

Figure 20-52 Renal cell carcinoma. **A,** Clear cell type. **B,** Papillary type. Note the papillae and foamy macrophages in the stalk. **C,** Chromophobe type. (Courtesy Dr. A. Renshaw, Baptist Hospital, Miami, Fla.)

delicate branching vasculature and may show cystic as well as solid areas. Most tumors are well differentiated, but some show nuclear atypia with bizarre nuclei and giant cells.

As tumors enlarge they may bulge into the calyces and pelvis and eventually fungate through the walls of the collecting system to extend into the ureter. **One of the striking characteristics of renal cell carcinoma is its tendency to invade the renal vein** (Fig. 20-51), in which it may grow as a solid column of cells that extends up the inferior vena cava, sometimes as far as the right side of the heart.

Papillary carcinomas, thought to arise from distal convoluted tubules, can be multifocal and bilateral. They are typically hemorrhagic and cystic, especially when large. The tumor is composed of cuboidal or low columnar cells arranged in

papillary formations. Interstitial foam cells are common in the papillary cores (Fig. 20-52B). Psammoma bodies may be present. The stroma is usually scanty but highly vascularized. **Chromophobe renal carcinoma** is made up of pale eosinophilic cells, often with a perinuclear halo, arranged in solid sheets with a concentration of the largest cells around blood vessels (Fig. 20-52C). **Collecting duct carcinoma** is a rare variant showing irregular channels lined by highly atypical epithelium with a hobnail pattern. Sarcomatoid changes arise infrequently in all types of renal cell carcinoma and are a decidedly ominous feature.

Clinical Features. The classic clinical features of renal cell carcinoma are costovertebral pain, palpable mass, and hematuria, but all three are seen in only 10% of cases. The most reliable clue is hematuria, but it is usually intermittent and may be microscopic; thus, the tumor may remain silent until it attains a large size, often greater than 10 cm. At this time it is often associated with generalized constitutional symptoms, such as fever, malaise, weakness, and weight loss. This pattern of asymptomatic growth occurs in many patients, so the tumor may have reached a diameter of more than 10 cm when it is discovered. Currently, an increasing number of tumors are being discovered in the asymptomatic state by incidental radiologic studies (e.g., computed tomographic scan or magnetic resonance imaging) performed for other indications.

Renal cell carcinoma is considered one of the great mimics in medicine, because it tends to produce a diversity of systemic symptoms not related to the kidney. In addition to fever and constitutional symptoms mentioned earlier, renal cell carcinomas produce a number of syndromes ascribed to abnormal hormone production, including polycythemia, hypercalcemia, hypertension, hepatic dysfunction, feminization or masculinization, Cushing syndrome, eosinophilia, leukemoid reactions, and amyloidosis.

A particularly troublesome feature of renal cell carcinoma is its tendency to metastasize widely before giving rise to any local symptoms or signs. In 25% of new patients with renal cell carcinoma, there is radiologic evidence of metastases at the time of presentation. The most common locations of metastasis are the lungs (more than 50%) and bones (33%), followed in frequency by the regional lymph nodes, liver, adrenal, and brain.

The average 5-year survival rate of persons with renal cell carcinoma is about 70% and as high as 95% in the absence of distant metastases. With renal vein invasion or extension into the perinephric fat, the figure is reduced to approximately 60%. Radical nephrectomy has been the treatment of choice, but partial nephrectomy to preserve renal function is recommended for T1a tumors (<4 cm) as well as larger tumors when technically feasible. Drugs that inhibit VEGF and various tyrosine kinases are used as an adjunct to therapy in patients with metastatic disease.

Urothelial Carcinoma of the Renal Pelvis

Approximately 5% to 10% of primary renal tumors originate from the urothelium of the renal pelvis (Fig. 20-53). These tumors range from apparently benign papillomas to invasive urothelial (transitional cell) carcinomas.

Renal pelvic tumors usually become clinically apparent within a relatively short time, because they lie within the