Survival after aortic dissection in giant cell arteritis

Acute aortic dissection in giant cell arteritis is rare and usually fatal. It is associated with hypertension and is commoner in females. We describe two patients who were diagnosed in life and who survived on medical treatment alone for a prolonged period: one for two years; the other still alive at three years. A third patient who required aortic grafting has survived 14 years. A literature review revealed 43 other cases, with only three survivors on medical care alone.

Case reports

PATIENT 1
A 68 year old white male presented with acute chest pain on exertion radiating to the back, accompanied by dyspnoea and sweating. He had noted weight loss of more than 7 kg over the previous months. A history of back and neck ache was ascribed to spondylitis, and shoulder pain to a rotator cuff lesion. His pulse was 60 beats/min regular, and his blood pressure was 150/70 mm Hg. An ejection murmur was noted at the left sternal edge. Examination was otherwise unremarkable.

The patient developed intermittent pyrexia over the next three weeks, and experienced frequent left sided stabbing chest pain. The leucocyte count increased from normal to 14.5 with a neutrophilia. Electrocardiogram and echocardiogram showed left ventricular hypertrophy, confirmed by thoracic echocardiogram, which showed no other abnormality. Chest radiograph showed minor atelectasis at both lung bases. Indium leukocyte scan showed some mediastinal uptake. Computed tomography (CT) of the thorax, without intravenous contrast medium (because of asthma), showed a normal mediastinum but a small rim of pleural fluid on the left.

At follow up, the patient complained of right thigh claudication. Radio femoral delay was noted. CT of the abdomen showed internal displacement of calcified atheroma in the aorta, suggesting aortic dissection. Magnetic resonance imaging (MRI) confirmed this as distal to the origin of the left subclavian artery, extending to the aortic bifurcation. Eight weeks after discharge, he developed headaches, aching thighs and jaw claudication with temporal artery tenderness. Prednisolone 30 mg daily was begun with rapid improvement. Clinical improvement correlated with his ESR decreasing to normal and with gradual resolution of the radio femoral delay.

PATIENT 2
A 68 year old white women presented with five days of worsening anterior pleuritic chest pain radiating to the left, sweats, nausea, and an increased temperature. She had a history of osteoarthritis, eutrophic Graves’ ophthalmopathy, Raynaud’s phenomenon, and longstanding hypertension. Temporal arteritis had been diagnosed on biopsy eight years previously, following a characteristic illness accompanied by an ESR of 121 mm/1st h. Prednisolone for three years had achieved remission.

On admission, the patient was pyrexial at 37.7°C. The heart rate was 110 beats/min with no radiofemoral delay, and the blood pressure was 140/90 mm Hg, equal in both arms. There was dullness on percussion at both lung bases and bronchial breathing at the left base. ESR was 86 mm/1st h, and the leucocyte count 11.4 with a neutrophilia. ECG showed left ventricular hypertrophy. Chest radiograph showed bilateral small pleural effusions and a markedly widened mediastinum. CT of the thorax and abdomen revealed a dilated thoracic aorta with a dissection in the descending portion. Transoesophageal echocardiography (TOE) initially suggested dissection commencing at the aortic root; however, repeat CT, arch aortography and MRI confirmed type B dissection. The patient was treated with a β blocker to maintain normal blood pressure, and prednisolone at a dose of 20 mg. The pain resolved and she was discharged, remaining well and normotensive for two years. Mediastinal widening then began to accelerate, MRI imaging confirming a grossly dilated dissecting descending aorta and dilatation of the ascending aorta (figure). She died soon afterwards.

Most of these patients showed evidence of hypertension in life or at postmortem examination. Twenty-two of 27 reported cases were histologically confirmed - disproportionate to the 1:2 male:female ratio in giant cell arteritis. The gender of 16 cases with aortic dissection in the large series reported by Evans et al.\(^7\) was not specified.

The diagnosis of acute aortic dissection may be made by four different imaging techniques. Retrograde aortography has a sensitivity of only 81–91%, though specificity may be more than 90%.\(^8\) It can demonstrate involvement of branch vessels and coronary arteries. CT has a sensitivity of 83–100% and a specificity of 90–100%. MRI has been shown to be up to 100% sensitive and specific in some studies. TOE may be 97–100% sensitive, but specificity as low as 68% has been reported, probably reflecting operator dependence.

The cases described had clinical evidence of giant cell arteritis, biopsy proven in two cases, and aortic dissection. The two presentations with fever and chest pain did not immediately suggest the diagnosis. Clinicians should be aware of potential life threatening large vessel disease in giant cell arteritis, particularly in female patients with hypertension. The outcome may be more favourable than previously believed, if the arteritis is adequately controlled with steroids. The diagnosis of giant cell arteritis should be considered in patients presenting with acute dissection of the aorta.

**Correspondence to:** Dr A M L Lever.


**Antibodies to collagens in sera from patients receiving bovine cartilage graft.**

The production of autoantibodies against different collagen types has been described in association with several systemic rheumatic pathological entities. Antibodies to collagen type II have been demonstrated in relapsing polychondritis and in rheumatoid arthritis.1,2

Cartilage grafts for tissue reconstruction have been used in many surgical specialties, especially in otorhinolaryngology and maxillofacial surgery. Autograft cartilage has the tendency to warp. Further, harvesting cartilage is time consuming. The material of the harvest requires an additional operative procedure. Recently, we observed humoral reactivities against cartilage antigens in patients with degenerative nasal deformities. These observations have led to investigations of autologous cartilage grafts.3 These
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M P Richardson, A M Lever, A M Fink, A K Dixon and B L Hazleman

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