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ISOLATED ULNAR ARTERY OCCLUSION WITH CONCURRENT INTERNAL JUGULAR VEIN THROMBOSIS IN SEVERE IRON-DEFICIENCY ANAEMIA

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ABSTRACT

Introduction: Severe iron deficiency anaemia (IDA) is increasingly recognized as a cause of a reversible hypercoagulable state, although simultaneous arterial and venous thrombosis are rare. Several mechanisms have been proposed, including reactive thrombocytosis, endothelial dysfunction, and changes in erythrocyte rheology, which may be secondary to reduced tissue oxygenation.

Case description: We report the case of a 41-year-old woman with chronic menorrhagia due to fibroids who developed digital cyanosis and neurologic deficits due to severe microcytic anaemia (Hb 6.7 g/dl), associated with severe iron deficiency and mild thrombocytosis (442 $\times 10^9$/l). She was discharged after blood transfusion with therapy for Raynaud phenomenon (amlodipine) but returned to the emergency room due to an acute right ulnar artery occlusion complicated by internal jugular vein thrombosis that was managed with embolectomy and anticoagulation. Her digital function fully recovered, and she remained free of thrombophilia.

Conclusion: Our case highlights the potential for severe IDA to cause both arterial and venous thrombosis, even in the setting of only modest platelet elevations. Chronic tobacco use may have contributed to her presentation, and clinicians should remain aware of the possibility of thrombosis in patients presenting with severe IDA who do not have other known pro-thrombotic conditions.

KEYWORDS

Iron deficiency anaemia, arterial thrombosis, venous thrombosis, digital ischemia, reactive thrombocytosis

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LEARNING POINTS

- Severe iron deficiency anaemia can cause both arterial and venous thrombosis even in the setting of only mildly elevated platelet count, so platelet count alone should not be used as a determinant for thrombotic risk.
- Painless discoloration of the digits with diminished or non-existent pulse on examination in a young patient with no vascular risk factors requires vascular imaging prior to arriving at a diagnosis of Raynaud phenomenon, as the presentation can mimic acute arterial occlusion and require urgent intervention.
- A negative workup for thrombophilia and autoimmune disease cannot be used to rule out the possibility of a prothrombotic state, as severe iron deficiency anaemia can act as a prothrombotic trigger.

INTRODUCTION

Almost one-third of nonpregnant women worldwide are anaemic due to decreased iron levels, and this condition increases the risk of venous thromboembolism[1,2]. Severe iron deficiency anaemia (IDA) has recently been implicated in arterial occlusion events such as cerebral infarction, carotid artery thrombosis, and thrombosis of the ophthalmic artery[3]. Several mechanisms have been proposed, including reactive thrombocytosis, endothelial dysfunction, and changes in erythrocyte rheology, but isolated thrombocytosis rarely explains occlusion[2-4], whereas reduced tissue oxygenation may act synergistically with continued tobacco use to decrease the thrombotic threshold[5,6]. Isolated involvement of arterial or venous circuits during concurrent arterial and venous thrombosis is uncommon during severe IDA, with the majority of cases reporting only venous or only arterial involvement[3]; we present a case of both.

CASE DESCRIPTION

A 41-year-old woman with uterine fibroids and menorrhagia presented with painless blue discoloration of the right index and middle fingers and palm that had been present for 3 days *(Fig. 1)* plus headache, dizziness, and visual disturbances for 2 weeks. She denied any trauma, cold exposure, infection, chest pain, or dyspnoea; family history of Raynaud phenomenon, autoimmune disease, or thrombosis; or personal history of Raynaud phenomenon, autoimmune disease, pregnancy loss, or thrombosis. The patient was a former smoker with a 5 pack-year history.

On examination she looked pale and tired with blue, cold fingers and capillary refill of >3 seconds on the right index and middle fingers and palm. The radial and ulnar pulses were normal and symmetrical except the right radial artery was weaker than the left. There was no cyanosis of the rest of her body.

Laboratory investigations showed severe microcytic hypochromic anaemia with profound iron deficiency and mild thrombocytosis, along with a mildly elevated erythrocyte sedimentation rate, normal C-reactive protein levels, and mildly elevated D-dimer *(Table 1)*; beta-human chorionic gonadotropin, renal, liver, and coagulation function tests were normal. She received one unit of blood and iron therapy with gradual improvement of her hemoglobin up to 8.1 g/dl and was discharged home with oral iron, analgesia, and a course of amlodipine for



Figure 1. *Bluish discoloration of the right index and middle fingers on initial presentation (arrows), consistent with acute digital ischemia.*

Test	Result	Reference range
Haemoglobin (g/dl)	6.7	12–15
MCV (fl)	60.1	80–100
Ferritin (ng/ml)	6	12–150
Transferrin saturation (%)	3	20–50
Platelet count ($\times 10^9$/l)	442	150–410
ESR (mm/hr)	25	<20
CRP (mg/l)	4.4	<5
D-dimer (µg/ml)	0.61	<0.5

Table 1. *Initial laboratory investigations.*
Abbreviations: MCV, mean corpuscular volume; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein.

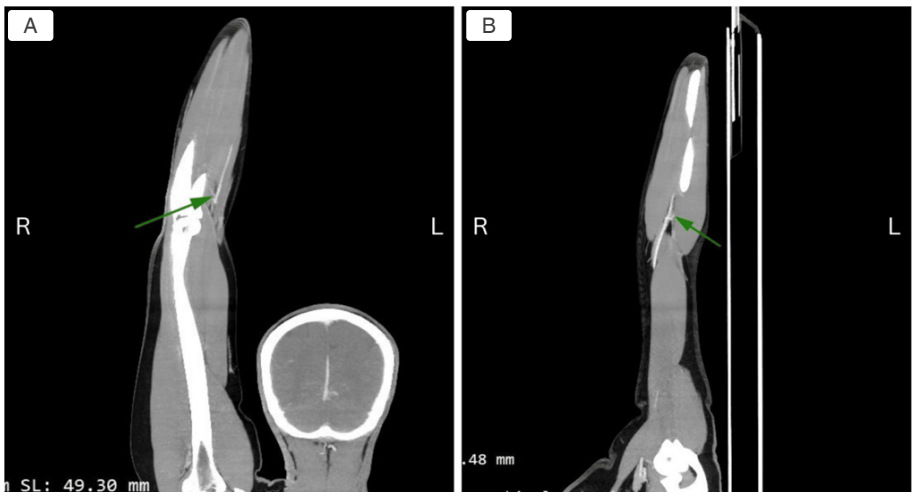


Figure 2. A) Coronal computed tomography angiography of the right upper limb at the level of the brachial artery bifurcation in coronal B) axial views both showing abrupt loss of opacification of the right ulnar artery (arrow), with normal contrast filling of the radial artery, consistent with ulnar artery occlusion.

presumed vasospasm alongside urgent rheumatology review. Two days later she presented with more severe discoloration of the fingers, intense pain, numbness, and inability to bend her fingers. On examination the right ulnar pulse was absent with normal radial/brachial pulses and normal comparative findings on the left. Bedside Doppler showed triphasic radial artery flow, but monophasic ulnar artery and palmar arch with no deep palmar arch flow. Computed tomography angiography (CTA) showed complete occlusion of the right ulnar artery up to the wrist with normal remainder arteries *(Fig. 2A and 2B)*. Notably it also showed a filling defect in the right internal jugular vein at the level of C2 – C3 suggesting partial thrombosis *(Fig. 3)*, which was not evident on a separate venous study.

The patient underwent an emergency open embolectomy with thrombus evacuation from the right ulnar and radial artery *(Fig. 4 and 5)*. The patient was placed on a regimen of therapeutic subcutaneous enoxaparin and intravenous alprostadil. Distal pulses were normal with improvement in digital perfusion, and motor power and flexion were normal with the exception of mild sensory loss and discoloration of the tip of the index finger. Extensive tests for thrombophilia and autoimmunity were performed and proved to be negative *(Table 2)*. The patient was discharged after 1 week of anticoagulation with a follow-up with haematology, vascular surgery, and gynaecology for her fibroid uterus and menorrhagia.

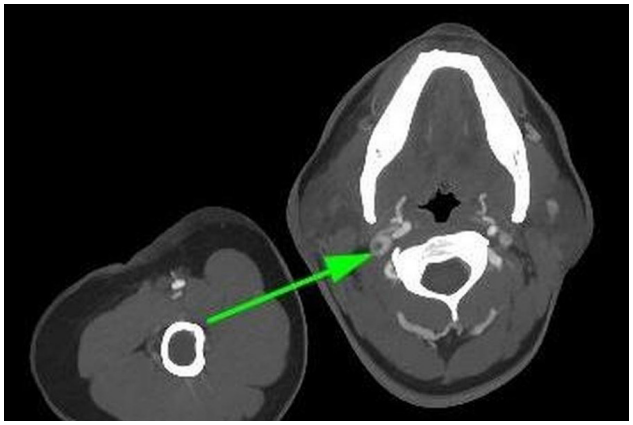


Figure 3. Axial venous-phase computed tomography image at the C2–C3 level showing an intraluminal filling defect within the right internal jugular vein (arrow), consistent with partial thrombosis.

DISCUSSION

This case is notable for an association of upper extremity arterial and internal jugular vein thrombosis within the same distribution as part of a hypercoagulable state; thrombosis of both systems simultaneously has been described in only a few previously reported cases of IDA[3]. Ischemia without significant cardiac risk factors was thought to be multifactorial; vasospastic disease was



Figure 4. Intraoperative photograph showing surgical exploration of the right ulnar and radial arteries during urgent open embolectomy.

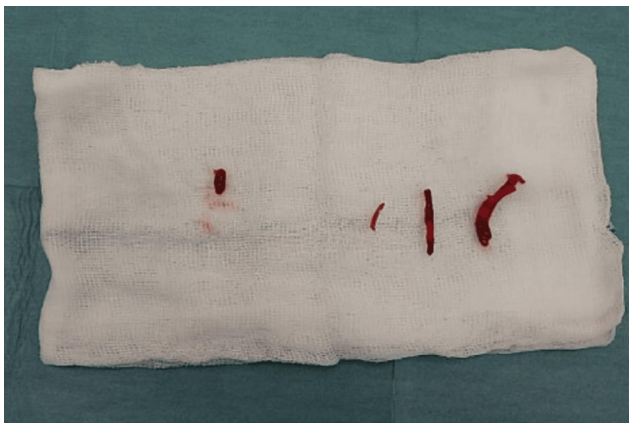


Figure 5. Retrieved arterial thrombi following open surgical embolectomy of the ulnar and radial arteries.

Investigation	Result
Antiphospholipid antibodies	Negative
Antinuclear antibodies	Negative
Extractable nuclear antigen profile	Negative
Anti-neutrophil cytoplasmic antibody	Negative
Protein C activity	Normal
Protein S activity	Normal
Antithrombin activity	Normal
Activated protein C resistance	Normal
Factor V Leiden mutation	Negative
Prothrombin G20210A mutation	Negative
JAK2 mutation	Negative
Flow cytometry for paroxysmal nocturnal haemoglobinuria	Negative

Table 2. Thrombophilia and autoimmune evaluation.

initially favoured, as the discoloration was initially painful and responded to amlodipine. However, the fixed nature of the ischemia, with documentation of arterial occlusion, suggests an irreversible process. The mildly diminished radial pulse on the right at presentation also supported this diagnosis; earlier consideration of vascular imaging could have expedited workup and therapy. Vasculitis was also a concern given the elevated erythrocyte sedimentation rate, but this was not supported by autoimmune serology or clinical findings, although it should be noted that seronegative vasculitis cannot be definitively excluded without a biopsy. Cardioembolism was felt to be less likely given the patient's young age and limited past medical history, although this remains possible as no cardiac workup or investigations for a source of embolism were performed for the jugular vein thrombosis. Inherited and acquired coagulopathy was similarly ruled out, leaving severe iron deficiency as a diagnosis of exclusion.

The role of thrombocytosis must also be discussed, as the patient's platelet count of 442 $\times 10^9$/l was only modestly elevated beyond her laboratory's reference maximum of 410 $\times 10^9$/l and significantly lower than diagnostic thresholds of >450 $\times 10^9$/l for thrombocytosis according to the World Health Organization (WHO) and 409 $\times 10^9$/l for the 99th percentile of normal adult women[4], compared to the >1000-2000 $\times 10^9$/l often seen in cases of IDA-associated thrombosis[3]. It is notable that the majority (>80%) of thrombosis events associated with thrombocytosis occurred in patients with platelet counts >600 $\times 10^9$/l, and that even extreme thrombocytosis (>1000 $\times 10^9$/l) only carries a 4-6% absolute risk of thrombosis[4]. Consequently, thrombocytosis likely played only a minor role, if any, in the development of this patient's multi-system thrombosis: it was considered to be part of a prothrombotic environment typical for severe anaemia, in which decreased erythrocyte deformability, increased tissue hypoxia, and release of prothrombotic mediators also contribute[2,5,7]. Mechanisms of IDA-associated thrombosis beyond reactive thrombocytosis have in fact been recognized since at least 2008, including iron-dependent elevations in coagulation factors VIII and fibrinogen, increased plasma homocysteine, impaired erythrocyte deformability, and direct iron-related endothelial dysfunction, all of which may act independently of platelet count[8].

Chronic tobacco use was a likely permissive factor due to its role as an independent risk factor for arterial thrombosis through endothelial dysfunction and impaired nitric oxide production[6], although it is difficult to assess the risk independently in the setting of severe anaemia. Overall, the case represents acute thrombosis of multiple vascular beds uncommon for IDA, namely upper extremity arteries and large cerebral veins, with no history of central venous catheterization[1,3,9,10]. It was noted that, in a previously described cohort of 9 patients with chronic IDA-associated thrombosis, only one had experienced multi-system thrombosis, and that it occurred years apart with much more severe thrombocytosis than observed in this case[10].

The present report's limitations are inherent to the patient's circumstances and the nature of the study. As a single-case report, no definitive conclusions can be drawn regarding causality; the association of IDA with multi-system thrombosis should be interpreted as a probable, but not proven, link. The diagnosis of upper extremity arterial ischemia was complicated by the lack of a dedicated venous CTA: jugular vein thrombosis was found incidentally on an arterial CTA. Vascular imaging at a later date to confirm resolution and guide further treatment and follow-up, as well as a transthoracic echocardiogram to rule out cardioembolism were not performed. Lastly, the role of chronic tobacco use as a prothrombotic agent, as well as the possibility of an alternative diagnosis not suspected by the treating physicians cannot be completely discounted despite extensive testing.

CONCLUSION

The presented case highlights the phenomenon of severe IDA provoking simultaneous arterial and venous thrombosis, even in the context of only slightly increased platelet counts, by causing a prothrombotic state through multiple mechanisms, not thrombocytosis alone, and chronic smoking may have contributed to the development of the prothrombotic state. Severe IDA is a potentially reversible cause of thrombosis that should be suspected in patients with fixed digital ischemia or an asymmetric pulse. Vasculitis, embolism, thrombophilia, and other vascular causes should also be ruled out. The most important lesson is that the correction of severe IDA and the underlying mechanism should be the primary focus of management.

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