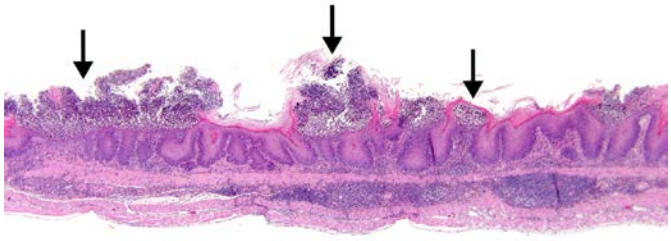
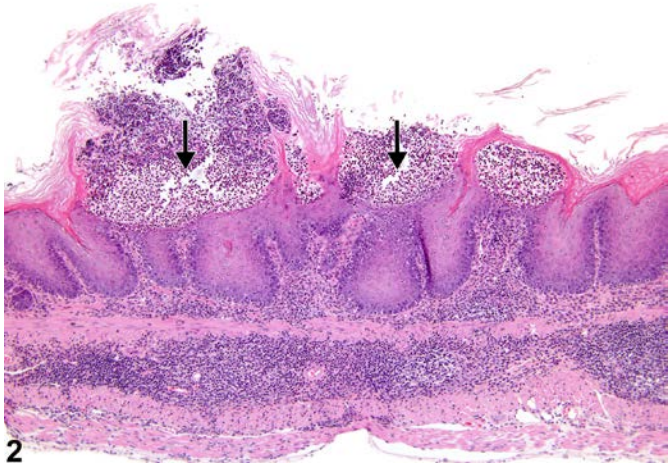
NTP Nonneoplastic Lesion Atlas

Stomach, Forestomach – Erosion



1



2

Figure Legend: Figure 1 Stomach, Forestomach - Erosion in a female B6C3F1 mouse from a chronic study. The superficial layers of the hyperplastic squamous epithelium have been lost (arrows). **Figure 2** Stomach, Forestomach - Erosion in a female B6C3F1 mouse from a chronic study (higher magnification of Figure 1). The superficial layers of the hyperplastic squamous epithelium have been lost (arrows).

Comment: An erosion (Figure 1 and Figure 2) is defined as the loss of superficial epithelial layers of the mucosa, whereas an ulcer is the loss of all epithelial cell layers, extending through to the submucosa. Erosions can occur anywhere in the gastrointestinal tract, but the forestomach is a relatively common location. Erosions often occur in areas of preexisting epithelial hyperplasia. Alternatively, the presence of an erosion can induce epithelial cell proliferation (as a healing response), which can result in epithelial hyperplasia. Inflammation is common in tissues around erosions. Erosion of the forestomach can be spontaneous, secondary to the gavage procedure, or treatment related (especially when the test article is an irritant), but in many cases, the exact cause of erosions is not known.

Recommendation: Erosion of the forestomach should be diagnosed whenever present. Erosions should be graded based on the extent, number, and depth of the lesions. Secondary lesions, such as edema, inflammation, and hyperplasia of the adjacent epithelium, should not be diagnosed separately unless they are prominent components of the lesion or they are considered a primary lesion (e.g., the hyperplasia occurred prior to the erosion). Necrosis of the epithelium is diagnosed instead of erosion if

the necrotic epithelium is still present and attached to the underlying lamina propria (see Stomach, Forestomach - Necrosis).

References:

Bertram TA, Markovits JE, Juliana MM. 1996. Non-proliferative lesions of the alimentary canal in rats GI-1. In Guides for Toxicologic Pathology. STP/ARP/AFIP, Washington, DC, 1-16.
Full-Text: https://www.toxpath.org/ssdnc/GINonproliferativeRat.pdf

Betton GR. 1998. The digestive system I: The gastrointestinal tract and exocrine pancreas. In: Target Organ Pathology (Turton J, Hooson J, eds). Taylor and Francis, London, 29-60.
Abstract: http://www.amazon.com/Target-Organ-Pathology-Basic-Text/dp/0748401571

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

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

Puurunen J, Huttunen P, Hirvonen H. 1980. Is ethanol-induced damage of the gastric mucosa a hyperosmotic effect? Comparative studies on the effects of ethanol, some other hyperosmotic solutions, and acetyl-salicylic acid on rat gastric mucosa. Acta Pharmacol Toxicol (Copenh) 47:321-327.
Abstract: http://onlinelibrary.wiley.com/doi/10.1111/j.1600-0773.1980.tb01567.x/abstract

Authors:

Linda H. Kooistra, DVM, PhD, DACVP
Pathologist
Charles River Laboratories, Inc.
Research Triangle Park, NC

Abraham Nyska, DVM, Diplomate ECVP, Fellow IATP
Expert in Toxicologic Pathology
Visiting Full Professor of Pathology
Sackler School of Medicine, Tel Aviv University
Timrat Israel