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Long term oral steroid medication induce hip dislocation in pediatric : a case report

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Abstract

Introduction: Oral corticosteroids play a major role in the treatment of diseases such as dermatitis. In adults, osteoporosis is a well-known and major complication of oral corticosteroid treatment. A recent study reported that the risk of hip fracture and dislocation was doubled in adults using higher doses of oral corticosteroids. Its findings suggested that the adverse skeletal effects of oral corticosteroids manifest rapidly. Several studies have reported decreased bone density in children taking oral corticosteroids. No studies have evaluated the risk of dislocation in children taking oral corticosteroids. **Case description:** This case report describes an 3 year old female with hip pain and a history of fall to ground hit the left hip. **Discussion:** Several risk factors that play a role in the hip dislocation such as history of dermatome disease, age, dislocation type, taking oral corticosteroid medication induce hip dislocation in children. **Conclusion:** Hip dislocation in pediatric are uncommon and differ from elderly. Corticosteroid has a high risk of dislocation and fracture. Complications can be minimized by early mobilization with adequate nutrition and rehabilitation are needed to bring a good outcome for the patient.



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Introduction

Traumatic hip dislocations in childhood are rare injuries [2]. The current evidence is based mainly on many case reports and a few small case series. Unlike adults, traumatic hip dislocation in children can be caused by minor trauma and therefore hip fracture – dislocation are not as frequent in pediatric[2]. The mainstay of treatment is urgent close reduction to decrease the risk of avascular necrosis and the goal is to achieve a congruent and stable hip joint [3]. The treatment recommendation in childhood is to perform a close reduction under general anesthesia to allow for muscle relaxation to help facilitate the reduction under fluoroscopic monitoring [4]. The goal is to allow for adequate healing of the torn capsule, acetabular labrum and other periarticular soft tissue to decrease the risk of recurrent hip dislocation [5]. Traumatic hip dislocation in childhood is a rare injury with a reported incidence of approximately 7.5% of all hip dislocation; between 2% and 5% of all joints dislocation. male predominance was reported with a ratio of 2:1 to 3:1.² The mechanism of injury is dependent on the patients age and skeletal maturity status [6]. The majority of pediatric hip dislocations are posterior, representing 71-94% of these cases [2]. The energy required to dislocate a pediatric hip increases with age and skeletal maturity. While in children less than 10 years of age, a minimal or low energy mechanism can be associated with hip dislocation and children older than 10 years of age typically required a much higher energy mechanism to cause a dislocation. The direction of most

pediatric hip dislocation is posterior. Likely due to the laxity of soft tissues around the hip in younger children [7]. Traumatic hip dislocation has been classified broadly according to the direction of dislocation (anterior, posterior or central) and by pathoanatomy. The most popular classification system for posterior hip dislocation is that introduced by Thompson and Epstein in 1951.⁷ this classification differentiates simple dislocation (without associated fractures; type I) from those with either associated acetabular rim fractures (type II and III), acetabular floor injury (type IV) of femoral head fractures (type V) [8]. Glucocorticoids are naturally synthesized primarily in the zona fasciculata of the adrenal cortex or can be administered as pharmacologic agents. Glucocorticoids act by binding to the cytosolic glucocorticoid receptor, which is widely expressed among varying cell types. Non-canonical pathways play less important roles, but these nongenomic effects have also been shown to contribute to various mechanisms of downregulating inflammatory markers [9]. In terms of the direct impact on bone, endogenous glucocorticoids at physiologic concentrations may have a role in promoting osteogenesis, while excess glucocorticoids increase osteoclastogenesis and suppress osteoblastogenesis in cell culture, murine and human models. Local metabolism of glucocorticoids in bone cell is controlled by a pair of complementary enzyme, 11 β -hydroxysteroid dehydrogenase types 1 and 2 (Hsd11b1 & Hsd11b2) [9]. In addition, there are indirect effects of glucocorticoids on skeletal health. They

suppress IGF-1, a hormone crucial for general growth and bone also collagen formation. They inhibit calcium absorption from the intestine. Effects of glucocorticoids on suppressing estrogen and testosterone production may also augment deleterious effects on bone. Finally, many of the underlying disease states for which chronic glucocorticoids are prescribed are themselves linked to lower bone mineral density (BMD). Children represent a different approach as bone development is still critical. In children, weight gain and growth retardation are the most frequent adverse reaction to chronic oral glucocorticoid use, but a recent meta-analysis that included a total of 6817 children noted a 21% incidence of decrease bone mineral density [9].

Case History

A 3 years-old girl (weight 13kg) came to emergency department of Wangaya general hospital because of deformity and pain on left waist. Her left thigh hit the ground while playing at her yard. Although the exact mechanism of injury was not known, it was attributed to a low-energy trauma. The physical examination revealed swelling of the left thigh with deformity. Her left thigh found in flexion, adduction and internal rotation. In addition, distal sensory perception and capillary refill time were normal.

The pain score using the face, legs, activity and cry, scale was 3-4. Moreover the hip and knee range of motion was limited due to pain, the extension of the ankle and great toe was normal. We noted leg length discrepancy between both legs about 2cm. fig 01a, 01b and 1c showed the clinical picture and hip x-ray of the patient.

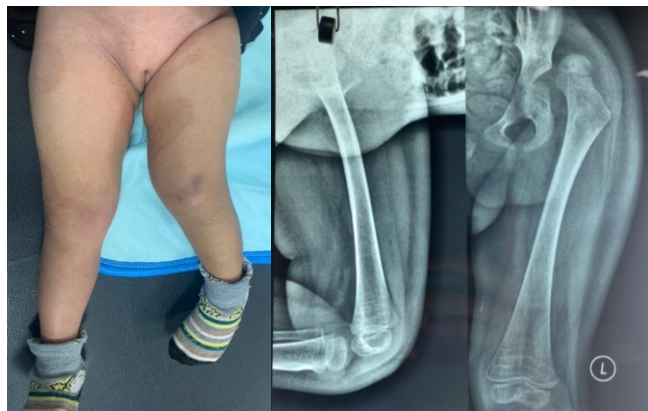


Fig 01a
Fig 01a Clinical appearance at admission
Fig 01b
Fig 01b X-ray examination of the left femur

Since 2 weeks year old, the patient take Triamcinolone corticosteroid oral and interhistine anti-histamine oral twice a daily until 2 years old. Patient also delayed in walk, patient can walk at 13 month years old and still lame in every step on left leg. The patient is the second child of his parents and was born via normal delivery. Her birth weight was 3100gr. During the gestational period, antenatal care was provide routinely by an obstetrician.

Her antenatal care and ultrasonography examination revealed no abnormalities. Due to her dislocation, we decided to treat the dislocation conservatively with a unilateral hip spica cast (fig 02a and 02b). after a period of observation, the patient was discharged.



Fig 02a
Fig 02a clinical appearance post close reduction and unilateral hip spica cast
Fig 02b
Fig 02b X-ray post close reduction and unilateral hip spica cast



Fig 03: X-ray post remove hip spica cast for treated 1 month

Hip spica cast treatment was carried out for 1 month and we decided to remove the hip spica cast (fig 03). At the time of removal, evaluated with ortolani and barlow tests.

Discussion

Traumatic hip dislocation in childhood is a rare injury with a reported incidence of approximately 7.5% of all hip dislocations. Traumatic hip dislocation has been classified broadly according to the direction of dislocation (anterior, posterior or central) and by pahtoanatomy. The most popular classification system for posterior hip dislocation is that introduced by Thompson and Epstein in 1951. This classification differentiates simple dislocation from those with either associated acetabular rims fractures, acetabular floor injury or femoral head fractures [8].

Table 01: Thompson-Epstein classification of posterior hip dislocation

Type I	With or without minor fracture
Type II	With single large fracture of the posterior acetabular rim
Type III	With a comminuted fracture of the posterior rim of the acetabulum with or without a major fragment
Type IV	With fracture of the acetabular rim and floor
Type V	With fracture of the femoral head

The prolonged use of corticosteroids interrupts the bone turnover cycle and causes osteoporosis. Which results in pathological fracture or dislocation. In addition, corticosteroids decrease osteoprotegerin (OPG) and increase the receptor activator of nuclear factor kB ligand (RANKL) expression, which stimulates the osteoclast production. Moreover, corticosteroid affect the stimulation of the osteoclast activity, which in turn elevates bone resorption [10].

Corticosteroids decrease the calcium absorption in the intestine and increase the calcium excretion in the kidneys. Furthermore, corticosteroids decrease the androgen and oestrogen production. Which in turn increases the bone resorption. (pdf2) Corticosteroids induce apoptosis of osteoblast and interfere with their function. Corticosteroids also induce apoptosis of osteocytes, which cause osteonecrosis. Furthermore, corticosteroid suppresses osteogenic factors production of osteoclasts, which decrease the bone formation [11]. The elevated fracture and dislocation risk depends on the dose and duration of the steroid's treatment. The higher risk is in the first 3 month, and then slowly decreases, although it doesn't return to normal. In addition, intermittent corticosteroid use exerts a cumulative effect on bones, but smaller than the continuous dose [12].

In this case, the patient already treated by corticosteroid for years before hip dislocation. Although the patient in this case was complained by cant walk normally from the beginning and worsening could be prevented by good collaboration between the orthopaedic surgeon and dermatologist on treat the steroid induce hip dislocation on psoriasis patient.

Conclusion

Hip dislocation in pediatric are uncommon and differ from elderly. Corticosteroid has a high risk of dislocation and fracture, furthermore treating physicians must be made aware of the possible side effect on the bone and joint. Understanding of steroid induced needs to be known more deeply both by the orthopaedic and by every paramedic. The first treatment for hip dislocations is maintain the pain and prepare for the reduction as soon as possible. Good and appropriate cooperation can prevent unwanted events and of course reduce the risk worsening of dislocations. Complications can be minimized by early mobilization with adequate nutrition and rehabilitation are needed to bring a good outcome for the patient.

Disclosures

Conflict of Interest

The Author Reports No Conflicts Of Interest In This Work.

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Author Contribution

All Authors Contribute In Concepting, Designing, Conduct The Study, Prepare The Manuscript And Agree For This Final Version Of Manuscript To Be Submitted To This Journal.

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