# 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 pathophysiologic 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 directed medical treatment.

The clinical significance of our patient exam, prior medical history and treatments allowed prompt diagnosis and supportive care to meet individual perfusion needs limiting brain ischemia. Neuroscience nurses gather detailed patient information including medication reconciliation; this case demonstrates the important relevance of pharmacologic agents to disease process.

## 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
  - SSRI use linked to Syndrome
  - Oral contraceptives
  - Vasoactive drugs (pseudoephedrine)
  - Illicit drugs (cocaine, amphetamines)
  - Anti-migraine agents
  - Cyclosporine
- Conditions associated with Call-Fleming Syndrome:
  - Hypertensive encephalopathy, eclampsia, postpartum period, porphyria, CEA, IV immunoglobulins
- Treatment is supportive. Simple Analgesics for headache; Calcium channel blockers (nimodipine) for vasospasm
- Syndrome is benign with excellent (>95%) recovery

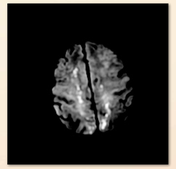
## Case Presentation

- 57 year-old female with PMHx migraine, HTN, depression, osteoporosis, GERD, lower back pain and IBS, 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"
- Laboratories: Serum WBC 20.8, BUN 24/ Creatinine 1.4 (out-pt w/u); Thrombotic 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, glucose 76, protein 43, cultured negative.

## 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.
- Neuro exam on discharge: Easily distracted but alert and oriented x3. Right sided strengths +5/5. Left arm strength +4/5; Left leg remained plegic. Able to transfer with min/mod assist of 1. Speech returning but still experienced mild cognitive and linguistic deficits.

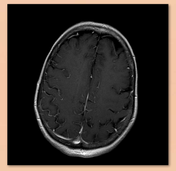
## Day 1



**MRI/MRA Admission:** Multiple foci 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. + Left side neglect.

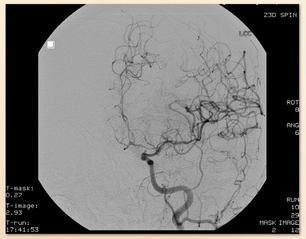
## Day 2



**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

## Day 3

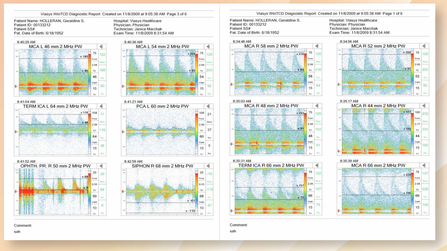



**Cerebral angiogram Day 3:** Diffuse central and peripheral vascular narrowing of bilateral anterior and posterior circulation. Appearance reminiscent of vasospasm; no subarachnoid hemorrhage

**Day 3**: Left side weakness (leg > arm) begins

## Day 4

TCD demonstrates severe vasospasm



**Day 4:** Left arm antigravity; Left leg plegic

Remains with Left leg plegia throughout stay, Left arm does regain strength

## Implications for Practice

- Thorough work-up including clinical history, medication reconciliation, and previous treatments
- Specialized neurological nursing monitoring; correlating physical exam to neuroimaging
- Aggressive neurorehabilitation
- Persistence in unusual cause of altered mental status led to an uncommon definitive diagnosis and adjustment of treatment plan

**References**

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