Letter to the Editor

NEPHRON

Nephron 1998;79:381–382

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Successful Transitory External Cardiac Electrostimulation in a Patient with Excessive Hyperkalemia and Bradycardia

Dear Sir,

We present the case of a young hypertensive patient (35 years old, body weight 73.5 kg) with insulin-dependent diabetes mellitus diagnosed 18 years ago and multiorgan complications related to diabetes such as retinopathy with advanced chronic renal failure, metabolic 'renal' acidosis, blindness, and excessive hyperkalemia (9.0 mmol/l).

The blood glucose concentration and arterial pressure were not satisfactorily controlled, 1 year ago (HbA1c >7.5%, mean arterial pressure varies between 118 and 144 mm Hg). At the same time, chronic azotemia was detected (creatinine blood concentration 150 μmol/l). During the last few months, the chronic azotemia progressed to advanced uremia.

Because of repeated bilateral intravitreal hemorrhages, the patient was treated four times in Moscow, Russia (Fjodorov Institute). In August 1996, advanced nephrotic syndrome was detected with simultaneous aggravation of the renal function (blood urea nitrogen 12–21 mmol/l, serum creatinine 250–540 μmol/l, total urinary proteins 6.4–2.9 g/l).

The patient's general health status worsened abruptly in October 17, 1996 (unconsciousness, hypotonia to 80/50 mm Hg, depressed heart beats, excessive bradycardia to 25 beats/min, bradypnea to 10 breaths/min). Routine laboratory analyses demonstrated acute hyperglycemia to 35 mmol/l, hyperkalemia to 9.0 mmol/l, blood urea nitrogen to 28 mmol/l, advanced anaemia to 64 g/l for hemoglobin, 2.0×10^{12}/l for RBC, and 0.17 for hematocrit. The decompensated renal and ketometabolic acidosis (urine-positive glucose and acetone) was characterized by pH 6.8, BE –30.1 mmol/l, and total HCO_3 6.3 mmol/l. After that, the patient was admitted to the Department of Nephrology (Medical Faculty, Skopje). Before acute dialytic therapy, the patient was initially treated (Medical Center Gostivar) with subcutaneous application of 4 mg atropine, 16 U crystal insulin and intravenous perfusion with orciprenaline (1 mg in total) because of excessive bradycardia (24–32 beats/min) [1] hyperglycemia to 29 mmol/l, and advanced arterial hypotension (0/0, 80/40, 95/40 mm Hg) [2] (fig. 1, 2).

Because of acute transverse cardiac failure dyspnea, painful hepatomegaly, anasarca, and ascites related to diabetic myocardiopathy, hyperkalemia, and acidosis, a transitory extracorporeal cardiac electrostimulation was performed through the right subclavian vein with regular ventricular response and sufficient circulatory stability (mean arterial tension 85 mm Hg, heart rate 74/min). In the meantime, a femoral venous catheter was placed and 3-hour hemodialysis instituted with good results (K^+ 9.0–4.5 mmol/l, blood urea nitrogen 28–18, PO_4 2.2–1.6, blood sugar, 35–20.4 mmol/l, pH 6.8–7.4, BE –30.1 to –5.6 mmol/l, total HCO_3 6.3–17.0 mmol/l) and normalization of cardiac performance (clinical, ECG, and echographic monitoring). The calculated creatinine clearance [3] was 15.6 ml/min and the measured clearance from the 24-hour urine and blood creatinine (5.2 μmol/l) was three times lower (only 6.1 ml/min). During the next 3 weeks, the patient was regularly treated with hemodialysis and insulin. After 3 U RBC, hemoglobin was 98 g/l, RBC 3.4×10^{12} and hematocrit 0.29.

Besides frequent nonmalignant ventricular extrasystolies during the first 7 days after the patient was admitted to the hospital, cardiac function was normal and stable with

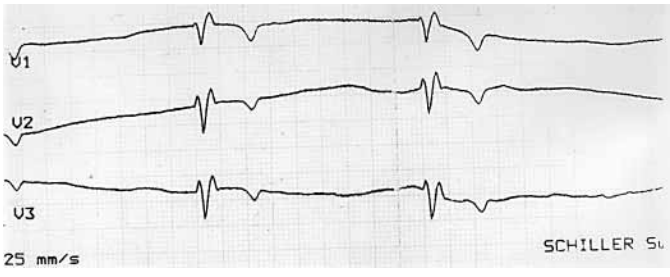

Fig. 1. Hyperkalemic bradycardia.

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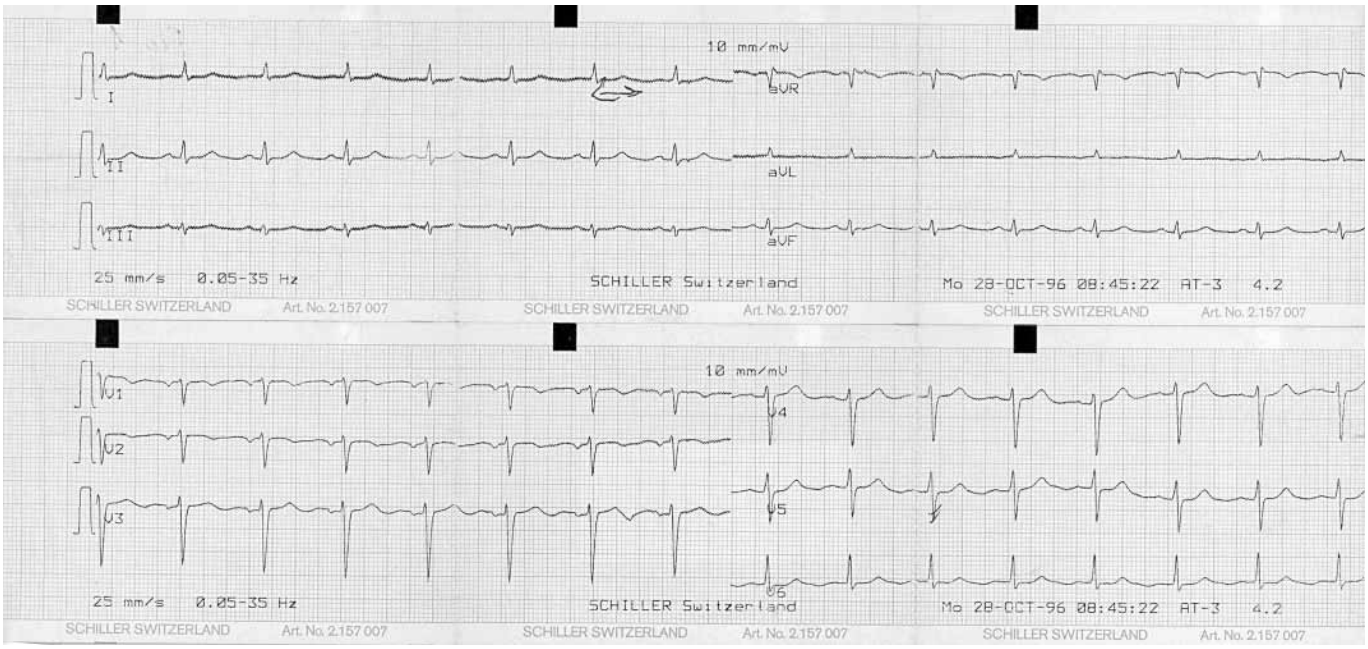

Fig. 2. ECG immediately after the disconnection of the stimulator at the end of dialysis.

reestablishment of arterial hypertension (mean arterial pressure during the last week before discharge from the hospital between 124 and 152 mm Hg) and residual diuresis (800–1,600 ml/day). The blood glucose level was not so well regulated (glycemia between 2.1 and 26 mmol/l) despite the regular diabetologic survey.

Renal echography showed both kidneys to be normal sized (100 × 50 mm) and structural signs characteristic of diabetic nephropathy. Echocardiography demonstrated a moderate interventricular septal hypertrophy (15 mm), marginal systolic function (ejection fraction 60%), and a thick epicardium.

An arteriovenous fistula of the left forearm was placed, and the patient was regularly dialyzed (total 10 hemodialysis sessions in our department) during arteriovenous fistula maturation. The patient left the hospital with the following therapy: amlodipine 10 mg/day, enalapril 20 mg/day, furosemide 250 mg together with 2 mg polythiazide every 2nd day, and calcium carbonate 3.0 g/day besides the regular antidiabetic treatment (Insulatard 24 + 8 IU/day).

This is the first case in our experience where hyperkalemia and malignant bradycardia (20–30 beats/min) were successfully treated by temporary extracorporeal cardiac electrostimulation, giving enough time to normalize the serum level of potassium during the 3 h of hemodialysis. After dialysis, spontaneous cardiac automatism, excitability, and regular rhythm reappeared. The previous standard antikalemic procedures (bicarbonate, calcium, and hypertonic glucose infusion with insulin supplements) were ineffective.

The simultaneous dialytic treatment of metabolic acidosis, possibly complementarily ameliorates the cardiac function through the process of better enzymatic myocytic activity.

References

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