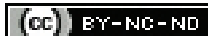


Primary Costochondral Gout with Concomitant Streptococcal Anterior Chest Wall Abscess: A Rare Diagnostic Challenge

NAUSHAD AHAMED¹, NANTHAKUMAR LOGITHASAN², G SOWMYA³, PREETAM ARTHUR⁴

ABSTRACT

Gout is a metabolic disorder caused by deposition of monosodium urate crystals in joints and soft-tissues. Gout classically affects peripheral joints such as the first metatarsophalangeal joint (podagra). Atypical presentation involves deposition in axial and other thoracic structures. Costochondral joint involvement is exceedingly rare and mimics infectious, inflammatory, or neoplastic aetiologies. The authors hereby, report the case of a 42-year-old male with poorly controlled diabetes and poor compliance to medications, presented with acute-onset chest pain with fullness in the left side of chest and high-grade fever for the past three days. On examination there was tenderness over the anterior chest wall and imaging revealed a localised large anterior chest wall collection adjacent to the costochondral junction involving the pectoral muscles and nearby structures. Image-guided aspiration yielded purulent material, which grew *Streptococcus* species. Cytological examination showed acute inflammatory infiltrates with monosodium urate crystal deposition consistent with gouty arthritis. He was managed with intravenous antibiotics, urate lowering agents, anti-inflammatory agents and analgesics resulting in significant clinical and radiological improvement. This case shows the diagnostic complexity associated with atypical presentation of gout, in patients with metabolic risk factors. High index of suspicion and early recognition with appropriate therapy is essential for a favourable outcome.

CASE REPORT

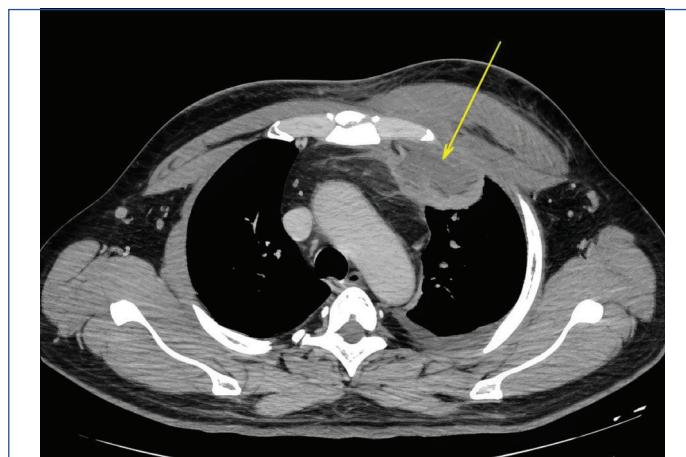
A 42-year-old male presented to the General Medicine Outpatient Department with complaints of acute-onset chest pain and associated fullness and swelling in the left side of the chest and high-grade intermittent fever, associated with malaise without chills and rigours for three days. The pain was sudden-onset, sharp, progressive radiating to the left shoulder causing movement restriction. There was no preceding trauma, dyspnoea, orthopnoea or syncope. The patient was a known case of long-standing diabetes with poor adherence to daily medications.

On examination, he was conscious, febrile, blood pressure of 130/90 mmHg, pulse rate of 110 beats per minute and other system examination was unremarkable. Local examination of the left anterior chest wall revealed a diffuse swelling which was warm and tender on palpation. There was no active discharge or sinus formation with restriction of range of movements of ipsilateral shoulder joint. Examination of the right wrist and right ankle showed swelling, warmth and tenderness. Multifocal nature of involvement was suggestive of an acute inflammatory arthritis.

Baseline investigations showed haemoglobin 11.8 g/dL, total count-18,180 cells/mm³, 90.8% neutrophils, suggestive of neutrophilic leucocytosis. Inflammatory markers were, Erythrocyte sedimentation rate was 110 mm/hour and C-reactive protein was 23.1 mg/litre. Renal and liver function parameters were within normal limits. Glycated haemoglobin was 10.7%, indicating patient has poor glycaemic control; uric acid levels were 3.4 mg/dL. Antinuclear Antibodies (ANA), anti-double stranded Deoxyribonucleic Acid (DNA) antibodies, anti-cyclic citrullinated peptide antibodies and rheumatoid factor were negative. Contrast-Enhanced Computed Tomography (CECT) of the thorax demonstrated a localised collection measuring 5.7×5.6×5.9 cm (≈90-95 cc) in the left anterior chest wall involving the pectoralis major, extending to mediastinum and extending from first rib to third rib [Table/Fig-1]. All these imaging features were suggestive of left chest wall abscess. Patient underwent ultrasound-guided aspiration of the chest wall collection in the first left costochondral joint, which yielded

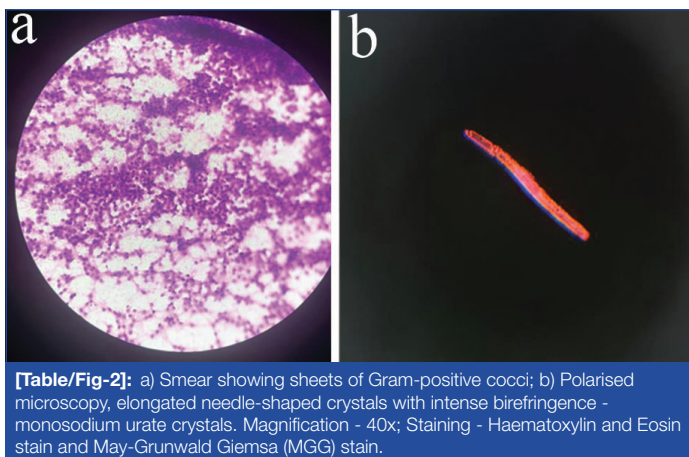
Keywords: Arthritis, Crystal arthropathies, Infectious, Uric acid

purulent material. Analysis showed total cell count: 15,000 cells/mm³, neutrophilic predominance (85%) and was negative for fungal elements and acid-fast bacilli. Gram stain showed Gram-positive cocci and Culture grew *Streptococcus* species and was negative for mycobacteria [Table/Fig-2a]. Cytological examination revealed suppurative inflammatory infiltrates with crystal deposition with refractive crystals on polarisation suggestive of gout [Table/Fig-2b].



[Table/Fig-1]: Contrast-enhanced CT thorax showing thick-walled collection (volume approx. 90-95 cc) involving the anterior chest wall and costochondral joint with extension into the mediastinum.

Pyogenic chest wall abscess was considered due to localised swelling and marked tenderness. Neutrophilic leucocytosis on complete blood count supported this possibility. However, simultaneous involvement of multiple joints was against the diagnosis initially. Septic arthritis was also considered, but there was no involvement of the sternoclavicular joint which argued against the diagnosis. Neoplastic lesions like bone tumours, may present as chest wall masses but presence of fever, imaging and cytological findings were against malignant aetiology. Tuberculous osteomyelitis was also considered as patient is in a region, which is endemic for tuberculosis, tuberculous involvement



of the sternum or ribs is an important differential diagnosis. However, the smear was negative for mycobacteria, absence of granulomatous inflammation on histopathology flagged against the diagnosis.

Based on these findings, he was diagnosed with gouty arthritis involving the costochondral joint, wrist and ankle joints with a secondary pyogenic abscess involving the anterior chest wall by *Streptococcus* species. Despite normal serum uric acid levels, Ultrasound-guided arthrocentesis of the chest wall collection was done with monosodium urate crystal deposition on polarising microscopy confirms the diagnosis of gouty arthritis. Patient was managed with intravenous antibiotics, piperacillin-tazobactam 4.5-gram i.v. thrice daily for seven days, which was later de-escalated to amoxicillin-clavulanate 1.2-gram i.v. twice daily for five days based on culture sensitivity reports. Analgesics and supportive therapy were provided to alleviate his pain and to improve the mobility. Anti-gout therapy, Colchicine 0.5 mg twice daily was prescribed for the inflammatory process and urate lowering therapy Febuxostat 40 mg once daily. Patient was managed with oral medications for diabetes.

After receiving appropriate therapy, clinical recovery was observed within three weeks of treatment. The chest wall swelling and pain gradually subsided. A repeat CECT thorax showed near complete resolution of previously noted collection measuring 1.5×1.9×1.9 cm (2.7 millilitre) [Table/Fig-3]. Patient was educated regarding the nature of his chronic illness. Dietary and lifestyle modifications were recommended. On follow-up after three weeks, patient remained adherent to the medical therapy. Glycaemic control was well maintained. There was marked improvement in his functional and health status, this was positively acknowledged by the patient's family.



[Table/Fig-3]: Follow-up imaging showing thick-walled residual collection (volume approx. 2.7 mL).

DISCUSSION

Classically gout affects peripheral joints. Atypical involvement of axial and other structures is currently being recognised. Atypical

presentations involve unusual anatomical sites like, axial skeleton to internal visceral organs [1], which often manifests in areas of lower physiological temperature or higher mechanical stress [2]. Risk factors include post-menopausal women with polyarticular involvement [3], patients on calcineurin inhibitors and elderly patients with multiple co-morbidities.

The present case illustrates the involvement of costochondral joint, an anatomical site that is infrequently implicated. Overlap in the clinical and radiological features often leads to diagnostic dilemma and delay in appropriate management [4]. As the patient resided in a tuberculosis-endemic zone, tuberculous osteomyelitis remained an important differential diagnosis.

Co-existence of secondary bacterial infection adds an additional layer of complexity to the present case. Although uncommon, co-existence of gouty and septic arthritis has been sparsely described in literature [5-7]. Previous studies have shown co-existing septic and gouty arthritis with diabetes and chronic kidney disease being the most common risk factors with *Staphylococcus aureus* being the predominant organism [6,7]. Crystal-induced arthritis can obscure the underlying joint infection and most of the patients had favourable outcomes.

Local inflammation caused by crystal deposition may predispose to bacterial colonisation, with underlying diabetes mellitus, increases the susceptibility to infection. Isolation of *Streptococcus* from aspirated material confirmed the presence of superadded infection, necessitating dual therapeutic approach. Imaging modality may be less valuable in the present case where the atypical site of involvement makes identification of the aetiology challenging.

Advanced imaging techniques like dual-energy CT can detect urate deposits with acceptable performance with, sensitivity and specificity ranging from 90% to 95%, but their limited availability restricts their routine clinical use [8-10]. Therefore, definitive diagnosis depends on demonstrating monosodium urate crystal deposition as observed in the present patient. This case highlights the importance of tissue diagnosis involving atypical sites. Clinical acumen played a pivotal role in the present case, as patient had diabetes mellitus and polyarticular involvement suggested an on-going systemic inflammatory process rather than an isolated infection.

The novelty of this case lies in the rare anatomical site of involvement of costochondral joint, atypical clinical presentation and coexistence of dual pathology, which collectively posed a challenging scenario [11].

Management of such cases needs a comprehensive multidisciplinary approach. Prompt initiation of appropriate antimicrobial therapy, with simultaneous treatment of acute gout with agents like colchicine and urate-lowering therapy prevents recurrence and disease progression. Optimising the underlying co-morbid condition especially glycaemic control played crucial role in reducing the infection and inflammatory burden. Key learning points in this case include that gout can involve atypical anatomical sites and may present as chest wall swelling, particularly in the presence of underlying metabolic risk factors. Furthermore, the co-existence of dual pathology necessitates the management of both conditions. A definitive diagnosis requires aspiration and demonstration of monosodium urate crystals. Normal levels of uric acid do not exclude the diagnosis.

CONCLUSION(S)

This case highlights an unusual presentation of gout involving the costochondral junction, mimicking an anterior chest wall abscess with a coexistent bacterial infection. The present case shows the need for considering gout in atypical anatomical locations, especially in patients with metabolic risk factors presenting with chest wall swellings. The presence of a secondary infection can mask the underlying metabolic condition, requiring clinicians to look beyond localised symptoms. Relying on tissue diagnosis and polarised microscopy remains the gold standard for navigating such diagnostic dilemmas. Early recognition of dual pathology

enables timely initiation of antimicrobial and anti-gout therapies. This comprehensive approach is vital for achieving complete clinical resolution and prevents long-term complications and local recurrence.

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PARTICULARS OF CONTRIBUTORS:

1. Postgraduate Student, Department of Internal Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.
2. Assistant Professor, Department of Internal Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.
3. Professor, Department of Internal Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.
4. Professor, Department of Internal Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Naushad Ahamed,
Postgraduate Student, Department of Internal Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai-600116, Tamil Nadu, India.
E-mail: naushad1296@gmail.com

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