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Extracorporeal cardiopulmonary resuscitation for sudden cardiac arrest induced by septic shock-related adrenal crisis

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Abstract:

Extracorporeal cardiopulmonary resuscitation is being increasingly used to treat refractory in-hospital cardiac arrest (IHCA). Etiologies of IHCA may differ from etiologies of out-of-hospital cardiac arrest. We report a case of a 50-year-old man who was admitted to a local hospital, presenting with drowsiness, hypotension, and severe metabolic acidosis. After being transferred to our tertiary center, he quickly progressed to cardiac arrest and required extracorporeal cardiopulmonary resuscitation (eCPR) with veno-arterial extracorporeal membrane oxygenation (VA ECMO). Initially, due to high levels of inflammatory markers, sepsis became the most probable diagnosis. The patient responded well to antibiotics and supplemental corticosteroid therapy. Subsequent investigation revealed sepsis-induced absence of cortisol based on previously unknown hypopituitarism. Following corticoid administration, rapid myocardial recovery occurred with successful ECMO weaning. The patient was discharged from the ICU after 13 days with a favorable neurological outcome. Therefore, VA ECMO seems to be a feasible method to provide a bridge to recovery in patients with sudden hemodynamic collapse due to an adrenal crisis.

Keywords:

Adrenal crisis, extracorporeal cardiopulmonary resuscitation, septic shock, veno-arterial extracorporeal membrane oxygenation

Introduction

Extracorporeal cardiopulmonary resuscitation (eCPR) is increasingly being used to treat refractory in-hospital cardiac arrest. The etiologies of in-hospital cardiac arrest (IHCA) may differ from those of out-of-hospital cardiac arrest.[1] Severe adrenal insufficiency (adrenal crisis) is a rare, life-threatening medical condition, caused by a critical lack of glucocorticoid, with or without deficiency of mineralocorticoids and adrenal androgens. Usually, it is caused by disorders of the adrenal glands (i.e., primary adrenal insufficiency or Addison's disease), or by an impairment of the function of the hypothalamic-pituitary-adrenal axis, resulting in the deficiency of adrenocorticotropic hormone (secondary adrenal insufficiency).[2] The clinical manifestations are often nonspecific, with a broad range of symptoms, making the diagnostics challenging. In most severe cases, the adrenal crisis may manifest as a sudden hemodynamic collapse or cardiac arrest, with the need for cardiopulmonary resuscitation.[3]

We present the case of a 50-year-old male with previously undiagnosed hypopituitarism

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who developed sepsis followed by circulatory arrest. eCPR using veno-arterial extracorporeal membrane oxygenation (VA ECMO) was required until full recovery was achieved.

Case Report

A 50-year-old male presented to the local emergency department with symptoms of drowsiness, fatigue, hyponatremia, hypokalemia, and hypotension (85/50mmHg) [Table 1]. The symptoms commenced one day prior and progressively worsened. A significant weight loss of 30 kg over the previous year was reported. His medical history was unremarkable, except for an episode of myopericarditis one year earlier. Laboratory investigations revealed elevated inflammatory markers, with a C-reactive protein level of 177 mg/L and a procalcitonin level of 19.9 ng/L. In addition, his serum level of N-terminal pro-B-type natriuretic peptide (NT-proBNP) was elevated at 8332 ng/l [Table 1]. The initial computed tomography (CT) scan, performed at a local hospital, revealed bilateral pulmonary infiltrates, making sepsis the most probable diagnosis. After being transferred to our hospital, he was already on high doses of vasopressors to maintain a mean arterial pressure ≥65 mmHg (0.8 µg/kg/min norepinephrine and 2.4 IU/min vasopressin). Elevated lactate level, interleukin 6, CRP and procalcitonin levels were recorded, with a qSOFA score of 3 and a SOFA score of 14 points. Initial transthoracic echocardiography performed in the emergency department showed a well-preserved left ventricular ejection fraction (LVEF) of 55% with no signs of pericardial effusion or pulmonary embolism. A significant increase in NT-proBNP was observed compared to the results reported by the local hospital [Table 1].

After transferring the patient to the ICU, he experienced severe bradycardia and asystole, leading to cardiopulmonary resuscitation (CPR) using an automated CPR device (CorPuls CPR, Elektromedizinische Geräte G. Stemple, Germany). No return of spontaneous circulation could be achieved within 15 min of conventional CPR. eCPR was performed as per our local protocol. A peripheral VA ECMO (Cardiohelp, Maquet, Getinge, Sweden) was implanted, and full ECMO flow was reached within the next 50 min. Although the patient had previously exhibited normal left ventricular function, a repeated echocardiogram after eCPR revealed a severely reduced LVEF of 5%. To support left ventricular unloading, an intra-aortic balloon pump was inserted. The ECG after eCPR revealed severe sinus tachycardia with diffuse repolarization changes in the precordial leads. Due to ongoing hemodynamic instability, ECG after eCPR and increasing levels of high-sensitivity troponin, coronary angiography was performed, which revealed normal findings.

To treat the septic shock, broad-spectrum antibiotic treatment with cefotaxime 2 g every 8 h along with azithromycin 500 mg every 24 h was initiated as per the local protocol. Due to high doses of vasopressors, including norepinephrine (0.5ug/kg/min) and vasopressin (0.03 IU/min) to maintain an adequate mean arterial pressure on ECMO, 200 mg of hydrocortisone per day was administered (50 mg every 6 h). The next day, we observed a significant decrease in vasopressor requirements and a gradual improvement in cardiac function (LVEF improved to 25%). Six days after admission, the patient's cardiac function had recovered sufficiently, and the patient was weaned from ECMO with an LVEF of 40%. The following day, the patient was successfully liberated from mechanical ventilation without notable neurological impairment. Soon after, the patient underwent intensive rehabilitation and began oral intake.

After ECMO weaning, the patient's endocrine test results revealed low cortisol (7 nmol/l) and adrenocorticotropic hormone (ACTH) (<0.22 pmol/l) levels that were suggestive of adrenal crisis, which became clinically apparent due to septic shock [Table 2]. Therefore, further diagnostic tests, including brain magnetic resonance imaging, were conducted and revealed empty sella turcica syndrome.[4]

Table 1: The initial laboratory results

Parameter	Value	Reference range
C-reactive protein (mg/L)	159	0.0–5.0
Interleukin 6 (ng/L)	>3300	0–4.4
Procalcitonin (ug/L) – local hospital	19.85	0–0.1
Procalcitonin (ug/L) – admission	35.33	0–0.1
Leucocytes ([10×9]/L)	3.0	4–10
NT-pro BNP (ng/L) – local hospital	8332	5–125
NT-pro BNP (ng/L) – admission	>35,000	5–125
High sensitive troponin I (ng/L) – local hospital	161.3	0–64
High sensitive troponin I (ng/L) – admission	380.9	0–64
Sodium (mEq/L)	125	136–145
Potassium (mmol/L)	3.0	3.5–5.1

NT-pro BNP: N-terminal pro b-type natriuretic peptide

Table 2: Hormonal profile after transferring the patient to the endocrinology unit

Hormone	Value	Reference range
Cortisol (nmol/L)	7	145.4–619.4
Adrenocorticotropic hormone (pmol/L)	<0.22	1.1–10.1
Free trijodthyronin (pmol/L)	1.73	3.50–6.50
Free thyroxin (pmol/L)	8.38	11.50–22.70
Anti thyreoglobulin antibodies (kIU/L)	0.18	<115
Anti thyroidperoxidase antibodies (kIU/L)	0.99	<34
Sex hormone-binding globuline (nmol/L)	32.4	20.6–76.7
Luteinizing hormone (IU/L)	2.91	1.7–8.6
Follicle-stimulating hormone (IU/L)	3.77	1.5–12.4
Prolactin (ng/mL)	9.06	3.8–21.2
Insulin-like growth factor 1 (ng/mL)	165	70.9–205

At the time of diagnosis, the patient was still receiving 200 mg of hydrocortisone daily intravenously. After consulting an endocrinologist, the replacement therapy was changed to 20 mg of hydrocortisone orally every 8 h. He was discharged from the ICU after 13 days and then referred to the endocrinology department for further treatment. A hormonal profile is shown in Table 2. Six months after being released from the hospital, he is alive, resuming routine activities, and taking prednisone 5 mg orally as a daily substitution treatment. The patient has given written consent approving the publication.

Discussion

Over the past decade, eCPR has been increasingly utilized worldwide as a rescue technique among patients with refractory cardiac arrest. Etiologies of IHCA may differ from etiologies of out-of-hospital cardiac arrest. The most frequent causes of IHCA in general ward settings are hypoxia, acute coronary syndrome, arrhythmias, hypovolemia, and septic shock.[1] The decision-making for eCPR is often time-critical and relies on incomplete information. Therapy is most frequently indicated as a bridge to recovery in cardiogenic shock due to various causes.[5]

An adrenal crisis is a medical emergency that typically manifests during stressful situations. The clinical presentation of hypopituitarism, regardless of the underlying cause, varies depending on the type and extent to which hormones are deficient.[3,4] In our case, the dysfunction of the ACTH axis was the primary clinical feature, as the levels of other pituitary hormones were within normal ranges. Due to the near-complete absence of ACTH, affected patients are unable to produce enough of the stress hormone cortisol to cope with the extreme stress. Patients with hypopituitarism present a clinical challenge due to the rarity of the disease and a wide range of unspecific symptoms associated with cardiovascular collapse.[3,6] In our case, hypopituitarism emanated from previously undetected empty sella syndrome (ESS). Empty sella" refers to the neuroradiological or pathological finding of an empty sella turcica that contains no pituitary tissue.[7]

At the time of the intervention, we did not suspect any underlying hormonal disorder. After confirming bilateral pneumonia in CT scan (realized in a local hospital), using the Sepsis 3 definition, the diagnosis of septic shock was suspected and treated.[8] In sepsis, dysregulation of the hypothalamic–pituitary–adrenal axis, alterations in cortisol metabolism, and tissue resistance to glucocorticoids can all result in relative adrenal insufficiency or critical illness-related corticosteroid insufficiency.[9] We suspect that in the case of our patient, this phenomenon was significantly aggravated by previously undiagnosed pre-existing hypopituitarism, which led to progressive hypotension and circulatory arrest. Given the refractory asystole, the eCPR with VA ECMO was necessary as a last-resort intervention. Corticosteroid substitution, as recommended for severe sepsis, led to fast cardiac recovery. This prompted us to evaluate the hormone profile, leading to the diagnosis of adrenal crisis and ultimately to a magnetic resonance imaging confirming ESS. Reports about patients suffering a circulatory arrest as a consequence of an adrenal crisis are scarce. Lüsebrink *et al*. reported a case of a 19-year-old woman with cardiogenic shock as the first manifestation of her previously unknown Addison's disease. She had nonspecific clinical manifestations (i.e., lethargy, fatigue, arthralgia, dyspnoea, and hypotension) comparable to those of our presented patient. Venoarterial extracorporeal membrane oxygenation was also successfully used as a salvage intervention in this case, as well.[10]

Thus, the evaluation of a hormone profile should be taken into consideration in acute admissions with a history of unclear fatigue, hyponatremia, and hypotension after ruling out the common cardiovascular causes. The early diagnosis and an appropriate substitution therapy, along with the proper education of the patients (increasing the doses of corticosteroids during stressful situations, acute illness, or prior to an operation), are very important, contributing significantly to an increased quality of life and reduction of the morbidity and mortality rates.[7,9] In our case, with appropriate replacement therapy, the patient made a full recovery and was discharged from the hospital without neurological impairment.

Conclusion

An adrenal crisis is a life-threatening condition that requires immediate treatment. In rare cases of circulatory arrest due to a lack of glucocorticoids, achieving the return of spontaneous circulation using conventional CPR may not be possible. eCPR with VA-ECMO may thus be used as a bridge to effective treatment with corticoid substitution, which is essential to achieving recovery.

Author contributions statement

MP: Conceptualization, Resources, Visualization, Writing - original draft, Writing, Reviewing and Editing. TG, DR, MM, MI: Resources, Writing, Reviewing. TG, DR, MM, MI: Writing – Extensive Review and Editing.

Conflicts of interest

None Declared.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal.

The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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