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The Pathogenesis of Gastric Carcinoma

Elroy Patrick Weledji
Senior lecturer in Anatomy and Clinical Surgery, University of Buea, S. W. Region, Cameroon

Abstract
Gastric cancer is one of the leading causes of cancer mortality in the world. Gastric adenocarcinomas account for more than 95% of gastric tumours, and these epithelial tumours result from the accumulation of multiple genetic defects which leads to uncontrolled growth. The most plausible pathway for gastric carcinoma indicates that the underlying mechanisms may be different for the diffuse and intestinal types of tumour. The distinct subtypes - proximal, diffuse and distal gastric cancer that are different from a histological and epidemiological standpoint, can also distinguished by gene expression data. The disease classification of the epithelial tumours may lead to different treatment paradigms for individual gastric cancer subtypes. The changes present can be classified as consisting of abnormalities in DNA content, the karyotype (including allele loss), oncogene and tumour suppressant gene expression (or deletion), cell cycle regulation and DNA repair genes. This article reviewed the biological/molecular differences of the gastric carcinoma subtypes, the risk factors and precursors of gastric carcinoma and the implications on early diagnosis and response to adjuvant treatment. Despite the heterogeneity of multiple somatic alterations in the neoplastic lesion, the implication of a molecular classification is its exploitation to identify prognostic and predictive biomarkers and to identify targets for therapy.

Key words: gastric carcinoma, pathogenesis, molecular, epidemiology