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Primary Group A *Streptococcus* Peritonitis from Intrauterine Device

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Background	Primary peritonitis (PP), defined as diffuse peritoneal inflammation without an identifiable intra-abdominal source, is an uncommon diagnosis, particularly in young, immunocompetent individuals. While various etiologies exist, Group A *Streptococcus* (GAS) as a causative agent is exceptionally rare, especially in the context of an intrauterine device (IUD). This report details a case of GAS PP in a young, healthy female with a recently inserted IUD, whose initial presentation mimicked more common causes of acute abdomen.
Summary	A previously healthy 23-year-old woman presented with acute-onset abdominal pain, vomiting, diarrhea, and fever. An IUD had been inserted several weeks prior. Initial empirical treatment for common etiologies such as urinary tract infection (UTI) and gastroenteritis proved ineffective, necessitating hospital admission for further investigation. Diagnostic laparoscopy, performed to evaluate for other causes of an acute abdomen, revealed frank peritonitis without an identifiable visceral source, thereby establishing the diagnosis of primary peritonitis. Despite broad-spectrum intravenous antibiotics, the patient's clinical condition worsened. Following removal of the IUD, her condition began to improve significantly. Subsequent microbiological cultures from both intraoperative peritoneal fluid and the IUD itself grew GAS.
Conclusion	Primary peritonitis is rare in young, healthy patients. Signs and symptoms may be subtle, but it can progress rapidly with critical complications such as sepsis, shock, and even death. GAS is an exceptionally rare cause of PP, especially in young, immunocompetent patients, and has seldom been associated with IUDs. This case report contributes to the existing knowledge of IUD-associated GAS PP by demonstrating that removal of an IUD once PP is suspected may result in recovery and should be strongly considered as empirical treatment in this patient population, even in the weeks, months, and years after IUD insertion. Furthermore, we recommend that any type of intrauterine or vaginal device should be removed empirically in patients presenting with an acute abdomen of unknown etiology.
Key Words	primary peritonitis; laparoscopy; intrauterine device; Group A *Streptococcus*; acute abdomen

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Case Description

A previously healthy 23-year-old female presented to the emergency department with a one-day history of progressively worsening, cramping lower abdominal pain associated with nausea, vomiting, diarrhea, and subjective fever. An intrauterine device (IUD) had been inserted approximately six weeks prior. Since IUD placement, she reported irregular menstrual cycles with intermittent spotting, but the remainder of her genitourinary review of systems was negative. On initial presentation, the patient was tachycardic but afebrile and otherwise hemodynamically stable. Abdominal examination revealed diffuse tenderness to palpation but was soft, non-peritonitic, and without palpable masses. Initial laboratory studies demonstrated a neutrophilic leukocytosis and urinalysis findings consistent with a urinary tract infection (UTI). Concurrently, a transvaginal ultrasound revealed a benign-appearing left ovarian cyst, a small amount of free fluid in the cul-de-sac, and a correctly positioned IUD. Based on these findings, the patient was diagnosed with gastroenteritis and UTI and was discharged home on nitrofurantoin.

The following day, the patient re-presented to the emergency department with new-onset dyspnea refractory to her home asthma inhalers. While her diffuse abdominal pain had transiently improved, she now reported worsening pain localized to the right lower quadrant, accompanied by an escalation of her constitutional and gastrointestinal symptoms. Repeat complete blood count (CBC) and urinalysis were within normal limits. A computed tomography (CT) scan of the abdomen and pelvis demonstrated a normal to slightly prominent appendix containing air and fluid within its lumen but without significant appendiceal wall inflammation or periappendiceal fat stranding. Trace free fluid was noted in Morison's pouch and the cul-de-sac. Given her escalating symptoms and the unclear etiology of her condition, the patient was admitted to the hospital for further evaluation and management. Subsequent laboratory workup revealed new-onset lactic acidosis and bandemia without significant leukocytosis. Other blood counts and chemistries were largely unremarkable. Empiric broad-spectrum intravenous antibiotics (piperacillin-tazobactam and vancomycin) were initiated. An obstetrics and gynecology consultation did not identify a clear gynecological source for her symptoms. Evaluation by the emergency general surgery service revealed rebound tenderness in the right lower quadrant and a positive Rovsing sign, prompting urgent diagnostic laparoscopy for appendicitis versus other intra-abdominal pathology.

Upon laparoscopic entry into the abdomen, diffuse serositis was immediately apparent, marked by a large volume of purulent fluid and extensive fibrinous exudate overlying the uterus, fallopian tubes, ovaries, sigmoid colon, and portions of the small bowel most pronounced in the right lower quadrant. The distal small bowel was inflamed and mildly distended. The appendix exhibited mild periappendicitis without evidence of perforation, and an appendectomy was performed. The previously identified left ovarian cyst was visualized and benign in appearance. The remainder of the intra-abdominal organs appeared normal. A surgical drain was placed in the right lower quadrant prior to closure.

During the first postoperative day, the patient demonstrated initial clinical and laboratory improvement. At this time, despite a normal pelvic examination and absence of urinary symptoms, the urine culture obtained during her initial emergency department visit returned positive for Group A Streptococcus (GAS). Over the ensuing few days of her hospitalization, the patient's condition slowly deteriorated with worsening abdominal pain, recurrent emesis, dyspnea, intermittent fevers up to 39.4°C (103°F), persistent leukocytosis, and increasingly purulent drain output. Three days after her surgery, with a clearly deteriorating clinical state and persistent signs of intra-abdominal sepsis, her IUD was suspected as a potential nidus of infection and was thus removed.

Over the next few days, the patient then began to demonstrate gradual and sustained symptomatic and clinical improvement. Histopathological examination of the appendix revealed periappendicitis with acute inflammation confined to the serosa, without significant acute inflammation within the appendiceal wall itself. Crucially, intraoperative peritoneal fluid cultures and cultures obtained from the extracted IUD grew GAS. Her antibiotic regimen was briefly switched from piperacillin-tazobactam to amoxicillin and metronidazole due to concerns for elevating liver function tests during her hospital stay, but was ultimately reverted to piperacillin-tazobactam due to recurrence of vomiting and abdominal pain. Eight days after her operation and four days after IUD extraction, the patient was deemed medically stable and was discharged home on oral amoxicillin-clavulanate, with clinic follow-up arranged with infectious disease for evaluation following completion of her antibiotic regimen.

Discussion

Primary peritonitis, also referred to as spontaneous bacterial peritonitis in certain contexts (the latter typically implying pre-existing ascites), is defined by diffuse inflammation of the peritoneal cavity without an identifiable intra-abdominal source of infection. Common causative organisms include Escherichia coli, Klebsiella pneumoniae, and Streptococcus pneumoniae.[1] Group A Streptococcus, or Streptococcus pyogenes, is an exceptionally rare cause of PP, particularly in young, immunocompetent individuals. A review of existing literature revealed fewer than 70 reported cases, with limited comprehensive reviews as of 2021.[2] GAS infections are more typically associated with pharyngitis, skin and soft tissue infections, or, in the gynecological context, pelvic inflammatory disease (PID) and acute complications following gynecological procedures, including IUD insertion or removal. This association with recent procedures can make diagnosis challenging when symptoms arise weeks later, as in our patient whose IUD was placed six weeks prior.[3] Although rare, primary GAS peritonitis can present insidiously and progress rapidly to life-threatening sepsis and shock.[1] Epidemiologically, most reported cases of GAS PP occur in female patients in an approximate ratio of 4:1 compared to males, with the presumed pathogenesis most often attributed to an ascending genitourinary tract infection.[1] Other less common proposed routes of GAS transmission leading to PP involve orogenital contact and complications of surgical abortions.[4] Our literature search identified only one other case report of IUD-associated GAS PP presenting beyond the acute post-insertion period (i.e., >2 weeks) and in this case, manifesting years after IUD placement.[5] There has also been a reported case of GAS PP in a young female utilizing a menstrual cup.[6] It is noteworthy that PP in the setting of concurrent IUD use has been associated with other pathogens, including Streptococcus pneumoniae and Actinomyces species.[7,8] Most reported cases of IUD-associated GAS infections are characterized by sepsis, shock, or streptococcal toxic shock syndrome (STSS) occurring within mere days of IUD insertion.[3,10,11]

Nonspecific findings such as symptomatic dyspnea, pleural effusions, and transaminitis, as noted in other case reports of PP, were found to be present in our patient and contribute to an increasingly murky clinical picture.[1] Prompt diagnosis of GAS PP is imperative, as systemic infection can rapidly escalate to severe sepsis, shock, and mortality.[5,9,10] In our patient, the diagnosis of PP was established intraoperatively during diagnostic laparoscopy. This is consistent with many reports in the literature describing PP as mimicking more common pathologies such as enteritis and appendicitis, ultimately leading to definitive diagnosis made via surgical exploration and microbiological cultures.[5] In other instances, diagnosis of PP has been established non-operatively through a combination of abdominal imaging (to exclude secondary causes) and analysis of peritoneal fluid obtained via paracentesis.[11]

The decision to remove the IUD in our patient was delayed, largely due to the atypical nature of her disease course and initial unremarkable gynecological workup, which did not strongly implicate an ascending infection. Furthermore, intraoperative findings did not definitively point to the IUD as the source. The early positive urine culture for GAS was considered unusual and was thought to be related to contamination or transient bacteruria secondary to severe intra-abdominal sepsis rather than a primary urinary source. However, as the patient's condition deteriorated despite broad-spectrum antibiotics and surgical drainage, the IUD was increasingly suspected as a potential source of infection. It was therefore removed, even before definitive culture results from the implant were available. This action proved pivotal, as the patient began to show sustained improvement immediately following IUD extraction. While the patient had been receiving appropriate antibiotic coverage for GAS, the definitive management in this case was establishing source control via IUD extraction. There is a paucity of literature regarding antibiotic regimens for GAS PP with existing recommendations being somewhat controversial. However, the general consensus supports penicillin for sensitive strains in uncomplicated infections and broad-spectrum antibiotics as the mainstay for patients presenting with sepsis.[1,12]

It remains challenging to definitively ascertain whether the IUD served as the primary source of her infection (e.g., introduced during insertion or colonized thereafter) or rather became a secondary nidus for colonization. Regardless of the initial mechanism, its removal resulted in prompt recovery. This raises an important clinical question: should all patients with IUDs or other indwelling gynecological devices who present with PP have these devices removed empirically? Current literature and guidelines which tackle this question are lacking at best. In an analogous disease, PID, current recommendations are to consider IUD removal if there is no clinical improvement after 48–72

hours of appropriate antibiotic therapy, although studies have not consistently shown a difference in treatment outcomes between women who retained their IUD and those who had it removed.[13]

Conclusion

Although existing literature regarding the management of primary peritonitis in the setting of concurrent IUD use is limited and in some instances, controversial, this case proposes that early removal should be strongly considered in female patients presenting with a constellation of symptoms indicative of PP, particularly in the acute and subacute post-placement timeframe. Empiric IUD extraction may expedite recovery by eliminating a primary or secondary source of infection, even if the IUD was not recently placed.

Lessons Learned

Primary peritonitis can manifest with ambiguous clinical features, often leading to diagnostic delays and potentially severe sequelae if not promptly addressed. Therefore, PP should be included in the differential diagnosis for any patient presenting with signs of sepsis and a constellation of nonspecific symptoms. Management should include prompt initiation of broad-spectrum antibiotics and a comprehensive diagnostic workup, which may also necessitate operative intervention. Importantly, this case highlights that intra-abdominal infections related to intrauterine devices, including PP, can occur well beyond the acute post-insertion period. Consequently, clinicians should maintain a high index of suspicion for IUD-associated PP in any appropriate female patient presenting with unexplained acute abdominal symptoms and maintain a low threshold for IUD removal, even in the absence of localizing genitourinary symptoms or definitive laboratory evidence directly implicating the device early in the clinical course.

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