Eastern Journal of Medicine 3 (2): 78-79, 1998.

Leydig cell tumor of the testis

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Key words Testicle, Leydig cell tumor, adult

Introduction

Leydig cell tumor is a rare form of testicular neoplasm. It represents only 1 to 3 percent of all testicular tumors although it is the most common form of the sex cord-mesenchyme tumors. The majority have been recognized in males between the ages of 20 and 60 years. However approximately one fourth have been reported before puberty (1).

The etiology of Leydig cell tumors is unknown. In contrast to germ cell tumors, there is no correlation with cryptorchidism. Causing experimental production of Leydig cell tumor in mice following chronic estrogen administration or intrasplenic testicular autografting shows its hormonal basis (1). In addition, estrogen and progesterone receptors were detected in about 70 per cent of the Leydig tumor cells in an immunohistologic study, though no receptor was observed in normal Leydig cells (2).

Case report

A 26-year-old man was referred with a 6 months history of right testicular swelling. On genital examination, a mass of approximately 2x2 cm in diameter was detected in the right testicle and was confirmed by scrotal ultrasonography. There was neither clinical nor chemical manifestation of endocrinopathy. Serum estradiol and testosterone levels were in normal ranges. The patient underwent right inguinal orchiectomy. The testis contained a well- circumscribed tan-colored tumor 2x2 cm in diameter. The histological appearance was that of a Leydig cell tumor exhibiting no mitotic figure (Fig 1-2). Chest radiography and CT scanning of the abdomen showed no evidence of metastatic spread.

Discussion

Leydig tumors may be hormonally active, secreting a variety of steroid hormones including primarily testosterone, together, or to a lesser degree with estrogen and its derivates. In adult patients with Leydig cell tumor of the testis, endocrinologic signs occur in 20 % of cases and often precede the onset of a palpable testicular mass (3). Virilizing types of congenital adrenocortical hyperplasia may also produce the endocrine signs and symptoms of Leydig cell tumors. The adrenal cortex and testicle are of common mesodermal origin and the histologic pattern may overlap. Gotoh et al. reported a case who had bilateral testicular Leydig cell tumor with adrenocortical adenoma and suggested to examine the adrenal gland in patients with testicular Leydig cell tumors (4). Adult patients may present with gynaecomastia, loss of libido, feminine hair distribution and genital under-development. Testicular swelling is usually present, but where a discrete tumor mass is not palpable , ultrasound is extremely useful in confirming the presence of a tumor (5). In our case, the patient admitted with tescular swelling as reported usually, not with hormonal disturbance.

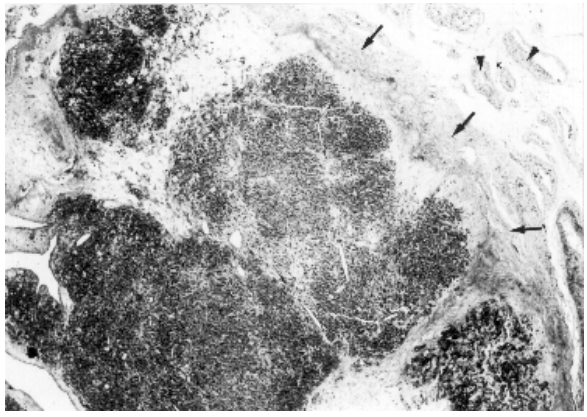

Figure 1. Leydig cell tumor presents sharply delimited solid nodul embedded within the testis (Arrows). Arrow heads show seminiferous tubules. Haematoxylin and eosin X 10.

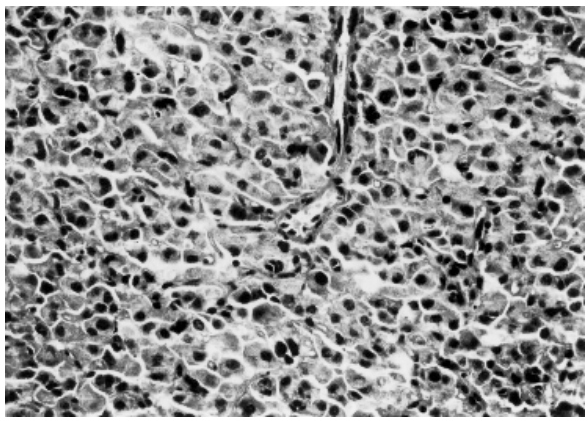

Figure 2. Neoplasm was characterized by solid growth of polygonal cells with abundant granuler acidophilic cytoplasm. Haematoxylin and eosin X 100.

The incidence of bilateral Leydig cell tumor was reported 3-10% in some reports (3,6). 10-to 20% of Leydig cell tumors are malignant (1,7). Malignancy is likely if the tumor is greater than 5 cm in size. Histological features suggesting malignancy are: numerous and atypical mitoses, invasion of vascular

Accepted for publication: 02 June 1998

channels, extension of the tumor into the spermatic cord, invasion of the capsule or the presence of marked cellular pleomorphism (8). However, the only true indicator of malignancy is the presence of metastases. None of these findings were present in our case.

Generally metastatic spread occurs within two years of the primary Leydig cell tumor, and the patient dies within two years of the discovery of metastatic disease (9). The tumor is highly resistant to both radiation and chemotherapy. There fore, some authors suggested to perform retroperitoneal node dissection before or after chemotherapy for staging and also for therapeutic reasons (10).

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