

Myocardial infarction secondary to unintentional ingestion of hydrogen peroxide

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Abstract

Ingestion of acid-containing household products, either accidentally or as a suicide attempt, is a common form of intoxication. A clear and odorless liquid, hydrogen peroxide is an oxidizing agent found in most households and many industrial environments. Cardiovascular manifestations of hydrogen peroxide ingestion are extremely rare. Here we report a 60 year-old woman with acute inferolateral myocardial infarction (MI) after hydrogen peroxide ingestion, who had no history of coronary artery disease. Physicians dealing with hydrogen peroxide ingestion in the emergency department should be aware of the probability of MI and obtain an electrocardiogram, even if the patient has no cardiac complaint. (Cardiol J 2012; 19, 1: 86–88)

Key words: hydrogen peroxide, intoxication, myocardial infarction

Introduction

A clear and odorless liquid, hydrogen peroxide (HP) is an oxidizing agent found in most households and many industrial products. Used as disinfectants, solutions of HP are typically at low concentrations (3–6%). Higher concentrations (33–35%) are used as bleaching agents [1]. HP can be immediately absorbed through the gastric wall, with a subsequent release of oxygen into the vascular system. When the amount of oxygen exceeds its maximal blood solubility, embolization can occur [2]. HP ingestion can lead to serious toxicity, including death. Although ingesting HP is generally not fatal in adults, oxygen gas emboli resulting from ingestion have been recognized as the cause of brain infarcts, portal venous gas emboli, hemorrhagic gastritis and arterial emboli [3]. We present a case of acute myocardial infarction (AMI) following HP ingestion, which we believe is the first of its kind in the literature.

Case report

A 60 year-old woman was admitted to the emergency room with complaints of nausea, vomiting and discomfort in the form of a feeling of compression on her chest for the previous two hours. She had no other cardiovascular risk factor other than hypertension. On physical examination, blood pressure and heart rate were found to be 150/90 mm Hg and 100 bpm, and there was no additional abnormal finding. The electrocardiogram (ECG) revealed sinus rhythm and ST elevation at inferolateral leads with reciprocal ST segment depression at D1 and aVL (Fig. 1). Emergency cardiac catheterization was performed, which revealed normal epicardial coronary arteries. The control ECG taken in the coronary intensive care unit showed ST segment resolution and negative T waves (Fig. 2). The echocardiogram revealed inferior hypokinesia. The troponin-I value at the seventh hour was 7.3 ng/mL (normal limits < 0.1 ng/mL) having been

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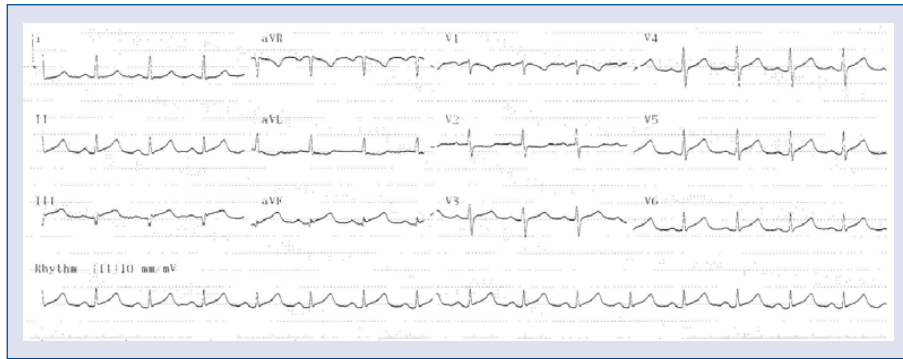


Figure 1. Electrocardiogram of the patient showing ST-segment elevations in leads II, III, aVF, V4, V5 and V6, accompanied by reciprocal ST-segment depressions in leads aVR, aVL, V1 and V2 which is compatible with acute inferolateral myocardial infarction.

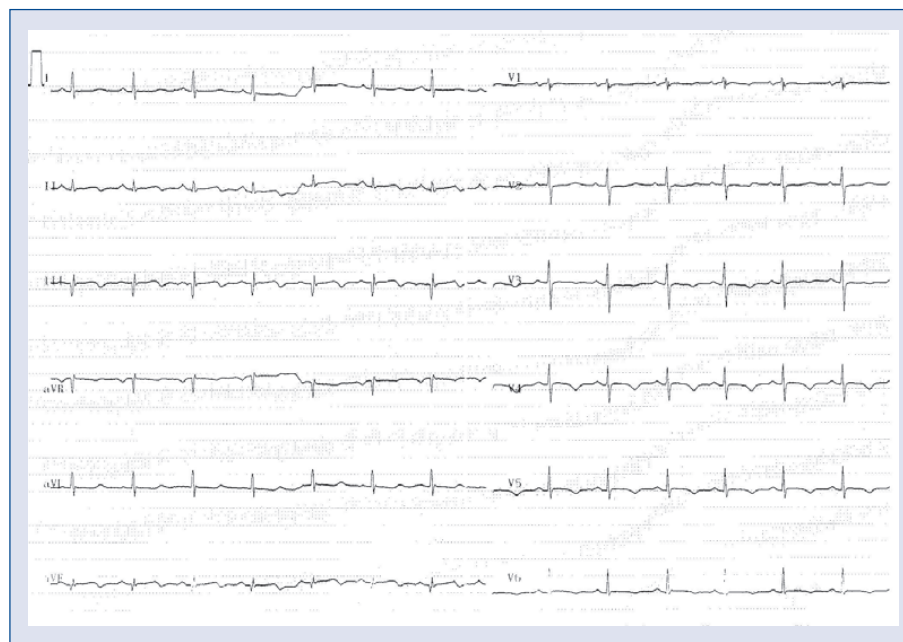


Figure 2. Control electrocardiogram showing ST segment resolution and negative T waves.

non-detectable on admission. All other laboratory parameters (biochemical and hematological) were within normal limits. Chest X-ray showed no evidence of mediastinitis, subdiaphragmatic free air or increased cardiothoracic ratio. When a detailed history was taken, it emerged that the patient had unintentionally drunk a mouthful of a 35% HP solution. The patient completely recovered and was discharged without any complications after three days of hospitalization. At one month follow-up, she was symptom free and her ECG and echocardiogram was found to be normal, with no systolic wall motion abnormality because of early spontaneous reperfusion,

Discussion

Ingestion of HP at low concentrations has usually benign consequences, with mucosal toxicity being the most commonly reported sign. However, ingestion of even small amounts of HP in higher concentrations can be fatal. Previous complications reported following HP ingestion have included hemorrhagic gastritis, respiratory collapse, pneumomediastinum, seizures, portal venous gas, air in the right ventricle, and death [1]. There are three proposed mechanisms for arterial gas embolism following HP ingestion. Formed oxygen bubbles in the venous system could pass directly to the

arterial system through a cardiac defect. Secondly, oxygen bubbles or undissociated HP could pass through a pulmonary arterio-venous fistula or the pulmonary capillary bed. Or finally, aspiration could result in HP being absorbed inside the pulmonary veins [1]. Small gas emboli in skeletal muscle or visceral vessels are usually well tolerated. But cerebral, coronary and pulmonary emboli can result in serious morbidity and mortality.

Many reports have described the hazards of an application of HP. Ghai and Martin [2] reported a portal vein gas resulting from ingestion of HP, where a 31 year-old woman accidentally drank a mouthful of 35% HP. Moon et al. [4] reported a 25 year-old woman who ingested one mouthful of 3% HP and presented to the emergency room with persistent vomiting and epigastric pain. Bollen et al. [5] reported a case of a 13 year-old girl who developed colonic ulceration and rectal bleeding after receiving an enema containing 10% HP and 1 L of water to resolve her constipation. Another study reported a patient who unintentionally ingested 33% HP and presented with symptoms and magnetic resonance imaging findings suggestive of multiple cerebral arterial gas emboli [1]. We present a case of AMI following HP ingestion which we believe is the first such report in the literature.

In our presented case, our patient was admitted to hospital because of inferolateral AMI with normal coronary arteries following HP ingestion. Coronary angiography was immediately performed as a result, but no thrombotic occlusion was observed. Although they have normal epicardial coro-

nary arteries in angiography, some patients were exposed to AMI. This can be explained by coronary artery spasm, as in our case. We have not tested for coronary spasm because it is contraindicated in a setting of AMI. Furthermore, even if testing is negative, it does not rule out the possibility of coronary spasm.

Conclusions

Despite extremely low cardiovascular manifestations of HP ingestion, such ingestion can lead to AMI that can be fatal. Physicians dealing with HP ingestion in the emergency department should be aware of this possibility and always obtain serial ECG, even if the patient has no cardiac complaint.

Conflict of interest: none declared

References

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