Aortic Compression and Cross Clamping in a Case of Placenta Percreta and Amniotic Fluid Embolism: A Case Report

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ABSTRACT

Amniotic fluid embolism (AFE, also known as anaphylactoid syndrome of pregnancy) at the time of surgery for placenta percreta has been previously reported. We report here a case in which AFE and associated cardiac arrest occurred following a hysterectomy for placenta percreta. In this case, subhepatic manual aortic compression during the cardiac arrest and chest compressions followed by infrarenal aortic cross-clamping during volume infusion and reversal of the coagulopathy were associated with a successful resuscitation and good maternal outcome.

KEYWORDS: Amniotic fluid embolism, placenta percreta, aortic cross clamping, bispectral index monitoring, zinc protoporphyrin

CASE REPORT

The patient was a 33-year-old Hispanic woman (gravida 6, para 4) with three prior transverse lower uterine segment cesarean sections (and one ectopic pregnancy) who presented for a repeat cesarean section and total abdominal hysterectomy for known placenta previa percreta. Antenatal ultrasound and magnetic resonance imaging had confirmed percreta into the bladder dome. She was managed in hospital for the last 3 weeks of her pregnancy. Planned cesarean–hysterectomy was performed at $36^0/_7$ weeks. Bilateral ureteric stents and internal iliac artery balloon catheters (uninflated) were placed prior to surgery. Internal jugular central venous access and a radial arterial line were present. A BIS (Bispectral Index) monitor (BIS, Aspect Medical System Inc., Newton, MA) was placed prior to surgery for cerebral activity monitoring (target BIS level = 40 to 50) in accordance with standard practice at the facility for cases with potential for significant blood loss.[1] General endotracheal anesthesia was induced. Delivery via a fundal and posterior uterine hysterotomy through a midline infra- and supraumbilical incision was made difficult by dense adhesions between the abdominal wall and the uterus and omentum. There was approximately a 10-minute interval between starting the surgery and delivery. The baby was obtunded at birth (male, 3802 g, Apgars 2 and 8 at 5 and 10 minutes, respectively) with blood-stained but otherwise clear amniotic fluid. He responded to resuscitation and subsequently did well.

The internal iliac artery catheters were inflated following delivery. The entire lower uterine segment was highly vascular with clear invasion of the placenta into the posterior bladder wall. There were multiple large abnormal blood vessels noted extending from the pelvic

side walls to the lower segment and placental mass. No attempt was made to separate the area of percreta, and a 5 × 5-cm portion of the bladder was excised and removed with the uterus. The patient had lost 2500 mL of blood to this point and had received 5 U of packed red blood cells (PRBCs) and 4 U of fresh frozen plasma (FFP). She had been hemodynamically stable (blood pressure = 110 to 120/40 to 50 with heart rate of 110 beats per minute) to this point (3 hours into the case), and her hematocrit was 36% with normal PT/PTT, fibrinogen, and pH. Blood replacement was controlled and contemporaneous with operative bleeding. Approximately 15 minutes after removal of the uterus and cervix, during closure of the bladder, the patient experienced a sudden drop in oxygen saturation from 100 to 78%, followed by bradycardia that proceeded to asystole and a pulseless state in under 5 minutes. The advanced cardiac life support protocol was initiated and cardiac compression and epinephrine were given. The surgeon immediately reached into the upper abdomen and identified and compressed the abdominal aorta just below the liver. The effectiveness of the CPR was gauged by the presence of a detectable pulse on the pulse oximeter. A transesophageal echocardiogram (TEE) probe was placed after the cardiac arrest and confirmed complete asystole with no evidence of fibrillation. The right ventricle was noted to contain obvious debris suggestive of amniotic fluid embolism (AFE) and was significantly dilated and motionless. Within minutes of the arrest, the patient was noted to have developed coagulopathy and she began bleeding massively from the unsutured bladder and abdominal wall incisions, previously dry surgical pedicles, as well as from her nose and mouth. Chest compressions continued for ~27 minutes (11:30 to 11:57 a.m.) with intermittent stops to check for cardiac activity (asystole confirmed with TEE). Ventricular fibrillation then began and defibrillation with 200J × 2 was used to convert the patient to a sinus tachycardia. Over the next 30 minutes, aggressive resuscitation continued with blood loss being controlled using manual subhepatic aortic compression and pelvic pressure packs. Bleeding persisted despite all efforts, and ventricular fibrillation again occurred for approximately 10 minutes, necessitating chest compressions and two further 200J shocks to reestablish sinus tachycardia. The vascular surgeon was then asked to cross-clamp the infrarenal aorta, and manual aortic compression was discontinued. The patient finally stabilized after 45 minutes of aortic cross-clamping during which the internal iliac arteries were ligated and bleeding in the pelvis was controlled. Once the patient had established a sinus rhythm and a stable blood pressure, the aortic cross-clamp was removed and distal limb pulses were checked and were present bilaterally. Bowel and renal perfusion were observed and noted to be satisfactory. The patient remained in disseminated intravascular coagulation

with no observed clotting for a further 30 to 40 minutes, during which time FFP and cryoprecipitate were given and abdominal and pelvic packing was maintained. Estimated blood loss was 21,000 mL, and volume resuscitation was performed with blood and crystalloid (54 U PRBCs, 15 U FFP, five "jumbo" packs each of platelets and cryoprecipitate, and 9000 mL of crystalloid). She was also given activated factor VII (90 μg/kg) × two doses.

Once the vascular surgeon was satisfied that no embolectomy was required and the patient had started clotting, large-bore abdominal drains were placed, the bladder and rectus fascia were closed (but not the skin), and the patient was taken to the intensive care unit for postoperative care. Total time in the operating room was approximately 8 hours. The BIS decreased from the optimum anesthesia level of 40 to 50 down to 30 for ~45 minutes during the resuscitation and recovered back to prearrest levels immediately after reestablishing sinus rhythm.

She remained ventilated overnight and received 2 further U of PRBCs. She was extubated the following morning uneventfully with no apparent central nervous system or permanent organ system deficit. Her creatinine and cardiac and liver enzymes increased and her thyroid function diminished (Table 1) transiently, but there was no long-term organ failure. A zinc protoporphyrin level was checked on a specimen of blood taken ~15 minutes before she developed hypoxia and bradycardia, and the level was markedly elevated for an adult (813 nmol/L; normal adult level is 0 to 637 nmol/L).

The patient was discharged home with an indwelling bladder catheter on postoperative day 11 after her skin was closed secondarily. Her immediate postoperative complications included a transient Sheehan syndrome and a vesicovaginal fistula that was repaired definitively at 6 weeks postpartum. She never developed any central nervous system complications, and her family and spouse did not notice personality or cognitive function deficits during her first 6 weeks postpartum. Table 1 shows selected blood results just before the cardiac arrest occurred (preevent) and again during surgery and in the postpartum period.

DISCUSSION

AFE is a clinical diagnosis that hinges on three major features: acute onset hypoxia, fulminant right heart failure with cardiovascular collapse, and rapid-onset coagulopathy.[2] The syndrome is believed to be the result of an anaphylactoid-type response with pulmonary hypertension and activation of coagulation as hallmarks.[2,3] It could be argued that our patient simply suffered the effects of massive hemorrhage. However, the sudden onset of hypoxia while being ventilated, the rapid development of asystole and coagulopathy in the space of

Table 1 Selected Blood Tests

Test	Intraoperative Preevent	Intraoperative Postevent	POD 1	POD 2	POD 3
Hct (%)	36.6 (2 min before collapse)	21	31.3	26.9	30.1
Hgb (g/dL)	11.6	7	11.1	9.3	10.7
Platelets (1000/μL)	214	52	95	81	100
PT (s)	13 (10.7–13.9)	17.6	12.7	11.7	
PTT (s)	31 (23–36)	113	30	25	
Fibrinogen (mg/dL)		100 (200–400)	356		
D-dimer (μg/mL)		1.0 (<0.5)	2	2	
Creatinine (mg/dL)		0.7 (0.4–1.0)	1.1	1.4	1.3
BUN (mg/dL)		8	10	16	27
AST/ALT (U/L)		165/59	168/62	67/52	38/42
TSH (uIU/mL) FT4 (ng/dL)		0.68 (0.71–1.85) 5.96 (0.45–4.67)			
Prolactin (ng/mL)			250 (1.4–24.2)		
Cortisol (μg/dL)			46 (3–16)		
Creatine kinase (U/L)		261 (20–200)	314		
CKMB (ng/mL)		18.4 (0–5.0)	16.3		
Troponin I (ng/mL)		47.2 (0.3–2.0)	10	5	
K (mmol/L)		7.1 (3.5–5.0)	4.4	3.5	4.3
pH		6.88 (7.35–7.45)	7.48		
Base deficit (mmol/L)		29 (2–10)			
PaO$_2$ (mm Hg)		113 (80–105)			
SaO$_2$%	100	78	100	98	98
Zinc protoporphyrin (nmol/L)		813 (0–637)			

The postevent data represent the highest (or lowest) representative level for that specific parameter on each day. The normal range for our laboratory is included in parentheses. ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CKMB, creatine kinase MB; FT4, thyroxine free; Hct, hematocrit; Hgb, hemoglobin; K, potassium; PaO$_2$, partial pressure of oxygen; POD, postoperative day; PT, prothrombin time; PTT, partial thromboplastin time; SaO$_2$, oxygen saturation; TSH, thyroid stimulating hormone.

minutes in a previously stable patient who was not bleeding uncontrollably, and the TEE images of an enlarged right ventricle[4] and intraventricular debris point much more strongly to AFE. This has previously been reported in placenta percreta.[5] The significance of the elevated maternal zinc protoporphyrin in blood drawn just before the AFE event is unclear. Zinc levels are increased in meconium, and elevated coproporphyrin levels have been reported in AFE.[6] In this case, zinc coproporphyrin was ordered but zinc protoporphyrin was inadvertently measured. Elevated zinc protoporphyrin is considered an indicator of iron-deficiency anemia or chronic lead exposure toxicity and is not known to be elevated in normal pregnancy.[7,8] Unfortunately, zinc protoporphyrin tested by hematofluorometry may also be artificially elevated by many interfering substances, which we were not able to rule out in this case.[9] We speculate that zinc protoporphyrin may be a marker of AFE but encourage further research.

The remarkable outcome of this patient is difficult to explain given her ~35 minutes of asystole or ventricular fibrillation. We attribute it to the preparations for the management of massive hemorrhage that were in place, the coordinated efforts of all of the people involved, and the persistence with CPR despite what appeared to be a hopeless situation (pH = 6.88, pulseless

electrical activity, asystole). The patient never recorded a hematocrit of < 21% (optimal for oxygen delivery and consumption) despite a blood loss of 21,000 mL. Although there was a coagulopathy, her platelets were never below 50,000/mL, and the coagulopathy was rapidly reversed once she started perfusing again. The recording of BIS before, during, and after the arrest is unique and has not been reported in AFE. The fact that BIS never fell below 30 was very reassuring during the code and encouraged persisting with our CPR efforts given the evidence of adequate brain perfusion and activity despite asystole. Although BIS is not a complete electroencephalogram, there are data that show that BIS does accurately reflect brain activity and specifically brain death.[10]

We speculate that the immediate compression of the aorta beneath the liver at the time of the cardiac arrest decreased the bleeding from the abdomen and increased the efficiency of the cardiopulmonary resuscitative efforts. Massive volumes of blood products and fluid were infused into the central veins, and the volume of distribution was to some extent limited to the upper body and brain. The perfusing effect of the cardiac compressions could be seen as a perturbation on the pulse oximeter and reassured us that despite the prolonged cardiac arrest and resuscitation, an adequate level

of peripheral (and presumably brain) perfusion was occurring. The peripheral oxygen saturation remained between 77% and 85% throughout the periods of chest compression. The TEE was useful in allowing the anesthesiologists to gauge the cardiac status and to see when the right heart failure reversed and the pulmonary hypertension abated. Although visualization of the asystole was most disconcerting, we could see the state of filling and when the pulseless electrical activity converted to ventricular fibrillation (which was amenable to defibrillation).

The infrarenal cross-clamping and pelvic pressure packs diminished the active bleeding and allowed us further time to resuscitate the patient and reverse the coagulopathy.[11–13] The coagulopathy that was present protected our patient from thrombosis, and no embolectomy was needed. We should stress, however, that following aortic cross-clamping, every effort should be taken once the patient is stabilized to rule out ischemic thrombosis, and if necessary, embolectomy should be performed. In this case, we released the aortic clamp before the coagulopathy was reversed and before clotting was noted.

We feel that our patient makes the case for subhepatic aortic compression and aortic cross-clamping in patients with massive postpartum hemorrhage and/or cardiac arrest. Given the recommendation that the uterus be emptied in pregnant women with cardiac arrest[14] to help in the resuscitative efforts (decreased shunt, decreased caval compression), we feel that once the abdomen is open and cardiac arrest has occurred, subhepatic aortic compression until cardiac activity is restored and then infrarenal aortic cross-clamping until bleeding is controlled may be lifesaving.

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