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Use of Extracorporeal Cardiopulmonary Resuscitation in a Fatal Case of Pneumococcal Septic Shock Post-Splenectomy: A Case Report and Literature Review

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Abstract

Overwhelming Post-Splenectomy Infection (OPSI) is a rare but recognized life-threatening infection in patients with a history of splenectomy. These patients are susceptible to severe infections with encapsulated bacteria, especially in unvaccinated individuals. The role of Venoarterial Extracorporeal Membrane Oxygenation (VA-ECMO) in adult patients with sepsis remains disputed. While the European Resuscitation Council endorses Extracorporeal cardiopulmonary Resuscitation (ECPR) as a rescue strategy when standard Advanced Life Support (ALS) measures fail, its application in cases of cardiac arrest secondary to sepsis is not addressed in the latest guidelines. Similarly, the latest revision of the Surviving Sepsis Campaign Guidelines provides no specific recommendations regarding VA-ECMO in circulatory collapse. We report a case of a 34-year-old male, post-splenectomy due to Evans syndrome, who presented with flu-like symptoms that rapidly deteriorated into septic shock and cardiac arrest. ALS was initiated immediately. Following the witnessed In-Hospital Cardiac Arrest (IHCA), the ECPR protocol was implemented. Despite comprehensive interventions, the patient died. Blood cultures proved positive for streptococcus pneumoniae. Although numerous case reports describe successful use of VA-ECMO or ECPR in septic shock, reports of fatal outcomes remain exceedingly rare. With this case we intend to offer a more balanced perspective on the potential limitations and risks of VA-ECMO or ECPR in septic shock, addressing the likelihood of publication bias that favors reports of positive outcomes.

Keywords

Streptococcus Pneumoniae, Septic Shock, Asplenic, Overwhelming

Post-Splenectomy Infection, Extracorporeal Cardiopulmonary Resuscitation

1. Introduction

Evans syndrome is a rare autoimmune disorder marked by a concurrent or successive onset of Autoimmune Hemolytic Anemia (AIHA) and Immune Thrombocytopenia (ITP), sometimes accompanied by neutropenia, without an identifiable cause [1]-[3]. The understanding of Evans syndrome in adults is sparse. Initial treatment typically involves corticosteroids or Intravenous Immunoglobulin (IVIG), while second-line therapies include splenectomy and other immunosuppressive agents [2] [3]. Complications, primarily comprising bleeding, infection, and thrombosis, typically occur within the first couple of years following diagnosis. Their frequency appears to increase with the number of treatment lines, suggesting an additional iatrogenic effect [4].

Regarding infectious complications, the relatively rare phenomenon of immune neutropenia might contribute to the risk of severe sepsis. Additionally, treatment with corticosteroids is a known risk factor for infection and splenectomy significantly increases the risk of infections, particularly those caused by encapsulated bacteria such as streptococcus pneumoniae [5]-[7]. In some cases, infections in patients that underwent a splenectomy can lead to severe sepsis known as Overwhelming Post-Splenectomy Infection (OPSI) [7] [8]. Adequate medical follow-up, vaccination strategies and anti-infectious prophylaxis are essential and significantly reduce the risk of infection with encapsulated bacteria in this subset of patients [8] [9].

The use of Venoarterial Extracorporeal Membrane Oxygenation (VA-ECMO) in septic shock is still controversial. Extracorporeal Cardiopulmonary Resuscitation (ECPR) can be defined as the implantation of VA-ECMO in a patient who experienced a sudden cardiac arrest [10]. While the European Resuscitation Council (ERC) recommends ECPR as a rescue strategy when standard advanced life support (ALS) measures are unsuccessful, its application in cases of cardiac arrest secondary to sepsis is not specifically addressed in the latest guidelines [11]. The latest revision of the Surviving Sepsis Campaign Guidelines (SSCG), the international practice guideline for sepsis, endorses Venovenous Extracorporeal Membrane Oxygenation (VV-ECMO) for severe Acute Respiratory Distress Syndrome (ARDS). It however does not provide specific recommendations regarding VA-ECMO or ECPR for cases involving circulatory collapse [12].

This report details a case of a patient who suffered a therapy-refractory pneumococcal septic shock post-splenectomy. It is complemented by a literature review on adult cases of severe septic shock and the use of VA-ECMO and ECPR.

2. Case Description

A 34-year-old male with a medical history of splenectomy 14 years prior due to

Evans syndrome and in remission since, presented to the emergency department admitted by prehospital Emergency Services (EMS). Upon EMS arrival at the patient's home, he reported flu-like symptoms since the night before and appeared stable. He had a normal body temperature after taking 1 g of paracetamol one hour before. The vital parameters were normal, apart from oxygen saturation, which could not be measured due to cold extremities (Table 1). Oxygen therapy and 500 mL of Intravenous (IV) Plasmalyte were initiated.

Table 1. Evolution of vital parameters.

Time	BP (mmHg)	HR (/min)	Sat (%)	Temp (˚C)	RR (/min)
7:01 AM	114/87	53	?	35.9	18
7:19 AM	111/60	110	99 + 15LO2	/	25

BP: Blood Pressure; HR: Heart Rate; Sat: Oxygen Saturation; Temp: Temperature; RR: Respiratory Rate; AM: Ante Meridiem.

On arrival at the hospital at 7:18 AM (see Table 2 for timeline of important events), the patient demonstrated rapid clinical deterioration (Table 1). Physical examination revealed signs of septic shock, including livedo reticularis, a prolonged capillary refill time and decreased consciousness (Glasgow Coma Scale 13/15). He was tachypneic and reported widespread myalgias. Fluid resuscitation was continued with an additional 500 ml of IV balanced crystalloids. An arterial blood gas confirmed tissue hypoperfusion with a markedly elevated lactate (Table 3). Blood samples and cultures were obtained. The chest radiograph was normal. Within the first hour, the patient received 2 g of IV ceftriaxone and 2 g of IV amikacin, following hospital-specific guidelines for septic shock post-splenectomy. Due to mild hypoglycaemia, 10 g of IV glucose was administered. Aggressive fluid resuscitation was continued and 2 liters of balanced crystalloids were administered rapidly. Norepinephrine was started at 0.1 mcg/kg/min peripherally. He received 100 mg of IV hydrocortison. Due to hyperkalaemia and acidosis, bicarbonate 8.4% 100 ml IV was administered. In total, the patient received 2.5 litres of balanced crystalloids in the first hour.

Table 2. Timeline of important events.

Time	Event
7:01 AM	Emergency services arrival at the patient's home
7:18 AM	Patient arrived at the hospital
7:45 AM	Both antibiotics administrated
8:01 AM	First cardiac arrest
8:05 AM	Return of spontaneous circulation
8:08 AM	Second cardiac arrest
8:30 AM	Start of ECPR cannulation

Continued

8:49 AM	Cannulation successful, ECPR pump-on
9:14 AM	Ventricular fibrillation, defibrillation
9:30 AM	Additional cannula placement in right jugular vein

ECPR: Extracorporeal Cardiopulmonary Resuscitation.

Table 3. Evolution of blood gas analyses.

Time	7:26 AM	8:07 AM	9:05 AM	9:19 AM	10:15 AM	
Test	**ABG**	**VBG**	**ABG**	**Extracorporeal BG**	**Extracorporeal BG**	**Reference Range ABG**
pH	7.03	/	6.98	6.83	6.77	7.37 - 7.45
PO_2 (mmHg)	199	35	359	378	406	80 - 108
PCO_2 (mmHg)	23	87	37	38	18	35 - 48
HCO_3 (mmol/L)	6.3	/	8.8	6.4	2.5	22 - 29
Sodium (mmol/L)	141	146	153	148	146	136 - 146
Potassium (mmol/L)	4.7	6.1	6.4	6.0	7.1	3.5 - 4.5
Glucose (mg/dL)	51	75	41	251	99	70 - 105
Haemoglobin (g/dL)	17.1	15.7	7.2	6.5	9.1	14.0 - 18.0
Lactate (mmol/L)	20	23	30	31	>31	0.5 - 2.2

ABG: Arterial Blood Gas; VBG: Venous Blood Gas; Extracorporeal BG: Arterial Blood Gas taken out of extracorporeal circulation.

Despite the initiated therapy, deterioration continued with further declining consciousness and respiratory failure which necessitated tracheal intubation and mechanical ventilation (see follow-up venous blood gas Table 3). Ketamine 100 mg IV, fentanyl 100 mcg IV and rocuronium 100 mg IV were used for rapid sequence induction. Peri-induction adrenaline 100 mcg was administered to stabilize the hemodynamic status. Minutes after uncomplicated intubation, the patient experienced a first in-hospital cardiac arrest presenting with Pulseless Electrical Activity (PEA). ALS was initiated, after 1 mg of IV adrenaline and 4 minutes of ALS, Return Of Spontaneous Circulation (ROSC) was achieved. Nevertheless, a second PEA cardiac arrest subsequently occurred minutes later and ALS was resumed. Following multidisciplinary consultation, the decision was made to acti-

vate the ECPR team. VA-ECMO cannulation commenced at 8:30 AM during ongoing mechanical chest compressions provided by a Lund University Cardiopulmonary Assist System (LUCAS). Cannulation was performed under bedside Transesophageal Echocardiography (TEE) guidance to confirm accurate placement. Echocardiography revealed no pericardial effusion or right-ventricular dilatation. The aortic valve did not open. Severe mitral regurgitation and markedly reduced left ventricular systolic function were observed, along with a mobile structure in the right atrium possibly representing a thrombus.

Additional laboratory results indicated severe thrombocytopenia (Table 4), necessitating platelet transfusion. An additional 80 mg of IV methylprednisolone was administered. A central venous line was placed in the right jugular vein. No IVIG was administered due to resuscitation setting.

Table 4. Additional testing.

Time	Sample	Test	Result	Units	Reference Range
	Blood chemistry testing				
		CRP	91	Mg/L	≤5
		WBC	7.25	×10^9/L	4.0 - 10.0
		Haemoglobin	16.8	g/dL	14.0 - 18.0
		Platelet count	7	×10^9/L	150 - 450
		LDH	564	U/L	135 - 250
		Bilirubin	2.31	Mg/dL	≤1.18
7:29 AM		ALT	139	U/L	≤38
		AST	141	U/L	≤42
		GGT	231	U/L	≤60
		CK	110	U/L	≤190
		Albumin	36.4	g/L	35.0 - 52.0
		Creatinine	2.17	Mg/dL	0.67 - 1.17
	Nasopharyngeal PCR	Influenza A and B	Negative		
		COVID-19	Negative		
	Serological testing				
8:05 AM		Irregular antibodies (LISS/COOMBS)	Negative		
		COVID-19	Negative		

CRP: C-Reactive Protein; WBC: White Blood Cell count; LDH: Lactate Dehydrogenase; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; CK: Creatine Kinase.

Further several adjunctive therapies were administered during ongoing ALS and ECPR cannulation, based on potential benefit and a safe side effects profile, rather than based on strong evidence. Bicarbonate IV was repeated in response to pronounced acidaemia, while 10 g of IV glucose was repeated to address potential hypoglycaemia. Additionally, 500 mg of IV thiamine was administered in case of impairment of cellular metabolism [13]. Clindamycin 600 mg IV was added to reduce potential exotoxin production [14].

Cannulation was successfully performed, and ECPR was ongoing at 8:49 AM. Initial blood flow was limited to 2 L/min; fluid resuscitation was continued. The cardiac rhythm deteriorated to ventricular fibrillation which was promptly terminated with one defibrillation attempt of 200 Joules. Subsequent blood gas analysis showed a decrease in haemoglobin (**Table 3**). Due to concerns for potential bleeding secondary to thrombocytopenia, two units of O-negative packed red blood cells were transfused, along with 2 g of IV calcium chloride. Despite rigorous fluid administration, the maximum ECPR flow reached 2.5 L/min; temporarily halting fluids resulted in a decrease of flow to approximately 1.5 L/min. As a last resort measure to optimize blood flow, our cardiac surgery team decided to place an additional venous drainage cannula in the right jugular vein via guidewire exchange over the central venous line.

The central venous line was replaced in the left femoral vein. Albumin was administered in conjunction with ongoing crystalloid resuscitation. Despite all interventions, flow remained persistently low, and lactate levels continued to rise, reaching more than 30 mmol/L. Following multidisciplinary consultation, the team agreed that further resuscitative efforts were futile. The family was informed after which ECPR was discontinued. The patient deceased shortly after. The family requested an autopsy, which was authorized by the medical team. Hours later, all blood cultures returned positive for gram-positive cocci, setting the stage for a diagnosis of therapy-refractory septic shock.

An autopsy was performed days later. No clear primary infection focus was identified; however, the tonsils were red and swollen. Additional findings included severe congestion and tissue damage in lungs and liver, with evidence of accelerated autolysis in the liver. Mild interstitial inflammation and haemorrhage were observed in the myocardium, along with minor interstitial bleeding in the adrenal glands. Signs of Disseminated Intravascular Coagulation (DIC), including thrombi in the microcirculation of heart, lungs, and kidneys were present. Definitive results from the ante-mortem blood cultures were positive for streptococcus pneumoniae serotype 12F with a sensitivity to all tested antibiotics except levofloxacin, for which there was intermediate sensitivity. The autopsy findings confirmed the diagnosis of a sepsis-related death with multi-organ failure due to pneumococcal sepsis.

3. Discussion

This case exemplifies the manifestation of OPSI, in a patient post-splenectomy for

Evans syndrome. The patient initially presented with mild symptoms, which rapidly escalated into therapy-refractory septic shock and repeated cardiopulmonary arrests. Despite prompt suspicion and initiation of adequate therapy for septic shock following the SSCG—including broad-spectrum antibiotics, fluid resuscitation, vasopression, IV-hydrocortisone- and eventually ECPR, the outcome was fatal. The patient's last mengingococcal, pneumococcal and haemophilus influenza vaccinations dated from 2011, 13 years prior, whereas pneumococcal vaccination is supposed to be repeated after 5 years [5]. Furthermore, Serotype 12F is among the pneumococcal serotypes that are typically covered by current vaccine strategies, offering protection against these invasive infections. Literature reveals that up to 85% of post-splenectomy patients are unaware of their increased vulnerability to certain infections [5]. This case represents a missed opportunity for vaccination, underscoring the need for increased awareness of adequate vaccination prophylaxis and a high index of suspicion for sepsis in post-splenectomy patients presenting with fever or malaise.

The application of VA-ECMO or ECPR in septic shock remains a topic of ongoing debate. The ERC recommends ECPR as a rescue strategy when standard ALS measures are unsuccessful; however, its specific use in cases of cardiac arrest secondary to sepsis is not explicitly addressed in the latest guidelines [11]. Similarly, the latest revision of the SSCG, the international practice guideline for sepsis, endorses VV-ECMO for managing severe ARDS but does not provide specific recommendations regarding VA-ECMO for cases involving circulatory collapse [12].

Recent retrospective studies investigated the use of VA-ECMO in septic shock, with outcomes reported to be variable [15]-[17]. An international multicentre cohort study on ECPR identified higher occurrences of haematological conditions, sepsis and acute renal failure in a group of non-survivors, suggesting that conditions outside the cardiovascular system are associated with poorer ECPR outcomes [18]. Further, several studies indicate poor overall survival rates in sepsis, particularly when initiation of VA-ECMO occurs after cardiac arrest or when pre-ECMO lactate values are high [19]-[21]. Evidence suggests that this treatment option may offer benefits in patients with septic shock when it is associated with septic cardiomyopathy or myocardial injury [19]-[23].

In our case, ECPR was initiated despite initial high lactate levels. The relationship between initial lactate levels and survival is inconsistent across studies, and there is limited evidence for a definitive lactate threshold precluding ECPR initiation [24]. Although septic shock was strongly suspected as the cause of the patient's cardiac arrest, there was diagnostic uncertainty at the time of decision-making for ECPR. Furthermore, our hospital's inclusion and exclusion criteria for initiating ECPR do not consider lactate levels, and the patient did not meet any exclusion criteria.

Faced with suboptimal ECPR flow, we implemented several measures to try to optimize blood flow. Hypovolemia was addressed with rigorous fluid administra-

tion, decreasing haemoglobin concentration and potential bleeding was addressed by administering packed red blood cells, cannula malposition was ruled out via TEE and an additional venous drainage cannula was placed in the superior vena cava. We hypothesize that persistent inadequate pump flow in this case resulted from a combination of sepsis-induced vasodilation, DIC, microcirculatory dysfunction, and endotheliopathy with capillary leakage [25], a hypothesis supported by autopsy findings.

Several case reports have documented successful use of ECPR in septic shock, often associated with septic-induced cardiomyopathy [26]-[28]. To our knowledge, this represents the first documented case of ECPR following IHCA in the context of OPSI. Notably, this case report is among the few documented instances of septic shock where ECPR was employed, ultimately culminating in a fatal outcome. We consider it important to report this case to contribute to the limited literature on this relatively rare intervention, particularly given its negative outcome. Publication of such cases is vital to counteract publication bias, which tends to favor successful outcomes. This might provide a more balanced understanding of the potential role and limitations of ECPR in septic patients.

4. Conclusions

This case highlights the ambiguity of the use of VA-ECMO or ECPR in patients with septic shock, raising important questions regarding the cost-effectiveness and overall utility in critically ill septic patients. While single fatal case reports can offer valuable insights, their main limitation lies in their inability to provide generalizable findings, underscoring the need for further prospective research to define patient subsets that might truly benefit from these invasive and resource-intensive therapies.

By reporting this case, we additionally aim to emphasize the need for clinicians to be vigilant and stay on top of prevention strategies post-splenectomy, because even despite prompt recognition and maximal therapeutic interventions, some lives can not be saved.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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