Emergency Tips

MANAGEMENT OF HYPERTENSIVE EMERGENCIES

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Hypertension is defined as a blood pressure (BP) above 95th percentile for the age(1). Blood pressure readings between 90th and 95th percentile values for the age on at least 3 occasions is labelled as high normal BP, while a systolic or diastolic BP >99th percentile is termed severe hypertension (*Table I*).

Prompt recognition of the clinical manifestations and early implementation of individualized treatment is of critical importance. To emphasize proper approach to clinical evaluation and treatment of high blood pressure in the emergency setting it is differentiated into Hypertensive Urgency and Emergency(2).

Hypertensive emergency (HTE) is a BP high enough to cause acute end organ damage requiring reduction of arterial pressure within one hour. The end-organ damage may manifest as encephalopathy, intracranial bleed, acute left ventricular failure and pulmonary edema, acute renal failure, and retinopathy. It may occur at lower levels of BP in patients with recent onset hypertension than in those with long standing hypertension. Thus, there is no specific BP level that constitutes an HTE.

Hypertensive encephalopathy is the most common manifestation of HTE in children and is defined as transient cerebral dysfunction that occurs when blood pressure rapidly increases.

Hypertensive urgency (HTU) is defined as high blood pressure which does not pose any immediate risk to cardiovascular or other end-organs; the blood pressures may be reduced slowly in 24 hours. These patients, in contrast to those of HTE, may be managed on an ambulatory basis.

Table II gives the common causes of hypertensive emergencies and urgencies in children. In our population severe hypertension is most likely to be secondary hypertension of renal etiology or vascular cause; essential hypertension is rare(3)

Management

Initial Assessment: A symptomatic hypertensive patient constitutes hypertensive emergency which needs immediate attention. The initial assessment should be directed towards identification of affected end-organs and possible cause (*Table III*). The history taking and examination should be brief and relevant. The diagnostic work up must be individualized according to the history and physical examination findings (*Table IV*). Complicated diagnostic tests

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Accepted: June 2, 1992

TABLE I—*95th and 99th Percentile Limits for Pressure at Different Ages. [Adopted from Task Force Recommendations(1)].*

Age	95th-99 Centile		>99th Centile	
	Systolic	Diastolic	Systolic	Diastolic
<7 days	>96	–	106	–
7-30 days	104	–	110	–
1 mo-2 yrs	112	74	118	82
3 yrs-5 yrs	116	77	124	84
6 yrs-9 yrs	122	78	130	86
10 yrs-12 yrs	126	82	134	90

TABLE II—*Etiology of Hypertensive Crises in Pediatrics*

Renal

1. Glomerulonephritis: acute poststreptococcal, Henoch-Schonlien or chronic
2. Pyelonephritis: acute and chronic
3. Hemolytic uremic syndrome
4. Congenital malformations (polycystic disease, dysplasia, hypoplasia)
5. Obstructive uropathy

Renovascular diseases

1. Coarctation of aorta
2. Aorto-arteritis
3. Renal artery anomalies
4. Vasculitis

Endocrine

1. Pheochromocytoma
2. Neuroblastoma
3. Adrenogenital disease
4. Cushing' syndrome

Miscellaneous

1. Corticosteroid administration
2. Gullain-Barre Syndrome
3. Raised intracranial pressure
4. Essential hypertension

and transport of the patient to other departments for evaluation should not be done until BP is adequately controlled(4).

General Principles: The patients with HTE should be hospitalized, have an intravenous access established and placed on a BP monitor. The drug therapy should be initiated without delay.

Each patient needs a precise and individualized management and choice of medications. The two most important considerations in the treatment are etiology and duration of hypertension. Etiology determines the selection of the most effective antihypertensive agent, while acuteness of

TABLE III—*Clinical Findings in Presence of which Hypertensive Emergency Must be Looked For.*

Symptoms	Signs
Headache	Short stature, peripheral edema, pallor
Dizziness	Tachycardia, increased sweating, flushing
Excessive crying	Moon faces, obesity
Epistaxis	Absent or delayed femoral pulses, leg pressure significantly lower than arm pressure
Failure to thrive	Abdominal bruit
Joint pain	Hypertensive fundoscopic changes
Convulsions	Neurologic deficit, *e.g.*, absent pupillary reflex, hemiparesis
Hemiplegia	
Irritability, confusion	
Visual disturbances	
Personality changes	

TABLE IV—*Diagnostic Work up in Hypertensive Emergency*

General	Specific
Hemogram:	Ultrasound abdomen
Renal disease—low hemoglobin	Intravenous pyelography
Urine—microscopic and routine for	Echocardiography
albumin, RBCs, pus cells and casts	CT scan
Renal parameters—urea, creatinine	Plasma renin
Electrolytes—renal disease	Urinary 17 ketosteroids,
mineralocorticoid excess	Vanil-mandelic acid and
Chest *X*-ray	Catecholamines
ECG	

BP elevation defines the rapidity and degree by which it should be reduced.

Therapeutic Goal: The goal of emergency management of HTE with an acute rise in BP is to lower the mean arterial pressure by 25% over a period of minutes to one hour(4). It is preferable to use one drug when the patient's condition is acute. The BP in HTU (severely hypertensive but asymptomatic patients) should be decreased by one third of planned reduction in first 6 hours, by another third during the next 12-36 hours and the last third during next 48-96 hours. Precipitous reduction of BP to normotensive levels provokes an end organ ischemia or infarction due to loss of autoregulation(5). More gradual reduction using oral drugs obviates this problem. Complications of hypertension should be managed appropriately.

Monitoring: The effectiveness of therapy should be monitored by serial

measurement of BP, ideally by direct intra-arterial monitoring. Serial neurologic examinations are also important in the child with hypertensive encephalopathy.

Drugs Used: The drugs used in management of hypertensive emergency fall in two groups: direct vasodilators (such as sodium nitroprusside, diazoxide, hydralazine, nifedipine, *etc.*) and sympathetic inhibitors (methyldopa, labetolol; reserpine is rarely used today). The important advantages and disadvantages, and the dosage of the drugs are listed in *Table V*. Diazoxide, trimethaphan, parenteral hydralazine and prazosin which have been used in HTE are not included in the discussion. Of the drugs available for hypertensive urgency nifedepine, clonidine, captopril and labetalol are safest and most useful.

The Choice of Drugs: This depends on patient's condition, present medication, suspected etiology and the end-organ involved. In HTE it is preferable to use the drugs that allow close monitoring of reduction of BP, such as those given by incremental infusions. If intracranial bleed is suspected one would avoid drugs that increase cerebral blood flow such as nifedipine. If heart failure is present, a beta-blocker should be avoided. Nitroprusside is generally recommended as the drug of first choice(3). However, more recently sublingual use of nifedipine as an alternative modality is finding increasing acceptance.

Sodium nitroprusside: Nitroprusside causes direct vasodilatation of capacitance and resistance vessels, it is the most potent and predictable antihypertensive drug. It does not evoke tachycardia, and by reducing the preload, it improves borderline

TABLE V–*Drugs Used in Hypertensive Emergencies*

Drug	Onset of action duration	Dose & route	Advantages/ disadvantages
Sodium nitroprusside	<30 sec; 1-5 min	0.5-8 μg/kg/min by continuous drip	Requires continuous monitoring, Thiocyanate poisoning if >72 h use. Solution photosensitive
Nifedipine	10 min; 4-6 h	0.25-0.5 mg/kg/dose (aspirate in syringe), sublingual	Occasional hypotension
Labetolol	<5 min; up to 24 h	Bolus: 0.5 mg/kg over 2 min IV. Double dose q 10 min Max: 5 mg/kg and total 300 mg	Contraindicated in asthma, CCF, heart-block; hypotension, rash; Easy to titrate
Captopril	15 min; 8-12 h	Age >6 mo 0.3-2.0 mg/kg, oral	Very useful for renal hypertension
Diazoxide	<10 min; peak at 30 min	0.1-0.5 mg/kg IV push at 10-15 min interval	Potent and convenient
Hydralazine	<30 min; 4-12 h	0.1-0.5 mg/kg IV Maximum 25 mg	Tachycardia, flushing, acute hypotension

myocardial dysfunction and cerebral blood flow. It is the preferred drug for severe long standing hypertension, hypertensive encephalopathy, hypertensive emergency associated with acute LV failure, head injury and pheochromocytoma.

It requires an infusion pump for administration, and careful and constant bedside monitoring. Since light causes rapid degradation of the drug, bottle and the tubings need to be covered with a black sheet. Although thiocyanate, a by-product of nitroprusside metabolism, gradually accumulates in oliguric patients, it does not preclude the use of the drug for up to 2 days even in anephric patients. Thiocyanate toxicity (toxic levels >100 mg/L) results in nausea, weakness, disorientation, tinnitus, muscle spasm, acidosis and psychosis(6).

Nifedipine: Nifedipine, a calcium channel blocker has a powerful vasodilator action with little effect on cardiac conduction. In clinical practice the potential negative chronotropic, inotropic and dromotropic effects are seldom a problem. Its dilating effect on the coronary vessels and the decrease in afterload results in a lesser myocardial oxygen demand. The impaired rennin and aldosterone secretion may also contribute to its antihypertensive action.

Side effects are uncommon. Hypotension is rare. Minor side effects are related to vasodilatation; these include peripheral edema, headache, flushing and parasthesias which usually diminish with the use.

The drug has been considered to be a safe and effective antihypertensive agent for infants and children(7). In a study involving 21 children, sublingual nifedipine in the dosage 0.25-0.5 mg/kg decreased arterial pressure from 183/136 to 132/98 mmHg in 30 minutes. The antihypertensive effect started within 10 minutes, packed at 30 minutes and continued for 6 hours. An increase in heart rate was reported in most of the patients in this study(7).

In our emergency room we are using sublingual nifedipine for almost all the hypertensive emergencies since 1989, in children 6 months and above. In the first year of our experience it was possible to achieve a 25% reduction in BP within 30 minutes in 19/21 children with a sublingual dose of 0.5 mg/kg (*Fig.*). Of the two patients who did not respond one had rapidly progressive glomerulonepheritis and another pheochromocytoma.

In view of its predictable and reliable response convenience of route of administration, and lack of side effects which are often associated with centrally acting agents (sedation, CNS depression) or with diuretics (electrolyte imbalance, hypovolemia) this drug may currently be considered as the drug of choice for management of hypertensive emergency and urgency in children in setting where facility for sodium nitroprusside infusion and its monitoring are inadequate.

Labetolol: Labetolol is a combined nonselective antagonist of α- and β-adrenergic receptors. It decreases heart rate, total systemic vascular resistance and renin activity while renal perfusion is maintained. It must be used with caution in heart failure and bronchial asthma due to its beta-antagonism. Its experience in pediatric age group is limited though it is considered to be safe for use(8).

Captopril: Captopril is a angiotensin converting enzyme inhibitor and therefore particularly useful in renin dependent hypertension. It is started in a dose of 0.3 mg/kg orally, and doubled every two hourly till the desired effect or a dose of 2 mg/kg is reached. It does not affect CNS.

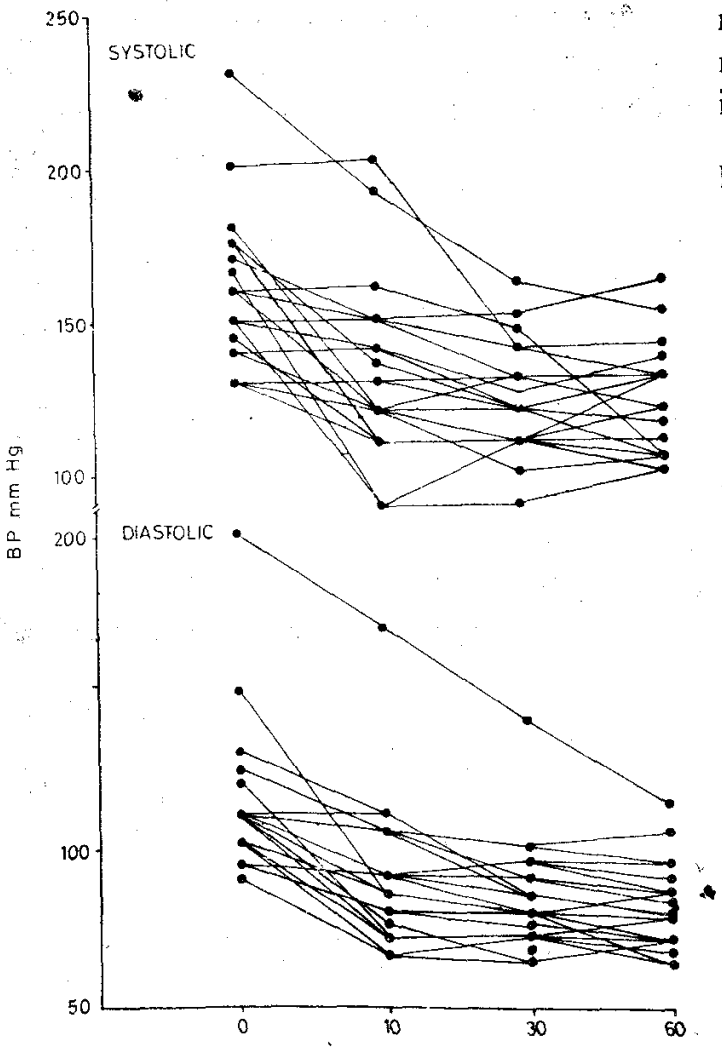

Fig.. Blood pressure changes in 19 patients at 10, 30, 60 minutes after sublingual nifedipine (0.25-0.50 mg/kg). Each line indicates the values for one patient.

Outcome

The exact mortality of hypertensive emergency in children is not known, but the risk of death has been diminished with early recognition of the condition and use of more efficacious drugs. The patients may require further management after initial control of BP such as correction of renal artery stenosis, removal of tumor, correction of coarctation of aorta, or it may be incurable as in chronic glomerulonephritis.

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