Kidney: Metanephric adenoma

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Identity

Other names
Embryonal adenoma
Nephrogenic nephroma

Note
A benign tumor resembling differentiation toward early embryonic metanephric tubules.

Classification

Note
Metanephric adenoma belongs to the category of Metanephric tumor in the WHO histological classification of tumors of the kidney.

Clinics and pathology

Epidemiology
Metanephric adenomas are uncommon but occur across a wide range of patients, from infants to elder individual. The peak age frequency is 40 to 69 years. There is a female predominance (2.3:1). Ten percent of patients present with polycythemia.

Clinics
Mostly are incidentally discovered. Some patients present with hematuria and abdominal or flank pain.

Pathology
Grossly, the tumors are solid, gray to tan in color, and homogenous on cut surface. Most tumors are well-demarcated but lack a capsule. Microscopically, the tumor is chiefly composed of tightly packed primitive tubules and acina, with frequent papillary or glomeruloid structures.

The individual tumor cells have scant cytoplasm, round-to-oval nuclei with smooth chromatin, inconspicuous nucleoli. Stromal hyalinization, dystrophic calcification and psammoma bodies are occasionally seen. Mitotic figures are rare or absent. The tumor cells are positive for WT1, PAX2 and CD57, but negative for CK7, CD56 and AMACR.

Treatment
Surgical excision.

Prognosis
The vast majority of metanephric adenomas behave in a benign fashion. Cases of regional lymph node metastasis and sarcomatoid changes have been individually reported.

Cytogenetics

Note
Karyotyping of a total of five cases revealed negative findings, with the exception of a dual balanced translocation of t(1;22)(q22;13) and t(15;16)(q21;p13) in one case. Sporadic allelic imbalances involving chromosomes 2p, 7, 8p, 12q, 16q, and 20q were identified in 18-56% of cases. A deletion in chromosome 2p was described, and a tumor suppressor gene region on 2p13 was delineated.
One study of array comparative genomic hybridization (aCGH) reported no copy number changes in six cases, while another study of a CGH on 9 cases showed normal chromosome copy number in 4, sporadic chromosomal imbalances in 3, and multiple chromosomal gains and losses in 2.
A. The tumor is composed of tubular and solid areas of uniform cells. B. The tumor is immunohistochemically positive for WT-1.

The gain of chromosome 19 was the most common finding (five cases). The gains of chromosomes 7 and 17 noted in papillary renal cell carcinoma, as well as the chromosome gains of 1q, 7q, and 12, and losses of 11p and 16q commonly seen in Wilms’ tumor have not been detected in metanephric adenoma. It should be noted that the trisomies of chromosomes 7 and 17 and loss of Y reported in the series of Brown et al. could not be reproduced in later investigations.
References


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