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CASE REPORT
Peripheral Nerve

Heterotopic Ossification Complicating Traumatic Ulnar Nerve Palsy in a Child

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Summary: Heterotopic ossification (HO) refers to the process of forming bony tissues in extra-skeletal sites such as muscles and soft tissues. This pathological process most commonly commences following trauma, surgery, and fractures. Rarely, HO can compress nearby neurovascular structures. To the best of our knowledge, however, no previous reports exist of HO serving as second-hit to partially injured ulnar nerve in the pediatric population. We present a case of a 6-year-old girl with complete high ulnar nerve palsy caused by medial epicondylar fracture followed by development of HO around the elbow. The patient was initially managed conservatively. Four months after the first visit, she presented with complete ulnar nerve palsy that was not evident on the initial presentation. Based on further investigations, she was taken to OR for release of the cubital tunnel and anterior transposition of ulnar nerve. On further follow-up, the patient regained sensory and motor functions of the ulnar nerve with minimum sequalae. Post-traumatic ulnar nerve injury is well described in the literature as resulting from initial trauma or as an iatrogenic injury following fracture reduction and fixation. HO in the pediatric age group is considerably rare, occurring after trauma and burn injuries. Surgical timing of HO release remains controversial. No previous reports of HO complicating traumatic ulnar nerve palsy in pediatric patients exist. In the current case report, partial ulnar nerve injury was initially due to medial epicondylar fracture, but it had progressed to full blown ulnar palsy secondary to superimposed HO. Early HO takedown combined with decompression of ulnar nerve are crucial to ensure complete nerve recovery. *(Plast Reconstr Surg Glob Open 2022;10:e4089; doi: 10.1097/GOX.0000000000004089; Published online 9 February 2022.)*

Heterotopic ossification (HO) refers to the process of forming bony tissues in extraskeletal sites such as muscles and soft tissues. This pathological process most commonly commences following trauma, surgery, and fractures.[1] Formation of lamellar bones in muscles and soft tissue can occur in genetically predisposed individuals or in a nongenetic fashion.[2] HO can present with different forms depending on the location and progression. It may start as a localized pain and tenderness. However, it can progress to a firm swelling that can block the range of motion. Rarely, it can result in peripheral nerve compressions[3] as the ulnar nerve passes through a potentially tight tunnel behind the medial epicondyle. The most common cause of ulnar nerve palsy in children above the age of 5 years is supracondylar fracture of the humerus.[4] The prevalence of neurovascular involvement in displaced supracondylar fracture ranges from 10% to 20%.[5] The affected nerves reported in the literature following such fracture are median and anterior interosseous nerves.[6] To the best of our knowledge, however, no previous reports exist of HO complicating traumatic ulnar nerve neuropathy in the pediatric population. We present a case of progressive posttraumatic high ulnar nerve palsy complicated by HO around the elbow in a pediatric patient.

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CASE PRESENTATION

A 6-year-old girl presented to the emergency department 4 weeks after sustaining a fall on an outstretched

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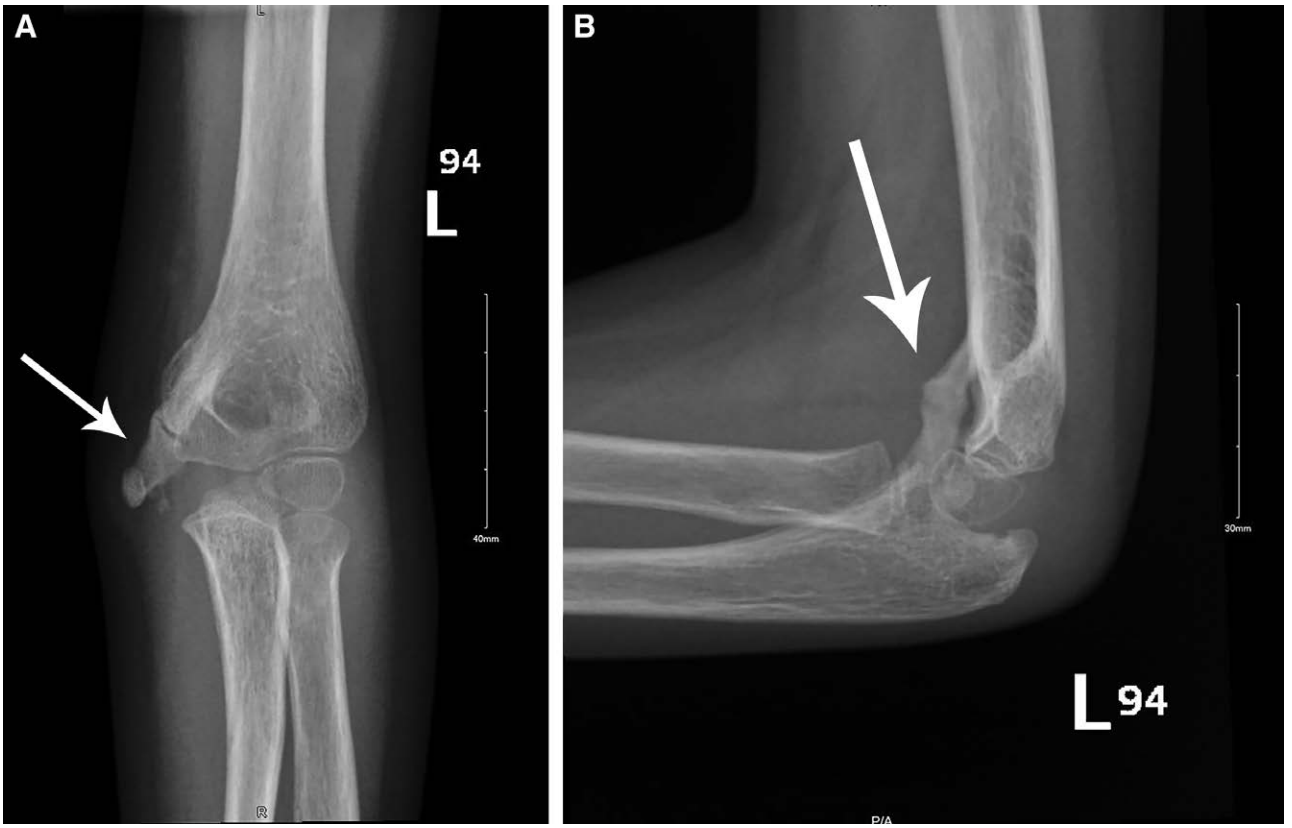

Fig. 1. Initial X-ray images upon presentation showing bridging ossification at the elbow. A, AP view. B, lateral view.

elbow. She was initially diagnosed and managed conservatively at another hospital for an occult left supracondylar fracture. Surgery was offered to the family for fracture fixation, but they elected to seek another opinion and conservative management with immobilization was done. Initial assessment at the emergency department revealed pain at the elbow with limited painful motion with a point of maximal tenderness over the medial epicondyle and no tenderness elsewhere. There was no swelling or bruises or open wounds. Neurovascular evaluation showed warm well-perfused hand with normal capillary refill and intact median and radial nerve functions. Ulnar nerve function was found to be moderately compromised as sensation over the ulnar aspect of the hand, along with abduction of fingers, was markedly decreased. Radiologic evaluation with x-rays showed medial epicondyle fracture. A CT scan showed a comminuted displaced medial epicondylar fracture. Due to the late presentation and status of the fracture, orthopedics elected to continue on the conservative management.

Table 1. Preoperative and Postoperative Nerve Conduction Study of Ulnar Nerve Function

Stim. Site	Onset (ms)	Normal Onset (ms)	O-P Amp (mV)	Normal O-P Amp	Velocity (m/s)	Normal Velocity (m/s)
Preoperative left ulnar motor						
Wrist	**5**	<4.2	**2.2**	>3	**22**	>53
Postoperative left ulnar motor						
Wrist	3	<4.2	8.4	>3	54	>53
Right ulnar motor						
Wrist	2	<4.2	11.2	>3	64	>53
Stim. Site	**Response**	**Peak (ms)**	**Normal Peak (ms)**			
Preoperative left ulnar sensory (fifth digit)						
Wrist	**No response**	**No peak**	**<3.7**			
Postoperative left ulnar sensory (fifth digit)						
Wrist	**Yes**	**3.4**	**<3.7**			
Right ulnar sensory (fifth digit)						
Wrist	**Yes**	2.7	<3.7			

Text in bold highlights the extent of ulnar nerve damage preoperatively and nerve recovery postoperatively.

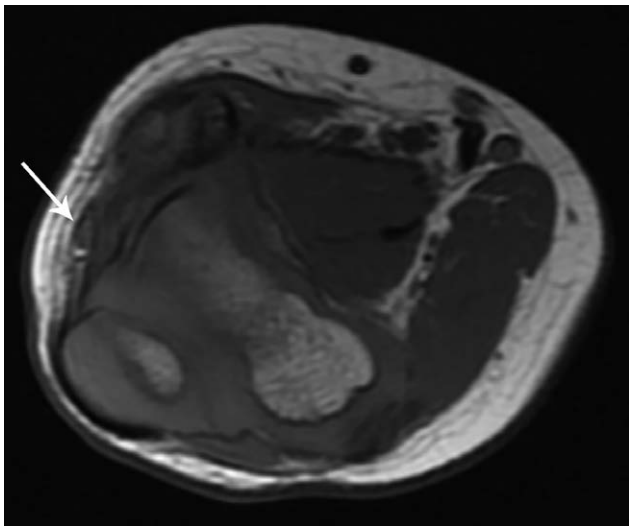

Fig. 2. Post contrast T1 weighed axial magnetic resonance imaging showing enhancement and thickening around ulnar nerve suggestive of nerve compression.

Ulnar nerve weakness was followed closely as it was considered more likely to be a posttraumatic neuropraxic injury at this stage. Patient returned to clinic 4 months after initial injury when she was found to have worsening of her high ulnar nerve palsy with full-blown clawing and absent sensation of the fourth and fifth digits. Intrinsic muscle function was severely impaired. Physiotherapy was started and elbow range of motion improved to restore −30 degrees of extension to 90 degrees of flexion. Follow-up X-rays revealed a large exophytic HO lesion adjacent to medial epicondyle (Fig. 1A, B). In the meantime, nerve conduction study was done to evaluate the function of ulnar nerve. It showed evidence of left sided axonal injury involving motor and sensory fibers suggestive of compression at the level of elbow joint (Table 1). Magnetic resonance imaging was done to assess the position and course of ulnar nerve in cubital tunnel which revealed avulsed medial epicondyle with dystrophic calcified ridge and distorted cubital tunnel architecture with fibrosis and inability to visualize the ulnar nerve within the tunnel (Fig. 2). It is likely the ulnar nerve injury was related to the initial fracture and later compromised by the developing HO. Early surgical intervention to decompress the nerve and take down the HO was deemed necessary.

Operative exploration of ulnar nerve combined with orthopedics team for HO take down was performed 5 months following the initial presentation to our hospital. HO was found extending from the medial epicondyle and spanning the roof of the cubital tunnel. With careful dissection, HO was removed and ulnar nerve was found to be severely compressed with an hour-glass appearance under the area of bony growth proximal to the cubital tunnel (**see figure, Supplemental Digital Content 1**, which shows hour-glass appearance of compressed ulnar nerve, **http://links.lww.com/PRSGO/B910**). External neurolysis was done to transpose the nerve anteriorly. Branches of the superior ulnar collateral artery were kept with the nerve to maximize its blood supply. The nerve was transposed anteriorly in a subcutaneous plane and stabilized using a fascial sling.[7] The patient was placed in backslab for 3 weeks and physiotherapy was started afterwards. Six weeks postoperatively, marked improvement in both sensory and motor functions was noted. At 1-year follow-up, the patient showed complete recovery of the ulnar nerve function, which was confirmed with normal nerve conduction studies (Table 1).

DISCUSSION

Posttraumatic ulnar nerve injury is well described in the literature as resulting from initial trauma or as an iatrogenic injury following fracture reduction and fixation. A retrospective study investigated the possibility of iatrogenic ulnar nerve injury following surgical treatment of displaced supracondylar fracture by closed reduction and percutaneous pinning. The study concluded that the use of crossed pin configuration resulted in higher incidence of ulnar nerve injury.[8] Furthermore, a systematic review of pediatric medial epicondylar fractures found the incidence of posttraumatic ulnar nerve injury to be as high as 9.6%.[9]

HO in the pediatric age group is considerably rare.[8,9] It typically occurs following an enticing event such as traumatic fractures and burn injuries. Surgical timing of HO release remains controversial. Historically, surgical intervention is delayed until complete HO maturity between 12 and 18 months to reduce the risk of recurrence. In a recent systematic review of timing of surgical release of posttraumatic elbow stiffness, better results were noticed in the early intervention group (6–10 months) compared to intermediate (11–20 months) and late (>20 months) groups.[10]

Management of worsening ulnar nerve injury secondary to HO involves early HO takedown and nerve decompression, which is well described in the adult population. The presence of symptoms suggestive of ulnar nerve injury proposes a need for surgical intervention to decompress the nerve. The functional outcomes and recovery of ulnar nerve were found to be excellent in early surgical intervention.[11] Yang et al[12] reported seven cases of ulnar nerve palsy associated with HO secondary to elbow trauma in adult patients (four elbow dislocations, one medial epicondyle fracture, one coronoid fracture, and one radial neck fracture). All patients underwent early surgical treatment of HO (mean time of 6 months postinjury) using posterior approach and anterior transposition of ulnar nerve. Ulnar nerve motor and sensory functions recovered in all patients at 12 months postoperatively.[12] Another report of ulnar nerve compression in a 20-year-old patient caused by HO secondary to the radial head fracture was treated by surgical release and nerve in situ decompression, which resulted in complete nerve recovery.[13] Eight cases of brain-injury-related HO of the elbow with ulnar nerve compression in adult patients were reported. All cases underwent early surgical release and transposition of ulnar nerve with good to excellent outcomes in seven of eight patients.[14]

Brooke et al[15] reported a case of ulnar nerve compression due to HO in a major burn patient which appeared 3 months after his burn. He was treated by early surgical release of HO and anterior transposition of ulnar nerve with good nerve recovery.[15]

Despite being rare, ulnar nerve palsy in the pediatric age group occurs mainly following acute trauma to the elbow region. No previous reports of HO complicating traumatic ulnar nerve palsy in pediatric patients exist. In the current case report, partial ulnar nerve injury was initially due to medial epicondylar fracture but it had progressed to full-blown ulnar palsy secondary to superimposed HO. Early HO takedown combined with decompression of ulnar nerve was deemed necessary and resulted in complete nerve recovery.

CONCLUSIONS

Worsening ulnar nerve function following fractures should raise the suspicion for another insulting event such as HO. In such cases, early HO takedown and surgical decompression of the ulnar nerve are warranted to maximize nerve recovery.

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