Dear Sir,

Total parathyroidectomy (tPTX) with autotransplantation (AT) has been widely performed as a standard operative procedure for secondary hyperparathyroidism (HPT) in dialysis patients. One of the main problems associated with this procedure is the gradual development of recurrent HPT. Recently, ethanol injection to parathyroid masses has been performed in patients with primary or secondary HPT [1-4]. We report a patient with autograft-dependent recurrent HPT who was successfully treated by percutaneous ethanol injection to the autograft.

A 59-year-old man underwent tPTX with AT for secondary HPT in 1987, 11 years after starting hemodialysis. All 4 excised glands showed hyperplasia. For AT, 10 pieces measuring $1 \times 1 \times 2$ mm were cut out of the smallest gland and implanted into the left forearm. Three months later, serum carboxy-terminal parathyroid hormone (PTH-c) decreased from 48.5 to 1.6 ng/ml (normal: < 1.3).

However, over a period of 40 months, serum PTH-c increased gradually to 4.4 ng/ml and serum calcium (Ca) before hemodialysis was elevated to 6.5 mEq/l (normal: 4.5-5.5). Serum alkaline phosphatase (ALP) was also elevated to 302 U/l (normal: 94-292). Ultrasound, computed tomography and combined $^{201}$Tl and $^{99m}$Tc scan did not demonstrate the existence of parathyroid glands in the neck or mediastinum. Moreover, there was a gradient of intact PTH between the two arms (left: 519, right: 152 pg/ml). According to these findings, he was diagnosed as having autograft hyperfunction and underwent a surgical excision of 3 enlarged autografts in May 1990. All excised autografts showed hyperplasia. After the excision, serum PTH-c, Ca and ALP transiently decreased to 2.4 ng/ml, 5.0 mEq/l and 237 IU/l, respectively, but showed subsequent increases.

On September 11, 1991, subtotal gastrectomy with Billroth II reconstruction was per-
formed for early gastric cancer. In predialysis laboratory tests before gastrectomy, serum Ca was 5.6 mEq/l, serum phosphorus was 5.8 mg/dl (normal: 2.5-4.3), serum ALP was 520 IU/l, serum PTH-c was 8.3 ng/ml, and serum albumin was 4.5 g/dl (normal: 3.5-5.0). Three days after the operation, he was diagnosed as having a subphrenic abscess. Food and liquids were withheld, and he received intravenous hyperalimentation containing 8.5 mEq per day of Ca. He was kept at bed rest throughout the day. For 2 weeks, serum Ca increased to 7.3 mEq/l (fig. 1). This increase in serum Ca, which was considered to be caused by immobilization in the presence of recurrent HPT, could not be ameliorated by changing the solutions to ones containing no Ca or administration of eel calcitonin.

In the left forearm, only 1 parathyroid fragment with a well-defined boundary was detected under ultrasound and its size was measured at 21 × 16 x 8 mm. There was a significant gradient of intact PTH between the left (5,280 pg/ml) and the right forearms (141 pg/ml). We performed ultrasonically guided ethanol ablation under local anesthesia. For ultrasound guidance, a 7.5-MHz transducer (ASU-32WS, Aloka Company, Tokyo, Japan) with a lateral puncture adaptor was used. We used a commercially available 22-gauge needle, 7 cm in length, for the ethanol injection. Ethanol injection was performed on October 3 and 7, and the volume of absolute ethanol injected was 0.5 and 0.6 ml, respectively. No pain or hematoma developed at the site of injection. Serum Ca became within the normal range 2 days after the second injection, and 1 month later, the levels of serum PTH-c in the right forearm and intact PTH in the left forearm decreased to 2.5 ng/ ml and 279 pg/ml, respectively. Moreover, the parathyroid fragment was reduced in size to 10 × 7 × 3 mm 4 months after the injections.

A surgical removal of some autografts has so far been the only therapeutic method for autograft-dependent recurrent HPT. In this letter, we report an ultrasonically guided percutaneous ethanol injection to a parathyroid autograft as a new treatment.

In the present patient, ethanol injection to a parathyroid autograft could be easily performed under local anesthesia with no adverse effects. Moreover, the ethanol injection could immediately reduce the levels of serum Ca and PTH, and later, the size of the parathyroid autograft significantly decreased. With regard to safety and effectiveness, ethanol ablation may be in no way inferior to surgical removal.

The great advantage of ethanol ablation is that the volume of parathyroid tissue and the level of serum PTH can be gradually reduced by repeated injection of a small amount of ethanol. In this patient, surgical removal might lead to hypoparathyroidism because he had only 1 enlarged parathyroid fragment in the forearm. If 2 or more enlarged autografts are seen, an effective treatment must include percutaneous ethanol ablation of each lesion step by step. On the other hand, ethanol ablation has a few disadvantages. First, it is suitable only for patients with ultrasonically detectable autografts. Second, it is impossible to confirm the histology of the autografts. Since a few authors [5, 6] have reported patients with transplantation of parathyroid tissue subsequently diagnosed as having parathyroid
carcinoma, ethanol ablation should be limited to patients having autografts with a well-defined boundary under ultrasound.

References

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Takeda/Michigishi/Takazakura Percutaneous Ethanol Injection to Parathyroid Autografts