

The role of helicobacter pylori in vitiligo

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Degree in Dermatology and Andrology***

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Abstract

Introduction. Recent evidence suggests that helicobacter pylori infections play a role in the pathogenesis of a variety of skin diseases. Various mechanisms have been proposed in an attempt to explain the extra intestinal manifestations of H. pylori infections. They are based on the ability of H. pylori to initiate systemic immune response whether humoral or T cell mediated. Vitiligo is a generalized depigmented disorder manifesting as acquired white patches due to loss of melanocytes. Immune dysregulation play an important role in the pathogenesis of vitiligo. So, we may propose that H. pylori may play a role in vitiligo through triggering an immune response. **Aim of the Work.** To clarify if helicobacter pylori plays a role in the pathogenesis of vitiligo or not. **Patients and methods.** The study included 40 patients with different types of vitiligo without GIT symptoms as well as 40 healthy volunteers served as control. Anti Helicobacter pylori Igs (IgG, IgA, IgM) was assessed in the serum of vitiligo patients and controls using ELISA Test. **Results.** An overall anti H. pylori IgG antibody seropositivity of 57.5% (n = 23) was found in vitiligo patients compared with an overall value of 30% (n = 12) for the control group. The difference in seropositivity between patients and controls was statistically significant (P-value = 0.013). An overall anti H. pylori IgA antibody seropositivity of 32.5% (n = 13) was found in vitiligo patients compared with an overall value of 15% (n = 6) for the control group. The difference in seropositivity between patients and controls was not statistically significant (P-value = 0.066). Anti H. pylori IgM antibody seroprevalence of the vitiligo patients and controls was negative. When comparing the IgG level in the patient and control groups, the mean expression level of IgG in the serum of vitiligo patients was higher than in the control serum,

and this difference is statistically significant (P Value = 0.041). While the mean expression level of IgA, IgM in the serum of vitiligo patients was higher than in control group but this difference is statistically not significant (P-value= 0.060, 0.580 for IgA, IgM respectively).

Conclusion. Increased prevalence of H. pylori infection in association with vitiligo, together with the high level of anti H. pylori IgG in vitiligo patients compared to control are with our concept that H. pylori infection may play a role in the pathogenesis of vitiligo.

Keywords. H. pylori, vitiligo, ELISA Test, Anti Helicobacter pylori Igs.



Introduction

Introduction

Helicobacter pylori is a Gram negative, microaerophilic bacterium that can inhabit various areas of the stomach and duodenum. It is one of the most common pathogens affecting humans, infecting approximately 50% of the world's population. It is found more frequently in developing countries than in industrialized countries, presumably due to poor sanitary condition (*Lehours and Yilmaz, 2007*).

The outcome of the infection depends on a combination of factors: bacterial virulence, host factors, and environmental factors (*Uemura et al., 2001*). It is strongly linked to the development of duodenal and gastric ulcers as well as cancer stomach. Over 80% of individuals infected with the bacterium are asymptomatic (*Boyanova, 2011*).

The bacteria have been isolated from faeces, saliva and dental plaques of infected patients, which suggests that the fecal oral route is a possible transmission mode (*ndip et al., 2003*).

Recent evidence suggests that helicobacter pylori infections play a role in the pathogenesis of a variety of skin diseases. The best evidence for such a link is found for two diseases: chronic urticaria and immune thrombocytopenic purpura. Other diseases that have a purported, but not yet proven link to H. pylori are: cutaneous pruritus, Behçet's disease, nodular prurigo and lichen planus. Lastly, single or few case reports have documented associations between Helicobacter pylori infection and rosacea, aphthous stomatitis, atopic dermatitis, alopecia areata, Henoch-Schoenlein purpura and Sjögren syndrome, but these are only descriptive in nature (*Hernando et al., 2009*).

Various mechanisms have been proposed in an attempt to explain the extra intestinal manifestations of *H. pylori* infections. These include: atrophic gastritis, an increase in gastric vascular permeability during infection, release of inflammatory mediators, molecular mimicry and systemic immune response. As an example, antigastric autoantibodies have been found in more than 30% of patients who are infected with *H. pylori* (**Lehours and Yilmaz, 2007**).

An increase in permeability of the gastric and intestinal mucosa in infected patients has also been demonstrated (**Magalhaes Queiroz and Luzza, 2006**), and may result in increased exposure to alimentary antigens. Of note is that it is well known that the immunological response elicited by *H. pylori* is an important determinant of the amount of gastric mucosal damage. Thus the production of large amounts of various proinflammatory substances, such as cytokines, eicosanoids, and proteins of the acute phase, follows gastric colonisation by *H. pylori* (**Negrini, 1996**). This inflammatory response may lead to the development of antigen-antibody complexes or cross-reactive antibodies (by molecular mimicry) resulting in damage to other organs (**Uemura et al., 2001**).

It has been proposed that *H. pylori* induces a phenomenon similar to that seen in the molecular mimicry between hemolytic streptococcus group A antigens and host proteins resulting in both humoral and cell mediated autoimmune reactions and ultimately causing rheumatic fever and rheumatic heart disease (**Fae et al., 2008**). Based on these observations, investigators have examined the role of *H. pylori* as a pathogenic determinant for idiopathic extra intestinal diseases, in which immune dysregulation is implicated. A competing theory that is also being discussed is that an infection induced immune response continues after the pathogen has been eradicated. This could explain why patients with confirmed eradication therapy failed to show improvement in short term observations (**Hernando et al., 2009**).

Vitiligo is one of the most common skin disorders with a prevalence of 1-2% in different populations. The condition occurs when pigmented cells are destroyed, causing patches of skin to lose their normal color and appear whiter (*Alkhateeb et al., 2003*).

The etiology of this disorder is not clear, it is a multifactorial polygenic disorder with a complex pathogenesis. At present, there have been various causative factor implicated, these include genetic predisposition, autoimmune, neurogenic and biochemical theories (*Ongenaek et al., 2003*).

From these factors, autoimmune disorder is the most common cause and some of the patients have antibodies to melanocytes or melanocytic proteins. Also, there is some evidence that cell-mediated immunity plays role in melanocyte destruction (*Yu et al., 1993 & Vanden et al., 2000*).

Thus immune dysregulation play a role in the pathogenesis of vitiligo. As *H. pylori* was found to play a role in the pathogenesis of some skin disease most probably through induction of immune dysregulation (*Hernando et al., 2009*). We purpose that *H. pylori* infection may play a role in the pathogenesis of vitiligo. To the best of our knowledge, no previous studies were reported on the relation between helicobacter pylori and vitiligo.



Aim of the Work

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To clarify if helicobacter pylori plays a role in the pathogenesis of vitiligo or not.



Review of literature
