Research Journal of Medical Sciences 9 (2): 39-42, 2015
ISSN: 1815-9346

The Modern View of the Issue of *Helicobacter pylori* Infection

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Abstract: The modern view of the issue of *Helicobacter pylori* infection is considered. It was demonstrated that *Helicobacter pylori* is the opportunistic pathogen of the mucosal stomach flora and oral mucosa. The secondary reservoir of *Helicobacter pylori* is the oral cavity. The treatment of the *Helicobacter pylori* associated diseases shall be combined, individualized, ethiopathogenic, substantiated, consistent, dynamic, expectant. By treatment of the *Helicobacter pylori* associated oral mucosa diseases immunomodulators shall be prescribed, remedial measures aimed at sanation of the chronic centers of infection, equalization of the microbial landscape of both the oral and stomach mucosa shall be taken.

Key words: *Helicobacter pylori*, mucosal stomach microflora, eradication, dysbacteriosis, oral mucosa diseases, local immunity of oral mucosa

INTRODUCTION

As of today it is known that Helicobacter pylori (*H. pylori*) is the microaerophilic, small, non-spore-forming gram-negative bacterium of primarily S-form with the length 2.5-4 μm and the width 0.5 μm. On the one pole there are 4-7 flagella 5-10 μm long that ensure the bacterium ability for active movement (Aebischer, 2010; Wadstrom *et al*., 1996). Flagella are covered by sheathes reaching up to 30 nm in width at the end of which there are club thickenings. Vegetative and spiral forms of *H. pylori* with flagella may be transformed into ovoid or coccus forms. This more frequently occurs in older cultures or under unfavorable conditions (Zimmermann, 2011). According to findings of the molecular-biological studies, the 'age' of *H. pylori* does not exceed 10-11000 years (Krylov and Myths, 2006).

For long it was generally, thought that due to the low pH a human stomach does not contain microorganisms. The main role in the development of gastritis and ulcer disease was assigned to the hydrochloric acid, diet violations and psycho-physical loads (Schwarz, 1910).

For the first time the concept of the infectious onset of the ulcer disease appeared at the end of the 19th century and the confirmation, thereof were spiral bacteria found in the human and animal stomach. The first one to find them in 1874 in a canine stomach was Bottcher Zimmermann (2013) and Krienitz (1906) found the similar spiral bacteria in the gastric juice and autopsy tissue of a human stomach. Rosenow and Sanford (1915) in their researches confirmed presence of the curved bacteria on the surface of the human stomach mucosa (Krienitz, 1906; Rosenow and Sanford, 1915).

In 2005, Barry J. Marshall and Robin Warren were awarded the Nobel Prize in medicine and physiology for discovery of the *H. pylori* bacterium and its role in the development of gastritis and ulcer diseases, development of the first eradication schedules. The studies of these two researchers have proved that a stomach is not sterile; microorganisms can exist in an acid environment. *H. pylori* produce a lot of enzymes decomposing urea to ammonia and carbon dioxide that form a thick protective layer around bacteria (Mayev and Samsonov, 2006; Amieva and Omar, 2008).

MAIN PART

By now the modern methods of microbiological study have proved that the amount of various mucosal microflora in the stomach of healthy people makes 10^3-10^4 mL^{-1} (3 Ig CFU g^{-1}) including in 44.4% of cases there was diagnosed *H. pylori* (5.3 Ig CFUKOE g^{-1}) in 55.5% streptococci (4 Ig CFU $Г^{-1}$) in 61.1% staphylococci (3.7 Ig CFU $Г^{-1}$) in 50% lacto bacteria (3.2 Ig CFU $Г^{-1}$) in 22.2% candida fungi

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(3.5 Ig CFU Γ^{-1}). Besides, there have been screen out bacteroids, corynebacteria, micrococci in the amount 2.7-3.7 Ig CFU g^{-1}. It shall be noted that *H. pylori* was determined only in association with other bacteria. The stomach environment appeared to be sterile in healthy people only in 10% of cases (Blaser *et al*., 2008; Kon'kova, 2012; Roos, 2005).

By different diseases (chronic gastritis, ulcer disease and stomach cancer) the number and variety of bacteria colonizing the stomach are increased significantly (Bondarenko, 2007; Halme *et al*., 2008). By chronic gastritis the most of mucosal microflora was found in the antral part by ulcer diseases in the periulcerous area (in the inflammatory cushion). It is often that it is not *H. pylori* but streptococci, staphylococci, enterobacteria, micrococci, lacto bacteria, candida fungi that prevail (Zimmermann, 2011; Halme *et al*., 2008; Sasaki, 2012).

Thus, *H. pylori* is an opportunistic microorganism of the mucosal stomach microflora. According to the famous researcher Blaser *et al*. (2008), *H. pylori* is a part of the human microbiocenosis and depending on the particular conditions (circumstances) may act both as commensals and pathogens. He believed that there is kind of homeostasis between *H. pylori* and human organism: as long as they not try to kill it, it does not do any harm to the 'host' (Blaser *et al*., 2008).

A man acquires it in the early childhood, usually during the first year of his life. The transmission route fecal-oral, oral-oral and vomit-oral. After entering the stomach, upon absence of administration of antibiotics *H. pylori* persists for decades in most cases during the entire life of the host. *H. pylori* strains feature great variability, the interaction of macro organism and bacteria is affected by different sites of *H. pylori* genes (Chereshnev, 2006).

The secondary reservoir of *H. pylori* is the oral cavity (Kaspina, 2003). *H. pylori* was found in saliva, soft calcular deposits, purulent content of gingival pockets on the dental prostheses (Kaspina, 2003; Arutyunov, 2005). There was noted the correlation between presence of *H. pylori* in the oral cavity and its poor hygienic condition (Anand, 2006; Salmanian *et al*., 2008).

Presence of *H. pylori* in the gastrointestinal tract is not the ground for immediate therapy with the use of antibiotics or other drugs (Blaser *et al*., 2008). The strategy of the Maastricht conference of 2010 'test and treat' suggesting prescription of eradication in bacteria carriers is absolutely not unreasonable and insubstantial as 3.5-4 billion of healthy people infected with *H. pylori* cannot be subjected to treatment. Moreover, mass eradication of the microorganism in healthy people will inevitably result in the catastrophic and irreversible spreading of *H. pylori* strains resistant to antibiotics therapy (Zimmermann, 2013; Mayev *et al*., 2006).

By eradication of *H. pylori* we mean complete destruction of the vegetative and coccus microorganism forms proven 4-6 weeks after completion of the therapy by at least two diagnostic methods (urea tests, histologic examination of the gastric biopsy specimen, bacteriological method) (Mayev *et al*., 2006; Jafri *et al*., 2008; Uberti *et al*., 2013). Eradication includes at least two antibacterial drugs (for example, Clarithromycin 500 mg and Amoxicillin 1000 mg twice a day) during 7-10 days. There have been designed over 120 schedules of eradication therapy. However, none of them is 100% efficient (Kim, 2013; Ozturk, 2013; Zullo *et al*., 2007).

Besides, prescription of eradication is often not followed by prescription of pre and probiotics (Zheng, 2013; Zou, 2009). In some cases, mono-antibacterial therapy is still used. Metronidazole is often included in the treatment schedule; resistance of *H. pylori* to it in Russia makes 70-90% (Goh, 2011).

An important aspect of assignment of anti-helicobacter therapy includes adverse effects and in general the tolerance and safety of the massive antibiotic therapy causing allergic, toxic and dysbiotic changes in the human body (Mayev *et al*., 2006). The antibacterial therapy is the cause of development of up to 30% of all adverse effects related to administration of drugs (Rafalsky, 2007). In 30-40% of patients the adverse effects in the form of the colonic dysbiosis and antibiotics-associated diarrhea (Krylov and Myths, 2006). It is known that diseases of the gastrointestinal tract and liver are often accompanied by changes in the oral cavity. This is explained by the morphofunctional similarity of the oral and gastrointestinal mucosa. Besides, the oral mucosa is a wide receptive field receiving the reflex influence from any inner organ (Lukinykh *et al*., 2004). A patient visits a doctor after eradication with complaints of dryness, burning of the oral mucosa, taste perversions, bitter flavor in the mouth, bad breath.

The consequences of destruction of *H. pylori* can be seen in the oral cavity these are xerostomia, candidiasis, andidosis, leptotrichosis, benign migratory glossitis, hyperplasia of clavate papillae, chronic recurrent ulcerative stomatitis.

SUMMARY

Based on the foregoing we believe that eradication with the use of antibacterial agents is expectant treatment. Ethiopathogentic treatment of *H. pylori* is improvement of the local and systemic immunity, sanation of the chronic

centers of infection (chronic tonsillitis, chronic pharyngitis, etc.) and first of all, sanation of the oral cavity, professional hygiene of the oral cavity, rational teeth replacement, rational hygiene of the oral cavity. In light of the data provided, the following is of utmost importance today:

- To diagnose the oral mucosa diseases, their occurrence rate and severity in patients depending on the *H. pylori* content in the stomach
- To investigate the indicators of the local immunity of the oral cavity: secretory IgA, IgA, IgG, IgM, Liz activity level, coefficient of balance of the local immunity factors in patients infected with *H. pylori*
- To investigate the effect of the professional oral cavity hygiene on the efficiency of the combined, individualized ethiopathogenetic treatment and (or) prevention of the *H. pylori* associated diseases of the oral mucosa. To define the place and role of the rational and professional oral cavity hygiene in this category of patients
- To design and to implement on the basis of microbiological and immunological data obtained the schedule of the combined, individualized ethiopathogenetic treatment and (or) prevention of the *H. pylori* associated diseases of the oral mucosa

CONCLUSION

Thus in this literature review, we have combined the historical aspects and our view of the issue of the *H. pylori* infection, pointed out to the necessity of development of the individualized ethiopathogenetic treatment and/or prevention of Helicobacteriosis aimed at improving the quality of the patient's life.

REFERENCES

Aebischer, T. 2010. Inflammatorion Immunity and Vaccines for Helicobacter. Meyer, T. and L. Andersen (Eds.) Helicobacter., 15 (1): 21-28.

Amieva, M.R. and E.M. Omar, 2008. Host-bacterial interactions in *Helicobacter pylori* infection. Gastroenterology, 134 (l): 306-323.

Arutyunov, S.D., 2005. Peculiarities of the state of the periodontal tissue in patients with the *Helicobacter pylori* associated ulcer disease. Mayev, I.V. and N.V. Romanenko (Eds.). Periodontology, No. 3, pp: 30-33.

Anand, P.S., 2006. Are dental plaque, poor oral hygiene, and periodontal disease associated with *Helicobacter pylori* infection?. Nandakumar, K. and K.T. Shenoy (Eds.). Periodontol., 77 (4): 692-698.

Blaser, M.J., Yu Chen and J. Reibman, 2008. *Helicobacter pylori* a friend or a foe? Health of Ukraine, 6 (1): 70-71.

Bondarenko, V.M., 2007. Intestinal dysbacteriosis as a clinical-laboratory syndrome: status update of the problem. M: GEOTAR-Media, pp: 302.

Chereshnev, V.A., 2006. Biological laws and human life and activities (method of multifunctional restorative bio-treatment). Perm: GSHA, pp: 300.

Goh, K.L., 2011. High *Helicobacter pylori* resistance to metronidazole but zero or low resistance to clarithromycin, levofloxacin and other antibiotics in Malaysia. Helicobacte, 16 (3): 241-245.

Halme, J. Arola, K. Hockerstedt and I. Lautenschlager, 2008. Human herpesvirus 6 infection of the gastroduodenal mucosa L. Clin. Infect. Dis., 46 (3): 434-439.

Jafri, N.S., C.A. Hornung and C.W. Howden, 2008. Meta-analysis sequential therapy appears superior to standart therapy for *Helicobacter pylori* infection in patients naïve to t reatment Ann. Intern. Med., 19: 35-41.

Krylov, N. and N. Myths, 2006 and realias of the modern theory of the ulcer disease. Collection of thesis of the All-Russian research and practical conference Gastroduodenal ulcer. J. Interventional Gastroenterol., No. 1, pp: 40.

Krienitz, W., 1906. Uber das Auftreten von spirocheten vershiedener form im Mageninhalt bei Carcinoma Ventriculi. Dtsch. Med. Wschr. Bd., 22: 872-882.

Kon'kova, N.I., 2012. Relationship of gastric microflora and neopterin content in gastric juice in children with chronic gastroduodenitis. Eksp Klin Gastroenterol., No. 6, pp: 123-126.

Kaspina, A.I., 2003. Impact of the *Helicobacter pylori* infection on the condition of the oral mucosa. Institute for Dentistry, 4 (21): 68-69.

Kim, B.J., 2013. Substitutions in Penicillin-Binding Protein 1 in Amoxicillin-Resistant *Helicobacter pylori* Strains Isolated from Korean Patients. Gut Liver. 7 (6): 655-660.

Lukinykh, L.M., 2004. Diseases of the oral cavity/Nizhny Novgorod: NGMA, pp: 510.

Mayev, I.V. and A.A. Samsonov, 2006. Modern standards of treatment of the acid-dependent diseases associated with *H. pylori* (materials of the consensus Maastricht-3). Consilium Medicum, 8 (1): 3-8.

Ozturk, Y., 2013. Preventive approaches for intrafamilial *H. pylori* transmission as an efficient target strategy to decrease the prevalence of the infection in developing countries. Turk. J. Gastroenterol., 24 (3): 297-298.

Rosenow, E.S. and A.N. Sanford, 1915. The bacteriology of ulcer of the stomach and duodenum in men. J. Inf. Dis. Vol. 17, pp: 210.

Roos, S., 2005. *Lactobacillus gastricus*, *Lactobacillus antri*, *Lactobacillus kalixensis* and *Lactobacillus ultunensis* isolated from human stomach mucosa. Engstrand, A. and I. Jonsson (Eds.). Int. J. Syst. Avil. Microbiol., No. 55, pp: 77-82.

Rafalsky, V.V., 2007. Adverse drug reactions and interactions. Guidebook for a polyclinic doctor. No. 4, pp: 79-86.

Schwarz, K. 1910. Ueber penetrierende Magen-und Jejunalgeschwure. Beitr. Klin. Chir., 67: 96-128.

Sasaki, K., 2012. Candida-associated gastric ulcer relapsing in a different position with a different appearance. World J. Gastroenterol., 18 (32): 4450-4453.

Salmanian, A.H., F. Siavoshi, F. Akbari and A. Afshari, 2008. Yeast of the oral cavity is the reservoir of *Helinobacter pylori*. J. Oral. Pathol. Med., 37 (6): 324-328.

Uberti, A.F., D. Olivera-Severo, G.E. Wassermann *et al*., 2013. Pro-inflammatory properties and neutrophil activation by *Helicobacter pylori* urease. J. Toxicon., No. 69, pp: 240-249.

Wadstrom, T., S. Hirmo and T. Boren, 1996. Biochemical aspects of *Helicobacter pylori* colonization of the human gastric mucosa. Aliment. Pharmacol. Ther., 10 (S.1): 17-27.

Zimmermann, J.S., 2011. Issues of etiology and pathogenesis of ulcer disease: re-reading V.H. Vasilenko (Ed.). Clinical Medicine, No. 1, pp: 14-19.

Zimmermann, J.S., 2013. Unsolved and disputable issues of the modern gastroenterology. MED Press-Inform, pp: 224.

Zou, J., 2009. Meta-analysis: Lactobacillus containing quadruple therapy versus standard triple first-line therapy for *Helicobacter pylori* eradication. Helicobacter, 14 (5): pp: 97-107.

Zullo, A., F. Pema, C. Hassan *et al*., 2007. Primary antibiotic resistance in *Helicobacter pylori* strains isolated in northern and central Italy. Pharmacol. Ther., 25 (6): 1429-1434.

Zheng, X., 2013. Lactobacillus-containing probiotic supplementation increases Helicobacter pylorieradication rate: Evidence from a meta-analysis. Rev. Esp. Enferm Dig., 105 (8): 445-453.