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Gout in the Spine: Imaging, Diagnosis, and Outcomes

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Abstract Gout is characterized by the deposition of monosodium urate crystals and by acute and chronic inflammation in response to crystals so deposited. Multiple case reports and series describe the deposition of monosodium urate in the spine as a rare manifestation of gout, but the actual prevalence of spinal involvement is unknown and likely to be higher than generally anticipated. Here we review the characteristics of 131 previously reported cases of spinal involvement in gout. We focus in particular on the use of imaging modalities and the extent to which they correlate with presenting symptoms and tissue diagnoses. The recent innovation of using dual-energy computerized tomography to identify urate crystal deposition holds promise for reducing the need for surgical intervention and for establishing a true prevalence rate for spinal gout.

Keywords Gout · Spine · Tophus · Back pain · Dual-energy CT · DECT

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Introduction

The incidence of gout, a monosodium urate crystal deposition disease, has been increasing, and gout now affects almost 4 % of all adults in the USA [1]. Gout most commonly affects the first metatarsophalangeal joint, knees, ankles, wrists, and hand joints. With unknown frequency, gout may also affect the spine. The first case of gout in the spine was published in 1950 [2]. Since then, an accruing series of cases has been reported in the literature, but limitations in diagnostic methodology, and lack of studies assessing for spinal gout in asymptomatic or mildly symptomatic patients, have made an accurate assessment of incidence and prevalence difficult to attain. Several authors have speculated that the incidence of spinal gout has been, and remains, more common than previously believed [3–5].

Comprehensive reviews have disagreed on the best approach to diagnosis and treatment of this condition. Although some authors have advocated for computerized tomography (CT) as the best method to identify spinal urate deposition [4, 6], others have asserted that imaging was inherently nonspecific. These authors believed that only a tissue sample could provide a correct diagnosis, despite the risks of surgery and low yield of biopsies [7]. In this manuscript, we review the literature concerning spinal gout, including symptomatology, abnormal laboratory results, pertinent imaging findings, diagnostic methods, and treatments. We then examine cutting-edge methods to diagnose spinal gout and possibly uncover the prevalence of this condition.

Methods

A literature search using the terms “spine,” “vertebral,” “tophus,” and “gout” was carried out using PubMed. Articles that

discussed cases of gout involving the spinal column, including case reports, retrospective trials, and review articles, were included. Additional cases were added after cross-referencing previously published review articles. Articles describing gout affecting only the sacroiliac joints (37 cases) [5, 18, 66] were excluded, in order to focus specifically on cases involving the spinal column.

A total of 133 cases of spinal gout were identified. Two cases were excluded (one was in another language and the other could not be accessed), leaving 131 total cases that were included in the review [1–132]. Ours is the most comprehensive review of spinal gout to date.

Results

Demographics

Of the 131 cases reported, 129 included a gender. Ninety-eight (76 %) of those 129 were males and 31 (24 %) were females. In two cases, the gender was not reported. The ages ranged from 17–87, with a median age of 58.9 (with most cases occurring between 44 and 74 years).

Symptoms

Patients with spinal gout presented variably, with acute, subacute, or chronic symptoms. In two cases, patients had no symptoms and the diagnosis was made incidentally on autopsy for other reasons [43, 73]. Symptom chronicity ranged from 1 day to 6 years [92, 112]. One patient had new MRI lesions develop over only 3 months, concomitantly with her symptoms. [33]

Of the total 131 cases, 130 mentioned presenting symptoms of the patient. The most common symptom was back pain, which was present in 89 (68.5 %) cases. When present, back pain was usually but not consistently reported in the general area of the spinal level affected by urate deposition. Tkach et al. found a frequent coexistence of primary osteoarthritis and gout in patients who had pain in their spines, but whether the pain was due to gout, osteoarthritis, or both was not made clear [113]. The second most common complaint was neurological impairment. Patients variously presented with radiculopathy, loss of sensation, motor weakness, bowel/bladder dysfunction, or quadriparesis. Not surprisingly, back pain and neurologic impairment were not mutually exclusive. Eighty-five (65.4 %) of the 130 cases where the presenting symptom was mentioned had neurological findings, either as a symptom or as a sign on physical exam. These neurologic symptoms or signs were frequently concerning for compression of the spinal cord or exiting spinal nerves by the mass (ultimately identified as tophus), prompting the pursuit of tissue diagnosis, often in the context of surgical management. In many cases, diagnosis of spinal urate deposition was accomplished in the course of a workup over concern for a mass or tumor. Aside from back pain and/or neurologic involvement, no other indications for referral were reported.

Of the 131 total cases, 129 included mention of the spinal level where the gouty tophus was found. Most cases (80.6 %) only involved one region of the spine. In 32 (24.8 %) cases, the tophus affected only the cervical spine. The lumbar spine alone was affected in 49 (38.0 %) cases. The thoracic spine alone was involved in 23 (17.8 %) cases. Eighteen (14.0 %) cases affected both the lumbar and upper sacral (S1) spine. A single (0.7 %) case involved both cervical and thoracic spine. Six (4.7 %) cases affected both thoracic and lumbar spine. There were two cases where the spinal level was not mentioned. When including the patients with multiple spinal regions involved, 33 patients had gouty tophi in the cervical spine, 73 patients had lumbar spine pathology, and 29 cases had gout in their thoracic spine.

Prior History of Gout

Of the 131 cases, 122 addressed a past history of gout. The majority of patients (75.4 %) had a history of gout or the known gout risk factor of hyperuricemia. Eighty-seven patients (71.3 %) had pre-existing gout, 5 (4.1 %) had recognized hyperuricemia without gouty attacks, and 30 (24.6 %) had no prior history of either gout or hyperuricemia. However, it is likely that many patients without a known previous history of gout had not had a prior serum urate level, resulting in underestimation of the prevalence of pre-existing hyperuricemia. Nine of the case reports made no mention of any prior history. Among the 100 gout patients for whom the presence or absence of visible peripheral tophi was reported, 59 had tophi on exam and 41 did not have tophi; there was no mention in the remaining 31.

Regarding risk factors for gout, there were 21 cases with a history of kidney disease and 4 cases that had received a renal transplant after end-stage renal disease: two due to IgA nephropathy [3, 9], one due to progressive membranoproliferative glomerulonephritis [45], and one without clear reason for renal failure [94].

Laboratory Values

The most frequently abnormal values encountered in patients with spinal gout included the serum uric acid (sUA) concentration, the erythrocyte sedimentation rate (ESR), the C-reactive protein (CRP), the white blood count (WBC), and the serum creatinine (Cr). For the purposes of analyzing and unifying the data, we defined the normal value for uric acid as <7 mg/dL, the normal ESR as <20 mm/h, the normal CRP as

<3 mg/L, the normal WBC as <12,000/μL, and the normal Cr as ≤1.2 mg/dL.

Of the 131 cases reviewed, the sUA was reported in 104. Of those cases, 18 (17.3 %) had a sUA <7 mg/dL and 83 (79.8 %) had a sUA >7 mg/dL. In three (2.9 %) additional cases, the sUA was reported to be normal but a numeric value was not provided. The median sUA was 9.9 mg/dL (range, 1.9–19.1 mg/dL) [29, 64]. Thus, patients with spinal gout often but not always had an elevated sUA at the time of evaluation, and a normal sUA did not exclude spinal gout from the diagnosis.

ESR and CRP were also found to be elevated in some cases. The ESR was only provided in 36 (27.5 %) cases; in 33 of those (92 %), the ESR was elevated, and the mean ESR was 75.9 mm/h. CRP was similarly mentioned in only 37 (28.2 %) cases but was elevated in 34 of those (92 %), with a mean of 117.13 mg/L. In the aforementioned cases, 15 had an elevation in both ESR and CRP.

The WBC was reported in 49 of the 131 cases. It was elevated in 14 (28.6 %) cases and normal in 27 (55.1 %), with 8 (16.3 %) additional cases listed as normal without a numeric value provided. In 82 cases, the WBC was not mentioned. The average WBC was 11.7. Eleven cases reported concurrent elevations in WBC, ESR, and CRP; in the other cases with an elevated WBC, the ESR and CRP were not reported.

The serum Cr was mentioned in 52 of the 131 cases. It was >1.2 mg/dL in 40 (76.9 %) of the 52 cases, ≤1.2 mg/dL in four (7.7 %) of those cases, not mentioned but presumed elevated in one (1.9 %) patient who had end-stage renal disease, and listed as normal without a value in seven (13.5 %) cases. The Cr was not mentioned in the remaining 79 cases.

Overall, the laboratory abnormalities were often consistent with an inflammatory state such as is often seen in acute gouty arthropathy in other joints. However, 13 of the 23 cases whose initial presentation included fever also had an elevation in ESR, CRP, or WBC, if not all three. In these cases, infection was higher on the differential than gout, making the diagnosis without a tissue sample more challenging.

Imaging Modalities

The imaging studies most often performed on patients with spinal gout were plain radiographs, magnetic resonance imaging (MRI), and computed tomography (CT) (including scans with contrast, positron emission tomography, and dual-energy formats). Much less frequently, patients had myelography or technetium bone scans. In addition to spinal imaging, many of the patients who had peripheral arthritis symptoms also had plain radiographs of those joints.

Sixty-three patients underwent plain radiography of the spine, with 51 (81.0 %) of the 63 having abnormal radiographs. The other 12 (19 %) had unremarkable findings on their X-rays. Generally speaking, many of the abnormalities reported on plain radiographs were nonspecific for spinal gout. Abnormalities reported include spondylosis [21], spondylolisthesis [115], degenerative changes [11], and diffuse spinal hyperostosis [50]. In other cases, radiographs were read as normal [116]. Patients with severe neurological symptoms were often the ones with the most abnormal X-ray findings, showing focal narrowing or destructive changes, including atlanto-axial subluxation [124]. In subsequent evaluation, these findings were typically associated with large invasive tophi. However, there was no consistent relationship between the X-ray findings and the laboratory results.

MRI with gadolinium contrast was the most common method used to evaluate patients with spinal gout; 90 of the cases received an MRI, and 89 (98.9 %) of those cases were abnormal, revealing a high degree of sensitivity. However, findings on MRI were usually nonspecific, with only 19 (21 %) of the 90 cases interpreted specifically as a gouty or tophaceous lesion (Fig. 1) [17–19, 28, 47, 75, 88, 90, 103, 104, 107, 112, 114, 116, 125]. Changes on MRI may mimic osteomyelitis or tuberculosis, creating a challenge for making a specific diagnosis. Although classically believed to be hypointense on T1 and hyperintense on T2 [114], tophi were variously reported as hypointense or isointense soft tissue

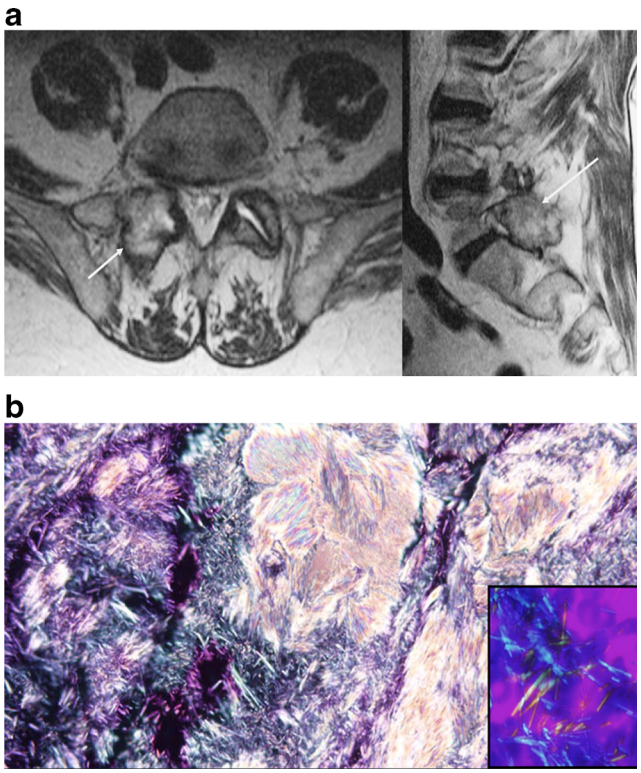

Fig. 1 **a** Presence of a mass in the lumbosacral region of the spine, as seen on MRI in coronal and sagittal views (mass indicated by *white arrows*). **b** Confirmation of mass as a tophus through surgical biopsy and examination of recovered material under polarizing microscopy. Reprinted from Samuels et al. [101] (with permission)

densities on T1 and ranged from hypointense to hyperintense onT2 [17, 47, 66, 112]. Among patients who received gadolinium contrast as part of their MRI, tophi often demonstrated either homogenous or heterogeneous peripheral enhancement [3, 5, 7, 11, 76, 93].

CT appears to be more sensitive and specific than plain radiography for the diagnosis of spinal gout, but given the fact that most studies involved case reports, an accurate denominator is lacking. Fifty-seven (96.6 %) of the 59 CT scans performed on patients with spinal gout were read as abnormal, with 11 (18.6 %) scans reported as highly suspicious for tophaceous gout [37, 42, 49, 57, 66, 75, 86, 88, 90, 108, 112]. The most common findings included bone or joint erosions with well-defined sclerotic margins, facet or intervertebral bone neoformation, or juxta- or intra-articular masses that were denser than the surrounding muscle [4, 132]. Other CT findings that were less frequently reported included degenerative changes (5 cases) [120], lytic lesions(18 cases) [17, 39, 96], and spinal stenosis (7 cases) [14, 123]. One subject underwent a PET-CT to evaluate spinal gout, which revealed increased uptake around the lesion [18], consistent with the fact that tophi are complex structures that include surrounding zones of metabolically active leukocytes and fibroblasts [22]. Variability of standard CT findings appears to depend in part upon where the uric acid crystals deposit within the joints. For example, epidural compression by a mass can easily be confused for a tumor or abscess and cannot be differentiated without a tissue diagnosis.

A newer method of imaging gout is dual-energy CT (DECT) scanning. DECT scanners capture images at two separate energy levels, compared to the standard single-energy CT. The separate energy levels allow substances of different chemical composition to appear distinct based on their differential X-ray photon energy [87], and more effectively differentiate tophi from other types of masses. Using DECT, researchers have been able to identify subclinical gouty tophi and directly measure their volume [23]. In a recent study, Hu et al. found the sensitivity and specificity of DECT in identifying gouty tophi at 91.9 and 85.4 %, respectively, when adjusting certain CT scanner settings for performing imaging and analysis [48]. More recent studies suggest that DECT may be less sensitive at identifying diffuse as opposed to dense lesions [26]. To date, most reports and studies of DECT scanning in gout have focused on tophi in peripheral appendages.

In two reported cases, DECT has been used as part of the workup of the spinal gout patient. Dhaese et al. used DECT to confirm urate deposition in the costovertebral joints of the thoracic spine and treated the patient conservatively, with complete resolution of all symptoms (Fig. 2) [29]. Similarly, Parikh et al. employed DECT on a patient who had gout with worsening back pain and discovered deposition of uric acid crystals in the lumbar facet joints [93]. While the urate depositions were clearly seen on DECT, a conventional CT image only showed nonspecific areas of mildly increased attenuation where the tophi were located. This patient was treated conservatively and improved.

The relative specificity of DECT may permit, for the first time, an assessment of the actual prevalence of spinal involvement in gout. In a report published in abstract form, Law et al. performed DECT of the thoracic and lumbar spine on 17 patients with tophaceous gout. They observed that eight (47 %) patients had DECT evidence of MSU deposits, most commonly in the lumbar spine [69]. Back pain was reported to be a common complaint, but actual prevalence of back pain was not provided. These observations suggest that, at least among gout patients with peripheral tophi, spinal deposition of urate may be more common than previously appreciated, may contribute to the total body burden of urate, and may contribute to back pain.

Diagnosis

Definitive diagnosis of the tophus was most commonly made during surgery, revealing a white chalky mass visualized macroscopically, and negatively birefringent urate crystals seen microscopically under polarizing conditions (Fig. 1). Surgery was performed in 75 cases. An additional 32 cases were diagnosed via needle aspiration or bone biopsy of the suspected lesions. Eleven cases were diagnosed without tissue by consideration of the overall clinical picture, including history, physical exam, elevated uric acid, and abnormal imaging findings. Four additional cases diagnosed the patient on autopsy with tophi found in the spine [2, 43, 67, 73]. In three cases, the patient was diagnosed indirectly by obtaining crystals from other joints, combined with abnormal findings on imaging studies and a high suspicion for spinal gout [71, 104, 125]. In six reports, the means of diagnosis was not mentioned.

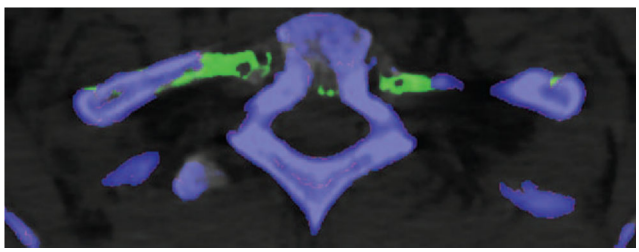

Fig. 2 Presence of monosodium urate crystal deposition at the level of thoracic vertebrae 1 and 2, on axial projection, imaged using dual-energy computed tomography. In this image, the green areas are indicative of uric acid. From Dhaese et al. [29] (reproduced with permission of Lippincott Williams & Wilkins in the format journal/magazine via Copyright Clearance Center)

Treatment

Forty-three (38 % of all treated patients) cases of spinal gout were reported to be managed with surgery alone; 26 (23 % of all treated patients) cases were managed with a combination of surgery and pharmacologic treatment. Forty-two patients (37.2 % of all treated patients) were treated conservatively (i.e., nonsurgically) with various combinations of colchicine, steroids, allopurinol, and/or febuxostat. Two (1.8 % of all treated patients) patients failed initial conservative treatment and were subsequently treated surgically with improvement in their symptoms. Four patients died of other causes prior to receiving treatment. In 14 reports, treatment information was not provided. Forty (37.7 %) patients had complete resolution of symptoms with treatment. Fifty-five (51.9 %) patients noted some improvement without complete mention of resolution. Two (1.9 %) patients did not improve after being treated, and nine (8.5 %) died of various causes (one after an initial report of gout improvement). Of those reported to be treated only surgically, 18 (47.4 %) had complete resolution of symptoms, 18 (47.4 %) improved, and 2 (5.2 %) died post-operatively (both of pneumonia). No data was available regarding symptom resolution in the remaining five patients treated only surgically. It is possible that some of these patients were subsequently treated medically but that the results were not reported. Among those subjects reported to be treated first surgically, then medically, four (15.4 %) cases completely resolved, 19 (73 %) cases improved, one (3.8 %) died from post-operative infection, one (3.8 %) did not improve, and one case had no treatment outcomes mentioned. Of those patients treated medically only, the outcome was mentioned in 38 of the 42 cases. Seventeen (44.7 %) of those 38 cases had complete resolution of symptoms, 18 (47.4 %) cases improved, one (2.6 %) initially improved but died from aspiration pneumonia, another case (2.6 %) died of pneumonia, and one (2.6 %) case did not improve. In the remaining four cases, the authors did not provide an outcome.

Based on the above data, conservative medical treatment appears to be as effective as surgical treatment while avoiding post-operative complications. Often, patients who presented with focal neurologic deficits received surgery for salvage. Fifty-nine (84.3 %) patients with neurological symptoms were treated with surgery (with or without medications), compared to only 11 (15.7 %) without neurological findings who received surgery. On the other hand, patients who had no neurological findings were more likely to be treated conservatively. Twenty-five (59.5 %) patients whose chief complaint was back pain without neurologic deficits were treated surgically, in contrast to 17 (40.5 %) with neurological findings who were treated conservatively.

Discussion

Spinal gout can present in a variety of ways. Symptoms—mainly back pain, and/or nerve or spine compression—are not specific for gout and can occur within a short time frame or develop over many years. Patients most commonly have an extensive prior history of gout or hyperuricemia, but spinal involvement can also be the first manifestation of gouty symptoms. The uric acid is usually elevated, as are inflammatory markers such as ESR, CRP, and WBC. However, none of these satisfactorily excludes infection from the differential diagnosis, and vigilance is required, especially if a patient presents with fever.

Although a variety of imaging studies have been used to evaluate spinal gout, most are nonspecific and cannot definitively exclude other causes of spinal mass. Therefore, imaging usually leads to a more invasive measure to diagnose the condition, such as open surgery or needle biopsy (which may not yield diagnostic tissue or may require multiple attempts) [3], to differentiate gout from infection or tumor. DECT is a newer imaging modality that is both sensitive and specific for monosodium urate deposition in peripheral joints. Further study is warranted to determine whether the sensitivity and specificity of DECT are sufficient to obviate the need for invasive procedure and tissue diagnosis in the case of a spinal mass observed on imaging. At the present time, however, DECT is not widely available and is more expensive than conventional CT, and many radiologists will need to be trained before they can effectively read the images. [48]

In gout patients who present with back pain not responding to conservative measures, urate crystal deposition in the spine should be higher on the list of differentials, and DECT should be considered to work up the patient, if available. If DECT is not available, CT and/or MRI with biopsy (if needed) can also be used. Treatment for nonurgent cases should initially be conservative and resemble the treatment of other forms of chronic gout, with anti-inflammatory prophylaxis accompanied by urate lowering therapy, e.g., a xanthine oxidase inhibitor (allopurinol or febuxostat) with or without probenecid, or pegloticase in the case of inadequate urate lowering with the initial regimen. If symptoms do not improve, surgery may be the next step. In patients with urgent neurological symptoms, especially evidence of spinal compression, surgery may be the first treatment option in order to remove the gouty mass.

The prevalence of spinal gout is still unknown, and the use of conventional imaging modalities has shed only limited light on the question. Among a sample of patients with gout, Jajic et al. reported that 12 of 54 (22 %) had abnormal radiographs with hyperostotic spondylosis or diffuse idiopathic spinal hyperostosis [50]. The results suggest that gout patients commonly have spine disease, but the findings they report are not specific to gout and do not directly reflect the presence of urate. Konatalapalli et al. studied the prevalence of gout

with a retrospective study using CT scans and reported that nine of 64 (14 %) gout patients in the study had evidence of uric acid crystal deposition on their CT scans, while the remainder did not have the characteristic findings [4]. The lumbar spine was most commonly affected, occurring in 80 % of all abnormal CT scans. Of the patients studied, only three had gotten CT scans for back pain, while the rest had gotten CT scans for other indications. Of those three patients, two had no CT findings of spinal gout, and only one had a CT consistent with axial gout. However, the study had a small sample size and had no histological confirmation making the data more difficult to interpret. Although the number of subjects was once again small, the unpublished DECT study by Law et al. suggests that spinal gout involvement may be much more common than previously appreciated, possibly approaching 50 % in patients with clinically appreciated tophi [69].

Overall, we conclude that spinal involvement in patients with gout may be common, may be an underappreciated source of axial pain, and may require consideration as a urate reservoir and a barrier to urate lowering therapy. Additional DECT studies are clearly warranted, both to better understand the modality and to better understand the incidence and prevalence of spinal gout.

Compliance with Ethical Standards

Conflict of Interest Dr. Pillinger has received grant support from Takeda Inc. and Crealta. He has also served as a consultant for AstraZeneca and Crealta.

Dr. Krasnokutsky and Dr. Toprover has nothing to disclose.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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