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Case report

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A case of ST-elevation and nystagmus—when coronary thrombosis is not to blame

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Summary

We present the case of a post-menopausal female who, following a prolonged period of vomiting and diarrhoea, presented acutely with Wernicke's encephalopathy, chest pain, ST-segment elevation and congestive cardiac failure associated with hypotension. Coronary angiography demonstrated no abnormality. Haemodynamics improved significantly in the short-term following intravenous thiamine replacement, with complete resolution of all ST-segment abnormalities and normalization of left ventricular function at six-week follow-up. Shoshin beriberi in the context of metabolic alkalosis, secondary to severe electrolyte depletion, is exceptional and is a differential diagnosis to be considered in all malnourished patients presenting with symptoms and signs suggestive of an acute coronary syndrome, especially those with a history of alcohol-dependence.

In March 2009, a 59-year-old woman presented to hospital acutely confused. She had a 3-week history of progressive unsteadiness of gait, poor appetite, vomiting, diarrhoea and a 2-h history of central chest discomfort. In addition to chronic alcohol dependence and depression, she had a history of non-insulin-dependent diabetes mellitus, hypertension and hyperlipidaemia, and was a cigarette smoker of 20 per day for 40 years. Her regular medications included aspirin, bumetanide, ramipril, simvastatin and fluoxetine, although she had not taken these in the preceding 2 weeks due to nausea and reduced oral intake.

On admission she was afebrile, tachycardic (132 b.p.m.) and hypotensive (64/40 mmHg). Clinical examination revealed horizontal nystagmus, dysdiadokinesis, an elevated venous pressure and bibasal inspiratory crepitations. She was clinically euthyroid. Initial laboratory investigations included Hb 17.4 g/dl [Normal range (NR) 13.0–18.0 g/dl], Na^+ 139 mmol/l (NR 135–145 mmol/l), K^+ 2.5 mmol/l (NR 3.5–5.0 mmol/l), urea 6.0 mmol/l (NR 3.3–8.8 mmol/l), creatinine 62 μmol/l (NR 40–110 μmol/l) and Mg^{2+} 0.27 mmol/l (NR 0.75–1.15 mmol/l). Arterial blood gas analysis revealed: pH 7.49 (NR 7.35–7.45), pCO_2 5.6 kPa (NR 4.7–6.0 kPa) and HCO_3^- 33.1 mmol/l (NR 22–28 mmol/l). Cardiac troponin T (cTnT) was elevated at 1.42 μg/l (NR < 0.03 μg/l). Thyroid function and urinary catecholamine output were within normal limits. Electrocardiogram (ECG) demonstrated a prolonged QT_c and ST segment elevation in the anterolateral praecordial leads (Figure 1). She was treated with intravenous thrombolysis as standard. Her deficiencies in both Mg^{2+} and K^+ were corrected by intravenous replacement. Ninety-minutes post-thrombolysis, with no electrocardiographic evidence of reperfusion, ongoing chest discomfort and hypotension, she proceeded to rescue angioplasty. Coronary angiography showed no significant coronary artery stenoses or intracoronary thrombus and revealed severely

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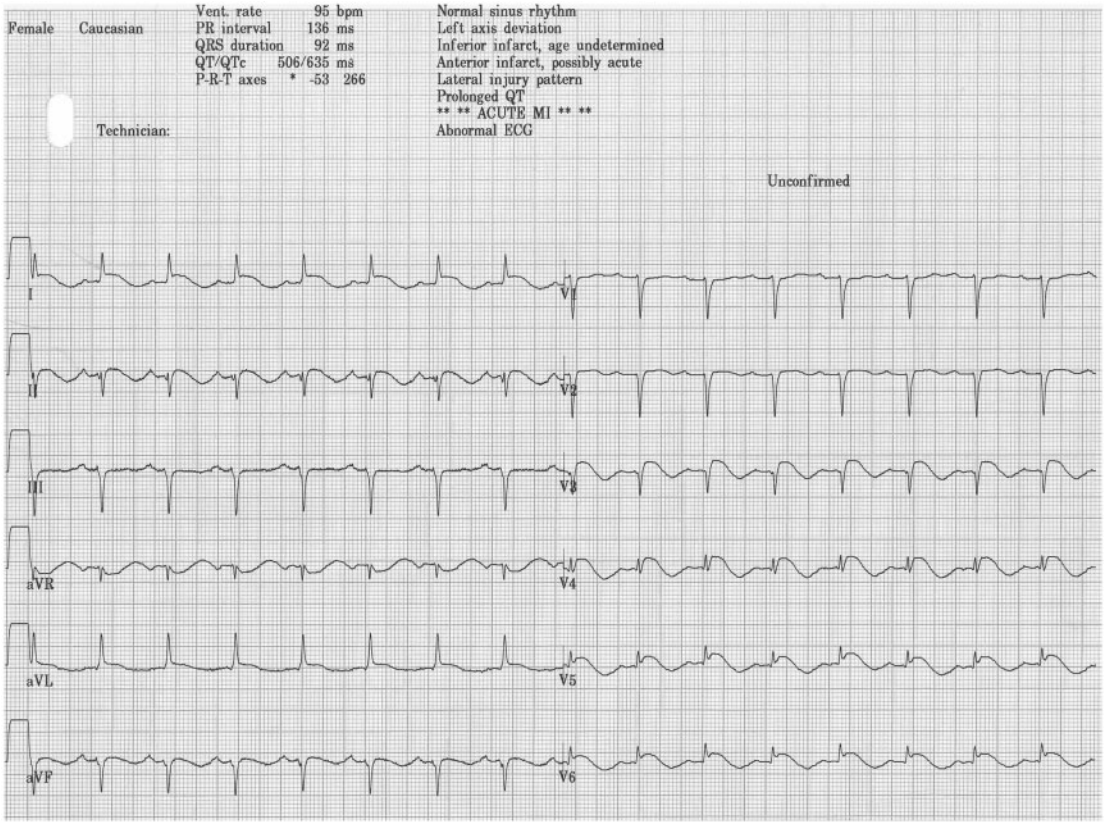

Figure 1. Electrocardiogram (ECG) on presentation demonstrating a prolonged QT interval and anterolateral ST-segment elevation.

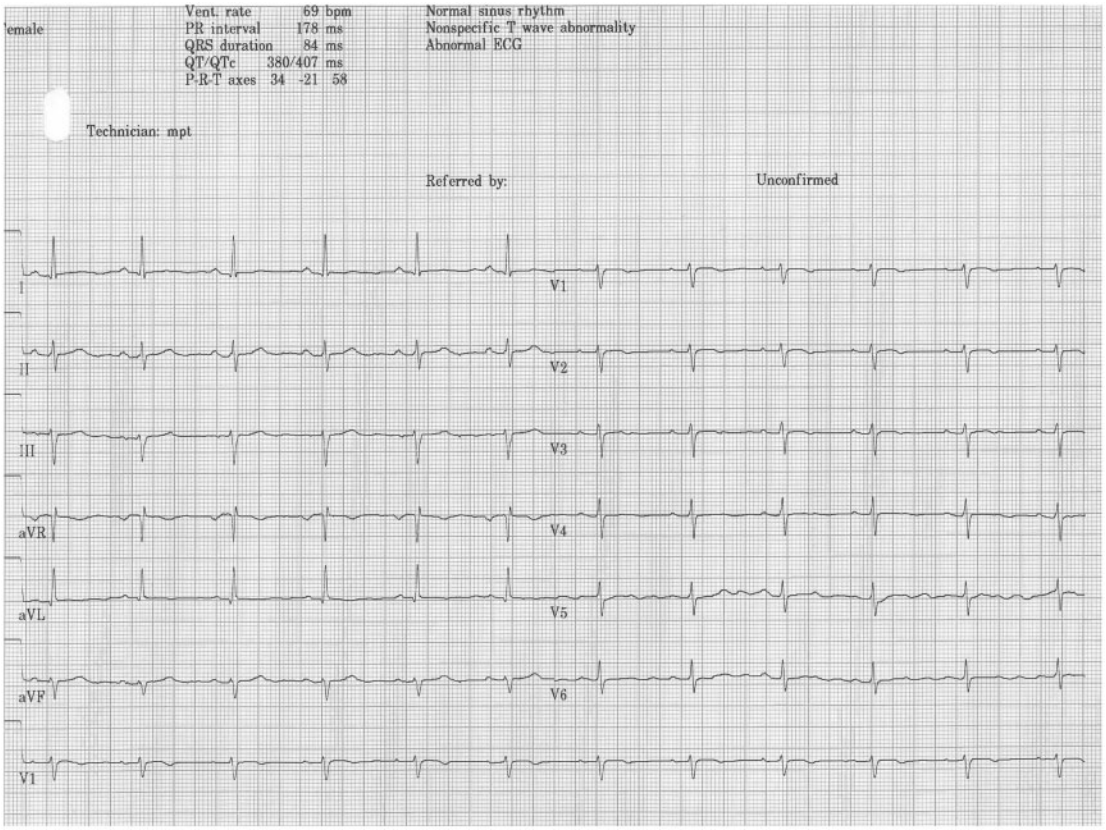

Figure 2. ECG six-weeks post-discharge showing complete resolution of all ST-segment abnormalities.

impaired left ventricular (LV) systolic function with diffuse anteroapical hypokinesis. Transthoracic echocardiography estimated LV ejection fraction to be 21%. Upon return to the Coronary Care Unit she remained hypotensive, with a systolic blood pressure (SBP) of 68 mmHg. Subsequent treatment with intravenous thiamine hydrochloride 500 mg resulted in an immediate improvement in SBP: 78 mmHg at 1-h, 94 mmHg at 4-h and 102 mmHg at 8-h post-treatment. Peak cTnT (10-h post-admission) was significantly elevated at 10.08 μg/l, with NT-proBNP 938 ng/l (NR < 222 ng/l). She was discharged 7 days later on oral thiamine with no clinical evidence of Wernicke's encephalopathy, haemodynamically stable and with a normalized K^+, Mg^{2+}, NT-proBNP and cTnT. Follow-up echocardiogram 6 weeks post-discharge revealed normal LV systolic function with an ejection fraction of 58%. ECG at this time showed normalization of the QT_c interval, no pathological Q wave development and complete resolution of all previous ST-segment abnormalities (Figure 2).

Thiamine deficiency may present in four classical clinical forms: peripheral polyneuropathy, muscular weakness and anorexia (dry beriberi); high output cardiac failure with signs of congestion (wet beriberi); berberi associated with shock (Shoshin beriberi) and Wernicke's encephalopathy. Beriberi heart disease, originally described in Asia by Aalsmeer in 1929[1] remains relatively uncommon in the west.[2] Characteristic ECG changes include biphasic or inverted T-waves, prolongation of the QT_c and sinus tachycardia. While thiamine deficiency is known to be associated with ST-segment elevation in animal models, there has been only one reported case in humans.[3]

Diagnostic tests for thiamine deficiency are rarely performed in the acute care setting since their result is often delayed beyond the period of acute illness. Diagnosis is commonly based on monitoring the therapeutic response to thiamine replacement.[4] In this case, the rapid improvement in haemodynamics following the treatment of Wernicke's encephalopathy with intravenous thiamine, in the absence of any other precipitant of reversible ventricular dysfunction, is diagnostic. Acute heart failure accompanied by ST-segment elevation in patients with a history of alcohol abuse, malabsorption states, malnutrition or eating disorder should prompt the clinician to consider the presumptive diagnosis of cardiac beriberi.[5]

References

1. Aalsmeer WC, Wenkebach KF. The heart and circulatory system in beriberi. *Am J Heart* 1929; **4**:630.
2. Attas M, Hanley HG, Stultz D, Jones MR, McAllister RG. Fulminant beriberi heart disease with lactic acidosis: presentation of a case with evaluation of left ventricular function and review of pathophysiologic mechanisms. *Circulation* 1978; **58**:566–72.
3. Kawano H, Koide Y, Toda G, Yano K. ST-segment elevation of electrocardiogram in a patient with Shoshin beriberi. *Intern Med* 2005; **44**:578–85.
4. Meurin P. Shoshin beriberi: a rapidly curable haemodynamic disaster [French]. *Presse Med* 1986; **25**:1115–18.
5. Loma-Osorio P, Penafiel P, Doltra A, Sionis A, Bosch X. Shoshin beriberi mimicking a high-risk non-ST-segment elevation acute coronary syndrome with cardiogenic shock: when the arteries are not guilty. *J Emerg Med* 2009 (in press).