Endometrial receptivity in adenomyosis and/or endometriosis

Paul Pirtea, M.D., Dominique de Ziegler, M.D., and Jean Marc Ayoubi, M.D., Ph.D.

Department of Obstetrics, Gynecology and ART, Hospital Foch, Paris, France

A narrative review of endometrial receptivity in adenomyosis and/or endometriosis revealed that this parameter is difficult to assess in natural conception because both disorders alter natural fertility. Recent data emanating from assisted reproductive technology have allowed the study of endometrial receptivity in women affected by adenomyosis and endometriosis. This has upended our views on the effects of these 2 disorders on embryo implantation. Today, the very existence of altered receptivity in assisted reproductive technology is questioned. In this context, we now know that frozen euploid blastocyst transfers in estradiol and progesterone cycles have unaltered outcomes in both adenomyosis and endometriosis. (Fertil Steril® 2023;119:741–5. ©2023 by American Society for Reproductive Medicine.)

Key Words: Endometriosis, adenomyosis, endometrial receptivity, IVF failure, frozen embryo transfer (FET)

This narrative review is part of a series of articles focusing on the similarities and differences existing between adenomyosis and endometriosis and how both disorders affect human reproduction. The specific aim of this review is to analyze the effects of adenomyosis and endometriosis on endometrial receptivity.

Classically, adenomyosis and endometriosis have been considered disorders that affect fertility and receptivity. This belief notably stems from the presence of significant genic and proteomic alterations that have been documented in the eutopic endometrium, as discussed further in this article and in other articles of this series. The past decade, however, has brought new elements that offer a more discriminative view of the specific effects of endometriosis and adenomyosis on endometrial receptivity. Indeed, we can now distinguish the effects that these disorders exert on both natural fertility and endometrial receptivity to embryo implantation in assisted reproductive technology (ART). Therefore, our review will focus on the consequences of adenomyosis and endometriosis on these 2 parameters, natural fertility, and the actual ART outcome. We will also determine whether certain measures may either enhance embryo implantation chances, be useless, or have detrimental effects in the case of adenomyosis and endometriosis. Likewise, we will review whether different ART protocols and/or possible pre-ART treatments may impact the ART outcome in the case of adenomyosis and/or endometriosis.

MATERIALS AND METHODS

We searched all publications listed in PubMed over the past 5 years under the following keywords: "endometrial receptivity endometriosis" (90 hits); "endometrial receptivity adenomyosis" (27 hits); "IVF and adenomyosis" (49 hits); "IVF and endometriosis" (383 hits); "fertility and adenomyosis" (175 hits); and finally, "fertility and endometriosis" (1059 hits). All references were first checked according to their abstract. Ultimately, 187 articles were identified as pertinent, and the full articles were retrieved. Finally, 36 of these publications were quoted in the present manuscript together with 17 classic references.

THE CONCEPT OF ENDOMETRIAL RECEPTIVITY

Lessons Learned From Donor-Egg ART

Back in pre-ART times, uterine receptivity was believed to decline with age, as evidenced by the fact that older women had decreased pregnancy chances and increased miscarriage rates. At that time, this observation was logically taken as evidence for an age-related decline in the uterine capacity to host embryos. Of course, the advent of ART—with donor egg in its wake—has upended our understanding of endometrial receptivity and uterine function as a whole. We now know that when properly primmed—with estradiol (E_2), followed by progesterone—and in the absence of anatomical pathologies, uterine receptivity is only a little altered by advancing age (1). Hence, donor-egg ART taught us that uterine—endometrial—receptivity is practically constant throughout reproductive years. The parameters of endometrial receptivity defined through the donor-egg ART model have provided information of general value, which

Received February 13, 2023; revised March 1, 2023; accepted March 3, 2023; published online March 11, 2023.

P.P. has nothing to disclose. D.d.Z. has nothing to disclose. J.M.A. has nothing to disclose.

Reprint requests: Paul Pirtea, M.D., Hospital Foch, 131 Avenue de Malakoff, Paris 75116, France (E-mail: paulpirtea@gmail.com).

Fertility and Sterility® Vol. 119, No. 5, May 2023 0015-0282/$36.00

https://doi.org/10.1016/j.fertnstert.2023.03.004

notably allows us to study the impact of adenomyosis and endometriosis on receptivity.

The 2 primary lessons of donor-egg ART regarding receptivity are as follows: the necessity for sufficient estrogen priming is very permissive, with no difference in embryo implantation rates related to the duration (2, 3) and/or dosing of E_2 priming (4), within reasonable limits of course; and the duration of progesterone exposure controls the period of endometrial receptivity—also known as the window of receptivity (WOR)—after proper E_2 priming (5). The readily recognized efficacy of donor-egg ART has led to the use of the same priming regimens for timing frozen embryo transfers (FET), first with ovarian suppression with a gonadotropin-releasing hormone (GnRH) agonist (GnRH-a) (6) and later without it (7). Recent prospective data using transfers of frozen embryos indicate that the WOR is rather broad, contrary to prior reports. Indeed, randomized control trials have shown that embryonic endometrial synchrony is attained when transferring cleavage-stage embryos on days 3–5 (8) and blastocyst-stage embryos on days 5–7 of progesterone administration (8, 9). In reality, it is likely that the WOR is determined solely by the days of progesterone administration and that the key is attaining embryonic endometrial synchrony.

Assessing Endometrial Receptivity

The WOR has been linked to histologic changes taking place in the endometrium, as assessed by biopsies specimens examined according to changes seen in the menstrual cycle (10). E_2 and progesterone supplement cycles used in donor-egg ART actually showed slight differences in endometrial morphology as compared with the menstrual cycle findings on the 6th day of the luteal phase. Indeed, Navot and Bergh (11) reported a slight delay in endometrial gland development—the persistence of subnuclear vacuoles on the 6th day of progesterone—and advanced predecidualization of the stroma, as compared with the menstrual cycle.

More recently, endometrial changes induced by progesterone and, in particular, the control of WOR have been studied by genic expression assessed in endometrial biopsies. This has led to the development of new modes of receptivity determination and, notably, the so-called "endometrial receptivity assay" (12). Despite monumental publicity, there is now evidence that the endometrial receptivity assay test is of no practical use for determining whether embryos will implant or not and/or for "personalizing" embryo transfers according to the duration of progesterone exposure (13, 14).

Other modalities for testing of endometrial receptivity have been proposed. One is based on the overexpression of B-cell lymphoma 6 (BCL6) protein in the endometrium (15). This too failed to truly predict endometrial receptivity when frozen euploid blastocysts were transferred. Indeed, Klimczak et al. (16) reported that embryo implantation and live birth rates (LBRs) in patients with BCL6 positive biopsies did not significantly differ from those in patients whose biopsies were negative. Chronic endometritis, which has an increased prevalence in endometriosis (17), does not seem to affect implantation rates after transfers of euploid blastocysts in E_2 and progesterone cycles (18). Still, other endometrial receptivity tests have been proposed for assessing endometrial receptivity (19, 20). Yet no proof of efficacy exists regarding this too because none of these tests have been evaluated by proper randomized control trials (19, 20). Considering that the very existence of altered endometrial receptivity is seriously questioned, as discussed further in this article, these latter receptivity tests (19, 20) should be considered practically useless.

Disordered Endometrial Receptivity: Myth or Reality?

Patients undergoing embryo transfers after ART and failing to become pregnant are prone to blame themselves—their uterus—for the failure. Indeed, women failing ART are led to believe that if the "nice" embryo that was transferred in their womb did not implant, the problem must originate from their endometrium. This belief has been the starting point for proposing numerous treatments and measures—collectively called "add-ons"—in the case of failed ART. Yet, there is now mounting evidence that in more than 95% of ART cases, failed implantation in a woman whose uterus is anatomically and functionally normal, as per ultrasound assessment, is rooted in embryo quality and not endometrial receptivity (21–24). Indeed, successive euploid embryo transfers, up to 3, lead to practically identical sustained implantation rates—with ultrasound fetal heart activity—and a cumulative sustained implantation rate of 95%. De facto, this amounts to practically exclude that the vast majority of women who fail ART and suffer from altered endometrial receptivity (21). Hence, modern data indicate that impaired endometrial receptivity—if it really exists in rare cases—probably plays no or little (in $\leq$5% of cases) role in ART failures (22). Therefore, this also applies in the case of endometriosis and adenomyosis. In this context, it is understandable that the slew of proposed "add-ons," none of which has been adequately tested, are essentially useless and some possibly harmful (22, 25).

ENDOMETRIAL ALTERATIONS IN ENDOMETRIOSIS AND ADENOMYOSIS

The article by Bulun et al. (26) in this series of Views and Reviews on adenomyosis and endometriosis sums up in detail the most recent understanding of endometrial changes encountered in these 2 disorders. In both cases, the eutopic endometrium is markedly altered by inflammation-related changes, namely through gene mutations in the glandular epithelium and epigenetic alterations in the endometrial stroma (26). In summary, these changes amount to local production of E_2 by in situ CYP-19 activation and some degree of progesterone resistance. At first glance, the sum of these changes would lead to the belief that endometrial receptivity is altered in the case of adenomyosis and endometriosis. Yet, recent ART data suggest otherwise. Indeed, as discussed further in this article, ART data accumulated in the last years fail to confirm that endometrial receptivity is altered in adenomyosis (27) and endometriosis (28).

FERTILITY IN ADENOMYOSIS AND ENDOMETRIOSIS

The links between endometriosis and infertility, albeit not absolute, are well established and rarely challenged. Classically, endometriosis is encountered in nearly 40% of infertile women, whereas its prevalence is only 10% in the general population of reproductive age (29, 30). Today, the primary mechanism by which endometriosis is believed to hamper the chances of natural conception is the pelvic toxic condition that interferes with sperm-oocyte interactions (31). This concept outlines that inflammatory conditions prevailing in the pelvic cavity—with an elevation of a slew of cytokines and other markers of inflammation—constitute a toxic environment for gametes and early-stage embryos (31).

Numerous reports have concurred to indicate that surgery enhances the chances of natural conception (32). Conversely, all currently available medical treatments—using GnRH-a, oral contraceptives, or progestins—have no proven effect on natural fertility. All these medical treatments are indeed contraceptive. Hence, for any of these treatments to actively enhance the chances of natural conception, you would have to postulate the existence of a rebound of fertility after stopping such treatments. Although long hoped for, an increase in natural fecundity after stopping the medical treatment of endometriosis does not occur (33).

In endometriosis, it is difficult to assess the role of endometrial receptivity in women who fail to conceive naturally. Indeed, as mentioned earlier in this article, the primary disorder responsible for infertility in endometriosis is believed to be an effect on gamete interactions that cause delayed toxic effects on the developing embryos (31). Hence, we cannot determine whether endometriosis affects endometrial receptivity in the case of natural conception.

The issue of adenomyosis and natural fertility is more complex. The primary observation regarding the prevalence of adenomyosis and fecundity has linked histologically proven adenomyosis at the time of hysterectomies to multiparity (34, 35). However, this association between adenomyosis and multiparity has been challenged in a recent epidemiological study (36). Moreover, Mishra et al. (37) recently reported an observation linking adenomyosis and infertility. This directly stems from the fact that adenomyosis can now be diagnosed noninvasively (38–41). One in 10 women with subfertility has a diagnosis of isolated adenomyosis with a prevalence that varies according to coexisting endometriosis and/or fibroids (37).

ENDOMETRIAL RECEPTIVITY IN ADENOMYOSIS AND ENDOMETRIOSIS

Classic Beliefs

Endometriosis has long been blamed for altering the ART outcome by affecting both oocyte quality and endometrial receptivity to embryo implantation. These purported effects were believed to parallel the infertility associated with endometriosis. Adenomyosis was also described to alter the ART outcome (42, 43), possibly even increasing the harm caused by endometriosis to endometrial receptivity (44). Classically, endometriosis found during diagnostic laparoscopies—previously performed nearly routinely in fertility workups—was surgically removed for optimizing the ART outcome. It was only approximately a decade ago that surgery was seen to not improve the ART outcome (45). However, this dominant view, which prevails nowadays, is challenged by some investigators (46), including certain surgeons (47). Moreover, mounting evidence arose suggesting that surgery for endometriosis is potentially prone to harm the ovarian reserve, even more than the disease itself (48, 49).

Recent ART Evidence

As mentioned earlier in this article, Hamdan et al. (28) were the first to largely report that even the presence of endometriomas did not alter ART results. Today, several reports have indicated that the transfer of euploid blastocysts emanating from women with endometriosis provides a similar outcome—LBR, miscarriage rates—to that seen in disease-free controls (50, 51). Likewise, adenomyosis diagnosed on ultrasound did not alter implantation rates after euploid embryo transfers (52). However, it is important to note that all these latter studies report transfers of euploid blastocysts conducted in hormone replacement cycles using progesterone administered by intramuscular injections. The ovarian suppression induced by the E_2 and progesterone hormone replacement regimen may indeed play a role in suppressing endometrial alterations linked to ovarian function in endometriosis and adenomyosis.

A Call for Action?

In light of the number of publications detailing alterations of the eutopic endometrium in the case of adenomyosis and/or endometriosis, it is important to query whether certain measures may counteract such effects. For example, Juárez-Barber et al. (53) reported that women with adenomyosis have an altered expression of genes involved in decidualization. Because progesterone resistance has been described as the hallmark of uterine alterations encountered in the case of adenomyosis and endometriosis, these investigators questioned whether increasing progesterone doses may be beneficial. Their results indicate that adjusting progesterone doses before FETs did not improve the ART outcome in patients with adenomyosis. Other molecular mechanisms beyond progesterone resistance may be involved in implantation failure (53). In short, no measure has been reported to modify endometrial receptivity in cases of adenomyosis and endometriosis, which appears to not be altered anyway in case of frozen transfers in E_2 and progesterone cycles (50–52).

Differences Between Various ART Treatments

Originally, ART was conducted under the enduring belief that long ovarian suppression by GnRH-a for 3–6 months improved ART outcome in the case of endometriosis, as originally reported by Surrey et al. (54). Subsequently, a similar benefit of ART outcome has been reported with 6–8 weeks of pretreatment with oral contraceptives (55). More recently, however, GnRH-antagonist protocols have been widely

adopted in ART notably because it decreases the risks of ovarian hyperstimulation syndrome while maintaining similar ART outcomes.

In endometriosis, we have reported that ovarian stimulation for ART using an antagonist protocol and triggering of the final step of ovulation with a GnRH-a did not increase endometriosis-related symptoms (56, 57). The use of GnRH-a for triggering ovulation is a wise step in the case of endometriosis to avoid cyst formation in a context where endometriomas may exist. Furthermore, the use of GnRH-a for triggering ovulation also implies implementing a freeze-all and deferred embryo transfer approach, also used in the case of preimplantation genetic testing for aneuploidy. Indeed, embryo transfers conducted in E_2 and progesterone cycles may have a beneficial effect on endometrial receptivity. Of note, a few reports, however, suggested that GnRH-a protocols offer higher pregnancy and LBR than those offered by GnRH-antagonist protocols (58, 59). Today, European Society of Human Reproduction and Embryology guidelines have revoked the recommendation for prolonging GnRH-a suppression before ART (60).

CONCLUSION

Numerous alterations take place in the eutopic endometrium in case of adenomyosis and endometriosis. This has led to questions and suspicions that endometrial receptivity is altered in adenomyosis and endometriosis. However, endometrial receptivity cannot be tested in natural conceptions notably because in vivo endometriosis alters oocyte-sperm interactions and early-stage embryo quality. On the contrary, ART allows to test the chances of embryo implantation, and recent data have changed our views on this topic. Indeed, contrary to earlier beliefs, the chances of embryo implantation transferred in E_2 and progesterone replacement cycles appear to not be altered in case of endometriosis or asymptomatic adenomyosis. It is important to note the possible benefit of FETs conducted in E_2 and progesterone cycles (56–58). Hence, until more is known, we recommend that a freeze-all and deferred embryo transfer approach be opted for cases of adenomyosis and endometriosis.

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