Diagnosis of carbon monoxide-induced acute myocardial injury using a bone scan

Introduction

Carbon monoxide poisoning-induced myocardial injury is not infrequent. Although it can be diagnosed using electrocardiogram, cardiac biomarkers and echocardiogram, there were no readily usable imaging tools. This article presents a case of carbon monoxide poisoning-induced myocardial injury detected by bone scan. The positive finding on the bone scan (hot uptake) can be easily interpreted by non-expert clinicians, and the bone scan can detect injured muscles.

Discussion

Traditionally, a diagnosis of carbon monoxide-induced myocardial injury depends on ischaemic changes in the electrocardiogram (ST- or T-wave changes and ST segment elevation), elevated levels of cardiac biomarkers (cardiac troponin I >0.7 ng/ml), and abnormal echocardiograms (abnormal left ventricular function, regional wall motion abnormality) (Satran et al, 2005).

Newer imaging modalities such as magnetic resonance imaging have been used to try and identify myocardial damage in moderate to severe carbon monoxide intoxication (Henry et al, 2008; George et al, 2014). Henry et al (2008) suggested the utility of cardiac magnetic resonance imaging in a patient with a completely normal echocardiogram. However, the diagnostic ability and prognostic values of cardiac magnetic resonance imaging have not been evaluated.

In the current case, a bone scan was performed to evaluate the extent of rhabdomyolysis. Tc99-labelled hydroxymethylene diphosphonate can react with calcium released from damaged muscle, and the bone scan can therefore

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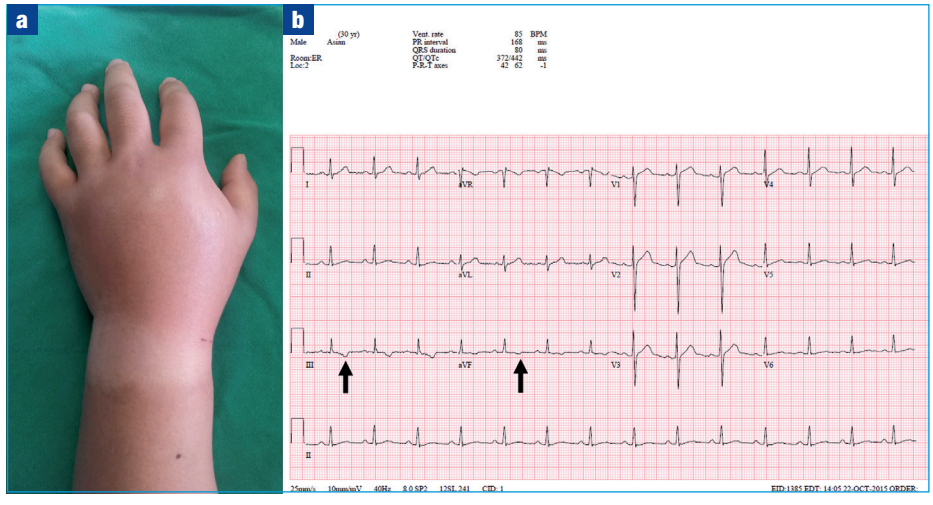

Figure 1. **a.** The patient's arm swelling and **(b)** electrocardiogram at presentation. Black arrows indicate T-wave abnormalities (down-sloping ST segment depression and T-wave inversion).

CASE REPORT

A 30-year-old man visited the emergency department with a 1-day history of chest discomfort. Two days earlier, he had lost consciousness for 36 hours. His last memory was of grilling steaks over a charcoal fire in his one-room apartment. His medical history was unremarkable.

Physical examination showed swelling and tenderness in his forearms and hands (*Figure 1a*). Although he could not clench his fists because of the swelling and felt paraesthesia in both hands, the distal circulation was not impaired. The electrocardiogram showed down-sloping ST segment depression and T-wave inversion in leads III and aVF (*Figure 1b*). The laboratory results were: creatine phosphokinase 13 050 IU/litre, creatine kinase MB isoenzyme 10.86 ng/ml, troponin-I 2.659 ng/ml, myoglobin 454.98 ng/ml, aspartate aminotransferase 348 IU/litre, alanine aminotransferase 144 IU/litre, lactate dehydrogenase 786 IU/litre, and S-100 0.094 ng/ml. According to a co-oximeter, the patient's oxyhaemoglobin was 96% and carboxyhaemoglobin was 2.0%. He scored 29 points on the mini mental state examination. There were no abnormal findings on brain computed tomography.

Although the patient's carboxyhaemoglobin level was within normal limits, he was diagnosed with acute myocardial injury and rhabdomyolysis caused by carbon monoxide because of the time interval between carbon monoxide inhalation and the emergency department visit. He was treated with crystalloid hydration (Hartmann's solution 160 ml/h).

Although serum creatine phosphokinase and troponin-I levels had decreased to 3642 IU/litre and 1.168 ng/ml respectively on hospital day 3, his chest discomfort continued. The swelling in both forearms and hands had also worsened. Echocardiography and a Tc99-labelled hydroxymethylene diphosphonate bone scan were performed to evaluate cardiac function and rhabdomyolysis on hospital day 4. Echocardiography showed no regional wall motion abnormalities and normal global left ventricular systolic function (ejection fraction 68%). However, the bone scan showed uptake not only in both forearms but also in the heart (*Figure 2*).

He was started on an angiotensin-converting enzyme inhibitor (ramipril 2.5 mg daily) and beta-blocker (carvedilol 3.125 mg twice a day) on hospital day 4. Intravenous hydration was continued. The chest discomfort improved and disappeared by hospital day 8. The uptake in the bone scan had decreased in a follow-up image on hospital day 8. The follow-up electrocardiogram also changed from T-wave inversion to non-specific T-wave abnormality. He was discharged uneventfully.

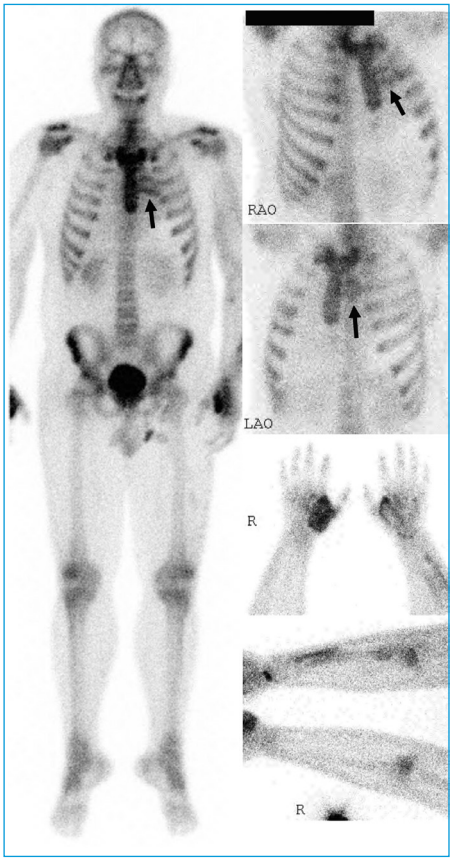

Figure 2. Tc99-labelled diphosphonate bone scan checked on hospital day 4. The black arrows show uptake in the injured myocardium.

show radiolabelling of damaged muscle (Walsh and Fan, 2009). Since Esnault et al (2007) reported that Tc99-labelled hydroxymethylene diphosphonate scintigraphy could localize injured muscles in rhabdomyolysis, bone scans have been used as a non-invasive imaging diagnostic tool for rhabdomyolysis.

However, 99Tc uptake was not limited to the areas of rhabdomyolysis – there was also uptake in the injured myocardium. Although the echocardiogram was normal, the bone scan detected evidence of myocardial injury. This uptake by the heart decreased after the patient's symptoms were relieved. **BJHM**

LEARNING POINTS

- A bone scan simultaneously detected the rhabdomyolysis and myocardial injury caused by carbon monoxide.
- Although the cut-off value and prognostic significance of the bone scan are unclear, it could be considered an imaging modality for the diagnosis of carbon monoxide-induced myocardial injury, especially when combined with rhabdomyolysis and a normal echocardiogram.

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