Case Report

Autoinfarction of the parathyroid gland diagnosed by power Doppler ultrasonography in a patient with secondary hyperparathyroidism

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Introduction

Spontaneous remission due to parathyroid infarction of secondary hyperparathyroidism (SHPT) [1,2] is very rare compared to that of primary hyperparathyroidism [3,4]. It is possible that the diagnosis of parathyroid infarction is missed in patients with SHPT because parathyroid infarction often occurs in only one of the enlarged glands. Lately, neck ultrasound examination has become a more beneficial and specific method for the diagnosis of enlarged parathyroid glands, in contrast to classic diagnostic techniques such as computed tomography (CT), magnetic resonance imaging (MRI) and scintigraphy [1,2]. The diagnosis of parathyroid infarction reported in previous studies, however, was often based on CT, MRI and scintigraphy findings and only a few studies have reported such diagnosis by emergency power Doppler ultrasonography of the neck.

Here, we present a haemodialysis patient with autoinfarction of the left parathyroid gland diagnosed by emergency power Doppler ultrasonography of the neck. The spontaneous infarction of the parathyroid gland was associated with a small to moderate decrease in serum calcium and parathyroid hormone (PTH) values, but PTH remained markedly elevated.

Case report

The patient was a 35-year-old woman who had been on peritoneal dialysis since 1974 for chronic renal failure caused by chronic glomerulonephritis. She received renal transplantation from her mother in 1975. However, renal function gradually deteriorated and she had undergone thrice-weekly haemodialysis since 1993. In February 2004, the patient was found to have a high serum PTH concentration (919 pg/ml; intact-PTH; Nicols Institute Diagnostics; San Clemento, CA; normal range, 14–66 pg/ml; 14–66 ng/l), a high bone metabolic marker and two enlarged parathyroid glands with hypervascularity by power Doppler ultrasonography. Moreover, a significantly low bone mineral density was detected. Since persistently high intact-PTH and high calcium levels were noted in spite of 15 mg/week of intravenous maxacalcitol treatment (normal dose range of maxacalcitol; 7.5–30 mg/week), she was scheduled for parathyroidectomy (PTx) in 2004, with a diagnosis of SHPT refractory to medical treatment.

In August 2004, the patient reported mild pain in the left neck and ultrasonography of the neck was immediately performed. As shown in Figure 1, the size and volume of the left parathyroid gland (10.0 × 12.8 × 11.6 mm; 772 mm³) was significantly larger than before this episode (8.0 × 10.1 × 9.5 mm; 399 mm³). The size of the right parathyroid gland (8.8 × 10.5 × 9.8 mm; 471 mm³) did not change before and after this episode. The volume of the parathyroid glands was computed by three-dimensional measurement using the formula (π/6 × a × b × c), where a, b, and c represent the three dimensions of the gland on ultrasonography, as described previously [5]. The hypervascular findings of the left enlarged parathyroid gland detected earlier changed to hypovascular findings by power Doppler ultrasonography, suggesting...
autoinfarction of the left parathyroid gland. A CT scan of the neck revealed a low-intensity 1 cm tumour in the lower left parathyroid gland, but no hot spot was detected in either gland by 99mTc-201Tl scintigraphy. These results indicated autoinfarction of the left parathyroid gland.

Soon after this episode, serum calcium concentration precipitously decreased from 10.1 to 8.2 mg/dl and persistently low serum calcium and phosphorus concentrations were noted thereafter. The spontaneous infarction of the parathyroid gland was associated with a small to moderate decrease in serum calcium and PTH values, but PTH remained markedly elevated. However, with a 4-fold increase in the dose of maxacalcitol to 60 μg/week, the intact-PTH concentrations diminished to 302 pg/ml (302 ng/l) and serum calcium levels increased in late October 2004 (Figure 2). In October 2004, power Doppler ultrasonography revealed a significant reduction in the size and volume (3.2 × 7.0 × 4.7 mm; 55 mm³) of the left parathyroid gland but no change in the right parathyroid gland (Figure 1).

Discussion

Although about 30 cases of spontaneous remission of primary hyperparathyroidism due to necrosis of parathyroid adenoma have been reported [3,4], spontaneous remission due to parathyroid infarction of SHPT is rare compared to that of primary hyperparathyroidism. One possibility is that a parathyroid adenoma is more vulnerable to infarction than parathyroid hyperplasia. Another possibility is that the presence of two equal sized glands in SHPT could mask the clinical presentation of a spontaneous infarction of one gland.

Previous studies [1,2] of parathyroid infarction in primary hyperparathyroidism and SHPT reported marked falls in serum calcium and PTH values and although marked hypocalcaemia developed, PTH values did not increase and remained quite low. These patients often presented with neck pain or other associated clinical findings. Based on a review of the pathological findings, Nylen et al. [6] classified the pattern of parathyroid haemorrhage/infarction into two types, moderate intracapsular haemorrhage/infarction and massive extracapsular haemorrhage/infarction. However, our present case would seem to be different from previous reports [1–4,6] of parathyroid infarction in primary hyperparathyroidism and SHPT because of the minimal biochemical changes that accompanied the clinical presentation.

Here, we report the emergency and follow-up ultrasonographic findings of parathyroid infarction in SHPT. The most interesting finding in this case was that one of the two enlarged glands underwent autoinfarction while the remaining gland continued to secrete PTH. In the presented case, parathyroid gland infarction was associated with a moderate decrease in serum calcium and a small decrease in PTH, but PTH values remained extremely high (>600 pg/ml). While part of the increased PTH could be from stimulation by hypocalcaemia, the PTH value of >600 pg/ml shows a relatively intact secretory apparatus. Spontaneous infarction of one parathyroid gland was documented in this patient only because of the use of power Doppler ultrasound; otherwise,
it would have been missed if diagnosis was solely based on clinical presentation.

It is important to document the incidence of two simultaneous predominant hyperplastic glands in SHPT. Tominaga et al. [7] and Ellis [8] showed a marked asymmetry of the parathyroid gland weight on PTx in patients with SHPT. Ellis [8] reported that six of 14 patients with SHPT have two approximately equal size parathyroid glands. Similar conclusions could be drawn from a study [9] in which ethanol was directly injected into the parathyroid glands with refractory SHPT. Kitaoka et al. [9] reported that five of nine patients with SHPT had two enlarged parathyroid glands by ultrasonography. In almost all these patients (four of five patients) who had two enlarged parathyroid glands, ethanol injections into two glands were necessary to achieve the target PTH level, suggesting that the marked fall in serum PTH level was not always induced by a single moderate intracapsular infarction. Based on these reports [7–9], we suggest that our case may not be uncommon and without a power Doppler ultrasound study, the parathyroid autoinfarction could have been easily missed.

In conclusion, it could be suggested that spontaneous infarction of a parathyroid gland may occur more commonly than previously thought in SHPT, and unless sophisticated imaging studies are performed, it could be missed clinically.

Conflict of interest statement. None declared.

References


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