Osteolytic skull lesions secondary to trauma

Report of two cases

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Two patients with posttraumatic osteolytic skull lesions are presented and discussed. One was operated on, and pathological examination confirmed an inflammatory reactive process within the bone lesion.

KEY WORDS • skull injury • osteolytic skull defect • children

Two cases are reported in which osteolytic skull lesions occurred in the frontal bones of children following trauma. Soft-tissue swelling and periosteal inflammatory reaction was present at the site directly overlying the skull lesions. The skull lesions involved both the inner and outer tables. The dura was not invaded in either case, although reactive inflammatory changes were noted on the surface of the dura in one patient who underwent surgery. There were no systemic or local signs of infection in either child.

Case Reports

Case 1

This 14-year-old boy fell from a motorcycle and struck the right side of his forehead, causing localized swelling. Three weeks later he noted a depression in the skull in the area of previous swelling.

Examination. A 2-sq cm nontender, nonpulsatile depression was present in the right frontal area. Neurological examination was within normal limits. The patient had no clinical signs or symptoms of renal, thyroid, or parathyroid disease and denied any history of drug or metal ingestion. Skull x-ray films demonstrated a 2-sq cm lytic lesion in the right frontal bone (Fig. 1). Magnetic resonance imaging did not show any intracranial extension, but the process involved both the inner and outer tables. A bone scan showed increased uptake over the right frontal bone (Fig. 2). A long-bone skeletal survey showed no evidence of other lytic lesions.

Operation. Thickened periosteal granulation tissue covered a 2.5-cm diameter hole involving both the inner and outer tables. The peristeum was excised. The diploic space and surrounding bone margin was curetted and rongeured for possibility of any tumor involvement. Thickened tissue was scraped from the dural surface. An autologous bone cranioplasty was performed, and postoperative radiographs showed good healing.

Pathological Examination. All tissue cultures were negative for bacterial growth. Microscopic examination of material from the dural surface showed granulation tissue composed of fibrous connective tissue infiltrated with small lymphocytes. The bone fragments contained thin areas of fibrous connective tissue and lymphocytes. There were no eosinophils. The periosteal granulation tissue was composed of strips of fibrous connective tissue containing scattered mononuclear leukocytes, hemosiderin-laden macrophages, and multinucleated giant cells with brown pigment. Three pathologists independently concluded that the findings represented a reactive process secondary to previous trauma.

Case 2

This 6-year-old girl struck her head and developed considerable localized swelling on the right side of the forehead, self-described as a bluish “goose egg.” She had two other episodes of lesser trauma to that same area. The patient was otherwise entirely healthy with no drug history.

Examination. The patient had a prominent, nontender swelling in the right frontal area, measuring 4 sq cm 1 month following trauma. A lytic lesion was visible on skull x-ray film. A computerized tomography (CT) scan nearly 2 months following initial trauma showed...
Traumatic osteolytic skull lesions

FIG. 1. Anteroposterior skull film showing the right frontal posttraumatic osteolytic defect.

Fig. 2. Case 1. Technetium-99 bone scan, anteroposterior projection, showing increased uptake in the right frontal bone.

FIG. 3. Computerized tomography scans in Case 2. Left: A right frontal full-thickness skull defect is seen with overlying soft-tissue swelling. Right: Incomplete healing of the frontal lytic lesion is visible 18 months following trauma.

the bone scan showed solitary uptake in the right frontal bone.

Course. No surgery was performed. The patient was followed and the skull defect began to close. Six months after injury, the lytic lesion could no longer be appreciated on skull films, and there was only slight depression in the right frontal scalp area. A CT scan obtained 18 months after trauma showed incomplete healing of the skull lesion (Fig. 3 right). The child remained neurologically intact.

Discussion

Osteolysis of bone following trauma has been described involving the clavicle and pubis bone where fracture was not present. Posttraumatic osteolysis involving the skull has not been reported. We report two cases in which osteolytic skull lesions occurred in the frontal bone following trauma. Hematoma and soft-tissue swelling occurred prior to the development of skull lesions. Bone erosion involved both inner and outer tables. The lesions were approximately 2.5 cm in width. The dural layer was not compromised in the first case, although reactive inflammatory changes occurred on the surface of the dura. There were no systemic or local signs of infection in either child. Regrowth of bone began in the unoperated child 2 months following the initial trauma.

In Case 1, the osteolytic process was assumed to be neoplastic. The association of trauma was made only after histological specimens were obtained at surgery. Based on this experience, the child in Case 2 was followed without surgery; the posttraumatic osteolysis in this case was self-limited over several months.

The literature concerning skull lesions in association with soft-tissue changes of the scalp does not record any osteolytic process. Repeated head trauma and chronic subgaleal hematoma resulted in partial calcification within the subgaleal space in one patient. A
case of aggressive osteoblastoma was associated with a 10-year-old subgaleal hematoma causing erosion of the outer table. Cephalohematoma in the neonate at either the subgaleal or subperiosteal level has rarely been associated with calcifications and never with osteolysis. 

Gorham’s disease is a condition of massive osteolysis which rarely affects the skull in isolation. The role of angiomatosis in Gorham’s disease (a nonneoplastic proliferation of vascular channels resulting in resorption of bone and its replacement by fibrosis) is not well established in isolated skull lesions. Two cases in the neurosurgical literature have been diagnosed as massive osteolysis where biopsy was not performed; in both, the lesions were large, chronic, and not associated with trauma. Two other cases are reported with large, progressively enlarging osteolytic calvarial lesions involving the full thickness of the skull. Pathological examinations showed dense epidermal connective tissue changes at the site of the bone defects with normal vascularity of involved bone.

The cranial osteolytic lesions presented here resemble the changes reported in the clavicle and pubis following trauma. Cranial osteolytic changes were present within 1 month following head trauma. Levine, et al., could detect radiographic osteolysis in the clavicle as early as 2½ weeks after trauma. In both of our cases soft-tissue swelling of the scalp was prominent and persisted for several days to weeks. Soft-tissue swelling in the acromioclavicular joint occurred prior to erosion of the distal clavicle. Technetium bone scans exhibited increased uptake in osteolytic lesions of the skull, clavicle, and pubis. Histopathological findings for clavicular and pubic bone osteolytic lesions have varied considerably, ranging from necrosis to bone degenerative change, nonspecific inflammatory change, and fibro-ossseous proliferation. The heterogeneity of pathological findings would suggest different disease processes but could also be attributed to the difficulty in sampling a process in evolution.

Of the many agents that induce resorption, one possible common chemical pathway is the release of organic acids together with enhanced biosynthesis and release of many lysosomal enzymes. At a cellular level, hemotactic factors (such as collagen degradation products) activate osteoclasts, which mediate bone resorption. Direct bone resorption can occur via human monocytes in vitro. Other known activators of bone resorption, such as vitamin A, excess parathyroid hormone, thyroxine, 1,25 (OH) vitamin D3, lead, and heparin, are all well identified but have generalized, not focal, effects. Given the multiplicity of factors involved in the lysis of bone, it is not surprising that lesions with similar radiographic appearance have diverse histopathological findings.

It is concluded that minor trauma to the skull can precipitate an osteolytic lesion. The trauma triggers a nonspecific inflammatory reaction involving the periosteum. The periosteal inflammatory reaction can be quite aggressive and result in focal osteolysis. Over the course of several months the osteolytic process ceases and remodeling ensues. If lytic skull lesions can be unambiguously associated with previous blunt head trauma and concomitant soft-tissue changes, then it may be prudent to follow them expectantly. If the defects do not enlarge after 2 months and there is no evidence of intradural extension or infection, conservative treatment is justified.

References