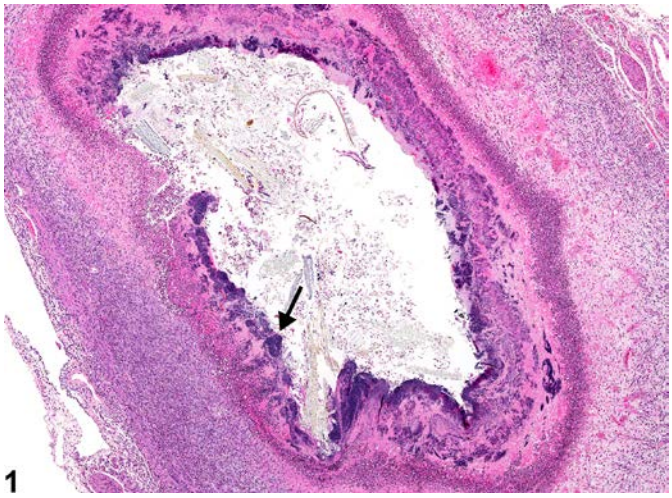
National Toxicology Program
U.S. Department of Health and Human Services

NTP Nonneoplastic Lesion Atlas

Esophagus – Inflammation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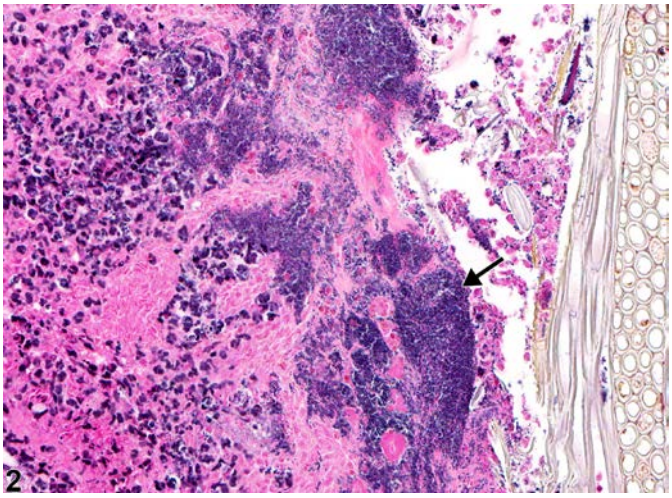
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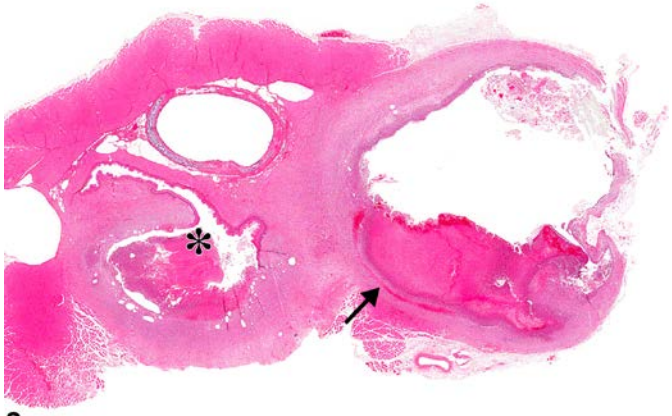
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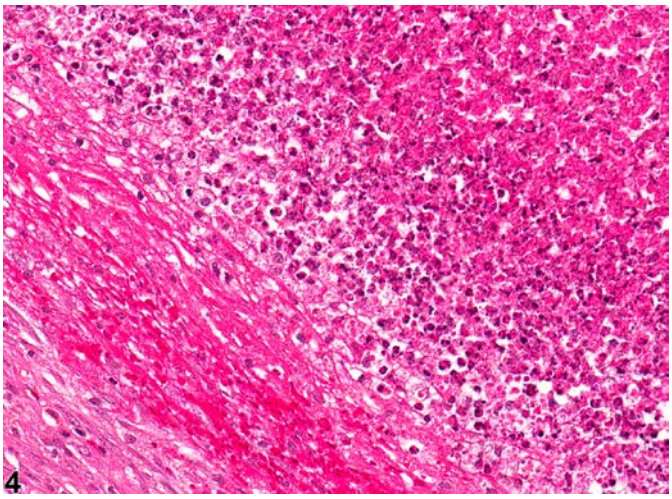
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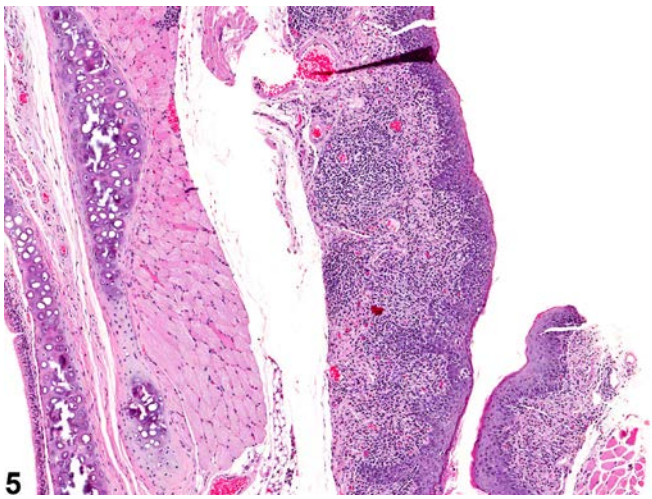
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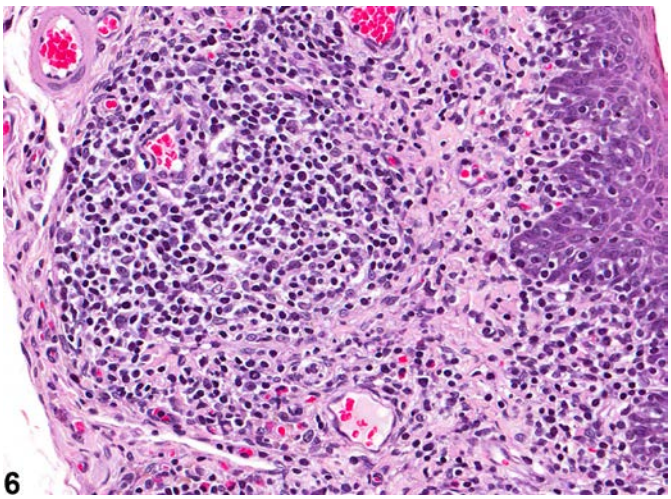
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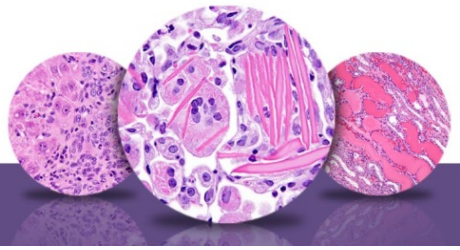
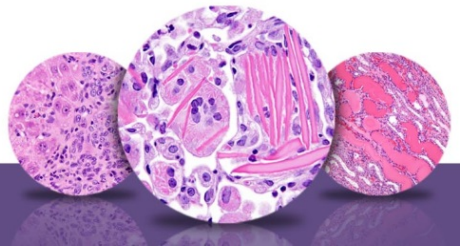


Esophagus – Inflammation

Figure Legend: Figure 1 Esophagus - Inflammation, Suppurative in a female F344/N rat from a subchronic study. Note the bacterial colonies (arrow). **Figure 2** Esophagus - Inflammation, Suppurative in a female F344/N rat from a subchronic study (higher magnification of Figure 1). Note the bacterial colonies (arrow). **Figure 3** Esophagus - Inflammation, Suppurative in a male F344/N rat from a chronic study. There is inflammation in the esophagus (asterisk) and in the periesophageal tissue (arrow). **Figure 4** Esophagus - Inflammation, Suppurative in a male F344/N rat from a chronic study (higher magnification of Figure 3). Suppurative inflammation in the periesophageal tissue. **Figure 5** Esophagus - Inflammation, Chronic in a female F344/N rat from a chronic study. The inflammation is composed primarily of lymphocytes and plasma cells. **Figure 6** Esophagus - Inflammation, Chronic in a female F344/N rat from a chronic study (higher magnification of Figure 5). The inflammation is composed primarily of lymphocytes and plasma cells.

Comment: Direct toxic effects of chemicals on the esophagus are rare in NTP studies. Esophageal inflammation in rodents is most commonly caused by gavage trauma and can be associated with ulceration, necrosis, and perforation. In NTP studies, there are five standard categories of inflammation: acute, suppurative, chronic, chronic active, and 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 a significant number of neutrophils are also present. 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. Suppurative inflammation with secondary bacterial infection due to gavage trauma is relatively common in the esophagus (Figure 1, Figure 2, Figure 3, and Figure 4).



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Recommendation: Whenever present, inflammation should be diagnosed and graded. The diagnosis should include a modifier indicating the duration or type of inflammation (i.e., acute, suppurative, chronic, chronic active, or granulomatous). The severity grade depends on the extent of the lesion in the esophagus and the density of the cellular infiltrate. Lesions consistent with an abscess are diagnosed as suppurative inflammation. Associated necrosis or inflammation is not generally recorded unless the inflammation or necrosis is a significant component of the lesion.

References:

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Ackermann MR. 2007. Chronic inflammation and wound healing. In: Pathologic Basis of Veterinary Disease, 4th ed (McGavin MD, Zachary JF, eds). Mosby, St Louis, MO, 153-191.

Brown HR, Hardisty JF. 1990. Oral cavity, esophagus and stomach. In: Pathology of the Fischer Rat (Boorman GA, Montgomery CA, MacKenzie WF, eds). Academic Press, San Diego, CA, 9-30. Abstract**:** http://www.ncbi.nlm.nih.gov/nlmcatalog/9002563

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