ANTIBIOTIC RESISTANCE OF HELICOBACTER PYLORI IS THE CURRENT STATE OF THE PROBLEM

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Introduction. Helicobacter pylori (HP) is the most common infection, the carrier of which is almost every second person in the world. The standard of treatment for diseases of the gastrointestinal tract associated with HP is anti-helicobacter therapy, which includes a combination of a proton pump inhibitor and two antibiotics, which are reflected in international and Ukrainian recommendations for the treatment of gastroenterological patients. Unfortunately, the effectiveness of this therapy is constantly decreasing, which is associated with an increase in the number of HP strains resistant to metronidazole or clarithromycin. This is due to the fact that HP has the ability to form a biofilm, which contributes to the resistance of the bacterium to antibiotic therapy and protects it from the host's immune response. Antihelicobacter therapy can be successful only if the resistance of HP in the region does not exceed 10-12% for clarithromycin, 30% for metronidazole.

Materials and methods. The purpose of the study was to study the current state of antibiotic resistance in Europe. Antibiotic resistance of H. pylori in Europe has been monitored every 10 years since 1998. In 2018, an observational multicenter study was conducted to assess the prevalence of H. pylori antibiotic resistance in Europe using a common standard protocol. Cases of primary antibiotic resistance of H. pylori were studied in 24 medical centers in 18 European countries.

Results and their discussion. The results of an observational multicenter European study showed that the frequency of antibiotic resistance (among 1211 adult patients) was 21.4% for clarithromycin, 15.6% for levofloxacin, and 38.9% for metronidazole. At the same time, the level of resistance was higher in the countries of Central, Western and Southern Europe compared to the countries of Northern Europe.

This study confirmed a significant association between antibiotic resistance to clarithromycin and the use of macrolides in the population (p=0.0003), as well as between antibiotic resistance to levofloxacin and the use of fluoroquinolones (p=0.0002) and second-generation fluoroquinolones.

The results of this study confirmed that the antibiotic resistance of H. pylori grows proportionally with using macrolides and fluoroquinolones in this region. These data suggest that HP eradication therapy with clarithromycin and levofloxacin should not be started without determination of antibiotic sensitivity.

Conclusions. The obtained results indicate the importance of rational use of antibiotics and avoiding their unnecessary use. The conducted research is important for

optimizing the therapy of Helicobacter-associated diseases and continuing the use of standard eradication therapy.

VIRUS-VIRUS INTERACTION

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Introduction. Several respiratory viruses can circulate during the same period and can concurrently or sequentially infect the respiratory tract, leading to virus–virus interactions. At the host level, the course of infection of 1 virus might be influenced by prior or concurrent infection by another virus. Infection by a first virus could enhance or reduce infection and replication of a second virus, resulting in positive (additive or synergistic) or negative (antagonistic) interaction.

Positive virus–virus interaction corresponds to a co-infection that might result in an increased disease severity and pathogenesis (e.g., severe acute respiratory syndrome coronavirus 2 [SARS-CoV-2] and influenza A[H1N1]). Negative virus-virus interaction can be homologous or heterologous depending on whether the 2 viruses belong to the same family or to different serotypes or families. Homologous virus-virus interaction implies that cross-reactive immunity against a first virus prevents infection with a second virus (e.g., among different influenza subtypes or lineages). Heterologous viral interference relies on induction of a nonspecific innate immune response by a first virus that reduces or prevents infection and replication of a second virus (e.g., influenza A virus [IAV] and respiratory syncytial virus [RSV]). The type of virus-virus interaction (negative or positive) is probably dependent on the respiratory viruses involved, the timing of each infection, and the interplay between the response of the host to each virus. In this perspective, we focus more specifically on viral interference.

Materials and Methods. A literature search was conducted in Google Scholar, PubMed, Scopus and Web of Science databases.

Results and Discussion. The more probable mechanism of negative viral interactions relies on the induction of a transient innate immunity by the interfering virus. Structural components of viruses are sensed by pattern recognition receptors in epithelial and immune cells. This recognition triggers the expression of interferon (IFN) – stimulated genes (ISGs) and type I (i.e., IFN-α/β) and type III (i.e., IFN-λ) IFNs. The IFN-α/β receptor is expressed on most cell types, whereas the IFN-λ receptor is predominantly present on epithelial cells of the gastrointestinal and respiratory tracts. Secreted IFNs bind to receptors present at the surface of infected and neighboring cells to amplify the expression of ISGs. This process leads to an antiviral defense program consisting in the