

Introduction

Understanding the molecular basis of gastric cancer is essential to develop more effective methods of primary prevention and secondary prevention (early diagnosis and treatment). Although the molecular mechanisms in gastric carcinogenesis have not been completely delineated, some important advances in the molecular biology of gastric cancer have been made⁽¹⁾.

Several abnormalities in oncogenes, tumor suppressor genes, and growth factor expression have been identified in gastric cancer. P53 is a tumor suppressor gene which is usually mutated up to 67.9% in gastric carcinoma⁽¹⁾; these mutations are usually missense point mutations leading to genetic instability and uncontrolled cell proliferation⁽²⁾. These mutations impair P53 anti-cancer gene inducing effects, so restoring its function would be a major step in curing many cancers, including gastric cancer⁽³⁾, especially the ability of P53 to control apoptosis in response to DNA damage which has important practical therapeutic implications to enhance the effect of radiation and chemotherapy, or even evaluating the effect of adenovirus mediated re-introduction of wild type P53 as a potential clinical utility in gene therapy of gastric cancer⁽⁴⁾. Thus, it is accepted that P53 plays a fundamental role in tumorigenesis and hence is an obvious choice for therapeutic exploitation. However, conflicting evidence and insufficient knowledge about the P53 pathways in detail and the fact that other mechanisms exist to modulate P53 activity leave this useful tool a hope for the future as regards use in the clinic⁽⁵⁾.

So this study aims to assess the immunohistochemical expression of p53 protein in gastric carcinoma and to study the correlation between p53 protein expression and different

clinicopathological variables like: age, gender, site, gross pattern, histological type, grade, and stage of the tumor in gastric carcinoma cases.

Patients, materials and methods

From October 2006-May 2007, forty formalin fixed paraffin embedded gastric carcinoma tissue blocks (partial or total gastrectomy specimens) from the archived materials of the Department of Pathology of Baghdad Teaching Hospital and the Center of Gastrointestinal and Hepatic Diseases, and other private laboratories were included in this study.

Clinicopathological parameters as the age and gender, site, gross appearance, histological type, grade and stage of tumor were obtained from histopathological reports.

A four micrometer thick tissue sections were obtained from representative area, one section was stained with Hematoxylin and eosin (H&E) and then reviewed, while two sections were stained immunohistochemically for p53. The histologic type was classified according to Lauren classification 1965.

The positive control tissue used in the present study was a specimen from poorly differentiated ductal breast carcinoma tissue, which was known to be positive for monoclonal anti p53 protein.

Untreated sections with primary antibody (by omission of the primary antibody) were considered as technical negative control, while normal gastric tissue sections were considered as *tissue negative control* and were used for each set of slides. These tissues should show absence of specific staining.

All the slides were examined by light microscope; a random selection of the fields was used for analysis of all cases. Positive p53 results give