

Serum H Pylori Antibodies and Thyroid Disorder in Chronic Urticaria

Shantha Kumari.N¹, Karthik G², Nataraj B³

1. Associate professor, Department of Biochemistry, Dr B R Ambedkar Medical College, Bangalore, India.

2. Assistant professor, Department of Biochemistry, Dr B R Ambedkar Medical College, Bangalore, India

3. Assistant professor, Department of Biochemistry, Dr B R Ambedkar Medical College, Bangalore, India

Corresponding Author: Karthik G

Abstract:

Introduction: Chronic urticaria (CU) is the most common pruritic condition with multiple casual factor characterized by erythematous, edematous and pruritic lesion of dermis and hypodermis 50% of patients with chronic urticaria have autoimmune basis. Thyroid disorders and H pylori have been a major contribution to chronic urticaria.

AIMS and objectives: To estimate RBS, H pylori IgG, IgM, TSH in patients with chronic urticaria and comparing with control group. To evaluate the importance of these biochemical parameters in chronic urticaria patients.

Methodology: 30 patients with symptoms of chronic urticaria and 28 controls were considered for the study. Random Blood samples was collected and evaluated for H pylori IgG, IgM, RBS and TSH. H pylori antibodies was analysed by Elisa method. TSH was estimated by immunoassay method and RBS by hexokinase- G6PD method.

Results: RBS in cases and controls was 119.07 ± 62.9 and 90.67 ± 11.17 , respectively with significant p value of 0.022. TSH in cases and controls was 4.11 ± 2.26 and 2.47 ± 1.42 , respectively with significant p value of 0.02. H pylori IgG in cases and controls was 0.83 ± 0.58 and 0.46 ± 0.43 , respectively with significant value of 0.034. H pylori IgM in cases and controls was 0.79 ± 0.55 and 0.52 ± 0.35 , respectively, with significant value of 0.028.

Conclusion: RBS, TSH, H pylori IgG and IgM was significantly high in cases than controls, suggesting a strong role of diabetes, and autoimmune role in urticaria.

Key words: Urticaria, Hpylori IgG, Hpylori IgM, TSH.

Date of Submission: 22-02-2018

Date of acceptance: 08-03-2018

I. Introduction

Chronic urticaria (CU) is the most common dermatologic disorder characterized by erythematous, edematous and pruritic lesion of dermis and hypodermis with complex pathophysiologic mechanisms. Various causes are viral, bacterial fungal and protozoal infections, psychological factors, food and food additives, auto antibodies, drugs like, salicylates and NSAIDS, premenstrual period in women alcohol etc.,. Sometimes etiology remains unclear, known as chronic idiopathic urticaria.

H pylori is a gram negative micro aerophilic spiral, flagellated bacteria, that affect humans at any age or sex (1). It is oxidase, catalase and urease positive bacilli. Because of its mobility, it resides and colonizes in stomach (2). It is the causative agent for gastrointestinal condition like chronic gastritis, peptic ulcer, gastric and duodenal carcinoma. Hpylori also have extra-intestinal pathologies like autoimmune, metabolic, neurologic and dermatological disease (3). In South India, very minimal studies are done to substantiate association of chronic urticaria with H pylori and other autoimmune disorders. Our study aims to correlate chronic urticaria with H pylori Ig G, Ig M, TSH and sugar levels.

II. Methodology

Study group consists of 60 participants with age group between 20 to 50 yrs of both sex. 30 participants diagnosed with chronic urticaria was considered as cases. 30 controls are normal subjects without any features of urticaria were taken as controls. Under aseptic precautions, 5 ml of venous blood was collected and serum was estimated for RBS, TSH, H pylori IgG and IgM. RBS was estimated by Hexokinase-G6PD method using commercially available kit in Seimens dimension Xpand autoanalyser. TSH was estimated by immunoassay method using commercially available kit in Advia centaur CP immunoassay system. H pylori was estimated by

ELISA method using commercially available calbiotech kit in Alere Elisa instrument. The data was analysed using SPSS version16, students independent ‘t’ test and p value significance.

III. Results

Table 1 shows that the mean age of cases and controls was 37.5 ± 2.5 and 38 ± 3.2 years, respectively and age was matched. Table 2 shows that sex was also matched between cases and control groups. Table 3 shows the biochemical values of RBS, TSH, H pylori IgG and H pylori IgM. RBS in cases and controls was 119.07 ± 62.9 and 90.67 ± 11.17 , respectively with significant p value of 0.022. TSH in cases and controls was 4.11 ± 2.26 and 2.47 ± 1.42 , respectively with significant p value of 0.02. H pylori IgG in cases and controls was 0.83 ± 0.58 and 0.46 ± 0.43 , respectively with significant value of 0.034. H pylori IgM in cases and controls was 0.79 ± 0.55 and 0.52 ± 0.35 , respectively, with significant value of 0.028. This shows that RBS, TSH, H pylori IgG and IgM is increased in cases compared to controls suggesting a strong role in chronic urticaria.

Table 1: Age wise comparison in two groups studied

	Cases	Controls	P value
Age	37.5 ± 2.5	38 ± 3.2	0.508

Table 2: Sex wise comparison in two groups studied

Sex	Test group		Control group		p-value
	No	%	No	%	
Male	16	55.17	13	44.83	0.599
Female	14	48.28	15	51.72	

Table 2: Comparison of Biochemical parameters in cases and controls

Parameter	Cases	Controls	P value
RBS	119.07 ± 62.9	90.67 ± 11.17	0.022*
Ig G	0.83 ± 0.58	0.46 ± 0.43	0.034*
Ig M	0.79 ± 0.55	0.52 ± 0.35	0.028*
TSH	4.11 ± 2.26	2.47 ± 1.42	0.002**

* Significant, **Highly significant

IV. Discussion

30% of patients with chronic urticaria suffer from H pylori infection and the prevalence of chronic urticaria decreased drastically after anti-H pylori treatment suggesting H pylori cause for chronic urticaria (2, 4).

H pylori infection increases gastric acid pepsin secretion. This enhanced acid load will induce gastric metaplasia and inflammation with recruitment of eosinophils, neutrophils and lymphocytes. These Histamine releasing factors mediate the granules, which when released cause mast cell degranulation. The interference with pro inflammatory cytokines and with the expression of epitopes of adhesion to the endothelial cells, triggers systemic immune response. H pylori also produce urease, protease, phospholipase and cytokines which trigger complement response (5).

The other study showed that H pylori increases the permeability of stomach lining and thus increases the exposure to allergens in the gastro intestinal tract. Also, the immune response to H pylori produces antibodies which release histamine in skin (6). H pylori toxin release initiate complement activation process, that is anaphlotoxin C3a and C5a. These complement fragments with specific receptors on mast cells and basophiles release Histamine (7). IgG & IgM antibodies to H pylori associated lipoprotein were found in the pathogenesis of CU (3). But Hellmig et al concluded that there is no evidence that H pylori eradication improves the outcome in patients with CU (8).

There is high prevalence of H pylori infection with type I/II Diabetes mellitus correlating dyspeptic symptoms, esophagitis, peptic ulcer with cardio vascular autonomic neuropathy. It is related to reduced gastric motility and changes in gastric mucosa following non enzymatic glycosylated process and non specific immunity in diabetic patients (9).

The virulent strains of H pylori are associated with macroangiopathy, neuropathy and micro albuminuria in type II DM, is due to an immune mediated response in the endothelium caused by systemic immune response to infection, leading to albumin leakage (10). There is high chances of H pylori infection in patients with DM due to changes in gastric microvasculature, leading to reduced absorption of antibiotics and also development of resistant H pylori stains (11). H pylori infection many have pathogenic role in the development of insulin resistance (12).

Infection with H pylori initiates immunological response by increasing production of interleukin - 8, platelet-activating factor, leukotriene B4 and C4. The release of auto antibodies by immunogenic bacterial cell wall of H pylori are similar to antiperoxidase antibodies. The monoclonal antibodies against H pylori strains can cross react with follicular cells of thyroid gland. Also the eradication of H pylori antigens is followed by gradual decrease in levels of thyroid auto antibodies, suggesting H pylori antigens involvement in auto immune course of thyroid disease(5).

V. Conclusion

This is understood that chronic urticaria is commonly associated with diabetes mellitus. H pylori antibodies and thyroid antibodies is also associated with chronic urticaria. We infer that diabetic control will minimize the symptoms of urticaria. Treatment of H pylori will also reduce the symptoms of chronic urticaria.

References

- [1] Johanna Hook-Nikanne, Elina Varjonen, Rauno J Harvima and Timo U Kosunen. Is Helicobacter pylori infection associated with chronic urticaria? *Acta Derm Venereol* 2000; 80: 425-426.
- [2] PV S Prasad. Urticaria; *Indian J of Dermatol Venereol Leprol* 2001; 67:11-20.
- [3] Majid Mogadam Rostamy. Prevalance of the Helicobacter pylori infection in chronic urticaria. *Journal of Pakistan Association of Dermatologists* 2010; 20:142-145.
- [4] Nasreen Qazi, Azam J Samadani, Shahnawaz Jamali, et al. Chronic Idiopathic urticaria and helicobacter pylori infection: Effect of eradication therapy on the relief of symptoms. *JLUMHS*: 2013;vol 12: No. 03
- [5] Sheema Mohsin Mustafa and Ahmed Bolad: The prevalence of Helicobacter pylori among patients with chronic idiopathic urticaria. *Vol.3, No.16 ISSN 1858-6155*.
- [6] Buhner S, Reese I, Kuehl F, et al. Pseudoallergic reactions in chronic urticaria are associated with altered gastroduodenal permeability. *Allergy* 2004; 59:1118-1123.
- [7] Rossano Valsecchi and Paolo Pigatto. Chronic Urticaria and Helicobacter pylori. *Acta Derm Venereol (stockh)* 1998; 78:440-442.
- [8] S. Hellmig, K. Troch, S. J. otto. et al. Role of H pylori infection in the treatment and outcome of chronic urticaria. *Helicobacter*. 2008; 13: 341-345.
- [9] Konstantinos X Papamichael, Garyphallia Papaioannou, et al. Helicobacter pylori infection and endocrine disorders: Is there a link. *World J Gastroenterol* 2009, 14; 15 (22): 2701-2707.
- [10] Pietroiusti A, Giuliano M, et al. Cytotoxic-associated gene A strains of Helicobacter pylori represent risk factor for the development of microalbuminuria in type 2 diabetes. *Diabetes care* .2006;29:1399-1401.
- [11] Sargyn M, Uygur Bayramicli O, et al. Type 2 diabetes mellitus affects eradication rate of Helicobacter pylori. *World j Gastroenterol* 2003; 9: 11261128
- [12] Polyzos. SA, Kountouras J, Zaros C. Deretzi .G. The association between H pylori infection and insulin resistance a systemic review. *Helicobacter* 2011;16: 79-88.

IOSR Journal of Biotechnology and Biochemistry (IOSR-JBB) is UGC approved Journal with Sl. No. 4033, Journal no. 44202.

Shantha Kumari.N " Serum H Pylori Antibodies and Thyroid Disorder in Chronic Urticaria" IOSR Journal of Biotechnology and Biochemistry (IOSR-JBB) 4.2 (2018): 09-11