

# Slipped capital femoral epiphysis in a boy with hypogonadism

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*Sri Lanka Journal of Diabetes Endocrinology and Metabolism* 2011; 1: 48-50

## Abstract

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 14-year old boy referred by an orthopedic surgeon with the clinical presentation of lack of age appropriate sexual maturation. He had bilateral slipped capital femoral epiphysis and underwent surgical correction. Further examination and hormonal assessment revealed hypogonadotrophic hypogonadism which persisted over the next six months.

## Introduction

Slipped capital femoral epiphysis (SCFE) is unusual in the adolescent hip. Although endocrine, traumatic, mechanical, and toxic 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 boy with hypogonadism and SCFE. The possible mechanisms, associations and causes of SCFE are reviewed.

## Case report

A boy aged 14 years was referred from an orthopedic unit because of lack of secondary sexual characteristics. He was presented to the orthopedic unit with a one-year history of chronic, insidious-onset pain in his hip, poor tolerance of weight bearing and limping of three weeks. The patient denied a history of any major trauma. He was investigated and found to have bilateral SCFE for which he underwent surgical correction.

The patient had a normal birth at term. His growth and development were normal and since 11 years old he rapidly gained weight. There was no history of delayed dental maturation, anosmia, hyposmia or a family history of delayed puberty. His parents noticed he had lack of secondary sexual characteristics compared to his peers. He was studying in grade nine with average academic grades and normal social activity.

On examination, his vital signs were normal. He was 155 cm tall and 61.5 kg weight with a body mass index (BMI) of 25.6 kgm<sup>-2</sup>. His arm span was 157 cm and lower body segment (floor to pubis) was 1.5 cm longer than upper body segment (pubis to crown). He had no midline defects. His thyroid was not palpable. He lacked axillary and pubic hair, and the external genitalia showed a

micropenis with a phallus length of 2 cm and testicular volume of 3 ml and 4 ml in right and left testes respectively: consistent with a Tanner's pubertal stage of 1.

Neurologic examination including visual field proved normal.

Laboratory evaluation revealed the following data (normal values in parenthesis): Thyroid stimulating hormone (TSH) 1.55 IU/ml (0.4-4.0 IU/ml), free T4 1.16 ng/dl (0.8-2.0 ng/dl), prolactin 8.3 ng/ml (2.8-29.2 ng/ml), follicle-stimulating hormone (FSH) 0.473 mIU/ml (4.0-9.0 mIU/ml), luteinizing hormone (LH) 0.10 mIU/ml (1.6-9.0 mIU/ml), total testosterone 0.20 ng/ml (1.95-11.05 ng/ml), 9:00 a. am. fasting plasma cortisol 19.7 microgram/dl (5-25 microgram /dl), and Insulin like growth factor 1 (IGF1) 338 ng/ml which was appropriate for his age and sex.

Anteroposterior and lateral radiographs revealed bilateral SCFE (Figure).



Figure

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The bone age was 13 years (with the standard deviation of 11 months) at the chronological age 14 years and 5 months.

He had a normal MRI (magnetic resonance imaging) brain.

The diagnosis of hypogonadotropic hypogonadism was made.

At six months the levels of FSH, LH, and plasma testosterone were not much changed and were still in the prepubertal level. At this stage it may be contended that this patient had either an isolated hypogonadotropic hypogonadism or a delayed onset of puberty. But since the hormonal parameters and the testicular volume had not changed in six months, the diagnosis of hypogonadotropic hypogonadism appeared more likely.

Due to the stress caused by the lack of sexual characteristics of patient compared to his peers he was started on testosterone enanthate 50mg deep intramuscular injection monthly for three consecutive months. He was observed for pubertal signs over a further three months (six months in all) which remained unchanged. The LH, FSH and plasma testosterone levels repeated at six months were also unchanged.

## **Discussion**

SCFE occurs when shearing forces applied to the femoral head exceed the strength of the capital femoral physis (1, 2). The factors that weaken the physal plate are not fully clarified, but are thought to include: adolescence growth (3), trauma (4), obesity (5), inflammatory changes, genetic predisposition (6-9), endocrine and metabolic disorders such as hypothyroidism and growth hormone deficiency (10-14), growth hormone therapy (15, 16) and sex hormone deficit (10-14). In many instances a combination of the features may operate to cause SCFE.

In our case the diagnosis could be either delayed onset of puberty or secondary hypogonadism. The distinction between delayed puberty and secondary hypogonadism can only be made over time, by observing whether LH, FSH, plasma testosterone level and clinical features are consistent with the onset of puberty. Since our patient had reached the age of fifteen and he had not demonstrated any pubertal signs and his LH, FSH, plasma testosterone levels had remained in the prepubertal range, it was concluded that he was most likely to be a patient with hypogonadotropic hypogonadism.

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. Androgens indeed increase the strength of the physal plate, and low levels of androgens may delay puberty and weaken the physal plate. Low androgen levels may therefore be a possible etiologic factor for SCFE.

As a mechanical factor, obesity is also a predisposing factor for SCFE. It increases the shear stress placed across the physal plate. If this stress is combined with a weak and immature physal plate due to architectural irregularities resulting from endocrine abnormalities such as hypogonadism, 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. 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.

Bone remodeling, which is primarily stimulated by estrogen, is important in maintaining healthy bone throughout life. Some studies found the occurrence of more than one case of SCFE in a particular family.

In our case the contributory factors appeared to be hypogonadism and obesity. These lead to poor development of skeletal muscle, delayed epiphyseal closure and the mechanical factor of obesity.

The complications of SCFE includes osteonecrosis and osteoarthritis, which leads to a poor joint functional outcome.

## **Conclusion**

Careful clinical examination and hormonal assessment is required for all patients with SCFE to exclude an associated endocrinopathy. Hypothyroidism should be screened first in all such patients as primary hypothyroidism, the commonest endocrine cause may cause retardation of osseous development and delay in epiphyseal plate closure. Pituitary deficiency should be considered in those who have a relatively short stature for their age. Hypogonadism is a specially relevant aetiological factor when sexual development is absent.

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