Diagnostic, Treatment And Outcome Of Pulmonary Embolism In Delivery

D Damnjanovic, M Zamurovic

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Abstract

Pulmonary embolism is an extremely rare yet life-threathening condition which can lead to acute death of both the mother and the child. To prevent sudden maternal death, urgent treatment with cardiopulmonary resuscitation, hemodynamic stabilization and correction of haemostasis disorders is necessary, followed by urgent extraction of the fetus and timely initiation of intensive therapy. This paper presents diagnostics, anesthesiological treatment and consequences of pulmonary embolism during the expulsion phase of a delivery in epidural anesthesia and considers the differential diagnostic dilemma whether it is a case of amniotic fluid embolism or thromboembolism.

INTRODUCTION

Pulmonary embolism in delivery is a potentialy life-threathening condition which can lead to acute death of not just the mother but of the child as well (1). Maternal treatment is primarily supportive, whereas prompt delivery of the mother who has sustained cardiopulmonary arrest is critical for improved newborn outcome (2). This entity is an interdisciplinary challenge because of its presentation with sudden cardiac arrest without immediately obvious cause, the lack of specific diagnostic tests, the difficulty of establishing the diagnosis and excluding competing diagnoses, and the complex treatment required, including cardio-pulmonary resuscitation. The crucial points for successful resuscitation are prompt obstetric anesthesiologist involvement and good communication with obstetricians and midwives (3).

This paper presents diagnostics, anesthesiological treatment and consequences of pulmonary embolism during the expulsion phase of a delivery in epidural anesthesia and considers the differential diagnostic dilemma whether it is a case of amniotic fluid embolism or thromboembolism.

CASE REPORT

An expectant mother, whose condition is described in this paper, was admitted to the Clinic for delivery started by spontaneous uterine contractions. Anamnesis and previous medical history showed that gestational diabetes was verified during pregnancy while no other conditions were present before that. The patient was examined by the anesthesiologist and her current state was classified as ASA class 1. In cooperation with the obstetrician, after entering the delivery room and at a convenient cervical dilation, the epidural catether was placed and 0,25% bupivacaine in bolus dose was administered, followed by continuous analgesia through the pump (0,125% bupivacaine 4ml/h). Fentanyl was used as adjuvant. Delivery was lead actively, with synthetic oxitocin stimulation by intravenous drip. When cervical dilation was almost complete, the epidural anesthetic dose was reduced, thus causing the patient feel uterine contractions followed by labor pains. Then began the second stage of labor – fetus expulsion. Immediately during the second expulsive effort the parturient complained of nausea and the urge to vomit. She than felt a sharp pain in her chest, started to cough, cough got deeper, bloody secretion appeared from the mouth, after which the patient became cyanotic. These symptoms were followed by loss of consciousness. Arterial tension was immeasurable, puls in the large blood vessels was impalpable. Cardiac arrest occured. The baby was extracted by vacuum extractor and immediately placed under pediatric care. Apgar score was 5/6. Simultaneously with child extraction, the patient was intubated and ventillated by Ambu balloon accompanied by external cardiac massage and all measures of cardiopulmonary resuscitation. Adrenalin and amiodarone as

well as heparin in bolus dose of 5000 units IV were administered because of suspected pulmonary embolism, indicated by the clinical picture.

The placenta was manually extracted, followed by manual revision of uterine cavity, while the perineal rupture was surgically treated. CPR was continued simultaneously with the obstetric interventions in the fourth stage of labor. Since heart function was not restored regardless of urgent CPR and administered therapy, heart defibrillation was performed. Heart function was restored only after the third defibrillation cycle and stabilized after 2-3 minutes. CPR lasted a total of 20 min.

Wet rustling sounds presented in the lungs, considerable amount of discharge was aspirated through the tubus, SpO2 was maintained at 88-95%. After cardiac function was restored, the patient started to bleed vaginally, probably due to previous intravenous administration of heparine. Manual uterine cavity revision was performed again, accompanied by therapy with uterotonic and protamine sulfate as antidote for heparine after which uterine bleeding ceased.

Another problem which appeared after a profuse uterine bleeding in the fourth stage of labor, caused by intravenous administration of heparine, was massive transfusion and its consequences, as well as disseminated intravascular coagulation (DIC-PTT>160,PT 30,3,INR 2,37,Fibr.2,52,D-dimer>360mg/l.). Thus, the patient`s condition was very complex and required close cooperation between anesthesiologist, internist, cardiologist, pulmonologist, neurologist and hematologist, i.e., an interdisciplinary approach in order to devise the best therapy for the current state. ROTEM test of thrombocyte function showed coagulation factor deficit. Visible effect of anticoagulation by heparin as well as presence of noneffective hemostasis with high risk of hemorrhage was present as well as apparent fibrin polimerization disorder and unstable blood clot formation. Necessary therapy was administered (tranexamic acid, cryoprecipitates, fresh frozen plasma).

Mechanical ventilation mode IPPV was continued in the ICU. Arterial pressure was maintained with dopamine stimulation, and the pulse was stabilized at approximately 100 bpm. Oxygen saturation was kept at 88-95%. Heart function was rhythmical, pupils medium dilated, centered, slow left accomodation. Lividity present in the upper body after reanimation was slowly disappearing. Vital parameters were stable, diuresis was restored.

The neurologist ascertained that the bulbi were medially located, pupils 6mm in size, slightly reacting to light, with bilateral absence of conjugated moves during cephalovestibular reflex. Atonia of the left arm and both lower extremities was present. Soles were not responding, on both sides. GSK 3/15. It was concluded that patient state corresponded to hypoxic-ischemic encephalopathy that was a consequence of cardiac arrest due to acute pulomary embolism.

Differential diagnostic dilemma appeared whether this was a case of amniotic fluid embolism or thromboembolism, cerebrovascular insult, cardiomiopathy, miocardial infarction. Considering the acute condition, administered therapy (especially heparin in bolus dose), postpartal uterine bleeding requiring urgent compensation of blood, derivatives, plasma expanders and liquids, poor lab results including coagulation factor disorder, as well as the inability to perform additional and invasive diagnostics, open question remained whether this was a case of amniotic fluid embolism or thromboembolism even though all clinical parameters indicated that this was indeed amniotic fluid embolism.

After administered therapy the patient`s condition stabilized and first signs of improvement were visible. This is when endocranial MR examination was performed, verifying subacute ischemic lesions in temporal, frontal and parietal lobe and to a lesser extent in the occipital lobe with lacunar infarctions in the right part of the corpus callosum. MRA of the head shows there were no pathological lesions in magistral blood vessels of the brain, aneurisms, AV malformations or significant stenoses. Treatment was continued under the supervision of a neurologist. Tracheotomy was performed. Signs of improvement were quickly visible, the patient was conscious but the degree of orientation was impossible to assess because of the global aphasia. The patient was afebrile, eupneic, and skin and visible mucous membranes were normal in color. The bulbi were in primary position, medially located, movable in all directions. The pupils react to light and accomodation. Central facial palsy was present on the right. GKS 13. The patient was breathing spontaneously through the tracheostomy tube and achieved satisfactory saturation; breathing was bronchovesicular with present wet murmurs of a more basal nature. Neurological findings pointed to a rightside spastic hemiparesis of the arm, a light hemiparesis of the right leg and positive Babinski sign on the right. Aphasia was globally present in speech and the patient

pronounced word by word and partially understood imparted orders without verbal comunication. The patient was included in a physio and work therapy. Speech treatment was also conducted. The condition continued to improve and after thirty days of speech and physical therapy the patient was independently mobile, the speech skills improved and clinically corresponded to expressive aphasia syndrom type 2 (Broca's aphasia). Further speech therapy was proposed.

The patient recovered with cerebral hypoxia sequels. As a consequence, hemiparesis of the right arm and global aphasia remained. Rehab accompanied by speech therapy was continued and the patient responded well so that now just mild, almost imperceptible, hemiparesis of the right arm remained, while auditive comprehension skills significantly improved.

DISCUSSION

Acute pulmonary symptomatology with clinical symptoms of pulmonary embolism in delivery is a very difficult problem both from diagnostic and therapeutic point of view and its outcome is uncertain. Differential diagnosis can include thromboembolism, amniotic fluid embolism, cardiomyopathy with acute pulmonary edema, myocardial infarction, arrhythmia, acute cerebrovascular hemorrhage etc. Rapid development of the clinical picture and condition that may have a fatal outcome both for the mother and the fetus in matters of minutes and requires urgent treatment without definite diagnostics about the condition. According to data from references, AFE can be diagnosed with high serum sialyl-Tn antigen and zinc-coproporphyrin (4, 5). Serum-based diagnostics of AFE, however, is possible only in big centers, i.e., in cases of lethal outcome, during autopsy. Acute diagnostics of pulmonary thromboembolism is also not possible in the given situation, since saving patient and her baby is much more important than precise diagnostics. Anticoagulant therapy by heparin in bolus dose, administered intravenously, can be useful in saving mother`s life but can have adverse effects such as uterine bleeding that can also potentially endanger the mother`s life. Hemorrhaging caused by intravenous administration of heparin sometimes requires massive transfusion making already hard and challenging ICU treatment even more difficult.

Multidisciplinary treatment must be led by both obstetrician and anesthesiologist. Delivery must end as soon as possible either vaginally or by urgent Caesarean section (depending on labor stage, i.e., cervical dilation and obstetrician`s assessment). Anesthetic treatment with CPR measures and intensive therapy must be performed simultaneously with obstetrical treatment and delivery of the child.

Adverse effects of pulmonary embolism in delivery depend on the length of hypoxia in the tissues, especially in the CNS as well as on the ability of the tissue to recover from hypoxia, i.e. depend on the speed of recognition of the acute condition and urgent initiation of CPR, tissue oxygenation and intensive therapy. Consequently, the ability to manage obstetric emergencies is especially important for obstetric anesthesiologists who must be correspondingly trained regardless of the fact that such urgent conditions seldom occur in delivery.

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Author Information

Dijana Damnjanovic, MD, Specialist for Anestesiology
University Clinic for Obstetrics and Gynecology “Narodni front”
Belgrade, Serbia

Milena Zamurovic, Pd (PhD) Specialist for Obstetrics and Gynecology
University Clinic for Obstetrics and Gynecology “Narodni front”
Belgrade, Serbia
mzamurovic@gmail.com