Case Report

Premature Physeal Closure after a Non-Displaced Physeal Fracture of Distal Fibula

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ABSTRACT

Premature physeal closure (PPC) may lead to clinically significant progressive angular deformity or leg length discrepancy. Many variables seem to play a role in determining which injuries result in PPC. A 8-year-old boy sustained a non-displaced physeal fracture of distal tibia and fibula. He showed no signs of PPC at 7 months post-injury. Seven years later, his ankle became painful. He had developed PPC of distal fibula causing angular ankle deformity, which was treated successfully by corrective osteotomy. To our knowledge, this is the first reported case of a non-displaced fracture of the distal fibula leading to PPC several years after the initial injury, which in our understanding was impossible to predict.

Background

Distal tibial and fibular physis are responsible for approximately one-third of the final length of the lower leg producing between 3-5 mm of longitudinal growth per year [1]. Distal tibia physis is the second most common location of physeal fractures [2]. Risk of premature physeal closure (PPC) of the injured distal tibial growth plate has varied between 5-42% [3-7]. The incidence of the physeal injuries of distal fibula and the related risk of PPC is not known. PPC may lead to clinically significant progressive angular deformity or leg length discrepancy depending on its location and extent [8]. After physeal injury has been established, a repeat radiography is recommended every 3 months until normal growth has been documented for at least 6 months [8]. Many variables seem to play a role in determining which injuries result in PPC: Mechanism of injury, trauma energy, fracture pattern and primary and post-reduction displacement [3-6, 9-12].

Case Report

A 8-year-old previously healthy boy injured his ankle from a fall while playing football in July 2008. Bony injuries were not seen on radiographs, but non-displaced Peterson III physeal fractures of the tibia and fibula were suspected (Figure 1a). He was treated with a short leg cast for 5 weeks. His growth plates looked normal in follow-up radiographs 7 months after the injury (Figure 1b). In May 2015, he twisted his ankle during football practice. Prior to this new injury, he had experienced progressive ankle pain but had been able to play football with his ankle taped. Radiographs showed now a total PPC of the fibula and abnormal distal tibial physis and a substantial shortening of the fibula and angular ankle deformity (Figure 1c). He was then referred to our hospital, where a complete fibular PPC and a vertical cavity in the distal tibial physis were diagnosed by a CT scan. His anterior distal tibia angle (ADTA) was abnormal in the lateral part of the tibia, in addition to significant valgus angulation. He had a clinically obvious valgus deformity and instability of his ankle joint.

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MRI showed damage of the tibial joint surface, which was verified by arthroscopy (Alter-Bridge level I damage of distal tibial including the medial malleolus). Closing wedge osteotomy of the tibia to correct both the valgus and ADTA and intraoperative 10 mm lengthening of fibula were performed (Figure 1d). ADTA was intentionally slightly under corrected laterally. The patient’s recovery was uneventful (Table 1) and he went back to playing football. His hardware was removed in December 2017. He remained symptomless despite slight instability of his ankle in the frontal plane at the last follow-up.

Table 1: Recovery after corrective surgery of ankle deformity caused by premature physeal closure of distal fibula in a 15-year-old boy 7 years after the injury.

| Examiner               | Function                                                                 |
|------------------------|--------------------------------------------------------------------------|
| Six weeks              | Orthopaedic surgeon                                                      |
| Walks with a limp.     | Stable ankle.                                                            |
| Two months             | Physiotherapist                                                          |
| Does not need pain     | medication. Walks without crutches. Decreased ankle range of motion.     |
| Four months            | Physiotherapist                                                          |
| Normal gait and        | symmetrical ankle joint range of motion.                                 |
| Six months             | Orthopaedic surgeon                                                      |
| Returned to work and   | sports 4 months from surgery.                                            |
| Eighteen months        | Orthopaedic surgeon                                                      |
| Discomfort from        | hardware. Osteotomy completely healed on radiographs                     |

Figure 1: A 8-year-old boy sustained an ankle injury without radiographic evidence of fractures a) anteroposterior and lateral radiographs at the time of injury, b) 7-months later, c) 7-years later and d) after corrective osteotomy.

Discussion

To our knowledge, this is the first reported case of a non-displaced Peterson III (SH-I) fracture that developed a significant angular deformity several years after the initial injury. The most significant causative factors of PPC are high trauma energy and fracture displacement [5, 13-15]. Non-displaced Peterson III are believed to be low-risk fractures that are treated in a short leg, non-weight-bearing cast for 4 to 6 weeks [6, 16]. Our patient probably sustained a non-displaced physeal fracture of the distal fibula from a low energy trauma, which led to a premature physeal arrest and significant angular deformity caused by the shortened fibula. We believe that due to fibular PPC traction from the syndesmotic ligaments and interosseous membrane caused the growth disturbance in the distal tibial growth plate.

Early recognition of PPC is important in order to prevent functionally significant deformity. Asymmetric Harris lines can predict PPC already at three months, whereas angular deformity becomes evident later but usually during the first 2 years from the injury [12, 17, 18]. We do not know when our patient’s ankle deformity started to develop since he was discharged at 7 months from the injury when no signs of PPC were seen clinically or on radiographs. We do not arrange further follow-up or imaging studies for asymptomatic patients with open normal looking physis and symmetric Harris lines. PPC might have been diagnosed earlier. Follow-up would have been longer, but it is not reasonable to follow-up on all patients for several years. Further research is needed to improve our understanding of the predictive factors of PPC and follow-up protocols in physeal fractures.
Conflicts of Interest

None.

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