Editorial

WEAR GLOVES TO PROTECT NOT ONLY YOUR HANDS BUT ALSO YOUR STOMACH DURING UPPER GI PROCEDURES!

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In this issue of Marmara Medical Journal, Tözün et al. (1) evaluate seroprevalence of H. pylori infection in endoscopists and ICU nurses. Although they found higher prevalences in endoscopy and ICU personnel with respect to controls, the difference did not reach to statistical significance. They propose that in areas with H.pylori endemicity, where acquisition occurs at a relatively earlier age, neither endoscopy nor ICU care increase the acquisition of H.pylori by close contact with patients.

Endoscopy and ICU personnel may be exposed to gastric secretions more frequently during upper GI endoscopy or nasogastric intubation and aspiration procedures. Furthermore, several studies have shown instrument contamination after the gastroscopy of H.pylori infected patients (2-4). Handling these intubated instruments, especially when they are inadequately disinfected, with bared hands may increase the hazard of transmission of H.pylori.

Although several studies indicated higher prevalence of H.pylori infection in endoscopists, particularly in those who did not wear gloves (5,6), not all studies were conclusive. As Tözün et al. (1) study, most studies from developing countries with high H. pylori overall prevalence suggested that the infection rate in endoscopists was not significantly higher than in non-endoscopists (7). However, studies from countries with high H.pylori infection rates need large sample sizes to be able to prove a prevalence difference between various groups. Tables, nomograms and computer programs have been developed to estimate the sample size according to requirements of the study design (8-10).

For the clinical studies, the type I error level usually set at 5 %, and the type II error level, whose complement to unity represents the power of the study. The latter is the ability to demonstrate a difference, when this difference does exist, at least at a level judged to be possible and of clinical importance. For example, according to a type I error level of 5% and a power of 95%, if H.pylori prevalence is 20% in the control group and if you expect an 80% rate within the risk group, you need about 16 people per group. But if the prevalence is 90% within the control, approximately 1500 people should be enrolled to the study to demonstrate a prevalence greater than 95% in the risk group.

However, negative results were also published from developed countries with lower H.pylori infection rates (11, 12). But in one of these studies, a significant difference was found between physicians and nurses, when compared to controls who had no patient contact (12). This result indicates that, if the primary cutcome is highly related to a patient characteristic, a so-called risk factor, stratification should be used in the experimental design of the clinical trial.

As Tözün et al. (1) implied in their study, H.pylori infection is frequently acquired early during childhood years in Türkiye and probably will continue to be a major health problem in the future years.

Although eradication of H.pylori with antibiotics is an accepted practice in peptic ulcer patients, therapeutic intervention for other H.pylori infected people remains more controversial. Because; no one regimen with optimal efficacy, safety and compliance has yet been established; these people are frequently asymptomatic, so any therapeutic intervention will necessitate screening programs; in addition, high drug costs, unjustifiable risk of side-effects and risk of increasing the incidence of drug-resistant strains, thus rendering the current successful therapies useless make therapeutic intervention impractical. And, again, in developing countries there may be a high reinfection risk in contrast to developed countries.

Just as, in a pilot interventional trial in Venezuela. H.pylori eradication was achieved in only 6.5 % of the treatment group compared to 2% of the placebo group suggesting that either the therapy was ineffective or that reinfection rates were very high (13). So control and prevention remain the most meaningful strategy, as the public pressure increases for large-scale treatment against Hp, especially in populations in which gastric cancer continues to be a significant problem.

But, it should be noted that any prevention programme would have to be a very long-term strategy as infection is thought to be acquired early in

life and prevention would, primarily, therefore have to be targeted at children. It would be 20-50 years or more before any reduction was seen for example in the rates of peptic ulcer or gastric cancer which are primarily diseases of adults or elderly, respectively.

Secondly, it is worth noting that H.pylori infection rates are already declining at least in developed countries and any measures designed to further reduce these rates would have to be very cost-effective to achieve more than is already occurring in the absence of any planned intervention.

Thirdly, if the apparent reduction in infection rates in developed countries has resulted from improvements in living and sanitation conditions, it is unlikely that further improvements will occur sufficiently quickly to bring rates much lower. Moreover, the developing countries that would benefit most from such improvements are probably least able to afford to implement them.

In anyway, there may be two options for prevention of infection; 1. Reducing exposure to the organism. 2. Vaccination against the organism.

The first option, reducing exposure to the bacteria, is unlikely to be a viable option in the foreseeable future. Because; practically, human is the most important reservoir, and it is nearly impossible to eliminate carrier state with current therapies; we do not know accurately the infectious dose, the exact mode of transmission, and the role of environment as a reservoir, yet; although improvements in economic development and hence a better standard of living and hygiene have paralleled reductions in H.pylori infection in birth cohorts, these evolvements need time, education and money.

The second strategy for prevention of infection, prophylactic vaccination would also have some difficulties. First of all, for any prophylactic vaccination schedule risk groups should be defined and screening programmes have to be implemented; and secondly, any vaccine should be cost-effective. A cost-benefit analysis showed that costs depend heavily on the anticipated trends in H.pylori infection rates and they suggest that vaccine in children, in the developed world would take many decades to be cost effective; no analysis, however, was done on the cost-benefit of use of vaccine in areas where there are high infection rates in children.

Animal models suggest that effective immunization is possible and vaccine can also help to eradicate already established infection. Although it is not fully known how vaccine works and there are still problems related to protective immunogen and adjuvant etc. (14), if these studies can be confirmed in human models and it is proved to be effective in eradication of H.pylori infection, especially with no reinfection risk, besides its preventive potential in noninfected people, it may be indicated in all H.pylori infected patients including GI staff, either as a primary or adjunctive therapy.

REFERENCES

1. *Tözün N, Durademir A, Avşar E, et al. Prevalence of Helicobacter pylori in endoscopists. Marmara Med J. 1997;10:33-35.*
2. *Frickner CR. Adherence of bacteria associated with active chronic gastritis to plastics used in the manufacture of fiberoptic endoscopes. Lancet 1984;1:800.*
3. *Fantry GT, Zheng 0-X, James SP. Conventional cleaning and disinfection techniques eliminate the risk of endoscopic transmission of helicobacter pylori. Am J Gastroenterol 1995;90:227-232.*
4. *Tytgat GNJ. Endoscopic transmission of Helicobacter pylori. Aliment Pharmacol Ther 1995; 9 (suppl. 2): 105-110.*
5. *Mitchell HM, Lee A, Carrick J. Increased incidence of Campylobacter pylori infection in gastroenterologists: further evidence to support person-to-person transmission of C. pylori. Scand J Gastroenterol 1989;24:396-400.*
6. *Morris A. Lloyd G, Nicholson G. Campylobacter pyloridis serology among gastroendoscopy clinic staff. NZ Med J 1986;99:819-820.*
7. *Matysiak-Budnik T, Gosciniak G. Brugmann D, et al. Seroprevalence of Helicobacter pylori infection in medical staff in Poland. Eur J Gastroenterol Hepatol 1994:6:309-311.*
8. *Casagrande JT, Pike MC, Smith PG. The power function of the exact test for comparing two binominal distributions. Appl Statist 1978;27:176-180.*
9. *Altman DG. Statistics and ethics in medical journals: III How large a sample? Br Med J 1980;281:1336-1338.*
10. *Dupont WD, Plummer WD Jr. Power and sample size calculations: a review and computer program. Controlled Clin Trials 1990:11:116-128.*
11. *Pristautz H, Eherer A, Brezinschek R, et al. Prevalence of Helicobacter pylori antibodies in the serum of gastroenterologists in Austria. Endoscopy 1994;26:690-696.*
12. *Braden B, Duan LP, Lembcke B. Caspary WF. Upper GI endoscopy is not a risk factor for Hp infection-but medical practice is. Gastroenterology 1994:106: A 56.*
13. *Buiatti E, Munoz N, Vivas J, et al. Difficulty in eradicating Helicobacter pylori in a population at high risk for stomach cancer in Venezuela. Cancer Causes and Control 1994, 5:249-254.*
14. *Lee A. Therapeutic immunization against Helicobacter infection. Gastroenterology 1996;110:2003-2006.*