

## HYPERTENSIVE ENCEPHALOPATHY

### Introduction

- A hypertensive crisis is present when the diastolic pressure rises above 120 mmHg.
- The clinical classification into emergencies and urgencies will determine the management.
- In hypertensive emergency acute and progressive vascular and/or organ compromise is present and reduction of the blood pressure within minutes to hours is necessary.
- In hypertensive urgency the patient has no evidence of end organ damage or vascular compromise, but is at risk of progression to an emergency. This requires reduction in blood pressure within 24-48 hours
- There is a continuum between the clinical syndrome of hypertensive urgency and emergency, hence their distinction may not always be clear and precise.
- It is more the compromise to vital organ function rather than the magnitude of the pressure that determines the management.

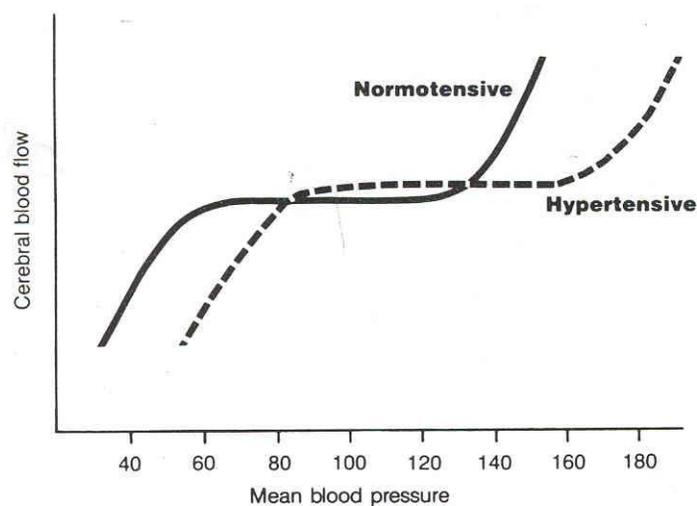
Other examples of hypertensive emergencies besides hypertensive encephalopathy include:

- Acute Pulmonary Edema
- Dissecting aortic aneurysm
- Severe pre-eclampsia and eclampsia
- Acute myocardial ischemia or infarction
- Acute renal failure
- Subarachnoid hemorrhage.

### Pathophysiology

The 3 major organ systems affected by high blood pressure are the CNS, cardiovascular system, and renal system.

- The CNS is affected as the elevated blood pressure overwhelms the normal cerebral autoregulation.
- Under normal circumstances, with an increase in blood pressure, cerebral arterioles vasoconstrict and cerebral blood flow (CBF) remains constant.
- During a hypertensive emergency, the elevated blood pressure overwhelms arteriolar control over vasoconstriction and autoregulation of CBF.
- There is a generalized vasodilation with loss of integrity of the blood brain barrier and transudate leak across capillaries leading to clinically apparent cerebral dysfunction and papilledema, the hallmark of “malignant hypertension”. The end result is hypertensive encephalopathy.
- It should further be noted that in chronically hypertensive individuals, the cerebral autoregulatory range is gradually shifted to higher pressures as an adaptation to this chronic elevation of systemic blood pressure. This can occur over a period of weeks to months.



*Cerebral autoregulation of blood flow: changes seen with chronic hypertension.*

- In addition to the CNS, the cardiovascular and renal systems are at risk of compromise. The cardiovascular system is affected by increased cardiac workload. This may be complicated by pulmonary edema, myocardial ischemia, or myocardial infarction. The renal system may show worsening function, hematuria, red blood cell (RBC) cast formation, and/or proteinuria.

### Causes

Most common cause of hypertensive encephalopathy is abrupt elevation in the chronically hypertensive patient.

Other conditions predisposing a patient to acutely elevated blood pressure can cause the same clinical situation.

Examples include:

1. Chronic renal parenchymal disease
2. Acute glomerulonephritis
3. Renovascular hypertension
4. Sudden withdrawal from chronically used hypotensive agents (eg, clonidine)
5. Pheochromocytoma
6. Sympathomimetic agents (eg, cocaine, amphetamines)
7. Eclampsia and preeclampsia
8. Ingestion of tyramine-containing foods or tricyclic antidepressants in combination with monoamine oxidase (MAO) inhibitors

### **Clinical Features**

1. Markedly elevated blood pressure.
  - The diastolic pressure will be above 120 mmHg.
2. Fundoscopic findings
  - Papilloedema is often present, together grade 3 or 4 retinopathy, if the patient has chronically elevated blood pressure.
3. Altered mental status, reflecting cerebral edema. Symptoms tend to develop gradually over 24-48 hours.
  - Headache
  - Confusion
  - Nausea and vomiting
  - Drowsiness
  - Seizures
  - Coma

## Investigations

Hypertensive encephalopathy is a diagnosis of exclusion.

Investigations are therefore directed at:

- Ruling out other causes of cerebral dysfunction.
- Finding a possible cause for the hypertension.
- Looking for complications of the hypertension

Investigation should thus include:

### Blood tests

- FBE
- U&Es / glucose
- Cardiac enzymes if myocardial ischemia is suspected.

### Urine

- FWT
- Urine toxicology screen is important in diagnosing drug-induced hypertensive encephalopathy.
- Urinary catecholamine levels, if pheochromocytoma is suspected.

### ECG

- Look for evidence of chronic hypertension or myocardial ischemia

### CXR

- Look for cardiomegaly or evidence of heart failure.

### CT scan of the brain

- To rule help out other causes of encephalopathy.

## Management

1. Attend to any immediate ABC issues.
2. IV access

3. An arterial line is desirable, but attempts at its insertion should not be allowed to delay definitive management.
4. IV diazepam may be needed for agitation or confusion.
5. IV narcotic may be required for severe headache.
6. Blood pressure management
  - Control of the blood pressure over a period of one to two hours. This needs to be done with caution in patients who have chronically elevated blood pressure, as adequate cerebral flow will not be maintained during periods of low arterial pressure that normotensive patients would easily tolerate, (see graph above). There is thus a risk of cerebral infarction if the pressure is lowered too far on these people.
  - A decrease in the mean arterial pressure by 25 % is generally safe.
  - Alternatively a diastolic pressure of 100-110 mmHg should be aimed for.
  - This target level is then maintained for several days prior to further reduction to the normal level. The latter is achieved over the ensuing weeks.
  - The agents of choice in achieving this blood pressure lowering are nitroprusside or GTN.

References:

1. Lee M: Hypertension in Textbook of Adult Emergency Medicine 1<sup>st</sup> Ed Cameron et al 2000 p. 188.
1. Murphy C 1995 Hypertensive Emergencies. Emergency Clinics of North America 13 (4): 1249-1265

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