Preeclampsia, PRES and Intracranial Hemorrhage

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Patient

- 18 yo G1P0 with no PNC presented to ER from home due to progressive lethargy and decreased responsiveness (617pm)

- After evaluation by the pedi ER, OB was consulted (1145pm)

- When OB arrived in ER, pt found in fetal position with complaint of inability to see

- She responded appropriately to questioning after aggressive stimulation
OB assessment

- On PE, she appeared to be 38-39 weeks GA, but she and parents denied the pregnancy

- She reported no complaints other than vision loss, history of 2 day headache, decreased responsiveness, N/V

- Bedside sono revealed fetal FL of 38+2 wks, oligo and fetal hydranencephaly vs holoprosencephaly

- BP in ER was 142-159/86-92
OB assessment

- Labs (1006pm 7/4): H/H 11.1/32.7, pl 304, cr 0.94, beta 43754, UDS neg, UA 300mg/dL protein

- She was immediately transferred to LD with working diagnosis of preeclampsia/PRES (arrived 1205am 7/5)

- On LD, 4gm bolus MgSO4 given immediately, labs sent

- BP 143/81, cervix 1/80/hi
OB assessment

- NST with repetitive late decels and then bradycardia
- Section called by MFM faculty
- In OR for stat section (1232am)
- Viable female infant delivered at 1246am, Apgars 5/9, thick meconium
Anesthesia preoperative assessment

- Informed that patient would be coming up to the floor for possible C/S with working diagnosis of severe preeclampsia and PRES

- Room was prepared for both general anesthetic and neuraxial blockade
- 18 yo G1P0 female
- No prenatal care
- Somnolent
- Answering questions appropriately
- Acutely Blind
- Nausea/Vomiting
- No neuroimaging available
Differential Diagnosis

- PRES
  - Seizures
  - Headache
  - Visual Disturbances
- AMS
  - Kernig’s Sign
  - Photophobia
  - Hypertension
  - Nausea
  - Vomiting
- Liver Tenderness
- Edema
- Proteinuria
- HELLP Syndrome

*SUBARACHNOID HEMORRHAGE*

*PREECLAMPSIA*
Intraoperative Events

- The patient was positioned in the sitting position and prepped and draped in a sterile fashion.
- During this time, the patient was following commands and conversant.
- On barbotage for spinal anesthesia the CSF was noted to contain frank blood.
- Uncomplicated delivery occurred 15 minutes after entering the operating room and the total case time was approximately 30 minutes.
- Patient immediately taken to CT for neuro-imaging.
Neurosurgery events

- Neurosurgery consulted at **130am**
- At bedside in ICU within 5 minutes-BP 200/100
- Decision made for ventriculostomy after examination and imaging of patient
  - Intraparenchymal hemorrhage 3.8 x 2.2cm at level of left caudate nucleus with intraventricular extension and 5mm left-to-right midline shift
- On exam, patient obtunded, left gaze preference with hemineglect, localization to painful stimuli on left arm
- A-line, intubation and ventriculostomy placement by **220am**
Neurosurgery assessment

- BP management with nicardipine gtt with transition to oral enalapril and amlodipine
- Ventriculostomy was removed POD4
- Decision made for lumboperitoneal shunt
- Pt was discharged home on POD5
- Follow-up at 3 months: no h/a, intact neuro exam
- Post-op course consisted of BP management and neurologic/neurosurgical care
Follow-up baby

- Initial diagnosis was schizencephaly
- VP shunt placed
- Final diagnosis of porencephalic cyst/ in utero vascular infarct
- Baby with mom and grandparents
- Expect neurodevelopmental delay
Posterior reversible encephalopathy syndrome (PRES)

- A clinical neuroradiologic syndrome of heterogenous etiologies that are grouped together because of similar findings on neuroimaging studies

- Incidence is unknown
PRES

- First described in a 1996 case series (15 patients) (Hinchey et al., 1996)
- Headache
- Confusion or decreased consciousness
- Visual changes (visual neglect or cortical blindness)
- Seizures
- Assoc posterior cerebral white matter edema
Pathophysicsology

- Pathogenesis is unclear, but appears related to disordered cerebral autoregulation and endothelial dysfunction.
- Often related to an acute increase in arterial blood pressure.
- Clinically indistinguishable from hypertensive encephalopathy.
- Given vast array of etiologies, several other pathways are possible.
Likely due to vasogenic edema secondary to an acute increase in arterial blood pressure, which overwhelms the autoregulatory capacity of the cerebral vasculature, causing arteriolar vasodilation and endothelial dysfunction, leading to extravasation of fluid (i.e., preeclampsia) (Thackeray and Tielborg, 2007).

Or an acute and significant episode of hypertension that causes cerebral vasoconstriction with subsequent ischemia and edema (Thackeray and Tielborg, 2007).
Normal autoregulation
Autoregulatory failure

- Normal autoregulation maintains constant cerebral blood flow over a range of systemic blood pressures.

- When the upper limit is exceeded, the arterioles dilate allowing breakdown of the blood-brain barrier thus allowing extravasation of fluid and blood into the brain parenchyma.
Autoregulatory failure

- In chronic hypertension, the range is “reset” allowing severely elevated BPs

- Less severe elevations can cause PRES in the acute setting (ie preeclampsia)
Endothelial dysfunction

- Implicated in cases associated with preeclampsia or cytotoxic therapies

- Capillary leakage leads to blood-brain barrier disruption, which leads to vasogenic edema

- In preeclampsia, markers of endothelial dysfunction (elevated LDH, abnormal RBC morphology) may precede the clinical syndrome
Associated conditions

- Hypertensive encephalopathy
- Preeclampsia/eclampsia
- Immunosuppressive therapy
- Others: renal failure, chemotherapeutic drugs, hypercalcemia (Stott et al, 2005; Kastrup et al., 2002)
Clinical

- **Headache**
  - Constant, nonlocalized, moderate to severe, not relieved with analgesics

- **Altered consciousness**
  - Mild somnolence to confusion and agitation. May progress to stupor or coma

- **Visual disturbances**
  - Hemianopia, auras, hallucinations, cortical blindness

- **Seizures**
  - May be the presenting manifestation
  - Generalized tonic-clonic
Clinical

- Differential diagnosis
  - Stroke (thrombotic, embolic, hemorrhagic)
  - Venous thrombosis
  - Toxic or metabolic encephalopathy
  - Demyelinating disorders
  - Vasculitis
  - Encephalitis
Radiologic

- Neuroimaging is essential for the diagnosis

- Bilateral white matter edema and hypodensities in the posterior cerebral hemispheres (white and gray matter)

- Lesions are usually parieto-occipital (98.7%), but brainstem (18.4%) and cerebellar (34.2%), thalamus (30.3%), basal ganglia (11.8%) and temporal (68.4%) and frontal lobe (78.9%) involvement can occur (Gasco et al., 2008)

- Findings usually not confined to a singular vascular territory

- With prompt treatment, resolution of the findings is expected, but not always observed
Radiologic

- CT or MRI
- Can have similar findings to stroke
Diagnosis

- Clinical symptoms and radiologic evidence support the diagnosis

- Should be recognized promptly so treatment can be initiated

- In our patient population with preeclampsia/eclampsia, this typically means delivery and treatment of blood pressures
Treatment

- Acute-onset, severe systolic ($\geq 160$) or severe diastolic ($\geq 110$) hypertension lasting $> 15$ minutes is considered a hypertensive emergency

- This can develop in pregnant patients with chronic HTN or in those with no history (ACOG CO 514, 2011)
Treatment

- Severe hypertension can cause CNS injury

- The degree of systolic hypertension may be the most important predictor of cerebral injury and infarction (ACOG CO 514, 2011)

- Treatment goal is not normal BP, but a range of 140-160/90-100
  - Prevent repeated, prolonged exposure to severe systolic hypertension and loss of cerebral vasculature autoregulation (ACOG CO 514, 2011)
First-line treatment (ACOG CO 514, 2011)

- IV labetalol and hydralazine

- Risks:
  - Labetalol-neonatal bradycardia; avoid use in women with asthma or heart failure
  - Hydralazine-maternal hypotension

- If no IV access give 200mg labetalol po and repeat in 30 minutes if needed
First-line treatment  (ACOG CO 514, 2011)

- Labetalol:
  - 20mg IV over 2 minutes
  - Repeat with 40mg in 10 minutes if needed
  - Repeat with 80mg in 10 minutes if needed
  - Wait 10 minutes

- Hydralazine:
  - 10mg IV over 10 minutes

- Switch agents
First-line treatment  (ACOG CO 514, 2011)

- Hydralazine:
  - 5-10mg IV over 2 minutes
  - Repeat with 10mg in 20 minutes if needed
  - Wait 20 minutes

- Labetalol:
  - 20mg IV over 20 minutes
  - Repeat with 40mg in 10 minutes if needed

- Switch agents
Second-line treatment *(ACOG CO 514, 2011)*

- If labetalol and hydralazine fail, consider labetalol or nicardipine by infusion pump
Prognosis

- Most cases of PRES are reversible in days to weeks with removal of the inciting factor and treatment of the blood pressure.

- Secondary cerebral infarction or hemorrhage.

- Death and permanent neurologic impairment are possible.
Intracranial hemorrhage

- Preeclampsia/eclampsia as causative factors of PRES is well established
  - Presence of hemorrhagic events with PRES is reported once

- Changes in the blood brain barrier as a consequence of cerebral autoregulatory dysfunction could be responsible for the initial encephalopathy (PRES), and this disruption could create a predisposition of the microcirculation to a hemorrhagic event (Gasco et al., 2008)
The favorable outcome of our patient was due to rapid delivery, blood pressure control and immediate ventriculostomy placement once the intraventricular hemorrhage was identified.
Obstetricians must be vigilant and investigate suspicious acute CNS complaints with radiologic studies in antepartum/intrapartum/postpartum patients, especially with hypertension.