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VIRTUAL LAF CONFERENCE

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SUBJECT: Helicobacter pylori & Afib

Could the following fascinating research be discussed in the Conference Room in the future? If the research is on the right track it holds great promise for afibbers.

http://www.reutershealth.com/en/index.html

NEW YORK (Reuters Health) - Patients with atrial fibrillation are nearly 20 times more likely to be infected with the common gastric microbe Helicobacter pylori than are healthy controls, with the association strongest in patients with persistent rather than paroxysmal atrial fibrillation, according to a report in the July issue of *Heart*. Atrial fibrillation patients also had higher levels of C-reactive protein (CRP) than did controls, suggesting that H. pylori's effects on this arrhythmia may be mediated through an inflammatory pathway. While the exact mechanism is unknown, Dr. A. S. Montenero, from Policlinico Multimedica in Milan, Italy, and colleagues hypothesize that it may relate to autoantibodies that develop in some H. pylori-infected patients. These antibodies, which normally attack a proton pump found on gastric cells, may instead attack a similarly appearing pump on cardiac cells, ultimately triggering atrial fibrillation. The new findings are based on a study of H. pylori positivity and CRP levels in 59 patients with atrial fibrillation and 45 healthy controls. The atrial fibrillation group was further divided into 29 patients with persistent disease and 30 with paroxysmal disease. In the overall analysis, 97.2% of atrial fibrillation patients were seropositive for H. pylori compared with just 5.3% of controls. As noted, CRP levels were significantly higher among atrial fibrillation patients as well ($p < 0.001$). Further analysis showed that persistent atrial fibrillation was tied to a higher rate of H. pylori seropositivity ($p = 0.027$) and to higher CRP levels than was paroxysmal disease ($p = 0.041$). The findings show a highly significant link between H. pylori seropositivity and atrial fibrillation, the authors note. However, "more data will be necessary from controlled studies to further identify how H. pylori can influence the pathogenesis of atrial fibrillation."

Dean

The whole paper is here:

http://press.psprings.co.uk/heart/july/960_ht36681.pdf

George

I was intrigued to find that H.Pylori also may cause migraine headaches, which I also suffer from.

H Pylori + Migraine + AF..... Could there be a common thread here?

Rob P.

Rob:

I also have migraines and LAF. H. Pylori connection. In my questioning members of this BB, is seems this combination is fairly common.

Afib episodes ~3 or 4 times per month.
Migraines ~ 2 or 3 times per month.

Pam

By the way, I think the article quoted: "In the overall analysis, 97.2% of atrial fibrillation patients were seropositive for H. pylori compared with just 5.3% of controls" is a poor interpretation of the data in the paper.

What the paper actually says is "In the AF group H pylori seropositivity (97.2 (50.5–100.0) IU/ml) and CRP (8 (6–10) mg/l) were significantly higher (p , 0.001) than in the control group (5.3 (5.0–33.9) IU/ml and 1 (0–2) mg/l, respectively)"

What this means is that the average serum level of H Pylori is 97.2 IU/ml in the afibbers vs 5.3 IU/ml in the controls.

Just goes to show you that journalists don't always accurately convey the information in the publications.

George

Pam,

Yes it doesn't say +ANA but when it says autoantibodies develop in some HP infected patients, I take it as +ANA which is equal to autoantibodies. Based on all my blood test and the symtoms I have it, this fits me perfectly.
I agree with Dean's suggestion. I really believe this maybe the beginning of curing underlying afib.

When I was researching into HP to find out why all of sudden I was diagnosed with HP, maybe I got it from my cats? - we have 3 cats. I never had animals living with me until 10 yrs ago. I talked to veterinarian to test HP on our cats but she said there is no test for it on animals. She said they are working on it but it’s not available yet, that was couple yrs ago. She said a lot of animals carry HP but there is no data on weather it's transmittable to animal to human or vs; I'm pretty sure you can to human to human. My husband took blood test and he doesn't have it. I know co-work and her son has, but not her husband.

I never had migraine headaches but lately I do. I wasn't sure this is related to sinus headaches or just migraine. It’s hard to tell which is which. I have all the data but what do I do with it. According to my GI, I do not have HP but I do have high +ANA. I will be seeing rheumatologist next wk. I really appreciate what questions I should be asking regards to this issue.

Thanks,
Mira

Mira:

While one article I read and quoted here, they name other antibodies that are autoantibodies, I think it is primarily ANA. Possibly in response to H. P.

Oddly, I am a cat person also. I've always had cats and used to breed Himalayans until I got so sick with my ablation. After that I got my 5 Himalayans fixed. One died, but I still have the four left.

Please let us know what you find out.

Pam

Found this interesting article. Looks like cats are carriers and possible transmitters of H. Pylori.

http://www.vetmed.wisc.edu/pbs/zoonoses/Helicobacter/helicoanimals.html

Pam

Pam,

I'm sorry about your cat. They really keep us company. My three cats, each one has different personalities and they are unique in their own way.

When I first saw Rheumatologist, he done some physical test and said you do have rheumatoid arthritis and he sent me home. So I went 2nd Rheumatologist, he said you don't have any symptoms of join problems, etc. But I asked him for blood test just to be sure which I took couple wks ago and next appointment will be just follow up for that. But I will bring over the article and will ask for blood test just for antibodies for HP. I'm not sure this will take care of it or are there another kind of blood test you have to do? I believe the test he ordered (listed below) was for rheumatoid arthritis but not for antibodies for HP? Am I asking right questions?

Here are blood work:
Anti-dsDNA
Anti Sm(ENA)
SSA and SSB ab
C3 and C4

Mira

Mira:

I'm lost here. I think Anti-dsDNA and Anti-Sm (ENA) are autoantibodies.
The rest I will have to look up. C3 and C4 I think are called compliments, but I will have to look that up as well. Let us know when you find out.

Did you read about link about cats and H. Pylori? I should have an ANA titer drawn too.

Pam

I just typed in below the excerpt from the link Wil has provided. This is same article Dean posted above but with more details. I thought this particular article is very interesting.

Based on this article, antibodies are hard to tell from H+/K+ - ATP and Na+/K+ - ATPase so it ended up attach to cardiac cells instead of proton pump of gastric cells which causes afib.

HP is a very strong bacterium, with features that allow it to cross gastric mucus and to attach to epithelial cells, thereby evading the immune response. HP infection is responsible for determining an important system and mucosal humoral response, but these antibodies do not eradicate the infection and may contribute to tissue damage. Different HP strains can determine the specific systemic antibody response in infected patients. Some patients with HP have utoantibodies to H+/K+ - ATPase of gastric parietal cells that can determine corpus atrophy. Considering that there is a similarity

between H+/K+ - ATPase, the proton pump of gastric cells, and Na+/K+ - ATPase, the pump of cardiac cells, which are inhibited by cardiac glycoside such as digoxin, an interesting hypothesis is that these autoantibodies to H+/K+ - ATPase may also be antibodies to Na+/K+ - ATPase, thus determining atrial damage. In fact, cardiac Na+/K+ - ATPase and H+/K+ -ATPase have a similar 35 kDa glyco-protein necessary for their catalytic activity. The role of these pumps is to maintain IONIC homeostasis by hydrolysing ATP and therefore loss of this balance may trigger AF by determining abnormal automaticity or triggered activity that causes delay after DEPOLARISATION inducing very rapid premature atrial contractions. Several issues remain unresolved. We have shown the highly significant link between AF and HP in our relatively small sample and confirmed that CRP is a good marker for the inflammatory process. More data will be necessary from controlled studies to further identify how HP can influence the pathogenesis of AF.

Mira

Pam,

I really don't have any idea except that its related +ANA.
That is why I listed here. Hopefully you and others will help out.

Yes, I have read your article about cats and HP. Thank you!

I bet a lot of people on bb have cats or dogs and should be test for ANA titler as well.

Mira

I'm actively searching for information on the etiology of H. pylori.... I've noted it is also implicated in stomach cancer, big time, and gallstones.... along with this new news on AF. But, what I want to learn is how does H. pylori become pathogenic? Apparently, the pathogen is ingested and then it takes time to flourish. Note the last paragraph in this post from the articles I've included from my cursory research.

Jackie

Here's something from 5 years ago:

1: *Annu Rev Microbiol. 2000;54:615-40. Related Articles, Links*

The disease spectrum of Helicobacter pylori: the immunopathogenesis of gastroduodenal ulcer and gastric cancer.

Ernst PB, Gold BD.

Department of Pediatrics, Sealy Center for Molecular Sciences, University of Texas Medical Branch, Galveston, Texas 77555-0366, USA. pernst@utmb.edu

Helicobacter pylori is a gram-negative bacterium that resides under microaerobic conditions in a neutral microenvironment between the mucus and the superficial epithelium of the stomach. From this site, it stimulates cytokine production by epithelial cells that recruit and activate immune and inflammatory cells in the underlying lamina propria, causing chronic, active gastritis.

Although epidemiological evidence shows that infection generally occurs in children, the inflammatory changes progress throughout life. H. pylori has also been recognized as a pathogen that causes gastroduodenal ulcers and gastric cancer. These more severe manifestations of the infection usually occur later in life and in a minority of infected subjects. To intervene and protect those who might be at greatest risk of the more severe disease outcomes, it is of great interest to determine whether bacterial, host, or environmental factors can be used to predict these events.

To date, several epidemiological studies have attempted to define the factors affecting the transmission of H. pylori and

the expression of gastroduodenal disease caused by this infection. Many other laboratories have focused on identifying bacterial factors that explain the variable expression of clinical disease associated with this infection.

An alternative hypothesis is that microorganisms that cause lifelong infections can ill afford to express virulence factors that directly cause disease, because the risk of losing the host is too great. Rather, we propose that gastroduodenal disease associated with H. pylori infection is predominantly a result of inappropriately regulated gastric immune responses to the infection.

In this model, the interactions between the immune/inflammatory response, gastric physiology, and host repair mechanisms would dictate the disease outcome in response to infection.

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Here's another informational article

Edited by Marvin Turck, MD
Best Practice of Medicine.

Posted February 2, 2001

This is certainly an exciting time in the field of infectious diseases. Many clinical syndromes and afflictions of previously uncertain cause turn out to have a microbial etiology—or, at least, are triggered by microbial antigens acting in concert with genetic and environmental influences. During the past few years, illnesses such as peptic ulcer and atherosclerosis have been linked to infections, and even evolutionary biologists and the mass media are paying attention. Certainly the discovery of retroviruses and the world pandemic of HIV infection have moved infectious diseases to the forefront of public health concerns. I frequently tell my medical students, tongue in cheek, that all diseases of unknown etiology are infectious—we just haven't proven this yet!

The H. pylori rap sheet

It has been 18 years since Helicobacter pylori was implicated in clinical infection. Most experts presently agree that infection by this bacterium account for about 75% of stomach ulcers. In addition, H. pylori is now associated with two cancers: gastric carcinoma and lymphoma of the mucosa-associated lymphoid tissue (MALT lymphoma).

The presence of H. pylori confers about a sixfold increased risk of gastric cancer, and biopsies show that 90% of MALT lymphomas are associated with the bacterium. In the stomach, H. pylori is related to the production of BCA-1, a chemokine that attracts lymphocytes and may lead to lymphomas. Some investigators have suggested that H. pylori may also contribute to heart disease: 62% of individuals with atherosclerosis, but only 40% of those without, were seropositive for infection. They speculated that H. pylori causes low-grade, life-long infection and smoldering inflammation of the arteries. Although Chlamydia pneumoniae is presently the microbe most widely implicated in heart disease, one wonders if previous studies and those in progress purporting the benefits of antibiotics in preventing myocardial infarction have controlled for the presence or absence of Helicobacter. We also know now that not all H. pylori are the same; conceivably the presence or absence of CagA protein or certain lipid moieties are the real culprits.

What else has Helicobacter been up to? In a recent study, investigators found that 47% of people with chronic urticaria, primarily children, were infected with H. pylori; 26% had associated gastritis. Improvement or complete disappearance of urticaria occurred in most treated with antibiotics, whereas only 50% of untreated seropositive patients showed remission.

Another group of investigators found a positive association between H. pylori infection and food allergy in children. They speculated that factors other than CagA may be involved in the pathogenesis of H. pylori infection in children. Finally, children with type 1 diabetes infected with Helicobacter had higher insulin requirements and higher glycosylated hemoglobin A levels than uninfected children Today, the complete genome of H. pylori, containing 1590 genes, has been unmasked and published on the internet, so one may be optimistic that these findings will translate into clinical practice in the foreseeable future.

http://merck.micromedex.com/index.asp?page=bpm_edupdate&specialty=BPM01ID&action=editor

The Merck Manual of Diagnosis and Therapy
Section 19. Pediatrics
Chapter 268. Gastrointestinal Disorders

Etiology and Pathophysiology

As in adults with peptic ulcer, Helicobacter pylori may be present, and if so, its elimination may cure the ulcer, suggesting that it is the cause in these patients. H. pylori infection is less common in children than in adults with ulcer disease, and is more likely to be found in children of parent(s) with peptic ulcer. H. pylori and NSAIDs may disrupt the normal mucosal defense and repair, making the mucosa more susceptible to the attack of acid.

http://www.merck.com/mrkshared/mmanual/section19/chapter268/268b.jsp

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This from emedicine (http://www.emedicine.com/ped/topic2987.htm)

Frequency: The prevalence of peptic ulcer disease in infants, children, and adolescents is approximately 5 per 10,000 population. Helicobacter pylori colonization in the stomach has been recognized as an important risk factor in the development of peptic ulcers in children. In developing countries, approximately half of children have colonization of H pylori by age 10 years, whereas colonization in childhood is rare in developed countries. However, in children with a low socioeconomic status, colonization rates in developed countries are similar to those in developing countries.

Etiology: H pylori, described by Warren and Marshall in 1982, is a spiral gram-negative organism with a smooth surface and multiple unipolar flagella. Since its discovery, this bacterium has gained worldwide recognition as an important factor in the pathogenesis of gastritis and peptic ulcer disease. H pylori has the ability to grow in an acidic environment because of its urease-mediated ammonia production, which neutralizes gastric acid. Most organisms settle in the mucus of the gastric antrum without invading the mucosa. Flagella enable H pylori to resist gastric peristalsis, and the bacterium's adhesion properties allow this organism to attach to the gastric epithelium. Mucosal reaction consists of acute and chronic inflammation with neutrophil and lymphocyte infiltration and, in children, lymphonodular hyperplasia.

Numerous studies link H pylori colonization with chronic gastritis in children. Furthermore, H pylori gastritis seems to correlate strongly with duodenal ulcers in this group, in which a 90-100% prevalence of H pylori gastritis has been reported. However, a link between H pylori antral colonization and gastritis is less likely as the etiologic factor of pediatric gastric ulcers. Whether the organism causes the ulcers or plays an important role in maintaining them is unclear; however, eradicating H pylori in affected adults and children heals and cures their recurrent peptic ulcers.

Pathophysiology: Mediators of mucosal inflammation, such as lymphokines and oxygen-free radicals, are involved in the development of gastritis or ulcer disease. Mucosal defenses include intestinal and pancreatobiliary sources of bicarbonate, surface-active phospholipids, mucosal blood flow, and rapid cell exchange rates influenced by substances such as epidermal growth factor. Prostaglandins protect the gastric mucosa by directly inhibiting parietal cell function and acid secretion, increasing mucus production, and promoting mucosal blood flow. Although the presence of hydrochloric acid is necessary for the development of ulcers, gastric acidity tends to be normal or decreased in gastric ulcers (particularly secondary ulcers) and increased in pyloric and duodenal ulcers. Therefore, factors associated with mucosal resistance are more prominent in the etiology of gastric ulcers, and factors affecting acidity predominate in the etiology of pyloric and duodenal ulcers.

The effects of H pylori colonization of the gastric mucosa may take years to become evident. This organism possesses large amounts of urease, allowing it to convert urea to ammonia and carbon dioxide and, thus, gain a survival advantage in an acidic environment. Production of oxygen dismutase and catalase enables H pylori to resist damage from phagocyte-released oxygen free radicals. The microaerophilic metabolism of this bacterium permits its

proliferation in the semipermeable layer of mucus that lines the stomach.

After ingestion of the organism, a period of florid proliferation occurs, with resulting gastric inflammation. Usually, the mounted immune response, which may take weeks to months to develop, is ineffectual in eliminating the infection, and chronic superficial gastritis ensues.

Hello Pam,

Below is link to my blood work but I haven't found the link for C3 and C4 yet.

Anti-dsDNA
Anti Sm(ENA)
SSA and SSB ab
C3 and C4

http://www.labtestsonline.org/understanding/analytes/autoantibodies/glance-2.html

http://www.labtestsonline.org/understanding/analytes/autoantibodies/sys_ab_table.pdf

Mira

What documentation exists for the connection between HP and migraine?

I've never been able to successfully articulate a description of my migraine symptoms. The conventional description is "pain"; but that is an inadequate description in my case, though pain is present when the migraine peaks. My wild guess has always been that migraine is actually a symptom of brain inflammation. That guess might well be consistent with a connection between migraine and HP.

Wil

Migraines and H. Pylori
This article submitted by on 11/3/99.

New Scientist, 12 September 1998
Mario Giacovazzo of La Sapienza University in Rome and his colleagues found that of 225 migraine sufferers, 48 per cent were infected with Helicobacter pylori, a bacterium implicated in stomach ulcers and certain stomach cancers. A week's course of antibiotics eradicated the bacteria in 84 per cent of the infected migraine sufferers. Almost a quarter experienced no further headaches, and most of the rest had fewer and less severe headaches over the next year, the researchers told the Migraine Trust International Symposium in London last week.

http://neuro-www.mgh.harvard.edu/forum_2/DepressionF/11.3.995.25PMMigrainesand.html

H. pylori Infection. People who are infected with the bacteria H. pylori , the major cause of peptic ulcers, are at higher risk for migraines.

http://www.umm.edu/patiented/articles/who_gets_migraine_headaches_000097_4.htm

H. Pylori is further implicated with heart disease, gum disease, rosacea, asthma, and chronic headaches or migraines.

http://www.acu-cell.com/dis-hpy.html

Headache might be linked to infection with a common stomach "bug," a preliminary report suggested. A study presented April 26 at an infectious diseases conference in Milan found that about 18% of chronic migraine sufferers were infected with the gastrointestinal bacteria Helicobacter pylori, which has been linked to an increased incidence of ulcers. In the study, Italian scientists divided 130 migraine patients who were infected with H. pylori into two groups. One group was given a 3-week course of antibiotics while the other received 3 weeks of antibiotics plus Lactobacillus, a "friendly" bacteria or probiotic found in yogurt. The probiotic group took 3 Lactobacillus doses a day for 3 months, then 1 dose a day for the next 9 months. At the end of the year, 50% of the people treated with antibiotics alone were still getting migraines, compared with 20% in the group who took the probiotics for a year. Headaches in the probiotic group occurred less often, were milder and went away more quickly than they did in the antibiotics group. The infection findings were similar. After a year, the bacteria were reduced 40% in the group who took antibiotics and 70% in the group getting the combination treatment. This new "germ theory" of headache is interesting but the study is inconclusive because it did not include a placebo group. Placebo-treated groups frequently show improvement rates up to 30%. Also, the study has not yet been published, so experts in the field cannot yet review the data and the study methods. Infection with H. pylori is very common, but the incidence rate varies greatly with age, ethnicity and region.

The 18% infection rate among these migraine sufferers may not be any greater than the general population, which makes a causative link with migraine less likely.

http://www.achenet.org/news/older/052002.php

Aspirin and Helicobacter Pylori

According to an article published in *The American Journal of Gastroenterology (Am J Gastroenterol 2001;96:1751-1757*), while aspirin and Helicobacter pylori infection have each been linked to gastric mucosal injury, it seems that these factors also interact to increase the risk of damage.

For the study, 29 healthy subjects with H. pylori infection and 32 similar subjects without infection were randomized to receive placebo or low-dose aspirin therapy for 45 days. Results of endoscopic examinations showed the presence of aspirin-related erosive disease in 50% of the infected subjects, but only in 16% of the uninfected subjects.

Furthermore, aspirin caused significantly greater gastric antrum damage in the infected subjects than in the uninfected subjects. Two of the aspirin-treated infected subjects developed antral ulcers. The differences in aspirin-induced damage could not be accounted for by differences in prostaglandin levels since infected and uninfected subjects showed similar reductions in prostaglandin synthesis.

According to the authors, these findings indicate that the inflammation that is present with H. pylori infection predisposes to more damage from aspirin. They add that the implication from the current findings is that if one could identify these high-risk patients and eradicate the infection prior to aspirin therapy, one may reduce the risk of significant GI bleeding and other ulcer complications.

http://www.targethealth.com/ontarget/2001/08052001.htm

H. PYLORI and low Stomach Acid: Nutritional Causes, Prevention and Therapies

While "stress" was a popular basis for stomach ulcers years ago, Helicobacter Pylori has become the ever popular cause for peptic and duodenal ulcers since its discovery by two Australian doctors, Robin Warren, M.D., and Barry Marshall, M.D., in the early 80's. Some doctors place the bacterium's involvement as high as 90%, however ulcer-inducing drugs, alcohol, and other lifestyle stimulants are still a more common factor in the development of these ulcers than given credit lately.

H. Pylori may be inhibited by raising stomach acid or lowering its pH, provided this is done before much damage is done by the bug. This is the reason why people who manage to maintain normal stomach acid levels are generally

asymptomatic and don't get ulcers unless they are taking specific drugs, they consume excessive amounts of alcohol (although alcohol itself inhibits Helicobacter Pylori), or they supplement too much calcium and magnesium, which in turn can lower stomach acid levels too much. Coffee consumption is another factor known to aggravate the symptoms of H. Pylori infections.

Unfortunately, people with reduced acid levels frequently suffer from what they assume is elevated stomach acid (heartburn, bloating, nausea, frequent burping), and as a result often take acid-lowering drugs or remedies.

By doing so, they encourage greater H. Pylori activity and thus increase the risk to develop peptic or duodenal ulcers, pancreatic / gastric cancer, and mucosa-associated lymphoid tissue (MALT) lymphoma.

H. Pylori infections can also lead to some forms of arthritis (calcification, spurs), iron-deficiency anemia, and Vitamin B12 deficiency that may develop as a result of lowered stomach acid levels and damage to parietal cells which produce the intrinsic factor. H. Pylori is further implicated with heart disease, gum disease, rosacea, asthma, and chronic headaches or migraines.

If patients had indeed high acid levels (as some physicians still have them believe), then why do symptoms improve when acid levels are raised?

The confusion usually stems from the fact that esophageal reflux (GERD) causes heartburn from acid getting up into the esophagus, which doesn't have the acid-protective mucus coating of the stomach, and where - if acid was not reduced - could eventually cause ulcerations or esophageal cancer. In such an event, stomach acid will indeed have to be controlled by acid-lowering medications, as well as other lifestyle changes such as not overeating at any meal, not bending down or lying down following meals, and to sleep with the upper body more elevated until the reflux situation is resolved. Bromelain supplementation with meals is often beneficial in such situations because of its anti-inflammatory and pro-digestive properties.

Because of its acid-inhibiting effect, H. Pylori actually reduces the risk of developing esophageal adenocarcinoma, which is a form of cancer that may result from chronic esophageal reflux, and also Barrett's esophagus (Barrett's syndrome). However, as mentioned above, H. Pylori increases the risk for developing various other cancers, including esophageal squamous-cell carcinoma.

If a Stomach Ulcer has developed as a result of H. Pylori infection, then along with the usual antibiotic treatments, acid-lowering therapy is also necessary to allow the ulcer to heal. Once the course of drugs is finished and the ulcer has healed, then normal acid levels should be maintained from there on.

After the discovery of H. Pylori, and once medical science accepted it as being a significant factor with ulcers, predictions were made that ulcers and related stomach complaints would become a thing of the past.

However, there are as many patients as ever complaining of stomach problems, including those who had been "cured" of H. Pylori. The reason is very simply low stomach acid - which had not been corrected as part of the treatment. Antibiotic-resistant Helicobacter Pylori cases have now become a commonplace occurrence as well, and there are also plenty of patients who simply don't tolerate any of a number of antibiotics used in the treatment of H. Pylori.

Physicians don't generally test for Helicobacter Pylori unless a patient exhibits gastric complaints, and even then, patients themselves have to frequently convince - or even beg their GPs to run a test after learning about H. Pylori through a news flash or reading about the long-term risks associated with its infection. Unfortunately, if a patient suffers from non-gastric symptoms, there is little chance of the average practitioner relating a condition to a possible Helicobacter Pylori infection.

For example: One of my patients developed asthma after having no prior history of this condition. An Acu-Cell Analysis revealed greatly reduced stomach acid levels and very high calcium and magnesium levels - also with no prior history. Although supplementing digestive aids to raise acid levels improved that patient's asthma, the fact that her bismuth and lithium levels were lower as well led me to suspect H. Pylori involvement, with turned out positive. Following successful antibiotic therapy, her magnesium, calcium, bismuth and lithium levels returned to normal, her stomach acid returned to near normal without the need for any more digestive supplements, and she became and remained completely

asthma-free.

Without eradicating the H. Pylori bug, she would have likely had to remain on asthma medication for much of her life, and/or there would have been the additional risk to develop other - potentially serious -
H. Pylori-related medical conditions, including cancer.

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Following are some "Natural Remedies" that have been used with mixed results for H. Pylori:

A 5% solution of "Manuka Honey" from New Zealand worked well in vitro to kill the bug, with almost two dozen studies backing up the claims, however I have yet to see a single patient ending up with an actual "cure" after taking Manuka Honey. There are also claims that pure Alcohol taken on an empty stomach early in the morning will kill H. Pylori. Only one of my patients tried that approach, and although the symptoms did indeed disappear for a while, they eventually returned as severe as before.

Regular consumption of sulfur-containing sources such as Garlic, Onions, or MSM is supposed to be helpful for H. Pylori symptoms according to some sources. Similar claims are made for regular intake of Licorice and Cinnamon, larger amounts of Vitamin C, as well as Coconut oil, or spicy foods such as Hot (Chili) Peppers. All had shown to somewhat inhibit Helicobacter Pylori in various trials, but again, none of these have really proven to be effective in actual clinical settings on a long-term basis.

Probiotic-types of remedies (friendly bacteria) consisting of Lactobacillus Acidophilus and Bifidus - although not a cure in themselves - are an important addition to any therapy for H. Pylori infection to help inhibit the bug, and to counteract any headaches, early-morning nausea, or general dyspepsia associated with low stomach acid alone, or following antibiotic therapy, with the acidophilus being best taken at bedtime. Some patients only tolerate the lactobacillus acidophilus, without the bifidus.

An optional adjunct remedy in the treatment of H. Pylori is Bismuth, which is also part of OTC products such as Pepto-Bismol. Cellular bismuth and lithium levels routinely test below normal with low acid levels, respectively to upper stomach involvement (bismuth), and lower stomach / duodenal involvement (lithium). The only problem might be that the high magnesium present in some products (that contain bismuth) generally worsens already low acid levels.

"Mastic" is another remedy that has made the news. It is derived from a tree resin (Pistacia lentiscus) that has been used as a food ingredient in the Mediterranean region for thousands of years, and which is now dried and sold in capsules. Using 1-2g a day, there are reports of H. Pylori symptoms clearing in 90% of patients, and stool samples being H. Pylori-free in 80% of patients after only two weeks. In vitro studies have shown Mastic Gum to be effective against at least seven strains of Helicobacter Pylori, and an increasing number of human trials show similar results, backed by urea breath tests coming back negative.

A number of scientific studies around the world are currently being conducted, including trials to find out whether killing H. Pylori in the oral cavity by chewing mastic gum - not just in the stomach alone – would more permanently eradicate the bug. Unfortunately, as is the case with many therapies and remedies, mastic (mastica) is not tolerated too well by a small percentage of patients who report stomach upsets similar or even worse compared to the discomfort experienced from the H. Pylori bacteria itself. Most patients however experience no problems during the two week mastic treatment, however despite the promising results of some of the above studies, there have been other clinical trials conducted with mastic/mastica that showed no efficacy whatsoever.

Sulforaphane is a promising compound that inhibits extracellular, intracellular, and antibiotic-resistant strains of Helicobacter Pylori. This effect was identified by scientists at the Johns Hopkins University School of Medicine in Baltimore while investigating sulforaphane - one of a class of chemicals called isothiocyanates - for its protective effect against cancer. Sulforaphane is found in broccoli and other cruciferous vegetables such as cauliflower, cabbage, and kale, with broccoli sprouts containing anywhere from 30 to 50 times the concentration of the chemical as contained in the mature plants. Daily recommended amounts of sulforaphane from broccoli sprout extracts are in the 0.2-0.4mg range.

Most patients who don't produce enough stomach acid will continue to experience problems, even if antibiotic therapy - or any other "natural" approach - has successfully killed the bug, but not everyone necessarily always suffers from "heartburn"- like symptoms, or bloating.

Low Stomach Acid can be a factor with allergies, asthma, headaches, chronic fatigue, non-specific aches and pains, osteoarthritis, osteoporosis and other calcium metabolism-impaired problems -- all the way to various cancers. Many of these complaints are rectifiable by normalizing stomach acid, and from personal clinical observation, I'm convinced that even several non-gastric types of cancers could be prevented, since they never seem to develop in the presence of normal acid levels.

To help the symptoms, or until any of several possible causes for low stomach acid are resolved, a digestive aid containing Glutamic acid + Betaine + Pepsin should be taken with every larger meal. Some formulations contain Bromelain as well, which provides additional digestive support. The only contraindications are gastritis, the presence of an ulcer, or when stomach acid levels are not low, in which case acid-raising digestive aids should not be supplemented, although Bromelain alone may still be a consideration for its anti-inflammatory effect.

When antibiotics and natural approaches have not been successful in eradicating Helicobacter Pylori, or when there is intolerance to most of the remedies that are usually helpful with low-acid symptoms, than regularly drinking Pineapple Juice with meals, or sipping it slowly throughout the day may be another option that has helped many patients keep their symptoms to a minimum and improve general digestion.

(see also Acu-Cell Nutrition "Calcium & Magnesium" for a description of a number of causes for low stomach acid).

http://www.acu-cell.com/dis-hpy.html

Hello Jackie,

Thank you so much for your research into H. Pylori! All the articles you have listed here are very interesting, especially Nutritional Causes, Prevention and Therapies. I need some time to read them over and over again to digest it.

I have tried antibiotic 4X in the past 2 yrs; 2X was from my GI and 2X was from my GP. I was already taken antibiotic from my GI but yr later my GP ordered me blood test for HP which showed positive. Later I found out that when you had taken antibiotic, you will have positive blood test. I think it has to do with antibodies in your system? I don't know its forever or it goes away yrs later? But if you never taken antibiotic for HP, then blood test could confirm it.

I have been taking digest enzyme and probiotic on and off for more than couple yrs now. I also took many natural remedies listed here but few not taken yet. I would like to try the Mastic next time.

Thanks Jackie!

Mira

Hello All,

Luckily, I have an appt. to have my esophagus problem (Schatzky's Ring) taken care of again, so I'm going to have the testings done for H. Pylori and I'll report back any significant findings.

Thoughts to ponder. In Jackie's above post it speaks of Sulforaphane inhibiting H. Pylori. Is this another reason to believe that we simply need more sulfur in our diets due to our hazardous environment? Also, is there a possiblity that H. Pylori has always been in our systems, but a particular antibiotic that we have all taken, has caused this to become a "super bug" or mutate?

This topic is certainly an interesting one.

Richard

Just a few thoughts and questions...

1. I'm gaining the impression that HP is difficult to test for ACCURATELY: just what is THE definitive test for HP? Indeed, is there one, or can ALL available tests give false results one way or another?

2. If one is predisposed to HP, and then one gets rid of HP with combined pharmaceutical therapy intervention, then I'm guessing that the return of HP is almost inevitable?

3. Whilst I note that decreased stomach acid makes conditions may well create more favourable for HP, there are some here (Dean, Peter & myself) for whom PPIs have virtually eradicated AF for getting on for 2 years and more. Surely if PPIs create more favourable HP conditions, then PPIs would assist in precipitating more HP-->inflammation-linked AF, rather than LESS?

4. Given that probiotics are cited above as being useful in controlling HP, any thoughts as to how useful consumption of natto food might be as regards similarly controlling HP?

This is a really fascinating subject. There has ALWAYS been - despite the usual and wider indifference of many within the medical community - a strong correlation for soooo many AFrs between gastric distress and ectopy and eventually AF. Its good to see some preliminary endeavours in research circles as to try and unravel the relationship between AF and HP. Let's hope that this is merely the start of something REALLY exciting and useful.

Mike F.

Jackie,

I know that my Mg & K supplements helped neutralize my stomach acid, which did not aid my digestion. I now try to take them apart from my meals had have added Betaine HCl when I take supplements & eat to increase my stomach acid. I've not completely got this figured out yet, but my digestion is much improved.

George

From - http://www.cdc.gov/ulcer/md.htm

How is H. pylori infection diagnosed?

Several methods may be used to diagnose H. pylori infection. Serological tests that measure specific H. pylori IgG antibodies can determine if a person has been infected. The sensitivity and specificity of these assays range from 80% to 95% depending upon the assay used. Another diagnostic method is the breath test. In this test, the patient is given either 13C- or 14C-labeled urea to drink. H. pylori metabolizes the urea rapidly, and the labeled carbon is absorbed. This labeled carbon can then be measured as CO2 in the patient's expired breath to determine whether H. pylori is present. The sensitivity and specificity of the breath test ranges from 94% to 98%. Upper esophagogastroduodenal endoscopy is considered the reference method of diagnosis. During endoscopy, biopsy specimens of the stomach and duodenum are obtained and the diagnosis of H. pylori can be made by several methods: The biopsy urease test - a colorimetric test based on the ability of H. pylori to produce urease; it provides rapid testing at the time of biopsy. Histologic identification of organisms - considered the gold standard of diagnostic tests. Culture of biopsy specimens for H. pylori, which requires an experienced laboratory and is necessary when antimicrobial susceptibility testing is desired.

What are the treatment regimens used for H. pylori eradication?

Therapy for H. pylori infection consists of 10 days to 2 weeks of one or two effective antibiotics, such as amoxicillin, tetracycline (not to be used for children <12 yrs.), metronidazole, or clarithromycin, plus either ranitidine bismuth citrate, bismuth subsalicylate, or a proton pump inhibitor. Acid suppression by the H2 blocker or proton pump inhibitor in conjunction with the antibiotics helps alleviate ulcer-related symptoms (i.e., abdominal pain, nausea), helps heal gastric mucosal inflammation, and may enhance efficacy of the antibiotics against H. pylori at the gastric mucosal surface. Currently, eight H. pylori treatment regimens are approved by the Food and Drug Administration (FDA) (Table 1); however, several other combinations have been used successfully. Antibiotic resistance and patient noncompliance are the two major reasons for treatment failure. Eradication rates of the eight FDA-approved regimens range from 61% to 94% depending on the regimen used. Overall, triple therapy regimens have shown better eradication rates than dual therapy. Longer length of treatment (14 days versus 10 days) results in better eradication rates.

FDA-Approved Treatment Options

Omeprazole 40 mg QD + clarithromycin 500 mg TID x 2 wks, then omeprazole 20 mg QD x 2 wks

-OR-

Ranitidine bismuth citrate (RBC) 400 mg BID + clarithromycin 500 mg TID x 2 wks, then RBC 400 mg BID x 2 wks

-OR-

Bismuth subsalicylate (Pepto Bismol) 525 mg QID + metronidazole 250 mg QID + tetracycline 500 mg QID* x 2 wks + H2 receptor antagonist therapy as directed x 4 wks

-OR-

Lansoprazole 30 mg BID + amoxicillin 1 g BID + clarithromycin 500 mg TID x 10 days

-OR-

Lansoprazole 30 mg TID + amoxicillin 1 g TID x 2 wks**

-OR-

Rantidine bismuth citrate 400 mg BID + clarithromycin 500 mg BID x 2 wks, then RBC 400 mg BID x 2 wks

-OR-

Omeprazole 20 mg BID + clarithromycin 500 mg BID + amoxicillin 1 g BID x 10 days

-OR-

Lansoprazole 30 mg BID + clarithromycin 500 mg BID + amoxicillin 1 g BID x 10 days

*Although not FDA approved, amoxicillin has been substituted for tetracycline for patients for whom tetracycline is not recommended.

**This dual therapy regimen has restrictive labeling. It is indicated for patients who are either allergic or intolerant to clarithromycin or for infections with known or suspected resistance to clarithromycin.

Are there any long-term consequences of H. pylori infection?

Recent studies have shown an association between long-term infection with H. pylori and the development of gastric cancer. Gastric cancer is the second most common cancer worldwide; it is most common in countries such as Colombia and China, where H. pylori infects over half the population in early childhood. In the United States, where H. pylori is less common in young people, gastric cancer rates have decreased since the 1930s.

How do people get infected with H. pylori?

It is not known how H. pylori is transmitted or why some patients become symptomatic while others do not. The bacteria are most likely spread from person to person through fecal-oral or oral-oral routes. Possible environmental reservoirs include contaminated water sources. Iatrogenic spread through contaminated endoscopes has been documented but can be prevented by proper cleaning of equipment.

Hi Mike,

Just a quick post. I have to absorb all of this info. I tested NEGATIVE to H. Pylori in 2002 when I had a GI scope. I wonder if I am still negative. I too have had cats for years and many fish aquariums and ponds.

Had to visit the GI surgeon yesterday for opinion on operating on my loose LES. Showed the Research paper on H. Pylori and afib to him and he said "I have never come across this view before, interesting" and that was that! Told me I was H.Pylori negative in 2002. He is the leading Upper GI surgeon in Australia and helped set up a special surgery section for Upper GI in UK. The results of the consultation was that he will not operate on me as I am a "boarder line" case and it is not worth the risks so he told me I would be on PPI's for the rest of my life unless my GERD deteriorates and he will then consider an op. Asked him about PPI's and cancer and he said there is a slight risk of stomach cancer but the link has not been proven. Hmmm.....
So now I have had no luck with my attempted ablation and now no surgery to fix LES. I have spent a fortune to find all this out! I am doing a better job using this website to cure myself! Thank God for Hans.

Dean

Dean,

You are quite right, this website and also that of the blood type diet are the best I've come across in the battle to control one's a-fib!

Thank goodness for folks like Hans, Dr D and all their helpers!

Joyce

Sorry to have been silent on such an interesting topic. I've been on a bit of a holiday Down Under for the past fortnight. Fortunately the sharks on the Reef didn't find out that I'm on coumadin.

The association of Helicobacter with AF is most titillating. The proton pump autoantibody theory is fascinating, but IMHO it's the inflammation connection again at work. Vagal stimulation secondary to upper GI irritation is probably providing assistance in initiating AF. Although Hp infection causes a drop in gastric HCl (proton pump antibody effect), it does cause ulcers. As such it is the opposite of GERD (increased gastric HCl). Kinda like hypo and hyperthyroidism both being associated with AF.

Presumably by damaging the proton pump (H+/K+ pump) this antibody is decreasing gastric HCl thereby increasing intracellular H+ and decreasing intracellular K+. If this antibody is also attacking the cardiac H+/K+ pump, then one can see why Hp infection might predispose to AF. If this is happening, then one might expect to see an increase in AF in

those on PPIs (proton pump inhibitors). I don't think Dean has experienced such.

LAF is a most complicated disease and Hp induced autoantibodies which may or may not be at work in the heart are at most a side issue. To further underscore the importance of inflammation in initiating and maintaining AF please read "Inflammation Of Atrium After Cardiac Surgery Is Associated With Inhomogeneity Of Atrial Conduction And Atrial Fibrillation" (5/31/05) at
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15927979&dopt=Abstract
There are many other articles underscoring the association of inflammation with LAF.

P cells are still needed to trigger the PACs. Inflammation makes the cardiac substrate more fertile for the development of AF. In fact I believe that P cells are not damaged in LAF, but over time the cardiac substrate does become damaged and that is what determines the age at onset of AF. That is what ultimately causes the "AF begets AF". And the RAS (renin angiotensin system) is at the "heart" of this. Perhaps we can revisit that and relevant recent findings in another CR session.

PC

PC,

Presumably by damaging the proton pump (H+/K+ pump) this antibody is decreasing gastric HCl thereby increasing intracellular H+ and decreasing intracellular K+. If this antibody is also attacking the cardiac H+/K+ pump, then one can see why Hp infection might predispose to AF.

If this theory apply to Dean, his AF should get worse because PPI will increase intracellular H+ and decrease intracellular K+ right? So since he stopped his afib by taking the PPI, it actually helped him. So that means antibody is not attacking his cardiac? Me on the other hand antibody is attacking my cardiac because when I started taking the antibiotic and PPI at the same time which I was diagnosed with Afib. So in my case this theory applied because PPI increased intracellular H+ and decreased intracellular K+. Right?

I also tested for CRP on 10/03 and was 0 and CRP test on 3/31/05 was .3 reference range is .9. I know has it gone up but based on test, I do have elevated CRP.

Correction: I meant to said I do not have elevated CRP.

Mira

Mira,

You and PC state that the proton pump (H+/K+ pump) and the cardiac pump (H+/K+ pump) are the same but the research paper says the cardiac pump is "similar" and is written (Na+/K+ pump) - Sodium! I think that puts a different slant on things.

I tested Negative for H. Pylori in 2002 when I was still having AF attacks. Haven't been tested since so I don't know if I've been infected in the meantime. As my AF has disappeared for the last 2yrs and 4 mths I would have to assume I am still H.Pylori free.

I'm not the only one having success with PPI's. There are 3 others several yrs afib free and one who would be heading for his 5th yr afib free.

If I'm not infected with H.Pylori then maybe the others having success on PPI's are not infected as well.

If this is the case then it sort of poo poo's the Italians research. The only way to answer this properly would be for all of us to be tested for H. Pylori.

By the way, what is the correct and most accurate test for H. Pylori? Is it the breath test or the blood serum test?

Interesting side note - My mother is being breath tested for H.Pylori on Friday. She is recovering from bowel cancer and doctor thinks she may now be infected.

Dean

Dean,

I don't mean to said they are same but you're correct that they are similar. However, because of it, it might mistakenly attached to cardiac cell: Antibodies to H+/K+ - ATPase may also be antibodies to Na+/K+ - ATPase, thus determining atrial damage.

But on the other hand, Jackie's posting above article said H. Pylori may be inhibited by raising stomach acid or lowering its pH which you get helped by PPI, which lower your stomach acid. I understand some of you guys got helped from PPI and I really would like to find out why. Also your posting today, you mentioned of your PH to be above 7 which is opposite of acidic and its due to you taking PPI I think?

Its hard to sort out these two because there aren't that much studies out there about afib and HP. Maybe PC could explain to us simple terms:)

I hope your mother is not infected HP but if she is, then this could pass on to person to person.

The way that I understood from GI is that if you never taken antibiotic for HP, then you can do blood test for HP but if you took it in the past, then you have take the breath test for it. Otherwise, it will show positive even though you got rid of HP. I think it has to do with antibodies on your blood. I don't know how long it stayed in your system but I was told long time.

Mira

Mira and Dean,

The whole ion pump/Hp correlation sounds very tenuous to me and I wouldn't put too much stock in it.

Just a bit of clarification. PPIs inhibit the H+/K+ pump, thereby decreasing extracellular H+ (raising pH). Hp infection appears to effect the same change in gastric cells.

Also, most clinicians looking for Hp infection use the breath test. Go to
http://www.questdiagnostics.com/kbase/topic/medtest/hw1531/descrip.htm
for more detail on all the diagnostic tests for Hp. I don't think a test for IgM antibodies to Hp is yet available (detects recent infection). A positive test for IgG antibodies to Hp would only indicate a past infection (recent or remote).

PC

Hi PC,

Glad to hear the sharks didn't get you in Aussie. A couple of yrs back the reef tour operators left a pair of Yanks behind who were swimming out on the reef and the tiger sharks got them. Hope you enjoyed your trip.

Yes, I agree with you that whole ion pump/Hp correlation sounds very tenuous. I do agree with the Italian research

that the majority of afibbers have some sort of gastric problems. The number of posts you read on this Bulletin board mentioning heartburn, reflux, GERD etc. is staggering. There must be a link with afib somewhere.

Dean

PC - Thanks for your input. I really do think that this is the area I should be focusing on since the research report is very similar to my situation.

I do agree with Dean that lot of us having reflux, GERD, etc. tied to afib but yet we don't know why and there has been no research into this area.

However, you do agree that if person has autoantibodies attacking the cardiac would cause afib? Below is your quote.

Presumably by damaging the proton pump (H+/K+ pump) this antibody is decreasing gastric HCl thereby increasing intracellular H+ and decreasing intracellular K+. If this antibody is also attacking the cardiac H+/K+ pump, then one can see why Hp infection might predispose to AF. If this is happening, then one might expect to see an increase in AF in those on PPIs (proton pump inhibitors). I don't think Dean has experienced such.

Mira

Mira,

Here is some of PC's older posts about PPI's, proton pump descriptions and why they could reduce afib. I am sure this could help your research:

From PC,
“Use of a PPI to raise blood potassium (in this case anorexia nervosa) is exactly what I suggested in the present CR topic of discussion. You'll recall my query to you about this mechanism operating in addition to decreased vagal stimulation for the improvement in LAF with such treatment.

"Another interesting question is why proton pump inhibitors (PPI) not only relieve GERD but also appear to relieve AF. Is it only because there is less irritation of the lower esophagus (and less vagal stimulation)? Or is it also because of an improvement in potassium balance? Gastric acid production is a two step process. First, gastric parietal cells secrete KCl into the lumen. Then the gastric cell H+/K+ pump goes into action and the end result is HCl in the lumen. This latter is the proton pump, because H+ is no more than a proton. By inhibiting it less H+ is lost in the gastric juice. Less potassium is lost in the urine, because the blood is less alkaline (since the gastric fluid is less acidic). Who knows what the critical factor is? Perhaps

“Regarding the stomach acid/potassium thing and gastric parietal cells. H+ and Cl- go into the gastric fluid from these cells. At the same time K+ and HCO3- go from these cells into the blood on a one for one basis. HCO3- makes the blood alkaline and HCO3- must be eliminated in the urine to maintain the proper blood pH. To maintain electrical neutrality the HCO3- must drag a cation out with it. And this cation has to be either H+ or K+. Excreting H+ will only result in a more alkaline blood. So, it excretes K+.

Therefore, less gastric acidity translates to less blood alkalinity which translates to less HCO3- (and K+) excreted in the urine. This means a smaller potassium gradient, which means less PACs (and PVCs)”
PC

Mira, you seem to be very knowledgeable about this topic. I hope you can solve the puzzle for us.

Dean

Great Smokies Diagnostic Lab says: "HpSA (Helicobacter pylori stool antigen)...Direct stool testing of the antigen (HpSA) is highly accurate and is appropriate for diagnosis and follow-up for infection." So they say.

Trent

Dean,

I'm not sure I'm knowledgeable on this topic but I'm trying to understand why we ended up with Afib without explanation.

Even though I maybe helped with PVA but I ended up having 2nd PVA in 5/05. So there must be underlying problem causing this.

It seems to me antibody is attacking the cardiac cell because when I had HP, it produced antibody and some people develop antibody to proton pump but in my case, didn't and it ended up attacking cardiac cell, thus, I ended up with afib. I too, afib is always related to digest issue. This theory is based on positive blood tests for HP and autoantibodies, as well as digest problems and afib.

Let said I got rid of HP but I still have antibodies in my blood which it may still attacking cardiac cell.

I took blood tested for autoantibodies and I will be seeing Rheumatologist tomorrow for the test results. I will be asking him if there are autoantibodies for HP. If I have autoantibodies, then I need to find out how to get rid of it? I hope there is solution to getting rid of autoantibodies! I will update you after I talk to Rheumatologist tomorrow.

Mira

I just got back from visiting Rheumatologist about my autoantibodies blood test results. He said all came out negative, thus, he will not answer me questions about H. Pylori to autoantibodies. I showed him the Itarian Research about HP and antoantibies but he pointed out that the title says A Possible pathogenic link that he doesn't want to get into "possibilities". He said anything could be possible under these circumstances and nothing has been proven. He said I do not have medical back ground to asking these questions. He is kind of Dr that he doesn't like patient asking questions to Dr's. He said when it comes to Afib, you don't have to have reason for it.

Anyway, I was disappointed but good news is that these tests came out negative. As Hans said this is a do it yourself project.

Thanks
Mira

I was at the CCF yesterday with a friend who was consulting in the Gastroenterology department and I picked up a printout of an NIH publication on H. pylori .... These are the etiology highlights--

" No one knows for sure how H. pylori spreads, so prevention is difficult. Researchers are trying to develop a vaccine to prevent infection."

"Researchers are not certain how people contract H. pylori, but they think it may be through food or water."

"Researchers have found H. pylori in the saliva of some infected people, so the bacteria may also spread through mouth-to-mouth contact such as kissing."

"H. pylori is able to survive stomach acid because it secretes enzymes that neutralize the acid. This mechanism allows

H. pylori to make its way to the 'safe' areas - the protective mucous lining. Once there, the bacterium's spiral shape helps it burrow through the mucous lining."

(I would presume that in people with low stomach acid, it's a 'shoe-in' to become infected.)

Resource:
http://digestive.niddk.nih.gov/ddiseases/pubs/hpylori/

Jackie

I found this to be a bit interesting. Awhile back, I believe it was James, but can't be sure, said that his brother had thrombocytopenia. This was a June 2005 study:

Idiopathic thrombocytopenic purpura (ITP), an autoimmune disease caused by sensitization of platelets by autoantibodies leading to platelet destruction, has been associated with some infectious agents, including Helicobacter pylori. The study by Suzuki et al., published in this issue, provides further evidence of the role of H. pylori infection in the pathogenesis of ITP, as confirmed by the increase in the platelet count in patients with ITP, following H. pylori eradication. Interestingly, H. pylori infection has also been shown to play a role in other diseases in which autoimmune mechanisms may be predominant, such as acne rosacea, idiopathic chronic urticaria, and atherosclerosis. While H. pylori eradication is usually recommended in patients with gastric diseases, there are no specific indications for extraalimentary diseases. In the light of the recent findings, a revision of the current guidelines for the management of H. pylori infection may be needed.

http://www.blackwell-synergy.com/doi/abs/10.1111/j.1572-0241.2005.50224.x;jsessionid=lJIkjkdWucR4?cookieSet=1&journalCode=ajg

You all might find this article of interest.

http://www.vianet.net.au/~bjmrshll/newscientist/page1.htm

Richard

I was googling another topic and happened upon the below article. It seems to underscore the probable vagal (v. autoantibody) connection between Hp and AF.

Autonomic control of heart period in duodenal ulcer patients - insights from spectral analysis of heart rate variability.

Lucini D, Cerchiello M, Basilisco G, Cainelli M, Bianchi PA, Fiorelli G, Malliani A, Pagani M.
Centro Ricerca sulla Terapia Neurovegetativa, Medicina Interna I, Ospedale L. Sacco, CNR, Universita di Milano, Italy.

Beyond the fundamental pathogenetic importance of Helicobacter Pylori a possible additional role of vagal innervation in favouring or modulating the clinical history of duodenal ulcer (DU) has been suggested by old studies employing invasive methodologies. Aim of this study was to assess whether vagal prevalence in autonomic modulation was present in healed DU patients (n=20) as compared to controls,(n=50), using a validated non-invasive methodology, based on spectral analysis of cardiovascular variability. This approach provides markers of the sympathetic and vagal modulations of the SA node, respectively by way of the normalized low frequency (LF(RR)) and high frequency (HF(RR)) components of RR interval variability; LF/HF ratio furnishes a marker of sympatho-vagal balance. In addition, sham feeding (SF) provided a means to assess, in DU patients, neurally mediated acid secretion, as the SF acid output (SAO) to basal acid output (BAO) ratio (SAO/BAO). Results showed that LF(RR) was smaller in DU patients than in controls (40.3+/-3.9 vs. 52.3+/-2.3 normalized units, nu; P<0.05). On the contrary, HF(RR) was greater (52.1+/-3.7 vs. 35.7+/-2.3 nu; P<0.05). Conversely the LF component of SAP variability, a marker of sympathetic vasomotor modulations, and the index alpha, a measure of baroreflex control of the SA node, as well as respiratory patterns, were similar in the two groups. SAO/BAO ratio was significantly correlated with markers of autonomic control of the SA node

(r = -0.67, P<0.0083 with HF(RR)). In conclusion results suggest an enhanced vagal modulation of heart period in DU patients at rest, that appears linked to indices of neurally mediated gastric acid secretion response.
Auton Neurosci. 2000 Nov 1;84(3):122-9
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=11111844&query_hl=20

PC

My 3 black cats are happy -

I got my serum Helicobacter pylori test back & no antibodies were detected. After I asked my GP to order the test, I saw Trent's post on 6/21 and remembered that I had the Great Smokies Diagnostic Lab CDSA test last fall which also detected no Helicobacter pylori stool antigen. In addition, my CRP at that time was low, so little inflammation. If I had remembered about the CDSA test, I probably wouldn't have ordered the recent serum test, however it is good confirmation.

George

George - That is great news! Now you have one less thing to worry.

Mira
