Thoracic disc herniation and acute myelopathy: clinical presentation, neuroimaging findings, surgical considerations, and outcome

Clinical article

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Object. Thoracic disc herniations (TDHs) may occasionally present with an acute myelopathy, defined as a variable degree of motor, sensory, and sphincter disturbances developing in less than 24 hours, and resulting in a Frankel Grade C or worse. Confronted with such a patient, the surgeon has to decide whether to perform an emergency operation and whether to use an anterior or posterior approach. The authors analyze their own experience and the pertinent literature, focusing on clinical presentation, imaging findings, surgical timing, technique, and outcome.

Methods. Among 250 patients who underwent surgery for symptomatic TDH, 209 had at least 1 year of follow-up at the time of writing, including 8 patients who presented with an acute myelopathy. They were surgically treated using standard thoracoscopic microdiscectomy, careful blood pressure monitoring, and intravenous methylprednisolone. The authors analyzed pre- and postoperative neuroimaging, and Frankel scores preoperatively, at discharge, and 1 year postoperatively.

Results. Although 5 patients had multiple TDHs, the symptomatic TDH was invariably situated between T9–10 and T11–12. Seven TDHs were giant, 6 were calcified, 6 were accompanied by myelomalacia, and 4 were accompanied by segmental stenosis. Although sudden dorsalgia was the initial symptom in 6, a precipitating event was noted in only 1. All patients had severe neurological deficits by the time they underwent surgery. Frankel grades improved from B to D in 2 patients, from C to E in 4, and from C to D and B to E in 1 patient each. All patients regained continence and ambulation. Transient complications were CSF leak (in 2 patients), and intraoperative blood loss greater than 1000 ml, reversible ischemic neurological deficit, and subileus (in 1 patient each).

Conclusions. Approximately 4% of TDHs present with an acute myelopathy. They are often situated between T9–10 and T11–12, large or giant, and even calcified. They almost invariably cause important cord compression (sometimes aggravated by an associated segmental stenosis) and myelomalacia. Their clinical presentation may be misleading, and diagnosis may be delayed until other causes (especially vascular) have been excluded and the clinical picture has become more complete. Interestingly, whereas a precipitating event or trauma is rarely present, dorsalgia frequently precedes profound myelopathy and may help to make an early diagnosis. Remarkable recovery is possible even with profound neurological deficit, a delay of several days, in the elderly, and in the presence of myelomalacia, provided the spinal cord is adequately decompressed and intraoperative hypotension is strictly avoided. Although alternative approaches more familiar to most neurosurgeons may be used, the anterior transthoracic approach has the advantage of reaching the TDH in front of the compromised spinal cord, avoiding any manipulation. In experienced hands, thoracoscopic microdiscectomy combines the advantage and versatility of an anterior approach with minimal postoperative discomfort. The authors conclude that TDH-related acute myelopathy may have a favorable outcome when managed correctly, and they strongly recommend that every single patient should undergo surgical treatment.

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Abbreviations used in this paper: MABP = mean arterial blood pressure; TDH = thoracic disc herniation; TMD = thoracoscopic microdiscectomy.
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cord) may remain asymptomatic for a long time,26 and when they do produce symptoms, it rarely is in an acute setting. Nevertheless, an occasional TDH will present with an acute myelopathy defined as a variable degree of motor, sensory, and sphincter disturbances developing in less than 24 hours, and resulting in a Frankel Grade C or worse.

Although some authors have stated that TDHs more frequently cause an acute paraplegia than their cervical counterparts,12,25 there are actually few case reports concerning this entity.5,6,12,14,15,17,24,28 Interestingly, we have treated 8 patients affected by an acute myelopathy (including 3 with paraplegia, 2 with severe paraparesis, and 1 with severe monoparesis) at our institution over the past 8 years. When confronted with such a patient, the surgeon has to decide whether to perform an emergency operation and if so, whether to use an anterior or posterior approach, considering the anticipated risk of such an attempt. Unfortunately, the literature brings little, if anything, that helps in making the right decision.5,6,12,15,17,24 In this study, we therefore focus on clinical presentation, imaging findings, surgical timing, surgical technique, and outcome. We hope this will contribute to our understanding of this entity and improve our care for these patients.

Methods

Overall Patient Population

Our medical center functions as a tertiary referral center mainly for Belgium and the Netherlands. Between October 2000 and December 2009, 250 patients with 1, 2, or occasionally 3 symptomatic TDHs at anatomical levels ranging from T2–3 to T12–L1 underwent surgery at our institution. For the purpose of this study, however, only patients with a minimum follow-up of 1 year at the time of writing were selected (209 patients). All patients underwent surgery performed by a single surgeon (E.C.) using an anterior approach, most frequently a video-assisted TMD20–22 (179 patients [85.6%]), less frequently a minithoracotomy (27 patients [12.9%]), and rarely a transaxillary approach (2 patients [level T3–4, 1.0%]) or transsternal approach (1 patient [level T2–3, 0.5%]). The minithoracotomy technique was used for some so-called giant calcified TDHs19 (20 patients [9.5%]) and/or in case of previous posterior surgery at the involved level (6 patients [2.9%]), as both necessitated simultaneous anterior stabilization, as well as in 1 patient with a history of pulmonary lobectomy presenting with a symptomatic TDH on the same side.

We retrospectively identified 8 patients (3.8%) including 4 men and 4 women who presented with an acute myelopathy, defined as a variable degree of motor, sensory, and sphincter disturbances developing in less than 24 hours, and resulting in a Frankel Grade C or worse. We classified each patient’s neurological status according to the Frankel classification1 before surgery, at discharge from the hospital, and at follow-up examination 1 year postoperatively (Table 1).

Standard Neuroimaging Evaluation

Preoperative neuroimaging included MR imaging of the entire cervicothoracic spine to allow correct identification of the symptomatic level, and CT myelography or occasionally standard CT scanning to check for calcifications in disc herniation and intervertebral disc and to localize the involved level preoperatively.1 Postoperative neuroimaging included CT scanning with reconstructed images to assess stability before discharge, and MR imaging to assess accuracy of spinal cord decompression after 3 months. Moreover, we assessed the presence or absence of an intramedullary high signal intensity (myelomalacia) at the symptomatic level on axial and sagittal T2-weighted MR images obtained preoperatively as well as 3 months postoperatively.

Surgical Strategy in Patients Presenting With an Acute Myelopathy

All patients presenting with an acute myelopathy (8 patients) underwent surgery using standard TMD technique, 30° optics (Karl Storz GmbH & Co. KG), and a pneumatic arm holding the endoscope (Endoboy, Geyer S.A.); however, the patient in Case 7 initially underwent surgery performed by the attending neurosurgeon through a posterior approach. This patient’s condition deteriorated postoperatively, and she underwent reoperation via an anterior approach 4 days later. All patients underwent surgery during daytime hours, except for the patient in Case 2 who presented early in the series and underwent surgery overnight. This patient presented to our hospital directly where his clinical condition rapidly deteriorated to complete paraplegia, a T-12 sensory level, decreased anal sphincter tone, and priapism.

Adhering to a prospective study protocol initiated when we introduced the TMD technique in our department in 2000, all patients were carefully monitored perioperatively with MABPs close to preinduction values and above 70 mm Hg at all times. Moreover, all patients with significant cord compression and clinical and/or radiological myelopathy were treated with intravenous methylprednisolone according to the NASCIS-II protocol (National Acute Spinal Cord Injury Study)4 to protect the already compromised cord against iatrogenic trauma due to manipulation.

Results

Patient Characteristics and Clinical Presentation

Eight patients with an acute presentation (3.8%) included 4 men and 4 women with a mean age of 53 years (range 38–76 years), whereas 201 patients without an acute presentation included 82 men and 119 women with a mean age of 49.2 years (range 23–83 years). Preoperatively, moderate paraparesis was seen in Cases 4 and 5; severe monoparesis of the right leg was noted in Case 6; severe paraparesis was noted in Cases 1, 7, and 8; and complete paraplegia was noted in Cases 2 and 3. Moreover, patients in Cases 1, 2, 4, 7, and 8 demonstrated a thoracoabdominal sensory level corresponding to the TDH level; those in Cases 3 and 6 demonstrated a sensory level limited to the legs (the latter with a typical Brown-Séquard syndrome); and the patient in Case 5 had normal sensation with paresthesias limited to his right

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an acute presentation included 18 patients with a symptomatic TDH at T10–11, and 2 at T11–12, whereas 201 patients without an acute presentation included 2 patients with a symptomatic TDH at T9–10, 4 at T10–11, and 1 at T9–11, representing a total of 68 patients (33.8%). Moreover, 5 of 8 patients presenting with an acute myelopathy had at least 2 TDHs (Cases 2, 3, 5, 7, and 8) (Figs. 1–4).

Thus, all patients (100%) with an acute presentation had an acute neurological impairment. Moreover, 5 of 8 patients presenting with an acute presentation had preoperative CT myelography in patients undergoing elective surgery.5 Of these neurologically unstable patients did not receive intrathecal contrast to avoid intrathecal manipulation (Cases 2, 3, 4, 6, and 7). Interestingly, we observed a completely calcified TDH in Cases 1 (Fig. 1), 7, and 8 (Fig. 4), and a partially calcified TDH in Cases 3 and 5. On the other hand, we observed a single soft nucleus pulposus fragment surrounded by many harder, yet uncalcified, fragments in the patient in Case 4 (Fig. 3), a 49-year-old man who had suffered transient paraparesis 11 years earlier. Moreover, we observed a noncalcified TDH strongly adherent to the posterior longitudinal ligament in the patient in Case 2 (Fig. 2), and an enormous anular fragment that may have originated at T12–L1 (collapsed intervertebral disc space) and was sequestered and trapped in a T11–12 stenosis (normal disc space, hypertrophic facets and flaval ligaments) in Case 6 (Table 1).

As such, the symptomatic disc herniation clearly had a fresh aspect (that is, noncalcified, nonadherent to the posterior longitudinal ligament and dura) in only 1 (12.5%) of 8 patients presenting with an acute myelopathy (Case 6), and 2 (1%) of 201 patients presenting without an acute myelopathy. The patient who presented with acute myelopathy was a 38-year-old man in whom an enormous sequestered anular fragment was encountered during surgery. The patients who presented without an acute myelopathy were a 23-year-old woman suffering from mild myelopathy for several months and a 26-year-old woman suffering from axial pain after a car crash 7 months before surgery. Both disc herniations consisted of a single soft nucleus pulposus fragment.

Percentages of Canal Overcrowding and Segmental Stenosis. We assessed the percentage of spinal canal surface occupied by the disc herniation using preoperative CT scanning, CT myelography, and MR imaging. Ac-
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<table>
<thead>
<tr>
<th>Case No.</th>
<th>Clinical Presentation</th>
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<tr>
<td>1</td>
<td>see text</td>
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<td>2</td>
<td>46-yr-old M suddenly had severe pain in back, abdomen, &amp; rt leg while walking through the house. He fell on the floor, was unable to get up, &amp; was transferred to the ED, &amp; his condition rapidly deteriorated to paraplegia, priapism, decreased sphincter tone, &amp; T-12 sensory level (Frankel Grade B).</td>
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<tr>
<td>3</td>
<td>76-yr-old F suddenly had severe pain in lower thoracic spine, which quickly resolved, but reappeared the next day, accompanied by a rapidly progressive paraparesis. Within hrs, she was paraplegic, with some residual sensation in legs, &amp; UR (Frankel Grade B).</td>
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<tr>
<td>4</td>
<td>see text</td>
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<tr>
<td>5</td>
<td>47-yr-old M suffering from unexplained pain in thoracic spine for &gt;1 yr suddenly developed a mod paraparesis, paresthesias in both legs, UI, &amp; fecal incontinence (Frankel Grade C).</td>
</tr>
<tr>
<td>6</td>
<td>38-yr-old M suddenly developed an incomplete Brown-Séquard syndrome (severe motor loss &amp; hypesthesia in rt leg, &amp; loss of pain &amp; temperature sensation in lt leg) during a soccer game (Frankel Grade C).</td>
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<td>7</td>
<td>51-yr-old F suddenly had severe pain &amp; mod paresis in her legs, not in the back. Admitted into another center &amp; transferred the next day when her condition had deteriorated to severe paraparesis, thoracoabdominal sensory level, &amp; UR (Frankel Grade C).</td>
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<tr>
<td>8</td>
<td>62-yr-old F had hypesthesia in both legs shortly before going to bed. The next morning, she woke up with more severe paraparesis, thoracoabdominal sensory level, &amp; UR. By the time she arrived at the ED, she was paraplegic (Frankel Grade B).</td>
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* ED = emergency department; mod = moderate.

According to the scoring system proposed by Hott et al., a greater than 40% occupation would indicate a giant TDH, which was present in all cases except for Case 3 (Table 1). This scoring system, however, does not account for an eventual associated segmental stenosis, which in our experience is quite common especially in the lower thoracic segments. Such stenosis may aggravate the amount of cord compression, and in fact was present in 3 of 7 TDHs classified as giant TDHs (Cases 4, 6, and 7) (Fig. 3) as well as in Case 3 (Table 1).

Presence of Myelomalacia. We observed an intramedullary high signal intensity (indicating myelomalacia) on axial and sagittal T2-weighted MR images either at the level of the disc herniation (Cases 4, 5, and 6) (Fig. 3) or immediately above and/or below in cases of major cord compression, making assessment at the exact level impossible (Cases 2, 3, and 8) (Figs. 2 and 4). Thus, myelomalacia was present in all but 2 patients (Cases 1 and 7); however, the latter did show a small spot of intramedullary high signal intensity on postoperative MR imaging.

Surgical Timing, Outcome, and Complications

With regard to surgical timing, the patient in Case 2 is the only one who underwent surgery the day myelopathy started. Those in Cases 7 and 8 underwent surgery the next day; in Cases 3 and 5, 2 days later; in Case 1, 6 days later; and in Cases 4 and 6, the patients underwent surgery 9 days later, mainly because of delayed referral (the patient in Case 4 was actually repatriated from Costa Rica). The patient in Case 7 is unique as she underwent surgery performed 1 day after admission by the attending neurosurgeon through a posterior approach, which resulted in inadequate cord decompression. Her neurological condition deteriorated over the following 3 days and she underwent reoperation on the 4th day through an anterior thoracoscopic approach, which resulted in complete resection of a calcified, adherent disc herniation. Additional posterior stabilization was performed 17 days later. Notwithstanding this surgical delay, all patients had a favorable outcome, regaining continence and ambulation as reflected in their 12-month postoperative Frankel scores. Indeed, 4 patients improved from Grade C to E, 2 from Grade B to D, 1 from Grade C to D, and 1 from Grade B to E (Table 1). Seven patients showed significant improvement within a week, whereas 1 patient (Case 3) showed no improvement at all within 3 weeks postoperatively. This 76-year-old woman initially exhibited persisting paraplegia, then started to move her toes at 3 weeks and continued to gradually recover for approximately 1 year, achieving independent ambulation and full bowel and bladder control.

Complications in this series were as follows: intraoperative blood loss greater than 1000 ml (in 1 patient [Case 8]), CSF leak (in 1 [Case 7]), possible CSF leak (in 1 [Case 2]), reversible ischemic neurological deficit (in 1 [Case 1]), and subileus requiring prolonged medical treatment (in 1 patient [Case 3]). There were no infections. The CSF leaks were successfully treated with a combination of Spongostan (Ethicon BioSurgery, Johnson & Johnson Medical, Ltd.), an autologous fat graft obtained through one of the incisions, and Tisseel fibrin sealant (Baxter BioSurgery, Baxter Healthcare Corp.). Postoperatively, we avoided suction on the chest tube, and we ordered bedrest and an external lumbar drain during 5 days in the patient with a proven CSF leak. One patient awoke from surgery with a right hemiparesis. Although still young, she had a history of 50 smoking pack years, and fortunately recovered completely within the next few days. As such, there were no permanent complications.

Illustrative Cases

Case 1

Presentation and Examination. Getting out of bed, this healthy 55-year-old woman with chronic low-back pain and 2 previous lumbar disc operations suddenly lost strength in her right leg and fell on the floor. She experienced pain in the back and right groin, paresis of the right leg, and hypesthesia of both legs. After admission, she rapidly progressed to become paraplegic with bilat-
eral extensor plantar reflexes and incontinence. After bed rest and dexamethasone, by the next day strength had improved to 1–2/5 on the right and 3–4/5 on the left according to the American Spinal Injury Association classification. A T-8 sensory level with intact pain and temperature sense was noted, her knee jerks were subclonic, her ankle jerks were vivid, and her plantar reflexes were repeatedly normal. Computed tomography and MR imaging demonstrated a very large, calcified right mediolateral TDH at T9–10, causing cord compression and deviation to the left (Fig. 1).

**Operation.** Five days after admission, she was transferred to our hospital in unchanged neurological condition and underwent surgery the next day. We performed a right-sided TMD with piecemeal resection of the disc herniation, using an autologous iliac crest graft to bridge the vertebral defect. The patient awoke from surgery with a right hemiparesis. Although still young, she had a history of 50 smoking pack years, and was lucky to recover completely within the next few days.

**Postoperative Course.** At discharge 12 days postoperatively, the right leg scored 4/5, and the left leg scored 5/5. At 3 months, MR imaging confirmed adequate decompression without myelomalacia. At 6 months, clinical examination was nearly normal, including discrete hyperreflexia in the legs, normal gait, and continence. Her condition 4.5 years postoperatively is stable.

**Case 4**

**Presentation and Examination.** During snorkeling in Costa Rica, this 49-year-old man suddenly developed paraparesis and paresthesias in both legs but more pronounced on the right. Interestingly, 11 years earlier he had been treated conservatively at another institution for a transient paraparesis attributed to a TDH at T10–11. He was repatriated 5 days later, and his condition remained unchanged, including moderate paraparesis, bilateral hyperreflexia, extensor plantar reflexes, a T-11 sensory level, decreased vibration sense in both legs, and normal sphincter control with variable urinary retention.

Magnetic resonance imaging demonstrated a large, soft, left mediolateral TDH at T10–11, causing severe cord compression, as well as multiple smaller midthoracic disc herniations. The hyperintense signal of the severely flattened cord on axial T2-weighted images strongly suggested myelomalacia.
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Operation. The patient was transferred to our hospital and underwent surgery 9 days after symptom onset. We performed a left-sided TMD with careful resection of a fibrous disc herniation, which had perforated the posterior longitudinal ligament.

Postoperative Course. At discharge 9 days postoperatively, both legs scored at least 4/5; however, the patient’s gait was still unstable. After 6 weeks, he returned to work and was able to walk even longer distances without support, his right leg was still a little numb and difficult to control, and his urination was completely normal. At 3 months, MR imaging confirmed adequate decompression and residual myelomalacia (Fig. 3D and E). The patient has no residual deficit 1.5 years later, except for a vague numbness in his right leg.

Discussion

Particular Features of TDHs Presenting With Acute Myelopathy

When analyzing our personal series and case reports in the literature,6,12,13,15,17,24,28 it seems that TDHs presenting with an acute myelopathy share some particular radiological and clinical features.

Radiological Features

In our entire series, we observed the symptomatic disc herniation at a level between T9–10 and T11–12 in all patients presenting with an acute myelopathy as opposed to 33.8% of those presenting without an acute myelopathy (Table 1). Interestingly, the same is true for most patients reported on in the literature: 2 had a disc herniation at T9–10,15 1 at T10–11,17 1 at T11–12,3 and 1 at T12–L1.12 On the other hand, we found 2 reports of an acute myelopathy at T2–324 and T4–5.5 Thus, we conclude that lower TDHs (including at T12–L1) are more prone to present with an acute myelopathy. Moreover, we hypothesize that this may be related to a greater mobility of the spine, a more delicate vascularization of the cord, and/or the lack of a protective layer of peripheral white matter surrounding the fragile gray matter of the conus.

We observed a fresh-looking disc herniation in only 1 patient (a sequestered anular fragment) and an at least partially calcified disc herniation in 5 patients presenting with an acute myelopathy. The same is true for at least 3 cases in the literature: a combination of cartilaginous endplates and disc material,3 a calcified disc herniation,15 and an adherent disc herniation.17 Other authors reported a noncalcified sequestered anular fragment,24 multiple

![Fig. 3. Case 4. Sagittal (A and D) and axial (B and E) T2-weighted MR images and an MR myelogram (C). A: Image demonstrating a left mediolateral TDH with slight cranial extension at T10–11. Aggravated by some degree of segmental stenosis, the disc herniation causes severe cord compression and deviation to the right and posteriorly (A and B), resulting in myelomalacia (B) and a complete MR myelographic block (C). D and E: Sagittal (D) and axial (E) images obtained 3 months postoperatively, demonstrating adequate decompression and some residual myelomalacia (D).](image1)

![Fig. 4. Case 8. A–C: Sagittal (A) and axial (B) T2-weighted MR images demonstrating a large central TDH at T9–10, causing severe cord compression and myelomalacia. A CT myelogram (C) demonstrates that the disc herniation is largely calcified. D and E: Sagittal (D) and axial (E) T2-weighted MR images obtained at 3 months, demonstrating adequate decompression and no residual myelomalacia.](image2)
noncalcified nonsequestered disc herniations, an intradural disc herniation, and a disc herniation adherent to the dura. Thus, we conclude that TDHs may be present (and may even be calcified) long before they suddenly become symptomatic.

We observed a giant disc herniation according to the scoring system by Hott et al. in all patients presenting with an acute myelopathy except for the patient in Case 3. Again, the same is true for at least 3 cases in the literature reported by Bose, Chen et al., and Sasaki et al. Moreover, we observed myelomalacia in 6 of 8 patients: 3 at the level of the disc herniation and 3 immediately above and/or below. In another patient (Case 7), we observed a small spot of myelomalacia postoperatively that may have been missed on the preoperative MR image because of major cord compression. Finally, we observed an associated segmental stenosis in 4 patients (including the patient in Case 3 with a relatively small disc herniation) aggravating the amount of cord compression. Thus, we conclude that the relatively rare TDHs presenting with an acute myelopathy invariably cause important cord compression, which may be aggravated by an associated segmental stenosis. Moreover, they almost invariably cause local myelomalacia.

As in many patients in our entire series, we observed multiple (2 or more) TDHs in 5 of 8 patients presenting with an acute myelopathy; however, we could always identify the symptomatic disc herniation rather easily using clinical and radiological clues. Important radiological clues include TDH size, relative to the kyphotic curve (the cord is closer to the intervertebral disc at the apex of the curve), cord compression (flattening) best appreciated on axial T2-weighted images, associated segmental stenosis, and last but not least myelomalacia at the involved level (6 of 8 cases [Table 1]).

Clinical Features

The clinical presentation of a TDH-related acute myelopathy may be misleading, and therefore, diagnosis may be delayed until other causes (especially vascular) have been excluded. Often, by that time the patient’s neurological condition has deteriorated, and the clinical picture has become more complete. Interestingly, although Brennan et al. reported a case of paraparesis due to a small T4–5 TDH in an 11-year-old boy who suffered a minor trauma, a precipitating event or trauma is absent in most cases. On the other hand, more or less severe dorsalgia frequently precedes profound myelopathy. This sequence of events was observed in several case reports and as well as in 6 patients (75%) in the present series. In fact, dorsalgia was present in 2 patients (Cases 2 and 3) who were initially examined for a vascular cause, until their neurological condition had dramatically deteriorated and a severe thoracic myelopathy had become evident a few hours later. As such, dorsalgia typically had a sudden onset preceding myelopathy by several hours, except for one patient in whom dorsalgia and myelopathy developed suddenly and simultaneously, and another in whom dorsalgia had started approximately 1 year before acute neurological impairment.

Theoretically, the pathophysiological mechanism behind dorsalgia may be analogous to the one behind cervicalgia preceding acute paraplegia in case of a soft cervical disc herniation. Sudden stretching of the annular fibers and posterior longitudinal ligament may cause severe pain; however, this may not explain dorsalgia in cases of a calcified TDH (Cases 1, 3, 5, and 7) that must have been present for some time already. Nevertheless, sudden-onset dorsalgia is an important sign that may guide the clinician to an early diagnosis, before a more complete clinical picture develops. Indeed, 6 patients in our series had developed severe mono- or paraparesis (4 patients) or even paraplegia (2 patients), and only 1 patient had normal sphincter control by the time the TDH was diagnosed (Table 1).

Surgical Considerations in Case of Acute Myelopathy

Theoretically, the gold standard in cases of spinal cord compression is immediate decompression; however, the relevant time frame in TDH-related acute myelopathy is unclear. Our results show that remarkable recovery is possible even with profound neurological deficit and a delay of several days, provided that the spinal cord is adequately decompressed. Since the introduction of TMD in our department 10 years ago, it has been our policy to surgically treat every TDH with an acute myelopathy referred to our center, even in cases in which myelopathy was present for several days, and even in cases of paraplegia. In view of our encouraging results, we will continue this policy and strongly recommend that every single patient undergo surgical treatment.

Given these considerations, we are convinced that it is even more important to perform the operation correctly rather than urgently. Standard laminectomy entails a high risk of neurological deterioration (as illustrated in Case 7) and is no longer an accepted method of approaching these challenging lesions. In fact, its use even in an emergency situation can never be justified. Although alternative approaches that may be more familiar to most neurosurgeons (costotransversectomy, or a lateral extracavitary or transpedicular approach) may be used successfully, the anterior transthoracic approach has the advantage of reaching the disc herniation anterior to the already compromised cord, thus avoiding any manipulation of the cord except at the interface with the disc herniation. In experienced hands, TMD combines the advantage and versatility of an anterior approach with improved visualization and minimal postoperative discomfort.

Furthermore, we strongly advocate keeping the MABP close to preinduction values and at all times above 70 mm Hg as long as the spinal cord has not been adequately decompressed, avoiding aggravated hypoperfusion and additional ischemic cord damage during surgery. Recent studies recommend an even higher MABP above 90 mm Hg, including a study by Chi et al. who reported a personal series of severely myelopathic patients treated with a so-called mini-open transpedicular thoracic discectomy. In this regard, we would agree with Crock et al. who stated “the vascular factor may be the most neglected one in acute spinal cord injury in general.” Of note, we are continuously evaluating our practice and are currently reconsidering the use of intravenous methylprednisolone.
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Indeed, a beneficial effect with regard to surgical trauma has never been proven, and evidence of negative side effects is accumulating in recent literature, although not reflected in our own series. The issue of multiple TDHs and acute myelopathy has briefly been addressed in a case report by Chen et al.6 who stated, “Since the presentation of TDH is variable and difficult to correlate with imaging findings, decompression at all lesion levels in a patient with symptomatic multilevel TDHs may be necessary to achieve complete symptom relief and satisfactory results.” We disagree and believe it should be possible to locate the symptomatic level even in cases of acute presentation, considering marked tenderness over the involved level (often present although not obligatory), an eventual sensory level (5 of 8 patients demonstrated a thoracoabdominal sensory level corresponding to the level of the disc herniation), and important radiological clues as described above. As such, it should be possible to do a selective decompression in most cases, as we successfully did in all 5 cases presenting with multiple TDHs.

All patients regained continence and ambulation as reflected in their pre- and 12-month postoperative Frankel scores. Of note, early postoperative clinical appearance does not necessarily reflect final outcome, as even patients with persistent, and almost complete paraplegia early after surgery subsequently may improve considerably.6 According to these authors, the greatest neurological recovery commonly occurs within 6 weeks, but some patients may continue to improve for up to 2 years. Our data support these findings, as 6 patients showed significant improvement within 1 week, whereas 1 patient (Case 3) showed no improvement at all the first 3 weeks postoperatively, yet ultimately achieved independent ambulation and full bowel and bladder control. Thus, we conclude that good to excellent recovery is not infrequent and is possible even with profound neurology and a delay of several days, provided the spinal cord is adequately decompressed.

Pathophysiology of Acute Myelopathy

Very little is known about TDH pathophysiology in general,16 let alone in case of an acute myelopathy. Interestingly, in our entire series (250 patients), there does not appear to be a clear correlation between TDH dimensions, radiological myelomalacia, and clinical myelopathy (unpublished data). We hypothesize that the lack of correlation may be explained by mechanical and vascular factors interacting with each other over a variable amount of time and resulting in a more or less profound and rarely acute myelopathy. Many questions remain; however, we plan to elaborate on this issue in a future publication, trying to improve our understanding and ultimately our care for these patients.

Counseling the Asymptomatic Patient

As mentioned before, this study clearly shows that TDHs may be present (and may even be calcified) long before they suddenly become symptomatic, causing a rapidly progressive myelopathy. This raises some questions with regard to counseling the asymptomatic patient, especially in case of a large or giant disc herniation with significant cord compression and myelomalacia. At this point, however, we do not know the natural evolution in such cases, and realize that patients referred for surgical treatment are only a fraction of those actually having a thoracic disc herniation.26-27 On the other hand, there is little doubt that such patients are at risk, and therefore they should at least be carefully counseled. We suggest balancing factors such as age, comorbidity, and occupation with the perceived risks of surgery on an individual basis.

Conclusions

Approximately 4% of TDHs present with an acute myelopathy. They are often situated between T9–10 and T11–12, are large or giant, and are even calcified, indicating their presence long before they become symptomatic. They almost invariably cause significant cord compression, which may be aggravated by an associated segmental stenosis, and myelomalacia. The clinical presentation of a TDH-related acute myelopathy may be misleading, and diagnosis therefore may be delayed until other causes (especially vascular) have been excluded. Often, by that time the patient’s neurological condition has deteriorated and the clinical picture has become more complete. Interestingly, whereas a precipitating event or trauma is rarely present, more or less severe dorsalgia frequently precedes profound myelopathy and may help the clinician to make an early diagnosis.

Our results show that remarkable recovery is possible even with profound neurological deficit, a delay of several days, in the elderly, and in the presence of myelomalacia, provided that the spinal cord is adequately decompressed and intraoperative hypotension is strictly avoided. Standard laminectomy entails a high risk of neurological deterioration and is no longer acceptable. Although alternative approaches more familiar to most neurosurgeons may be used successfully, the anterior transthoracic approach has the advantage of reaching the disc herniation in front of the compromised spinal cord, avoiding any manipulation. In experienced hands, TMD combines the advantage and versatility of an anterior approach with minimal postoperative discomfort.

In conclusion, this study demonstrates that TDH-related acute myelopathy may have a favorable outcome when managed correctly. We strongly recommend that every single patient should undergo surgical treatment.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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