Cardiac Decompression Sickness After Hypobaric Chamber Training: Case Report of A Coronary Gas Embolism

Alçak Basınç Çemberi Eğitimi Sonrasında Gelişen Kardiyak Dekompresyon Hastalığı: Koroner Gaz Embolismi Olgusu Takdimi

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Introduction

Air embolism is an uncommon but potentially catastrophic event, which occurs as a consequence of the entry of air into the vasculature. Surgery, instrumentation of the central venous system, positive pressure ventilation, trauma and decompression are the most common causes of air embolism. Decompression sickness is an illness caused by reduced pressure on the body that results in formation of bubbles of an inert gas and specific related symptoms. Decompression sickness is still a risk for both aviators and divers (1). Here we report a cardiac decompression sickness case due to air embolism.

Case Report

A 22-year-old male pilot candidate was evaluated for sudden dyspnoea and chest pain after hypobaric chamber training (Fig. 1). This training is given in order to simulate high altitude hypoxia. The pilot candidate had periodic medical examination, which revealed to be completely normal prior to hypobaric chamber training. He was exposed to hypobaric environment for about one hour (total time for ascending and descending) staying at the maximum 35,000 feet atmospheric pressure for about 15 min. Two hours after the training he was transferred to emergency department because of chest pain at rest typical of myocardial infarction (MI). On admission, he was anxious with profuse sweating. He was normotensive, nondiabetic, nonsmoker and had no family history of coronary artery disease. Blood pressure was 140/90 mmHg, heart rate was 90 beats per minute. Physical examination was unremarkable. His electrocardiogram (ECG) revealed ST segment elevation in the derivations DII, DIII, AVF, V5, V6; ST segment depression in DI and aVL (Fig. 2). Considered as an acute MI case due to decompression cardiac sickness (DCS), he was immediately taken into hyperbaric chamber (Fig. 3), because it is a general rule for decompression sickness that diagnostic procedures must not cause a delay in the specific treatment. The patient denied any risk factors known for DCS such as SCUBA diving, strenuous exercise or Rapid Decompression (cabin depressurization) in the previous days.

He was given hyperbaric oxygen therapy (HBOT) according to US-Navy Treat: Table 6 (2), aggressive hydration and 100% oxygen breathing with a tight fitting mask (3), resulting in rapid resolution of the symptoms at 15th minute of HBOT. After HBOT ECG disclosed changes compatible with an acute inferolateral MI (1 mm ST segment elevations and significant decrease in R amplitude (poor R wave progression) in DII, DIII, aVF, V5, V6; 0.5 mm ST segment depression and increased T amplitude in V1 and V2 derivations) (Fig. 4). On admission to coronary care unit, the patient was stabilised and free of chest discomfort, he was then monitored. He experienced ventricular extrasystoles and rare couplet forms suggestive of reperfusion. Chest X-ray, complete blood count and blood chemistry (including lipid profile) other than cardiac enzymes were within normal limits. Since he was considered as decompression sickness and responded to HBOT dramatically, no additional medication, including antiaggregant, anticoagulant, thrombolytic or antiischemic drugs were administered. Creatine phosphokinase, creatine phosphokinase MB fraction and aspartate aminotransferase enzymes presented early peak at 24th hour and early wash-out compatible with early reperfusion. In serial ECGs, ST segment elevations and decreased R amplitude in lateral derivations regressed and ST segment elevations in inferior derivations returned to baseline. He was discharged on the 7th day of hospitalisation having no other symptoms and complications.

Two months after discharge, transthoracic echocardiography revealed inferobasal hypokinesia and mild mitral insufficiency. Ejection fraction was slightly below normal limits. A symptom-limited exercise ECG performed up to Bruce stage V, showed no evidence of myocardial ischaemia and hyperventilation test was also normal. Myocardial perfusion scintigraphy with Thallium 201 was normal. Coronary angiography disclosed neither lesion nor coronary anomaly. Left ventriculography revealed mild hypokinesia in posterobasal segments. Three months after the episode he was allowed for full flight

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duties. The rationale for this decision was the intact structure of coronary arteries. He has been in active duty as a fighter pilot for 3 years without any cardiac event.

Discussion

Decompression sickness may cause potentially fatal outcomes by means of gas embolism. Although it is mainly observed in divers after rapid ascent, it may also occur in aviators during high altitude flights or simulated training conditions. Decompression sickness, also known as “bends”, was originally described as "caisson disease" when it was first recognised in 1843 among tunnel workers following return from the compressed environment of the caissons to the normal atmospheric pressure (2).

Figure 1. Hypobaric chamber

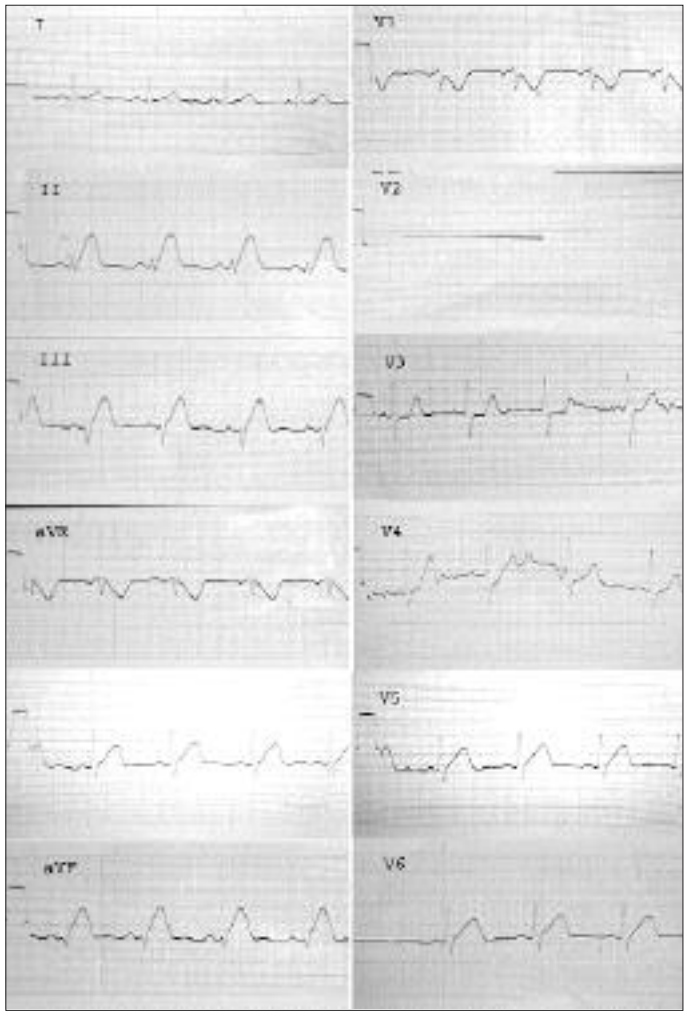

Figure 2. Patient’s initial electrocardiogram on admission.

Under higher atmospheric pressures the tissues become loaded with increased quantities of oxygen and nitrogen. As atmospheric pressure decreases, ie. while divers ascend to surface or aviators climb up to higher altitudes, the sum of the gas tensions in the tissue may exceed the ambient partial pressure of the gas and lead to the liberation of free gas from the tissues in the form of bubbles. The liberated gas bubbles can alter organ function by blocking vessels, rupturing or compressing tissue, or activating clotting and inflammatory cascades (3).

Overall incidence of DCS occurring after hypobaric chamber training was found about 0.19-0.32 (4). Among these cases incidence of cardiac DCS cases, which are characterized with coronary gas embolus or cardiovascular collapse is 0.2% (5). However a detailed description of such a case was not available in the medical literature.

Approximately 75% of patients with decompression sickness develop symptoms within 1 hour and 90 percent within 12 hours of exposure; only a small number of the cases become symptomatic after 24 hours. Symptoms differ according to organ systems involved (6). Although it is suspected that there is a tendency for a person who develops DCS under given conditions to again develop DCS under similar circumstances, it has not been proven yet. It is also interesting that bubble formation may not always result in symptoms or embolus (7).

Since sudden loss of cabin pressure is a major risk for DCS, pilots and candidates who take this type of training in

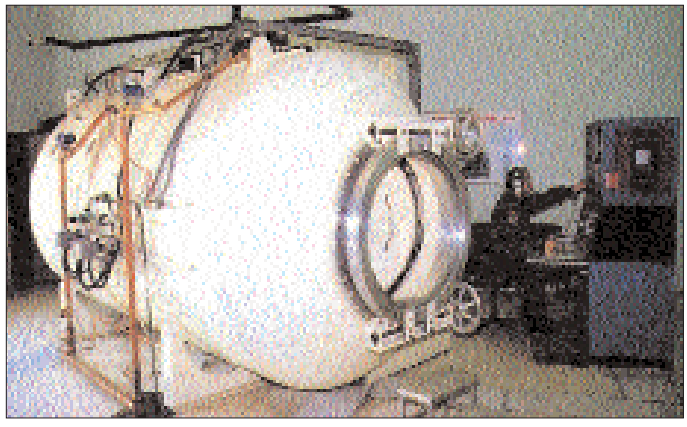

Figure 3. Hyperbaric oxygen chamber

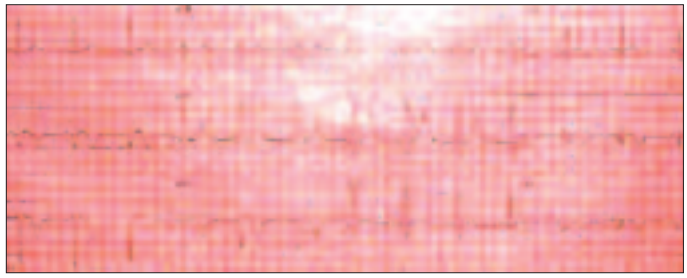

Figure 4. Patient’s electrocardiogram after hyperbaric oxygen therapy

hypobaric chamber must adequately be informed of these dangers. Sudden cabin explosion of airline transport planes at high altitudes generally would not pose a serious risk because of the rapid descent, however signs or symptoms of MI occurring after such an event must be evaluated carefully.

Atherosclerosis of coronary arteries resulting in MI is still one of the most important causes of morbidity and mortality. Besides atherosclerosis, gas and air emboli are among rare causes of MI. As little as 0.5 ml of air in the coronary circulation can lead to dysrhythmias, myocardial infarction, and/or cardiac arrest. Gas emboli are encountered in divers and aviators due to decompression sickness; whereas the most common causes of air emboli in daily clinical practice are surgery (especially open heart surgery), trauma, central venous catheterisation, barotrauma due to positive pressure ventilation, cardiac catheterisation and ruptured angioplasty balloon (8).

Therapeutic approach in management of MI differs greatly in case of gas or air emboli; therefore it must be kept in mind to prevent potentially fatal outcomes in patients with compatible history.

The primary aims of the treatment are identification of the source of air or gas, prevention of further embolisation, removal of embolised gas and restoration of circulation. Nitrogen washout by means of high flow supplemental oxygen with a tight fitting mask, supine positioning, supportive measures in addition to HBOT are the main therapeutic strategies. Hyperbaric oxygen therapy reduces air bubble size, accelerates nitrogen resorption, and increases the oxygen content of arterial blood, potentially ameliorating ischaemia. Although prompt initiation of HBOT is preferred, it may improve outcome even if delayed up to 30 hours (9).

Acute MI in our patient was considered to be due to gas embolus. Being evaluated medically normal prior to hypobaric chamber training, absence of cardiac risk factors and underlying systemic disease, development of symptoms two hours after decompression and dramatic response to recompression were the key factors in diagnosis. Time-gap between decompression and onset of the symptoms is due to circulating silent bubbles before lodging.

Clinical causes of air/gas emboli such as open heart surgery, trauma, pulmonary barotrauma, cardiac catheterisation and ruptured angioplasty balloon can be seen in daily practice. In such cases HBOT will be helpful as well. In order to better understand the mechanisms acting in the pathological process of Cardiac Decompression Sickness, controlled experimental studies should be planned.

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