

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 vertebrae, 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, spondylolisthesis, 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

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ZW and YK contributed equally to this work.

Before the commencement of the study, the approval of the Ethics Committee of first hospital of Jilin University was sought, and written informed consent was obtained from the patient.

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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.

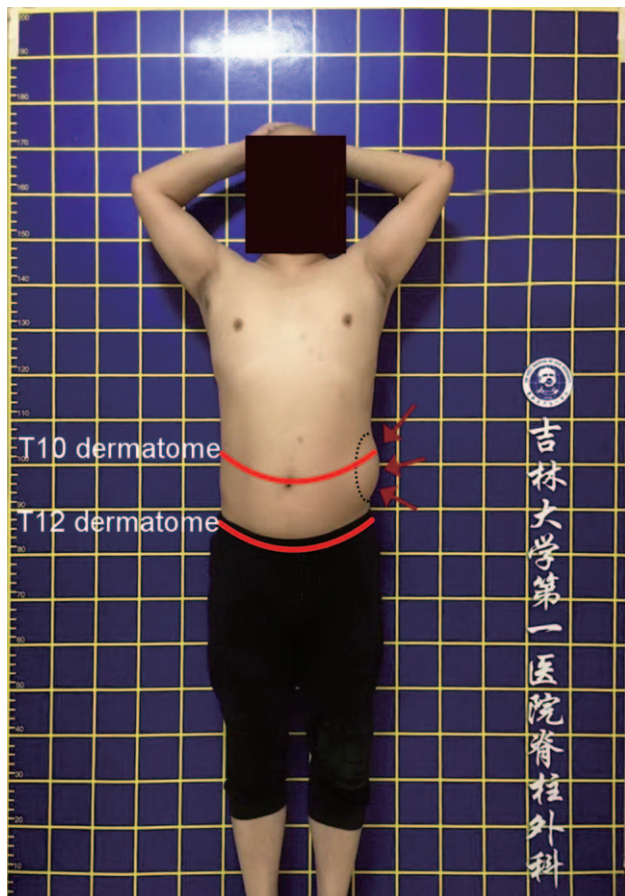


Figure 1. Abdominal wall protrusion appeared between T10 dermatome and T12 dermatome in standing position.

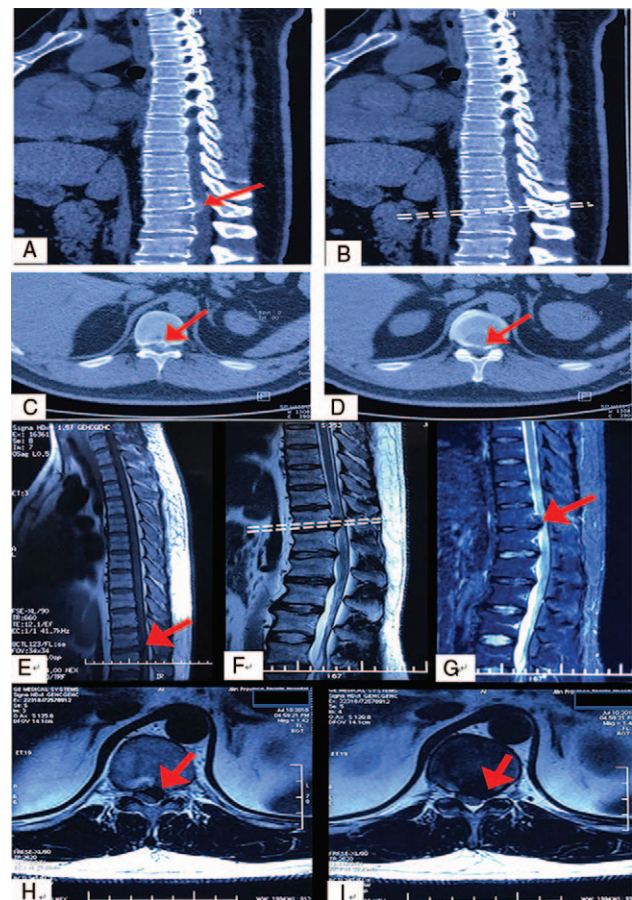


Figure 2. Preoperative CT and MRI images: (A) Sagittal plane showed calcification surrounding the herniated disk at T11/T12 levels. (B) Positioning phase. (C and D) Axial plane showed disk herniation and spinal stenosis at T11/T12 levels. (E) T1-weighted image shows the lesions at T11/T12 levels. (F) T2-weighted image shows the lesions at T11/T12 levels. (G) T2 fat suppression images shows the lesions at T11/T12 levels. (H and I) Axial images show the lesion is close to spinal cord and enters the nerve root canal.

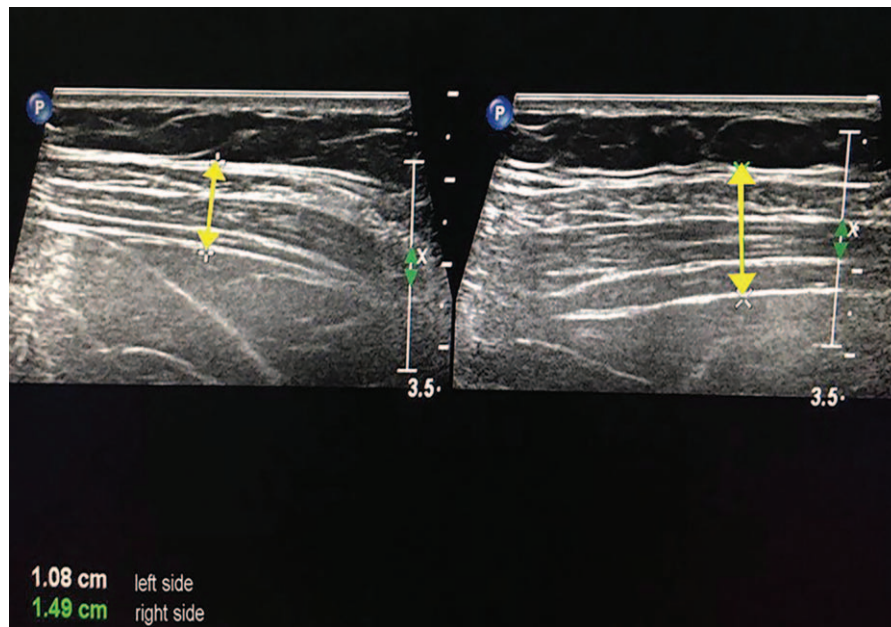


Figure 3. Preoperative color Doppler ultrasound: The image showed that the left anterolateral abdominal wall was thinner than the right anterolateral abdominal wall.

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) \times 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) \times 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 segment.^[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

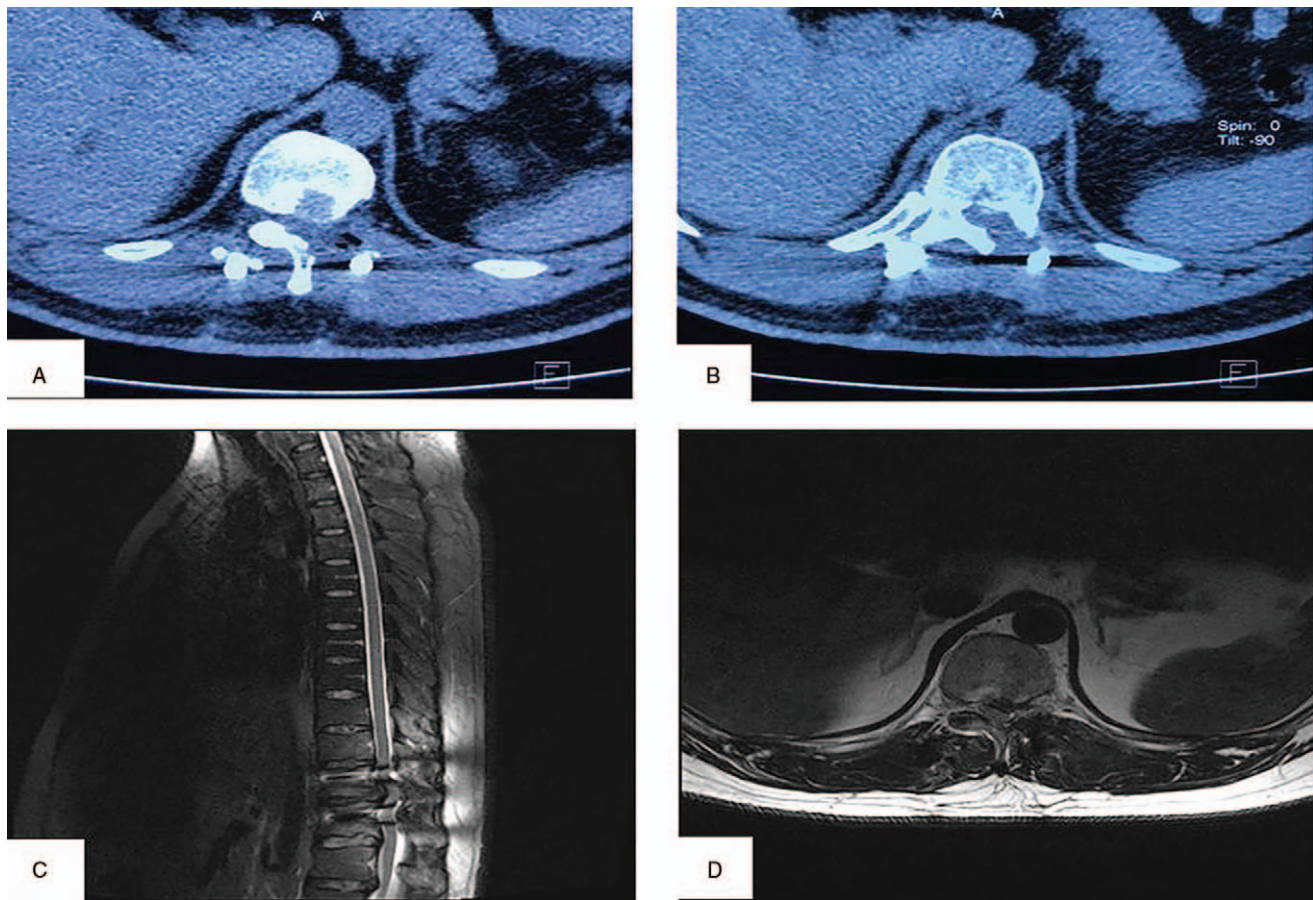


Figure 4. Postoperative CT images and MRI images: (A and B) Postoperative CT images showed adequate semi-lamina decompression at T11/T12 levels. (C and D) Postoperative MRI images showed adequate semi-lamina decompression at T11/T12 levels.

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 relieved 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

Table 1

Demographic and clinical characteristics of patients with axial gout.

Author	Publication year	Country	Sex	Age (year)	Gout history	Neurological symptoms	Clinical description of gout	Serum uric acid level (μmol/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	Decompression	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 ^[12]	1983	Australia	Male	33	5	Midthoracic pain; bilateral lower limb paralysis and weakness; urinary retention	Severe, polyarticular tophi	560	T7–T11	Extradural space; pedicles	Operation	Laminectomy	Improved
Downey et al ^[28]	1987	UK	Male	73	NR	Bilateral leg paralysis; gait disturbance	NR	NR	T1	Extradural space	Operation	NR	NR
Yasuhara et al ^[3]	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 ^[17]	1999	France	Female	59	16	Bilateral lower extremity paralysis and weakness	Severe, polyarticular tophi	340	T2–T9	Extradural space	Operation	Laminectomy	Improved
Hausch et al ^[30]	1999	USA	Male	59	3	Back pain	Polyarticular tophi	726	T4; T7	Vertebral bodies	Needle biopsy	Conservative	Improved
Kaye et al ^[18]	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 ^[16]	2000	Taiwan	Male	82	5	Bilateral lower extremity weakness; urinary retention	Polyarticular, no tophi	506	T10–T11	Extradural space	Operation	Laminectomy	Improved
St George et al ^[19]	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; urinary retention	Polyarticular, no tophi	601	T9–T10	Facet joint; ligamentum flavum	Operation	Laminectomy	Resolved
Hsu et al ^[21]	2002	Taiwan	Male	83	2	Bilateral lower extremity weakness and numbness	No tophi	375	T9–T11	Ligamentum flavum	Operation	Laminectomy	NR
Souza et al ^[22]	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 Sandif et al ^[23]	2004	USA	Male	32	NA	Back pain	No tophi	620	T7–T9	Extradural space	Operation	Laminectomy	Improved
Popovich et al ^[23]	2006	USA	Female	36	NA	Bilateral lower extremity weakness; sensory impairment	No tophi	571	T4–T7	Pedicles	Operation	Laminectomy	Resolved
Chan et al ^[29]	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 ^[24]	2013	Canada	Male	25	NR	Back pain	No tophi	462	T9; T10; T12	Costovertebral joint	Needle biopsy	Conservative	Decreased
Nasser et al ^[25]	2013	USA	Male	30	NR	Back pain; bilateral lower extremity weakness	NR	NR	T10–T11	Facet joint	NR	NR	NR
Yoon et al ^[27]	2013	Korea	Male	64	8	Back pain; bilateral lower extremity weakness	NR	726	T6–T7	Facet joint; pedicles	Operation	Laminectomy	Improved
Zheng et al ^[2]	2015	China	Male	54	12	Progressive back pain; unilateral lower extremity weakness	Severe, polyarticular tophi	320	T3–T4	Ligamentum flavum	Operation	Laminectomy	Improved
Arealo-Saenz et al ^[8]	2017	Spain	Female	52	NR	NR	NR	NR	T10–T11	Extradural space	Operation	Hemilaminectomy and spinal decompression	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 the spine. In patients with a history of gout, axial gout should be considered as one of the differential diagnoses.

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