Heterotopic Ossification in Polytrauma

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Background: Heterotopic ossification (HO) is the pathologic development of ectopic bone in muscle and soft tissue. HO is a known complication that can arise in cases of fracture, surgery, burn injury, and nervous system injury. The co-occurrence of traumatic brain injury (TBI) and fracture increases the risk of HO development by 37-50%. This review aims to examine and synthesize the mechanisms involved in HO development after polytrauma, available animal models to study these mechanisms, and possible treatment options for patients.

Project Methods: A literature search was conducted using PubMed and EMBASE with search terms: heterotopic ossification, fracture, and traumatic brain injury. This search produced 33 unique articles.

Results: TBI and fracture contribute to HO development by triggering the release of inflammatory cytokines, neuropeptides, and hormones in response to nervous system and bone damage. Bone morphogenetic protein (BMP), vascular endothelial growth factor (VEGF), and Substance P (SP) are among the known contributors to HO formation. Damage to the blood brain barrier and release of nervous system factors may also contribute to the osteogenic environment that promotes ectopic bone development. Rodent modeling of the biologic mechanisms underlying HO can be exploited to better understand the molecular causes of the condition. There are also options for both prophylaxis and treatment of HO. Prophylaxis options include radiation treatment, non-steroidal anti-inflammatory drugs (NSAIDs), and bisphosphonates. Treatment options include physical therapy and surgical resection.

Potential Impact: HO has a high occurrence rate in military personnel exposed to combat trauma and can lead to restriction of joint movement, pain, and infections in soft tissue. There are limited options for treatment, pointing to a need for further study.