AANP Teaching Rounds: Neuropathology of Brain Swelling and Herniation

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Outline

- Review of etiologies leading to brain swelling
- Definitions of swelling/edema
  - Cellular bases
  - Radiographic appearances
- Patterns of herniation, with case examples
  - Tissue displacement – various compartments
  - Secondary processes
    - Herniation “contusions”
    - Territorial infarctions
    - Brainstem (Duret) hemorrhages
    - Cerebellar “emboli” to spinal canal
  - Alterations induced by intervention (EVD, SDH evacuation, craniectomy)
  - Appearances in survivors
- Brain death

Primary etiologies leading to brain swelling

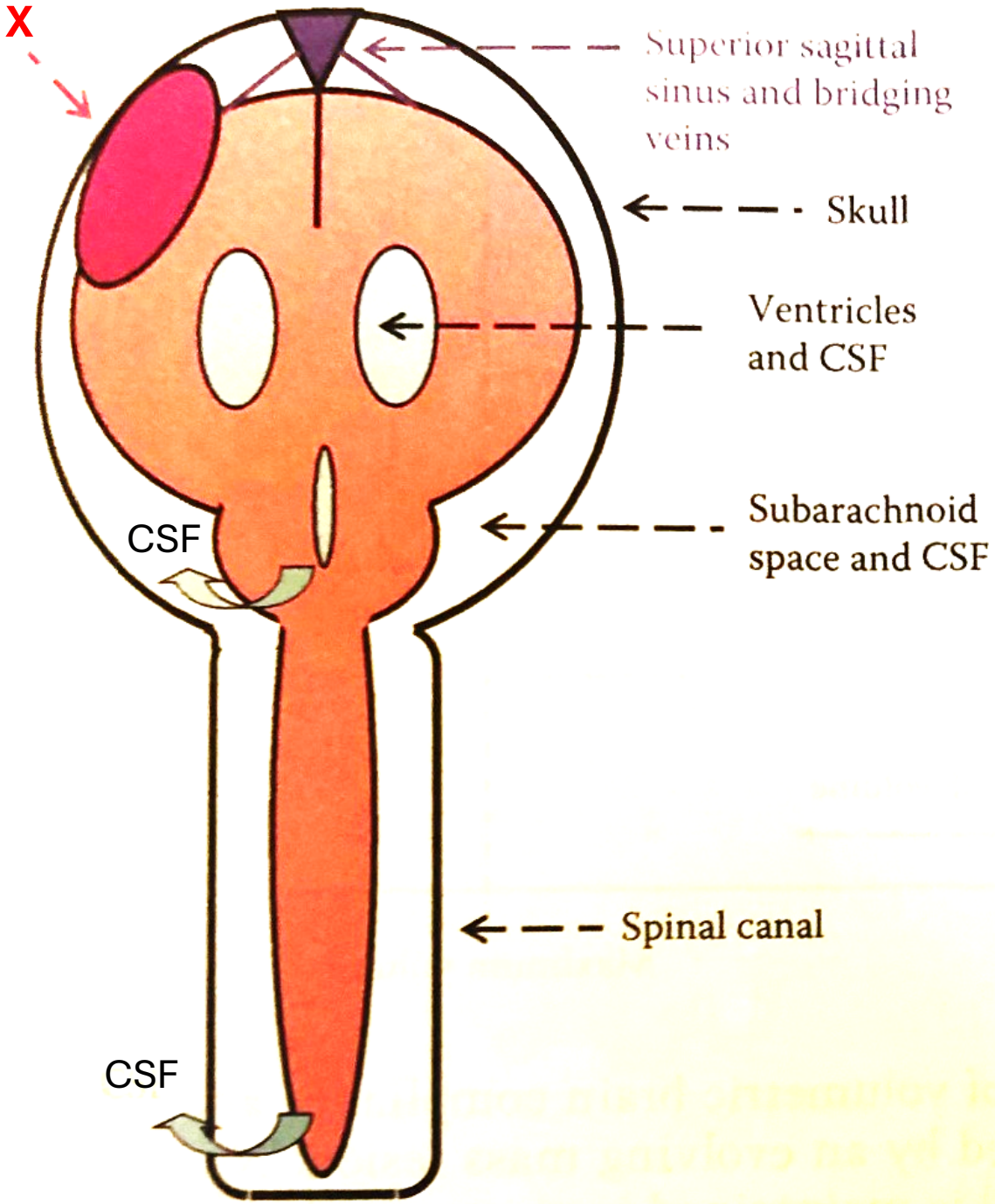
- Trauma
  - Localized impact, e.g., contusions, hematomas
  - Torsional (diffuse axonal) injury
- Non-traumatic mass lesions (neoplasia, cysts, abscesses)
  - Localized lesions
  - Leptomeningeal/ventricular involvement leading to hydrocephalus
- Hypoxia-ischemia
  - Generalized, e.g., opioid overdose, metabolic derangements (hepatic/renal failure, glucose derangements)
  - Localized, e.g., hypertensive hemorrhage, ischemic stroke
- Dural venous sinus thrombosis
- Infectious meningoencephalitis

- Non-traumatic subarachnoid hemorrhage
- Inflammatory/demyelinating lesions
- Epilepsy, especially status epilepticus
- High altitude, toxins (heavy metals, CO)

Brain swelling =

- Increased volume over baseline
  - Baseline = “normal” brain tissue volume (1400ml) + intravascular blood volume (150ml) + CSF (150ml) within fixed cranial volume
    - Range – dynamic (cellular metabolism, blood pressure fluctuation, CSF pulsation)
  - Increase can be in:
    - Brain tissue volume (cells + interstitial fluid) (*edema*)
    - Intravascular blood volume
    - CSF volume (*hydrocephalus*)
  - Rate of increase and equilibration
- Intracranial pressure = summation of normal volume range
  - Increased ICP = ≥20cm water via intracranial monitor placement (normal, 5-15)

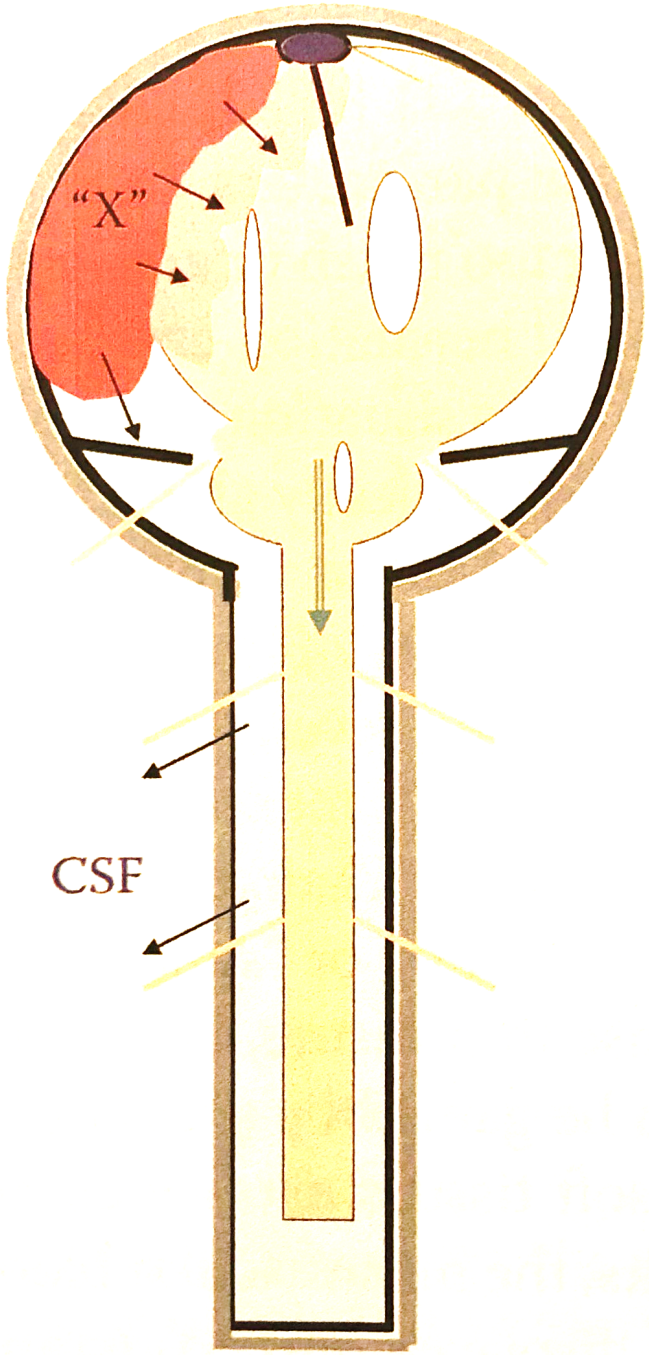
- ***“Brain swelling” is best term***
  - Specific cellular basis may be unknown and/or multiple, especially at autopsy
  - Histology usually unhelpful



$$\text{Volume}_{\text{total}} = V_{\text{brain/cord}} + V_{\text{blood}} + V_{\text{CSF}} + V_{\text{"X"(subdural)}}$$

- Intracranial volume is fixed by rigid skull
- When $V_{\text{"X"}}$ increases, CSF is absorbed to make room until no more CSF is available

From Leestma, Forensic Neuropathology, 3rd ed., 2014

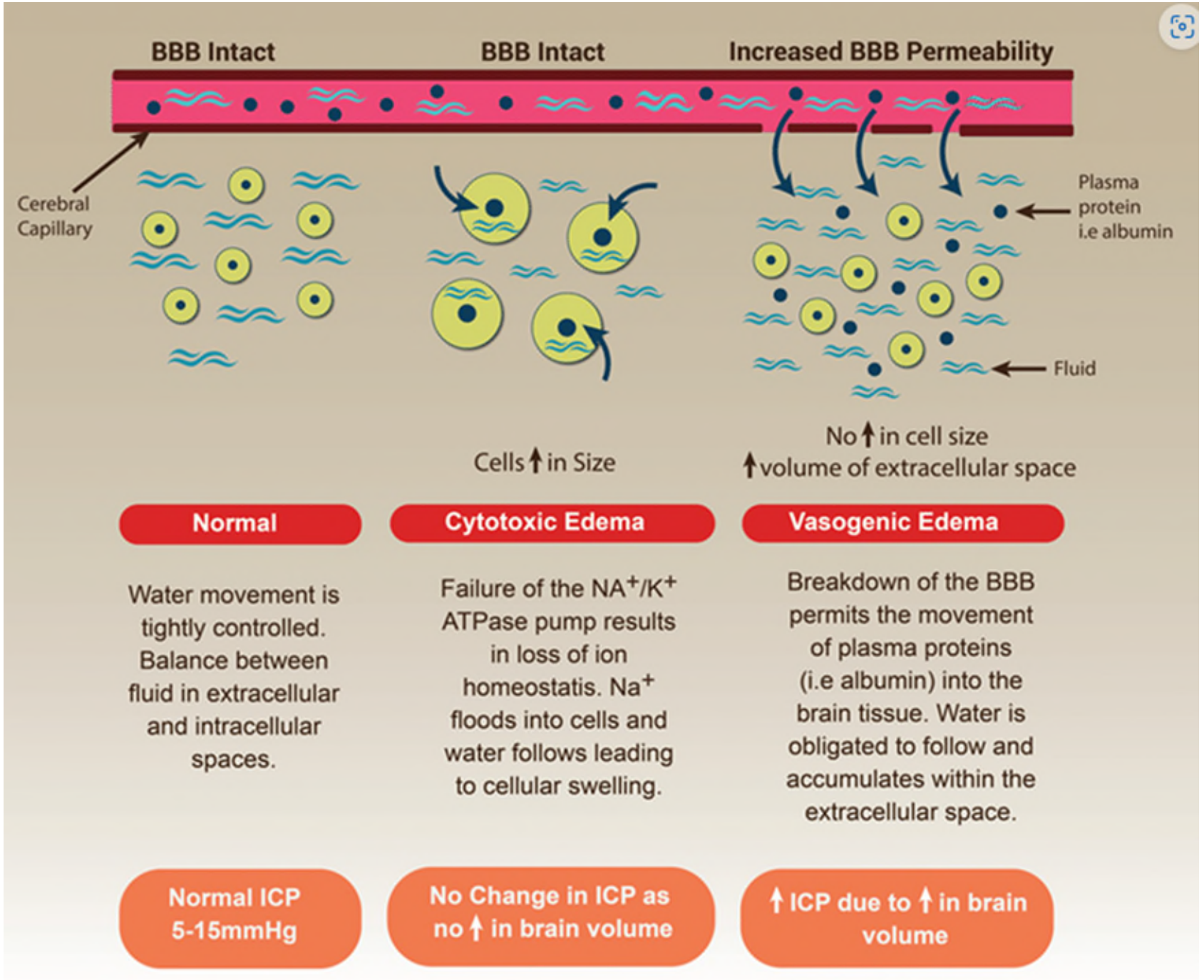


- When hematoma (or mass) expands, CSF is driven out to try to maintain normal pressure.
- Brain shifts (herniates) away from mass. Ischemia and edema at edge of mass may add to mass effects.
- When cranial CSF is mostly absorbed, spinal CSF is next.
- Brain stem herniation follows with loss of consciousness and respiration, often precipitously.

From Leestma, Forensic Neuropathology, 3rd ed., 2014

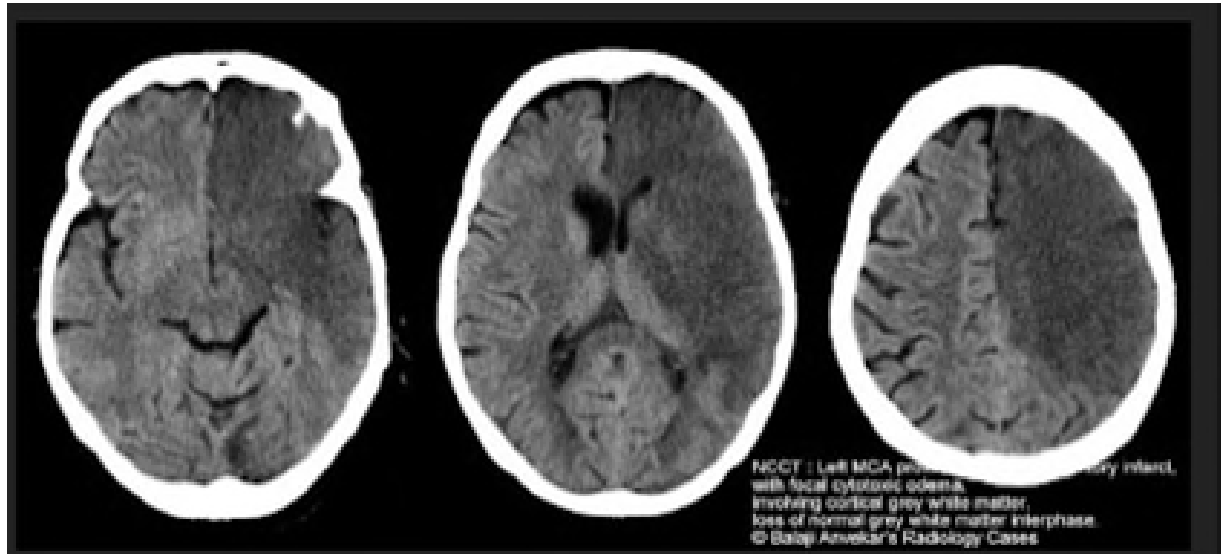
Cellular bases of brain swelling

- Cytotoxic = disruption of cell membrane homeostasis, with cell swelling
  - TBI, stroke
- Vasogenic = disruption of blood-brain barrier (BBB), plasma components entering perivascular space and interstitium
  - Tumors, abscesses (rim-enhancement on contrast imaging), TBI
- Osmotic = disturbance of blood osmolality causing fluid shift to/from interstitium
  - Rapid correction of hyperglycemia, hyponatremia
- Interstitial = pressurized movement of CSF into interstitium
  - Hydrocephalus

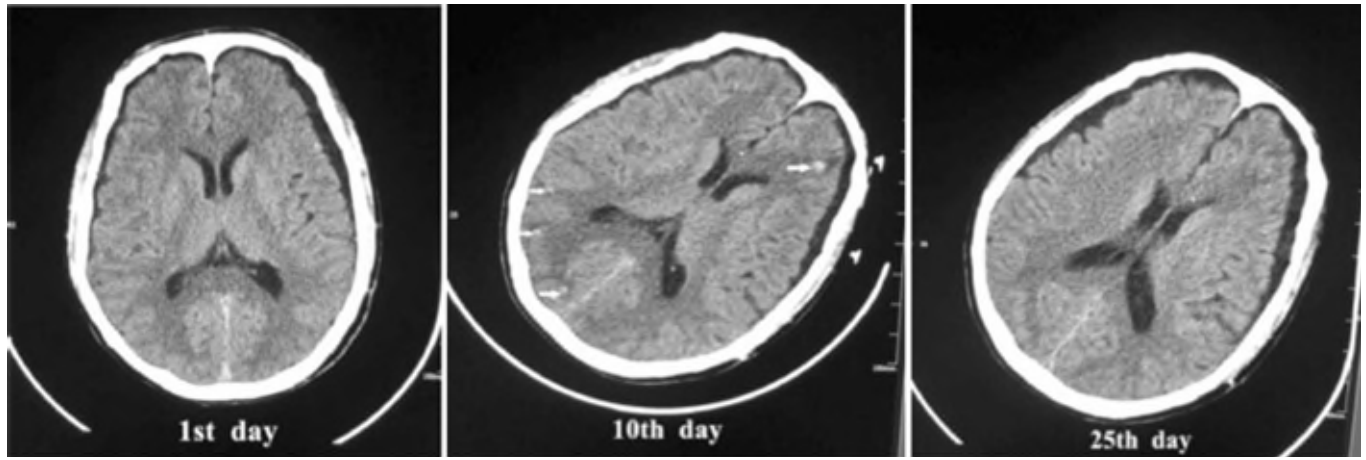

BBB Intact
BBB Intact
Increased BBB Permeability
Cerebral Capillary
Plasma protein i.e albumin
Fluid
Cells ↑ in Size
No ↑ in cell size
↑ volume of extracellular space
Normal
Cytotoxic Edema
Vasogenic Edema
Water movement is tightly controlled. Balance between fluid in extracellular and intracellular spaces.
Failure of the NA+/K+ ATPase pump results in loss of ion homeostatis. Na+ floods into cells and water follows leading to cellular swelling.
Breakdown of the BBB permits the movement of plasma proteins (i.e albumin) into the brain tissue. Water is obligated to follow and accumulates within the extracellular space.
Normal ICP 5-15mmHg
No Change in ICP as no ↑ in brain volume
↑ ICP due to ↑ in brain volume

Cytotoxic edema

- BBB intact, edema due to failure of ATP-dependent ion transport, resulting in cellular retention of water
- Astrocytes involved – gray and WM – poor distinction
- Examples:
  - (Initial) ischemic infarct
  - Toxic conditions (may have WM>GM – intramyelinic edema)



Ischemic infarct



Trimethyltin toxicity

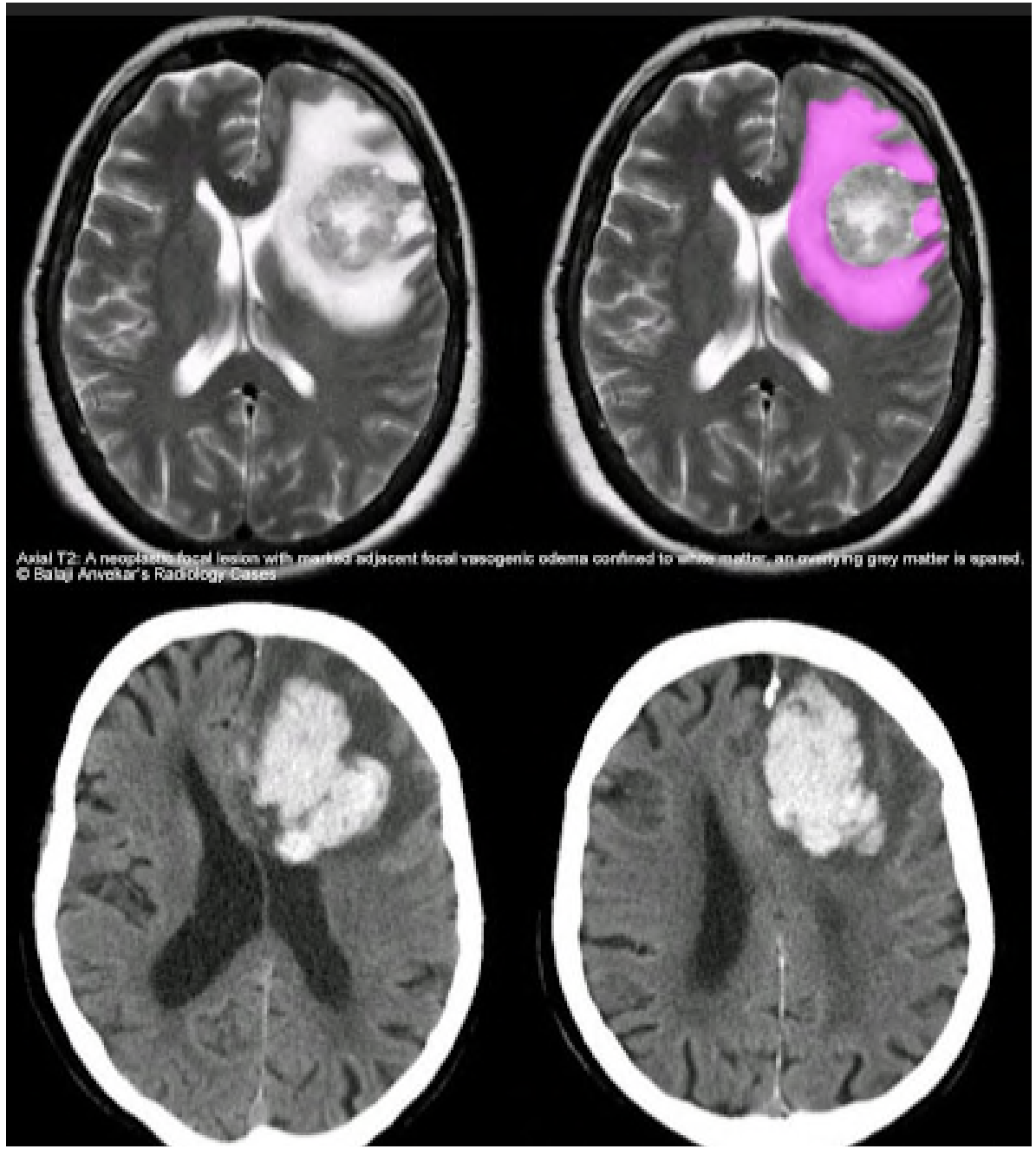
Dr. Balaji Ankevar, http://www.neuroradiologycases.com/2011/.html
https://www.sciencedirect.com/science/article/abs/pii/S0002962921000616

Vasogenic edema

- BBB compromise
  - Physical disruption by arterial hypertension causing direct transmission of pressure to capillary bed, with transudation into extracellular space, WM>GM
  - Examples:
    - Tumor or abscess with vasoactive factor expression (arachidonic acid, excitotoxins, histamine, VEGF)
    - Intracerebral hemorrhage
    - Trauma with bleed
    - PRES

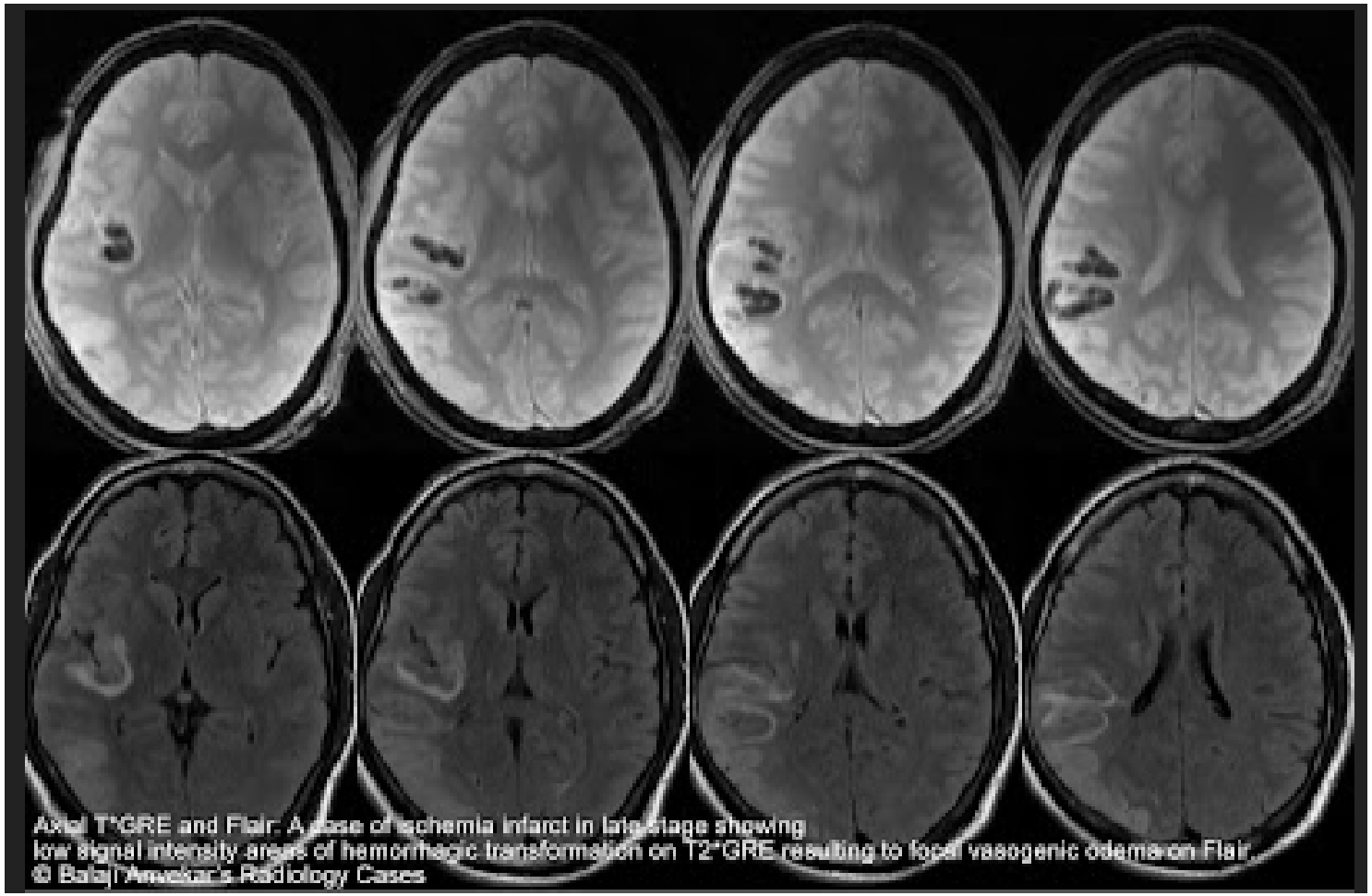
Metastatic tumor

Lobar hemorrhage



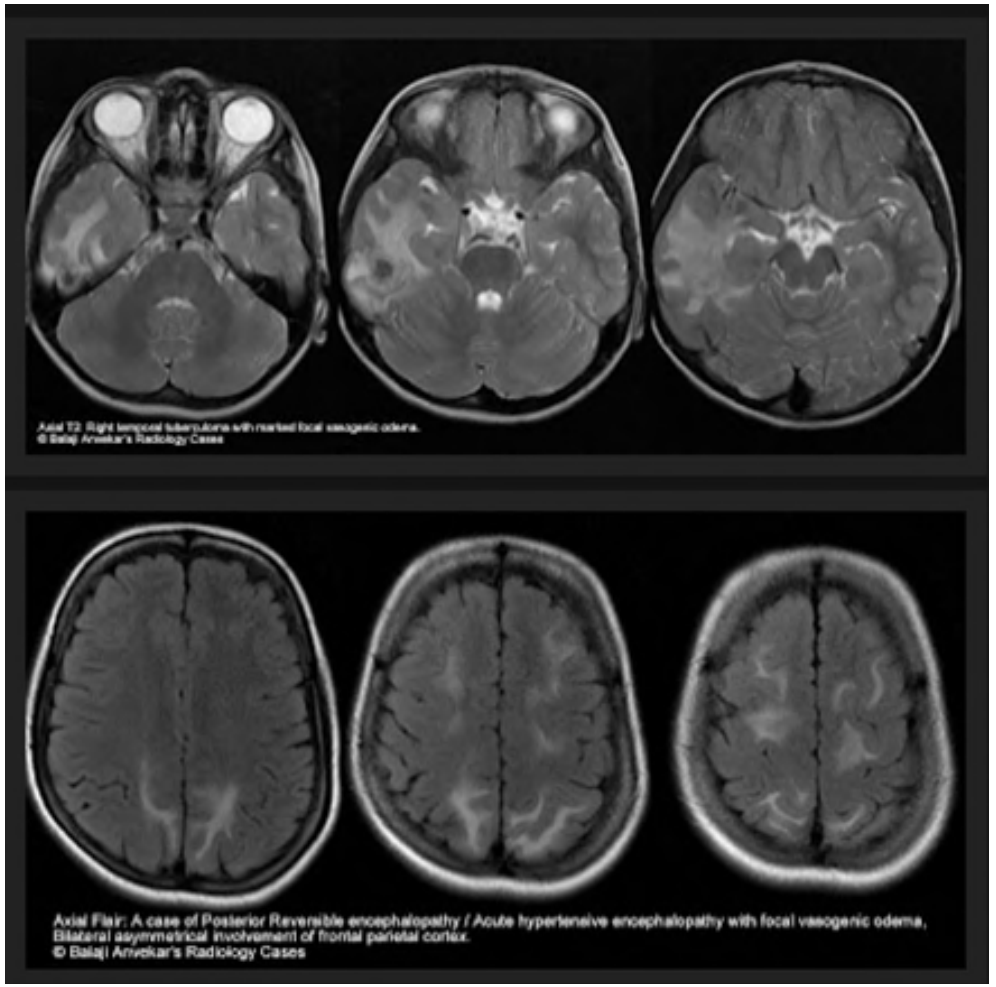
Dr. Balaji Ankevar, http://www.neuroradiologycases.com/2011/.html

Vasogenic edema



Reperfusion hemorrhage complicating ischemic infarct

Tuberculoma

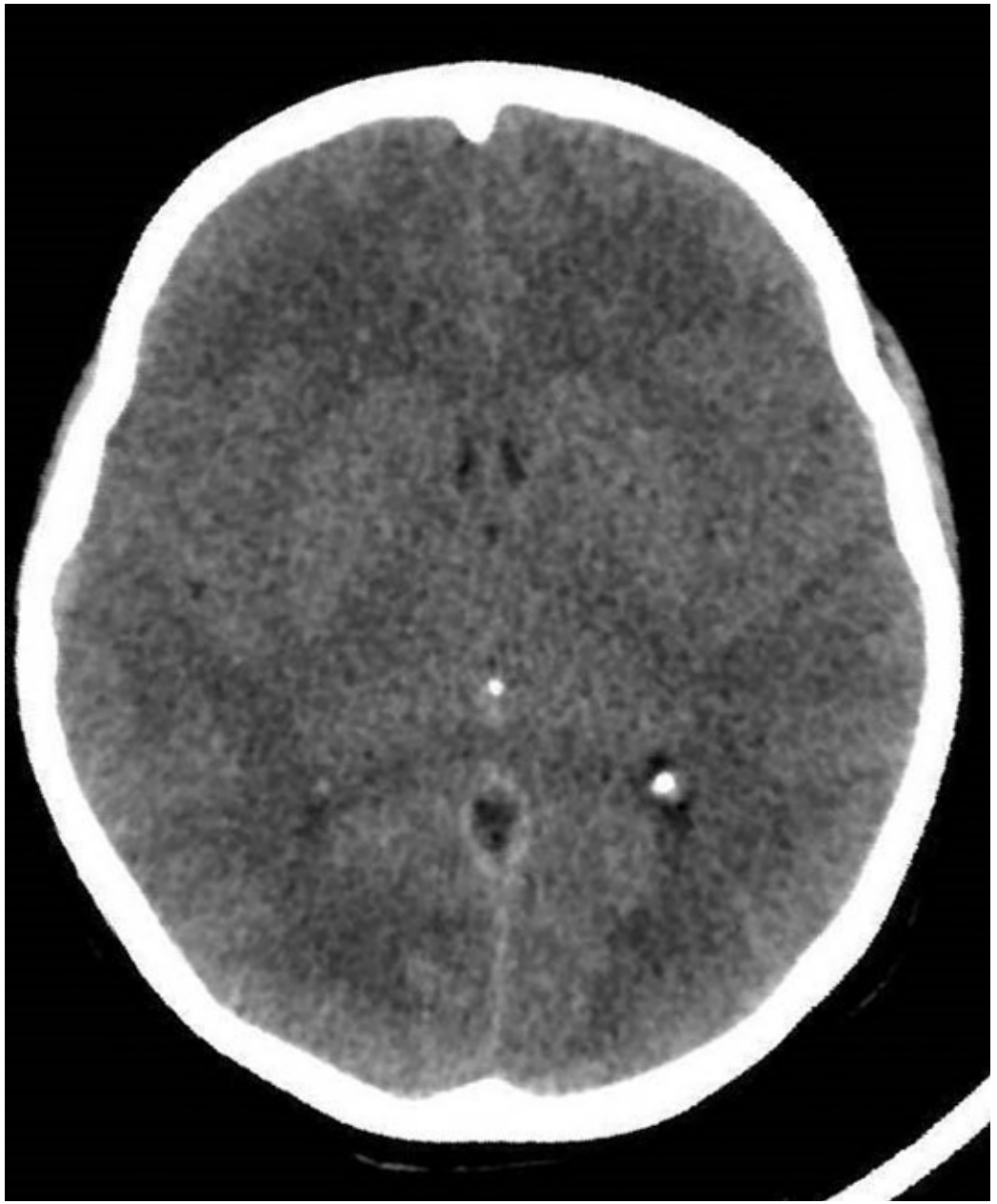


Post. Reversible Encephalopathy Syndrome

Dr. Balaji Ankevar, http://www.neuroradiologycases.com/2011/.html

Osmotic edema

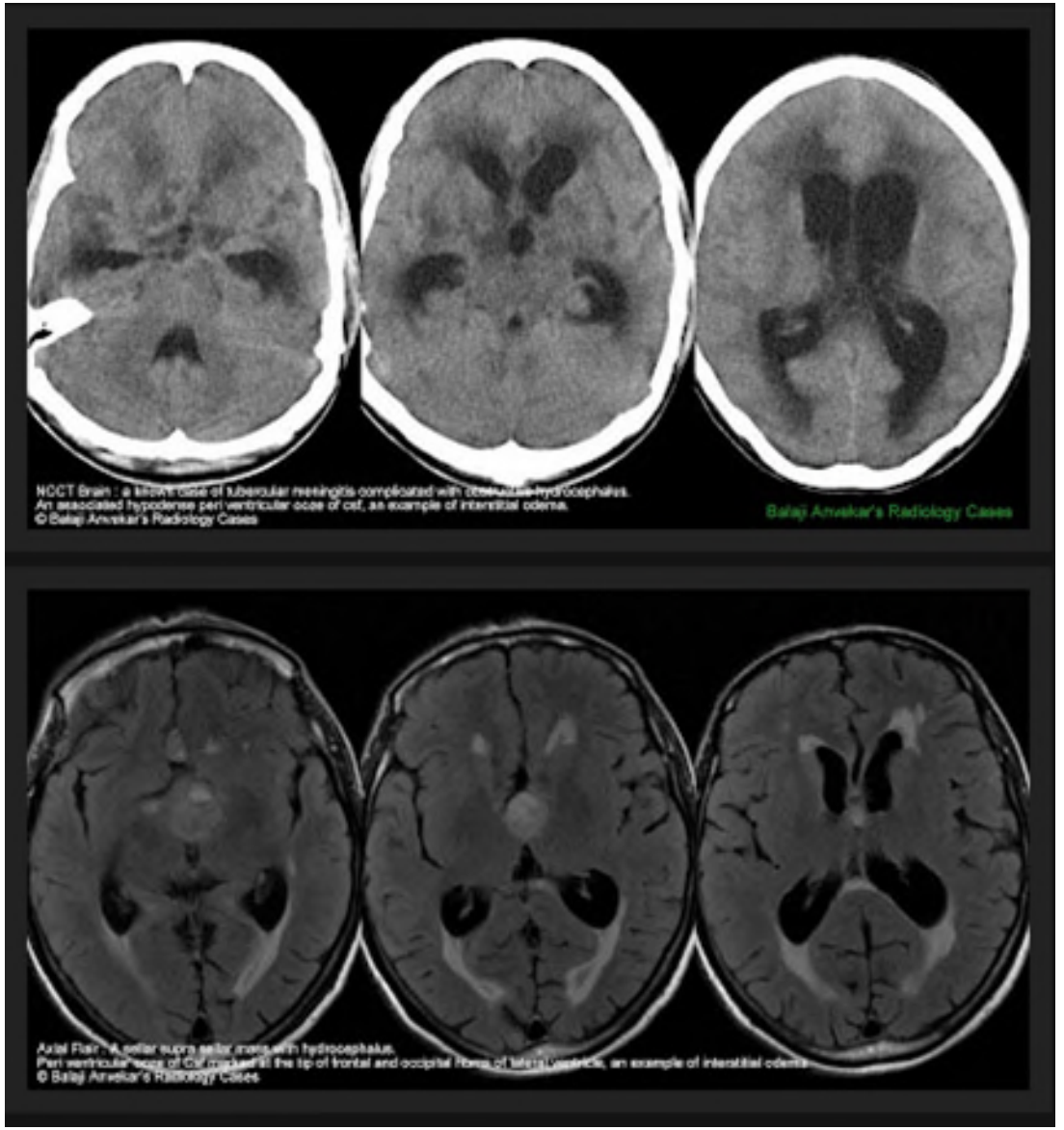
- CSF and extracellular brain fluid has slightly lower osmolality than plasma. When plasma is diluted (excessive water intake, SIADH, hemodialysis, rapid glucose lowering in hyperglycemic non-ketotic acidosis), water enters interstitium
- Diffuse, with relative preservation of G-W distinction



https://radiopaedia.org/cases/cerebral-edema-due-to-severe-hyponatremia

Interstitial edema

- Obstructive hydrocephalus, compromise of CSF-brain barrier, transependymal flow of CSF to extracellular spaces, especially in periventricular WM
- (protein low in transudate)

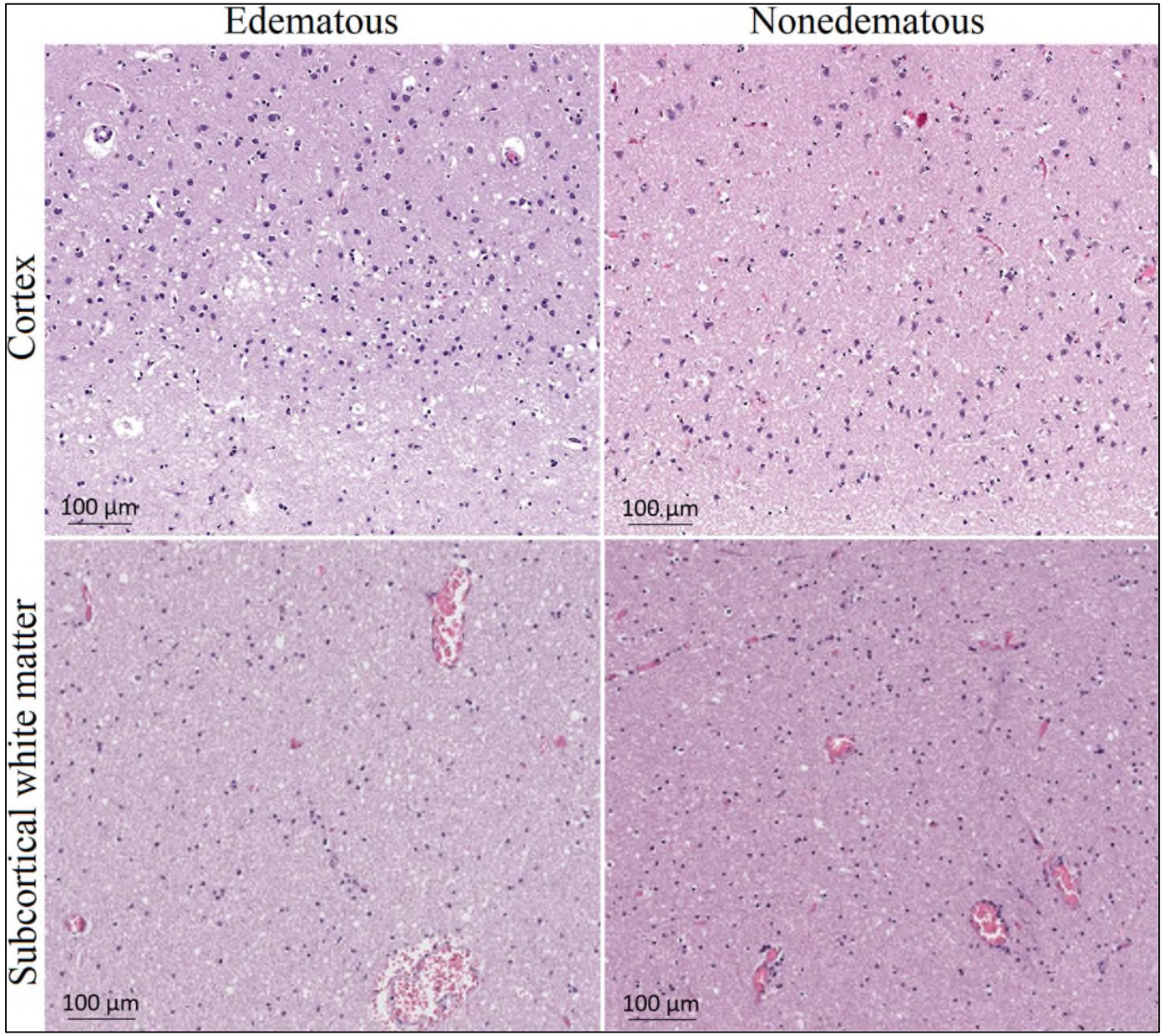


Dr. Balaji Ankevar, http://www.neuroradiologycases.com/2011/.html

Edema – microscopy

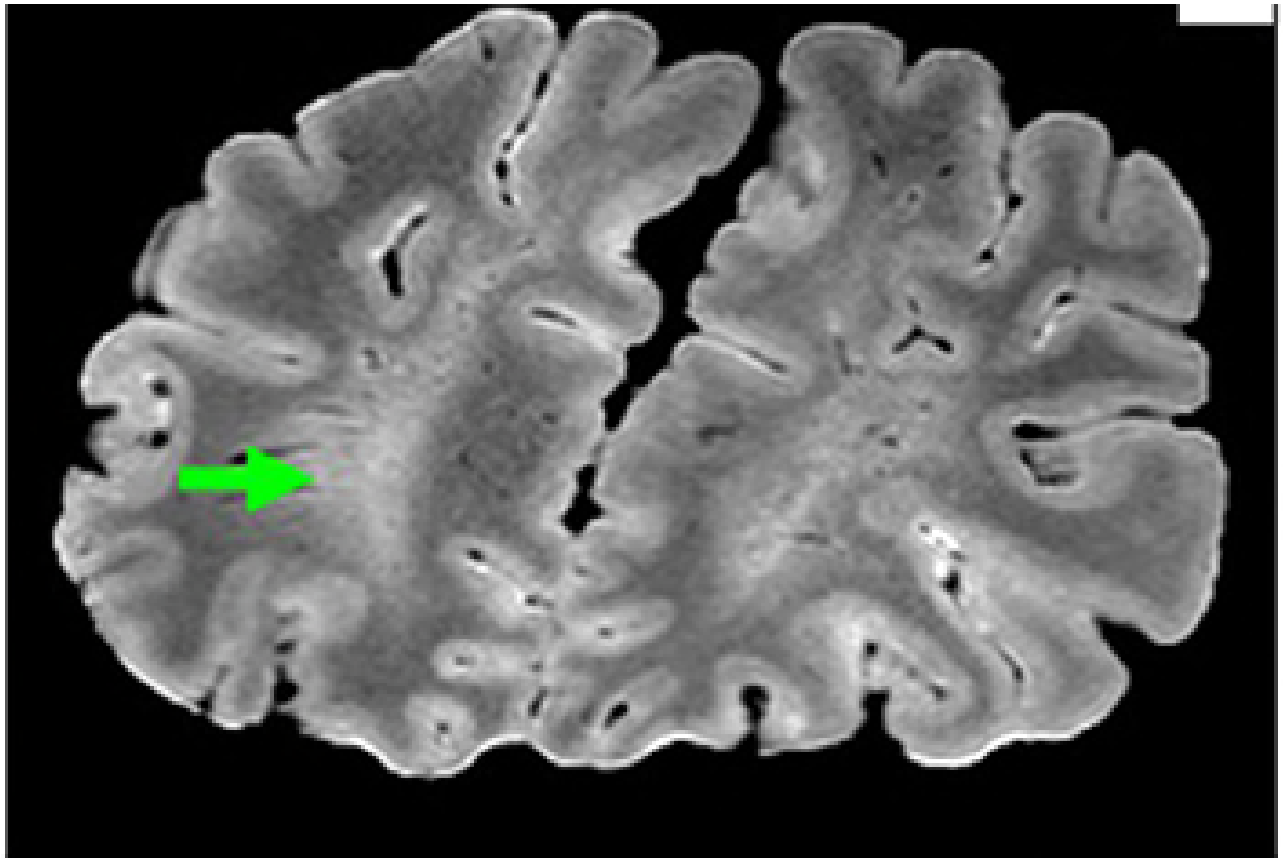
- Pallor of myelin
- Distention of perivascular and pericellular spaces
- Sieve-like appearance of myelinated areas
- Rarefaction of subpial spaces
- Vacuolar appearance of gray matter neuropil
- Pools of protein-rich fluid

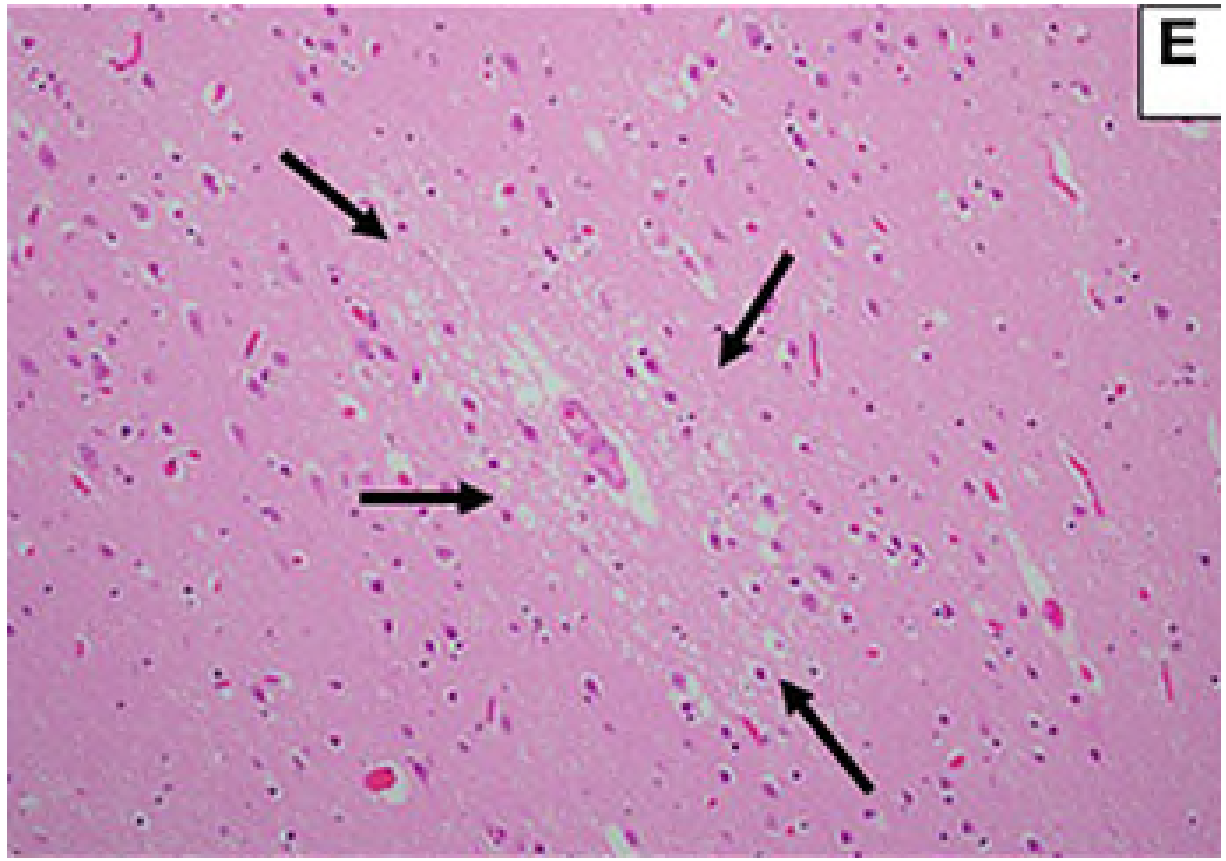
Often, poor distinction among edema subtypes



Bauer et al, 2021

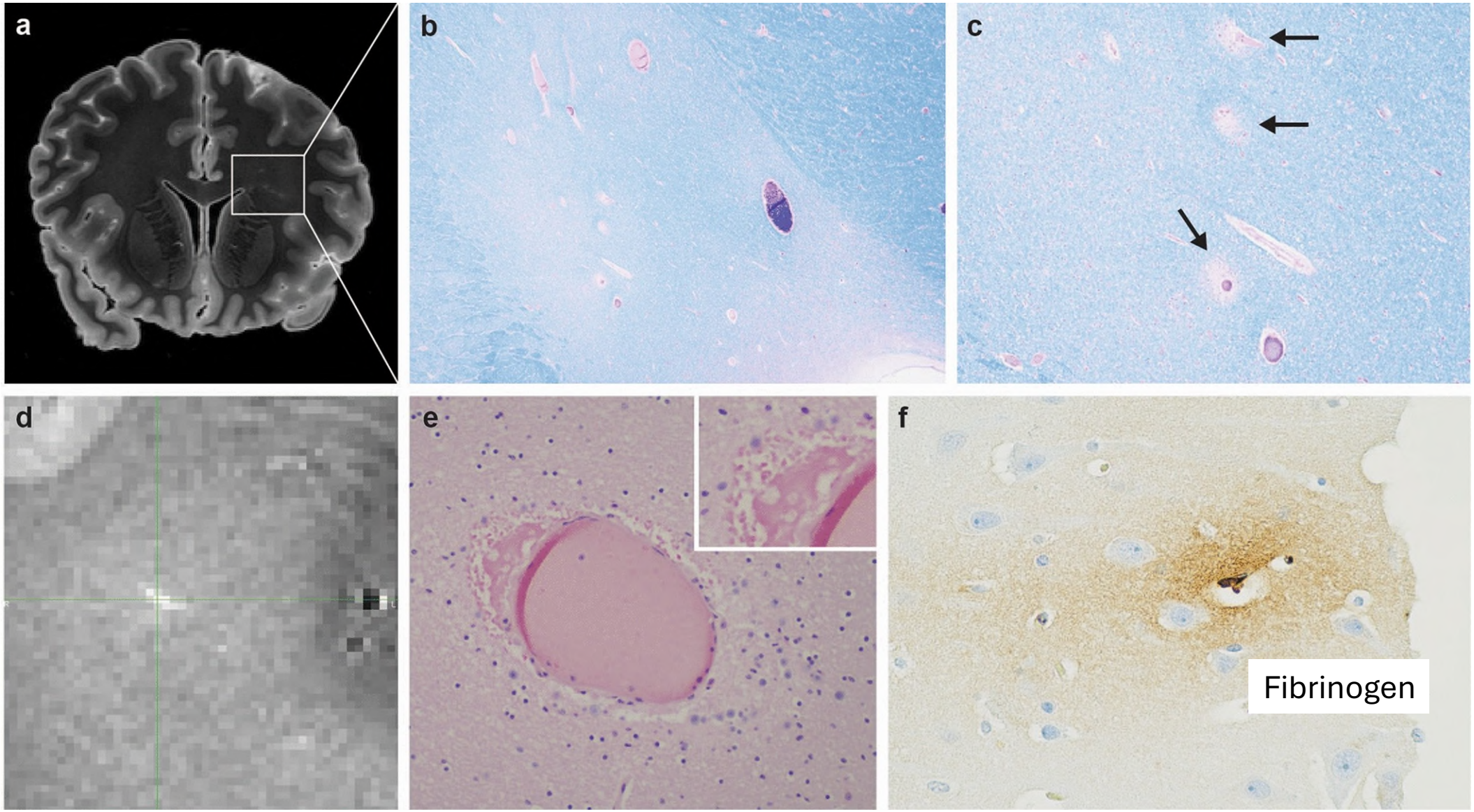
Edema - microscopy





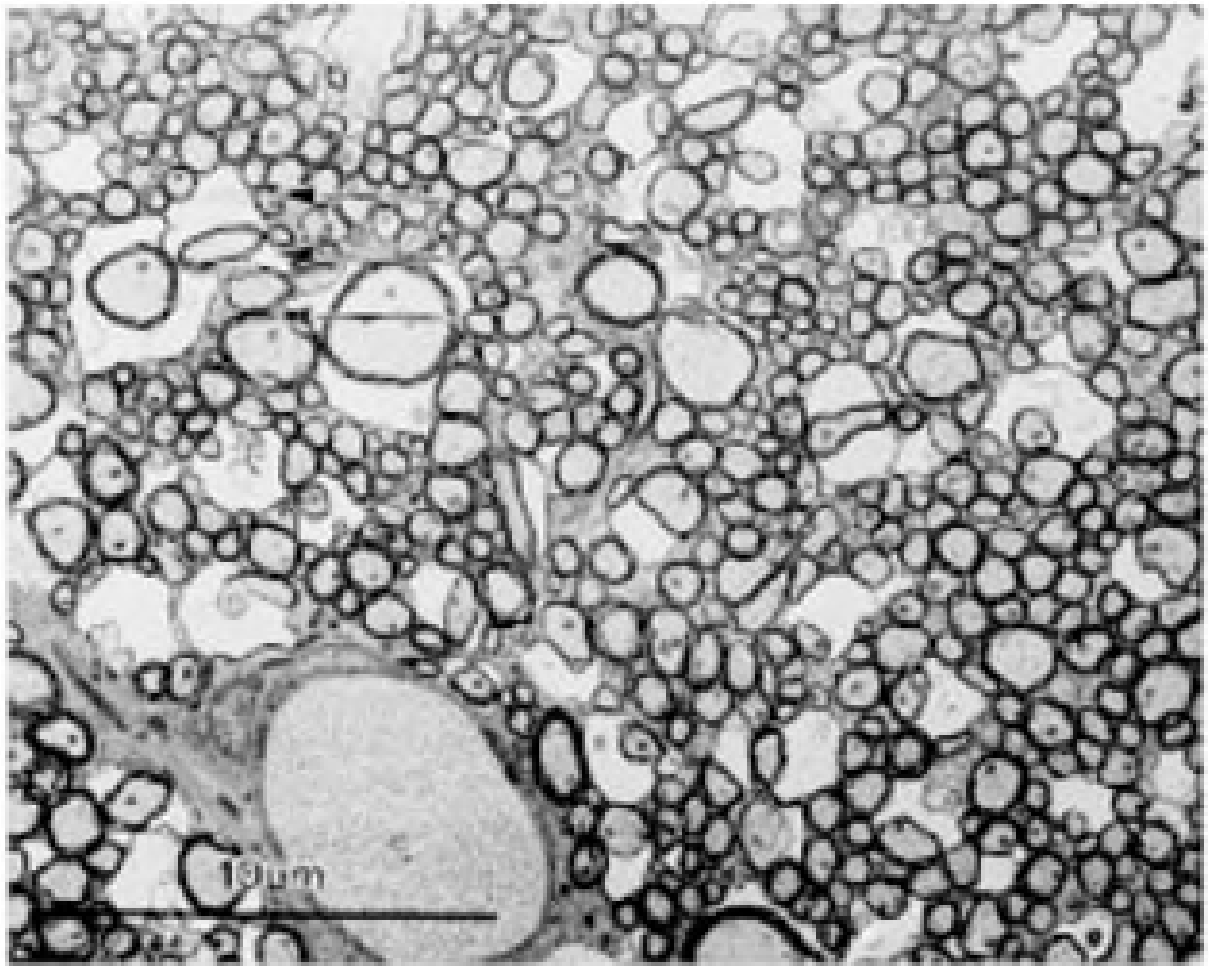
Stram et al, 2022

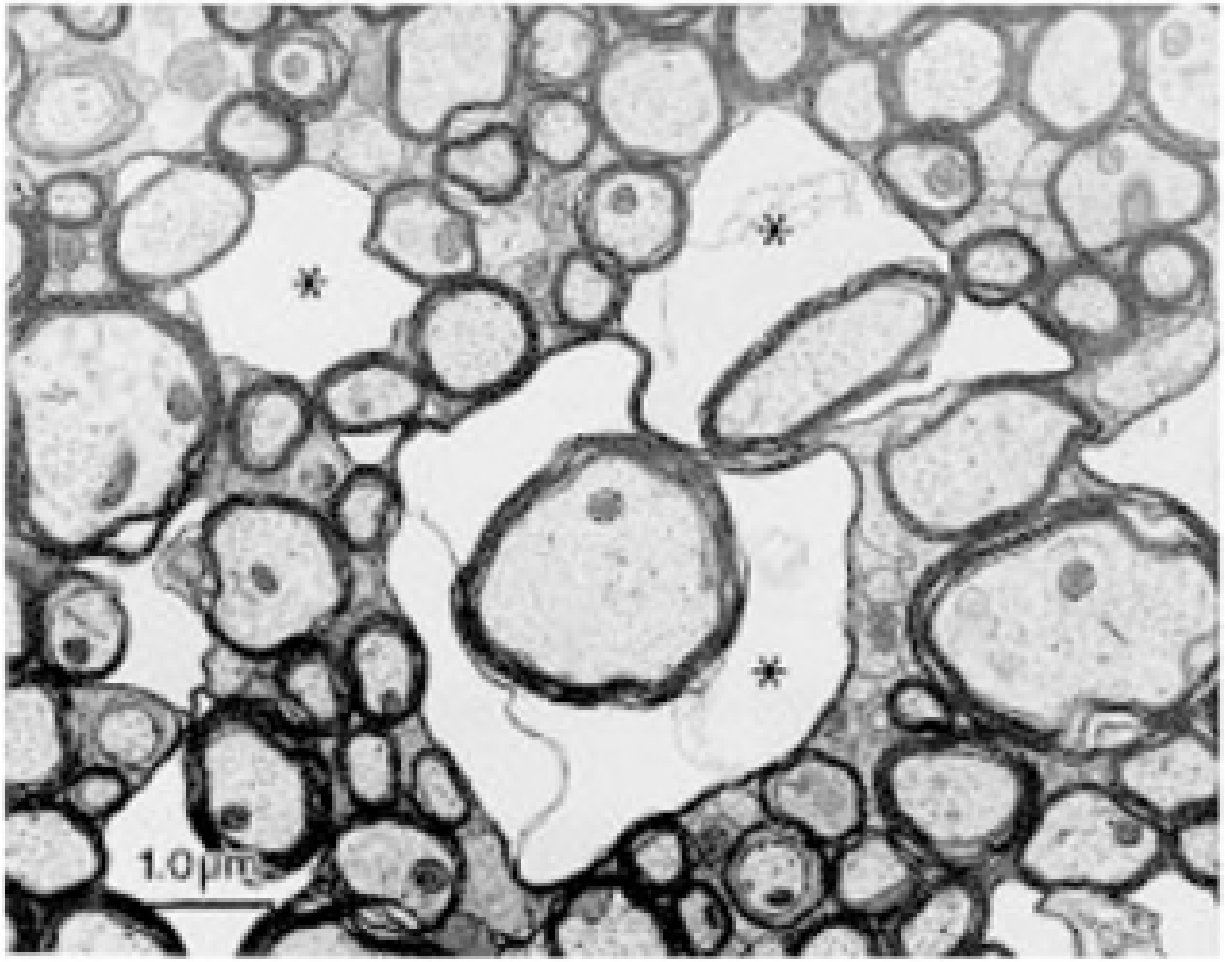
BBB compromise – neuroimaging/path correlation



Dams-O'Connor et al, 2023

Edema – electron microscopy *(triethyltin toxicity)*

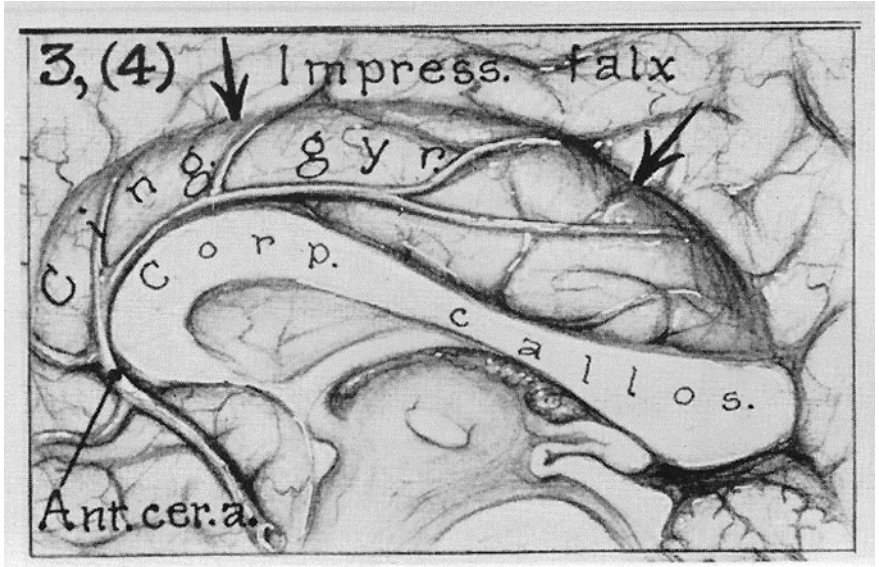


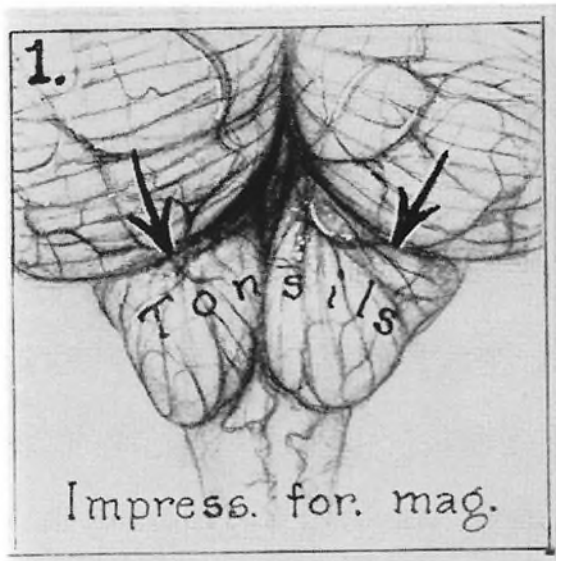


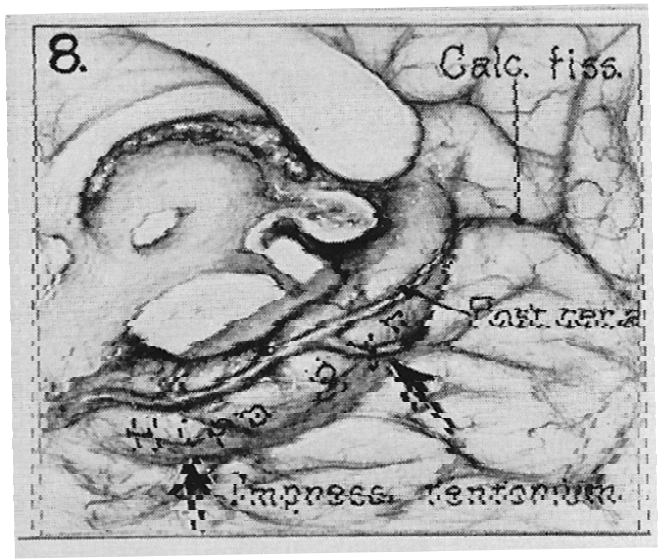
https://www.sciencedirect.com/topics/pharmacology-toxicology-and-pharmaceutical-science/triethyltin

Patterns of herniation

- Subfalcine (cingulate gyrus)
- Transtentorial (uncinate process)
- Transforaminal (cerebellar tonsillar)



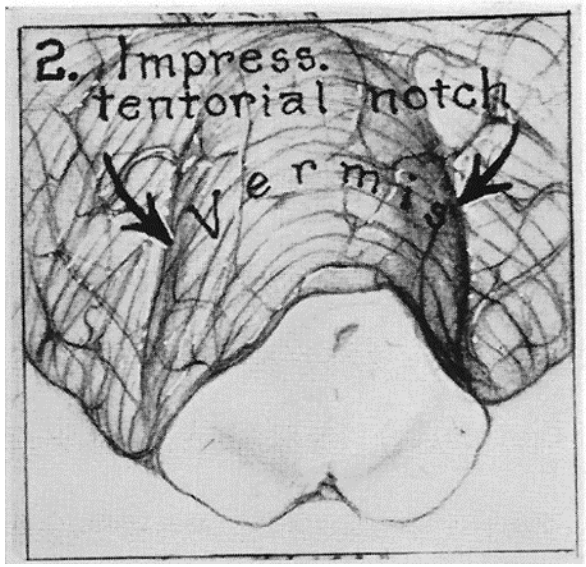


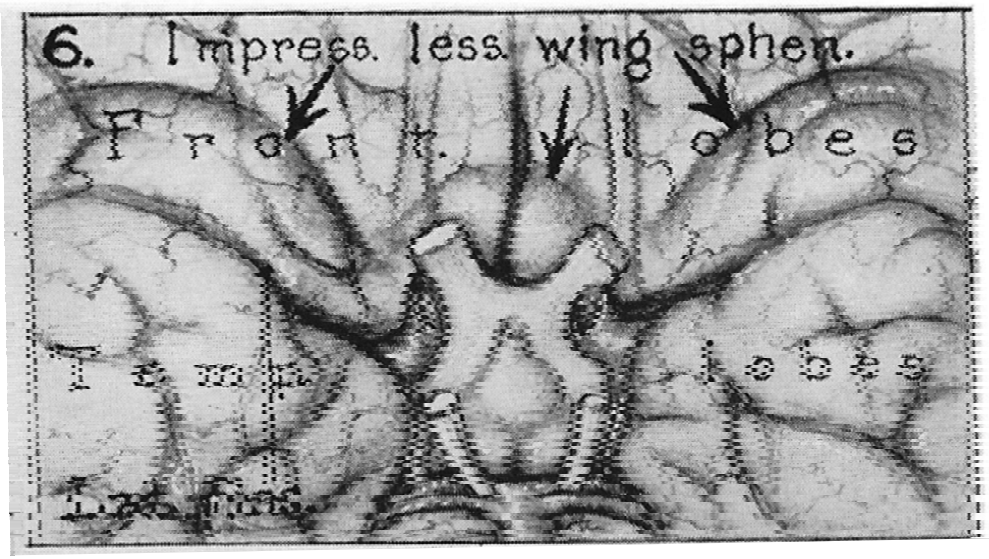


From Finney and Walker, Transtentorial Herniation, 1962.

Patterns of herniation (cont'd)

- Lesser sphenoidal (posterior orbital frontal)
- Supratentorial (superior cerebellar)

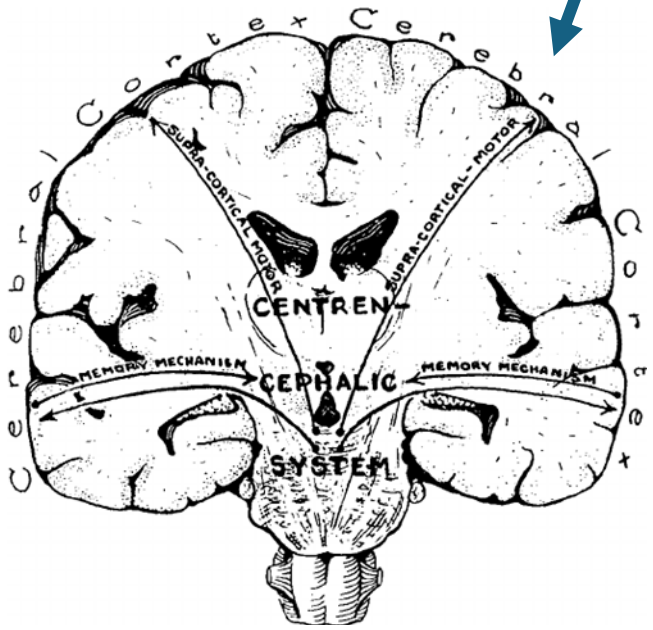


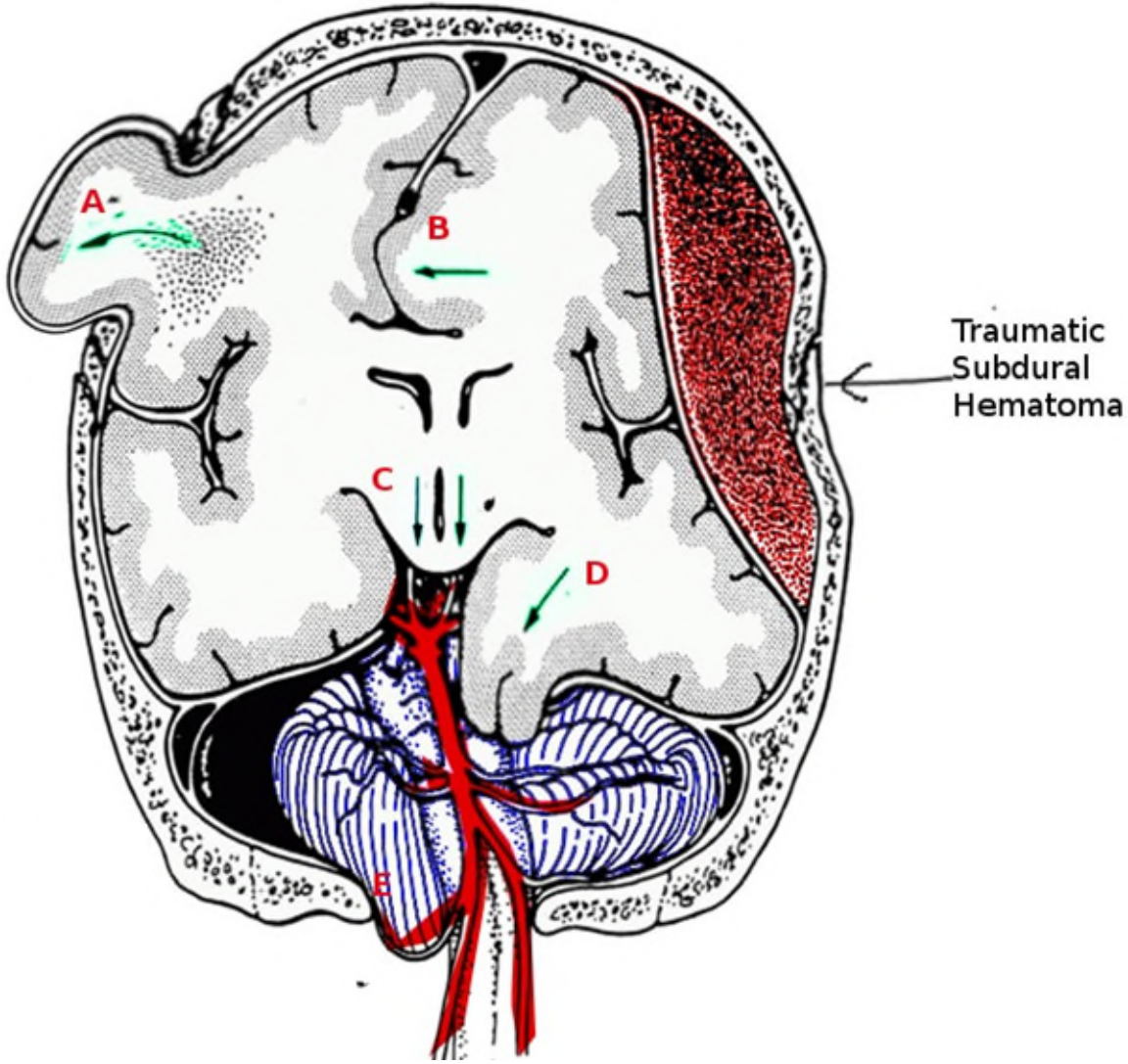


From Finney and Walker, Transtentorial Herniation, 1962.

Patterns of herniation (cont'd)

- External (*fungus cerebri*)
- Centrencephalic





Centrencephalic herniation

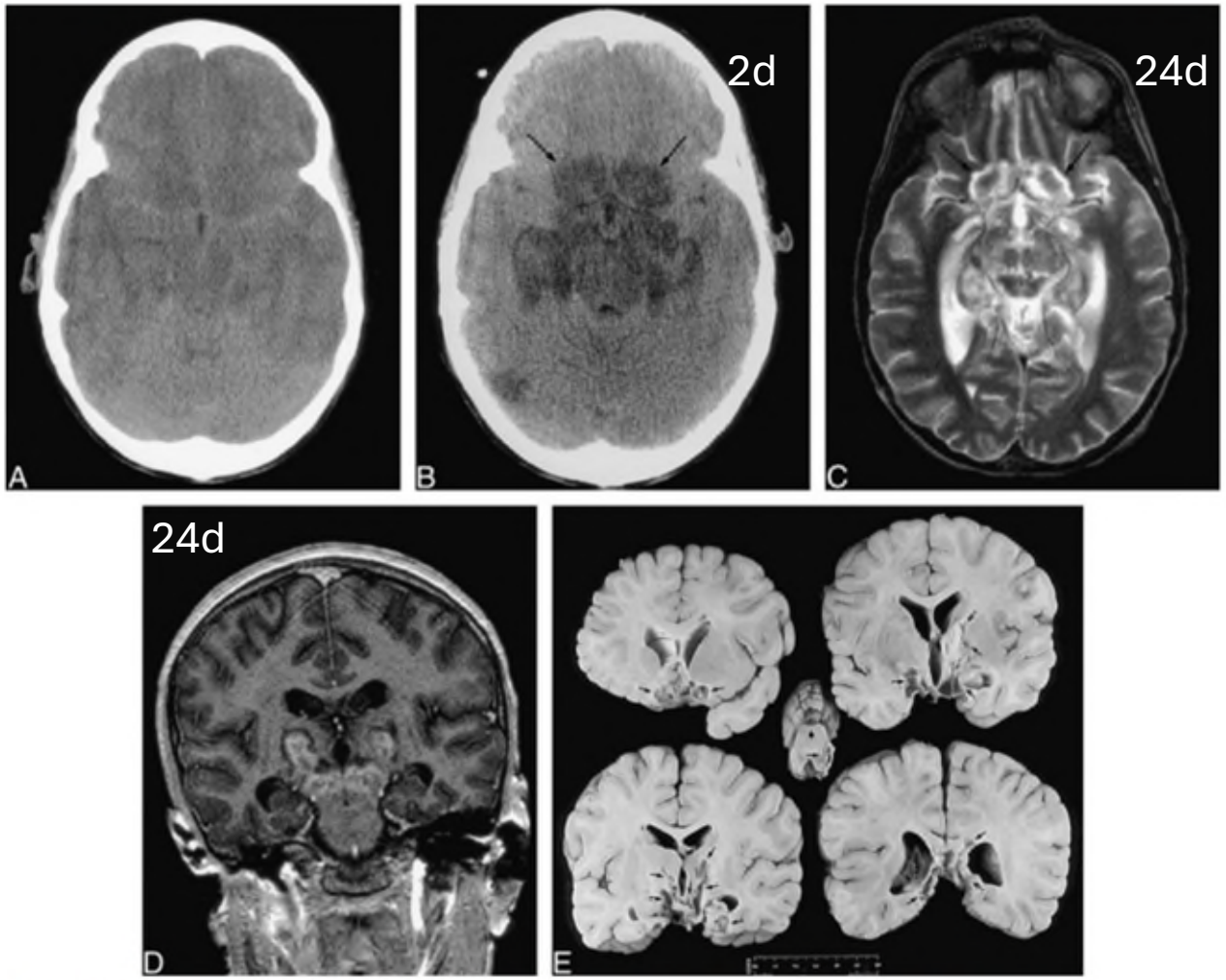


FIG 1. Nine-year-old boy with diffuse cerebral edema and central herniation secondary to treatment of DKA.
A, Axial noncontrast CT scan shows diffuse cerebral edema with effacement of sulci and basal cisterns.
B, Axial noncontrast CT scan obtained 2 days after *A* shows marked low-density infarcts in the gyrus recti and medial orbital gyri (*arrows*), globus pallidi, hippocampi/parahippocampal gyri, hypothalamus, midbrain, and posterior right temporal lobe.
C–D, Axial noncontrast T2-weighted (4000/105/1) (*C*) and coronal postcontrast T1-weighted SPGR (14.4/3.7/1) (*D*) MR images obtained 24 days after *A* show cavitary infarcts in the gyrus recti and medial orbital gyri (*arrows*), medial temporal lobes, midbrain and thalami. Enhancement is present within the thalamic and midbrain lesions. There is diffuse cerebral atrophy.
E, Coronal gross pathologic sections through cerebrum and axial section through midbrain show remote infarctions involving the basilar cerebral structures, basal ganglia, thalami, hippocampi and midbrain (*arrows*).

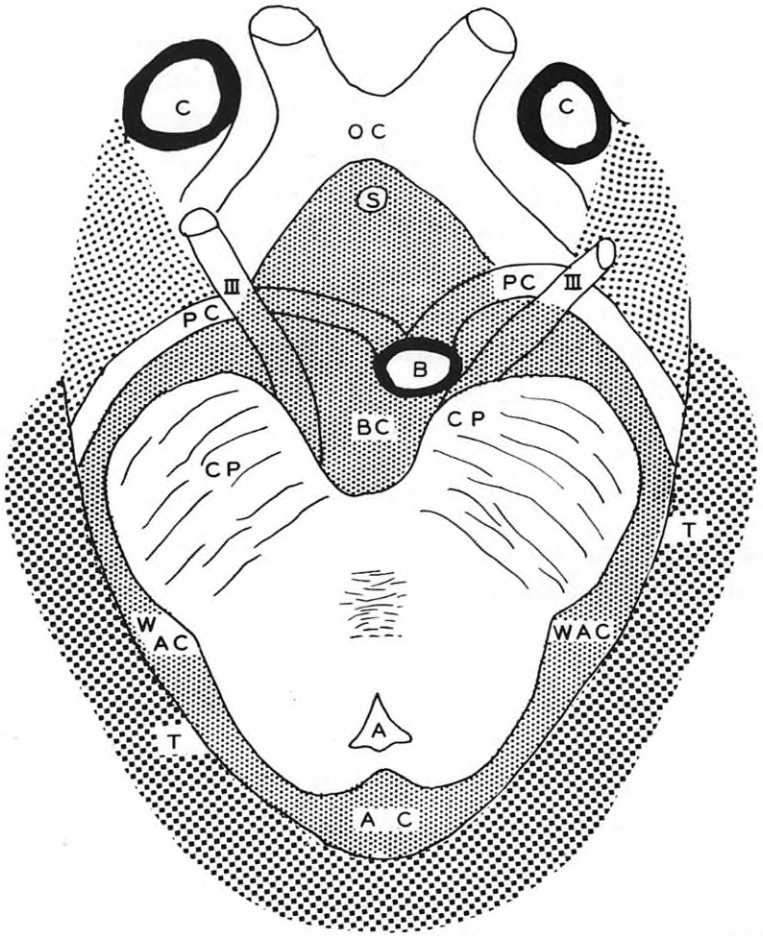


Fig. 7. Diagrammatic sketch to indicate the position and extent of the ambient and basal cisterns, as seen looking at the tentorium from below. A—aqueduct of Sylvius, A.C.—ambient cistern, B—basilar artery, B.C.—basal cistern, C—internal carotid artery, C.P.—cerebral peduncle, O.C.—optic chiasm, P.C.—posterior cerebral artery, S—stalk of the pituitary, T—tentorium cerebelli, W.A.C.—wings of the ambient cistern.

From Finney and Walker, Transtentorial Herniation, 1962.

Examples of herniation

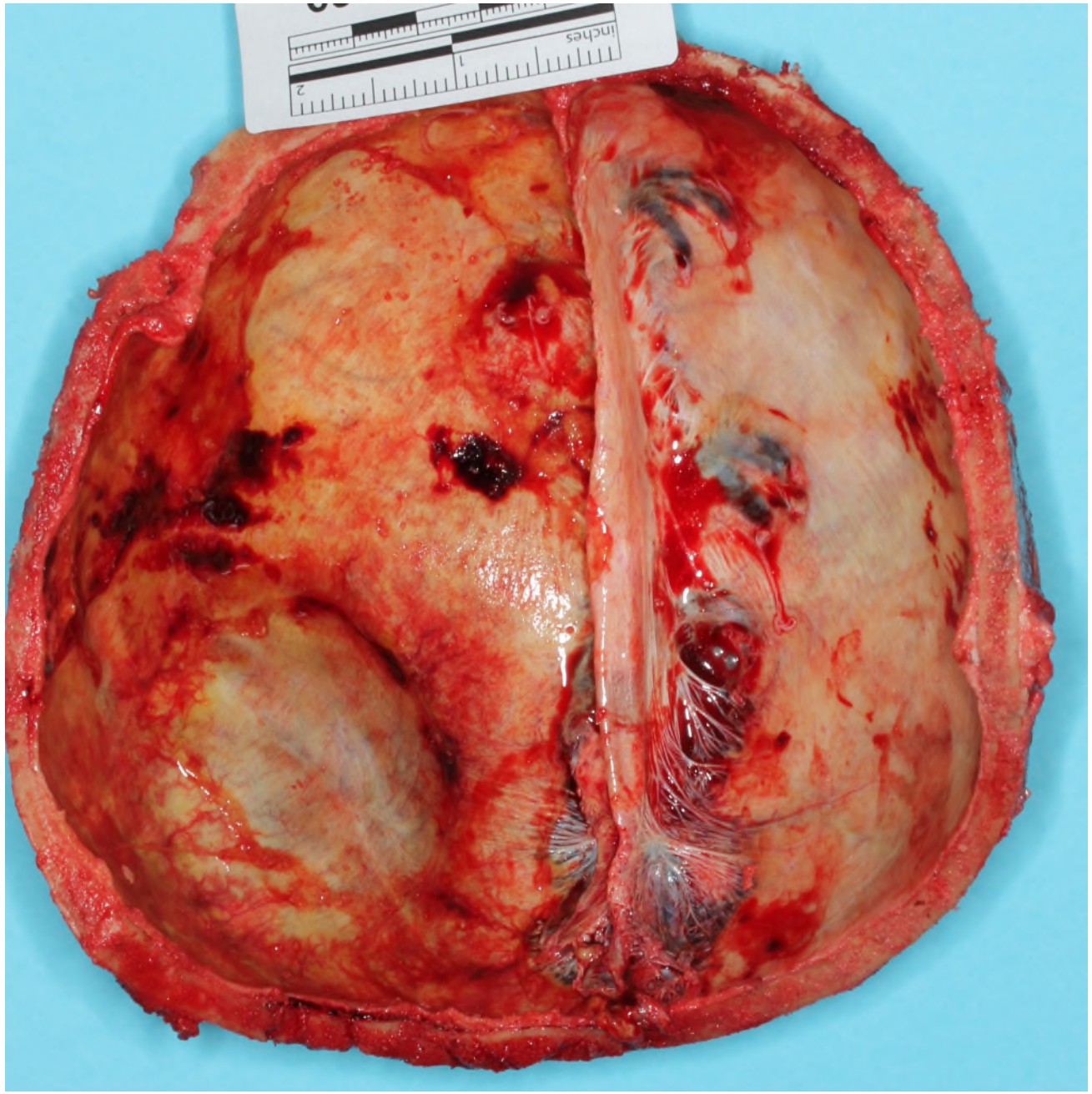
- Acute and subacute – including pediatric
- Chronic
- S/P interventions
  - External ventricular drain
  - Craniectomy

- Supervening processes
  - Secondary compression infarcts, reperfusion hemorrhage
  - “Herniation contusions”, “Kernohan’s Notch Phenomenon”
  - Brain death changes

Cases 1-5: Acute and subacute TBI with hematomas (EDH, SDH, contusion hematomas) and secondary changes

- “50yo ejected from car in MVA”

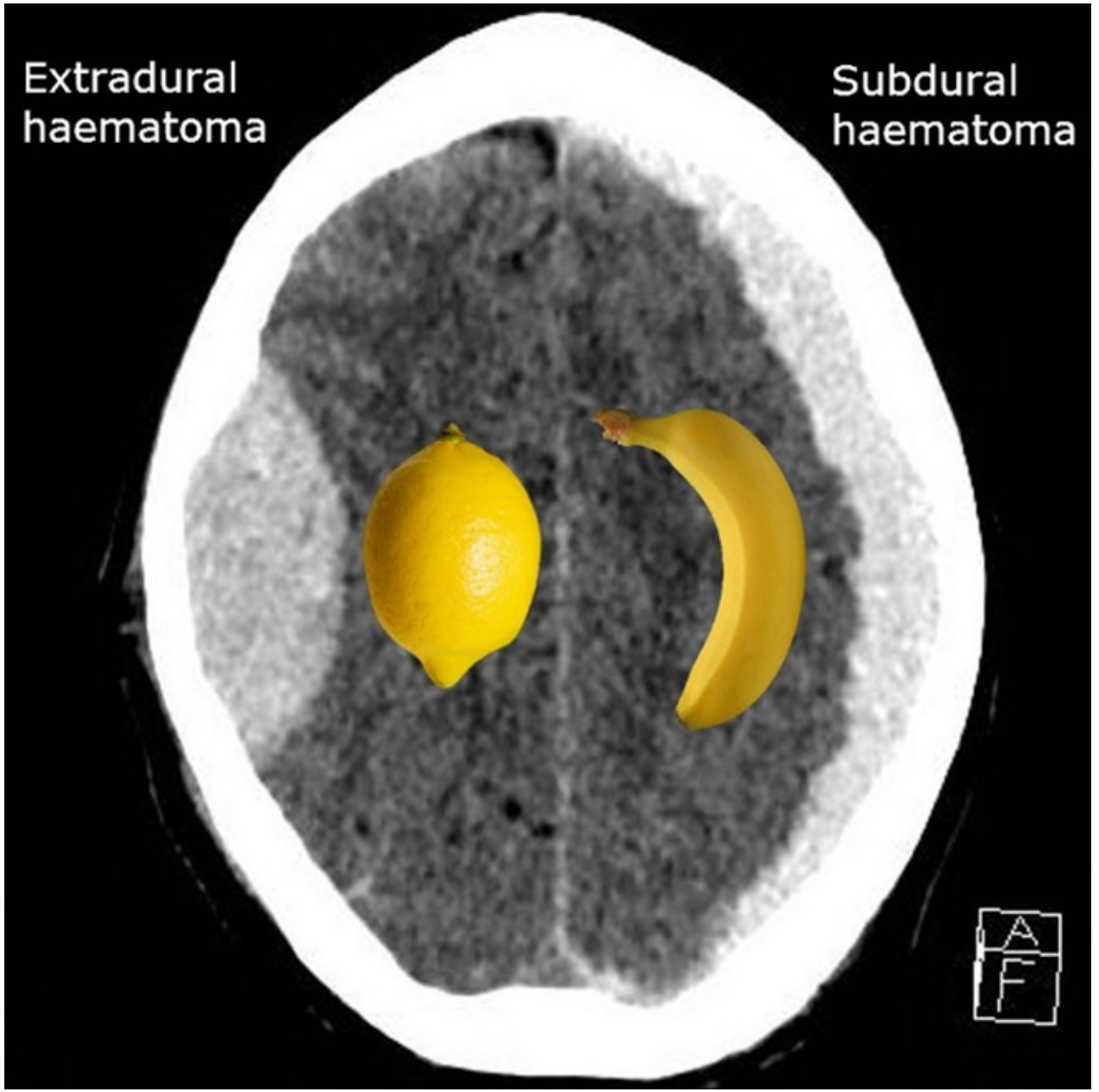
Case 1: R parietal fracture with EDH



FNPDx:

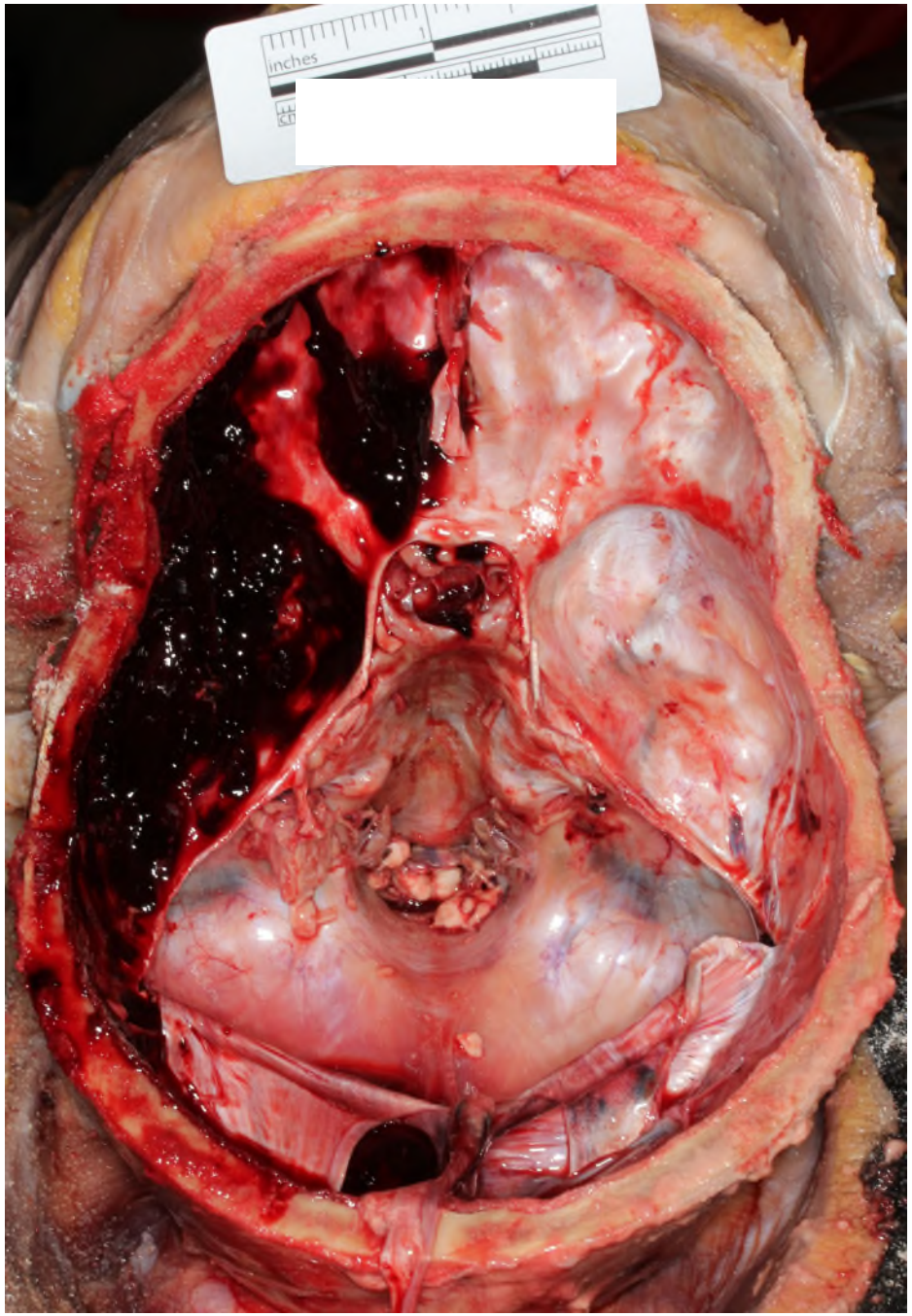
- Blunt impact injury of head, acute, with:
  - R Parietal fracture and epidural hematoma
  - (not shown)
    - Right-to-left midline shift
      - R subfalcine and transtentorial hernias
    - Secondary brainstem (Duret) hemorrhage

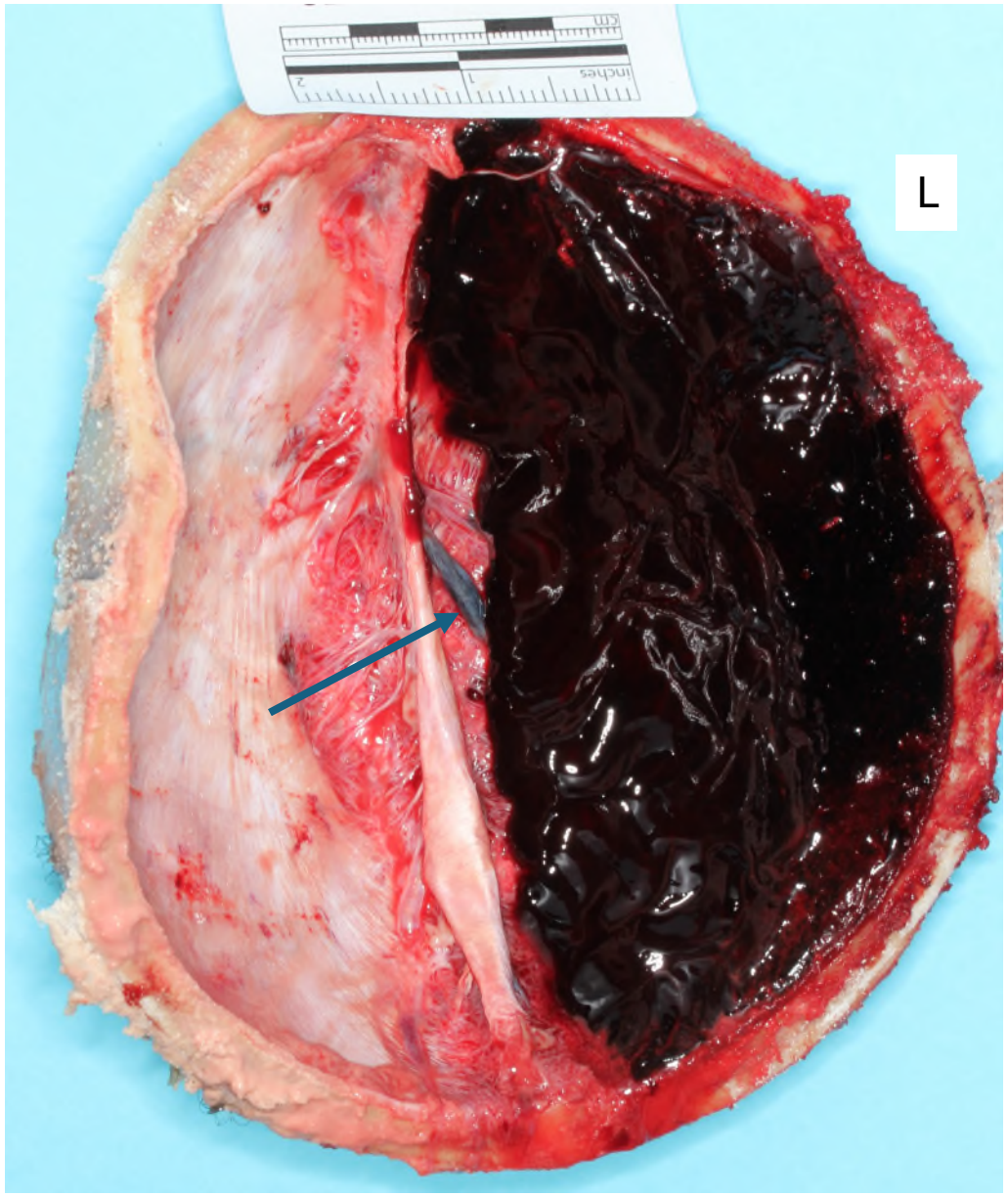
(Case 1)



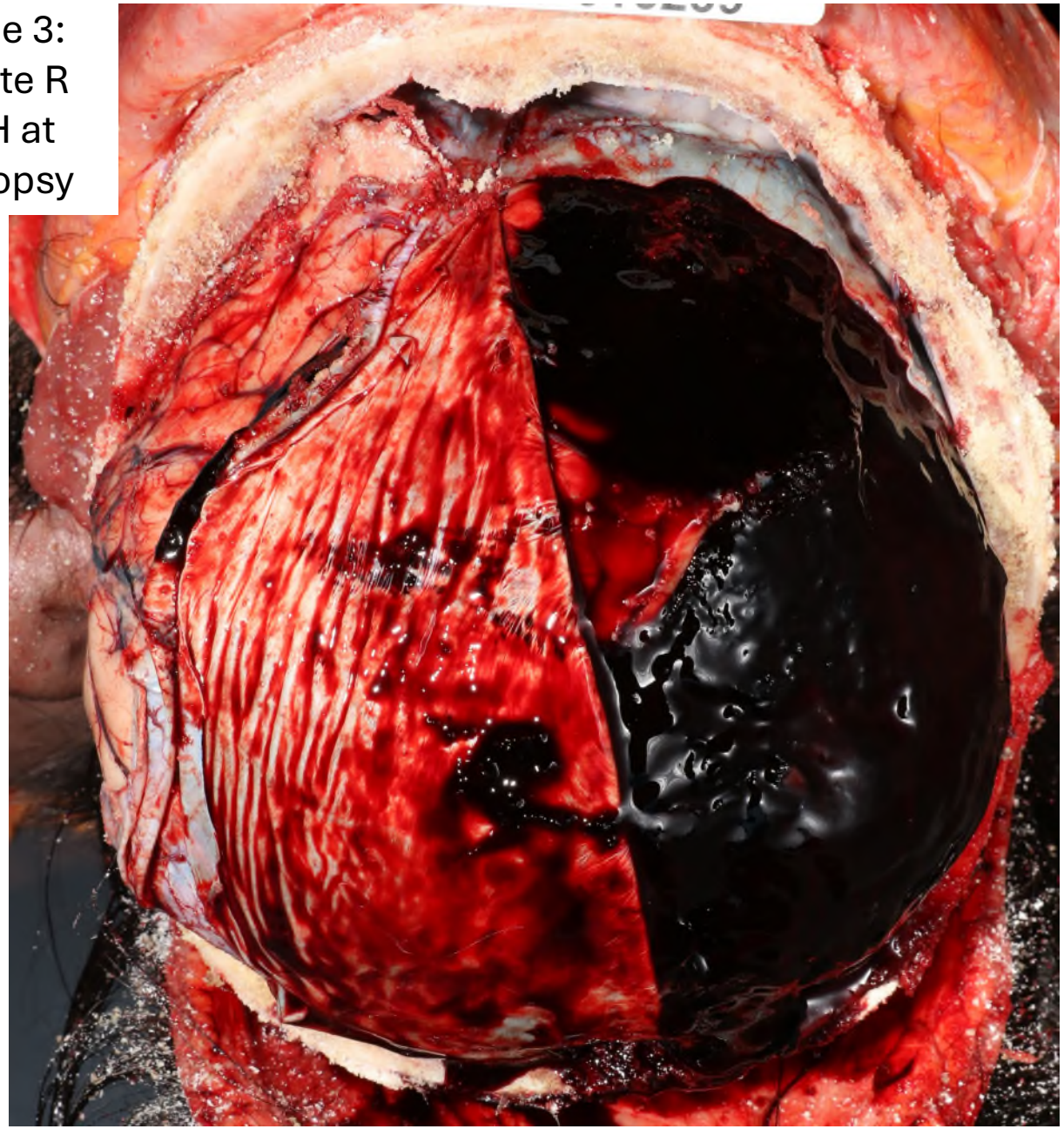
Radiopaedia

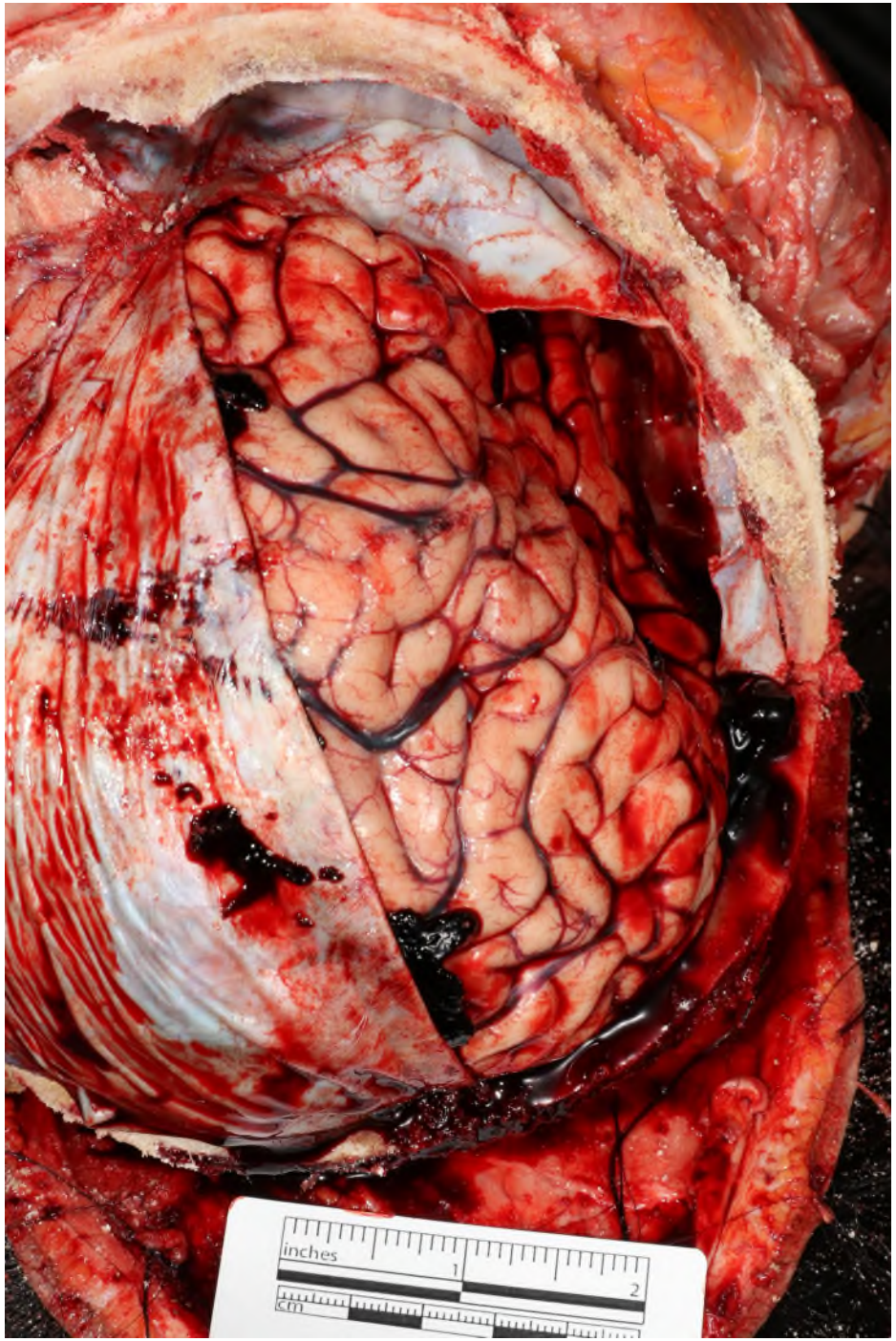
Case 2: Acute L SDH at autopsy



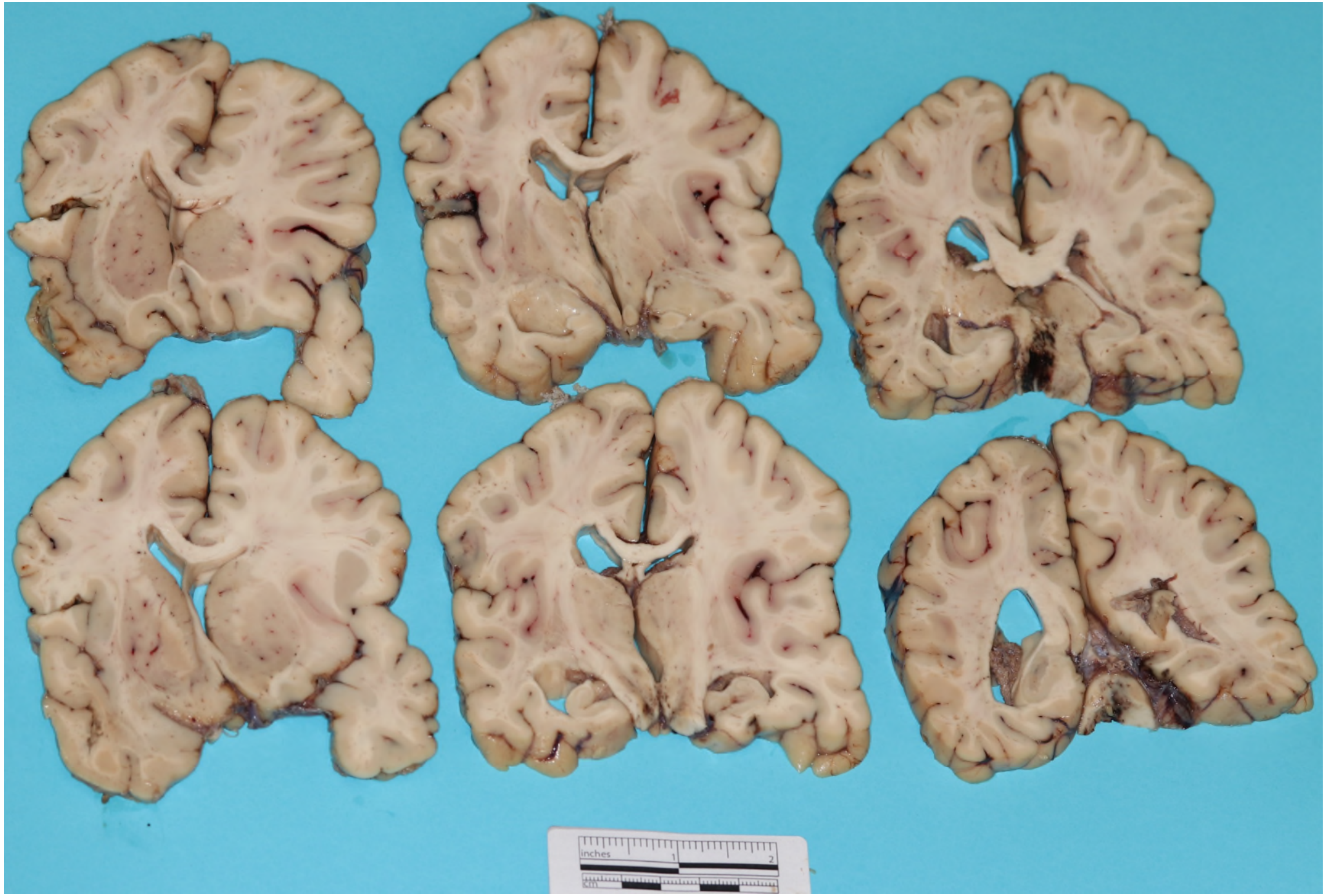


Case 3: Acute R SDH at autopsy





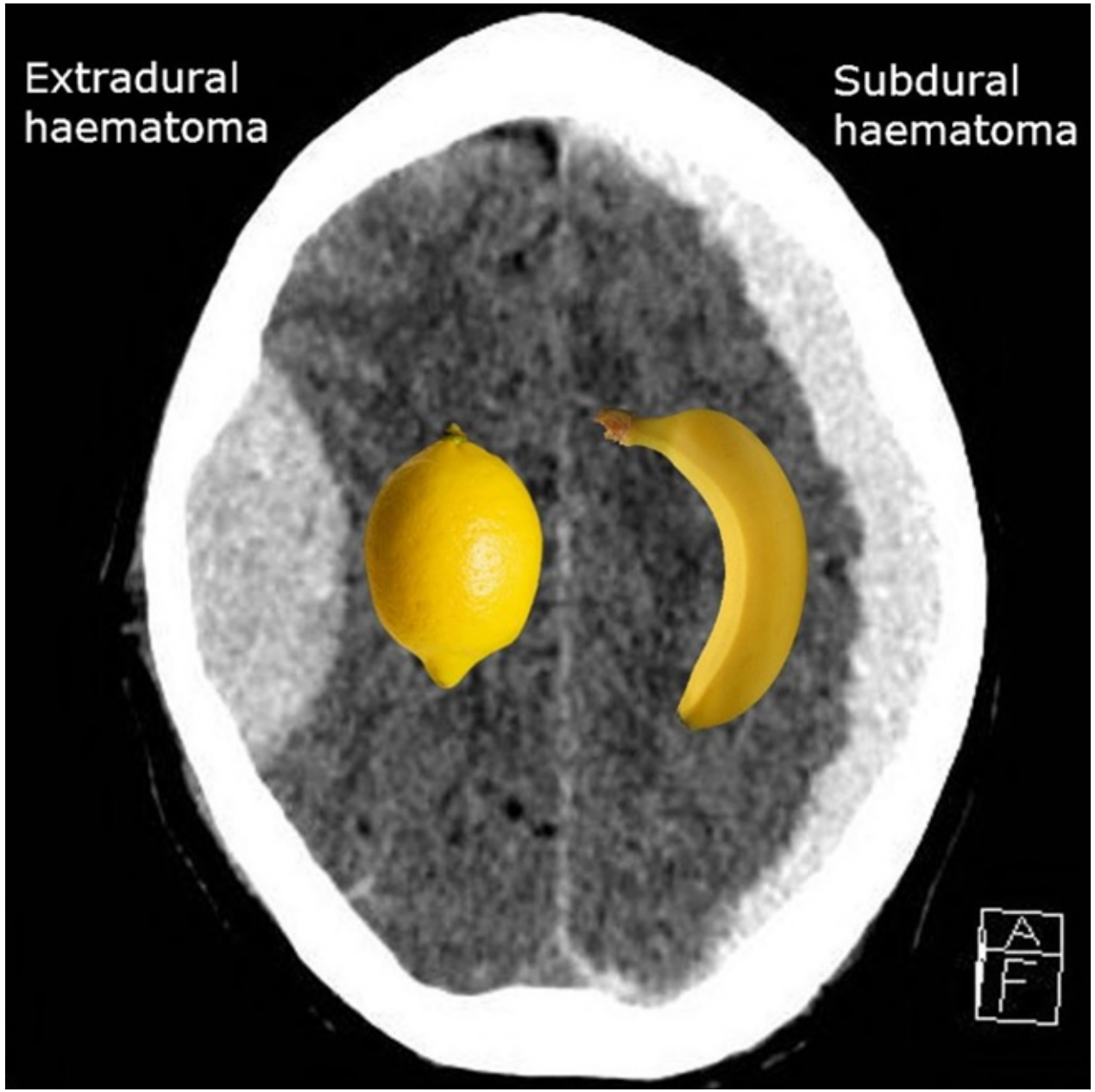
Case 3:
Acute R SDH
after fixation



FNPDx:

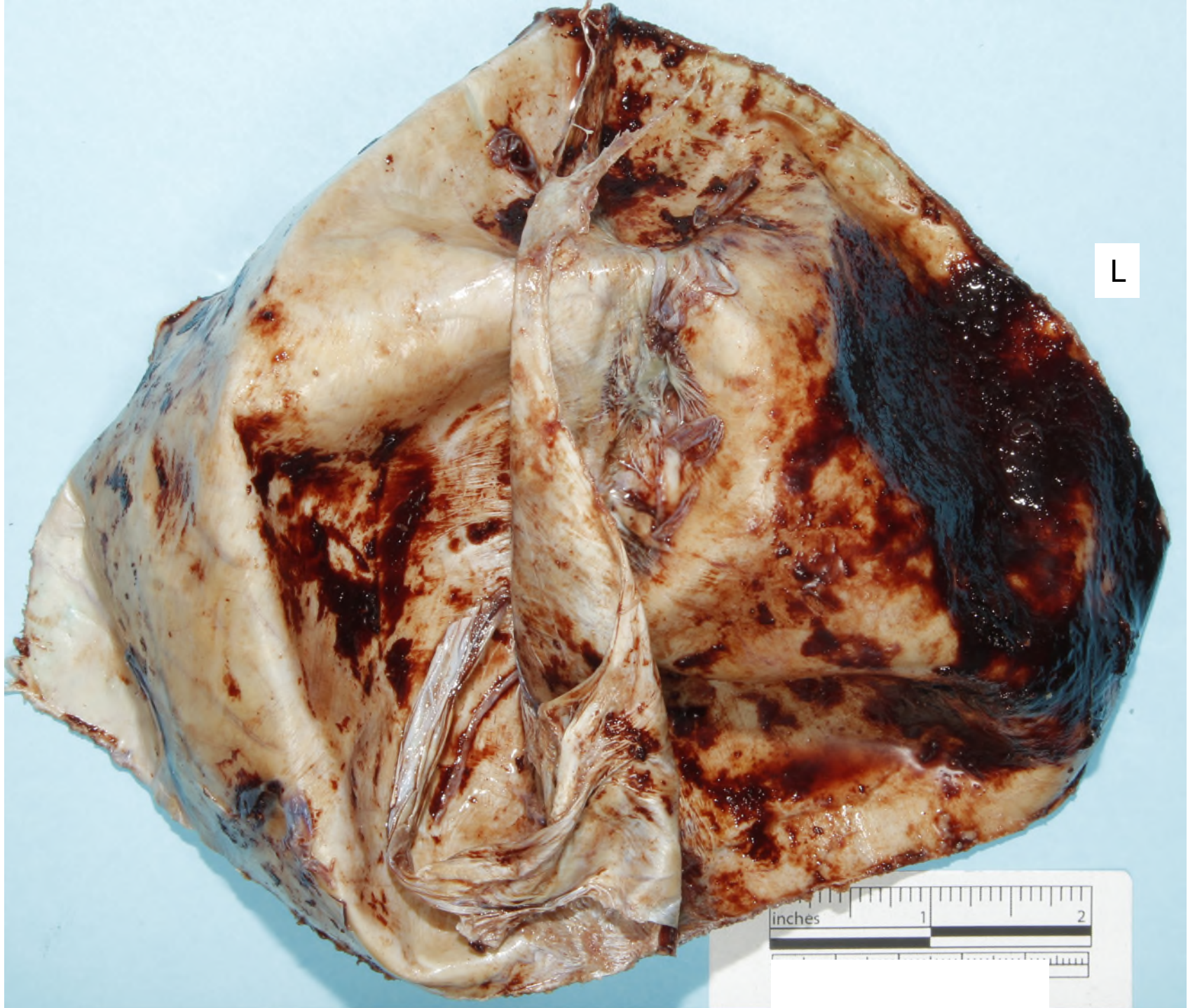
- Blunt impact injury of head, acute, with:
  - Subdural hematoma
  - Midline shift
    - Subfalcine and transtentorial hernias
    - Entrapped contralateral ventricle
  - Secondary brainstem (Duret) hemorrhage

(Case 2, 3)

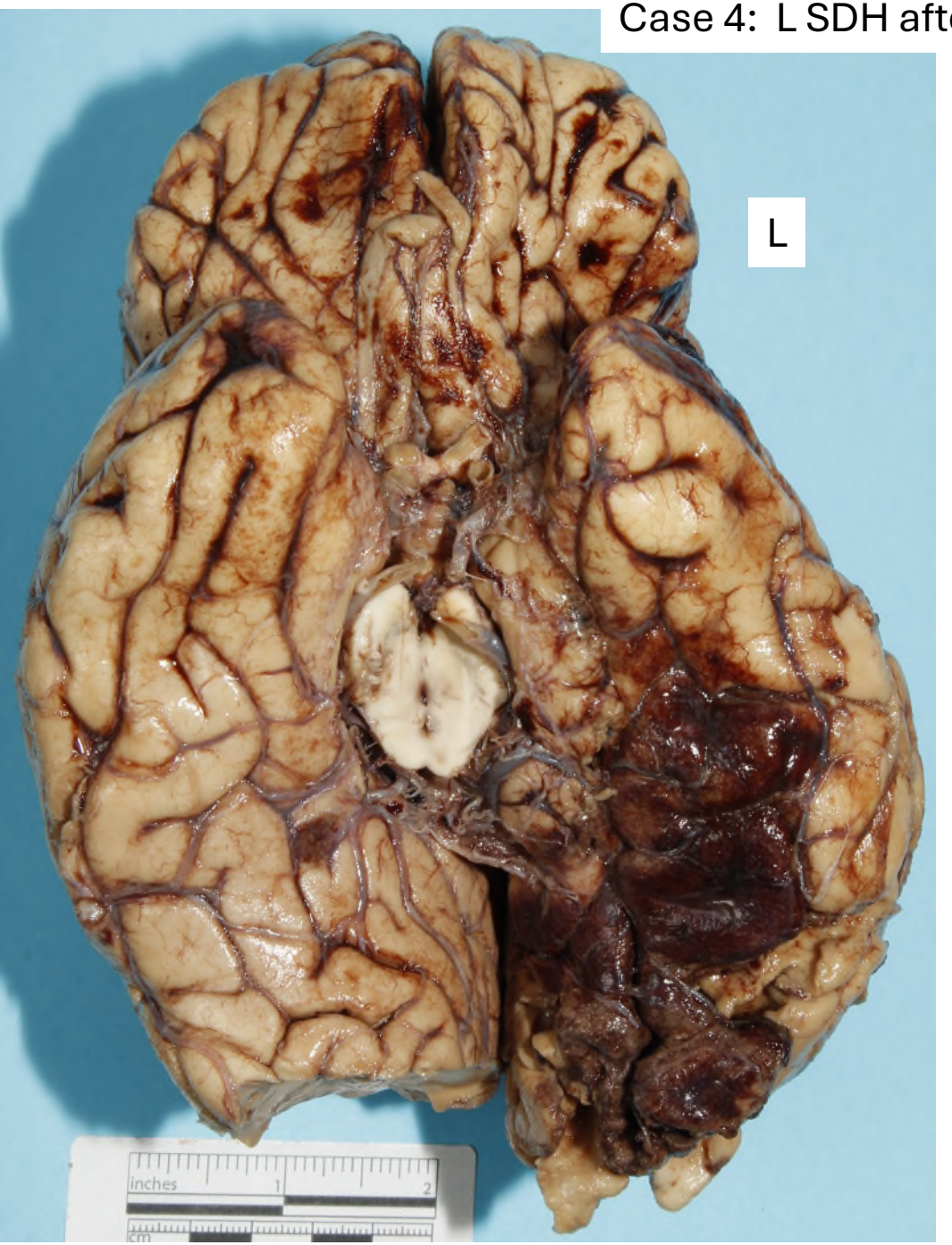


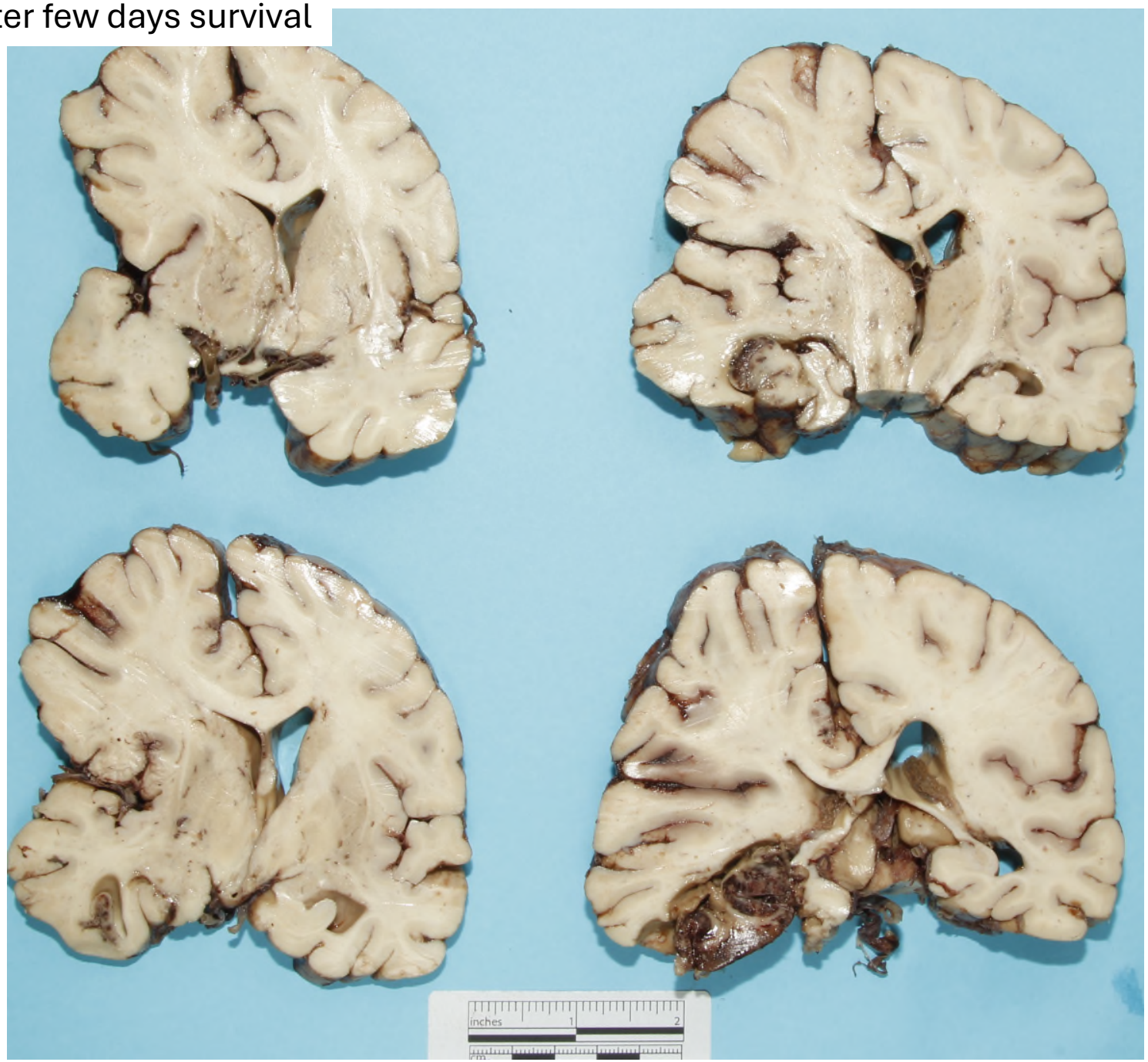
Radiopaedia

Case 4: L SDH after few days survival



Case 4: L SDH after few days survival



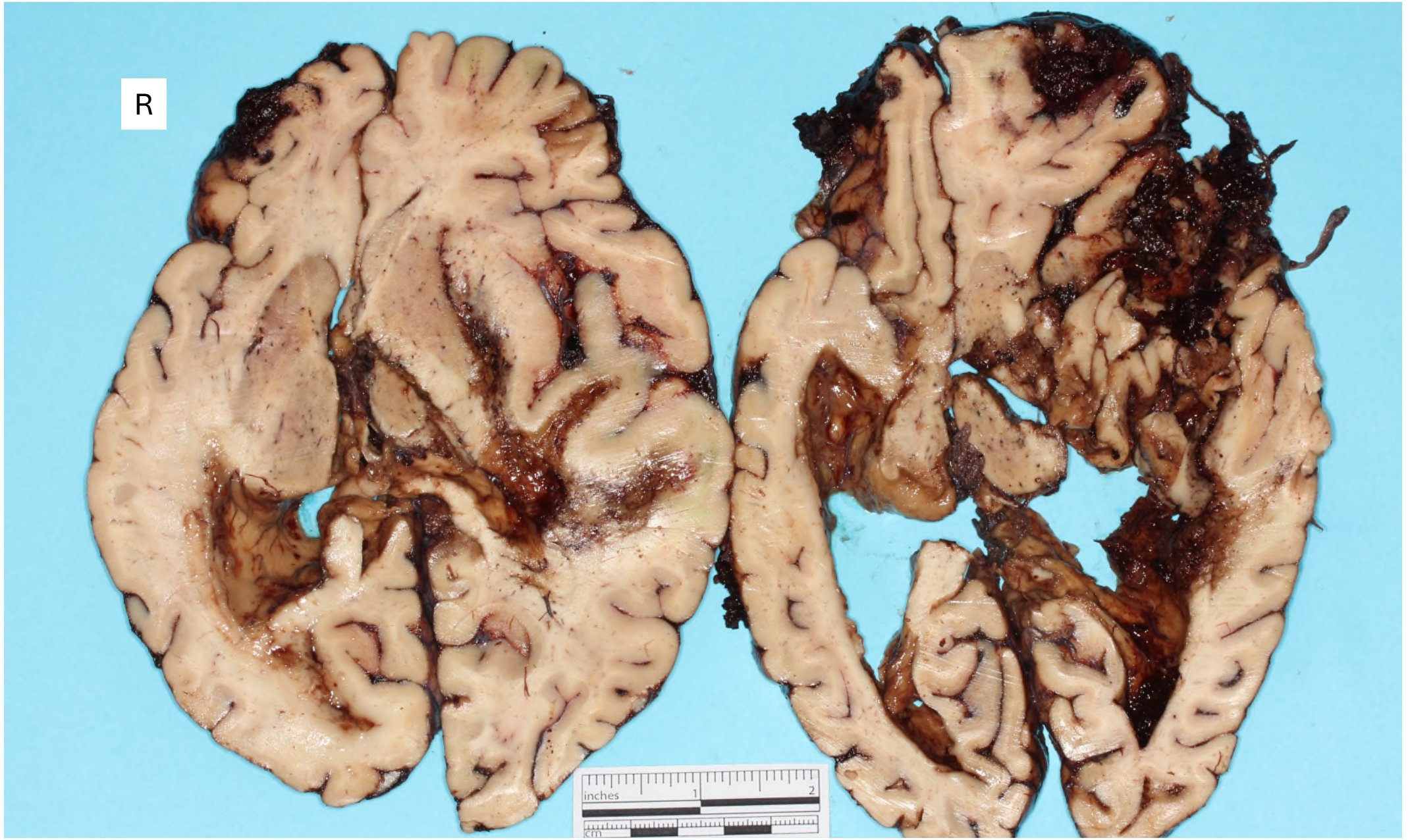


FNPDx:

- Blunt impact injury of head, subacute, with:
  - L subdural hematoma
  - Left-to-right midline shift, with:
    - L subfalcine and transtentorial hernias, with compression ischemia and reperfusion hemorrhage (“herniation contusions”)
    - Entrapped R lateral ventricle
  - Secondary compression ischemia of L posterior cerebral artery distribution
  - Secondary brainstem (Duret) hemorrhage

(Case 4)

Case 5: L SDH after few days survival (axial section)



Case 5: L SDH after few days survival (axial section)

FNPDx:

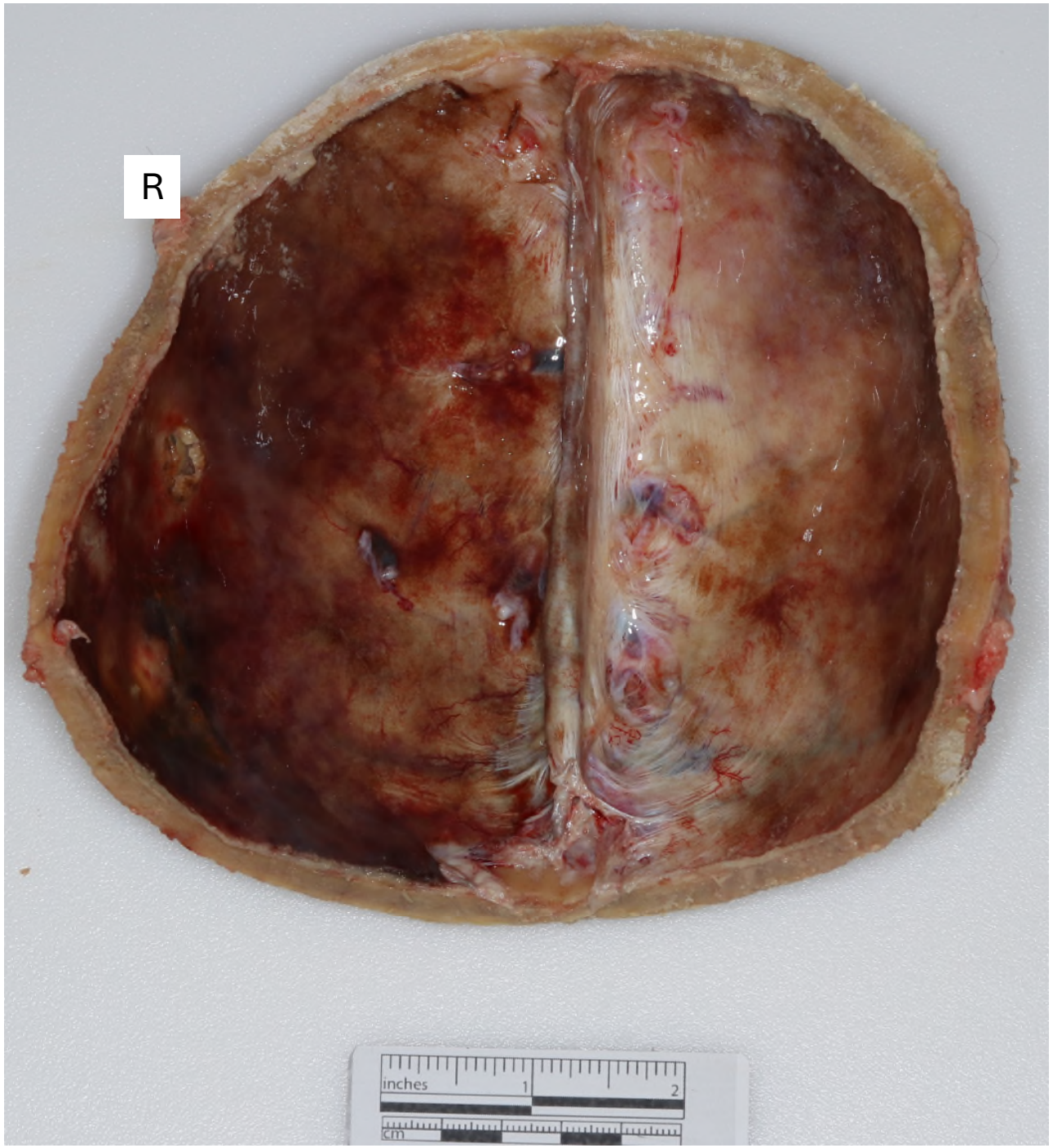
- Blunt impact injury of head, subacute, with:
  - L subdural hematoma
  - Orbitofrontal and L temporal contusions, with L frontotemporal contusion hematomas
  - Left-to-right midline shift, with:
    - L subfalcine and transtentorial hernias
    - R transtentorial compression ischemia (“herniation contusion”, “Kernohan notch phenomenon”)
    - Entrapped R lateral ventricle
  - Secondary brainstem (Duret) hemorrhage

(Case 5)

Case 6: Chronic SDH

- 76yo M, DM, prostate CA, prior “CVA”, temporally related to assault 5mos prior
  - Was punched by another pt while in hospital for foot infection, had “brain bleed”/SDH, s/p surgery
  - Went to nursing home with hemiparesis, sepsis, trach/peg, continued decline
  - Palliative extubation


inches
1
2
cm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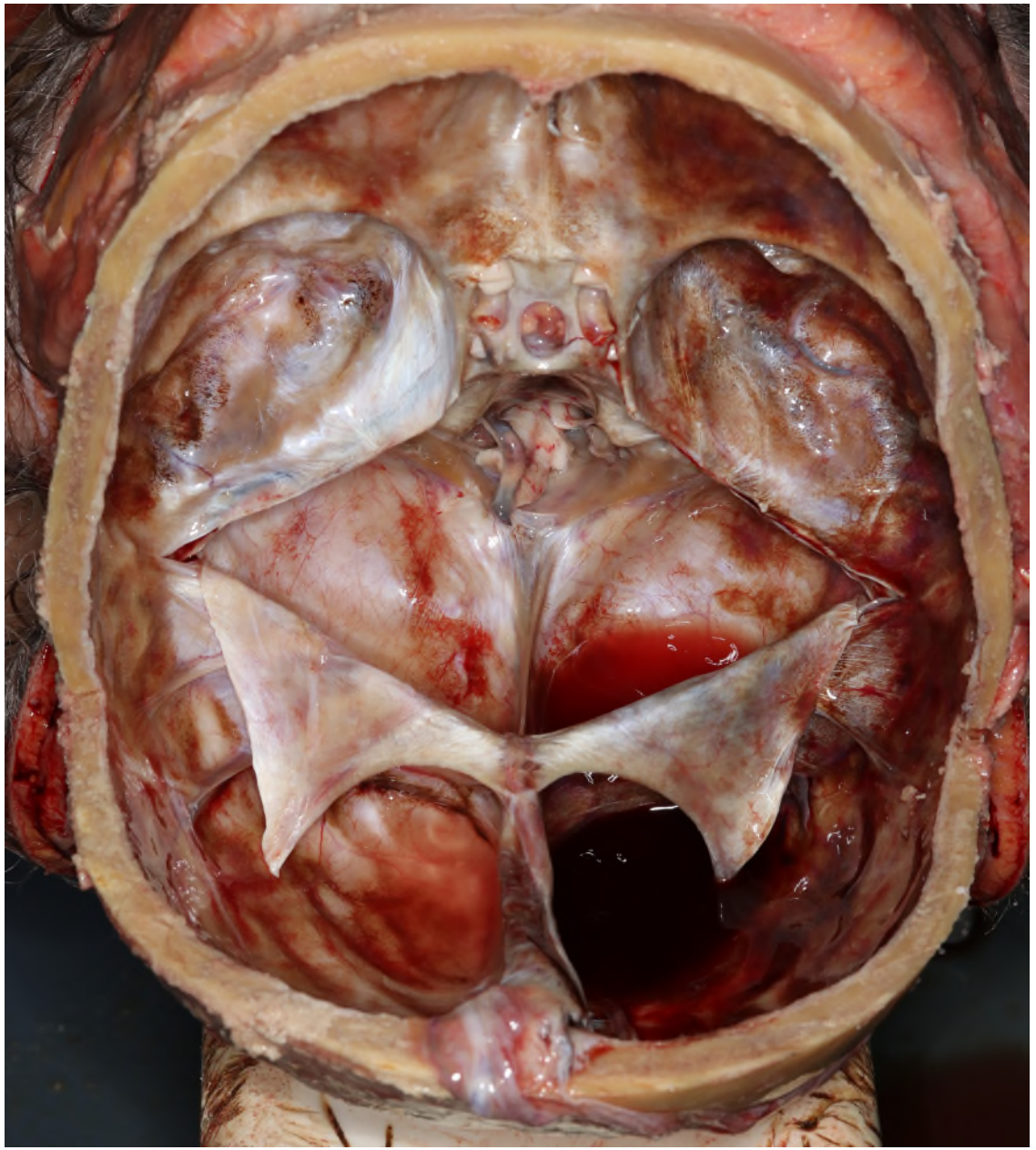


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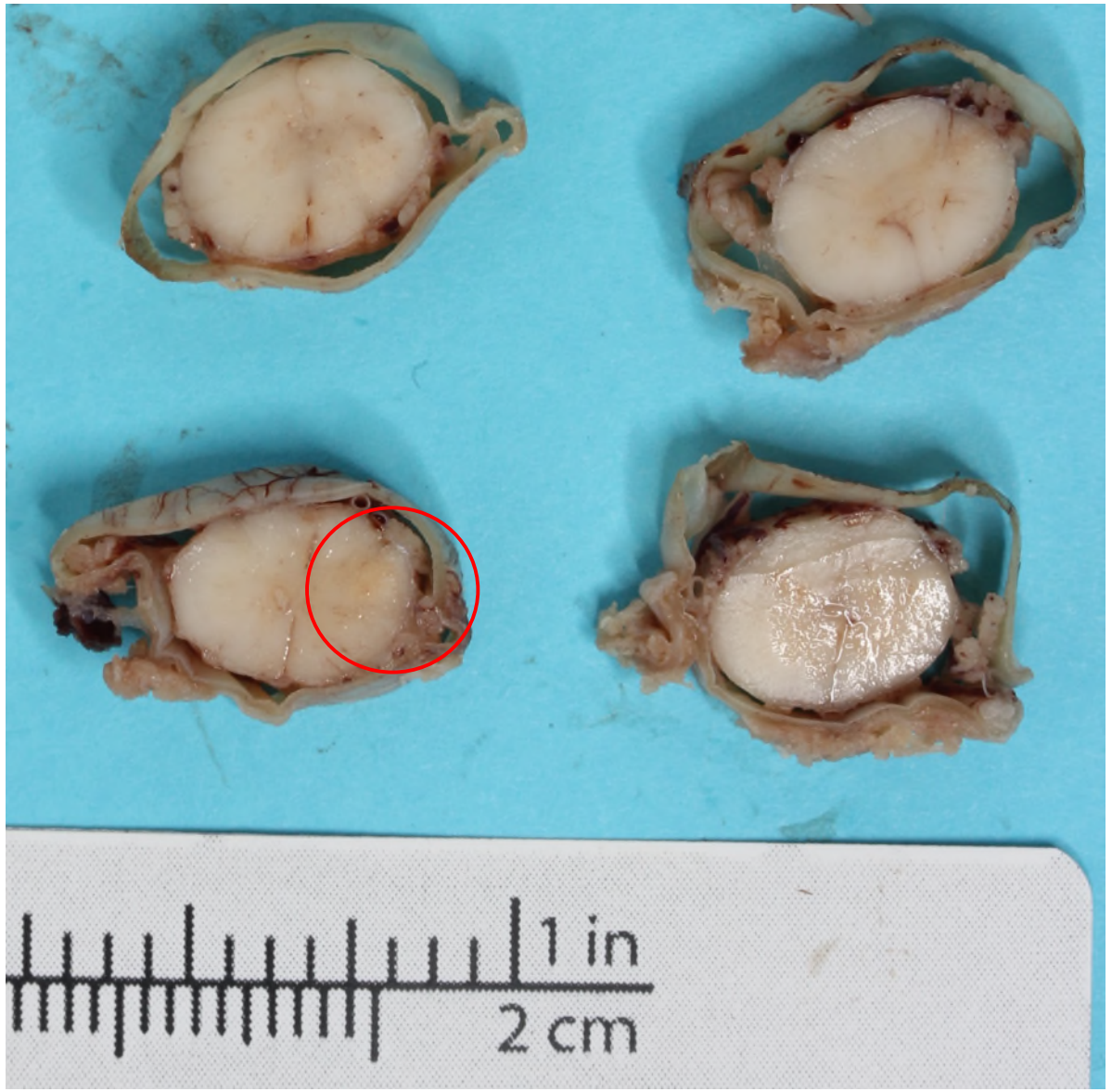
1 in
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FNPDx:

I. Blunt impact injury of head, old
    A. Subdural neomembranes, bilateral
        i. Status post neurosurgical intervention(s)
    B. Subarachnoid hemorrhage, right frontotemporal *(not shown)*
II. Cerebrovascular disease
    A. Atherosclerosis, moderate *(not shown)*
    B. Arteriolosclerosis, marked
        i. Lacunes, left basis pontis
            a. Secondary degenerative and reactive changes of left pyramid and right-sided corticospinal tracts in spinal cord

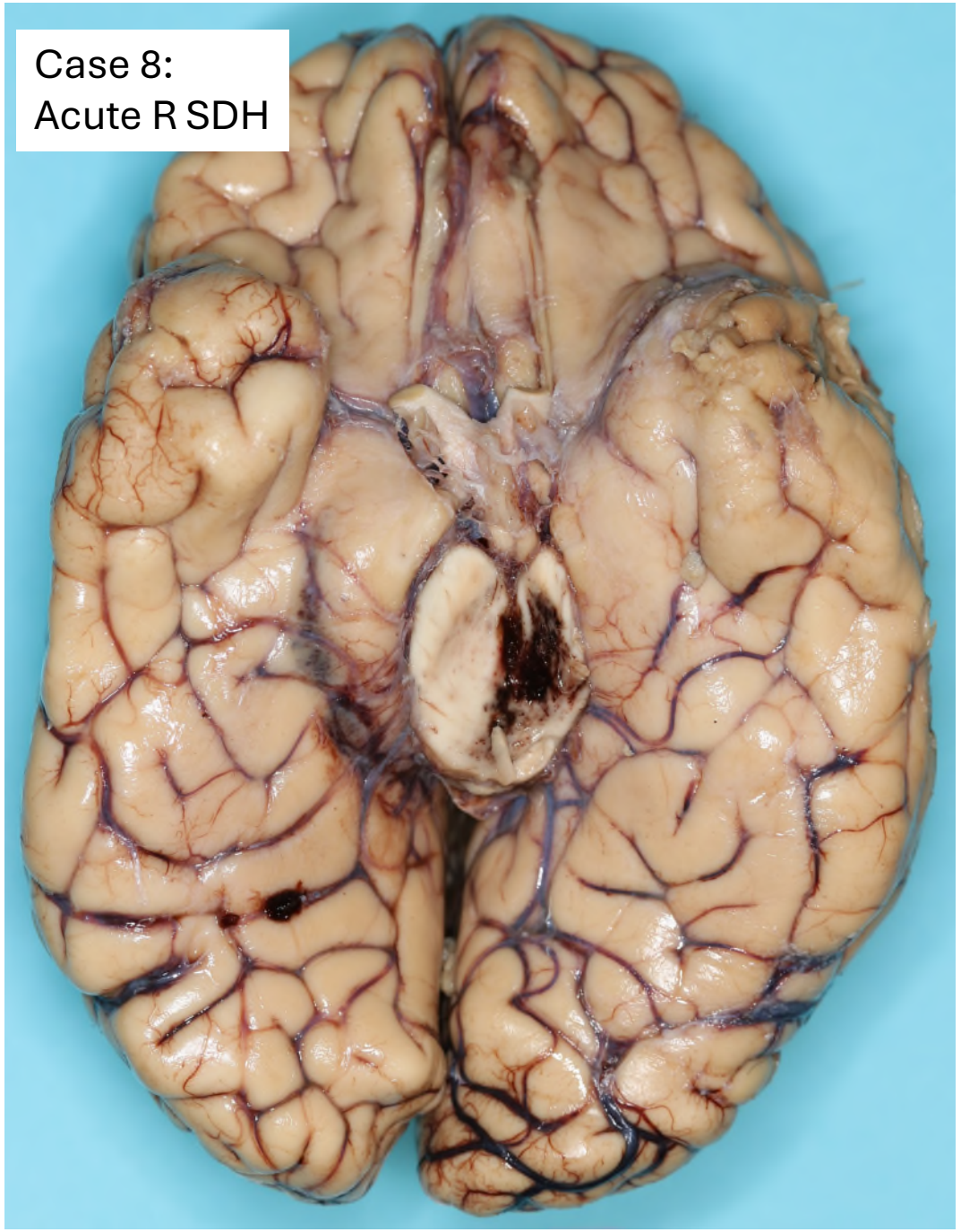
(Case 6)

Cases 7-12: Duret hemorrhages

- “50yo bicyclist struck by truck at intersection”

Case 7:
Acute L SDH

Case 8:
Acute R SDH



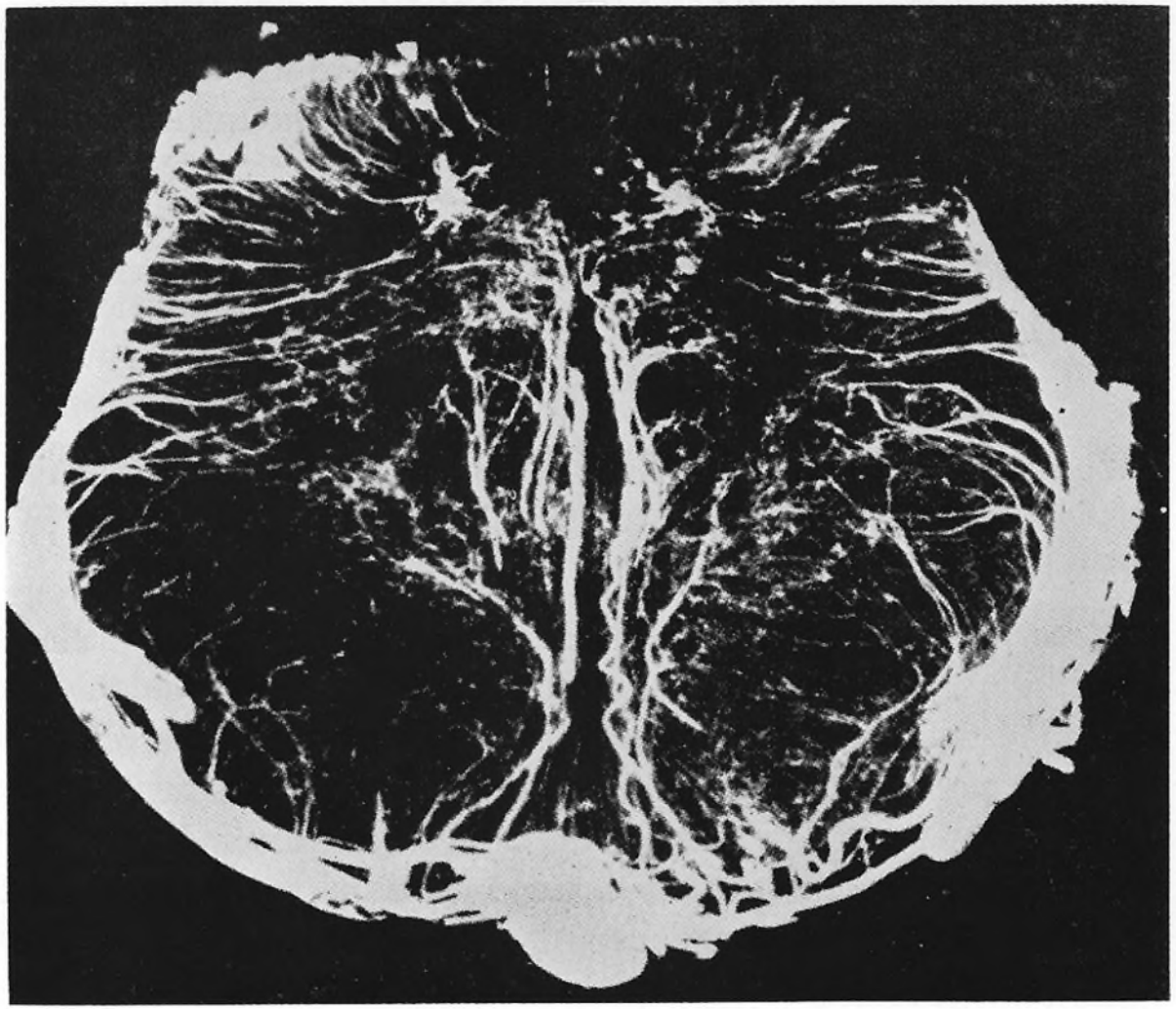


Fig. 6. Cross-sectional view of the pons with injected arteries, demonstrating the penetrating paramedian and circumferential systems of vessels (reprinted with the permission of Kaplan).

From Finney and Walker, Transtentorial Herniation, 1962.

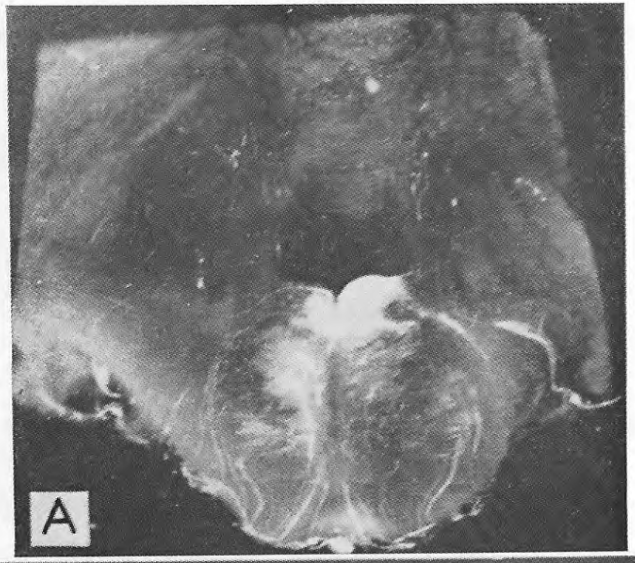
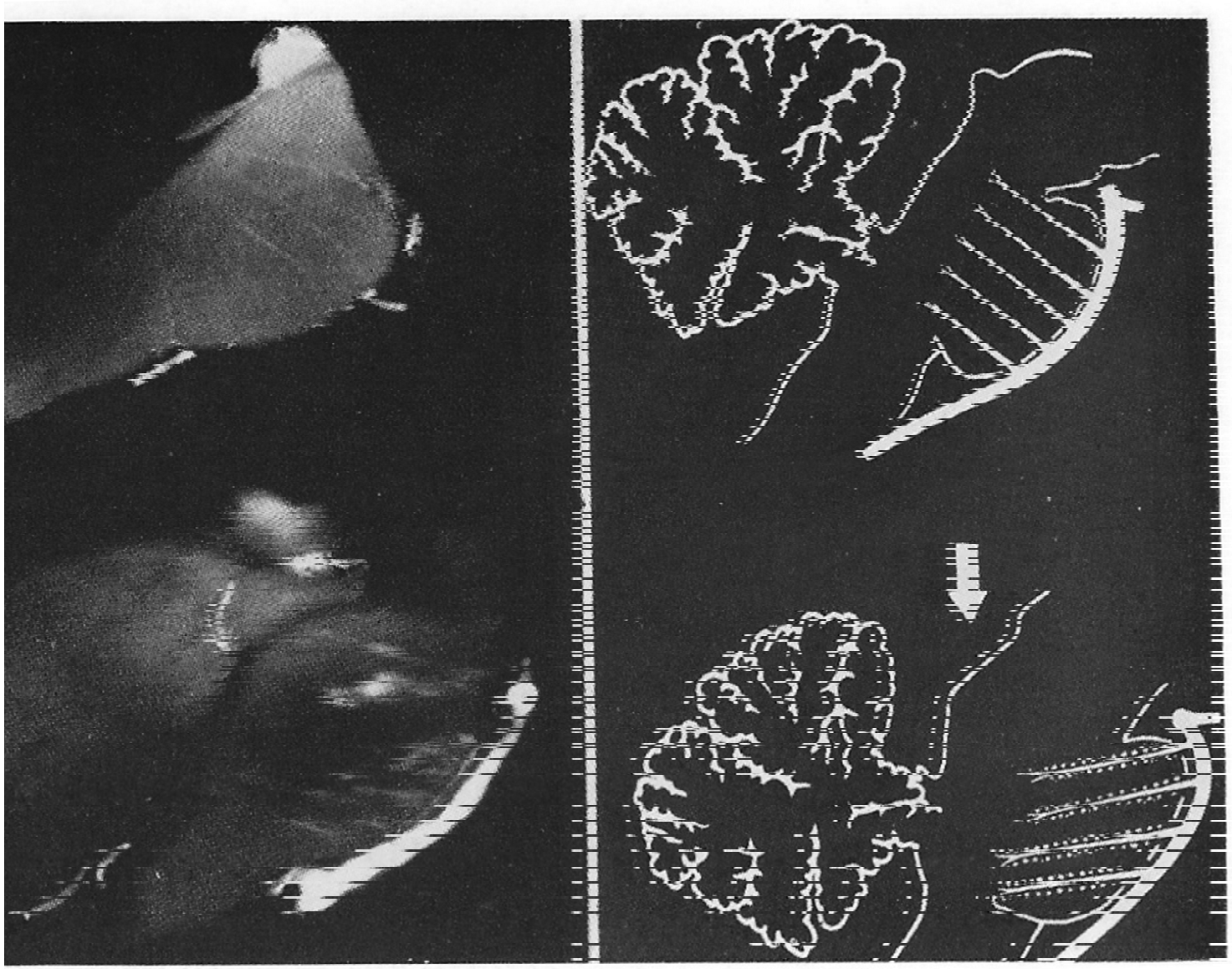


Fig. 21. a. Radiograph of a transverse slice of pons from a "normal" brain. Lateral pressure was applied to the upper pons and midbrain while the arteries were injected with silver iodide mixture. The resulting "hemorrhages" closely simulate those occurring naturally.

b. Sagittal section of a "normal" brainstem. Intra-arterial silver iodide solution was injected while lateral pressure was applied only to the midbrain. Resultant "hemorrhages" are confined to that region (reprinted with the permission of Johnson and Yates).



From Finney and Walker, Transtentorial Herniation, 1962.

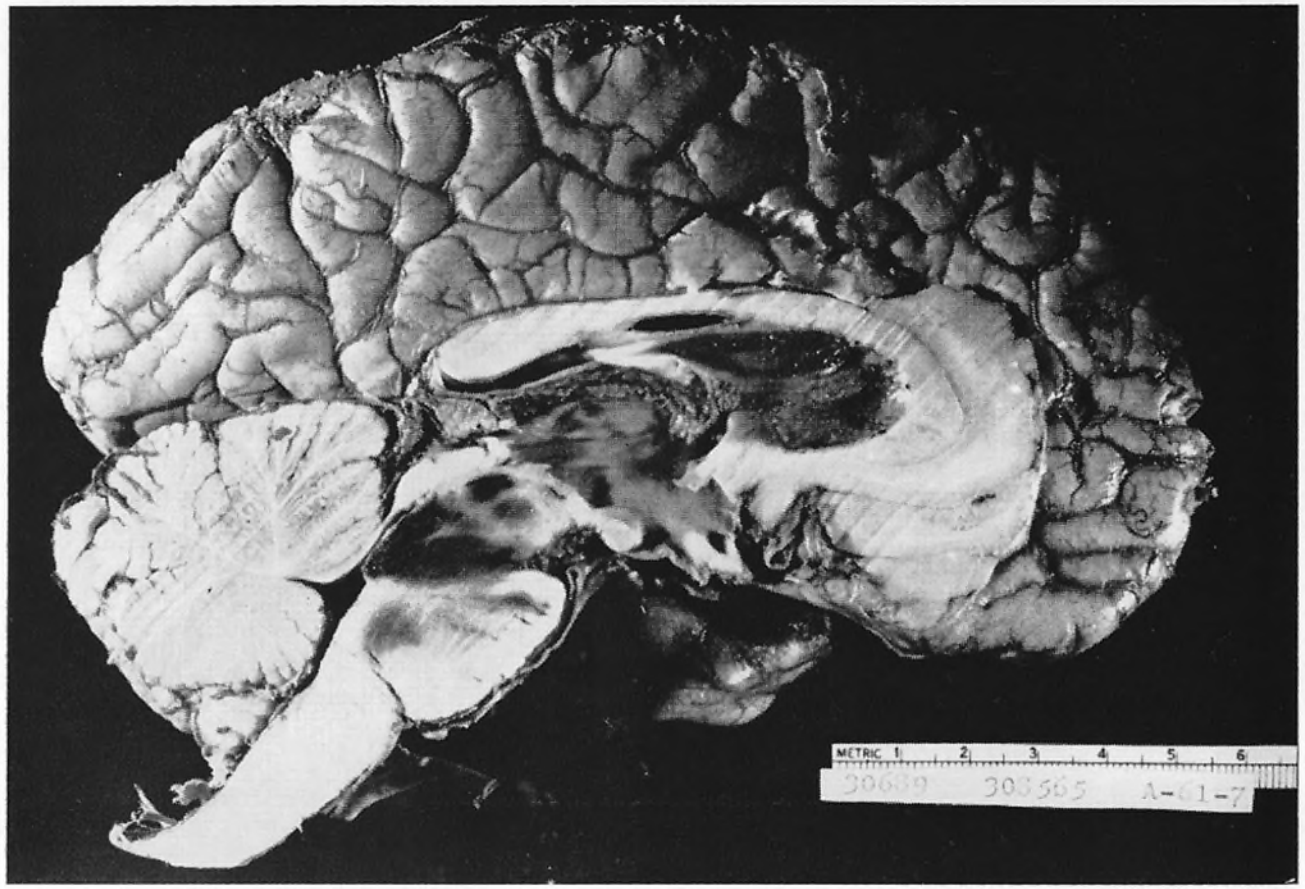
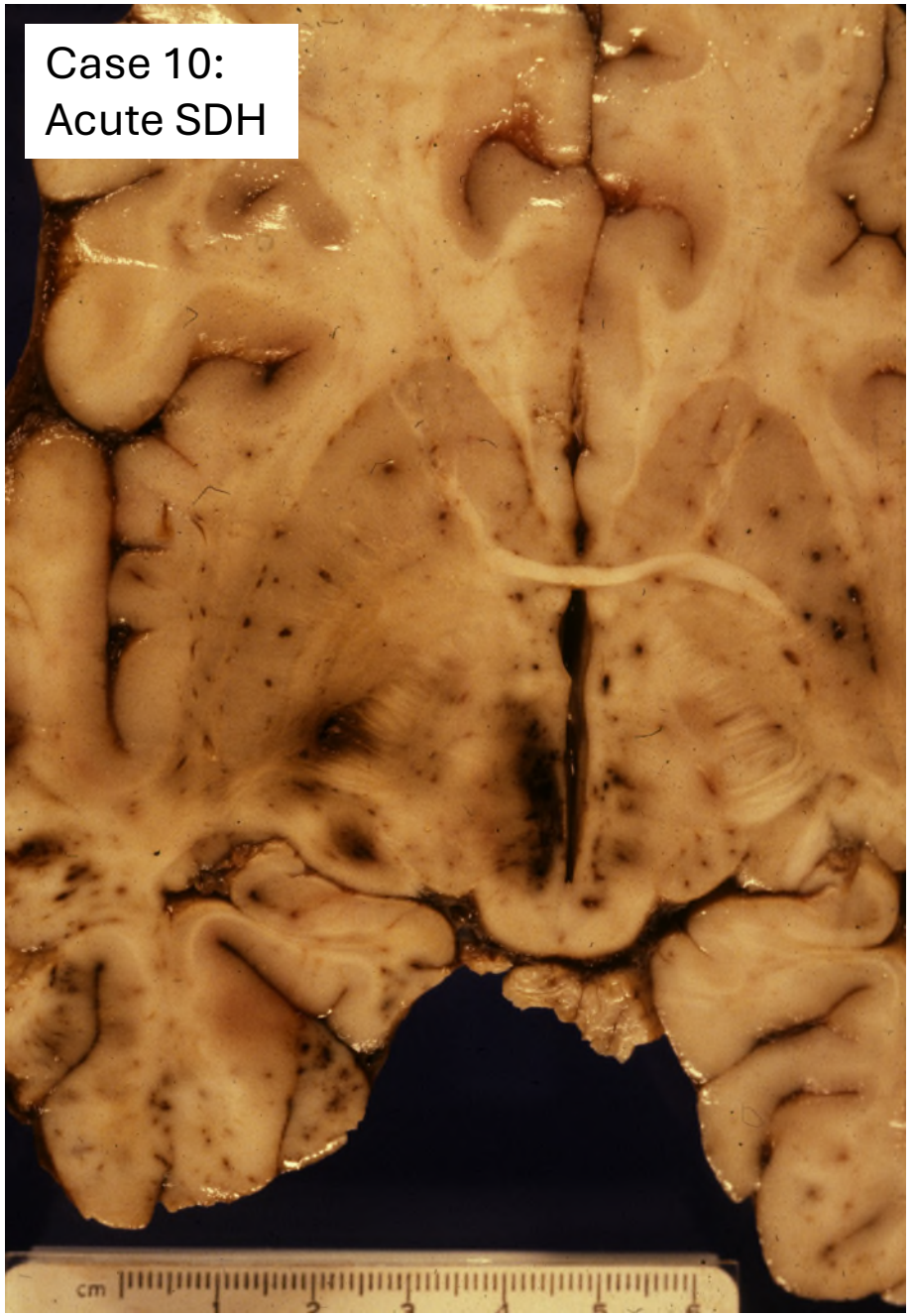


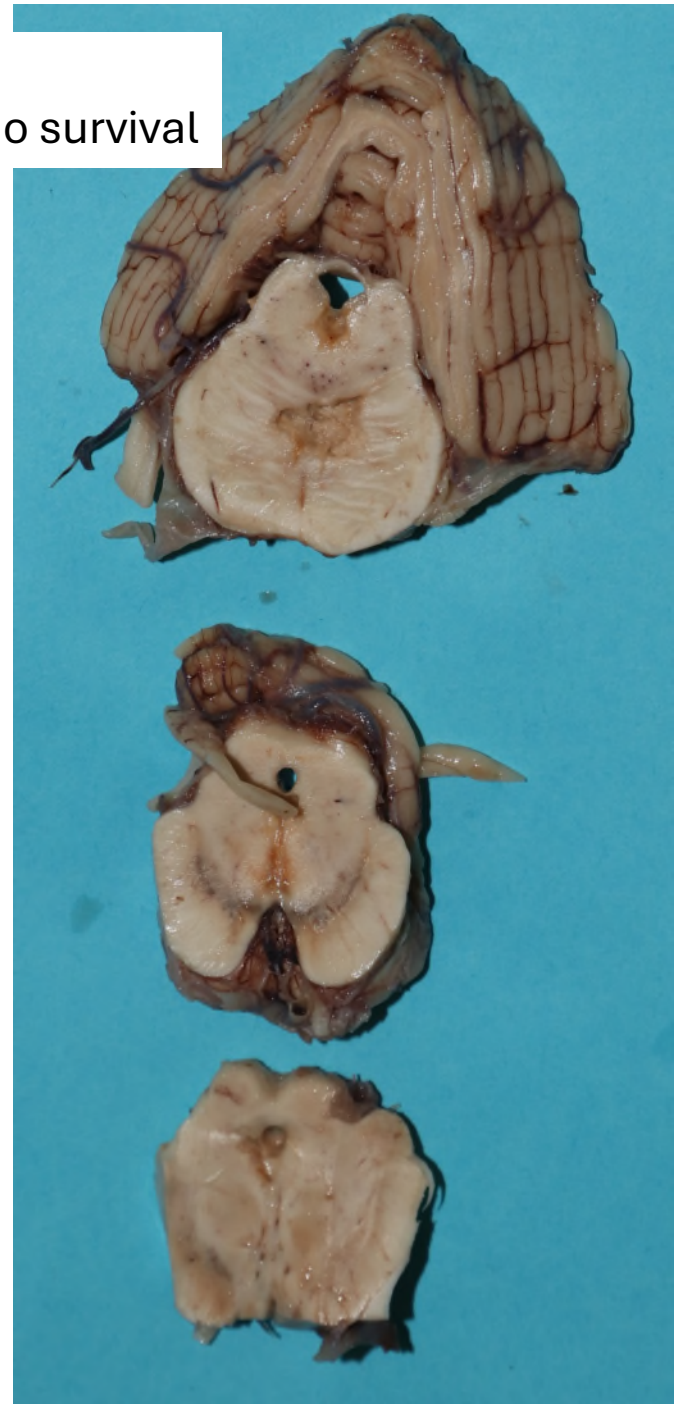
Fig. 22. (A-61-7) Sagittal section of the brain of a 58 year old man who suffered a right frontal hemorrhage and secondary transtentorial herniation producing distortion of the brainstem, and pontine hemorrhages along the distribution of the penetrating paramedian arterioles of the pons.

From Finney and Walker, Transtentorial Herniation, 1962.

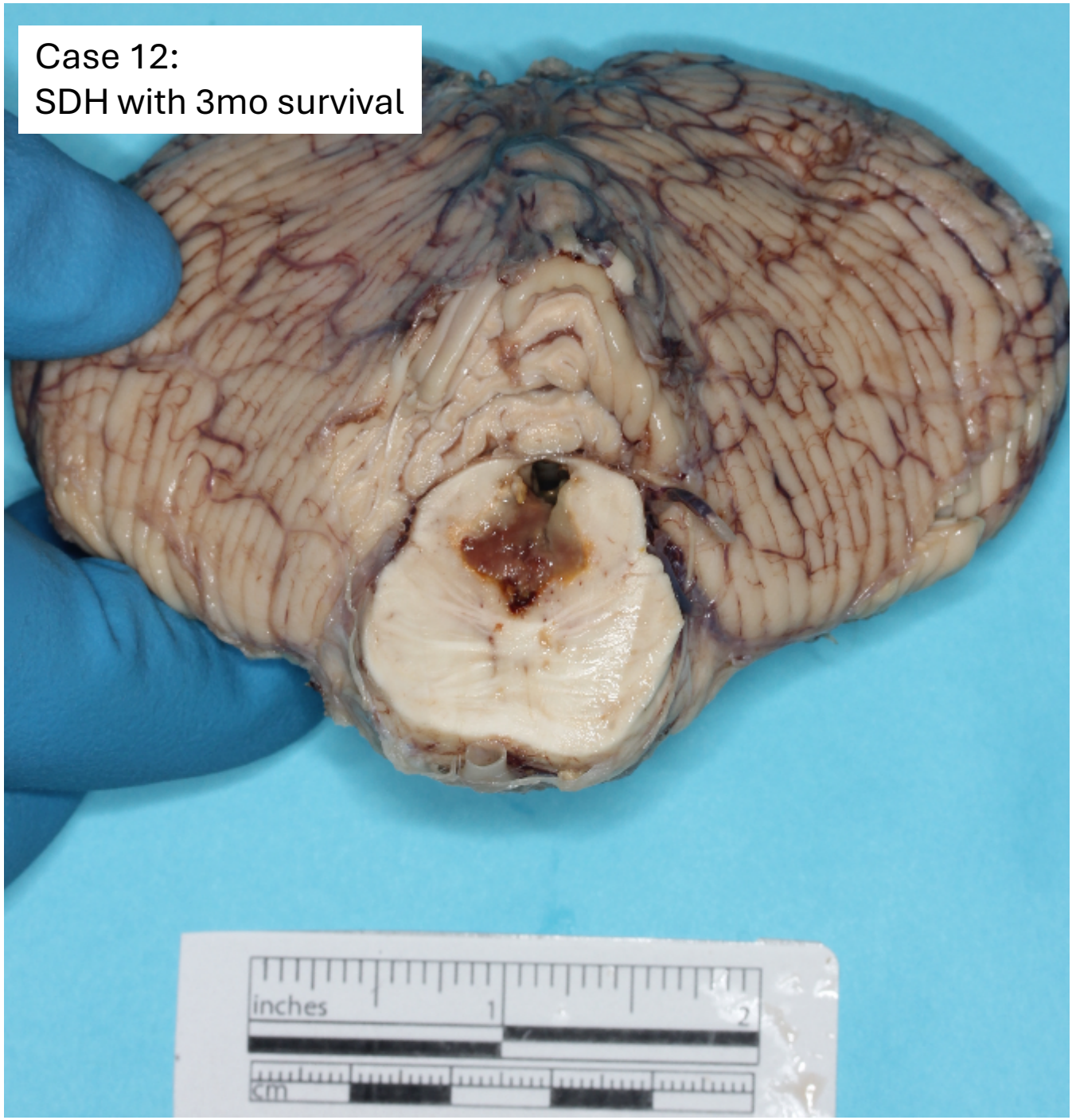


Courtesy Prof. U. de Girolami

Case 11:
SDH with 1.5mo survival



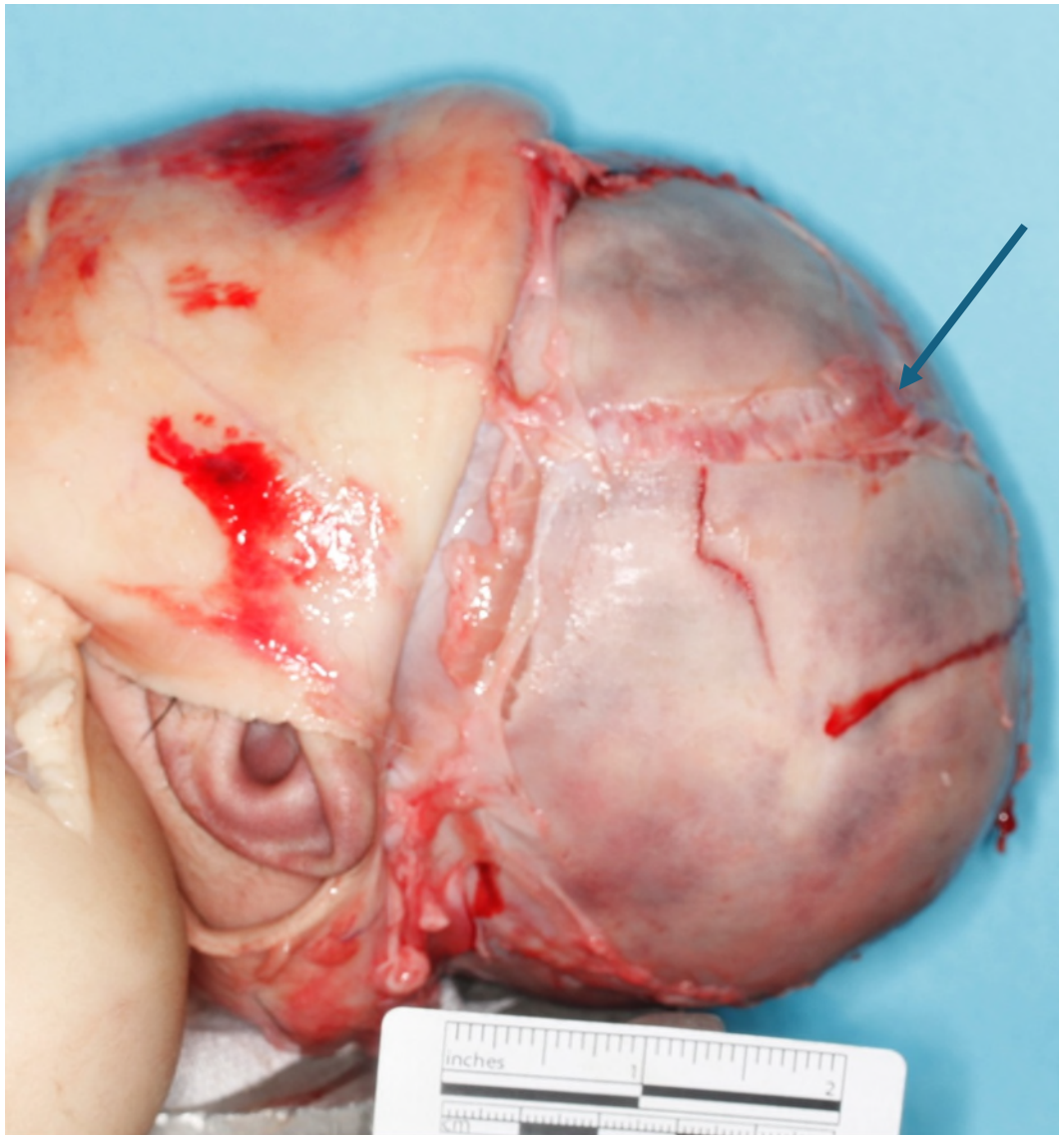
Case 12:
SDH with 3mo survival

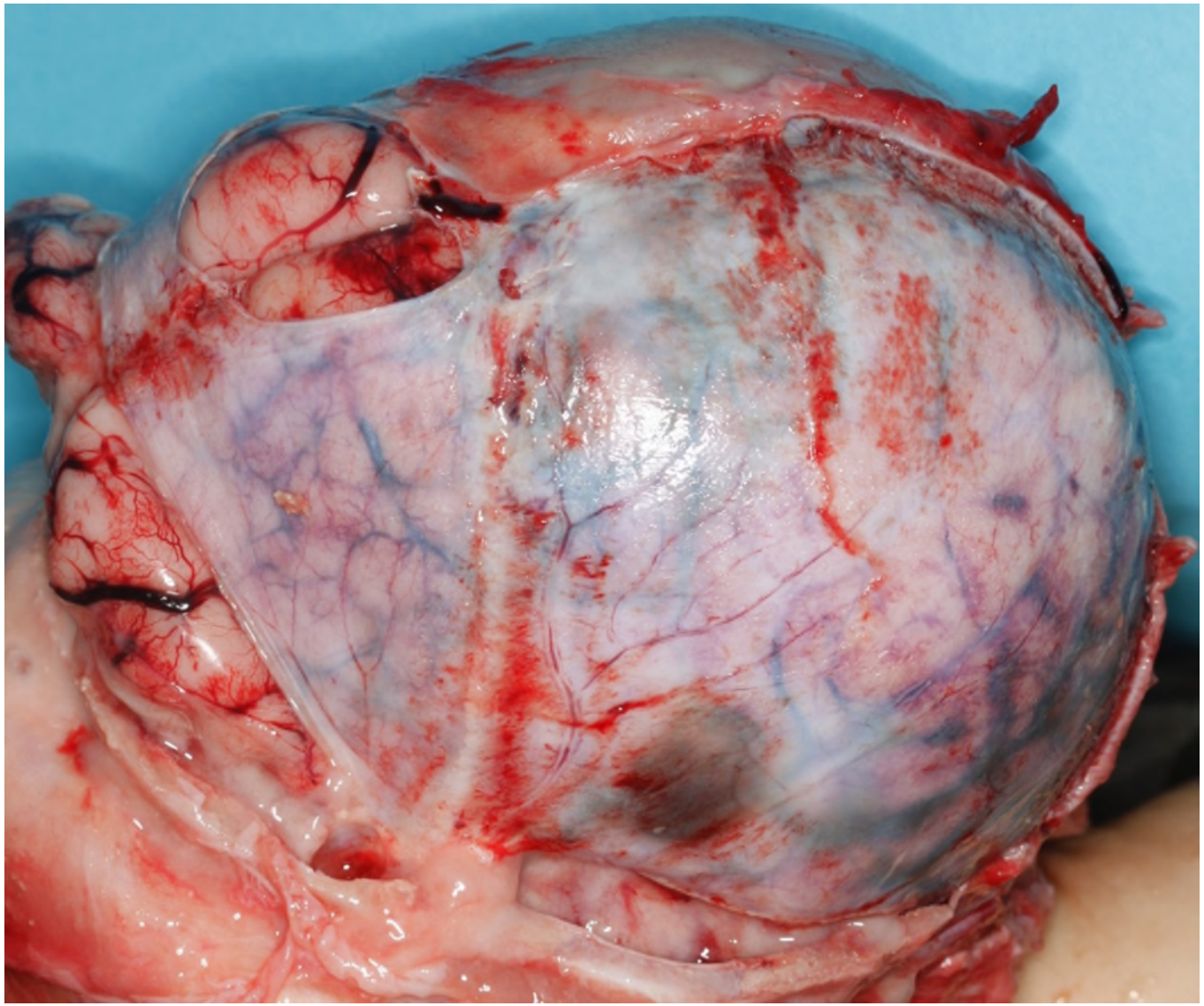


Cases 13-15: Pediatric acute and subacute TBI

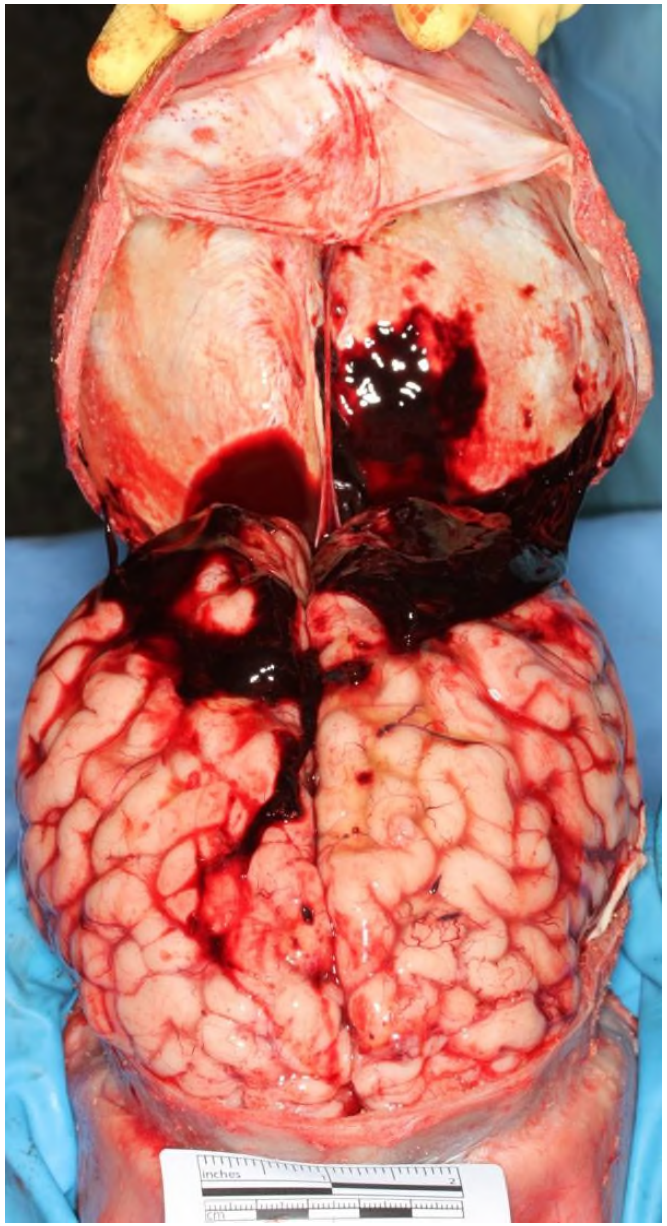
- “2mo brought to ER unresponsive, without adequate explanation (i.e., suspicious circumstances)”

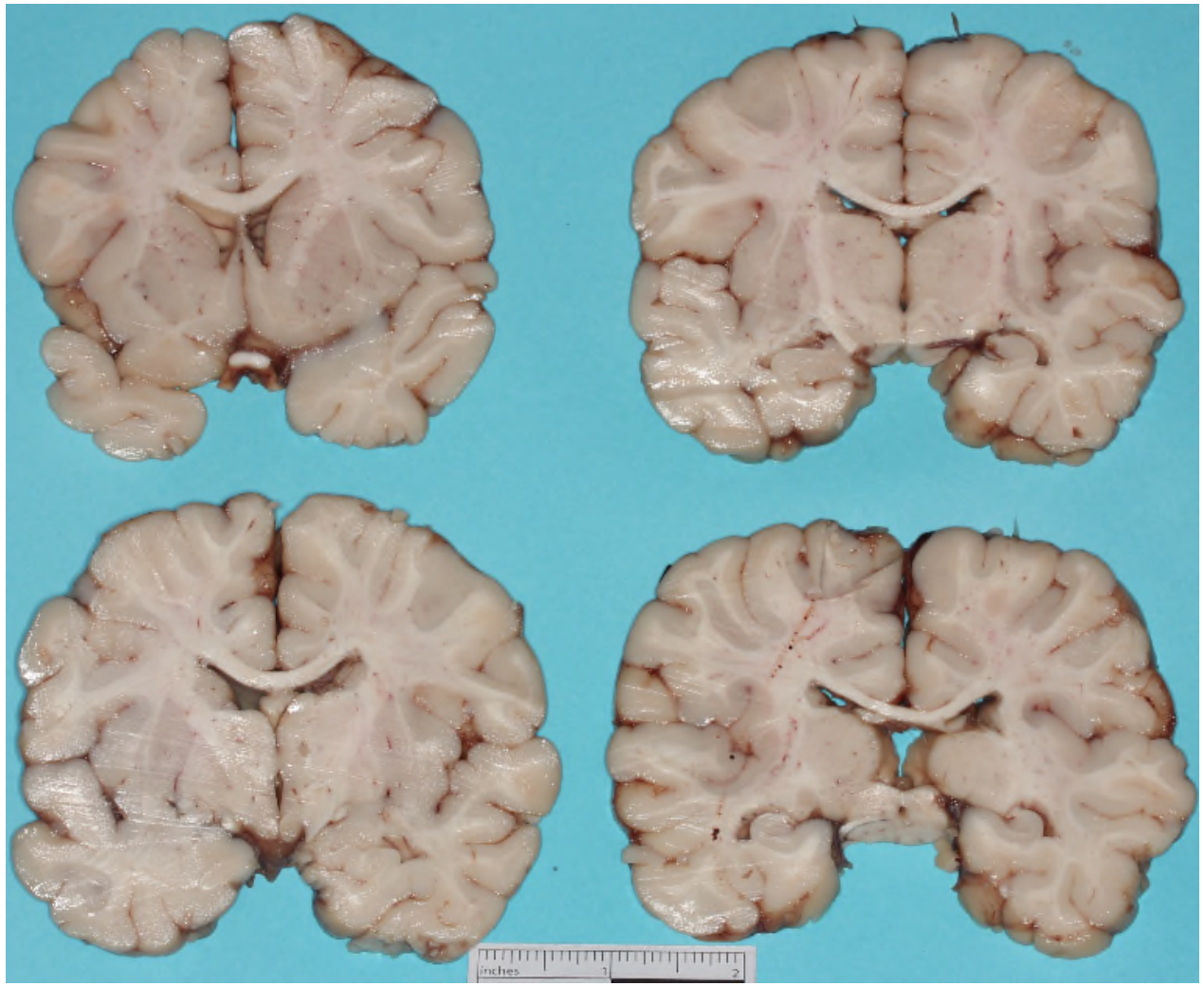
Case 13: Acute L parietal skull fracture, slight EDH, cranial sutural diastasis from brain swelling





Case 14: Acute SDH and brain swelling, without herniation





FNPDx:

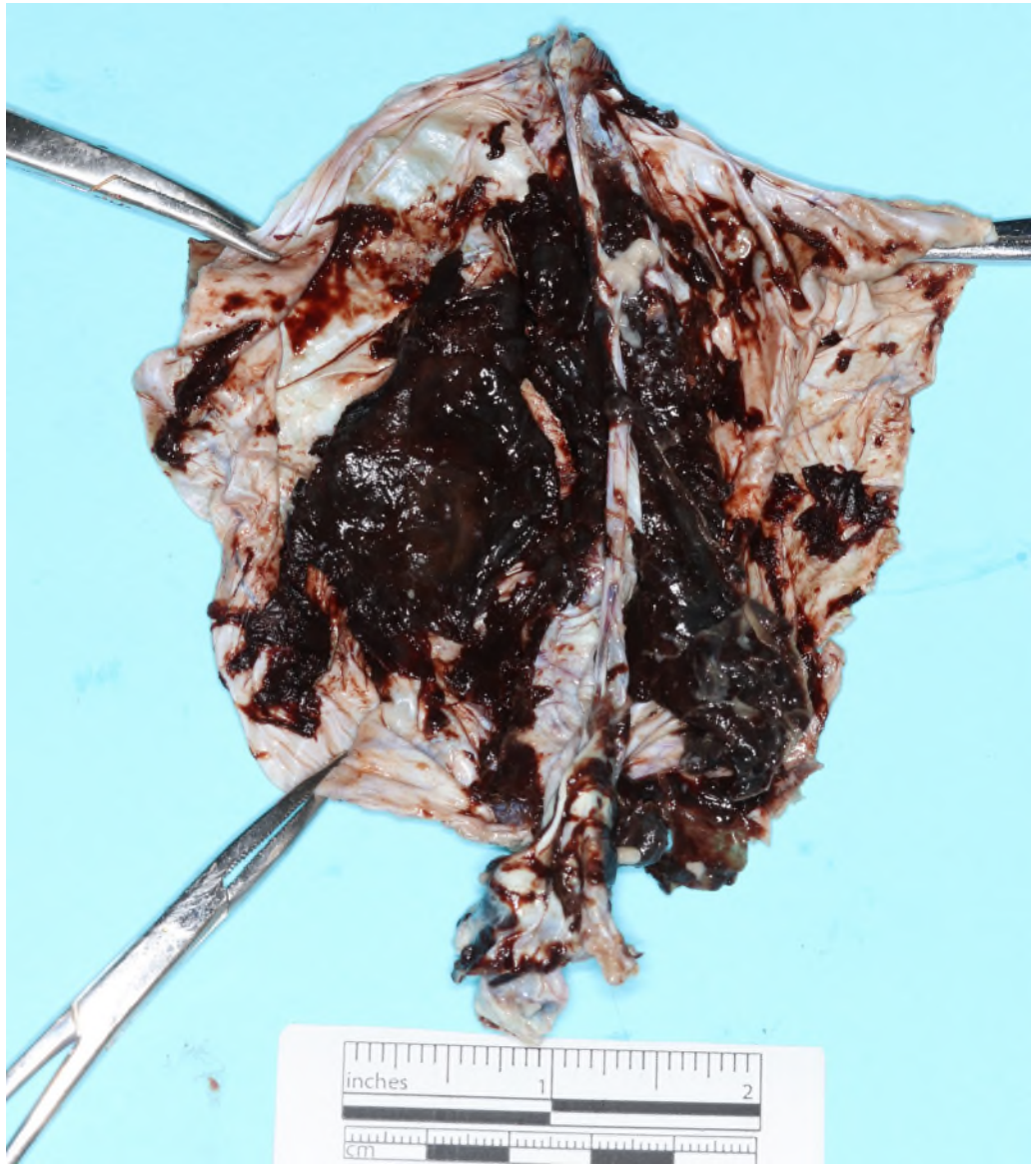
- Blunt impact injury of head, acute, with:
  - L parietal skull fracture, with slight epidural hemorrhage
  - Brain swelling
    - Cranial sutural diastasis

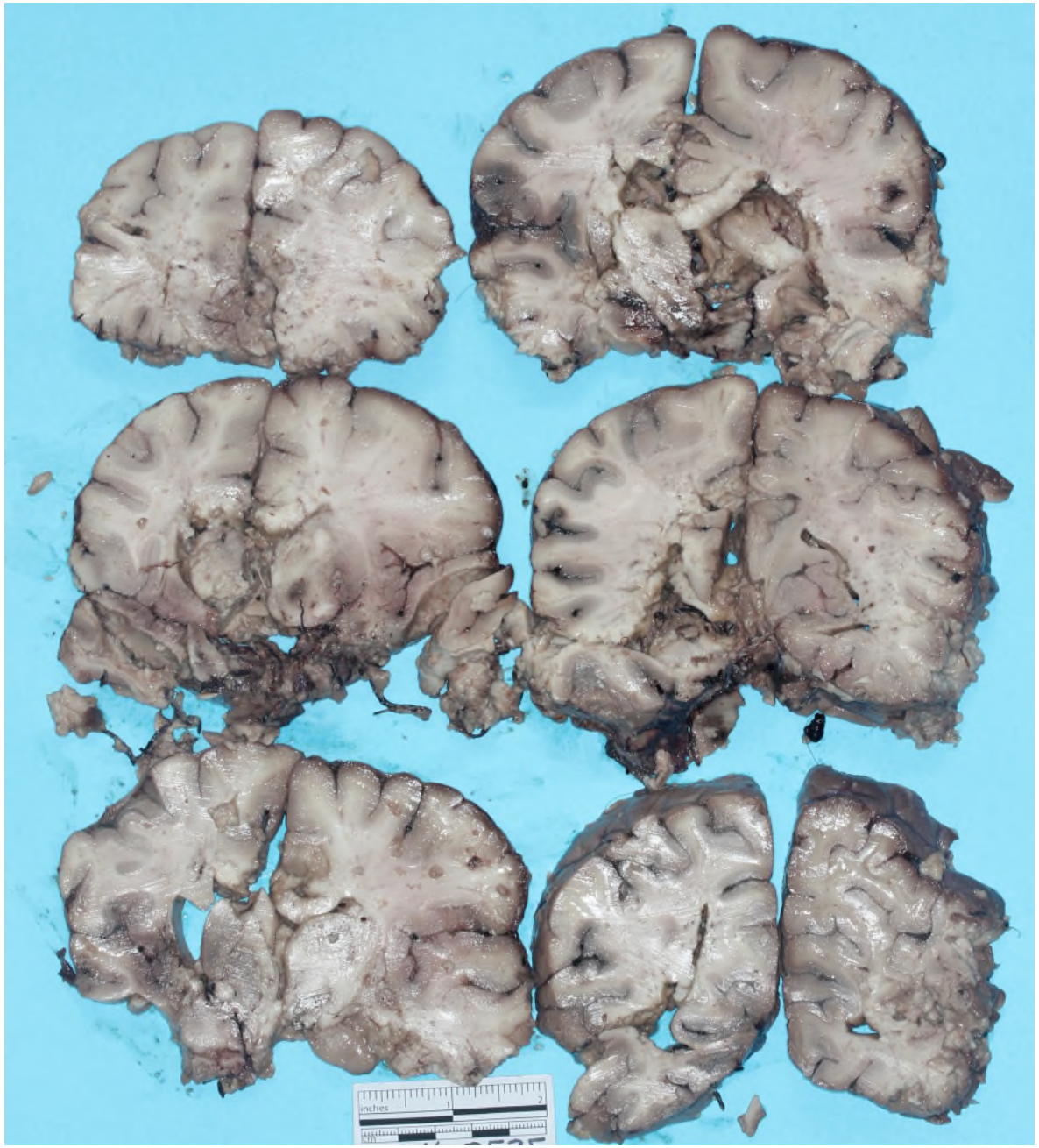
(Case 13)

- Traumatic injury of head, acute, with:
  - Bilateral subdural hemorrhage
  - Brain swelling
    - Cranial sutural diastasis

(Case 14)

Case 15: Subacute SDH and ocular hemorrhages, 3 days' survival





Case 15: Subacute cervical nerve root hemorrhages

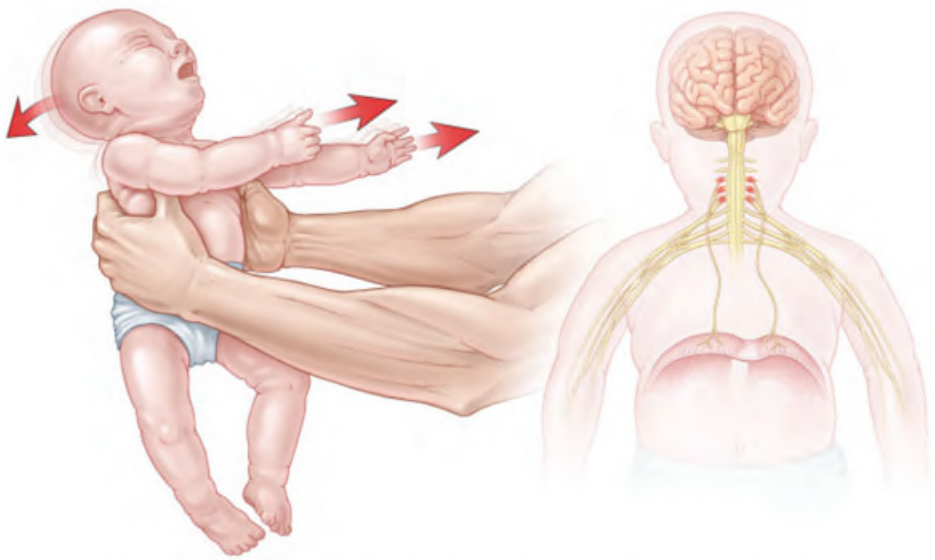
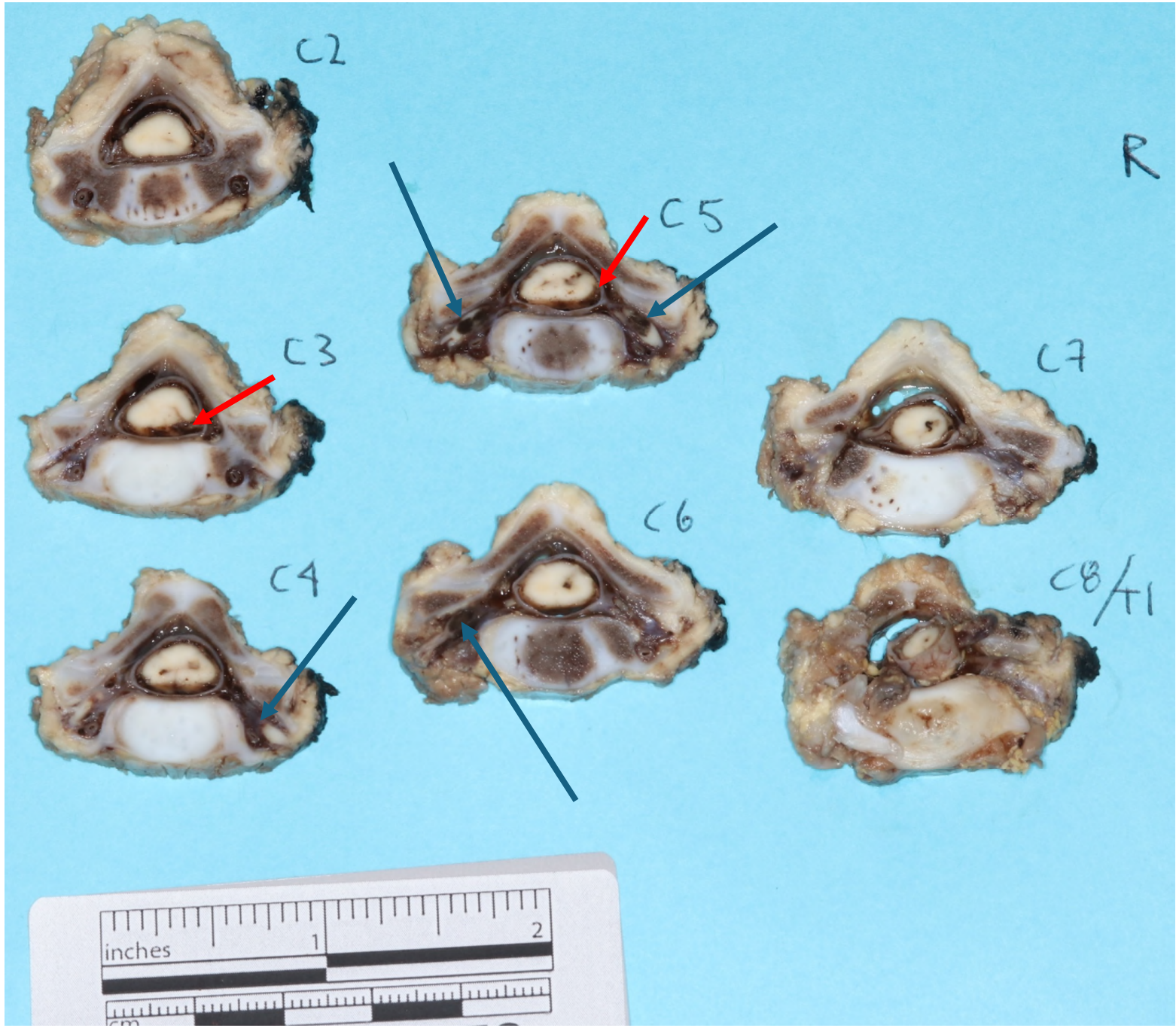


FIGURE 2: Illustration of the forces an infant is subjected to when shaken. A - The size and weight of the head combined with the lack of developed neck musculature is expected to lead to alternating hyperflexion and hyperextension of the cervical spine. B – Cervical spine anatomy, showing levels C3, C4 and C5 highlighted, with nerve roots innervating the diaphragm.

Matshes et al, 2011



FNPDx:

- Traumatic injury of head and neck, subacute, with:
  - Bilateral subdural hemorrhage
  - Brain swelling
    - Cranial sutural diastasis
    - Secondary hypoxia-ischemia of:
      - Brain
        - Cerebellar cortical “emboli” to spinal canal
      - Central cervical spinal cord, with reperfusion hemorrhage
  - Bilateral optic nerve sheath hemorrhages
  - Bilateral, multilevel endoneurial cranial nerve root hemorrhages

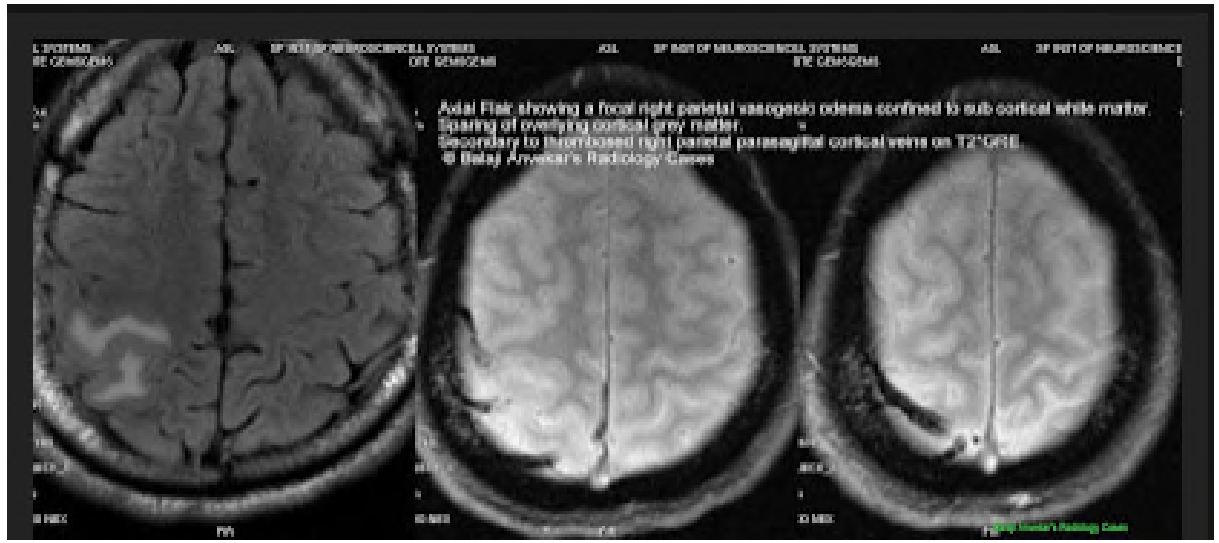
(Case 15)

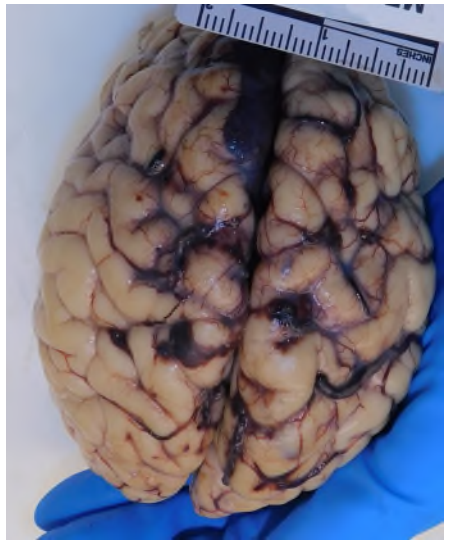
**autolysis, “brain death” changes*

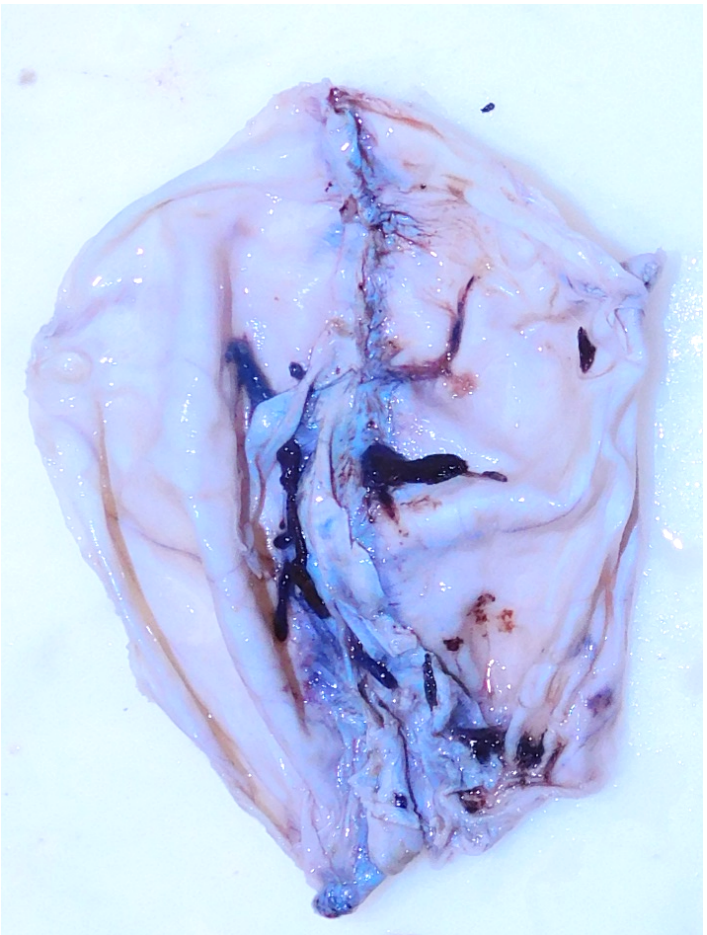
Case 16: Cortical vein thrombosis

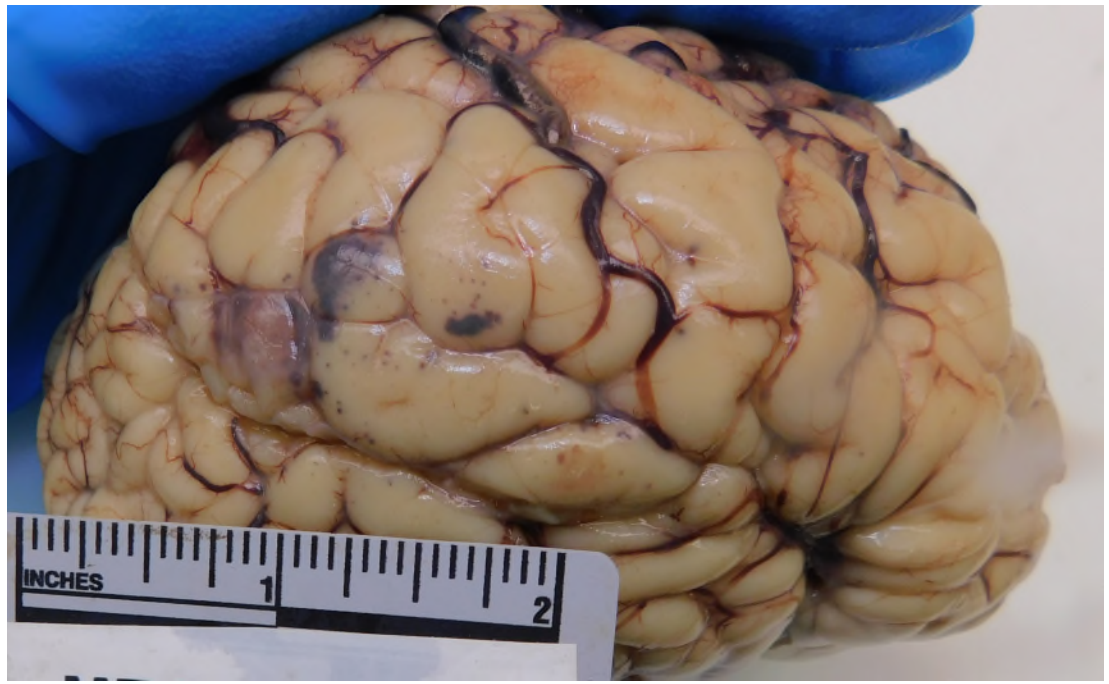
- “2mo brought to ER unresponsive, without adequate explanation (i.e., suspicious circumstances)”

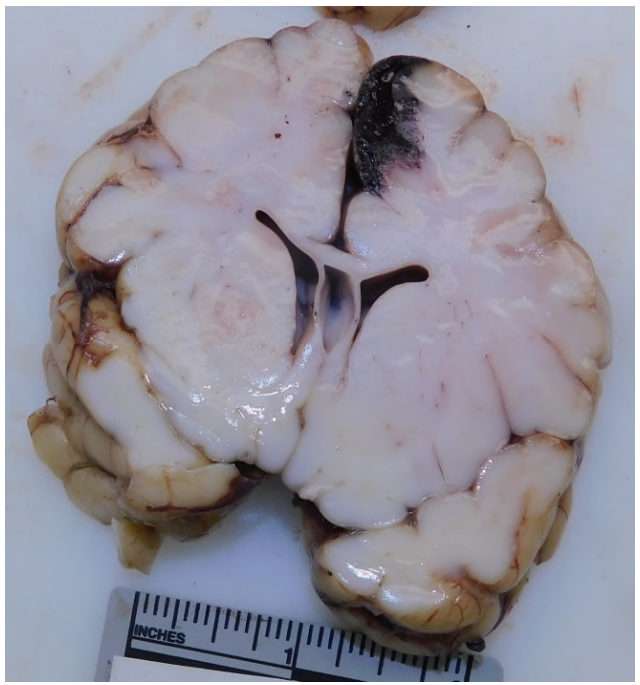
Case 16: Cortical vein and dural venous sinus thrombosis



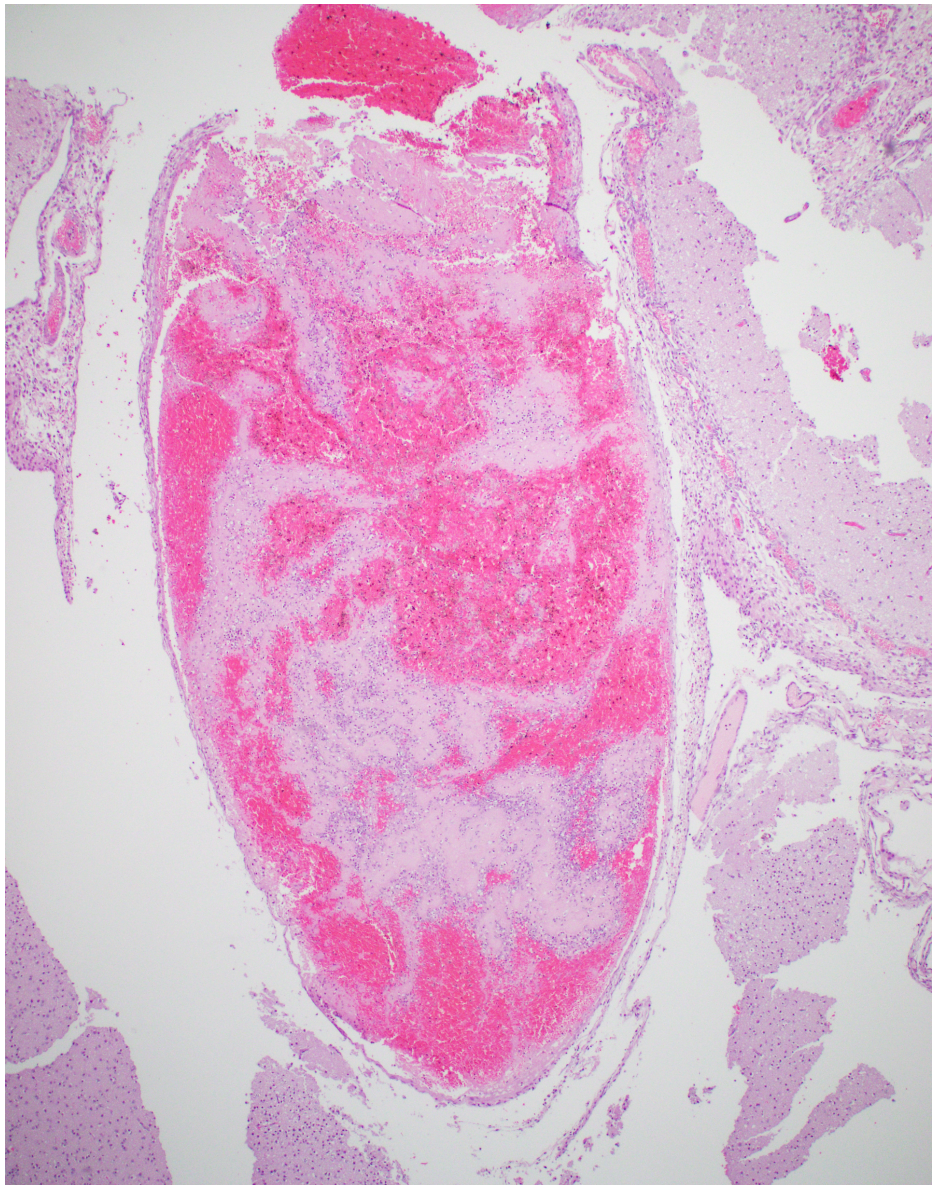


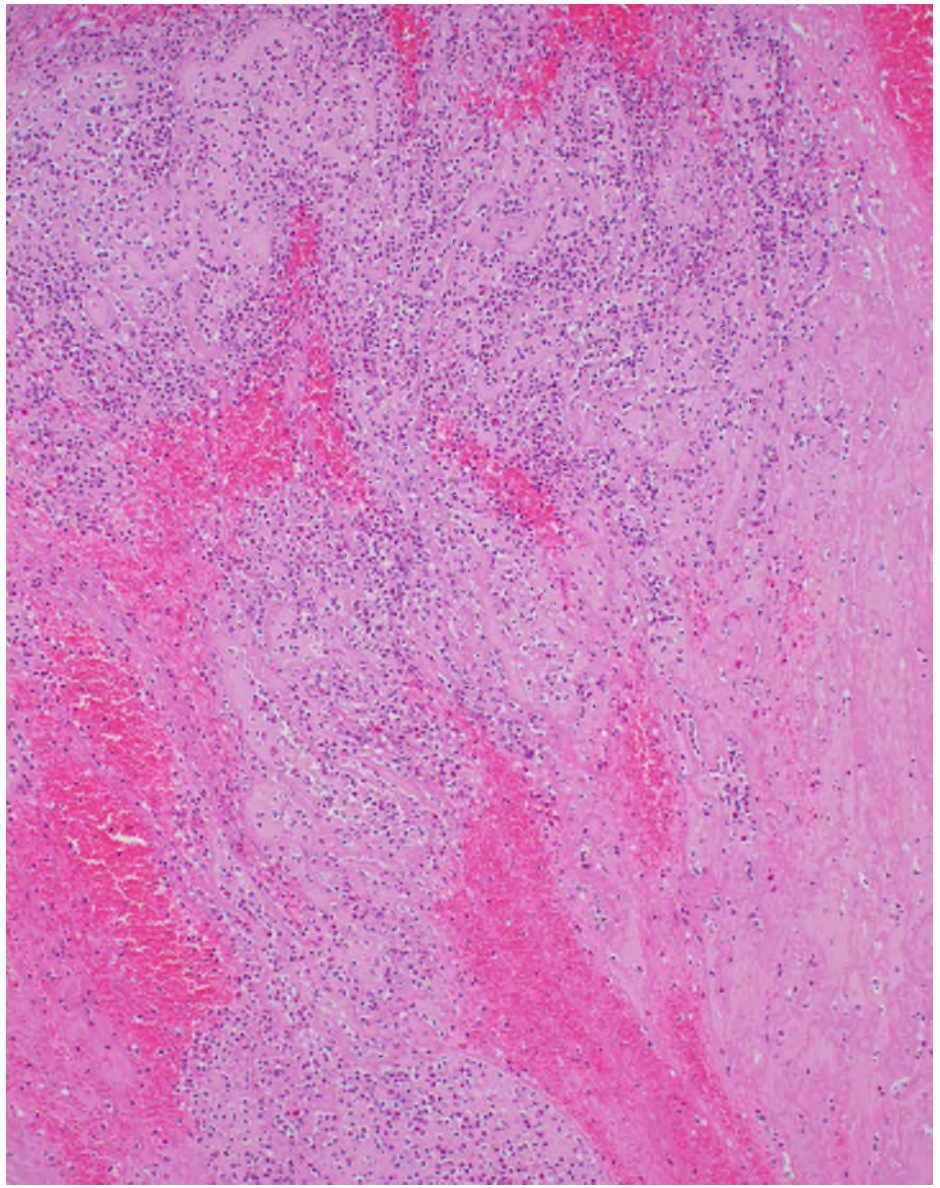


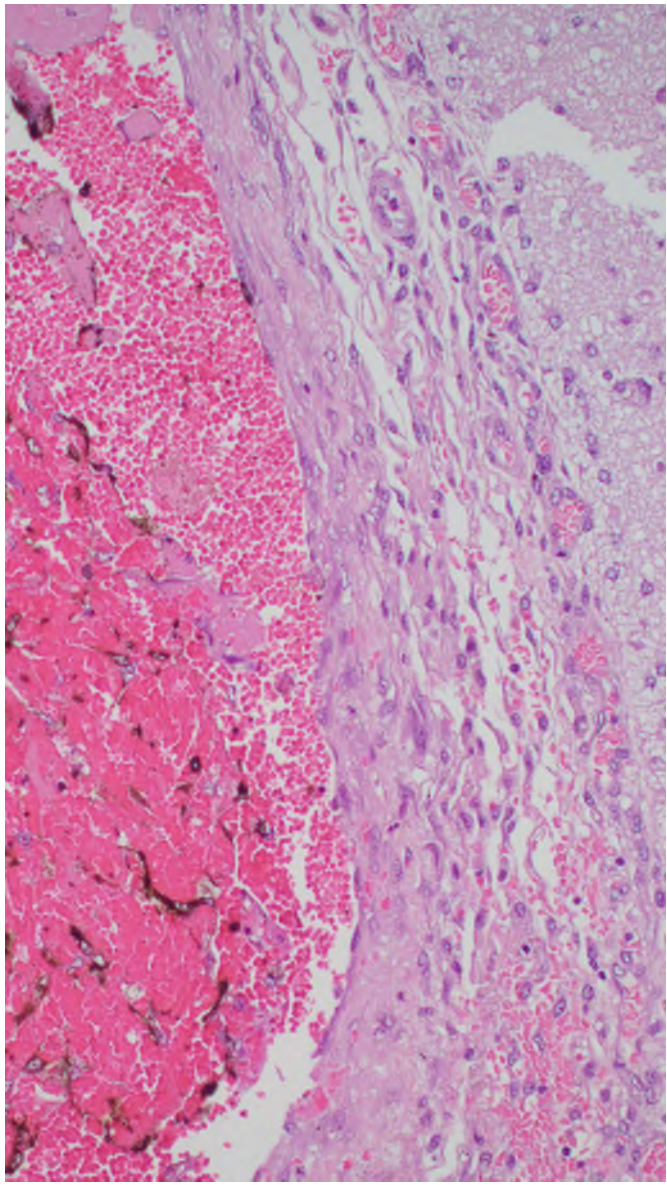




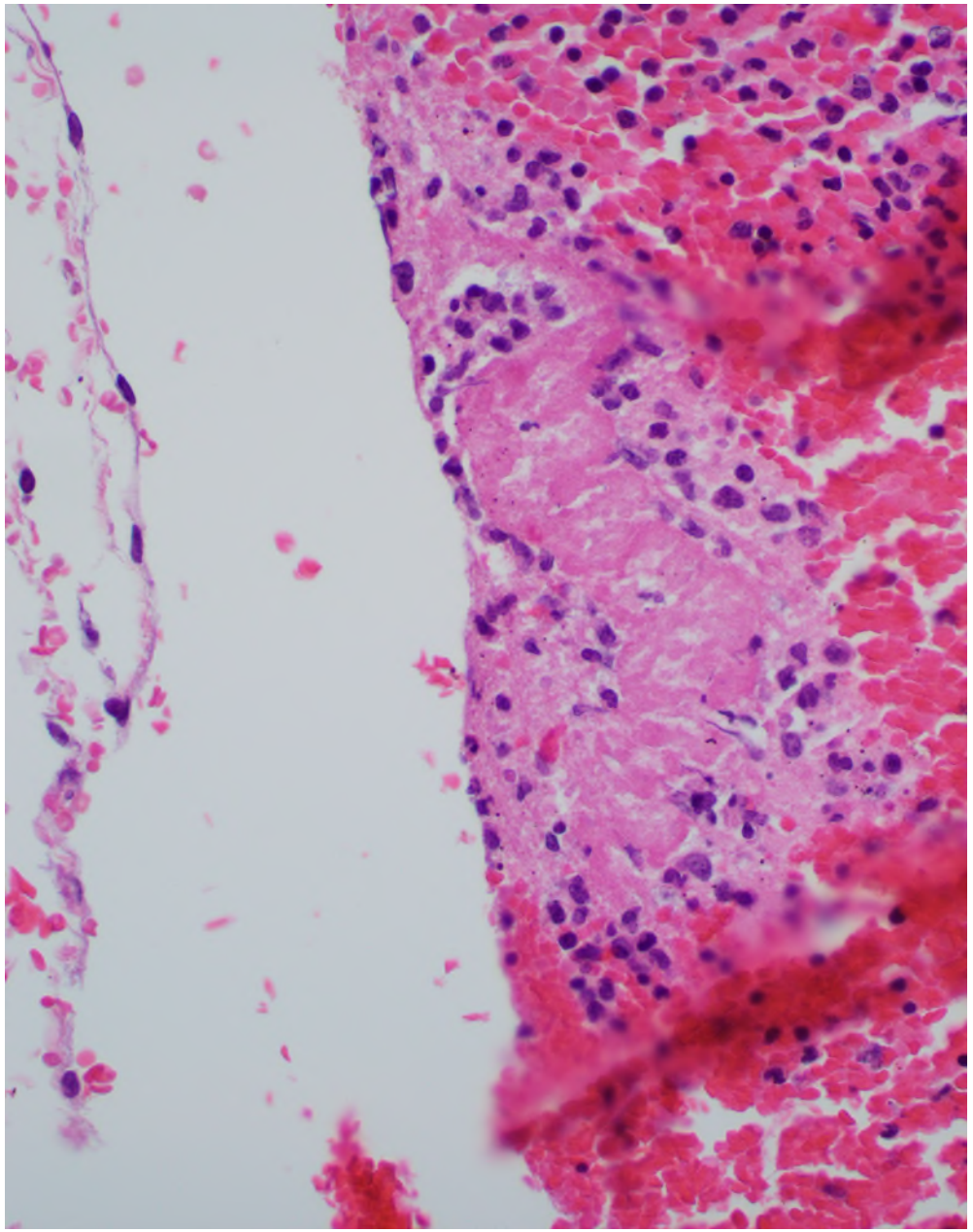
Case 16: Cortical vein thrombosis

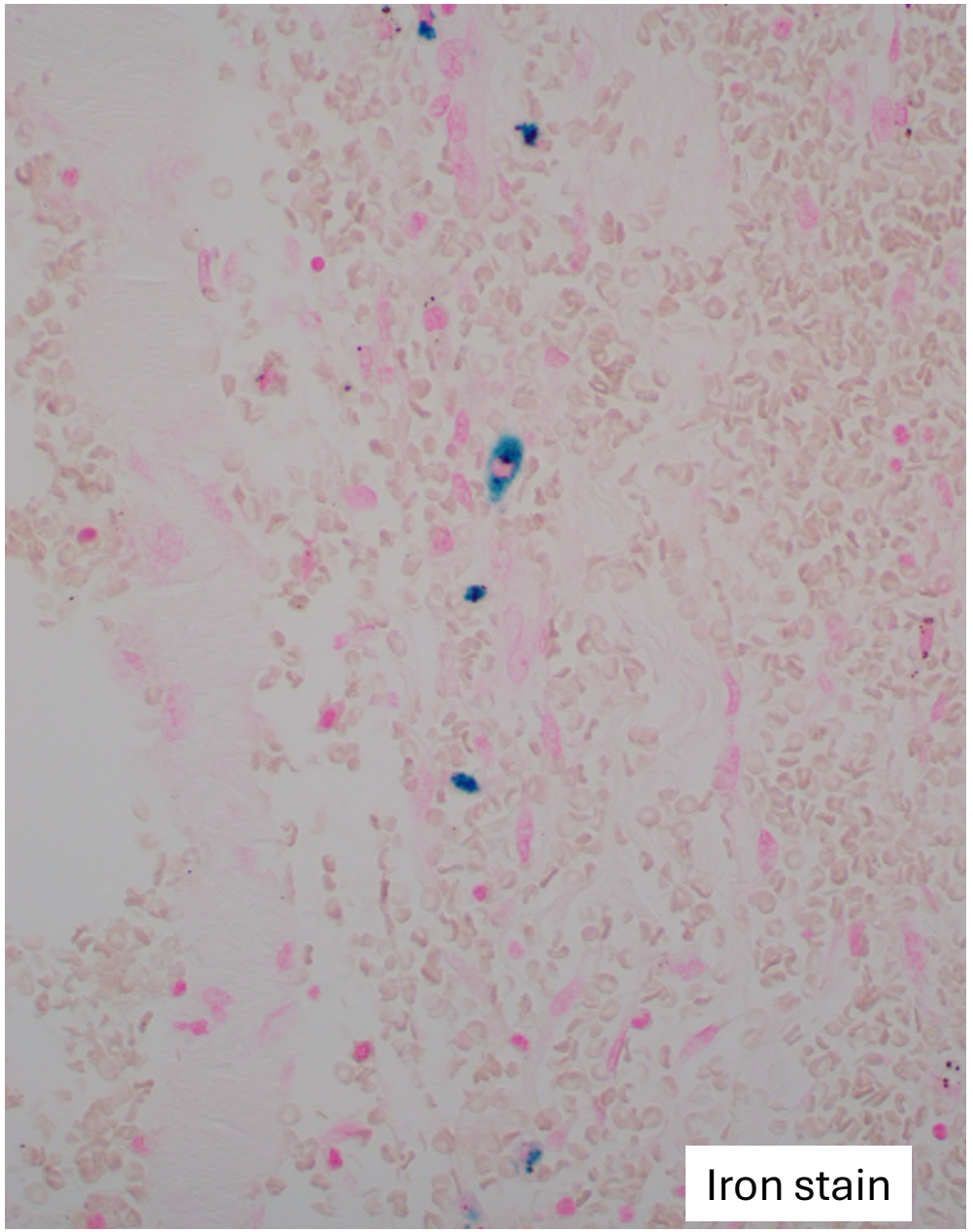






Case 16: Dural sinus clots





FNPDx:

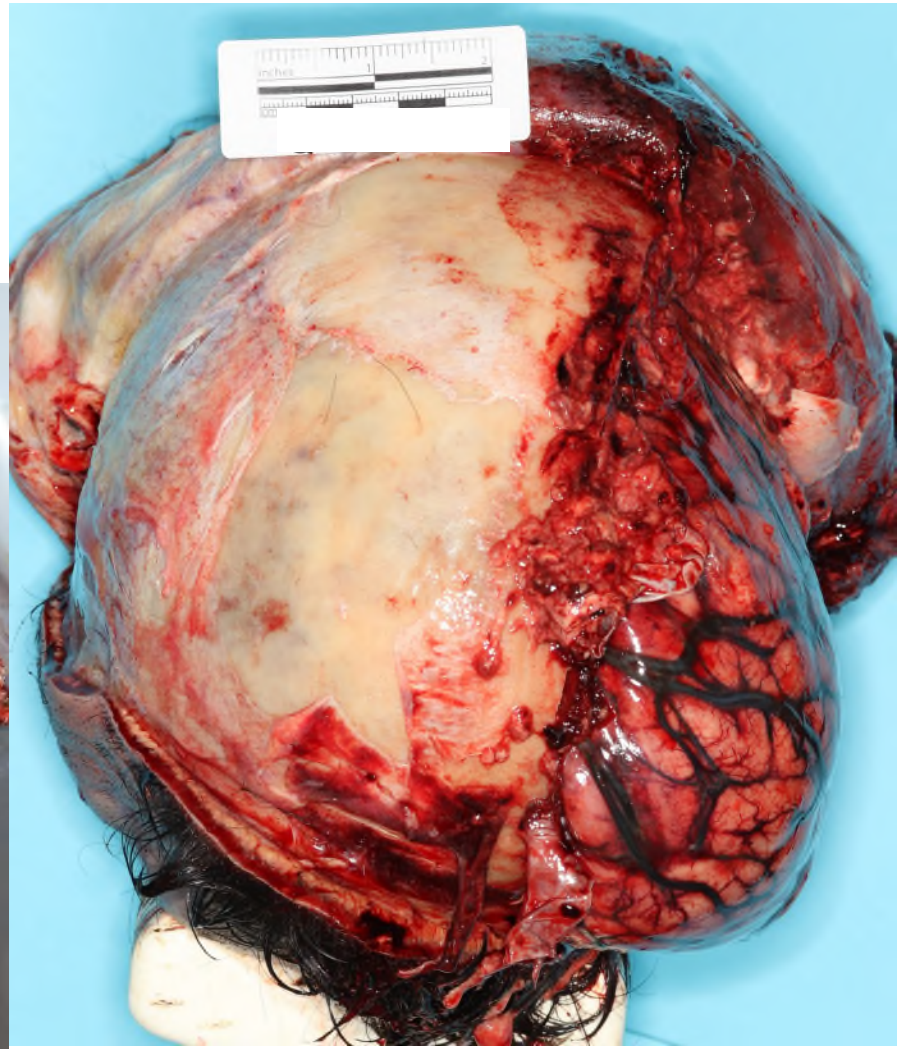
- Dural bridging vein and superficial cortical vein thrombosis, with early organization
  - Secondary venous hemorrhage in left frontal and temporal cortices
  - Secondary mural thrombi in dural venous sinus
  - Reactive changes in dura and leptomeninges
  - Brain swelling
  - Secondary acute neuronal ischemia, brain and spinal cord

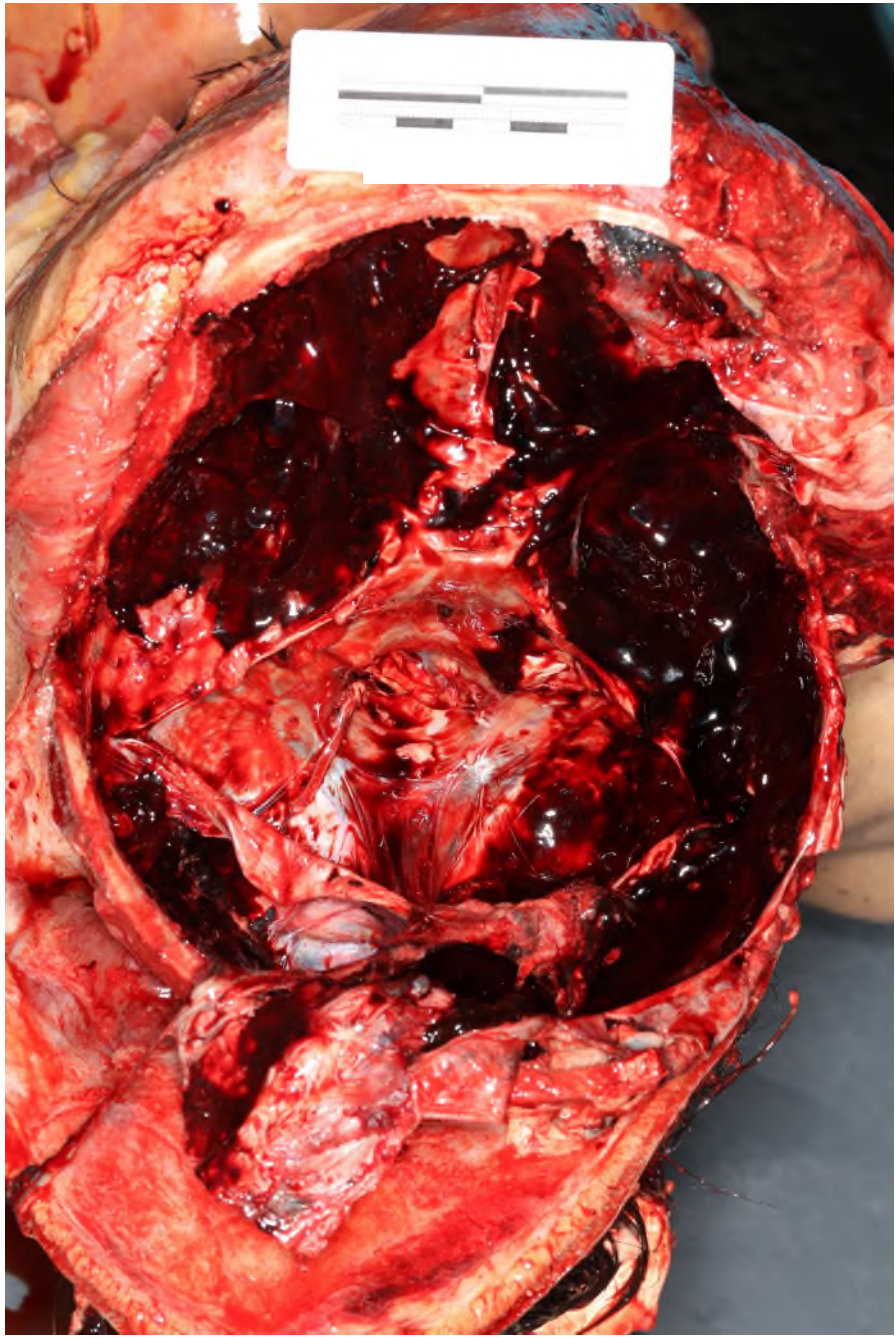
(Case 16)

Case 17: Acute TBI, s/p craniectomy

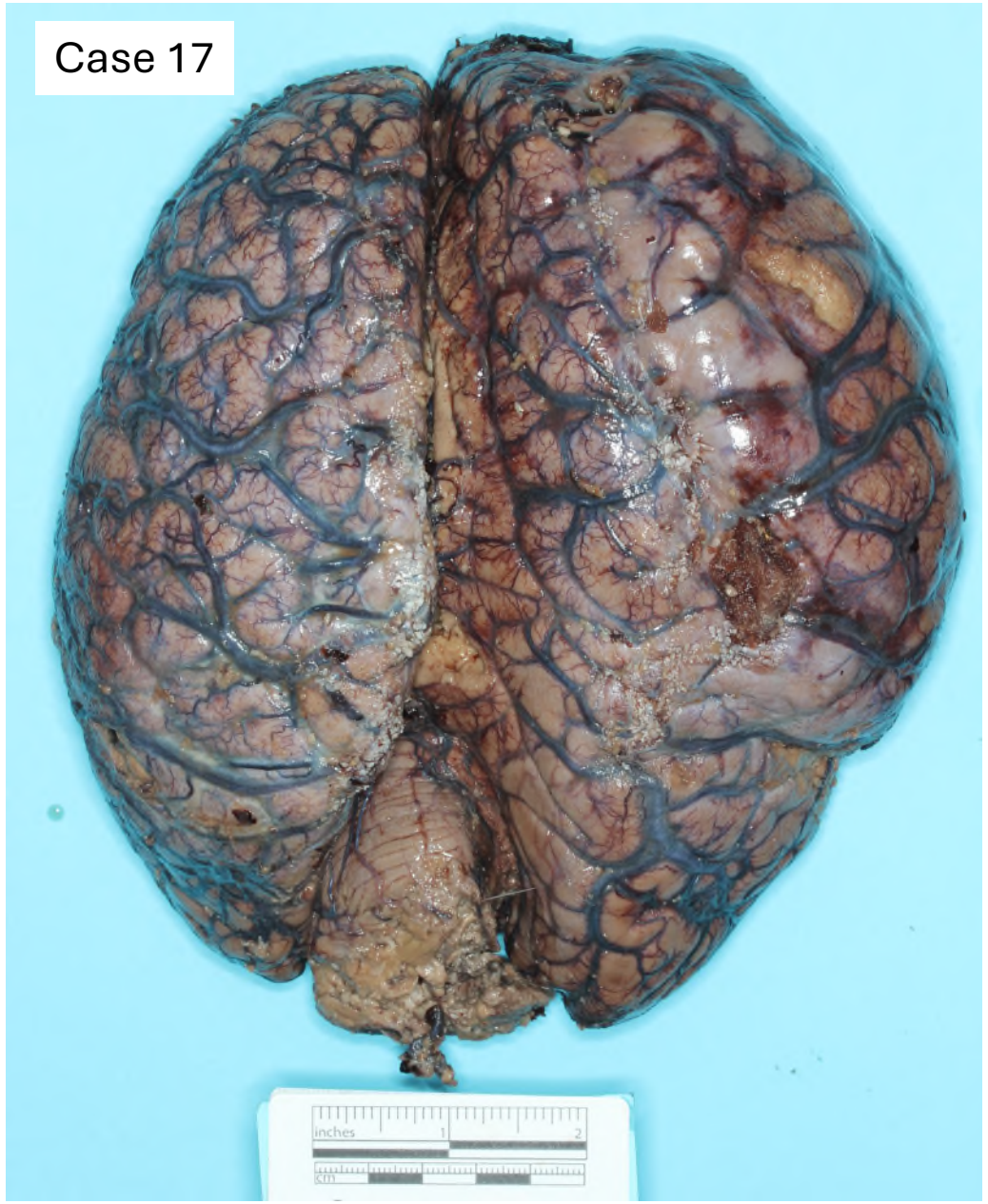
- Unknown male, found down outside
- Brought to ER, underwent craniectomy
- Survived 2 days, “brain death” protocol

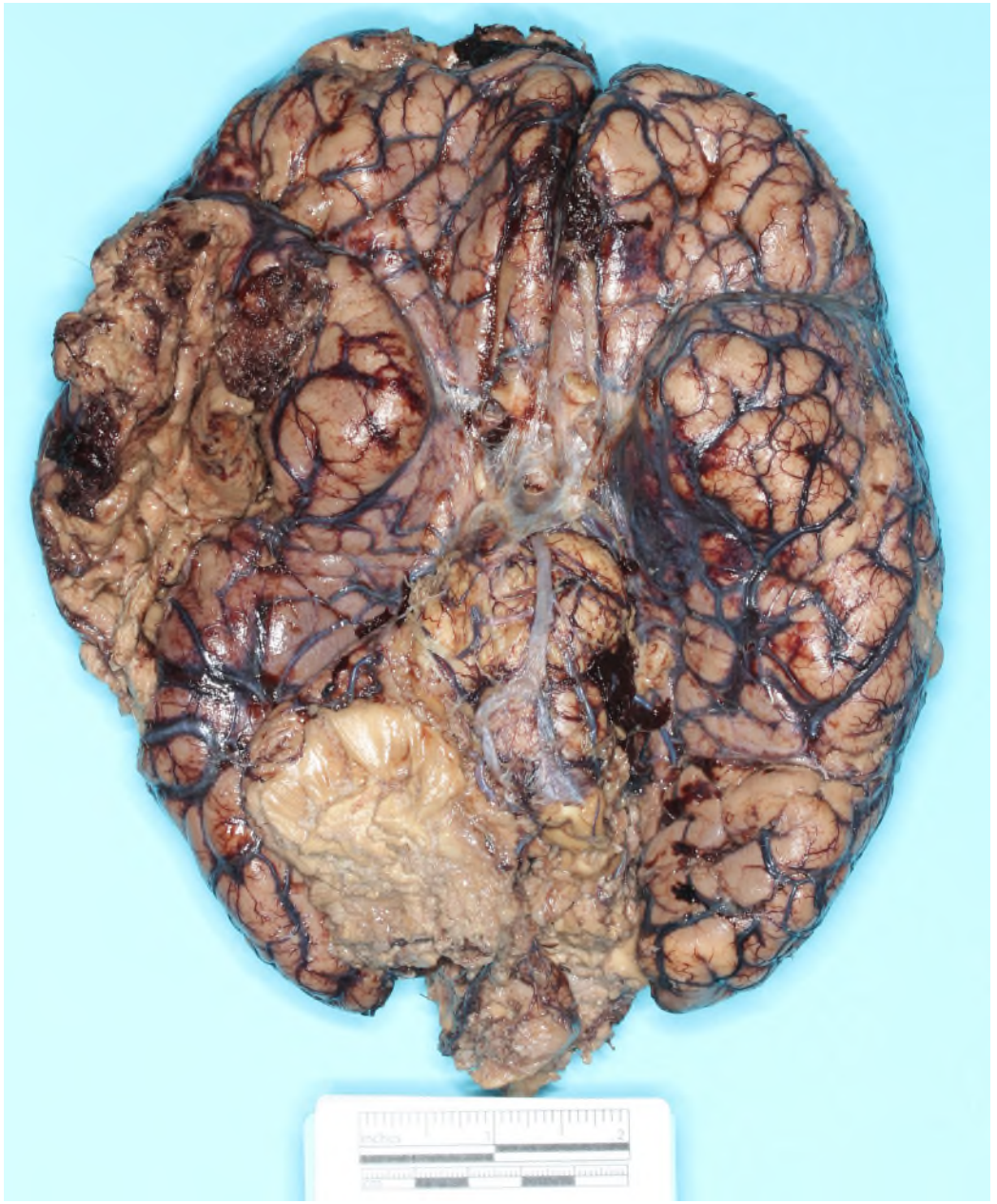
Case 17

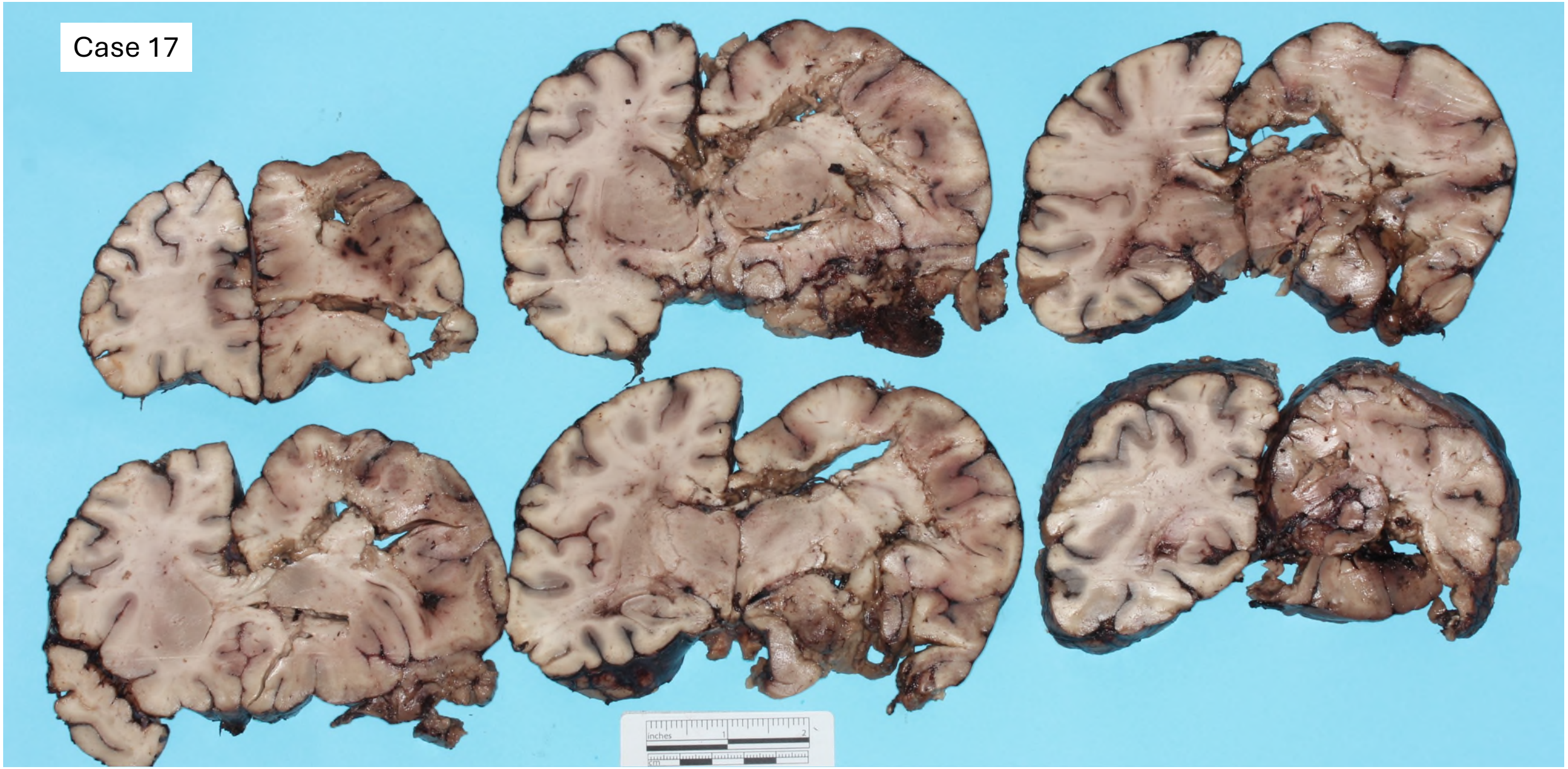




Case 17






Case 17
inches
1
2
cm

Case 17

FNPDx:

- Blunt impact injury of head, recent:
  - Nondisplaced fracture of R occipital skull *(not shown)*
  - S/P neurosurgical interventions:
    - R hemicraniectomy and evacuation of subdural hematoma
      - External R frontoparieto-occipital hernia
        - Secondary marginal compression ischemia with focal reperfusion hemorrhage ("herniation contusions")
    - R external ventricular drain placement
      - Focal residual intraventricular blood clot
  - Residual subdural hemorrhage
  - Subarachnoid hemorrhage
  - Diffuse hypoxia-ischemia with autolysis*

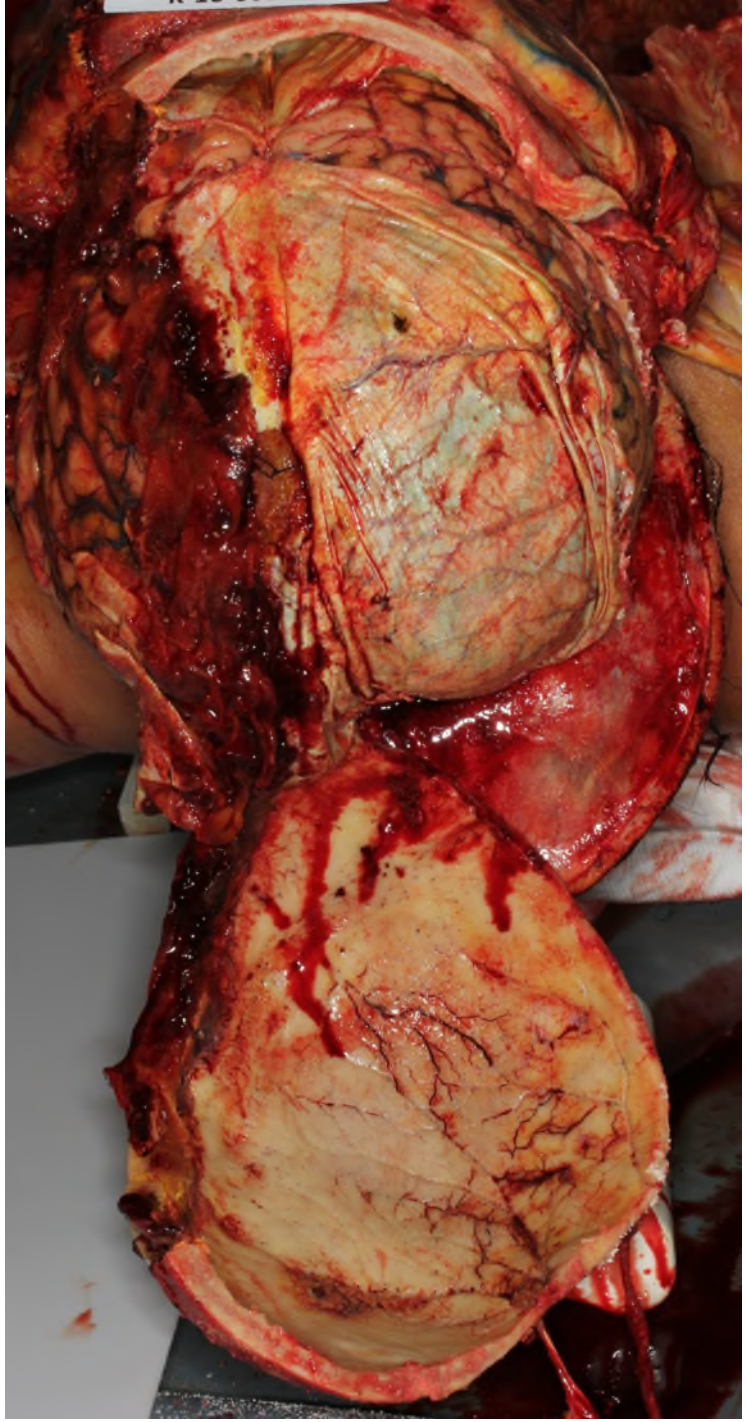
(Case 17)

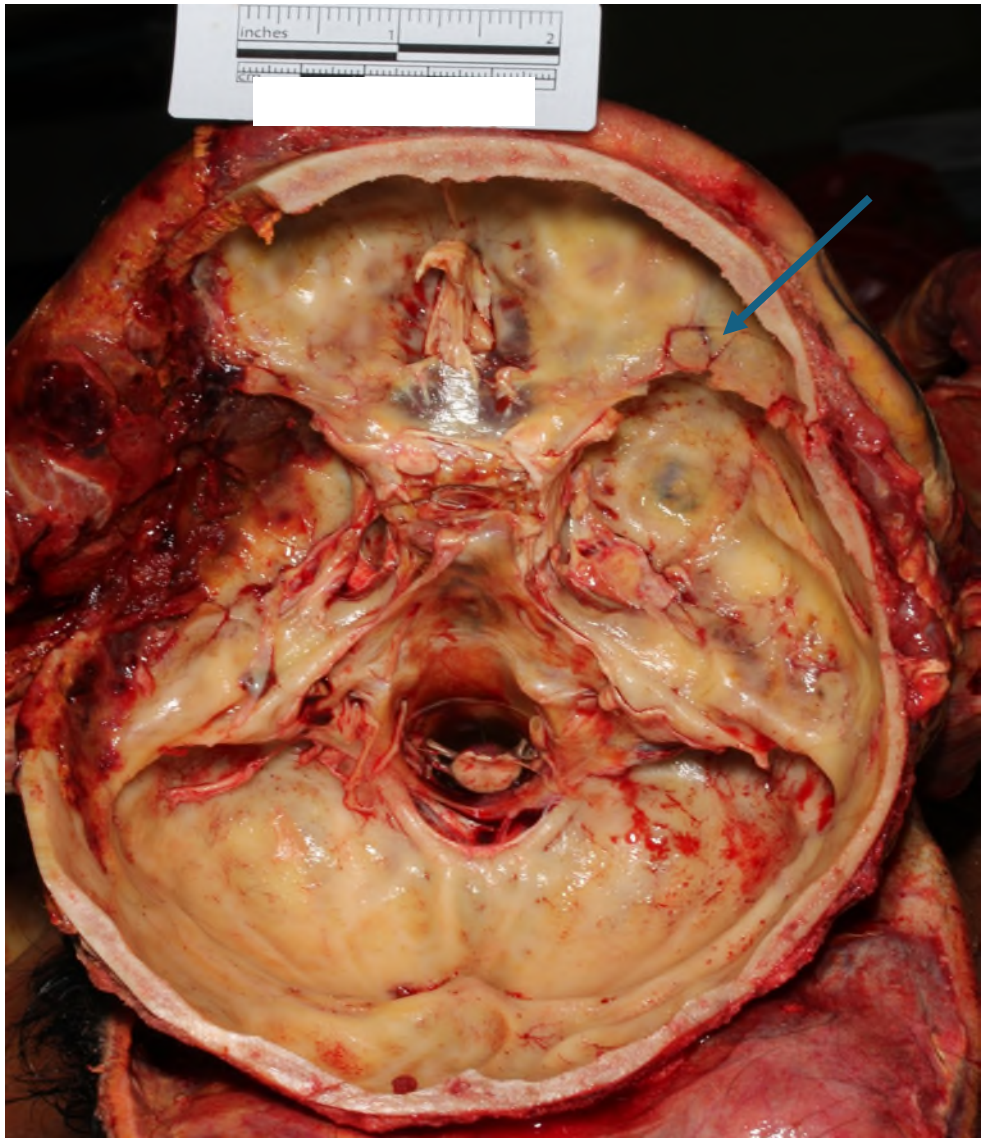
**autolysis, "brain death" features*

Case 18: Old TBI, s/p craniectomy

- Unknown male, 20-40yo F
- Found unresponsive on street
- High BAL, GCS 13
- CT → R scalp hematoma, R temporal fx, L SDH
- L hemicraniectomy, R external ventricular drain placement
- Eventual sepsis and death after 1.5mos

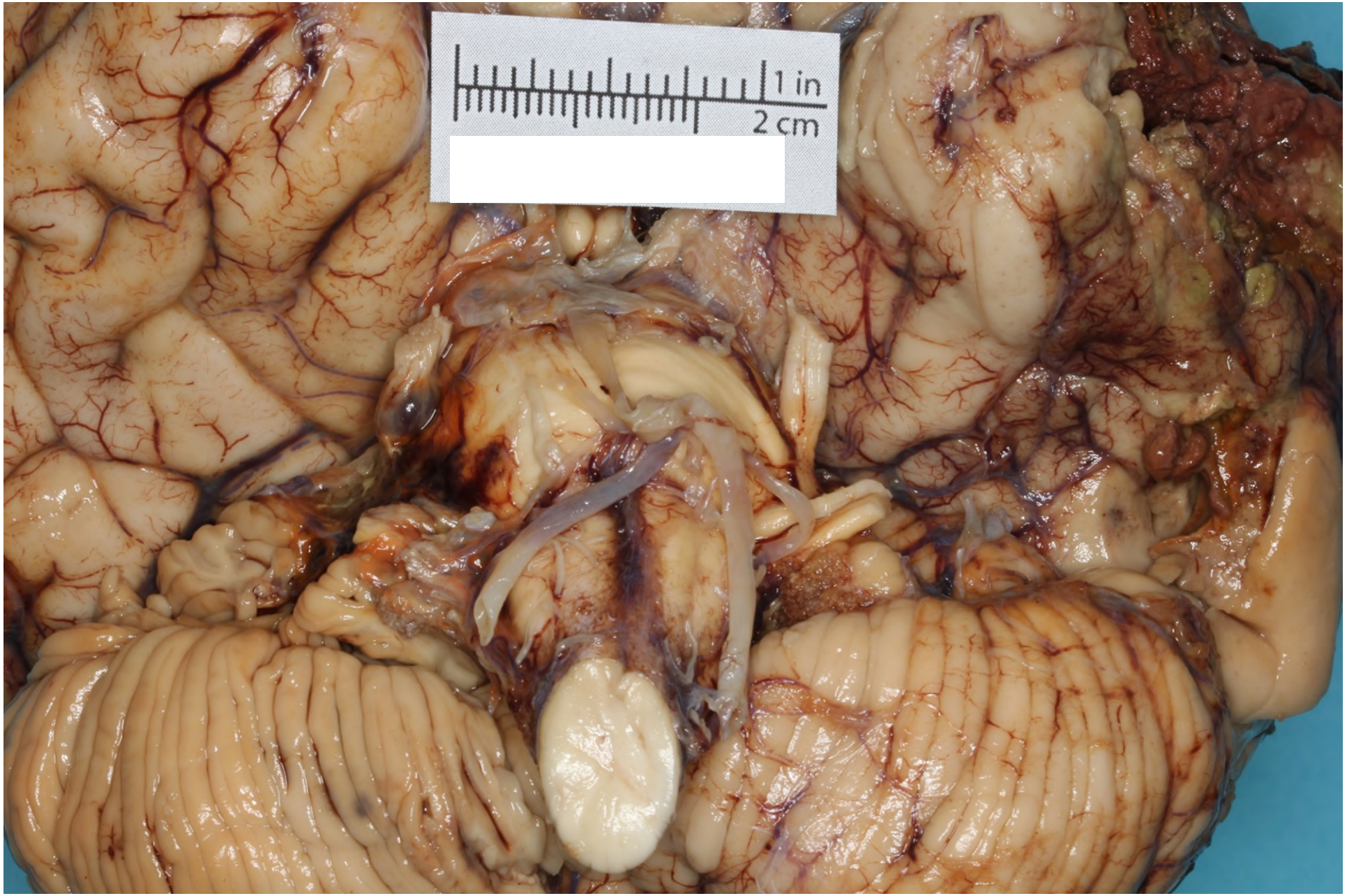
Case 18



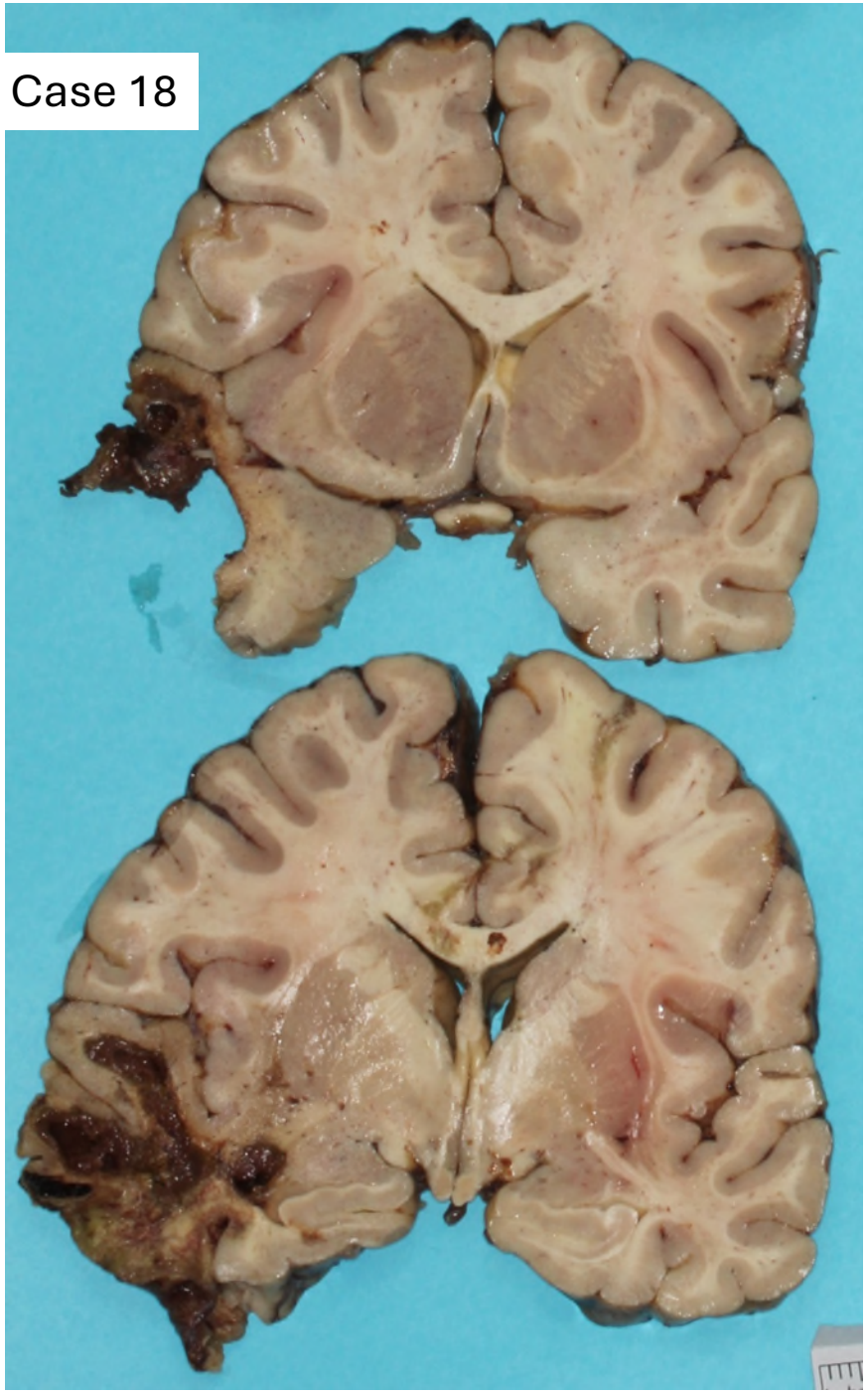


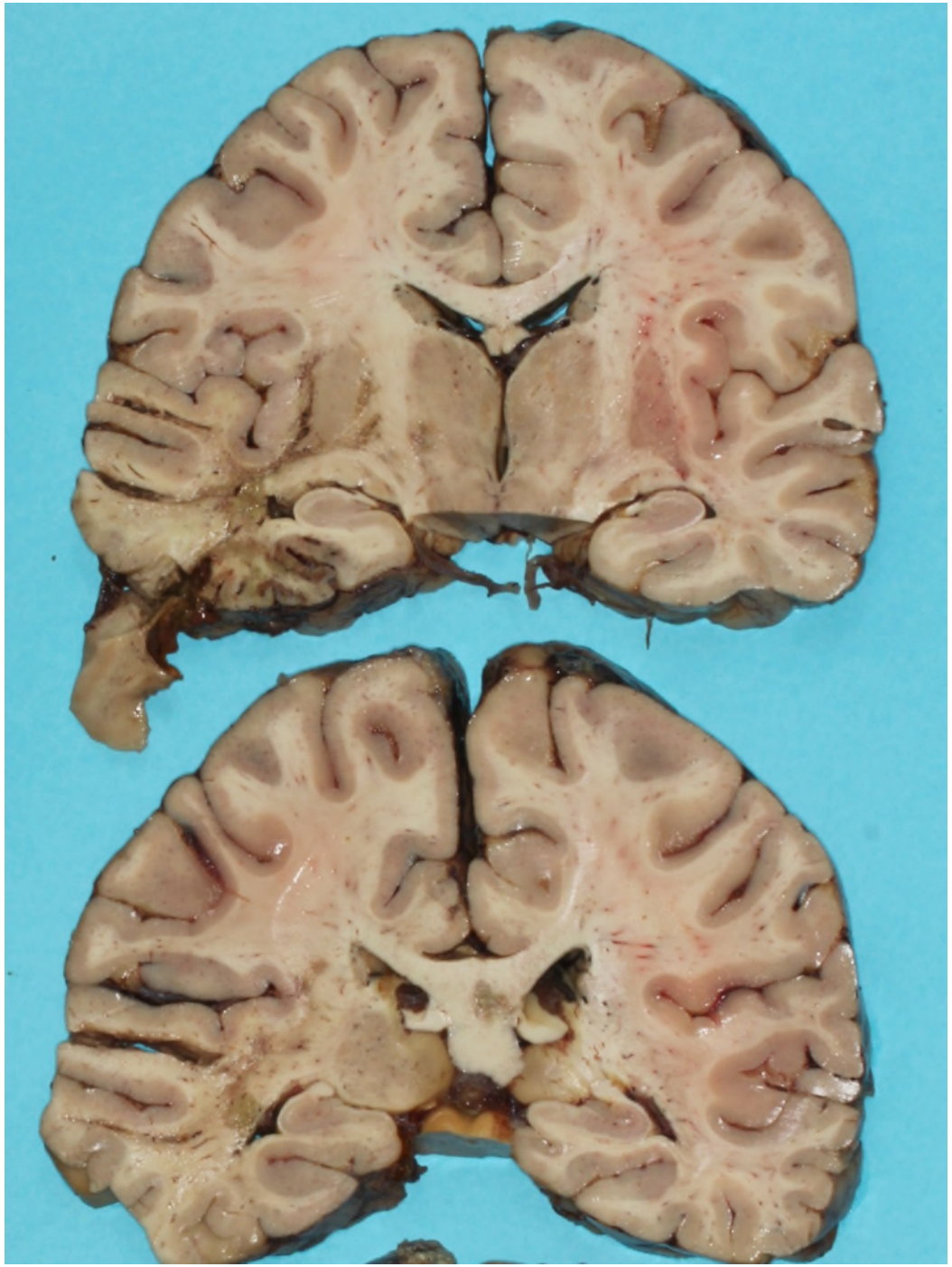
Case 18

Case 18



Case 18





Case 18

FNPDx:

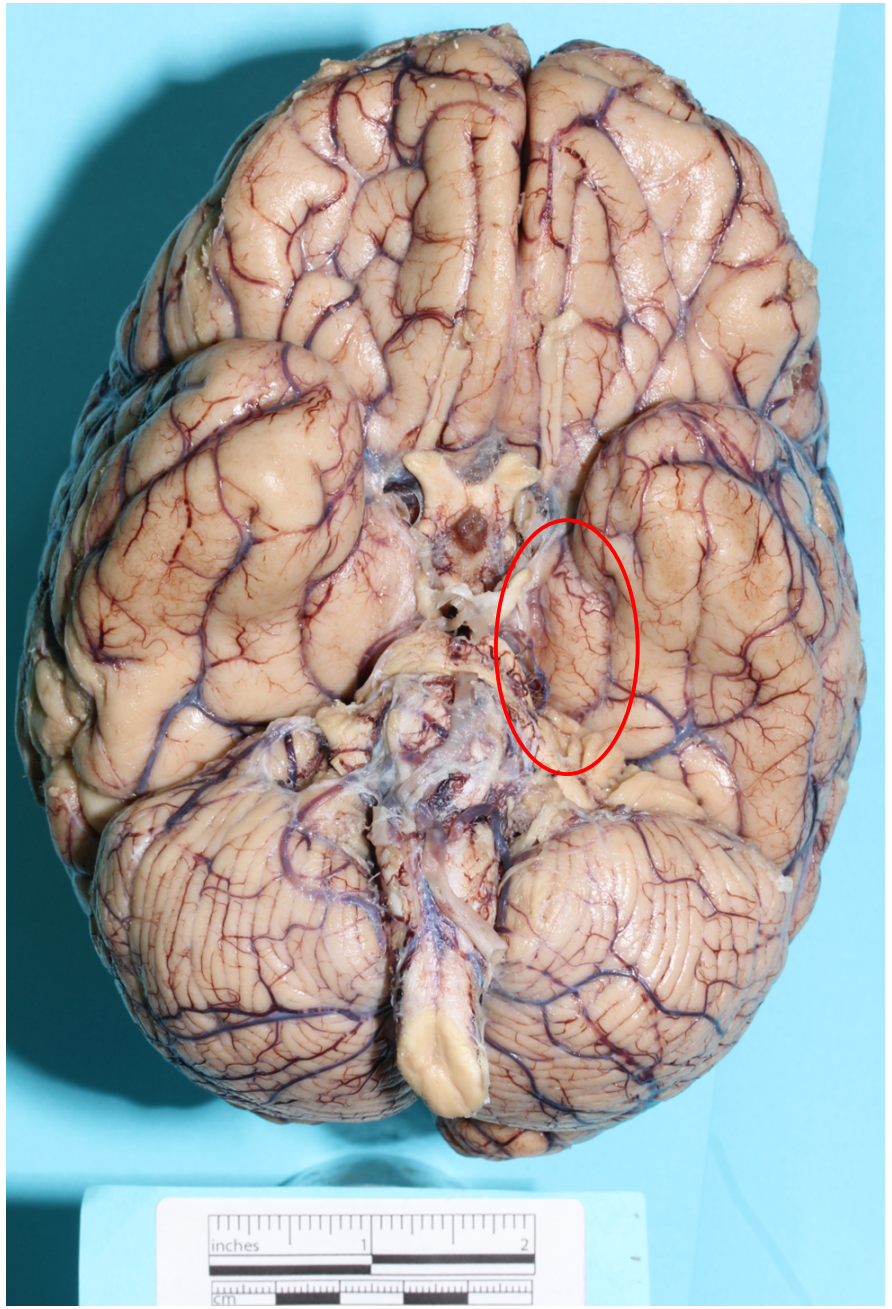
- Blunt impact injury of head, old (1.5mo prior to death), with:
  - R temporal skull fracture
  - Organizing L temporal contusion hematoma
  - S/P neurosurgical interventions:
    - L hemicraniectomy and evacuation of L subdural hematoma, with:
      - Secondary organizing compression ischemia with reperfusion hemorrhage (“herniation contusions”) involving:
        - L temporal lobe at margins of craniectomy
        - R lateral midbrain and pons (“Kernohan’s notch phenomenon”)
      - Residual organizing subdural hemorrhage
    - R external ventricular drain placement with organizing track in frontal cerebral mantle and corpus callosum

(Case 18)

Case 19: Old TBI

- 27yo M
- Hx of TBI from assault 10yrs prior, now wheelchair-bound, with seizures
- Found dead at home

Case 19




Case 19
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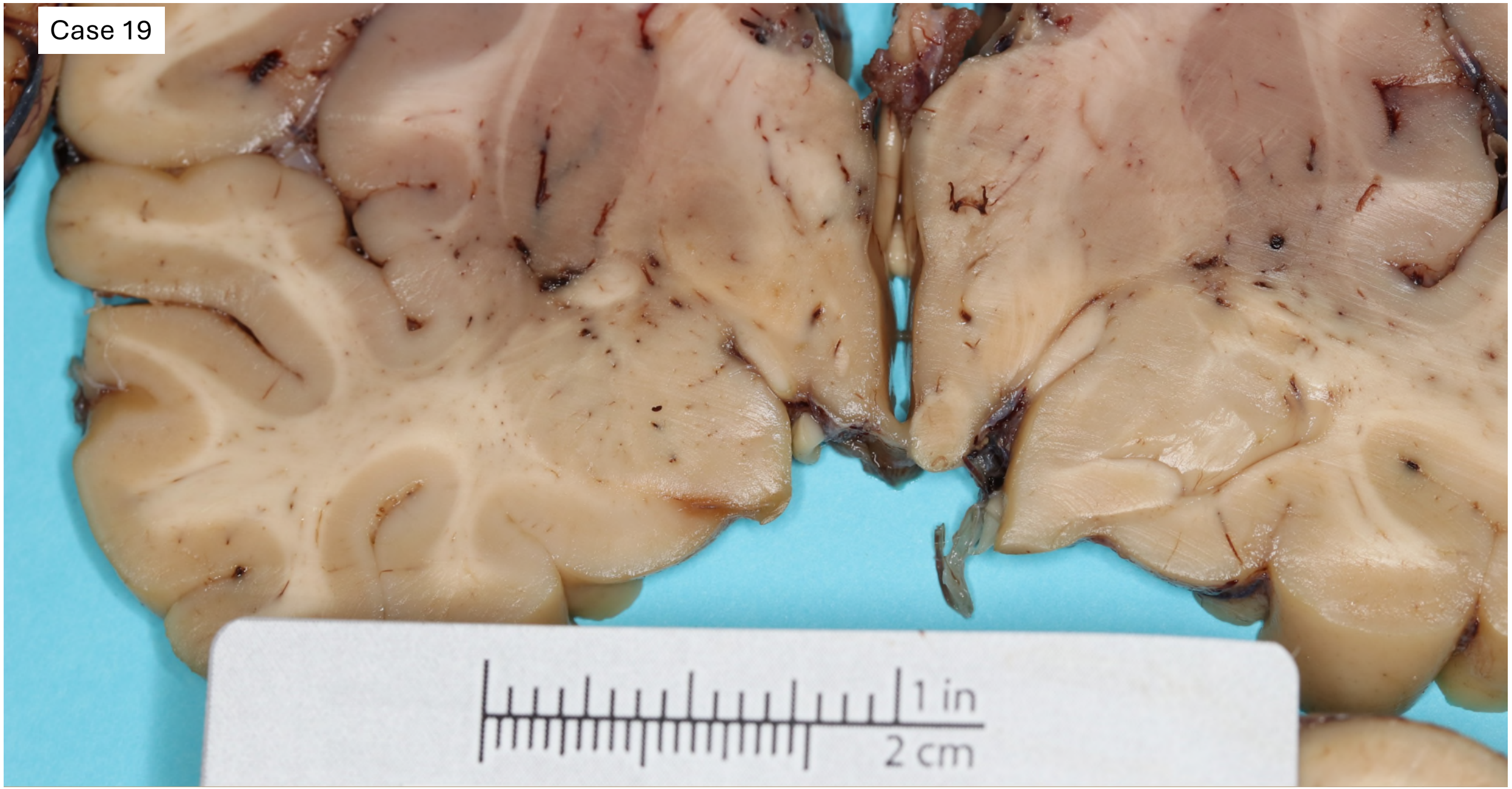
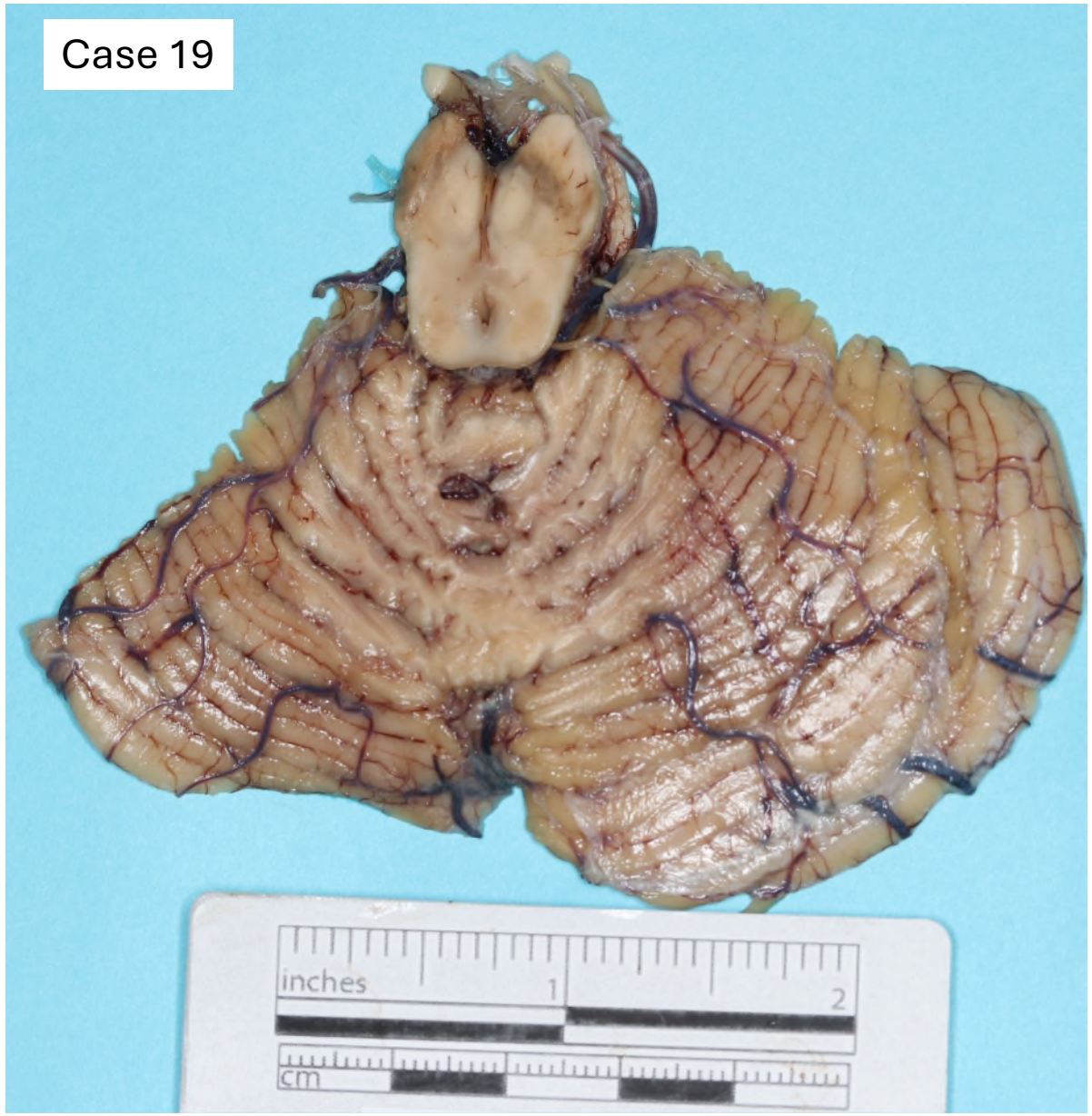

Case 19
1 in
2 cm

Case 19



Case 19

Final NP Dx

- (History of blunt force trauma of head, old) (see autopsy report)
  - Tentorial notch (“herniation”) contusions, left medial temporal lobe
  - Secondary atrophy of left cerebral peduncle, basis pontis, and pyramid, and of right lateral spinal funiculi

(Case 19)

Cause and manner of death????

Final NP Dx

- (History of blunt force trauma of head, old) (see autopsy report)
  - Tentorial notch (“herniation”) contusions, left medial temporal lobe
  - Secondary atrophy of left cerebral peduncle, basis pontis, and pyramid, and of right lateral spinal funiculi

(Case 19)

Cause and manner of death????

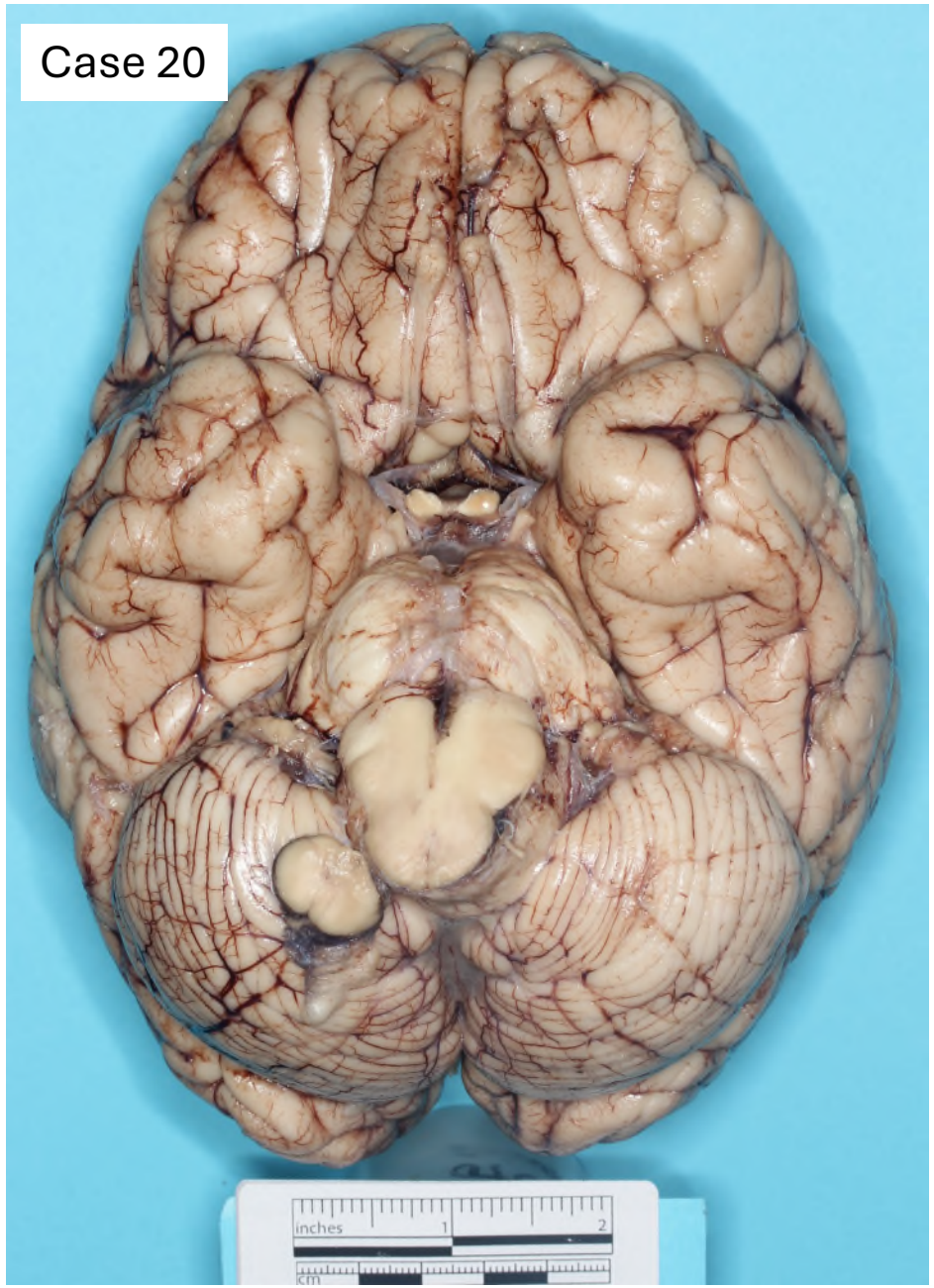
-PE, complicating hemiparesis due to old blunt force trauma of head

- Homicide

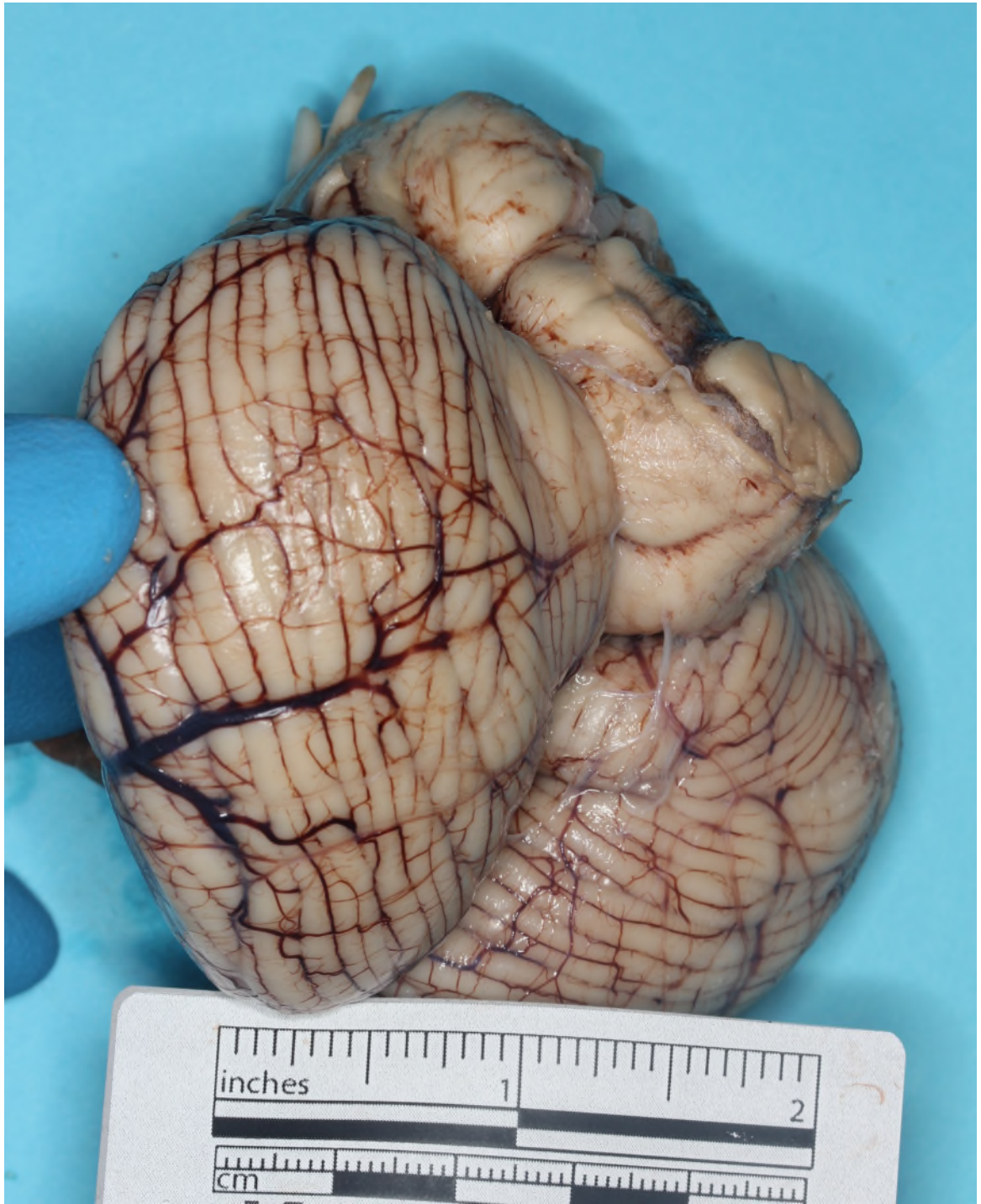
Case 20: Undiagnosed tumor

- 3yo boy, choked on PB&J
  - *Suspicion raised when caregiver says “choked”…..*
- Autopsy completely negative, except for brain
- Histology c/w angiocentric glioma

Case 20



Case 20



Case 20

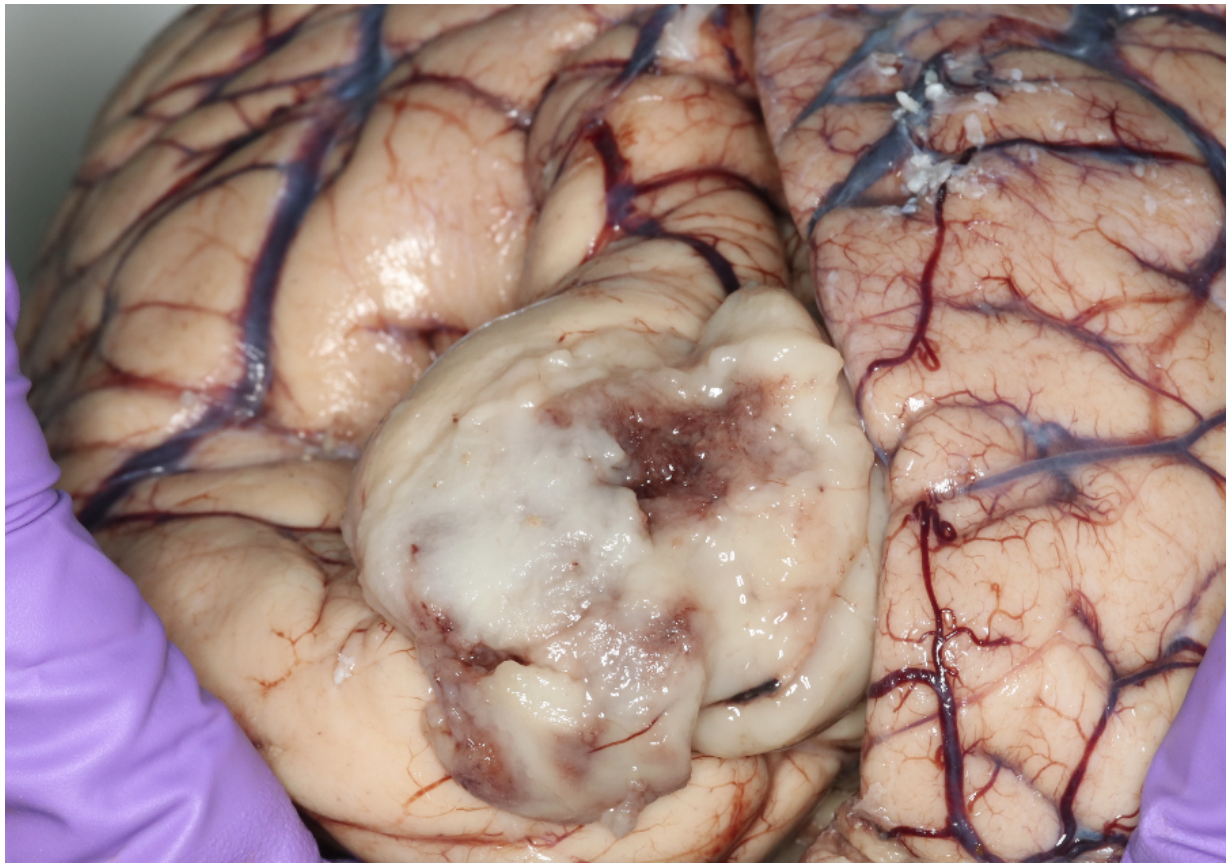

Case 20
inches
1
2
cm

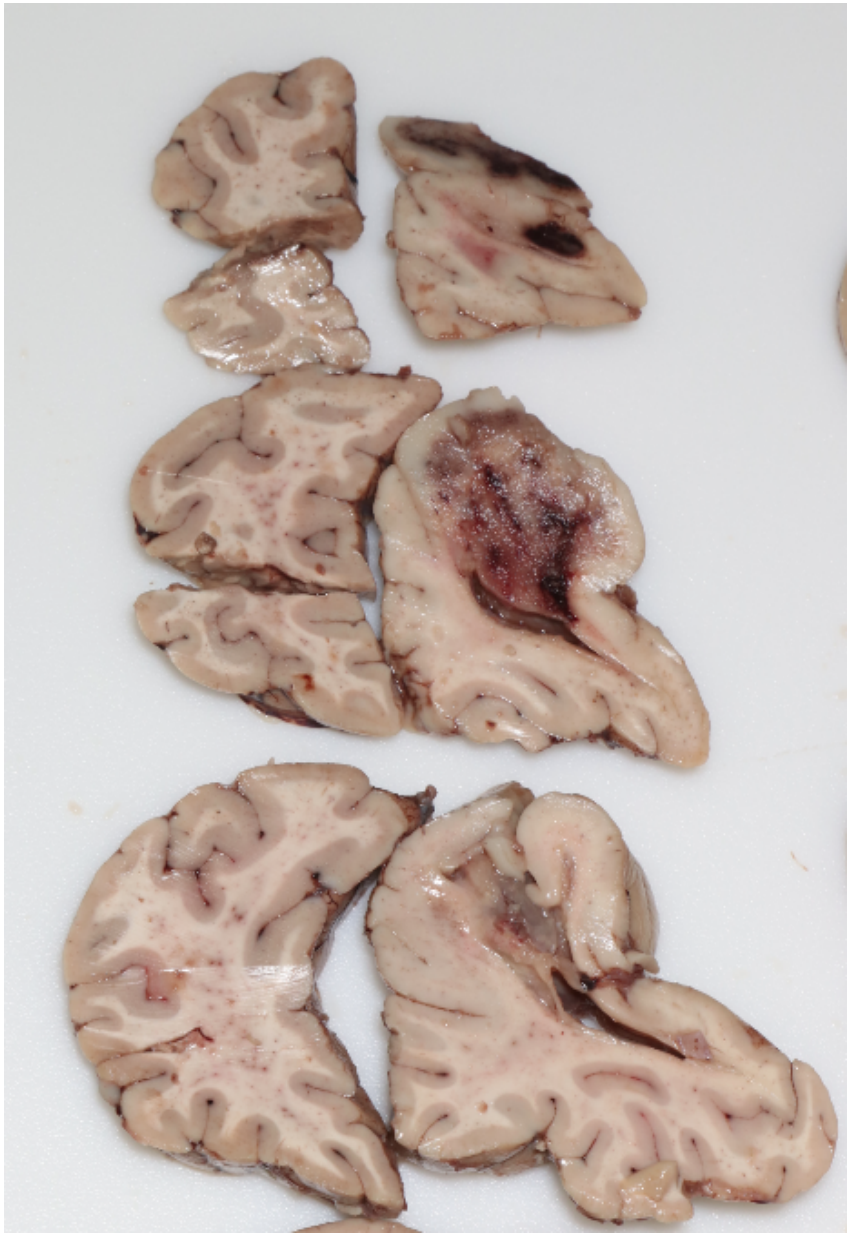

inches
1
2
cm

Case 21: Undiagnosed tumor

- 24-year-old
- Complaints of headaches in the days prior to death
- Found dead in locked/secured room
- Histology was most suggestive of an anaplastic oligo, with acute intratumoral hemorrhage

Case 21

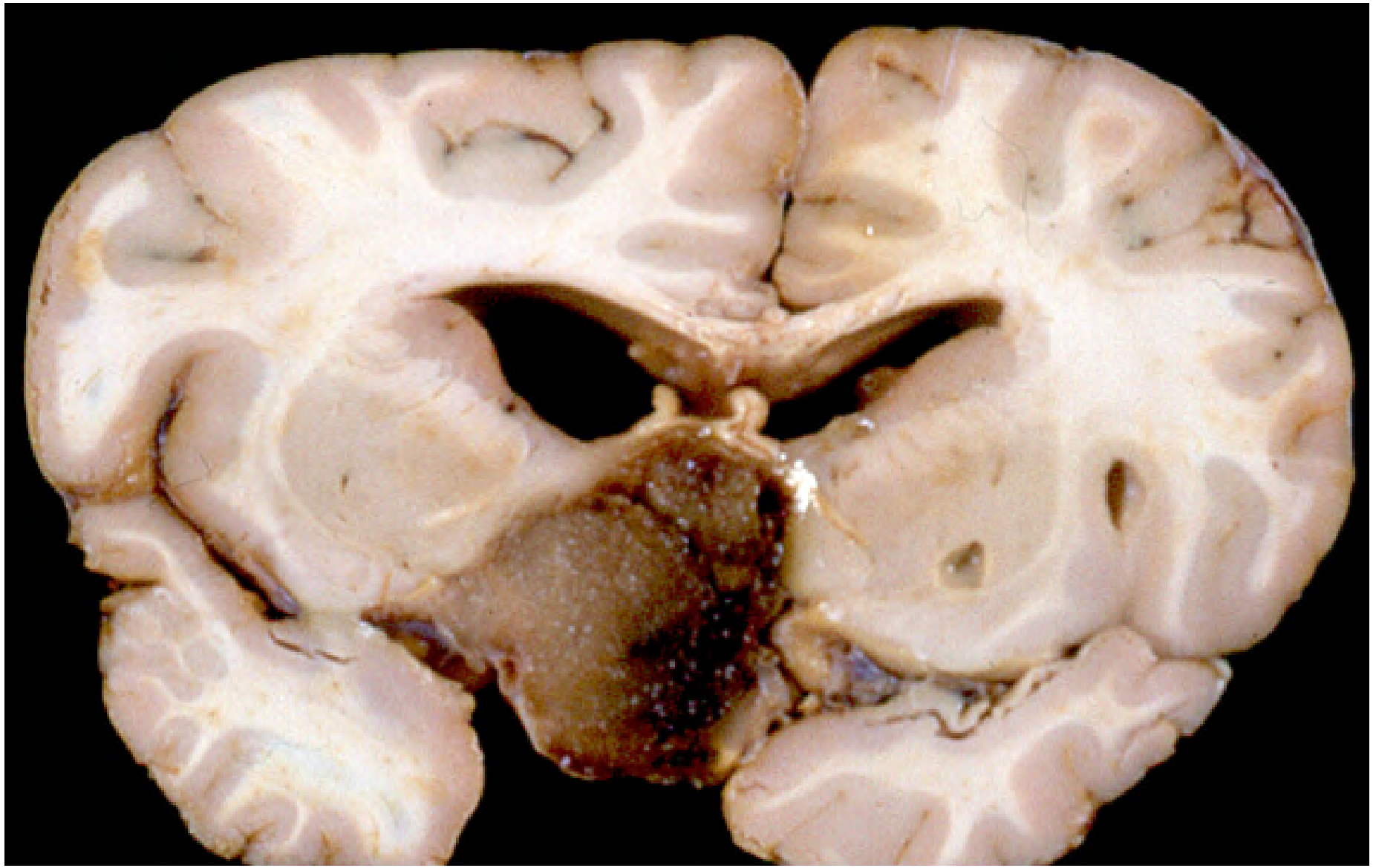




Courtesy Dr. H. Maioli, NYC OCME

Case 22: 7yo with hx of pilocytic astrocytoma of optic system, found dead at home:

- Acute intratumoral hemorrhage causing sudden death

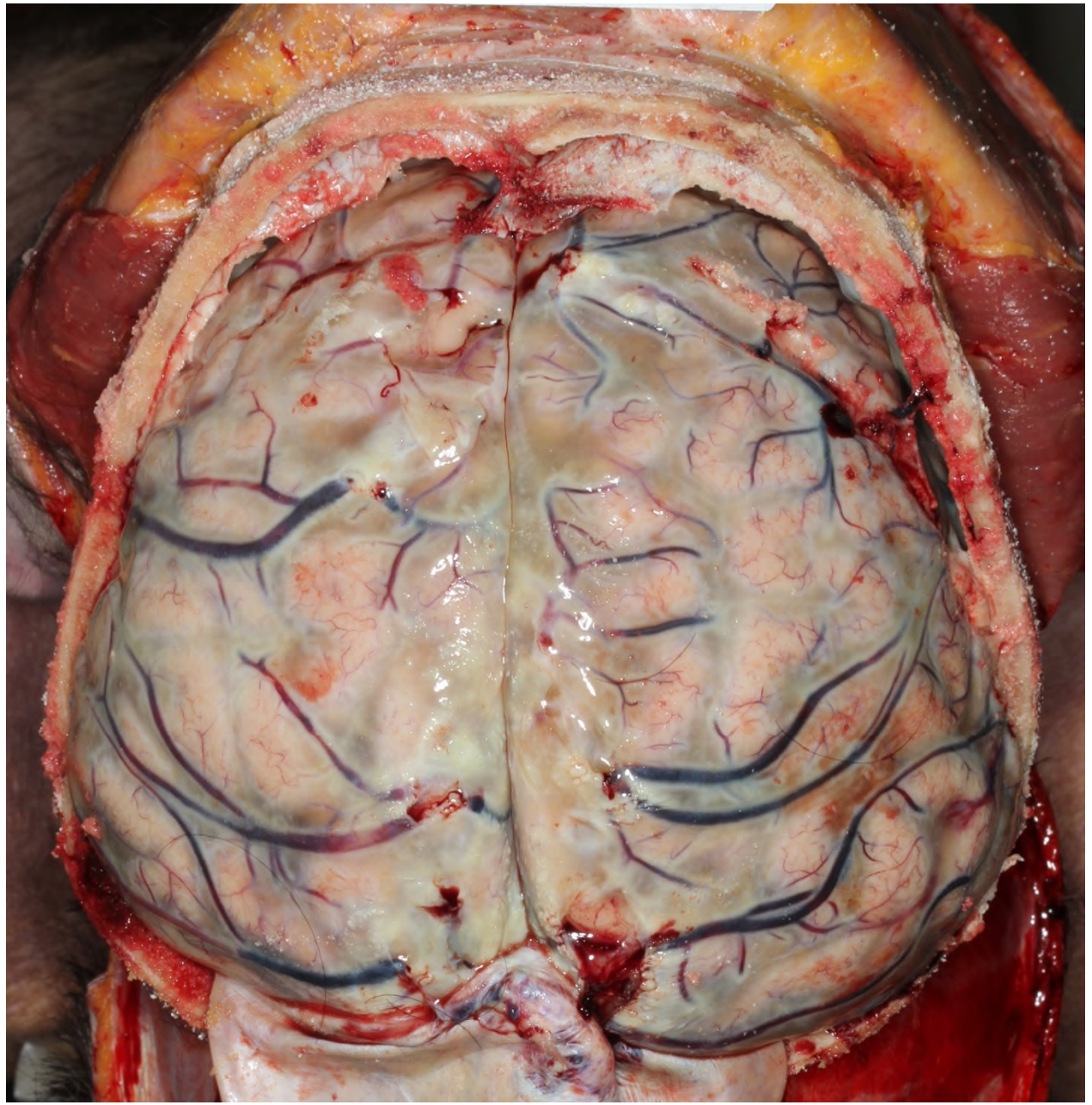


Escourolle&Poirier

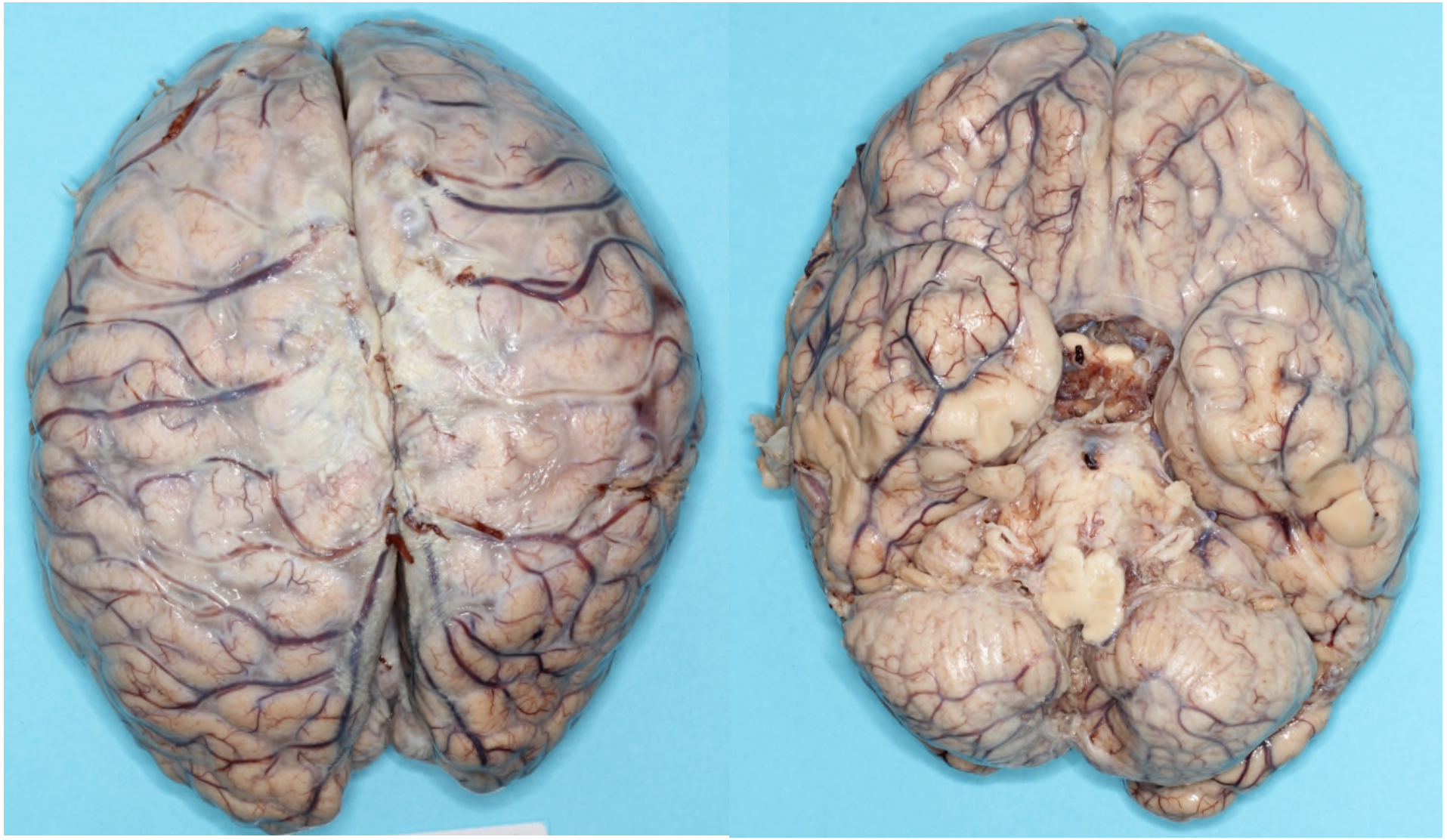
Case 23: Undiagnosed infection

- 46M, alcohol and tobacco abuse
- Found dead inside locked SRO bedroom by neighbor
- Pneumococcal pneumonia at autopsy

Case 23



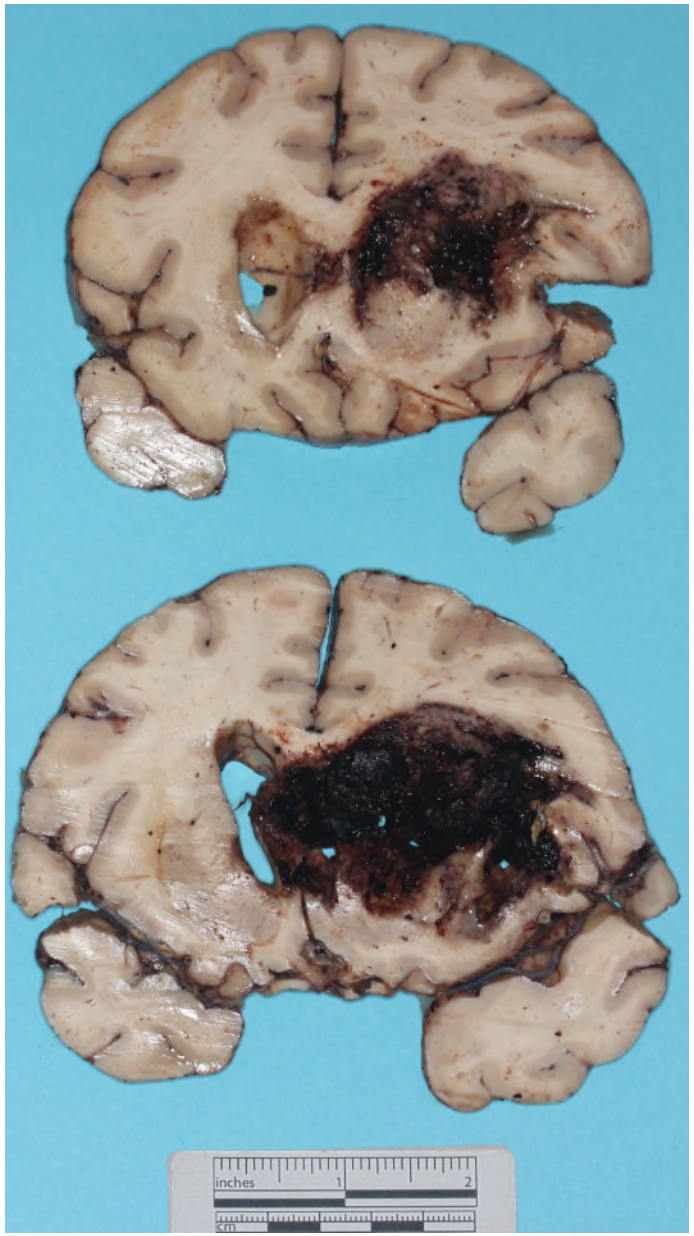
Case 23

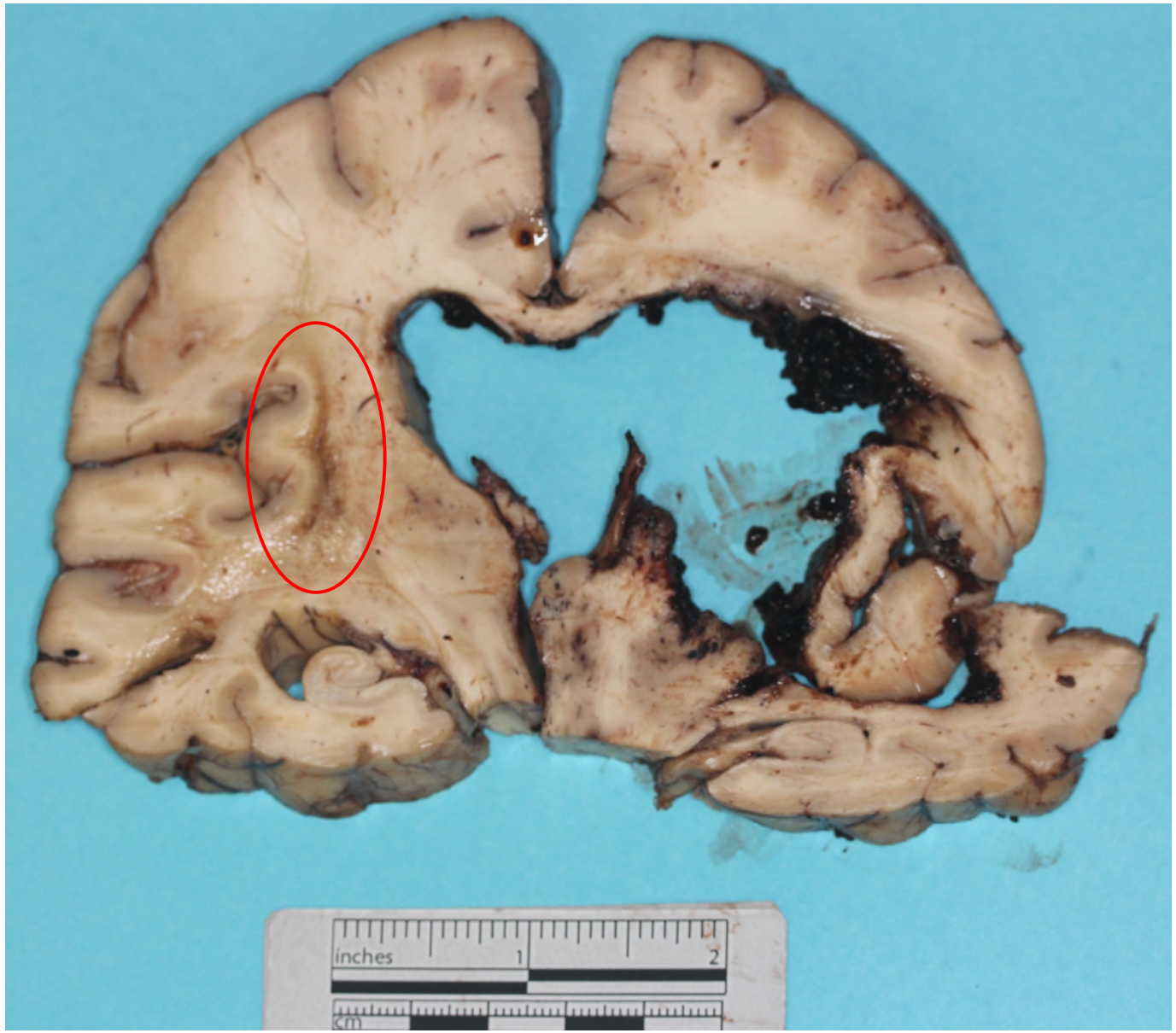


Case 24: Hypertensive ICH

- 79yo found dead at home
- Medications at scene include anti-hypertensives

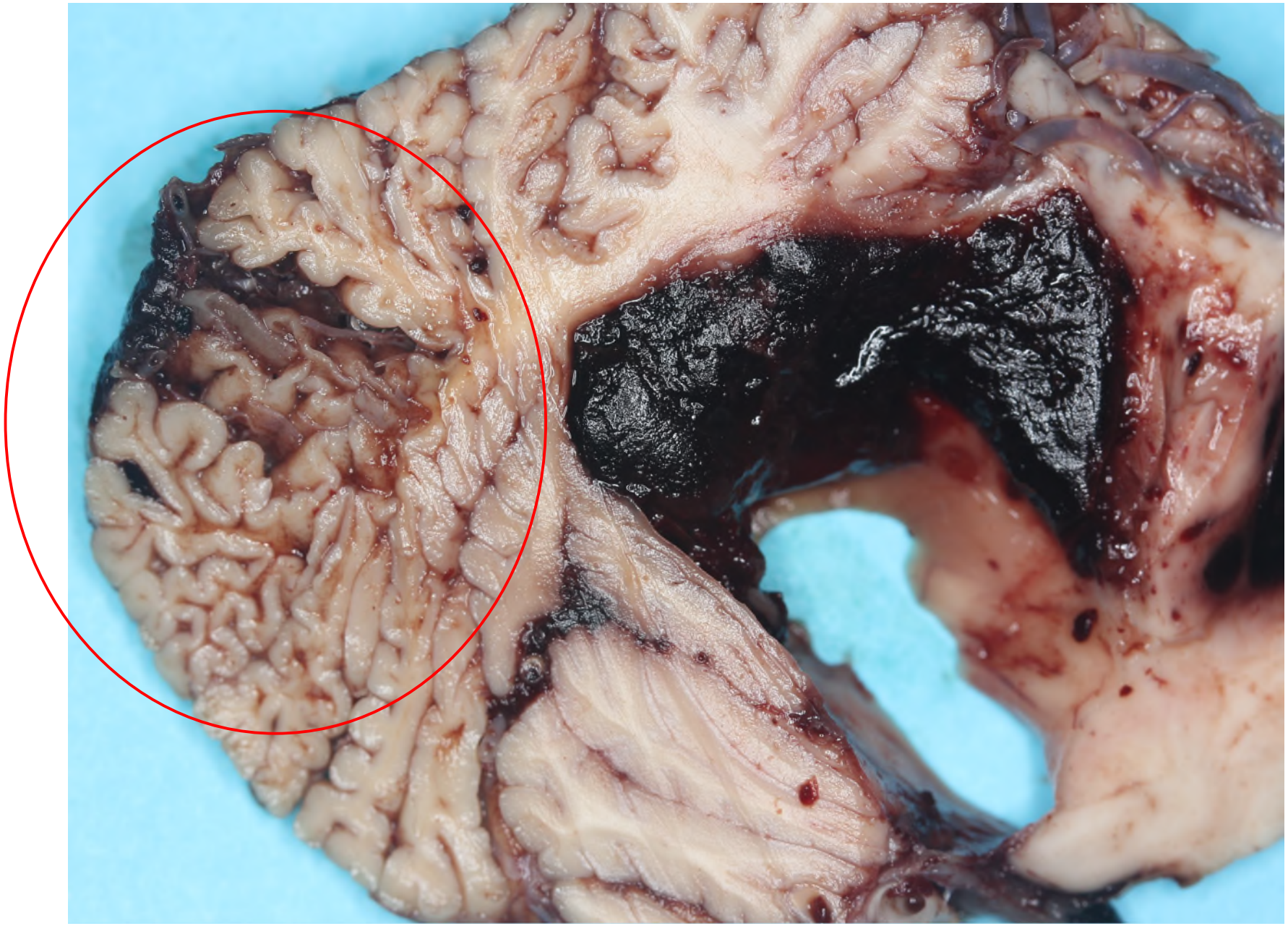
Case 24: Acute intracerebral (hypertensive) hemorrhage in R basal ganglia with intraventricular extension , *and*...



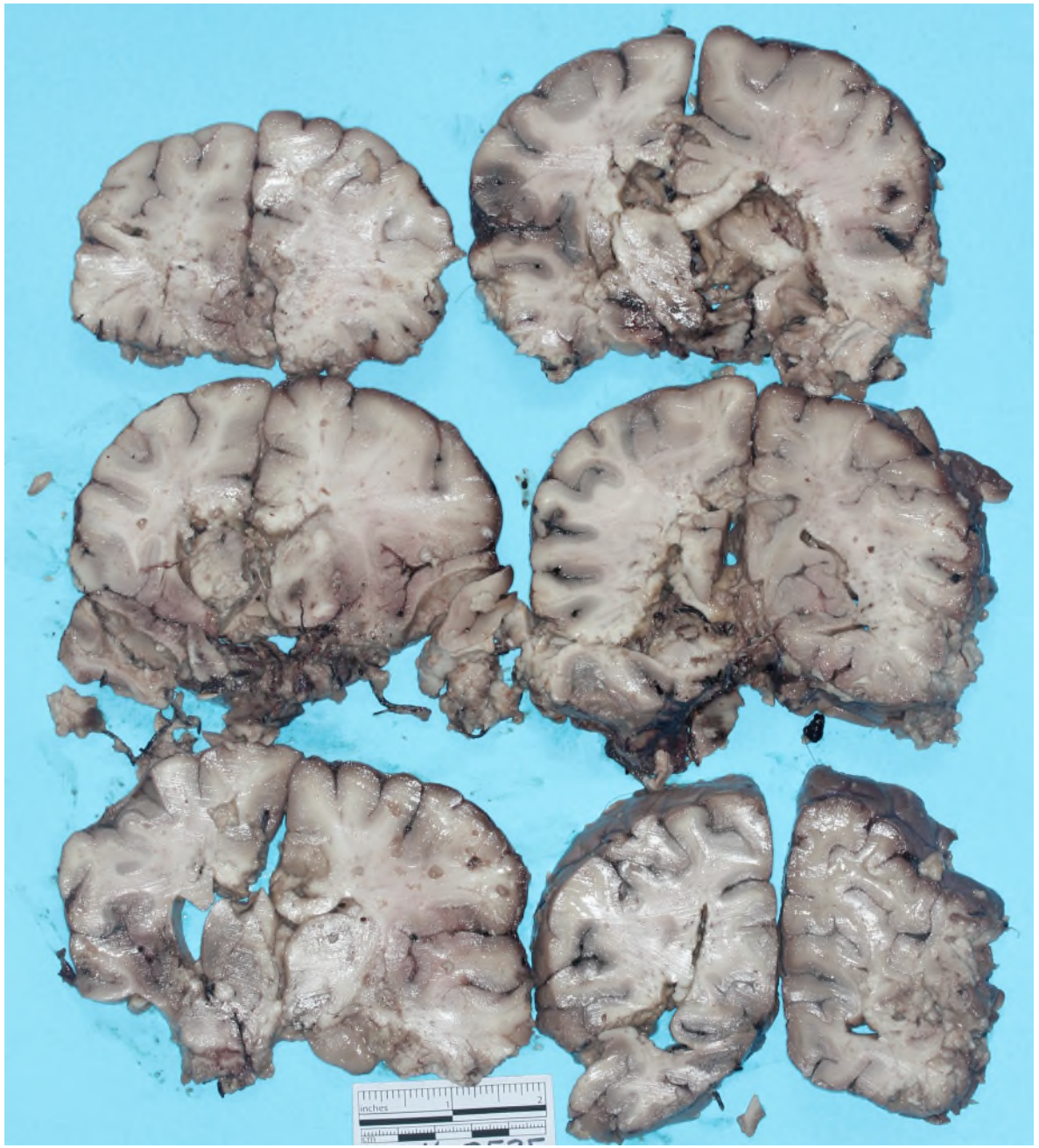


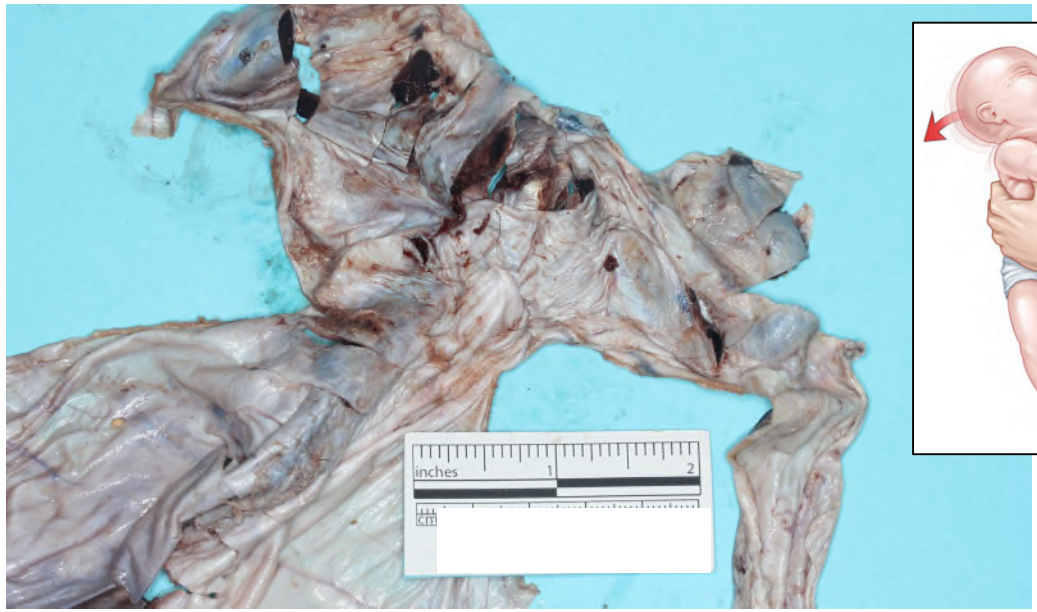
Hypertensive ICH

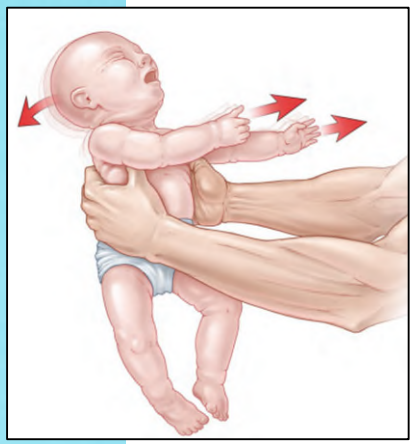
Case 24: Acute intracerebral (hypertensive) hemorrhage in deep cerebellar white matter, *and*...

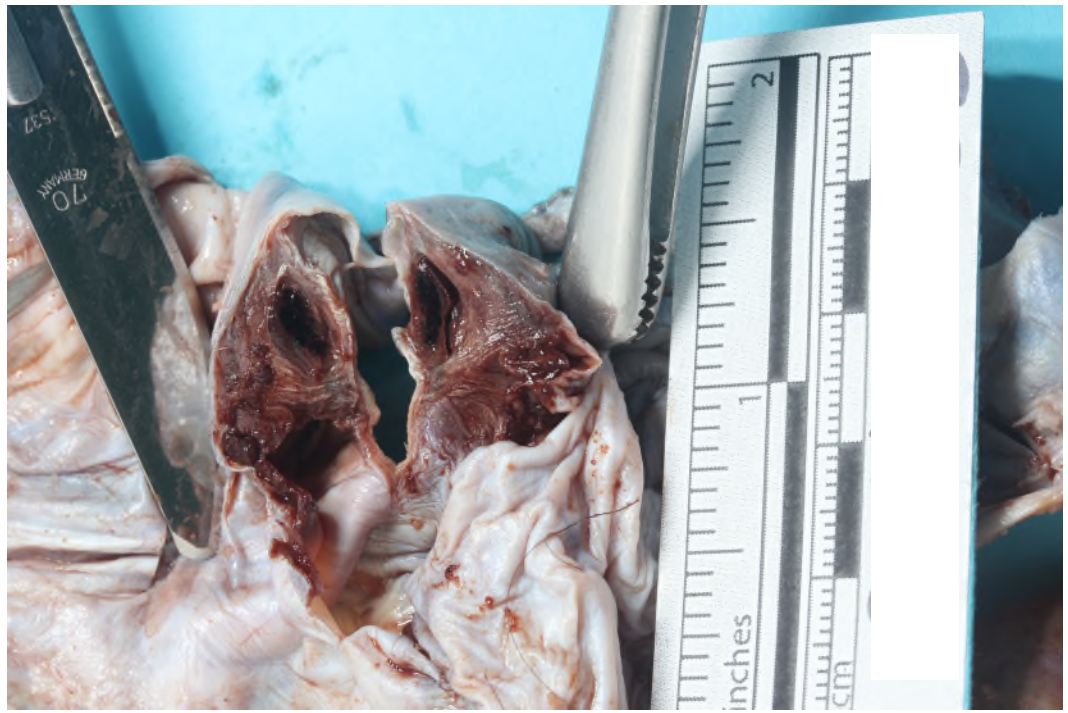


Case 15: 2mo, brought to ER, “suspicious circumstances”, with subacute SDH and ocular hemorrhages, 3 days’ survival









FNPDx:

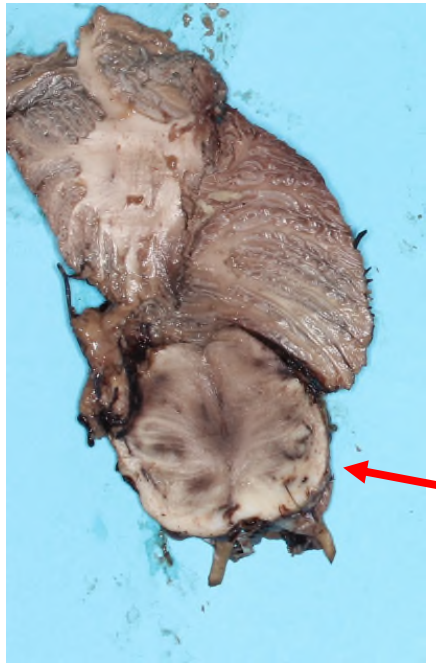
- Traumatic injury of head and neck, subacute, with:
  - Bilateral subdural hemorrhage
  - Brain swelling
    - Cranial sutural diastasis
    - Secondary hypoxia-ischemia of:
      - Brain and central cervical spinal cord, with focal reperfusion hemorrhage
        - Cerebellar cortical “emboli” to spinal canal
  - Bilateral optic nerve sheath hemorrhages
  - Bilateral, multilevel endoneurial cranial nerve root hemorrhages

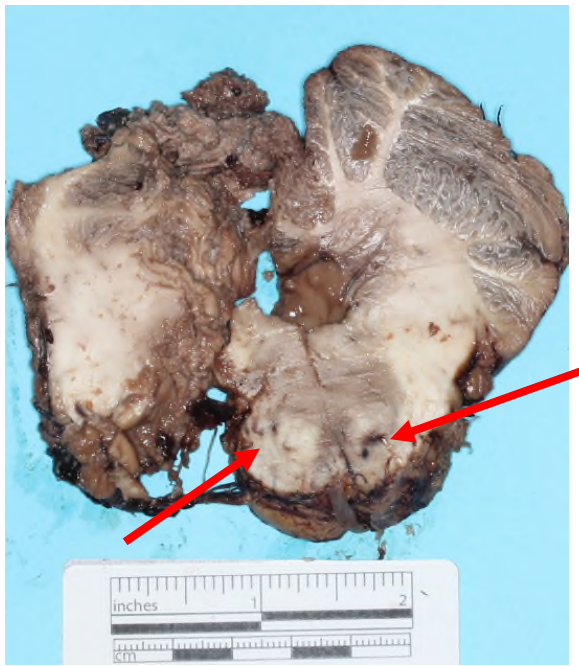
(Case 15)

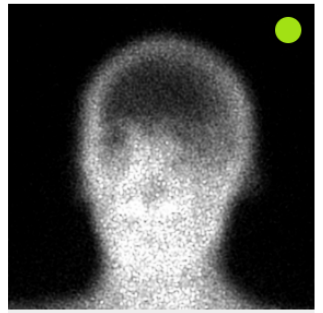
autolysis, “brain death” changes

***autolysis, "brain death" changes**

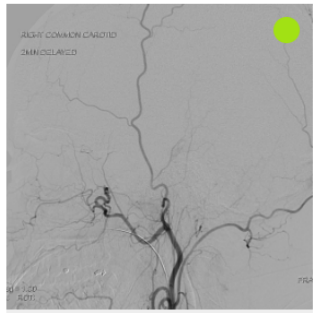
- Intracerebral (cranial) pressure exceeds cerebral perfusion (systolic) pressure
- Minimal entry/exit of arterial/venous blood into tissue
- "Clock stops" on reactive changes
- Focal capillary bed openings allow pinpoint reperfusion hemorrhage
  - Basal, subpial, with minimal reactive changes on microscopy
  - "Last gasp" hemorrhages



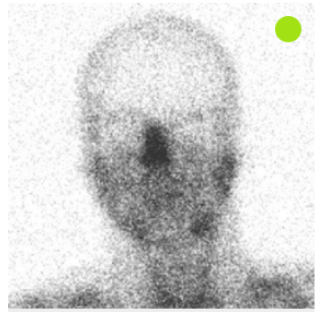




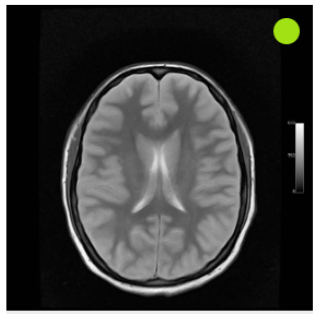
Case 1



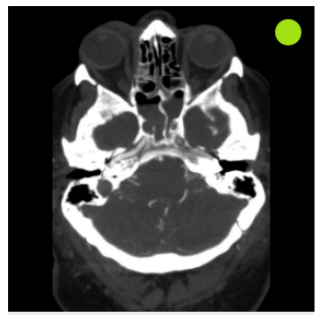
Case 2



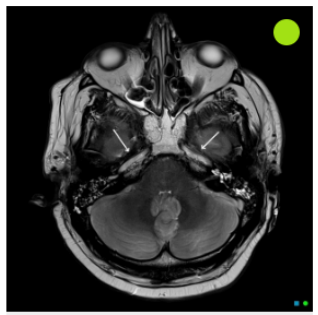
Case 3: with hot nose sign



Case 4



Case 5



Case 6

https://radiopaedia.org/articles/brain-death-2

Acknowledgments

- NYC OCME
  - Jason Graham, Chief
  - Heather Maioli, Neuropathologist
- AANP!!!!!