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CASE REPORT

# Idiopathic hypertrophic spinal pachymeningitis: a case report and review of literature

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**Abstract**
*Purpose* To report an unusual case of idiopathic hypertrophic spinal pachymeningitis (IHSP) with a review of relevant literature and to discuss the etiology, clinical features, imaging, treatment and prognosis of IHSP.
*Methods* The case of a 44-year-old woman is reported. MEDLINE was used to search relevant literatures written in English since 2004.
*Results* The patient suffered from progressive mild thoracic backache followed by truncal and lower extremity weakness, numbness and urinary retention. The diagnosis was confirmed by magnetic resonance (MR) imaging and histopathologic examination. Although she received corticosteroid therapy and decompressive surgery, the patient suffered a rapid relapse probably because of the withdrawal of postoperative steroid therapy.
*Conclusions* IHSP is a rare disease characterized by inflammatory hypertrophy of the dura mater without identifiable cause and featured clinical progress of radiculalgia to myelopathy. It is a diagnosis of exclusion. In our view, surgical decompression with postoperative steroid therapy may be optimal. Furthermore,we speculated that increased levels of protein and cell count in cerebrospinal fluid (CSF) might be positively related to the disease progression. High inflammatory signs or CSF protein and cell levels before surgery or postoperative residual lesions are possible reasons of poor prognosis in patients with IHSP.

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**Keywords** Idiopathic hypertrophic spinal pachymeningitis · Radiculalgia · CSF · Therapy · Prognosis

## Introduction

Hypertrophic pachymeningitis is a chronic inflammatory disease. It can be divided into hypertrophic cranial pachymeningitis (HCP) and hypertrophic spinal pachymeningitis (HSP) depending on the location of the lesion [1]. The majority cases of HSP are of no identifiable cause and called IHSP. Since 2004, through MEDLINE, we found only 13 cases of IHSP confirmed by biopsy reported in English-speaking literature. Here, we reported a case of IHSP, analyzed the factors that probably affect prognosis and tried to discuss optimal therapy. Although Zhao et al. [2] reported the features of idiopathic hypertrophic pachymeningitis in China in 2014, they only focused on HCP. To our knowledge, this is the first report about IHSP in China. In our view, the low incidence rate of this disease in China is due to missed diagnosis.

## Case report

A 44-year-old woman presented with acute symptoms of truncal and lower extremities weakness, numbness from the mid-chest down to the feet, and sphincter dysfunction in March 2014. Before that, she had been complaining of thoracic backache radiating to left truck for 1 month. The radiculalgia was aggravated at rest and partially relieved after movement. The weakness and numbness of lower extremities started and rapidly progressed to disabled 1 day later. There was no history of trauma, tuberculosis or any other illnesses.

Cranial nerve examination showed no abnormalities. Motor and sensory functions in the upper extremities were intact, but muscle strength in the lower extremities was approximately grade 4/5. In addition, superficial sensation was decreased and deep sensation was lost below the mid thoracic level. Laboratory examinations revealed that the level of inflammatory biomarkers was within normal range and immune indices were negative. A tuberculin skin test and a MycoDot TM were also negative, while the T-SPOT-TB test was positive. A MR imaging scan of the spine was performed (Fig. 1). The sagittal images of MR imaging showed predominantly ventral thickening of the dura mater with compression of the spinal cord from the T-2 to T-6 levels. Images with enhancement showed hypointense on T2-weighted and isointense on T1-weighted following intravenous gadolinium injection. Axial images after the administration of gadolinium disclosed a crescent-enhancing lesion which was mainly located in the anterior aspect of the spinal canal. Lumber punctures had been performed five times and the biochemical examination of CSF was variable accompanying with fluctuations of clinical features. Among them, the maximum leukocytosis count with lymphocyte predominance was up to $150 \times 10^6$/L and protein content was 6106.70 mg/L. Special stains were negative for fungi, bacteria, mycobacteria and CSF tuberculosis antibody test was noncontributory (Table 1).

The patient was treated with methylprednisolone pulse therapy (500 mg daily) followed by maintenance treatment with oral prednisone (60 mg daily). Two weeks later, there was no improvement in her condition and MR imaging (Fig. 2). Then antituberculous treatment started as tuberculosis could not be eliminated and prednisone therapy (60 mg daily) was continuing at the same time. On the tenth day after antituberculous treatment, the patient suffered sudden and severe, continuous back pain. Besides, her muscle strength in the lower extremities dropped to grade 0/5 and the level of decreased sensation rose to the nipple line within the following 36 h. She was completely paraplegia. The lesion revealed by MR imaging extended upwardly and backwardly, and it became a ring shape at the axial images (Fig. 3).

Since neurological function deteriorated rapidly, a T1–T7 laminectomy was performed for decompression immediately. There was a large amount of lipoid tissue encompassing and compressing the dural sac. The dura mater of the lesion area was thicker than normal. For the sake of safety, only posterior dura was excised, though hypertrophic dura mater compressed the spinal cord anteriorly and posteriorly. Histologic examination of the dura mater revealed adipose tissue with extensive fibrosis and chronic inflammation (Fig. 4). Immunohistochemistry analysis showed inflammatory cells were mainly T cells and B cells, no atypical cells, granulomas or vasculitis were identified. With the help of neurological rehabilitation, the patient's muscle strength in the lower extremities recovered slowly after the surgery. The steroid therapy was not continuously applied after the surgery. However, 1 month later, the patient relapsed both clinically and radiologically (Fig. 5). She was completely paraplegia again. Her muscle power dropped to grade 0/5 and sensation decreased below the nipple line.

## Discussion

HSP is a chronic progressive disease characterized by diffuse inflammatory fibrosis and thickening of the dura mater [3]. HSP was first reported by Charcot and Joffroy in

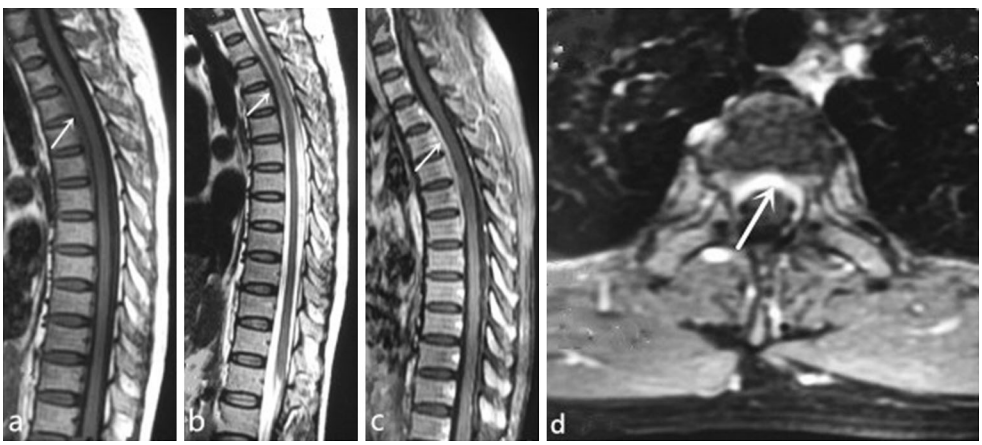


**Fig. 1** Initial MR imaging. A dural-based mass in the anterior aspect of the spinal canal extending from T2 to T6 level which is uniform signal intensity in sagittal T1-weighted image (**a**), low signal intensity in sagittal T2-weighted image (**b**) and enhanced in sagittal postcontrast T1-weighted image (**c**). Axial postcontrast T1-weighted image (**d**) reveals the enhanced lesion is crescent shaped and mainly located in the anterior aspect of the spinal canal

**Table 1** Details of CSF analyses

| Date | Pressure ($mmH_2O$) | White blood cells (WBC)/$mm^3$ (% lymphocytes) | Protein (mg/L) | Glucose (mmol/L) | Other examinations of CSF |
|---|---|---|---|---|---|
| March 26, 2014 | 100 | 150 (90) | 6106.70 | 4.31 | [b] |
| March 28, 2014 | 135 | 143 (97) | 165.7 | 2.99 | Tuberculosis antibody test was negative |
| April 2, 2014 | 110 | 90 (99) | 242.0 | 4.44 | [b] |
| April 9, 2014 | 92 | 4 ([a]) | 1126.0 | 4 | Acid-fast stain was negative |
| April 15, 2014 | 140 | 150 (75) | 3638.1 | 3.64 | ADA2.6 μ/L acid-fast stain was negative |

Special stains were negative for fungi, bacteria, mycobacteria

[a] Not mentioned

[b] Not performed

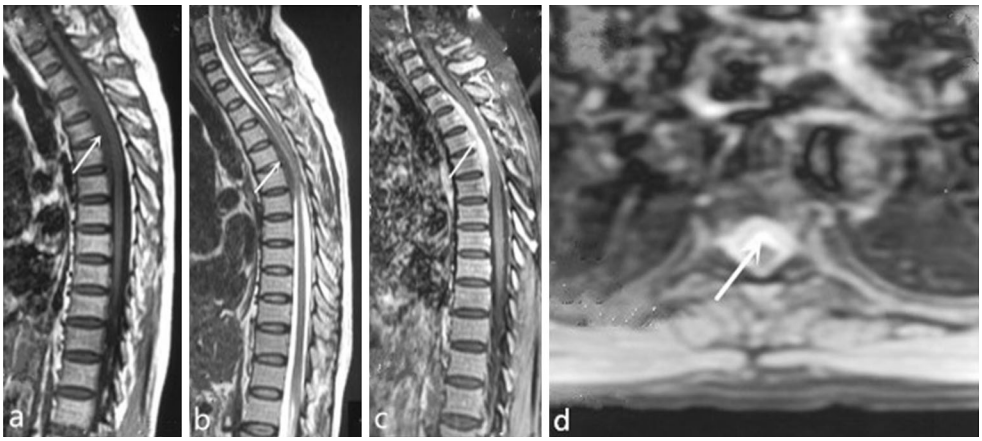


**Fig. 2** Follow-up MR imaging after half a month of steroid therapy. The lesion was almost unchanged

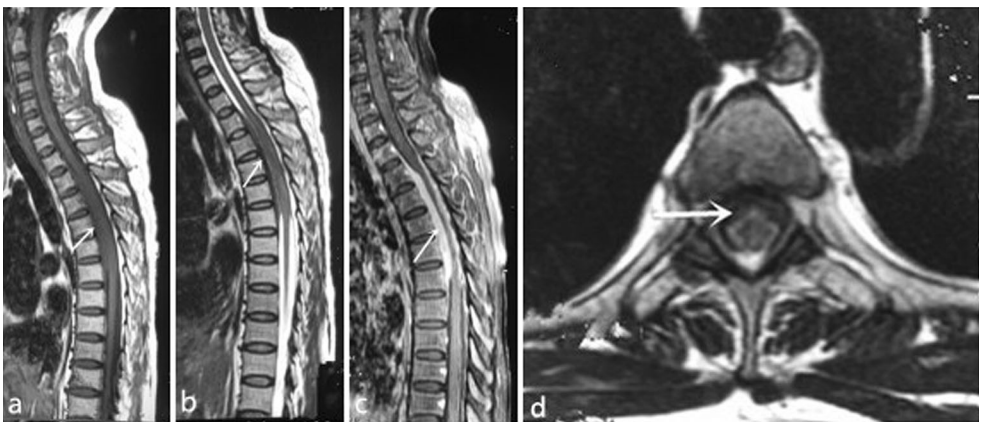


**Fig. 3** MR imaging when the patient's condition was deteriorated. Sagittal T1-weighted image (**a**), sagittal T2-weighted image (**b**) and enhanced in sagittal postcontrast T1-weighted image (**c**) show the lesion have obviously increased in size. **d** The axial postcontrast T1-weighted image

1869. Its cranial form of hypertrophic pachymeningitis was later described, which is more common than the spinal form. In 2003, Esparcia et al. [1] reviewed all 58 cases of idiopathic hypertrophic pachymeningitis reported during the past 35 years, among which, 43 cases were HCP and 11 cases were HSP.

Possible causes of HSP are various, including infectious diseases such as syphilis [4], tuberculosis infection [5],

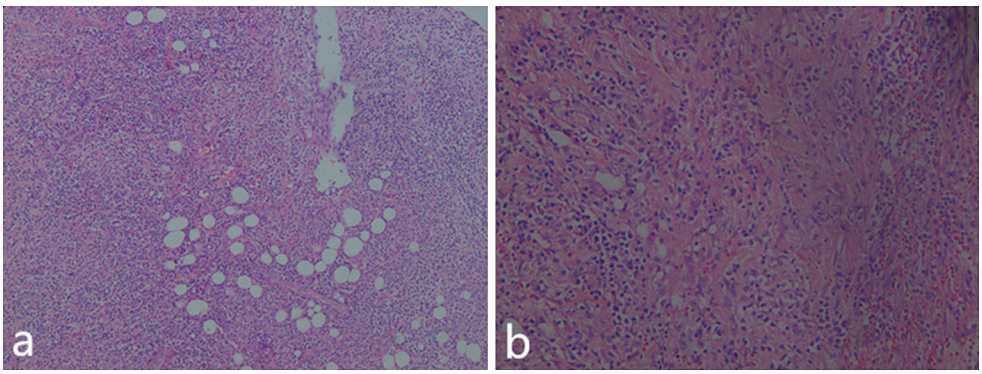


**Fig. 4** Photomicrograph showing adipose tissue with extensive fibrosis. The fibers are infiltrated by a large amount of chronic inflammatory cells. A typical cell, granulomas, vasculitis were not identified (hematoxylin and eosin stain, original magnification ×10 and ×40)

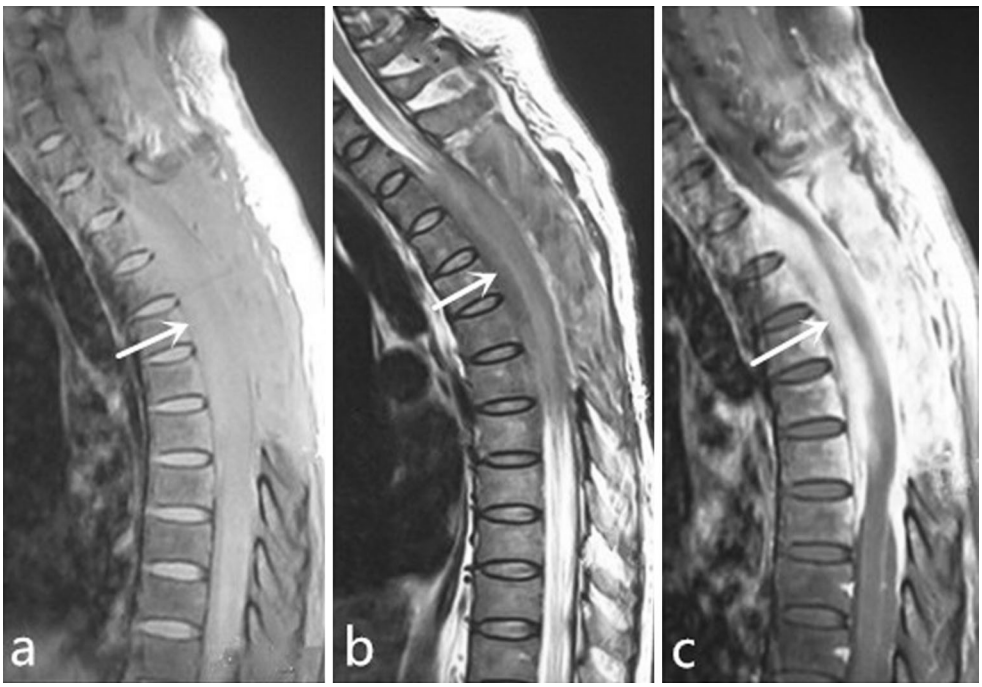


**Fig. 5** Follow-up MR imaging at clinical relapse (1 month). Meningeal lesions have increased again

fungal infection and autoimmune disorders [6–10] like rheumatoid arthritis, Wegener's granulomatosis and p-ANCA positive diseases. Other factors subsume neoplastic diseases, intrathecal drug administration [11], trauma and so on. However, most cases remained idiopathic. Though the pathogenesis of IHSP is not well understood, increasing evidence have suggested that abnormal autoimmune may play a role [12]. In our case, increased CSF lymphocytes were identified and the biopsy showed increased lymphocytic infiltration, which strongly supported the argument above. Moreover, we found the increase of lymphocyte and protein is consistent with disease progression.

Only thirteen IHSP cases confirmed by biopsy were reported in English literature since 2003. All these cases were described in Table 2 including ours. These cases showed a female preponderance (71 %). Although all ages were affected, people in the age range of 40–60 are at an obviously higher risk of IHSP (57 %). IHSP affects most commonly the cervical and thoracic spine (86 %), involvement of the entire spine was relatively unusual (7 %). Ventral dura of the spinal canal was more susceptible than dorsal side [13]. The lesion in our case was mainly located in the front of thoracic spinal canal. At present, paralysis was the most common symptom, occurring in ten of fourteen patients (71 %). Other common symptoms

**Table 2** Published English cases of IHSP which are confirmed by biopsy

| Case no. | References | Age (years)/gender | Clinical presentation | Laboratory examination | CSF | Levels | MR imaging signal intensity | Location in spinal canal | Treatment | Improvement | Recurrence |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Sridhar et al. [20] | 48/F | Weakness<br>Heaviness<br>Back pain | → | [a] | C7–T11 | Hypointense (T1)<br>Hypointense (T2) | Circumferential | Steroid antituberculous | Y | N |
| 2 | Claus et al. [18] | 31/F | Back pain<br>Hypoesthesia | → | Protein↑<br>Cells↑ | T6–T12 | Ispointense (T1) | Dorsal and ventral | Steroid | Y | Y |
| 3 | Pai et al. [17] | 68/M | Weakness<br>Urinary retention<br>Decreased sensation<br>Poor rectal tone | [a] | [a] | C3–T1 | Ispointense (T1)<br>Hypointense (T2) | Ventral | | Y | N |
| 4 | Pai et al. [14] | 47/F | Weakness<br>Numbness<br>Bowel and bladder incontinence<br>Increased reflexes | [a] | [a] | T1–T6 | Hypointense (T1)<br>Hypointence (T2) | Ventral | Operation | Y | N |
| 5 | Aburahma et al. [15] | 3.5/M | Vomiting<br>Irritability<br>Decline in activity<br>Weight loss | → | Protein↑<br>Cells↑ | Entire spinal meninges | [a] | [a] | Steroid<br>Cyclophosphamide<br>Cal cytarabine | N | Y |
| 6 | Lowdon et al. [21] | 42/F | Numbness | ESR↑<br>Immunological examination→ | → | T2–T5 | Hypointense (T2) | Ventral | [a] | [a] | [a] |
| 7 | Ranasinghe et al. [17] | 65/M | Decreased position sense<br>Gait ataxia | [a] | [a] | T4–T9 | Ispointense (T1)<br>Hypointense (T2) | Circumferential | | N | N |
| 8 | Ranasinghe et al. [17] | 77/F | Ambulatory dysfunction<br>Decreased strength<br>Decreased sensation | [a] | [a] | C4–T11 | Ispointense (T1)<br>Hypointense (T2) | Circumferential | | Y | N |

**Table 2** continued

| Case no. | References | Age (years)/gender | Clinical presentation | Laboratory examination | CSF | Levels | MR imaging signal intensity | Location in spinal canal | Treatment | Improvement | Recurrence |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 9 | Ranasinghe et al. [17] | 43/F | Paresthesias | → | [a] | T1–T6 | Hypointense (T1)<br>Hypointense (T2) | Circumferential | Operation steroid | Y | N |
| 10 | Tsutsui et al. [22] | 28/M | Spastic gait<br>Numbness<br>Back pain<br>Urinary retention | ESR↑<br>CRP↑ | Protein↑<br>Cells↑ | T5–L2 | [a] | Circumferential or crescent shaped | Operation | Y | Y |
| 11 | Kim et al. [23] | 45/F | Fever<br>Quadriplegia | WBC↑<br>ESR↑<br>CRP↑ | [a] | C1–C4 | Hypointense (T1)<br>Hypointense (T2) | Ventral | Operation steroid | N | Y |
| 12 | Kim et al. [23] | 55/F | Backache<br>Paraparesis<br>Numbness<br>Voiding difficulty | ESR↑<br>CRP↑ | [a] | C6–T8 | Hypointense (T1)<br>Hypointense (T2) | Ventral | Operation steroid | Y | Y |
| 13 | Jee et al. [24] | 58/F | Weakness of lower extremities<br>Clumisiness of hands | ESR↑ | [a] | C5–C7 | Hypointense (T1)<br>Hypointense(T2) | Dorsal | Operation steroid (temporarily) | Y | N |
| 14 | Present case, 2014 | 44/F | Weakness<br>Numbness<br>Urinary retention | → | Table 1 | T2–T6 | Ispointense (T1)<br>Hypointense (T2) | Dorsal and ventral (mainly) | Operation<br>Steroid empirical antituberculous | Y | Y |

All of them showed typical histopathology findings which were thicken and fibrosis of the dura, with chronic inflammatory cell infiltration composed mainly of lymphocytes and plasma cells. Erythrocyte sedimentation rate and serum C-reactive protein are abbreviated to ESR and CRP, respectively

↑, increase; →, normal; *F* female, *M* male, *C* cervical, *T* thoracic, *L* lumbar, *Y* yes, *N* no

[a] Not performed, or not mentioned

included numbness (64 %), which was also the only symptom in one case, and bladder and rectal dysfunction (43 %). There were 4 cases (29 %) with back pain as the initial symptom including this case. As far as we know, direct compression of vascular compromise or neural structure might attribute to these symptoms of nerve root or spinal cord compression.

In ten cases where patients underwent peripheral blood study, non-specific inflammation was seen in half of the cases (50 %). The CSF analyses were performed in five cases, most of the results (80 %) showed different levels of abnormality. In our case, CSF white blood cells increased and mononuclears prevailed. This might be considered to be local aseptic inflammatory response which may be related to autoimmune system. Increased CSF protein levels might result from the blocking of CSF circulation. High protein in CSF, in turn, undoubtedly, aggravated the blocking of CSF circulation. Kupersmith et al. [13] noted that patients with abnormal CSF protein tended to have more diffused dural involvement on MR imaging than CSF cellular response. In our case, the levels of protein and cell in CSF were variable accompanying with fluctuations of clinical features. Based on the simultaneous changes of clinical symptoms and levels of protein and cell in CSF, we further speculated that increased levels of protein and cell count in CSF may be positively related to the disease progression.

MR imaging is a preferred imaging examination. After reviewing reported cases of IHSP that included MR imaging findings, Pai et al. [14] proposed that a long extramedullary mass of low T2 signal intensity with predominantly peripheral enhancement represented a specific MR imaging finding which was highly suggestive of IHSP. Biopsy is still the gold criteria for diagnosis. All of 13 cases in Table 2 showed characteristic histopathology findings which were thickening and fibrosis of the dura, with chronic inflammatory cell infiltration composed mainly of lymphocytes and plasma cells. In particular, adipose tissue seen in our case might be a sign of degeneration.

Up to now, IHSP is a diagnosis of exclusion. When all the known etiologies such as infectious, tumor, autoimmune disorders are ruled out and typical pathological findings are displayed, this diagnosis can be established. Accordingly, once imaging test suggests HSP, physicians should make a thorough etiological investigation to find the evidence of some underlying conditions.

The treatment of IHSP is contested. Given that IHSP is a chronic inflammatory process, steroid treatment is often used. However, there is no clear consensus on the treatment doses and duration of steroid. In addition, radiotherapy, immunosuppressive therapy such as azathioprine and cyclophosphamide have also been tried in some cases [15]. As they believe the underlying cause of some of the idiopathic cases was occult infection of mycobacterium tuberculosis, Parney et al. [16] recommended empirical antituberculous therapy on patients of apparent idiopathic pachymeningitis. Unfortunately, no convincing evidence showed this treatment method can slow the progression of the disease. In Table 2, most of the ten cases received the decompressive surgery showed dramatic improvement in neurological deficit. Usually, the application of surgical decompression with subsequent maintenance steroid therapy demonstrates the positive effects and may be the optimal treatment [17]. However, in our patient, steroid therapy was not continued postoperatively which was probably one reason why she relapsed so quickly.

Relapse is indeed the main problem of long-term IHSP [18, 19]. Many authors reported that patients who recovered well after operation still had a high recurrence rate during follow-up. Some researchers believed that patients with active dural inflammatory signs, like fever, increased sedimentation rate, leukocytosis, or increased CRP before surgery had a poorer prognosis and a higher recurrence rate than patients without inflammatory signs [3, 12]. Beyond this, some other conditions like residual pachymeningitis or arachnoiditis can also influence prognosis [17]. In our case, only posterior dura was excised during the operation and residual pachymeningitis or arachnoiditis might be another reason of relapse. Steroid treatment significantly decreased the high levels of CSF protein and cell in this case. However, as IHSP progressed with recurrent increments of CSF protein and cell levels, clinical manifestations and MR imaging showed a rapid deterioration. We suspected that increased CSF protein and cells may contribute to disease progression. Moreover, there could be a positive correlation between CSF protein, cell levels and patient's condition, as well as prognosis.

In conclusion, IHSP is a rare disease which can cause disastrous neurological damage. Physicians should pay attention to the typical MR imaging of this disease and actively exclude any other causes for HSP. Surgical decompression with postoperative steroid therapy may be the best treatment option. Inflammatory signs or increased CSF protein and cell levels before surgery or postoperative residual lesions may indicate poor prognosis in patients with IHSP.

**Conflict of interest** The authors have no actual or potential conflicts of interest to report.

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