

Slipped Capital Femoral Epiphysis in an Adult Patient with Panhypopituitarism

Shikha Gupta*, Kavita Vani**, Rahul Bansal*, Bindu Kulshreshtha*

Abstract

Slipped capital femoral epiphysis (SCFE) most commonly affects early adolescent age group while the epiphysis is still unfused. There are only anecdotal case reports of adults with SCFE. Hypothyroidism and hypogonadism, two endocrine conditions associated with delayed epiphyseal fusion are the most common causes of SCFE post-adolescence. We present here a case of twenty-three-year-old male who presented with pain hip due to SCFE. He was later diagnosed to have panhypopituitarism in association with partial empty sella.

Keywords: SCFE, adult, partial empty sella.

Introduction

Slipped capital femoral epiphysis is the most common hip disorder affecting early adolescents^{1,2}. It usually occurs in children going through a pubertal growth spurt, possibly because the immature proximal physis is unable to bear shear stress³. Most SCFE patients are younger than eighteen years because proximal femoral physis fuses at this age. However, it can also appear in adults with delayed skeletal maturation. This suggests that SCFE only has the opportunity to appear while physis is still open. At the cellular level, the physis in these cases shows loss of regular longitudinal cell column formation in cartilage and increased intercellular matrix⁴.

Slipped capital femoral epiphysis in adults is uncommon, with only fifty-five cases reported in literature⁵. Post-adolescence, endocrinopathy is the most common cause of SCFE. This includes hypothyroidism and hypogonadism—two endocrine conditions that are commonly associated with delayed fusion of the epiphysis⁶. Macia-Villa *et al* reviewed all previous reported cases of adults with SCFE in 2015 and observed that among fifty-five adults, nineteen (35%) had hormonal causes for SCFE⁵.

Here, we describe a twenty-three-year-old male who presented with SCFE in the setting of partial empty sella with panhypopituitarism.

Case report

This twenty-three-year-old male consulted an

orthopaedician for pain in the lower back and left lower limb. He had developed lower back pain two years back while he was lifting some heavy object. He did not pay much attention to pain initially; however, the pain gradually became severe and later involved the left lower limb and he started limping. He consulted an orthopaedician who observed a shortened and externally rotated left leg. X-ray revealed left slipped capital femoral epiphysis. He was given calcium, vitamin D and analgesics and surgery was advised. Meanwhile he was referred to our Endocrine department for lack of secondary sexual characters. He reported that he had not gained height in the past years and had observed small genitalia and absence of pubertal changes unlike his peers. He felt lethargic and his appetite was poor. On examination patient was 156 cm tall (upper segment/lower segment ratio -0.72) with BMI of 18 kg/m². He was prepubertal (A1B1P1) with stretched penile length of 4.5 cm and testicular volume of 1 cc on both sides. There were no axillary or pubic hairs. He denied any history of anosmia, headache, head trauma or any chronic diseases. Family history was non-contributory.

Investigations revealed mild anaemia with normal liver and kidney function tests. Hormonal investigations revealed central hypothyroidism, hypogonadotropic hypogonadism and hypocortisolism suggestive of panhypopituitarism (Table I). Gn RH stimulation test was non-stimulable with peak LH post-stimulation of 0.33 m IU/ml. DEXA scan for bone density was done which showed osteoporosis (Z score of -3.8 at lumbar spine, -2.55 at left hip and -4.6 at left forearm).

*Professor and Head, Department of Endocrinology, **Department of Radiodiagnosis, PGIMER Dr Ram Manohar Lohia Hospital, Baba Kharak Singh Marg, New Delhi - 110 001.

Corresponding Author: Dr Bindu Kulshreshtha, Professor and Head, PGIMER Dr Ram Manohar Lohia Hospital, Baba Kharak Singh Marg, New Delhi - 110 001. Phone: 011-23404789, E-mail: drbindu25@yahoo.co.uk.

Table I: Details of hormonal profile in the patient.

Hormones (units)	Results	Range
FT3(pg/ml)	2.43	2 - 4.4
FT4 (ng/dl)	0.43	0.6 - 2.2
TSH (mIU/ml)	10.3	0 - 5.5
FSH (mIU/ml)	2.85	1.55 - 9.74
LH (mIU/ml)	< 0.216	1.8 - 7.8
Testosterone (nmol/l)	< 0.170	0.198 - 2.67
Prolactin (ng/ml)	30	3 - 18.6
PTH (pg/ml)	48.9	14 - 75
VitD3 (pg/ml)	35.8	30 - 56
Cortisol (nmol/l)	39.9	123 - 626

MRI of brain revealed partial empty sella syndrome with CSF herniation and compressed pituitary gland (Fig. 1). X-ray pelvis and MRI Pelvis was suggestive of slipped capital femoral epiphysis (SCFE) (Fig. 2 and 3). The bone age was delayed (fifteen-years compared to his chronological age of twenty-two years). He was started on steroids initially followed by throxine and injectable testosterone.

Discussion

Slipped capital femoral epiphysis (SCFE) is an uncommon disorder occurring during adolescence while the epiphysis is still open. Rarely, patients undergo slippage at atypical ages when physis are supposed to be already closed⁵. The aetiology of SCFE is unclear. Various factors have been postulated for its occurrence including obesity, mechanical,

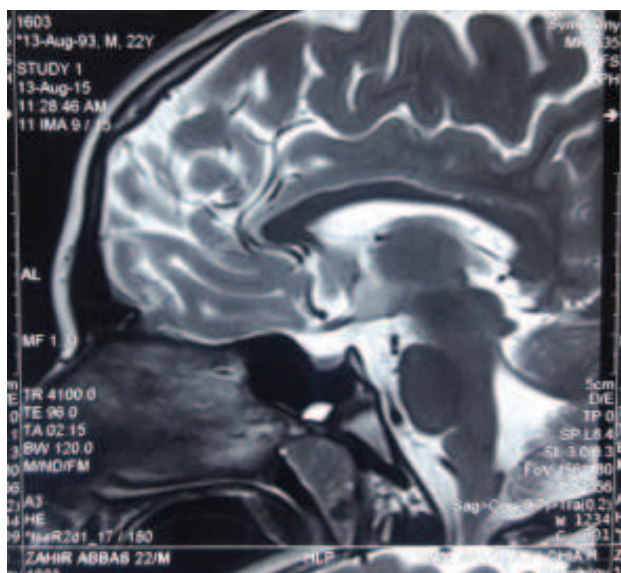


Fig. 1: MRI Brain suggestive of partial empty sella syndrome.



Fig. 2: X-ray Pelvis suggestive of left slipped capital femoral epiphysis.

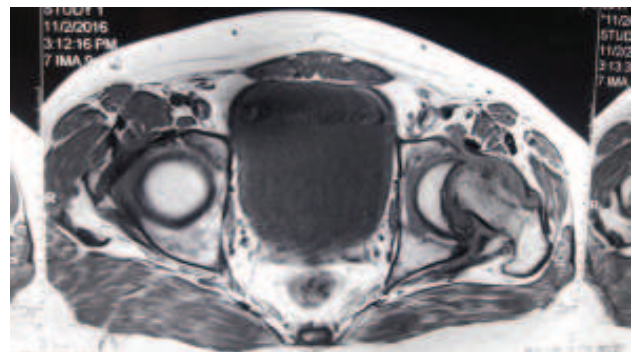


Fig. 3: MRI Pelvis suggestive of left slipped capital femoral epiphysis.

hormonal, genetic factors and some toxic drugs. Some familial cases have also been reported whereby hereditary factors like collagen type II or proteoglycan defects have been implicated⁷.

The hormonal milieu during adolescence, combined with a deficiency in the physal area of the growth plate has been postulated as a possible aetiology for SCFE. Hypothyroidism and hypogonadism, two endocrine conditions associated with delayed epiphyseal fusion are the most common causes of SCFE post-adolescence⁶. GH excess either due to pituitary tumour or GH treatment has also been associated with SCFE^{4,9,10}. Both thyroid and growth hormone are necessary for growth and maturation of the cartilage, with subsequent calcification and replacement by mineralisation. Vitamin D deficiency with secondary hyperparathyroidism in the setting of chronic renal failure is another rare cause of SCFE. Rare cases of SCFE due to steroids have also been reported^{11,12}.

Only anecdotal case reports of SCFE during adulthood have been reported. The proximal femoral epiphysiolysis in adults is commonly associated with endocrinal disturbances. Till 2016, fifty-five cases of SCFE in adults more than twenty years of age were reported. Of fifty-five, twenty-eight cases had endocrinological causes of SCFE like hypothyroidism in five, hypopituitarism in twenty-two and acromegalic gigantism in one patient. Other cases were mostly

idiopathic or traumatic⁵. Soleymanha *et al* reported a case of SCFE in an adult patient with craniopharyngioma aged twenty-eight years. He had a combination of secondary hypothyroidism, hypogonadism and hypocortisolism¹³.

Our case presented during adulthood and was diagnosed as SCFE with panhypopituitarism. The endocrine abnormalities diagnosed were hypogonadotropic hypogonadism (as evidenced by non stimuable GnRH stimulation test), central hypothyroidism and hypocortisolism. Our patient was initiated on hormonal therapy including thyroxine, low dose steroids and androgens.

In conclusion, this was a rare case of SCFE in an adult patient with hypopituitarism. Therefore endocrine disorders like hypothyroidism and hypogonadism must be excluded in patients presenting with SCFE.

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