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Gonorrhea: Update

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Gonorrhea is a worldwide sexually transmitted disease (STD) caused by *Neisseria gonorrhoeae*. Gonorrhea is the second most often reported STD in the United States behind chlamydia. An estimated 600,000 people each year in the United States are infected. Only about half this number of cases are reported. From 1975 through 1997, the national gonorrhea rate declined 74.3%. After a small increase in 1998, the gonorrhea rate has decreased each year since 1999. In 2003 the South had the highest gonorrhea rate among the four regions of the country. Antimicrobial resistance remains an important consideration in the treatment of gonorrhea. In 2003 the Gonococcal Isolate Surveillance Project found about 16% of collected isolates were resistant to penicillin and/or tetracycline. Since 1998 the number of ciprofloxacin-esistant isolates has been increasing with 270 (4.1%) being reported in 2003. Oral gonorrhea is rare, nonspecific, and varied and may range from slight erythema to severe ulceration with a pseudomembranous coating. The patient with gonorrhea poses little threat of disease transmission to the dentist. However, patients who have or have had gonorrhea should be approached with a measure of caution because they are in a high-risk group for additional STDs. The CDC has published recommendations for standard precautions to be followed in controlling infection in dentistry that have become the standard for preventing cross-infection. Strict adherence to these recommendations will, for all practical purposes, eliminate the danger of disease transmission between dentist and patient. Dentists should be aware of local statutory requirements regarding reporting STDs to state health officials. Syphilis, gonorrhea, and AIDS are reportable diseases in every state. Local health departments or state STD programs are sources of information regarding this matter. **(Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101:137-43)**

Gonorrhea is a worldwide sexually transmitted disease (STD) caused by *Neisseria gonorrhoeae*. Gonorrhea is the second most often reported STD in the United States behind chlamydia.[1-3] It produces symptoms in men that usually cause them to seek treatment soon enough to prevent serious sequelae but may not be soon enough to prevent transmission to others.[4] Infections in women often do not produce recognizable symptoms until complications have occurred.[1-4] Because gonococcal infections among women often are asymptomatic, an important component of gonorrhea control in the United States continues to be the screening of women at high risk for STDs.[1-4]

Transmission is more efficient from an infected male to a female partner during vaginal intercourse, and the rate is approximately 50% to 70% per sexual contact.[2] Infected women transmit gonorrhea to the urethra of their male partners at 20% per vaginal intercourse episode, and this rate increases to 60% to 80% after 4 or more exposures.[2] The difference likely reflects the size of the inoculum of *N gonorrhoeae* deposits on a susceptible mucosal surface, as well as the inherent efficiency of internal deposition of infective secretions.[5] Transmission by rectal or oral intercourse has not been quantified but appears to be efficient.[2]

INCIDENCE AND PREVALENCE

N gonorrhoeae is an important cause of sexually transmitted disease, infecting an estimated 600,000 people each year in the United States.[3] Only about half this number of cases are reported.[3] The incidence declined dramatically in most industrialized countries from the 1970s to the mid-1990s, but the rate remained stable in the United States from 1997 to 2001. The incidence of gonorrhea is lowest in Western Europe, but the disease remains epidemic in Eastern Europe, much of Africa, the Indian subcontinent, and parts of Asia and South America.[5]

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From 1975 through 1997, the national gonorrhea rate declined 74.3% following implementation of the national gonorrhea control program in the mid-1970s.[6] After a small increase in 1998, the gonorrhea rate has decreased each year since 1999.[6-10] In 2003, 335,104 cases of gonorrhea were reported in the United States.[6] The rate of reported gonorrhea in the United States was 116.2 cases per 100,000 population in 2003, (132 in 1999, 131 in 2000, 128 in 2001, 125 in 2002) which was the lowest rate of reported gonorrhea ever.[6-9] As in 2002, in 2003 only 8 states and 1 outlying area had gonorrhea rates below the Healthy People 2010 (HP2010) national target of 19 cases per 100,000 population.[6]

As in previous years, in 2003 the South had the highest gonorrhea rate among the 4 regions of the country.[6] However, the gonorrhea rate in the South has declined by 23% from a rate of 195.1 per 100,000 population in 1999 to 149.8 in 2003. In contrast, the gonorrhea rate in the West has increased by 25% from 51.3 cases per 100,000 population in 1999 to 64.0 in 2003. Rates in the Northeast (91.1 in 2003) and the Midwest (136.3 in 2003) have shown minimal change since 1999.[6] Prior to 1996, rates of gonorrhea among men were higher than rates among women. Since then, rates among women and men have remained similar. In 2003 the gonorrhea rate among women was 118.8 and the rate among men was 113.0 cases per 100,000 population.[6]

Antimicrobial resistance remains an important consideration in the treatment of gonorrhea. In 2003 the Gonococcal Isolate Surveillance Project (GISP) found about 16% of collected isolates were resistant to penicillin and/or tetracycline.[6] Resistance to ciprofloxacin was first identified in the GISP in 1991.[6] From 1991 through 1998 less than 9 ciprofloxacin-resistant isolates were found each year.[6] Since 1998 the number of resistant isolates has been increasing, with 270 (4.1%) being reported in 2003.[6] Increased resistance to azithromycin also has been reported in some regions of the country.[7,8]

The highest incidences occur in young (aged 15 to 30 years) single persons of low socioeconomic and educational attainment, in inner city residents, and in some rural settings, especially in the southeast.[5,11-13] The incidence of reported gonorrhea is 30-fold higher in African Americans than in persons of European, Asian, or Pacific Island ancestry; the rates in those of Hispanic or Native American ethnicity are 3-fold and 4-fold higher, respectively, than in nonhispanic whites.[5] The differences between racial and ethnic groups are reflections of differing sex partner network structures, socioeconomic attainment, education, and access to health care.[5]

Gonorrhea in prepubertal children older than 1 year almost always results from sexual abuse. Most infections in females cause no symptoms or only mild ones that do not lead to a health care visit. By contrast, the large majority of urethral infections in men cause overt, symptomatic urethritis, with no more than a few percent asymptomatic infections.[5]

PATHOLOGY

Gonorrhea attaches to different types of epithelial cells via a number of different structures on its surface. *N gonorrhoeae* has the ability to alter its surface structures, particularly pilin, lipo-oligosaccharide antigens, and less frequently protein 1 antigens, which helps it evade the host response. It is ingested by the cell and can cause asymptomatic or symptomatic infection.[2]

Neisseria are gram-negative diplococci that are flattened on the adjoining sides, giving the pair the shape of a coffee bean. These aerobic bacteria have stringent nutritional requirements and grow best on enriched media such as lysed sheep's blood agar ("chocolate" agar). The 2 clinically significant *Neisseria* are *N meningitidis* and *N gonorrhoeae*.[1] *N meningitidis* is a significant cause of bacterial meningitis, particularly among people between 5 and 19 years old.[1] The organism is a common colonizer of the oropharynx and is spread by the respiratory route.[1] Approximately 10% of the population is colonized at any one time, and each episode of colonization lasts, on average, for several months.[1]

Although *N gonorrhoeae* usually manifests as a local infection in the genital or cervical mucosa, pharynx, or anorectum, disseminated infections may occur. *N gonorrhoeae* is much more likely to become disseminated in people who lack the complement proteins that form the membrane attack complex (a multimolecular complex of complement C5b, C6, C7, C8, and C9, that possesses cytolytic activity for host protection).[1] Two surface proteins (pil E and pil S) of *Neisseria*, both of which adhere the bacteria to host cells, undergo antigenic variation through different mechanisms.[1] There is a single complete gene for pili in the bacterial chromosome.[1] Adherence of *N gonorrhoeae* to epithelial cells is initially mediated by long pili, which bind to CD46, a complement-regulatory protein expressed by all human nucleated cells.[1]

The expression pili loci include the regulatory elements for gene expression, as well as the entire protein-coding region.[1] There are also 10 to 15 silent pili genes in the chromosome; these encode antigenically variant pili proteins but lack the regulatory elements (promoter) as well as the DNA encoding the N-terminal domain of the protein.[1] Homologous recombination between the silent loci and the expression loci for pili shuttles variant genes into the complete loci, resulting in expression of new pili proteins.[1] Because part or all of the coding region of a silent locus may recombine