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Case Report

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Leydig Cell Hyperplasia: A Difficult Clinical Diagnosis

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Abstract

A man in his 50s presented to the urology clinic with a history of left sided orchalgia. Testicular ultrasound showed multiple hypoechoic areas in both testes. Serum tumour markers for testicular cancers were not raised and CT imaging did not show any suspicious of metastasis. He underwent a radical inguinal orchidectomy which demonstrated Leydig cell hyperplasia on histological examination.

Keywords: Leydig cell hyperplasia, Leydig cell tumor, Testicular turmor, Testicular Cancer, Leydig cell

Introduction

Leydig cell hyperplasia (LCH) is a rare benign pathology of the testes and ovary which is often clinically difficult to distinguish from tumour [1,2]. It is associated with Klinefelter's syndrome, alcoholism and pernicious anaemia [1,3].

Case Report

A 53-year-old gentleman presented to the urology outpatient department with a history of left testicular pain. He had a past medical history of secondary adrenocortical insufficiency, Crohn's disease, osteoporosis, polyarthralgia and previous alcohol excess.

At examination no inguinal or abdominal masses were palpable. The right testicle was mildly tender but otherwise normal. The left was found to be high in the scrotum and atrophic but had no palpable masses. Routine blood tests were unremarkable as well as tumor markers (PSA 1.3 ng/ml, β HCG <2 mIU/mL, AFP 3.8 ng/ml LDH 472 U/L). Testicular ultrasound scan revealed multiple small hypoechoic areas without convincing vasculature (Figure 1), mostly on the left side.

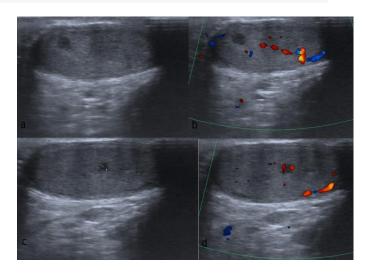


Figure 1: Ultrasound appearances of the left testicle (a and c: left testicular nodules b and d: colour doppler appearances of left testicular nodules).

CT of the chest abdomen and pelvis demonstrated an indeterminate ground glass nodule in the right upper lobe of likely infective origin (which disappeared at follow up). No evidence of metastasis was seen. At Multidisciplinary Team Meeting the decision was made to undertake a left radical inguinal orchidectomy. The patient was offered sperm banking but declined.

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Following removal, the testicle appeared macroscopically unremarkable. Microscopically, the background testis revealed extensive atrophy with Leydig cell hyperplasia (Figure 2), maintaining interstitial distribution of hyperplastic Leydig cell nodules (Figure 3). An inhibin immunostrain highlighted Leydig cells (Figure 4). There was no increase in mitotic activity, evidence of intratubular germ cell neoplasia or invasive malignancy.

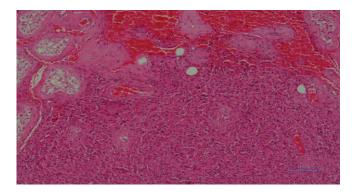


Figure 2: Hematoxylin and eosin strain, high power view with leydig cells seen between atrophic seminiferous tubules.

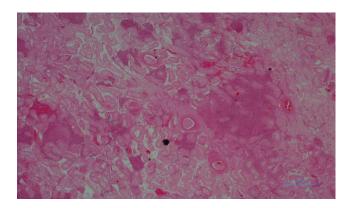


Figure 3: hematoxylin and eosin strain, low power view of testis showing interstitial growth pattern with nodules.

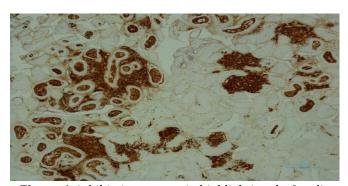


Figure 4: inhibin immunostain highlighting the Leydig cells clusters.

Discussion

Leydig Cell hyperplasia (LCH) Is a rare benign condition which is both clinically and radiologically difficult to differentiate from Leydig Cell Tumour (LCT). Clinically both LCH and LCT typically present with a painless testicular mass. Histologically LCH is defined as an increase in number of cells which can be nodular or diffuse. In both LCH and LCT Leydig cells are normally identified as single cells or clusters around seminiferous tubules [1,4].

Normal Leydig cells are differentiated mesenchymal cells surrounding the testicular stroma and seminiferous tubules. They are large and spherical with small nuclei and contain a small amount of chromatin. They have a large amount of cytoplasm with refractory granules and Reinke's crystals. LCH maintains many of the above features but is differentiated by multifocal areas of large nodular cells without destruction of seminiferous tubules and fewer Reinke's crystals, there are varying degrees of spermatogenic failure with atrophic tubules. They lack cellular atypia, frequent mitosis invasion and necrosis [4].

Management

Most cases of LCH will be diagnosed following orchidectomy or open biopsy for a testicular mass or abnormal ultrasound findings. For patients presenting with gynaecomastia and a testicular mass with or without raised tumour markers, standard management is radical inguinal orchidectomy. If physical examination and ultrasonography are normal, serial examination and ultrasound examination should be carried out for observation. Difficult arises for patients with impalpable suspicious ultrasound findings. If tumour markers are normal then open biopsy and frozen section could be carried out, followed by enucleation.

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