Meyer Dysplasia in the Differential Diagnosis of Hip Disease in Young Children

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Objectives: To describe a rare developmental disorder of the femoral capital epiphysis in infants and children that is often misdiagnosed and to suggest an evaluation protocol to differentiate it from other hip problems.

Design: Case series.

Setting: Tertiary care center.


Intervention: All clinical and imaging data were collected.

Results: Two of the 5 patients were initially diagnosed as having osteomyelitis and 3 as having Perthes disease. The diagnosis of Meyer dysplasia was confirmed by plain film of the pelvis, a negative bone scan, or normal bone marrow findings on magnetic resonance imaging. The limping resolved without treatment in all patients within 1 to 3 weeks.

Conclusions: Meyer dysplasia is a benign condition that should be included in the differential diagnosis of hip disease in infants and children. Awareness of this condition may prevent unnecessary hospitalization and treatment.


Editor's Note: Keep this in mind next time you see a limping toddler or preschooler.  

Catherine D. DeAngelis, MD

MEYER dysplasia is a symptomless developmental disorder of the hip manifested by delayed, irregular ossification of the femoral epiphysial nucleus.1 The dysplasia is noted during the second year of life and usually disappears by the end of the sixth year without treatment.2 This rare condition may easily be mistaken for other hip problems, leading to unnecessary diagnostic procedures and treatments.

We describe 5 children who were ultimately diagnosed as having Meyer dysplasia and suggest guidelines for the evaluation of this condition based on our findings and a review of the literature.

Table 1 presents clinical findings and Table 2 presents imaging findings in the 5 patients. The 5 children included 4 males and 1 female aged 9 to 48 months. All presented with acute onset of limping of 2 to 30 days’ duration. Patient 3 was being observed by a pediatric endocrinologist for short stature, and patient 4 had a family history of congenital dislocation of the hip and Perthes disease. Otherwise, the histories were unremarkable.

Physical examination revealed a decreased range of motion of the hip joint in 3 of the 5 patients; in 1 (patient 2), it was accompanied by fever. Acute-phase reactants (high erythrocyte sedimentation rate, high white blood cell count) were found in 2 patients (patients 1 and 2). Hip radiographs revealed various epiphysial changes compatible with Meyer dysplasia in all patients, 3 of whom had bilateral involvement (Figure 1).

The initial diagnosis in patients 1 and 2 was acute osteomyelitis, based on the clinical presentation and the presence of an elevated sedimentation rate with leukocytosis and radiolucent lesions of the femoral head. However, the normal findings on bone scan ruled out this possibility and supported the diagnosis of Meyer dysplasia.
The 3 other patients (patients 3, 4, and 5) were first suspected to have Perthes disease, based on the clinical presentation, lack of signs of infection, and the presence of epiphysial changes on the hip radiographs. Again, normal bone scan findings excluded this diagnosis. Because of a family history of Perthes disease in patient 4, magnetic resonance imaging was performed, revealing multiple centers of ossification of the femoral heads. The normal bone marrow intensity in all sequences ruled out edema and ischemia and confirmed the diagnosis of Meyer dysplasia (Figure 2).

In all patients, the limping resolved without treatment within 1 to 3 weeks. Bone survey did not disclose epiphysial dysplasias in other bones in any of the children. Results of thyroid function tests were normal. Bone age (by Greulich Pyle) was retarded in all patients.

Table 1. Clinical Findings in Meyer Dysplasia

<table>
<thead>
<tr>
<th>Patient No./ Sex/Age, mo</th>
<th>Clinical Presentation</th>
<th>Maximal Temperature</th>
<th>Physical Findings</th>
<th>Laboratory Findings*</th>
<th>Initial Diagnosis</th>
<th>Initial Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/9</td>
<td>Guarding of left leg</td>
<td>Normal</td>
<td>Limited internal rotation of left hip joint</td>
<td>ESR, mm/h: 60 WBC, $\times 10^9$/L: 15</td>
<td>Osteomyelitis</td>
<td>Cefuroxime</td>
</tr>
<tr>
<td>2/M/24</td>
<td>Limping in left leg</td>
<td>39.5° C</td>
<td>Tonsillitis, limited internal rotation of left hip joint</td>
<td>ESR, mm/h: 62 WBC, $\times 10^9$/L: 26</td>
<td>Osteomyelitis, tonsillitis</td>
<td>Cefuroxime</td>
</tr>
<tr>
<td>3/M/48</td>
<td>Limping, waddling gait</td>
<td>Normal</td>
<td>Normal</td>
<td>ESR, mm/h: 25 WBC, $\times 10^9$/L: 8.8</td>
<td>Perthes disease</td>
<td>None</td>
</tr>
<tr>
<td>4/M/30</td>
<td>Limping in left leg</td>
<td>Normal</td>
<td>Limited internal rotation of left hip joint</td>
<td>ESR, mm/h: 25 WBC, $\times 10^9$/L: 10</td>
<td>Perthes disease</td>
<td>None</td>
</tr>
<tr>
<td>5/M/36</td>
<td>Limping</td>
<td>Normal</td>
<td>Normal</td>
<td>ESR, mm/h: 11 WBC, $\times 10^9$/L: Not done</td>
<td>Perthes disease</td>
<td>None</td>
</tr>
</tbody>
</table>

*ESR indicates erythrocyte sedimentation rate; WBC, white blood cell count. Cefuroxime was given as cefuroxime sodium.

Table 2. Imaging Findings of the Hip Joints in Meyer Dysplasia

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Affected Side</th>
<th>X-Ray Film</th>
<th>Bone Scan</th>
<th>Ultrasound</th>
<th>Magnetic Resonance Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Left</td>
<td>Small, irregular epiphysis with radiolucent lesion</td>
<td>Normal</td>
<td>Not done</td>
<td>Not done</td>
</tr>
<tr>
<td>2</td>
<td>Left</td>
<td>Flattening, irregularity, sclerosis, and radiolucent foci of femoral head</td>
<td>Normal</td>
<td>Fluid within joint</td>
<td>Not done</td>
</tr>
<tr>
<td>3</td>
<td>Bilateral</td>
<td>Bilateral dysplastic changes of femoral heads</td>
<td>Normal</td>
<td>Not done</td>
<td>Not done</td>
</tr>
<tr>
<td>4</td>
<td>Bilateral</td>
<td>Bilateral small and irregular femoral heads</td>
<td>Normal</td>
<td>Normal</td>
<td>Multiple centers of ossification of femoral heads (with normal bone marrow intensity)</td>
</tr>
<tr>
<td>5</td>
<td>Bilateral</td>
<td>Bilateral fragmentation of femoral heads</td>
<td>Normal</td>
<td>Not done</td>
<td>Not done</td>
</tr>
</tbody>
</table>

Pedersen and Meyer were the first to describe a subgroup of patients with Perthes disease who showed a distinct radiological and clinical picture found rarely among the clinical conditions affecting the hip joint in childhood. Meyer estimated that this subgroup accounted for 10% of all patients with Perthes disease and was characterized by a male predominance and young age (<5 years). He reported that this dysplasia is noted during the second year of life and usually disappears by the end of the sixth year. It is more often bilateral than typical Perthes disease (42% vs 7%) and has a higher familial frequency of other hip diseases such as congenital dislocation and Perthes disease. Meyer traced the pathogenesis to circulatory disturbances that caused delayed, irregular ossification. Other authors have attributed the dysplasia to congenital focal hypoplasia of the femoral epiphysis.

The possibility that we are dealing with a physiologic variant of ossification of the femoral head cannot be ruled out in those who completely recover with normal epiphysial shape and size.

Radiologically, affected patients show a marked delay in the development of the femoral epiphysial nucleus and its pathological appearance at a later date. Maturation is retarded in other parts of the skeleton as well. In Meyer dysplasia, ossification of the femoral head does not usually set in until 2 years of age. The ossification cen-
ter, instead of the normal single unit, appears as a small epiphyseal nucleus composed of multiple independent bony foci. These foci gradually grow, coalesce, and finally fuse into a single center. A small, single ossification center with a cortical defect on the articular surface has also been described.

Magnetic resonance imaging shows multiple centers of ossification of the femoral head, with a normal signal intensity in all sequences and reduced height of the cartilaginous epiphysis. Normal findings of bone scanning are typical for Meyer dysplasia. The differential diagnosis consists mainly of Perthes disease, which is characterized by both abnormal bone scan findings and bone marrow signals in the femoral head on magnetic resonance imaging. Uniform massive condensation on hip radiographs in Perthes disease is another feature that distinguishes it from Meyer dysplasia, and that does not require expensive imaging techniques. However, this finding is usually a late sign and therefore not helpful in the early stages. Whenever bilateral changes are present, multiple epiphyseal dysplasia and hypothyroidism should be excluded.

Despite the prominent radiological findings, clinical signs are usually mild or absent, and the lesions are usually discovered incidentally. Some patients with bilateral involvement may have a mild inconsistent waddling gait. Pain, limping, or limitation of movement are rarely observed and are usually transient.

As healing is complete, Meyer dysplasia needs no treatment. Patients show continuous improvement with steady unification and growth of the epiphysis, usually over a 3-year period. The final outcome is a normal hemispherical shape and size of the femoral head, although some have reported diminished height. Function is probably normal. Nevertheless, biomechanically induced degenerative joint changes may be anticipated in adulthood in those with smaller femoral heads. In Perthes disease, by contrast, the natural course is associated with increased fragmentation that may result in permanent deformity of the femoral head if left untreated. However, in children younger than 5 years, the disease is quite benign and usually requires no intervention.

The results of a recent prospective, clinical, and radiological study of 18 children with Meyer dysplasia have confirmed most of Meyer’s observations. Half the cases were bilateral and boys were affected 5 times more often than girls. The imaging studies showed a delayed appearance of the ossification centers, with complete healing by an average age of 5½ years, except for a slight loss in epiphysial height. Most of the patients were asymptomatic. Although 6 (20%) of Meyer’s 30 patients showed a shift from the benign course of dysplasia to Perthes disease, no cases of Perthes disease were observed in this study. This was also true for another case of bilateral femoral dysplasia, which had a favorable natural course.

We described 5 patients with Meyer dysplasia, 2 of whom were initially misdiagnosed as having osteomyelitis and 3 of whom were thought to have Perthes disease. The diagnosis of Meyer dysplasia in our patients was confirmed by the normal bone scan findings, normal bone marrow signal on magnetic resonance imaging, and complete resolution of the limping. Since all our patients had been asymptomatic in the past, we assume that the acute onset of limping may have been due to an acute event, such as toxic synovitis, unrelated to Meyer dysplasia. The elevation of the sedimentation rate in patients 1 and 2 may be attributed to an intercurrent infection that resolved without treatment.
Once diagnosed, these patients should be followed up both clinically and radiographically (with plain films) by the pediatric orthopedic surgeon. If there is not clinical deterioration (limping, limited joint movement), hip radiographs should be performed once a year until resolution is noted.

In conclusion, Meyer dysplasia is a rare condition with a benign clinical course that may mimic other, more severe hip diseases. A greater awareness of this condition would prevent unnecessary hospitalization and treatment and allay parental fears regarding the prognosis.

Accepted for publication February 8, 1999.

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REFERENCES


Error and Omission in Text. In the article by Strauss titled “Self-reported Weight Status and Dieting in a Cross-sectional Sample of Young Adolescents: National Health and Nutrition Examination Survey III,” published in the July issue of the ARCHIVES (1999;153:741-747), an error and an omission appeared in the text. On page 742, in the “Sample” subsection of the “Subjects and Methods” section, the fourth sentence should have read as follows: “Of the children enrolled, 1097 (56.8%) were white, 743 (38.5%) were black, and 92 (4.8%) were other.” Also, on that same page in the “Statistics” subsection of the “Subjects and Methods” section, a new sentence should have been inserted after the second sentence and read as follows: “Whites and Hispanics were grouped together because of sample size limitations and herein are identified as ‘white.’”