CASE REPORT

Knee Flexion Deformity and Increased Posterior Tibial Slope Associated with Osgood Schlatter Disease: A Case Report and Review of Literature

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Abstract

Genu recurvatum associated with Osgood-Schlatter disease (OSD) has been reported in several studies. In this report, we describe a rare complication of a case of OSD with flexion contracture (tfighat is the exact opposite of the knee deformity classically associated with OSD) and increased posterior tibial slope.

In the current article, we report a 14-year-old case of OSD referred to our center with a fixed knee flexion contracture. Radiographic evaluation revealed a tibial slope of 25 degrees. There was no limb length discrepancy. Bracing that was prescribed in the primary center

before referring to us was not successful in treating this deformity. He underwent anterior tibial tubercle epiphysiodesis surgery. After a year, the flexion contracture of the patient was significantly reduced. The tibial slope decreased by 12 degrees and reached 13 degrees.

The present report suggests that OSD may affect the posterior tibial slope and lead to knee flexion contracture. Surgical epiphysiodesis can correct the deformity.

Level of evidence: IV

Keywords: Flexion deformity, Genu recurvatum, Osgood-Schlatter, Posterior Tibial slope, Tibial tubercle apophysitis

Introduction

Osgood-Schlatter disease (OSD) was first introduced in 1903 by Robert Osgood, and Carl Schlatter is an overuse condition of the knee, causing pain and tenderness in the tibial tuberosity.[1] It is now described as apophysitis at the anterior part of the tibial tubercle.[2] It is a common disorder of the anterior knee in growing children due to repetitive microtrauma caused by frequent quadriceps contraction at the attachment site of the patellar tendon to the unfused tibial tubercle apophysis.[3,4]

OSD is a self-limited condition that often presents insidious onset of activity-dependent pain, tenderness, and prominence over the tibial tubercle. It generally involves girls and boys at the age of 8-13 and 10-15 years, respectively.[4] Several risk factors have been proposed for OSD, including weight, quadriceps muscle tightness, and the flexibility of the hamstring muscles.[5] It is more common in the athletic population repeatedly perform physical stress-causing activities such as jumping and sprinting.[3] Sports such as soccer, basketball, and volleyball are associated with a higher risk of OSD.[4] Regular assessment of these groups of athletes for muscle strength and flexibility could reduce the incidence rate of OSD and its complications by implication of training regimens balancing the load on the tibial tubercle.[6]

The diagnosis is mainly centered on clinical symptoms. Besides, the radiographic evaluation may be helpful to rule out other differential diagnoses and when it is a

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concern about other diagnoses.[7] The overall treatment is dictated according to the level of pain and includes the management of symptoms with ice, non-steroidal anti-inflammatory drugs (NSAIDs), and activity modification.[3]

According to Léonard et al., in nearly 10% of cases, OSD results in a range of complications.[8] These complications mainly include nonunion of the bony fragment to the tibia, pseudarthrosis, patellar subluxation, patellar tendon avulsion, patella Alta, and genu recurvatum.[9]

Physeal arrest of the proximal tibia and its subsequent genu recurvatum has been attributed to several conditions such as undiagnosed trauma, prolonged immobilization of the knee in the cast, and tibial traction. OSD has also been reported as a condition causing premature fusion of tibial epiphysis and genu recurvatum.[10]

To the best of our knowledge, flexion deformity associated with OSD has not been reported in English literature. This report presents a rare presentation of OSD complicated with flexion deformity, which is the opposite of the knee deformity classically associated with OSD. We also describe the management approach and treatment outcome in such a rare complication of OSD. Informed parental permission for this publication is obtained.

Case Presentation

A 14-year-old male case of OSD was referred to our orthopedic department with a clinical and radiologic knee flexion contracture [Figure 1]. Diagnosis of OSD was made based on patient history and physical examination findings two years ago at another center.

He had no history of trauma, infection, or family history relevant to knee deformity. To conservatively treat the OSD, NSAIDs consumption, bracing, and activity modulation was prescribed in another primary center for two years, but no improvement was achieved.

Parents reported a gradual increase of knee flexion deformity on the right side but no progress of deformation on the left knee. On the clinical examination, fixed flexion deformity of the right knee was observed without coronal deformity. Hamstring tightness and gastrocnemius muscle contracture were ruled out by appropriate physical testing and confirmed under anesthesia preoperatively. Tenderness on the tibial tubercle was evoked on both sides, more pronounced on the right knee. The patient complained of activity-dependent pain. No limb length discrepancy was detected. The ligamentous examination was normal in both knees. The right tibial tubercle physeal plate seemed active in radiographic evaluation, while the posterior tibial slope was abnormal at 25 degrees. The contralateral knee (left side) also had an active tibial tubercle physeal plate and an upper limit normal posterior tibial slope of 12 degrees [Figure 2].

The patient was first treated conservatively with a knee brace for three months, but the deformity progressed. Since the conservative approach did not lead to good results, surgery was scheduled.

We aimed to correct the deformity by preventing the tibial tubercle growth from diminishing its possible stimulatory effect on the anterior part of the proximal tibial physeal plate. We approached the anterior tibial physis through a 4-centimeter incision, just medial to the tibial tubercle.

Subsequently, the proximal physeal plate was located and explored under fluoroscopic control. Tibial tubercle epiphysiodesis was performed by removing the physeal plate using a curette and inserting a 4-millimeter cancellous screw [Figure 3].

The knee was immobilized in a knee immobilizer after surgery for four months. However, intermittent early range of motion (ROM) was initiated from the first postoperative day. Full weight-bearing with walking crutches -as a pain reliever- was allowed immediately after the surgery. Exercises to improve the joint ROM began afterward. The patient was visited sequentially for one year. During the follow-up period, the knee flexion deformity was gradually corrected. Furthermore, the posterior tibial slope was decreased by 13 degrees on

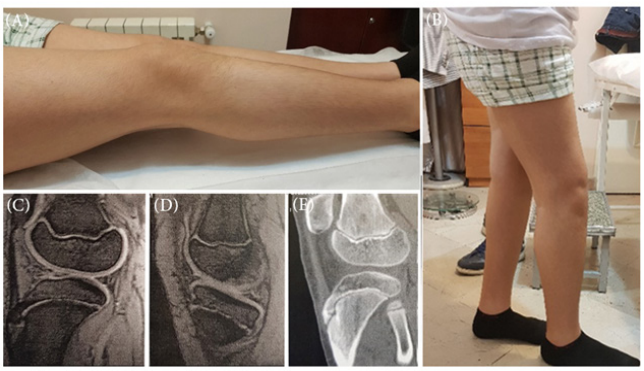

Figure 1. Preoperative photographs (A & B), MRI (C), CT scan (D), and radiograph of the patient showing a knee flexion contracture.

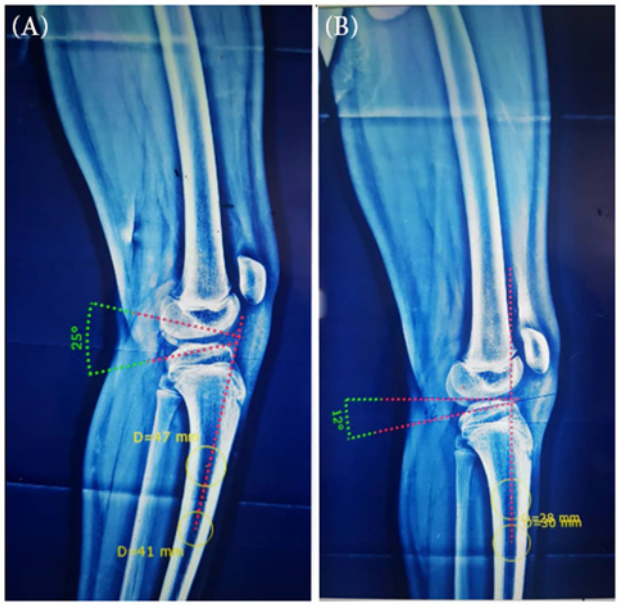

Figure 2. Preoperative lateral radiographs showing a posterior tibial slope of 25º in the involved knee (A) and 12 º in the contralateral knee (B).

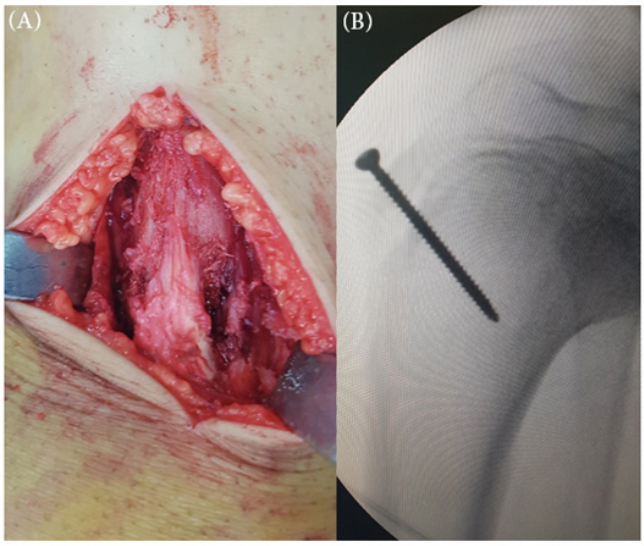

Figure 3. Intraoperative photograph (A) and lateral fluoroscopic view.

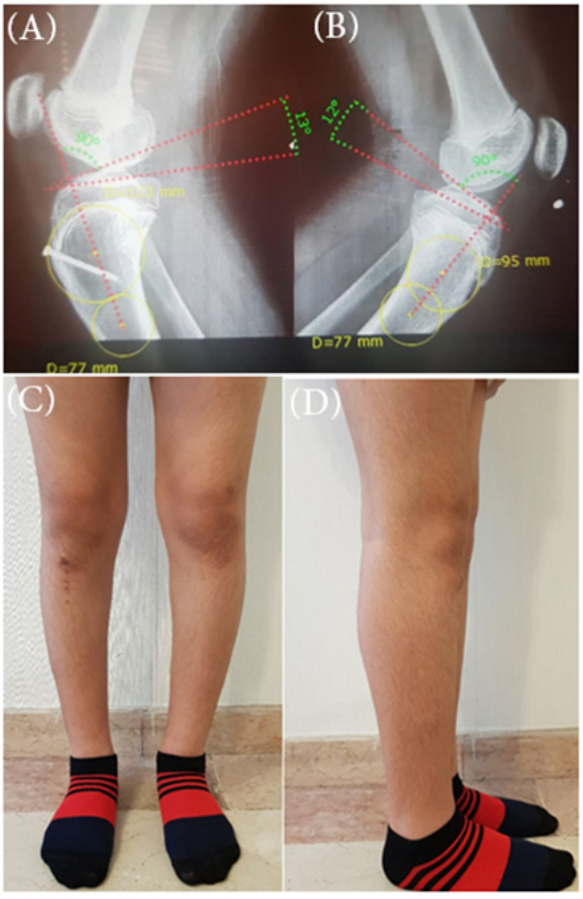

Figure 4. Lateral radiographs show the posterior tibial slope of the involved knee (A) and contralateral knee (B) at the final follow-up; Anteroposterior and lateral photographs of the patient's knees at the final follow-up (C & D).

final postoperative radiographs [Figure 4]. Postoperative complication was not detected, and the limb length was preserved.

Discussion

In this report, we presented a 14-year-old boy with bilateral OSD associated with fixed right knee flexion deformity and increased posterior tibial slope, which are rare complications of this entity. The patient was treated by epiphysiodesis of the tibial tubercle physeal plate. One year after the surgery, the flexion contracture was significantly improved. The tibial slope was decreased by 12 degrees.

Based on several studies, pro-inflammatory cytokines that are released following the inflammatory conditions, such as tumor necrosis factor α, interleukin 1β, and IL6, directly affect the growth plate to induce apoptosis and suppress bone growth. Induction of the growth plate and its subsequent flexion contracture secondary to OSD, as was reported in this study, might contradict this evidence. One explanation could be the balance of proinflammatory and anti-inflammatory factors released in the inflammation site, which needs to be further investigated.[2] In other words, OSD might result in the upregulation of anti-inflammatory factors in some cases. However, this hypothesis needs to be evaluated in-depth.

As a complication of OSD, growth disturbance has been reported in a few studies. In 1965, Jeffreys reported a case of bilateral OSD in an 11-year-old boy who was referred with activity-dependent pain, enlargement, and tenderness in tibial tuberosity. He was treated conservatively.

However, their patients returned with pain recurrence in the right knee four years later. In the radiographic evaluation, the physis was beginning to fuse. The pain was relieved after a month with no intervention. The patient returned with a nearly 20 degrees genu recurvatum of the right knee two years later. The anterior part of the upper right tibial epiphysis in the radiographs was fused.[11]

Bellicini et al. reported a case of genu recurvatum secondary to OSD in a 15- year-old boy presented with right knee pain. The knee was in 20 degrees of hyperextension. The affected leg was 1.5 cm shorter than the contralateral side. The deformity was not improved by bracing. Therefore, it was managed by proximal tibial osteotomy and distraction with a Taylor spatial frame. The genu recurvatum was corrected in the follow-up radiographs, and the limb length discrepancy was resolved. Symmetric full ROM was achieved.[12] Closure of the proximal tibial physis with subsequent genu recurvatum secondary to OSD has been reported in many other studies.[12-16]

Similarly, Green et al., in a recent study, showed an association between posterior tibial slope and OSD, but they did not find any cause-and-effect relationship.[17]

In the current study, the patient had signs and symptoms of OSD at the onset of diagnosis. He has experienced a gradual increase in his knee flexion deformity and posterior tibial slope, which confirms that OSD is the only justification for these deformities. In addition, the study conducted by Green et al. suggested that evaluating tibial slope in high-demand athletic adolescents with

OSD through long leg lateral knee and leg x-ray may prevent increasing posterior tibial slope and subsequent anterior cruciate ligament (ACL) injury in these groups of patients.[17] Since the pathologic increase in tibial slope predisposes patients to an ACL injury, physeal modulation surgery, as a minimally invasive technique with less complication than osteotomy, can be beneficial and must be considered after physeal closure at an older age.

A variety of etiologies can describe the occurrence of knee flexion contractures, such as hamstring tightness, quadriceps weakness, and neurological disorders. Knee flexion contracture could also be a compensatory mechanism for pes calcaneus, pes equinus, or fixed hip flexion.18 To the best of our knowledge, flexion contracture associated with OSD is not reported in 154 the earlier studies. Similar to the genu recurvatum, a cause-and-effect relationship between OSD and flexion deformity cannot be concluded according to the present report. Still, the sequence of symptoms, progressive knee deformity, and impressive deformity correction after epiphysiodesis can introduce the effect of OSD on this deformity. On the other hand, Perry et al. illustrated that knee flexion contracture directly increases the quadriceps force on the tibial tubercle, which may be an underlying cause of OSD applied to the knee.[19] However, flexion contracture was not observed in this case at the onset of the diagnosis of OSD.

We would follow this patient and consider epiphysiodesis of the entire proximal tibial physis in the future to avoid a progressive decrease of the tibial slope due to continued posterior growth. If we observe over-correction, we will consider arresting the reminding of the physeal plate to prevent the reversing of slope and genu recurvatum.

For future cases, we would recommend surgeons not to repeat the same procedure and consider a much less percutaneous insertion of screws across the physis of the tibial tubercle to achieve that same result with much less tissue injury.

The present report suggests that OSD may affect the posterior tibial slope leading to knee flexion deformity. Surgical epiphysiodesis can be an effective approach to correcting this deformity.

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References

1. Maher PJ, Ilgen JS. Osgood-Schlatter disease. BMJ Case Rep. 2013; 2013:bcr2012007614. doi:10.1136/bcr-2012-007614
2. Sailly M, Whiteley R, Johnson A. Doppler ultrasound and tibial tuberosity maturation status predicts pain in adolescent male athletes with Osgood-Schlatter's disease: a case series with comparison group and clinical interpretation. Br J Sports Med. 2013; 47(2):93-97. doi:10.1136/bjsports-2012-091471
3. Vaishya R, Azizi AT, Agarwal AK, Vijay V. Apophysitis of the Tibial Tuberosity (Osgood-Schlatter Disease): A Review. Cureus. 2016; 8(9):e780. doi:10.7759/cureus.780
4. Ladenhauf HN, Seitlinger G, Green DW. Osgood-Schlatter disease: a 2020 update of a common knee condition in children. Curr Opin Pediatr. 2020; 32(1):107-112. doi:10.1097/MOP.0000000000000842
5. Nakase J, Goshima K, Numata H, Oshima T, Takata Y, Tsuchiya H. Precise risk factors for Osgood-Schlatter disease. Arch Orthop Trauma Surg. 2015; 135(9):1277-1281. doi:10.1007/s00402-015-2270-2
6. Itoh G, Ishii H, Kato H, Nagano Y, Hayashi H, Funasaki H. Risk assessment of the onset of Osgood-Schlatter disease using kinetic analysis of various motions in sports. PLoS One. 2018; 13(1):e0190503. doi:10.1371/journal.pone.0190503
7. Jamshidi K, Mirkazemi M, Izanloo A, Mirzaei A. Benign bone tumours of tibial tuberosity clinically mimicking Osgood-Schlatter disease: a case series. Int Orthop. 2019; 43(11):2563-2568. doi:10.1007/s00264-019-04397-9
8. Léonard JC, Albecq JF, Leclet H, Morin C. Complications de la maladie d'Osgood-Schlatter: les pièges d'une maladie réputée banale Complications of Osgood-Schlatter's disease: a certainly deceitful disease. Science & sports. 1995; 10(2):95-101. doi.org/10.1016/0765-1597 (96)89533-5
9. Ogden JA,eds. Skeletal injury in the child. 3st ed. Springer Science & Business Media; 2006.
10. Bellicini C, Khoury JG. Correction of genu recurvatum secondary to Osgood-Schlatter disease: a case report. Iowa Orthop J. 2006; 26:130-133.
11. Jeffreys TE. Genu recurvatum after Osgood-Schlatter's disease: Report of a case. J Bone Joint Surg Br. 1965; 47:298-299.
12. Docquier J, Soete P, Twahirwa J, Piotte M. Genu recurvatum et maladie d'Osgood-Schlatter. Acta Orthop Belg. 1985; 51(6):945-54.
13. Lynch MC, Walsh HP. Tibia recurvatum as a complication of Osgood-Schlatter's disease: a report of two cases. J Pediatr Orthop. 1991; 11(4):543-544. doi:10.1097/01241398-199107000-00025
14. Pappas AM, Anas P, Toczylowski HM Jr. Asymmetrical arrest of the proximal tibial physis and genu

recurvatum deformity. J Bone Joint Surg Am. 1984; 66(4):575-581.
15. Zimbler S, Merkow S. Genu recurvatum: a possible complication after Osgood-Schlatter disease. Case report. J Bone Joint Surg Am. 1984; 66(7):1129-1130.
16. Sederquist B, Fernandez-Vojvodich P, Zaman F, Sävendahl L. Recent research on the growth plate: Impact of inflammatory cytokines on longitudinal bone growth. J Mol Endocrinol. 2014; 53(1):T35-T44. doi:10.1530/JME-14-0006
17. Green DW, Sidharthan S, Schlichte LM, Aitchison AH, Mintz DN. Increased Posterior Tibial Slope in Patients With Osgood-Schlatter Disease: A New Association. Am J Sports Med. 2020; 48(3):642-646. doi:10.1177/0363546519899894
18. Cottalorda J, Violas P, Seringe R; French Society of Pediatric Orthopaedics. Neuro-orthopaedic evaluation of children and adolescents: a simplified algorithm. Orthop Traumatol Surg Res. 2012; 98(6 Suppl):S146-S153. doi:10.1016/j.otsr.2012.04.015
19. Perry J, Antonelli D, Ford W. Analysis of knee-joint forces during flexed-knee stance. J Bone Joint Surg Am. 1975; 57(7):961-967.