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**Management details**

Management of patients presenting with peripheral microemboli in the upper or lower limbs.

**Resources searched**

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

**Database search terms:** peripheral* adj2 microembol*; (small OR micro) adj2 (embol* OR “blood clot” OR clot*); microembol*; microembol* adj2 (minor OR peripheral OR outly* OR marginal*); minor; peripheral; outly*; marginal*; exp EMBOLISM; small; micro; min*; tiny; limb*; exp LOWER EXTERMITY; exp UPPER EXTERMITY; arm*; leg*; ankle* knee*; foot; feet; toe*; thigh*; calf; calves; shin*; hand* finger*; wrist*; forearm*; elbow*; extremit*; exp MICROEMBOLISM;

**Evidence search string(s):** peripheral microemboli (limb* OR leg* OR arm* OR hand* OR wrist* OR foot OR feet OR ankle* OR knee* OR elbow* OR thigh* OR calf OR calves OR shin* OR toe* OR finger* OR digit*)

(Peripheral OR minor OR marginal) (small OR micro OR minute OR miniscule OR tiny) (embolus OR emboli) (limb OR limbs OR leg OR legs OR arm OR arms OR hands OR thighs OR feet)

**Google search string(s):** (Peripheral OR minor OR marginal) (microembolus OR microemboli) (limb OR limbs OR leg OR legs OR arm OR arms OR hands OR thighs OR feet)

(Peripheral OR minor OR marginal) (small OR micro OR minute OR miniscule OR tiny) (embolus OR emboli) (limb OR limbs OR leg OR legs OR arm OR arms OR hands OR thighs OR feet)
Summary

There is some research on this topic, but not a huge amount. A lot of it relates to the effect of microemboli occurring in the limbs and travelling through the blood to affect other areas of the body, such as the brain, heart and lungs. There are also quite a few papers on detection, but limited research on treatment, unless the treatment options for emboli would also apply to microemboli.

Guidelines and Policy

None found.

Evidence-based reviews

None found.

Published research – Databases

1. Blue toe syndrome - A description of positive evolutions through patient adapted therapy

Author(s) Weise M., Muhlberg K. S., Pfeiffer D.

Citation: Vasa - Journal of Vascular Diseases, September 2013, vol./is. 42/(97) (12 Sep 2013)

Publication Date: September 2013

Abstract: Introduction: The blue toe syndrome is a skin manifestation related to diverse diseases characterized by either decreased arterial flow e.g. embolism/thrombosis, vasoconstriction and inflammation or abnormal composition of circulating blood with consecutive end-organ damage. As it is a rare and often misdiagnosed sickness standardized therapy guidelines are non-existent yet. The following cases present blue toe syndromes in multimorbid elderly patients caused by microembolization and their successful therapeutic approach. Case descriptions: We present two different patients with blue toe syndrome who obtained an individual therapy according to their concomitant diseases. 1st case: A 77-year old multimorbid male presented to us with a bilateral very painful blue toe syndrome. Two months earlier he had suffered a myocardial infarction treated by PCI (drug-eluting stent). His medical history consisted furthermore of arterial hypertension, non significant peripheral macroangiopathy and benign prostatic hyperplasia. Laboratory findings demonstrated newly occurred renal failure (GFR 15 ml/min/1.73 m2) and proteinuria. Cholesterol embolization was histopathologically confirmed in renal biopsy. It was assumed that peripheral perfusion deficit was caused by cholesterol embolization, too. Due to the recent myocardial infarction application of prostaglandin derivatives was not possible. Under treatment with prednisolone and statin, pain and blue toe coloration could be reduced and renal function improved markedly (GFR 28 ml/min/1.73m2). This status could be hold even after end of therapy with prednisolone. 2nd case: A 64-year old female discovered painful blue coloration and swelling of all toes 14 days before referral. Except for an arterial hypertension and nicotine abuse there were no pre-existing conditions. Duplex sonography and angiography could not demonstrate microembolization or peripheral stenosis, but abundant atherosclerotic formations in pelvic and femoral vessels. We administered alprostadil intravenously. Since thrombotic microembolization could not been ruled out surely (atrial fibrillation never was notified), the patient further was treated with anticoagulant therapy for three months, then continuing with antiplatelet agents as well as statin. The livedo reticularis disappeared completely over time and optical pulse oxymetry showed an increased arterial perfusion. Discussion: Until now reliable therapies of blue toe syndrome do not exist mostly for the rareness of this syndrome causes the lack of controlled randomized studies. Nevertheless all treatments search for an improvement of rheology. Statin and antiplatelet agents are recommended after case-series and expert opinions but the use of anticoagulant therapy is controversial due to its possible impact. These cases illustrate that medical treatment with different starting points can lead to very
satisfactory results if it is adapted to the patient, his medical history and the assumed agent(s). Follow-up and re-evaluation of therapy is highly important to prevent end-organ damage and to ensure individual outcome.

**Source:** EMBASE

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2. Evaluating embolic protection strategies in a consecutive group of 55 complex atherectomy procedures

**Author(s)** Makam P.

**Citation:** EuroIntervention, May 2013, vol./is. 9/(65), 1774-024X (May 2013)

**Publication Date:** May 2013

**Abstract:** Aims: Evaluate dual protection strategy (the concurrent use of SpiderFX Embolic Protection Device (Covidien/ev3, US) and Proteus balloon which has debris capturing capability (Angioslide, Israel) vs. mono protection strategy, which uses the latter only. In a set of lower limb atherectomy procedures we compared (i) the embolic load in various lesion types and (ii) in different atherectomy techniques; and (iii) debris capture effectiveness. Methods and results: The mean number of particles in the total embolic shower was 269±455; mean aggregated surface area was 22±20 mm². The majority of particles were captured by Proteus (395±524 particles, 27±21 mm²) and a small fraction by SpiderFX (36±41 particles, 12±12 mm²), p<0.001. A similar distribution was found for the various subgroups of lesions analysed. Embolic load by lesion type: Restenotic and thrombotic lesions produced in total more than double the number of particles and surface area than non-thrombotic and de novo lesions (p<0.03). Highly calcified lesions yielded 42-44% more debris particles compared with low-grade calcification lesions, although their surface area was approximately the same. High grade stenotic lesions (75-95%) produced more (33%) debris than occluded lesions in terms of surface area, which was spread among fewer particles (p<0.079). Procedures done on TASC II D lesions produced approximately double the surface area of debris than TASC II B and C lesions, spread between 30% and 38% more particles, respectively (p<0.002). Embolic load by procedure type: Directional atherectomy produced in total the largest number of particles, for which Proteus accounts for 97% (p<0.001). Laser atherectomy produced 36% more debris in terms of surface area, spread over 24% less particles than directional atherectomy (p<0.011). In Laser atherectomy a somewhat larger proportion of particles were captured by SpiderFX compared to its performances in other procedures. Orbital atherectomy yielded the least debris of all procedure types in terms of aggregated surface area (p<0.0032). All this debris was captured by Proteus, as SpiderFX cannot be used with orbital atherectomy. Small particles: Debris was comprised of large (surface area >0.3 mm²) and small particles, the latter accounting for up to 50% of the total aggregate surface area. Nevertheless, while Proteus captured large and small particles fairly evenly, small particles were largely missed by SpiderFX (p<0.001). Conclusions: This study concurs with previous publications on the high embolic load generated in complex atherectomy procedures. Sub analysis unveils the importance of addressing the full range of particle sizes, both macro- and, previously disregarded, micro-emboli. Dual protection strategy, composed of SpiderFX and Proteus as complementary means, is an effective measure in capturing large quantities of embolic matter.

**Source:** EMBASE

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3. Initial experience with the 5 x 300-mm Proteus embolic capture angioplasty balloon in the treatment of peripheral vascular disease.

**Author(s)** Zeller T, Schmidt A, Rastan A, Noory E, Sixt S, Scheinert D

**Citation:** Journal of Endovascular Therapy, December 2012, vol./is. 19/6(826-33), 1526-6028;1545-1550 (2012 Dec)

**Publication Date:** December 2012

**Abstract:** PURPOSE: To describe the use of the 5 x 300-mm Proteus embolic capture angioplasty (ECA) balloon catheter to reduce embolic burden in complex TASC II
(TransAtlantic Inter-Society Consensus) C and D femoropopliteal interventions. METHODS: A non-randomized safety and feasibility study was conducted at 2 centers enrolling 15 subjects (9 women; mean age 72.5 + 9.5 years, range 53-85) suffering from Rutherford-Becker category 2 to 4 occlusive disease. Of the 20 lesions in 15 limbs, 16 were TASC II D and 4 were TASC II C. Average baseline stenosis was 95% + 12%; 16 lesions were totally occluded. Half of the lesions were de novo, 5 were restenotic, and 5 were in-stent stenoses. Average lesion length was 284 + 50 mm. In addition to using the ECA device, 18 of the target lesions were treated with stents and 4 with rotational thrombectomy devices. Distal angiography was performed before and after use of the ECA device to locate any periprocedural embolic events. RESULTS: Procedural success was achieved in 100% lesions. The ECA balloon was used for predilation in 11 lesions and for postdilation in 9. No distal embolization or flow-limiting vessel dissections were observed despite the complex nature of the cases. Three non-device-related complications were reported (pseudoaneurysm, myocardial infarction, acute renal failure) and resolved without sequelae within 30 days. Analysis of the particles recovered from 5 ECA balloons demonstrated a mean 257 + 185 particles, with a mean major axial dimension of 0.54 + 0.04 mm (range 0.11-7.54). There were a mean 7.67 + 6.03 particles >2 mm in diameter, and all samples contained 1 to 3 particles >4 mm in diameter. CONCLUSION: In this small series, the 5 x 300-mm ECA embolic capture balloon catheter was an effective tool for avoiding embolic events in long peripheral lesions, with a good safety profile. The device might be considered as part of routine clinical practice for complex TASC II C/D femoropopliteal lesions.

Source: Medline

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Author(s) Sawalhi S.I., Hamad H.

Citation: Journal of Taibah University Medical Sciences, August 2012, vol./is. 7/1(41-44), 1658-3612 (August 2012)

Publication Date: August 2012

Abstract: 'Blue toe syndrome' presents as a cyanotic toe caused by fibrino-platelet microemboli occlusion of the small digital arteries, with palpable pedal pulses originating from a proximal ath-eromatous source (cardiac, aortic, femoral or popliteal aneurysm). We report a case in a 43-year-old woman with a history of hypertension and diabetes. She was taking warfarin for extensive right deep-vein thrombosis 6 months before her current admission. She presented with sudden onset of severe pain, paraesthesia and bluish discolouration of the right big, second and third toes, in spite of palpable distal pulses. Computerized tomography angiography revealed an atheromatous plaque causing 70% stenosis of the infra-renal aorta. We therefore performed aortotomy, thrombectomy and endartectomy. Blue toe syndrome is of clinical importance in etiology, diagnosis and treatment. Our case had blue toe syndrome despite anticoagulant therapy. 2012 Taibah University.

Source: EMBASE

5. Epithelioid hemangioendothelioma as a rare cause of blue toe syndrome

Author(s) Heldenberg E., Rabin I., Cheyn D., Lorber J., Elkabetz E., Sandbank J., Bass A.

Citation: Journal of Vascular Surgery, September 2011, vol./is. 54/3(854-856), 0741-5214;1097-6809 (September 2011)

Publication Date: September 2011
**Abstract:** Blue toe syndrome (BTS), is a well-known entity of toe gangrene and rest pain secondary to micro emboli lodged within the digital arteries. BTS among young patients should alert physicians to look for causes such as trauma, connective tissue disease, hypercoagulability state, and others. We hereby describe a 32-year-old female with right BTS. A mass obstructing 80% of the right popliteal artery lumen was the source of embolism. The histologic results of the replaced arterial segment revealed a thrombus on top of epithelioid hemangioendothelioma. This is the first description of the association between primary vascular tumor and BTS. 2011 Society for Vascular Surgery.

**Source:** EMBASE

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6. Epithelioid hemangioendothelioma as a rare cause of blue toe syndrome.

**Author(s)** Heldenberg E, Rabin I, Cheyn D, Lorber J, Elkabetz E, Sandbank J, Bass A

**Citation:** Journal of Vascular Surgery, September 2011, vol./is. 54/3(854-6), 0741-5214;1097-6809 (2011 Sep)

**Publication Date:** September 2011

**Abstract:** Blue toe syndrome (BTS), is a well-known entity of toe gangrene and rest pain secondary to micro emboli lodged within the digital arteries. BTS among young patients should alert physicians to look for causes such as trauma, connective tissue disease, hypercoagulability state, and others. We hereby describe a 32-year-old female with right BTS. A mass obstructing 80% of the right popliteal artery lumen was the source of embolism. The histologic results of the replaced arterial segment revealed a thrombus on top of epithelioid hemangioendothelioma. This is the first description of the association between primary vascular tumor and BTS. Copyright 2011 Society for Vascular Surgery. Published by Mosby, Inc. All rights reserved.

**Source:** Medline

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7. Getting to the heart of strokes

**Author(s)** Gogia N., Kanuri S., Hunter C.

**Citation:** Journal of General Internal Medicine, May 2011, vol./is. 26/(S523-S524), 0884-8734 (May 2011)

**Publication Date:** May 2011

**Abstract:** LEARNING OBJECTIVES: 1. Recognize the association between migraines, cryptogenic stroke (CS) and Patent Foramen Ovale (PFO) 2. Management of cryptogenic stroke associated with PFO. CASE INFORMATION: A 41 year old Caucasian female presented to the emergency room with three day duration of worsening migraine headaches, dizziness, nausea and vertigo. She has a history of typical migraine headaches with visual aura for the last twenty years. Home medications include progesterone only contraceptives. Neurologic examination revealed left facial droop with associated sensory loss and negative gag reflex. Upper and lower extremity strength, sensation and deep tendon reflexes were intact bilaterally. Heart and lung examination were normal. She received aspirin after a negative brain CT scan. Subsequently an MRI revealed infarct in the territory supplied by the left Postero-inferior cerebellar artery. MRA revealed no significant occlusive lesions. TEE revealed a PFO and atrial septal aneurysm (ASA). Lower
extremity dopplers were negative for DVT. Her hospital course was uneventful; she was discharged to home on full dose aspirin. On subsequent follow up she opted for elective percutaneous closure. Literature search revealed an association of cryptogenic stroke with PFO and migraine with aura which were interestingly present in our patient.

IMPLICATIONS/DISCUSSION: Cryptogenic strokes occur in patients less than 55 years without identifiable etiology and constitute 20% of all ischemic strokes. A higher prevalence of PFO (40-50%) was noted in these patients as compared to 20% in the general population. Small emboli can travel from legs to the right atrium across the PFO and travel to the brain and cause a stroke during straining activities. The existence of this mechanism was documented in studies by the detection of thrombus lodged in the PFO in patients with embolic events. Interestingly it was noted that patients with migraine and associated aura are four times more likely to have a PFO than the general population. It is unclear if there is a causal relationship or mere coexistence. One hypothesis suggests that passage of blood directly from the right to left atrium, bypassing the lungs, allows higher concentrations of serotonin, nitric oxide, kinins or other migraine precipitating chemicals to reach the brain and trigger migraine attacks. In patients with cryptogenic strokes due to PFO, the annual rate of recurrence is reported to be between 1.5-12%. The size of the septal separation seen on TEE, presence of atrial septal aneurysm contributes to the increased risk. The four major treatment modalities include medical therapy (anti platelets, anti coagulants), percutaneous device closure and surgical closure. As per the ACC/AHA recommendations, for patients with an ischemic stroke or TIA and a PFO, antiplatelet therapy is reasonable to prevent a recurrent event. Warfarin is indicated in high-risk patients with underlying hypercoagulable states or history of deep vein thrombosis. PFO closure may be considered for patients with recurrent cryptogenic stroke despite optimal medical therapy. The optimal management remains unclear due to the lack of randomized trials, which are currently in progress.

Source: EMBASE

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8. When it all goes badly wrong - What do you do?

Author(s) Kessel D.O.

Citation: CardioVascular and Interventional Radiology, September 2010, vol./is. 33/(65-66), 0174-1551 (September 2010)

Publication Date: September 2010

Abstract: Endovascular management of acute severe lower limb ischaemia using angioplasty, stenting and thrombolytic are all associated with complications. Fortunately, most are minor with minimal sequelae. A small proportion of cases go badly wrong. Serious complications are life-and limb-threatening and require immediate recognition and
Development of a large haematoma can make further arterial compression ineffective due to a combination of local pain and inability to feel the pulse. When a haematoma develops, it is best to start forming contingency plans straight away. Explain what is happening to the patients and reassure them. Ask how they feel and check the pulse and blood pressure. If the patient is hypotensive, consider whether this might be a vasovagal attack (hypotension and bradycardia). Remember that trends in physiological measurements are more useful than an isolated value; a hypertensive patient may become normotensive as a result of hypovolaemia. Make sure that there is venous access in case fluid resuscitation or transfusion is required. If the patient has not had blood taken for blood grouping, now is a good time to send a specimen. Consider checking/reversing anticoagulation if heparin has been administered. The size of the haematoma should be marked on the skin as a baseline to allow comparison with later observations so that increase in size will be recognised. Continue puncture site compression for at least another 10-15 minutes before checking again. If there is any suggestion of ongoing bleeding, i.e. physiological deterioration (increasing pulse and falling blood pressure), restlessness, yawning, and sweating, then urgent resuscitation is needed. Call the vascular surgeon. If the patient remains stable, then arrange immediate CT to demonstrate whether there is active bleeding and if so the exact location; this is important if the bleeding is in the pelvis. If the patient is becoming unstable despite fluid resuscitation, then consider placing an occlusion balloon into the aorta or iliac artery from the contralateral approach. Controlling the inflow will allow the patient to be stabilized and further angiography to be performed. A decision can then be made as to whether to treat the bleeding by surgery or endovascular means. Stent grafts are usually reserved for cases when the puncture site is in the external iliac artery.

Treatment related: The principle treatment related problems are dissection and arterial rupture. Dissection is a necessary part of angioplasty related to plaque rupture. There are of course degrees. Flow limiting dissection requires attention and is usually simply managed. The first step is to try prolonged low pressure balloon inflation in the hope that this sticks the flap back against the artery wall; surprisingly, this low technology solution often works! If this fails, consider stenting the dissection; this should only be used in locations where a stent would normally be considered, i.e. avoid points of flexion. If the situation is not suitable for stenting, assess the limb; it may be no worse than at the start of the procedure. If so, then stopping is usually the wisest option. If there is significant clinical deterioration, then notify the vascular surgeon and assess the patient together to plan further treatment. This is usually surgery but sometimes it is felt more appropriate to stent. If you are stenting at a point of flexion, then use a self-expanding stent. At present, there is no design which is proven immune to fracture. Deep dissections are a grey area. A simple way of thinking about them is, are they flow limiting? Was the angioplasty associated with significant pain? Is there abnormal flow in the dissection? Is there any extravasation of contrast (rupture)? If any of these is the case, then additional therapy is needed. Unless there is rupture, simple stenting is usually the solution. Arterial rupture is normally but not always heralded by severe pain during angioplasty. It is most likely to occur in small heavily calcified vessels particularly if extraluminal recanalization has been performed. Trying to prevent rupture is the reason for asking patients to report any pain during balloon inflation. Rupture is particularly likely if pain persists or worsens after the balloon is deflated. If rupture is a possibility, then be prepared to inflate the angioplasty balloon proximal to the rupture or across the injury to tamponade the leak. The staples of management are balloon occlusion to stop haemorrhage and stabilize the patient followed by definitive surgery or stent grafting. Remote: There are two categories of remote complication, those directly related to the treatment and those related to other factors such as contrast media or underlying conditions, e.g. myocardial ischaemia. These latter problems will not be considered further. Remote complications related to treatment of underlying stenosis or occlusion is almost always due to distal (or contralateral) embolization. This is said to occur in 1-4% of cases. Small emboli into branch vessels are of little clinical relevance. Emboli are important if they occlude important runoff vessels. Abrupt occlusion of the single runoff vessel may lead to acute ischaemia which mandates therapy. Occlusion of one of several runoff vessels needs to be considered on merit. For example, a small embolus into the
If the peroneal was the dominant vessel, then the situation would be different and attempts to aspirate or lyse the embolus would be appropriate. In the event of a large embolus, then the options are for surgical embolectomy or stenting. Learning points: - Serious complications will sometimes occur even in the best hands, be alert and prepared to manage them.
- Be honest with the patient and explain what is happening and keep a clear record of events.
- Resuscitate the patient if they are haemodynamically unstable.
- Do not hesitate to use an occlusion balloon.
- Do not hesitate to ask for surgical assistance.

Source: EMBASE

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9. Aortic aneurysm and orchitis due to Wegener's granulomatosis.

Author(s) Minnee RC, van den Berk GE, Groeneveld JO, van Dijk J, Turkcan K, Visser MJ, Vahl AC

Citation: Annals of Vascular Surgery, November 2009, vol./is. 23/6(786.e15-9), 0890-5096;1615-5947 (2009 Nov-Dec)

Publication Date: November 2009

Abstract: We present a patient with Wegener's granulomatosis (WG) with involvement of the abdominal aorta, testis, peripheral nerve system, and skin. A 51-year-old man presented at our outpatient clinic with lower back pain. He had a history of smoking, hypertension, and an embryonal carcinoma of the left testis, treated 13 years ago with orchidectomy and chemotherapy. One month earlier, he underwent a partial orchidectomy of the right testis due to testicular swelling. Abdominal computed tomography showed a 3.8 cm wide aneurysm of the distal part of the aorta with inflammation. One week later he was admitted to the hospital with numbness of his hands and feet. Physical examination showed signs of peripheral microemboli. Serological laboratory tests revealed elevated antineutrophil cytoplasmic antibody titers with positive reactions against proteinase-3, indicating Wegener's disease. The chest X-ray was normal. Pathological examination of the right testis showed necrotizing vasculitis of a small artery. He was treated with cyclophosphamide and prednisolone. WG with extrapulmonary involvement occurs infrequently, and reports of manifestations of WG in aorta, testis, the peripheral nerve system, and skin are even more uncommon. Small- and medium-vessel vasculitis can precede large-vessel vasculitis or occur in the absence of small-vessel involvement. Therefore, WG should be included in the work-up of large-vessel vasculitis, which can give rise to periaortic inflammation.

Source: Medline


Author(s) McBane RD, Hodge DO, Wysokinski WE

Citation: Thrombosis & Haemostasis, May 2008, vol./is. 99/5(951-5), 0340-6245;0340-6245 (2008 May)

Publication Date: May 2008

Abstract: Although infrequent, embolic occlusion to non-cerebral arteries may result in limb loss, organ failure, and death. The aim of this study was to define clinical and echocardiographic characteristics determining thromboembolism destination in non-valvular atrial fibrillation. An inception cohort of individuals (n=72) were identified with incident peripheral embolism in the setting of non-valvular atrial fibrillation (1995-2005). A randomly selected group of atrial fibrillation related stroke patients (n=100) were identified for comparison. Arteries of the extremities were the most common site of embolism (85%);
lower extremity involvement was twice as common compared with the upper extremity. Clinical features distinguishing peripheral embolism from stroke included age>75, heart failure and hypertension. Severe left ventricular dysfunction, spontaneous echo contrast and left atrial thrombus were 2-3 fold more common in peripheral embolism patients. Mean CHADS-2 scores were low and comparable for both groups. By multivariate analysis, age>5 years (hazard ratio [HR] 2.3, 95% confidence interval [CI] 1.3-3.9; p=0.05) was predictive of peripheral embolism. After adjustment for age>75 years, severe left atrial enlargement (HR 1.8, 95% CI 0.99-3.1; p=0.055) and CHADS score (HR 1.2, 95% CI 0.99-1.6; p=0.06) were of borderline significance. In conclusion, several clinical and echocardiographic measures distinguish the clinical presentation of thromboembolism in non-valvular atrial fibrillation. Small emboli are destined to lodge in the cerebral circulation as a result of hydrodynamic, anatomic, and physical factors. Advanced age, atrial enlargement and other co-morbidities may increase the propensity for the formation of larger thrombi which may bypass the carotid orifice merely as a function of size.

Source: Medline

11. Intermittent claudication secondary to a traumatic arteriovenous fistula

**Author(s)** Kotelis D., Klemm K., Teng-Kobligk H.V., Allenberg J.-R., Bockler D.

**Citation:** Vasa - Journal of Vascular Diseases, November 2007, vol./is. 36/4(285-287), 0301-1526 (November 2007)

**Publication Date:** November 2007

**Abstract:** Arteriovenous fistula (AVF) formation is a recognized complication of arterial trauma. A 63-year-old man with no known risk factors for atheroma and a history of a 20-year delay in the diagnosis and treatment of a traumatic AVF presented with right calf claudication (maximal walking distance = 150 m). A duplex ultrasound scan and a magnetic resonance angiography showed an aneurysmatic dilatation and partial thrombosis of the infrarenal aorta and the right leg feeding arteries. Selective angiography revealed a complete occlusion of the right popliteal artery in the segment 2 and the infrapopliteal arteries with good collateral formation in the lower leg. At this time, there is no indication for infragenual arterial revascularisation in this patient. Instead, therapy consists of exercise training and antiplatelet therapy. We concluded, that peripheral microembolism from the aneurysmatic aorta, iliac or femoral arteries is most probably the reason for the popliteal occlusion in this patient, representing a rare, nonatherosclerotic cause of claudication. Verlag Hans Huber, Hogrefe AG, 2007.

**Source:** EMBASE

12. Detection of peripheral microemboli through collateral circulation by Doppler ultrasound monitoring-report of 2 cases.

**Author(s)** Kudo T, Inoue Y, Nakamura H, Hirokawa M, Sugano N, Iwai T

**Citation:** Vascular & Endovascular Surgery, January 2005, vol./is. 39/1(103-8), 1538-5744;1538-5744 (2005 Jan-Feb)

**Publication Date:** January 2005

**Abstract:** It is possible for a proximal arterial source to lead to distal atheroembolism even in the presence of chronic occlusive disease. However, no monitoring technique has been established regarding detection of peripheral emboli through the collateral circulation in the lower limbs. We report a 60-year-old woman and a 73-year-old man with iliac stenosis and complete occlusion of the ipsilateral superficial femoral artery in whom Doppler ultrasound successfully detected microembolic signals (MES) at the tibioperoneal trunk during percutaneous transluminal angioplasty (PTA) and stent placement. By means of continuous Doppler ultrasound monitoring, 29 MES were successfully detected immediately after PTA or stent placement (MESp) and 64 MES were detected immediately after the contrast medium administration (MESc). MESc generated significantly higher intensities (median 28, range 7 to 38) as opposed to MESp (median 21, range 9 to 35, p = 0.017). In addition, the intensity of MES after prestent PTA (n = 8, 25 dB, 12-35 dB) and stenting (n = 18, 22 dB, 9-35 dB) was significantly higher than that of MES after poststen PTA (n = 3, 13 dB, range; 5-16 dB), respectively (p = 0.041, p = 0.034). Iliac PTA and stent placement were
successful. Ankle/brachial pressure index and the symptoms improved in both patients, who showed no embolic symptoms after the procedure. This study suggested that it was possible to detect peripheral microemboli through the collateral circulation by Doppler ultrasound monitoring and that this technique would be helpful to investigate the mechanism of embolization in patients with PTA and stent placement.

Source: Medline
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Available in fulltext from Vascular and Endovascular Surgery at Free Access Content

13. Does patent foramen ovale promote cryptogenic stroke and migraine headache?.

Author(s) Tobis MJ, Azarbal B

Citation: Texas Heart Institute Journal, 2005, vol./is. 32/3(362-5), 0730-2347;0730-2347 (2005)

Publication Date: 2005

Abstract: Cryptogenic stroke is a diagnosis of exclusion. These are strokes that occur in people who are usually less than 55 years old, without an identifiable cause. Our sensitivity to these events has been heightened because of the new definitions of a transient ischemic attack. Transient ischemic attack (TIA) is a clinical diagnosis of a neurologic deficit without MRI abnormalities: if there is an MRI abnormality, whether or not that person is symptomatic, it is now defined as a stroke. With these new definitions, and the sensitivity of MRI, we are seeing more cryptogenic strokes. It has been hypothesized that many cryptogenic strokes are caused by small emboli that travel from the legs to the right atrium; during straining (such as a Valsalva maneuver) these emboli can go across a PFO into the left atrium and then travel to the brain, producing a stroke. The problem is that these are very small emboli, approximately 1 to 3 mm, and we can't actually show these small emboli crossing from right to left. However, large emboli have been observed by echocardiography to be trapped in the PFO. So the diagnosis of cryptogenic stroke is a diagnosis of exclusion that is impossible to verify. What is the scope of the problem? Of the 700,000 strokes per year in the United States, 80% of them are ischemic, and 20% of those are defined as cryptogenic. The prevalence of PFO among this cryptogenic stroke population is about 40% to 50%; in the general population, it's only about 20%. Current estimates are that somewhere between 30,000 and 60,000 strokes per year in the U.S. are caused by paradoxical embolism through a PFO. There are some other fascinating associations: scuba divers with PFOs are more susceptible to decompression illness. Platypnea-orthodeoxia is a condition of desaturation that occurs when you're standing up but not when you're lying down; these patients are quite symptomatic, with arterial saturations in the low 80s. They also frequently have PFOs; if you close the PFO, the arterial desaturation is alleviated. Fat emboli during orthopedic surgery or air emboli during neurosurgery may also travel through the venous system. If you don't have a PFO, the fat or the air is trapped in the lungs and doesn't cause much of a problem unless it's massive; but if you have a PFO, then the embolus can go from right to left atrium up to the brain, with devastating neurologic consequences.

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14. Doppler ultrasound monitoring for detection of microembolic signals in peripheral arteries

Author(s) Kudo T., Inoue Y., Sugano N., Iwai T.

Citation: European Journal of Vascular and Endovascular Surgery, July 2002, vol./is. 24/1(37-42), 1078-5884;1532-2165 (July 2002)

Publication Date: July 2002

Abstract: Objective: to use Doppler ultrasound to detect peripheral microembolulation. Methods: standard Transcranial Doppler equipment was used to peripheral detect peripheral embolic high intensity transient signals (HITSs) in a pig model following injection of microparticles and atheroma, and in 23 patients who underwent open repair of an abdominal aortic aneurysm (AAA), six patients with blue toe syndrome and 10 age matched healthy subjects. Results: the pig study showed increasing signal intensity with particle size. Particles of 100 (n = 24), 200 (n = 17), and 400 mum (n = 31) elicited 14, 25, 33 dB signals, respectively (p < 0.05). During AAA surgery, the intensity (median) of HITSs before clamping (n = 226) and after declamping (n = 1216) were 14, and 20 dB, respectively (p < 0.001). Quite a few HITSs were detected after surgery. In patients with blue toe syndrome, a total of 63 HITSs could be detected, and the frequency of HITSs (median: 5.72/30 min) was significantly higher than that in patients with AAA before surgery (0.065/30 min) (p < 0.001). Conclusions: Doppler ultrasound technique may be a clinically useful test to guide the treatment of patients at risk of distal atheroembolic events. 2002 Elsevier Science Ltd. All rights reserved.

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