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COMPLICATIONS AND OUTCOMES IN 3 OBESE PREGNANT FEMALES- A CASE SERIES

Obstetrics & Gynaecology

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ABSTRACT

Obesity has been implicated in causing numerous antenatal, intrapartum and postpartum complications in the mother as well as the baby.This is a case series of 3 obese pregnant females, they had complications like GDM, gestational hypertension, hypothyroidism, failed labor induction, difficulty in delivery, delayed wound healing, etc. In this case series we have elaborated the preventive measures, complications and their management as they occured.

KEYWORDS

Obesity, Gestational hypertension, Gestational diabetes mellitus

INTRODUCTION

The prevalence of obesity in the general population among women of childbearing age has increased dramatically during recent years. Extensive weight gain is a major health problem today in many affluent societies. The adverse health aspect of obesity is staggering and includes increased risk of type 2 diabetes, hypertension, heart diseases and osteoarthritis. Importantly obese women and their fetuses are predisposed to numerous pregnancy related complications like GDM, gestational hypertension, increased number of labor inductions, failed induction, cesarean deliveries etc.

Case Summary-1

A 33 year old G3P1L1A1 morbidly obese women booked at Preethi hospital. Her height is 165 cm, weight 122 kg, BMI -44.8 kg/m2(morbidly obese).

Past obstetric history: (1) Her last child birth was 5 years back, during which she had gestational hypertension from 22 weeks of gestation, managed with antihypertensives. Labor was induced at 39 weeks of gestation and she had delivered a female baby of 2.9 kg vaginally in a private hospital. Her blood pressure was not reduced after pregnancy and diagnosed as chronic hypertension. She was on antihypertensives since then. (2) In 2020 she had a missed miscarriage at 12 weeks of gestation, for which she underwent dilatation and evacuation in a private hospital.

Present Pregnancy

First trimester: spontaneous conception. Booked at Preethi hospital. First trimester: GCT showed impaired glucose tolerance (2 hour-125 mg/dL), started on medical nutritional therapy. BP was elevated (>140/90 mmHg). Pregnancy safe antihypertensives, Tab.Labetalol 100 mg twice daily was started and her ARBs were stopped .Had history of hypothyroidism as TSH was found to be 6.6 mIU/L and hence Started on Tab. Thyroxine 25 mcg once daily. NT screening with double marker test showed low risk.

During 2nd trimester: BP was high (>140/90 mmHg) without proteinuria or any signs of end organ damage, Tab. Labetalol 100 mg changed to thrice daily dose. Anomalies ruled out at 20th week of gestation. At 24 weeks diagnosed as GDM as her GTT found to be abnormal (FBS-119 mg/dL, 1 hour-161 mg/dL, 2 hour-117 mg/dL, 3 hour- 104 mg) and hence was on insulin along with MNT . During 3rd trimester: growth scan was corresponding to gestational age, BP was elevated (>150/90 mmHg), urine albumin was 1+, diagnosed as pre-eclampsia superimposed on chronic hypertension. BP was controlled after adding tab. Nifedipine 20 mg –TDS. Physician opinion obtained in all trimesters. Cardiac diseases ruled out with ECG and ECHO. Total weight gain during entire pregnancy was 5.9 kg. At term labor induced with dinoprostone gel, thrice 6 hours apart, continuous CTG monitoring was done though it was difficult due to obese abdomen. She had a long latent period. Labor was augmented with oxytocin, she had a prolonged augmentation to delivery interval. Intrapartum period: BP was monitored and dual antihypertensives were continued. Due to poor maternal efforts and lax abdomen vacuum delivery was done. She delivered an alive female baby of 2.65 kg. Injection carbetocin was given for prevention of PPH and PPH was prevented. BP was controlled with the same dual antihypertensives (labetalol and nifedipine) in the postpartum period and then changed to her usual ARB. GTT and thyroid function test done after 6 weeks of delivery was normal. She was advised to have regular follow-up with the physician for hypertension.

Case Summary -2

A 31 years old obese primigravida, conceived 3 years after marriage, presented at 7 weeks of gestation, her height-154 cm, weight 96 kg, BMI-40.47 kg/m2(morbidly obese), not a known hypertensive/ diabetic/ thyroid disorder. During 1st trimester, Tab. Ecosprin 150 mg was started as a prevention of pre- eclampsia and continued throughout pregnancy till 34 weeks, routine investigations including GCT was normal. NT screening with double marker test was negative. During 2nd trimester: weight gain was 0.7-0.8 kg/month, Anomaly scan at 20 weeks showed growth corresponding and no gross anomalies. GTT done at 24 weeks was abnormal (FBS-113 mg/dL, 1 hour-190 mg/dL, 2 hour-162 mg/dL). Started on MNT and sugars monitored, later on insulin was added to control sugars. BP was elevated first at 25+5/7 weeks without proteinuria or signs of end organ damage, diagnosed as gestational hypertension and started on Tab. Labetalol 100 mg BD.

During 3rd trimester: weight gain was 1 kg/month. Interval growth scan corresponding to gestational age, sugars were controlled and continued on MNT and insulin. BP was maintained <140/90 mmHg with Labetalol. Developed pedal oedema and abdominal wall edema from 36 weeks of gestation, proteinuria or signs of end organ damage were absent. Total weight gain during entire pregnancy was 7.8 kg. Labor induced at term with dinoprostone gel. 2 doses of dinoprostone gel (6 hours apart) was required for induction. Continuous fetal monitoring was difficult due to obese abdomen. Labor augmented with oxytocin, labour progressed well, vacuum was used to deliver the baby in view of narrow outlet and inadequate uterine contractions. She delivered an alive female baby of 2.515 kg. Had PPH and managed with oxytocin and carboprost.

Thromboprophylaxis given .BP continued to be elevated up to 4 weeks post-partum, during which she was on labetalol and later on labetalol was stopped and BP was normal. Advised to follow up her BP regularly. GTT at 6 weeks postpartum was normal. Baby developed jaundice on day 3 of life (total bilirubin 26 mg/dL), required phototherapy.

Case Summary -3

A 36 years old obese primigravida, known case of chronic hypertension on regular treatment, IVF conception, presented after 30 days of embryo-transfer with positive UPT. Her height-152 cm,

weight-85 kg, BMI-36.79 kg/m2(obese).

During 1st trimester: Routine investigations including GCT were normal. Labetalol 100 mg –BD was started since the beginning of pregnancy in view of chronic hypertension. NT scan with double marker test was normal. During second trimester, weight gain was 0.6 kg/month, BP was under control with labetalol 100 mg BD up to 20 weeks, then escalated to labetalol 100 mg TDS . Anomalies ruled out. Urine protein and signs of end organ damage was absent. GTT done at 25 weeks was normal. Weight gain during 2nd trimester was 1.2 kg/month. BP was elevated even with labetalol 100 mg-TDS and hence nifedipine 20 mg was added as OD and then escalated to TDS. Urine albumin and signs of end organ damage was absent, grade-l pedal oedema appeared after 27 weeks of gestation. During 3rd trimester, growth scan was corresponding to gestational age, weight gain during third trimester was 1 kg/month, At 35 weeks BP was 160/110 mmHg even with dual antihypertensives, proteinuria was present. Diagnosed as severe pre-eclampsia. Labetalol IV regimen was started. Prophylactic MgSo4 was given as prevention of eclampsia. Pregnancy was terminated by emergency lower segment cesarean section in view of severe pre-eclampsia with imminent eclampsia. An alive female baby of weight 2.3 kg delivered. Carbetocin was given for prevention of PPH and LMWH for thrombo-prophylaxis. Her BP was monitored and controlled with antihypertensives. Delayed wound healing was present and she required repeated dressing in the postpartum period. Baby was healthy and required no interventions after delivery.

DISCUSSION

Causes of maternal obesity are complex and multifactorial. The adverse outcomes in pre-pregnancy obese women are uncertain. The increased risk of complications may be due to obesity itself or the presence of comorbidities such as type 2 diabetes mellitus, chronic hypertension etc. In studies it is proven that obese women have increased risk of GDM, gestational hypertension, pre-eclampsia, FGR, LGA, difficulty conceiving, miscarriages, post term deliveries, labor inductions, failed inductions, dystocia, cesarean deliveries, delayed wound healing, post natal complications, NICU admissions. In our cases, 2 patients had gestational hypertensions, 2 patients had GDM and 1 patient had chronic hypertension with superimposed pre-eclampsia. We encountered difficulty in managing hypertension. Labor induction required multiple doses for 2 normal deliveries. Duration of labour was prolonged. Continuous fetal monitoring during labor was difficult due to obese abdomen. Vacuum had to be used in normal deliveries for delivering the head since uterine contractions and mother's effort were inadequate. We encountered PPH in one patient which was managed with uterotonics. There is increased risk of thrombo-embolism in obese women, hence thromboprophylaxis was given. Delayed wound healing was also observed. None of the babies had severe complications after delivery.

CONCLUSIONS

Maternal obesity is associated with an increased risk of gestational hypertension, preeclampsia, gestational diabetes mellitus, dystocia, labor induction, failed induction, difficulty fetal monitoring, difficulty delivering baby, cesarean delivery, PPH, thrombo-embolism, delayed wound healing etc. Careful management of antenatal period, intra-partum period and post partum period can lead to good outcome.

Compliance With Ethical Standards :

Conflict Of Interest : The authors declare that he has no conflict of interest.

Informed Consent : informed consent was taken from the patients.

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