



5/19/20 Morning Report with @CPSolvers



Case Presenter: Moses Murdock (@haematognomist) **Case Discussants:** Kimberly Manning (@gradydoctor) Gurpreet Dhaliwal (@Gurpreet2015)

CC: Right leg weakness and paresthesias

HPI: 69 year-old man presented with right leg weakness and paresthesias.

He was ambulating normally last night. This morning, right leg felt unstable and weak. Gait was uncoordinated, slamming his right foot with each step and scuffing his toes.

He also experienced numbness and tingling in his distal right lower extremity at the calf, ankles, and foot.

Denies fevers, chills, fatigue, weight loss, chest pain, palpitations, or dyspnea. He has no headaches, vision changes, or weakness in his face or other extremities. The remainder of his review of systems was negative.

PMH:
COPD, HLD, HTN

3 prior CVAs (6 weeks prior). Aortic arch atheroma. Small PFO. No vegetations. No A-fib. Periodontal disease

Lumbar Stenosis w/ prior laminectomy
Teeth extraction 18mo.

Meds:
ASA, Plavix, Atorvastatin
Lisinopril

Fam Hx:
Non-contributory

Soc Hx:
Lives in central valley of CA
Married, monogamous, sexually active

Health-Related Behaviors:
1/2ppd tobacco use. 1-2 drinks 4-5 nights/week

Allergies:
None

Vitals: T: 37C HR: 76 BP: 147/73 RR: 14 SpO₂: 92% on RA

Exam:
Gen: Well appearing, sitting in bed
HEENT: Poor dentition. Multiple missing molars. No LAD. PERRL.
CV: regular rhythm, old II/VI harsh mid-peaking SEM at RUSB
Pulm: Normal work of breathing. Mild diffuse wheezes
Neuro: CN II-XII intact. Known L biceps weakness. RLE ¼ strength hip and knee flex/ext. Unable to dorsiflex the R foot. Diminished sensation to touch and proprioception of RLE. Brisk RLE patellar reflex. Foot drop with ambulation

Notable Labs & Imaging:
Hematology:
WBC: 10.4 (72% PMN, 19% Lymphs, 5% monos, 2% Eos) Hgb: 16.9 Plt: 318
Chemistry:
Na: 141 K: 3.4 Cl:106 CO₂: 29 BUN: 10 Cr:0.7 glucose: Ca: 9.5 Liver Enzymes, Alk phos, Albumin: normal
ESR: 62, CRP: 128 A1c: 6.3% TC: 204, HDL: 72, LDL: 120, Trigs: 59
HIV: Negative
BCX: Negative

Imaging:
CXR: Hyperinflation. No masses or effusions.
CT Brain w/o Contrast: Chronic R frontal lobe infarcts. White matter edema of superior L parietal lobe with central hypoattenuation.
MRI Brain: Chronic R frontal infarcts. 2cm x 2cm x 1.6cm ring enhancing lesion in the L parietal lobe with surrounding vasogenic edema.
CT C/A/P: No signs of occult malignancy

Prior MRI 6 weeks ago: No abnormalities in the currently affected area of the L parietal lobe
TTE: Normal ejection fraction, moderate diastolic dysfunction. No vegetations.
TEE: Negative for vegetations
FNA: Histopathology revealed no malignancy. Gram stain showed GPCs in chains. Culture grew out *S. intermedius* of the *S. anginosus* group

Problem Representation:
69 year old man with acute onset right leg weakness and paresthesias, found to have a ring-enhancing brain lesion that had developed over 6 weeks, with FNA revealing no malignant cells but gram-positive cocci that speciated as *S. intermedius*.

Teaching Points (Jack):
Problem representation: “Marie Kondo your brain.” Searching for something within a lot of information. The “who”, “when,” and “how.”
The Who: Elderly gentleman with risk factors for malignancy and CVD
The When and the What: Acute onset weakness
The How → Localizing the neurologic process.

Localizing the neurologic process: Laterality decreases the likelihood of a primary muscle disease, but disease in any other portion of the neuraxis is still possible.
Toe Scuffing: Suggests a steppage gait, which is a sign of a foot drop, often suggestive of a peripheral nerve process, but can also be due pathology anywhere in the neuraxis.
What suggest UMN vs LMN pathology here? Known cardiovascular risk factors, prior CVAs, as well as an atheroma and brisk reflexes increase UMN/CNS probability. Foot drop is more often LMN/PNS but the CNS is still possible.

Acuity of symptoms suggests acuity of pathophysiology, but this not always the case. For example, neuropathy is not generally an acute pathophysiologic process, but patients may experience it acutely.
When finding a pivot point: Ensure that the potential pivot point explains the initial clinical syndrome.

An approach to a ring-enhancing brain lesion: Malignancy (e.g., cancer that metastasizes to the brain, even without a prominent primary finding or primary brain cancer) Infections (e.g., bacteria, mycobacteria, fungi (e.g., fung-oma), parasites like neurocisticercosis, toxoplasmosis), and autoimmune diseases (sarcoidosis, GPA, multiple sclerosis). Look outside of the brain and across time.
S. Intermedius: *S. intermedius* is part of the *S. anginosus* group, which are abscess forming viridans streptococci.