

Clinical Article

Irritable Hip in Childhood: Possible role of calcium pyrophosphate?

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Abstract

The cause of irritable hip in children has so far eluded in depth investigation. We present the case of a four-year-old boy with a limp. Ultrasound guided aspiration of the affected hip joint revealed calcium pyrophosphate crystals. The symptoms settled with rest and oral anti-inflammatory agents. Could crystal deposition disease be one of the causes of irritable hip in this young age group? *Int Pediatr.* 2002;17(4):213-215.

Key words: irritable hip, limp, calcium pyrophosphate crystals, crystal deposition disease

Introduction

The child with a limp and without a history of injury is a common problem in accident and emergency departments. Limping is never normal and few parents tolerate the problem long before seeking medical attention. In a recent study,¹ the main diagnosis was irritable hip/transient synovitis in 39.5% while Perthes' disease accounted for 2%. Forty-percent had a preceding illness. Most of the children were treated in the accident and emergency Department. The cause of irritable hip has so far eluded detailed investigation. We present an interesting case of a four year-old boy who presented with a limp to the Accident and Emergency Department at a District General Hospital.

Case Report

A four-year-old was presented with a two-day history of pain in the right groin and a limp. There was no history of a preceding illness. The pain started as a mild discomfort but became progressively worse, limiting his activities. He was the only child in the family and there was no relevant personal or family history.

On examination, he looked well and was febrile. The right hip was held in slight flexion and external rotation,

and he was tender in the groin. The movements of the right hip were only limited towards the extremes of extension and external rotation. There were no systemic signs of other rheumatological disease or a septic focus.

X-rays, which included an antero-posterior view of the pelvis and a frog lateral, showed no bone abnormalities suggestive of Perthes' disease, osteomyelitis, neoplasms, or hip joint effusion. The white cell count was 9.4 and the ESR was 16mm in the first hour.

The working diagnosis was that of transient synovitis. At ultrasonography, 8mls of a cloudy yellow fluid were aspirated. The Gram stain and culture were negative. However, calcium pyrophosphate crystals in the presence of lymphocytes and macrophages were found at cytology.

His symptoms settled overnight after aspiration, rest and non-steroidal analgesics. He was reviewed in the outpatient clinic ten days later; his symptoms had subsided completely and he had normal pain free movement of the right hip and a normal gait.

Discussion

The etiology of transient synovitis is still unknown, but the natural history is that of a self-limiting disorder with no residual sequelae. Recurrence is possible. Most cases can be managed with bed rest at home and administration of a non-steroidal anti-inflammatory medication.

The differential diagnosis includes trauma, infection, neoplasia and inflammatory, congenital, neuromuscular or developmental disorders. A single test for discrimination between these conditions is currently not available. Several protocols for the investigation of the child with a limp have been suggested.²⁻⁵

Biochemically, elevation of the erythrocyte sedimentation rate and of the C-reactive protein are important in differentiating septic arthritis from transient synovitis.⁶ The combination of an erythrocyte sedimentation rate of more than 20 mm/hr and/or a temperature of more than 37.5 C identified 97% of all cases of septic arthritis of the hip.⁷

Ultrasound as a primary investigation is encouraged,^{4,8-12} especially with the hip extended and abducted.¹³ An effusion is suggested by an increased

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anterior capsule distance (more than 2-4 mm and a side difference of more than 2 mm)^{14,15} or by a change in appearance of the hip capsule (a bulging convex appearance when fluid is present compared to a concave capsule that parallels the long axis of the femoral neck in the absence of an effusion).¹⁶ Because of an asymptomatic synovitis in the contralateral hip in 25% of cases,¹⁷ a comparison with age-related normal values is important. Reliance on the echopatterns emitted by the various constituents of the effusion (sterile, purulent or hemorrhagic) is not to be encouraged because of variations in interpretation.¹⁸⁻²⁰

The finding of calcium pyrophosphate crystals in the synovial fluid of this child has several implications on the possible etiology of transient synovitis. About 15% of children will suffer from a recurrence of transient synovitis.²¹ Recurrences of calcium pyrophosphate deposition (CPPD) disease in a much older age group are well known and are adequately managed by rest and non-steroidal anti-inflammatory agents. The same management seems to work very well for patients with irritable hip.

It is estimated that about 5% of the adult population has deposits of calcium pyrophosphate crystals in the knees and that prevalence increases with age. The clinical presentation is variable, from pseudogouty forms, pseudorheumatoid arthritis or secondary degenerative joint disease, to asymptomatic cases (20%).²² In the systematic evaluation, family history should be sought (primary form), and metabolic diseases such as gout, diabetes, hemochromatosis, hyperparathyroidism and hypothyroidism (secondary form) should be ruled out.

Though CPPD is prevalent in older generations, certainly above the second decade, early-onset osteoarthritis and chondrocalcinosis are linked to chromosome 8q^{23,24} and chromosome 5p.^{25,26} A syndrome of chondrocalcinosis associated with recurrent childhood seizures is linked to chromosome 5p.²³ Several recent reports show CPPD crystal disease occurring in association with Gitelman syndrome, the hypocalcuric-hypomagnesaemic variant of Bartter syndrome.²⁶

The concentration of CPPD per ml of fluid and the total mineral content per joint were greater early on in the acute stage of the synovitis than during the recovery phase.²⁷ The importance of concentration of crystals in the generation of the synovitis may explain the finding that following aspiration of the hip joint effusion, capsular distension was significantly and permanently lower when compared to the non-aspirated cases.²⁸ It is well known that damage to the articular cartilage is a precipitant for further recurrences of CPPD.^{29,31}

In an in vivo study, inflammation due to the CPPD crystals was maximal at about 9 hours following the start of the attack.³² Inflammatory cells appearing at around this time played a major part in dissolving the crystals, as did a change in pH and pyrophosphate activity. CPPD crystals also reduce greatly in number if the synovial fluid sample is left to stand overnight prior to being examined.³³ The fact that we found crystals in our sample may be related to the prompt aspiration of the joint as well as the prompt examination of the synovial fluid. The larger crystal size associated with acute attacks of CPPD²⁷ could account for the finding of these crystals in our sample.

Is it possible that CPPD crystals were not previously detected in the synovial fluid of these children because, in view of the very young age, they were not specifically looked for? Ultrasound of the hip joint in patients with irritable hip may have been delayed while observing the child with routine blood tests and plain radiographs; a sample of synovial fluid then obtained did not contain crystals because they had dissolved by the inflammatory cells.

The discovery of CPPD crystals in the synovial fluid aspirate of a four-year-old child presenting with transient synovitis/irritable hip is a provocative and possibly an important one. Could crystal deposition disease be one of the causes for synovitis in this young age group?

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