QUESTION 81

A 28yo woman presents to the emergency department with a 4 day history of headache and blurring of vision. She has previously been well. She is on no medication.

On examination she is very confused and disorientated. Her blood pressure is 230/140 mmHg. Her JVP is not elevated, heart sounds are normal and lung bases clear. Her reflexes are generally brisk. Fundoscopy shows bilateral haemorrhages, exudates and papilloedema.

Which of the following is the most appropriate treatment?

A. Intravenous sodium nitroprusside
B. Intravenous frusemide
C. Oral nifedipine
D. Intramuscular hydralazine
E. Oral enalapril

MALIGNANT HYPERTENSION

- Malignant HT is marked HT with retinal haemorrhages, exudates or papilloedema
- Hypertensive encephalopathy is the presence of signs of cerebral oedema caused by breakthrough hyperperfusion from severe and sudden rises in BP

Mechanism:

- With mild to moderate elevations in BP above normal the initial response is arterial vasoconstriction --> tissue perfusion maintained at constant level
- With more severe elevations in BP, autoregulation fails --> vasodilation --> rise in pressure in arterioles and capillaries --> damage to vascular wall --> narrowing or obliteration of vascular lumen
- In the brain breakthrough vasodilation leads to the development of cerebral oedema and hypertensive encephalopathy
- BP at which vascular damage occurs depends on baseline BP
- If normally hypertensive, pt will have arteriolar hypertrophy that minimises transmission of pressure to the capillaries so malignant HT will occur at higher levels (eg: DBP >120)
- If usually normotensive, malignant HT can occur with DBP >100
- Patients with impaired autoregulation may also develop malignant HT at relatively low BPs

Clinical Manifestations:

- Retinal haemorrhages and exudates (representing ischaemic damage and leakage of blood/plasma from affected vessels) and papilloedema
- Malignant nephrosclerosis --> ARF, haematuria and proteinuria
- Neurological symptoms due to bleeding, lacunar infarcts or hypertensive encephalopathy
- Hypertensive encephalopathy = insidious onset of headache, nausea, vomiting followed by non-localising neurological symptoms such as restlessness and confusion --> SZ, coma if not treated
- MRI (T2): oedema of white matter of the parieto-occipital regions = RPLS
- When MRI reveals primarily pontine abnormalities the condition is called hypertensive brainstem encephalopathy

**Treatment:**

- Nitroprusside infusion (first choice): arteriolar and venous dilator
  i. Acts within seconds
  ii. Duration of action 2-5 mins
  iii. Potential for cyanide toxicity limits prolonged use, esp in renal impairment
  OR
- Diazoxide IV
- Hydralazine IV
- Clonidine IV or IM
- Oral agents can be used if less urgent – nifedipine, captopril, amlodipine, felodipine, methyldopa, prazosin
- Risk of ischaemia if BP lowered too much

**Aim of Treatment:**

- Initial goal is to reduce BP by no more than 25% in first 2 hours
- Then aim about 160/100 within 2 to 6 hours
- More aggressive hypotensive therapy is unnecessary and may cause ischaemia
- Once BP controlled, change to oral therapy
- Aim DBP 85 – 90 mmHg over 2-3 months
- Often associated with a transient decline in renal function

**Prognosis:**

- Most patients have moderate to severe acute and chronic vascular damage so at risk of vascular disease
- 90% survival

Answer: A

**REVERSIBLE POSTERIOR LEUKOENCEPHALOPATHY SYNDROME**

- Not always reversible
- Not always confined to the white matter
- Clinical syndrome of:
  i. Insidious onset of headache (usually constant, non-localised)
  ii. Confusion
  iii. Decreased consciousness
  iv. Visual changes (hemianopia, neglect, auras, hallucinations etc)
  v. Seizures
  vi. Associated characteristic MRI findings of cerebral white matter oedema
- Need to distinguish from ischaemic stroke as treatment very different (can do DWI on MRI – infarct hyperintense)

**Pathogenesis:**

- May be due to autoregulatory failure with malignant HT or cerebral ischaemia due to reactive focal vasoconstriction (autoregulatory failure more likely)
- May also be associated with endothelial dysfunction
- Combination of acute hypertension and endothelial damage results in hydrostatic oedema w leakage of serum through capillary walls, protein extravasation and fibrinoid necrosis
- More likely in white matter as grey matter more tightly packed
- Not sure why the posterior brain is involved more

**Causes:**

- Hypertensive encephalopathy
- Acute or chronic renal diseases
- Vasculitis – SLE, PAN
- Endocrine disorders – phaeochromoctoma, primary aldosteronism
- Porphyria
- Thermal injury
- Cocaine/amphetamines/other stimulants
- Eclampsia
- TTP
- HUS
- Hypercalcaemia
- Immunosuppressants
- Other medications such as HAART, EPO, GCSF, IVIg
- Blood transfusions
- Contrast exposure