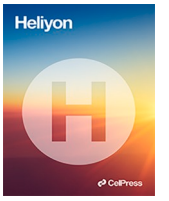
Heliyon 10 (2024) e23337

Contents lists available at ScienceDirect

Heliyon

journal homepage: www.cell.com/heliyon



Case report: Maternal cardiac arrest at 12 hours postpartum

Patchareya Nivatpumin *, Saranya Lertkovit

Department of Anesthesiology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand

ARTICLE INFO

Keywords:
Maternal cardiac arrest
Postpartum
Preeclampsia
Pulmonary edema

ABSTRACT

Maternal cardiac arrest is a rare occurrence. In this case report, we present a detailed account of a 37-year-old pregnant woman with preeclampsia with severe features who underwent cesarean delivery. The patient experienced dyspnea and hypoxia at 12 hours postpartum, leading to cardiac arrest in the maternity ward. Advanced cardiac life support measures, including 15 minutes of chest compressions, were performed until spontaneous circulation was restored. This study explores the underlying factors contributing to maternal cardiac arrest during the postpartum period. Additionally, it highlights the effective strategies employed by our multidisciplinary team in managing and resolving this critical medical event.

1. Introduction

Maternal cardiac arrest is very rare. The primary causes of maternal cardiac arrest are hemorrhage, preeclampsia, cardiovascular issues, medication errors or overdosage, amniotic fluid embolism (AFE), and anesthetic-related factors [1,2]. In this report, we present a case of a patient with preeclampsia who experienced cardiac arrest in the maternity ward 12 hours after undergoing a cesarean delivery. Our objective is to systematically examine the potential causes of cardiac arrest and thoroughly analyze the resuscitative measures and management implemented by the medical team.

2. Case report

A 37-year-old gravida 3, para 2-woman of Thai ethnicity, with the gestational age of 34 weeks (96 kg, 168 cm) was admitted to our hospital in June 2020. She presented with hypertension and the symptom of edema in her legs and eyelids. Her previous two pregnancies were uneventful, with the first pregnancy resulting in a normal vaginal delivery and the second pregnancy being a cesarean delivery three years ago. She had no known underlying diseases and had received three antenatal care visits with no history of previous hypertension.

On arrival, her initial blood pressure was 206/127 mmHg, with a heart rate of 100 beats per minute. Her lung examination revealed clear lungs, and her oxygen saturation on room air was 97 %. Laboratory investigations showed the following results: hemoglobin 13.5 g/dL, hematocrit 39.2 %, platelets 245,000/mm^3, blood urea nitrogen (BUN) 5.4, creatinine (Cr) 0.61, uric acid 4.9 mg/dL, total bilirubin 0.62 mg/dL, direct bilirubin 0.30 mg/dL, aspartate transaminase 17 unit/L, alanine transaminase 11 unit/L, and serum lactate dehydrogenase (LDH) 238 unit/L. Urinalysis showed 3+ proteinuria and a urine protein per creatinine ratio of 4.5. A diagnosis of preeclampsia with severe features was made.

* Corresponding author. Division of Obstetric Anesthesia, Department of Anesthesiology, Faculty of Medicine Siriraj Hospital, Mahidol University, 2 Wanglang Road, Bangkok Noi, Bangkok, 10700, Thailand.
E-mail address: patchareya.niv@mahidol.ac.th (P. Nivatpumin).

https://doi.org/10.1016/j.heliyon.2023.e23337
Received 23 October 2022; Received in revised form 8 November 2023; Accepted 1 December 2023
Available online 4 December 2023

The patient received continuous intravenous drip of magnesium sulfate, intravenous labetalol (a total of 120 mg), and intramuscular dexamethasone 5 mg every 12 hours (a total of 20 mg). The preoperative serum magnesium level was 4.0 mg/dL (3.3 mEq/L). An urgent cesarean delivery was performed due to the history of previous cesarean delivery and the presence of preeclampsia accompanied by difficult-to-control blood pressure. The operation was done under spinal anesthesia using 0.5 % heavy bupivacaine (11 mg) and morphine (200 mcg). Intraoperatively, a total of 1200 ml of intravenous fluids was administered, with an estimated blood loss of 600 ml and 80 ml of urine throughout the procedure. The operation proceeded without complications, and there were no postanesthetic care unit complications. Following the operation, the patient was transferred to the maternity ward. The patient was prescribed a continuous infusion of magnesium sulfate at a rate of 2 g per hour for 24 hours after delivery. Intravenous infusion of 5 % dextrose in NSS/2 solution was administered at a rate of 120 ml per hour, resulting in a cumulative urine output of 400 ml over approximately 8 hours postoperatively. The patient did not receive venous thromboembolism prophylaxis.

At 12 hours after surgery, the patient developed dyspnea and desaturation, with an oxygen saturation of 88 %. An O_2 mask with a flow rate of 10 LPM was administered, resulting in an increase in oxygen saturation to 95 %. Subsequently, the patient experienced tachypnea with a respiratory rate of 30 breaths per minute. Mild drowsiness was observed, along with normal deep tendon reflexes. The planned treatment involved intubation; however, prior to the intubation attempt, the patient became unconscious. Initial monitoring using a 3-lead cardiac monitor showed electrical activity, but no pulse was detected. As a result, chest compressions were initiated and continued for 15 minutes. During this time, the patient received 3 doses of 1 mg adrenaline and 10 ml of 10 % calcium gluconate. The infusion of magnesium sulfate was discontinued. After initial unsuccessful attempt, the second intubation was successful. As a result, return of spontaneous circulation was achieved. The patient regained consciousness, albeit with a slow response to commands. Twelve-lead electrocardiograms revealed sinus tachycardia without ST-segment elevation. No signs of anemia or external bleeding were observed. Auscultation of the lungs revealed rales in both lungs. Subsequently, the patient was transferred to the cardiac intensive care unit (Fig. 1).

A portable chest X-ray revealed marked cardiomegaly with pulmonary congestion in both lungs (Fig. 2). Laboratory investigations performed in the patient ward, approximately 30–60 minutes after the cardiac arrest, revealed a hemoglobin concentration of 12.9 g/dL and a serum magnesium level of 6.9 mg/dL (equivalent to 5.7 mEq/L). Arterial blood gas analysis revealed a pH of 7.222, PaO_2 of 106 mmHg (FiO_2 1), $PaCO_2$ of 39 mmHg, bicarbonate of 15.8 mmol/L, and a base excess of −11.1 mmol/L. The serum sodium level was measured at 133 mEq/L, while serum glucose, renal function, and coagulogram were within the normal range. The serum troponin-T level was 99.6 ng/L. Transthoracic echocardiogram findings indicated a cardiac ejection fraction of 58 %, with no abnormalities in regional wall motion. The left ventricle showed concentric hypertrophy, and there was evidence of diastolic dysfunction (abnormal relaxation). Trivial regurgitation was observed in the pulmonic valve and tricuspid valve, with a right atrial pressure of 10 mmHg. Pulmonary hypertension was noted, with a pulmonary artery pressure (PAP) reading of 44/18 mmHg and a mean PAP of 30

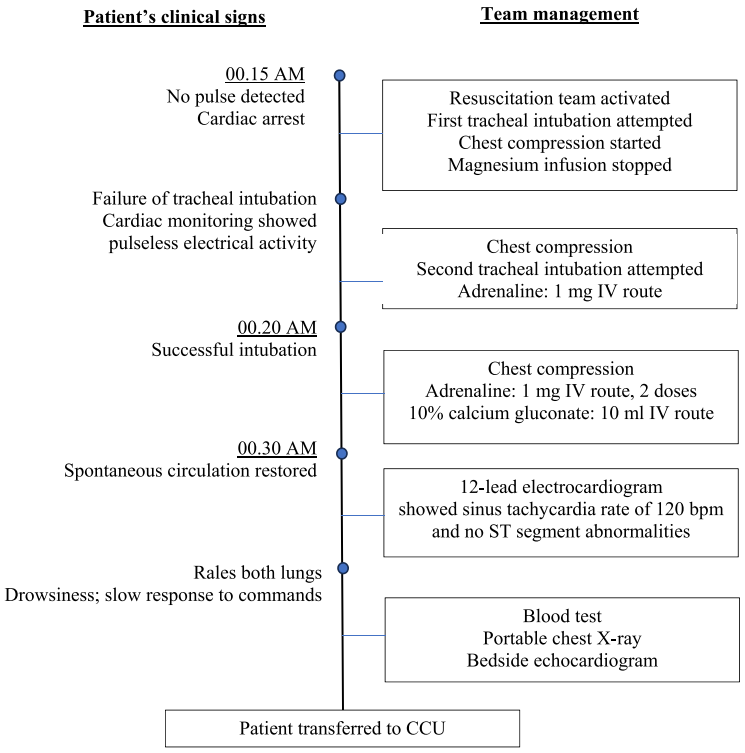


Fig. 1. Timeline of cardiac arrest
Abbreviations: bpm, beats per minute; CCU, cardiac intensive care unit; IV, intravenous; ml, milliliter.

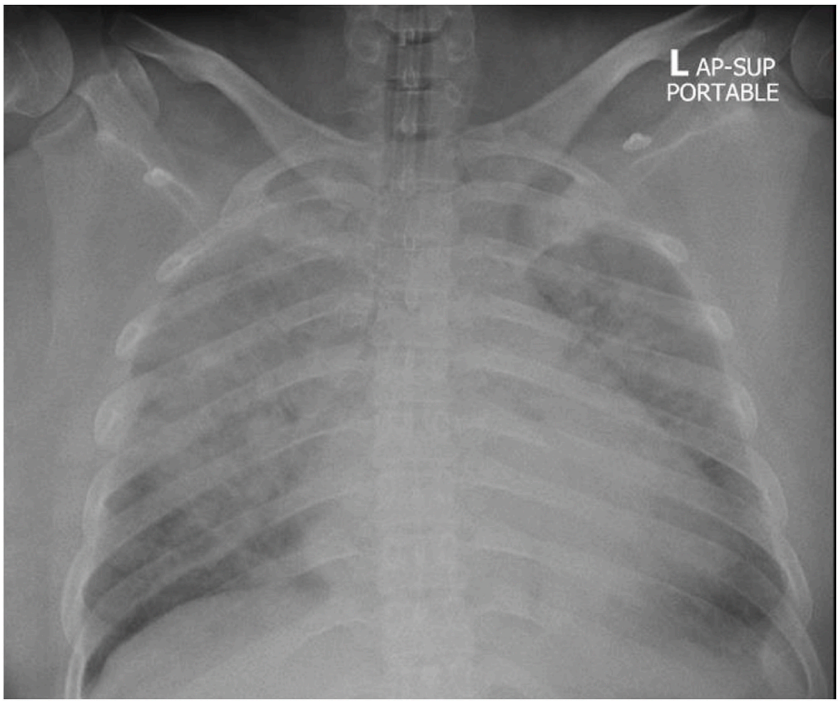


Fig. 2. Patient's chest X-ray immediately after cardiac arrest and return of spontaneous circulation.

mmHg (calculated using Abbas's formula). No impairment of right ventricular function or abnormalities in the size of the aorta were observed. The patient was diagnosed with pulmonary edema and congestive heart failure.

In the cardiac intensive care unit, the patient received pressure-controlled ventilation with a peak inspiratory pressure of 14 cmH_2O, positive end-expiratory pressure of 5 cmH_2O, and FiO_2 of 0.4. Intravenous furosemide was administered with a total dosage of 200 mg. The patient also received intravenous nitroglycerin and a continuous drip of intravenous nicardipine. After diuresis with a

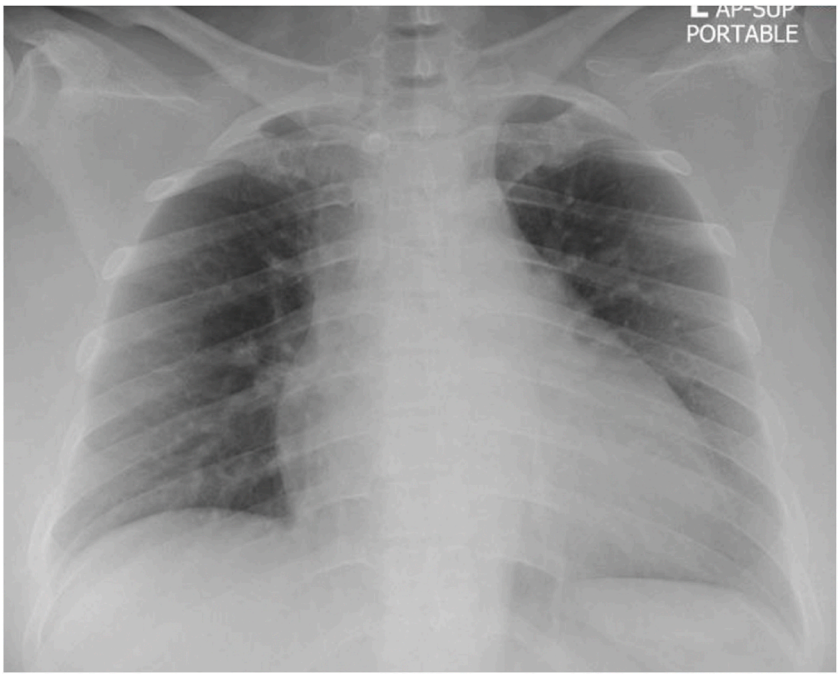


Fig. 3. Patient's chest X-ray 3 days postpartum.

negative fluid balance of 3500 ml over 2 days, the patient was extubated on postpartum day 3 and subsequently transferred to the maternity ward. A follow-up portable chest X-ray revealed improvement in bilaterally pulmonary congestion (Fig. 3). The patient's neurological examination was within normal limits, except for transient amnesia on the day of cardiac arrest. She was prescribed oral antihypertensive medications (manidipine and enalapril) and was discharged from the hospital on day 12. Subsequently, she followed up with a cardiologist and underwent an echocardiogram in April 2021. The echocardiogram revealed a good left ventricular ejection fraction (LVEF) of 66 %, mild concentric left ventricular hypertrophy, no regional wall motion abnormality, no significant valvular lesions, mild right atrial dilatation with a normal right ventricular size, and good right ventricular contraction. The diagnosis of hypertensive heart disease was confirmed. The patient is currently in good functional status and continues to receive oral antihypertensive medication (manidipine and losartan).

3. Discussion

Several causes of postpartum cardiac arrest have been reported, such as massive postpartum hemorrhage, pulmonary embolism, peripartum cardiomyopathy (PPCM), magnesium toxicity, and anaphylaxis [3]. Our patient was diagnosed with preeclampsia with severe features, a condition characterized by multiorgan involvement, with the majority of clinical manifestations being derangements of the cardiovascular system, primarily systemic hypertension [4]. Preeclampsia is associated with a reduction in plasma colloid oncotic pressure and alterations in endothelial permeability, which predispose individuals to pulmonary edema [4].

Upon arrival at the hospital, our patient presented with an acute hypertensive crisis. This contributed to the development of pulmonary edema through sympathetic nervous system activation, venoconstriction, and vasoconstriction, resulting in increased afterload and fluid redistribution from the peripheral to the pulmonary circulation [4]. Furthermore, perioperative fluid administration played a significant role in triggering pulmonary edema in our patient. She underwent cesarean delivery with spinal anesthesia, which required a substantial amount of fluid administration to maintain stable hemodynamics after the spinal block. Acute pulmonary edema in pregnancy commonly occurs in the postpartum period due to intravenous fluid administration aimed at increasing intravascular volume or correcting oliguria [4]. Even though our patient had normal renal function, her preoperative condition and the treatments she received led to the development of acute pulmonary edema, left ventricular heart failure, hypoxic respiratory failure, and ultimately cardiac arrest during the postpartum period. These findings were confirmed by echocardiography. The echocardiogram revealed concentric left ventricular hypertrophy and diastolic dysfunction, indicating left-sided heart failure with associated pulmonary edema.

However, a differential diagnosis should be conducted to identify and address the underlying cause of cardiac arrest. Postpartum hemorrhage is the most common cause of maternal cardiac arrest and death [1,5]. In this patient, the presence of obvious bleeding was examined and confirmed through normal hemoglobin levels. Another diagnosis including acute myocardial infarction can be a cause of peripartum cardiac arrest [1,2], no ST-T segment abnormalities were observed on the electrocardiogram, and the echocardiogram revealed no regional wall motion abnormalities.

Amniotic fluid embolism (AFE) cannot be ruled out in this presentation, given the cardiovascular collapse and hypoxia, as the signs and symptoms of AFE can resemble those of other obstetric causes [6,7]. However, no other clinical abnormalities associated with AFE, such as neurological symptoms (e.g., seizures) or hematological abnormalities (e.g., disseminated intravascular coagulation), were observed [6,7]. Furthermore, considering that AFE typically occurs during the labor and delivery period, it is unlikely to be the cause of cardiac arrest in this patient [6].

This patient is classified as obese, with a body mass index (BMI) of 34.0 kg/m^2. Obesity during pregnancy is associated with an increased risk of obstetric and anesthetic complications in the perinatal period, including hypertension, diabetes, difficult intubation, and thromboembolism [8,9]. The "CAPS study" conducted in the United Kingdom investigated cardiac arrest in pregnancy and found that 37.8 % (25/66 patients) of the patients had a BMI exceeding 30 kg/m^2 [10]. Additionally, among the patients who experienced cardiac arrest related to obstetric anesthesia, 12 out of 16 were obese [10]. Thromboembolism also can be a cause of cardiac arrest in the peripartum period and is a significant concern for obese parturients [10]. Reports have demonstrated cases of cardiac arrest resulting from pulmonary embolism in morbidly obese parturients during the perioperative period [11,12]. Given the absence of thromboprophylaxis in our patient, thromboembolism should be considered as a potential differential diagnosis. The echocardiogram performed on the patient did not reveal characteristic signs associated with pulmonary embolism, such as interventricular septal bulging into the left ventricle (D-shape septum) and pulmonary artery dilatation.

Another potential cause of cardiac arrest in this patient was peripartum cardiomyopathy (PPCM). Factors such as patient age over 30, preeclampsia, and maternal hypertension can predispose individuals to PPCM [13,14]. The presenting signs and symptoms of PPCM include heart failure symptoms, such as dyspnea and pulmonary edema, which align with this patient's presentation [14]. Similarly, the patient's chest X-ray revealed pulmonary congestion and cardiomegaly, which could be attributed to both preeclampsia with pulmonary edema or PPCM. To obtain a definitive diagnosis, an echocardiogram was necessary. The echocardiogram findings for PPCM typically demonstrate an ejection fraction lower than 45 % and global hypokinesia [14].

This patient was diagnosed with preeclampsia with severe features and received magnesium infusion, which led to respiratory failure prior to the onset of cardiac arrest. According to the updated 2020 American Heart Association guideline for cardiac arrest in pregnancy, it is recommended to consider administering calcium chloride or calcium gluconate when a patient is receiving intravenous magnesium and the infusion is stopped [15]. This precaution is taken due to the potential for magnesium toxicity, which can result in respiratory failure and cardiovascular collapse [16]. The therapeutic range for serum magnesium is typically 5–9 mg/dL (4–7 mEq/L), while levels exceeding 12 mg/dL (10 mEq/L) and 30 mg/dL (25 mEq/L) can lead to respiratory paralysis and cardiac arrest, respectively [16]. Although this patient did not exhibit signs of magnesium overdose, such as the absence of deep tendon reflexes,

considering the uncertain cause of cardiac arrest, it would have been prudent to administer calcium chloride or calcium gluconate initially.

After the diagnosis of cardiac arrest in our patient, it was crucial to immediately initiate chest compressions and activate a resuscitation team [1]. Our patient received comprehensive cardiopulmonary resuscitation, which involved a multidisciplinary team comprising obstetricians, anesthesiologists, and internal medicine physicians. Despite the absence of a delay in chest compression, the patient's obesity and preeclampsia presented challenges during intubation due to airway edema. As a result, a second tracheal intubation attempt was necessary. Prolonged delays in intubation can worsen hypoxia in this clinical scenario.

Following return of spontaneous circulation, the patient was promptly transferred to the cardiac intensive care unit and required positive pressure ventilation. Performing an echocardiogram on the patient provided several advantages, including confirming the diagnosis of cardiac arrest and guiding subsequent therapeutic interventions. Notably, the patient's troponin T level was elevated; this finding aligns with research indicating an increase in cardiac enzyme levels following cardiopulmonary resuscitation, even without defibrillation, due to cardiac injury [17,18]. Interestingly, subsequent monitoring of troponin T levels was not conducted as myocardial infarction was ruled out based on the findings of the ECG and echocardiogram. However, it is worth noting that the N-terminal pro-B-type natriuretic peptide (NT-pro BNP) level was not assessed, and this measurement could have provided valuable insights into the diagnosis of heart failure in this patient [19].

There are several aspects that warrant improvement in our case. It is strongly recommended to identify indicators suggestive of magnesium overdose, such as diminished deep tendon reflexes, somnolence, dyspnea, or respiratory distress. Judicious fluid administration throughout the preoperative, intraoperative, and postoperative stages is of paramount importance. Patients who have preeclampsia with severe features are inherently at risk of developing pulmonary edema, making prudent fluid management and meticulous monitoring of urine output crucial factors to consider.

4. Conclusions

Postpartum pulmonary edema with congestive heart failure associated with preeclampsia is a very rare cause of cardiac arrest; nevertheless, this cause is preventable. Although there were no signs of pulmonary edema upon arrival at the hospital, intraoperative fluid administration, along with liberal fluid management in the postpartum period, can contribute to fluid overload. We strongly recommend judicious fluid management during the perioperative period and increased awareness of this potential situation in patients with preeclampsia. Additionally, prompt cardiopulmonary resuscitation with a multidisciplinary team approach and correction of the underlying cause of cardiac arrest can lead to successful restoration of the patient's condition.

Funding

None.

Ethical statement

Approval from the ethics committee was not required for this study. Consent for publication was directly obtained from the patient. The authors obtained permission from the hospital's director to utilize the patient's data and chest X-ray for research purposes.

Data availability statement

No data was used for research described in the article.

CRediT authorship contribution statement

Patchareya Nivatpumin: Conceptualization, Data curation, Project administration, Supervision, Validation, Writing – original draft, Writing – review & editing. **Saranya Lertkovit:** Conceptualization, Data curation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] F.M. Jeejeebhoy, et al., Cardiac arrest in pregnancy: a scientific statement from the American heart association, Circulation 132 (18) (2015) 1747–1773.
[2] Y. Helviz, S. Einav, Maternal cardiac arrest, Curr. Opin. Anaesthesiol. 32 (3) (2019) 298–306.
[3] A. Morton, Postpartum collapse, Obstet. Med. 14 (1) (2021) 46–49.
[4] A.T. Dennis, C.B. Solnordal, Acute pulmonary oedema in pregnant women, Anaesthesia 67 (6) (2012) 646–659.
[5] B.-O. Committee on Practice, Practice bulletin No. 183: postpartum hemorrhage, Obstet. Gynecol. 130 (4) (2017) e168–e186.
[6] A.A. Shamshirsaz, S.L. Clark, Amniotic fluid embolism, Obstet. Gynecol. Clin. N. Am. 43 (4) (2016) 779–790.
[7] J. Moore, M.R. Baldisseri, Amniotic fluid embolism, Crit. Care Med. 33 (10 Suppl) (2005) S279–S285.

[8] A. Tonidandel, et al., Anesthetic and obstetric outcomes in morbidly obese parturients: a 20-year follow-up retrospective cohort study, Int. J. Obstet. Anesth. 23 (4) (2014) 357–364.
[9] M.C. Smid, et al., Maternal obesity and major intraoperative complications during cesarean delivery, Am. J. Obstet. Gynecol. 216 (6) (2017) 614 e1–e614 e7.
[10] V.A. Beckett, M. Knight, P. Sharpe, The CAPS Study: incidence, management and outcomes of cardiac arrest in pregnancy in the UK: a prospective, descriptive study, BJOG 124 (9) (2017) 1374–1381.
[11] D.K. Ho, et al., Anesthetic management of a super morbidly obese obstetric patient with a body mass index of 109 kg/m2 presenting for her fourth caesarean delivery, Cureus 12 (11) (2020), e11803.
[12] P. Nivatpumin, et al., Comparison of perioperative outcomes and anesthetic-related complications of morbidly obese and super-obese parturients delivering by cesarean section, Ann. Med. 55 (1) (2023) 1037–1046.
[13] Z. Arany, U. Elkayam, Peripartum cardiomyopathy, Circulation 133 (14) (2016) 1397–1409.
[14] M.C. Honigberg, M.M. Givertz, Peripartum cardiomyopathy, BMJ 364 (2019) k5287.
[15] American Heart Association, Cardiac Arrest in Pregnancy In-Hospital ACLS Algorithm, American Heart Association, 2020. http://cpr.heart.org/-/media/CPR-Files/CPR-Guidelines-Files/Algorithms/AlgorithmACLS_CA_in_Pregnancy_InHospital_200612.pdf. (Accessed 12 December 2022).
[16] Gestational hypertension and preeclampsia: ACOG practice bulletin, number 222, Obstet. Gynecol. 135 (6) (2020) e237–e260.
[17] C.C. Lin, et al., The influence of cardiopulmonary resuscitation without defibrillation on serum levels of cardiac enzymes: a time course study of out-of-hospital cardiac arrest survivors, Resuscitation 68 (3) (2006) 343–349.
[18] C. Palmiere, et al., Cardiac troponins and NT-proBNP in the forensic setting: overview of sampling site, postmortem interval, cardiopulmonary resuscitation, and review of the literature, Forensic Sci. Int. 282 (2018) 211–218.
[19] M. Weber, C. Hamm, Role of B-type natriuretic peptide (BNP) and NT-proBNP in clinical routine, Heart 92 (6) (2006) 843–849.