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Gout in the spine☆

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ABSTRACT

Axial gout can affect all segments of the spine. It is manifested as back pain, as pain associated with neurological symptoms, and as neurological impairment without pain in 17.9%, 75.8% and 4.2% of cases, respectively. These manifestations were the first presentation of gout in many patients. Although X-rays as well as computed tomography and especially magnetic resonance scans can be very suggestive, histopathological, cytological and crystal analyses are the diagnostic gold standard. In most cases involving neurological manifestations, the patient underwent surgery, leading to satisfactory results. There are, however, some reports of full recovery following the usual clinical treatment for gout, suggesting that such treatment may be the initial option for those subjects with a history of gout and radiological findings of axial involvement.

Gota axial

Palavras-chave:
Gota
Coluna vertebral
Tofo
Radiculopatia

RESUMO

A gota axial pode afetar todos os segmentos da coluna vertebral. Ela se manifesta como dor nas costas, dor associada com sintomas neurológicos, e como comprometimento neurológico sem dor em 17,9%, 75,8% e 4,2% dos casos, respectivamente. Essas manifestações foram a primeira apresentação da gota em muitos pacientes. Embora radiografias, bem como tomografia computadorizada e especialmente ressonância magnética, possam ser muito sugestivos, análises histopatológicas, citológicas e pesquisa de cristais são o padrão ouro de diagnóstico. Na maioria dos casos que envolveram manifestações neurológicas, o paciente foi submetido à cirurgia, levando a resultados satisfatórios. Há, no entanto, alguns relatos de recuperação total após o tratamento clínico habitual para gota, o que sugere que esse tratamento pode ser a opção inicial para os indivíduos com histórico de gota e sinais radiológicos de envolvimento axial.

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Introduction

Spinal gout was first described by Kersley et al.[1] in 1950, and in 1953 Koskoff et al.[2] reported the first case of myelopathy due to gout. Since then, several cases of spinal gout have been reported, with manifestations ranging from an asymptomatic clinical picture to serious complications, such as paraplegia and quadriparesis (see Table 1 with case reports cited). This study reviews the literature related to clinical manifestations, diagnosis and treatment of spinal involvement due to gout.

Methods

A bibliographical search for the terms *spine, gout, tophus,* and *myelopathy* was carried out on PubMed and Medline, and a selection of articles describing spinal gout, including case reports, letters to the editor, radiological findings, systematic reviews and observational trials was made. Cross-referencing cases among these reports, that were absent in initial search, were also included in the current review. The final number of articles was 94, and a total of 113 subjects were included in these reports. Our search also found two larger studies regarding axial gout and radiological image, one with a retrospective design and the other one being a prospective study. No previous reviews regarding this subject included these two studies, and neither had such a comprehensive search for case reports.

Results

Clinical manifestations

The mean age of the 113 patients was 60.3 ± 14.4 years ranging from 17 to 85 years,[3,4] and 70.8% were male. A previous history of gout was observed in 62 patients (65.9% of the reports that mentioned this information), and 31 of them were tophaceous. There was no reference to a history of hyperuricemia and/or gout in 19 (16.8%) patients.

Serum uric acid was measured at the diagnosis in 69 patients and 48 (69.6%) presented with high levels. Renal insufficiency was reported in 25 cases (22.1%) and a previous history of renal transplant in another 7 (6.2%).[4-6] Alcohol use and diuretic regimen was reported only in 7 (6.2%) and 12 (10.6%) cases, respectively.

Spinal involvement was the initial manifestation of gout in 28 patients (24.8%). All spinal segments were affected: the lumbar spine in 66 of the 113 patients (58.4%); the cervical spine in 28 (24.8%); and the thoracic spine in 24 (21.2%). The involvement of S1 was observed in 15 cases (13.3%), associated with lumbar spine lesions in 13 (86.7%) of them. Two patients (1.8%) had both cervical and thoracic lesions,[7,8] and another four (3.5%) had simultaneous thoracic and lumbar involvement.[5,9-11]

Gout may affect any spinal structure, such as intervertebral discs, facet joints, laminae, vertebral bodies, pedicles, ligamentum flavum, filum terminale and the soft tissues adjacent to the spinal column.[12]

Neurological symptoms were observed in 88 patients (77.9%), and there was an association with cervical, thoracic or lumbar pain in 80 (90.9%) of these cases. Pain with no neurologic symptom was reported in 23 patients (20.4%), 3 (2.7%) in the cervical spine, 1 (0.9%) in the thoracic spine, 20 (17.7%) in the lumbar spine (one case with both cervical and lumbar pain, and one report with both lumbar and thoracic involvement) and 1 (0.9%) with pain over the sacrum. Two (1.8%) patients were asymptomatic and were only diagnosed on autopsy (Table 2).[13,14]

Radiculopathy (motor dysfunction or dysesthesia along the course of a specific nerve caused by compression of its root) was the most frequent neurological symptom, occurring in 39 patients (34.5%), followed by claudication in 23 (20.4%), crural paraparesis in 14 (12.4%), quadriparesis in 8 (7.1%), and paraplegia in 5 (4.4%). Atlanto-axial subluxation with the presence of tophus was observed in two cases of cervical pain leading to quadriparesis,[15,16] and in one case of multiple cranial nerves palsy.[17] Neurologic symptoms without pain were related in 8 patients (7.1%).

Bladder and/or bowel dysfunction was observed in 10 subjects, all of them presenting with other neurological manifestations, and 5 with back pain. Thirty-four (38.6%) of the 88 cases with neurological impairment exhibited acute onset of symptoms (four weeks or less prior to diagnosis).

Fever higher than 38 °C was reported in 15 patients (10.6%), all of them with elevated erythrocyte sedimentation rate (ESR) and C reactive protein; therefore, exclusion of an infectious process was mandatory. Additionally, further 12 cases without fever had elevated ESR.

Imaging studies

As observed by King et al.,[18] X-rays may be normal or reveal soft tissue edema, signs compatible with osteoarthritis (new bone formation and/or reduction of intervertebral space), clearly-defined subchondral bone cysts, erosions with sclerotic borders, erosion of the odontoid process, atlanto-axial subluxation and pathologic fracture.[18,19] Among the findings listed above, the most common were those suggestive of osteoarthritis, seen in 26 (65%) of the 40 reports in which a spinal radiography was described. Fig. 1 shows a thoracic radiography of a patient followed at our clinic due to longstanding gout and thoracic (T7) spinal involvement.

Although not frequently mentioned in the literature, computed tomography may show erosions located in facet joints[20] and damage to soft tissue with the presence of low-density nodule or mass in some cases.[21]

In magnetic resonance imaging (MRI), tophaceous gout is usually characterized by a homogeneous image with a signal ranging from intermediate to low on T1 (with the same signal intensity as muscle), and on T2, the image appears homogeneous and may show low or high intensity.[22-25] When contrast (gadolinium) is used, peripheral heterogeneous or homogeneous contrast enhancement may occur, revealing reactive vascularization.[22,26] Fig. 2 shows the MRI of the lumbar spine of the same patient described above.

The tophus does not present a characteristic image and so it is difficult to differentiate it from other types of lesion, such as neoplasms, infections and abscesses. As a result, the final diagnosis often requires histopathological or cytological analyses.

Table 1 – Spinal gout case reports and series reviewed but not cited in the text.

No.	Authors	No. of cases reported	Year	Reference
1	Adenwalla HN, Usman MH, Bagir M et al.	1	2007	South Med J. 2007 Apr;100(4):413-4
2	Arnold MH, Brooks PM, Savvas P et al.	1	1988	Aust N Z J Med. 1988 Dec;18(7):865-7
3	Barrett K, Miller ML, Wilson JT	1	2001	Neurosurgery. 2001 May;48(5):1170-2
4	Beier CP, Hartmann A, Woertgen C et al.	1	2005	J Neurosurg Spine. 2005 Dec;3(6):485-7
5	Bonaldi VM, Duong H, Starr MR et al.	1	1996	AJNR Am J Neuroradiol. 1996 Nov-Dec;17(10):1949-52
6	Burnham J, Fraker K, Steinbach H	1	1977	AJR Am J Roentgenol. 1977 Dec;129(6):1116-9
7	Cabot J, Mosel L, Kong A et al.	1	2005	Skeletal Radiol. 2005 Dec;34(12):803-6
8	Chan AT, Leung JL, Sy AN	1	2009	Hong Kong Med J. 2009 Apr;15(2):143-5
9	Chang IC	4	2005	Clin Orthop Relat Res. 2005 Apr(433):106-10
10	Clerc D, Marfeuille M, Labous E et al.	1	1998	Clin Exp Rheumatol. 1998 Sep-Oct;16(5):621
11	Das De S	1	1988	J Bone Joint Surg Br. 1988 Aug;70(4):671
12	Dhote R, Roux FX, Bachmeyer C et al.	1	1997	Clin Exp Rheumatol. 1997 Jul-Aug;15(4):421-3
13	Diaz A, Porhiel V, Sabatier P	1	2003	Neurochirurgie. 2003 Dec;49(6):600-4
14	Draganescu M, Leventhal LJ	1	2004	J Clin Rheumatol. 2004 Apr;10(2):74-9
15	Duprez TP, Malghem J, Vande Berg BC et al.	1	1996	AJNR Am J Neuroradiol. 1996 Jan;17(1):151-3
16	El Sandid M, Ta H	1	2004	Ann Intern Med. 2004 Apr 20;140(8):W32
17	Ferreira A, Silva Junior BA, Braga FM et al.	1	1989	Arq Neuropsiquiatr. 1989 Dec;47(4):479-83
18	Fontenot A, Harris P, Macasa A et al.	1	2008	J Clin Rheumatol. 2008 Jun;14(3):188-9
19	Gines R, Bates DJ.	1	1998	Am J Emerg Med. 1998 Mar;16(2):216
20	Hasegawa EM, Goldenstein-Schainberg C, Fuller R	1	2007	Rev Bras Reumatol. 2007 Jul-Aug;47(4):300-2
21	Jacobs SR, Edeiken J, Rubin B et al.	1	1985	Arch Phys Med Rehabil. 1985 Mar;66(3):188-90
22	Justiniano M, Colmegna I, Cuchacovich R	1	2007	J Rheumatol. 2007 May;34(5):1157-8
23	Kao MC, Huang SC, Chiu CT et al.	1	2000	J Formos Med Assoc. 2000 Jul;99(7):572-5
24	Kelly J, Lim C, Kamel M et al.	1	2005	J Neurosurg Spine. 2005 Feb;2(2):215-7
25	Kern A, Schunk K, Thelen M	1	1999	Rofo. 1999 May;170(5):515-7
26	Ko KH, Huang GS, Chang WC	1	2009	Arthritis Rheum. 2009 Jan;60(1):198
27	Ko KH, Huang GS, Chang WC	1	2010	J Clin Rheumatol. 2010 Jun;16(4):200
28	Ko PJ, Huang TJ, Liao YS et al.	1	1996	Changgeng Yi Xue Za Zhi. 1996 Sep;19(3):272-6
29	Lam HY, Cheung KY, Law SW et al.	4	2007	J Orthop Surg (Hong Kong). 2007 Apr;15(1):94-101
30	Leaney BJ, Calvert JM	1	1983	J Neurosurg. 1983 Apr;58(4):580-2
31	Lievre JA, Leroux-Robert J, Bacri J	1	1961	Presse Med. 1961 Jul 8;69:1525-6
32	Litvak J, Briney W	1	1973	J Neurosurg. 1973 Nov;39(5):656-8
33	Miller JD, Percy JS	1	1984	J Rheumatol. 1984 Dec;11(6):862-5
34	Niva M, Tallroth K, Konttinen YT	1	2006	Clin Exp Rheumatol. 2006 Jan-Feb;24(1):112
35	Nygaard HB, Shenoi S, Shukla S	1	2009	Neurology. 2009 Aug 4;73(5):404
36	Paquette S, Lach B, Guiot B	1	2000	Neurosurgery. 2000 Apr;46(4):986-8
37	Peeters P, Sennesael J	1	1998	Nephrol Dial Transplant. 1998 Dec;13(12):3245-7
38	Pfister AK, Schlarb CA, O'Neal JF	1	1998	AJR Am J Roentgenol. 1998 Nov;171(5):1430-1
39	Reynolds AF, Jr., Wyler AR, Norris HT	1	1976	Arch Neurol. 1976 Nov;33(11):795
40	Riddell CM, Elliott M, Cairns AP	1	2008	J Rheumatol. 2008 Oct;35(10):2076-7
41	Sabharwal S, Gibson T	1	1988	Br J Rheumatol. 1988 Oct;27(5):413-4
42	Saketkoo LA, Robertson HJ, Dyer HR et al.	1	2009	J Med Sci. 2009 Aug;338(2):140-6
43	Samuels J, Keenan RT, Yu R et al.	1	2010	Bull NYU Hosp Jt Dis. 2010;68(2):147-8
44	Sequeira W, Bouffard A, Salgia K et al.	1	1981	Arthritis Rheum. 1981 Nov;24(11):1428-30
45	Souza AW, Fontenele S, Carrete H et al.	1	2002	Clin Exp Rheumatol. 2002 Mar-Apr;20(2):228-30
46	St George E, Hillier CE, Hatfield R	1	2001	Rheumatology (Oxford). 2001 Jun;40(6):711-2
47	Staub-Schmidt T, Chaouat A, Rey et al.	1	1995	Arthritis Rheum. 1995 Jan;38(1):139-41
48	Suk KS, Kim KT, Lee SH et al.	1	2007	Spine J. 2007 Jan-Feb;7(1):94-9
49	Tkach S	1	1970	Clin Orthop Relat Res. 1970;71:81-6
50	Vaccaro AR, An HS, Cotler JM et al.	1	1993	Orthopedics. 1993 Nov;16(11):1273-6
51	van de Laar MA, van Soesbergen RM, Matricali B	1	1987	Arthritis Rheum. 1987 Feb;30(2):237-8
52	Varga J, Giampaolo C, Goldenberg DL	1	1985	Arthritis Rheum. 1985 Nov;28(11):1312-5
53	Vervaeck M, De Keyser J, Pauwels P et al.	1	1991	Clin Neurol Neurosurg. 1991;93(3):233-6
54	Vinstein AL, Cockerill EM	1	1972	Radiology. 1972 May;103(2):311-2
55	Wald SL, McLennan JE, Carroll RM et al.	1	1979	J Neurosurg. 1979 Feb;50(2):236-9
56	Wang LC, Hung YC, Lee EJ et al.	1	2001	J Formos Med Assoc. 2001 Mar;100(3):205-8
57	Yasuhara K, Tomita Y, Takayama A et al.	1	1994	J Spinal Disord. 1994 Feb;7(1):82-5
58	Yen HL, Cheng CH, Lin JW	1	2002	Acta Neurochir (Wien). 2002 Feb;144(2):205-7

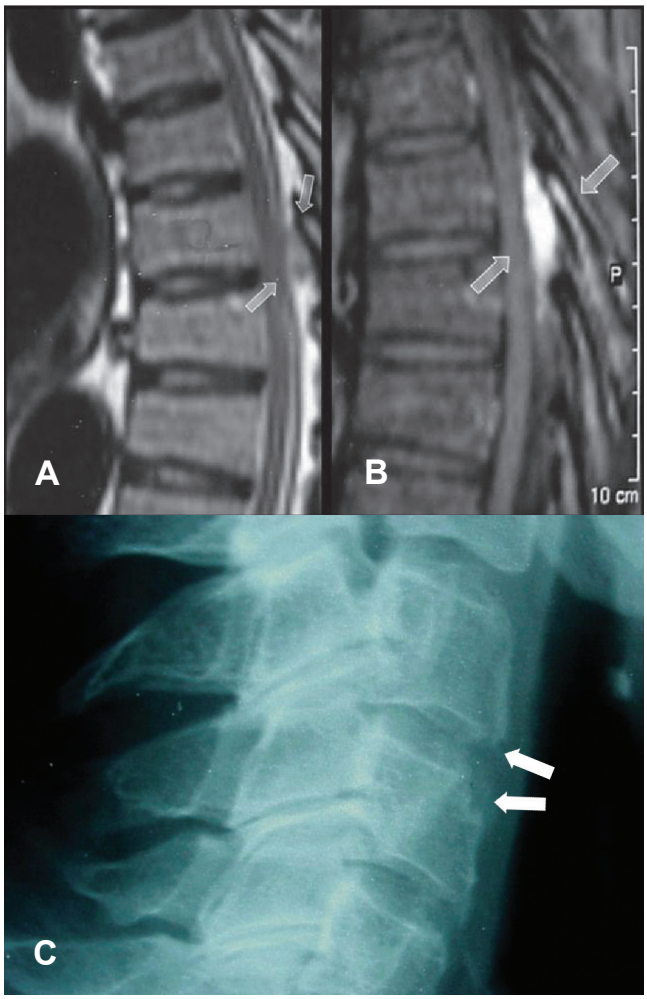

Fig. 1 – (A and B) MRI of thoracic spine on lateral view. Note the presence of a low signal lesion in T1 indicated by the arrows (A) before and (B) after contrast administration, with a homogeneous enhancement. (from: Hasegawa EM, Goldenstein-Schainberg C, Fuller R. Rev Bras Reumatol. 2007;47(4):300-2). (C) Lateral radiography of cervical spine in a patient with chronic tophaceous gout. The arrows show erosions in the anterior aspects of the vertebral bodies.

The most common findings in imaging study are listed in Table 3.

Diagnosis

In 103 of the 113 cases (91.2%), a diagnosis was achieved through cytological or histopathological studies. A histological study of the tissue removed during the surgical excision of the lesion or decompressive laminectomy was performed in 87 patients (77%). Guided puncture was done in 16 patients (14.2%), biopsy being unnecessary; and an open biopsy was performed in one (0.9%).[27] A pasty, chalky-white mass was usually observed during the surgery.

In seven patients (6.2%),[8,17,23,24,28-30] histological or cytological tests were not performed, and diagnosis was presumed based on clinical and imaging findings or on arthrocentesis of other joints. In another four cases (3.5%) diagnosis was made during autopsy,[1,2,13,14] but none of them died because of spinal involvement by gout.

In 17 patients who reported pain without neurological manifestation, guided puncture or surgical intervention was performed based on the presence of abnormalities in CT and/or MRI (mass lesion), and eventually fever and elevation in ESR at onset.

The histological description includes classic gout aspects, such as the presence of histiocytes and multinucleated giant cells and fibroblasts surrounding eosinophilic debris or amorphous material, and may contain needle-shaped crystals with negative birefringence under polarized light. Occasionally, only the negative image of the crystals is observed in the neutrophils, as they are dissolved during fixation in aqueous medium.[31-33]

Treatment

Among the 88 patients with neurological symptoms, 74 (84.1%) underwent surgery. The most common procedure was decompressive laminectomy. Full recovery of neurological manifestations was observed in 55 of 74 patients (74.3%); partial recovery followed surgery in nine, and two patients[31,34] reported no recovery after surgery, although one of them[34] improved after subsequent clinical treatment with non-steroidal anti-inflammatory (NSAIDs) and hypouricemic drugs. In another six cases there was no follow-up description and thus no data about response to surgery could be retrieved. Only one patient without neurological symptoms was managed surgically but there was no mention to outcomes. Two patients died in the post operatory status due to bronchopneumonia,[15,28] one of them had not shown any improvement, while the other was actually getting better from the neurological symptoms before the infectious complication.

Twenty-five patients (22.1%) received clinical treatment alone (NSAIDs, colchicines and oral, intravenous or epidural corticosteroid) with recovery from neurological deficits and/or pain. In three cases managed conservatively the outcome was not reported.[6,23,35] Three patients who did not undergo surgery died from bronchopneumonia,[1,9,17] one of them had been improving from the symptoms related to gout, whilst the other two had not. In eight cases there was no mention to management and outcome. Treatment outcomes are summarized in Fig. 2.

Discussion

Spinal involvement in gout arthritis is increasingly recognized as an unusual manifestation; however its prevalence is clearly underestimated because only those patients with neurological deficits and/or fever, and those who do not improve with clinical treatment are investigated with imaging and subsequent histopathological studies to confirm the diagnosis.

Although considered to be a rare manifestation of gout, mainly because most of the cases reported are of symptomatic clinical scenarios (98.2% of the 113 patients reviewed in this article), some evidence point out to the fact that spinal

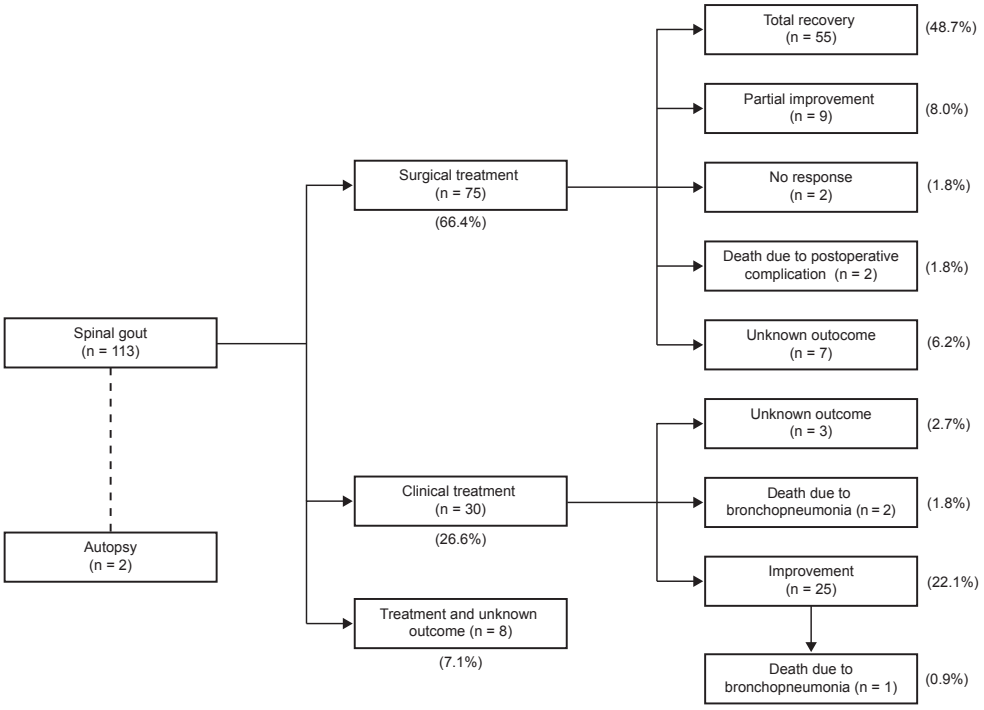

Fig. 2 – Treatment and outcome summary.

Table 2 – Clinical manifestations.

Symptoms	n	%
Neurologic deficit with back pain	80	
Radiculopathy	39	34.5
Claudication	23	20.4
Paraparesis	14	12.4
Tetraparesis	8	7.1
Paraplegia	5	4.4
Pain without neurologic deficit	23	20.4
Cervical	3	2.7
Thoracic	1	0.9
Lumbar	20	17.7
Sacral	1	0.9
Neurologic deficit only	8	7.1
Unknown*	2	1.8

* Diagnosis made by autopsy

Table 3 – Imaging findings in spinal gout.

Method	Findings
X – Ray	Osteoarthritic changes: osteophytes, subchondral cysts and bony sclerosis
CT	Osteoarthritic changes
	Low-density mass
	Facet joint erosion
MRI	T1 – homogeneous, low to intermediate signal
	T2 – homogeneous, low to high signal
	Contrast (Gadolinium): homogeneous, peripheral enhancement
	Normal bone marrow signal of the adjacent vertebrae

CT, computed tomography; MRI, magnetic resonance imaging.

alterations in gout might be much more common than previously thought. Konatalapalli et al.[36] retrospectively reviewed 64 CT images of the spine from patients with gout, and found out that 14% of them presented features of spinal gout. The same research group performed a prospective study in which 48 subjects with a diagnosis of gout had a spinal CT scan.[37] Thirty-five percent of the patients had CT scan evidence of spinal gout erosions or tophi. Since these two studies were not case reports and therefore did not mention any individual clinical info on each patient, they were not included in our analysis.

Axial gout should be considered in the differential diagnosis of patients with previous diagnosis of gout or a history of hyperuricemia who present symptoms suggesting spinal cord involvement and lumbar or cervical pain. Although usually not mentioned in many case reports, risk factors for the development of acute gout such a as renal failure, drugs (diuretics, low-dose aspirin), diet, alcohol intake, and infection should also be considered for the presumptive diagnosis of spinal gout.

The mechanism associated with axial gout is not yet clear. However, it is assumed that the same factors leading to the peripheral picture such as pH, temperature, trauma and joint degeneration are involved in crystal deposits.[33,38] Finally, the presence of spinal osteoarthritis perhaps also facilitates further crystal deposition.

In patients with no history of gout or hyperuricemia, the diagnosis may be presumed from CT scan, MRI study, clinical and laboratorial findings, previous medical history, and associated risk factors. Plain radiography is a relatively limited diagnostic resource.

Histological or cytological analysis is necessary for a definite diagnosis and to exclude infectious processes or neoplasms which may have similar clinical and imaging aspects,

especially in patients without history of gout and/or those with signs and symptoms of red flags for back pain. The sample should be preserved in alcohol to prevent urate crystals dissolution.[33]

Patients without neurological involvement may initially be treated with NSAIDs, as indicated in acute gout attacks,[39,40] and subsequently with hypouricemic drugs. In the presence of neurological symptoms, clinical treatment may also be tried, as it leads to improvement in some patients. The favorable response to clinical treatment alone may suggest that the inflammatory process, rather than a compression due to tophi itself, should be the main mechanism for the development of symptoms.

In a previous review of spinal gout made by Hou et al.,[22] after an initial clinical, laboratorial and imaging evaluation, biopsy is suggested as definite diagnosis procedure. If there are progressive neurologic deficits, surgery may be preferred. If biopsy confirms the diagnosis of gout, conservative treatment may be tried, and when symptoms persist or recur after initial improvement, surgery must be performed. We recommend guided puncture rather than open biopsy in those patients without severe or progressive neurological involvement, and in those with pain as sole manifestation that did not improve with clinical treatment.

In conclusion, gout should be included in the differential diagnosis of acute spinal pain episodes, associated or not with neurological manifestations in patients with a history of gout and hyperuricemia. In the cases without or with mild to moderate neurological manifestations, we recommend guided puncture as initial diagnostic procedure, and conservative treatment with NSAIDs and/or corticosteroids. Surgery must be reserved for those patients with no improvement or those with progressive neurologic deficits, despite clinical treatment.

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