

# Post resuscitation care of a postpartum cardiac arrest patient: A case report

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## ABSTRACT

We are presenting a case of a 30-year-old pregnant lady with Para2 Gravida2. She underwent an earlier lower segment caesarean section (LSCS) for her first baby. Her second delivery was also through LSCS but had excessive bleeding and hence subtotal hysterectomy was done. During this procedure, patient went in for an “on table” cardiac arrest. She was resuscitated and referred to our hospital in post cardiac arrest status for post-resuscitation care. We share our post-resuscitation care experience of this patient.

**Key words:** Cardiac arrest, Postpartum, Post-resuscitation care

## INTRODUCTION

The incidence of primary cardiac arrest in the hospital is approximately 1.5-3.0/1000 admissions<sup>[1]</sup> and rates of survival to hospital discharge are approximately 17%.<sup>[2]</sup> Pregnant and postpartum women needing admission to an intensive care unit (ICU) are a distinctive but rare group, constituting <1% of all ICU admissions of which very few are postpartum with obstetric complications. Postpartum in-hospital cardiac arrest is very uncommon, especially in a healthy patient.<sup>[3]</sup> The pathological events occurring at the time of cardiac arrest prolong and extend into the post-arrest phase causing more organ damage.<sup>[4]</sup> The cardiac arrest cases with a Return of spontaneous circulation (ROSC) are admitted to an ICU for further care. The post-resuscitation phase starts at the location where ROSC is achieved but, once stabilized; the patient is transferred to an ICU for intensive care management.

## CASE REPORT

Our patient was a 30-year-old pregnant south Indian lady, a homemaker, Para2 Gravida2 with no antenatal

history of any maternal disease. She had undergone an earlier lower segment caesarean section (LSCS) of her first child 2 years earlier. The patient reported for a second full-term delivery with an anterior placental presentation in a limited facility private hospital elsewhere. She underwent LSCS and a healthy baby was delivered. During caesarean section, she developed severe intractable postpartum hemorrhage for which sub-total hysterectomy was planned and carried out. During caesarean section, the patient went in for an “on table” cardiac arrest. Following successful resuscitation and return of spontaneous circulation after about 30 min, the patient was transferred to the emergency room (ER) at our hospital after about 6 h.

On receiving the patient at the ER, the patient was already intubated with a 7.5 size endotracheal tube and was fixed at 19 cm at the level of the incisors. She was on bag mask valve ventilation with bilaterally equal air entry with SPO<sub>2</sub> of 96% on 4L O<sub>2</sub>. Arterial blood gas showed respiratory acidosis. Her blood pressure was 110/70 mmHg on dopamine support (20 mics/kg/min) with an 18G intravenous (IV) cannula at her right arm and heart rate was 140/min.

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All peripheral pulses were felt. AB +ve blood was being transfused while receiving in the ER. Serum lactate was 2.54 mmol/L. Electrocardiogram was taken which showed sinus tachycardia. Bedside 2D-ECHO was done which showed good cardiac contractility, no right atrial/right ventricular enlargement and with an inferior vena cava (IVC) diameter around 0.5 cm (more than 50% collapsible). Hence, fluid boluses were administered until IVC diameter was <50% collapsible. Screening for deep vein thrombosis (DVT) was done and ruled out. Glasgow coma scale (GCS) was  $E_1V_1M_1$ ; pupils were 3 mm bilaterally reactive to light. Capillary blood sugar in the ER was high (capillary blood glucose- 322 mg/dL), hence the patient was started on insulin infusion using a syringe pump. She had a nasogastric (NG) tube and a Foley's catheter in-situ draining adequate amount (around 100-120 ml/h) of urine. She had no pressure sores. LSCS scar was seen and head to toe examination was otherwise normal. Triple lumen central venous line was placed in the right internal jugular vein (IJV). Therapeutic hypothermia (32-34°C) was chosen for the comatose patient. Therapeutic hypothermia was induced by external cooling using ice packs (kept at the patient's axillae and groins) and an ice blanket. As an esophageal probe was not available, an NG tube was slit longitudinally, and a conventional multiparameter thermometer probe (connected to the monitor) was inserted into the slit NG tube and introduced into the esophagus of the patient up to 30 cm from the incisors. Samples were taken for investigations before induction of hypothermia. All essential investigations were done. Hemoglobin was 7.9 g/dL and total count was found to be 30300 cells/cu mm. The bleeding profile was found to be normal. The patient was shifted to the ICU after inducing hypothermia and changing all tubes and lines as per the hospital protocol.

In the ICU, (day 1), controlled ventilation was initiated as per the standard recommendations.<sup>[5]</sup> The patient was connected to mechanical ventilator which was put on volume control mode with the following parameters - 450 ml tidal volume, respiratory rate of 12 breaths/min was started to achieve partial pressure of end-tidal carbon dioxide of 35-40 mmHg, inspiratory: Expiratory ratio was kept at 1:2, 10 cm H<sub>2</sub>O pressure support, positive end-expiratory pressure of 5 cmH<sub>2</sub>O. Controlled re-oxygenation is done by adjusting the FiO<sub>2</sub> to achieve an arterial oxygenation of 94-98%. Blood pressure was 110/70 mmHg on dopamine infusion at 20 mics/kg/min (started outside) and was continued as per the guidelines. The patient was sedated and paralyzed with 3 mg/h

infusions of midazolam and vecuronium to prevent tremors.

Elevated blood sugar was continuously being controlled by IV insulin Infusion with a target range for blood glucose concentration of up to 8 mmol/L (144 mg/dL) and should be <10 mmol/L (180 mg/dL). HBA1c (glycated hemoglobin) was within the normal range. She was started on these antibiotics, injection cefoperazone + sulbactam 1.5 g IV BD; injection amikacin 500 mg IV BD, injection metrogyl (metronidazole) 500 mg IV TDS as a post-operative order by the surgeon; injection rantac (ranitidine) 50 mg IV BD as stress ulcer prophylaxis and low molecular weight heparin (LMWH) clexane (enoxaparin) 40 mg S/C OD which was started as DVT prophylaxis. During the course of her stay, pulmonologist and neurologist opinions were obtained. Computed tomography brain (plain + contrast) and Doppler of neck vessels were done and were normal.

On the day 2, therapeutic hypothermia was stopped after 24 h of cooling. Two units of albumin were transfused in view of Anasarca (hypoalbuminemia [2.2 g/dL]). LMWH was withheld due to mild bleeding per vagina treated by local packing. She gained consciousness and her GCS improved to  $E_3V_1M_6$ . Insulin infusion was stopped (as the sugars were controlled) after initiating subcutaneous insulin. On the day 5, dopamine was tapered and stopped. On day 6, general condition of the patient improved and was fit to be extubated. After extubation, she was shifted to the ward. She was given regular physiotherapy and was mobilized. On day 9, the patient was made to ambulate; sutures were removed, and the patient was discharged.

## DISCUSSION

Unconscious mechanically ventilated survivors of cardiac arrest account for 5.8% of all admissions to ICUs in the United Kingdom (UK). Organ injury caused by ischemia and hypoxia during prolonged cardiac arrest is compounded by reperfusion injury when a spontaneous circulation is restored. A systemic inflammatory response, similar to that associated with sepsis, causing multiple organ dysfunctions is evident termed as a "sepsis-like" syndrome.<sup>[6]</sup> Prompt and effective interventions in the post-resuscitation period have a significant influence on the ultimate outcome.<sup>[7]</sup>

Usually according to the guidelines, therapeutic hypothermia has to be induced within 2 h of arrest but in our patient, therapeutic hypothermia could not be induced within 2 h as the patient was received only

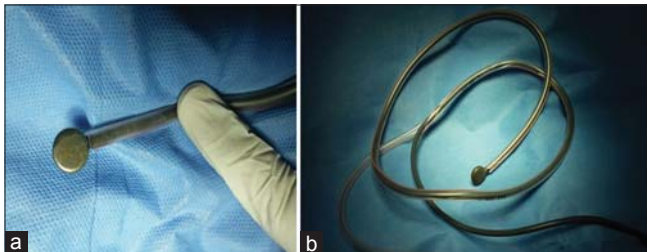
after 6 h of arrest but the survival without any neurological deficit, could be achieved with delayed induction of hypothermia.

In general, in western countries, therapeutic hypothermia is to be induced actively using both internal and external cooling devices. Instead in this case, we could successfully induce therapeutic hypothermia externally using only ice packs and iced blanket which was monitored by an esophageal “thermometer” probe. As an esophageal probe was not available, a slit was made longitudinally on an NG tube and a conventional (multiparameter) thermometer probe (connected to the monitor) was inserted within the NG tube which was then introduced into the esophagus of the patient (Figure 1a and b).

Some clinicians advocate the administration of albumin solution because of two possible advantages over fluid repletion with an isotonic saline solution: (1) more rapid plasma volume expansion, since the colloid solution remains in the vascular space (in contrast to saline, three-quarters of which enters the interstitium), and (2) lesser risk of pulmonary edema, since dilutional hypoalbuminemia will not occur.<sup>[8]</sup>

Albumin administration improves organ function in critically ill hypoalbuminemic patients.<sup>[9]</sup> Our patient was given albumin as it is known to reduce cerebral edema and is of prognostic value. A study has shown that the survival rate and neurological outcomes are more favorable in patients with higher serum albumin following ROSC after cardiac arrest.<sup>[10]</sup> However, Randomized trials and meta-analyses have failed to demonstrate benefits from the use of albumin.<sup>[11-14]</sup>

Although hypoalbuminemia is associated with increased mortality, the use of albumin in critically ill patients with a serum albumin concentration  $\leq 25$  g/liter is not associated with reductions in mortality, duration of ICU stay or mechanical ventilation, or in the use of renal replacement therapy.<sup>[15]</sup>



**Figure 1:** (a and b) “conventional (multiparameter) thermometer probe” was inserted into a slit nasogastric tube which was introduced into the esophagus of the patient

## CONCLUSION

Intensive care of a post-cardiac arrest resuscitated patient requires collaboration across different specialties. Implementation of the post-cardiac arrest care bundle<sup>[16]</sup> integrating management should be standard protocol adjusted for local needs in all ICU. The best possible resuscitation care must be given always even if placed at a periphery with limited facilities. The outcome of patients admitted to ICU after cardiac arrest should also be a subject of the regular audit.

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