Hypertension in the Pediatric Intensive Care Unit

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Abstract

Keywords

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- pediatric intensive care unit
- hypertensive emergency

Hypertension in the pediatric intensive care unit (PICU) is common and it contributes to the overall morbidity and mortality. Patients may present with hypertensive emergencies or hypertension can manifest itself later in PICU course. Although hypertension can be seen in most patients during hospitalization, patients with some specific diseases and conditions are more prone to hypertension. Hypertension should be recognized promptly and treated accordingly. Different pathophysiologic mechanisms can be responsible for the hypertension and management differs based on the underlying etiology. Any patient with a hypertensive emergency must be admitted to PICU, and treatment and diagnostic workup should be initiated immediately.

Introduction

There is paucity of information about hypertension in pediatric intensive care settings. Ehrmann et al found a hypertension prevalence of 25% in their retrospective cohort.¹ In the study preferred hypertension definition based on high sensitivity was having systolic blood pressure and/or diastolic blood pressure greater than 99% + 5 mm Hg in three readings for a day. In bivariate analyses, children with hypertension were younger and had a higher proportion of mechanical ventilation, PRISM-III (pediatric risk of mortality III) scores, use of anti-infectives, and diuretics. Hypertension was independently associated with nearly threefold increased odds of AKI and 1.5-day increase in length of stay in multivariable analyses. Mortality was rare (< 1%) but was associated with hypertension. Normal blood pressure (BP) values were defined by the guidelines of the National High Blood Pressure Education Program Working Group (NHBEP) in 1987 and its updates in 1996 and 2004.^{2,3} BP values are based on sex, age, and height for 50th, 90th, 95th, and 99th percentiles. Hypertension is defined as average systolic BP and/or diastolic BPs that are 95th percentile or greater for sex, age, and height on three or more separate occasions. Condition of BP levels that are 90th percentile or greater or 120/80 mm Hg or greater but

less than 95th percentile is termed *prehypertension*. Prevalence of hypertension is between 2 and 4% in general pediatric population.^{4–6} Also, it should be kept in mind that detection of hypertension is hard in pediatric population because of normal ranges change in sex, age, and height. A study showed that of 507 children and adolescents (3.6%) who had hypertension, only 131 (26%) had a diagnosis of hypertension or elevated BP documented in the electronic medical record.⁷

BP measurements should be done accurately. Any abnormal result should be repeated. Although oscillometric measurements are commonly used for BP measurements in both inpatient and outpatient settings, NHBEP recommends clinical sphygmomanometric measurements by using stethoscope placed over the brachial artery pulse proximal and medial of the cubital fossa. Cuff appropriate to the child's upper right arm should be used to avoid false measurements. Typically oscillometric measurements are 5 to 10 mm Hg higher compared with auscultation.⁸ Also, patients in intensive care units (ICUs) usually have arterial catheters. Holt et al compared direct arterial, automated oscillometric, and sphygmomanometer with gold standard Doppler ultrasound measurements in pediatric intensive care patients. There was no clinically significant difference between methods in the normotensive group. In the hypertensive group, the systolic

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arterial BP values were higher and indirect BP values using the automated oscillometric device readings were lower than gold standard Doppler ultrasound.⁹

Etiology

The etiology of hypertension in intensive care population is broad. According to the 4th report of NHBEP, primary hypertension in childhood is usually characterized by mild or stage 1 hypertension and is often associated with a positive family history of hypertension or cardiovascular disease.² With the obesity, epidemic incidence of primary hypertension is increasing in the pediatric population, but secondary hypertension still remains to be more common in children than adults. Children with known hypertension are admitted to the ICUs due to hypertensive emergencies, seizures, cerebrovascular accidents, and congestive heart failure. Pain and agitation are significant causes of elevated BPs in the intensive care settings. In addition, hypertension can be a side effect of medications used such as albuterol and steroids. Use of vasopressors in septic patients can result in inadvertent high BPs, and deliberate induction of hypertension can be a desired goal in patients with traumatic brain injury while targeting specific BPs to maintain cerebral perfusion pressures (CPPs).

Neonatal ICU patients after discharge are a specific population that deserves special attention. They are usually admitted to the pediatric intensive care units (PICU) frequently in first couple years of life. Many epidemiologic studies have found an inverse association between low birth weight and hypertension.^{10–16} This is most likely related to the congenital deficit in nephron number, resulting in a decreased filtration surface area and glomerular filtration rate.¹¹ Congenital malformations like renal artery stenosis, renal artery hypoplasia, abdominal aortic atresia, coarctation of the aorta, kidney cystic disease, and reflux nephropathies can cause hypertension in the neonates and infants.¹⁷ Umbilical catheterizations can cause renal artery stenosis and lead to hypertension.¹⁸

Intoxications with psychoactive substances can cause life-threatening hypertensive emergencies and arrhythmias. Hypertensive emergencies are commonly reported with cocaine and amphetamines. Designer drugs like methylenedioxypyrrovalerone (MDPV) also known as “bath salts” has gained special attention in the literature due to increasing abuse in the adolescent population.^{19,20} Accidental ingestion of medications should be suspected in any toddler and young child presenting with altered mental status and hypertension. Physicians have to get a detailed history and ask about any medications and substances at home. A detailed history about medications used by adults at home is very helpful. Parents should be recommended to bring the bottles of any medication at home and missing pills should be counted.

Diagnostic Evaluation

Most hospitalized children with identified high BP have a secondary form of hypertension. The diagnostic evaluation of a hospitalized hypertensive child should be undertaken

concurrent with lowering BP, especially in the setting of hypertensive urgency and emergencies. After ruling out influence of pain and anxiety as the cause of hypertension, the evaluation should focus on the assumption of a secondary cause until proven otherwise. This evaluation for secondary hypertension can be done in a stepwise fashion and individualized based on findings of the history, physical examination, special tests, and child's clinical condition aside from pain and anxiety. The most common cause of secondary hypertension in hospitalized children is renal parenchymal disease.^{21,22} **Fig. 1** provides an algorithm for the diagnostic evaluation of hypertension in the hospitalized child.

In the absence of an obvious precipitating factor contributing to the child's hypertension, a comprehensive history is crucial such as prenatal history of prematurity, placement of umbilical catheters, acute renal injury (AKI), repeated upper urinary tract infections during first few years of life, history of glomerulonephritis as evidenced by fever, preceding sore throat, joint pain and hematuria, family history of renal disease, cardiovascular disease is helpful in determining the cause of hypertension. Acute or chronic use of pain medications such as nonsteroidal anti-inflammatory drug (NSAID), oral contraceptives, corticosteroids, stimulants used for attention deficit disorder, decongestants, calcineurin inhibitors (CNIs), and recreational drugs can also cause hypertension in children.

History of chronic or acute headaches, chest pain, or changes mental status changes are associated with chronic or acute hypertension. Migraine headaches and sleep disturbances are often associated with chronic hypertension that can improve with normalization of BP.²³ Sweating, palpitations, flushing, and abdominal cramps are associated with state of catecholamine excess as in pheochromocytoma.

The physical examination should be complete especially focused on evaluating end-organ damage, and should include examination of the cardiorespiratory system, genitourinary system, abdomen, neurologic changes, retina, thyroid, skin, and four limb pulse and pressures.²⁴

Toxicology screening of serum, sodium, potassium, chloride, phosphorus, blood urea nitrogen (BUN), creatinine, magnesium, glucose, carbon dioxide, liver profile, plasma serum, renin, aldosterone, and, when indicated, urine catecholamines should be done. Other laboratory investigation to be considered includes serologic profile for vasculitis, systemic lupus, complement levels, and lipid profile. Urine analysis may reveal hematuria, proteinuria, pyuria, casts and bacteruria, aiding in the diagnosis of renal parenchymal disease, renal vascular hypertension, and infection. Presence of electrolyte abnormality is helpful to diagnose renal dysfunction.

Renal ultrasound with Doppler of the renal arteries is a useful screening test to rule out hydronephrosis, renal dysplastic and cystic disease, renal blood flow velocities, and renal parenchymal disease. The size of the kidney could help establish chronicity of kidney disease. Computed tomographic (CT) scan of the head in patients with neurologic symptoms is useful to rule out acute bleeding or other changes in the brain. After stabilization of the patient objective evidence of

Fig. 1 Proposed algorithm for the management of hypertensive crisis in children. CT, computed tomography; DMSA, dimercaptosuccinic acid; ECG, electrocardiogram; ECHO, echocardiogram; MAG3, mercaptoacetyl triglycine; PICU, pediatric intensive care unit. (Modified from Chandar and Zilleruelo.²⁴)

end-organ damage should be sought and should include an echocardiogram, ophthalmologic examination, and renal scan (dimercaptosuccinic acid [DMSA], mercaptoacetyl triglycine [MAG3] renal scan with furosemide) (–Fig. 1).

Special Causes of Hypertension

We will include special diseases and conditions that are relatively unique to PICU population. We will briefly talk about the some of the causes, pathogenesis, and management of hypertensive crisis.

Aortic Coarctation

Aortic coarctation is caused by narrowing of the aortic arch, which may be related to discrete narrowing or more diffuse hypoplasia of the arch. It is a relatively common defect accounting for 5 to 8% of all congenital heart diseases with an estimated incidence of 1 in 2,500 newborns.^{25,26} Presentation is either in newborn period when the ductus arteriosus starts to close due to critical coarctation or later in life related to the degree of narrowing. Those infants with critical coarctation need early surgical repair, and early mortality is less than 1% currently in most centers with the improvements in cardiothoracic surgery and postoperative care in the last three decades. The most common complication in the early postoperative period is hypertension. Morphogenesis of aortic coarctation shows progressive accumulation of fibroelastic tissue in the extracellular space. Sehested et al examined

resected aortic tissues and observed reduced tension induced by potassium, noradrenaline, and prostaglandin in the pre-stenotic versus poststenotic aorta.^{27,28} It is postulated that with aortic stiffness and abnormal vessel dynamics, baroreceptors may have a higher threshold for activation. Kenny et al measured noninvasive indices of autonomic balance, large artery stiffness, and cardiac output in an 8-month-old male child with native coarctation and his monozygotic twin.²⁹ They found that spontaneous baroreceptor reflex sensitivity was markedly lower in the infant with coarctation suggesting dampening of the baroreceptor reflex. Dysregulation of renin angiotensin system and endothelial dysfunction may be the other mechanisms contributing to the hypertension.^{30–32} Despite early repair, hypertension remains to persist in most patients. In the literature median prevalence of hypertension in patients who have undergone aortic coarctation repair is 32.5% with a range of 25 to 28%.³³ As previously mentioned, aortic stiffness and altered shape and elasticity of aortic arch, endothelial dysfunction with abnormal nitric oxide synthesis and impaired baroreceptor function play a role in hypertension later in life.

Early hypertension after surgery may be related to inability of baroreceptors to adjust to the new hemodynamics and elevated distal BPs.^{27,28,34} After resetting of the baroreceptors, BPs normalize, but in the initial period patients need aggressive BP management to avoid exposure of fresh sutures to high systemic pressures. Hypertension, usually referred as

paradoxical hypertension, is managed by intravenous agents started in the operating room. Combined α - and β -blockers or rapid-acting IV β -blockers like esmolol have been used successfully in the immediate management of postoperative hypertension.^{35–37} Nitroprusside is also commonly used for management of hypertension but may be inferior to IV esmolol in control of BPs.³⁷ Postoperative hypertension usually resolves in a week, but recurrence of hypertension is very common as patient gets older. Treatment of the hypertension in the long-term period is beyond the scope of this review.

Hypertension Associated with Skeletal Traction

Hypertension is a frequent complication after surgery. In a study, 45% of surgical patients had a new discharge diagnosis of hypertension after surgery. Of those patients, 18% were sent home on an antihypertensive agent.³⁸ Hypertension after tractional orthopedic surgeries is also very common. Heij et al have found an arterial hypertension incidence of 62% in children treated with skeletal traction for forearm and leg fractures and orthopedic diseases.³⁹ Different etiologies for postoperative hypertension in orthopedic patients have been suggested in the literature. Pain and anxiety should be considered in the etiology of hypertension in all surgical patients and treated appropriately. Increased sympathetic activity and traction on pelvic soft tissues can be responsible of increased cardiac output as well as peripheral vascular resistance.^{40,41} Although hypercalcemia is thought to be a cause of postoperative hypertension previously, Heij et al did not find any difference in the occurrence of hypertension in normocalcemic and hypercalcemic children.^{39,42–44} Pain control is an important adjunct therapy in addition to antihypertensive medications.

Hypertension after Kidney Transplant

Hypertension is a common and dreaded complication after renal transplant. It can be seen in up to 90% of renal transplant recipients.^{45,46} Incidence of early posttransplant hypertension is around 80%.^{47,48} Hypertension is associated with cardiovascular diseases and increased graft loss and mortality. It can be early and late onset with possible different pathophysiologic mechanisms being responsible. Because of lack of autoregulation of transplanted kidney in the initial postoperative period up to 48 hours, high BPs are required to decrease the risk of allograft damage.⁴⁹ In pediatric patients following kidney transplant, adult BPs level is desired to maintain the perfusion of the graft. However, one has to avoid high BPs that can cause injury to the central nervous system (CNS). Usually lower BP goals are more applicable to the posttransplant patients at week 4 and beyond.^{50,51}

Immunosuppressive therapies like steroids and cyclosporine, allograft failure, allograft renal stenosis, native kidneys (by producing excess renin), recurrent essential hypertension, and volume overload are important etiologies of hypertension after renal transplant.^{50,52} CNIs are routinely used after solid-organ and bone marrow transplants. They decrease the incidence of acute and chronic rejection, but rates of posttransplant hypertension almost doubled after the CNI era.⁵³ The mechanisms of CNI-induced rise in BP are due to systemic vasoconstriction and its effects on the renal allograft by causing afferent vasoconstriction and reduction in glomerular filtration rate (GFR) and increase in tubular sodium reabsorption. Systemic vasoconstriction is caused by imbalance between vasoconstrictive and vasodilators, increase in vasoconstrictive substances (endothelin-1 and thromboxane A2), decrease in vasodilator compounds like prostacyclin and nitric oxide, increase in intrarenal renin activity,

Fig. 2 Mechanism of hypertensive crisis. (Modified from Dinish Sing et al.⁷³)

accentuation of angiotensin II action, and increase in sympathetic activity (►Fig. 2).⁵⁰

Transplant renal artery stenosis (TRAS) is a significant complication of renal transplantation that can lead to uncontrolled hypertension and graft loss.⁵⁴ Hurst et al found an incidence rate of 1.9% according to the US Renal Data System Registry.⁵⁵ TRAS-associated hypertension may not respond to medical management and may require percutaneous intervention or surgery. Interestingly, Henning et al showed that after successful angioplasty the mean dose of antihypertensive therapy did not change.⁵⁶ We therefore speculate that TRAS might not be responsible for hypertension in some cases but can be a predictor of ongoing renal damage and nephron loss due to chronic rejection and other causes.

Hypertension after Head Trauma and Surgery

Hypertension after neurologic injuries can be frequently seen. Excessive fluid resuscitation, pain, and anxiety are common causes of hypertension in trauma patients. In severe neurologic injuries paroxysmal sympathetic overactivity, most often referred as *storming*, can be responsible of hypertension as well as tachycardia, agitation, sweating, and posturing. It is thought to be due to hypothalamic disturbances and loss of inhibition of cortical and spinal centers on the catecholaminergic system.^{57–59} β -Blockers are used in treatment of sympathetic storming.

Acute kidney injury (AKI) may contribute to the hypertension seen in patients with neurotrauma. According to a study, renal failure incidence was 1.5% in a prospective cohort in patients with severe head injury.⁶⁰ AKI is a significant risk factor for in-hospital mortality in patients with critical illness.⁶¹ It can be related to the nature of the trauma, posttraumatic hypotension and acute tubular necrosis, rhabdomyolysis, and vasoconstriction due to sympathetic dysautonomia. Fang et al found that cumulative doses of mannitol is a risk factor for developing AKI.⁶²

It should be kept in mind that arterial hypertension may be a requirement to maintain CPPs (mean arterial pressure – intracranial pressure) in patients with severe head injury. Recommended CPPs for adults target a range between 50 and 70 mm Hg.⁶³ There are no strict target CPP goals recommended by the guidelines for children, but in most institutions CPP less than 50 mm Hg is avoided.⁶⁴ Patients are supported with vasopressors and fluids to sustain desired CPP goals.

Management of Hypertension in the Pediatric Intensive Care Unit

An intravenous drip is the preferred method to handle hypertension crises, as the drug dosing can be titrated. Based on previous observations and experience, the initial drop in BP should not be greater than 25 to 30% of the original value accomplished over 6 to 8 hours and followed by gradual reduction of more than 24 to 48 hours.²

The choice of antihypertensive medication depends on the etiology of hypertension. Hypertensive emergencies should be treated with an agent that has rapid onset of action and a short half-life.

►Fig. 1 provides an algorithm for the treatment of hypertensive crisis. Intravenous nicardipine is a dihydropyridine calcium channel blocker that reduces peripheral vascular resistance. It does not have negative inotropic effect and can be used in the presence of bronchospasm in patients with hepatic and renal failure. It has rapid onset of action and half-life is 10 to 15 minutes. It is well tolerated and efficacious. Tachycardia and flushing are side effects of therapy, but nicardipine can be used for a longer period of time and should be considered as the first line of therapy in children with hypertension. Short-acting sublingual nifedipine is not recommended because of erratic absorption and is associated with high incidence of adverse events. Labetalol is an α - and β -blocker that reduces peripheral vascular resistance. It has a long half-life (3–5 hours), and this should be considered while titrating the dose of the drug. It should not be used in patients with tendency for bronchospasm or congestive heart failure as it has a negative inotropic effect. Labetalol has potential to worsen hyperkalemia and should be used with caution in children with renal failure. It can be given as a bolus, which can be advantageous when an infusion cannot be started quickly.²⁴

Esmolol is used in hypertensive crisis and is particularly useful in patients with tachycardia. Esmolol is a cardioselective β -blocker that is rapidly acting and has a half-life of 10 minutes. It is particularly useful after repair of congenital heart disease in children. Some of the side effects are congestive heart failure and broncho spasm. It is contraindicated in hypertensive crisis caused by catecholamine excess as hypertension is perpetuated by persistent α stimulation.²⁴

Fenoldopam is a rapidly acting vasodilator and is a selective dopamine receptor agonist, and binds with moderate affinity to α adrenoreceptors. It can be used in renal insufficiency.⁶⁵ Fenoldopam is mostly used in children after cardiopulmonary bypass surgery for congenital cardiac disease. In neonates it can promote diuresis after fluid load following cardiac surgery.⁶⁶ It promotes renal perfusion. Its toxicities are reflex tachycardia and tachyphylaxis.

IV hydralazine has been used in neonates, children, and pregnant teenagers to control severe hypertension. In very sick and low-birth-weight neonates for whom enteral administration of medications is not possible, IV hydralazine is a good therapeutic option. It is a potent vasodilator with an onset of action within 10 minutes and its effect lasts for 2 to 4 hours. It can be administered intramuscularly when immediate IV access is not possible. The side effects of hydralazine are tachycardia and vasodilation.^{24,67}

Sodium nitroprusside is a powerful arteriolar and venous vasodilator. Its rapid onset of action with short half-life has made it a first-line option to treat hypertensive crisis and emergency. Nitroprusside is a potent vasodilator. Its use is limited, however, by its toxicity of metabolites nitroprusside if converted to cyanide by tissue and this cyanide is converted to thiocyanate in the liver. Patients with liver disease and reduced renal function are prone to develop the cyanide toxicity. Patients on nitroprusside for more than 72 hours and renal insufficiency and a GFR of less than 60 mL/min/1.73^{m2} should be monitored

Table 1 Review of drugs used in pediatric hypertensive patients

	Mechanism of action	Dose	Onset of action	Considerations/side effects
Nicardipine	Calcium channel blocker	0.5–1 µg/kg/min Max dose: 4–5 µg/kg/min	Within minutes	<ul style="list-style-type: none"> Chest pain, ECG abnormalities, abdominal pain, headache, and dizziness
Labetalol	Combined αβ blocker	0.25–3 mg/kg/h	2–5 min	<ul style="list-style-type: none"> Caution with hyperreactive airway disease and asthma Caution in patients with diabetes mellitus; may mask symptoms of hypoglycemia May depress myocardial activity and can worsen congestive heart failure Can cause ECG abnormalities
Esmolol	β-Blocker	50–250 µg/kg/min (500 µg/kg/min loading dose recommended)	2–10 min	<ul style="list-style-type: none"> Caution with hyperreactive airway disease and asthma Caution in patients with diabetes mellitus; may mask symptoms of hypoglycemia May depress myocardial activity and can worsen congestive heart failure Caution in patients with renal impairment; active metabolite retained
Fenoldopam	Postsynaptic dopamine agonist	0.2 µg/kg/min Max dose: 0.8 µg/kg/min	5 min	<ul style="list-style-type: none"> Caution in patients with glaucoma and intraocular hypertension Caution in patients with increased intracranial pressure; not studied in this population Tachycardia
Hydralazine	Direct vasodilation of arterioles (little effect on veins)	0.1/0.2 mg/kg/dose (IV) every 4–6 h as needed	5–20 min	<ul style="list-style-type: none"> Discontinue if patients develop SLE-like syndrome or positive ANA
Nitroprusside	Direct arterial and venous vasodilator	0.3–0.5 µg/kg/min Max dose: 8–10 µg/kg/min	Within 2 min	<ul style="list-style-type: none"> May cause precipitous hypotension Cyanide and thiocyanate toxicity may occur
Enalaprilat	ACE inhibitor	0.08 mg/kg/d (enteral) Max dose: 5 mg (enteral)	Within 1 h	<ul style="list-style-type: none"> Caution with renal impairment Avoid in renal artery stenosis May cause cough Contraindicated in pregnancy and idiopathic and hereditary angioedema
Furosemide	Loop diuretic	0.05 mg/kg/h Max dose: 0.4 mg/kg/h	5 min	<ul style="list-style-type: none"> May cause electrolyte and acid-base abnormalities Contraindicated in sulfonyleurea allergies
Bumetanide	Loop diuretic	0.015–0.1 mg/kg/dose every 6–24 h	Within a few minutes	<ul style="list-style-type: none"> May cause electrolyte and acid-base abnormalities Contraindicated in sulfonyleurea allergies
Hydrochlorothiazide	Distal tubule diuretic	1 mg/kg/d (oral) Max dose: 3 mg/kg/d	Within 2 h	<ul style="list-style-type: none"> May cause electrolyte and acid-base abnormalities Contraindicated in sulfonyleurea allergies Caution in patients with hypertriglyceridemia

Abbreviations: ACE, angiotensin-converting-enzyme; ANA, antinuclear antibodies; SLE, systemic lupus erythematosus.

for thiocyanate toxicity such as neurotoxicity, metabolic acidosis, pink skin, methemoglobinemia, and altered consciousness.^{68,69}

Enalaprilat is the IV form of the angiotensin-converting enzyme (ACE) inhibitor enalapril. Its use is appropriate for those scenarios where renin-mediated hypertension is confirmed. Enalaprilat is longer acting than many other IV antihypertensive medications and is dosed every 8 to 24 hours in infants and children.⁷⁰ Enalaprilat makes a unique drug of choice that addresses specific mechanisms of hypertension, and is useful in treating hypertension in neonates and children with cystic kidney disease, urologic malformations, or clinical conditions where afterload reduction is desired. It should be used with caution in renovascular disease as renal perfusion is rennin dependent. Its use can lead to decrease in GFR and/or acute renal failure. Careful monitoring of serum creatinine and potassium is warranted to avoid development of renal failure.

Diuretics are often used as adjunct to antihypertensive medications. Loop diuretics are potent diuretics such as furosemide and bumetanide, and distal tube diuretics are less potent such as hydrochlorothiazide and chlorothiazide. They are useful in treating fluid overload conditions. Diuretic therapy can mitigate volume expansion caused by vasodilating antihypertensive medications. Furosemide and bumetanide can be administered as a continuous infusion for refractory volume overload. Oral medications should be initiated as the intravenous antihypertensive medications are weaned off in preparation to transfer patients from ICU into the ward.

► **Table 1** reviews the drugs most often used in pediatric hypertensive patients. The choice of oral antihypertensive agents should address underlying pathophysiology of hypertension. Children with fluid overload related to glomerulonephritis should be treated with diuretics and vasodilators. Hyperreninemic hypertension related to renal scarring should be treated with ACE inhibitors or angiotensin receptor blockers (ARBs). Generally dose is escalated until maximum dosing has been reached at which point second medication is added and dose is adjusted to reach optimal BP before discharge from the hospital. The use of fewest medications is optimal to encourage adherence.

Conclusion

Hypertensive crisis in children is a medical emergency and should be treated promptly to avoid acute and long-term end-organ damage. In children etiology of hypertension is usually secondary in nature and differs in various age groups. Renal vascular hypertension, parenchymal disease, and anomalies are most common causes of secondary hypertension. It is important to determine the etiology of hypertension so that appropriate reduction in BP is achieved with suitable antihypertensive medications without compromising blood flow to the vital organs. In acute and chronic neurologic complications, visual and acute renal failure has been described with rapid reduction in BP in children.^{71,72}

In hypertensive crisis, IV infusions of antihypertensive medications are preferred to manage controlled decrease in BP. In children nicardipine, labetalol, sodium nitroprusside, and hydralazine are the most frequently used IV drugs. However, the choice of antihypertensive should be based on primary cause of hypertension. After stabilizing hypertension, long-term follow-up in the clinic is warranted.

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