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Manual of Obstetric Critical Care

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10 CHAPTER

Nonthrombotic Pulmonary Embolism

Ahsan Ahmed, Anirban Bose

INTRODUCTION

Nonthrombotic pulmonary embolism is a rare but life-threatening condition due to embolization to the pulmonary circulation by various cells/tissues, organisms, foreign materials, or gas. The purpose of this review is to describe the different subtypes of nonthrombotic pulmonary embolism observed in obstetric patients.

AMNIOTIC FLUID EMBOLISM

Introduction: Amniotic fluid embolism is a rare but catastrophic syndrome usually occurring during labor, delivery or in the postpartum period. The first case was reported in 1926 but it was recognized widely in 1941.

Epidemiology and magnitude of the problem: The incidence of amniotic fluid embolism varies from one in 12,000 to one in 50,000. Although it is rare, it is common in the population of women who died during pregnancy. Actual incidence and mortality due to amniotic fluid embolism is confounded by many factors such as variable clinical definition, overlapping symptoms, and an absence of gold standard diagnostic tests. It is usually a diagnosis of exclusion.[1]

Implications for maternal health: This life-threatening condition is initiated by the entry of amniotic fluid, fetal cells, hair, and other debris in maternal circulation. Usual risk factors are cesarean section, instrumental delivery, cervical trauma, placenta previa and abruption, eclampsia, polyhydramnios, multiple gestation, maternal age and parity, and male fetus.[1]

Clinical presentation is not uniform, although it classically presents as sudden onset hypotension and hypoxemia, followed by coagulopathy.[2] The mechanism of hemodynamic alteration is poorly understood. The mechanical obstruction theory is losing popularity as recent evidence shows activation of systemic inflammatory response syndrome (SIRS) like inflammatory responses causing transient pulmonary and systemic vasoconstriction, leading to myocardial depression and circulatory collapse. Hypoxemia occurs due to profound shunting followed by pulmonary edema and/or lung injury such as acute respiratory distress syndrome. Amniotic fluid activates platelet factor III, factor X, complement factors as well as the coagulation cascade leading to diffuse intravascular coagulation and hemorrhage. Neurological symptoms ranging from altered mentation to tonic-clonic seizures are due to injury to the central nervous system by inflammatory mediators, hypotension, and hypoxemia.[1-3]

Management: Diagnosis of amniotic fluid embolism is largely clinical with classical

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triad of hypoxemia, hypotension, and coagulopathy along with onset during labor and delivery or within first 30 minutes of postpartum period.[2] Differential diagnoses such as pulmonary thromboembolism, air embolism, anaphylaxis, high spinal anesthesia, peripartum cardiomyopathy, myocardial infarction, septic shock, transfusion reaction, hemorrhage, eclampsia, placental abruption, and uterine rupture should also be kept in mind.[1]

The mainstay of treatment is cardiopulmonary stabilization. The initial phase consists of right ventricular failure, which should be treated with preload optimization, vasopressors, and mechanical ventilatory support with emphasis on avoidance of hypoxemia, hypercarbia, and acidosis to prevent increase in pulmonary vascular resistance. Dobutamine and milrinone are used to improve right ventricular output while sildenafil, inhaled, or intravenous prostacyclin and inhaled nitric oxide are used to reduce pulmonary vascular resistance. However, the data is not promising. Gradually, the left ventricle fails and causes further hemodynamic alteration.[1] At this stage patient may need intra- aortic balloon pump (IABP) or extracorporeal membrane oxygenation (ECMO) support but severe coagulopathy restricts its use.[2,4]

Coagulopathy and bleeding should be managed by blood component therapy as per standard guidelines. Massive blood transfusion protocol may need to be initiated.[1,3] Use of activated recombinant factor VIIa has been reported but significant adverse effects limit its use.[4]

In case of cardiac arrest, resuscitation and post-resuscitation care should be done as per advanced cardiovascular life support (ACLS) guidelines. Removal of fetal monitor during defibrillation to prevent formation of electrical arch has theoretical benefits. If an undelivered viable fetus is present, expeditious delivery by operative vaginal delivery if patient is on labor, or cesarean section otherwise, is indicated. Perimortem cesarean section is indicated if spontaneous circulation is not restored within 4 minutes of cardiac arrest. Preparation for perimortem cesarean section should be started along with cardiopulmonary resuscitation.[2]

FAT EMBOLISM

Introduction: Fat embolism was first described way back in 1861. Fat embolism is defined as the release of fat into the circulation but fat embolism syndrome is a rare clinical consequence, characterized by respiratory distress, altered mental status, and petechial rash.

Epidemiology and magnitude of the problem: Fat embolism occurs in nearly all patients with pelvic or long bone fractures undergoing endomedullary procedure or joint prosthesis placement, due to disruption of sinusoids, but fortunately, fat embolism syndrome is rarely observed.

Fat embolism is also common after blast injury without skeletal injury where the soft tissue is likely to be the source. Fat embolism can also occur in patients receiving intraosseous transfusions, bone marrow infarcts due to sickle cell anemia and prolonged steroids therapy. Acute pancreatitis, mobilization of fat in viral hepatitis in preexisting fatty liver, liquefying hematoma, liposome-embedded drugs, total parenteral nutrition are very unusual causes of fat embolism. In rare circumstances, fat embolism has been reported after normal delivery and cesarean section and in acute fatty liver of pregnancy.[4,5]

Implications for maternal health: Pathophysiology of fat embolism syndrome

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remains unclear. Two theories have been proposed, the mechanical theory and the biochemical theory. In the mechanical theory, small fat globules obstruct the pulmonary capillary causing interstitial hemorrhage and edema as well as hypoxic pulmonary vasoconstriction while large fat globules obstruct a major pulmonary artery causing major hemodynamic instability. Some fat globules enter the systemic circulation via a pulmonary capillary or any arteriovenous shunt. They may get deposited in the brain and can cause petechial hemorrhage. They are also deposited in the skin, causing petechial rash. In the biochemical theory, the release of high concentration of glycerol and toxic free fatty acids in circulation initiates the inflammatory cascade leading to end-organ damage, causing pulmonary hemorrhage and edema, acute renal injury and disseminated intravascular coagulation.[4,6]

Patients usually present with hypoxemia, altered neurological status (encephalopathy, focal neurodeficit, seizure, and coma) and petechial rash. Other associated findings are fever, anemia, thrombocytopenia, altered renal and liver function and retinopathy. In severe cases, patients may have right ventricular followed by biventricular dysfunction, acute respiratory distress syndrome, coagulopathy, and shock.[4,6]

Management: Diagnosis is usually clinical and supported by imaging and laboratory values. The Gurd and Wilson's criteria are widely used for diagnosis. One of the three major criteria (respiratory distress, petechial rash, cerebral symptoms in nonhead injury patient) and four of the eight minor criteria (tachycardia, fever, jaundice, renal changes, retinal changes, acute anemia, acute thrombocytopenia, elevated erythrocyte sedimentation rate and fat macroglobulinemia) are required to diagnose the case. Investigations such as complete blood picture, inflammatory markers, blood culture, liver, and renal function, chest radiograph, echocardiography, and magnetic resonance imaging (MRI) brain should be carried out.[4,6]

Differential diagnosis should include pulmonary thromboembolism, amniotic fluid embolism, air embolism, and acute myocardial infarction. Sepsis must also be kept in mind.

Treatment is largely supportive as fat embolism is self-limiting. Cardiopulmonary support along with usual critical care management is all that is needed. Severe cases may need ECMO support. Cardiac arrest and post-resuscitation care should be done as per ACLS protocol.[4,6]

TROPHOBLASTIC MATERIAL EMBOLISM

Introduction: Gestational trophoblastic disease is a group of lesion originating from abnormal proliferation of placental trophoblastic epithelium. It includes hydatidiform mole (partial or complete) and malignancies (invasive mole, choriocarcinoma, and placental site trophoblastic tumors). It was first described way back in 1893.

Epidemiology and magnitude of the problem: In both normal and abnormal pregnancies, a good amount of trophoblastic material enters into the maternal circulation but trophoblastic embolism is usually seen in gestational trophoblastic disease and rarely in eclampsia and placental side. The actual incidence of trophoblastic material embolism is unknown. A total of 3–25% molar evacuation patients become symptomatic.[4]

Implications for maternal health: The most common symptom is shortness of breath, which is rarely severe and self-limited.

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Trophoblastic tissue in circulation and in the lung presents in majority of the patients but severe symptoms such as circulatory collapse and cardiac arrest are rarely reported. Severe symptoms are most likely due to pulmonary edema due to accompanying hyperthyroidism, dilutional anemia, preeclampsia, fluid overload, or any combination of these.[4,7,8]

Implications for Fetal Well-being

Management: Diagnosis is usually clinical in view of background history. Radiographic features include alveolar, nodular and miliary patterns and signs of pulmonary artery occlusion.

Treatment is largely symptomatic and supportive. Oxygen therapy and diuretics are the mainstay of treatment. In severe cases, patients need ventilatory and vasopressor support. Patients with cardiac arrest need resuscitation and post-resuscitation care as per ACLS protocol. Emergency pulmonary embolectomy was also reported in a case.[4,7,8]

SEPTIC EMBOLISM

Introduction: Septic pulmonary embolism is a rare but life-threatening condition usually associated with right-sided infective endocarditis, infected deep venous thrombosis, infected vascular catheter, periodontal disease, infection elsewhere with septal defect, and immunodeficiency.

Epidemiology and magnitude of the problem: The reported incidence of infective endocarditis in pregnant women is 0.006% and septic embolism is even rarer. Early recognition of infection and use of appropriate antimicrobial therapy has reduced the incidence significantly but a little increase is seen recently due to increased number of intravenous drugs users.[9]

Implications for maternal health: Staphylococcus aureus (*S. aureus)* is the most commonly involved organism. *Staphylococcus* infection has a rising trend due to an increasing number of drug users. Fungal emboli are usually found in malignancy patients. Anaerobic organisms are commonly involved in oropharyngeal and maxillofacial infections.[4,9]

Clinical features include features of extra pulmonary infections along with cough, hemoptysis, chest pain, and shortness of breath.

Management: Investigations include complete hemogram, infection markers, blood cultures, renal and liver function tests, urine analysis, echocardiography (transesophageal is superior to transthoracic to see vegetation, abscess, and leaflet perforation), CT chest with angiography and other investigations according to the source. Chest radiograph may show patchy pneumonia, feeding vessel sign, cavities, or abscess formation.[4,9]

The mainstay of treatment is management of sepsis with appropriate antibiotics and other supportive care. Inferior vena cava filter has theoretical benefit in proximal deep venous thrombosis with sepsis but the efficacy and safety is questionable. Although surgery is indicated in some severe cases, it should be balanced with the high risk of mortality.[4,9]

GAS EMBOLISM

Introduction: Gas embolism is a well-recognized phenomenon in obstetric population. It was first reported in the obstetric population in 1850. Two types of gas embolism can occur, arterial, and venous. Among these, venous air embolism is more common and is usually iatrogenic.

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Epidemiology and magnitude of the problem: Venous air embolism is the most common embolism in obstetric population but fortunately, the amount of air entering is not significant to cause symptoms. Most commonly, air enters during cesarean section due to exteriorization of the uterus. Oral sex by insufflating large amount of air in vagina during pregnancy or puerperium can cause air embolism. Other infrequent causes are placenta previa, manual extraction of placenta, uterine rupture, criminal abortion, manipulation of central venous catheter, and hemodialysis catheter, infusion through peripheral venous cannula, and invasive and non-invasive ventilation.[10]

Implications for maternal health: Usually, the pulmonary capillaries prevent entry of air into the systemic and coronary circulations. Large accumulation of air in the pulmonary circulation can cause right ventricle outflow obstruction leading to right ventricular failure. Adhered and absorbed air bubbles can activate the inflammatory cascade leading to lung edema, microthrombi formation, and coagulopathy. Rarely, air bubble can enter the systemic circulation via an arteriovenous shunt or by overwhelming the pulmonary capillary trapping mechanism causing end organ infarcts such as cerebral or cardiac infarcts.[10,11]

Cough, dyspnea, tachypnea, chest pain are common symptoms and acute cardio-respiratory failure can occur in severe cases. Paradoxical embolism can cause neurological symptoms such as focal neurodeficit, visual disturbance, headache, seizure, and acute coronary syndrome like symptoms. Cyanosis, pallor, mottled skin, pallor of mucous membranes, pallor in tongue, air in retinal vessels, and a "mill-wheel" murmur can be found.[10,11]

Management: Diagnosis is largely done by history of the event and clinical scenario. End-tidal carbon dioxide ($EtCO_2$) monitor can facilitate early detection. Intracardiac air can be detected by precordial Doppler, transthoracic Doppler, and trans-esophageal Doppler (most sensitive). A computed tomography scan of the chest may show pulmonary edema, dilatation of pulmonary artery, and air in the pulmonary artery or right heart. An MRI brain can pick up a cerebral infarct and electrocardiogram (ECG), echocardiography, and cardiac enzymes can detect coronary insufficiency.

The first step of treatment for venous air embolism is to stop further entry of air. Semi-Fowler position with leftward tilt is advisable to prevent further air entry and promote movement of air from right ventricle to pulmonary capillary. Maintaining adequate preload, providing 100% oxygen, and vasopressor support is an absolute necessity. If the patient is under general anesthesia, Nitrous oxide should be stopped and ventilation should be adequate. Suction of the air from right ventricle by multiple orifice central venous or pulmonary artery catheter, keeping it's tip 2 cm below the junction of right ventricle and superior vena cava is recommended as it can suck out up to 50% of entrained air. Arterial air embolism is managed by flat position, hyperbaric oxygen therapy and management of cerebral and coronary infarct. Nitric oxide can be used and role of fluorocarbon is under investigation.[4,10,11]

IMPLICATIONS FOR FETAL WELL-BEING

Non-thrombotic pulmonary embolism causes cardiopulmonary issues in various degrees. The fetus can suffer from hypoxic and hypotensive insult in severe cases and prognosis is usually guarded. There are no

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consensus recommendations for the delivery of the fetus. In case of severe cardiorespiratory instability and fetal age more than 23 weeks, prompt delivery is usually indicated. If the fetal age is <23 weeks and maternal condition is not life-threatening, decision should be taken on a case-to-case basis.

TAKE HOME MESSAGES

- Nonthrombotic pulmonary embolism is a rare but life-threatening condition.
- Nonthrombotic pulmonary embolism should be suspected along with other causes, if a patient has cardiorespiratory symptoms in the peripartum period.
- There is no gold standard test for diagnosis.
- Diagnosis, in a majority of cases, is done by exclusion.
- Treatment is usually supportive, along with good critical care practice.

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This work is a unique compilation of easy-to-read articles in the field of Obstetric Critical Care. This manual is meant to be a basic reading material for the ISCCM Online Course in Obstetric Critical Care. However, it is also a good standalone resource for the latest in the field. The authors are all accomplished practitioners of their respective fields and have made a conscientious effort to provide the most current information in an easy-to-understand format. The editorial board is confident that this treatise will be a valuable asset to all practitioners who manage critical-ill obstetric patients.

About the Editors

This resourceful publication has been edited by senior practitioners of the fields of Critical Care and Obstetrics. The editors bring in more than a couple of decades of professional experience and knowledge in the Art and Science of Obstetric Critical Care. The editors have carefully selected the aspects to be covered in this compilation and have spent significant time in fine-tuning the information that has been provided in this publication.

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