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CASE REPORT

Death Due to Traumatic Amniotic Fluid Embolism Following a Fall From a Bed: A Case Report

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Abstract

Amniotic Fluid Embolism (AFE) is a rare obstetric emergency with a potentially fatal outcome, occurring in approximately 1.9 to 6.1 cases per 100,000 deliveries, with variations observed among different countries. Obstetric management continues to be the most common preceding event associated with AFE, making it the primary contributing factor. However, traumatic AFE cases are being typically reported as isolated case reports in literature. While previous case reports on the traumatic basis of AFE have primarily associated AFE with blunt-force abdominal trauma resulting from car accidents and evident gross injuries, this article presents a unique case of AFE following minor blunt trauma from a self-fall from a height of 2.5 ft, where no macroscopic lesions were observed. The autopsy lung histopathology exhibited alveolar spaces filled with serous fluid and lymphocytes, as well as emboli composed of fatty material in blood vessels. Some blood vessels contained keratinous emboli. Severe pulmonary oedema and embolized vessels were consistently observed throughout the lung sections. We hypothesise that a simple fall from a height of 2.5 feet onto a firm surface, leading to transmitted blunt force to the abdomen, may trigger AFE by disrupting the flow dynamics of the foeto-placento-maternal circulation. Despite the absence of gross injuries, the underlying pressure transmission is emphasized as a significant factor in AFE initiation due to the disruption of the microvasculature. This case report highlights the importance of recognizing the possibility of AFE even in cases of seemingly minor trauma without visible external signs.

Keywords: Amniotic Fluid Embolism, Disseminated Intravascular Coagulation, Respiratory Distress Syndrome, Obstetric Trauma, Anaphylactoid Syndrome

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Introduction

Amniotic Fluid Embolism (AFE) is a potentially fatal obstetric emergency characterized by sudden cardiorespiratory collapse and disseminated intravascular coagulation. The onset is abrupt, with sudden cardiorespiratory collapse, severe coagulopathy, and refractory to resuscitation. The incidence of amniotic fluid embolism (AFE) is estimated at approximately 1.9 to 6.1 cases per 100,000 deliveries. The exact prevalence is unknown due to incorrect diagnosis and failure to record nonfatal occurrences [1].

The onset of AFE requires two necessary conditions. Firstly, an influx of foetal components into the maternal circulation, and secondly, a significant pulmonary embolus or maternal immune/ anaphylactoid reaction against the amniotic fluid or foetal components [2].

The introduction of amniotic fluid into maternal circulation can transpire through multiple ways, including amniotomy (artificial rupture of foetal membranes for labour induction or augmentation), uterine or cervical tears during vaginal delivery, or caesarean section procedures. This influx can occur via several potential sites of entry: the endocervical veins, uterine trauma sites, or the placental attachment site. Subsequent proposed responses include either a reaction secondary to the obstruction of pulmonary blood flow or a proinflammatory reaction secondary to the release of cytokines derived from arachidonic acid from the amniotic fluid, resulting in an anaphylactoid reaction. In either case, the series of events result in pulmonary oedema and corresponding pulmonary hypertension leading to acute respiratory distress syndrome, hypotension, and hypoxia [3].

In obstetric practice, AFE is encountered usually during the process of labour and its management. However, forensic pathologists are concerned about the traumatic origins of amniotic fluid embolism. When an expectant mother dies as a result of obstetric trauma, it is critical for the pathologist to determine how the trauma occurred. This finding is extremely important in legal processes because such accidents result in the death of both the mother and the foetus. It is also critical to emphasise that even seemingly slight injuries during pregnancy pose major harm to the developing foetus's well-being. In this article, we present a case of a seemingly insignificant trauma that led to a fatal amniotic fluid embolism to the mother and both the mother, and the foetus succumbed to the events following the trauma.

Case report

A 21-year-old female, gravida 2, para 1, at 8 months of gestation, was found unconscious in a left lateral position on a marble floor late one night, adjacent to her usual sleeping place on the bed (the height of the bed is 2.5 ft. from the ground). Despite immediate transportation to the hospital, she was declared dead upon arrival. Given the absence of antecedent symptoms or known risk factors for adverse pregnancy outcomes, an autopsy was conducted to determine the cause of her sudden demise. Throughout her antenatal care, all clinical and laboratory parameters remained within normal ranges. The police registered the case as a suspicious death, with the presumed cause being "death due to accidental fall from a bed." The postmortem examination was conducted by a team of doctors with expertise in forensic pathology, obstetrics, and surgical pathology.

On external examination, the corpse showed generalized cyanotic features. Two contusions measuring 3 x 3 cm each were present over the posterior aspect of the left shoulder joint and the posterolateral aspect of the upper part of the left arm. Internally, the autopsy revealed no outer surface abnormalities in the uterus. On further dissection, placental attachments were normal, and liquor was adequate and clear. There were no signs of haemorrhage or injury in the entire uterine cavity. A 08-month-old male foetus was found inside the uterus, measuring 40 cm in length, and weighing 2.6 kg, showing no injuries. All major visceral organs were congested and did not yield any positive contributory finding toward the cause of death. Apart from the routine viscera for chemical analysis (liver, kidneys, blood, and stomach & intestine along with contents), swabs from the vagina, cervix, and anus were collected to rule out any presence of a foreign body or chemical poisons. However, both of the investigations turned negative.

Histopathological examination (HPE) of lung sections displayed alveolar spaces filled with serous fluid and lymphocytes within the septa. The blood vessels exhibited red blood cells and emboli, identified as globules of fatty material. The interstitium showed evidence of serous fluid. The grossly grey-white appearing areas of the lung cut section revealed lymphoid aggregates, fibro-collagenous bundles, nerve bundles, and dilated, congested blood vessels in microscopy. Notably, some blood vessels contained granular eosinophilic emboli (keratinous). Severe pulmonary oedema and embolized vessels were evident in all the lung sections studied. Other organ histopathology yielded normal findings, except for the expected pregnancy-related changes in the uterus. More particularly, the placental histology was also normal.

Based on the positive lung histopathological findings (Figures 1-3), postmortem findings, and other negative ancillary investigation results, traumatic amniotic fluid embolism was considered the most probable cause of death. Immunohistochemistry was not attempted in this case due to logistic constraints.

This unusual case highlights the significance of considering traumatic amniotic fluid embolism (AFE) in the setting of falls during pregnancy, as it could lead to maternal mortality. To the best of our knowledge, this is the first reported instance of AFE resulting from a fall from a bed onto a hard surface.

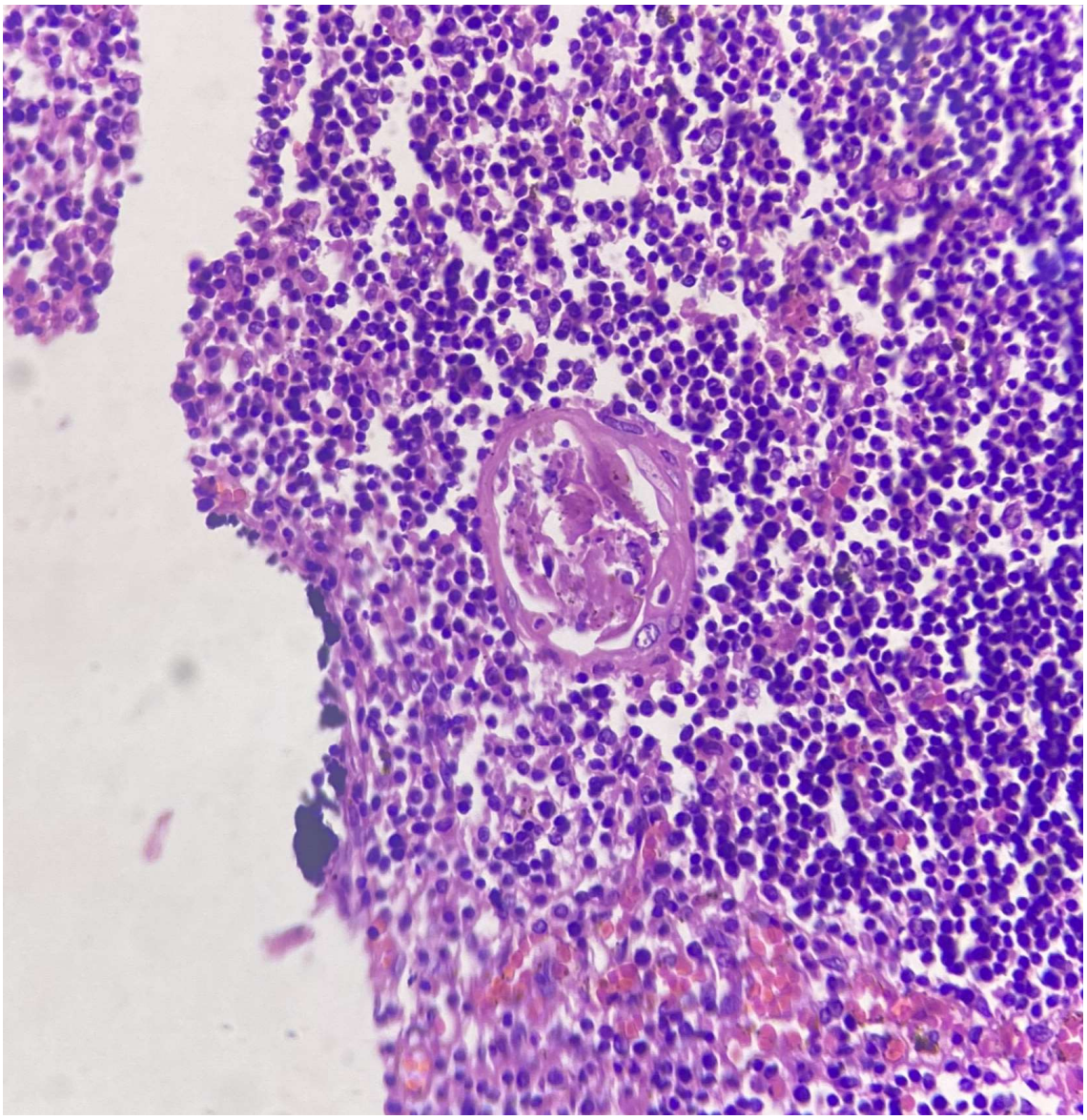

Figure 1. Photomicrograph of HPE Lung: Abundant lymphoid aggregates & blood vessel with granular eosinophilic emboli (keratinous), 40X Haematoxylin and Eosin.

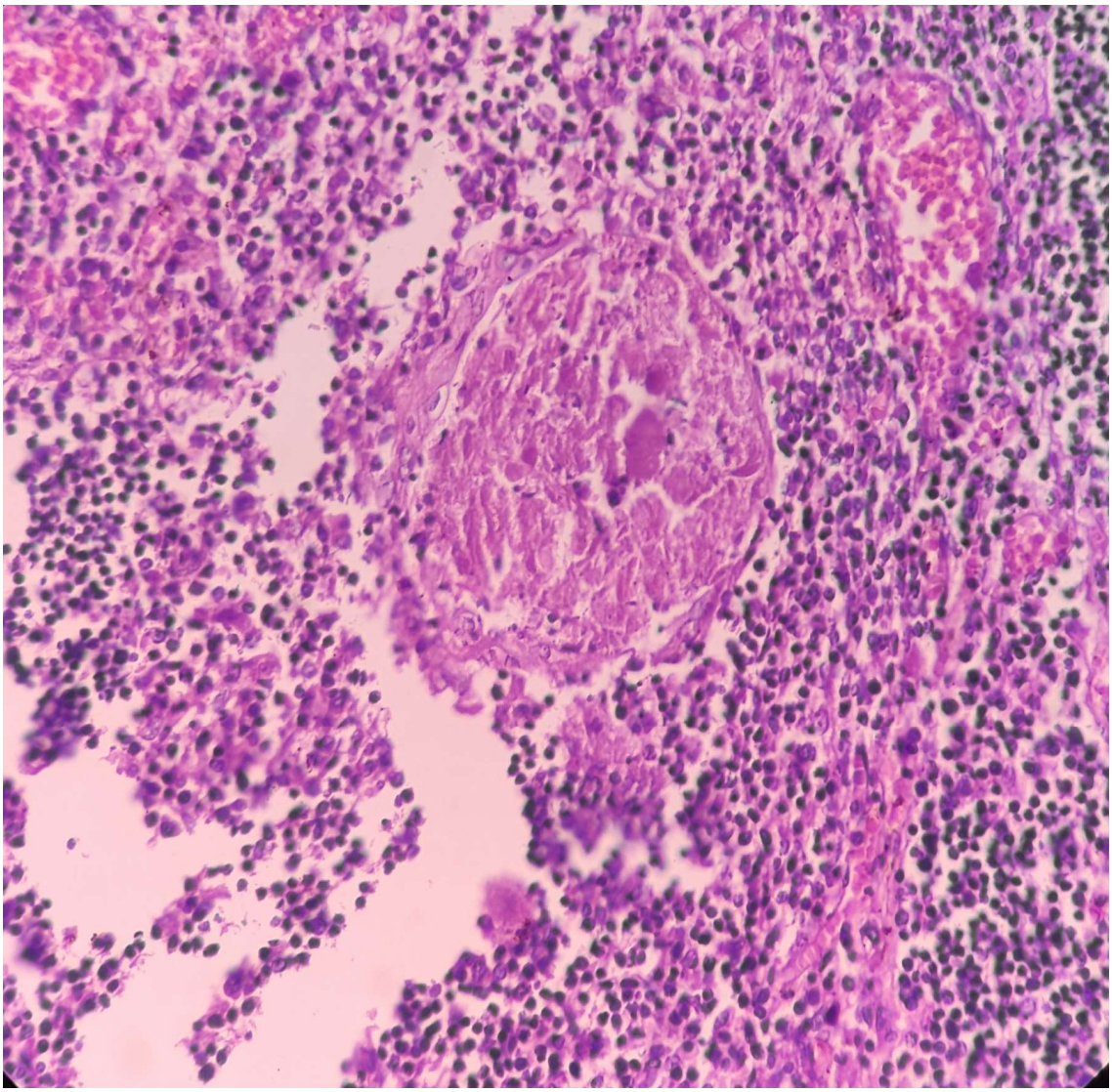

Figure 2. Photomicrograph of Lung HPE: Abundant lymphoid aggregates & blood vessel showing granular eosinophilic emboli (keratinous), 40X Haematoxylin and Eosin.

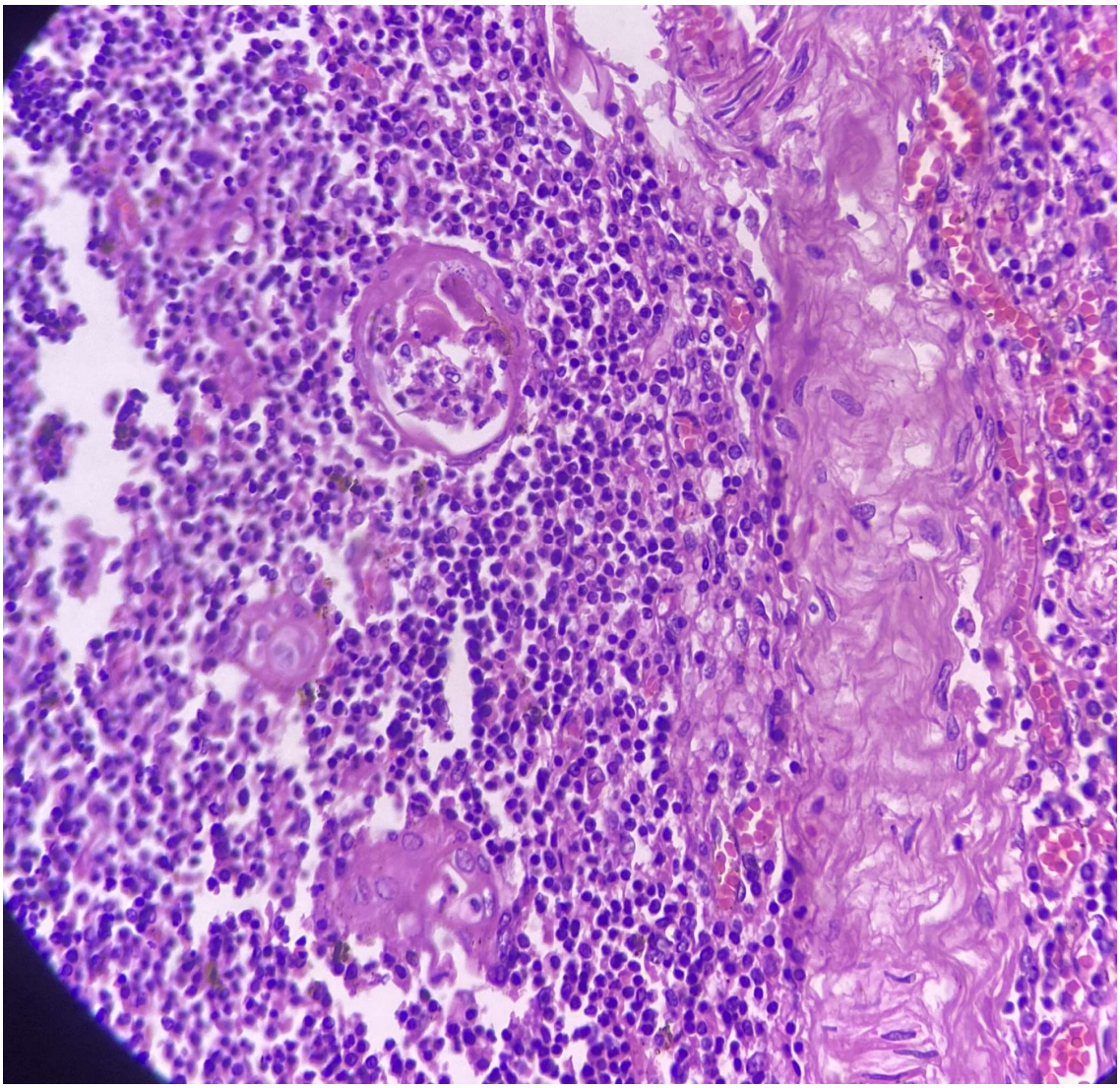

Figure 3. Photomicrograph of Lung HPE: Abundant lymphoid aggregates & blood vessel showing granular eosinophilic emboli (keratinous), 40X Haematoxylin and Eosin.

Discussion

Blunt-force abdominal trauma during pregnancy can pose a significant risk for AFE, as it may cause direct injury to the uterus, leading to the entry of amniotic fluid into the maternal circulation and the subsequent pathophysiological responses described above. Trauma-induced AFE occurs when there are tears in the uterus or cervix due to various scenarios, such as road accidents, falls from beds, or direct blows to the abdomen.

The main question we need to address for clarity and understanding in our case is whether Amniotic Fluid Embolism (AFE) can be caused by blunt force trauma, even when there are no obvious tears or visible damage in the maternal tissues. It is plausible to consider that blunt force trauma could lead to changes in the flow dynamics of the foeto-placento-maternal circulation, potentially originating from the large placental surface, even if there are no

apparent macroscopic lesions or visible signs of damage.

In this scenario, it is hypothesized that the impact of blunt force may damage delicate microvascular structures, leading to subtle microscopic alterations not immediately observable. This trauma-induced disruption may affect the placental or uterine microvasculature, causing the release of amniotic fluid components into the maternal bloodstream. The formation of small emboli from these micro-scale disruptions can obstruct pulmonary vessels, resulting in a rapid increase in pulmonary vascular resistance, or can cause pulmonary vasospasm apart from initiating the cytokine storm.

In our specific case, the second crucial question to be answered is whether there was a de novo or spontaneous occurrence of Amniotic Fluid Embolism (AFE) that led to the agony and fall, or if the trauma was the initial event. Precisely put, we need to determine whether AFE occurred independently, without any external trigger, or if the trauma occurred first and potentially contributed to the development of AFE. It is important to note that existing literature tends to heavily lean against the possibility of spontaneous AFE, indicating that there is usually an identifiable event or trigger that precedes its occurrence.

In this context, pregnancy-related and pathology-related anaphylactoid syndromes of pregnancy are not relevant to our discussion. However, a summary of the clinical picture, clinical parameters for evaluation, autopsy related gross and histological findings in cases of AFE are summarised in Table 1.

Table 1: A Summary of Amniotic Fluid Embolism/Anaphylactoid Syndrome of Pregnancy [4-13]

1	Clinical features/ Risk factors	Hypoxia, Hypotension, Seizures, Disseminated Intravascular Coagulation (DIC), Altered mental status, Shortness of breath Cardiac arrest, Shock, Death Risk factors: Age > 35years, African/ other American, Polyhydramnios, Blunt abdominal trauma and surgical trauma, Procedures like pregnancy termination, amniocentesis, Pre-eclampsia/ eclampsia, Induction of labour, Placenta previa/ abruption, Foetal distress, Instrumental delivery, Vaginal breech delivery, Caesarean delivery Cervical laceration/ uterine rupture, Manual removal of placenta, Multifetal pregnancy, Gestational age < 37weeks Postdated pregnancy, Foetal macrosomia.
2	Pathogenesis	A breach in physical barriers between maternal and foetal compartments at the endocervical veins, uterine trauma sites, and placental attachment site is one of the primary prerequisites. Foetal components in amniotic fluid cause pulmonary vasospasm due to anaphylactoid reaction. It is an immune inflammatory reaction called an anaphylactoid syndrome during pregnancy

		AFE: necessitates two components: 1. Foetal components in maternal circulation. 2. Significant pulmonary embolus or maternal immune/ anaphylactoid reaction. • In the cardiovascular type of AFE: Physical obstruction in maternal micro vessels of various organs results in shock and loss of consciousness. • DIC type of AFE: Clinical AFE with secondary Post Partum Haemorrhage (PPH) of unknown aetiology. • Amniotic fluid into uterine vessels: Anaphylactoid reaction leading to the oedematous uterus. • In uterine type AFE: Histamine, bradykinin, inflammatory cytokines such as IL 8 and procoagulant substances lead to endothelial activation and an eventual massive inflammatory reaction. The severity of an anaphylactoid reaction depends on the balance between the inflow amount and quality of the amniotic fluid and the potential of biological inhibitors.
3	Laboratory Parameters/ Investigations	Increased levels of fibrin products, Decreased levels of fibrinogen, Increased levels of PT and PTT, Thrombocytopenia, Echocardiography ECG CXR CBC, LFT, RFT, Coagulation profile, Serum electrolytes, arterial pH, ABG, Glucose, cardiac enzymes(Sialyl-Tn <46IU/ml), ZnCP1 <1.6pmol/L- detection indicates AFE, Complement 3 (80-140mg/dl) - decreased Complement 4 (11-34mg/dl) – decreased IL 8 (<20pg/ml) – increased C1 esterase inhibitor – decreased
4	Treatment / Monitoring	Massive transfusion protocols (fresh frozen plasma preferred over RBCs), Hemofiltration, and plasma exchange transfusions, High dose corticosteroids, C1 inhibitor concentrate, Volume replacement by crystalloids/ colloids. (500ml of fresh blood increases fibrinogen by 12.5mg per 100ml and it also adds 10000 to 15000 platelets per cu mm. 1-unit FFP raises fibrinogen by 5-10mg/dl, 1 unit cryoprecipitate raises fibrinogen by 5-10mg/dl, 1 unit platelet concentrate raises the platelet count by 7500/ml, 1-unit Packed RBC raises HB by 1g/dl, which increases oxygen carrying capacity)

		In acute conditions: IV heparin 5000 units at 4-6 hours intervals, Fibrinolytic inhibitors: EACA: inhibits plasminogen and plasmin. Aprotinin (Trasylol) Cardiotocography Pulse oximetry
5	Postmortem Appearances:	The postmortem examination reveals several findings related to Amniotic Fluid Embolism (AFE) in the lung, including pulmonary oedema, congestion, and focal atelectasis. Additionally, in cases of Uterine type AFE, features of acute myometritis are observed. Diagnosing AFE primarily relies on histological analysis. To confirm the diagnosis, an examination of pulmonary artery blood is necessary. Furthermore, investigation for genital tract trauma, such as tears or ruptures in the cervix, vagina, uterus, or adjacent soft tissues, should be conducted. In cases where no apparent abnormalities are observed during the evisceration process, a comprehensive examination of the entire group of organs is crucial. Histopathological findings in the right ventricular myocardium may indicate an acute increase in pressure in the pulmonary circulation, further supporting the diagnosis of AFE.
6	Histopathology / Immunohistochemistry	Foetal components: Routine H&E staining (epithelial squamous cells, lanugo hair, fat) Alcian blue: mucin, Attwood's stain – stains the keratin red and mucus turquoise blue. Lendrum Stain: Contains phloxine tartrazine detects squames by staining them red. Sudan black or oil red – vernix caseosa. IHC: Cytokeratin AE1/AE3: foetal squamous cells, ZnCp-1 stain for meconium C5a receptor(CD88) stain: complement activation and anaphylactoid formation in various organs Amniotic components in pulmonary vessels: Alcian blue and ZnCp-1 stain AE1/AE3 staining of the lung: Intravascular positivity of foetal squamous cells. Immune histochemical staining for CD 88: positive in stromal cells around pulmonary capillaries and inflammatory cells in the alveolus. In Myometrium of DIC type PPH: tryptase halos around activated mast cells, elastase positive neutrophils, CD 68 positive macrophages.

In the context of Amniotic Fluid Embolism (AFE) and its association with trauma, it is worth noting that although trauma, especially from road traffic accidents, is acknowledged as an important cause of AFE in Western countries, there are relatively few reported cases in the medical literature. Specifically, there has been limited case reports that link trauma to AFE. Some of these reports involved car accident cases where both chorionic villi embolism and AFE were observed. Additionally, there have been instances of the successful revival of pregnant women from AFE, as reported by researchers. However, a significant difference between several of these reported cases in the literature and the present case is the absence of noticeable gross features of an injury identified as the site of breach responsible for triggering AFE [14-19].

Conclusion

In conclusion, our hypothesis suggests that a simple fall from a height of 2.5 feet onto a hard surface, resulting in blunt force transmitted to the abdomen, has the potential to cause Amniotic Fluid Embolism (AFE) without any gross lesions. Our reasoning is based on the notion that such trauma could lead to alterations in the flow dynamics of the foeto-placento-maternal circulation, potentially affecting the microvasculature. Although there were no visible macroscopic injuries, the underlying changes in microvascular structure could play a significant role in triggering AFE in these cases.

Conflicts of Interest: None to declare.

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Ethical Considerations: Addressed by the authors.

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