Aggressive jaw brown tumor in a 28-year-old man with long-lasted chronic kidney disease

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Brown tumors are bony lesions triggered by rapid osteoclastic activity, which rarely involved jaws (1-3). In fact brown tumors or osteoclastomas are erosive bony lesions appearing as a complication of hyperparathyroidism. Renal osteodystrophy is the result of secondary hyperparathyroidism and is associated with various pathogenetic mechanisms, such as disorder of calcium-phosphate metabolism, increased parathyroid activity that lead to extreme concentrations of parathormone and impaired metabolism of vitamin D (1-7).

We report a case of jaw enlargement in a 28-year-old patient with a ten years history of urinary obstruction due to multiple renal stones and history of chronic renal failure detected by laboratory records. One year before admission, the patient underwent bilateral DJ insertion. At the admission, patient had a GFR of 30 cc/min and low bilateral kidney size.

Three years before admission, isotopic renal scintigraphy revealed a severely decreased function (around 8%) of right kidney and around 10% reduced function of left kidney. A 66×23 mm of right kidney size and a 150×65 mm of left renal size with sever hydronephrosis, by sonography was detected.

According to the low kidney size, patients was not scheduled for renal biopsy and treatment with enalapril; 20 mg/day, allopurinol 50 mg/day and atorvastatin 10 mg/daily was started.

Two months after, patients returned to nephrology clinic with a new complain of jaw protrusion (Figure 1) which was supported by a cystic lesion of the jaw in MRI (Figure 2).

A conducted mandibular, anterior side incisional biopsy, detected a giant cell lesion with no evidence of malignancy. The result of laboratory test was as the follow; Serum creatinine; 3.9 mg/dl, intact PTH; 119 (10.9-54.8) pg/ml, calcium; 8.2 mg/dl, phosphorus 5 mg/dl and alkaline phosphatase was 466 IU/l. Vitamin D level; 35 ng/ml and serum calcitonine was 16.4 (0.2-27.7) pg/ml. Patient was decided to treat firstly by cinacalcet and then

Implication for health policy/practice/research/medical education

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Figure 1. Brown tumor of the jaw due to secondary hyperparathyroidism. A, B; Jaw protrusion. C; dental displacing by tumor.
we decided to refer him for surgical repair of teeth and tumor.

In patients with end-stage kidney failure, brown tumors are uncommon skeletal manifestations (1-5). Brown tumors are tumors of the bone that happen due to augmented osteoclastic activity, which are usually seen in severe forms of secondary hyperparathyroidism (5-8). Secondary hyperparathyroidism, which develops from phosphate retention and impaired calcitriol (1,25-dihyroxyvitamin D₃) synthesis in end-stage kidney failure, can cause increased osteoclastic activity, which may emerge as cysts in the bone and may ultimately progress to represent as brown tumors if left untreated (8-10). Brown tumors are defined in 1.5%–1.7% of patients with chronic kidney disease and happen most often in the pelvis, ribs, and mandible (7-12). Initial management involves the correction of hyperparathyroidism, which usually results to regression of the tumors (6-12).

Authors’ contributions
All authors contributed to paper equally.

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