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3

Fallopian tube subtle pathology☆

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A B S T R A C T

The aim of the present review is to give a comprehensive overview of fallopian subtle lesions and suggest the impacts of these abnormalities on fertility. Tubal subtle variations, including tubal diverticula, Morgagni hydatids, accessory fallopian tube, accessory ostium of the fallopian tube, tubal phimosis, agglutination, and sacculation, have been described and cited as making significant contributions to infertility. This review summarizes characteristics of these subtle abnormalities and provides an update of recent knowledge of the diagnosis and management of these variations. We hope that the present contribution may help to bring more attention to the clinical field to recognize these abnormalities and consequently aid in improving fertility.

Tubal pathology is one of the major causes of female subfertility, with a prevalence of approximately 30%–35% in subfertile couples [1]. The objective of this review is to provide a detailed description of the tubal normal and abnormal anatomy and to document the effects of fallopian subtle defects on reproductive outcomes. Failure to recognize these subtle abnormalities may otherwise result in patients being diagnosed as idiopathically infertile, which in most instances would exclude many patients from being offered a possibly successful treatment through microsurgery.

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Part 1

Anatomy and embryology of the tube

Embryologic development

During the 5th to 6th weeks of gestation, the paired paramesonephric ducts form from the coelomic epithelium. The cranial portions of the paramesonephric ducts develop into the fallopian tubes, and the caudal portions fuse to form the uterus. Defects in canalization of the Müllerian system may result in duplication of the ostia of the oviducts. The cranial end of the fallopian tube remains open, thereby communicating with the peritoneal (coelomic) cavity, with multiple invaginations of the free edge forming the fimbriae. The caudal end of the fallopian tube communicates with the uterine cornua.

Congenital anomalies of the fallopian tubes include aplasia, hypoplasia, accessory ostia, paratubal cysts, accessory tubes, and congenital diverticula.

Normal anatomy

The fallopian tubes are paired, with tubular uterine appendix located in the superior edge of the broad ligament bilaterally. The fallopian tubes extend from the ovaries to the uterus and serve to transport the ovum.

The fallopian tubes are 10–12 cm in length and 1–4 mm in diameter [2]. In general, as traced laterally from the uterus, the fallopian tubes pass at first horizontally. The peritoneal reflection draping over the salpinges forms the mesosalpinx [3]. The tubes then laterally and posteriorly pass along the uterine pole of the ovary. The fallopian tubes extend anteriorly from the posterosuperior aspect of the uterine fundus and open into the peritoneal cavity, where the fimbriated end approaches the medial surface of the ovary [4]. Many variations in length, size, and direction exist and are usually considered as normal.

The fallopian tube is divided into four anatomic segments.

(1) Intramural—the uterine tube that passes through the muscular wall of the uterus. It is approximately 1 cm in length and varies from 0.2 mm to 0.4 mm in diameter [4]. The myosalpinx consists of an outer layer of longitudinal fibers, an intermediated circularly arranged layer, and an inner longitudinal layer. The endosalpinx is composed of four or five folds.
(2) Isthmus—the narrowest portion of the fallopian tube with fewer mucosal folds and a thick muscularis layer. It is 2–3 cm in length and varies from 0.2 mm to 2 mm in diameter. It is the densest muscular segment of the tube. The myosalpinx consists of inner and outer longitudinal layers and a middle circular layer. The inner longitudinal layer is defined at its uterine end.
(3) Ampulla—Closer to the ovary, the tube widens to form the ampullary segment, which is the usual site for fertilization. It accounts for over one half of its total length. The diameter of the lumen of the ampullar varies between 1 mm and 1 cm. The myosalpinx is thin and arranged into an outer longitudinal layer and inner circular layer. In the ampullar, the inner longitudinal muscle layer is present as discontinuous fibers. The endosalpinx is extensively folded; there are 6–8 primary folds, comprising numerous complex secondary and tertiary folds.
(4) Infundibulum—the funnel-shaped open end of the uterine tube with fimbriae, which opens into the peritoneal cavity by its abdominal ostium. It is approximately 3 mm in diameter [4]. The infundibulum has a thin layer of muscle consisting of an outer longitudinal and an inner circular layer. The internal surface of the infundibulum is occupied by the complex folds of mucosa, and these folds evert to form numerous irregular slender processes called fimbriae. The fimbria is suspended over the ovary by the fimbria ovarica and captures the ovum once it is released.

Very often, there is a normal cyst of 5 mm in diameter called appendix vesiculosa, which is attached to the fimbrial end. Sometimes, appendix vesiculosa is called as Morgagni hydatid (MH), whereas some authors call only the bigger paratubal cysts as MH (see part 2) (Fig. 1).

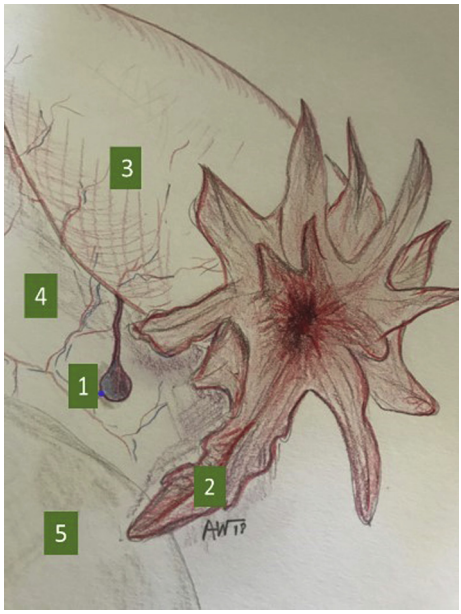

Fig. 1. Normal anatomy of the distal part of the tube. 1-Appendix vesiculosa; 2-fimbria ovarica; 3-ampulla; 4-mesosalpinx; 5-ovary.

The histological structure of the fallopian tube

The wall of the fallopian tube is composed of three layers: mucosa, muscularis, and serosa. Mucosal folds form finger-like projections called plicae that increase in complexity from the medial to the distal part of the tube. The plicae coalesce at the infundibulum, where they are contiguous with the fimbria. The epithelium is composed of secretory and ciliated cells, which produce tubular fluid and facilitate transport of gametes, respectively [5]. Ciliated epithelium and mucosal plicae propel the ovum toward the uterine cavity. In contrast to the ovum, fluid secreted by the epithelium progresses toward the fimbrial end of the tube and is released into the peritoneal cavity. Ciliated cells are found predominantly on the apex of the mucosal folds. Their reactivity is affected by a variety of hormonal and neuronal stimuli. Many pathological conditions associated with infertility and ectopic pregnancies have been shown either to destroy cilia or to reduce ciliary motion or both [6]. The muscular layer consists of longitudinal and circular muscular fibers, continuous with those of the uterus. Their thickness depends on the segment of the tube. The external layer or serosa is peritoneal. It surrounds the fallopian tube (except for the fimbrial end, which is the only organ with the ovary to *be stricto sensu* extra peritoneal) by a double fold or peritoneum and fuses inferiorly to form the mesosalpinx, the primary supporting mesentery of the tube. Between the leaves of the mesosalpinx, varying amounts of connective tissue are present along the nerves, vessels, and lymphatics.

Conclusion

The four anatomical segments of the tube can be identified depending on the anatomical position, thickness of the smooth muscle, complexity of mucosal folds, and cellular composition of the mucosa. These four anatomical segments are consistent with their different functions, i.e., ovum capture, sperm and embryo transport, retention, and release of early embryos for arrival at the uterine cavity for implantation.

Part 2

Tubal subtle abnormalities

The fallopian tubes play an important role in sperm transport, oocyte capture and transport, fertilization, and early embryo development. Abnormalities in any one of these functions cause defective transport of the oocyte and impaired fertilization, through alterations in tubal peristaltic or ciliary activity, which may affect one or both fallopian tubes. Some investigators reported that infertile women had significantly more tubal variations, including fimbrial pathology, than did their fertile counterparts [7].

Subtle variations in tubal anatomy, including tubal diverticula, MHs (paratubal cysts), accessory fallopian tube, accessory ostium of the fallopian tube and phimosis, agglutination, and sacculation, have been cited to make significant contribution to infertility (Fig. 2).

Incidence of subtle tubal lesions: the SUTUBA study

There are few works on the general incidence of such subtle tubal abnormalities because, until recently, they were not reported to have any impact on fertility. We described below that this affirmation should be revised, but the first question was: What is the incidence of subtle tubal abnormalities? For that purpose, we designed an observational study (Subtle Tubal Abnormalities, SUTUBA) (unpublished data, communication in the joint ESHRE/BSGE meeting, Edimburgh 2017), where we asked various centers worldwide to detect and describe these abnormalities encountered while performing surgical operations during a two-week time. Seventeen centers were enrolled in France, China, Russia, Belgium, Algeria, Italia, Slovaquia, the United Kingdom, Hungary, Austria, and Romania.

The results are summarized in Table 1.

Incidence of subtle abnormalities was slightly higher than the one observed in the literature [8], as out of 208 patients, 113 had at least one subtle tubal abnormality (54.3%). We asked to report any

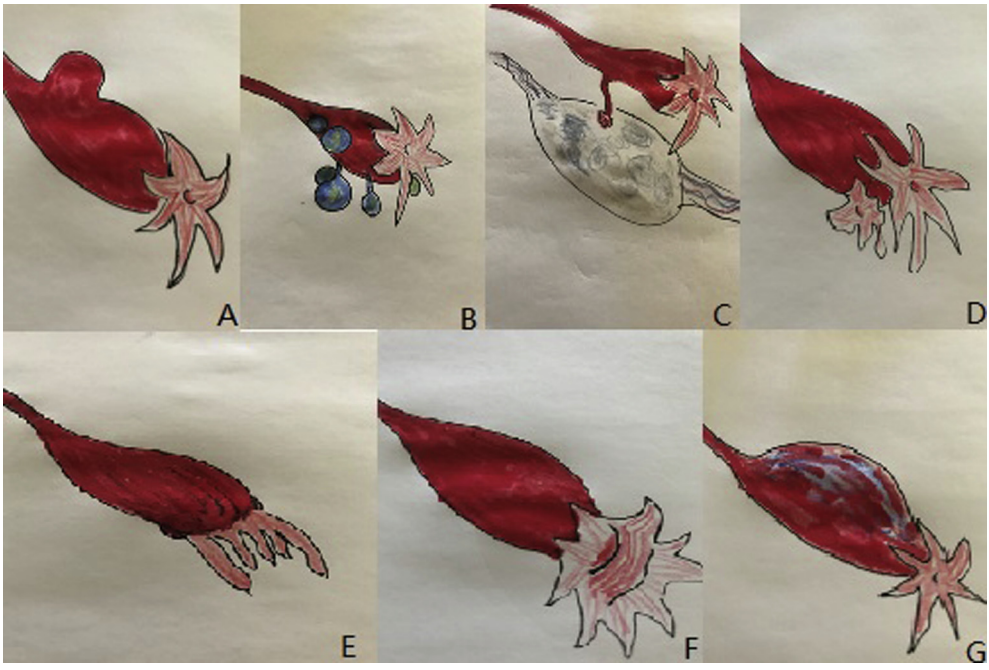

Fig. 2. Subtle variations in tubal anatomy. A. tubal diverticula; B. Morgagni hydatids; C. accessory fallopian tube; D. accessory ostium of the fallopian tube; E. phimosis; F. agglutination; G. sacculation.

Table 1
Patients' characteristics.

	Number (n)	Percentage (%)
Total	208	
Infertility	101	48.7
Unexplained infertility	48	23
Population		
Caucasian	164	78.7
Asian	33	15.9
African	11	5.3
Operation modes		
Laparoscopy	173	83
Fertiloscopy/TVE	7	3.3
Laparotomy	26	12.5

variation observed, and interestingly, the appendix vesiculosa, which is a 5 mm small cyst described in classical anatomy as normal anatomic variation, was reported in 35 cases (31%) depending on the site with no statistical difference between fertile and infertile women; however, the impact on fertility seemed to be important in the case of MH. The difference observed with fertile and infertile patients is highly statistically significant, especially when cysts are bilateral: no fertile patients were found to have bilateral cysts. In itself, this observational study is a confirmation of the work of Rasheed et al. [8]. Further studies are needed, but this study is one more argument to evaluate and consider these abnormalities as pathologies that potentially impact infertility.

Systematic description and fertility impact

Tubal diverticula

Characteristics. Tubal diverticula identified at laparoscopy are small, thin-walled outpouches present in the tubal isthmus and ampullary portion of the fallopian tube (Fig. 3) [9]. The literature reports that diverticula were diagnosed by hysterosalpingography (HSG) and that diverticula were located in the tubal isthmus and ampulla [10]. HSG may be helpful in enabling the diagnosis of tubal diverticula, but it can be misdiagnosed as a distal occlusion with dilatation of the ampullary tract [10]. In 1995, Muzii et al. [11] reported one patient with bilateral solitary tubal diverticula, which was falsely diagnosed as bilateral tubal occlusion. In 1996, Muzii et al. [12] demonstrated that of 25 distal fallopian tube occlusions diagnosed at HSG, 3 were tubal diverticula, confirmed at laparoscopy. In 2014, 13 cases of solitary tubal diverticula were analyzed in our own center, at HSG in 11 patients, in which 5 were diagnosed as tubal occlusions; however, at laparoscopy, 4 were confirmed as tubal diverticula and the remaining was hydrosalpinx [9]. In our experience, HSG findings for diverticula were as follows: i) the shape of the fallopian tube is normal and no rigid tube is observed; ii) the tubal ampulla is enlarged or appears to be a cyst; iii) contrast medium may spill into the peritoneum; and iv) the dissemination

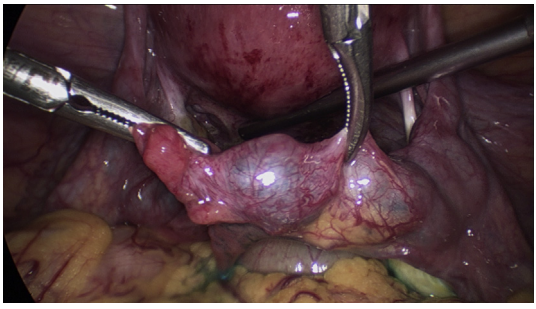

Fig. 3. Tubal diverticula (laparoscopic view).

phase shows good dissemination, and cloudy or patchy dissemination may be observed. The dissemination phase of HSG could help to differentiate the diverticulum and hydrosalpinx; in tubal diverticula, the residual contrast medium would finally drain into the pelvic cavity, whereas in the hydrosalpinx, there would not be contrast medium in the pelvic cavity. Salpingoscopic findings can be normal tubal mucosa, or thin, enlarged, and transparent tubal wall in the diverticulum [13]. Laparoscopic surgery is considered as the golden accessing technique. During laparoscopy, the fallopian tube, especially the ampulla, should be carefully examined when diluted methylene blue is injected. The diverticulum outpouches after the injection of diluted methylene blue. If the physician observes only whether the diluted methylene blue has drained from the fimbria but not the entire fallopian tube, tiny fallopian tube lesions may be overlooked.

Tubal diverticulum is a rare condition with an incidence ranging from 0.9% to 2%. A literature review revealed that most of the studies were case reports [11,14]. Two major tubal diverticula are classified as follows: multiple diverticula, for example, genital tuberculosis, salpingitis isthmica nodosa, and endometriosis tubae interna, and solitary diverticulum, which refers to a thin-walled pouch, first described by Treoll in 1970 [15]. The disease could be considered a normal tubal variant with unknown etiology, though many factors have been incriminated, such as congenital, hyperplastic, and inflammatory [14]. In our own study, 13 tubal diverticula were observed during laparoscopic surgical procedures performed because of infertility, accounting for 2.11% of the infertile patients undergoing laparoscopy [9]. Yablonski [16] reported that the incidence of the variant in infertile women was approximately 2%, which is consistent with our results.

The histology of tubal diverticulum was first documented by Troell [15] and showed the diverticulum to be lined by tall columnar epithelium surrounded by concentric layers of the fibromuscular tissue as would be shown in a normal fallopian tube. The removed tubes with the diverticulum in our own center were also examined historically. Similar to the previous results, the diverticulum was lined by tall columnar epithelium, with layers of the surrounding fibromuscular tissue, and with dilated blood vessels and sporadic inflammatory cells [9].

Impact on fertility. Until now, it is still unclear whether tubal diverticula are closely associated with infertility. However, some investigators believe that tubal diverticula may be associated with infertility and tubal pregnancy. Yablonski et al. [16] reported that 2 tubal diverticula were observed in 100 infertile women at laparoscopy, whereas no tubal diverticula were observed in 100 women undergoing cesarean section (C/S). The authors believed that the tubal diverticula were associated with infertility and proposed the theory that the tubal diverticula delayed the egg entering the uterus. Similar to the study above, in our own study, 13 tubal diverticula were observed during laparoscopy because of infertility, accounting for 2.1% of the infertile patients undergoing laparoscopic surgery [9]. However, several case reports also suggested that a tube with a diverticular lesion will permit gamete and embryo transport, resulting in a normal intrauterine conception, thus casting doubt on the suggested association between infertility and tubal diverticula [14,15]. Therefore, it is unclear whether tubal diverticula are closely associated with infertility.

In the previously mentioned report of 13 cases [9], we demonstrated that tubal diverticulum was more prevalent in women with endometriosis than in infertile patients (6.7% vs 2.1%; $p = 0.000$). Eleven patients (84.6%) had endometriosis, which was in an early stage (I or II) in 9 patients (81.8%). All 13 cases were diagnosed by laparoscopy, and the diverticula were resected during surgery. To our knowledge, this is the first report focusing on the relationship between tubal diverticula and endometriosis. Further studies are therefore needed to assess better the significance of this anomaly in terms of fertility.

Complications. In literature, 6 articles on tubal diverticula were published as case reports, involving 8 tubal diverticula [13,17–21]. Three cases that were secondary to ectopic pregnancy were reported. Although normal intrauterine pregnancies have been reported with this condition [17], these patients are still at risk of developing an ectopic pregnancy.

The experience with fallopian tube diverticula is limited and the prognosis is unknown. Tulandi et al. [22] conducted a 5-year follow-up of 10 patients with tubal diverticula and found that only 1 patient had a natural pregnancy, which indicates that the pregnancy rate in patients with tubal diverticula is lower than that in other infertile populations. Because tubal diverticula may interfere with transport of the egg and has a predisposition to ectopic gestation, surgical intervention may be

discussed. Improvement in fertility potential after surgical correction of tubal diverticula has been reported [9].

Morgagni hydatids (MHs)

Characteristics. Hydatid cysts of Morgagni are simple cysts that originate from the remnants of paramesonephric (Müllerian) or mesonephric (Wolffian) ducts present during urogenital embryologic development [23]. Hydatid cysts of Morgagni or MHs are the small pedunculated structures attached to the uterine tubes near their fimbriated end and represent remnants of the mesonephric ducts [24]. MHs are among the most common benign conditions of the fallopian tubes [25]. It was reported that MH occurred most frequently (45.6%) as adnexal masses in pregnancy [25]. MHs are thin-walled, smooth, pedunculated cysts arising from the fallopian tube and contain clear fluid. Figs. 4–6 show the presence of MH.

The true incidence rates are not clearly known because these cysts are usually found incidentally during operative procedures for other indications [26]. According to a prospective case series of 683 patients with adnexal masses by Marana et al. [27], MHs were found on histopathological examination in 8% of patients. The prevalence of MHs in a setting of 240 fertile patients undergoing C/S was compared with that reported in 204 infertile patients undergoing laparoscopy [26].

MHs cannot always be documented during a routine transvaginal ultrasonography (TV-US) examination unless they are larger than 2 cm in diameter. Similarly, HSG may not provide informative clues about MH. In addition, patients with MHs are asymptomatic, and MHs are discovered as incidental findings at laparotomy or laparoscopy.

Impact on fertility. The relationship between MH and infertility is unclear. Several studies evaluated the effects of MH on infertility. The fact that Gupta found significantly higher rates of MH in infertile women may be an indirect proof of the effect of MH in the pathophysiology of infertility [28]. Of the 330 women in the study period who underwent surgery for suspected endometriosis, 63 had infertility, of which 24 (38.1%) had MHs. Of the 239 patients who did not have infertility, 40 (16.7%) patients had the disease [28]. A similar trend was also described by Cebesoy et al. [24]. Cebesoy and colleagues compared the MH frequency in women known to be fertile (having a spontaneous conception and delivering by C/S without any infertility therapy and infertile). According to Cebeoy et al., the incidence of MH in fertile women was 15/240, while the rate was increased to 36/204 in infertile patients [24]. A nonrandomized controlled trial study addressed the issue of the role of MH in unexplained infertility [8]. The prevalence of these cysts in patients with unexplained infertility (52.1%) is considerably higher than those with explained infertility (25.6%), thus supporting the probable impact of MH on fertility. However, citing MH as a single cause of infertility in patients with infertility would be grossly incorrect, as many unknown factors may also be related with infertility. Recently, a study suggested that endometriosis is present in MHs in infertile women or those with pelvic pain at the rate of 11.3% [28]. It should be noted that this study is limited by the small sample size. Fifty patients were confirmed to have endometriosis, with only 7 patients having endometriosis in MHs. Therefore, larger randomized controlled studies are needed to further evaluate the relationship between endometriosis and MHs.

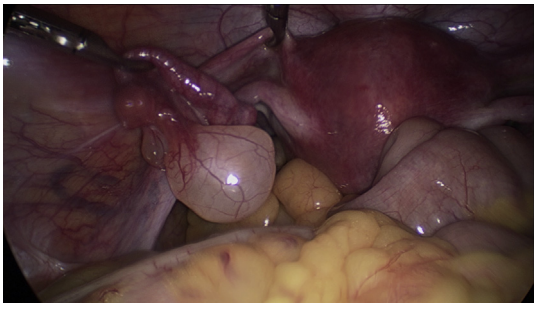

Fig. 4. Fimbrial Morgani hydatids (laparoscopic view).

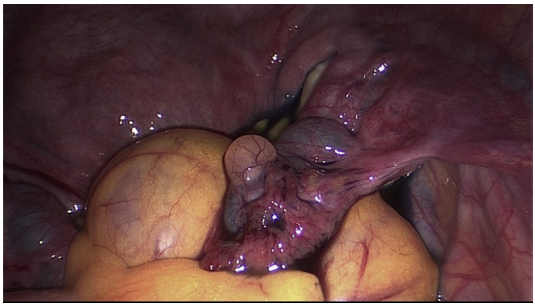

Fig. 5. Sessile Morgani hydatids (laparoscopic view).

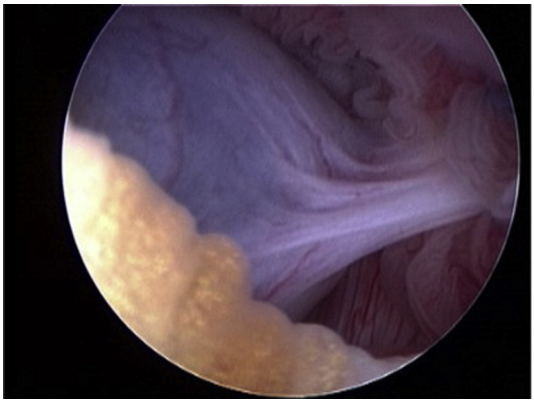

Fig. 6. Intrafimbrial Morgani hydatid (fertiloscopic view).

The involvement of MHs in the pathogenesis of infertility remains unclear, although possible mechanisms involving the disruption of tubal motility and ovum pick-up of the fimbriae caused by the abnormal weight of the MH in the distal part of the tube may explain the effects on infertility [24]. As MHs have been identified in a research as a possible manifestation of endometriosis, it is likely that endometriosis-related pro-inflammatory mediators also impede fertility.

Excision of the MHs when found incidentally during a surgery is recommended [29]. The author observed a high pregnancy rate after removal of the cysts and suggested that MH as a single pelvic pathology might hinder fertility. Consistent with the study, Rasheed et al. also advocated the policy of removing these cysts by laparoscopy in infertile patients. The remarkably higher pregnancy rate (58.7%) after laparoscopic excision of these cysts than that in the control group (20.6%) is a finding that supports the efficiency of surgical intervention [8].

Complications. MH is an extremely rare cause of isolated torsion of the hydatid cyst or torsion including fimbria [30,31]. The presence of the cyst of the MH at the fimbrial end is probably responsible for tubal torsion because the free end of the tube becomes heavier, excessively mobile, and more susceptible to rotation [31]. Torsion occurs often at the time of ovulation and frequently involves the right fallopian tube. Patients with tubal torsion should be followed up after conservative treatment and recommended to seek medical advice immediately if they experience similar symptoms in the future.

Accessory fallopian tube

Characteristics. The fallopian tube is the part of the uterus that carries the ovum from the ovary to the uterus, whereas the accessory fallopian tube is the congenital anomaly attached to the ampullary part of

the main tube. The accessory fallopian tubes are congenital and developmental anomalies resulting from the bifurcation of cranial ends of Müllerian ducts. During the embryologic development of the female urogenital system, the Müllerian ducts fuse cranially from their caudal tips forming a tube with a single lumen known as the uterovaginal primordium, which develops into the upper segment of the vagina, fallopian tube, and uterus. Many of these malformations are those resulting from varying degrees of failure of fusion of the paramesonephric ducts [32,33]. Gandhi et al. [34] found duplication of the fallopian tube on the right side in a young female cadaver during routine and described that accessory fallopian tubes are nonpatent cylindrical structures attached to the ampullary part of a normal-sized tube. One of the tubes was the main tube of normal size and the other one was thin and hypoplastic arising from the ampullary portion of the main fallopian tube with their own fimbriae. Both the ovaries and uterus were normal, and no renal anomalies were found. Dahan et al. [35] suggested a similar conclusion that when congenital ampullary atresia occurs without concomitant Müllerian anomalies, they would not be expected to be associated with an increase in renal abnormalities. Beyth et al. [36] assessed 200 abdominal operations and described all accessory tubes were attached to the ampullary segment of the oviduct. Examined histologically, accessory tubes were cylindrical in structure, and their length stretched from 15 to 20 mm, with fimbriated end. They were composed of a stalk with relatively large tortuous vessels with hyalinized walls, fibrous tissue, and smooth muscle. The fimbrial end was open in all tubes, which had fine fimbriae lined with normal epithelium similar to that of the main fallopian tube. No communication was found in any of the cases with the lumen of the main fallopian tube [36].

Accessory tubes are not a rare finding. Their exact incidence is not known because they are inconspicuous structures and usually overlooked by surgeons. Kossman was the first to report the incidence of accessory tubes in 1894, which was found in 4%–10% of all women [37]. Since then, many authors reported the anatomy, histology, and pathogenesis [35,38,39]. Gardner et al. reviewed accessory tubes partially in 1948 [37]. Zolcinski et al. [39] found the disease in nearly 5% of their abdominal operations. Beyth et al. [36] assessed 200 operations and indicated that the incidence of accessory tubes in their experience is approximately 6%.

Impact on fertility. The accessory fallopian tube is an anatomical variation, which may cause gynecological complications such as infertility. How and why an accessory tube leads to infertility? Beyth et al. discussed the phenomenon of infertility in patients having accessory fallopian tube [36]. Accessory tube is obliterated at its attachment with the main tube, and the ova is wasted, leading to infertility [36]. Alternatively, the other entry may impair ciliary activity and adversely affect fluid transport mechanism of the oviduct [40]. In addition, peritoneal migration of sperm is a well-known phenomenon [41]. Sperm that enter the peritoneal cavity through the main tubal ostium may migrate to the fimbria of the accessory tube and fertilize a captured ovum there, resulting in an ectopic pregnancy in the accessory tube [36]. The accessory fallopian tubes are one of the possible contributory factors of infertility as their fimbria pick the ovum instead of fimbria ovarica of normal tube.

Complications. Ectopic pregnancy, torsion, cystic swelling, and pyosalpinx are other complications related to the accessory tube [40,42–44]. In case fertilization occurs, this leads to ectopic pregnancy, another life-threatening complication [42,44]. Ectopic pregnancy within the accessory tube leading to excessive bleeding and surgical intervention is widely described in literature. Torsion of the accessory tube is a rare. A review of literature shows three reports of torsion of an accessory fallopian tube, all described in young girls [40,45]. In a more recent case report, a twisted right accessory fallopian tube arising from the ampullary portion of the main fallopian tube was noted in laparoscopy [40]. The torsion of the right fallopian tube is more common because the left tube is fixed in the left hemipelvis by the sigmoid colon and mesentery [32]. Pyosalpinx in the left accessory fallopian tube was briefly described by Macnaughton almost a century ago [43]. Since then, no such complication has been investigated.

To improve fertility outcomes and for the prevention of these complications, some authors recommended surgical excision when found incidentally [34,40]. Failure to recognize the abnormalities may otherwise result in patients being diagnosed as idiopathic infertility. Systematic examination and appraisal of every oviduct during gynecological surgery may help in the identification and interpretation of such anomalies. Accessory tubes are recommended to be carefully removed surgically, especially in infertile women.

Accessory ostium

Characteristics. Fallopian tube accessory ostium or secondary ostium is a rare condition that occurs when an ectopic fimbria is observed at a distance from the fimbriated end as a natural variation of the fallopian tube (Fig. 7) [46]. Cohen et al. reported that the accessory ostia were all in the mid-ampulla of the fallopian tubes in the patients they studied [47]. Accessory tubal ostia are characterized by two separate fimbrial openings, that is, greater (primary) and lesser (accessory) ostia, and are thought to result from the bifurcation of the distal ends of the Müllerian ducts [44]. Accessory tubal ostia are divided into two subtypes depending on the distance between the primary and accessory ostia seen during laparoscopy. Those occurring <1 and ≥ 1 cm from the primary ostia are called terminal and ampullary accessory ostia, respectively [46].

The accessory tubal ostia are considered rare fallopian variants, the prevalence of which varies among different studies. One early study [47] suggested that the prevalence of this condition could be 5%–10%. Yablonski et al. [16] reported that the incidence of fallopian tube accessory ostium was higher among infertile women than among their fertile peers (10.0% vs. 0.0%). Isherwood et al. [48] detected fallopian tube accessory ostium in 4 of 82 patients undergoing gamete intrafallopian transfer—a frequency of 5.0%. Beyth et al. [35] reported the incidence of fallopian tube accessory ostium to be 6.0%. Zheng et al. [46] evaluated 1113 patients who underwent laparoscopy for infertility and found that 21 (1.9%) were diagnosed with fallopian tube accessory ostium, which is lower than that previously reported.

Diagnosis of fallopian tube accessory ostium can be very difficult prior to surgery because of a lack of clinical features, e.g., patients can have no history of pelvic inflammatory disease and no positive findings after gynecologic examination. Additionally, the HSG does not provide informative clues in many cases, thus necessitating laparoscopy, particularly in the presence of dysmenorrhea or primary infertility. During laparoscopic surgery, it is recommended that surgeons not only focus on whether the fallopian tube is patent but also carefully examine the entire tube (especially the distal portion) to enable the discovery of fimbrial pathology during chromotubation. Lack of careful evaluation of the fimbria once the blue dye is seen, suggesting tubal patency, may account for the failure of detecting such subtle pathology.

Impact on fertility. Previous studies have reported accessory tubal ostia, at least co-incidentally, in patients undergoing treatment for infertility [16,35,44,46–49]. Nevertheless, the association between accessory tubal ostia and infertility remains controversial, and most of the relevant findings were published at least 25 years ago. In a retrospective study, Yablonski et al. [16] reported that the incidence of fallopian tube accessory ostium was higher among infertile women (10%) than among their fertile controls (0%). A study performed by Novak et al. demonstrated that this condition was associated with tubal ectopic pregnancy [49]. We previously investigated the clinical features of fallopian tube accessory ostium and found that it might also be associated with endometriosis [46]. In this study, the prevalence of fallopian tube accessory ostium was markedly higher among women with endometriosis than among those without endometriosis. Nineteen out of 21 (90.5%) patients with accessory ostia were detected to have endometriosis, mainly at an early stage (I–II) [1]. However, Yablonski et al. [16] suggested that fallopian tube accessory ostium had no association with endometriosis or pelvic

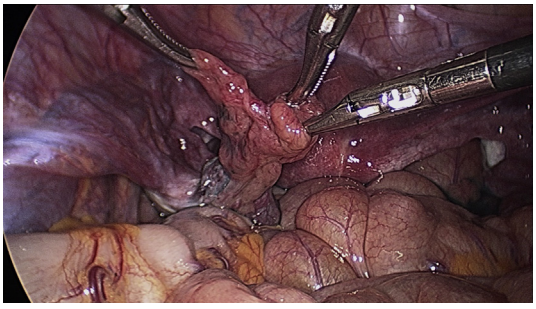

Fig. 7. Accessory ostium of the fallopian tube (laparoscopic view).

inflammatory disease. Cohen et al. [47] proposed that fallopian tube accessory ostium was predominantly a congenital condition. Therefore, it remains unclear whether or not fallopian tube accessory ostium is associated with endometriosis.

How fallopian tube accessory ostium induces infertility remains controversial, but several mechanisms could contribute to pathogenesis. One possible pathogenesis is that the ovum can escape from the tube through the accessory ostium. Alternatively, the additional opening could allow derangement of the normal ciliary and fluid transport mechanism of the oviduct and interfere with the fertilization process. In addition, a possible mechanism that a "double" ostium may impair ovum pick-up by decreasing the size of the funnel of the normal ostium should not be excluded. Finally, infertility might also be associated with coexisting endometriosis.

Desirable fertility outcomes have been reported after surgical treatment of accessory tubal ostia. Surgical treatment of accessory tubal ostia can be achieved through the laparoscopic or abdominal approach. Terminal accessory ostia can be treated with an incision made from the fimbria to the accessory ostium, with a mattress suture of the fimbria. In contrast, ampullary accessory ostia can be treated with a monofilament purse-string suture at the base of the accessory ostium followed by resection or electrocoagulation of ostial stump [46]. In a study of 21 infertile women with accessory ostia, Cohen et al. [47] reported a live birth rate of 47% with natural conception after tubal microsurgery. Our experience is that ampullary accessory ostia can be treated with a monofilament purse-string suture at the base of the accessory ostium followed by resection or electrocoagulation of ostial stump. Using a laparoscopic approach in 18 patients with accessory ostium, 66.7% of patients achieved a successful clinical pregnancy [46].

Phimosis

Characteristics. Phimosis (fimbrial conglutination) is a concentric stricture of the fallopian tube at its distal end noted at the infundibular fimbrial junction [21]. The phimosis was considered as mild when the distal tube was narrowed, while moderate to severe phimosis was defined when the tube was constricted partially and required a cuff salpingostomy [50]. Phimosis, which presumes tubal patency, was distinguished from fimbrial occlusion. This pathology can be misdiagnosed as fimbrial occlusion by hysterosalpingography, whereas laparoscopy could disclose that the tube is patent with conglutination of the fimbrial folds [51]. During tubal perfusion in these patients, there was a dilation of the distal ampullary portion of the fallopian tube, and the dye was spilled out as a narrow stream.

Data about the prevalence of phimosis are limited. Lapido et al. investigated the tubal patency in 86 infertile patients, and the results showed that 13 cases of fimbrial phimosis were diagnosed by laparoscopy [51]. Abuzeid et al. [7] suggested the presence of fimbrial phimosis in many infertile patients with an early stage of endometriosis. The study group consisted of 315 infertile women who were found to have stages I and II endometriosis. Women with a documented history of pelvic inflammatory disease were excluded. The control group consisted of 152 infertile women without endometriosis. The prevalence of fimbrial pathology was significantly higher in the study group (50.2%) than in the control group (17.8%). The author also pointed out that fimbrial phimosis was the most common type of fimbrial pathology in the group of infertile women with endometriosis. The incidence of fimbrial primroses (unilateral and bilateral) was significantly higher in infertile patients with early stages of endometriosis than in infertile patients without endometriosis. The data may also suggest the cause and effect between endometriosis and some cases of fimbrial phimosis, or they may reflect a correlation only [7].

How endometriosis induces fimbrial phimosis remains unclear, but several mechanisms could contribute to pathogenesis. One possibility is that certain inflammatory factors not only interfere with fallopian tubal function but also may damage the tube anatomy, causing subtle variations including fimbrial, agglutination, fimbrial blunting, and fimbrial phimosis [52,53]. Alternatively, the presence of fimbrial pathology may be associated with a subtle developmental anomaly during differentiation of the Müllerian ducts. Therefore, the relationship between endometriosis and phimosis may be a subtle clinical picture of what has already been described in the literature of higher incidence of endometriosis in patients with nonobstructive anomalies [54,55].

Impact on fertility. A previous study indicated that infertile women had significantly more tubal variations including fimbrial pathology than did their fertile counterparts [16]. Cohen and Katz reported that convoluted oviducts may be associated with infertility, endometriosis, and fimbrial phimosis [47]. Savaris et al. [56] evaluated the expression of β3 integrin subunit in the endometrium of infertile patients with tubal phimosis and found that the expression of β3 integrin subunit is significantly reduced in the presence of tubal phimosis during the window of implantation. The study suggested the detrimental effects of tubal phimosis on markers of uterine receptivity.

If phimosis is present, it should be corrected by laparoscopic fimbrioplasty. Fimbrial grasping forceps were used to grasp and elevate the fimbriated end. A laparoscopic pointed probe was then introduced through the narrow ostium. A small incision was made along the antimesenteric site, followed by an exstrophy mattress suture of the fimbria. In a study of 85 patients with fimbrial phimosis, Abuzeid et al. [57] reported that after laparoscopic fimbrioplasty, a cumulative conception rate of 74.2% after 24 months of follow-up was achieved. In another study on 122 patients with fimbrial pathology and early stages of endometriosis laparoscopic surgery (79 patients with stage I and 43 patients with stage II endometriosis), the authors reported cumulative conception rates of 66% and 64% after 12 months of follow-up [58].

Fimbrial agglutination

Characteristics. Fimbrial agglutination was defined as one or more adhesive bridges of fimbria across the ostium (Fig. 8) (interfimbrial bridges or adhesions) [7]. There are no data available on the incidence of fimbrial agglutination in fertile women.

A retrospective study showed that there was no significant difference in the incidence of fimbrial agglutination (unilateral or bilateral) between patients with or without endometriosis [7]. However, the authors admitted that the sample size was not large enough to achieve strong power and suggested that if a large group of patients is added, statistical significance may be achieved. Alternatively, it may simply be a coincidence.

Impact on infertility. Although the association of fimbrial agglutination with primary infertility remains contentious, some authors postulate that the anatomic and functional aberrations of the fallopian tube as the fimbrial agglutination may contribute to the pathogenesis of infertility [16,21]. Yablonski et al. [16] described that infertile patients had a significantly higher incidence of tubal anomalies, including fimbrial pathology, than did their fertile counterparts. Fakih and Marshall in their study on a selective group of patients with endometriosis who underwent gamete intrafallopian transfer also reported a similar conclusion [21]. They reported that when controlling for age, semen characteristics, number of mature oocytes transferred, and tubal status, the extent of anatomical tubal abnormalities (tubal disorders included phimosis, fimbrial agglutination, etc.) was the most important prognostic factor in determining pregnancy outcome.

It remains unclear how fimbrial agglutination induces infertility, but several mechanisms could contribute to pathogenesis. First, interference with the ovum pick-up mechanism was cited as the most possible reason for infertility in patients who suffer from this pathology. Second, the presence of

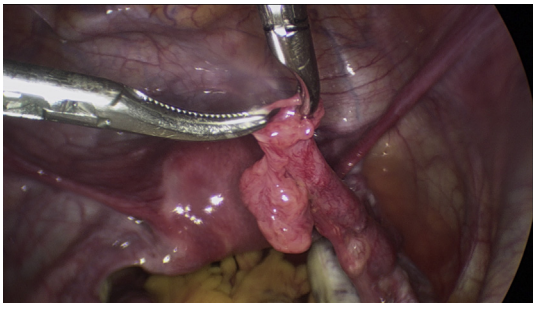

Fig. 8. Tubal agglutination (laparoscopic view).

certain factors in the peritoneal fluid of patients with endometriosis may also play a role as proposed by Abuzeid et al. [7]. Third, if fimbrial pathology is due to pelvic inflammatory disease, associated peritubal adhesions and the presence of a factor of endosalpinx damage may be present as an additional cause of infertility.

Fimbrial agglutination may be filmy or relatively dense. To correct this lesion, laparoscopic fimbrioplasty is recommended. The fimbrial end is grasped using fimbrial grasping forceps, and the bank of agglutination is elevated using a laparoscopic probe. The monopolar diathermy needle tip is then used to divide the bank of agglutination over the laparoscopic probe. In a large prospective study on 295 patients with distal nonocclusive tubal lesions, Saleh and Dulgi reported a cumulative conception rate of 58% post laparoscopic fimbrioplasty after 24 months of follow-up [50].

Tubal sacculation

Characteristics. In the list of subtle abnormalities, one is rather frequent, the tubal sacculation, in which there is a sort of ampullary hernia of length from 1 to 4 cm (the whole ampulla in this case) due to the lack of a muscularis layer (Figs. 9 and 10).

Tubal sacculation, or tubal hernia, is different from tubal diverticula. In tubal diverticula, the three layers of the tube are respected (i.e., serosa, muscularis, and mucosal layers); however, in tubal sacculation, there is muscularis hypoplasia.

Diagnosis is easily made by laparoscopy or fertiloscopy. In laparoscopy, the blue dye fills into the "tubal hernia" and the thinnest is rather evident the ampulla becoming blue. During a fertiloscopy (transvaginal endoscopy), a salpingoscopy may be performed; in this case, the tube wall appears so thin that the fatty tissue or the bowel is seen by transparency [59,60].

The frequency is probably underestimated due to the lack of proper tube examination during laparoscopy because these abnormalities are not occlusive. In the SUTUBA study, we found 26.7% of infertile patients presented with tubal sacculation, and in 24.5% patients, the infertility was "unexplained" (Table 2); these values were higher than those reported by Yablonski et al. [16]. In a personal series, we previously observed almost the same rate, with 18 of 67 (26.8%) presenting with tubal sacculation [61].

Impact on fertility. The impact on fertility is not well known, but we may anticipate that the lack of a proper muscularis layer may impair the ovum pick-up mechanism as well as the ovum or sperm transport.

Potentially, it could present an increased risk of ectopic pregnancy. Hence, when such conditions are encountered, we treat them promptly. The treatment proposed is a racket-form salpingostomy: an incision is made on the hernia until the fimbrial end, and then, the eversion is performed and maintained using fine sutures (6x0). Preliminary results seem to indicate that it is worst to treat this kind of hernia with a pregnancy rate of 48% in the following 6 months in our short series [61]. Indeed, these results have to be confirmed in a larger prospective study.

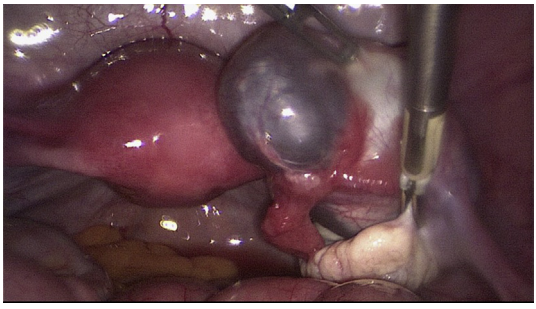

Fig. 9. Tubal sacculation (laparoscopic view).

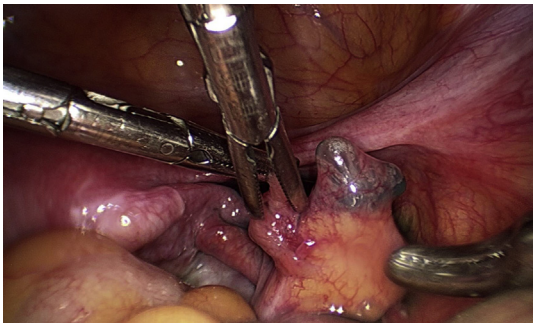

Fig. 10. Tubal sacculation (laparoscopic view).

Table 2
Global results of the SUTUBA study.

	Total n (%)	Infertility n (%)	Unexplained infertility n (%)
Accessory tube			
L	9 (4.3)	6 (5.9)	4 (9.8)
R	7 (3.3)	6 (5.9)	5 (10.4)
Sacculation			
L	18 (8.6)	14 (13.8)	7 (12)
R	25 (12.0)	22 (21.8)	11 (22.9)
Bilateral	11 (5.2)	9 (8.9)	5 (10.4)
Bridge adhesion			
L	20 (9.6)	15 (14.8)	8 (16.6)
R	12 (5.8)	11 (10.9)	5 (10.4)

Summary

It is important to understand the variety of tubal subtle abnormalities. Their frequencies are grossly underestimated because they all have a common point, i.e., they are nonobstructive lesions. Therefore, they are often missed during laparoscopy and the dye test (lap and dye test), where only patency is checked. Hence, one should be strongly recommended to perform a complete evaluation of the tube, especially of the distal part of the tube where most of the lesions occur. This will enable surgeons to detect and treat such abnormalities.

We have increasing evidence of the impact of these abnormalities on fertility, and even if many studies remain to be conducted in this field, current evidence is sufficient to propose to treat these abnormalities. The laparoscopic approach is preferred. Usually, the correction of these abnormalities is not very difficult provided that the surgeons respect the integrity of the tube and apply the principles of microsurgery very well defined in the eighties by Winston and Gomel [62,63].

By implementing this change of paradigm in the management of tubal infertility, we can decrease the number of so-called “unexplained infertility,” which may lead to unnecessary IVF or lack of treatment when access to IVF is difficult.

Practice points

- Large epidemiological studies of tubal subtle abnormalities are lacking, and their frequencies are grossly underestimated.
- Fallopian subtle lesions are associated with an increased risk of infertility.
- Early identification of these variations may be beneficial in preventing progression and infertility.
- Pregnancy rate could be improved through the correction of these tubal subtle lesions.

Research agenda

- Prospective study evaluating the impact of tubal subtle lesions on fertility
- Randomized controlled trials evaluating surgical correction of tubal subtle lesions

Conflict of interest

The two authors have no conflict of of interest to declare regarding the content of this article.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bpobgyn.2018.12.012.

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