Guided meditations on the stages of

Helicobacter pylori is a gram-negative helium bacterium involved in the development of gastritis, ulcers and associated with stomach cancer. It was discovered in 1983 by Australian pathologists Robin Warren and Barry Marshall by studying the mucous membranes of a person's stomach. Even Marshall experimented with himself, producing material contaminated with bacteria, where he discovered that he was producing gastritis, and was able to check for bacteria in his own stomach biopsy. He also checked to see if he was responding to antibiotic treatment. Helicobacter pylori thus dismantled the old theory that gastritis was produced by consuming spicy food or stress. For this reason, Warren and Marshall were awarded the Nobel Prize in Medicine in 2005. Common characteristics For its great resemblance to the genus Campylobacter, in the early days it was called Campylobacter pyloridis, and then Campylobacter pylori, but was later reclassified to a new genus. It is believed that once the microorganism is first acquired, it can remain for years or for life, in some cases imptomatic. On the other hand, the stomach does not seem to be the only place where the microorganism can be placed, it is believed that H. pylori can consolidate in the mouth before colonizing the stomach. It is also possible that H. pylori present in the mouth may re-infect the stomach after treatment. This is amplified by discovering that some amptomatic children have had their plaques of microorganisms isolated. However, although helicobacter pylori infection is imptomatic in some people, it is not harmless since it has been linked to 95% duodenal ulcer, 70% ulcer disease and 100% chronic gastritis location. In addition, Helicobacter pylori has been classified as a Class I carcinogen by the International Agency for Cancer Research for its link between infection and stomach cancer. Habitat Helicobacter pylori was found in the following guests: male, monkey and cat. This bacterium requires a microaerophilic atmosphere (10% CO2, 5% O2 and 85% N2) to be grown, with iron being an important element for its growth and metabolism. The optimal growth temperature is 35 to 37 oC, although some strains are capable of developing at 42 oC. Similarly, a certain degree of moisture contributes to its growth. Helicobacter pylori slowly grows in the lab, may require 3 to 5 and even up to 7 days for the colony to be evident in the middle. Non-selectively blood-supplemented media can be used for cultivation. On the other hand, Helicobacter pylori is characterized by its mobile and because of its spiillary shape allows it to have propeller movements as if it were screwed. This will help you mobilize through gastric mucus. It is also catalysis and positive oxidase and a major manufacturer of urease, the latter performs a vital function for microorganisms. Urease allows you to survive in an acidic pH environment by generating ammonia, which helps you to alcalize your pH. The microorganism needs pH from 6 to 7 to reproduce. To do this, in addition to the use of urease, it is established to live below the stomach mucosa, where gastric mucus protects it from the extreme acidity of gastric light (pH 1.0 - 2.0). On the other hand, proteases released by bacteria modify stomach mucus, reducing the likelihood of acid spreading through mucus. The Virulence Factors of Flagella Bacteria Movement is a factor of virulence because it helps to colonize the stomach mucosa. The adhesive bacterium has pilis and fimbrial hemagliutin, which act when microorganisms stick to gastric and duodenal cells. Adhesion is a strategy of bacteria to resist the peristilization of the mucous layer where they reside and then migrate to epithelial cells. On the other hand, specific hemagglutinins of sialic acid on the surface of the mucosa delay adhesion and the intake of H. pylori. Lipopolisaccharides (LPS) are endotoxic, as are LPS other gram-negative bacteria. The purified antigen can cause apoptosis. Urease Bacteria uses the production of urease to degrade urea in ammonia and carbon dioxide. This action allows you to maintain the alkaline pH around you and thus avoid the destruction of the stomach with the salric acid, ensuring its survival. This property is encoded by the Ura A. Citotoxine vacuolizing gene (VacA) This is a protein that causes vacuoles in the epithelial cells of the stomach, hence tissue ulcers. It is encoded by the VacA genome. Cytotoxin (CagA) Strains with the CagA gene are more virulent. They are associated with severe gastritis, atrophic gastritis, duodenitis and/or stomach cancer. This cytotoxin cagA increases the proliferation of gastric cells without apoptosis, which leads to a change in the normal model of renewal of the stomach epithelium. Superoxide dismutase and catalysis is essential to protect against O2-dependent death from neutrophils. It works by degrading hydrogen peroxide, a metabolite that is toxic to bacteria. Nitrogen oxide induction syntasis The bacterium induces iNOS and macrophases in vitro. This finding suggests that high production of nitric oxide by induction of this litase, due to immune activation, is involved in tissue damage. Phospholipalase, lipase and mcinase allow the infestation of microorganisms under the stomach mucosa and then alter the mucus so that it functions as a waterproof layer that protects it from stomach-churning mild acid. Also in this place the immune response is completely ineffective. Domain taxonomics: Phylum Bacteria: Proteobacteria Class: Epsilonproteobacteria Order: Campylobacterales Family: Helicobacteraceae Genus: Helicobacter Species: pylori Morphology Helicobacter pylori is a thin helium bacillus gram negative, small, curved and slightly plump. It is about 3 mm long and 0.5 mm wide. It is mobile due to the presence of several polar disasters (in the plume), 4 to 6 in total, which is characteristic of the shell. The shell that covers the flagellae contains proteins and lipopolysacchaid is equivalent to the components of the outer membrane. However, its function is unknown. It doesn't form a spore, and it's not a capsule. The cell wall is similar to the wall of other gram-negative bacteria. Colonies of Helicobacter pylori tend to be small gray and translucent. When colonies age (long-term cultures), bacillary forms become cocoids. Diagnosis For diagnosis Helicobacter pylori There are many methods and they are classified as invasive and non-invasive. -Invasive methods of gastric mucosa biopsy It is taken through endoscopy, the most sensitive method for diagnosing Helicobacter pylori. Microorganisms can be observed during tissue incisions, and the mucous membrane will have pathological characteristics of their presence. The downside is that the distribution of H. pylori through the stomach is not homogeneous. Rapid testing of urease is a method of indirect detection of bacteria. Parts of the samples can be immersed in urea broth with pH (phenol red) and less than an hour results can be observed. The medium of urea broth virah is yellow to fuchsia due to a change in pH caused by the production of ammonia from urea, the action of urease. The sensitivity of this test depends on the bacterial load in the stomach. Growing samples of the stomach mucosa Part of the sample taken by the endoscopy may be intended for cultivation. Negative culture is the most sensitive indicator of treatment after therapy. A sample of a biopsy of the stomach or duodenum should be recent, and its transport is not more than 3 hours. They can be stored for up to 5 hours at 4oC and the tissue must be moist (container with 2 ml sterile saline solution). Before sowing the sample, maceration should be done for greater sensitivity. The sample can be sown in Brucella Agar, an infusion of the brain's heart, or soy triptyase supplemented with 5% ram or equestrian blood. Polymerase chain reaction (PCR). Tissue cuts can be exposed to molecular biology techniques to detect DNA from microorganisms. The advantage of PCR is that it can be used in analysis of samples such as saliva allowing the diagnosis of H. pylori in a non-invasive way, although the fact that the bacteria in the saliva does not necessarily indicate a gastric infection. -Non-invasive methods of serology This method has a sensitivity of 63 -97%. It consists of measuring IgA, IgM and IGG antibodies using the ELISA method. This is a good diagnostic option, but has limited usefulness for controlling treatment. This is because the antibodies can remain elevated for 6 months after the microorganism has been removed. The advantage of this method is that it is faster, simpler and cheaper than the method requiring an endoscopy biopsy. It should be noted that the antibodies generated against H. pylori serve to diagnose, but do not prevent colonization. Thus, people who acquire H. pylori tend to develop chronic diseases. Breathing test For this test the patient must pro disclose carbon-labeled urea (13C or 14C). This compound, when exposed to urease produced by bacteria, is converted into marked carbon dioxide (CO2 C14) and ammonium (NH2). Carbon dioxide enters the blood stream, and from there into the lungs, where exhaled breathing. A patient's breath sample is collected in a hot air balloon. A positive test is a confirmation of infection with this bacterium. Modified breath test This is the same as above, but in this case a 99mTc colloid is added that is not absorbed into the digestive system. This colloid allows you to visualize the production of urea in the place of the digestive system, where it is generated by the gamma chamber. The life cycle of Helicobacter pylori in the body behaves in two ways: 98% of the population of H. pylori freely live in stomach mucus. It serves as a reservoir for adept bacteria that will serve to transfer. While 2% are attached to epithelial cells that support infection. Thus, there are two populations, adherents and non-appearances, with different characteristics of survival. Pathogenesis Once the bacteria enters the body, it can primarily colonize the joint using virulence factors it has. Bacteria can last a long time installed in the stomach mucosa, sometimes for life without causing discomfort. Invades and colonizes the deep layers of gastric and duodenal mucus coating through proteas and phospholias. It is then attached to the surface epithelial cells of the stomach mucosa and duodenum, without invading the wall. It is a strategic place that takes bacteria to protect against the extremely acidic pH of the stomach light. In this regard, on this site the bacterium deploys urea to further poke the environment and remain viable. Most of the time there is a continuous inflammatory reaction in the stomach mucosa, which in turn changes the mechanisms of regulation of the secretion of stomach acid. This is how some ulcer mechanisms are activated such as: inhibiting the function of the parietal cell by inhibiting somatostatin, where insufficient gastrinate production is conducive. Ammonia is produced as well as cytotoxin VacA abuse epithelial cells, thereby causing lesions in the stomach or duodenum of the mucosa. Therefore, there are degenerative changes in the epithelial surface, including the depletion of mucin, cytoplasmic vacuolization and deorganization of the mucous glands. Inflammatory penetration of the aforementioned lesions leads to the fact that the mucous membrane and own foil invade the dense penetration of inflammatory cells. Initially, penetration can only be minimal with mono-nuclear cells. But then inflammation can spread with the presence of neutrophils and lymphocytes, which cause damage to mucous and parietal cells and may even be the formation of microbeads. Cytotoxin CagA, for its part, enters the gastric epithelial cell, where several ensimatic reactions are triggered, which cause the reorganization of the actin cytoskeleton. The specific mechanisms of carcinogenesis are unknown. However, prolonged inflammation and aggression are thought to cause metaplasia and prolonged cancer. Pathology Chronic surface gastritis usually occurs within weeks or months after the bacteria have been identified. This gastritis can progress to ulcers and subsequently cause gastric lymphoma or adenocarcino disease. Аналогичным образом, Helicobacter pylori инфекции является условием, которое предрасполагает к страданиям от лимфомы MALT (лимфома лимфомы лимфомы лимфомы лимфомы, связанные с слизистой оболочки). On the other hand, recent studies mention that Helicobacter pylori causes extragastric diseases. These include: iron deficiency anemia and thrombocytopenia purple Also skin diseases such as rosacea (the most common skin diseases associated with H. pylori), chronic itching, chronic idiopathic hives, psoriasis and others. In pregnant women it can cause hyperemesis gravidaus. Other less common sites where H. pylori is thought to have some involvement causing pathology is at the level: middle ear, nasal polyps, liver (hepatocellular carcinoma), gallbladder, lungs (bronchiectaz and chronic obstructive pulmonary COPD disease). It has also been linked to eye disease (open angle glaucoma), cardiovascular disease, autoimmune disorders, among others. Clinical manifestations This pathology can take amptomatic up to 50% of adults. Otherwise, the primary infection can cause nausea and pain in the upper abdomen, which can last up to two weeks. Symptoms then go away to appear some time after gastritis and/or ulcers are established. In this case, the most common symptoms are nausea, anorexia, vomiting, epigastric pain and even less specific symptoms such as belching. Ulcers can cause severe bleeding, which can be complicated by peritonitis from leaking stomach contents into the abdominal cavity. Infection People with Helicobacter pylori can secrete bacteria through the stool. Thus, water consumption can be contaminated. Therefore, the most important way of infecting a person is fecal-oral. It is believed to be in water or some vegetables that are usually eaten raw, such as lettuce and cabbage. These products can be contaminated by pouring contaminated water. However, the microorganism has never been isolated from water. Another method of infection is a rare oral oral, but it has been documented in Africa by the custom of some mothers to pre-mask the food of their children. Finally, iatrogenic contagion is possible. This pathway involves contamination through contaminated or poorly sterilized material in invasive procedures related to contact with the stomach lining. Helicobacter pylori treatment in vitro is prone to various antibiotics. These include: penicillin, some cephalosporins, macrolides, tetracyclines, nitroimidazole, nitrofurans, quinolones and vismutous salts. But they are inherently resistant to receptor blockers (cimetidine and ranitidine), polymyxin and trimethoprim. Among the most successful treatments you have: a combination of medications, including 2 antibiotics and 1 proton pump inhibitor. The most commonly used combination of antibiotics is claritromycin and metronidazole or claritromycin, amoxicillin or claritromycin or metronidazole and tetracycline. The proton pump inhibitor may be Omeprazole or esomeprazole. Some treatments may also include consumption of bismuth salts. The therapy must be performed for at least 14 days, as recommended by the FDA. However, in some patients this therapy is difficult to tolerate. It is recommended to combine treatment with the use of products containing probiotics. These treatments are effective, but in recent years resistance from Helicobacter pylori to metronidazole and claritromycin has been reported. Microorganisms can be eradicated, but re-infection is possible. In the second re-infection therapy recommended the use of levofloxacin. Links Koneman E, Allen S,

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