Tumour-induced osteomalacia masquerading as spondyloarthritis: a case report

Sirs,

Tumour-induced osteomalacia (TIO) is a rare acquired paraneoplastic disorder caused by excess fibroblast growth factor 23 (FGF23), resulting in renal phosphate wasting and impaired bone mineralisation (1). Adult patients typically present with progressive musculoskeletal pain, proximal muscle weakness, and fragility fractures. Axial involvement and sacroiliac abnormalities on MRI may mimic axial spondyloarthritis (axSpA), leading to escalation of anti-inflammatory therapy that does not address the pathology (2). Misdiagnosis is associated with prolonged disability, repeated imaging, and avoidable costs from ineffective drugs, monitoring, and clinic visits. Diagnostic delay is frequent, and serum phosphate is the most actionable but often overlooked clue (1, 2). We report a case of TIO masquerading as axSpA to outline the clinical, biochemical, and imaging pitfalls and promote early recognition.

A 38-year-old man was referred in July 2025 for refractory axial pain. Symptoms began two years prior with low back pain and morning stiffness, initially attributed to seronegative ankylosing spondylitis despite negative HLA-B27 status. Treatments with celecoxib, sulfasalazine, and adalimumab provided minimal benefit. He later developed severe nocturnal pain and functional decline. Review of systems revealed an earlier incidental rib insufficiency fracture without significant trauma. Physical examination demonstrated axial tenderness over sacroiliac joints and multiple thoracic spinous processes, though provocative sacroiliac testing was negative.

Laboratory assessment revealed normal inflammatory markers, renal function, and parathyroid hormone. Hypophosphataemia (0.48 mmol/L) with elevated alkaline phosphatase (220 U/L) were found. Renal phosphate wasting was confirmed by elevated fractional excretion of phosphate (FEP: 27.9%, FEP >5% indicating renal phosphate wasting (3)) and reduced tubular maximum for phosphorus/glomerular filtration rate (TMP/GFR: 0.354 mmol/L). Meanwhile, imaging presented a diagnostic challenge. Bone scintigraphy showed symmetric axial uptake (Fig. 1A) and pelvic MRI demonstrated bilateral sacroiliitis with associated bone marrow oedema (Fig. 1B). Multiple vertebral and rib fractures with osteopenia were present. Though imaging findings were highly suggestive of SpA, the combination of refractory renal hypophosphataemia, low bone density, and multiple fractures raised strong suspicion for an underlying metabolic disorder.

A conventional 18F-FDG-PET/CT was unrevealing. Following a multidisciplinary team discussion, serum FGF23 was measured and found to be markedly elevated at 1040.0 pg/mL (reference range: 23.3–95.4 pg/mL). An octreotide scan including the standard field from mid-brain to proximal femora was negative. However, a subsequent whole-body AI18F-NOTA-LM3 PET/CT successfully identified a posterolateral distal tibia soft-tissue lesion (Fig. 1C). Retrospective physical examination confirmed a subtle, previously overlooked prominence at that site (Fig. 1D). Resection confirmed a phosphaturic mesenchymal tumour (PMT). Serum phosphate normalised within 24 hours, with marked symptom and function improvement at one month.

This case shows how TIO can mimic axSpA with axial pain, sacroiliac bone-marrow oedema, and diffuse bone scan uptake, yet the decisive signal was unexplained hypophosphataemia with renal phosphate wasting and markedly elevated FGF23. Localisation pitfalls, as reported in prior cases, included non-diagnostic FDG-PET/CT (4-7) and imaging with a limited field of view that omitted distal extremities (7, 8). In contrast, whole-body somatostatin receptor (SSTR)-PET/CT revealed the occult lesions amenable to curative resection in axSpA-like TIO cases (4-10). A meticulous physical examination, including distal and craniofacial palpation, is another essential but often overlooked component.

Taken together, TIO is an important and treatable mimic of axSpA. In refractory axial pain with fractures, a phosphate-centered pathway that includes early phosphate testing, confirmation of renal phosphate wasting, FGF23 measurement, and whole-body SSTR-PET/CT for localisation enables timely recognition and curative surgery, reducing disability and avoidable healthcare costs.

M. Li, *MD**
G. Zhou, *MS**
J. Lv, *MS*
J. He, *MS*
P. Zeng, *MS*
Q. Wang, *MD*

**Contributed equally.*

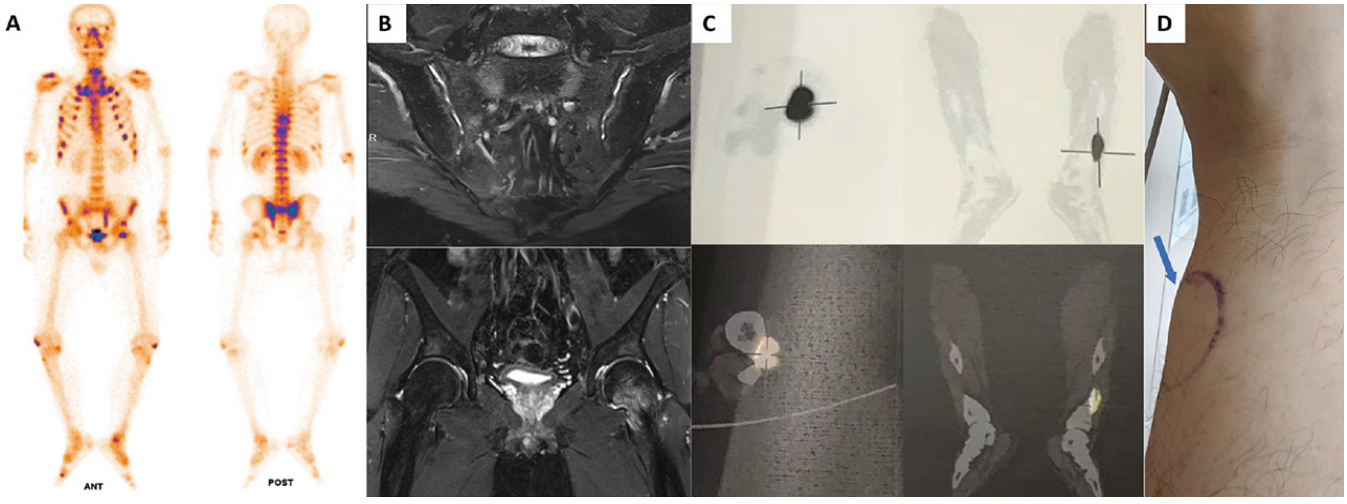


Fig. 1. Axial SpA-like imaging features with definitive tumour localisation in TIO.
A: Whole-body 99mTc-MDP bone scintigraphy (anterior/posterior) showing symmetrically increased uptake in the sternum, cervical-thoracolumbar-sacrococcygeal spine, multiple bilateral ribs, the left proximal femur, pubic symphysis, and multiple peripheral joints, most marked at the bilateral sacroiliac joints.
B: Pelvic T2-weighted MRI demonstrating bilateral sacroiliitis with bone-marrow oedema in both acetabula and the left femoral head and neck.
C: Somatostatin-receptor PET/CT with AI-18F-NOTA-LM3 identifying a focal avid soft-tissue lesion along the posterolateral distal left tibia.
D: Clinical photograph showing the external appearance of a subtle mass over the lateral distal left tibia (blue arrow).
TIO: tumour-induced osteomalacia; SpA: spondyloarthritis; MRI: magnetic resonance imaging; SSTR: somatostatin receptor; PET: positron emission tomography; CT: computed tomography.

Department of Rheumatology and Immunology, Peking University Shenzhen Hospital, Shenzhen Key Laboratory of Inflammatory and Immunology Diseases, Shenzhen, China.

Please address correspondence to:
Qingwen Wang
Peking University Shenzhen Hospital, Shenzhen Key Laboratory of Inflammatory and Immunology Diseases,
no. 1120 Lianhua Road, Futian District, 518036 Shenzhen, China.
E-mail: wangqingwen@pkuszh.com

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