

Neck swelling secondary to severe hyperplasia of autotransplanted parathyroid tissue following parathyroidectomy

Drasko Pavlovic,^a Lidija Orlic,^b Hrvojka Tomic-Brzac,^c Nikola Pavlovic,^d Vlado Petric^e

From the ^aDepartment of Nephrology and Dialysis, Sestre Milosrdnice University Hospital, ^bDepartment of Nephrology and Dialysis, University Hospital Rijeka, ^cDepartment of Nuclear Medicine and Radiation, University Hospital Zagreb, ^dDepartment of Medicine and ^eEar, Nose, Throat and Neck Surgery, Sestre Milosrdnice University Hospital, Croatia

Correspondence and reprints: Drasko Pavlovic MD · Department of Nephrology and Dialysis, Sestre Milosrdnice University Hospital · Vinogradska c. 29 Zagreb 10000 Croatia · T: +3851-378-7526 F: +3851-376-9067 · drasko.pavlovic@bol-svduh.htnet.hr · Accepted for publication June 2008

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Secondary hyperparathyroidism, characterized by an increase in the synthesis and secretion of parathyroid hormone (PTH) and parathyroid hyperplasia, is a very common complication of chronic kidney disease.¹ Despite significant advances in medical therapy, in 5% to 10% of patients with chronic renal failure, parathyroidectomy (PTX) may be indicated.^{2,3} Unfortunately, recurrent or persistent hyperparathyroidism can be seen in up to 10% of patients after PTX.^{4,5} Our case was a 44-year-old woman with recurrent hyperparathyroidism after total PTX with autotransplantation into the sternocleidomastoid muscle, who presented with an enlarged neck structure. She had chronic renal failure due to chronic glomerulonephritis and was on hemodialysis. Four years later, total PTX with autotransplantation in a single area into the ster-

nocleidomastoid muscle was performed. Seven years after the surgery, the intact PTH level was 2630 pg/mL (normal range, 6-62 pg/mL), and total alkaline phosphatase was 682 IU/L (normal range, 60-170 IU/L). The patient refused a new operation. Two years later an enlarged structure in the neck was observed (Figure 1). At that time, intact PTH was 2632 pg/mL and total alkaline phosphatase was 818 IU/L. By ultrasound, five enlarged structures suspected of having hyperplastic parathyroid tissue were found; a small one (dimension 0.9×0.9×0.8 cm) with uniform echoes and four larger ones with a nodular pattern of hyperplasia. The largest one, with dimensions of 2.5×1.3×2.2 cm was detected subcutaneously and by palpation, corresponding to the nearby enlarged structure, the left sternocleidomastoid muscle. We assumed that the largest nodule was



Figure 1. Enlarged neck structure, near the left sternocleidomastoid muscle.

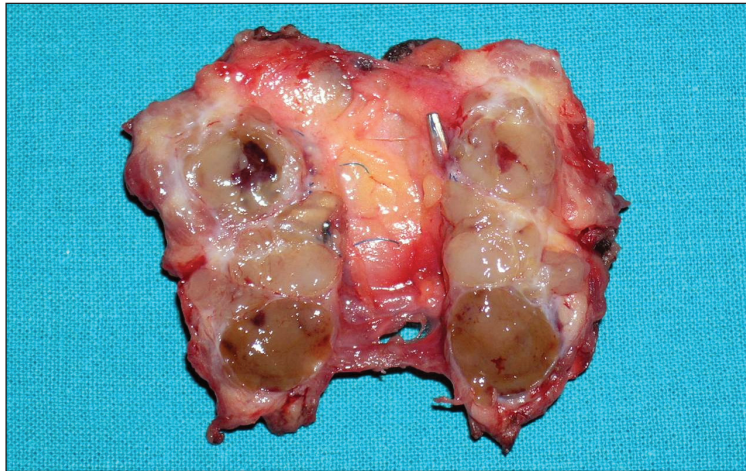


Figure 2. Single large dissected autotransplanted parathyroid nodule.

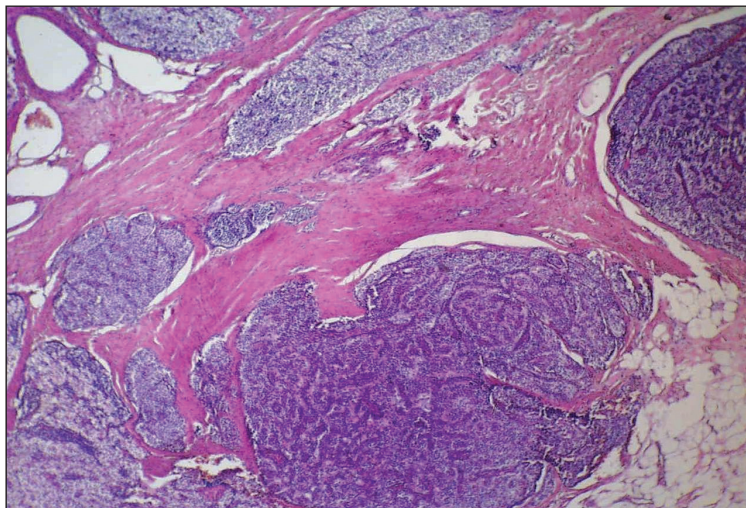


Figure 3. Parathyroid nodular hyperplasia dispersed between muscle cells (hematoxylin-eosin $\times 40$).

a consequence of hyperplasia of the autotransplanted parathyroid tissue while the other hyperplastic nodules were a consequence of the scattered parathyroid cells. Proliferation of parathyroid cells was confirmed by fine needle biopsy of the largest nodule. Surgery was performed and all enlarged structures, i.e. parathyroid tissue, were removed (Figure 2). Histopathological ex-

amination showed nodular hyperplasia of parathyroid tissue, including the largest one (Figure 3), and diffuse hyperplasia of the smallest one. One month later, intact PTH was 374.2 pg/mL and total alkaline phosphatase did not change significantly (929 IU/L). Unfortunately, a year later her PTH was 656.4 pg/mL and therapy with paricalcitol was started.

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