Call-Fleming Syndrome: More Than A Crazy Migraine

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Call-Fleming Syndrome: More Than A Crazy Migraine

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Abstract
Occasionally symptoms do not always follow typical disease patterns which challenges prompt diagnosis. Call-Fleming Syndrome, also known as reversible segmental vasoconstriction, is a self-limited acute neurological process. It is a reversible cerebral spasm that commonly affects women in their 20-50s and may be associated with SAH. Challenges in caring for this patient include maintaining cerebral perfusion and preventing brain ischemia. Patients present with a variety of non-specific neurological symptoms which delays primary diagnosis. Advanced neuro-imaging identifies pathophysiological changes in cerebral vasculature. Utilizing a clinical case presentation, our poster will detail the pathophysiology of Call-Fleming Syndrome, describe the clinical work-up, and illustrate goal of Call-Fleming Syndrome, describe

Case Presentation
•  57 year-old female with PMHx migraine, HTN, depression, osteoporosis, GERD, lower back pain and BLS, smoker
•  Home medications: Coreg, Effexor, Paxil, and Prilosec
•  Presents with 10 day progressive deterioration in gait and mental status including agitation, confusion, impulsivity, and hallucinations
•  Began with headache—felt like “head was splitting apart”
•  Laboratory workup: Serum WBC 20.8, BUN 24/creatinine 1.4 (out-patient w/u), Thrombocytopenia Risk Profile negative; MIP #1 0.08, MIP #2 0.10, MIP #3 0.08, Blood, sputum, and urine cultures negative. CSF: RBC 967, WBC 3, protein 43, cultured negative.

Case History
•  In our case the patient was treated for vasospasm (permissive hypertension, nimodipine, hydration, close neuro monitoring)
•  Discharged Day 15 to acute in-patient rehab.

Cerebral Angiogram Day 2: Diffuse bilateral temporal, parietal and occipital areas with prominent vascular narrowing bilaterally and posterior circulation. Appearance reminiscent of vasospasm; no subarachnoid hemorrhage

Cerebral Angiogram Day 3: Diffuse central and peripheral vascular narrowing of bilateral anterior and posterior circulation. Appearance reminiscent of vasospasm; no subarachnoid hemorrhage

Cerebral Angiogram Day 4: Diffuse narrowing of the extracerebral vessels bilaterally in the posterior circulation. Appearance reminiscent of vasospasm; no subarachnoid hemorrhage

Objectives
•  List signs and symptoms of Call-Fleming Syndrome
•  Discuss medications associated with development of cerebrovascular drug reactions

Call-Fleming Syndrome
•  Syndrome first described in 1988 in a case series of 4 patients with transient fully reversible vasoconstriction around the Circle of Willis

•  Common characteristics: severe thunderclap headache with or without SAH, fluctuating motor or sensory deficits, encephalopathy, seizures, angiographic vasospasm, and normal CSF

•  Medications commonly involved in cerebrovascular disorders
•  SSRIs use linked to Syndrome
•  Oral contraceptives
•  Vasoactive drugs (pseudoephedrine)
•  Blood drugs (cocaine, amphetamines)
•  Anti-migraine agents
•  Cyclosporine

•  Conditions associated with Call-Fleming Syndrome:
•  Hypertensive encephalopathy, eclampsia, postpartum period, pheochromcytoma, CEA, IV immunoglobulins

•  Treatment is supportive. Simple Analgesics for headache: Calcium channel blockers (nimodipine) for vasospasm

•  Syndrome is benign with excellent (>95%) recovery

Day 1
MRI/MRA Admission: Multiple foca of restricted diffusion in the centrum semiovale bilaterally and the parietal and occipital lobes, suggestive of watershed ischemia.

No Hemodynamically significant stenosis.

Neuro exam on admission: Brief periods of logical conversation otherwise speech is repetitive. Able to follow simple commands.

Day 2
MRI/MRA Day 2: Prominent vascular enhancement in the posterior parietal, temporal and occipital lobes which could represent collateral flow given the bilateral watershed ischemia noted on the prior study. Severely diminished flow in the anterior and posterior cerebral arteries. Multiple intracranial stenoses in the right MCA which demonstrates decreased flow compared to the left.

Day 2: Exam essentially the same

Analysis of MRA Day 1: Day 3: Severely diminished flow in anterior MCA compared to the left.

Day 3
MRI/MRA Day 3: No Hemodynamically significant stenosis.

Day 4
MRI/MRA Day 4: Collateral circulation noted in the anterior and posterior cerebral arteries. Day 3: Left leg weakness (leg > arm) begins

Cerebral Angiogram Day 3: Diffuse central and peripheral vascular narrowing of bilateral anterior and posterior circulation. Appearance reminiscent of vasospasm; no subarachnoid hemorrhage

Day 1: Neuro exam on admission: Brief periods of logical conversation otherwise speech is repetitive. Able to follow simple commands. + Left side neglect

Patient Outcome
•  In our case the patient was treated for vasospasm (permissive hypertension, nimodipine, hydration, close neuro monitoring)
•  Discharged Day 15 to acute in-patient rehab.

Implications for Practice
•  Thorough work-up including clinical history, medication reconciliation, and previous treatments

References


Day 2: Left arm antigravity; Left leg plegic

Day 3: Left leg plegic throughout stay, Left arm does regain strength