Pseudomyocardial Infarction, Digitalis Intoxication and Hyperkalemia

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CASE REPORT

A 57 year old farm hand was transfered to Invensive Care Unit because of anuria and acute myocardial infarction. Several years earlier nephrolithiasis had been confirmed on both sides. Recently she had creatinine clearance 64 ml/min., RR 200/115 mm Hg (26,7/15,3 kPa). From the beginning of 1983 her general condition deteriorated, she had melena on three occasions. On the day of transfer to the Rebro hospital she was unable to urinate, and hyperkalemia was present and an electrocardiogram indicated acute myocardial infarction (Fig. 1.).

Status on admittance: adipose (97 kg), height 160 cm, tachypnea 26/min., pale. Heart and lungs: normal. RR 110/80 mm Hg (14,7/10,7 kPa), c/p 116/min. Liver 16 cm, hard edge, spleen 4 cm, no signs of fluid in the abdomen. Extremities: edema of both legs, greater of the left leg. Digitorectal: melena.

Laboratory: The hematocrit 19 to 29 per cent, erythrocytes 1,9 to 2,8 x 10^{12}/L, hemoglobin 5,7 to 7,9 g/100 ml, leukocytes 16,1 to 14,7 x 10^{9}/L. Urine: mass of white cells. Potassium in serum: 5,6-4, 8-5,0-7, 1-4, 9 mmol/L. The CPK 17 to 13 U/L, the LDH 1230-1161 U/L. The total bilirubin 31/umol/L, the alkaline phosphates 345-386 U/L, AST 64-76, ALP 58-45 U/L. The index of renal damage (the ratio between the sodium in urine and the creatinine urine-serum ratio was 0,87 indicating prerenal oliguria). The excretion fraction of filtered sodium was 0,64 (the sodium urine-serum ratio and creatinine urine-serum ratio) also indicating prerenal oliguria. Scann of the liver using Tc 99 m: enlarged liver, hepatic masses with hypofixation of Tc 99 m in both sections, enlarged spleen, with homogenous activity, extrahepatic fixation of the radioisotope in the spine. Digoxin concentration in serum (after taking undetermined amount of medigoxin out of hospital, during 9 days): 6.1 nmol/L. and one day later: 4,4 nmol/L

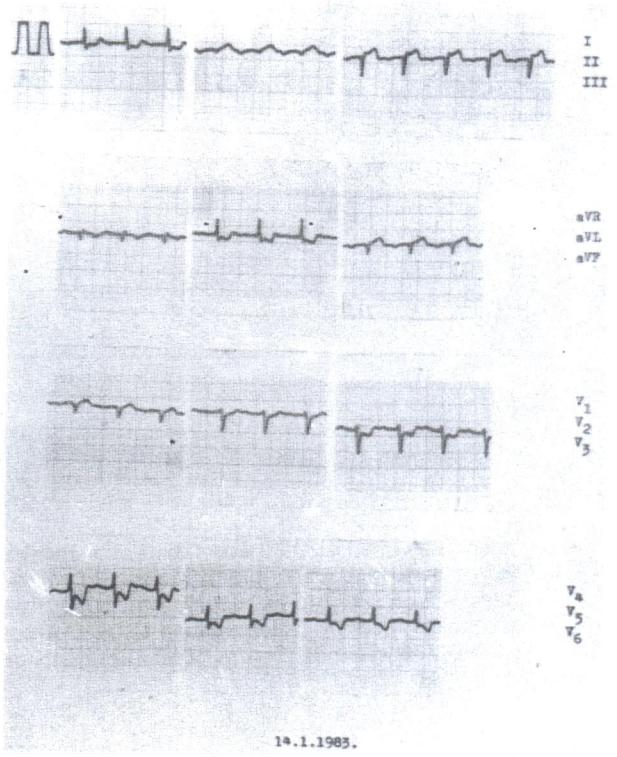

FIGURE — 1.

ECG January 14, 1983 at 9.00 a.m.: Sinus tachycardia of frequency 124/min., first degree atrioventricular block, a picture of acute myocardial infarction of diaphragmatic location with formed Q wave, anterolateral S-T segment depression with negative T wave to 4 mm, microvoltage QRS. Serum digoxin concentration: 6,1 nmol/L. Corrected Q-T interval=0,388 seconds, P-T-Q index=2,472. Serum potassium concentration=5,6 mmol/L.

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The digoxin half-life was 50.9 hours: 1/4 longer than normal because of acute renal failure. Course of illness: Six hours after admittance and following correction of hypovolemia, anemia and hyperkalemia, electrocardiogram no longer showed signs of myocardial infarction, although a first degree atrioventricular block was present, a corrected Q-T interval of 0,333 seconds and P-T-Q index of 2,666 as a results of digitalis effect. Two days later the patient had again hyperkalemia of 7,1 mmol/L, and poor R wave progression apical location, a second degree atrioventricular block Mobitz I type ratio 4:3 (Fig. 2.) After repeated correction of hyperkalemia this ECG signs disappeared. The daily diuresis values were as follows: 30-220-550-450-1125-1100-1000 ml. The patient died in hepatic failure five days later, of progressive malignant disease of the liver. During autopsy a primary carcinoma of the liver was found together with metastases, but no lesion of myocardium-coronary disease was found.

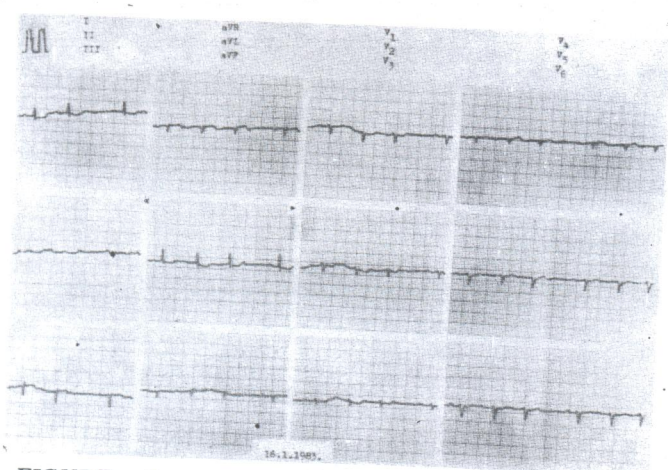

FIGURE — 2.

ECG. January 16, 1983 at 9.00 a.m.: Left axis deviation, second degree atrioventricular block Mobitz I type ratio 4:3, ventricular rhythm 80/min., a picture of poor R wave progression apical location, microvoltage QRS. Corrected Q-T interval=0,275 seconds. Serum potassium concentration=7,1 mmol/L.

DISCUSSION

The case was one of hypovolemia and acute renal failure, together with hyperkalemia and digitalis intoxication as a result of the above, with signs of myocardial infarction. The hyperkalemia disappeared together with signs of myocardial infarction. During autopsy no signs of myocardial infarction were found, indicating once again hyperkalemia as the cause of the electrocardiogram picture of myocardial infarction. Digitalis intoxication, together with acute renal failure, was the cause of hyperkalemia. In literature isolated reports exist on electrocardiogram changes of the pseudomyocardial infarction with hyperkalemia in diabetic ketoacidosis primarily (3,4), with S-T segment elevation, and rare(1) with pathological Q wave. Explanation of the picture of pseudomyocardial infarction is still not definite, and attempts are being made to look for the answer in the changes of the transmembranous potential of potassium during its increased concentration (1,2,5) decreased amplitude and duration of depolarization, and also the conduction of excitation.

References :

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