The use of therapeutic hypothermia in the management of amniotic fluid embolism

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Summary

Background: Amniotic fluid embolism (AFE) is a rare peripartum obstetric emergency where patients seldom survive neurologically intact. The exact pathophysiology is not completely understood and treatment remains mainly supportive.

Case: A 34-year-old African American woman gravida 1, para 0-0-0-0 at 38 weeks and four days induced for chronic hypertension with superimposed preeclampsia experienced an AFE during labour. Supportive treatment included early use of therapeutic hypothermia resulting in a normal neurological outcome.

Conclusion: This case demonstrates the timely use of therapeutic hypothermia in a patient surviving an AFE and suffering no neurological sequelae. Therapeutic hypothermia should be considered in the supportive treatment of AFE.

Keywords: high-risk pregnancy, hypertension, intensive care medicine, maternal–fetal medicine, maternal mortality

INTRODUCTION

Amniotic fluid embolism (AFE) is a rare obstetric emergency. Approximately 15% of patients survive neurologically intact according to the national registry¹ as opposed to findings of 6% with persistent neurological impairment in the UK national registry mentioned in one meta-analysis.² Furthermore, 10% or less of women suffering an AFE and cardiac arrest survive neurologically intact.³ In AFE, fetal cells enter the maternal circulation which is then associated with cardiopulmonary distress. The exact cascade of events leading to this outcome is still unknown, although evidence suggests a systemic inflammatory response syndrome associated with the inappropriate release of endogenous inflammatory mediators.³ This often leads to a significant risk of fetal and maternal morbidity and mortality. Incidence has been reported to be anywhere from 7.7 per 100,000 deliveries⁴ to an incidence of 2.0 per 100,000 deliveries in the UK.⁵

Due to the devastating sequelae of AFE, it is important to have early recognition and timely implementation of supportive treatment. Since AFE is a rare event, much of the literature on management is based on case reports. This case report demonstrates the successful use of therapeutic hypothermia in the setting of AFE where the patient survived neurologically intact.

CASE REPORT

A 34-year-old African American woman gravid 1, para 0-0-0-0 at 38 weeks and four days was admitted to labour and delivery

for induction of labour secondary to chronic hypertension with superimposed preeclampsia at term. Her hypertension was controlled with oral labetalol. She underwent cervical ripening with misoprostol 25 µg per vagina every four hours for a total of three doses. Intravenous magnesium for seizure prophylaxis was not initiated at this time. Early morning of hospital day 2, she had spontaneous rupture of membranes. Four minutes later the patient began to have shortness of breath with a pulse oximetry of 78%. She was tachypnoeic with bilateral coarse breath sounds noted on exam. Given that the patient had no known cardiopulmonary disease other than hypertension and the timing of her symptoms in relation to spontaneous rupture of membranes, she was suspected to have an AFE. She underwent emergency primary caesarean section, during which she suffered a cardiac arrest. A live healthy infant was delivered with Apgar scores of 3, 7 and 9, at 1, 5 and 10 minutes, respectively. During maternal resuscitation, copious frothy secretions were noted from the endotracheal tube. Cardiopulmonary resuscitation was successful 43 minutes after efforts were initiated.

The patient was stabilized and transferred to the medical intensive care unit, where she remained intubated. Therapeutic hypothermia via a cooling blanket was initiated (target temperature 32–34°F for 24 hours) to optimize her neurological outcome. Transoesophageal echocardiogram was performed which showed a left ventricular ejection fraction of 10–15%, and on postoperative day 1 an ejection fraction of 5–10% with diffuse hypokinesia thought to be secondary to both peripartum and ischaemic cardiomyopathy. On postoperative day 2, re-warming began over the course of 10–12 hours. She was successfully extubated on postoperative day 3 and discovered to be neurologically intact.

On postoperative day 5 she underwent surgery for a bivalve implantable cardioverter defibrillator, improving her ejection fraction to 20–25%. On postoperative day 7, the patient was

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discharged home. Two years after suffering an AFE our patient has no neurological deficits.

COMMENT

AFE remains one of the most devastating obstetric emergencies due to its significant morbidity and mortality. This is the first report using therapeutic hypothermia in a patient suspected to have suffered an AFE.

Our patient had risk factors for AFE including preeclampsia,⁴ induction of labour and non-white ethnicity and age greater than 35 years.⁵ Additionally, our patient presented with dyspnoea, acute hypoxia, acute hypotension and cardiac arrest shortly after spontaneous rupture of membranes which are consistent with AFE.^{3,5} Although no specimen was collected for histological evidence for AFE, this does not exclude AFE in this case as it is a diagnosis of exclusion and management should be initiated immediately based on clinical suspicion.^{1,3} Our patient suffered from a cardiac arrest which 30–87% of patients experience with AFE.² Given the patient's symptoms, risk factors and hospital course, we described a successfully treated AFE with therapeutic hypothermia.

As demonstrated in this case, the use of therapeutic hypothermia in the setting of AFE resulted in a neurologically intact and otherwise healthy patient. A search of PUBMED and OVID MEDLINE (English language; 1960 to present; search terms 'amniotic fluid embolism,' 'therapeutic hypothermia,' 'pregnancy') was performed. Of the articles returned in these searches, none discussed hypothermia in the maternal management of AFE. Other case reports have discussed supportive management such as factor VII in treating disseminated intravascular coagulopathy secondary to AFE⁶ and nitric oxide has been used to improve cardiorespiratory status.⁶ To provide haemodynamic stability in the setting of AFE the use of cardiopulmonary bypass,⁷ arteriovenous extracorporeal membrane oxygenation⁸ and temporary right ventricular assist device have been described.⁶

Although the specific aetiology of AFE is unknown, it is thought to have an immunological component following rupture of membranes. Immune mediators lead to myocardial depression, pulmonary hypertension and shock with or without disseminated intravascular coagulopathy.¹ We believe that prompt administration of supportive management may improve maternal outcome. The use of therapeutic hypothermia can allow for adequate perfusion to the brain decreasing neurological complications while immunological mediators dissipate and cardiogenic sequelae resolve particularly in the setting of AFE and cardiac arrest. According to the

International Resuscitation Guidelines, therapeutic hypothermia is utilized to improve neurological outcomes and decreased mortality in patients who survive cardiac arrest.⁹ One case describes the successful use of therapeutic hypothermia after cardiac arrest during pregnancy.¹⁰

This case demonstrates timely use of therapeutic hypothermia shortly after cardiac arrest secondary to AFE resulting in a neurologically normal outcome. The judicious use of therapeutic hypothermia most likely contributed to her successful neurologic resolution. Much research is needed in the area of management of AFE. Clinicians should individualize their treatment for AFE and consider all of the available treatment options, including therapeutic hypothermia.

DECLARATIONS

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