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Case Report

Group A streptococcal toxic shock syndrome secondary to necrotizing pelvic inflammatory disease in a postmenopausal woman

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ABSTRACT

Group A β-hemolytic streptococcus (GAS) is well known to cause upper respiratory tract or cutaneous infections, but some more virulent species of GAS can lead to a rapidly progressive life threatening soft tissue necrotizing infection and streptococcal toxic shock syndrome (STSS). In the modern era, GAS infections within the female reproductive tract leading to STSS are unusual and are often the result of retained products of conception or intrauterine devices. This report describes a case of GAS necrotizing pelvic infection in a previously healthy menopausal woman with no obvious portal of entry. Her clinical course rapidly progressed to septic shock and multiorgan failure. She required multiple surgeries in addition to targeted antimicrobials and aggressive management of shock and organ failures. After a prolonged hospital stay, she had a full recovery.

Introduction

The incidence of severe and invasive GAS infection was significantly reduced in the early 20th century, however, since the late 1980s, there has been a resurgence of severe GAS infections [1]. A proposed theory for the resurgence of more serious GAS related disease revolved around genetic shifts of M serotypes leading to GAS species with greater virulence [2]. GAS is mainly transmitted through respiratory droplet spread, however, skin to skin transmission has also been described. It has rarely been reported to cause vulvovaginitis and PID in nonpregnant women. To the best of our knowledge, this is the first report describing an invasive GAS infection of the pelvic organs in a menopausal woman that resulted in streptococcal toxic shock syndrome.

Case report

A 55 year-old previously healthy woman presented with a one day history of rapidly progressive lower abdominal and pelvic pain. Early on the day of admission, she felt well and actually went for a jog; however, later that evening, she developed a crampy lower abdominal pain that progressed rapidly in intensity, radiating to the pelvis and lower back. The pain was associated with nausea, vomiting, malaise, generalized body aches, a severe headache, and subjective fever.

She had no significant past medical history and she was post-menopausal. Her surgical history included an uncomplicated C-section 21 years ago and an uneventful IUD removal six months ago. She denied tobacco, alcohol or illicit drug use. She exercised regularly, worked as a kindergarten teacher and has been recently exposed to children who had sore throats in the days preceding the onset of her symptoms. She denied any symptoms of upper respiratory or genitourinary infection. The patient has no history of sexually transmitted diseases and she denied wearing any sanitary products such as tampons.

On admission, the patient appeared in moderate distress, was afebrile and her vital signs were within normal range. Her abdomen was tender to palpation, especially over the lower quadrants, without peritoneal signs. The vaginal speculoscopy showed edematous and ecchymotic vaginal walls with small amount of dark blood in the cavity. The cervix could not be visualized due to the amount of edema. The bimanual palpation revealed an enlarged tender uterus and a right adnexal mass. The laboratory data showed a normal leukocyte count and a platelet count of $111 \times 10^3/\mu l$. The bicarbonate was 18 mmol/L and the

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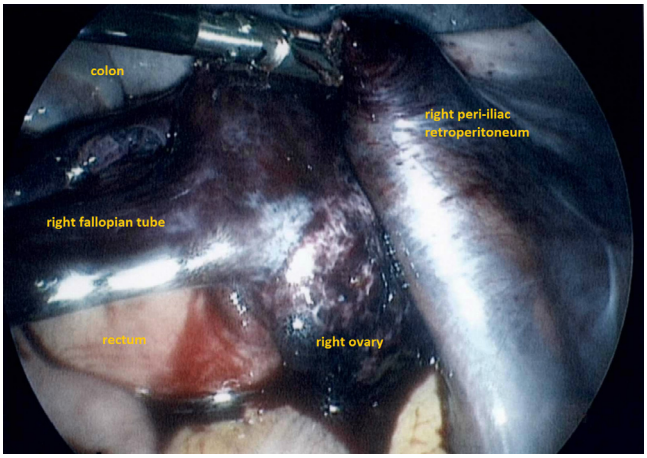

Fig. 1. Laparoscopy picture showing necrotic tissues of right fallopian tube and ovary.

anion gap was 15 consistent with an anion gap metabolic acidosis. Other blood chemistry tests and urine analysis were normal. An abdomen and pelvis CT scan showed predominately right-sided pelvic fluid collection without visualization of the appendix. A pelvic ultrasound revealed two mural uterine fibroids, the largest measuring 3 cm in diameter. The left ovary was unremarkable, the right ovary was not visualized.

The patient underwent a diagnostic laparoscopy, which revealed a completely necrotized right fallopian tube and ovary (Fig. 1) with dark sanguineous free fluid in the cul-de-sac. The left ovary, uterus, appendix, and bladder appeared normal. Surgeons performed a right salpingo-oophorectomy. A pelvic exam done post-surgery revealed a 2.5 cm $\times$ 3 cm right cervix hematoma. She received empirical antibiotic therapy with vancomycin and piperacillin/tazobactam.

On hospital day 2, her condition deteriorated rapidly overnight; she developed septic shock with multi-organ failure including disseminated intravascular coagulation (DIC). She had a fever of 103 °F and tachycardia of 136 bpm. The leukocyte count increased to 22.5 $\times$ $10^3/\mu l$ in several hours. Serum calcium and albumin levels drastically decreased. She was resuscitated with fluid and blood products. A subsequent examination revealed a small amount of vaginal bleeding and an ecchymotic cervix. The patient was taken again to the operating room because of the concern for progressing disseminated GAS. The surgery revealed extensive necrosis of the pelvic organs, a total abdominal hysterectomy and a left salpingo-oophorectomy were performed. Blood culture that was drawn on admission grew GAS. Culture results of uterine and cervix tissue that were collected from the second surgery confirmed GAS, therefore penicillin and clindamycin were started.

On hospital day 3, the patient returned to the operating room for a third time for a closure, during which the right infundibulopelvic (IP) ligament appeared necrotic and the gallbladder looked inflamed. The right IP ligament and gallbladder were excised and the abdomen was closed.

The patient developed acute respiratory distress syndrome and remained on mechanical ventilation for seven days. She eventually made a complete clinical recovery and was discharged home.

Discussion

Acute pelvic inflammatory disease (PID) is usually caused by organisms ascending from the genital tract. GAS is not a common organism in the vaginal flora thus an unusual cause of PID. With the resurgence of invasive GAS infections since the mid-1980s, cases of intrapelvic infection in healthy non-pregnant pre-menopausal women have been reported. These cases have occurred in association with prior disease and procedures such as surgeries, skin infections, child birth or intrauterine devices [3,4].

Traditionally, GAS is mainly transmitted through respiratory droplets, although direct skin to skin transmission may occur. It rarely colonizes as a vaginal flora, carriers are usually asymptomatic [5]. In a study of pregnant women, the vaginal-rectal colonization rate of GAS was 0.03% [6]. In the few reported cases of GAS genital tract infection in non-pregnant women, it was hypothesized that the bacteria was inoculated from the person's own pharynx or from a close contact source. While dermal or respiratory infection or carrier state serves as a likely source, sexual and hand contact increases the risk of genital-rectal inoculation, which presumably serves as a reservoir for ascending infection [7].

Mechanical disruption of the genital tract membrane by foreign bodies such as IUD, instrumental or surgical procedures or child birth have been considered possible portals of entry for pelvic infection related with GAS [3]. Less obviously menses and tampon use is reported as likely cause of ascending infection by GAS. In menopausal women, vaginal atrophy has been proposed to be a risk factor for genital GAS infection [8]. Literature reports about postmenopausal women and GAS PID is lacking. In this population, most frequently isolated microorganisms in PID were *E. coli* or *Klebsiella* [9]. The closest case related to this case was a 57 year old woman developed septic shock with DIC who was found to have GAS cervicitis during autopsy [10].

Prompt recognition with source control (*i.e.* surgical removal of infected tissue) and penicillin therapy are crucial to survival of invasive GAS infection. In our patient, the rapid progression of pelvic soft tissue necrosis warranted multiple surgical interventions. Clindamycin is a potent suppressor of bacterial exotoxin and inflammatory factors. The drug also facilitates phagocytosis of GAS by inhibiting M-protein synthesis [11]. Therefore it is recommended that high dose penicillin G and clindamycin be given in suspected invasive GAS infection. In our case, steroid was also administered, considering severe STSS can be associated with Waterhouse–Friderichsen syndrome [12].

Conclusion

Group A streptococcus is a rare cause of genital tract infection in nonpregnant women. Most of the invasive female genital tract infections by GAS were related with child birth, retained contraceptive product or sanitary products such as tampons, *etc.* We described a case of STSS secondary to extensive intra-pelvic tissue necrosis caused by GAS. We believe this case represents the first reported case of GAS STSS that results from rapidly spreading necrotizing inflammation of the genital tract in a previously healthy post menopausal woman. Of particular interest, she has no obvious trauma, intrauterine foreign body or recent surgical procedure that can serve as a portal of entrance. The crucial steps that lead to her full recovery are prompt source control with surgical excision and diagnosis, early targeted antimicrobial regimen, as well as aggressive management of shock and multi-organ failure.

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