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Seven Cases of Subtotal Parathyroidectomy for Renal Hyperparathyroidism*)

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ABSTRACT

Seven patients under maintenance hemodialysis were performed the subtotal parathyroidectomy from January 1979 to August 1984. Their ages ranged from 8 to 49 years. The primary renal disease included chronic glomerulonephritis in 4 patients, polycystic kidneys in 2 patients and renal aplasia in one patient. We preserved 20 to 180 mg of parathyroid tissue on intact vascular pedicle. All histology of these glands showed parathyroid hyperplasia.

The PTH levels were elevated in all patients preoperatively from 1.1 to 35.4 times the upper limit of normal. Postoperatively, these values dramatically decreased in all but one patient. All of these six patients had a decrease in serum calcium levels as well as resolved clinical symptoms. Roentgenographic improvement also occurred in all of these six patients.

INTRODUCTION

The enlarged parathyroid glands associated with chronic renal failure were first recognized by Albright et al.[1] in 1934, Pappenheimer and Wilens[12] in 1935 and Castleman and Mallory[5] in 1937. It was not an important clinical problem before the dialysis era, since patients usually died of the renal disease without any symptom of renal hyperparathyroidism. In these 20 years, however, hemodialysis prolonged the lives of many of these patients, and the hyperparathyroid state certainly became a clinical problem in the dialysis population.

Secondary hyperparathyroidism associated with chronic renal failure is related to disordered calcium and phosphorus metabolism rusulting from changes in parathyroid hormone (PTH) secretion and vitamin D metabolism.

Stanbury et al.[13] reported subtotal parathyroidectomy performed upon patients with renal disease in 1960. The clinical results were encouraging. They recommended subtotal parathyroidectomy to reduce the mass in the functioning parathyroid gland, thereby enabling serum calcium and phosphorus levels to return to normal. This procedure also allowed the oral administration of calcium and vitamin D with subsequent skeletal remineralization and correction of the osteomalacia. The frequency of severe secondary hyperparathyroidism with the associated skeletal and soft tissue manifestations in patiends with chronic renal failure was also recognized by Wilson et al.[15]. They confirmed the validity of subtotal parathyroidectomy as a means of preventing the progression

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of osteitis fibrosa. The later series of Geis et al.[11] and Blake et al.[2] also gave support for this operation.

In this paper, we report seven cases of the surgical treatment of renal hyperparathyroidism.

PATIENTS MATERIALS AND METHODS

The indications for operation were clinical disease with laboradory confirmation of secondary hyperparathyroidism or biochemical abnormalities that progressed despite aggressive medical management. According to our criteria, seven patients, under maintenance hemodialysis, 5 males and 2 females, were performed the subtotal parathyroidectomy from January 1979 to August 1984 (Table 1). Their ages ranged from 8 to 49 years. The cause of renal failure was glomerulonephritis (4 patients), polycystic kidneys (2 patients) and renal aplasia (1 patient). The length of time dialysis ranged from 2 to 10 years.

Our usual surgical procedure is to use a transverse collar incision with subplatysmal flap dissection and midline splitting of the cervial strap muscles. The thyriod gland is mobilized anteromedially. Recurrent laryngeal nerves are identified and protected. A systemic research for parathyroid tissue is then began. Four glands were found in 4 patients, five glands in 2 patients, and three glands in one patient. The removed parathyroid tissue weighed 0.47 to 5.42 g. We preserved 20 to 180 mg of parathyroid tissue on intact vascular pedicle in 7

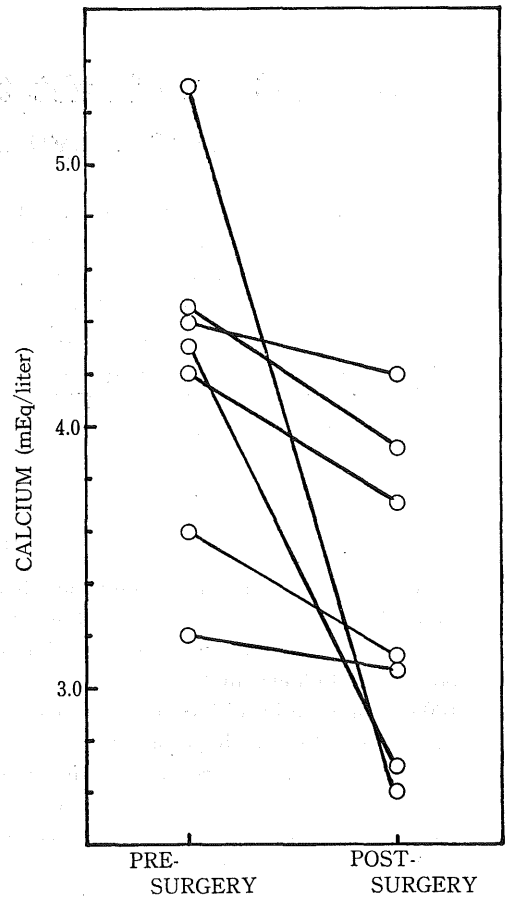

Fig. 1. Serum calcium levels presurgery and post (one month)-surgery in seven patients with renal hyperparathiroidism. Normal value: 4.3 to 5.5 milliequivalents per liter.

Table 1. Clinical data on seven patients requiring subtotal parathyroidectomy

Patient No.	Age (yrs)	Sex	Chief Complaint	Cause of Renal Failure	Duration of Dialysis (yrs)	Operation	Wt. of Extirpated Glands (g)	Wt. of Residual Gland (g)
1.	16	Male	Deformity of lower extremities.	CGN*	6	3.5 gland excision	0.90	0.03
2.	8	Male	Deformity of extremities.	Renal aplasia	2	3.5 gland excision	0.47	0.06
3.	33	Male	Arthralgia and deformity of chest	CGN*	10	3.5 gland excision	2.20	0.18
4.	41	Male	Deformity of chest	CGN*	6	3.5 gland excision	2.50	0.06
5.	35	Female	Arthralgia	Polycystic kidney	2	2.5 gland excision	1.15	0.05
6.	34	Male	Bone pain and deformity of chest	CGN*	7	4.5 gland excision	5.42	0.02
7.	49	Female	Arthralgia	Polycystic kidney	8	4.5 gland excision	2.68	0.05

*CGN; Chronic glomerulonephritis.

patients. The tissue removed was verified by frozen section. Some tissue routinely cryopreserved for the potential of hypoparathyroidism. All histology of these glands showed parathyroid hyperplasia.

We allowed preoperative and postoperative oral administration of calcium lactate (3.0 to 12.0 g/day), 1α-OH-D_3 (1.0 to 2.0 μg/day), and $Al(OH)_3$ (3.0 to 9.0 g/day).

Values of the serum calcium, ionized calcium, phosphorus, ALP and PTH (c-terminal) were determined preoperatively and one month postoperatively.

RESULTS

Biochemical observation:

The serum calcium and phosphorus levels preoperatively and postoperatively of 7 patients are shown in Figs. 1 and 2. The preoprative calcium levels were normal in 5 of 7 patients. We have no patients with markedly elevated serum calcium levels. The postoperative calcium levels declained in all of 7 patients. The preoperative phosphorus values were elevated in 3 of 7 patients (Fig. 2). The postoperative phosphorus levels decreased in 5 of 7 patients. However, one patient showed almost no change within the normal range, and the value of phosphorus remained to be elevated in another patient. Changes in serum ionized calcium is shown in Fig. 3. Values of ionized calcium were determined in 3 patients. The postoperative levels of ionized calcium markedly decreased in all of these three patients.

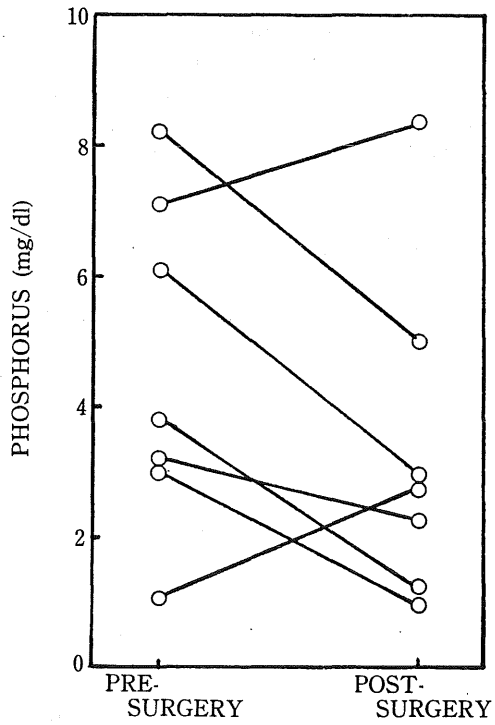

Fig. 2. Serum phosphorus levels presurgery and post (one month)-surgery in seven patients with renal hyperparathyroidism. Normal value: 2.5 to 4.5 milligrams per deciliter.

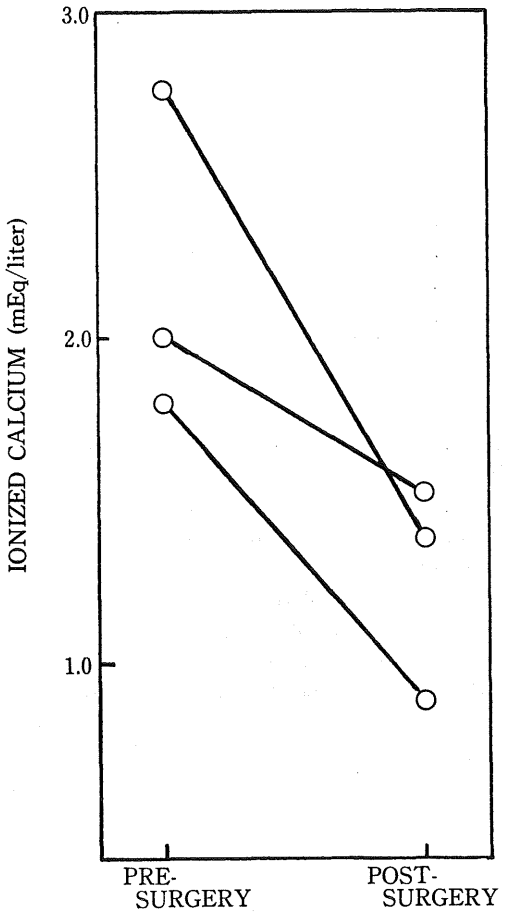

Fig. 3. Serum ionized calcium levels presurgery and post (one month)-surgery in three (Patient No. 1, 2, 3) with renal hyperparathyroidism.

The PTH levels were elevated in all patients preoperatively from 1.1 to 35.4 times the upper limit of normal (Table 2). Postoperatively, these values dramatically decreased to 0.08 to 1.23 times the upper limit of normal in all but one patient. It is interesting that all of these six patients had a decrease in serum calcium levels

Table 2. Correlation between PTH levels and clinical obsevation

Patient No.	Preop. PTH (ng/ml)	Postop. PTH (ng/ml)	Clinical Observation
1.	1.36	0.16	improved
2.	8.56	0.10	improved
3.	9.84	0.95	improved
4.	29.5	0.60	improved
5.	40.8	8.40	unchanged
6.	40.1	0.40	improved
7.	46.0	1.60	improved

as well as resolved clinical symptoms. Preoperative PTH values were compared to the size of the parathyroid glands determined by linear measurement, and a poor correlation was found. PTH values were also compared with serum calcium levels, phosphorus levels, severity of clinical disease and duration of dialysis. No definite correlations could be established among these sets of variables.

Preoperative ALP levels were elevated in 4 patients from 1.50 to 31.6 times the upper limit of normal. Values of ALP were normal in 3 patients. Postoperative values of ALP were almost unchanged in all of 7 patients.

Clinical observation:

Clinical bone disease was present in 6 patients who had roentgenographic evidence of hyperparathyroidism. Roentgenographic improvement occurred in all of these six patients. Bone pain was present in 7 patients and was relieved postoperatively in 6 patients, was unchanged in one patient (Patient No. 5) (Table 2). Four patients (Patient No. 3, 4, 6, 7) had spontaneous fractures of ribs preoperatively. Following operation, their fractures healed, and no further spontaneous fractures occurred.

Vascular calcifications were present in one patient (Patient No. 4) preoperatively and there were unchanged postoperatively.

Major operative complications was not observed in all cases.

DISCUSSION

It has been well known that for nearly a half century that high PTH levels and parathyroid hyperplasia are the common findings of the hyperparathyriod state in chronic renal failure[5,13]. The pathogenesis of secondary hyperparathyroidism is not well understood. However, two general mechanisms has been proposed. As reviewed by DeLuca[7], defects in vitamin D metabolism are present in renal failure. Reduction of renal mass is resposible for lesser quantities of 25-hydroxycholecalciferol being hydoxylated to 1, 25-dihydroxycholecalciferol which is the predominant active form of vitamin D in the intestine. Bricker et al.[3,4] described a retention of phosphorus due to decreasing nephron population, resulting in increasing serum phosphorus levels with a reciprocal decrease in ionized serum calcium and excessive stimulation of PTH secretion. The high PTH levels found in renal failure may be, in part, due to the decreased breakdown and clearing of PTH molecules[10].

PTH levels were not influenced by the age and sex of the maintenace hemodialysis patients but they are elevated as the period of hemodialysis became longer[2]. The correlation between the level of PTH and secondary hyperparathyroidism was good in our cases. Diethelm et al.[8] reported that the duration of renal impairment as well as the length of time dialysis was thought to have a direct relationship to the incidence of secondary hyperparathyroidism.

The severity of bone disease varies from one patient to another who has renal disease. Not all of these patients will require an operation on the parathyriod glands, since in some, the bone disease will reverse with medical therapy[2,9]. Therefore, the indication for operation are clinical disease with laboratory confirmation of secondary hyperparathyroidism or biochemical abnormalities that progress despite aggressive medical management.

The classical approach to diffuse parathyroid hyperplasia has been complete removal of all but one of the parathyroid glands and subtotal resection of this last gland, as described by Cope[6]. The results with this procedure were

satisfactory in the present series, except for one patient. These results are consistent with Geis's report[11]. The advantage of this approach was that no permant hypoparathyroid state were created. Blake et al.[2] reported that the standard subtotal parathyroidectomy, leaving 30 to 60 mg of tissue in the neck, has been quite satisfactory and effective. We also leaved 20 to 180 mg of the gland in neck.

In 1973, Geis et al.[11] and in 1975, Wells et al.[14] published the early results of total parathyroid excision with autografting of parathyroid tissue in patients with renal hyperparathyroidism. However, the procedure cannot as yet be recommended for general use because of its potetial disadventages. Immediately postoperatively, all patients require calcium supplementation for several weeks while the autograft becomes established. Furthermore, failure of the implant to be vascularized could lead to permanent hypoparathyroidism. Geis et al.[11] and Blake et al.[12] reported that subtotal parathyroidectomy will remain the operation of choice for diffuse parathyroid hyperplasia.

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