The etiology of slipped capital femoral epiphysis (SCFE) is unknown, though hormonal as well as mechanical factors have been implicated. We report a case of gigantism who presented with SCFE. This case provides an insight into the genesis of SCFE, which in this case was related to growth hormone excess and sex hormone deficiency.

**INTRODUCTION**

Slipped capital femoral epiphysis (SCFE) is a disorder characterized by displacement of the capital femoral epiphysis from the metaphysis. Puberty-related biomechanical factors and endocrine alterations in the growth plate have been postulated to explain this disorder.1 We report a patient with gigantism who developed SCFE; this gives support to the hypothesis that SCFE occurs when there is a combination of growth hormone excess and sex hormone deficiency.

**CASE REPORT**

A 19-year-old boy presented with tall stature. Two years ago, he had developed a discomfort in the medial aspect of the right thigh, associated with a limp that had developed insidiously. A year ago, he was found to have a slipped epiphysis of the right femur, for which he had undergone fixation. He noticed diminution in vision during the last one year, involving both eyes, but the right more than left. His libido had decreased, and he had noticed regression of axillary and pubic hair. Relatives had noticed that he had been growing very fast since the last 3 years. There was no history of headache, vomiting, memory disturbance, polyuria or seizures. On examination, his pulse was 80/min, blood pressure 100/70 mm Hg. On standing, the blood pressure dropped to 80/60 mm Hg. There was a soft diffuse goiter. Hands and feet were enlarged. Axillary hairs were absent and pubic hairs were sparse. Testes were soft and 6 ml in size bilaterally. Height was 191 cm, and the arm span was 210 cm. Upper segment: lower segment ratio was 83/108. Nervous system examination revealed that visual acuity in the left eye was 6/18 and in the right eye only finger counting at 3 feet was possible. There was a left temporal field defect on confrontation perimetry. Examination of the fundus showed bilateral optic atrophy. Systemic examination was otherwise unremarkable.

**Investigations**

- Hemogram, electrolytes and renal functions were normal.
- Basal growth hormone level was 276 µg/L and the value was 247 µg/L two hours after glucose load (normal < 5).
- Other hormonal profiles:
  - T4 - 48 nmol/L (normal 64-154),
  - T3 - 1 nmol/L (normal 1.1-2.9),
  - TSH - 3.8 mU/L (normal 0.4-5),
  - LH < 1 IU/L (normal 1.3-13),
  - FSH - 1 IU/L (normal 0.9-15),
  - Testosterone : 6 nmol/L (normal 10-35),
  - 8 am cortisol 100 nmol/L (normal - 140/690), and
  - Prolactin 440 ug/L (normal 2-15).

A previous X-ray of the hip showed slipped capital femoral epiphysis (Fig. 1). MRI pituitary showed a large pituitary adenoma 19x14x12 mm in size extending suprasellarly, compressing the chiasma. The patient was started on replacement therapy with prednisolone (7.5 mg/d) and L thyroxine (0.15 mg/d). The patient was also started on bromocriptine (7.5 mg/d) and referred to the neurosurgeon for pituitary surgery.

**DISCUSSION**

Slipped capital femoral epiphysis (SCFE) is a rare disorder characterised by displacement of the capital femoral epiphysis over the metaphysis; classic findings are pain in the groin, limping gait and a limitation of medial rotation of the hip in extension. Treatment of chronic SCFE involves surgical stabilization of the displacement. The etiopathogenesis of slipped capital femoral epiphysis (SCFE) is still unknown.1 It is generally agreed that this is a disorder of puberty, where an increasing biomechanical load due to accelerated growth is coupled with a relative weakening of the growth plate, leading to the displacement of the epiphysis from the metaphysis. The reason for the weakened growth plate has been attributed to alterations in the hormonal balance of thyroid hormone, growth hormone, testosterone, and estrogen.2 Consequently, a variety of endocrinopathies have been associated with...
SCFE: hypothyroidism, obesity, hypogonadism and hypopituitarism.

Currently, SCFE is thought to be mediated by a combination of biochemical and biomechanical factors at puberty. In addition to circulating hormones, local concentrations of hormones (especially IGF-1) are known to have effects on the growth plate.

An interesting theory states that SCFE is due to an imbalance between the excess effects of growth hormone and decreased effects of sex hormones. This theory, called “Harris’s Hypothesis” has never been proven and is a subject of much controversy. Experimentally, growth hormone given to castrated rats has been shown to result in decreased shear strength to which the growth plate is exposed; in addition, hypertrophic chondrocytes in the growth plate are associated with deficient matrix and this renders the growth plate fragile.

Sex hormone administration to normal rats led to increased shear strength, rendering the epiphyseal plate less likely to separate. Clinical support for this hypothesis has come from the well-recognized association between growth hormone therapy and SCFE, and only a single earlier report of gigantism - associated with SCFE. The occurrence of SCFE in our patient with gigantism, who had both hypogonadism and growth hormone excess, supports the hypothesis that SCFE is related to an increased ratio of serum growth hormone to sex hormone action.

References