



NTP Nonneoplastic Lesion Atlas

Stomach, Glandular Stomach – Inflammation

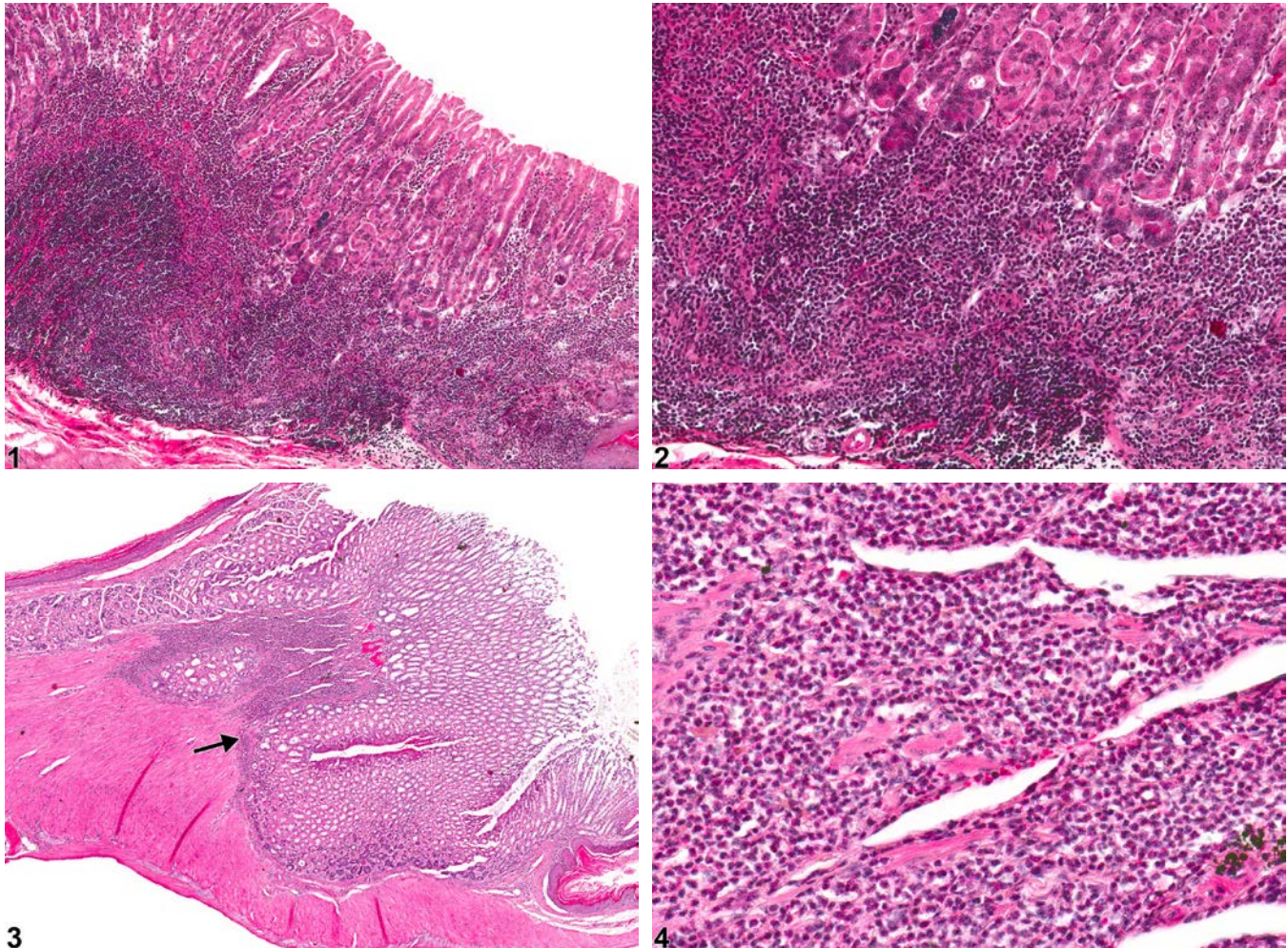


Figure Legend: **Figure 1** Stomach, Glandular stomach - Inflammation, Chronic in a male B6C3F1 mouse from a chronic study. Lymphocytes (primarily) are present within the submucosa and lamina propria. **Figure 2** Stomach, Glandular stomach - Inflammation, Chronic in a male B6C3F1 mouse from a chronic study (higher magnification of Figure 1). Lymphocytes (primarily) are present within the submucosa and lamina propria. **Figure 3** Stomach, Glandular stomach - Inflammation, Eosinophilic in a male B6C3F1 mouse from a chronic study. Eosinophils (primarily) are present within the submucosa and lamina propria (arrow). **Figure 4** Stomach, Glandular stomach - Inflammation, Eosinophilic in a male B6C3F1 mouse from a chronic study (higher magnification of Figure 1). Eosinophils (primarily) are present within the submucosa and lamina propria.



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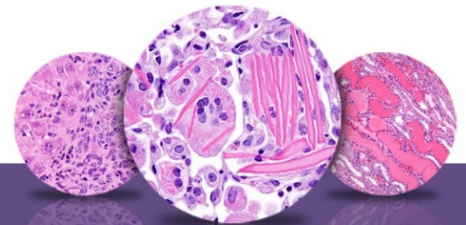
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Comment: Inflammation (Figure 1, Figure 2, Figure 3 and Figure 4) can occur in the glandular stomach as in elsewhere in the gastrointestinal tract. Lesions can be spontaneous (idiopathic), related to gavage procedure, or due to direct/indirect chemical toxicity.

Inflammation can be diagnosed as acute, suppurative, chronic, chronic active, or granulomatous. In *acute inflammation*, the predominant infiltrating cell is the neutrophil, though fewer macrophages and lymphocytes may also be present. There may also be evidence of edema or hyperemia. The neutrophil is also the predominant infiltrating cell type in *suppurative inflammation*, but the neutrophils are aggregated, and many of them are degenerate (suppurative exudate). The exudate may also contain cell debris, both from the resident cell populations and from infiltrating leukocytes; proteinaceous fluid containing fibrin; fewer macrophages; occasional lymphocytes or plasma cells; and, possibly, an infectious agent. Grossly, these lesions would be characterized by the presence of pus. In the tissue surrounding the exudate, there may be fibroblasts, fibrous connective tissue, and mixed inflammatory cells, depending on the chronicity of the lesion. Lymphocytes predominate in *chronic inflammation*. Lymphocytes also predominate in *chronic active inflammation*, but there are also a significant number of neutrophils. Both lesions may contain macrophages. *Granulomatous inflammation* is another form of chronic inflammation, but this diagnosis requires the presence of a significant number of aggregated, large, activated macrophages, epithelioid macrophages, or multinucleated giant cells.

Eosinophilic inflammation (Figure 3 and Figure 4) is not one of the five main types of inflammation ordinarily diagnosed by the NTP but may be diagnosed if the inflammatory cells are primarily eosinophils. The pathologist may deviate from the 5 main inflammation diagnoses as long as the reason for the deviation is discussed in the narrative.

Recommendation: Whenever present, inflammation should be diagnosed and given a severity grade and a modifier that indicates the duration or type of inflammation (e.g., acute, suppurative, chronic, chronic active, or granulomatous). The severity grade depends on the extent of area of glandular stomach affected, the density of the cellular infiltrate, and the extent of changes in the tissue (e.g., edema, hemorrhage, degeneration, necrosis). Lesions consistent with an abscess are diagnosed as suppurative inflammation. Occasionally the pathologist may find it necessary to use different modifiers than the five stated above. The reasons for the use of other modifiers, such as “eosinophilic,” should be



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explained in the narrative. Inflammation in areas of erosion or ulcer is not generally recorded unless the inflammation is a significant component of the lesion.

References:

Leininger JR, Jokinen MP, Dangler CA, Whiteley LO. 1999. Oral cavity, esophagus, and stomach. In: Pathology of the Mouse (Maronpot RR, ed). Cache River Press, St Louis, MO, 29-48.
Abstract: <http://www.cacheriverpress.com/books/pathmouse.htm>

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