Contribution of dna hypomethylation in gastric carcinogenesis

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Abstract

Introduction

Gastric cancer (gc) is among the most common malignancies worldwide with high mortality rate. to design more effective diagnostic and therapeutic strategies, recognition of novel biomarkers that are epigenetically regulated during gastric carcinogenesis has been center of attention in recent years.

Methods

To review current knowledge regarding epigenetic modifications in gastric cancer, published papers including key words gastric carcinoma, dna hypomethylation and epigenetic modification, were extracted from pubmed, scopus, web of science, and google scholar.

Results

Local hypermethylation occurs in cpg islands and resulted in aberrant silencing of genes such as tumor-suppressor, cell cycle regulator and dna-repair genes. nevertheless, global dna hypomethylation, particularly in repetitive sequences, is generally associated with wide range of events including chromosome instability, loss of imprinting, repression of transposable elements and activation of oncogenes. in gastric carcinogenesis, dna hypomethylation, which has been discovered in a number of genes, was associated with clinicopathological features of the disease. for instance, demethylation of synuclein-Î³ was common in patients with lymph node metastasis, while hypomethylation of cyclin d2 was frequently observed in advanced stages of gc. in addition, hypomethylation of mage (melanoma antigen gene) and line-1 (long interspersed element-1) was associated with poor prognosis of gc. hypomethylation of genes could also be induced by h. pylori during gastric carcinogenesis, as reported for alu and satÎ±.

Conclusion

Overall, epigenetic modification in the form of hypomethylation could be used as a potential screening marker for early detection of gc, and a monitor for evaluating responses to therapeutic strategies.

Keywords

Gastric carcinoma, dna hypomethylation, epigenetic modification