



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

Saeed Mardani¹, Mohammad-Reza Tamadon², Heshmatollah Shahbazian³, Seyed Seifollah Beladi Mousavi³, Mohammad-Reza Ardalan⁴, Hamid Nasri^{5*}

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

Brown tumors are bony lesions triggered by rapid osteoclastic activity, which rarely involved jaws. Brown tumors are tumors of the bone that happen due to augmented osteoclastic activity, which are usually seen in severe forms of secondary hyperparathyroidism.



Figure 1. Brown tumor of the jaw due to secondary hyperparathyroidism. A, B; Jaw protrusion. C; dental displacing by tumor.

Received: 12 September 2014, Accepted: 4 November 2014, ePublished: 20 January 2015

¹Department of Nephrology, Shahrekord University of Medical Sciences, Shahrekord, Iran. ²Department of Nephrology, Semnan University of Medical Sciences, Semnan, Iran. ³Chronic Renal Failure Research Center, Ahvaz Junishapur University of Medical Sciences, Ahvaz, Iran. ⁴Chronic Renal Failure Research Center, Tabriz University of Medical Sciences, Tabriz, Iran. ⁵Department of Internal Medicine, Isfahan University of Medical Sciences, Isfahan, Iran.

*Corresponding author: Prof. Hamid Nasri, Email: hamidnasri@yahoo.com

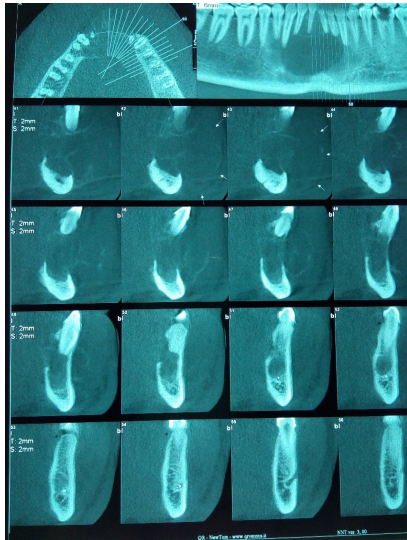


Figure 1. Cystic lesion of the jaw

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-dihydroxyvitamin 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.

Ethical considerations

Ethical issues (including plagiarism, informed consent, misconduct, double publication and redundancy) have been completely observed by authors.

Conflict of interests

The authors declared no competing interests.

Funding/Support

No funding from any source.

References

1. Nabi Z, Algailani M, Abdelsalam M, Asaad L, Albaqumi M. Regression of brown tumor of the maxilla in a patient with secondary hyperparathyroidism after a parathyroidectomy. *Hemodial Int* 2010; 14(2): 247-9.
2. Pechalova PF, Poriázova EG. Brown tumor at the jaw in patients with secondary hyperparathyroidism due to chronic renal failure. *Acta Medica (Hradec Kralove)* 2013; 56(2): 83-6.
3. Pinar Sumer A, Arik N, Sumer M, Karagoz F. A rare complication of secondary hyperparathyroidism. Brown tumor of the maxilla and mandible. *Saudi Med J* 2004; 25(12): 2010-2.
4. Nasri H, Baradaran A, Naderi AS. Close association between parathyroid hormone and left ventricular function and structure in end-stage renal failure patients under maintenance hemodialysis. *Acta Med Austriaca* 2004; 31(3): 67-72.
5. Zangeneh F, Clarke BL, Hurley DL, Watts NB, Miller PD. Chronic Kidney Disease-Mineral and Bone Disorders (CKD-MBDs): What the Endocrinologist Needs to Know. *Endocr Pract* 2014; 20(5): 500-16.
6. Khouzam NM, Wesseling-Perry K, Salusky IB. The role of bone in CKD-mediated mineral and vascular disease. *Pediatr Nephrol* 2014 Aug 29.
7. Nasri H. Linkage of elevated CaxPO4 product with inflammation in maintenance hemodialysis patients. *Minerva Urol Nefrol* 2006; 58(4): 339-45.
8. Vervloet MG, Massy ZA, Brandenburg VM, Mazzaferro S, Cozzolino M, Ureña-Torres P, et al. Bone: a new endocrine organ at the heart of chronic kidney disease and mineral and bone disorders. *Lancet Diabetes Endocrinol* 2014; 2(5): 427-36.
9. Pinto LP, Cherubinim K, Salum FG, Yurgel LS, de Figueiredo MA. Highly aggressive brown tumor in the jaw associated with tertiary hyperparathyroidism. *Pediatr Dent* 2006; 28(6): 543-6.
10. Lamb EJ, Delaney MP. Does PTH Offer Additive Value to ALP Measurement in Assessing CKD-MBD? *Perit Dial Int* 2014 2; 34(7): 687-91.
11. Goldsmith DJ, Massy ZA, Brandenburg V. The Uses and Abuses of Vitamin D Compounds in Chronic Kidney Disease-Mineral Bone Disease (CKD-MBD). *Semin Nephrol* 2014; 34(6): 660-8.
12. Bover J, Ureña P, Brandenburg V, Goldsmith D, Ruiz C, DaSilva I, et al. Adynamic Bone Disease: From Bone to Vessels in Chronic Kidney Disease. *Semin Nephrol* 2014; 34(6): 626-40.

Please cite this paper as: Mardani S, Tamadon MR, Shahbazian H, Beladi Mousavi SS, Ardalan MR, Nasri H. Aggressive jaw brown tumor in a 28-year-old man with long-lasting chronic kidney disease. *J Parathyroid Dis* 2015; 3(1): 8-9.
Copyright © 2015 The Author(s); Published by Nickan Research Institute. This is an open-access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.