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ORIGINAL PAPER

Risk factors associated with post-operative neurological deterioration in patients with thoracic disc disorders with myelopathy

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Abstract

Purpose Post-operative neurological deterioration (ND) is a severe complication. However, limited literature exists on the ND in thoracic disc disorders with myelopathy (TDM). This study describes the risk factors of neurological deterioration in TDM with instrumentation and fusion.

Methods A single-centre review of TDM with instrumentation and fusion during 2006–2019 was performed. Post-operative neurological deterioration was defined as the deterioration of pre-existing neurological function or the appearance of new neurological symptoms. Patients were then grouped into two groups depending on neurological deterioration (ND group) or not (non-ND group). Demographics, radiographic parameters, and surgical characteristics were compared between the two groups.

Results A total of 257 cases were included, and neurological deterioration occurred in 16 (6.23%) cases. Multivariate analysis revealed spinal canal occupancy ratio > 75%, U-shaped compressed spinal cord in axial MRI, calcified herniated disc, anterior approach, and intra-operative blood loss > 1500 mL were associated with ND. Ten patients (62.5%) had complete neurological recovery within six months, and four patients (25%) had progressive neurological function improvement and equal or better than pre-operation within nine months.

Conclusions The rate of neurological deterioration is 6.23%, and a higher spinal canal occupancy ratio, U-shaped compressed spinal cord, calcified herniated disc, anterior approach, and massive intra-operative blood loss were associated with neurological deterioration. Long-term outcomes of neurological deterioration are favourable, and 62.5% of patients experienced complete neurological recovery within six months. Patients with TMD who undergo surgery should be properly informed of the potential risks of neurological deterioration, despite its usually transient nature in most patients.

Keywords Thoracic disc disorders with myelopathy · Neurological deterioration · Spinal canal occupancy ratio

Introduction

Thoracic disc herniation (TDH) is one of the pathological factors for progressive and severe thoracic myelopathy caused by anterior compression. Literature reports that the rate of clinically symptomatic TDH is between 1 in 1000 and 1 in 1000,000 patient-years, accounting for 0.1–3% of all spinal disc herniation, evidencing a relatively unusual condition [1, 2]. The presentation of TDH is a combination of ataxia, the motor deficit in lower limbs, paresthesia, and bowel and bladder symptoms. Patients presenting with neurological symptoms of thoracic myelopathy due to TDH typically require surgery [3]. The main goal of surgery is to provide sufficient decompression and minimize manipulating the compressed spinal cord. Although this disease is uncommon, various surgical approaches have been developed. These techniques include the transpedicular, extracavitary, costotransversectomy approach, and anterior techniques, such as thoracotomy and thoracoscopy [4].

The anterior approach (transthoracic or retroperitoneal) has advantages in providing a direct view of the herniated discs and taking the discs away from the cord ventrally. However, the visualization provided by the anterior transthoracic approach is still inadequate because that the neural elements on the other

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side of the spinal canal are not in direct view for most of the decompression process [5]. Furthermore, the anterior approach is technically demanding and associated with higher complications [6]. The posterolateral/lateral approaches, including costotransversectomy and extracavitory approach, all essentially performed from one side, most suitable for lateral, soft herniated discs [3]. However, these approaches do not permit clear visualization and smooth excision of the lesion on the other side of the spinal canal that is not exposed in central TDHs, which may predispose to incomplete decompression or inadvertent cord manipulation. We previously reported the posterior circumspinal decompression and fusion procedure, which includes a laminectomy plus bilateral facet joint excision and transforaminal decompression, could offer a genuinely wide surgical view, and it allows bilateral, interactive manipulation of the central hard disc matter, thus facilitating decompression and reducing the risk of cord injury. However, it required wide resection of normal structures and instrumentation and fusion [5, 7].

Timely adequate decompression around the spinal cord plays a critical role in neurological recovery in TDM. Still, thoracic discectomy has a wide array of reported complications, with a complication rate of 20 to 30% [1, 8, 9]. Neurological deterioration is a serious post-operative complication and can result in a worsening of neurological function, paraplegia, or incontinence [10]. The majority of the neurological deficits are transient, but permanent injuries are possible [11]. The pathogenesis of neurological deterioration is unknown, although several hypotheses have been suggested: medullary inhibition, spinal shock, medullary contusion, or vascular problem [2]. Characteristics of the herniated disc (such as giant calcified TDH), vascular underperfusion following cord manipulation, surgery approach, intraoperative neurophysiological monitoring (IONM) changes, and pre-existing neurological symptoms have been found associated with a risk of new neurological deterioration [1, 3, 8, 12–14]. However, all of these case series suffers from small sample size and a lack of data on various possible complications, which fail to explore post-operative neurological deterioration risk factors.

Due to low incidence and specific pathological characteristics, TDM still is a great challenge in terms of surgical technique and potential adverse events. We present the largest single-center consecutive cohort to date of TDM treated with instrumentation and fusion. The goals of the present study were to report the incidence and identify risk factors for neurological deterioration.

Materials and methods

Patients

The study was approved by the Medical Science Research Ethics Committee of our institution (IRB00006761-M2020049) and registered in the Chinese Clinical Trial Registry (registration ID: ChiCTR2000032957). This study was conducted according to the principles of the Declaration of Helsinki. The informed consent was waived because this was a retrospective study. A consecutive retrospective examination of thoracic disc disorder with myelopathy who received instrumentation and fusion during 2006-2019 was performed. Thoracic myelopathy caused by diffuse idiopathic skeletal hyperostosis, spinal infections, spinal compression fractures, malignant tumours of the spine, thoracic kyphosis, and scoliosis rotational deformity was excluded. Finally, 257 patients were enrolled: 194 males, 63 females; mean age 48.13 ± 14.15 years (range, 19 to 84 years). All surgical strategies and approaches were discussed and decided by these surgeons before the operation. In all cases included in this study, meticulous surgery was performed by the senior authors, who had at least 15 years of thoracic spinal surgery experience before. All surgical strategies and approaches were discussed and decided by these surgeons before the operation. One hundred and ninety-four patients underwent posterior decompression and fusion (unilateral transforaminal thoracic interbody fusion [TTIF] or posterior circumspinal decompression and fusion [PCDF]), and 63 cases decompressed and fused by anterior transthoracic approach. No loupes, microscope, or video-assisted endoscopy were used in operation.

Data collection

Electronic medical records were reviewed for the patients, and demographic, radiographic values, and surgical data were collected. Neurological deficits were defined as a worsening of neurological status compared to baseline or the development of new neurological symptoms. Lower limb muscle strength was assessed using a 0–5 scale: 0/5, no movement; 1/5, the flicker of movement; 2/5, full range of motion (ROM) without gravity; 3/5, full ROM against gravity; 4/5, full ROM against slight resistance; and 5/5, full ROM against strong resistance [14]. According to the occurrence of postoperative neurological deterioration, patients were divided into two groups: the neurological deterioration (ND) group and the non-ND group. Demographics, radiographic parameters, surgical characteristics, and neurological complications were compared between the two groups.

Neurophysiological monitoring

Intra-operative use of somatosensory evoked potentials (SSEP) and motor evoked potential (MEP) monitoring for neurological monitoring was also recorded. Any amplitude reduction of more than 50% from baseline was defined as neuromonitoring alerts [15]. When neurophysiological monitoring alerts, the surgeon was notified, and a surgical pause was done to explore for possible aetiologies. The

anesthesiologist evaluated mean arterial pressure, temperature, haematocrit, pCO2, and pH. Electrode and body position was checked, and possible electrical interferences were investigated. Last, screw positioning and decompression sites were analyzed.

Statistic analysis

SPSS software (version 24.0; IBM) was used for statistical analysis. Simple comparisons of continuous data between groups were carried out with the Student's t-test or Mann-Whitney U-test. Categorical variables were compared using the chi-square test or Fisher's exact test. Variables with p values smaller than 0.05 in the univariate analyses and variables reported in the previous study were entered into a multivariate logistic regression model. Odds ratios and a 95% confidence interval (CIs) were estimated for significant factors. All tests were 2-sided, and p values < 0.05 were considered significant. In the logistic regression model, the areas under the receiver operating characteristic (ROC) curves were calculated to evaluate the overall diagnostic performance of the continuous variables for ND. The optimal cutoff value of the continuous variables was calculated by Youden's index, which was estimated by summarizing sensitivity and specificity minus 1 [16]. The optimal cutoff value was used to convert the continuous variable into the dichotomous variable.

Results

Postoperative neurological deterioration was developed in 16 of 257 patients (6.23%). Detailed case descriptions were summarized in Table 1. Fourteen patients were with motor deterioration, one with sphincter, and one with motor and sensory deterioration. Eleven patients worsen the neurological status than pre-operative, and five developed new neurological symptoms. All patients were treated immediately with neurotrophic drugs, corticosteroids, and dehydration. Ten patients (62.5%) had complete neurological recovery within six months. Two patients (12.5%, cases 15 and 16) had progressive neurological function improvement and better status than pre-operation, and another two cases (12.5%, cases 5 and 8) returned to baseline levels at discharge and three months, respectively. Case 10 with paraplegia had permanent neurological deterioration in the last follow-up, and one patient (case 6) was lost to follow-up.

No statistical difference was seen in the ND group and non-ND in age at operation, gender composition, body mass index, and symptoms duration. The ND group had a lower pre-operative mJOA score (4.81 ± 2.16 vs. 5.99 ± 2.03, $p = 0.026$) and a higher spinal canal occupancy ratio (68.33 ± 16.53 vs. 57.16 ± 14.31%, $p = 0.003$). Furthermore, the ND group had a higher proportion of U-shaped compressed spinal cord in axial MRI (56.3% vs. 12.4%, $p < 0.001$) and calcified herniated disc (43.8% vs. 7.1%, $p < 0.001$). The surgery approach presented a significant difference between the two groups because the posterior approach was more common in the non-ND group. The intra-operative blood loss (800 vs. 500 mL in median, $p = 0.007$) and operation time (196.88 ± 63.47 vs. 164.54 ± 59.01 min, $p = 0.036$) in the ND group were much more than the non-ND group. No association was found between neurological deterioration and herniated disc location, kyphosis correction, mean atrial pressure, and fixed and fused levels. Patients with a neurological deficit had a longer duration of admission with discharge after a mean of 16.75 days compared to 11.16 days ($p = 0.001$) (Table 2).

Intra-operative neurophysiological monitoring(IONM) was only performed in 46 patients (5 in the ND group). Neurophysiological monitoring alerts were observed in four cases (1 in the ND group, $p = 0.379$): neuromonitoring of three patients in the non-ND group returned to baseline, and one patient (case 2) in the ND group had complete disappearance of bilateral MEP intra-operatively. The right side of case 2 had a recovery when then decompression finished and had neurological deterioration post-operatively with a muscle strength decreased at the left lower extremity. The absence of intra-operative neural monitoring alarms during these four surgical procedures in the ND group had different origins: "true" false negative where no waveform change meeting the alarm criteria occurred despite the appropriate intra-operative neural monitoring (case 4); case 9 and case 10 only recorded SSEP, and the SSEPs of them were normal during the surgery; the absence of interpretable IONM baseline data which precluded any alarm was seen in case 16. We found seven cases that showed neurological monitoring signal improvement compared with baseline, and 35 patients had no change (31 in the non-ND group and 4 in the ND group).

The risk factors for neurological deterioration in univariate and multivariate logistic regression models are summarized in Table 3. Six variables obtained statistical significance ($p < 0.05$) in the univariate logistic regression models and were further analyzed using multivariate logistic regression. After model selection, pre-operative mJOA less than 7 (OR = 1.968, 95% CI = 0.502–7.715, $p = 0.331$) was excluded from the multivariate logistic regression model. Hence, higher pre-operative spinal canal occupancy ratio, U-shaped compression of the spinal cord, calcified TDH, anterior approach, and massive intra-operative blood loss were identified as risk factors of neurological deterioration.

Discussion

The present study retrospectively reviewed a total of 257 patients with thoracic myelopathy caused by the thoracic disc herniation and analyzed the incidence and possible risk factors of post-operative neurological deterioration after surgical

Table 1 Case descriptions in 16 patients with neurological deterioration

Case no.	Age(yrs), Sex	Symptoms duration (mos)	Characters of herniated discs and compressed cord	Levels	Operation	Neuro complications	Neuro outcome
1	37, M	3	TDH with PBA	T11–12	AEA	Lt LE sensory deficits Lt IP: 4/5→3/5	Full recovery in 3 mos
2	54, M	54	TDH with calcification U-shaped spinal cord	T8–9	PCDF	Loss of Rt LE MEP Rt LE:3/5→1/5	Full recovery w/in 6 mos
3	52, M	48	TDH with PBA	T10–11	AEA	Uroschesis	Full recovery at discharge
4	65, F	48	TDH with PVO	T8–9	Unilateral TTIF	No neuromonitoring alerts Lt LE: 5/5→3/5	Full recovery in 6 mos
5	49, M	84	THDO	T11–12	AEA	Rt TA:2/5→0/5	Back to baseline at 3mos
6	43, M	7	TDH with calcification U-shaped spinal cord	T9–10	ATA	Lt 2–3/5, Rt 3–5/5→0/5 Bilat	Lost in FU
7	60, F	8	TDH with PBA	T12–L1	AEA	Lt TA: 5/5→4/5	Full recovery at discharge
8	67, F	120	TDH with PVO	T12–L1	AEA	Lt IP:4/5→0/5	Back to baseline at discharge
9	38, M	12	TDH with calcification U-shaped spinal cord	T6–7	PCDF	No neuromonitoring alerts 2–4/5→0/5 Bilat	Full recovery in 2 mos
10	37, F	65	TDH with calcification U-shaped spinal cord	T10–11	PCDF	No neuromonitoring alerts 3–5/5→0/5 Bilat	Persistent paraplegia
11	52, F	6	TDH with calcification U-shaped spinal cord	T3–4	PCDF	Lt LE: 4–5/5→ 0–3/5	Full recovery in 6 mos
12	62, F	36	Giant thoracic osteophyte; U-shaped spinal cord	T2–3	PCDF	Lt LE:5/5→3/5	Full recovery at discharge
13	46, M	72	TDH with PBA	T11–12	AEA	Bilat TA:5/5→ 2/5	Full recovery at discharge
14	57, F	3	TDH with calcification U-shaped spinal cord	T9–10	AEA	Bilat LE:4/5→ 2/5	Full recovery in 3 mos
15	52, M	12	TDH with calcification U-shaped spinal cord	T11–12	Unilateral TTIF	Lt 0/5, Rt 3–4/5→Bilat 0/5	Progressive improvement of strength w/in 6 mos; 4/5 strength bilat LE
16	23, M	48	TDH with PBA; U-shaped spinal cord	T10–11	PCDF	No neuromonitoring alerts Lt 3/5, Rt 0/5 → Bilat 0/5	Progressive improvement of strength w/in 9 mos; 3–4/5 strength bilat LE

TDH, thoracic disc herniation; *THDO*, THD with annulus fibrosus or posterior longitudinal ligament ossification; *TDH with PBA*, TDH with posterior bony avulsions of the vertebrae; *TDH with PVO*, TDH with posterior vertebral osteophytes; *GTO*, giant thoracic osteophyte. *PCDF*, posterior circumspinal decompression and spinal fusion; *ATA*, anterior transpleural approach; *AEA*, anterior extrapleural approach; *TTIF*, transforaminal thoracic interbody fusion; *LE*, lower extremity; *IP*, iliopsoas, *TA*, tibialis anterior

treatment. In the current study, 6.23% of the patients (16/257) occurred neurological deterioration. Results showed that neurological deterioration was significantly associated with the higher pre-operative spinal canal occupancy ratio, U-shaped compression of the spinal cord, calcified TDH, anterior approach, and massive intra-operative blood loss. The severity and prognosis of post-operative neurological complications varied. Deficits can result in paraparesis, but most are transient in nature and recovered within nine months.

The literature on neurological deterioration following thoracic surgery in the treatment of TDH is limited to small case series. Furthermore, the criteria used for ND diagnosis are different among the studies. The majority of authors use the mJOA score, ASIA score, or Frankel grade to evaluate post-operative findings. To obtain a more complete view, we defined neurological deterioration as the deterioration of pre-existing neurological function or the appearance of new neurological symptoms. Neurological deterioration was seen in

Table 2 Comparison of characteristics between patients with neurological deterioration and those without

Variable	ND(n = 16)	Non-ND(n = 241)	p value
Age (years)	49.63 ± 11.86	48.34 ± 14.23	0.725
Female (n, %)	7(43.8%)	56(23.2%)	0.122
BMI (Kg/m^2)	25.71 ± 3.69	26.85 ± 4.05	0.275
Duration of symptoms (months)	24 (6.5–56.5)	24(6–48)	0.778
Pre-operative mJOA (score)	4.81 ± 2.16	5.99 ± 2.03	**0.026***
Pre-op Frankel (A: B:C:D)	0:6:3:7	3:77:12:149	0.110
ASA (I: II: III)	3:13:0	97:138:6	0.161
Spinal canal occupancy ratio (%)	68.33 ± 16.53	57.16 ± 14.31	**0.003****
U-shape of spinal cord (n, %)	9(56.3%)	30(12.4%)	**< 0.001****
Calcified TDH (n, %)	7(43.8%)	17(7.1%)	**< 0.001****
Location(upper/middle/lower)†	2:2:12	8:21:212	0.265
Kyphosis correction(°)	2.55(0.00–5.30)	3.90(0.70–7.50)	0.363
MR T2-hyperintensity	11 (68.8%)	111 (46.1%)	0.078
Surgical approach (n, %)			
Anterior	8(50%)	55 (22.8%)	**0.032***
Posterior	8(50%)	186(77.2%)	
Operation time (min)	196.88 ± 63.47	164.54 ± 59.01	**0.036***
Blood loss (mL)	800(575-2250)	500(300-900)	**0.007****
MAP (mmHg)	81.44 ± 6.46	82.65 ± 7.66	0.535
Fixed and fused levels	1.5(1–3)	1(1–2)	0.398
Length of hospital stay in days	16.75 ± 7.33	11.16 ± 6.22	**0.001****

ND, neurological deterioration; *BMI*, body mass index; *mJOA*, modified Japanese Orthopaedic Association score; *ASA*, American Standards Association; *TDH*, thoracic disc herniation; *MR*, magnetic resonance; *MAP*, mean arterial pressure

Values are presented as the number of cases (%), mean ± SD, or median (IQR)

† Location of the lesion (upper thoracic: T1–5, middle thoracic: T5–9, lower thoracic: T9–L1)

*$p < 0.05$, **$p < 0.01$

16 (6.23%) patients and is consistent with the incidences of ND in the previous studies, which are reported to be between 0% and 15.09% [3, 8, 10–12, 17–19].

Although most of our patients were affected in the lower thoracic spine (Th10/11, Th11/12, or Th12/L1), the configuration of the herniated disc and the relationship with the spinal

Table 3 Univariate and binary logistic regression analysis of risk factors for neurological deterioration

	Univariate analysis			Multivariate analysis		
	p	OR	95% CI	p	OR	95% CI
Pre-op mJOA < 7	**0.046***	4.634	1.030–20.851	0.331	1.968	0.502–7.715
SCCR > 75%	**<0.001****	8.397	2.922–24.128	**0.040***	4.094	1.002–16.726
U-shape of the cord	**<0.001****	9.043	3.136–26.080	**0.010***	4.812	1.547–25.321
Calcified TDH	**<0.001****	10.248	3.398–30.910	**0.030***	4.812	1.163–19.899
Anterior approach	**0.020***	3.382	1.213–9.427	**0.013***	4.896	1.402–17.095
MAP < 81 mmHg	0.296	1.723	0.621–4.778	0.373	1.719	0.522–5.659
Op time > 200min	0.088	2.454	0.875–6.880	0.929	1.069	0.243–4.707
IBL > 1500 mL	**0.005****	5.311	1.671–16.879	**0.036***	4.031	1.098–14.799

OR, odds ratio; *CI*, confidence interval; *mJOA*, modified Japanese Orthopaedic Association score; *SCCR*, spinal canal occupancy ratio; *TDH*, thoracic disc herniation; *MAP*, mean arterial pressure; EBL, interoperative blood loss

*$p < 0.05$, ** $p < 0.01$

cord are likely important determinants of the surgical risk. Anatomically, the thoracic spinal canal is smaller than the cervical or lumbar region, thus providing less room for the spinal cord. Besides, TDHs are frequently centrally located and present as "hard discs" [5, 20]. Hott et al. [21]defined giant thoracic disc herniations (gTDHs) as those occupying at least 40% of the spinal canal's diameter based on pre-operative CT myelography, MR imaging, or both. In their study, 16% and 5% gTDH patients worsened 1 Frankel grade post-operatively and at final follow-up, respectively [21]. Giant TDHs are a surgical challenge because they may be densely calcified or ossified, erode the dura, and severely compress the spinal cord [3, 8]. A larger spinal canal occupancy ratio and incorporation with the thoracic dura make the spinal cord vulnerable to even mild traction or slight vibration.

Another possible reason for the ND risk associated with a higher spinal canal occupancy ratio is reperfusion injury after decompression of a chronic compressive diseased cord. The thoracic spinal cord has a more tenuous blood supply and collateral potential than other regions [22]. Compression of the spinal cord within a higher spinal canal occupancy ratio can cause mechanical neural injury and poor blood flow, resulting in spinal cord ischemia and edema, which always present intramedullary MR T2-hyperintensity (Fig. 1c). Therefore, spinal cord ischemia, such as that due to spinal cord swelling after "surgical trauma," can have devastating effects such as paraparesis. In cases 6 and 9, post-operative MRI revealed enlargement with an expansion of the T2 high-signal intensity (Fig. 2c), which is compatible with the reperfusion injury hypothesis. Following a typical ischemic injury to the spinal cord, the release of free fatty acids, an increase of Ca^{2+} intake, and vascular permeability impair tissue metabolism. These effects can restrict the possibility of enhanced recovery and deteriorate neurologic function in the reperfusion period [23].

In the present study, the spinal cord was deformed into a U-shape in axial T2MR owing to severe anterior compression by the herniated discs in nine cases. In this situation, the herniated disc indented the spinal cord, and the lateral aspect of the cord was, in fact, ventral to the herniated discs. Most previous studies recommended a transthoracic or thoracoscopic approach for those cases [3, 8, 10, 11]. However, handling those cases was extremely intractable, whether by anterior transthoracic or lateral approaches, including extracavitory and costotransversectomy. These approaches are essentially performed one-sided, and the neural elements on the other side of the spinal canal are not in direct view for most of the decompression process. It is necessary to access the dura through the compressive lesion with the anterior approach (transthoracic or retroperitoneal), predisposing the patient to inadvertent cord injury. Roelz et al. [10] reported that 17 patients treated by mini-thoracotomy and two patients (11.76%) experienced a transient post-operative neurological decline. In another cohort of 33 cases of giant TDH, 23 patients underwent thoracotomy, nine costotransversectomy, and two transpedicular approaches; however, all three neurological deteriorations were seen in the thoracotomy group (13.04%) [3]. Brauge et al. [8] reported that 53 patients due to gTDH operated by the thoracoscopic approach, and eight patients (15.09%) had a new neurological deficit post-operatively. Another study with a slightly larger sample size showed that 5.4% of the mini-transthoracic (56 patients) and 2% of the transpedicular discectomy (44 patients) worsened neurologically [11]. The present study showed 12.70% of anterior and 4.12% of posterior occurred neurological deterioration. Our study used a posterior circumspinal decompression and fusion procedure (PCDF) to treat central giant TDH. This procedure involved laminectomy and bilateral resection of facet joints before removing the herniated discs. This procedure offered a genuinely broad surgical view and allowed bilateral, interactive manipulation of the central hard disc matter, thus facilitating decompression and reducing the risk of spinal cord injury [5].

This study found that intra-operative blood loss of more than 1500 mL was associated with ND, while no difference was noted in the MAP between the ND and the non-ND group. Recent studies demonstrated that besides the major blood supply through conduit arteries, a huge collateral network also participated in the spinal cord blood supply, which involved an extensive axial arterial network in the spinal canal, the paravertebral tissues, and the paraspinous muscles, in which vessels anastomose with one another and with the nutrient arteries of the spinal cord. The paravertebral muscles ensure blood supply to the spinal cord and endanger the central nerves by the so-called steal phenomena. During a steal phenomenon, blood is redistributed by alternate routes or reversed flow, causing hypoperfusion in the vessel bed from which blood is withdrawn [24, 25]. A massive blood loss during exposure and decompression might endanger the blood supply of the spinal cord via a steal phenomenon. On the other hand, massive blood loss might complicate the operative procedure, which was a risk factor for post-operative neurological complications. The deterioration might be partly caused by iatrogenic damage to the spinal cord during the decompression procedure due to poor visualization of the surgical site due to bleeding. However, the retrospective nature of this survey did not allow us to reach any conclusion in this regard. Therefore, the effective measures to prevent cord ischaemia are to improve surgical technique, shorten operation time, and pay more attention to haemostasis.

There were several potential limitations in the present study. First, this study was a retrospective study, including cases from a single institution, and some patients were lost in follow-up. However, this study aimed to report on perioperative neurological deterioration. Second, the present study has several biases: selection bias, performance bias. Another limitation is that the number of 16 neurological

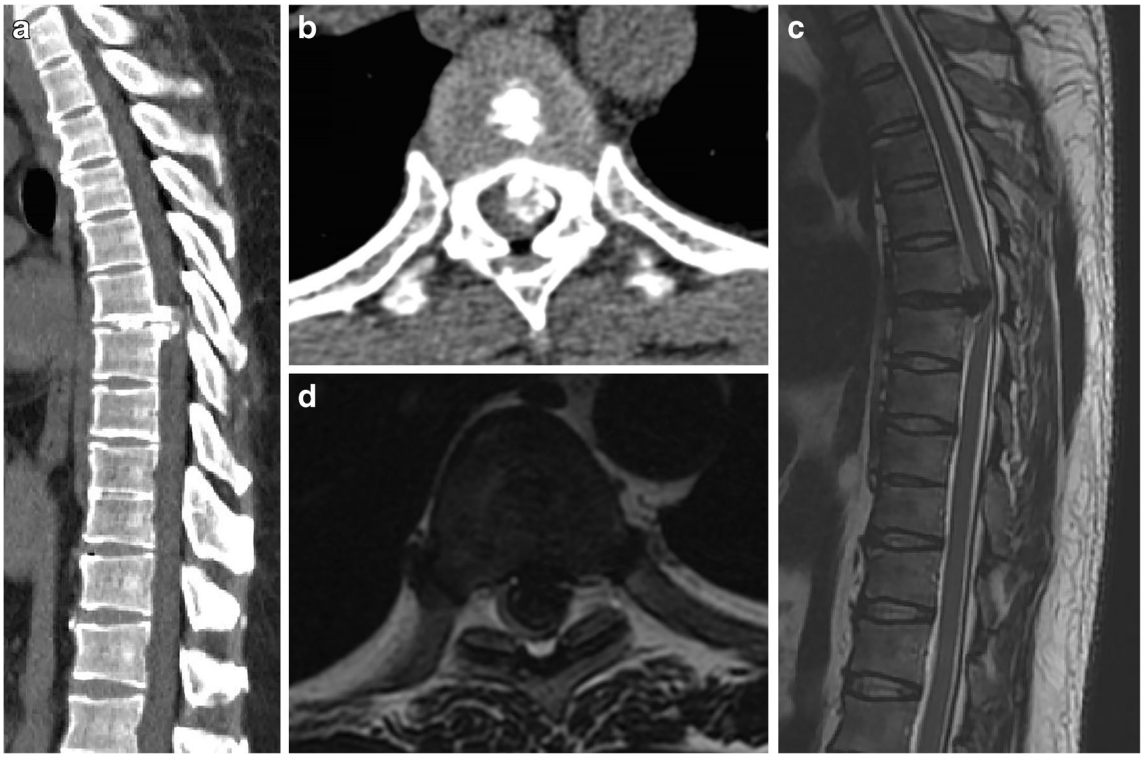

Fig. 1 Sagittal (**a**) and axial (**b**) CT images demonstrating a thoracic disc herniation with calcification of T6-7. C and D: Sagittal (**c**) and axial (**d**) T2-weighted MR images demonstrating a large central TDH at T6-7, causing severe cord compression with a spinal canal occupancy ratio of 80% and high signal in MRI. Axial MRI showed a calcified herniated thoracic disc compressing the spinal cord to a U-shape (Case 9)

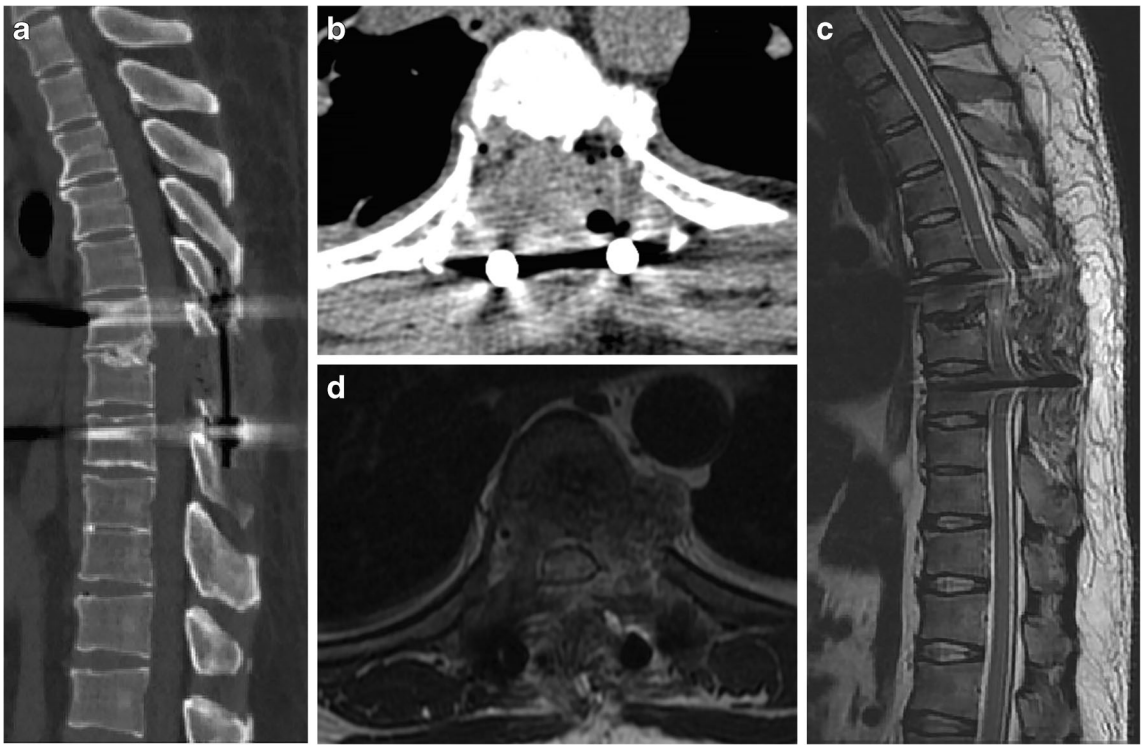

Fig. 2 Post-operative sagittal (**a**) and axial (**b**) CT image demonstrating that the herniated hard disc and the bilateral facet joints had been removed and satisfactory decompression. Post-operative sagittal (**c**) and axial (**d**) MRI image demonstrating enlargement with an expansion of the T2 high-signal intensity

deterioration cases is too low to give significant results regarding valuable statements regarding the risk factors. Meta-analyses and prospective studies with larger sample sizes are required to confirm our observations.

Conclusions

In a total of 257 cases, new or progressive neurological deterioration occurred in 16 cases (6.23%). Patients with a high occupying ratio of TDH, U-shaped compressed spinal cord, calcified herniated disc, anterior approach surgery, and massive intra-operative blood loss were at an increased risk of developing neurological deterioration. Long-term outcomes of new neurological deficits are favourable, and 62.5% of patients experienced complete neurological recovery within six months. Patients with TMD who undergo surgery should be properly informed of the potential risks of neurological deterioration, despite its usually transient nature in most patients.

Author contribution The authors confirm that all authors have made substantial contributions to all of the following:

The conception and design of the study, or acquisition of data, or analysis and interpretation of data.

Drafting the article or revising it critically for important intellectual content.

Final approval of the version to be submitted.

Sound scientific research practice.

Data availability All data generated or analyzed during this study are included in this published article.

Declarations

Ethics approval Ethics approval was obtained from the Peking University Third Hospital Medical Science Research Ethics Committee (IRB00006761-M2020049) and registering in the Chinese Clinical Trial Registry (registration ID: ChiCTR2000032957. http://www.chictr.org.cn/index.aspx).

Consent to participate This study was approved by the Ethical Committee of our hospital and was conducted according to the principles of the Declaration of Helsinki. The informed consent was waived because this was a retrospective study.

Consent to publish Not applicable.

Competing interests The authors declare no competing interests.

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