Current Concepts in Neurogenic Heterotopic Ossification

JONATHAN LIU, MD; NOAH GILREATH, BA; SYDNEY ROZENFELD, MD; SANDI CAUS, MD; SARAH CRIDDLE, MD; EDWARD J. TESTA, MD; ANDREW EVANS, MD

ABSTRACT

Heterotopic ossification (HO) is abnormal bone deposition, most commonly in the hip or elbow, that can significantly impair patients due to pain, stiffness, and contractures, which prevents them from carrying out activities of daily living. A traumatic brain (TBI) or spinal cord injury may prompt the formation of heterotopic ossification, creating so-called neurogenic heterotopic ossification (NHO). The pathophysiology of this condition is not fully understood, but probably involves a complex interplay between a biological scaffold of demineralized bone, CNS signaling, and local tissue signal mediators that often result from trauma. This environment is conducive to HO formation. Diagnosis relies on laboratory testing, plain radiographs, and advanced imaging such as triple phase bone scan or computed tomography scan. Treatment involves medical management with anti-inflammatory drugs, bisphosphonates, radiation therapy, or surgical excision, although outcomes are variable both in regards to prevention and treatment. A mainstay of treatment is physical therapy to maintain range of motion. This paper also presents a case study of a poly-traumatized patient with a TBI and multi-level spinal column injury who developed NHO after acetabular fixation.

KEYWORDS: Heterotopic ossification; traumatic brain injury; bisphosphonates; indomethacin; radiation therapy; rehabilitation; physical therapy

INTRODUCTION

Neurogenic heterotopic ossification (NHO) occurs in the setting of neurological disorders and is characterized by abnormal bone deposition in extraskeletal tissue.[1] While NHO is usually seen after traumatic injury to the brain (TBI) or spinal cord (SCI), it is also associated with disorders such as Guillain-Barre syndrome, cerebral anoxia, stroke, infections, and brain tumors.[2] It has been reported to occur in up to 20% of TBI patients and 30% of SCI patients.[3-5] Additional risk factors for NHO may include male gender, polytrauma, delayed rehabilitation, and prolonged hospital length of stay.[6,7]

Patients with NHO may present with pain, reduced joint range of motion, warmth, and swelling. Typical sites of NHO include the hips, knees, elbows, shoulders, hands, and spine.[8–11] Due to its location and associated symptoms, NHO can cause significant impairment of activities of daily living (ADLs). While the exact mechanisms behind the development of NHO are not completely understood, the complex relationship of traumatic injury, localized and systemic inflammation, and neural regulation are all thought to contribute to its development.[12–14]

This article will review the pathophysiology, diagnosis, and treatment of NHO in the context of a male patient who developed NHO after surgical fixation of a complex left acetabular fracture following a polytraumatic motor vehicle collision.

PATHOPHYSIOLOGY

The pathophysiology of NHO is not entirely understood, but it is generally recognized as a complex interplay between traumatic injury, local and systemic inflammatory responses, and neuromodulation.[15,16] NHO affects approximately 20% of people with a spinal cord injury or TBI, so it is imperative to understand NHO and its associated morbidity. Generally, HO involves osteogenesis outside the appendicular or axial skeleton and instead within soft tissue (i.e., muscle).[16] It has been described as the formation of benign ectopic bone which undergoes osteogenesis through endochondral rather than intramembranous ossification.[17] Although ectopic bone formation can occur at any extraosseous site, the hip is the most common, followed by the elbow.[18-20] Specifically, the demineralized bone matrix that becomes embedded in muscle will undergo osteogenesis, which is contrary to that in other tissues (i.e., adipose).[15] Herein, we will further explore mediators of the local and systemic inflammatory responses as well as neuromodulatory responses that contribute to extraosseous bone formation.

Perhaps most critical for the development of HO is the biological scaffold that promotes bone formation, an environment that responds to an inducing agent (i.e., trauma) and contains osteogenic precursors.[16] Our understanding of the relationship between the central nervous system (CNS) and bone continues to evolve. Dense innervations of the periosteum provide a mechanistic route by which the CNS can modulate osteogenesis, through neurotransmitters including glutamate, calcitonin gene-related

protein, substance P, and catecholamines. Altogether, these transmitters upregulate osteoblastic activity while down regulating osteoclasts.[16]

Apart from the nervous system as a modulator for HO, local mediators are often further upregulated in the setting of TBI or SCI. Osteoprogenitor cells within skeletal muscle respond to the local environment, specifically to inflammatory mediators that create a hypoxic environment for osteogenesis.[17] Pro-inflammatory cytokines such as transforming growth factor-beta (TGF-β), interleukin-1 (IL-1), interleukin-6 (IL-6), insulin-like growth factor (ILGF), platelet-derived growth factor (PDGF) and fibroblast growth factor (FGF) contribute to the differentiation of osteoprogenitor cells.[1,3] Furthermore, the relatively hypoxic environment contributes to an influx of pro-inflammatory cells, including macrophages, neutrophils, and mast cells; this environment is pro-osteogenic. This leads to a cascade including upregulation of hypoxia inducible factor-1 and endothelial growth factor (FEGF), which stimulate angiogenesis and the migration of osteoprogenitor cells, which are stimulated by FEGF to differentiate into fibroblasts and chondrocytes. Ultimately, this process leads to an up-regulation of SOX-9 and the production of chondrocytes, which begin to form lamellar bone.[17] Overall, at the cellular level there are multiple contributors that lead to the up-regulation of chondrocytes and formation of heterotopic ossification.[15-17]

DIAGNOSIS

Early NHO can manifest as joint stiffness, decreased range of motion, erythema, swelling, and pain.[3,5] Without clinical suspicion, the diagnosis of early NHO can easily be missed. NHO usually occurs 3–12 weeks after the injury/trauma, but it can take more than six months to present in some cases.[21] Common differential diagnoses that should be ruled out include deep vein thrombosis (DVT), tumor, and septic arthritis.

Laboratory studies can provide cost-effective and important information in the workup of NHO, especially in its early inflammatory phase. Non-specific inflammatory markers such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are often elevated in the inflammatory phase of NHO, though it is important to consider and rule out mimicking inflammatory or infectious pathologies.[22] Alkaline phosphatase and osteocalcin are also associated with NHO, though in a non-specific manner.[23] These markers increase in the first six to 12 weeks after trauma, with serum levels greater than 250 (IU/L) associated with early HO formation.[5,24-26] Additionally, increasing serum creatine kinase may be correlated with more severe disease and resistance to medical therapies, which may help further guide diagnosis and treatment.[3,27]

A venous duplex ultrasound can be ordered to quickly and easily rule out a DVT. Radiographs are specific but not sensitive in the early phase of NHO. Later on, it can be seen as circumferential bone formation at or around a joint. While triple-phase bone scan is the most sensitive test for NHO, as early as 2.5 weeks after injury, it has low specificity.[3] Computed tomography (CT) scans can determine the extent and three-dimensional structure of NHO, which is helpful to prepare for operative intervention but not as useful in diagnosis.[16,28] Magnetic resonance imaging (MRI) reliably detects NHO in a three-dimensional fashion, as early as one to two days after the onset of symptoms, but the specificity is low.[29] Other imaging techniques used are ultrasound and 3-dimensional stereolithography, but they are less popular. Early diagnosis is important because it allows for initiation of interventions that may halt its progression.

TREATMENT

Treatment varies based on symptom severity and patient-specific risk factors for developing NHO. Studies vary in the reported incidence of asymptomatic NHO; patients without clinical symptoms from early-grade cases may be monitored closely.[30] Radiographic severity may not correlate directly with loss of function or range of motion, though high-grade cases are more likely to cause debilitating symptoms.[31] There is a wide range of treatment options, prophylactic and definitive, varying from NSAIDS and other oral medications to radiation therapy to surgical excision.[32]

The main medical therapy for NHO prophylaxis continues to be non-steroidal anti-inflammatory drugs (NSAIDs), which targets the early inflammatory phase.[30,33] Traditionally, indomethacin is considered the gold standard for prophylaxis following high-risk surgical procedures for the development of NHO. The recommended dose of indomethacin is 75 to 100 mg/day for seven to 14 days postoperatively, with monitoring for side effects such as ulcers, gastritis, or kidney injury.[32] Recent literature has suggested that less potent, nonselective NSAIDs such as ibuprofen or selective COX-2 NSAIDs such as celecoxib may be equally effective, with cost savings and a lower incidence of postoperative bleeding and side effects.[34] Bisphosphonate therapy is also effective in NHO prophylaxis, which can be especially useful in patients with contra-indications to NSAIDs.[35] As early prophylaxis, bisphosphonate regimens such as a three-day IV course of etidronate followed by a six-month oral course have effectively halted progression.[36] Limitations of bisphosphonate treatment include greater costs and treatment duration when compared to NSAIDs.[37] Additionally, bisphosphonates may be ineffective when started in the late stages of NHO (with positive radiographs) and have risks of severe associated side effects.[35]

Radiation therapy is also effective for NHO prophylaxis. This treatment involves the irradiation of pluripotent mesenchymal cells, which are thought to form heterotopic bone.[38] External beam radiation therapy is often prescribed at a 7 to 8

Gy fraction dose and typically given within 24 hours preoperatively or within 72 hours postoperatively.[30] Some studies have found radiation therapy to be superior to NSAIDs in preventing clinically significant NHO, while others report equivocal outcomes.[39,40] At present, there is no consensus on the most effective treatment, so either can be utilized based on provider preference, patient factors, and institutional protocols. It is important to consider radiation side effects such as wound healing delays, joint swelling, bony nonunion, and the rare incidence of secondary malignancy.[30]

Ultimately, high-grade NHO with functional impairment and pain may require surgical excision [**Figure 1A,B,C**]. Excision should be performed after the growth and maturation phases, as confirmed by serial radiographs, which can take over 1.5 years for TBI.[5] Surgeons should weigh the risks of prolonged debilitation and surgical complexity

Figures. 37-year-old male with neurogenic heterotopic ossification (NHO) of the left hip after surgical fixation of a complex left acetabular fracture in the setting of a polytraumatic motor vehicle collision with traumatic brain injury.

Figure 1. 3-view radiographs of the pelvis including AP [**A**], iliac oblique [**B**] and obturator oblique [**C**] views demonstrating extensive heterotopic ossification formation about the left hip in the setting of a prior posterior column acetabular fracture fixation four months post-operatively.

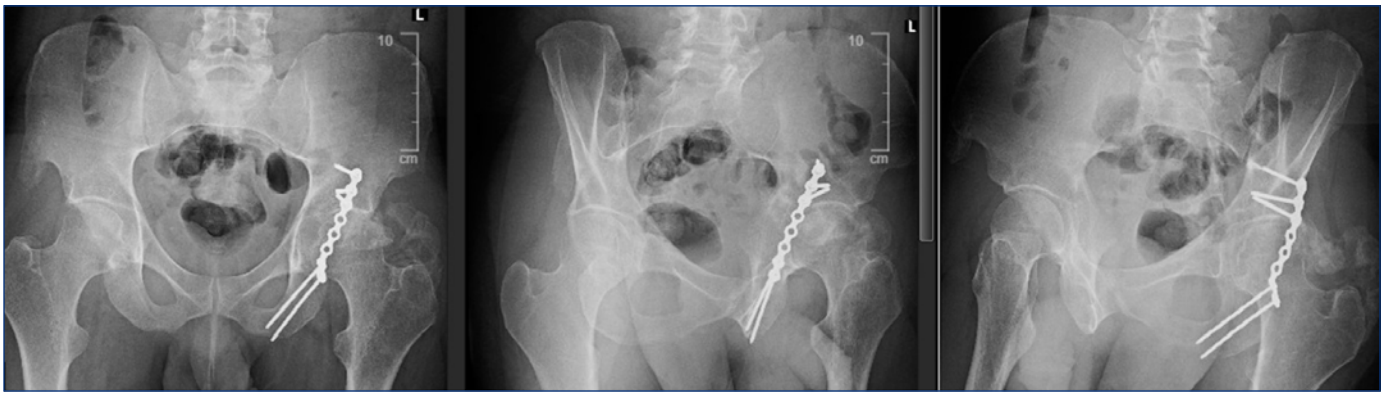


Figure 2. Computed tomography axial cuts at level of acetabulum [**A**], femoral head [**B**], greater trochanter [**C**] further characterizing the extent of heterotopic ossification at 4 months post-operatively

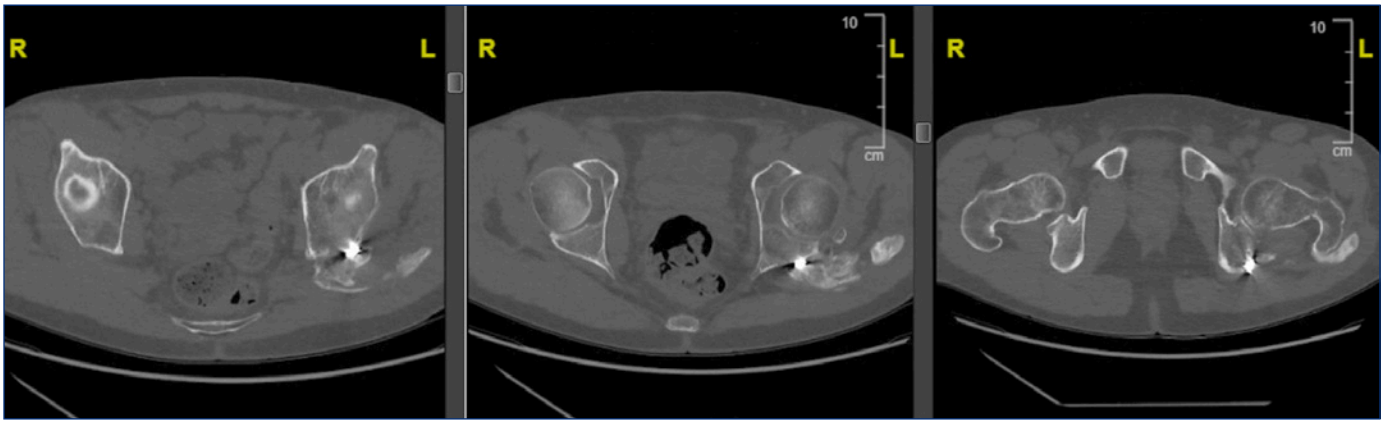
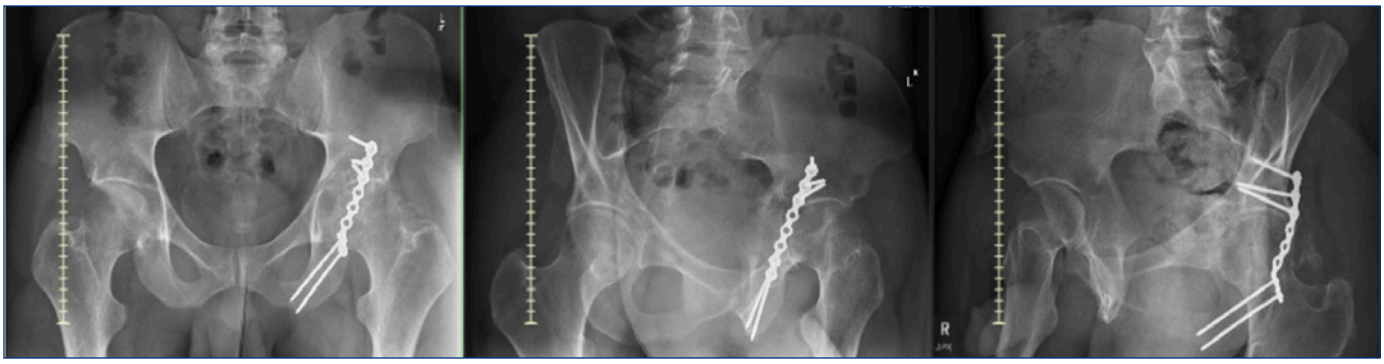


Figure 3. 3-view radiographs of the pelvis including AP [**A**], iliac oblique [**B**] and obturator oblique [**C**] at 4 months after surgical excision of prior NHO.



when considering surgical timing. Advanced imaging such as CT may be utilized for surgical planning, to further characterize the extent of heterotopic bone formation [**Figure 2A,B,C**]. Surgical management of NHO is challenging because excision may not fully address clinical symptoms and incomplete resection has recurrence rates as high as 33%.[41] Outcomes in the literature have been variable; many patients have improved pain and range of motion (ROM), but few achieve full resolution without recurrence.[42] While further high-quality investigations are required at this time, surgical excision is indicated in the patients with high-grade NHO and symptoms refractory to nonsurgical management. Additionally, due to high recurrence rates, surgical excision of heterotopic bone should be supplemented with medical treatments.[33] Traditional prophylaxis with NSAIDs, bisphosphonates, and radiotherapy is effective in reducing recurrence rates after surgical excision.[43] The role of additional medical management is further supported by findings in post-operative patients that new foci of ectopic bone are likely due to de novo formation rather than extension of unresected bone.[14] Therefore, the goal of resection, with medical prophylaxis for recurrence, is to improve range of motion while minimizing soft tissue trauma and surgical morbidity [**Figure 3A,B,C**].[44]

Regardless of the stage of NHO or treatment phase, physical therapy (PT) should be implemented throughout the disease course in conjunction with pharmacologic or surgical treatments.[33] While PT has not been shown to independently prevent NHO, range-of-motion exercises are crucial for preserving joint motion and preventing soft tissue contractures.[5] PT regimens may vary by institution and case-specific characteristics; however, early passive and active ROM of restricted joints in a controlled setting is recommended to preserve or optimize function prophylactically and post-treatment.[45,46]

CONCLUSIONS

Neurogenic heterotopic ossification is a difficult problem to treat in poly-traumatized patients with neurologic injuries due to the significant functional limitations it can place on already debilitated patients. While prevention is effective, no consensus exists on optimal treatment and surgery has variable results. Physical therapy remains an important mainstay of treatment in order to maintain range of motion. Further studies on the pathophysiology of this condition are crucial in order to develop treatment and prevention efforts and minimize the negative impact of NHO on patients.

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Authors

Jonathan Liu, MD, Brown University, Department of Orthopaedics, Providence, RI.

Noah Gilreath, BA, University Orthopaedics, Inc., East Providence, RI; UMass Chan Medical School, Worcester, MA.

Sydney Rozenfeld, MD, Brown University, Department of Orthopaedics, Providence, RI.

Sandi Caus, MD, Brown University, Department of Orthopaedics, Providence, RI.

Sarah Criddle, MD, Brown University, Department of Orthopaedics, Providence, RI.

Edward J. Testa, MD, Brown University, Department of Orthopaedics, Providence, RI.

Andrew Evans, MD, Brown University, Department of Orthopaedics, Providence, RI; University Orthopaedics Inc, East Providence, RI.

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Correspondence

Jonathan Liu, MD

593 Eddy Street, Providence RI, 02903

jonathanliu_liu2@brown.edu