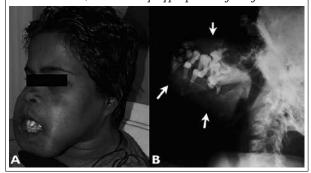
Jaw enlargement in a haemodialysis patient

E. Hoornenborg¹*, F. van Delft^{1,2}, L. Vogt³

¹Department of Internal Medicine, Drs. L. Mungra Streekziekenhuis, Nieuw Nickerie, Suriname, ²Department of Gastroenterology & Hepatology, VU University Medical Center, Amsterdam, the Netherlands, ³Department of Internal Medicine, Nephrology Division, Academic Medical Center, University of Amsterdam, Amsterdam, the Netherlands, *corresponding author: e-mail: elske.hoornenborg@zonnet.nl

A 38-year-old woman presented to our district hospital in Suriname (South America) because she was unable to close her mouth. Physical examination demonstrated severe facial deformity with bony protrusion of both the maxilla and mandible (figure 1A). Oral inspection showed mandibular and palatal exophytic swelling and displacement of teeth. The patient's medical history was significant for end-stage renal disease of unknown origin and 11 years of adequately dosed haemodialysis treatment. She also had secondary hyperparathyroidism complicated by pelvic and hip deformations. Her laboratory tests revealed mild hypocalcaemia (2.20 mmol/l, normal value 2.10-2.55 mmol/l), hyperphosphataemia (1.76 mmol/l, normal value 0.8-1.4 mmol/l) and an extremely high parathyroid hormone value (336 pmol/l, normal value 2-7 pmol/l). Facial radiography confirmed bony enlargement of the jaws (figure 1B; arrows indicate boundaries).

Figure 1. Enlarged maxilla and mandible with inability to close mouth, due to bony hyperplasia of the jaws



WHAT IS YOUR DIAGNOSIS?

See page 233 for the answer to this photo quiz.

Netherlands The Journal of Medicine

ANSWER TO PHOTO QUIZ (PAGE 229)

JAW ENLARGEMENT IN A HAEMODIALYSIS PATIENT

DIAGNOSIS

A diagnosis was made of severe bony deformation with hypertrophia of the jaws caused by uncontrolled secondary hyperparathyroidism.

Secondary hyperparathyroidism is a common complication in chronic renal failure. It is caused by disruption of the calcium and phosphorus homeostasis. The rise of parathyroid hormone results in renal osteodystrophy with different clinical manifestations such as osteitis fibrosa or osteomalacia. Mixed bone disease often develops, characterised by increased number and activity of osteoblasts with marked bone turn-over, in conjunction with proliferation and differentiation of osteoclasts leading to superimposed mineralisation defects. Thus, fractures and bone deformities develop as late consequences.¹

Treatment consists of reduction of dietary phosphorus intake, oral phosphate binders, vitamin D derivatives,

calcimimetics or parathyroidectomy. The last two treatments are, however, not widely available in middle-income countries such as Suriname due to high costs and limited expertise in neck surgery, respectively. Although better access to dialysis in middle-income countries significantly improves survival for renal insufficiency, adequate management of dialysis-associated hyperparathyroidism remains cumbersome due to lack of resources.

REFERENCE

 Cunningham J, Locatelli F, Rodriguez M. Secondary hyperparathyroidism: pathogenesis, diseases progression, and therapeutic options. Clin J Am Soc Nephrol. 2011;6:913-21.