The Endovascular Management of Blue Finger Syndrome

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Objectives: to review our experience of the endovascular management of upper limb embolisation secondary to an ipsilateral proximal arterial lesion.

Design: a retrospective study.

Materials and methods: over 3 years, 17 patients presented with blue fingers secondary to an ipsilateral proximal vascular lesion. These have been managed using transluminal angioplasty (14) and arterial stenting (five), combined with embolectomy (two) and anticoagulation (three)/anti-platelet therapy (14).

Results: all the patients were treated successfully. There have been no further symptomatic embolic episodes originating from any of the treated lesions, and no surgical amputations. Complications were associated with the use of brachial arteriotomy for vascular access.

Conclusions: endovascular techniques are safe and effective in the management of upper limb embolic phenomena associated with an ipsilateral proximal focal vascular lesion.

Key Words: Embolism; Embolism, cholesterol; Upper extremity; Arm; Hand; Fingers; Ischaemia; Angioplasty, balloon; Angioplasty; Stents; Embolectomy.

Introduction

The phenomenon of peripheral embolisation from a proximal arterial lesion is well recognised. Microemboli from such lesions may occlude digital arteries, most commonly in the feet, resulting in the blue toe syndrome as described by Karmody. The condition is associated with a significant risk of further microembolisation or macroembolisation threatening the viability of the involved limb. Several series have described the management of patients with blue toe syndrome, both by surgical1±3 and, more recently, by endovascular techniques.4,6 However, few cases of the upper limb equivalent, blue finger syndrome, have been reported. We present our experience of treating patients with blue finger syndrome, using a variety of endovascular techniques.

Patients and Methods

Over a period of 3 years we have treated 17 patients referred with sudden onset of ischaemic lesions affecting one or more fingers of one hand consistent with a diagnosis of “blue finger syndrome”. These patients were treated at three different vascular centres and included both acute referrals, and referrals following an earlier symptomatic episode. The group consisted of 14 women and three men with a mean age of 58 years (range 32–86 years years). Fourteen had a history of smoking, three were diabetic and one had had radiotherapy to the axilla for breast carcinoma. One patient had had previous surgery for a cervical rib.

The patients’ records were reviewed to determine the presenting signs and symptoms, including, where documented, the palpable pulses in the symptomatic arm and the brachial-brachial index (BBI – brachial arterial pressure in the symptomatic arm divided by that in the contralateral arm). The angiographic lesions identified and the subsequent interventional procedures performed were noted, together with the immediate results of such treatment, including the post
Complementary medical treatment was also recorded. A combination of case note review and contacting the patient’s general practitioner, other clinician, or the patient himself or herself was used to determine the subsequent efficacy of the treatment, particularly regarding any further embolic events. Any subsequent imaging of the relevant vessels was also recorded.

**Results**

**Presenting features**

A total of 17 patients presented with either “blue finger” (digital microembolisation) or blue hands (macroembolisation).

Fourteen patients initially presented with signs of digital microembolisation including one or more blue fingers (13), splinter haemorrhages (three) and ulceration (two). Of these, five first presented to other centres who subsequently referred them on for further management. Four of these had been started on aspirin, and did not have any further embolic episodes in the intervening period. However, one patient, who was not treated with aspirin or warfarin, represented with new signs of macroembolisation including a blue hand and loss of previously recorded radial and ulnar pulses (KD – Table 1). Only four patients with clinical microembolisation had normal peripheral pulses in the asymptomatic arm. One patient (CF), who had an occluded haemodialysis arteriovenous fistula, had normal brachial and ulnar pulses but an absent radial pulse. The remainder had weak or absent brachial, radial, and ulnar pulses due to the proximal vascular lesion.

Three patients had evidence of macroembolisation with blue hands, at initial presentation. All patients with macroembolisation had normal brachial pulses and absent radial or ulnar pulses.

Two patients gave a history of paraesthesia in the symptomatic hand, but only one patient described symptoms of arm claudication.

**Investigation and treatment**

**Stenotic disease.** At angiography 15 of the patients were found to have proximal stenotic lesions in the symptomatic arm, with a marked left sided preponderance (Table 1). The recorded BBIs in this group were in the range 0.62–1.0 (median 0.78). In only one patient (KD) was proximal thrombus found associated with a subclavian stenosis together with distal thromboembolus at the brachial bifurcation. Fourteen patients with stenotic lesions were treated by balloon angioplasty in the first instance, with subsequent stent placement in two for significant residual stenoses. The patient who had a stenosis following axillary radiotherapy (MC) was stented *de novo*. In one patient (LC) the stenotic lesion was overlying the origin of the vertebral artery, in which there was still antegrade flow. A 3F balloon catheter was therefore used to occlude the vertebral artery during subclavian angioplasty, to reduce the risk of cerebral embolisation. Eleven of these patients were continued on long-term aspirin 150 mg per day, nine patients had heparin, and two patients had long-term warfarin (including the patient with visible thrombus on the subclavian stenosis). Two patients also had an iloprost infusion, one of whom was intolerant of aspirin.

**Aneurysmal disease.** One patient (EB) had an aneurysm immediately distal to a previously resected cervical rib which was treated with a covered stent (Cragg endoprosthesis). A second patient (MB) had an aberrant right subclavian artery with an aneurysm segment containing thrombus. A Wallstent was placed to stabilise the thrombus. Both patients were treated with aspirin and heparin and the patient with the aberrant right subclavian artery aneurysm was continued on long term warfarin.

**Approach.** Endovascular treatment of the vascular lesions was performed using a variety of vascular access routes including brachial (either percutaneous or surgical cut down), femoral and combined brachial and femoral access. In two patients the use of a surgical brachial cut down permitted balloon embolectomy of thrombus lodged at the brachial bifurcation, and also in one case (KD), removal of thrombus on the subclavian stenosis.

**Outcome**

The primary treatment was technically successful in all the patients (residual stenosis of less than 50%, exclusion of the aneurysm, completed embolectomy) and the patients have been followed-up for a mean of 13.7 months (range 3–36 months).

**Stenotic disease.** In the patients with stenotic lesions the BBIs recorded following treatment were in the range
Table 1. Angiographic findings, their treatment and outcomes in 17 patients with blue finger syndrome

<table>
<thead>
<tr>
<th>Case initials</th>
<th>Age</th>
<th>Sex</th>
<th>Risk factors</th>
<th>Signal/symptoms</th>
<th>Angiographical findings*</th>
<th>Intervention</th>
<th>Medical treatment</th>
<th>Follow up</th>
<th>Outcome</th>
<th>Vascular imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>KD</td>
<td>39</td>
<td>F</td>
<td>Smoker</td>
<td>Blue left hand</td>
<td>L SA stenosis</td>
<td>Embolectomy</td>
<td>Heparin/ warfarin</td>
<td>14 m</td>
<td>Occasional</td>
<td>Duplex – 14 m</td>
</tr>
<tr>
<td>SB</td>
<td>47</td>
<td>M</td>
<td>Smoker</td>
<td>Ulcer left finger</td>
<td>SA thrombus</td>
<td>Angioplasty</td>
<td>Aspirin</td>
<td>22 m</td>
<td>Asymptomatic</td>
<td>None</td>
</tr>
<tr>
<td>SM</td>
<td>56</td>
<td>F</td>
<td>Hypertension</td>
<td>Ex-smoker</td>
<td>Blue left finger</td>
<td>Angioplasty</td>
<td>Heparin</td>
<td>31 m</td>
<td>Asymptomatic</td>
<td>Duplex – 7 m</td>
</tr>
<tr>
<td>AT</td>
<td>32</td>
<td>F</td>
<td>Smoker</td>
<td>Blue left finger</td>
<td>SA stenosis</td>
<td>Embolectomy</td>
<td>Aspirin</td>
<td>20 m</td>
<td>Asymptomatic</td>
<td>None</td>
</tr>
<tr>
<td>EB</td>
<td>86</td>
<td>F</td>
<td>Smoker</td>
<td>Blue right hand</td>
<td>L SA stenosis</td>
<td>Embolectomy</td>
<td>Aspirin</td>
<td>24 m</td>
<td>Asymptomatic</td>
<td>Duplex – 6 m</td>
</tr>
<tr>
<td>LC</td>
<td>69</td>
<td>F</td>
<td>Smoker</td>
<td>Blue left finger</td>
<td>SA stenosis</td>
<td>Angioplasty</td>
<td>Heparin/ warfarin</td>
<td>6 w</td>
<td>Asymptomatic</td>
<td>Duplex – 13 m</td>
</tr>
<tr>
<td>MB</td>
<td>66</td>
<td>F</td>
<td>Smoker</td>
<td>Blue right finger</td>
<td>R SA (aberrant)</td>
<td>Wallstent</td>
<td>Heparin/ warfarin</td>
<td>13 m</td>
<td>Asymptomatic</td>
<td>None</td>
</tr>
<tr>
<td>EH</td>
<td>69</td>
<td>F</td>
<td>Hypertension</td>
<td>Blue left finger</td>
<td>L SA stenosis</td>
<td>Angioplasty</td>
<td>Aspirin</td>
<td>7 m</td>
<td>Asymptomatic</td>
<td>None</td>
</tr>
<tr>
<td>MW</td>
<td>79</td>
<td>F</td>
<td>Smoker</td>
<td>Blue right finger</td>
<td>R SA stenosis</td>
<td>Angioplasty</td>
<td>Iloprost</td>
<td>9 m</td>
<td>Asymptomatic</td>
<td>Duplex – 9 m</td>
</tr>
<tr>
<td>JG</td>
<td>39</td>
<td>F</td>
<td>None</td>
<td>Blue left finger</td>
<td>L SA stenosis</td>
<td>Angioplasty</td>
<td>Iloprost</td>
<td>15 m</td>
<td>Asymptomatic</td>
<td>None</td>
</tr>
<tr>
<td>RP</td>
<td>61</td>
<td>M</td>
<td>Smoker</td>
<td>Blue left finger</td>
<td>L SA stenosis</td>
<td>Angioplasty</td>
<td>Heparin</td>
<td>36 m</td>
<td>Asymptomatic</td>
<td>Duplex – 36 m</td>
</tr>
<tr>
<td>AA</td>
<td>54</td>
<td>F</td>
<td>Smoker</td>
<td>Blue left finger</td>
<td>L SA stenosis</td>
<td>Angioplasty</td>
<td>Heparin</td>
<td>12 m</td>
<td>Asymptomatic</td>
<td>None</td>
</tr>
<tr>
<td>WS</td>
<td>77</td>
<td>M</td>
<td>Smoker</td>
<td>Blue right finger</td>
<td>R SA stenosis</td>
<td>Angioplasty</td>
<td>Heparin</td>
<td>8 m</td>
<td>Asymptomatic</td>
<td>None</td>
</tr>
<tr>
<td>JS</td>
<td>57</td>
<td>F</td>
<td>Smoker</td>
<td>Blue left hand</td>
<td>L SA stenosis</td>
<td>Angioplasty</td>
<td>Heparin</td>
<td>5 m</td>
<td>Asymptomatic</td>
<td>None</td>
</tr>
<tr>
<td>BS</td>
<td>72</td>
<td>F</td>
<td>Diabetes mellitus</td>
<td>Ulcer</td>
<td>L SA stenosis</td>
<td>Angioplasty</td>
<td>Heparin</td>
<td>4 m</td>
<td>Asymptomatic</td>
<td>Duplex – 4 m</td>
</tr>
<tr>
<td>CF</td>
<td>45</td>
<td>F</td>
<td>Smoker</td>
<td>Blue left fingers</td>
<td>L SA stenosis</td>
<td>Angioplasty</td>
<td>Heparin</td>
<td>5 m</td>
<td>Asymptomatic</td>
<td>Duplex – 2 m</td>
</tr>
<tr>
<td>MC</td>
<td>42</td>
<td>F</td>
<td>R axillary radiotherapy</td>
<td>Splinter haemorrhage</td>
<td>R SA/AA stenosis</td>
<td>Memotherm Stent</td>
<td>Heparin</td>
<td>3 m</td>
<td>Forearm/band pain – 3 w</td>
<td>Duplex – 3 w</td>
</tr>
</tbody>
</table>

*SA: subclavian artery; AA: axillary artery; BA: brachial artery; UA: ulnar artery.

0.86–1.12 (median 0.95). The only immediate complication encountered was when, for technical reasons, a stent was deployed distal to the stenotic lesion being treated (CF). The stenosis was then dilated from the brachial approach, but the deflated balloon could not be withdrawn through the stent without displacing it, and it had to be recovered via the femoral artery.

Fourteen people have remained asymptomatic even though one case developed a recurrent stenosis at 3 years (on ultrasound) and another, whilst the subclavian angioplasty site remained patent, occluded the distal axillary and brachial artery proximal to the arteriotomy site 2 months following treatment. There has been healing of the lesions in both the patients who presented with finger ulceration. Two patients whose blue fingers developed gangrenous tips, have either undergone, or are proceeding to spontaneous auto-amputation. One patient, who had not had any further vascular symptoms, died 6 weeks after treatment from an unrelated illness. Two patients have had further symptoms following initial treatment. One patient has continued to have occasional paraesthesia in the finger that had been the site of microembolisation at presentation, but has had no new symptoms fol-
lowing treatment. The second patient (MC) developed pain in the forearm and hand 3 weeks after having a stent inserted via a brachial cut down. The brachial artery was re-explored and clot removed from the brachial artery which was thought to be related to an intimal flap at the arteriotomy site. The artery was repaired and the patient has subsequently remained asymptomatic.

_Aneurysmal disease._ Six months after treatment, the patient who had a covered stent inserted for an aneurysm, developed arm claudication. An angiogram confirmed occlusion of the stent, which was thought to be due to poor alignment of the stent within the artery because of the rigidity of the device. There was, however, good distal collateral flow. Her symptoms soon resolved and she remains asymptomatic. The second patient with aneurysmal disease remains asymptomatic.

Discussion

The blue finger syndrome is uncommon. Whilst there are no randomised trials to compare any intervention (conventional surgical or endovascular) against best medical therapy, it would seem reasonable to correct the underlying source of emboli to stop further events, and, where the lesion is occlusive, improve flow to the threatened tissue. As illustrated by one of our cases (KD), if treatment is delayed then further micro- or macroemboli may threaten the viability of the arm.

Vascular lesions may give rise to emboli of several different types including cholesterol-rich debris and fibrinoplatelet material, which cause microembolisation, and larger thrombi which cause macroembolisation.1-6 Fibrinoplatelet material probably accounts for most microemboli in blue digit syndrome,14,6 and this is supported by the reported effectiveness of antiplatelet/anticoagulant therapy in such patients.47 However, this treatment alone is not always sufficient. One patient (EB) was already on long-term aspirin when she first presented with embolic symptoms. The formation of fibrinoplatelet material is triggered by a combination of abnormal vascular endothelium and/or abnormal flow within the vessel. The underlying proximal lesion may be recognised as an area of intimal irregularity, which may or may not be haemodynamically significant, or may take the form of an aneurysm. Patients often do not give a history of arm claudication, even if there is a haemodynamically significant stenotic lesion. In addition, endothelial lesions causing little or no stenosis can still be the origin of distal emboli. As a result, signs and symptoms of micro- or macroembolisation may be the only presenting features of the syndrome, and whilst the possibility of a cardiac origin should be considered, a proximal vascular lesion in the affected arm should be sought, and if present, treated.

Endovascular therapy, using balloon angioplasty or stents, is aimed at correcting these abnormalities by establishing more normal flow patterns, and stimulating the formation of a stable neo-intima. Angioplasty alone is usually sufficient treatment for simple stenotic lesions, but significant residual stenoses, eccentric stenoses and aneurysms may all benefit from the use of stents.

Compared with the blue toe syndrome, a number of different techniques and access may be required for the successful endovascular treatment of blue finger syndrome. Angioplasty of subclavian and axillary stenoses is now a well-recognised and accepted technique, with a predominance of left-sided lesions often being reported.9,10 It often benefits from the use of a brachial approach, which provides both greater stability when trying to cross a difficult stenosis, frequently near the origin of the vessel, and also a straighter route which aids both angioplasty and stent deployment. Combined brachial and femoral access allows simultaneous vascular imaging during the interventional procedure. A surgical arteriectomy may facilitate brachial access, particularly if the brachial artery is not palpable. If there is intravascular thrombus present, either proximally or distally, then balloon embolectomy can be performed at the same time as treatment of the proximal vascular lesion. Subclavian angioplasty also carries the additional risk of vertebral embolisation and occlusion, particularly if the lesion is close to the origin of the vertebral artery. In some patients the subclavian steal phenomenon may provide protection, but this is not always the case (e.g. LC), and balloon occlusion of the vertebral artery during angioplasty may be indicated.11 The use of a Wallstent to manage loose thrombus within an anomalous subclavian artery aneurysm is novel and clearly would not be appropriate where the aneurysm was having a mass effect.

In our series of 17 patients with blue finger syndrome, endovascular treatment combined with antiplatelet/anticoagulant therapy has proved to be effective in preventing further emboli from a variety of underlying proximal vascular lesions. Recurrent stenoses may develop (e.g. RP) but the more stable neointima has so far not led to any new embolic episodes. Two patients developed brachial thrombus/
occlusion soon after having a brachial arteriotomy which was thought most likely to represent a local complication of the arteriotomy. Only one patient was asymptomatic but arteriotomy should probably be restricted to the more complex cases. Two patients had minor ischaemic tissue loss but no patients required a surgical amputation. No patients have had recurrent symptoms.

Conventional surgical techniques offer an alternative to the endovascular approach. Embolectomy is effective in large vessels and was used in this series in two patients, but the technique is difficult for peripheral emboli and does not correct the underlying proximal lesion. In addition, it is apparent from this series that the outcome for peripheral embolisation distal to the brachial trifurcation is good if the proximal lesion alone is attended to. Thrombolysis is successful in acute occlusion of the upper limb in 76–92% of cases but runs the risk of stroke in up to 4% of cases. For this reason, if large vessel embolisation is considered to require intervention, we prefer to use a combination of embolectomy and proximal endovascular intervention. Surgery to the proximal disease would include subclavian carotid transposition and carotid subclavian bypass with proximal ligation if appropriate. These carry a perioperative death rate of 0–2% and stroke rate of 1.5–3%. In addition, 1–2% of grafts become infected and phrenic nerve injury, thoracic duct laceration and Horner’s syndrome are all well recognized. Where intrathoracic surgery is required then the associated mortality rate is 3–15%. Conventional surgical procedures do have excellent patency rates that probably exceed endovascular procedures. However, where the underlying pathophysiology of the condition is one of peripheral embolisation rather than reduced flow, long-term patency of the surgery may not be the best outcome measure. The situation is analogous to carotid endarterectomy for symptomatic carotid disease where the relevant outcome measure is the cerebral ischaemia event rate and not particularly whether the patient has a degree of symptomatic restenosis. Endoscopic sympathectomy is best used for hyperhidrosis, the long-term benefits for vascular ischaemia are less convincing.

**Conclusion**

The safety and efficacy profile demonstrated in this series would suggest that, where appropriate, patients with the blue-finger syndrome due to proximal arterial disease should primarily be treated using endovascular techniques.

**References**


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