GENETIC FACTORS IN PEPTIC ULCER

Pages with reference to book, From 280 To 281

S.M Abbas Rizvi (PMRC Research Centre, Jinnah Postgraduate Medical Centre, Karachi.)

Chronic peptic ulcer occurs in the portions of the alimentary tract which come in contact with the gastric juice like lower portion of the eosophagus, stomach, duodenum, jejunum and in the Meckel's diverticulum containing ectopic gastric mucosa. The frequency and type (GU/DU) with which peptic ulcer occurs, makes the aetiology and management of this ulcer difficult and problematic. Although lot of work has been done on the aetiology of peptic ulcer, yet we know little about the various causes of the development and persistence of ulcer; why ulcers heal or relapse? or, why and how patients with ulcers may or may not have pain, bleeding or perforation? Various factors commonly studied in the aetiology and pathogenesis of peptic ulcer include epidemiological, etiological, genetic and psychologic factors. Much information about the aetiology of the ulcer disease can potentially be obtained from the analysis of occurrence of ulcers in various populations within the same and in different geographic areas of the world[1]. Etiological factors like cigarette smoking, alcohol consumption, diet and drug ingestion have been studied. [2,3] Studies on psychological factors have shown no experimental confirmation that such factors can produce ulcers, even in animals. [4] These studies can indicate the etiology and, accordingly, pathogenesis of the peptic ulcer but the data is at best crude and not as reliable as rigorous scientific and laboratory based experiments. Work done on the genetic factors in peptic ulcer has shown that it occurs 2 to 3 times more frequently in the first degree relatives of the individuals with peptic ulcer as in the relatives of the controls or in general population. [5,6] For first degree relatives of duodenal ulcer patients and controls, the endoscopy showed the presence of ulceration in 13% and 3.9%, respectively. [5] In another study, the frequency of a positive family history in the controls and in the families of positive duodenal ulcer patients was found to be 13.0% and 52.5%, respectively. In studies of twins, one study showed that monozygotic twins showed concordance in half the cases and 3 times more frequently than dizygotic pairs, leading to the conclusion that hereditary factors are involved in ulcerogenesis[7]. To establish the role of the genetic factors in the peptic ulcer, different markers have been studied. These include the studies on blood groups, secretor status, alkaline phosphatase and pepsinogen[8] in ulcer patients. The results of studies on the genetic markers indicate the importance of genetic factors in the aetiology of the peptic ulcer. However, the data from one study cannot set a standard and separate studies in various areas are necessary, particularly while working for a disease as universal aspeptic ulcer. In a study on pepsinogen values in ulcer patients in Pakistan; approximately 14% of the patients had elevated pepsinogen values (Shahid and Zuberi - unpublished data). In a similar study in India[8], 83% of the patients had hyperpepsinogenaemia. Considering the general similarities of the diet and natural habits between the people of Pakistan and India, this difference is remarkable. This is a comparison of just one study on one genetic marker. The studies on the prevalence of HLA antigens also give an idea of the importance of genetic factors in peptic ulcer. While studying the prevalence of HLA antigens in patients with duodenal ulceroit was found that the frequency of HLA-B5[9], , HLA-B 12[10] and HLA-B35[11] were significantly increased. The relative risk for developing a duodenal ulcer for persons with HLA -B12 was stated to be 2.1:1. However, other reports did not note any significant association between the frequency of any antigen and gastric or duodenal ulcer[12,13]. It is therefore, necessary that a detailed study on the genetic markers and peptic ulcer for the establishment of the role of genetic factors is required. The high prevalence of the disease in our country warrants an elaborate study plan on this important aspect of the disease.

REFERENCES

1. Donaldson, R.M. Factors complicating observed associations between peptic ulcers and other diseases. Gastroenterology, 1975; 68: 1608.
2. Wormsley, K. G. Smoking and duodenal ulcer. Gastroenterology, 1978; 75: 39.
3. Friedman, G. D., Siegelaub, A.B. and Seltzer, C. C. Cigarettes, alcohol, Coffee and peptic ulcer. N. Engi. J. Med., 1974; 290 : 469.
4. Feldman, EJ. and Sabovich, K.A. Stress and peptic ulcer disease. Gastroenterology, 1980; 78 (Pt.1): 1087.
5. Lorpita, SJ.M., Pikkavaiven, P., Vuoristo, M. and Ibaruake, T. Endoscopic and clinical findings in first degree relatives of duodenal ulcer patients and control subject. Scand. J. Gastroenterol., 1982; 17 : 503.
6. Doll, R. and Buch, J. Hereditary factors in peptic ulcer. Ann. Eugenics, 1950; 15: 135.
7. Eberhard, G. Peptic ulcer in twins. A study in personality, heredity and environment. Acta Psychiatr. Scand., 1968; 44 (Suppl. 205):1.
8. Habibullah, C.M., All, M.M., lshaq, M., Prasad, R., Pratap, B. and Saleem, Y. Study of duodenal ulcer disease in 100 families using total serum pepsinogen as genctic marker. Gut, 1984; 25: 1380.
9. Hotter, J.I., Limoin, D.L., Gursky, J.M., Tevasaki, P. and Sturdevant, R.A. HLA-B5 associated with duodenal ulcer. Gastroenterology, 1977; 73: 438.
10. Elis, A. and Woodrow, J.C. ULA and duodenal ulcer. Gut, 1979; 9:760.
11. Cough, MJ., Rajah, S.M. and Giles, G.R. HLA antigens in relation. ship to duodenal ulceration, gastric acid secretion and the clinical result followingg vagotomy. Br. J. Surg., 1982; 69: 105.
12. O'Brien, B.D., Thompson, A.B.R. and Dossetor, J.B. HLA and peptic ulcer. Dig. Dis. Sci., 1979; 24 :314.
13. Kaug, J.Y., Doran, T., Grampton, R., Mclenehan, W. and Piper, D.W. HLA antigen and peptic ulcer disease. Digestion, 1983; 26:99.