A heterotopic ossification related to temporary placement of a skull bone flap

Case report

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✓ The authors report on a 19-year-old man who sustained a traumatic brain injury. Following decompressive craniotomy, he suffered from an unusual heterotopic ossification due to the temporary placement of the skull bone flap in his thigh. To the best of the authors’ knowledge, this is the first time that a possible causal relationship between these entities has been reported in the literature. (DOI: 10.3171/JNS/2008/108/2/0370)

KEY WORDS • heterotopic ossification • skull bone flap • thigh • traumatic brain injury

HETEROTOPIC ossification is the deposition of mature, lamellar bone in soft tissue, especially around the large joints such as the hip, knee, and elbow. The precise cause of HO after TBI is still unidentified, and there may be multiple predisposing factors such as immobility, limb spasticity, prolonged coma, and deteriorated local circulation. The role of trauma (for example, surgery) has also been pointed out by several authors for the mechanism of ossification.

We report on a 19-year-old man who had suffered HO in the left hip joint after decompressive craniectomy. To the best of our knowledge, this is the first report of an HO occurring near a temporary implanted skull bone flap in the thigh.

Case Report

History and Examination. This 19-year-old man who suffered a TBI after a motor vehicle accident was admitted to our rehabilitation center after a 40-day stay in the intensive care unit. The patient had undergone emergency decompressive craniotomy at another hospital, and his skull bone flap had been preserved subcutaneously in the left thigh. He had not sustained any other bone fractures or any spinal cord injury.

The patient was found to be confused and inappropriate (Rancho Los Amigos Level V) on admission. His upper- and lower-extremity motor functions were Grade 4/5 on the right side and Grade 3/5 on the left side. The left hip joint motions were painful and severely limited during internal rotation and less during flexion/extension. The serum alkaline phosphatase level was higher than normal 1226 U/L (normal range 34–155 U/L). Radiological evaluation of the hip joints disclosed a left-sided HO near the skull bone flap in the thigh (Fig. 1).

Treatment. The patient was started on combined therapy of etidronate 20 mg/kg/day and indometacin 100 mg/day. In the meantime, he underwent physical and cognitive therapy. After a 4-week uneventful hospital stay, he was transferred to the neurosurgery department for cranioplasty.

Posttreatment Course. Three months after surgery, he was seen for a follow-up visit. The range of motion in his hips was found to be more limited than it had been, and a repeated x-ray of the hip joint showed increased HO formation (Fig. 2). He was functionally independent during daily activities. One year after this visit he will undergo examination for possible surgery to treat his HO.

Discussion

The incidence of HO after TBI has been reported in the literature to be between 10 and 20%. The hip joint and the elbow are the most common locations for this condition. Heterotopic ossification commonly occurs at 3 sites around the hip. Anterior HO courses in a plane from the anterior superior iliac spine to the greater trochanter. Inferomedial HO is positioned distally to the hip joint and medially to the femoral shaft. Posterior HO occurs immediately posterior to the femoral head and neck. Occasionally, combined patterns or even localization in the abductor muscle may occur. In our case, the HO was present postcranioplasty to the left proximal femoral shaft and extended 10 cm below the trochanter minor.

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Heterotopic ossification related to skull bone flap

The disturbance in local circulation caused by either a change in muscular function or by the production of substances such as fibroblast growth factor that stimulate the local development of bone tissue. Besides significant spasticity, a coma lasting longer than 2 weeks and proximity to fractures in long bones have been considered the main risk factors of HO in association with TBI. None of these factors were present in our patient.

Although its exact mechanism is yet unclear, the role of trauma (for example, surgery) has been noted by several authors in the literature as causing the formation of HO. It may occur through an effect on muscle and connective tissue, leading to activation of undifferentiated mesenchymal cells, or through an effect on existing osteogenic cells in the adjacent periosteum and the skeleton. We hypothesize that in our patient, implantation of the skull bone flap subcutaneously in the thigh might have induced a traumatic form of HO. The fact that the HO occurred at an unusual site extending down to the femoral shaft and that it was aggravated significantly after the second surgery seem to be relevant with our hypothesis. On the other hand, concomitant contribution of the initial TBI on the formation of HO should not be underestimated.

In conclusion, by presenting the first case of HO development related possibly to a skull bone implant in the thigh of a patient with TBI, we alert surgeons to this rare entity. Furthermore, we suggest that other possible areas of safe bone implantation such as abdominal or chest wall, may be preferable especially in patients with the aforementioned risk factors.

References


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