Evaluation of the value of laparoscopy in diagnosis & subsequent treatment of non-palpable testes

Thesis
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To my Mom and Dad

Contents

Introduction

Cryptorchidism or undescended testis refers to a developmental defect in which the testis fails to descend completely into the scrotum. Although cryptorchidism is one of the most common congenital anomalies of the genitourinary system, its pathogenesis is uncertain.

Cryptorchidism is a term that has been used interchangeably with the term undescended testis. Both terms refer to an abnormally positioned testis, but cryptorchidism literally means “hidden testis.” Therefore, an undescended testis may be a more appropriate term because most testes that are not within the scrotum at birth are detectable by palpation.

Its incidence can reach 3% in full term male newborn , decreasing to 1% in male infants aged 6 months to 1 year rising to 30% in premature male newborn **(Thong et al,1998)** with approximately 80–90% unilateral and 10–20% bilateral and about 20% of undescended testicles are non-palpable **(Wenzler et al, 2004).**

The term non-palpable testis implies that the testis cannot be detected on physical examination.

It is sometimes difficult to accurately classify the position of an undescended testis during physical examination as body habitus, testicular position, and compliance of the patient can significantly complicate the clinical evaluation thus more accurate assessment occurs at the time of surgery. Undescended testicular position is most simply described at the time of exploration as intra-abdominal, intra-

canalicular, extra-canalicular (suprapubic or infrapubic), or ectopic **(Caesar and Kaplan, 1994).**

Despite a variety of non-operative imaging techniques have been used to localize non-palpable testis but none of them provide reliable results as ultrasonography, computed tomography & magnetic resonance imaging lack sensitivity & specificity **(Siemer et al, 2000)** considering their high negative results minimally invasive techniques have been introduced to solve this dilemma. One such technique, first described by Cortesi et al 1976, is laparoscopy.

Laparoscopic procedure proved to be highly accurate in providing exact information about the presence, position, size of non-palpable testes & relative size of the vas & the gonadal vessels in addition of being a safe &suitable for all age groups **(Orlando et al, 2003).**

Besides being used as a diagnostic tool, recently laparoscopy is a common technique for treating non-palpable testes**.** It has several advantages over open surgery including clear demonstrations of the anatomy, ability to achieve extensive vascular dissection, less morbidity, fewer complications, decrease length of hospital stay & better cosmesis **(Barqawi et al, 2003)**.

Diagnosis & subsequent management of non-palpable testis remains a challenge to all urologists.

Aim of the study

The aim of this study is to evaluate potential value of laparoscopy in diagnosis & subsequent treatment of non-palpable testes assessing the success rate & potential morbidity of this procedure.

Review of literature

Gonadal embryology

Gonadal Development

During the 5th week, primordial germ cells migrate from the yolk sac along the dorsal mesentery to populate the mesenchyme of the posterior body wall. Under the influence of SRY (the sex-determining region of the Y chromosome), cells in the medullary region of the primitive sex cords begin to differentiate into Sertoli cells, while the cells of the cortical sex cords degenerate. Sex cord cells differentiate into Sertoli cells only if they contain the SRY protein.

During the 7th week, the differentiating Sertoli cells organize to form the testis cords. At puberty these testis cords associated with germ cells undergo canalization and differentiate into seminiferous tubules. Direct cell-to-cell contact between developing Sertoli cells and primordial germ cells is thought to play a key role in the proper development of male gametes. This interaction occurs shortly after the arrival of the primordial germ cells in the presumptive genital ridge.

The testis cords distal to the presumptive seminiferous tubules also develop lumen and differentiate into a set of thin-walled ducts called the rete testis. Just medial to the developing gonad, the tubules of rete testis connect with 5 to 12 residual tubules of nephric ducts, called efferent ductules. The vas deferens also develops from the nephric

duct. At this time, the testicle begins to round up, reducing its area of contact with the surrounding mesonephros. As the testicle continues to develop, the degenerating cortical sex cords become separated from the coelomic (peritoneal) epithelium by an intervening layer of connective tissue called the tunica albuginea.

As the developing Sertoli cells begin their differentiation in response to the SRY, they also begin to secrete a glycoprotein hormone called müllerian-inhibiting substance (MIS). MIS causes the paramesonephric (müllerian) ducts to regress rapidly between the 8th and 10th weeks. Small müllerian duct remnants can be detected in the developed male as a small tissue protrusion at the superior pole of the testicle, called the appendix testis, and as a posterior expansion of the prostatic urethra, called the prostatic utricle.

During the 9th and 10th weeks, Leydig cells differentiate from mesenchymal cells of the genital ridge in response to the SRY protein. These endocrine cells produce testosterone. At an early stage of development, testosterone secretion is regulated by placental chorionic gonadotropin, but eventually the pituitary gonadotropins assume control of androgen production.

Between the 8th and 12th weeks, testosterone secretion by Leydig cells stimulates the nephric (wolffian) ducts to transform into the vas deferens. The cranial portions of the nephric ducts degenerate, leaving a small remnant of tissue protrusion called the appendix epididymis,

and the region of nephric ducts adjacent to the presumptive testicle differentiates into the epididymis.

During the 9th week, 5 to 12 nephric ducts in the region of the epididymis make contact with the sex cords of the future rete testis. It is not until the 3rd month, however, that these tubules actually establish communication with the rete testis as the efferent ductules. Meanwhile, the nephric duct–derived tubules near the inferior pole of the developing testicle degenerate, sometimes leaving a remnant of tissue protrusion called the paradidymis **(John M Park, 2007)**

Gonadal descent

Prior to gonadal differentiation, the testicle lies near the developing kidney, loosely held in place by two ligamentous structures. The dorsal ligament is referred to as the cranial suspensory ligament (CSL), whereas the ventral ligament later develops into the gubernaculum.

Between 10 and 15 weeks, the testicle is anchored near the inguinal region by both enlargement of the gubernaculum and regression of the CSL. Insulin-like 3 (INSL3) causes the swelling and enlargement of gubernaculum and androgens cause an involution of cranial suspensory ligament (CSL)**.**

Starting in the 7th month, the gubernaculum begins to bulge beyond the external inguinal ring and descends to the scrotal location, while, simultaneously, it is hollowed out by the evaginating peritoneal

diverticulum called the processus vaginalis. The processus vaginalis allows the intra-abdominal testicle to exit the abdominal cavity.

Shortening of the cord may be an important mechanism to position the testicle over the inguinal ring to permit abdominal pressure to push the testicle out of the abdomen although intra-abdominal pressure may not be a factor during the initial transabdominal descent; it is thought to be important during transit through the inguinal canal and the subsequent scrotal migration**.**

Insulin-like 3(INSL3) is a hormone identified by Adham et al as a novel gene product of the Leydig cells in 1993. Insulin-like 3(INSL3) is similar in structure to the peptide hormones relaxin and insulin and is expressed in both fetal and adult Leydig cells in a differentiation-dependent manner. Lacking functional insulin-like 3 (INSL3) genes demonstrate intra-abdominal cryptorchidism but otherwise no obvious defects in other male reproductive organs **(John M Park, 2007)**