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Editorial

Beware of Long Covid-19 Orthopedic Complication: Osteonecrosis

Asep Santoso

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Several orthopaedic complications have been reported related to COVID-19. It includes symptoms related to muscles, bones, and joints.[1,2] One important complication in the musculoskeletal area is osteonecrosis/ avascular necrosis (AVN). Several studies have reported cases of osteonecrosis. It can occur in the hip, knee, or jaw. Sulewski et al. found ten cases of osteonecrosis in large joints adjacent to the epiphyses of long bones and the spine.[3] Angulo-Ardoy M. *et al* reported osteonecrosis of the knee related to COVID-19.[4]

It is known that hip joints are susceptible to various diseases.[5,6] Several authors have reported osteonecrosis of the femoral head after COVID-19 infection. Agarwala et al. reported three cases of avascular necrosis of the femoral head in patients with long covid.[7] The interesting findings are that patients complained of symptoms with evidence of avascular necrosis around 58 days after a COVID-19 diagnosis. It is much faster when compared to steroid exposure, which shows that it generally takes six months to a year to develop osteonecrosis. The recent preliminary data showed that the angiogenic pathogenesis of SARS Cov-2 and treatment with high-dose corticosteroids increased the risk of osteonecrosis in Covid-19 patients.[8,9] A recent meta-analysis showed that 40% of COVID-19 patients received corticosteroid treatment. Among them, some 32% of the cases had osteonecrosis. It is also reported that low corticosteroid exposure might also lead to avascular necrosis.[10] We seem to face "double-trouble" conditions.[8] A general recommendation for assessing and managing the risk of glucocorticoid-induced osteonecrosis in patients with COVID-19 has been developed.[10]

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Original Research Article

Direct Lateral Versus Posterior Approach in Patients Undergoing Hip Arthroplasty: Short Term Functional Outcome and Review of Recent 5-year Literature

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ABSTRACT

Introduction: There is still limited evidence to support either the direct lateral approach (DLA) or the posterior approach (PA) for hip arthroplasty. The aim of this study is to compare the short-term clinical outcomes of both approaches and discuss the findings from recent literature.

Methods: This is a cohort study of the hip arthroplasty registry at our institution from January 2019 until January 2020. Functional evaluation using the Harris Hip Score was carried out before surgery, three months after surgery, and six months after surgery. The primary clinical endpoint was determined as the intraindividual rise at the six-month evaluation versus its preoperative level. Statistical analysis with T-test and Mann Whitney Test.

Results: A total of 68 patients underwent the surgery, equally distributed between the LA and PA group (n=34 for each). Postoperatively, no significant difference in HHS score was found between the two groups at three months for the pain score ($p = 0.534$) and functional score ($p = 0.772$), as well as at six months for both the pain ($p = 0.995$) and functional score ($p = 0.790$). The change in total HHS score from preoperative assessment to postoperative 3rd and 6th months between both surgical approaches was also not significantly different ($p = 0.693$ and $p = 0.505$, respectively).

Conclusion: Hip arthroplasty that performed with posterior and lateral approaches resulted in similar intraoperative morbidity assessed through amount of blood loss and also similar clinical and functional outcome as assessed through length of stay and Harris Hip Score.

Keywords: Harris Hip Score, hip arthroplasty, direct lateral approach, posterior approach
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INTRODUCTION

Hip fracture is regarded as a "fragile" fracture in the elderly with osteoporosis or osteopaenia brought on by a little fall. A hip fracture patient is typically 77 years old. Hip fractures occurred in 1.7 million people globally in 1990; by 2050, that number is anticipated to increase to 6.3 million.[1]

Displaced intracapsular neck of femur fractures may be treated with joint replacement operations. There are two main joint replacement techniques for treating misplaced intracapsular neck of femur fractures: partial hip replacement and total hip replacement. Partial hip replacement is the preferable option because the large diameter hemiarthroplasty "head" portion lowers the risk of dislocation in the frail, low mobility group. Total hip replacement is preferable for those who are more active since it can result in a better functional outcome.[2]

The posterior approach (PA) and the direct lateral approach (DLA) are the two surgical techniques most frequently employed for the hip region. In terms of maintaining gait, the posterior approach is preferable because the superior gluteal nerve is protected more and the abductor muscles are not divided. A direct lateral approach, on the other hand, may cause a late Trendelenburg gait and a delay in the restoration of abductor strength. The advantage of DLA is the acetabulum's clear visibility, which promotes cup alignment and may lower dislocation rates. Additionally, because the surgical location is farther away, there is a reduced risk of sciatic nerve damage.[3]

The strategy adopted is frequently determined by surgeon preference, and current recommendations are based on a small body of evidence. Dislocation rates have historically been used to form recommendations. Due to technological advancements and a better awareness of patient priorities, outcomes like post-operative function and pain may be viewed as more important in the current situation. This study compares the functional results of elderly patients who underwent hip arthroplasty using direct, lateral, and posterior surgical methods.

SUBJECTS AND METHODS

A prospectively established cohort was used in this investigation, which was carried out between January 2019 and January 2020. The inclusion criteria were met by patients who were brought to our facility with a displaced intracapsular hip fracture who underwent arthroplasty. With the approval of their next of kin or legal guardian, patients who were unable to consent were allowed to participate in the study. Inability to provide permission and pathological fractures requiring a particular prosthesis or therapy were exclusion criteria. The surgeon's preference determined the surgical strategy.

Similar preoperative, intraoperative, and postoperative care were given to the patient. The patient's membership in the organization was disclosed to the physician performing the surgery. The medical staff caring for the patient during their hospital stay and those evaluating the results, however, were blinded to the surgical technique used.

Surgical Procedure

Direct Lateral Approach

A longitudinal incision is made 3–5 cm proximal and 5–8 cm distal to the tip of the greater trochanter with the patient in the lateral decubitus position. The fascia is split in line with the skin incision between the tensor fascia latae and the gluteus maximus, then retracted with a retractor. The gluteus medius splits at the midpoint between the muscle's most anterior and posterior ends. The gluteus minimus and the joint capsule are divided around the neck of the femur. External rotation and flexion of the hip and knee are performed to dislocate the femoral head. To safely perform a femoral neck osteotomy with an oscillating saw, Hohmann retractors are inserted around the femoral neck.[4]

Posterior Approach

In the lateral decubitus position, the skin incision begins 5 cm distal to the greater trochanter and proceeds proximally to the greater trochanter, based on the diaphysis of the femur. It then curves for 6 cm toward the posterior superior iliac spine.[4]

Overlying the gluteus maximus, the fascia latae is incised, splitting the muscle bluntly down to the short external rotators. The gluteus maximus is retracted with a Charnley retractor, and care must be taken for the sciatic nerve that runs posterior to the short external rotators. The tenotomy of the short external rotators and piriformis is then performed at their insertion onto the greater trochanter. After incising the posterior joint capsule, the femoral head was dislocated by internally rotating the hip, and then the femoral neck was osteotomized.[4]

Postoperative Management

A standardized physiotherapy program was planned for all patients until hospital discharge at the fifth postoperative day, including a walking training with partial weight-bearing started from first postoperative day. After hospital discharge, physiotherapy continued on an individual basis, and walking training was done using a walker. Forced internal and external rotation was prohibited, and hip flexion was limited to 90° during the first four weeks of postoperative management.

Primary Clinical Outcome

The Harris Hip Score (HHS) was used as the primary outcome. Data were collected for gender, age, the body region being operated on, the procedure performed, the length of stay, and the surgical approach. Patients were scheduled for follow-up in the outpatient clinic three months and six months after surgery. The primary clinical outcomes evaluated with HHS were function, pain, and range of motion (ROM). The main clinical endpoint was described as the intraindividual increase at the six-month evaluation compared with its preoperative measurement. The maximum score of HHS is 100 points, covering function (seven items, 0–47 points), pain (one item, 0–44 points), absence of deformity (one item, four points), and ROM (two items, five points). Function is subdivided into gait (33 points) and activities of daily living (14 points). A cumulative score of 90–100 indicates an excellent result, 80–90 indicates a good result, 70–80 indicates a fair result, and <70 indicates a poor result.

RESULTS

There were a total of 68 patients undergoing the surgery, equally distributed between the group using the Lateral Approach (LA) and Posterior Approach (PA) (n=34, each). The mean age of patients was 69.68 ± 14.66 years in the LA group and 68.59± 16.52 years in the PA group (p = 0.775), with an equal distribution of gender (p = 0.575). The most common cause of pathology was trauma in both groups. The lateral approach was most commonly used in pathology around the femoral neck (50%), followed by hemiarthroplasty (91.4%). However, the posterior approach was mostly used in pathology around the intertrochanteric area (35.3%) and in hemiarthroplasty procedures (70.6%). There was no significant difference regarding length of stay between the LA group (11.34 ± 6.40 days) and the PA group (10.26 ± 3.85 days) (p = 0.490). (Table 1).

Parameters	Lateral Approach Group	Posterior Approach Group	p-value
Age (years); mean ± SD	69.68 ± 14.66	68.59± 16.52	0.775
Sex			0.575
Male; n (%)	10 (29.4%)	7 (20.6%)	
Female; n (%)	24 (70.6%)	27 (79.4%)	
Cause			0.510
Trauma; n (%)	30 (88.2%)	27 (79.4%)	
Non-Trauma; n (%)	4 (11.8%)	7 (20.6%)	
Region Involved			0.000
Intertrochanteric; n (%)	14 (41.2%)	12 (35.3%)	
Femoral Neck; n (%)	17 (50.0%)	11 (32.4%)	
Other; n (%)	3 (8.8%)	11 (32.4%)	
Procedures			0.017
Total Hip Arthroplasty; n (%)	3 (8.8%)	10 (29.4%)	
Hemiarthroplasty; n (%)	31 (91.2%)	24 (70.6%)	
Blood Loss; mean ± SD	323.33 ± 187.91	425 ± 251.845	0.177
Length of Stay (LOS); mean ± SD	11.26 ± 5.79	10.21 ± 3.57	0.820

Table 1. Baseline Characteristic

HHS Score		Group		
Time	Domain	Lateral Approach	Posterior Approach	p-value
Pre-op	Pain	9.12 ± 2.88	8.82 ± 3.27	0.692
	Functional	20.97 ± 3.90	21.91 ± 3.07	0.196
	Total HHS	30.09 ± 4.65	30.74 ± 4.32	0.555
3rd month	Pain	31.18 ± 5.91	32.06 ± 6.41	0.526
	Functional	22.82 ± 6.09	22.18 ± 6.33	0.693
	Total HHS	54.09 ± 4.28	55.06 ± 3.00	0.325
6th month	Pain	39.18 ± 4.62	38.59 ± 5.11	0.722
	Functional	33.59 ± 6.64	33.41 ± 6.00	0.909
	Total HHS	72.76 ± 5.74	72.00 ± 5.21	0.781

Table 2. Distribution of Harris Hip Score (HHS)

Changes in HHS Score	Group		
	Lateral Approach	Posterior Approach	p-value
Changes in Pre- op to 3rd Month	24.00 ± 4.73	24.32 ± 3.76	0.756
Changes in Pre- op to 6th Month	42.68 ± 7.40	41.26 ± 7.29	0.431

Table 3. Change in Total Harris Hip Score (HHS)

Preoperatively, no significant difference in HHS score was found between the two groups in terms of pain score (p = 0.692) and functional score (p = 0.198). Postoperatively, there was expected improvement in the HHS score. However, no significant difference in HHS score was found between the two groups after three months for the pain score (p = 0.534) and functional score (p = 0.772), also after six months for both the functional score (p = 0.790) and pain (p = 0.995). The complete result of our study was displayed in Table 2. As a result, the change in total HHS score from preoperative assessment to postoperative 3rd and 6th months between both surgical approaches was also not significantly different (p = 0.693 and p = 0.505, respectively). (Table 3).

DISCUSSION

This study adds yet another factor to take into account when choosing a hip arthroplasty technique. After three and six months, there was no significant difference in the functional results between the two strategies. Similar morbidity, as measured by intraoperative blood loss and duration of stay, was also discovered. This is presumably due to the extensive body of material that has already been written about both surgical methods, which helped us identify their respective flaws. (Table 4)

The incidence of abductor insufficiency is the main difference between the posterior and lateral approaches. There is an increased incidence of abductor insufficiency following the use of the direct lateral approach in THA procedures. For the posterior approach, the reported incidence ranges from 0% to 16%, whereas the direct lateral approach has a range of 4% to 20%.[4] A randomized controlled trial study by Witzleb et al. (2009) showed slightly better functional outcomes in patients implanted via the posterior approach after three months.[3]

In the last five years, studies comparing both surgical approaches have concluded that four out of five functional outcomes were comparable. Kristensen et al. (2016), however, found that patients undergoing hip arthroplasty with PA had less pain and a better quality of life. The author assumed that this was related to the use of the posterior approach in the group with uncemented implants. Patients who are in good physical condition are more likely to use uncemented implants.[5–9]

As for intraoperative morbidity, a previous study showed that PA is superior to DLA regarding intraoperative blood loss.[9] However, there is conflicting evidence regarding the operation time. A randomized controlled trial study with 60 patients undergoing cementless total hip arthroplasty found a significantly shorter operating time using a direct lateral approach compared to a posterior approach (67 minutes vs 76 minutes, p < 0.001).[3]

No	Reference	Conclusion	Outcome					Follow up period	Adverse event
			Pain	Blood Loss	Outcome Score	Dislocation Rate	Time Operation		
1	Hongisto et al 2018	Hemiarthroplasty using a lateral approach predisposed to the need for ambulatory aids 1 year after hip fracture. The posterior approach, however, predisposed to hip dislocation.	No difference	N/A	N/A	PA 4 (3.4%); DLA 0 (0%)	N/A	1 year	N/A
2	Kristensen et al 2016	Hemiarthroplasty for hip fracture per- formed through a posterior approach rather than a direct lateral approach results in less pain, with better patient satisfaction and better quality of life. The risk of reoperation was similar with both approaches.	Mean 4 month PA 23; DLA 25 (p=0.01) 12 month PA 18; DLA 21 (p=0.001) 36 month PA 17; DLA 20 (p=0.02)	N/A	EQ 5D index Score 4 month PA 0.47; DLA 0.45 (p=0.2) 12 month PA 0.58; DLA 0.55 (p=0.01) 36 month PA 0.6 ; DLA 0.56 (p=0.08)	N/A	PA 67 min; DLA 76 min	3 years	Walking problem in DLA > PA No Walking Problem : 4 month PA 27% ; DLA 20% (p<0.001) 12 month PA 40% ; DLA 30% (p<0.001) 36 month PA 35% ; DLA 42% (p=0.009)
3	Mukka et al 2016	Patients treated with HA for FNF using either the DL or PL approaches had comparable functional outcome after 1 year. The PL approach had a tendency towards a higher reoperation rate.	N/A	PA 239 (± 186); DLA 254 (± 141)	1 Year Follow Up WOMAC PA 79 (± 24.0); DLA 79 (± 22) Harris Hip Score PA 72 (± 17); DLA 71 (± 18)	Single dislocation PA 1; DLA 2 Recurrent dislocation PA 6; DLA 1	PA 66 (± 18) min; DLA 90 (± 21) min	1 year	Number of hips with any complication PA 15 (18.1%); DLA 9 (8.8%) ; (p=0.08)
4	Petis 2018	Temporal gait parameters were similar following THA for all approaches. Differences in gait kinematics and kinetics exist; however, given the small absolute differences, the clinical importance of these changes remains undetermined.	N/A	N/A	Step Length (m) Preop PA 0.53 [0.35–0.67]; DLA 0.48 [0.39–0.53]; p=0.89 6 weeks PA 0.58 [0.51–0.69]; DLA 0.48 [0.31–0.58]; p=0.18 12 weeks PA 0.60 [0.53–0.71]; DLA 0.56 [0.43–0.64]; p=0.19 Stride Length (m) Preop PA 1.06 [0.83–1.29]; DLA 0.95 [0.76–1.05]; p=0.08 6 weeks PA 1.14 [0.99–1.36]; DLA 0.98 [0.66–1.11]; p=0.19 12 weeks PA 1.20 [1.11–1.37]; DLA 1.12 [0.91–1.29]; p=0.15 Gait Velocity (m/s) Preop PA 0.86 [0.53–1.06]; DLA 0.76 [0.60–0.95]; p=0.10 6 weeks PA 0.98 [0.75–1.15];DLA 0.82 [0.36–0.96]; p=0.36 12 weeks PA 1.09 [0.99–1.33];DLA 1.00 [0.84–1.14]; p=0.33	N/A	N/A	12 weeks	N/A
5	Apparajit et al 2017	The rate of early surgical complications and outcome measures after posterior and lateral approaches is not significantly different.	N/A	PA 144.75±17.68; DLA 148.38±15.03	Harris Hip Score 3 mo :PA 67.22±7.61; DLA 65.01±7.35; p=0.191 6 mo :PA 76.45±6.31; DLA 74.06±6.81; p=108 12 mo :PA 85.62±6.04; DLA 83.40±5.09; p=0.094	PA 2 (5%) ; DLA 0 (0%)	PA 48.43±5.38 min; DLA 47.50±7.59 min	1 year	infection PA 17.50%; DLA 12.5%

Table 1. Summary of literature search about current study on surgical approach of hip arthroplasty

A prospective comparative study comprising 80 patients with intracapsular neck femur fractures between 50 and 80 years of age treated with hemiarthroplasty found similar operation times between the posterior and direct lateral approaches (48.43±5.38 and 47.50±7.59 minutes, respectively). A longer operation time is known to correlate with the amount of blood loss. As for our study, the similarity of intraoperative morbidity between the two approaches can be attributed to the well-known pitfalls of the two approaches.

The direct lateral approach has a lower risk of prosthetic dislocation and a lower rate of the secondary procedure compared to the posterior approach. The incident of prosthetic dislocation was high with posterior approach (5%) as compared to lateral approach (0%) and the rate of secondary procedures was high with posterior approach (12.5%) as compared to lateral approach (7.5%).[9] This unfavorable outcome might be related to the sacrifice of external rotator muscles in the posterior approach. We attempt prevent this in our cohort by advocating safe hip position to the patients and their caregivers.

Our study has several limitations. The first is the limited number of samples. Despite that, the sample size is sufficient to draw conclusions with sufficient power. There was also no randomization, and the selection of the surgical approach was entirely attributed to surgeon preference. These limitations suggest the need for further studies with a larger number of samples and the use of a randomized control study.

CONCLUSION

Both posterior and lateral methods of hip arthroplasty provided comparable intraoperative morbidity as measured by blood loss, as well as equal clinical and functional outcomes as measured by hospital stay and the Harris Hip Score.

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Ethical Approval

The research protocol for Ethical Clearance from the Research Ethics Commission at the Faculty of Medicine, Udayana University/Sanglah Hospital Denpasar was submitted before the research was carried out. Subjects who met the study criteria were given an explanation of the purpose of the study and asked to fill out written informed consent. Researchers have also attached a secondary data collection permit in the form of a medical record at Sanglah Hospital Denpasar.

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Original Research Article

Novel Biomolecular Target for OA Therapy

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ABSTRACT

Osteoarthritis (OA) is a chronic degenerative joint disease that affects many people worldwide, in which the loss of articular cartilage is the main cause of the pathology. The anatomical changes of OA include cartilage degradation, inflammation of the synovium, subchondral bone changes, and osteophyte formation. Damage to or loss of the extracellular matrix (ECM) composed of collagen, proteoglycan, and water, serves as the pathologic process of OA. The current available treatment options only include symptomatic or pain relief and surgical procedures toward joint replacement for correcting the deformity as a severe complication of OA. These, however, do not provide an adequate strategy for slowing the progression of OA, not to mention completely ceasing or reversing the resultant joint damage. Consequently, several alternatives for OA management have been recently proposed, including therapies targeting several enzymes and substrates playing important roles in OA, namely proteases, aggracanases, matrix metalloproteinases, and sialic acid.

Keywords: Osteoarthritis, protease, aggrecanase, sialic acid
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INTRODUCTION: OSTEOARTHRITIS

Osteoarthritis (OA) is a chronic degenerative joint disease affecting many people globally. It may cause disability, joint stiffness, and even the loss of joint function, especially in the older population. Anatomical changes occurring in OA include cartilage degradation, synovial inflammation, subchondral bone changes, and finally osteophyte formation.[1]

Nowadays, the management of OA is only symptomatic and surgical, namely joint replacement.[2] There is no single medication that can completely cure or prevent OA from becoming a full-blown disease. Therefore, a novel therapeutic strategy has been developed to target the main cause of OA, which is the loss of cartilage. To date, a biomolecular approach in the treatment of OA can be a promising novelty in order to improve patient's quality of life.

PATHOPHYSIOLOGY OF OSTEOARTHRITIS

The underlying etiology of OA is not fully understood; however, several mechanical factors have been associated with its development and progression. These factors include injury and obesity, along with other risk factors like age, gender, or genetics. The main pathology of osteoarthritis is a degenerative process involving the cartilage breakdown and, finally, the dysfunction of joints.

Cartilage is tissue formed by chondrocytes and surrounded by an extracellular matrix (ECM). Water, collagen (especially type II collagen), and proteoglycan build up this ECM. Collagen contributes to the strength of the cartilage to sustain tensile, while the proteoglycan absorbs water to sustain compression. Damage to or loss of these two important components, collagen and proteoglycan, are the main pathologies in OA.[3] Several enzymes are also responsible for ECM degradation, including matrix metalloproteinase (MMP), aggrecanases, serine proteinase, and cysteine proteinase.[4,5]

Type II collagen is the most common collagen found in cartilage. This type II collagen forms a fibril-woven structure. As previously mentioned, collagen degradation and the activity of aggrecanases are the main features of OA. Aggrecan plays an important role in protecting collagen from degradation. Although damage to proteoglycan is reversible by nature, damage to collagen is irreversible and cartilage repair would be impossible if the collagen were damaged.[6,7]

Matrix metalloproteinases (MMP) are endopeptidases bound to zinc that contribute to growth, development, wound recovery, and various pathological conditions, including arthritis and cancer. Its main role is to degrade ECM.[8] It has been observed that several MMPs are secreted by osteoblastic cells, including MMP-2, MMP-3, MMP-8, MMP-9, MMP-13, and MT1-MMP, while MMP-9 is mainly expressed by osteoclasts. MMP has been proven to degrade osteoid and activate bone remodeling in experimental animals and humans. MMP-13 is considered essential for osteoclastogenesis and is primarily associated with the mineralization of bone matrix. It also plays an important role in the degradation of type I collagen in the bone matrix. Abnormal expression of several MMPs in osteoblastic cells is also observed in patients with estrogen deficiency, since estrogen decreases MMP-13 levels in osteoblastic cells, resulting in inhibition of bone resorption and a decline in the rate of bone turnover.[9-11] MMP-13 is mainly produced in human chondrocytes in normal circumstances, but it undergoes rapid endocytosis and degradation. It is mainly expressed in OA cartilage but not in normal cartilage. According to several previous studies, MMP-13 levels in synovial fluid are known to correlate with the severity of OA. High expression of MMP-13, which induces joint abnormalities in OA, was observed in studies using a transgenic animal model. Studies evaluating MMP-13 inhibitors observed that MMP-13 inhibition provided protection against human and bovine cartilage cultures and provided an in vivo chondro-protective effect.[12,13]

OA, being one of the most common forms of arthritis, is characterized by joint cartilage destruction. Collagen type II, along with various proteoglycans, including aggrecans, chondroitin sulfate, and hyaluronan, are the main constituents of the joint tissue. The triple-helical structure of collagen type II contributes to the tensile strength of joint cartilage. Meanwhile, MMP-13 has been observed to be a major collagenase responsible for joint cartilage degradation in OA. The initial alteration of OA in animal and human models is controlled by a significant up-regulation of MMP gene expression. This MMP gene expression is increased by proinflammatory cytokines produced by activated synoviocytes or chondrocytes, such as interleukin (IL)-1β and tumor necrosis factor (TNF)-α.[12,14]

Another important process contributing to the pathogenesis of cartilage destruction is mechanical stress. In numerous in vivo and in vitro experiments, MMP-13 seemed to be involved in the early stages of OA development. In a mouse model of mechanical stress-induced OA, Kamekura et al. found MMP-13 in the early stages of OA in vivo. MMP-13 expression rose rapidly in chondrocytes in vitro due to mechanical stress.[15] There are more than 20 proteinase enzymes that make up MMP, each of which is a product of a variety of different genes. Some MMPs are created in abundance by chondrocytes and synovial cells in inflamed or arthritic joints. There are five sub-groups of MMPs according to their substrate specificity: collagenase, stromelysin, gelatinase, and membrane-type MMP.[16]

Four membrane-type MMPs (membrane-type MMP/ MT-MMP) have been identified in cartilage tissue, namely MT1-MMP (MMP-14), MT2-MMP (MMP-15), MT3-MMP (MMP-16), and MT4-MMP (MMP-17). It has been shown that chondrocytes and synovial cells express "membrane-related members" of the MMP group (except MMP-14) at low levels. MT1-MMP is the most dominant in cartilage tissue and is regulated by various cytokines (TGF-beta1, IL-1beta, and TNF-alpha) and epidermal growth factors (EGF). Additionally, the MT1-MMP is also capable of initiating the activation of pro-MMP-2 and pro-MMP-13. These two enzymes are specifically believed to be involved in mediating type II collagen webbing structure breakdown in cartilage tissue.[17,18]

Various other proteinases are further classified as disintegrins and metalloproteases with the recurrent thrombospondin family (ADAM-TSx), in which they can digest the core aggrecan protein. They have been identified and named aggrecanase-1 and aggrecanase-2, respectively. Various cytokines (IL1-beta, IL-6, IL-17, and TNF-alpha) and EGF regulate these aggrecans with a mechanism that differs from MMP regulation. Aggrecanase-2 creates a different "abrasion site" than that produced by MMP (Asu341-Phe342) in aggrecan molecules. In addition, it was recently discovered that aggrecanase-1 can cleave aggrecan molecules at the site of action of MMP (Glu373-Ala374).[8]

Tissue inhibitors of metalloproteinase (TIMP)-1, TIMP-2, TIMP-3, and TIMP-4 can inhibit MMP. According to previous studies, once TIMP is activated, it can inhibit MMP, and the balance between MMP and TIMP is indispensable in maintaining joint cartilage homeostasis. TIMP-3 has also been shown to inhibit aggrecanase activity in vitro.[19]

The synthesis rate of MMP in cartilage with OA far exceeds the up-regulation of TIMP-1, -2, -3, and -4 gene expression. Even though the expression of TIMP-1 mRNA by chondrocytes in cartilage with OA is higher than that in normal cartilage, the chondrocytes in OA do not produce sufficient amounts of the TIMP isoform to inhibit the existing MMP levels. MMP will degrade both endogenous and the newly synthesized ECM molecules. Finally, this process will result in the total loss of cartilage integration.[12]

DAggrecan is one of the main constituents of articular cartilage ECM and is largely responsible for the high resistance to compression of this load-bearing tissue. Its high osmotic pressure from the negatively charged GAG2 chains gives rise to the resistance to compression of the cartilage. The formation of aggregates is physiologically critical for the retention of aggrecan in collagen tissue.In numerous pathological conditions, including OA, the degradation of ECM macromolecules outperforms their synthesis, resulting in a decreased net cartilage matrix.[20]

Cartilage aggrecan degradation was initially assumed to only involve MMPs. But over time, other enzymatic activities have been proposed to be responsible for the response to the breakdown of aggrecan core molecules and proteins in the peptide bonds Glu373-Ala374 in the interglobular domain of these molecules. Aggrecanase is the term to describe this enzymatic activity. Many researchers throughout the 1990s debated whether aggrecanase belonged to the MMP group. The debate ended in 1999 when the ADAMTS group, a disintegrin and metalloproteinase with thrombospondin, was first introduced. This protease was initially named aggrecanase-1, also known as ADAMTS4, and sometime later, aggrecanase-2, or ADAMTS5, which has a specificity like that previously identified.[20-22]

It is also suggested that ADAMTS5 is present in human chondrocytes and synovial cells. The increased expression of ADAMTS5 mRNA in chondrocytes and synovial cells is not influenced by stimulation of IL-1, TNFα, or TGFβ, unlike ADAMTS4, whose levels are influenced by other cytokines. However, several follow-up studies showed different regulation of ADAMTS5 mRNA expression in bovine chondrocytes, in which the enzyme responded to IL-1 stimulation, unlike ADAMTS4 mRNA expression.[20,22]

TIMP-3 is a potent biological inhibitor to inhibit ADAMTS5 activity with *K*i values only in the subnanomolar range. The interaction between aggrecan and the C-terminal domain of ADAMTS5 can modulate it well. TIMP 3 is the only TIMP that hinders ADAMTS5 activity, although there are still many other TIMPs.[22]

Aggrecan constitutes the main proteoglycan in cartilage, and therefore severe damage, which has been associated with its aggrecanase action, is an important manifestation of OA. Aggrecanase was identified as the proteinase responsible for cleaving the matrix proteoglycan and can increase in its level in several circumstances; this activity is a feature of cartilage degradation during inflammatory diseases such as OA. A rise in the cleavage of ADAMTS family proteins at the aggrecanase site has been shown in vitro. Of these, ADAMTS-4 and ADAMTS-5 are the most efficient aggressors and have generally been proposed as the most likely candidates for a role in the pathological mechanism of OA. Significant protection against ex vivo proteoglycan degradation and decreased OA severity is provided by ADAMTS-4 and ADAMTS-5 in animal models. ADAMTS-4 and ADAMTS-5 both play important roles in mediating aggrecan loss in normal cytokine-stimulated cartilage and the ongoing degradation seen in cartilage OA. Consequently, a potential therapeutic strategy for OA can be directed towards the inhibition of their proteinase activity.[11,20]

In cultured bovine and swine chondrocyte models or cartilage explants, ADAMTS4 was induced after stimulation with IL-1, TNF-α, oncostatin M or transforming growth factor (TGF), but not ADAMTS5. A recent study showed that although ADAMTS4 gene expression could be increased through treatment with IL-1, TNF- α or oncostatin M, there was little effect on ADAMTS5 in either human chondrocytes or cultured human cartilage explants. On the contrary, an additive effect of combination treatment with oncostatin M and either IL-1 or TNF- α in this system was present, and this led to induction of ADAMTS4 as well as some induction of ADAMTS5 gene expression. In the synovium or cartilage undergoing OA, the aggressiveness of the activity and the expression of ADAMTS4 and ADAMTS5 are present constitutively, without the requirement for catabolic stimulation. Previous studies showed that ADAMTS4 upregulation relies on TNF- α and IL-1 produced by synovial macrophages, whereas these cytokines do not alter ADAMTS5 levels.[22]

In bovine nucleus pulposus tissue, upregulation of aggrecanase activity, ADAMTS4 in particular, in an NFkB-dependent manner, is induced by TNF treatment, although the specificity of the small NFkB inhibitor molecule used in this study remains unproven. In human OA synovial fibroblasts, upregulation of ADAMTS4 was observed in treatment with IL-1 or TNF-a but not with phorbol ester, while ADAMTS5 was unaffected. In this model, NFkB can be specifically inhibited by adenoviral gene transfer from the endogenous inhibitor IkBa without affecting other signaling pathways or causing apoptosis. Whereas ADAMTS5 gene expression was not altered by IkBa gene transfer, NFkB regulation potentially inhibited ADAMTS4 induction by IL-1 or TNF-a. A loss of the IL-1 response to the luciferase gene reporter vector ADAMTS4 results from mutation of one of the three identified NFkB binding sites, indicating that two or more NFkB binding sites located in the region 5 'flanking of this gene highly affect the increased transcription of the IL-1 stimulated ADAMTS4 gene.[23] These studies strongly suggested that the upregulation of ADAMTS4 induced by IL-1 or TNF-a is NFkB dependent. However, the role of NFkB in regulating ADAMTS5 expression remains unproven.[24,25]

Sialic acid, a form of carboxylated sugar, has a distinct pattern and tissue specificity. It can occur in free form or as constituents of glycoproteins and many forms of saccharydes, including polysaccharides, oligosaccharides, lipopolysaccharides, and lipooligosaccharides. Besides, membrane anchors and keratan surfaces have also been shown to contain sialic acid.[26] Cell behavior, including its growth, migration, inflammation, and matrix production, highly depends on specific sialylation motifs that lead to different effects of glycoproteins. Glycosilation is an important process in modifying cell surfaces and ECM proteins. In articular cartilage, chondrocytes and ECM high in collagen and proteoglycan have a high content of glycosylated proteins, and there is a thick coating of carbohydrates on the cell surfaces. The glycosylation process is heavily involved in the modification of the cell surface and ECM.Arthritis has been shown to be associated with a change in glycoproteins containing certain chains of sialic acid. Recent studies showed that OA cartilage expresses sialylated transmembrane mucin receptors.[27,28]

The interaction between glycan-binding proteins (GBPs) and glycan-protein interactions are essential for physiological or pathological process regulation, including inflammation and arthritis. Two major groups of GBPs consist of lectins and glycosaminoglycan-binding proteins. Lectins function as identifiers for specific patterns of glycan molecules. Under pathological circumstances, the interaction between lectin and glycan controls the activation of inflammatory pathways. For instance, galectin-1, a part of beta-galactoside-binding lectin, has been observed to induce inflammatory responses in OA through enhancement of the secretion of effectors of the degeneration process, including NF-B. Certain modifications in lectin and glycan result in the development and progression of certain disorders. In degenerative joint diseases, the expression of the -2,3-sialylated glycoprotein PDPN receptor stimulates degenerative joint changes by specifically involving tissue development, repair, and inflammation processes.[27]

MATRIX METALLOPROTEINASE-13 AS POTENTIAL TARGET THERAPY

The early pathological phase of chondrocytes is highly affected by MMP-13 since it promotes the release of the Low-density lipoprotein Receptor–related Protein-1 (LRP1) protein in cartilage. LRP1 plays an important role in regulating the clearance of ADAMTS-5. Therefore, to promote LRP1, MMP-13 interacts with another protein through activation of the latent form of the MMP-13 protein. A potential therapeutic target for early OA is the pro-MMP-13 activator because the activation of the zymogenic form of MMP-13 occurs rather early in the progression of OA. There are many other transcription factors involved during various stages of OA, these include LEF1, NF-κB, ELF3, HIF2α, and Runx2, in which they either directly or indirectly impact MMP-13 transcription.[9]

The chondrocytes and synovial cells produce MMP as a latent pro-enzyme. Thus, the process of OA involves several activation pathways, in which therapeutic intervention can be addressed on to these activation events. Plasminogen activator synthesized by chondrocytes synthesize plasminogen activator (plasminogen activator/PA) produce plasmin. It is known that the PA form found in urine (uPA) and tissue (tPA) are both produced by chondrocytes. The uPA form may be more important in the process of cartilage damage since plasmin, identified as a generalized MMP activator, is able to convert pro-MMP-13 into the active form of MMP-13. Pro-collagenase is then activated by this active form of MMP-13. The plasmin/PA pathway is regulated by a plasminogen activator inhibitor (PAI). The plasmin concentration is significantly higher than normal and the observed level of activated MMP is also higher in joint cartilage with OA. Another activation pathway has also been found in membrane-type MMP (MT1-MMP; MMP-14), activating MMP-2 (gelatinase A) and collagenase-3. Samples taken from patients with early-stage OA have also shown significantly increased MMP activity. Hence, therapeutic interventions can be directed and designed to inhibit MMP activity, and they will specifically be useful for managing OA.[11,19]

AGGRECANASE: ANOTHER POTENTIAL TARGET THERAPY

According to the most recent data presented in this review, while ADAMTS4 and ADAMTS5 were similar, they were two very different enzymes for their regulation. At least in human cells, ADAMTS4 responds to IL-1 and TNF-a, while ADAMTS5 does not. Another difference is that ADAMTS4 upregulation depends on the transcription factor NFkB, whereas ADAMTS5 is NFkB independent and has no kB element in its promoter. With this in mind, it is interesting to note that treatment to prevent IL-1-induced aggrecan depletion can be achieved through the use of the small molecule IkB kinase inhibitor in bovine cartilage explants, suggesting this process occurs in an NFkB-dependent manner. This dissimilarity in the regulation of ADAMTS4 and ADAMTS5 has implications for the potential development of disease-modifying osteo-arthritis medications. A therapeutic strategy inhibiting cytokine-induced inflammatory responses would tend to downregulate ADAMTS4, as would NFkB inhibitors. However, no strategy is likely to affect ADAMTS.[29]

The pharmaceutical industry has set its sights on the design of small-molecule aggrecanase inhibitors. For such an approach to work, there is a need to appreciate that ADAMTS4 and ADAMTS5 are configured differently. Further identification of the primary aggrecanases (ADAMTS4 or ADAMTS5) involved in human OA is therefore necessary.[24,30]

TARGETING SIALIC ACID IN OA PATHOGENESIS

Lectin, a type of sialic acid, has been utilized in differentiating malignant from benign tumors, and its use as a therapy for cancer by inhibiting cell proliferation has been widely proposed. Targeting sialic acid as therapy for OA involves the regulation of signaling cascade events aiming to protect cartilage from catabolic effects inducing ECM degradation. Lectin has potential effects on cartilage structure and primary chondrocytes in which it preserves cartilage structure and function by interfering with the transmembrane receptors under multiple factors inducing arthritis. Regulation of signaling cascades would prevent catabolic effects inducing ECM degradation on the cartilage, so invasive therapeutic methods, namely surgery can be prevented. Sialic acid occurs in free form or as a constituent of glycoproteins and saccharides, important components of a cell, playing a role in its growth, migration, and inflammation. ECM and chondrocytes contain high levels of glycosylated proteins, and glycosylation is important in cell surface modification and ECM production. Since arthritis involves a change in glycoproteins with sialic acid resulting in degenerative joint changes, a specific therapy targeting sialic acid focuses on controlling the interaction and modification between glycan and lectin, a form of sialic acid, resulting in the inhibition of inflammation and degenerative changes.[26,27]

SUMMARY

OA is a complex degenerative joint disease in which the underlying pathologic process requires further understanding so that therapy can be aimed at preventing OA from happening, slowing the pathologic process, and preventing its complications rather than correcting the debilitating resultant deformity. Treatment strategies targeting proteinase, aggrecanase, and sialic acid offer a promising future to prevent further degradation in OA; however, further research on this topic is highly necessary as a whole new alternative to symptomatic therapy and joint replacement surgery commonly performed in OA.

Conflict of Interest

The author declares that there is no conflict of interest related to the material discussed in this manuscript.

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Original Research Article

Psychological Problems in Parents of Children with Orthopedic Pediatric Congenital Disorders

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ABSTRACT

Background: Parenting a child born with a congenital disorder can be very unsettling and stressful for a long period of time. It takes patience, strength, and fortitude as well as a high sense of attention. Parents of children with congenital disorders are expected to collaborate along with healthcare professional and expect to receive support in return. Other than the medical team, parents are hopeful to find acceptance and comfort from the community around them. However, information regarding congenital disorders in society may not be as easily understood which limits their responses.

Main Discussion: This review explores the psychological issues faced by parents of children with congenital disorders of the musculoskeletal system. It is only natural for parents to experience emerging feelings of distress after knowing the unexpected truth. The additional care required for the child's medical and social aspects adds to the parent's personal and emotional baggage. Either by stigmatizing or providing support, society's response to the child with congenital disorders is pivotal to determine the mental health of these parents. Eventually, the coping mechanism opted for by parents might affect their decision making process and eventually, the quality of care they provide for their child.

Conclusion: By recognizing the potential roots of distress one family might endure, healthcare professionals are expected to provide holistic and comprehensive services for parents of children with congenital disorders, including their psychological state.

Keywords: psychology, parents, congenital disorders, orthopedics
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INTRODUCTION

One of the basic desires as a human being is to be accepted by society and interact with other individuals. In the past, individuals born with congenital disorders were segregated as their differences were stigmatized by society. The ancient Greeks perceived having a congenital disorder as a form of punishment from God or as a retribution for evil behavior in a past lifetime. The ancient Romans apparently held a similar viewpoint, as they were apparently instructed to murder babies born with congenital disorders.[1]

Children with congenital disorders were seen as embodiments of the devil and were linked to parents who practiced black magic throughout the Middle Ages.[2] Around the same time, Martin Luther also considered devil reincarnation in the form of a congenital disorder and recommended killing the child.[1] As we entered the 19th century, a shift of attitude toward congenital disorders by ruling out these superstitious explanations and associated these congenital differences with environmental factors from study results instead.[1]

Parenting a child born with congenital disorder can be frustrating and a continuous source of stress. In addition to the common duties of caring for young children, these parents are also faced with different expectations from the birth of a healthy and normal child.

PARENT'S INITIAL REACTION

According to Drotar *et al*. in 1975, parents would react in 5 different stages when they welcome a child with congenital disorder which include shock, denial, sadness, anger, adaptation and reorganization (Figure 1).[3,13] The first state of shock is often accompanied by feelings of guilt, sadness, and hopelessness.[8] Parents of a child with congenital disorder diagnosis may feel waves of emotions comparable to those with suicidal thoughts, and the extent of distress felt by parents, particularly mothers, may even continue to depression.[12] Compared to mothers whose children have no such diagnosis, a congenital abnormality diagnosis serves as an extra emotional burden for parents. According to Sapkota *et al* (2017, p. 29) children raised by depressed mothers showed developmental disturbances compared to other children.[12]

Figure 1. Five stages of parenteral reaction

The sooner parents, especially mothers, recognizes their children could possibly have a congenital disorder, the better their coping mechanism will be, which is essential in determining family's perception and acceptance for the diagnosis.[11] A survey study by Skotko (2005, p. 71-2) reported a direct testimony from a mother who received printed medical information regarding their daughter's condition from a social worker and would much preferred to obtain the information straight from their doctor.[11] Thus, providing prompt counselling sessions for these parents is a critical intervention point.

Figure 2. Clubfoot Deformity

PSI-SF subscale	Typical Stress Percentile	High Stress Percentile	Clinically significant Percentiles
Parental Distress (PD) - Reflective of adjustment to parenthood	15-80	81-89	90-100 Referral to community support
Parent-Child Dysfunctional Interaction (P-CDI) - Reflective of bonding with child	15-80	81-84	90-100 Referral for professional intervention
Difficult Child (DC) - Reflective of self-regulatory processes (physical or temperamental problems) or child's cooperativeness	15-80	81-89	90-100 **<18 months old** Referral to pediatrician **≥ 2 years old** Referral for professional support
Total Stress	15-80	81-89	90-100

Table 1. PSI-SF guide and interpretation

IMPACT OF OBLIGATION IN DECISION MAKING

Inadequate explanation of the condition to parents may negatively impact a future child's care plans. Collaboration between parents and medical team is pivotal in coordinating follow-up care and is expected to be achieved through thorough education of the disorder.[14]

A study conducted by Beresford *et al* (2007, p.2) reported that most parents' main goal is to ensure their children can become self-reliant in their activities and for themselves to have a balanced life between being a caretaker and as an individual.[4]

Rosenberg *et al* described that the more visible the child's condition, the higher the level of stress their parents are enduring.[13] A similar result is expressed by Bawalsah (2016, p.16) where the severity of physical dysfunction in children is positively related to stress occurring in parents.[5] In contrast, a qualitative analysis by Cousino and Hazen (2013, p. 821-2) reported that parenting stress was not related to children's illness period or severity but rather associated with the responsibility for managing the disorders' treatment and their poorer ability to adjust psychologically.[6] Orthopedic congenital disorders, such as those in the hands and feet, have the possibility of increasing stress related to physical appearance disorders, making parents actively seek treatment and medical care.[12]

Regarding parents' medical decision-making for their children, Madrigal *et al.* (2012, p. 2876) concluded that parents tend to participate in a collaborative role with the medical team (semi-active or collaborative role).[7] Although parents do want to have a thorough understanding of and coverage for their child's congenital disorder care, they do not want to take over the roles of healthcare professionals but rather seek support when parents implement their acquired skills.[4] The Parenting Stress Index (PSI) and its shorter form, the PSI-SF, are available to evaluate stress level through questionnaire components reported by the parents themselves (Table 1). In parents with a higher Parenting Stress Index (PSI) score, however, medical decisions were taken over by the mothers instead (active role). Kim *et al* (2019, p. 7-8) rationalized this significant association of an active decision-making role with a higher stress level due to maternal guilt and an attempt to make amends to her own feelings.[13]

ASPECTS OF PARENTAL BURDEN

Medical and rehabilitation services are just the tip of the iceberg of financial strain that may impose on parents' funds. Depending on how parents seek treatment, "doctor shopping" or even seeking non-medical treatments such as those offered by faith healers adds to the extra costs associated with caring for children with congenital disorders.[12] Parents with small incomes or even those who do not have insurance protection

Figure 3. Congenital tibial hemimelia which affect child's mobility

certainly have a much higher stress level. Beresford *et al* (2007, p. 4) highlighted the importance of providing parents with funding services in order to safely maintain financial support for the special-needs child.[4] The family economy would also be weighed down by the additional assistance parents might need in providing long-term care for their children with congenital disorders. Other than basic medical and rehabilitation assistance, parents may need help in the developmental areas of a child, including social and emotional development, academic progression, and independent living skills.[5]

Parents may receive assistance in one area but not in others. Although parents tend to seek psychological and emotional support from friends or other family members, they will not necessarily feel comfortable asking for their time to take care of their children or other domestic duties in order to personally rest.[4,8] It is important for parents to identify the strengths and weaknesses of their support system and then develop novel approaches to bridge the gaps that exist. Reminding parents of patients that doing what is best for their child with a congenital disorder does not mean doing everything by themselves is important.

ADAPTATIONS AND COPING MECHANISMS

As there is no specific way to react when welcoming children with congenital disorders into a household, a combination of patience, strength, and a high sense of attention should be the basis of this personalized approach. As time goes by, parents will try to adapt and find new ways to interact as a family. By acknowledging the different reactions of family members, openness allows lingering cynical thoughts to be released. In response, active listening between family members is key to helping parents rearrange their emotions and allowing them to provide proper care to these special children with a clear head.[1,8]

Mothers of children with congenital disorders must make many changes in their lives to be able to meet the needs of attention and care for their children. Heiman (2021, p.648) reported confession by one of the mothers with special need child, the dynamic in parental relationship shifted as the mother of became sole primary carer of the child whilst the father ensures financial income to support the family." Frustration and exhaustion are commonly expressed by mothers with this parenting arrangement, as they feel limited in their ability to live their own lives.[8]

An important source of coping mechanisms for stress is social support. Social support is as important in reducing stress, boosting trauma resilience, and lessening hardship.[9] The presence of another individual could improve the self-confidence of the parents, especially when the attention came from those who are expected to give help, such as extended family members.[8] Farrell and Corrin in Mason *et al* (2001, p.55) suggested the benefits of social support groups that promoted extensive information regarding congenital disorder conditions, obtaining a sense of acceptance by society, and strengthening healthy coping mechanisms so parents would not feel lonely facing their child's congenital disorder diagnosis.[1,10]

SOCIAL STIGMA AND HEALTH CARE PROFESSIONAL ROLE

Society's stigma towards children with congenital disorders is one of the most influential factors in the

Figure 4. Preaxial polydactyly is frequently stigmatized due to appearance abnormality

psychological health of their parents.[1] Parents may be reluctant to admit the embarrassment they feel is unavoidable. However, the rejection and exclusion that their children had to endure hurt them the most, especially when they reach school age and social interaction becomes a child's way to make friends.[8]

Community preparedness in responding and accepting children with congenital abnormalities maybe insufficient to provide the already-stressed parents the reassurance they needed.[1] Benjamin in Mason *et al* (2001, p. 68) pointed that due to high level of burden in parents' mind may perceive obtained sympathy into a pitiful act.[15] Thus, it is important to provide parents of children with congenital disorders with the adequate psychological and emotional support they are seeking by assessing their stress level.

Other than relaying comprehensive medical information regarding their child's condition, healthcare professionals are responsible for ensuring the caregivers' physical and mental health. After all, the caretakers, which in most situations would be the parents, will be the ones looking after the child. By informing parents of suspected or found abnormalities within their child and the possible diagnosis as soon as possible and providing multidisciplinary support are ways a healthcare professional can do to reduce stigma. Combining a sense of empathy with knowledge and skills in providing sensitive care reminds us that humanity should live within the hearts of healthcare professionals too.[15]

CONCLUSION

By recognizing the potential social stigma a family might endure, healthcare professionals are expected to provide holistic and comprehensive services for parents of children with congenital disorders, including their psychological state. Early provision of information regarding the child's condition, even when it is still a suspicion, might prepare parents to develop better coping mechanisms. Communication skills are key when delivering the news, as it is a very sensitive subject. As trust is built between caretakers and healthcare professionals, it is hoped that children with congenital disorders may receive proper collaborative care for their condition.

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Original Research Article

A Comparative Review on ACL Reconstruction vs Internal Brace Augmentation

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ABSTRACT

Increasing knee injuries mainly of Anterior Crucial Ligament have led to the development of different surgical procedures for its treatment. ACL reconstructive surgery is the most frequently used surgery in the orthopaedic field. It is performed by either a bone-patellar tendon-bone (BPTB) or semitendinosus and gracilis tendon (STG) graft. Earlier the ACL injury was treated by reconstructing the ligament but recurrence of 2nd injury after surgery was reported. This led to the development of a suture to tie up the graft in its place which provides more knee stability and good functional outcomes. The functional outcome of the surgeries was evaluated by some outcome measures like IKDC, KOOS, Lysholm score, etc. The patients who underwent surgery were asked to perform some physical tests to evaluate the success rate of surgery. The results of these tests determined the motion, functional activity, and efficacy in sports. This review focuses on understanding the benefits of suture augmentation in combination with ACL reconstruction and also discusses the combination of these two modalities that has led to a revolutionary change in the future of ACL ligament surgery.

Keywords: ACLR, anterior cruciate ligament, grafts, knee injury
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INTRODUCTION

One of the most frequent knee injuries in teen athletes is the fracture of ACL ligament.[1,2] The most commonly damaged part of the knee is the anterior cruciate ligament (ACL), responsible for around 50 percent of all injuries to the knee ligament.[3] The injury is prevalent among athletes, especially females. Reasons attributed for gender-based observation include the difference in genders in neuro-muscular build and physique. The anatomical pattern of the pelvis and legs have a hormonal influence of Oestrogen. During athletic events, the numbers of accidents arise. Soccer is one of the games that have the greatest ACL injury incidence.[4,5] The individual's response to cope-up with the injury varies when allowed to heal without any interventions. The partial nature of the injury may heal without intervention. However, it takes more than 90 days, and the symptoms may persist in many individuals. Severe injuries are the potential candidates for surgical management. The meniscus, when damaged, often demands more attention than any type of injury.

With the incidence of tearing of the ACL and the constant need for enhanced reconstructive procedures, surgeons are continuously searching for future developments in surgical techniques. While some studies have shown strong results in ACL regeneration[6,7] utilizing allograft tissue, there is a high risk of surgical failure in younger athletes.[8,9] Besides, the risk of an additional surgical site often prevails with autograft. The identification and rectification of this complication will be a potential therapeutic approach to enhance the reconstructive procedure for the management of used in ACL injuries.[10]

STRUCTURE OF ACL

The ACL is the knee's main static stabilizer against tibia-to-femur anterior translation. The ACL is a circle-shaped ligament that derives from the medial portion of the lateral femoral condyle and extends posteriorly through the intercondylar notch. The attachment's anterior surface is nearly vertical, while the posterior part is convex. In the direction of the tibia, the ligament runs distally, anteriorly, and medially. The ligament's strands move slightly to the exterior throughout the duration of its existence. The ligament averages 38 mm in length and 11 mm in width on average.[11]

ACL RECONSTRUCTION

The most important surgical procedure performed in the orthopaedic field is the reconstruction of the Anterior Crucial Ligament (ACL). Once torn, the fan-shaped complex ACL lacks the ability to repair or regenerate by itself. With rising life expectancy and quality of life changes in developed nations, athletic standards and demand are increasing among older aged patients.[12,13] Injured athletes forced to compete the professional game event are typically recommended for reconstructive operation. The ideal choices of the graft may include BPTB & STG.[14] Many competitors suffering from injured ACLs fail to recover back to their degree of pre-injury activities successfully[15,16] and one of the biggest explanations for this may be that athletes may not recover to their complete potential.[17]

SUTURE AUGMENTATION

To speed up postoperative healing, SA has been employed to establish fast stabilization before the graft integration. With aims close to ACLR, this procedure has been utilized for posteromedial corner and medial collateral ligament reconstructions and repairs, Achilles tendon repairs,[18] posterior cruciate ligament avulsion fracture repairs, elbow ulnar collateral ligament repairs, and lateral ankle weakness reconstructions.

DRAWBACKS OF ACLR

While ACL reconstructions have a high progress rate, they also have a high failure rate which may contribute to chronic damage following the procedure. ACL replacement patients are unlikely to do as well as they did previous to the operation. Following treatment, the early results of ACLR showed gradual degradation. These effects were linked to comprehensive soft tissue deconstruction and cast immobilization, which resulted in a high rate of discomfort, rigidity, and dysfunction. Although ACL reconstruction improves anterior-posterior knee flexibility, there is a reduction in knee strength and work done by the muscles around the damaged knee post-operatively, indicating that donor site morbidity contributes to the changed knee kinematics found after an ACL injury, according to Kowalk et al. The number of research focused on examining gait and knee kinematics after ACL reconstruction indicates an increase in gait pattern relative to pre-surgery, but compensatory muscle usage mechanisms continue in the number of people, suggesting sub-optimal graft results.[19]

MEASURES USED FOR OUTCOMES OF ACL RECONSTRUCTION

Knee-specific success tests are widely used as an assessment during knee surgery, especially during anterior cruciate ligament reconstruction surgery.

A. *Anterior Posterior Knee Laxity*

On both knees, anterior-posterior laxity values were calculated by a certified physical therapist with the KT-1000 Knee Arthrometer.[20] Three manual limit measurements were carried out and averaged the displacement readings. The gap between the legs was measured and used for the study (surgical knee-contralateral intact knee).

B. *Knee Injury and Osteoarthritis Outcome Score*

For the analysis of patient-reported performance, the KOOS[21] is applied. The KOOS assesses 5 domains: quality of life linked to the knee (QOL), the role of sports and exercise, everyday living tasks, symptoms, and discomfort. On a scale varying from 0 to 100, the sub-scores were presented, with 100 showing a perfect knee.

C. *ACL Return to Sport After Injury (ACL-RSI) Scale*

This scale is used for the evaluation of the patient's ability to return to normal functional activities. it is an effective questionnaire that is comprised of 12 questions that include unique features, like management of risk and trust of the patient, and is related to the preparation of an athlete to get back to functional activity.[22] The rating of this scale varies from 0 to 100, reflecting the status of patients who can return to their sports after assessing their score. A score of 56 or less on the ACL RSI scale has accurately defined the status of older patients who, because of psychological reasons, can struggle to get back to their sport after their surgical procedure.

D. *International Knee Documentation Committee (IKDC)*

An IKDC questionnaire is a quantitative scale that assesses the overall functional activity of the patient by providing scores according to the question category. The questionnaire is meant to include three categories: complaints, involvement in activities, and knee activity. Problems such as pain, fatigue, swelling, and knee giving-away appear to be assessed by the subscale of symptoms.

E. Lysholm score

It is a scale that provides 100 points rating for the evaluation of a patient's knee-specific problems, including mechanical locking, pain, discomfort, inflammation, stair climbing, knee instability, and squatting, which is the Lysholm score. Currently, the Lysholm Scale includes eight elements that are scored as given below:

ELEMENTS	SCORE
Pain	25
Instability	25
Locking	15
Swelling	10
Limping	5
Ascending stairs	10
Squatting	5
Need for support	5

On an increasing scale, any query answer has been given an arbitrary ranking. The number of each answer to the eight questions is the overall score, which can vary from 0-100. Higher scores reflect a stronger performance and fewer signs or disorders.

ACLR SURGICAL TECHNIQUE USING SUTURE TAPE[23]

Graft Preparation

An anterior dissection is used to extract a normal bone patellar tendon-bone autograft using 20- to 25-mm bone plugs for autografts. Achilles' allograft with the bone block is another choice for allograft. After that, a 2-mm hole is drilled into the superior plug to scale and ready the graft (and inferior bone plugs for an autograft). Suture tape is then wrapped across the distal end of the femoral bone block and threaded through the graft with a loose needle to the intended anterior side.

Graft Passage

A normal femoral tunnel is created across the anterior medial portal, and the tibia is drilled anterogradely. The graft is then threaded into the tibial tube and fixed in the femur with an intrusion pin. The anterior medial portal's suture tape augmentation tails (initially labeled) are then recovered. After that, the graft is cycled, and the isometric point is verified. For the posterior drawer, the leg is almost completely extended.

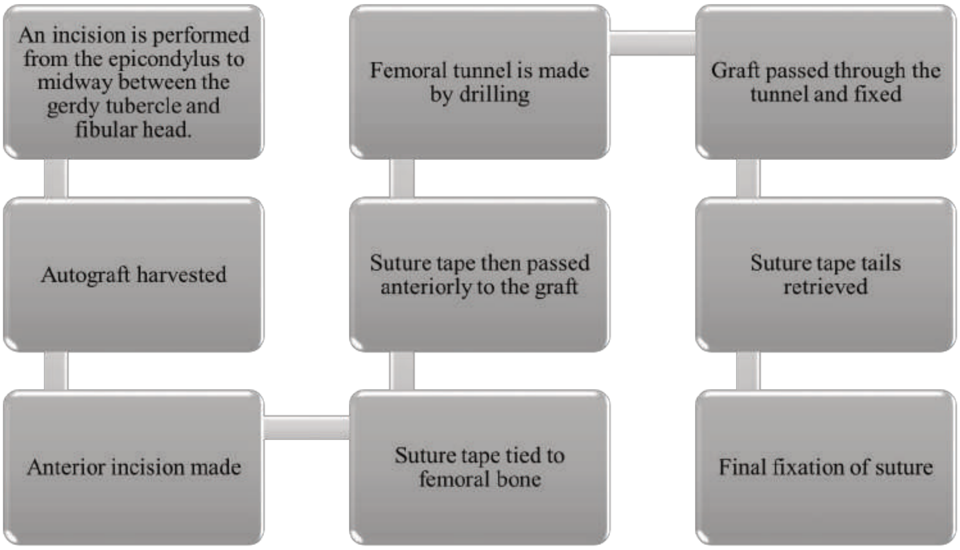

Figure 1. Depicting the surgical procedure of ACLR with SA

Suture Tape Augmentation Fixation

Crucially, during the graft, the FiberTape internal brace is clamped individually. After the allograft ACL has been placed on the tibia and fibula, the focus is shifted to the internal brace's final fixing. At this point, the knee can be tested to confirm that it has a complete scope of movement.

After the patient has shown a possibly optimally functioning quadriceps muscle and strong leg coordination, the range of movement is established using a CPM simulator, and weight-bearing is advanced as acceptable. Closed-chain strengthening is stressed, and patients are normally permitted to return to sports 6 to 9 months following surgery.

Functional measures	ACLR with SA	Standard ACLR	P value
Preoperative			
KOOS	48.44±13.85	49.78±12.04	.712
Pain	47.08±17.95	50.56±15.82	.429
IKDC	30.68±13.78	34.37±13.82	.385
ADL	62.74±17.30	67.58±12.15	.253
Post-operative			
KOOS	92.19±8.89	87.13±10.54	.068
Pain	94.74±9.54	89.63±8.25	.053
IKDC	87.55±14.05	73.24±20.09	.006
ADL	98.07±4.76	94.66±8.05	.073

Table 1. Comparative pre- and post-patient related outcomes

BENEFIT OF ACLR OVER SA

Due to the additional mechanical intensity, it may offer in the initial recovery and healing period, SA is presently being employed to assist ACLR. The internal brace has the added benefit of strengthening the overall build, which protects the graft during the remodeling and revascularization phase.

Strong associations among SA and better periods of recovering from preinjury activity level and percentage of preinjury activity level were found by Bodendorf et al. with a tendency toward an enhanced frequency of returning to preinjury activity level.[24] In table 1, the comparative results pre- and post-surgery are addressed.

The findings revealed that there were no substantial difference in –pre-operative scores among the SA and normal ACLR categories. SA had slightly higher IKDC and KOOS ratings after surgery. SA had higher comparative KOOS, ADL, and pain sub ratings, but this disparity still trended toward relevance. This showed that participants in the SA community returned to pre-injury activity levels much faster than those in the traditional ACLR group.

Biomechanical experiments utilizing SA to test ACLR have shown positive results. Cook et al. used a canine model to evaluate their theory.[25] Six months after treatment, the findings of a quadriceps tendon allograft with SA showed no major variations in force at fixed displacement sites or rigidity relative to the original

ACL. In this study, the SA showed consistent healing and no signs of osteophyte, cartilage, or meniscal abnormalities.

As opposed to graft alone, a biomechanical analysis conducted by Bachmaier et al. of bovine ACLRs supplemented by suture tape showed dramatically reduced graft dynamic elongation during load applied and enhanced failure load.[26] This impact was observed to be particularly powerful with grafts of limited diameter. This research also discovered that the suture tape's load-sharing role would not take control until the graft had significantly elongated, implying that the suture augment will not protect the graft from loads of low tension. These findings indicate that the SA would offer improved dynamic stabilization, particularly soon in the healing phase of the fragile graft, that may be beneficial to the recovered ACLR before the graft is secure.

On three pediatric patients, Smith et al. effectively implemented temporary usage of SA for ACL repair.[27] Short-term clinical progress has been shown by DiFelice et al. utilizing a SA construct to offer support for primary ACL repairs.[28] Interestingly, Peterson et al. observed no long-term substantial variations in return to operation or KOOS ratings between the augmented and nonaugmented ACL groups utilizing a common conceptual approach.

LIMITATIONS OF ACLR WITH SA

The primary disadvantage regarding the use of an internal brace would be the risk of over-constraining the joint and leading to loss of motion if the internal brace is too tight. For this reason, the internal brace is fixated separately from the graft and always at full hyperextension. Another concern would be potential stress shielding of the graft itself, but this also can be avoided by placing a hemostat tip underneath the FiberTape at the time of tibial fixation to build in a bit of slack with the internal brace. This ensures that the graft sees load, which is important in the tissue revascularization and remodeling process.

CONCLUSION

ACL tears can be distressing. However, the right surgical procedure can get patients walking again. In most cases, ACL reconstruction has long-term benefits. However, there may be some cases where ACL reconstruction along with suture augmentation will be successful, with shorter recovery. Both techniques have their advantages and disadvantages. Both have equal success and failure rates. The failure rate of ACL reconstruction earlier has led to the development of SA combined with ACLR which provides more patient compliance and better player performance.

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Case Report

Neglected Traumatic Posterior Hip Dislocation In a 4-Years-Old Child: A Case Report

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ABSTRACT

Introduction: Traumatic hip dislocation in pediatrics is relatively rare. Low-energy mechanisms of injuries such as trivial falls and slips are the usual causes, especially in the young age group (<5 years). Long-term complications may arise, especially if the hip dislocation is neglected. The reduction of a hip dislocation that has been neglected becomes more difficult over time.

Case Report: We present a 4-year-old male patient with a neglected traumatic posterior hip dislocation after falling from a parked motorcycle two weeks before admission. The posterior hip dislocation was diagnosed by physical examination and X-Ray. There was no disturbance in either the neurological or vascular status of the patient. The dislocation was reduced by closed reduction using the Allis maneuver.

Result: Post-reduction radiograph using X-ray showed a concentric reduction, and six weeks of immobilization using hip spica were initiated afterward. The patient showed normal gait and a painless range of hip movement without complications at two months follow-up

Keywords: neglected, traumatic, posterior hip dislocation, children
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INTRODUCTION

Traumatic posterior hip dislocation is infrequent in pediatrics. accounts for about less than 6%.[1,2,3] The causing trauma usually results from low-energy mechanisms, especially in children under five. This is mostly due to the laxity of their joints and their soft pliant cartilage compared to adults.[1,2,4] AVN of the head of the femur, sciatic nerve injury, degenerative arthritis, and recurrent dislocation are among the complications of posterior hip dislocation and may be associated with delayed reduction. Neglected in musculoskeletal injuries can be defined as injuries that are presenting late, lack or without comprehensive treatment and the time frame varies between body regions.[5] Neglected hip dislocation is still vaguely defined in the literature. A study by Garret et al. (1979) stated that a dislocated hip that was not reduced in 72 hours was called old unreduced hip dislocation.[6,7] Also, closed reduction of a dislocated hip that has been neglected becomes less possible over time due to fibrous tissue formation after the trauma.[8] Therefore, early diagnosis and prompt reduction are of utmost importance.[1,2,9,10,11]

PRESENTATION OF CASE

A 4-year-old boy was brought by his caregiver to our hospital with pain felt in his left thigh. He fell from a parked motorcycle two weeks before admission and had difficulty extending his left leg after the incident. The left hip was internally rotated, adducted, and slightly flexed with tenderness over the joint on examination. The left leg also appears shorter than the opposite leg by approximately 2 cm (Figure 1A). There were no distal neurovascular disturbances. The hip range of movement was limited on both active and passive examinations due to pain. Radiological evaluation using hip X-ray showed a displacement of the femoral head from the acetabulum to a superior and lateral direction without concomitant fracture (Figure 1B). Closed reduction of the hip using the Allis maneuver was then performed under general anesthesia. Post-reduction radiograph using another X-ray showed an anatomical reduction of the dislocated hip (Figure 3). We then immobilized the reduced dislocated hip with a hip spica for six weeks before weight-bearing (Figure 4). The patient showed normal gait, no pain, and a full range of hip movement at the follow-up two months later (Figure 5). We also found no signs of early AVN on the follow-up hip X-Ray. We advised the patient and his caregiver to remain on follow-up.

DISCUSSION

Traumatic hip dislocation is infrequent in pediatrics.[1,2,3] Trivial fall or slip (a low-energy mechanism of injury) is the usual cause, especially in the young age group (<5 years). In older children (>5 years), hip dislocation is more often caused by an athletic injury or a motor

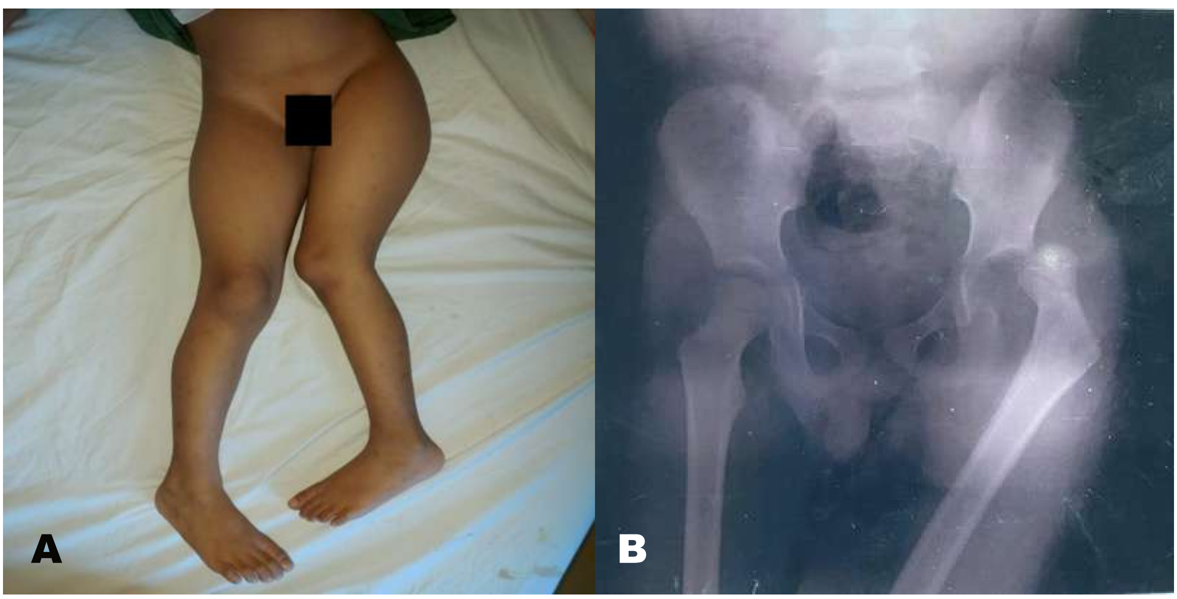

Figure 1. The patient's left hip before reduction (A). A posterior hip dislocation without associated fracture was seen on the AP hip radiograph (B)

vehicle accident (high energy mechanism of injury).[1,4,9,10,12] Hip dislocation in children can be classified into posterior, anterior, and central. Posterior dislocations constitute 75–90% of hip dislocations in children.[1]

A force that directs the femur posteriorly causes the typical posterior dislocation. Flexion, adduction, and medial rotation of the involved thigh are characteristics of posterior hip dislocation. The dislocated hip makes the leg on that same side appear shorter than the opposite leg. The examiner may palpate the femoral head posteriorly. The leg does not rest in a specific position in a central hip dislocation, and there is no leg length discrepancy. In an anterior hip dislocation, the dislocated limb may appear in a flexed, abducted, or externally rotated position. The examiner can palpate the femur head in the region of the obturator foramen, and the dislocated limb may appear longer than the contralateral side.[1] The examiner should also look for possible sciatic nerve and femoral nerve injuries that may occur in posterior and anterior hip dislocations, respectively.[1,2,10]

An AP view of a pelvic radiograph should be obtained to exclude associated injuries. In addition, more advanced imaging like MRI and CT can further exclude associated fractures and injuries that are not visible on X-ray. The radiographic finding of posterior hip dislocation is the typical displacement of the femoral head from the acetabulum in a superior and lateral direction.[1,2,10] Our patient is under the age of five, and the causative trauma mechanism was trivial. The characteristics of posterior hip dislocation were present, including a flexed, adducted, and medially rotated thigh. The AP pelvic radiograph showed displacement of the femoral head in a superior and lateral direction. Therefore, our findings were in accordance with the literature.

In our case, we consider the patient's condition to be an untreated or neglected hip dislocation because the injury occurred late and was not treated.[5] The time frame was in concordance with the criteria by Garret et al. (1979), because of the prolonged dislocation to a reduction time interval (2 weeks).[6] To the best of our knowledge, the definition of "neglected hip dislocation" is still vaguely defined in the literature, or there is no clear agreement. Early reduction by the closed method under general anesthesia is sufficient in most hip dislocation cases, hence why we chose to do so in our

Figure 2. An anatomical reduction of the left hip joint was seen on the post-closed

Figure 3. The patient’s left leg was immobilized with a hip spica

case. If the closed reduction fails or there are concomitant fractures, an open reduction should be considered.[1] Another piece of literature suggests that three trials of closed reduction be tried before an attempt at open reduction.[12,13] The treatment of a hip dislocation that has been neglected becomes more difficult over time. This difficulty may be due to fibrous tissue formation in the acetabulum, making closed reduction less possible.[8] Fibrous tissue begins to develop as early as 3 weeks and later filled the capsule by 8-10 weeks in an experimental animal study, and similar findings from another study. Hence, it is suggested that a closed reduction can still be done on a dislocated hip in less than 3 weeks.[6,7,11,14,15,16]

Furthermore, children, especially the younger groups (under the age of 5 years), whose acetabulum is mostly soft, pliant cartilage and tends to have more laxity in their joint compared with adults, require minor trauma to dislocate the joint. Thus, it's mostly not associated with other injuries or soft tissue or osteochondral interposition that may have made the close reduction more difficult.[17] Therefore, despite being neglected, we were still able to do a closed reduction on the patient's dislocated hip. We hypothesized that our case findings were no different from or in concordance with the literature just mentioned. These findings include: 1) the mechanism of injury of our patient was relatively minor (he fell from a parked motorcycle); 2) the younger age profile (under the age of 5); and 3) supposedly unabundant fibrous tissue formation (less than 3 weeks).

We treated the patient with closed reduction under general anesthesia using Allis maneuver because it's relatively one of the easiest, the most common, and the most effective treatments based on literature and our own experience. In this method, the patient's hip and knee are flexed 90 degrees, slightly adducted, and medially rotated. The surgeon then gives anterior traction by holding the patient's knee with one or both of their hands while the assistant holds the patient's anterior superior iliac spine.[1,12] Post-reduction X-ray showed an anatomical reduction. For the follow-up, we used criteria developed by Garret et al. (1979) to assess the follow-up result as excellent, good, fair, or poor. A painless, non-limping hip with a full range of hip movements is considered an excellent result. A painless, non-limping range of hip movements of 75% is considered good. A non-disabling pain with a moderate limp and a 50% range of hip movement is considered a fair result. A poor result is defined as disabling pain and markedly limited hip movement.[6] Our patient was excellent at follow-up.

Figure 4. Xray of both hips at two months follow up

There is some literature with a case similar to ours regarding the dislocation to a reduction time and the chosen reduction method. Baidoo and Adegah (2017) reported similar findings in their study. Their patient was also successfully treated with closed reduction using Allis maneuver, with two weeks being also the dislocation to time interval. Their patient was then followed up by six months, with no limping and painless range of motion.[18] A study by Hung (2012) reported 22 patients with five patients having a similar dislocation time interval to ours (interval of 12 days to 17 days). The five patients were treated by closed reduction using the Allis maneuver. The results were excellent in two, good results in two, and fair results in one.[15] These findings further support the idea of still being able to reduce the dislocation within 3 weeks.

There have been no conclusions regarding rehabilitation or when weight-bearing will be allowed. According to some studies, six weeks of immobilization with a spica cast before the patient is allowed to bear weight is sufficient for healing the soft tissue.[1,2,10] Traumatic hip dislocations in children can lead to complications, especially if the reduction is delayed. AVN of the head of the femur, sciatic nerve injury, degenerative arthritis, and recurrent hip dislocation are among the complications. AVN of the head of the femur is the most common complication. Factors that may be associated with the development of AVN are severe trauma and a delay in reduction of 6 hours or more from the time of dislocation.[1] The risk of AVN can increase by 20 times for every 6 hours of delay in its reduction.[19] Serial follow-up radiographs should be checked for at least two years after the original dislocation/trauma because radiographic findings of AVN can develop up to two years later.[1] Our patient had no complications at his last follow-up and was advised to check for any potential complications regularly.

CONCLUSION

Traumatic hip dislocation is infrequent in pediatrics. The mechanism of injury is usually trivial. Early diagnosis and prompt reduction should be done early for good outcomes, thus reducing the risk of potential complications. Long-term follow-up and monitoring are necessary.

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