Immersion Pulmonary Edema in the Setting of Takotsubo Cardiomyopathy

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Immersion Pulmonary Edema (IPE) is a unique medical condition being increasingly described in the medical literature as sudden-onset edema in the setting of scuba diving and/or swimming. We report on three patients with unique presentations of IPE with associated development of Takotsubo cardiomyopathy (TTC). All three cases occurred in Oahu, Hawaii and were seen by the same cardiologist within a span of seven years.

Each patient was scuba diving with sudden onset dyspnea with pulmonary edema on chest x-ray. Cardiac catheterization revealed no significant epicardial stenosis or thrombosis. EKGs showed typical evolution of symmetric T wave inversion. Wall motion abnormalities resolved. IPE and TTC may occur together and may be more common than initially thought. Physical and emotional stressors are known to trigger TTC. TTC should be considered as a possible complication of IPE. Initial workup should include EKGs, cardiac enzymes, echocardiogram and, in the appropriate situation, cardiac catheterization.

Takotsubo cardiomyopathy (TTC) was first described in Japan in 1991, named for the unique left ventricular morphological features.1,2 Takotsubo is a Japanese word that translates to “octopus pot.” Also known as Apical Ballooning Syndrome and Broken Heart Syndrome, TTC is a type of acquired stress cardiomyopathy. This syndrome is now being increasingly recognized in the literature and the American Heart Association has formally incorporated this disease in the class of acquired cardiomyopathies. Usually seen in perimenopausal or menopausal women, TTC is stimulated by a type of emotional or physical stress.3 Presentation often mimics ST-Elevation Myocardial Infarction, but without significant coronary artery stenosis. Based on retrospective studies, approximately 2% of all patients with suspected acute coronary syndrome were found to have TTC.3,4

There is no consensus on diagnostic criteria for TTC. The Mayo Clinic proposed diagnostic criteria in 2004 and this has been commonly adopted throughout the literature.5 This criterion is used by the International Takotsubo Registry (InterTAK Registry).

Scuba divers’ pulmonary edema (SDPE) is a unique medical condition being increasingly described in the medical literature as sudden-onset pulmonary edema in the setting of scuba diving. SDPE is one type of Immersion Pulmonary Edema (IPE), which presents with shortness of breath, tachypnea, fatigue, and cough with frothy white or blood-tinged sputum.6 A 62-year-old Italian-Mexican female was in her normal state of health prior to a dive where she followed appropriate ascent rates of no more than one foot every two seconds (30ft/9m per minute) and made necessary decompression stops. On ascent, she experienced dyspnea and cough. When she reached the surface, she was given an oxygen mask and coughed up white frothy fluid with blood-tinged sputum. There was no aspiration of seawater, chest pain, or palpitations.

She initially presented with an oxygen saturation of 82% on non-rebreather oxygen mask. Her lung examination was significant for diffuse bilateral rhonchi. Electrocardiogram showed deep pathologic Q waves in V1, V2, and V3 and T wave inversions in lead I, aVL, and V6. Initial troponin I value was 0.69 μg/L and peaked at 0.84 μg/L. Other significant lab values on admission were Creatine Kinase 523 U/L, Brain Natriuretic Peptide (BNP) 570 ng/L, and mild leukocytosis. Transthoracic echocardiogram (TTE) showed an ejection fraction of 32% and severe global hypokinesis of the left ventricle. She underwent left heart catheterization, selective coronary arteriography, and left ventriculography. There was no evidence of coronary artery disease. There were findings of apical ballooning with hyperkinetic base supportive of TTC (Figure 1). Her ejection fraction at that time was 45% measured by planimetry. The patient was discharged home in stable condition. One month later, she had a repeat TTE. There were no wall motion abnormalities, apical hypokinesis or apical ballooning. Ejection fraction improved to 55%.

The next patient is a 60-year-old Caucasian male with no history of coronary artery disease and was in his normal state of health prior to the dive. When he reached the water’s surface, he was dyspneic...
and immediately began coughing up brown-tinged foamy sputum. There was no aspiration of seawater, chest pain, or palpitations. The patient led an active lifestyle and had a negative treadmill stress test (12 minutes on Bruce protocol) done two months prior. On admission, his Creatine Kinase-MB (CK-MB) was 4.7 ng/ml, Troponin-I 0.04 ug/L, BNP 137 ng/L. Troponin-I peaked at 0.53 ug/L and CK-MB levels at 8.7 ng/ml. Chest x-ray showed pulmonary edema suggestive of congestive heart failure. Electrocardiogram showed regular sinus rhythm with no ST or T wave changes. TTE showed a severe deterioration of left ventricular ejection fraction to 25% with global hypokinesis in his left chamber with characteristic apical ballooning. Subsequent cardiac catheterization showed an estimated ejection fraction of 60% with mild anterior hypokinesis and no significant coronary artery disease. The patient was discharged the next day in stable condition.

The third patient is a 47-year-old Japanese female who ascended per diving protocol and was noted to be hyperventilating. She successfully surfaced, but lost consciousness while boarding the vessel. Oxygen saturation was found to be 88% on room air on arrival to the hospital. On admission, her CK-MB was 39 ng/ml, Troponin-I 0.49 ug/L, BNP 46 ng/L. Troponin-I peaked at 3.49 ug/L. Echocardiogram revealed normal sinus rhythm with mild increase in QTC to 479, left ventricular hypertrophy, 1-mm ST elevations in leads V1, V2, and V3, ST depressions in leads V4, V5, and V6, and T wave inversions in lead V2. Chest x-ray was significant for bilateral mild to moderate infiltrates. On admission, his Creatine Kinase-MB (CK-MB) was 4.7 ng/ml,

References