SERUM HAPTOGLOBIN PHENOTYPES AND DUODENAL ULCER

A. TSOUKANTAS, A. ARCHIMANDRITIS, G. THEODOROPOULOS, K. KATSAVOCHRISTOS, K. MELISSINOS

Department of Pathologic Physiology, Medical School, University of Athens, Greece

Serum haptoglobin phenotypes were studied in 100 Greek patients suffering from duodenal ulcer by vertical starch gel electrophoresis. A sample of 2026 healthy subjects served as control. No statistically significant differences were found in Hp phenotypes and gene frequencies between patients and healthy controls.

INTRODUCTION

Although the ultimate cause of duodenal ulcer remains unknown, the race, sex and familial predisposition are thought to play an important role in its pathogenesis. Besides, it is well-known that the prevalence of duodenal ulcer is greater in blood group 0 non-secretors as compared to secretors of other blood groups (Clarke et al. 1959).

Many polymorphic systems have appeared in medical genetics recently. The haptoglobins (Hp) belong to the α_2-glycoproteins and are inherited through at least two autosomal codominant alleles, HpI and Hp2.

Three Hp phenotypes, HpI-I, HpI-2, and Hp2-2, have been discovered by starch-gel electrophoresis. The distribution of Hp genes and phenotypes varies considerably all over the world (Giblett 1969). On the other hand, a relationship seems to be possible between the HpI gene and some diseases, i.e., leukemia (Peacock 1966) and liver cirrhosis (Hirayama et al. 1975).

In the international literature we have found only one record of the distribution of Hp phenotypes among patients of peptic ulcer (Wendt et al. 1968). Nevertheless, the results concerned both duodenal and gastric ulcer.

This work was undertaken to study the Hp phenotypes among patients suffering from duodenal ulcer.

MATERIAL AND METHODS

Sera from 100 hospitalized patients of both sexes suffering from duodenal ulcer and originating from various areas of Greece were studied by vertical starch-gel electrophoresis (Smithies 1959). A sample of 2026 healthy Greeks, examined previously in the same laboratory under the same conditions, served as controls (Angelopoulos et al. 1966). Diagnosis was made by the history and x-ray examination in all patients and was established surgically in 67. Patients also suffering from other diseases were excluded from this study. Statistical analysis was performed by conventional methods (fourfold tables with Yates' correction, Brandt and Snedecor formula).

RESULTS AND DISCUSSION

As shown in the Table, the Hp phenotype and gene frequencies are those expected for Europeans (Giblett 1969). No statistically significant differences were found between our patients and healthy controls, and our results are in close agreement with those reported by Wendt et al. (1968) for peptic ulcer and suggest that there is no relationship between the Hp system and duodenal ulcer.

Table. *Hp phenotypes and gene frequencies in patients with duodenal ulcer and in controls*

Material	N	Hp phenotypes 1-1	2-1	2-1	Gene frequencies Hp^1	Hp^2
Patients	100	14	43	43	0.355	0.645
Controls	2026	224	927	875	0.339	0.641

χ^2 between genes = 0.144; $p > 0.7$.
χ^2 among phenotypes = 0.909; $p > 0.6$.

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Dr. G. Theodoropoulos, 2 Dryadon Street, Chalandrion, Athens, Greece.