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Original Research Article

SARS-CoV-2 Mediated Placental Dysfunction and its Impact on Maternal Hormonal Homeostasis During Pregnancy

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Abstract: ***Background*:** Severe acute respiratory syndrome coronavirus 2 (COVID-19) can result in systemic inflammatory reactions that could disrupt endocrine and placental physiology in the course of pregnancy. The presence of the key pregnancy-supportive hormones, which are critical to keep the gestation and placental activity, includes progesterone, estradiol, and 2-human chorionic gonadotropin (2-hCG), whereas cortisol and thyroid-stimulating hormone (TSH) indicates the maternal stress and thyroid-regulatory pathways. Although the evidence of COVID-19-induced changes in physiological changes is accumulating, the impact of severe infection on maternal hormonal profiles during pregnancy is not defined well enough. ***Aim*** :This research was meant to assess the effect of severe COVID-19 on chosen maternal hormonal parameters in second-trimester pregnant women. ***Methods*** :The study was a hospital-based case-control study carried out at Al -Nasiriyah Teaching Hospital in Dhi Qar Province in the year 2020. It involved 200 pregnant women between the ages of 25 and 40 years with a gestation period of 1620 weeks. The sample was split into two groups of which 100 women were confirmed to have severe COVID-19 that needed oxygen therapy and 100 healthy pregnant controls. Each of the participants was a singleton, with comparable body weight (73 75 kg), and chronic disease or thyroid disorders were excluded. Blood samples were taken to the venous blood used to measure serum progesterone, estradiol (E2), 8-hcg, cortisol and TSH concentrations in the serum by the use of enzyme linked immunosorbent assay (Elisa) kit. The levels of hormones in the infected group were measured about one month after infection and statistical comparisons with the appropriate parametric or non-parametric tests were conducted on the levels. ***Results*** **:**The women with the worst cases of COVID-19 had much lower progesterone, estradiol and 2-hCG levels than the controls. On the other hand, the levels of cortisol were dramatically increased in the infected group, whereas the levels of TSH have decreased slightly but significantly. ***Conclusions*** **:**Major endocrine changes in severe COVID-19 in the second trimester comprise low placental hormones, high cortisol, and low TSH. These results indicate possible effects of a serious case of viral infection on placental endocrine functions and maternal stress mechanisms, and hormonal surveillance of such pregnancies is critical.

Keywords: SARS-CoV-2; COVID-19; pregnancy; second trimester; progesterone; estradiol; β-hCG; cortisol; TSH; maternal hormones.

1. INTRODUCTION

The worldwide manifestation of COVID-19 which was caused by Severe-acute respiratory syndrome coronavirus 2 has been met with immense health issues all over the world and has brought up issues concerning the vulnerable groups especially pregnant women. Pregnancy is associated with an intricate system of physiological, immunologic, and hormonal changes that could contribute to the predisposition to viral infections and the severity of the disease. A number of clinical reports have suggested that pregnant women infected with SARS-CoV-2 can develop more adverse outcomes than their non-pregnant counterparts, such as respiratory complications, higher rates of hospitalization, and negative obstetric outcomes such as preterm birth and preeclampsia [1-2]. The process of pregnancy is set with a complex endocrine control that promotes fetal development and maternal-fetal homeostasis. The

placenta mainly secretes hormones that include progesterone, estradiol and 2-human chorionic gonadotropin (2-hCG) and these are vital in pregnancy sustainment. The roles of progesterone, estradiol, and 2 -hCG in uterine quiescence, maternal immune tolerance, uterine blood flow, placental development, and corpus luteum functioning, respectively. The disruptions in such hormonal pathways can hinder the functioning of the placenta and worsen pregnancy complications. There is growing evidence that viral infections may cause endocrine pathway dysregulation by systemic inflammation, immune dysfunction, and potential direct endocrine gland or placental tissue actions [3-4]. The placenta is an important endocrine gland besides a physical barrier that separates maternal and fetal blood. Recent research studies have revealed SARS-CoV-2 infection in pregnancy could cause structural and functional changes in placental tissue. Defects of the vascularity, thrombosis, and villous edema as well as evidence of disrupted maternal-fetal circulation in the placenta of the infected mothers have been reported using histopathology. Pathological observations of this nature imply that the process of placental perfusion and endocrine functions could be impaired by the presence of viral infection, which can have an impact on the production and maintenance of pregnancy-related hormones [5-6]. Systemic endocrine pathways can also be impaired in response to SARS-CoV-2 infection in addition to placental hormones. The Hypothalamic-pituitary-adrenal axis (HPA) axis aids the secretion of cortisol that is important in physiological response to stress and infection. The state of severe illness and systemic inflammation is often related to an elevated level of cortisol which is the adaptive stress response of the body. It has been commonly noted that high cortisol levels are associated with patients having severe COVID-19 and can affect immune and metabolic functions. Viral infection may also affect thyroid functioning. During pregnancy, thyroid hormones are necessary in metabolism and neurodevelopment of the fetus. A number of the researches have documented changes in the thyroid parameters such as thyroid-stimulating hormone (TSH) among patients with COVID-19. Such changes can be caused by the activity of inflammatory cytokines, the systemic stress reaction, or the direct virus impact on the endocrine tissues [7-8]. The other critical process involved in SARS-CoV-2 pathophysiology is its connection with host cellular receptors, including angiotensin-converting enzyme-2 (ACE2). The ACE2 receptors are abundant in different tissues such as the placenta, ovaries and endometrium. Reproductive and endocrine functions can be affected by viral entry by these receptors. Recent literature has proposed that SARS-CoV-2 infection can impact the reproductive health of women by disruptions in hormonal control, ovarian activity, and menstrual cycles (915). Although the studies on obstetric outcome among pregnant women with COVID-19 are growing, the implications of SARS-CoV-2 infection on maternal hormonal patterns during pregnancy have not been thoroughly studied. The second trimester is a crucial phase of placental growth and hormonal level stabilization and any alteration of endocrine balance during this time may impact maternal well-being and fetal development. Thus, the current study experiment sought to examine how severe COVID-19 affects the subsequent maternal hormonal profiles such as progesterone, estradiol (E2), 9-hCG, cortisol, and TSH in second-trimester pregnant women who attended Al-Nasiriyah Teaching Hospital in Dhi Qar Province to help explain possible endocrine changes in response to severe infection during pregnancy.

2. MATERIALS AND METHODS

Study design, setting, and period

The study is a case-control case study based in a hospital, Al-Nasiriyah Teaching Hospital, Dhi Qar Province, Iraq, in the year 2020.

Participants: 200 pregnant women (2540 years) in the second trimester (1620 weeks of gestation) were recruited and randomly assigned to:

Severe COVID-19 group (cases): n = 100

Healthy control group (controls): n = 100.

The participants were similar in terms of the range of maternal weight (7375 kg), singleton pregnancies, and no chronic disease or thyroid disorders history.

Case and control: Cases were pregnant women with laboratory-confirmed SARS-CoV-2 infection (RT-PCR positive) during the index pregnancy and severe disease necessitating supplemental oxygen (all cases needed oxygen therapy). After the samples were collected, hormonal measurements were done at approximately 16 weeks of gestation, at a time when participants were of 16 20 weeks of gestation. The treatment protocol followed in all of the cases was identical as per the hospital guidelines.

The controls were healthy pregnant women who were getting routine antenatal care in the same hospital and time. Cases and control group were treated equally by means of laboratory tests.

Laboratory tests and blood samples: The blood samples were taken through venous blood samples, and the serum was centrifuged based on the standard procedures. ELISA determined serum levels of progesterone (ng/mL), estradiol (E2; pg/mL), 2-hCG (mIU/mL), cortisol (250 ug/dL), and TSH (mIU/mL) by the manufacturer guidelines along with in house quality-control.

Statistical analysis: Normality tests were done on continuous variables and shown as mean SD (or median IQR where non-normal). The independent samples t-test was used or the Mann Whitney U-test in case of between-group comparisons. Stratification of analyses was further

done using gestational age (1618 vs 1920 weeks old). A p less than 0.05 on either side was taken to be statistically significant.

Ethics: The institutional committee that was used made an ethical approval and the informed consent was written and signed by all participants.

3. RESULTS

The analysis was based on 200 pregnant women in the second trimester (1620 gestation weeks) who includes 100 pregnant women with severe SARS-CoV-2 infection and 100 healthy pregnant controls. The two groups were similar in terms of maternal age and weight and had a history of neither chronic nor thyroid disorders.

Table 1 has demonstrated that there were notable differences in a number of the hormonal parameters between the two groups. Pregnant women who had a severe case of COVID-19 had pronouncedly low levels of progesterone (36.5 ± 9.8 ng/mL) as compared to the healthy controls (44.7 ± 10.7 ng/mL) with a mean difference of -8.2 ng/mL ($p < 0.001$). On the same note, estradiol E2 (E2) was largely lower in the severe Covid-19 (4,876 ± 1,321 pg/mL) compared to the controls (5,726 ± 1,440 pg/mL; $p < 0.001$).

Furthermore, the concentration of β-human chorionic gonadotropin (2-hCG) was also significantly lower in the groups of the infected individuals (18,052 6,451 mIU/mL) than in healthy pregnant women (22,351 6,900 mIU/mL; $p < 0.001$). The decreases of placental-related hormones are represented in Figures 1 to 3.

On the other hand, the mean cortisol level of serum of pregnant women with severe COVID-19 (32.3 ± 8.1 µg/dL) was significantly higher than the control (24.8 ± 6.5 µg/dL), and the difference between the means is significant (7.6 µg/dl) as shown in Figure 4.

In terms of thyroid activity, TSH levels differed in the severe COVID-19 group (0.93 ± 0.47 mIU/L) and the control group (1.29 ± 0.62 mIU/L) with a slight significant difference of $p = 0.002$ as depicted in Figure 5.

Altogether, these results suggest that serious SARS-CoV-2 infection in the second trimester is accompanied by a substantial change in maternal hormonal profiles, with a lower level of placental hormones (progesterone, estradiol, and 5 -hCG) and an increase in the level of stress-related cortisol, but a slight drop in TSH. These differences are graphically demonstrated in Figures 1-5 which clearly prove the statistically significant hormonal changes as summarized in Table 1.

Table 1: Hormonal comparison in pregnancies with severe COVID-19 infection in comparison to healthy controls during the second trimester

Hormone	Severe COVID-19 group (n=100) Mean ± SD	Healthy control group (n=100) Mean ± SD	Mean difference	P value
Progesterone(ng/mL)	36.5 ± 9.8	44.7 ± 10.7	−8.2	<0.001
Estradiol(E2) (pg/mL)	4,876 ± 1,321	5,726 ± 1,440	−850	<0.001
β-hCG (mIU/mL)	18,052 ± 6,451	22,351 ± 6,900	−4,300	<0.001
Cortisol (µg/dL)	32.3 ± 8.1	24.8 ± 6.5	+7.6	<0.001
TSH (mIU/L)	0.93 ± 0.47	1.29± 0.62	−0.36	0.002

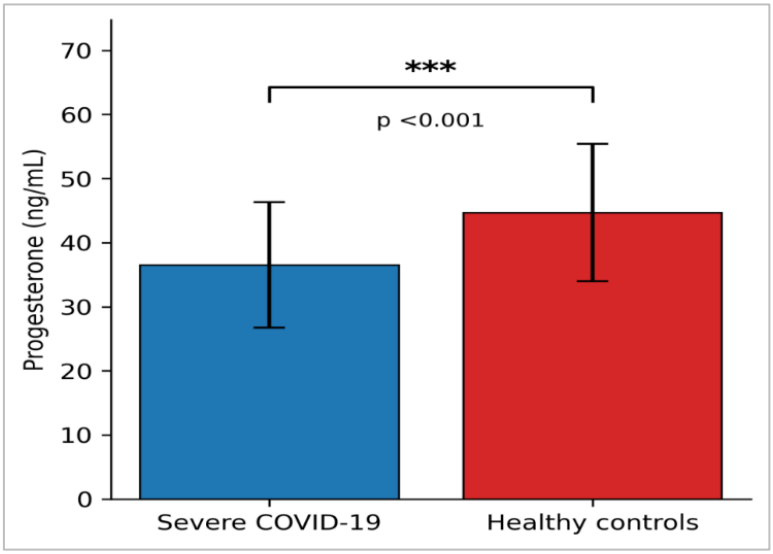

Figure 1: Serum progesterone levels in severe COVID-19 and healthy pregnant women

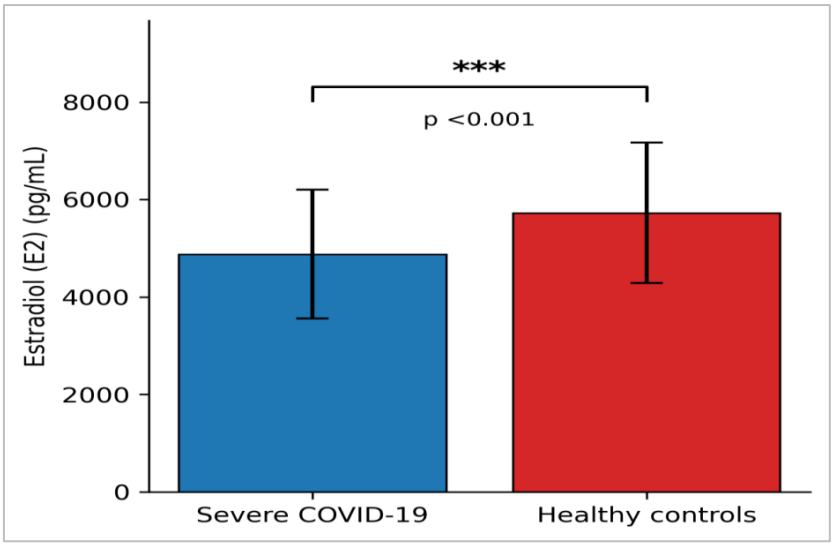

Figure 2: Serum estradiol (E2) levels in severe COVID-19 and healthy pregnant women

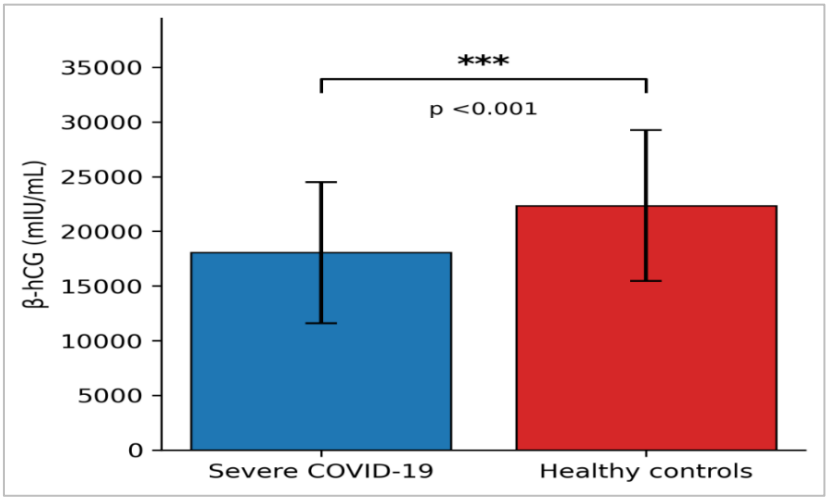

Figure 3: Serum β-hCG levels in severe COVID-19 and healthy pregnant women

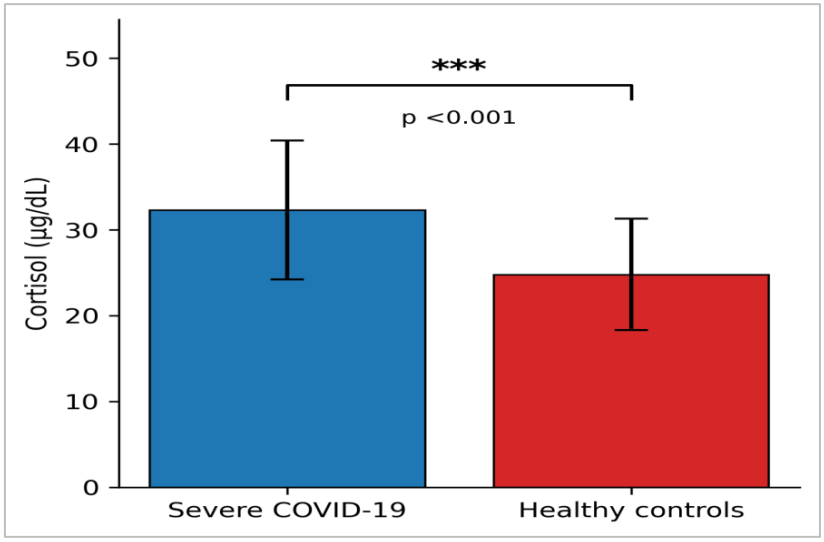

Figure 4: Serum cortisol levels in severe COVID-19 and healthy pregnant women

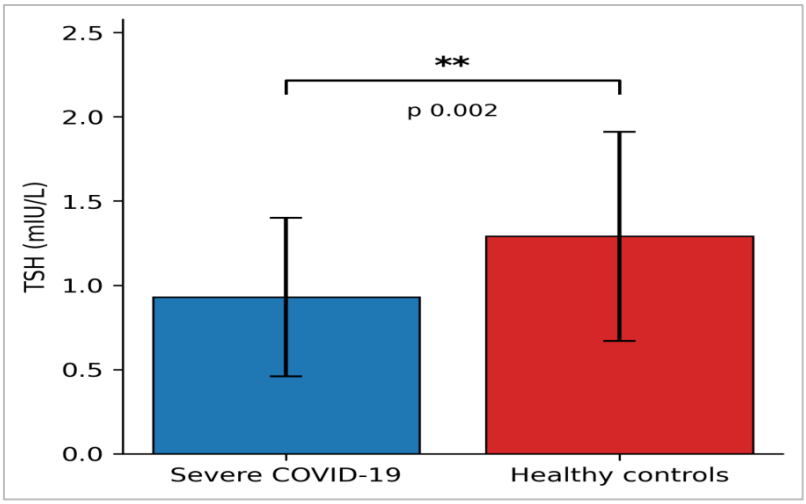

Figure 5: Serum TSH levels in severe COVID-19 and healthy pregnant women

4. DISCUSSION

The current case-control research problem was the effect of severe COVID-19 due to Severe-acute respiratory syndrome coronavirus 2 on maternal hormonal profiles during the second trimester of pregnancy. The results indicated evident endocrine changes in the pregnant women who had recovered following severe infection, with a total of reduced concentrations of progesterone, estradiol, and 8 -human chorionic gonadotropin (8 -hCG) and an increase of cortisol concentrations and a moderate decrease in thyroid-stimulating hormone (TSH) relative to healthy pregnant controls. These findings suggest that serious cases of SARS-CoV-2 infection can affect placental endocrine function and maternal systemic hormone control, which is a reflection of the interplay between the effects of viral infection, inflammatory responses, and endocrine physiology. One of the most important findings in this research was that the level of progesterone decreased significantly in pregnant women who had severe COVID-19. Of utmost importance is the role that progesterone has in sustaining pregnancy by ensuring uterine quiescence, immune tolerance to the fetus and also developing the placenta. The low levels of progesterone could thus indicate placental stress or a defect in the trophoblastic activity. Past research has also shown that inflammatory cytokines and viral infections have the ability to interfere with steroidogenic pathways in placental tissues, which may limit the production of progesterone [15]. Besides, progesterone has characterized anti-inflammatory and immunomodulatory effects, which could potentially affect how hosts respond to viral infections, including SARS-CoV-2 [16]. Therefore, the low progesterone level could indicate the dysfunction of the placenta and disregulation of immunity in the case of serious infection.

On the same note, the concentrations of estradiol (E2) were much lower in the pregnant women who were infected. Estradiol is an imperative controller of uteroplacental circulation, angiogenesis of the placenta, and fetal growth. The low level of estradiol can diagnose as such low levels may refer to dysfunctional placental endocrine functions. The problem of placental abnormalities during COVID-19-complicated pregnancies has been stated in a few studies, where vascular lesions, inflammatory alterations, and maternal vascular malperfusion [17] are identified. Such pathological changes can disrupt hormone production in placenta tissues. Moreover, the molecular research on the placentas of infected women revealed that the genes of the estrogen signaling pathways are being altered, implying that the endocrine control, at the placental level, might be broken by the SARS-CoV-2 infection [18]. These results are aligned with the low estradiol levels that were found in the current study.

The fact that the levels of β-hCG decreased is another indication of a potential placental dysfunction as 2 -hCG is created by syncytiotrophoblast cells and is one of the major forms of placental activity and pregnancy containment. A decrease in β-hCG levels can be a sign of the dysfunction of trophoblasts or the decreased secretion of placental hormones after the acute viral infection. Past studies have postulated that COVID-19 has the potential to impact placenta physiology and biomarkers of placenta functioning [19]. Inflammatory lesions and thrombotic alterations have also been described in placentas of infected mothers by histopathological studies that may impair trophoblastic integrity and endocrine functions [20]. The resulting reduction in progesterone, estradiol, and 2-hCG indicated by the current study consequently leads to the conclusion that there is a regular pattern of placental endocrine dysregulation in the case of severe SARS-CoV-2 infection. Unlike placental hormones, the levels of cortisol were very high in the case of severe COVID-19 pregnant women. The main glucocorticoid hormone that is released by the stimulation of hypothalamic-pituitary-adrenal (HPA) axis is cortisol, a hormone that reacts to physiological stress and inflammation on a systemic level. Viral infections that cause severe responses have been known to trigger intense neuroendocrine mechanisms, which elevate cortisol

release. High levels of cortisol recorded in this paper are probably the physiological stress reaction linked to serious infection. Past clinical studies have also reported the elevation of cortisol in hospitalized COVID-19 patients especially in patients with severe disease that needed respiratory assistance [21]. High levels of cortisol can also affect immune response and metabolism in case of infection and also underscores the interrelation between metabolism and immune response. The other interesting observation was a slight decrease in the level of TSH in pregnant women who were infected. During systemic infections and inflammatory states, thyroid dysfunction is frequent in case of systemic factors. Lower levels of the TSH can happen as a non-thyroidal illness syndrome which is a temporary change in the regulation of the thyroid hormones in case of severe illnesses. Various reports have reported some disorders in thyroid in patients with COVID-19, such as lowering the TSH level and a change in the thyroid hormone metabolism [22]. Moreover, SARS-CoV-2 infection has been linked with more widespread endocrine dysfunctions that included thyroid, adrenal, and pituitary gland dysfunctions [23. Thus, the decrease in TSH that was found in this study could be attributed to infection-related endocrine disregulation and the physiological changes of pregnancy .On the whole, the results of the hormonal profile indicate two key endocrine effects of acute SARS-CoV-2 infection in pregnant women. First, the reduction in placental hormones suggests the possible endocrine dysfunction of the placenta caused by inflammatory damage, vascular defects, or trophoblastic dysfunction. Second, the higher level of cortisol and the lower level of TSH indicate that the systemic neuroendocrine responses to acute infection and inflammatory stress. These conclusions are consistent with the recent evidence, which stated that COVID-19 can impact not only placental physiology but also maternal endocrine pathways, which can have an impact on the health of pregnancy [24].

Such hormonal changes can be of clinical significance. The hormones such as progesterone, estradiol and 2hCG are vital in the retention of pregnancy in the body and in promoting the growth of the fetus. The disturbances of these pathways could be the cause of placenta disturbance and pregnancy problems. Recent systematic reviews have found evidence of higher risks of adverse outcomes, including preterm birth and an admission to neonatal intensive care among women with severe COVID-19 [25]. Though the effects of pregnancy were not directly evaluated in the current study, the identified hormonal disruptions serve as additional confirmation of the idea that dire cases of SARS-CoV-2 infection can affect maternal endocrine balance during pregnancy.

5. CONCLUSIONS

The results of the research suggest that serious COVID-19 in the second trimester of pregnancy is linked with the major changes in maternal hormonal profiles. The placental hormones, progesterone, estradiol, and b-human chorionic gonadotropin, and plasma cortisol concentrations and slight decreases in thyroid stimulating hormone (TSH) were lower in the women who were pregnant and recovered after severe infection, than in healthy pregnant controls .These endocrine alterations indicate that the extreme infection of SARS-CoV-2 can influence placental endocrine and global maternal hormonal equilibrium. These decreased placental hormones can be signs of subclinical placental dysfunction or trophoblastic stress associated with inflammatory and vascular changes, whereas elevated cortisol concentrations probably demonstrate the activation of the hypothalamic-pituitary-adrenal stress response .In general, these results indicate the need to observe the endocrine and placental activity in pregnant women who experienced severe COVID-19 to follow up. More extensive longitudinal studies combining hormonal, inflammatory and placental measurements are required to explain the clinical importance of these endocrine changes.

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