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Editorial

Gastric Lymphoma and Epstein-Barr Virus

Epstein-Barr virus (EBV) is the major biological cofactor contributing to a number of human cancers including B-cell neoplasms (Burkitt's lymphoma, Hodgkin's lymphoma (HL and immunoblastic lymphomas, certain forms of T-cell lymphoma, and some epithelial tumours (nasopharyngeal carcinomas and gastric carcinomas). Recent studies have revealed the association of EBV with about 10% of gastric carcinoma cases worldwide. EBV DNA in cancer biopsies shows monoclonality, indicating that carcinoma arises from a single EBV-infected cell.[1]

Primary malignant gastric lymphomas account for less than 15% of gastric malignancies. Most gastric lymphomas are B-cell lineage NHL. They usually arise from mucosal associated lymphoid tissue (MALT). Other types of gastric lymphomas include diffuse large B-cell lymphomas, mantle cell lymphomas and T cell lymphomas.[2]

Seventy to 100% of MALT lymphoma cases are associated with *Helicobacter pylori*, a bacterium proven to be the cause of gastritis and peptic ulcer and the progression to gastric carcinoma. Lower *H. pylori* infection rates have been reported in gastric lymphoma in Asian populations (50%-60%).[3]

Gastric lymphoma association with EBV has not been extensively studied. The pilot study by Sood *et al* published in this issue of Journal reports EBV detection in neoplastic cells in 36% of 11 cases,[4] higher than reports from Hong Kong, Korea and China (2% to 8% in series of 53, 33 and 49 cases respectively).[3,5] However, the small sample size does not allow any meaningful conclusion.

Detection of EBV in tumour cells by immunohistochemistry or in situ hybridization is no proof of its implication in the pathogenesis. EBV might either play a role in the development of gastric lymphoma, particularly in *H. pylori* negative cases, or be present in neoplastic cells as a secondary phenomenon. Demonstration of EBV clonality in EBV-associated gastric lymphomas would be able to give a definitive answer.

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