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Spinal gout diagnosis in chiropractic practice: narrative review

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Objective: *To review and summarize the recent literature, increase awareness and provide guidance for chiropractic physicians regarding the diagnosis of spinal gout.*

Methods: *A search of PubMed was undertaken for recent case reports, reviews and trials relating to spinal gout.*

Results: *Our analysis of 38 cases of spinal gout revealed that 94% of spinal gout patients presented with back or neck pain, 86% displayed neurological symptoms, 72% had a history of gout, and 80% had raised serum uric acid levels. Seventy-six percent of cases proceeded to surgery. A combination of clinical findings, laboratory tests and appropriate utilization of Dual Energy Computed Tomography (DECT) has the potential to improve early diagnosis.*

Diagnostic de la goutte spinale dans la pratique chiropratique : analyse narrative

Objectif : *Examiner et résumer la littérature récente, sensibiliser les médecins chiropraticiens et les guider dans le diagnostic de la goutte spinale.*

Méthodes : *Une recherche a été entreprise dans PubMed pour trouver des rapports de cas, des études et des essais récents concernant la goutte spinale.*

Résultats : *Notre analyse de 38 cas de goutte spinale a révélé que 94 % des patients souffrant de goutte spinale présentaient des douleurs dorsales ou cervicales, 86 % des symptômes neurologiques, 72 % des antécédents de goutte et 80 % une élévation du taux d'acide urique sérique. Soixante-seize pour cent des cas ont donné lieu à une intervention chirurgicale. La combinaison des résultats cliniques, des tests de laboratoire et de l'utilisation appropriée de la tomographie informatisée à double énergie (DECT) peut améliorer les chances d'un diagnostic précoce.*

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Conclusion: *Gout is an uncommon cause of spine pain; however, it must be considered in the differential diagnosis as outlined in this paper. Increased awareness of the signs of spinal gout and earlier detection and treatment has the potential to improve the quality of life of patients and reduce the need for surgery.*

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KEY WORDS: chiropractic, gout, spine

Conclusion: *La goutte est une cause peu fréquente de douleur vertébrale, mais elle doit être prise en compte dans le diagnostic différentiel, comme indiqué dans le présent document. Une meilleure connaissance des signes de la goutte spinale et une détection et un traitement plus précoces pourraient améliorer la qualité de vie des patients et réduire la nécessité d'une intervention chirurgicale.*

(JCCA. 2023;67(1):50-66)

MOTS CLÉS : chiropratique, goutte, colonne vertébrale

Introduction

Gout is an inflammatory arthritis caused by the deposition of monosodium urate crystals (MSU) in the synovial fluid and other tissues. Hyperuricaemia is correlated with gout incidence and recurrence.[1] Gout usually presents as monoarticular and is more likely to affect the distal joints, most frequently the first metatarsophalangeal joint, ankle, and mid-foot, however it may also affect the hands, wrists, elbows, knees, hips and spine. Polyarticular gout is typically associated with chronic gout symptoms.[2]

The prevalence of gout worldwide is estimated to be 1-4%. It is more common in men (3:1 to 10:1) and older age groups, and is increasing in many developed countries (New Zealand, Canada, United Kingdom, Korea).[3–5] In 2017, the greatest point prevalence estimates for gout were in New Zealand, Australia and the United States of America (US).[5]

In the US, general gout prevalence is 5.2% in males and 2.7% in females.[6] With age greater than 80 years the prevalence increases to 9% in men and 6% in women. Prevalence of hyperuricaemia remained stable in the US from 2007-2016. However, Chen-Xu *et al*.[7] propose that a new wave of rising obesity in younger age groups may lag a future increase in gout prevalence. It is suspected that Western diets, sedentary lifestyle, an increased frequency of obesity and hypertension are contributing factors to raised uric acid levels.

Spinal gout, once thought to be a rare condition, may be more common than previously appreciated. Spinal gout has been identified in 14 to 29% of patients with a confirmed diagnosis of extremity gout.[8–10] Prevalence may be significantly underestimated as only patients with severe symptoms, such as neurological deficit and fever, or those failing to respond to other therapies are investigated further.[11]

Advancements in imaging technologies such as Computed Tomography (CT), Magnetic Resonance Imaging (MRI) and Dual Energy Computed Tomography (DECT) have significantly improved the detection of spinal gout. DECT is an emerging technology designed to detect the presence of MSU crystalline deposits. The technology is well established for assisting with the diagnosis of extremity gout but is yet to be commonly utilized for detecting spinal gout. Several case reports summarized in our paper mention DECT as an important diagnostic tool for confirming spinal gout.[12–14]

In this paper we analyse 38 case reports of spinal gout from between 2017 and 2022. Consistent with previous reviews, a high proportion (76%) of cases of spinal gout proceed to surgery with diagnosis only confirmed after excision and lab analysis.[15–17] This highlights the need for earlier and less invasive detection methods for spinal gout, with particular application in primary care settings. In this paper we review advances in clinical diagnosis and imaging and recommend tools to improve recognition and earlier diagnosis of spinal gout.

Methods

A search of PubMed was undertaken for case reports, systematic reviews, literature reviews, observational studies,

clinical trials and radiological investigations relating to spinal gout. Keywords searched were "gout", "spinal", "axial", "vertebral", "tophaceous", "prevalence", "imaging", "case reports", and "case review". Reference lists of included and key articles were searched to identify other potentially suitable articles. Data from included studies were extracted by one review author including data such as participant characteristics (age/sex, history of back and neck pain, neurological signs and symptoms), location, history of gout, imaging and treatment (Table 1). As this is a narrative review, no critical appraisal of the included articles or confirmation of data extraction was undertaken. Cases containing incomplete data were not excluded from analysis, however when specific data was missing (e.g. laboratory findings) the case was excluded from analysis for that item only. For example, Serum Uric acid (sUA) above 6.0 mg/dL = 28 cases. Three of our 38 cases did not report sUA and were therefore excluded from our calculations resulting in the formula 28/35 (80% of cases)

There have been several comprehensive spinal gout reviews covering case reports between 1950-2017.[11,15–17] In order to avoid case report duplication with previous reviews we limited our search for case reports to the period between January 2017 and April 2022.

Results

Forty-five case reports, four systematic reviews, fourteen literature reviews and nine observational studies were identified. The systematic reviews provided case series analysis of 454 cases of spinal gout (including duplicated cases) between 1950 and 2017. After removing duplicates, Zhang *et al*.[16] found 287 cases of spinal gout for this period, however only 142 cases met criteria for further analysis. To avoid duplication, we excluded seven case reports (which appeared in previous reviews[16,18]), leaving 38 cases for inclusion in our analysis. Our case study findings are summarized in Table 1.

Our case review analysis revealed that 85% of patients diagnosed with spinal gout were male. Ninety-four percent

Table 1.

Summary of findings from case studies. From the following 38 case reports, specific facts and figures emerge on the diagnosis and treatment of spinal gout. These statistics are taken from and grouped into pertinent and clinically related data on the demographics, diagnostic imaging, anatomical location, diagnosis and the ultimate impression of gout.

Authors	Age/ Sex	Hx BP/NP	Neuro S/s	Hx Joint Pain	Location	Hx Gout	sUA >6.0 mg/dL	Imaging Findings	Tx
Wu Z *et al*. 19	48 M	8/52 sharp LBP	Y	Y	L2-3 Epidural Space	10yrs	Y	MRI - extradural mass/ lesion to the left	Surgery
Wu Z *et al*. 19	63 M	2yrs LBP	Y	NR	L4-5 Epidural Space	30yrs	Y	MRI and CT – nerve root compression, LCSS	Surgery
Sullivan *et al*.20	63 M	Weeks severe LBP	Y	Y	Lx, SIJ's	Y	Y	DECT - diffuse MSU deposition Lx Spine, sacrum and SIJ's	Medication
Wang *et al*. 21	48 M	1/52 severe LBP	N	NR	Lx Erector Spinae	N	N	MRI - large mass (intermediate attenuation)	Medication
Sanchez *et al*. 22	41 M	4/52 LBP	Y	Y	L5-S1 Facet Joints	7yrs	Y	CT - erosive changes L5-S1 facet joint with hypertrophy	Medication
Park *et al*. 12	49 M	NR	Y	NR	C1-2 Epidural Space	NR	NR	CT - erosive appearance, lytic bone lesions, DECT - extradural tophus (central-posterior to C1 body)	Deceased (before Tx)

Authors	Age/ Sex	Hx BP/NP	Neuro S/s	Hx Joint Pain	Location	Hx Gout	sUA >6.0 mg/dL	Imaging Findings	Tx
Romero *et al*. 23	82 M	Neck Pain	Y	Y	C1-2 Vertebra, transverse ligament, Epidural Space	40yrs	N	CT - rat-tooth erosions, retrodental mass arising from transverse ligament of atlas - 80% central CSS	Medication
Si *et al*. 24	49 F	2yrs severe LBP	N	NR	L3 Intradural	NR	N	MRI and CT - calcified mass causing displacement of dural sac and nerve roots	Surgery
Yip *et al*. 25	43 M	7/52 severe LBP	Y	Y	L4-5 Epidural Space	20yrs	Y	MRI with gadolinium - epidural lobulated lesion	Surgery
Martins *et al*. 26	55 M	1/52 LBP	Y	Y	L4-5 Epidural Space	8yrs	N	CT and MRI - calcified mass causing central and foraminal stenosis and compressing/ displacing the dural sac	Surgery
Emsen *et al*. 27	63 F	NR	NR	NR	C3-T1 Facet Joints	NR	NR	FDG PET/CT - multiple osteolytic lesions C3-T1 with joint erosions	Surgery
Ayoub *et al*. 28	27 M	4/52 LBP	NR	Y	L5-S1 Facet Joint Lamina, Epidural Space	5yrs	Y	CT and MRI - impingement of S1 nerve root in lateral recess	Medication
Wang *et al*. 13	32 M	acute severe LBP	N	Y	L3-S1 Facet joints	Y	Y	MRI, DECT - focal erosions at right L3-4, L4-5 and L5-S1 facet joints	Medication
Wang *et al*. 13	74 M	6/52 LBP	N	Y	L4/5 Facet Joints	N	Y	CT and MRI, DECT - erosion of L4-5 facet joint, calcified peri-articular mass, marked stenosis, compression of the L5 descending nerve root,	Medication
Matos *et al*. 29	50 F	Neck Pain	Y	N	C7 Lamina Facet Joints	N	Y	MRI - spinal canal stenosis, CT - lytic lesions of C6 spinous and posterior elements	Surgery
Mishra *et al*. 30	33 M	9Mths BP	Y	N	T9-11 Intradural, Ligamentum Flavum	NR	Y	Thoracic plain film normal, MRI - posterolateral lesion at T10-11, focal hyperintense cord signal	Surgery
Thuraikumar *et al*. 31	68 M	1yr LBP	Y	Y	L5-S1 Vertebra Anterior Longitudinal Ligament	Y	N	MRI - end-plate destruction of L5-S1, partial vertebral body destruction, cystic fluid collection, ALL spread and LCSS	Surgery
Kao *et al*. 18	38 M	8/52 BP	Y	Y	T11-T12 Epidural Space	10yrs	Y	CT - T11-12 epidural mass causing CCS and left foraminal stenosis	Surgery

Authors	Age/ Sex	Hx BP/NP	Neuro S/s	Hx Joint Pain	Location	Hx Gout	sUA >6.0 mg/dL	Imaging Findings	Tx
Chen *et al*. 32	64 M	11Mths LBP	Y	N	L4-5 Epidural space	N	N	CT and MRI - consistent with a large disc protrusion causing canal stenosis and left lateral recess stenosis	Surgery
Salazar *et al*. 33	44 M	1yr severe LBP	Y	Y	L4-5-S1 FJ Epidural space Interspinous space	NR	Y	X-Ray- lytic spondylolisthesis, MRI - facet joint and interspinous space lesion	Surgery
Zhou *et al*. 34	50 M	Neck Pain 5yrs	Y	Y	C5-6 IVD Vertebra	20yrs	Y	CT and MRI - osteolytic destruction of vertebral endplates	Surgery
Akhter *et al*. 35	26 M	NR	Y	Y	T6-8 Facet Joints Epidural space	N	Y	MRI - epidural mass right posterolateral, severe central canal stenosis, lytic/erosive lesions facet joints	Surgery
Ma *et al*. 36	45 M	2yrs LBP	Y	Y	C4-5 and L5-S1 Epidural space, Ligamentum Flavum	14yrs	Y	CT and MRI - herniated lesion with cord compression C4-5, L5-S1 LDH	Surgery
Ma *et al*. 36	61 M	2yrs LBP	Y	Y	L4-5 Facet Joints	Y	Y	CT and MRI - bony erosion	Surgery
Ma *et al*. 36	70 F	8 yrs Neck Pain	Y	Y	C3-6 Ligamentum Flavum Epidural space	Y	Y	CT and MRI - high density mass extending into spinal canal	Surgery
Ma *et al*. 36	24 M	1yr LBP	N	Y	L3-4 Facet Joint	Y	Y	CT - bony erosion, high density	Surgery
Ma *et al*. 36	78 M	N	Y	Y	C2-5 Posterior Longitudinal Ligament	Y	Y	CT and MRI – bony erosion, high density	Medication
Wan *et al*. 37	42 M	2yrs LBP	Y	Y	L4-S1 Facet Joints, Lamina	4yrs	N	MRI - end-plate erosions, Hyperintense disc signal L4-5 & L5-S1	Surgery
Xie *et al*. 38	73 M	N	Y	N	C5-6 Ligamentum Flavum	N	Y	CT - spinal stenosis due to LF ossification - left side	Surgery
Liu *et al*. 14	35 M	LBP	Y	Y	L2-5 Facet Joints Lamina Spinous Process Epidural space	Y	Y	XR - facet joint erosions L2-5, MRI and DECT - LCSS due to tophi	Surgery
Al-Jebaje *et al*. 39	26 M	NR	Y	Y	C3-T1 and T7-11 Epidural space	Y	Y	MRI - severe LCSS at C6-7 and right C7 foraminal stenosis, multilevel degeneration, spinal stenosis T12-L1	Surgery

Authors	Age/ Sex	Hx BP/NP	Neuro S/s	Hx Joint Pain	Location	Hx Gout	sUA >6.0 mg/dL	Imaging Findings	Tx
Gago *et al*. 40	55 M	12/52 LBP	Y	Y	L3-5 Facet Joints	Y	Y	MRI - exophytic lesion extending posteriorly from L3-4 facet joint, L4-5 facet joint inflammation	Surgery
Ding *et al*. 41	36 M	2/52 BP	Y	Y	T9-10 Intradural	2 yrs.	Y	CT - spinal stenosis due to occupied lesions T9 and T10, MRI - cord ischemia	Surgery
Cheng *et al*. 42	23 M	LBP 6yrs	Y	Y	Thx and Lx Epidural space	Y	Y	MRI - epidural collection C4-T11 along anterior thecal sac, CT - bone erosions L2-3 facet	Surgery
Borges *et al*. 43	56 M	2yrs LBP	Y	Y	T8-9 Epidural space	15 yrs	Y	CT and MRI - space occupying lesion at T8-9 - indentation of dural sac, multi-level facet joint hypertrophy and erosion of articular surfaces	Medication
Ng *et al*. 44	66 M	1/52 Neck Pain	Y	N	C4-5 Vertebra	N	Y	XR - C5-6 end-plate erosions, C5 vertebral body destruction, CT-prevertebral fluid C2-5, MRI - C5-6 CSS and myelomalacia	Surgery
Ng *et al*. 44	68 M	8/52 LBP	Y	Y	L4-5 Epidural space Ligamentum Flavum, Interspinous, Supraspinous Ligament	N	Y	MRI - epidural soft tissue signals around bilateral L4-5 pars defects causing severe LCSS and bilateral foraminal stenosis, cauda equina compression	Surgery
Vergara *et al*. 45	60 F	16/52 severe LBP	Y	NR	L3-4 Epidural space	NR	NR	CT Myelogram - severe LCSS L3-4, moderate LCSS L2-3	Surgery

Legend: *M=Male, F=Female, LBP= Low Back pain, BP=Back pain, Y= Yes, N= No, sUA = Serum Uric Acid, NR= Not reported, Mths= Months, /52 = weeks, Lx= Lumbar, Thx= Thoracic, Cx= Cervical, ALL= Anterior Longitudinal Ligament, LDH: Lumbar Disc Herniation XR= X-ray, CT= Computed Tomography, MRI= Magnetic Resonance Imaging, DECT= Dual Energy Computed Tomography, Yrs= Years, LCSS= Lumbar Central Spinal Stenosis, CSS= Cervical Spine Stenosis, Hx= History, Tx= Treatment*

presented with back or neck pain and 86% displayed radiating/neurological symptoms. Back pain varied from mild to severe with onset months to years prior to presentation. Only two patients did not report a history of axial pain[36,38] (four case studies did not report this clinical data[12,27,35,39]). Neurological symptoms and signs included upper and lower limb radiating pain, arm or leg paresthesias/numbness/weakness, claudication, muscle atrophy, bowel or bladder incontinence, clumsiness, and falls. Patients with spinal gout were more likely to have a history of extremity joint pain (84%) and peripheral gout (72%) with the feet, ankles, knees, wrists, and hands commonly affected. Subdermal gouty tophi were observed in 69% of cases.

The lumbar spine (61%) was the most affected spinal region, particularly the facet joints (37%) and epidural space (50%). Spinal gout was reported less commonly in the ligamentum flavum (13% of cases[30,36,38,44]), vertebrae (11% of cases[23,31,34,44]) and the lamina (11%[14,28,29,37]). There were isolated cases involving other spinal elements (intervertebral disc[34], spinous process[46], anterior longitudinal ligament[31], posterior longitudinal ligament[36], interspinous/supraspinous ligament[44], transverse ligament[23], erector spinae[21]). The cervical spine was involved in 29% of cases and the thoracic spine in 21% of cases. The most involved vertebral segments for each spinal region were L4-5 and L5-S1, T8-9 and T9-10, and C4-5 and C5-6. Spinal gout was also identified in the sacroiliac joints by a DECT scan in one of our cases.[20]

Laboratory findings demonstrated serum uric acid (sUA) levels above 6 mg/dL in 80% of patients, raised C-Reactive Protein (CRP) in 48%, and raised Erythrocyte Sedimentation Rate (ESR) in 48% of patients. Twenty-nine percent of patients had a sUA level over 10 mg/dL[13,29,30,33,36,39,40,42,43]. Twenty percent of patients were reported to have sUA levels in the normal range[21,23,24,26,31,32,37]. Three cases failed to report sUA levels[12,27,45], one case reported sUA as 'normal'[24], and one case reported sUA as 'elevated'[35]. In most cases X-ray, CT and MRI scans did not offer a definitive diagnosis. DECT scans assisted in the diagnosis of spinal gout in five of our cases[12,13,20,46]. Wang *et al*.[13] identified two cases of spinal gout utilizing DECT. Comorbid conditions were present in 75% of spinal gout patients. Obesity, type two diabetes, renal impairment, and hypertension were the most common comorbidities.

Twenty-eight cases (76%) in our review proceeded to surgery. One case was scheduled for surgery but passed before surgery was performed. Of our 38 cases, only eight cases were suspected as having spinal gout.[12,20,23,25,28,33,43,46] The most common presumptive diagnoses (79% of cases) were infection[21,22,31,34,37,40,42,44] (osteomyelitis, septic arthritis, spondylodiscitis, epidural abscess, tuberculosis), tumour[19,21,26,30,36,41] (meningioma) and disc herniation/degenerative stenosis[18,19,32,36,38,39,45]. Laminectomy/decompression was the most common surgical procedure. The majority of patients treated by surgery experienced an improvement in their symptoms. Seven of our nine cases treated conservatively with anti-inflammatories and uric acid lowering medication also experienced symptomatic improvement.[20–23,36,43] There were two cases treated with medication which did not report the outcome.[13,28] Our analysis is summarized in Table 2 below.

Criteria for the clinical diagnosis of gout

Recognizing the importance for earlier detection of gout in primary practice, Janssens *et al*.[47] developed diagnostic criteria emphasizing the clinical features of gout in preference to diagnosis by joint aspiration. Various clinical features were studied and given a point rating according to their association with gout. In combination they found that male sex, involvement of the first metatarsophalangeal joint, and elevated serum uric acid (> 5.88 mg/DL, 0.35 mmol per L) were highly correlated with a diagnosis of gout. Low scores, <4.0 points were found to have 97% sensitivity for excluding gout whilst high scores, >8.0 points were found to have 80% sensitivity for diagnosing gout (confirmed by joint aspiration).[47] Kienhorst *et al*.[48] validated Janssens *et al*.'s diagnostic rule (Table 3). Joint aspiration and crystal analysis remains the gold standard for gout diagnosis, however the challenges posed by joint aspiration, particularly in the case of spinal gout, limits the viability of this diagnostic tool.

The American College of Rheumatology and the European League against Rheumatism (ACR/EULAR) include joint aspiration and crystal analysis in their diagnostic criteria[49], however as per Janssens *et al*., clinical features are also strongly represented. Importantly, a negative joint aspiration does not necessarily exclude a diagnosis of gout if other clinical features score highly.[49] In contrast to the Janssens *et al*. criteria, the ACR/EULAR criteria only apply if there is a history of at least one episode of 'swelling, pain or tenderness of a peripheral joint or bursa.'

Table 2.
Summary of case report analysis

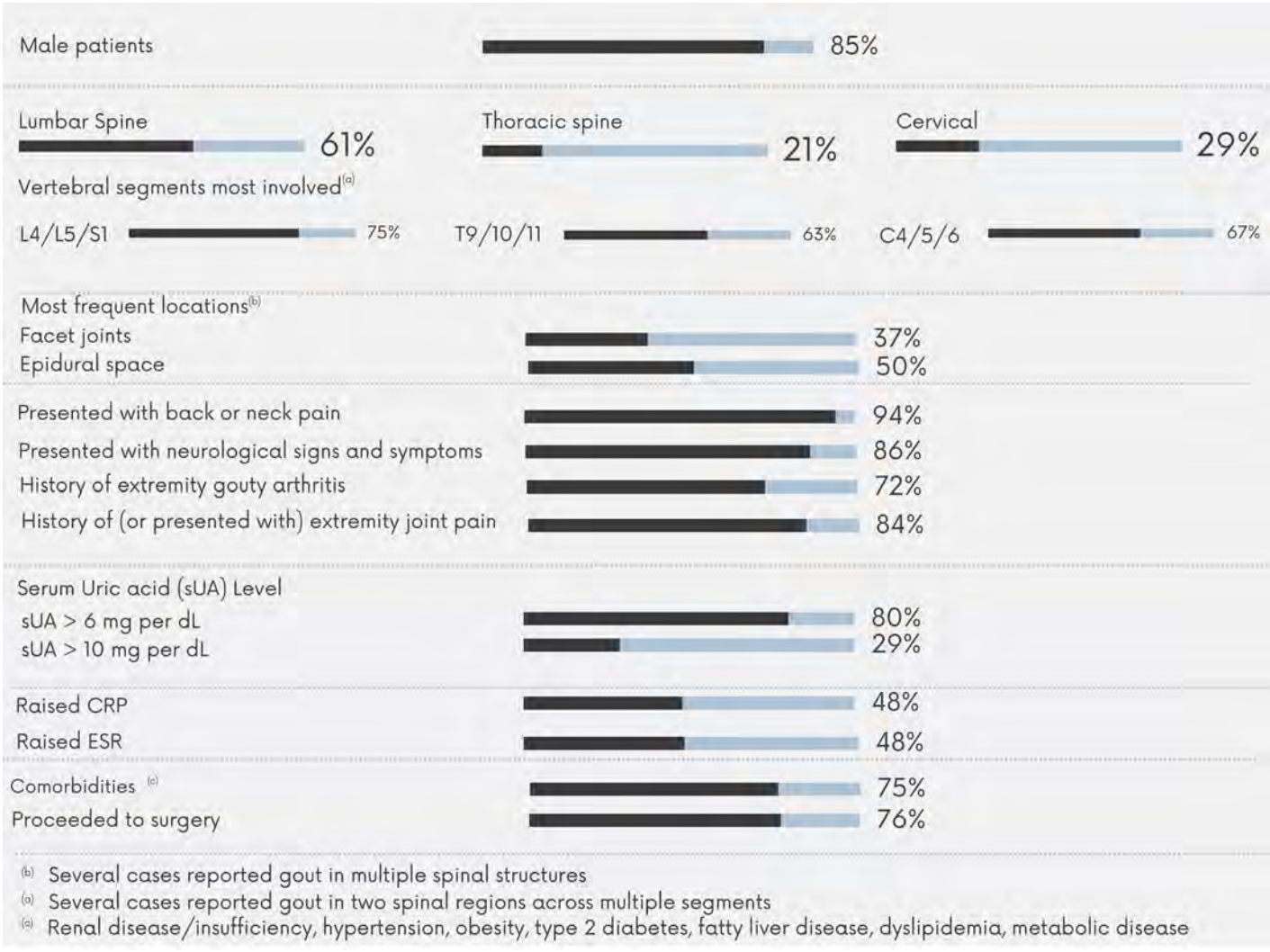

Table 3.
Diagnostic rule for gout diagnosis in clinical practice

Patient With Monoarthritis	
Clinical variables	
Male Sex:	2 points
Previous attack of patient reported arthritis attack	2 points
Onset within 1 day:	0.5 point
Joint redness	1 point
Involvement of the first MTP joint:	2.5 points
Hypertension or >1 cardiovascular diseases*	1.5 points
Serum uric acid > 5.88 mg/dL (0.35 mmol per L)	3.5 points
Total score: (maximum: 13 points)	
Interpretation:	
>8 Points	Gout in 87%
>4 and <8 Points	Uncertain diagnosis, Perform joint fluid analysis
<4 Points	Non-gout in 95% cases, consider non-gout diagnosis – CPPD, septic arthritis, reactive arthritis, rheumatoid arthritis, osteoarthritis, or psoriatic arthritis

*—Cardiovascular diseases include angina pectoris, myocardial infarction, heart failure, stroke, transient ischemic attack, and peripheral vascular disease.
CPPD: calcium pyrophosphate dihydrate deposition disease

Reprinted with permission: Kienhorst et al. The validation of a diagnostic rule for gout without joint fluid analysis: a prospective study. Rheumatology. 2015;54(4):609-614.

Imaging of spinal gout

Imaging modalities utilized for detecting spinal gout have been well researched. Patients presenting to primary practice for management of neck/arm pain or back/leg pain may typically undergo further assessment with x-ray, CT scan, MRI or DECT. Bony erosions and spinal tophi are well demonstrated by CT and MRI, however both technologies often lack the ability to differentiate spinal gout from other space occupying lesions such as abscess, tumour and disc extrusion.[50]

Based upon the imaging findings presented in Table 1, we provide the following summary of the radiological features suggestive of spinal gout, as observed using each of the most common imaging modalities:

X-ray

- Signs may have a similar appearance and be difficult to distinguish from osteoarthritis (OA) and age related changes – subchondral bone cysts, facet joint erosions with sclerotic borders, odontoid process erosion, soft tissue oedema.[11]

Computed tomography (CT scan)

- Bone or facet joint erosions with well-defined sclerotic margins.[11,15] Bone resorption occurs under the articular surface and may be accompanied by high-density tophi around the joint.[50]
- Facet or intervertebral bone neoformation, or juxta-articular or intra-articular masses that have increased density compared to surrounding muscle.[15]
- Tophi appear as a high-density mass and need to be distinguished from other space occupying lesions.[15,50]

Note: Patients presenting with an acute first episode of extremity gout are less likely to have evidence of joint erosions on imaging.[51]

Magnetic resonance imaging (MRI)

Tophi appearance

- T1 – hypointense to isointense signal, homogenous mass.
- T2 – hypointense to hyperintense signal, homogenous mass (tophi). Peripheral heterogenous contrast enhancement patterns.[11,15,52]

Disc space appearance:

- T1 hypointense signal. T2 – variable hypointense to isointense signal, contrast enhancement of the disc which can mimic discitis, degenerative changes or CPPD.[11,15,52]

Vertebra:

- Normal bone marrow signal of adjacent vertebrae.[11]

Dual energy computed tomography (DECT):

DECT scanning has been utilized for more than a decade for detecting and monitoring monosodium urate (MSU) deposits in gout patients. It has been found to have high sensitivity and specificity[53–55]. Dalbeth *et al*.[56] observed MSU deposits in 24% of individuals with asymptomatic hyperuricaemia, 79% with early gout (<3 years) and 84% with late gout. MSU deposits occurred in joints and tendons and were of a larger volume in the symptomatic patients.

False negatives on DECT scans are more commonly observed during a first flare of gout (< 6 weeks) or when MSU deposits are smaller in volume, such as those found in the facet joints. DECT is being utilized increasingly for detecting spinal gout, however there is ongoing research regarding the optimization of parameters/thresholds to minimize false positives and negatives.[51] Artefacts mimicking MSU deposits can be caused by beam hardening and image noise (patient movement). Beam hardening may occur due to the presence of metal implants, metal rings/piercings or dense cortical bone. Post-processing software may produce artefacts.[55] Toprover *et al*.[57] also propose that aged costochondral cartilage, which contains low concentration calcium deposits, may be mistaken for MSU deposits (due to having similar DECT attenuation properties).

Whilst further research is needed regarding the utility of DECT for diagnosing spinal gout, when combined with clinical findings, lab results and other imaging (CT and MRI)[52], DECT may prove to be a useful tool for improving diagnostic certainty in primary care settings. Figure 1 through 6 feature CT, MRI and DECT scans demonstrating spinal gout.

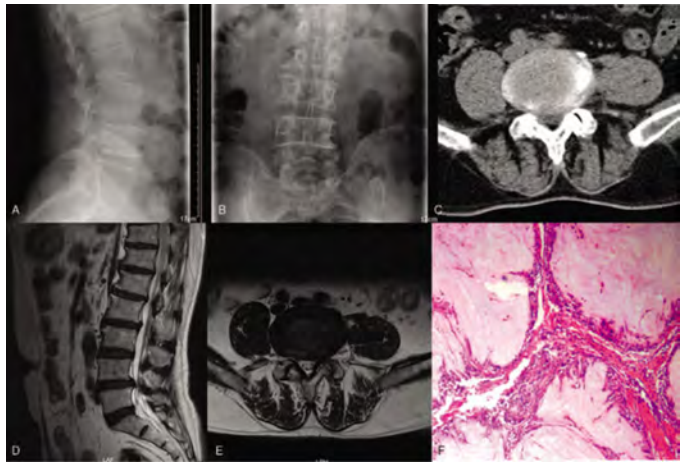

Figure 1.

Spinal gout imaging: x-ray, CT, MRI and histology of a 64-year-old male with an 11-month history of lower back pain and worsening left sided sciatica/leg weakness. (A) Lateral x-ray & (B)A-P x-ray show mild degenerative changes. (C) Axial CT shows left sided lateral recess stenosis at L4/5. (D) Sagittal MRI and (E) Axial MRI showing L4/5 disc herniation and left lateral recess narrowing. (F) Pathology examination revealing abundant MSU crystals surrounded by a foreign body-type giant cell reaction.

Reprinted with permission (CC-BY): Chen et al. Percutaneous transforaminal endoscopic decompression for the treatment of intraspinal tophaceous gout: a case report. Medicine. 2020;99(21)[32]

A proposed diagnostic algorithm for spinal gout in chiropractic practice

As far as the authors are aware, with reference to primary care settings, a clinical criterion specific to spinal gout diagnosis is yet to be developed. We reference current gout classification criteria guidelines[47,49] and combine them with clinical and diagnostic data derived from this, and previous spinal gout reviews,[11,15,16] to develop a diagnostic algorithm for spinal gout (Figure 7). We recommend this algorithm be utilized as an adjunct to standard diagnostic procedures used by chiropractic physicians rather than

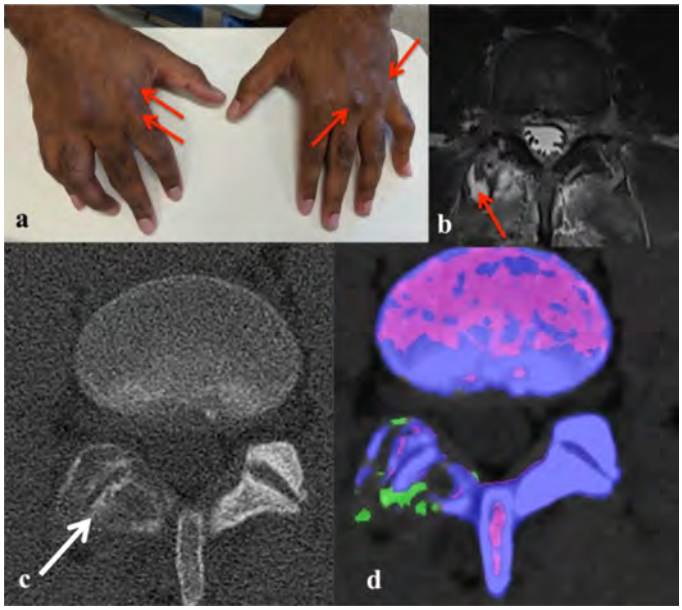

Figure 2.
Spinal gout imaging: CT, MRI, DECT of a 32-year-old man with fever, acute severe lower back pain, history of left knee pain and gout.(a) A 32-yo man with probable gouty tophi affecting both hands (red arrows), (b) Axial MRI of his Lumbar spine (T2) reported as showing an abscess surrounding the right L4/5 facet joint (red arrow). (c) Axial CT of lumbar spine showing erosions (white arrow); and (d) corresponding DECT image (Siemens Somatom ForceTM) showing MSU crystal deposition (green).

Reprinted with permission (CC-BY): Wang et al. The utility of dual energy computed tomography in the management of axial gout: case reports and literature review. BMC Rheumatol. 2020;4: 22.[13]

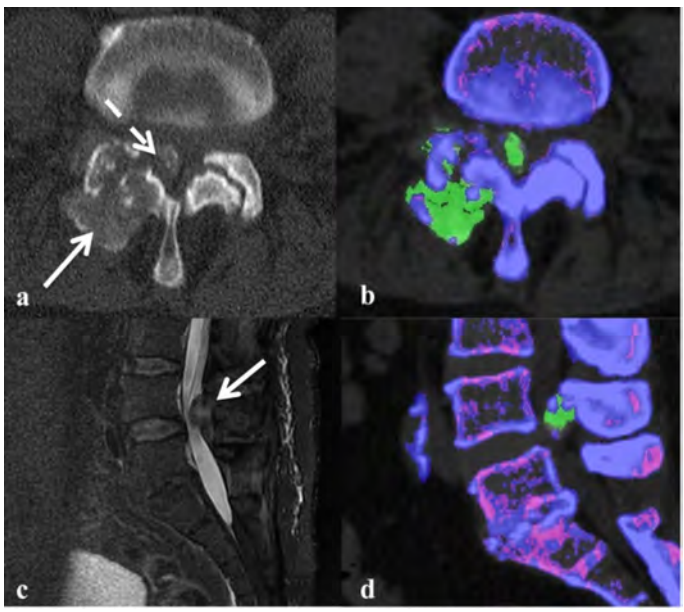

Figure 3.
Spinal gout imaging: CT, MRI and DECT of a 74-year-old male, six-week history of lower back pain and longstanding bilateral knee pain.

(a) Axial CT showing right L4/5 facet joint erosion (white arrow) with calcified peri-articular mass encroaching on the lumbar canal (dashed white arrow) (b) Corresponding DECT image (Siemens Somatom ForceTM) showing MSU crystal deposition (green). (c) Sagittal T2-fat suppressed MRI image of the lumbar spine showing the soft tissue mass seen in (a) and (b) causing marked lumbar canal stenosis, and (d) corresponding DECT image showing attenuation consistent with MSU crystal deposition.

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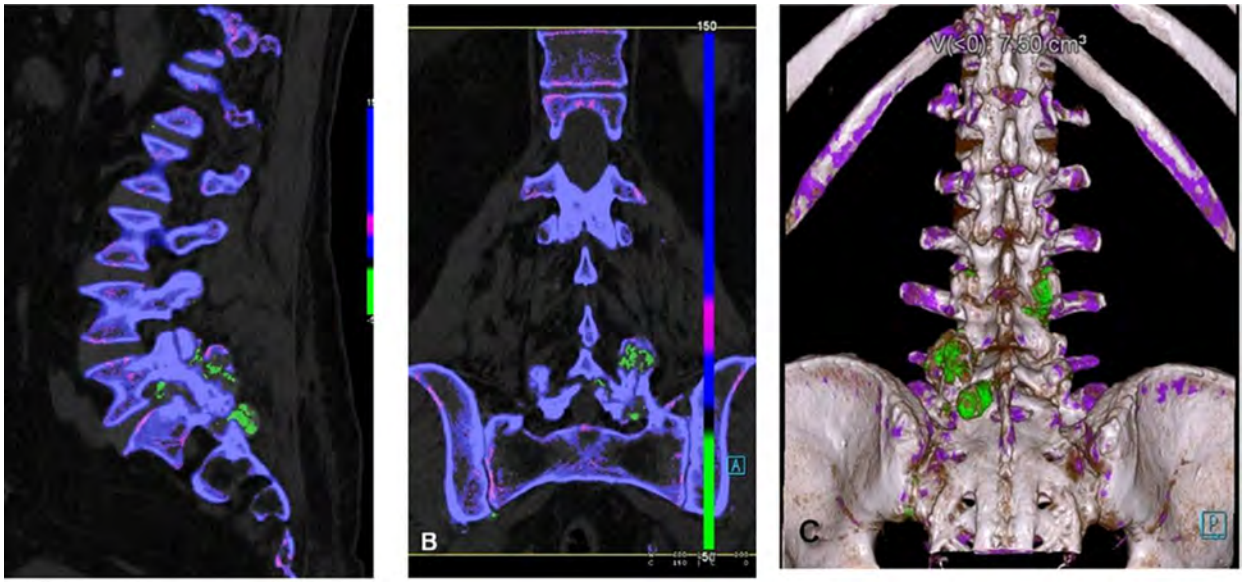

Figure 4.

Spinal gout imaging: DECT of a 67-year-old male patient presented with an exacerbation of acute-on-chronic lower back pain.

Spinal urate deposits Lumbar spine DECT (A) Sagittal view (B) Coronary view (C) 3D rendered images showing urate deposits in facet joints (green).

Reprinted with permission (CC-BY): Ahmad et al. Urate crystals; beyond joints. Front Med. 2021;8:649505.[58]

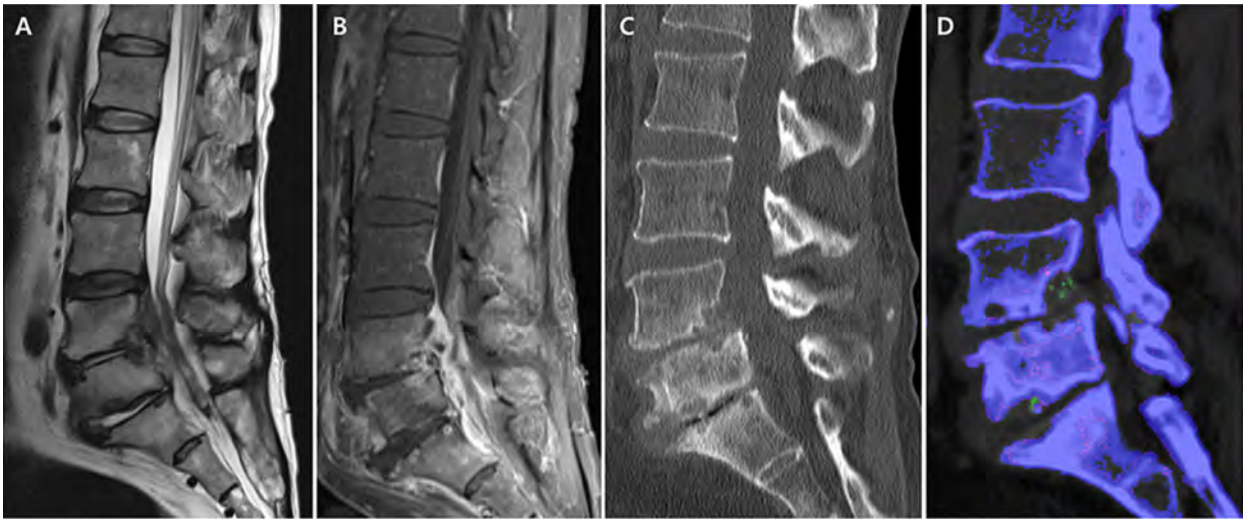

Figure 5.

Spinal gout imaging: MRI, CT and DECT of a 54-year-old with chronic lower back pain and a five-year history of gout. Presented to emergency with severe lower back pain and right buttock pain.

Lumbar spine MRI and CT in axial gout. (A) Increased signal intensity on T2 weighted imaging of L4-5 and L5-S1. Intervertebral disc and erosive changes on the posterior cortices and endplates of L4-L5-S1 vertebra. (B) Enhancement of epidural space on T1WI. (C) Erosive changes in L4-5 and L5-S1 endplates on conventional CT. (D) MSU deposits (green) in the erosive foci of endplate on DECT.

Reprinted with permission (CC BY): Jin et al. The frequency of axial deposition in Korean patients with gout at a tertiary spine center[8]

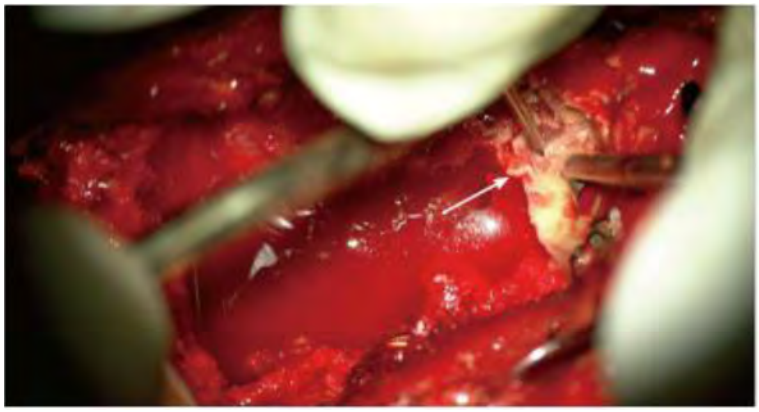

Figure 6.
Spinal gout: tophus removal. Intraoperative photograph taken by the surgical microscope showed a well-marked chalky white tophus lesion (arrow).

a replacement. When navigating the algorithm, the term 'usual spine care' indicates the usual process of diagnosis and management performed by chiropractors, including identifying other potential red flags.

Discussion

Spinal gout may present in an acute, subacute or a chronic stage of disease.[15] Spine related symptoms have been reported as the 'first manifestation of gout' in up to 25% of cases.[11] In a review of 131 cases of spinal gout, Toprover *et al*.[15] reported that patients usually described pain in the general area of urate crystal deposition, although this was not consistent.

To the best of our knowledge, the current paper is the first to provide analysis of spinal gout case reports published since 2017. We found 94% of patients in our review presented with spinal pain, and 86% displayed neurological symptoms and/or signs. This is consistent with a review by Hasegawa *et al*.[11] in which spinal pain was present in 93% of cases and neurological signs in 77.9% of cases. However, in a large cases series involving 142 cases of spinal gout, Zhang *et al*.[16] found 79.6% of patients presented with pain and 45.8% with neurological symptoms.

Consistent with previous reviews we found the lumbar spine to be the most common region reported for urate crystal deposition (61%). The thoracic spine (21%) and cervical spine (29%) were also frequently affected, noting several of our cases reported simultaneous gout in more than one spinal region.[27,36,39,42]

MSU deposits and tophi were found most frequently to involve the facet joints (14 cases) and epidural space (19 cases). Although occurring less frequently, MSU deposits were also reported in the sacroiliac joints, vertebrae, lamina, intervertebral discs, spinous processes, spinal ligaments (ligamentum flavum, anterior and posterior longitudinal ligaments, interspinous ligament, transverse ligament, and supraspinous ligaments) and soft tissues (erector spinae). Toprover *et al*.'s analysis of 131 spinal gout cases found 18 of 131 cases (14%) reported gout in the sacroiliac joint.[15] One case in our series reported gout in the sacroiliac joint.[20]

In twenty-five of our cases (68%) urate crystal deposition was confirmed by surgical excision and lab analysis. In one case, a chalky white material resembling gouty tophi was observed during surgical excision, however confirmation by histological analysis was not reported.

MSU deposition in the facet joints (and other spinal structures) may be more frequent than currently appreciated. Jin *et al*.[8] reviewed the spinal CT imaging of 95 patients diagnosed with gout finding 15.8% of patients demonstrated CT evidence of spinal gout. Yang *et al*.[50] reviewed the spinal CT scans of 17 patients with a diagnosis of spinal gout finding 84% of patients had evidence of facet joint bone resorption and erosion (41% involving multiple facet joints) and all patients demonstrating high density spinal tophi. Tophi were observed anterior and/or posterior to the facet joints, with several cases showing intrusion into the epidural space. Sullivan *et al*.[20,57] compared the spinal DECT scans of 50 gout patients with non-gout controls finding 20 to 40% of gout patients had MSU-coded lumbosacral lesions.

In our case series, most patients presenting with spinal pain and/or neurological symptoms were confirmed as having spinal gout following surgery. Prior to surgery, the most common presumptive diagnosis was infection or malignancy/tumour. Seven cases were suspected of having disc herniation and/or degenerative spinal stenosis.

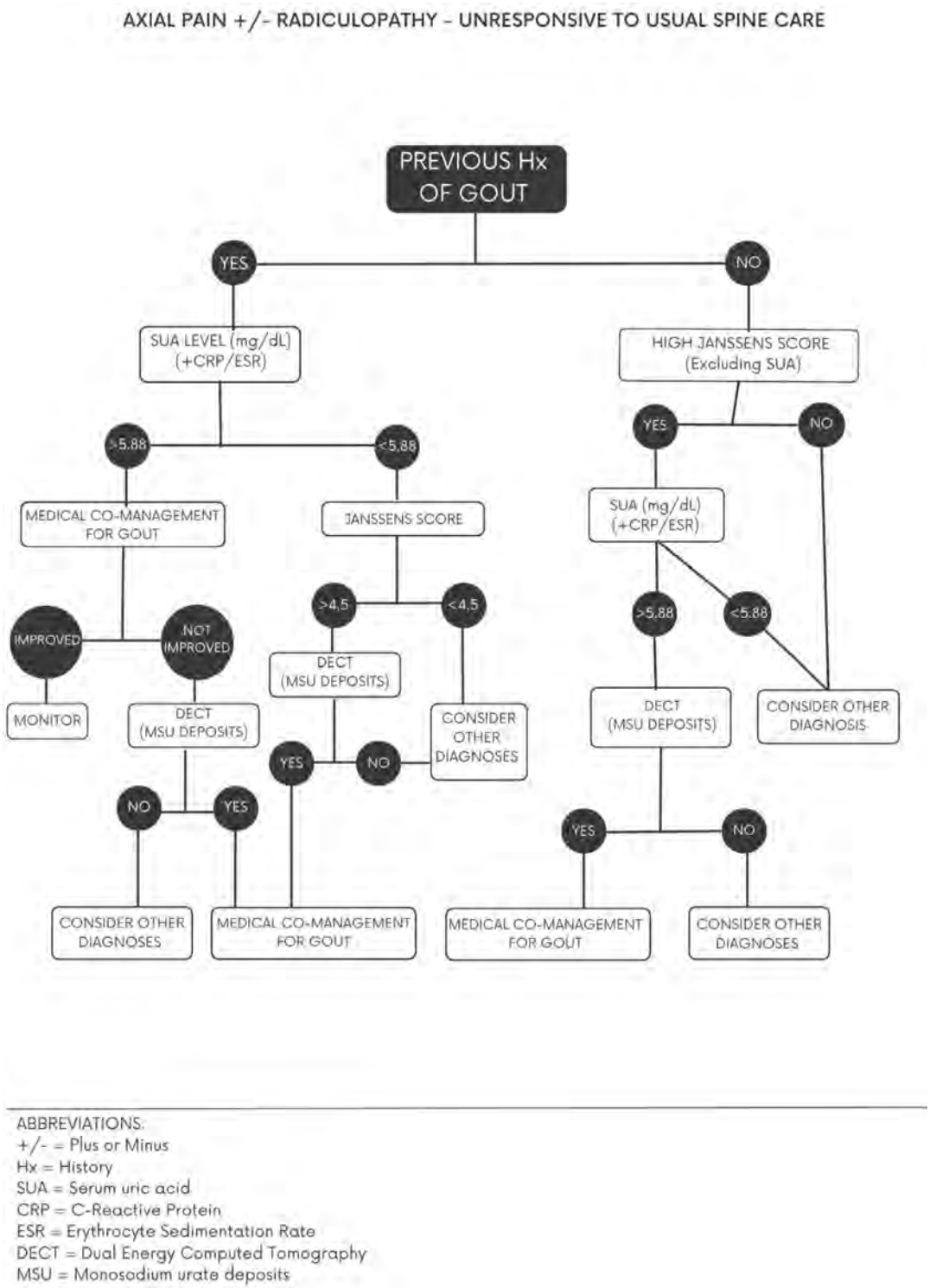

Figure 7.
Spinal gout: proposed diagnostic / treatment algorithm

Chen *et al*.[32] presented a case of a 64-year-old man with an 11 month history of lower back pain and worsening left sciatica. CT and MRI findings were consistent with a large L4/5 disc herniation causing neurological compromise, uric acid levels were normal, and there was no history of gout recalled. However, during surgery, gouty tophus was identified surrounding the disc extrusion (Figure 1). Consistent with previous literature reviews[16,17,59], our review found a continuing pattern of late diagnosis and surgical treatment of spinal gout. Seventy-six percent of the cases reviewed proceeded to surgery.

It has been proposed that spinal gout may develop asymptomatically, or with only mild symptoms, months or years prior to diagnosis.[60] We combined Zhang et al.'s review of 142 cases of spinal gout (1950-2017) with our review of 38 cases to calculate the percentage of patients presenting without a history of gouty arthropathy (a total of 34 cases were excluded due to incomplete data). We found 26% of the cases did not record a history of gout, although we note that by excluding some cases our figure may have overestimated this finding. Other literature reviews have reported similar percentages of patients presenting without a history of gout episodes (16.8-24.6%).[11,15]

Nine of our cases were managed conservatively with anti-inflammatories and uric acid lowering medications. Symptom duration in this small group was generally short: five patients presented within one to six weeks after the onset of symptoms. Three cases did not define symptom duration and one case described intermittent symptoms over a 2-year period. Larger prospective studies are required regarding early presentation and patient outcomes.

Limitations and future studies

A proportion of the spinal gout case reports we reviewed contained incomplete data, therefore our calculations and consequent conclusions may be inaccurate, however, as detailed in this paper, our findings are consistent with similar spinal gout reviews. As this is a narrative review no confirmation of data extraction or critical appraisal of the included articles was undertaken.

Historically, a majority of case reports involving spinal gout have described patients in an advanced state of the disease. Most often patients presented to medical facilities for evaluation before proceeding to surgery. Patients falling into this demographic may be less likely to have primary contact with a chiropractic physician. The clinical presentation of spinal gout in the chiropractor's office may therefore differ substantially from the patients included in this paper. Further research is required to improve our clinical understanding of spinal gout in primary care settings.

Secondly, the frequency of spinal gout locating to the facet joints (and other spinal tissues) may be under-estimated in our study. MSU deposits in the facet joints are difficult to verify without joint aspiration or DECT scanning. In addition, whilst MSU deposits may be present, they may be too small in volume to be detected (especially earlier in the disease). As most of the cases we reviewed did not utilize DECT or facet joint aspiration, a greater prevalence of facet joint involvement cannot be excluded.

Conclusion

Spinal gout, once considered a rare manifestation of peripheral gout, is being reported with increasing frequency and may be a more common source of spine related pain than previously appreciated. Chiropractic physicians are primary care providers for spine complaint patients and as such are responsible for the diagnosis and appropriate care of the patient. Chiropractors have an important role in the early detection of spinal gout potentially influencing the course of conservative management and reducing the burden of surgery to health care systems. Patients meeting clinical criteria may be referred for further screening and medical co-management.

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