Please find below the results of your literature search request.

If you would like the full text of any of the abstracts included, or would like a further search completed on this topic, please let us know.

We’d appreciate feedback on your satisfaction with this literature search. Please visit http://www.hello.nhs.uk/literature_search_feedback.asp and complete the form.

Thank you

Literature search results

<table>
<thead>
<tr>
<th>Search completed for:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Search required by:</td>
<td>14 August 2013</td>
</tr>
<tr>
<td>Search completed on:</td>
<td>12th August 2013</td>
</tr>
<tr>
<td>Search completed by:</td>
<td>Richard Bridgen</td>
</tr>
</tbody>
</table>

Search details

Efficacy, treatment duration and prevention of next psychotic relapse of olanzapine and aripiprasole in drug-induced psychoses.

Resources searched

NHS Evidence; TRIP Database; Cochrane Library; EMBASE; MEDLINE; PsychINFO; Google Scholar

**Database search terms:** olanzapine; zyprexa; symbyax; aripiprazole; abilify; psychotic adj2 disorder*; exp PSYCHOTIC DISORDERS; schizophren*; schizoaffective adj2 disorder*; delusion* adj2 disorder*; exp SCHIZOPHRENIA; schizoaffective; delusion*; psychosis; psychoses; "drug induced psychos*"; "drug-induced psychos*"; drug-induc*; drug* adj2 induc* adj2 psychos*; medicine* adj2 induc*; substance* adj2 induc*; pharmacol* adj2 induc*; medication* adj2 induc*; stimulant* adj2 induc*; exp ADVERSE DRUG REACTION;

**Evidence search string(s):** (inducing OR induced) (medicine OR medicines OR drug OR drugs OR medication OR pharmacology OR pharmaceutical OR stimulant OR stimulants OR substance OR substances ) (psychosis OR psychoses OR psychotic OR schizophrenia OR schizoaffective OR delusion OR delusions OR delusional) (olanzapine OR aripiprasole OR zyprexa OR symbyax OR abilify)

**Google search string(s):** (drug-inducing OR drug-induced) (psychosis OR psychoses OR psychotic OR schizophrenia OR schizoaffective OR delusions) (olanzapine OR aripiprazole OR zyprexa OR symbyax OR abilify)

Summary

There is a lot of research even given your search, amended to include drug-induced psychoses. Overall the efficacy of olanzapine and aripiprasole look promising, and there
Guidelines and Policy

British Association of Psychopharmacology
Evidence-based guidelines for the pharmacological treatment of schizophrenia: recommendations 2011

NICE
CG155 Psychosis and schizophrenia in children and young people 2013
CG120 Psychosis with coexisting substance misuse 2011
CG82 Schizophrenia (update) 2009
CG35 Parkinson's disease 2006

Evidence-based reviews

Cochrane Database of Systematic Reviews
Treatment for amphetamine psychosis 2008

Only one RCT of treatment for amphetamine psychosis has been published. Outcomes from this trial indicate that antipsychotic medications effectively reduce symptoms of amphetamine psychosis, the newer generation and more expensive antipsychotic medication, olanzapine, demonstrates significantly better tolerability than the more affordable and commonly used medication, haloperidol.

There are other two studies that did not meet the inclusion criteria for this review. The results of these two studies show that agitation and some psychotic symptoms may be abated within an hour after antipsychotic injection.

Whether this limited evidence can be applied for amphetamine psychotic patients is not yet known.

The medications that should be further investigate are conventional antipsychotics, newer antipsychotics and benzodiazepines. However, naturalistic studies of amphetamine psychotic symptoms and the prevalence of relapse to psychosis in the presence of amphetamine, are also crucial for advising the development of study designs appropriate for further treatment studies of amphetamine psychosis.

Database of Abstracts of Reviews of Effects
Antipsychotic drugs and extrapyramidal side effects in first episode psychosis: a systematic review of head-head comparisons 2013

Second generation antipsychotics offer an advantage over first generation drugs with respect to extrapyramidal adverse effects in patients with first episode psychosis, but the evidence largely related to comparisons with haloperidol.

Antipsychotic agents for the treatment of substance use disorders in patients with and without comorbid psychosis 2010

Studies of patients with comorbid psychosis suggested that atypical antipsychotics, especially clozapine, may decrease alcohol and drug use disorders. Studies of patients without comorbid psychosis suggested that atypical antipsychotics be beneficial in treating alcohol dependence in some alcoholic subpopulations, but may not be effective in treating stimulant dependence and aggravated the condition in some cases.

A meta-analysis of the risk of acute extrapyramidal symptoms with intramuscular antipsychotics for the treatment of agitation 2008

Intramuscular second-generation antipsychotics had a lower risk of acute extrapyramidal
symptoms than haloperidol alone, for acute anxiety. However, the extrapyramidal symptoms risk associated with second-generation antipsychotics and with haloperidol plus promethazine was comparable.

Second-generation versus first-generation antipsychotic drugs for schizophrenia: a meta-analysis 2008

There is substantial variation in the properties of second-generation antipsychotic drugs. Small to medium effects are possible following treatment with amisulpride, clozapine, olanzapine and risperidone in terms of overall efficacy and positive and negative symptoms. Second generation drugs can also result in fewer extrapyramidal side effects, but can induce weight gain.

Treating dopamimetic psychosis in Parkinson's disease: structured review and meta-analysis 2007

Evidence only supports the use of clozapine for the treatment of drug-induced psychosis in patients with Parkinson’s disease; olanzapine should not be used, and quetiapine should not be used unless proven to be effective.

New generation antipsychotics versus low-potency conventional antipsychotics: a systematic review and meta-analysis 2003

Optimum doses of low-potency conventional antipsychotics might not induce more EPS than new-generation drugs. The authors also concluded that new-generation drugs may have higher efficacy than the conventional treatments.

Efficacy and extrapyramidal side-effects of the new antipsychotics olanzapine, quetiapine, risperidone, and sertindole compared to conventional antipsychotics and placebo: a meta-analysis of randomized controlled trials 1999

This meta-analysis suggests that patients treated with olanzapine and risperidone showed a greater overall improvement than patients treated with haloperidol or other conventional antipsychotics. Both quetiapine and sertindole turned out to be as effective as haloperidol and all compounds analysed were superior to placebo with regards to antipsychotic efficacy. In addition, all new antipsychotics were associated with less frequent use of antiparkinson medications than haloperidol, with risperidone appearing to have slightly less favourable EPS-profile than the other new antipsychotics. This meta-analysis confirms that not only atypical but also conventional antipsychotics such as haloperidol are effective against negative symptoms in general.

NIHR Health Technology Assessments

Randomised controlled trials of conventional antipsychotic versus new atypical drugs, and new atypical drugs versus clozapine, in people with schizophrenia responding poorly to, or intolerant of, current drug treatment 2009

Atypical Antipsychotic Drugs in Schizophrenia 2003

**Published research – Databases**

1. Aripiprazole for treating cannabis-induced psychotic symptoms in ultrahigh-risk individuals

**Author(s)** Rolland B., Geoffroy P.A., Jardri R., Cottencin O.

**Citation:** Clinical Neuropharmacology, May 2013, vol./is. 36/3(98-99), 0362-5664;1537-162X (May-June 2013)

**Publication Date:** May 2013

**Abstract:** Cannabis-induced psychotic symptoms (CIPSs) have both similarities and differences with positive symptoms of schizophrenia, and it remains unclear whether CIPSs result from dopaminergic mechanisms and can be treated with antipsychotics. We report the case of a 22-year-old male patient with ultrahigh risk criteria for psychosis, who reported cannabis addiction and recurrent CIPSs. Aripiprazole 10 mg/d could totally and durably suppress CIPSs in the patient, but had no effect on the smoking level. Treating
CIPSs in ultrahigh risk individuals who cannot stop or refuse stopping cannabis might fit a harm-reduction strategy by preventing transition into psychosis. Copyright 2013 by Lippincott Williams & Wilkins.

Source: EMBASE

2. A new perspective for schizophrenia: TAAR1 agonists reveal antipsychotic- and antidepressant-like activity, improve cognition and control body weight


Citation: Molecular Psychiatry, May 2013, vol./is. 18/5(543-556), 1359-1418;1476-5578 (May 2013)

Publication Date: May 2013

Abstract: Schizophrenia is a chronic, severe and highly complex mental illness. Current treatments manage the positive symptoms, yet have minimal effects on the negative and cognitive symptoms, two prominent features of the disease with critical impact on the long-term morbidity. In addition, antipsychotic treatments trigger serious side effects that precipitate treatment discontinuation. Here, we show that activation of the trace amine-associated receptor 1 (TAAR1), a modulator of monoaminergic neurotransmission, represents a novel therapeutic option. In rodents, activation of TAAR1 by two novel and pharmacologically distinct compounds, the full agonist RO5256390 and the partial agonist RO5263397, blocks psychostimulant-induced hyperactivity and produces a brain activation pattern reminiscent of the antipsychotic drug olanzapine, suggesting antipsychotic-like properties. TAAR1 agonists do not induce catalepsy or weight gain; RO5263397 even reduced haloperidol-induced catalepsy and prevented olanzapine from increasing body weight and fat accumulation. Finally, TAAR1 activation promotes vigilance in rats and shows pro-cognitive and antidepressant-like properties in rodent and primate models. These data suggest that TAAR1 agonists may provide a novel and differentiated treatment of schizophrenia as compared with current medication standards: TAAR1 agonists may improve not only the positive symptoms but also the negative symptoms and cognitive deficits, without causing adverse effects such as motor impairments or weight gain. 2013 Macmillan Publishers Limited All rights reserved.

Source: EMBASE

3. Prior haloperidol, but not olanzapine, exposure augments the pursuit of reward cues: implications for substance abuse in schizophrenia.

Author(s) Bedard AM, Maheux J, Levesque D, Samaha AN

Citation: Schizophrenia Bulletin, May 2013, vol./is. 39/3(692-702), 0586-7614;1745-1701 (2013 May)

Publication Date: May 2013

Abstract: Drug abuse and addiction are excessively common in schizophrenia. Chronic antipsychotic treatment might contribute to this comorbidity by inducing supersensitivity within the brain's dopamine system. Dopamine supersensitivity can enhance the incentive motivational properties of reward cues, and reward cues contribute to the maintenance and severity of drug addiction. We have shown previously that rats withdrawn from continuous haloperidol (HAL) treatment (via subcutaneous minipump) develop dopamine supersensitivity and pursue reward cues more vigorously than HAL-naïve rats following an amphetamine (AMPH) challenge. Atypical antipsychotic drugs are thought to be less likely than typicals to produce dopamine supersensitivity. Thus, we compared the effects of HAL and the atypical antipsychotic olanzapine (OLZ) on the pursuit of reward cues. Rats were trained to associate a light-tone cue with water then treated with HAL or OLZ. Following antipsychotic withdrawal, we assessed AMPH-induced enhancement of lever pressing for the cue. Withdrawal from HAL, but not from OLZ, enhanced this effect. HAL, but not OLZ, also enhanced AMPH-induced psychomotor activation and c-fos mRNA expression in the caudate-putamen. Thus, prior HAL, but not OLZ, enhanced conditioned reward following an AMPH challenge, and this was potentially linked to enhanced behavioral sensitivity to
AMPH and AMPH-induced engagement of the caudate-putamen. These findings suggest
that HAL, but not an atypical like OLZ, modifies reward circuitry in ways that increase
responsiveness to reward cues. Because enhanced responsiveness to reward cues can
promote drug-seeking behavior, it should be investigated whether atypical antipsychotics
might be a preferential option in schizophrenic patients at risk for drug abuse or addiction.

Source: Medline


Author(s) Mangewala, Vikas, Sarwar, Sajjad R, Shah, Kavit, Singh, Tanvir

Citation: Innovations in Clinical Neuroscience, February 2013, vol./is. 10/2(10-11), 2158-
8333:2158-8341 (Feb 2013)

Publication Date: February 2013

Abstract: Presents a case study of a 15-year-old boy with no previous psychiatric history.
He came to the emergency room (ER) with complaints of agitation and psychotic
symptoms. Reportedly, the patient smoked marijuana that was laced with bath salts. Soon
after use, the patient became paranoid, barricading himself in his father’s home. Police
were called to gain forcible entry into the house, and the patient was brought to the ER.
During his inpatient psychiatric stay, the patient was treated with combination of olanzapine
5mg once daily and lorazepam 0.5mg twice daily. The dose of olanzapine was increased to
7.5mg daily, and this combined with the lorazepam improved his symptoms of psychosis
and agitation. As a result he began to interact with the peers on the unit. Symptoms of
paranoia improved within three days of treatment, and the patient was discharged home on
olanzapine 7.5mg once daily and lorazepam 0.5mg twice daily with outpatient follow-up.
The patient had no relapse of symptoms in the eight-week follow-up period. He denied
current substance use. No further adjustments in the medication dosages were required. In
conclusion these substances are ingested, smoked, and injected as legal alternatives to
stimulants that are detected in routine drug testing. Our case adds to a rapidly evolving
literature that indicates the potential for acute psychiatric toxicity due to recreational use of
compounds. (PsycINFO Database Record (c) 2013 APA, all rights reserved)

Source: PsycINFO

Available in fulltext from Innovations in Clinical Neuroscience at National Library of
Medicine

5. Benzodiazepines for psychosis-induced aggression or agitation.

Author(s) Gillies D, Sampson S, Beck A, Rathbone J

Citation: Cochrane Database of Systematic Reviews, 2013, vol./is. 4/(CD003079), 1361-
6137:1469-493X (2013)

Publication Date: 2013

Abstract: BACKGROUND: Acute psychotic illness, especially when associated with
agitated or violent behaviour, can require urgent pharmacological tranquillisation or
sedation. In several countries, clinicians often use benzodiazepines (either alone or in
combination with antipsychotics) for this outcome.OBJECTIVES: To estimate the effects of
benzodiazepines, alone or in combination with antipsychotics, when compared with
placebo or antipsychotics, alone or in combination with antihistamines, to control disturbed
behaviour and reduce psychotic symptoms.SEARCH METHODS: We searched the
Cochrane Schizophrenia Group’s register (January 2012), inspected reference lists of
included and excluded studies and contacted authors of relevant studies.SELECTION
CRITERIA: We included all randomised clinical trials (RCTs) comparing benzodiazepines
alone or in combination with any antipsychotics, versus antipsychotics alone or in
combination with any other antipsychotics, benzodiazepines or antihistamines, for people
with acute psychotic illnesses.DATA COLLECTION AND ANALYSIS: We reliably selected
studies, quality assessed them and extracted data. For binary outcomes, we calculated
standard estimates of relative risk (RR) and their 95% confidence intervals (CI) using a
fixed-effect model. For continuous outcomes, we calculated the mean difference (MD)
between groups. If heterogeneity was identified, this was explored using a random-effects
MAIN RESULTS: We included 21 trials with a total of n = 1968 participants. There was no significant difference for most outcomes in the one trial that compared benzodiazepines with placebo, although there was a higher risk of no improvement in people receiving placebo in the medium term (one to 48 hours) (n = 102, 1 RCT, RR 0.62, 95% CI 0.40 to 0.97, very low quality evidence). There was no difference in the number of participants who had not improved in the medium term when benzodiazepines were compared with antipsychotics (n = 308, 5 RCTs, RR 1.10, 95% CI 0.85 to 1.42, low quality evidence); however, people receiving benzodiazepines were less likely to experience extrapyramidal effects (EPS) in the medium term (n = 536, 8 RCTs, RR 0.15, 95% CI 0.06 to 0.39, moderate quality of evidence). Data comparing combined benzodiazepines and antipsychotics versus benzodiazepines alone did not yield any significant results. When comparing combined benzodiazepines/antipsychotics (all studies compared haloperidol) with the same antipsychotics alone (haloperidol), there was no difference in improvement in the medium term (n = 155, 3 RCTs, RR 1.27, 95% CI 0.94 to 1.70, very low quality evidence) but sedation was more likely in people who received the combination therapy (n = 172, 3 RCTs, RR 1.75, 95% CI 1.14 to 2.67, very low quality evidence). However, more participants receiving combined benzodiazepines and haloperidol had not improved by medium term when compared to participants receiving olanzapine (n = 60, 1 RCT, RR 25.00, 95% CI 1.55 to 403.99, very low quality evidence) or ziprasidone (n = 60, 1 RCT, RR 4.00, 95% CI 1.25 to 12.75 very low quality evidence). When haloperidol and midazolam were compared with olanzapine, there was some evidence the combination was superior in terms of improvement, sedation and behaviour.AUThORS’ CONCLUSIONS: The evidence from trials for the use of benzodiazepines alone is not good. There were relatively little good data and most trials are too small to highlight differences in either positive or negative effects. Adding a benzodiazepine to other drugs does not seem to confer clear advantage and has potential for adding unnecessary adverse effects. Sole use of older antipsychotics unaccompanied by anticholinergic drugs seems difficult to justify. Much more high quality research is needed in this area.

Source: Medline
Available in fulltext from Cochrane Library, The at Wiley


Author(s) Dannaram, Srinivas, Borra, Dileep, Pulluri, Madhuri, Jindal, Prachi, Sharma, Ashish

Citation: Innovations in Clinical Neuroscience, October 2012, vol./is. 9/10(10-11), 2158-8333;2158-8341 (Oct 2012)

Publication Date: October 2012

Abstract: Presents a case report of a 50-year old man who presented with a first psychotic episode secondary to an increase in dose of levetiracetam. The patient was admitted to our medical floor with a first episode of psychosis. His medical history was significant for partial complex seizures since he was three years old, migraines, gastroesophageal reflux disease (GERD). Medications on admission were levetiracetam 1000 mg orally twice daily, gabapentin 600mg orally at bedtime, valproic acid 500mg orally twice daily, and esomeprazole 40mg orally once daily. Physical examination was normal through out the hospital stay. Diagnostic work up included a complete blood cell count differential, comprehensive metabolic panel, thyroid function tests, blood alcohol level, urine analysis, urine drug screen, computed tomography scan and magnetic resonance imaging of the head, electroencephalography, and valproic acid and levetiracetam levels, which were all normal. Based on history, physical examination, and diagnostic tests, a diagnosis of levetiracetam induced psychosis was made. Levetiracetam was stopped and olanzapine 5mg orally, as needed, every six hours, was started. The patient recovered from his psychotic symptoms over next 48 hours and was discharged home. (PsycINFO Database Record (c) 2013 APA, all rights reserved)

Source: PsycINFO
Available in fulltext from Innovations in Clinical Neuroscience at National Library of Medicine
7. Synthetic cannabinoid-induced psychosis: Two adolescent cases.

**Author(s)** Oluwabusi, Olumide O, Lobach, Liudmila, Akhtar, Umair, Youngman, Branden, Ambrosini, Paul J

**Citation:** Journal of Child and Adolescent Psychopharmacology, October 2012, vol./is. 22/5(393-395), 1044-5463;1557-8992 (Oct 2012)

**Publication Date:** October 2012

**Abstract:** Presents case reports of two adolescent patients with synthetic cannabinoid induced psychosis. The first patient is a 16-year old Hispanic male who was admitted to a psychiatric unit with new onset psychosis. On the psychiatric unit, he was stabilized on quetiapine, however this was changed to aripiprazole shortly after discharge secondary to an acute dystonic reaction. He became stable and returned to baseline functioning, but he relapsed after 3 months of treatment with aripiprazole. At this presentation, he developed severe agitation, lability of mood, increased irritability, increased energy, pressure of speech, flights of ideas, paranoid and grandiose delusions, musical auditory hallucinations, and colorful visual hallucinations. He returned to his premorbid level of functioning after a few days of inpatient care, and he was discharged home for follow-up with outpatient psychiatric services. The second patient is a 17-year old Hispanic male who was admitted to an adolescent psychiatric unit with new onset psychosis. He was readmitted 7 months later with paranoid ideations, ideas of reference, somatic delusions, decreased motivation, thought blocking, and bizarre behavior. His psychotic symptoms subsided within a few days after he recommenced olanzapine at a maintenance dose of 15 mg. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

**Source:** PsycINFO


**Author(s)** Sulaiman A.H., Gill J.S., Said M.A., Habil M.H., Zainal N.Z., Guan N.C.

**Citation:** Klinik Psikofarmakoloji Bulteni, July 2012, vol./is. 22/2(121-129), 1017-7833;1302-9657 (July 2012)

**Publication Date:** July 2012

**Abstract:** Objective: This study aimed to explore the therapeutic effects and tolerability of aripiprazole in the treatment of psychosis among methamphetamine dependent patients. Methods: This was an open label single arm prospective study conducted at the University Malaya Medical Centre (UMMC). The study subjects included treatment naive patients with a current diagnosis of methamphetamine dependence with co-occurring acute psychotic symptoms based on the Diagnostic and Statistical Manual of Mental Disorders-IV (DSMIV). Eligible patients were treated with an initial dose of 5-10 mg aripiprazole followed by flexible doses (5-15mg/day) from day 2 to 14. Results: Out of 49 patients enrolled, 41 patients (83.7%) completed the study. At baseline the mean PANSS total score was 79.2+/−13.7 and the mean CGI-S score was 4.3+/−0.5. There was a statistically significant decline in the mean PANSS-total and CGI-S scores over the course of the study. The mean reduction was 27.6+/−21.4 point (p<0.05, 95% CI (-34.8, -20.4)) from baseline on day 14 for total PANSS score and 2.0+/−1.2 point (p<0.05, 95% CI (-2.4, -1.6) for CGI-S. Aripiprazole was generally well tolerated during the study. Adverse events were reported in 10 (20.4%) patients. No statistically significant changes were noted with respect to movement-related adverse events. Conclusions: This study found that aripiprazole improved the psychotic symptoms associated with methamphetamine use. It was generally well tolerated with mild to moderate adverse events. Based on these results aripiprazole might be an effective and safe option for the treatment of methamphetamine induced psychosis.

**Source:** EMBASE

9. Pattern of antipsychotic use and its determinants in Iran

**Author(s)** Mirabzadeh A., Khodaei M.R., Feizzadeh G., Samiei M.
**Citation**: European Psychiatry, 2012, vol./is. 27/, 0924-9338 (2012)

**Publication Date**: 2012

**Abstract**: Introduction: Antipsychotic monotherapy is recognized as the treatment of choice for patients with psychosis but concurrent use is increasingly common among both inpatients and outpatients. Objective: Previous studies of the prescription patterns of psychotropic medications in psychotic patients have highlighted a high rate of antipsychotic polypharmacy, but data in Iran are sparse. This study seeks to analyze prescribing patterns of antipsychotic use and to estimate associated risks in this patient group. Aims: The aim of this study was to investigate the prevalence and its determinants of Antipsychotic Use in patients with psychiatric disorders in the greatest psychiatric hospital (Razi) in Iran. Methods: This study was on patients with psychiatric disorder that have discharged from the hospital during four months. We have assessed all patients with psychiatric interview and evaluation of their psychiatric documentations. Results: Results of this study indicated that the most prevalent of psychiatric diagnosis was schizophrenia (33.42%) and then Bipolar I Disorder, Schizoaffective Disorder, Mental Retardation and Amphetamine-Induced Psychotic Disorder. 90.7% of all of patients had taken antipsychotic medications and antipsychotic polypharmacy was in 27.2% of these patients. The most prevalent component of antipsychotic polypharmacy was consisting of Chlorpromazine, Haloperidol and Chlorpromazine, Risperidone and then Chlorpromazine, Olanzapine respectively. There were significant relations between pattern of antipsychotic use and gender, occupation status, type of psychiatric ward, duration of hospitalization and cost of treatment but no relationship with age, educational status and duration of illness. Conclusion: This study suggests that prevalence of antipsychotic polypharmacy is high in in-patient psychiatric patients.

**Source**: EMBASE

---

10. **Effect of aripiprazole on anxiety associated with ethanol physical dependence and on ethanol-induced place preference**

**Author(s)** Shibasaki M., Kurokawa K., Mizuno K., Ohkuma S.

**Citation**: Journal of Pharmacological Sciences, 2012, vol./is. 118/2(215-224), 1347-8613;1347-8648 (2012)

**Publication Date**: 2012

**Abstract**: In the present study, we investigated the effect of aripiprazole, a dopamine system stabilizer, on ethanol-induced psychological and physiological dependence and anxiety-like behavior. First we determined the effect of aripiprazole, a dopamine system stabilizer, on the development and expression of ethanol-induced place preference. Both the development and expression of ethanol-induced place preference was significantly suppressed by treatment of aripiprazole. Next, the withdrawal score gradually increased with increasing duration after the withdrawal from ethanol for 6 days in vehicle-treated mice and the maximal score was observed 10 h after the ethanol withdrawal. Aripiprazole caused no changes in the withdrawal score as compared to vehicletreated mice. Under these conditions we investigated the effect of aripiprazole on the anxiety-like behavior of ethanol physical dependent mice, which were animals subjected to ethanol vapor for 6 days. The significant decrease of time spent in the open arms and number of open arm entries characterize the anxiety-like behavior in ethanol physical dependent mice, compared to control mice. These decreases were reversed by treatment of aripiprazole, which were inhibited by WAY100635, a serotonin 5-HT<sub>1A</sub> receptor antagonist. The present findings suggest that aripiprazole was efficient for reversing ethanol-induced place preference and anxiety-like behavior. The Japanese Pharmacological Society.

**Source**: EMBASE

---

11. **Temazepam withdrawal induced psychosis.**

**Author(s)** Alexander, Jacob, Tibrewal, Prashant, Fantasia, Robert

**Citation**: Asian Journal of Psychiatry, September 2011, vol./is. 4/3(224-225), 1876-2018;1876-2026 (Sep 2011)
Abstract: Presents a case report of 58-year-old lady with a long history of major depressive disorder that appeared to be well controlled on a regime of mirtazapine and temazepam. She presented to emergency services following the acute onset of psychotic symptoms in conjunction with temazepam cessation during the preceding week. She satisfied criteria for a substance induced psychotic disorder with onset during withdrawal. There was no family history of psychosis and no history of substance abuse involving hallucinogens. Her psychotic symptoms resolved rapidly in 3 days with the institution of lorazepam leading to a discharge from hospital on the 6th day. No antipsychotic medication was prescribed for the management of psychotic symptoms apart from a single dose of olanzapine (10 mg) at arrival in the emergency services. The patient was successfully weaned off benzodiazepines over the next few months. Perceptual distortions, hallucinations, delusions and paranoid thoughts have been documented with benzodiazepine withdrawal. A greater knowledge and appreciation of this complication has important implications for the appropriate management of benzodiazepine withdrawal. In light of the above, some clinical discretion should be exercised while prescribing benzodiazepines and antidepressants together. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

12. Comparative pharmacology of antipsychotics possessing combined dopamine D2 and serotonin 5-HT1A receptor properties.

Author(s) Newman-Tancredi A, Kleven MS

Citation: Psychopharmacology, August 2011, vol./is. 216/4(451-73), 0033-3158;1432-2072 (2011 Aug)

Abstract: RATIONALE: There is increasing interest in antipsychotics intended to manage positive symptoms via D(2) receptor blockade and improve negative symptoms and cognitive deficits via 5-HT(1A) activation. Such a strategy reduces side-effects such as the extrapyramidal syndrome (EPS), weight gain, and autonomic disturbance liability.OBJECTIVE: This study aims to review pharmacological literature on compounds interacting at both 5-HT(1A) and D(2) receptors (as well as at other receptors), including aripiprazole, perospirone, ziprasidone, bifeprunox, lurasidone and cariprazine, PF-217830, adoprazine, SSR181507, and F15063 METHODS: We examine data on in vitro binding and agonism and in vivo tests related to (1) positive symptoms (e.g., psychostimulant-induced hyperactivity or prepulse inhibition deficit), (2) negative symptoms (e.g., phencyclidine-induced social interaction deficits and cortical dopamine release), and (3) cognitive deficits (e.g., phencyclidine or scopolamine-induced memory deficits). EPS liability is assessed by measuring catalepsy and neuroendocrine impact by determining plasma prolactin, glucose, and corticosterone levels.RESULTS: Compounds possessing "balanced" 5-HT(1A) receptor agonism and D(2) antagonism (or weak partial agonism) and, in some cases, combined with other beneficial properties, such as 5-HT(2A) receptor antagonism, are efficacious in a broad range of rodent pharmacological models yet have a lower propensity to elicit EPS or metabolic dysfunction.CONCLUSIONS: Recent compounds exhibiting combined 5-HT(1A)/D(2) properties may be effective in treating a broader range of symptoms of schizophrenia and be better tolerated than existing antipsychotics. Nevertheless, further investigations are necessary to evaluate recent compounds, notably in view of their differing levels of 5-HT(1A) affinity and efficacy, which can markedly influence activity and side-effect profiles.

Source: Medline

Available in fulltext from Psychopharmacology at EBSCOhost


Author(s) Ekinci, Ozalp, Sabuncuoglu, Osman

Citation: Progress in Neuro-Psychopharmacology & Biological Psychiatry, March 2011,
**Publication Date:** March 2011

**Abstract:** Presents the case report of MG, a 17 year old male adolescent, who was admitted for the complaints of difficulty paying attention at school, forgetfulness and making careless mistakes in exams. A diagnosis of attention-deficit/hyperactivity disorder (ADHD) was made. MPH long acting preparation (Concerta) was started and gradually increased. At his first month visit, for the target symptoms of impulsive aggressive behaviors, risperidone was initiated. At his 3rd month visit, despite the stable improvement in his school grades, MG's impulsive aggressive behaviors were reported to be still present. A switch from risperidone to aripiprazole was planned and risperidone dose was gradually tapered keeping MPH dose constant. In the 3rd day of aripiprazole treatment, MG was admitted to the emergency clinic by the complaints of hearing strange voices, talking to himself and thoughts of having a demon inside of him. With the diagnosis of a medication induced psychotic reaction, aripiprazole was stopped and risperidone was re-started. The MPH dose was also lowered. As a result, his complaints decreased. In his follow up, regular monthly visits continued for three months and no recurrence of psychotic symptoms was reported. Thus, this case report highlights the need to be cautious when switching from risperidone to aripiprazole in adolescents who are on MPH. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

**Source:** PsycINFO

---

**14. Caffeine induced psychotic exacerbation.**

**Author(s)**: Tibrewal, Prashant, Dhillon, Rohan

**Citation:** Australian and New Zealand Journal of Psychiatry, February 2011, vol./is. 45/2(179-180), 0004-8674;1440-1614 (Feb 2011)

**Publication Date:** February 2011

**Abstract:** Presents a case report of a 52-year-old man who lives in a supported residential facility (SRF). He has a long-standing history of paranoid schizophrenia that has been relatively stable with mild residual positive symptoms over the last few years. He was being managed with parenteral risperidone 100 mg intramuscular injection fortnightly. He presented with two weeks' history of psychotic relapse characterized by predominant delusions of persecution and reference of people following him in order to rape him. He was admitted to our inpatient unit and was started on olanzapine to control his agitation and psychotic symptoms. His acute psychotic symptoms settled within ten days of closed ward admission without any changes to his regular psychotropic prescription. He was discharged to his SRF with recommendations to monitor and manage his coffee intake. The patient represented after two months with similar acute psychotic symptoms in the background of increasing coffee consumption. The rapid resolution of psychotic symptoms without additional drug therapy and with the restriction of caffeine intake would suggest that increased dopaminergic activity was the primary mechanism to explain the acute worsening of the patient's psychosis. Our case report offers evidence that the screening of caffeine consumption can be of importance in a certain group of vulnerable psychotic patients. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

**Source:** PsycINFO

Available in fulltext from Australian and New Zealand Journal of Psychiatry at EBSCOhost

---

**15. Probable psychosis associated with levetiracetam: A case report.**

**Author(s)**: Aggarwal, Ashish, Sharma, Dinesh D, Sharma, Ravi C, Kumar, Ramesh

**Citation:** Progress in Neuro-Psychopharmacology & Biological Psychiatry, January 2011, vol./is. 35/1(274-275), 0278-5846 (Jan 15, 2011)

**Publication Date:** January 2011

**Abstract:** Presents a case report of a 20 year old male who was brought to a psychiatry outpatient department with complaints of violent abusive behavior, agitation, muttering and
gesticulating to self with disturbed biological functions for the past 3 days. Past history revealed that the patient had poor scholastic performance since the age of 11 years after he had developed febrile encephalopathy. He was also suffering from generalized tonic clonic seizures as well as left simple partial seizures. The patient also had history of bilateral Chronic Serous Otitis Media and had marked decrease in hearing. He was initially given divalproex up to 2000 mg per day but his seizures were not adequately controlled. He would have around one seizure in about 15 day. The patient reported improvement in symptoms from the second day and was asymptomatic by 7th day. His olanzapine and lorazepam were stopped and he was maintained on levetiracetam 1000 mg per day along with divalproex 1000 mg per day without any recurrence of seizure or psychosis for the next 3 months of follow up. In our case, the close temporal relationship between psychosis and increase in levetiracetam dose and prior reports of levetiracetam induced psychosis and rapid resolution of symptoms following decrease in the dose of levetiracetam supported our hypothesis of levetiracetam induced psychosis. To conclude, there is no doubt that antiepileptic drugs may lead to various psychiatric adverse effects, including psychosis. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

16. Effects of asenapine, olanzapine, and risperidone on psychotomimetic-induced reversal-learning deficits in the rat.

Author(s) McLean SL, Neill JC, Idris NF, Marston HM, Wong EH, Shahid M

Citation: Behavioural Brain Research, December 2010, vol./is. 214/2(240-7), 0166-4328;1872-7549 (2010 Dec 25)

Publication Date: December 2010

Abstract: BACKGROUND: Asenapine is a new pharmacological agent for the acute treatment of schizophrenia and bipolar disorder. It has relatively higher affinity for serotonergic and alpha(2)-adrenergic than dopaminergic D(2) receptors. We evaluated the effects of asenapine, risperidone, and olanzapine on acute and subchronic psychotomimetic-induced disruption of cued reversal learning in rats.METHODS: After operant training, rats were treated acutely with d-amphetamine (0.75 mg/kg intraperitoneally [i.p.]) or phencyclidine (PCP; 1.5mg/kg i.p.) or subchronically with PCP (2mg/kg i.p. for 7 days). We assessed the effects of acute coadministration of asenapine, risperidone, or olanzapine on acute d-amphetamine- and PCP-induced deficits and the effects of long-term coadministration of these agents (for 28 additional days) on the deficits induced by subchronic PCP.RESULTS: Deficits in reversal learning induced by acute d-amphetamine were attenuated by risperidone (0.2mg/kg i.p.). Acute PCP-induced impairment of reversal learning was attenuated by acute asenapine (0.025 mg/kg subcutaneously [s.c.]), risperidone (0.2mg/kg i.p.), and olanzapine (1.0mg/kg i.p.). Subchronic PCP administration induced an enduring deficit that was attenuated by acute asenapine (0.075 mg/kg s.c.) and by olanzapine (1.5mg/kg i.p.). Asenapine (0.075 mg/kg s.c.), risperidone (0.2mg/kg i.p.), and olanzapine (1.0mg/kg i.p.) all showed sustained efficacy with chronic (29 days) treatment to improve subchronic PCP-induced impairments.CONCLUSION: These data suggest that asenapine may have beneficial effects in the treatment of cognitive symptoms in schizophrenia. However, this remains to be validated by further clinical evaluation. Copyright (c) 2010 Elsevier B.V. All rights reserved.

Source: Medline

17. A 40-year-old man with acute psychosis.

Author(s) Kumar, Sanjeev, Bankole, Aziza

Citation: Psychiatric Annals, December 2010, vol./is. 40/12(600-603), 0048-5713;1938-2456 (Dec 2010)

Publication Date: December 2010

Abstract: Presents a case report of a 40-year-old white man who was admitted to inpatient service involuntarily on a court order for acute psychosis and later diagnosed with alcohol-induced psychotic disorder (AIPD). He had been expressing bizarre delusions about "the
devil" and winning the lottery. He expressed paranoid delusions about people being after him for his money. He also experienced auditory and visual hallucinations. He did not have any suicidal or homicidal thoughts and was involuntarily committed to treatment based on his inability to take care of himself. The patient had a history of generalized anxiety disorder and had been prescribed citalopram, sertraline, and quetiapine. He was not taking any of these medications at the time of his hospitalization. He gave a history of chronic alcohol use with recurrent remissions and relapses. There was no euphoria or pressured speech, and the patient was fully alert and oriented to time, place, and person. He had a mini-mental status examination (MMSE) of 28/30. He was given vitamins, including thiamine and mineral supplements. Psychosis was managed with olanzapine titrated up to 20 mg. He responded well to the medication, and the intensity of his delusions decreased. He was also started on citalopram for anxiety. He was discharged with outpatient case management services on 20 mg of olanzapine and 40 mg of citalopram once daily after 10 days of hospital stay. His delusions and hallucinations had ceased. He remained symptom free for several months after this hospital stay. Patients who are misdiagnosed with schizophrenia are likely to get prolonged antipsychotic treatment, which might not be warranted in all the cases. Also, failure to distinguish AIPD from withdrawal states may result in the withholding of treatment, resulting in prolonged morbidity. The need for increased awareness among clinicians and further large-scale prospective studies cannot be overemphasized. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

18. Clinical comparison of schizophrenia spectrum disorder with and without cannabis dependency

Author(s) Makkos Z., Fejes L., Inczedy-Farkas G., Kassai-Farkas A., Faludi G., Lazary J.

Citation: Neuropsychopharmacologia Hungarica, October 2010, vol./is. 12/(30-31), 1419-8711 (October 2010)

Publication Date: October 2010

Abstract: Background Numerous clinical studies support the relation between cannabis abuse and psychotic disorders and by now several ways are known where they are linked. Although incidence of schizophrenia is higher among cannabis users and marijuana is the most common abused drug by adolescents, etiological linkage between schizophrenia and cannabis use is still not clarified. Clinical experiences suggest that regular cannabis user can show similar psychotic episode to schizophrenic disorders but it still unclear if cannabis induced psychotic disorder is a distinct entity requiring special therapy or regular cannabis use consequently lead up to schizophrenia. Therefore, we compared retrospectively psychotic patients with and without cannabis use by clinical profile.

Methods Clinical data of 85 patients with schizophrenia spectrum disorder were analyzed retrospectively. Cannabis use was not reported by 43 persons (Cnbs0 subgroup) and 42 patients used regularly cannabis during at least 1 year (Cnbs1 subgroup). Comparison of anamnesis, family history, social-demographic condition, positive and negative symptoms, acute and long-term therapy recorded by clinical interviews was performed with chi square tests, logistic binary regression and t-tests using SPSS 13.0 for Windows software.

Results Men were over-represented in cannabis abuser group while mean age was lower among them compared to Cnbs0 subgroup. Prevalence of suicidal attempt was increased in men without cannabis use (OR=5.25, p=0.016). Patients without cannabis use spent more time in hospital (p=0.026) and smoking was more frequent among them (OR=1.36, p=0.047). The chance to get olanzapine for acute therapy and aripiprazol for long term therapy was more than two fold in Cnbs1 subgroup (OR=2.66, OR=3.67, respectively). However, aripiprazol was used for acute therapy with significantly lower risk in Cnbs1 subgroup (OR=0.47, p=0.023). Olanzapine was admitted for long term therapy in a higher dose to Cnbs0 patients (p=0.040). Also, higher dose of risperidon LAI was used in women without cannabis dependency compared to women of Cnbs1 subgroup (p=0.020). Positive and negative symptoms and family history did not differ significantly between the two subgroups.

Conclusion Although symptom profile was similar, hospitalization time, suicidal anamnesis, smoking habit and aldosage, intensity and lasting of therapy was different between the two subgroups. Further prospective studies are required for investigation of the clinical and molecular background of this discrepancy to determine relevant protocol of prevention and treatment of the chronic cannabis use related psychotic
19. Aripiprazole attenuates established behavioral sensitization induced by methamphetamine.

**Author(s)** Futamura, Takashi, Akiyama, Satoshi, Sugino, Haruhiko, Forbes, Andy, McQuade, Robert D, Kikuchi, Tetsuro

**Citation:** Progress in Neuro-Psychopharmacology & Biological Psychiatry, August 2010, vol./is. 34/6(1115-1119), 0278-5846 (Aug 16, 2010)

**Publication Date:** August 2010

**Abstract:** Psychostimulant-induced behavioral sensitization is an experimental model of the stimulant psychosis and the vulnerability to relapse in schizophrenia. This study investigated the effects of aripiprazole, an antipsychotic drug that has dopamine D2 receptor partial agonist activity, on established sensitization induced by methamphetamine (MAP) in mice. Repeated treatment with MAP (1.0 mg/kg, s.c.) for 10 days progressively increased the ability of MAP to increase locomotor activity. The enhanced locomotion induced by a challenge dose of MAP (0.24 mg/kg, s.c.) also occurred after withdrawal from MAP pretreatment. Repeated treatment with aripiprazole from days 10 to 14 during withdrawal from MAP administration attenuated the effect of MAP pretreatment, enhancing the motor response to a challenge dose of stimulant 3 days after the aripiprazole preparation. In contrast, sulpiride, a dopamine D2 receptor specific antagonist, and risperidone, a serotonin 5-HT2 and dopamine D2 receptor antagonist, did not show effects similar to aripiprazole. The attenuation effect of aripiprazole was blocked by pretreatment with the specific serotonin 5-HT1A antagonist WAY100635. These results of aripiprazole suggest that the attenuation effect of aripiprazole was mediated by 5-HT1A receptors and imply that aripiprazole may have therapeutic value in treating drug-induced psychosis and schizophrenia. (PsycINFO Database Record (c) 2012 APA, all rights reserved) (journal abstract)

**Source:** PsycINFO

---

20. The effect of olanzapine on a tetrahydrocannabinol-induced increase on the positive and negative syndrome scale

**Author(s)** Kleinloog H.D., Liem-Moolenaar M., Jacobs G.E., De Kam M.L., Van Gerve J.M.A.

**Citation:** European Neuropsychopharmacology, August 2010, vol./is. 20/(S265), 0924-977X (August 2010)

**Publication Date:** August 2010

**Abstract:** Introduction: Psychosis is a very complex derangement of the human central nervous system, which cannot be modelled adequately in preclinical models. An in-human model using a psychoactive drug to induce psychotomimetic symptoms in healthy volunteers could constitute an alternative. Tetrahydrocannabinol (THC) has previously been described to induce psychomimetic symptoms in healthy volunteers [1], which could be significantly reduced following co-administration of haloperidol in a study with a crossover design [2]. Objective: To further validate an in-human model of psychosis and antipsychotic drug action. Methods: This was a double-blind, randomized, placebo-controlled, five-way cross-over interaction trial in healthy volunteers with previous mild cannabis exposure. Intrapulmonary inhalations of purified THC in three subsequent dosages of 2, 4 and 6 mg with a 90 minute interval were used to induce psychomimetic symptoms. A single oral dose of olanzapine 10 mg, diphenhydramine 2x15 mg (as positive control), or matching placebos was administered before inhalations of THC or placebo. The positive subscale of the Positive and Negative Syndrome Scale (PANSS) was scored independently by two raters, following a structured clinical interview at baseline and after each THC inhalation. The visual analogue scales (VAS) for psychomimetic symptoms are self-report questionnaires previously described by Bowdle [3] that were administered frequently throughout the study days. They were subdivided into VAS internal perception, VAS external perception and VAS feeling high. Results were logtransformed for analysis.
and back-transformed for descriptive purposes. Results are presented only for responders, which are defined as subjects that show an increase on the positive PANSS following THC administration. Results: Of the 49 included subjects, 39 were classified as responders. THC induced an increase on the positive PANSS by 1.9 points (95% CI 1.2–2.5; p < 0.0001) compared to placebo. Co-administration of olanzapine reduced this increase by 47.6% (0.9 point reduction; 95% CI 0.3–1.4; p = 0.0046), whereas coadministration of diphenhydramine had no effect. THC induced an increase on VAS feeling high of 7.7mm (95% CI 5.0–11.4; p < 0.0001) compared to placebo, which could be reduced by olanzapine by 38.8% (3.0mm reduction; 95% CI 0.5–4.8; p = 0.0218). VAS internal perception and VAS external perception were increased following THC administration by 0.2mm (95% CI 0.1–0.4; p = 0.0011) and 0.7mm (95% CI 0.4–1.0; p < 0.0001) respectively and not influenced significantly by either olanzapine or diphenhydramine. However, olanzapine showed a trend towards reduction of the effect on VAS external perception of 32.6% (0.2mm reduction; 95% CI 0.0–0.5; p = 0.0985). Conclusions: THC induces transient psychotomimetic effects in healthy volunteers, as measured on the positive subscale of the PANSS and VAS Bowdle. This effect can be reduced by coadministration of olanzapine, but not diphenhydramine. It seems as if the effect of THC on cannabinoid CB1 receptors can be influenced by the activity of olanzapine on dopamine D2 and serotonin 5-HT2 receptors. This model could be used to test the effect of novel drugs in development for psychosis in an early clinical phase.

Source: EMBASE

21. A double-blind, randomized, placebo-controlled, five-way cross-over interaction trial to investigate the inhibitory effect of olanzapine on a tetrahydrocannabinol-induced increase on the positive and negative syndrome scale

Author(s) Kleinloog D., Liem-Moolenaar M., Jacobs G., De Kam M., Van Gerven J.

Citation: International Journal of Neuropsychopharmacology, June 2010, vol./is. 13/(97-98), 1461-1457 (June 2010)

Publication Date: June 2010

Abstract: Objective: To further validate an in-human model of psychosis and antipsychotic drug action. Methods: Intrapulmonary inhalations of purified tetrahydrocannabinol (THC) in three subsequent dosages of 2, 4 and 6 mg were used to induce psychomimetic symptoms in healthy, male, mild cannabis users. These symptoms were measured using the positive subscale of the Positive and Negative Syndrome Scale (PANSS), based on a structured clinical interview, and visual analogue scales (VAS) as described by Bowdle. A single oral dose of olanzapine 10 mg, or diphenhydramine 2x15 mg (as positive control), or matching placebo was administered before inhalations of THC or placebo in a randomized, five-way cross-over design. (Figure Presented) Results: 49 subjects were included in the study, of whom 39 showed an increase on the positive PANSS following THC administration. THC alone induced an increase on the positive PANSS by 25.1% (95%CI 16.6–34.1; p < 0.0001). Co-administration of olanzapine reduced this increase by 9.5% (95%CI 3.1–15.5; p=0.0046), whereas diphenhydramine had no effect. VAS feeling high showed an increase of 0.695 log(mm) (95% CI 0.554–0.836; p<0.0001), which was inhibited by olanzapine by 0.161 log(mm) (95%CI 0.024–0.298; p=0.0218). Conclusion: THC induces transient psychotomimetic effects in healthy volunteers, as measured on the positive subscale of the PANSS. This effect could be reduced by co-administration of olanzapine. The inhibitory effect on feeling high seems less strong. It seems as if the effect of THC on cannabinoid CB1 receptors can be influenced by the activity of olanzapine on serotonin 5-HT2 and dopamine D2 receptors. This model could be used to test the effect of novel drugs in development for psychosis in an early clinical phase of drug development.

Source: EMBASE

22. Psychopathological symptoms and neurocognitive tests performance in polish 1200 schizophrenic patients treated with olanzapine (Olzapin)

Author(s) Borkowska A., Blinski M., Bucinski A., Kacprzyk P., Drozdz W.

Citation: Biological Psychiatry, May 2010, vol./is. 67/9 SUPPL. 1(80S), 0006-3223 (01 May
**Abstract:** Background: Cognitive dysfunctions in schizophrenia show an association with negative symptoms and brain abnormalities. Studies on correlations between psychometric ratings and cognitive functions in schizophrenic patients revealed inconsistent results and therefore assessment in both cross-sectional and longitudinal surveys has been suggested. The aim of the study was to assess the correlation between PANSS scale and performance on cognitive tests in a cohort of schizophrenic patients treated with olanzapine (Olzapin) for at least one month. Methods: Patients with the ICD-10 diagnosis of schizophrenia treated with efficacious dose of olanzapine were enrolled in 38 centres in Poland. Neuropsychological testing was based on short computerized battery which consisted of five tasks: simple reaction time test (SRT), Verbal Memory Test (VM), GoNoGo test (GNG), Stroop Test (STR) and Visual Working Memory Test (VWM). On the same day an assessment of psychopathology with the PANSS was performed. Advanced multidimensional data analysis, (i.e. Principal Component Analysis and Artificial Neural Networks methods), were used in statistical analysis. Results: Overall 1200 patients aged 18-53 (mean 43,7) years were enrolled. Duration of the illness was 1-15 years (mean 8+/-6). The average dose of olanzapine was 10,5+/-6 (5-25) mg/day. Mean intensity of PANSS total was 70+/-17. Reaction time, visual memory, GoNoGo test and Stroop test performance were associated with intensity of both positive and negative symptoms on PANSS scale. Conclusions: Most cognitive measures were correlated with the intensity of negative symptoms on PANSS scale, and this indicates strong association between negative symptoms and cognitive impairment in the majority of domains.

**Source:** EMBASE
Publication Date: April 2010

Abstract: Background: Mood stabilizing properties including anti-depressive effects have been disclosed for some of the second generation antipsychotics (SGAs). We have previously demonstrated differential antipsychotic effectiveness among olanzapine, quetiapine, risperidone, and ziprasidone. The main aim of this rater-blind, randomized, naturalistic study was to determine whether differences exist among these first-line SGAs regarding relief of symptoms related to depression and suicidal ideation. Methods: Patients (age <sup>3</sup> 18 years) acutely admitted with psychosis to a single-site hospital were eligible for the study. The hospital is responsible for all the acute admissions in the catchment area of about 400000 inhabitants, thus supplying information from a diverse population representing everyday clinical practice. The patients were randomized to risperidone, olanzapine, quetiapine, and ziprasidone, and tested at intervals for up to 2 years. The main outcome measures were change of the scores of item G6 (Depression) in the Positive and Negative Syndrome Scale (PANSS), general psychopathology subscale; and of the 9 items Depression, hopelessness, self depreciation, guilty ideas of reference, pathological guilt, morning depression, early wakening, suicide, observed depression, in the Calgary Depression Scale for Schizophrenia (CDSS); as well as the use of concomitant mood stabilizers, antidepressants, and benzodiazepines. Results: A total of 226 men (67.3%) and women (32.7%) were included. At baseline mean age with standard deviation (SD) was 34.1 (13.5) years and 44.2% were antipsychotic drug naive. The distribution of ICD-10 diagnoses were: schizophrenia and related disorders 44.5%; acute and transient psychotic disorder 21.3%; drug-induced psychotic disorder 13.4%; mood disorders with psychotic symptoms 10.6%; non-organic unspecified psychotic disorder 5.1%; miscellaneous psychotic disorders 5.1%. Baseline CDSS total score with SD was 6.5 (5.3) and PANSS G6 score was 3.2 (1.6). Baseline PANSS total score with SD was 74.0 (13.4), the Clinical Global Impression - Severity of Illness scale score was 5.2 (0.6), and the Global Assessment of Functioning scale - Split Version, Functions scale score was 30.7 (6.0). Mean doses in milligrams with SD for the antipsychotics were for olanzapine 14.5 (5.0); quetiapine 339.3 (193.4); risperidone 3.3 (1.1); and ziprasidone 100.3 (42.2). There were no substantial differences among the SGAs with regards to change of any depression item in the rating scales. There were no substantial differences among the SGAs regarding concomitant use of mood stabilizers, antidepressants, or benzodiazepines. Discussion: Differential effectiveness among the SGAs against symptoms of depression was not disclosed in this heterogeneous sample. The results do not support preference of any particular agent among the SGAs under investigation for targeting symptoms of depression in a patient acutely admitted with psychosis.

Source: EMBASE

25. [Action of amygdala dopamine and effect of antipsychotics].

Author(s) Oshibuchi H, Inada K, Ishigooka J

Citation: Nihon Shinkei Seishin Yakurigaku Zasshi, April 2010, vol./is. 30/2(93-9), 1340-2544;1340-2544 (2010 Apr)

Publication Date: April 2010

Abstract: Although emotional dysfunction in patients with schizophrenia is thought to be associated with poorer outcomes in terms of overall quality of well-being, only a few basic studies have been done on the biochemical effect of antipsychotics on the fear response of a neurotransmitter (i.e. dopamine). To examine the reaction to emotional stress, conditioned fear stress has been developed as a form of psychological stress based on classical conditioning theory. Using this model, the amygdala was found to be one of the most potent modulators of the mechanisms responsible for the emotional memory system. Methamphetamine-induced sensitization (reverse tolerance phenomenon) in rats has been widely and successfully used as an animal model of stimulant-induced psychosis and schizophrenia in terms of the paranoid psychotic state and its vulnerability to relapse. Methamphetamine-sensitized animals show significantly higher extracellular dopamine release in the amygdala than unsensitized rats after exposure to a conditioned stimulus. This hypersensitivity of dopamine release is considered to be a biochemical marker of hypersensitivity and vulnerability to stress in psychosis. Aripiprazole and haloperidol equally suppressed the marked increase in extracellular dopamine levels in fear-conditioned rats, whereas haloperidol increased and aripiprazole decreased basal
dopamine levels. The effect of antipsychotics attenuates the dopamine fear response in the amygdala, modulating basal dopamine level.

Source: Medline

26. Drugs associated with hemorrhagic pancreatitis in the food and drug administration adverse event database and mitochondrial toxicity

Author(s) Burkhart K.K., Szafrman A., Lyndly J.

Citation: Clinical Toxicology, March 2010, vol./is. 48/3(295-296), 1556-3650 (March 2010)

Publication Date: March 2010

Abstract: Objective: To identify drugs with a disproportional high postmarketing reporting of hemorrhagic pancreatitis (Table Presented) and evaluate the mechanisms of action that may be involved. Methods: The FDA's Adverse Event Reporting System (AERS) was searched for the drugs that had the highest adjusted disproportionality reporting ratio for the preferred MedDRA term, hemorrhagic pancreatitis. We identified these signals by using the Multi-item Gamma Poisson Shrinker (MGPS) statistical algorithm that applies a Bayesian model to simultaneously analyze disproportionality of reporting ratios for each event in the huge AERS database (including hemorrhagic pancreatitis) relative to all other events in the whole database. To help reduce false positives, MGPS systematically "shrinks" unstable observed/expected ratios and adjusts for background differences in relative reporting rates by using stratification. The final score is the Empirical Bayesian Geometric Mean (EBGM) score. We found that the list of drugs generated by the algorithm included drugs known to cause mitochondrial toxicity. A PubMed search was performed on each drug to determine effects on mitochondrial function. Results: MGPS generated the following top EBGM scores and number of reports (EBGM, N) for the following drugs: asparaginase (25.1, 16), valproic acid (19.2, 88), stavudine (18.2, 30), pegaspargase (12.5, 5), didanosine (11.2, 19), pentamidine (10.5, 6), fluphenazine (7.1, 7), lamivudine (5.8, 18), efavirenz (5.3, 12), nelfinavir 4.7, 8), prednisolone (4.4, 10), drotrecogin-alpha (4.2, 5), olanzapine (3.9, 20) and fenofibrate (3.7, 5). The drugs inhibit a number of mitochondrial complexes. Impaired mitochondrial complexes included asparaginase (II), valproate (II), stavudine (I, II, IV), fluphenazine (I), furosemide (II, III), prednisolone (IV, V), olanzapine (II) and fenofibrate (I, II, III, IV). Other actions of the drugs included uncoupling of oxidative phosphorylation (pentamidine) and inhibition of electron transport (furosemide, prednisolone). Conclusion: Most drugs that are associated with a high adjusted relative postmarketing reporting ratio for hemorrhagic pancreatitis appear to impair mitochondrial function and/or electron transport. Further research into the potential role of mitochondrial toxicity and hemorrhagic pancreatitis appears warranted. Likewise, antidotes such as L-carnitine that improve mitochondrial oxidation may warrant further study. Analysis of the AERS database can be used to generate hypotheses about the mechanisms for drug-induced toxicity.

Source: EMBASE

Available in fulltext from Clinical Toxicology at EBSCOhost

27. Hyperglycemia and antipsychotic treatment in patients on methadone maintenance

Author(s) Barbadillo L., Zapirain E., Lopez A., Usoz E., Gil A.V., Calvo A.

Citation: European Neuropsychopharmacology, 2009, vol./is. 19/(S554-S555), 0924-977X (2009)

Publication Date: 2009

Abstract: The American Psychiatric Association set a new standard of care by collaborating with the American Diabetes Association and others in recommending how to manage the potential for increased risk of obesity, diabetes, and lipid disorders when using atypical antipsychotics. The prevalence of hyperglycemia and/or diabetes seems to be greater in schizophrenic patients than in the general population. This has been associated with factors such as the existence of harmful hygienic-diet habits and the adverse effects of anti-psychotic treatment [1]. Some studies have published that prolonged treatment with
2nd generation or atypical antipsychotic can lead to weight gain and alteration of glucose and lipids in the metabolism. They have also described that Olanzapine and Clozapine are the anti-psychotic drugs with the greatest risk of causing diabetes. The increasing incidence of diabetes in the population makes it difficult to assess the relationship between atypical antipsychotic use and blood glucose abnormalities. Moreover, the risk of diabetes may be elevated in patients with schizophrenia, whether or not they are receiving medications. Diabetes and disturbed carbohydrate metabolism may be an integral component of schizophrenia itself [2]. A direct relationship is also suggested with the duration of this treatment used chronically to treat patients with psychotic disorders.

Objectives: Determine the prevalence of hyperglycemia in patients with dual diagnosis of psychosis and dependency on opiates, in continual treatment antipsychotic drugs and methadone maintenance. Determine the incidence of diabetes mellitus, after introducing antipsychotic treatment. Determine whether there is correlation between continued treatment time with antipsychotic drugs and hyperglycemia. Method: Transverse study including patients on a maintenance programme with methadone, with dual diagnosis of schizophrenia, schiz-o-emotional disorders and psychotic disorder induced by substances plus dependency on opiates. All of them, followed treatment with antipsychotic. Blood glucose values were collected by means of blood test and hyperglycemia was considered to be >=110mg/dl on an empty stomach, and diabetes for those who presented blood glucose figures >=126mg/dl on an empty stomach on two consecutive occasions or symptoms of diabetes with casual blood glucose >=200 mg/dl. Results: 19 patients were included; with an average age of 40.2 years age drug dependency and 4.5 (SD 2.8) in treatment with antipsychotic. Three patients (15.8%) had presented hyperglycemia during the treatment period; of these, two with olanzapine and one with olanzapine and aripiprazole, although no statistically significant relationship was relating to the type of treatment (p = 0.411). During the treatment two patients developed diabetes, corresponding to 10.5% of the sample, both treated with olanzapine, one in monotherapy and the other one combined with aripiprazole, no significant association could be demonstrated (p = 0.292). No linear relationship was found between the blood glucose values and the time measured in years of antipsychotic treatment (p = 0.215).

Conclusions: Approximately one in seven patients treated with antipsychotic and methadone develop hyperglycemia as a treatment side effect. The incidence of diabetes in patients treated with antipsychotic as approximately one in ten. A correlation has not been found between blood glucose values and increase in treatment time for antipsychotic.

Source: EMBASE

28. What can we learn from evidence based medicine studies with clozapine and quetiapine for psychosis in PD

Author(s) Rabey J.M.

Citation: European Journal of Neurology, 2009, vol./is. 16/S3(625), 1351-5101 (2009)

Publication Date: 2009

Abstract: Parkinson's disease (PD) is a multisystem disease including not only motor symptoms but also many non motor as autonomic, cognitive and psychiatric. Psychiatric symptoms include depressive, compulsive and psychotic features. Psychotic symptomatology such as hallucinations and delusions are a predictor of nursing home placement and are related to mortality (Fenelon, et al, Brain 2000). In contrast to motor symptoms treatment options for non-motor symptoms are still very limited (Chaudhuri et al, Lancet Neurology, 2006). Concerning the treatment of psychosis, classical antipsychotics have the capability to worsen motor symptomatology (by blocking D2 receptors in the striatum) and it was then logical that neurologists tried to utilize new atypical antipsychotics (clozapine, risperidone, olanzapine, ondansetron, quetiapine) which were introduced to the clinics as lacking the capability to produce parkinsonism. However very soon it became clear that some of them (risperidone, olanzapine, ondansetron) can also induce a detrimental effect on the motor system (Goetz et al, Neurology 2000; Ellis et al, Clin Neuroscti 2000, etc). In a review of the atypical antipsychotic agents used for the management of drug-induced psychosis in PD the only drug with confirmed benefits (3 randomized clinical studies (RCT); NEJM 1999; Lancet 1999; Neurology 2000) without worsening PD was clozapine (Friedman and Factor, Mov Dis,2000). On the other hand due to the concern of potential bone marrow inhibition induced by clozapine, many neurologists prescribe quetiapine, another atypical antipsychotic, which although has been proved
without efficacy in 3 RCT (Ondo et al, Mov Dis 2005, Rabey et al, Mov Dis 2007, Kurland et al, Mov Dis 2008) is still widely used in clinical practice. We hope that new drugs (like pimavanserin) will soon be available for the treatment of psychosis in PD.

Source: EMBASE
Available in fulltext from European Journal of Neurology at EBSCOhost

29. Aripiprazole for psychosis-induced aggression or agitation

Author(s) Pagadala B., Jayaram M.B., Mitra L.
Citation: Cochrane Database of Systematic Reviews, 2009, vol./is. /4, 1469-493X (2009)
Publication Date: 2009
Source: EMBASE
Available in fulltext from Cochrane Library, The at Wiley

30. Treatment for amphetamine psychosis

Author(s) Shoptaw S.J., Kao U., Ling W.
Citation: Cochrane Database of Systematic Reviews, 2009, vol./is. /1, 1469-493X (2009)
Publication Date: 2009

Abstract: Background: Chronic amphetamine users may have experience of paranoia and hallucination. It has long been believed that dopamine antagonists, such as chlorpromazine, haloperidol, and thioridazine, are effective for the treatment of amphetamine psychosis. Objectives: To evaluate risks, benefits, costs of treatments for amphetamine psychosis. Search strategy: MEDLINE (1966-2007), EMBASE (1980-2007), CINAHL (1982-2007), PsychINFO (1806-2007), CENTRAL (Cochrane Library 2008 issue 1), references of obtained articles. Selection criteria: All randomised controlled and clinical trials (RCTs, CCTs) evaluating treatments (alone or combined) for people with amphetamine psychosis Data collection and analysis: Two authors evaluated and extracted the data independently. Dichotomous data were extracted on an intention-to-treat basis in which the dropouts were assigned as participants with the worst outcomes. The Relative Risk (RR) with the 95% confidence interval (95% CI) was used to assess the dichotomous data. The Weighted Mean Difference (WMD) with 95% CI was used to assess the continuous data. Main results: The comprehensive searches found one randomised controlled trial of treatment for amphetamine psychosis meeting the criteria for considering studies. The study involved 58 participants and compared the efficacy and tolerability of two antipsychotic drugs, olanzapine (a newer antipsychotic) and haloperidol (a commonly used antipsychotic medication used as a control condition), in treating amphetamine-induced psychosis. The results show that both olanzapine and haloperidol at clinically relevant doses were efficacious in resolving psychotic symptoms, with the olanzapine condition showing significantly greater safety and tolerability than the haloperidol control as measured by frequency and severity of extrapyramidal symptoms. Authors' conclusions: Only one RCT of treatment for amphetamine psychosis has been published. Outcomes from this trial indicate that antipsychotic medications effectively reduce symptoms of amphetamine psychosis, the newer generation and more expensive antipsychotic medication, olanzapine, demonstrates significantly better tolerability than the more affordable and commonly used medication, haloperidol. There are other two studies that did not meet the inclusion criteria for this review. The results of these two studies show that agitation and some psychotic symptoms may be abated within an hour after antipsychotic injection. Whether this limited evidence can be applied for amphetamine psychotic patients is not yet known. The medications that should be further investigate are conventional antipsychotics, newer antipsychotics and benzodiazepines. However, naturalistic studies of amphetamine psychotic symptoms and the prevalence of relapse to psychosis in the presence of amphetamine, are also crucial for advising the development of study designs appropriate for further treatment studies of amphetamine psychosis. 2009 The Cochrane Collaboration. Published by John Wiley & Sons, Ltd.

Source: EMBASE
31. Aripiprazole ameliorates phencyclidine-induced impairment of recognition memory through dopamine D1 and serotonin 5-HT1A receptors

Author(s): Nagai T., Murai R., Matsui K., Kamei H., Noda Y., Furukawa H., Nabeshima T.

Citation: Psychopharmacology, January 2009, vol./is. 202/1-3(315-328), 0033-3158;1432-2072 (January 2009)

Publication Date: January 2009

Abstract: Rationale: Cognitive deficits, including memory impairment, are regarded as a core feature of schizophrenia. Aripiprazole, an atypical antipsychotic drug, has been shown to improve disruption of prepulse inhibition and social interaction in an animal model of schizophrenia induced by phencyclidine (PCP); however, the effects of aripiprazole on recognition memory remain to be investigated. Objectives: In this study, we examined the effect of aripiprazole on cognitive impairment in mice treated with PCP repeatedly. Materials and methods: Mice were repeatedly administered PCP at a dose of 10mg/kg for 14 days, and their cognitive function was assessed using a novel-object recognition task. We investigated the therapeutic effects of aripiprazole (0.01-1.0mg/kg) and haloperidol (0.3 and 1.0mg/kg) on cognitive impairment in mice treated with PCP repeatedly. Results: Single (1.0mg/kg) and repeated (0.03 and 0.1mg/kg, for 7days) treatment with aripiprazole ameliorated PCP-induced impairment of recognition memory, although single treatment significantly decreased the total exploration time during the training session. In contrast, both single and repeated treatment with haloperidol (0.3 and 1.0mg/kg) failed to attenuate PCP-induced cognitive impairment. The ameliorating effect of aripiprazole on recognition memory in PCP-treated mice was blocked by co-treatment with a dopamine D1 receptor antagonist, SCH23390, and a serotonin 5-HT1A receptor antagonist, WAY100635; however, co-treatment with a D2 receptor antagonist raclopride had no effect on the ameliorating effect of aripiprazole. Conclusions: These results suggest that the ameliorative effect of aripiprazole on PCP-induced memory impairment is associated with dopamine D1 and serotonin 5-HT1A receptors. 2008 Springer-Verlag.

Source: EMBASE

Available in fulltext from Psychopharmacology at EBSCOhost

32. Is aripiprazole the only choice of treatment of the patients who developed antipsychotic agents-induced leucopenia and neutropenia? A case report.

Author(s): Yalcin, Demet Ozen, Goka, Erol, Aydemir, M. Cigdem, Kisa, Cebrail

Citation: Journal of Psychopharmacology, May 2008, vol./is. 22/3(333-335), 0269-8811;1461-7285 (May 2008)

Publication Date: May 2008

Abstract: Leucopenia and neutropenia could be side effects of anti-psychotic drugs, especially clozapine. However, there is evidence that other anti-psychotics can cause leucopenia and neutropenia. We present the clinical follow-up and treatment process of a patient, who had initially developed quetiapine and amisulpride related neutropenia, but not with aripiprazole. (PsycINFO Database Record (c) 2012 APA, all rights reserved) (journal abstract)

Source: PsycINFO

33. Olanzapine and risperidone block a high dose of methamphetamine-induced schizophrenia-like behavioral abnormalities and accompanied apoptosis in the medial prefrontal cortex.

Author(s): Abekawa, Tomohiro, Ito, Koki, Nakagawa, Shin, Nakato, Yasuya, Koyama, Tsukasa

Citation: Schizophrenia Research, April 2008, vol./is. 101/1-3(84-94), 0920-9964 (Apr
Abstract: This study aims to propose a comprehensive new model for schizophrenia, which shows PPI disruption at baseline state as an endophenotype, the development of cross-sensitization to an NMDA receptor antagonist, MK-801 as a clinical phenotype of the progression into treatment-resistance, and accompanied induction of apoptosis in the medial prefrontal cortex as a critical possibility during the progression. Repeated administration of a high dose of methamphetamine (METH) (2.5 mg/kg), which could increase glutamate levels in the medial prefrontal cortex (mPFC), induced TUNEL-positive cells in this region, accompanied development of behavioral cross-sensitization to MK-801 in response to a challenge injection of MK-801, and PPI disruption at baseline state without a challenge injection. Olanzapine (OLZ) (1.0 mg/kg) and risperidone (RIS) (0.1 mg/kg), which inhibited and remarkably attenuated METH (2.5 mg/kg)-induced increases in glutamate levels, respectively, blocked not only the induction of TUNEL-positive cells in the mPFC but also the accompanied development of above behavioral abnormalities. These findings suggest that repeating the METH-induced glutamate release produces behavioral abnormalities as a clinical phenotype of schizophrenia, accompanied apoptosis as a critical possibility during the progression, and suggest that sufficient dose of olanzapine and risperidone can block the development of these behavioral abnormalities and accompanied apoptosis during the progression. (PsycINFO Database Record (c) 2012 APA, all rights reserved) (journal abstract)
schizophrenia, while its effects on glutamate-based paradigms have remained to be further characterized. Due to its unique mechanism of action, aripiprazole has also been considered as a replacement medication for psychostimulant abuse. Thus, in the present study we tested the hypothesis that aripiprazole would prevent the motor hyperactivity induced by psychostimulant and psychotomimetic drugs that act either by dopaminergic or glutamatergic mechanisms. Male Swiss mice received injections of aripiprazole (0.1-1 mg/kg) followed by drugs that enhance the dopamine-mediated neurotransmission, amphetamine (3 mg/kg) or cocaine (5 mg/kg), or by glutamate NMDA-receptor antagonists, ketamine (60 mg/kg) or MK-801 (0.4 mg/kg). Independent groups also received aripiprazole (0.1-1 mg/kg) or haloperidol (0.5 mg/kg) and were tested for catalepsy. All doses of aripiprazole were effective in preventing the motor stimulant effects of amphetamine and cocaine. Moreover, the higher dose also prevented the effects of ketamine and MK-801. The present study reports the effects of aripiprazole in dopaminergic and glutamatergic models predictive of antipsychotic activity, suggesting that both may be useful for screening novel partial agonists with antipsychotic activity. It also shows that aripiprazole may prevent the acute effects of psychostimulant drugs without significant motor impairment.

Source: Medline


Author(s) Curtis, L, Bartolomei, J, Merlo, M. C. G

Citation: Schweizer Archiv fur Neurologie und Psychiatrie, January 2008, vol./is. 159/1(7-15), 0258-7661 (Jan 2008)

Publication Date: January 2008

Abstract: Today's psychotic patient is more and more at risk from metabolic syndrome. This syndrome, whose core criteria were standardised by expert consensus in 2004, represents a significant cause of increased morbidity and mortality in such a psychiatric population. The constellation of risk factors of metabolic syndrome associates central obesity with at least two of the following: hyperlipidaemia, hypercholesterolaemia, arterial hypertension or fasting hyperglycaemia. The reasons why psychotic patients are more at risk for these complications are still debated although one can reasonably suggest a multifactorial aetiology. Among the causes which seem to contribute significantly and moreover which the clinician may be able to have a positive influence over are antipsychotic medication and patient lifestyle. Since the introduction of second-generation antipsychotics complications of the metabolic syndrome have increasingly drawn clinicians' attention. Based on current guidelines, we propose a classification of risk for metabolic syndrome according to specific medication: the high-risk group consists of clozapine and olanzapine, the medium-risk group of risperidone,quetiapine and amisulpride, the low-risk group of aripiprazole and sertindole. The mechanisms through which antipsychotic medication induces metabolic-syndrome symptoms are now actively researched. A probable hypothesis implicates deregulation of energy homeostasis in the central nervous system (essentially at the hypothalamic level). This deregulation would be exerted through specific neurotransmitter systems with which medication interferes. At the moment, most data support the relevance of interaction at the histaminergic and serotoninergic receptors. However, other hypotheses supported by recent research suggest a significant medication interaction at the peripheral level, such as the pancreas or adipose tissue. For the clinician the priority remains how to use today's knowledge to best prevent and treat metabolic syndrome in his or her patients. We propose a therapeutic attitude intervening at several levels with early detection of possible signs, judicious adaptation of antipsychotic medication, lifestyle intervention and, if necessary, specific treatment of metabolic syndrome symptoms. (PsycINFO Database Record (c) 2012 APA, all rights reserved) (journal abstract)

Source: PsycINFO

37. Olanzapine monotherapy for late-onset vocal tics in a schizophrenic patient.

Author(s) Cheng, Wan-Ju, Liu, Hsing-Cheng, Huang, Ming Chyi

Citation: Psychiatry and Clinical Neurosciences, December 2007, vol./is. 61/6(700-701),
Abstract: Olanzapine is an atypical antipsychotic with diverse receptor activities. Its greater affinity for serotonin 5HT2A receptors over dopamine D2 receptors accounts for the lower risk of extrapyramidal symptoms. Tic can be observed in transient tic disorder, Tourette's disorder, or medication-induced tic disorder, which could be a variant expression of tardive dyskinesia. Dearrangement of the dopaminergic pathway, including dopamine excess or super-sensitivity of post-synaptic dopaminergic receptors, is the main neurotransmitter abnormality of tic. The present report shows the advantage of olanzapine monotherapy in treating 48-year-old woman with late-onset simple vocal tics in a chronic schizophrenia patient following withdrawal of long-term antipsychotic treatment. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO
Available in fulltext from Psychiatry and Clinical Neurosciences at EBSCOhost

38. Aripiprazole-induced psychosis: a case report of reexposure by stepwise up-titration.
Author(s) Kapusta ND, Mossaheb N, Barnas C, Fischer P
Citation: Journal of Clinical Psychiatry, September 2007, vol./is. 68/9(1445-6), 0160-6689;1555-2101 (2007 Sep)
Publication Date: September 2007
Source: Medline
Available in print at Grantham Hospital Staff Library

Author(s) Kapusta N.D., Mossaheb N., Barnas C., Fischer P.
Citation: Journal of Clinical Psychiatry, September 2007, vol./is. 68/9(1445-1446), 0160-6689 (September 2007)
Publication Date: September 2007
Source: EMBASE
Available in print at Grantham Hospital Staff Library

40. Metabolic side effects of antipsychotic medication
Author(s) Tschoner A., Engl J., Laimer M., Kaser S., Rettenbacher M., Fleischhaeker W.W., Patsch J.R., Ebenbichler C.F.
Citation: International Journal of Clinical Practice, August 2007, vol./is. 61/8(1356-1370), 1368-5031;1742-1241 (August 2007)
Publication Date: August 2007
Abstract: The use of second-generation antipsychotics (SGAs) is associated with metabolic side effects including weight gain, diabetes mellitus and an atherogenic lipid profile. These adverse effects are not only the risk factors for cardiovascular disease, insulin resistance and diabetes mellitus leading to increased morbidity and mortality but may also impair the patient's adherence to treatment. SGAs in particular are associated with significant weight gain with clozapine and olanzapine carrying the highest risk, whereas newer agents, such as risperidone and aripiprazole, are considered to be less prone to cause weight gain. Consequently, a consensus development conference convened issuing recommendations on patient monitoring when treated with SGAs. The metabolic effects of antipsychotic drugs should be of concern when planning a patient's treatment strategy. Baseline screening and regular follow-up monitoring whose intervals should depend on the individual predisposition are advised. Possible therapeutical
strategies for the management of drug-induced obesity include therapeutic approaches, such as lifestyle change and pharmaceutical intervention. Drugs with a weight reducing effect become more important because of the lack of compliance with behavioural intervention. Topiramate, histamine-antagonists, dopaminergic- and serotonergic agents have shown positive results in the management of psychotropic medication induced weight gain. However, further trials are required to support a specific therapeutical approach as well as studies to investigate the underlying mechanisms for future drug development. 2007 The Author.

Source: EMBASE
Available in fulltext from International Journal of Clinical Practice at EBSCOhost

41. Psychosis with sibutramine.

Author(s) Rosenbohm, Angela, Bux, Christoph J, Connemann, Bernhard J
Citation: Journal of Clinical Psychopharmacology, June 2007, vol./is. 27/3(315-317), 0271-0749;1533-712X (Jun 2007)
Publication Date: June 2007
Abstract: Sibutramine (l-(4-chlorophenyl)-N,N-dimethyl-a-(2-methylpropyl)-cyclobutanemethanamine (Meridia and Reductil), a norepinephrine and serotonin reuptake inhibitor originally developed as an antidepressant, has met with considerable success in the treatment of obesity. Typical adverse effects are dry mouth, constipation, tachycardia, hypertension, and headache. Regarding the central nervous system, anxiety, depression, and somnolence are well-known side effects, whereas delusions or hallucinations have been reported only once, without fully establishing causality. We report 2 additional cases of psychosis apparently induced by sibutramine. A 48-year-old German prison officer was admitted because of imperative voices telling him to shoot his superior. He reported hearing these voices during the past 3 months and also lately receiving signs and reading thoughts. The patient was treated with sertindole 16 mg OD; full recovery was achieved within 3 weeks. A 43-year-old Thai woman was admitted after setting her furniture on fire, because it appeared "dark," and attacking her husband fiercely, biting and scratching him when he tried to rescue her from the fire. The patient was treated with olanzapine 15 mg OD and recovered completely within 4 weeks. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

42. The atypical antipsychotic, aripiprazole, blocks phencyclidine-induced disruption of prepulse inhibition in mice.

Author(s) Fejgin, Kim, Safonov, Sergej, Palsson, Erik, Wass, Caroline, Engel, Jorgen A, Svensson, Lennart, Klammer, Daniel
Citation: Psychopharmacology, April 2007, vol./is. 191/2(377-385), 0033-3158;1432-2072 (Apr 2007)
Publication Date: April 2007
Abstract: Rationale: The psychotomimetic drug, phencyclidine, induces schizophrenia-like behavioural changes in both humans and animals. Phencyclidine-induced disruption of sensory motor gating mechanisms, as assessed by prepulse inhibition of the acoustic startle, is widely used in research animals as a screening model for antipsychotic properties in general and may predict effects on negative and cognitive deficits in particular. Dopamine (DA) stabilizers comprise a new generation of antipsychotics characterized by a partial DA receptor agonist or antagonist action and have been suggested to have a more favourable clinical profile. Objective: The aim of the present study was to investigate the ability of first, second and third generation antipsychotics to interfere with the disruptive effect of phencyclidine on prepulse inhibition in mice. Results: Aripiprazole blocked the phencyclidine-induced disruption of prepulse inhibition. The atypical antipsychotic clozapine was less effective, whereas olanzapine, and the typical antipsychotic haloperidol, failed to alter the effects of phencyclidine on prepulse inhibition. Conclusions: The somewhat superior efficacy of clozapine compared to haloperidol may be explained by its lower affinity.
and faster dissociation rate for DA D2 receptors possibly combined with an interaction with other receptor systems. Aripiprazole was found to be more effective than clozapine or olanzapine, which may be explained by a partial agonist activity of aripiprazole at DA D2 receptors. In conclusion, the present findings suggest that partial DA agonism leading to DA stabilizing properties may have favourable effects on sensorimotor gating and thus tentatively on cognitive dysfunctions in schizophrenia. (PsycINFO Database Record (c) 2012 APA, all rights reserved) (journal abstract)

Source: PsycINFO
Available in fulltext from Psychopharmacology at EBSCOhost

43. Treating dopamimetic psychosis in Parkinson’s disease: structured review and meta-analysis.

Author(s) Frieling H, Hillemacher T, Ziegenbein M, Neundorfer B, Bleich S
Citation: European Neuropsychopharmacology, February 2007, vol./is. 17/3(165-71), 0924-977X;0924-977X (2007 Feb)
Publication Date: February 2007
Abstract: Psychosis due to dopamimetic treatment is a difficult problem in patients with Parkinson’s disease (PD). The aim of this structured review with meta-analysis was to evaluate which neuroleptic drugs can efficiently be used to treat drug-induced psychosis (DIP) in Parkinson’s disease. Electronic databases were screened for the key words Parkinson's disease and psychosis. Only 7 trials with a satisfactory allocation concealment and data reporting were included into the study. Two trials compared low-dose clozapine versus placebo with a significantly better outcome for clozapine regarding efficacy and motor functioning. In one trial clozapine was compared against quetiapine showing equivalent efficacy and tolerability. However, in two placebo controlled trials quetiapine failed to show efficacy. In two further placebo controlled trials olanzapine did not improve psychotic symptoms and significantly caused more extrapyramidal side effects. Based on randomized trial-derived evidence which is currently available, only clozapine can be fully recommended for the treatment of DIP in PD. Olanzapine should not be used in this indication.

Source: Medline

44. Interferon--induced persistent psychosis.

Author(s) Ginsberg, David L
Citation: Primary Psychiatry, October 2006, vol./is. 13/10(26-27), 1082-6319 (Oct 2006)
Publication Date: October 2006
Abstract: Reports a case in which a patient presents with persistent and refractory psychosis after treatment with interferon (IFN)--. A 50-year-old white male was brought to the emergency room by a family member who had become concerned by his increasing paranoia and bizarre behavior. His psychiatric history was significant for a 25-year history of narcotic dependence, two brief admissions to substance-dependence treatment facilities, and a remote suicide attempt. The patient was treated with olanzapine and showed substantial, albeit incomplete, remission of his symptoms. However, shortly thereafter the patient became nonadherent with treatment and experienced a worsening psychosis that necessitated readmission. Olanzapine was increased to 25 mg/day. One more month of inpatient treatment resulted in a decrease in auditory hallucinations, but there was no reduction in the persecutory delusions. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

45. Psychotic disorder induced by oxybutynin: Presentation of two cases.

Author(s) Gulsun M, Pinar M, Sabanci U
Citation: Clinical Drug Investigation, 2006, vol./is. 26/10(603-6), 1173-2563;1173-2563
Abstract: Anticholinergic agents are muscarinic receptor antagonists that suppress the activity of the acetylcholine system in the brain. Some of these agents also increase the concentration of dopamine in the synaptic cleft, which may result in psychotic symptoms. Oxybutynin is an antimuscarinic drug that may have adverse effects on the CNS, including memory impairment, confusion, delirium and hallucinations in elderly patients. To date, several case reports have been published about the association between oxybutynin and psychotic symptoms in elderly subjects, but we were unable to find any case reports describing oxybutynin-induced psychotic disorders in young people. Here we report on two patients, a 7-year-old boy and a 21-year-old man, who developed a brief psychotic disorder that may have been caused by oxybutynin. The first patient was kept under observation with vital functions supported but no medication. All his psychotic symptoms regressed and his general condition improved. The second patient was treated with olanzapine 10 mg/day. His psychotic symptoms resolved within 3 weeks. Our two case reports provide evidence that oxybutynin may induce psychotic disorders, and in younger patients.

Source: Medline

46. Atypical case of neuroleptic malignant syndrome caused by olanzapine and carbamazepine.

Author(s) Woo, Benjamin K. P, Obrocea, Gabriela V
Citation: Psychiatry, December 2005, vol./is. 2/12(23-24), 1550-5952 (Dec 2005)
Publication Date: December 2005

Abstract: We report a case of NMS occurrence when carbarnazepine was added to an atypical neuroleptic, olanzapine. Ms. E, a 31-year-old woman with bipolar disorder, presented to our hospital with consciousness disturbance, increasing persecutor delusions, and depressive symptoms. Poly substance induced rhabdomyolysis might have occurred. The addition of carbamzepine might have modified the presentation of NMS as the patient was a febrile. Lastly, our case reiterates the finding that decreased serum iron level is associated with neuroleptic malignant syndrome. In this case, the serum iron level was back to normal after the episode resolved. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO
Available in fulltext from Psychiatry at National Library of Medicine

47. Aripiprazole-induced psychosis

Author(s) Ginsberg D.L.
Citation: Primary Psychiatry, December 2005, vol./is. 12/12(30), 1082-6319 (December 2005)
Publication Date: December 2005
Source: EMBASE

48. Pathological gambling associated with drug therapy? Blood dyscrasias associated with thiazolidinediones olanzapine can cause metabolic problems if used in mentally healthy patients flecainide-induced neuropathy acute psychosis with an OTC cold medicine topiramate-induced psychosis SIADH - caused by SSRI or nicotine? Fentanyl patch advisory

Author(s) Shuster J.
Citation: Hospital Pharmacy, November 2005, vol./is. 40/11(946-950+1016), 0018-5787 (November 2005)
Publication Date: November 2005
49. **Drug-induced psychosis in Parkinson disease: Phenomenology and correlations among psychosis rating instruments.**

**Author(s):** Chou, Kelvin L, Messing, Susan, Oakes, David, Feldman, Peter D, Breier, Alan, Friedman, Joseph H

**Citation:** Clinical Neuropharmacology, September 2005, vol./is. 28/5(215-219), 0362-5664;1537-162X (Sep-Oct 2005)

**Publication Date:** September 2005

**Abstract:** Objectives: To describe further the phenomenology of drug-induced psychosis (DIP) in patients with Parkinson disease (PD) and assess which items on two common psychosis rating instruments—the Brief Psychiatric Rating Scale (BPRS) and the Neuropsychiatric Inventory (NPI)—are the best measure of DIP by comparing them with the Clinical Global Impression Scale (CGIS). Methods: Baseline data from two placebo-controlled, double-blind studies of olanzapine in PD patients with DIP were collected and analyzed. Results: A total of 157 of 160 patients had hallucinations, with visual hallucinations being the most common (97% of subjects), followed by auditory (48%), tactile (23%), and olfactory (16%). Seventy-six percent of subjects experienced delusions, and all types of delusions occurred with relatively equal frequency. The CGIS correlated with suspiciousness, hallucinatory behavior, unusual thought content, and hostility on the BPRS; and delusions, hallucinations, agitation, aberrant motor behavior, and sleep on the NPI. Conclusion: Nonvisual hallucinations and delusions may occur more frequently in DIP than previously thought. These symptoms, plus agitation and hostility, may ultimately be the best measure of DIP in patients with PD. (PsycINFO Database Record (c) 2012 APA, all rights reserved) (journal abstract)

**Source:** PsycINFO

50. **Treatment of dementia with Lewy bodies and Parkinson's disease dementia.**

**Author(s):** Poewe W

**Citation:** Movement Disorders, August 2005, vol./is. 20 Suppl 12/(S77-82), 0885-3185;0885-3185 (2005 Aug)

**Publication Date:** August 2005

**Abstract:** Cognitive decline and dementia affect approximately 30% to 40% of patients with idiopathic Parkinson's disease during the course of their illness. PD-dementia (PDD) and dementia with Lewy bodies (DLB) are second to Alzheimer's disease in causing degenerative dementia in the elderly. The nosological distinction of the conditions has remained controversial because of broad clinical and pathological overlap. Treatment issues in both clinical settings are virtually identical. Treatment of Parkinsonism is often complicated by drug-induced psychosis and reduced levodopa responsiveness. Cognition, alertness, attention, as well as apathy or aggressive behavior have been shown to respond to treatment with cholinesterase inhibitors in randomized controlled trials both in DLB and PDD. Such treatment may also improve hallucinosis, but many patients will require add-on treatment with atypical neuroleptics to control drug-induced psychotic reactions. Clozapine and quetiapine are the drugs most commonly used and, contrary to classic neuroleptics, risperidone or olanzapine do not seem to cause severe side effects according to published data. Copyright 2005 Movement Disorder Society.

**Source:** Medline

51. **Clozapine decreases [3H] CP 55940 binding to the cannabinoid 1 receptor in the rat nucleus accumbens.**

**Author(s):** Sundram S, Copolov D, Dean B

**Citation:** Naunyn-Schmiedebergs Archives of Pharmacology, May 2005, vol./is. 371/5(428-33), 0028-1298;0028-1298 (2005 May)

**Publication Date:** May 2005
Abstract: Antipsychotic drugs are effective in the treatment of cannabis-induced psychosis, but only clozapine appears effective in the treatment of comorbid schizophrenia and cannabis use. The unique effects of clozapine on cannabis use could, therefore, be due to an as yet unidentified interaction between clozapine and the endogenous cannabinoid system. To address this hypothesis, we used in situ radioligand binding and quantitative autoradiography with the selective cannabinoid CB1 receptor agonist, (-)-cis-[2-hydroxy-4-(1,1-dimethylheptyl)phenyl]-trans-4-(3-hydroxypropyl)cyclohexanol (side chain-2,3,4(N)-3H) ([3H]CP 55940) to measure the density of the CB1 receptor in frontal cortex, hippocampus, nucleus accumbens and striatum from rats treated with a variety of antipsychotic drugs. Clozapine significantly decreased [3H]CP 55940 binding in the nucleus accumbens compared with vehicle after 1 (35.0+-14.0 vs. 71.2+-8.5 fmol/mg estimated tissue equivalent (ete); P = 0.03) and 3 months (42.3+-4.0 vs. 71.1+-16.3 fmol/mg ete; P < 0.04) of treatment, an effect not observed with haloperidol, chlorpromazine or olanzapine. In rats treated with clozapine for 3 months and then left for 1 month without treatment, [3H]CP 55940 binding was not different in the nucleus accumbens (100.5+-22.2 vs. 100.9+-25.4 fmol/mg ete; P > 0.10). By contrast, there were significant increases in accumbal [3H]CP 55940 binding in rats treated with haloperidol (136.5+-14.2 fmol/mg ete; P < 0.05), chlorpromazine (137.4+-12.7 fmol/mg ete; P < 0.05) and olanzapine (144.7+-10.1 fmol/mg ete; P < 0.01). These data indicate that in the nucleus accumbens clozapine differs from other antipsychotic drugs in its effects on [3H]CP 55940 binding. If these results can be extrapolated into humans, then this effect of clozapine on the CB1 receptor may be a mechanism that makes it uniquely effective in schizophrenia and comorbid cannabis use.

Source: Medline
Available in fulltext from Naunyn-Schmiedeberg's Archives of Pharmacology at EBSCOhost

52. Schizophrenia and amphetamine dependence. A case report.

Author(s) Dervaux, A, Krebs, M. -O, Laqueille, X

Citation: L'Encephale: Revue de psychiatrie clinique biologique et therapeutique, March 2005, vol./is. 31/2(247-250), 0013-7006 (Mar-Apr 2005)

Publication Date: March 2005

Abstract: Whereas observations of psychotic disorders induced by amphetamines are common, few observations described the impact of chronic amphetamine abuse on schizophrenic patients. We report the case of a schizophrenic patient who presented with amphetamine dependence for several years, without other accompanying addiction. CASE REPORT: During his adolescence, Mr. X. gradually developed delusional beliefs of persecution and telepathy. He believed that the other pupils and teachers spoke about him in malicious terms. At the age of 23, Mr. X began to consume 60-100 mg/week of amphetamines orally. He consumed amphetamines during 7 years. The delusions, in particular the auditory hallucinations worsened after the use of amphetamines. Subsequently, he married and was declared unfit for national service due to the psychotic disorders. Mr. X received neuroleptic treatment with moderate effects on the psychotic symptoms. Between the age of 24 and 30, the patient presented persecutory, megalomaniac and physical transformation beliefs, delusions of being controlled as well as auditory, somatic-tactile and visual hallucinations. At the age of 30, while he had stopped his consumption of amphetamines for 9 months, the patient, overwhelmed with the delusions, murdered his wife. He was sent to jail for 13 months, and subsequently hospitalized for one year in a high security psychiatric department and 7 years in our psychiatric department. The neuroleptic treatment was effective, particularly against the hallucinations. Following stabilisation, the symptomatology of the patient was marked by a disorganization syndrome, including prominent thought disorder, disorganized speech, associative loosening, frequent derailments and negative signs of schizophrenia, in particular affective flattening and blunting of emotional expression. When the patient was 43, a trial discharge was authorized owing to improvement of his condition. The neuroleptic treatment was switched with single-drug olanzapine therapy, 10 mg/day which improved the negative symptoms. Mr. X. resumed part-time professional activities and remarried. Discussion: The patient fulfilled the DSM IV criteria for schizophrenia and for amphetamine dependence assessed using the Composite International Diagnostic Interview (CIDI). He presented, in particular, withdrawal syndrome when amphetamines were discontinued. The
amphetamine consumption was followed by a marked deterioration in the delusions, particularly the hallucinations. Worsening of the positive symptoms in schizophrenic patients by amphetamines has been established in single dose studies, in particular characterized by persecutory delusions and hallucinations. On the other hand, amphetamines tend to transiently and moderately reduce the negative symptoms. Some studies have shown that amphetamine consumption promoted violent acting out in non-schizophrenic subjects. In our observation, the acting out may be not related to the acute effects of these substances, since it occurred 9 months after stated discontinuation of amphetamine consumption. However, the cerebral toxicity and psycho-behavioural disturbances related to amphetamines might be prolonged after withdrawal. In non-schizophrenic patients, the existence of prolonged neurotoxicity of amphetamines and related psycho-behavioral disturbances has been suggested. The prolonged administration of amphetamines to animals produces neuro-axonal degeneration in the striatum, the frontal cortex, the nucleus accumbens and the amygdala. In human, there are some evidence of persistent deteriorations of the serotonergic and dopaminergic systems in the caudate nucleus, the putamen and the nucleus accumbens following amphetamine consumption. CONCLUSION: The neurobiological and psycho-behavioural effects of amphetamines may be prolonged following withdrawal in both schizophrenic and non-schizophrenic patients. (PsycINFO Database Record (c) 2012 APA, all rights reserved) (journal abstract)

**Source:** PsycINFO

53. [Clozapine and olanzapine in the treatment of the psychotic disorders in Parkinson's disease]. [Spanish] Clozapina y olanzapina en el tratamiento de los trastornos psicóticos de la enfermedad de Parkinson.

**Author(s)** Chacon J

**Citation:** Revista de Neurologia, October 2004, vol./is. 39/7(655-660), 0210-0010;0210-0010 (2004 Oct 1-15)

**Publication Date:** October 2004

**Abstract:** AIMS: The most important and serious neuropsychiatric disorders in Parkinson's disease (PD) are the medication-induced psychoses. They are remarkably frequent and are linked to a dopaminergic over-stimulation caused by the drugs administered over a long period of time. This complication increases the morbidity and mortality rates in PD and in the past it was very difficult to treat. Yet, the atypical antipsychotics have transformed its prognosis.DEVELOPMENT: We review the general pharmacology of the atypical antipsychotics, in particular clozapine and olanzapine, which we have wide experience with. We present two series in which these drugs were used with the psychosis in PD. Our studies were performed using a strict prospective methodology, with scales that are valid for psychosis, and the patients' motor signs. Both atypical antipsychotics were seen to improve the positive and negative symptoms of the patients, giving rise to no or scarce improvement of the motor signs of PD. The side effects of the two drugs were not important. Our two series are compared with others from the literature, with similar results as regards the improvements in the psychoses and the absence or only slight deterioration of Parkinson's disease.CONEClUSIONS: The psychoses induced by medication in PD are frequent and severe. The appearance of the atypical antipsychotics has come as a true 'revolution' in their treatment, owing to the good results obtained in the treatment of the psychoses and the scant deterioration of the motor signs of PD.

**Source:** Medline

54. Clozapine and olanzapine in the treatment of the psychotic disorders in Parkinson's disease [Spanish] Clozapina y olanzapina en el tratamiento de los trastornos psicóticos de la enfermedad de Parkinson

**Author(s)** Chacon Pena J.

**Citation:** Revista de Neurologia, October 2004, vol./is. 39/7(655-660), 0210-0010 (01 Oct 2004)

**Publication Date:** October 2004
Abstract: Aims. The most important and serious neuropsychiatric disorders in Parkinson's disease (PD) are the medication-induced psychoses. They are remarkably frequent and are linked to a dopaminergic over-stimulation caused by the drugs administered over a long period of time. This complication increases the morbidity and mortality rates in PD and in the past it was very difficult to treat. Yet, the atypical antipsychotics have transformed its prognosis. Development. We review the general pharmacology of the atypical antipsychotics, in particular clozapine and olanzapine, which we have wide experience with. We present two series in which these drugs were used with the psychosis in PD. Our studies were performed using a strict prospective methodology, with scales that are valid for psychosis, and the patients' motor signs. Both atypical antipsychotics were seen to improve the positive and negative symptoms of the patients, giving rise to no or scarce improvement of the motor signs of PD. The side effects of the two drugs were not important. Our two series are compared with others from the literature, with similar results as regards the improvements in the psychoses and the absence or only slight deterioration of Parkinson's disease. Conclusions. The psychoses induced by medication in PD are frequent and severe. The appearance of the atypical antipsychotics has come as a true 'revolution' in their treatment, owing to the good results obtained in the treatment of the psychoses and the scant deterioration of the motor signs of PD. 2004, Revista de Neurologia.

Source: EMBASE

55. Neuroleptics election and clinical outcomes in acute psychosis: A comparative study of risperidone versus other neuroleptics [Spanish] Eleccion de neuroleptico y respuesta clinica en psicosis aguda: Un estudio comparativo de risperidona frente a otros neurolepticos

Author(s) Pedros Rosello A., Tenias J.M.

Citation: Anales de Psiquiatria, April 2004, vol./is. 20/4(167-171), 0213-0599 (April 2004)

Publication Date: April 2004

Abstract: Background: Acute psychosis is continuously under revision, with insufficient criteria (DSM-IV, ICD- 10). The aims of this study are to identify a clinical frame when treatment is selected and determine the efectivity of the treatment in acute psychosis. Material and methods: 54 patients diagnosed of acute psychosis were included (brief psychosis, non specified psychosis, drug induced psychosis or schizophreniform psychosis). We analysed two groups: treated with risperidone (n=34) and treated with other antipsichotic agents: olanzapine, haloperidol and pimocide (n=20). Results: Acute episodes with depressive clinic were treated mainly with risperidone and episodes with maniac and agitation-aggressive symptoms with other neuroleptics. The clinical outcomes evaluated were similar in both groups. Antiparkinsonian agents were prescribed less frequently in the risperidone group. Conclusion: In spite of differences in independent variables clinical efectivity was equivalent in both groups of treatment.

Source: EMBASE

56. Aripraxole for Drug-induced Psychosis in Parkinson Disease: Preliminary Experience

Author(s) Fernandez H.H., Trieschmann M.E., Friedman J.H.

Citation: Clinical Neuropharmacology, January 2004, vol./is. 27/1(4-5), 0362-5664 (January/February 2004)

Publication Date: January 2004

Abstract: Aripiprazole is the newest atypical antipsychotic (AA) drug to be released in the US. It is the only AA that is a partial agonist at the D2 and 5HT1a receptors and an antagonist at 5HT2a receptors. It also has a high 5HT2/D2 ratio and may therefore carry a low risk of extrapyramidal side effects and alleviate psychosis in Parkinson-vulnerable populations. We report our preliminary experience in 8 patients with probable Parkinson disease (PD) treated with aripiprazole for drug-induced psychosis. Two patients were neurolepticaive, 5 patients were "quetiapine failures", and 1 patient was switched from olanzapine to aripiprazole. Aripiprazole was started at 5 mg to 10 mg a day and slowly
increased over 3 to 7 days until side effects or improvement of psychosis occurred. Only 2 out of 8 patients experienced near complete resolution of their psychosis using aripiprazole. The other six patients discontinued aripiprazole within 40 days, 2 of whom discontinued due to motor worsening. Our preliminary experience with aripiprazole is mixed but not very encouraging. Controlled studies are needed to evaluate aripiprazole in parkinsonian patients.

Source: EMBASE

57. Aripiprazole for Drug-Induced Psychosis in Parkinson Disease: Preliminary Experience.

Author(s) Fernandez, Hubert H, Trieschmann, Martha E, Friedman, Joseph H

Citation: Clinical Neuropharmacology, January 2004, vol./is. 27/1(4-8), 0362-5664;1537-162X (Jan-Feb 2004)

Publication Date: January 2004

Abstract: Aripiprazole is the newest atypical antipsychotic (AA) drug to be released in the US. It is the only AA that is a partial agonist at the D2 and 5HT1a receptors and an antagonist at 5HT2a receptors. It also has a high 5HT2/D2 ratio and may therefore carry a low risk of extrapyramidal side effects and alleviate psychosis in Parkinson-vulnerable populations. We report our preliminary experience in 8 patients with probable Parkinson disease (PD) treated with aripiprazole for drug-induced psychosis. Two patients were neuroleptic-naive, 5 patients were “quetiapine failures”, and 1 patient was switched from olanzapine to aripiprazole. Aripiprazole was started at 5 mg to 10 mg a day and slowly increased over 3 to 7 days until side effects or improvement of psychosis occurred. Only 2 out of 8 patients experienced near complete resolution of their psychosis using aripiprazole. The other six patients discontinued aripiprazole within 40 days, 2 of whom discontinued due to motor worsening. Our preliminary experience with aripiprazole is mixed but not very encouraging. Controlled studies are needed to evaluate aripiprazole in parkinsonian patients. (PsycINFO Database Record (c) 2012 APA, all rights reserved) (journal abstract)

Source: PsycINFO


Author(s) D’Innella P, Zaccala G, Terazzi M, Olgiati P, Torre E

Citation: Recenti Progressi in Medicina, July 2003, vol./is. 94/7-8(343-4), 0034-1193;0034-1193 (2003 Jul-Aug)

Publication Date: July 2003

Source: Medline

59. Anti-cataleptic effects of clozapine, but not olanzapine and quetiapine, on SCH 23390- or raclopride-induced catalepsy in rats.

Author(s) Ahlqvist J, Isacson R, Wahlestedt C, Salmi P

Citation: European Neuropsychopharmacology, May 2003, vol./is. 13/3(177-82), 0924-977X;0924-977X (2003 May)

Publication Date: May 2003

Abstract: The present study investigated potential anti-cataleptic properties of the prototype atypical antipsychotic clozapine and two newly developed atypical antipsychotics, olanzapine and quetiapine, which are structurally related and display similar pharmacological profiles to clozapine. Clozapine (2.5 mg kg(-1), s.c.), but not olanzapine (2.0 mg kg(-1), s.c.) and quetiapine (20.0 mg kg(-1), s.c.), blocked catalepsy induced either by the dopamine D(1/5) receptor antagonist SCH 23390 (50.0 microg kg(-1), s.c) or the selective dopamine D(2/3) receptor antagonist raclopride (4.0 mg kg(-1), s.c.). Such findings are consistent with the beneficial effects of clozapine in the management of drug-
induced psychosis in parkinsonian patients, and suggest that neither olanzapine nor quetiapine may be a safe alternative to clozapine in this field. Furthermore, the results indicate that clozapine has a unique pharmacological profile that distinguishes it from olanzapine and quetiapine. The mechanisms underlying anti-cataleptic or anti-parkinsonian properties of clozapine are unclear but may be related to dopamine D(1) receptor agonism of clozapine.

Source: Medline

60. Treatment of psychosis in Parkinson's disease: Safety considerations

Author(s) Fernandez H.H., Trieschmann M.E., Friedman J.H.

Citation: Drug Safety, 2003, vol./is. 26/9(643-659), 0114-5916 (2003)

Publication Date: 2003

Abstract: Psychosis only rarely occurs in patients with untreated Parkinson's disease. Much more commonly, psychosis is induced by drug therapy for Parkinson's disease and is the strongest known risk factor for nursing home placement. Delusions are less frequent than hallucinations, but are more concerning as they are often paranoid in nature. Treatment begins with a search for correctable infectious, toxic, and metabolic aetiologies. If symptoms persist, anti-Parkinson's disease medications are slowly reduced. However, withdrawal of these drugs usually worsens parkinsonism and is often not tolerated. Certain atypical antipsychotics can be used to treat psychosis without compromising motor function. The choice of atypical antipsychotic is largely based on ease of use and adverse effect profile as most have comparable efficacy in improving psychosis. Currently, there are five marketed atypical drugs - clozapine, risperidone, olanzapine, quetiapine and ziprasidone. Ziprasidone is the only agent whose adverse effect profile has not been reported in Parkinson's disease. The most common adverse effects of clozapine in Parkinson's disease are sedation, orthostatic hypotension and sialorrhoea. Sedation is generally helpful since these patients are frequently awake at night and tend to have worse behavioural problems then. Clozapine does not induce deterioration of motor function, but it has the potential to cause agranulocytosis, which is idiosyncratic and not dose-related. In risperidone-treated Parkinson's disease patients, reported adverse effects include somnolence, sialorrhoea, dizziness, palpitations, constipation, delirium, fatigue, leg cramps, depression, urinary incontinence and hypotension. Although in some Parkinson's disease studies, risperidone has been well tolerated, others have shown that many patients are unable to tolerate the drug due to deterioration of motor function. While an initial study of olanzapine in Parkinson's disease psychosis showed the drug to be effective without deterioration of motor function, succeeding reports demonstrated a deleterious effect of the drug on motor functioning. The most common adverse effects of quetiapine in Parkinson's disease patients are sedation and orthostatic hypotension. There is a lack of double-blind trials; however, cumulative reports involving >200 Parkinson's disease patients strongly suggest that quetiapine is well tolerated and effective. Unlike clozapine, it does not improve tremor and may induce mild deterioration of motor function. Recently, cholinesterase inhibitors have been reported to alleviate psychosis in Parkinson's disease. Although ondansetron, an antiemetic with antiserotonergic properties, has been reported to relieve psychosis in Parkinson's disease, its prohibitive cost has prevented further study in this population. Electroconvulsive treatment is generally reserved for the patient with psychotic depression who is unable to tolerate any pharmacological therapy.

Source: EMBASE

Available in fulltext from Drug Safety at EBSCOhost

61. Atypical antipsychotics in the EPS-vulnerable patient.

Author(s) Friedman, J. H

Citation: Psychoneuroendocrinology, January 2003, vol./is. 28/Su[pp]l(39-51), 0306-4530 (Jan 2003)

Publication Date: January 2003

Abstract: "Typical' antipsychotic agents can lead to a variety of extrapyramidal symptoms
including parkinsonism. The efficacy of a number of atypical antipsychotics in reducing psychosis without a detrimental effect on motor function has been studied in the group of patients most vulnerable to EPS, those who already have parkinsonian symptoms. Multiple open-label studies with clozapine strongly suggested that at low doses the drug was an effective antipsychotic and did not impair motor function. A disadvantage of clozapine is that it can cause agranulocytosis and therefore patients require ongoing hematological monitoring. Studies with both risperidone and olanzapine have produced conflicting results. As with clozapine, multiple open-label studies with quetiapine have consistently demonstrated that it improves psychosis without impairing motor function. Some experts recommend quetiapine as the drug of choice for treatment of drug-induced psychosis in patients with parkinsonism. The atypical antipsychotics have also been tested in the largest group of EPS-vulnerable patients, the demented elderly. Results from a number of trials are described here; the evidence to date indicates a generally low incidence of tardive dyskinesia with atypical antipsychotics. (PsycINFO Database Record (c) 2012 APA, all rights reserved)
appears in PD patients. The results obtained show OLZ to be an effective antipsychotic drug. However, the data on its capacity to deteriorate motor function is contradictory and it has not been possible to pinpoint the reasons why this adverse side effect appears in some patients and not in others. The causes that have been suggested, although they do not account for all the cases, are the use of high doses of OLZ and the prior existence of dementia. Moreover, some cases of OLZ induced agranulocytosis have been detected, although it was thought that this side effect within the AA was exclusive to clozapine.

CONCLUSION: Although it is effective as an antipsychotic drug, there exists contradictory data about the capacity of OLZ to deteriorate the state of patients suffering from Parkinson's disease, which means that it does not seem to be the first choice drug in the DIP that appears in PD.

Source: Medline


Author(s) Breier, Alan, Sutton, Virginia K, Feldman, Peter D, Kadam, Deborah L, Ferchland, Iris, Wright, Padraig, Friedman, Joseph H

Citation: Biological Psychiatry, September 2002, vol./is. 52/5(438-445), 0006-3223 (Sep 2002)

Publication Date: September 2002

Abstract: Reports results from 2 placebo-controlled, double-blind studies of olanzapine for treatment of dopamimetic drug-induced psychosis in patients with Parkinson's disease (PD). Patients were treated with olanzapine or placebo for 4 wks while dopamimetic therapy was held constant. Olanzapine was initiated at 2.5 mg/day, with 2.5-mg/day increases allowed every 3 to 4 days up to the maximum dose of 15 mg/day. Olanzapine patients showed significant improvements from baseline on positive symptoms and most efficacy measures, but no significant treatment-group differences were observed. Olanzapine performed significantly worse than placebo in both studies on the Unified Parkinson's Disease Rating Scale (UPDRS) total, Motor, and Activities of Daily Living scales, but not the UPDRS Tremor item or Complications scores. Corrected QT interval, vital signs, and body weight were not significantly different from placebo. Findings did not demonstrate superior efficacy of olanzapine for treatment of dopamimetic-induced psychosis in PD. The initial dose-titration schedule and mild baseline levels of psychosis may account for these findings. Future studies involving gradual dose titration are needed to explore further olanzapine's optimum use for patients with PD with treatment-related psychosis. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

65. Improvement of levodopa-induced psychosis and dyskinesias in Parkinson's disease patients with low-dose olanzapine: An open-label trial

Author(s) Lin J.-J., Chang D.-C., Yueh K.-C.

Citation: Acta Neurologica Taiwanica, September 2002, vol./is. 11/3(128-134), 1019-6099 (September 2002)

Publication Date: September 2002

Abstract: Sixteen consecutive patients with Parkinson's disease (PD) complicated by levodopa-induced psychosis were treated with low-dose olanzapine (OLZ). This started at 2.5 mg daily and slowly increased in 2.5-mg increments as needed. Their psychotic symptoms were rated by the Scale for the Assessment of Positive Symptoms (SAPS). The Visual Hallucinations item (number 9) from the SAPS and the Brief Psychiatric Rating Scale (BPRS) were used as the behavioral outcome measures. Furthermore, the Unified Parkinson's Disease Rating Scale (UPDRS) total motor activity of daily living (ADL) subscales were used for measuring their parkinsonian symptoms. The Modified Dyskinesia Rating Scale (MDRS) was used for measuring their dyskinesias induced by levodopa. A comparison of these rating scales was made before and three months after the treatment. OLZ could significantly reduce SAPS total scores by 65.9%, SAPS subscores for the specific visual hallucination items by 60.5%, and BPRS total scores by 42.9% in these PD
patients with psychosis. Furthermore, this drug could also significantly reduce the mean score for dyskinesia by 51.4%. Although treatment with OLZ could slightly worsen the UPDRS motor and ADL subscales, the degree of worsening was not significant. Overall, there was no significant increase in levodopa dose in this study. In conclusion, the results of our study revealed that treatment with low-dose OLZ could improve levodopa-induced psychiatric symptoms and dyskinesias without worsening motor function in PD patients with psychosis.

Source: EMBASE


Author(s) Chacon JR, Duran E, Duran JA, Alvarez M

Citation: Neurologia, January 2002, vol./is. 17/1(7-11), 0213-4853;0213-4853 (2002 Jan)

Publication Date: January 2002

Abstract: BACKGROUND: To evaluate the antipsychotic efficacy of olanzapine (OLZ) in patients with Parkinson's disease (PD) and drug-induced psychosis (DIP) and its repercussion on the motor function. METHODS: Ten patients (5 women and 5 men) diagnosed of PD and DIP, aged 67 years (range: 50-81), with PD duration of 11.1 years (range: 6-23), treated chronically with levodopa per day, received a dose of 2.5 or 5.0 mg OLZ daily. Data concerning improvement of psychosis and worsening of motor function was based on Positive And Negative Symptoms Scale (PANSS) and Unified Parkinsons Disease Rating Scale (UPDRS) motor. RESULTS: Psychotic symptoms were improved in all patients. In most of them the improvement was almost total. Seven patients increased levodopa dose on OLZ, but significant worsening of motor function was reported just in one patient. None of the patients had agranulocytosis in the blood monitoring. Two patients presented weight gain. Seven patients improved their cognitive status. CONCLUSIONS: We conclude that OLZ at the doses studied may have efficacy for DIP which appears in PD and does not induce worsening of motor function in most of the patients.

Source: Medline

67. Usefulness of olanzapine in the levodopa-induced psychosis in patients with Parkinson's disease [Spanish] Utilidad de la olanzapina en la psicosis inducida por levodopa en pacientes Parkinsonianos

Author(s) Chacon J.R., Duran E., Duran J.A., Alvarez M.

Citation: Neurologia, 2002, vol./is. 17/1(7-11), 0213-4853 (2002)

Publication Date: 2002

Abstract: BACKGROUND: To evaluate the antipsychotic efficacy of olanzapine (OLZ) in patients with Parkinson's disease (PD) and drug-induced psychosis (DIP) and its repercussion on the motor function. METHODS: Ten patients (5 women and 5 men) diagnosed of PD and DIP, aged 67 years (range: 50-81), with PD duration of 11.1 years (range: 6-23), treated chronically with levodopa per day, received a dose of 2.5 or 5.0 mg OLZ daily. Data concerning improvement of psychosis and worsening of motor function was based on Positive And Negative Symptoms Scale (PANSS) and Unified Parkinson's Disease Rating Scale (UPDRS) motor. RESULTS: Psychotic symptoms were improved in all patients. In most of them the improvement was almost total. Seven patients increased levodopa dose on OLZ, but significant worsening of motor function was reported just in one patient. None of the patients had agranulocytosis in the blood monitoring. Two patients presented weight gain. Seven patients improved their cognitive status. CONCLUSIONS: We conclude that OLZ at the doses studied may have efficacy for DIP which appears in PD and does not induce worsening of motor function in most of the patients.

Source: EMBASE

68. Ecstasy-induced psychotic disorder: Six-month follow-up study
Objective: To describe the psychiatric symptoms manifested by persons diagnosed for the first time as having ecstasy-induced psychotic disorder and to explore the evolution of their symptoms over a 6-month period. Design: Observational study with a 6-month follow-up. Method: The subjects studied were 32 ecstasy consumers who were treated at two drug-dependency outpatient centers for hallucinatory-delusional manifestations and who were diagnosed as having ecstasy-induced psychotic disorder according to DSM-IV criteria. For the assessment of the intensity of the syndrome and its follow-up, the Brief Psychiatric Rating Scale (BPRS), the Hamilton Depression Rating Scale (HDRS) and the Clinical Global Impression (CGI) were used at the outset and after 1, 3 and 6 months. All subjects received treatment with olanzapine. Results: The treatment program was completed by 96.9% of the patients. At the baseline assessment, a high incidence of symptoms of a severe psychiatric disorder was observed. From the first month the psychotic symptoms (BPRS) were considerably reduced with treatment, with the most severe positive symptoms remitting in the first 3 months. The three assessment indicators (BPRS, HDRS and CGI) showed a statistically significant clinical reduction over the 6 months of the assessment period. Furthermore, no relevant side effects were noted. Conclusions: In its initial manifestations, a drug-induced psychotic syndrome includes marked symptoms meeting the criteria of a severe psychotic disorder, with the presence of considerable positive and negative symptoms. Olanzapine has been shown to be very effective in these situations and its use is suggested as first-choice therapy. Copyright 2002 S. Karger AG, Basel.

Source: EMBASE
Available in fulltext from European Addiction Research at EBSCOhost

69. Persistent psychosis after a single ingestion of ‘ecstasy’.

Author(s) Van Kampen, Janice, Katz, Mark

Abstract: Describes the case of a female (aged 18 yrs) who developed prolonged psychosis after a single recreational use of methylenedioxymethamphetamine (MDMA). It is argued that the diagnosis of MDMA-induced psychotic disorder seems most appropriate given the absence of any sustained mood disturbance and the lack of evidence of any prodromal symptoms. Her psychosis was treated successfully with olanzapine. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

70. Treatment options for depression and psychosis in Parkinson’s disease.

Author(s) Poewe W, Seppi K

Abstract: Neuropsychiatric symptoms are a frequent feature of advancing Parkinson’s disease (PD). The reported prevalence of depression varies greatly between different studies but there is general consensus that between 40 and 50% of patients will be affected. Depression may antedate motor manifestations of Parkinson’s disease and is usually of moderate or mild intensity. However, depression is of major impact on the quality of life in PD patients according to a recent survey. Drug-induced psychosis is one of the major therapeutic challenges in Parkinson’s disease and may occur in up to 6% in otherwise uncomplicated de novo patients when first receiving dopaminergic therapy. It
increases in frequency, in advanced disease and particularly in patients with dementia where up to 22% may be affected. There is an amazing lack of controlled clinical trials assessing the effects of antidepressants in clinical trials including more than 20 patients and assessing efficacy of antidepressants specifically in the context of mood changes in Parkinson's disease. A comprehensive literature search yielded only a total of 17 articles of which a majority included less than 20 patients and/or did not use valid depression ratings. The only randomized controlled trial was conducted more than 20 years ago using nortryptiline while no controlled trials were available on the use of serotonin reuptake inhibitors. Studies assessing the antidepressant action of dopaminergic therapies are fewer and inconclusive. Thus, while tricyclic antidepressants or SSRIs are widely used in clinical practice, there is still a need for controlled clinical trials proving their efficacy specifically in parkinsonian depression. Three randomized controlled trials are now available assessing the efficacy of the atypical neuroleptics clozapine and olanzapine in the treatment of drug-induced psychosis. While clozapine is of proven efficacy at least in the short-term management of this complication without negative impact on the motor symptoms, olanzapine in currently used doses of 2.5 to 15 mg/d seems to aggravate motor symptoms with lesser effect on psychosis compared to clozapine. Currently, clozapine is the atypical neuroleptic of choice for the treatment of drug-induced psychosis in Parkinson's disease.

Source: Medline
Available in fulltext from Journal of Neurology at EBSCOhost

71. [Pharmacological modulation of the effects induced by ketamine at subanesthetic doses]. [French] Modulation pharmacologique des effects induits par la ketamine a des doses subanesthesiques.

Author(s) Mechri A, Micallef J, Blin O, Saoud M, Dalery J, Gaha L
Citation: Therapie, September 2001, vol./is. 56/5(617-22), 0040-5957;0040-5957 (2001 Sep-Oct)
Publication Date: September 2001
Abstract: The similarity between ketamine effects and endogenous psychoses has created interest in the capacity of antipsychotic medications to block ketamine effects. In healthy subjects, a sub-anaesthetic single dose of lorazepam, typical neuroleptics, such as haloperidol, and atypical neuroleptics, such as clozapine and olanzapine, failed to block ketamine-induced positive and negative symptoms resembling schizophrenia. However, haloperidol is able to decrease ketamine-induced impairment in executive cognitive functions. Recently, lamotrigine reduced ketamine-induced psychotic symptoms, perceptual alterations, and cognitive impairments. In schizophrenic subjects, single doses of olanzapine do not decrease the effects of ketamine. However, long term treatment with clozapine has been reported to decrease ketamine-induced positive symptoms. Pharmacological modulation of the effects of NMDA receptor antagonists, such as ketamine, may lead to development of novel therapeutic agents for psychiatric illnesses such as schizophrenia.

Source: Medline

72. Olanzapine as alternative therapy for patients with haloperidol-induced extrapyramidal symptoms: Results of a multicenter, collaborative trial in Latin America.

Author(s) Costa E Silva, Jorge A, Alvare, Nelson, Mazzotti, Guido, Gattaz, Wagner Farid, Ospina, Jorge, Larach, Veronica, Starkstein, Sergio, Oliva, Daniel, Cousins, Lynne, Tohen, Mauricio, Taylor, Cindy C, Wang, Jeff, Tran, Pierre V
Citation: Journal of Clinical Psychopharmacology, August 2001, vol./is. 21/4(375-381), 0271-0749;1533-712X (Aug 2001)
Publication Date: August 2001
Abstract: Patients with schizophrenia and related disorders who were taking haloperidol (N = 94; mean dose = 12.7 mg/day; aged 18-53 yrs) and had extrapyramidal symptoms (EPS; Simpson-Angus Scale [SAS] > 3) were directly switched to 6 wks of open-label olanzapine
treatment (mean dose = 11.4 mg/day). There were significant mean improvements from baseline to endpoint on the SAS, the Barnes Akathisia Scale, and the Abnormal Involuntary Movement Scale, and anticholinergic use decreased from 47.9% to 12.8%. Significant mean baseline to endpoint improvements were observed on the Positive and Negative Syndrome Scale (PANSS), the PANSS-extracted Brief Psychiatric Rating Scale, and the Clinical Global Impressions Severity scale. Spontaneously reported treatment-emergent adverse events with a greater than 5% incidence were somnolence, increased appetite, weight gain, headache, anxiety, dizziness, and insomnia. Criteria for a successful switch were met by 90.5% of patients. Psychotic symptom exacerbation was experienced by 30.9% of patients at any time during the study and by 11.7% of patients at endpoint. Results suggest that a direct switch to olanzapine is a therapeutic option when patients with haloperidol-induced EPS are unable to tolerate a more gradual switch. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

Available in fulltext from Journal of Clinical Psychopharmacology at East Midlands Ovid Archive Collection

73. Parkinson's disease: the treatment of drug-induced hallucinations and psychosis.

Author(s) Molho ES, Factor SA

Citation: Current Neurology & Neuroscience Reports, July 2001, vol./is. 1/4(320-8), 1528-4042:1528-4042 (2001 Jul)

Publication Date: July 2001

Abstract: Drug-induced psychosis is one of the most disabling complications of advancing Parkinson's disease. It has also been one of the most difficult to treat. Clozapine was the first medication shown to be safe and effective in this setting, and it remains the standard by which newer atypical antipsychotics are measured. However, due to the small but significant risk of agranulocytosis and the need for frequent blood testing, alternatives have been sought. Risperidone, olanzapine, and quetiapine are new atypical antipsychotics that have each been proposed as an alternative to clozapine, but the literature concerning their use in Parkinson's disease is conflicted and confusing. Although quetiapine appears to be the best current choice, none of these medications have equaled clozapine's ability to safely treat drug-induced psychosis without the risk of worsening parkinsonism.

Source: Medline

74. Pharmacological modulation of the effects of subanaesthetic ketamine: A review [French] Modulation pharmacologique des effets induits par la ketamine a des doses subanesthesiques

Author(s) Mechri A., Micallef J., Blin O., Saoud M., Dalery J., Gaha L.

Citation: Therapie, 2001, vol./is. 56/5(617-622), 0040-5957 (2001)

Publication Date: 2001

Abstract: The similarity between ketamine effects and endogenous psychoses has created interest in the capacity of antipsychotic medications to block ketamine effects. In healthy subjects, a subanaesthetic single dose of lorazepam, typical neuroleptics, such as haloperidol, and atypical neuroleptics, such as clozapine and olanzapine, failed to block ketamine-induced positive and negative symptoms resembling schizophrenia. However, haloperidol is able to decrease ketamine-induced impairment in executive cognitive functions. Recently, lamotrigine reduced ketamine-induced psychotic symptoms, perceptual alterations, and cognitive impairments. In schizophrenic subjects, single doses of olanzapine do not decrease the effects of ketamine. However, long term treatment with clozapine has been reported to decrease ketamine-induced positive symptoms. Pharmacological modulation of the effects of NMDA receptor antagonists, such as ketamine, may lead to development of novel therapeutic agents for psychiatric illnesses such as schizophrenia.
75. Efficacy and safety of clozapine and olanzapine: An open-label study comparing two groups of Parkinson's disease patients with dopaminergic-induced psychosis

Author(s) Gimenez-Roldan S., Mateo D., Navarro E., Gines M.M.

Citation: Parkinsonism and Related Disorders, 2001, vol./is. 7/2(121-127), 1353-8020 (2001)

Publication Date: 2001

Abstract: Clozapine, an atypical neuroleptic agent, improves dopaminergic-induced psychosis in parkinsonian patients without increasing motor disability. However, because of the risk of agranulocytosis periodic hematological controls are mandatory. Olanzapine, another atypical neuroleptic, does not require such monitoring, which may represent a practical advantage. Therefore, for 12 weeks we compared the tolerability and efficacy of clozapine and olanzapine in two groups of nine consecutive parkinsonian psychotic patients treated with these compounds. All the patients on clozapine (mean starting dose: 13.1 +/- 7.9 mg/d) completed the study despite reporting a number of adverse events, including somnolence, falls, orthostatic hypotension, and syncopate. In contrast, early withdrawal occurred in three of the nine patients receiving olanzapine, due to severe gait deterioration and drowsiness (mean starting dose: 3.9 +/- 1.3 mg/d). Psychotic symptoms improved in both groups, as reflected by a reduction of 71.7% in clozapine and a 61.7% reduction in olanzapine, in five selected items from the Neuropsychiatric Inventory. On conclusion of the study, parkinsonism had improved in the clozapine group with a 19.7% decrease in the raw scores and a 7.9% decrease in the weighted scores according to the Cornell University Rating Scale for parkinsonism (mean dose: 16.9 +/- 10.3 mg/d). Conversely, the six patients receiving olanzapine who finished the study experienced aggravated parkinsonian symptoms, with a 25.5% worsening in the raw scores and a 24.6% worsening in the weighted scores (mean dose: 4.7 +/- 2.3 mg/d). We postulate that the early drop-outs in the olanzapine-treated parkinsonian group may be attributable to a non-specific effect of the drug as a result of starting at too high a dose, and that the worsening of parkinsonism following prolonged treatment may have been caused by the drug's blocking effect on striatal D2 receptors. Copyright 2001 Elsevier Science Ltd.

Source: EMBASE

76. Drug-induced psychotic symptoms in Parkinson's disease. Problems, management and dilemma

Author(s) Kuzuhara S.

Citation: Journal of Neurology, Supplement, 2001, vol./is. 248/3(28-31), 0939-1517 (2001)

Publication Date: 2001

Abstract: Psychotic symptoms develop in 20-30% of patients with Parkinson's disease (PD) receiving chronic anti-PD medications, and visual hallucinations with or without delirium and paranoid delusions are the most frequent symptoms. Psychotic symptoms disturb ADL and QOL of PD patients and tax caregivers far more than the motor disabilities do, and good management of drug-induced psychotic symptoms is potentially important. Withdrawal of anti-PD drugs relieves the patients from psychotic side effects, but worsens the parkinsonian motor symptoms. The first step of treatment is to eliminate triggering factors other than anti-PD drugs, such as infections, metabolic disorders, subdural hematoma, and hallucinogenic drugs. The second step is to eliminate anti-PD drugs in the following order; first anticholinergics, amantadine and selegiline, second dopamine agonists, and finally levodopa/carbidopa. Anti-PD medications should be reduced to the point of improving psychotic side effects without drastically worsening parkinsonian motor symptoms. When the above adjustments fail to sufficiently alleviate psychotic side effects, the third step is consideration of antipsychotic drugs although they have potential capacity to antagonize dopamine D2 receptors and worsen parkinsonism. Atypical antipsychotics such as clozapine and olanzapine are recommended, though the former is not available in Japan.
77. Dopamine transporter density in young patients with schizophrenia assessed with [123I]FP-CIT SPECT.

Author(s) Lavalaye, Jules, Linszen, Don H, Boonj, Jan, Dingemans, Peter M. A. J, Reneman, Liesbeth, Habraken, Jan B. A, Gersons, Berthold P. R, van Royen, Eric A

Citation: Schizophrenia Research, January 2001, vol./is. 47/1(59-67), 0920-9964 (Jan 2001)

Publication Date: January 2001

Abstract: Disturbances in the dopamine (DA) system are thought to play a major role in schizophrenia. Amphetamine-induced release of endogenous DA is shown to be enhanced in schizophrenia, as is striatal [8F]FDOPA uptake in the striatum. It is not clear if the density of DA neurons is altered in schizophrenia. By studying the DA transporter with [123I]FP-CIT SPECT, the density of nigrostriatal dopaminergic cells can be studied. DA transporter density in the striatum was studied in 36 young patients (19-22 yrs old) with schizophrenia. Ten patients were antipsychotic (AP)-naive, 15 were treated with olanzapine, 8 with risperidone, and 3 were AP-free. A control group of 10 age-matched volunteers was included. Striatal [123I]FP-CIT binding was not significantly different between AP-naive patients (2.87), patients treated with olanzapine (2.76), patients treated with risperidone (2.76), AP-free patients (2.68), and controls (2.82). Unexpectedly, striatal [123I]FP-CIT binding in females was significantly higher than in males. In conclusion, functional changes in the dopaminergic system in schizophrenia are not likely to be reflected in change in DA transporter density. Moreover, DA transporter density does not seem to be altered by AP medication. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

78. One further case of Pancytopenia induced by olanzapine in a Parkinson’s disease patient.

Author(s) Onofrj, Marco, Thomas, Astrid

Citation: European Neurology, 2001, vol./is. 45/1(56-57), 0014-3022;1421-9913 (2001)

Publication Date: 2001

Abstract: Olanzapine is a new atypical antipsychotic agent, marked for treatment of refractory schizophrenia with a low risk of extrapyramidal side effects, that apparently does not share the known hematological side effects with clozapine. Early reports suggested that olanzapine could be safely used in patients affected by psychosis in Parkinson's disease (PD) and might therefore become a substitute of clozapine in the treatment of these disturbances. For this reason, the authors used olanzapine for the treatment of psychotic disturbances with hallucinations and paranoid delusions in a 67-yr-old male affected by PD for at least 15 yrs. The present report shows that olanzapine improved L-DOPA-induced psychosis, but unexpectedly induced pancytopenia in the S, accompanied by worsening of motor symptoms. (PsycINFO Database Record (c) 2013 APA, all rights reserved)

Source: PsycINFO

Available in fulltext from European Neurology at EBSCOhost

79. Switching clozapine responders to olanzapine.

Author(s) Littrell, Kimberly H, Johnson, Craig G, Hilligoss, Nicole M, Peabody, Carol D, Littrell, Steven H

Citation: Journal of Clinical Psychiatry, December 2000, vol./is. 61/12(912-915), 0160-6689 (Dec 2000)

Publication Date: December 2000

Abstract: Determined the effectiveness and safety of a method of slow cross-titration from clozapine to olanzapine among patients responsive to clozapine treatment but experiencing
medication-induced adverse events. Changes in symptomatology, mood, subjective response, and safety were examined in 20 outpatients meeting Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV) criteria for schizophrenia or schizoaffective disorder who converted from clozapine to olanzapine. Patients were considered clozapine-responsive as evidenced by improved social function and decreased symptoms with clozapine therapy; however, they were interested in alternative pharmacologic treatment because of clozapine-related side effects. Equivalent efficacy of olanzapine to clozapine was found in 90% of the patients (18/20) in the study group, without rehospitalization or suicidal behavior in any of the patients. Also notable was a reduction in drug-induced side effects and improved subjective response to pharmacotherapy. The successful conversion from clozapine to olanzapine has the potential to provide great benefits for the patient, including reducing drug-induced side effects while maintaining symptom control. (PsycINFO Database Record (c) 2013 APA, all rights reserved)

Source: PsycINFO

80. Serotonin and dopamine antagonism in obsessive-compulsive disorder: effect of atypical antipsychotic drugs.

Author(s) Ramasubbu R, Ravindran A, Lapierre Y

Citation: Pharmacopsychiatry, November 2000, vol./is. 33/6(236-8), 0176-3679;0176-3679 (2000 Nov)

Publication Date: November 2000

Abstract: BACKGROUND: Previous reports suggest that some atypical antipsychotics may have obsessogenic as well as antiobsessional effects. Given their higher affinity for serotonin 5HT2 receptors than dopamine D2 receptors, it has been speculated that atypical antipsychotics may induce obsessive-compulsive (OC) symptoms, even at low doses, due to high 5HT2 antagonism, whereas improvement in OC symptoms is thought to occur only at high doses due to high D2 antagonism.METHOD: In this open case series, the dose-response relationship of atypical antipsychotic augmentation in the treatment of obsessive compulsive disorder (OCD), and the dose-severity relationship in atypical anti psychotic-induced OC symptoms were examined. Three patients were identified who had either refractory OCD or OC symptoms following administration of atypical antipsychotics such as olanzapine and risperidone.RESULTS: Case 1: A linear dose-response relationship between increasing doses of olanzapine and improvement in OC symptoms was observed in an OCD patient resistant to 5-HT reuptake inhibitors. 2: OC symptoms induced by low doses of risperidone (1 mg) were reversed by increasing the doses of risperidone (3 mg) in a bipolar disorder patient suggesting an inverse dose-severity relationship. 3: No inverse dose-severity relationship was noted between olanzapine induced OC symptoms and its dosage in an asymptomatic OCD patient. Treatment-emergence OC symptoms responded to increasing the doses of maintenance clomipramine treatment.CONCLUSIONS: Controlled studies are needed to investigate the dose-response or dose-severity relationships between OCD and atypical antipsychotics.

Source: Medline

81. Quetiapine for (I)-dopa-induced psychosis in PD.

Author(s) Fernandez, Hubert H

Citation: Neurology, September 2000, vol./is. 55/6(899), 0028-3878;1526-632X (Sep 2000)

Publication Date: September 2000

Abstract: Presented a similar case to that reported by W. J. Weiner et al (see record 2000-03198-009) in which a 77-yr-old male patient with a 12-year history of Parkinson's disease (PD) was treated successfully for his drug-induced psychosis with quetiapine after the patient failed to respond to olanzapine. In this case, olanzapine caused worsening of parkinsonism and the benefit of clozapine was negated by the cumbersome blood-monitoring requirement. Quetiapine controlled psychosis without worsening parkinsonism and without the need for blood monitoring. The case also demonstrates that a switch over from clozapine to quetiapine may be attempted even among psychiatrically-stable patients
82. Olanzapine treatment of methamphetamine psychosis.

Author(s): Misra, Lalith K, Kofoed, Lial, Oesterheld, Jessica R, Richards, George A

Citation: Journal of Clinical Psychopharmacology, June 2000, vol./is. 20/3(393-394), 0271-0749;1533-712X (Jun 2000)

Publication Date: June 2000

Abstract: Describes a case of a 50-yr old male with a history of alcohol, cannabis, and cocaine dependencies who developed a persistent paranoid-hallucinatory state after 1 yr of methamphetamine use. On 2 separate occasions, olanzapine effectively treated both acute and residual psychotic states. It is suggested that atypical antipsychotics can be effective in the treatment of acute and residual methamphetamine-induced psychosis. In this case, the patient relapsed into methamphetamine abuse and psychosis and recovered when he resumed olanzapine. Adherence to olanzapine treatment for approximately 8 wks also effectively controlled his cravings for methamphetamine. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

Available in fulltext from Neurology at the ULHT Library and Knowledge Services' eJournal collection

83. Manic psychosis induced by long term alpha-interferon treatment for hepatitis C.

Author(s): Howes, Oliver D, McKenzie, Kwame J

Citation: International Journal of Psychiatry in Clinical Practice, June 2000, vol./is. 4/2(161-162), 1365-1501;1471-1788 (Jun 2000)

Publication Date: June 2000

Abstract: There is increasing evidence of psychiatric side-effects following long-term alpha-interferon treatment, but no previous reports of psychosis as a side effect. There is little evidence to suggest the best treatment of interferon-related psychiatric illness. A case of manic psychosis developing after long-term alpha-interferon treatment is reported in a 38-yr-old woman with no previous psychiatric history. The patient did not respond to termination of alpha-interferon therapy. She responded partially to olanzapine but completely recovered after sodium valproate was added, with no deleterious effects. Psychiatric side-effects, including psychosis, are appreciable problems of alpha-interferon. This report suggests that the pathological mechanisms of early and late side-effects are different. Sodium valproate proved to be a safe and effective treatment. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

Available in fulltext from Journal of Clinical Psychopharmacology at East Midlands Ovid Archive Collection

84. Atypical antipsychotics in the treatment of drug-induced psychosis in parkinson's disease

Author(s): Friedman J.H., Factor S.A.

Citation: Movement Disorders, 2000, vol./is. 15/2(201-211), 0885-3185 (2000)

Publication Date: 2000

Abstract: Our experience with atypical antipsychotics in patients with PD is that their motor effects are not predictable. The multiple reports concerning clozapine’s beneficial effects on tremor, dystonia, nocturnal akathisia, and dyskinesias all underscore this observation. However, the appearance of even minor degrees of parkinsonism in normal volunteers or
schizophrenics should suggest that an antipsychotic will not be well-tolerated in patients with PD. The treatment of PD is probably the most stringent test of a drug’s freedom from parkinsonian side effects. The data from trials in schizophrenia concerning parkinsonian effects cannot always be confidently interpreted. Virtually all subjects in these trials have been treated with typical neuroleptics until shortly before study entry. Because the parkinsonian side effects of these drugs may persist for several months, patients may still show declining levels of parkinsonism even when placed on a drug that induces it if this effect is milder than that induced by the pre-study neuroleptic. Depending on the pre-study drug used and the duration of the study, distinguishing placebo from a low-potency neuroleptic may be impossible. Furthermore, the standard measure of parkinsonism in psychiatric studies is the Simpson-Angus scale which is heavily weighted toward rigidity and may underscore bradykinesia, gait, and posture abnormalities. The prolactin response to an antipsychotic drug may turn out to be a good predictor of its freedom from parkinsonian side effects. That would fit with the data presented above of clozapine and quetiapine having less parkinsonian effects, olanzapine having more but variable effects and risperidone being poorly tolerated. With the data presented above, comprising a current review of all reports of the use of atypical antipsychotics in PD that we could locate, we can say little with certainty. The only drug with confirmed benefit without worsening parkinsonism is clozapine. Open-label trials involving over 400 patients and two multicenter, placebo-controlled, double-blind trials have demonstrated that it is effective in treating the psychosis. It improves tremor, does not worsen other motor functions to any significant extent, and is safe at low doses. Limited data provide conflicting information on both risperidone and olanzapine. Quetiapine seems to be well-tolerated with some, but definitely less, worsening of PD motor features than risperidone and olanzapine. Based on the current literature, our personal experience, and anecdotal experience of other PD specialists which we solicited, we will venture our own interpretation and recommendations. We think risperidone is poorly tolerated and should be used only as a last resort; that olanzapine is better than risperidone but will, in a majority of patients with PD, worsen motor function. We are optimistic, but not yet convinced, that quetiapine may prove to be as effective and better tolerated than clozapine. It will not require cumbersome monitoring because it does not induce a blood dyscrasia. We therefore recommend that DP be treated in the following manner. First, the anti-PD medications should be simplified and reduced as much as tolerated. We think, in general, side effects multiply more with increasing numbers of drugs than with drug dose, so that patients are more likely to tolerate a higher dose of levodopa than a lower dose of levodopa combined with other adjunctive anti-PD medications. In reducing anti-PD medications, we recommend tapering and stopping, if necessary, the drugs with the highest risk-to-benefit ratio first. Anticholinergics are stopped first, then selegiline, dopamine agonists, amantadine, and finally COMT inhibitors, which have no psychotomimetic action of their own. Finally, levodopa is reduced. Generally, a point is reached at which the anti-PD medications cannot be reduced without jeopardizing motor function. If psychosis persists at this point, then an antipsychotic is added. We first recommend a trial of quetiapine because of the ease of using it, beginning with 12.5 mg at bedtime, then increasing by 12.5 mg every 4-7 days until on 25 mg twice a day or 50 mg at night. Changes are then made as indicated. How quickly the increase is made varies with the individual case and depends on the side effects and the response. If the patient is not sedated, the increase can be made daily or every other day. If quetiapine is not tolerated or not effective, we recommend clozapine as the second-line choice. We begin clozapine at 6.25 mg at bedtime, and increase by the same amount every 4-7 days until psychosis remits or side effects occur. Most psychotic patients with PD can be treated with clozapine at bedtime only using doses of 50 mg or less. Should quetiapine and clozapine fail, olanzapine is our third-line choice, beginning with 2.5 mg. We suggest weekly increases of 2.5 mg until psychosis remits or parkinsonism worsens. Failure to respond to any antipsychotic is rare and should be treated either with electroconvulsive therapy or a more drastic reduction of anti-PD medications until the psychosis resolves. Levodopa is then cautiously restarted at a low dose and gradually increased. Additional atypical antipsychotics will be available soon, making this a work in transition.

Source: EMBASE

85. Worsening of motor features of parkinsonism with olanzapine.

Author(s) Molho ES, Factor SA

Citation: Movement Disorders, November 1999, vol./is. 14/6(1014-6), 0885-3185;0885-
Abstract: Clozapine is the current treatment of choice for drug-induced psychosis (DIP) occurring in Parkinson's disease. However, alternative medications have been sought because of the small but significant risk of agranulocytosis and the need for frequent blood testing. The new "atypical" antipsychotic olanzapine (OLZ) has recently been proposed as a safe and effective option for treating psychosis in this setting. To investigate this, we retrospectively evaluated all 12 of our patients treated with OLZ for DIP. Symptoms of psychosis were improved in nine of 12 patients, but nine of 12 patients also experienced worsening of motor functioning while on OLZ. The worsening was considered dramatic in six of these patients. Overall, there was no significant increase in levodopa doses on OLZ. Only one patient remained on OLZ at the time of the analysis. Nine patients were switched to alternative treatment for DIP. We conclude that although OLZ may improve symptoms of psychosis in parkinsonian patients, it can also worsen motor functioning. In some patients, the degree of motor worsening may be intolerable.

Source: Medline

86. Olanzapine-induced psychotic mania in bipolar schizoaffective disorder.

Author(s) Benazzi, Franco

Citation: The Canadian Journal of Psychiatry / La Revue canadienne de psychiatrie, August 1999, vol./is. 44/6(607-608), 0706-7437;1497-0015 (Aug 1999)

Abstract: Presents the case of 54-yr-old female with bipolar schizoaffective disorder who had an episode of psychotic mania induced by olanzapine. To treat her hypomania, risperidone was discontinued and olanzapine was started the following day. The S became irritable and agitated. She also developed logorrhea, delusions, and auditory hallucinations. Olanzapine was discontinued and risperidone was restarted. All symptoms had disappeared after 2-4 days after the discontinuation of olanzapine. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

Available in fulltext from Canadian Journal of Psychiatry at EBSCOhost

87. Olanzapine in an intensive care unit.

Author(s) Anand, H. S

Citation: The Canadian Journal of Psychiatry / La Revue canadienne de psychiatrie, May 1999, vol./is. 44/4(397), 0706-7437;1497-0015 (May 1999)

Abstract: Presents the case of a 64-yr-old male who was hospitalized with pancreatitis and cholelithiasis that required surgery. During the ordeal the S became increasingly depressed. Treatment with fluvoxamine and risperidone was at first successful, but then the patient became more irritable and angry. Olanzapine treatment was initiated, and his mental state improved. A short time later his cardiac status declined, requiring pacemaker treatment. The fluvoxamine was discontinued, and his mood declined rapidly. Fluvoxamine was reinstated, and the patient improved both physically and mentally. The use of olanzapine in agitated, somewhat psychotic states induced by medical conditions is not well documented. In this case it appeared that risperidone was extremely useful initially; its efficacy appeared to diminish with time. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

Available in fulltext from Canadian Journal of Psychiatry at EBSCOhost

88. A comparison of olanzapine with haloperidol in cannabis-induced psychotic
disorder: A double-blind randomized controlled trial.

Author(s) Berk, M, Brook, S, Trandafir, A. I

Citation: International Clinical Psychopharmacology, May 1999, vol./is. 14/3(177-180), 0268-1315;1473-5857 (May 1999)

Publication Date: May 1999

Abstract: Little controlled data exist on the treatment of substance induced psychotic disorders. In this study, 30 patients (aged 19-58 yrs) meeting DSM-IV criteria for cannabis induced psychotic disorder were randomly allocated to receive either olanzapine or haloperidol in a 4-week double-blind clinical trial. There were no significant outcome differences between the two groups on any of the primary outcome measures, the Brief Psychiatric Rating Scale, Clinical Global Impression (CGI) severity scale, or the CGI improvement scale. The haloperidol group however, developed significantly more extrapyramidal side-effects as measured by the Simpson Angus Scale. Significantly more biperidin was used for extrapyramidal side-effects in the haloperidol than in the olanzapine group. Olanzapine appears to be as effective as haloperidol in the treatment of cannabis induced psychotic disorder, but is associated with a lower rate of extrapyramidal side-effects. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Source: PsycINFO

89. Quetiapine for the treatment of drug-induced psychosis in Parkinson's disease.

Author(s) Fernandez HH, Friedman JH, Jacques C, Rosenfeld M

Citation: Movement Disorders, May 1999, vol./is. 14/3(484-7), 0885-3185;0885-3185 (1999 May)

Publication Date: May 1999

Abstract: Quetiapine is an atypical antipsychotic with clozapine-like pharmacology but without associated agranulocytosis. We report our complete experience with quetiapine for the treatment of drug-induced psychosis (DIP) in Parkinson's disease (PD). Thirty-five patients with PD and DIP aged 75 years (range, 58-89) with a mean PD duration of 8.4 years on an average of 427 mg levodopa per day received a mean dose of 40.6 mg quetiapine daily. Twenty of 24 neuroleptic-naive patients reported marked improvement of psychosis without a decline in motor function as assessed by the Unified Parkinson's Disease Rating Scale (UPDRS-motor). Ten patients had a baseline and 4-week follow-up assessment using the Mini-Mental Status Examination (MMSE) and Brief Psychiatric Rating Scale (BPRS). The improvement in BPRS score (32.6 versus 22.8) was clinically and statistically significant (p = 0.024). Three of 24 were unable to tolerate quetiapine because of orthostatic hypotension, headache, nausea, and persistence of hallucinations. One patient died of an unrelated cause. We also tried to switch 11 psychiatrically stable patients on clozapine (eight) and olanzapine (three). Five patients made this transition without a loss of effect as measured on BPRS and MMSE. Six did not (five on clozapine, one on olanzapine) because of confusion, erratic behavior, and increased hallucinations. No crossover failure had worsened PD except for increased tremor in one. Quetiapine is useful and well-tolerated as a first drug to treat DIP in PD but must be used cautiously to replace other atypical antipsychotic drugs.

Source: Medline

90. The role of atypical antipsychotics in the treatment of movement disorders

Author(s) Fernandez H.H., Friedman J.H.

Citation: CNS Drugs, 1999, vol./is. 11/6(467-483), 1172-7047 (1999)

Publication Date: 1999

Abstract: An atypical antipsychotic drug is loosely defined by its ability to produce an antipsychotic effect without inducing extrapyramidal symptoms (EPS). To date, 4 atypical antipsychotics have been released in the US: clozapine, quetiapine, olanzapine and risperidone, which are listed in decreasing order of ‘atypicality’ based on clinical and preclinical studies. While we await the outcome of trials with quetiapine on parkinsonian
patients (considered the most stringent test of the atypicality of a drug), clozapine remains the prototypic atypical antipsychotic drug. Disappointing reports of risperidone-induced parkinsonism raise questions about the atypical nature of this drug. Olanzapine appears to be intermediate between risperidone and clozapine in inducing EPS. Drug-induced psychosis in Parkinson's disease and antipsychotic-induced movement disorders in psychotic patients are the most common indications for an atypical antipsychotic in patients with movement disorders. In drug-induced psychosis in Parkinson's disease, the antiparkinsonians are first reduced until psychosis resolves. Unfortunately, motor function is often compromised as a result. The addition of an atypical antipsychotic drug, without altering the regimen of antiparkinsonians, often controls psychosis without compromising motor function. Depending on the atypical antipsychotic used, the dosage required may be substantially lower than that for schizophrenic patients. No treatment strategy has been proven to be clearly superior in suppressing antipsychotic-induced movement disorders such as tardive dyskinesia, tardive akathisia and dystonia. Nonetheless, a review of the available data strongly suggests that clozapine has substantially less risk of inducing tardive dyskinesia as compared with conventional antipsychotic agents. No case of tardive dyskinesia developing in patients who have taken clozapine as their only antipsychotic has yet been reported. Although there is evidence that clozapine may have an active therapeutic effect against pre-existing tardive dyskinesia, this remains inconclusive. Data on the use of clozapine for tremor in Parkinson's disease suggest significant benefit. Clozapine has also been reported to be useful in a variety of movement disorders including levodopa-induced dyskinesia, nocturnal akathisia and dystonia in Parkinson's disease, but the number of patients involved is small. No definitive conclusion on the role of atypical antipsychotic agents in other behavioural disorders such as depression, anxiety and sleep fragmentation in Parkinson's disease, as well in other movement disorders, can be made until well planned long term double-blind trials have been performed.

Source: EMBASE

91. Olanzapine-induced psychotic mania in bipolar schizoaffective disorder [6]

Author(s) Benazzi F.

Citation: Canadian Journal of Psychiatry, 1999, vol./is. 44/6(607-608), 0706-7437 (1999)

Publication Date: 1999

Source: EMBASE

Available in fulltext from Canadian Journal of Psychiatry at EBSCOhost

92. Substituting clozapine for olanzapine in psychiatrically stable parkinson's disease patients: Results of an open label pilot study

Author(s) Friedman J.H., Goldstein S., Jacques C.

Citation: Clinical Neuropharmacology, September 1998, vol./is. 21/5(285-288), 0362-5664 (September/October 1998)

Publication Date: September 1998

Abstract: Psychosis induced by the standard drugs for treating idiopathic Parkinson's disease (PD) occurs, in the long term, in approximately 5-8% of patients. Until the development of atypical antipsychotic drugs, treatment of this psychosis was extremely limited and unsatisfactory. Clozapine has been reported to be an effective and well-tolerated treatment for this problem. Clozapine, however, is a difficult drug to use as a result of the monitoring system mandated by the Food and Drug Administration because of the potential for agranulocytosis. Treatment with risperidone has been shown to be poorly-tolerated. This article reports on the authors' open label experience with the newest atypical antipsychotic, olanzapine, and the switching of stable patients from clozapine to olanzapine. Nine of twelve subjects were unable to make the transition because of worsened parkinsonism. Most subjects preferred taking clozapine, despite the onerous monitoring procedure.

Source: EMBASE
93. *Isoniazid-induced psychosis.*

**Author(s)** Alao AO, Yolles JC

**Citation:** Annals of Pharmacotherapy, September 1998, vol./is. 32/9(889-91), 1060-0280;1060-0280 (1998 Sep)

**Publication Date:** September 1998

**Abstract:** OBJECTIVE: To report a suspected case of isoniazid-induced psychosis in a 31-year-old woman.CASE SUMMARY: A 31-year-old white woman without a prior psychiatric history presented with psychotic symptoms suspected to be related to prophylactic treatment with isoniazid after she tested positive to a tuberculin (purified protein derivative) test. The psychotic symptoms resolved partially after isoniazid was discontinued and completely after treatment with olanzapine was begun. The patient remained symptom-free 11 months after discharge from the hospital.DISCUSSION: Cases of isoniazid-related psychiatric disorders reported in the literature include psychosis, obsessive-compulsive neurosis, and mania. With the increasing prevalence of tuberculosis in the US, more people are expected to receive treatment for tuberculosis. Pyridoxine deficiency may play a role in the pathogenesis of isoniazid-induced psychosis. Such deficiency states may be detected indirectly by measuring urinary metabolites of tryptophan.CONCLUSIONS: Clinicians should be aware of this adverse effect of isoniazid and that it may present with a broad clinical picture.

**Source:** Medline

94. *Conventional psychotropic-induced tremor extinguished by olanzapine.*

**Author(s)** Strauss AJ, Bailey RK, Dralle PW, Eschmann AJ, Wagner RB

**Citation:** American Journal of Psychiatry, August 1998, vol./is. 155/8(1132), 0002-953X;0002-953X (1998 Aug)

**Publication Date:** August 1998

**Source:** Medline

Available in fulltext at *American Journal of Psychiatry*; Notes: Username: ulhtlibraries/Password: POL_828094394

95. *Olanzapine in the treatment of dopaminomimetic psychosis in patients with Parkinson's disease*

**Author(s)** Wolters E.Ch., Jansen E.N.H., Tuynman-Qua H.G., Bergmans P.L.M.

**Citation:** Neurology, October 1996, vol./is. 47/4(1085-1087), 0028-3878 (October 1996)

**Publication Date:** October 1996

**Abstract:** We studied the effect of olanzapine (1 to 15 mg/d) in 15 nondemented parkinsonian patients with drug-induced psychosis. Psychotic symptoms decreased significantly during treatment, and there was no worsening of extrapyramidal symptoms. These results suggest that olanzapine is a well-tolerated and effective treatment for drug-induced psychosis in nondemented patients with Parkinson's disease.

**Source:** EMBASE

Available in fulltext from *Neurology* at the ULHT Library and Knowledge Services’ eJournal collection

**Published Research - Google Scholar**

*From the first 50 results…*

Open-label flexible-dose pilot study to evaluate the safety and tolerability of aripiprazole in
patients with psychosis associated with Parkinson's disease

JH Friedman, RM Berman, CG Goetz… - Movement …, 2006 - Wiley Online Library

... drug-induced psychosis in Parkinson's disease; hallucinations; delusions; atypical antipsychotic drugs; aripiprazole: Parkinson's ... antipsychotic drugs in patients with schizophrenia and primary psychoses is commonly ... than what is used for the treatment of psychosis in patients ...

Cited by 77 Related articles All 4 versions Cite

Olanzapine treatment for dopaminergic-induced hallucinations

WG Ondo, JK Levy, KD Vuong, C Hunter… - Movement …, 2002 - Wiley Online Library

... This use would require caution, because Manson and associates reported that olanzapine improved dyskinesia ... at the tested doses, for the chronic, uninterrupted management of drug-induced psychosis in PD. ... Psychotic symptoms in Parkinson's disease patients with dementia. ...

Cited by 103 Related articles All 4 versions Cite

Clozapine for the treatment of drug-induced psychosis in Parkinson's disease: Results of the 12 week open label extension in the PSYCLOPS trial

SA Factor, JH Friedman, MC Lannon… - Movement …, 2001 - Wiley Online Library

... Olanzapine in the treatment of hallucinations/ delusions in Parkinson's disease ... L, Sweitzer D, Yeung P, Jewart RD, Nemeroff C. Quetiapine improves psychotic symptoms associated ... Jacques C, Rosenfeld M. Quetiapine for the treatment of drug-induced psychosis in Parkinson's ...

Cited by 69 Related articles All 4 versions Cite

Olanzapine can worsen parkinsonism


... 1 Clozapine, which binds preferentially to D 4 receptors, 2 is a useful treatment for drug-induced psychosis in PD. 3 Olanzapine, another new neuroleptic, has been recently proposed as an effective and safe treatment for dopaminomimetic psychosis in PD. ...

Cited by 51 Related articles All 4 versions Cite

Olanzapine for the treatment of psychosis in patients with Parkinson's disease and dementia

L Marsh, C Lyketsos, SG Reich - Psychosomatics, 2001 - Elsevier

... Prevalence and clinical correlates of psychotic symptoms in Parkinson Disease: a community ... Low-dose clozapine for the treatment of drug-induced psychosis in Parkinson's disease. ... Olanzapine in the treatment of dopaminomimetic psychosis in patients with Parkinson's disease ...

Cited by 32 Related articles All 4 versions Cite

A placebo-controlled, double-blind study of the efficacy and safety of aripiprazole in patients with acute bipolar mania


Cited by 422 Related articles All 24 versions Cite

More

Aripiprazole in psychosis associated with Parkinson's disease


... 1,2,4,6. Aripiprazole is an agent for treating schizophrenia that has a novel pharmacological profile. ... Three years later, he developed memory impairment and psychosis. ... We found a remarkable improvement in all of his psychotic symptoms. ...

Cited by 28 Related articles All 21 versions Cite

More

Olanzapine and clozapine Comparative effects on motor function in hallucinating PD
and any prior history of a primary psychiatric illness including schizophrenia, psychotic depression, or be tested carefully for effects on motor function as well as psychotic symptoms before. Low dose clozapine for the treatment of drug-induced psychosis in Parkinson's disease. ... A randomized, open-label comparison of 2 switching strategies to aripiprazole treatment in patients with schizophrenia: add-on, wait, and tapering of previous ... Extrapyramidal symptoms-related side effects were evaluated biweekly using the Drug-Induced Extrapyramidal Symptoms ... to be safe and well tolerated without the worsening of psychotic symptoms and ... up-regulation, a net effect, in turn, may result in supersensitivity psychosis. ... Adverse events related to olanzapine. RR Conley, HY Meltzer - The Journal of clinical psychiatry, 1999 - europepmc.org to using the newer agents as preferred treatment for schizophrenia and related psychoses. ... Or filter your current search. Akathisia, Drug-Induced. Agents that control agitated psychotic ... They are used in SCHIZOPHRENIA; senile dementia; transient psychosis following surgery; or ... Psychosis in Parkinson's disease W Poewe - Movement disorders, 2003 - Wiley Online Library ... efficacy and safety of olanzapine specifically in the treatment of drug-induced psychosis in Parkinson's. Cholinesterase inhibitors, therefore, may be another treatment option for psychotic behaviour specifically ... future role of these agents regarding the treatment of psychosis in PD ... Atypical antipsychotics: CATIE study, drug-induced movement disorder and resulting iatrogenic psychiatric-like symptoms, supersensitivity rebound psychosis and ... G Chouinard, VA Chouinard - Psychotherapy and psychosomatics, 2008 - karger.com ... studies: the American CATIE study [5] and the European Schizophrenia Outpatient Health ... go to top of outline Supersensitivity Withdrawal Syndromes and Supersensitivity Rebound ...
Deterioration of parkinsonian symptoms following treatment of dopaminergic hallucinosis with olanzapine

J Rudolf, M Ghaemi, S Schmülling - European psychiatry, 1999 - Elsevier

... of dopaminergic psychosis, but due to the considerable risk of drug-induced leukopenia, the ... While the use of risperidone in dopaminergic psychosis was abandoned due to intolerable ... Due to the severity of the psychotic phenomena (optic hallucinations), an initial drug dose ...

Cited by 18 Related articles All 5 versions Cite

Management of psychosis in Parkinson's disease

EC Wolters, HW Berendse - Current opinion in neurology, 2001 - journals.lww.com

... often paranoid in type and mainly involve persecution, spousal infidelity or jealousy [9] . Hallucinations, at first often with preserved insight into the drug-induced aetiology, are ... Alternatively, they may be induced by late-life psychosis or, as a psychotic behavioural syndrome ...

Cited by 55 Related articles All 4 versions Cite

<table>
<thead>
<tr>
<th>Search Strategy - Databases</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>MEDLINE olanzapine.ti,ab</td>
<td>6251</td>
</tr>
<tr>
<td>MEDLINE Zyprexa.ti,ab</td>
<td>57</td>
</tr>
<tr>
<td>MEDLINE symbyax.ti,ab</td>
<td>10</td>
</tr>
<tr>
<td>MEDLINE 1 OR 2 OR 3</td>
<td>6259</td>
</tr>
<tr>
<td>MEDLINE (psychotic adj2 disorder*).ti,ab</td>
<td>5494</td>
</tr>
<tr>
<td>MEDLINE exp PSYCHOTIC DISORDERS/</td>
<td>38011</td>
</tr>
<tr>
<td>MEDLINE schizophren*.ti,ab</td>
<td>91042</td>
</tr>
<tr>
<td>MEDLINE (schizoaffective adj2 disorder*).ti,ab</td>
<td>3563</td>
</tr>
<tr>
<td>MEDLINE (delusional adj2 disorder*).ti,ab</td>
<td>709</td>
</tr>
<tr>
<td>MEDLINE (delusion* adj2 disorder*).ti,ab</td>
<td>815</td>
</tr>
<tr>
<td>MEDLINE exp SCHIZOPHRENIA/</td>
<td>85676</td>
</tr>
<tr>
<td>MEDLINE schizoaffective.ti,ab</td>
<td>4380</td>
</tr>
<tr>
<td>MEDLINE delusion*.ti,ab</td>
<td>7914</td>
</tr>
<tr>
<td>MEDLINE (psychosis OR psychoses).ti,ab</td>
<td>29857</td>
</tr>
<tr>
<td>MEDLINE abilify.ti,ab</td>
<td>52</td>
</tr>
<tr>
<td>MEDLINE aripiprazole.ti,ab</td>
<td>3251</td>
</tr>
<tr>
<td>MEDLINE 4 OR 15 OR 16</td>
<td>8678</td>
</tr>
<tr>
<td>MEDLINE 5 OR 6 OR 7 OR 8 OR 9 OR 10 OR 11 OR 12 OR 13 OR 14</td>
<td>150646</td>
</tr>
<tr>
<td>MEDLINE 17 AND 18</td>
<td>4913</td>
</tr>
<tr>
<td>MEDLINE 5 OR 6 OR 8 OR 10 OR 11</td>
<td>117154</td>
</tr>
<tr>
<td>MEDLINE 17 AND 20</td>
<td>3909</td>
</tr>
<tr>
<td>EMBASE 45 [Limit to: Publication Year 2008-Current and (Human Age Groups Adult 18 to 64 years or Aged 65+ years)]</td>
<td>1461</td>
</tr>
<tr>
<td>MEDLINE 7 OR 8 OR 11 OR 12</td>
<td>111718</td>
</tr>
<tr>
<td>MEDLINE 17 AND 47</td>
<td>4152</td>
</tr>
<tr>
<td>Database</td>
<td>Query</td>
</tr>
<tr>
<td>----------</td>
<td>-------</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>48 [Limit to: Publication Year 2008-Current and (Age Groups All Adult 19 plus years)]</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>&quot;drug induced psychos**&quot;.ti,ab</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>&quot;drug-induced psychos&quot;.ti,ab</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>&quot;drug-induced psychos**&quot;.ti,ab</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>drug-induc*.ti,ab</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>(drug* adj2 induc* adj2 psychos*).ti,ab</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>(53 adj2 18).ti,ab</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>55 OR 56</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>4 AND 57</td>
</tr>
<tr>
<td>EMBASE</td>
<td>olanzapine.ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>Zyprexa.ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>symbyax.ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>60 OR 62 OR 63</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(psychotic adj2 disorder*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>exp PSYCHOTIC DISORDERS/</td>
</tr>
<tr>
<td>EMBASE</td>
<td>schizophren*.ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(schizoaffective adj2 disorder*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(delusional adj2 disorder*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(delusion* adj2 disorder*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>exp SCHIZOPHRENIA/</td>
</tr>
<tr>
<td>EMBASE</td>
<td>schizoaffective.ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>delusion*.ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(psychosis OR psychoses).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>abilify.ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>aripiprazole.ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>65 OR 78 OR 79</td>
</tr>
<tr>
<td>EMBASE</td>
<td>67 OR 68 OR 69 OR 70 OR 71 OR 73 OR 74 OR 75 OR 76 OR 77</td>
</tr>
<tr>
<td>EMBASE</td>
<td>80 AND 81</td>
</tr>
<tr>
<td>EMBASE</td>
<td>67 OR 68 OR 70 OR 73 OR 74</td>
</tr>
<tr>
<td>EMBASE</td>
<td>80 AND 83</td>
</tr>
<tr>
<td>EMBASE</td>
<td>45 [Limit to: Publication Year 2008-Current and (Human Age Groups Adult 18 to 64 years or Aged 65+ years)]</td>
</tr>
<tr>
<td>EMBASE</td>
<td>69 OR 70 OR 74 OR 75</td>
</tr>
<tr>
<td>EMBASE</td>
<td>80 AND 86</td>
</tr>
<tr>
<td>EMBASE</td>
<td>87 [Limit to: Publication Year 2008-Current and (Age Groups All Adult 19 plus years)]</td>
</tr>
<tr>
<td>EMBASE</td>
<td>&quot;drug induced psychos**&quot;.ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>&quot;drug-induced psychos&quot;.ti,ab</td>
</tr>
<tr>
<td>Database</td>
<td>Query</td>
</tr>
<tr>
<td>------------</td>
<td>----------------------------------------------------------------------</td>
</tr>
<tr>
<td>EMBASE</td>
<td>&quot;drug-induced psychos**&quot;.ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>drug-induc*.ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(drug* adj2 induc* adj2 psychos*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(53 adj2 18).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>93 OR 94</td>
</tr>
<tr>
<td>EMBASE</td>
<td>65 AND 95</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>olanzapine.ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>Zyprexa.ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>symbyax.ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>98 OR 100 OR 101</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(psychotic adj2 disorder*).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>exp PSYCHOTIC DISORDERS/</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>schizophren*.ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(schizoaffective adj2 disorder*).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(delusional adj2 disorder*).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(delusion* adj2 disorder*).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>exp SCHIZOPHRENIA/</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>schizoaffective.ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>delusion*.ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(psychosis OR psychoses).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>abilify.ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>aripiprazole.ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>103 OR 116 OR 117</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>105 OR 106 OR 107 OR 108 OR 109 OR 111 OR 112 OR 113 OR 114 OR 115</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>118 AND 119</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>105 OR 106 OR 108 OR 111 OR 112</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>118 AND 121</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>107 OR 108 OR 112 OR 113</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>118 AND 123</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>124 [Limit to: Publication Year 2008-Current and (Age Groups All Adult 19 plus years)]</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>&quot;drug induced psychos**&quot;.ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>&quot;drug-induced psychos&quot;.ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>&quot;drug-induced psychos&quot;.ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>drug-induc*.ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(drug* adj2 induc* adj2 psychos*).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(53 adj2 18).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>130 OR 131</td>
</tr>
<tr>
<td>Database</td>
<td>Query</td>
</tr>
<tr>
<td>-------------------</td>
<td>-----------------------------------------------------------------------</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(medicine* adj2 induc*).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(medicine* adj2 induc* adj2 psychos*).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(substance* adj2 induc*).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(pharamcol* adj2 induc*).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(medication* adj2 induc*).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(stimulant* adj2 induc*).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(stimulant* adj2 psychos*).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>134 OR 136 OR 137 OR 138 OR 139</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(119 adj2 141).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>142 and 103</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>140 OR 141</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>118 AND 144</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(medicine* adj2 induc*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(medicine* adj2 induc* adj2 psychos*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(substance* adj2 induc*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(pharamcol* adj2 induc*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(medication* adj2 induc*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(stimulant* adj2 induc*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(stimulant* adj2 psychos*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>146 OR 148 OR 149 OR 150 OR 151</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(119 adj2 141).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>154 and 103</td>
</tr>
<tr>
<td>EMBASE</td>
<td>152 OR 153</td>
</tr>
<tr>
<td>EMBASE</td>
<td>118 AND 156</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(pharmacol* adj2 induc*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>146 OR 148 OR 149 OR 150 OR 151 OR 158</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(81 adj2 159).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>80 and 160</td>
</tr>
<tr>
<td>EMBASE</td>
<td>80 and 153</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(drug* adj2 induc*).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>159 OR 163</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(81 adj2 164).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>165 and 80</td>
</tr>
<tr>
<td>EMBASE</td>
<td>81 and 164</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(81 adj0 164).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>80 AND 81 AND 164</td>
</tr>
<tr>
<td>EMBASE</td>
<td>((medicine* OR drug* OR medication* OR pharmacol* OR substance* OR stimulant*) adj2 (psycho* OR schizo* OR</td>
</tr>
<tr>
<td>Database</td>
<td>Search String</td>
</tr>
<tr>
<td>-----------</td>
<td>-------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(((medicine* OR drug* OR medication* OR pharmacol* OR substance* OR stimulant*) adj0 induc*) adj2 (psycho* OR schizo* OR delusion*)).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>171 and 80</td>
</tr>
<tr>
<td>EMBASE</td>
<td>exp ADVERSE DRUG REACTION/</td>
</tr>
<tr>
<td>EMBASE</td>
<td>68 OR 74</td>
</tr>
<tr>
<td>EMBASE</td>
<td>173 AND 174</td>
</tr>
<tr>
<td>EMBASE</td>
<td>80 and 175</td>
</tr>
<tr>
<td>EMBASE</td>
<td>(induc* adj2 (psycho* OR schizo* OR delusion*)).ti,ab</td>
</tr>
<tr>
<td>EMBASE</td>
<td>80 and 177</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>(((medicine* OR drug* OR medication* OR pharmacol* OR substance* OR stimulant*) adj0 induc*) adj2 (psycho* OR schizo* OR delusion*)).ti,ab</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>179 and 80</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>(induc* adj2 (psycho* OR schizo* OR delusion*)).ti,ab</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>80 and 181</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>17 AND 179</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>17 and 181</td>
</tr>
<tr>
<td>MEDLINE</td>
<td>180 OR 184</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(((medicine* OR drug* OR medication* OR pharmacol* OR substance* OR stimulant*) adj0 induc*) adj2 (psycho* OR schizo* OR delusion*)).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>(induc* adj2 (psycho* OR schizo* OR delusion*)).ti,ab</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>17 AND 186</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>17 and 187</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>118 and 186</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>118 and 187</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>190 OR 191</td>
</tr>
</tbody>
</table>