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CLINICAL CASE

Transverse testicular ectopia: diagnostic and therapeutic algorithm

Ectopia testicular transversa: algoritmo diagnóstico y terapéutico

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What do we know about the subject matter of this study?

Transverse testicular ectopia has an incidence of 1 in 4 million children. It should be suspected in patients with a non-palpable testicle and contralateral inguinal hernia. It is essential to rule out the presence of Müllerian remnants for proper management.

What does this study contribute to what is already known?

Our work provides a diagnostic and therapeutic evaluation algorithm that allows for an organized approach to the comprehensive assessment required by patients with transverse testicular ectopia.

Abstract

Transverse testicular ectopia (TTE) is an uncommon anatomical anomaly where a testicle is mistakenly guided into the contralateral inguinal canal and may be associated with persistent Müllerian duct syndrome (PMDS). **Objective:** To describe 2 cases of TTE and to propose a diagnostic and therapeutic algorithm. **Clinical Cases:** Case 1: Term newborn, male, with bilateral non-palpable testicles. At one month of age, two structures with testicular consistency were palpated in the right inguinal canal. Ultrasound showed left TTE. Karyotype 46XY. At 8 months, he underwent bilateral open orchiopexy and resection of pelvic Müllerian remnants. Low anti-Müllerian hormone levels. Findings were consistent with PMDS. After 3 months, cystoscopy and laparoscopy were performed, ruling out Müllerian remnants and allowing for a new descent of the ascended left testicle. Postoperative recovery was favorable. Case 2: Term newborn, male, with a non-palpable right testicle and left inguinal volume increase at birth. Ultrasound at one month of age showed a left inguinal hernia and right TTE. Karyotype 46XY. Normal hormonal study. Pelvic ultrasound and exploratory laparoscopy showed no Müllerian remnants. At 9 months, cystoscopy was normal, and laparoscopic

Keywords:
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right orchiopexy and left inguinal hernioplasty were performed. Postoperative follow-up was without complications. **Conclusions:** TTE is an infrequent condition that requires careful evaluation, including preoperative ultrasound, karyotype, and hormonal tests, as well as intraoperative cystoscopic and laparoscopic assessment to rule out the presence of Müllerian remnants. Treatment depends on the association with PMDS.

Introduction

Transverse testicular ectopia (TTE) or crossed testicular ectopia was described for the first time in 1886 by Michael Von Lenhossek[1]. It consists of both testes descending through the same inguinal canal into the same hemiscrotum or inguinal region. An incidence of 1 in 4 million children has been reported[2] and, in up to 30% of cases, it can be associated with persistent Müllerian duct syndrome (PMDS), of which about 300 cases have been reported in the literature[3].

The development of the gonads, the internal and external genitalia in the human embryo, consists of 3 sequential stages: a) undifferentiated stage, b) gonadal determination in testes or ovaries, and c) differentiation of internal and external genitalia. In the XY male, until the 8th week, the internal genitalia are undifferentiated with 2 pairs of Wolffian ducts and 2 pairs of Müllerian ducts. Testicular differentiation in the XY embryo begins in the 7th week, with the activation of *SRY* gene expression, located on the short arm of the Y chromosome, and regulated, among others, by genes such as *SF1* and *WT1*, which trigger the expression of the *SOX9* gene which, at the same time, stimulates a chain of critical factors for the differentiation of the undifferentiated gonadal crest towards the testes (*SOX3*, *SOX8*, and *SOX10*, among others) and suppresses the expression of ovarian development factors (RSPO1/WNT4/FST/B-catenin). At the same time, Sertoli cells differentiate and produce anti-Müllerian hormone (AMH), which is responsible for the regression of the Müllerian ducts. Starting from the 8th week, Leydig cells, stimulated by the beta subunit of human chorionic gonadotropin (β-hCG) and later by fetal luteinizing hormone (LH), secrete testosterone, which drives the differentiation of the Wolffian ducts into the epididymis, vas deferens, seminal vesicles, and ejaculatory ducts. Additionally, testosterone is converted into dihydrotestosterone by the action of 5-alpha-reductase, leading to virilization of the external genitalia. When AMH production or secretion fails in males, its secretion is delayed, or there is a failure in the response of the target organs, the Müllerian ducts do not regress, resulting in the development of the fallopian tubes, uterus, and the upper third of the vagina, leading to persistent Müllerian duct syndrome (PMDS)[4].

On the other hand, normal testicular descent involves two stages: the intra-abdominal stage (from weeks 8 to 15) and the inguinoscrotal stage (between weeks 23 and 35), which concludes with the proper fixation of the gubernaculum to the scrotum and the obliteration of the processus vaginalis[5].

The exact etiology of TTE is not yet understood. However, some causal factors have been described, such as the development of both testes from the same germinal crest, early fusion of the developing Wolffian ducts, testicular adhesion to Müllerian structures, and obstruction of the inguinal ring, which may cause failure of testicular descent on the ipsilateral side. The most accepted theory is the abnormal fixation of the gubernaculum[6].

The diagnosis of TTE has changed in recent years, shifting from an intraoperative finding to a preoperative diagnosis, in which physical examination and ultrasound evaluation play a fundamental role[6]. However, due to its low frequency, there is still a lack of knowledge about this pathology, regarding its diagnostic evaluation, the importance of studying the presence of Müllerian remnants, and the available therapeutic options.

The objective of this report is to describe 2 cases with TTE and to propose a diagnostic and therapeutic algorithm to guide the medical team in performing a comprehensive and orderly evaluation of this pathology.

Clinical Cases

Case 1

Male term newborn (38 weeks), with bilateral non-palpable testes, normal scrotal and penile development. At one month of age, two structures consistent with testes were palpated in the right inguinal canal; inguinal and scrotal ultrasound showed both testes located in the right inguinal canal, with a symmetrical appearance (Figure 1 A). Karyotype: 46,XY. At 8 months, bilateral testicular descent was performed through a right inguinal incision which was later extended to the left (Pfannenstiel type) (Figure 1B and C), with transabdominal reposition of both testes in their respective hemiscrotum and careful resection,

using magnification, of pelvic structures suggestive of Müllerian remnants, which were confirmed in the deferred biopsy. Figure 1 D shows the schematic representation of left transverse testicular ectopia associated with Müllerian remnants.

Since the cleavage plane between the Müllerian structures and the noble structures was not easily recognizable, the biopsy reported a sample of Wolffian origin (vas deferens). At 11 months of age, AMH level was 4 ng/ml (normal range: 95.80-326.19), measured by enzyme immunoassay (EIA) method; follicle-stimulating hormone (FSH), LH, and testosterone were within normal ranges for age.

At 12 months of age, the case was presented to the different sexual development team as a case of PMDS. 3 months after, a study of persistent Müllerian remnants was completed with cystoscopy and laparoscopy, ruling out their presence. In addition, in the same surgical procedure, a new scrotal descent of the ascended left testis was performed, likely secondary to scarring in the new descent pathway, since the ectopic testis was properly positioned in the scrotum during the first post-descent follow-up visit but ascended in subsequent visits. At the 12-month follow-up after the last surgery (2 years and 3 months of age), both testes were in the scrotum, with the left one slightly larger than the right one, and the abdominal scar was uncomplicated.

Case 2

Male term newborn (39 weeks), non-palpable right testis at birth, left testis in the scrotum, and left inguinal enlargement. Inguinal and scrotal ultrasound at one month of life showed a left inguinal hernia with adipose tissue and the right testis in the left inguinal canal with Valsalva maneuver, both testes symmetrical (0.2 cc). He was evaluated by the urology team at 5 months of age. Physical examination revealed a normal-appearing penis and scrotum, with a non-palpable right testis. The findings were consistent with right TTE.

The case was presented to the different sexual development team and a study was completed to rule out PMDS. Karyotype 46,XY. Hormonal study (FSH, LH, AMH, and testosterone) within normal ranges for age. Pelvic ultrasound without evidence of Müllerian remnants. Inguinal and scrotal ultrasound showed the right testis located near the left deep inguinal ring (volume: 0.35 cc) and the left testis within the inguinal canal (volume: 0.5 cc) (Figure 2A-D). Figure 2E shows a schematic representation of right TTE.

At 9 months, cystoscopy was performed, without evidence of Müllerian remnants or prominent utricle; exploratory laparoscopy confirmed absence of Müllerian remnants (Video 1; Figures 2F-H). A right testicular descent was performed using the Fowler-Stephens approach, along with a left inguinal hernioplasty during the same surgical procedure. Postoperative follow-up at three months showed no complications, with both testes located in their respective hemiscrotum.

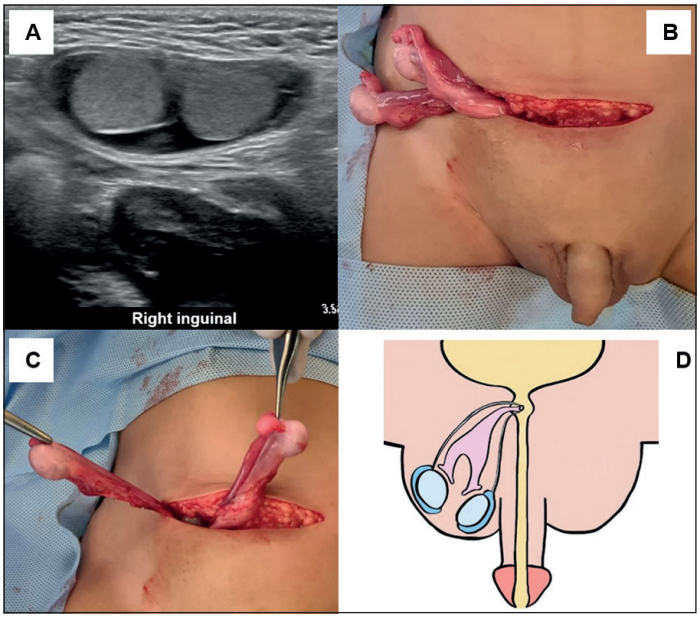

Figure 1. Imaging and intraoperative findings Case 1. Inguinal ultrasound showing both testes in the right inguinal region **(A)**. Intraoperative images of both testes in the right inguinal region, associated with Müllerian remnants (**B** and **C**). Schematic representation of left transverse testicular ectopia associated with Müllerian remnants **(D)**.

Discussion

Clinical suspicion of TTE is essential for a complete preoperative evaluation and appropriate management. Based on the clinical findings, TTE is classified into 3 types according to the location of the testes and Müllerian structures (Figure 3)[2,6]. PMDS is defined as the presence of structures derived from the Müllerian duct (Müllerian remnants) in a male with normal phenotype (male) and genotype (46,XY)[7]. It is of autosomal recessive genetic cause in 85% of the cases due to a defect in the AMH synthesis gene on chromosome 19 (PMDS type I, 45%) or a defect in the AMH receptor gene (*AMHR2*) on chromosome 12 (PMDS type II, 40%). In 15%, the cause is unknown (idiopathic PMDS)[3].

The clinical presentation of TTE has traditionally been described as an intraoperative finding during surgeries for other conditions such as inguinal hernia, hydrocele, or cryptorchidism[6,8]. In a retrospective review, Zhou et al.[6] reported that 93.8% of cases were diagnosed preoperatively by physical examination and ultrasound. The mean age of presentation in the

literature is 24 months and in their case series, it was 16.7 months. TTE should be suspected in patients with non-palpable testis and contralateral inguinal hernia, or when both testes are palpable in the same inguinoscrotal region. It should be noted that some cases may present as an incarcerated inguinal hernia[9,10] or as testicular torsion of the ectopic testis, due to its inadequate fixation[7,11].

In addition, it is essential to suspect and rule out PMDS in patients diagnosed with TTE due to the high probability of association of both conditions[7]. In PMDS, Wolffian and Müllerian duct structures coexist in a male phenotype, since external virilization is complete due to the presence of testosterone[3]. Therefore, the urethra opens at the tip of the penis. The absence of external genital ambiguity differenti-

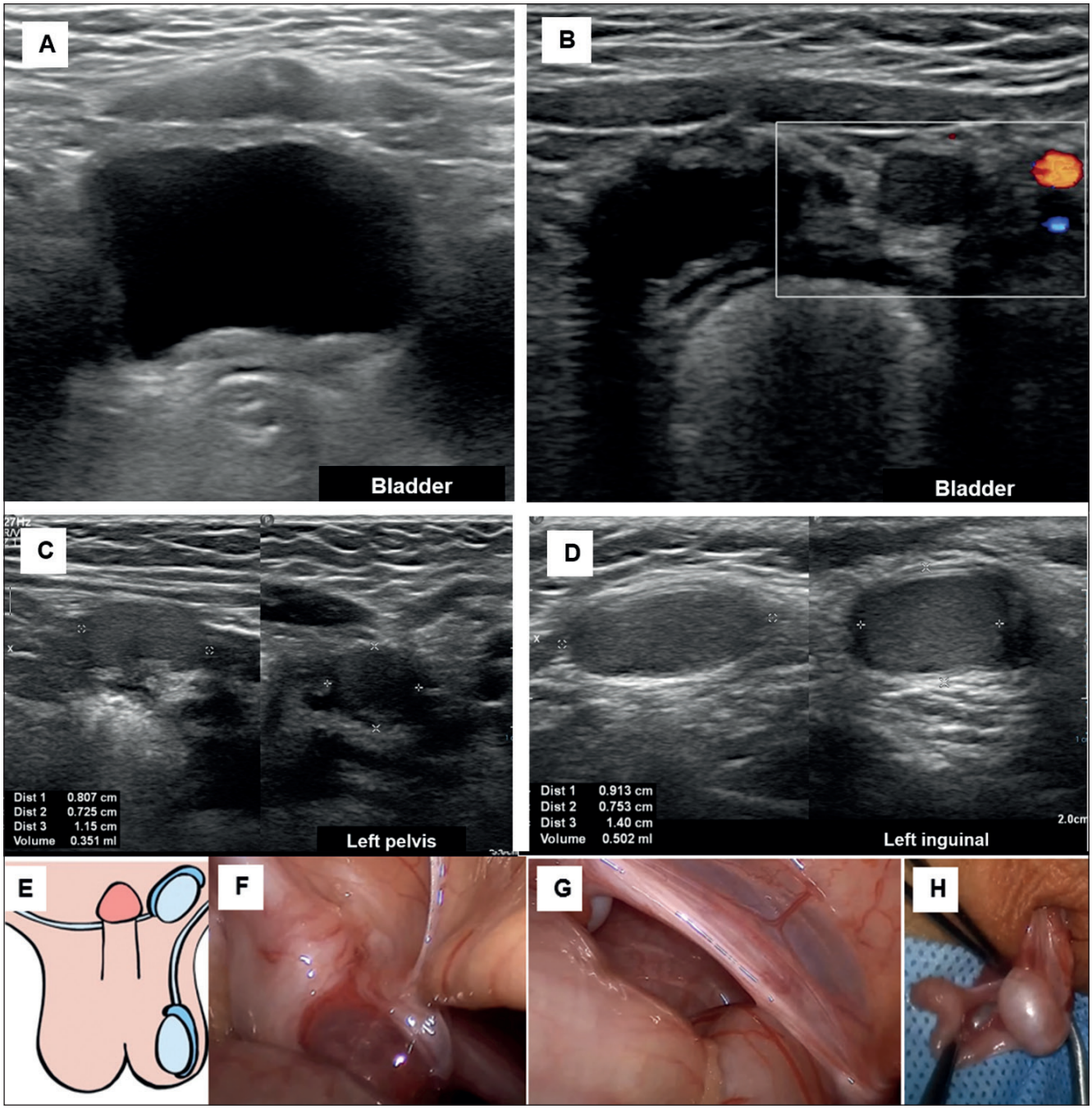

Figure 2. Imaging and intraoperative findings Case 2. Pelvic ultrasound without evidence of Müllerian remnants (**A**), right testis on the left, lateral to the bladder (volume 0.35 cc) (**B** and **C**). Inguinal and scrotal ultrasound showing the left testis in the ipsilateral inguinal canal (0.5 cc) (**D**). Schematic representation of right transverse testicular ectopia (**E**). Intraoperative images of the right testis adjacent to the left deep inguinal orifice (**F** and **G**). Right testis descended into the ipsilateral scrotum (**H**).

ates PMDS from mixed gonadal dysgenesis, a completely different type of different sexual development affecting both Leydig and Sertoli cells[7]. It has been described in adults, that PMDS can manifest with cyclic hematuria, urinary tract infections, and urolithiasis[7,12].

Video 1. Case 2. Exploratory laparoscopy revealed the right testis adjacent to the left internal inguinal ring, which was widely open. Additionally, the absence of pelvic Müllerian remnants was confirmed. Right testicular descent was performed through the route described by Fowler Stephens, with the testis reaching the ipsilateral scrotum without tension.

Note: To scan video codes, you must focus on the image with your smartphone's camera and open the link that appears. Sometimes you'll need to install a QR code reader app.

Preoperative ultrasound in TTE allows for determining the anatomical location of the testis, testicular volume, and the presence of inguinal hernia. In a study published by Zhou et al.[13] it was found that, in TTE, the correlation between physical examination and ultrasound was low; however, the correlation between ultrasound and laparoscopy in the diagnostic evaluation was 100%. In addition, they noted that the volume of the ectopic testis is usually smaller than the contralateral one. Knowing the anatomical location before surgery may improve surgical performance and minimize the risk of complications[13]. In addition, pelvic ultrasound allows assessment for the presence of Müllerian remnants[7,13], which are described as a cord-like hypoechoic mass or a cystic anechoic mass between the bladder and rectum. While it is a useful technique, it has a lower diagnostic yield in PMDS than laparoscopy. This may be related to factors such as pelvic development in infancy and special technical requirements because an acoustic window is needed to visualize the deep pelvic structures (full bladder), which is not easy to achieve in young children[13].

In patients with TTE associated with PMDS, the karyotype is normal (46,XY)[6,7]. In this group of patients, and depending on the level of AMH, molecular genetic analysis of mutations of the AMH synthesis gene or its receptor can be considered[14]. In these cases,

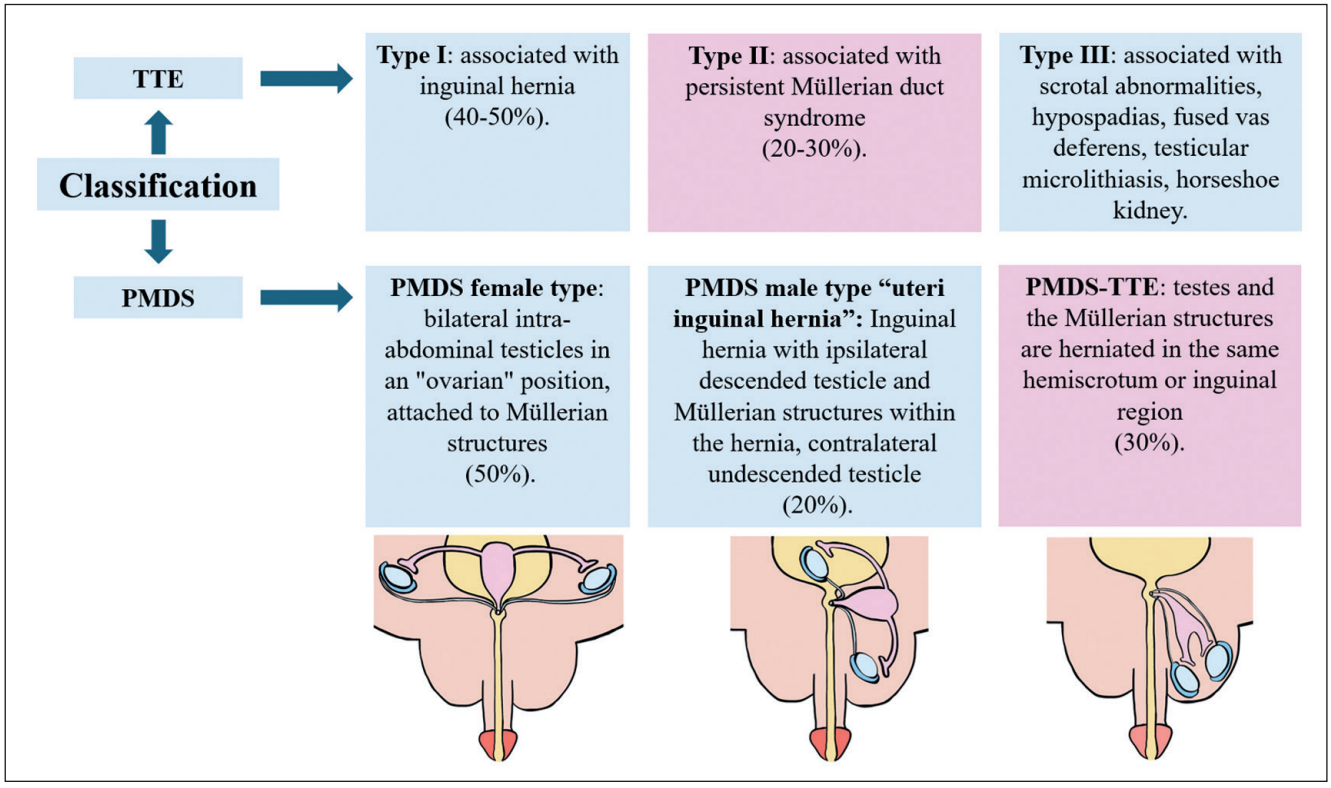

Figure 3. Classification of transverse testicular ectopia and persistent Müllerian duct syndrome[3,6,7].TTE: transverse testicular ectopia, PMDS: persistent Müllerian duct syndrome.

the hormone tests completes the preoperative evaluation and should include function evaluation of Sertoli cells (FSH and AMH) and Leydig cells (LH and testosterone). In PMDS, AMH synthesis gene mutations, with some exceptions, are associated with an unstable protein and therefore with very low or undetectable serum AMH in prepubertal male patients, compared to normal AMH levels for age in AMH receptor defects, because testosterone and FSH concentrations are normal in these patients[15,16].

In the intraoperative evaluation, it is initially beneficial to perform diagnostic cystoscopy and laparoscopy, complementing the search for Müllerian remnants. Cystoscopy allows for the identification of any communication between the urethra and Müllerian remnants, which originate at the opening of the prostatic utricle at the verumontanum, in the posterior urethra[12].

Diagnostic laparoscopy is useful to confirm the diagnosis of TTE, map the anatomy, evaluate the cord structures and vascular supply, verify the presence of Müllerian remnants, and plan definitive surgical management. The most common laparoscopic findings in TTE include: ectopic testis, with crossing of vessels and the vas deferens over the midline toward the contralateral internal inguinal ring; and associated with PMDS: the presence of rudimentary Müllerian structures, such as the uterus, round ligament, and fallopian tubes[6,17].

The objectives of TTE management are to preserve gonadal function and fertility potential and to reduce the risk of testicular malignancy, which has been reported in 5-18% of cases. Tumors typically present in adulthood, with seminomas and other germ cell tumors being the most common[7]; in the case of associated PMDS, it is also important to be aware of the risk of malignancy in Müllerian remnants, which has been reported with a rate of 3.1 to 8.4%, with an age of onset ranging from 4 to 68 years. Malignant neoplasms reported include adenocarcinoma and adenosarcoma[3]. Additionally, surgery is recommended to be performed after 6 months of age and no later than 18 months of age[6]. Orchidopexy, as in cryptorchidism due to other causes, does not eliminate the risk of malignant degeneration, but it likely allows for early detection[18]. Furthermore, because an inguinal hernia is almost invariably associated with testicular ectopia, it should be searched for and repaired during surgery[6].

Surgical options include laparotomy, inguinal approach, laparoscopically-assisted inguinal approach, and laparoscopy[6]. Laparoscopy is increasingly becoming the preferred method for both diagnosing and treating TTE and its associated anomalies[19,20].

For orchidopexy, both transseptal and transabdominal approaches have been described. The transseptal approach (Ombredanne technique) is recommended if there is an adequate length of the vas deferens to allow the ectopic testicle to be placed without tension in the correct scrotum. A transseptal incision is made, through which the ectopic testis is passed and fixed to the correct hemiscrotum. However, the spermatic cord still crosses the midline and passes through the contralateral inguinal canal. Another option is to proceed with a "contralateral" transseptal orchiopexy (modified Ombradanne technique), in which the orthotopic testis with an adequately long vas deferens can cross transseptally, while the ectopic testis with inadequate length undergoes transseptal fixation. A transabdominal orchidopexy is performed if the length of the vascular supply of the vas deferens is inadequate, despite dissection of the proximal insertions up to the level of the internal inguinal ring[17,21,22]. When transseptal orchidopexy is not possible, another option described is to fix both testes in the same hemiscrotum[2]. If an atrophic ectopic testis is found, it should be removed. A staged procedure to bring the ectopic testis into the correct canal has also been described[23].

In cases of TTE not associated with PMDS, as in case 2, we recommend performing the orchidopexy laparoscopically, since the crossed ectopic testis can usually be rescued intra-abdominally from the contralateral side and carefully separate the ipsilateral testis and cord which, due to its condition, has an adequate length that facilitates ipsilateral testicular descent.

The removal of Müllerian remnants is still controversial[6]. On the one hand, there is the risk of malignancy described and there are studies that recommend their removal[18], and on the other hand, dissection of the Müllerian remnants can cause damage to the vas deferens closely related to the testis or its vascularization[6,18]. Infertility is the most common complication of PMDS[7]. Some causes of this can be late orchidopexy, damage to the testis and vas deferens during surgery, and abnormal anatomical connection of the testis to the excretory ducts[16].

For this reason, some authors recommend leaving Müllerian remnants in place and monitoring them. If testicular descent remains restricted due to the short length of the Müllerian remnants, even after sacrificing the gonadal vessels, the main body of the Müllerian structure (uterus) can be divided sagittally, allowing for the lateral separation of each half to facilitate proper descent[7].

If the Müllerian remnant is left in place, the literature recommends regular ultrasound monitoring, e.g., annually, to evaluate changes in size or new mass lesions. Soft tissue magnetic resonance imaging is also a

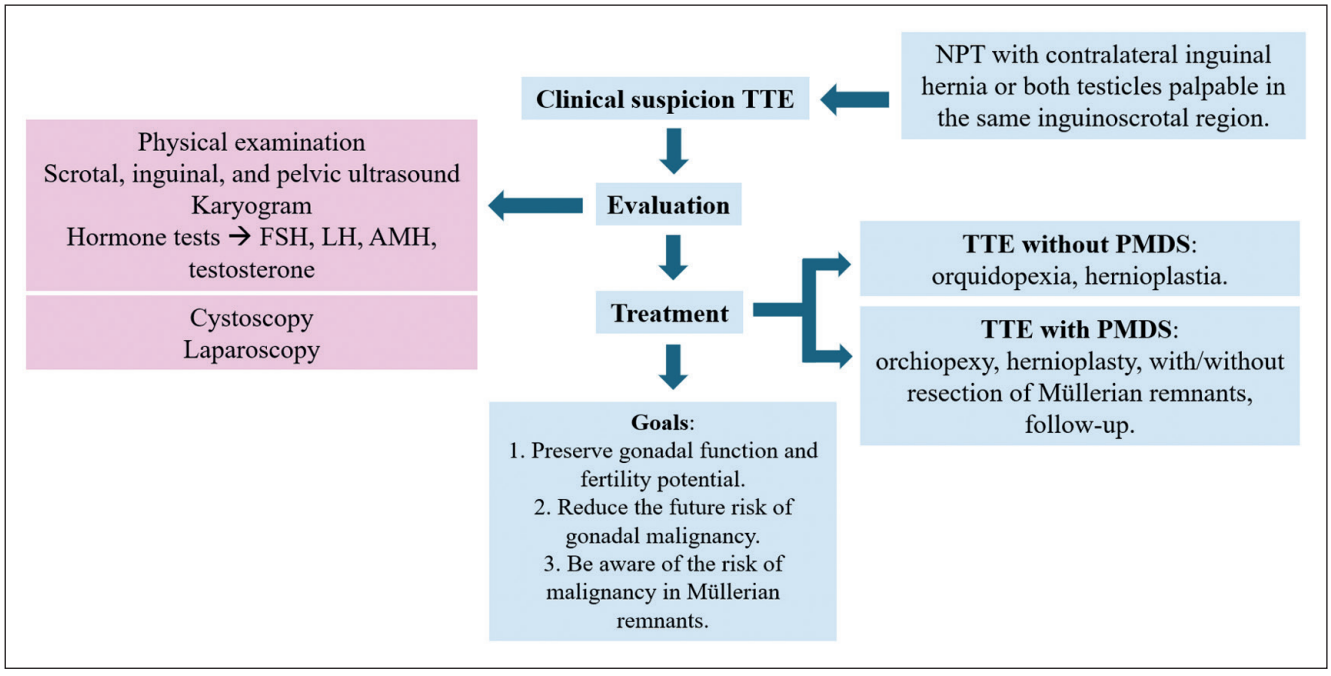

Figure 4. Diagnostic and therapeutic algorithm for transverse testicular ectopia. TTE: transverse testicular ectopia, NPT: non-palpable testis, FSH: follicle-stimulating hormone, AMH: anti-Müllerian hormone, LH: luteinizing hormone, PMDS: persistent Müllerian duct syndrome.

useful modality for monitoring and evaluating changes in the Müllerian remnant. Currently, there is no data suggesting whether one modality is better in terms of monitoring, nor is there guidance on the frequency of monitoring. If a change is detected during follow-up, the appropriate intervention should be determined[3]. Zhou et al. detected no malignancies from residual Müllerian remnants during 134 months of follow-up. There is agreement that these patients should be under follow-up for life[6].

In the cases reviewed in this study, case 1 was compatible with a TTE type II, which corresponds to PMDS associated with TTE, and, case 2, with a TTE type I. Case 1 was the first patient presenting with this pathology in our center, which motivated us to perform an exhaustive review of the subject; thus, case 2 had a complete preoperative evaluation and led our team to perform this evaluation algorithm. In case 2, laparoscopy allowed us to perform the diagnosis and treatment in the same procedure, and the adequate length of the cord facilitated the ipsilateral testicular descent.

We present a diagnostic and therapeutic algorithm that allows the healthcare team to guide the evaluation of patients with TTE (Figure 4). Finally, we highlight the importance of performing a preoperative evaluation according to the proposed algorithm in this pathology and having the participation of a multidisciplinary team composed of a urologist, a geneticist, and an endocrinologist, thus a comprehensive therapeutic approach can be offered to the patient.

Conclusions

TTE is a rare pathology that requires thorough evaluation, including ultrasound, karyotype, and preoperative hormone tests, as well as cystoscopy and laparoscopy to rule out the presence of Müllerian remnants. Treatment depends on the association with PMDS.

Ethical Responsibilities

Human Beings and animals protection: Disclosure the authors state that the procedures were followed according to the Declaration of Helsinki and the World Medical Association regarding human experimentation developed for the medical community.

Data confidentiality: The authors state that they have followed the protocols of their Center and Local regulations on the publication of patient data.

Rights to privacy and informed consent: The authors have obtained the informed consent of the patients and/or subjects referred to in the article. This document is in the possession of the correspondence author.

Conflicts of Interest

Authors declare no conflict of interest regarding the present study.

Financial Disclosure

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