

# SEVERE HYPERTENSION

(Last updated 07/22/2019; Reviewers: Philippe R. Bauer, MD; C. Schmickl, MD, MPH; Chun Wan, MD)

**PRESENTING COMPLAINT:** headache, shortness of breath, nausea/vomiting, blurry vision, altered consciousness

## FINDINGS

- **A** Check Airway
- **B** RR↑, increased work of breathing (if pulmonary edema)
- **C** BP↑, HR↓/N, or palpitation, arrhythmias
- **D** Variable altered (V,P,U,D)\*
- **E** Papilledema/ophthalmoscope
- **L<sub>PC</sub>** ↑/N creatinine, urea, cardiac biomarker, BNP, Abnormal urinalysis
- **U<sub>PC</sub>** B lines if pulmonary edema, left ventricular hypertrophy and/or enlargement

\*V (verbal), P (pain), U (unconsciousness), D (delirious)

U<sub>PC</sub> (point of care ultrasound) L<sub>PC</sub> (point of care labs)

## DEFINITIONS

- **Severe hypertension:** systolic blood pressure (SBP) >180 mmHg and/or diastolic blood pressure (DBP) >120 mmHg
- **Hypertensive urgency:** severe hypertension without acute end-organ damage
- **Hypertensive emergency:** severe hypertension with acute end-organ damage
  - Encephalopathy, retinopathy, acute kidney injury, cardiovascular manifestations

## OTHER HISTORY

- Dizziness, poor attention, hypomnesia, tachycardia, chest distress, increased nocturia, headache, confusion, nausea/vomiting, seizure, coma (encephalopathy)
- **Signs and Symptoms**
  - Retinopathy (retinal hemorrhages, exudates +/- papilledema), compare blood pressure (BP) on both arms, acute kidney injury (hematuria and proteinuria), cardiovascular manifestations (acute pulmonary edema, acute coronary syndrome, aortic dissection, acute hemolytic anemia, thrombocytopenia)
- **Predisposing Conditions**
  - Non-compliance with medication, salt indiscretion, fluid overload

## DIFFERENTIAL DIAGNOSIS

- Renal parenchymal lesion, renal vascular hypertension, primary aldosteronism, pheochromocytoma, hypercortisolism, aortic coarctation, OSAS, drug-induced hypertension
- Rule out pain, drug overdose, intracranial hypertension, and kidney disease

## OTHER INVESTIGATIONS

- **Monitoring**
  - BP (frequent/continuous), ECG, creatinine, plasma renin activity, level of aldosterone, cortisol hormone, catecholamines
- **Ultrasound**
  - Renal artery
- **Imaging**
  - Consider CT head (e.g. encephalopathy vs stroke), CTA, or MRA of aorta and renal artery; CT or MRI of kidney and adrenal; sleep respiratory monitoring

## THERAPEUTIC INTERVENTIONS

- **General**
  - Reduce BP by 10-20% within one hour and no more than 25% within 24 hours
- **Hypertensive urgency**
  - Use labetalol (if tachycardic) or hydralazine (if not tachycardic) IV
  - May use oral agents: beta-blockers or calcium channels blockers; do not use sublingual nifedipine
- **Hypertensive emergency**
  - Use labetalol (if tachycardic) or hydralazine (if not tachycardic) IV
  - May use other agents (furosemide, metoprolol, esmolol, nicardipine, nitroprussiate) ischemic stroke and not candidate for thrombolytics
    - Do not treat hypertension unless  $> 220/120$  mmHg
  - Ischemic stroke and candidate to thrombolytics: treat hypertension to goal SBP  $< 185$  mm Hg and DBP  $< 110$  mmHg
  - Acute coronary syndrome: use morphine, nitroglycerin; do **not** use beta blocker first
  - Aortic dissection: use beta blocker or labetalol to rapidly reduce SBP to 100 mmHg and heart rate to 60 bpm
- **Acute sympathetic activity** (e.g. cocaine use): benzodiazepine, nitroglycerin, calcium channel blockers; do **not** use beta blockers
- **Pregnancy** (preeclampsia): use labetalol or hydralazine

## ONGOING TREATMENT

- **Treatment**
  - Switch to oral medications after BP-stabilization (after 6-12h)
  - Initiate long-term treatment specific to etiology
- **Prognosis**
  - Malignant hypertension: without treatment, one-year survival is 10-20%; with treatment, five-year survival is up to 70%

## CAUTIONS

- **Complications**
  - Severe hypertension may cause permanent kidney damage or other potentially fatal disease (cardiovascular events)
  - Avoid rapid fluctuations (risk of hypotension, PRESS)
- **Rapid (over-) correction**
  - Autoregulation of cerebral/coronary blood flow is often dysregulated in patients with chronic hypertension, such that lowering the BP to values relatively high in normotensive patients (e.g. SBP 140 mmHg) may cause ischemic symptoms
- **Ischemic stroke, subdural/intracranial hemorrhage**
  - Weigh benefits of lowered BP with risk of reduced cerebral perfusion pressure
  - Consider neurology input

## REFERENCES & ACKNOWLEDGMENTS

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