

A new manifestation of thrombocytopenia: myocardial haemorrhage with symptomatic arrhythmia

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SUMMARY We describe a patient with thrombocytopenia who developed episodes of dyspnoea due to recurrent cardiac arrhythmia. Necropsy revealed that the apparent mechanism was extensive myocardial haemorrhage.

Complications of thrombocytopenia, such as cutaneous bruising, intracerebral and gastrointestinal haemorrhage are well recognised. Subendocardial and pericardial haemorrhage is a frequent necropsy finding in thrombocytopenic patients. However, the findings are generally considered to be of no clinical significance. We describe a patient with thrombocytopenia who developed breathless attacks for several weeks before death. Twenty-four hour electrocardiograph recordings showed intermittent cardiac arrhythmias and necropsy demonstrated the apparent mechanism was myocardial haemorrhage.

Case report

In 1971, a 49-year-old woman was found to have essential thrombocythaemia and was treated with ³²P (total dose 33 mCi).

She presented again in May 1980 with tiredness and easy bruising. Investigations showed a haemoglobin concentration of 11 g/dl and a platelet count of $10 \times 10^9/l$. Bone marrow examination revealed myelofibrosis with an increase in blast cells. Some remission in her symptoms was achieved by treatment with vincristine and prednisolone. However, she remained severely thrombocytopenic with skin purpura and episodes of epistaxis and she required platelet transfusion. She was transfused a total of 20 single donor units of platelets.

In August 1980, she developed recurrent transient episodes of dyspnoea lasting for up to several minutes. There were at first irregular occurring no more than twice weekly but subsequently became more troublesome, increasing in frequency to several times daily. Examination during an attack showed tachycardia (apical rate 120–150/min) but

no evidence of clinically detectable pulmonary oedema. A continuous twenty-four hour electrocardiograph revealed that the dyspnoea was associated with sinus tachycardia; this was followed by gradual slowing and the development of an atrioventricular (AV) junctional (nodal) escape rhythm with the occasional ventricular extrasystole. Plasma sodium, potassium, calcium and magnesium, serum thyroxin and acid base balance were normal. The cause of the arrhythmia was uncertain and the episodes of dyspnoea continued up to the time of her death in September 1980. The attacks were not related to platelet transfusions and the patient was not taking drugs.

NECROPSY

Necropsy demonstrated that the immediate cause of death was a massive gastrointestinal haemorrhage associated with numerous petechiae in the gastric mucosa. There were also scattered petechiae in the skin, brain, and renal pelvis.

The heart macroscopically (Fig. 1) showed numerous haemorrhages on the epi- and endocardial surfaces of the right atrium, right atrial appendage and at the base of the interatrial septum. The conducting system was dissected using the method described by Davies² and histological examination showed severe widespread haemorrhage into the myocardium of the right atrium with distortion and separation of the muscle fibres (Fig. 2). Haemosiderin-laden macrophages indicated that previous haemorrhagic episodes had occurred. Haemorrhage was also present around the nerve fibres of the subpericardial plexus and in the fibrous ring adjacent to the AV nodal artery. The sinoatrial (SA) artery, muscle fibres of the SA and AV nodes and bundle of His were normal.

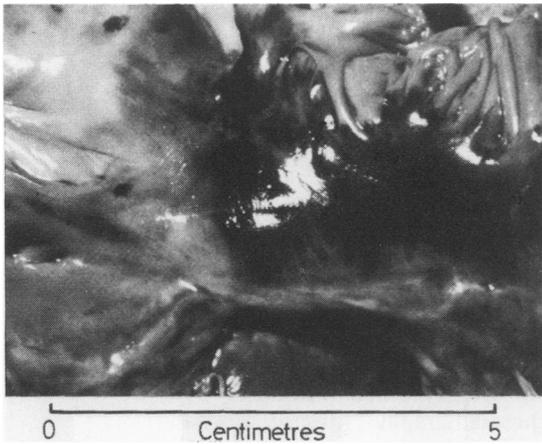


Fig. 1 Right atrial haemorrhage above the tricuspid valve.

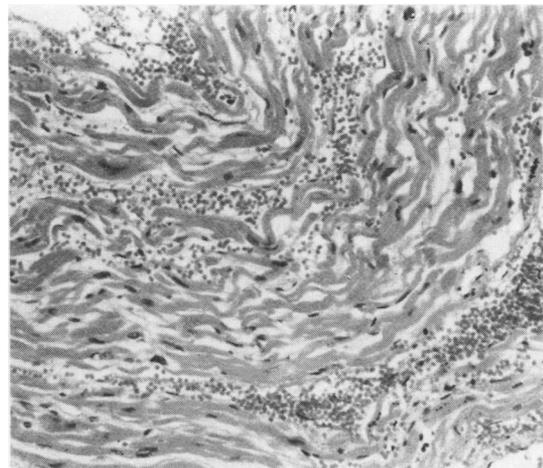


Fig. 2 Extensive right atrial haemorrhage causing separation and distortion of the myocardial fibres. Haematoxylin and eosin $\times 295$.

The left atrial muscle and ventricular myocardium were largely devoid of haemorrhage. All coronary arteries showed less than 5% luminal occlusion by atheroma and the lungs were histologically normal.

Discussion

The terminal phase of our patient's disease was characterised by thrombocytopenia. She was transfusion-dependent and showed intermittent cutaneous haemorrhagic signs. The troublesome transient episodes of dyspnoea were difficult to understand and it was not until the twenty-four hour electrocardiograph was performed that a relation was shown between the dyspnoea and an arrhythmia.

Myocardial haemorrhage due to operative trauma in patients undergoing cardiac surgery is recognised to be as a cause of atrial arrhythmia³ and complete heart block.⁴ Arrhythmias have also been associated with haemorrhage in a patient with diphtheria⁵ and it is interesting that Menzies states that traumatic myocardial contusion is consistently associated with a tachycardia.⁶

Necropsy studies of patients dying from leukaemia have demonstrated the presence of both petechial haemorrhages and cellular infiltrates in the conducting system.⁷ Although the occasional report has shown a relation between leukaemic infiltration and a cardiac arrhythmia,⁸ myocardial haemorrhage has not been previously reported as a cause of arrhythmia in a leukaemic or thrombocytopenic patient.

It is possible that this complication of thrombocytopenia occurs not infrequently and Dresdale

*et al*⁹ suggested that, without specific electrocardiological investigations, various arrhythmias in haematological patients may be overlooked due to a coexistent tachycardia associated with anaemia and pyrexia.

In the absence of any other predisposing factor, it appears probable that the cause of the AV junctional rhythm in our patient was an escape rhythm after disruption of the normal conducting pathways between the SA and AV node.

We are grateful to Professor EK Blackburn for permission to report this case and to Dr J Fleming for interpretation of the 24-hour electrocardiogram.

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