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BROWN TUMOR OF THE ORBITAL ROOF, ISOLATED MANIFESTATION OF SECONDARY HYPERPARATHYROIDISM: REPORT OF A CASE

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ABSTRACT

Introduction: Brown tumor is a non-neoplastic lesion, secondary to disturbance in bone metabolism in the context of the primary or secondary hyperparathyroidism. **Subject of the Study:** We report a clinical case to illustrate the rarity and highlight the clinical, therapeutic and evolutionary characteristics of this condition. **Observation:** it's Mr. AE 34 years old, followed in nephrology for chronic renal failure on hemodialysis, consult in ophthalmology for a progressive instalation of exophthalmia without effects on visual acuity. A cranial ct-scan Shows bone mass on the orbital roof, without optic nerve compression. An excisional biopsy of the mass was conducted. Histological examination was in favor of osteoblastic giant cell tumor. Laboratory tests showed hyperparathyroidism. **Discussion and Conclusion**: brown tumor is a secondary lesion to primary hyperparathyroidism or most often secondary to chronic renal failure. It mostly affects the ratings, pelvis, femur and mandible. Reaching the roof of the orbit is exceptional. A female predominance is noted. Before the non-specic histology, only the association of osteoclastic giant cells hyperparathyroidism can confirm the diagnosis. treatment of hyperparathyroidism alone can lead to the regression of the expansive process.

KEYWORDS: Brown tumor - hyperparathyroidism secondary- orbit.

INTRODUCTION

The brown tumor is a complication of hyperparathyroidism, which may be secondary most often to a parathyroid adenoma or rarely to chronic renal failure.[1] The diagnosis of the brown tumor is essential because the specific treatment of hyperparathyroidism can lead to the regression of the tumor process without surgical treatment.

OBSERVATION

H.E. is a 32-year-old patient, followed in the Nephrology Department for chronic renal failure on hemodialysis. He consulted in ophthalmology for a left exophthalmia of progressive evolution without diminution of the visual acuity nor repercussion on the ocular motricity.

A cranial CT scan (figure 1: expansive process at the roof of the left orbit) was made and showed bone mass at the roof of the orbit responsible for significant exophthalmia without optic nerve damage.

The patient underwent an excisional biopsy of the lesion, and the pathological study: osteoblastic giant cell tumor. Cervical ultrasound showed parathyroid hyperplasia. A biological assessment has shown high levels of parathyroid hormone, and phosphorus with hypocalcemia. A radiological assessment of the skeleton in search of another concomitant localization was made, and it proved normal. A medical treatment based on calcium and vitamin D, as well as phosphorus chelators was started. The evolution was marked by the regression of exophthalmia. Close monitoring has been advocated.

DISCUSSION

Secondary hyperparathyroidism is a common complication of chronic renal failure, and is often due to abnormal phosphocalcic metabolism.[1] The bone manifestations of hyperparathyroidism are usually late and rare, they are in the order of 5 to 15% of cases.[2] Hyperparathyroidism secondary to renal failure is usually responsible for diffuse bone demineralization rather than tumors.[3] The incidence of brown tumors is 1.5 to 1.7% in secondary hyperparathyroidism while it is 3% in primary hyperparathyroidism.[2] It mainly affects the sides, the pelvis, the femur and the mandible.[4] The orbital location is extremely rare.[5] The revealing clinical symptomatology is heterogeneous depending on the seat and the proximity of the nerve and muscle structures; exophthalmia, diplopia, pain, decreased mobility and visual acuity.[5] A complete ophthalmological examination to appreciate occulomotricity and visual acuity is necessary. A cranio-facial CT scan is requested systematically. It makes it possible to highlight the tumor process, its size, its extension and its relation to

neighborhood structures. Radiological assessment of the skeleton and technetium scintigraphy is often required to eliminate other concomitant lesions. A cervical ultrasound allows to objectify the parathyroid hyperplasia. Biological disturbances can often guide the diagnosis. Renal failure with high parathyroid hormone, hyperphosphoremia and hypocalcemia confirms the secondary nature of hyperparathyroidism. Pathologically, the brown tumor is formed by highly vascularized connective tissue with giant osteoclastic cells distributed around the foci of hemorrhage. The diagnosis is based on a bundle of biological and radiological and Histological arguments.[1]

Histological differential diagnosis occurs with giant-cell tumors, giant-cell central repair granuloma, cholesteatomas, adenoid cystic carcinomas of the lacrimal gland, osteosarcomas, and aneurysmal bone cysts.[5,6,7] Secondary hyperparathyroidism is usually controlled by a hypophosphoremic regimen and medical treatment including calcium, phosphorus chelators and active metabolites of vitamin D. Surgery may be necessary if medical treatment or severe form with very high parathormon levels associated or not with bone deformities or pathological fractures.[1]

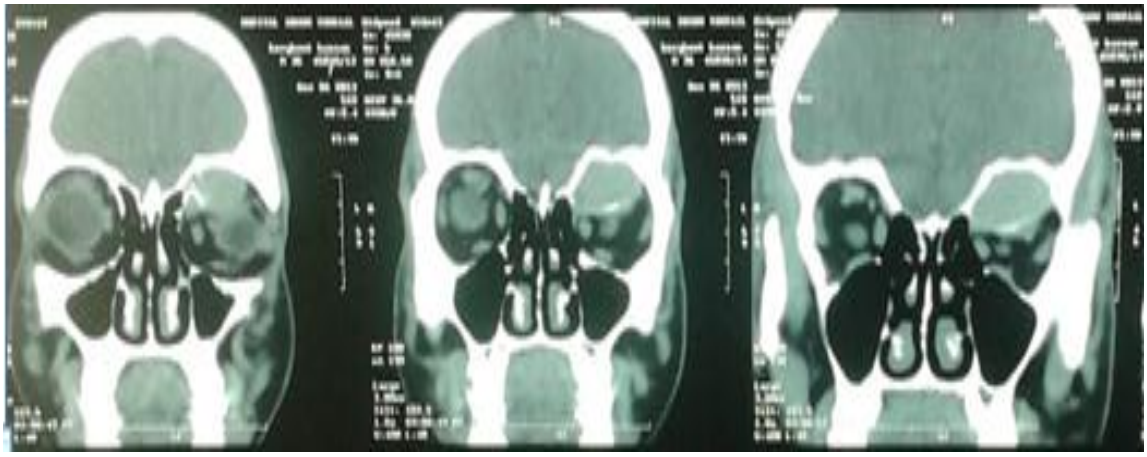

Figure 1: Expansive process at the roof of the left orbit.

CONCLUSION

The brown tumor of the orbit is an extremely rare manifestation of secondary hyperparathyroidism. A diagnostic delay can bring into play the functional prognosis of the eye. Treatment of hyperparathyroidism can lead to regression of the tumor. The use of surgery may be necessary for large symptomatic tumors.[5]

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