An unusual cause of iron deficiency anaemia in an intravenous drug user

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CASE REPORT

A 40-year-old intravenous drug user was admitted to the department of medicine with a painful swollen left leg. He freely admitted to injecting heroin (diamorphine) intravenously and had recently injected into his left leg. He had a 10-year history of illicit drug use and had previously attempted to stop with the use of methadone. There was no history of hepatitis, human immunodeficiency virus (HIV) infection or the sharing of syringes used for injection.

Clinical examination revealed a thin, unkempt, middle-aged gentleman, who had pallor of the skin and the mucous membranes. He had evidence of drug 'tramline' marks, venous thrombophlebitis in his groins and both upper and lower limbs (*Figure 1*). There was no evidence of lymphadenopathy or signs of endocarditis. Chest and abdominal examination revealed no abnormalities. He had evidence of swelling, erythema and tenderness in the anterior lower left leg and there was no calf tenderness. The signs in his leg were consistent with cellulitis. Initial investigations revealed a severe hypochromic microcytic anaemia, a haemoglobin level of 4.3 g/dl with low serum iron, low ferritin and raised iron binding capacity consistent with iron deficiency anaemia. Electrolytes, liver function tests and clotting were normal.

He was transfused four units of red packed cells and treated with intravenous antibiotics. He denied any history of maleana, haematemesis, rectal bleeding or haematuria. He denied usage of non-steroidal inflammatory drugs or aspirin. A Doppler ultrasound of his leg revealed no evidence of thrombus. A chest X-ray revealed no abnormalities. A transoesophageal echocardiogram showed no evidence of endocarditis. Tests for HIV, hepatitis B or C revealed no abnormalities. A gastroscopy revealed no abnormality and duodenal biopsies revealed no evidence of coeliac disease. Colonoscopy revealed no abnormality. Red cell isotope scanning using 51-chromium-labelled red cells and mesenteric angiography revealed no cause for blood loss. Repeated samples of urine revealed no red blood cells. He eventually admitted to injection with syringes into his groin, often hitting his femoral artery rather than the femoral vein, with concomitant loss of arterial blood on multiple occasions. We assumed that this was the most likely cause of his iron deficiency anaemia.

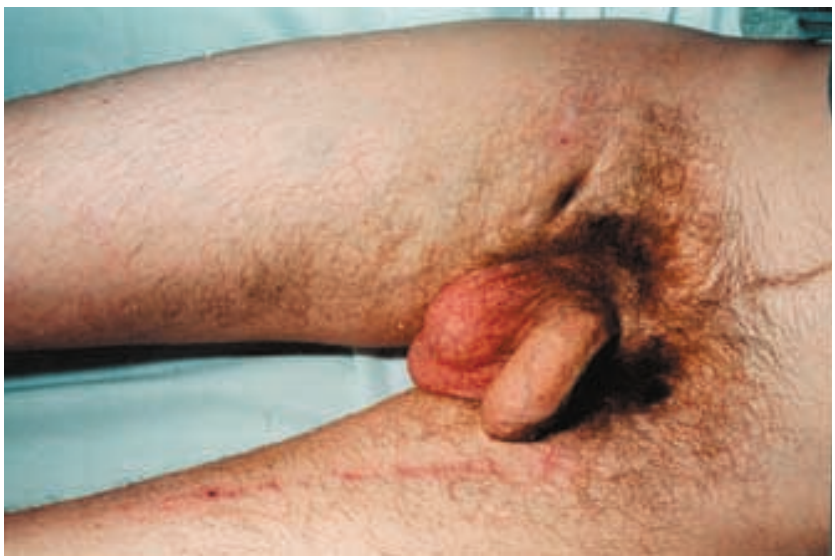

Figure 1. Patient's left thigh reveals evidence of venous thrombophlebitis, 'tramline' marks and there is scarring in the right groin from needle injections.

INTRODUCTION

An unusual cause of severe iron deficiency anaemia is described in a patient who used intravenous drugs. We believe this to be an important cause of anaemia in the differential diagnosis of such patients.

DISCUSSION

There are few studies on the reported prevalence of anaemia in intravenous drug users. In one study of American drug users attending a clinic 20% of them were found to be anaemic (Brown et al, 1993).

Anaemia may arise from a number of causes and may be multifactorial. Chronic infections such as infective endocarditis, human immunodeficiency virus (HIV), tuberculosis and localized abscesses are recognized complications in intravenous drug users and can result in anaemia as a consequence of chronic disease, usually resulting in a normocytic normochromic anaemia. Endocarditis can also result in a haemolytic anaemia as a result of red cell fragmentation (Gradon et al, 1996; Naidoo et al, 1998).

A possible cause of iron deficiency anaemia in this patient is dietary deficiency of iron as a result of severe malnutrition. Malnutrition resulting in folate or iron deficiency has been well recognized in patients with alcoholism (Savage and Lindenbaum, 1996), but although malnutrition is recognized in intravenous drug users (Santolaria-Fernandez et al, 1995), malnutrition

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severe enough to cause iron deficiency anaemia has not previously been documented.

Blood loss from the gastrointestinal tract causing iron deficiency anaemia can result from bleeding oesophageal varices or from coagulopathy as a result of chronic liver disease caused by hepatitis B or C viruses. This was essentially excluded by gastroscopy in our patient. Extensive investigations of the gastrointestinal tract revealed no other cause for blood loss.

We believe our patient had iron deficiency anaemia as a result of blood loss from self-injection. Factitious anaemia with severe iron deficiency has been described in a Japanese non-drug user medical student who had secretly performed repeated self-venesection (Nakamo et al, 1998), and other cases of factitious anaemia resulting from self-venesection have been reported (Granacher, 1982; Maruyama et al, 1987).

An extensive review of the medical literature using Medline has reported only one case report of blood loss as a result of self-injection in an intravenous drug addict; in this report the patient similarly had iron deficiency anaemia as a result of arterial injection and blood loss (Halperin, 1973).

CONCLUSIONS

We believe chronic blood loss from self-venesection in intravenous drug addicts to be an important consideration in the differential diagnosis of iron deficiency anaemia in such patients, and may prevent the need for multiple investigations. HM

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