Pattern of helicobacter pylori resistance to antibiotics in Saudi Arabia

Seari,

The weight of scientific literature is in support of helicobacter pylori having a causal association with active chronic gastritis, gastric and duodenal ulcer, non-ulcer dyspepsia and possibly gastric carcinoma.1,2,3 With the recognition of the importance of H. pylori in the pathogenesis of gastritis and peptic ulcers, treatment options have focused on the eradication of the organism. This therapeutic approach has shown that eradication of the organism leads to improvement in the clinical condition of the patients and significant reduction in the recurrence rates and symptoms of the preceding diseases.2,3,4

For some years now the mainstay of therapy to eradicate H. pylori has been the use of bismuth salts together with antimicrobials either singly or in combination. The most commonly used agents are metronidazole, amoxicillin and tetracycline.2,3,4

Many factors have been reported to affect the clinical efficacy of antimicrobials in the treatment of H. pylori, such as insufficient concentration of active drugs in gastric mucosa, instability of some agents in an acidic pH, inappropriate formulation of the drug; insufficient duration of treatment, variable compliance of patients and rapid acquisition of resistance to antibiotics.1 H. pylori demonstrates in vitro susceptibility to a large number of antimicrobial agents such as penicillins, cephalosporins, aminoglycosides, macrolides, tetracyclines, rifampicin and metronidazole.2 However, very recently primary resistance to metronidazole has been reported in strains of H. pylori isolated from patients. In Europe, resistance was found to range from 6-27% and was higher in women (probably related to prior use of metronidazole to treat gynecological infections). On the other hand, in Africa, where metronidazole is widely used to treat gastrointestinal infections, resistance to metronidazole was found to be 84% with no difference reported between women and men.3

In Saudi Arabia, metronidazole is commonly used to treat giardiasis, amebiasis and gynecological infections. We therefore decided to determine the susceptibilities of H. pylori isolated in our hospital to metronidazole and some of the common antibiotics used in the therapy of H. pylori.

Patients attending our Gastroenterology Unit who had endoscopic examination had gastric biopsy obtained for histological and microbiological examination and culture. H. pylori was isolated using a chocolate plate and Columbia sheep blood agar with incubation under microaerophilic conditions for 72 hours. H. pylori isolates were identified by characteristic colonial appearance, Gram stain, oxidase test, catalase test and urease test. Isolates were stored frozen at -20 degrees C until ready for sensitivity testing. Sensitivity testing was performed on 215 isolates by the Kirby-Bauer technique4 by inoculating a plate of Mueller-Hinton 5% blood agar with a saline suspension of the organism adjusted to McFarland 0.5 turbidity (corresponding to 106 CFU). The following antibiotics discs were applied to the sensitivity plate viz. metronidazole 5 mg; ampicillin 10 mg; erythromycin 15 mg; gentamycin 10 mg; rifampicin 5 mg; tetracycline 30 mg. Inhibition zone diameters were measured after 48 and 72 hours incubation at 37 degrees C under microaerophilic conditions.5,6 Isolates giving a zone diameter of 30 mm or more were regarded as sensitive while those giving 29 mm or less were regarded as resistant. Controls were included in each batch of tests.

Of the 215 isolates tested by this technique, 142 (66.1%) were resistant to metronidazole, 3 (1.4%) to tetracycline, 6 (2.8%) to erythromycin, 16 (4.7%) to rifampicin and 4 (1.9%) were found resistant to ampicillin.

The resistance of our isolates mainly from residents of Jeddah area appears to be higher than that reported in Europe but less than that in Africa.3 It has been reported that in vivo resistance of H. pylori to metronidazole develops very readily when monotherapy is used, while the use of combination therapy (double or triple) reduces the risk of development of antibiotic resistance.4 Clearly it is desirable that in our locality the sensitivity of H. pylori must be determined, so that appropriate antibiotics could be prescribed if eradication of the organism is the desired goal. As far as we are aware this is the first report on the antibiogram of H. pylori in the Kingdom. There is therefore a need for similar studies to be carried out in other centers so that the overall picture in the Kingdom can be ascertained. Current literature shows there is a growing importance in the determination of the susceptibility of H. pylori to antimicrobial agents, since it appears that primary or acquired resistance to various drugs may be
responsible for failure to eradicate the organism from the gastric mucosa.  

References


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