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The acute blue finger: management and outcome

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ABSTRACT

INTRODUCTION The objective was to assess the management, and short- and longer-term outcome of patients presenting with an acute blue finger.

PATIENTS AND METHODS This was a retrospective, case-note review and prospective follow-up by telephone and general practitioner enquiry. All patients who presented with sudden onset blue discolouration of a finger within the previous 72 h, with normal radial and ulnar pulses, were included.

RESULTS From 2000 to 2006, 22 patients, 15 female, 7 male, were reviewed. Median age was 56 years (range, 19–88 years). Median time from onset of blue finger was 6 days (range 1 day to 3 months). In most cases (17), no underlying cause was identified. Five patients had an underlying cause; two had symptoms compatible with Raynaud's phenomenon, one patient had signs (later confirmed on MRA) of arterial thoracic outlet syndrome and two had polycythaemia (haemoglobin > 17 g/dl). Otherwise, all laboratory investigations were normal. Upper limb duplex, echocardiogram and 24-h cardiac tapes were normal in all cases. Median follow-up was 19 months. Three patients had recurrent symptoms in the finger. No patient suffered tissue loss or loss of digit(s), and none had stroke or arterial embolisation.

CONCLUSIONS The acute blue finger is a benign condition not suggestive of arterial embolisation. Tissue or digit loss is not a threat and, in the longer term, there is no threat of embolisation to other vascular sites.

KEYWORDS

Vascular – Blue – Finger

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The acutely blue finger is an uncommon, isolated problem and may have many causes (Table 1). It has only been described in a small series of case studies.[1,2] This study aimed to examine the management, outcome and aetiology of patients presenting with this condition. In addition, the study aimed to follow-up these patients to assess whether the acute blue finger was a marker for an increased risk of major arterial embolisation later on.

Patients and Methods

All patients referred between 2000 and 2006 with an acutely blue finger to one tertiary centre were assessed. The vascular department has five consultant surgeons and serves a population of 1.2 million people. Details of all referrals to the department are routinely entered prospectively into a vascular database as part of the morbidity and mortality audit. Patients were identified from this database. Patients presenting with an acute bluish discolouration of one of more digits, with normal pulses and no difference in blood pressures between both upper limbs were included. Patients with chronic upper limb peripheral vascular disease or clinical evidence of major upper limb embolisation to the brachial, radial or ulnar arteries were excluded.

Table 1 Suggested causes for an acutely blue finger. Trauma may include local bruising or bleed into a tendon sheath.

- Raynaud's syndrome
- Vasospasm
- Trauma
- Atherosclerosis
- Thoracic outlet syndrome
- Vibration-induced injury
- Buerger's disease
- Micro-emboli
- Venous thrombosis
- Frost-bite
- Cryoglobulinaemia

Data were collected on the symptoms and signs at presentation, past history of cardiovascular risk factors, smoking and occupation. The presence of Raynaud's phenomenon was assessed by questioning according to a standard description of repeated episodes of colour change with associated numbness and paresthesia upon exposure to cold.[3] Initial management, investigations and imaging requested at presentation by the vascular surgeon were noted.

Follow-up was undertaken by review of the case notes. The patient and patient's general practitioner were contacted directly. The symptoms associated with the bluish colouration of the digit, the length of time of symptoms, any diagnosis made and further episodes of symptoms were reviewed at presentation, 6 weeks' follow-up in out-patients and in July 2006 by direct contact and correspondence with the patient and patient's general practitioner.

Results

In 6 years, 22 patients were seen with an acutely blue finger. Median age was 56 years (range, 19–88 years). Fifteen cases were female. In 14 cases, the left hand was affected. The index finger was affected in nine cases; the thumb in four, the middle finger in six, and the little finger in two and one case involved all fingers on one hand. All 22 cases presented with blue discolouration of upper limb digits. All but three had other symptoms as well: ache (2) or pain (8), altered sensation (pins and needles or numbness; 9), coldness (6), and swelling (5). No patient had a previous history of an acute blue finger. Most patients (17) were seen within 24 h of developing symptoms; all were seen within 72 h of onset.

Nine patients were smokers (mean 25 pack years); a further three were occasional smokers and one an ex-smoker. Six had risk factors for cardiovascular disease, none of which were diabetes. Two had symptoms compatible with Raynaud's phenomenon (aged 55 years and 68 years); the latter had previously been diagnosed with scleroderma.

On examination, all patients had normal brachial, radial and ulnar pulses. No significant difference in blood pressure was recorded between the upper limbs. One patient had reproducible symptoms on abduction and external rotation of the right shoulder suggestive of thoracic outlet syndrome. Polycythaemia was seen in two patients (haemoglobin 18.9 g/dl and 17.7 g/dl; normal range, 13–17 g/dl); mean haemoglobin was 14.1 g/dl. All other laboratory investigations were normal.

Initial management varied between cases: five were admitted, one was placed on intravenous heparin and later warfarin, whilst the majority (15) were discharged on antiplatelet agents (13 aspirin and two clopidogrel). The median duration of symptoms was 6 days (interquartile range, 2–9 days).

In summary, at presentation no cause was identified in 17 patients. Two had Raynaud's phenomenon, two were polycythaemic, and one had signs compatible with thoracic outlet syndrome.

Patients underwent imaging investigations either as an in-patient at acute presentation or as an out-patient within a few weeks of presentation, except the two patients with Raynaud's phenomenon and one who was lost to follow-up. No proximal cause or source of embolism was identified on upper limb arterial duplex ultrasound examination or transthoracic echocardiogram and 24-h cardiac tape.

At 6-week follow-up, 17 patients had no further symptoms. Two had persistent symptoms that had not fully resolved. Three had sporadic symptoms of swelling or discolouration or coldness of varying digits, but of a lesser severity than the initial presentation. One patient was lost to follow-up. No further patients were diagnosed with an underlying problem. Of the five with a diagnosed problem, the two patients with Raynaud's syndrome remained asymptomatic. One patient had thoracic outlet syndrome confirmed on MRA and was offered surgical decompression but opted for conservative management. Of the two patients found to be polycythaemic both underwent treatment and follow-up with the haematologists; one had persistent symptoms. Tissue loss was not seen in any patient.

Median follow-up was 19 months. Only one patient, who initially presented with all fingers affected, had a recurrent episode of an acute blue finger 3 years later that was again self-limiting. One patient with polycythaemia suffered an acute ischaemic upper limb (of the same arm) 4 months later (1 month after stopping warfarin). Arterial duplex was normal at initial presentation but subsequent angiogram revealed thrombosis at a subclavian stenosis. She died 3 years later from other medical conditions. One patient with persistent symptoms at 6-week follow-up was lost to further follow-up. One further patient was lost to longer follow-up. Antiplatelet therapy was continued indefinitely in all patients who were started on this treatment at the initial presentation of their acute blue finger.

In summary (Table 2), following initial presentation, five patients were found to have an underlying diagnosis. At long-term follow-up, one patient with polycythaemia had persistent symptoms and one case of acute self-limiting recurrence occurred.

Discussion

The acute blue finger is a rare condition that appears to occur in all age groups, predominately affecting the female, middle-aged population. Echocardiography was negative in all cases where performed and does not appear to be helpful in the investigation of this problem. Investigations performed showed no evidence of arterial embolisation.

Table 2 Summary of diagnosis at presentation, outcome at short- and long-term follow-up of patients presenting with an acute blue finger

Diagnosis at presentation	Symptoms at 6-week follow-up	Long-term symptoms and outcome
Scleroderma	No	No
Raynauld's syndrome	No	No
Thoracic outlet	No	No
Polycythaemia	Persisting	Persisting
Polycythaemia	No	Subclavian artery thrombosis
Nil	Persisting	LTF
Nil	Sporadic	No
Nil	Sporadic	No
Nil	Sporadic	No
Nil	LTF	LTF
Nil	No	Short recurrence at 3 years
Nil	No	Died
Nil	No	No
Nil	No	No
Nil	No	No
Nil	No	No
Nil	No	No
Nil	No	No
Nil	No	No
Nil	No	No
Nil	No	No
Nil	No	No

No diagnosis found at presentation (Nil), symptoms at follow-up (no, sporadic, persisting) or lost to follow-up (LTF), long-term symptoms and outcome.

Although transoesophageal echocardiography is idea to rule out a cardiac cause of embolism, this is invasive and these data show that, in the long term, the acute blue finger is not associated with further embolic events. This condition does not appear to have the same poor prognosis associated with upper limb emboli. The acute blue finger appears to be a benign condition not associated with any threat to limb or digits, with minimal risk of further episodes.

Only a few previous small-scale studies and case reports have described the acutely blue finger.[1,2] The most recent by Khaira *et al.*[2] in 2001 described 11 patients presenting in this way. Our study reinforces the benign short-term effects of the problem; also, longer follow-up confirms both the lack of continuing symptoms and adverse prognostic significance for the patient. The small numbers do not allow comment on the effectiveness of antiplatelet agents for these patients.

The population affected by the acute blue finger appears to be different than that affected by Raynaud's phenomenon and that affected by peripheral vascular disease. Peripheral vascular disease tends to affect an older population with a male predominance. In primary Raynaud's syndrome, the median age of onset is 14 years, with an onset after 30 years rare.[4] The acute blue finger appears to affect an older population (mean age, 57 years) and does not appear to be associated with other rheumatological conditions. Secondary Raynaud's syndrome affects a slightly older population with the age of onset usually greater than 30 years in patients with clinical features suggestive of connective tissue disease (arthritis, abnormal lung function). Connective tissue disease only occurs in 2% of patients with vasospastic disease alone who were seronegative for antinucleotide antibody and rheumatoid factor.[5] Up to a fifth of patients with Raynaud's phenomenon, who have autoantibodies or abnormalities in nail-fold capillaries and who do not initially meet the criteria for a connective tissue disease, will progress ultimately to do so, usually within 2 years.[6,7] Therefore, it seems reasonable to suggest, as the aetiology of the acute blue finger still appears to be unclear, a thrombophilia and vasculitis screens could be sent and those patients with a negative screen are unlikely to have, or develop, a connective tissue disease.

Spontaneous digital venous thrombosis appears to occur rarely with a female predominance. The population affected by the acute blue finger appears to correlate more with the venous thromboses population.[8] van Rossum *et al.*[9] described six cases of spontaneous digital thrombosis and reviewed the literature of a further 20 cases. Digital thrombosis of the palmer veins appears to present with a nodule and bluish discolouration at the level of or distally to the proximal interphalangeal joint. Most cases appear to resolve spontaneously but surgery may be considered in the cases where the symptoms persist or the nodule enlarges. The acute blue finger could represent a venous phenomenon. Hofer[10] described three different subtypes of digital thrombosis and reinforced the importance of excluding an acquired or inherited hypercoagulable state in people presenting with digital thromboses. It seems reasonable to investigate patients presenting with an acute blue finger for hypercoagulable states and if symptoms suggest thoracic outlet obstruction.

Conclusions

The acute blue finger does not appear to be an arterial phenomenon. All cardiac investigations looking for sources

of emboli were negative and appear unhelpful in the management of the acute blue finger. Arterial imaging studies do not appear to play a useful role in the diagnosis of this condition. The case of thoracic outlet syndrome was diagnosed clinically and later confirmed by MRA. The later case of subclavian thrombosis had a normal upper limb duplex examination on initial presentation. The longer follow-up data here suggest that these patients are not at an increased risk of any future cardiovascular events. In patients presenting with an acute blue finger, it seems reasonable to investigate for connective tissue diseases and hypercoagulable states but no other investigations appear to be necessary. If no systemic disease is identified, then patients can be re-assured that the condition should resolve spontaneously and that further episodes are unlikely.

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