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Image-guided transthoracic transpedicular microdiscectomy for a giant thoracic disc herniation: patient series

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BACKGROUND This case series reports on five consecutive patients who underwent image-guided transpedicular transthoracic microdiscectomy. The authors retrospectively reviewed five patients who had undergone Stealth image-guided transpedicular transthoracic microdiscectomy between 2015 and 2021.

OBSERVATIONS Image guidance with O-arm verified critical anatomical landmarks in the setting of large central calcified and/or soft tissue disc prolapse. This allowed limited rib head resection, pedicle removal, and corpectomy to give adequate access and not require interbody fusion. The authors performed a partial posterior corpectomy anterior to the affected disc prolapse and microsurgical delivery of the affected disc anteriorly into the corpectomy cave away from the thecal sac. Electronic and radiographic records were analyzed at their initial presentation and at follow-up. The median patient age was 51 years (range, 44–56 years), with 80% of the patients being males. Four of five patients had significant improvement of their presenting clinical symptoms. One patient had a complicated postoperative recovery with a pneumothorax and subsequent bilateral pneumonia requiring intensive care. Another patient developed delayed postoperative worsening of paraparesis.

LESSONS The use of Stealth image guidance with O-arm for transthoracic microdiscectomy for complex calcified thoracic disc herniation is an effective operative technical adjunct to verify anatomical landmarks and limit the microsurgical procedure.

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KEYWORDS thoracic microdiscectomy; image guidance; giant thoracic disc herniation; delayed onset paraparesis

Thoracic disc herniation (TDH) has an incidence of 1 per 1,000,000 persons[1] and comprises 0.15% to 4% of surgical cases of disc herniation.[2,3] A significant proportion of TDHs are diagnosed incidentally due to increasing use of magnetic resonance imaging (MRI), where the herniation may be large yet asymptomatic and without neurological deficit.[4] TDH most commonly affects the intervertebral discs between T8 and L1,[5] and people between 30 and 50 years of age with equal prevalence in both sexes.[5] TDH manifests as back pain and/or intercostal neuralgia in 92% of cases. In a study of 168 patients, 60% had sensory and/or motor neurological deficits resulting from cord compression.[3,5] However, most cases do not present typically and may have progressively worsening signs such as an ataxic gait, a pyramidal tract syndrome in the lower limbs, and/or vesico-sphincter signs.[3,5] Patients with atypical symptoms frequently experience diagnostic delay.[5] Myelopathy and pain resulting from TDH depends on the vulnerability of the thoracic spinal cord to multiple factors. These include: a greater diameter of the spinal cord relative to the thoracic spinal canal that leaves less space surrounding the spinal cord, a compromised thoracic intramedullary vascularization known as the watershed zone,[5] the extent of potential intradural extension causing cord compression, the degree of thoracic kyphosis that bow-strings the cord against the disc, and the nature of the denticulate ligaments that reduce cord mobility.[4]

Disc herniation that occupies more than 40% of the spinal canal on MRI and computed tomography (CT) is labeled a giant TDH.[2] Giant TDHs are often calcified,[5] whereas ordinarily 42% of TDHs are calcified.[3] TDHs demonstrate intradural extension in 0.26% to 0.3% of cases, with this being 15% to 70% for giant calcified

ABBREVIATIONS CT = computed tomography; DPND = delayed postoperative neurological deficit; MRI = magnetic resonance imaging; PLL = posterior longitudinal ligament; TDH = thoracic disc herniation.

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TDHs,[5] reflecting the difficulty that surgeons face finding the correct extradural microdissection plane. Giant calcified TDHs cause myelopathy in 95% of affected patients,[5] and are associated with increased incidence of postoperative complications.[4] A history of trauma is present for 3% to 37% of people with TDH, and TDH is more common in patients with Scheuermann's disease.[5]

Many patients with TDH benefit from nonoperative management.[1] However, surgery is indicated when there is worsening myelopathy, persistent axial back pain, ongoing intercostal neuralgia, and intractable radicular pain.[1] Symptoms can develop slowly or rapidly, because of the vulnerability of the thoracic spinal cord to compression.[5]

Historically, the mainstay operative technique for relieving pressure caused by TDH was a thoracic laminectomy, but its operative complications led clinicians away from this method.[5] The best surgical approach is one that provides the desired amount of decompression while minimizing tissue distortion and damage to the vulnerable thoracic spinal cord.[4] There are three primary surgical approaches utilized now: a posterolateral transpedicular approach, a lateral approach (costotransversectomy or extended lateral extracavitary), and an anterior approach (transpleural thoracotomy, thoracoscopy and mini-thoracotomy with retropleural variation).[5] Calcified TDHs in the midline are approached anteriorly by performing a thoracotomy to allow adequate visualization of dural tears, whereas lateralized soft hernias may be treated with a posterolateral approach because they are less likely to be adherent to the dura.[5]

In our case series we present five cases of giant TDH that underwent a transthoracic discectomy utilizing Stealth image guidance with O-arm between 2015 and 2021. The patients underwent a thoracotomy, Stealth-assisted removal of the pedicle and an adequate partial corpectomy anterior to the affected disc, and microsurgical delivery of the disc anteriorly away from the thecal sac.

Study Description

Case 1

A 50-year-old male presented to his local emergency department with worsening sharp, localized pain in his lower thoracic spine, which was preceded by mild progressive lower limb numbness. Prior to surgery he had worsening myelopathy with reduced sensation bilaterally in the lower limbs, clonus, and a unilateral positive Babinski reflex but normal sphincter function. Preoperative CT and MRI showed a preoperative calcified disc prolapse at T9–10 with cord compression, and postoperative imaging showed decompression of the thecal sac (Fig. 1).

Case 2

A 53-year-old male had two episodes of severe sharp back pain located in the midthorax during heavy lifting, associated with leg weakness and altered sensation below the mid thorax. Prior to surgery he had bowel and bladder dysfunction, with sensory changes below the T10 dermatome. Preoperative CT and MRI demonstrated a focal soft disc prolapse at the T4–5 level which impinged the anterior surface of the spinal cord. Postoperative MRI showed decompression of the thecal sac (Fig. 2).

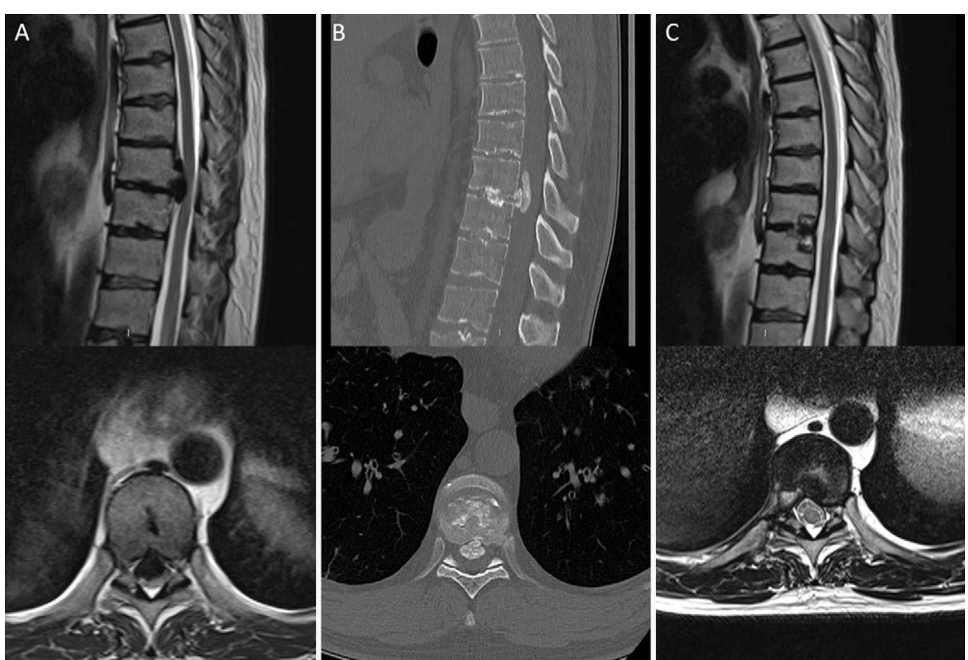

FIG. 1. Case 1. **A:** Preoperative T2-weighted MRI demonstrates the thoracic spinal cord being impinged at T9–10 with flattening of the cord in the sagittal (*upper*) and axial (*lower*) views. **B:** Preoperative sagittal (*upper*) and axial (*lower*) CT shows calcification of the posterior longitudinal ligament and disc prolapse extending into the spinal canal. **C:** Postoperative sagittal (*upper*) and axial (*lower*) T2-weighted MRI shows the limited removal of the rib head and neck, the limited corpectomy cave and decompression of the thecal sac and cord.

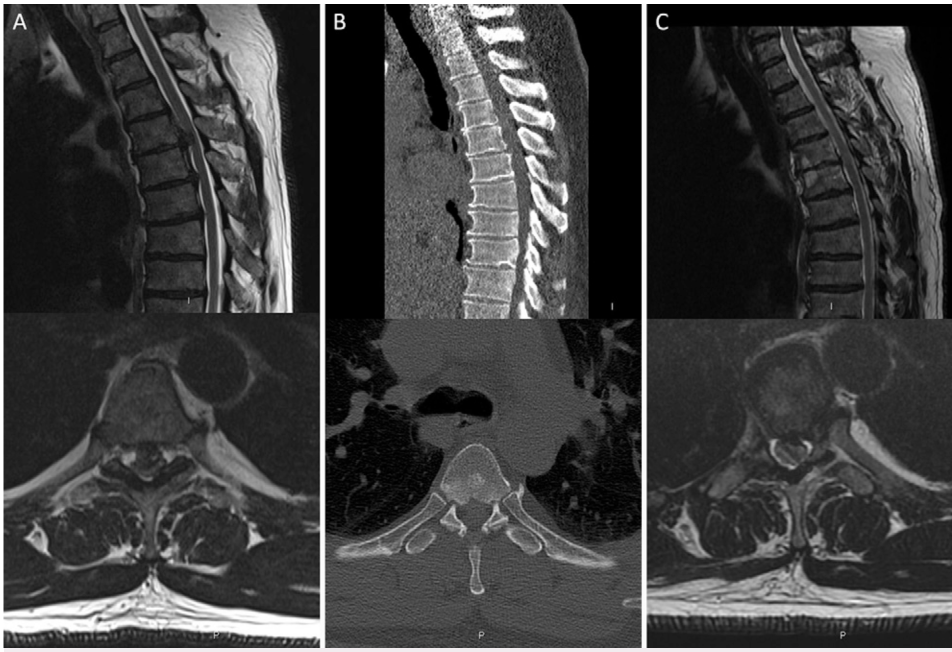

FIG. 2. Case 2. **A:** Preoperative sagittal (*upper*) and axial (*lower*) T2-weighted MRI shows the large central soft tissue disc prolapse causing flattening of the cord. **B:** Preoperative sagittal (*upper*) and axial (*lower*) CT shows the soft tissue prolapse without calcification. **C:** Postoperative sagittal (*upper*) and axial (*lower*) T2-weighted MRI shows decompression of the thecal sac and cord.

Case 3

A 56-year-old female presented with a 6-month history of progressive myelopathy with incoordination, mild spasticity, and decreased sensation in her lower limbs. Examination findings demonstrated mild bilateral weakness in hip flexion, intact lower limb sensation, lower limb hyperreflexia, a positive Babinski reflex, and mild spastic gait. Preoperative CT and MRI demonstrated T6–7 calcified disc prolapse with significant cord compression and cord signal change. Postoperative CT showed decompression of the spinal canal with a shell of calcification that was left in situ disconnected from the vertebra and posterior longitudinal ligament but attached to the dura (Fig. 3).

Case 4

A 44-year-old male presented with a 2-year history of unsteadiness, diffuse lower limb sensory loss, and urinary urgency with incontinence. On examination he had normal lower limb power, brisk patellar reflexes bilaterally, unilateral clonus but normal sensation and gait. Preoperative imaging with CT and MRI demonstrated a large, calcified disc sequestrum at the T6–7 level impinging the spinal cord. Postoperative CT showed the bony access with removal of the rib head and pedicle, with decompression of the spinal canal (Fig. 4).

Case 5

A 51-year-old male who presented with a 4-week history of unsteadiness, reduced lower limb power with altered sensation, and urinary and bowel dysfunction; he developed worsening lower limb power 48 hours before surgery. On examination he had moderate weakness of bilateral hip flexion, knee flexion and ankle dorsiflexion, lower limb hyperreflexia, and impaired light touch sensation in his feet. Preoperative CT and MRI demonstrated a large central calcified disc extrusion at T8–9 with cord compression. Postoperative CT and MRI showed decompression of the cord and no myelomalacia (Fig. 5).

Operative Technique

Patients were positioned in the left lateral position with the neck in neutral position. After skin preparation and draping, a small incision was made in the midline over the mid- to upper-level thoracic spinous processes for attachment of a reference frame. An O-arm CT spine was then performed and merged with the Stealth navigation system.

A posterolateral thoracotomy was performed adjacent to the level of the disc prolapse, verified by the Stealth image guidance. Once the lung was deflated, the parietal pleura was peeled away from the lateral vertebral bodies and costovertebral joints. Under microscope magnification the head and neck of the rib up to the facet and the pedicle were removed to expose the lateral epidural fat. With the disc identified and verified on Stealth image guidance, a partial corpectomy was performed in the adjacent vertebrae. This created a cavity above and below as well as lateral to the preserved disc prolapse, adequate to manipulate the dissecting instruments, which was verified with Stealth navigation. A shell of outer cortex of the posterior vertebral margin was left in situ anterior to the posterior longitudinal ligament (PLL) above and below the disc prolapse. This shell of bone was then dissected free of the PLL and removed, exposing the dural sac above and below the disc prolapse. The soft partially calcified disc material centrally within the prolapse where possible was then debulked using microdissectors and rongeurs. Where identifiable the PLL was separated to expose

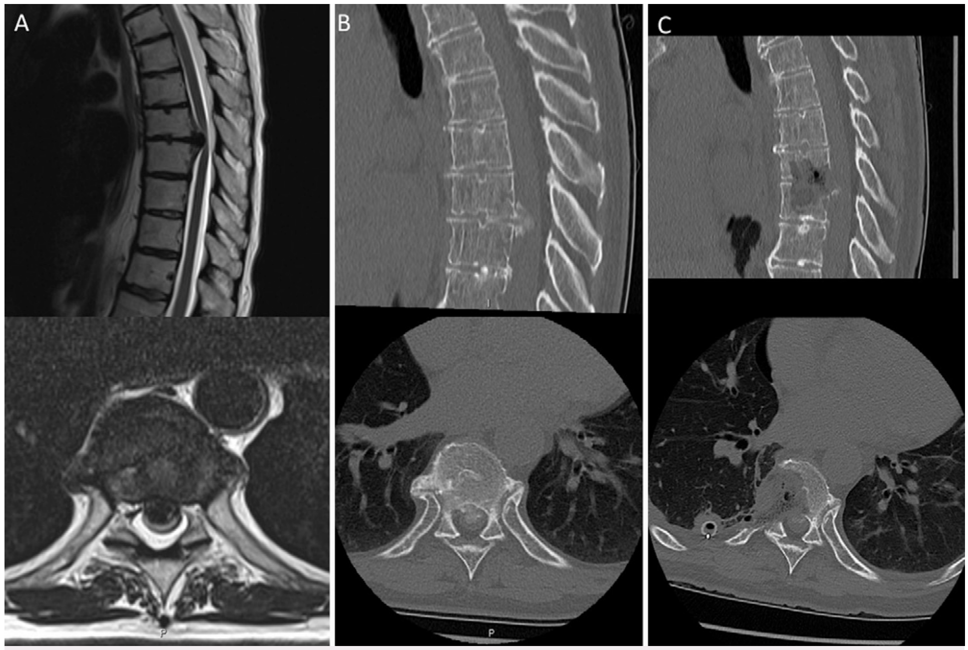

FIG. 3. Case 3. **A:** Preoperative sagittal (*upper*) and axial (*lower*) T2-weighted MRI shows a large central disc prolapse with marked flatting of the spinal cord. **B:** Preoperative sagittal (*upper*) and axial (*lower*) CT shows the calcified disc prolapse extending into the spinal canal and the calcification at the right costovertebral joint. **C:** Postoperative sagittal (*upper*) and axial (*lower*) CT shows removal of the head of the rib, pedicle and vertebral body on the right side; the shell of mobilized calcification at the posterior margin of the thecal sac can also be seen.

the dural sac and the herniated disc. The adherent calcified disc was mobilized anteriorly away from the dura while avoiding traction of the dura. This allowed delivery of the prolapsed disc en bloc or in fragments into the space created by the partial corpectomy, with decompression of the thecal sac. Care was taken to avoid excess use of bipolar diathermy of the epidural veins and adjacent soft tissue.

For case 1, a defect was noted in the midline of the dura, with egress of cerebrospinal fluid. As the dura at the opening was too friable for primary closure the repair was completed with layered DuraMatrix (Stryker) and Tisseel (Baxter) glue. With a chest drain and an intercostal catheter in situ the thoracotomy wound was closed in a standard method.

Outcomes

The postoperative recovery was complicated for case 4, who developed a significant pneumothorax, subcutaneous emphysema, and pneumonitis. After intensive care for respiratory support he responded well and was extubated and stepped down to the ward after 2 days.

The postoperative outcome was also complicated for case 5, as he progressively worsened 2 weeks after surgery with severe weakness, after initially remaining stable. Postoperative MRI and CT showed decompression of the thecal sac. Several postoperative MRIs over 3 months showed no myelomalacia or other changes in the cord or epidural space to explain the delayed postoperative deterioration. He was transferred to the spinal rehabilitation unit.

Case 1 was followed for 8 months. He had a normal gait, but was experiencing mild wound hypersensitivity, and hyperalgesia in the lower limbs and perineal region, with normal urinary and bowel function. Case 2 was followed for 8 months. He had a normal gait, and his sensory deficit improved from involving his entire lower limbs to just the soles of his feet, with no residual bowel/bladder dysfunction. Case 3 was followed for 16 months. His myelopathy improved significantly, and he was mobilizing independently without neurological deficit. Case 4 was followed for 6 months and so far, has no ongoing respiratory symptoms. His lower limb sensation had normalized, and he only experienced mild thoracic wound tenderness. Case 5 had documented follow-up for 21 months, remains with severe lower limb weakness, mobilizes in a wheelchair, and requires sphincter support.

Discussion

Surgery for giant TDH is technically demanding due to limited surgical access and proximity to the thoracic spinal cord. Potential complications include, symptom worsening with failure to treat, post operative neurological damage, pneumothorax, subarachnoid-pleural fistula, and a dural tear.[4] There is consensus that a transthoracic anterior approach is preferred for calcified midline TDHs.[6–12] Innocenzi et al.[13] report performing a posterior transpedicular approach for TDHs using a three-dimensional navigation system; image guidance has also been used in the thoracoscopic approach.[14]

Observations

In this case series we present five patients who had Stealth-assisted transthoracic microdiscectomy with O-arm in the setting of large central calcified and/or soft tissue disc prolapse and complications. Stealth navigation with O-arm was used to identify the correct

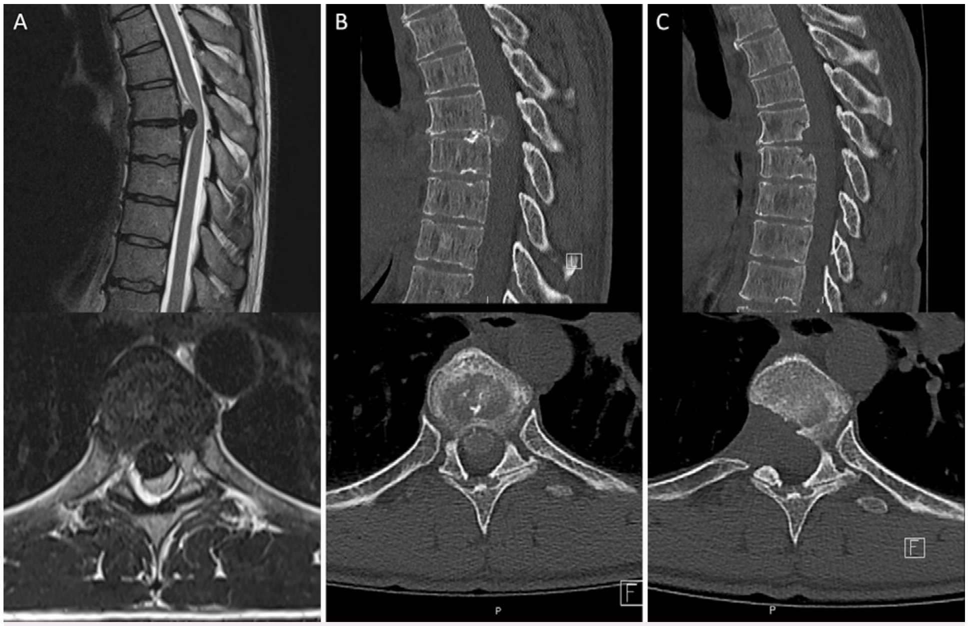

FIG. 4. Case 4. **A:** Preoperative sagittal (*upper*) and axial (*lower*) T2-weighted MRI shows the central disc prolapse with effacement of the thecal sac and moderate cord compression. **B:** Preoperative sagittal (*upper*) and axial (*lower*) CT shows calcification of the disc prolapse extending into the spinal canal. **C:** Postoperative sagittal (*upper*) and axial (*lower*) CT shows the partial corpectomy and bony access with decompression of the spinal canal.

spinal level and verify anatomical landmarks, which allowed for head of rib resection, pedicle removal, and limited corpectomy to give the necessary access and exposure for microsurgical delivery of the disc anteriorly away from the thecal sac into the corpectomy cave. In all cases, the limited exposure utilizing image guidance helped to avoid interbody fusion.

Despite the advent of minimally invasive techniques[15–18] and the use of image guidance,[14,19,20] further improvements to reduce the risk of complications and maximize accuracy in treating giant TDHs by identifying key anatomical structures are necessary. This is pertinent as the complication rate of thoracic discectomy remains at 20% to 30%.[4] In our case series, useful Stealth assisted technical nuances include verification of the limits of exposure and removal of the head and neck of the rib, the margins of the pedicle, the extent of the partial corpectomy just beyond the dimensions of the disc prolapse, and verification of the position of the neural foramen and thecal sac.

All patients except case 5 had significant improvement of their neurological symptoms at follow up. The postoperative outcome of delayed postoperative paraparesis for case five was unexpected as the operative procedure was uneventful. However, uneventful surgery with stable peri- and intraoperative vital signs and normal postoperative examination findings can still result in delayed postoperative neurological deficit (DPND).[21] The DPND for case 5 may have resulted from tissue destruction, ischemia, or a functional disorder. However, the dura was not injured with no apparent cerebrospinal fluid leak, the thecal sac was well decompressed without incident and several postoperative MRIs showed no myelomalacia or other cord abnormality. Also of note, during the procedure several epidural veins were cauterized using bipolar diathermy, but there was no imaging evidence of myelitis, infarction, or hematoma.

Complications after thoracic discectomy have improved significantly since the 1990s,[22,23] but more so since the advent of minimally invasive approaches for lateral prolapse.[4,24] Nevertheless, complications are encountered after approaches to central giant calcified thoracic disc prolapse with a vulnerable spinal cord.[25,26] DPND is reported after correction of severe kyphotic deformity,[27] but is not an ordinary consequence following minimally invasive thoracic discectomy[28] or thoracotomy for image guided microdiscectomy with normal postoperative imaging.

The research into DPND is sparse, and largely in the context of spinal deformity surgery. DPND is exceedingly rare, with an estimated incidence of 1 per 9,910 cases (0.01%) in patients undergoing spinal deformity surgery.[21] Despite this, 23% of surgeons surveyed had experienced at least one DPND in the previous 10 years.[21] Of 92 cases of DPND, the most common cause of injury included ischemic injury (38%) and cord compression (15%). A delayed ischemic spinal cord event may be attributable to postoperative hypotension, instrumentation-related compression, or an epidural hematoma.[21] Postoperatively, only 73% of patients with DPND achieve final neurological status by 6 months, demonstrating that some patients do not recover.[21]

Lessons

The etiology of DPND remains unknown and is likely to be multifactorial. A possible explanation for the outcome for case 5 is postoperative spinal cord edema or vascular spasm causing occlusion, leading to ischemia, but in this case the postoperative MRIs were normal. Auerbach et al.[21] highlight several ways to minimize the risk of DPND: (1) adequate perioperative volume repletion to maintain mean arterial pressures; (2) transfusion with packed red blood cells to maintain normal hemoglobin concentrations; (3) continuous

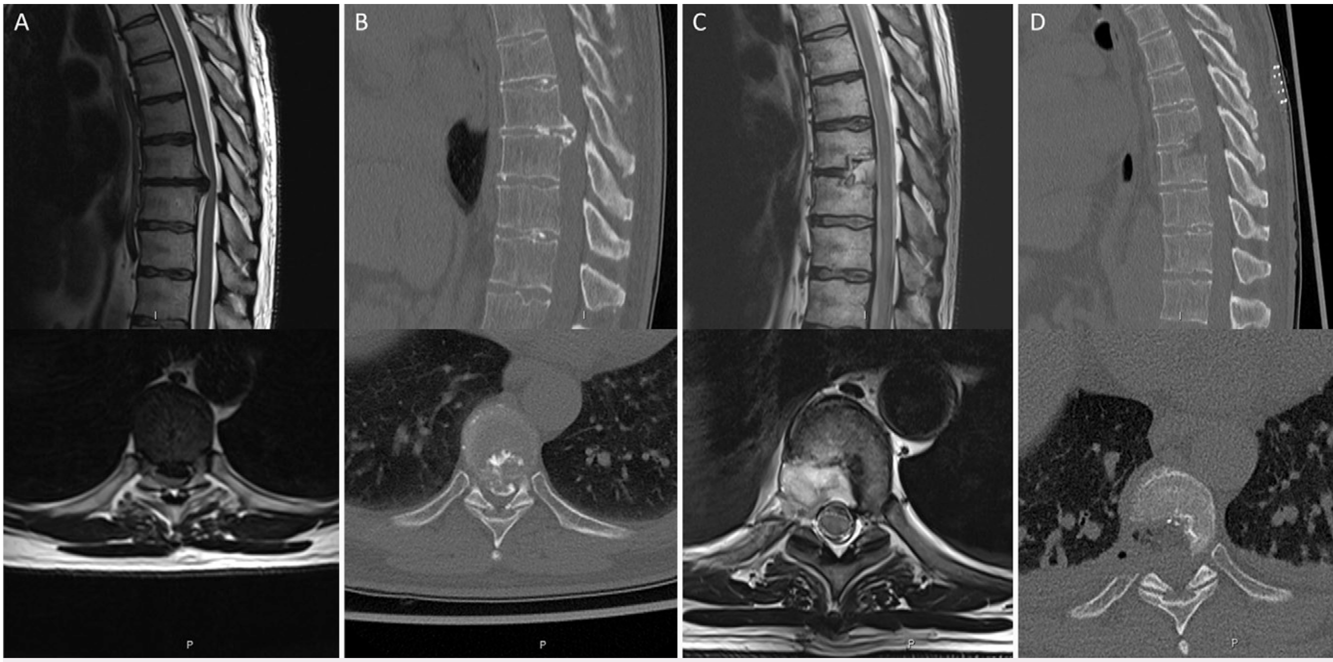

FIG. 5. Case 5. **A:** Preoperative sagittal (*upper*) and axial (*lower*) T2-weighted MRI shows the large central disc prolapse with severe cord compression. **B:** Preoperative sagittal (*upper*) and axial (*lower*) CT shows the calcification of the disc prolapse. **C:** Postoperative sagittal (*upper*) and axial (*lower*) T2-weighted MRI shows decompression of the cord and no T2-weighted MRI signal change within the cord. **D:** Postoperative sagittal (*upper*) and axial (*lower*) CT shows the bony access and decompression of the spinal canal.

arterial pressure monitoring to maintain arterial pressure between 80 and 90 mm Hg; (4) frequent neurological examination; and (5) minimization of patient-controlled analgesia to maximize cooperation with postoperative neurological examination.[29] Due to risk of bronchopulmonary injury, thoracoscopy is advised for small, noncalcified TDHs between T4 and T11 in those who have not previously had thoracic surgery.[30] However, its efficacy and safety are similar to open approaches, and its use has a steep learning curve.[28]

We report on this case series to highlight the use of Stealth image guidance with O-arm for transthoracic microdiscectomy for complex calcified TDHs as an effective operative technical adjunct to verify anatomical landmarks and limit the microsurgical procedure.

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Disclosures

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

Author Contributions

Conception and design: Wickremesekera, Koeck. Acquisition of data: Guy, Wickremesekera. Analysis and interpretation of data: all authors. Drafting the article: Kilmister, Guy, Wickremesekera. Critically revising the article: Kilmister, Wickremesekera, Koeck. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Kilmister. Administrative/technical/material support: Kilmister, Wickremesekera. Study supervision: Wickremesekera.

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