A 13-year-old boy sustained an avulsion fracture of the left greater trochanter eight months after surgical stabilisation of a slipped capital femoral epiphysis on the same side. In this specific case, avulsion of the greater trochanter after slipping of the capital femoral epiphysis may have been facilitated by weakening of the trochanteric physis.

INTRODUCTION

Apophyseal avulsion fractures of the pelvis and lesser trochanter are well recognised in the existing literature (6, 14, 15). However, avulsion fractures rarely involve the greater trochanter (1, 11), presumably because of the high mechanical forces which are necessary to avulse the greater trochanter. Hormonal changes at puberty combined with biomechanical stresses due to obesity have been implicated as possible causative factors for slipped capital femoral epiphysis (5, 10, 12, 13, 20). However, they are not considered as a possible cause for apophyseal fractures. We have treated a 13-year-old boy with an avulsion fracture of the left greater trochanter, which occurred 8 months after surgical stabilisation of a slipped capital femoral epiphysis. In this specific case, possible weakening of the greater trochanteric physis may have been a contributing factor for its avulsion after slipped capital femoral epiphysis (SCFE).
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Fig. 1a. — Frog-leg lateral view of the left hip: there is a grade I slip with a femoral head-shaft angle of 17° according to the Southwick method.

(fig 1a) demonstrated a stable grade I slipped capital femoral epiphysis with a 17° femoral head-shaft angle according to the Southwick method (18). There was no evidence of bone remodeling at the junction between femoral head and neck. MRI was performed using standard pulse sequences (T1-weighted spin echo, fat-suppressed T2-weighted spin echo, and STIR) in order to exclude bilateral involvement. MRI confirmed a minimal left-sided SCFE with morphologic distortion of the physis, bone marrow oedema in the adjacent metaphysis and epiphysis, and joint effusion (fig 1b). The physis of the greater trochanter apophysis appeared normal. Endocrinopathies were ruled out by hormonal investigations. The femoral head was fixed in situ with a single percutaneous 7-mm cannulated screw (Synthes AO/ASIF) under fluoroscopic control (fig 2). The patient made an uneventful recovery.

Eight months postoperatively, as he was landing after jumping over a barrier, he felt pain in his left hip once again. Physical examination revealed tenderness on palpation over the greater trochanter. No limitation of passive hip motion was present, but he reported acute pain on active abduction of the hip and during the Trendelenbourg manoeuvre. An anteroposterior radiograph of the pelvis showed an apophyseal avulsion of the greater trochanter with slight displacement (fig 3). As the presence of the stainless steel screw would have interfered with MRI study, a technetium bone scan was performed and ruled out a vascular impairment of the femoral capital epiphysis; it showed increased uptake in the greater trochanter physis (fig 4). The patient was treated successfully with non-weight bearing on the left lower extremity for 8 weeks.

Seven months later, he developed right hip pain that increased during physical activity and was relieved by rest. Radiographs revealed a stable grade I slipped capital femoral epiphysis. He
underwent *in situ* percutaneous fixation with a 7-mm screw on the right side.

**DISCUSSION**

Slipped capital femoral epiphysis is related to puberty and usually occurs during the adolescence growth phase (17). Obesity is commonly noted, as is male predominance (8). SCFE is usually secondary to multiple factors. These result in a weakened physis that is loaded with higher than normal shear stresses. Displacement of the capital femoral epiphysis occurs in the widened hypertrophic zone, which has become weakened due to alterations in chondrocyte maturation and endochondral ossification (2, 7). Histological and histochemical changes have been noted in adolescents with slipped capital epiphysis and with abnormal widening of the growth plate (7). Widening of the growth plate can be of major magnitude and may contribute to further slippage (7). It is also worth noting, that the physeal perichondral ring, which functions as a stabiliser for the physeal plate, decreases in strength during adolescence (4). The physiological hormonal changes occurring during puberty alter physeal physiology and could be considered as a possible aetiologic factor for slippage (10, 12, 20). Hypogonadism, hypothyroidism, decreased vitamin D intake or absorption, increased levels of parahormone, ischaemia of the metaphysis, and deficient enchondral ossification represent other pathological factors which increase the width of the physis and alter its strength (16). Mechanical factors contributing to slipping of the capital epiphysis include obesity, a retroverted femoral neck, and an oblique orientation of the physis. These result in increased shear forces across the capital femoral epiphysis (5, 13).

Avulsion fractures at muscle insertions on the pelvis and of the lesser trochanter are uncommon but these injuries are becoming more and more frequent due to the greater number of adolescents participating in athletics (6, 14, 15). Avulsion fractures occur in adolescents participating in sports and are usually caused by powerful contraction, unbalanced contraction or overpull of the attached muscle (15). Avulsion fractures of the greater trochanter are rare (1, 11), even in sporting activities, which may be due to the fact that the abductors do not apply as much stress as other muscles like hamstrings and quadriceps during normal physical activity (11). Many reports have noted an increased prevalence of physeal fractures and apophyseal

**Fig. 3.** — The boy experienced recurrence of left hip pain after a jump. Apophyseal avulsion of the greater trochanter with slight displacement was noted on the AP pelvis radiograph.

**Fig. 4.** — Technetium bone scan showed increased uptake over the apophyseal cartilage of the greater trochanter.
avulsion fractures during adolescence. This seems to support the existence of a temporary physeal weakening during puberty (3, 6, 14, 15). In slipped capital femoral epiphysis, transient weakness of the growth plate during adolescence has been suggested by the high frequency of bilateral involvement and by the age at which the condition usually occurs (9). Other case reports seem to confirm the hypothesis of a physeal weakness. Association of tibia vara with slipped capital femoral epiphysis has been reported by Yanici et al and Takikawa et al (19, 21). Slipped capital femoral epiphysis and Blount’s disease are felt to have histopathological similarities (19, 21). These factors have led us to postulate that there may be a generalized physeal weakness and that every physis or apophysis could potentially be at risk for slipping or avulsion if mechanical constraints exceed the load tolerance of this structure.

CONCLUSIONS

Hormonal changes occurring during puberty contribute to an abnormal widening of the growth plate, which has been recognised as resulting in weakening of the physis. Lesions could thus occur without evidence of an endocrinologic disturbance, due to excessive mechanical forces exerted on the epiphysis or apophysis with regard to their load tolerance. Slipped capital femoral epiphysis is the lesion that occurs with the greatest frequency. However, every physis or apophysis may be at risk for such a lesion if mechanical constraints exceed its load tolerance.

REFERENCES