European Journal of Obstetrics and Gynecology 305 (2025) 132–141

Contents lists available at ScienceDirect

European Journal of Obstetrics & Gynecology and Reproductive Biology

journal homepage: www.journals.elsevier.com/european-journal-of-obstetrics-and-gynecology-and-reproductive-biology

Review article

The Pivotal role of hysteroscopy in diagnosing subtle uterine lesions in infertile patients: Seeing the unseen can make the difference

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ARTICLE INFO

Keywords:
Hysteroscopy
Subtle uterine lesions
Female infertility
Dysmorphic uterus
Adenomyosis
Endometritis
Endometrial osseous metaplasia

ABSTRACT

Background: Subtle uterine lesions are abnormal intracavitary conditions that, while not causing significant anatomical changes, can adversely impact fertility. Identifying these "subtle" lesions is challenging due to arbitrary interpretation, varied management strategies, unclear clinical significance, and insufficient clinician training in recognizing them. Hysteroscopy offers direct visualization and the capacity to obtain targeted biopsies, making it an invaluable tool for the diagnosis and treatment of these often overlooked conditions.

Purpose: The lack of standardized and universally accepted terminology complicates the diagnosis and treatment of subtle uterine pathologies. A thorough understanding of how to identify and effectively manage these lesions is essential for the treatment of infertile patients. This review summarizes the critical and evolving subtle uterine lesions that clinicians frequently overlook during hysteroscopic procedures, highlighting the importance of accurate identification and its potential to improve reproductive outcomes.

Methods: This literature review followed the Scale for the Quality Assessment of Narrative Review Articles (SANRA) guidelines. All articles discussing infertility-related issues and subtle lesions diagnosable with hysteroscopy were deemed eligible for inclusion.

Results: We identified several subtle uterine lesions that are often overlooked during routine hysteroscopic evaluations but can adversely impact fertility. These include morphological abnormalities such as the T-shaped uterus, cystic lesions of the uterine wall, adenomyosis, cervicitis, cervical and tubal endometriosis, and endometrial conditions such as chronic endometritis, thin endometrium and endometrial calcifications.

Conclusions: Effective diagnosis and treatment of subtle uterine lesions is essential to fertility care. Knowing which lesions are commonly missed and understanding their endoscopic features and potential clinical implications, is crucial for enhancing the diagnostic capacity of hysteroscopy. This, in turn, may improve reproductive outcomes and the overall quality of care of infertile patients.

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https://doi.org/10.1016/j.ejogrb.2024.12.013
Received 31 October 2024; Received in revised form 1 December 2024; Accepted 8 December 2024
Available online 9 December 2024
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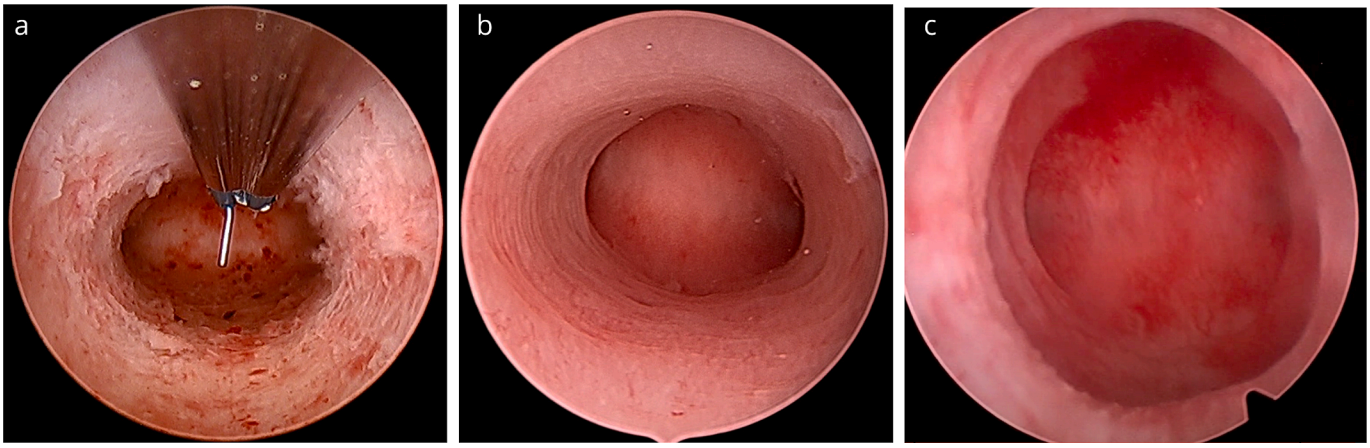


Fig. 1. Dysmorphic uteri. (a) T-Shaped Uterus: The uterine cavity has a tubular appearance with the tubal ostia not visible from the isthmus; (b) Y-Shaped Uterus: The uterine cavity appears tubular with a distinctive indentation at the fundus, giving it a Y shape appearance; (c) I-Shaped Uterus: The uterine cavity is uniformly tubular up to the fundus, with a significantly reduced interostial distance, making both tubal ostia easily visible due to their close proximity.

Introduction

Infertility affects approximately 9 % of couples of reproductive age, with female factors contributing to 20–35 % of all cases [1]. Among these, uterine cavity abnormalities are identified in about 10–16 % of women and in as high as 50 % of women experiencing recurrent implantation failure with assisted reproductive techniques [2,3].

Hysteroscopy is widely recognized as the "gold standard" for evaluating and treating intrauterine pathologies. It offers valuable insights into the accessibility and structure of the uterine cavity, crucial for procedures like intra-uterine insemination and embryo transfer [4,5].

Despite its effectiveness, even experienced hysteroscopists can occasionally miss intracavitary lesions during the procedure, potentially leading to undiagnosed pathology [6].

Hysteroscopic findings are frequently categorized into major and minor abnormalities based on their impact on the uterine anatomy. Major abnormalities, such as congenital uterine malformations (classified as U2 in the ESHRE/ESGE system), myomas, large polyps and adhesions, significantly alter uterine structure [7–12]. In contrast, minor or "subtle" lesions, such as diffuse polyposis, endometrial hypervascularization, strawberry patterns of the endometrium, mucosal elevation, endometrial defects, and subtle malformations like U1 sec. ESHRE/ESGE system, do not cause significant anatomical changes and their clinical significance remains debated [13].

A prospective randomized trial highlighted the differences in hysteroscopic findings between infertile patients and those with abnormal uterine bleeding [13]. Infertile patients more frequently exhibited major abnormalities such as congenital malformations, polyps, and adhesions, as well as subtle lesions like U1 malformations, strawberry patterns, and hypervascularization [13]. Conversely, patients with abnormal uterine bleeding primarily had major pathologies like myomas and polyps, with mucosal elevation being a common subtle lesion.

The definition of "subtle" lesions varies due to frequent arbitrary interpretation, differences in management, undetermined clinical significance and best management strategy, and the lack of training for many clinicians in recognizing these challenging lesions [14,15].

While the clinical significance of subtle lesions remains unproven, they may act as early indicators of underlying pathologies or precursors to more severe conditions, thus guiding further sub-endometrial and myometrial evaluation.

This review explores the crucial role of hysteroscopy in detecting and understanding subtle uterine lesions in infertile patients, emphasizing the importance of identifying these often-overlooked abnormalities to enhance the reproductive outcomes.

Materials and methods

We adhered to the quality standards for narrative reviews, as defined and quantified by the scale for the quality assessment of narrative review articles (SANRA)[16]. The relevant publications were identified after a systematic query of PubMed, Google Scholar, Scopus, Web of Science, and research registers (such as Clinicaltrials.gov), complemented by cross-checking the reference lists. We used a combination of the search terms "hysteroscopy", "infertility", and "lesion". No language restriction was applied. All articles describing infertility-related issues and lesions that can be diagnosed through hysteroscopy were considered eligible for review. Relevant aspects of each article were identified and explored, with attention to the type of treatment applied and the outcomes described.

Uterine abnormalities

Morphological abnormalities

The anatomical integrity of the uterine cavity is crucial for achieving and maintaining a pregnancy, as it allows for proper implantation and placentation. Defects in uterine canalization can lead to reproductive issues such as infertility, recurrent miscarriage, and obstetric complications such as intrauterine growth restriction and preterm labor [17,18]. Recent studies have demonstrated that genetically normal embryos may fail to implant if the uterine environment is not supportive [17,18].

While diagnosing and treating a uterine septum is relatively straightforward, identifying a T-shaped dysmorphic uterus, characterized by a reduced endometrial cavity volume, can be more challenging. This malformation is characterized by a narrowed uterine cavity with lateral indentations, giving it a T-shaped appearance due to thickened lateral walls [19]. Common diagnostic techniques for identifying a dysmorphic uterus include hysteroscopy, Magnetic Resonance Imaging (MRI), 2D or 3D ultrasonography, and hysterosalpingography (HSG) [19]. When evaluating the uterine cavity of a dysmorphic uterus with hysteroscopy, the uterine cavity appears tubular, and the tubal ostia are not visible from the isthmus [19] (Fig. 1a). The severity of the deformity should also be considered, as a pronounced T-shaped uterus represents an obvious lesion, while a subtle T-shaped uterus is more difficult to diagnose.

The European Society of Human Reproduction and Embryology (ESHRE) and the European Society for Gynaecological Endoscopy (ESGE) have classified the dysmorphic uterus as a Class 1 uterine anomaly. This classification includes three subgroups: U1a, which refers

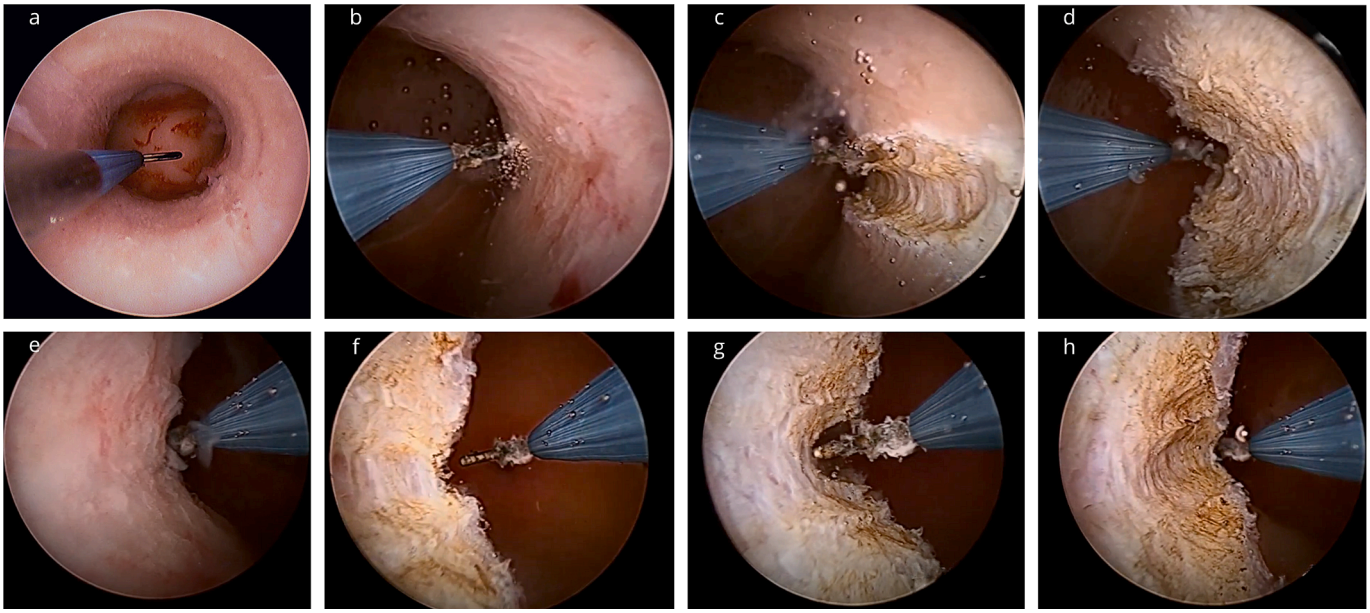


Fig. 2. Hysteroscopic metroplasty to expand a T-Shaped uterus. (a) Initial hysteroscopic view of the T-shaped uterus with a narrow, tubular cavity due to thickened lateral walls, where the tubal ostia are not visible from the isthmus; (b-c-d) Lateral incisions made with the monopolar hysteroscopic hook, perpendicular to the left uterine sidewall, from the fundus to the isthmus, gradually expanding the cavity by breaking the constriction rings. (e, f, g) Similar lateral incisions performed on the right uterine sidewall with the monopolar hysteroscopic hook, further increasing the space within the cavity.

to a T-shaped uterus; U1b, indicating an infantilis uterus; and U1c, characterized by a normal uterine outline accompanied by other abnormalities within the uterine cavity [7].

Recently, it has been proposed that, besides the T-shaped uterus, two additional types of differentiated uteri could also be identified using 3D ultrasonography and hysteroscopy [20]. The Y-shaped uterus is characterized by a narrowing in the middle third of the cavity due to thickened lateral walls and additional thickening at the fundus, resulting in a slight indentation into the uterine cavity. When viewed via hysteroscopy, the uterine cavity appears tubular with this indentation at the fundus, giving it a Y shape appearance and moving the tubal ostia further apart and laterally, thus reducing the inter-ostial distance compared to a T-shaped uterus (Fig. 1b) [20]. Another type, the I-shaped uterus, features thickened lateral walls extending from the internal cervical os up to the uterine fundus. This type of abnormal uterine cavity has almost no variation in its width. During hysteroscopy, the I-shaped uterus has a uniformly tubular cavity up to the fundus, with a markedly reduced inter-ostial distance, making both tubal ostia easily visible due to their proximity (Fig. 1c) [20].

The dysmorphic uterus, previously thought to be associated with diethylstilbestrol (DES) exposure during fetal development, has often been underestimated, misdiagnosed, and not clearly defined by diagnostic criteria [21–23]. The prevalence of T-shaped uterus and its impact on reproductive outcomes are still largely unknown. Uterine anomalies are not typically screened for in asymptomatic individuals of the general population. As a result, studies focusing on T-shaped uteri mainly include patients with infertility or adverse reproductive outcomes [24,25]. Several historical studies have reported poor reproductive outcomes in women with untreated T-shaped dysmorphic uteri [22,23]. However, a direct connection between dysmorphic uterus and infertility has not been established. The observed reproductive failures may be due to alterations in the endometrial structure, such as excessive myometrium in the uterine walls and increased uterine contractility [26].

The management of the dysmorphic uterus is also controversial due to the lack of standardized diagnostic criteria and subjectivity in treatment [25,27,28]. The most common surgical technique, hysteroscopic metroplasty, involves making two lateral incisions perpendicular to the uterine sidewalls to interrupt the fibromuscular constriction rings and expand the volume of the uterine cavity. These incisions are made from the fundus to the isthmus in the same groove until a depth of 6 to 7 mm is reached, as indicated by the visualization of the tubal ostia from the isthmus and a triangular, symmetrical, more spacious uterine cavity [29,30] (Fig. 2). Studies have demonstrated that anti-adhesion strategies post-hysteroscopic metroplasty can be beneficial [27,30,31]. Technological advancements, including the development of miniaturized mechanical instruments, bipolar energy devices and laser technologies, have enabled the treatment of the dysmorphic uterus in outpatient settings, thereby reducing the need for more invasive procedures [32–35]. Although systematic reviews and meta-analyses have demonstrated promising reproductive outcomes and the cost-effectiveness of hysteroscopic metroplasty, there is still insufficient evidence of its impact on reproductive outcomes [25,28]. This is primarily due to the absence of randomized clinical trials comparing the treatment with non-intervention. This limitation underscores the ongoing uncertainty about the association between dysmorphic uteri and infertility, as well as the true effectiveness of hysteroscopic surgery in improving reproductive outcomes. Therefore, further studies are urgently needed to clarify whether T-shaped uterus and subtle uterine anomalies negatively impact fertility and whether surgical treatment is necessary [36].

Cystic lesion of the uterine wall

A cyst or a cyst-like lesion within the myometrium is relatively uncommon and can be linked to benign and malignant conditions [37]. Benign causes may include adenomyosis, venous congestion, cystic degeneration of fibroids, or a cyst at the site of a previous endometrial scar [37,38]. Malignant causes might involve an invasive mole or a placental site trophoblastic tumor [37,38].

Cystic degeneration of a fibroid refers to a structural change where cavities filled with serous, mucinous, or hemorrhagic fluid develop within the fibroid tissue. This transformation can occur when the fibroid experiences rapid growth not matched by an adequate blood supply, a scenario that may occur during pregnancy [38].

Adenomyosis, the most common cause of myometrial cystic lesions, is identified as ectopic, non-cancerous endometrial glands and stroma within the myometrium. This disorder can anatomically manifest as one

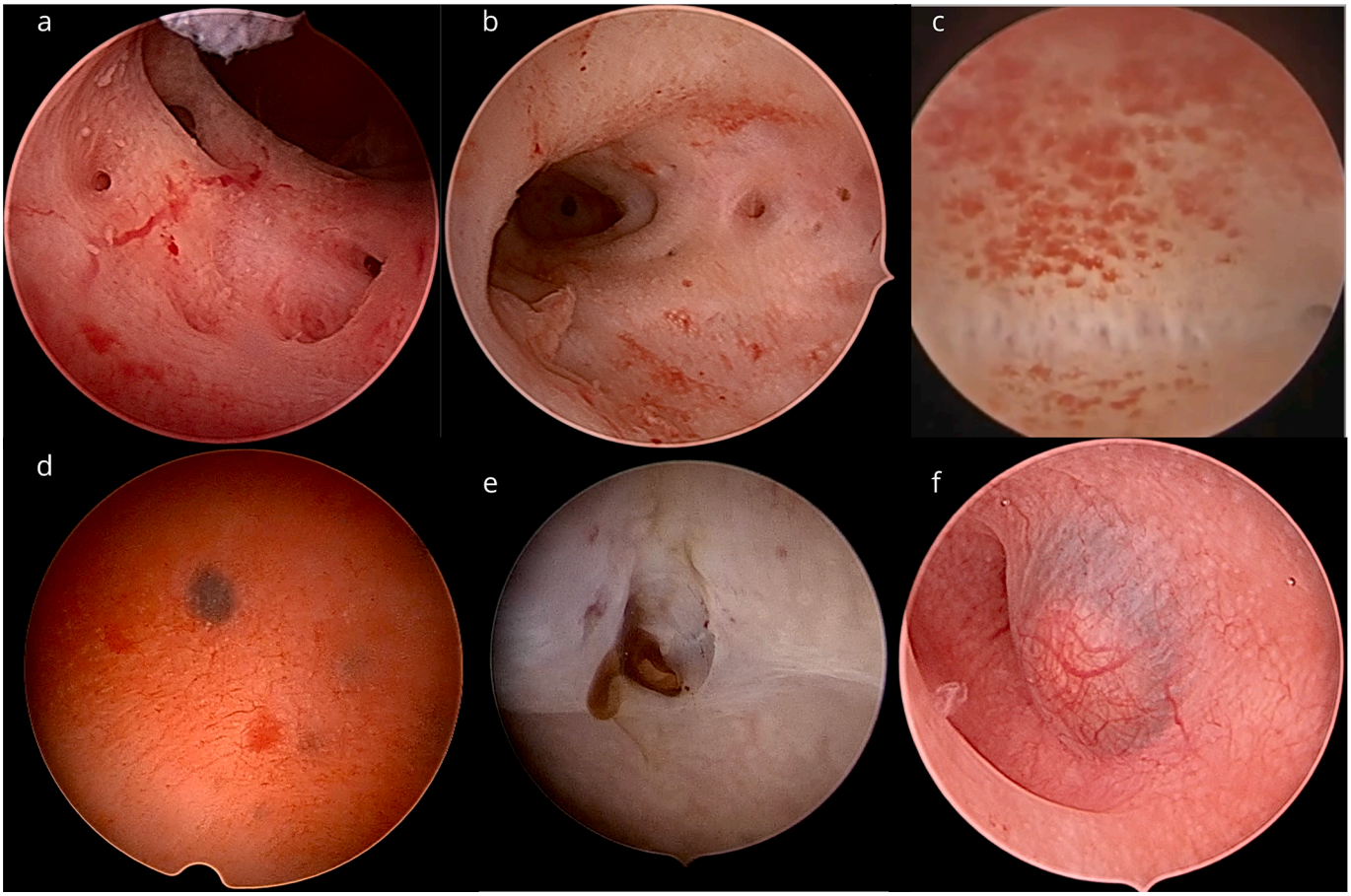


Fig. 3. Hysteroscopic view of adenomyosis: (a-b) Tiny adenomyotic openings scattered across an irregular endometrial surface; (c) Typical 'strawberry' pattern of the endometrium, characterized by hyperemia with bright red areas surrounding white central dots; (d-e-f) Hemorrhagic cystic lesions exhibiting a dark blue to chocolate brown appearance.

or a combination of internal myometrial thickening, areas of focal or diffuse disease, and involvement of the outer myometrium [39]. Depending on its extent, adenomyosis is categorized into two types: diffuse adenomyosis, where the endometrial glands or stroma are widely distributed within the myometrium, and focal adenomyosis, where nodular aggregates are circumscribed to a well-defined area. In rare instances, it can present as a large cyst, termed an adenomyotic cyst or cystic adenomyoma [39].

Although the evidence is not conclusive, adenomyosis generally has a detrimental effect on fertility and decreases the success of assisted reproductive technologies [40]. This is thought to be due to changes in the typical architecture of the myometrium and its function caused by dysfunctional uterine peristalsis and decreased endometrial receptivity [40]. In some cases, adenomyosis can be found in uteri with congenital anomalies, especially in infertile patients [41].

Hysteroscopy is a valuable adjunct diagnostic tool for evaluating adenomyosis, offering the advantage of direct visualization of the uterine cavity and the option of collecting targeted biopsy samples under direct visualization [13,42]. Diagnostic hysteroscopy, although unable to definitively diagnose adenomyosis due to its limited ability to visualize pathology beyond the endometrial surface layer, provides evidence suggesting the condition through the observation of irregular endometrium with small openings, pronounced hypervascularization, an endometrial pattern resembling a 'strawberry', hemorrhagic cystic lesions that appear dark blue or chocolate brown, fibrous cystic lesions following multiple episodes of intra-myometrial hemorrhage [43] (Fig. 3). In patients with adenomyosis, hysteroscopy can reveal an irregular endometrial vascular distribution pattern during the proliferative and secretory phases. These superficial vascular anomalies can only be adequately evaluated by decreasing the intracavitary pressure during the procedure [43]. Accurate identification of uterine adenomyosis during hysteroscopy is influenced by the menstrual cycle phase in which the examination is conducted. It is recommended to perform the procedure in the early post-menstrual phase when the lesions are more likely to impact the uterine cavity, enhancing the likelihood of a correct diagnosis [44]. Hysteroscopy also allows the collection of biopsy samples from the endometrium and underlying myometrium using mechanical instruments like biopsy or grasping forceps, scissors, or bipolar electrodes [45]. Alternatively, an instrument called 'Spirotome' (Bioncise NV, Wellen, Belgium) can be used to collect samples from the sub-endometrial myometrium, providing additional information regarding the depth of penetration of the pathology [46].

Hysteroscopy is not the primary treatment option for adenomyosis but is a valid surgical option in selected cases of superficial sub-endometrial adenomyosis. It is used to evacuate cystic hemorrhagic lesions or enucleate superficial focal adenomyomas less than 1.5 cm in diameter [13,43,46]. Mechanical instruments and/or 5 Fr bipolar electrodes or a 15 Fr resectoscope can be used for these procedures [43].

Superficial diffuse adenomyosis may be treated with variable success using endomyometrial ablation (endomyometrectomy). This technique differs from classical endometrial ablation as it involves resecting not only the endometrium but also the superficial myometrial layers up to visualize healthy myometrium, concluding with the coagulation of endometrial residues [43]. Endomyometrectomy utilizes 3-mm or 5-mm straight loops for ablation of the fundus and cornual recesses, and classic cutting loops for the uterine walls [43]. The degree of intramyometrial

extension of the pathology correlates with the duration of surgery, its technical difficulty, and risks such as incomplete removal of adenomyotic tissue, excess fluid intravasation, uterine perforation, and intraoperative hemorrhage. A second-stage surgical procedure may be necessary for persistent or recurrent disease. During preoperative assessment, using ultrasound, meticulous care must be taken to evaluate the thickness of the myometrium between the outer margin of adenomyosis and the uterine serosa [43].

Endomyometrectomy may cause the dissemination and proliferation of ectopic endometrial cells, promoting the progression of the pathology and the development of new adenomyosis [47]. As an adjunct to surgery, or as an alternative option, local progestin-based therapy with a levonorgestrel-releasing IUD can be used [48,49]. The continuous controlled release of levonorgestrel directly at the uterine mucosal level may induce regression of adenomyotic lesions and relieve pain symptoms by targeting progestin-sensitive endometrial cells [48].

Cervicitis

Cervicitis, defined as inflammation of the cervix, can result from various causes, including infections, chemical irritants, or physical trauma. Common infectious agents include *Chlamydia Trachomatis* and *Neisseria Gonorrhoeae* [50]. While cervicitis has been linked to infertility, the evidence supporting this connection is still limited and not entirely conclusive [50,51]. Nonetheless, it is plausible that the inflammatory state associated with cervicitis could negatively affect sperm by being toxic to them. Additionally, if the inflammation leads to the narrowing or destruction of cervical crypts, it could potentially reduce sperm lifespan and interfere with their hyperactivation [50,51]. Despite the need for additional studies, clinicians should consider the cervix as a potential site of pathology when evaluating patients for infertility, especially since cervicitis is often asymptomatic and can be easily overlooked [50,52,53]. While much of the focus in infertility evaluations tends to be on the cervical canal and the uterine cavity, it is crucial to also assess the cervix itself for signs of chronic inflammation. Hysteroscopy can be instrumental in identifying subtle signs of cervicitis that might otherwise go undetected, such as hyperemia or engorged capillaries on the cervix [52]. Since cervicitis is often asymptomatic, it is frequently noted as an incidental finding during hysteroscopy. However, attention to this subtle finding could have a significant impact in cases of unexplained infertility [52].

Chronic endocervicitis can sometimes be complicated by endocervical ossification. Although the precise mechanism by which endocervicitis leads to endocervical ossification remains unclear, it is hypothesized that the release of increased intracytoplasmic calcium following the death of cervical cells may trigger further calcium deposits, leading to the formation of macroscopic bone structures [54]. During hysteroscopy, the diagnosis of endocervical ossification and chronic endocervicitis can be supported by the presence of white, sponge-like endocervical fragments that have a hard consistency upon contact with the hysteroscope [54].

Cervical and tubal endometriosis

Cervical endometriosis, though less common than endometriosis in other locations, can still present significant clinical challenges. This rare condition, with an incidence between 0.11 % and 2.4 %, affects women of reproductive age [55]. Clinically, it may manifest through symptoms such as heavy bleeding, dyspareunia, dysmenorrhea, and chronic pelvic pain [56–58]. During hysteroscopic examination, cervical endometriosis is diagnosed by the presence of small mucosal lesions on the ectocervix or endocervix, often associated with oozing hemorrhagic material [37]. Polypoid forms of this condition can closely mimic cervical polyps, making histopathological evaluation essential for a definitive diagnosis [59].

Similarly, tubal endometriosis is a cause of tubal infertility, found in 25 % of patients undergoing assisted reproductive technology (ART) [60]. Studies have shown that patients with endometriosis experience lower implantation rates, with gamete transport, fertilization, and embryo implantation being impaired due to the inflammatory environment created by endometriotic lesions [60,61]. The inflammatory cytokines secreted by these lesions and the oxidative stress they induce can affect sperm motility and lead to apoptotic events in the embryos [60,61].

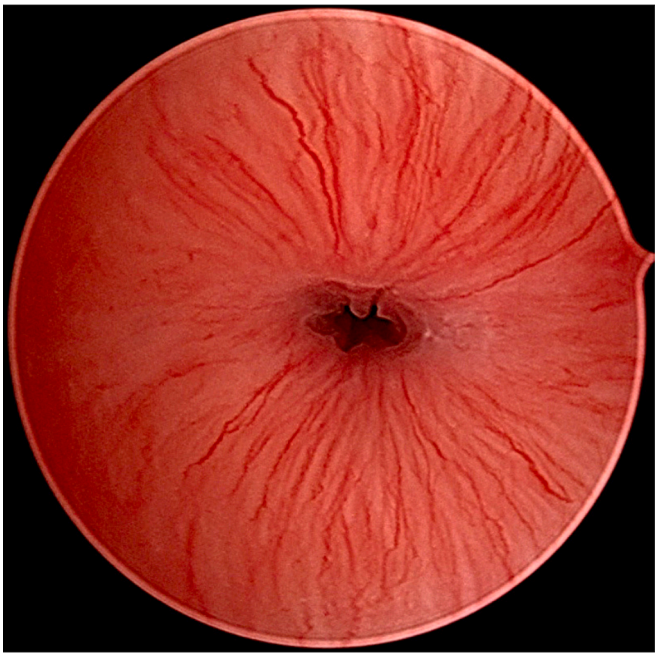


Fig. 4. The “tubal ostium sunhine” for chronic salpingitis: pronounced vascularity forming a ’sunshine’ pattern around the tubal ostia.

Endometriotic lesions on the serosa of the Fallopian tube are typically not visualized on ultrasonography, computed tomography, or magnetic resonance imaging, even when the tubes appear patent [62]. Diagnosis of tubal endometriosis generally involves laparoscopy with direct visualization and histopathological confirmation with biopsy [61]. However, a less invasive diagnostic approach might include hysteroscopy, where the observation of brown fluid emerging from the tubal ostium when intrauterine pressure is decreased could suggest the presence of intrinsic tubal endometriosis [61,63].

Tubal ostium sunshine

Abnormalities of the Fallopian tubes, such as tubal occlusion, acute and chronic salpingitis, hydrosalpinx, peritubal adhesions, and salpingitis isthmica nodosa, are significant contributors to female infertility, accounting for 30 % to 40 % of all cases [64]. A distinctive hysteroscopic finding associated with chronic salpingitis is the “*Tubal Ostium Sunshine*”, characterized by pronounced vascularity forming a ’sunshine’ pattern around the tubal ostia [64] (Fig. 4).

Tubal polyps

Tubal polyps, also referred to as cornual polyps, have been sporadically reported in gynecologic literature, with a widely varying prevalence ranging from 1.2 % to 33 % [65]. These polyps are ectopic islands of normal endometrial tissue located within the interstitial portion of the fallopian tube. During hysteroscopic evaluation, it is crucial to closely inspect the tubal ostia to identify the presence of these polyps (Fig. 5), as they are associated with infertility, with reported rates between 20 % and 61.5 % [65]. Hysteroscopic polypectomy can be performed using mechanical and electrosurgical systems; however, this procedure

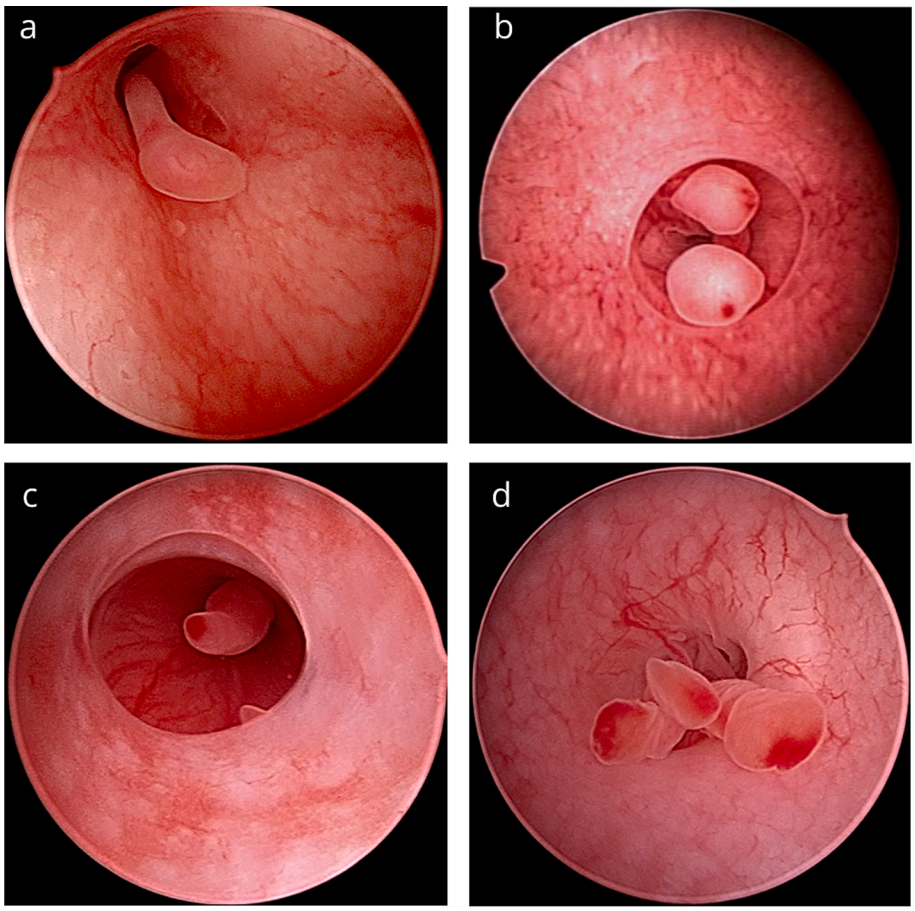


Fig. 5. Tubal Polyps: (a-b) Tubal polyp protruding from the right tubal ostium; (c-d) Tubal polyp protruding from the left tubal ostium.

requires careful technique due to the higher risk of complications in this area, such as uterine perforation or tubal damage [66]. Some studies have demonstrated a significant increase in pregnancy rates following the resection of polyps located at the uterotubal junction, as opposed to other areas of the uterine cavity [67,68].

Endometrial abnormalities

Chronic endometritis

Chronic endometritis (CE) is a condition marked by inflammation of the endometrium. This inflammation can either follow an acute phase, which is the more commonly observed type of CE, or it may emerge subtly as chronic inflammation from the onset, bypassing any acute stage [69].

For years, the clinical significance of CE has been debated. However, increasing evidence now suggests that CE can adversely affect fertility, contributing to implantation failure and recurrent pregnancy loss [70]. This is primarily due to an inadequate microenvironment that disrupts normal implantation and endometrial receptivity [70].

Clinical examination and transvaginal ultrasound are often nonspecific for diagnosing CE [71].

Hysteroscopy can effectively identify signs of endometrial inflammation, especially when performed during the proliferative phase of the menstrual cycle. According to the criteria proposed by Cicinelli et al. [72] the hysteroscopic diagnosis of chronic endometritis is characterized by several features: hyperemia (Fig. 6 a-b), where the vascular network appears accentuated, especially at the periglandular level; stromal edema (Fig. 6 c), where the endometrium presents as both pale and thickened during the proliferative phase; micropolyps (Fig. 6 d-e-f), manifesting as small pedunculated vascularized protrusions of the uterine mucosa (less than 1 mm) covered by endometrium and characterized by an accumulation of inflammatory cells, including lymphocytes, plasma cells, and eosinophils, scattered with normal stromal cells [72]. CE is often categorized as a subtle lesion for several reasons. Despite the importance of standardized criteria for diagnosis, some practitioners may interpret hysteroscopic findings as non-pathological. Additionally, CE can sometimes present as unclear or indistinct hysteroscopic images, leading to frequent misdiagnosis. Given these challenges, the definitive diagnosis of CE traditionally relies on endometrial biopsy and histopathological examination, where the presence of endometrial stromal plasma cells is a key diagnostic marker [71]. However, identifying these plasma cells using conventional staining methods (such as hematoxylin-eosin) can be difficult, as they are often indistinguishable from endometrial stromal fibroblasts and monocytes [73]. The use of immunohistochemical staining for CD-138 (syndecan-1), a specific plasma cell marker, has been shown to improve diagnostic accuracy and reduce the risk of misdiagnosis [74]. It is also important to note that CE may have an asymmetric distribution within the endometrium, meaning that a blind biopsy could miss the affected areas. For this reason, it is crucial for clinicians involved in the workup of infertile patients to recognize the hysteroscopic signs indicative of this condition. The treatment currently considered most effective for restoring fertility in cases of CE is the administration of oral antibiotics [75].

Thin endometrium

Thin endometrium, also known as refractory or atrophic endometrium, has been observed in up to 9 % of patients undergoing Assisted Reproductive Technology (ART) [76]. This condition can arise from

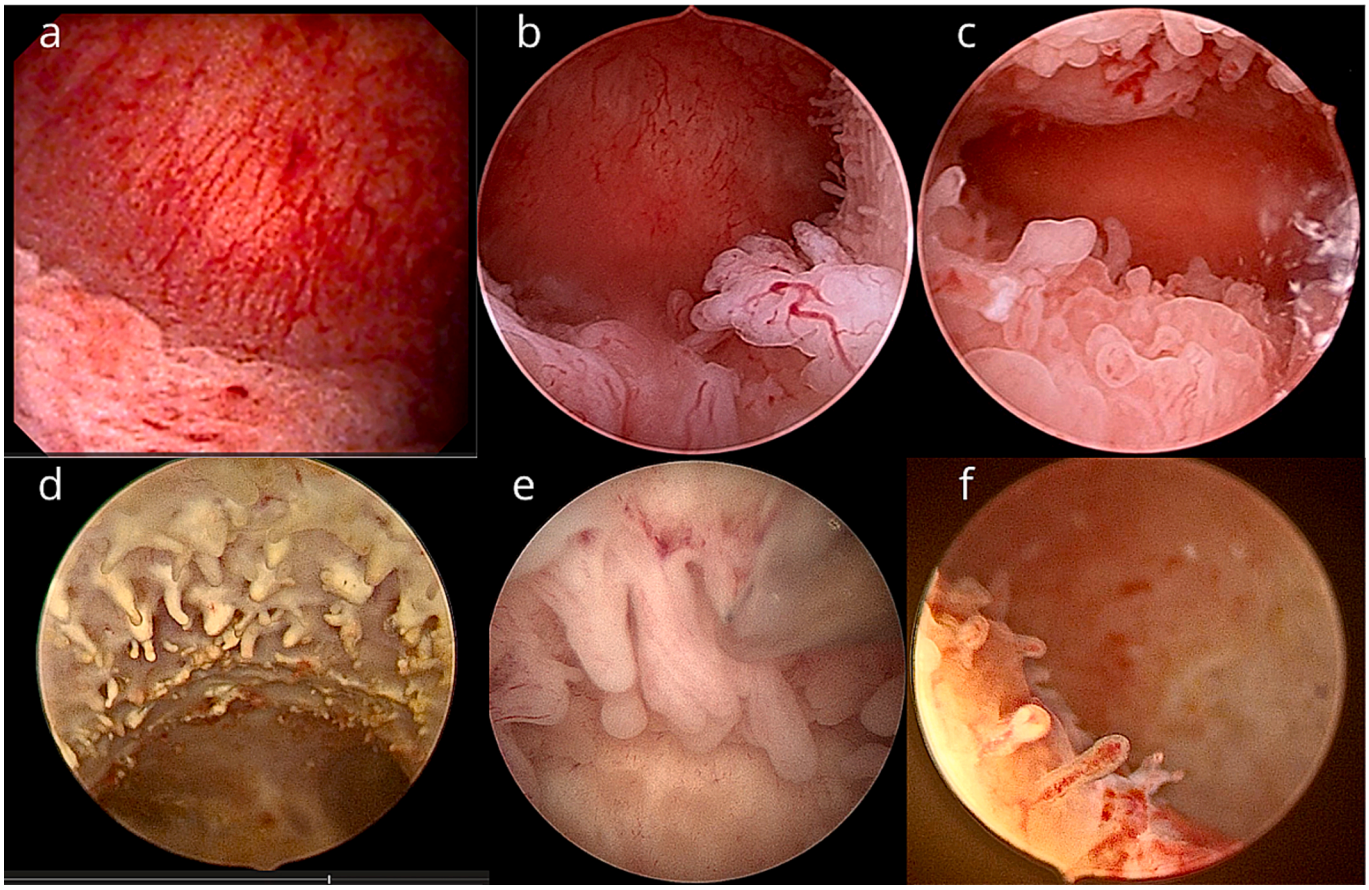


Fig. 6. Hysteroscopic appearance of chronic endometritis**:** (a) The endometrial mucosa displays diffuse hyperemia; (b) The posterior uterine wall is covered with micro-polyps, and the fundus shows endometrial hyperemia; (c) Stromal edema accompanied by micro-polyps; (d-e-f) Micro-polyps covering various uterine walls.

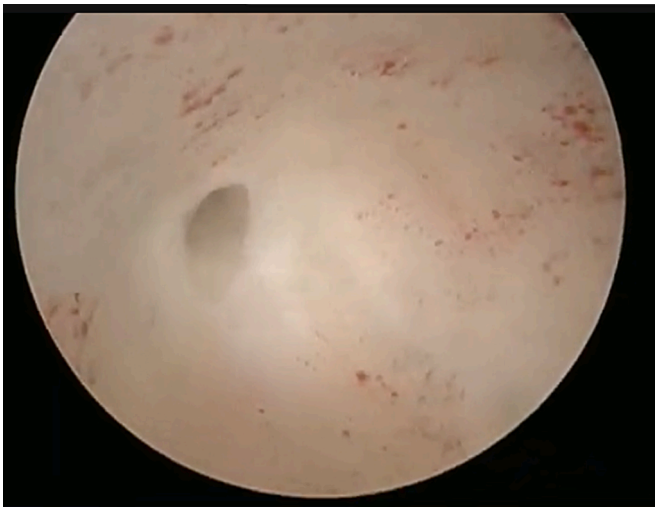


Fig. 7. Irregular "thin" endometrium with subendometrial hemorrhage.

various factors, with inflammatory and iatrogenic causes being the most common [77]. Additionally, poor vascularity and low estradiol levels can contribute to insufficient endometrial growth, and in some women, the endometrium may be inherently thin [76]. Inflammatory causes, such as acute or chronic infections, can destroy the basal layer of the endometrium [78]. Iatrogenic causes, which are related to medical interventions, can also cause a thin endometrium [77]. Repeated or vigorous curettage during surgical procedures, such as hysteroscopic myomectomy, polypectomy, or laparoscopic myomectomy, can damage the basal layer, potentially leading to intrauterine adhesions formation [79].

Idiopathic thin endometrium can also occur without any clear precipitating disease. This can be due to an individual's unique uterine structure or intrinsic endometrium properties that affect its growth [77]. A thin endometrium is associated with implantation failure due to its lack of the structural characteristics typical of a receptive endometrium, making it unable to provide the necessary environment for embryo implantation [77].

During hysteroscopic evaluation, a thin or atrophic endometrium appears smooth and thin, with visible deep endometrial vessels—an unusual feature not seen in healthy endometrial tissue. (Fig. 7). Hysteroscopy is a highly sensitive method for diagnosing atrophic endometrium [61].

Histopathological examination of biopsy samples typically reveals reduced endometrial glands and densely compacted stromal cells [61]. In ultrasonographic assessments, an endometrial thickness of less than 7 mm indicates a refractory or thin endometrium associated with lower implantation, pregnancy, and live birth rates [77,78].

Endometrial osseous metaplasia

Endometrial osseous metaplasia is a rare condition, with nearly 200 cases documented in the literature. It is characterized by the presence of mature or immature bone tissue within the endometrium [80]. The most widely accepted hypothesis regarding its pathogenesis suggests that it derives from embryonic remnants, with a process of endochondral ossification occurring from omnipotent endometrial cells [80]. Another proposed mechanism involves metaplasia of mesenchymal tissue, often

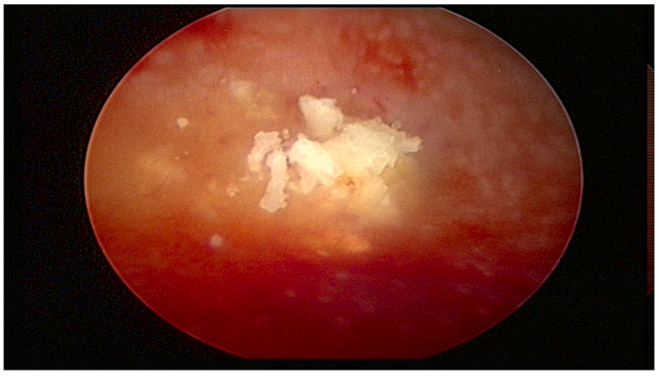


Fig. 8. Endometrial osseous metaplasia.

secondary to inflammatory processes in the endometrium, typically following an abortion [81]. Interestingly, a few cases in the literature have reported endometrial osseous metaplasia in women who have never been pregnant [81,82]. In such instances, the pathogenesis may be linked to heterotopia, where mature endometrial stromal cells undergo metaplasia [81,82].

Clinically, patients with this condition may present with symptoms such as dysmenorrhea, pelvic pain, menstrual irregularities, or, more commonly, infertility [80,81]. Ultrasound imaging often reveals a hyperechoic endometrial appearance, prompting suspicion of this diagnosis [83]. However, the gold standard for diagnosis is hysteroscopy, which typically reveals multiple osseous fragments within the uterine cavity, often partially embedded in the endometrial mucosa [84,85] (Fig. 8). The removal of these lesions is performed by hysteroscopy using mechanical forceps or bipolar electrodes. For larger osseous fragments, a resectoscopic approach is generally preferred to ensure complete removal [84,85].

Endometrial calcifications

Endometrial calcifications are rare clinicopathologic findings observed frequently in menopausal women but can also be found in women of reproductive age [85]. Clinically, this condition is often associated with infertility and abnormal uterine bleeding [86,87]. Most commonly, endometrial calcifications are identified as secondary to osseous metaplasia, dystrophic calcification of necrotic tissue, or calcified foci within uterine fibroids [86]. These calcifications can also be linked to infectious diseases, such as chronic endometritis caused by Mycobacterium tuberculosis[86]. Although the exact significance of endometrial calcifications remains unclear, their association with infertility suggests a potential compromise of endometrial function [87]. Hysteroscopy is an essential diagnostic tool for diagnosing this condition and can be highly effective in removing the affected tissue (Fig. 9) [85].

Conclusion

The diagnosis and management of subtle lesions during hysteroscopy present significant challenges due to their elusive nature and the complexity of their clinical implications. It is essential for gynecologists to have a clear understanding of the endoscopic characteristics of these subtle lesions and their potential impact on fertility. By recognizing these nuances, gynecologists can significantly enhance the diagnostic accuracy of hysteroscopy, ensuring that even the most subtle abnormalities are identified and appropriately managed. This attention to detail is crucial in optimizing the quality of care for infertile women.

The integration of advanced ultrasound techniques, such as 3D endovaginal ultrasound, with hysteroscopy can further enhance the detection and management of subtle lesions. Ultrasound can provide preoperative insights into uterine abnormalities, enabling targeted hysteroscopic evaluations. Additionally, correlating ultrasound findings with hysteroscopic observations allows for a more comprehensive understanding of subtle lesions, optimizing both diagnostic accuracy and clinical outcomes. This synergistic approach can be particularly valuable in identifying conditions that may complicate reproductive treatments, such as cervical stenosis or subtle malformations.

To advance the field of reproductive medicine, it is essential to develop and adopt a standardized, universally accepted terminology and classification system for these infertility-related subtle lesions. Such standardization would improve diagnostic consistency and treatment strategies across the field. Moreover, despite their rarity, clinical trials are necessary to validate the effectiveness of treating these subtle abnormalities.

In light of these challenges, refining residency and fellowship programs is crucial to ensure that trainees are well-versed in recognizing and understanding these lesions., even if they do not encounter them directly during their education. Training should emphasize the importance of keen observation and attention to detail because “seeing what is commonly unseen can make the difference!”.

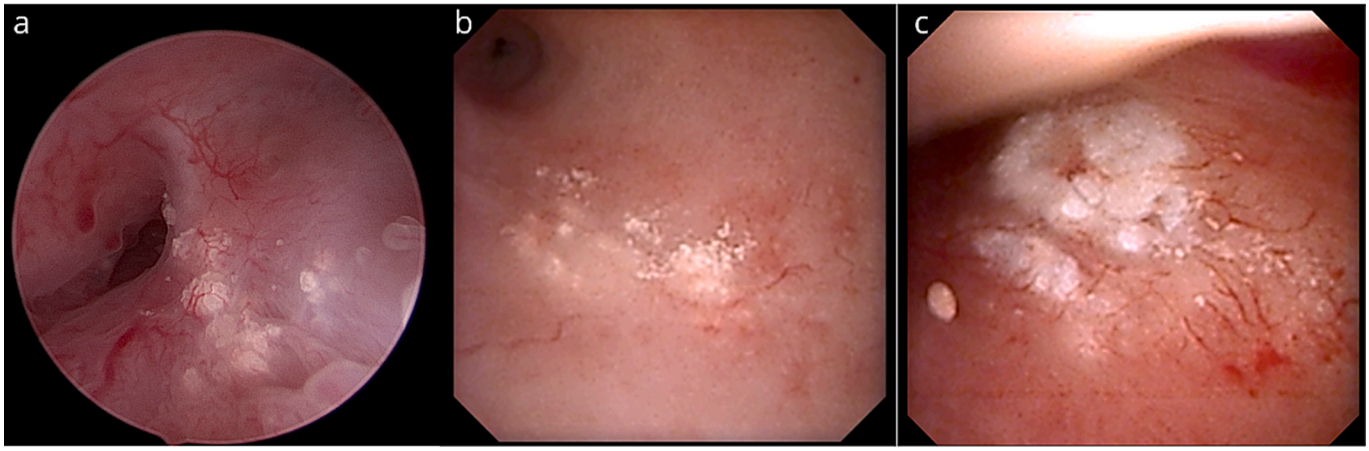


Fig. 9. Endometrial Calcifications: (a) Diffuse white plaques visible on the endometrial surface. These plaques are indicative of endometrial calcifications, which are scattered irregularly across the cavity. The surrounding endometrium appears mildly inflamed, with visible blood vessels suggesting underlying chronic inflammatory processes; (b) Concentrated region of white, plaque-like calcifications on the endometrial surface. These calcifications are distinctly separated from the surrounding tissue, which shows signs of mild erythema, suggesting possible chronic irritation or previous inflammatory activity; (c) Cluster of endometrial calcifications where the white, dense lesions are more pronounced. The calcifications are grouped together, creating a textured appearance on the endometrial lining. The surrounding tissue exhibits a reddish hue, likely due to inflammation or vascular changes associated with the calcification process.

Funding sources

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

CRediT authorship contribution statement

Stefania Saponara: Writing – review & editing, Writing – original draft, Investigation, Data curation, Conceptualization. **Stefano Angioni:** Writing – review & editing, Methodology, Investigation, Conceptualization. **John Preston Parry:** Writing – review & editing, Resources, Data curation. **Luis Alonso Pacheco:** Writing – review & editing, Resources, Data curation. **Jose Carugno:** Writing – review & editing, Investigation. **Nash Moawad:** Writing – review & editing, Methodology, Investigation. **Sergio Haimovich:** Writing – review & editing, Resources, Formal analysis. **Ricardo Bassil Lasmar:** Writing – review & editing, Resources, Formal analysis. **Davide Dealberti:** Writing – review & editing, Validation, Formal analysis. **Ece Aksakal:** Writing – review & editing, Investigation. **Bulent Urman:** Writing – review & editing, Validation, Methodology. **Luigi Nappi:** Writing – review & editing, Validation, Methodology. **Salvatore Giovanni Vitale:** Writing – review & editing, Writing – original draft, Supervision, Project administration.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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