The Journal of Emergency Medicine, Vol. 50, No. 2, pp. 277–280, 2016

0736-4679/$ - see front matter

ELSEVIER

http://dx.doi.org/10.1016/j.jemermed.2015.09.045

Selected Topics: Sports Medicine

STRESS CARDIOMYOPATHY CAUSED BY DIVING: CASE REPORT AND REVIEW OF THE LITERATURE

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☐ **Abstract—Background: Stress cardiomyopathy is characterized by transient myocardial dysfunction that mimics a myocardial infarction in the absence of obstructive coronary artery disease. The onset is frequently triggered by an acute illness or intense physical or emotional stress. Case Report: We describe the case of a 47-year-old woman who was brought to the emergency department with acute onset shortness of breath while scuba diving. She was found to have acute pulmonary edema radiographically. Her troponins were noted to be positive. Initial echocardiogram showed basal hypokinesis with hyperkinesis of apex. She was treated with noninvasive ventilation and intravenous diuretic therapy and her symptoms significantly improved. She subsequently underwent cardiac catheterization which revealed nonobstructive coronary artery disease. An exercise stress echocardiogram was performed 2 days later that revealed resolution of the wall motion abnormality and no ischemia at high levels of exercise. A diagnosis of reverse stress (Takotsubo) cardiomyopathy was made based on Mayo Clinic Diagnostic criteria. *Why Should an Emergency Physician Be Aware of This?*: This case brings to light the risk of stress cardiomyopathy in divers. The diagnosis should be considered in patients presenting with acute pulmonary edema during diving. © 2016 Elsevier Inc.**

☐ **Keywords—acute coronary syndrome; cardiomyopathy; diving; pulmonary edema; stress; stress-induced cardiomyopathy**

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INTRODUCTION

Takotsubo cardiomyopathy—also known as stress-induced cardiomyopathy, broken heart syndrome, and apical ballooning syndrome—consists of transient myocardial systolic dysfunction that is frequently triggered by intense physical or emotional stress (1,2). It mimics a myocardial infarction and is accompanied by electrocardiographic changes and elevated cardiac enzymes, but in the absence of obstructive coronary artery disease. The most common pattern of cardiomyopathy is mid- and apical hypokinesis with hyperkinesis of the base leading to apical ballooning and resembling the shape of an octopus trap, therefore called Takotsubo (an ancient octopus trap used in Japan). Less commonly, there may be hypokinesis of the mid-ventricle or hypokinesis of the base with sparing of the mid-ventricle and apex, also referred to as inverse or reverse Takotsubo (3). There have been few reported cases of Takotsubo cardiomyopathy associated with diving (4,5). We describe a case of stress cardiomyopathy caused by diving and review the available literature on this subject.

RECEIVED: 27 September 2015;
ACCEPTED: 30 September 2015

CASE REPORT

Our patient is a 47-year-old woman who was undergoing lessons in recreational SCUBA diving at a local quarry. She had submerged for her fourth dive, and while about 30 feet beneath the surface of the water she became acutely anxious and short of breath. With the help of her instructor, she quickly ascended to the surface with persistent shortness of breath. Emergency medical services were called and she was placed on continuous positive pressure ventilation (CPAP) because of respiratory distress and hypoxia. On arrival to the emergency department, her blood pressure was 150/110 mm Hg, her heart rate was 119 beats/min, her respiratory rate was 28 breaths/min, and her O_2 saturation was 87% on CPAP. On examination, she was tachypneic with diffusely decreased breath sounds and basal rales on auscultation. She was maintained on CPAP and was given a dose of intravenous furosemide. A chest radiograph revealed pulmonary edema (Figure 1). An electrocardiogram performed at that time found sinus tachycardia with mild ST depressions in the inferior and anterolateral leads (Figure 2). Her initial troponin level was 0.09 (normal value, <0.04) and her probrain natriuretic peptide was 579 pg/mL (normal value, <125 pg/mL). Subsequently, her troponin trended up to 0.88 and peaked at 1.10. Based on her symptoms and troponin elevation, it was thought that she had an acute coronary event. An echocardiogram was performed that revealed hypokinesis of the basal left ventricular segments and hyperkinesis of the apex (Video 1). She was taken for an emergent cardiac catheterization, and coronary angiography revealed mild nonobstructive coronary artery disease and normal blood flow. Intravascular ultrasonography was also performed to rule out unstable plaque. There was no dissection and only soft plaques were noted. Over the course of a couple of days, her symptoms improved with diuretic therapy. A follow-up exercise stress echocardiogram was performed 2 days later. She exercised on Bruce protocol for 9 min 26 seconds, indicating good functional capacity. She did not experience dyspnea or chest pain during exercise. An echocardiogram revealed resolution of previously noted wall motion abnormality and no ischemia at peak exercise (Video 2). The final impression of this case was that the patient had suffered from a stress cardiomyopathy caused by the stress of SCUBA diving. She was discharged home on aspirin, statins, and beta-blocker therapy with subsequent follow-up arranged with cardiology.

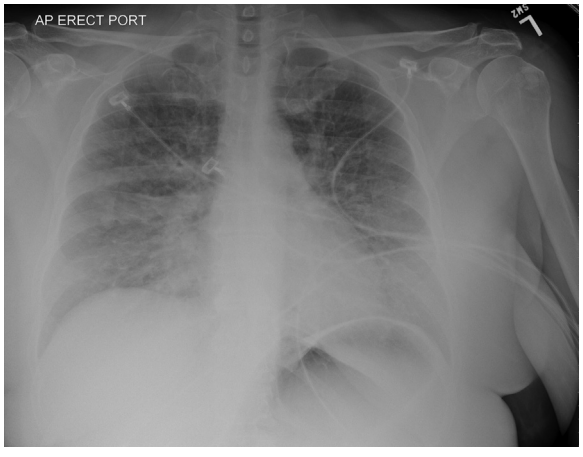

Figure 1. Chest radiograph revealing acute pulmonary edema.

DISCUSSION

Our case describes the potential of stress cardiomyopathy and associated pulmonary edema occurring with diving in an otherwise healthy individual without evidence of heart disease. Postulated mechanisms involved in pathogenesis of stress cardiomyopathy include catecholamine excess, microvascular dysfunction and vasospasm (6–8). We performed a literature search through PubMed and Medline of all available English language articles pertaining to stress cardiomyopathy and diving. We came across only 2 articles addressing this issue (4,9).

Chenaitia et al. reported a case of Takotsubo cardiomyopathy in a 51-year-old woman after diving to a depth of 35 meters. She presented with chest discomfort, pulmonary edema, an abnormal electrocardiogram, and elevated cardiac markers. Her echocardiographic results revealed apical ballooning with hyperkinesis of the basal portion of the left ventricle. She had normal coronaries on coronary angiography. She recovered full myocardial function with no wall motion abnormalities on follow-up.

Most recently Gemppe et al. published a case series describing determinants of pulmonary edema in 54 consecutive divers (9). Of these, 15 had transient myocardial dysfunction in the form of wall motion abnormalities and reduced left ventricular ejection fraction. Most of the wall motion abnormalities were limited to vascular territories. This may suggest that rapid decompression syndrome with air emboli may have contributed to the presentation. However, they reported 3 cases that had global left ventricular hypokinesis that reversed to normal on follow-up. These cases perhaps represent true stress-induced cardiomyopathy. The authors proposed that sympathetic activation with catecholamine excess and tissue hypoxia during diving might contribute to this syndrome. Female sex was not associated with an increased risk of pulmonary edema and cardiomyopathy in this case series. This is in contrast to the published data for Takotsubo cardiomyopathy, in which women account for 80% to 100%