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CASE REPORT

Transient osteoporosis of the hip: A Case report

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Abstract Transient osteoporosis of the hip (TOH) is a rare, self-limiting condition of unknown aetiology characterised by sudden onset pain without trauma, primarily affecting middle-aged men, and women in the third trimester of pregnancy. Patients typically present with a limping gait. MRI is the imaging modality of choice and reveals focal bone marrow oedema, joint effusion and preserved joint space. TOH must be differentiated from avascular necrosis due to the different associated treatment strategies. A case of TOH is described in a healthy 65 year old man including clinical presentation, differential diagnosis, pathophysiological considerations, imaging studies and management.

Implications for practice

- Transient Osteoporosis of the Hip should be included in the differential diagnosis for middle-aged men presenting with severe acute hip pain without trauma.
- MRI is the imaging modality of choice for its detection.
- TOH must be differentiated from avascular necrosis to avoid potential joint failure.

Introduction

Bone marrow oedema syndrome (BMES) refers to transient conditions with uncertain pathophysiological mechanisms affecting the hip, knee or ankle, including transient osteoporosis and regional migratory osteoporosis (RMO),[1] and in some classifications, chronic regional pain syndrome (CRPS) type I.[2] Transient osteoporosis of the hip (TOH) was first described by Curtiss and Kincaid in 1959[3] as a rare, self-limiting condition which typically affects healthy middle-aged men, and women in the third trimester of pregnancy or immediately post-partum.[1,4—8] Male to female

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incidence is 3:1.[2,6] The term RMO is used when, in approximately 20%[2] of cases, TOH takes a migratory course, usually affecting the contralateral hip or a neighbouring joint[9] and in some instances recurring in the original joint.[10,11] RMO most commonly occurs within a year of the initial episode.[2,12]

Hip pain is a common presenting complaint seen by manual therapists in the UK.[13] Individuals affected by TOH present with sudden onset hip pain, a limping gait, normal range of motion and usually without a history of trauma.[4] Pain improves with rest and nocturnal pain is not usually present. Passive range of motion may be preserved until the last few degrees at which point pain is elicited.[8] The hip is the most frequently affected joint.[14] Average time to resolution is between six and twelve months[10,11] although cases have been reported lasting up to 24 months.[7] It is likely that many cases go undiagnosed because TOH, being a self-limiting condition, is often an incidental finding on MRI.[1] Consent was received from the patient for publication of this case report. The Consensus-based Clinical Case Reporting Guidelines were used to structure this case report.[15]

Case history

A 65 year old man presented with sudden onset right hip pain of 24 h duration. His symptoms were aggravated by walking and only partially relieved by recumbency. The patient rated his pain as 7/10 at rest and 9/10 on bearing weight. He denied any recent history of trauma, weight loss, loss of appetite, nocturnal pain, fatigue or foreign travel, and he had no gastro-intestinal or genito-urinary symptoms. His medical history was noncontributory but there was some family history of osteoporosis.

The patient was ambulatory with a right-sided limp and a positive standing Trendelenburg test. Active and passive hip movements were within the normal range but considerable apprehension was noted on full hip flexion with a preference for external rotation (Drehmann's sign present[16]). Straight leg raise, FABER and FADIR tests were negative. No abnormality was detected on examination of the spine or abdomen. Power, reflexes, sensation and peripheral pulses were normal. Some tenderness was elicited on deep palpation of the anterior right hip and in the corresponding gluteus medius and psoas major. The constellation of signs and symptoms led me to refer the patient to his GP with a suggestion that an MRI scan be performed. MRI was requested over plain films because of the concern that pain of this severity and an inability to weight-bear may have been caused by avascular necrosis (AVN) in this patient who had no history of arthritis nor any prior hip pain.

MRI revealed extensive oedema within the head and the neck of the right femur with accompanying joint effusion (Fig. 1). The radiology report concluded that these observations were consistent with TOH. There were no findings suggestive of AVN. The patient was referred to a rheumatologist and pamidronate bisphosphonate was administered intravenously. Advice was given to avoid excessive weight-bearing activity due to the risk of stress fracture. A follow up MRI was performed two months later, demonstrating near complete resolution of bone marrow oedema and reduction of the joint effusion (Fig. 2). By this time the patient was pain free and a programme of joint mobilisation and Pilates was commenced to restore function.

Discussion

TOH is a rare, self-limiting condition which is likely under-diagnosed because conventional X-rays are normal in the early stages.[5] Its rarity has resulted in a lack of epidemiological studies to date,[17] but some potential risk factors have been identified, including low calcium intake, liver fibrosis, high alcohol intake and tobacco smoking.[9]

Differential diagnosis

A thorough history and physical examination should help the clinician to distinguish TOH from osteoarthritis, infectious arthritis, reactive arthritis, rheumatoid arthritis, crystal-induced arthritides, osteomyelitis, malignancy and tuberculosis.[7] A history of trauma and the presence of secondary changes including skin atrophy, sensorimotor changes and contractures may distinguish CRPS type I from TOH.[7] AVN must be ruled out by MRI to avoid potential joint failure.[2,4,7]

Pathophysiology

An early view in the era preceding MRI was that TOH represented a non-traumatic variety of Südeck's atrophy, later termed reflex sympathetic dystrophy (RSD) and now known as CRPS type I.[8,18] Some authors believe that TOH represents an early stage of AVN because histological studies reveal necrosis and marrow oedema which are similar to findings in early stage AVN,[1,19,20] but a study of 155 TOH patients found that none progressed to AVN.[2]