INTRODUCTION

Hypertensive (HTN) disorders are the most common medical complications of pregnancy and are an important cause of maternal and perinatal morbidity and mortality.\[1\] HTN is present in 6–8% of young women of childbearing age,\[2\] but the prevalence increases with age and in women with diabetes mellitus, primary renal disease, or collagen vascular diseases.

The majority of foetal complications occur due to prematurity and hypoxia.\[3\] Foetal complications related to the severity of preeclampsia, duration of the disease, and degree of proteinuria Spasms of the utero-placental circulation lead to foetal distress, accidental haemorrhage, IUGR, IUD, low birth weight, low APGAR score, NICU admissions, and early foetal death.\[4\] Perinatal morbidity is increased due to spontaneous preterm labour or iatrogenic preterm induction. Hence, an attempt was made to evaluate the types of delivery and parameters of HTN and the clinical manifestations of neonates.

MATERIALS AND METHODS

150 HTN pregnant women regularly visiting the obstetrics and gynaecology departments of Nimra Institute of Medical Sciences, Vijayawada, were studied. They were followed until delivery and 6–8 weeks post-delivery.

Inclusive Criteria

Pregnant women with gestational HTN preeclampsia or patients with essential HTN were selected for study.

Exclusion Criteria

Pregnant women with chronic renal failure, chronic liver disease, endocrine diseases, gestational diabetes mellitus, or a history of epilepsy were excluded from the study.

Method

Antenatal check up (ANS) gestational age was determined by the last menstrual period (or first trimester USG) recorded. HTN disorders and obstetric problems like preterm labour were recorded. In addition to this, neonatal parameters like gestational age, birth weight, respiratory distress syndrome, congenital pneumonia, and convulsions were also recorded.

Blood examination included: – Routine Blood examination, serum electrolyte coagulation CBC, CT, BT, Foetal monitoring included – DFMC, FHR.
NST, umbilical Doppler. USG was done to assess foetal serial growth. AFI, BPP placental location and maturity was noted.

Treatments included rest, control of blood pressure by using drugs Nefidipine, Methyldopa, labetalol, depending upon the severity of Blood pressure. To control seizures, anticonvulsants MgSO4 IV was given slowly over 5-10 minutes and 5 gm of 50% MgSO4 IM 4 hours (in alternate gluteal region).

In mild preeclampsia and gestational HTN, a termination of pregnancy was done by inducing labour at 37 week at his gestational age. In patients < 37 weeks of gestation termination of pregnancy was warranted only if maternal condition deterioetd or if there was foetal compromise.[5]

Pregnancy was terminated by LSCS for urgent termination for maternal shake as in acute fulminating preeclampsia and eclampsia when cervixes were not ripe and also for foetal sake. When foetus was in jeopardy as indicated by deranged Doppler studies (reverse diastolic flow), severe IUGR, meconium staining of liquor or foetal distress.[6]

The duration of study was August-2019 to August-2020

Statistical Analysis: Types of delivery, disorders of HTN, gestational ages, body weight of neonates, outcomes of birth were classified with percentage. The statistical analysis was done in SPSS software.

RESULTS

Table 1: Types of delivery in HTN-pregnant women 97 (64.6%) LCSC, 39 (26%) NVD, 5 (3.3%) forceps delivery, 9 (6%) consanguity.

Table 2: HTN disorders in pregnancy 30 (20%) patients had < 140 systolic, 90 diastolic; 80 (53.3%) patients had 140-159 systolic, 90-190 diastolic; 40 (26.6%) patients had > 160 systolic, >110 diastolic.

Table 3: Distribution of neonates according to gestational ages: 21 (14%) < 32 weeks, 40 (26.6%) 32–36 weeks, 89 (59.3%) 37–42 weeks.

Table 4: Study of Birth Weight in Neonates 70 (46.6%) neonates were LBW (1.5–2.5 kg), 17 (11.3%) neonates were VLBW (1–1.5 kg), 33 (22%) neonates had a normal birth weight, and 30 (20%) were IUGR neonates.

Table 5: Study of foetal outcomes in HTN pregnancies: 118 (78.6%) were alive, 32 (21.3%) were neonatal deaths.

Figure 1: Types of Delivery HTN pregnant women

Figure 2: Study of HTN disorders in pregnant mothers

Figure 3: Distribution of Neonates according to gestational age

Figure 4: Study of birth weight in Neonates

Figure 5: Foetal outcome at birth in HTN pregnancy
**DISCUSSION**

Present study of HTN disorders in pregnancy and their outcome in Neonate types of delivery were: 97 (64.6%) LCSC, 39 (26%) NVD, 5 (3.3%) for caps, 9 (6%) consonguity [Table 1]. HTN disorders in pregnancy were: 30 (20%) patients had <140 systolic, 90 diastolic, 80 (53.3%) had 140-159 systolic, 90-190 diastolic, 40 (26.6%) had >160 systolic, and < 110 diastolic BP (mm/Hg). [Table2]. Distribution of neonatal age: 21 (14%) were <32 weeks, 40 (26.6%) were between 32-36 weeks, 89 (59.3%) were between 37-42 weeks [Table 3]. The birth weight of neonates was 70 (46.6%) LBW, 17 (11.3%) VLBW, 33 (22%) had a normal birth weight, and 30 (20%) were IUGR [Table 4]. Foetal outcome at birth in HTN pregnancy: 118 (78.6%) were alive, 32 (21.3%) were neonatal deaths [Table 5]. These findings are more or less in agreement with previous studies.7-9 Maternal risk factors associated with chronic HTN include superimposed preeclampsia, deterioration of renal function, cerebrovascular accidents, congestive cardiac failure, and haemorrhage secondary to placental abruption. HTN disorders in pregnancy predispose women to acute or chronic utero-placental insufficiency, resulting in ante- or intra-partum hypoxia and even anoxia that may lead to foetal death.10 Perinatal outcome is strongly influenced by gestational age and the severity of HTN, as expressed by the need for anti-hypertensive treatment irrespective of the underlying syndrome.11 Foetal growth is a useful marker for foetal well being. A high incidence of IUGR/SGA infants in women who have pre-eclampsia has been reported.12 Pre-term delivery is due to maternal HTN disorders during pregnancy.

**Limitation of Study**

Owing to the tertiary location of the research centre, the small number of patients, and the lack of the latest techniques, we have limited findings and results.

**CONCLUSION**

Pregnancies complicated by HTN are associated with adverse foetal and neonatal outcomes in terms of the prevalence of IUGR, LBW, VLBW, and neonatal deaths. Expectant management with temporising treatment should be performed to lengthen the gestation, which may be associated with enhanced perinatal survival. Proper intensive care Close monitoring during labour, judicious timing of delivery, and NICU facilities are required for a better foetal and neonatal outcome. But this study demands further hormonal, pathophysiological, genetic, angiological,
and nutritional studies because the exact aetiology of HTN during pregnancy is still unclear.

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