Case Report

Amniotic Fluid Embolism

Dr. Attiya-tul-Waheed and Dr. Anwar Ul Haque

Amniotic Fluid Embolism is a serious complication of pregnancy. Renal involvement can produce protean manifestation which may not be recognizable unless the pathologist is aware of the history. A young lady developed postpartum anuria. The biopsy revealed amniotic fluid embolism obstructing renal vessels and some renal glomerular capillary lumina.

Key words: Amniotic Fluid Embolism, Renal Emboli, Acute Renal Failure.

Report of A Case

A 27 year old low socio-economic group, married house wife with four children presented to Emergency Room with post partum vomiting that began one day after spontaneous vaginal delivery. She had 7-8 episodes of vomiting every day. The vomitus was copious containing food particles but no blood. Two days after delivery she also developed anuria. At that time her hemoglobin was 8.7 g dl and total leukocyte count was 15,600 per cubic millimeter. Her serum urea was 190 mg/dl and creatinine was 7.5 mg dl. She was brought to Pakistan Institute of Medical Sciences after 9 days of delivery. She was given 3 units of blood and she remained anuric for another 3 days. The past history was not significant. She had no significant medical or surgical illnesses. There was no history of previous blood transfusions. The menstrual history was regular with 5/28 days cycle. She did not smoke and no history of any other addiction. On physical examination, a young lad was lying comfortably in the bed. She was well oriented in time and space. Her pulse was 130 and her blood pressure was 130/80 mm Hg. She has thrombophlebitis of left hand. No rash and jaundice were noted. She had normal vesicular breathing with no added sounds. She had a palpable un-involved uterus The rest of the physical examination was essentially normal. A clinical diagnosis of acute renal failure was made. Possibility hypovolemia due to excessive vomiting or concealed hemorrhage was considered. Pregnancy related Hemolytic Syndrome was also considered. On admission (After 9 days of delivery) her White blood cell count was 13,500. Her hemoglobin was 13.2 g dl and the platelets were 216000. The Liver Profile revealed bilirubin of 1 mg dl, ALT of 28 and Alkaline phosphatase of 381. The Fibrinogen Degradation Products were more than 40. The Urea was 83 mg dl (N: 13-43), creatinine was 8.9 (N: 0.5- 1.1), potassium was 3.6 (3.5-5.1) and calcium 8.5 (8.4-10.2) The coagulation profile revealed essentially normal APTT and PT. The catheterized sample of urine on routine examination showed a trace protein, blood ++ and numerous red blood cells while WBC 7-8 / High power field. On imaging both kidneys were normal in size, shape and contour with normal calyceal system. There was no hydronephrosis and there was no evidence of nephrolithiasis. Each pyramid was dilated. The renal parenchyma was normal in thickness and echogenecity with normal well preserved corticomedullary differentiation. The urinary bladder was empty and there was un-involved uterus with no evidence of retained products of conception.

Patient was treated on the lines of ARF and was dialyzed on alternate days. During her stay, she developed pulmonary edema twice that improved with dialysis. Even after two weeks after her admission, she remained oligo-anuric, so her renal biopsy was done. The renal biopsy revealed glomeruli containing occasional pale stained off white amniotic fluid emboli. The interstitium showed edema and there was loose fibrosis. Extravasations of RBC’s were seen. The tubular epithelium was damaged and denuded. Many tubules showed necrosis. Their lumina contained red blood cells and hemoglobin casts. The vessels were thickened and some contained non nucleated pale stained off white foreign material consistent with amniotic fluid embolus similar to seen...
in glomeruli. The final diagnosis of acute tubular necrosis along with amniotic fluid embolism was rendered. Patient was put on dialysis. She gradually started passing urine.

Discussion

Amniotic fluid surrounds the fetus during pregnancy and is produced by the fetal membranes and the fetus. The fluid has a neutral pH and increases from 50 mL at 12 weeks to 1000 mL at 38 weeks. Amniotic fluid contains fetal components such as squamous cells from the skin, mucin, vernix, lanugo hairs, platelet activating factor, prostaglandins, complement activating factors, procoagulants, and sometimes meconium.

Amniotic Fluid Embolism is a dreaded complication of pregnancy. It gives no signals and warning signs and the beautiful event of delivery culminating into a live healthy baby and very satisfied mother ends up in grave tragedy of loss of mother in most cases. The unfortunate event is sudden and drastic and may kill patient in no time despite aggressive management in good set up. The patient if survive requires intensive care and follow up most likely in Intensive Care Unit (ICU) (1). The condition is also referred to as anaphylactoid syndrome of pregnancy is a leading cause of maternal mortality.[2] Exact incidence of the condition is not known and may have been under reported. In United States of America the condition occurs in about 1 in 20,000 to 1 of 80,000 pregnancies.[3]

Maternal mortality related to this syndrome ranges from 26% to 61%; however, as few as 15% may survive without neurological impairment.[4,5] Of all affected patients, 50% die within the first hour. [6] Although 79% of neonates survive, 50% of these infants are neurologically impaired.[4]. Amniotic fluid embolism was first described in 1926 by Meyer, [7] Steiner and Lushbaugh in 1941, described autopsy findings from 8 pregnant women in whom pulmonary edema and shock developed during labor. [8]. They suggested that powerful or tetanic contractions caused an embolism of squamous cells, mucin, and/or other amorphous debris, presumably from the fetus, to lodge in the patients’ pulmonary vasculature. They termed this as “maternal pulmonary embolism by amniotic fluid” characterized by shock that developed during labor or shortly after delivery. Liban et al reported cyanosis, respiratory distress, hypotension, and coagulopathy in 14 patients with amniotic fluid embolism. The squamous cells were detected in the heart, liver, kidney, spleen, pancreas, and brain of several patients. [9]

Benson presented a new clinical definition after
summarizing 3 case reports of women who survived amniotic fluid embolism. The definition included sudden onset of

Amniotic Fluid Embolism (Anaphylactoid Syndrome of Pregnancy) seems to occur after maternal intravascular exposure to fetal tissue. This exposure routinely occurs during normal labor and delivery and can also take place after placement of an intrauterine catheter, after uterine rupture, during cesarean section, or during spontaneous or surgical abortion. Under certain conditions, when amniotic fluid and its fetal components enter the maternal circulation along with endogenous mediators (e.g., prostaglandins, leukotrienes, histamine, bradykinin, cytokines, thromboxane, complement-activating factors, and platelet activating factor). The former may physically block the microvasculature of lungs, heart, brain, kidney and other viscera. Symptoms depend upon the amount and distribution patterns of the circulation obstruction. The powerful chemical mediators may incite a striking anaphylactic reaction. Both types of reaction may be seen together or separately. The pathological maternal response results in hypoxia, homodynamic instability, and/or consumptive coagulopathy [3, 4] These 3 characteristic clinical manifestations do not occur uniformly in all patients; any component may dominate or be entirely absent. [11, 12]. No consistent markers or patterns of markers have been detected [13, 14]

Differential diagnoses for this condition includes; hemorrhagic shock, sepsis, pulmonary embolism, air embolism, eclampsia, placental abruption, uterine rupture, uterine atony, uterine inversion, myocardial infarction, cardiomyopathy, anaphylactic reaction to local anesthetic agents and Hemolytic uremic syndrome of pregnancy. [3, 14, 15, 16]

Amniotic Fluid Emboli may reach many different organs. The route from pulmonary circulation to systemic circulation may be via from veins to left heart and from there to distant organs. It is conceivable that small droplets of the amniotic fluid may coalesce and form bigger masses after reaching to the systemic arterial circulation. The amniotic fluid emboli are described in brain, kidney, adrenal glands and many other organs. It is quite possible that many cases of pregnancy related Hemolytic Uremic syndrome may be in reality manifestation of amniotic fluid embolism. Renal and skin biopsies with meticulous examination of vasculatures of these organs may furnish more insight into the entity.

References