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Case Report

Pulmonary artery rupture during Swan-Ganz catheterisation: A case report

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ABSTRACT

Catheter-induced pulmonary artery rupture is an infrequent complication that may occur during invasive cardiopulmonary monitoring. Fatal cases are uncommon and result from hemoptysis and flooding of the opposite lung with resulting hypoxia. Alpha-1-antitrypsin deficiency is a rare genetic disorder characterised by low serum levels of alpha-1-antitrypsin, critical in maintaining connective tissue integrity. Besides pulmonary emphysema, recent observations suggest that alpha-1-antitrypsin deficiency may also be involved in vascular wall weakening, thereby predisposing arteries to dissection and aneurysm formation. In this article, we describe an autopsy case of pulmonary artery iatrogenic rupture due to insertion of a Swan-Ganz catheter in an 82-year-old woman suffering from pulmonary hypertension and alpha-1-antitrypsin deficiency. The exact source of bleeding could not be precisely identified during autopsy due to the extent of tissue hemorrhage, though postmortem angiography revealed a contrast medium extravasation from a branch of the left pulmonary lower lobar artery. The case herein emphasises the importance of postmortem angiography in facilitating the detection of vascular injuries, the importance of familiarity with intensive care techniques and procedures on behalf of forensic pathologists as well as in-depth knowledge of all possible contributing conditions and predisposing disorders in the pathogenesis of death.

1. Introduction

Pulmonary artery rupture is an uncommon, though often lethal, complication of catheterisation during invasive cardiopulmonary monitoring. Comprehensive review of the literature has reported incidence rates averaging 0.01–0.47%, with a mortality of 50% that can reach as high as 75% in anticoagulated patients [1]. Vessel rupture usually occurs on the right side and is most common in elderly women undergoing cardiopulmonary bypass. Other predisposing factors are anticoagulation therapy and pulmonary artery hypertension. In most cases, the mechanism of injury is arterial wall damage caused by the advancing catheter tip or by normal or eccentric balloon inflation, especially when the catheter is wedged [2]. If death occurs, it is usually secondary to asphyxia rather than hypovolemic shock. Indeed, most fatal cases result from consequences of hemoptysis and opposite lung flooding with resulting hypoxia and asphyxiation [1,3].

Alpha-1-antitrypsin (AAT) deficiency is a rare genetic disorder characterised by hepatitis in neonates, childhood and adulthood and pulmonary emphysema with or without hepatitis in adulthood [4]. The disease is characterised by low serum levels of AAT, the main protease inhibitor in human serum, with clinical manifestations that may vary widely among patients, ranging from the absence of symptoms to fatal liver or lung disease [5]. Due to its anti-protease activity, AAT is critical in maintaining connective tissue integrity. Recent observations have suggested that AAT deficiency (AATD) may be responsible for nonarteriosclerotic vessel disease. AATD might predispose the arterial walls to dissection or aneurysm formation because the extracellular matrix has been inherently compromised. To date, AATD has been linked to the dissection of several arteries including the aorta, internal carotid, cervical, common iliac and coronary arteries as well as aneurysms of the splenic and mesenteric arteries [6].

In this article, we describe a case of fatal iatrogenic rupture of a branch of the left pulmonary lower lobar artery due to insertion of a balloon-tipped, flow-directed (Swan-Ganz) catheter in an 82-year-old woman suffering from pulmonary hypertension and AATD with severe pulmonary emphysema. The patient developed massive hemoptysis and died intraoperatively. Postmortem angiography allowed the source of bleeding to be detected while

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histology showed the presence of periodic acid-Schiff (PAS) positive granules in the hepatocytes, thus confirming the AATD diagnosis.

2. Case report

An 82-year-old woman with past medical history significant for pulmonary hypertension and AATD with severe pulmonary emphysema was admitted to the hospital due to progressive, worsening dyspnea at rest and shortness of breath, associated with fatigue. The patient also suffered from hypercholesterolemia, arterial hypertension and refractory atrial fibrillation, which had been treated by ablation of focal triggers (ablation of the atrioventricular node and pacemaker implantation).

Increased blood pressure was noted from the physical examination on admission. The chest roentgenogram was unremarkable, though the 2-dimensional echocardiogram showed severe tricuspid regurgitation without regional wall motion abnormalities. The right cardiac chambers were dilated with mild hypertrophy of the right ventricle.

The patient was transferred to the cardiac intensive care unit for hemodynamic monitoring. The following day, with no improvement in the patient's dyspnea, she was scheduled for pulmonary catheterisation using a right femoral approach. Catheterisation was performed under combined radioscopy and echocardiographic guidance with a Swan-Ganz catheter. The catheter was advanced and the balloon wedged into the pulmonary artery. Pulmonary capillary wedge pressure was found high after Swan-Ganz balloon inflation in a branch of the left pulmonary artery. Suddenly after Swan-Ganz deflation and retrieval, the catheter tip lunged forward, resulting in laceration of the artery. The patient started coughing and soon afterward experienced massive hemoptysis with loss of at least 200 ml of fresh blood.

Urgent anesthesiologist assistance was required. The patient was sedated and an endotracheal tube was immediately placed for airway protection. The culprit vessel was not identified and the massive bleeding could not properly be controlled. The patient died minutes thereafter despite resuscitation attempts.

A medico-legal autopsy was requested by the public prosecutor since the death occurred in the hospital and a correlation between pulmonary catheterisation, hemorrhage and fatal outcome could not be formally excluded.

A multi-phase postmortem computed tomography angiography was performed prior to autopsy and revealed an evident, intrapulmonary extravasation of contrast medium from a branch of the left pulmonary lower lobar artery. The precise localisation of the source of bleeding was therefore identified Fig. 1.

External examination was unremarkable except for fresh injection marks due to medical intervention on the upper and lower limbs and cardio-pulmonary resuscitation marks on the chest.

Internal examination revealed rib fractures and intercostal space hemorrhagic infiltrations consistent with cardio-pulmonary resuscitation marks. On autopsy, the lungs were obtained en bloc with mainstem bronchi and trachea. The pericardial sac showed no adhesions and contained 50 ml of clear fluid. The heart weighed 400 g. Heart examination revealed right cardiac chamber dilatation and mild right ventricle hypertrophy. The coronary arteries had a normal anatomic course and revealed mild atheromatous disease without significant stenosis. Examination of the pleural cavities showed bilateral hemothorax (approximately 100 ml blood in each cavity). Large amounts of blood were found in the upper and lower respiratory tracts. Gross examination of the lungs revealed diffuse bilateral hemorrhages. On cut section, both lungs (right 930 g, left 900 g) showed diffuse emphysema and hemorrhagic infiltrations, especially in the left inferior lobe. The exact source of bleeding could not be precisely identified due to the extent of tissue hemorrhage. Pulmonary embolism was not observed. The spleen, liver, kidney and brain did not show any significant, macroscopic changes.

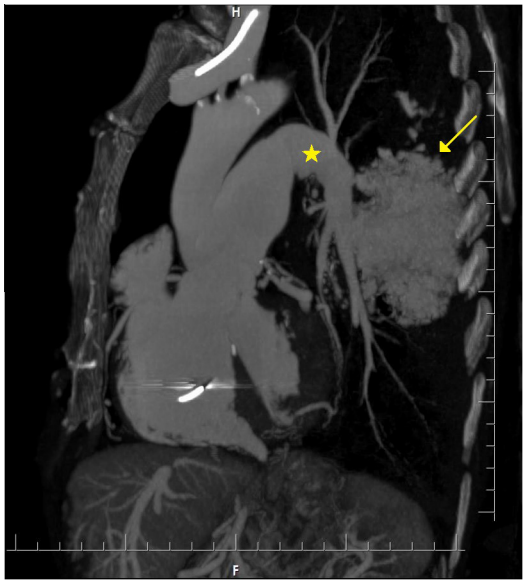

Fig. 1. Visualisation of the hemorrhage originating from a branch of the left pulmonary lower lobar artery after the veinous phase of the multi-phase postmortem computed tomography angiography. Left sagital-oblique maximum intensity projection reconstruction showing the intra-pulmonary extravasation of contrast medium from a branch of the left pulmonary lower lobar artery (arrow) and the left pulmonary artery (star).

Sections of most of the organs were examined microscopically. Neuropathology was unremarkable. The heart revealed right ventricular hypertrophy with no evidence of subendocardial hemorrhage or acute myocardial infarction. The lungs showed generalised alveolar septal wall destruction and diffuse hemorrhagic infiltrations. Pulmonary artery examination (hematoxiline eosine stain) failed to reveal structural abnormalities. Periportal round to ovale eosinophilic globules, characteristic of AATD, were identified in the liver with PAS stain Fig. 2.

Femoral blood, vitreous humor and cerebrospinal fluid as well as gastric content, hair and tissue samples were recovered for toxicology and biochemistry. Toxicological analyses were performed on blood. These analyses included ethanol determination as well as screening for common drugs and illegal substances by gas chromatography–mass spectrometry (GC–MS), high-performance liquid chromatography with diode-array detection (HPLC-DAD) and headspace-gas chromatography flame ionisation detection (HS-GC-FID). The results of the toxicological analysis were negative for ethanol and all screened drug substances. Postmortem biochemical investigation results were not contributory.

Based on the clinical information as well as postmortem investigation findings, the cause of death was determined to be hemorrhagic shock and massive aspiration of blood into the airways due to the rupture of a branch of the left pulmonary lower lobar artery following pulmonary catheterisation. The death was classified as a therapeutic accident resulting from pulmonary artery laceration during the Swan-Ganz catheterisation.

Although Swan-Ganz catheter manipulation was estimated to be the main factor leading to the vascular rupture, AATD was postulated to have played a role in the pathogenesis of death by possibly compromising the extracellular matrix and weakening the arterial wall, thereby predisposing or facilitating the rupture.