PREOPERATIVE ENDOVASCULAR EMBOLISATION OF A VERTEBRAL HAEMANGIOMA

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We describe the successful relief of compression of the spinal cord due to a vertebral haemangioma by transcatheter embolisation using cyanoacrylate compounds before operation, and provide a brief review of the literature.

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Embolisation for vertebral haemangioma causing paraplegia was first reported in 1972.¹ The traditional treatment in these circumstances had been surgical decompression, with or without postoperative radiotherapy. Embolisation is playing an increasingly important role since it can avoid the need for surgery in some cases.^{1,2} A combined approach with preoperative embolisation minimises bleeding during operation and reduces the risk of a postoperative epidural haematoma.

CASE REPORT

A 33-year-old Caucasian man noted paraesthesiae in both feet after performing leg presses in the gymnasium. His legs became increasingly stiff, the paraesthesiae became worse and he had difficulty in walking. He also had some low back pain which increased on lying down, but he did not experience problems with his bladder or bowel. Examination showed a spastic paraparesis with grade-IV weakness in both legs, worse on the left. He had patchy sensory loss in both legs, with clonus at both ankles and extensor plantar responses.

MRI showed haemangiomatous replacement of the T10 vertebra with a soft-tissue component compressing the spinal cord (Fig. 1). A high signal was present in the cord at that level consistent with oedema.

The patient underwent spinal angiography and embolisation. A size 5 French Cobra catheter (Cook UK Ltd, Letchworth, UK) was inserted via the right femoral artery and selective catheterisation of the intercostal arteries supplying T9 to T11 was performed. The spinal artery arose from the left T12 intercostal artery. The arterial supply to the haemangioma was from both the left and right T10 intercostal arteries with a large feeder from the right, less supply from the left, and small branches from the right T11 vessel. Embolisation was carried out as a two-stage procedure over two days. First, occlusion of the feeder from the right side of the T10 intercostal artery was carried out with 0.5 ml of cyanoacrylate/lipiodol (50:50) injected through a Tracker 18 (Target Therapeutics, St Albans, UK) catheter placed into the nidus of the haemangioma. On the next day, the left T10 intercostal artery was occluded with 0.4 ml of cyanoacrylate/lipiodol (50:50) since it was impossible to catheterise selectively the small feeders which arose from this artery. A small vessel from the right T11 artery feeder was also occluded with 0.2 ml of cyanoacrylate/lipiodol (50:50). After embolisation no arterial supply to the haemangioma was visible angiographically (Figs 2 to 5).

MRI was performed after the embolisation. A high signal return was again seen at T10, consistent with replacement of the vertebral body by haemangiomatous tissue. The soft-tissue component previously seen within the canal posteriorly showed marked resolution, with minimal compression of the cord. Although a high signal was again seen within the posterior aspect of the cord at the level of T10, the extent was less, indicating that the oedema was resolving (Fig. 6).

The relative youth of the patient gave a high chance of recurrence, fracture and compression. For this reason, vertebrectomy and stabilisation with an iliac-crest bone graft was performed.

At operation the vertebral body was drilled out, leaving a partial shell of cortical bone. The cancellous bone of the vertebra was coarsely trabeculated and thrombosed vessels were seen. Peroperative bleeding was minimal. After operation, the patient made very good progress and is now fully mobile, with no residual neurological deficit.

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Fig. 1a

Fig. 1b

Pre-embolisation MRI. Figure 1a – T1-weighted sagittal image of the thoracic spine, showing a high signal in the vertebral body of T10 consistent with haemangioma. An extradural component with a high signal is seen to compress the spinal cord. Figure 1b – T1-weighted axial image at T10. There is a high signal within the vertebral body, with paravertebral spread (white arrow) and an extradural component (curved black arrow) causing marked cord compression.

Fig. 2

Fig. 3

Fig. 4

Fig. 5

Figure 2 – Pre-embolisation digital subtraction angiography (DSA). A Tracker 18 microcatheter has been inserted via a guiding catheter and the tip advanced to the nidus of the haemangioma at T10 via the right T10 intercostal artery. Angiography shows a vascular blush in the haemangioma. Figure 3 – Postembolisation DSA with cyanoacrylate compounds. There is no residual vascular blush, indicating successful embolisation of the supply from the right side. Figure 4 – Pre-embolisation DSA. A microcatheter has been inserted into the left T10 intercostal artery showing further vascular supply to the right side of the haemangioma. Figure 5 – Postembolisation DSA. Most of the vascular supply has been occluded by the cyanoacrylate/lipiodol compounds.

Fig. 6a

Fig. 6b

Postembolisation MRI. Figure 6a – A T1-weighted sagittal image of the thoracic spine showing marked resolution of the high signal in the vertebral body of T10. The extradural component has largely resolved. Figure 6b – A T1-weighted axial image at T10. The vertebral body now shows a mainly low signal with some residual high signal in the left posterolateral aspect. The extradural component has largely resolved with no evidence of cord compression.

DISCUSSION

The incidence of vertebral haemangioma in the thoracic spine at postmortem is about 11%,^{3,4} but most have been asymptomatic. Compression of the cord or cauda equina may occur, however, if there is expansion of the vertebra, extension of the haemangioma into the extradural space, a compression fracture of the vertebra or a secondary extradural haematoma.^{5,6} Spinal-cord compression in our patient was due to extradural tumour. This was well shown on MRI which has replaced myelography as the primary means of imaging cord compression.

Various methods of treatment are available including embolisation, surgery, and a combination of embolisation followed by surgery or radiotherapy.

We agree with other authors⁷⁻¹⁰ that embolisation should be the treatment of choice, because it may avoid the need for surgery. Endovascular embolisation may fail as the sole treatment for cord compression either because of tortuous vessels or if the arterial supply to the cord arises from the feeding intercostal arteries or tiny feeders and capillaries which cannot be occluded. When embolisation is performed before surgery, intraoperative bleeding is greatly decreased, reducing the mortality which used to be encountered when operating on these tumours.

Embolisation has been carried out by percutaneous methods using methylmethacrylate,¹¹ intraoperatively with methylmethacrylate¹² and endovascularly using sponge fragments,¹³ but to our knowledge, endovascular embolisation with cyanoacrylate components has not

previously been described.

Selective spinal angiography is essential for demonstrating the arterial supply to the tumour and the artery of Adamkiewicz, which if inadvertently embolised will lead to infarction of the spinal cord.

Cyanoacrylate compounds are often used for embolisation of intracranial arteriovenous malformations, since they have low tissue reactivity and toxicity, and their liquid consistency allows easy injection into small-bore catheters. Unlike methylmethacrylate compounds there is no late solidification which may make surgery difficult.¹¹ The use of microcatheters allows easier access to the nidus, increasing the chance of success of embolisation.

We have demonstrated an excellent result with pre-operative endovascular embolisation using cyanoacrylates. Transcatheter embolisation has a low morbidity and mortality in experienced hands, and can now complement surgery in the management of arteriovenous malformation of bone¹⁴ and in vertebral haemangioma.

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