Axial Gouty Arthropathy

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Abstract: Gouty involvement of the spinal column is not as rare as generally perceived. Tophaceous gout involving the spinal column is a well-documented cause of myelopathy and frank cord compression. It takes several years of gout before bony destruction is radiologically apparent. If erosive or tophaceous gout is present, magnetic resonance imaging signal enhancement offers diagnostic guidance. Non-tophaceous gout of the spine may also show signal enhancement consistent with inflammation. The sequelae of cord compression can be reversed with timely surgical intervention and maintenance of uric acid–lowering therapy; in some cases, medical therapy alone can reverse the findings of radiculopathy. Growing evidence suggests that the tangled web of hypertension, diabetes, and atherosclerotic disease are risk factors for gout and hyperuricemia and may, in fact, be the result of higher than physiologically tolerable levels of uric acid in humans. Here, 52 additional cases to the 73 collated by Hou et al (Surg Neurol. 2007;67:65–73), reinforce that gout is a major contender on the differential diagnosis of back-related presentations in patients at high risk for gout. The pervasiveness of cardiovascular disease and chronic back pain warrants a closer look into a possible occult contributor to the prevalence of chronic back pain: gout.

Key Indexing Terms: Gout; Hyperuricemia; Cord compression; Spinal; Vertebral; Uric acid. **[Am J Med Sci 2009;338(2):140–146.]**

BACKGROUND

Gouty involvement of the spinal column is not as rare as generally perceived with tophaceous gout of the spine being well documented in presentations of myelopathy and frank cord compression. Hou et al[1] undertook a literature review collating disparate case series and reports revealing the striking prevalence of such spinal disease. Here, we add 52 additional cases to the 73 catalogued by Hou et al; reinforcing that gout is a major contender on a differential diagnosis for back-related presentations. We also review the associations of uric acid to cardiovascular morbidity as a hypothetical explanation that speaks to the prevalence of axial gouty arthropathy.

Hyperuricemia and Gout

Monosodium urate (MSU) crystal disease, or gout, is a metabolic disorder of purine nucleotide degradation and is defined by the presence of negatively birefringent crystals under polarized light found in body tissue or joint spaces. The limit of uric acid solubility in serum is 7 mg/dL at normal body temperature. At levels above this, serum experiences supersaturation of uric acid and crystals are precipitated, resulting in an increased risk for nephrolithiasis and inflammatory crystal arthritis. A high serum uric acid level, hyperuricemia, without overt clinical manifestations is not generally considered to be gout.

Gout may be the result of a primary or secondary hyperuricemia. Primary hyperuricemia is a consequence of congenital enzymatic defects, which are of several types. Primary hyperuricemia usually becomes apparent in the third or fourth decade with recurrent gouty arthritis and renal stone formation. In contrast, secondary hyperuricemia results from a preexistent or acquired condition (ie, renal failure, hypertension, or tumor lysis), which results in increased serum uric acid levels. Epidemiological studies suggest that the prevalence of secondary gout is around 1% in the United States and is increasing.[2] Secondary asymptomatic hyperuricemia and gout are linked to the coexistence of hypertension, hyperlipidemia, insulin resistance, chronic kidney disease, cardiovascular disease, dietary excess, alcohol consumption, and obesity. The presence of gout should initiate an investigation to identify and correct associative causes.[3]

Gout and the Musculoskeletal System

Gout is commonly accepted as a peripheral joint disease of males. Indeed, at younger ages, gout is more apparent in males but this distribution is equalized between the sexes with age and the onset of menopause.[2] Initial attacks of gout and the development of tophi—accumulations of MSU deposition—commonly manifest peripherally in the first metatarsal phalangeal joint (podagra), ankle, knee, or elbow. However, gout is a frequent and adept mimic of many diseases. For example, gout in its polyarticular form has become recognized as a common occurrence in rheumatologic practice with many patients having been mistakenly treated for rheumatoid arthritis.[4]

Gouty Spinal Involvement: Under-recognised in Prevalence

Axial disease has been perceived to be quite rare; however, a simple literature search is rife with cases of spinal gout, which begs the true prevalence of spinal disease. Table 1 catalogues 125 cases of spinal gout. A Pubmed query in September 2008 crossing "gout" with commonly associated joints, such as "knee," "hand," "foot," "toe," and "elbow" yielded 348, 326, 252, 95, and 63 entries, respectively. From this perspective, spinal gout makes a pretty strong showing.

Several retrospective investigations suggest that gouty involvement of the spine is overlooked. Alarcon-Segovia et al[5] found, of 143 patients with gout, 17% had axial involvement specifically of the sacroiliac (SI) joints. More recently, Bhandaru et al[6] found that of 64 computed tomography scans of patients with gout that were available for retrospective review, 17% had changes consistent with gouty involvement in SI joints and vertebral spine with multiple levels of vertebral involvement in the majority (67%) of patients. Lumbar involvement predominated at 78% with the most common finding being facet joint erosions, 30% had thoracic

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TABLE 1. List of patient cases on spinal gout in the literature presented chronologically

Patients	Author	Symptoms	Location
1	Alarcon-Segovia et al, 1973	Chronic low back pain, cord compression	C6-C7, SI joints
2	Koskoff et al, 1953	LE paralysis, BBD	T9-T11
3	Levin et al, 1956	Postmortem finding	L-spine
4	Hall et al, 1960	Postmortem finding	L4-S1
5	Lievre et al, 1961	Radicular pain	C3
6	Vinstein et al, 1972	Neck pain	C3-C4
7	Litvak et al, 1973	LE weakness	L3-L5
8	Reynolds et al, 1976	Paraparesis	L4-L5
9	Burnham et al, 1977	LE weakness	L4-S1
10	Wald et al, 1979	Back pain	L3-L5
11	Magid et al, 1981	Back pain, LE weakness, BBD	T2-T9, C7
12	Sequueira et al, 1981	Quadriparesis, BBD	C3-C4
13	Lagier et al, 1983	Chronic back pain	L2
14	Leaney et al, 1983	Chronic back pain, LE weakness, fever	T7-T11
15	Miller et al, 1984	Neck pain, UE radicular pain	C5-C7
16	Jacobs et al, 1985	Quadriparesis	C1-C2
17	Varga et al, 1985	Chronic back pain, radiculopathy	L4-L5
18	Alarcon et al, 1987	Neck pain	C6-C7
19	De Vries et al, 1987	Claudication, radiculopathy	C1-C2
20	Downey et al, 1987	Chronic LE weakness, BBD	T1
21	Van De Laar, 1987	Chronic LE weakness, myelopathy	C-spine
22	Arnold et al, 1988	Chronic back pain and calf pain	L4
23	Das De, 1988	Back pain, fever	L5-S1
24	Sabharwal et al, 1988	Chronic neck pain	C2-C5
25	Leventhal et al, 1990	Acute back pain, fever	L-spine
26	Vervaeck et al, 1991	Acute back pain, paraparesis	L2-L3
27	Vaccaro et al, 1993	Quadriparesis	C2-C6
28	Murshid et al, 1994	Chronic UE pain, quadriparesis	C1-C2
29	Yasuhara et al, 1994	Acute thoracic myelopathy	T6-T7
30	Fenton et al, 1995	Chronic back pain, radicular pain	L4-L5
31	Fenton et al, 1995	Chronic back pain	L4-L5
32	Staub-Schmidt, 1995	Lower back pain, *Staphylococcus* sepsis	L5
33	Bonaldi et al, 1996	Acute back pain, fever	L3-L5
34	Duprez et al, 1996	Quadraplegia	C3-C6
35	Ko et al, 1996	Chronic back pain, claudication	L2-L3
36	Miller et al, 1996	Back pain, LE weakness	L2-L5
37	Dhote et al, 1997	New onset LE weakness and pain	T4-T9
38	King et al, 1997	Acute back pain, radicular pain	L5-S1
39	Clerc et al, 1998	LE weakness	L4-L5
40	Gines, 1998	Acute back pain, fever	L1-L5
41	Giuliano et al, 1998	UE radicular pain	C6-C7
42	Peeters et al, 1998	Chronic back pain, radicular pain	L4-L5
43	Pfister, 1998	Acute back pain, BBD, LE weakness	T8-T9
44	Bret et al, 1999	Cord compression	T2-T9
45	Hausch et al, 1999	Chronic back pain, fever	T4, T7, L4, L5
46	Kaye et al, 1999	Chronic back pain, LE weakness, BBD	T8
47	Marsaudon et al	Back pain	L3-L4
48	Kao et al, 2000	LE weakness	T10-T11
49	Mekelburg et al, 2000	Chronic back pain	L2-L3
50	Paquette et al, 2000	Chronic back pain, radicular	L3
51	Thornton et al, 2000	Acute back pain	L3-L4
52	Barrett et al, 2000	Acute back pain, fever, radicular pain	L5-S1
53	St George et al, 2001	Acute LE weakness, BBD	T1-T2
54	Wang et al, 2001	Acute LE weakness	T9-T10

(*Continued*)