*Case Series*

# A case series: The hip thing

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**Background**

Rehabilitation is a complicated process, no give or take there. To help improve the functional status of a patient we must be aware of the incidence, progression & treatment of expected complications, of the disease the patient is suffering from. The presence of Cognitive issues, Conative problems & Communication issues in patients also makes rehabilitation all the more challenging. This field is also becoming the hip thing globally, as the unmet need for rehabilitation is slowly catapulting this field to eminence. Entities that cause pain affect rehabilitation potential of patients to a great extent. In this article I wish to dilate on a pain condition which is sometimes neglected & is quite disabling if overlooked. Let's delve into 3 cases to better understand this pain entity.

**Keywords:** Traumatic Tetraparesis; Heterotopic Ossification; morphogenetic protein; Ultrasonography

## 1. Case Presentation

### *1.1 Case 1*

The first patient is a 20-year-old female who had Haemorrhagic Encephalitis sequelae & was Aphasic. She was assessed 5 months' post insult. There was severe ROM restriction of the Right hip joint & any movement causes her to wince & withdraw from further examination. Because of this pain she was not tolerating Therapeutic sitting & standing properly. Lab investigations revealed anaemia & elevated S. ALP levels (#). No features of infection (TLC, CRP levels were within limits) were obtained & D-Dimer levels were normal. We X-rayed her hip (bedside). The images are discussed below (of all the three patients).

**Citation:** Bineesh Balakrishnan. A case series: The hip thing. Kauverian Med J. 2025;2(9):1-5.

Academic Editor: Dr. Venkita S. Suresh

ISSN: 2584-1572 (Online)

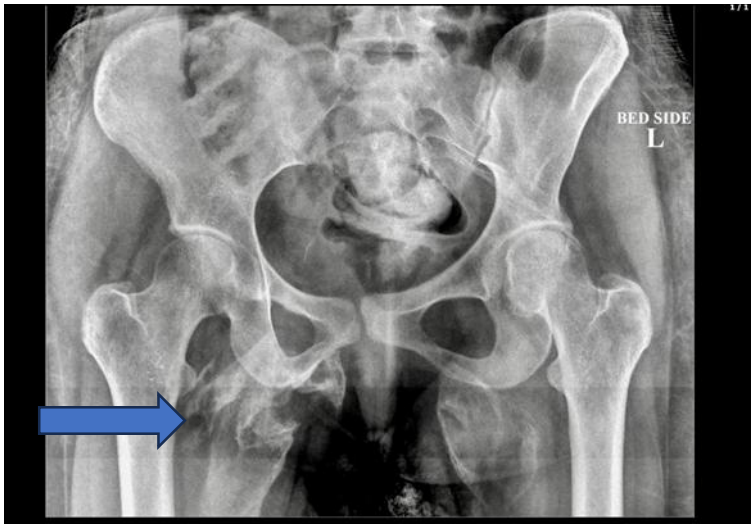


**Fig (1)**: Bedside X-ray of both hips AP view of Case 1

***1.2. Case 2***

A 58-year-old male with Traumatic Tetraparesis (S/P Spine fixation), who had a history of Ankylosing Spondylitis, was assessed at 6 months' post insult. He had restriction of ROM of the Left hip associated with severe pain. Because of the pain he couldn't tolerate supported standing. Blood investigations showed anaemia & elevated S. ALP (#). D-Dimer levels were normal as were the TLC & CRP. Bedside X-ray of his Left hip was taken.

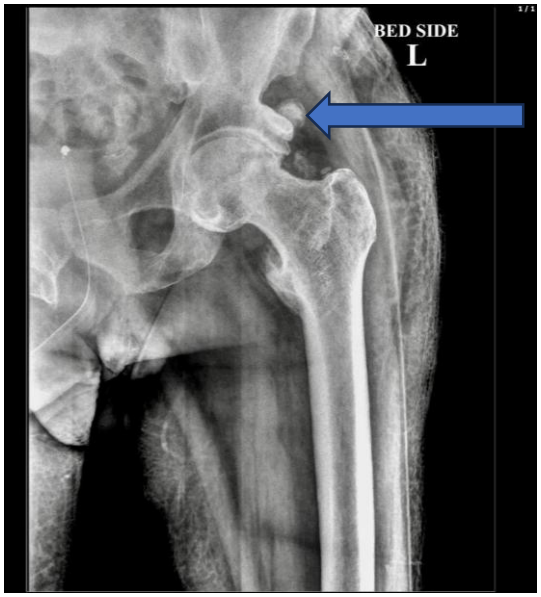


**Fig (2)**: Bedside X-ray of Left hip AP view of Case 2

***1.3. Case 3***

A 54-year-old male with Traumatic Tetraparesis (S/P Spine fixation). He was a Diabetic & Hypertensive on treatment. Around 6 months' post insult, on examination, he had pain & ROM restriction in the Left hip. This affected how long he tolerated therapeutic standing. S. ALP was elevated (#). D-Dimer, TLC & CRP were normal in this patient. Left hip X-ray(bedside) of this patient was also taken.

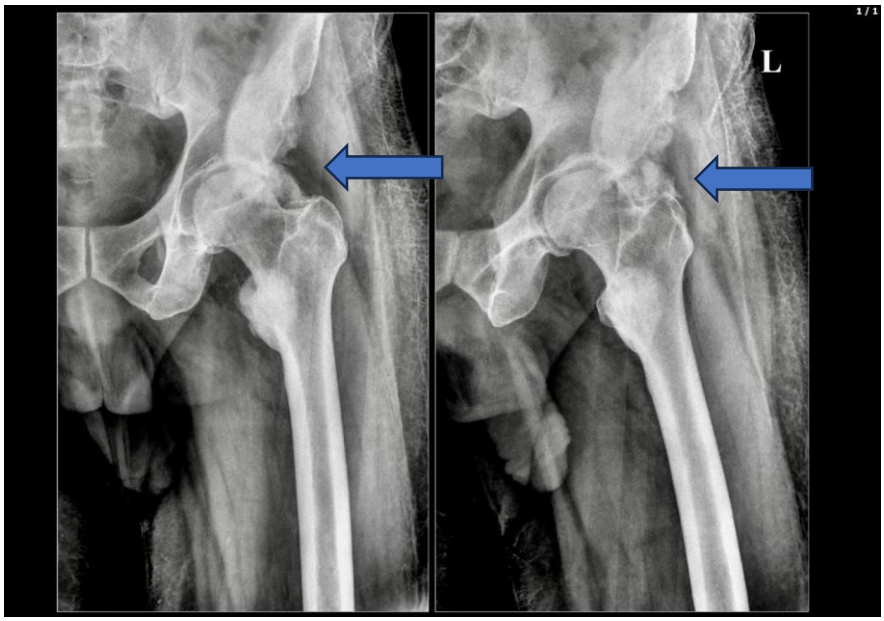


**Fig (3)**: Bedside X-ray of the Left hip AP of Case 3

So, in all three patients there was a CNS insult which eventually resulted in abnormal bone growth around the hip joints with elevated levels of S. ALP. This eventually caused significant pain & ROM restriction around the joints which compromised their rehabilitation potentials.

**The X-rays**: All the These X-rays were taken at bedside in our centre. The bone growth is conspicuous all the same.

**2. The Diagnosis**

The entity we're dealing with is called Heterotopic Ossification. It can be hereditary or acquired. Hereditary HO is a rare but life-threatening genetic disorder that mainly comprises fibrodysplasia ossificans progressiva (FOP, divided into classic and atypical types) and progressive osseous heteroplasia (POH). Evidence has shown that hereditary HO results from spontaneous mutations in a specific gene. FOP is characterized by a large amount of ectopic ossification in postnatal muscles and tendons, which has been proven to be caused by gain-of-function mutations in the bone morphogenetic protein (BMP) type I receptor, located in the ACVR1 (also called ALK2) gene located on chromosome 2. In 2002, Kaplan et al confirmed that POH was associated with a mutation in the GNAS gene.

**3. Acquired Heterotopic Ossification**

Acquired HO is the most common form, is considered aberrant tissue repair, and can be broadly categorized into four etiologic subtypes according to the diversity of trigger factors:

1. Neurogenic injury
2. Orthopedic surgeries
3. Severe burns

4. Combat-related injuries.

Pain & restriction of ROM are the most common clinical findings at presentation. Other clinical signs include local swelling, erythema, warmth in the joint, muscle guarding, and low-grade fever.

The hip is the most common site of HO after TBI, SCI & Stroke, followed by the elbows, shoulders, and knees. Diagnosing HO first begins with a clinical suspicion based on its clinical presentation and ruling out other causes such as infection, venous thromboembolism (VTE), or undiagnosed orthopaedic injury.

**4. On S. ALP**

An elevated serum alkaline phosphatase may be found during early formation of HO. S. ALP is a sensitive indicator, increasing in the advance of symptoms and radiographic findings. Levels become abnormal approximately 2 weeks after onset and reach a peak, typically 3.5 times normal by about 8 weeks later. Unfortunately, S. ALP has poor specificity, as it can be elevated for multiple other reasons like surgical intervention for fractures, hepatotoxicity, cholestasis, etc. Although not specific for HO, patients with HO may have elevated creatinine kinase levels, which may be helpful in treatment planning and evaluation of response to treatment**.**

**5. Imaging in HO**

Ultrasonography has been used in the early diagnosis of HO about the hip joints. However, it is an operator-dependent examination, and no data are available on the value of ultrasonography in the diagnosis of HO in other joints. Plain radiographs are often unremarkable early in the process of bone formation, revealing evidence of HO weeks to months later. The most sensitive imaging modality for early detection and assessing the maturity of HO is the three-phase technetium-99m (99mTc) methylene diphosphonate bone scan. CT and MRI may be useful in delineating local anatomy prior to resection, but the role of these imaging modalities in the evaluation of other aspects of HO has not been well established.

**6. Treatment options**

**6.1. Drugs & Radiation Therapy**

Though there's no consensus about which drug to use, NSAIDs & Bisphosphonates are the most commonly used ones for managing this condition. Indomethacin remains the gold standard for HO prophylaxis following THA, although other NSAIDs, including naproxen and diclofenac, are equally as effective and can be considered as alternative first-line treatments. Indomethacin prescribed for 3 weeks in a dose of 75 mg/d after spinal cord injury reduced the incidence of HO by a factor of 2 to 3. For most patients undergoing THA, a 7-day course of indomethacin has been recommended as a prophylaxis against HO. Other drugs routinely used include Etoricoxib & Bisphosphonates (Etidronate infusions being the most studied).

Radiation therapy (RT) has been found to be more effective than NSAIDs in multiple clinical trials and may be administered at a dose of 700–800 cGy either 2–4 h preoperatively or within 24 hr postoperatively. Active stretching exercises should be advocated to avoid permanent loss of motion or joint ankylosis in patients with preexisting HO.

### 6.2. Surgery

Surgical excision of HO should be considered in cases where the intervention is anticipated to improve function, such as standing posture and ability to sit or ambulate or independently perform activities of daily living. Excision should also be considered for patients in whom an underlying bone mass contributes to repeated pressure injuries. Planned excision typically occurs 12 to 18 months postinjury to allow for maturation of HO. Garland has recommended different schedules for surgical intervention, depending on the aetiology of the condition underlying the HO: 6 months after direct traumatic musculoskeletal injury, 1 year after spinal cord injury, and 1.5 years after TBI.

The ideal candidate for surgical resection of HO before 18 months (after TBI) will have no joint pain or swelling, a normal alkaline phosphatase level, and a three-phase bone scan indicating mature HO. Complications of surgical removal of HO include haemorrhage, wound healing problems, cellulitis, infection including osteomyelitis, and possible recurrence of HO.

## 7. Conclusion

Our patients responded to T. Indomethacin & a stretching protocol. Repeat X-rays showed reduction in bone growth. All 3 patients tolerated therapeutic sitting & standing for longer durations & had significant pain relief. Managing this complication greatly improved their rehabilitation outcomes. For early detection of this entity, a high index of suspicion is needed & three-phase technetium-99m (99mTc) methylene diphosphonate bone scan should be considered.

Neglecting this entity can greatly reduce the rehabilitation potentials of the patients. TBI-associated HO is associated with significantly longer inpatient rehabilitation hospitalizations and poorer FIM scores. More specifically, both admission and discharge FIM scores are lower, mostly accounted by ADL and mobility subscores, when compared with matched persons with TBI without clinical stigmata of HO.

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