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Slipped capital femoral epiphysis (SCFE) – Epidemiology, Aetiology, Pathomechanics & Outcomes

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Abstract

Slipped Capital Femoral Epiphysis is a common paediatric hip disease and the incidence is on rise over the years. Various epidemiological factors have been associated with it and there is also a trend of changing patterns with respect age of presentation and pathomechanics. This article primarily reviews the epidemiology, aetiology, pathomechanics and outcomes in slipped capital femoral epiphysis.

Keywords: Slipped capital femoral epiphysis, epidemiology, Pathomechanics

Introduction

Slipped capital femoral epiphysis (SCFE) is the most common adolescent hip disorder which is associated with both short-term and long-term morbidity to the hip function. Over the years there is a steady increase in incidence of SCFE globally and there is a clear association established between increasing childhood obesity and increase in the incidence(1–6). The knowledge of post-SCFE deformity leading to femoroacetabular impingement and the controversies surrounding the management make SCFE one of the most discussed topics in the orthopaedic literature.

Epidemiology and demographics

Incidence: The overall prevalence of SCFE is between 0.71 to 10.8 per 100,000 children(7). The reported incidence is variable in different parts of the world. It ranges from as low as 0.2 per 100,000 in Eastern Japan to as high as 17.15 per 100,000 in Northeastern United States(8).The incidence in India is not known as there are no population based studies reported in SCFE.

A steady increase in the incidence of SCFE over the past few decades has been reported in many studies across the world. In 2008, Benson et al reported a dramatic increase in the incidence of SCFE in New Mexico to 6 per 100000 children in the highest risk age group compared to that in 1970(2). Hagglund et al published that there is increase in SCFE incidence in Sweden in 1984 and concluded that it followed a periodic pattern with peak once in 20 years(5). Song et al from Korea and Noguchi et al from Japan have reported a significant increase in the incidence rates of SCFE over the last three decades(4,6). Nguyen et al in 2011, reported increased incidence of SCFE in South Australia, particularly in the indigenous Australians.(9)

Racial variation: There is significant difference in the incidence of SCFE based on the race. It is reported to be highest in the blacks and lowest in Whites and native Americans. The relative racial frequencies of SCFE (using the white population as a reference of 1.0) were estimated as 4.5 for Polynesians,2.2 for blacks,1.05 for Amerindians,0.5 for Indonesian-Malay (Asian) and 0.1 for Indo-Mediterranean people based on an international multicentre study(3). Though difference in the acetabular morphology has been proposed as a reason for incidence in racial difference, Loder et al in a study has shown that the racial variation in CE and Sharp's angle did not follow the same pattern of racial variation in incidence of SCFE(10).

Gender and Age: Overall there is a male predominance of this disorder irrespective of the age of presentation. However, there are differences in gender by race, with Indo-Mediterraneans having the highest proportion of boys(90%) and Polynesians with an equal distribution in males and females(3). Many studies have shown that over the years the age of onset has decreased significantly and this could probably be due faster skeletal maturation of this generation of children(1,5,8).

Bilaterality: The reported prevalence of bilateral SCFEs is between 20 and 80% (11). About 50-60% of them present with simultaneous bilateral involvement and in those presenting with sequential hips, most of them happen within the next 18 months(8). Bilaterality is reported to be highest in Africans, followed by Asians, Hispanics and Whites in that

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order(3).

In a study by Bhatia in 2006, they found that children who had Bilateral SCFE had significantly higher BMI compared to those who had unilateral SCFEs and stressed that patients with high BMI are prone for developing bilateral SCFEs(12). BMI for age is more appropriate for defining obesity in children rather than BMI. It has been shown that there is an association between high BMI for age and bilateral SCFE at presentation(13).

Seasonal variation: Seasonal variations in the incidence of SCFE has been reported in literature based on population based studies(14,15). Slight seasonal variations in the incidence has been reported based on the geographic locality. More than half of the SCFE occurred during the summer months in north of 40 degrees latitude and during the winter months in south of 40 degrees latitude(1).

Aetiology

Slipped capital femoral epiphysis could either be idiopathic or secondary to endocrinopathy, renal failure or radiation therapy which are termed as "atypical SCFEs". It has been shown that atypical SCFEs are more common in patients less than 10 years and more than 16 years of age and those with body weight < 50th percentile for that age. This led way to the description of "age-weight test" by Loder et al in 2001(16). The age-weight test is said to be negative when the age of the patient is <16 years and weight is more than 50th percentile. In a child with negative test, there is 93% chance that the SCFE could be idiopathic rather than atypical. In other words the negative predictive value of age-weight test is 93%.

Though the exact cause of SCFE is not known, over the years we do have an understanding of the possible pathogenesis of the problem. The failure of the physis is the final event occurring due to a combination of weak physis and abnormal forces through the weak physis. Dennis Weiner stated that "it should be considered as a disorder of puberty due to a combination of pubertal mishaps occurring in concert with normally evolving biologic events."(17)

Both biomechanical and biochemical factors play a role in the pathogenesis of SCFE. Obesity, femoral retroversion and increased obliquity of the physis can lead to abnormally high forces passing through the physis. Endocrine and metabolic disorders can lead to weakness of the physis due to changes happening at the cellular level.

Biomechanical factors

Femoral morphology: The mean femoral anteversion in obese children is much less compared to their normal counterparts. Also, patients with SCFE have increased vertical orientation of physis compared to the normal hips. This relative femoral retroversion along with increased physeal obliquity increases the forces transmitted through the physis(18).

Acetabular morphology: Lateral overcoverage of the femoral head with relative medialisation can lead to increase in the shear forces across the physis. Kitadai et al reported increased center edge angles in a series of 104 patients with SCFE as compared with controls matched for age, sex, and race and suggested that increased femoral head coverage may indeed be part of etiology in SCFE(19).

There are varying reports on the version of acetabulum in children with SCFE. There are few studies which report significant retroversion of the acetabulum even in contralateral hips of children with SCFE(20,21) while some authors have reported the acetabular retroversion to be normal(22).

Histology of physis : The physis in SCFE has many histologic and ultrastructural differences compared to a normal physis and may be rightly called as "sick physis". The thickness of the growth plate is increased upto 12 mm and the proliferating and hypertrophic zones are increased occupying 60-80% of the width of the physis. The chondrocytes are arranged in clusters with loss of normal columnar arrangement. The extra-cellular matrix has abnormal longitudinal septa with loss of collagen and reduced proteoglycan content(23). Ultrastructural studies have revealed that the chondrocytes are elliptical and smaller than normal and the collagen fibers are thinner in the physis with SCFE(24).

Hormonal influence on the physis: Since obesity and pubertal growth are considered as important risk factors it is pertinent to know about the influence of various hormones on the growth plate. There are five main hormone groups which influences the structure of physis. They are

1. Growth Hormone & IGF – 1 (Insulin like growth factor -1)
2. Sex hormones
3. Leptin
4. Thyroid hormones
5. Glucocorticoids

The secretion of Growth hormone (GH) increases two to three fold during the pubertal growth spurt and this has direct and indirect (through IGF-1) effects on the physis(23). It increases chondrocyte proliferation in the physis.

Androgens and estrogens are responsible for the pubertal growth spurt. They have direct growth stimulating effect on physis and also indirect effect through enhancement of GH secretion which increases circulating IGF-1 and stimulates clonal expansion and chondrocyte growth in the proliferating zone(23). Estrogens play an important role in regulating linear growth in both the sexes and subsequent closure of the physis. Patients with SCFE often have delayed sexual maturation and thus have delay in the closure of the physis.

This along with increased force transmission due to obesity may potentially lead to slip of the epiphysis.

Leptin is a protein secreted by the adipocytes and the circulating leptin levels are directly proportional to BMI(23). Leptin acts directly on the physis through leptin receptors and causes increase in width of the proliferative zone. A recent study by Helverson et al has shown five times increased odds between elevated serum leptin levels and SCFE, irrespective of the BMI(25). This has opened up a new avenue for research to study the association between leptin levels, SCFE and obesity.

Tri-iodo thyronine (T3) is the active form of thyroid hormone which is essential for differentiation of resting zone cells and hypertrophic chondrocytes. It also has an indirect effect on growth plate by influencing GH secretion. Hypothyroidism leads to growth failure and delayed skeletal maturity. This effect is mediated through Indian hedgehog parathyroid hormone –related hormone (IHH-PTHrH) which is an important negative feedback loop in chondrocyte differentiation(23).

Glucocorticoids facilitates normal growth in physiological concentrations. But, high concentration supresses physis chondrocyte proliferation and cases growth retardation. Also, it increases the apoptosis in the physis mediated through glucocorticoid receptor.

Vitamin D & SCFE: There are few reports published on the association between Vitamin D deficiency and SCFE(26,27) while some authors have shown no correlation between low vitamin D levels and development of SCFE(28). Vitamin D is a fat soluble vitamin and its role in calcium metabolism and homeostasis is well established. Calcitriol, the biologically active form of vitamin D stimulate epiphyseal chondrocytes in a dose-dependent manner and there is also a possible link between Vitamin D and IGF-1(23).

Familial SCFE: The occurrence of SCFE in the same family has been described in the literature(29,30). X-linked dominant, autosomal dominant with variable penentrance and autosomal recessive patterns of transmission has been proposed, though none has been proved.

Pathomechanics

The role of shearing forces across the physis in slipped capital femoral epiphysis has been studied and described with various experimental studies(31–33). All of them have concluded that abnormal shear forces due to pre-existing anatomical changes (coxa vara, retroversion of femur) and over weight across a weak and susceptible physis leads to posterior and inferior displacement of the capital femoral epiphysis in relation to the neck.

Tayton in 2007 proposed that the epiphysis rather rotates over a peg like tubercle called the 'epiphyseal tubercle'(34). The epiphyseal tubercle is described as a conical projection of bone from the epiphysis into the metaphysis measuring about 4mm in length. The presence of this in adolescent hips was studied both on cadaveric adolescent femoral heads as well as radiologically by CT scan(34,35). According to this hypothesis, the epiphysis gradually rotates posteromedially and the epiphysis overhangs the metaphysis. Relative stability is maintained till the point that the epiphysis tilts backwards and the tubercle disengages from the metaphysis and an acute-on-chronic slip happens where it separates completely. This could possibly explain the long history in chronic stable slips.

Similarly, change in the shape of the epiphysis from pleated to flat in adolescence is also described as one of the aetiological factors causing SCFE. A radiological study in about 100 hips has shown the change in shape of the growth plate from childhood into adolescence with decrease in the inter digitations of the physis as the age increases(36).

Natural history and outcomes

The natural history of this condition is directly related to the onset and severity of slip and how it was treated. All acute slips are very painful and unstable and presents usually like traumatic epiphyseal separation. Most of them seek medical attention and they undergo either in-situ pinning or capital realignment. The reported rates of AVN in unstable slips vary between 0 and 58%(37). There has been few recent reports that the surgical dislocation of hip and development of retinacular flap for capital realignment actually decreases the AVN rates in an unstable setting(7). According to a meta-analysis published in 2010, unstable slips had 9.4 times increased risk of developing AVN compared to stable slips, irrespective of the type of intervention done(38).

An untreated mild slip is bound to progressively slip further until it is stabilised by some means either by an implant or till the physis matures by itself. A classical chronic stable slip which is progressively slipping results in severe retroversion with an elongated neck and a posterior curvature simulating the shape of a banana in the axial section of CT scans or MRI (Fig 1). The varus component of the deformity is not as much as the retroversion as the epiphysis tends to slip posteriorly gradually. Typically these patients present with a painless long standing limp and an out-toeing gait pattern due to retroverted hip or a painful limp due to a labral tear. A trivial trauma can lead to an acute on chronic slip where the presentation would be similar to that of an unstable slip.

In 1991, the Iowa group published the long-term outcomes of SCFE with a mean follow-up of 41 years. This is the largest series with the longest follow-up which stated that the natural history of a malunited slip depends on the severity of the slip and the complications related to treatment(39). They

concluded that irrespective of severity of the slip, in-situ pinning gave the best long term outcome in terms of delay in degenerative arthritis. Following this there has been many studies which have concluded that in-situ pinning in general has good functional outcomes in the long-term with minimal complications(40–42).

In 2009, Pablo Castenada from Mexico reported the mid-term results of 105 patients with grade III slips treated by in-situ pinning and concluded that all of them had excellent to good outcomes(43). In 2013, the same group from Mexico published their long-term results of in-situ pinning and concluded that even patients with Grade I slip developed the typical "pistol grip" deformity of the proximal femur and alluded that FAI due to this deformity could be the cause for early osteoarthritis in these patients(44).

We now know that slips treated by in-situ pinning leads to an abnormal morphology of the proximal femur which can potentially cause a dynamic conflict in movement between the acetabulum and proximal femur. This conflict is termed as "femoro-acetabular impingement(FAI) " and is a precursor of osteoarthritis. But, the question which is still unanswered is that how many of these patients with radiological FAI would turn out to be symptomatic and would need some surgical intervention at a later date. This probably depends on multiple factors like activity level of the patient, severity of the slip, obesity and whether the patient is putting that hip into the position of impingement in day-to-day activities which would all lead to increased wear and tear. At this point in time, we could safely conclude that all the patients with radiological abnormalities needs to followed-up for symptoms of FAI and needs to be addressed once they develop symptoms to prevent progression of FAI into frank osteoarthritis.

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