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Slipped Capital Femoral Epiphysis in a Woman with Hypogonadism

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The etiology of slipped capital femoral epiphysis (SCFE) remains uncertain. The frequent findings of growth abnormalities in affected patients lead to the hypothesis that various endocrine disorders cause this condition. We encountered a 25-year-old woman with chronic slipping of the femoral epiphysis, and the endocrine abnormality of hypogonadism was identified after further investigation. The radiographs revealed SCFE type I (less than one-third displacement of the epiphysis) of the left hip. Although a chromosomal study showed a karyotype of 46XX without chromosomal abnormality, gynecologic sonography showed the absence of a uterus and an ovarian body. No abnormal tumor was detected in the sella turcica, but brain MRI indicated that the pituitary fossa was relatively small for the patient's age. Abnormal serum levels of sexual hormones were measured. Surgical stabilization with percutaneous pinning of the left hip was performed and asymptomatically full weight-bearing gait with full motion of the left hip was found when the patients was examined in the outpatient department a year after the surgery.

Key words: slipped capital femoral epiphysis, hypogonadism

INTRODUCTION

Slipped capital femoral epiphysis (SCFE) is unusual in the adolescent hip. Although endocrine[1-5], traumatic, mechanical[6], and toxic[7,8] causes are all possible, the definitive etiology of this condition is still unknown. SCFE often occurs during the adolescent growth spurt and may be associated with endocrinopathies. We present a young adult woman with SCFE and hypogonadism and review her clinical course.

CASE REPORT

A woman aged 25 years four months presented with a one-year history of chronic, insidious-onset pain in her left hip and poor tolerance of weight bearing with limping for three weeks. The patient denied history of any major trauma, and no cause of her problem was immediately obvious.

The patient had been born normally after a full-term pregnancy. Her growth and development were normal until adolescence. She was 160 cm tall and weighed 54 kg, which was not unusual in her family. She had graduated from high school with average grades and normal social activities. She was heterosexual and had dyspareunia, with primary amenorrhea.

On physical examination, the patient's vital signs were within normal ranges. Her thyroid was barely palpable and no nodules were found. She lacked breast development and had scanty axillary and terminal pubic hair, though her external genitalia and vagina were normal. Findings on neurologic examination were within normal limits. She had equal leg lengths but an antalgic gait on ambulation. The range of motion in her left hip was limited, with 80° flexion and 15° extension, 20° internal rotation and 80° external rotation in flexion, and 30° abduction and 40° adduction.

Findings on anteroposterior and lateral radiographs included a subtle, posterior step-off of the epiphysis on the metaphysis at the physeal level. The Shenton line remained intact but the superior border of the epiphysis was on the Klein line on the left side. The impression was that of SCFE type I (less than one-third displacement) (Fig. 1). Wrist radiography demonstrated a delayed bone age approximating 16 years. Brain MRI showed a relatively small pituitary fossa for the patient's age, and a hyperintense ectopic spot in the posterior pituitary was noted in the medial eminence of the hypothalamus. No abnormal tumor was found in the sella turcica or other visual fields. Gynecologic sonography showed an absent uterus and no obvious ovarian body on

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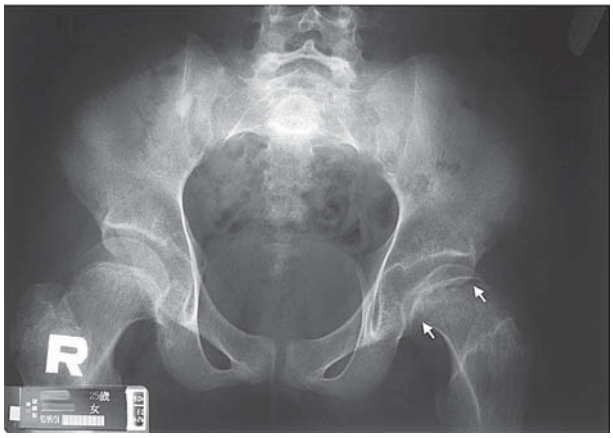

Fig. 1 Preoperative anteroposterior radiographies demonstrate subtle posterior step-off of the epiphysis on the metaphysis at the physeal level. The superior border of the epiphysis lies on Klein's line of the left hip.

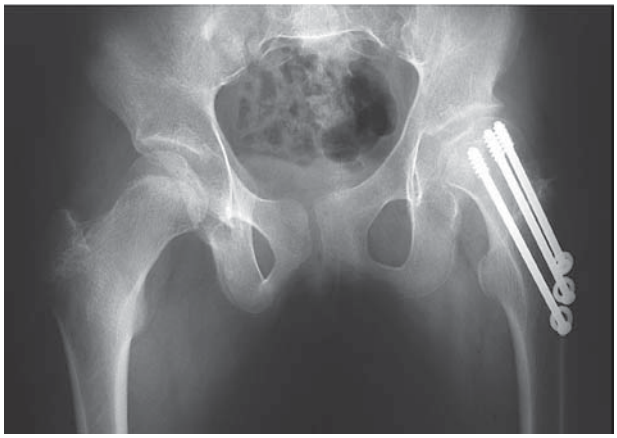

Fig. 2 Postoperative radiographies demonstrate the pinning position of Knowles pins in the slipped capital femoral epiphysis of the left hip.

either side.

Laboratory evaluation gave the following data: thyroid-stimulating hormone (TSH), 2.69 IU/ml (normal range 0.3-5 IU/ml); free T4, 0.78 ng/dl (normal range 0.8-2 ng/dl); growth hormone, 0.1 ng/ml; adrenocorticotropic hormone, 6.6 pg/ml (normal range 9-52 pg/ml); prolactin, 17 ng/ml (normal range 2.8-29.2 ng/ml); follicle-stimulating hormone (FSH), 0.79 mIU/ml; luteinizing hormone, 0.01 mIU/ml; testosterone, < 10 ng/dl (normal range 14-76 ng/dl); and estradiol, < 10 pg/ml. Cortisol levels were 0.85 μg/dl at 8:00 A.M. and 0.21 μg/dl at 4:00 P.M.

The karyotype was 46XX, as expected in a woman without chromosomal abnormality.

The initial impression was one of hypogonadism. Because of the patient's worsening symptoms and because of a desire to prevent further slippage, surgical stabilization with percutaneous pinning of the slipped epiphysis was performed (Fig. 2). The patient was referred to an endocrinologist and received a series of hormone-stimulation tests and subsequent treatment.

No complications occurred during hospitalization, and, by the seventh postoperative day, the patient was able to tolerate ambulation well, with continuous and partial weight bearing. Six months after surgery she recovered nearly the full range of motion in the left hip, without disability. At one year after surgery, the patient was examined in the outpatient department and was asymptomatic and capable of a full weight-bearing gait.

DISCUSSION

The etiology of SCEF is still unclear, and the role of endocrine abnormalities in the occurrence of SCEF is still controversial. Some reports indicate that a deficit of sex hormones relative to growth hormones can result in widening of the growth plate and subsequent reduction of the shearing force necessary to displace the epiphysis[9-11]. Androgens indeed increase the strength of the physeal plate, and low levels of androgens may delay puberty and weaken the physeal plate. Low androgen levels may therefore be a possible etiologic factor for SCFE[12]. Loder et al.[5] examined 85 patients with endocrine disorders and SCFE and found that 40% had hypothyroidism, 25% had growth-hormone deficiency, and 35% had other conditions, such as panhypopituitarism, craniopharyngioma, hyperparathyroidism, etc.

As a mechanical factor, obesity is also a predisposing factor for SCFE[11-13]. It increases the shear stress placed across the physeal plate. If this stress is combined with a weak and immature physeal plate due to architectural irregularities resulting from endocrine abnormalities, slippage of the epiphysis may result.

In both sexes, hypogonadism accelerates the loss of bone and the development of osteoporosis. Sex steroids also influence circulating levels of growth hormone and insulin-like growth factor-1, and the interaction among these hormones is likely to be important in the acquisition and maintenance of normal bone mass[16]. Androgens directly bind to androgen receptors or form aromatic compounds with estrogens and subsequently interact with estrogen receptors. Both pathways are important for skeletal health. The former is especially important in early skeletal development and in the determination of dimorphic sexual traits[17]. Bone remodeling, which is primarily

stimulated by estrogen, is important in maintaining healthy bone throughout life. Results of studies of bone modeling and remodeling in both rats and humans support the complex interaction of androgens and estrogens in the development and maintenance of healthy bone[18-21].

Some studies and case reports have focused on the occurrence of more than one case of SCFE in a particular family. Genetic and environmental factors were involved in the etiologies[22-24]. Environmental triggers associated with obesity or subtle hormonal imbalances were emphasized, but the relative importance of their roles remains unknown[24]. Previous studies showed a high percentage of bilaterality in patients with endocrine-associated (Loder et al., 61%, Wells et al., 100%) or nonendocrine-associated (Loder et al., 41 %) SCFE[25].

The progression of slippage results in complications, such as osteonecrosis and osteoarthritis, and leads to a poor outcome regarding the joint[26]. Delay in diagnosing SCFE has important implications in terms of the severity of slippage and the long-term outcome for the hip. In fact, a delay in diagnosis is essentially related to the severity of slippage[27].

In conclusion, careful clinical and radiographic examination for SCFE is required for all patients with endocrinopathy and hip problems with thigh or knee pain. Clinicians should be aware of the potential for undiagnosed endocrinopathies in patients with SCFE. Hypothyroidism should be screened first in all such patients as primary hypothyroidism may cause retardation of osseous development and delay in epiphyseal plate closure. It is important to distinguish primary from secondary hypothyroidism. The most helpful study is the determination of TSH level, because elevated TSH is the most sensitive indicator of primary hypothyroidism. Pituitary deficiency should be considered in those who have a relatively short stature for their age. Hypogonadism should be considered in those who lack appropriate sexual maturation for their age. Other associated orthopedic symptoms may be the poor development of skeletal muscles and also delayed epiphyseal closure resulting in long arms and legs. The causes of hypogonadism should be differentiated from hypogonadotropic hypogonadism, hypergonadotropic hypogonadism and genetically caused hypogonadotropic hypogonadism. The initial laboratory study should measure FSH, LH, prolactin, testosterone or estradiol levels and should include thyroid function tests. Patients with unilateral SCFE should receive close follow-up to detect changes in the asymptomatic or unaffected hip to prevent delays in diagnosis.

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