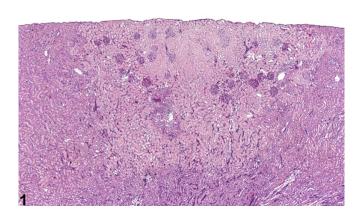
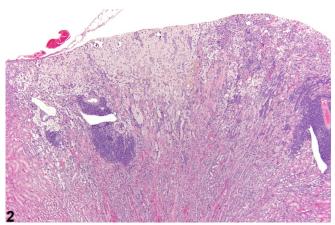


NTP Nonneoplastic Lesion Atlas

Kidney – Infarct





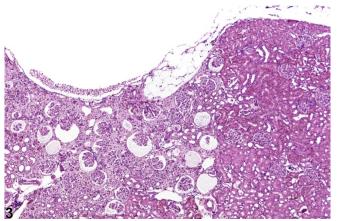


Figure Legend: Figure 1 Kidney - Infarct in a female F344/N rat from a chronic study. A wedged-shaped cortical area of acute coagulative necrosis is present. **Figure 2** Kidney - Infarct in a female B6C3F1 mouse from a chronic study. This infarct has a prominent area of basophilic inflammatory cellularity associated with an infarcted area. **Figure 3** Kidney - Infarct in a female B6C3F1 mouse from a chronic study. This chronic or healed infarct is characterized by cortical depression and interstitial fibrosis.

Comment: Renal infarcts usually appear as well-demarcated, wedge-shaped or triangular areas of coagulative necrosis that extend from the capsular surface into the medulla. The characteristic shape results from the kidney's unique vascular supply. Infarcts can arise spontaneously from a number of causes that compromise the vascular supply, such as neoplastic infiltrates or nephrotoxicants. Acute infarcts have a central area of necrosis with little peripheral inflammation (Figure 1). As the lesion





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progresses, neutrophilic to mononuclear infiltrates become evident near the area of necrosis (Figure 2). Chronic infarcts are characterized by renal parenchymal interstitial fibrosis and capsule surface depression (Figure 3). However, ascribing an infarct to all areas with cortical depression and fibrosis may not be warranted since some might represent areas of previous interstitial inflammation that are resolving.

Recommendation: Infarcts should be diagnosed as infarcts rather than diagnosing the individual components (e.g., necrosis, inflammation, fibrosis). They should be graded according to the extent of renal involvement. In the pathology narrative, infarcts should be designated as either acute or chronic, and if possible, the cause of the infarct should be identified.

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