

Right frontal intraparenchymal hemorrhage case study



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History of Present Illness: The patient is a 30 year old G5P5 woman with no significant PMH, on postpartum day 11, who is being transferred from the NICU to the stroke service for further management of a right frontal intraparenchymal hemorrhage and diffuse cerebral vasculopathy on angiography. The patient had a normal spontaneous vaginal delivery on 9/1 and presented to Kimball Hospital on 9/4 with sudden onset, severe headache. Her headache was left sided and associated with neck pain that was worse with lying down and turning. She was admitted and found to have a BP of 152/88 and a normal head CT. On 9/5, the patient was transferred to Monmouth Hospital for “ better care” per family.

At Monmouth, her headache generalized and intensified to 7/10. An MRI showed a 2.1cm long oval area of T2 flair hyperintensity in the right cerebellar hemisphere interpreted as posterior reversible encephalopathy syndrome (PRES). Her MRV was negative. On 9/6, her headache worsened and she became disoriented and agitated. She was sent for an interventional directed LP to rule out viral meningitis given her elevated WBC and LFT's. The LP showed opening pressure of 27, RBC of 1,030,000, WBC 4700, neutrophils 94, lymphocytes 4, monocytes 2, protein 1161, and glucose 26. A repeat head CT showed a right frontal 4x6cm intraparenchymal hemorrhage with intraventricular hemorrhage, 9mm midline shift, and cistern effacement. In addition, her neurological exam was significant for left facial droop and left arm drift.

The patient underwent emergency right frontal craniotomy and placement of an external ventricular drain. She was alert and oriented x3 and had no focal neurological deficits after the surgery. On 9/8 at 8am, the patient as noted <https://assignbuster.com/right-frontal-intraparenchymal-hemorrhage-case-study/>

by neurosurgery to be somnolent with occasional grunting. Her ICP was 43, her pupils were 7-8mm and fixed with no response to sternal rub. She was given mannitol 75g. A repeat head CT showed no changes. She was also started on hypertonic saline. Because she remained minimally responsive, she was transferred to CUMC for further management.

Hospital course: On admission, the patient underwent a head CT which showed no changes. She also underwent MRI, MRV and MRA which showed no signs of venous thrombosis or AVM. Her EVD was not functioning and was removed. Angiography showed diffuse large and small vessel vasculopathy consistent with postpartum vasculopathy. There were no aneurysms or vascular malformations. EEG showed right frontal seizures, prompting the addition of dilantin. On 9/10, the patient had a fever of 102. 5. She underwent a repeat LP which showed an opening pressure of 55, WBC 1352, RBC 974, neutrophil 94, glucose 36, and protein 103. The patient was started on empiric antibiotics – vancomycin, cefepime, and ampicillin – for presumed bacterial meningitis. CSF cultures have been negative. The patient was extubated on 9/12 and transferred to the stroke service.

- Past Medical/Surgical History: G5P5 with no complications.
- Family History: Father had a stroke in the past.
- Social History: The patient is married with 5 children. She denies smoking and drinking.

Medications:

STANDING MEDS

- Ampicillin Inj 2 G IVPB q4h

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- Cefepime +R+ Inj 2 G IVPB q8hr
- Vancomycin Inj +R+ 1 G IVPB q8hr
- Phenytoin Maintenance Inj 100 MG IVPB q8hr
- Acetaminophen Rectal 650 MG RECTAL q6hr
- Levetiracetam Inj 1000 mg IVPB q12hr
- Enoxaparin Inj 40 MG SUBQ daily 9am
- Docusate Sodium Oral 100 mg PO tid-(10, 14, 20)
- Esomeprazole (Nexium) Inj 40 mg IVPB daily 9am
- Polyethylene Glycol Oral 1 packet PO q24h
- Senna Oral 2 tab PO bedtime
- Hydrocodone/APAP 5/500 Oral 1 TAB PO q4h
- Multivitamin Oral 1 TAB PO daily 9am

PRN MEDS

- Acetaminophen Oral 650 mg PO q4h
- Maalox Plus Oral Liq 30 ML PO q4h
- Milk of Magnesia Oral Liq 30 ML PO q6hr

Allergies: No known allergies.

Physical Exam:

VITALS (last 24h):

Tc: 37. 7 Tmax: 38. 8

HR: 78 (66 – 83)

BP: 154/86 (121/72 – 154/86)

RR: 19 (18 – 19) | SpO2: 99% (95% – 99%)

- General: Lying in bed in no apparent distress.

CVS: RRR, normal S1 and S2.

- Pulmonary: Lungs clear to auscultation. No wheezing/rales/rhonchi.
- Abdominal: No pain on palpation.
- Extremities: No edema.
- Neurological:
 - MSE: Alert and oriented to person and date but thinks that she is at home. Can repeat 3/3 and recall 3/3. Can spell “ world” backwards.
 - CN: PERRLA. EOMI. VFF. Light touch intact in V1-3. Hears finger rub bilaterally. Tongue protrudes symmetrically. Uvula elevates in midline.
 - Motor: 5/5 strength in R arm, and both legs. 3/5 strength in L arm.
 - Sensory: Light touch, vibratory, proprioception, pain, and temperature sensation intact in all extremities.
 - Reflexes: 3+ biceps, brachioradialis, patellar reflexes bilaterally. 2+ Achilles reflexes bilaterally. No Babinski signs and no clonus.
 - Coordination: FNF without dysmetria.

Labs (last 24h)

ABG: pH: 7. 50, pCO2: 29, pO2: 206, HCO3: 23, SaO2 (calc): 99. 9

140 | 108 | 15

—————< 98 Ca: 7. 9 P: 3. 7 Mg: 1. 9

4. 0 | 24 | 0. 5

WBC: 13. 1 / Hb: 9. 5 / Hct: 29. 0 / Plt: 266

AST: 11 / ALT: 50 / AlkPhos: 66 / Bili: 0. 5 / Dir: 0. 1 / Prot: 5. 4 / Alb: 3. 1

Troponin: <0. 02

Imaging:

CT Head without contrast (9/8/10)

1. Postoperative changes, as described.
2. Parenchymal hemorrhage and extra-axial hemorrhage in the right frontal region, as described with focal mass effect in the right frontal region and right lateral ventricle
3. Partial effacement of the suprasellar and basal cisterns. Moderate 5 mm right-to-left midline shift.
4. Mild prominence of the temporal horn of the left lateral ventricle.
5. Right frontal hypoattenuation, which may represent edema, but evolving infarction may also have this appearance. Underlying mass cannot be excluded.

MRI of brain without contrast (9/9/10):

1. Parenchymal hemorrhage in the right frontal lobe with intraventricular extension. Associated restricted diffusion and wedge shape suggest that this may have been a primary arterial or venous infarct with hemorrhagic transformation. A less likely possibility is a hemorrhagic contusion.

2. Focal increased FLAIR/T2 hyperintensity in the right cerebellum is consistent with edema, possibly due to resolving infarct or resolving PRES.
3. Thin subdural hematoma over the right temporal lobe.
4. Soft tissue swelling over right orbit of unknown etiology; possibly trauma or related to craniotomy.

MRA of brain without contrast (9/9/10):

Decreased visualization of MCA branches in region of hemorrhage, but no aneurysm, AV malformation, or major vessel occlusion. Variant anatomy as described.

MRV of brain without contrast (9/9/10):

No evidence of thrombosis of the superficial or deep venous systems.

Echocardiogram:

1. LV size is normal. There is mild LVH. Left ventricular systolic function is normal. There is Doppler evidence of impaired LV relaxation. No left ventricular thrombus is noted.
2. Left atrial size is normal. Right atrial size is normal. Agitated saline contrast study failed to demonstrate right to left shunting at the atrial level.
3. Right ventricular size is normal. The right ventricular systolic function is normal.
4. The aortic valve is structurally normal. No aortic insufficiency is present. Aortic root dimension is normal.

5. The mitral valve is structurally normal. No mitral regurgitation is present.
6. The tricuspid valve is structurally normal. Trace tricuspid regurgitation is present. The structure of the pulmonic valve is normal. No significant pulmonary regurgitation is present.
7. No pericardial effusion.

Assessment: The patient is a 30 year old, previously healthy, recently postpartum woman who presented to an outside hospital with sudden onset severe headache. She went on to develop a right frontal intraparenchymal hemorrhage requiring a craniotomy and shows diffuse vasculopathy on angiography consistent with postpartum angiopathy. She has also developed culture negative meningitis.

Localization: Loss of strength in the patient's left arm suggests a lesion involving the right motor cortex in the territory of the MCA. Intact cranial nerve exam and sensory exam suggest that the lesion is limited to the frontal lobe.

When the patient first presented to the outside hospital with a sudden onset severe headache, i. e. a thunderclap headache, the differential was broad and included subarachnoid hemorrhage, cerebral venous thrombosis, cervical artery dissection, pituitary apoplexy, ischemic stroke, infection, and postpartum angiopathy. The most important pathology to rule out was subarachnoid hemorrhage (SAH). In terms of her symptoms, her neck pain at the time may have indicated meningismus, though she had no other symptoms of SAH such as altered consciousness and vomiting. The diagnosis

of SAH was ruled out by her normal result on brain CT without contrast, which has a 100 percent sensitivity and 98 percent specificity for SAH when performed within 12 hours of headache onset (Edlow 2000). In cases of suspected SAH, lumbar puncture is recommended as an additional diagnostic tool. Findings include an elevated opening pressure, elevated red blood cell count, and xanthochromia. It is unclear whether an LP was performed as part of the initial evaluation for this patient at the OSH. Subsequent testing with MRI/MRV/MRA ruled out some of the other etiologies for thunderclap headache such as cerebral venous thrombosis, cervical artery dissection, pituitary apoplexy, and ischemic stroke.

In this patient, the angiographic finding of diffuse constriction of large and small vessel is consistent with a diagnosis of postpartum angiopathy, cerebral vasculitis, or intracranial atherosclerosis. Intracranial atherosclerosis tends to present as a progressive syndrome in older patients with risk factors such as hypertension and diabetes. It is therefore unlikely in this patient. Postpartum angiopathy and cerebral vasculitis can be difficult to distinguish because both are associated with headache, focal deficits, stroke, seizures, and angiographic abnormalities. One way to differentiate between the two conditions is by their disease course. Postpartum angiopathy presents with an acute onset thunderclap headache and is self-limited, resolving spontaneously over the course of a few days to 3-4 months. In contrast, cerebral vasculitis presents with a subacute onset headache that is progressive (Ducros 2007). The distinction is important because cerebral vasculitis is definitively diagnosed by brain biopsy, an invasive procedure

that can be avoided in this case. Given the acute onset of the patient's headache, postpartum angiopathy is the more likely diagnosis.

Postpartum angiopathy is a member of a group of cerebral vasoconstriction syndromes. It presents in postpartum women with thunderclap headaches and often nausea, photophobia, and encephalopathy. The angiographic finding of reversible segmental vasoconstriction and vasodilation of the intracranial arteries is diagnostic. The pathophysiology of postpartum angiopathy is unknown, though it has been postulated that pregnancy related hormonal factors may lead to vasospasm. Postpartum angiopathy has been associated with intraparenchymal hemorrhage, as occurred in this patient. One suggested mechanism is that the focal stenoses found in postpartum angiopathy weaken vessel walls and lead to vessel rupture (Geocadin 2002). An additional risk factor for intraparenchymal hemorrhage in this patient is her history of repeated childbirths. One case-control study has shown that each additional birth increases the risk of intraparenchymal hemorrhage with an odds ratio of 1.27 (Jung 2010). The prognosis for this patient is good as postpartum angiopathy is self-limited. Management of postpartum angiopathy is described in the treatment plan.

This patient's meningitis is likely bacterial in etiology given the predominance of neutrophils. Empiric therapy with ampicillin, cefepime, and vancomycin has been initiated. Antibiotic regimen will be adjusted following results of blood, urine, and CSF cultures.

Plan:

Neurological

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1. Nimodipine 30mg q4hr to prevent vasoconstriction. Calcium channel blockers such as nimodipine and nifedipine have proven effective in relieving headaches and vasoconstriction (Geocadin 2002).
2. Maintain SBP <160. The patient's blood pressure should be kept within the normal range because acute hypertension has been known to precipitate the postpartum angiopathy while hypotension can reduce cerebral perfusion in the setting of vasoconstriction (Singhal 2005).
3. Levetiracetam Inj 1000 mg IVPB q12hr and Phenytoin Maintenance Inj 100 MG IVPB q8hr for seizure prophylaxis. Given the patient's intraparenchymal hemorrhage, seizure prophylaxis should be used.
4. Repeat LP if change in mental status.
5. Daily transcranial doppler is useful in monitoring the effect of treatment and demonstrating the reversibility of vasoconstriction in less than 3 months. (Zunker 2002)
6. Hydrocodone/APAP 5/500 Oral 1 TAB PO q4h for pain management.

Gastrointestinal:

1. Senna Oral 2 tab PO bedtime
2. Docusate Sodium Oral 100 mg PO tid-(10, 14, 20)
3. Esomeprazole (Nexium) Inj 40 mg IVPB daily 9am
4. Polyethylene Glycol Oral 1 packet PO q24h

The patient should be prescribed stool softeners because valsalva maneuver can precipitate worsening headaches in postpartum angiopathy (Singhal 2005).

Infectious Disease:

1. Ampicillin Inj 2 G IVPB q4h
2. Cefepime +R+ Inj 2 G IVPB q8hr
3. Vancomycin Inj +R+ 1 G IVPB q8hr
4. Follow up blood, urine, and CSF cultures.

Hematological:

1. Enoxaparin Inj 40 MG SUBQ daily 9am for DVT prophylaxis.
2. Venodynes.