CASE REPORT

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Endomyocardial Fibrosis in a Scuba Diving Death

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ABSTRACT: A death of an amateur SCUBA diver with undiagnosed late phase endomyocardial fibrosis is reported. This 27-year-old man found himself in difficulties during a Sub-Aqua Club outing and drowned.

Autopsy revealed features of drowning in addition to severe endomyocardial fibrosis (EMF) with collagenization of the left ventricle and interventricular septum with foci of chronic myocarditis. Other organs were essentially normal and toxicological studies were negative.

Examination of the air tank revealed a normal oxygen circulation with elevation of the carbon dioxide content above recommended limits. Sudden ventricular arrhythmia underwater due to EMF is believed to have precipitated drowning.

Stricter medical monitoring of amateur divers should prevent similar accidents in the future.

KEYWORDS: pathology and biology, scuba diving, endomyocardial fibrosis, arrhythmias, drowning, accidental death

Scuba diving is the use of self-contained underwater breathing apparatus for diving either for commercial purposes or as a sport. Common causes of death, particularly among amateur recreational divers are barotrauma, air or fat embolism with or without thrombo-occlusive phenomena, hypoxia and drowning [*1–6*]. Natural diseases, especially those of cardiac and pulmonary origin, can also cause sudden death underwater and may precipitate drowning or worsen barotrauma and/or decompression sickness [*6–9*].

Statutory regulations (embodied in an Act of Parliament of the United Kingdom entitled the Health and Safety at Work Act (1974)), closely control industrial practices, equipment and premises. As an integral part of the provisions of this Act, a body known as the Health and Safety Executive is constituted with a mandate to investigate potential dangers in working practices and to investigate accidental and other infringements of safety during employment. This body has produced strict legislation regulating commercial diving especially regarding physical fitness of the diver. The British Sub-Aqua Club is an organization of amateur divers who take part in diving as a recreational activity. It makes recommendations to its members that apply only to affiliated clubs. However, anyone can purchase diving equipment and engage in the sport without previous training

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in the science and art of diving. The potential dangers of this sporting activity are further encouraged where the amateur is known to be physically unfit or has an undiagnosed medical condition.

A case of fatal scuba diving in an amateur with undiagnosed late phase endomyocardial fibrosis is presented. The pathological findings, cause and mechanisms of death are discussed in addition to suggestions for preventing future occurrences.

Case Report

Circumstances Leading to Death

J.S.B. was a 27-year-old self-employed motor engineer; he was a member of a Sub-Aqua club and engaged in various sports. He was described as being in excellent health and had not needed to consult a doctor for several years. His smoking and drinking habits are not known.

He went diving during autumn (September) in company of other club members at a small harbor on the North Sea Coast of East Lothian (Scotland). The divers went down in pairs. J.S.B.'s initial dive with his partner was aborted after 5 minutes due to poor sea visibility and both moved to another location. During a second dive, J.S.B. descended slightly faster than his mate who later saw him sitting on a rock about 8 meters (26.7 feet) below the surface and raising his left arm. J.S.B.'s mate initially thought that nothing was wrong, but, after about 30 seconds, he realized that J.S.B. was unresponsive and air bubbles were seen to be emerging from the air bleed valves on his sleeve and mouth piece. The rescuer quickly inflated J.S.B.'s life jacket, dragged him onto a partially submerged rock and commenced cardiopulmonary resuscitation (CPR). They were joined by a co-diver and a boat took them to the shore; all these maneuvers lasted a couple of minutes. Meanwhile CPR was maintained and foam was observed around J.S.B.'s mouth.

HM Coastguard and the police were alerted and the emergency medical team at the harbor cut open the diving suit and applied a defibrillator, but all resuscitative measures proved futile and life was pronounced extinct. The dry diving suit and breathing equipment were retained by the police for further specialist examination while the body was transferred to the mortuary for an autopsy.

Autopsy Findings

Autopsy revealed no petechiae and there were no recent external or internal injuries except for those that could be attributed to resuscitation. There was no evidence of barotrauma or decompression sickness. The lungs were markedly distended, almost entirely filling the pleural space and covering the pericardial sac; the tracheobronchial tree contained copious frothy fluid. From the heavy lungs (R = 950 g, L = 930 g), copious frothy fluid readily oozed out on sectioning. Watery fluid was found within the stomach mixed with scanty semisolid food material.

The major pathological finding was in the heart, which weighed 360 g and showed marked endomyocardial fibrosis affecting predominantly the left ventricle and interventricular septum; the papillary muscles were similarly affected. On gross sectioning the fibrosis was shown extending to involve up to 25% of the myocardial thickness. The coronary arteries and great vessels were essentially normal, as were other viscera. Death was attributed to drowning with endomyocardial fibrosis as a contributory factor.

Histology showed severe pulmonary congestion and edema with focal presence of "heart failure" cells (pigmented macrophages). The heart showed beneath the pavemented endothelial lining of the endocardium the presence of a band of dense hypocellular fibrocollagenized tissue with some entrapped blood vessels (Fig. 1). The band of fibro-

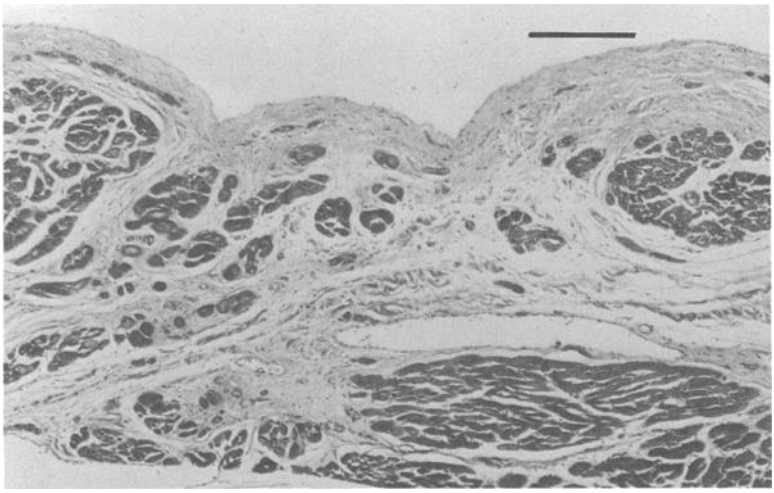

FIG. 1—*Section of the heart showing the myocardium with the endocardial surface uppermost. Below the layer of flattened endothelial cells is a dense fibrocollagenous stroma infiltrating the underlying myocardial fibers, which are entrapped and fragmented. This dense stroma is also seen to encircle some blood vessels. (Masson Trichrome stain; the bar is equivalent to 500 microns.)*

collagenous tissue extended for up to 1798 microns into the total ventricular thickness of 7210 microns. In some areas individual myocytes or groups of myofibers were submerged in the fibrous stroma (Fig. 2). This became less prominent toward the pericardial surface; fingerlike projections of fibrous tissue occasionally encircling blood vessels were seen. Foci of infiltrating lymphocytes mixed with a few eosinophils were present; small aggregates of these cellular infiltrates were observed scattered within the myocardium (Figs. 3 and 4). There were no areas of active necrotizing eosinophilic myocardial damage.

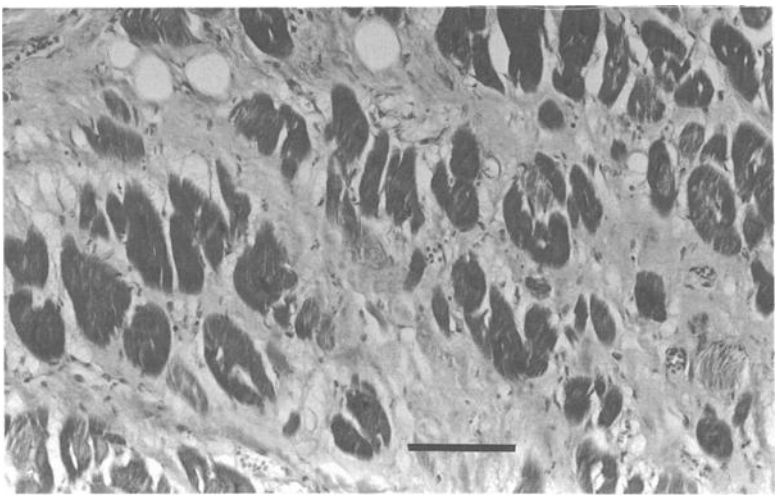

FIG. 2—*Higher magnification of the section of myocardium is showing fragmented and partly wavy cardiac muscle entrapped within a fibrocollagenous stroma. Some large adipocytes are seen in the upper left hand corner and the stroma is mildly infiltrated by lymphocytes. (Hematoxylin and Eosin stain; the bar is equivalent to 50 microns.)*

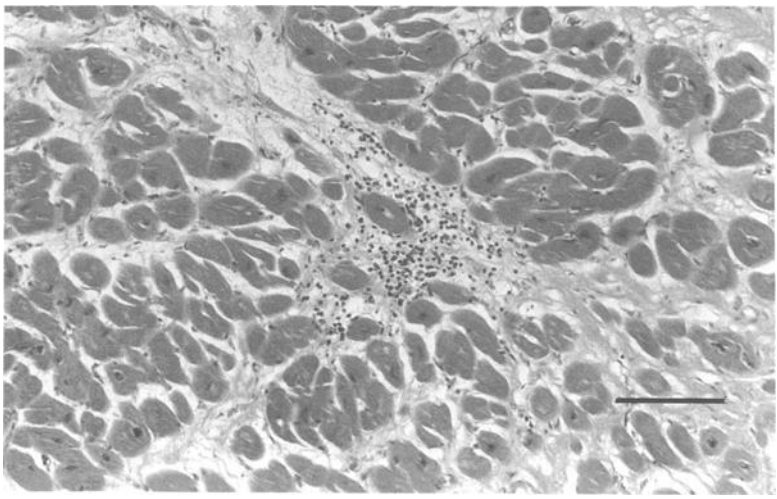

FIG. 3—*Section of myocardium showing a focus of dense infiltration by lymphocytes located within the fibrous stroma and also in close proximity to the fragmented muscle fibers. The degenerating myofibers show mild to moderate nuclear hyperchromasia with a striking shift away from the normal peripheral localization and a tendency toward an ovoid appearance rather than the normal rectangular shape. (Hematoxylin and Eosin stain; the bar is equivalent to 500 microns.)*

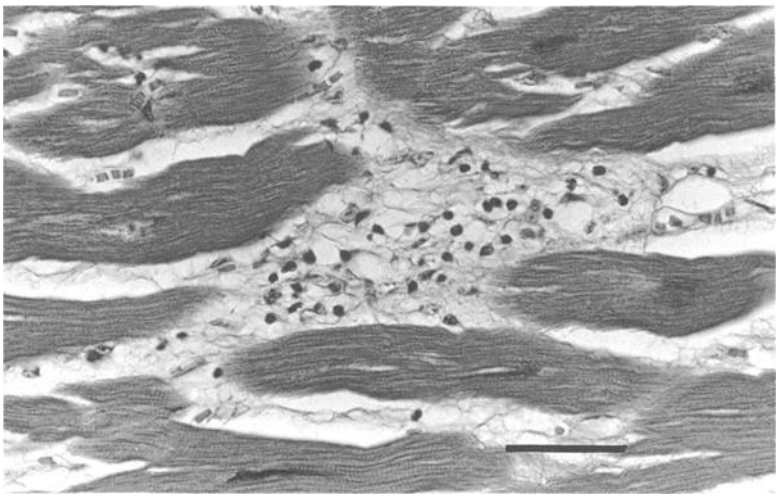

FIG. 4—*Higher magnification of another focus of chronic myocarditis with presence of occasional eosinophils and loose connective tissue stroma. The myofibers are slightly wavy but cross-striations are still very much visible. Note that the inflammatory cells are in parts intimately applied to the myofiber membrane. (Hematoxylin and Eosin stain; the bar is equivalent to 50 microns.)*

No active myocarditis or vasculitis was seen. The other major organs showed mild congestion only.

Toxicological studies showed no evidence of alcohol or drugs in blood, urine, or vitreous.

Examination of the air tank and valve revealed no defects. The tank contained 110 bar of air; oxygen = 20.9%, carbon monoxide (CO) = less than 1 part per million

(ppm), carbon dioxide (CO_2) = 850 ppm (recommended maximum is 500 ppm), water = 0.16 g/m^3, oil = 0.77 mg/m^3 and the odor was normal. No defects were present in the diving suit.

Discussion

Fatal scuba diving is occasionally observed among commercial divers [*2*] and is also more commonly reported among amateurs [*1,10*]. The account of the diver and subsequent autopsy findings confirmed drowning as the cause of death in this case. Of greater practical importance in all diving deaths are any pre-existing conditions that make the diver more susceptible to an accident; in this case cardiac disease was almost certainly the underlying cause of death.

Endomyocardial fibrosis (EMF) is a type of restrictive cardiomyopathy. The restrictive element of the condition is attributed to fibrosis; on occasion this is aggravated by superimposed mural thrombosis producing further narrowing of the ventricular inflow tract. The disease spectrum comprises eosinophilic and noneosinophilic varieties with three clinicopathological stages identifiable, namely, the acute, healing and late stages. The first is characterized by a necrotizing myocarditis with or without pronounced eosinophilic infiltration, the second by granulation tissue and the third by endomyocardial replacement with dense fibrocollagenized tissue. The terminal phase can be reached within two years of the known onset of the disease [*11*].

Several studies indicate that the myocardial damage is due to the cardiomyocytes' interaction with products of eosinophils, specifically the major basic protein (MBP) [*12–15*].

Following an appropriate stimulus, probably viral or related to antigen-antibody complexes with complement, the activated eosinophil degranulates, releasing the MBP, which is cardiotoxic and also a potent stimulator [*13,15*]. Myofiber damage and thrombosis are induced simultaneously. Studies have localized MBP on myofibers [*13–15*] especially during the active phase.

Clinical diagnosis is best made by echocardiography with ventricular angiography; endomyocardial biopsy is also helpful but requires caution in its execution so as to avoid dislodgement of mural thrombus. An antemortem diagnosis was not made in this case; the deceased did not present with any complaint of illness.

The histological findings following autopsy were consistent with those of the late phase EMF with subtle chronic myocarditis. Autopsy did reveal some evidence of chronic heart failure but no other natural disease process. Though treatment is more specifically directed at the initiating hypereosinophilic syndrome, the management is directed at treating heart failure or arrhythmias [*11,16*]. Of forensic importance, and particularly in this case, is the possibility of arrhythmia. Cardiomyopathy, as indeed myocarditis or disorders of the cardiac conduction system, may present clinically for the first time with arrhythmias [*17–20*]; this can lead to collapse and even sudden unexpected death.

Arrhythmia is known to occur in divers in the absence of underlying cardiac pathology; the mechanism is obscure, but McDonough et al. observed it to be more than 22 times more common underwater than out of it [*21*]. They attribute this to the pooling of blood centrally with resultant dilatation of the cardiac chambers, in particular the right side during descent underwater. Melamed et al. further suggest that the strain of diving, breath holding, hyperventilation and sudden changes in temperature are also arrhythmogenic in diving conditions [*21*]. EMF and chronic myocarditis posed a heightened risk of arrhythmia in this case; added to this was the known hazard of diving in autumn when the North Sea water was very cold. Why death occurred on this particular occasion and not on a previous dive cannot be explained.

Autopsy did not reveal evidence of mechanical asphyxia; such evidence is usually lacking in true drowning. It appears likely that arrhythmia during rapid descent must have precipitated collapse and consequent drowning in this case.

It is noteworthy that the CO_2 level in the air tank was higher than the recommended level. Though this did not significantly affect the relative percentage of inspired oxygen and probably played no direct part in death in this case, it has been suggested that divers tend to react idiosyncratically to elevated inspired air CO_2 concentration [2]. Increased CO_2 concentration does not necessarily stimulate ventilation in all divers and might in fact encourage heat loss and enhance the development of nitrogen narcosis [2].

Amateur divers, like professionals, should subject themselves to regular physical and medical checkups that must include exercise tests, perhaps with prolonged electrocardiographic monitoring. Those with congenital heart and lung disease, chronic obstructive airways disease, bronchial asthma, middle ear and sinus problems, diabetes, epilepsy, and those prone to spontaneous pneumothorax or arrhythmia should be closely monitored if not firmly discouraged from diving [3,6,8,9,22]. The death of J.S.B. reinforces the well-recognized view that scuba diving is a potent physiological stressor capable of activating or revealing latent disease [6]. The prudent diver will make sure that his or her health has been totally tested and not merely assumed to be adequate before venturing into the water.

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