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From Biopsy to Peritonitis: A Rare Case of Invasive Group a Streptococcus Pyogenes

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ABSTRACT

Group A Streptococcus pyogenes (GAS) is a pathogenic bacteria capable of a wide spectrum of illness to include peritonitis: a rare but life-threatening condition. Initially diagnosed with a small bowel obstruction, this patient ultimately proved to have GAS peritonitis from a uterine biopsy - a complication not previously described.

Introduction

Group A Streptococcus pyogenes (GAS) is a Gram-positive bacteria responsible for a wide range of pathology in humans [1]. These can range from mild infections such as pharyngitis or skin and soft tissue infections to more invasive pathologies including myonecrosis, necrotizing fasciitis, and streptococcal toxic shock syndrome [10]. This pathogen is responsible for an estimated 18.1 million individuals contracting infections worldwide each year [3]. Understanding the spectrum of disease caused by this pathogen is of critical importance, not only because invasive GAS infections are estimated to have a mortality rate approximating 20% [3,4], but the Center for Disease Control (CDC) reports a rising incidence of invasive disease from these bacteria over the past decade [5].

Acute peritonitis due to GAS is a very rare life-threatening condition not well described in the current literature. One study, reviewing diagnoses from hospital discharge records on pediatric patients in Finland, estimated that primary peritonitis represents 5% of invasive GAS infections.[6] Interestingly, the two most common diagnoses from invasive GAS infections in this study were skin and soft tissue infections (46%) and sepsis (26%) [6]. This case highlights an unusual presentation of invasive GAS infection as primary peritonitis in a 59-year-old female presenting to the emergency department with abdominal pain initially diagnosed as a small bowel obstruction. The aim of this case report is to raise diagnostic awareness of invasive GAS infections and provide strategies for therapeutic management. Prompt recognition of this atypical manifestation of invasive GAS is paramount to improving patient outcomes and guiding future medical management.

Case Description:

A 59-year-old female with hyperlipidemia, no prior abdominal surgical history, and family history of ovarian cancer presented to the emergency department with progressively worsening abdominal pain two days after a uterine biopsy had been performed to investigate new onset dysmenorrhea. These symptoms were accompanied by intractable nausea, multiple episodes of nonbloody nonbilious vomiting, intolerance of oral intake, generalized fatigue, and chills. Initial examination revealed hypotension, tachycardia, a moderately distended and rigid abdomen, with diffuse abdominal tenderness worst in the right lower quadrant, consistent with peritonitis. Notably, the patient was afebrile, and the remainder of her physical examination revealed no significant clinical findings. The patient's hypotension and tachycardia demonstrated minimal improvement despite appropriate intravenous fluid resuscitation. Laboratory studies revealed hypokalemia, hyponatremia, hypomagnesemia, acute kidney injury, and severe lactic acidosis. Leukocytosis was notably absent. A contrast-enhanced computed tomography (CT) scan of the abdomen and pelvis reported a small bowel obstruction at the ileocecal junction, with proximally dilated small bowel and a severely dilated stomach. The general surgery team was consulted, a nasogastric tube placed with 2.4 liters of immediate bilious output, and the patient was taken emergently to the operating room for an exploratory laparotomy.

Intraoperative findings included at least 1 liter of purulent fluid throughout the abdomen and pelvis, diffuse peritoneal inflammation, and bilateral hydrosalpinx with purulent fluid exuding from the right Fallopian tube. There was no mechanical bowel obstruction discovered. Intraoperative culture samples were obtained, gynecologic surgery was consulted, and a bilateral salpingo-oophorectomy was performed. Postoperatively, the patient was successfully extubated and transferred to the intensive care unit (ICU) for blood pressure support by vasopressor infusion and close clinical monitoring. Broad spectrum antibiotics were initiated with piperacillin-tazobactam and linezolid.

The intraoperative fluid culture grew Group A Streptococcus pyogenes on postoperative day 1, at which time the antibiotic regimen was narrowed to ceftriaxone and metronidazole based upon culture sensitivities and continued for a 14-day course. The patient was transferred to the general medicine floor after a successful wean of vasopressor support and appropriate clinical improvement, where she remained for a total hospital stay of 18 days. Her hospital course was complicated by postoperative ileus requiring nasogastric tube with trans-parenteral nutrition as well as a pelvic abscess requiring percutaneous drainage by interventional radiology. The patient was ultimately discharged with follow-up with her primary care provider as well as the surgical team.

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Discussion:

Invasive GAS infections are uncommon, and primary peritonitis from these bacteria is exceedingly rare. Literature review of the PubMed database revealed 37 case reports of GAS peritonitis from 2000 to 2025. Females represented approximately 87% of infections, and children represented 24% of these cases. The presumed etiology of infection in this case was translocation of bacteria following an endometrial biopsy, as no other risk factors were apparent in the patient's clinical history. The majority of the case reports describing primary GAS peritonitis find no clearly identifiable point of entry [7]. It is not clear from the literature review performed what percentage of total cases of primary peritonitis are caused by GAS infections. Additionally, peritonitis as a complication from uterine biopsy has not been described in the existing literature. Chronic diseases including diabetes, cardiovascular disease, alcohol use disorder, and human immunodeficiency virus (HIV) are suspected to increase the risk of invasive GAS infections [2,8], although these risk factors are notably absent in this case.

GAS has several known virulence factors proposed to contribute to more severe invasive infections. The M protein is a surface protein that contributes to the virulence of GAS by disrupting phagocytosis and contributes to a proinflammatory response through the activation of neutrophils, monocytes, and keratinocytes through several unique mechanisms once released from the cell wall [9]. The streptococcal pyrogenic erythrogenic exotoxin (SpeB) is another protein produced by GAS that promotes virulent activity. It functions by degrading numerous host proteins involved in immunity and structure, which facilitates invasive bacterial spreading and circumvention of host immune surveillance [9]. The previously mentioned proteins, including other novel proteins such as toxic shock syndrome toxin-1 (TSST-1), have the ability to behave as superantigens, which have marked immune activating modulation capabilities. Notably, this activating capability has been identified to be related to the interaction of antigen-presenting cells and T-cells, specifically when superantigens bind with the product of the MHC class II complex and the T-cell receptor [4]. A marked downstream cytokine induction is initiated once superantigens are bound to this complex, significantly contributing to the virulence of invasive GAS [4]. While this is not an exhaustive list of virulent proteins produced by this bacteria, it illustrates several pathogenic mechanisms related to severe disease.

For mild GAS infections such as pharyngitis, penicillin or amoxicillin are effective antibiotic regimens. As this patient presented in septic shock, broad spectrum antibiotics including piperacillin-tazobactam and linezolid were initiated. Once culture data confirmed the presence of GAS and reported culture sensitivities, antibiotic therapy was narrowed appropriately. Current antibiotic recommendations for GAS infections, compiled from 2012 Infectious Diseases Society of America (IDSA) guidelines for GAS pharyngitis and literature review for invasive GAS disease, are summarized in Table 1 [4].

Source control is essential to successful management of invasive GAS infections. One prior case report noted that intraperitoneal drainage alone was effective in achieving source control [10], although the cornerstone of management in invasive GAS infections remains surgical source control, which is reported in up to 88% of cases [11]. Exploratory laparotomy with bilateral salpingo-oophorectomy was performed to establish definitive source control in this patient. There is debate regarding the role of intravenous immunoglobulin (IVIG) therapy in invasive GAS infections; the proposed mechanism is the suppression of immune activation through the neutralization of circulating superantigens based on animal models, although several retrospective studies have shown no mortality benefit when using this therapy [12]. This patient had a favorable outcome following surgical source control with broad initial and subsequent targeted antibiotic therapy, without IVIG administration.

Conclusion

This case report describes a rare case of primary peritonitis due to Group A Streptococcus pyogenes. It emphasizes the importance of a broad differential diagnosis in patients presenting with shock physiology and peritonitis, and the critical importance of obtaining a thorough medical and surgical history in all patient encounters. Surgical source control and prompt antibiotic administration are critical to optimize patient outcomes under such circumstances.

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Table 1: Antibiotic and Adjunctive Therapies to Manage GAS Infections.

Antibiotic	Class	Mechanism of Action	Role in Treatment	Notes
Penicillin G	Beta-lactam	Inhibits cell wall synthesis	First-line for invasive GAS	Highly effective; no resistance in S. pyogenes
Cephalexin	Cephalosporin	Inhibits cell wall synthesis	For patients with penicillin allergy	Useful in mild disease with concomitant penicillin allergy
Clindamycin	Lincosamide	Inhibits 50S ribosomal subunit (protein synthesis)	Adjunctive therapy with penicillin	Suppresses toxin production; active in stationary phase; not affected by inoculum size
Vancomycin	Glycopeptide	Inhibits cell wall synthesis	For penicillin-allergic patients	Use if severe allergy to beta-lactams
Linezolid	Oxazolidinone	Inhibits protein synthesis	Alternative in severe beta-lactam allergy or toxin suppression	Also suppresses exotoxins; costly
IVIG (adjunctive)	Immunoglobulin	Neutralizes superantigens and toxins	Used in streptococcal toxic shock syndrome (STSS)	Not an antibiotic; may reduce mortality when used with antimicrobials

Conflict of Interest/Disclosures: None.

Disclaimer: The views and opinions expressed are those of the authors and do not necessarily reflect the official policy or position of the United States Navy, Department of Defense, or United States Government.

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