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CASE REPORT
Inherited cardiac conditions/cardiomyopathies

Sudden cardiac death during scuba diving: a case report of a patient with unknown hypertrophic cardiomyopathy

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Background Scuba diving is a recreational activity usually considered at low impact on cardiovascular system. However, when diving, increased ambient pressure exerts several effects on the cardiovascular and pulmonary systems, mainly due to redistribution of peripheral blood into the central circulation. This phenomenon, also known as blood shift, may produce a significant overload on a non-healthy heart.

Case summary We present the case of a female patient who experienced sudden cardiac death during scuba diving: post-mortem cardiac magnetic resonance and autopsy revealed that the patient was affected by previously unknown hypertrophic cardiomyopathy.

Discussion Diving exposes the body to significant physiological changes that may overstress a diseased heart. This case suggests the need for some cardiovascular exams, such as an echocardiogram or, at least, an electrocardiogram, for screening cardiovascular abnormalities in subjects who wish to practice scuba diving.

Keywords Case report • Scuba diving • Hypertrophic cardiomyopathy • Sudden death

ESC curriculum 8.1 Sports Cardiology • 9.7 Adult congenital heart disease

Learning points

- An echocardiogram may reveal cardiac abnormalities that can increase the risk of adverse events when diving that could be done for primary prevention.

Introduction

Scuba diving is a water sport that involves breathing air from a tank while underwater. Scuba is an acronym for Self-Contained Underwater Breathing Apparatus. Technical certification for the use of this apparatus is recommended, while no clinical evaluation is usually required. Nonetheless, scuba diving may impose a significant stress on the cardiovascular system. Our understanding of the cardiac stressors induced by scuba diving is still incomplete.[1,2] During scuba diving, the subjects breathe a gas delivered to a pressure adapted to the ambient pressure, which counteracts the transfer of blood towards the thorax induced by water immersion. Left ventricular (LV) preload even seems to decrease during scuba diving, as demonstrated by a significant reduction in left atrial diameter, LV end-diastolic diameter, and stroke volume.[1] The decreasing ambient pressure during the ascent promotes blood return to the lungs and increases LV preload.[3] The shifts in

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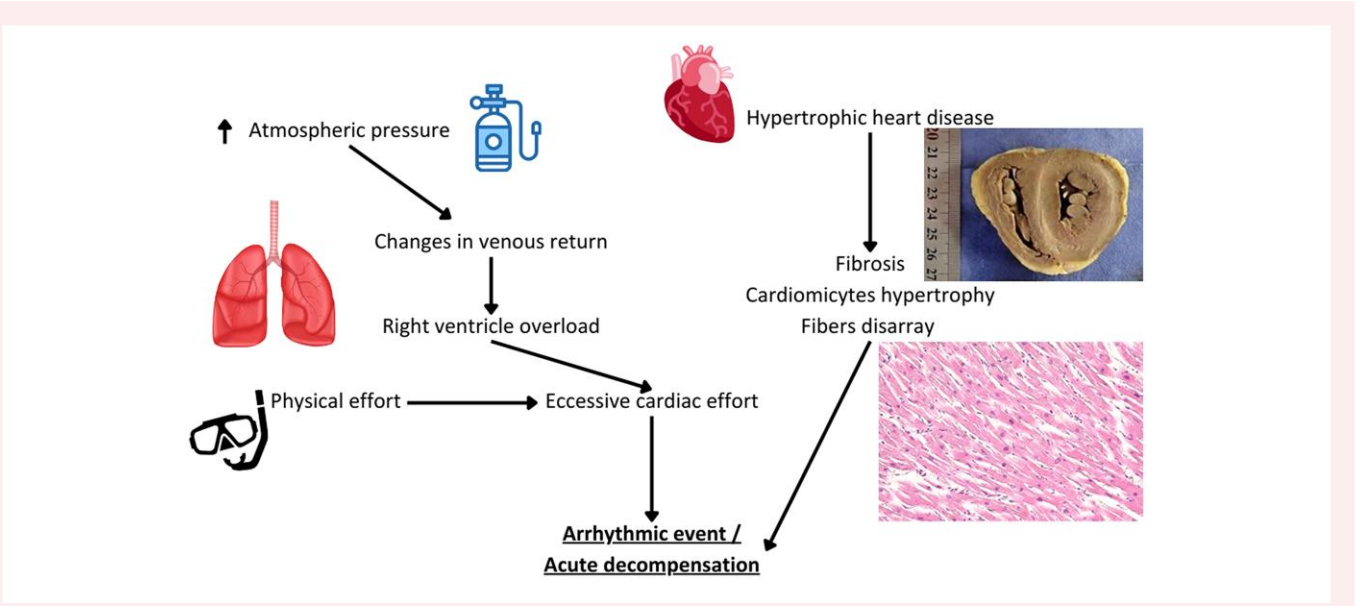
central haemodynamics may be particularly dangerous in patients with subclinical cardiac disorders. For example, patients with hypertrophic cardiomyopathy (HCM) can display an impaired diastolic filling causing a fall in cardiac output when the preload decreases, and a steep increase in pulmonary pressures when the preload increases.[4,5] These patients are then susceptible to tissue hypoperfusion during the dive, and to pulmonary oedema during a rapid ascent. We may add that HCM has an estimated prevalence of 1 out of 500 in the general population, often affects young individuals, and is also associated with an increased risk of ventricular arrhythmias,[6] possibly elicited by the haemodynamic stress due to scuba diving. The risks of this sport in individuals with HCM are clearly shown by the case we are presenting.

Summary figure



Case presentation

A 53-year-old Caucasian woman with unremarkable clinical history underwent a training for advanced scuba diving without any specific clinical evaluation, except for a standard visit with the GP and an electrocardiogram, both resulted normal, as required by Italian legislation for non-competitive sports activities.[7] She had already completed a basic scuba diving course (maximum depth of 18 m) without any problem. Once surfaced after her first dive at 30 m, the woman displayed a transient congested cough and rales disappearing after a few minutes. Roughly 15 min into her second dive, the woman started experiencing nausea. Her instructor assisted her in conducting a swift ascent. While at a depth of 12 m, the woman's condition deteriorated rapidly. Upon reaching the surface, she experienced intense bouts of coughing and proceeded to vomit a yellowish mucus. Subsequently, she lost consciousness. When emergency services arrived, the patient had no pulse and no shockable rhythm. She was successfully resuscitated through basic life support manoeuvers but did not regain consciousness. A contrast-enhanced computed tomography scan of the brain was suggestive of anoxic brain damage, while there was no evidence of air embolism.

At the time of hospitalization, an echocardiogram was not immediately performed because the woman promptly underwent oxygen therapy in a hyperbaric chamber. Troponin values showed pathological levels (T 416.0 ng/L) while, in the following 2 days, two echocardiograms were performed, documenting respectively a non-dilated left ventricle with wall thickness at higher normal values, with hypokinesia of the base segments and hyperkinesia of the apical segments (suspected reverse Takotsubo), and then, an akinesia of the basal segments and normal contractility of the apex. Both examinations, however, did not show signs of HCM.

From the interview with the family, it emerged that the patient did not suffer from pathologies and did not usually take medications. The use of oral contraceptives was also excluded, and no history of familial cardiomyopathies was reported.

After 4 days in the intensive care unit, brain death was declared.

Because of the suspicion of cardiac hypertrophy on the echocardiogram, a post-mortem magnetic resonance scan was performed. The exam confirmed LV concentric hypertrophy (*Figures 1* and *2*) and showed myocardial fibrosis in the most hypertrophied area as well as extensive oedema (as a sign of acute myocardial damage; *Figure 2*).[8–10]

On macroscopic examination, the heart weighed 300 g and wall thickness was up to 20 mm (in the basal septum) and 18 mm (in the basal anterior and lateral segments; *Figure 3*). No abnormalities of the heart valves or coronary arteries were noted.

Histological examination of the myocardial tissue showed diffuse hypertrophy and disarray of cardiomyocytes with foci of fibrosis and mid-adventitial thickening of subendocardial arterioles (*Figure 4*).

In conclusion, the autopsy findings combined with evidence provided by microscopic examinations of the lungs have allowed us to highlight the presence of a massive acute pulmonary oedema. This condition was deemed to be secondary to the rapid ascent from the dive, which, however, was carried out by the woman due to the acute cardiac symptoms she had developed.

Discussion

We describe a case of a woman with previously unknown hypertrophic cardiomyopathy who suddenly died during scuba diving. According to the 'The Sports and Fitness Industry Association 2015' report, 3 million people in the USA join scuba dives at least once per year; the rate of mortal accidents is about two cases per 100 000 dives.[11] Sudden

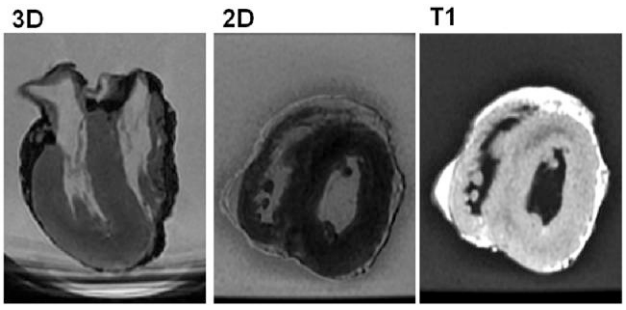


Figure 1 Post-mortem cardiac MRI images. Four-chamber view (left) and short-axis view (centre); steady state free precession (SSFP) acquisition; increased T1 signal in the mid-wall portion of the anterior septum (right).

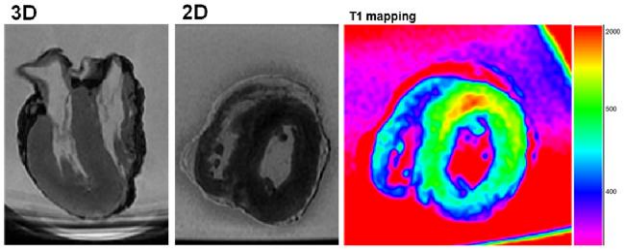


Figure 2 T1 and T2 mapping analysis of the explanted heart. Increased myocardial T1 in the mid-wall layer of the anteroseptal and anterior wall. Myocardial T2 is also increased in the same regions, as well as in the inferior septum.

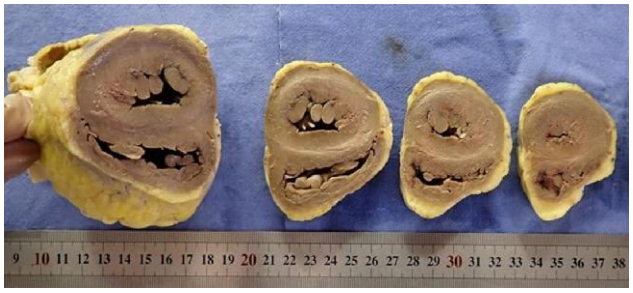


Figure 3 Macroscopic examination. Consecutive longitudinal cardiac sections.

cardiac death (SCD) accounts for 20–30% of fatal cases.[12] No epidemiological data are available, but acute pulmonary oedema and ventricular arrhythmias account for many of these cases. In a case series, approximately one-fourth of SCD cases had LV hypertrophy.[13] Transthoracic echocardiography has been proposed to become part of a screening examination before scuba diving, to detect LV hypertrophy:[14] the value of transthoracic echocardiography could be helpful in detecting cardiac abnormalities, valvular defects, or patent foramen ovale, which occurs in at least 25% of the population.[15] Alternatively, an initial screening with an electrocardiogram followed by echocardiogram only in presence ECG (electrocardiogram) abnormalities has been also proposed as a more cost-effective approach.[16,17] Electrocardiogram plays a fundamental role in the diagnosis of cardiomyopathies, acute coronary syndromes and in the identification of pathological conditions that may lead to sudden death.[18] In particular in hypertrophic heart disease, the ECG performed at rest may show deviation of the repolarization axis to the left, signs of left ventricular hypertrophy or abnormalities in ventricular repolarization (namely negative T waves) raising the suspicion of a cardiac disease and indicating the need for further investigations: however, in some cases, the ECG can also be normal.

The transthoracic echocardiogram (TTE) and the ECG therefore represent two fundamental and complementary tests that can help identify HCM. In particular, the TTE is believed to represent an even more important test that can detect wall thickness increase even when electrocardiographic findings are negative.

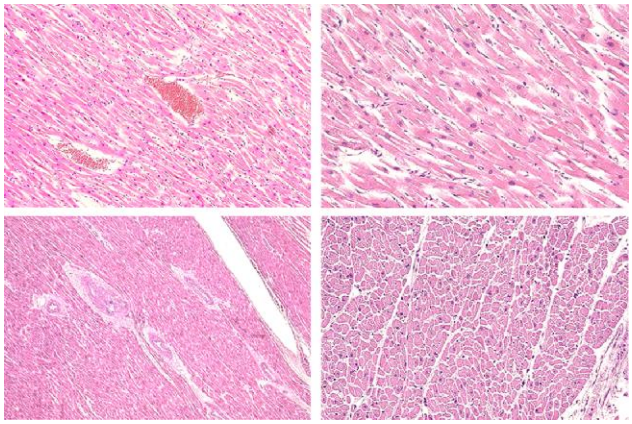


Figure 4 Disarray of myocardial fibres.

Concerning the mechanism of arrhythmogenesis during scuba diving, it can be hypothesized that the increased venous return may have produced and increased the pressure in the lungs, leading to additional stress on an already affected heart that may have triggered arrhythmias and/or acute decompensation.

In conclusion, diving exposes the body to significant physiological changes that may overstress a diseased heart. Our case report suggests that people who want to practice sports such as scuba diving should undergo deeper diagnostic examination, including at least clinical history and ECG in order to exclude previously undiagnosed cardiac pathologies. Adding an echocardiogram may result in a more efficient recognition of cardiac abnormalities reducing the risks of adverse events when diving.

Lead author biography

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Consent: The patient reported in this case is deceased. The authors have been unable to contact the patient's next-of-kin to obtain consent for publication, but every effort has been made to anonymize the case. This situation has been discussed with the editors.

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Data availability

The data underlying this article cannot be shared publicly due to Italian Law limitation. The data will be shared on reasonable request to the corresponding author.

References

1. Marabotti C, Scalzini A, Menicucci D, Passera M, Bedini R, L'Abbate A. Cardiovascular changes during SCUBA diving: an underwater Doppler echocardiographic study. *Acta Physiol (Oxf)* 2013;**209**:62–68.
2. Marabotti C, Piaggi P, Menicucci D, Passera M, Benassi A, Bedini R, et al. Cardiac function and oxygen saturation during maximal breath-holding in air and during whole-body surface immersion. *Diving Hyperb Med* 2013;**43**:131–137.
3. Boussuges A, Rostain JC. What cardiovascular changes during SCUBA diving? *Acta Physiol (Oxf)* 2013;**209**:9–10.
4. Leite-Moreira AF. Current perspectives in diastolic dysfunction and diastolic heart failure. *Heart* 2006;**92**:712–718.
5. Rapezzi C, Aimo A, Barison A, Emdin M, Porcari A, Linhart A, et al. Restrictive cardiomyopathy: definition and diagnosis. *Eur Heart J* 2022;**43**:4679–4693.
6. Husser D, Ueberham L, Jacob J, Heuer D, Riedel-Heller S, Walker J, et al. Prevalence of clinically apparent hypertrophic cardiomyopathy in Germany—an analysis of over 5 million patients. *PLoS One* 2018;**13**:e0196612.
7. Pelliccia A, Sharma S, Gati S, Bäck M, Börjesson M, Caselli S, et al. 2020 ESC guidelines on sports cardiology and exercise in patients with cardiovascular disease. *Eur Heart J* 2021;**42**:17–96.
8. Aquaro GD, De Gori C, Faggioni L, Parisella ML, Cioni D, Lencioni R, et al. Diagnostic and prognostic role of late gadolinium enhancement in cardiomyopathies. *Eur Heart J Suppl* 2023;**25**:C130–C136.
9. Aquaro GD, De Gori C, Faggioni L, Parisella ML, Aringhieri G, Cioni D, et al. Cardiac magnetic resonance in Fabry disease: morphological, functional, and tissue features. *Diagnostics (Basel)* 2022;**12**:2652.
10. Aquaro GD, Masci P, Formisano F, Barison A, Strata E, Pingitore A, et al. Usefulness of delayed enhancement by magnetic resonance imaging in hypertrophic cardiomyopathy as a marker of disease and its severity. *Am J Cardiol* 2010;**105**:392–397.
11. The Sports and Fitness Industry Association (SFIA), Report 2015. https://sfia.org/resources/all-sfia-individual-single-sport-research-reports-are-now-available.
12. Denoble PJ, Caruso JL, Dear GDL, Pieper CF, Vann RD. Common causes of open-circuit recreational diving fatalities. *Undersea Hyperb Med* 2008;**35**:393–406.
13. Buzzacott P, Anderson G, Tillmans F, Grier JW, Denoble PJ. Incidence of cardiac arrhythmias and left ventricular hypertrophy in recreational scuba divers. *Diving Hyperb Med* 2021;**51**:190–198.
14. Shenasa M, Shenasa H. Hypertension, left ventricular hypertrophy, and sudden cardiac death. *Int J Cardiol* 2017;**237**:60–63.
15. Koutroulou I, Tsivgoulis G, Tsalikakis D, Karacostas D, Grigoriadis N, Karapanayiotides T. Epidemiology of patent foramen ovale in general population and in stroke patients: a narrative review. *Front Neurol* 2020;**11**:281.
16. Marian AJ, Braunwald E. Hypertrophic cardiomyopathy: genetics, pathogenesis, clinical manifestations, diagnosis, and therapy. *Circ Res* 2017;**121**:749–770.
17. Graziano F, Zorzi A, Cipriani A, De Lazzari M, Bauce B, Rigato I, et al. The 2020 "Padua Criteria" for diagnosis and phenotype characterization of arrhythmogenic cardiomyopathy in clinical practice. *J Clin Med* 2022;**11**:279.
18. Corrado D, Zorzi A, Cipriani A, Bauce B, Bariani R, Brunetti G, et al. Scarring/arrhythmogenic cardiomyopathy. *Eur Heart J Suppl* 2023;**25**:C144–C154. doi: 10.1093/eurheartjsupp/suad017