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Adenomyosis and Pregnancy: A Review

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ABSTRACT
Studies have shown that ectopic EM glands that develop into the myometrium are the cause of the prevalent uterine illness known as AD. Studies have concluded that an increasing number of women of reproductive age are being diagnosed with AD, which now ranges from 20% to 35% of these women. Numerous studies have found that the hormonal changes that take place during pregnancy may have an impact on the appearance of AD, which may be diffuse, focal, or cystic. Furthermore, studies have concluded that awareness of these imaging changes during pregnancy is required in order to correctly diagnose adenomyosis, a benign illness that may fool doctors into thinking it is a major placental and myometrial abnormality. Thus, in our review, we have discussed AD and PG in terms of etiology, histopathology, complications and radiography.
Keywords: *AD, PG , Etiology, Histopathology, Complications, Radiography, EM.*

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INTRODUCTION

Studies have concluded that adenomyosis(AD) is a common benign ovarian disorder that affects 20% to 35% of women of childbearing age.[1] According to studies, it is identifiable by endometrial cells or stroma growing outside of the uterine myometrium.[2] Studies have concluded that changes may happen to these uterine glands on a regular basis during pregnancy(PG) and during your period.[3] Furthermore, studies have concluded that only about one-third of people with AD have any symptoms at all.[1] However, studies have concluded that, two-thirds of those people may have different symptoms, such as menorrhagia, dysmenorrhea, or metrorrhagia.[1] Studies have concluded that in the past, parous women aged 40 to 50 years old made up the bulk of adenomyosis patients; cases involving women younger than 40 years old were rare.[1] Results from prior research that relied heavily on hysterectomy specimens for diagnosis are criticized for possibly skewing the results.[1] Studies have concluded that due to the development of less intrusive diagnostic criteria, AD has been diagnosed in younger women in recent years.[1] In women aged 18 to 30 who were symptomatic and undergoing US, diffuse AD was found in up to 34% of the cases.[4] An MRI investigation of symptomatic women aged 18 to 42 years old, both with and without an additional diagnosis of severe endometriosis, found a diffuse AD rate.[5] Studies have concluded that factors that contribute to the development of AD include advancing age, having multiple pregnancies, undergoing cesarean delivery, and terminating pregnancies.[1] Additionally, studies have concluded that conditions associated with high levels of estrogen, such as early onset of menstruation, obesity, and short menstrual cycles, can also increase the risk.[1] Studies have concluded that AD often occurs alongside other gynecologic conditions like fibroids, polyps, and endometriosis.[2] Therefore, research has shown that looking at adenomyosis by itself is hard because other health problems can affect its effects on symptoms, especially infertility.[2] There is some conflicting evidence, but it is believed that AD may be linked to infertility. This could be due to issues with sperm transport caused by dysfunctional uterine peristalsis or problems with decidualization, which can affect implantation.[5] Thus, in our review, we have discussed AD and PG.

ETIOLOGY & HISTOPATHOLOGY

Although studies have shown that two main ideas have been widely accepted, the precise cause of adenomyosis remains largely unknown.[1] Researchers have also looked into the idea that endometrial tissue can move into the myometrial junctional zone through the basalis layer. This idea has gotten a lot of attention from researchers, and most of them agree with it.[1] Additionally, according to studies by experts, a distressing event or other elements like pregnancy or surgical complications may have caused

the migration. Studies have also shown that having ectopic endometrial tissue(EM-T) in a certain place can cause inflammation, fibrosis, and an increase in the movement of the uterus. Studies have also shown that this tissue may contain both ducts and stomas. In addition to this, studies have concluded that these responses are believed to trigger additional EM migration by causing further damage in a cyclical manner, leading to more damage.[6] Another theory that has been put forward recently says that adenomyosis may be a birth defect caused by fetal Müllerian remnants settling in the junctional zone or endometrial stem cells differentiating within the myometrium. The idea is backed up by the fact that deep EM was found in the posterior outer wall of the uterus. Case reports of AD in people with the Mullerian development disorder Mayer-Roktansky-Koster-Hauser syndrome add to the evidence for this theory that it starts in birth.[6]

In addition to this, various studies have also shown that during pregnancy, hormonal changes occur due to the presence of progesterone and other complex molecular pathways. These changes lead to the decidualization of the EM, both within and outside the uterus. This process is like the way that decidualized endometriomas cause problems, which has been talked about in earlier studies.[7,8,9] Research has shown that decidualized (DD) EM is a commonly encountered complication that can pose a diagnostic challenge in pregnant patients. Thus, studies have also shown that, in this theory, AD has the potential for similar DD.[1] More than that, different studies have also shown that the ectopic endometrium is what causes adenomyosis to look abnormal under a microscope. Studies have also shown that ectopic EM produces hyperplasia and the expansion of myometrial smooth muscle.[1] Additionally, researchers have discovered that ectopic EM and abnormalities in smooth muscle hyperplasia and hypertrophy are the main causes of imaging signs of AD [10]. Besides this, research has shown that myometrial cysts and cystic adenomyosis are likely caused by a disease process involving hormone-controlled growth and release that happens in cycles. [10] In addition to this, studies have concluded that the process behind the development of AD is unclear, given that it occurs in the non-functional basal layer of the endometrium. Studies have shown that AD, on the other hand, is more likely to be hormonally regulated or to have spontaneous bleeding.[10]

RADIOGRAPHICALLY

Researchers have found that patterns of adenomyosis in the uterus of women who are not pregnant have been documented in both US and MRI literature.[10] Studies have also shown that imaging results mostly show changes in fibrosis, smooth muscle hyperplasia, and hypertrophy around endometrial glands that are not in the uterus.[10] Studies have concluded that these patterns may be seen in non-gravid uteri that have not been pregnant. Several studies have also found that in 2015, the worldwide committee in charge of the Morphological Uterus Sonographic Assessment (MUSA) put together a list of the most common sonographic findings that should be reported when myometrial lesions are present.[11,12] Van den Bosch et al.,and others proposed a sonographic categorization system that is unique to AD.[12] Researchers used this system to demonstrate that findings can be general or specific, and they might also include myometrium cyst-filled areas.[13,14] Furthermore, research has shown that putting the condition into groups based on how the junctional zone looks and how involved it is doesn't help the pregnant patient because the junctional zone can't be seen after implantation. Some research has also shown that adenomyosis can show up in three different ways on a sonogram of a pregnant uterus: diffusely, focally, and cystically.[1]

COMPLICATION

Researchers have found that before and during pregnancy, adenomyosis is associated with a number of complications that may have an adverse impact on the patient as well as the developing baby.[1] Studies have concluded that complications may take many different forms, including infertility, early pregnancy loss, growth restriction, preterm delivery, and preeclampsia. Furthermore, studies have concluded that there are ideas for each of the direct effects of adenomyosis; however, the data does not support these concepts very well.[15] Studies have concluded that defective uterine peristalsis, morphological distortion of the uterus, and improper uterotubal transport are some of the possible explanations for the relationship between adenomyosis and infertility.[15] In a similar manner, studies have concluded that an abnormal uterine morphology is likely to have secondary effects that lead to early pregnancy loss. Studies have also found that these secondary effects include a problem with the metabolism of the endometrium, which can affect the early development of the placenta and the implantation of the gestational sac.[1] Researchers have found that fetal growth restriction and early delivery may be linked to more free radicals and inflammation in the uterus, as well as changes in the placenta and the amount of blood that can flow between the fetus and the mother.[15] This may happen through a process called vascular steal. Studies have concluded that growth restriction and preterm delivery may result in

significant unfavorable effects, such as lung immaturity, brain damage, and long-term postnatal health issues.[1]

IMPACT ON PREGNANCY

Studies have revealed that "adenomyosis is a recognized reproductive illness, and an increasing number of publications are demonstrating that the condition may affect not only fertility but also the outcome of a pregnancy".[16] This contradicts previous beliefs by different studies that the "disorder just affects fertility. Studies have also shown that adjusted odds ratio [aOR] 1.84 (95% confidence interval [CI] 1.32–4.31) and preterm premature rupture of membranes (aOR 1.98, 95% confidence interval [CI] 1.39–3.15) were shown to be higher in patients with adenomyosis".[16] "These discoveries were made after a late pregnancy had already been completed, and the results were analyzed. These results were confirmed in a small cohort of women who were diagnosed by ultrasound or MRI before becoming pregnant, showing a significantly higher risk of cesarean delivery (OR 4.5, 95% CI 2.1–9.7), small for gestational age (SGA) fetuses (OR 4.3, 95% CI 1.8–10.3), postpartum hemorrhage (OR 6.5, 95% CI 2.2–19.0), and fetal malpresentation".[17] According to recent "retrospective case-control research, adenomyosis is also associated with an increased risk of second-trimester miscarriage (OR 11.2, 95% CI 2.2–71.2), pre-eclampsia (OR 21.0, 95% CI 4.8–124.5), and placental malposition (OR 4.9, 95% CI 1.4–16.3) in pregnant women. These risks are associated with adenomyosis as well. These findings are the result of a meta-analysis of the individual studies that came before it".[18] Studies have also shown that the "increased rates of pregnancy-induced hypertension and uterine infection in diffuse-adenomyosis patients compared to those with focal-adenomyosis patients may have an impact on the outcome of the pregnancy".[19] Studies have revealed that "very recently, a prospective Japanese nationwide birth cohort study was published, and according to results obtained from self-reported questionnaires, adenomyosis was a risk factor for PTB of less than 37 weeks (aOR 2.49, 95% CI 1.89–3.41), PTB of less than 34 weeks (aOR 1.91, 95% CI 1.02–3.55), low birth weight of less than 2500 g (aOR 1.83, 95% CI 1.36–2.45), low birth weight of less than 1500 g (aOR 2.39, 95% CI 1.20–4.77), and SGA neonates (aOR 1.68, 95% CI 1.13–2.51)".[20] According to the studies, "pathogenic mechanisms described in one study, it is suspected to be responsible for obstetric issues such as adenomyosis, inflammation, increased myometrial prostaglandin production, altered uterine contractility, and increased intrauterine pressure, which may all be responsible for the association with PTB".[21] Additionally, studies also concluded that "activation of local and systemic inflammatory pathways was shown to occur in adenomyosis".[21] "Studies have also concluded that this activation had an effect on the interactions between the decidua and the trophoblasts early in the pregnancy, as well as the interactions between the chorion and the decidua, which had the potential to activate the PTB mechanisms later in the pregnancy".[21]

Studies have shown that obstetrical syndromes in women with adenomyosis may be caused in part by problems with myometrial spiral artery remodeling and deep placentation. Studies have also concluded that "these are two of the most common causes of these conditions. This is because the disease is associated with a greater rate of placenta-related disorders than the average pregnancy does. Studies have also concluded that, in fact, changes in the uterine JZ as well as the inner myometrium have been the subject of much research and have been described".[22,23] On the other hand, further research "processes are required from both an epidemiological and a physio-pathological point of view. Studies have also concluded that pre-pregnancy diagnoses of adenomyosis can only be made using uniform and shared imaging diagnostic criteria and classification, in fact. Studies have also concluded that, in a similar vein, there is a lack of understanding regarding the molecular mechanisms that lead to obstetric complications in adenomyosis. This is due to the fact that adenomyosis is a rather rare condition".[24]

IMPACT ON FERTILITY

Several studies have shown that "adenomyosis has been considered the typical uterine condition in multiparous women; nevertheless, an increasing amount of evidence suggests a connection with infertility and reproductive failure".[25,26,27,28] Studies have also concluded that a "recent cross-sectional study on infertile women found that the prevalence of adenomyosis was 24.4% in women over 40 years old and 22% in women under 40 years old. This percentage went up to 38.2% in cases of recurrent pregnancy loss, whereas it dropped to 34.7% in cases of past ART failure".[29]

Studies have shown that "adenomyosis is today considered to be one of the possible clinical presentations of infertility, and several theories have been given to explain the underlying mechanisms that are responsible for this disorder".[30] Studies have concluded that abnormal utero-tubal transport seems to be a significant mechanism that leads to infertility. This is because of morphological distortion of the uterine cavity as well as altered uterine peristalsis and sperm transport.[31] Studies have also concluded that this is "due to the fact that aberrant utero-tubal transport seems to be a significant contributor to

infertility as a mechanism. The JZ is the root cause of the faulty hyperperistalsis and high intrauterine pressure that are seen in the inner myometrium and, in particular, the JZ". In addition, when adenomyosis is present, ultrastructural myometrial abnormalities are what cause a disruption in normal myocyte contractility, which ultimately results in a loss of normal rhythmic contraction. This is because adenomyosis causes abnormalities in the ultrastructure of the myometrium.[32]

Studies have shown that when analyzed with an altered receptivity, the eutopic endometrium of infertile women who have adenomyosis shows a wide variety of molecular alterations.[33,34] According to studies ,"this includes altered pathways for sex steroid hormones, increased inflammatory markers and oxidative stress, reduced expression of implantation markers, a lack of expression of adhesion molecules, and an altered function of the gene for embryonic development (HOXA 10 gene), all of which can cause an impairment of implantation in women who have adenomyosis".[30]

Studies have shown that there aren't any easy-to-find studies on spontaneous conception in people with adenomyosis. However, some articles have looked at the "effects of adenomyosis in women who are getting ART or surgery for DIE, and their results are mixed".[35] Studies have also concluded that "Vercellini et al.'s 2014 meta-analysis found rates of miscarriages to be 31% in women with adenomyosis and 14.1% in women who did not have the condition (relative risk (RR) = 2.12, 95% confidence interval (CI) = 1.20–3.75)".[25] In contrast, studies have also concluded that the results of a "case-control study conducted on a group of women undergoing in vitro fertilization (IVF) showed that the implantation rate was not significantly impacted in those diagnosed with adenomyosis at TVUS but asymptomatic for AUB when compared to those who were not afflicted by the condition".[36] In a multicenter prospective study, "Mavrelos et al.[37] found that the chance of clinical pregnancy dropped from 42.7% in women who did not have adenomyosis to 22.9% in women who had four ultrasound signs of adenomyosis and 13.0% in women who had all seven ultrasound signs of adenomyosis".[37] This suggests that the severity of the illness, which is reflected in a number of morphological abnormalities on ultrasound, makes the reproductive result worse. Studies have concluded that the "deleterious impact of the uterine condition on reproductive results was corroborated by the findings of the most recent systematic review and meta-analysis on IVF treatment outcomes in adenomyosis".[26] This review and analysis included 11 studies and 519 patients with a TVUS or MRI diagnosis of adenomyosis.

Studies have shown that "women with adenomyosis have much lower rates of implantation, clinical pregnancy per cycle, clinical pregnancy per embryo transfer, continuing pregnancy, and live birth. On the other hand, they had a much higher risk of miscarriage".[38] The study also found that a "pre-IVF treatment including the use of gonadotropin-releasing hormone analog (GnRHa) down-regulation may be advantageous to the pregnancy rate".[38] The fact that the study found supported this "view in terms of the relationship between endometriosis, adenomyosis, and fertility, the findings of a systematic review and meta-analysis that was published in 2014 on women who had undergone surgery for rectovaginal and colorectal endometriosis reported a 68% reduction in the likelihood of pregnancy in women who wanted to become pregnant".[38] The study was conducted on women who had undergone surgery for rectovaginal and colorectal endometriosis. The study was conducted on "women who had undergone surgery for endometriosis in the colorectal and recto-vaginal regions".[39] The prevalence of adenomyosis was shown to have an impact on the rate of live births as well as the rate of miscarriages in a recent retrospective cohort study of women undergoing IVF. This study was conducted on women who had previously been analyzed. Adenomyosis patients, in particular, exhibited a significantly lower clinical pregnancy rate (26.4% as compared to 12.5%), as well as a significantly lower rate of live births.[39]

CONCLUSION

In conclusion, 20%–35% of women of reproductive age are affected by adenomyosis, a benign uterine illness that is increasingly discovered in young women. Given the trend toward later maternal ages and improvements in imaging tools, the occurrence of adenomyosis during pregnancy is seen with increasing frequency.The hormonal shifts that occur during pregnancy alter the appearance of adenomyosis, making it seem similar to other, more important myometrial and placental pathologic disorders, such as neoplasms. This is because AD is a benign condition. The appearance of AD during pregnancy may vary based on the patterns of distribution of the ectopic endometrial glands within the myometrium. These patterns include diffuse, focal, and cystic endometrial endometriosis. AD can also emerge during the early stages of pregnancy. Because AD in pregnancy is linked to adverse pregnancy outcomes like spontaneous abortion, preterm birth, and fetal growth restriction, as well as being a risk factor for preeclampsia and associated maternal complications, accurate diagnosis of the condition during pregnancy is essential for the health of both the fetus and the mother.

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