

Thunderclap Headache

Katie Guillen PA-C Mayo Clinic Arizona Department of Neurology

Disclosures

Non-Declaration Statement: I have no relevant relationships with ineligible companies to disclose within the past 24 months. (Note: Ineligible companies are defined as those whose primary business is producing, marketing, selling, re-selling, or distributing healthcare products used by or on patients.)

Objectives

- Define Thunderclap Headache (TCH)
- Discuss headache red flags and SNOOP criteria to understand when emergent evaluation is needed
- Discuss clinical presentation and diagnostic evaluation of TCH including pertinent imaging, labs, and procedures
- Review most common causes of TCH, as well as treatment
- Apply the information presented in a clinical case



What is a Thunderclap Headache?

A sudden onset, severe headache that begins and reaches maximal intensity within 1 minute



SNOOP Criteria

S - Systemic signs or symptoms of disease (fever, chills, myalgias)

N - Neurologic deficits

O - Sudden onset

O - Onset after the age of 40

P - Pattern, any change in headache pattern

Clinical Presentation

- Severe, rapid onset headache, reaching maximal intensity within 60 seconds
- Sentinel Headache
- Altered level of consciousness
- Vision changes
- Neurologic deficits
- Seizures
- Neck or back pain

Diagnostic Evaluation

- CT Head w/o contrast always first!
 - Evaluate for blood, intracranial masses, ischemic stroke
- Lumbar puncture
 - Evaluate for subarachnoid hemorrhage
 - Bonus: infection, increased intracranial pressure, inflammation etc.

Diagnostic Evaluation

- If CT Head/LP are not diagnostic pursue
 - MR Brain w/wo contrast
 - Vessel imaging (CTA H/N or MRA H/N)
- CBC, CMP, CXR, Urinalysis, UDS, EKG

Subarachnoid Hemorrhage

- Most common cause of TCH
- Acute bleeding into subarachnoid space
- Most commonly from intracranial aneurysm rupture
- Clinical presentation:
 - TCH, ALOC, +/- sentinel headache



Hunt and Hess Scale

Grade	Hunt and Hess Scale
1	No sx, mild HA, +/- nuchal rigidity
2	Mod - severe HA, nuchal rigidity, CN palsy
3	Mild AMS, +/- mild focal neuro deficit
4	Stupor and/or hemiparesis
5	Comatose and/or decerebrate rigidity and/or no motor response



Subarachnoid Hemorrhage

- Once SAH confirmed, urgent vascular imaging performed to look for vascular malformation
 - CTA 90-97% sensitivity in detecting intracranial aneurysm
 - Digital subtraction angiography is the gold standard

SAH Management

- Emergent stabilization if needed
 - · Airway, breathing, circulation
 - Blood pressure management
 - EVD placement
- Monitoring in ICU/PCU
 - Secondary complications
 - Monitor for cardiac and pulmonary complications, electrolyte abnormalities

SAH Management

Treatment

- Endovascular intervention or open ablation
- Timing for treatment balances risk of rebleeding/complications
- Management of complications
 Calcium channel blockers for cerebral vasospasm, initiation of AEDs for seizures

Reversible Cerebral Vasoconstriction Syndrome (RCVS)

- Most common cause of TCH
- Diagnosed based on key clinical features
 - TCH or severe, recurrent headache
 - Cerebral vasoconstriction in at least 2 different arteries
 - Resolution of vasoconstriction within 3 months
 - No PACNS or aneurysmal SAH

RCVS

<u>Clinical Presentation</u>:

- TCH, nausea/vomiting, light/sound sensitivity, altered LOC, seizures, focal neuro deficits
- CTH and MR Brain are often normal
- CTA will note multifocal vasoconstriction in intracranial arteries
 - "String of Beads" appearance

RCVS on Imaging



Case courtesy of Assoc Prof Frank Gaillard, Radiopaedia.org, rID: 4533





Case courtesy of Prof Peter Mitchell, Radiopaedia.org, rID: 34462

RCVS Management

- Withdrawal of vasoactive agents, analgesia, observation
- Initiation of calcium channel blocker
 - Treatment duration varies, usually 4-8 weeks
 - Goal: resolution of vasoconstriction on imaging
- Manage secondary complications of RCVS
- Avoidance of headache triggers
- Outpatient follow up with Neurology
- RCVS is a self-limited course

Case 1

59 YOF with hx of hypertension presents to ED with complaint of headache. Patient reported she was in a meeting when she realized her left leg couldn't move. This coincided with abrupt onset of 10/10 headache that reached maximum intensity within 15-30 seconds. She reported associated blurry vision and nausea. No vomiting, slurred speech, aphasia, or other focal neurologic deficit.

Patient also reported headache the day prior to presentation which peaked to 7/10 within minutes of onset. It resolved after patient took Naproxen.

Case 1 Continued

Vital Signs: BP: 150/99 HR: 65 RR: 16 T: 36.5 C SpO2: 97% General: WDWN, resting in bed, at times tearful but NAD Mental Status: Awake, alert, oriented x3. Disoriented to date. Language: Speech is slow but no dysarthria or aphasia. CN: PERRLA. VFI. EOMI. Normal sensation V1-3. Facial strength full and symmetric. Motor: Full strength in all extremities. Sensation: Intact to light touch in multiple dermatomes in all 4 extremities

CT Head without contrast



Case 1 Continued

- CTA H/N: L cervical ICA with focal area of luminal expansion and irregularity
- EVD placed to manage hydrocephalus
- Cerebral angiogram performed noted R ICA aneurysm
 - Flow Diverting Embolization performed
- Initiated on Keppra 500mg BID
- Initiated on Nimodipine 60 q4hours
- Daily TCDs to assess for vasospasm

Complete Thunderclap Headache Differential

- Subarachnoid Hemorrhage*
- RCVS*
- CVST
- Cerebral Infection
- Cervical Artery Dissection
- Complicated Sinusitis
- Hypertensive Crisis
- ICH
- Ischemic stroke
- Spontaneous Intracranial Hypotension
- Subdural Hematoma

- Aqueductal Stenosis
- Brain Tumor
- Cardiac Cephalgia
- Giant Cell Arteritis
- Pituitary Apoplexy
- Pheochromocytoma
- Retroclival Hematoma
- Spontaneous Spinal Epidural Hematoma
- Third Ventricle Colloid Cyst
- Primary or Idiopathic Thunderclap Headache
- Unruptured Intracranial Aneurysm

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