of vaginitis and vulvovaginal symptoms include retained foreign bodies (e.g., tampons), cervical caps, vaginal spermicides, vaginal antiseptic preparations or douches, vaginal epithelial atrophy (in postmenopausal women or during prolonged breast-feeding in the postpartum period), allergic reactions to latex condoms, vaginal aphthae associated with HIV infection or Behçet's syndrome, and vestibulitis (a poorly understood syndrome).

MUCOPURULENT CERVICITIS

Mucopurulent cervicitis (MPC) refers to inflammation of the columnar epithelium and subepithelium of the endocervix and of any contiguous columnar epithelium that lies exposed in an ectopic position on the ectocervix. MPC in women represents the "silent partner" of urethritis in men, being equally common and often caused by the same agents (*N. gonorrhoeae*, *C. trachomatis*, or—as shown by case–control studies—*M. genitalium*); however, MPC is more difficult than urethritis to recognize. As the most common manifestation of these serious bacterial infections in women, MPC can be a harbinger or sign of upper genital tract infection, also known as *pelvic inflammatory disease* (PID; see below). In pregnant women, MPC can lead to obstetric complications. In a prospective study in Seattle of 167 consecutive patients with MPC (defined on the basis of yellow endocervical mucopus or ≥30 polymorphonuclear leukocytes [PMNs]/1000× microscopic field) who were seen at STD clinics during the 1980s, slightly more than one-third of cervicovaginal specimens tested for *C. trachomatis*, *N. gonorrhoeae*, *M. genitalium*, HSV, and *T. vaginalis* revealed no identifiable etiology (Fig. 163-4). More recently, a study in Baltimore using NAATs for these pathogens still failed to identify a microbiologic etiology in nearly one-half of the 133 women with MPC.

The diagnosis of MPC rests on the detection of cardinal signs at the cervix, including yellow mucopurulent discharge from the cervical os, endocervical bleeding upon gentle swabbing, and edematous cervical ectopy (see below); the latter two findings are somewhat more common with MPC due to chlamydial infection, but signs alone do not allow a distinction between the causative pathogens. Unlike the endocervicitis produced by gonococcal or chlamydial infection, cervicitis caused by HSV produces ulcerative lesions on the stratified squamous epithelium of the ectocervix as well as on the columnar epithelium. Yellow cervical mucus on a white swab removed from the endocervix indicates the presence of PMNs. Gram's staining may confirm their presence, although it adds relatively little to the diagnostic value of assessment for cervical signs. The presence of ≥20 PMNs/1000× microscopic field within strands of cervical mucus not contaminated by vaginal squamous epithelial cells or vaginal bacteria indicates endocervicitis. Detection of intracellular gram-negative diplococci in carefully collected endocervical mucus is quite specific but ≤50% sensitive for gonorrhea. Therefore, specific and sensitive tests for *N. gonorrhoeae* as well as for *C. trachomatis* (e.g., NAATs) are always indicated in the evaluation of MPC.

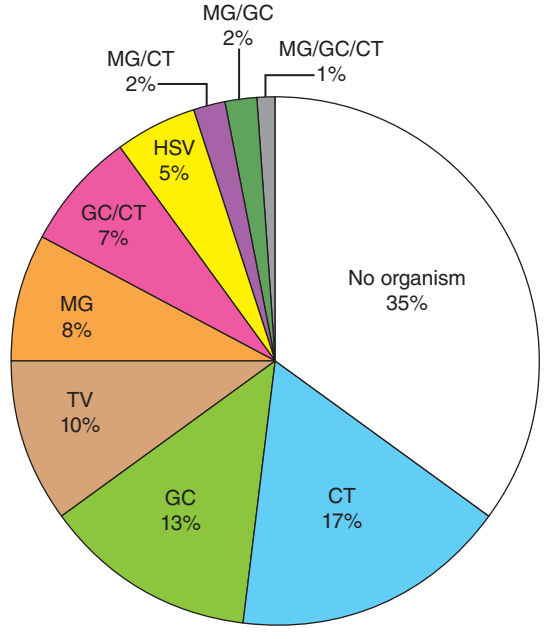

FIGURE 163-4 Organisms detected among female sexually transmitted disease clinic patients with mucopurulent cervicitis (n = 167). CT, *Chlamydia trachomatis*; GC, gonococcus; MG, *Mycoplasma genitalium*; TV, *Trichomonas vaginalis*; HSV, herpes simplex virus. *(Courtesy of Dr. Lisa Manhart; with permission.)*

TREATMENT MUCOPURULENT CERVICITIS

Although the above criteria for MPC are neither highly specific nor highly predictive of gonococcal or chlamydial infection in some settings, the 2010 CDC STD guidelines call for consideration of empirical treatment for MPC, pending test results, in most patients. Presumptive treatment with antibiotics active against *C. trachomatis* should be provided for women at increased risk for this common STI (risk factors: age <25 years, new or multiple sex partners, and unprotected sex), especially if follow-up cannot be ensured. Concurrent therapy for gonorrhea is indicated if the prevalence of this infection is substantial in the relevant patient population (e.g., young adults, a clinic with documented high prevalence). In this situation, therapy should include a single-dose regimen effective for gonorrhea plus treatment for chlamydial infection, as outlined in Table 163-4 for the treatment of urethritis. In settings where gonorrhea is much less common than chlamydial infection, initial therapy for chlamydial infection alone suffices, pending test results for gonorrhea. The etiology and potential benefit of treatment for endocervicitis not associated with gonorrhea or chlamydial infection have not been established. Although the antimicrobial susceptibility of *M. genitalium* is not yet well defined, the organism frequently persists after doxycycline therapy, and it currently seems reasonable to use azithromycin to treat possible *M. genitalium* infection in such cases. With resistance of *M. genitalium* to azithromycin now recognized, moxifloxacin may be a reasonable alternative. The sexual partner(s) of a woman with MPC should be examined and given a regimen similar to that chosen for the woman unless results of tests for gonorrhea or chlamydial infection in either partner warrant different therapy or no therapy.

CERVICAL ECTOPY

Cervical ectopy, often mislabeled "cervical erosion," is easily confused with infectious endocervicitis. Ectopy represents the presence of the one-cell-thick columnar epithelium extending from the endocervix out onto the visible ectocervix. In ectopy, the cervical os may contain clear or slightly cloudy mucus but usually not yellow mucopus. Colposcopy shows intact epithelium. Normally found during adolescence and early adulthood, ectopy gradually recedes through the second and third decades of life, as squamous metaplasia replaces the ectopic columnar epithelium. Oral contraceptive use favors the persistence or reappearance of ectopy, while smoking apparently accelerates squamous metaplasia. Cauterization of ectopy is not warranted. Ectopy may render the cervix more susceptible to infection with *N. gonorrhoeae*, *C. trachomatis*, or HIV.

PELVIC INFLAMMATORY DISEASE

The term *pelvic inflammatory disease* usually refers to infection that ascends from the cervix or vagina to involve the endometrium and/or fallopian tubes. Infection can extend beyond the reproductive tract to cause pelvic peritonitis, generalized peritonitis, perihepatitis, perisplenitis, or pelvic abscess. Rarely, infection not related to specific sexually transmitted pathogens extends secondarily to the pelvic organs (1) from adjacent foci of inflammation (e.g., appendicitis, regional ileitis, or diverticulitis) or bacterial vaginosis, (2) as a result of hematogenous dissemination (e.g., of tuberculosis or staphylococcal bacteremia), or (3) as a complication of certain tropical diseases (e.g., schistosomiasis). Intrauterine infection can be primary (spontaneously occurring and usually sexually transmitted) or secondary to invasive