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Original Article

Impact of Antenatal Anxiety and Depression

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Abstract

Introduction: Pregnancy is generally accepted to be a time of happiness and emotional well-being for a woman. However, for many women, pregnancy increases their vulnerability to psychiatric illnesses such as depression and anxiety disorders (ANYAXs), and these are usually overlooked. This study was conducted to determine the effect of maternal depression and ANYAXs on the course of pregnancy and on fetal health.

Materials and Methods: A total of 310 women attending the antenatal clinic were interviewed as a part of this cohort study, of which 284 completed the study. The participants were assessed twice in the second trimester and once in the third trimester. Depressive and ANYAXs were diagnosed using ICD-10 criteria. Severity was assessed using relevant scales.

Result: The prevalence of depressive disorders was 41 (63%) and ANYAXs 24 ((36.9%). There was a significant association between the presence of psychiatric disorders (PSYDS) and obstetric complications ($P < 0.05$) and between depression in pregnancy and the development of hypertensive disorder of pregnancy ($P < 0.05$). ANYAX in pregnancy was significantly associated with fetal growth disorder ($P < 0.05$). The presence of a PSYDS was strongly associated with fetal distress measured by the Apgar score at birth ($P < 0.05$) and with preterm birth ($P < 0.05$).

Conclusion: An interdisciplinary approach is needed for improving interventions to prevent maternal and fetal complications.

Key words: Antenatal, Anxiety, Depression, Fetal growth, Pregnancy

INTRODUCTION

Pregnancy and its associated complications have been an issue of public health concern throughout the world. Pregnancy and the transition to parenthood involve major psychological and social changes in the mother, which have been linked to symptoms of anxiety and depression.[1] Approximately, 21% of women experience a mood disorder and 30% anxiety disorder (ANYAX) at some points in their lives.[2] Although historically it was believed that pregnant women are at lower risk of anxiety and mood disorders,[3] recent studies do not support this belief. Rather between 10% and 27% of women experience depressive symptoms during pregnancy including 2-11% who experience major depressive disorder.

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Studies have indicated that depression and anxiety during pregnancy affect the neonatal outcome. In particular, attention has focused on the increased risks of spontaneous preterm delivery, low birth weight, operative delivery (cesarean section and instrumental vaginal delivery), and admission to a neonatal intensive care unit among offspring of women with antenatal depression.[4-6] Not only does depression tend to shorten pregnancy but also major life events, if they are perceived as stressful, tend to shorten it as well.[7,8] Antenatal stress has been suggested to cause preterm delivery through activation of the placental-maternal pituitary-adrenal axis.[9] This hypothesis is further supported by a relation between preterm birth and elevated levels of corticotropin-releasing hormone (CRH) in maternal plasma and in placenta.[9-11]

Studies have shown a relationship between maternal anxiety in pregnancy and increased uterine artery resistance index.[12] It suggests a mechanism by which the psychological state of the mother may affect fetal development, and may explain epidemiological associations between maternal anxiety and low birth weight. High levels of anxiety during pregnancy have been associated with increased risk for preeclampsia.[12]

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The correlation between plasma levels of cortisol in the mother and in the fetus may have implications for the developing fetal brain.[13]

Depression during pregnancy has extensive negative effects on mothers and babies including increased risks for preterm birth and low birth weight.[14,15] Posttraumatic stress disorder during pregnancy was shown to increase the risk for preterm birth.[16]

This study was conducted to determine the effect of maternal depression and ANYAXs on the course of pregnancy and on fetal health.

MATERIALS AND METHODS

This cohort study was carried out in the outpatient Department of Obstetrics and Gynecology at Goa Medical College, Bambolim - Goa, over a period of 1 year. Ethical approval was obtained from the Local Ethics Committee before commencing the study.

A total of 310 antenatal women were interviewed during this period. All the women were in the second trimester at the time of interview. This was essential because most women presented for the first antenatal visit during the second trimester or toward the end of the first trimester. Furthermore, the time period required to obtain the reports of routine investigations before induction in the study resulted in all women being inducted in the second trimester.

Informed consent was obtained from all patients before inclusion. Only those women who intended to carry out future antenatal visits and delivery at Goa Medical College were included in the study.

Antenatal visits in Goa Medical College (GMC) are carried out at intervals of 4 weeks up to 28 weeks, at intervals of 2 weeks up to 36 weeks, and weekly thereafter till term. Participants were assessed on 3 occasions; twice in the second trimester and once in the third trimester. Hence, antenatal assessments in the study were separated by a gap of at least 4 weeks.

Potential confounding factors or effect modifiers identified were age, parity, current substance use, caloric intake, obstetrical complications (OBSDYSs) in previous pregnancy which are likely to recur in subsequent pregnancies, any abnormality detected on routine investigation or booking ultrasound scan, medical and surgical illness during pregnancy, gynecological disorders in pregnancy, and if the patient was currently receiving medication for any psychiatric disorder (PSYDS).

After limiting the confounding factors and effect modifiers, participants were inducted in the study.

Inclusion criteria were:
1. Maternal age between 20 and 35 years
2. No history of substance use
3. No history of recurrent obstetric complications
4. Adequate caloric intake
5. Not receiving treatment for a major PSYDS currently
6. No abnormality detected on initial routine antenatal screening or on initial ultrasound.

Depressive and ANYAXs were diagnosed using the ICD-10[17] criteria.

Patients thus diagnosed were then rated to analyze the longitudinal course of the illness using relevant scales.

Data (psychiatric and medical) were recorded, coded, analyzed, and interpreted using the SPSS software.

RESULTS

A total of 310 antenatal women participated in the study. Of these, 12 women did not follow-up for further antenatal visits at GMC; 5 patients underwent a therapeutic abortion for severe complications in the second trimester, a further 5 patients developed PSYDSs following an adverse obstetric event (depression - 3 and panic disorder - 2) and 4 patients were started on medications during pregnancy for psychiatric symptoms (mania = 1, depression = 2, and generalized ANYAX = 1).

The number of participants who completed the study was 284 (attrition rate = 8.39%).

Findings in the study are described below.

Of the 284 patients who participated in the study, 65 (22.89%) were diagnosed as having a PSYDS.

The mean age of the patients who had a PSYDS was 23.88 (standard deviation 2.713) and the mean age of those who did not was 23.20 (standard deviation 2.415) as shown in Table 1.

Of the total of 65 patients with PSYDS, 41 (63%) patients were diagnosed to have depressive disorders and 24 (36.9%) ANYAXs. The overall distribution of the various depressive and ANYAXs is summarized in Table 2.

Of the total of 65 patients who were diagnosed as having a PSYDS, 24,i.e., (36.9%) developed OBSDYSs.

Of the remaining 219 patients who did not have any psychiatric diagnosis, 44 (20.1%) developed OBSDYSs as is shown in Table 3.

A significant association was found between the presence of PSYDSs and OBSDYSs (Chi-square = 8.381, $P = 0.004$).

Out of 41 patients with depression, 11 (26.8%) patients were diagnosed to have hypertension as compared to 31 (12.8%) patients out of 243 without depression.

Depressive disorders were significantly associated with the development of hypertensive disorder in pregnancy (Chi-square = 5.513, $P = 0.019$) as shown in Table 4.

Of the 24 (36.9%) patients diagnosed to have ANYAXs, 10 (41.7%) had fetal growth disorder (FGD) as compared to 43 (16.5%) patients with no ANYAX as shown in Table 5.

A significant association was found between the presence of ANYAXs in pregnancy and FGDs (Chi-square = 13.451, $P < 0.05$).

Of the 65 patients with anxiety and depressive disorders, 27 (41.5%) had abnormal Apgar score as compared to 51 (23.3%) with no PSYDS.

The presence of a PSYDS was strongly associated with fetal distress as measured by the Apgar score at birth as shown in Table 6 (Chi-square = 8.381, $P = 0.04$).

16 (24.6%) patients of those with PSYDS had preterm delivery as compared to 26 (11.9%) without PSYDS.

PSYDS in pregnancy was significantly associated with preterm birth as shown in Table 7 (Chi-square 6.459, $P < 0.01$).

DISCUSSION

The study was undertaken to determine the effect of anxiety and depression in the antenatal period and on the course of pregnancy and fetal health.

65 patients met the diagnosis of anxiety and depression. Of these, 63% were diagnosed to have depressive disorder and 36% ANYAXs. This is in sharp contrast with the study by Andersson *et al.*,[18] wherein the point prevalence of mood and ANYAXs during the second trimester of pregnancy in a population-based sample of pregnant women were analyzed using the Primary Care Evaluation of Mental Disorders questionnaire was found to be 10.2% for depression and 6.6% for ANYAXs. Studies anxiety

Table 1: Mean age and standard deviation of participants

Psychiatric dysfunction	Number	Mean age	Standard deviation
Present	65	23.88	2.713
Absent	219	23.20	2.415

Table 2: Distribution of patients with depressive and ANYAXs

PSYDS	Number of patients
Depressive disorder	41 (63%)
Recurrent depressive disorder	18
Depressive episode	8
Depressive episode and generalized ANYAX	9
Depressive episode and panic disorder	4
Depressive episode and obsessive compulsive disorder	1
ANYAXs	24 (36.9%)
Generalized ANYAX	12
Obsessive-compulsive disorder	4
Panic disorder	6
Agoraphobia	1
Social phobia	1

PSYDS: Psychiatric disorder, ANYAX: Anxiety disorder

Table 3: Association between PSYDSs and obstetric dysfunction

PSYDS	OBSDSY		Total
	Yes	No	
Present			
Number	24	41	65
Percentage	36.9	63.1	100.0
Absent			
Number	44	175	219
Percentage	20.1	79.9	100.0
Total			
Number	68	216	284
Percentage	23.9	76.1	100.0

PSYDS: Psychiatric disorder, OBSDYS: Obstetrical complication, Chi-square=8.381, P=0.004

Table 4: Association between depression and HTDIS

DEP	HTDIS		Total
	Absent	Present	
Absent			
Number (%)	212	31	243
Percentage	87.2	12.8	100.0
Present			
Number (%)	30	11	41
Percentage	73.2	26.8	100.0
Total			
Number (%)	242	42	284
Percentage	85.2	14.8	100.0

DEP: Depressive disorder, HTDIS: Hypertensive disorder in pregnancy, Chi-square=5.513, P=0.019

Table 5: Association between ANYAX and FGD

ANYAX	FGD		Total
	No	Yes	
0			
Number	217	43	260
Percentage	83.5	16.5	100.0
1			
Number	14	10	24
Percentage	58.3	41.7	100.0
Total			
Number	231	53	284
Percentage	81.3	18.7	100.0

ANYAX: Anxiety disorder, FGD: Fetal growth disorder, 0 - Group without psychiatric disorder, 1 - group with anxiety disorder, Chi-square=13.451, *P*<0.05

Table 6: Association between PSYDSs and depressed Apgar score

PSYDS	Apgar		Total
	Normal	Abnormal	
Present			
Number	38	27	65
Percentage	58.5	41.5	100.0
Absent			
Number	168	51	219
Percentage	76.7	23.3	100.0
Total			
Number	206	78	284
Percentage	72.5	27.5	100.0

PSYDS: Psychiatric disorder (Chi-square=8.381, *P*=0.04)

Table 7: Association between PSYDSs and preterm birth

PSYDS	Preterm birth		Total
	Yes	No	
Present			
Number	16	49	65
Percentage	24.6	75.4	100.0
Absent			
Number	26	193	219
Percentage	11.9	88.1	100.0
Total			
Number	42	242	284
Percentage	14.79	85.21	100.0

PSYDS: Psychiatric disorder, (Chi-square 6.459, *P*<0.01)

in pregnancy anxiety from different parts of the world reported a prevalence rate of 14-54%.[9,19-21]

However, the findings of our study are comparable with those reported in developing countries. In Brazil, the prevalence rate of anxious symptoms among pregnant women was estimated to be 60%, and the rate for depressive symptoms was about 20%.[22] Furthermore, in Bangladesh, the prevalence rate of depressive symptoms among pregnant women was estimated to be 33% and 42.7% in Pakistan.[23] The prevalence of anxiety (41%) and depression (57%) was found in Nicaraguan pregnant women.[24]

Depressive and ANYAXs during pregnancy were strongly associated with the development of OBSDYSs in the present study. Andersson *et al.*[18] analyzed the obstetric outcome and health-care consumption during pregnancy, delivery, and the early postpartum period in an unselected population-based sample of 1495 pregnant women diagnosed with antenatal depressive and/or ANYAXs, compared with healthy participants. Significant associations were found between depression and/or anxiety and increased nausea and vomiting, prolonged sick leave during pregnancy, and increased number of visits to the obstetrician, specifically, visits related to fear of childbirth and those related to contractions. Planned cesarean delivery and epidural analgesia during labor were also significantly more common in women with antenatal depression and/or anxiety.

Bonari *et al.*[14] in a review of perinatal risks of untreated depression concluded that depression in pregnancy was significantly associated with preterm delivery and growth retardation, preeclampsia, spontaneous abortion, and impaired perinatal development.

Pathophysiological mechanisms to explain the factors responsible for adverse obstetrical outcomes in physically healthy women suffering from PSYDSs during pregnancy have focused on hormonal mechanisms to explain the findings.

Nadelson and Dickstein[25] Kammerer *et al.*,[15] Mastorakos and Ilias[26] Ochedalski and Lachowicz[27] and Halbreich[28] in five independent papers, have proposed several mechanisms which may interact to produce an adverse obstetric outcome. The neuroendocrine stress response, immunosuppression, and the role of genetics have been implicated as some of the mechanisms leading to OBSDYSs.

Depression in pregnancy was found to be significantly associated with the development of hypertensive disorders in pregnancy. This finding is in accord with that of Kurki *et al.*[29] who found that depression and anxiety in early pregnancy were significantly associated with preeclampsia, and Evans *et al.*,[1] who reported a significant association between depression in pregnancy and development of preeclampsia. However, Larsson *et al.*[30] did not find any association between depression and preeclampsia.

Depressive disorders may be harmful through an altered secretion of vasoactive hormones and other neuroendocrine transmitters. This may in turn cause vasoconstriction and

increased uterine artery resistance and, therefore, elevate blood pressure. Depressive disorders during pregnancy increase stress resulting in increased levels of pressor agents,i.e., epinephrine, norepinephrine, and angiotensin 2, Halbreich.[28] An increased level of prostaglandins is seen during stress and may contribute to vasoconstrictor effects.

This immune response in early pregnancy is primarily a Th-1 response with the secretion of interferon-gamma, interleukin 2 (IL-2), and tumor necrosis factor-beta which promote cellular immunity. If the Th-1 response persisted beyond early implantation, the pregnancy may not survive; thus, there is a switch in the immune system to a Th-2 with a different set of cytokines, IL-4, IL-10, and IL-13 that promote humoral immunity and a decreased risk of rejection. However, depression and anxiety can cause an increase in the Th-2 response resulting in an inflammatory cascade and endothelial injury.[31]

Further depressive disorder can cause an abnormal immunological reaction thus making the individual susceptible to infection.

Interestingly, the above mechanisms have also been postulated in the pathophysiology of preeclampsia.[31] Hence, depression in pregnancy may increase susceptibility to preeclampsia by increasing stress.

ANYAXs in pregnancy were significantly associated with FGDs, i.e., small for gestational age babies. Dole *et al.*[8] in a prospective cohort study of 1962 pregnant women in central North Carolina between 1996 and 2000, found a significant association between ANYAXs in pregnancy and preterm birth. Similarly, Kent *et al.*[8] reported a significant association between ANYAXs in pregnancy and preterm birth. This could be explained on the basis of several mechanisms.

Placental CRH plays an important role in the communication between the placenta and the maternal and fetal adrenal gland for the production of precursors of estrogen production which are important for uterine growth and perfusion. First, elevations in CRH appear in the fetal circulation, as shown by Goland *et al.*[33] suggesting that this peptide is available to activate the fetal pituitary-adrenal axis, which is considered to be mature during the second trimester. The relationship between maternal endocrine events, placental CRH, and the fetal pituitary-adrenal axis could be an attempt by the maternal-fetal-placental unit to bring about early fetal maturation to increase the chances for survival if delivered early, Hobel *et al.*[34]

Anxiety in pregnancy is associated with increased uterine artery resistance as demonstrated by Texeira *et al.*[21,32] This may reduce uteroplacental perfusion and exchange and contribute to intrauterine growth retardation.

Third, elevated levels of pituitary hormones such as oxytocin and prostaglandins may result in premature uterine contractions and contribute to the initiation of premature labor.

Furthermore, elevated stress levels resulting in immunosuppression may predispose to the development of infections, thus hampering fetal growth and causing prematurity.

Finally, patients with depressive and ANYAXs might be more likely to engage in poor health behaviors such as inadequate diet, or smoking, or might be less likely to avail adequate prenatal services. However, this factor was controlled for in the present study; it appears the findings in this study are more likely due to neuroendocrine parameters.

Although most studies have found a significant association between ANYAXs in pregnancy and FGD, a large meta-analysis by Littleton *et al.*[35] did not find any relationship between ANYAX and OBSDYSs.

The risk of preterm birth increased significantly in women who had a PSYDS compared to cohorts without a psychiatric diagnosis. Steer *et al.*[36] found elevated risks for preterm delivery (<37 weeks), low birth weight (<2500 g), and babies small for their gestational age (<10th percentile) among women who had scores of 21 or more on the Beck Depression Inventory and who were not receiving active treatment.

The neuroendocrine disturbances described above possibly increase uterine irritability resulting in preterm labor and subsequent delivery.

Similarly, it was observed that women with PSYDSs were more likely to deliver babies with low Apgar score at birth. Apgar score is a measure of fetal distress due to a deleterious intrauterine environment or birth trauma.

Chung *et al.*[5] using serial ultrasonography demonstrated that the fetus of a mother suffering from depression spends more time in sleep and exhibits less body movement than the fetus of a mother without depression. A similar study by Allister *et al.*[37] using ultrasonography suggested that maternal depression may affect fetal heart rate response to vibroacoustic stimulation. This test produces cardio acceleration typical of a healthy fetus and is commonly used to assess fetal well-being. In women with untreated depression, there was a delayed fetal response to a

vibroacoustic stimulus applied to the maternal abdomen. These findings are suggestive of fetal distress. Zax *et al.*[38] and Patel *et al.*[39] found significant associations between maternal depression in pregnancy and lower Apgar scores in the offspring. The causative factors may be placental insufficiency, prematurity, or superimposed infections.

CONCLUSION

The findings of this study suggest that contrary to popular belief, a substantial number of women suffer from PSYDSs in the antenatal period. Development of PSYDSs may precipitate OBSDYSs and thus jeopardize the course of pregnancy with adverse consequences both for the mother and developing fetus.

However, PSYDSs during pregnancy are under diagnosed, ignored, or undertreated. Routine assessment of the mental health of pregnant women at a primary health-care level has the potential to identify early those women experiencing distress or with significant risk factors for perinatal disorders.

Clinicians, families, and women themselves need to be educated about the perils of untreated PSYDSs in pregnancy so that they can make truly informed treatment decisions.

Currently, the multiple clinical and research disciplines that are concerned with the various aspects of pregnancy, delivery, and postpartum period are not conceptually and practically integrated. Specifically, obstetricians are more concerned with delivery complications, whereas mental health professionals are concerned with the treatment of PSYDSs. An interdisciplinary approach is needed for better understanding of psychological processes and the development of measurements and interventions to prevent long-term impact on the offspring.

In the light of these findings, further research is necessary to determine the causal mechanisms by which PSYDSs result in OBSDYSs, as well as long-term effects on the mental health of children. In addition, studies are necessary to determine the optimum treatment of these disorders during pregnancy as well as effectiveness of psychosocial interventions.

LIMITATIONS OF THE STUDY

1. The study was carried out at a tertiary care hospital in Goa; hence, the findings cannot be extrapolated to the general population
2. As the primary focus of the study was to assess whether PSYDSs caused OBSDYSs, life events, and support systems that might influence the development and course of PSYDSs during pregnancy were not considered.

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