Presence of Epstein–Barr virus in gastric adenocarcinoma in Indian patients

Dear Editor,

Rymbai et al. reported in the recent issue of this journal, the presence of Epstein–Barr virus (EBV) in a small proportion of gastric adenocarcinomas (GC) in patients presenting to a Tertiary Care Centre in South India.[1] EBV is a lymphotropic herpes virus implicated in the aetiology of several human malignancies including Burkitt’s lymphoma, nasopharyngeal carcinoma and GC. It is estimated that the global burden of deaths from EBV-associated malignancies is approximately 150,000.[2] The virus is transmitted via the oral route and infection, often takes place early in childhood, particularly in developing countries. By adulthood, more than 90% of the individuals worldwide are asymptomatic carriers, with the virus establishing life-long latency in memory B-cells.[3] Thus, the mere detection of EBV DNA/RNA in clinical samples using polymerase chain reaction (PCR), as reported by Rymbai et al., cannot be

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simply interpreted to indicate that the virus is involved in the disease process. In this context, we have several points which we would like to raise regarding the study of Rymbai et al.

It is well-established that EBV is aetiologically associated with the pathogenesis of approximately 10% of GC. In these cases, the virus can be detected in the malignant cells of GC by Epstein–Barr encoded RNA (EBER) in situ hybridisation or Epstein–Barr nuclear antigen 1 (EBNA-1) immunohistochemistry. Rymbai et al. reported an association between EBV and GC based on the detection of EBNA-1 mRNA using real-time PCR (RT-PCR). We find this to be an insufficient approach to conclude a virus-disease association, bearing in mind that EBV is ubiquitous in the healthy population. Furthermore, the detection of EBNA-1 mRNA does not necessarily mean that the corresponding protein is expressed at the translational level, and it certainly does not indicate which cells within the tumour are expressing it. The authors reported 6% (6/100) of their cases to be positive for EBNA-1 mRNA. In 2 of the 6 positive cases, EBNA-1 mRNA was also detected in non-tumour tissues. It is impossible to know if the cellular source of EBNA-1 mRNA in these 2 cases was malignant cells which had metastasised from the main tumour or non-malignant tumour infiltrating lymphocytes which happened to be EBV positive. This could have easily been resolved by performing EBNA-1 immunohistochemistry instead of RT-PCR. Alternatively, the authors could have examined their cases for the presence of EBV using EBER in situ hybridisation. This highly sensitive and specific method is widely used as the gold standard for the detection of EBV in histological tissues. This approach would have given a much better indication of the true proportion of EBV-associated GC cases in the Indian population.

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Conflicts of interest

There are no conflicts of interest.

References


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