Mandibular Brown Tumor Linked to Tertiary Hyperparathyroidism: A Case Report

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Background

- Brown Tumors (BT) are rare, osteolytic lesions caused by excessive osteoclastic activity due to hyperparathyroidism (HPT).
- Typically affect long bones, with craniofacial involvement being rare, especially in children.
- Pediatric patients with chronic kidney disease (CKD) and renal osteodystrophy are at higher risk due to secondary or tertiary HPT. Early diagnosis is crucial, as BTs often regress with metabolic control of HPT.
- This case emphasizes the need for a multidisciplinary approach for accurate diagnosis and management.

Case Presentation

- **Patient:** 9-year-old female.
- **Chief Complaint:** Facial swelling with mandibular expansion and mobility of incisors (#23-26).
- **Medical History:** ESRD (hypoplastic kidneys), severe malnutrition, rickets, tertiary HPT (PTH: 2494 pg/mL), on hemodialysis, vitamin D, and calcimimetics.
- **Clinical Findings:** Right mandibular expansion (5 cm), firm swelling, intact mucosa, Class II mobility of incisors.
- **Radiographic Findings:** Diffuse ground-glass bone pattern, osteolytic lesion (11X13X12 mm) with cortical thinning, root displacement, alveolar bone loss.
- **Histopathologic Findings:** Sheets of spindle cells with diffuse multinucleated giant cells, hemosiderin deposition, and reactive bone formation, consistent with Brown Tumor (BT).

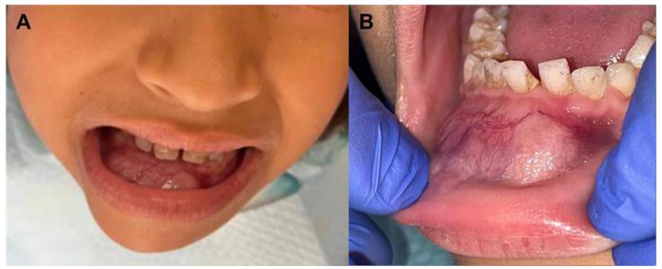

Figure 1: Extraoral (A) and intraoral (B) clinical photograph of the lesion.

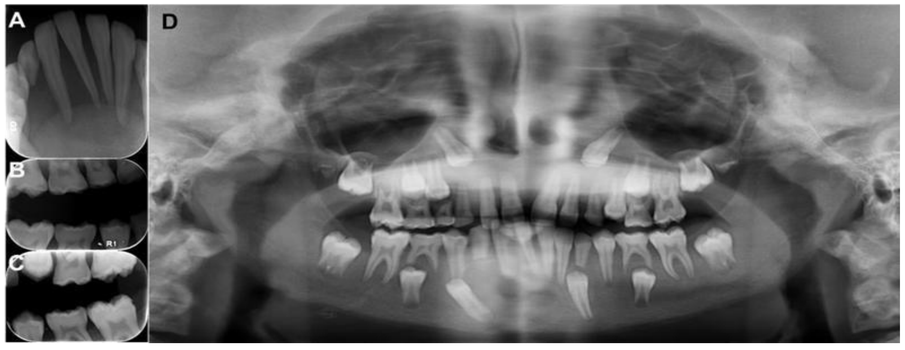

Figure 2: Intraoral periapical radiograph of mandibular anterior region (A), right (B) and left (C) bitewings, and panoramic radiograph (D).

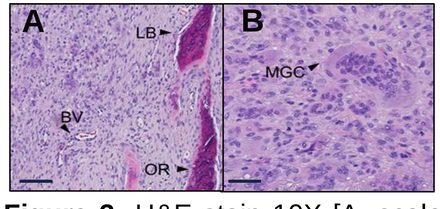

Figure 3: H&E stain 10X [A, scale bar 100µm, multinucleated giant cells (MGCs), blood vessels (BV), and lamellar bone (LB) with inconspicuous osteoblastic rimming (OR)]. 40X (B, scale bar 50µm).

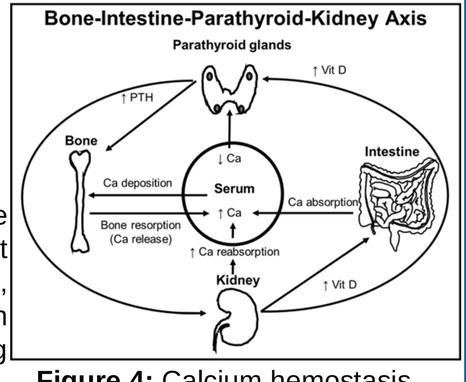

Figure 4: Calcium hemostasis

Discussion

- Bone-Intestine-Parathyroid-Kidney (BIPK) axis dysfunction contributed to bone resorption and hemorrhage, leading to BT.
- Renal osteodystrophy and metabolic bone disease weakened bone structure, increasing lesion size.
- Differential diagnosis for renal osteodystrophy, a generalized disease, includes rickets. In the jaws, the ground-glass appearance of the bone mimics craniofacial fibrous dysplasia.
- Radiographically and microscopically, the BT resembles central giant cell granuloma, giant cell tumor and aneurysmal bone cyst.
- Medical management (hemodialysis, calcimimetics, phosphate binders) aimed to prevent PTH elevation and lesion progression. Surgical excision was deferred, as medical stabilization may allow for partial or complete lesion regression.
- Early recognition of BT in pediatric ESRD patients is crucial to prevent craniofacial and dental deformities.

Conclusions

- Pediatric craniofacial BT is rare, with no standardized management protocols.
- Delayed diagnosis due to healthcare barriers led to an expansile jaw lesion consistent with BT.
- Early diagnosis and reporting of chronic renal disease and HPT is essential for multidisciplinary care and long-term follow-up.

References available upon request