Leading article

The gay bowel

Through the early 1970's with increasing freedom of sexual expression a subpopulation of homosexual men, particularly in the larger cities of North America and Europe, engaged in sexual intercourse with large numbers of casual and often anonymous partners.[1] There followed increases in conventional sexually transmitted diseases (STDs), those not previously recognised as such and the appearance of the acquired immune deficiency syndrome (AIDS).

The anorectum is a sexual organ and in the United Kingdom a conservative estimate indicates that between 2 and 2·5 million people use it for sexual fulfilment.[2] A wide variety of organisms can be locally transmitted through penile-anorectal contact. The rectal mucosa is more fragile than that of the vagina and breaks in it more readily facilitate the transmission of organisms such as hepatitis B virus, cytomegalovirus (CMV) and probably human T-lymphotropic virus III (HTLV III). A wider sexual contact involving the anorectum, mouth, and hands allows for the transmission of enteric organisms.

The 'gay bowel syndrome' was first used to describe not a syndrome, but a list of conditions.[3] The term hides the problems facing the gastroenterologist. Firstly, the sexual orientation of a patient may not be easily ascertainable in the setting of a general outpatient clinic. Secondly, many infections of the gay bowel are asymptomatic and are missed without full microbiological screening. Thirdly, coinfection is common and the organism isolated may not be causing the symptoms and signs. Finally, the bowel has limited and non-specific clinical and histopathological responses to many infections.

Gonorrhoea is one of the commonest STDs in gay men and the rectum is the only site infected in 40%, most patients being asymptomatic.[4] Others present with anorectal symptoms and non-specific inflammation. Diagnosis can be made on a Gram stained rectal smear in only 50% of cases so that culture is essential for confirmation. A common treatment is a single dose of ampicillin 3 g with 1 g of probenecid orally. Penicillinase-producing *N gonorrhoeae* (PPNG) is now endemic in the United Kingdom, rather than imported, but is less common in homosexual than in heterosexual men.[5 6] Syphilis is now largely seen in its early infectious stages (the first two years of infection) and approximately 80% of syphilis cases seen in central London clinics have been homosexually acquired.[7]

The anorectal chancre may be atypical. It is easily missed by the patient and physician, may be multiple, tender or mimic an anal fissure. Perianal lesions of secondary syphilis may coalesce to form *Condylomata lata* which must be distinguished from the more common anogenital warts *(Condylomata acuminata)*. Dark ground microscopy can be used to identify

T pallidum from primary and secondary lesions. The disease then enters its early latent phase with no clinical signs of infection. For this reason gay men at risk should be regularly screened three monthly with serological tests and for *N gonorrhoeae* in exposed sites, throat and/or urethra and/or rectum. Penicillin remains the treatment of choice for all stages of syphilis.

Chlamydia trachomatis serotypes D-K (non-LGV) may cause an asymptomatic rectal infection or a mild proctitis.[8][9] The LGV serotypes tend to cause more severe inflammation which may extend beyond the rectum with ulceration or perianal abscesses, fistulas and occasionally strictures resembling Crohn's disease, but the relationship between serotype and severity of disease is not always clear cut.[10] An aetiological association between *C trachomatis* and Crohn's disease is not supported by serological studies.[11] Diagnosis is made primarily by isolation of the organism. Serology with paired sera using micro-immunofluorescence may be useful and with LGV the complement fixation test titre is usually raised ($\geq$1:16 and often $\geq$1:64).[9] Treatment is with oxytetracycline.

As might be expected with oral-anal contact, infection with *Shigella, Salmonella* and *Campylobacter* species have been described.[12][13] More recently campylobacter-like organisms (CLOS) have been implicated as a cause of proctocolitis.[13] Spirochaetes other than *T pallidum* are commonly found in the gay rectum, but like their counterparts in the upper gastrointestinal tract their pathogenic role is unclear.[14][15]

Giardia lamblia and *Entamoeba histolytica* have long been regarded as 'exotic' organisms, but are 'hyperendemic' among gay men attending STD clinics with up to 20% excreting cysts.[16][17] Infections are usually asymptomatic and even when symptoms are present they correlate poorly with the presence of organisms.[18] *E histolytica* trophozoites have been divided into pathogenic and non-pathogenic types by isoenzyme patterns (zymodemes) and non-pathogenic types seem to be common in gay men.[19] The symptomatic patient should be treated with metronidazole with other infections confidently excluded, because as many as 50% of symptomatic gay men may have one or more additional pathogens. Treatment of the asymptomatic patient with cysts remains controversial.

In the United Kingdom between 1972 and 1982 the annual number of cases of the two commonest viral STDs, genital herpes and warts, has increased by 230% and 105% respectively. Primary or initial infection with herpes simplex virus (HSV, usually type 2) is a common cause of proctitis and was isolated from 23 of 102 (23%) consecutive homosexual men attending an STD clinic with rectal symptoms.[20] Anorectal pain, tenesmus, constipation, perianal lesions (papules, ulcers or crusts), inguinal lymphadenopathy, fever, neurological symptoms such as difficulty with micturition, sacral parasthesias and posterior thigh pain are important distinguishing features from other causes of proctitis. Again coexisting pathogens may be found in as many as 35% of patients. Acyclovir given by intravenous or oral routes reduces the duration of viral shedding, time to healing and the duration of symptoms, but the frequency of recurrences is unaltered.[21] Treatment must be started after a clinical diagnosis before the results of confirmatory viral isolation.

Genital warts are caused by the human papilloma virus (HPV) and found in 8–12% of gay men attending clinics.[18][22] Restriction enzyme digestion and nucleic acid hybridisation reveal many subtypes with at least

four; HPV 6, 11, 16, 18 apparently specific for the urogenital tract.[23] They are usually asymptomatic and discovered during a checkup or examination after presentation with another STD and approximately 60% of homosexual men with perianal warts will be found to have rectal warts. To some extent genital warts have been regarded as trivial and certainly frustrating to treat, with high recurrence rates after treatment with caustic agents such as podophyllin, cryotherapy and surgery.[24] A strong association between the urogenital HPVs and cervical neoplasia has been established and there have been case reports of anorectal carcinoma in gay men. Furthermore an association in men has been shown between squamous and transitional anorectal carcinoma and past syphilis and never being married, both markers for a gay lifestyle.[25] The focus of attention of the cervical molecular biologists is now being directed towards the anorectum of gay men.

A number of miscellaneous conditions complete the list of gay bowel diseases. Pruritis ani may be caused by helminth infections such as *Enterobius vermicularis* (pin worms, threadworms) or arthropod infections namely *Pthirus pubis* (the pubic louse, 'crabs') or *Sarcoptes scabiei*. Traumatic anorectal conditions from anorectal intercourse, the insertion of foreign bodies or 'fisting' are well publicised but not often seen in the STD setting.[26]

To deal with the gay bowel the gastroenterologist has to determine the sexual orientation of his patient, regard the anorectum as a sexual organ, remember that asymptomatic and coinfections are common and have full microbiological support. A useful guide to management was put forward by Quinn and his colleagues.[27] An immediate diagnosis can be made at initial physical examination and proctoscopy which may show the typical maculopapular rash of secondary syphilis, a chancre with *T pallidum* on dark ground microscopy, or perianal ulcers of an HSV proctitis. Proctoscopy with gram stain of a rectal smear may show *N gonorrhoeae,* but will miss 50% of cases. Serology for syphilis should always be taken together with swabs for *N gonorrhoeae* from the rectum and other exposed sites. Specific therapy can then be started after the appropriate confirmatory culture, or isolation. If no immediate diagnosis is made, a combination of the symptoms and signs at sigmoidoscopy may help. If the symptoms are essentially enteric – for example, diarrhoea, abdominal cramps, bloating, and sigmoidoscopy normal, consider *G lamblia*. If symptoms are predominantly anorectal and signs limited to the distal rectum swabs should be taken for culture of *N gonorrhoeae* and herpes simplex and isolation of *C trachomatis*. Chlamydial serology may be helpful if isolation is negative. If inflammation extends beyond 15 cm consider *Campylobacter, Shigella, E histolytica* and an LGV *C trachomatis* infection. Persistent symptoms and signs with no evidence of infection will need further investigation with biopsy and contrast studies, a home game for the gastroenterologist. The physician in genitourinary medicine has greater facilities for on the spot microbiological support for screening, strict protocols in follow up for proof of cure, health advisers to assist in tracing contacts and most importantly, health education. The ideal management of the gay bowel is a combined effort by departments of gastroenterology and genitourinary medicine.

Human T-lymphotropic virus (HTLV III) infection is now endemic

among homosexual men in the United Kingdom. One in five gay men attending an STD clinic in London have antibodies to HTLV III and at least 2600 gay men in London had been infected by July 1984.[28] Human T-lymphotrophic virus III infection is characterised by chronicity and the virus has been isolated from the blood, semen and saliva of anti-HTLV III positive individuals.[29] [30] There will be a move to use anti-HTLV III testing to determine to whom one should apply the recommended safety precautions for care, laboratory containment and the disinfection of instruments used in invasive procedures, even though nosocomial transmission has been shown to be very rare with the established guidelines.[31–35] Before adopting such a policy one should recognise that a negative test does not exclude infection and that anti-HTLV is not a measure of infectivity. Furthermore, one should consider the implications to the patient of a positive test in terms of its meaning, the social and sexual consequences and anxieties concerning confidentiality. With the rising prevalence in London, an alternative policy would be to consider all identified gay men at risk as potentially infected and to take appropriate precautions.

One of the end-points of HTLV III infection is AIDS and the bowel is one of the commonest organs for the tumours and opportunistic infections.[36] Kaposi's sarcoma alone is the presenting feature in 21% and gay men have a higher incidence than other risk groups. There is widespread involvement of skin, mucous membrane, lymph node and viscera, with lesions occurring in the gut from the oropharynx to the rectum and ranging from small, flat lesions seen at endoscopy, but not well demonstrated by contrast studies, to larger nodular lesions.[37] The median survival of patients with Kaposi's sarcoma alone is about 18 months, compared with seven months in those with opportunistic infections. Although bleeding may occur from Kaposi's sarcoma lesions infection is the commonest terminal event. Single and combined chemotherapy is being used with the risk of further immunosuppression and opportunistic infection, but early trials with alpha interferon look promising.[38] Other tumours such as non-Hodgkins B cell lymphomas may present *de novo,* in a setting of prodromal benign lymphadenopathy, opportunistic infection or Kaposi's sarcoma and extranodal involvement is common.[39] They involve primarily the brain, bone marrow, and gut, and response to treatment is poor.

Candidiasis is one of the commonest of the opportunistic infections.[40] Oral and oesophageal candida may present with other manifestations of AIDS or as a prelude to them. Oesophageal involvement has to be shown preferably by endoscopy to fulfil the AIDS criteria. Many patients are asymptomatic but some present with oral discomfort, dysphagia, or retrosternal burning. In severe cases there is ulceration on barium swallow. Oral nystatin, amphotericin or ketoconazole can be used in treatment but infection tends to recur.

Cryptosporidium is a protozoan only recently shown to cause diarrhoea in man, mild transient and selflimiting in the immunocompetent, severe and protracted in the immunocompromised.[41] This infection presents with weight loss and severe, watery, non-bloody diarrhoea, which can be continuous or intermittent. Abdominal pain, nausea and vomiting may also be present. The protozoan can be found in biopsy material from the

pharynx to the rectum, but infection seems to be most severe in the small bowel. There may be varying degrees of villous atrophy, but the organism does not appear to be invasive.[42] Oocytes 4–5μm in diameter can be shown by modified and acid fast stains of faecal smears, or with stool concentration methods. A variety of treatments have been used unsuccessfully, but recently some patients have responded to spiramycin.[43]

Atypical mycobacteria are ubiquitous organisms, resistant to antituberculous drugs, that show little virulence for the immunocompetent host. In AIDS disseminated infection with *M avium-intracellulare* can involve the lung, liver, lymph nodes, brain, and gut.[40] A common presentation is with unexplained fever, weight loss, lymphadenopathy, and diarrhoea. Diagnosis is made by biopsy, acid fast staining and culture, although the organisms can be cultured from blood on suitable media. There is sparse granuloma formation. The small bowel may show prominent folds with PAS positive foamy macrophages and intracellular organisms, which mimics Whipple's disease.[44]

Two members of the herpes virus group: HSV and CMV, may cause ulceration of the gut anywhere from the mouth to the anus. Cytomegalovirus may cause diarrhoea with ulceration of the colon shown by contrast studies, or colonoscopy.[45][46] Diarrhoea, diffuse ulceration, and non-specific inflammation in a young man without other features of AIDS would have to be differentiated from other causes of inflammatory bowel disease. Biopsies may show non-specific inflammation, but typical intranuclear inclusions may be seen. Cytomegalovirus can be isolated from the lesions. There is no specific treatment of proven value. Herpes simplex virus infections often present as severe, gradually enlarging mucocutaneous ulceration.[47] Involvement of the mouth, perianal skin and rectum is common and there is often a good response to acyclovir given intravenously, or orally.

Although a large number of different organisms have been associated with the diarrhoeal syndromes in patients with AIDS, many have no demonstrable pathogen in spite of intense investigation.[40] This suggests that other mechanisms, or as yet unidentified pathogens are involved. Indeed, an enteropathy has been described with jejunal abnormalities including partial villous atrophy, other non-specific changes in the small and large bowel and secretory IgA deficiency.[48] Whether such changes are primary, or secondary to some unidentified pathogen, remains to be seen.

It is plain from this review that the gastroenterologist will become increasingly involved in the diagnosis and treatment of the gay bowel and the amplification of the conditions caused by HLTV III infection. The gastroenterologist will be called upon in a caring role as a general physician, as a specialist with certain invasive techniques needed for diagnosis, and as a scientist to assist in the investigation of the pathogenic mechanisms involved and the evaluation of specific treatments.

I V D WELLER

Academic Department of Genito Urinary Medicine,
Middlesex Hospital Medical School,
London W1

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