

Slipped capital femoral epiphysis in 6 of 8 first-degree relatives

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We treated 3 siblings for slipped capital femoral epiphysis (SCFE). The first, a girl, was admitted when she was 13 years old who had had left hip and knee pain for 3 months. Radiography showed a SCFE with a slip angle of 60° on the Lauenstein view. The girl was overweight, 62.5 kg, while her height was 148 cm. Menarche had not taken place yet, the size of the mammae was Tanner I. She was treated with a subtrochanteric osteotomy (Southwick 1967).

5 years later, a boy aged 14 years from the same family was examined because of pain in the left hip. The radiograph showed a SCFE on the left, with 40° slip angle. The boy was treated with open reduction and screw fixation. His body weight was 66 kg and his height was 154 cm. The size of the testis was 10 cm^3 .

The third child, also aged 14 years, was admitted with a 1 month history of left hip pain irradiating into the knee. The slip of the epiphysis was mild (10°) and in situ fixation was performed with pins. His body weight was 67 kg and height 156 cm. The size of the testis was 10 cm^3 .

The whole family was now screened. The siblings were examined physically and radiographs (anteroposterior and Lauenstein view) were taken. They were asked about previous hip or knee complaints. We were able to examine the parents and their 6 children. The uncles and aunts were interviewed. It was not possible to obtain useful data about the grandparents (Figure 1).

In addition to the 3 patients, 2 other siblings, a 21-year-old man (weight 85 kg, height 168 cm) and a 24-year-old woman (weight 92 kg, height 160 cm), were affected. Radiographs showed that a slip had occurred earlier; the contour of the femoral head was typically deformed. None of them had had any hip complaints in their childhood.

Both hips of the man were affected (right 25° , left 20°), while the female had a right SCFE (15°). The

man had bilateral hip pain after activities; the woman had no complaints. The youngest brother was 9 years old (weight 31 kg, height 124 cm) and had normal hips.

The mother (56 years, weight 58 kg, height 155 cm) and 3 of her brothers had normal hips. The father, aged 66 years at the time of the analysis, had a latent SCFE. He had never had hip pain, but the radiographs showed bilateral SCFE (right: 15° , left 20°) with mild arthrosis. His body weight was 74 kg, while his height was 169 cm. The father had 3 sisters who had no hip complaints. Except for the father who had ischemic heart disease, all the siblings were healthy.

The level of thyroid hormones was normal in all cases. In the siblings treated surgically, the level of the sex hormones was lower than normal. In the girl, the level of FSH was 2.8 iu/L and the LH was 9.5 iu/L. In the second case, the level of testosterone was $0.22\text{ }\mu\text{g}/100\text{ mL}$, while the other boy had $0.18\text{ }\mu\text{g}/100\text{ mL}$. However, they had a normal pubertal development later. The other siblings had a normal sex hormone status. Unfortunately, we have no data about the level of somatotropin.

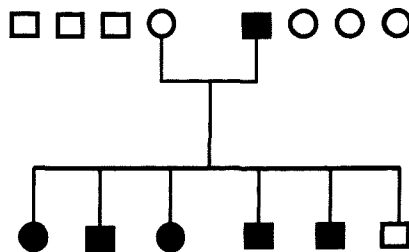


Figure 1. Pedigree of the affected family. □ male, ○ female, black affected, and white not affected

Discussion

Harris (1950) concluded that SCFE had an endocrine etiology. Heyerman and Weiner (1984) found that SCFE was associated with hypothyroidism. A multifactorial endocrine background was assumed by Al-Aswad et al. (1978) and Wilcox (1988). Bone et al. (1985) found an increased excretion of parathormone.

A familial occurrence of SFCE is rare, 3% according to Sørensen (1968). Familial SFCE was first reported by Kirmisson (1918) and later by others (Schreiber and Schmied 1967, Rennie 1982, Hägglund and Hansson 1986). Rennie (1982) reported that first degree relatives were affected in 14 cases out of 214; 23 second-degree relatives were also affected. According to Exner (1986), familial SCFE is caused by inherited tendencies to increased weight and pathological constitution. In the family we analyzed, except for the unaffected mother and the youngest brother, all were overweight. The 3 children treated surgically were hypogonadal.

Hägglund et al. (1986) analyzed 49 families and found manifest SCFE in 4 families, whereas the disease had occurred in a latent form in 13 families. Some authors have stated that SCFE is transmitted by an autosomal dominant gene with varying penetrance, but a multifactorial origin cannot be excluded (Rennie 1982, Crossan and Wynne-Davies 1986).

When the familial history is positive the risk of a new case is higher. According to Hägglund and Hansson (1986) the incidence could be 15% in such a family. We shall follow the youngest boy in this family.

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