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Case report

Lethal lung tear and coronary artery gas embolism in shallow water apnea diving exercise – PMCT, PMMRI, autopsy and histology

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ABSTRACT

A 45 year old man with 10 year history of accident and incident free recreational apnea diving finished an apnea warmup exercise still apparently well, gave a thumb up, and after two breaths collapsed. No success at subsequent resuscitation attempts. Post mortem computed tomography showed two pneumatoceles, a gas filled right coronary artery and some gas in the left cardiac ventricle and aorta. There were post mortem MRI T2 signal alterations in the left ventricle's posterior wall. Sinuses were normal. At autopsy, subpleural hemorrhages were found on both lungs with a predominance of lobe edges, indicative of mechanical strain as in mechanical breathing suppression or suffocation. Selective post mortem CT angiography of the right lung veins revealed extensive intra-alveolar leakage of contrast agent into the middle lobe, but not into the upper or lower lobes. Histology showed that fibrotic tissue and elastic fibers were not increased or decreased across lobes. The right middle lobe also contained numerous siderophages. So this is a case of recurrent pulmonary barotrauma with arterial gas embolism to the right coronary artery. This is the first case where consequences of lung pressure or barotrauma acquired at water surface by apnea as such could be documented at autopsy.

1. Introduction

This case report details aspects of the death of an apnea diver during shallow water warm up exercises during training.

Apnea diving consists in holding one's breath while diving. Training practice consists in repeated breath-hold dives over several hours, practiced at the surface of the water with 1–2 min duration. In between, there are recovery periods of 2–3 min. Also, training may contain static immersion breath holding exercises [1]. Other than that, no disease history or other findings suggested any other assumption than simple breath holding had killed the victim.

In this instance, presence of two pneumatoceles and right coronary artery gas suggested a diffuse pressure induced lung injury of the right lung's middle lobe that was confirmed by selective pulmonary vein angiography.

2. Material and methods

2.1. Case

A 45 year old man had an apparent history of a decade of accident free and incident free recreational apnea diving. No pre-existing medical conditions including illnesses or post traumatic residual states were known, in particular there was no history of asthma, diabetes, mental illness or epilepsy. However, he was reported to have experienced a metallic taste in his mouth after diving in recent times. Also, he was known to use a technique dubbed as "packing" [1].

He had conducted a static breath holding warmup exercise as part of his club training. It was conducted in a heated indoor swimming pool with chlorinated water and a depth of 1.2 m. The water temperature was 28.2 °C. Wearing a neoprene suit, the man was performing the static float exercise positioned on the surface horizontally in the water, while a colleague was watching and supervising him.

The sequence conducted was apnea for 1:30 min while being submerged in waist deep water, then the athlete would stand up to breathe and rest for 4 min, again apnea for 2:30 min, stand up and rest for 4 min, then again apnea for 4 min and again getting up, standing and breathing.

After that preceding exercise he still was apparently well. The man was reported to having given a thumb up sign after he had gotten up from his submerged floating position to standing. After taking about two breaths when not being submerged any longer but in fact standing, he collapsed. He was retrieved from the pool right away, but subsequent resuscitation attempts were not successful.

2.2. Post mortem CT and MRI scanning

A dual source CT (computed tomography) scanner (Somatom Definition Flash, Siemens, Erlangen, Germany) was used for PMCT.

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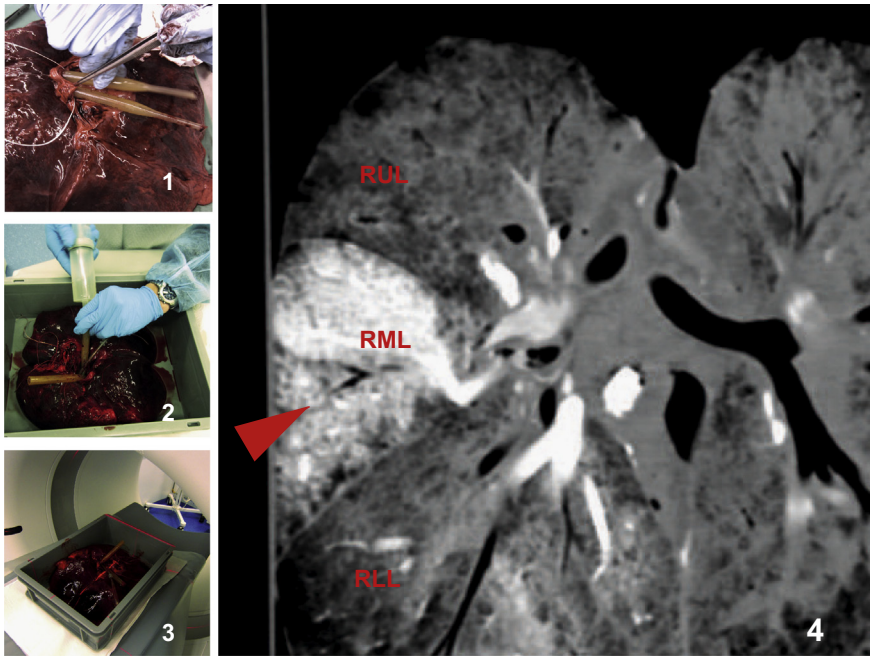

Fig. 1. Preparation for selective angiography of the lung veins contained placement of access tubes (1), filling with contrast agent (2) and subsequent CT scanning (3). Resulting lung scan (4) shows right upper lobe (RUL), right middle lobe (RML) and right lower lobe (RLL) with a predominant accumulation of contrast agent in the lung tissue of the right middle lobe, indicating extravasation due to diffuse vessel injury.

Scans were obtained at 120 kV, reference mAs 400 and 128 × 0.6 mm collimation with automated dose modulation (CARE dose4D, Siemens, Erlangen, Germany).

Slice thickness was 1 mm with an increment of 1 mm. Image reconstruction was performed with a hard and soft kernel in abdominal and lung window, respectively. Multiplanar reconstructions and volume rendering were performed using standard workstation equipment ("Somaris/7 syngo 2011A" software on a "Leonardo" workstation, Siemens, Erlangen, Germany) [2].

After autopsy removal of the lungs, the lung veins were inserted with tubes (see Fig. 1). As contrast agent, polyethylene glyocol and Gastrografin® were used with a 15:1 mixing ratio [3–6]. The solution was manually injected into lung vein mounted tubes. Then, isolated lung PMCT was performed (Fig. 1).

Post mortem MRI was obtained on a 3T MR scanner (Achieva 3.0 TX, Philips Medical Systems, Netherlands) using a 16 channel SENSE torso XL coil. The sequences relevant to this study included a T2-weighted Turbo Spin Echo sequence (T2W TSE: TR 3037ms, TE 100ms) and a T2-weighted Turbo Spin Echo sequence featuring Spectral Presaturation with Inversion Recovery (SPIR) as selective fat suppression technique (T2W TSE SPIR: TR 2013ms, TE 60ms) [7].

2.3. Microscopy

Autopsy was supplemented with standard histology of the major organs. For the lungs, Hematoxylin–Eosin, Van Gieson Elastin and Prussian Blue slides were obtained. Microscopic (microscope model 'Axio Lab.A1', Carl Zeiss AG, Oberkochen, Germany) images were captured using a digital micro scanner camera (Progres C14, Jenoptik, Jena, Germany).

3. Results

Post mortem computed tomography showed a gas filled right coronary artery (Fig. 2(6)), and two regions containing traumatic pneumatoceles in immediate vicinity of pulmonary hemorrhage in the right lung's middle lobe (Fig. 2), as well as minimal amounts of gas in the left cardiac ventricle and aorta. There was no cerebral gas embolism.

MRI showed a left ventricular posterior wall with low signal in T2W TSE (39 ± 8.4) compared to the left lateral wall (45 ± 6.9, statistically significant difference in t-test $p < 0.0001$) and a low signal in T2W TSE SPIR (60 ± 8.1) relative to the left lateral wall (69 ± 7.5, statistically significant difference in t-test $p < 0.0001$) (Fig. 3).

At autopsy, numerous small lentil shaped subpleural hemorrhages were found on both lungs with a predominance of lobe edges (Fig. 4(2) and (3)). The main coronary artery to supply blood to the left ventricular posterior wall was the right coronary artery (RCA) (see Fig. 3). The liver exhibited a nutmeg pattern on its cut surface, and a slight orange yellow discoloration.

Post mortem CT angiography of the right lung revealed extensive leakage of contrast agent into the tissue of the middle lobe, but not into the upper or lower lobes (Fig. 1).

Histology of the lungs showed extensive tearing of alveolar walls in the middle lobe of the right lung (Fig. 4, image 4). The lower lobe did not show as much tearing of alveolar walls (Fig. 4, image 5).

There were large numbers of partially densely packed siderophages, particularly in the right middle lobe (Fig. 4, image 6). Fibrous tissue and elastic fibers appeared to be neither decreased or increased across the lung slides.

There were no signs of coronary artery atherosclerosis, chronic emphysema, pulmonary artery atherosclerosis, hypertrophy of the cardiac right ventricle, pneumothorax or soft tissue emphysema. Sinuses and mastoid bone were normal. The heart did not exhibit macroscopic or microscopic pathology, in particular there were no signs of ischemic damage. Also, the posterior left ventricular wall was free of any histologic signs of peracute cell damage such as vacuoles or contraction band necroses.

The athlete was of a normal bodily build (176 cm, 76.7 kg, BMI 24.8). There were fractures of the ribs and the sternum after resuscitation had been attempted. There was a mild degree of atherosclerosis of the arteries. There was mildly increased fibrous