CASE REPORT

Acute blue finger: a diagnostic challenge

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SUMMARY

The management of the acute blue finger is controversial with many regarding it as a benign condition. However, we would argue that it should always be considered as an emergency. We present a challenging case of a 43-year-old woman who presented with a 1-week history of sudden onset blue discolouration of the left fifth digit, and a 6-week history of episodic joint problems. Examination showed bilateral normal radial and ulnar pulses. Following blood investigations, an initial working diagnosis of early rheumatoid arthritis with associated Raynaud's phenomenon was made. Also, infective endocarditis was considered due to temporary misleading physical signs. Later, CT angiography of the left upper limb arteries showed a significant proximal left subclavian stenosis. Subsequently, a diagnosis of the left subclavian arteritis associated with digit ischaemia from embolic debris was made and the patient underwent a left subclavian angioplasty. However, delayed management resulted in a necrotic digit, which was left to autoamputate.

BACKGROUND

Acute blue finger can occur in a wide variety of medical conditions. The common causes include Raynaud's disease or syndrome, infective endocarditis, peripheral vascular disease, thoracic outlet obstruction, subclavian artery stenosis, frostbite,

trauma and certain poisons. In a prospective follow-up of 22 patients who had presented with sudden onset blue discolouration of a finger with normal radial and ulnar pulses, it was concluded that acute blue finger is generally a benign condition not suggestive of arterial embolisation and with no threat from digit loss.¹ However, a recent case report has suggested considering a benign aetiology only for patients who present with tip sparing and a lack of history of rheumatological diseases or other embolic phenomena.²

CASE PRESENTATION

A 43-year-old Caucasian woman was referred urgently by the general practitioner with a 6-week history of tiredness and generalised episodic medium to large joint pains, stiffness and swelling (mainly shoulders, elbows and knees) and more importantly a 1-week history of sudden onset blue discolouration of the left fifth digit distal phalanx with bilateral mild tenderness over the finger pulps (figure 1). There was no significant medical history or family history, and she was on no regular medications. There was no recent history of trauma, skin rash or oral contraceptive use over the past 10 years. The patient was a light smoker and denied alcohol ingestion. Physical examination, including cardiovascular, neurological and rheumatological assessments, was unremarkable. Bilateral

Figure 1 Blue discolouration of the left fifth digit.

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radial and ulnar pulses were normal with no significant blood pressure (BP) difference between both arms (less than 20 mm Hg). Blood investigations showed strongly positive rheumatoid factor (RF=256) and high C reactive protein (CRP) (32 mg/L) and erythrocyte sedimentation rate (ESR; 40 mm/h). Rest of the routine blood investigations including full immunological screening and lipid profile were normal. Duplex ultrasound examination of the left upper limb arteries showed normal axillary, brachial, radial and ulnar arteries with no significant stenosis or occlusion. A presumptive diagnosis of probable seropositive inflammatory arthritis (possible new onset rheumatoid arthritis—ACR/EULAR RA classification score=6),³ associated with atypical Raynaud's phenomenon was made, and the patient was started on a high-dose oral corticosteroid therapy and a 5-day course of prostacyclin infusion (PGI₂; Flolan).

Three days later, a significant improvement of the joint pains and finger pulp tenderness was noted; however, the patient started to complain of severe pain of the left fifth digit with worsening of finger discoloration. Also, low-grade fever was reported. The full septic screen showed a normal chest radiograph, ECG, urinalysis and a high white cell count and neutrophil count of 13.8×10^9 and $10 \times 10^9/L$, respectively. This, along with the fever, was presumed to be secondary to either systemic inflammatory response or corticosteroid therapy. Physical examination at this point revealed a collapsing pulse, and a soft, ejection systolic (grade II) murmur heard best over the aortic area. In view of the new findings, a revised differential diagnosis considered subacute infective endocarditis, as suggested by the modified Duke criteria.⁴

At 48 h, three blood cultures showed no evidence of growth, and the transthoracic echocardiogram (TTE) showed no evidence of valve dysfunction or any obvious vegetation. Interestingly, as the prostacyclin infusion had stopped, the collapsing pulse and the soft systolic murmur disappeared suggesting that they were temporary findings (ie, large volume pulse and functional murmur) secondary to the vasodilation and hyperdynamic circulation caused by vasodilator drugs. As the signs resolved, transoesophageal echocardiogram (TOE) was not performed.

At day 7 postadmission, the patient was referred urgently for a vascular surgical assessment. A CT angiography of the left upper limb arteries showed a significant narrowing (70% stenosis) of the left subclavian artery origin with otherwise normal arteries (figure 2A, B). As the relationship between seropositive inflammatory arthritis and vasculitis had been well established,⁵ a final diagnosis of the left subclavian artery stenosis secondary to early arteritis associated with digit ischaemia from emboli was made, with a further inclusive diagnosis of seropositive inflammatory arthritis being considered. The other major differential for vasculitis would have been atherosclerosis causing subclavian stenosis. However, despite the fact that the patient smoked, the patient's age, clinical presentation, absence of other risk factors and the CT angiographic findings made the likelihood of atherosclerosis low.

TREATMENT

After confirming the diagnosis, a combination therapy with heparin anticoagulation and a single antiplatelet agent was started. At day 13, revascularisation via percutaneous intervention with stenting of the left subclavian artery was undertaken (figure 3), without complications. Despite revascularisation, the left fifth digit could not be salvaged, becoming more necrotic distally, and as expected intensely painful despite copious

Figure 2 (A) CT angiography. Axial plane showing severe eccentric narrowing of lumen of the origin of left subclavian artery. (B) CT angiography. Coronal plane showing severe eccentric narrowing of lumen of the origin of left subclavian artery.

analgesia. Although surgical amputation is usually the treatment of choice,⁶ autoamputation was allowed to ensue in view of the advanced digital ischaemia and tissue necrosis. Also, autoamputation was the patient's choice. Opioid analgesics, antiplatelet therapy, tapering dose steroids, a finger glove for support and smoking cessation advice and leaflets were given on discharge.

OUTCOME AND FOLLOW-UP

At 4 weeks of follow-up, the distal phalanx of the left fifth digit looked darkest in colour, more ischaemic and beginning to demarcate. Steroid therapy was stopped and the patient had not had any major exacerbations of her joint signs or symptoms. Inflammatory markers including ESR and CRP showed a trend towards reduction. The decision to start disease modifying antirheumatoid drugs was left to be made in the future, as appropriate.

DISCUSSION

Subclavian artery stenosis is uncommon. It can lead to critical symptomatic ischaemic changes affecting the brain, the heart in some cases and more frequently the upper extremities. Arm claudication, rest pain and finger necrosis from embolic debris can occur.⁷ The left subclavian artery is involved more than the right. Atherosclerosis is considered the most common cause of this condition. English and colleagues reported a prevalence of left subclavian artery stenosis of 4.3% in patients with a history of smoking, 4.3% in hypertensive patients, 6.8% in patients with diabetes mellitus and also in patients with peripheral vascular disease and cerebrovascular disease.⁸ Treatment of subclavian artery stenosis is generally according to the presenting signs and

Figure 3 Left subclavian angioplasty. Stenosis (red arrow), inflated balloon (blue arrow) and stent in situ (black arrow).

symptoms. Medical, endovascular or surgical options are available. The medical therapy involves antiplatelet agents, statin therapy, BP and diabetes control and smoking cessation. Endovascular techniques include all means of endovascular revascularisation (eg, transluminal angioplasty with stenting). Open surgical subclavian bypass is usually reserved for those with severe symptoms who have failed endovascular intervention.⁹

Our patient's presentation was rather challenging and complex. Not only because the pathology is rare, but also because of the interesting, thought-provoking sequence of events. Early exclusion of a vascular cause remains the ultimate goal of management to prevent potentially catastrophic complications.

Learning points

- ▶ Acute blue finger should be always treated as an emergency that requires prompt management.
- ▶ Early arterial imaging with angiography is almost always required to prevent potential adverse outcomes in patients presenting with acute blue finger, even with the presence of palpable pulses.
- ▶ Acute blue finger is a diagnostic challenge. A vascular surgical opinion should be sought early in the presentation.
- ▶ Although subclavian artery stenosis can be a cause of significant morbidity, it is usually overlooked in our patient population. Therefore, it is important that medical practitioners become aware of the diagnosis and management of this condition.

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