

Special Article - *Helicobacter pylori*

Biological Polymorphisms of *Helicobacter pylori* on Drug-Susceptibility Test in Clinical Laboratory

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Letter to Editor

The drug-susceptibility test is necessary and important examination to perform effective antibiotic therapy for individual infectious disorders irrespective to acute and chronic infections. The methodology for drug-susceptibility test in clinical laboratory crucially contributes to select the effective antibiotics in the regimen. Routinely, a strain of the microorganisms causing the infectious disease is subjected to the drug-susceptibility test in clinical laboratory according to application of health issuance and cost-performance. However, the treatment with the antibiotics chosen based on the drug-susceptibility test provides not always successful outcome. Concerning with the importance of the problem, we generally accept the bacteria and host-related factors as follows; minor antibiotic resistant which is not detected and poor adherence and individual pharmacokinetics/dynamics in the body.

One of the chronic infectious disorders, the persistent *Helicobacter pylori* infection in the human stomach causes various manifestations of gastro and extra-gastric disorders as reviewed [1,2]. *H. pylori* is acquired in childhood and the stable colonization continues lifelong unless treated successfully [3]. *H. pylori* possesses a high genetic diversity/rearrangement, and is persistently colonizing in the stomach due to adapt to the microenvironment of stomach according to the change in the circumstances. The persistent *H. pylori* infection has been made possible by the coexistence of a variety of strains consisting with newborn mutant strains as a flexible *H. pylori* community (*H. pylori* flora) in individual stomachs, leading to biological polymorphism of *H. pylori* in the stomach [4,5]. Nowadays, the eradication therapy is performed worldwide, however, the eradication failure with antibiotics based on the drug-susceptibility test using a strain is increasing and is worthy of note. Thus, many scientists and medical practitioners are struggling to develop the best regimen for treatment of *H. pylori* infection. They are investigating to figure out more effective combination therapy with antibiotics and gastric acid secretion inhibitors via the randomized clinical trials [6]. Simultaneously the drug-susceptibility test before antimicrobial prescription should be performed with due consideration for the characteristics of the examinations and bacterial features. The

difference of drug-susceptibility tests e.g. E-test and agar dilution sometimes differs the values of Minimum Inhibitory Concentration (MIC) [7,8]. Furthermore, in particular, *H. pylori* utilizes the high proper adaptation strategies mentioned above and survives as *H. pylori* flora in the stomach [4,5], which probably affects the results of the drug-susceptibility test. In fact, in the case of eradication failure, the discrepancy between the result from the drug-susceptibility test and clinical outcome is reported [6,9-11]. When a single strain is used in the examination, it is hard to provide the accurate result/MIC value for the effective antibiotic therapy. Thus, we are analyzing with at least each 10 strain from individual patients to clarify the biological polymorphism of *H. pylori* on the drug-susceptibility. Four antibiotics such as clarithromycin, amoxicillin, metronidazole and sitafloxacin, were used in the drug-susceptibility test. So far, the diversity of MIC value among the multiple strains in the stomach is confirmed by the drug-susceptibility test in clinical laboratory. Interestingly, antibiotic-sensitive and -resistant strains coexisted even in a stomach. Moreover, the MIC values differ among the resistant strains in a stomach. These results seem to relate with the anatomical site where *H. pylori* exists and the history of antibiotic therapy. We need more investigations with the increased cases including primary treatment and eradication failure to clarify the effect of such biological polymorphism of *H. pylori* on the drug-susceptibility.

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