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Subtle Fallopian Tube Abnormalities in Infertility: Anatomy, Functional Implications, and Diagnostic Evaluation

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ABSTRACT

Subtle fallopian tube abnormalities refer to minor, non-obstructive morphological changes affecting the distal portion of the tube, including fimbrial agglutination, fimbrial phimosis, hydatids of Morgagni, tubal diverticula, accessory ostia, and accessory fallopian tubes. These abnormalities were historically considered normal anatomical variants without clinical significance; however, growing evidence suggests that they may negatively impact tubal function and fertility. Because these lesions do not cause complete tubal obstruction, they are frequently overlooked during conventional infertility investigations such as hysterosalpingography and routine laparoscopy, which primarily assess tubal patency.

Assessment of fallopian tube structure and function is a critical step in the infertility work-up because tubal factors contribute to 25–35 % of female infertility cases worldwide. Proper evaluation helps identify occlusion, peritubal adhesions, or mucosal damage that might impede fertilization or zygote transport. The diagnostic approach combines imaging, endoscopic, and functional techniques that range from non-invasive screening to direct visualization procedures.

Keywords: Fallopian tubes; Infertility; Laparoscopy; Hysterosalpingography; Tubal abnormalities

INTRODUCTION

Infertility is recognized as a disease and a condition of the reproductive system, characterized by the inability to achieve a successful pregnancy. This may be determined based on a patient's medical, sexual, and reproductive history, age, physical findings, diagnostic testing, or a combination of these factors [1].

Infertility can be classified as either primary or secondary. Primary infertility is a delay in becoming pregnant for a couple with no previous pregnancies. Secondary infertility is a delay in conception for a couple who had previously conceived and cannot establish subsequent clinical pregnancy, however, it's possible that the pregnancy didn't complete, either due to ectopic pregnancy or miscarriage. [2].

Tubal factors account for approximately 25% of infertility cases among women, making them one of the most common causes of female subfertility. So, tubal condition and wellness must be investigated thoroughly during infertility work up. There are multiple methods to evaluate tubal condition including hysterosalpingogram (HSG), hysterosalpingo-contrast sonography (HyCoSy), Saline infusion Sonohysterosalpingography(SIS) and laparoscopy [3].

Unexplained infertility is defined as the absence of a clear cause for a couple's inability to conceive despite at least 12 months of unprotected, regular sexual intercourse and normal findings in standard fertility evaluations. This condition affects up to 30% of infertile couples worldwide. Many clinicians question the accuracy of the term unexplained infertility, emphasizing that its use depends on the scope, quality, and interpretation of diagnostic tests applied [3].

The absence of identifiable abnormalities after these assessments does not necessarily indicate the absence of an underlying cause. Therefore, further in-depth investigations are recommended to detect subtle or complex causes of infertility [4].

ANATOMY AND DEVELOPMENT OF THE FALLOPIAN TUBES

The Fallopian tubes, classically referred to as the oviducts or uterine tubes, are paired, tubular, muscular structures situated within the superior margin of the broad ligament of the uterus (the mesosalpinx). Extending laterally from the uterine cornu toward the ovaries, each adult Fallopian tube measures approximately 10 to 12 cm in length and varies significantly in luminal caliber across its distinct anatomical divisions [5].

From medial to lateral, the Fallopian tube is anatomically partitioned into four well-defined segments:

Interstitial (Intramural) Segment: Traverses the thick muscular wall of the uterine cornu for approximately 1 cm. It exhibits the narrowest lumen, measuring 0.7 to 1.0 mm in diameter, and is encased directly by myometrial smooth muscle fibers. [5].

Isthmus: A narrow, thick-walled muscular segment spanning 2 to 3 cm in length with a luminal diameter of 1 to 2 mm. The thick circular smooth muscle layer of the isthmus functions as a physiological sphincter that regulates sperm ascent and delays blastocyst transit until uterine receptivity is optimal [5].

Ampulla: The longest and widest portion of the tube, measuring 5 to 8 cm in length with a luminal diameter expanding up to 1 cm. The ampulla is characterized by an extensive, highly folded mucosal architecture (plicae) and represents the primary site of human fertilization [5].

Infundibulum and Fimbriae: The trumpet-shaped distal extremity measuring 1 to 2 cm, opening into the peritoneal cavity via the abdominal ostium. It terminates in 20 to 30 finger-like mucosal projections termed fimbriae. The longest single fimbria, the fimbria ovarica, is structurally tethered to the superior pole of the ovary to guide ovulated oocytes into the tubal lumen [5].

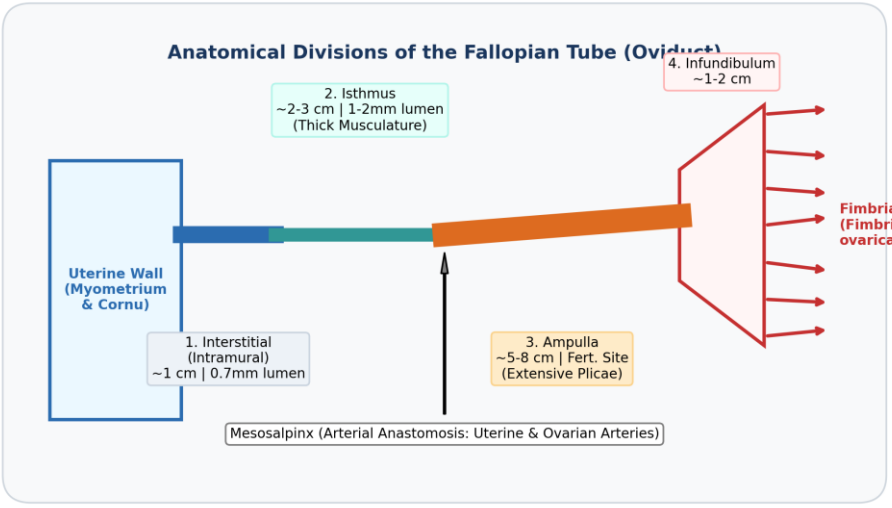


Figure 1: Schematic anatomical structure of the Fallopian tube detailing the four main segments (interstitial, isthmus, ampulla, and infundibulum/fimbriae) along with luminal caliber and vascular attachments. (Han et al., 2023).

Histological Architecture

The tubal wall consists of three concentric tissue layers: an inner mucosa (endosalpinx), a middle muscularis (myosalpinx), and an outer serosa (perisalpinx):

Endosalpinx (Mucosa): Composed of a simple columnar epithelium lying on a delicate lamina propria. The endosalpinx forms intricate longitudinal mucosal folds (plicae), which are most complex in the ampulla and simple in the isthmus. The epithelium contains two major functional cell types: ciliated columnar cells and non-ciliated secretory cells (peg cells), alongside resting reserve stem cells.

Myosalpinx (Muscularis): Arranged in an inner circular and outer longitudinal layer of smooth muscle. In the interstitial and isthmic regions, an innermost longitudinal layer is also present, facilitating complex peristaltic contractions.

Perisalpinx (Serosa): An outer visceral peritoneal layer composed of mesothelial cells resting on loose connective tissue containing abundant blood vessels, lymphatics, and nerve fibers. [6]

Embryological Development

The development of the female reproductive tract is a tightly regulated morphogenetic process occurring between the 4th and 20th weeks of human embryogenesis. The Fallopian tubes derive specifically from the cranial, non-fused portions of the paired paramesonephric (Müllerian) ducts [7].

During the 6th week of development, in an embryo lacking a Y chromosome and the sex-determining region Y (SRY) gene, the paramesonephric duct arises as a longitudinal invagination of the coelomic epithelium on the anterolateral surface of the urogenital ridge. The craniolateral funnel-shaped opening of this invagination remains in open communication with the coelomic cavity, establishing the future abdominal ostium and fimbriae of the Fallopian tube. The paired Müllerian ducts extend caudally, running parallel and lateral to the mesonephric (Wolffian) ducts. In the absence of Anti-Müllerian Hormone (AMH, also known as Müllerian Inhibiting Substance [MIS]), which is produced exclusively by Sertoli cells in male embryos, the Müllerian ducts persist and undergo robust differentiation. The cranial vertical portion and middle horizontal portion of each duct form the ipsilateral Fallopian tube, whereas the caudal vertical portions fuse in the midline to form the uterovaginal primordium (giving rise to the uterine body, cervix, and upper two-thirds of the vagina). [8].

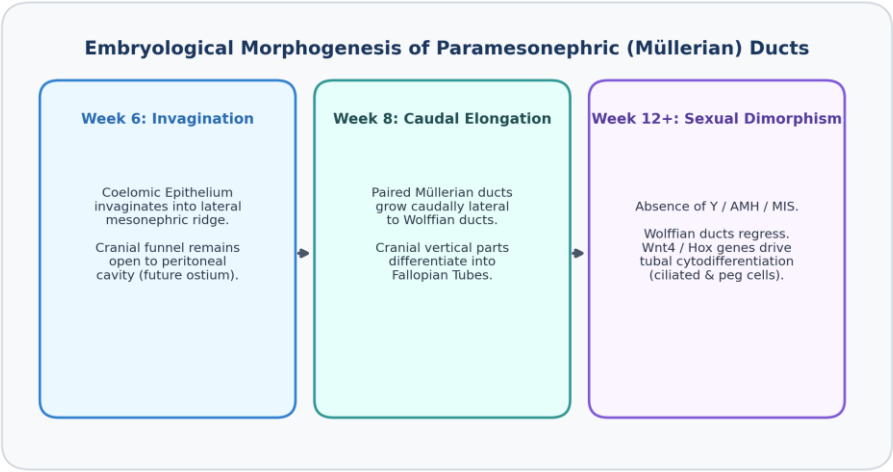


Figure 2: Chronological sequence of paramesonephric (Müllerian) duct morphogenesis, detailing coelomic invagination, caudal elongation, and hormone-mediated female differentiation. (Libretti & Aeddula, 2023).

Molecular Signaling in Tubal Development

The execution of tubal morphogenesis requires orchestrated expression of key transcriptional factors and signaling molecules:

WNT4 and WNT7a: WNT4 is essential for initial Müllerian duct formation and maintenance; its knockout leads to total absence of Müllerian structures. WNT7a regulates axial patterning along the anteroposterior axis of the duct and is required for smooth muscle differentiation in the myosalpinx.

HOX Genes (HOXA9, HOXA10, HOXA11, HOXA13): The regional specificity of the Müllerian duct is directed by differential HOX gene expression. HOXA9 is specifically expressed in the cranial segment destined to become the Fallopian tube, directing tubal epithelium cytodifferentiation. HOXA10 and HOXA11 specify the uterus, while HOXA13 dictates upper vaginal differentiation.

PAX2 and Lim1 (LHX1): Essential transcription factors governing ductal elongation and epithelial cell survival during early mesodermal migration. [9]

PHYSIOLOGY OF THE OVIDUCT

The Fallopian tube is not a passive conduit, but a highly dynamic biological microenvironment that actively supports gamete maturation, fertilization, and early embryo development.

Transport of oocytes, spermatozoa, and embryos depends on a synergistic interaction between mucosal ciliary beat and myosalpingeal smooth muscle contractions:

Ciliary Function: Ciliated cells possess approximately 200-300 cilia per cell, beating in a coordinated, metachronous wave directed toward the uterine cavity. Estrogen increases ciliary beat frequency (CBF) and promotes ciliogenesis during the follicular phase, reaching a peak frequency of 8 to 10 Hz around ovulation. Progesterone depresses CBF during the luteal phase.

Smooth Muscle Peristalsis: Myosalpingeal contractions are controlled by autonomic inputs and local paracrine factors (prostaglandins E2 and F2α). During the pre-ovulatory phase, high estrogen levels increase myometrial and myosalpingeal contractility to assist sperm transport toward the ampulla. Post-ovulation, progesterone reduces overall contractility, allowing smooth, controlled movement of the cleavage-stage embryo [10].

Non-ciliated peg cells actively secrete oviductal fluid, a complex biochemical milieu essential for gamete survival and pre-implantation embryo development. The fluid is produced via selective transudation from blood capillaries combined with active epithelial secretion. Key constituents include:

Oviduct-Specific Glycoprotein 1 (OVGP1): A major estrogen-dependent chaperone protein that binds to the zona pellucida and sperm membrane, enhancing sperm capacitation, acrosome reaction, fertilization rate, and zona hardening to prevent polyspermy.

Energy Substrates: High concentrations of pyruvate and lactate are present in the ampullary fluid, serving as primary metabolic substrates for the unfertilized oocyte and early cleavage-stage blastomeres before embryonic genome activation.

Antioxidants and Immunomodulators: Contains superoxide dismutase, catalase, and complement regulatory proteins that protect gametes from reactive oxygen species (ROS) and maternal immune destruction. [11].

CONGENITAL ANOMALIES OF THE FALLOPIAN TUBES

Congenital anomalies of the Fallopian tubes result from disruptions in embryonic paramesonephric duct formation, elongation, fusion, or canalization. While isolated tubal anomalies are rare (occurring in 0.1% to 0.5% of the general population), they frequently co-occur with uterine anomalies (Class I–VII AFS/ASRM classifications) and renal tract malformations (due to the shared development of the mesonephric ridge) [5].

According to the American Society for Reproductive Medicine (ASRM) and the European Society of Human Reproduction and Embryology (ESHRE/ESGE) classifications, congenital tubal defects are categorized into four major pathological patterns [12].

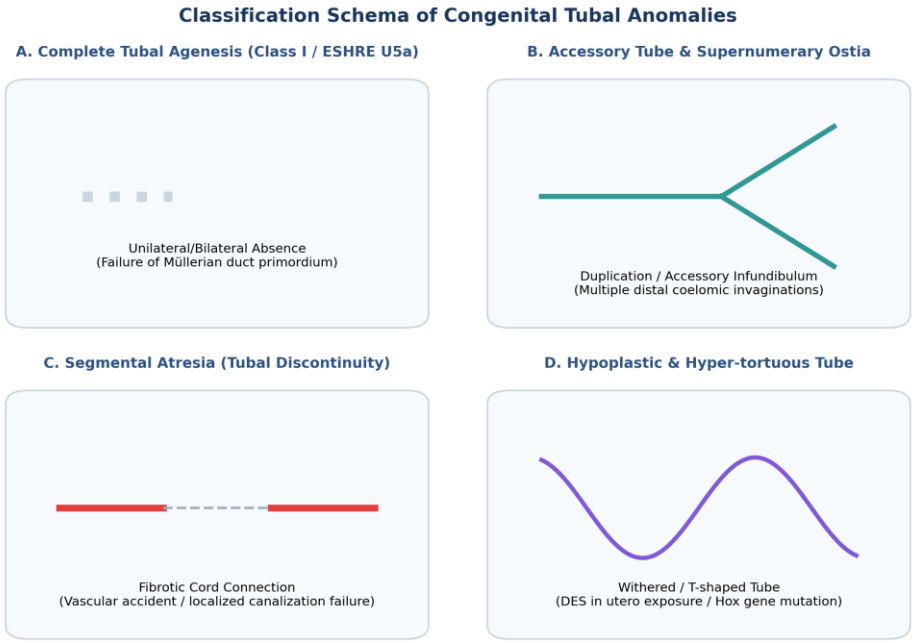


Figure 3: Schematic representation of major tubal congenital anomalies: A. Complete tubal agenesis; B. Accessory Fallopian tube and supernumerary ostia; C. Segmental tubal atresia; D. Hypoplastic and hyper-tortuous tube. (Csöbönyeiová et al., 2022).

Tubal agenesis involves the total absence of one or both Fallopian tubes. Unilateral agenesis (Class Ia / ESHRE U5a) is commonly associated with unicornuate uterus (Class II) and ipsilateral renal agenesis (in up to 40% of cases) due to failure of embryonic Müllerian duct primordium initiation. Bilateral tubal agenesis is extremely rare and typically seen in Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome, where both Müllerian ducts fail to develop. [12].

Characterized by the congenital absence of a segment of the Fallopian tube, leaving proximal and distal tubal ends connected by a thin fibrous cord or completely separated. Segmental atresia arises from localized canalization failure or early fetal vascular accidents (e.g., partial adnexal torsion in utero) [9]

Duplication defects range from supernumerary fimbriated ostia on an otherwise single tube to complete duplication of the Fallopian tube (accessory tube). Accessory ostia occur in up to 2% to 5% of infertile women and result from multiple distal coelomic invaginations along the embryonic mesonephric ridge [5].

Hypoplastic tubes are abnormally short, thin, or withered structures with poorly developed muscular layers and sparse ciliated epithelium. Hyper-tortuous (coiled) tubes demonstrate extreme convolution. Historically, severe tubal hypoplasia and T-shaped uterine cavity anomalies were linked to in utero exposure to diethylstilbestrol (DES), which disrupted WNT7a and HOXA expression [10].

Clinical and Reproductive Implications

Congenital tubal anomalies carry profound reproductive consequences:

Infertility: Agenesis and segmental atresia create absolute mechanical blockages preventing sperm-oocyte interaction. Accessory ostia can cause 'gamete entrapment', where the ovulated cumulus-oocyte complex enters a blind-ending accessory infundibulum.

Ectopic Pregnancy: Impaired ciliary motility or structural narrowing in hypoplastic, tortuous, or partially atretic tubes significantly delays blastocyst transit. This leads to premature blastocyst hatching and implantation within the tubal mucosa [13].

Surgical & ART Management: Corrective microsurgery (e.g., tubal re-anastomosis) may be performed for short segmental atresia, but in vitro fertilization and embryo transfer (IVF-ET) remains the gold-standard therapeutic intervention for severe congenital tubal factor infertility [10].

TUBAL FACTORS OF INFERTILITY

Tubal factor infertility occurs when the structure or function of the fallopian tubes is compromised, preventing successful fertilization or transport of the embryo to the uterine cavity. Normally, the Fallopian tubes capture the ovulated oocyte via the fimbrial end, permit meeting with sperm, enable fertilization (usually in the ampulla), and then transport the zygote toward the uterus by coordinated ciliary action and peristalsis [14].

Common pathophysiologic mechanisms include tubal obstruction (complete or partial), altered tubal motility, damaged mucosal lining (loss of cilia), altered intraluminal fluid environment, and peri-tubal adhesions that distort the anatomy. For example, inflammation from infection can destroy ciliated epithelial cells, leading to impaired transport of gametes and embryos. Furthermore, when the distal tube is occluded or distended (as in hydrosalpinx), fluid may reflux into the uterine cavity and create a toxic environment for embryo implantation, reducing fertility and success rates in assisted reproductive techniques [14].

Causes and Risk Factors

Pelvic Inflammatory Disease (PID): Often resulting from sexually transmitted infections (e.g., Chlamydia trachomatis, Neisseria gonorrhoeae), PID is one of the most prevalent causes of tubal damage leading to infertility. Infection can lead to salpingitis, scarring, occlusion and ciliary loss.

Endometriosis: Endometrial tissue implantation in and around the tubes and pelvis can cause adhesions, distortion, and blockade of tubal fimbriae.

Prior pelvic or abdominal surgery: Surgeries such as tubal ligation/reversal, ectopic pregnancy surgery, appendectomy, or pelvic operations can lead to adhesions or tubal trauma that impairs function.

Hydrosalpinx and tubal occlusion: When a tube is blocked and distended with fluid, it not only blocks transport but may also have a toxic effect on embryo implantation.

Congenital tubal anomalies: Less common, but anatomical variations (e.g., salpingitis isthmica nodosa, tubal spasm, or developmental abnormalities) may contribute to tubal infertility.

Subtle Fallopian tube abnormalities may contribute to infertility such as fimbrial phimosis, accessory tubal ostia...etc [15].

Empirical studies identify multiple risk factors that increase the likelihood of tubal factor infertility:

History of multiple PID episodes correlates with higher risk of tubal occlusion. For example, after three or more episodes of salpingitis the risk of infertility may rise to 54–75%.

Previous ectopic pregnancy, previous tubal surgery or sterilization, documented tubal pathology, and prior genital infections significantly elevate risk (relative risks vary).

Low socioeconomic status, younger age at first infection, use of intrauterine devices in certain settings, sexual behaviour (e.g., multiple partners), and inadequate access to health care have been associated with increased tubal damage.

Tubal microbiota disruption is an emerging risk factor; research suggests that changes in tubal microbial environment may influence tubal epithelial health and patency. [14].

EVALUATION OF FALLOPIAN TUBE STATUS

Assessment of fallopian tube structure and function is a critical step in the infertility work-up because tubal factors contribute to 25–35 % of female infertility cases worldwide. Proper evaluation helps identify occlusion, peritubal adhesions, or mucosal damage that might impede fertilization or zygote transport. The diagnostic

approach combines imaging, endoscopic, and functional techniques that range from non-invasive screening to direct visualization procedures [14].

Hysterosalpingography

Hysterosalpingography is a radiographic technique in which contrast medium is injected through the cervix to outline the uterine cavity and fallopian tubes under fluoroscopy. It provides information about tubal patency and uterine contour.

Advantages: Widely available, inexpensive, outpatient procedure.

Limitations: Cannot evaluate peritubal adhesions or differentiate between tubal spasm and true occlusion. It may cause discomfort and allergic reactions to contrast media.

Interpretation: Free spill of contrast into the peritoneal cavity indicates patent tubes, while absence of spill suggests blockage at the proximal, mid, or distal segment. Recent studies also suggest that oil-based contrast may transiently increase pregnancy rates by flushing debris from the tubes [16].

Sonohysterography and HyCoSy

HyCoSy uses ultrasound with echogenic contrast agents (foam or microbubbles) to assess tubal patency and surrounding structures.

Advantages: Avoids radiation exposure, provides concurrent evaluation of the uterus and ovaries, and allows dynamic assessment of peritubal environment.

Limitations: Operator dependent and may have reduced accuracy in cases of partial obstruction or severe adhesions. Color Doppler HyCoSy and three-dimensional (3D) HyCoSy have improved visualization, sensitivity (up to 95 %), and specificity (about 90 %) compared with conventional HSG. [17].

Transvaginal Ultrasound

Transvaginal ultrasound provides anatomic details of the fallopian tubes, especially when hydrosalpinx, tubo-ovarian masses, or cysts are present. It can reveal thickened tubal walls, dilatation, or peritubal adhesions indirectly. 3D ultrasound offers better visualization of tubal architecture, while high-resolution Doppler imaging may indicate vascular changes associated with chronic inflammation or scarring [18].

Laparoscopy and Chromopertubation

Laparoscopy remains the gold standard for assessing tubal pathology. During the procedure, diluted methylene blue dye is injected transcervically while the surgeon observes spillage through the fimbrial end.

Advantages: Allows direct inspection of the tubes, fimbriae, ovaries, and pelvis for endometriosis, adhesions, and hydrosalpinx.

Limitations: Invasive, requires anesthesia, and may not be suitable for all patients. Nevertheless, laparoscopy enables simultaneous therapeutic intervention, such as adhesiolysis or tubal reconstruction. [19].

Other Endoscopic Techniques

Hysteroscopy is primarily used to evaluate the uterine cavity but can help assess the tubal ostia at the uterine end. Combined hystero-laparoscopy provides a comprehensive assessment of both intrauterine and peritoneal factors contributing to infertility [18].

Salpingoscopy involves inserting a micro-endoscope into the fimbrial end during laparoscopy to inspect the mucosal folds and cilia.

Falloposcopy is a transcervical method that allows visualization of the entire tubal lumen using a flexible fiber-optic system. [18].

Functional and Adjunctive Assessments

Functional tubal patency can also be evaluated using radionuclide techniques, such as technetium-99m pertechnetate scintigraphy, which provides physiologic information about ciliary transport. Magnetic resonance imaging (MRI) with contrast enhancement has emerged as a non-ionizing alternative that delineates soft-tissue details and detects peritubal adhesions or endometriotic nodules with high sensitivity. [20].

Recent research investigates biomarkers such as cytokines (IL-6, TNF-α) and gene expression patterns in tubal fluid to assess inflammation, ciliary damage, and oxidative stress that may impair tubal function [20].

Diagnostic Approach

In clinical practice, evaluation is often stepwise beginning with non-invasive screening (HSG or HyCoSy) followed by confirmatory laparoscopy if abnormal or inconclusive results are obtained.

Normal findings: No obstruction or adhesions; patients may proceed to expectant management or other fertility treatments.

Abnormal findings: Hydrosalpinx, proximal/distal block, or adhesions may necessitate surgical correction or consideration of assisted reproductive technologies (ART) such as IVF. Evidence suggests that removing hydrosalpinx before IVF significantly improves implantation and pregnancy rates [21].

SUBTLE FALLOPIAN TUBE ABNORMALITIES

Subtle fallopian tube abnormalities refer to minor, non-obstructive morphological changes affecting the distal portion of the tube, including fimbrial agglutination, fimbrial phimosis, hydatids of Morgagni, tubal diverticula, accessory ostia, and accessory fallopian tubes. These abnormalities were historically considered normal anatomical variants without clinical significance; however, growing evidence suggests that they may negatively impact tubal function and fertility. Because these lesions do not cause complete tubal obstruction, they are frequently overlooked during conventional infertility investigations such as hysterosalpingography and routine laparoscopy, which primarily assess tubal patency. [22].

Fimbrial Phimosis: Fimbrial phimosis is characterized by narrowing of the fimbrial opening due to fibrosis or chronic inflammation, reducing the efficiency of ovum pickup [23].

Hydatids of Morgagni: Hydatids of Morgagni are benign paratubal cysts derived from Müllerian remnants, commonly located near the fimbrial end and potentially interfering with fimbrial mobility [22].

Tubal diverticula: Tubal diverticula are thin-walled small outpouchings of the tubal wall that may alter ciliary transport and peristalsis, negatively affecting gamete and embryo movement [24].

Tubal sacculation: Tubal sacculation involves a herniation of the tubal ampulla arising from deficient or absent the layer of tubal musculosa, often secondary to previous inflammation, and may compromise normal tubal transport without complete obstruction [24].

Accessory ostium: An accessory ostium is an additional opening in the fallopian tube, which can disrupt normal tubal physiology and has been associated with infertility and ectopic pregnancy [25]

Accessory Fallopian tube: Accessory fallopian tubes are rare congenital anomalies involving duplication or additional tubal segments, which may impair fertility by altering normal tubal anatomy and function [25]

Fimbrial agglutination: Fimbrial agglutination refers to partial adherence of the fimbriae, which may impair oocyte capture despite preserved tubal patency [22].

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