Stroke and Headache

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Objectives

- Understand cerebrovascular causes of headache
- Identify secondary headaches
- Evaluate and Manage stroke related headaches
Epidemiology
Migraine vs Stroke

- Headaches and Migraine are more common than stroke
- Migraines are most frequent in young patients
- Stroke is more frequent after age 65
- Stroke can occur in young patients
- Migraine and aura can start after age 50 - 60
What type of Stroke causes headache?

- Traumatic – subdural, epidural hematoma
- Subarachnoid hemorrhage
- Intracerebral hemorrhage
- Ischemic stroke
- Vasculitides
Secondary headaches

SNOOP

Systemic Symptoms
Fever, myalgia, weight loss, history of systemic illness

Neurologic Signs and symptoms

Onset - Sudden, with valsalva, positional

Older Age: > 50

Pattern change from prior headaches
ICHD II – Part Two – The Secondary Headaches
6 : Headache attributed to cranial or cervical vascular disorder

- 6.1 Headache due to stroke or TIA
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- 6.7 Headache due to other intracranial arterial disorder
  - PRES, RCVS
  - Genetic disorders – CADASIL, CARASIL and MELAS
A 62 year old man presents to the ER with a sudden, severe headache, holocranial with vomiting and vertigo BP : 190/98, neuro exam is normal in stretcher and CT head was normal. He is discharged with diagnosis of benign positional vertigo and when he stands to leave the ER he falls and is unable to stand up
Infarct in Cerebellum - MRI
Ischemic Stroke - Headache

- 27% (7-65%) have headache at onset of stroke
- Headaches are more common in:
  - Women
  - Migraine patients
  - Cerebellar strokes, basilar territory
  - Younger age
- Headaches are less common with Lacunar stroke
- Headache is not associated with stroke severity or outcome
- Headache does not predict cause of stroke
TIA or Migraine?

- TIA is sudden, migraine is a slow march with positive phenomena
- Headache is unusual with TIA –
  - Think instead: migraine with aura or aura without headache
- Typical aura – 20-60 minutes - visual, sensory or speech
- Brainstem aura – dysarthria, vertigo, tinnitus, diplopia, ataxia, change in LOC, hypacusis, visual, sensory or speech
- Hemiplegic migraine – clarify weakness not sensory only
ICHDI II – Part Two – The Secondary Headaches

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76 year old man with atrial fibrillation and hypertension, had the sudden onset of a severe, holocranial headache, right sided weakness and speech difficulty.

What is the most important question for this patient?
Intracerebral hemorrhage
Intracerebral Hemorrhage

- Sudden headache + **focal neurologic symptoms**
- Uncontrolled hypertension is primary cause, anticoagulation
- Deep location- putamen, thalamus, caudate and cerebellum

- Headache most common with caudate, cerebellar and least with thalamic hemorrhage ( < 1/3)

- Cortical hemorrhage from amyloid in elderly
- AVM, aneurysm, vasculitis, venous thrombosis, anticoagulation, trauma, drugs, tumors
Pituitary Apoplexy – ICHD II

- Severe, acute retro-orbital or diffuse headache with **at least one of:**
  - Nausea and vomiting
  - Ophthalmoplegia, low VAcuity
  - Decreased LOC
  - hypo pituitarism
  - hypotension
  - fever

- Neuroimaging: acute pituitary hemorrhagic infarction
- Headache occurs with the pituitary hemorrhage
- Headache and symptoms resolve within 1 month
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A 26 year old with a history of menstrual migraines is brought to the emergency room with the sudden onset of the worst headache of her life. The headaches reached a 10/10 intensity in about 30 seconds. She reports that this headache feels different than her usual migraine headaches.
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Subarachnoid hemorrhage
Subarachnoid hemorrhage

- 80% from ruptured saccular aneurysms
- Other causes: AVM, dural AV fistula, drugs/cocaine, vertebral dissection,
- 95% CT head shows hemorrhage
  - CT head sensitivity highest 6-12 hours after SAH
  - Sensitivity decreases to 75% by day 3, 58% by day 5
  - 5% it is negative acutely – need to do a LP
- Need Angiography – CT angiogram, ? 4 vessel angiogram
  - 14-22% angiogram is negative
Subarachnoid hemorrhage and Headaches

- 12-51% of SAH are initially misdiagnosed
- **SAH is the Most common cause of Thunderclap headache**
  - it is often the only symptom!
- 10-43% have sentinel headache before rupture
  - Occur days to weeks before aneurysm ruptures
  - Small leak of blood into subarachnoid space
  - Hemorrhage after this up to 50% mortality
Perimesencephalic Subarachnoid Hemorrhage
A 54 year old woman who had left hemiplegia after embolization in NYC hospital 20 years previously for her Sturge Weber presents with recurrent episodes of left face, arm, leg tingling with severe, pounding right sided headache with light/noise sensitivity and nausea
6.3 Headache due to Unruptured Vascular Malformation

- AVM – headache gone within 72 hrs, no ICH, SAH
  - Can have: cluster, paroxysmal Hemicrania, SUNCT
  - 58% women with AVM have migraine with aura
  - Usually is same side of AVM, not usually presenting symptoms
- Dural AV fistula
  - SAH/ICH r/o
  - Painful pulsatile tinnitus, painful Ophthalmoplegia
- Cavernous angioma
  - These can be incidental in migraine patients
- Sturge Weber
- Can you treat with triptans??
ICHD II – Part Two – The Secondary Headaches

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Temporal Arteritis

- **Age > 55 years, usually > 60**
- **Primary presentation is headache**, can be any type
- **Other features predicting positive biopsy**
  - Transient monocular vision loss
  - Jaw claudication
  - Elevated ESR, C reactive protein
  - Can also have -myalgia, fever, fatigue, anemia
- **After first eye involved, second involved ≤ 1 week**
Temporal Arteritis

- Biopsy at least 3 cm, consider bilateral biopsies
- Immediate prednisone: 1 mg/kg, do not wait for biopsy
  - Biopsy still be positive up to weeks after starting steroids
- Temporal artery biopsy is 85% sensitive
  - Increased if all clinical features present and esr/crp elevated
- Imaging studies
  - Contrast enhanced MRI of the temporal artery
  - Ultrasound to look for halo (only 40-69% sensitive)
Headache Due to Primary CNS angiitis

- A. Any new, persisting headache with D and E
- B. Encephalic signs of any type – stroke, seizures, confusion, change in LOC
- C. CNS angiitis proven by cerebral or meningeal biopsy OR suspected on angiography without systemic arteritis
- D. Headache in close temporal relation to encephalic signs
- E. Headache better within 1 month steroids/immunosuppr

*Stepwise presentation, MRI with strokes of different ages
*Absence of headache and CSF pleocytosis- Diagnosis is unlikely!
Secondary CNS Angiitis

- Systemic vasculitis- Wegners, PAN, Sjogren's,
- Headache in 50-80%
- Encephalic signs:
  - Stroke
  - Seizures
  - Change in cognition
  - Change in LOC
- Headache improves within 1 month of steroid/immunosuppressant meds
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A 28 year old, with a history of migraines, has had a prolonged labor and has transient loss of vision in right eye and severe right sided headache. Labor continues and four hours after delivery she has onset of left hemiplegia and lethargy.
Peri-partum stroke with Headache

- Cerebral venous thrombosis
- Pre-eclampsia/eclampsia
- Cerebral artery dissection
- RCVS
Right Carotid Artery Dissection
Left Carotid Artery Dissection
Carotid Artery Dissection

- Carotid and vertebral arteries are pain sensitive
- Pain is referred to the face/head
- Carotid dissection usually causes headache
  - Ipsilateral Headache in 55 – 100%
  - Often precedes ischemic symptoms
- Ipsilateral Horner’s, Pulsatile tinnitus, carotid bruit
- Ipsilateral neck pain is also common

*Cerebral artery dissection- increased in migraine patients*
Vertebral artery dissection

- Headache in 50 – 70%
- SAH with intradural dissection
- Sudden headache in occiput, nape of neck
- Stroke, SAH occur **1-10 days after headache**
- If detected after headache, may prevent stroke
Cervical Artery dissection - Imaging

- CT angiogram, MR angiogram – usually adequate
- Need intracranial and extra cranial angiograms
- MRI neck with fat saturation protocol
  - Intramural blood, crescent sign,
- Angiogram
  - Long tapered or flame shape
    - stenosis - 48%
    - occlusion – 35% (can recanalize later)
  - Intimal flap
  - Double lumen
  - Later: pseudoaneurysm
Right Vertebral Artery Dissection
Right Vertebral Artery dissection
Cerebral Venous Thrombosis

- Headache usually is the presenting feature
- Can have:
  - Papilledema, vision loss due to increased ICP
  - Seizures
  - Focal neuro deficits, confusion, decreased LOC
- Often has a hemorrhagic component + edema
- Does not conform to arterial territory, need CTV
- MR venogram - can overestimate, transverse sinus with anatomic variation is very common - CT venogram is better!
- Causes: postpartum, hereditary thrombophilias
- 23% progress, can herniate -> anticoagulation, thrombolysis
Empty Delta Sign
Cerebral venous thrombosis
Sagittal Sinus Thrombosis
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A 76 year old man with labile hypertension and renal insufficiency is admitted with a blood pressure of 240/140, with his wife noting he is variably compliant with his blood pressure medications. You are called to see him when he begins to have recurrent generalized seizures.
PRES
Clinical Features

- Slow onset headache, can have thunderclap
- Confusion, decreased level of consciousness
- Visual Changes, focal neurologic symptoms
- Seizures!
- 75% have moderate to severe hypertension
PRES
Associated Disorders

- Hypertensive encephalopathy
- Eclampsia
- Acute and Chronic Renal disease, Dialysis
- Hypercalcemia, hypomagnesium, hyponatremia
- Porphyria
- Blood products – RBC transfusion, ivIg
- Contrast media – intra arterial
- Sepsis, H1N1 A, ischemic bowel disease
- TTP, hemolytic uremia syndrome
- Vasculitis – SLE, PAN, Wegners, cyroglobulinemia,
Medications that can cause PRES

- Immunosuppressant drugs
  - Cyclosporine A
  - Tacrolimus
  - Sirolimus
  - Bevacizumab
  - Cisplatin
  - Cytarabine
  - Gemcitabine
  - Ipilimumab
  - Vincristine
  - Interferon alpha
  - Methotrexate
  - Rituximab
  - Tyrosine kinase inhibitors-sorafenib, sunitinib
  - Granulocyte colony stimulating factor
Clinical Spectrum of PRES
Lee VH et al Arch Neurol 2008; 65: 205-210

- Retrospective review of 36 patients with PRES

- 20 F, 16 M, mean age: 45 years
  - 68% had hypertension
  - 11% had eclampsia
  - 21% on dialysis
  - 24% s/p bone marrow or solid organ transplant
  - 32% had malignancies
Clinical Spectrum of PRES
Lee VH et al Arch Neurol 2008; 65: 205-210

- Seizures 87%
- Encephalopathy 92%
- Headache 53%
- Visual change 39%
- Intubation 39%
- Mean systolic BP 187 mm Hg

5/36 died
28/36 with mean f/u of 1.8 years – full recovery, no seizures
PRES
Imaging

- Symmetric WM hyper intensities- + DWI, no mismatch
  - Restricted diffusion in 11-26%- cytotoxic edema vs infarction
- Border zone regions – cortex, subcortical and deep WM
- Primarily posterior –parietal/ occipital-spares calcarine cortex
- Can be holo hemispheric – ie ACA/MCA borderzone
- Gyriform signal enhancement with gadolinium can occur
- Can look similar to post seizure MRI if mild
- Brainstem and cerebellum are often involved
- Severe cases: anterior cortex, basal ganglia
PRES Pathophysiology

- Impaired autoregulation and Dysfunctional endothelium

- If autoregulation is exceeded CPP increases with BP increase
  - Brain hyper perfusion, Vasogenic edema
  - Breakdown of BBB
  - Extravasation of fluid/blood products
  - Arterial border zone – capillary level
  - GM more packed than WM, resists edema
  - Posterior circulation has less sympathetic innervation

- Alternate: immune system (T cell) and endothelial cell activation with vasoconstriction with hypo perfusion and activation of coagulation and leukocyte trafficking

- Cytotoxic meds – may have direct toxicity on endothelium
A 44 year old with primarily menstrual migraines, calls your office on a Friday afternoon to report that 2 hours previously she had the sudden onset of a bilateral severe headache, “the worst of her life”, that reached peak intensity in less than 1 minute. She took a maxalt and repeated it in 2 hours and it is slowly resolving. Of note her psychiatrist just increased her Zoloft to 200 mg and she has been taking Claritin D for an upper respiratory infection
Thunderclap Headache
Thunderclap Headache (TCH)

- Subarachnoid hemorrhage
  - 10-43% have sentinel thunderclap like headache
  - 12% have worst headache of life with SAH
- Cerebral venous thrombosis
  - 2-13% have a TCH at onset
- Arterial dissection
  - TCH in up to 20%, ipsilateral to the dissection
- Spontaneous intracranial hypotension
  - 14% with TCH
- Meningitis - 2.7% with TCH
- RCVS – 85% present with thunderclap
RCVS
Reversible cerebral vasoconstriction eponyms

- Crash migraine
- Benign angiopathy of the CNS
- Migrainous vasospasm or crash migraine
- Call-Fleming syndrome
- Post partum angiopathy
- Isolated benign cerebral vasculitis
- Drug-induced cerebral vasoconstriction
  - Ergotamine, pseudoephedrine, phenylpropanolamine, cocaine, amphetamine, ecstasy, bromocriptine, cannabis
Headache due to RCVS

- **A.** Diffuse, severe headache of abrupt or progressive onset, with or without focal neurological deficits and/or seizures and fulfilling C and D

- **B.** “Strings and beads” or Sausage on a string - appearance on angiogram and SAH ruled out by appropriate tests

- **C.** One or both of the following
  - 1. headache develops at onset of neuro deficits/seizures
  - 2. headache results in angiogram noting strings and beads

- **D.** Headache + neuro deficits resolve spontaneously in 2 months
RCVS Features

- Headache – severe, acute, diffuse or thunderclap
- SAH ruled out – CT, LP (protein $< 80$, WBC $< 10$)
- MRA/CTA – multifocal segmental vasoconstriction and dilation in proximal and distal branches- circle of Willis
- MRI DWI- can have PRES changes, watershed infarctions
- Vasoconstriction resolves within 3 months
The Thunderclap Headache of RCVS and its Triggers

- RCVS
  - Primary – 1/3
  - Secondary – most common- 2/3
- Recurrent Thunderclap headache is hallmark
- 80% with trigger including: sexual activity, defecation, stressful situation, urination, coughing, sneezing, laughing, sudden bending down, singing
Secondary RCVS

- Pregnancy and post partum

- Drugs: phenylpropanoloamine, ephedrine, pseudoephedrine, ergotamine, methergine, bromocriptine, SSRIs, lisuride, sumatriptan, isomethptetine, cocaine, ecstasy, amphetamines, cannabis, LSD, tacrolimis, cytoxan, erythropoietin

- Blood products: iv Ig, red blood cell transfusions

- Metabolic: hypercalcemia, pheochromacytoma, porphyria, carcinoid tumors, unruptured aneurysms, head trauma, neurosurgery, endarterectomy, spinal subdural hematoma,
The clinical and radiological spectrum of RCVS. A prospective series of 67 patients
Ducros A et al  Brain 2007:130:3091-3101

- Prospective study, single center, same neurologist
- RCVS diagnostic criteria:
  - Sudden, severe headache, +/- neuro deficits/seizures
  - Cerebral vasoconstriction on MRA or angiogram with at least 2 narrowings per artery, on two different intracranial arteries
  - Disappearance of artery narrowing in less than 3 months
The clinical and radiological spectrum of RCVS-Ducros A

Results

- Average age: 42.5 (19-70) years
- Women > Men - 43 female, 24 male
- RCVS was primary in 37% and secondary: 63%
- Focal neuro deficits: 14 patients
  - transient (up to 4 hours): 11
  - Lasting > 24 hours: 5
- Seizures: 2/67
- BP >160/90 in 22 (only 2 had prior hypertension)
The clinical and radiological spectrum of RCVS-Ducros A Thunderclap Headaches

- TCH was presenting symptom in all, bilateral
- 76% TCH was the only symptom
- 63 (93%) had recurrent TCH- mean: 4.5 days after first
- 13 patients had h/o migraine – all noted TCH different
- 79% had trigger- primarily valsalva-cough, sneeze, sex
- Nausea/vomit: 57%
- Agitation: 32%
- Photophobia: 30%
The clinical and radiological spectrum of RCVS-Ducros A
Secondary causes

- 25 patients no precipitating factor
- 5 women were postpartum - 1 had bromocriptine
- 37 patients used vasoactive substances
  - Cannabis: 20
  - SSRIs: 13
  - OTC decongestants 8
  - Cocaine 3
  - Interferon 2
  - Nicotine patch 1
- Cannabis more in men and SSRI more in women
The clinical and radiological spectrum of RCVS-Ducros A MRI results

- 28% MRI brain were abnormal
- Cervical artery dissection - 4/67 patients
- 19 (28%) had abnormal MRI
  - C SAH 15
  - At least one stroke 6
  - ICH 4
  - Infarct with symptoms 2
  - Silent infarct 1
  - RPLS/PRES 6
The clinical and radiological spectrum of RCVS-Ducros A Vascular Imaging

- MRA showed diffuse, segmental artery constriction in 59 (88%)
  - 6- vasoconstriction only on second MRA – 14 days later
  - TCD in 64 - 44 (69%) had increased velocities MCA, carotid
- Angiogram – 45/67 patients
  - All had multifocal segmental artery constrictions
  - Including those with normal MRA and 6 with normal TCD
- Other arterial abnormalities in 7
  - 4 with small unruptured aneurysms – (2-3.8 mm)
    - None of these patients had SAH on MRI or CSF exam
  - 4 had vertebral artery dissection
Time Course of RCVS

- Monophasic course, no new symptoms after 1 month
- Days 1-7 Thunderclap headache, can be recurrent
- Days 7-21 Peak of vasoconstriction
- Week 12 Resolution of Vasoconstriction
- Stroke complications: 5-10%
  - RISK factors for stroke: h/o migraine, severe vasoconstriction
  - Week 1-2: SAH, ICH, seizures, PRES
  - Week 2-3: TIA's and Ischemic stroke
Listen to the patient!
Review medication list!
CT head
Toxicology screen
LP
  • r/o SAH, meningitis, inflammatory/vasculitis
  • Check opening pressure
MRI brain with and without
MRA/V and/or CTA/V
If negative- consider 4 vessel angiogram
**RCVS - Pathophysiology**

- Unknown- transient deregulation of cerebral arterial tone resulting in Vasoconstriction

Possibly due to:
- Endothelial dysfunction
  - Increased sympathetic tone
  - Oxidative stress

- Possible hormonal component

- SAH – possible minor leaks, rupture of small vessels
- c SAH occurs early – suggesting small vessel involvement first
- Stroke/TIAs later in course suggest larger vessels involved later
RCVS treatment

- Stop all vasoactive drugs
- Monitor BP, avoid high or low BP
- Nimodipine – 30-60 mg q 4-8 hours for 4-12 weeks
  - This reduces the thunderclap headaches within 48 hours
  - Does not change the time course of vasoconstriction
  - Can also use verapamil and magnesium
- Avoid glucocorticoids – can worsen, increase sympathetics
- Angiogram can cause transient neurologic symptoms in 9%
MELAS- Mitochondrial encephalopathy, lactic acidosis and stroke like episodes

- Mutation in MtDNA – maternally inherited
- Stroke like events, strokes
- Lactic acidosis, ragged red fibers – muscle
- Migraine with/ without aura, hemiplegic
- Seizures can occur
- CoQ10 may help
CADASIL
Cerebral Autosomal dominant Arteriopathy with Subcortical Infarcts and leukoencephalopathy

- Missense mutation in the NOTCH 3 gene on chromosome 19p13 – over 50 mutations
- Migraine with aura in 30-40%, (onset: 30 yrs+)
- MRI WM abnormalities – 30-40s- 2/3 micro bleeds
- Strokes in 50s – 85% have stroke or TIA
- Subcortical dementia in 60s
- Psychiatric Sx in 20-30%, depression, bipolar
Migraine that Mimics Stroke
Familial Hemiplegic Migraine

- 3 genes have been identified
- Slowly progressive hemiparesis
- Hemisensory loss, can have aphasia
- Visual aura or homonymous vision loss
- Sx spread slowly 30-60 minutes
- Severe pounding headache, contralateral follows symptoms
- Can have drowsiness, coma, symptoms can be prolonged
Headaches in Stroke Patients

Treatment

- Avoid triptans, ergots, methylsergide
- SAH – no triptans for 1-2 months after bleed
- ICH – avoid daily NSAID use
- Aggrenox can cause more headaches in migraine patients
- Selective beta blockers only as non-selective can increase vascular risk
- Calcium channel blockers, ie verapamil SR
- Topamax, Neurontin can be used
How to avoid Stroke in Migraine Patients

- Control Vascular Risk Factors
- Stop Smoking
- Aggressive headache management to decrease migraine and aura frequency
- No proven role for daily aspirin
- PFO closure does not decrease stroke risk
Conclusions

- A Sudden severe headache- think secondary headache (vascular)
- SAH, RCVS and cerebral dissection increased in migraine patients
- Rarely does Migraine cause stroke, consider other causes
- Stroke and TIAs can mimic migraine
- Migraine can mimic stroke and TIAs
- The correct diagnosis is essential!
THE END
Thank you