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CASE REPORT

Calcific discitis with giant thoracic disc herniations in adults

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Abstract
Purpose Calcific discitis is a self-limiting process most commonly seen in the cervical spine of children. Rare literature exists regarding the natural history and management of this condition in adults, especially when it presents as a giant thoracic disc herniation into the spinal canal. Giant herniations in the thoracic spine are typically surgically removed to reduce the chance of permanent neurologic deficit from spinal cord compression. However, when associated with calcific discitis, they may undergo spontaneous regression with the need for surgery obviated.
Methods Medical records and radiographic studies of two adult patients with calcific discitis and myelopathy due to spinal cord compression by giant thoracic disc herniations were retrospectively reviewed. Search of the literature on calcific discitis in adults and spontaneous regression of calcified thoracic disc herniations was separately performed.
Results Both patients were young male adults presenting with back pain and early signs of myelopathy. With restriction of activities and oral NSAIDs, their symptoms were relieved within 3 months. Four adult cases of calcific discitis (characteristic central calcification confined within the *nucleus pulposus*) and three instances of spontaneous regression of small- to medium-sized thoracic calcified disc herniations were identified from the literature.
Conclusions The demonstration of spontaneous resorption of giant calcified thoracic disc herniations in two adult patients with calcific discitis supplements the existing literature and provides the first evidence that giant calcified thoracic disc herniations may still undergo spontaneous remission and a "wait and watch" strategy may be justified at least in the initial management of these patients, even with the presence of mild myelopathy.

Keywords Thoracic disc herniation · Thoracic myelopathy · Calcific discitis · Giant herniation · Spontaneous regression

Introduction

Calcific discitis in children is a well-described diagnosis with hundreds of cases reported in the literature and its etiology, clinical characteristics, and outcomes have been excellent reviewed [1]. It most commonly presents as axial pain in the lower cervical spine of children between the ages of 6 and 10 and typically only the *nucleus pulposus* was calcified, while asymptomatic involvement of the thoracic and lumbar spine has also been reported [2]. A single level calcification was exhibited in 70 % of patients, but multiple discs may be affected [3]. The prognosis is generally excellent, with symptoms resolving within several months following conservative treatment of oral anti-inflammatory medications and activity restriction, even in rare cases with giant disc herniations [4].

Only four instances of calcific discitis in the adult population have been reported [5–7]. In all of them, the calcification was invariably confined within the *nucleus*

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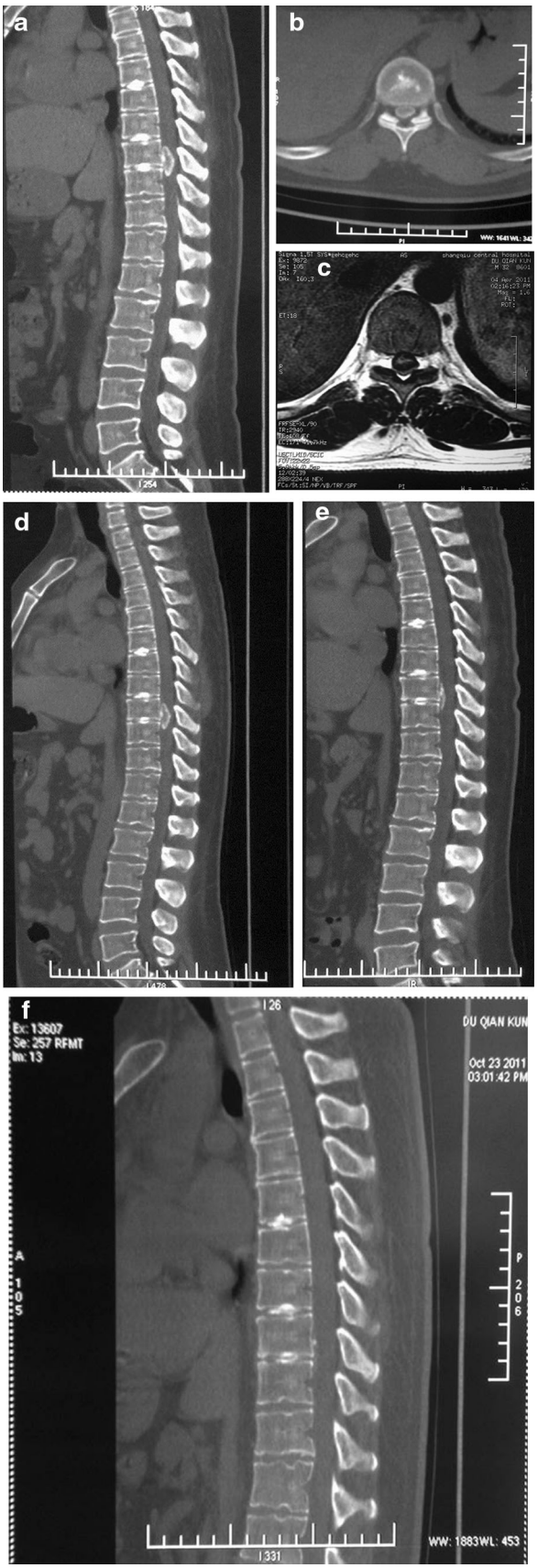

Fig. 1 *Patient 1* At first presentation, calcification of the *nucleus pulposus* at the intervertebral levels of T6/7, T8/9, and T9/10, and a giant calcified HTD at the T9/10 level was demonstrated in the sagittal CT reconstruction (**a**). Axial views of CT and MRI at the initial presentation (**b**, **c**). Partial resorption of the HTD observed 1 month (**d**) and 3 months later (**e**). Complete resorption of the HTD after 6 months of conservative treatment of oral NSAIDs and restriction of activities (**f**)

pulposus. Rare literature exists regarding the natural history and management of this condition in adults, especially when it presents with a giant disc herniation into the spinal canal.

We herein report the first two cases of adult calcific discitis with giant herniations of thoracic discs (HTDs). A giant herniation was defined as occupying more than 40 % of the spinal canal based on pre-operative magnetic resonance imaging (MRI) according to Hott et al. [8]. The non-operative management and outcomes of these two cases may be of particular interest to thoracic spine surgeons as they highlighted the self-limiting nature of calcific discitis in adult patients and the potential for spontaneous regression of symptoms even in those with early signs of myelopathy secondary to ventral compression by HTDs. Both patients were followed for more than 3 years and no long-term sequelae or recurrences were noted.

Case report

Case 1 A previously healthy 31-year-old man presented with symptoms of mid-back pain of abrupt onset (VAS 7), numbness in the lower extremities, and difficulty in walking noted for 2 weeks. There was no recent history of trauma, fever, or weight loss, and the patient otherwise had no significant medical or surgical comorbidities. On physical examination, the patient had tenderness to palpation over the spinous processes of the mid-thoracic spine. The neurologic exam revealed bilateral quadriceps hyperreflexia but no motor or sensory deficits. No pathological signs were elicited. The patient had no bladder dysfunction. Blood panel and erythrocyte sedimentation rate results were within normal limits. CT scan was performed to rule out spinal cord compression and revealed multi-level calcification of the central portion of the intervertebral discs at the T6/7, T8/9, and T9/10 levels, and a giant, calcified HTD at the T9/10 level with soft-tissue density centrally and a calcific rim (Fig. 1a, b). The ovoid mass was 1 cm in size and slightly hypo-intense relative to the vertebral body marrow in the periphery and hyper-intense centrally on MRI (Fig. 1c). The patient was started on a course of oral NSAIDs [5] and activity restriction (i.e., no active sports, daily activities of living as tolerated but bed rest preferred).

At the 1-month and 3-month follow-up visits, his symptoms had improved and partial resorption of the HTD was observed (Fig. 1d, e). Seven months after the beginning of treatment, the patient became symptom-free and exhibited complete radiographic resorption of the HTD but the intradiskal calcification was not absorbed (Fig. 1f).

Case 2 A 55-year-old man presented with a 3-week history of progressive back pain (VAS 8) and numbness and weakness in the lower extremities. His medical history was unremarkable. The symptoms began 3 weeks ago after waking up from a nap at noon. At the time of presentation the patient was able to walk but not run. There was no bladder dysfunction noted in the history. On physical examination, he was tender to palpation over the spinous processes of the mid-thoracic spine and had positive Babinski's sign bilaterally. Deep tendon hyper-reflexia was elicited on both sides. There was hyposensitivity to pain in both legs but muscle strength was all V on the Oxford scale. Sagittal reconstruction of the CT scan revealed T8/9 disc central calcification and a giant HTD (Fig. 2a). Axial view of the same diskal level demonstrated ventral calcification of the giant HTD and its dorsal one-half was hyperintense relative to the vertebral body marrow on MRI (Fig. 2b). He was treated conservatively with oral NSAIDs and activity restriction in a similar fashion to the first patient. Three months after the initial presentation, he complained of no pain and revealed improved upper motor neuron signs on examination of the legs. Axial and sagittal reconstruction of CT scan revealed resorption of the soft-tissue density part of the giant HTD (Fig. 2c, d). At the 6-month follow-up, his symptoms had disappeared and the patient was able to run again. A CT scan confirmed complete resorption of the giant HTD including the ventral calcification, with only residual asymptomatic calcification of the *nucleus pulposus* (Fig. 2e).

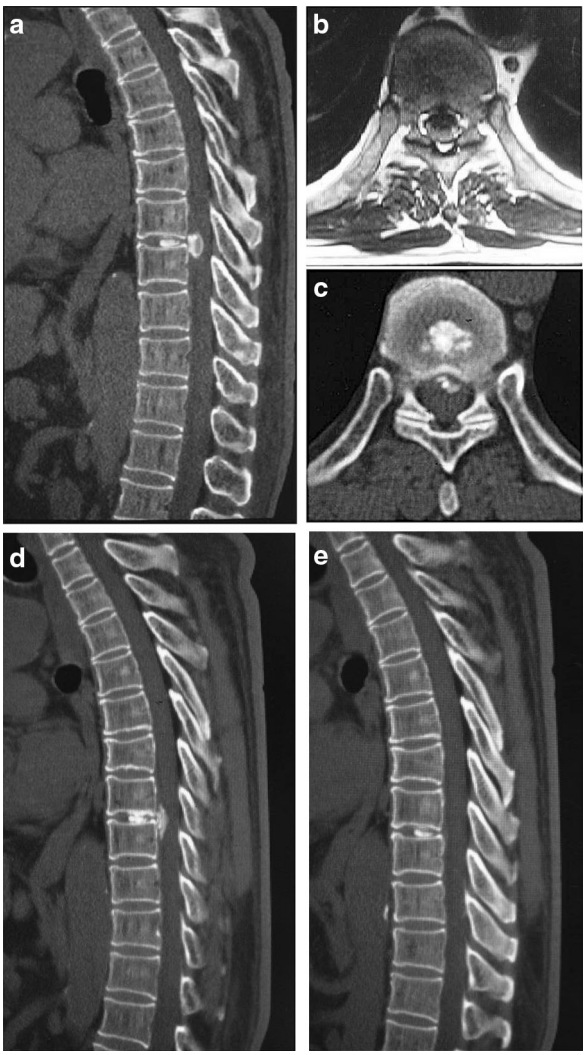

Fig. 2 *Patient 2* At first presentation, sagittal reconstruction CT demonstrated calcification of the *nucleus pulposus* along with a giant calcified HTD at the intervertebral levels of T8/9 (**a**). Axial views of MRI (**b**) of the lesion at the initial presentation. Partial resorption of the HTD observed 3 months later on axial (**c**) and sagittal CT reconstruction (**d**). Complete resorption of the HTD after 6 months of conservative treatment of oral NSAIDs and restriction of activities (**e**)

Discussion

Both of our patients initially had an abrupt onset of severe pain in the mid-thoracic spine and presented to our hospital during the acute phase (i.e., within 3 weeks) with mild myelopathy. Diagnostic imaging studies confirmed intradiskal calcification and a giant HTD. A course of conservative management consisting of oral NSAIDs and activity restriction proved effective for complete symptomatic relief 6 months after the beginning of treatment.

Of note, both patients remained to show calcification within the *nucleus pulposus* at the last follow-up, a phenomenon that is characteristic of calcific discitis, which needs to be distinguished from the more common condition of intervertebral disc calcification (IDC). Causes of IDC include alkaptonuria, hemochromatosis, amyloidosis, hyperparathyroidism, and in the vast majority of cases, degenerative changes [11]. The previously reported prevalence of IDC was 5 % [12, 13] and the primary site involved was the *annulus fibrosus* in the lower thoracic spine of asymptomatic elderly patients [11]. In contrast, calcific discitis is a self-limiting disorder and generally considered as only occurring in children. A total of four instances of this condition in adults were identified from the literature (patient 1–4 in Table 1). Their average age was 38.3 years, younger than a typical degenerative spine patient. Two presented to the ER with acute severe pain and the other two had a history of moderate pain for several weeks. No one had any signs of myelopathy or HTDs on radiologic studies. All recovered within 6 months of

Table 1 Literature review identified four cases of adult calcific discitis (patient 1–4) and three cases of resorption of small/medium-sized HTDs (patient 5–7)

Author	Gender	Age	Duration of symptoms	Pain	Neurologic exams	Disc level	Treatment	Time to complete recovery
Pt 1 Bazzi	F	32	Emergency	Severe	Normal	T7/8	Jewett brace, anti-inflammatory medications, activity restriction	1 month
Pt 2 Bazzi	F	59	4 months	Severe	Normal	T10/11	Anti-inflammatory medications, PT, transcutaneous electrical nerve stimulation	Recovered but details not available
Pt 3 Nogueira-Barbosa	M	40	15 days	Moderate	Normal	T9/10	Analgesics	20 days
Pt 4 Azizaddinni	F	22	Emergency	Severe	Normal	C2/3	NSAIDs and benzodiazepines	6 months
Pt 5 Piccirilli	F	36	5 months	Moderate	Normal	T7/8	Analgesics + Ketoprofene	3 months
Pt 6 Eap	M	48	6 months	Severe	Normal	T8/9	Analgesics	6 months
Pt 7 Eap	F	45	Incidental finding	No	Normal	T12/L1	None	12 months

conservative treatment consisting of different combinations of bracing, oral anti-inflammatory medications, bed rest alone, physical therapy, transcutaneous electrical nerve stimulation, analgesics, or benzodiazepines.

Surgical decompression is often indicated for HTDs associated with severe neurologic compromise that is recalcitrant to conservative therapy for 6–8 weeks. Barbanera et al. reported a series of seven patients with giant calcified HTDs. None showed signs of regression with conservative management and thus were all surgically treated [9]. More recently, Quraishi et al. published a retrospective cohort of 13 patients with giant calcified HTDs who all underwent surgery [10]. However, thoracic decompression is associated with a high incidence rate of surgery-related adverse events [9, 10, 14]. In particular, some anatomic features may predispose the thoracic spinal cord to injury during surgery. The high cord occupancy ratio, tenuous blood supply, draping of the cord over the posterior longitudinal ligament, and limited posterior displacement due to tethering by the dentate ligament are all contributing to this disposition. Furthermore, giant calcified HTDs display unique surgical challenges due to their large volume and high likelihood of adherence to the dura mater and intra-dural growth [8, 15]. This current study suggests that the need for surgery might in some cases, i.e. when there is concurrent calcification of the *nucleus pulposus*, be obviated. A similar course of recovery was described by Wu et al. in an 8-year-old boy with calcific discitis and a giant calcified HTD [4] but has never been reported in adults. The only three adult cases of spontaneous regression of HTDs found in the literature were all of small or medium-size [16, 17] (patient 5–7 in Table 1) and the patients had no signs of neurologic abnormality. Our study provided the first evidence that complete, spontaneous resorption was possible even with giant calcified HTDs in adult patients.

Martinez-Quinones et al. summarized the current hypotheses for explaining the phenomenon of spontaneous regression of calcified HTDs as follows: re-accommodation of the intervertebral herniation, absorption of the herniated fragment following dehydration, increased intra-spinal hydraulic pressure on the HTD, and interaction of various inflammatory factors [18]; but there was no consensus from the literature. Due to a lack of histological confirmation of the exact nature of the herniated fragments, it was not possible to draw conclusions regarding the mechanism for their spontaneous regression from this current study. Examination of surgical specimens in the future in cases with more rapid progress of neurologic deterioration and thus indicated for surgery [8, 19] may help reveal the nature of the underlying pathophysiological process. Report on the use of oral NSAIDs in the management of this condition in ours and several other reports [5, 16, 20] seemed to support the hypothesis that an inflammatory reaction was involved.

In conclusion, myelopathy caused by giant HTDs in the setting of calcification of the *nucleus pulposus* may be self-limiting in adult patients and the mainstay of treatment should be oral NSAIDs and restriction of activities as detailed in the case description. A high degree of vigilance should be maintained in the future to distinguish patients with giant calcified HTDs in association with calcific discitis from those with degenerative diseases. It is also worth noting that osteochondroma is a differential

diagnosis consideration that should be ruled out by their characteristic peripheral ossification and the low-density central marrow space on CT scans [21].

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Compliances with ethical standards

Conflict of interest The authors had no conflict of interest regarding the content of this article.

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