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A Rare Case Of Transverse Testicular Ectopy (With Sertoli-Cell-Only Syndrome) With Persistent Mullerian Duct Structures & A Comprehensive Review Of Literature

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Abstract:
In this article we will deal with two developmental anamolies that usually coexist in 20 to 49% [1] of cases.
One of the entities is Transverse Testicular Ectopy, also known as Crossed Testicular Ectopy, as the name suggests, is a condition in which both the testes descend through the same inguinal canal into the same hemi-scrotum. TTE is a rare case that occurs only 1 in 4 million patients [2].
The other entity is Persistent Mullerian Duct Syndrome, which is a rare form of male pseudo-hermaphroditism in which there are remnants of the Mullerian structures in an otherwise genotypically (46XY) and phenotypically normal male.
The first case of TTE was reported in 1886 by Lenhossek [3], and nearly 200 cases were documented since then in the literature. Inspite of this history, this condition is not well recognized by the clinicians, and nearly 65% of cases are diagnosed intraoperatively [4].
We present to you a case of TTE with PMDS operated at our institute, along with a comprehensive review of the literature of both the entities.

Keywords:*tte; transverse testicular ectopy; cte; crossed testicular ectopy; PMDS; PMD; persistantmullerain duct syndrome*

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I. Introduction:

Transverse Testicular Ectopy, as the name describes, is a condition in which the testes are located in a region other than where they are designated to be. It is not completely similar to undescended testis on the grounds that both the testes are located on the same side:

1. Either both may be undescended – completely empty scrotum or
2. One of the testes enters the hemi-scrotum, and the other testis is also on the same side, presenting as an inguinal hernia (a majority of the cases), i.e., ipsilateral empty scrotum and contralateral inguinal hernia.

So far in the literature, the following names were used to describe this condition –

a. Crossed testicular ectopia
b. Testicular pseudo-duplication
c. Unilateral double testis
d. Transverse aberrant testicular maldescent

Coming to the PMD – usually during the development of a male child, the Paramesonephric ducts,aka. Mullerian ducts are regressed in response to the Anti-Mullerian Hormone, aka. Mullerian Inhibiting Substance secreted by the Sertoli cells of the testes. Due to some abnormalities which are described later in the course of this article, the Paramesonephric ducts do not regress and develop into adulthood. The remnants could be all or a part of the Uterus, Fallopian tubes, and upper part of the Vagina. This condition is described in the literature as 'hernia uteri inguinalis' by Nelson in 1939 [5] – which gives us an idea that the uterus (a remnant of Mullerian duct) presents as an inguinal hernia.

Apart from the above two entities, an issue with the vas deferens has also been identified in the present case – both the vasa are fused. When the vasa diferentia are fused, a difficulty is experienced during surgical dissection – careful dissection has to be done to prevent stripping off the blood supply.

Case Presentation:

A 25 year old male patient presented with left inguinal hernia since first noticed 5 years back which gradualy increased in since then. Classic features of hernia were present and there is no significant history related to the swelling. There is no history of suggestive of any causative factors for hernia (chronic cough, wt. lifiting etc.). No previous surgical history. He has been married for 3 years without any children – this history must have been probed, but wasn't probed into much.

On examination, the general condition of the patient is normal, secondary sexual characters appeared normal.

After proper consent, the abdomen of the patient is exposed from Nipple to mid thigh in a well lit room and examined. Hernia (of size 10*12 cms, pyriform shaped) was noted in the left inguino scrotal region with visible and palpable cough impulse. The scrotum did not appear full. A diagnosis of Left indirect inguinal hernia was made and the content was assumed to be omentum as it was more firm than soft and elastic, and no bowel sounds were heard over the swelling.

Ultrasound abdomen revealed the content to be omentum, bowel and probably an undescended testis. The condition was explained to the patient and proper consent was taken.

His preanaesthetic work up and check up revealed no abnormalities and the patient was given fitness for surgery. He was prepped for aExploration and proceed further under spinal anaethesia.

Intra-operatively:

After the sac is opened, a single globular swelling of size 3*4 cms with smooth surface and with a pear shaped appendage at one side (probably epididymis) and a long non pulsatile tubular structure with a diameter of about 1cm extending from the narrow end of the appendage to inside the abdomen along with other tubular pulsatile structure in its close proximity – corresponding to the testis, epidydimis, ipsilateral vas and probably contralateral vas respectively.

The vas is followed inside with minimal traction on the testis brought outside the abdomen. The bowel loops that protruded outside the sac are carefully pushed inside. The other end of the tubular structure connected to a swelling that is similar in consistency with the first swelling – the contralateral testis.

The other testiswas also carefully delivered out of the abdomen. A long tubular structure is found connecting both the swellings with a small dilation in the middle – (probably premnant of Mullerian duct)alongwith two separate pulsatile tubular structures extending into the abdomen (probably the blood supply)

The observation of the hernial content was suggetive of some anatomical anomaly related to testicular descent with additional mass of tissue connecting both the testes. (Transverse Testicular Ectopy with Persistant Mullerian duct syndrome). Both the testes though have a blood supply appeared atrophic and the vas deferens of both sides were fused and embedded together with the additional tissue mass. Mobilization of testes and vas by meticulous dissection seemed to be difficult.

As the patient was conscious, a brief but informative explanation of his condition was given, and no promising assurance was given related to to restoring fertility as performing an orchidopexy seemed to be technically demanding and also both the testes appeared to be atrophic (smaller than the expected size at that age).

Further the risk of developing malignancy from the testes as well as remnants of Mullerian ducts is explained.

After the options available were give to the patient and his wife, they refused orchidopexy, and microTESE was not available at our institute, after proper consent from the patient and his wife the entire structure – both the testes, fused vasa deferentia(bilateral orchidectomy) and the excision of the additional mass was done and sent for histopathological examination.

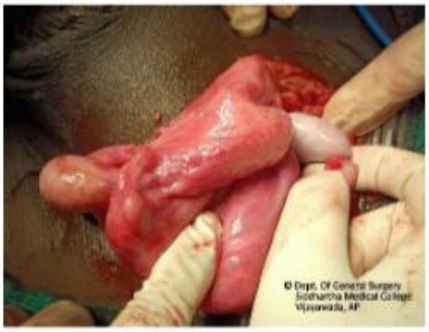

Figure 1 - Intraoperative Photograph

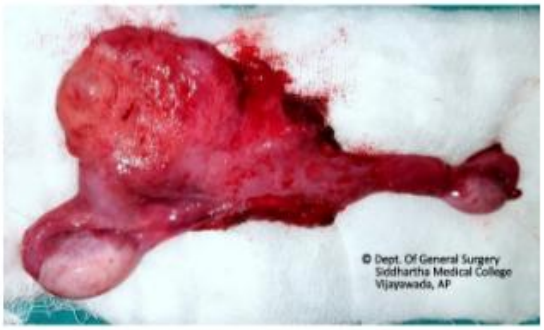

Figure 2 – Specimen excised – both testes and a central mass

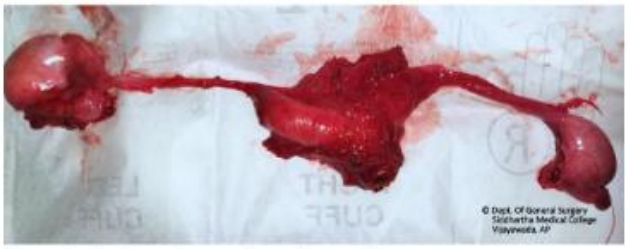

Figure 3 – Specimen (excess tissue dissected)

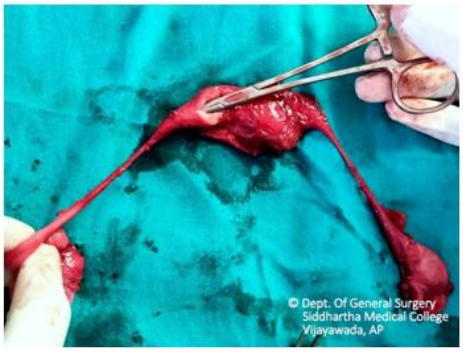

Figure 4– Exploring the Specimen

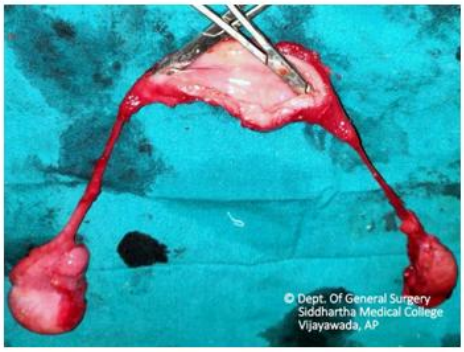

Figure 5– Central tubular structute opened to confirm the communication

Histopathology:

1. Both the testes were **devoid of germ cells** – **Sertoli-cell-only syndrome**. Semeniferous tubules with empty lumens were present. No evidence of malignancy was found – this was perhaps because of long standing cryptorchidism which led to destruction of germ cells. The patient could have not been fertile even if orchidopexy was performed because of the complete absence of germ cells.

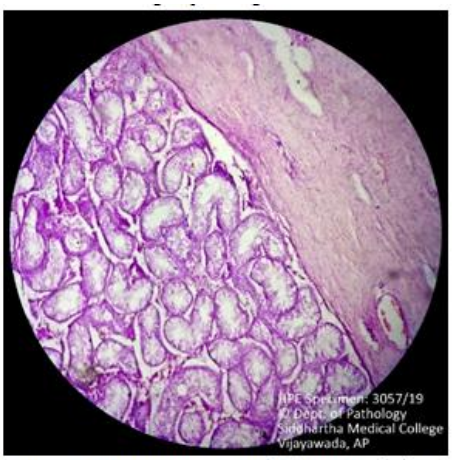

Figure 6– Microscopic view of testis part of the Specimen

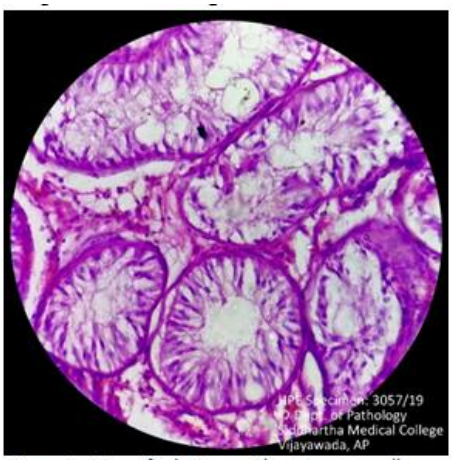

Figure 7– Magnified view – Absent germ cells

2. The vas of both the sides appeared to be normal.
3. The intervening tissue was **Uterus like** with endometrial lining, glands and myometrial tissue. A limited bit of tissue from this part showed features of endocervial canal – hence histopathological diagnosis of Persistent Mullerian Duct syndrome was made.

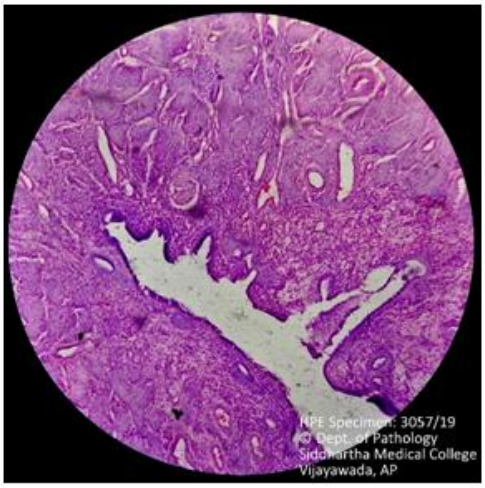

Figure 8– Microscopic view of central tubular structure of specimen – resembles Uterus

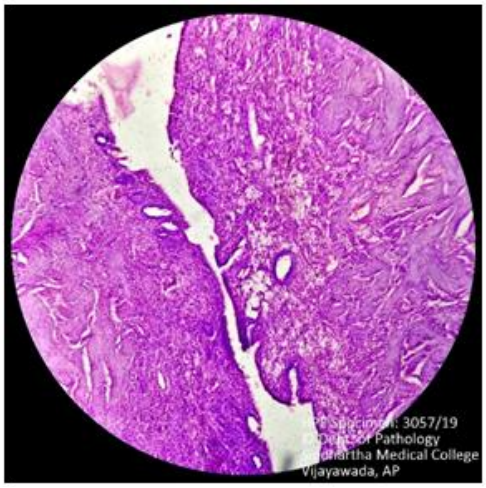

Figure 9– Magnified view demonstrating glands

A diagnosis of TTE with Sertoli-cell-only syndrome along with PersistantMullerain Duct syndrome was made after receiving the histopathological report.

II. Discussion:

a. Etiopathogenesis:

The exact etiology of TTE and PMDS is unknown, but some hypotheses have been proposed:

For TTE:
Josso N. et al. [6] proposed that anatomical factors – defective implantation, rupture or tearing of gubernaculum, obstruction of internal ring, development of adhesion of testes to adjacent structures (like remnants of Mullerian ducts), late closure of umbilical ring, etc. may be causative or inducible factors for TTE;
There is another possible theory suggested by Clarnette TD et al. [7] that states that the two testes might have taken origin from the same genital ridge (in addition to the anatomical factors mentioned above).

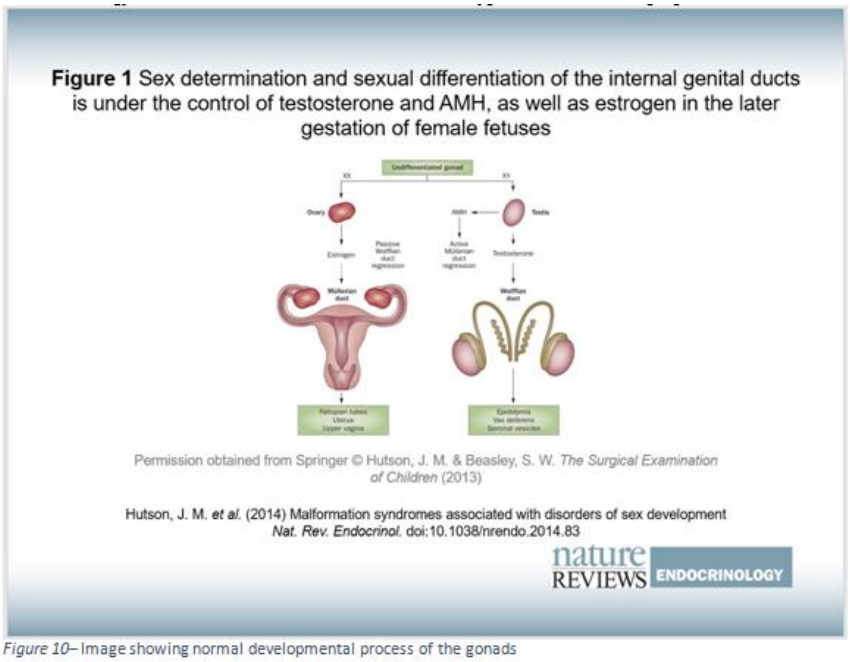

Figure 10– Image showing normal developmental process of the gonads

For PMDS:
Beginning at 7 weeks of age,Sertoli cells release Anti Mullerian Hormone (AMH), which leads to regression of Mullerian ducts; in PMDS, there is a defect in either AMH or AMH Receptor, which leads to the presence of Mullerian structures in otherwise normal male [8] – Josso N. Belville et. al.
Renuka et al. [9] in 2011 documented the following probable causes: failure of synthesis or release of MIH, failure of an organ to respond to MIH (may be due to receptor defects), a defect in the timing of the release of MIH.

For fused vas:
Probably the reason may be single vas originated from a common mesonephric duct, or from two separate counterparts fused early during the development when one testis crosses over to the opposite side [10].

Normally, the Leydig cells secrete testosterone, which has a local effect on mesonephric (Wolffian) ducts and helps them differentiate into the epididymis, vas deferens, and seminal vesicles, whereas dihydrotestosterone help in the formation of urogenital sinus and external genitalia. Another important function of AMH is to initiate testicular descent, principally by its postulated regulatory control over the gubernaculum testis [11] – Robby SJ et. al. These two functions can possibly explain the coexistence of TTE and PMD.

b. Classification:
Classification of **TTE**:
The first classification documented dates to 1967 by C. G. Thevathasan [12] but it is replaced by the present classification system byGauderer et al. in 1982 [13]
a. Type I – presents as hernia only (40 to 50%)
b. Type II – Hernia + PMD (30%)
c. Type III – Hernia + PMD + associated defects in Urogenital system (13 to 20%)
Associated abnormalities other than PMD include hypospadias, pseudo-hermaphroditism, bifid scrotum, seminal vesicle aplasia/hypoplasia, renal anomalies, and seminal vesicle cysts
This classification is based on the associated developmental abnormalities and is? Practically useful, but this classification system does not discriminate between a solitary and two distinct vasa deferentia, which may radically affect the therapeutic approach applied [10].

Classification of **PMDS**:
Three possible types of PMD are documented in the literature.
The types are:
a. Male type, one testis is usually found within the scrotum; the uterus and ipsilateral fallopian tube are either in the inguinal canal or can be brought into it by gentle traction on the presenting testis
b. In some cases, the contralateral testis and tube are also in the hernial sac; transverse testicular ectopia can also occur
c. The Female type is characterized by bilateral cryptorchidism with testes embedded in the broad ligaments in an ovarian position with respect to the uterus, which is fixed in the pelvis
Shabnam Azar et. al. [14] documented the types in order of their prevalence they studied.

c. *Presentation*:
This is the actual area of clinical interest.

The presenting complaint depends on the age of presentation. If presented in the pediatric age group, the presenting complaint would be an undescended testis (empty scrotum) with or without an inguinal hernia on the contralateral side. The mean age of presentation in the pediatric age group was documented as 8.2 years [15]. To make a diagnosis, a high degree of clinical suspicion is required.

Next, if the patient presents to the hospital a little later (the 20s to 40s), the main presenting complaint would be an inguinal hernia,and infertility can also be the presenting complaint.

If the patient still delays coming to the hospital (age 50's), the presenting complaint would probably be a mass per abdomen, which is increasing in size – which gives us a hint that there might be a malignant change in the undescended testis or the Mullerian remnants.

Though the patient is infertile, the secondary sexual characters are normal, and there are no problems related to erection as well. This is probably because the testosterone levels (Leydig cells) are normal, the actual pathological abnormality would be the damage to the germ cells attributed to cryptorchidism,or there might be abnormalities in the vas, seminal vesicles, or the pathway (sperm collection and transit) due to MIH and PMD related pathology – in Type III TTE the structures like seminal vesicles are not formed properly, and hypospadias may also contribute to infertility.

The germ cell development may not be entirely hampered; it may range from a complete absence of germ cells – as in our case, Sertoli-cell-only syndrome;arrestat some phase of development, or sometimes the patient might not have a complaint of infertility because of the presence of normal germ cells.

Type III presentation was reported by Chrysovalantis et al. [10] – a single fused vas deferens and bilateral hypoplastic seminal vesicles. The patient had a low ejaculate volume, azoospermia, and elevated FSH levels (26.22 mIU/mL).

Another interesting presentation was documented by Smith Harrison et al. [16] – the patient was a 27 years old male with status post orchidopexy at 18 months of age, presented with intermittent (cyclical), painless gross hematuria and hematospermia. On investigation, it was found that there was a uni-cornuate uterus with an endocervical canal that terminated in the right seminal vesicle with patent communication. The hematuria and hematospermia corresponded to the shedding of endometrium in the remnant of PMD.

A case reported by Renuka et al. [9] in 2011 was that of a 25-year-old male patient, married and had two children, presented with a complaint of lump abdomen sincethree years gradually progressive in size, but rapidly increased in size in the last four months – suggestive of some malignant pathology.

Of all the above presentations, the most common presentation would be unilateral cryptorchidism and contralateral inguinal hernia [17].

This is the spectrum of possible clinical presentations documented in the literature so far. A high index of clinical suspicion is necessary to make the diagnosis of TTE.

d. *Scope of Investigations*:

A preoperative diagnosis allows the assessment of the length of spermatic vessels and the anatomy of two vasa deferentia before committing to a particular mode of orchidopexy [1]; hence proper preoperative evaluation is essential.

The investigations that can be done are:

1. Ultrasound and Doppler imaging – to know the status of undescended testis, its vascularity, and any associated structures
2. Semen analysis
3. Hormonal Assay
4. CECT Abdomen
5. MRI
6. MR Venography
7. Retrograde urethro-cystogram – to show any structures of Mullerian remnants connected with patency to the seminal vesicle/prostate.
8. Karyotyping
9. Prader's Orchidometer – to assess the development of testes
10. Diagnostic Laparoscopy
11. Gonadal biopsy

Laparoscopy is the gold standard [18], [19], and has the advantage of biopsy of the testis [1] and Mullerian derivatives, if any.

Ultrasound has a sensitivity of 82% to 88% in the detection of an impalpable testis – Lam WW et al. [20].

An approach of a line of the workup for an impalpable testis, as recommended by Lam et al. [20], involves USG as the first line of imaging modality due to its easy availability and lack of any ionizing radiation. If USG findings are negative, MRI is recommended as the next line of investigation (comparable sensitivity of 84%) followed by MRV and third line of investigation. MRV has a sensitivity of 100% with the main objective of localizing the testicular vessels instead of testicular tissue.

e. *Histopathology and Malignant transformation / Malignant degeneration*:

One of the factors advocating surgical management is the risk of malignant transformation of the undescended testes or malignant degeneration of the PMD structures. A careful insight into this section guides us to make a proper decision.

Initially, let us know the possible histopathology reports when the excised specimen is studied. Mohammad M. Shalbay et al. [15] reported that biopsies of 84% of testes showed normal study, 11% has Sertoli-cell-only syndrome (as in this case report), and 5% showed atrophic glands.

Recently Eastham et al. [21] and Y. Zhu et al. [22] documented that it is uncertain whether TTE possesses and an independent risk factor for testicular malignancy, but surely it has been linked to both seminomatous& non-seminomatous germ cell tumors and teratomas.

Renuka et al. [19] documented two cases TTE with PMDS, both presented as lump abdomen (both without any history of infertility) – in one of the cases, aged 25 years, there was a large mass on the right side of the pelvis adherent to the pelvic organs and in other case aged 35 years a tumor was present in retroperitoneum

with para-aortic lymph nodes. Histopathology of both the surgical specimens showed features of classical seminoma and, additionally, in the first case, showed secondary deposits on omentum and internal iliac lymphnodes.

Huston JM et al. [23] documented that risk of testicular malignancy increases by 35 to 50 times with cryptorchidism.

Wood et al. [24] showed that the risk of malignancy of undescended testes is decreased if orchidopexy is performed before the age of 10 to 12 years.

As far as the testis component, the risk of malignancy is documented as above; moving to the remnants of Mullerian ducts, earlier reports argued that there was no absolute indication for the removal of Mullerian remnants since the risk of malignant degeneration has never been established [25], but this has changed in the course of time.

Mohammad M. Shalbay et al. [15] observed that, though all the Mullerian tissues that were removed were free of malignancy, it was found that these tissues were more vascular in older patients; this type of growth rather than involution is a warning sign regarding the possible complications and hence justifies excision.

However, a more recent report by Farikullah et al. in 2012 [26] has demonstrated malignant degeneration in Mullerian duct remnants – they concluded with 90% confidence that PMDS patients have a 3.1 to 8.4% risk of developing Mullerian malignancies – however they acknowledged that the accuracy of this value is limited since all cases of PMDS are diagnosed and not all Mullerian duct malignancies are reported.

Studies suggestive of malignant transformation of Mullerian structures are [block].

There are documented reports of embryonal cell carcinoma, seminoma, yolk sac tumors, and teratoma in patients with PMDS – Shinmura Y et al. [27].

From this section, we can understand that both the components undescended (ectopic) testes and remnants of Mullerian structures posses a potential risk of transforming into malignancy.

f. *Treatment Modalities*:

Goals of therapy:

1. Manage infertility
2. Avoid malignancy

The primary goal of treating the patients with TTE with or without PMDS is to spare/restore the fertility because ectopic testes have an increased risk of infertility, and also have a 5 to 10-fold increases lifetime risk of testicular neoplasia [28], and the second task is to excise the remnants of Mullerian ducts (if any) as they possess a potential risk of malignant degeneration.

Structures to be addressed during surgery:

1. the testes
2. the vasa deferentia
3. the remnants of Mullerian ducts

Trans-septal orchidopexy or Fowler-Stephens orchidopexy are the surgical procedures that can be undertaken based upon the level of testes. Avoid excessive dissection and mobilization of the closely placed testes – as over mobilization or dissection carries the risk of stretching and stripping off of blood supply to the testes.

Further, complications arise when remnants of Mullerian ducts exist; because the vasa deferentia, which are the conduit for the sperms, are embedded in the broad ligament or exist in very close proximity to these remnant structures, thus resulting in difficult dissection while attempting the mobilize the vasa and excise the remnant structures.The blood supply to these vasa might be stripped off during this process, thus resulting in infertility. Hence excessive dissection without a clear picture of the vascular supply is not advisable.

Coming to the PMDS, Mullerian structures should be removed where ever possible to avoid the risk of malignant transformation [15]. If the decision is made to remove the Mullerian duct remnants, extra precaution should be taken to ensure that there is no vascular compromise of vas deferens, which has a blood supply close to Mullerian derivatives [28].

Shalby et al.[15] stated that in a case where there is a risk of stripping the testes off their vascular supply during the excision of PMDS due to short spermatic vessels in patients presented with neglected cryptorchidism, it is better to leave the PMD and have a long term follow up along with an attempt to preserve fertility than risking the loss of fertility for excising the PMDS.

It is also documented that the preservation of Mullerian derivatives is incompatible with successful orchidopexies because, with sexual maturation, the uterus may become hypertrophic and cause discomfort or may present as a mass whose origin is unknown Clemente et al. [29].

Thus, the overall funda related to remnants of Mullerian ducts is to excise them where ever possible but not at the cost of losing the fertility; subsequently, after the patient completes his family, he should be taken for surgery to excise the leftover PMDS later on.

In the experience of Smith et al. [16] with robotic surgery in an adult with a delayed diagnosis of PMDS, they stated that the visualization and precision provided by the robotic arms were critical to the difficult nerve-sparing surgery.
Chrysovalintis et al. [10] documented a case in which, due to the presence of fused vas deferens, reconstruction, and transseptal transfer of ectopic testis to the contralateral side were technically difficult because the fused vas would restrict the testicular dissection and mobilization.
Robby SJ et al. [11] – Orchidectomy in testes that cannot be mobilized or have undergone a malignant change (our present case).

Further, Microsurgical Testicular Sperm Extraction (microTESE) is better done for preserving the sperm in all possible situations where the risk of infertility due to the procedure is anticipated (cases of rigorous dissection where there is a risk of compromise of blood supply) – approx. 6% of the cases the complication encountered intraoperatively was inadvertent cut of vas during primary orchidopexy [15].

A small note of signs of PMDS as observed in laparoscopy [15] – most common initial finding was an abnormally raised peritonea, fold tenting over the bladder, gubernacular seemedto be joining in the midline, and the testes in 90% of the cases were medical and below the level of external iliac vessels.

g. ***Post-op care and follow up*****:**

Routine post-op care would suffice. If orchidopexy and fertility-sparing procedures are performed, the patient is to be closely monitored – immediate post-op for arterial doppler to assess the supply to the testes and in the long run for semen analysis.

Prepubertal patients with PMDS had testicular volumes confirming to the reference values for normal testes [30], and their testes showed better compensatory growth than those of adult patients after orchidopexy, which may support the theory that testicular damage is due to neglected cryptorchidism rather than a part of the syndrome [15].

Proper guidelines are yet to be established for the follow up of these patients.

III. CONCLUSION

Always have a high degree of clinical suspicion when examining any case presenting with an inguinal hernia on one side and cryptorchidism on the other side. A careful probing into the marital and family history may provide a clue regarding infertility – the patient may not present with infertility and, in some cases, may not confess the condition unless questioned in privacy and confidence.

Proper preoperative assessment with the multitude of investigations available apart from the semen analysis – aiming to understand the underlying anatomy of testes, vasa deferentia& remnants of Mullerian ducts (if any) – this helps in a proper plan for the surgical procedure and prevents any sudden surprises intraoperatively.

As previously discussed, the priority is fertility over prevention on malignancy – based on the age group, family status, and, more importantly, the decision of the patient.

MicroTESEwith cryopreservation can always be kept as a safe standby for any form of surgical procedure planned and any unplanned intraoperative complications.

Persistent Mullerian Duct structures are removed in wherever possible unless there is a risk of compromising the fertility (due to extensive dissection that might be required to excise them) – because these remnants are observed to grow rather than involute as the patient ages and hence possess a definite risk of malignancy in the later years. Orchidectomy is the last retort (after a proper consent from the patient and family) when there is a risk of losing the testes during the mobilization as well as in situations where the testes appear unviable, atrophic, or malignant.

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