

CASE REPORT

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Treatment of amniotic fluid embolism using a modified advanced cardiac life support pulseless electrical activity protocol

Dmitri Chamchad, Maria Lebedev, Erin Langan, Jay Horrow

ABSTRACT

Introduction: Several treatment paradigms exist to resuscitate patients who suffer amniotic fluid embolism (AFE). None has achieved universal acclaim.

Case Report: A 34-year-old G2P1 woman with 38-week twin gestation underwent primary cesarean section with combined spinal-epidural anesthesia. Shortly after delivery of twin A, maternal blood pressure (BP) was 49/22 mmHg, SpO₂ 60%; she lost consciousness. During 12 minutes of resuscitation, she received gentle mask ventilation, hydrocortisone, diphenhydramine, ondansetron, and two doses of epinephrine. Vital signs stabilized. This approach minimized pulmonary vascular resistance by (1) using epinephrine; (2) avoiding intubation, positive end-expiratory pressure (PEEP), and fluid resuscitation; (3) blocking serotonin with ondansetron; and (4) blocking thromboxane with hydrocortisone, then ketorolac. These interventions decrease pulmonary edema and congestion.

Conclusion: We report a case of successful resuscitation from AFE that avoided intubation and PEEP. Successful AFE treatment should focus on keeping pulmonary vascular resistance low.

Keywords: Amniotic fluid embolism, Pulmonary capillary leak, Pulmonary hypertension, Pulseless electrical activity

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INTRODUCTION

Amniotic fluid embolism (AFE), a rare, often rapidly fatal complication of labor and delivery, occurs when fetal amniotic fluid enters the maternal circulation [1]. The hypothesized pathophysiology cites an anaphylactoid reaction to fetal antigens leading to a massive cytokine storm. The ensuing release of vasoactive and procoagulant mediators causes pulmonary hypertension, systemic hypotension, and subsequent cardiovascular collapse [1]. In developed countries, AFE comprises half of maternal deaths occurring in the first hour of delivery and 10% of all peri-partum maternal deaths [2, 3]. Successful treatment requires rapid recognition and intervention [1–4]. Protocols for AFE management remain poorly studied. The A-OK protocol consists of atropine, ondansetron, and ketorolac [5], while the American Heart Association Advanced Cardiac Life Support (ACLS) protocols would employ epinephrine and hydrocortisone for circulatory support [6].

This report demonstrates how a novel combination of the A-OK protocol [5] and the ACLS pulseless electrical activity protocol [6], modified to avoid positive end-expiratory pressure (PEEP), can prevent the need for intubation and subsequent institution of extracorporeal membrane oxygenation (ECMO).

Written Health Insurance Portability and Accountability Act authorization has been obtained from the patient.

CASE REPORT

A 34-year-old G2P1 woman at 38 weeks gestation with twins underwent a planned, primary cesarean section due to breech-transverse presentation. The placentas were noted to be anterior. Following an adequate block with combined spinal–epidural anesthesia, the surgeons made a low transverse hysterotomy, delivering twin A in breech position and twin B in cephalic after internal version. Suddenly after delivery of twin A, the mother lost consciousness; her blood pressure (BP) decreased to 49/22 mmHg and oxygen saturation (SpO₂) to 60% (Table 1).

Suspecting either an air embolism or AFE, caregivers administered 0.3 mg IV epinephrine, resulting in a

transient increase in BP to 187/117 mmHg. She then received IV hydrocortisone 100 mg, diphenhydramine 50 mg, and IV ondansetron 4 mg. The patient’s BP decreased to 84/52 mmHg (Table 1), necessitating a second dose of 0.3 mg IV epinephrine that yielded a peak BP of 221/132 mmHg. The BP remained above 140/80 mmHg thereafter. SpO₂ returned to 100% within 2–3 minutes of diagnosis, without intubation. The patient received only gentle mask ventilation without increased PEEP. Her immediate post-operative course included IV 30 mg ketorolac.

Placement of a radial arterial catheter 20 minutes after initial hypotension, and soon after circulatory stabilization, provided an arterial blood sample with the following values: pH 7.44, PCO₂ 31 mmHg, PO₂

Table 1: Serial intraoperative vital signs around the time of loss of consciousness

Time (min)	SpO ₂	BP (mmHg)	NIBP (mean) mmHg	HR (bpm)	Treatment
931	100%			76	
932	100%			66	
933	100%			66	
934	100%	134/85		60	
935	100%	122/92	105	67	
936	99%			65	
937	99%			131	
938	97%			96	
939	60%	49/22	31	79	Epinephrine 0.3 mg
940	.			70	
941	100%			98	Hydrocortisone 100 mg
942	100%			106	diphenhydramine 50 mg ondansetron 4 mg
943	100%			127	
944	100%	187/117	146	114	
945	100%	136/73	98	99	
946	100%			94	
947	100%	105/61	78	83	
948	100%			83	
949	100%	84/52	64	92	Epinephrine 0.3 mg
950	100%			93	
951	100%			131	
952	100%	221/132	167	128	
953	100%	162/116	134	134	
954	100%			127	
955	100%	143/86	110	124	
956	100%			115	
957	100%	147/87	112	110	
958	100%			109	
959	100%	151/95	118	109	
1000	100%			107	
1005					Chemistry and coagulation studies

330 mmHg (15 L/min face mask oxygen), bicarbonate 21.1 mEq/L, base deficit 2.4 mEq/L, lactate 1.1 mEq/L, glucose 71 mg/dL, sodium 136 mEq/L, potassium 3.3 mEq/L, ionized calcium 0.98 mEq/L, hemoglobin 100 g/L, activated partial thromboplastin time 29 seconds, prothrombin time 13.7 seconds, international ionized ratio 1.1, and fibrinogen 354 mg/dL.

DISCUSSION

Amniotic fluid embolism is a catastrophic complication of delivery; its pathogenesis remains poorly understood. Initially, pulmonary congestion occurs, presumably from pulmonary vascular obstruction with contributions from cytokine-mediated pulmonary vasoconstriction, erythrocyte aggregation, and compression of the pulmonary vasculature [3, 7]. Cardiogenic shock and consumptive coagulopathy may follow. The initial pulmonary hypertension causes severe ventilation/perfusion mismatching and hypoxemia [7]. Damage to the endothelial-alveolar membrane and capillary leak result in pulmonary edema [7]. Circulatory collapse, hypoxemia, and sudden loss of consciousness, occurring in the reported case immediately following delivery of twin A, strongly suggested AFE.

Current treatment paradigms include chest compressions, intubation, positive pressure ventilation with PEEP, fluids, vasopressors, and ultimately ECMO. The sequence of actions presented in this report avoids positive pressure ventilation, PEEP, and fluid resuscitation to minimize compression of the pulmonary vasculature. Rather, this approach treats AFE as pulseless electrical activity, using epinephrine to increase peripheral resistance and decrease pulmonary resistance. This mitigates pulmonary edema and congestion and avoids increasing pulmonary vascular obstruction (Figure 1). Both hydrocortisone and ketorolac decrease thromboxane activity to minimize cytokine storm and platelet dysfunction [8]. The blockade of serotonin receptors from ondansetron helps to relieve pulmonary hypertension. This protocol omits atropine since epinephrine sufficiently increases heart rate.

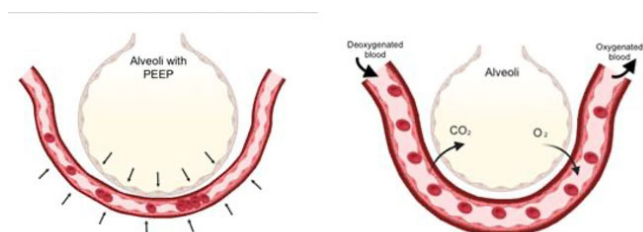


Figure 1: Left: Normal alveoli-capillary gas exchange. Right: Alveoli with PEEP in the setting of AFE-induced pulmonary vasoconstriction and vascular compression.

CONCLUSION

The reported case demonstrates successful resuscitation from AFE using epinephrine, hydrocortisone, ondansetron, and diphenhydramine. This combination includes appropriate ACLS approaches, eliminating the potentially harmful aspects of intubation and PEEP on pulmonary vascular resistance, and avoiding ECMO. Additional evaluation is needed to determine its effectiveness in treating AFE.

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Author Contributions

Dmitri Chamchad – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Maria Lebedev – Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Erin Langan – Conception of the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Jay Horrow – Analysis of data, Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission

The corresponding author is the guarantor of submission.

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Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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