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A man in his sixties with acute chest pain

EDUCATIONAL CASE REPORT

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BACKGROUND

Chest pain is a frequent symptom of acute myocardial infarction, but the cause is not always coronary atherothrombosis. We present a case where the patient himself had made a correct non-cardiac diagnosis, but this was initially overlooked by the doctor.

CASE PRESENTATION

A man with a history of cardiac infarction was admitted with acute chest pain and troponin elevation. Electrocardiogram suggested ST elevations, echocardiography showed a possible slight hypokinesia, and we primarily suspected an acute coronary syndrome. However, invasive coronary angiography was negative and a primary acute myocardial infarction was less likely. A renewed interview revealed that the patient had worked with a propane burner indoors without adequate ventilation. The patient himself suspected carbon monoxide poisoning. Arterial blood gas showed HbCO 27.4 %, which was unfortunately overlooked initially. The patient had carbon monoxide poisoning with symptoms of dizziness, hand ataxia and myocardial ischaemia. He received 100 % oxygen and HbCO was normalised.

INTERPRETATION

The imbalance between oxygen demand and supply resulted in a type 2 cardiac infarction. A thorough medical history is crucial for correct diagnosis but can unfortunately be missed on a busy shift. This case illustrates the importance of the patient's own diagnostic assumption.

A man in his sixties was admitted to a busy emergency department with acute chest pain and elevated troponin. Only after invasive coronary angiography revealed normal findings did the team on duty have to acknowledge that the patient himself had made the correct non-cardiac diagnosis.

The patient was a man in his sixties with vascular disease, haemochromatosis and previous branch retinal vein occlusion in one eye. He was taking medicinal treatment for hypertension. Several years earlier, he had undergone percutaneous coronary intervention (PCI) with stenting of the diagonal branch of the left coronary artery for an anterior wall myocardial infarction.

He was admitted in the daytime when he became acutely unwell, with dizziness and retrosternal chest pain radiating to the neck and jaw after he had been doing manual labour on a boat along with two friends. The pain was accompanied by dyspnoea, but not nausea. Glyceryl trinitrate had provided relief in pre-hospital care.

He continued to have chest pain in the emergency department. His skin was dry and warm. His blood pressure was 130/82 mmHg in the right arm and 149/89 mmHg in the left arm. His respiratory rate was 13 breaths per minute, and arterial blood gas analysis revealed oxygen saturation of 100 % on room air. His pulse was regular at 88 bpm.

Electrocardiography (ECG) showed sinus rhythm at a rate of 90 bpm and suggested ST elevations in leads II, III and aVF, all corresponding to the inferior wall of the heart. Echocardiography in the emergency department found good left ventricular function with normal ejection fraction, but it was queried whether there might be slight hypokinesia in the posterior apical septum. No valve defects were detected, and there was no pericardial effusion. Based on information about previous coronary heart disease, acute chest pain, slight ischaemic changes on ECG and possible slight regional hypokinesia in the left ventricle on echocardiography, our primary suspicion was acute coronary syndrome.
The patient suddenly developed substantial tremor in his hands, but denied feeling cold. He did not have a fever. His rectal temperature was 36.4 °C. He also complained of increasing dizziness.
Sepsis and aortic valve disease were now both considered in the differential diagnosis.

A CT scan of the aorta found no signs of aortic valve disease. The results of blood samples taken immediately after admission were as follows: leukocytes 12.4 × 10^9/L (reference range 3.5–10.0 × 10^9/L), haemoglobin 14.8 g/dL (13.4–17.0), CRP <0.6 mg/L (<5.0), creatinine 74 µmol/L (60–105) and troponin T 28 ng/L (0–14), with only troponin T being abnormal. Therefore, it was concluded that the diagnosis was likely to be acute non-ST-elevation myocardial infarction, even though the diagnosis of acute myocardial infarction cannot be made based on one isolated troponin result.
The patient received oral antiplatelet therapy with aspirin 300 mg and ticagrelor 180 mg, before rapid performance of invasive coronary angiography. The investigation found that the coronary stent inserted previously was patent and that there were no new coronary artery stenoses.
In terms of the differential diagnosis, the patient's condition now appeared to be unexplained. Primary acute myocardial infarction was less likely with negative coronary angiography, but a thrombosis that was no longer present could not be entirely ruled out.

Another review of the patient's case history revealed that he had worked with a propane burner indoors for several hours without adequate ventilation of the space. He himself suspected carbon monoxide poisoning. Results of arterial blood gases taken on arrival, but not adequately evaluated, were as follows: pH 7.40 (7.36–7.44), pO_2 9.0 kPa (10.5–14.0), pCO_2 4.8 kPa (4.7–6.0), HbCO 27.4 % (0–1.5) and lactate 1.5 mmol/L (0.5–2.2).
Therefore, carbon monoxide pressure was considerably elevated, which was unfortunately overlooked initially. A repeat arterial blood gas analysis three hours after admission showed that HbCO had fallen to 16.1 %.
The patient had acute, severe carbon monoxide poisoning with symptoms consisting of dizziness, malaise, hand ataxia and myocardial ischaemia. This was only recognised after the findings of coronary angiography were revealed to be normal.

He received 100 % oxygen through a non-rebreather mask with a reservoir (10 L/min), and his HbCO levels had returned to normal the next day. The patient was discharged in a good condition.

Discussion

Chest pain is a common symptom in acute myocardial infarction and a common cause of admission to medical departments. Approximately 10,000 people have an acute myocardial infarction in Norway each year, and patients with an established history of coronary artery disease are at increased risk of suffering a further myocardial infarction (1, 2).

The diagnosis of acute myocardial infarction is based on a rise and/or fall in the cardiac marker troponin as well as at least one of the following additional criteria a) ischaemic symptoms, b) ECG changes, c) imaging evidence of loss of viable myocardium or new regional wall motion abnormality, or d) identification of intracoronary thrombus by angiography or autopsy (3). Our patient fulfilled the diagnostic criteria for acute myocardial infarction if his chest pain was interpreted as ischaemic symptoms or the queried hypokinesia on echocardiography was considered conclusive.

However, in this case, the cause was not coronary atherothrombosis. In this situation of carbon monoxide poisoning, there was an imbalance between oxygen demand and supply in the myocardium since oxygen was displaced from haemoglobin in the blood. The myocardial infarction can be defined as a type 2 infarction.

In a busy emergency department, it is easy to follow the first diagnostic assumption, which is often 'inherited' from the referring doctor or ambulance service. Our patient was admitted with typical clinical findings of acute coronary syndrome, with relief provided by glyceryl trinitrate in the ambulance. Although the patient himself suspected carbon monoxide poisoning and communicated this, the findings and test results were interpreted as confirmation of coronary syndrome. This type of confirmation bias is probably recognisable to everyone working in emergency medicine, but is something we should always be alert to. A thorough case history with probing questioning about relevant information is essential for correct diagnosis and treatment, but can unfortunately be easily missed on a busy shift. Our case report illustrates the importance of being attentive to the patient's own assumption about the diagnosis.

Carbon monoxide (CO) is an odourless, colourless and non-irritant gas produced by the incomplete combustion of organic material due to insufficient oxygen supply. Carbon monoxide poisoning is likely to be responsible for over half of all fatal cases of poisoning worldwide (4). Carbon monoxide that is not accompanied by smoke can only be detected using a CO detector, but should be suspected in all fires and when combustion devices are used in confined spaces.

Symptoms of carbon monoxide poisoning can develop gradually over minutes to hours. Signs and symptoms of hypoxia are most prominent in the acute phase. The heart and brain have a high oxygen consumption and are thus most sensitive to oxygen deficit with the earliest impact on organ function. Central nervous system symptoms, such as headache and dizziness, occur frequently.

Other common symptoms of hypoxaemia are tachypnoea and dyspnoea. Non-ST-elevation myocardial infarction (NSTEMI) on ECG or type 2 infarction are rarer, but not uncommon (5).

Patients with acute carbon monoxide poisoning usually have HbCO levels above 10 % (6), but severity correlates poorly with the levels measured because these decrease over time, particularly with oxygen treatment. HbCO levels above 40 % are reported to be fatal (7, 8), although many people have survived with higher levels. Carbon monoxide binds to the oxygen binding site on the haemoglobin molecule with much higher affinity than oxygen and thus blocks normal oxygen transport in the blood.

Oxygen is vital for the maintenance of aerobic metabolism, enabling energy-carrying adenosine triphosphate (ATP) molecules to be created. This ATP production takes place mainly in the mitochondria. Carbon monoxide also binds to cytochromes, myoglobin and guanylyl cyclase (9). Binding to cytochrome 3A and cytochrome C-oxidase in the mitochondria blocks oxidative metabolism. This results in the production of reactive oxygen compounds, leading to oxidative stress. Carbon monoxide competes with nitric oxide (NO) for binding to proteins, which in turn results in increased NO levels. Reactive oxygen compounds and nitric oxide appear to play an important role in the development of oxidative brain injury with peroxidation and demyelination, and may explain the delayed neurological sequelae experienced by some patients following severe poisoning.

Most pulse oximeters do not differentiate between oxyhaemoglobin (HbO_2) and carboxyhaemoglobin (HbCO) and therefore cannot be used for diagnosis. In this particular case, the pulse oximeter showed oxygen saturation (SpO_2) of 100 %, even though 27.4 % of haemoglobin was occupied by carbon monoxide.

The treatment of carbon monoxide poisoning is primarily with normobaric oxygen. Carbon monoxide is excreted via the lungs once exposure stops, and elimination follows the concentration gradient. In addition, oxygen will compete for the binding sites and facilitate elimination. The higher the oxygen concentration, the faster excretion will take place. The half-life of HbCO in the blood of patients on room air (21 % oxygen) is 4–6 hours, but this decreases to approx. 1.5 hours with the administration of 100 % oxygen (10).

If the amount of oxygen is increased with the use of a pressure chamber, which is referred to as hyperbaric oxygenation, the half-life will decrease further to approx. 20 minutes with 100 % oxygen at 3 atmospheres of pressure, equivalent to a depth of 20 metres underwater. A pressure tank or hyperbaric oxygen therapy has not been shown to reduce mortality in the acute phase because normobaric oxygen therapy is enough to counter the hypoxia, but the method has been used to limit the delayed neurological sequelae (9). This is controversial though, and the results of clinical trials have not been conclusive (11). The treatment rapidly reduces the concentration of HbCO, but does not assist in reducing the concentration of oxygen radicals.

Although treatment in modern pressure chambers is safe on the whole, any hyperbaric therapy is associated with some risk of barotrauma (ear and sinus injuries, pneumothorax) and oxygen toxicity (12, 13). Hyperbaric oxygen therapy is still used in Norway, but the trend is to restrict who is offered this

treatment. According to the guidelines of the Norwegian Poisons Information Centre and Oslo University Hospital, hyperbaric oxygen therapy is recommended if HbCO > 25–30 % (for pregnant women HbCO > 15 %), in patients with loss of consciousness, severe metabolic acidosis (pH <7.25) or evidence of end-organ damage (e.g. ECG changes, elevated cardiac markers, respiratory failure, neurological deficit or altered mental status).

Our patient was not assessed for hyperbaric oxygen therapy. Even though his HbCO levels were high enough, he did not have metabolic acidosis or loss of consciousness while admitted, and it was a long way to travel to a chamber. New treatment principles that have not yet been fully clarified include normocapnic hyperventilation, in which carbon dioxide is added to inspired air to increase respiratory minute volume. This lowers the half-life of carbon monoxide by an equivalent amount to a pressure tank, but without causing such a high oxygen load. Medicinal treatment of the inflammatory processes with steroids has also been proposed (14), but this has also not yet been studied in enough depth for it to be recommended as routine practice.

The patient has consented to the publication of the article.

The article has been peer-reviewed.

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