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SYSTEMATIC REVIEWS

Spinal gout: A review with case illustration

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Abstract

AIM

To summarize clinical presentations and treatment options of spinal gout in the literature from 2000 to 2014, and present theories for possible mechanism of spinal gout formation.

METHODS

The authors reviewed 68 published cases of spinal gout, which were collected by searching "spinal gout" on PubMed from 2000 to 2014. The data were analyzed for clinical features, anatomical location of spinal gout, laboratory studies, imaging studies, and treatment choices.

RESULTS

Of the 68 patients reviewed, the most common clinical presentation was back or neck pain in 69.1% of patients. The most common laboratory study was elevated uric acid levels in 66.2% of patients. The most common diagnostic image finding was hypointense lesion of the gout tophi on the T1-weighted magnetic resonance imaging scan. The most common surgical treatment performed was a laminectomy in 51.5% and non-surgical treatment was performed in 29.4% of patients.

CONCLUSION

Spinal gout most commonly present as back or neck pain with majority of reported patients with elevated uric acid. The diagnosis of spinal gout is confirmed with the presence of negatively birefringent monosodium urate crystals in tissue. Treatment for spinal gout involves medication for the reduction of uric acid level and surgery if patient symptoms failed to respond to medical treatment.

Key words: Spinal; Gout; Tophi; Monosodium urate

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Core tip: Gout is a common inflammatory arthritis that rarely affects the spine. In such cases, patients may experience back pain, myelopathic symptoms and radiculopathy. Clinical findings are non-specific. Therefore, it is necessary to have an awareness of the diagnosis,

especially in patients with a clinical history of gout and/or elevated inflammatory markers and hyperuricemia. While magnetic resonance imaging is the major non-invasive diagnostic method, all suspicious findings on imaging require surgical sampling for pathological confirmation. While typical uric acid lowering medications are first-line therapy, cord compression or continued symptoms may necessitate operative intervention if medications fail.

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INTRODUCTION

Gout is a common inflammatory arthritis with an increase in prevalence over the last 20 years. It currently affects over 8 million Americans. The clinical presentation of gout depends on the site of monosodium urate (MSU) crystals precipitation and the subsequent inflammatory response that ensues in the synovial joints and soft tissues. Gout usually manifests as a monoarticular arthritis in the lower extremities. If untreated, nodular masses of MSU crystals called tophi may eventually deposit in extraarticular locations, such as, the axial skeleton. Although traditionally thought of as a rare complication, recent study suggests that axial gout may be more prevalent than suspected[1]. Gout affecting the spinal column will typically present with neurological compromise, localized pain, and lytic vertebral lesions[2,3]. Spinal gout can affect the facet joint, laminae, ligamentum flavum, as well as the epidural space[4].

From 2000 to 2014, approximately 68 case reports have been published on spinal gout. The current manuscript summarizes the most common presenting features, imaging findings, and treatment choices based on the 68 published cases. A case is also presented to provide illustration on the topic.

MATERIALS AND METHODS

Literature review

A PubMed literature search using the key words spinal gout, from January 2000 to December 2014, limited to human studies and restricted to English language literature resulted in 221 publications. Abstracts and articles were then reviewed for content. Articles kept for review included patients who underwent treatment for spinal gout. Furthermore, data required for inclusion in the study included: Patient demographics, clinical presentation, laboratory findings, imaging studies, and treatment methods were collected (Table 1). Articles excluded from the study were those that did not have patients diagnosed with spinal gout and those that did not include patient demographics, clinical presentation, laboratory findings, imaging studies, and treatment methods. After review, a total of 54 peer reviewed articles met the above criteria and were included for data collection.

RESULTS

The 54 articles accounted for 68 cases of spinal gout with 51 (75%) males and 17 (25%) females and an average age of 59.2 years, 41(60.3%) had prior history of peripheral gout (Table 1).

Clinical presentations

Of the 68 spinal gout patients reviewed, 47 (69.1%) presented with localized back/neck pain, 38 (55.9%) with some form of spinal cord compression, defined as weakness, numbness, loss of bladder or bowel control, and decreased sensation below the compression level, 17 (25%) with spinal nerve root compression or radiculopathy, defined as motor dysfunction or dysesthesia along the course of a specific nerve caused by compression of its root, 13 (19.1%) with fever, 1 (1.5%) with cranial nerve palsy, and 2 (3.0%) with atlanto-axial subluxation (Table 2). Furthermore, among the sites of involvement in the 68 spinal gout patients, 38 (55.9%) were located in the lumbar region, 15 (22.1%) in the thoracic region, 15 (22.1%) in the cervical region, and 1 (1.4%) in an unspecified region (Table 3). One patient demonstrated soft tissue nodularity consistent with gouty tophi on biopsy in both the thoracic and lumbar spinal segments.

Laboratory studies

Laboratory studies of the 68 recorded cases showed 45 (66.2%) with elevated uric acid level at the time of diagnoses, 17 (25%) had elevated erythrocyte sedimentation rates (ESR), 19 (27.9%) had increased C-reactive proteins (CRP) level, 11 (16.2%) had renal insufficiency, 9 (13.2%) had leukocytosis, and 5 (7.4%) had anemia (Table 4).

Imaging studies

On the T1-weighted magnetic resonance imaging (MRI) images, 28 (41.2%) did not report findings, 31 (45.5%) were hypointense, 8 (11.8%) were isointense, and 1 (1.5%) was heterointense. On T2-weighted images, 24 (35.3%) did not report findings, 18 (26.5%) were hypointense, 12 (17.6%) were heterointense, 11 (16.2%) were hyperintense, and 4 (5.9%) were isointense. A gadolinium (Gd)-enhanced MRI scan was obtained from 32 (47.1%) patients. These findings are referenced in (Table 5).

Thirty-seven cases (54.4%) did not report X-ray findings, 12 (17.6%) showed spondylosis or spondylisthesis, 8 (11.8%) showed bony erosion, 6 (8.8%) were unremarkable, and 5 (7.4%) showed degenerative changes. In addition, 35 (51.5%) did not report computed tomography (CT) findings, 13 (19.1%) showed bony erosion and high density attenuation, 13 (19.1%)

Table 1 List of patient cases of spinal gout in the literature since 2000

No.	Year	Ref.	Age/ sex	Site	Sx/Signs	Duration	Hx Gout	Tophi	Relevant Hx	Hi Urate	MRI T1	T2	Gad	Tx
1	2000	Kao *et al*[5]	82 M	T10-T11	LE weakness	1 mo	Y	NA	NA	Y	Iso	Hypo	NA	T9-T11 lamina
2	2000	Mekelburg *et al*[6]	60 M	L2-3	Back pain	5 mo	Y	NA	NA	Y	NA	NA	NA	Cervical lamina
3	2000	Paquette *et al*[7]	56 M	L3	Back pain, radicular pain	6 yr	N	NA	Arthritis	NA	NA	Hypo	NA	Surgery
4	2000	Thornton *et al*[8]	27 M	L3-L4	Back pain	1 d	Y	NA	RT	Y	Hypo	NA	Y	Medical NOS
5	2001	Barrett *et al*[9]	70 M	L5-S1	Back pain, radicular pain, fever	2 d	Y	NA	RI	N	NA	Hyper	Y	Lamina
6	2001	St George *et al*[10]	60 M	T1-T2	LE weakness, BBD	6 wk	Y	N	NA	NA	NA	Hypo	NA	T1-T2 lamina
7	2001	Wang *et al*[11]	28 M	T9-T10	LE weakness	1 d	Y	N	NA	NA	NA	NA	NA	T9-T10 lamina
8	2002	Hsu *et al*[12]	72 M	L4-S1	Back pain, radicular pain	18 mo	Y	NA	NA	Y	Hypo	Hyper	Y	Lamina
9	2002	Hsu *et al*[12]	77 M	L3-L5	Back pain, radicular pain	12 mo	N	NA	NA	Y	Hypo	Hypo	Y	Lumbar lamina
10	2002	Hsu *et al*[12]	83 M	T9-T11	LE weakness	1 mo	Y	NA	NA	N	Hypo	Hypo	Y	Lamina
11	2002	Hsu *et al*[12]	27 M	L2-S1	Back pain	6 mo	Y	NA	NA	Y	Hypo	Hyper	Y	Medical NOS
12	2002	Souza *et al*[13]	49 M	T9-T10	Back pain, LE weakness	6 mo	Y	NA	NA	NA	Iso	Hypo	Y	T9-T11 lamina
13	2002	Yen *et al*[14]	68 M	C4-C5	Quadriparesis	2 wk	Y	NA	RI	Y	Hypo	Hypo	NA	Surgery
14	2003	Diaz *et al*[15]	74 M	C4-C5	Quadriparesis	1 wk	Y	Y	NA	Y	NA	NA	NA	C4-C5 lamina
15	2004	Draganescu *et al*[16]	48 F	L4	Radicular pain	1 d	Y	Y	Diuretic	Y	NA	Hetero	Y	L4-L5 lamina
16	2004	El Sandid *et al*[17]	32 M	T7-T9	Back pain, fever	Acute	Y	NA	NA	Y	NA	NA	NA	Lamina
17	2004	Nakajima *et al*[18]	39 M	L4-5	Low back pain	NA	Y	Y	Arthritis	Y	NA	NA	Y	Medical
18	2005	Beier *et al*[19]	29 M	L4-L5	Back pain, L5 radiculopathy	Acute	N	N	NA	Y	NA	NA	NA	L4-L5 lamina
19	2005	Celik *et al*[20]	48 M	C1-C2	Neck pain, radiculopathy, paresthesias	2 mo	N	Y	Alcohol	Y	Hypo	Hyper	Y	Medical NOS
20	2005	Chang[21]	60 M	L3-L4	B/L L4 radiculopathy	NA	Y	NA	NA	Y	Hypo	Hypo	Y	Surgery
21	2005	Chang[21]	72 M	L4-S1	Back pain, claudication	2 wk	Y	NA	NA	Y	Hypo	Hypo	Y	Surgery
22	2005	Chang[21]	66 F	L4-L5	Back pain, claudication	1 mo	Y	NA	NA	Y	Hypo	Hypo	Y	Surgery
23	2005	Chang[21]	63 M	L3-S1	Back pain, claudication, fever	2 wk	NA	NA	NA	N	Hypo	Hypo	Y	Surgery
24	2005	Kelly *et al*[22]	56 F	L4	Back pain, LE weakness	1 mo	Y	NA	RA, DM, RI	NA	Iso	Hypo	Y	L4-L5 lamina
25	2005	Mahmud *et al*[23]	47 M	L4-L5	Radiculopathy	3 mo	Y	NA	NA	Y	NA	NA	NA	L4-L5 lamina/ facet
26	2005	Mahmud *et al*[23]	71 F	L4-L5	Back pain, radiculopathy	4 mo	N	NA	NA	N	NA	Hetero	NA	L4-L5 lamina/ fusion
27	2005	Mahmud *et al*[23]	58 M	L4-L5	Back pain, claudication	6 mo	N	NA	NA	N	NA	Hyper	NA	L5 lamina
28	2005	Wazir *et al*[24]	66 F	C1-C2	Chronic neck pain, A-A subluxation, quadriparesis	2 mo	N	N	Arthritis	Y	NA	NA	NA	Lamina/ fusion
29	2005	Yen *et al*[25]	65 F	L5-S1	Back pain, LE weakness	10 mo	N	NA	NA	NA	Iso	Hetero	Y	L5-S1 lamina
30	2006	Dharmadhikari *et al*[26]	66 F	C3-C7	Cord compression, quadriparesis, falls	2-3 mo	N	N	NA	NA	Hypo	Hypo	N	C3-C6 vertebrectomy
31	2006	Hou *et al*[27]	37 M	L5-S1	Back pain, fever	5 d	Y	N	RT	Y	Iso	Iso	Y	Medical NOS
32	2006	Oaks *et al*[28]	32 M	T5-T8	Back pain, myelopathy	NA	Y	NA	NA	NA	Hetero	Hetero	Y	Lamina
33	2006	Pankhania *et al*[29]	68 M	C4-C5	Neck pain, quadriparesis, sensory dysfunction	1 mo	N	N	NA	N	Iso	Hetero	Y	Lamina
34	2006	Popovich *et al*[30]	36 F	T2-T9	Paraplegia	2 wk	Y	NA	NA	Y	Hypo	Hypo	Y	T5-T7 lamina
35	2007	Adenwalla *et al*[31]	77 M	L5-S1	Severe low back pain, LE weakness	1 wk	N	N	Diurectic	Y	NA	NA	NA	Prednisone and colchicines
36	2007	Lam *et al*[32]	65 M	L3-L4	LE pain and numbness, BBD	Acute	Y	Y	RI	Y	NA	NA	NA	L3-L4 lamina
37	2007	Lam *et al*[32]	63 M	L4-S1	Chronic LE pain and paresthesia, claudication	1 yr	Y	N	NA	N	NA	NA	NA	L4-L5 lamina/ fusion

38	2007	Suk *et al*[33]	55 M	L4-L5	Back pain, LE weakness and paresthesia, fever	1 wk	N	N	Alcohol	Y	Hypo	Hetero	Y	L4-L5 lamina/ fusion
39	2008	Fontenot *et al*[34]	85 F	L3-L4	Low back pain	2 mo	N	N	Diuretics	Y	NA	Hyper	NA	Prednisone and colchicines
40	2009	Chan *et al*[35]	76 M	T8, T10	LE weakness		Y	Y	NA	Y	Iso	Hetero	NA	Medical NOS
41	2009	Nygaard *et al*[36]	75 M	L4-L5	Low back pain, fever	5 d	Y	N	NA	Y	NA	NA	NA	NA
42	2009	Tsai *et al*[37]	64 F	T8-T9	Fever, low back pain, LE weakness	1 d	N	N	DM, RI	N	Hypo	Iso	Y	T8-T9 discectomy and partial corpectomy
43	2010	Coulier *et al*[38]	62 F	C6-C7	Neck pain	NA	N	Y	NA	Y	NA	NA	NA	NA
44	2010	Ko *et al*[39]	63 M	L5-S1	Low back pain	2 mo	N	N	NA	Y	Hypo	Hypo	Y	Lamina
45	2010	Murphy *et al*[40]	82 M	NA	Back pain	3 mo	N	Y	NA	NA	NA	NA	NA	NA
46	2010	Ntsiba *et al*[41]	43 M	T9-T10	Spastic paraplegia	6 mo	Y	Y	Alcohol	NA	Hypo	Hyper	NA	T10 lamina
47	2010	Samuels *et al*[42]	75 M	L5-S1	Low back pain, radiculopathy, b/l groin pain	Acute	Y	N	DM, RI, arthritis	NA	NA	NA	NA	Steroid injection and allopurinol
48	2011	Ibrahim *et al*[43]	70 F	T1-T2	UE and LE weakness	1 yr	Y	N	RI	Y	Hypo	Hyper	NA	Lamina/fusion
49	2011	Levin *et al*[44]	34 M	T2-T5	Paraplegia	Acute	Y	N	RI, DM	Y	NA	NA	NA	Lamina
50	2011	Thavarajah *et al*[45]	57 M	C1-C2	Neck pain, UE and LE tingling	1 yr	Y	N	NA	NA	NA	NA	NA	C0-C6 fusion
51	2011	Tran *et al*[46]	73 M	C1-C2	CN IX, X, XII palsies, fever, cough,	3 d	Y	Y	RI	Y	NA	Hetero	NA	Allopurinol, rasburicase
52	2012	Federman *et al*[2]	66 M	C4-C6	Neck pain	4 mo	N	N	DM	Y	Hypo	Hetero	NA	Allopurinol, colchicine, narcotic analgesics
53	2012	Hasturk *et al*[4]	77 F	L4-L5	Low back pain, radiculopathy	5 mo	N	N	NA	N	Hypo	Hypo	Y	Surgery
54	2012	Sakamoto *et al*[3]	69 M	L1-L2	Back pain, radiculopathy	Acute	N	N	Heart Failure	Y	Hypo	Hypo	Y	Medical NOS
55	201	Yamamoto *et al*[47]	58 F	C4-C7	Malaise, fever, back pain	3 yr	N	Y	Arthritis, RI	Y	NA	NA	NA	Prednisolone, allopurinol, benzbromarone
56	2012	Lu *et al*[48]	29 M	L4-S1	Severe pain, paresthesia, acratia of LLE	3 yr	Y	Y	Chronic alcohol abuse, chronic gout	Y	Hypo	Hypo	NA	L4-L5/L5-S1 decompression/ fusion
57	2012	Sanmillan *et al*[49]	71 M	C3-C4	Progressive Quadriparesis	4 mo	Y	Y	Hypertension, dislipidemia	Y	Hypo	Hyper	NA	C3-C4 micro-discectomy/ fusion
58	2013	Wendling *et al*[50]	54 M	C5-C6	Inflammatory neck pain and cervicobrachial neuralgia	Acute	Y	N	Hyperch-olesterolemia	Y	Hypo	NA	Y	Colchicine
59	2013	Wendling *et al*[50]	52 F	Lumbar posterior facet joint	Low back pain	NA	N	Y	Polychondritis	N	NA	NA	NA	Surgery
60	2013	Wendling *et al*[50]	72 M	C5-C6	Acute neck pain, knee arthritis	Acute	Y	N	Hypertension	Y	NA	NA	NA	Colchicine
61	2013	Wendling *et al*[50]	65 M	L4-L5	Inflammatory low back pain	Acute	Y	Y	Cardio-myopathy, hypertension	Y	NA	NA	NA	Colchicine
62	2013	Wendling *et al*[50]	87 M	L3-L5	Inflammatory low back pain	Acute	N	Y	Hypertension, heart failure, chronic kidney failure	Y	Hypo	NA	Y	Colchicine
63	2013	Komarla *et al*[51]	69 F	L3-S1	Back pain, fever	Acute	N	Y	Alchohol abuse, chronic low back pain	N	NA	Hyper	NA	Allopurinol, colchicine, glucocorticoids
64	2013	de Parisot *et al*[52]	60 M	C1-C2	Walking disorders, urinary and bowel incontinence	6 mo	Y	Y	NA	Y	Hypo	Hyper	Y	C1-C2 Arthrodesis
65	2013	Kwan *et al*[53]	25 M	T9-T10, L3-S1	Pain, swelling, and decreased ROM in multiple joints	1 wk	N	Y	CKD	Y	Hypo	Hetero	NA	Prednisone, allopurinol

66	2013	Yoon *et al*[54]	64 M	T5-T7	Weakness B/L LE, back pain rad to left anterior chest, paraparesis	Weakness: 1 wk; back pain 1 mo	Y (8 yr ago)	Y	Acute gout arthritis 8 yr prior	Y	Hypo	Hetero	Y	T5-T7, laminectomy, facetectomy, pedicle screw fixation w/PL fusion
67	2013	Jegapragasan *et al*[55]	24 M	L4-S1	Progressively worsening LBP w/rad, weakness of RLE, fever	3 yr LBP rad lat thight	Y (4 yr Hx)		4 yr Tophaceous gout, CKD, 3 yr LBP, rad pain down later thigh (Rt > Lf)	Y	Iso	Hetero	NA	Decompressive laminectomy L4-S1, resection of intraspinal canal and perineural lesion; post-op: colchicine, allopurinol, brief burst of prednisone
68	2014	Cardoso *et al*[56]	69 W	L4-5, SI joints	LBP rad to buttocks and hips, low fever	NA	N	Y	Constrictive pericarditis, chronic renal insufficiency, HTN, DM	Y	Hypo	Iso	Y	Colchicine, allopurinol

M: Male; F: Female; Sx: Symptoms; Hx: History; RT: Renal transplant; Hi: High; R: Right; LE: Lower extremity; RI: Renal insufficiency; BBD: Bowel/bladder dysfunction; Y and N: Yes and no; Hypo: Hypointense; Iso: Isointense; Hyper: Hyperintense; Hetero: Heterointense; UE: Upper extremity; Lamina: Laminectomy; Tx: Treatment; Gad: Gadolinium.

Table 2 Clinical features

No. of patients	
Localized back/neck pain	47
Spinal nerve root compression, in general	14
Radicular pain	6
Radiculopathy NOS	8
Spinal cord compression, in general	38
LE weakness	19
Quadriparesis	6
Claudication	5
Paraplegia	4
BBD	3
Myelopathy NOS	1
Cranial nerve palsy	1
Atlanto-axial subluxation	2
Fever	13

NOS: Not otherwise specified; UE: Upper extremity; LE: Lower extremity; BBD: Bowel/bladder dysfunction.

Table 3 Anatomic location of spinal gout

No. of patients	
Lumbar spine	38
Thoracic spine	15
Cervical spine	15
Not mentioned	1
Total[1]	68

[1]Patient No. 65 had gout in two sites, thoracic and lumbar regions.

displayed bony erosion only, 5 (7.4%) demonstrated lytic lesions, and 2 (2.9%) were unremarkable.

Treatments

Forty-five (66.2%) patients had surgical treatment. Thirty-five (51.5%) patients had laminectomies, 8 (11.8%) of whom also had fusions with laminectomies, 7 (10.3%) had surgeries not otherwise specified, 1 (1.5%) had a vertebrectomy, 2 (2.9%) had discectomies with partial corpectomies. Twenty (29.4%) received medical treatment alone and 3 (4.4%) did not report any treatment (Table 6).

Table 4 Laboratory studies

No. of patients	
Elevated uric acid	45
Elevated ESR	17
Elevated CRP	19
Renal insufficiency	11
Leukocytosis	9
Anemia	5

ESR: Erythrocyte sedimentation rate; CRP: C-reactive protein.

Case illustration

A 58-year-old female presented with a chief complaint of low back and radicular pain over left L4, 5 dermatomes that had been progressively worsening over a four-month duration to the point where she was unable to walk. The patient denied any saddle paresthesia or change in bowel and bladder function. She has a history of cardiovascular disease, chronic kidney disease (stage Ⅰ), type Ⅱ diabetes mellitus, hypertension, obesity, and obstructive sleep apnea. The patient also described an acute gouty arthropathy that was diagnosed in her right hand about 4 mo prior for which she was taking colchicine. An inflammatory workup was ordered which showed CRP of 3.58 (n < 1.0), ESR 25 (0-20), WBC 6.2 (4.0-10.0), uric acid 11.4 (2.5-6.8); HLA-B27, anti-DNA, Rheumatoid factor, and complement labs were negative.

Plain radiograph of the lumbar spine was unremark-

Table 5 Imaging studies

No. of patients	
X-ray	
Not performed	37
Spondylosis/-listhesis	12
Bony erosion	8
Unremarkable	6
Degenerative changes	5
CT	
Not performed	35
BE and HDA	13
BE only	13
Lytic lesions	5
Unremarkable	2
MRI	
T1	
Not reported	28
Hypointense	31
Isointense	8
Heterointense	1
T2	
Not reported	24
Hypointense	18
Heterointense	12
Hyperintense	11
Isointense	4
Gadolinium enhancement	
No	36
Yes	32

BE: Bony erosion; HAD: High density attenuation; CT: Computed tomography; MRI: Magnetic resonance imaging.

Table 6 Treatment

No. of patients	
Laminectomy only	24
Nonsurgical treatment	20
Surgery not specified	7
Laminectomy and fusion	5
Not reported	3
Fusion only	2
Laminectomy and facetectomy	3
Laminectomy and facetectomy and fusion	1
Vertebrectomy	1
Discectomy and partial corpectomy	2
Total	68

able. Plain radiograph of the right hand showed osseous erosive changes at the 4th finger distal interphalangeal (DIP) joint (Figure 1). MRI showed intraspinal extradural lesion causing spinal canal stenosis at L4-S1 (Figure 2). A CT showed that the lesion was calcified with erosive changes noted at the left L4-5 facet joint and L 4 lamina (Figure 3). The patient was treated with L4-S1 decompression, instrumentation and fusion. The surgical microscope was used during excision of the intraspinal lesion, which appeared chalky white, non-adherent and easily pealed off the thecal sac without sustaining dural tear (Figure 4). Postoperatively the patient noted significant improvement in both low back and radicular pain. The patient received allopurinol treatment for gout and remained asymptomatic at the last follow up two years after the index procedure.

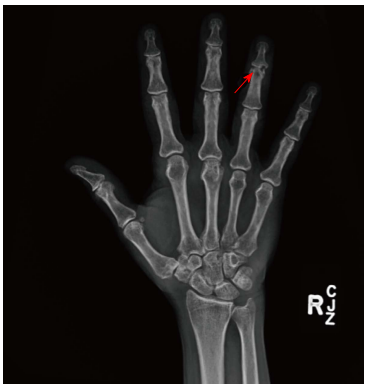

Figure 1 Plain radiograph anteroposterior view right hand showed osseous erosive changes at the 4th finger distal interphalangeal joint (arrow).

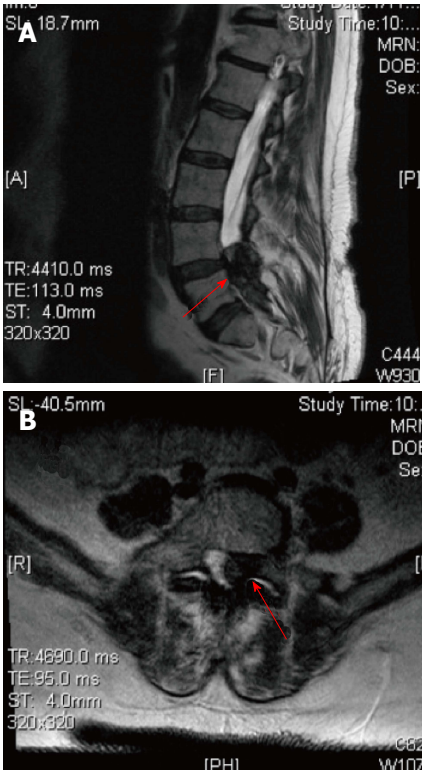

Figure 2 T2 weighted magnetic resonance imaging scan mid sagittal (A) and axial (B) showed intraspinal extradural hypodense lesion causing spinal canal stenosis at L4-S1.

DISCUSSION

Gout is a common form of inflammatory arthritis caused by the deposition of MSU crystals in synovial joints that result into erosion and joint damage. Soft tissue masses of MSU crystals known as tophi are usually found in the hand and extensor surface of the forearm[4,32,57]. Tophi are seen in patients with long-standing gout, but can also be one of the first symptoms amongst a cluster of metabolic disorders leading to hyperuricemia, especially

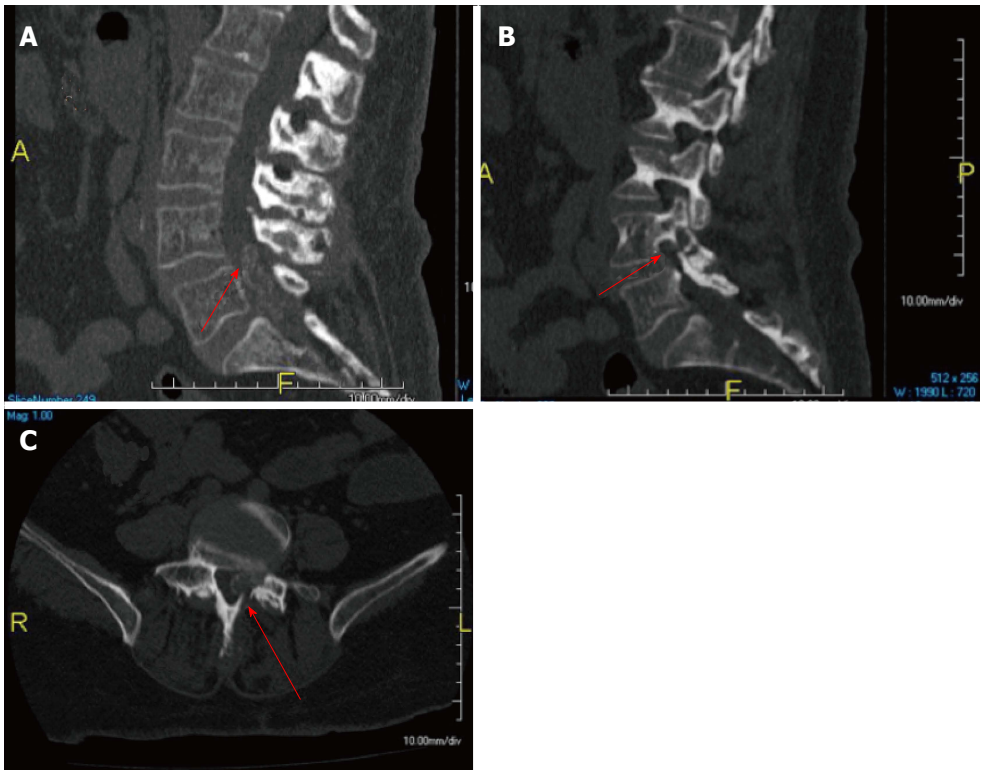

Figure 3 Computed tomography scan mid sagittal (A), left parasagittal (B), and axial (C) views showed the intraspinal lesion was calcified with erosive changes at the left L4-5 facet joint and L4 lamina (arrows).

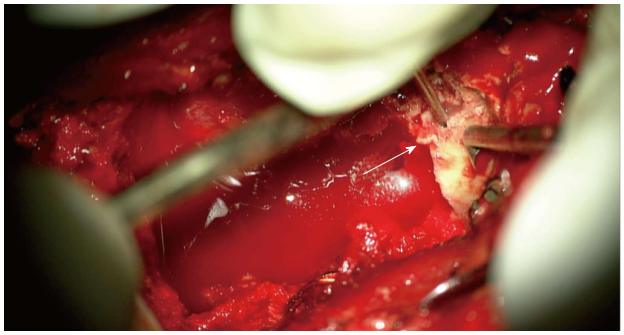

Figure 4 Intraoperative photograph taken by the surgical microscope showed a well-demarked chalky white tophous lesion (arrow).

among those with long-standing renal impairment[2,33]. Tophi are a common manifestation of gout, but spinal manifestations are considered rare. Recent research by de Mello *et al*[1], however, suggests that tophi in the axial skeleton may be more prevalent than first suspected.

Although no studies have been able to conclude the exact mechanism for axial involvement in gout, the likely theory is, as gout usually involves joint spaces, facet joint may be the initial deposition location for MSU crystals. Another theory is based on the fact that high uric acid and other inflammatory markers are often elevated in gout. This increase in uric acid in the blood could signal a corresponding increase in cerebrospinal fluid (CSF) leading to the obstruction of the canal or foramen.

Literature review showed that the lumbar spine was the most commonly involved region followed by thoracic and cervical regions. The most common clinical presentation was back pain associated with lumbar radiculopathy, or neurogenic claudication. The most frequent laboratory finding was hyperuricemia defined as uric acid above 7 mg/dL. Renal insufficiency was also found in many patients. Plain radiograph findings are usually non-specific. The most consistent image findings of the intraspinal extradural tophi were hypointense signal on the T1-weighted MRI and heterointense signals on the T2-weighted MRI. Spinal gout is usually diagnosed with cytological or histopathological studies. However, for patients treated with surgery, a pasty chalk-white mass are usually present. Clinical presentations and radiological findings of spinal gout are often non-specific and one has to consider the differential diagnoses of intraspinal extradural mass. The most frequent etiology with similar clinical presentations and imaging findings is herniated disc. Other causes include synovial cyst, tumor, epidural abscess, arteriovenous malformation.

Pharmacotherapy for spinal gout is the same as those used for gout involving typical joints. Acute gouty attack is most often treated with nonsteroidal anti-inflammatory drugs (NSAIDs), such as, naproxen or indomethacin. In patients with chronic kidney disease, duodenal or gastric ulcer, heart disease or hypertension, NSAID allergy, or anticoagulant treatment, colchicine is an alternative treatment. While NSAIDs and colchicine are effective in symptomatic reduction during an acute attack, they do not prevent the development of bony

erosions or tophi deposits in tissues. To prevent further gouty attack, maintenance medications are often prescribed with the goal of keeping uric acid level less than 6 mg/dL. Xanthine oxidase inhibitors, such as allopurinol, febuxostat, and oxypurinol, are the first line choices for reduced production of uric acid. Allopurinol can precipitate gouty attack or worsen current attack, thus, it is used for maintenance after acute attack has resolved. Uricosuric agents, such as, probenecid and sulfinpyrazone, are second line prophylactics aimed to increase uric acid excretion since decreased uric acid excretion is responsible for 85% to 90% of primary or secondary hyperuricemia[58].

Surgical interventions may be needed if patient has symptoms of spinal cord or nerve root compression. The mainstay of surgical treatment is decompression and excision of the tophi. The role of fusion at the time of the decompression remains controversial. The need for fusion is influenced by symptomatic preoperative instability as evidenced by dynamic radiographs, erosion of the facet joint seen on CT scan, or intraoperative instability that may be created by iatrogenic resection of spinal structures such as the pars interarticularis or the facet joints.

Although this article provides a broad overview of cases involving spinal gout since January 2000, there are some limitations. The absence of certain information, such as the post-treatment outcomes, limited the depth of our analysis in certain cases. Furthermore, the literature review could not always account for individual variation among the 68 cases reviewed including the particular method of diagnosis, which was not standardized across all patients included in the study. In addition, the individual articles did not provide information regarding prior uric acid lowering treatments, which could possibly inflate the number of spinal gout cases with normal uric acid levels.

The majority of clinical features for spinal gout such as back pain and neurological symptoms are nonspecific. Thus, one must rule out other common diagnoses, such as disc herniation, tumor, infection prior to diagnosing a patient with spinal gout. Laboratory study indicative of gout is elevated uric acid levels. In this literature review, the majority of the cases utilized MRI as the radiological study of choice in detecting spinal gout. While MRI was the major non-invasive diagnostic method, all suspicious findings on imaging required surgical sampling for pathological confirmation of negatively birefringent MSU crystals presence.

COMMENTS

Background

Gout is a common inflammatory arthritis with an increase in prevalence over the last 20 years. It currently affects over 8 million Americans. The primary aim of this review is to summarize the most common presenting features, imaging findings, and treatment choices based on the 68 published cases.

Research frontiers

Literature review showed that the lumbar spine was the most commonly involved region followed by thoracic and cervical regions. The most common clinical presentation was back pain associated with lumbar radiculopathy, or neurogenic claudication. The most frequent laboratory finding was hyperuricemia defined as uric acid above 7 mg/dL.

Innovations and breakthroughs

Traditionally gout thought of as a rare problem characterized by a sudden, severe attacks of pain, redness and tenderness in joints, often the joint at the base of the big toe. Recent studies suggest that axial gout may be more prevalent than suspected. Spinal gout can affect the facet joint, laminae, ligamentum flavum, as well as the epidural spaces.

Applications

The majority of clinical features for spinal gout such as back pain and neurological symptoms are nonspecific. Suspicious findings on MRI imaging required surgical sampling for pathological confirmation of negatively birefringent monosodium urate crystals presence.

Peer-review

It is a good review concerning the spinal gout consisting of the symptom and signs, treatment option and lab data analysis.

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