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Rapid progression of spinal epidural lipomatosis after percutaneous endoscopic spine surgery mimicking disc herniation

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ABSTRACT

INTRODUCTION: Spinal epidural lipomatosis (SEL) is well known but uncommon complication of endoscopic spine surgery. Here, we present a case of SEL that progressed focally and rapidly after endoscopic spinal surgery.

PRESENTATION OF CASE: A 67-year-old man presented with back and Lt. leg radiating pain. MRI of the lumbar spine demonstrated severe foraminal stenosis at Lt. L4/5/S1. He underwent endoscopic posterior foraminotomy at Lt. L4/5/S1. After surgery, his leg pain disappeared. A month after surgery, the patient developed Lt. leg pain again. MR images showed focally progressed epidural fat posterior to the L5 body. After removal of lipomatosis via endoscopic posterior decompression at L3/4/5, his symptoms improved.

DISCUSSION: Mostly, the epidural fat accumulates insidiously, and distributes widely across several levels. And the increased accumulation of fat is predominantly posterior and posterolateral within the spinal canal. As in this case, focally progressed SEL anterior to the dural sac is rare. And focally progressed SEL can be misdiagnosed for disc herniation.

CONCLUSION: SEL should be recognized as a rare complication of endoscopic spine surgery.

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1. Introduction

Spinal epidural lipomatosis (SEL) is characterized by the overgrowth of epidural adipose tissue inside the spinal canal and may present with symptoms of spinal stenosis or nerve root compression [1]. SEL was first reported in 1975 with the use of corticosteroids to prevent rejection of a kidney transplant [2]. Known causes of SEL are exogenous steroid use, exposure to endogenous steroid resulting from endocrine abnormalities, obesity, and idiopathic [3]. There have been several reports of SEL occurring after open spinal surgery [4,5]. However, we have found no previous reports of SEL that occurred after endoscopic spine surgery. We present a case of SEL that progressed focally and rapidly after endoscopic spinal surgery. The work has been reported in line with the SCARE criteria [6].

2. Presentation of case

A 67-year-old man presented with spinal claudication, back and Lt. leg radiating pain. MRI of the lumbar spine demonstrated severe foraminal stenosis at Lt. L4/5/S1 (Fig. 1). He got three times of nerve root block at the pain clinic before visiting our hospital.

His medical history included hypertension and depressive mood disorder those were well controlled with antihypertensive and antidepressant drugs. The patient was not obese (Body mass index, BMI; 24.7 kg/m²) and denied any previous systemic steroid treatments. We performed endoscopic posterior foraminotomy at Lt. L4/5/S1 (Fig. 2). After surgery, his leg pain disappeared. Postoperative MR images also showed enlarged foramen at Lt. L4/5/S1 (Fig. 3). A month after surgery, the patient developed Lt. leg pain again. The patient was treated with medications, but his symptoms persisted. MRI was checked at five months postoperatively and showed focally progressed epidural fat posterior to the L5 body, causing tight spinal canal stenosis and compressing the cauda equina (Fig. 4). The MRI performed before and after L4/5/S1 foraminotomy surgery did not show the SEL (Fig. 5). After removal of lipomatosis via endoscopic posterior decompression at L3/4/5 (Fig. 6), his symptoms improved. Histologic analysis revealed unencapsulated adipose tissue.

3. Discussion

Epidural fat is adipose tissue located within the epidural space, that is, the area located between the dura mater and the vertebral wall of the spine. Epidural fat buffers pulsatile dural sac movements, allows sliding of the dural sac along the surface of the vertebral arch during flexion and extension, and protects the dural sac during lashing and rotational motions [7,8].

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Fig. 1. Sagittal MR image of the lumbar spine demonstrated severe foraminal stenosis at Lt. L4/5/S1.



Fig. 3. Postoperative MR image showed enlarged foramen at Lt. L4/5/S1.

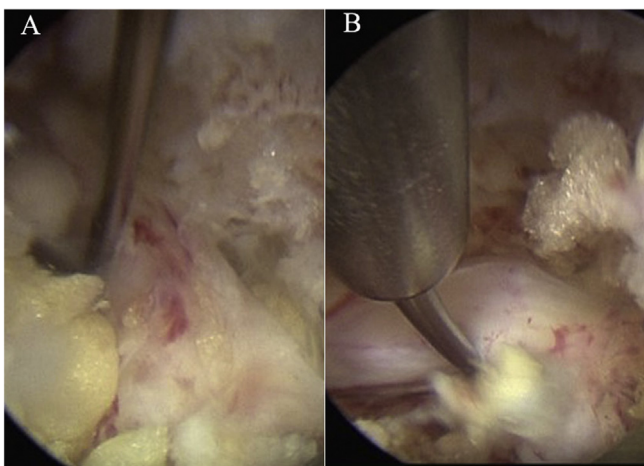


Fig. 2. A flexible probe under endoscopic view was used to confirm the decompression by mobilizing the Lt. (A) L4/5 and (B) L5/S1 exiting nerve roots.

However, excessive amounts of epidural fat could lead to compression of neighbouring structures. Spinal epidural lipomatosis (SEL) refers to an excessive accumulation of fat within the epidural space.

Mostly, the epidural fat accumulates insidiously, and distributes widely across several levels. And the increased accumulation of fat is predominantly posterior and posterolateral within the spinal canal. As in our case, focally progressed SEL anterior to the dural sac is rare. And focally progressed SEL anterior to the dural sac can be misdiagnosed for disc herniation [9].

SEL can be classified into 5 main categories according to pathogenesis: exogenous steroid use, endogenous steroid hormonal disease, obesity, surgery induced, and idiopathic [1].

Many studies have reported that obesity is an imputed cause of spinal epidural lipomatosis. In 1959, Ramsey [10] stated that obese patients had more epidural fat than normal individuals and other authors supported the hypothesis that obese patients would therefore, be at higher risk of spinal canal stenosis [11–13]. Jaimes and

Rococo [14] reported that the probability of developing EL is linear with increasing BMI between 28 and 35. Obesity leads to an increase in 11 β -HSD-1 which increases cortisol by the local conversion of corticosteroids to the biologically active cortisol [15]. Elevated cortisol could raise blood glucose concentration, and the raised blood glucose will stimulate insulin release. Then, insulin stimulates lipogenesis. The prevalence of SEL is greater than initially thought with the current literature suggesting a rate between 6.2% and 8.6% [16]. This may represent a combination of increased imaging, rising obesity, and an aging population. Valcarenghi et al. also reported the case of a 48-year-old obese man with a history of chronic back pain and sciatica secondary to SEL, which subsequently resolved completely after sleeve gastrectomy over a 6-month follow-up period [17]. Idiopathic SEL denotes patients without a history of systemic steroid therapy or endocrinopathy; however, the vast majority of idiopathic cases have an increased BMI [18].

It is believed that corticosteroids cause epidural lipomatosis by inducing hypertrophy of adipose tissue that is normally present in the epidural space of the spinal canal [19]. Administration of large amounts of glucocorticoids is known to cause progressive hypertrophy of fat in the neck, face, and trunk [20]. The exact mechanism by which glucocorticoids preferentially induce hypertrophy of adipose tissue in selected areas of body, including the epidural space has not been clearly elucidated. And, the pathogenesis has not been fully explained the induction of epidural lipomatosis by local epidural injections. But Jaimes and Rococo [14] reported that a strong correlation between the number of subsequent epidural steroid injections and SEL occurrence in their logistic regression model. The corticosteroid from the epidural steroid injection might provide the substrate for the formation of cortisol.

In our case, epidural steroid injection and surgery may be considered as causes of SEL. The patient did not use systemic steroids, but had several times of nerve root blocks before surgery, and those injections contained steroid.

There have been some reports of rapid progression of SEL after spinal surgery. Greenish et al. [5] reported SEL that occurred 2 days after bilateral spinal decompression surgery for L4/5 spinal steno-

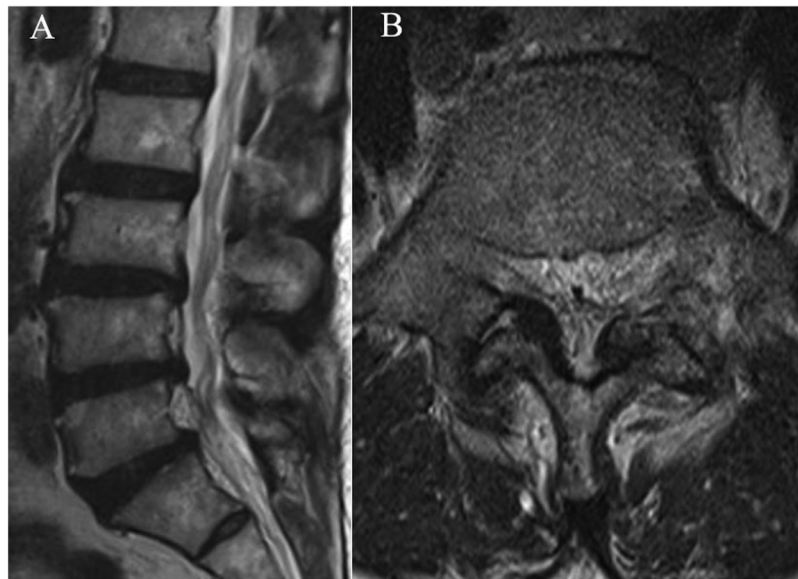


Fig. 4. (A) Sagittal and (B) transverse MR images showed focally progressed epidural fat posterior to the L5 body, causing tight spinal canal stenosis and compressing the cauda equina.

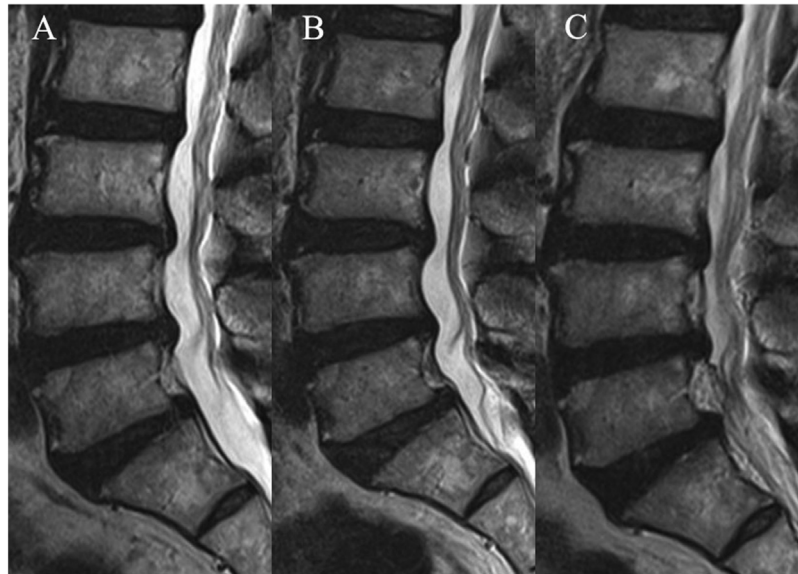


Fig. 5. The MR images performed before and after L4/5/S1 foraminotomy surgery did not show the spinal epidural lipomatosis.

A: Sagittal MR image performed before L4/5/S1 foraminotomy surgery.

B: Sagittal MR image performed after L4/5/S1 foraminotomy surgery.

C: Sagittal MR image performed at 5 months after L4/5/S1 foraminotomy surgery.

sis. The patient in that case had none of the known risk factors for SEL. Choi et al. [4] reported two cases of rapid progression of SEL after spine surgery. Both of those cases underwent epidural steroid injection before surgery. But surgical intervention may induce the accumulation of epidural adipose tissue. Mostly, the epidural fat accumulates slowly and insidiously. But these SEL patients developed neurologic symptoms less than 5 months after surgery. Surgery is a stressor that induces a systemic stress response that includes increased adrenal cortisol production. Several factors like invasiveness of surgery, anesthetic technique and perioperative care can affect the cortisol stress response to surgery [21]. Elevated cortisol levels can lead to lipogenesis by the mechanism described above.

There is no known direct evidence or mechanism that spinal surgery causes SEL. However, considering the rapid progress of SEL after spinal surgery, the study should be carried out with the hypothesis that spinal surgery itself may contribute to the occurrence of SEL.

4. Conclusion

SEL can occur and progress rapidly after endoscopic spine surgery. As in our case, focally progressed SEL anterior to the dural sac could be misdiagnosed for disc herniation. SEL should be recognized as a rare complication of endoscopic spine surgery.

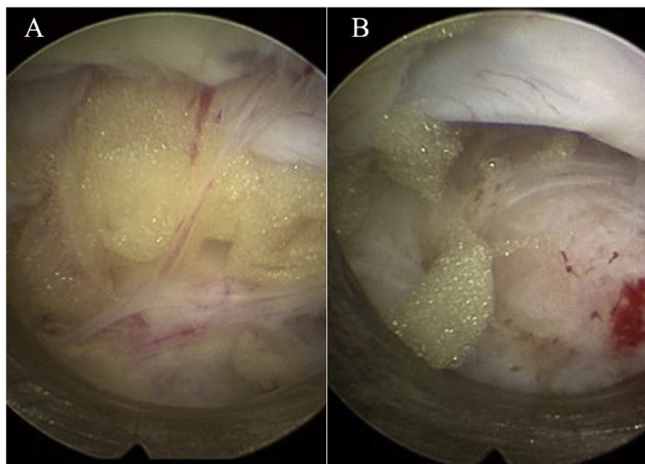


Fig. 6. Endoscopic views (A) before and (B) after epidural lipomatosis removal surgery.

Conflicts of interest

The authors declare no conflict of interest.

Sources of funding

We have nothing to declare.

Ethical approval

This is exempt from ethnical approval in our institution.

Consent

Informed consent was obtained from the patient for publication of this case report and accompanying figures.

Author contribution

Myung Soo Youn: Performing the surgery, Final approval of the version to be submitted.

Young Ha Woo: Acquisition of data, analysis and interpretation of data.

Jong Ki Shin: Conception and design the study, drafting the article.

Registration of research studies

Not applicable.

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References

- [1] K. Kim, J. Mendelis, W. Cho, Spinal epidural lipomatosis: a review of pathogenesis, characteristics, clinical presentation, and management, *Glob. Spine J.* 9 (2019) 658–665.
- [2] M. Lee, J. Lekias, S.S. Gubbay, P.E. Hurst, Spinal cord compression by extradural fat after renal transplantation, *Med. J. Aust.* 1 (1975) 201–203.
- [3] G.R. Fogel, P.Y. Cunningham 3rd, S.I. Esses, Spinal epidural lipomatosis: case reports, literature review and meta-analysis, *Spine J.* 5 (2005) 202–211.
- [4] K.C. Choi, B.U. Kang, C.D. Lee, S.H. Lee, Rapid progression of spinal epidural lipomatosis, *Eur. Spine J.* 21 (2012) S408–S412.
- [5] D. Greenish, K. Watura, I. Harding, Spinal epidural lipomatosis following bilateral spinal decompression surgery, *BMJ Case Rep.* 12 (2019), e226985.
- [6] R.A. Agha, M.R. Borrelli, R. Farwana, K. Koshy, A.J. Fowler, D.P. Orgill, SCARE Group, The SCARE 2018 statement: updating consensus Surgical CAse REport (SCARE) guidelines, *Int. J. Surg.* 60 (2018) 132–136.
- [7] R. Beaujeux, R. Wolfram-Gabel, P. Kehrl, M. Fabre, J.L. Dietemann, D. Maitrot, P. Bourjat, Posterior lumbar epidural fat as a functional structure? Histologic specificities, *Spine* 22 (1997) 1264–1268.
- [8] M.A. Reina, C.D. Franco, A. López, J.A. Dé Andrés, A. van Zundert, Clinical implications of epidural fat in the spinal canal. A scanning electron microscopic study, *Acta Anaesthesiol. Belg.* 60 (2009) 7–17.
- [9] E. Duran, K. Ilik, T. Acar, M. Yildiz, Idiopathic lumbar epidural lipomatosis mimicking disc herniation: a case report, *Acta Med. Iran.* 54 (2016) 337–338.
- [10] H.J. RAMSEY, Fat in the epidural space in young and adult cats, *Am. J. Anat.* 104 (1959) 345–379.
- [11] J.P. Badami, V.C. Hinck, Symptomatic deposition of epidural fat in a morbidly obese woman, *AJNR Am. J. Neuroradiol.* 3 (1982) 664–665.
- [12] A.C. Borstlap, W.J. van Rooij, M. Sluzewski, A.C. Leyten, G. Beute, Reversibility of lumbar epidural lipomatosis in obese patients after weight-reduction diet, *Neuroradiology* 37 (1995) 670–673.
- [13] K. Kumar, R.K. Nath, C.P. Nair, S.P. Tchang, Symptomatic epidural lipomatosis secondary to obesity. Case report, *J. Neurosurg.* 85 (1996) 348–350.
- [14] R. Jaimes 3rd, A.G. Rocco, Multiple epidural steroid injections and body mass index linked with occurrence of epidural lipomatosis: a case series, *BMC Anesthesiol.* 14 (2014) 70.
- [15] R.H. Unger, The physiology of cellular liporegulation, *Annu. Rev. Physiol.* 65 (2003) 333–347.
- [16] A.J. Wells, M.J. McDonald, S.J. Sandler, N.J. Vrodos, Lumbosacral epidural lipomatosis causing rapid onset cauda equina syndrome, *J. Clin. Neurosci.* 21 (2014) 1262–1263.
- [17] J. Valcarenghi, O. Bath, H. Boghal, M. Ruelle, J. Lambert, Benefits of bariatric surgery on spinal epidural lipomatosis: case report and literature review, *Eur. J. Orthop. Surg. Traumatol.* 28 (2018) 1437–1440.
- [18] C.G. Kellett, V. Siva, I.C.F. Norman, J. Jung, G. Grahovac, P. Minhas, Symptomatic idiopathic spinal epidural lipomatosis in 9 patients: clinical, radiologic, and pathogenetic features, *World Neurosurg.* 126 (2019) e33–e40.
- [19] S.C. Robertson, V.C. Traynelis, K.A. Follett, A.H. Menezes, Idiopathic spinal epidural lipomatosis, *Neurosurgery* 41 (1997) 68–74.
- [20] J.N. Fain, Inhibition of glucose transport in fat cells and activation of lipolysis by glucocorticoids, *Monogr. Endocrinol.* 12 (1979) 547–560.
- [21] A. Prete, Q. Yan, K. Al-Tarrah, H.K. Akturk, L.J. Prokop, F. Alahdab, et al., The cortisol stress response induced by surgery: a systematic review and meta-analysis, *Clin. Endocrinol. (Oxf.)* 89 (2018) 554–567.

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