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HYPODYNAMIC HYPERTENSION IN PREGNANCY: ROLE OF THE CLINICAL PHARMACIST IN A GYNECOLOGICAL WARD-REVIEW ARTICLE

Saba mubeena[1*], Supriya Kulkarni[1], E. Sreeja[1], P. Swathi[1], Dr. P. Soma Sekhar[2]

[1]Students of pharm D 4th year, [2]student of pharm D 4th year, [3]student of pharm D 4th year, [4] student of pharm D 4th year ,

[2]Assistant professor, Vision College of Pharmaceutical Sciences & Research, Hyderabad, Telangana, India,

Abstract:

Background: *Hypertensive disorders of pregnancy (HDP) remain a leading contributor to global maternal and perinatal morbidity. Among the hemodynamic subtypes, hypodynamic hypertension — defined by elevated total peripheral vascular resistance (TPVR), low cardiac output (CO), and reduced plasma volume — represents a particularly high-risk phenotype frequently associated with early-onset preeclampsia and fetal growth restriction (FGR). Despite growing evidence that hemodynamic profiling can guide rational pharmacotherapy, this approach remains underimplemented in most clinical settings.*

Literature Review: *A comprehensive narrative review of peer-reviewed literature published after 2020 was conducted using PubMed, ScienceDirect, and Google Scholar databases. Search terms included 'hypodynamic hypertension,' 'hemodynamics-guided therapy pregnancy,' 'clinical pharmacist hypertension pregnancy,' and related MeSH terms.*

Results: *Hypodynamic hypertension is characterised by concentric left ventricular (LV) remodeling, high TPVR, and low CO/stroke volume. The landmark multicenter PHTT study (2024) and the systematic review by Kluge et al. (2024-2026) demonstrated that antihypertensive therapy tailored to the hemodynamic phenotype — preferring dihydropyridine calcium channel blockers and nitric oxide donors over beta-blockers in hypodynamic states — significantly improved blood pressure control and reduced severe hypertension events. Clinical pharmacists are uniquely positioned to participate in hemodynamic-guided medication selection, therapeutic drug monitoring, patient education, medication reconciliation, and adverse drug reaction (ADR) surveillance.*

Conclusion: *Hemodynamic-guided pharmacotherapy represents an evidence-based paradigm shift for managing hypodynamic hypertension in pregnancy. Integration of clinical pharmacists into multidisciplinary obstetric teams — particularly within government hospital settings such as ESI Hospital — can optimise therapeutic outcomes, enhance medication safety, and reduce maternal-fetal complications. Strengthening pharmacist competencies in obstetric cardiovascular pharmacology is critical for the PharmD curriculum.*

Keywords: *Hypodynamic hypertension; Hypertensive disorders of pregnancy; Hemodynamic-guided therapy; Cardiac output; Total peripheral vascular resistance; Clinical pharmacist; Preeclampsia; Fetal growth restriction; Nifedipine; Labetalol; Antihypertensive therapy; Medication reconciliation; PharmD; ESI Hospital*

Corresponding author:

Saba Mubeena,
Student of pharm D 4th year,
Vision College of Pharmaceutical Sciences & Research ,
Boduppal, Hyderabad, Telangana
Gmail : sabamubeena2004@gmail.com

QR CODE

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1. INTRODUCTION:

Hypertensive disorders of pregnancy (HDP) complicate approximately 10-15% of all pregnancies worldwide and constitute one of the leading causes of maternal and neonatal morbidity and mortality, second only to obstetric hemorrhage [7]. These disorders encompass a clinical spectrum ranging from gestational hypertension and preeclampsia to eclampsia and HELLP syndrome, each carrying distinct pathophysiological mechanisms necessitating individualised management strategies.

Traditionally, clinical management of HDP has been predicated almost exclusively on blood pressure (BP) values, with pharmacological intervention triggered by threshold readings of >=140/90 mmHg for non-severe and >=160/110 mmHg for severe hypertension. However, two pregnant women may present with identical BP readings yet harbour fundamentally divergent cardiovascular profiles — one characterised by elevated cardiac output (CO) with low vascular resistance (hyperdynamic), and the other by low CO with markedly elevated TPVR (hypodynamic). Treating both identically is pharmacologically irrational and may cause harm.
The concept of hemodynamic phenotyping in pregnancy has gained substantial scientific traction. Evidence from the landmark multicenter 'Preeclampsia and Hypertension Target Treatment' (PHTT) study [3] and a 2024-2025 systematic review on hemodynamics-guided therapy [2] consistently demonstrate that antihypertensive therapy matched to the hemodynamic profile yields superior BP control and reduces adverse maternal-fetal outcomes. In hypodynamic hypertension, vasoconstriction is the dominant abnormality; accordingly, vasodilating agents such as dihydropyridine calcium channel blockers (e.g., nifedipine) and nitric oxide donors are rationally superior to beta-blockers that further suppress cardiac output.

Despite this evidence, hemodynamic-guided therapy remains largely underimplemented in routine obstetric practice, particularly in high-volume government hospitals such as ESI Hospital, India. The clinical pharmacist — equipped with pharmacokinetic expertise, medication safety knowledge, and proximity to both prescribers and patients — occupies a uniquely strategic role in bridging the gap between evidence and bedside practice. This narrative review addresses the pathophysiology, classification, identification, clinical implications, and management of hypodynamic hypertension in pregnancy, with a dedicated focus on the multifaceted role of the clinical pharmacist in the gynecological ward setting.

2. PATHOPHYSIOLOGY

2.1 Normal Hemodynamic Adaptations in Pregnancy

Healthy pregnancy is associated with profound cardiovascular adaptations. From the first trimester, plasma volume expands by approximately 40-50%, CO rises by 30-50% above non-pregnant baseline, and systemic vascular resistance (SVR) falls due to vasodilation mediated by nitric oxide, prostacyclin, progesterone, and the low-resistance uteroplacental circulation. Blood pressure characteristically decreases in the second trimester, reaching a nadir around 20-24 weeks, before gradually returning toward pre-pregnancy values at term. This hyperdynamic, low-resistance state ensures adequate placental perfusion and supports fetal growth.

2.2 Pathogenesis of Hypodynamic Hypertension

In a subset of hypertensive pregnant women — particularly those who develop early-onset preeclampsia or FGR — the anticipated hyperdynamic adaptation fails to occur. Instead, the maternal cardiovascular system adopts a hypodynamic profile, often before clinical manifestations of hypertension appear. Hemodynamic studies consistently demonstrate that such women exhibit reduced CO, low stroke volume, concentric LV geometry (increased wall thickness relative to cavity size), and markedly elevated TPVR — frequently from the first trimester [1,21].

The mechanistic basis is multifactorial:
Defective Trophoblastic Invasion: Impaired spiral artery remodeling results in high-resistance uteroplacental flow, placental hypoxia, systemic endothelial dysfunction, and disproportionate generation of anti-angiogenic factors — particularly soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin — relative to pro-angiogenic placental growth factor (PlGF), promoting systemic vasoconstriction.
Endothelial Dysfunction and Vasoconstriction: Elevated sFlt-1 neutralises circulating vascular endothelial growth factor (VEGF) and PlGF, impairing nitric oxide-dependent vasodilation. The net effect is peripheral arterial vasoconstriction, elevated TPVR, and compensatory reduction in venous capacitance.
Relative Hypovolemia: Women with hypodynamic hypertension exhibit attenuated plasma volume expansion. This reduced preload directly contributes to low stroke volume and CO.
Concentric LV Remodeling: Chronic pressure overload from elevated TPVR induces concentric LV hypertrophy, reducing LV cavity size and diastolic compliance, further constraining CO.

The net result is a self-perpetuating cycle: high TPVR elevates afterload, reduces stroke volume and CO, triggers compensatory venous constriction, worsens hypovolemia, and exacerbates uteroplacental insufficiency.

2.3 Differentiating Hypodynamic from Hyperdynamic Hypertension

The distinction between these two hemodynamic subtypes is clinically critical because their pharmacological management is diametrically opposed. In hyperdynamic hypertension (high CO, low-to-normal TPVR), beta-blockers are physiologically appropriate. In hypodynamic hypertension (high TPVR, low CO), agents that reduce vasoconstriction — calcium channel blockers, hydralazine, nitrates — are pharmacologically rational, while beta-blockers may worsen uteroplacental perfusion and precipitate fetal compromise [2,3].

3. CLASSIFICATION OF HYPERTENSION IN PREGNANCY

The contemporary classification of HDP is based on guidelines of the International Society for the Study of Hypertension in Pregnancy (ISSHP), the American College of Obstetricians and Gynecologists (ACOG), and the 2024 European Society of Cardiology (ESC) guidelines [14,25]. Hypertension in pregnancy is defined as a systolic BP >=140 mmHg and/or diastolic BP >=90 mmHg on two measurements at least four hours apart. Four principal categories are recognised.

Table 1: Classification of Hypertensive Disorders of Pregnancy and Hemodynamic Tendencies

Category	Onset	Key Features	Hemodynamic Tendency
Chronic (Pre-existing) Hypertension	Before 20 weeks or pre-pregnancy	Persists >12 weeks postpartum; may be primary or secondary	Variable; often hyperdynamic early, may shift hypodynamic
Gestational Hypertension	>=20 weeks; no proteinuria/organ damage	Resolves within 12 weeks postpartum	Often hyperdynamic; hypodynamic in FGR-associated cases
Preeclampsia	>=20 weeks; with proteinuria and/or organ damage	End-organ involvement; can progress to eclampsia/HELLP	Early-onset: typically hypodynamic; late-onset: often hyperdynamic
Superimposed Preeclampsia	Chronic hypertension + new-onset proteinuria/organ damage	Higher risk of adverse maternal-fetal outcomes	Usually hypodynamic; significant vasoconstriction

Within the framework of hemodynamic phenotyping, early-onset preeclampsia (before 34 weeks) is most strongly associated with hypodynamic cardiovascular profiles, concentric LV geometry, high TPVR, low CO, and FGR. Late-onset preeclampsia (after 34 weeks) more frequently presents with a hyperdynamic or mixed profile — a distinction with direct implications for pharmacological selection [1,21]. The 2024 ESC guidelines define severe hypertension as systolic BP >=160 mmHg or diastolic BP >=110 mmHg, requiring urgent intervention within 30-60 minutes [14].

4. IDENTIFICATION AND DIAGNOSIS

4.1 Clinical Assessment of Hemodynamic Profile

The gold standard for maternal hemodynamic assessment is transthoracic echocardiography (TTE), which allows direct quantification of CO, stroke volume, LV geometry, and diastolic function. However, TTE requires trained personnel and equipment often unavailable in high-volume Indian public hospitals [1]. Non-invasive cardiac output monitors, such as the Ultrasound Cardiac Output Monitor (USCOM) used in the PHTT multicenter study, offer accurate, reproducible hemodynamic profiling without the complexity of echocardiography, measuring CO, stroke volume, and TPVR via suprasternal Doppler auscultation [3,4].

4.2 Surrogate Clinical Identification Without Devices

In resource-limited settings, simultaneous BP and heart rate measurement provides a reasonable surrogate estimation of hemodynamic profile, as described by Novelli, Vasapollo et al. (2025) [1]:

Hypodynamic Pattern: High BP + Low/Normal Heart Rate — indicates high TPVR with insufficient compensatory tachycardia; CO likely reduced.
Hyperdynamic Pattern: High BP + Elevated Heart Rate — indicates high CO with compensatory tachycardia; TPVR relatively low.
While this surrogate method lacks precision, it provides a clinically pragmatic starting point for pharmacological decision-making in technology-limited settings.

4.3 Biomarker-Assisted Diagnosis

The sFlt-1:PlGF ratio has been validated as a powerful biomarker for the prediction and diagnosis of preeclampsia, particularly early-onset disease. An sFlt-1:PlGF ratio >=38 effectively identifies women at imminent risk of preeclampsia with a negative predictive value exceeding 99%. Elevated sFlt-1 reflects anti-angiogenic dysregulation characteristic of hypodynamic placental disease, though its availability remains limited in developing-country public hospital settings.

4.4 Doppler Ultrasound and Fetal Assessment

Uterine artery Doppler assessment revealing increased pulsatility index and/or notching in the first or early second trimester identifies women at elevated risk of placental insufficiency and hypodynamic HDP. Serial umbilical artery Doppler monitoring guides decisions about timing of delivery in women with established hypodynamic hypertension and suspected FGR. Absent or reversed end-diastolic flow in the umbilical artery signals critical fetal compromise and mandates immediate obstetric intervention.

5. CLINICAL IMPLICATIONS

5.1 Maternal Complications

Hypodynamic hypertension carries a disproportionately high burden of severe maternal outcomes. Concentric LV remodeling and diastolic dysfunction predispose affected women to acute pulmonary edema. High TPVR and endothelial dysfunction contribute to hypertensive encephalopathy, intracranial hemorrhage, and acute kidney injury. HELLP syndrome is more commonly encountered in women with early-onset, hypodynamic preeclampsia. Furthermore, inappropriate use of beta-blockers in hypodynamic hypertension — facilitated by the absence of hemodynamic assessment — further reduces CO, worsens uteroplacental blood flow, and increases the risk of FGR and perinatal mortality, constituting a critical preventable medication harm [2,3].

5.2 Fetal and Neonatal Implications

Hypodynamic maternal hemodynamics directly compromise uteroplacental perfusion: high TPVR reduces the pressure gradient across the placental bed; hypovolemia decreases uterine blood flow; endothelial dysfunction impairs spiral artery remodeling. Collectively, these changes result in chronic fetal hypoxia, FGR, oligohydramnios, and abnormal Doppler findings. Neonates are at elevated risk of preterm birth, low birth weight, NICU admission, respiratory distress syndrome, necrotising enterocolitis, intraventricular hemorrhage, and long-term neurodevelopmental sequelae, imposing a substantial economic and social burden.

5.3 Long-Term Cardiovascular Risk

Women who experience preeclampsia — particularly the early-onset hypodynamic subtype — face a significantly elevated lifetime cardiovascular risk. Evidence from a 2024 systematic review indicates a 2-8-fold increased risk of hypertension, a 2-fold increased risk of ischemic heart disease, and an elevated risk of stroke in later life [11]. The concentric LV remodeling that characterises hypodynamic preeclampsia often persists postnatally, representing a subclinical cardiovascular risk state warranting long-term cardioprotective follow-up.

6. MANAGEMENT

6.1 Non-Pharmacological Management

Non-pharmacological interventions serve as foundational adjuncts to pharmacotherapy and are particularly important in mild hypertension (140-159/90-109 mmHg) as initial or concurrent management.
Dietary Sodium Restriction: Moderate restriction (2-4 g/day) is generally recommended. However, severe sodium restriction is contraindicated in hypodynamic hypertension because it exacerbates pre-existing relative hypovolemia, further reducing preload, stroke volume, and CO.
Rest and Activity Modification: Complete bed rest is no longer routinely recommended due to thromboembolic risk. Lateral decubitus positioning (left lateral tilt) improves venous return and reduces aortocaval compression from mid-pregnancy onwards [23].
Low-Dose Aspirin Prophylaxis: ACOG, NICE, and ISSHP recommend low-dose aspirin (75-150 mg/day, initiated before 16 weeks) in women at high risk for preeclampsia [25]. Aspirin reduces the risk of preterm preeclampsia by approximately 60% (ASPRE trial) [24] by inhibiting thromboxane A2-mediated vasoconstriction — a mechanism particularly relevant to hypodynamic vasoconstrictive pathophysiology.
Calcium Supplementation: WHO recommends calcium supplementation (1.5-2 g/day) in populations with low dietary calcium intake, as it is associated with a reduced risk of preeclampsia — relevant in many Indian public hospital populations.

Antenatal Monitoring: Frequent BP monitoring, serial fetal growth scans, and Doppler ultrasonography are non-pharmacological cornerstones of HDP management.

6.2 Pharmacological Management

6.2.1 Principles of Hemodynamic-Guided Therapy

The central principle of hemodynamic-guided antihypertensive therapy — validated by the PHTT study [3] and the systematic review by Kluge et al. [2] — is that pharmacological agents should be selected not only to lower BP but to correct the underlying hemodynamic abnormality:

Hypodynamic Profile (High TPVR, Low CO): Use vasodilating agents. Preferred: dihydropyridine calcium channel blockers (nifedipine), direct vasodilators (hydralazine), alpha-methyldopa. Avoid or use with extreme caution: pure beta-blockers and high-dose labetalol, as these reduce CO and worsen the hemodynamic state.

Hyperdynamic Profile (High CO, Low-Normal TPVR): Beta-blockers (labetalol, metoprolol) and centrally acting agents (alpha-methyldopa) are appropriate. Calcium channel blockers may also be used.

6.2.2 First-Line Antihypertensive Agents

Table 2: First-Line Antihypertensive Agents in Pregnancy and Their Hemodynamic Suitability

Drug	Class	Mechanism	Hemodynamic Effect	Dose (Oral)	Use in Hypodynamic HDP
Nifedipine (extended-release)	Dihydro pyridine CCB	Blocks L-type Ca2+ channels; arterial vasodilation	↓ TPVR, ↑ CO	30-90 mg/day	PREFERRED — vasodilates arterial bed, corrects high TPVR
Alpha-methyldopa	Central alpha-2 agonist	Reduces sympathetic outflow; mild vasodilation	Modest ↓ TPVR, neutral CO	250-2000 mg/day (TID-QID)	Acceptable — limited hemodynamic advantage over CCBs
Hydralazine (IV)	Direct vasodilator	Direct smooth muscle relaxation	↓ TPVR, ↑ CO (reflex)	5-10 mg IV bolus	Preferred IV agent in acute severe hypodynamic hypertension
Labetalol (IV/oral)	alpha/beta blocker	Blocks alpha-1 and beta-1/beta-2 receptors	↓ CO and ↓ TPVR	20-80 mg IV; 100-400 mg BD oral	CAUTION — reduces CO; irrational as monotherapy in hypodynamic state
Atenolol / Metoprolol	Selective beta-1 blocker	Reduces HR and CO	↓ CO significantly	Varies	AVOID — worsens low CO; associated with SGA/FGR in hypodynamic women
Isosorbide mononitrate	Nitric oxide donor	NO-mediated venous and arterial vasodilation	↓ TPVR, ↑ venous capacitance	10-20 mg BD	Beneficial — corrects arterial and venous vasoconstriction

6.2.3 Acute Severe Hypertension

Acute severe hypertension (systolic >=160 mmHg or diastolic >=110 mmHg) requires urgent pharmacological intervention within 30-60 minutes to prevent maternal stroke. First-line parenteral agents include intravenous labetalol (20-80 mg IV bolus, repeated every 10 minutes to a maximum of 300 mg) and intravenous hydralazine (5-10 mg IV bolus every 20 minutes). Oral nifedipine immediate-release (10 mg orally) is an acceptable alternative when IV access is unavailable [22]. Intravenous sodium nitroprusside is reserved as a last resort due to fetal cyanide toxicity risk and should not be used for more than four hours. In the hypodynamic patient, hydralazine is hemodynamically preferable to labetalol as the initial IV agent, as it reduces TPVR while maintaining or reflexively increasing CO [5].

6.2.4 Magnesium Sulfate for Seizure Prophylaxis

Magnesium sulfate (MgSO4) is the drug of choice for prevention and treatment of eclampsia in women with severe preeclampsia. The standard Pritchard regimen comprises a 4 g IV loading dose over 20 minutes, followed by 1-2 g/hour IV maintenance. MgSO4 also exerts mild vasodilating properties, making it doubly beneficial in the hypodynamic context. Monitoring for toxicity — loss of patellar reflexes (Mg^{2+} 7-10 mEq/L), respiratory depression (>10 mEq/L), and cardiac arrest (>15 mEq/L) — is mandatory. Calcium gluconate (1 g IV) serves as the antidote.

6.2.5 Corticosteroids for Fetal Lung Maturation
In pregnancies with anticipated preterm delivery before 34 weeks, antenatal corticosteroids (betamethasone 12 mg IM x 2 doses, 24 hours apart; or dexamethasone 6 mg IM x 4 doses, 12 hours apart) are recommended to reduce the risks of respiratory distress syndrome, intraventricular hemorrhage, and neonatal mortality. Corticosteroids may transiently increase BP in the 24-48 hours following administration, requiring intensified monitoring.

6.2.6 Postpartum Management
Postpartum hypertension is a frequently underappreciated risk. The maximum BP following delivery typically occurs between days 3-7 postpartum as fluid shifts back into the intravascular compartment. Antihypertensive therapy should be continued postpartum if initiated antenatally. After delivery, ACE inhibitors (captopril, enalapril) become an additional option; both are compatible with breastfeeding at standard doses. NSAIDs — frequently used for postpartum analgesia — should be avoided in women with hypertension, as they cause sodium retention and can precipitate hypertensive crises. Women should receive written discharge instructions detailing BP monitoring schedules and warning symptoms.

7. ROLE OF THE CLINICAL PHARMACIST
The clinical pharmacist in the gynecological ward occupies a pivotal position within the multidisciplinary obstetric team. In hypodynamic hypertension, where the stakes of inappropriate pharmacotherapy are exceptionally high — carrying risks of FGR, maternal stroke, and organ failure — pharmacist involvement addresses critical gaps in medication safety, rational prescribing, patient education, and care coordination.

7.1 Hemodynamic-Guided Pharmacotherapy Facilitation
The pharmacist serves as a knowledge resource and active clinical consultant regarding the hemodynamic rationale for drug selection. When a prescriber orders a beta-blocker as first-line therapy in a patient with signs of hypodynamic hypertension (low heart rate, high BP, FGR), the clinical pharmacist should initiate a structured pharmacist-physician consultation, present the hemodynamic evidence — citing the PHTT study [3] and Kluge et al. [2] — and propose a hemodynamically appropriate alternative such as extended-release nifedipine or hydralazine. This constitutes a clinical medication review rather than a mere dispensing function.

7.2 Medication Reconciliation
Pregnant women admitted to the gynecological ward frequently have pre-existing medications requiring adjustment or discontinuation. The pharmacist performs comprehensive medication reconciliation at admission, identifying and resolving:

Teratogenic agents: ACE inhibitors and angiotensin receptor blockers are absolutely contraindicated in the second and third trimesters (cause fetal renal failure, oligohydramnios, and neonatal anuria); direct renin inhibitors (aliskiren) are similarly contraindicated.

Potentially harmful combinations: NSAIDs (cause oligohydramnios and fetal ductus arteriosus constriction after 28 weeks), aminoglycosides (nephrotoxicity), and ergot alkaloids (severe vasoconstriction in an already vasoconstricted patient).

Dose adjustments for physiological changes: Pregnancy increases glomerular filtration rate by 50%, hepatic enzymatic activity, and plasma volume — collectively altering pharmacokinetics of many antihypertensives. Methyldopa requires more frequent dosing due to increased renal clearance; labetalol's volume of distribution expands significantly.

7.3 Therapeutic Drug Monitoring and ADR Surveillance
The clinical pharmacist is responsible for ongoing monitoring of antihypertensive therapy efficacy and toxicity. Key monitoring parameters include: $MgSO_4$ toxicity monitoring (urine output >25 mL/hr, respiratory rate >12/min, patellar reflexes; calcium gluconate maintained at bedside); nifedipine monitoring for reflex tachycardia, peripheral edema, hypotension (particularly with concomitant $MgSO_4$), and headache; hydralazine monitoring for lupus-like syndrome and reflex tachycardia; and labetalol monitoring for neonatal bradycardia, hypoglycemia, and respiratory depression when used antepartum. ADR documentation and reporting to the Pharmacovigilance Programme of India (PvPI) is a core pharmacist responsibility in ESI Hospital settings.

7.4 Patient and Family Education
PharmD students and clinical pharmacists deliver structured, individualised medication counseling to pregnant women with HDP. Evidence from multiple post-2020 studies — including Gholami et al. (Front Cardiovasc Med, 2022) [15] and a 2024 Indian hospital-based trial (IJPRA, 2024) [10] — confirms that pharmacist educational interventions significantly improve women's knowledge, correct beliefs about medication safety in pregnancy, and enhance adherence. Key counseling components

include: rationale for antihypertensive treatment and consequences of non-adherence; correct administration technique (e.g., swallowing extended-release nifedipine whole; not crushing); warning symptoms requiring immediate attention (severe headache, visual disturbances, epigastric pain, decreased fetal movements); safe use of supplements; and postpartum BP monitoring. In ESI Hospital, counseling should be adapted using simple language, visual aids, and teach-back verification methods.

7.5 Interdisciplinary Collaboration and Documentation

The clinical pharmacist participates in ward rounds, communicates drug-related problems to obstetricians and anaesthesiologists, documents clinical pharmacy interventions in the medical record, and contributes to clinical audit and drug usage evaluation. Within ESI Hospital, where institutional protocols may not yet incorporate hemodynamic-guided therapy, the pharmacist can champion protocol development and staff training to standardise care and reduce prescribing variability.

7.6 Evidence of Pharmacist Impact

The systematic review by Fitria (J Basic Clin Pharma, 2024) [8] and the review by Boachie-Ansah et al. (J Clin Hypertens, 2025) [6] demonstrate that pharmacist involvement in HDP management is associated with improved BP control, enhanced medication adherence, reduced medication errors, and greater patient satisfaction. The pharmacist-led model is particularly effective in settings with heavy physician workload, where the pharmacist's continuous ward presence enables real-time medication safety surveillance.

Table 3: Summary of Clinical Pharmacist Roles in Hypodynamic Hypertension Management

Pharmacist Role	Key Activities	Evidence/Outcome
Hemodynamic-guided pharmacotherapy	Drug selection consultation; proposing nifedipine/hydralazine over beta-blockers in hypodynamic states	Improved BP control; reduced FGR (PHTT study)
Medication reconciliation	Identifying teratogenic agents (ACEi, ARBs); adjusting doses for pregnancy pharmacokinetics; resolving drug interactions	Reduced medication errors; enhanced patient safety
Therapeutic drug monitoring	MgSO4 toxicity surveillance; nifedipine/hydralazine/labetalol monitoring; ADR documentation and PvPI reporting	Early detection of toxicity; regulatory compliance
Patient and family education	Structured counseling on administration, warning symptoms, adherence; teach-back verification	Improved adherence and knowledge (Gholami et al., 2022; IJPRA, 2024)
Interdisciplinary collaboration	Ward rounds participation; documentation; protocol development; staff training on hemodynamic-guided therapy	Reduced prescribing variability; improved team communication

8. CHALLENGES IN CLINICAL PRACTICE

8.1 Limited Hemodynamic Assessment Infrastructure

The most fundamental barrier to implementing hemodynamic-guided therapy in settings like ESI Hospital is the absence of point-of-care hemodynamic monitoring devices. Echocardiography is not universally available in district and sub-district public hospitals, and USCOM devices are expensive and require training. Until affordable, validated, portable monitoring tools are widely deployed, clinicians will continue to rely on surrogate clinical markers, which, while informative, are imprecise.

8.2 Institutional Prescribing Inertia and Protocol Gaps

Established prescribing habits — particularly the default use of labetalol or methyldopa as universal first-line agents regardless of hemodynamic profile — are deeply entrenched in many obstetric units. The absence of institutional protocols incorporating hemodynamic phenotyping perpetuates this pattern. Changing prescriber behaviour requires robust evidence dissemination, institutional leadership support, and the development of locally adapted clinical decision algorithms.

8.3 Pharmacist-Physician Communication Barriers

In hierarchical hospital environments, clinical pharmacists may face reluctance from physicians when suggesting therapeutic modifications. Developing a culture of collaborative, non-judgmental pharmacist-physician communication — grounded in shared patient safety goals — is essential for intervention acceptance. Formal clinical pharmacy consultation models, supported by institutional policy, are superior to informal suggestions in ensuring intervention uptake.

8.4 Rapidly Changing Evidence Base

The field of hemodynamic-guided therapy in HDP is evolving rapidly. PharmD students and pharmacists must maintain currency with the latest evidence, including PHTT trial results and ongoing trials testing hemodynamic phenotyping platforms, through continuous professional development, journal clubs, and structured review of emerging guidelines.

8.5 Pharmacokinetic Complexity in Pregnancy

Pregnancy-induced pharmacokinetic changes — including increased volume of distribution, altered protein binding, enhanced hepatic metabolism via CYP3A4 induction, and increased renal clearance — affect the disposition of virtually every antihypertensive drug. Failure to account for these changes can result in subtherapeutic concentrations, inadequate BP control, or unexpected toxicity. The pharmacist is uniquely trained to identify and address these challenges.

9. FUTURE PERSPECTIVES

The field of hemodynamic-guided therapy for HDP is at an inflection point. Several important directions are emerging:

Affordable Point-of-Care Hemodynamic Monitoring: Development and validation of low-cost, portable, operator-independent hemodynamic monitoring tools adapted for resource-limited settings will be transformative. Smartphone-based photoplethysmography and AI-driven cardiac output estimation hold particular promise for public health systems such as the ESI Hospital network.

Artificial Intelligence and Precision Medicine: Machine learning algorithms trained on large maternal hemodynamic datasets may enable individualised prediction of optimal antihypertensive drug selection and titration, integrating biomarker data (sFlt-1:PlGF ratios), Doppler parameters, and clinical variables.

Digital Health and Remote Monitoring: Telehealth-based BP monitoring platforms — evaluated in the BUMP2 and Safe@Home studies (JACC Advances, 2024) [11] — demonstrated high patient satisfaction, improved postpartum BP surveillance, and reduced hospitalisations. Integration of these platforms into ESI Hospital outpatient workflow could address critical gaps in postpartum hypertension management.

Nitric Oxide Donors in Hypodynamic Preeclampsia: Ongoing trials investigating isosorbide mononitrate and L-arginine supplementation as adjunctive vasodilatory therapies in early-onset hypodynamic preeclampsia show preliminary benefits on umbilical artery Doppler parameters and fetal growth, warranting Phase III evaluation.

Pharmacist-Led Hemodynamic Clinics: As pharmacist prescribing authority expands globally, pharmacist-led antenatal hypertension clinics incorporating BP monitoring, medication titration, and hemodynamic risk stratification represent a scalable, cost-effective model for ESI Hospital and similar public health institutions.

Postpartum Cardiovascular Follow-Up: Given the substantially elevated long-term cardiovascular risk following hypodynamic preeclampsia, integrated postpartum cardiovascular surveillance programmes — including echocardiographic assessment, lipid profiling, and glucose tolerance testing at 3 and 12 months — are needed. Clinical pharmacists can coordinate and manage these programmes within hospital-based chronic disease management frameworks.

10. DISCUSSION

The evidence presented in this narrative review underscores a critical and actionable paradigm shift in the management of hypertensive disorders of pregnancy. For too long, antihypertensive prescribing in obstetrics has been guided almost exclusively by BP thresholds, without consideration of the underlying hemodynamic phenotype. As demonstrated by the PHTT multicenter study [3] and Kluge et al.'s systematic review [2], this hemodynamically agnostic approach is not only suboptimal but potentially harmful in women with hypodynamic hypertension, in whom beta-blockers — a cornerstone of standard hypertension management — can precipitate worsening uteroplacental insufficiency and fetal compromise.

The surrogate clinical approach of correlating BP with heart rate patterns, as validated by Novelli and Vasapollo et al. [1], offers a pragmatic solution for resource-limited settings. In ESI Hospital, where echocardiography and USCOM devices may not be readily available, this simple bedside assessment can meaningfully inform first-line drug selection, reducing the risk of prescribing hemodynamically inappropriate agents.

The role of the clinical pharmacist in this context extends far beyond drug dispensing. As demonstrated by Boachie-Ansah et al. [6] and Fitria [8], pharmacist-led interventions in HDP are associated with measurable improvements in BP control, adherence, and medication safety. In the high-volume, resource-constrained environment of ESI Hospital's gynecological ward, the pharmacist's continuous ward presence and clinical expertise make them an indispensable member of the obstetric team. Pharmacist-driven medication reconciliation at admission, structured patient education, and real-time ADR surveillance directly address the most common and preventable sources of medication-related harm in this patient population.

The integration of hemodynamic-guided therapy into institutional protocols, supported by pharmacist-led education and clinical decision algorithms, represents a feasible and cost-effective strategy for improving maternal and fetal outcomes in public hospital settings. The PharmD curriculum must accordingly evolve to include foundational competencies in maternal cardiovascular pharmacology, hemodynamic pharmacotherapy, and obstetric medication safety.

11. CONCLUSION:

Hypodynamic hypertension in pregnancy — characterised by elevated TPVR, low CO, concentric LV remodeling, and relative hypovolemia — represents a high-risk hemodynamic phenotype most commonly associated with early-onset preeclampsia and fetal growth restriction. The traditional blood-pressure-centric approach to antihypertensive management, while clinically indispensable, is insufficient. Current evidence unambiguously supports pharmacological therapy informed by the underlying hemodynamic profile — vasodilating agents (nifedipine, hydralazine) for hypodynamic states, with judicious avoidance of pure beta-blockers that further suppress an already compromised cardiac output.

The clinical pharmacist in the gynecological ward is an essential, evidence-based contributor to optimal management of hypodynamic HDP. From hemodynamic-guided pharmacotherapy consultation and medication reconciliation to patient education, ADR surveillance, therapeutic drug monitoring, and interdisciplinary collaboration, the pharmacist's role firmly positions the profession as a cornerstone of obstetric patient safety. In the context of Indian public hospitals such as ESI Hospital — where physician-to-patient ratios are high and complex obstetric comorbidities are common — the integration of well-trained PharmD clinical pharmacists into multidisciplinary obstetric teams is both beneficial and necessary.

PharmD students must develop foundational competencies in maternal cardiovascular pharmacology, hemodynamic pharmacotherapy principles, and obstetric medication safety to fulfil this role effectively. The evidence reviewed herein provides a scholarly foundation for that development and underscores the urgency of implementing hemodynamic-guided, pharmacist-supported care models for hypertensive disorders of pregnancy.

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