

CASE REPORT

Sudden death in water: Diagnostic challenges



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Abstract: The authors report a case of sudden death in a breath-holding diver and highlight the forensic diagnostic difficulties in opining the cause of sudden death in water. The autopsy showed increased thickness of the left ventricular wall with a distinct pattern of concentric hypertrophy, evident particularly in the subaortic interventricular septum. Histological examination revealed diffuse interstitial fibrosis and associated findings of multifocal myocyte disarray especially evident in the subaortic interventricular septum. The analysis and discussion of this case made it possible to attribute sudden death to a lethal arrhythmia following myocyte disarray and hypoxia caused by breath-holding, the triggering factor of apnea. This case demonstrates the importance of a thorough forensic investigation, particularly in histological terms, in subjects found dead in water, in order to ascertain the real cause of death, which may not be always ascribable to drowning.

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1. Introduction

The scientific community has long debated on the need to distinguish between subjects dying of asphyctic syndrome from drowning and subjects found dead in water. In 1999 Modell

et al. deemed it appropriate to apply to the latter the notion of “drowning without aspiration”.¹

These implications are even more important in cases where forensic investigations provide evidence that the presence of pre-existing pathological conditions may have contributed to and/or caused death in water.

This issue is further complicated by the inherent difficulty of ascertaining that sudden death in water had occurred at such an early stage that no drowning fluid could enter the airways and generally the gastrointestinal tract. In addition, other than in deaths caused by the inhibitory nervous mechanism or asphyctic syndrome from laryngospasm, the main mechanisms underlying sudden death in water do not necessarily involve

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such a sudden arrest of cardiac function and breathing as to allow concomitant drowning to be ruled out.²

These considerations are extremely relevant given the growing number of diving incidents that require medical assessment, as competitive and/or non-competitive breath-held diving activities have become increasingly popular.

The authors report a case of sudden death in a breath-holding diver hunting underwater.

2. Case report

A 44-year-old man died during breath-held diving while hunting under water. His body was found on the seabed about 6 h after plunging. The police authority then requested a post-mortem examination.

The external examination of the body, weighing 77 kg and measuring 174 cm in length, revealed conjunctival hyperaemia and moderate leakage of reddish fluid from the nostrils. There was no evidence of traumatic injuries on the body.

The internal examination showed in particular that the heart weighed 400 g and had a significant concentric left ventricle hypertrophy particularly in the subaortic interventricular septum (asymmetrical form with significant Van Noorden index), bilateral atrial enlargement and a mild prolapse of the posterior mitral leaflet. The lungs showed some subpleural petechiae and bronchial leakage of pinkish foam.

Samples of tissues and fluids were collected for further histological and toxicological testing.

Toxicology tests were performed on the heart, blood, urine and bile. All specimens tested negative for alcohol, illegal and psychotropic drugs.

Histological tests reported hyperventilated alveoli often converging into emphysematous spaces. Seventy-five percent of these spaces were optically empty (Fig. 1), while the other 25% contained scarce red-blood cells (Fig. 2). The examination of the heart revealed multifocal myocyte disarray affecting 35% of the left ventricular sections studied associated with diffuse interstitial fibrosis mainly in the subaortic interventricular septum (Fig. 3). It also showed significant myocyte hypertrophy and a multifocal wave-like pattern (Fig. 4). The examination of the other organs reported no pathological signs.

3. Discussion

The analysis of this case and in particular of the macroscopic and histological cardiac findings demonstrated evidence of previously undiagnosed hypertrophic cardiomyopathy.

This disease is a common cause of sudden death in young adults. It can occur during both routine daily tasks and even mild physical activities.^{3,4} Genetic studies demonstrated that it is associated with dominant autosomal mutations of some genes, including MYH7, MYBPC3 and TNNT2. However, these mutations were reported in slightly less than 50% of clinically affected subjects.⁴

In this disease, the most typical anatomopathological findings are an asymmetrical increase in the thickness of the ventricular walls (particularly in the septum) with a potential shrinkage of the cavity, a common endocardial fibrosis of the mitral leaflet, atrial enlargement and mitral valve thickening. The most common histological findings are an uneven pattern of enlarged and irregular myocardial fibres (“disarray”),

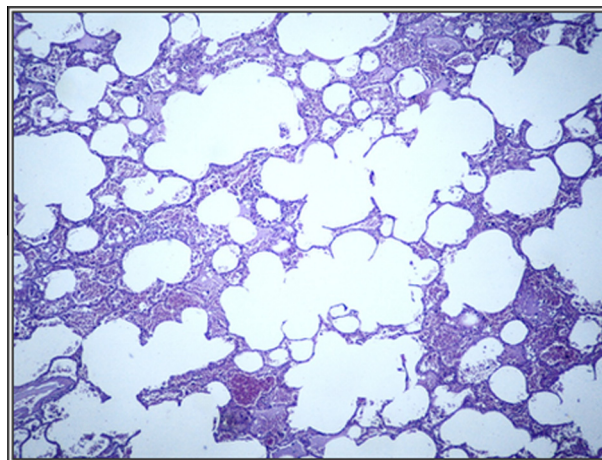


Figure 1 Hyperventilated alveoli converging into optically empty emphysematous spaces (EE 25).

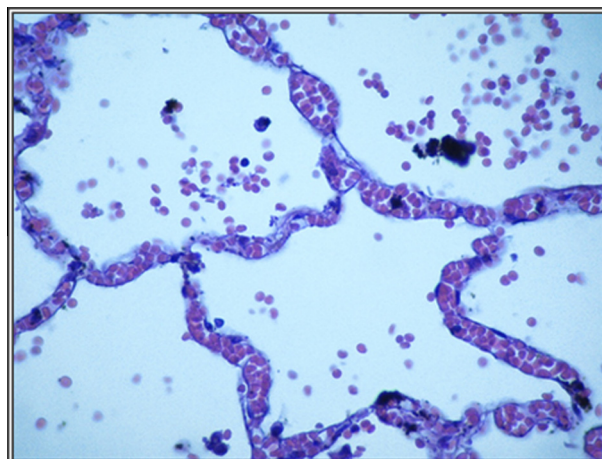


Figure 2 Alveoli sprayed with red blood cells (EE 40).

increased interstitial matrix and fibrotic hyperplasia of intramural arterioles.⁵⁻⁷

Hemodynamic and electrophysiological studies demonstrated that the clinical symptoms of hypertrophic cardiac disease are attributable to mechanisms associated with structural abnormalities of the ventricular cavity and abnormal ejection due to a defective release of myocytes. This mechanism can be promoted by anterior systolic motion of the mitral valve with subsequent congestive cardiac failure.⁸ In other cases, abnormal electrocardiographic findings can be attributed to ischemic events due to the increased myocardial mass. However this finding apparently contradicts the absence of any evidence of myocardial infarction or extensive replacement fibrosis even in severe hypertrophic cardiomyopathy.^{4,9}

Sudden death in hypertrophic cardiomyopathy is a frequent occurrence. It is generally caused by cardiac arrhythmias associated with re-entry mechanisms or excitable foci or a reduced ventricular cavity or small vessel disease.⁵

Although lethal mechanisms underlying arrhythmias associated with sudden death are well understood, the same cannot be said for the cause and timing of the initial triggering event.

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