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Hyperkalaemia as a differential diagnosis in chest pain

EDITORIAL

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The treatment of patients with suspected acute myocardial infarction generally works well. Sometimes other suspected diagnoses become so urgent that treatment must be initiated rapidly.

When acute myocardial infarction is suspected, the standard treatment protocol is immediate transportation to a hospital able to offer percutaneous coronary intervention, called a PCI centre. If the patient's condition necessitates it or the time required for transportation is too long, the shortest journey to a local hospital for thrombolytic treatment is chosen (1, 2).

The Journal of the Norwegian Medical Association is now publishing a case report by Rasmussen and Bjerring regarding a woman in her forties with chest pain, dyspnoea and bradycardia whose condition rapidly deteriorated and became life-threatening (3). Thanks to quick-witted diagnostic testing and treatment, the correct diagnosis was made (severe hyperkalaemia) and the correct treatment initiated promptly, so the patient survived. The only formula followed was vigilance and improvisation combined with good clinical judgement and logistical guidelines for patients with suspected myocardial infarction.

The patient's unusual ECG findings meant that myocardial infarction could not be ruled out initially. It is then important to perform echocardiography on arrival in hospital to confirm or rule out acute myocardial infarction and possible pulmonary embolism. Overly strict use of routine logistical procedures according to established rules can lead to use of thrombolytic treatment or referral for percutaneous coronary intervention on an incorrect basis (2). Rapid collection of relevant diagnostic data provided a sound basis for making the correct diagnosis and initiating the correct treatment for this patient. The diagnosis of severe hyperkalaemia was confirmed once the results of electrolyte analysis were available.

Potassium is extremely important for the maintenance and regulation of the resting membrane potential of cells, which is generated by the sodium-potassium pump. Potassium is the most abundant cation in the body, and normal levels are maintained by an ordinary varied diet. The vast majority is intracellular (98 %), but routine clinical practice only measures the small, but equally important, extracellular levels. Among all the ions, sodium and potassium are the predominant ions in creating action potential in cells. Major changes in their levels outside the upper or lower reference ranges results in muscle weakness, tiredness, numbness, nausea and vomiting, chest pain and abdominal pain (4). Minor changes in potassium levels are usually asymptomatic. The reference range is defined slightly differently for serum or plasma analysis, and errors in blood sample collection, including a tight tourniquet and haemolysis, are not uncommon causes of falsely elevated results.

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Both low and high extracellular potassium levels result in cardiac arrhythmias, everything from palpitations to life-threatening arrhythmia (5). Excessively high or low potassium intake has the same consequences as other causes of altered potassium balance such as renal failure, acid-base changes, diarrhoea, diuretics, medicinal treatment of heart failure, insulin and beta-blockers (4, 6). Many patients admitted to hospital with acute illness are diagnosed with catecholamine-induced hypokalaemia (6). This is due to increased adrenaline, induced by the stressful situation, causing a rapid shift of potassium across the cell membrane from the extracellular to the intracellular space.

The situation usually normalises without action. Severe ventricular arrhythmias can be caused by mild hypokalaemia (3–3.5 mmol/L) in cases of acute myocardial ischaemia due to potentiating effects of catecholamines (5). People with good cardiac function do not have a similar risk, and in these people severe ventricular arrhythmias or atrioventricular block only develop with hypokalaemia below 2.0 mmol/L. Corresponding adjustments in risk are not made for hyperkalaemia because values over 6.5 mmol/L are considered to be of potentially serious significance in all cases (4).

Acute ST-elevation myocardial infarction is characterised by the onset of acute chest pain and ST elevation on ECG. However, there are a number of causes of ST elevation on ECG that are unrelated to ischaemia and infarction (7). It is easy to misinterpret ST elevation as a sign of infarction, especially in cases with widening of the QRS complex. Therefore, it is important that staff in the emergency medical communication centres, who interpret the ECG and give necessary feedback to the ambulance teams, have up-to-date knowledge in this field.

There are good reasons for adhering to a well-known and well-established logistical procedure when acute myocardial infarction is suspected. This type of treatment protocol has worked extremely well for many years and has been subject to continual improvement. In practice, the protocol works as a checklist that must be followed point-by-point. In the case report presented by Rasmussen and Bjerring, it meant that the patient arrived at hospital earlier, where planned diagnostic testing according to the routine logistical procedure gave early confirmation that the patient had not had a myocardial infarction. This enabled the correct diagnosis to be made, and targeted and life-saving treatment to be initiated.

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