Dear Sir,

We report on a 31-year-old dialysis patient who was successfully resuscitated after ventricular fibrillation (VF). The patient underwent parathyroidectomy (PTX) 8 weeks before the described event. This report suggests that autotransplantation (ATP) of parathyroid tissue is not always effective in preventing cardiac arrhythmias due to hypocalcemia.

A 31-year-old man with Alport syndrome had a 5-year history of renal failure, treated by peritoneal dialysis until May 1993 and by hemodialysis three times a week since May 1993. There was a 3-year history of recurrent tissue calcifications that had obliged the patient to undergo three surgical interventions to remove calcified tissue tumors from the right shoulder and his fingers despite therapy with phosphate binders. In May 1993, PTX with ATP of parathyroid tissue was performed. Since then the patient described a strong improvement in his tissue calcification. Calcium (3 × 2 g/day) and vitamin D substitutes (3 × 0.25 mg/day) were given orally, but the patient’s compliance with therapy was known to be poor. Eight weeks after PTX, the patient lost consciousness due to cardiac arrhythmia. His brother being present started cardiopulmonary resuscitation (CPR). On arrival, the emergency physician found the patient unconscious and cyanotic with a systolic arterial pressure of 70 mm Hg. The ECG monitor showed sinus arrhythmia with about 60 beats/min with multifocal ventricular extrasystoles. The T wave was elevated with prolongation of the Q-T interval. An intravenous line was established and the patient intubated and ventilated with 100% oxygen. Two grams of calcium gluconate was given which reduced the number of ventricular extrasystoles. Suddenly the VF appeared 10 min after intubation, and CPR was immediately performed. The VF did not respond to four defibrillations by an external defibrillator, 200 mg lidocaine, and 1.5 mg epinephrine. CPR was performed during 30 min, while calcium substitution and glucose/insulin infusion were continued. Thereafter, the patient had the ECG...
shown in figure 2. Hemodialysis was continued and the patient extubated 2 h later. Calcium substitution was continued over 3 days at the concentration indicated above to maintain a serum value between 1.8 and 2.2 mmol/l. After 1 week the patient was discharged in good condition. Serum calcium and parathormone levels were normal.

When cardiac arrhythmias in uremic patients occur, it seems that always another metabolic condition, such as severe hypocalcemia or hypomagnesemia, is present together with hyperkalemia [1]. Incidence studies on hyperkalemia alone [2] show that there is no uremic patient among those who died from malignant arrhythmias, but patients with acute renal failure in whom potassium levels were normal 36 h before. Uremic patients had an even higher kalemia (potassium > 7 mmol/l) than the patients who died from arrhythmia, and there were no ECG changes found in these patients.

Hypocalcemia is a common phenomenon in uremic patients. Secondary hyper-parathyroidism is responsible for the fact that hypocalcemia is usually mild and without clinical symptoms. A normal response to hypocalcemia after PTX with ATP by an increase in parahyroid hormone levels has been described [3]. However, persistent hypocalcemia despite normal or elevated parathyroid hormone levels has been found in one fourth of the patients after PTX with ATP for secondary hyper-parathyroidism [4].

Ionized calcium is known to play a critical role in normal cardiovascular function. Acidosis seems to prevent arrhythmias in patients with hypocalcemia by increasing the ionized fraction of total serum calcium. Metabolic acidosis is always present in cardiorenal failure.

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Table 1. Serum parameters on admission to the intensive care unit

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<th>Component</th>
<th>SI</th>
<th>Conventional</th>
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<tr>
<td>Potassium</td>
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<tr>
<td>Sodium</td>
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<td>Calcium (total)</td>
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<td>Urea</td>
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<td>Creatinine</td>
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<td>Glucose</td>
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piratory arrest. One can speculate that in the case presented respiratory compensation of the acidosis by hyperventilation after intubation may have further decreased the ionized fraction of the low total serum calcium level and thus triggered VF. This feature stresses the danger of antagonizing metabolic acidosis by sodium bicarbonate. It has been shown [5] that sodium bicarbonate does not have any effect on hyperkalemia in uremic patients. It has to be considered contraindicated in this situation.

CPR in VF due to hypocalcemia has been described as refractory to defibrillations in hypocalcemic patients [6]. It is reasonable to limit the number of defibrillations because of the potentially dangerous effects on the myocardium. CPR must be continued while high-dose calcium is given intravenously. Acidosis should be tolerated until cessation of VF. Hemodialysis may be necessary to normalize hyperphosphatemia and hyperkalemia [7].

To our knowledge, VF after PTX in secondary hyperparathyroidism has not yet been reported. This case suggests that ATP of parathyroid tissue is not always effective in preventing cardiac arrhythmias due to hypocalcemia.

References

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