



NTP Nonneoplastic Lesion Atlas

Oral Mucosa – Inflammation

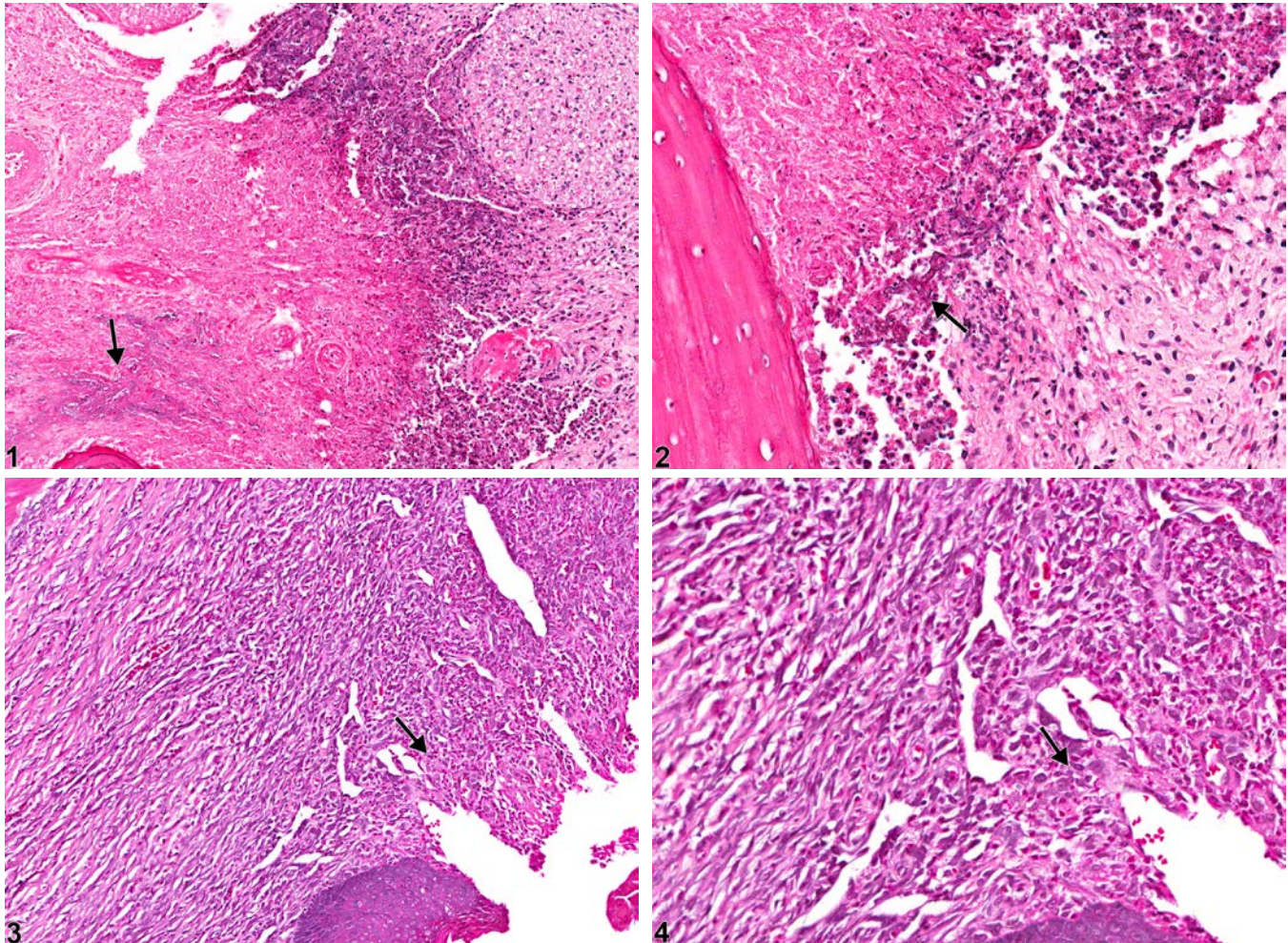
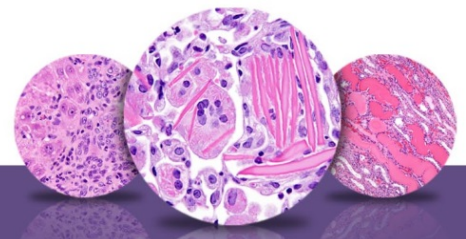


Figure Legend: Figure 1 Oral mucosa - Inflammation, Suppurative in a male F344/N rat from a chronic study. There is an area of suppurative inflammation with necrosis and bacterial organisms (arrow).

Figure 2 Oral mucosa - Inflammation, Suppurative in a male F344/N rat from a chronic study. There is an area of suppurative inflammation with necrosis and fungal organisms (arrow). **Figure 3** Oral mucosa - Inflammation, Chronic active in a male F344/N rat from a chronic study. There is chronic active inflammation subjacent to a mucosal ulceration (arrow). **Figure 4** Oral mucosa - Inflammation, Chronic active in a male F344/N rat from a chronic study (higher magnification of Figure 3). There are neutrophils and mononuclear cells subjacent to a mucosal ulceration (arrow).

Comment: Inflammation in the oral cavity is often secondary to traumatic injury from foreign bodies or gavage procedure or to necrosis from chemical agents. Infectious agents, usually opportunistic



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organisms such as bacteria and fungi, may be seen within the lesion (Figure 1 and Figure 2). In NTP studies, there are five standard categories of inflammation: acute, suppurative (Figure 1 and Figure 2), chronic, chronic active (Figure 3 and Figure 4), 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). 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 may also be present in within the exudate. Grossly, these lesions would be characterized by the presence of pus. The tissue surrounding the exudate may contain 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.

Recommendation: Whenever present, inflammation should be diagnosed, graded, and given a modifier that indicates the duration or type of inflammation (i.e., acute, suppurative, chronic, chronic active, or granulomatous). The severity grade depends on the extent of the area affected and the density of the cellular infiltrate. Lesions consistent with an abscess are diagnosed as suppurative inflammation. Inflammation that is secondary to another lesion, such as ulcer, erosion, or necrosis, should not be diagnosed separately unless warranted by severity. If, however, the inflammation is considered the primary lesion and is the cause of the associated lesions (ulcer, erosion, necrosis, etc.), then the inflammation should be diagnosed and the secondary lesions described in the narrative. Bacteria, fungi, or other opportunistic organisms are not diagnosed separately. Foreign material within the lesion should be diagnosed only if it considered the primary lesion (i.e., the cause of the inflammation). If the pathologist feels the foreign material was introduced subsequent to the inflammation, then the foreign material should be described in the pathology narrative but not diagnosed separately.



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References:

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