Abstract

Purpose: The aim of this study was to assess the safety and efficacy of Intertrochanteric Imhäuser osteotomy (IO) combined with osteochondroplasty in treatment of moderate-severe stable slipped capital femoral epiphysis (SCFE) on short term basis.

Methods: Nineteen Patients (20 hips) with moderate-severe stable SCFE were surgically treated, between 2014 - 2016, by IO combined with osteochondroplasty and followed up for 12-24 months. The cases were between 12 and 18 years at the time of surgery, and complaining of variety of symptoms that included pain, limping, limited range of motion (ROM), and/or abductor weakness. The outcomes were assessed using clinical and radiological parameters as well as functional outcomes measures (HHS and WOMAC index).

Results: There were statistically significant improvement of ROM particularly flexion, internal rotation (IR), and abduction. HHS and WOMAC index were improved significantly with final follow up scores at 86.76 and 6.4%, respectively. The radiological parameters showed significant improved as regard Southwick angle (mean 12.8°), alpha angle of Nötzli (mean 29.85°), Hilgenreiner epiphyseal angle (mean 37.65°), neck shaft angle (NSA) (mean 140.63º), and acetabulo-trochanteric distance (ATD) (median14.1mm) at the end of follow up period.
Conclusions: IO combined with osteochondroplasty surgery is a good option in moderate-severe stable SCFE treatment.

Key words: slipped capital femoral epiphysis, stable SCFE, Imhäuser osteotomy, osteochondroplasty.

Level of Evidence: Level IV, prospective case-series therapeutic study.

INTRODUCTION

Slipped capital femoral epiphysis (SCFE) is an adolescent hip disorder where the femoral neck and shaft displace relative to the femoral epiphysis via the physis. Its incidence is 0.2 to 10 per 100.000. The mean age at diagnosis is 13.5 years in boys and 12 years in girls.[1] Most of the displacement is in varus (upward, anterior, and external rotation displacement of the neck) but can occasionally be in valgus (downward, posterior, and internal rotation displacement of the neck).[2] Its pathogenesis and etiology are still unknown, but it is the result of high load across abnormally weak physis. Obesity, endocrine, and chronic systemic diseases are known to be associated with SCFE.[3]

SCFE is classified both clinically and radiologically. Clinically, it can be classified into acute (those with symptoms last less than 3 weeks), chronic (those with symptoms last more than 3 weeks), and acute on top of chronic (those with chronic symptoms initially with subsequent development of acute symptoms).[4] Another clinical classification, Loder et al [5] classification, depends on physeal stability that classified it into stable (those who are able to walk with or without crutches) and unstable (those who are unable to walk even with crutches) with avascular necrosis (AVN) risk as high as 47 %. Radiological classification (Southwick), which is based on

lateral head shaft angle, places the slip into mild (< 30^0), moderate ($30\text{-}50^0$), and severe (>50^0). [6]

The long-term prognosis of SCFE is highly influenced by the severity of the slip. In mild slips, in situ pinning is a well-established treatment method where remodeling of the residual deformity is usually the case.[7] In moderate and severe slips, the prominent metaphysis may impinge anteriorly on the acetabulum causing femoro-acetabular impingement (FAI) especially with hip flexion.[8] The repetitive mechanical abrasions of the metaphysis against the acetabular cartilage can trigger osteoarthritis.[9] The cam lesion developed in SCFE can be quantified using alpha angle of Nötzli: the angle on axial views between two lines: a line from the center of the femoral head through the middle of the femoral neck and a line through a point where the contour of the femoral head-neck junction exceeds the radius of the femoral head. An angle above 55^0 is abnormal and indicates a cam lesion. [10]

The treatment of moderate to severe SCFE is highly controversial. The primary objective of SCFE treatments is stopping further slippage, prevention of impingement, osteoarthritis, chondrolysis, and AVN. [11] Some authors recommend in situ pinning, but it cannot correct the deformity present in severe slippage and there is a distinct correlation between it and osteoarthritis (OA) 20 years after treatment.[12] Dunn [13] described subcapital femoral neck osteotomy to

correct the deformity where the tension on posterior flap was reduced by wedge resection. Although it corrects the deformity completely, the risk of AVN reported range from 10 – 100 %.[14] In 1992, Ganz et al [15] described a modification to Dunn procedure where a safe surgical dislocation (SSD) used to solve AVN problem, but it is technically demanding with lack of long term results studies and is used usually for unstable cases [14] or stable cases with open physis.[7] Although Ganz et al [15] and others [9],[11] reported no AVN risk in their case series, other studies reported AVN risk ranges from 4.5% to 26% [16-18] with the majority occurred in unstable cases with open physis. Others also reported increased incidence of AVN in skeletally mature patients. [19] Kramer et al [20] described an extra-capsular basal neck osteotomy in an attempt to decrease the risk of AVN. However, AVN and chondrolysis have still been reported.

The use of Intertrochanteric osteotomy in correcting the deformity has the advantage of being a safe option with no risk of AVN theoretically, but the ability to correct the deformity has limits. Southwick described an osteotomy at the level of lesser trochanter which corrects the varus and extension deformity with no derotation to avoid abductor weakness.[21] Imhäuser [22] described an osteotomy proximal to the lesser trochanter which involves valgus, flexion, and internal rotation. Several studies reported the use of Imhäuser osteotomy with satisfactory outcomes with clinical and radiological

improvement on long term follow up as long as 39 years and hence the delay of total hip replacement (THR) need. [23-25] As it cannot correct the deformity completely, being away from the center of deformity, some admitted the need of additional procedure to deal with the residual deformity persisted after its use that may explain the incomplete improvement of some cases.[7],[24],[25] Bali et al added open neck osteochondroplasty to correct the residual deformity with promising results.[7]

Based on the previous mentioned facts, we hypothesize that Imhäuser osteotomy offers a good balance of correcting the deformity of SCFE while avoiding as much as possible the most feared complication which is AVN. To try to avoid long term complications of FAI, we chose to add osteochondroplasty to the treatment policy.

AIM OF THE WORK

The aim of this study is to assess the short-term outcomes of Imhäuser intertrochanteric osteotomy combined with osteochondroplasty in treatment of moderate-severe stable SCFE.

Chapter 1
EPIDEMIOLOGY AND PATHOLOGY OF SCFE

Epidemiology

SCFE is the most common hip pathology affecting adolescents between 8 – 15 years old.[26] It is a proximal femoral disorder in which the femoral neck and shaft displace in relation to the femoral head, that stabilized in the acetabulum. Most of the displacement is in varus (the epiphysis appears in postero-caudal position and medial in relation to the neck); however, it can occasionally be in valgus (the epiphysis appears in antero-cranial position and lateral in relation to the neck).[2]

The incidence is 0.33-24.58/ 100,000 children 8-15 years old. Variations of the incidence among different geographic areas are evident. It is 10.8/ 100,000 in the United States, 0.76 in females – 2.22 in males /100,000 in Japan, 9.66/ 100,000 in Scotland, and 4.4 in girls – 5.7 in boys /10,000 in Sweden.[2],[27].

There are obvious differences of the occurrence among ethnic groups. The relative racial frequency, in comparison to Caucasian who are referred to 1, was 5.6 for Polynesians, 3.9 for Blacks, and 2.5 for Hispanic. [2] These differences are attributed to different reasons. Firstly, the average body weight among different races are variant with the highest in Polynesians and Blacks, that reflect the impact of obesity on

SCFE occurrence. Secondly, dark colored skin people have less vitamin D absorption potentiality compared to fair colored individuals increasing the susceptibility of vitamin D deficiency, a hormone that is essential for physis growth and mineralization.[26] Furthermore, it is claimed that people with deeper acetabulum have higher risk of shearing forces among the physis increasing the incidence of SCFE among them. This is proved by Loder et al [28] who found that Blacks have the deepest acetabulum (39^0 average center edge angle "CE angle") compared to Caucasians (38^0 average CE angle) and Japanese (32^0 average CE angle), which are the shallowest.

The figure has been rising in the recent decades at certain locations. It has been tripled in New Mexico over 4 decades, the same with Scotland over 2 decades, and 5-fold rise in Japan over 3 decades. These were attributed to the global increase of obesity.[2]

There is a male predominance of occurrence over females with a ratio at 1.1:1 – 4.1:1 according to the race. [27] The age of symptoms onset has decreased significantly over the last century. It has dropped from 13.5-year-old to 12-year-old in boys and from 12-year-old to 11.2-year-old in girls over 15 years. [2]

There are many literatures depicted seasonal variation in the onset of symptoms of SCFE patients with different explanations. The first study discussed this issue was conducted

by Ferguson and Howorth in the United States in 1931. They noticed a seasonal peak between July and November among boys and girls. They attributed these to the increased playing activities through this period. In 1958, Andren and Borgstrom demonstrated seasonal variation in Sweden, blaming it to a substance ingested in milk as it peaked at months of milk pastry. Another Swedish study demonstrated a peak between May and August among females only. Maffuli and Douglas studied the variations of the onset among 1103 patients all over Scotland. They observed an autumn peak, which was significant among males rather than females. They did not identify a specific etiological explanation for this feature, but vitamin D deficiency, a result of insufficient sunlight exposure, and increased weight during these months were accused. [29]

Further studies were conducted to confirm this observation at a wide geographical area and among ethnically diverse populations. Loder et al [30] studied 1630 children from 33 different centers worldwide. They found seasonal variation at areas north to 40^{o} latitude, with North America peak at June and Europe peak at July, but not at areas south to it. No gender or ethnic based groups differences were noted. A state of subclinical rickets with vitamin D deficiency, owing to little sun exposure, was suggested to cause these variations. The picture was supported by Brown who analyzed patient records of 4690 patients all over the United States. He concluded a peak between August and October which was evident in

northern states compared to southern ones, in whites compared to blacks, and to some degree in girls compared to boys. [29]

Recently, Loder et al has published the largest ever study with 10350 patients records over 10 years. They described for the first time a double peak at the areas between 31^0 – 35^0 latitudes and a peak above 35^0 Latitudes; nevertheless, no variation existed below 30^0 latitudes. They also found a correlation between the variations and the climate. Areas with humid cold climate demonstrated seasonal variations in comparison to the hot arid ones. The same trend in areas with sun exposure <2500 hours per year compared to the sunny areas. The incidence of SCFE reached the top at mid-August, but no difference with various ethnic or gender groups. [31]

Based on the facts that Vitamin D level in children vary throughout the year, its deficiency is more in obese Blacks compared to non-obese Caucasians. [32] , and the difference of SCFE incidence in relation to sunlight exposure, they make a link between vitamin D deficiency and SCFE occurrence. This supports the view of some authors who believe that SCFE represents a state of subclinical rickets with defective physeal mineralization. [30],[33] Furthermore, it is known that the maximum weight velocity among adolescents occurs at the seasons of fall and winter which move in tandem with SCFE occurrence. [34]

Histopathology

The pathogenesis of SCFE is not exactly clear, but it is the result of high loads on normal physis, normal loads on weakened physis, or both. [3]

High loads on the physis occur in cases of increased body mass index (BMI), femoral retroversion, or increased physeal obliquity. [35] There is a strong relation between increased BMI and SCFE occurrence as it increases the shearing stress across the physis, and adolescents with 95% or more BMI for age are at significant risk of its development. Increased BMI is associated with increased risk of bilateralality of the disease. Patients with bilateral SCFE have significantly higher BMI for age compared to unilateral counterparts. Moreover, patients with unilateral SCFE who progress to be bilateral have considerably higher BMI for age in comparison to those who do not progress. [36] Regarding femoral retroversion, there is a correlation between it and reduced femoral neck shaft angle (NSA) that is together with the tendency of increased physis obliquity in adolescents period change the forces applied on the femoral head from compression to shear increasing the risk of physis instability and slippage. [37] Some authors also identified head neck asphericity to be a mechanical factor predisposing to SCFE rather than being the result of it. Subclinical contact between the head neck junction and the acetabulum would accumulate a mechanical stress across the physis that end eventually in its failure and slippage. [38]

Physeal weakness might be at the cellular level of chondrocytes in the hypertrophic layer as a result of dysregulation of different pathways signals that are affected by hormonal imbalance or systematic diseases.[3] Hypothyroidism, hypogonadism, hypopituitarism, vitamin D deficiency, renal osteodystrophy (ROD) as well as radiation exposure have been incriminated individually to be involved in SCFE pathogenesis.[27]

The normal physis is formed of four different zones. Resting zone contains inactive germ-like cells with abundant extracellular matrix rich in collagen, proliferative zone comprises proliferating chondrocytes arranged in layers parallel to the bone axis, hypertrophic zone with maturating non-dividing chondrocytes with less extracellular matrix, and zone of provisional calcification with vascular invasion and islands of calcified cartilages. [39] [figure 1] The growth plate is surrounded by dense fibrous perichondrial ring, which is important in the stability of the physis. [3]

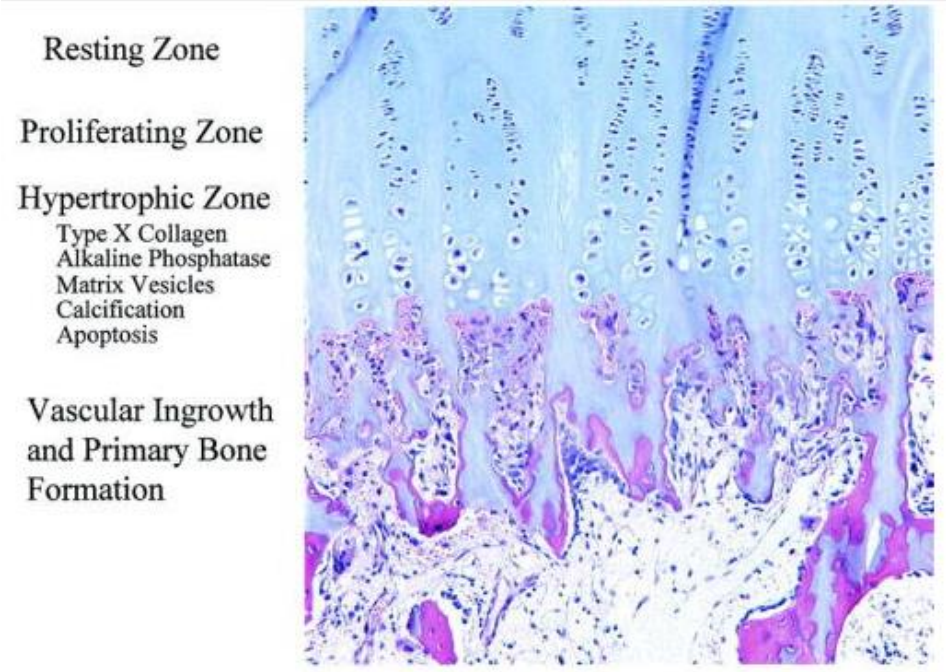

Figure (1): Histological appearance of the human growth plate. (magnification, ×100). [3]

In SCFE, there are histological changes in the physis, both cellular and extracellular. However, it is not clear whether it represents an etiology or a result of the condition. [3]

Regarding resting zone, there are no many changes in it apart of cartilage cells clusters at some tissue samples. On the other hand, hypertrophic and proliferative zones face the main changes. Chondrocytes lose the normal longitudinal arrangement in the growth plate, decreased their numbers as a result of increased apoptosis, and increased nucleus and cytoplasmic density with increased glycogen contents. Extra-cellular matrix shows decreased collagen content with

abnormal septa, decreased normal proteoglycan with increased abnormal ones, and islands of calcification and callus formation as a consequence of increased matrix vesicles rich in calcium phosphates, hydroxyapatite and matrix metalloproteinases. [3] [figure 2]

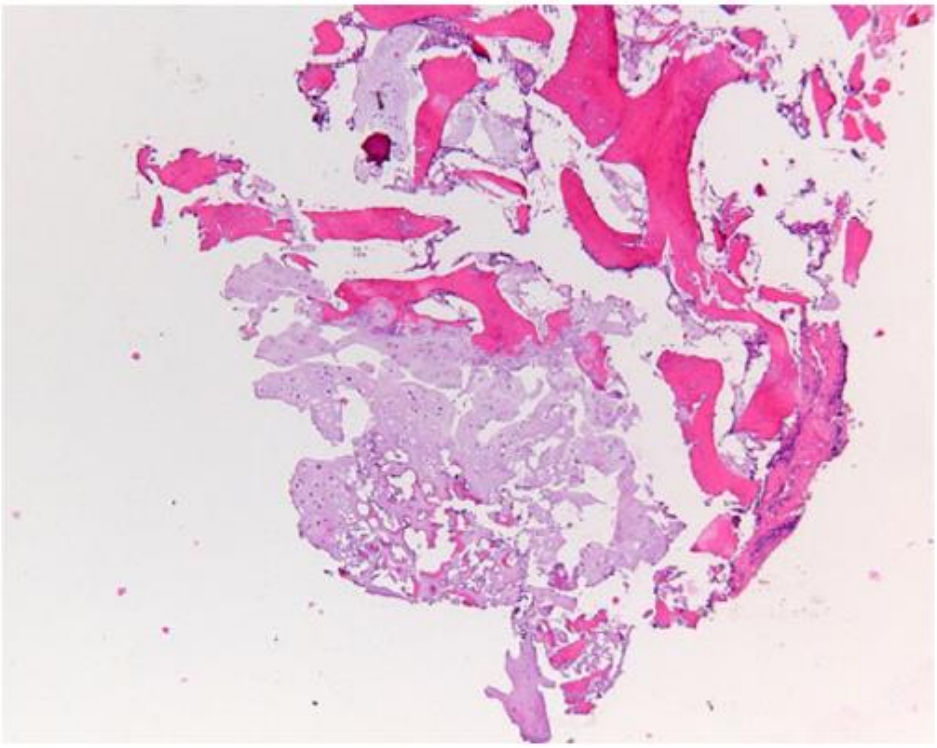

Figure (2): Abnormal physis of SCFE. At the top-right of the image, ossification is visible, and at the bottom, the disorganized cartilage from the growth plate is visible. The normal regular arrangement is lacking. [3]

Another theory of physis weakness discussed a cumulative stress, for example hormonal changes or minor traumas, around epiphyseal tubercle which induces a reactive process resulting in focal osteolysis around it. Epiphyseal

tubercle is an eccentrically posteriorly situated blunt projection from the epiphysis into the metaphysis that acts as key stone stabilizer for the epiphysis in the skeletally immature individuals. Peri-tubercle osteolysis could eventually result in a rotational deformity of the epiphysis around its fulcrum, the epiphyseal tubercle. This theory is valid for stable SCFE in which the progression is slow but not for unstable ones in which complete tubercle dislodgment from its metaphyseal groove occur. [40]

During puberty, linear bone growth is induced and controlled by synergetic effects of hormonal and growth factors especially growth hormone (GH), insulin like growth factor-1 (IGF-1), sex hormones, glucocorticoids, and thyroid hormones. Local IGF-1 plays a major role in controlling the growth by expressing receptors to several hormones and growth factors and it is the key for growth regulation. [3]

GH, a peptide hormone produced by pituitary gland, stimulates clonal expansion of the chondrocytes in the proliferating zone directly and indirectly, stimulating the release of hepatic IGF-1 and acts on local IGF-1 to produce such effect. [3] Its production increases 2-3 folds in both basal rate and pulse amplitude during the puberty and its abnormalities are associated with weakened physis raising a risk of SCFE development. [3],[27]