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Original Research Paper

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CLINICAL PROFILE,MATERNAL MORBIDITY AND NEONATAL OUTCOMES IN LATE-ONSET SEVERE PRE-ECLAMPSIA

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ABSTRACT

Background: Late-onset severe pre-eclampsia remains an important cause of preventable maternal and neonatal morbidity in tertiary referral settings, particularly where women present late or are referred after development of severe-range hypertension and end-organ dysfunction. The present study aimed to evaluate maternal and perinatal outcomes among women with late-onset severe pre-eclampsia managed at a tertiary healthcare centre.**Methods:** This prospective observational study was conducted from April 2024 to March 2026 among 120 women with late-onset severe pre-eclampsia admitted and delivered at the study institution. Maternal and perinatal outcomes were analysed descriptively.**Results:** The mean age was 29.7 ± 6.9 years. Rural residence was recorded in 63.3%, unbooked status in 38.3%, and referral from peripheral facilities in 65.0%. Primigravidae constituted 44.2% of the study population. The mean gestational age at diagnosis was 37.1 ± 1.7 weeks. Pedal oedema was documented in 93.3%, and headache in 30.8%. Severe-range systolic blood pressure was present in 77.5%, and 3+/4+ proteinuria in 55.0%. HELLP syndrome occurred in 18.3%. All patients received oral labetalol and magnesium sulphate; nifedipine was required in 57.5%, and intravenous labetalol in 20.8%. Total vaginal delivery was achieved in 57.5%, including spontaneous vaginal delivery in 27.5%, induced vaginal delivery in 25.0% and assisted vaginal delivery in 5.0%; caesarean section was performed in 42.5%. Maternal complications included placental abruption (15.0%), postpartum haemorrhage (12.5%), acute renal failure (8.3%), pulmonary oedema (5.8%), post-admission eclampsia (6.7%), ICU admission (25.0%), and maternal mortality (1.7%). The live birth rate was 94.2%, while stillbirth/IUFD accounted for 5.8%. Low birth weight was reported in 43.4%, small-for-gestational-age status in 31.0%, a 5-minute Apgar score <7 in 15.9%, and NICU admission in 57.5% of live births.**Conclusion:** Late-onset severe pre-eclampsia was associated with substantial maternal and perinatal morbidity despite standard antihypertensive therapy, magnesium sulphate prophylaxis and timely delivery. Early detection, effective referral systems, critical-care readiness and neonatal intensive care support are essential to improve outcomes.

KEYWORDS : Late-onset pre-eclampsia; severe pre-eclampsia; hypertensive disorders of pregnancy; maternal outcome; perinatal outcome; NICU admission; tertiary care centre; India

INTRODUCTION

Hypertensive disorders of pregnancy are among the leading medical complications of pregnancy and remain major contributors to maternal and perinatal morbidity worldwide. Pre-eclampsia is defined by new-onset hypertension after 20 weeks of gestation, with proteinuria and/or evidence of maternal end-organ dysfunction, and severe disease is characterised by severe-range blood pressure or features such as thrombocytopenia, renal dysfunction, hepatic involvement, pulmonary oedema, persistent neurological symptoms or visual disturbance.[1]

Global estimates suggest that pre-eclampsia and eclampsia together affect a substantial proportion of pregnancies and contribute disproportionately to adverse outcomes in low- and middle-income countries.[2] Hypertensive disorders accounted for a notable share of global maternal deaths in WHO analyses, and the burden is amplified where antenatal detection, emergency transport, blood-bank access, critical care and neonatal services are limited.[3,4] Severe pre-eclampsia is therefore not only an obstetric diagnosis but also a marker of systemic maternal illness and fetal risk requiring coordinated multidisciplinary management.

Pre-eclampsia is clinically classified into early-onset and late-onset subtypes, with late-onset disease generally defined as onset at or beyond 34 weeks of gestation. This distinction is clinically useful because early-onset disease is more strongly related to placental dysfunction, fetal growth restriction and prematurity, whereas late-onset disease often reflects a complex interaction between maternal cardiovascular-metabolic vulnerability and placental stress near term.[5,6,7]

Although late-onset pre-eclampsia is often perceived as less severe because it occurs closer to fetal maturity, the presence of severe features changes the clinical risk profile substantially. Late-onset severe pre-eclampsia may be complicated by eclampsia, HELLP syndrome, placental abruption, postpartum haemorrhage, acute renal failure, pulmonary oedema, cerebrovascular accident, ICU admission, blood transfusion and maternal mortality. Perinatal consequences include fetal growth restriction or small-for-gestational-age status, low birth weight, neonatal depression, NICU admission, prematurity-related morbidity, stillbirth and perinatal mortality.[8,9,10] Present study was conducted to evaluate the maternal and perinatal outcomes in women with late-onset severe pre-eclampsia managed at a tertiary healthcare centre.

MATERIALS AND METHODS

This was a prospective observational study conducted among pregnant women diagnosed with late-onset severe pre-eclampsia. The study was conducted in the Department of Obstetrics and Gynaecology at Government Medical College, Chhatrapati Sambhajinagar, Maharashtra. The study was conducted over two years, from April 2024 to March 2026. The study was conducted after Institutional Ethics Committee approval. Written informed consent was obtained in the participant's preferred language.

The calculated minimum sample size was approximately 111; allowing for attrition and feasibility, 120 women were enrolled. Late-onset pre-eclampsia was defined as pre-eclampsia occurring at or beyond 34 completed weeks of gestation. Severe pre-eclampsia was diagnosed based on severe-range

blood pressure and/or maternal end-organ dysfunction, including thrombocytopenia, renal dysfunction, hepatic dysfunction, neurological symptoms, visual disturbance, pulmonary oedema or other severe features. Proteinuria was recorded as part of disease assessment.

Inclusion criteria

- Pregnant women with a confirmed diagnosis of late-onset pre-eclampsia at gestational age ≥34 weeks.
- Women admitted to and delivered at the study institution.
- Women willing to participate in the study.

Exclusion criteria

- Women with chronic hypertension predating pregnancy or diagnosed before 20 weeks of gestation.
- Women with gestational hypertension without severe features.
- Women with mild or non-severe pre-eclampsia.
- Women with gestational age <34 weeks.
- Women who delivered outside the study institution.
- Women unwilling to participate or not providing consent.
- Women with major fetal congenital anomaly where perinatal outcome assessment would be confounded.

A structured case record form was used for data collection. Sociodemographic details included age, residence, religion, booking status and referral status. Obstetric details included gravida, parity, gestational age at diagnosis and admission, previous obstetric history and presence of risk factors. Clinical assessment included presenting symptoms, general examination, blood pressure measurement, pedal oedema, neurological symptoms, visual complaints, epigastric pain, respiratory symptoms, obstetric examination and fetal assessment. Blood pressure was measured using standard technique and classified according to severity. Fundoscopy was performed when clinically indicated to assess hypertensive retinopathy.

Laboratory evaluation included complete blood count, haemoglobin, platelet count, urine protein, liver function tests, renal function tests, serum creatinine, serum uric acid where available, coagulation profile, blood group/Rh typing, serology, LDH and peripheral smear when HELLP syndrome was suspected. Obstetric ultrasonography with Doppler assessment was used to evaluate fetal biometry, amniotic fluid index, placental location and umbilical artery Doppler findings.

Women were managed according to departmental protocol. Oral labetalol was used as first-line antihypertensive therapy, nifedipine was added when blood pressure control was inadequate, and intravenous labetalol was used for acute hypertensive crises. Magnesium sulphate was administered for seizure prophylaxis using the Pritchard regimen as standard; the Zuspan regimen was used where intramuscular administration was contraindicated or unsuitable. Magnesium toxicity was monitored by urine output, patellar reflexes and respiratory rate. Maternal stabilisation, fetal surveillance, timing of delivery, induction of labour, operative delivery and caesarean section were decided according to maternal condition, fetal status, gestational age, cervical favourability and obstetric indications. Postpartum monitoring included blood pressure surveillance, urine output, symptoms of worsening disease, laboratory reassessment and management of complications.

Maternal outcomes included mode of delivery, indications for caesarean section, postpartum haemorrhage, placental abruption, acute renal failure, pulmonary oedema, HELLP syndrome, hypertensive retinopathy, cerebrovascular accident, post-admission eclampsia, ICU admission, duration of ICU stay, blood transfusion and maternal mortality. Perinatal outcomes included live birth, intrauterine fetal death/stillbirth, birth weight, SGA status, APGAR score at 1 and 5 minutes, neonatal resuscitation, NICU admission, primary indication for NICU admission, duration of NICU stay and perinatal mortality.

Data were entered in Microsoft Excel and analysed using SPSS version 26.0. Categorical variables were expressed as frequencies and percentages. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, as appropriate.

RESULTS

A total of 120 women with late-onset severe pre-eclampsia were included in the study. The mean age was 29.7 ± 6.9 years. Women aged 21–30 years constituted the largest age group (43.3%). Rural residence was more frequent than urban residence (63.3% versus 36.7%). Most women were booked (61.7%), while unbooked cases accounted for 38.3%. Referral from peripheral healthcare facilities was documented in a substantial proportion of cases (65.0%), reflecting the tertiary referral nature of the study setting.

Table 1. Sociodemographic and antenatal profile of study participants

Variable	Category	n	%
Age group, years	≤20	12	10.0
	21–25	27	22.5
	26–30	25	20.8
	31–35	19	15.8
	>35	37	30.8
Residence	Rural	76	63.3
	Urban	44	36.7
Booking status	Booked	74	61.7
	Unbooked	46	38.3
Referral status	Referred	78	65.0
	Direct admission	42	35.0

Primigravidae represented the largest obstetric group (44.2%). Gravida 2 and gravida 3 accounted for 28.3% and 20.8%, respectively, while gravida 4 and above constituted 6.7%. The mean gestational age at diagnosis was 37.1 ± 1.7 weeks. Gestational age was almost equally distributed across 34–36 weeks (34.2%), 37–38 weeks (31.7%) and >38 weeks (34.2%). Overweight and obesity were frequent, with obesity class I forming the largest BMI category (31.7%).

Table 2. Obstetric profile, gestational age and BMI

Variable	Category	n	%
Gravidity	Primigravida	53	44.2
	G2	34	28.3
	G3	25	20.8
	G4 and above	8	6.7
Gestational age at diagnosis	34–36 weeks	41	34.2
	37–38 weeks	38	31.7
	>38 weeks	41	34.2
BMI category	Normal weight	30	25.0
	Overweight	33	27.5
	Obese class I	38	31.7
	Obese class II	14	11.7
	Obese class III	5	4.2

Pedal oedema was the most frequent clinical finding (93.3%). Headache was reported in 30.8%, followed by blurring of vision (15.0%), epigastric pain (6.7%) and nausea or vomiting (5.0%). Severe-range systolic blood pressure of ≥160 mmHg was present in 77.5%, while diastolic blood pressure ≥110 mmHg was present in 70.0%. Proteinuria of 3+ or 4+ was documented in 55.0%. Anaemia was the most frequent laboratory abnormality (58.3%), followed by elevated creatinine (24.2%), thrombocytopenia (18.3%), elevated LDH (18.3%) and elevated liver enzymes (15.0%). Fundoscopy was normal in 75.0%, while abnormal fundoscopy findings were

documented in 25.0%

Table 3. Clinical and laboratory profile at admission

Variable	Category	n	%
Clinical symptom/sign*	Pedal oedema	112	93.3
	Headache	37	30.8
	Blurring of vision	18	15.0
	Epigastric pain	8	6.7
	Nausea/vomiting	6	5.0
Systolic BP	140–159 mmHg	27	22.5
	160–179 mmHg	68	56.7
	≥180 mmHg	25	20.8
Diastolic BP	90–109 mmHg	36	30.0
	≥110 mmHg	84	70.0
Proteinuria	1+	15	12.5
	2+	39	32.5
	3+	48	40.0
	4+	18	15.0
Laboratory abnormality*	Anaemia	70	58.3
	Thrombocytopenia	22	18.3
	Elevated creatinine	29	24.2
	Elevated AST/ALT	18	15.0
	Elevated LDH	22	18.3
Fundoscopy	Normal	90	75.0
	Grade I retinopathy	20	16.7
	Grade II/III retinopathy	10	8.3

*Multiple-response variable.

All patients received oral labetalol and magnesium sulphate prophylaxis (100.0%). Oral nifedipine was additionally required in 57.5%, while intravenous labetalol was used in 20.8%. The Pritchard regimen was the preferred magnesium sulphate regimen (83.3%), while the Zuspan regimen was used in 16.7%. Vaginal delivery was achieved in 57.5%, including spontaneous vaginal delivery (27.5%), induced vaginal delivery (25.0%) and assisted vaginal delivery (5.0%). Caesarean section was performed in 42.5%. Among caesarean deliveries, fetal distress was the leading indication (31.4%), followed by failed induction (29.5%), previous caesarean section (23.5%), malpresentation (7.8%) and placental abruption (7.8%).

Table 4. Treatment received, mode of delivery and LSCS indications

Variable	Category	No. of cases (n=120)	%
Antihypertensive treatment	Oral labetalol	120	100.0
	Oral nifedipine	69	57.5
	Intravenous labetalol	25	20.8
Magnesium sulphate regimen	Pritchard regimen	100	83.3
	Zuspan regimen	20	16.7
Mode of delivery	Spontaneous vaginal delivery	33	27.5
	Induced vaginal delivery	30	25.0
	Assisted vaginal delivery	6	5.0
	Total vaginal delivery	69	57.5
	Caesarean section	51	42.5
LSCS indication, n=51	Fetal distress	16	31.4
	Failed induction	15	29.5
	Previous LSCS	12	23.5
	Malpresentation	4	7.8
	Placental abruption	4	7.8

HELLP syndrome was the most frequent major maternal complication (18.3%). Placental abruption was observed in 15.0%, postpartum haemorrhage in 12.5%, acute renal failure in 8.3%, post-admission eclampsia in 6.7%, pulmonary oedema in 5.8%, cerebrovascular accident in 5.0% and hypertensive retinopathy in 2.5%. ICU admission was required in 25.0%, and blood transfusion was required in 20.8%. Maternal mortality was 1.7%.

Table 5. Maternal complications and maternal outcome

Maternal outcome	No. of cases (n=120)	%
HELLP syndrome	22	18.3
Placental abruption	18	15.0
Postpartum haemorrhage	15	12.5
Acute renal failure	10	8.3
Post-admission eclampsia	8	6.7
Pulmonary oedema	7	5.8
Cerebrovascular accident	6	5.0
Hypertensive retinopathy	3	2.5
ICU admission	30	25.0
Blood transfusion	25	20.8
Maternal death	2	1.7

Live birth was recorded in 94.2%, while IUFD/stillbirth accounted for 5.8%. The mean birth weight was 2.49 ± 0.84 kg. Low birth weight was documented in 43.4% of live births, and SGA status was recorded in 31.0%. APGAR score <7 at 1 minute was observed in 27.4%, while APGAR score <7 at 5 minutes was observed in 15.9%. Neonatal resuscitation was required in 15.9%.

Table 6. Perinatal and neonatal outcome

Variable	Category	n	%
Birth status, n=120	Live birth	113	94.2
	IUFD/stillbirth	7	5.8
Birth weight, n=113 live births	Mean birth weight	2.49 ± 0.84 kg	—
	Low birth weight <2.5 kg	49	43.4
	SGA	35	31.0
APGAR at 1 minute, n=113	<7	31	27.4
	≥7	82	72.6
APGAR at 5 minutes, n=113	<7	18	15.9
	≥7	95	84.1
Neonatal resuscitation n=113	Required	18	15.9

NICU admission was required in 57.5% of live births. The principal indications for NICU admission were prematurity (33.8%), low birth weight (26.2%), respiratory distress syndrome (20.0%), birth asphyxia (13.8%) and neonatal sepsis (6.2%). The mean NICU stay was 12.7 ± 5.0 days. Stillbirth/IUFD was recorded in 5.8% of pregnancies.

Table 7. NICU admission, indications and perinatal mortality

Variable	Category	n	%
NICU admission, (n=113 live births)	Required	65	57.5
	Not required	48	42.5
NICU indication, n=65	Prematurity	22	33.8
	Low birth weight	17	26.2
	Respiratory distress syndrome	13	20.0
	Birth asphyxia	9	13.8
	Neonatal sepsis	4	6.2
Perinatal mortality (n=120 pregnancies)	IUFD/stillbirth	7	5.8
NICU stay	Mean duration	12.7 ± 5.0 days	—

DISCUSSION

The present prospective observational study evaluated 120 women with late-onset severe pre-eclampsia managed at a

high-volume tertiary referral centre. The present prospective observational study evaluated maternal and perinatal outcomes in women with late-onset severe pre-eclampsia managed at a tertiary referral centre. The study demonstrates that late-onset severe pre-eclampsia was associated with substantial maternal morbidity, including HELLP syndrome, placental abruption, postpartum haemorrhage, acute renal failure, pulmonary oedema, post-admission eclampsia, ICU admission, blood transfusion and maternal mortality. Perinatal morbidity was also clinically relevant, with stillbirth/IUFD, low birth weight, SGA status, low Apgar score and NICU admission documented in the cohort. These findings support the need to treat late-onset severe pre-eclampsia as a high-risk obstetric condition despite presentation after 34 weeks of gestation.

Wadhwani et al.,[8] reported that women with early-onset and late-onset pre-eclampsia differed in clinical characteristics, but both subgroups contributed to maternal and neonatal risk in tertiary settings. In the present study, referral burden and unbooked status emphasise the need for antenatal risk stratification and early transport before development of severe features.

Primigravidity accounted for 44.2% of cases, consistent with the known association between nulliparity and pre-eclampsia. The proportion of women with obesity was also high, with 47.5% having BMI ≥30 kg/m². This finding is clinically plausible for late-onset disease, in which maternal cardiovascular and metabolic susceptibility is considered more prominent than the severe placental phenotype typical of early-onset disease. Wójtowicz et al.,[7] demonstrated that early- and late-onset pre-eclampsia differ in laboratory and clinical patterns when classified using ISSHP criteria, while Dimitriadis et al.,[9] summarised late-onset disease as a condition in which maternal cardiovascular maladaptation plays a major role. The present study's BMI distribution reinforces the importance of pre-pregnancy counselling, weight optimisation and antenatal surveillance in overweight and obese women.

The clinical presentation was dominated by pedal oedema and severe hypertension, while headache, visual symptoms and epigastric pain occurred in smaller proportions in the results table. Severe-range systolic blood pressure was observed in 77.5% and diastolic blood pressure ≥110 mmHg in 70.0%, indicating a high-risk admission profile. Heavy proteinuria was documented in 55.0% of patients. Although modern definitions no longer require proteinuria when end-organ dysfunction is present, the coexistence of heavy proteinuria and severe hypertension still identifies a group requiring close maternal and fetal surveillance.[1,5]

The laboratory profile showed anaemia, thrombocytopenia in HELLP cases, elevated serum creatinine and markedly elevated LDH. HELLP syndrome was present in 18.3%, predominantly Mississippi Class II. This is important because HELLP represents a severe microangiopathic phenotype with hepatic, haematological and renal involvement. In the present study, HELLP was linked clinically with critical illness, transfusion need, ICU admission and one maternal death due to multi-organ dysfunction. These findings align with the ISSHP and ACOG emphasis on platelet count, liver enzymes, renal function and neurological symptoms as markers of severe disease rather than relying only on blood pressure and proteinuria.[1,5]

All women received oral labetalol and magnesium sulphate, reflecting adherence to standard management principles. The Magpie Trial established magnesium sulphate as the anticonvulsant of choice for preventing eclampsia in women with pre-eclampsia.[11] Labetalol, nifedipine and methyldopa are accepted antihypertensive options in pregnancy, and comparative trial data support the use of oral regimens for severe hypertension when clinically appropriate.[12] In this cohort, nifedipine was required in 57.5% and intravenous labetalol in 20.8%, suggesting that a substantial proportion required combination or escalation therapy. This reinforces the need for readily available antihypertensive drugs, monitoring charts and escalation protocols at both referral and tertiary levels.

Delivery planning in late-onset severe pre-eclampsia requires balancing maternal risk against fetal maturity and neonatal readiness. In this study, total vaginal delivery was achieved in 57.5%, including assisted vaginal delivery in 5.0%, while caesarean delivery was performed in 42.5%. Fetal distress, failed induction and previous caesarean section were the leading indications for LSCS. The HYPITAT-II trial and related evidence indicate that management between 34 and 37 weeks must be individualised, because immediate delivery may reduce maternal risk but can increase neonatal respiratory morbidity when performed before full term.[13] In a tertiary referral environment, the decision often depends on maternal stabilisation, cervical status, fetal condition and availability of neonatal intensive care. The present caesarean rate reflects both disease severity and obstetric indications rather than pre-eclampsia alone.

Maternal morbidity was substantial: placental abruption occurred in 15.0%, postpartum haemorrhage in 12.5%, acute renal failure in 8.3%, pulmonary oedema in 5.8%, post-admission eclampsia in 6.7%, cerebrovascular accident in 5.0%, ICU admission in 25.0% and blood transfusion in 20.8%. Two maternal deaths occurred, one due to cerebrovascular haemorrhage and one due to HELLP-related multi-organ dysfunction. These outcomes reinforce the need for obstetric critical-care systems, aggressive blood pressure control and protocolised response to neurological symptoms. Panda et al.,[14] in a prospective Indian hospital-based study of hypertensive disorders, similarly highlighted the contribution of severe disease to maternal complications and adverse perinatal outcomes.

Perinatal outcome was also affected. Live birth occurred in 94.2%, while stillbirth/IUFD occurred in 5.8%. The mean birth weight was 2.49 kg, low birth weight was reported in 43.4% and SGA in 31.0% of live births. Neonatal depression at 5 minutes occurred in 15.9%, and NICU admission was required in 57.5%, mainly for prematurity, low birth weight and respiratory distress syndrome. These findings are consistent with the biological pathway linking severe maternal hypertension, uteroplacental insufficiency, fetal growth restriction and neonatal compromise.[15] Even in late-onset disease, fetal compromise can occur because severe maternal vasoconstriction, placental dysfunction, abruption and iatrogenic early delivery influence neonatal outcomes.

The clinical implication of this study is straightforward: late-onset severe pre-eclampsia requires the same structured surveillance and emergency preparedness as other severe hypertensive disorders of pregnancy. A practical protocol should include early recognition at the peripheral level, rapid referral when severe-range blood pressure or danger symptoms occur, immediate antihypertensive therapy, magnesium sulphate prophylaxis, expedited laboratory evaluation, fetal assessment, clear delivery planning and postpartum surveillance for delayed eclampsia or pulmonary oedema. For tertiary centres, availability of ICU beds, blood components, trained anaesthesia support and NICU services is central to reducing preventable morbidity and mortality.

This study has limitations: it was conducted at a single tertiary referral centre, which limits generalisability and introduces referral bias; the sample size was modest; a normotensive

comparison group was not included; long-term maternal follow-up, neurodevelopmental follow-up and infant growth outcomes were not assessed. The strengths of the study include its prospective design, focus on the clinically important subgroup of late-onset severe pre-eclampsia, inclusion of both maternal and perinatal outcomes, tertiary referral setting, and detailed documentation of antihypertensive therapy, magnesium sulphate use, delivery mode, maternal complications, ICU admission, blood transfusion, NICU admission and perinatal outcome.

CONCLUSION

In the present prospective observational study, late-onset severe pre-eclampsia was associated with considerable maternal morbidity despite standard management with antihypertensive therapy, magnesium sulphate prophylaxis and timely delivery. The cohort largely represented a tertiary referral population, with a high proportion of rural, referred and unbooked cases. Severe-range hypertension, significant proteinuria and laboratory evidence of end-organ involvement were frequent findings. HELLP syndrome, placental abruption, postpartum haemorrhage, acute renal failure, pulmonary oedema, post-admission eclampsia, cerebrovascular accident, ICU admission and blood transfusion requirement contributed to the maternal disease burden.

Perinatal outcome was also affected, with stillbirth, low birth weight, small-for-gestational-age status, low APGAR scores, neonatal resuscitation and NICU admission observed in the study population. These findings emphasise the need for early antenatal identification of hypertensive disorders, prompt referral before development of severe features, rapid maternal stabilisation, close fetal surveillance and timely individualised delivery planning.

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