Acute Hypoparathyroidism after Percutaneous Fine-Needle Ethanol Injection (PFNEI) in a Patient on Haemodialysis

P.L.	Pier Luigi	Bedani ^a
L.	Luciano	Feggi ^b
N.	Napoleone	Prandini ^b
P.	Paolo	Gilli ^a

^aDivision of Nephrology and ^bInstitute of Nuclear Medicine, Santa Anna Hospital, Ferrara, Italy

Dr. Pier Luigi Bedani, Division of Nephrology, S. Anna Hospital, Corso Giovecca 203, I-44100 Ferrara (Italy)

Dear Sir,

Percutaneous fine-needle ethanol injection (PFNEI) is a valid procedure for the treatment of secondary hyperparathyroidism (sHPT) in uraemic patients undergoing chronic dialysis, particularly when parathyroid hyperactivity recurs after subtotal para-thyroidectomy (PTX) [1-3]. The side-effects of PFNEI are limited (remitting local pain, transient dysphonia, light parathyroid swelling owing to oedema or haematoma) and usually do not promote acute changes in serum calcium and phosphorus concentrations, because the correction of sHPT is slow and progressive [2, 3].

We report a case of acute hypoparathyroidism after PFNEI of an enlarged parathyroid gland in a haemodialyzed patient who had been previously submitted to subtotal PTX.

Case Report

A 38-year-old woman on RDT for 35 months because of chronic glomerulonephritis had been submitted to PTX for severe sHPT. Histological study had confirmed the removal of three parathyroid glands, i.e. the two apical and the lower left. The day after the operation, the patient had a transient light hypocalcaemia favourably corrected by oral calcium and calcitriol administration. A few months later, the patient experienced musculoskeletal pains, symptoms of polyneuropathy, persistent hyperphosphataemia, which could not be corrected by overdoses of aluminium-containing phosphate binders, and very elevated serum alkaline phosphatase (1,280 U/l; reference interval 99-310 U/l).

Fig. 1. Acute changes in the serum PTH (•), calcium (Ca, □), phosphorus (P, ■) and alkaline phosphatase (ALP, O) after the second percutaneous ethanol injection.

Sonographic study of the anterior region of the neck showed an enlarged gland on the right basal side.

An ultrasonically guided fine-needle aspiration biopsy was performed, but histological study indicated, erroneously, the presence of thyroid tissue. Some months later, with persisting bone pain and intractable pruritus, due to hyperphosphataemia (7.5-8.2 mg/dl, 2.5-2.7 mmol/l; reference intervals 2-5 mg/dl, 0.7-1.7 mmol/l) and elevated Ca × P product (75-80 mg/dl), the patient underwent an

MNR of the neck which showed the presence of a lower right enlarged parathyroid gland. A further fine-needle aspiration biopsy was performed. The cytological examination confirmed

hyperplastic parathyroid tissue. The patient was submitted to PFNEI according to the technique of Giangrande et al. [2]. A week after the first injection of 0.8 ml of 95% ethanol, no significant decrease of serum PTH was proved, so a second ethanol injection was performed. The total volume of absolute ethanol injected was 1.6 ml. Some hours after the

last injection, the patient experienced mild dysphonia and local transient pain, and about 36 h later she reported diffuse paresthesia and shivering. Serum calcium concentration was 6.8 mg/dl (1.7 mmol/l; reference interval 8.5-10.5 mg/dl, 2.1-2.6 mmol/l), ionized calcium 0.82 mmol/l (reference interval 1.12-1.30 mmol/l), phosphataemia 3.7 mg/dl (1.2 mmol/l), and serum intact PTH was un-detectable. Ultrasonographic examination of the lower right parathyroid gland showed that its volume had enlarged from 2 to 5 cm³ probably due to an acute reactive oedema. The dosage of oral calcitriol was increased gradually (from 1.0 to 3.0 µg/day), and every day the patient received an infusion of CaCl₂ at an appropriate dosage. This treatment was necessary also in the later period of haemo-dialysis or shortly after dialysis. Some weeks later, after an important therapy with a daily oral administration of 3.0 µg calcitriol and 4 g calcium carbonate, serum calcium concentrations returned to within the normal range, and the serum PTH level increased slightly (fig. 1). The serum alkaline phosphatase, which had dramatically increased a week after the second ethanol injection, fell to its initial value (fig. 1), indicating that the 'hungry bone' was partially repleted. Ultrasonically, gland volume appeared significantly decreased (from 5 to 2.5 cm³) with a marked echogenicity, probably due to induced fibrosis.

This case allows us to make the following comments. First, PFNEI may be a very effective alternative to the surgical approach or administration of high-dose 1,25(OH)₂D₃ in uraemic patients with sonographically verified single or multiple enlarged parathyroid glands [3]. Second, its side-effects are usually of limited clinical importance and well endured [2,3], but it is possible that acute chemical inhibition of parathyroid activity produces profound and sustained hypocalcaemia, particularly when there is radio-graphic evidence of bone resorption. For reasons that are uncertain, tetanic seizures most often occur during the later period of haemo-dialysis or shortly after it, when the degree of hypocalcaemia is undoubtedly less marked than at other times [4]. In our opinion, it is thus prudent to observe the following procedure: (a) careful monitoring of serum calcium (total and ionized), phosphorus and PTH in the immediate post-PFNEI period until the degree of hypocalcaemia is established; (b) no administration of phosphate-binding antacids if serum phosphorus falls below 3.0 mg/dl (1 mmol/l); however, serum phosphorus should not be allowed to increase above 3.5-1.0 mg/dl (1.2-1.3 mmol/l) because of the risk of aggravating the hypocalcaemia; (c) early administration, some days before the PFNEI, of high calcitriol and calcium carbonate supplements, particularly in patients with severe sHPT and fibrous osteitis [5], (d) and finally, temporary use of dialysate that contains higher calcium concentrations (to 2 mmol/l) in order to balance the diffusion of the ion during dialytic treatment particularly in those patients who have a highly reduced skeletal pool of calcium.

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