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Recurrent spontaneous spinal epidural haematoma treated with and without surgery

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1. Introduction

Spontaneous spinal epidural haematomas (SSEHs) are a rare cause of spinal epidural haematoma (SEH) in the paediatric population. The accumulation of blood within the epidural space causes compression of the spinal cord and signs and symptoms of sensory and/or motor deficits. The potential causes include hypertension, coagulopathies, increased venous pressure and vascular malformations. We report a case of SSEH that initially resolved with non-surgical management, but recurred, was further investigated and was then treated with surgical decompression.

2. Case report

A nine-year old, previously well boy of Chinese decent presented to a peripheral New Zealand hospital, with a two month history of upper thoracic back pain followed by sudden onset of weakness. Five hours later, upon arrival at Starship Hospital, Auckland, clinical examination found tenderness over vertebra T3–T6, 3/5 power to all myotomes in the right leg and 3/5 power to the left leg, excluding ankle and toe dorsiflexion and plantar flexion which had 4/5 power. He had bilateral numbness below the umbilicus, symmetrical brisk reflexes and bilateral up going plantar reflexes. Perianal sensation was intact, but he was unable to pass urine and an indwelling catheter drained 150 ml residual bladder volume.

Initial investigations included thoracic and lumbar radiographs, and a full coagulopathy screen, all of which was normal. Subsequent spinal MRI revealed a posterolateral epidural haematoma, extending from T1 to T5 vertebral body levels, and mild cord compression, without intrinsic oedema (Fig. 1). After the scan the neurological findings began to improve. Power returned to 5/5 bilaterally, light touch sensation returned and catheter sensation was intact. After discussion with the Neurosurgical team, the decision was made to treat this boy conservatively. He commenced 2 mg dexamethasone four times a day for three days, followed by 1 mg twice a day for two weeks. A repeat MRI showed near complete resolution of the left posterolateral epidural haematoma. Fig. 2. The child made a complete recovery and was discharged.

This child then represented 6 months later with pain over his lower thoracic spine and tingling in both legs and the saddle area, after a 10-h plane flight. An MRI of the spine found a small rebleed at the site of the original epidural haematoma, in the mid-thoracic spine, but no extraaxial lesion or cord compression (Fig. 3).

Clinical findings settled with conservative management, but three days later our patient had a further episode. An MRI and contrast enhanced time resolved MR angiogram of the spine was performed, Fig. 4. The latter demonstrated a small epidural AV malformation at the T2/3 vertebral body level. Subsequently a formal digital subtraction angiogram was performed to confirm the findings and acquire more detail regarding the feeding vessels (Fig. 5).

He did not present again acutely, but three months later underwent an elective left sided T3 hemilaminectomy and obliteration of the dural arteriovenous fistula. There were no operative complications. Post-operatively he complained of transient altered sensation over his entire body, which settled completely prior to discharge. A follow up spinal angiogram showed no evidence of any residual AV fistula.

Our patient has had no further presentations and has remained neurologically intact.

3. Discussion

SSEH's are a rare cause of spinal cord compression. They are most common after the fourth and fifth decades,¹³ with few cases reported in children.¹³ The most common spinal level affected in children is from C5 to T1¹³ and there is no gender predominance.¹³ The haematoma is often confined to two or three vertebral segments, with the majority in a dorsal location, because the dural sac is fixed to the posterior longitudinal ligament ventrally.³

MRI is a rapid and accurate method for the diagnosis of SSEH.^{4,16} It is able to identify the location of the lesion, its extent and the



Case report

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Fig. 1. MRI. (a) Sagittal T1 flair image reveals a left posterolateral epidural collection which is isointense to the spinal cord. (b) On Sagittal T2 the epidural collection is centrally hypointense. Signal intensity is consistent with central deoxyhaemoglobin.

degree of cord compression and oedema. The T1 and T2 signal intensity changes with the age of the haematoma. Hyperacute haematoma is isointense on T1 weighted images and hyperintense on T2 weighted images, consistent with oxyhaemoglobin. Later the haematoma will return iso or high signal on T1 weighted images and low signal on T2 weighted images, reflecting deoxyhaemoglobin and later intracellular methaemoglobin.¹⁶

The cause of SSEH's is not always clear. Some authors believe the bleeding is venous in origin, with rupture of the valveless epidural plexus veins due to increased intra-abdominal or intrathoracic pressure.^{13,2} There is an association with the use of anticoagulants¹⁵ and coagulation disorders.¹³ Spontaneous rupture of arteriovenous malformations has also been suggested as a prominent cause in children.² These can be dural or epidural in location, although this origin can be difficult to diagnose.¹¹ MRI/ MRA has greatly helped in the diagnosis of arteriovenous malformations,⁵ as shown in this case, but angiography still remains the gold standard.^{5,10,12} As well as detection of spinal dural arteriovenous malformations, by localising the level of abnormality, contrast enhanced time resolved MRA can reduce the radiation dose and volume of contrast agent associated with subsequent digital subtraction spinal angiography.⁹

The mainstay of treatment of SSEH's has been surgical decompression and evacuation of the haematoma. The length of



Fig. 2. Sagittal T2 (a) and axial T2 (b) show near complete resolution of the T2 low signal thoracic epidural haematoma. Intradural CSF flow voids are present.

time between onset of symptoms and treatment is important for neurological recovery. Surgical decompression within 36 h for patients with complete spinal dysfunction, and within 48 h in patients with incomplete deficits, has resulted in favourable outcomes.⁶ However, in the patient with minimal neurological deficit or rapidly improving neurological deficit, non-operative treatment may be appropriate.^{7,8,12,13} These patients need to be followed with repeat neurological examination and MRI imaging. If neurological deterioration occurs, or the improvement plateaus at an unacceptable level, then haematoma evacuation is indicated.

Typically a SSEH is an isolated event. There are two documented cases of recurrent SSEH's in children in the literature.^{1,14} The first was in a 10-year-old female who presented with two episodes of transient lower extremity paralysis followed by a third episode that resulted in permanent paralysis.¹ Spinal angiogram revealed "an unusual vascular abnormality" in the epidural space, but only benign fibrous tissue was found at time of explorative laminectomy. The other case was in a 16-year-old female who presented three times with a SSEH in the ventral epidural space who was non-operatively managed for the first episode, followed by decompressive hemilaminectomies at the two further occurrences. She was subsequently found to have syringomyelia at the affected cord level.¹⁴

In this case, the patient presented with spontaneous paraplegia due to a SSEH which resolved without surgical treatment. Further





Fig. 3. Sagittal T2 (a) shows a hypointense T2 epidural collection, which is isointense on T1 axial (b) acquisitions.



Fig. 4. Contrast enhanced time resolved MRA of the thoracic spine shows a tangle of thoracic vessels in the left side of the spinal canal at T2/3.



Fig. 5. Selective DSA demonstrates an AV fistula fed by two small branches passing underneath the left T3 pedicle.

symptoms occurred six months later and MRI/MRA at this time revealed a recurrent epidural haematoma due to an A–V malformation. This case highlights the importance of investigating for vascular malformations and we would strongly recommend initial investigation with a MRI/MRA. This allows the lesion to be surgically treated and prevent further occurrences.

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