Hip Ankylosis Caused by Heterotopic Ossification: A Case Report

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ABSTRACT

We present the case of a 59-year-old patient with heterotopic ossification in the right hip. This condition developed as a result of right hemiplegia secondary to a traumatic brain injury sustained in a traffic accident. Subsequently, the patient suffered an intracapsular fracture of the left hip, which required joint replacement surgery. Due to the patient's neurological limitations and postoperative immobility, Brooker grade IV heterotopic ossification with ankylosis developed in the non-operated hip. Surgical resection of the ossification was necessary, and it was found to be highly vascularized and in close proximity to the pelvic neurovascular bundle. Postoperatively, the patient was treated with radiation therapy and non-steroidal anti-inflammatory drugs. At the one-year follow-up, significant improvements in range of motion and independence were observed, with no signs of recurrence.

Keywords: Heterotopic ossification; hip; hip replacement; Brooker.
Level of Evidence: IV

Anquilosis de cadera por osificación heterotópica: reporte de un caso

RESUMEN

Se presenta el caso de un paciente de 59 años con una osificación heterotópica en la cadera derecha. Este trastorno se desarrolló como resultado de una hemiplejia derecha secundaria a un traumatismo craneoencefálico sufrido en un accidente de tránsito. Posteriormente, el paciente sufrió una fractura intracapsular en la cadera izquierda que requirió una cirugía de reemplazo articular. Debido a sus limitaciones, derivadas de su condición neurológica y el reposo posoperatorio, se desarrolló una osificación heterotópica grado IV de Brooker, con anquilosis en la cadera no operada. Esta osificación requirió una resección quirúrgica, se detectó una notoria vascularización y proximidad al paquete neurovascular inguinal. Luego de la cirugía, el paciente recibió radioterapia y antinflamatorios no esteroides. Se observó una notable mejoría en los arcos de movilidad y en la independencia durante el seguimiento de un año, sin evidencia de recurrencias.

Palabras clave: Osificación heterotópica; cadera; reemplazo articular de cadera; clasificación de Brooker.
Nivel de Evidencia: IV

INTRODUCTION

Heterotopic ossification (HO) is defined as the formation of lamellar bone in non-skeletal tissues, such as muscles, tendons, or other soft tissues. Although its etiology remains unknown, it is frequently observed in bedridden patients, those with traumatic brain injury (TBI), spinal cord injuries, or those who have undergone orthopedic surgery, as well as individuals experiencing tissue hypoxia, inflammatory states, burns, or who have a genetic predisposition.[1] HO was first described in 1883 by Reidel, but it was not until 1918 that Dejerine and Ceillier noted a higher prevalence of HO among soldiers who had suffered spinal cord trauma during World War I.[2]

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The annual prevalence of HO is approximately 1.3% over a 10-year period, with the most frequently affected joints being the hip (89.4%), knee (15.7%), elbow (14.5%), and shoulder (8.5%). Typically, the condition correlates with the side of the body that experienced trauma or neurological sequelae: in 83% of cases, HO is ipsilateral; in 14.9%, it is bilateral; and in 2.12%, it is contralateral. [3]

The absence of specific signs and symptoms makes HO a diagnostic challenge, especially in its early stages. Early-stage HO may present with pain, fever, edema, erythema, and mild reduction in the range of motion. At this stage, it can be mistaken for soft tissue infections, cellulitis, thrombophlebitis, or osteomyelitis. Eventually, it can lead to severe restriction of motion and complete joint ankylosis. [2]

We present a clinical case of severe spontaneous HO of the hip, of atraumatic origin, secondary to immobility and hemiplegia due to TBI in a 59-year-old patient.

CLINICAL CASE

The patient is a 59-year-old man who sustained a severe TBI in a motorcycle accident on January 21, 2021. The accident required a craniotomy and drainage of an intraparenchymal hematoma in the left temporoparietal region. As a result of the accident, he developed right hemiplegia and mixed aphasia with a predominance of motor impairment. In March 2021, the patient experienced a seizure that caused a fall, resulting in an intracapsular fracture of the left hip. At another medical center, he underwent hip replacement surgery with a dual-mobility cup prosthesis.

He was later referred to the specialized hip team for evaluation. Radiographic follow-ups revealed a bony bridging formation of HO extending from the femoral neck to the iliac wing on the non-operated hip. This condition was classified as Brooker grade IV HO, attributed to immobility and hemiplegia, leading to ankylosis and a flexion contracture limiting the range of motion to 45°. This restriction made sitting, independent mobilization, and walking difficult.

A pelvic CT scan was requested to assist in treatment planning, and the patient was evaluated by the Physiatry and Anesthesiology teams (Figure 1).

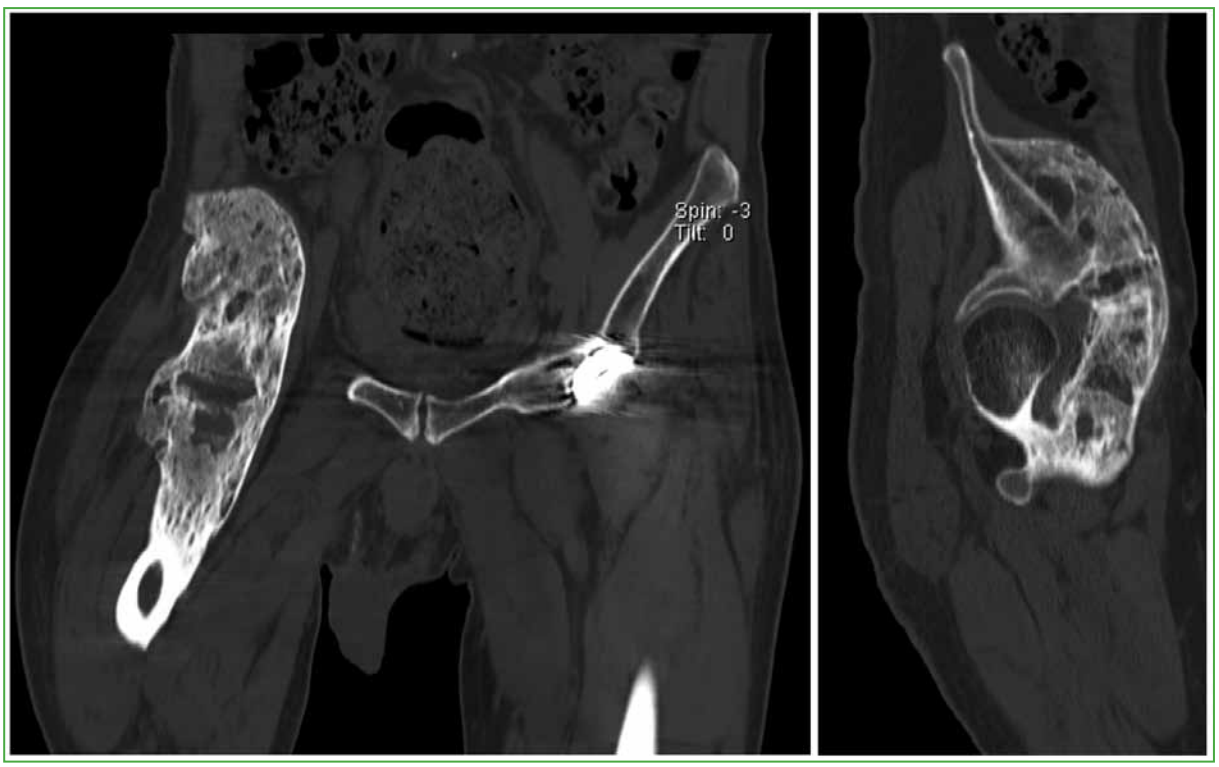

Figure 1. Computed tomography of the pelvis. Heterotopic ossification formation with bony bridging from the femoral neck to the iliac crest at the level of the non-operated hip. Brooker classification grade IV. [8]

The Physiatry team recommended starting physical therapy, along with the application of local antispasticity treatment to the hamstring muscles, in order to improve the gait pattern. While there was some improvement, the patient did not achieve independent walking. As a result, the decision was made to proceed with surgical resection of the HO, performed by the hip surgery and orthopedic oncology team.

During surgery, a Brooker grade IV HO was confirmed, with involvement from the acetabular rim to the lesser trochanter of the right hip, and significant vascularity was noted within the lesion. An iliofemoral approach to the right hip was made, during which the vessels and femoral nerve were identified and retracted medially. Proximal and distal dissection was performed to locate both foci of ossification, followed by a central osteotomy. The medial third and distal region were resected, and an additional osteotomy was performed at the level of the lesser trochanter. The hip capsule was exposed, and a proximal osteotomy was performed, removing the ossification en bloc. The range of motion was then reassessed, showing significant improvement, and hip stability was confirmed (Figure 2). The procedure concluded with the application of hydrogen peroxide, irrigation with 3000 cc of 0.9% saline, meticulous hemostasis, and the use of bone wax. Two Spongostan® sponges were left in place due to significant bleeding, necessitating observation in the Intensive Care Unit. The patient received a transfusion of 5 units of red blood cells, 3 units of fresh plasma, and an apheresis of platelets, in addition to vasopressor support.

A consultation with the radiotherapy team resulted in a recommendation for 3D conformal radiotherapy to the hip. Celecoxib was prescribed for 30 days.

Figure 2. A. Flexion contracture, before surgery, under general anesthesia. **B.** Recovery of extension in the immediate postoperative period.

Immediate post-operative radiographic follow-up and imaging three months later (Figures 3 and 4) confirmed complete resection of the ossification. Clinically, one year after surgery, the patient demonstrated marked improvement in the range of motion, achieving full extension and 110° of flexion. He resumed walking with the aid of a walker and continues physical therapy and quadriceps strengthening under the supervision of the Physiatry team, with no additional pain or complications to date.

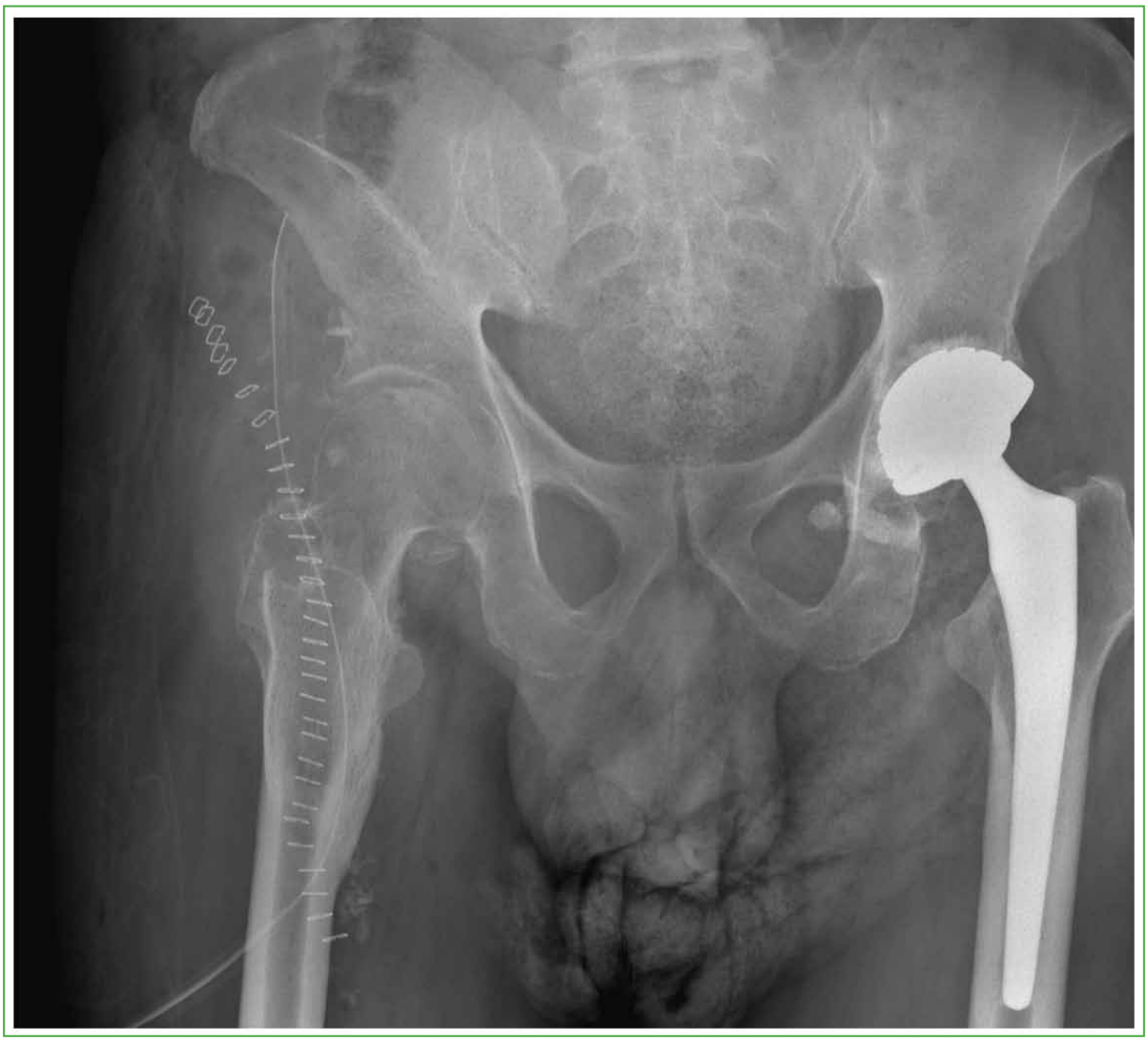

Figure 3. Anteroposterior follow-up radiograph of the pelvis in the immediate postoperative period. Wide bone resection and recovery of normal anatomy.

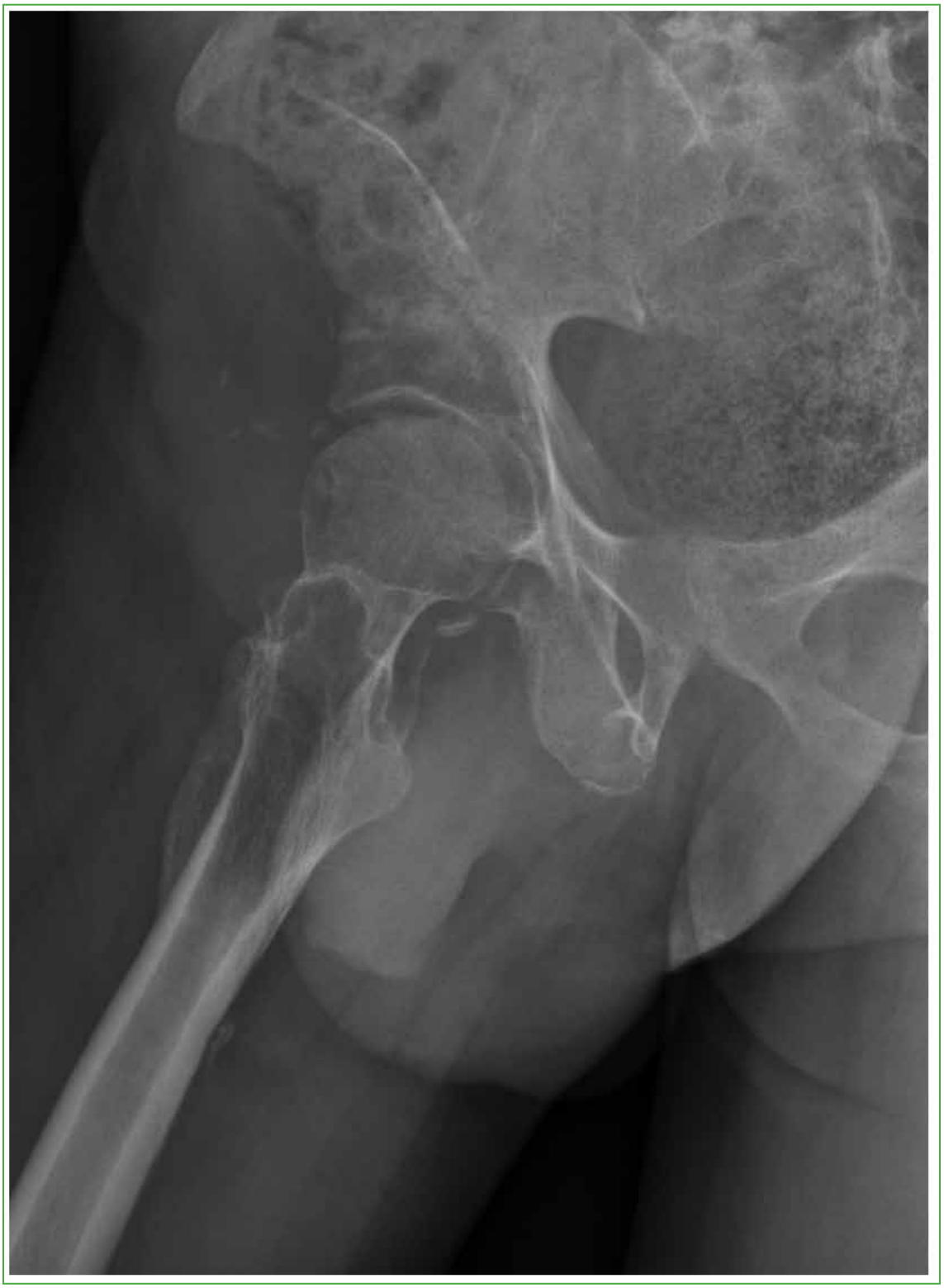

Figure 4. Follow-up radiograph three months after surgery. Resection and preserved anatomy.

DISCUSSION

Heterotopic ossification (HO), also known as hypertrophic osteoarthropathy or myositis ossificans, is a common condition characterized by the ectopic formation of bone tissue in soft tissues. It can be classified by both etiology and severity. In terms of etiology, it may be congenital or acquired. Among congenital conditions, fibrodysplasia ossificans progressiva and progressive osseous heteroplasia are specifically associated with extensive ectopic bone formation. However, causes of acquired origin are more prevalent, occurring in 44% of patients undergoing arthroscopy or hip replacement, 10-20% of those with central nervous system or spinal cord injuries, and 4% of those with burns affecting more than 30% of their body surface.[4-6] DeBaun et al. reported rates of up to 90% and 40% of HO in patients undergoing total hip arthroplasty and open reduction with internal fixation of the hip, respectively.[7] In terms of severity, the Brooker classification has historically been used to assess the level of ossification, with grade IV representing the most severe form, including bony ankylosis of the joint (Table).[8]

Table. Brooker's classification

Type I	Bone islands within soft tissues
Type II	Ossification originating from the pelvis or proximal femur, leaving at least 1 cm between opposing bone surfaces
Type III	Ossification originating from the pelvis or proximal femur, reducing the space between opposing bone surfaces to less than 1 cm
Type IV	Ossification that makes a complete bridge between the proximal femur and the pelvis (bone ankylosis).

The pathophysiological mechanisms underlying HO formation are not yet fully understood. The literature suggests a variety of possible precursor cells, including myosatellite cells, smooth-muscle cells, and even endothelial cells.[9] The presence of multipotent cells in local tissues has been identified as a trigger for this condition. HO formation requires an inducing agent, an osteogenic precursor, and a conducive environment for osteogenesis.[9] Bidner et al. propose that dysregulation of the immune system leads to an uncontrolled inflammatory response, which releases factors that promote HO.[10] Further research by Salisbury et al. identifies bone morphogenetic protein 2 as a proinflammatory agent that stimulates the release of substance P and calcitonin gene-related peptide from sensory nerves.[11] Other proposed contributors include prostaglandins, specifically prostaglandin E2, which mediates progenitor cell differentiation, as well as tissue hypoxia and imbalances between parathyroid hormone and calcitonin.[2] Kurer et al. conducted a study using blood samples from four paraplegic patients with HO and four without. The samples were incubated with human osteoblasts in tissue culture, and their metabolic activity was quantitatively measured. The results showed that patients with abnormal bone formation had significantly higher levels of factors stimulating osteoblastic activity, potentially contributing to HO pathogenesis.[12] A review by Cholok et al. highlights the involvement of multiple potential cell lineages and signaling pathways, underscoring the current lack of comprehensive understanding of HO formation.[13] In summary, the exact mechanisms underlying HO remain unclear and require further investigation.

Computed tomography (CT) optimizes preoperative planning by providing enhanced three-dimensional visualization of HO in relation to relevant anatomical landmarks. In certain cases, magnetic resonance imaging (MRI) may be necessary to define more precisely the extent of neurovascular or local soft tissue involvement. These imaging studies are most effective when HO is located near anatomical structures within the potential operative field.[14]

Multiple pharmacological and non-pharmacological interventions have been described for preventing HO. These include non-steroidal anti-inflammatory drugs (NSAIDs), both selective and non-selective, radiotherapy, physiotherapy, and combinations of these strategies.

Selective and Non-selective Non-steroidal Anti-inflammatory Drugs

NSAIDs prevent HO by inhibiting the osteogenic differentiation of progenitor cells. Prostaglandin E2 plays an important role in HO formation, fracture healing, and bone regeneration.[15,16] Numerous dosing regimens are used; indomethacin is the most common non-selective NSAID for HO prophylaxis in patients undergoing total hip arthroplasty. A dose of 75-100 mg/day is typically administered 24-48 hours before surgery and continued for 7-14 days.[17] A large meta-analysis, which included both randomized clinical trials and observational studies, found that both selective and non-selective NSAIDs reduced the risk of HO after total hip arthroplasty compared with placebo (odds ratio [OR] -1.35; 95% confidence interval [CI] -1.83 to -0.86, and OR -1.58; 95% CI -2.41 to -0.75, respectively).[18]

While NSAIDs are effective for HO prophylaxis, their impact on fracture healing should also be considered. HO prophylaxis with indomethacin increases the risk of pseudarthrosis in long bones and exposes patients who are also on anticoagulation therapy for other medical conditions to a higher risk of bleeding. Given these risks, NSAIDs should be administered with caution following orthopedic injuries due to the potential risk of pseudarthrosis. Consideration should be given to using NSAIDs alongside proton pump inhibitors to reduce the risk of gastroduodenal injury and subsequent gastrointestinal bleeding. The optimal timing, dosage, and duration of NSAID therapy for HO prophylaxis remain to be determined.

Radiotherapy

Radiation therapy has shown good results in the hip; however, the effectiveness of radiation prophylaxis in joints other than the hip has not been adequately studied. Radiation can be administered at a dose of 700-800 cGy in a single fraction, from 24 hours preoperatively to 48-72 hours postoperatively.[21] Both preoperative and postoperative radiation have been found to be equally effective in the hip, with no significant differences in complication rates.[22] Although no cases of malignancy have been reported following prophylactic radiation, it remains a theoretical risk. Additional potential side effects include progressive soft tissue contracture, delayed wound healing, pseudarthrosis, and inhibition of press-fit hip implant ingrowth. [23]

Physical Therapy

There are differing opinions on the value of physical therapy in the treatment of HO, as there is no clear evidence regarding the effect of joint motion on the progression of the condition. Some believe that excessive movement immediately after injury exacerbates HO, while others argue that the condition progresses due to insufficient movement. Although there is no consensus, physical therapy may be beneficial when worsening range of motion begins to limit daily function.[24]

In a meta-analysis comparing radiotherapy and NSAIDs for the prevention of HO after major hip surgeries, Pakos et al. found that radiotherapy tended to be more effective than NSAIDs in preventing Brooker grade III or IV HO (relative risk 0.42; 95% confidence interval [CI] 0.18-0.97) or any grade of HO (relative risk 0.75; 95% CI 0.37-1.71). However, there was significant heterogeneity between studies in the latter analysis. The overall absolute risk difference for Brooker grade III or IV was small (-1.18%; 95% CI -2.45% to 0.09%).[25]

CONCLUSIONS

HO is a significant concern in patients with the aforementioned risk factors. Despite the multiple theories surrounding its pathophysiology, the impact of HO can be severe in advanced stages, potentially leading to bony ankylosis that severely limits joint mobility, as seen in the presented patient with severe atraumatic HO secondary to rest and hemiplegia due to traumatic brain injury (TBI). Therefore, comprehensive medical management—including physiotherapy, NSAIDs, and radiotherapy—is essential to reduce the risk, progression, and recurrence of HO. Additionally, surgical intervention should be considered a crucial option, as it becomes the mainstay treatment to improve quality of life and mobility.

Conflict of interest: The authors declare no conflicts of interest.

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