Acute myelomonocytic leukaemia complicated by an acute aortic thrombosis

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Summary

Acute aortic thrombosis is a rare condition, occurring mainly as a result of trauma or atherosclerosis and occasionally secondary to hypercoagulable states. We report a patient with relapsed acute myeloid leukaemia who developed an unusual complication, acute aortic thrombosis.

Keywords: acute myeloid leukaemia, lupus anticoagulant, acute aortic thrombosis

Introduction

Acute aortic thrombosis is relatively rare (see box).¹⁻⁴ We report a patient with relapsed acute myeloid leukaemia who developed an acute aortic thrombosis.

Case report

KJ, a 38-year-old Malay man, presented as an acute emergency when he spontaneously developed sudden pain and numbness over his lower limbs with weakness, progressing to complete paralysis within one hour. He had urinary retention. One day earlier, he had had numbness and pain over his lower limbs lasting 15 minutes without any weakness. This resolved spontaneously.

He had been treated for acute myelomonocytic leukaemia a year earlier. He achieved remission after two induction courses consisting of daunorubicin and cytosine arabinoside. This was consolidated with two courses of chemotherapy consisting of VP 16 and cytosine arabinoside. He relapsed six weeks prior to the current presentation. He had refused reinduction with chemotherapy.

On examination, he was in severe pain. Marked pallor with gross gum hypertrophy was present. He did not have any petechial haemorrhages. Blood pressure was 190/100 mmHg. Both lower limbs were mottled and cold. The femoral and all lower limb pulses were absent. He had Grade 0 power in both lower limbs. Reflexes were absent. A sensory level at T10 was present. No abdominal bruit was heard. Bowel sounds were present. No fundal haemorrhages were seen. There was no lymphadenopathy or hepatosplenomegaly.

A full blood count showed a haemoglobin level of 5.3 g/dl, platelet count $84 \times 10^9/l$, white blood cells $50.4 \times 10^9/l$ with 60% blasts.

A dynamic CT scan of the abdomen revealed an aortic thrombus beginning just below the renal arteries and progressing down to both common iliac arteries (figure). The thrombus had almost occluded the whole lumen of the aorta. The para-aortic lymph nodes were normal and no abnormal masses were seen. Echocardiography was normal.

The lupus anticoagulant was detected using the kaolin clotting time test and the tissue thromboplastin index. Antithrombin III and proteins C and S were found to be normal. Antiphospholipid antibody testing was not performed (table).

The perfusion of his lower limbs improved with heparin infusion. The mottled appearance had disappeared six hours later and his limbs were warmer. He regained grade 1 power to his

Figure Dynamic CT scan image of the abdomen just below the renal arteries demonstrating an irregular hypodense filling defect consistent with a thrombus within the aorta, occupying 75% of the lumen (arrow). The aortic diameter is normal

Acute aortic thrombosis: causes

- trauma
- atherosclerosis
- antithrombin III deficiency
- protein C deficiency
- lupus anticoagulant
- spinal metastases
- following arterial catheterisation
- following heparin-induced thrombocytopenia

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Acute aortic thrombosis: features
<ul style="list-style-type: none">• sudden lower limb ischaemia• sudden hypertension

lower limbs and his sensory level improved to L3 level. All pulses in his lower limbs remained impalpable. His blood pressure returned to 150/85 mmHg after a day. He was able to micturate and also had normal bowel opening by the third admission day. He was subsequently started on warfarin. A repeat CT scan six days later did not show any improvement nor any progression of the thrombosis.

Unfortunately, he discharged himself against medical advice 14 days after admission when he was improving and pain free. He did not wish to have any further treatment for his relapsed acute myelomonocytic leukaemia. He passed away at home 10 days after discharge.

Discussion

The patient had developed acute aortic thrombosis following the relapse of acute myeloid leukaemia. He had no known risk factors for an acute thrombosis except possibly the lupus anticoagulant. Acute leukaemia should predispose him to bleeding rather than thrombosis.

Acute aortic thrombosis is an uncommon problem. The classical presentation is sudden symptoms and signs of lower limb ischaemia, which may mimic post-mortem changes.¹ A proportion of patients develops sudden hypertension.⁵ This may be due to renal artery occlusion or increase in peripheral vascular resistance. The blood pressure in the patient was only transiently high, which might have been partly contributed by severe pain. The important feature is impalpable pulses in the lower limbs. In patients with infrarenal aortic thrombosis,⁶ the arterial supply to the spinal cord is preserved. The neurological deficit is caused by ischaemic peripheral neuropathy causing sudden extreme pain in the lower limbs followed by paraesthesia. This appears to have been the case in this patient. Mesenteric infarction can occur if the inferior mesenteric artery is involved.

Table Results of the lupus anticoagulant tests

Test	Result
Kaolin clotting time	
Normal plasma	61.0 s
Normal plasma + patient plasma	72.2 s
Patient plasma	146.3 s
Index	11.0 s
Platelet neutralising procedure	
Patient's plasma + freeze-thawed platelets	57.0 s
Patient's plasma + TRIS buffered saline	56.9 s
Tissue thromboplastin index	
1/100	1.9
1/1000	2.2
Normal ratio $\leq$ 1.3	
Russell viper venom	
Normal plasma	23.2 s
Patient's plasma	21.7 s
Ratio	0.93
Normal ratio	<1.2

Two tests for the lupus anticoagulant were positive. This may explain the thrombotic tendency in this patient. There is one previous report of a case of aortic thrombosis associated with the antiphospholipid antibody.⁴ It is possible that immunological perturbations in leukaemia had contributed somehow to the development of the lupus anticoagulant or some other procoagulant leading to aortic thrombosis. A white blood cell thrombus may be another explanation but the total white cell count was only $56 \times 10^9/l$.

The treatment of choice for acute aortic thrombosis is emergency surgery with aortic reconstruction or a bypass procedure that carries a mortality of 60%.^{1,6} It would probably have been futile in this patient, who had thrombocytopenia and relapsed acute myeloid leukaemia. Thus, anticoagulation with heparin and warfarin was used. Fibrinolytic agents like streptokinase or urokinase would have been considered but the risks of a major haemorrhage with thrombocytopenia precluded their use. We are unaware of any report of its use in this situation. The improvement in circulation was probably due to restoration by collaterals and flow through an extremely narrowed aortic lumen.

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