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Review Article

The Management of Amniotic Fluid Embolism

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Abstract

Amniotic Fluid Embolism (AFE) as a devastating complication in obstetrics remains one leading causes of maternal mortality in developed country. During the process of resuscitation, there are some vital deficiencies which mainly involve neglect of high risk factors, delayed recognition of early signs and symptoms, poor knowledge about emergency resuscitation, invalid of management of heart failure, coagulopathy and multi-organ failure. This mini review mainly presented correlative high risk factors, early recognition initial signs or symptoms, and effective therapeutic measures including medicine selection of vasopressors and inotropes, application of Mechanical Circulatory Support (MCS), management of consumptive coagulation, and maintenance of homeostasis.

Keywords: Amniotic fluid embolism; Systemic inflammatory reaction syndrome (SIRS); Initial signs and symptoms; Cardiac arrest; Consumptive coagulopathy

Introduction

Amniotic Fluid Embolism (AFE) as a devastating complication in obstetrics remains one leading causes of maternal mortality. Because of AFE rarity, its reported incidence varies widely with rates varying from 1.9 per 100,000 to 6.1 per 100,000, maternal mortality rate varies from 15-55%, mainly because of absence of uniformed AFE diagnostic criteria [1-4]. Centers for Disease Control and Prevention report, AFE caused 5.5% of maternal deaths in the United States from 2011 to 2013 due to an earlier diagnosis combined with prompt initiation of supportive care measures [5]. National Perinatal Epidemiology Unit of United Kingdom reported that the estimated incidence of AFE ranged from 0.8-1.8 per 100,000 maternities, and the proportion of women with AFE who died or had permanent neurological injury ranged from 30%-41% [6].

Although many rigorous researches in past two decades have greatly improved our understanding of this syndrome, its etiology and pathophysiology remain uncertain. Systemic Inflammatory Reaction Syndrome (SIRS) and Anaphylactoid Syndrome of Pregnancy (ASP) as the primary two theories tend to be considered extensively [7-9]. However, either SIRS or ASP, all suggested that maternal-fetal barrier disruption in parturition or cesarean section promote fetal antigen entrance into maternal circulation, thereafter, result in activation of humoral and cellular immunity, release massive inflammatory mediators that produce clinical manifestation of cardiopulmonary collapse and consumptive coagulopathy, it is called "immunologic storm" or "waterfall type inflammatory response syndrome" [10]. The aim of this review is to present relative risk factors associated with AFE, initial signs and symptoms that manifest in the early stage of AFE, valuable and useful management measures.

High Risk Factors of AFE

Regarding to risk factors for AFE, many systematic reviews and retrospective studies reveals a wide range of inconsistency. Advanced maternal age, placenta previa and abruption, and ethnic minority status have been found to be significantly associated with amniotic fluid embolism [1,7,11]. Labor induction methods including oxytocin and prostaglandin, and parturition mode association with AFE were of conflicting conclusion, other factors such as parity, instrumental delivery, cervical trauma and male fetus whether or not associated with AFE were not well understood [12-14]. During stage of maternal age from 28 to 36, AFE incidence gradually increase, and the morbidity of AFE is highest during maternal age stage of 34 to 36. Although multipara morbidity of AFE is higher than primigravida, this tendency coordinate with the rate of mutipara in women population, consequently, mutipara should not considered as a risk factor for AFE. The vast majority of AFE occurs in the third trimester of pregnancy, and very few cases happened in the process of evacuation or amniocentesis, and present a kind of phenomenon that the smaller the gestational age, the milder the symptoms, consequently the higher success rate of treatment. Labor induction and cesarean delivery as significant risk factors for AFE had been proposed by previous study based on the Nationwide Inpatient Sample (NIS) and other population-based studies from Canada, Australia, the UK, and the Netherlands [15-18]. Clark [8] consider that there is no causative link between uterine stimulation and amniotic fluid embolism, as well as conclusions of the American College of Obstetricians and Gynecologists. Fitzpatrick et al. [19] reported six times the odds of AFE in women who had labor induced with prostaglandin, and more than double the odds of AFE among women who received oxytocin in labor or had both oxytocin and prostaglandin. Ugo Indraccolo et al. [14] consider oxytocin augmentation of labor and labor induction could be at least inter-correlated with AFE, and suggested that oxytocin use in labor associates with death if AFE onsets. In our point of view, due to the increased frequency of labor induction and the rarity of AFE, the correlation between labor induction involving oxytocin, misoprostone, prostaglandin and AFE onset require a larger availability of data and more extensive epidemic investigations. Knight et al. [1] suggested cesarean delivery was associated with postnatal amniotic fluid embolism based on population-based cohort study and nested case-control analysis. Kramer et al. [15] based on the US Nationwide Inpatient Sample from 1999 to 2008, proposed

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conclusion that increased risks of AFE associated with cesarean delivery have implications for elective use of these interventions. Based on many systematic review [16-21], cesarean section and operation delivery not only were not associated with the AFE onset, but also as a vital emergency intervention to treat AFE, however, clinician should rationally apply these interventions.

Pregnancy complications significantly increased the risk of AFE onset. Placenta previa and abruption have been found to be significantly associated with AFE because of increased circulation exchange between maternal and fetal compartments [15]. Based on current pathophysiologic mechanism of AFE, these risk factors either promote inflammatory cytokines production or fetal compartment antigenic challenge to enhance endogenous mediators release, consequently induce SIRS or "immunologic storm".

Clinical Manifestation and Early Recognition

The typically AFE clinically manifest sudden onset of a triad of hypotension, hypoxia, rapidly ensued cardiovascular pulmonary collapse, coagulopathy usually presents in concurrent or afterward of cardiopulmonary collapse. In fact, the initial signs and symptoms of vast majority of AFE are not uniform between cases. Partial patients firstly manifest respiratory symptoms such as dyspnea, shortness of breath, dry cough, respiratory arrest, pulmonary edema, Acute Respiratory Distress Syndrome (ARDS); a portion of patients forepart initially neurological signs including loss of consciousness, confusion, seizures or convulsion and restlessness; prime cardiovascular system mainly manifest as sudden profound hypotension, tachycardia, cardiac dysrhythmia (ventricular fibrillation, asystole, and pulseless electrical activity) and lethal cardiac arrest. Obstetric presentation primarily manifest as fetal distress which showed in electronic fetal monitoring with late decelerations, prolonged decelerations, loss of variability and terminal bradycardia. 83% of patients presented with consumptive coagulopathy or DIC, this kind of symptom usually as an accompanied or secondary symptom manifested as postpartum hemorrhage or oozing blood from wounds and puncture sites, vaginal bleeding, and hematuria [8,11]. Clark et al. [11] reported 70% of cases of AFE occur during labor, 11% after a vaginal delivery, and 19% during a cesarean delivery, rare cases of AFE happened during the process of evacuation, amniocentesis and trauma.

Because manifestations of AFE is of variety and overlaps with other complication such as pulmonary embolism, peripartum cardiomyopathy, epilepsia and postpartum hemorrhage. Fetal distress is the most common first sign, fetal heart rate abnormality resulting from hypoperfusion uncommonly accompany or even precede recognizable maternal signs and symptoms of AFE. Therefore, clinician should consider the possibility of onset AFE when encounters acute fetal distress with being difficult to correct. The second common signs are neurology system signs of loss of consciousness, seizure or convulsion, usually occur during the labor or operation, sometimes, in the state of surgical anesthesia, Bispectral Index reflecting electroencephalogram activity presents sudden lowering to less than 30, even to undetected level, these phenomenons all reflect the acute hypoxia of brain tissue [22]. Hypotension is primary manifestation of hemodynamic abnormality, and recurrent refractory hypotension commonly is an omen of cardiac collapse, and tachycardia or cardiac dysrhythmia frequently is a vanguard of cardiac arrest, which hint the probable necessity of cardiopulmonary support device except vasoactive agent. AFE as an obstetric fatal emergency with characteristic of absence of premonitory and warning presentations, it is vital for clinician being able to quickly and to accurately recognize initial signs and symptoms of AFE. Therefore, requiring clinician familiarize with the common first signs and symptoms, which will be very helpful for further management of AFE.

Management

Although its etiology and pathophysiology is not fully understood and its treatment still is a huge challenge for health providers involving emergency cardiopulmonary resuscitation, dealing with consumptive coagulopathy, and supportive therapy of ensuing multiple organ dysfunction. However, its treatment make a great progress not only due to many successful saved patients and failure lesson, but also rapid development of critical medicine and presented new theory such as ASP and SIRS. By systematically reviewed AFE case reports published, we eager to screen out some valuable and useful therapeutic strategy. Therefore, evaluating and providing a series of useful and valid treatment measures will be helpful for practice of obstetrician and midwife.

Resuscitation Management

Facing with the sudden occurrence of cardiopulmonary collapse for caregivers, Standard Basic Life Support and Advanced Cardiac Life Support algorithms should be applied according to American Heart Association (AHA) scientific statement of Cardiac Arrest in Pregnancy [23]. These emergent resuscitation should be of crucial role for ameliorating outcomes and prognosis of AFE women. Several researches had pointed out knowledge deficits and poor resuscitation skills could be major causes of poor outcomes once cardiac arrest has occurred. Appropriate training and preparation for such events will be the valid measures for reducing AFE induced mortality despite its rarity.

At the same time of resuscitation, laboratory investigations should be repeated frequently in the first few hours, as coagulopathy may develop in the initial phase of AFE and acidaemia will aggravate pulmonary vasospasm and pulmonary hypertension, and injured multiple organs function. Laboratory investigations should include complete blood count with platelets, blood type and antibody screening, serum electrolytes, arterial blood gas, cardiac enzymes, coagulation profile (prothrombin time, partial thromboplastin time, international normalized ratio, and fibrinogen), D-dimer, liver and kidney function test, except for these blood biochemical tests, other auxiliary diagnosis and differential diagnosis measures should also been applied in women with AFE, involving 12-lead electrocardiogram, echocardiogram, chest radiograph, etc.

Hypotension and Hypoxia Treatment

Hypotension and hypoxia in the initial stage of AFE should be treated by a series strategies for maintaining cardiopulmonary stabilization and vascular perfusion. Blood pressure should be supported with fluids and pressor agents, it is optimal of norepinephrine or vasopressin administrated by deep venous catheter, and different from other type hypotension, the hypotension presentation of AFE originate in acute right ventricle failure with

preload-dependent, so that, during the process of fluid resuscitation, should avoid excess fluid administration, because it will aggregate the overdistention of the right ventricle and may increase the risk of right myocardial infarction and later pulmonary edema [7,11,24]. Pulmonary artery hypertension is the initial basic pathophysiological changes of AFE and induce hypoxia, therefore, relieving pulmonary artery hypertension is the first treatment of hypoxia, sildenafil, inhaled or intravenous prostacyclin, and inhaled nitric oxide were recommended for pulmonary hypertension induced acute right heart failure by Europe Heart Failure Association and Europe Heart Disease Association [11,25]. Another important measure is to ensure adequate ventilation and oxygenation, endotracheal intubation and delivering 100% oxygen are necessary for correcting hypoxia and hypercapnia which could promote pulmonary vasoconstriction and further increase the afterload of the right ventricle, and oxygen therapy should be used to keep an arterial oxygen saturation more than 90% [24, 25]. Diuretics should not been as an routine application except woman with signs of venous and systemic congestion.

Echocardiography Assessment of AFE

Pulmonary artery hypertension induced acute right ventricle failure, ensued left ventricle failure and globe ventricle failure, which is the typical pathophysiological mechanism of cardiopulmonary collapse in AFE. The common characteristic of echocardiography included acute right atrium dilatation, interauricular septum left shift, following right ventricle dilatation and left shift of interventricular septum, tricuspid regurgitation. With pericardial constraint and septum shift, left ventricle became volume reduced, hypokinesia, cardiac ejection fraction decreased. These changes may contribute to low cardiac output state by decreasing left ventricular distensibility, preload, and ventricular elastance. Most part of cases presented increased pulmonary arterial pressure, one case showed transient right atrial embolus [26,27]. Therefore, echocardiography plays an extremely crucial role in the assessment of heart size, function and load during the AFE emergency management [25,28]. Firstly, it can be used to exclude other causes induced acute heart failure, such as pericardial tamponade, pulmonary artery embolus and pulmonary embolism by ultrasound evaluation of the lower extremity veins; secondly, echocardiography could provide all available views of globe ventricle changes from different ultrosound section including apical four-chamber right ventricle focused and subcostal views, accurately estimate right ventricle enlargement, sphericity, and degree of right ventricle dilation compared with left ventricular dilation. Thirdly, latest advances in echocardiography could quantitatively evaluate global and segmental right ventricle function by measuring following parameters: fractional area changes, tricuspid annular plane systolic excursion, systolic S velocity of the tricuspid annulus, right venticle index of myocardial performance, regional longitudinal shortening, and three-dimensional right ventricle volumes and ejection faction. All these parameters will be valuable for clinician to diagnosis AFE, to evaluate therapeutic effect and decide further treatment strategy [28,29]. When women do not respond to initial management, invasive monitoring with pulmonary artery catheter may be required [25,30].

Acute Heart Failure Therapy

Pharmacotherapy

Refractory cardiac arrest, mutiple organ failure and consumptive coagulopathy were the common fatal causes of AFE. Acute cardiac arrest is the main lethal cause of AFE, therefore, it will be the most important role in resuscitation for taking timely and effective measures to cardiac arrest. Cases successful revived women should be due to prompt resuscitation and validly application of vasopressors and inotropes. According to the European Society of Cardiology (ESC) guideline about acute heart failure therapeutic strategy, vasopressors and inotropes are indicated to support the failing heart with hemodynamic instability [31]. Noradrenalin are primarily indicated to restore blood pressure and improve cerebral, coronary, and other organ perfusion, and improve systemic hemodynamics by improvement of ventricular systolic interaction and coronary perfusion without change in pulmonary vascular resistance. Dobutamine and phosphodiesterase III inhibitors such as milrinone, levosimendan could improve contractility and increase cardiac output. Phosphodiesterase III inhibitors exert a positive inotrope effect on the right ventricle without the deleterious effects on pulmonary vascular resistance that occur with catecholamines [24,25,31].

Mechanical circulatory support

In some cardiac arrest patients, although succeed in emergency cardio-pulmonary resuscitation and administrated pharmacotherapy including noradrenalin, dobutamine, and milrinone, patients persisted in hemodynamic instability manifested recurrent cardiac arrest, refractory hypotension with critical organ hypoperfusion, globe heart hypokinetic state, mechanical circulatory support should be considered. Most cases manifested acute heart failures, who survived with the support of mechanical circulatory device including AV-ECMO, IABP and Impella CP. Confronting AFE progressive clinical manifestations, acutely decompensated heart failure, and acute myocardial damage, the time to make a decision about MCS device implantation may be extended. Recent years, MCS device application in refractory cardiac arrest tend to increase in popularity, it can provide a great opportunity to enhance patient prognosis especially decreasing neurological deficiency complications and secondary multiple organs dysfunction in AFE [32-34]. Some researches presented that massive vasoactive agent and inotrope medicine application in cardiac arrest would induce poor clinical prognosis [31,32,35]. So that, MCS devices can be proposed very early in the management of acute heart failure, to replace inotrope therapy, and because of rapidly deteriorating hemodynamic state of acute heart failure, most MCS devices are also used to complement the usefulness or insufficiency of inotrope therapy [31,36]. In China extracorporeal membrane oxygenation expert consensus propose early application ECMO for refractory cardiac shock with no contraindications, suggest that MCS device should be provided for patient with no recovery effective autonomous circulation after 10 minutes persistent valid Cardiac Pulmonary Resuscitation (CPR) [37]. To optimize management of patients with cardiac arrest, a multidisciplinary discussion before implantation is required in most cases. According to AHA expert consensus, the choice of MCS must be based on the aetiology of cardiogenic shock, the patient's hemodynamic and respiratory status and an estimate of the "time to support" [24]. In cases of AFE pathophysiology trait of acute right ventricular failure, and ensuing biventricular failure, multiorgan failure or associated respiratory failure, ECMO is preferred. In contrast to the other devices, ECMO provides complete cardiopulmonary support, leaves time for myocardial recovery, and prevents irreversible mutiple

organs failure [25,38,39].

Management of consumptive coagulation

Consumptive coagulopathy or DIC usually occurs simultaneously or rightly after cardiac arrest, manifest as hyperfibrinolysis, massive consumption of coagulation factors, sometimes with microthrombosis of tissue organ microcirculation, mucous membrane bleeding or wound oozing, and uterus hemorrage. Its therapeutic strategy includes supplement mutiple coagulation factors by transfusion massive fresh frozen plasma, cryoprecipitate, and fibrinogen concentration. Early initiate a Massive Blood Transfusion Protocol (MTP) play a critical role in this process. rVIIa usage in AFE achieved bleeding decrease and reduced overall blood component transfusions, and no report of organ thrombosis. rVIIa can combine with circulating tissue factor to enhance the formation of intravascular clots, some of rVIIa complications of survivors such as deep venous thrombosis, acute renal failure, and organ thrombosis limit rVIIa as a common treatment. Because of AFE consumptive coagulpathy progressively development, most of experts recommend that the initial therapy of AFE associated consumptive coagulopathy should consist of blood component replacement, and rVIIa should be used in AFE patients when the hemorrage cannot be stopped by massive blood component replacement [7,40]. Early usage of tranexamic acid will be helpful for improving hyperfibrinolysis. The usage of C1 esterase inhibitors (C1-INH) in early stage of AFE, presented very good therapeutic effect. C1-INH has effect of regulating immunological reactions including the complement and kinin-kallikrein systems, and inhibits coagulation factor a, a, bradykinin releasing enzyme, and plasmin [41-43]. Obstetrical intervention Bakri balloon compression or uterine packing compression were initially used hemostatic measures, B-Lynch suture may be of worse effect, if these measures failed, vascular ligation, uterine arteries embolism, even hysterectomy will be effective measures to cease bleeding. In the state of systemic DIC, when hysterectomy still cannot stop bleeding, pelvic packing should be considered. In all, actively correct systemic coagulopathy is of critical role in management of hemorrage at the time of performing obstetrical intervention.

Maintenance of homeostasis

After initial cardiac pulmonary resuscitation, AFE patients multiple organs system experience hypoperfusion, hypoxia and microthrobosis formation of microcirculation attack, ensue presenting metabolic disorder of water electrolysis, maintenance of homeostasis will be crucial for preventing multiorgan failure and irreversible organ function damage. Review successful resuscitation cases, the hemodialysis in CHDF mode and plasmapheresis were timely applied during initial resuscitation golden 24-48 h. The primary measures include clearance of massive inflammatory mediators, correction of metabolic disorder of water electrolysis and acidosis, and ensuring full oxygenation of tissues and organs, hemodialysis is the strong effective treatment for accomplishing these targets [44,45]. Persistent massive inflammatory factors releasing and immune complex formation are the vital pathophysiological process of AFE, therefore hemodialysis usually take the mode of CHDF, and continuous CHDF is more preferable than intermittent mode in the period of initial 72h. Meanwhile, hemodialysis can clear massive water in body, regulate and maintain electrolysis, except that, plasmapheresis with hemodialysis device is the key measure to treat refractory consumptive coagulopathy which cannot be corrected by supplementing massive coagulation factors and MTP. The delay application of hemodialysis or plasmapheresis would induce irreversible organ damage, ultimately presenting multiorgan failure. Blood gas and lactate content reflect homeostasis disorder state, and may be as crucial criteria for decision of initiating hemodialysis.

Conclusion

From the first report of amniotic fluid embolism by Mayer in 1926, there are a lot of case reports of amniotic fluid embolism rather than systematic clinical research. Although its etiology and pathophysiology is not fully understood, its treatment make a great progress mainly acquired not only from many successful saved patients and failure lessons, but also rapid development of critical medicine. Increased awareness and proper training for AFE will hopefully result in reduced fatality rate and improved maternal outcomes for this rare and devastating complication. Clinicians in practice should pay more attention to correlated high risk factors of AFE, to familiarize with initial signs or symptoms in order to early recognize and management. Multidisciplinary cooperation play a key role in successful resuscitation of cardiopulmonary collapse. In future, the theory "immunologic storm" or "waterfall type inflammatory response syndrome" of AFE needs more basic researches to prove. Collaborative interstate and international programs will provide more powerful management of AFE.

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