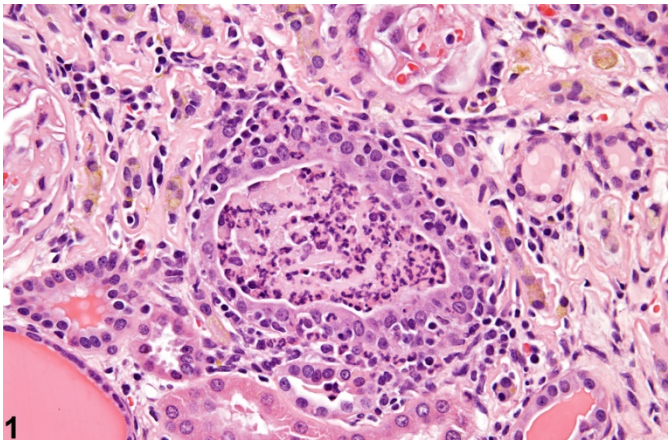
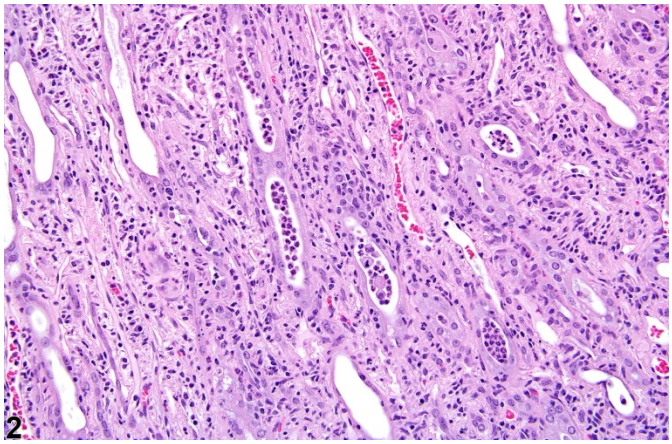
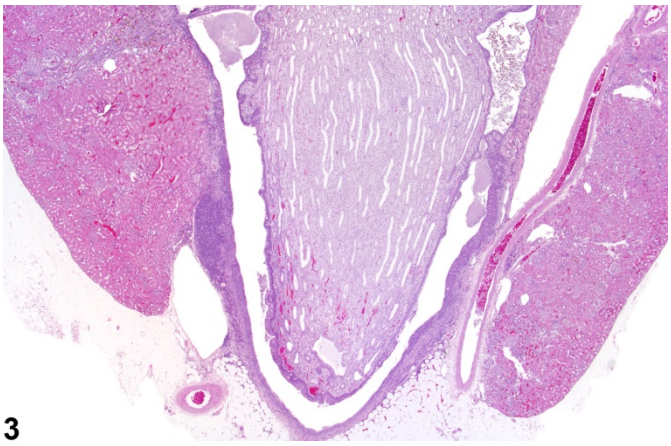
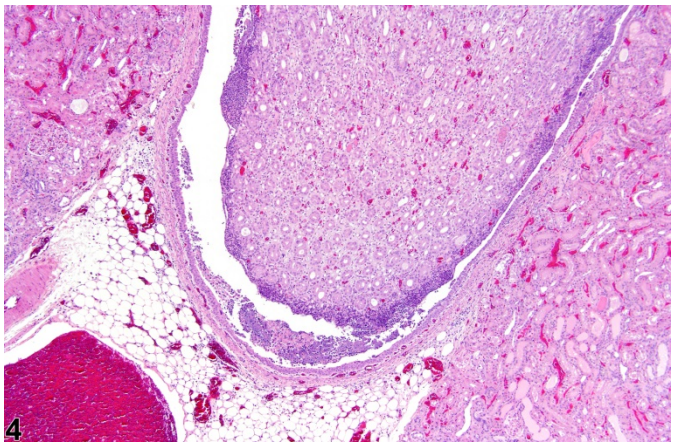
NTP Nonneoplastic Lesion Atlas

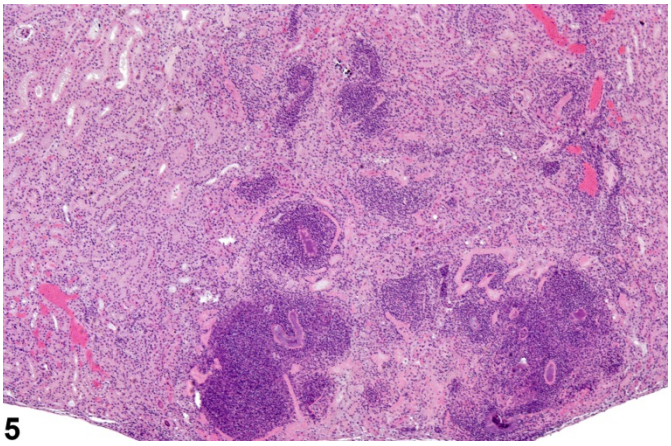
Kidney – Inflammation

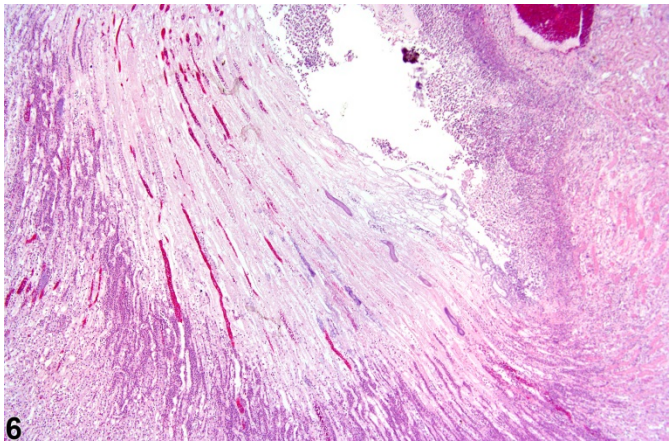

1


2

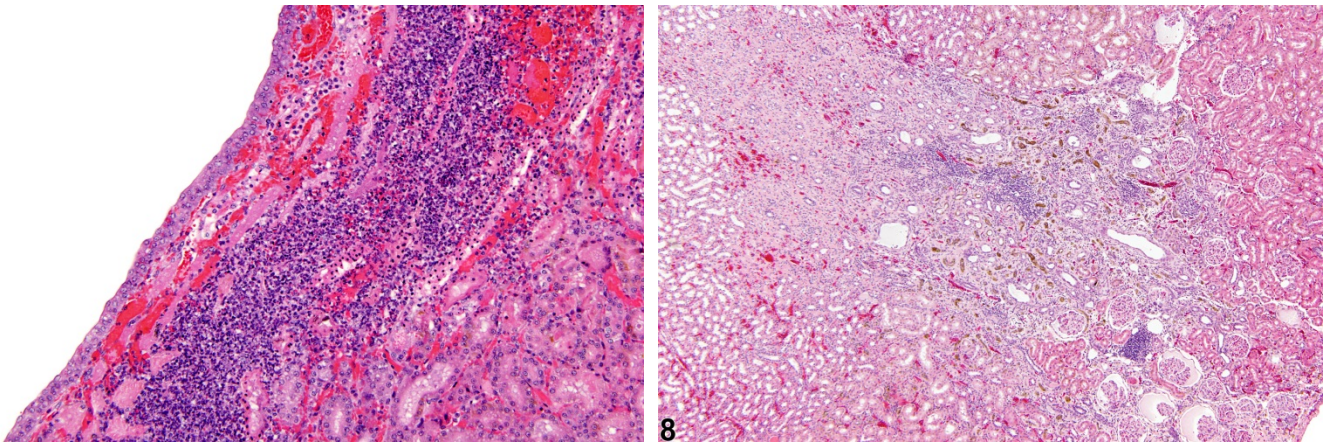

3


4


5


6

Kidney – Inflammation



7 8

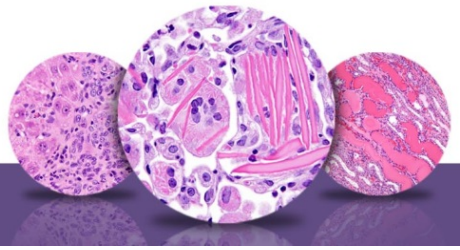
Figure Legend: **Figure 1** Kidney, Renal tubule - Inflammation, Acute in a male F344/N rat from a chronic study. Acute inflammatory cells are present in the renal tubule. **Figure 2** Kidney, Renal tubule - Inflammation, Acute in a female F344/N rat from a chronic study. Acute tubule inflammatory cells associated with extension from renal pelvis inflammation are present. **Figure 3** Kidney, Renal Pelvis - Inflammation, Chronic in a female F344/N rat from a chronic study. Inflammation involves the renal pelvis with infiltrates of inflammatory cells in the pelvis and peripelvic tissue; note the hyperplasia of the papillary epithelium and urothelium. **Figure 4** Kidney, Renal Pelvis - Inflammation, Acute in a male F344/N rat from a chronic study. Acute inflammation is present within the renal pelvis and along the renal papilla. **Figure 5** Kidney, Renal Pelvis - Inflammation, Suppurative in a male B6C3F1 mouse in a chronic study. Microabscesses may result from extension of renal pelvic inflammation into the renal medulla and cortex. **Figure 6** Kidney, Renal Pelvis - Inflammation, Acute in a female F344/N rat from a chronic study. Necrosis of the renal papilla may result from marked inflammation of the renal pelvis. **Figure 7** Kidney, Interstitium - Inflammation, Acute in a male F344/N rat from a chronic study. An acute interstitial inflammatory cell infiltrate and hemorrhage are present adjacent to the renal pelvis. **Figure 8** Kidney, Interstitium - Inflammation, Chronic in a female F344/N rat from a chronic study. An area of chronic interstitial inflammation is characterized by mononuclear inflammatory cell infiltrates and fibrosis.

Comment: In NTP studies, there are five standard categories of inflammation, according to the predominant inflammatory cell type present: 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 and hyperemia. The neutrophil is also the predominant cell type in *suppurative inflammation*, but the neutrophils are aggregated, and many of them are degenerative (suppurative exudate). Lymphocytes predominate in *chronic inflammation*. Lymphocytes also predominate in *chronic active inflammation*, but there are also a significant number of neutrophils. Both lesions 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. In addition, there may be necrosis and hemorrhage, which accompanies the inflammatory reaction. Inflammation should be distinguished from cellular infiltrates. Inflammation in the kidney can occur in the renal tubule, interstitium, renal pelvis, or glomeruli. Inflammatory reactions in the glomerulus are considered a separate entity and covered in a separate document (see Kidney - Glomerulonephritis).

Renal tubule inflammation (tubulitis) (Figure 1 and Figure 2) may be associated with a number of causes that may include the deposition of crystals, extension from the lower urinary tract (pyelonephritis), infectious processes, chronic progressive nephropathy, previous infarction, or direct chemical administration. Tubule inflammation is characterized by the presence of inflammatory cells within the tubule lumen, epithelium, or both.

Inflammation can also be localized to the renal pelvis (Figure 3, Figure 4, Figure 5, and Figure 6) or the interstitium (Figure 7 and Figure 8). Renal pelvis inflammation usually arises from conditions related to localized infections, which ascend from the lower urinary tract and extend into the renal papilla, medulla, and cortex. However, renal pelvis inflammation without lower urinary tract involvement may also be seen. There may be a number of causes for inflammation. Renal pelvis inflammation typically is acute or suppurative, with more chronic lesions noted in peripelvic tissues. Another diagnostic term occasionally used by pathologists to describe a similar condition is “pyelonephritis,” which emphasizes renal involvement along with pelvis inflammation. Microabscesses may be noted in marked cases, due to either an ascending infection or a coinciding septicemic infection. Renal papillary ulceration and/or necrosis may occur as a result of renal pelvis inflammation. Reactive urothelial and/or papillary epithelial hyperplasia are often noted in more chronic cases. Interstitial inflammation, a cellular reaction



to tissue injury from a variety of causes, may be observed frequently in the kidney of rodents. Chronic interstitial inflammation is often seen as a component of chronic progressive nephropathy (see Kidney - Nephropathy, Chronic Progressive).

Recommendation: Inflammation in the kidney should be diagnosed and graded when it is a primary lesion or is severe enough to warrant a separate diagnosis. The diagnosis should include a site modifier (e.g., interstitium, renal tubule, glomerulus) and the type of inflammation (e.g., acute, chronic, chronic active). Inflammatory reactions in the glomerulus should be diagnosed as glomerulonephritis and are covered in a separate document (see Kidney - Glomerulonephritis). The pathologist should use his or her judgment in deciding whether or not secondary lesions such as necrosis or hemorrhage associated with inflammation are prominent enough to warrant a separate diagnosis. Inflammation as a component of chronic progressive nephropathy should not be diagnosed separately.

Reference:

Cattell V, Jennette JC. 1998.. Mechanisms of acute inflammatory and immunologic renal injury. In: Heptistall's Pathology of the Kidney (Jennette JC, Olson JL, Schwartz MM, Silva FG, eds). Lippincott-Raven, Philadelphia, 85-136.

Authors:

John Curtis Seely, DVM, DACVP
Senior Pathologist
Experimental Pathology Laboratories, Inc.
Research Triangle Park, NC

Amy Brix, DVM, PhD, DACVP
Senior Pathologist
Experimental Pathology Laboratories, Inc.
Research Triangle Park, NC