

Frequency of Helicobacter Pylori Infection in Patients with Lichen Planus (A Hospital Based Cross Sectional Descriptive Study)

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Abstract: To determine the frequency of Helicobacter pylori infection in patients with lichen planus presented to tertiary care hospital Hyderabad. *Patients and Methods* This cross sectional descriptive study of six months was carried at Liaquat University Hospital Hyderabad Sindh, Pakistan from February 2008 to July 2008. All patients above 12 years of age, of either gender, were known / already diagnosed and newly diagnosed cases of lichen planus (LP) were came through outdoor patient department (OPD), indoor patient and causality outdoor department (COD) were evaluated and enrolled in the study. All such patients were screened for Helicobacter pylori infection by Helicobacter pylori stool antigen (HpSA). For getting result the data was saved and analyzed in SPSS version 10.00. *RESULT:* One hundred and five patients (105) were identified as lichen planus and screened for Helicobacter pylori infection. The Helicobacter pylori stool antigen (HpSA) was positive in 81 (77%) subjects with mean age 42.89 ± 13.32 (SD). Majority of the patient were presented in the department of dermatology; were females and belonged to the rural areas of Sindh province. Other features and presentation identified in such patients were epigastric pain (69%), retrosternal pain (17%) and haemetemesis in 07 patients. *CONCLUSION:* The patients with lichen planus are more prone to acquire the Helicobacter pylori infection.

Key words: Lichen planus % Helicobacter pylori % Helicobacter pylori stool antigen (HpSA)

INTRODUCTION

Lichen planus (LP) is a pruritic dermatosis of unknown etiology that affects skin, oral and genital mucous membranes, nails and hair. The classic form presents with symmetrically distributed, violaceous, papules commonly involving the flexor aspects of wrists, legs and lower back. LP is mostly self limiting and resolves after a variable period ranging from few months to years, leaving behind hyperpigmentation and/or scarring. LP is uncommon in children accounting for less than 2-3% of all adult cases [1]. No significant differences in incidence are noted between male and female patients, but in women, LP may present as desquamative inflammatory vaginitis [2]. More than two thirds of patients are aged 30-60 years; however, LP can occur at

any age [3]. The typical rash of lichen planus is well-described by the "5 P's": well-defined pruritic, planar, purple, polygonal papules.

The mucous lesions are most commonly found on the tongue and the buccal mucosa; they are characterized by white or gray streaks forming a linear or reticular pattern on a violaceous background. Oral lesions are classified as reticular, plaque like, atrophic, papular, erosive and bullous. Ulcerated oral lesions may have a higher incidence of malignant transformation in men, but this observation may be confounded by other factors, such as smoking and chewing tobacco. Lesions may also be found on the conjunctivae, the larynx, the tonsils, the bladder, the vulva and the vaginal vault; throughout the gastrointestinal tract; and around the anus.

The genital involvement is common in men with cutaneous disease. Typically, an annular configuration of papules is seen on the glans. Less commonly, linear white striae, similar to the lesions on the vulva and the vagina, can be seen on male genitalia. Vulvar involvement can range from reticulate papules to severe erosions. Dyspareunia, a burning sensation and pruritus are common. Vulvar and urethral stenosis can also be present. Two reports documented that more than 50% women with oral LP had undiagnosed vulvar LP [4-5]. The patients with a cutaneous eruption may also have follicular and perifollicular violaceous, scaly, pruritic papules on the scalp.

Direct immunofluorescence study reveals globular deposits of immunoglobulin M (IgM) and complement mixed with apoptotic keratinocytes. The histopathologic features distinguish LP based on the presence of irregular acanthosis and colloid bodies in the epidermis with destruction of the basal layer. The upper dermis has a bandlike infiltrate of lymphocytic (primarily helper T) and histiocytic cells with many Langerhans cells. The infiltrate is very close to the epidermis and often disrupts the dermal-epidermal junction.

The drugs used to treat lichen planus are oral and topical steroids, oral retinoids, immunosuppressant medications, hydroxychloroquine, tacrolimus, dapsone, UVB Narrow Band Phototherapy [6] and Aloe vera [7]. The world wide prevalence of LP is 0.73% [8].

Helicobacter pylori, a gram negative bacillus discovered in 1983, is the most popular pathogenic bacteria in the world. In addition to its roles in causing gastritis, gastric ulcer, duodenal ulcer and adenocarcinoma of stomach, it has been shown to cause nongastrointestinal disease [9]. Recently, there have been some studies on the possible role of HP infection in the pathogenesis of various extragastric diseases involving dermatologic conditions [10]. There is a relationship observed between peptic ulcer and HP infection with LP [11]. The measurement of *Helicobacter pylori* antigens in human stools (HpSA) has been proposed as a valuable, non-invasive, diagnostic tool with specificity and sensitivity of 94% [12].

The rationale of this study is to determine the frequency of *Helicobacter pylori* infection in the patients with lichen planus. Early detection and eradication of *Helicobacter pylori* infection can prevent such patients to develop complications as gastritis, gastric and duodenal ulcer.

MATERIALS AND METHODS

This cross sectional descriptive study was carried out in the department of dermatology at Liaquat University Hospital (a tertiary care 1500 bedded hospital) Hyderabad from February 2008 to July 2008. All patients above 12 years of age, of either gender, were known / already diagnosed and newly diagnosed cases of lichen planus (LP) were came through outdoor patient department (OPD), indoor patient and causality outdoor department (COD) were evaluated and enrolled in the study. The referred suspicious patients of lichen planus referred from different departments were also included in our study. The technique used for sample collection was non probability purposive. The data was collected through a pre-formed proforma / questionnaire. The detail history of all such patients was taken; complete clinical examination and routine investigation were performed. For relevant investigation all such patients were advised for *Helicobacter pylori* stool antigen (HpSA) for the detection of *Helicobacter pylori* infection. Informed consent was taken from every patient or from attendant of patients after full explanation of procedure regarding the study and all such maneuvers were under medical ethics. The data was collected, saved and analyzed in SPSS version 10.00. The frequency and percentage of *Helicobacter pylori* infection in lichen planus was calculated by detecting the number of positive and negative cases. The frequency and percentage was also calculated for gender distribution. The Mean and standard deviation was calculated for age. The exclusion criteria were 1. The patients who were already on *Helicobacter pylori* eradication therapy. 2. The non cooperative patients or who refused to give consequent or did not have interest to participate in the study were considered to be in exclusion criteria.

RESULTS

In this study one-hundred seventy three (105) patients were evaluated and diagnosed as lichen planus in the dermatology department, of which eighteen 18 (17%) were referral patients from different units for expert opinion. 72 (69%) patients had history of epigastric pain, 18 (17%) patients complaint of retrosternal pain and 07 patients had history of haemetemesis. The age with mean and standard deviation, gender, frequency and percentage of *Helicobacter pylori* infection in the patients with lichen planus and demographical distribution of patients was given in Table 1-3 and Fig. 1.

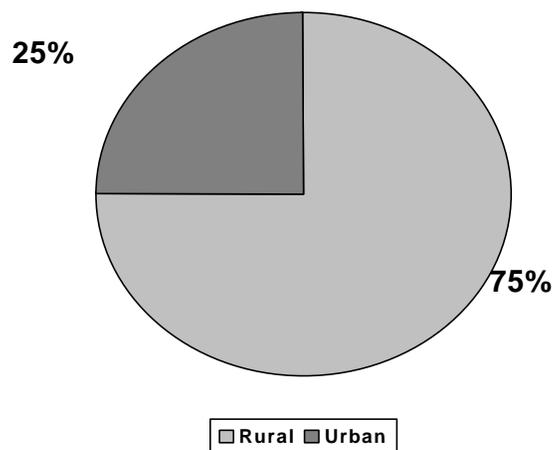


Fig. 1: Demographical Distribution of Helicobacter Pylori Infected Patients

Table 1: Results of Helicobacter Pylori Stool Antigen (Hpsa) Test in Patient with Lichen Planus

Lichen Planus	n = 105	%
Helicobacter pylori stool antigen (HpSA)		
Positive	81	77
Negative	24	23

Table 2: Age Distribution of Patients with Lichen Planus

Age	n = 105	%	Mean	Standard deviation (SD)
12 - 20	09	8.57		
21 - 30	18	17		
31 - 40	16	15		
41 - 50	46	44	42.8952	±13.3207
51 - 60	09	8.57		
61 - 70	05	4.76		
71 - 80	02	1.9		

Table 3: Gender Distribution of Patients Infected with Helicobacter Pylori Infection

Gender	n = 81
Male	33
Female	48

DISCUSSION

The etiology of lichen planus is unknown, but it is often connected with infections. LP has been associated with multiple disease processes and agents, such as viral and bacterial infections, autoimmune diseases and medications. In recent years peptic ulcer disease has also been closely linked with an infectious agent, Helicobacter pylori [11].

In present study, the Helicobacter pylori infection was detected in 81(77%) patients by Helicobacter pylori stool antigen (HpSA) in the subjects diagnosed as lichen planus, while another study detected approximately three-fold increased risk of peptic ulcer in patients with chronic/repeating lichen planus [11].

A high prevalence of HP in Iran leads us to assess the relationship between HP and LP [9]. Several other studies have also been reported the relationship between HP and LP. Dauden E, *et al* [13] conducted a study in which 61 patients with LP were subjected to urea breath test (UBT) revealed the positive Helicobacter pylori infection in 75.4% subjects. The study by Riggio [14] detected Helicobacter pylori infection in recurrent aphthous and oral lichen planus (OLP) by polymerase chain reaction (PCR) in 28 patients with aphthous showed the positive PCR for DNA of HP in 3 patients. Another related study was conducted on patients with oral mucosal ulcerative disorder for the screening of Helicobacter pylori, 03 cases of erosive lichen planus showed H. pylori infection. In our study the 22 patients had family history of peptic ulcer in which 13 were females and 09 were males.

In present study the Helicobacter pylori infection was predominant in females and this is similar in the study of Kaffes [15]. The Epidemiological studies have shown that the prevalence of Helicobacter pylori infection in a community and occupational health are closely related to lifestyle and socio-economic status. By occupation most of the patients in our study were industrial workers, mainly uneducated and did not have a proper drinking water facility that is the main predisposing factor to acquire bacterial infection in our setup. The

present study showed history of smoking in 27 patients whereas 09 patients had history of alcoholism and both are the predisposing factors for the Helicobacter pylori infection [16]. Such factors are associated with a decrease in efficacy of eradication therapy for H. pylori infection [17]. In our study 07 Helicobacter pylori infected patients had history of haemetemesis and out of which 04 were already on non steroidal antiinflammatory drugs (NSAID). However a study conducted at Turkey showed the half of the patients with lichen planus were positive for Helicobacter pylori infection in gastric and duodenal ulcer and most of them were symptom free [18].

In our series, the mean age and standard deviation of patients with lichen planus is 42.8952 ± 13.3207 where as a study conducted on lichen planus at Imam reza hospital, Iran that showed the mean age 43.3 ± 12.8 years in patients with lichen planus [19].

According to the comparative study of Moravvej *et al.* the active HP infection was found in 82.5% of the patients with LP and 61.25% of the patients in control group; the difference was statistically significant and displayed that HP could be at least the provoking factor behind LP, particularly as a chronic infecting agent maintaining LP [20]. However, the etiology of LP is probably multifactorial and the possibility still remains that HP may trigger LP in certain individuals. An autoimmune mechanism has been suggested in LP and HP has been associated with autoimmune processes in susceptible patients.

Therefore, patients with Lichen planus are more prone to acquire Helicobacter pylori infection, larger and further studies in different setups are needed yet to delineate and evaluate such predisposition, the appropriate and effective measures are the best tools that must be taken to protect the patients with lichen planus from different life threatening complications of Helicobacter pylori infection.

CONCLUSION

From our study we had concluded that patients with lichen planus are more prone to acquire the Helicobacter pylori infection. The patients with lichen planus must be evaluate and examine thoroughly. The routine and susceptible relevant investigation must be performed. Such screening and protocols can safe the patients from different life threatening complications. As far as Helicobacter pylori infection is concerned eradication therapy can prevent the patient to develop severe complications such as gastritis, gastric and duodenal ulcer and carcinoma.

REFERENCES

1. Singal, A. and D. Pandhi, 2004. Images in clinical practices. Indian Pediatrics, 41: 1062-63.
2. Murphy, R. and L. Edwards, 2008. Desquamative inflammatory vaginitis: what is it?. J. Reprod Med., 53(2): 124-8.
3. Balasubramaniam, P., M. Ogboli and C. Moss Lichen, 2008. Planus in children: review of 26 cases. Clin Exp Dermatol, 33(4): 457-9.
4. Belfiore, P., O. Di Fede, D. Cabibi, G. Campisi, G.S. Amaru and S. De Cantis, *et al.*, 2006. Prevalence of vulval lichen planus in a cohort of women with oral lichen planus: an interdisciplinary study. Br J. Dermatol, 155(5): 994-8.
5. Di Fede, O., P. Belfiore, D. Cabibi, S. De Cantis, E. Maresi A.R. Kerr, *et al.*, 2006. Unexpectedly high frequency of genital involvement in women with clinical and histological features of oral lichen planus. Acta Derm Venereol, 86(5): 433-8.
6. Pavlotsky, F., N. Nathansohn, G. Kriger, D. Shpiro and H. Trau, 2008. Ultraviolet-B treatment for cutaneous lichen planus: our experience with 50 patients. Photodermatol Photoimmunol Photomed, 24(2): 83-6.
7. Choonhakarn, C., P. Busaracome, B. Sripanidkulchai and P. Sarakarn, 2008. The efficacy of aloe vera gel in the treatment of oral lichen planus: a randomized controlled trial. Br. J. Dermatol, 158(3): 573-7.
8. Abdallat, S.A. and T.J. Maaita, 2007. Epidemiological and clinical features of Lichen Planus in Jordanian patients. Pak J. Med. Sci., 23(1): 92-4.
9. Malekzadeh, R., M. Mohamadnejad, F. Siavoshi and S. Massarrat, 2004. Treatment of Helicobacter pylori infection in Iran: Low efficacy of recommended western regimens. Arch Iranian Med., 7: 1-9.
10. Nilsson, H.O., A. Pietroiusti, M. Gabrielli, M.A. Zocco, G. Gasbarrini, A. Gasbarrini, 2005. Helicobacter pylori and extragastric diseases - other Helicobacter. Helicobacter, 10: 54-65.
11. Vainio, E., S. Huovinen, M. Liutu, J. Uksila and R. Leino, 2000. Peptic ulcer and Helicobacter pylori in patients with lichen planus. Acta Derm Venereol, 80: 427-429.
12. Faruqi, A.N., U. Majid, L. Ahmed, M. Khalil and M. Hassan, 2007. Helicobacter pylori stool antigen the diagnosis of gastric infection. J Coll Physician Surg Pak., 17(6): 316-9.

13. Daud4n, E., M.A. Vazquez-Carrasco, P.F. Pablo, J.M. Pajares and A. Garcia-Diez, 2000. Association of *Helicobacter pylori* infection with Psoriasis and Lichen planus: Prevalence and effect of eradication therapy. *Arch Dermatol*, 136: 1275-6.
14. Riggio, M.P., A. Lennon and D. Wray, 2000. Detection of *Helicobacter pylori* DNA in recurrent aphthous stomatitis tissue by PCR. *J. Oral Pathol. Med.*, 29: 507-13.
15. Kaffes, A., J. Cullen, H. Mitchell and PH Katelaris 2003. Effect of *Helicobacter Pylori* Infection and Low-Dose Aspirin Use on Iron Stores in the Elderly. *Gastroenterol Hepatol*, 18(9): 1024-1028.
16. Sillakivi, T., M. Peetsalu, M. Mikelsaar and A. Peetsalu, 2001. An attempt at *Helicobacter pylori* eradication with intravenous clarithromycin in perforated peptic ulcer patients. *Scand J. Gastroenterol*, 36: 1119-20.
17. Boixeda, D., D.C. Martin, C. Argila and R. Canton, 1994. Prevalence of *helicobacter pylori* in symptomatic adults. *Rev Esp Enferm Dig.*, 86(2): 569-76.
18. Sanli, H., A.H. Çetinkay, U. Türsen, M. Kaya, I. Kuzu and A. Gürler, 2002. Upper gastrointestinal findings in oral lichen planus. *The Turkish Journal of Gastroenterology*, 13(1): 31-4.
19. Ghaderi, R. and Z. Makhmalbaf, 2007. The Relationship between Lichen Planus and Hepatitis C in Birjand, Iran. *Shiraz E-Medical J.*, 8(2): 34-7.
20. Moravvej, H., H. Hoseini, B. Barikbin, R. Malekzadeh and G.M. Razavi, 2007. Association of *Helicobacter pylori* with lichen planus. *Indian J. Dermatol.*, 52: 138-40.