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Acute cauda equina syndrome secondary to free fat graft following spinal decompression

Y Imran,Y Halim

ABSTRACT

A 63-year-old man developed acute cauda equina syndrome due to fat graft compression. Following decompressive laminectomy and posterior instrumented fusion with pedicle screw fixation for spinal stenosis of L5 and S1 vertebral levels, free fat grafting was performed to cover the exposed dura. The patient developed gradual neurological deficit three days postoperatively. This started with sensory loss and weakness of the affected dermatomes and myotomes, followed by bowel incontinence on the 12th postoperative day. Intraoperatively, significant dural compression by the fat graft was confirmed. Immediate removal of the fat graft resulted in recovery from cauda equina syndrome.

Keywords: cauda equina syndrome, free fat graft, postoperative complications, spinal surgery

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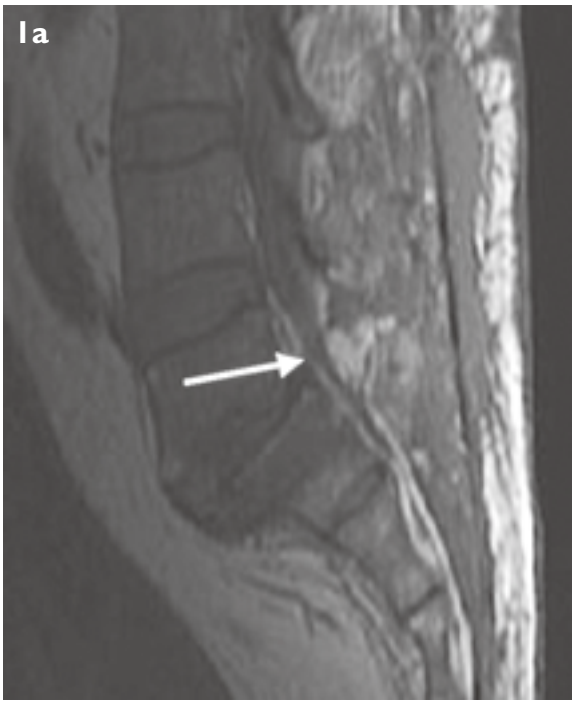
INTRODUCTION

Laminectomy is a common surgical procedure for spinal decompression. As leaving the dura exposed may cause dural adhesion and fibrosis, autologous free fat grafting is routinely performed following laminectomy to prevent these complications. Its main advantages are its efficiency, availability and compatibility. Complications following this procedure are rare. We describe a case of acute cauda equina syndrome following free fat grafting in a patient who had spinal decompression for spinal stenosis, and its outcome following its removal.

CASE REPORT

A 63-year-old man presented with symptoms of spinal stenosis of two years duration and a claudication distance of 50m. On examination, neurological deficit was noted at his right L5 and S1 distribution in which his sensation over the dorsum and plantar aspects of his right foot were markedly reduced. His right toe dorsi- and plantar flexion were also weak. Magnetic resonance (MR) imaging showed circumferential disc



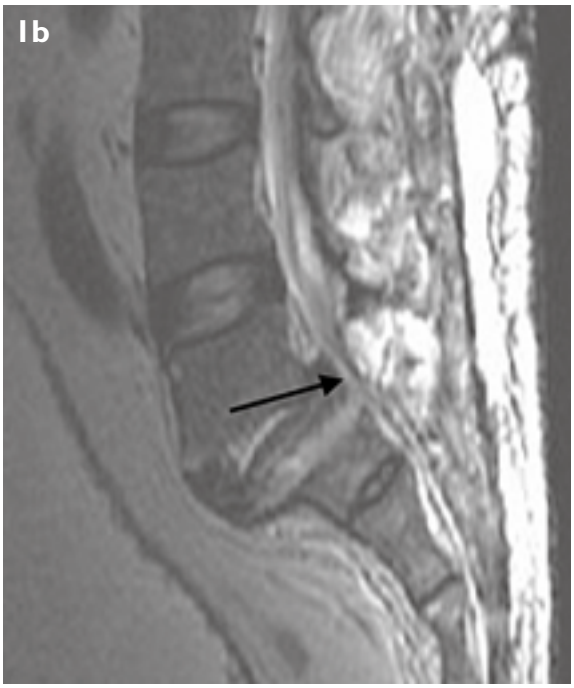


Fig. 1 Sagittal MR images show severe constriction (arrow) of the lumbosacral dura sac by a posterior mass. The mass has (a) mixed high and low signal intensity on T1-W and (b) high signal intensity on T2-W images.

Department of Orthopaedics
School of Medical Science
Universiti Sains Malaysia
Jalan Raja Perempuan Zainab 2
16150 Kubang Kerian
Kelantan, Malaysia

Y Imran, MD, MMed
Lecturer

Y Halim, MBBCh, MMed
Lecturer

Correspondence to:
Dr Imran Yusof
Tel: (60) 9766 1700 ext: 4518
Fax: (60) 9765 3370
Email: drimran93@yahoo.com

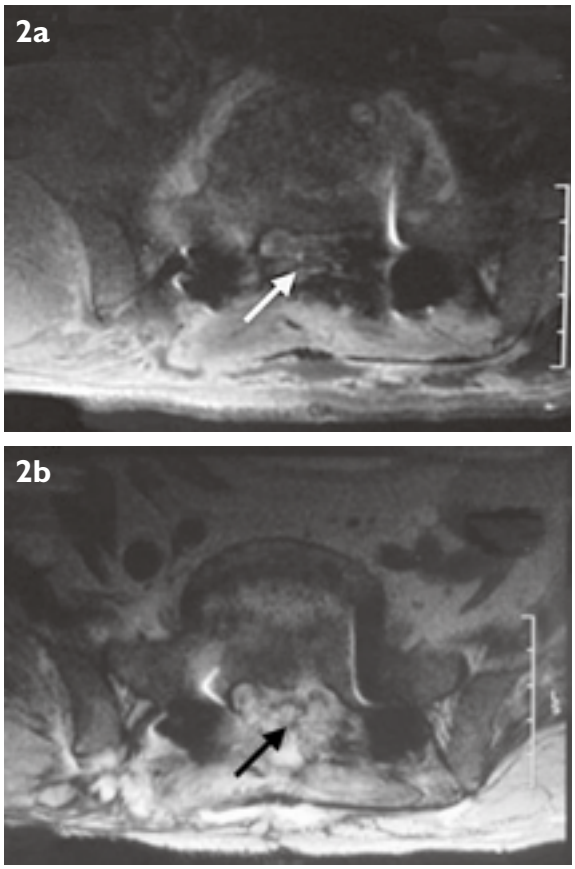


Fig. 2 Axial MR images show severe constriction (arrow) of the lumbosacral dura sac by a posterior mass. The mass shows (a) mixed high and low signal intensity on T1-W and (b) high signal intensity on T2-W images.

prolapse at L5/S1 vertebral level with compression of the thecal sac. Both L5 nerve roots were impinged with thickening of the ligamentum flavum and facet joint arthropathy.

He underwent spinal decompression, posterolateral fusion and instrumentation. Fat graft (3.5 x 2 x 1 cm) was laid over the exposed dura without being sutured to adjacent tissue. Postoperatively, his neurological deficit partially recovered. However, three days postoperatively, he suffered from neurological deficit at the right L5 and S1 dermatomes and myotomes. His condition deteriorated until day 12 postoperatively when he developed bowel incontinence. Urgent MR imaging was performed and fat tissue compressing the dura at the level of spinal decompression was documented (Figs. 1 & 2). Emergency spinal decompression was done and the fat graft was removed. After two days, the patient recovered completely from the cauda equina syndrome. However, there remained weakness in his right extensor hallucis longus and flexor hallucis longus.

DISCUSSION

Postoperative cauda equina syndrome following lumbar decompression is not common. Fat graft, haematoma formation and retained disc material are the possible causes of acute cauda equina syndrome following surgery. Retained intervertebral disc material is unlikely to cause the cauda equina syndrome in this patient as its size was too small to produce significant dura compression in the spinal canal that had been completely decompressed posteriorly[1]. Injury to the cauda equina by the surgical implants would cause cerebral spinal fluid leakage and is therefore identified by the surgeon intraoperatively. Haematoma collection causing postoperative cauda equina syndrome had been reported[2]. However, MR imaging features and intraoperative findings ruled out haematoma collection as the cause of the problem. The early onset of the symptoms of cauda equina syndrome ruled out epidural fibrosis as epidural fibrosis takes some time to develop.

Epidural fibrosis following dura exposure commonly results in failed back syndrome. To prevent this complication, free fat grafting is considered a routine procedure. The efficiency of autologous fat graft in preventing epidural fibrosis had been shown[3]. Its main advantages include its availability, compatibility and viability. The fate of autogenous fat graft after laminectomy had been studied extensively by Kanamori et al[4]. They found that grafted fat tissue survived with increase in collagen fibres and hyperplasia of blood vessels within the graft.

The true incidence of neural compression by fat graft is not known but it is likely to be very low. Autologous fat graft may cause neural compression as early as three days postoperatively, as reported in our case, or as long as six years[5]. Factors that contribute to the incidence are not known. However, the thickness and quality of the graft are important factors. No guidelines exist for determining the size of fat graft[4,6], but based on the facts that it will shrink to 30-50% of its original size, Kanamori et al suggested that the graft should be 5mm thick[4] or thinner[7]. Mayer and Jacobsen recommended fat graft thickness of between one-half and one centimetre thick, to protect the dura sufficiently and to allow for some shrinkage of the graft without the formation of fibrous tissue[6]. However, fat graft of 1cm or greater in thickness have been successfully used in patients without any incidence of dural compression[8].

The other parameters of the graft used would depend on the size of the dural exposure. Graft which is smaller than the bony defect will not serve the purpose. But if it is too big, the graft may be infolded into the canal and might increase its thickness,

causing dural compression. Dural compression in the early stage secondary to graft hypertrophy is very unlikely. As reported by Kanamori et al[4], during the first one year, it undergoes hypotrophy. Fat graft hypertrophy was noted at 10 years follow-up in another study[9].

Fat graft displacement that causes dural compression has been reported[10]. In our practice, fat graft is not routinely sutured to the adjacent tissue to prevent this complication and we have not encountered any previous incidence of neurological complication. However, based on this experience and another report[7], we have now considered the possibility of routinely placing a suture in the fat graft to prevent similar complication. Preserving the ligamentum flavum to avoid the use of fat graft may not be applicable in all cases. In degenerative spinal stenosis, the hypertrophied ligamentum flavum needs to be excised. The use of fat graft is still advantageous, although there are possible complications. However, the early recognition of fat graft causing acute cauda equima syndrome is important, as immediate surgical decompression is usually followed by neurological recovery.

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