Orbital Brown tumor: a case report and literature review

Tumor de Brown orbital: relato de caso e revisão da literatura

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ABSTRACT

The objective of this report is to describe a rare case of an orbital brown tumor secondary to hyperparathyroidism in a pediatric patient with end-stage renal failure and highlight the importance of timely multidisciplinary management. An 18-year-old male with end-stage renal failure and longstanding secondary hyperparathyroidism presented with progressive proptosis, diplopia, and globe dystopia. Imaging revealed an expansile lytic lesion of the right orbital roof compressing the globe and threatening multiple ocular structures including the optic nerve. Surgical excision preserved visual acuity at 6/9 bilaterally and restored extraocular movements. Histopathology confirmed a brown tumor. Subsequent total parathyroidectomy resulted in normalization of parathyroid hormone levels. Orbital brown tumors are rare but potentially vision-threatening. Early recognition and combined surgical management of the orbital lesion and underlying hyperparathyroidism can preserve vision and reduce morbidity in ESRF patients.

RESUMO

O objetivo deste relato é descrever um caso raro de tumor marrom orbitário secundário ao hiperparatireoidismo em um paciente pediátrico com doença renal em estágio terminal e destacar a importância do manejo multidisciplinar precoce. Paciente do sexo masculino, de 18 anos, com doença renal em estágio terminal e hiperparatireoidismo secundário de longa evolução, apresentou proptose progressiva, diplopia e distopia do globo ocular. Os exames de imagem evidenciaram uma lesão lítica expansiva no teto da órbita direita, comprimindo o globo ocular e comprometendo múltiplas estruturas oculares, incluindo o nervo óptico. A ressecção cirúrgica preservou a acuidade visual de 6/9 em ambos os olhos e restabeleceu a motilidade ocular extrínseca. O exame anatomopatológico confirmou o diagnóstico de tumor marrom. Posteriormente, foi realizada paratireoidectomia total, resultando na normalização dos níveis do hormônio da paratireoide (PTH). Os tumores marrons orbitários são raros, porém podem representar uma ameaça significativa à visão. O reconhecimento precoce e o tratamento cirúrgico combinado da lesão orbitária e do hiperparatireoidismo subjacente são fundamentais para preservar a visão e reduzir a morbidade em pacientes com doença renal em estágio terminal.

INTRODUCTION

Hyperparathyroidism (HPT) is an endocrine disorder caused by excessive parathyroid hormone secretion, leading to increased osteoclastic activity and abnormal bone remodeling. In rare cases, this results in brown tumors, which are non-neoplastic reactive bone lesions characterized by marked bone resorption, multinucleated giant cells, microfractures, hemorrhage, and hemosiderin deposition responsible for their brown coloration.[1]

The incidence of primary HPT (PHPT) ranges from 0.4 to 21.6 cases per 100,000 person-years and is most commonly due to parathyroid adenomas (≈ 80%), followed by hyperplasia (10 to 25%), multiple adenomas (≈ 5%), and rarely carcinoma (< 1%).[1,2] Osteoporosis is the most frequent skeletal manifestation (39 to 62.9%), while up to 20% of PHPT patients develop cystic bone lesions.[1,2] Secondary HPT (SHPT) is a common complication of advanced chronic kidney disease and affects up to 54% of hemodialysis patients.[3]

With improved screening, brown tumors have become uncommon, with an overall prevalence of approximately 0.1%.[1,2] They occur in up to 4.5% of PHPT patients and 1.5 to 1.7% of those with SHPT.[1,2] Lesions may involve any bone, most often long bones, pelvis, ribs, and clavicles, while facial involvement is rare (≈ 4.5%).[1,2] Radiographically, brown tumors appear as well-defined expansile lytic lesions with thin cortical margins and internal trabeculae and may mimic bone metastases when multiple.[1,2] Due to their rarity, particularly in SHPT and the facial skeleton, diagnosis may be delayed, leading to larger lesions at presentation.

The orbit is rarely affected, even less than the maxilla, mandible, maxillary sinus, and nasal cavity.[1,2]

The objective of this report is to describe a rare case of an orbital brown tumor SHPT in a pediatric patient with end-stage renal failure and highlight the importance of timely multidisciplinary management.

CASE REPORT

An 18-year-old male with SHPT due to ESRF, which resulted from bilateral vesicoureteral reflux and posterior urethral valves, was referred for painless facial asymmetry. He had been on continuous ambulatory peritoneal dialysis for nine years. His medical history included chronic rheumatic heart disease, hypertension, bilateral hip avascular necrosis, and multiple pathological fractures involving the right radius, ulna, and left femur. Secondary HPT had been diagnosed more than five years earlier, and he was treated with calcium carbonate, alfacalcidol, and subcutaneous erythropoietin, without improvement in anemia.

He reported progressive right proptosis and inferior globe dystopia for six months, with diplopia in primary gaze developing a few months after onset. On examination, he was underweight, with short stature. Visual acuity was 6/9 in both eyes, with no relative afferent pupillary defect. The right eye showed mild proptosis, inferior globe dystopia (Figure 1), and mild chemosis. Ocular motility was markedly restricted on upgaze. Intraocular pressure and fundus examination were normal bilaterally.



Figure 1. Clinical photograph showing the right eye with mild proptosis, inferior globe dystopia, and minimal conjunctival chemosis

Laboratory investigations showed normal serum calcium, persistently elevated phosphate (1.87 to 2.28 mmol/L), markedly elevated alkaline phosphatase (769 IU/L), and elevated parathyroid hormone levels (1,066 to 1,826 pg/mL), consistent with SHPT. Contrast-enhanced computed tomography revealed a benign-appearing expansile lytic lesion with internal calcifications at the right orbital roof, extending into the orbit and compressing the globe (Figure 2). The patient underwent multidisciplinary surgical resection involving the oculoplastic and oral and maxillofacial surgery teams (Figures 3 and 4). Histopathology showed cellular fibroblastic stroma with multinucleated giant cells and hemosiderin deposits, confirming the diagnosis (Figure 5).

Based on clinical, radiological, biochemical, and histopathological findings, a diagnosis of orbital brown



Figure 2. CECT of the brain and orbits demonstrates a benign-appearing expansile lytic lesion involving the right orbital roof, with multiple small internal calcifications (arrow)



Figure 3. Intraoperative finding of a hollow cyst with multiple bony deposits



Figure 4. Biopsy postsurgical resection

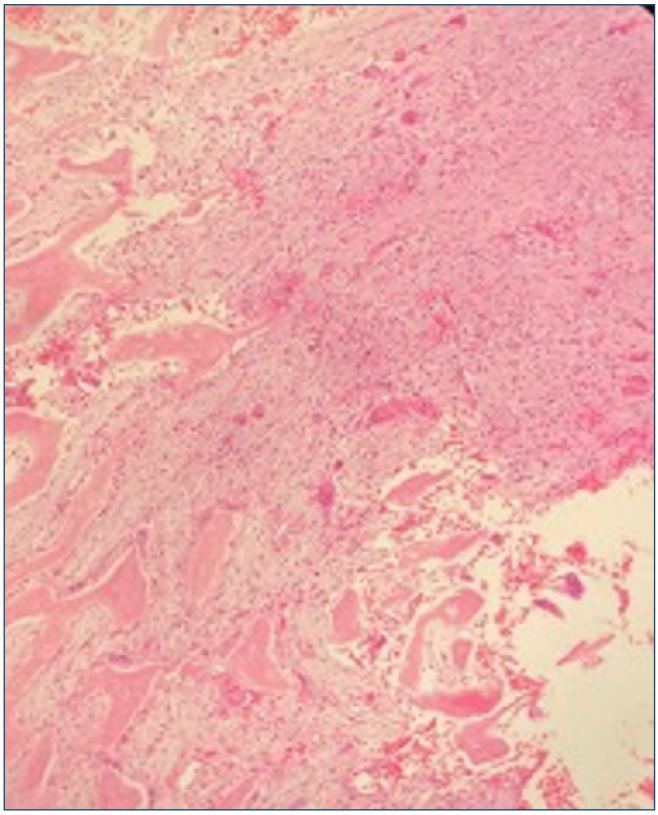


Figure 5. Biopsy showing cellular fibroblastic stroma with scattered multinucleated giant cells and hemosiderin deposits, consistent with brown tumor (hematoxylin and eosin, x10)

tumor secondary to SHPT was established. Surgical excision was followed by total parathyroidectomy in a separate setting. All four parathyroid glands were hyperplastic, measuring 2.0 × 1.1 cm to 3.5 × 2.0 cm.

Two months postoperatively, parathyroid hormone levels normalized to 51.8 pg/mL, although serum 25-hydroxyvitamin D remained deficient at 12.60 nmol/L.

Visual acuity remained 6/9 bilaterally, with full restoration of extraocular movements and resolution of proptosis, though mild inferior dystopia persisted, potentially requiring future orbital reconstruction (Figure 6).



Figure 6. Post parathyroidectomy with right orbital dystopia

This study was approved by the local ethics committee on March 16, 2026.

DISCUSSION

Only 4.5% of brown tumors involve facial bones, the most common bone being the mandible followed by the maxilla, and rarely, the orbit.[1,2] We reviewed ten cases of orbital brown tumors published in PubMed® from 2000 to 2025. Table 1 summarizes age, sex, presentation, management, and outcomes, if mentioned.[4-12]

Among the ten reported cases of orbital brown tumors published between 2000 and 2025 (Table 1), patient age ranged from 18 to 63 years, with a slight female predominance (6/10, 60%). Secondary HPT was the most frequent underlying etiology (7/10, 70%), while PHPT accounted for four cases (40%), most commonly due to parathyroid adenoma, with one case related to parathyroid carcinoma. Parathyroid hormone levels were markedly elevated in all cases with available data, ranging from 450 to 2,220 pg/mL. When mentioned, almost all of the cases with PHPT and SHPT had normal calcium and phosphate levels. There was only one case of hypercalcemia (3.00 mmol/L) that was associated with PHPT in a case of parathyroid carcinoma, and only one case with hyperphosphatemia (2.39 mmol/L) which was associated with SHPT. Alkaline phosphatase levels, when reported, were consistently elevated (191 to 1,928 IU/L), indicating high bone turnover.

Clinically, proptosis was reported in three out of ten cases (30%), while orbital or facial swelling was noted in five cases (50%). Diplopia occurred in three cases (30%), and optic nerve compression was documented in one case (10%). Anatomically, the lateral orbital wall (4/10; 40%) and medial orbital wall (4/10; 40%) were the most frequently involved orbital sites, followed by the orbital roof (3/10; 30%), orbital rim (2/10; 20%), and orbital floor (2/10; 20%). Contiguous involvement of orbital bones with adjacent craniofacial bones was observed in all ten cases. The most commonly affected extraorbital bones were the frontal bone (3/10; 30%), maxilla or maxillary sinus (3/10; 30%), and ethmoid bone or sinus (3/10; 30%), followed by mandible (2/10; 20%), sphenoid sinus or skull base (2/10; 20%), and zygoma (1/10; 10%). As for the management, 60% (6/10) of the cases underwent excision of the orbital brown tumor and among the cases with reported outcomes, there was improvement in terms of pain, size of swelling, proptosis, and diplopia. These findings highlight the aggressive expansile nature of orbital brown tumors and the importance of early recognition and multidisciplinary management to prevent advanced craniofacial involvement.

Primary HPT is caused by a solitary parathyroid adenoma in 80% of cases, whereas four-gland hyperplasia accounts for 10 to 15%, multiple adenomas for 5% and parathyroid cancer for < 1% of cases.[2,3] SHPT arises from progressive renal dysfunction, leading to impaired phosphate excretion, reduced vitamin D activation, hypocalcemia, and compensatory PTH overproduction. Additional contributors include fibroblast growth factor-23 overexpression and reduced calcium-sensing and vitamin D receptor expression in CKD patients.[13] Tertiary HPT is most often the result of a long-standing, severe SHPT, which has turned autonomous even when the cause of the SHPT has been removed.[14,15]

Brown tumors are histologically indistinguishable from other giant cell lesions, making biochemical correlation essential.[1-3] Elevated PTH levels are key to diagnosis.

Table 1. Summary of previous literature on orbital brown tumor published in PubMed from 2000 to 2025

Reference	Patient, age, year/sex	Primary or secondary hyperparathyroidism	Tumor location		Symptom	Blood investigation				Management	Outcome
			Orbit	Other		Calcium (mmol/L) Normal range: 2.2 to 2.7 mmol/L[3]	Phosphate (mmol/L) Normal range: 0.81 to 1.45 mmol/L[3]	Alkaline phosphate (IU/L) Normal range: 30 - 130 IU/L[3]	Parathyroid hormone (pg/mL) Normal range: 16.0 to 59.6 pg/mL[6]		
Zwick at al.[4]	29/M	Secondary	Orbital roof, Lateral orbital wall	Frontal bone	Swelling, pain, proptosis	-	2.39	1821	450	Cranio – orbitomy with surgical excision biopsy	Not stated
Monteiro[5]	40/F	Secondary	Orbital rim	Frontal bone	Swelling, proptosis	-	-	-	-	Surgical excision biopsy, parathyroidectomy	Orbital lesion reduced size, pain and proptosis resolved
Gonzalez – Martinez et al.[6]	30/F	Secondary	Orbital roof	Anterior cranial fossa	Proptosis	-	-	-	-	Surgical excision biopsy	Not stated
Cecchetti et al.[7]	24/M	Secondary	Orbital floor, medial & lateral wall	Maxillary bone, ethmoidal bone and sinuses, palate, zygoma, mandible	Not stated	2.245	1.34	1928	1987	Not stated	Not stated
	21/F	Secondary	Lateral wall	Maxillary bone and sinus, ethmoidal bone	Not stated	2.24	0.78	413	2220	Not stated	Not stated
Hannah et al.[8]	62/M	Primary: parathyroid carcinoma	Orbital floor, medial wall	Maxillary bone, ethmoid sinus	Diplopia Nasal blocked	3.00	0.53	191	880	Total parathyroidectomy offered but declined	Swelling regress, diplopia resolved
Borbolla-Pertierra et al.[9]	63/F	Secondary	Orbital rim	Nasal fossa	Painless swelling	2.49	-	722	1942	Surgical excision biopsy	Not stated
Alves at al.[10]	44/M	Primary : parathyroid adenoma	Infero medial wall orbit	Pelvis	Painless swelling	2.59	-	-	998	Surgical excision biopsy parathyroidectomy	Pain improved, swelling regress
Taddart et al.[11]	22/F	Primary : parathyroid adenoma	Lateral orbital wall	Frontal bone, temporal region, mandiblenbase of skull	Mandible swelling, painless, impaired chewing	1.65	1.43	-	1932	Surgical excision biopsy parathyroidectomy	Swelling regress
Faiz et al.[12]	42/F	Secondary	Medial orbital wall, compressing optic nerve	Sphenoid sinus, sella floor	Diplopia Fatigue	-	-	-	-	Not stated	Not stated

The management of brown tumor in SHPT is centered around controlling the underlying chronic kidney disease. This includes hemodialysis, dietary phosphorous restrictions, calcimimmetic drugs such as cinacalcet, vitamin D sterols, phosphate binders, bisphosphonates and intralesional steroid therapy.[13]

Although brown tumors may regress with medical therapy, early surgical excision is indicated in vision-threatening cases.[2,13,14] In retrospect, this patient would likely have benefited from earlier parathyroidectomy, given persistently elevated parathyroid hormone levels despite maximal medical therapy. Current guidelines recommend parathyroidectomy in ESRF patients with PTH levels >800 pg/mL for more than six months, particularly in the presence of parathyroid hyperplasia and erythropoietin-resistant anemia.[14,15] All of these criteria were met in this case. Earlier surgical intervention may have prevented tumor formation, pathological fractures, and subsequent morbidity.

Orbital brown tumors are exceedingly rare and often present with advanced disease. Ophthalmologists, particularly oculoplastic surgeons, play a critical role in diagnosis and management. Timely multidisciplinary intervention, addressing both the orbital lesion and underlying HPT, results in favourable visual and functional outcomes.

AUTHOR'S CONTRIBUTION

Ibrahim Suhaila and Muhammad-Saifullah Ahmad-Anwaar were involved in the clinical evaluation and management of the patient and prepared the initial draft of the manuscript. A-Rahim Haslinda and Ahmad-Alwi Azma-Azlina contributed to the surgical management of the orbital lesion and participated in manuscript revision. Ibrahim Suhaila and Muhammad-Saifullah Ahmad-Anwaar also conducted the literature review and contributed to the discussion. Ismail Shatriah provided overall supervision of patient care and manuscript development and performed the final critical review. All authors approved the final version of the manuscript and accept responsibility for its accuracy and integrity.

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