

A STUDY OF H₂ RECEPTOR ANTAGONISTS IN THE TREATMENT OF CHRONIC INTRACTABLE PHARYNGITIS

Pages with reference to book, From 217 To 219

Shabih H. Zaidi (Department of E.N.T. and Head and Neck Surgery, Jinnah Postgraduate Medical Centre, Karachi.)

ABSTRACT

In a controlled study of 75 patients suffering from chronic pharyngitis: 30 were treated with H₂-receptor antagonist, 30 with Hi-receptor antagonist, local decongestants and antiseptic gargles: and 15 treated with alum, salt or Dispirin gargles acted as controls. Best response was obtained with the first mode of treatment, raising the possibility of role of the H₂-receptors in causing chronic pharyngitis. The difference was highly significant (P <0.001) (JPMA 40 : 217, 1990).

INTRODUCTION

Chronic intractable pharyngitis is mainly caused by gastrooesophageal reflux and is most commonly seen in patients with gastric, hyperacidity, peptic ulcer or other digestive disorders¹. In a previous study, it has been established that such patients do not present with features of acute sore throat seen in infective pharyngitis, nor do they have allergic manifestations of the upper respiratory tract¹. The present study investigates the possible role of H₂ receptors in causing or promoting chronic intractable pharyngitis; and its management with H₂-receptors antagonists as against Hi-receptor antagonists and topical therapy as is customarily used in clinical practice.

PATIENTS AND METHODS

Between October 1989 - March 1990, a total of 75 patients were investigated for the best possible treatment of chronic intractable pharyngitis. After excluding the bacterial causes of sore throat, rhinosinusitis, smoking, pan chewing, supari sucking, niswar sucking and chronic tonsillitis, (as the major sufferers of chronic intractable pharyngitis and chronic tonsillitis are usually adults), these patients were divided into 3 groups. There were 42 females and 33 males, of 23-50 years age group (mean age: 37 yrs) belonging to multiple ethnic groups resident in Karachi for years. The 1st group was treated with Ranitidine (Zantac) one tablet twice daily for 4 weeks. The second group of 30 patients was treated with short term antihistamines in the day (Fendina, Teldane, Incidal) and long term antihistamines (Tavegyl - Avil) at night, also for the same duration. The 3rd group of 15 patients acted as controls. They were kept on simple gargles of Dispirin, salt or alum. In this group, there were 9 females and 6 males of 23-50 years age group, with no history of G.E. reflux. Each group was checked on weekly basis for four weeks, when the treatment was discontinued, and they were further observed for four weeks to note the progress.

RESULTS

Since fever, toxemia and constitutional disturbances are not the requisite features of chronic intractable pharyngitis, two signs and two symptoms, commonly encountered were taken as parameters. Sore throat and persistent discomfort or tightness in throat are the common complaints, and

hyperaemia of the posterior pharyngeal wall, and the pharyngeal bands as well as the appearance of granulomas on the posterior pharyngeal wall are the diagnostic signs in such cases. After four weeks of treatment group A showed 85% improvement in sore throat, 80% improvement in tightness and discomfort, 90% had improved state of pharyngeal hyperaemia and 92% showed either improvement or disappearance of granular appearance. In comparison group B, showed improvement of sore throat in 34%, tightness and pharyngeal discomfort in 30%, improved state of hyperaemia in 40% and, of pharyngeal granulomas in 10%. Group C showed improvement in these features upto 26%, 20%, 20% and 15% respectively for sore throat, tightness and discomfort, state of hyperaemia and the presence of granulomas. After six weeks, progress in group A was even better while in group B, there was slight increase in the number of patients with diminished soreness (38%), but otherwise the picture remained same. Group C, on the other hand showed deterioration or at least some fall in number of improved patients. After eight weeks, group A continued to show progress, and only a few patients, i.e. less than 5%, remained as prior to the treatment. Group B, showed some progress in improvement of symptoms, but not really in the clinical signs, of hyperaemia and granulomas. And group C, showed diminution in the number of improved patients. The inference to be made therefore was that the patients treated with H₂ receptor antagonists did significantly better ($P < 0.001$) than those treated with H₁ receptor blockers, or topical decongestant therapy alone as shown in the accompanying figure.

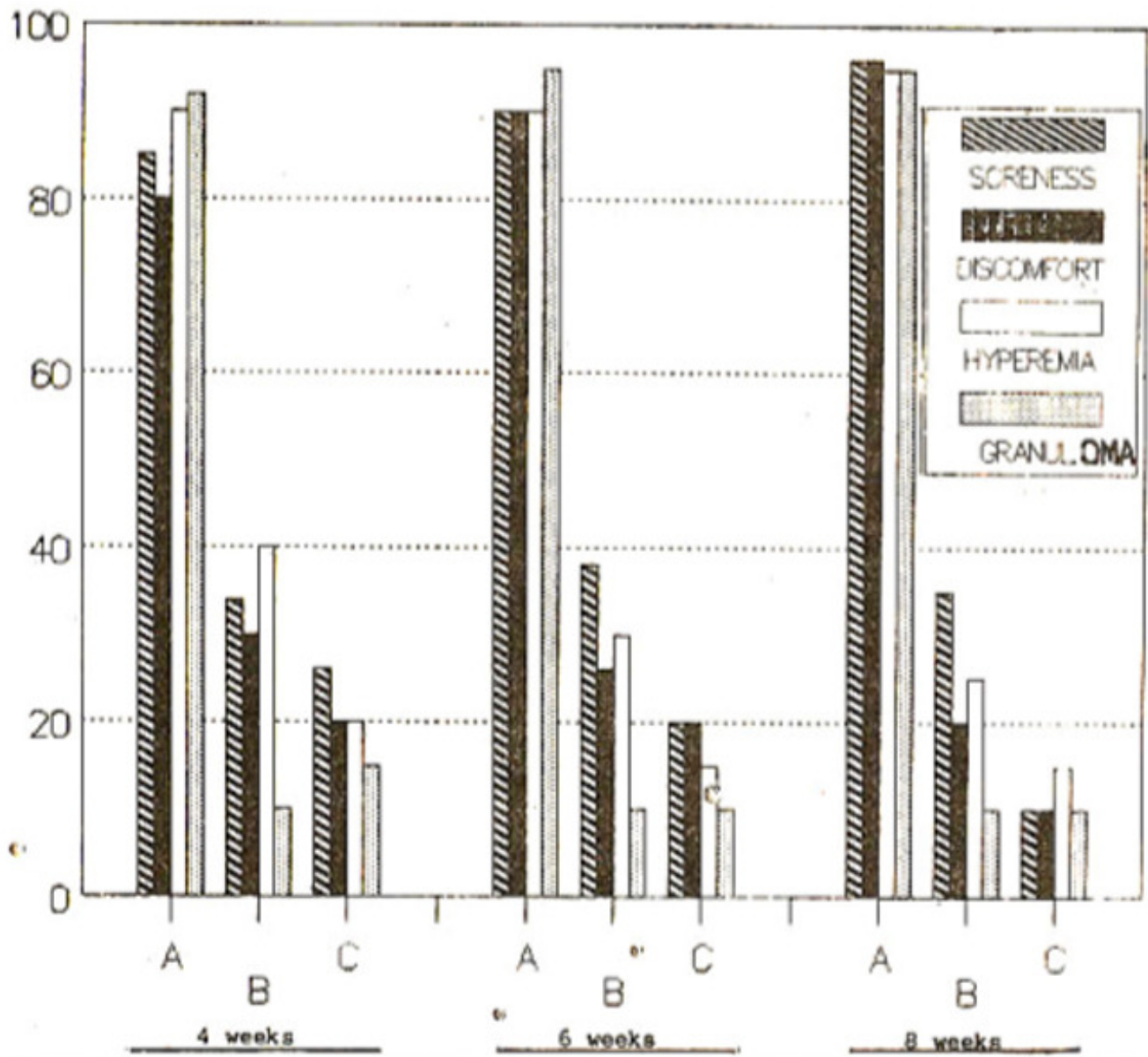


Figure. Satisfactory response at 4,6,8 weeks with different regimens.

DISCUSSION

Chronic pharyngitis may be of infective, allergic or nonspecific origin. Innumerable factors have been suggested in causing chronic nonspecific pharyngitis, such as atmospheric pollution, consumption of contaminated food, and use of aerated drinks, sensitivity to change in temperature etc. Amongst the recent developments is the role of gastro-oesophageal reflux in causing or promoting it¹. The alteration of pH in pharyngeal mucosa, defective mucociliary flow or the presence of hiatus hernia have been thoroughly researched by many workers³⁻⁷. Amongst the possible explanation of GER causing acid pharyngitis numerous factors have been incriminated such as alteration of lower oesophageal sphincter⁸ impaired oesophageal motor function with delayed clearing. Upper oesophageal sphincter dysfunction,

impaired gastric emptying, presence of hiatal hernias, alteration of duodenal function, gastric distention, increased production of gastric acid and pepsin², and postural aggravation as in the position of 'ruku' and sajda after heavy meals — are all possible causes of GER¹, along with drugs like salicylates¹. The present study raises yet another possibility, viz., the presence of H₂-receptors in the pharyngeal mucosa. The improvement in symptoms of soreness, dryness and irritability in the pharynx, and obvious clinical improvement in hyperaemia, and granulomatosis of the posterior pharyngeal wall, with H₂-receptor antagonists -zantac, within two weeks of therapy and further improvement in the next 4-6 weeks, appears to be a cognizable finding (P < 0.001). The pharyngeal granuloma, comprises of freshly developed capillaries, chronic lymphocytes, histiocytes embedded in the columnar, pharyngeal epithelium. The response to H₁ receptor antagonists over the same period was not as noticeable as with H₂-receptor antagonists over the same period (P < 0.05). In the third group, where only topical treatment was given, showed little or negligible improvement in the features described above (P < 0.05). The age and sex were uniformly distributed in all three groups. Two obvious queries arise out of these observations, namely: was this improvement in signs and symptoms of pharyngitis brought about by improvement in GE reflux and diminished gastric acidity or was it due to direct effect of H₂-receptor blockers on H₂-receptors spread across the pharyngeal mucosa? G.I. endoscopy or Barium studies were not carried out in this study. Circumstantial evidence points out towards the latter as these receptors have been identified in the bronchial system⁹ which has a common embryological origin with the pharynx, indeed the entire aerodigestive system¹⁰. However further investigation is required to confirm the presence of H₂-receptors in the pharyngeal mucosa. And if so, and it appears to be a strong possibility, then the whole philosophy of management of chronic pharyngitis would need to be modified so as to counteract the H₂-receptors - a radical diversion indeed from conventional practice of treating it with H₁-receptor antagonists or with local medication alone.

REFERENCES

1. Zaidi, S.H. and Jafri, R. G.E. reflux, as a cause of chronic intractable pharyngitis. Pakistan J. ORL., 1989; 6: 20.
2. Gaynor, E.B. Gastroesophageal reflux as an etiologic factor in laryngeal complications of intubation. Laryngoscope, 1988; 98: 972.
3. Olson, N.R. The problem of G.E. reflux. Otolaryngol Clin. North Am., 1986; 19: 119.
4. Goldberg, M., Noyek, A. and Pritzker, K.P.H. Laryngeal granulomas secondary to GE. reflux. J. Otolaryngol., 1978; 7: 196.
5. Wynne, J.W., Ramphal, R. and Hood, C.I. Tracheal mucosal damage after aspiration. Am. Rev. Respir. Dis., 1981; 124: 728.
6. Ward, P.H. and Berci, G. Observations on the pathogenesis of chronic non-specific pharyngitis and laryngitis. Laryngoscope, 1982; 92:1377.
7. Pearson, J.E.G. and Wilson, R.S.E. Diffuse pulmonary fibrosis and hiatus hernia. Thorax, 1971; 26: 300.
8. Johnson, L.F. New concepts and methods in the study and treatment of gastroesophageal reflux disease. Med. Clin. North Am., 1981; 65: 1195.
9. Hamilton, N.J. and Harrison, R.J. Anatomy of the mouth, pharynx and oesophagus, in Scott-Browns diseases of ear, nose and throat. 3rd ed. London, Butterworths, 1971, Vol. 1, P. 189.