



10/15/24 Morning Report with @CPSolvers

"One life, so many dreams" Case Presenter: Khashayar Khosravi Case Discussants: Gerardo Luna Peralta (@gerarlunap)



CC: Left sided weakness

HPI: 48yo R handed woman that presented with L sided weakness since 4 hours ago. Symptoms began suddenly without progression. She has difficulty in speech (dysarthria), but no dysphagia, no vertigo, no blurred vision, no diplopia, no dysphonia, no n/v. Four months earlier began presenting with episodes of R side weakness and facial paresis, dysarthria. Went to another hospital and she had a recurrent attack before admission. Symptoms always resolved by themselves within a day.

PMH:

HTN

Meds:

ASA 80 mg, Amlodipine 2.5mg, Plavix 75mg, Valsartan 80 mg, Atorvastatin 40 mg, Pantoprazol 40 mg, Citalopram 20mg, Chlordiazepoxide 5mg.

Fam Hx:

Father died at 52yo
2/2 complications of CVA.

Soc Hx:

Stay at home mom of 3. No hx of abortions.

Health-Related

Behaviors:

No smoking or EtOH.

Allergies:

Vitals: T: nl HR: 82 BP: 120/80 RR: 12

Exam:

Gen: patient awake, alert and oriented x3.

HEENT: No diplopia, no blurred vision, normal acuity, normal sclera and conjunctiva. NI optic disk no papilledema.

CV: Normal heart sounds w/o murmurs.

Neuro: Motor: no atrophy, normal tone, L side force $\frac{2}{3}$ R side 5/5. Plantar reflex down, L Babinski +, DTR +2. Sensory examination normal to light touch, pain and temperature.

Notable Labs & Imaging:

Hematology:

WBC: 6700 Hgb: 12.9 Plt: 279k

Chemistry:

Na: 135 K: 3.5 Cr:1 glucose: 92 Ca: 9.2 AST: 19 ALT:15 Alk-P:262 Albumin: 4.5 INR 1 PTT 29 TG 219 Cholesterol 145 LDL 68 HDL 47 ESR 11 CRP 39.4 ANA 0.1 Anti Ds DNA 1.2, Anti B2 glycoprotein IgG and IgM: Neg, anti cardiolipin: neg, anti phospholipid: neg, lupus anticoagulant: 40 (nl<45)

Imaging:

EKG: Normal sinus rhythm. EKG Holter (24h): normal.

Echocardiogram: Normal

Initial CT and MRI: normal.

Carotid doppler sonography: normal.

CTA: Narrow L internal carotid in the neck. No plaque or thrombosis.

DSA: bilateral ICA and A1 M1 severe narrowing.

Dx: Moyamoya disease.

Problem Representation: 48yoF w/ hx of HTN, p/w sudden L sided weakness and dysarthria w/ previous recurrent episodes alternating w/ R sided weakness for 4 months. PE reveals diminished L sided weakness, L Babinski and normal DTRs. DSA showed bi ICA and A1 M1 severe narrowing.

Teaching Points (Mukund 🙌):

Primary ddx for acute lateralized weakness: stroke vs TIA vs Todd's paresis
Neurologic manifestations of hyperglycemia include hemichorea vs seizure.

Young patient with symptoms "disseminated in space and time" -> could consider MS. However, here: symptom onset and resolution is too rapid (MS flares are acute but not sudden & resolve over weeks).

Classic MS syndromes: optic neuritis, myelitis, acute cerebellar syndromes (cerebellar peduncles rich in white matter).

Acute recurrent syndromes: seizure (unwitnessed w/postictal weakness vs seizure with negative symptoms) vs autoimmune (MS, per above) vs migraine (e.g. hemiplegic migraine) vs recurrent TIA (risk factors include: atherosclerosis vs hypercoagulability vs vasculopathy/vasculitis).

With more sensitive imaging (DWI), we are now seeing many presentations clinically c/w TIA are in reality small strokes.

Schema: stroke in a young patient, includes -

CADASIL: cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy; NOTCH3 mutation on chromosome 19. Presents with classic symptoms of recurrent stroke, migraine w/aura, psych disturbances.

Localizing the lesion: most likely in the internal capsule or pons above the facial nucleus. Without MRI findings to correlate -> consider vascular imaging.

Moyamoya: primary genetic vs secondary etiologies exist. Progressive terminal ICA stenosis leads to transient ischemic symptoms.

Encephaloduroarteriosynangiosis (EDAS) to treat moyamoya

NEJM review: <https://www.nejm.org/doi/full/10.1056/NEJMra0804622>