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Pathology of *Helicobacter pylori*-associated lymphomas

AXEL GREINER
HANS K. MÜLLER-HERMELINK
Institute of Pathology,
University of Würzburg, Germany

The stomach is the commonest site of MALT-type lymphoma development, although the gastric mucosa is primarily devoid of lymphoid tissue. *Helicobacter pylori* leads to an active chronic gastritis and to the recruitment to of both B cells and T cells to mucosa forming mucosa-associated lymphoid tissue (MALT).

In the recent decade evidence accumulated that *Helicobacter pylori* infection is linked to the pathogenesis of gastric MALT-type lymphoma. Antigen- with T-cell stimulation may have important impact on the initial phase of tumor development, while *Helicobacter pylori* eradication can result in tumor regression in most cases.

The histological features of MALT-type lymphomas closely resemble those of the peyer's patch. Because of the difficulty in distinguishing between acquired MALT and MALT-type lymphoma, particularly in small biopsies, molecular clonality analyses may

be combined with histology for diagnosis and follow-up.

Gastric MALT-type lymphomas disseminate both locally and systemically to other mucosal sites and the spleen more frequently than was originally believed. Cytologically they are low-grade neoplasm and correspond to the marginal zone but tend to progress in a third of cases into diffuse large B-cell lymphomas. Although MALT-type lymphomas of other sites share the same morphologic and immunophenotypic features they may differ genetically.

In particular, it has now been shown that gastric MALT-type lymphomas resistant to eradication carry the t(11;18). Therefore it is likely that the tumor cells are not reliant to the signals from the microenvironment and the molecular changes override the normal signalling pathways that may have important implications for future diagnostic and therapeutic management.