CASE REPORT

Intrauterine device infection causing concomitant streptococcal toxic shock syndrome and pelvic abscess with *Actinomyces odontolyticus* bacteraemia

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SUMMARY

Intrauterine devices (IUDs) are rarely associated with serious infections. We report an unusual concomitant infection of group A *Streptococcus* (GAS) causing toxic shock syndrome and pelvic abscess with *Actinomyces odontolyticus* associated with an IUD in a healthy 50-year-old patient. The IUD was subsequently removed and the patient recovered on the appropriate antibiotics. This case highlights the importance of clinicians' high index of suspicion of an IUD infection and prompt removal of the infected foreign body to obtain source control.

BACKGROUND

The use of intrauterine devices (IUDs) has been associated with several serious infections, including invasive group A streptococcal disease (GAS)¹ and toxic shock syndrome (TSS), due to *Staphylococcus* and *Streptococcus* spp.,^{2–4} as well as to *Actinomyces* infections.^{5–8} Although TSS was originally described in children in the late 1970s, the syndrome gained national attention when gynaecological causes began to affect healthy young women.⁹ The association between the use of an absorbent tampon and TSS has since been well-described in the literature. IUD use has also been associated with TSS and septic shock in rare cases.^{1–4 10–13} Early recognition of the syndrome for prompt treatment is crucial in preventing fatality, as described in several of the IUD-associated case reports.^{2 4} Pelvic actinomycosis is another rare but serious complication associated with long-term use of IUDs. *Actinomyces* organisms often colonise the genital tract and can cause polymicrobial pelvic abscesses or mass-like structures, which can be mistaken for malignancy.¹⁴ We describe an unusual case of concomitant GAS TSS and *Actinomyces odontolyticus* and *Streptococcus sanguinis* bacteraemia associated with pelvic abscesses and presumed septic emboli in an immunocompetent host.

CASE PRESENTATION

A 50-year-old previously healthy woman with a medical history of hypertension presented with generalised weakness, nausea, vomiting and diarrhoea. She had been in her usual state of health until 2 days prior to presentation, when she first developed generalised malaise with nausea and vomiting, followed by watery diarrhoea. On the day of presentation, she became intolerant to oral intake due to severe nausea. She denied any sick contacts, recent antibiotic use, travel or unusual

foods. She also denied any history of inflammatory bowel disease.

In the emergency department, she was found to be hypotensive with a systolic blood pressure of 70 mm Hg and a temperature of 38.3°C (100.9°F). She was given aggressive fluid resuscitation with 4 L of normal saline, with minimal clinical improvement. A central line was placed and norepinephrine started for blood pressure support. She also received an additional litre of fluids before being admitted to the medical intensive care unit (ICU) for further diagnostic work up and resuscitation.

INVESTIGATIONS

The patient's initial laboratory results were remarkable for leucocytosis with white cell count (WCC) count of $12.1 \times 10^9/L$, 33% bandemia, lactic acid of 7.7 mmol/L and acute kidney injury with creatinine of 229 µmol/L from her baseline of normal renal function. An abdominal and pelvic CT scan without intravenous contrast showed proctitis and the presence of an IUD. Admission urinalysis, blood cultures, liver enzymes, chest imaging, respiratory viral studies and stool studies were unrevealing for a source.

DIFFERENTIAL DIAGNOSIS

The initial working diagnosis was shock, secondary to likely mixed hypovolemic and septic shock, from an unknown source. Given that the only remarkable findings on the CT scan of the abdomen and pelvis were proctitis, and the presence of an IUD, the patient was initially given intravenous ciprofloxacin 400 mg and intravenous metronidazole 500 mg, which was then broadened to intravenous aztreonam 1 g every 8 h (the patient has penicillin allergy) and intravenous metronidazole 500 mg every 6 h on day 2 in the medical ICU, for a presumable enteric or pelvic source. Proctoscopy on day 3 showed only mild erythaema, thought not to have caused the septic shock. Further inquiry revealed that this patient had no gastrointestinal (GI) symptoms until 24 h prior to admission, and history of neither inflammatory bowel disease, proctitis nor other GI diseases over her lifetime to suggest a GI source for her initial presentation. Therefore, although her initial infectious work up was remarkable for proctitis on the CT scan of the abdomen and the pelvis, this was determined to be of little clinical significance by the treating physicians. Intravenous vancomycin was added as the

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patient remained in shock with no convincing source of infection. The patient's IUD was removed on day 4 due to the lack of an alternative infectious source. The IUD removal was uncomplicated. She endorsed having a non-copper (hormonal) IUD placed 5 years prior without any complications. A thorough gynaecological history revealed that the patient was postmenopausal, with her last menstrual period at the time of the IUD insertion 5 years earlier. She denied any history of sexually transmitted infections, and was only sexually active with her husband. She also denied any vaginal douching or tampon use (for over 5 years) preceding her admission.

The patient progressed to acute liver injury with total bilirubin of 34.2 $\mu\text{mol/L}$ (direct 20.5 $\mu\text{mol/L}$), alkaline phosphatase that peaked at 2.7 $\mu\text{kat/L}$, γ -glutamyl transpeptidase of 1.9 $\mu\text{kat/L}$, international normalised ratio of 1.5, as well as anuric renal failure requiring dialysis on hospital day 4. On day 5, her WCC peaked at $40.0 \times 10^9/\text{L}$. A subsequent chest–abdomen–pelvis CT scan with intravenous contrast showed scattered opacities concerning for septic emboli (figure 1), as well as an evolving presacral fluid collection without rim enhancement. One of three blood cultures from day 4 showed Gram-positive cocci and Gram-positive rods on Gram stain, prompting a transthoracic echocardiogram, which showed no vegetations. On day 6, the IUD culture grew GAS (*Streptococcus pyogenes*), sensitive to ampicillin, ceftriaxone, clindamycin and levofloxacin. Eventually, the patient's blood culture from day 4 was finalised as *Streptococcus sanguinis/gordonii*, sensitive to ceftriaxone, clindamycin and erythromycin; and *A. odontolyticus*, sensitive to ampicillin/sulbactam, penicillin, piperacillin/tazobactam and intermediate to metronidazole, identified using the automated VITEK 2 system (bioMérieux, La Balme les Grottes, France). A subsequent CT of the oral cavity revealed no dental caries, no abscess and no osteomyelitis. On examination, the patient appeared to have very good oral hygiene with no visible caries, no gingivitis and no dental abscesses. The patient denied any history of dental procedures within the preceding 12 months, and suggested to the physicians caring for her during her admission that she was actually due to follow-up with her dentist for an annual visit.

TREATMENT

Once the blood cultures were finalised, the patient's antibiotics were changed to intravenous clindamycin 600 mg every 8 h and intravenous ampicillin-sulbactam 3 g every 6 h, with the concern of TSS and pelvic actinomycosis, and to cover for *Streptococcus sanguinis* bacteraemia. Her renal function recovered and dialysis was discontinued on day 8. A repeat CT scan of the chest, abdomen and pelvis with intravenous contrast on day 12 showed

Figure 1 Scattered opacities in lungs bilaterally, concerning for septic emboli.

Figure 2 Rim-enhancing pelvic abscess.

resolving pulmonary opacities and a growing large rim-enhancing pelvic abscess (figure 2), which was subsequently drained, sent for culture and had no growth after 5 days. Unfortunately, the patient's pelvic fluid culture was not held beyond 5 days to monitor for growth of *Actinomyces* spp or other anaerobes due to delayed involvement of the infectious disease consultant and a low initial index of suspicion from the primary team for this infection. Ampicillin-sulbactam was chosen over penicillin therapy alone in order to provide additional anaerobic coverage for anaerobes that might not have been isolated from the patient's pelvic fluid within 5 days. Another concern was the unclear relevance of the patient's proctitis on initial imaging, which might have implicated Gram-negative organisms commonly found within the distal colon. Likewise, no tissue samples were taken to evaluate for sulfur granules on histopathology. The patient eventually underwent a transoesophageal echocardiogram, which showed no vegetations.

OUTCOME AND FOLLOW-UP

The patient recovered with an extended course of intravenous antibiotics and her fevers resolved prior to discharge. She was discharged on day 18, with a plan to treat her for a total of 3 weeks with intravenous ampicillin/sulbactam 3 g every 6 h, followed by oral penicillin 500 mg every 6 h for another 6–12 months to treat the pelvic actinomycosis. The patient's symptoms continued to improve in clinic follow-up, with good tolerance to oral penicillin. While she did not develop desquamation during her hospital stay, she eventually experienced desquamation of the skin on her hands and feet as well as hair loss approximately 2 weeks after discharge, which was only noted during her follow-up visits with her infectious disease doctor. The patient's abdominal pain completely resolved, and her kidney and liver function completely recovered within 2 months of hospital discharge. A repeat CT scan of the abdomen and pelvis showed improvement in the size of the previously found pelvic fluid collection with some associated rim enhancement, and a new rim-enhancing fluid collection in the right lower quadrant, concerning for a persistent nidus for infection. The patient continues to be followed in the infectious disease clinic for close clinical monitoring and remains on oral penicillin for treatment of the pelvic fluid collection due to pelvic actinomycosis.

DISCUSSION

Infections associated with an IUD can rarely present as severe manifestations such as TSS or pelvic actinomycosis. Both TSS and pelvic actinomycosis can be challenging to diagnose.

TSS is associated with protean clinical manifestations that can be difficult for providers to recognise. It is a syndrome of acute onset characterised by fever, hypotension and variable multi-organ involvement, often accompanied by vomiting and diarrhoea due to GI mucosal damage. GAS (*Streptococcus pyogenes*) and *Staphylococcus aureus* are frequently associated with TSS. Different diagnostic criteria have been developed by the Centers for Disease Control and Prevention (CDC) to distinguish GAS (box 1)¹⁵ from non-GAS TSS (box 2).¹⁶ While rash is a necessary criterion for a confirmed non-streptococcal TSS case, only

Box 1 Streptococcal toxic-shock syndrome (*Streptococcus pyogenes*) 2010 Case Definition by the Centers for Disease Control and Prevention (CDC)¹⁵

Clinical criteria

An illness with the following clinical manifestations*

- ▶ Hypotension defined by a systolic blood pressure ≤ 90 mm Hg for adults or less than the 50th centile by age for children aged <16 years
- ▶ Multiorgan involvement characterised by two or more of the following
 - Renal impairment: creatinine ≥ 2 mg/dL (≥ 177 μ mol/L) for adults or greater than or equal to twice the upper limit of normal for age. In patients with pre-existing renal disease, a greater than twofold elevation over the baseline level
 - Coagulopathy: platelets $\leq 100\,000/\text{mm}^3$ ($\leq 100 \times 10^6/\text{L}$) or disseminated intravascular coagulation, defined by prolonged clotting times, low fibrinogen level and the presence of fibrin degradation products
 - Liver involvement: alanine aminotransferase, aspartate aminotransferase, or total bilirubin levels greater than or equal to twice the upper limit of normal for the patient's age. In patients with pre-existing liver disease, a greater than twofold increase over the baseline level
 - Acute respiratory distress syndrome: defined by acute onset of diffuse pulmonary infiltrates and hypoxaemia in the absence of cardiac failure or by evidence of diffuse capillary leak manifested by acute onset of generalised oedema, or pleural or peritoneal effusions with hypoalbuminaemia
 - A generalised erythematous macular rash that may desquamate
 - Soft-tissue necrosis, including necrotising fasciitis or myositis, or gangrene

*Clinical manifestations do not need to be detected within the first 48 h of hospitalisation or illness, as specified in the 1996 case definition. The specification of the 48 h time constraint was for purposes of assessing whether the case was considered nosocomial, and not whether it was a case or not

- ▶ Isolation of group A *Streptococcus*

Case classification

Probable: A case that meets the clinical case definition in the absence of another identified aetiology for the illness and with isolation of group A *Streptococcus* from a non-sterile site

Confirmed: A case that meets the clinical case definition and with isolation of group A *Streptococcus* from a normally sterile site (eg, blood or cerebrospinal fluid or, less commonly, joint, pleural, or pericardial fluid)

Box 2 Toxic shock syndrome (other than streptococcal) 2011 Case Definition by the Centers for Disease Control and Prevention (CDC)¹⁶

Clinical criteria

- ▶ Fever: temperature $\geq 102.0^\circ\text{F}$ ($\geq 38.9^\circ\text{C}$)
- ▶ Rash: diffuse macular erythroderma
- ▶ Desquamation: 1–2 weeks after onset of rash
- ▶ Hypotension: systolic blood pressure ≤ 90 mm Hg for adults or less than 50th centile by age for children aged <16 years
- ▶ Multisystem involvement (3 or more of the following organ systems)
 - Gastrointestinal: vomiting or diarrhoea at onset of illness
 - Muscular: severe myalgia or creatine kinase level at least twice the upper limit of normal
 - Mucous membrane: vaginal, oropharyngeal, or conjunctival hyperaemia
 - Renal: blood urea nitrogen or creatinine at least twice the upper limit of normal for laboratory or urinary sediment with pyuria (≥ 5 leukocytes per high-power field) in the absence of urinary tract infection
 - Hepatic: total bilirubin, alanine aminotransferase enzyme, or aspartate aminotransferase enzyme levels at least twice the upper limit of normal for laboratory
 - Haematological: platelets $<100\,000/\text{mm}^3$
 - Central nervous system: disorientation or alterations in consciousness without focal neurological signs when fever and hypotension are absent

Laboratory Criteria for Diagnosis

Negative results on the following tests, if obtained

- ▶ Blood or cerebrospinal fluid cultures blood culture may be positive for *Staphylococcus aureus*
- ▶ Negative serologies for Rocky Mountain spotted fever, leptospirosis or measles

Case classification

Probable: A case that meets the laboratory criteria and in which four of the five clinical criteria described above are present

Confirmed: A case that meets the laboratory criteria and in which all five of the clinical criteria described above are present, including desquamation, unless the patient dies before desquamation occurs

about 10% of the cases of GAS TSS develop a diffuse erythematous rash, followed by desquamation,¹⁷ making diagnosis a challenge.

Our patient, even without the rash, was a definitive case for GAS TSS, based on diagnostic criteria established by the CDC:¹⁵ isolation of GAS from her IUD (normally a sterile site), hypotension (systolic blood pressure ≤ 90 mm Hg), and renal and liver dysfunction.

Since the withdrawal of several highly absorbent tampons from the market in the early 1980s due to their association with TSS,⁹ the incidence of TSS has dramatically decreased.² We performed a thorough review of the existing literature, which revealed several cases of septic shock and a few cases of TSS associated with IUD use. Agnew and Pritchard¹⁰ described possibly the first case of septic shock caused by an IUD in 1966. Klug *et al*² described a case of fatal TSS due to an IUD infected with *Staphylococcus aureus*, and Cho and Fernando⁴ described the first confirmed fatal case of streptococcal TSS associated with an IUD. Of note, the patient referenced in the

Cho and Fernando case report presented without a rash, similar to our patient's presentation. Similarly, Gisser *et al*¹ reported a case of streptococcal TSS from an IUD that became colonised via oral sex. The patient described by Gisser *et al* ultimately had two exploratory laparotomies, a bilateral salpingectomy and a total abdominal hysterectomy due to the severity of her infection. Furthermore, Venkataramanasetty *et al*¹⁸ reported the first case of GAS causing TSS after insertion of a copper IUD, which resulted in an emergent total abdominal hysterectomy with bilateral salpingectomy. Other serious complications after insertion of an IUD have been described as well, including severe adnexitis,¹⁹ peritonitis and salpingitis.²⁰ One case, described by Zuckerman and Stubblefield,¹¹ involved *Escherichia coli* septicaemia associated with a Dalkon Shield IUD resulting in septic abortion. Thereafter, several additional cases of sepsis, including a few deaths, were reported, leading to the eventual withdrawal of the Dalkon device in the US market in 1974.²¹ Additionally, one case described staphylococcal septicaemia after insertion of the Lippes Loop device,¹² and another a case of meningococcal septic shock associated with an IUD.¹³

The mechanism by which IUDs cause invasive infections remains unknown. Some clinicians have hypothesised that the vaginal string appendage of the IUD may break the protective barrier of the cervical mucosa, serving as a portal of entry for commensal bacteria in that region.²² Others have postulated that the minor trauma caused by the presence of an IUD results in disruption of the cervical mucosa, allowing for bacterial infection and systemic spread.

TSS is associated with other gynaecological infections as well, even in the absence of IUD use. One recent paper by Hikone *et al*²³ described two cases of streptococcal TSS secondary to GAS vaginitis. Lee *et al*²⁴ described an association between TSS and barrier contraceptives, while Wilson²⁵ described a case of TSS associated with diaphragm use.

Treatment for TSS requires prompt recognition of the syndrome, as not only is source control critical, but initiating medications with the antitoxin effect is paramount as well. TSS is a condition mediated by superantigens via the toxins produced by the bacteria.²⁶ Studies have demonstrated that clindamycin inhibits transcription of streptococcal pyrogenic exotoxin superantigen production,^{27–28} and may lead to better survival rates even with delayed administration, in animal studies.²⁹ Even though clindamycin is widely recommended as an adjunctive treatment to a β -lactam antibiotic for TSS,³⁰ as it had been shown to reduce mortality,³¹ it is still often underused.³¹ Our patient made rapid clinical improvement immediately after the addition of high-dose intravenous clindamycin, which we feel is not a coincidence but rather a testament to the potent toxin inhibition of this drug.

In addition to TSS related to infected IUDs, abdominopelvic actinomycosis is another dreaded infectious complication associated with IUD use. *A. israelii*, described for the first time by Israel in 1978,³² is a filamentous and branching commensal Gram-positive anaerobic bacterium.³² It is a rare disease that is challenging to diagnose because as fastidious anaerobes, *Actinomyces* spp are difficult to grow. It is uncommon to isolate *Actinomyces* spp in the blood, as seen in our case, due to the fastidious nature of this organism. The most frequent locations of infections are the head and neck (50–60%), and thoracic areas (20–25%).^{32–33} While abdominopelvic actinomycosis is rare, its prevalence has been raising with the increasing use of IUDs,^{6–7} first reported in 1973.⁸ In fact, it has been reported that the bacterium is identified in 8–20%

of IUD carriers.³⁴ Prolonged IUD use (over 4 years) has been associated with pseudo tumour pelvic actinomycosis³⁵ and abdominal wall actinomycosis mimicking peritoneal carcinomatosis.³⁶

The evolution of multiple pelvic abscesses due to actinomycosis from an infected IUD is very rare. Only two other case reports described this clinical phenomenon, the first associated with prolonged use of an IUD causing an abdominal wall abscess and secondary intra-abdominal abscesses.³⁷ The other by Galata *et al*¹⁴ reported a case of abdominopelvic actinomycosis requiring an urgent exploratory laparotomy for multiple abscesses secondary to a neglected IUD. Unfortunately, preoperative diagnosis of actinomycosis is very difficult. In fact, only 17% of pelvic actinomycosis are diagnosed preoperatively,³⁸ requiring clinicians' heightened suspicion to start empirical treatment with antibiotics for cure.³⁶

Of note, infections due to *Actinomyces* spp are often polymicrobial due to the ability of this organism to produce biofilms, as was the case in our patient. We suspect that the IUD was the most likely source of the polymicrobial infection in our patient with the evidence of rim-enhancing pelvic abscesses and, as a CT scan of the oral cavity did not reveal any dental caries, nor abscess, nor osteomyelitis. Removal of the infected device may be critical to establishing source control in such cases, as an in vitro study has shown that *Actinomyces* spp is capable of developing a biofilm over copper surfaces in IUDs.³⁹

Finally, in addition to pelvic actinomycosis, our patient had radiographic imaging suggestive of septic emboli from bacteraemia, possibly due to either *A. odontolyticus* and/or *Streptococcus sanguinis/gordonii*. In the literature, there have been case reports of *Actinomyces* spp causing infectious endocarditis (IE), including *A. israelii*^{40–41} and *A. neuii*.⁴² However, IE caused by *A. odontolyticus* has not been reported previously. On the other hand, *Streptococcus sanguinis* is an important oral bacterium that is known to cause IE,⁴³ as demonstrated by numerous case reports of IE caused by *Streptococcus sanguinis* in the literature.^{44–47} It has been demonstrated that a particular lipoprotein is critical for *Streptococcus sanguinis* virulence for endocarditis.⁴⁸ Our patient, however, did not have any evidence of vegetation on either transthoracic or transoesophageal echocardiograms. Therefore, we may only presume from her CT of the chest that she may have had endocarditis, caused by either *Streptococcus sanguinis*, a well-known pathogen for IE, or *A. odontolyticus*, which had not been previously reported as a known pathogen for IE. There may be an association of septic pulmonary thromboembolism in streptococcal TSS in the absence of endocarditis, as described first in 1995 in a case report where the pulmonary microcirculation of the patient on autopsy was filled with thrombi that contained numerous Gram-positive cocci.⁴⁹ Given that our patient did not have evidence of IE on her echocardiograms, this may be the most plausible explanation. While we may attribute the patient's TSS to the presence of GAS on her IUD and her pelvic abscess to actinomycosis given the blood culture data, the clinical significance of the presence of *Streptococcus sanguinis* in her blood remains unclear.

Our patient had a very unusual clinical scenario without a clear explanation as to why the blood cultures were positive after multiple antibiotics, although the inadequate source control (the lack of removal of the IUD) may be one possibility. While it is possible that the bacteraemia could have resulted from the IUD removal as only the blood culture from the day the IUD was removed (day 4) grew an organism, what argues against this phenomenon is the fact that we were not

able to identify any alternative explanation for the patient's initial presentation and her prompt improvement after IUD removal on tailored antibiotic therapy. This clinical course suggests that the patient's illness was driven by an infection of the IUD and inadequate source control until removal of the device. Moreover, the IUD removal was uncomplicated and there was no haemodynamic compromise, no fever and no clinical decompensation after removal of the IUD to suggest that the removal itself triggered this patient's infectious illness.

Learning points

- Prompt diagnosis of TSS, especially in the absence of skin involvement, is paramount to preventing associated morbidity and mortality via early antibiotic treatment and mitigation of toxin-associated pathology.
- TSS and pelvic actinomycosis are infectious syndromes associated with IUD use; both diagnoses require providers to have a heightened index of suspicion in those with an IUD in order to recognise and solidify these difficult-to-recognise clinic syndromes, especially because both syndromes have substantial associated morbidity and mortality if treatment is delayed.
- Prompt removal of the nidus of infection (in our case, the patient's IUD) is a key step in establishing source control in patients with TSS or pelvic actinomycosis.

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