Recurrence Group A Streptococcal Vulvovaginitis in Adult Women: Family Epidemiology

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Group A β-hemolytic streptococcal (GAS) vulvovaginitis has been reported in prepubertal girls. In adult women, a vaginal carrier state has been described, but vulvovaginitis is rarely reported. We describe 2 cases of recurrent GAS vulvovaginitis in women whose husbands were gastrointestinal carriers of GAS. Characterization of the isolated strains demonstrated that identical emm types of GAS were shared by partners. Treatment of both partners resulted in resolution of vulvovaginitis. On the basis of negative vaginal culture results obtained after treatment of each individual episode of vulvovaginitis, we believe that the female patients were reinfected as a result of exposure to their husbands, with shedding likely to have occurred in bed. These cases reiterate the necessity for adequate screening of the patient’s family and contacts in cases of recurrent GAS infection by culturing all potential areas of GAS carriage.

Group A β-hemolytic streptococcal (GAS) vulvovaginitis has generally been reported in prepubertal girls [1–5]. Between 11% and 20% of all swab samples obtained from girls with signs and symptoms of vulvovaginitis revealed GAS in culture [1]. In adult women, a vaginal carrier state has been described, but vulvovaginitis is only rarely reported. We describe 2 cases of recurrent GAS vulvovaginitis in women whose husbands were gastrointestinal carriers of GAS and likely shedders of these pathogenic organisms.

Case reports. Patient 1 was a 42-year-old woman who was referred to the clinic because of recurring episodes of symptomatic vaginitis in the previous 7 months. The patient had been in stable health until ~7 months before presentation, when she developed pruritus, soreness, severe erythema, and swelling of the vulvovestibular area. She experienced similar attacks almost monthly, and she was empirically treated with antifungal medication without any improvement. Repeated vulvovaginal cultures obtained during successive attacks revealed Streptococcus pyogenes, for which the patient was treated with azithromycin and cefuroxime. After each course of antibiotic therapy, the patient’s symptoms improved, and although her vaginal culture results became temporarily negative for S. pyogenes, symptoms recurred after a short period of time. The last course of antibiotic therapy was completed 1 week prior to her referral to our clinic (Wayne State University Vaginitis Clinic; Detroit, Michigan).

The patient was married and had 3 healthy children. There was no other pertinent past medical history. The patient was allergic to penicillin. Her husband had a history of a hospital admission for GAS necrotizing myofasciitis in his left upper thigh, which had occurred several months before our patient was referred to our clinic.

Our patient’s general physical examination had normal findings except for a nonradiating, grade 2 midystolic murmur in the apical area. Findings of a physical examination of her genitalia were normal. The vaginal pH was 4.1, the patient’s vaginal smear had normal findings, and the result of vestibule-vaginal culture was negative for yeast and GAS.

One month later, the patient returned with complaints of pruritus, soreness, and discharge. At this time, physical examination of her genitalia revealed erythema of the vaginal, vestibular, and perianal area. The vaginal pH was 4.7, a vaginal saline microscopy smear showed numerous cocci in chains, and cultures of samples from both the vaginal and perianal area were positive for GAS. At that time, the patient’s throat culture also was positive for GAS. Culture of a perianal sample obtained from the patient’s husband was negative for GAS, as was a stool culture; however, a throat culture showed no growth. Typing of the vaginal GAS isolates from the index patient and the GAS isolates obtained from her husband revealed the strains to be identical (T agglutination pattern 28, M protein [emm sequence] 28). Three children had samples cultured, none of whom had oral or rectal cultures positive for GAS. The patient was treated with clindamycin (300 mg 4 times per day) for 5 days, and her husband received penicillin G (500 mg 4 times per day) for 10 days.

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References


One week after finishing her treatment, the patient reported resolution of her symptoms, which was confirmed by a significant decrease in erythema and swelling on physical examination of her genitals. Her vaginal culture was negative for GAS. On her follow-up visits, she remained asymptomatic and culture negative. However, cultures of her husband’s perianal samples continued to be positive for GAS 2 weeks after completing the course of oral penicillin. Accordingly, he was treated with moxifloxacin administered at a dose of 400 mg daily for 14 days, and the patient was treated with moxifloxacin 400 mg daily for 7 days. Since then, the patient has remained asymptomatic and culture negative. Her husband has also remained rectal culture negative throughout a 6-month follow-up period.

Patient 2 was a 39-year-old woman who was referred to our clinic because of recurrent episodes of vulvovaginitis over a 6-month period, characterized by pruritus, vaginal discharge, and vulva redness and swelling. She received several courses of both antimycotic and antibacterial therapy from her family practitioner, with only short-term benefit. It is noteworthy that, on 2 occasions, cultures of vaginal and urine samples were found to be positive for S. pyogenes, but the finding was ignored and thought not to be relevant. She saw a urologist and a gynecologist, but her illness remained undiagnosed; she was given empirical anti–herpes virus therapy, because her type 2 herpes simplex virus: IgG titer was elevated.

Physical examination revealed diffuse erythema of the patient’s vulva, vestibule, and vagina, together with the presence of copious purulent secretions. Vaginal pH was elevated at 4.8, and 4+ polymorphonuclear leukocytes were seen on saline microscopic examination. A 10% potassium hydroxide examination for yeast had negative findings, and Gram stain of vaginal secretions revealed gram-positive cocci as singlets, pairs, and chains. Vaginal and rectal cultures (but not throat cultures) were positive for GAS. Similarly, cultures of rectal samples (but not of throat samples) obtained from the patient’s husband were positive for GAS. Cultures of throat samples obtained from the patient’s 3 children showed no growth. Characterization of the patient’s vaginal and rectal streptococci and of her husband’s rectal streptococci revealed identical strains of M protein type (emm sequence) 44/61.

The patient was treated with 2% vaginal clindamycin cream administered daily for 14 days, followed by amoxicillin administered at a dosage of 500 mg for 14 days. The patient’s husband received oral levafloxin (500 mg daily) for 28 days. The patient returned 28 days after the initiation of therapy and was entirely asymptomatic; she had normal physical examination findings, and both vaginal and rectal cultures were negative for GAS. Similarly, her husband’s rectal culture was negative for GAS. Further follow-up 3 months later confirmed clinical cure.

**Discussion.** GAS vaginitis is a rare condition, most often found in prepubertal girls and rarely found in adult women [1–5]. Patients complain of a purulent vaginal discharge, discomfort, and itching. Dysuria, pain, and bleeding have also been reported. Physical examination of the genitalia reveals erythema and tenderness of the vulvovaginal area. Light microscopic examination of Gram-stained vaginal secretions reveals gram-positive cocci in chains, as well as many polymorphonuclear leukocytes.

Although rates of vaginal carriage of GAS in adult women remain low, GAS vaginitis has been reported [6, 7]. In an investigation of vaginal colonization rates for group A β-hemolytic streptococci, Mead et al. [8] studied 6944 vaginal and rectal swab samples obtained from all patients delivering of infants at a Vermont hospital during a 38-month period and showed a 20.1% colonization rate for group B streptococcus but only a 0.03% colonization rate for GAS. Thus, GAS is seldom present in the normal vaginal milieu and is rarely the cause of vaginitis in adult women.

Carriage or exposure to a carrier is an important pathogenic factor in recurrent GAS infection, although it is often ignored. Although mostly found in the nasopharynx, GAS can colonize the perineum, anus, vagina, and normal skin [4, 9–14]. Skin colonization is mostly noted in people with dermatological conditions, such as psoriasis, eczema, and wounds. Patients with GAS pharyngitis spread the bacteria through droplets and physical contact. Gastrointestinal and perianal carriage may be evident in patients with pharyngitis even after pharyngeal infection has resolved and negative pharyngeal culture results have been obtained [9]. Perianal S. pyogenes shedding in the immediate environment may lead to contamination of bed sheets and mattresses, as was probably the case in our patients. Air contamination can also result from carriers, regardless of the colonized site [9, 15].

Reported outbreaks in health care facilities are infrequent but are indicative of serious complications [10, 15–18]. One study that reviewed postoperative wound infections due to GAS revealed GAS carriage by staff members, most often anesthesiologists and nursing staff members [15]. The anus and vagina were the most common carrier sites involved [14]. Relapse was also common months after treatment, often as the result of a GAS carrier in the patient’s household. It is unclear how long a colonized health care worker must be monitored. In recurrences of GAS infection, characterization of the GAS isolates is useful in tracing the original carrier.

Because both patients were finally effectively treated with clindamycin, the possibility of vaginal anaerobes producing penicillin-destroying β-lactamases should not be excluded as a cause for the prior recolonization of the vagina. However, in contrast to streptococcal pharyngitis, little is known about the clinical relevance of β-lactamase production in the vagina.

Following several months of recurrent GAS vulvovaginitis, evaluation of the patient’s families revealed only anorectal car-
riage in both husbands. All other culture sites, including the nose and throat, had samples with negative culture results. Characterization of the GAS isolates demonstrated identical strains in respective partners. Only after treatment of the partners following the patients’ clindamycin therapy was resolution of the recurrences achieved. After the failure of penicillin G therapy to eradicate intestinal carriage in the first patient’s husband, a high-dose, prolonged regimen of oral quinolones successfully eradicated GAS in the gut. We believe, on the basis of the negative vaginal culture results obtained after treatment of each individual episode of vaginitis, that the women were reinfected as the result of exposure to their male partners. Shedding is likely to have occurred in bed. The first couple was not sexually active throughout the period of recurrent vaginitis. However, in the second couple, sexual transmission could not be ruled out. These cases reiterate the necessity for adequate screening of the patient’s family and contacts in cases of recurrent GAS infection by culturing all possible areas of carriage.

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References