## Non-gonococcal urethritis, Helicobacter pylori infection and fellatio: a new ménage à trois?

There have been a number of organisms that are associated with sexually transmitted urethritis (see Table 1); however, the majority of non-gonococcal urethritis cases (up to 50%) are due to Chlamydia trachomatis (Shahmanesh, 2001). No other micro-organism has been shown to cause any larger proportion of the remaining non-gonococcal urethritis cases, although a large number of organisms have been isolated from the urethra of young males (Kumar et al., 1995). In addition, oral sex is a very common sexual activity with nearly  $80\,\%$  of single whites and almost  $50\,\%$  of African Americans engaging in the activity, although variations in the prevalence of oral sex exist due to differences in gender, marital status, race and ethnicity (Billy et al., 1993). Helicobacter pylori, a gastric organism discovered more than 20 years ago, is responsible for chronic gastritis and peptic ulcer disease and is associated with an increased risk of developing stomach cancer (Eslick et al., 1999).

**Table 1.** Organisms that cause or are associated with sexually transmitted urethritis

Organism	No. of cases (%)
Gonococcal	
Neisseria gonorrhoeae	
Non-gonococcal	
Chlamydia trachomatis	15-50
Ureaplasma urealyticum	10-40
Other	20-40
Mycoplasma genitalium	15-25
Trichomonas vaginalis	Rare
Yeasts	Rare
Herpes simplex virus	Rare
Adenoviruses	Rare
Haemophilus sp.	Rare
Other bacteria? (Helicobacter)	Unknown

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*H. pylori* infection has also been implicated in numerous enterohepatic and extragastric diseases (Eslick *et al.*, 2002; Solnick & Schauer, 2001; On *et al.*, 2002).

The aim of this article is two-fold: firstly, to postulate that *H. pylori* may be transmitted sexually through the act of fellatio and thus, secondly, to highlight the possibility that *H. pylori* may be another micro-organism responsible for urethritis among males.

The idea that *H. pylori* or another species of *Helicobacter* could cause urethritis has never before been proposed. There have been three conflicting studies conducted to determine if sexual contact plays any role in the transmission of *H. pylori* (Aceti *et al.*, 1987; Polish *et al.*, 1991; Perez-Perez *et al.*, 1991). These studies were inadequate in terms of sample size, study design and causal information regarding sexual practices by the participants.

Non-gonococcal urethritis is predominantly developed through sexual activities, although rare reports exist of non-sexual transmission (Kleist & Moi, 1993; David, 1997). The sexual routes for the transmission of urethritis among males include vaginal intercourse, anal intercourse and oral sex (Martinez-Garcia et al., 1996; Jensen et al., 1993; Burstein & Zenilman, 1999). Moreover, uropathogens including Escherichia coli have been shown to be sexually transmitted between sex partners (Foxman et al., 1997). Oral sex (fellatio) is one of the main routes for the spread of common oral flora (Group A β-haemolytic streptococci, Neisseria meningitidis) causing pathology (Fisk & Riley, 1995; Edwards & Carne, 1998). Studies have reported that 0-90 % of H. pylori-infected individuals may permanently/transiently carry H. pylori in their mouth and saliva, from which further infections may arise (Dowsett & Kowolik, 2003; Thomas et al., 1997). The bacterial load of H. pylori in saliva has not been estimated; however, if techniques like PCR can detect low numbers of the bacteria then this suggests that H. pylori may exist in sufficient quantity to have pathogenic consequences (Dowsett & Kowolik, 2003).

What about the urethral tissue - can

H. pylori live or colonize in this location? It must be remembered that *H. pylori* does not invade the tissues of the host to any great extent, it is a mucosal pathogen. The urethral epithelium varies depending on the location within the urethra. For example, the navicular fossa can have either stratified squamous or stratified columnar ciliated epithelium (Sternberg, 1997). H. pylori usually colonizes the gastric columnar epithelium, but can survive on the squamous epithelium of the mouth (tongue, cheek, palate) (Thomas et al., 1997; Parsonnet et al., 1999). Thus, H. pylori could inhabit the urethra and perhaps colonize the tissues leading to inflammation and/or pathology.

The natural infective dose of *H. pylori* is unknown. Based on the analogy of other enteric pathogens (*Shigella*, *Giardia lamblia*, *Entamoeba histolytica*) it has been suggested that the infectious dose of *H. pylori* is small (Feldman *et al.*, 1998). If the minimum infective dose of *H. pylori* is small, then the chance of a urethral infection is greater than if a high inoculum were needed. One would suspect that low doses of *H. pylori* should be sufficient to causes urethritis given the correct environmental conditions of temperature and pH.

If H. pylori can survive in the extreme acidic environment of the stomach, can it live in the urethra with acidic/neutral or even alkaline urine flowing through it? Based on previous studies of H. pylori and environmental pH it is possible for H. pylori to survive in pH ranges between 2.2 and 7.2 (Clyne et al., 1995). Moreover, the optimal pH for H. pylori survival is between 4.5 and 7.0 which is also the pH range for human urine. The urinary tract may provide an ideal environment for H. pylori to thrive in once colonization has been established. Will the flow of urine affect the colonization of H. pylori? I suspect no more than any other sexually transmitted infection. In the stomach, H. pylori adheres to gastric epithelium using adhesins interacting with the host-cell receptor (Noach et al., 1994). It does this to avoid being washed away into the intestines by peristalsis. The urethra should support *H. pylori* (microaerophilic) just as it does a number of other organisms (i.e. lactobacilli, enterococci,  $\beta$ -haemolytic streptococci) (Kumar et al., 1995).

In conclusion, oral sex is one of the most common sexual practices in the world and it is possible that H. pylori could be transmitted via the act of fellatio to the urethra leading to infection. This organism may be the 'missing link' in explaining the large proportion of males with non-gonococcal urethritis where no other responsible organisms can be isolated. This is the first article to suggest a link between H. pylori infection and urethritis. Studies will be required to determine if H. pylori is transmitted via oral sex to the urethra; what, if any, interactions might occur with other uropathogens; if any pathologies arise from such an association [e.g. prostate (prostatitis), epididymitis, seminovesculitis, testicles (orchitis, cancer) and urinary calculi)]; and potential treatment modalities.

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