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Detection of Epstein Barr Virus in gastric lesions

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Different lines of evidence support an association between Epstein-Barr virus (EBV) and gastric cancer (GC). The main understood risk factor to develop GC is infection by Helicobacter pylori (H. pylori), which triggers a local inflammatory response critical for progression from gastritis to GC. The role of EBV in early inflammatory gastric lesions has been poorly studied. A recent study proposed a cutoff value of 2000 EBV particles to identify patients with increased chances of infection of the gastric epithelium, which may be favoring the inflammatory process. To better understand the role of EBV in cancer progression, we analyzed a total of 548 gastric samples; 98 samples of GC, 162 control samples of non-tumor gastric tissue derived from GC patients, 17 biopsies of gastric metaplasia, eight atrophic gastritis (AG) samples and 168 biopsies from patients with non-atrophic gastritis (NAG). In addition 76 samples of pediatric patients diagnosed with NAG were included and 19 control samples of patients undergoing bypass surgery. A first-round PCR was used for EBV detection in tumor and non-tumor controls and a more sensitive nested PCR for gastritis samples; both PCRs with lower detection limits above the proposed cutoff value. With this strategy 9.18% of GC, 1.23% of non-tumor controls, 17.64% of gastric metaplasia, 0% of AG, 5.9% of NAG in adult patients, 11.84% of NAG in pediatric patients and 0% of the bypass samples were found positive. An EBER1 ISH showed EBV infection of epithelial cells in GC and in a third of NAG samples, while in the other NAGs infection was restricted to mononuclear cell infiltrate. EBV positive GCs were enriched in lace and cribriform patterns, while these rare patterns were not observed in EBV negative samples. Our results support a role for EBV in GC and early precursor inflammatory lesions.

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