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Case Report

Amniotic fluid pulmonary embolism and COVID-19: Case report ☆,☆☆

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ABSTRACT

Amniotic fluid embolism is a rare complication of peripartum. It is caused by the entry of fetal components into the maternal systemic circulation. There are 2 main types: typical; it presents with the triad of hemodynamic collapse, respiratory distress and disseminated intravascular coagulation type coagulopathy, while atypical; disseminated intravascular coagulation does not occur. SARS CoV-2 infection causes coagulopathy due to the alteration of Virchow's triad and coagulation factors. We present the case of a 21-year-old pregnant woman who consulted for premature rupture of membranes, with an indication for cesarean section, and during surgery presented bradycardia, hypotension, and desaturation until cardiorespiratory arrest. An angiotomography showed amniotic fluid embolism associated with pulmonary edema, ruling out differential diagnoses associated with the disease, leaving as the only cause of the infection confirmed by COVID-19, which, it was inferred, was closely related to the immunological disorder suffered by the patient.

Introduction

Amniotic fluid embolism (AFE) was first described in 1941 by Steiner and Luschbaugh. It is a rare medical emergency, characterized by a systemic inflammatory process mediated by the entry of amniotic fluid and placental debris into the maternal systemic circulation, which triggers an immune and coagulant storm [1,2], causing the classic triad of AFE; hypotension or cardiorespiratory arrest, hypoxia and disseminated intravascular coagulation (DIC) [1–3].

AFE affects 1.7:100,000 mothers; mortality is estimated at 61%-86% of cases, with a fetal survival rate of 70% [4,5]. Diagnosis is by exclusion [5,6] and treatment is based on interdisciplinary work, ensuring the airway, guaranteeing adequate oxygenation, and circulatory support [4–7].

SARS-CoV-2 virus, a zoonosis described in 2019, can present clinically asymptomatic forms or cause severe mani-

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festations, leading to death. An acquired syndrome known as COVID-19 coagulopathy has been described, caused by alterations in Virchow's triad and changes in coagulation factors, due to an exaggerated immune response causing DIC in pregnancy [7,8].

An exhaustive review of the literature found that there are no reported cases of AFE associated with COVID-19. We present the case of a young patient who developed this clinical spectrum, and was promptly managed by a multidisciplinary team with successful recovery.

Presentation of the case

A 21-year-old woman consulted the emergency department at 38 weeks of gestation for premature rupture of membranes (PROM) and occasional contraction-type abdominal pain. The pregnancy was unplanned, she had 5 well-controlled and tolerated prenatal check-ups. She was alert and vital signs were normal. Obstetric evaluation showed fetal movement, fetal heart rate of 145 beats per minute, and cervix without cervical changes.

The admission paraclinics reported; a normal hemogram and negative COVID-19 antigen test, normal biophysical profile (Table 1), reactive fetal monitoring, with irregular uterine dynamics. Pregnancy at term, PROM in the prepartum phase, and vaginosis due to whitish vaginal discharge were considered. She was hospitalized for labor induction with oxytocics. After 23 hours without cervical changes and 34 hours of PROM, induction was considered failed. During the procedure she presented bradycardia, hypotension, and desaturation until cardiorespiratory arrest, advanced resuscitation maneuvers were started for 6 minutes with return of spontaneous circulation, she required mechanical ventilation (MV), sulbactam ampicillin, 3 doses of tranexamic acid and dexamethasone. The newborn Apgar score was 7 points at 1 minute, 8 points at 5 minutes, and 9 points at 10 minutes.

Control paraclinical tests were fibrinogen 346 mg/dL (normal: 200-400 mg/dL), arterial blood gasses without oxygenation disorders or acid-base imbalance, normal coagulation times (prothrombin time PT: 13 seconds, thromboplastin time TPT 28 seconds) (normal: PT; 11-13. 5 seconds, PTT; 25-35 seconds), lactate dehydrogenase 1. 203 IU/L (normal: 105-333 IU/L) and D-dimer >10,000 ng/mL (normal: less than 500 ng/mL). An angiotomography showed pulmonary embolism due to amniotic fluid and pulmonary edema (Table 1, Fig. 1). Respiratory infection was considered in an asymptomatic patient, polymerase chain reaction was performed by nasopharyngeal swab, which was positive, confirming SARS-CoV-2 infection; elevated risk of pneumonia, mild respiratory distress syndrome (ARDS), sepsis of pulmonary origin and cardiogenic shock.

Table 1 – Diagnostic aids.

Examination	Images	Description
Biophysical profile	Abdominal	Movements: 2 Tone: 2 Amniotic fluid: 2 Amniotic fluid index: 6.7 cm Respiratory movements: 2 Heart rate: 143 bpm
Angiotomography	Chest	Interstitial-alveolar opacities in the upper and lower lobes. Bilateral posterior basal consolidation. Left pneumothorax.

Source: Own elaboration.

He remained in the intensive care unit for 6 days with favorable clinical evolution, and the MV was withdrawn. New paraclinical tests showed leukocytes 6250 × 103/mm^3 and neutrophils 60%, in normality with respect to the initial ones that were elevated (leukocytes 15,600 × 103/mm3 and neutrophils 85%) (normal range: leukocytes; 5000-10,000×103/mm^3, neutrophils; 50%-65%), procalcitonin <0.05 ng/mL (normal: <0.5 ng/mL), blood cultures, urine cultures and orotracheal tube culture were all negative. Given a satisfactory recovery, the patient was discharged from the hospital.

Discussion

AFE is an obstetric complication of unknown cause. It is caused by the entry of amniotic fluid and fetal components (meconium, mucus, sebum, lanugo, and amniotic cell debris) into the maternal circulation during labor, which causes exaggerated activation of the immune system [1,5].

Immune activation mediated by leukotrienes and proinflammatory cytokines causes macrophage and polymorphonuclear activation, releasing reactive oxygen species, proteases, and nitric oxide, stimulating numerous reactions by active and prothrombotic substances that generate smooth muscle contraction at the pulmonary level, generating vasoconstriction, bronchoconstriction, capillary leakage, distributive shock, ARDS and multiorgan failure as evidenced in this case report [3,9].

The risk factors for AFE remain unclear. One study shows that multiple pregnancy, maternal age, polyhydramnios, and induction of labor with prostaglandins and oxytocin predispose to developing AFE [3]. Another study points out that heart disease and cerebrovascular disorders increase the risk by 25-75 times, while eclampsia and renal disease increase the risk by 7-13 times [9].

The AFE in this case was not related to risk factors and, on the contrary, the patient was young, nulliparous, without diseases and the administration of oxytocics did not cause coagulation alterations. Duron González et al. [10] state that, given the unpredictable and unpredictable nature of AFE, there is no expert opinion to justify whether routine obstetric care may or may not be a risk factor for AFE.

There are two types of AFE: typical AFE: characterized by the classic triad: ARDS, hypotension and DIC. Atypical AFE is divided into cardiopulmonary AFE without DIC as in the case presented and, on the other hand, fibrinogen, coagulation times, platelets and liver function were normal. Finally, the other type, AFE with DIC without circulatory and respiratory alterations [1–5].

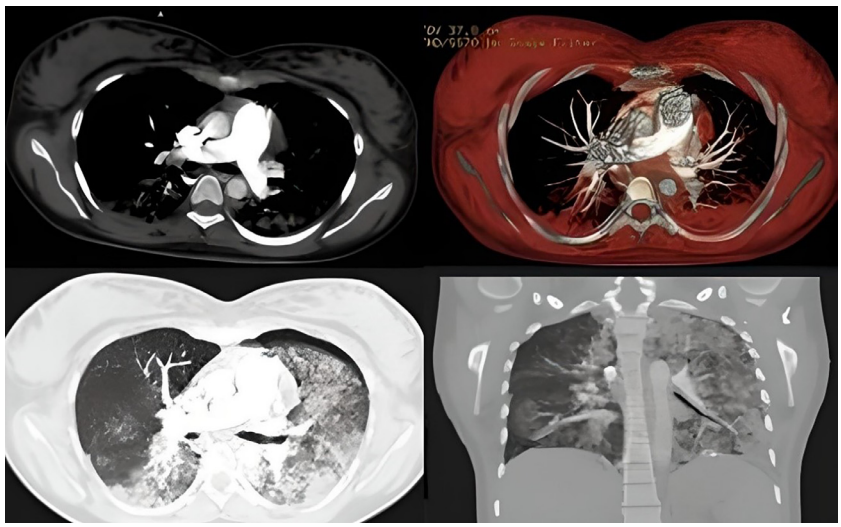

Fig. 1 – AFE associated with pulmonary edema and left pneumothorax. No pulmonary thromboembolism.

The diagnosis of AFE is clinical and of exclusion [3]. In this case, differential diagnoses such as coagulopathies, liver involvement, pulmonary thromboembolism, sepsis, heart disease, peripartum cardiomyopathy, and pulmonary infections were ruled out.

SARS-CoV-2 triggers cytokine-mediated immune cascades that increase proinflammatory and prothrombotic effects [6–8], leaving the incidental finding of COVID-19 infection as the sole cause, allowing the inference that AFE was secondary to SARS-COV-2 infection.

Conclusions

At present, there are no predictors and/or markers for a timely and reliable diagnosis of AEF, and there is a need to develop them to improve preventive and therapeutic interventions. Therefore, care by a multidisciplinary team remains the main therapeutic measure to increase survival, improve prognosis, and decrease morbidity and mortality of newborns and mothers.

Patient consent

Written, informed consent was obtained from the patient for publication of this case.

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