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Lessons of the month 2: A forgotten cause of transient T-wave inversion

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ABSTRACT

A 19-year-old patient presented with severe chest pain, which is not typical for cardiac angina. However, his smoking history and the strong family history of ischaemic heart disease coupled with evidence of progressive T-wave changes on his electrocardiogram (ECG) caused dilemma in deciding further management. His blood tests were normal apart from hypophosphataemia, and he had two negative troponin results. His arterial blood gases showed respiratory alkalosis. He was given analgesia for a diagnosis of musculoskeletal chest pain and the next morning his ECG, arterial blood gases and phosphate levels all normalised. He had a normal echocardiogram and was reviewed by the cardiologist who diagnosed musculoskeletal chest pain which led to distress and hyperventilation causing hypophosphataemia and transient T-wave inversion. This case is a reminder of an under-recognised physiological phenomenon involving the cardiac conduction during hyperventilation.

KEYWORDS: Hyperventilation, T-wave inversion, hypophosphataemia

Case presentation

A 19-year-old Caucasian male presented to the acute medical unit from the emergency department with severe chest pain which was worse on breathing and moving the arms. He had no previous medical history and was on no medications. He had no risk factors for venous thromboembolism. He smoked 10 cigarettes per day, drank alcohol occasionally and denied any recreational drug use. He works as a farmer and had busy work the previous few weeks in his farm. His father, who was a smoker, died of myocardial infarction at the age of 50.

Examination was unremarkable aside from mild tenderness on palpation of the anterior left chest wall. He was still complaining of chest pain and had tachypnoea (28 breaths per minute) with normal blood pressure, pulse and saturation. Chest X-ray was normal, and initial electrocardiogram (ECG) showed sinus rhythm with T-wave inversion in lead V3 (Fig 1). Serial ECGs were performed which showed further T-wave inversion in lead V4 (Fig 2) and then in lead V5 as well (Fig 3).

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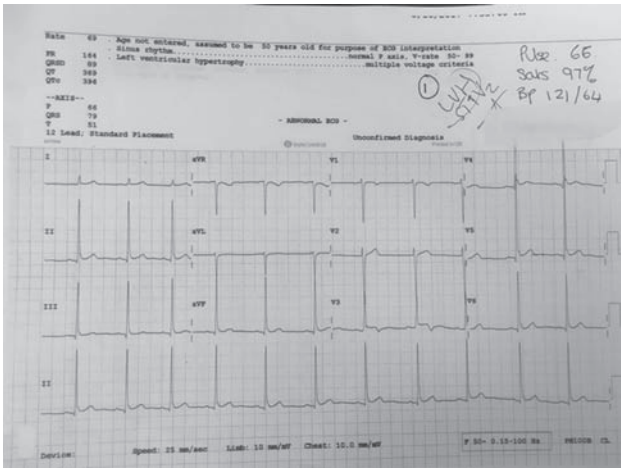

Fig 1. Initial electrocardiogram showing sinus rhythm with T-wave inversion in lead V3.

Blood tests including full blood count, renal function, liver function test, two troponin tests, D-dimer and inflammatory markers were all normal. Serum phosphate level was noticed to be

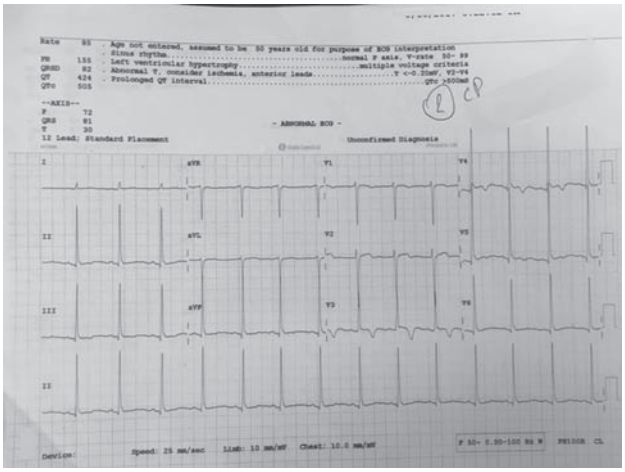

Fig 2. Electrocardiogram showing sinus rhythm with further T-wave inversion in lead V4.

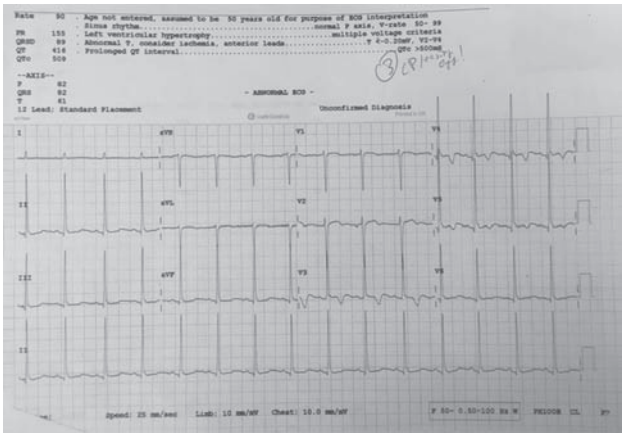

Fig 3. Electrocardiogram showing sinus rhythm with T-wave inversion in lead V5 as well.

significantly low at 0.3 mmol/L (0.8–1.5). His arterial blood gases (ABG) showed marked respiratory alkalosis with pH 7.641, pCO_2 2.72 kPa, pO_2 16.10 kPa and HCO_3 21.5 kPa which was thought to be due to pain-induced tachypnoea and hyperventilation. He had an echocardiogram which was normal. His pain sounded typical of musculoskeletal origin and he required regular strong analgesia during the night. The following morning he was pain-free and his ECG, ABG and PO_4 level had all normalised. He was reviewed by the cardiologist who diagnosed musculoskeletal chest pain which led to distress and hyperventilation causing hypophosphataemia and transient T-wave inversion.

Discussion

T-wave inversion on ECG is usually a concerning finding as it is often associated with myocardial ischaemia or ventricular strain. The respiratory variation in the T-wave morphology has been reported in the literature,[1] however the frequency of this observation remains unknown as this is not routinely evaluated. Hyperventilation has long been known to cause transient ECG repolarisation abnormalities[2] although it is rarely talked about in modern clinical settings. The exact physiological mechanism of these repolarisation changes is not fully understood. It has been posited that they are a direct consequence of respiratory alkalosis or hypocapnia;[3] however T-wave changes in some studies occurred after a short period of hyperventilation, which is unlikely to have caused significant pH change.[2] T-wave inversion has been demonstrated due to hyperventilation in high CO_2 atmosphere where hypocapnia and alkalosis did not occur.[2] An interesting study demonstrated that decreasing pCO_2 reduced the amplitude of T-waves at a fixed volume ventilation while separately increasing ventilation also decreased T-wave amplitude at a fixed pCO_2.[3] Extrapolation suggested extreme hyperventilation would decrease the T-wave amplitude enough for it to be inverted.[3] T-wave changes due to hyperventilation have also been shown to be unrelated to electrolyte disturbances or changes in heart position.[4] Another suggestion of a possible mechanism has been via a vagal reflex similar to the Hering–Breuer reflex that terminates inhalation to prevent overinflation of the lungs, and the reflex responses of coughing, airway constriction and hyperventilation.[2]

One study involving 474 healthy subjects showed that 15% developed repolarisation changes in response to hyperventilation.[3] The majority consisted of T-wave inversion – either from positive to negative, or negative to positive, eg lead III. A minority of the subjects that did show repolarisation abnormalities (13%) demonstrated ST depression. Most of the ST–T changes arose after brief (30–90 seconds) hyperventilation and soon after breathing returned to normal.[5]

Apart from the ECG changes and respiratory alkalosis our patient had hypophosphataemia on admission which the admitting team initially could not explain. Hypophosphataemia is common in hospitalised patients and the cause of hypophosphataemia is usually multifactorial, but the main mechanism is cellular redistribution, renal loss, malabsorption or gastrointestinal loss.[6] Hyperventilation is a common cause of hypophosphataemia in hospitalised patients and may not be obvious.[7] Hyperventilation causes a respiratory alkalosis which in turn mediates an increase in intracellular pH. This stimulates phosphofructokinase activity in the glycolytic pathway with a subsequent increase in demand for phosphate, which is driven intracellularly to form glycolytic intermediate metabolites.[8]

In this case the combinations of respiratory alkalosis, hypophosphataemia and T-wave inversion that all normalised with adequate analgesia have helped to reach the correct diagnosis.

Summary

Our young patient presented with severe chest pain, which is not typical for cardiac angina. However the smoking history and the strong family history of ischaemic heart disease coupled with evidence of progressive T-wave changes on ECG added to the dilemma in deciding further management. This case is a reminder of an under-recognised physiological phenomenon involving the cardiac conduction. In patients with a low risk of coronary artery disease who present with atypical chest pain and an abnormal ECG, arterial blood gas analysis should be considered to exclude hyperventilation and alkalosis. ■

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