CASE REPORT

A 32 year old man presented to the accident and emergency department with mild central chest pain. There was no history of precipitating factors, radiation of pain, vomiting or breathlessness. He gave a past history of non-Hodgkin’s lymphoma, which was treated by radiotherapy and chemotherapy 10 years earlier.

While being assessed, the patient suddenly complained of severe pain and loss of function in the lower limbs. Examination of the right lower limb revealed complete loss of power, reflexes and sensation below the knee. In the left lower limb the power was reduced to 3/5 but reflexes and sensation were normal. He had adequate perfusion and normal pulses in the lower limbs. The radial pulses were unequal with the left being weaker. The blood pressure in the right upper limb was 105/59 mm Hg and in the left was 85/59 mm Hg. Examination of the abdomen and chest showed no abnormalities. There was no bladder or bowel involvement. ECG showed a first degree heart block and the chest radiograph was unremarkable. His white cell count was raised at 14.6 x 10^9/l. Half an hour later, he continued to complain of mild dull aching central chest pain but there was full resolution of the lower limb symptoms. Re-examination of the lower limbs showed no evidence of neurological deficit. He was kept for observation in the department and repeated measurements indicated that the blood pressure returned to equilibrium with 111/73 mm Hg in the right upper limb and 108/63 in the left. Costochondral chest tenderness was also observed.

Although the possibility of aortic dissection was initially considered, resolution of clinical signs led to diagnosis of musculoskeletal chest pain being considered. The patient’s condition remained stable. When the initial clinical presentation was reviewed a few hours later, it was felt that diagnosis of aortic dissection was very likely. The diagnosis was subsequently confirmed by computed tomography of the chest and abdomen, which showed an aortic dissection extending from the aortic root, into the left subclavian artery and through the full length of the thoracic and abdominal aorta to the aortic bifurcation. Contrast was seen on both sides of the intimal dissection (fig 1).

The patient was immediately transferred to the cardiothoracic unit. He was found to have a congenital bicuspid aortic valve and underwent a successful operation with prosthetic aortic root replacement, coronary re-implantation and ascending aorta replacement with extending open distal anastomosis. He eventually made a satisfactory recovery.

DISCUSSION

Presentation of aortic dissection with neurological manifestation is not uncommon and the incidence varies from 18%–29% in different studies. Neurological abnormalities as a result of aortic dissection can be accounted for by three factors, ischaemia of the brain, ischaemia of the spinal cord and ischaemia of the peripheral nerves. It is most likely that our patient had ischaemia to the spinal cord, as there was no radiological evidence for extension of the dissection into the carotids or into the femoral arteries. Although in most patients, neurological deficits from aortic dissection are permanent, there are two previously reported cases of complete recovery of paraparesis. Waltimo and Karli reported another case of aortic dissection with paraparesis, which recovered the same day although no reference to the duration of the paraparesis was made in that report. The uniqueness of our case is the brief duration of the “transient ischaemic attack of the spinal cord”, which lasted for only half an hour. As far as we are aware, this has not been reported before.

Rosen suggested a possible mechanism for the transient nature of the paraplegia. An important source of blood supply to the lower spinal cord is the radicular artery called the artery of Adamkiewicz. Rather than thrombosis or shearing of the artery, a transient occlusive valve phenomenon attributable to progression of the dissection could have occurred, leading to transient ischaemia of the lower spinal cord explaining the clinical presentation. In this case the cause of dissection was congenital bicuspid aortic valve, which is known to predispose young patients to dissection.

Some clinical features posed diagnostic difficulties in this case of aortic dissection. These included absence of history of hypertension, young age, presence of costochondral tenderness, transient nature of neurological signs and the subsequent absence of significant blood pressure difference in both
arms. The past history of non-Hodgkin’s lymphoma was misleading and could have delayed diagnosis if the patient would have been investigated on the lines of a recurrence. However, the presence of chest pain, the initially observed difference in radial pulses and blood pressure between the arms suggested the possibility of aortic dissection. The marginally raised white cell count and the non-specific ECG changes, which are known to occur with aortic dissection, further pointed towards the diagnosis and resulted in prompt investigation and treatment, which, if delayed, could have proved fatal.

This report highlights the importance of considering the diagnosis of aortic dissection when a history of transient neurological symptoms in the lower limbs is obtained, even in the absence of abnormal neurological findings and irrespective of the patient’s age. Costochondral tenderness and absence of blood pressure difference in the upper limbs should not be interpreted as an evidence for less sinister causes of chest pain.

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REFERENCES