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Case Report
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Fulminant Pneumococcal Sepsis in the Emergency Department: A Case-Based Narrative Review of Overwhelming Post-Splenectomy Infection, Therapeutic Strategies, and Early Recognition

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Abstract

Overwhelming post-splenectomy infection (OPSI) is an aggressive and frequently fatal syndrome usually due to Streptococcus pneumoniae. It is a diagnostic dilemma to be addressed in an emergency based on nonspecific early presentation and aggressive course. This narrative review is based on a real-life clinical case and discusses pathophysiology, epidemiology, diagnosis, prognosis, and management of OPSI. A 68-year-old male with history of trauma splenectomy and no history of pneumococcal vaccination presented to the Emergency Department with fever, diarrhea and epigastric pain. Multiorgan failure (MOF) with coagulopathy due to pneumococcal sepsis developed within hours. This case emphasizes the need for early identification, empirical antibiotic management, and supportive care. Based on the last ten years of literature, the review will provide a useful perspective to emergency physicians on how to recognize OPSI early which will ultimately affect patient outcomes.

Keywords: Overwhelming Post-Splenectomy Infection, OPSI, Pneumococcal Sepsis, Streptococcus Pneumoniae, Splenectomy, Asplenia, Emergency Department, Disseminated Intravascular Coagulation, Case Report, Narrative Review, Critical Care, Sepsis Recognition, Unvaccinated Patient.

Introduction

OPSI is a rare but devastating syndrome that occurs in asplenic or functionally hyposplenic individuals, with an annual incidence of approximately 0.2 to 0.5 percent in adults and a fatality rate that can reach over 50 percent. A patient with Overwhelming Post-Splenectomy Infection (OPSI) may initially appear clinically stable: however, the condition can rapidly deteriorate and progress to fulminant sepsis. Individuals splenectomized or with hypersplenism exhibit a significantly compromised ability to mount an effective immune response, particularly against encapsulated pathogens.

Case Report

A 68-year-old man presented to the Emergency Department (ED) in the late afternoon complaining of fever, diffuse abdominal pain, and 2 days of diarrhea. His past medical history included a traumatic splenectomy in 2002, arteriopathy treated by valsartan, and prostate cancer treated surgically in 2008. He had received no post-splenectomy vaccination and had not previously received a pneumococcal vaccine (last vaccination performed 5 years before). On admission, he was hemodynamically stable with intact neurological status (GCS 15). Laboratory tests performed at the time of presentation revealed leukocytosis with severe neutrophilia, normal platelets and no evidence of any or-

gan failure. Upon admission the patient was febrile but hemodynamically stable.

His abdomen was soft on palpation, and no immediate signs of peritonism were noted. The ABCD assessment was within normal limits, and his Glasgow Coma Scale score was 15. During the subsequent twelve hours his condition became increasingly desperate. He became confused with psychomotor slowing and hypotensive with a decline in urine output to anuria. He had also experienced urinary incontinence. On physical examination, scattered petechiae and ecchymoses were observed over the thighs and conjunctivae, and there was evidence of peripheral hypoperfusion.

Blood testing had shown findings of acute kidney injury, lactic acidosis with lactate levels rapidly escalating to 14 mmol/L, a severe decrease in platelets, and a procalcitonin level of more than 110 ng/mL. Coagulation functions were deranged and urinalysis demonstrated massive hematuria and proteinuria.

Giving the clinical condition a comprehensive diagnostic workup, including blood cultures, urine analysis, CT scan and specialist consultation, was promptly initiated to identify the underlying source of infection. CT chest and abdomen demonstrated features of early pulmonary consolidation and renal hypoperfusion. Streptococcus pneumoniae was confirmed by a swift urinary antigen test.

The patient received aggressive crystalloid fluid resuscitation, vasopressor support, empiric broad spectrum antibiotics (piperacillin-tazobactam, meropenem, tigecycline), and transfusion of blood products; he developed disseminated intravascular coagulation and multiorgan failure within 24 hours of admission. There was a clinical presentation of OPSI (overwhelming post-splenectomy sepsis).

OPSI is challenging to diagnose in the ED as early symptoms are nonspecific and resemble those for respiratory infections. Fever, right-sided abdominal pain, and mild digestive symptoms are the only prevalent manifestations. The speed at which these transforms into hypotension, confusion, and multisystem involvement, is a very scary red flag. Laboratory findings are characterized by neutrophilic leukocytosis, increased levels of CRP, and procalcitonin, thrombocytopenia, and early signs of DIC. Pneumococcal urinary antigen testing provides rapid confirmation of infection; however clinical suspicion is required before diagnostic certainty.

Vital Signs

	25/06 19:31	25/06 23:49	26/06 02:32	26/06 07:36	26/06 09:31	26/06 09:50	26/06 09:53	26/06 10:12	26/06 12:10	26/06 13:40
HR	68	-	-	65	74	74	-	-	62	63
RR	68 (?)	-	-	-	-	-	-	-	14	14
BP max	106	-	-	117	105	115	118	110	109	165
BP min	66	-	-	78	62	68	70	75	70	78
SpO2	94	-	-	96	95	-	-	-	100	93
GCS	15	-	-	15	15	-	-	-	-	-
Temp °C	38,3	38,1	37,5	38,7	36,8	-	-	-	36	36

Figure 1: HR = Heart Rate; RR= Respiratory Rate; BP= Blood Pressure

Laboratory Tests

	25/06 19:33	**26/06 09:42**	**26/06 11:36**
RBC (4,5-6,0 x 10^6)	4,63	5,15	
WBC (4-11 x 10^3)	20.26	18,96	
Neutrophils (1,8-7 x 10^3)	18,7 (92,3%)	16,39 (86,4%)	
Lymphocytes % (20-50)	6,1	12,2	
Monocytes % (2-13)	0,8	0,6	
Eosinophils (0,1-0,7 x 10^3)	0,12	0,03	
Platelets (140-450 x 10^3)	176	27	
PT (seconds)	12.6	Clotted sample	
aPTT sec (25-37)	23	Clotted sample	
Potassium (3,5-5,1 mEq/L)	3,2	3,2	
Glucose (65-110 mg/dl)	94,1	-	
Creatinine (0,64-1,2 mg/dL)	1,12	2,29	
Total bilirubin (0,1-1,1 mg/dL)	1	1,3	
CRP (<0,5 mg/dL)	0,9	13,96	

Fibrinogen (200-400 mg/dL)	405	Clotted	
Sample			
LDH (<250 U/L)	267	699	
Clotted and slightly hemolyzed sample			
D-dimer (<500)	-	Clotted sample	
Urea (10-50 mg/dL)	45,3	66,9	
AST (5-45 UI/L)	22	101	
Procalcitonin (< 0,49 ng/mL)	-	110,95	

Figure 2: RBC = Red Blood Cells; WBC= White Blood Cells; PT= Prothrombin Time; aPTT = Activated Partial Thromboplastin Time; CRP= C-reactive protein; LDH = Lactate Dehydrogenase; AST = Aspartate Aminotransferase

Physiopathology

The spleen is rich in lymphoid tissue and splenic macrophages, which are essential for the phagocytosis of encapsulated microorganisms. By filtering approximately 10–15% of circulating blood volume per minute, the spleen plays a crucial role in the removal of senescent red blood cells and in maintaining immune homeostasis, due to its ability to link the innate and adaptive immune systems in the defense against infections.

In the absence of the spleen, the clearance of encapsulated pathogens is significantly impaired. Animal model studies have shown that splenectomy leads to the accumulation of polysaccharides in lymphoid tissues and a reduction in the number of memory B cells producing IgM, in decreased levels of cytokines such as IFN-γ and IL-4, which are essential for macrophage activation and antibody production. A deficiency in IFN-γ predisposes to infections by intracellular bacteria and viruses, while an IL-4 deficiency compromises B cell differentiation and survival. They can for this reason spread rapidly, resulting in a fulminant septic cascade with severe hemodynamic destabilization, DIC, and quickly progressing multi-organ failure [1-3].

Epidemiology

OPSI has a mortality rate of 50%-70%, with an annual incidence of approximately 0.23%. Although with early and aggressive medical intervention, the mortality rate can be reduced to around 10%. Therefore, timely recognition by emergency physicians is essential, enabling the prompt initiation of antibiotic therapy and the provision of appropriate supportive care.

The main causes of asplenia or hyposplenism include congenital conditions, trauma, hematologic diseases (such as sickle cell disease and thalassemia), advanced liver disease, ulcerative colitis, celiac disease, and systemic lupus erythematosus. Children under two years of age and patients splenectomized for hematologic diseases have a poorer prognosis compared to those splenectomized due to trauma, possibly due to the presence of accessory spleens or splenic implants that may provide partial immune function.

The most aggressive pathogens in asplenic patients are encapsulated bacteria, including Streptococcus pneumoniae, Haemophilus influenzae, and Neisseria meningitidis. Patients without a spleen have a 12 to 15-fold increased risk of developing invasive pneumococcal infections compared to the general population [4]

Clinical Presentation

Prodromal symptoms of OPSI may be mild and nonspecific, such as asthenia, nausea, and abdominal pain, followed by fever, myalgia, headache, vomiting, and eventually rash and petechiae. The clinical condition can rapidly deteriorate, progressing to multiorgan failure and death within 24–48 hours.

Management of OPSI in Emergency Department

First-line antibiotics: vancomycin plus ceftriaxone, dose-adjusted according to renal function. Vancomycin covers resistant pneumococci; ceftriaxone covers Gram-positive and Gram-negative bacteria such as Neisseria meningitidis and Haemophilus influenzae [5-8].

• Vancomycin 10-15 mg/Kg i.v q 12 if crCl> 60 mL/min (approzimates 1 g i.v q 12h for 70 Kg adult)
• PLUS
• Ceftriaxone 2 g i.v daily (children = 50 mg/Kg i.v q 12h) OR Cefotaxime 2 g i.v q 8h (children 25-50 mg/Kg i.v q 6h)
• Hemodynamic support: fluids, vasopressors, and intensive monitoring.
• IVIG: A dose of 0.4 g/kg/day for 3 days may be considered, despite limited evidence.

A 2021 Japanese study involving 69 OPSI cases showed a higher survival probability in patients treated with IVIG compared to controls: at 7 days, survival was 85% in the IVIG group versus 59.9% in the non-IVIG group. At 30 days, survival remained significantly higher—70% with IVIG versus 49% without. The efficacy of IVIG in terms of survival appears to be dose-dependent based on the amount of specific antibodies against causative bacteria. In addition to supplementing specific antibodies, IVIG can nonspecifically neutralize certain toxins and exert immunosuppressive effects on leukocytes, potentially preventing the late onset of immune energy [9].

Preventive strategies: Vaccinations

Preventive measures for OPSI are still the strongest weapon. Joint guidelines advise that all asplenic patients should be given pneumococcal conjugate and polysaccharide vaccines (PCV13 followed by PPSV23 (8 weeks later), then PPSV23 boosters every 5 years and, if needed, PCV13 revaccination), meningococcal vaccines (MenACWY vaccine administered in two doses 8–12 weeks apart, with subsequent boosters every 5 years.), and Haemophilus influenzae type b vaccine (Administration pre- or post-operatively depending on the type of splenectomy (elective vs. emergency).) . Immunity booster tactics and life-long report-

ing of splenectomised status are important, but there is evidence that both patients and providers fail to do so [10,11].

Our patient had not been appropriately vaccinated and there was no record of consideration for mitigation of infectious risk; this is an example of a predictable but preventable failure of care following splenectomy [12].

Prognosis

It is highly dependent on the initiation time of empiric antibiotic therapy and organ support. Survival is greatly reduced if treatment is delayed beyond the first few hours. Literature over the past 10 years reinforces the use of empiric third-generation cephalosporins or carbapenems, often in conjunction with vancomycin based on local resistance patterns. Supportive management is as important as it includes haemodynamic monitoring, platelets transfusion, plasma transfusion, renal replacement therapy and early ICU referral [13-16].

Conclusions

Pearls and Pitfalls for the EP

While the signs and symptoms of OPSI may be subtle, clinicians must remain suspicious of this condition when a patient who is known to have had prior splenectomy presents to the ED with vague or relatively minor symptoms such as fever, abdominal symptoms, or mental change. Not infrequently the transition is marked by rapid deterioration, confusion, petechiae, or evidence of coagulopathy. Rapidly worsening thrombocytopenia, creatinine, procalcitonin, and lactic acid elevations are further alerts stones. Empiric broad-spectrum antibiotics should never be held while waiting for microbiological confirmation. Start fluid resuscitation and observe for hemodynamic instability. Order a pneumococcal urinary antigen test, if possible, but keep in mind that the clinician's nose should take precedence over delayed diagnostics. Ask about past vaccinations, and on discharge make sure that there is a proper referral of the patient for follow-up at the infectious disease or primary care clinic. An alert, rapid and suspicious response can turn the tide in what could be fatal but avoidable emergency.

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