IJFANS INTERNATIONAL JOURNAL OF FOOD AND NUTRITIONAL SCIENCES

ISSN PRINT 2319 1775 Online 2320 7876

Research Paper © 2012 IJFANS. All Rights Reserved UGC CARE Listed (Group -I) Journal Volume 11, Iss 09, 2022

Prevalence of Helicobacter pylori in Recurrent Apthous Stomatitis

Jyoti Kiran[1*], Vishal Mehrotra[1], Kriti Garg[1], Rahul Srivastava[1], Sachin Kushwah[1], Shiv Kumar Singh[2]

[1]Rama Dental College, Rama University, Mandhana Kanpur, U.P, India

[2]Maharana Pratap Dental College, Kanpur

Email id: kiranjyioti876@gmail.com

Abstract

Background/Aims: The aim of this study was to determine probable Helicobacter Pylori infection in recurrent apthous stomatitis samples by Rapid Urease Test (RUT).

Materials and Methods: This in vivo cross-sectional study was approved by the Ethics Committee of Rama Dental College Hospital and Research Centre according to the ethical standards. A total of 30 patients with minor recurrent aphthous stomatitis and 20 healthy control groups were included in the study.

Results: Out of 30 patients with minor recurrent aphthous stomatitis, including 14 male and 16 female patients, with mean age of 47 and 38 years respectively, 19 patients (68%) were RUT (positive). Out of 20 healthy control groups, 10 males and 10 females, 3(11%) were RUT (positive) [Table 1]. Conclusion: HP may play a role in the etiology of RAS; it is likely that RUT may be rapid and reliable for investigation of HP in RAS lesions.

Conclusion: compared to healthy individual there was higher prevalence of H.pylori found in patient with recurrent Apthous stomatitis

Keywords: rapid urease test, antibody, infection, ulcer, stress

Introduction

The term recurrent aphthous stomatitis (RAS) refers to the most common inflammatory ulcerative disorder of the oral mucosa.[1] RAS is characterized by painful superficial ulcers on nonkeratinized or poorly keratinized mucosa and is generally localized.[2] The etiopathogenesis of RAS is unclear, although many possible predisposing factors have been implicated such as trauma, emotional stress, hormonal state, food hypersensitivity, viruses, bacteria, and immune dysregulation.[3] It has also been reported that type 1 helper genes are locally overexpressed and cytokines such as interleukin (IL)-2, tumour necrosis factor-α, and IL-6 are systemically produced by circulating mononuclear cells. IL-10 messenger RNA levels were reported to be decreased which suggests the immune system cannot effectively suppress the inflammatory reaction against the oral mucosa.[4,5] Previous studies have reported that increased cell-mediated immune responses against certain areas of the oral mucosa secondary to an abnormally regulated cytokine cascade lead to ulceration.[5] RAS etiopathogenesis may also include genetic factors, and RAS patients have an increased prevalence of HLA-B12 and Behcet's disease, which is a disorder characterized by recurrent aphthous ulcers and is associated with increased HLA-B5 levels.[6] It has been reported that IL-1β and IL-6 gene polymorphisms are associated with a significant risk for the development of RAS. However, associations between specific IL-10 or IL-12 gene polymorphism and RAS susceptibility were not demonstrated in prior studies.[7-9] Studies have shown that a G allele at the −174

ISSN PRINT 2319 1775 Online 2320 7876

Research Paper © 2012 IJFANS. All Rights Reserved UGC CARE Listed (Group -I) Journal Volume 11, Iss 09, 2022

position is associated with a higher IL-6 production than a C allele at the same position[10].Similar relationships between IL-1β −511 genotype and IL-1β production were also suggested.[11] The important etiological agents in the etiopathogenesis of RAS suggested as Helicobacter pylori (HP) in recent years. The HP infection is diagnosed by several methods, both invasive and non-invasive. The sensitivity and specificity of these assays range from 80% to 95% depending on the assay used.

Methodology

This in vivo cross-sectional study was approved by the Ethics Committee of Rama Dental College Hospital and Research Centre according to the ethical standards. A total of 30 patients with minor recurrent aphthous stomatitis and 20 healthy control groups were included in the study.

After obtaining a written consent, a detailed case history related to oral ulceration will be recorded, and thorough clinical examination will be carried out when the individual has active ulcerated lesion to confirm the diagnosis. Once the clinical diagnosis is done, the individual is selected to the study group. They had no chronic illness in both patients and control group. A detailed history (family history of the RAS, time of onset, annual recurrence rate, predisposing factors, number of aphthous lesions in last 3 months, localizations, diameters, and improvement time of the lesions) and complete dermatological examination were performed. Pathergy test, ophthalmologic examination, complete blood counting, routine biochemical tests, serum iron, folic acid, and Vitamin B12 levels were assessed. Hepatitis markers and anti-HIV antibodies were studied. The patients with normal laboratory examination were included in this study. Behcet's disease was excluded from this study. The major and herpetiform RAS was not included in the study. The diagnosis of recurrent oral aphthous stomatitis was given after taking a detail medical history, by thorough general and oral examination and by excluding or ruling out other deficiency states or other systemic or immunological conditions, through routine and special hematological tests.

The patients were given a questionnaire, which included questions on the presence of gastrointestinal disorders or a positive history of peptic ulcer. Only those who had not consumed any antibiotics in the last month and did not use any steroidal drugs were tested. The active aphthae of these patients were sampled with rapid urease test (RUT). The samples to be studied include tissue scrapings obtained from the ulcerated lesion using a sterile curette. 10% lidocain spray was applied to oral ulcer of the patient and buccal mucosa of the control. The samples were obtained by scraping the lesion of RAS in patients and buccal mucosa of the controls with a sterile curette. The collected samples were subjected to RUT for the detection of HP, the kits; helicorapt kit was obtained from zenith pharmaceuticals. RUT kit was stored at +4°C until usage. The scraping samples from ulcer and mucosa were stored at the room temperature then placed into kits. Color change was checked respectively at the 30th, 60th min and at the 4th h (as recommended by the company). If the color of the test is yellow or yellow-green, the result is negative but red or pink is considered as a positive test result. Grading of the results was be done as strongly positive, positive, and weakly positive. The results obtained were statistically analyzed.

ISSN PRINT 2319 1775 Online 2320 7876

Research Paper © 2012 IJFANS. All Rights Reserved UGC CARE Listed (Group -I) Journal Volume 11, Iss 09, 2022

Results

Out of 30 patients with minor recurrent aphthous stomatitis, including 14 male and 16 female patients, with mean age of 47 and 38 years respectively, 19 patients (68%) were RUT (positive). Out of 20 healthy control groups, 10 males and 10 females, 3 (11%) were RUT (positive) [Table 1]. The results obtained were statistically analyzed. The sensitivity and specificity to RUT in RAS patients were significantly higher (Fisher's test P = 0.0063). We have found that RUT is positive in 68% of the patients and 11% of the control group [Table 1]. RUT positivity in RAS patients was significantly higher than control group. We think that HP may play a role in the etiology of RAS;it is likely that RUT may be rapid and reliable for investigation of HP in RAS lesions. The results of studies with the relation of HP and RAS are controversial. In some studies, it has been said that HP had no role in the pathogenesis of RAS using polymerase chain reaction (PCR) and serology by Mansour-Ghanaei et al.,[12] Iamaroon et al.,[13] Shimoyama et al.[14] and Fritscher et al.,[15] whereas a positive correlation has been observed between HP and RAS, using PCR and serology by others.[4,8,16,17] However, there has been no clear conclusion in some studies.[18,19,20] This controversy is probably due to methodological and technical differences. When interpreting our results, it is important to note some of the limitations of our study. First, we had limited number of patients. Second, another diagnostic method showing the existence of HP as histopathology, PCR, antibody was not used. Therefore, false negative and the positivity of the test had not been possible to evaluate exactly. The major and herpetiform RAS was not included in the study.

Table 1: Sociodemographic features and rapid urease test results of the patient and control groups

	SEX		AGE (years) MEAN+SD	RUT (positive)
	Male	Female		
PATIENT (n = 30)	14	16	47.6+1.76	19(68%)
CONTROL GROUP (n = 20)	10	10	38.35+15.92	3(11%)

Risk factors for Helicobacter pylori

The risk factors for HP include poor social economic status, poor hygiene practice, absence of hygienic drinking water, and unsanitary food preparation.[21,22]

Transmission of Helicobacter pylori

The main route of organism entry has been charted as the following – oral to oral, gastro to oral, and faecal to oral. Transmission may occur in a vertical or horizontal mode. Oral carriage of HP may play a role in the transmission of infection.[22] Most infections are probably acquired in childhood, although the exact route of transmission is unknown. These bacteria are also found in plaque and feces, so the route of infection could be oral–oral or fecaloral.[23,24] It has long been speculated that dental plaque might harbour HP and therefore might be a source of reinfection of the gastric mucosa.[25] Because of similarities in the histologic characteristics of gastric ulcers and oral aphthous ulcers, it seems reasonable to assume that HP could play a role in the development of recurrent aphthous ulcers. The pathogenesis of HP infection in humans can be described in three steps[19]: Entry to, adherence to, and colonization of the human gastric mucosa[20]; avoidance, subversion, or exploitation of the human immune system and[26] multiplication, tissue damage, and transmission to a new

ISSN PRINT 2319 1775 Online 2320 7876

Research Paper © 2012 IJFANS. All Rights Reserved UGC CARE Listed (Group -I) Journal Volume 11, Iss 09, 2022

susceptible host or spread to adjacent tissue.[19,20,26] A virulence factor is defined by its involvement in one or more of these processes.

Pathogenesis

HP is well adapted to withstand low pH to gain entry to its preferred territory, the mucus layer of the mucous membrane. One of the survival capabilities is its ability to resist mucosa and migration toward epithelial cell. Once there, the bacterium resists the local and systemic immune responses. Colonization persists for life in the host if there is no exposure to antibiotics.[5] Once it escapes from the lumen, it modifies the bioenvironment in the area and starts releasing collagenases which degrade the collagen in the host enabling more space for the movement of the bacteria. The Fas Ag pathway of apoptosis is activated during HP infection. The combined action of the collagenases and stimulation of apoptosis leads to ulceration of the mucosa.[8]

Recently, HP has been proposed to be one of the important etiological agents in the pathogenesis of RAS. It has been showed that patients with recurrent oral ulcerations appear to suffer from active HP infection in a high percentage of cases.[10]

References

1. Robinson ND, Guitart J. Recalcitrant, recurrent aphthous stomatitis treated with etanercept) Arch Dermatol 2003;139:1259-63.

2. Jurge S, Kuffer R, Scully C, Porter SR. Mucosal Diseases Series, Number VI Recurrent aphthous stomatitis. Oral Dis 2006;12:1-21.

3. Embil JA, Stephens RG, Manuel FR. Prevalence of recurrent herpes labialis and aphthous ulcers among young adults on six continents. Can Med Assoc J 1975;113:627-30.

4. Borra RC, Andrade PM, Silvaet ID, et al. The Th1/Th2 immunetyperesponse of the recurrent aphthous ulceration analyzed by cDNA microarray J Oral Pathol Med 2004;33:140-6.

5. Buno IJ, Huff JC, Weston WL, Cook DT, Brice SL. Elevated levels of interferon gamma, tumor necrosis factor alpha, interleukins 2, 4 and 5, but not interleukin 10, are present in recurrent aphthous stomatitis Arch Dermatol 1998;134:827-31.

6. Riggio MP, Lennon A, Wray D. Detection of Helicobacter pylori DNA in recurrent aphthous stomatitis tissue by PCR. J Oral Pathol aMed 2000;29:507-13.

7. Bazrafshani MR, Hajeer AH, Ollier WE, Thornhill MH. IL-1β and IL-6 gene polymorphisms encode significant risk for the development of recurrent aphthous stomatitis (RAS). Genes Immun 2002;3:302-5.

8. Guimaraes AL, de Sa AR, Victoria JM, et al. Association of interleukin-1 beta polymorphism with recurrent aphthous stomatitis in Brazilianin dividuals. Oral Dis 2006;12:580-3.

9. Bazrafshani MR, Hajeer AH, Ollier WE, Thornhill MH. Polymorphisms in the IL-10 and IL-12 gene cluster and risk of developing recurrent aphthous stomatitis. Oral Dis 2003;9:287-91.

10. Dayanathi AR, Sunitha J, Ramamoorthy A, Jeddy N, Jeeva S. Helicobacter pylori and its role in oral diseases: A note on pathogenesis. J Indian Acad Dent Spec Res 2014;1:2.

11. Pociot F, Molvig J, Wogensen L, Worsaae H, Nerup J. A TaqIpolymorphism in the human interleukin-1 beta (IL-1 beta) 1992;22:396-402.

ISSN PRINT 2319 1775 Online 2320 7876

Research Paper © 2012 IJFANS. All Rights Reserved UGC CARE Listed (Group -I) Journal Volume 11, Iss 09, 2022

12. Mansour-Ghanaei F, Asmar M, Bagherzadeh AH, Ekbataninezhad S. Helicobacter pylori infection in oral lesions of patients with recurrent aphthous stomatitis. Med Sci Monit 2005;11:576-9.
13. Iamaroon A, Chaimano S, Linpisarn S, Pongsiriwet S, Phornphutkul K. Detection of Helicobacter pylori in recurrent aphthous ulceration by nested PCR. J Oral Sci 2003;45:107-10.
14. Shimoyama T, Horie N, Kato T, Kaneko T, Komiyama K. Helicobacter pylori in oral ulcerations. J Oral Sci 2000;42:225-9.
15. Fritscher AM, Cherubini K, Chies J. Association between Helicobacter pylori and Recurrent aphthous stomatitis in children and adolescents. J Oral Pathol Med 2004;33:129-32.
16. Jurge S, Kuffer R, Scully C, Porter SR. Mucosal disease series. Number VI. Recurrent apthous stomatitis. Oral Dis 2006;12:1-21.
17. Scully C, Porter S. Recurrent apthous stomatitis: Current concepts of etiology, pathogenesis and management. J Oral Pathol Med 1989;18:21-7.
18. Preeti L, Magesh K, Rajkumar K, Karthik R. Recurrent apthous stomatitis. J Oral Maxillofac Pathol 2001;15:252-6.
19. Marshall BJ, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. Lancet 1984;1:1311-5.
20. Lehours P, Yilmaz O. Epidemiology of Helicobacter pylori infection. Helicobacter 2007;12:1-3.
21. Makola D, Peura DA, Crowe SE. Helicobacter pylori infection and related gastrointestinal diseases. J Clin Gastroenterol 2007;41:548-58.
22. Assya K, Vladimir P, Adriana K, Angelina K. Oral cavity and systemic diseases — Helicobacter pylori and dentistry. Biotechnol 2011;25:2447- 57.
23. Dowsett SA, Archila L, Segreto VA, Gonzalez CR, Silva A, Vastola KA, et al. Helicobacter pylori infection in indigenous families of Central America: Sero-status and oral and fingernail carriage. J Clin Microbiol 1999;8:2456-60.
24. Kusters JG, van Vliet AH, Kuipers EJ. Pathogenesis of Helicobacter pylori infection. Clin Microbiol Rev 2006;19:449-90.
25. Dye BA, Kruszon-Moran D, McQuillan G. The relationship between periodontal disease attributes and Helicobacter pylori infection among adults in the United States. Am J Public Health 2002;92:1809-15.
26. Kilmartin CM. Dental implications of Helicobacter pylori. J Can Dent Assoc 2002;68:489-93.