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The Pathophysiology, Genetics and Management of Unilateral Undescended Testes

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Abstract

Undecended testes which effects 1% of children throughout life is an important condition for children and can affect the fertility of males. In addition, it can be associated with testicular malignancy. In the present review article, we present an overview on the currently-accepted pathophysiology, genetics and management protocols of unilateral undescended testes.

Keywords: Undescended testes; Pathophysiology; Management

Introduction

The normal scrotal position of the testis requires its midpoint to lie at or below the mid-scrotal line [1]. The significance of having scrotal position of testes was established many centuries ago. In the Middle Ages, the election of the cardinal to become the future Pope necessitated scrotal examination to confirm that he was a man announcing '*He has testicles, and they dangle nicely - testiculos habet, et bene pendentes*'[2].

Cryptorchidism, a Greek word for 'Hidden testicle', or undescended testis is defined as the failure of one or both testes to correctly lie in the scrotum, representing the most common birth defect in male neonates [3,4].

Testicular maldescent is noted to be more frequent between premature (33%) compared to full-term (2-5%) boys [1]. Nevertheless, more than two-thirds of undescended testes will eventually travel to their scrotal destination during infancy leaving only 1% of persistent cryptorchid testes throughout life [5].

The prevalence of cryptorchidism is variable in the literature depending on the definition employed. Using Scorer definition that considers high scrotal testis as undescended, a study in Denmark revealed 9% prevalence of cryptorchidism [6,7]. Recently, another Danish study reported 2.4% prevalence of cryptorchidism using classification of the John Radcliffe group which considers high scrotal testis to be normal [8,9].

Terminologies related to undescended testis can sometimes be used interchangeably generating clinical confusion [10], therefore, it is crucial to understand the exact meaning of each (Table 1).

Regardless the complicated classification of cryptorchidism into abdominal, inguinal, supra-scrotal, high scrotal or ectopic testis, a simpler clinical description is often used: palpable (80%) versus non-palpable (20%) and unilateral (60-70%) versus bilateral (30-40%) [11].

To date, the etiology of undescended testis remains largely unknown. In addition, the quality of up-to-date data supporting guidelines used for diagnosis and treatment of undescended testis is extremely low [12,13]. In this review article, we present an overview on the currently-accepted pathophysiology, genetics and management protocols of unilateral undescended testes.

Physiology of Normal Testicular Descent

A clear understanding of the pathophysiology of undescended testis requires familiarity with the physiology of normal testicular descent during the embryonic life. The gonads in human embryos are formed close to mesonephros, which replaces the pronephros [14]. The five crucial phases for testicular descent and gubernaculum development starting at week 5 of gestation are illustrated in Table 2.

Enlargement of the gubernaculum produced by cellular proliferation and laying down extracellular matrix is necessary to allow the inguinal canal to expand and allow testicular descent. Other key factors in testicular descent are detachment and migration of gubernaculums [15].

Although not completely clear, it is thought that Insulin-like factor 3 (INSL3), testosterone, anti-Mullerian hormone (AMH) and intact genitofemoral nerve play a role in caudal testicular migration and gubernaculum development [16,17].

Pathophysiology of Unilateral Undescended Testis

Environmental factors:

Following a noticeable rise in the incidence of cryptorchidism in male infants after maternal exposure to diethylstilbestrol (DES), fears of possible link between cryptorchidism and endocrine-disrupting environmental chemicals started to emerge [18].

A large study of 1197 men exposed to DES and 1038 unexposed men reported a relative risk of cryptorchidism of 1.9. The study also suggested that the risk of developing undescended testis rises with significant exposure at earlier gestational age [19].

Although significant number of studies indirectly correlates anti-androgenic and oestrogenic exposure to cryptorchidism [20-22], data are usually only suggestive with no high level of evidence.

In addition, it has been also shown that intrauterine exposure to "environmental endocrine disruptors" could cause male genital malformations such as cryptorchidism, recently [23]. It was presented that "environmental endocrine disruptors" that play a role in the

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