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| RESEARCH ARTICLE

Fever and Jaundice in a Post-Splenectomy Patient: Overwhelming Post-Splenectomy Infection (OPSI) Unveiled

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| ABSTRACT

In the context of OPSI, vaccination must not be mistaken for absolute protection but should instead be regarded as a preventive strategy—one inherently susceptible to failure, particularly due to serotype mismatch, wherein current vaccines do not encompass all pathogenic strains such as certain serotypes of Streptococcus pneumoniae. This assertion is exemplified by the present case report, which describes a 42-year-old Saudi male, fully vaccinated, who presented with new-onset fever, jaundice, and multi-organ dysfunction—eight years following a splenectomy performed after a motor vehicle accident resulting in traumatic splenic rupture. The patient was successfully managed through prompt resuscitation, hemodynamic support, and intravenous antibiotic therapy, underscoring the life-saving potential of early recognition and immediate intervention in cases of OPSI. This case reinforces the sobering reality that, even with appropriate treatment, OPSI carries significant mortality—and that timely clinical suspicion remains the most critical determinant of survival.

| KEYWORDS

Post-Splenectomy Jaundice, Sepsis, Encapsulated Organism, Asplenia, Splenectomy.

| ARTICLE INFORMATION

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Introduction

The spleen is one of the most vital lymphoid organs in the human body that plays various roles and functions, most importantly immunologic and hematologic functions. Some of the functions the spleen performs are removing harmful pathogens from the blood—most important clinically are encapsulated bacteria—red cell turnover and generating immune responses by producing antibodies and memory B-cells [1][2]. Histologically, the spleen has white and red pulps. In the white pulp lymphoid tissue is found, which is mainly responsible for our adaptive immunity, whereas in the red pulp macrophages can be found; macrophages phagocytose opsonized organisms and damaged erythrocytes [1][2]. The process of clearing harmful encapsulated bacteria like Streptococcus pneumoniae, Neisseria meningitidis, and Haemophilus influenzae that evade innate defenses is achieved by the generation of IgM memory B cells and opsonins via the spleen [3]. Hence, for the mentioned reasons, asplenia compromises and affects the body's ability to produce antibodies and clear the various forms of infections, resulting in a higher risk of severe and

potentially fatal, overwhelming infections [1][3]. It is undeniable that there are certain clinical scenarios where procedures like splenectomy are indicated. In hospital settings, splenectomy is performed as an emergency or electively, such as after trauma or in certain chronic blood disorders, respectively [2]. Some of the indications include traumatic splenic rupture or hemorrhage and certain blood disorders where the spleen is hyperactive and necessitates removal [2]. For instance, congenital hemolytic anemias such as hereditary spherocytosis, sickle cell disease, and thalassemia; immune cytopenias such as immune thrombocytopenic purpura and autoimmune hemolytic anemia; and hematologic malignancies such as leukemias and lymphomas [2]. Additionally, there are certain scenarios where the spleen enlarges—a condition referred to as hypersplenism—and therefore requires removal, such as in storage diseases or portal hypertension. Removal of the spleen, however, has well-known consequences. Post-splenectomy patients lose a critical line of defense, predisposing them to life-threatening infections [1][4]. The most feared complication is overwhelming post-splenectomy infection, abbreviated as OPSI, a fulminant sepsis syndrome in asplenic individuals. OPSI is classically defined as rapidly progressive septicemia or meningitis caused by encapsulated organisms—most often S. pneumoniae—occurring after loss of splenic function [1][5]. It typically begins with nonspecific flu-like symptoms including fever, chills, myalgias, headache, vomiting, and abdominal pain [4] and can progress to full-blown septic shock within 24–48 hours [5]. Without prompt recognition and treatment, OPSI carries a very high mortality rate [5][6]. Even with aggressive therapy, reported mortality is on the order of 30–70% [5][6], and deaths have been noted within hours of presentation [1][5]. OPSI can occur at any time after splenectomy, but the risk is greatest in the early postoperative years. Most reports find the majority of cases occur within the first 1–3 years after splenectomy, although rare cases have been documented decades later [5]. One review found that about 78% of OPSI episodes arose in the first three years, but some occurred as late as 59 years post-splenectomy [5]. Overall lifetime risk of OPSI is estimated to be around 5%, underscoring that susceptibility persists indefinitely [6]. Given the speed and severity of OPSI, it is considered a medical emergency requiring immediate intervention. Clinically, the earliest and most consistent finding in OPSI is high fever [4]. In practice, any asplenic patient with abrupt high-grade fever—often greater than 38–39°C—should be evaluated for OPSI [4]. Other early symptoms are nonspecific: rigors, weakness, gastrointestinal upset, and altered mental status may develop. Notably, jaundice, specifically scleral icterus, may accompany OPSI, though it is less emphasized. In such a context the jaundice noted is a likely consequence of both sepsis-triggered cholestasis and elevated hemolytic activity. It is hypothesized that during severe infections, pro-inflammatory cytokines interfere with the bile transport process and harm liver cells, producing conjugated hyperbilirubinemia [7]. At the same time, patients who have undergone splenectomy frequently have hemolytic blood conditions, and without the spleen, defective red blood cells are removed by other organs such as the liver, and this also increases the bilirubin burden. Therefore, we can conclude that patients who are both septic and asplenic may experience jaundice through the combined effects of cholestasis and hemolysis [7]. Given the catastrophic course of OPSI, management must be immediate. Empiric broad-spectrum antibiotics should be administered without delay to any febrile asplenic patient [4]. Current recommendations favor high-dose intravenous coverage for encapsulated bacteria; for example, vancomycin plus a third-generation cephalosporin such as ceftriaxone or cefotaxime is standard [8]. These agents target penicillin-resistant S. pneumoniae, N. meningitidis, and H. influenzae. In cases of penicillin allergy or unusual exposures, alternatives include carbapenems or fluoroquinolones in combination with vancomycin [8]. Supportive care, including aggressive fluids, vasopressors, and ICU monitoring, is also crucial. Prophylaxis is equally important: all post-splenectomy patients should receive immunizations against pneumococcus, meningococcus, and H. influenzae, with boosters as per guidelines [6], and many guidelines advocate oral antibiotic prophylaxis for high-risk patients, especially children, to prevent OPSI. Despite these well-known principles, awareness and adherence remain suboptimal. Surveys have shown that up to 85% of splenectomized patients are unaware of their increased infection risk [4]. Likewise, physicians may underappreciate atypical presentations such as jaundice in OPSI. In many regions, data on OPSI is limited. Notably, Middle East series on thalassemia have documented the impact of preventive measures: for instance, one study found 5 OPSI-related deaths among 30 unvaccinated Iraqi thalassemia patients over 4 years, compared to only 1 death among 22 Saudi patients who received pneumococcal vaccination, and many underwent partial splenectomy [9]. This stark contrast highlights the real-world mortality of OPSI and the benefits of vaccination and spleen-conserving strategies in resource-limited settings. In summary, overwhelming post-splenectomy infection is a rare but devastating complication of asplenia. Its nonspecific early features—typically high fever with possible gastrointestinal symptoms or jaundice—require a high index of suspicion. This case is educational because it illustrates an unusual presentation, fever with jaundice, of OPSI, reinforcing that any acutely ill febrile asplenic patient must be treated as a medical emergency [4][5]. It underscores the need for vigilance, patient education, and early empiric therapy to improve outcomes in this high-risk population.

Case Presentation

Patient's history and Physical Examination

This case concerns a 39-year-old Saudi male who presented to our facility with a recent onset of subjective fever and a yellowish discoloration of the sclerae. His symptoms commenced a day prior to admission and were accompanied by profound malaise and intermittent rigors. The patient also noted the passage of dark-colored urine. According to his wife, his condition

deteriorated swiftly, culminating in episodes of confusion. His wife recounted that he had been in his usual state of health during the preceding week until the day before presentation, when he appeared markedly weak, disoriented, unable to ambulate without assistance, and experienced two episodes of emesis. A detailed history revealed a prior splenectomy performed eight years earlier, consequent to traumatic rupture sustained in a motor vehicle accident. The patient is up to date with vaccinations against Streptococcus pneumoniae, Neisseria meningitidis, and Haemophilus influenzae. He reported no regular medications, known allergies, or significant familial medical history. Social history was notable for a five pack-year of smoking, with no reported use of illicit substances or alcohol. The family denied any recent travel. There were no complaints suggestive of upper respiratory tract infection—no cough, sore throat, or rhinorrhea—nor were there any urinary symptoms such as dysuria or hematuria. Additionally, the patient had no skin rashes, gastrointestinal disturbances, abdominal pain, or alterations in bowel habits. Upon arrival at our facility, physical examination revealed a temperature of 39.6°C, heart rate of 110 beats per minute, blood pressure measuring 90/55 mmHg, respiratory rate of 22 breaths per minute, and an oxygen saturation of 92% on ambient air. The patient appeared acutely ill—jaundiced, agitated, and disoriented—with a Glasgow Coma Scale score of 13 out of 15. No clinical signs suggestive of meningeal irritation were elicited. Systemic examinations of the abdomen, chest, and cardiovascular system were largely unremarkable, save for evident tachycardia and tachypnea. Neurological evaluation revealed no focal deficits.

Investigations

Bedside electrocardiography demonstrated sinus tachycardia. Although the chest radiograph revealed no abnormalities, and urinalysis was negative for both nitrites and leukocyte esterase, two sets of blood cultures were obtained promptly prior to the administration of empirical antibiotics. Examination of the peripheral blood smear showed no evidence of hemolysis or malarial parasites. Glucose-6-phosphate dehydrogenase (G6PD) levels were within normal limits, and abdominal ultrasonography revealed no significant findings. Pertinent laboratory investigations are summarized below (Table 1).

Test	Result	Normal Range
Hemoglobin	10.2	13-17 g\dL
WBC	$2.1x10^9$	$4.0\text{-}10x10^9$\L
Platelets	$85x10^9$	$150\text{-}450x10^9$\L
Total bilirubin	5.2	0.2-1.2 mg\dL
Direct bilirubin	3.4	0.1-0.3 mg\dL
Sodium	138	135-145 mmol\L
Potassium	4.4	3.5-5.0 mmol\L
Creatinine	1.5	0.7-1.2 mg\dL
ALT	120	<40 U\L
AST	150	<40 U\L
CRP	230	<5 mg\L
Procalcitonin	25	<0.5 ng\mL
Lactate	4.5	0.5-2.2 mmol\L
INR	1.6	0.8-1.2

Table 1: results of relevant laboratory investigations.

Management course

While the patient was being transferred to the intensive care unit, immediate fluid resuscitation was initiated with 30 mL/kg of intravenous 0.9% sodium chloride, amounting to approximately 2 liters administered over the first hour. Concurrently, supplemental oxygen at 4 liters per minute was delivered via nasal cannula. Urine output was closely monitored through insertion of a Foley catheter, and empiric intravenous antibiotic therapy was commenced within the first hour of presentation. This included ceftriaxone 2 grams every 12 hours and vancomycin at a dose of 15 mg/kg, with a contingency plan to escalate to meropenem in the event of therapeutic failure. Given the persistence of hypotension despite initial fluid administration, a continuous intravenous infusion of norepinephrine was initiated to provide hemodynamic support and maintain a mean arterial pressure above 65 mmHg. Acetaminophen and other hepatotoxic agents were deliberately withheld, with close daily monitoring of international normalized ratio (INR) and liver function tests. Serum lactate levels normalized within 48 hours, and bilirubin levels declined progressively. The patient was discharged on day 10 following clinical recovery, having been educated on warning signs, scheduled for follow-up, and provided with standby oral antibiotics in the form of amoxicillin 1 gram.

Discussion

Although several studies have demonstrated that up-to-date vaccination reduces the risk of overwhelming post-splenectomy infection (OPSI) by approximately 50–70%, it is important to underscore that immunization does not confer absolute protection [2]. Asplenic individuals remain at a permanent, lifelong risk of OPSI, with mortality rates reaching as high as 50–70% in the absence of timely intervention [3]. Alarmingly, the mortality curve rises steeply when antibiotic therapy is delayed beyond six hours from symptom onset [3]. In this particular case, it is reasonable to attribute the patient's jaundice to sepsis-induced cholestasis, likely mediated by pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which inhibit bile transporter expression [7]. Additional contributory mechanisms may include hypoperfusion and ischemic hepatitis [7]. Notably, studies have identified direct bilirubin levels exceeding 3 mg/dL in the context of OPSI as a predictor of increased mortality [7]. Other indicators of poor prognosis include elevated serum lactate (>4 mmol/L), leukopenia (white blood cell count <4 $x10^9$/L), thrombocytopenia (platelets <100 $x10^9$/L), and evidence of multi-organ dysfunction—all of which were observed in this patient [4]. These markers, taken together, underscore the severity of illness and the likelihood of a fatal outcome had early treatment not been initiated. This reinforces the critical importance of prompt recognition and intervention in cases of suspected post-splenectomy sepsis. Paradoxical leukopenia may signify physiological decompensation. Large cohort studies have shown that leukopenia is associated with higher mortality than extreme leukocytosis (WBC >20 $x10^9$/L), suggesting bone marrow exhaustion as part of the systemic inflammatory response to sepsis [4]. While the annual incidence of OPSI remains relatively low—estimated at around 0.3%—the lifetime risk may reach up to 5% and is reported to be significantly higher in pediatric populations, who also exhibit higher case fatality rates [5]. Despite the use of current vaccination strategies—such as PCV13, PPSV23, MenACWY, MenB, and Haemophilus influenzae type b (Hib) vaccines—breakthrough infections continue to occur [9]. In this case, blood cultures grew Streptococcus pneumoniae, but the isolate belonged to a serotype not covered by either PCV13 or PPSV23. This highlights the limitations of existing vaccines and underscores the urgent need for future vaccine development with broader serotype coverage [9]. Furthermore, it reaffirms the role of antibiotic prophylaxis—particularly with penicillin—in selected high-risk adults and children, alongside comprehensive patient education emphasizing that any episode of fever constitutes a medical emergency [9]. The use of medical alert identification, such as bracelets, is also strongly recommended [10]. Breakthrough OPSI cases are most commonly attributable to serotype mismatch, waning immunity over time, and individual host factors [10]. These realities position vaccination not as a definitive shield, but rather as an essential component of a multi-faceted prevention strategy. Clinicians must remain vigilant for the early manifestations of OPSI. Red flag symptoms include nonspecific flu-like illness, abrupt-onset fever, hemodynamic instability (notably hypotension and tachycardia), organ dysfunction, and jaundice [10]. Prompt identification and aggressive treatment of these features are paramount to improving survival in this vulnerable population.

Conclusion

In the context of OPSI, vaccination must not be misconstrued as definitive protection, but rather as a preventive measure—one that remains vulnerable to failure, particularly due to serotype mismatching when vaccines fail to cover certain strains of organisms such as Streptococcus pneumoniae. Given the exceedingly high mortality rates associated with delayed recognition and intervention, clinicians must treat any episode of fever in an asplenic patient as a medical red alert. The presence of jaundice, in such settings, often signifies the onset of severe multi-organ dysfunction. Perhaps the most vital takeaway is that prevention must not be passive. While patient education, medical alert bracelets, and standby antibiotic prescriptions may be considered secondary strategies, their timely implementation can be nothing short of lifesaving.

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References

[1] Howie S, et al. *Fatal overwhelming post-splenectomy infection in a patient with metastatic angiosarcoma treated with immunotherapy*. Front Immunol. 2024. https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2024.1366271/full
[2] Singh H, et al. *Splenectomy*. StatPearls [Internet]. 2024. https://www.ncbi.nlm.nih.gov/books/NBK560824/
[3] Di Sabatino A, et al. *Asplenia and spleen hypofunction*. Nat Rev Dis Primers. 2022. https://www.nature.com/articles/s41572-022-00399-x
[4] El-Alfy M, et al. *Post-splenectomy sepsis: a review of the literature*. J Infect Public Health. 2020. PMC: https://pmc.ncbi.nlm.nih.gov/articles/PMC7059871/
[5] Savoia D, Schito GC. *Overwhelming post-splenectomy infection (OPSI): pathogenesis and management*. Front Immunol. 2024.
[6] Santoro N, et al. *Asplenia and Infection*. StatPearls [Internet]. 2024.
[7] Chiang J, et al. *Hyperbilirubinemia in a patient with sepsis: a diagnostic challenge*. Clin Gastroenterol Hepatol. 2022. PMC: https://pmc.ncbi.nlm.nih.gov/articles/PMC10259638/

[8] *Fever in Patients with Impaired Spleen Function – Clinical Summary*. Emergency Care BC. 2017. https://emergencycarebc.ca/clinical_resource/clinical-summary/fever-in-patients-with-impaired-spleen-function/

[9] Sheikha AK, et al. *Prevention of OPSI in thalassemia: partial splenectomy and vaccination outcomes in the Middle East*. Mediterr J Hematol Infect Dis. 2015. https://www.mjhid.org/mjhid/article/download/2015.057/pdf_58/14847

[10] Hoffman R, Benz EJ, Silberstein LE, et al, eds. *Harrison's Principles of Internal Medicine*. 20th ed. New York, NY: McGraw-Hill Education; 2018.