Ventricular Tachyarrhythmia Treated by Parathyroidectomy in a Chronically Hemodialyzed Patient

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Dear Sir,

Cardiac arrhythmias have been frequently documented in patients with chronic renal failure receiving hemodialysis. Although several probable factors have been proposed for the arrhythmias, the pathogenesis has remained unclear.

We have examined the incidence and contributory factors of cardiac arrhythmias in chronically hemodialyzed patients by 72-hour continuous Holter ECG monitoring [1], and revealed that in the frequent ventricular arrhythmia group the percent fractional shortening of the left ventricle was significantly lower than those in the no arrhythmia group. In the frequent arrhythmia group, the values of the serum calcium concentration times those of phosphorus, which are thought to be an index of parathyroid function, were significantly higher than those of patients without ventricular arrhythmias. Thus we concluded that impaired cardiac performance and impaired calcium and/or phosphate metabolism may contribute to the pathogenesis of premature ventricular contractions, (PVCs). During our Holter ECG monitoring investigations, we experienced an interesting case whose ventricular arrhythmias were improved following parathyroidectomy.

A 43-year-old man was admitted to our hospital with secondary hyperparathyroidism and ventricular arrhythmias in December 1985. Fifteen years earlier in 1971, nephrotic syndrome was diagnosed and the patient received corticosteroids for 2 years. In 1973, chronic renal failure was diagnosed, and he began to receive regular hemodialysis. Although he sometimes complained of palpitations and pulse deficit during hemodialysis and ventricular arrhythmias were documented, antiarrhythmic agents were not administered. Thereafter, his azotemia had been well controlled by regular hemodialysis 12 h a week for 12 years. However, in October 1985, he began to suffer from a sharp pain in both knees and ankles on walking.

In December 1985, he was admitted to our hospital for parathyroidectomy and evaluation of the ventricular arrhythmias. On admission, he appeared well except for joint pain while walking. Physical examination showed typical uremic pigmentation over the entire skin. Skeletal survey revealed bone changes typical of secondary hyperparathyroidism, such as a 'pepper pot' skull, 'rugger jersey' spine and subperiosteal resorption on the phalanges. Laboratory data showed the presence of uremia (BUN 89 mg/dl, creatinine 14.0 mg/dl, Na 138 mEq/l, K 5.7 mEq/l,

hemoglobin 10.9 g/dl), and of secondary hyperparathyroidism (Ca 9.4 mg/dl, Pi 7.0 mg/dl, Al-P 745 mU/ml [normal value: < 230 mU/ml], the isozyme of Al-P being predominantly that of bone origin). The c-PTH level measured by radioimmunoassay was 56.1 ng/ml (normal value: < 0.5 ng/ml). Ultrasonic examination of the parathyroid glands revealed areas of apparent enlargement. Subtraction scintigram revealed maximal accumulation of radioisotope in all of four glands. ECG showed a regular sinus rhythm and normal corrected QT intervals (0.38 s). There were no abnormalities in ST, T waves to indicate the presence of coronary disease. Echo-cardiogram revealed normal cardiac function, and the left ventricular contraction was normal (percent fractional shortening 48%). The dimension of the left ventricle was 48 mm in diastole and 25 mm in systole, that of the left atrium 32 mm and that of the aortic root 30 mm. The thickness of the intraventricular septum and the left ventricular posterior wall were normal (10 and 10 mm, respectively). Valvular motion appeared normal. Examination of ventricular arrhythmias by 72-hour continuous Holter ECG monitoring revealed sporadic PVCs, 231 beats 24 h on a nonhemodialysis day, and frequent PVCs, 711 beats 24 h, on a day of hemodialysis. Frequency of ventricular arrhythmias increased markedly during hemodialysis and for 3 h afterwards. The patient underwent total parathyroidectomy; partial minced parathyroid gland was implanted over the right sterno-cleidomastoideus. Operative findings con-

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firming the existence of parathyroid hyperplasias in 3 of the 4 glands. Benign parathyroid hyperplasia was confirmed histologically. Transient hypocalcemia following the operation was treated by infusion of a sufficient amount of CaCl₂ together with oral administration of 5 µg of 1α-(OH)D₃. On the 7th day following the operation, 72-hour continuous Holter ECG monitoring was repeated. Only one ventricular arrhythmia was detected on the day of hemodialysis and no arrhythmias on the nonhemodialysis day. Plasma concentration of calcium was 7.1 mg/dl before hemodialysis and 9.2 mg/dl after hemodialysis; serum phosphate was 5.2 mg/dl before hemodialysis and 2.7 mg/dl afterwards. The plasma concentration of c-PTH was markedly depressed to 1.8 ng/ml. Although the calcium level decreased markedly, ECG revealed normal corrected QT intervals (0.37 s) before hemodialysis without any change of ST, T waves. On the 30th day following the operation, 72-hour continuous Holter ECG monitoring was performed again, and no arrhythmia was detected on both days of hemodialysis and nonhemodialysis, when PTH concentration was normalized to 0.4 ng/ml and calcium concentration had been maintained above 8.5 mg/dl.

Findings of the presented case and findings of previous Holter monitoring investigations [1] made us consider that PTH might be an arrhythmogenic factor in uremia. Ramirez et al. [2] also reported supportive data of our thesis of a significantly higher concentration of c-PTH in patients with cardiac arrhythmias than in patients without arrhythmias, suggesting that PTH might induce arrhythmias in uremic patients. It can also be argued that calcium is an arrhythmogenic agent [3], especially in cases of hypercalcemia with digitalis administration where it is thought that ventricular arrhythmias tend to be easily provoked [4]. In the present case, however, even after parathyroidectomy, serum concentrations of calcium were well maintained at values greater than 7.5 mg/dl by means of intravenous infusion of CaCl₂ and the oral administration of 1α-(OH)D₃. Thirty days after surgery, the Holter ECG monitoring procedure was repeated; PVCs were not detected when the PTH concentration returned to 0.4 ng/ml and the calcium

concentration had been maintained above 8.5 mg/dl. Thus, serum calcium itself might not be the major contributory factor in causing PVCs in uremia.

We therefore conclude that PTH may act as an arrhythmogenic agent in uremic patients.

References

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