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.

Keywords – Virtpoxy; Post mortem Computed Tomography; Angiography; Diving; Barotrauma.
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Figure 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.

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 minutes duration. In between, there are recovery periods of 2-3 minutes. 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.
Figure 2: In the case of this apnea diver, PMCT of the right middle lobe contained two areas suggestive of a traumatic pneumatocele adjacent to hemorrhage (first area 1 axial, 2 coronal reconstruction; second area 3 axial, 4 coronal reconstruction). There was a gas filled right coronary artery (6, arrow) which is usually not gas filled (5, arrow, shown in a control case).

2 Material and methods

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 was there 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 meters. 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 minutes while being submerged in waist deep water, then the athlete would stand up to breathe and rest for 4 minutes, again apnea for 2:30 minutes, stand up and rest for 4 minutes, then again apnea for 4 minutes 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.
Figure 4: The lungs (1) showed numerous small lentil-shaped oval subpleural hemorrhages (2, arrow; 3 shows an enlarged portion of 2). Histology of the right middle lobe exhibited tears and loss of alveolar structures (4, Elastin van Gieson stain) and massive accumulation of iron-containing macrophages (6, Prussian Blue, bottom left), also when compared to right lower lobe (5, Elastin van Gieson stain and bottom right, and 7, Prussian Blue). To allow for better delineation of alveolar walls, a portion of both histology images (4A, 5A) was manually outlined to allow for easier appreciation of the extent of alveolar fragmentation in the right middle lobe (4, 4A) as compared to right lower lobe (5, 5A). Bar 1 cm.

Post mortem CT and MRI scanning

A dual source CT (computed tomography) scanner (Somatom Definition Flash, Siemens, Erlangen, Germany) was used for PMCT. Scans were obtained at 120 kV, reference mAs 400 and 128 x 0.6 mm collimation with automated dose mod-
ulation (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 glycol and Gastrografin® were used with a 15:1 mixing ratio [3, 4, 5, 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].

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 (Figure 2/6), and two regions containing traumatic pneumatoceles in immediate vicinity of pulmonary hemorrhage in the right lung's middle lobe (Figure 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 ($69 \pm 7.5$, statistically significant difference in t-test $p<0.0001$) (Figure 3).

At autopsy, numerous small lentil shaped subpleural hemorrhages were found on both lungs with a predominance of lobe edges (Figure 4/2 and 4/3). 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 (Figure 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 (Figure 4, image 5).

There were large numbers of partially densely packed siderophages, particularly in the right middle lobe (Figure 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 tissue in the liver combined with a mild to moderate increase in fat vacuole deposits, as well as distended central and pericentral sinusoids, suggestive of chronic congestion.

4 Discussion

In this 45 year old man, barotrauma of the lungs induced by apnea diving, possibly assisted with buccal pumping or "lung packing", must have caused massive
transpleural pressures as also indicated by extensive hemorrhages on the visceral pleura, tearing across the right middle lobe of the lung, diffuse injury to the vessels with air embolism to the heart and the right coronary artery with what appear to be MRI signs of possible peracute hypoxic injury to the left posterior ventricular wall of the heart [8, 9] even in absence of concise histological evidence [10]. The lungs, particularly the left middle lobe, contained siderophages stained blue as sign of earlier injury there, and the liver showed signs of chronic congestion in absence of signs of structural heart disease [11]. There were no other concurrent diagnoses for cause of death, in particular were there no signs of drowning.

While circumstantial correlation appears to have lead to the assumption that post mortem MRI has the capacity to show myocardial regions whose the tissue is peracutely (but not acutely) damaged by obtaining a relatively low T2 signal [10], the finding was so far not reliably reproduced experimentally [8]. Thus, the question may have to therefore be taken into consideration, whether postmortem MRI is a possibly more sensitive method inasmuch as acute myocardial injury is concerned rather than a histological search for contraction band necroses.

The context of apnea diving and lung tears leading to gas embolism is unusual as seen rarely by forensic pathologists, but very plausible. Resuscitation was very unlikely to cause these findings [12]: no intraosseous access lines had been installed [13], no gas was found in the liver or other abdominal organs. There was no indication that resuscitation measures had caused the lung tears or relevant coronary air embolism. Also, no such findings were present in a review of resuscitation cases; secondly, pulmonary injury leaking air into coronary arteries causing myocardial damage was reported before [14].

When lung tissue tears up due to pressure, the pulmonary alveolar lining and vessel linings can get damaged. Then, these tears communicate openly with alveolar gas or air. Rupture causing hemorrhage into alveolar spaces can entail gas leaking into pulmonary veins, particularly when intraalveolar gas pressure is higher than pulmonary vein pressure [15].

To the best of our knowledge, this is the first case where specific consequences of excessive lung pressure or barotrauma acquired at water surface by apnea or breath holding were documented in a medicolegal autopsy. Diagnosis was made based on both conventional PMCT and selective pulmonary angiography inte-
grated with conventional autopsy and histology, all of which provided relevant findings. Properly sequencing post mortem tests still is a matter of individual judgment [16]. Sequencing can be a relevant issue as post mortem morphology recently has seen a massive boost in evidence collection through adding post mortem computed tomography (PMCT), magnetic resonance imaging, angiographic methods, post mortem ventilation, DTI studies, 3D surface scanning and more [17]. In our institute, we discuss step-by-step procedures for each case individually, and we also adapt further examinations based on previous results at each step. Here, a full body CT scan was captured as one of the first things after the body was admitted to our institute. Before any further tests were done, and before any further activities were undertaken, we had a thorough and close look at that CT scan [18]. On that basis, pressure caused lung tear injury was assumed immediately after PMCT imaging, and after a team discussion, selective angiography of the removed lungs was chosen as method. Given that many routine autopsies still are performed without adequate volume scanning to visualize gas or blood vessel injury in case specific ways, it is understandable that diagnoses such as in this instance are rare in literature.

Among pulmonary barotrauma in divers, barotrauma tends to be worse when it recurs. Then, it is typically associated with arterial gas embolism [19]. Regular extreme apnea in competitive breath-hold divers has been shown to correlate with functional cardiopulmonary abnormalities toward the onset of pulmonary hypertension [20]. In this instance, no evidence of pulmonary artery atherosclerosis or right ventricular hypertrophy was found but the liver exhibited signs of chronic congestion.

Pathophysiology of lethal barotrauma to the lungs at shallow water apnea diving appears to be somewhat obscure, however, because it cannot possibly be attributed to decompression [21, 22]. The only physiologically relevant activity that occurs here is related to techniques of breath holding, but there are no relevant external pressures or gas tank equipment to consider. Warm up exercises in apnea diving that contain breath holding exercises in shallow water, so far, are viewed as relatively harmless, particularly if performed by experienced swimmers or divers.

An examination of the history and of the clinical status of divers that do suffer pulmonary barotrauma from diving at shallow depths – particularly in
comparison to those that do not – will not yield an easily identifiable factor [19]. The only tangible evidence to correlate with the occurrence of pulmonary barotrauma appears to be a reduced pulmonary distensibility [23, 24]. Despite an absence of clinical diagnoses of a pulmonary disease, cigarette smokers tend to be more frequent among the divers suffering barotrauma at shallow depths. There is no evidence that previous episodes of pulmonary barotrauma make lungs stiffer, also based on analysis of repeated incidents in asthma patients [23]. In this instance, dense accumulations of siderophages did not go along with an increase in fibrous tissue or reduction in elastic fibers as judged, microscopically, per field of view. With increased age, total lung capacity and elastic recoil of the lungs decrease [25]. That can make it considerably harder for the more mature individual to be just as successful in breath holding diving as younger divers.

Age dependent restriction of total lung capacity and elasticity may not be a fact of life that may be acceptable to all divers. Buccal pumping (or ‘lung packing’) is a technique that is used by ‘free’ divers to increase total lung capacity. Buccal pumping was declared to not constitute a major risk for pulmonary barotrauma, despite an increase in intrapleural pressure from -22,7 (normal) to +56,7 cmH₂O [1]. Such a large increase in transpleural pressure would explain the rather extensive subpleural hemorrhages in this case.

From that, it might be deduced that if anything, one should – in addition to respecting existing guidelines – also be careful with breath holding exercises as a smoker or ex-smoker, as a person with evidence of lung cysts or increased scar or fibrous tissue, combined with an age over maybe 35 to 40 years. Depending on one’s condition, buccal pumping might not be completely safe, even though evidence that buccal pumping was employed in this instance is only circumstantial. And yet, many instances of pulmonary barotrauma and arterial gas embolism are survived. Body position, bubble buoyancy and gravity are not likely to dominate gas bubble distribution over the forces of arterial flow[26, 27].

References


