



Clinical and radiographic features of adult calcified thoracic disc herniation: a retrospective analysis of 31 cases

Lei Yuan^{1,2,3} · Zhongqiang Chen^{1,2,3} · Zhongjun Liu^{1,2,3} · Weishi Li^{1,2,3} · Chuiguo Sun^{1,2,3} · Xiaoguang Liu^{1,2,3}

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Abstract

Purpose To analyze the clinical and radiographic characteristics of calcified thoracic disc herniation (CTDH) and explore the possible pathogeneses.

Methods This is a retrospective clinical review of prospectively collected imaging data based at a single institute from 2004 to 2021. Clinical and radiographic parameters of CTDH patients were retrospectively collected and analyzed.

Results All 31 patients included presented thoracic myelopathy with a 17.05-month preoperative disease duration. Three (9.7%) patients had a history of trauma, and the rest had insidious onset. The average spinal canal ventral-occupying ratio was $74.90 \pm 15.16\%$. The most prominent radiographic feature was the calcification of the nucleus pulposus in the intervertebral disc and the calcified lesion contiguous with the disc space protruding into the spinal canal. Three main imaging forms of CTDH were found: calcium-ringed lesion (5), heterogeneous calcification lesion (19), and homogeneous calcification lesion (7). The radiographic manifestations, intraoperative findings, and postoperative pathologies of the three subtypes were different. The calcium-ringed lesion type was younger and had a shorter preoperative duration and significantly lower mJOA score. A special case was observed conservatively for five years, which suggested that a heterogeneous lesion could progress to a homogeneous lesion.

Conclusions Adult CTDH is a special thoracic disc disease with insidious onset, a long course, and a high spinal canal-occupying ratio. Calcium deposits in the spinal canal originate from the nucleus pulposus. The intraoperative findings and postoperative pathology of subtypes are different, which might indicate different pathological mechanisms.

Keywords Thoracic disc disease · Calcified thoracic disc herniation · Clinical characteristics · Radiographic features · Intraoperative findings · Pathophysiology

Introduction

Pediatric calcific discitis is well-described, most commonly at the lower cervical level, with a self-limiting process, and most symptomatic patients are recommended conservative

treatment, even in rare cases with giant disc herniations and mild neurological deficits [1, 2]. However, adult calcified thoracic disc herniation thoracic (CTDH) differs from pediatric calcific discitis with disc herniation in several respects, including the natural history, clinical presentation, treatment, and prognosis.

As a subtype of thoracic disc disease (TDD), calcified thoracic disc herniation thoracic (CTDH) only accounts for 7.3% of thoracic disk disease with myelopathy underwent surgery [3]. CTDH is characterized by calcification of the nucleus pulposus in the intervertebral space and calcified lesion contiguous with the disc space protruding into the vertebral canal and compressing the spinal cord. The CTDH needs to be distinguished from the more common condition of intervertebral disc calcification (IDC), which occurs in 5–6% of the general adult population, and the calcification was confined to the intervertebral space

✉ Zhongqiang Chen
zqchenby@163.com

¹ Department of Orthopedics, Peking University Third Hospital, 49 North Garden Street, Hai Dian, Beijing 100191, China

² Beijing Key Laboratory of Spinal Disease Research, Peking University Third Hospital, 49 North Garden Road, Beijing 100191, China

³ Engineering Research Center of Bone and Joint Precision Medicine, Ministry of Education, Peking University Third Hospital, 49 North Garden Road, Beijing 100191, China

without herniation [4]. Surgical decompression is recommended as early as possible for symptomatic CTDH patients with intractable pain, persistent neurological deficits, or progressive myelopathy after plenty of conservative treatment, including activity restriction and oral NSAIDs [5]. Adult CTDH greatly increases surgical difficulty because of the large volume, calcified and transdural nature, and the inherent vulnerability of the thoracic cord [6]. High rates of postoperative complications have been reported, including serious complications such as neurological deterioration [5, 7].

Some case reports have reported that spontaneous resorption of giant calcified thoracic disc herniations could happen in adult patients presenting back pain and even early signs of myelopathy [8–14]. In these spontaneous-regression cases, CT scans show that the calcified lesion protruding into the epidural space has a centrally soft tissue density [8, 13, 14]. However, adult CTDH was a rare, poorly understood condition. There is rare literature regarding the natural history, clinical presentation, treatment, and prognosis of CTDH in adults, and the natural history and radiographic features were lacking in the literature [8, 15, 16]. This article describes 31 cases of adult thoracic calcific discitis with herniation are described and divided into three subtypes according to the preoperative CT and MRI. We speculate that different types of CTDH may indicate different natural histories, pathogenesis, and clinical features.

Methods

Study design and subjects

CTDH was defined as calcification of the nucleus pulposus in the intervertebral space and calcified lesion contiguous with the disc space protruding into the vertebral canal and compressing the spinal cord and nerve root [3, 8, 13]. The exclusion criteria were diffuse idiopathic skeletal hyperostosis, spinal infections, spinal compression fractures, malignant spine tumors, thoracic kyphosis, and scoliosis rotational deformity. From January 2004 through May 2021, 31 consecutive adult patients diagnosed as CTDH with myelopathy (T1/2–T12/L1) were treated surgically at our hospital. Among them, 22 patients were males, and nine were females, with an average age of 50.03 ± 9.60 years (30–73 years). All patients had neurological symptoms caused by thoracic disc disease with myelopathy (TDM) that warranted surgery. This study was approved by the ethical committee of our hospital and was conducted according to the principles of the

Declaration of Helsinki. All participants provided written informed consent.

Data collection and radiographic measurements

Information on the demographic characteristics, the initial and preoperative complaints, predisposing factors, causes of aggravation, compressed segments, and preoperative disease duration was assessed. In addition, we evaluated the clinical examination findings, including deep tendon reflexes (DTRs), Babinski sign, and the modified Japanese Orthopedic Association (mJOA) scoring system (full score, 11 points) [17]. Preoperative lower limb muscle strength included assessment of the iliopsoas (IP), quadriceps femoris (QF), tibialis anterior (TA), and gastrocnemius (GS) using manual muscle testing (MMT 0–5 grades). Manual muscle testing grades < 4 were clinically weak [18]. Laboratory indicators were collected, such as leucocyte count, erythrocyte sedimentation rate, C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR). Intraoperative findings and postoperative specimen pathology were also analyzed.

According to preoperative radiographic examination, CTDH was divided into three types: calcium-ringed lesion type (type A): Preoperative CT showed that the central part of the components protruding into the spinal canal was with soft tissue density and a calcific rim and slightly hypointense relative to the vertebral body marrow in the periphery and hyper-intense centrally on T2-weighted MRI (Fig. 1a, b, c, d). Heterogeneous calcification lesion (type B): CT showed calcification in the intervertebral space, with some calcified tissue mixed with low-density tissue (heterogeneous density) in the spinal canal (non-uniformity calcification) (Fig. 2a, b, c, d). Homogeneous calcification lesion (Type C): CT showed uniform and high-density calcification in the spinal canal (dense calcification) (Fig. 3a, b, c, d).

Statistical analysis

Statistical analysis was performed using SPSS (IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.). Categorical and continuous data were compared using Chi-square or Fisher's exact test and the independent t test. The two-tailed significance level was set at $p < 0.05$. Comparisons among the subtypes of CTDH were made with the Mann–Whitney *U* test after the Kruskal–Wallis test, and a corrected $p < 0.05$ (corrected by the Bonferroni method) was used to indicate significance. Intra-observer reproducibility and inter-observer reliability were calculated using the intra-class correlation coefficient (ICC) for occupancy ratio and classification [19].

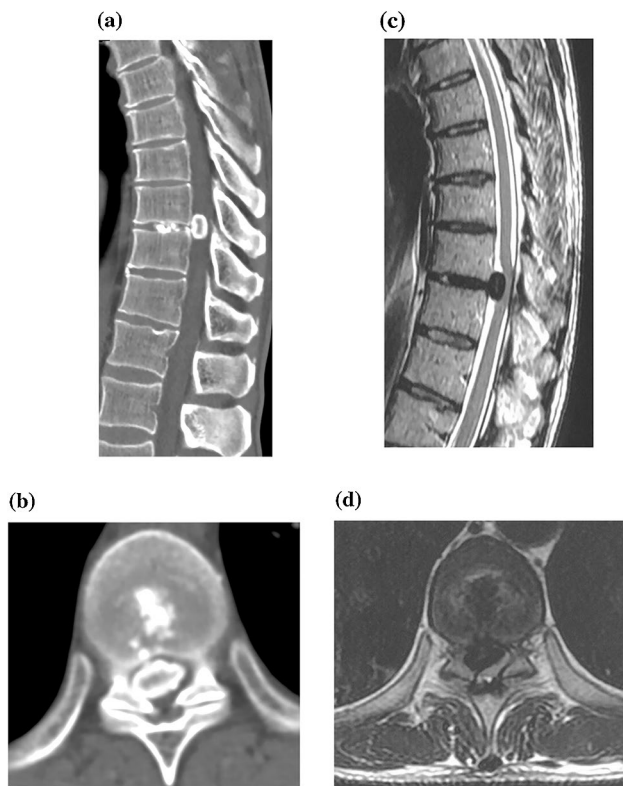


Fig. 1 Calcium-ringed lesion type: Calcification of the nucleus pulposus at the intervertebral level of T9-10 and a giant calcified HTD at the T9-10 level was demonstrated in the sagittal CT reconstruction (a). The central part of the protrusion into the spinal canal was with soft tissue density and a calcific rim and slightly hypointense relative to the vertebral body marrow in the periphery and hyper-intense centrally on MRI (c). Axial views of CT and MRI at the T9-10 level (b, d)

Results

Clinical and radiographic features of CTDH

The mean preoperative disease duration was 17.05 months (0.50–120.00 months). In 3 (9.68%) patients, their stories suggested an acute symptomatic onset [lifting heavy objects (1), fall damage (2)], different from the majority of patients with calcified disk herniations, which tend to be more insidious. The complaints at onset and preoperative are summarized in Fig. 4. At the onset, the most frequent complaint was low limb numbness (35.5%), followed by back pain (25.9%). Symptoms of 15 (48.4%) patients worsen significantly at 11.82 (0.30~35.00) months after onset, and only one patient has specific triggers (fall damage). The most frequent preoperative complaint was low limb numbness (80.6%), followed by subjective lower limb weakness (77.4%). The frequency of all complaints increased from onset to surgery. Twenty-one (67.7%) patients had decreased tactile sensation. Reduction of touch sensation, pain sensation, and both tough and

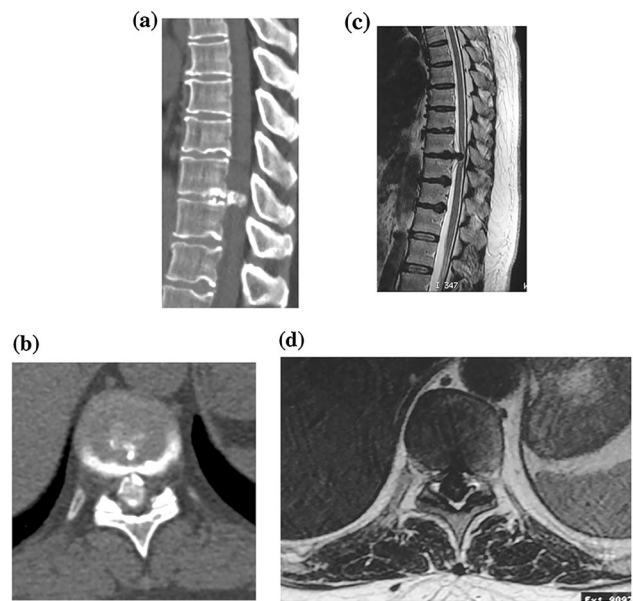


Fig. 2 Heterogeneous lesion type: CT showed calcification of the nucleus pulposus at the intervertebral level of T9-10 with some calcified tissue mixed with low-density tissue (heterogeneous density) in the spinal canal (non-uniformity calcification) (a, b). Sagittal c and axial d T2-weighted MR images demonstrating a large central TDH at T9-10, causing severe cord compression

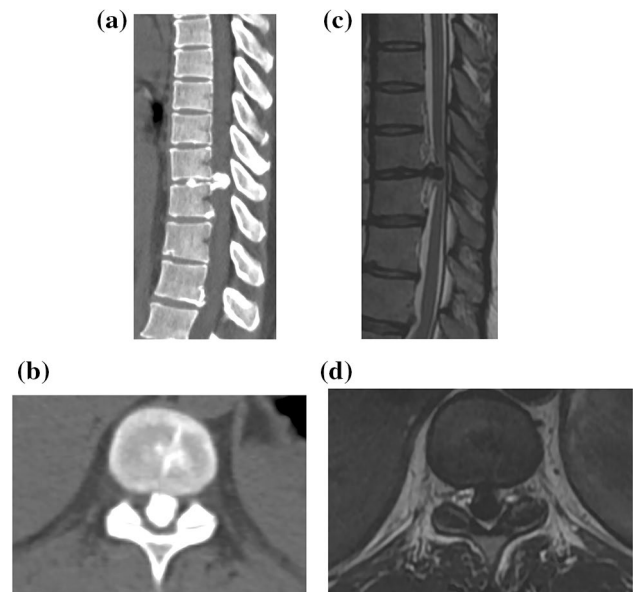


Fig. 3 Homogeneous lesion type: CT showed calcification of the nucleus pulposus at the intervertebral level of T10-11 a and high-density calcification in the spinal canal (dense calcification). Axial views of CT at the T10-11 level (b). Sagittal c and axial d T2-weighted MR images demonstrating a large central TDH at T10-11, causing severe cord compression with a spinal canal occupancy ratio of 80% and high signal in MRI. Axial MRI showed a calcified herniated thoracic disc compressing the spinal cord to a U-shape

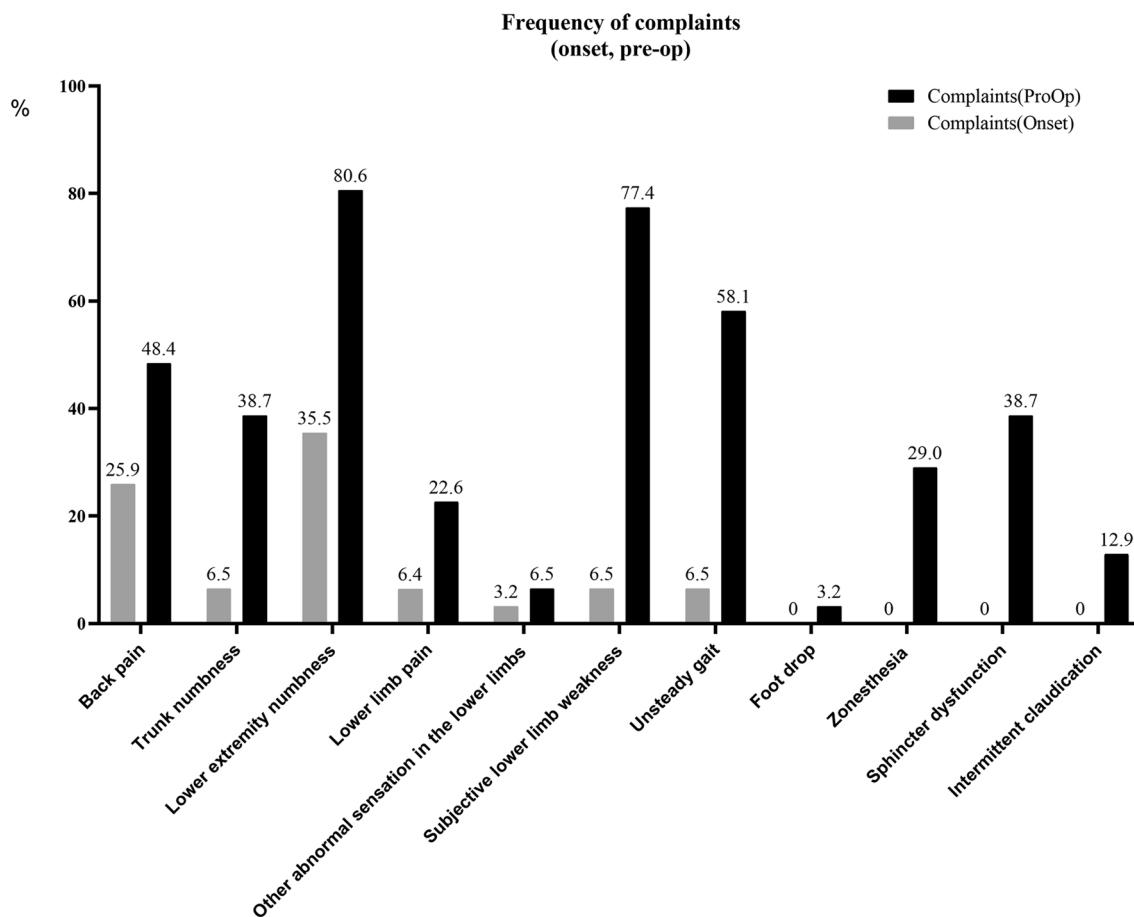


Fig. 4 Frequency of complaints (onset, pre-op). At the onset, the most frequent complaint was low limb numbness (35.5%), followed by back pain (25.9%). The most frequent complaint at preoperative

was low limb numbness (80.6%), followed by subjective lower limb weakness (77.4%). The frequency of all complaints increased from onset to surgery

pain sensation in the trunk were observed in 1 (3.2%), 2 (6.5%), and 9 (29.0%) patients, respectively. Reduction of touch sensation, pain sensation, temperature sensation, and both touch and pain sensation in low limbs were observed in 3 (9.7%), 7 (22.6%), 1 (3.2%), and 13 (41.9%) patients, respectively. Hyperalgesia in the low limb was observed in 1 (3.2%) patient. Four (12.9%) patients had lower limb muscle atrophy. Lower limb muscle weakness was observed in 12 (38.7%), 8 (25.8%), 10 (32.3%), and 11 (35.5%) patients for the IP, QF, TA, and GS, respectively. The average preoperative JOA score was 5.05 ± 2.01 (range 1–9). The preoperative Frankel grade was D in 18 patients, C in 4 patients, and B in 9 patients. No patient had a fever. The leucocyte count, erythrocyte sedimentation rate, CRP, and ESR were within normal limits.

Among the patients with CTDH, the distribution was predominantly at the T7–8 (16.1%), T8–9 (16.1%), T9–10 (19.4%), and T10–11 (22.6%) segments in 74.2% of all compressed segments (Fig. 5). The herniated discs had a mean canal occupancy of $74.90 \pm 15.16\%$ (42.15–90.03%). No

patient had a fever, and leucocyte count, erythrocyte sedimentation rate, CRP, and ESR were within normal limits.

Clinical and radiographic features of different types of CTDH

The classification of CTDH showed excellent intra-observer and inter-observer reliability (ICC = 0.98 and 0.94, respectively), indicating that the classification was reliable. There were 5 (16.1%) calcium-ringed lesions (type A), 19 (61.3%) heterogeneous lesions (type B), and 7 (22.6%) homogeneous lesions (Type C). The demographic data, clinical character, and radiographic features of three subtypes of CTDH are shown in Table 1. The age of patients with homogeneous lesions was 58.29 ± 7.50 years at surgery, older than patients with the calcium-ringed lesion ($P = 0.042$). The mean preoperative disease duration of patients with homogeneous lesions was longer than that of patients with calcium-ringed lesions

Fig. 5 Distribution of compressed segments based on anatomical pathology. The histogram demonstrates the segmental distribution of CTDH patients. Most CTDH is mid-lower thoracic segments between T7 and T11 (74.2%). The most common levels of involvement are T10-11 (22.6%), T9-10 (19.4%), T7-8 (16.1%), and T8-9 (16.1%)

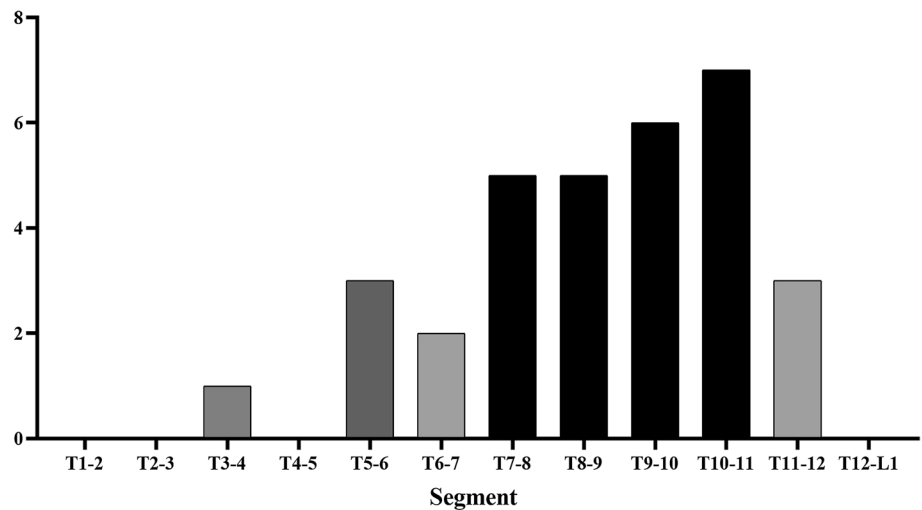


Table 1 Statistical comparison of patients with calcified thoracic disc herniation in three subtypes

	Calcium-ringed lesion (type A)	Heterogeneous lesion (type B)	Homogeneous lesion (Type C)	Three-group comparison p-value	Two-group comparison p-value
No. of patients	5(16.13%)	19(61.29%)	7(22.58%)		
Male: female	5:0	13:6	4:3	0.335	
Age at surgery, years (mean ± SD, range)	45.40 ± 9.63 (30–56)	48.21 ± 8.85 (35–62)	58.29 ± 7.50 (37–73)	0.024*	A–B: $p > 0.999$; A–C: $p = 0.042^*$ B–C: $p = 0.052$
Preoperative disease duration, months (mean ± SD, range)	3.50 ± 4.85 (0.50–12)	12.00 ± 11.39 (1–48)	40.43 ± 40.98 (2–120)	0.012*	A–B: $p = 0.110$; A–C: $p = 0.008^*$ B–C: $p = 0.343$
Preoperative mJOA score (mean ± SD, range)	3.00 ± 1.41 (1–4)	5.21 ± 2.21 (2–9)	6.07 ± 1.17 (5–8)	0.033*	A–B: $p = 0.092$; A–C: $p = 0.034^*$ B–C: $p = 0.966$
Ventral-occupying rate, % (mean ± SD, range)	77.81 ± 8.58 (66.11–86.75)	76.45 ± 9.97 (43.16–90.03)	69.01 ± 26.87 (20.01–90.00)	0.803	
Axial density of the herniation (HU)	369.88 ± 143.34	326.45 ± 134.37	684.13 ± 249.03	0.003*	A–B: $p > 0.999$; A–C: $p = 0.020^*$ B–C: $p = 0.002^*$
Sagittal density of the herniation (HU)	373.65 ± 106.83	344.06 ± 107.43	629.28 ± 180.16	< 0.001*	A–B: $p > 0.999$; A–C: $p = 0.012^*$ B–C: $p < 0.001^*$
Density of the intervertebral part (HU)	165.74 ± 60.41	214.79 ± 54.69	287.77 ± 67.36	0.004*	A–B: $p = 0.328$; A–C: $p = 0.004^*$ B–C: $p < 0.028^*$

SD Standard deviation, mJOA Modified Japanese Orthopaedic Association score, HU Hounsfield units

* $p < 0.05$

(40.43 months vs. 3.50 months, $p = 0.008$). Those with homogeneous lesions had higher preoperative mJOA scores than the calcium-ringed lesions ($P = 0.034$).

There was no significant difference in preoperative clinical symptoms and signs between the three types. No significant difference was seen in the ventral-occupying rate of the spinal canal among the three types of patients. The distribution of compressed segments based on anatomical pathology of different types of CTDH showed no significant difference ($P > 0.05$). The mean HU density of calcification in the homogeneous calcification type was higher than the other two types in the intraspinal calcified lesion and the intervertebral part.

Intraoperative finding and postoperative pathology of different types of CTDH

Six patients underwent anterior decompression and spinal fusion surgery, and 25 underwent posterior circumspinal decompression and spinal fusion [5, 17, 20]. In the calcium-ringed lesions, tough-texture bulbous cystic protrusions into the spinal canal were observed, and the white viscous fluid flowed out after the rupture of the cystic wall and then toothpaste/white sandy substance. Pathological examination revealed calcium deposition, infiltration of megakaryocytes and inflammatory cells, stromal degeneration, and formation of small vessels. The

calcified herniated disc was like irregular white gravel-like calcification in the heterogeneous lesion, and postoperative pathology showed multiple calcification foci with interstitial hyaline degeneration and necrosis, during which small vessels could be seen. In the homogeneous lesion, we observed osteoid structures with calcification and adhesion to the dura mater, and postoperative specimen pathology showed transparent cartilage and fibrous tissue with degeneration, necrosis, calcium deposition, and heterotopic ossification formation.

Five patients (16.1%) developed a cerebrospinal fluid leak (CSF), and the CSF rate in the homogeneous lesion (3 patients, 42.9%) was significantly higher for patients in the heterogeneous lesion (2 patients, 10.5%). Seven patients, 4 (21.1%) in the heterogeneous lesion and 3 (42.9%) in the homogeneous lesion, suffered transient neurological deterioration after surgery. Although there was no statistical difference, the proportion of transient neurological deterioration in the homogeneous lesion group was higher than in the heterogeneous lesion. Four recovered six months after surgery, and one gradually recovered after surgery and exceeded the preoperative level. One patient was lost at follow-up, and the mean follow-up mJOA score was 9.25 ± 1.72 . The postoperative Frankel grade was *E* in 18 patients, *D* in 7 patients, *C* in 2 patients, and *B* in 3 patients. At the final follow-up, 22 patients improved by at least 1 Frankel grade, and one case (1/31, 3.2%) in the homogeneous lesion had permanent neurological deterioration.

Case presentation of a special patient

A 32-year-old female came to the outpatient department, complaining of pain, weakness, and numbness in both lower limbs for five months and aggravation for two months with trunk numbness below the navel, without urination and defecation or sexual dysfunction. She did not report any trauma history. Her clinical examination revealed that the sensation was decreased below the T10 level, and the bilateral Babinski sign was positive. No muscle strength impairment or tendon reflex was reported. After clinical evaluation, MRI and CT scans showed a heterogeneous lesion in the spinal canal (Fig. 6a, b, c, d). The MRI revealed a massive T10-11 TDH with a median location, which deformed the anterior profile of the spinal cord. The sagittal HU density of the spinal canal part, axial HU density of the spinal canal part, and axial density of the intervertebral part were 327.3 ± 109.9 HU, 298.5 ± 118.2 HU, and 214.3 ± 131.4 HU, respectively. After fully communicating with the patient and family members about the benefits and risks, the patient refused the operation and required conservative treatment first. She was first treated with NSAIDs and neurotrophic drugs and recommended to follow up every two months. After

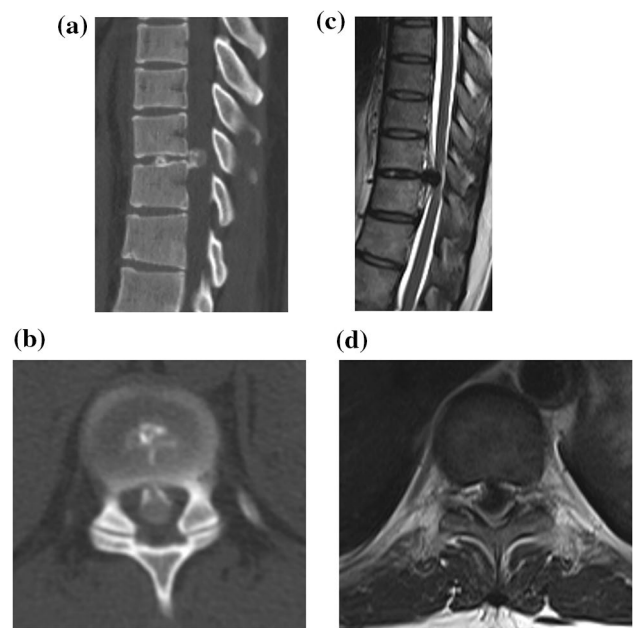


Fig. 6 CT showed calcification of the nucleus pulposus at the intervertebral level of T10-11 with some calcified tissue mixed with low-density tissue (heterogeneous density) in the spinal canal (non-uniformity calcification) (a, b). Axial views of CT at the T10-11 level (b). Sagittal c and axial d T2-weighted MR images demonstrating a large central TDH at T10-11

being discharged from the hospital, she was rechecked only once in three months, and her symptoms improved, but she has not been rechecked since then. The sagittal HU density of the spinal canal part, axial HU density of the spinal canal part, and axial density of the intervertebral part were 347.4 ± 137.1 HU, 325.5 ± 115.4 HU, and 243.4 ± 117.9 HU, respectively. All the mean HU densities showed higher values than the baseline.

After five years, the patient fell down and then developed unstable walking, numbness, and pain in both lower limbs, accompanied by temperature insensitivity to the skin of the right lower limb and intermittent claudication, frequent urination, and urgency. Her clinical examination revealed lower extremity muscle tension increased, and key muscle strengths of left and right lower extremities were grade II–III and IV, respectively. Bilateral PTR and ATR were hyperreflexia, and the bilateral Babinski sign was positive. After clinical evaluation, MRI and CT scans showed that herniation turned into the homogeneous lesion (Fig. 3). The sagittal HU density of the spinal canal part, axial HU density of the spinal canal part, and axial density of the intervertebral part were 674.1 ± 204.2 HU, 702.0 ± 237.9 HU, and 236.8 ± 192.9 HU, respectively. T10-11 posterior circumspinal decompression and T9, 10-T12 pedicle screw fixation were performed.

Discussion

We have presented a large case series of 31 patients with adult herniated thoracic calcific discitis and divided the CTDH into three subtypes according to preoperative imaging. The onset of CTDH was generally insidious, but some of the patients had apparent predisposing factors before the development of initial symptoms, and nearly half of the symptoms worsened significantly after onset. The compressed segment distribution was predominantly at the T7–11 level; all were giant thoracic disc herniation. There were significant differences in radiographic and clinical features, intraoperative findings, and postoperative pathology among patients with different types of CTDH. A special case observed five years demonstrated that the heterogeneous calcification type could switch to the homogeneous one.

The calcification mechanism of CTDH remains unknown. Most common theories believe that viral infection or trauma may cause this condition, but these theories contradict other studies [10]. In our present study, infection indicators were within normal limits. Moreover, less than 10% of patients have a clear history of trauma before developing initial symptoms. Previous reports have revealed that causes of intervertebral disk calcification (IDC) include alkaptonuria, hemochromatosis, amyloidosis, hyperparathyroidism, and degenerative changes in the vast majority of cases [21], and the prevalence of IDC was 5–6% [4]. None of the metabolic disorders mentioned above were diagnosed in all CTDH patients in the study. Adult CTDH is characterized by calcification of the nucleus pulposus in the intervertebral space and calcified lesion contiguous with the disc space protruding into the vertebral canal and compressing the spinal cord, which is different from IDC.

All the patients met giant thoracic disc herniations defined by Hott et al. [22], and postoperative pathology revealed calcium salt deposits. The homogeneous calcification type was significantly older and had a longer mean preoperative duration and higher preoperative mJOA scores. That shows that this calcification is slow progress, in which spinal cord compression and herniated discs adapt to each other. It can be assumed that calcification can develop long-term stable calcium deposits in the thoracic intervertebral discs. At this stage, no clinical symptoms can be observed. Subsequently, spontaneous liquefaction and inflammation destroyed the annulus fibrosus, eventually leading to thoracic disc herniation and severe neurological symptoms.

Calcific discitis is a self-limiting disorder that is generally considered only in children. Literature on the natural history and treatment of adults with this condition is

rare, especially when it manifests as a giant herniated disc protruding into the spinal canal. A few papers reported that a calcified HTD might sometimes regress spontaneously [8, 13, 23]. The previously reported cases of spontaneous absorption are mostly soft or central low-density types [8, 13, 23]. Martinez-Quinones et al. [24] described traditional theories that might explain the regression of calcified TDH: mechanical retraction and readjustment of the nucleus pulposus extrusion in the intervertebral space; dehydration and subsequent absorption of the hernia; and enzymatic degradation of the herniated disc. The calcium-ringed lesion contains a viscous fluid and permits rapid progression and absorption, allowing the possibility of conservative treatment. The case changing from the heterogeneous lesion to the homogeneous one implied that the heterogeneous and the homogeneous calcification types might not be spontaneously resolved. Conservative treatment is suggested for patients asymptomatic or with mild myelopathy in calcium-ringed lesions. However, it is necessary to recommend that patients regularly review imaging and monitor changes in symptoms. Decompression surgery should be performed as early as possible for patients with aggravating myelopathy or no signs of regression with conservative management. Attention should be paid to the hardness of the calcified and herniated intervertebral disc before surgery, which was important for subsequent surgical resection and provided good suggestions for the surgical management of future similar cases.

This study is a large sample single-center retrospective study. We review 31 cases of adult CTDH proposing a classification of CTDH and describing the clinical and radiographic characteristics. Furthermore, we compare the clinical features of different types of CTDH. According to our present knowledge, no literature has reported the above. However, this study also has some limitations. This study is a single-center retrospective study, and the low incidence of CTDH may have a specific impact on the results. And the specific composition of calcium salt deposits is unknown. Secondly, the cases we included were all CTDH with myelopathy needing surgery, and their radiographic and clinical features could not reveal the true natural course of the disease. Thirdly, it is impossible to judge whether these three subtypes are different states of the same disease or have different pathogenesis, which requires further research. Fourth, some case reports reported that the calcium-ringed lesion type could be absorbed spontaneously, but our data did not support a non-surgical approach to the calcific-rimmed type. Thus, we only recommend conservative treatment for patients resenting with back pain and without myelopathy.

Conclusion

Adult CTDH occupies a special rank in the field of TDH, with low incidence, insidious onset, and a high rate of spinal canal ventral-occupying ratio. Patients treated with surgery mainly manifest myelopathy, and only about 1/4 of patients complain of back pain at the onset. The calcium-ringed lesion type with a viscous fluid is why it rapidly progresses and can be absorbed in some reports, providing the possibility of conservative treatment in patients without myelopathy. The heterogeneous lesions can progress to a homogeneous lesions. Calcium deposits in the spinal canal originate from the nucleus pulposus, while the pathogens are unknown and still need further study.

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Declarations

Conflict of interest None of the authors has any potential conflict of interest.

Ethical approval The device(s)/drug(s) is/are FDA approved or approved by the corresponding national agency for this indication.

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