

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

Spinal gout diagnosed by dual-energy CT: A case report ^{☆,☆☆}

Pavan Brahmhatt, MD^{*}, Prasanna Vibhute, MD, Vivek Gupta, MD, John Murray, MD, Amit Desai, MD, Amit Agarwal, MD

Mayo Clinic, Florida, 4500 San Pablo Rd, Mayo Clinic, Jacksonville, FL 32224, USA

ARTICLE INFO

Article history:

Received 27 July 2022

Revised 4 August 2022

Accepted 8 August 2022

Keywords:

Dual-energy computed tomography

Spinal gout

Axial gout

Gout

CT

ABSTRACT

Axial gout is an atypical presentation of gout caused by monosodium urate deposition in the axial skeleton. Spinal gout presents nonspecifically and can be a difficult diagnosis. The diagnosis of gout is a clinical one, with imaging and labs providing supporting evidence. Current imaging modalities such as magnetic resonance, computed tomography, and X-ray can be nonspecific and lead to invasive procedures for diagnosis. Dual-energy computed tomography allows clear visualization of urate collection and is a valuable tool to make a confident diagnosis of spinal gout. Here, we present a case of a man with longstanding severe gout in which dual-energy computed tomography played a key role in diagnosis.

© 2022 The Authors. Published by Elsevier Inc. on behalf of University of Washington.

This is an open access article under the CC BY-NC-ND license

(<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

Introduction

Axial (Spinal) gout is defined by the deposition of monosodium urate crystals (MSU) in the axial skeleton [1]. Spinal gout was previously thought to be a rare manifestation of gout, however in those with longstanding gout the prevalence has been shown to be up to 35%. This is much higher than what previous studies have suggested and shows the importance of recognizing these atypical presentations of gout [1,2]. Depending on severity axial gout can present in a variety of ways ranging from asymptomatic, to fever, back pain, and elevations in erythrocyte sedimentation rate, C-reactive protein, and white blood cell counts [3,4]. On the

more severe spectrum, it may also present with radiculopathy and symptoms of neural compression [1,4,5].

Due to its nonspecific presentation, spinal gout may be confused for other pathologies such as vertebral osteomyelitis, malignancy, epidural abscess, and infections [3,6,7]. Imaging can be a valuable diagnostic aid in uncertain cases; when the initial complaint is back pain or neurological compromise, magnetic resonance (MR) is usually the preferred modality [5]. MR findings, however, can be nonspecific and may prompt further investigation through surgical or diagnostic procedures. Other imaging modalities, such as computed tomography (CT) and radiography (X-ray) are more specific but may not be sufficient to confirm the diagnosis. Dual-energy computed tomography (DECT) is a newer imaging protocol, and when com-

[☆] Competing Interests: None.

^{☆☆} Funding: None.

^{*} Corresponding author.

E-mail address: brahmhatt.pavan@mayo.edu (P. Brahmhatt).

<https://doi.org/10.1016/j.radcr.2022.08.009>

1930-0433/© 2022 The Authors. Published by Elsevier Inc. on behalf of University of Washington. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

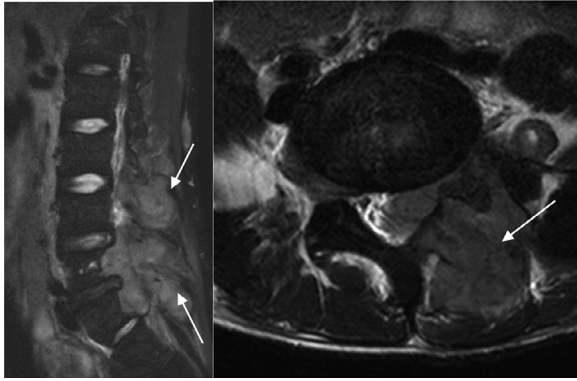


Fig. 1 – Extensive tophaceous gout along the left posterior elements from L3 to S1, extending into the neural foramina with encroachment upon the canal. Severe narrowing of the left L4-L5, L5-S1, and S1-S2 neural foramina with compression of the exiting nerve root (white arrows).

pared to classical CT, it has been shown to be highly effective in showcasing gout [8].

DECT imaging has the ability to differentiate urate deposition from the surrounding structures due to attenuation characteristics [8,9]. This allows clinicians to clearly visualize gout when compared to other imaging modalities and can be a useful tool diagnostically, preventing invasive procedures and obviating the need for biopsy/aspiration to confirm the presence of MSU crystals [8,10,11]. Here, we review the case of a 39-year-old male with a history of gout, who presented to the hospital with generalized weakness and lower left limb weakness. We aim from this review to highlight the atypical presentation of axial gout and how DECT can be a useful clinical tool in diagnosis and management.

Case presentation

A 39-year-old male presented to the emergency department with generalized weakness, chronic pain from head to toe, left limb weakness, and a chronic wound draining pus from his left foot. Following evaluation, he was admitted for symptomatic anemia and lower extremity limb weakness. The patient had a past medical history of severe chronic tophaceous gout, beta thalassemia, chronic anemia, splenomegaly due to extramedullary hematopoiesis, pancytopenia, and severe malnutrition. Despite being on allopurinol and colchicine, his gout progressed. On physical exam, he had bilateral paraspinal soft tissue masses, multiple tophi on extremities, including tophus throughout his left lateral foot wound draining thick yellow discharge. His uric acid on admission was 6.2 mg/dl (reference range 4.0–8.5 mg/dl). An MRI and X-ray of the foot were ordered to rule out osteomyelitis and showed erosions due to chronic gout. To evaluate his back pain and lower left limb weakness an MRI lumbar spine was done. MRI showed severe soft tissue abnormality involving both the lumbar region and the SI joints (Fig. 1) and due to the ambiguous findings on the MRI, a CT done with DECT protocol was indi-

cated. CT demonstrated amorphous soft tissue density consistent with extensive tophaceous gout. In Fig. 2, DECT are paired with their respective traditional CT scans, and DECT shows clear differentiation of urate deposition from the surrounding structures. The patient had severe tophaceous gout causing compression of the lumbar nerve roots and leading to lower left extremity weakness. Following consultation with rheumatology, he was switched to Febuxostat 80 mg with Prednisone 5 mg daily due to his nonresponse to allopurinol and colchicine and scheduled for outpatient rheumatology follow-up.

Discussion

Gout is the most common inflammatory arthritis in the world with a global prevalence of 1%–4% and higher in men and western countries. It is caused by an imbalance of urate production and excretion, leading to oversaturation and eventual deposition in tissue. The collection of MSU crystals in tissues and joints, leads to inflammation and pain [4,12]. While hyperuricemia (>6.8 mg/dl) is the most common risk factor for the development of gout, others include both modifiable and unmodifiable factors such as age, gender, alcohol intake, obesity, cardiovascular diseases, hemolytic disorders, etc. Typically, gout presents with acute or chronic manifestations. Acutely, it presents with fever, swollen erythematous joints, and severe pain. Chronically, the most seen phenomenon is tophi: a collection of urate crystals surrounded by granulomatous inflammation on bony prominences and joints [4,11].

Gout frequently presents in the distal extremities. While the first metatarsal joint is one of the most affected sites, gout may present anywhere in the body, including the spine [13]. In the spine, the most affected locations are the lumbar region and sacroiliac joints. Risk factors for developing axial gout and appendicular gout are the same, but spinal gout is associated with more severe gouty manifestations. Radiographic evidence of gouty changes in the peripheral skeleton (erosions, rat bite sign, tophi) were seen in 81% of patients with axial gout, indicating a poor degree of control [2,8]. Although it was previously hypothesized that the duration of gout may correlate with the occurrence of spinal gout, Lumezanu et al. [2] conducted a cross sectional study in which no correlation was shown. In our case, the patient had tophaceous involvement of multiple joints, indicating severe gout with poor control. A unique variable is his history of thalassemia. Thalassemia leads to a chronic hemolysis and extramedullary hematopoiesis, both of which contribute to increased cellular turnover, and therefore higher levels of serum uric acid. Chronically elevated levels of serum uric acid are a risk factor for gout, as well as renal and cardiovascular effects [8,14].

Gout is primarily a clinical diagnosis that can be aided by supporting evidence through laboratory investigations and imaging. However, the only way to confirm gout is by synovial fluid aspiration or a biopsy demonstrating negatively birefringent urate crystals [3,4,8]. When the clinical picture is equivocal, imaging can be useful in identifying the culprit. MR findings are nonspecific with tophi having a homogenous T1 hypointense signal and heterogeneous T2 hypo to hyperintense signal, which may mimic epidural abscess or tumor. If the

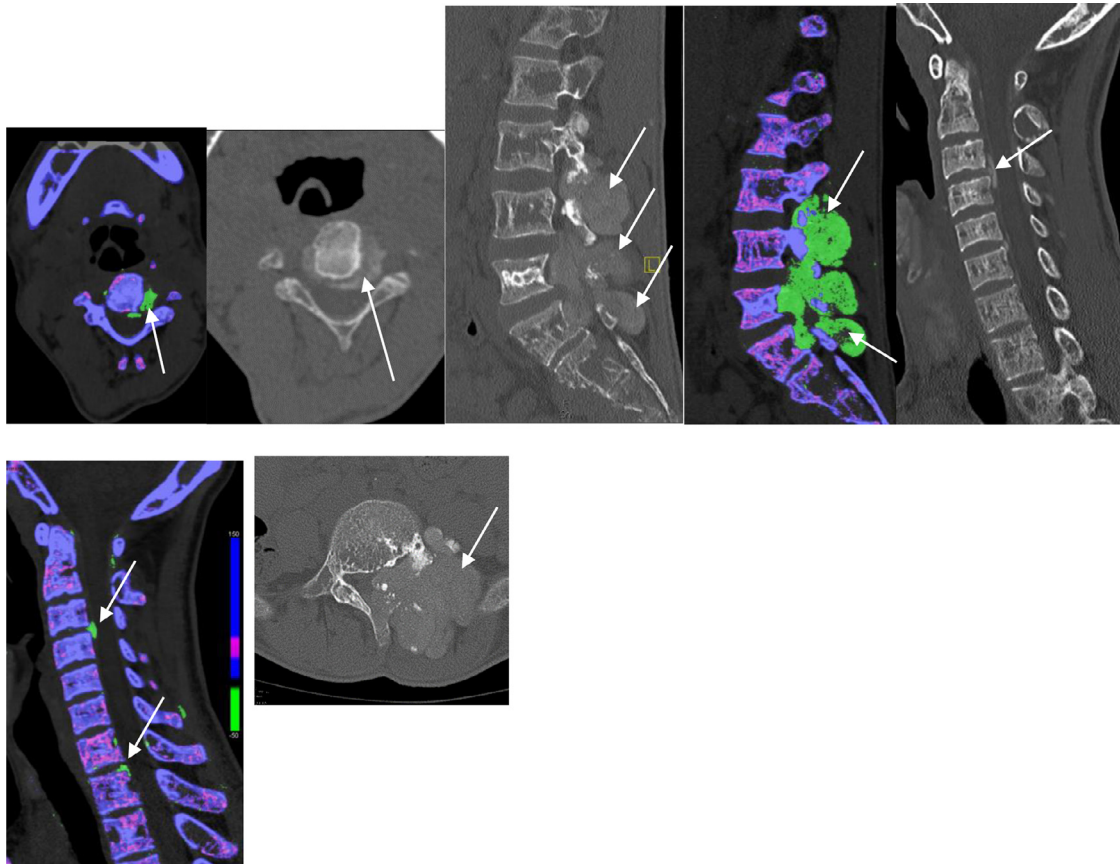


Fig. 2 – Extensive tophaceous gout with amorphous soft tissue density throughout the spinal axis, bilateral SI joints, costosternal junctions, multiple ribs, and subcutaneous soft tissues best depicted on the dual-energy color-coded scans (shaded green) (white arrows).

disk space is involved gout appears as T1 hypointense and T2 hypointense/isointense similar to degenerative changes and spondylodiscitis. CT findings show periarticular punched out erosions with overhanging margins and tophi appear as masses denser than surrounding tissues [10,15].

DECT uses 2 energy levels of X-ray beams (80 kV and 140 kV) to differentiate structures based on their attenuation characteristics in relation to each energy level. After post processing, materials are color coded to help identification, where urate is often coded green [8,9]. Initially, DECT was used to identify urate nephrolithiasis but has subsequently expanded its utility to diagnose gout. Its sensitivity and specificity for gout are 90% and 83%, respectively, and has diagnosed gout in those with negative synovial fluid analysis [8,16]. When compared to traditional CT and imaging, DECT has the unique ability to highlight MSU deposition and distinguish it from the surrounding calcium. In relation to spinal gout, DECT clearly demonstrates urate deposition, allowing for a more precise diagnosis compared to other imaging modalities. While spinal gout was typically an incidental pathologic diagnosis following biopsy or surgery for suspected malignancy or abscess, DECT may reduce the need for such invasive procedures [10,11]. Once identified, a patient can receive treatment with colchicine and allopurinol, which is the same as appendicular gout. Many patients experience rapid relief in symptoms with medical management alone; however, more serious

or resistant cases may require the addition of steroids and second line drugs such as febusostat or surgical intervention to relieve neuropathic compression.

In addition to diagnosis, DECT is highly effective and measuring even the smallest change (less than 2%) in tophi burden and can be used to monitor the treatment progress [8,9,11]. Currently, DECT scanners are nearly double the price of traditional CT, limiting its widespread availability. DECT also uses slightly higher radiation doses when compared to CT, which must be taken into consideration in patients undergoing multiple imaging studies to prevent higher cumulative radiation doses [8]. DECT postprocessing may lead to artifacts which do not correspond to urate deposition and must be accounted for to avoid false positives. Common sources of artifacts are nail beds, tendons, and advanced osteoarthritis. DECT is also limited in its ability to detect acute recent onset gout, which is the main cause of false negatives [16].

Conclusion

Spinal gout is more prevalent than previously thought and must be considered in patients with chronic gout with concurrent lumbosacral symptoms. Due to its nonspecific presentation, spinal gout can be difficult to confirm on traditional

imaging modalities. DECT imaging has the potential to be an invaluable tool to identify spinal gout and avoid invasive interventions.

IRB approval

We acknowledge that our institution does not require IRB approval for case reports or image submissions.

Patient consent

Written patient consent has been obtained as per Institutional policy. No patient image or identifying information included in this manuscript. All the images included are non-identifiable images consistent with Elsevier policies

REFERENCES

- [1] Konatalapalli RM, Demarco PJ, Jelinek JS, Murphey M, Gibson M, Jennings B, et al. Gout in the axial skeleton. *J Rheumatol* 2009;36(3):609–13.
- [2] Lumezanu E, Konatalapalli R, Weinstein A. Axial (spinal) gout. *Curr Rheumatol Rep* 2012;14(2):161–4.
- [3] Cordova SA, Bisen M, Khokhar F, May A, Ben Gabr J. Diagnosing spinal gout: a rare case of back pain and fever. *Case Rep Rheumatol* 2021;2021:7976420.
- [4] Fenando A, Rednam M, Gujarathi R, Widrich J. Gout. *StatPearls*. Treasure Island (FL): StatPearls Publishing Copyright © 2022, StatPearls Publishing LLC; 2022.
- [5] Konatalapalli RM, Lumezanu E, Jelinek JS, Murphey MD, Wang H, Weinstein A, et al. Correlates of axial gout: a cross-sectional study. *J Rheumatol* 2012;39(7):1445–9.
- [6] Nakajima A, Kato Y, Yamanaka H, Ito T, Kamatani N. Spinal tophaceous gout mimicking a spinal tumor. *J Rheumatol* 2004;31(7):1459–60.
- [7] Bonaldi VM, Duong H, Starr MR, Sarazin L, Richardson J. Tophaceous gout of the lumbar spine mimicking an epidural abscess: MR features. *AJNR Am J Neuroradiol* 1996;17(10):1949–52.
- [8] Khanna I, Pietro R, Ali Y. What has dual energy CT taught us about gout? *Curr Rheumatol Rep* 2021;23(9):71.
- [9] Desai MA, Peterson JJ, Garner HW, Kransdorf MJ. Clinical utility of dual-energy CT for evaluation of tophaceous gout. *Radiographics* 2011;31(5):1365–75.
- [10] Ayoub S, Rajamohan AG, Acharya J, Gross J, Patel V. Chronic tophaceous gout causing lumbar spinal stenosis. *Radiol Case Rep* 2021;16(2):237–40.
- [11] Ragab G, Elshahaly M, Bardin T. Gout: an old disease in new perspective—a review. *J Adv Res* 2017;8(5):495–511.
- [12] Zhu Y, Pandya BJ, Choi HK. Prevalence of gout and hyperuricemia in the US general population: the National Health and Nutrition Examination Survey 2007–2008. *Arthritis Rheum* 2011;63(10):3136–41.
- [13] Kotake S, Nanke Y. Spinal tophaceous gout. *Intern Med* 2012;51(3):237–8.
- [14] Chaloeuwong J, Tantiworawit A, Rattanathammethee T, Chai-Adisaksopha C, Rattarittamrong E, Norasetthada L, et al. Hyperuricemia, urine uric excretion, and associated complications in thalassemia patients. *Ann Hematol* 2019;98(5):1101–10.
- [15] Mogensen MA, DeConde RP, Sarikaya B. Spinal gout: Imaging and clinical features. *PM R* 2021;13(11):1304–6.
- [16] Bongartz T, Glazebrook KN, Kavros SJ, Murthy NS, Merry SP, Franz WB, et al. Dual-energy CT for the diagnosis of gout: an accuracy and diagnostic yield study. *Ann Rheum Dis* 2015;74(6):1072–7.