

Identical Twins with Legg-Calvé-Perthes Disease: A Report of Two Cases

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Learning Point of the Article:

Legg-Calvé-Perthes Disease may have a genetic contribution that should be further investigated.

Abstract

Introduction: Legg-Calvé-Perthes disease (LCPD) is an idiopathic avascular necrosis of the femoral head in children, with proposed etiologies, including genetic mutations and coagulation abnormalities. While familial clustering has been reported, the role of genetic predisposition remains incompletely understood, particularly in monozygotic twins.

Case Report: We report monozygotic male twins born at 35 weeks' gestation who developed LCPD sequentially. Twin A presented at 3 years and 4 months with a 2-week history of antalgic gait. Initial imaging was unremarkable, but follow-up radiographs demonstrated progressive changes consistent with LCPD, ultimately classified as lateral pillar Class A. He was managed conservatively with observation and showed clinical and radiographic improvement at 1 year. During Twin A's 1-year follow-up, Twin B presented with similar symptoms of antalgic gait and mild right hip pain. Imaging revealed early fragmentation and mild lateral pillar height loss, consistent with early-stage LCPD. Given the favorable course in Twin A, Twin B was also managed non-operatively.

Discussion: This case highlights the occurrence of LCPD in monozygotic twins raised in a shared environment, supporting a potential genetic contribution to disease pathogenesis. Prior studies have implicated hereditary thrombophilia and mutations in type II collagen in LCPD development. Although larger twin studies suggest low concordance rates, limited sample sizes restrict definitive conclusions. This case adds to existing reports suggesting a possible genetic predisposition, while also acknowledging the influence of shared environmental factors.

Conclusion: The sequential development of LCPD in identical twins underscores the importance of considering genetic susceptibility in disease etiology. Increased clinical vigilance and early evaluation in siblings may facilitate timely diagnosis. Further research is needed to better define the genetic mechanisms contributing to LCPD.

Keywords: Legg-Calvé-Perthes, Limping Child, Pediatric Orthopedics

Introduction

Legg-Calvé-Perthes disease (LCPD) is described as idiopathic avascular necrosis of the femoral head. Several etiologies have been proposed, including a mutation of type II collagen and thrombophilia causing necrosis of the femoral head [1]. LCPD has an incidence of approximately 1 in 10,000 children, affecting

boys 5 times as much as girls [2]. Children generally present at age 5–8 years with mild pain, a limp or decreased range of motion of the hip. Staging has been classified based on radiographic imaging (early sclerosis, fragmentation, re-ossification, and healing) [1]. The lateral pillar classification has been accepted as a prognostic indicator, with <50% of the height of the lateral

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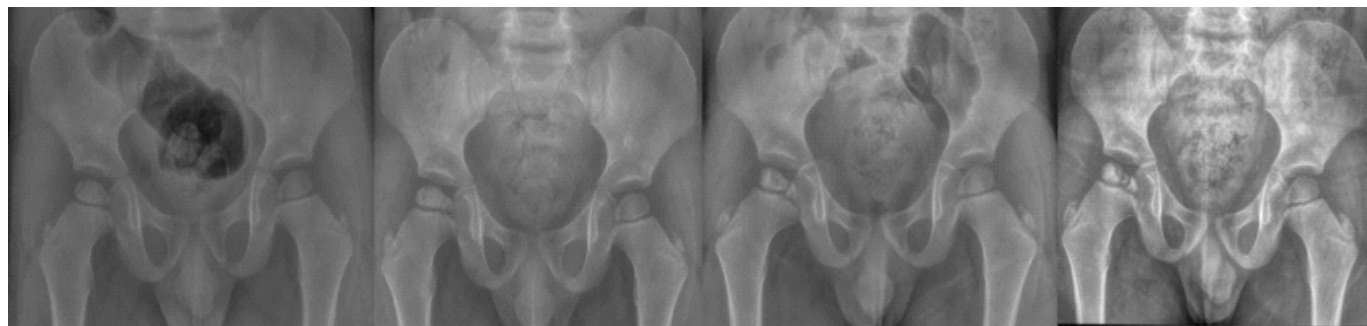


Figure 1: Sequential imaging of twin a at time of presentation, 3 months, 6 months and 1-year follow-up.

pillar of the femoral head indicative of poor outcomes [3]. Progression of the condition depends largely on the degree of femoral head deformation. Mild deformity may remain asymptomatic while more severe deformity may cause metaphyseal and acetabular changes, leading to pre-mature arthritis before the age of 50 [4].

Case Report

Twin A and Twin B are monozygotic males from a mother who received normal prenatal care and had no history of smoking. Each twin was raised in the same household. Pregnancy was complicated by pre-term pre-mature rupture of membranes at 35 weeks. Each twin was born through cesarean section at 35 weeks gestation. Twin A was born first, with no notable complications at birth. Twin B spent approximately 6 h on continuous positive airway pressure for decreased aeration at birth; however, there were no further complications at birth. Twin A presented to our office at 3 years 4 months of age with an antalgic gait for approximately 2 weeks in comparison to his brother, Twin B. Radiography at that time revealed no obvious abnormality to the bilateral hips. Lab work at that time, including inflammatory markers (erythrocyte sedimentation rate and C-reactive protein), complete blood count and comprehensive metabolic panel, which revealed an elevated alkaline phosphatase at 279 units/L, and no other significant

abnormalities. Twin A followed up in our office 2 weeks later with improvement of symptoms. Given his improvement and no obvious diagnosis at that time, the decision was made to continue to observe Twin A. At the 2-month follow-up, his symptoms had almost completely resolved. Radiography of his hips and pelvis revealed slight lucency of the right lateral femoral head at that time. He was then seen again at 6 months from symptom onset, where radiography revealed fragmentation of the right femoral head, lateral pillar Class A, and the diagnosis of LCPD was determined most likely. During his past follow-up at 1 year after initial presentation, he was found to be ambulating well without limitations. His radiography remained at lateral pillar Class A and was found to be consistent with the re-ossification stage (Fig. 1).

During the 1-year follow-up of Twin A, Twin B presented with approximately 2 weeks of antalgic gait and mild pain localized to the right hip. Radiography of the hips and pelvis revealed fragmentation of the medial aspect of the femoral head with slight height loss of the lateral femoral head, indicative of early LCPD. Twin B at 3-months follow-up continued to have a mild antalgic gait with minimal interval improvement. Given the fair prognosis of Twin A thus far, the decision was made to continue to observe Twin B in a similar fashion (Fig. 2).

Discussion

LCPD remains a condition of uncertain etiology, with environmental factors, such as second-hand smoke exposure and low socioeconomic status having been suggested to increase a patient's risk [5]. In recent years, research has aimed to identify genetic components associated with the development of LCPD. Given its rarity, documenting familial cases of LCPD, especially among siblings, is essential to expanding the data needed for larger studies on possible genetic contributions to its etiology. We present a case of identical twins developing LCPD confirmed by radiographic imaging.



Figure 2: Initial (left) and 3-month follow-up (right) imaging of Twin B.

Multiple genetic factors have been suggested to play a role in the development of LCPD. In the present literature, the most prominent genetic factor is the association of LCPD with hereditary thrombophilia. In a meta-analysis by Woratanarat et al., the presence of Factor V Leiden was shown to increase the risk of LCPD [6]. Other studies have reached similar conclusions. In a study of LCPD in Mexican patients, a significant difference in prothrombin time as well as Factor V and Factor IX levels were seen in those with LCPD [7]. In a study on 169 patients at two centers in the Netherlands, Factor V Leiden, prothrombin C20210A mutations, protein C deficiency and elevated Factor VIII were all significantly associated with LCPD, further suggesting genetic aberrations in coagulation increase a patient's risk of LCPD [8].

Interestingly, mutations in the type II collagen gene have also shown an association with LCPD. In a study of a Chinese family with known LCPD, a heterozygous mutation in exon 29 of COL2A1 was identified in family members specifically affected [9]. Similar familial studies have noted mutations at different sites of the COL2A1 gene in families with multiple family members affected by LCPD [10,11]. Interestingly, one case report also suggested an association of LCPD with osteogenesis imperfect [12].

Although genetic factors have been shown to increase the risk of developing LCPD, research specifically examining LCPD in twins remains limited. The largest twin study, involving 81 twin

pairs from the Danish Twin Registry, suggested a low likelihood of a distinct genetic contribution, as the concordance rate among monozygotic twins was low [13]. However, only 10 monozygotic twin pairs were included, limiting the strength of this conclusion. In contrast, multiple case reports have documented monozygotic twins both developing LCPD, supporting the possibility of a specific genetic component [14-16].

Conclusion

Given the potential influence of genetic factors in the development of LCPD, investigating familial associations remains essential. However, present literature offers relatively limited insight into the relationship between monozygotic twins and LCPD. Although constrained by the absence of genetic testing, our case contributes to the growing body of evidence suggesting a genetic component in the disorder's pathogenesis. Further research exploring the genetic mechanisms underlying LCPD is therefore warranted.

Clinical Message

There is a potential genetic contribution to LCPD pathogenesis. Recognition of a genetic component may warrant increased clinical vigilance and early screening in at-risk siblings to facilitate timely diagnosis and management.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given the consent for his/ her images and other clinical information to be reported in the journal. The patient understands that his/ her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

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