Key Points:
- Hypertensive emergencies are associated with end-organ damage and need to be treated immediately; hypertensive urgencies are asymptomatic but need to be treated within hours.
- Goal of treatment for hypertensive emergency is reduction of DBP to 100-110 mmHg OR reduction in MAP by 20-25%, whichever is the greater number, over the first 2-6 hours.
- Both TTP and hypertensive emergency can cause MAHA, renal failure, and altered mental status; TTP is usually not associated with the degree of elevation of BP seen with malignant HTN.

I. Definitions: What is a hypertensive emergency vs urgency vs malignant hypertension?
   a. Hypertensive emergency: HTN with evidence of end-organ damage (usually cardiovascular, CNS, or renal) that requires immediate BP control.
      i. CV: MI, aortic dissection, pulmonary edema, CHF.
      ii. CNS: encephalopathy (HA, N/V, progression to AMS/coma), seizures, CVA.
      iii. Renal: acute renal failure, hematuria, proteinuria.
      iv. Optho: retinal hemorrhages or exudates, papilledema.
   b. Hypertensive urgency: HTN that requires control within hours but without evidence of end-organ damage.
   c. Malignant HTN: marked HTN with papilledema, retinal hemorrhages or exudates (basically a subset of hypertensive emergency).

II. Causes of hypertensive emergencies
   a. Essential HTN
   b. Renal parenchymal disease: Acute GN, TTP/HUS, vasculitis.
   c. Renovascular disease: Renal artery stenosis.
   d. Endocrine: Pheo, Cushing’s, renin-secreting tumor.
   e. Drugs: Cocaine, amphetamines most common; reported with epo, cyclosporine; anti-hypertensive withdrawal.
   g. CNS disorders: head injury, CVA, increased ICP.
   h. Autonomic hyperreactivity: Guillain-Barre, porphyria.

III. Differentiation from TTP/HUS
   a. TTP pentad: Microangiopathic hemolytic anemia, thrombocytopenia, acute renal failure, fever, altered mental status. MAHA, ARF, and altered mental status are common manifestations of malignant HTN; thrombocytopenia and fever less common.
      i. Note that it is UNCOMMON for patients to present with all 5 parts of the pentad; only MAHA and thrombocytopathy without another clear source are truly required for dx.
   b. Differentiation between TTP and malignant HTN: Patients with TTP alone would be unlikely to have the degree of elevation of BP seen in malignant HTN; also, the fundoscopic changes are unique to malignant HTN. Note, however, that TTP is on the list of diseases that can cause malignant HTN; so the two diseases may co-exist.

IV. Management: Note that these recommendations are more consensus-of-experts quality than true RCT-proven guidelines.
      i. Goal is reduction of BP to DBP of 100-110 mmHg (but reduce MAP by no more than 20-25% of initial) over first 2-6 hrs. Careful monitoring for worsening of CNS status.
      ii. Rx choices:
         1. Sodium nitroprusside (Nypride): Usual first line therapy. Can cause cyanate or thiocyanate toxicity (after 24-48 hours of rx), which is more of a worry in
patients with underlying renal or hepatic dysfunction. Onset immediate, duration of action 1-2 minutes.

3. Fenoldopam: Peripheral D1-receptor agonist that causes direct vasodilation, renal-arterial dilation, and natriuresis.
4. Others: hydralazine, IV nitroglycerin, nicardipine

iii. Special situations:
1. Eclampsia: Deliver the baby; MgSO4
2. CVA: More permissive HTN

b. Hypertensive urgencies: Can usually be managed by oral agents