Idiopathic and Chronic Epidural Hematoma in the Lumbar Spine: A Case Report and Review of Literatures

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Abstract

Spontaneous and chronic epidural hematoma (SSEH) in the lumbar spine is rare, and idiopathic and chronic SSEH in the lumbar spine is extremely rare disease. Most of lumbar SSEH were acute and secondary with trauma, hematologic disorders, drug, and surgical procedure. Only 20 cases of chronic SSEH in the lumbar spine have been reported and 14 cases among them were considered to be idiopathic. Definitive guidelines for management of this condition are not clear and surgical total evacuation was performed in most of the cases. Some authors reported the epidural bleeding originates in the rupture of Batson’s plexus due to a rise in intra-abdominal pressure, but the mechanism is not clearly clarified. We report a surgical case of idiopathic and chronic SSEH. A 61-year-old woman suffered a sudden onset of severe lumbar pain during sleep. She had no history of trauma, spinal surgery, or hypertension. Magnetic resonance imaging revealed a lumbar chronic epidural hematoma which compressed the dural sac behind and extended from L2 to L5. This patient underwent the partial evacuation of the hematoma with partial hemilaminectomy on left at L2/3, resulting in immediate pain relief and resolution of symptoms and almost absorption of the hematoma within 1 week of the procedure. We presented this rare case and reviewed idiopathic and chronic epidural hematoma in the lumbar spine.

Key words: idiopathic, epidural hematoma, chronic, lumbar spine

Introduction

Spinal epidural hematoma (SEH) is a rare condition described in associated with trauma, hematologic disorders, anticoagulation and antiplatelet therapy, vascular malformations, neoplasm, trauma, or medical intervention such as epidural catheterization or spinal surgery. Most cases of spontaneous SEH were acute and secondary caused by the aforementioned risk factors. In 2009, Sarubbo et al.1 reviewed spontaneous and idiopathic chronic spinal epidural hematoma in the lumbar spine and one case was reported later in English.2 Surgical evacuation was performed in most of the cases and led to the good results. Here, we report a case of chronic and idiopathic SEH, which was successfully treated with surgical partial evacuation and reviewed about such cases.
Matraz 400), and T2* (TR 600 ms; TE 16 ms; NSA 2; FOV 180; Matraz 320) and surrounded by a thin membrane (Figs. 1b and 1c, Fig. 2a). Gadolinium-enhanced T1 images showed slight enhancement in the membrane (Fig. 2b). Computed tomography angiography (CTA) and magnetic resonance angiography (MRA) did not show vascular malformation and positron emission tomography (PET), CT showed no hot areas in the lesion. Based on the radiological findings, the mass was considered as chronic epidural hematoma and conservative therapy was chosen at first. But, the severe lumbar pain did not improve with enough medicine and bed rest, and surgical procedure was performed 1 week after admission.

A 3.5 cm midline skin incision was made at L2/3 disc space. The fascia was incised left to the midline, sequential dilators inserted and a quadrant retractor (Medtronic, Minneapolis, MN, USA) was placed at L2/3. When the yellow ligament was partially removed after partial laminectomy on left at L2 and L3 (Fig. 3), a jelly-like dark brown hematoma with a capsule was seen (Fig. 4a). After cutting the capsule, the hematoma was carefully removed piecemeal. Histopathologically, the membrane of the capsule was granulation tissue with neogenesis of blood capillary and had hyperplasia of fibroblast and invasion of inflammatory cells and no tumorigenesis (Fig. 4b). The hematoma showed only blood cells and fibrin, and the membrane was adherent to epidural fat tissue. Therefore, the hematoma was diagnosed as chronic SEH.

Lumbar pain was decreased immediately after the operation. Postoperative MRI on day 2 showed the hematoma was removed almost completely at L2/3, but remained mainly at L3/4 (Fig. 5a). The MRI on postoperative day 7 showed that the hematoma...
Fig. 3 Postoperative 3D CT (a) and CT on axial (b).

Fig. 4 Intraoperative image shows an encapsulated fibrous dark brown hematoma (a) and photomicrograph of the membrane shows granulation tissue and epidural fat tissue (b).

Fig. 5 T2 images on sagittal view after operation [(a) 2 days after operation, (b) 7 days, (c) 1 month].
disappeared almost completely and her lumbar pain vanished and the hematoma completely absorbed in 1 month after operation (Figs. 5b and 5c).

**Discussion**

Spinal epidural hematoma is uncommon condition and can be classified as spontaneous, secondary, and idiopathic but the definition was not clear. The most important causes of secondary SEHs are with hematologic disorders, anticoagulation and antiplatelet therapy, vascular malformations, neoplasm, trauma, or medical intervention, such as epidural catheterization or spinal surgery. Spontaneous SEH is defined as SEH with causative risk factors, such as minor trauma, chiropractic manipulation, Paget’s disease, ankylosing spondylitis, and rheumatoid arthritis. Our case never had above risk factors and was recognized as idiopathic SEH.

Chronic SEH is defined as spinal compression for months or years with mild symptom and all of the reported cases in the past occurred in the lumbar spine. In the past reports, 17 cases of chronic and spontaneous SEH were reported in English literature. As shown in Table 1, 14 cases of idiopathic and chronic SEHs were reported. Three cases were diagnosed on myelogram, four were on CT, and seven cases were on MRI. Though the hematoma was shown as various images on T1 and T2 images, chronic hematoma was in general low intensity on both T1 and T2 images. In our case, SEH which was spread from L2 to L5 body level was like crescent and showed low intensity on both of T1 and T2 images. Therefore, it was in chronic phase based on MRI even though the symptom occurred suddenly. The MR images in addition to the shape of the SEH indicated that the hematoma was enlarged little by little since the first onset of lumbar pain was 2 months before.

The locations and shapes of SEHs diagnosed with myelogram and CT were not correctly expressed. The SEHs were located at the lateral recess in five, at lateral behind the dural sac in five, at midline behind the dural sac in three, and not described in one. The SEHs were located at only one level in 11 and at two levels in three. In our case, SEH was spread from L2 to L5 body level. All of SEH cases presented with pain in the lumbar, buttock, and lower leg, and only two cases showed sudden severe pain and the other showed gradual progressive pain. Three cases complicated with slight motor weakness. The evacuation of SEHs was performed because of the severe pain in all the cases and none of the procedure was emergent. In one case, the operation was performed 3 days after admission because the pain was severe. In our case, it was 7 days after because of the same reason. The evacuation can be considered when enough medicine and rest therapy are not effective.

In past reports as shown in Table 1, 14 cases of idiopathic and chronic SEHs were operated, but two cases were not described in detail. About 11 cases were localized in the single level and three cases were spread at 2 levels. The SEH in our cases were spread from L2 to L5 body level. In past literatures, surgical evacuation resulted in good clinical courses and laminectomy in nine, hemilaminectomy in two and partial hemilaminectomy in one were performed. The SEHs located laterally were removed with laminectomy or hemilaminectomy and the SEHs located on midline were with laminectomy. The complete evacuation was done in five cases and partial evacuation was in one and the other eight cases were not described. Although the follow-up time was not described, the SEH removed partially vanished completely. Our case showed sudden severe lumbar pain and hypalgesia, and we choose surgery 1 week after the onset because the symptom did not improve. Because the SEH compressed dural sac the most severely at L2/3, partial hemilaminectomy at L2/3 and partial removal of SEH at the level was performed. The remaining SEH was completely disappeared 1 month after surgery. The residual chronic subdural hematoma in the cranial cavity after drainage is gradually absorbed and the hematoma does not need complete removal. For the same reason, we believe that partial removal and release of the capsule and epidural space can lead the drain to the epidural space and gradual absorption of the hematoma. Our procedure was considered as less invasive and effective to get enough results.

Recently, less invasive surgery is recommended and endoscopic surgery may one option for chronic SEH. In this case, the SEH was preoperatively diagnosed as idiopathic and chronic because MRA, CTA, and PET CT denied that vascular malformation and tumor caused the SEH. But the conclusive evidence was not preoperatively obtained and microscopic removal was chosen. As a result, partial removal of the SEH led to the release of lumbar pain and hypalgiesia, and early absorption of the hematoma 1 week after operation and complete absorption 1 month after operation.

The cause of idiopathic SEH was not clearly clarified. Some authors reported the epidural bleeding originates in the rupture of Batson’s plexus due to a rise in intra-abdominal pressure. On the other hand, other authors proposed that arterial bleeding or the rupture of epidural vascular malformation lead epidural hematoma. In this case, the cause of the epidural hematoma was not clearly
### Table 1  The summarize of all cases published in the literature of idiopathic and chronic epidural spinal hematoma in the lumbar spine

| No. | Author       | Year | Age | Sex | Level   | Location            | Shape   | Duration | Tool      | Surgery                        | Removal rate | Pathology |
|-----|--------------|------|-----|-----|--------|----------------------|---------|----------|-----------|---------------------------------|--------------|-----------|
| 1   | Harris       | 1969 | 66  | M   | L5/S1  | ?                    | Several M| Myelogram| Laminectomy L5–S1 | ?               | Yes        |
| 2   | Boyd         | 1972 | 66  | M   | L4–5   | Posterior lateral    | ?       | 7M       | Myelogram | Partial hemilaminectomy L4–5   | ?            | No        |
| 3   | Boyd         | 1972 | 75  | F   | L3/4   | Posterior lateral    | ?       | 6M       | Myelogram | Laminectomy                   | ?            | Yes       |
| 4   | Levitan      | 1983 | 58  | F   | L4     | Lateral recess       | ?       | Sudden   | CT: high | Yes but unknown              | ?            | No        |
| 5   | Levitan      | 1983 | 90  | F   | L3/4   | Posterior lateral    | ?       | 1.5M     | CT: high  | Yes but unknown              | ?            | No        |
| 6   | Nehls        | 1984 | 74  | M   | L3/4   | Lateral recess       | ?       | 2M       | CT: iso   | Laminectomy L3–4              | ?            | Yes       |
| 7   | De Almeida   | 1989 | 88  | M   | L3/4   | Posterolateral       | ?       | 4M       | CT: high  | Laminectomy L3–4              | ?            | No        |
| 8   | Nakgami      | 1992 | 58  | F   | L4     | Posterior            | Crescent | 2M       | T1, T2: high | Total laminectomy L4, partial laminectomy L3, L5 | Total        | Yes       |
| 9   | Lunardi      | 1995 | 45  | M   | L2–3   | Posterior midline    | Crescent | 2M       | T1, T2: high | Laminectomy L2–3              | Total        | Yes       |
| 10  | Riffaud      | 1999 | 70  | F   | L4/5   | Lateral recess       | Nodular | Sudden   | T1: iso, T2: low | Laminectomy L4              | ?            | Yes       |
| 11  | Vazquez-Barquero | 2000 | 75  | F   | L2–3   | Posterior            | Nodular | 7M       | T1: iso, T2: low | Laminectomy L2–3              | Total        | Yes       |
| 12  | Sarubbo      | 2009 | 65  | F   | L3/4   | Lateral recess       | Nodular | 3M       | T1: high  | Laminectomy L3–4              | Partial      | No        |
| 13  | Sarubbo      | 2009 | 85  | M   | L3     | Lateral recess       | Nodular | 2M       | T2: low   | Hemilaminectomy L3             | Total        | No        |
| 14  | Matsui       | 2014 | 78  | M   | L4     | Posterior lateral    | Nodular | 9M       | T1: high, T2: low | Hemilaminectomy L4, PLIF | Total        | Yes       |
| 15  | Our case     | 2015 | 61  | F   | L2–5   | Posterior            | Crescent | Sudden   | T1, T2: low | Partial hemilaminectomy L2–3  | Partial      | Yes       |
proved in pathological findings. But it seemed to be the rupture of venous plexus because arterial bleeding was not seen intraoperatively and the hematoma was jelly-like dark brown colored substance. The membrane was adherent to the epidural fat tissue and it may be considered that the venous bleeding from the epidural fat tissue caused the hematoma. The pathogenesis was not proved, but it seemed to be similar to chronic subdural hematoma of the intracranial cavity.

**Conclusion**

Epidural hematoma of the lumbar spine is rare condition and idiopathic and chronic epidural hematoma is even rarer. We must investigate the cause of the hematoma, such as vascular malformation and tumor. When the hematoma is diagnosed as idiopathic and chronic, partial removal of the hematoma can be one option as less invasive surgery depending on the condition of the hematoma.

**Conflicts of Interest Disclosure**

No potential conflicts of interest were disclosed.

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