Thoracic gout tophus with abdominal wall protrusion
A case report

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Abstract
Rationale: A patient presented the abdominal wall protrusion due to tophaceous gout of the spine. Similar cases were not reported in the literature. This study aimed to report a case of tophaceous gout of the spine with abdominal wall protrusion.

Patient concerns: A 38-year-old male patient had a 10-year history of gout and hyperuricemia. He complained of back pain and abdominal wall protrusion.

Diagnoses: The patient was diagnosed with tophaceous gout of the spine with abdominal wall weakness caused by T11 nerve root compression.

Interventions: A semi-lamina decompression was performed at T11-T12. The pathological examination of the specimen demonstrated tophaceous gout of the spine.

Outcomes: After the surgery, the patient’s back pain was completely relieved and the abdominal wall weakness significant improved.

Lessons: This case highlighted that axial gout could mimic thoracic disk herniation clinically. The abdominal wall weakness might also be due to single T11 nerve compression by the tophaceous gout of the spine. In patients with a history of gout, axial gout should be considered as one of the differential diagnoses.

Abbreviations: CT = computed tomography, DECT = dual-energy CT, MRI = magnetic resonance imaging.

Keywords: abdominal wall weakness, axial gout, dual-energy computed tomography, intercostal nerve

1. Introduction

Gout is inflammatory arthritis associated with hyperuricemia induced by monosodium urate crystals.[1] The incidence of gout is estimated to be 0.2% to 0.4% worldwide with an annual incidence of 0.01% to 0.015%.[2] When the concentration of uric acid in the blood surpasses the physiological dissolution threshold in humans, the urate crystals deposit in the joints, synovial bursa, and subcutaneous tissue.[3] The mechanism underlying axial gout is unclear and requires a widely accepted large-scale epidemiological survey. However, axial gout with neurological symptoms is rarely reported.

Gout involves all the segments of the spine. Toprover et al reviewed 131 cases of axial gout, showing that gout could occur at any level of the spinal cord. The proportion of lumbar vertebras, cervical vertebrae, and thoracic spine was 38%, 24.8%, and 17.8%, respectively.[4] Moreover, axial gout could impact any anatomic components of the spine, such as facet joint, vertebral bodies, pedicle, lamina, and ligamentum flavum.[5] Patients had symptoms of spinal stenosis, lumbar radiculopathy, spondylolysis, cauda equine syndrome, or spinal infection.

Abdominal wall weakness is rare in the clinic and also confused with abdominal wall hernia. It is caused by the looseness, weakness, or even defect of the myofascial tissue on the abdominal wall, leading to the leakage of the contents of the abdominal cavity. Abdominal wall weakness has been reported to result from intercostal nerve injury caused by surgical procedures or herpes virus infections.[6–8] However, abdominal wall weakness caused by the tophaceous gout of the spine has not yet been reported.

This study reported a rare case of tophaceous gout of the spine with abdominal wall weakness.

2. Case description

A 38-year-old Chinese male patient had a 2-month history of back and intercostal pain. Following pain for 1 month, a
protrusion was detected in the lower left abdomen of the patient when standing (Fig. 1), which disappeared in the lateral decubitus position. The patient was once misdiagnosed with abdominal wall hernia. However, computed tomography (CT) and abdominal color ultrasound did not support this diagnosis. The examination results showed that the bilateral abdominal wall muscle was continuous without breakage. The patient visited our hospital for a distinct diagnosis and further treatment. The medical records revealed that the patient had a 10-year history of gout and hyperuricemia but did not undergo standard and systemic gout treatment. Tophaceous deposits were present in the hands and toes for at least 5 years. Furthermore, the patient had high alcohol intake for 15 years. When he suffered gouty attacks, clindamycin and dexamethasone were given to relieve pain.

The physical examination exhibited a mild pain to percussion on T11–T12 spinous processes, but radiating pain was not evident. The numbness was experienced in the left T11–T12 intercostal area and the related lateral abdominal wall. Neurological examinations revealed normal sensory and motor functions of the lower limbs. Patients had no abnormal reflexes, and pathological reflexes were negative in both legs. Also, a decreased left lower abdominal reflex was found. Bilateral cremasteric reflexes were normal. The patient’s largest abdominal circumference across the core of the protrusion was 110 cm. The abdominal wall protrusion range was 12 cm (longitudinal diameter) × 20 cm (latitudinal diameter). The laboratory values were as follows:

- Uric acid, 701 μmol/L
- Erythrocyte sedimentation rate, 14 mm/1 h
- C-reactive protein, 11 mg/L

The CT of the thoracic spine revealed disk herniation and spinal stenosis at T11/T12 levels. The sagittal and axial planes of thoracic CT showed a high-density shadow surrounding the herniated disk (Fig. 2), which was considered as disk calcification on the magnetic resonance imaging (MRI) of the thoracic spine. The disks of T11/T12 showed posterior abdominal wall weakness. Furthermore, thoracic disk degeneration was evident at the level of T9/T10, T10/T11, and T11/T12.

MRI revealed abnormal hyperintensity in the disk on the axial plane (Fig. 2). In addition, the abdominal color Doppler ultrasound in the standing position showed that the left anterolateral abdominal wall was thinner than the right anterolateral abdominal wall (Fig. 3). The patient was diagnosed with thoracic disk herniation with abdominal wall weakness based on the symptoms and imaging examination. Posterior thoracic decompression, discectomy, and internal fixation were performed to achieve neurological decompression, improve abdominal wall weakness, and relieve back and intercostal pain.

Figure 2. Preoperative CT and MRI images: (A) Sagittal plane showed calcification surrounding the herniated disk at T11/T12 levels. (B) Positioning phase. (C and D) Axial plane showed disk herniation and spinal stenosis at T11/T12 levels. (E) T1-weighted image shows the lesions at T11/T12 levels. (F) T2-weighted image shows the lesions at T11/T12 levels. (G) T2 fat suppression images show the lesions at T11/T12 levels. (H and I) Axial images show the lesion is closer to spinal cord and enters the nerve root canal.
During the surgery, the left articular process and lamina were removed. A mass with a complete capsule was found on exploring the suspected thoracic disk herniation. After the capsule was opened, a white, silt-like, and granular crystal was observed, removed, and prepared for the pathological examination. Combined with the history of gout, the original diagnosis of thoracic disk herniation was negated and an intraoperative diagnosis of the tophaceous gout of the spine was made. Then, the left nerve root canal was decompressed, and the dorsal and ventral sides of the spinal cord were explored. Discectomy was not conducted because disk herniation was not found. Furthermore, due to the tight adhesion between the frontal mass and the dura mater, the complete resection of the mass capsule was aborted after the effective decompression of the left nerve root, to prevent spinal cord injury, dura tear, and cerebrospinal fluid leakage. Consequently, a tiny residue of the mass capsule was left on the ventral side of the spinal cord. However, decompression of the spinal cord and spinal nerve was achieved after the resection. A pedicle screw system was applied to prevent segmental instability. A pathological examination showed that the deposits of monosodium urate crystals were surrounded by multinucleated giant cells and monocytes. The pathological examination is consistent with our intraoperative diagnosis. According to pathological result, symptoms, laboratory value and imaging data, the final diagnosis was tophaceous gout of thoracic spine.

After the surgery, his back pain and numbness were significantly and immediately relieved. The patient had a normal gait 3 days after the surgery. However, the left abdominal wall protrusion in the standing position did not disappear immediately. The back pain and numbness disappeared during the first follow-up 3 months after the surgery. The range of the abdominal wall protrusion reduced to 12 cm (longitudinal diameter) × 18 cm (latitudinal diameter). During the second follow-up 6 months after the surgery, the patient underwent CT and MRI. MRI did not show any compression around the spinal cord and nerve root (Fig. 4). The thoracic CT also revealed that the calcification surrounding the tophaceous gout was removed, and no high-density shadow was detected in the spinal canal (Fig. 4). The range of the abdominal wall protrusion reduced to 11 cm (longitudinal diameter) × 18 cm (latitudinal diameter). Additionally, the abdominal circumference of the patient reduced to 106 cm. The abdominal color Doppler ultrasound showed that the left anterolateral abdominal wall was thicker than the preoperative thickness by 2 mm.

3. Discussion
This study reported a rare case of axial gout with tophaceous deposits in the thoracic spinal canal. The patient had severe pain and left abdominal wall thickness. A total of 23 cases of the tophaceous gout of the spine have been reported to date (Table 1).[2,9–30] The clinical manifestations were not consistent; the decrease in muscle power and positive pathological signs were the most common characteristics (Table 1). However, the tophaceous gout of the spine with abdominal wall weakness was not reported previously.

Typically, the ventral rami of the inferior six thoracic nerves (T7–T12)—also known as the intercostal or thoracoabdominal nerves—contribute to the innervation of all muscles of the anterolateral abdominal wall to varying degrees.[31] Fahim et al performed 32 cadaveric dissections to isolate the individual muscle layers and nerve supply. This anatomical study revealed a limited insertion of the T9 and T10 nerves into the anterolateral abdominal muscles. The most significant intercostal nerve contributions to this muscle were from the T11/T12 segments.[31] Intriguingly, these anatomical studies might explain the cause of abdominal wall weakness in the present case; the T11 thoracic nerve root was compressed by tophaceous gout. The weakness was primarily located in the anterolateral abdominal wall between the umbilicus and the inguinal ligament, which is...
mainly in T11 dermatome (Fig. 1). The number of patients diagnosed with the tophaceous gout of the spine is extremely low. The present case involved the T11/T12 segment, and the clinical manifestations were associated with spinal cord compression without T11 or T12 thoracic nerve root compression. This could explain why the tophaceous gout of the spine with abdominal wall weakness has not yet been reported.

Interestingly, the abdominal wall weakness is reported as one of the postoperative complications after abdominal surgery.[32] Typically, the iatrogenic intercostal nerve injury is the most common cause of abdominal wall weakness.[6–8] However, single nerve root compression is not considered as the cause of abdominal wall weakness because it results from the compression of a single thoracic nerve root, which has not been reported previously. However, the present case showed that single thoracic root compression resulted in abdominal wall weakness. Therefore, single T11 thoracic root compression should be listed as one of the putative causes of abdominal wall weakness.

In the present case, the neurological symptoms caused by T11 nerve root compression included no spinal cord compression. Therefore, hemilaminectomy was performed, and the nerve root was fully decompressed. This alleviated the pain, and the abdominal wall weakness improved significantly. Early and adequate decompression might have benefited neurological recovery. It also revealed that the anterolateral abdominal wall recovered after decompression.

The present case showed that the tophaceous gout of the spine appeared as homogeneous areas of intermediate-to-low signal intensity on T1-weighted images. Homogeneous hypointensity was found on T2-weighted images. This hypointensity might be due to immobile protons in tophaceous gout, such as regions of calcifications, mature fibrous tissue, or hemosiderin deposition.[2] The tophaceous gout is similar to a degenerated disk on T2-weighted images. Thus, MRI failed to differentiate tophaceous gout from thoracic disk herniation. However, the tophaceous gout of the spine can also appear as areas of hyperintensity on T2-weighted images, as reported previously.[2] The commonly used imaging modalities can help in the diagnosis of axial gout, but they are not specific for detecting the deposition of urate crystals.[33] Dual-energy CT (DECT) emerges as a promising, sensitive, and specific imaging modality for the identification and volumetric quantification of tophaceous gout.[33] The identification of urate deposition using DECT has already been included as one of the diagnostic criteria of gout.[34] Nonetheless, most of the published studies on DECT are based on patients with axial gout in the peripheral joints.

However, the mechanism underlying axial gout is unclear and requires a widely accepted large-scale epidemiological survey. Konatalapalli et al performed a cross-sectional study on axial gout, in which 17/48 (35%) participants had the CT evidence of spinal gout and 7/48 (15%) had spinal tophi.[35] However, the exact incidence of tophaceous gout of the spine might be much
| Author               | Publication year | Country | Sex | Age (year) | Gout history | Neurological symptoms | Clinical description of gout | Serum uric acid level (mmol/L) | Level of involvement | Location of involvement | Method of diagnosis | Treatment | Evaluation |
|----------------------|------------------|---------|-----|------------|--------------|-----------------------|-----------------------------|-------------------------------|------------------------|----------------------|----------------------|------------|------------|
| Koskoff et al [10]   | 1953             | USA     | Male | 44         | 12           | Bilateral leg paralysis; back pain; bilateral lower extremity weakness | Severe, polyarticular tophi | 625                           | T9–T11                  | Extradural space      | Operation           | Improved   |
| Levin et al [11]     | 1956             | USA     | Male | 56         | 35           | Normal               | Severe, polyarticular tophi | 774                           | T12–L1                 | Disk vertebral bodies | Autopsy             | ND         | NA         |
| Leaney et al [2]     | 1983             | Australia | Male | 33         | 5             | Multifaceted; no bilateral lower extremity weakness; urinary retention | Severe, polyarticular tophi | 560                           | T7–T11                  | Extradural space; pedicles | Operation           | Laminectomy | Improved   |
| Downey et al [20]    | 1987             | UK      | Male | 73         | NR           | Bilateral leg paralysis; gait disturbance | NR                           | NR                           | T1                      | Extradural space; pedicles | Operation           | NR         | NR         |
| Yasuhara et al [13]  | 1994             | Japan   | Male | 60         | 5             | Back pain; hypesthesia; bilateral lower extremity weakness | Mild, polyarticular tophi   | 619                           | T6–T7                  | Extradural space       | Operation           | Laminectomy | Improved   |
| Dhote et al [14]     | 1997             | France  | Male | 56         | 2             | Bilateral lower extremity paralysis and weakness | Severe, polyarticular tophi | 929                           | T4–T9                  | Extradural space       | Operation           | Laminectomy | Improved   |
| Pfister et al [15]   | 1998             | USA     | Female | 53        | 25            | Back pain; urinary retention; unilateral right leg weakness | Severe, polyarticular tophi | NR                           | T8–T9                  | Vertebral bodies       | Needle biopsy       | Laminectomy | Improved   |
| Bret et al [12]      | 1999             | France  | Female | 59        | 16            | Bilateral lower extremity paralysis and weakness | Severe, polyarticular tophi | 340                           | T2–T3                  | Extradural space       | Operation           | Laminectomy | Improved   |
| Hausch et al [14]    | 1999             | USA     | Male | 59         | 3             | Back pain | Polyarticular tophi | 726                           | T4; T7                  | Vertebral bodies       | Needle biopsy       | Conservative | Improved   |
| Kaye et al [21]      | 1999             | South Africa | Female | 59        | 4             | Bowel and bladder dysfunction; bilateral lower extremity weakness | Polyarticular, no tophi | NA                           | T8                      | Vertebral bodies       | Operation           | Laminectomy | NR         |
| Kao et al [18]       | 2000             | Taiwan  | Male | 82         | 5             | Bilateral lower extremity weakness | Polyarticular, no tophi | 506                           | T10–T11                | Extradural space       | Operation           | Laminectomy | Improved   |
| St George et al [24] | 2001             | UK      | Male | 60         | 10            | Unilateral left leg weakness | No tophi | NR                           | T1–T2                  | Extradural space       | Operation           | Laminectomy | Improved   |
| Wang et al [20]      | 2001             | Taiwan  | Male | 28         | 5             | Back pain; bilateral lower extremity paralysis and weakness | Polyarticular, no tophi | 601                           | T9–T10                 | Facet joint; ligamentum flavum | Operation           | Laminectomy | Resolved   |
| Hsu et al [19]       | 2002             | Taiwan  | Male | 83         | 2             | Bilateral lower extremity weakness and numbness | No tophi | 375                           | T9–T11                 | Ligamentum flavum      | Operation           | Laminectomy | NR         |
| Souza et al [23]     | 2002             | Brazil  | Male | 49         | 5             | Back pain; unilateral right leg weakness; sensory impairment | Severe, polyarticular tophi | NR                           | T9–T10                 | Extradural space       | Operation           | Laminectomy | Resolved   |
| El Sandik et al [21] | 2004             | USA     | Female | 32        | NA            | Back pain | No tophi | 620                           | T7–T9                  | Extradural space       | Operation           | Laminectomy | Improved   |
| Popovich et al [20]  | 2006             | USA     | Female | 36        | NA            | Bilateral lower extremity weakness; sensory impairment | No tophi | 571                           | T4–T7                  | Pedicles               | Operation           | Laminectomy | Resolved   |
| Chan et al [16]      | 2009             | Hong Kong | Male | 76        | NA            | Bilateral lower extremity weakness | Severe, polyarticular tophi | NR                           | T8; T10                | Pedicles; vertebral bodies | Needle biopsy       | Conservative | Improved   |
| Kwan et al [8]       | 2013             | Canada  | Male | 25         | NR            | Back pain | No tophi | 462                           | T9; T10; T12            | Facet joint; ligamentum flavum | Needle biopsy       | Conservative | Decreased |
| Nasser et al [8]     | 2013             | USA     | Male | 30         | NR            | Back pain; bilateral lower extremity weakness | No tophi | NR                           | T10–T11               | Pedicles               | Operation           | Laminectomy | Improved   |
| Yoon et al [9]       | 2013             | Korea   | Male | 64         | 8             | Back pain; bilateral lower extremity weakness | No tophi | 726                           | T6–T7                  | Facet joint; pedicles | Operation           | Laminectomy | Improved   |
| Zheng et al [9]      | 2015             | China   | Male | 54         | 12            | Progressive back pain; unilateral lower extremity weakness | Severe, polyarticular tophi | 320                           | T3–T4                  | Ligamentum flavum      | Operation           | Laminectomy | Improved   |
| Arevalo-Saenz et al [8] | 2017             | Spain   | Female | 52        | NR            | Back pain | No tophi | NR                           | T10–T11               | Extradural space       | Operation           | Laminectomy | Improved   |

F = female, M = male, NA = not applicable, ND = not done, NR = not reported.
higher than that detected. Also, axial gout should be a crucial differential diagnosis based on the gout history and neurological symptoms of patients. DECT may be recommended besides routine spinal MRI for patients suspected of having axial gout. A limitation of this study was the relatively short follow-up time, which should be increased to achieve better outcomes regarding the patient’s nerve and muscle function.

4. Conclusion

This case highlighted that axial gout could mimic thoracic disk herniation clinically. The abdominal wall weakness might also be due to single T11 nerve compression by the tophaceous gout of herniation clinically. The abdominal wall weakness might also be considered as one of the differential diagnoses.

Author contributions

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