Testicular Cancer

Testicle or testicular cancer develops in one or both testicles in men or boys. The annual age-adjusted incidence rate for this cancer is 4.5 cases per 100,000 men or approximately 6,500 cases in the United States per year (Schottenfeld 1996). Among Massachusetts' males, testicular cancer occurred at a rate of 4.3 cases per 100,000 in 1997. Testicular cancer is one of the most frequently diagnosed cancers in men between the ages of 20 and 44 (Schottenfeld 1996). Among this age group, the U.S. age-adjusted incidence of testicular cancer has increased at least fifty percent between 1973 and 1990 (Schottenfeld 1996). This increase is particularly evident in white males, but due to dramatic advances in the diagnosis and treatment of testicular cancer, age-adjusted mortality rates have declined in this population from 0.8 per 100,000 men in 1973 to 0.3 in 1990 (Schottenfeld 1996).

Over 90% of cancers of the testicle develop in certain cells known as germ cells (ACS 2000). There are two main types of germ cell tumors (GCTs) in men: seminomas and nonseminomas. About half of all testicle germ cell cancers are seminomas. They develop from the sperm-producing germ cells of the testicle. There are two main subtypes of these tumors distinguished by their appearance under the microscope: typical (or classic) and spermatocytic. The average age of men who are diagnosed with spermatocytic seminoma is 65, about 15 years older than the average age of men with typical seminomas (ACS 2000). Nonseminomas tend to develop earlier in life than seminomas, usually occurring in men in their 20's (ACS 2000). The main types of nonseminoma germ cell cancers are embryonal carcinoma, yolk sac carcinoma, choriocarcinoma and teratoma.

Tumors can also arise in the supportive and hormone-producing tissues, or stroma, of the testicles. These are known as gonadal stromal tumors. They account for 4% of adult testicle tumors and 20% of childhood testicular tumors (ACS 2000).

Scientists have found certain risk factors that make a person more likely to develop testicular cancer. The main risk factor for testicular cancer is a condition called cryptorchidism (undescended testicle(s)). About 14% of testicular cancer cases occur in men with a history of cryptorchidism (ACS 2000). Normally, the testes develop inside the abdomen of the fetus and descend into the scrotum before birth. In about 3% of males, however, the testes either do not make this decent or descend incompletely. In the past twenty years, studies have shown that men with a history of cryptorchidism are 2.5 to 11.4 times more likely to develop testicular cancer than men with no history of cryptorchidism (Schottenfeld 1996). Some researchers suggest that surgically correcting cryptorchidism before the onset of puberty may reduce the risk of developing testicular cancer later on in life (ACS 2000).

Most testicular cancers occur between the ages of 15 and 40, but this cancer can affect males at any age (ACS 2000). A family history of testicular cancer increases the risk of developing testicular cancer. If one man has the disease, there is an increased risk that one or more of his brothers will also develop it. The risk of testicular cancer among white men is about five times that of African-American men and more than double that of Asian-American men. The reason for this difference is not known (ACS 2000).

Research has suggested that prenatal exposure to synthetic estrogens, like the drug diethylstilbestrol (DES), which was used between 1945 and 1960 by pregnant women to prevent miscarriage, can lead to the development of testicular anomalies in male offspring (Schottenfeld 1996). However, there is no convincing evidence that DES exposure directly increases a man's risk for developing testicular cancer.

A recent study suggests that men with an occupational history of mining, food and beverage processing, employment in the utilities industry, and employment in the leather industry have an increased risk of developing testicular cancer (Knight et al. 1996). No particular chemicals, however, have been identified as contributing to the development of the disease. Researchers have also suggested that prolonged occupational exposure to extremely low and extremely high temperatures may increase the risk of developing testicular cancer (Zhang et al. 1995).

Recent research indicates that an association between exposure to polyvinyl chloride (PVC), during its production phase, and testicular cancer development may exist. Swedish researchers tentatively concluded that workers exposed to PVC, while it was being produced, had a significantly elevated risk of testicular cancer development (Hardell et al. 1997). One hypothesis on why PVC potentially elevates testicular cancer risk is that production of this plastic often involves the use of phthalates as additives. Phthalates have been reported to have estrogen-like effects (Jobling et al. 1995). Therefore, exposure to phthalates could increase the risk of development of testicular abnormalities. Other studies on exposure to PVC, however, found no such elevated risk of testicular cancer (Hansen 1999, Langard et al. 2000).

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