Imaging of Pediatric Pelvic Neoplasms

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KEYWORDS

• Testicle • Ovary • Germinoma • Sacrococcygeal • Teratoma • Bladder • Neoplasm

PRIMARY TESTICULAR NEOPLASMS

Testicular neoplasms represent approximately 1.0% to 1.5% of all childhood malignancies, with a peak incidence at 2 years of age.[1] Primary testicular tumors are classified by their tissue of origin and are divided into germ cell tumors, sex cord-stromal tumors, and mixed tumors. Germ cell tumors may differentiate into gonadal cell lines, in which case they are called seminomatous germ cell tumors. When they transform into undifferentiated totipotential cells they are termed nonseminomatous germ cell tumors.

A definitive diagnosis is not typically possible with radiological studies; however, imaging may be able to limit the differential diagnosis, especially when considered in the context of clinical history. Ultrasonography (US) is the initial modality of choice to image scrotal masses. Computed tomography (CT) is helpful for staging, but has the disadvantage of requiring ionizing radiation. Magnetic resonance (MR) imaging may be used for staging and as a problem-solving technique in some cases. The sensitivity of both sonography and MR imaging in differentiating between intratesticular and extratesticular lesion location is nearly 100%.[2] Testicular masses tend to have a nonspecific appearance of a solid mass on all cross-sectional imaging modalities. Useful generalizations are described as follows.

Intratesticular masses are more likely to be malignant than extratesticular masses. Aggressive tumors may invade the tunica albuginea, causing a more irregular appearance. Sonography may help distinguish simple versus reactive hydrocele, the latter being associated with testicular neoplasms in 15% to 25% of cases.[3–5] Reactive hydroceles (which may occur secondary to epididymitis, orchitis, testicular torsion, torsion of the appendix testis, trauma, or tumor) and invasion of the tunica albuginea suggest extratesticular involvement and are indications for further workup. Color Doppler sonography can determine the vascularity of a mass and may help to distinguish it from testicular torsion. Hypervascularity is present in 85% of neoplasms, although smaller tumors (<1.5 cm) are often avascular or hypovascular.[1,4] Unfortunately, hypervascularity on color Doppler US is a nonspecific finding seen in both malignancy and inflammation. Avascularity, an unusual finding in neoplasms, suggests a diagnosis of testicular torsion.

The metastatic workup of primary testicular masses includes CT of the chest, abdomen, and pelvis to screen for lung and lymph node involvement. It is also advisable to perform chest CT prior to surgery as postsurgical atelectasis may imitate metastatic lesions. Ultrasound should be used to screen the contralateral testicle for synchronous or metastatic lesions before and after orchiectomy.

Financial Disclosure: None.

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Radiol Clin N Am 49 (2011) 729–748
doi:10.1016/j.rcl.2011.05.007

One condition, testicular microlithiasis (TM), has a controversial association with testicular neoplasms. Although the cause of TM is unknown, some believe that degeneration of cells in the seminiferous tubules may cause formation of microliths. TM has been described in patients with undescended or delayed testicular descent. Some argue that because TM and certain testicular neoplasms are associated with undescended testicles, periodic screening with US may be warranted. Specific disorders that have been associated with TM include Klinefelter syndrome, male pseudohermaphroditism, Down syndrome, and pulmonary alveolar microlithiasis (**Fig. 1**).[6,7]

Germ Cell Tumors

Germ cell tumors represent 70% to 90% of childhood testicular neoplasms.[1] These tumors are classified on a pathologic basis into seminomatous, which are rare in the pediatric population, and nonseminomatous subtypes. Nonseminomatous germ cell tumors are further subdivided into yolk sac tumors, teratoma/teratocarcinoma, embryonal, and choriocarcinoma varieties.

Seminomatous germ cell tumors

Seminomas are most common in adult patients (age in their 40s), presenting as a painless testicular mass. These tumors are rare in the pediatric population. Seminomas are very sensitive to chemotherapy and radiation, and therefore cure rates are high.[8] Seminomas tend to be uniformly hypoechoic, rarely undergoing necrosis or hemorrhage, and on MR imaging are homogeneously hypointense on T1-weighted and hyperintense on T2-weighted imaging (**Fig. 2**).[9]

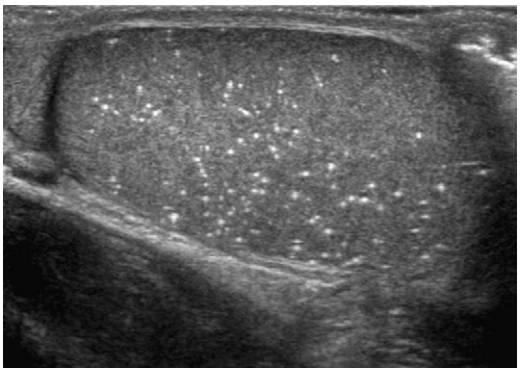

Fig. 1. Testicular microlithiasis in a 3-year-old male with scrotal pain. Longitudinal sonogram demonstrates innumerable foci of increased echogenicity with slight posterior shadowing scattered throughout the testicle. The contralateral testicle (not shown) had a similar appearance.

Nonseminomatous germ cell tumors

Yolk sac tumors Yolk sac tumors (endodermal sinus tumors) represent 80% to 90% of germ cell tumors in childhood, and up to 75% are diagnosed by the age of 2 years (**Table 1**).[10] Clinically, patients present with asymptomatic testicular enlargement and an elevated serum α-fetoprotein (AFP) level in 90% of cases.[9,11]

When used correctly, AFP levels can monitor the effectiveness of treatment, tumor recurrence, or presence of metastasis. However, infants younger than 6 months normally have elevated AFP. If the AFP does not normalize in a baby younger than 6 months after resection of the neoplasm, it is not necessarily an indication of persistent disease.

The majority of yolk sac tumors are confined to the scrotum at presentation, with the remainder having lymphatic metastases to regional/retroperitoneal lymph nodes or hematogenous spread to the lungs. Yolk sac tumors tend to be well circumscribed and heterogeneous in echogenicity on US, depending on the amount of internal hemorrhage, necrosis, and calcification. The appearance on color Doppler US has been described as chaotic and hypervascular (**Fig. 3**).[11] Patients older than 2 years often have a worse prognosis, and stage 1 yolk sac tumors have an 80% survival rate.[6]

Teratomas Testicular teratomas typically occur in boys younger than 4 years and are usually benign.[1] Up to a third of teratomas may metastasize to retroperitoneal lymph nodes within 5 years of diagnosis in postpubertal patients.[10] Postpubertal teratomas have a tendency to develop other components of germ cell tumors (such as choriocarcinoma, seminoma, and embryonal carcinoma), increasing the likelihood of malignancy. Therefore, orchiectomy is the treatment of choice in postpubertal teratomas. Testicular teratoma in a prepubertal patient is more likely to have a benign course, which may allow for tissue-sparing surgery. Metastatic disease from prepubertal teratomas (immature and mature) has not been reported.[12]

Testicular teratomas contain components of all 3 germ cell layers, including bony elements and adipose tissue. Their heterogeneity is reflected on US where they appear as complex masses with cystic and solid components. Bony elements (calcifications) are typically echogenic with posterior shadowing, and adipose tissue is echogenic without shadowing. In some cases superficial calcification will cause so much shadowing that the underlying lesion is hard to visualize. This US finding has been called “the tip of the iceberg” sign. Corresponding findings of mixed osseous, soft tissue, and fat attenuation are observed on CT and MR imaging.