Sexually transmitted gastrointestinal syndromes include proctitis, proctocolitis, and enteritis. These syndromes can be caused by one or multiple pathogens. Routes of sexual transmission and acquisition include unprotected anal intercourse and oral-fecal contact. Evaluation should include appropriate diagnostic procedures such as anoscopy or sigmoidoscopy, stool examination, and culture. When laboratory diagnostic capabilities are sufficient, treatment should be based on specific diagnosis. Empirical therapy for acute proctitis in persons who have recently practiced receptive anal intercourse should be chosen to treat Neisseria gonorrhoeae and Chlamydia trachomatis infections. In individuals infected with human immunodeficiency virus (HIV), other infections that are not usually sexually acquired may occur, and recurrent herpes simplex virus infections are common. The approach to gastrointestinal syndromes among HIV-infected patients, therefore, can be more comprehensive and will not be discussed in this article.

Brief Review: Acute Proctitis/Proctocolitis

The incidence of acute, sexually transmitted proctitis and proctocolitis has appeared to decrease over the past decade. With the emergence of AIDS and identification of HIV, unprotected anal intercourse was reported to be the most efficient mode of sexual transmission of HIV infection [1, 2]. Advocates of “safer” sexual practices, therefore, admonish unprotected sexual contact of any kind, especially rectal intercourse.

Since the national rates of rectal gonococcal infections decreased dramatically, particularly among homosexual men, from 1985 to 1995, it has been assumed that safer-sex guidelines are being followed and that the practice of anal intercourse has declined [3–5]. Data from cohort studies of homosexual men verify this assumption, but other recent reports of sexual behavior among homosexual and heterosexual adolescents, illicit drug users, and heterosexual adults suggest that unprotected anal intercourse is quite common [6–10]. Why, then, are acute sexually transmitted anorectal infections so rarely identified or reported in current clinical practice? Possible explanations include low prevalence of sexually transmitted pathogens, superior therapeutic regimens, or misdiagnosis. Since sexual activity predisposing to these syndromes continues to occur, awareness of their presentations and therapeutics must be maintained. This review describes the epidemiology, etiology, and clinical presentation of the acute proctitis/proctocolitis syndrome and discusses current diagnostic, therapeutic, and counseling options.

Epidemiology

Sites of infection and mode of transmission. Sexual practices of homosexual men and heterosexual men and women frequently involve direct or indirect contact of the rectal mucosal membranes [11–13]. Specific pathogens most commonly infect specific sites and have different modes of transmission. Fecal-oral passage of entero pathogens, parasites, and hepatitis A and B viruses occurs predominantly during direct oral-anal contact, or anilingus, or during oral-genital contact after rectal intercourse [14]. Exposure to as few as 10–100 organisms may precipitate infection. Chlamydia trachomatis and Neisseria gonorrhoeae infect columnar epithelium and infect the anorectal mucosa via oral-genital and rectal insertive intercourse. Herpes simplex virus (HSV), human papilloma virus, and Treponema pallidum infect stratified squamous epithelium and can be transmitted similarly to the anorectal region.

Risk factors. Factors associated with an increased risk of acquiring any STD include multiple partners, anonymous partners, and individual sexual practices that increase the risk of acquiring specific diseases, as previously mentioned [15, 16]. In the pre-AIDS era, homosexual men who engaged in these high-risk sexual practices were also at high risk for acquiring sexually transmitted proctitis/enteritis [14–16]. The updated Kinsey scales of homosexual behavior, published in 1989, reported that only 15% of homosexual men practiced sex frequently with multiple partners [12]. Thus, the high prevalence of STDs, including sexually transmitted proctitis/enteritis, reported among homosexual men during this time period probably occurred in a sexually active subpopulation. Since the advent of AIDS, this subpopulation has dramatically decreased in size, as reflected in diminishing rates of anorectal gonorrhea among homosexually active men [5].

Current Trends

Although unprotected anal intercourse and oral-anal contact have reportedly diminished, especially among older homosexual men, in part because of AIDS-intervention messages, anal intercourse as an AIDS-risk behavior for heterosexuals has received less attention [17–19]. Nonetheless, anal sex continues to be a prevalent sexual practice in both homosexual and
heterosexual populations. Several investigators have reported that as high as 43% of adult women have participated in anal intercourse [20–22]. A nationally representative study of the sexual behavior of men aged 20–39 years in the United States showed that 20% have engaged in anal intercourse, and only 2% of sexually active men have had any same-gender sexual activity during the past 10 years [10].

Among college-aged students, prevalence rates for rectal intercourse range from approximately one-fifth to one-third [23]. A study conducted among 137 sexually active, preadolescent and early adolescent low-income urban black youths showed that 36% had engaged in anal intercourse, including 35% of the sexually active boys and 43% of the sexually active girls [6]. A Canadian survey of street youths aged 15–20 years found that STD/HIV high-risk behaviors were frequent, with 73% of males and 75% of females inconsistently using condoms and 22% of males and 24% of females participating in anal intercourse [8].

To date, sexually transmitted anorectal infections have not been reported as a consequence of these sexual practices among these subpopulations. Unprotected anal intercourse, however, is being reported more frequently among several other subpopulations in which STDs are more prevalent and HIV is a constant risk, such as adolescent cocaine/crack users and heroin users, as well as drug-using and -nonusing prostitutes [9, 24–27].

Cultural Factors

Despite more prevalent AIDS awareness, more liberal laws, and more social consciousness, prejudices on the part of clinicians against anorectal sexual activity may foster higher rates of STDs. Closed social circles and anonymous sexual contacts promote disease spread and hinder contact-tracing and disease control. Clinicians, social workers, and disease-intervention specialists must therefore develop a tactful approach in eliciting information regarding anorectal sexual exposure, and this information may result in more rapid disease detection, treatment, and control.

Common Clinical Syndromes

Proctitis refers to inflammation of the rectal mucosa. Anatomically, infections may involve several areas. The perianal area up to the anal verge is lined by keratinized, stratified squamous dermal epithelium, and lesions caused by syphilis, HSV, or condyloma acuminata are generally classic in appearance. The epithelium gradually changes from stratified squamous to stratified cuboidal epithelium from the anal verge up to the anorectal (pectinate or dentate) line. This area has an extensive supply of sensory nerve endings, and infection is commonly very painful and sometimes results in constipation and tenesmus due to spasm of the anal sphincter muscle. From the anorectal line cephalad, the rectum is lined by columnar epithelium. Infections that involve the rectum but spare the anus are relatively painless. Symptoms of proctitis, therefore, include anorectal pain, mucopurulent or bloody rectal discharge, tenesmus, and constipation. Many infections are asymptomatic. Proctocolitis refers to infections that involve the rectum and the colon. Symptoms may include those of proctitis as well as abdominal pain, bloating, cramping, and sometimes diarrhea and fever.

Physical examination helps to classify patients with regard to these syndromes. Anoscopic examination of patients with either syndrome can reveal rectal exudates and/or rectal bleeding. In proctitis, sigmoidoscopic evaluation reveals diseases limited to the rectum, whereas with proctocolitis the disease process extends above 15 cm into the sigmoid colon.

Etiology

Specific pathogens have been associated with proctitis and proctocolitis (table 1) [14, 15]. Polymicrobial infection often occurs, causing an overlap of symptoms. Asymptomatic infections are also prevalent, and the clinician should routinely inquire about rectal exposure, regardless of the patient’s sexual preferences, so as to perform screening cultures or anoscopic evaluation when appropriate.

Individual Pathogens Causing Proctitis

N. gonorrhoeae. During the late 1970s and early 1980s, when sexually transmitted infections among homosexual men were being intently studied and characterized, gonococcal infection was the most common STD reported [28–31]. According to several studies, the rectum was the most common site for gonococcal infection. The concept that rectal gonorrhea was always asymptomatic was proved to be a misconception. Nonetheless, asymptomatic gonococcal infection of the rectum was common.

Since the AIDS epidemic began in 1982, the incidence of gonorrhea has declined markedly among homosexual men [32]. Outbreaks, however, continue to be reported, especially among young homosexual men who fail to follow safer-sex guidelines [33, 34]. Little has been reported for heterosexual populations, especially adolescents, so true prevalence rates are not known.

Symptoms associated with rectal gonorrhea may develop 5–7 days after exposure and include mild anorectal pain, itching, and mucopurulent discharge [15]. Occasionally, more severe symptoms may occur, which include impressive tenesmus and constipation. Anoscopic evaluation may reveal mucopus in the anal canal, especially around the anal crypts [29, 35]. The rectal mucosa may appear completely normal or may appear erythematous and friable, especially near the anorectal junction.

Rectal gram-staining is only 30%–40% as sensitive as culture, although it is highly specific [36]. However, in evaluation of men with symptomatic rectal gonorrhea, the sensitivity of gram staining was 79% when rectal samples were obtained via anoscopy, compared to 53% when they were obtained via an inserted swab [37]. Culture techniques, however, are similar regardless of the swabbing technique used. DNA assays are now available for gonococcal detection in urogenital specimens, but they have not been extensively studied for rectal
Table 1. Common sexually transmitted gastrointestinal syndromes.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Proctitis</th>
<th>Proctocolitis</th>
<th>Enteritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td>Rectal pain, discharge, tenesmus</td>
<td>Proctitis symptoms plus cramps, diarrhea</td>
<td>Diarrhea, cramps, bloating, nausea</td>
</tr>
<tr>
<td>Pathogen(s)</td>
<td><em>Neisseria gonorrhoeae</em></td>
<td><em>Entamoeba histolytica</em></td>
<td><em>Giardia lamblia</em></td>
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<td></td>
<td><em>Chlamydia trachomatis</em></td>
<td><em>Campylobacter jejuni</em></td>
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<td></td>
<td><em>Treponema pallidum</em></td>
<td><em>Shigella flexneri</em></td>
<td></td>
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<tr>
<td></td>
<td><em>Herpes simplex virus</em></td>
<td><em>C. trachomatis (LGV</em>)</td>
<td></td>
</tr>
<tr>
<td>Mode of acquisition</td>
<td>Receptive anal intercourse</td>
<td>Direct or indirect fecal-oral contact</td>
<td>Direct or indirect fecal-oral contact</td>
</tr>
<tr>
<td>Anoscopic findings</td>
<td>Rectal exudate ± friability</td>
<td>Rectal exudate, friability that may extend into the sigmoid colon</td>
<td>Normal</td>
</tr>
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</table>

* Lymphogranuloma venereum strains.

specimens. The PACE-2 DNA probe assay (GenProbe, San Diego, CA) was reported to be 100% sensitive in comparison with culture for detecting rectal gonorrhea in a population where the prevalence of anorectal gonorrhea was 4.7% [38].

Therapy for urogenital gonococcal infection includes single-dose regimens effective against β-lactamase-producing strains. A single 125-mg im injection of ceftriaxone cures 99.1% of uncomplicated urogenital and anorectal infections [39]. A single 400-mg oral dose of cefixime cures 97.1% of uncomplicated infections, and single oral 500-mg doses of ciprofloxacin or 400-mg doses of ofloxacin have resulted in cure rates of 99.8% and 98.4%, respectively [40–42]. Other regimens include cefixime, 500 mg im; cefotaxime, 500 mg im; cefotetan, 1 g im; and cephradine, 2 g im, plus probenecid, 1 g po. None of these regimens offers any advantage over ceftriaxone. Ceftriaxone, however, is reportedly useful in the treatment of incubating syphilis, in contrast to quinolones, and this should be considered in choosing treatment regimens for gonococcal infections in populations where incident syphilis rates are high [43].

Empirical treatment for acute proctitis/proctocolitis syndromes is the administration of a single dose of ceftriaxone (125 mg im) plus doxycycline (100 mg po) twice a day for 7 days [44]. This regimen covers the sexually transmitted pathogens that most commonly cause these symptoms (table 1) and is especially effective against gonorrhea, chlamydia, and incubating syphilis.

*C. trachomatis*. *C. trachomatis* infections involving the rectum are usually asymptomatic or mildly symptomatic when caused by the non–lymphogranuloma venereum (non-LGV) strains of *Chlamydia* [45]. Non-LGV strains or serovars can cause a mild proctitis with symptoms of rectal discharge, tenesmus, or anorectal pain. In contrast, direct rectal inoculation with LGV strains (L1, L2, or L3) of *C. trachomatis* may cause severe anorectal pain, a bloody mucopurulent discharge, and tenesmus [46, 47]. Although urogenital infections with LGV serovars of *C. trachomatis* are common in East and West Africa, South America, the Caribbean, and Southeast Asia, these strains occur only sporadically in the United States and Europe and therefore will not be discussed further in this review [48].

Non-LGV serovars of *C. trachomatis* that cause anorectal infections may differ from those causing cervical or urethral infections, and some non-LGV serovars may be associated with more severe urogenital disease [49]. More data are needed to clarify these associations with regard to anorectal infections.

Regardless of symptoms, chlamydial rectal infections are usually associated with friable rectal mucosa and mucopurulent discharge, as seen on anoscopy. Rectal-specimen gram stains show increased numbers of polymorphonuclear cells [45]. Sig-moidoscopy can yield normal findings or reveal mild inflammatory changes with small erosions or follicles in the lower 10–15 cm of the rectum.

*C. trachomatis* rectal infection may be confirmed by culture or by direct immunofluorescent staining [50]. Enzyme immunoassay assays for detection of chlamydial antigens in rectal samples are associated with high false-positivity rates [51]. Although DNA detection techniques have been well studied and are widely used for urogenital infections, these tests have not been evaluated adequately for diagnosing anorectal chlamydial infections.

Doxycycline (100 mg po twice daily for 7–10 days) is effective in the treatment of non-LGV chlamydial proctitis/proctocolitis. Azithromycin (1 g as a single oral dose) is effective for chlamydial urethritis and cervicitis and has been recommended for uncomplicated rectal infections [52].

*T. pallidum*. Rates of primary and secondary syphilis among heterosexual men declined dramatically in the United States during the late 1980s and throughout the 1990s [53]. Among heterosexual men and women, however, an increase was noted during this same period, and this increase coincided with an epidemic of crack cocaine use [54]. Currently, rates of primary and secondary syphilis for the nation as a whole are on the decline. Nevertheless, incident syphilis remains a problem for several Southern states and in populations where drug use, prostitution, and poverty persist regardless of geographic location [55].

Primary syphilis can present as an anorectal chancre. The classic chancre is painless, with well-demarcated indurated edges and a clean base [56]. It appears 2–6 weeks after exposure, at the site of sexual contact with an infected partner. Although the anorectal chancre may be asymptomatic, it may also present with itching, bleeding, rectal discharge, constipation, and tenesmus [57]. It can easily be misdiagnosed as a rectal fissure. A rectal mass lesion, condylomata lata, and/or mucous patches are other common clinical presentations associated with the secondary stage of syphilis [58–60].
If the patient reports any anorectal sexual exposure, anoscopy should be performed as a routine part of the physical examination. Fluid can be collected from any observed anorectal lesions, typically from the base of the chancre or condylomata lata, and should be examined under dark-field microscopy for detection of typical spirochetes and immediate diagnosis. Serological tests for syphilis include the rapid plasma reagin (RPR) and Venereal Disease Research Laboratory (VDRL) screening tests, of which positive results are confirmed by more specific tests such as fluorescent treponemal antibody absorption (FTA-ABS). These tests may miss 10%–25% of primary syphilis cases, involving patients who have not been infected long enough to mount a detectable antibody response [61]. Biopsy specimens of any suspicious lesions should be examined histologically with silver or specific immunofluorescent stains for T. pallidum [62].

The drug of choice for primary and secondary syphilis is benzathine penicillin (2.4 million units im). HIV-infected patients with primary or secondary syphilis who are allergic to penicillin may be managed and treated according to recommendations for HIV-negative patients. Patients with primary or secondary syphilis who are HIV-seronegative and allergic to penicillin can be treated with a 2-week course of doxycycline (100 mg twice daily). All sex contacts, regardless of signs, symptoms, or serological states, should be treated [63].

Herpes simplex virus. Anorectal herpes infection can be acquired by anal intercourse or through oral-anal contact. Most anorectal HSV isolates have been type 2 (HSV-2), but HSV type 1 (HSV-1) also is isolated. In a study of primary HSV proctitis, 70% of isolates were HSV-2 and 30% were HSV-1 [64]. At presentation a primary infection may involve the perianal skin and anal canal and may extend to the rectum. Pain may be severe, occurring with rectal discharge, tenesmus, and constipation. S4–S5 dysesthesia, sacral paresthesia, urinary retention, and temporary impotence have been reported in up to 50% of primary anorectal HSV infections. The patient may also have symptoms consistent with viremia, such as fever, chills, malaise, headache, and meningismus [65].

On examination of the perianal area, typical herpetic vesicles, pustules, or ulcerations may be seen. In severe cases, perirectal edema and erythema may be confused with a yeast infection. Anoscopy may be painful and may reveal an edematous, friable mucosa with ulcerations. In immunocompetent individuals, the infection rarely extends above 15 cm. Diagnosis is confirmed through culture or direct immunofluorescent staining.

In a prospective, double-blinded, placebo-controlled trial of therapy for primary HSV proctitis, acyclovir (400 mg po, 5 times daily for 10 days) was clinically efficacious in comparison with placebo [64]. No other controlled trials have been conducted recently, but some experts believe that acyclovir, valacyclovir, and famciclovir can be administered according to the same dosing schedules recommended for other genitourinary HSV infections.

In HIV-infected patients, severe mucocutaneous HSV infection, regardless of site, is treated with 5–10 mg/kg of iv acyclovir administered every 8 hours until clinical resolution is attained [66]. The patient may be given oral HSV suppressive therapy with acyclovir (400 mg twice daily). A recent double-blind, placebo-controlled, crossover trial of famciclovir (500 mg po twice daily) vs. placebo, administered for 8 weeks, revealed clinically and statistically significant reductions in the symptoms associated with HSV infection and in the symptomatic and asymptomatic shedding of HSV among HIV-positive persons. With this regimen, the percentage of days with genital lesions was reduced from 13.8% to 4.9%, and all recurrent anogenital lesions were due to HSV-2 [67].

Enteric Pathogens Causing Proctocolitis and Enteritis

Historically, enteric pathogens that cause proctocolitis and enteritis have not been considered agents of STDs. These infections are associated with the ingestion of fecally contaminated food or water. Certain sexual practices, especially analingus, may allow direct exposure to these pathogens and thus promote transmission of Campylobacter, Shigella, Salmonella, Entamoeba histolytica, Giardia, and several other enteric pathogens [15, 68–71].

Shigella. Most shigella infections in the United States are caused by Shigella sonnei and Shigella flexneri [72]. The infective dose for shigellosis is very small (10–100 organisms); thus, an infected individual can be very contagious. Symptoms may be absent or may include abrupt onset of watery or bloody diarrhea with associated nausea, tenesmus, cramping, and fever. Examination of stool shows RBCs and mucopus, and sigmoidoscopy may reveal an inflamed, friable mucosa extending above 15 cm. Diagnosis is confirmed by stool culture on selective media.

Treatment is supportive and consists mainly of hydration. Antimotility agents should not be given. Antibiotics are recommended by some experts to prevent complications in immunocompromised patients, and these should be selected according to regional antibiotic susceptibility patterns because of the widespread development of resistance. The value of treating sex contacts is unknown, but tracing them in order to detect asymptomatic carriers is an important public health measure.

Campylobacter. Campylobacter infections are usually acquired by ingestion of contaminated water or food, particularly chicken and unpasteurized milk [73]. Campylobacter organisms are microaerophilic, curved, motile, gram-negative rods. Campylobacter jejuni is one of the most commonly isolated bacterial agents from patients with acute diarrhea in the United States, and its annual incidence is reported to be 39-fold higher among patients with AIDS [74].

Campylobacter species isolates have been recovered in rectal and stool cultures as the sole pathogens in homosexual men presenting with proctocolitis. Atypical Campylobacter species, such as Helicobacter fennelae, Helicobacter cinaedi, and Campylobacter lari, have also been associated with bacteremic illnesses among AIDS patients with fever and among homosexual men with proctocolitis [75–77]. The most likely route of infection is fecal-oral. These Campylobacter species have also...
been associated with neutrophilic rectal secretions among asymptomatic homosexual men [78].

Symptoms associated with acute infection include diarrhea, abdominal bloating, and mucopurulent rectal discharge. Fever, chills, myalgias, and abdominal pain may occur, and in severe cases gastrointestinal infection can mimic acute appendicitis or inflammatory bowel disease. In mild cases, sigmoidoscopy and anoscopy can yield normal findings or show mucosal friability beyond 15 cm, similar to that seen with shigellosis. The diagnosis is confirmed by stool culture on selective media in a microaerophilic atmosphere.

Use of erythromycin (500 mg four times daily for 1 week) may benefit severely symptomatic individuals and is recommended for immunocompromised patients. This regimen may shorten duration of symptoms and fecal shedding, but bacteremia and recurrent diarrhea have been reported to occur following specific therapy [79]. Ciprofloxacin- and azithromycin-resistant isolates from Thailand have also been recently reported [80].

Salmonella. Salmonella has been reported as a cause of enteritis in homosexual men but has a 20-fold greater incidence among patients with AIDS [81, 82]. Salmonella bacteremia in an HIV-infected person is diagnostic of AIDS. In immunocompromised individuals these infections, which are usually due to Salmonella typhimurium and Salmonella enteritidis, are frequently associated with bacteremia and relapse despite directed therapy.

Diagnosis is made by culture of stool on selective media, and treatment is individualized according to severity of symptoms and antibiotic susceptibility findings. Immunocompromised patients with mild illness are usually given supportive therapy without antibiotics. Ciprofloxacin (750 mg twice daily for 2–4 weeks) is generally recommended for patients with AIDS, and ciprofloxacin (500 mg) is given indefinitely if relapse occurs [83].

**Parasitic Infections**

The role of sexual transmission in the spread of parasitic infections was suggested in 1972 when the prevalence of *Giardia lamblia* and *Entamoeba histolytica* was reported to be as high as 30%–40% in selected populations of homosexual men [84–87]. The presence of these parasites correlated better with a history of analgus than with travel to areas of endemicity.

*G. lamblia* is associated with symptoms of enteritis, since it is typically an infection of the small intestine. Symptoms include diarrhea, bloating, abdominal cramps, and nausea. Stool examination findings are often negative, and sampling of jejunal mucus by the string test (EnteroTest; Hedeco Corporation, Mountain View, CA) or by small bowel biopsy confirms the diagnosis. Therapy in the United States is with metronidazole (250–500 mg three times daily for 7 days). Alternatives include paromomycin (500 mg po three times daily) or furazolidone (100 mg four times daily), given for 7–10 days. A 2-g single oral dose of tinidazole is given in Europe for this infection, but this 5-nitro imidazole is not available in the United States [88].

Amebiasis is caused by two distinct species of *Entamoeba*, a pathogenic form (*E. histolytica*) and a nonpathogenic form (*E. dispar*), that are morphologically identical. *E. histolytica* has been found in the stool of both symptomatic and asymptomatic individuals [14]. Symptoms, if present, can vary from mild diarrhea to fulminant bloody amebic dysentery. Amebic proctocolitis may be visually indistinguishable on colonoscopy from other inflammatory bowel diseases. Diagnosis is best confirmed by the identification of *E. histolytica* cysts or trophozoites on examination of fresh stool specimens [89].

Identification of *E. dispar* would be of clinical importance, since infection with this nonpathogenic form may not necessitate therapy. Methods developed to distinguish these species, however, are either very time-consuming or involve laborious procedures for isolation of DNA [90]. Simpler methods to differentiate between these two species of *Entamoeba* are needed to enable full understanding of the epidemiology, clinical syndromes, and pathology of amebiasis.

When *E. histolytica* is identified in specimens from asymptomatic individuals, therapy is given to prevent continued infection and avoid transmission. Luminal amebicides are recommended. Therapy is with iodoquinol (650 mg po three times daily for 20 days). Alternative agents include paromomycin (25–30 mg/[kg·d] in three doses for 7 days) or diloxanide furate (500 mg three times per day for 10 days), which is available only through the Centers for Disease Control and Prevention [91]. Metronidazole (750 mg three times daily for 10 days) is the agent of choice for invasive, symptomatic infection. This regimen, however, does not eradicate organisms in the intestinal lumen and must be followed by a course of iodoquinol at the dosage indicated above.

Several other parasites frequently identified in the stool of homosexual men have included *Entamoeba nana*, *Escherichia coli*, and *Entamoeba hartmanni* [69–71]. *Iodamoeba butschlii* and *Dientamoeba fragilis* are less commonly identified, and the significance of any of these parasites as potential pathogens has not been established. Occurrences of severe intestinal infections with several protozoan parasites that include *Cryptosporidium*, *Isospora*, and *Microsporidium* have been reported among AIDS patients, with high frequencies among homosexual men with AIDS [92–95]. The epidemiology of these infections is not fully understood, and the role of sexual transmission has not been studied but should be addressed in future research.

**Discussion**

Although the incidence of acute sexually transmitted rectal and gastrointestinal syndromes appears to have decreased, the diligence of clinicians to pursue the diagnosis should persist. A careful history about specific sexual practices, as well as the method and frequency of condom or other barrier use should be obtained from all patients, regardless of their sexual preference. This will provide important clues to direct proper evaluation, specimen collection, therapeutic choices, and counseling, regardless of presenting symptoms.
Since the clinical presentations of infections with most pathogens that cause proctitis/proctocolitis are similar, a systematic approach is helpful. Often an immediate diagnosis can be made on the basis of distinct clinical presentations (primary HSV proctitis), rectal gram staining results (gonococcal proctitis), or the results of dark-field microscopy or nontreponemal serological tests (syphilis). Specific therapy is ideal but not always immediate or preferable, since many sexually transmitted gastrointestinal infections are symptomatic and polymicrobial. Identification of the specific pathogens takes time and relies on the results of appropriate cultures or other microbiological studies.

Empirical therapy is directed against gonococcal and chlamydial infections, but this recommendation is based upon studies that were conducted a decade ago [14, 15, 44]. Current data on the prevalence, epidemiology, and etiology of sexually transmitted rectal infections are needed. Furthermore, the role of sexual transmission should be studied for several relatively new pathogens such as Cryptosporidium and Microsporidium.

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