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Case Report

Recurrent Transient Osteoporosis Syndrome Presenting with Severe Limp, Synovitis and Multifocal Subchondral Fractures: A Case Report

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Abstract

Transient Osteoporosis Syndrome (TOS) is an uncommon but distinct clinical entity characterised by acute onset joint pain, reversible bone marrow oedema and significant functional impairment. Although classically self-limiting, recurrence across multiple joints spanning decades is extremely rare and poorly understood. A 58-year-old man presented with a two-month history of progressive left hip pain, resulting in severe limping and inability to weight-bear, requiring the use of two crutches. His left hip MRI demonstrated diffuse bone marrow oedema, moderate synovitis and multiple small subchondral fractures of the femoral head. Laboratory investigations showed mildly elevated inflammatory markers but were otherwise unremarkable. Historical records confirmed two previous episodes of Transient Osteoporotic Synovitis (TOS): one affecting the same hip at age 19 and another involving the left knee four years ago. The patient was treated conservatively with strict non–weight-bearing, opioid analgesia and a gradual rehabilitation plan. The diagnosis of recurrent, migratory TOS was made based on clinical, radiological and historical findings. This case highlights an unusually severe form of recurrent transient osteoporosis, emphasising the need for early MRI evaluation to distinguish it from avascular necrosis and avoid unnecessary invasive interventions.

Keywords: Transient Osteoporosis Syndrome; Bone-Marrow Oedema; Hip Pain; Synovitis; Subchondral Fractures; Migratory Osteoporosis; Avascular Necrosis Mimic

Introduction

Transient Osteoporosis Syndrome (TOS) is a rare, self-limiting disorder characterised by acute onset joint pain, rapid functional decline and reversible osteopenia. It most commonly affects middle-aged men, although it is also reported in late pregnancy [1]. The underlying pathophysiology remains unclear, with proposed mechanisms including transient ischaemia, microvascular dysfunction, neurogenic factors and accelerated regional bone turnover [2].

Diagnosis is often challenging because early symptoms and imaging may mimic Avascular Necrosis (AVN), septic arthritis or occult fractures [3,4]. Radiographs may remain normal initially, making MRI essential for identifying diffuse marrow oedema and excluding the characteristic serpiginous borders or double-line sign seen in AVN.

Recurrence is uncommon; when it occurs, it typically affects the same joint within a short interval, while migratory or widely spaced recurrences are extremely rare [2]. Subchondral fractures may arise in more severe presentations due to transient trabecular weakening [5]. Hormonal, metabolic and biomechanical factors may contribute to susceptibility, particularly in atypical or recurrent cases [6].

Recent studies support TOS as a dynamic, reversible metabolic bone process, with transient reductions in bone mineral density demonstrated by quantitative MRI and DEXA techniques [7]. Early off-loading and guided rehabilitation may shorten symptom duration and reduce the risk of complications [8]. Although TOS is generally benign, severe or recurrent forms require close follow-up to prevent mechanical insufficiency fractures or prolonged oedema syndromes [9]. Correctly differentiating TOS from AVN remains critical to avoid unnecessary invasive interventions [3]. This case highlights an unusually long recurrence interval nearly four decades and demonstrates the potential severity of TOS when complicated by marrow oedema–related subchondral fractures.

Case Report

A 58-year-old man presented with a two-month history of worsening left hip pain. The pain was severe, constant and poorly controlled despite oxycodone. He required two crutches and reported significant difficulty standing or walking even short distances. There was no preceding trauma, fever, systemic illness or weight loss.

Medical History

- Type 2 diabetes
- Hypertension
- Hyperlipidaemia
- Ischaemic heart disease
- Right supraspinatus calcific tendinosis treated previously with barbotage

Examination

- Groin tenderness
- Severe limitation of internal and external hip rotation
- Marked antalgic gait requiring bilateral crutches
- No erythema or warmth
- Knee examination normal

Laboratory Results

Laboratory investigations demonstrated a mildly elevated C-reactive protein (CRP) at 22 mg/L and an Erythrocyte Sedimentation Rate (ESR) of 35 mm/hour. The full blood count was unremarkable, with a white cell count of 10.7×10^9/L, haemoglobin level of 151 g/L and platelet count of 260×10^9/L. Autoimmune screening, including Antinuclear Antibodies (ANA), Rheumatoid Factor (RF) and anti-Cyclic Citrullinated Peptide (anti-CCP) antibodies, was negative. The uric acid level was normal at 0.17 mmol/L and liver, renal and thyroid function tests were all within normal limits. Collectively, these findings did not support infection, inflammatory arthritis or crystal arthropathy as the cause of his symptoms.

Pelvic Radiographs

- Subtle thinning of cartilage centrally in both hip joints is present but no significant bony changes of degenerative disease are identified and the SI joints are normal
- No osteophytes or sclerosis
- No femoral head collapse
- Normal sacroiliac joints

Left knee X-Ray was normal.

MRI of the left hip (Fig. 1) revealed:

- Extensive bone marrow oedema in the femoral head and neck
- Multiple small subchondral fractures in the posteromedial femoral head
- Moderate synovitis
- Absence of AVN signs (no serpiginous border, no double-line sign)

These findings strongly supported transient osteoporosis over avascular necrosis.

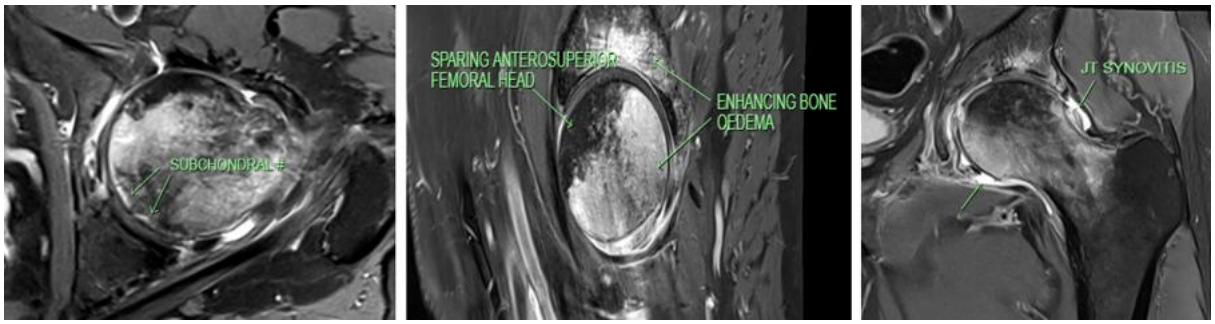

Figure 1: MRI of the left hip showing bone-marrow oedema, subchondral fractures and reactive synovitis consistent with transient osteoporosis.

Crucially, he had two prior documented episodes of transient osteoporosis:

1. Left hip at age 19, confirmed by MRI and nuclear bone scan
2. Left knee four years ago, MRI showing subchondral oedema consistent with TOS (Fig. 2)

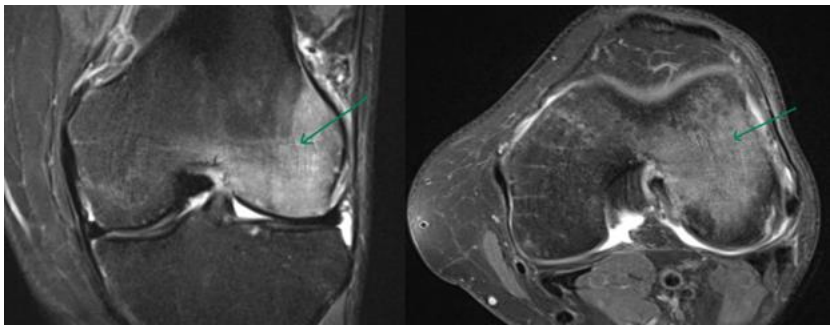

Figure 2: Left knee MRI; subchondral oedema of the lateral femoral condyle, more consistent with transient osteoporosis than an acute or subacute fracture.

Management

- Strict non–weight bearing
- Oxycodone and paracetamol
- Avoidance of NSAIDs due to comorbidities of diabetes and hypertension
- Gradual reintroduction of weight bearing as symptoms allowed

Discussion

Transient Osteoporosis Syndrome (TOS) is an uncommon bone marrow oedema disorder presenting with acute pain, functional impairment and reversible osteopenia, most frequently involving the hip. It may follow a self-limiting course but can occasionally present in recurrent or migratory patterns. Hormonal, metabolic and biomechanical influences have been proposed as potential contributors in such atypical cases [10]. A key clinical challenge is the similarity between early TOS and Avascular Necrosis (AVN), particularly when radiographs remain normal. MRI therefore plays an essential role in establishing the diagnosis and in identifying complications such as subchondral insufficiency fractures, which require strict off-loading to prevent progression or collapse [5,6].

This case is remarkable for its exceptionally long interval nearly 40 years between symptomatic episodes, as well as its migratory pattern involving two different joints many years apart. Such delayed recurrence is exceedingly rare and raises the possibility of an underlying predisposition to episodic regional bone vulnerability, potentially related to vascular, biomechanical or metabolic factors [11]. The presence of small subchondral fractures places this case at the severe end of the TOS spectrum. Transient demineralisation can weaken trabecular bone sufficiently to precipitate mechanical insufficiency, explaining the disproportionate severity of pain, the inability to weight-bear and the profound functional limitation observed.

Distinguishing TOS from AVN remains a key diagnostic challenge because both conditions can present with acute hip pain and disability. MRI provides crucial discriminatory features: TOS typically demonstrates diffuse bone marrow oedema without the serpiginous subchondral borders, focal collapse or double-line sign characteristic of AVN [4]. The detection of subchondral fractures is particularly important, as these lesions require strict non–weight-bearing to prevent progression or potential collapse of the articular surface. Early MRI evaluation therefore serves a dual purpose-confirming TOS and identifying high-risk structural features that alter management.

Although TOS is generally benign and self-limited, severe, migratory or recurrent presentations mandate closer clinical surveillance. Prolonged marrow oedema can compromise trabecular integrity and may predispose to structural collapse or persistent bone marrow oedema syndromes if inadequately off-loaded [12]. Accurate classification is essential to avoid misdiagnosis, which may otherwise result in unnecessary invasive interventions typically considered for early AVN. Conservative management-including protected weight-bearing, analgesia, physiotherapy-guided rehabilitation and serial imaging-remains highly effective when instituted early and appropriately tailored to disease severity [13].

Overall, this case demonstrates that:

- TOS can recur after many years and may follow a migratory pattern
- MRI is crucial for distinguishing TOS from avascular necrosis
- Subchondral fractures require careful protection with non–weight-bearing to prevent collapse
- Early recognition prevents misdiagnosis and supports effective conservative care

Conclusion

This case highlights an atypical but clinically important presentation of recurrent transient osteoporosis syndrome with severe marrow oedema, synovitis and spontaneous subchondral fractures. The migratory pattern spanning nearly four decades underscores the need for continued awareness of TOS as a reversible but potentially disabling condition. Accurate MRI-based differentiation from avascular necrosis and protective off-loading remains the cornerstones of management.

Conflict of Interests

The authors declare that there is no conflict of interest related to this study.

Patient Consent

The patient provided written informed consent for publication of all clinical information, including history, laboratory results and imaging descriptions.

Acknowledgement

The authors gratefully acknowledge the patient for his cooperation and willingness to share his clinical history for the purpose of advancing understanding of this rare condition. We also extend our appreciation to the reporting radiologist, whose careful review and correlation of historical MRI studies were instrumental in establishing the final diagnosis.

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