



Massive gas embolism in a child

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Abstract

A 16-month-old girl who was hospitalized with pneumonia and treated with antibiotics died after the nurse erroneously connected her intravenous left forearm catheter to the oxygen supply. Autopsy revealed an impressive gas embolism in the left subclavian and brachiocephalic veins, reduced crepitus and enlarged lung volume, and congestion of the meningeal vessels with some areas showing small air bubbles. Dilation of the right atrium and the right ventricle with efflux under pressure of large amounts of air bubbles were observed. The coronary arteries and veins were filled with air bubbles intercalated with segments containing blood. After exclusion of putrefactive artifacts as the source of such a large amount of gas in the body death was considered to be due to a massive air embolism. While embolisms are well recognized in adults, these cases are only infrequently encountered in forensic practice in younger decedents.

Keywords Massive gas embolism · Child · Neglect · Iatrogenic · Forensic autopsy

Case report

A 16-month-old girl hospitalized due to bilateral lobar pneumonia and intravenously treated with antibiotics via a peripheral line into her left forearm died following gas embolism caused when a nurse erroneously connected the serum catheter to the hospital oxygen system that supplied the nebulization mask. Prior to the event, the child was well, had been breastfed, and was ventilated. At the time of administering the antibiotherapy, the nurse noticed that the venous access was obstructed. Whilst attempting to clear the venous access,

the child became restless, and in the process of restraining the child whilst the effort continued, her nebulization mask became detached from the oxygen equipment and fell to the floor, leaving the oxygen efflux tube lying on the bed. In an attempt to reconnect the venous antibiotic catheter the nurse mistakenly picked up the oxygen tube and connected it directly to the venous access. The child became more agitated, with coughing, nausea, vomiting, cyanosis, convulsions and finally fainting. During this time, the error in the equipment was rectified; the oxygen line was removed from the venous access and the serum equipment was repositioned. The doctor was called and verified that the patient was in cardiorespiratory arrest but attempts to resuscitate her were not successful. The doctor was not informed that someone had introduced oxygen into the child's vascular system. The death certificate was completed with the diagnosis of myocarditis, pneumonia, or respiratory insufficiency. The Public Prosecutor's Office ordered an autopsy.

An autopsy was performed 12 h after death. External examination revealed subcutaneous emphysema with audible crepitus on the entire body surface. Autopsy also revealed venous congestion of the meningeal vessels with some areas showing small air bubbles inside the vessels between the cerebral convolutions (Fig. 1a). Large amounts of air bubbles under pressure were escaping through the opening of the left subclavian vein (Fig. 1b). An enlarged left lung with reduced crepitus was also recorded. The right lung exhibited increased volume, crossing the midline and with crepitus diminished in

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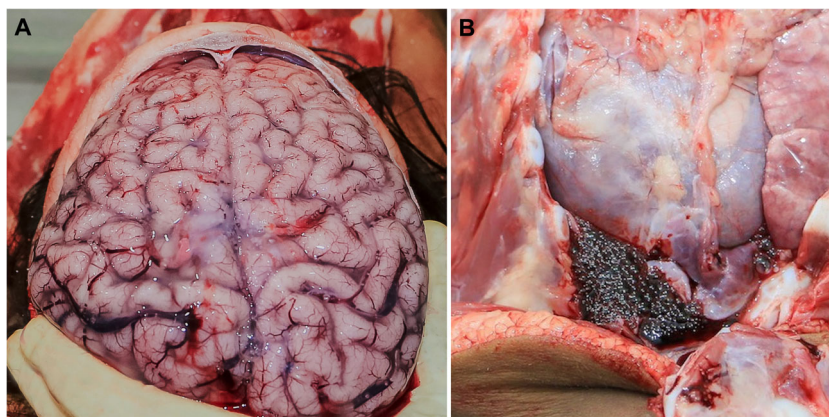
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Fig. 1 **a** Venous congestion of the meningeal vessels with some areas showing small air bubbles inside the vessels between the cerebral convolutions; **b** Air bubbles exit under pressure through the opening of the left subclavian vein



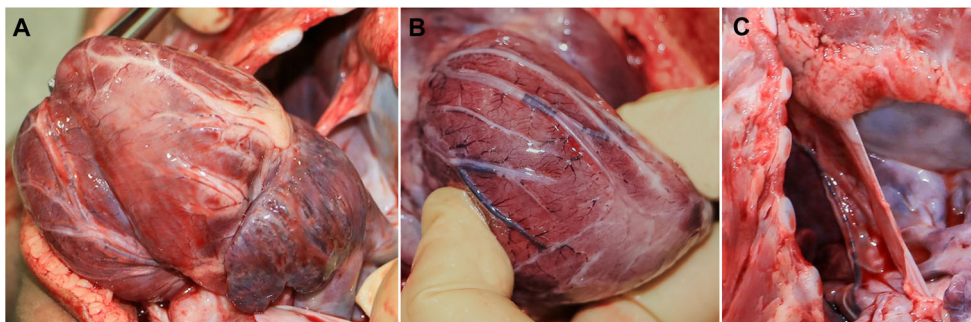
its apex and base and increased in its middle lobe. The heart showed dilation in its right atrium and ventricle (Fig. 2a) with large amounts of air bubbles flowing out of the scalpel incision in the right atrium. There were also air-filled coronary arteries and veins, and some segments with blood and air bubbles (Fig. 2b). Bubbles within the pericardial vessels (Fig. 2c) and an increased size and congestion of the spleen were also present. Signs of advanced putrefaction were not present, excluding putrefaction artifacts as the source of the massive amount of gas present in the body. Forensic autopsy indicated massive air embolism as the cause of death.

Discussion

Gas embolism, which is the entry of gas into the vascular system (i.e. veins, arteries or both) is a potentially life-threatening event and can result in serious morbidity and mortality [1]. It has mainly an iatrogenic cause as a complication of invasive diagnostic and therapeutic procedures, including direct air aspiration into the venous system through a disconnected peripheral intravenous catheter, direct air injection, surgical procedures, therapeutic abortion, caesarian delivery and positive pressure ventilation, bronchoscopy with fine needle aspiration, but also less commonly following barotraumas while scuba diving [2–7]. Venous gas embolism (VGE) occurs when gas enters the venous circulation, usually from

iatrogenic causes in the operating room or other invasive medical procedures. VGE may lead to cardiovascular collapse or to paradoxical arterial gas embolism (AGE) when gas that has entered the venous circulation migrates to the systemic arterial circulation. AGE is the entry of gas into the pulmonary veins or arterial circulation and most commonly results from pulmonary barotrauma (PBT) while scuba diving [8]. However, gas can also be injected directly into the arterial circulation during radiologic procedures and cardiac bypass surgery, intrapulmonary shunts, a patent foramen ovale, or any other right-to-left shunt, retrograde flow into cerebral veins through the superior vena cava or vertebral veins, leading to AGE [9]. The entry of gas into venous or arterial vessels requires a source of gas (usually the atmosphere or insufflation during arthroscopy or laparoscopy), a breach in the vascular wall, and a pressure gradient that favors the entry of gas into the vessel. Although the clinical consequences of VGE and AGE are different, therapeutic interventions may be similar [3]. The exact incidence of VGE is unknown because many cases of VGE are subclinical and unreported. The pathophysiology of VGE is related to the volume of gas that enters the vasculature and the accumulation rate of gas. Indeed, if a large quantity of air enters into the venous system, it reaches the right ventricle and finally the pulmonary circulation. As the pulmonary arterial pressure rises, pressure increases on the right ventricle, decreases the right ventricle outflow and the pulmonary venous return, which results in a diminished cardiac output and a cardiovascular collapse [1, 10].

Fig. 2 **a** Dilatation of the right atrium and ventricle; **b** Air-filled coronary arteries and veins, and some segments with blood fluid and air bubbles. **c** Air-filled pericardial vessels



We report an unusual and impressive case of gas embolism due to the introduction of oxygen into the peripheral venous system under high pressure, caused by a neglectful and reckless nurse during drug administration to a child in a pediatric hospital. Even though it happened accidentally, the case was considered as a qualified homicide due to the imprudence of the nurse. The doctor was free from judicial persecution but entered in a process for violation of *leges artis* by the Medical Association for having filled in the Death Certificate as natural causes. According to the literature, the presence of air in the vascular system is potentially fatal when it is abruptly injected directly into the venous system and reaches a volume around 3–5 mL/kg [1]. The volume required to cause cardiovascular collapse is quoted as being 10–480 mL in a healthy adult [11], although it depends on the velocity of injection of the air in the circulatory system and on the extension of the embolism (e.g. if only pulmonary or cerebral too). Other authors have reported that 300–500 mL of gas injected at a rate of 100 mL/s is fatal for humans [1, 12, 13]. Other reports suggest that a very small amount is required with death resulting from an injection into the cerebral circulation of as little as 2 mL, and into the pulmonary vein of as little as 0.5 mL [14, 15]. Taking into account these values and the hospital protocol that routinely uses a volume of 4–6 L/min of continuous oxygen flow for nebulization, and that the time elapsed from the recognition of the error and its reversal is at least 10 s, approximately 400–600 mL of oxygen was rapidly administered to the child's peripheral venous system under pressure. Since a volume of 60–70 mL is enough to cause cardiovascular collapse and death in a child weighing 13 kg, the amount of gas that was introduced is almost 10 times higher than the fatal amount.

In conclusion, gas embolism should be suspected in any patient with venous access who suddenly develops unexplained and sudden hypoxemia or hemodynamic cardiac and respiratory collapse, manifested by dyspnea, anxiety, dizziness, nausea, and imminent death, followed by neurological signs such as confusion, nausea, and loss of consciousness [16], signs and symptoms that were witnessed and reported by the child's mother. The temporal relation between the injection of air and the sudden development of symptoms must lead the physician to a presumptive diagnosis of possible gas embolism. Moreover, gas accumulated in the body can be precisely located and quantified using postmortem imaging, and an assessment of any postmortem changes can be made by a forensic radiologist to differentiate between an exogenous or an endogenous (i.e. putrefactive) origin of the gas [7, 17, 18]. Finally, aiming to reduce the interference of putrefactive artifacts of gas-forming bacteria, autopsy should be performed soon after death.

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Compliance with ethical standards

Conflict of interests The authors declare that they have no conflict of interest. No writing assistance was used in the production of this manuscript.

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