Abusive Head Trauma

The Kempe Center
FOR THE PREVENTION AND TREATMENT OF CHILD ABUSE AND NEGLECT

University of Colorado Anschutz Medical Campus

Children's Hospital Color

Overview

- Clinical presentation
- Victims/perpetrators
- Types of injury
- Medical evaluation and neuroimaging
- Retinal hemorrhages
- Differential diagnosis
- Timing of injury
- Outcomes

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What's in a Name?

- Abusive Head Trauma
- Inclusive of impact and inertial (shaking) mechanisms
- Can the injuries be explained by the reported history?
- Are the injuries abusive or accidental?

Epidemiology

- 0-12 month olds: 13-40/100,000 per year
- 12-24 month olds: 2.4/100,000 per year
- Many victims never present for medical care

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Presenting Clinical History

- Vomiting, fussiness, poor feeding
- Apnea, seizures, altered mental status
- BRUE (brief resolved unexplained event)
- Minor trauma (short fall)
- Macrocephaly

Spectrum of Clinical Presentation

- Vomiting
- Fussiness
- Poor feeding
- Altered mental status
- Apnea/abnormal breathing
- Seizures

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Components of AHT

- Head injury
  - Subdural hemorrhage most common
- Retinal hemorrhages
- Associated fractures?
- Often no external findings

Who Are the Victims?

- Most victims are <18 months of age
- Peak at 4-6 months
- Boys > girls
- Twin, Preterm birth
- Fussy/”colicky”

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Who are the Perpetrators?

- Predominantly male
  - Father, step-father, mother's boyfriend
- Babysitter
- Mother
- Mental illness, substance abuse, domestic violence, poverty, compromised prenatal care, criminal history

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Misdiagnosis of AHT

- 31% of children with AHT were misdiagnosed initially
- Often non-specific symptoms (vomiting, irritability)
- Mean of 2.8 medical visits and 7 days until correct diagnosis
- Young, Caucasian, 2 parent home

Clinical History

- What did you first notice was wrong?
- Timeline of events
  - Last normal feed and last normal play?
- PMHx – bruising or bleeding, increasing FOC
- Family Hx – bruising or bleeding, neurologic disorders

Types of Neurological Injury

- Primary (biomechanical)
- Secondary (cascade of effects)

- Location – from scalp to brain parenchyma

Primary (Biomechanical) Brain Injury

- Impact
  - Skull fracture
  - Localized bleeding from scalp bruise to parenchymal contusion
- Inertial (acceleration/deceleration)
  - Strain or deformation of tissues
  - SDH – bilateral, interhemispheric fissure, convexities
  - Diffuse axonal injury /deep white matter injury

Secondary Brain Injury

- Cerebral edema
  - Damage to neurons
  - Release of inflammatory mediators
  - Increased vascular flow
- Hypoxic ischemic encephalopathy
  - Apnea/bradycardia/hypotension/cardiac arrest
  - Increased intracranial pressure (edema or space-occupying lesion)
- Venous thrombosis
  - Injury to veins/sinuses

Where is the Blood?

Scalp
- Bruise or hematoma

Galea
- Subgaleal hematoma
- Cephalohematoma

Skull
- Epidural hemorrhage

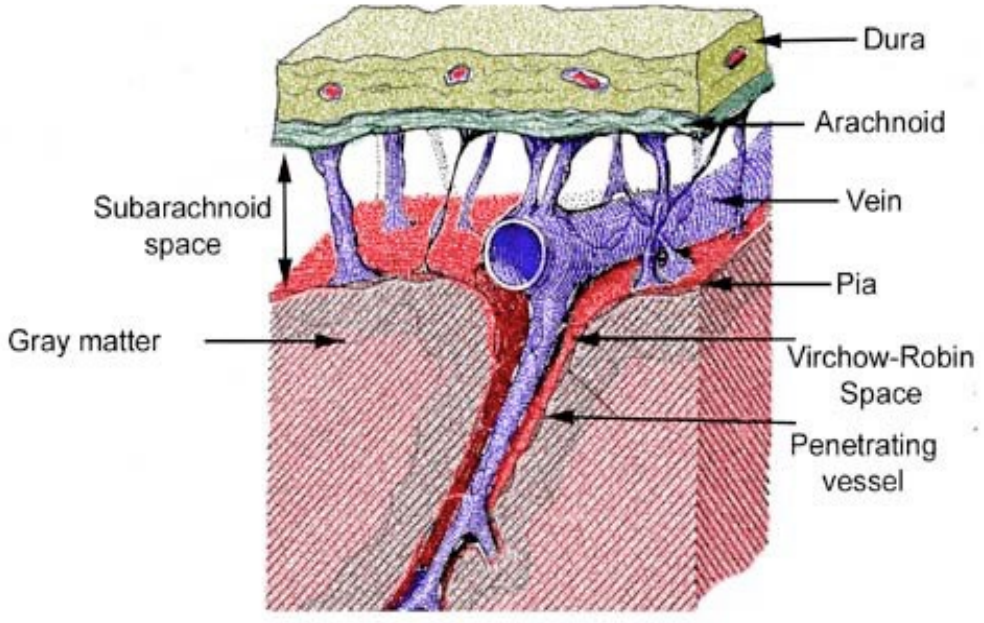
Dura mater
- Subdural hemorrhage

Arachnoid mater
- Subarachnoid hemorrhage

Pia mater

Brain
- Intraparenchymal hemorrhage
- Intraventricular hemorrhage

Subdural Hemorrhage



- Layers of dura divide to enclose dural venous sinuses
- Bridging veins that originate from the brain surface cross the sub-arachnoid and sub-dural spaces to drain into the venous sinuses
- Specific pattern – diffuse, interhemispheric, bilateral

Diffuse Axonal Injury

- Shearing injury of the white matter (axons)
- Result from angular acceleration/deceleration
- Commonly at gray-white junctions, corpus callosum, and brainstem
- Frequently no visible hemorrhage

Medical Evaluation

- Physical exam: especially skin, mouth, eyes, FOC, neuro
- Acute imaging: CT (or fast MRI)
- Follow-up imaging: MRI 48-72 hours later
- Hematology
  - CBC, PT/PTT
  - Factor VIII, IX
  - DIC panel (fibrinogen, D-dimer)
  - +/- Factor XI, XIII
  - +/- von Willebrand's Disease
  - +/- Thrombin time
- Skeletal survey
- AST/ALT

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Neuroimaging: CT Scan

- Readily available, fast, very sensitive for acute bleeds
- Do not require sedation
- Radiation exposure is not insignificant
- Not great for the details
- Description of blood as compared to CSF:
  - Hyperdense → Isodense → Hypodense
  - Mixed-density

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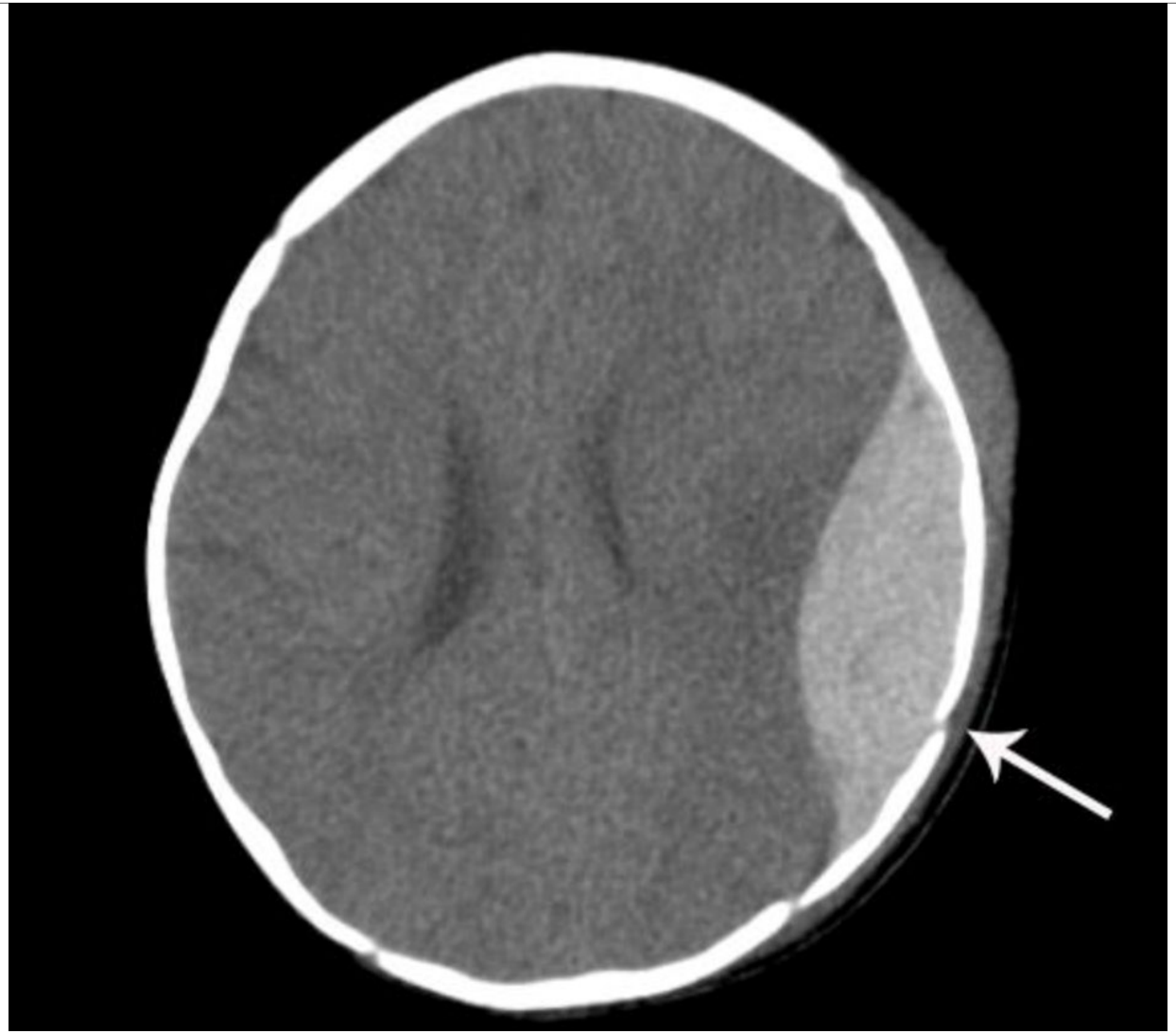
Epidural Hemorrhage



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Subdural Hemorrhage

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Chronic Subdural Hemorrhage

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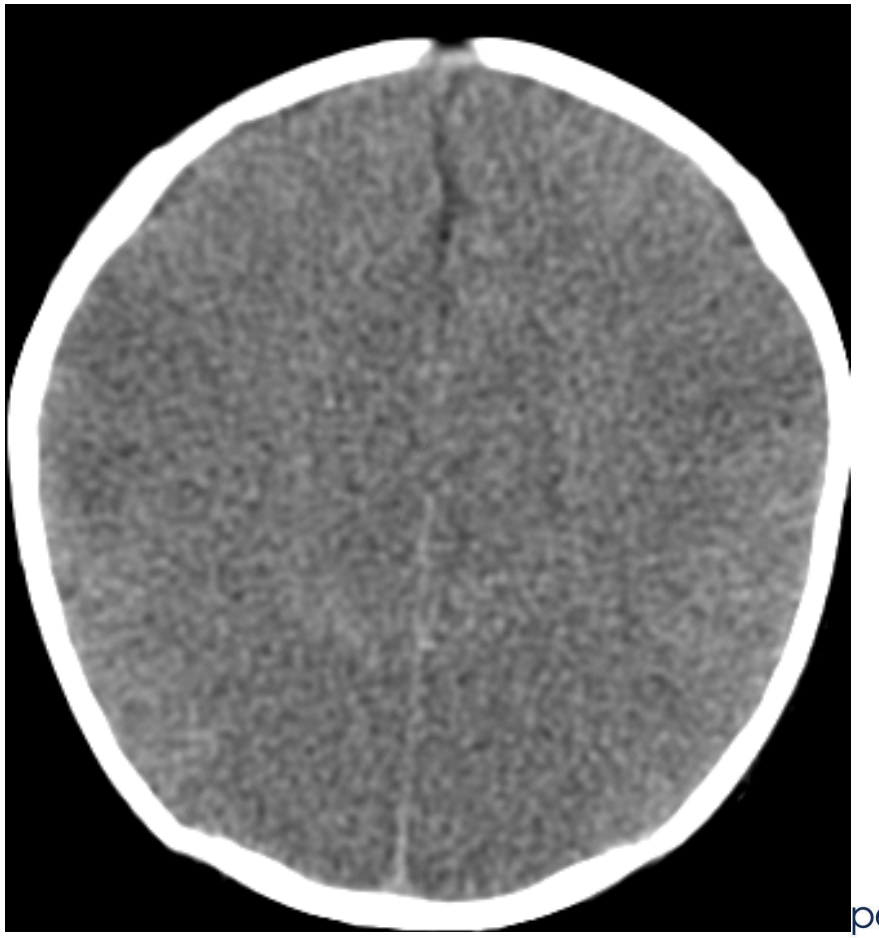
Diffuse Cerebral Edema



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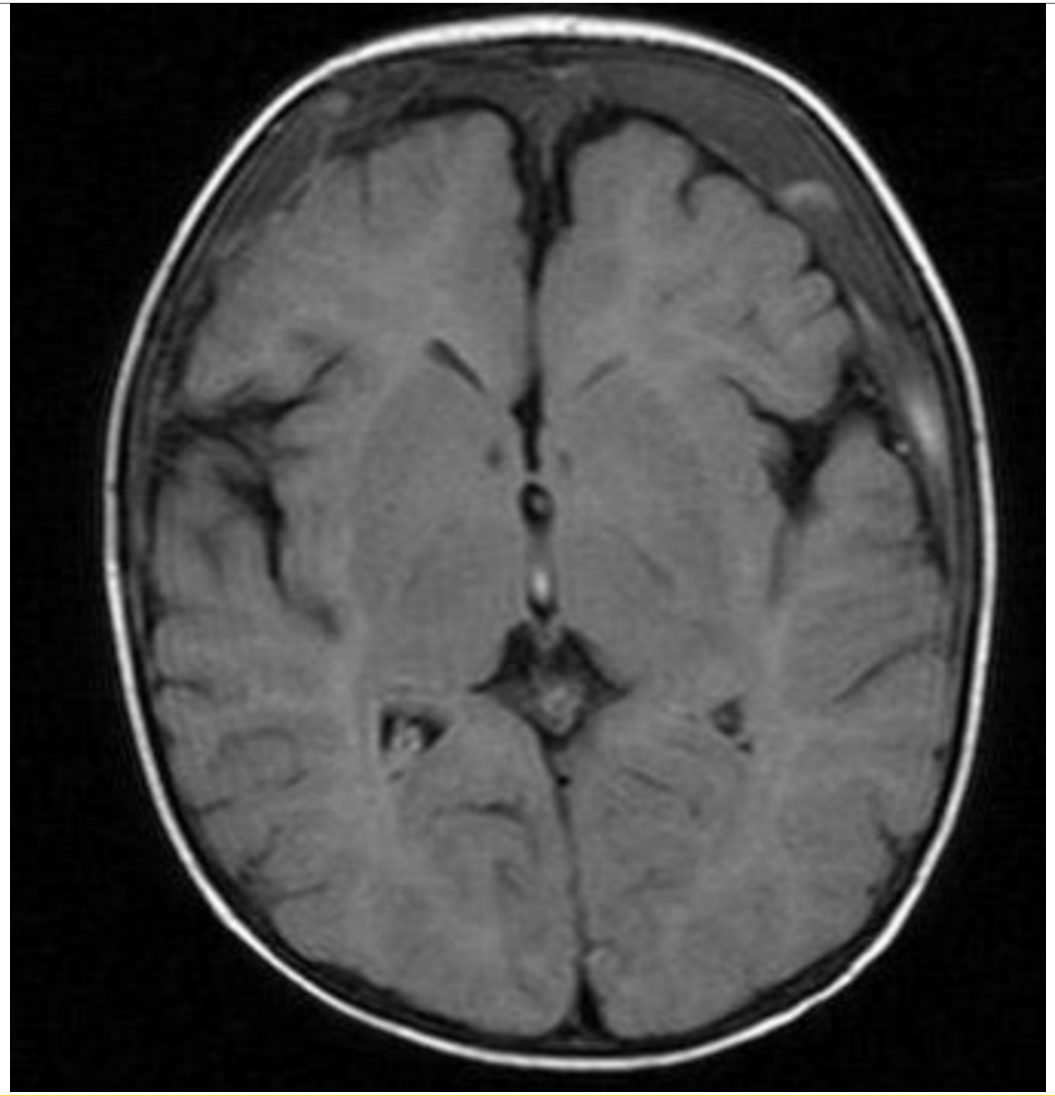
Fast MRI

- Motion tolerant
- Fast (minutes)
- No radiation exposure
- Similar to shunt series MRI
- Not as sensitive for skull fractures

Neuroimaging: MRI

- Expensive
- Slow – usually requires sedation
- Parenchymal lesions better defined
- More sophisticated description of blood appearance
- Usually obtained at 2-3 days post injury
- Many different sequences

Subdural Hemorrhage on MRI



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Spine and Neck Injuries

- C-spine and spinal cord injuries
- 71% of fatal AHT at autopsy
- 78% of non-fatal AHT on MRI
- Ligamentous injury
  - Usually at occipito-cervical junction (occiput to C2)
- Spinal SDH
  - More common in thoracic and lumbar spine

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Retinal Hemorrhages

- Present in 75-80% of AHT cases
- Usually bilateral – 80%
- Retinal hemorrhages don’t always mean abuse
- Absence of RH does not rule out abuse

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Ophthalmologic Exam

- Need an ophthalmologist to examine
- Dilated pupils
- Indirect ophthalmoscope
- Document with RetCam when possible

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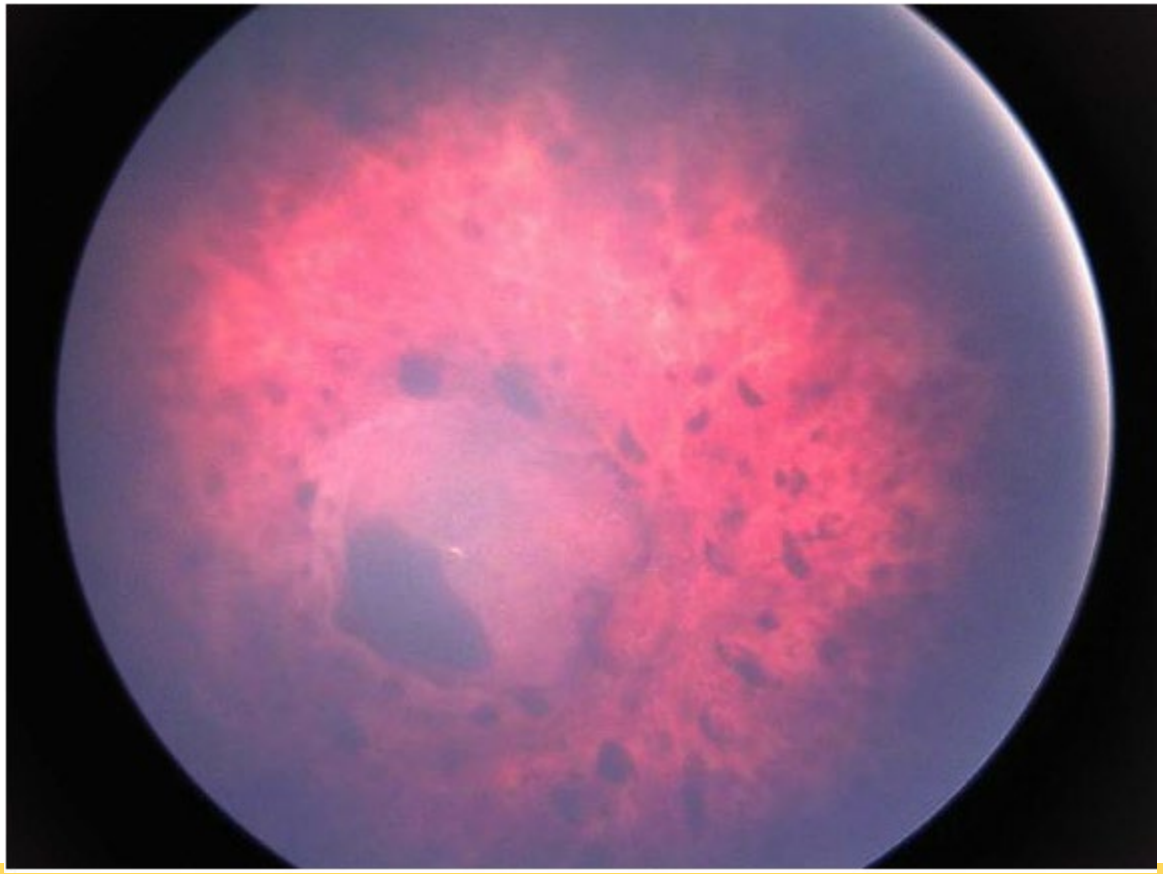
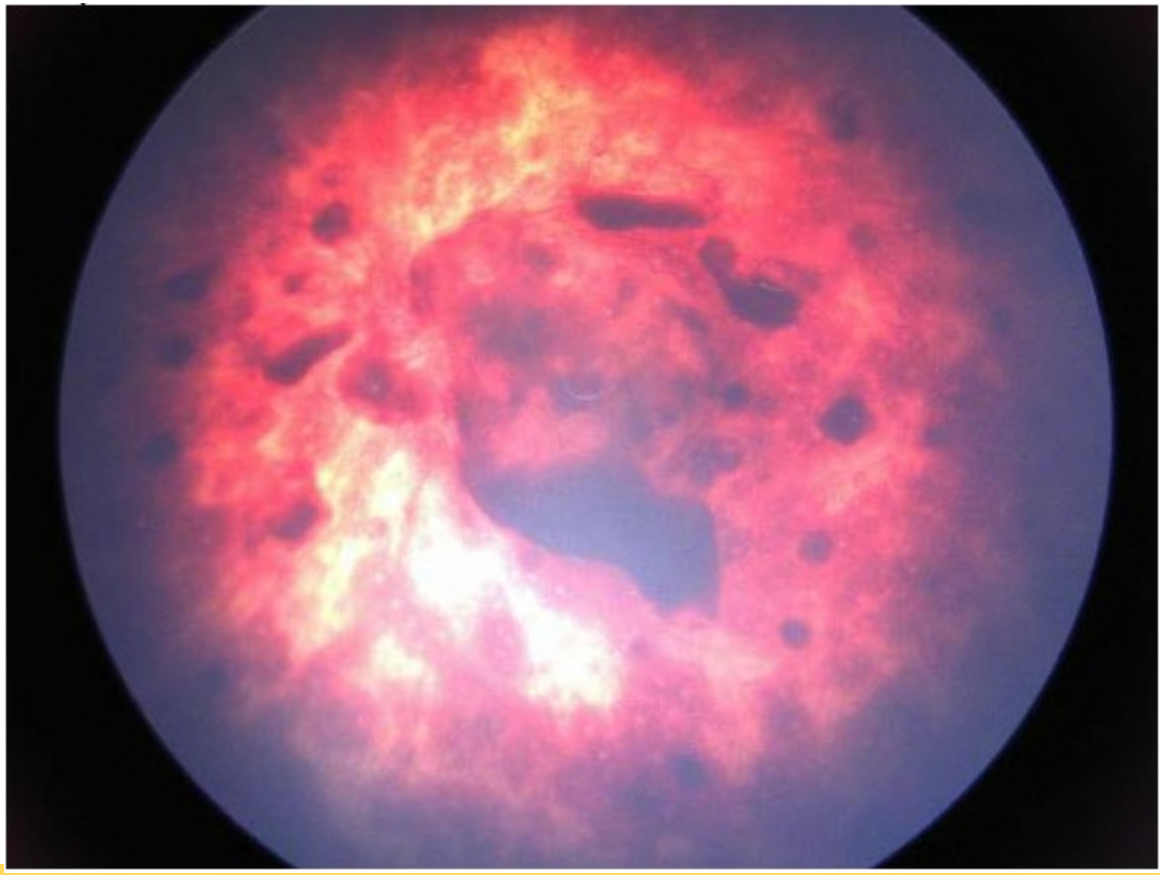
Retinal Hemorrhages in AHT

- Multiple hemorrhages (more than a few)
- Multiple layers of the retina
- Extend beyond the posterior pole to the periphery (ora serrata) of the retina
- Retinoschisis – splitting of layers of retina leading to cavity of blood or retinal fold

Eye Exam

retinoschisis, too numerous to count retinal hemorrhages





Timing of Injury

- Clinical presentation
- Appearance of blood on neuroimaging
- Could there be a lucid interval?
  - What does that mean in an infant?
  - Described in accidental head trauma – usually space occupying lesion (think EDH)
  - Perpetrators who have confessed describe immediate symptoms
- **Absent a complicating condition, 95% likelihood that a child with classically presenting AHT developed symptoms within a few minutes of the inciting trauma**

Differential Diagnosis of AHT

- Accidental trauma
  - Birth trauma – can cause asymptomatic SDH which resolves within ~4 weeks
  - Short fall – off furniture, from caregiver's arms, caregiver falls while holding infant, down stairs
- Medical
  - Benign extra-axial fluid of infancy (BEAF) or Benign expansion of subarachnoid spaces (BESS)
  - Coagulopathy
  - Glutaric aciduria type 1
  - Collagen disorder (osteogenesis imperfecta, Ehlers-Danlos syndrome)
  - Vascular disorder (AVM, aneurysm)
  - Menkes disease (kinky hair)
  - Alagille syndrome
  - Shunted hydrocephalus
  - Arachnoid cyst

BEAF/BESS

- Accumulation of CSF in the subarachnoid space
- Smooth acceleration of FOC over 15-18 months
- Usually cross 95%ile in first 6 months of life
- Family history of macrocephaly
- Occasional asymptomatic SDH without trauma history

Neurological Outcomes

- 25% mortality
- 25% normal
- 50% with variable levels of cognitive or neurologic impairment
- Often takes months or years to know sequelae
  - Seizures
  - Visual impairment
  - Cerebral palsy
  - Cognitive deficits
  - Behavioral disorder

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Selected References

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THANK YOU!

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