Slipped capital femoral epiphysis in adults: case report and review of literature

C.C. Macía-Villa[1], I. Sanchez-Lite[2], J. Medina-Luezas[1]

[1]*Department of Rheumatology, Clinical University Hospital of Valladolid, Valladolid, Spain;*
[2]*Department of Radiology, Muscleskeletal section, Clinical University Hospital of Valladolid, Valladolid, Spain*

SUMMARY

Slipped capital femoral epiphysis (SCFE) mainly affects overweight prepubertal children. It is usually idiopathic, but endocrinological diseases are proposed as the main cause. SCFE occurs before the closing of the femoral physis, which generally occurs at 18 years in males and 16 years in females, therefore it is considered a children's disease. However, there have been several reports of adults with SCFE and some familial cases. We present a case of bilateral SCFE in a 47 years old female with possible relationship with inhaled corticosteroids, and for the first time we collect and analyze all published cases in adults.

Key words: *Slipped; epyphisis; femur; adult; corticosteroids; bone.*

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INTRODUCTION

Slipped capital femoral epiphysis (SCFE) is one of the most frequent hip disorders in children. SCFE is due to the displacement of the epiphysis (femoral head, that keeps its location in the acetabulum) relative to the metaphysis (femoral neck) and shaft at the the physis level (1, 2). Most SCFE patients are younger than 18 years old because proximal femoral physis fuses at the age of 18 in boys and 16 in girls. However, it can also appear in adults with a delayed skeletal maturation. Anything able to decrease the growth plate resistance or to maintain the epiphysis opened might result in SCFE at any age.

The physiologic age when SCFE occurs is less variable than the chronologic age (3), which is called *the narrow bone age window*. This suggests that SCFE only has the opportunity to appear while the physis is still opened, and this depends on the bone age (or bone health) more than on the chronological age. At cellular level, the physis in these cases shows loss of regular longitudinal cell column formation in the cartilage and increased intercellular matrix. The damage in SCFE occurs between hypertrophy zones and provisional ossification zones, although it can extend towards the germinal zone or into the metaphysis (4).

Clinical expression of SCFE is usually groin and limp pain, with decreased rotations (mainly external) on physical examination (5). However there are several reports of asymptomatic cases, as it was demonstrated in a study in 2072 healthy Norwegian young people (18-20 years old) that found 6.6% cases of silent radiological SCFE (6). The reported incidence is variable depending on the countries, oscillating between 0.2 and 24.5:100,000 children 8 to 15 years old (1, 3, 5), but this number may underestimate the real incidence due to silent forms (7). SCFE is more frequent during the adolescent growth spur that happens to an average age of 13.5 years in boys and 12 years in girls. It is more prevalent in non-Caucasian males, and some series describe a seasonal variation in the incidence, being higher in spring and summer.

SCFE is frequently bilateral, reaching 50% of cases in some series. In unilateral cases, the age of presentation is usually younger, left hip is affected twice as often than the right, and an increased risk of contralateral damage appears in the next 18 months (3, 8). It is important to always check the contralateral hip as some series describe 40% prevalence of asymptomatic slips in this

Corresponding author
Cristina Clara Macía Villa
Department of Rheumatology
Clinical University Hospital of Valladolid
Avenida Ramón y Cajal 3
47005 Valladolid, Spain
E-mail: ccmacia@gmail.com

location (6). The only definite treatment is surgery, and most symptomatic cases need a total hip replacement.

The etiology of SCFE is unknown, but there are several disorders that have been related with its presentation and are summarized in Table I. Overweight with a body mass index higher than a 95% is reported in 63-80% of children with SCFE, and it has demonstrated to change the normal hip biomechanics (1, 5, 9).

In post adolescence, endocrinopathy is the most common cause of SCFE, mainly hypothyroidism and hypogonadism that produce delayed fusion of the epiphysis (10).

Several hypophysis dysfunctions have been associated with SCFE: Simmonds-Seehan disease and other causes of hypopituitarism (craniopharyngiomas, optic gliomas or chronic subdural hematomas), pinealomas and growth hormone (GH) pathologies. GH physiologically decreases the strength of the physis affecting the cells of the epiphyseal ossification center, explaining the cases of SCFE during GH treatment or pathologies with increased levels like acromegalic gigantism. Only elevated GH has been related with SCFE, there are any reported cases with isolated GH deficiency. In panhypopituitarism a GH decrease can be seen, but the effect of the other pituitary hormones seems to prevail (1, 2, 4).

In chronic renal failure the probability of epiphyseal separation is increased due to metabolic acidosis, secondary hyperparathyroidism and deficient vitamin D activation, that decrease bone mass and mineralization with changes in the cartilaginous plate of proximal femur. Calcitriol also has a direct action on epiphyseal chondrocytes, and some authors suggest that the seasonal variation of SCFE can be related to a decrease in vitamin D after several months without sunlight (5, 11). There is probably an hereditary factor involved in this disease: a decreased expression of collagen type II and proteoglycans has been reported in some patients, and SCFE has been described in Frolich's, Marfan's, Klinefelter's and Down's syndromes (12). Although the relationship between SCFE and glucocorticoids (endogenous or exogenous) has not been demonstrated yet (5), their skeletal effects on bone are widely known being the most common iatrogenic cause of secondary osteoporosis.

Regardless of the previously described associations with SCFE, the implication of these diseases remains controversial and diverse factors may play a role in each patient. The typical case normally appears in the absence of consistent biomechanical, biochemical or metabolic abnormalities (12).

We present an atypical case of SCFE in a female adult, and we collect and review all the adult SCFE cases published until the date.

Table I - Principal causes of slipped femoral capital epiphysis.

Mechanical factors	- Overweight - Traumas - Deeper acetabula - Coxa vara - Femoral and acetabular retroversion
Hormonal factors	- Hypophysis dysfunction - Hypothyroidism - Hypogonadism - Chronic renal failure - Calcitriol deficit - Other endocrine dysfunctions: hyperparathyroidism, adiposogenital syndrome, malnutrition
Genetic factors	- Decreased expression of collagen type II and proteoglycans - Frolich's, Marfan's, Klinefelter's and Down's syndromes
Toxics and drugs	- Radiation and chemotherapy, especially actinomycin D - Glucocorticoids
Idiopathic	

CASE REPORT

We present the case of a 47 years old Caucasian female with mild dyslipidemia (dietetic treatment), intrinsic asthma treated with inhaled corticosteroids for the last 6 years (fluticasone 500 mcgr bid: equivalent to 2000 mcgr/day of beclomethasone) and smoker of 20 cigarettes/day. Her menopause began two menopause two years ago and has not received hormone supplements. Previously asymptomatic, in August 2014 she developed without obvious cause a disabling left groin pain that became bilateral in a few days. Hip x-rays (Figure 1A) showed subtle lineal high-density images in both femoral necks, mainly on the left side. She was referred to Traumatology and Rheumatology departments. At clinical examination she was able to walk with bilateral groin pain, no leg length differences were noted, the left hip flexion was limited to 45º, and internal and external bilateral rotations to 15º. The study was amplified in the next months with more imaging tests (Figures 1B, 2, 3 and 4) and diagnosis of bilateral SCFE was made. A whole body Tc99 scintigraphy showed pathological findings only in hips, bone densitometry values were normal at lumbar spine, neck and total hip, and an exhaustive laboratory study including liver and renal function test, blood cell count, acute phase reactants, blood and urine calcium and phosphorus, alkaline phosphatase, bone turnover markers, celiac disease antibodies and hormones (parathormone, sex hormones, prolactine, cortisol, ACTH and somatomedine IGF-I) was normal except for severe vitamin D deficiency (25 hydroxycholecalciferol levels 7.74 ng/mL) and menopausal status. She had failed to symptomatic treatment prescribed by her General Practitioner. The

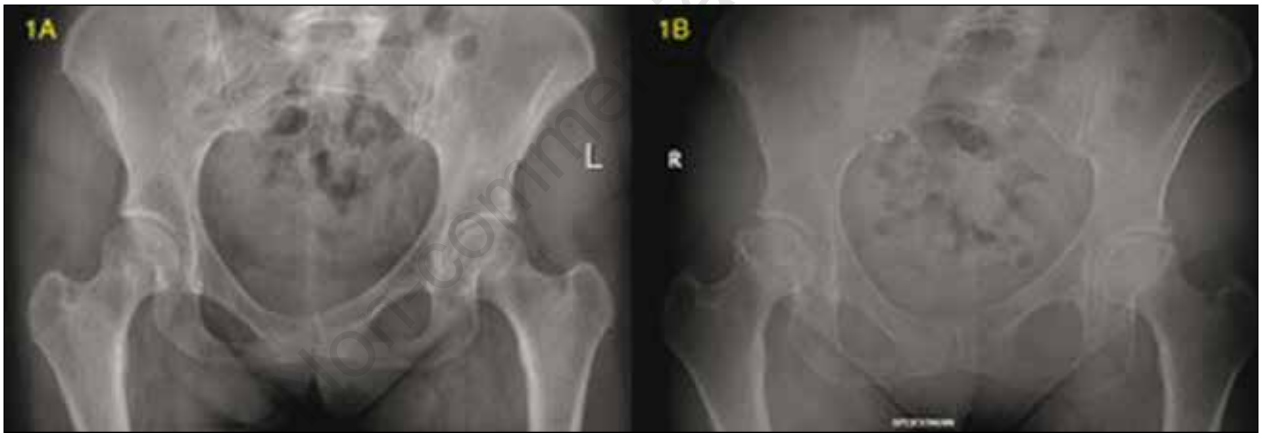

Figure 1 - Anteroposterior pelvis plain radiographs. A) At the moment of beginning of pain symptoms, minimal high density lineal images in both femoral necks, mainly on the left one. B) 6 months after Figure 1A, bilateral epiphyseal fracture with minimal displacement.

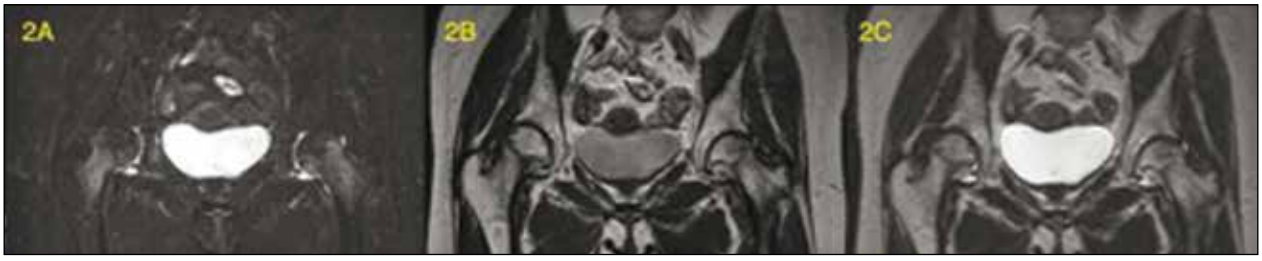

Figure 2 - Magnetic resonance images (MRI) showing results 3-month after Figure 1A. A) Coronal short tau inversion recovery MR image: extensive bone marrow edema in both femoral heads; B and C) Coronal T1-weighted MR image and coronal T2-weighted MR image sequences, respectively: fracture lines in femoral epiphyses.

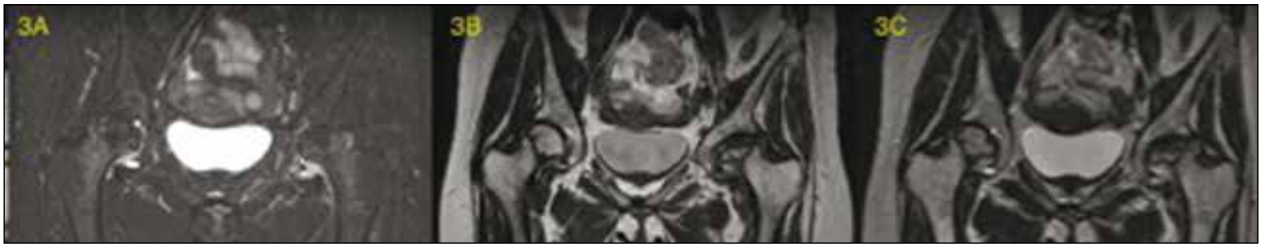

Figure 3 - Magnetic resonance images (MRI) showing results 7-month after Figure 1A. Fracture lines in both femoral heads with mild displacement, decreased bone marrow edema compared with Figure 2, and collapse and secondary osteonecrosis in left femoral head. Panels A, B and C are respectively coronal short tau inversion recovery MR image, coronal T1-weighted MR image and coronal T2-weighted MR image sequences.

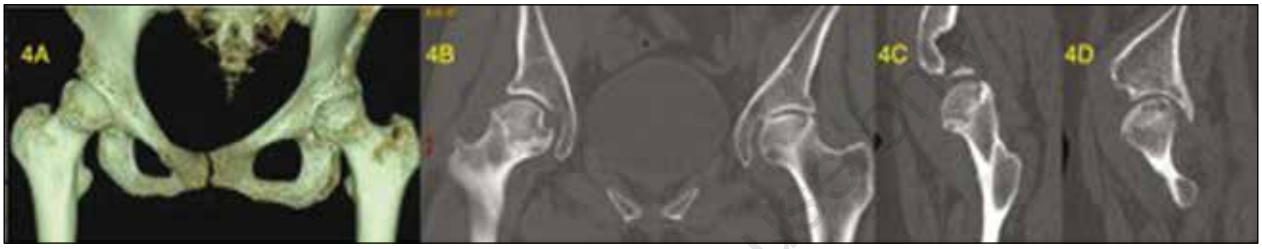

Figure 4 - Computed tomographic images 7-month after Figure 1A. A) 3D image; B) coronal view; C) sagittal right hip view; and D sagittal left hip view. Fracture lines in both femoral epiphyses with mild displacement and collapse signs in left femoral head.

pain mildly improved with walking restriction with wheelchair and crutches and the patient was discharged at home. Vitamin D supplements were prescribed with normalization of serum levels 3 months later. At the moment of publication, the patient has recently undergone surgery with a left total hip replacement.

DISCUSSION

Some patients undergo slippage at atypical ages when physis are supposed to be already closed. This situation, linked to the fact that many cases are asymptomatic and sometimes an unexpected radiological finding, makes the real prevalence of adult SCFE probably underestimated. Moreover, for some authors most cases of premature hip osteoarthritis are a sequel of known (and unknown) hip disorders as hip dysplasia, Legg-Calvé-Perthes, and also SCFE (13). Sometimes, the etiology of this atypical SCFE cases is found, but there are several idiopathic cases that leave us an unanswered question: are adult SCFE cases a childhood disease with a delayed diagnosis, or are they acute slippings in an adult non-closed physis?

All previously reported series of SCFE set the upper age limit at 18 years assuming that this is the normal limit of physiological physis closure. Only Hu MH and colleagues (8) searched for SCFE cases in adults older than 20 years between 1960 and 2009. We decided to do a non previously published review of SCFE cases in people older than 18 years: adult cases (14-44) searched in PubMed up to June 30, 2015 with keywords *slipped, femoral, epiphysis, head* (mainly searching for large series with possible adult cases) and same keywords adding *adult* (searching exclusively adult cases). References of retrieved articles were also reviewed. Inconclusive or incompletely described cases were rejected. Results are shown in Table II. Since 1940 (first report founded) we have found 60 cases, and 136 asymptomatic radiological SCFE in the Norwegian healthy cohort 18-20 years old (6). Excluding this cohort, there are 23 idiophatic cases, 17 secondary to hypophyseal abnormalities, 7 secondary to unspecified endocrine disorders, 5 re-

Table II - Slipped capital femoral epiphysis cases published until 2015 in people equal or older than 19 years old at time of diagnosis. In familial studies age is usually not provided, all cases of the table are reported as *mother*, *father* or similar. Inconclusive or incompletely described cases have been discarded.

Reference	Year	Type of report	Number of cases	Age of diagnosis (years)	Cause
Thrap-Meyer (14)	1940	Familial	1	Unknown, father	Unknown
Moore (15)	1945	Case	1	21	Traumatic
Irwin (16)	1946	Familial	1	Unknown, *father*	Unknown
Lofgren (17)	1953	Case	2	20 and 35	Hypophyseal abnormalities
Farrow (18)	1953	Case	1	26	Simmond´s disease
Burrows (19)	1957	Serie	1	41	Pituitary hypogonadism
Epps (20)	1963	Case	1	22	Hypothyroidism
Goldman (21)	1963	Case	1	27	Hypopituitarism and gigantism
Sarver (22)	1964	Case	3	20, 27 and 32	Functional hypopituitarism and giantism
Rennie (23)	1967	Familial	2	Unknown, *father and uncle*	Unknown
Zimmerman (24)	1967	Case	2	20 and 29	Hypopituitarism
Primiano (25)	1971	Case	1	19	Hypogonadism due to Klinefelter's syndrome
Kelsey (26)	1971	National serie	3	Two cases: 19 and 22 One case: 19	Unknown, both mentally and physically retarded Steroid therapy for bronchial asthma
Strunz (27)	1972	Case	1	51 yo	Panhypopituitarism
Heatley (28)	1976	Case	2	19 and 27 yo	Intracranial tumors causing hypogonadism
Al-Aswad (29)	1978	Case	1	35	Hypothyroidism
Goldman (30)	1978	Case	1	19	Renal osteodystrophy
Hennessy (31)	1982	Case	1	21	Hypothyroidism
Rennie (32)	1982	Familial	3	Unknown: *father, aunt and uncle*	Unknown
McAfee (12)	1983	Case	2	19 26	Radiation for sarcoma Panhypopituitarism
Hägglund (33)	1986	Familial	2	Unknown. 2 cases in *fathers*	Unknown
Cooperman (7)	1992	Post *mortem*	7	Unknown age of onset, only data of ages of death (26-68)	Unknown
Wells (34)	1993	Case	1	Unknown, older than 20	Craniopharyngioma with panhypopituitarism
Montskó (35)	1995	Familial	3	21, 24 and 66	Unknown
Feidy (36)	1997	Case	1	24	Acromegalic gigantism
Moreira (37)	1998	Familial	1	40 yo	Unknown
Diwan (38)	1998	Familial	1	Unknown, *mother*	Unknown
De Silva (39)	2000	Case	1	80 yo	Idiopathic
Noguchi (40)	2002	National serie	7	Unknown, older than 20	All with unspecified endocrine disorders (mentioned hypopituitarism and hypothyroidism)
Huang (41)	2007	Case	1	23	Craniopharyngioma with panhypopituitarism
Nourbakhsh (42)	2008	Case	1	24	Hypothyroidism
Brady (10)	2010	Case	1	22	Pituitary tumour with panhypopituitarism
Hu (8)	2011	Case	1	29	Craniopharyngioma with panhypopituitarism
Lehmann (43)	2012	Arthroplasty register	1	24	Unknown
Lehmann (6)	2013	National serie	136	Cohort of 2072 health Norweigans 18-20 Found x-rays compatible with asymptomatic SCFE in 6.6% (136 cases)	Unknown
Marquez (44)	2014	Case	1	28	Hypothyroidism

SCFE, slipped capital femoral epiphysis.

lated to hypothyroidism, 4 hypogonadism cases, and isolated case reports of trauma, renal osteodystrophy, post radiation, and asthma corticotherapy. This distribution matches with most frequent SCFE etiology in children: idiopathic and endocrine. Eight reports are family cases, mostly asymptomatic adult SCFE cases found in children relatives with symptomatic SCFE. Specifying the age of diagnosis in this category of studies is difficult, as frequently cases are reported in terms of family relationship (*mother, father, uncle*). Some authors postulate that there is probably a hereditary factor involved in SCFE, maybe in collagen type II and proteoglycans but this has not been clearly demonstrated, and there are several family reports without pathologic causes or background. There is also a *post mortem* study[7] with age of death between 26-68 years old that found seven unknown SCFE cases. Finally there are three national series of Norway (6), Connecticut and Southwestern United States (26) and Japan (40), and an arthroplasty register (43).

Our case is not the first case of adult SCFE with previous normal hip x-ray. De Silva and colleagues (39) reported an 80 year-old man with previous radiologically closed epyphisis who later developed an idiopathic SCFE (histology excluded malignancy, osteomalacia and avascularity). We propose that a normal x-ray in adults does not exclude microscopic physys pathology. Our case is the second report with a previous exposure to inhaled corticosteroids for asthma (26), but the time of exposure is not given. Oral glucocorticoids increase bone resorption, decrease bone formation and disrupt bone remodeling regulation resulting in a rapid loss of bone mineral density and bone quality even at doses as low as 2.5 mg of prednisolone per day or equivalent for more than 3 months (45). Studies focused on the bone effect of inhaled corticosteroids show disparate results. A Cochrane systematic review (46), a retrospective cohort (47) and another study (48) found no evidence of bone mineral density loss, bone turnover or vertebral fractures in patients receiving chronic inhaled corticosteroids. On the contrary, a meta-analysis (49) found a 12% increase in non-vertebral fractures and two other studies (50, 51) showed lower bone mineral density in these patients. A recent evaluation of publications in this topic by FRAX® group suggests an excess risk of fractures in those treated with high doses: 1600 mcgr/d of beclomethasone equivalent or greater (52). Overall there is no consensus, but data are consistent with a dose related decrease in bone mass and increased fracture risk in patients receiving long-term moderate to high dose–inhaled corticosteroids. In our case this is the most likely cause of SCFE we have found, although we cannot rule out the additive effect of accelerated bone loss related to recent menopause. The other pathological finding, vitamin D deficiency, can be explained by long rest at home and lack of exposure to sun after the beginning of SCFE symptoms.

CONCLUSIONS

SCFE is mainly a children's disease but it is also possible at older and atypical ages when physis are supposed to be closed. Although it is mandatory to rule out an underlying disease that can explain this lack of physiological consolidation, idiophatic cases prevail both in children and adults. This is the first review in literature of adults older than 18 years with SCFE, with 60 published cases since 1940 and 136 x-rays findings in a healthy cohort. In our case, we propose inhaled corticosteroids as the possible etiology with the exceptional finding of the absence of unclosed femoral physys in the initial hip x-ray interpreted as the presence of physis pathology at a microscopic level.

Conflict of interest: the authors declare no potential conflict of interest.

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