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Exploring the Role of Anatomical Factors in Female Infertility: A Review

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Abstract: *The main causes of female infertility include endometriosis, congenital/acquired uterine abnormalities, and post-infectious tubal damage. Septate uterus with myomas and synechiae may cause infertility, miscarriage, and other pregnancy issues. Pelvic inflammatory illness causes most tubal injuries. Surgery may cure tubal factor infertility with reproductive results equivalent to in vitro fertilization. Endometriosis, a common gynecologic condition, may cause pain and infertility in reproductive-age women. Immunological, genetic, and environmental factors may induce endometriosis-related infertility. The condition's cause is unclear. Despite its ubiquity, endometriosis' causes are unknown. Certain medical, surgical, and psychological treatments for endometriosis may enhance quality of life. In most cases, endometriosis surgery increases fertility. Endometriosis and the immune system are linked, therefore future treatments may use immunologic ideas.*

Keywords: Anatomical Causes, Management Strategies, Patient Care

I. INTRODUCTION

Anatomical anomalies: The most prevalent cause of female infertility?

Endometriosis, uterine myomas, congenital uterine malformations, tuboperitoneal abnormalities, and other rare reproductive system anomalies may cause female infertility.

Infertility assessments find tuboperitoneal involvement in 25%–35% of women [1,2]. Pelvic inflammatory disease (PID) causes most tubal damage. Monitoring data shows that 1.7% and 8% of UK and US women between 16 and 46 receive PID diagnoses annually, 15% of Swedish women will receive a diagnosis, and over a million American women receive PID treatment annually. Reports show 12% tubal infertility after one PID episode, 23% after two, and 54% after three. A recent review of 24 articles from the US and Europe found that up to 18% of women in these countries may lose their ability to conceive after showing signs of PID from any source [3]. STIs such Chlamydia trachomatis cause most PID in high-income countries [4]. Because tubal disease is generally asymptomatic, women seldom know they have it unless their medical history is taken. Other causes of tubal damage include postsurgical adhesions and endometriosis (stage III or IV).

Endometriosis affects 5%–15% of fertile women. Even while 20% to 25% of affected women are asymptomatic, the illness may cause pain and infertility. The cause of endometriosis-related infertility is unclear, although a complex interaction of immunological, genetic, and environmental factors is recognized, with mechanical factors prevailing in the late stages.

Miscarriages or infertility due to acquired (myomas and synechiae) or congenital (septate uterus) uterine cavity deformations indicate implantation failure [5]. Congenital uterine anomalies may cause infertility, early labor, poor fetal presentation, and pregnancy loss. Septate uteri, the most common defect, causes pregnancy losses of 60% and fetal survival rates of 6% to 28% [6, 7].

Uterine myomas affect 20%–50% of reproductive-age women. Submucous or intramural myomas hinder spontaneous conception and IVF [8]. Obliterating the uterine cavity with intrauterine synechiae, or adhesions, may induce subfertility and hypomenorrhea or amenorrhea. Synechiae may delay placental tissue removal or need repeated curettages following spontaneous miscarriage in 40% of women [9]. The cause of infertility is sometimes unknown despite all efforts.

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Post-infectious tuboperitoneal causes: Current impact of infertility

PTO, periadnexal adhesions, and DTO are post-infectious tubal damage (Fig. 1). Tubal patency testing before fertility therapy is the gold standard for infertile women [10,11]. When skilled, hysterosalpingography or contrast sonography may assess tubal patency in women without PID [11,12]. To avoid unnecessary IVF and embryo transfer, diagnostic laparoscopy should be done when results are abnormal [13]. When endometriosis, periadnexal adhesions, or tubal illness are present, laparoscopy is the preferred method for evaluating tubal factor infertility. Consider laparoscopy ahead of intensive empirical therapies with high costs and hazards [11].

Proximal tubal occlusion

In 10% of tubal diseased women, proximal tubal occlusion develops [12]. A true pathological occlusion from post-infectious fibrosis, an obstruction due to technical artifacts like cervical seal adequacy, intrauterine pressure, uterine-tubal ostium spasm, thick endometrium acting as a valve, or plugs of amorphous material of unknown etiology, often appearing t Between 42% and 95% of women identified with PTO do not have the illness [15,16].

A retrospective research by Al-Jaroudi et al. [16] examined reproductive results in women who had selective tubal catheterization for bilateral PTO. In 98 infertile women with bilateral PTO, a second hysterosalpingography was performed before selective tubal implantation. Twelve women had unilateral tubal patency, while 72 had bilateral PTO. Recanalization of both tubes was accomplished in 25 (34.7%) and at least one tube in 44 (61.1%) of 72 women who had selective tubal catheterization. Follow-up showed 23 conceptions within 24 months.

After 12, 18, and 24 months, the cumulative chance of conception was 28%, 59%, and 73%. Some individuals who failed tubal recanalization may have had a genuine occlusion due to fibrin scarring from salpingitis, endometriosis, or prior surgery. The standard of therapy is microsurgical excision of the occluded tubal part and tubocornual anastomosis of the patent distal tube to the intramural portion. 1, 2, and 3 years following surgery, live birth rates were 27%, 47%, and 53% [17].

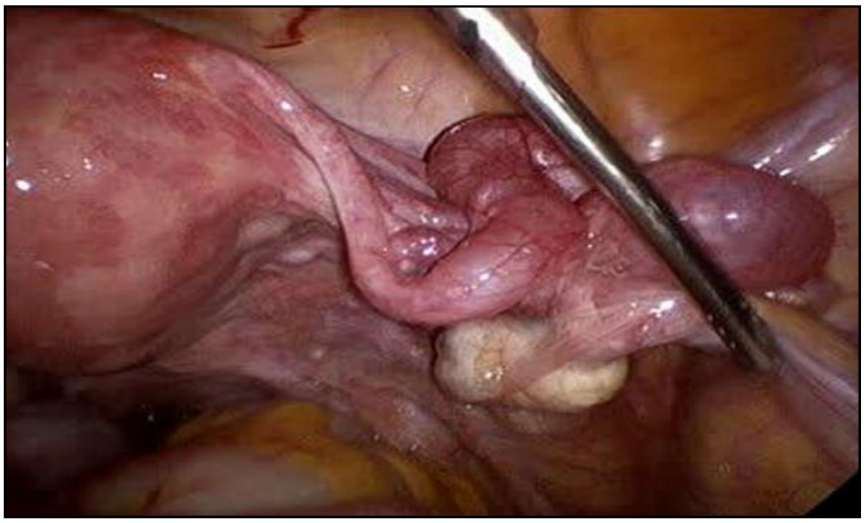

Fig. 1. Distal tubal occlusion of the right fallopian tube with mild periadnexal adhesions.

Marana et al. [18] reviewed 9 case series of 187 PTO patients and found a 49% term pregnancy rate and 4% ectopic pregnancy risk following laparotomy. The outcomes are comparable to IVF [10,18].

Periadnexal adhesions

Operative laparoscopy is the best salpingo-ovariolysis method. In non-selected individuals, laparoscopic salpingo-ovariolysis resulted in 51% to 62% intrauterine pregnancy and 5% to 8% ectopic pregnancy [10]. Salpingoscopy, the direct evaluation of the tubal mucosa by a dedicated, small-caliber endoscope during laparoscopy, is the most important prognostic factor for reproductive outcome after salpingo-ovariolysis, according to recent prospective studies. [19–23].

Brosens et al. [20] and Marana et al. [21–23] found that 80% of periadnexal adhesion patients have normal tubal mucosa, 70% will have a term pregnancy after laparoscopic salpingo-ovariolysis, and most pregnancies occur within a

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year. Most patients with periadnexal adhesions have retained tubal mucosa, therefore salpingectomy is not needed until hydrosalpinx causes extensive tubal damage.

Distal tubal occlusion

Marana et al. [24] found a 33% cumulative pregnancy rate per patient with microsurgical laparotomic salpingoneostomy in 10 investigations with 1128 participants. The pregnancies were 77% intrauterine, 61% term, 23% ectopic, and 15% spontaneous abortions.

A meta-analysis of 5 nonrandomized controlled trials compared laparotomic microsurgical tubal surgery with laparoscopic DTO therapy [25]. 138 (28.9%) of the 478 laparotomic patients and 104 (30.9%) of the 336 laparoscopic patients had intrauterine pregnancies. No significant difference was seen in intrauterine pregnancy rates between groups. Three trials provided sufficient information to compare surgical procedures at various stages of tubal disease. In the mild tubal disease categories, 83 (32.8%) of 253 laparotomy patients and 96 (39.5%) of 243 laparoscopy patients had intrauterine pregnancies. No significant change was seen in intrauterine pregnancy rates.

According to the Practice Committee of the American Society for Reproductive Medicine [26], surgical DTO therapy for moderate tubal illness (which accounts for 25% of DTO patients) has live birth rates of 39% to 59% and ectopic pregnancy rates of 4% to 10%. Ectopic pregnancy rates are comparable after reconstructive surgery and IVF (4%–10% versus 1%–13%) [26].

Schippert et al. [27,28] examined pregnancy rates in surgically treated women with mild or moderate acquired tubal illness. For salpingoneostomy, adhesiolysis, and tubal sterilization reversal, term pregnancy rates were 65%, 70%, and 80%. Ectopic pregnancy rates varied from 1% to 10% depending on tubal illness, although they were fewer than 10% among women who reversed tubal sterilization. Results after IVF varied from 2.1% to 11%.

In women having salpingoneostomy or salpingo-ovariolysis, the tubal mucosa on salpingoscopy is the most significant predictive indicator for reproductive outcome. Brosens [20] and Marana et al. [21–23] found that 35% to 45% of DTO patients had normal tubal mucosa and 65% will have a term pregnancy following laparoscopic salpingoneostomy. However, salpingectomy may be superior for badly injured tubal mucosa. The authors of this study recently disclosed a simplified salpingoscopy approach for women with DTO, introducing a small-caliber hysteroscope via an auxiliary trocar during laparoscopy [29].

Tubal reconstructive surgery vs IVF

IVF is the main treatment for tubal infertility, however tubal reconstructive surgery is still common. Many couples reject IVF for moral, spiritual, or financial grounds. Remember that surgery cures women with normal tubal mucosa, whereas IVF bypasses tubal injury. These ladies can conceive spontaneously and frequently without medication. They may also undergo pregnancy and delivery like women without tubal infertility, but without the hazards of IVF, such as OHSS, multiple pregnancies, preterm deliveries, and congenital abnormalities. Tubal surgery has little risks, including anesthesia and surgical problems, unlike IVF, which may cause OHSS [30]. Ovulation induction may cause this deadly disease. Intravascular depletion from OHSS may cause hemoconcentration-induced thrombosis, hypovolemia, dehydration, and electrolyte abnormalities. OHSS occurs in 1%–10% of IVF cycles, with 0.25%–2% having severe cases [31].

The 2007 American Society for Reproductive Medicine registry [32] analyzes assisted reproductive technology practices and outcomes since 2001 and shows a 27.2% live birth rate per cycle. In 2010, European statistics showed a clinical pregnancy rate of 29.0% per retrieval [33]. Although the European report lacks data to compute the live birth rate per cycle, a range of 21.0% to 22.5% may be inferred. Italy's latest statistics shows 16.8% live births each cycle [34]. Singleton, twin, and triplet births are 64.1%, 32.0%, and 3.7% in the US and 79.2%, 19.9%, and 0.9% in Europe after IVF. Because of this, the primary difficulty with IVF internationally, compared to spontaneous conception, is still multiple pregnancies, which increase the risk of preterm birth, cesarean delivery, and other complications [14, 35].

Their experiment had significant dropout rates, with 74% after the first unsuccessful effort, 61% after the second, and 69% after the third. This is significant to Sharma et al. [36]'s cumulative IVF pregnancy rates (66% after 4 rounds). Psychological stress and disappointment are the main reasons people quit treatment after many attempts [37].

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Recent studies have shown that IVF pregnancies, even singletons, have poor outcomes [38]. Birth abnormalities were 30–40% greater and low birth weight, premature delivery, and perinatal mortality were twice as prevalent as in spontaneously conceived pregnancies [38–49]. A 2010 Danish study [50] evaluated 20,166 singleton pregnancies. After adjusting for mother age, BMI, educational attainment, smoking status, and alcohol/coffee usage during pregnancy, IVF patients had roughly four times the risk of stillbirth compared to spontaneous conceivers.

IVF outcomes are improving, but tubal reconstructive surgery is still a good option for many couples. Surgery should be the initial step in diagnosing and treating tubal infertility. Surgical success depends on proper diagnosis and patient selection.

Endometriosis in the 21st century

Endometriosis contains endometrial glands and stroma outside the uterus [51]. Endometriosis affects 5–15% of reproductive-age women. Endometriosis causes dysmenorrhea, significant dyspareunia, pelvic pain, abnormal uterine bleeding, digestive difficulties, and infertility [51]. Endometriosis is more frequent in women with pelvic pain or infertility (40%–60% vs. 20%–30%) [52]. Laparoscopic endometriosis imaging and histology are the best diagnostic methods [53].

Three hypotheses explain endometriosis: embryonic origin [54], coelomic metaplasia [55], and the commonly recognized retrograde menstruation hypothesis [56], when endometrial pieces develop into the peritoneal cavity. It is unknown how endometriosis causes pain and infertility, although therapies targeting progesterone resistance, systemic immunological dysfunction, angiogenesis, inflammation, neurotropism, and pain transmission, including neuropathic pain, have been suggested [57].

Several authors have explained endometriosis' immunological system [58,59]. Endometrial cell development is promoted by peritoneal macrophage activation, decreased T and NK cell cytotoxicity, increased pro-inflammatory cytokines and growth hormones, and altered cellular immunity. Proliferation, inflammation, and angiogenesis result from these cells [60-63]. Recent study shows adult uterine stem cells, menstrual fluid, and endometrial implants beyond the uterus. Endometriosis may be caused by stem cells [64].

Peritoneal, ovarian, and deep-infiltrating endometriosis [65,66]. Endometriosis may infect the rectovaginal septum, retrocervical region, sigmoid, rectum, ureters, and bladder with lesions beyond 5 mm [67]. Minimal, mild, moderate, and severe are the American Society for Reproductive Medicine [68] classifications.

Cancer antigen 125 from human epithelial carcinoma is the most studied marker. It is a serum marker for endometriosis but has little diagnostic relevance [69,70]. Due to imaging advances, transvaginal ultrasonography is preferable for endometriosis diagnosis [71-73].

Current therapies tackle symptoms, not the disease. Medical, surgical, and psychological treatments may help endometriosis sufferers. Nonsteroidal anti-inflammatory medications, oral contraceptives, gonadotropin-releasing hormone agonists, danazol, and progestins relieve pain [74].

The cause of endometriosis-related infertility is uncertain. Altered folliculogenesis, ovulatory failure, decreased granulosa cell preovulatory steroidogenesis, sperm phagocytosis, poor fertilisation, early embryonic development toxicity, faulty implantation, and oocyte alterations [63]. Cervical stenosis, uterine abnormalities, PTO, DTO, and perimenbrial and peritubal adhesions may cause infertility (75).

Interesting, 50% of conception troubles are caused by male spouses or both. The most important test for male infertility is semen analysis [76].

Endometriosis-related infertility treatment is difficult. Although endometriosis treatment may ease pain, it does not improve infertility. Randomized clinical trials and meta-analyses have demonstrated that medical therapy alone is unsuccessful and that medical therapy with surgery is not better than surgery alone [77,78]. There are no randomized clinical trials comparing non-treatment to surgery for expectant management. Contrary to this technique, some investigations demonstrate very low spontaneous pregnancy rates without medication [78]. Surgery may fix endometriosis-related infertility. Surgery substantially enhanced conception rates in mild endometriosis (stages 1 and 2) patients compared to diagnostic laparoscopy [79]. Surgery is better to expectant care for advanced disease because it may achieve 50% to 67% postoperative pregnancy rates (Fig. 2) without randomized clinical trials [80,81].

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Due to the difficulties of randomized study, infertile women with severe endometriosis have no consensus on surgery versus reproductive technology. In the solitary randomized trial, Bianchi et al. [82], surgery outperformed IVF without surgery. Infertility-associated deep endometriosis needs further investigation to identify its importance and treatment options. Darai et al. [83] found that laparoscopy was more likely than laparotomy to promote spontaneous pregnancy in severe colorectal endometriosis.

Congenital and acquired uterine causes

Most female reproductive tract problems are congenital uterine malformations. Incomplete müllerian duct fusion causes problems in 4% of fertile women [7]. Septate, bicornuate, and arcuate uteri predominate [7]. Few unicornuate and didelphys uteri exist.

Uterine anomalies may induce recurring miscarriage, preterm labor, poor fetal presentation, and infertility [6,7]. Although simpler, septate uterus, the most common malformation, has the poorest reproductive results, with pregnancy losses of over 60% and fetal survival rates of 6% to 28% [6].

Incomplete septum, arcuate, bicornuate, and didelphys are the most frequent uterine anomalies. Hysterosalpingography and hysteroscopy may show the interior geometry of a double uterine cavity, but the fundus must be examined to identify problems. Single for septate or arcuate uterus, double for bicornuate or didelphys. Traditionally, laparoscopy and hysteroscopy assessed the uterine exterior. Uterine abnormality diagnosis now uses magnetic resonance imaging and 3-dimensional ultrasonography instead of laparoscopy. Hysteroscopy with intraoperative 3-dimensional ultrasonography may reduce partial uterine septum removal [84].

After two spontaneous abortions, uterine malformation surgery was often advised. As surgery has grown less invasive, prophylactically when no spontaneous abortion has occurred, particularly in women with a septate uterus, and infertility has been related to various uterine anomalies, surgical repair has been done. Surgical hysteroscopy addresses septate and arcuate uteri. Cold scissors or monopolar or bipolar electrosurgery may treat the deformity hysteroscopically with similar results. Post-septum removal term delivery is 75% [7]. Hysteroscopic surgery cannot fix complex abnormalities, which increase reproductive results if untreated. Surgery requires laparotomy.

The most common benign tumors in reproductive-age women are uterine myomas (20–50%) [85]. Submucosal, intramural, and subserosal myomas distort the uterine cavity (Fig. 3) and protrude [86].

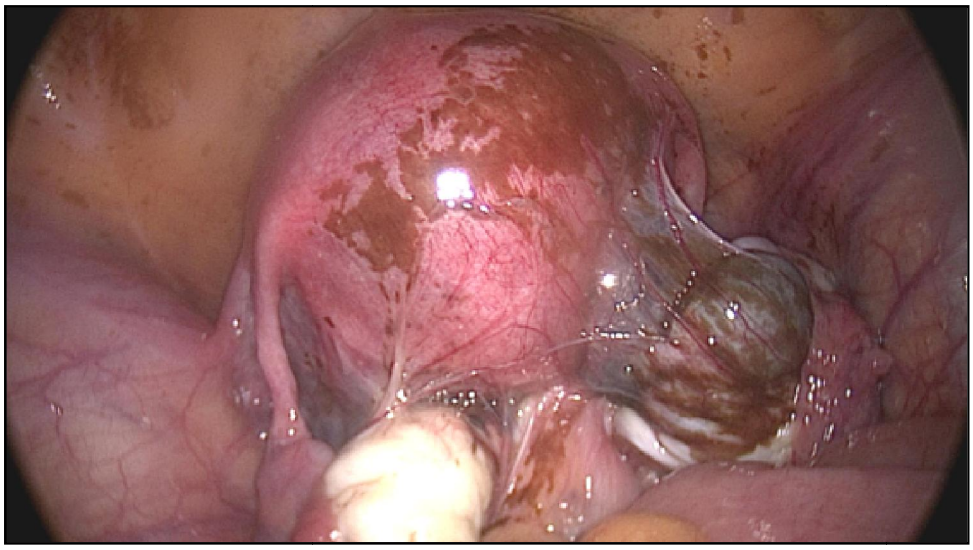

Fig. 2. Severe case of endometriosis, with bilateral ovarian-endometriomal adhesions and obliteration of the cul-de-sac.

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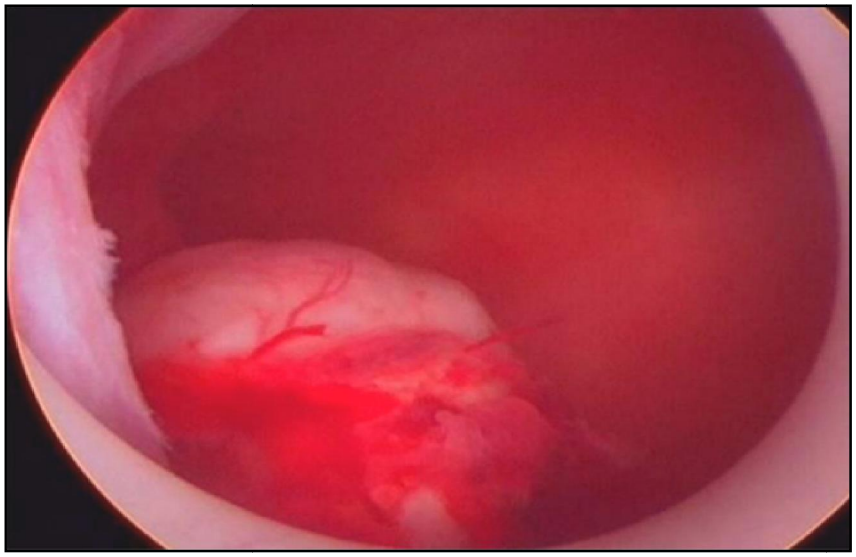

Fig. 3. Submucosal myoma with bleeding visualized during hysteroscopy.

Myomas can affect fertility through cervix displacement, uterine cavity enlargement, proximal fallopian tube obstruction, altered tubo-ovarian anatomy, uterine contractility, endometrial disruption, impaired blood flow, inflammation, and abnormal vasoactive substance secretion.

The tumors showed uterine myomas' main growth factors. MYOMAS have more estrogen and progesterone receptors, aromatase P450, and estrogen synthetase than the surrounding myometrium depending on the menstrual cycle [88,89].

Many women have asymptomatic uterine myomas. Signs include abnormal uterine bleeding, dysmenorrhea, pelvic pressure, pain, abdominal girth increase, urine or rectal symptoms, and reproductive failure [87]. By analyzing size, quantity, and location, transvaginal ultrasonography may determine whether a myoma should be treated hysteroscopically or abdominally [8,87]. Operative hysteroscopy is best for endometrial submucous myomas. Submucosal and intramural myomas that extend into the endometrial cavity diminish pregnancy and implantation rates but enhance them when removed in retrospective and case-control studies [90–92].

Gonadotropin-releasing hormone analogues before hysteroscopy may improve larger submucous myoma surgery [93]. Laparoscopy or laparotomy treat intraamural, subserous, and pustulated myomas, depending on amount and size. When fertility is not an issue and the patient accepts non-conservative treatment, hysterectomy may substitute myomectomy.

Intrauterine adhesions might completely block the uterine chamber. This affects 1.5% of infertile women [94]. Low menstrual flow and infertility are the most common symptoms [9,95]. Repeated curettage following abortions and delayed placental tissue removal may cause 40% of synechiae [96]. Diagnostic hysteroscopy detects intrauterine synechiae best [97].

The surgery is hysteroscopic adhesiolysis. Initial adhesion severity influences anatomy, menstruation, and pregnancy [95,97]. 3%–23% of adhesions reoccur, and severe adhesions are 20%–62% [95]. Physical and pharmacological supplements are widespread. These include endometrial estrogen stimulation, intrauterine contraceptive device insertion after surgery, Foley catheter insertion, or newer synthetic barriers that physically separate endometrial cavity walls [9,95,98–100].

Perspectives

Uterine congenital and acquired disorders may cause infertility and pregnancy loss. Diagnostic and treatment advances have improved care for women with uterine infertility. Better candidate selection for reconstructive tubal surgery in tuboperitoneal women may increase intrauterine pregnancy rates to 65%–70%. Complex endometriosis affects infertility management. Fertilization rates rise significantly following surgery. Despite its great incidence and huge physical, psychological, and economic cost, endometriosis' etiology is still unknown. There are evident links between endometriosis and the immune system, thus future treatments may involve immunological.

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II. CONCLUSION

Anatomical causes of female infertility, including uterine abnormalities, fallopian tube blockages, and ovarian issues, significantly impact a woman's ability to conceive. Understanding these underlying anatomical factors is crucial for effective diagnosis and management. Advances in medical imaging, such as hysterosalpin gography and laparoscopy, allow for precise identification of these issues, enabling targeted interventions. Management strategies may include surgical correction of anatomical abnormalities, assisted reproductive technologies like in vitro fertilization (IVF), or medications to stimulate ovulation. Addressing these anatomical causes not only enhances reproductive outcomes but also empowers women by providing them with informed choices regarding their fertility journey. Continued research and development of minimally invasive techniques hold promise for improving the management of female infertility and ultimately supporting women's reproductive health.

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