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CASE REPORT

Analysis of Death Due to Pulmonary Embolism, a Case Series

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ABSTRACT

Introduction: This autopsybased case series analyzes six fatalities resulting from pulmonary, fat, and amniotic fluid embolism, highlighting their clinical presentations, pathological features, and diagnostic challenges.

Case Report: This study included six medicolegal autopsy cases in which death was attributable to embolic phenomena. The presence of antemortem thrombi, fat globules with bone marrow elements, or fetal squames was used as confirmatory evidence of pulmonary, fat, and amniotic fluid embolism, respectively. Findings were correlated with premortem symptoms for diagnosis. Pulmonary thromboembolism was identified in two adults, they developed sudden dyspnea, tachycardia, or hypotension prior to collapse, and autopsy consistently revealed firm antemortem thrombi within the pulmonary trunk or arterial branches. Fat embolism was confirmed in two males, histology demonstrated fat globules and bonemarrow elements in the pulmonary arteries. Two postpartum females died

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suddenly after delivery and both demonstrated classic features of amniotic fluid embolism, including fetal squames and microthrombi within pulmonary vessels.

Conclusion: Across all patients, mechanisms of death included massive pulmonary arterial obstruction, hypoxia, rightheart strain, and disseminated intravascular coagulation. Amniotic fluid embolism and fat embolism typically manifest with nonspecific signs and sudden deterioration, limiting laboratory-based diagnosis during life. Thus, autopsybased histopathology continues to serve as the definitive diagnostic method for amniotic fluid embolism and fat embolism. Improved early recognition, appropriate thromboprophylaxis, timely stabilization of fractures, and rapid multidisciplinary management in obstetric emergencies are essential to reduce mortality and medicolegal burden associated with these embolic syndromes.

KEYWORDS

• Autopsy • Amniotic fluid • Embolism • Fat • Thrombus

INTRODUCTION

An embolus is a detached mass that is carried through the circulation from its site of origin to another location[1]. Pulmonary embolism causes its effects due to blockage of an artery in the lungs or through biochemical mechanisms involving interactions between blood constituents and the endothelium of the blood vessel wall. Among cardiovascular diseases, pulmonary embolism is the third most common cause of death.[2]

This case series describes findings from autopsies in which the mechanism of death involved the effects of blockage of pulmonary circulation due to antemortem thrombus, fat globules along with bone marrow elements, and amniotic fluid along with debris. According to the ICD-10[3] classification of diseases, pulmonary embolism is classified under I 26. Pulmonary embolism causes a sudden blockage of the pulmonary artery or one of its branches in the lungs. This can result in the permanent damage to lung tissue (infarction), decreased oxygenation of blood (hypoxia), and effects of hypoxia on other organs and tissues. The goals of treatment include detecting and correcting the cause of embolism, clearing the embolism from circulation, and providing supportive or definitive treatment aimed at re-establishing circulation. Pulmonary embolism may occur due to a variety of causes, both immediate and delayed. The signs and symptoms of fat embolism and amniotic fluid embolism are nonspecific; additionally, treatment in such cases is mainly supportive.

In developing countries such as India, it is particularly difficult for treating physicians to diagnose and manage fat and amniotic fluid embolism. However, in cases of thromboembolism, it is often possible for diagnosis and treatment before fatality occurs. The autopsy surgeon should study the clinical case history prior to postmortem examination, maintain a high index of suspicion for the possibility of fat and amniotic fluid embolism, and should also make the fixed lungs available for histopathological study. Physical examination and weight of the lungs cannot be used as a screening tool in suspected cases of pulmonary embolism at autopsy[4,5]. The present case series describes six cases in which the final pathophysiological state before the death of the victim, was because of emboli, which was eventually detected by postmortem histopathological examination. Antemortem thrombi, fat globules and bone marrow components, and fetal squames from amniotic fluid within pulmonary circulation were identified on histopathological examination using hematoxylin and eosin staining.

CASE REPORT

Patient 1: Antemortem thrombi in the pulmonary circulation

A 27-year-old male sustained a road traffic accident, which resulted in a comminuted fracture of the right tibia and fibula. He underwent a planned surgical intervention for fixation and correction of fracture involving the proximal tibia and fibula, and for the segmental fracture of the right fibula. Postoperatively, the right lower limb was supported by an external fixator to immobilize the limb. Despite this inpatient hospitalized care, he

died 25 days after the road traffic accident. On the day preceding death, he had complained of breathlessness and dyspnea. Auscultation of cardiovascular system revealed tachycardia and irregular heartbeats, which were confirmed by electrocardiography. The recorded blood pressure indicated hypotension. Auscultation of the respiratory system did not reveal any evidence suggesting crepitations or rhonchi. The medication administered during hospitalized care included aspirin, analgesics, and other supportive treatment to aid recovery following corrective surgical interventions for the fracture.

Autopsy findings: On External examination, therapeutic wounds resulting from surgical procedures, split-skin grafting, and application of the external fixator were present and were documented. There were noticeable signs of asphyxia, characterized by bluish discoloration (cyanosis) of the lips and nail beds, indicative of reduced oxygen saturation.

On internal examination, all organs were found to be congested. The right and left lungs weighed 830 grams and 630 grams, respectively. The lungs were fixed in formalin and subsequently dissected. On dissection, antemortem thrombi were observable within the pulmonary circulation, projecting slightly above the cut surface resembling toothpaste being extruded from a tube (Figure 1). Histopathological examination of lung tissue sections stained with hematoxylin and eosin demonstrated the presence of antemortem thrombi within the pulmonary blood vessels, as shown in Figures 2 and 3.

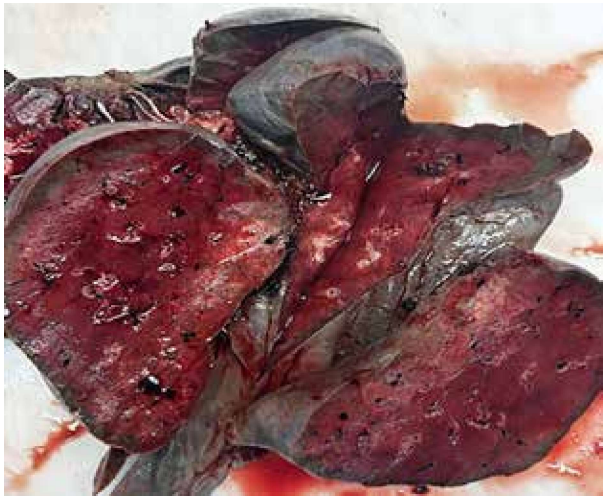

Figure 1: Gross photograph of the lungs demonstrating antemortem thrombi within the pulmonary vasculature, evident as firm, adherent intraluminal clots

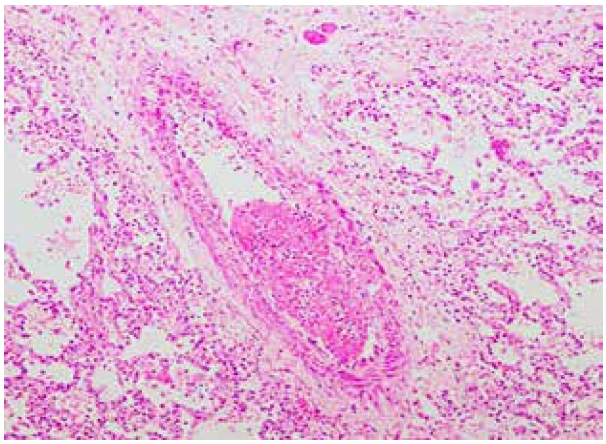

Figure 2: Photomicrograph of the lung showing antemortem thrombi within pulmonary vessels

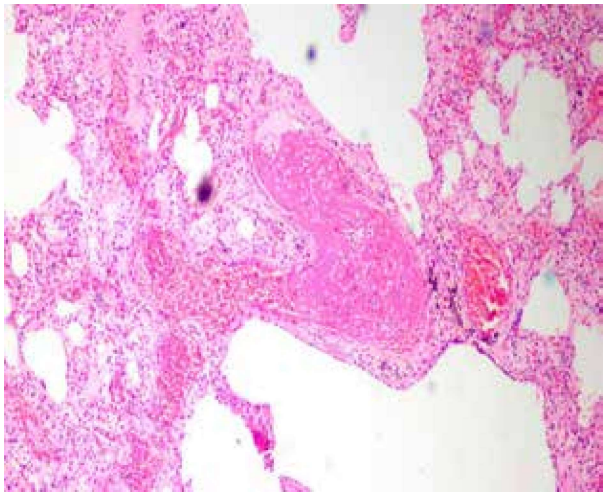

Figure 3: Photomicrograph showing a large pulmonary vessel containing a wellformed thromboembolus

Patient 2: Pulmonary thromboembolism following trauma

A 30-year-old male sustained a fall from a motorbike and experienced loss of consciousness immediately following the incident. He was promptly rushed to the emergency department of a hospital by good Samaritans on the road, where he was stabilized and treated. On initial examination, multiple abrasions were noticed over various parts of his body and appropriately cared. Subsequent radiological evaluation revealed a comminuted fracture of the shaft of the left humerus. The treating doctor explained the available treatment options, and a decision was made to proceed with surgical repair of the fractured humerus after a short period of conservative management. Surgery was scheduled for approximately 10 days after the injury. During this interim period,

he received analgesics, antibiotics, and supportive care. Eight days after the injury, the patient developed breathlessness and dyspnea. Pulmonary embolism was not clinically suspected at that time. Auscultation of cardiovascular system revealed tachycardia with an irregular heart rhythm, while the lung fields was clear and unremarkable. The patient died suddenly and unexpectedly while being shifted for further examination.

Autopsy findings: On external examination, multiple partially healed abraded wounds were present over the face, left upper limb, left knee joint and left foot. Musculoskeletal examination revealed a fracture with dislocation of the left humerus. On internal examination, both lungs showed multiple petechial hemorrhages on their surface and were markedly edematous. Dissection of the heart and great vessels revealed a firm clot within the trunk of the pulmonary artery, extending into and bifurcating within the pulmonary arteries inside the lung parenchyma. The clot was firm in consistency, firmly adherent to the lumen of the pulmonary artery, and was appreciable on macroscopic examination (Figure 4). The Histopathological examination of hematoxylin and eosin- stained sections of the clot demonstrated characteristic intertwining lines of Zahn, confirming its antemortem origin. Based on the autopsy and histopathological findings, the cause of death was opined as due to the consequences of pulmonary thrombosis resulting in the occlusion of the pulmonary arteries.

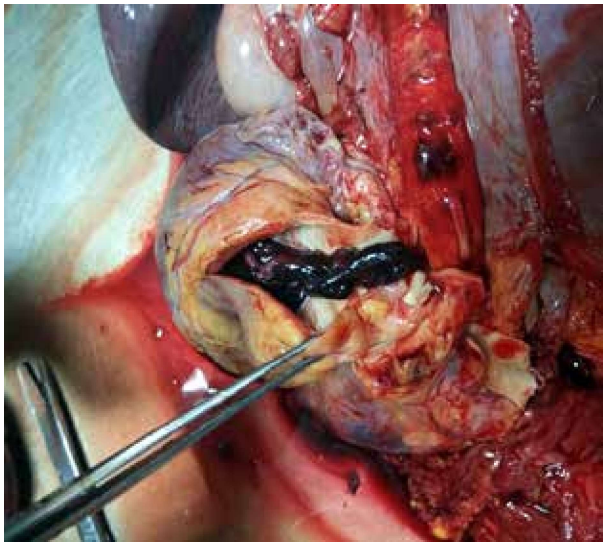

Figure 4: Dissected heart showing the right ventricle and pulmonary trunk, with a thrombus clearly visible within the pulmonary outflow tract

Patient 3: Fat embolism in the pulmonary circulation

A 35-year-old male, sustained injuries in a road traffic accident, which resulted in an open fracture of the left femur. The victim was stabilized at a tertiary care hospital, treated conservatively, and prepared for planned surgical correction of the fracture. On the fourth day following the accident, the patient developed breathlessness and dyspnea, and petechial hemorrhages were noticed on the skin. He also exhibited irritability and increased aggressiveness compared to his premorbid behavior. The planned surgery was postponed in view of the respiratory symptoms. Despite supportive management for the suspected fat embolism syndrome, his condition deteriorated, and he died on the fifth day following the road traffic accident. Antemortem diagnosis of fat embolism is difficult. Treatment at the hospital for signs of fat embolism, such as breathlessness and behavioral changes, is mainly supportive care.

Autopsy findings: Petechial hemorrhages were observed over both flanks. The weight of the right lung was 475 grams, and left lung was 410 grams. Histopathological examination of hematoxylin and eosin-stained sections of the lungs revealed fat globules and bone marrow components within the pulmonary arteries. The presence of bone marrow components (Figure 5) indicated that the fat globules originated from the marrow of the fractured femur bone.

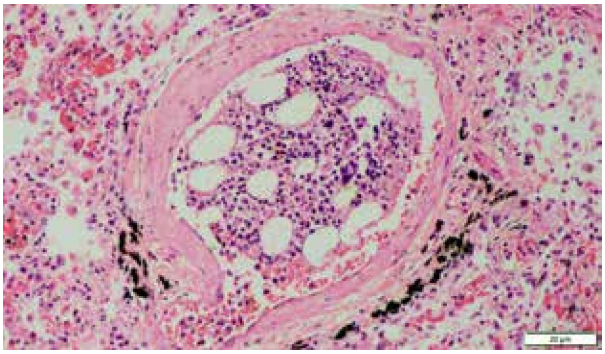

Figure 5: Photomicrograph showing adipocytes along with hematopoietic elements within the lumen of blood vessel with surrounding lung parenchyma showing septal congestion

Patient 4: Incidental findings of fat embolism in the lungs

A 38-year-old labourer, with a history of chronic alcohol use developed a headache and

was taken to a nearby hospital for treatment. He had a history of suffering from liver cirrhosis, for which he was on restricted diet. He was also receiving treatment for hypertension. He died suddenly and unexpectedly on the same day, and an autopsy was performed to determine the cause of death.

Autopsy findings: There were no external injuries on the body. Internal examination revealed a firm liver with micro- and macro nodules on its surface. The heart weighed 390 grams, and the coronaries felt gritty; horizontal section showed 60% luminal blockage. The lungs were congested. The lungs, heart, liver and kidneys were subjected for histopathological examination. Histopathological examination of hematoxylin and eosin-stained sections of the lungs revealed fat emboli, which are considered an incidental finding. Kidneys showed features suggestive of glomerulosclerosis.

Patient 5: Amniotic fluid embolism with multiple antemortem thrombi

A 35-year-old female, who had been normal throughout her routine antenatal examinations, was admitted during the latent phase of labor. Within a few hours, she progressed to active labor. During this stage, fetal bradycardia was noted. In view of this fetal distress, an emergency lower segment cesarean section (LSCS) was performed. However, during the procedure, she devoloped excessive hemorrhage and uterine atony. A hysterectomy was subsequently carried out in accordance with standard institutional guidelines to save the patient's life. A healthy baby was delivered, and the mother was transferred to the intensive care unit for postoperative recovery. Shortly after, she developed breathlessness followed by coughing spells and complained of dyspnea. Despite the best medical efforts, her condition deteriorated and progressed to arrhythmia and hypotension. The patient died one day after the surgery and delivery.

Autopsy findings: On external examination, apart from the sutures consistent with cesarean section and hysterectomy, bluish discolouration of the lips and nail beds was noted. On internal examination, approximately 100 ml of pale-yellow fluid was present in the pleural cavities. The right lung weighed 560 grams and the left lungs weighed 410 grams. Histopathological examination of hematoxylin and eosin-stained sections of the lungs (Figure 6) revealed fetal squames in the smaller pulmonary arteries. Multiple smaller arteries also showed the presence of antemortem thrombi.

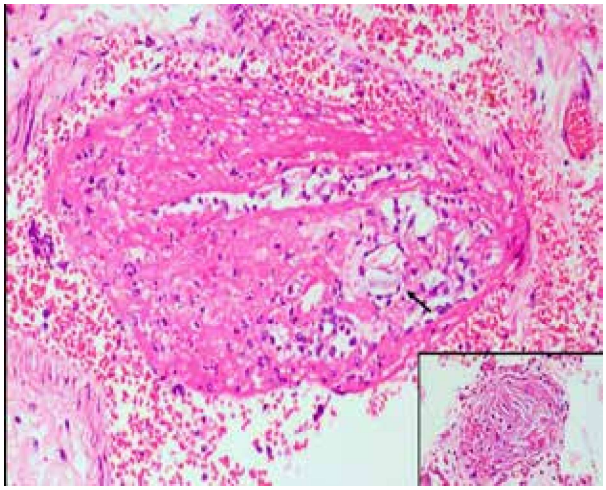

Figure 6: Photomicrograph showing a pulmonary vessel containing a thrombus composed of anucleate squamous cells (arrow). The inset demonstrates an alveolar capillary with similar anucleate squames.

Patient 6: Amniotic fluid embolism in pulmonary circulation

A 37-year-old female who had been regular with her antenatal check-ups, delivered a baby by normal vaginal delivery and died within three hours postpartum. Prior to death, she experienced dyspnea and tachycardia, for which appropriate intervention were provided according to institutional guidelines. She was being assessed for sudden breathlessness when her unexpected death occurred. An autopsy was performed to determine the cause of this sudden death.

Autopsy findings: On external examination, the features of recent vaginal delivery were evident. On internal examination, findings consistent with a recent delivery, including an enlarged uterus, were noted. Gross examination of the uterus, ovaries, and placenta, revealed no obvious cause of death. However, histopathological examination of hematoxylin and eosin-stained sections of the lungs showed intravascular squames in the blood vessels of the ecto-cervix and endo-cervix. The sections from the lungs presented anucleate squames and microthrombi.

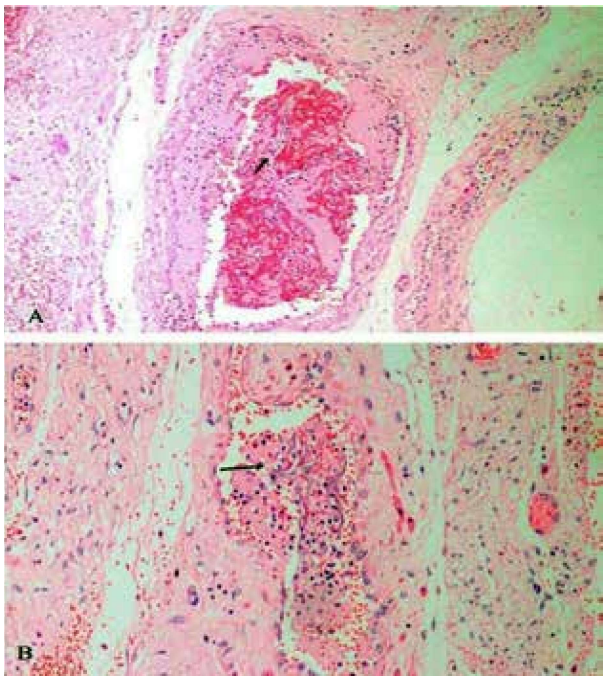

Figure 7: Photomicrograph of the uterine cervix showing dilated vessels containing microthrombi, with anucleate squamous cells present within and around the thrombotic foci

DISCUSSION

With respect to the first two cases, which involve antemortem thrombi, there is a paucity of data in the literature describing the signs and symptoms observed in hospitalized patients who die of antemortem thrombi. This lack of data may be because such patients are often intubated, comatose, or otherwise unable to communicate effectively.[6,7] Several different clinical conditions - such as myocarditis, rib fracture, myocardial infarction, pleuritis, and pericardial effusion, etc., present with signs and symptoms that are similar to the those associated with antemortem thrombi in the pulmonary circulation. Antemortem thrombi may cause death in several ways: 1) Their large size may obstruct the pulmonary artery or one of its main branches, leading to infarction of the pulmonary parenchyma distal to the blockage. 2) Antemortem thrombi may increase the back pressure on the right ventricle, resulting in cardiopulmonary arrest or rapidly progressing respiratory failure. 3) Vagal stimulation due to the mechanical effects of antemortem thrombi on the pulmonary vasculature may inhibit the cardiac function and ultimately cause death[8,9].

In the first patient, death resulted from increased back pressure on the right heart due to emboli in the branches of the pulmonary arteries. In the second case, the antemortem thrombi were found located in the pulmonary trunk and extended into both pulmonary arteries. There is lack of simple, risk-free, low-cost tests to reliably detect the presence of pulmonary thromboembolism (PTE). Therefore, treating clinicians must maintain a high index of suspicion regarding the possibility of PTE. Notably, no histopathological changes in the heart were observed in the heart suggestive of myocardial infarction or arrhythmia, which are expected in the initial few hours following infarction. Pulmonary thromboembolism was considered the cause of death based on the confirmed presence of intrapulmonary thromboemboli within the pulmonary vasculature, corroborated by treatment records indicating the absence of any other pathophysiological condition capable of causing death. From a medicolegal perspective, PTE is significant because a causal relationship must be established between any sustained trauma and the subsequent development of PTE. Additionally, the hospital practices must be evaluated, and institutional guidelines formulated when an inpatient - especially an accident victim - develops symptoms suggestive of PTE[10].

In the third case, fat embolism syndrome (FES) manifests following the entry of fat emboli into the circulatory system. These fat globules upon reaching the pulmonary capillaries, get lodged within it. Additionally, some fat emboli may also pass through pulmonary arteriovenous shunts into the arterial circulation and subsequently reach the brain and skin, leading to the neurological and cutaneous symptoms associated with FES[11,12], some of which was noticed in the case as described above. In the fourth patient, fat embolism may have originated during resuscitation efforts in a patient was known to be hypertensive with a history of alcohol addiction. Thus, the source of fat embolism is different between the two cases. Currently, there are no diagnostic methods for FES that are sufficiently sensitive or specific for use during the treatment of patients. Several diagnostic criteria have been proposed-such as Schoenfeld and Lindeque, Gurd and Wilson, etc., but none are universally accepted or clinically fully validated.[13-15]

Figure 5 shows bone marrow elements with fat emboli in the veins of a prepared slide and H&E-stained sections. Fat emboli trigger the release of lipase, which breaks down fat into glycerol and free fatty acids. These toxic metabolites damage endothelial cells and trigger an inflammatory response. This ultimately results in acute respiratory distress syndrome as observed in the reported case. The victim presented with irritability and dyspnea; however, despite the appropriate treatment provided, life could not be saved. The opinion that the cause of death was due to fat embolism is based on histopathological examination of lungs showing fat globules in the pulmonary circulation, along with symptoms and signs before death.[16-18]

For patients fifth and sixth, amniotic fluid embolism (AFE) during pregnancy also known as anaphylactoid syndrome of pregnancy, is a potentially catastrophic condition that may occur in a small number of females either during pregnancy or shortly after delivery.[19] The clinical diagnosis of AFE at the time of presentation is primarily based on the signs and symptoms observed by the treating clinician, after excluding other possible conditions with similar presentations. It is believed that a breach in the physical barriers between the maternal and fetal compartments allows amniotic fluid to enter the maternal circulation through the endocervical veins, uterine parenchymal tissue disruptions, or the placental attachment site[20-22]. This results in sudden cardiovascular collapse, manifesting as cardiac arrest, shock or severe hypotension accompanied by respiratory distress, altered mental status (such as seizure or coma), and disseminated intravascular coagulation. There are no bedside clinical tests available for the diagnosis of amniotic fluid embolism. Additionally, training staff and health care personnel to manage such emergencies remains challenging[23]. In patients five and six described above, amniotic fluid embolism was determined to be the cause of death.

In patient five, fetal squames were found in the lung parenchyma, and several and several antemortem thrombi were identified in the pulmonary circulation. These antemortem thrombi are part of disseminated intravascular coagulation (Figure 6), resulting from coagulation disorder caused by presence of amniotic fluid components in the maternal circulation. Aspirated food particles were also identified in the bronchioles. Aspiration may be the terminal event or may have occurred earlier when the patient was dyspneic and struggling to breathe. The combined presentations of aspiration and embolism highlight the need for a team of specialists from different medical streams, including obstetrics, anaesthesia, and emergency medicine, to work together assisting in reviving the dyspneic female patient.

In patient six, the uterine vessels from the ectocervix and endocervical regions also showed the presence of fetal squames. (Figure 7 A &B) Microthrombi were observed in the pulmonary circulation, similar to those in patient five. Therefore, amniotic fluid embolism is the cause of death in both patients five and six.

CONCLUSION AND RECOMMENDATIONS

Pulmonary embolism caused by the presence of thrombi, fat globules, and contents of the amniotic fluid are distinct with different etiologies. Pulmonary thrombo-embolism is largely preventable through timely risk stratification and appropriate thromboprophylaxis. Fat embolism can be significantly reduced by early stabilization and proper management of longbone fractures. Amniotic fluid embolism is neither predictable nor preventable, requires early recognition and rapid multidisciplinary intervention to improve both maternal and fetal outcomes. The nursing staff in a hospital should be sensitized regarding the signs and symptoms presented due to embolism. Maintaining a high index of suspicion, ensuring prompt diagnosis, implementing preventive strategies, and providing coordinated critical care are essential to reducing morbidity, mortality, and medicolegal implications associated with these embolic syndromes.

REFERENCES

1. Freund Y., Cohen-Aubart F., Bloom B. Acute pulmonary embolism: A review. *JAMA*. 2022; 328(13): 1336–1345. doi:10.1001/jama.2022.16815.
2. Goldhaber SZ, Bounameaux H. Pulmonary embolism and deep vein thrombosis. *Lancet*. 2012; 379(9828): 1835–1846. doi:10.1016/S0140-6736(12)60574-6.

3. International statistical classification of diseases and related health problems, 10th revision (ICD-10). Geneva: World Health Organization; 2019. Chapter IX: Diseases of the circulatory system (I00–I99). Code I26: Pulmonary embolism.
4. Lapner S.T., Kearon C. Diagnosis and management of pulmonary embolism. *BMJ*. 2013; 346: f757. pp. 1–9. doi:10.1136/bmj.f757.
5. Kakkar N., Vasishta R.K. Pulmonary embolism in medical patients: an autopsy-based study. *Clin Appl Thromb Hemost*. 2008; 14(2): 159–167. doi:10.1177/1076029607308389. Available from: http://cath.sagepub.com
6. Gong D.Y., Liu X.F., Huang F.J. Clinical feature analysis of fatal pulmonary thromboembolism: experiences from 41 autopsy-confirmed cases. *Eur Rev Med Pharmacol Sci*. 2013; 17: 701–706.
7. Christensen M.R., Larsen A.B., Boel L.W.T. Autopsy characteristics of deaths due to pulmonary thromboembolism in northern and western Denmark: a 10-year retrospective study. *Forensic Sci Med Pathol*. 2025; 21: 640–646.
8. Bergqvist D., Lindblad B. A 30-year survey of pulmonary embolism verified at autopsy: an analysis of 1274 surgical patients. *Br J Surg*. 1985; 72(2): 105–108.
9. Lucena J., Rico A., Vázquez R., Marín R., Martínez C., Salguero M., Miguel L. Pulmonary embolism and sudden–unexpected death: prospective study on 2477 forensic autopsies performed at the Institute of Legal Medicine in Seville. *J Forensic Leg Med*. 2009; 16(4): 196–201.
10. Theilade J., Winkel B.G., Holst A.G., Tfelt-Hansen J., Svendsen J.H., Haunsø S. A nationwide, retrospective analysis of symptoms, comorbidities, medical care and autopsy findings in cases of fatal pulmonary embolism in younger patients. *J Thromb Haemost*. 2010; 8(8): 1723–1729
11. Habashi N.M., Andrews P.L., Scalea T.M. Therapeutic aspects of fat embolism syndrome. *Injury*. 2006; 37 Suppl 4: S68–S73.
12. Bajuri M.Y., Johan R.R., Shukur H. Two variants of fat embolism syndrome evolving in a young patient with multiple fractures. *BMJ Case Rep*. 2013; 2013: bcr2013008631. doi:10.1136/bcr-2013-008631
13. Rudinská L.I., Delongová P., Vaculová J., Iannaccone S.F., Ihnát P. Fulminant pulmonary fat embolism – undetected silent killer. *J Forensic Leg Med*. 2025; 114: 102918.
14. Shin S.W., Goncalves B.S., Seabra L.M., Coelho H.A., Sansoni T.M., Calderan TRA, *et al*. Atypical presentation of fat embolism syndrome: A case report. *Trauma Case Rep*. 2025; 56: 101150
15. Yan X., Wu S., Zeng W., Kong J. Fat embolism syndrome caused by fracture or liposuction: a retrospective case series of nine patients. *Ann Med*. 2025; 57(1): 2447427. doi:10.1080/07853890.2024.2447427.
16. Morena D., Scopetti M., Padovano M., Turillazzi E., Fineschi V. Fat embolism: a systematic review to facilitate the development of standardised procedures in pathology. *Histopathology*. 2025; 86(6): 845–861. doi:10.1111/his.15355.
17. Chatzaraki V., Heimer J., Thali M.J., Ampanozi G., Schweitzer W. Approaching pulmonary fat embolism on postmortem computed tomography. *Int J Legal Med*. 2019; 133(6): 1879–1887
18. Qi M., Zhou H., Yi Q., Wang M., Tang Y. Pulmonary CT imaging findings in fat embolism syndrome: case series and literature review. *Clin Med (Lond)*. 2023;23(1):88–93
19. Cao X., Gui Q., Yan H., Wei Y., Chen X., Liang D., *et al*. A retrospective analysis of the incidence and risk factors for amniotic fluid embolism in cesarean deliveries. *BMC Pregnancy Childbirth*. 2025; 25: 432.
20. Society for Maternal-Fetal Medicine (SMFM), Pacheco L.D., Saade G., *et al*. Amniotic fluid embolism: diagnosis and management. *Am J Obstet Gynecol*. 2016; 215(5): B16–24.
21. Ghosh S., Kulkarni S., Nambier P., Davade T. Amniotic fluid embolism: pathophysiological insights through analysis of case series. *J Obstet Gynecol India*. 2025; 75(Suppl 1): S616–S618. doi:10.1007/s13224-024-02057-6.
22. Pranjic M., Borovac B., Juric T., Klaric M. Amniotic fluid embolism and uterine atony—two case reports and a literature review. *Wien Med Wochenschr*. 2025. doi:10.1007/s10354-025-01101-y.
23. Fong A., Chau C.T., Pan D., Ogunyemi DA. Amniotic fluid embolism: antepartum, intrapartum and demographic factors. *J Matern Fetal Neonatal Med*. 2015; 28(7): 793–8. doi:10.3109/14767058.2014.932766.