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Amniotic fluid embolism (AFE) occurs when there is a breaking barrier between maternal circulation and amniotic fluid. The fetal material has been documented in the pulmonary circulation of impotomatic women. Postulated mechanisms include: Obstruction of pulmonary vessels from fetal debris Inflammatory mediators leading to vasoconstriction and bronchospasm, rather than mechanical factors, most likely due to a combination of these factors. What leads to: The development of acute pulmonary hypertension and cortex is a sudden decrease in blood flow to the left ventricles, leading to a vascular collapse of ventilation/perfusion inconsistency, leading to hypoxemia and associated complications Activating the external coagulation pathway, leading to the spread of intrasodering coagulation (ziagulation) DIC Signs and Symptoms of Hypoxemia Hypotension Coagulopathy Altered Mental Status Seizures of Fetal Fever Fever Chills Nausea Diagnosis AFE Suspected. When women are present with the aforementioned signs and symptoms during : Amniocentesis Therapeutic Abortion Abdominal Trauma in pregnant individuals of intranatal amnioinfusion Differential diagnosis of venous thromboembolism air embolism infarction of myocardial Septic shock Preeclampsia/eclampsia postpartum hemorrhage Anaphyxia that can confirm the diagnosis. It is suspected when a woman who is pregnant or in the immediate postpartum period acutely presents with shock along with severe respiratory failure. Although most of the patients present in this way, there is a subset of patients whose initial presentation can be a severe hemorrhage of the secondary DIC. Sometimes the presentation can be subtle, with anxiety, altered mental state and/or hypoxemia. It is important to eliminate other fulminant conditions such as sepsis, myocardial infarction, anaphylaxis, and transfusion reactions. Laboratories: More than diagnostic help, these tests are useful in directing resuscitation. Arterial blood gas to assess ventilation and DIC hypoxemia profile: These patients may develop DIC, as evidenced by elevated PT, PTT, low fibrinogen and elevated fibrin split products (FSP). Full blood test: In the presence of DIC, hemoglobin and hematocrit tend to fall. Patients often develop thrombocytopenia, and they usually have reactive leukocytosis. Serum Chemistry Experimental Studies have shown that some laboratory tests may be useful: Serum triptasis and histamine markers activating mast cells (high) Sialyl Tn - Fetal antigen (high) Supplement C3 and C4 (low) zinc coproporphyrin (high) Although touted to be specific to AFE, they have no established role in AFE AFE diagnostics and remain experimental tools. Radiographic findings are not diagnostic. Most of these patients show diffuse infiltrations suggestive pulmonary edema. An electrocardiogram should be obtained to look for signs of ischemia. Echocardiogram to assess the pressure of the pulmonary artery, the right ventricle and the function of the left ventricle Historically, it was believed that the amniotic fluid of debris found by aspirational blood from the dist the port of the pulmonary artery catheter was pathognomonic AFE; however, recent studies have shown that fetal elements can be found in some asymptomatic pregnant women. Given the speed with which AFE represents, it is important to be vigilant and respond quickly. The cornerstone of therapy is supportive care. Hypoxemia Of Additional Oxygen when monitoring oxygen saturation If hypoxemia with hemodynamic instability is present, etonation and mechanical ventilation are shown. AFE patients with ARDS should be placed on low tidal volume and high PEEP when monitoring plateau pressure, as recommended by the ARDS network. Hemodynamic Instability These patients should be resuscitated with fluids and vasopressors. Transthoracic or transesophageal echocardiogram may be useful in steering fluid therapy. If patients are said to have left-handed lung edema, the therapy should include a combination of dobutamine along with phenylephrine or norradrenaline. The goal is to maintain the average blood pressure of 65 mmHg. St. Cardiac Arrest Advanced Cardiac Life Support, including medications should be administered immediately. During cardiopulmonary resuscitation before childbirth, the uterus should be shifted to the left to avoid compression of the aorta and lower vein. The goal is to restore the maternal hemodynamics and deliver the fetus as soon as possible. DIC Frequent cot-grade profile monitoring, including a full blood test and levels of fibrinogen blood transfusions for hemorrhage Patients should be poured over fresh frozen plasma and cryoprecipitate to correct clotting abnormalities. Platelet transfusions for thrombocytopenic patients who are actively bleeding given the increased risk of bleeding with coexisting coagulopathy, uterine atony should be treated aggressively with standard medical and surgical methods, including uterotonic drugs, uterine embolization or reassignment and hysterectomy. Fetus Number 2/3 of patients are in childbirth when they are diagnosed. In these cases, immediate delivery of the fetus is authorized to prevent further hypoxic damage. AFE patients should be constantly monitored in the intensive care unit. Continuous monitoring of the fetus before delivery is mandatory. All patients need central venous IV access, and arterial lines. Mortality 13-95% of survivors have neurological deficits. Fetal Outcome Mortality 21-22% 29-50% Survvror Survivors Deficit. Jijo John, MD, and Gary T. Kinasewitz, MD Amniotic Fluid Embolism is an example of a theme from pocket ICU management. To see other topics, please sign in or buy a subscription. 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