Comment on ''Helicobacter pylori infection may improve the survival of patients with carcinomas''

In this short correspondence, the authors hypothesized Helicobacter pylori infection may promote the development of immune response and thus improve survival rates of patients who have received complete resection of their tumors [1]. The authors based their hypothesis on the fact that patients with Helicobacter pylori infection had a high level of cytokine production. However, before considering it as one potential strategy for tumor therapy, we thought some issues need to be discussed.

Firstly, the Helicobacter pylori is a class I carcinogen and is associated with increased risk of gastric cancer while bacterial eradication substantially decreases incidence of gastric cancer [2]. If Helicobacter pylori conduce to outgrowth of gastric cancer which may represent the failure of immune surveillance but trigger an effective anti-tumor immunity which might improve the prognosis after the tumor resection, what might be the underlying mechanism responsible for this conversion?

Secondly, even though the cytokines may promote immune response, the negative effect of Helicobacter pylori product on anti-tumor immunity [3] should not be neglected.

Lastly, as for the production of cytokines, cytokine gene polymorphisms directly influence interindividual variation in the magnitude of cytokine response, and this clearly contributes to an individual's ultimate clinical outcome (duodenal ulcer phenotype, gastritis phenotype, or gastric cancer phenotype) [4]. If the cytokine imbalance might be responsible for disease progression and may decide about the disease outcome after Helicobacter pylori infection, whether it had effect on the intensity of anti-tumor immunity and the prognosis of gastric cancer after tumor resection?

In fact, we speculate that it is difficult to evaluate the impact of Helicobacter pylori infection on anti-tumor immunity. The Helicobacter pylori-induced inflammation itself might be a two-edged sword, whether promote or inhibit anti-tumor immunity is determined by the balance of positive and negative effects related to the complex host-bacterium interaction. Thus, only a portion of gastric cancer patients could benefit from Helicobacter pylori infection.

References

[1] Hong L, Han Z, Qiao T, et al. Helicobacter pylori infection may improve the survival of patients with carcinomas. Med Hypotheses 2008;70:206—7.
[2] Wong BC, Lam SK, Wong WM, et al. Helicobacter pylori eradication to prevent gastric cancer in a high-risk region of China: a randomized controlled trial. JAMA 2004;291:187—94.
[3] Betten A, Bylund J, Christophe T, et al. A proinflammatory peptide from Helicobacter pylori activates monocytes to induce lymphocyte dysfunction and apoptosis. J Clin Invest 2001;108:1221—8.
[4] Lochhead P, El-Omar EM. Helicobacter pylori infection and gastric cancer. Best Pract Res Clin Gastroenterol 2007;21:281—97.

Wei Zhang
Chengxiang Shan
Ming Qiu
Minimally Invasive Surgery Center,
Changzheng Hospital,
Second Military Medical University,
Shanghai 200003, PR China
Tel.: +86 21 63610109x73943; fax: +86 21 63610109
E-mail address: qiuming2006@yahoo.cn (M. Qiu)

Wei Tang
Department of Surgery,
Graduate School of Medicine,
University of Tokyo,
Tokyo 113-8655, Japan

doi:10.1016/j.mehy.2008.03.016

Parasympathetic nervous system: A silent killer in the early morning hours?

Dear Editor,

Myocardial infarction (MI) is one of the leading causes of sudden death in the world. The so called symbol of love, a four chambered organ, the heart, because of its importance has its own super specialty doctors like the cardiologist and the cardiac surgeon. Lot of research has been done and is in

progress on the etiology and prevention of MI. Leaving aside the major causes of MI, namely, high blood cholesterol level, hypertension and emboli formation, even the circadian pattern of blood pressure and heart rate is known to play a major role in MI with the event being more common during the early morning hours [1].

The heart being a visceral organ is supplied by the autonomic nervous system. At the primitive level, the sympathetic and parasympathetic nervous systems have opposite effect on the heart. The parasympathetic nervous supply to the heart is via the vagus nerve, which decreases the heart rate and constricts the coronary arteries. Being known as the resting nervous system, the parasympathetic nervous system is also necessary for the evacuation of the bladder and the rectum.

Even though micturition and defecation can be brought about by reflex arc at the spinal level, the higher centres play a major role in the control of these reflexes. The sacral part of parasympathetic nervous system is located in the lateral horns of S2, 3, 4 part of the spinal grey matter. The stretch sensation from the bladder and rectal wall will reach this spinal level by a visceral afferent neuron and to the higher centre by the ascending autonomic pathways. As mentioned earlier, the parasympathetic nervous system can contract musculature of the bladder and the rectum, and also relaxes the sphincters at the spinal level. However, major noradrenergic fibers from the brain stem descend down and end in various nuclei including the nucleus of vagus nerve. This pathway can stimulate the vagus nerve which acts on the heart.

Since there is a general tendency in humans to attend the call of nature in the mornings, the descending autonomic pathways can stimulate the vagus nerve. This in turn makes the vagus nerve to over shoot on a compromised heart, causing it to stop. This mechanism when coupled with the circadian pattern of blood pressure and heart rate, no doubt, makes the parasympathetic nervous system a silent killer in the early morning hours.

Reference

[1] Elliott WJ. Cyclic and circadian variations in cardiovascular events. Am J Hypertens 2001;14:291S—5S.

Stany W. Lobo
Department of Anatomy,
Melaka Manipal Medical College,
Manipal, India
Tel.: +91 9901727075
E-mail address: stanmcoms@yahoo.com

Ritesh G. Menezes
Tanuj Kanchan
Shrinidhi
K.S. Muhammed Sameer
Department of Forensic Medicine and Toxicology,
Kasturba Medical College, Mangalore, India

Abul Fazil
Department of General Medicine,
Kasturba Medical College,
Mangalore, India

Aman Chauhan
Undergraduate Student of Medicine,
Kasturba Medical College,
Manipal, India

doi:10.1016/j.mehy.2008.03.015

Etiology of dizziness, tinnitus, and nausea in idiopathic intracranial hypertension

Idiopathic intracranial hypertension (IIH) is a disease of unknown etiology predominantly affecting obese females of childbearing age [1]. The visual complications of IIH have been the most feared and studied to date. However, dizziness, tinnitus, and nausea are also associated with IIH and can be incapacitating for some patients. Although well documented as components of IIH, the etiology of dizziness, tinnitus, and nausea remains unexplained.

Visual loss in IIH is attributed primarily to optic nerve compression [1]. Recent reports have suggested a lack of neuroanatomic basis for preferential compression of the optic nerve in IIH compared to other cranial nerves [2]. The evenly elevated pressure within the cranial vault in IIH suggests a more global neurologic syndrome.

Our hypothesis is that dizziness, tinnitus, and nausea in IIH are secondary to a compressive