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Case report

Fatal amniotic fluid embolism with typical pathohistological, histochemical and clinical features

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ABSTRACT

Despite the decrease in maternal mortality rate, amniotic fluid embolism (AFE) is still one of the most feared complications of pregnancy due to the high rate of mortality in Japan. The authors present a fatal case of a healthy 39-year-old woman who died during delivery after a normal 40-week second pregnancy. Shortly after the arrival at hospital, an abrupt drop of foetal heart rate was observed, followed by deterioration of consciousness and cardiac arrest of the patient. Prompt cardiopulmonary resuscitation (CPR) was performed but the patient died about an hour and a half after her arrival at hospital. Forensic autopsy confirmed the pathohistological diagnosis of amniotic fluid embolism supported by histochemical analysis results and excluded other possible causes of death. This paper stresses the fundamental importance of autopsy in an unexpected maternal death in conjunction with the significance of data accumulation on maternal death.

1. Introduction

Despite earlier recognition and aggressive treatment, morbidity and mortality rates of amniotic fluid embolism (AFE) remain high. An estimated 5–15% of all maternal deaths in Western countries are due to AFE [1]. The reported maternal mortality rates for AFE range from 37% to over 80%, with one report stating that 25–50% of deaths occur within the first hour of diagnosis [2–4]. Diagnosis of AFE has historically been based on autopsy, revealing amniotic components in maternal pulmonary vasculature. Some recent studies introduced zinc coproporphyrin I (ZnCP-I) and sialyl-Tn (STN), both characteristic components in meconium, as less invasive, diagnostic markers for AFE [5,6]. Presented in this paper is a fatal AFE case in which the typical features were observed in pathohistological, histochemical and clinical findings.

2. Case history

The patient was a 39-year-old multiparous woman without any medical history or eventful course of pregnancy at 40-week gestation. She had no known allergies and was not taking any medication. Shortly after the membrane rupture at home, she was brought to the labour and delivery unit of the hospital by her husband's car, in active labour. Her cervix was dilated 4 cm and contraction occurred every 1.5 min on admission. Although the foetal heart rate (FHR) was 140–160 bpm, meconium staining was already observed at this stage. About 12 min later the FHR dropped to less than 120 bpm, and it became undetectable by a few minutes after the initial drop. An intravenous infusion of tocolytic agent was started to weaken labour pains, but the patient deteriorated, becoming unconscious, displaying the signs of cardiovascular collapse. Cardiopulmonary resuscitation (CPR) was begun immediately but the patient was pronounced dead about an hour and a half after her arrival at hospital. Because of the abrupt onset of symptoms and intensive CPR, an emergency caesarian section could not be carried out to deliver the foetus. The estimated blood loss was very little.

3. Autopsy findings

Forensic autopsy was performed approximately 18 h postmortem. The decedent was 162 cm in height and weighed 63 kg. No significant findings at external examination of body were present. All organs were congested. The weights of lungs were 442 g left and 535 g right, both strongly oedematous. The heart was of normal size, weighing 336 g, and the coronary arteries were free of atherosclerosis. No clots were found in the heart blood and a number of petechiae were seen in bilateral palpebral conjunctiva and epiglottis. A male foetus without remarkable anomaly was in

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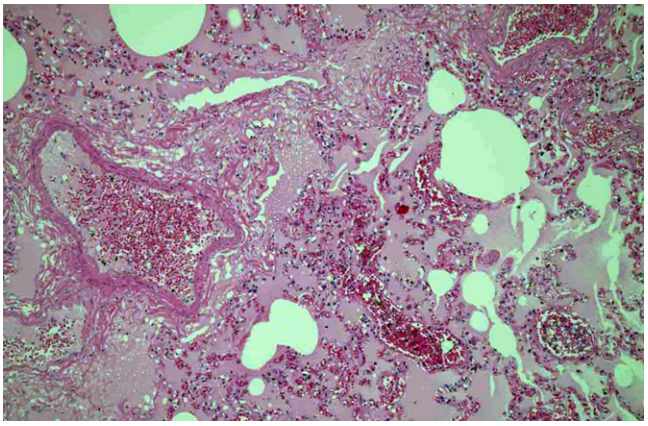

Fig. 1. Pathohistological section of the lung: blood congestion in capillaries (haematoxylin-and-eosin stain, original magnification 100×).

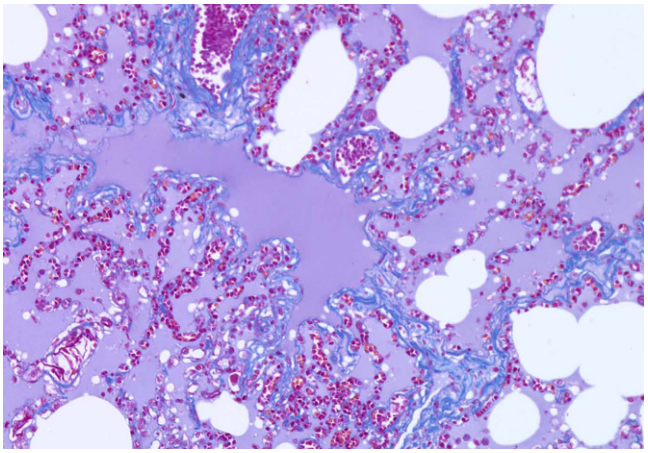

Fig. 3. Pathohistological section of the lung: blood congestion in capillaries (Azan stain, original magnification 100×).

the uterus, weighing 3800 g. The placenta was intact, and the umbilical cord was also normal. A small tear was observed on the posterior wall of cervix. Biological sample from heart blood was reserved but no urine could be obtained for toxicological investigation. Microscopic examination revealed extensive blood congestion in the pulmonary vasculature and microthromboemboli in the uterine microvasculature by haematoxylin-and-eosin and azan staining (Figs. 1–4). Furthermore, amniotic components were also detected inside the pulmonary vessels by alcian blue and ZnCP-I staining (Figs. 5 and 6). Immunohistochemical staining for C5a receptor (C5aR) was positive in stromal cells around the pulmonary capillaries and inflammatory cells in alveolus (Fig. 7). No remarkable pathological changes were observed in placenta. Alcohol and drugs were not detected by routine toxicological analysis. Concentrations of ZnCP-I and STN were 72.5 pmol/mL (normal: <1.6 pmol/mL) and 2630 U/mL (normal: <45 U/mL), respectively. These findings confirmed AFE as cause of death of the patient.

4. Discussion

The Japanese maternal mortality rate (number of maternal deaths per 100,000 live births) has been declining since the 1970s, being stable around 5 for the past decade [7]. Nevertheless, maternal death occurs on occasion, and the most frequently reported causes today include AFE, complications of pregnancy induced hypertension (PIH), pulmonary embolism, haemorrhage and infectious diseases [8–11]. The AFE syndrome was first described by Meyer in 1926 [12] and numerous case reports have been published from various countries to date. Despite its long, worldwide recognition, it is still challenging to save AFE patients due to the fulminant onset of symptoms and rapid clinical course [13].

AFE is generally characterized by a rapidly progressive clinical course with dyspnoea, hypoxaemia, hypotension and foetal bradycardia with subsequent and acute cardiorespiratory collapse, disseminated intravascular coagulopathy (DIC), neurological compromise, maternal and foetal death [14,15]. Since not all of these symptoms are evident on presentation, the differential diagnosis for AFE is broad and includes anaphylactic or haemorrhagic shock, eclampsia, cerebrovascular diseases and pulmonary embolism [16,17]. There are no universal diagnostic criteria to confirm AFE but some countries have their own for the national registry, including the United States of America, the United Kingdom and Japan [2,3,18]. A reliable diagnosis can be made only upon pathohistological examination, by the proof of amniotic fluid elements such as epithelial squamous cells, lanugo hair, and fat from vernix or infantile mucin in the pulmonary vascular bed of the mother [19]. These components of amniotic fluid could be identified in routine haematoxylin-and-eosin-stained sections, but the use of immunohistochemistry permits a more reliable assessment of the dimension of AFE. Special stains that have been used to demonstrate amniotic fluid include alcian blue stain to

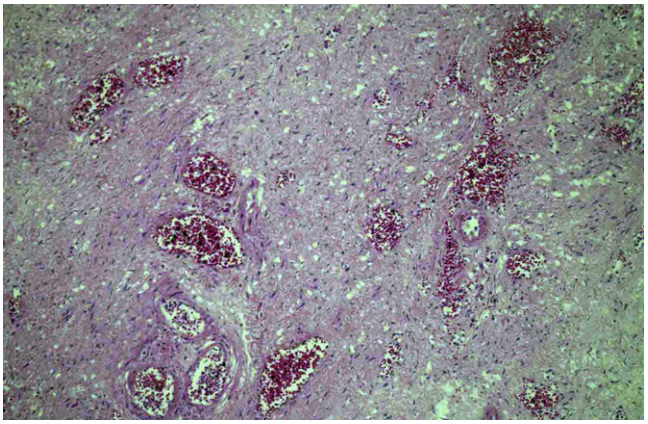

Fig. 2. Pathohistological section of the uterus: emboli in capillaries (haematoxylin-and-eosin stain, original magnification 100×).

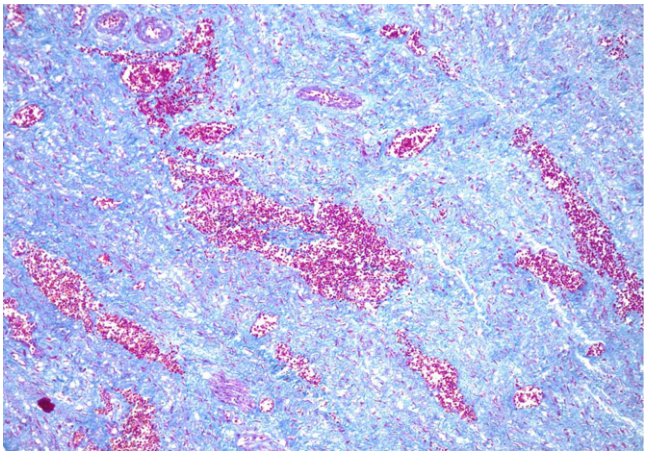

Fig. 4. Pathohistological section of the uterus: emboli in capillaries (Azan stain, original magnification 100×).