**Kidney – Cyst**

**Figure Legend:**

**Figure 1** Kidney, Renal tubule - Cyst in a male Tg.Ac (FVB/N) hemizygous mouse from a subchronic study. A spontaneous cortical cyst lined by flattened cells is present. **Figure 2** Kidney, Renal tubule - Cyst in a male Tg.Ac (FVB/N) hemizygous mouse from a subchronic study. A small tubule cyst is lined by cuboidal cells. **Figure 3** Kidney, Glomerulus - Cyst in a male B6C3F1 mouse from a chronic study. A glomerular cyst is present, with the glomerular tuft visible. **Figure 4** Kidney, Renal tubule - Cyst in an F344/N rat. These medullary cysts are the result of a toxic effect on the kidney. **Figure 5** Kidney, Renal tubule - Cyst in a rat. Multiple cysts are present in this polycystic kidney.

**Comment:** A variety of renal cysts can be seen in rodent kidneys. Cysts may be congenital, acquired, or associated with chronic progressive nephropathy or chemical administration and may be noted as isolated findings in the absence of other renal pathology (Figure 1 and Figure 2). They can be solitary or multiple and can occur anywhere in the renal parenchyma, cortex, medulla, or papilla. Cysts can arise from tubules or glomeruli (Figure 3). Some cysts may arise from progressive dilation, whereas others have been postulated to arise from tubules undergoing epithelial cell proliferation. Most cysts are lined by flattened to cuboidal epithelium and appear empty or may contain eosinophilic or amphophilic homogeneous fluid. Larger cysts may be associated with pericycstic and/or tubulointerstitial inflammation and fibrosis. Cysts may arise from chemical administration (Figure 4). Hereditary conditions manifesting as polycystic kidneys have been reported in rats and mice (Figure 5).

**Recommendation:** Primary renal cysts should be diagnosed. Their location should be indicated in the diagnosis with a site modifier (e.g., renal tubule, glomerulus). Cysts need not be given a severity grade unless grading would highlight a toxic effect. Cysts arising as secondary changes associated with a primary lesion such as with increasing severities of chronic progressive nephropathy, interstitial fibrosis, and/or retrograde nephropathy need not be recorded separately. However, the presence of cysts arising as a secondary mechanism needs to be carefully interpreted and diagnosed when appropriate with respect to chemical administration. Inflammation associated with renal cysts should be recorded if warranted by severity or if the pathologist deems the inflammatory reaction important to the overall understanding of the cystic change.
References:


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