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Clinical case

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Slipped capital femoral epiphysis in an adult with an unidentified Kallmann syndrome. Case report and etiology literature review

Epifisiólisis femoral capital deslizada en un adulto con síndrome de Kallmann no identificado. Informe de caso y revisión de la literatura sobre etiología

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Abstract. A 23-year-old male consulted for a subacute-onset left hip pain, being evident on radiographs a slipped capital femoral epiphysis (SCFE) while the contralateral proximal femur physis was still slightly open. Urgent percutaneous *in-situ* fixation with two cannulated screws was performed. Further investigations due to the atypical features of the case revealed that the patient had anosmia, which allowed for Kallmann syndrome (KS) diagnosis, being responsible for the delayed skeletal maturity of the patient. Very few cases of a delayed-onset SCFE in association with KS have been described in the literature but must be considered in the differential diagnosis.

Keywords: slipped capital femoral epiphysis, Kallmann syndrome, physiology, diagnosis.

Resumen. Paciente masculino de 23 años que consultó por dolor de cadera izquierda de inicio subagudo, evidenciándose en las radiografías una epifisiólisis femoral capital deslizada (*SCFE*, por sus siglas en inglés), mientras que la fisis del fémur proximal contralateral aún estaba ligeramente abierta. Se realizó fijación percutánea urgente *in situ* con dos tornillos canulados. Investigaciones adicionales debido a las características atípicas del caso revelaron que el paciente presentaba anosmia, lo que permitió el diagnóstico de síndrome de Kallmann (SK), siendo responsable de la madurez esquelética retrasada en este paciente. En la literatura se han descrito muy pocos casos de SCFE de inicio tardío en asociación con síndrome de Kallmann, pero deben ser considerados en el diagnóstico diferencial.

Palabras clave: epifisiólisis femoral capital deslizada, síndrome de Kallmann, fisiología, diagnóstico.

Abbreviations:
HH = hypogonadotropic hypogonadism
KS = Kallmann syndrome
SCFE = slipped capital femoral epiphysis

Introduction

Slipped capital femoral epiphysis (SCFE) is a condition of the proximal femoral physis in which there is an anterosuperior slide of the metaphysis relative to the epiphysis, occurring predominantly at puberty. It is more common in males, in specific ethnicities,[1,2] and associated with endocrine disorders, being obesity the single most significant risk factor.[3] Some authors describe it as one of the most common hip disorders in adolescents.[1,2] However, this condition is not exclusive to adolescents, since, although rare, it can also occur in skeletally immature adults, receiving the denomination of delayed-onset SCFE.

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On the other hand, Kallmann syndrome (KS) is a developmental genetic disorder characterized by congenital hypogonadotropic hypogonadism (HH) and anosmia or hyposmia secondary to aplasia or hypoplasia of the olfactory bulbs.[4]

To our knowledge, only three cases of a delayed-onset SCFE in association with KS have been described in the literature.[5,6,7] A new case that combines these conditions is presented here, and also a pragmatic literature review on the etiology of adult SCFE and its association with KS is performed.

Case report

A 23-year-old male consulted for hip pain with an onset two weeks ago after exercising. On physical examination, he was able to walk, although, with a limp, his left lower leg appeared externally rotated, he had limited hip internal rotation, painful flexion and abduction, and a positive Drehmann sign.

Simple radiographs showed a double density at the metaphysis (steel sign), widening and lucency of the physis compared to the contralateral, and a displacement of the epiphysis falling below the Klein line. Besides, the contralateral physis was still not totally fused *(Figures 1 and 2)*. For a better injury characterization, a CT scan was completed, revealing displacement of the left epiphysis relative to the metaphysis with minimal rotation *(Figure 3)*.

The diagnosis of delayed-onset left SCFE was made, and the patient was admitted for urgent surgical treatment. Under spinal anesthesia and fluoroscopic guidance, the patient was intervened by an *in situ* fixation with two partial threaded 7.3 mm diameter cannulated screws. No remarkable intraoperative incidences occurred; however, while in the operating theatre, absence of pubic, axillary, and facial hair was noted, and a micropenis was also observed, with no testicles palpable in the scrotum. Contralateral hip was not prophylactically fixed due to patients' preference; thus, close monitoring was decided.

His postoperative course was adequate, being the radiographic control satisfactory *(Figures 4 and 5)*; hence, partial weight-bearing was started immediately. Due to the abnormal findings and atypical characteristics of the case, a consultation to the endocrinologist was executed. As a complementary extension study, a complete blood test was made, which exhibited a normal hormonal profile except for FSH, LH, and testosterone levels, that stood below normal

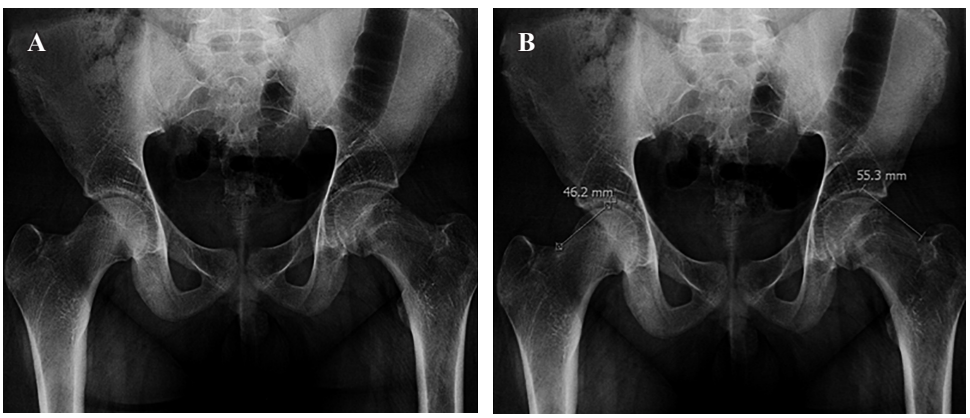

Figure 1:

Pelvis anteroposterior radiograph. **A)** Steel sign, widening, and lucency of the left proximal femoral epiphysis, which has no contact with the Klein. **B)** Representation of the Klein line or Trethowan sign as a line along the upper edge of the femoral neck that should intersect the lateral femoral head in a normal hip (right) and has no contact with the slipped hip (left).

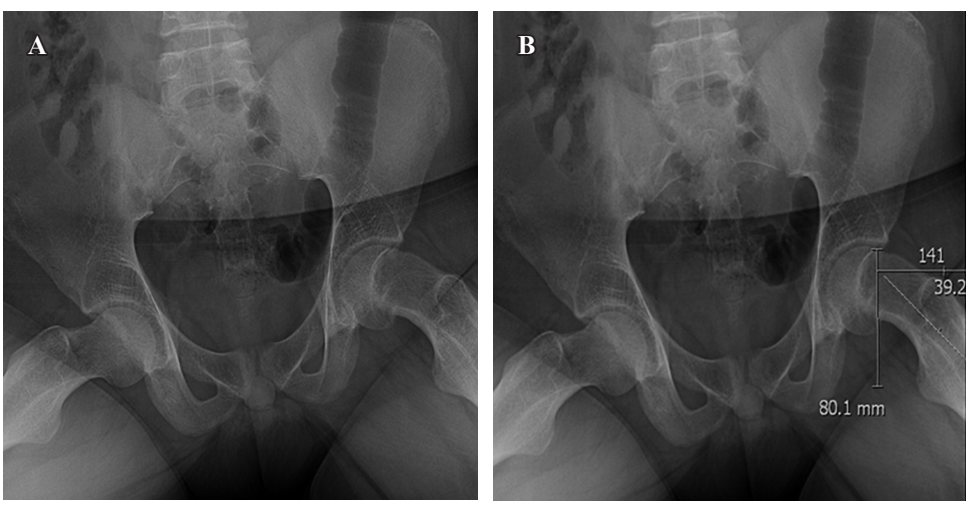

Figure 2:

Lateral frog-leg radiograph of both hips. **A)** Posterior displacement of the left epiphysis relative to the metaphysis that translates anteriorly. **B)** Southwick method for capito-diaphyseal angle calculation: angle formed by the intersection of a line perpendicular to the transepiphyseal line and another along the axis of the femoral neck. This demonstrates the degree of tilt. The magnitude of the affectation is calculated by the difference between the affected side and the healthy side (< 33o degree I; 34-50o degree II; > 50o degree III).

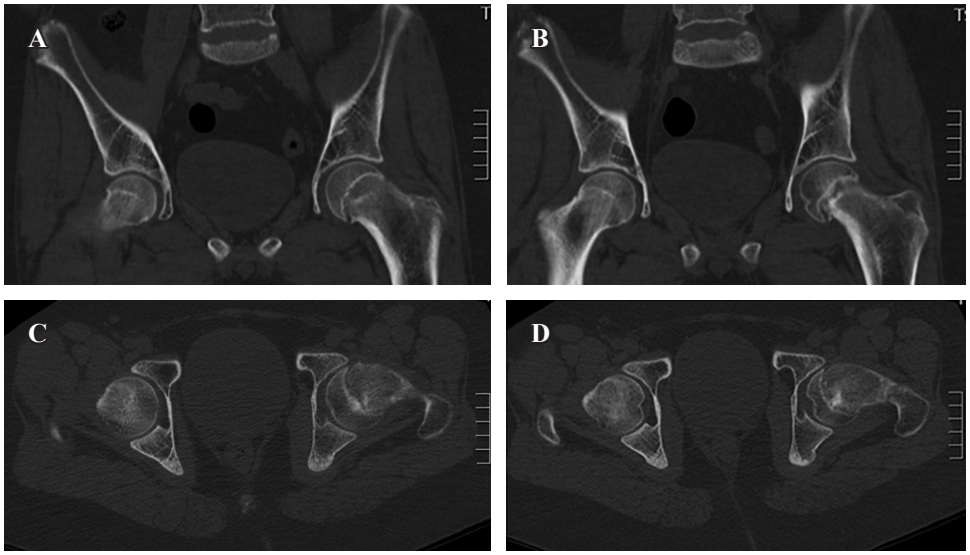

Figure 3:

A-B) Computed tomography (CT) scan coronal sections showing discontinuity between epiphysis and metaphysis. **C-D)** CT scan axial section showing posterior displacement of the epiphysis relative to the metaphysis.

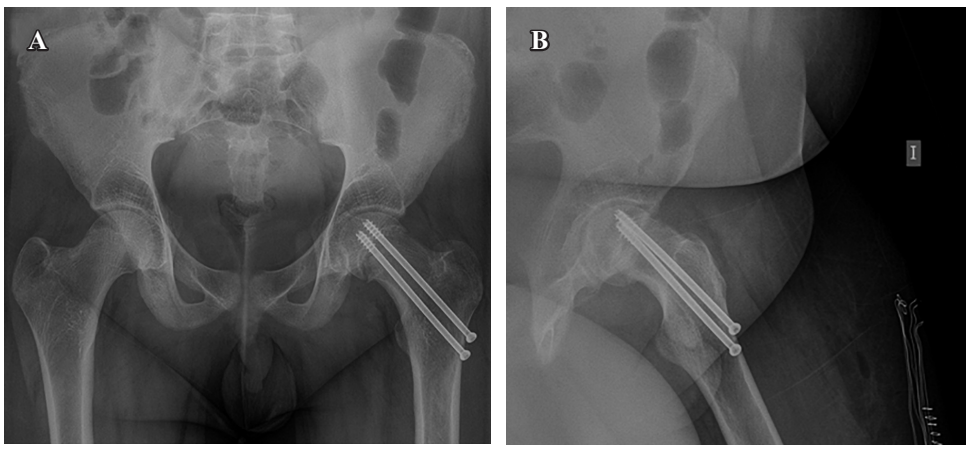

Figure 4:

Postoperative radiographs. **A)** Pelvis anteroposterior. **B)** Axial views showing the *in situ* osteosynthesis of the proximal femoral physis with two cannulated screws.

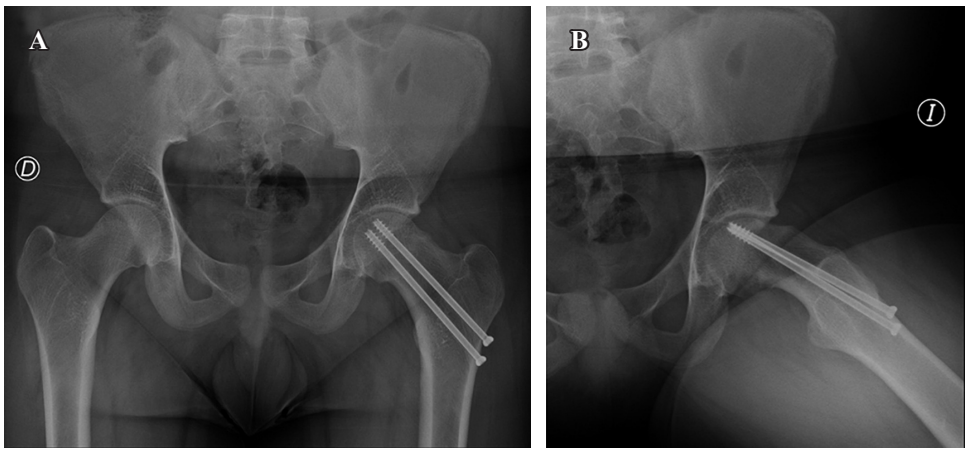

Figure 5:

Radiological evaluation at two-year follow-up. **A)** Pelvis anteroposterior. **B)** Axial radiographic views, showing consolidation and physis closure, without evidence of implant failure.

(0.4 mUI/ml, 0.1 mUI/ml, 0.06 ng/ml, respectively).

The physical and skeletal features of the patient, together with the laboratory data, indicated the possibility of a HH. A brain MRI showed no abnormalities, being the diagnosis hypothalamic-pituitary pathology discarded. Subsequently, a more detailed anamnesis during a follow-up visit revealed the presence of anosmia, that had gone unnoticed. This last discovery provided sufficient data to achieve the diagnosis of KS, which was shortly after confirmed by genetic testing. Consequently, hormonal replacement therapy was initiated.

Discussion

Herein, a case of a young male adult who developed a subacute SCFE and its urgent treatment is presented. The interest of the case lies in the further diagnostic process accomplished, promoted by the peculiar characteristics of the case, which in association with a later perceived anosmia, lead to the diagnosis of KS. The skeletal immaturity accompanying this syndrome could explain the development of a delayed-onset SCFE in the current patient.

KS is the most common form of isolated HH causing delayed puberty and skeletal maturation. Characteristically, there is a gonadotropin-releasing hormone (GnRH) deficiency associated and anosmia.[4] Its incidence is about 1/8,000 men, and 1/40,000 women. The majority of the reported cases are sporadic, but familial forms have also been described. It has an autosomal dominant, autosomal recessive or X-linked recessive inheritance pattern. The age of onset lies between 14-16 years when patients or their families consult for lack of sexual development. In the current case, neither the patient nor their family had previously consulted for delayed physical maturity. Also, the smelling impairment, as the main clinical feature associated with KS, had not been perceived before.

Else ways, delayed-onset SCFE is the definition for an SCFE that occurs during adulthood, when, theoretically, the bone physis should be fused. Its true prevalence is unknown,[8] owing to its asymptomatic course in some patients, appearing as a random radiographic finding. On the contrary, the incidence of SCFE in the adolescence is estimated in 10/100, 1,000 per year.[1,2] The SCFE pathogenesis is explained by a thinning and weakening of the perichondral ring and sliding through the growth plate during adolescence.[9] Histologically, the hypertrophic zone appears to be the debilitated layer of the physis. Moreover, in skeletally immature patients, the proximal femoral physis is still vertical, biomechanically resulting in increased shear forces.

The etiology of SCFE is not clear, presenting a multifactorial origin. These include mechanical factors (shear forces acting at a weakened physis), traumatisms, endocrine disorders (hypothyroidism, growth hormone deficiency, panhypopituitarism, etc.), genetic syndromes (Down syndrome, Klinefelter syndrome, renal osteodystrophy, etc.), toxics and drugs (chemotherapy, radiotherapy or prolonged exposure to corticosteroids) and idiopathic factors. Ensuing the previous, different authors have postulated a likely hereditary factor involved.[10,11,12]

Loder et al.[13] examined 85 patients with SCFE and associated endocrine pathology, finding that hypothyroidism was the primary endocrine disorder causing SCFE in adolescence, while HH predominated in adulthood. A review of 22 published cases,[7] attributed the adult SCFE to the endocrinopathy and reported that craniopharyngioma happens to be the most frequent hypothalamic tumor in adults with SCFE, as has also been reported in children.[14] A 2016 review on the subject performed by Macia-Villa et al.[8] summarizes the different causes for a delayed-onset SCFE since 1940, being endocrine disorders, predominantly pituitary pathology and hypothyroidism, and idiopathic causes, the most frequent etiologies, similarly to the described causes for the youth. Recently, Speirs et al.[15] carried out a new bibliographic review on the topic, gathering all the delayed-onset SCFE published cases, and all were in relation to underlying endocrine disorders or pituitary tumors, except for two. Other authors point out that due to the relation existing between sexual and growth hormones, the lack of androgen production can lengthen the time needed for physis closure in adults, resulting in an unstable and weak physis, prone to slippage.[16] Moreover, some authors affirm, that, several of the cases of premature hip osteoarthritis are sequelae of SCFE,[17] and it has been hypothesized whether it is a residual SCFE that happened during adolescence or an acute displacement in an adult with none-closed physis.[8]

After a detailed literature review, we were only able to find 3 cases of delayed-onset SCFE in relation to KS.[5,6,7] The case herein presented is the fourth published that reports this combination, in which the HH that accompanies a KS is the responsible for the delated physeal closure that plays a fundamental role in the development of the delayed-onset SCFE. Among the cases published, one is a female patient, and the other two correspond to males, as the patient of the current report. The range of age at diagnosis is 19-29, being the age of our case in-between these. Regarding the hip injury treatment, all of the patients underwent surgical intervention, one in-situ fixation, as our case, one had a closed reduction and internal fixation with two pins, while the other was operated three years after due to avascular necrosis and had a hemiarthroplasty implanted. Two of the three patients also had hormonal replacement therapy as a medical treatment. Contralateral prophylactic fixation was not performed in any of these patients. This is a controversial topic in children, being patients and family's preference crucial.[18] In adults, Song et al.[7] reported no diagnosis of sequentially affected contralateral hips and stated that careful observation was enough. We also consider that treatment of the underlying cause when possible is essential. Other data regarding the reviewed cases are displayed in *Table 1*. Amid these cases, to our knowledge, the current case is the only one in which the delayed-onset SCFE warned the surgeons to further investigate the cause of the retarded physical maturation of the patient, and after a thorough workup, the diagnosis of a KS was made, and appropriate hormonal replacement therapy was applied.

All in all, adult SCFE is highly associated with underlying endocrine disorders, pituitary tumors, or genetic syndromes that cause delayed skeletal maturity. When managing a patient with no previous medical history, a further investigation including a detailed history, physical examination, evaluation for endocrine disorders,

Table 1: Cases reported in the literature that present a slipped capital femoral epiphysis in the context of a Kallmann syndrome.

Autor (year)	Age (years)	Sex	Associated conditions	Trauma history	Side	Stability/acuteness	Severity (degree)	Interval between symptoms and treatment	SCFE treatment	KS syndrome	FU (years)
Takahashi, 1997[5]	22	Male	Arachnoid cyst, empty sella	—	Right	Acute	—	3 years	Hemi-arthroplasty	—	—
Choy WS, 1991[6]	18	Female	—	No trauma	Right	Acute	Moderate	2 weeks	CRIF 2 pins	HRT	1
Song, 2015[7]	29	Male	—	Long distance walking	Left	Stable	Mild	3 days	Single screw *in situ* fixation	HRT	7
Current report, 2020	23	Male	—	Exercise	Left	Stable	Mild	2 weeks	Two screws *in situ* fixation	HRT	2

CRIF = closed reduction and internal fixation. FU = follow-up. HRT = hormonal replacement therapy. KS = Kallmann syndrome. SCFE = slipped capital femoral epiphysis.

screening for pituitary tumors, and genetic testing should be accomplished to diagnose the primary condition and establish the appropriate treatment. Considering the current report, a total of four cases of delayed-onset SCFE have been described in association with a KS.

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