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Case Report

Death mechanism of amniotic fluid embolism was inclined to anaphylactic shock: a case report

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Abstract: A 38-year-old woman (Gestation 2, Production 1) was admitted to hospital for awaiting delivery for her conceiving for 41^{+1} weeks and experiencing abdominal pain for 1 day. Typical clinical manifestations of allergic shock performed twice on her respectively during the process of labour and after complete curettage of uterine cavity, and she died soon after the second event. So it was believed that the cause of maternal death was surely amniotic fluid embolism (AFE) according to the findings of forensic pathology autopsy and histopathological examination combined with the details of this case, but her clinical manifestations before death suggested that the death mechanism of amniotic fluid embolism was more likely to be allergic shock rather than pulmonary embolism.

Keywords: Anaphylactic shock, amniotic fluid embolism (AFE), autopsy, forensic pathology

Introduction

Amniotic fluid embolism (AFE) is a severe syndrome, an obstetric complication with a low incidence and a high fatality rate [1]. It mostly occurs at the end of first stage or/and in the second stage of the delivery with strong contractions, and may also happen in a short period of time after the delivery [2]. Due to maternal death frequently taking place in a short time after the urgent onset of AFE with an even dangerous disease tendency [3], it is very easily to cause a medical dispute, therefore, forensic identification for illustrating the cause and the mechanism of the death is very necessary. We encountered one case in our forensic practice not long ago, and now report it here to enrich the related data and discuss with colleagues for exchanging experiences on forensic identification of AFE.

Case description

A 38-year-old woman (gestation 2, production 1), who was previously healthy, was admitted to hospital for awaiting delivery after 41^{+1} weeks of pregnancy and one day of abdominal pain. The post admission examinations, including obstetric examination, indicated that her general condition was normal. The fetus was in a head-position, fetal presentation had been in engagement and the height was -3. The fetal heart rate was 140 beats per minute, and the estimated weight of fetus was about 3000 g. The cervical was in the middle, and its texture was soft and receptivity 60%. Uterus cervix had not opened at all, and the Bishop Score was 6. The amniotic membrane had not broken with a regular uterine contraction. The blood routine examination showed the number of erythrocyte decreased slightly (3.33×10^{12}/L), while urine routine test was no abnormal. The results: The biparietal diameter 9.3 cm and head circumference 33.5 cm, abdomen circumference 34.1 cm, the length of thighbone 7.2 cm, fetal heart pulse 122 bpm, the maxim con depth of amniotic fluid 7.0 cm, the thickness of posterior paries 3.3 cm, and maturity grade of placenta II, and no umbilical cord around the fetal neck from ultrasonic examination were in a normal range, too. At three thirty on the day after her admission, expectant mothers' cervical orifice opened fully. It was the moment that pale complexion, cyanosis of lips, tetany and drop of blood pressure (from 122/82 mmHg to 94/62

mmHg) broke out on the lying-in woman. After 3 minutes of oxygen uptake and intravenous injection of 10 ml Glucose Gluconate, maternal general conditions took a favourable turn: Convulsions stopped, lips got rosy, pulse was up to 94 bpm, breathing rate was 20 bpm, but blood pressure was still at a low level of 88/68 mmHg and the pale complexion persisted yet. The predelivery observation was carried on: The discipline of uterine contraction was 50 seconds/1-2 minutes and the height of fetal presentation +1. After then, at one to four, a baby boy was born by vaginal delivery under the episiotomy. Postpartum vaginal bleeding happened slightly and intermittently without soft birth canal laceration. The mother was sanity but with a pale look and a uterine inertia. Establishing vein-dual-channel, massaging the uterus and intramuscular injection of 250 ug Carboprost Tromethamine Injection were implemented. After 3 minutes, by pressing the bottom of womb, about 100 ml dark-red blood without clots was cleared out of the uterus with no obvious residual tissue. Then 20 units of oxytocin were injected into the cervix uterus. The uterus with a clear outline and rough inner membrance was observed. At twenty-three past six, just after her womb being cleared, pale face, dysphoria and polypnea appeared again on the puerpera who was sane with pulse being 50 bpm, pespiration 14 bpm, blood pressure 86/56 mmHg. Just two minutes later, apart from persistent pallor, unconsciousness even coma and adiaphoria began to arise. Then all indexes of pulse, respiration and blood pressure fell down to 0 followed by the stopping of heartbeat. After more than an hour of regular emergency rescue, her breathing and heartbeat had not yet recovered, so the clinical death was announced.

Major findings of forensic pathology autopsy and histopathological examination

The corpse frozen after death was routinely checked by comprehensive forensic pathology autopsy after 44 hours and 29 minutes from the death at a normal temperature. Every organ was checked via the macroscopic view, and samples from which were pathologically examined based on conventional paraffin pathology section, Haematoxylin-Eosin staining (HE) and light microscope observation.

The sclera, conjunctiva, lips, and all the nail beds of her hands and feet were pallor. There was some reddish liquid in bilateral pleural cavities and also in pericardial cavity. The spleen envelope shriveled. The uterus wall was osteoporosis and soft with a rough membrane surface of posterior paries and without laceration and perforation. Besides several vesicles that those diameters ranged 0.3 cm-0.5 cm found in the sections of cervix uteri, the mucous membrane was smooth. There was no obvious anomaly in the rest.

Amniotic fluid components, mainly cuticulated epithelium and meconium, were found in a number of small blood vessels and capillaries (**Figure 1A**, **1B**), and transparent thrombosis was seen in some capillaries (**Figure 1C**). A little homogeneous or cotton-shaped amorphous material pink-dyed was observed in some pulmonary alveoli (**Figure 1D**). A few hemorrhagic focuses were scattered in some pulmonary alveoli and pulmonary interstitiums (**Figure 1E**). The spleen blood sinuses were almost empty. Placental villi structures were tolerable. Endometrium was decidual tissues with scattered infiltration of nourish and inflammatory cells. Focal hemorrhages and a microvascular with the keratinizing epithelium were seen in the intrauterine membrane (**Figure 1F**). Mucous membrane of cervical epithelium fell off focally. Scattered focal inflammatory cell infiltrated in the mucous membrane with Nabothian cysts. There were some focal hemorrhages in the cervical tissue.

Discussion

AFE is a rare and severe obstetric complication which affects women during labor, delivery, or postpartum [2]. Some classic clinical manifestations such as dysphoria, shiver, emesis, bucking, dyspnea, cyanosis, rapid shock, and so forth can appear in the maternals with AFE [1-3]. The patients with a rapid onset may die within a few minutes [3]. And others, after the recovery of blood pressure, often suffer postpartum hemorrhage, blood discoagulate and sometimes systemic bleeding tendency, finally, then followed by renal failure, pulmonary failure, cardiac failure or multiple organ failure [4]. In our case, the typical clinical symptoms of anaphylactic shock, mainly microcirculation disorder symptoms caused by microvascular extensive expansion and central nervous system symptoms induced by brain hypoxia, successively performanced on the woman twice during and after the parturition according to

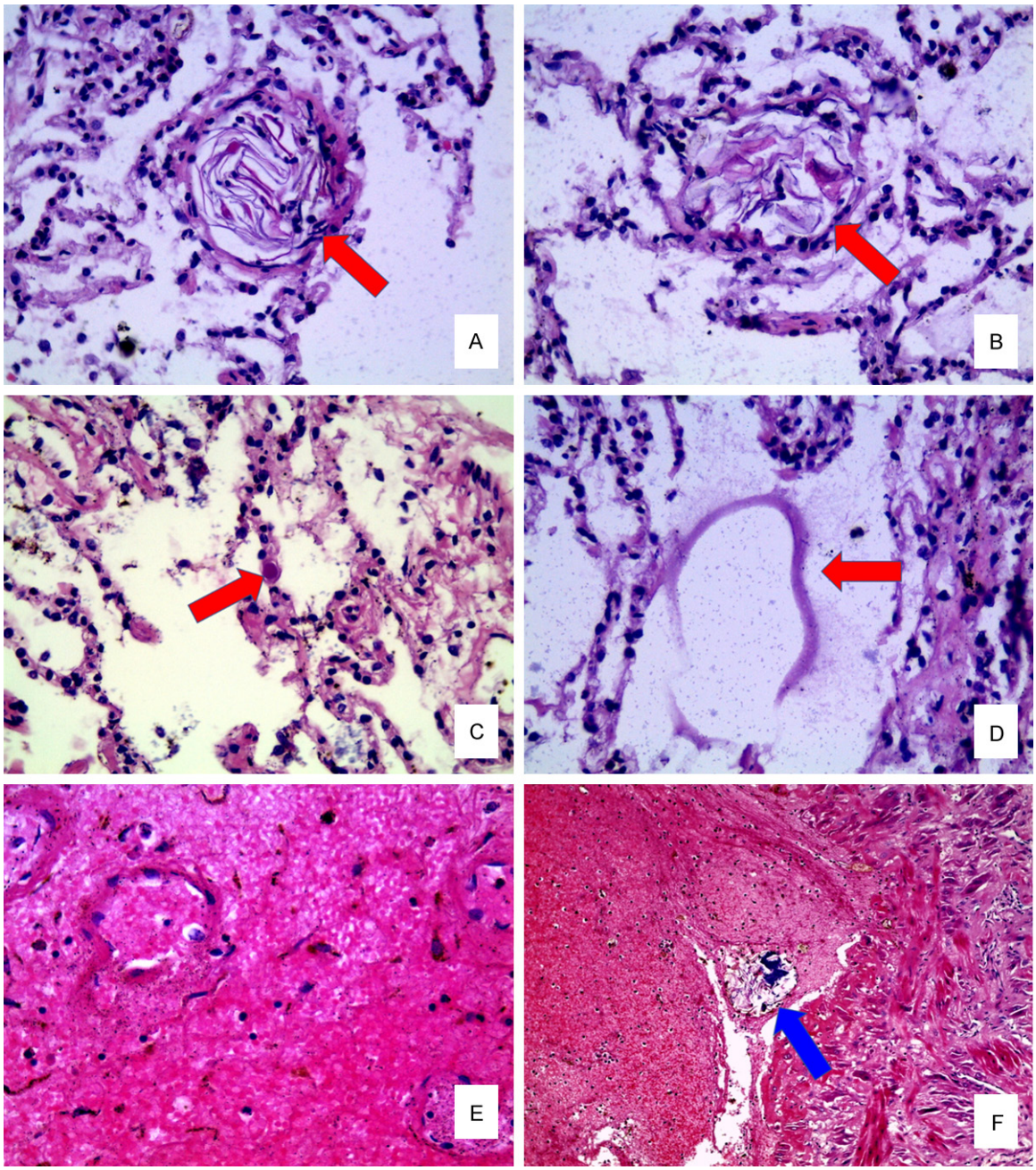

Figure 1. A, B: Amniotic fluid components, mainly cuticulated epithelium and meconium, were found in a large number of pulmonary small blood vessels and capillaries (pointed by the red arrows). HE×400. C: Transparent thrombosis were seen in some pulmonary capillaries (pointed by the red arrow). HE×400. D: A little homogeneous or cotton-shaped amorphous material pink-dyed was observed in some pulmonary alveoli (pointed by the red arrow). HE×400. E: A few hemorrhagic focuses scattered in some pulmonary alveoli and pulmonary mesenchyme. HE×400. F: Focal hemorrhages were seen in the intrauterine membrane, and a microvascular with the keratinizing epithelium (pointed by the blue arrow). HE×100.

her medical records. The one, including pallor, cyanosis of lips, carpopedal spasm and fall of blood pressure-from 122/82 mmHg to 94/62 mmHg, suddenly appeared in the maternal firstly at the time of uterus cervix fully opening. Although the seizures stopped and her lips got ruddy after oxygen inhalation and calcium gluconate intravenous slowly pushed, pale complexion still existed with pulse being quickened up to 94 times/minute. And her blood pressure

fell further to 88/68 mmHg with a postpartum vaginal persistent bleeding lasted more than 2 hours in the absence of soft birth canal laceration. And the other one happened after the implementation of complete curettage of uterine cavity, pallor, dysphoria and tachypnea performing on the postoperative maternal with Pulse 50 bpm, Respiration 14 bpm, Blood pressure 86/56 mmHg, but the woman was still sanity. And after only 2 minutes, the conditions of lying-in woman deteriorated to unconsciousness and coma, non-response and with Pulse, Respiration and Blood pressure falling down to 0, and the woman died.

For diagnosing AFE, the usual clinic practice is via the typical clinical symptoms: Dyspnea, cyanosis, cardiovascular dysfunction, hemorrhage, coma [5, 6], fetus compositions detected in the maternal blood [4], quantitative test of antigen Sialyl Tn [7, 8] and DIC. Considering the puerpera clinical symptoms and her high risk factors of AFE [1, 9-14]: A multipara aged 38, a history of complete curettage of uterine cavity and cervical oxytocin injection, the diagnosises: Suspicious DIC, delayed AFE, peripartum cardiomyopathy and doubtless full-term child birth were made.

For forensic science, different from clinical medicine, in addition to the details of a case and the clinical manifestations of the dead, the diagnosis of AFE is mainly dependent on the findings in obduction and histopathological examination.

In this case, amniotic fluid components, mainly cuticulated epithelium and meconium, were found in a number of small blood vessels and capillaries (**Figure 1A**, **1B**), and transparent thrombosis was seen in some capillaries (**Figure 1C**). Hyaline casts could be seen in some distal convoluted tubule lumens. A microvascular in uterine wall was blocked with the keratinizing epithelium (**Figure 1F**). In view of this, the diagnosis of amniotic fluid embolism was not difficult to make.

For the mechanism of death or pathogenesis of AFE, different from the cause of death or pathogeny, public opinions are divergent. Many scholars believe that the main reason for the mechanical obstruction of pulmonary vessels in AFE is the kind of tangible substance, such as fetal fat mass, the squamous cells and vellus hair falling from fetal skin and fetal meconium, entering the blood flow. On the other hand, it is worth noting that anaphylactic shock caused by amniotic fluid has been long before put forward as one of the important reasons for AFE death [15]. Some of the scholars, for instance, Clark believed that allergies might be the main cause of AFE attributed to the performance of AFE was similar to allergies or toxic shock (endotoxin) [3]. The amniotic fluid materials entering motherly circulation could lead to the clinical symptoms through endogenous mediators such as histamine, bradykinin, cytokines, prostaglandins, leukotrienes and thromboxanes [3, 15]. He even suggested that the name of amniotic fluid embolism should be replaced with anaphylactoid syndrome of pregnancy [3].

In this case, we found that the typical clinical symptoms of anaphylactic shock, mainly microcirculation disorder symptoms caused by microvascular extensive expansion and central nervous system symptoms induced by brain hypoxia, successively performanced on the woman twice during and after the parturition following by her death. So it was believed that the cause of the woman's death was AFE and the mechanism was more inclined to anaphylactic shock caused by amniotic fluid rather than pulmonary embolism.

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Disclosure of conflict of interest

None.

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