Case Report

Hypercalcemia due to Spontaneous Hemorrhage in a Parathyroid Adenoma during Recovery from Acute Renal Failure

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A 89-year-old woman showed transient and mild hypercalcemia (11.0 mg/dl) at the phase of recovery from acute renal failure. Serum PTH level elevated to 2.1 ng/ml when hypercalcemia was observed, and decreased to 1.1 ng/ml. Autopsy study revealed the bleeding within parathyroid adenoma. The transient and mild hypercalcemia seen in the patient was considered as the result of the bleeding within parathyroid adenoma. This unusual manifestation of parathyroid adenoma, bleeding, usually causes hypercalcemic crisis. The mild hypercalcemia observed in this patient despite of bleeding into parathyroid adenoma might be due to old age of the patient and the existence of renal failure in the patient.

Key Words: Parathyroid adenoma, Hemorrhage, Renal failure, Hypercalcemia, Aged

A rare subsequence of parathyroid adenoma, the spontaneous hemorrhage within adenoma, usually results in hypercalcemic crisis because of a tremendous release of parathyroid hormone into blood stream1-3).

We report here a first case of the spontaneous hemorrhage within a parathyroid adenoma without hypercalcemic crisis, but with mild hypercalcemia. The mild hypercalcemia in this case was observed during recovery phase from acute deterioration of chronic renal failure due to hyperpotassemic sinus arrest. We will discuss the pathogenesis of the mild and transient hypercalcemia in relation to renal failure and the hemorrhage within a parathyroid adenoma.

REPORT OF A CASE

A 89-year-old woman was referred to Tokyo metropolitan Geriatric Hospital on January 20th in 1983 for evaluation of progressive gait disturbance and weight loss of 25 kg in last 7 years. The careful intake of history failed to reveal any other significant symptoms related to hypercalcemia except for serious constipation.

Her body weight and body height was 52 kg and 134 cm, respectively. Her pulse was 72 beats per minute, blood pressure 160/92 mmHg, body temperature 36.2°C and respiration 18 per minute. No remarkable physical findings were observed except for generalized muscle weakness and atrophy with pathological reflexes in her extremities.

Neither apparent tumor nor tenderness in her neck could be detected. X-ray examination of her neck showed cervical spondylosis, and initial laboratory examination revealed that she had a moderate renal dysfunction (blood urea nitrogen; BUN; 46 mg/dl, serum creatinine; 2.0 mg/dl). Serum calcium level, phosphorus level and alkaline phosphatase activity were 9.3 mg/dl, 3.4 mg/dl and 34 IU, respectively.

Serum creatinine levels and BUN increased gradually and reached to 4.4 mg/dl and 75 mg/dl, respectively on 48th hospital day. At the same time, anuria and hyperpotassemia (serum K; 7.0

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mEq/L) were observed. Her electrocardiograms showed sinus arrest with ventricular escape beats. Her blood pressure fell to 70/42 mmHg and 2 hours urine output decreased to below 10 ml. After peritoneal dialysis for the following two days, serum K levels were normalized (4.8 mEq/L), and her urination was restored.

On 50th hospital day, hypercalcemia of 11.0 mg/dl was noticed. Higher serum immunoreactive PTH level (2.2 ng/ml) than that of the age matched control (less than 0.6 ng/ml6), was detected.

Forty-eight hours later (52nd hospital day), serum calcium levels decreased to 10.3 mg/dl without specific treatment against hypercalcemia except for infusion of 2000 ml Haltman solution per day. Serum PTH level decreased also to 1.1 ng/ml. Serum calcium levels remained to be within normal range until she died on 105th hospital day.

Fig. 1. A cross-up view of the cut surface of the adenoma with bleeding of parathyroid gland (left upper part).

The upper part and the lower part of the adenoma are composed of oncocytoma and chief cell adenoma of parathyroid, respectively.

Fig. 2. A lower field view of the bleeding within the chief cell adenoma.
The final clinical diagnosis at death was chronic renal failure secondary to arteriosclerotic contracted kidney.

Autopsy study demonstrated an adenoma, sized 1.0 x 0.8 cm, in the left upper parathyroid gland. The adenoma consisted of two parts histologically, upper part; oncocytoma, lower part; trabecular and partly follicular benign chief cell adenoma with fresh and old bleeding within adenoma (Figs. 1 and 2). Another two parathyroid glands were found at autopsy, contained oncocytoma composed of large oxiphyllic cells.

Fig. 3. A high power field view of the chief cell adenoma.
This part of the adenoma is composed of large water-clear cells arranged in trabecular and partly follicular pattern.

Fig. 4. A high power field view of the oncocytoma.
This part of the adenoma is composed of large oxiphilic cells. The abundant cytoplasm filled with very fine oxiphilic granules, and the dark-staining cytoplasm contrast with the pale-staining cytoplasm of the chief cell adenoma (Fig. 3).
Neither fibrotic ostitis nor nephrocalcinosis was observed. No malignant neoplasma which could be a cause of hypercalcemia was demonstrated. Arterioarteriolosclerotic contracted kidney (weight of right and left kidney was 65 and 70 g, respectively) was found. Therefore, the final pathological diagnosis of the patient was as follows; 1) parathyroid chief cell adenoma with bleeding into adenoma (Figs. 1–3). 2) parathyroid oncocytoma (Fig. 4). 3) arterioarteriolosclerotic kidneys. 4) cervical spondylosis with myelopathy (C3–C4).

**DISCUSSION**

We reported here a rare case who had mild and transient hypercalcemia inspite of the presence of spontaneous hemorrhage within parathyroid adenoma. Serum PTH levels elevated at the time of hypercalcemia, and decreased in accordance with the normalization of serum calcium levels. This observation suggested that PTH, probably released from destructed parathyroid adenoma by spontaneous hemorrhage, played a causal role for hypercalcemia.

Spontaneous hemorrhage within parathyroid adenoma was reported to cause hypercalcemic crisis\(^1\)–\(^5\). However, only mild hypercalcemia (serum calcium level; 11 mg/dl) was observed in this patient. It has been well-known that tissue responsiveness to PTH decreased with advancing of age\(^7\),\(^8\). Refractoriness for PTH was also demonstrated in the state of chronic renal failure\(^9\)–\(^13\). Thus age and renal failure of this patient might explain mild hypercalcemia regardless of high serum PTH levels induced by hemorrhage within parathyroid adenoma.

Hypercalcemia has been reported to occur in a variety of clinical situations associated with renal failure. These include the hypercalcemia that occurs in “uncomplicated” chronic renal failure, the hypercalcemia that follows hemodialysis for chronic renal failure, the hypercalcemia after renal transplantation and the hypercalcemia at the phase of diuresis of acute renal failure resulting in rhabdomyolysis\(^14\). In these cases, hypersecretion of PTH may not be responsible for the observed hypercalcemia, because, the PTH levels have not been consistently elevated\(^15\),\(^16\).

Thus, renal failure itself and the recovery of diuresis in this patient were not considered as a main causative factor of her hypercalcemia.

The microscopical examination showed old and fresh hemorrhage in parathyroid adenoma. This finding indicates that the hemorrhage in adenoma might occur at the phase of the recovery of anuria.

In conclusion, massive secretion of stored PTH might have occurred during the hemorrhage into adenoma of the parathyroid, which caused mild hypercalcemia but not hypercalcemic crisis because of the refractoriness to PTH due to the presence of renal failure and of her old age.

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**REFERENCES**


