Saudi Journal of Medicine and Public Health

https://saudijmph.com/index.php/pub

https://doi.org/10.64483/202522627

Nursing Management of Neonatal Hypertension in the Neonatal Intensive Care Unit-An Updated Review

Fahad Saud Mohammad Alsulaiman [(1)] , Khawlah Ali Hamad Hakami [(2)] , Hind Mkhoot Alfhigi [(3)] , Slama Abdallah Aldukhil Allah [(3)] , Salha Shahi Alanzi [(4)] , Arwa Ghunaim Aldhaferi [(5)] , Masiraha Hawaj Noman Aljameeli [(6)] , Saimah Hassan Ibrahim Habib [(7)] , Sameer Ali Ibrahim Zakri [(8)] , Ehdaa Kazim Mohammed Yahya [(9)] , Esahaq Yacop Aljubili [(10)] , Al-Hanouf Suleiman Al-Numan [(11)] , Atallah Ateya Badi Alruwaili [(12)]

(1) King Salman Hospital – Riyadh,Ministry of Health, Saudi Arabia,
(2) Jazan General Hospital,Ministry of Health, Saudi Arabia,
(3) Ministry Of Health, Saudi Arabia,
(4) Al-Khaleej 2 Primary Health Care Center – Riyadh,Ministry of Health, Saudi Arabia,
(5) Hafr Al-Batin Central Hospital,Ministry of Health, Saudi Arabia,
(6) Childbirth and Pediatrics,Ministry of Health, Saudi Arabia,
(7) Damad General Hospital – Jazan,Ministry of Health, Saudi Arabia,
(8) Jazan Health Cluster – External Treatment Requests,Ministry of Health, Saudi Arabia,
(9) King Salman Bin Abdulaziz Medical City,Ministry of Health, Saudi Arabia,
(10) Maternity and Children's Hospital – Al-Ahsa,Ministry of Health, Saudi Arabia,
(11) Al-Basatin Health Center,Ministry of Health, Saudi Arabia,
(12) Workplace-Jadidah ArarHospital, Ministry of Health, Saudi Arabia

Abstract

Background: Neonatal hypertension is increasingly recognized as a clinically significant condition, particularly among premature infants in neonatal intensive care units. Historically, limited normative blood pressure data and variability in measurement methods hindered early diagnosis and management. The condition arises from multifactorial etiologies, including prematurity-related complications, renal pathology, cardiovascular abnormalities, genetic factors, and environmental exposures.

Aim: This review aims to provide an updated, comprehensive overview of the nursing management of neonatal hypertension, emphasizing diagnostic approaches, etiologic considerations, monitoring techniques, and evidence-based treatment strategies.

Methods: A narrative review methodology was employed, synthesizing current literature related to blood pressure measurement techniques, pathophysiology, epidemiology, clinical presentation, diagnostic evaluation, management, prognosis, and interprofessional collaboration in neonatal hypertension.

Results: Accurate diagnosis relies on repeated blood pressure measurements using standardized invasive or noninvasive methods. Prematurity and renal disorders remain the leading causes. Nonrenal factors—such as bronchopulmonary dysplasia, endocrine disorders, and environmental exposures—also contribute. Management centers on addressing underlying causes, optimizing nursing surveillance, and employing pharmacologic or surgical treatments when indicated. Early intervention mitigates risks of end-organ damage, including renal injury, cardiac dysfunction, and neurologic sequelae. Effective care requires interprofessional coordination among neonatologists, nephrologists, nurses, pharmacists, and caregivers.

Conclusion: Neonatal hypertension is a complex, multifactorial condition requiring vigilant nursing assessment, accurate monitoring, and individualized treatment. Early recognition, thorough evaluation, and targeted management substantially improve outcomes, while structured interprofessional collaboration enhances continuity of care and long-term health.

Keywords: Neonatal hypertension, prematurity, blood pressure measurement, nursing management, renal pathology, NICU, antihypertensive therapy.

Introduction

Neonatal hypertension, although observed in clinical settings for more than four decades, has only relatively recently been acknowledged as a distinct and clinically relevant neonatal morbidity. Early recognition and systematic study of this condition were delayed largely due to the lack of robust and comprehensive normative blood pressure data specific to neonates. Without reliable reference standards, clinicians faced substantial difficulty in distinguishing pathological hypertension from physiological variation during the neonatal period. This challenge was compounded by historical limitations in both invasive and noninvasive blood pressure measurement technologies, which restricted the accuracy and consistency of blood pressure assessment in newborns, particularly in preterm and critically ill infants. The interpretation of neonatal blood pressure is further complicated by the inherent heterogeneity of the neonatal population. Blood pressure values vary

*Corresponding author e-mail: fsalsulaiman@moh.gov.sa (Fahad Saud Mohammad Alsulaiman).

Receive Date: 07 December 2025, Revise Date: 29 December 2025, Accept Date: 31 December 2025

considerably according to gestational age, postnatal age, birth weight, and sex, creating wide physiological ranges that evolve rapidly during early life. These dynamic changes have hindered the establishment of universally accepted thresholds for normal and abnormal blood pressure. As a result, the absence of standardized definitions historically limited both clinical decision making and research efforts aimed at understanding neonatal hypertension. Despite growing awareness, the natural history and long-term consequences of neonatal hypertension remain inadequately characterized. Current evidence provides limited insight into the progression of elevated blood pressure during infancy and childhood, as well as its potential association with future cardiovascular morbidity. Furthermore, uncertainty persists regarding the most appropriate pharmacological management of affected neonates. No clear consensus exists concerning the optimal timing, selection, or duration of antihypertensive therapy in this vulnerable population, reflecting gaps in high-quality clinical trials and long-term follow-up studies.[1] Consequently, management strategies often rely on extrapolation from older pediatric populations or on institutional experience rather than standardized evidence-based guidelines.

Ongoing research continues to refine the conceptual framework surrounding neonatal hypertension, with increasing focus on its definition, epidemiology, etiological factors, and pathophysiological mechanisms. Advances in neonatal monitoring and blood pressure measurement have improved diagnostic accuracy, enabling more consistent identification of affected infants. These developments have supported efforts to better delineate risk factors and to establish clearer diagnostic and therapeutic pathways. From a diagnostic standpoint, neonatal hypertension is defined by systolic or diastolic blood pressure measurements that meet or exceed the 95th percentile for postconceptual age, documented on three separate occasions to ensure persistence rather than transient elevation.[2] Values surpassing the 99th percentile are indicative of severe hypertension and necessitate prompt initiation of antihypertensive therapy, alongside a thorough evaluation to identify underlying causes.[3] This stratified approach underscores the importance of accurate measurement, repeated assessment, and contextual interpretation within the broader clinical profile of the neonate.

Blood Pressure Measurements

Blood pressure assessment in neonates can be performed using either invasive or noninvasive techniques, with the choice of method largely determined by the clinical condition, gestational maturity, and hemodynamic stability of the infant. Accurate measurement is essential, as neonatal blood pressure values are low, highly variable, and sensitive to technical factors. Errors in measurement may lead to misclassification, delayed diagnosis, or inappropriate management of neonatal hypertension. Among available techniques, invasive intra-arterial blood pressure monitoring is widely regarded as the reference standard for accuracy and reliability. This method involves the placement of an indwelling arterial catheter, most commonly in the umbilical artery, although radial or posterior tibial arteries may also be used. The catheter is connected to a pressure transducer, which converts mechanical pressure into an electrical signal displayed on a multichannel patient monitor, allowing for continuous real-time blood pressure monitoring. Due to its invasive nature and associated risks, this approach is generally reserved for critically ill, hemodynamically unstable, or extremely premature neonates who require close cardiovascular monitoring.[4] The accuracy of intra-arterial blood pressure measurement depends heavily on meticulous technique and strict adherence to procedural standards. The pressure transducer must be maintained at the level of the heart to ensure that hydrostatic pressure does not artificially alter the recorded values. Any deviation from this position may result in falsely elevated or reduced readings. The integrity of the monitoring system is also critical. Air bubbles within the tubing must be carefully eliminated, as they can dampen the pressure waveform, leading to an underestimation of systolic pressure and an overestimation of diastolic pressure. A properly displayed arterial waveform, characterized by a clearly defined dicrotic notch, serves as an important indicator of accurate signal transmission and appropriate catheter placement [4].

The physical properties of the tubing further influence measurement accuracy. Low-compliance tubing of minimal acceptable length is recommended, as excessive length or elasticity can absorb pressure waves and produce falsely low readings. Proper calibration of the system is achieved by zeroing the pressure transducer to atmospheric pressure prior to use, ensuring that measured values reflect true intravascular pressure. Continuous infusion of a heparinized solution is required to maintain catheter patency and reduce the risk of thrombus formation, which may compromise both measurement accuracy and vascular integrity. Catheter size also plays a role in the reliability of invasive monitoring. Inappropriately narrow umbilical artery catheters may underestimate systolic pressure due to increased resistance within the lumen. For this reason, appropriate catheter selection is essential. Additionally, umbilical artery catheters should not remain in situ for prolonged periods. Removal is generally recommended after five to seven days, as extended catheterization increases the risk of thrombotic complications, infection, and vascular injury, all of which may affect blood pressure readings and overall neonatal outcomes.[4]

Noninvasive Methods

Noninvasive blood pressure measurement represents the most frequently employed approach for hemodynamic assessment in neonatal intensive care units, particularly in clinically stable infants or those not requiring continuous invasive monitoring. Among available noninvasive techniques, automated oscillometric devices are the predominant modality due to their ease of use, rapid application, and reduced risk of complications when compared with intra-arterial methods. These devices function by detecting oscillations in arterial blood flow generated during cuff inflation and deflation. The point of maximal oscillation is interpreted as the mean arterial pressure, from which systolic and diastolic values are mathematically derived using manufacturer-specific proprietary algorithms. In general, oscillometric blood pressure measurements demonstrate reasonable correlation with invasive intra-arterial readings, supporting their widespread clinical use. However, important limitations affect their accuracy and clinical interpretation in neonates. Automated oscillometry has been shown to overestimate systolic blood pressure values by approximately 3 to 8 mm Hg when compared with direct intra-arterial measurements, a discrepancy that may lead to overdiagnosis of hypertension.[5] Conversely, the method becomes unreliable when mean arterial pressure falls below 30 mm Hg, reducing its sensitivity for detecting hypotension in critically ill infants. Additional inaccuracies have been reported in small-for-gestational-age neonates, in whom systolic blood pressure may be underestimated due to altered vascular compliance and limb size. Variability in reported accuracy across studies is partly attributable to differences in the proprietary algorithms used by various manufacturers, as each device applies distinct computational methods to derive systolic and diastolic pressures. Several investigations have demonstrated clinically relevant discrepancies between devices, underscoring the importance of consistency in equipment selection within individual clinical settings.[5]

Proper technique is essential to optimize the reliability of oscillometric measurements. Cuff size selection plays a central role in measurement accuracy. The recommended cuff width-to-arm circumference ratio ranges from 0.44 to 0.55, with the inflatable bladder covering approximately 80% of the length of the upper arm.[6] Failure to adhere to these parameters, particularly the use of an undersized cuff, results in falsely elevated blood pressure readings. Standardization of cuff size for repeated measurements is therefore necessary to ensure consistency and comparability of results. Patient positioning and physiological state at the time of measurement further influence accuracy. The infant should be placed in a supine position and remain calm, quietly awake, or preferably asleep. Measurements should ideally be performed one to one and a half hours after feeding to minimize the effects of agitation or postprandial physiological fluctuations. To confirm reliability, at least three readings should be obtained at intervals of approximately two minutes, with the right upper limb serving as the preferred measurement site due to its proximity to central arterial circulation.[7] Alternative noninvasive techniques are generally discouraged in neonatal practice. Manual sphygmomanometry is not recommended because Korotkoff sounds are typically too faint to be detected reliably in neonates.[8] Ultrasound Doppler methods are also infrequently used for routine monitoring, as they tend to underestimate systolic blood pressure and offer limited advantage over oscillometric devices in this population.[9]

Etiology

Prematurity constitutes one of the most influential determinants in the development of neonatal hypertension and accounts for the majority of reported cases in contemporary neonatal practice. Approximately three quarters of neonates diagnosed with hypertension are born preterm, highlighting the close relationship between immaturity, systemic illness, and dysregulation of blood pressure control. Hypertensive preterm infants frequently demonstrate higher overall illness severity, elevated neonatal illness scores, and extended durations of hospitalization within the neonatal intensive care unit. Prolonged exposure to intensive medical interventions, coupled with incomplete maturation of renal, vascular, and endocrine regulatory systems, creates a physiological environment that predisposes these infants to persistent elevations in blood pressure. Several prematurity-associated conditions contribute directly to the onset of neonatal hypertension that necessitates pharmacological management. Respiratory disorders, particularly bronchopulmonary dysplasia, represent a leading contributor. The pathophysiology involves chronic hypoxia, altered pulmonary vascular resistance, and systemic inflammation, all of which exert secondary effects on systemic blood pressure regulation. Pharmacologic agents commonly administered to premature infants further compound this risk. Caffeine, widely used for apnea of prematurity, and dexamethasone, employed in the management of chronic lung disease, have both been implicated in blood pressure elevation through mechanisms involving sympathetic stimulation and mineralocorticoid activity. Advanced supportive therapies, including extracorporeal membrane oxygenation, have also been associated with hypertension, reflecting both the severity of underlying disease and the hemodynamic consequences of prolonged circulatory support [10].

Renal pathology remains one of the most significant contributors to neonatal hypertension in premature infants. Structural and functional renal immaturity predisposes this population to parenchymal and renovascular disorders. Acute tubular necrosis, renal failure, and ischemic injury related to hypotension or sepsis can disrupt sodium

handling and activate the renin–angiotensin–aldosterone system, leading to sustained blood pressure elevation. Thromboembolic events related to umbilical artery catheterization further contribute to renovascular compromise and are well-recognized etiologic factors in this setting.[10] These complications underscore the vulnerability of the neonatal renal system and its central role in blood pressure regulation. Beyond renal causes, several nonrenal conditions have been associated with neonatal hypertension in premature infants. Neurologic complications, including seizures and intracranial pathology, may provoke transient or sustained increases in blood pressure through autonomic dysregulation. Cardiovascular abnormalities also play a contributory role, most notably coarctation of the aorta, which can present during the neonatal period with systemic hypertension proximal to the site of obstruction. Less commonly, a patent ductus arteriosus has been linked to hypertensive states, particularly when associated with altered systemic blood flow patterns. Collectively, these factors illustrate the multifactorial nature of hypertension in premature neonates and the necessity of comprehensive evaluation.[10] Among nonrenal etiologies, bronchopulmonary dysplasia emerges as the most prominent independent risk factor for hypertension in very low birth weight infants. The association persists even after adjustment for gestational age and illness severity, suggesting a direct pathophysiological link rather than a mere marker of prematurity. Chronic lung disease alters systemic vascular resistance and neurohormonal signaling, contributing to sustained hypertension. Premature infants with intraventricular hemorrhage have also demonstrated an increased risk, potentially related to central autonomic instability and inflammatory cascades affecting vascular tone.[11][12] In contrast, hypertension in term infants is comparatively rare and often linked to isolated structural abnormalities. Case reports have documented elevated blood pressure in association with ductal aneurysms, though such occurrences remain uncommon.

Additional reported causes of neonatal hypertension are infrequent and often described in isolated or anecdotal contexts. Adrenal hemorrhage has been implicated through disruption of corticosteroid metabolism and stress hormone release. Vitamin D toxicity, particularly when accompanied by renal calcinosis, can impair renal function and calcium homeostasis, leading to secondary hypertension. Exposure to total parenteral nutrition has also been suggested as a contributing factor, potentially through electrolyte imbalances or vascular effects. While these associations are not widely prevalent, they remain clinically relevant in selected cases and warrant consideration during etiologic evaluation. Genetic factors represent a rare but increasingly recognized cause of neonatal hypertension. Advances in molecular diagnostics have identified specific mutations associated with early-onset hypertensive phenotypes. Biallelic loss-of-function mutations in the NPR1 gene have been linked to isolated neonatal-onset systemic hypertension, reflecting impaired natriuretic peptide signaling and dysregulated vascular tone. Despite expanding recognition of such genetic contributions, a substantial proportion of neonatal hypertension cases lack a clearly identifiable cause. Approximately 57% of affected infants are classified as having idiopathic hypertension, emphasizing the limitations of current diagnostic frameworks and the complexity of blood pressure regulation in early life (see Image. Neonatal Hypertension Etiologies).[13][14] Environmental exposure within the neonatal intensive care setting has also been implicated in the pathogenesis of neonatal hypertension. Phthalates, particularly di-2-ethylhexyl phthalate, are plasticizers commonly used in medical devices such as continuous positive airway pressure interfaces, endotracheal tubes, intravenous fluid bags, and associated tubing. Emerging evidence has suggested a potential association between phthalate exposure and the development of idiopathic neonatal hypertension, typically manifesting around 40 weeks postmenstrual age. This phenotype is characterized by low plasma renin activity and demonstrates a favorable therapeutic response to spironolactone, indicating a mineralocorticoid-mediated mechanism.[15]

The proposed pathophysiological mechanism involves inhibition of 11β-hydroxysteroid dehydrogenase 2, an enzyme responsible for inactivating cortisol at the mineralocorticoid receptor. Inhibition of this enzyme permits cortisol-mediated activation of the receptor, leading to sodium retention and hypertension. Observational data from a single research center reported a marked reduction in neonatal hypertension incidence following the discontinuation of DEHP-containing intravenous fluids, with rates declining from 7.7% to 1.4%. Notably, reintroduction of these products was associated with a subsequent increase in hypertension prevalence to 10.1%.[16] While these findings suggest a potentially modifiable environmental risk factor, independent confirmation is lacking, and further investigation is required to establish causality and generalizability. Taken together, the etiology of neonatal hypertension reflects a complex interplay between prematurity, organ immaturity, medical interventions, genetic susceptibility, and environmental exposures. This multifactorial foundation underscores the importance of individualized assessment and highlights the need for continued research to refine etiologic classification and guide targeted prevention and management strategies.

PMA	BP percentile	Systolic BP (mmHg)	Diastolic BP (mmHg)	MAP (mmHg)
42-44 weeks				
	50th	85-88	50-50	62-63
	95th	98-105	65-68	76-80
	99th	102-110	70-73	81-85
38-40 weeks				
	50th	77-80	50-50	59-60
	95th	92-95	65-65	74-75
	99th	97-100	70-70	79-80
34-36 weeks				
	50th	70-72	40-50	50-57
	95th	85-87	55-65	65-72
	99th	90-92	60-70	70-77
30-32 weeks				
	50th	65-68	40-40	48-49
	95th	80-83	55-55	63-64
	99th	85-88	60-60	68-69
26-28 weeks				
	50th	55-60	30-38	38-45
	95th	72-75	50-50	57-58
	99th	77-80	54-56	63-63

Fig. 1: Blood pressure in neonates.

Epidemiology

Neonatal hypertension is considered an uncommon but clinically significant condition, with reported incidence rates varying widely across different neonatal populations. Among term newborns, the incidence has been estimated at approximately 0.2%, indicating that sustained elevations in blood pressure are relatively rare in otherwise healthy infants.[3][17] In contrast, the prevalence increases substantially in infants requiring admission to neonatal intensive care units, where reported rates reach up to 3%. This marked disparity reflects the concentration of medically complex and premature infants within intensive care settings, as well as increased surveillance and more frequent blood pressure monitoring in this population. The observed variability in incidence across studies is influenced by several methodological factors. Differences in study design, sample size, gestational age distribution, and clinical severity of enrolled infants contribute to inconsistent estimates. Additionally, the absence of a universally accepted definition of neonatal hypertension has led to heterogeneity in diagnostic thresholds and measurement techniques. Variations in the timing of blood pressure assessment and the criteria used to confirm persistent hypertension further complicate comparisons across studies. As a result, reported incidence rates should be interpreted within the context of the specific population and diagnostic standards applied. Prematurity represents a major epidemiological determinant of neonatal hypertension. Among preterm infants, approximately 1.4% require pharmacological antihypertensive therapy during their neonatal intensive care unit stay, compared with around 1% of term infants who receive similar treatment.[11] Although the absolute difference appears modest, it underscores the increased vulnerability of preterm infants to blood pressure dysregulation, particularly in the presence of comorbid conditions and prolonged exposure to intensive medical interventions. The burden of neonatal hypertension is particularly pronounced among infants diagnosed with bronchopulmonary dysplasia. In this subgroup, reported incidence rates range from 13% to 43%, highlighting a substantially elevated risk compared with both term and preterm infants without chronic lung disease. This association reflects the interplay between chronic respiratory pathology, systemic inflammation, and altered vascular regulation. Despite these observations, the true prevalence of neonatal hypertension across the broader preterm population remains incompletely defined, emphasizing the need for large, standardized epidemiological studies to better characterize its scope and determinants [11].

Pathophysiology

The pathophysiology of neonatal hypertension is multifactorial, with renovascular mechanisms representing the most common pathway. Reduced renal perfusion is central to the development of hypertension in most affected neonates, triggering activation of the renin–angiotensin–aldosterone system (RAS). Umbilical artery catheterization is a well-recognized contributor to this process. Endothelial injury from catheter placement can result in thrombus formation within renal vessels, impairing renal blood flow and stimulating RAS-mediated vasoconstriction and sodium retention, ultimately leading to sustained elevations in systemic blood pressure.[18] Premature infants with bronchopulmonary dysplasia demonstrate additional pathophysiological alterations. These infants often exhibit impaired free water excretion and elevated serum aldosterone levels, both of which contribute to fluid retention and systemic hypertension. Recent studies have also identified structural and functional vascular changes in this population, including increased peripheral arterial wall thickness and abnormal vasomotor regulation, providing a mechanistic basis for the observed hypertensive states.[12][19] Genetic and maternal factors may further influence neonatal blood pressure regulation. Infants of diabetic mothers and those with prothrombotic conditions such as Factor V Leiden mutation are at risk for renal vein thrombosis, which reduces renal perfusion and activates RAS. Neonatal asphyxia may precipitate renal tubular necrosis, either as a cause or consequence of hypertension, by impairing tubular sodium handling and fluid balance.

Neonatal tumors, including pheochromocytoma, neuroblastoma, Wilms tumor, and mesoblastic nephroma, contribute to hypertension through mechanical compression of renal vessels or ureters, or via secretion of vasoactive substances such as catecholamines. Structural cardiac anomalies, including coarctation of the aorta and patent ductus arteriosus, decrease forward systemic blood flow, reduce renal perfusion, and provoke RAS activation. Surgical interventions, such as closure of abdominal wall defects, can elevate intra-abdominal pressure and compromise renal perfusion, further exacerbating hypertensive responses. Extracorporeal membrane oxygenation (ECMO) represents another unique mechanism. Hypertension during ECMO is thought to

arise from increased stroke volume caused by augmented aortic return from the extracorporeal circuit, compounded by abnormal sodium and water handling in response to nonpulsatile arterial flow. Collectively, these diverse mechanisms illustrate the interplay between renal perfusion, neurohormonal regulation, vascular structure, and systemic hemodynamics in the pathogenesis of neonatal hypertension, emphasizing the complexity of its clinical management [12].

History and Physical

Neonatal hypertension is frequently asymptomatic, and its detection often occurs incidentally during routine blood pressure monitoring in the neonatal intensive care unit. When clinical features are present, they are generally nonspecific and variable, reflecting the immature and compensatory physiology of neonates. Affected infants may exhibit feeding difficulties, weak or uncoordinated sucking, irritability, hypotonia or hypertonia, vomiting, apnea, respiratory distress, and intermittent oxygen desaturation. More severe manifestations include tachycardia, congestive heart failure, cardiogenic shock, or seizures, which necessitate urgent evaluation and intervention. In term or older neonates, hypertension may be discovered during routine well-child visits or through standard vital sign assessments in the NICU. Infants previously discharged may present with signs such as persistent irritability or failure to thrive, prompting reassessment of cardiovascular status.[20] A comprehensive physical examination is essential to guide the diagnostic workup and identify potential underlying causes. General inspection may reveal dysmorphic features indicative of genetic syndromes associated with aortic coarctation, including Turner, Williams, or Noonan syndromes. Cardiovascular assessment may uncover a cardiac murmur, discrepancies between upper and lower limb blood pressures, or diminished femoral pulses, all of which suggest structural cardiac anomalies. Signs of congestive heart failure, including tachycardia, cyanosis, pallor, and mottling, may also be observed. Abdominal examination is crucial for detecting renal masses or structural abnormalities, including polycystic kidney disease, renal tumors, hydronephrosis, or evidence of renal vein thrombosis. Genitourinary evaluation can identify congenital anomalies or ambiguous genitalia, particularly in cases of congenital adrenal hyperplasia. Prenatal histories of oligohydramnios may indicate congenital renal malformations, which in severe cases are associated with distinctive physical features. Collectively, careful history taking and meticulous physical examination allow clinicians to recognize subtle presentations of neonatal hypertension, guide further diagnostic evaluation, and identify potentially treatable underlying conditions [20].

Evaluation

The evaluation of neonatal hypertension begins with a detailed maternal and perinatal history, alongside a comprehensive physical examination, to identify potential underlying causes. Most cases of neonatal hypertension arise from renovascular or renal parenchymal disorders, making early assessment of the renal system essential. Initial investigations typically include basic laboratory studies such as urine analysis, blood urea nitrogen measurement, and serum levels of creatinine, electrolytes, and calcium. Urinary testing for vanillylmandelic acid and homovanillic acid can help detect catecholamine-secreting tumors or metabolic abnormalities. Imaging of the renal system is a critical component of early evaluation, and aortic and renal ultrasonography with Doppler assessment is recommended to identify structural anomalies, vascular obstruction, or altered renal perfusion.[10] Subsequent investigations are guided by clinical presentation and the level of suspicion for specific etiologies. Endocrine evaluation may be warranted, including thyroid function tests, serum cortisol, aldosterone, and plasma renin activity, particularly in cases where mineralocorticoid or glucocorticoid excess is suspected. Measurement of plasma and urine catecholamines and metanephrines is indicated when pheochromocytoma or neuroblastoma is considered. Additional steroid profiling, such as serum 11-deoxycortisol, 11-deoxycorticosterone, and urinary 17-hydroxysteroids and 17-ketosteroids, provides further insight into congenital adrenal disorders or enzyme deficiencies that may contribute to elevated blood pressure. Imaging studies complement laboratory assessments and provide structural and functional evaluation. Chest radiographs are useful for assessing associated cardiopulmonary conditions, including bronchopulmonary dysplasia or congestive heart failure. Echocardiography evaluates cardiac anatomy and function, including coarctation of the aorta, while voiding cystourethrograms can identify lower urinary tract obstruction. Renal scintigraphy using captopril or dimercaptosuccinic acid helps delineate functional renal impairment or renovascular hypertension. Advanced imaging, such as computed tomographic angiography or abdominal magnetic resonance imaging, may be indicated for detailed assessment of the renal arteries and aorta. Head ultrasonography is employed to rule out intraventricular hemorrhage in premature infants. By combining laboratory and imaging studies, clinicians can establish the underlying etiology, guide management, and anticipate potential complications associated with neonatal hypertension [10].

Treatment / Management

Management of neonatal hypertension primarily focuses on addressing underlying, correctable causes, which often leads to resolution without the need for long-term pharmacologic intervention. One of the most common contributing

factors is the presence of an umbilical artery catheter, and prompt removal of the catheter can significantly reduce hypertension in affected neonates. Metabolic abnormalities, such as hypercalcemia, or fluid overload can be corrected through careful fluid management or the administration of diuretics. Medications that may exacerbate hypertension, including inotropes, corticosteroids, and caffeine, should be reviewed, with dose adjustments or discontinuation implemented as clinically appropriate. In cases where surgical conditions contribute to elevated blood pressure, timely operative intervention is essential, and adequate analgesia may also help mitigate pain-induced increases in systemic pressure. Endocrine disorders require targeted hormonal therapy to restore physiologic balance and normalize blood pressure levels. Despite these interventions, some neonates may continue to exhibit systolic blood pressure values above the 99th percentile, necessitating the initiation of antihypertensive therapy. For mild neonatal hypertension, defined by systolic pressures between the 95th and 99th percentiles, careful observation is frequently sufficient, particularly in asymptomatic infants. Therapy is generally reserved for cases in which blood pressure remains persistently elevated or when echocardiographic evaluation demonstrates evidence of end-organ effects, such as left ventricular hypertrophy. This approach allows clinicians to avoid unnecessary pharmacologic intervention while ensuring that evolving hypertension is promptly identified and treated. Moderate neonatal hypertension is characterized by systolic pressures at or above the 99th percentile without overt end-organ damage. Although evidence-based guidelines are limited, clinicians often utilize pharmacologic agents such as calcium channel blockers, vasodilators, diuretics, or beta-blockers. Angiotensin-converting enzyme inhibitors are generally avoided in preterm infants younger than 40 to 42 weeks postmenstrual age due to their potential adverse effects on nephron development.[21] Therapy is typically administered orally unless clinical instability necessitates intravenous intervention.

Severe neonatal hypertension requires intensive management with continuous intravenous antihypertensive infusions. Rapid reductions in blood pressure are avoided to prevent ischemic complications affecting the brain and kidneys. Continuous intra-arterial monitoring provides accurate, real-time measurements and guides titration of intravenous medications. Recommended intravenous agents, along with precise dosing protocols, are used to achieve gradual and controlled blood pressure reduction while minimizing the risk of complications. Surgical intervention is indicated in neonates with structural or obstructive causes of hypertension. These conditions include coarctation of the aorta, renal artery or vein occlusion, ureteropelvic junction obstruction, polycystic kidney disease, neuroblastoma, or Wilms tumor.[22] Early identification and correction of these abnormalities are critical to achieving long-term blood pressure control, preventing secondary organ damage, and improving overall neonatal outcomes. Timely multidisciplinary evaluation, including neonatology, pediatric nephrology, and pediatric surgery, ensures that appropriate medical or surgical management is implemented efficiently. Overall, the management of neonatal hypertension requires an individualized, stepwise approach that integrates careful monitoring, targeted correction of underlying causes, judicious pharmacologic therapy, and surgical intervention when indicated. Successful outcomes depend on early recognition, precise evaluation of etiology, and close hemodynamic surveillance to prevent complications and optimize long-term cardiovascular health [22].

Differential Diagnosis

Neonatal hypertension represents a clinical manifestation rather than a primary disease, and it may arise from disorders affecting multiple organ systems. Because the presenting symptoms are often nonspecific, a thorough differential diagnosis is essential to guide appropriate evaluation and management. Common presenting signs include respiratory distress, hypotonia, irritability, feeding intolerance, tachycardia, and, in more severe cases, seizures or signs of congestive heart failure. These features are not unique to hypertension and may overlap with other neonatal conditions, including sepsis, metabolic disturbances, congenital heart disease, and neurologic disorders. Distinguishing between these entities requires careful integration of maternal and perinatal history, physical examination, laboratory studies, and imaging. Renal etiologies constitute the most frequent causes of neonatal hypertension. Renal parenchymal diseases, congenital anomalies of the kidney and urinary tract, renal vein thrombosis, and obstructive uropathy should be considered in infants with elevated blood pressure. Renovascular abnormalities, including stenosis or thromboembolic events, can precipitate significant hypertension via activation of the renin–angiotensin–aldosterone system. Cardiac structural abnormalities, such as coarctation of the aorta or patent ductus arteriosus, must also be evaluated, particularly if there is asymmetry in upper and lower limb blood pressures or diminished femoral pulses. Endocrine disorders, including congenital adrenal hyperplasia or hyperaldosteronism, represent additional contributors that may present with nonspecific systemic symptoms. Rare genetic syndromes, environmental exposures such as phthalates, and medication-related effects must also be considered. Comprehensive evaluation ensures that reversible or treatable causes are promptly identified while avoiding misdiagnosis or unnecessary intervention [22].

Prognosis

The prognosis of neonatal hypertension depends on both the underlying etiology and the

severity at presentation. Transient causes, such as renal vein thrombosis, umbilical catheterization, or acute tubular necrosis, generally carry a favorable prognosis, with resolution expected following correction of the primary insult. The presence of end-organ involvement, including left ventricular hypertrophy, renal impairment, or neurologic sequelae, is associated with a poorer long-term outlook. Most infants require pharmacologic therapy only for a limited duration, with chronic therapy being relatively uncommon. A retrospective study by Xiao et al examined infants with idiopathic neonatal hypertension discharged from the NICU on antihypertensive therapy. Calcium channel blockers were the most frequently prescribed agents, representing 56% of all prescriptions. Following discharge, 60% of infants continued antihypertensive therapy, with 26% remaining on medication at one year and only 7% requiring therapy at two years. These findings indicate that the majority of neonates with idiopathic hypertension will discontinue therapy within two years post-discharge.[23] Long-term data on the progression of neonatal hypertension into late childhood and adulthood remain limited, emphasizing the importance of ongoing monitoring. Regular assessments of blood pressure and renal function are recommended, and care should be overseen by an interprofessional team to ensure appropriate management and early detection of potential complications [23].

Complications

Untreated or inadequately controlled neonatal hypertension can lead to significant morbidity through end-organ damage. Sustained elevated blood pressure increases the risk of vascular injury, left ventricular hypertrophy, hypertensive encephalopathy, and retinopathy. Early recognition and aggressive management are critical in preventing such complications. Chronic or severe hypertension may result in hypertensive nephropathy, characterized by variable degrees of renal dysfunction, as well as hypertensive cardiomyopathy, which can manifest as ventricular hypertrophy, dilation, or impaired cardiac function. The long-term sequelae of untreated hypertension compared with treated cases are not fully understood, though epidemiological studies in older populations indicate that childhood hypertension is associated with an increased risk of developing hypertension in adulthood. Therefore, prompt identification and careful blood pressure control are essential to mitigate the potential for irreversible organ damage [23].

Patient Education

Neonatal hypertension is frequently underrecognized, particularly in infants admitted to standard nursery settings. Early detection and timely intervention are crucial to prevent end-organ damage and reduce long-term cardiovascular risk. Pharmacologic management presents unique challenges due to the limited safety and efficacy data for antihypertensive agents in neonates. The absence of standardized normative blood pressure references and a universally accepted definition of neonatal hypertension complicates clinical decision-making. No established guidelines specify the optimal timing for initiating therapy or the management of mild hypertension in neonates. Many infants improve spontaneously or require only short-term pharmacologic therapy. Consequently, clinical judgment is paramount, considering the infant's overall condition, presence of end-organ effects, and individual risk factors. Patient and family education is equally important, as caregivers must recognize the need for follow-up, monitoring, and adherence to treatment plans. Careful, individualized management helps balance the benefits of intervention with the risks of unnecessary medication exposure in this vulnerable population [21][22][23].

Enhancing Healthcare Team Outcomes

Effective management of neonatal hypertension requires an interprofessional, coordinated approach. Neonatologists and pediatric nephrologists are responsible for accurate diagnosis, interpreting age-specific blood pressure percentiles, and guiding both invasive and noninvasive monitoring. Advanced practitioners contribute through comprehensive assessment, early recognition of hypertension, and initiation of appropriate evaluations, while ensuring meticulous documentation and monitoring. Nurses play a central role in bedside management, including precise blood pressure measurement, observation for clinical signs of hypertension, and identification of adverse medication effects. Pharmacists provide guidance on safe and effective pharmacologic therapy, accounting for neonatal pharmacokinetics, renal function, and comorbid conditions. Structured communication and care coordination are essential for optimizing outcomes. Regular interprofessional rounds facilitate collaborative decision-making, timely identification of risk factors, and clear delineation of responsibilities. Teams coordinate diagnostic evaluations, integrate maternal and perinatal history, and ensure smooth transitions from inpatient to outpatient care. Family education, supported by nurses and social workers, reinforces understanding, compliance, and engagement in follow-up care. Open communication, standardized protocols, and shared decision-making reduce diagnostic delays, prevent inappropriate interventions, and enhance both short- and long-term outcomes for neonates with hypertension [23].

Conclusion:

Neonatal hypertension represents a significant yet often underrecognized challenge in neonatal care. Its multifactorial origins—including prematurity, renal abnormalities, cardiovascular defects, endocrine disturbances, and environmental exposures—necessitate a comprehensive and

individualized approach to diagnosis and management. Early detection remains crucial, as many affected infants are asymptomatic or present with nonspecific signs that can easily be overlooked. Accurate blood pressure measurement, careful clinical assessment, and targeted investigations allow clinicians to identify reversible causes and initiate timely interventions. Most cases resolve with correction of underlying factors, while persistent or severe hypertension requires careful titration of pharmacologic therapy to avoid complications such as left ventricular hypertrophy, renal impairment, and neurologic injury. Because evidence-based guidelines remain limited, clinical judgment and interprofessional collaboration are essential. Nurses play a central role in monitoring, recognizing early changes, and educating families about follow-up and medication adherence. Pharmacists ensure appropriate drug selection and dosing, while pediatric specialists guide diagnostic and therapeutic decision-making. Ultimately, effective management depends on coordinated teamwork, standardized protocols, and continuous evaluation of each infant's evolving condition. With vigilant monitoring and individualized care, most neonates achieve favorable outcomes, emphasizing the critical importance of structured, multidisciplinary management strategies in the NICU.

References:

1. Adelman RD. Neonatal hypertension. Pediatr Clin North Am. 1978 Feb;25(1):99-110.
2. Seliem WA, Falk MC, Shadbolt B, Kent AL. Antenatal and postnatal risk factors for neonatal hypertension and infant follow-up. Pediatr Nephrol. 2007 Dec;22(12):2081-7.
3. Dionne JM, Abitbol CL, Flynn JT. Hypertension in infancy: diagnosis, management and outcome. Pediatr Nephrol. 2012 Jan;27(1):17-32.
4. Flynn JT. Hypertension in the neonatal period. Curr Opin Pediatr. 2012 Apr;24(2):197-204.
5. O'Shea J, Dempsey EM. A comparison of blood pressure measurements in newborns. Am J Perinatol. 2009 Feb;26(2):113-6.
6. Dionne JM, Bremner SA, Baygani SK, Batton B, Ergenekon E, Bhatt-Mehta V, Dempsey E, Kluckow M, Pesco Koplowitz L, Apele-Freimane D, Iwami H, Klein A, Turner M, Rabe H., International Neonatal Consortium. Method of Blood Pressure Measurement in Neonates and Infants: A Systematic Review and Analysis. J Pediatr. 2020 Jun;221:23-31.e5.
7. Flynn JT, Kaelber DC, Baker-Smith CM, Blowey D, Carroll AE, Daniels SR, de Ferranti SD, Dionne JM, Falkner B, Flinn SK, Gidding SS, Goodwin C, Leu MG, Powers ME, Rea C, Samuels J, Simasek M, Thaker VV, Urbina EM., SUBCOMMITTEE ON SCREENING AND MANAGEMENT OF HIGH BLOOD PRESSURE IN CHILDREN. Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children and Adolescents. Pediatrics. 2017 Sep;140(3)
8. de Swiet M, Dillon MJ, Littler W, O'Brien E, Padfield PL, Petrie JC. Measurement of blood pressure in children. Recommendations of a working party of the British Hypertension Society. BMJ. 1989 Aug 19;299(6697):497.
9. Nascimento MC, Xavier CC, Goulart EM. Arterial blood pressure of term newborns during the first week of life. Braz J Med Biol Res. 2002 Aug;35(8):905-11.
10. Sharma D, Farahbakhsh N, Shastri S, Sharma P. Neonatal hypertension. J Matern Fetal Neonatal Med. 2017 Mar;30(5):540-550.
11. Sahu R, Pannu H, Yu R, Shete S, Bricker JT, Gupta-Malhotra M. Systemic hypertension requiring treatment in the neonatal intensive care unit. J Pediatr. 2013 Jul;163(1):84-8.
12. Alagappan A, Malloy MH. Systemic hypertension in very low-birth weight infants with bronchopulmonary dysplasia: incidence and risk factors. Am J Perinatol. 1998 Jan;15(1):3-8.
13. Sharma D, Pandita A, Shastri S. Neonatal hypertension: an underdiagnosed condition, a review article. Curr Hypertens Rev. 2014;10(4):205-12.
14. Giri P, Roth P. Neonatal Hypertension. Pediatr Rev. 2020 Jun;41(6):307-311.
15. Jenkins RD. Phthalates cause a low-renin phenotype commonly found in premature infants with idiopathic neonatal hypertension. Pediatr Nephrol. 2023 Jun;38(6):1717-1724.
16. Jenkins R, Farnbach K, Iragorri S. Elimination of Intravenous Di-2-Ethylhexyl Phthalate Exposure Abrogates Most Neonatal Hypertension in Premature Infants with Bronchopulmonary Dysplasia. Toxics. 2021 Apr 02;9(4)
17. AlMaazmi A, Hagan J, Fernandes CJ, Gowda SH. Neonatal systemic hypertension across the PHIS database: An update. Int J Cardiol. 2023 Apr 01;376:49-53.
18. Kilian K. Hypertension in neonates causes and treatments. J Perinat Neonatal Nurs. 2003 Jan-Mar;17(1):65-74; quiz 75-6.
19. Sehgal A, Malikiwi A, Paul E, Tan K, Menahem S. Systemic arterial stiffness in infants with bronchopulmonary dysplasia: potential cause of systemic hypertension. J Perinatol. 2016 Jul;36(7):564-9.
20. Skalina ME, Kliegman RM, Fanaroff AA. Epidemiology and management of severe symptomatic neonatal hypertension. Am J Perinatol. 1986 Jul;3(3):235-9.
21. Gantenbein MH, Bauersfeld U, Baenziger O, Frey B, Neuhaus T, Sennhauser F, Bernet V. Side effects of angiotensin converting enzyme inhibitor (captopril) in newborns and young infants. J Perinat Med. 2008;36(5):448-52.

22. Stanley JC, Zelenock GB, Messina LM, Wakefield TW. Pediatric renovascular hypertension: a thirty-year experience of operative treatment. J Vasc Surg. 1995 Feb;21(2):212-26; discussion 226-7.
23. Xiao N, Starr M, Stolfi A, Hamdani G, Hashmat S, Kiessling SG, Sethna C, Kallash M, Matloff R, Woroniecki R, Sanderson K, Yamaguchi I, Cha SD, Semanik MG, Chanchlani R, Flynn JT, Mitsnefes M. Blood Pressure Outcomes in NICU-Admitted Infants with Neonatal Hypertension: A Pediatric Nephrology Research Consortium Study. J Pediatr. 2024 Jan;264:113765.

.