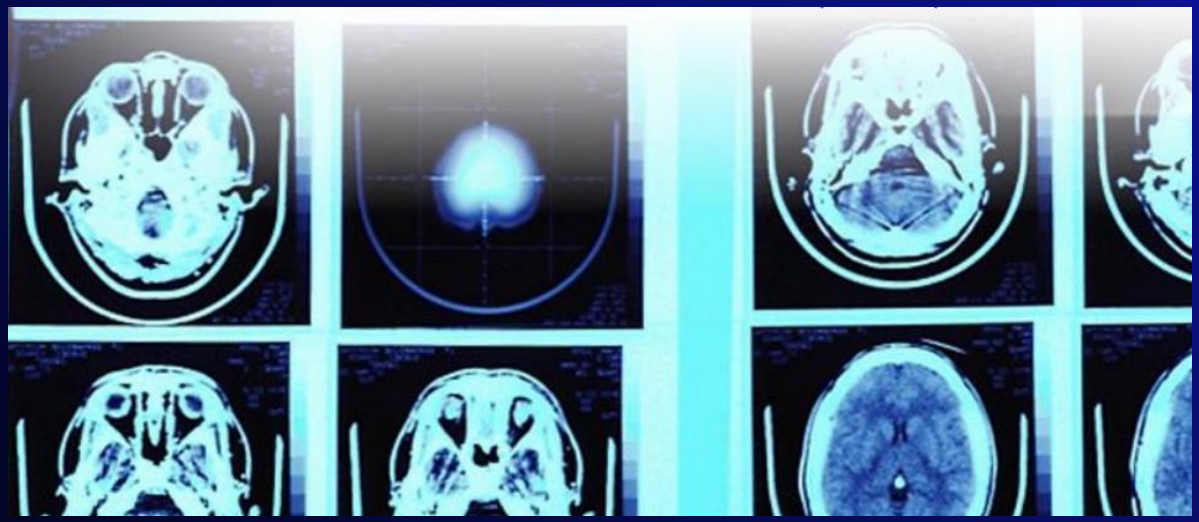
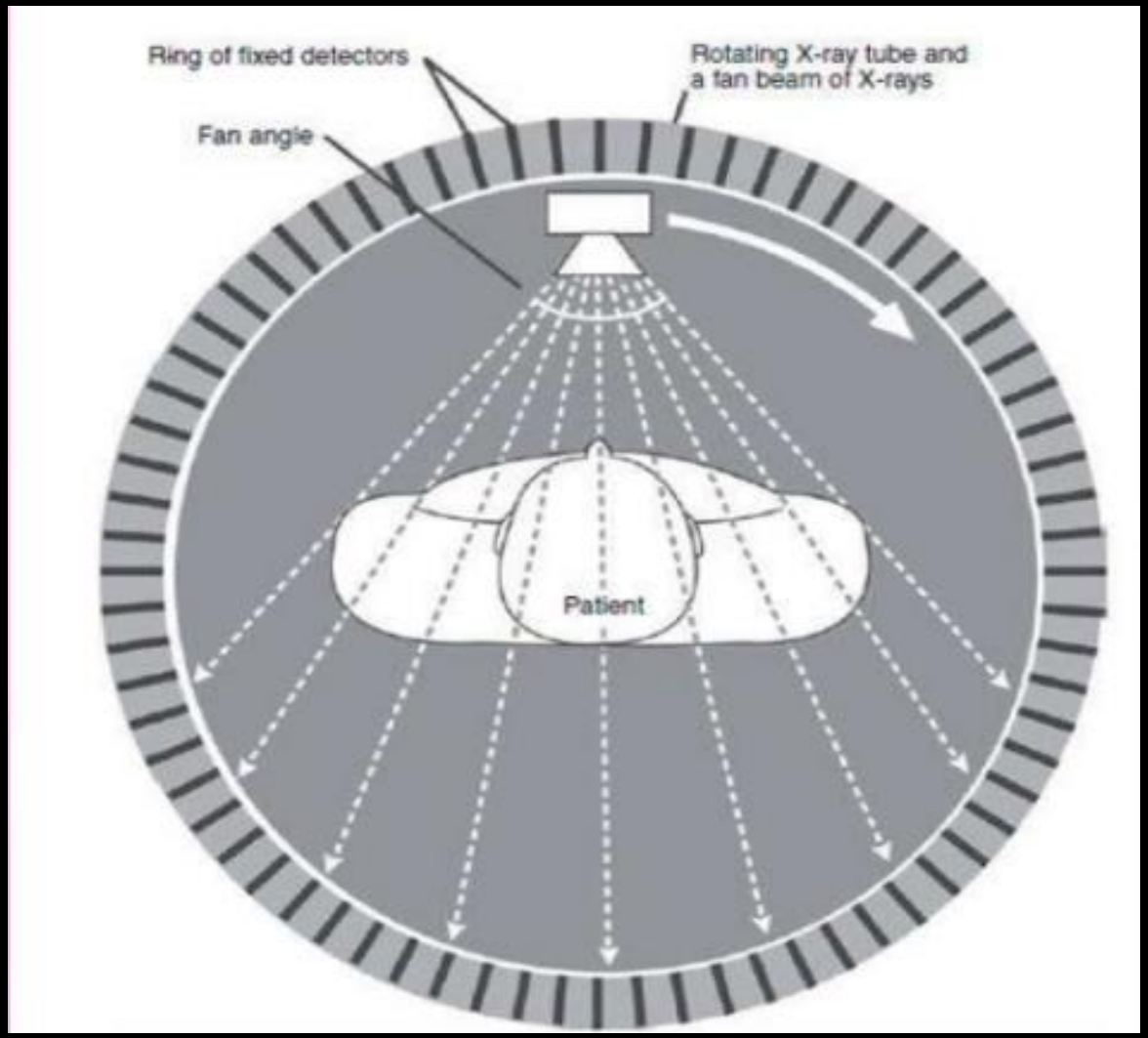
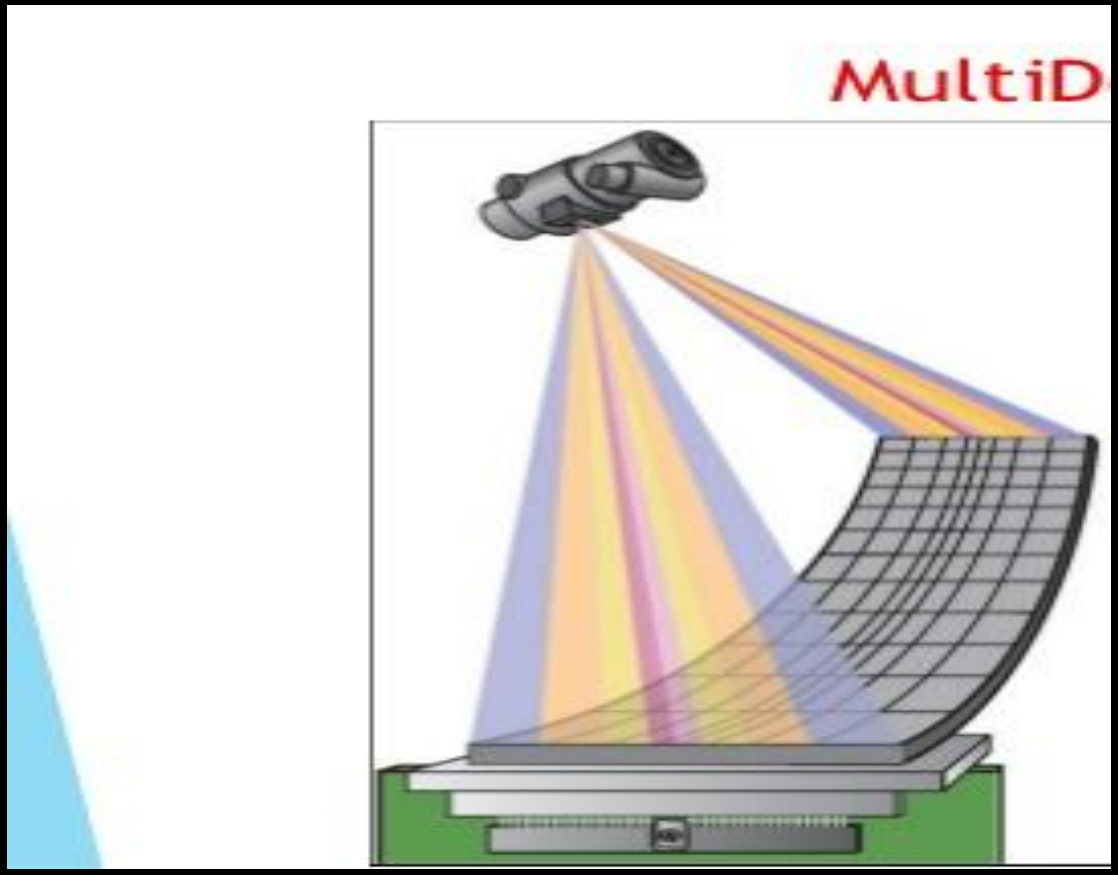
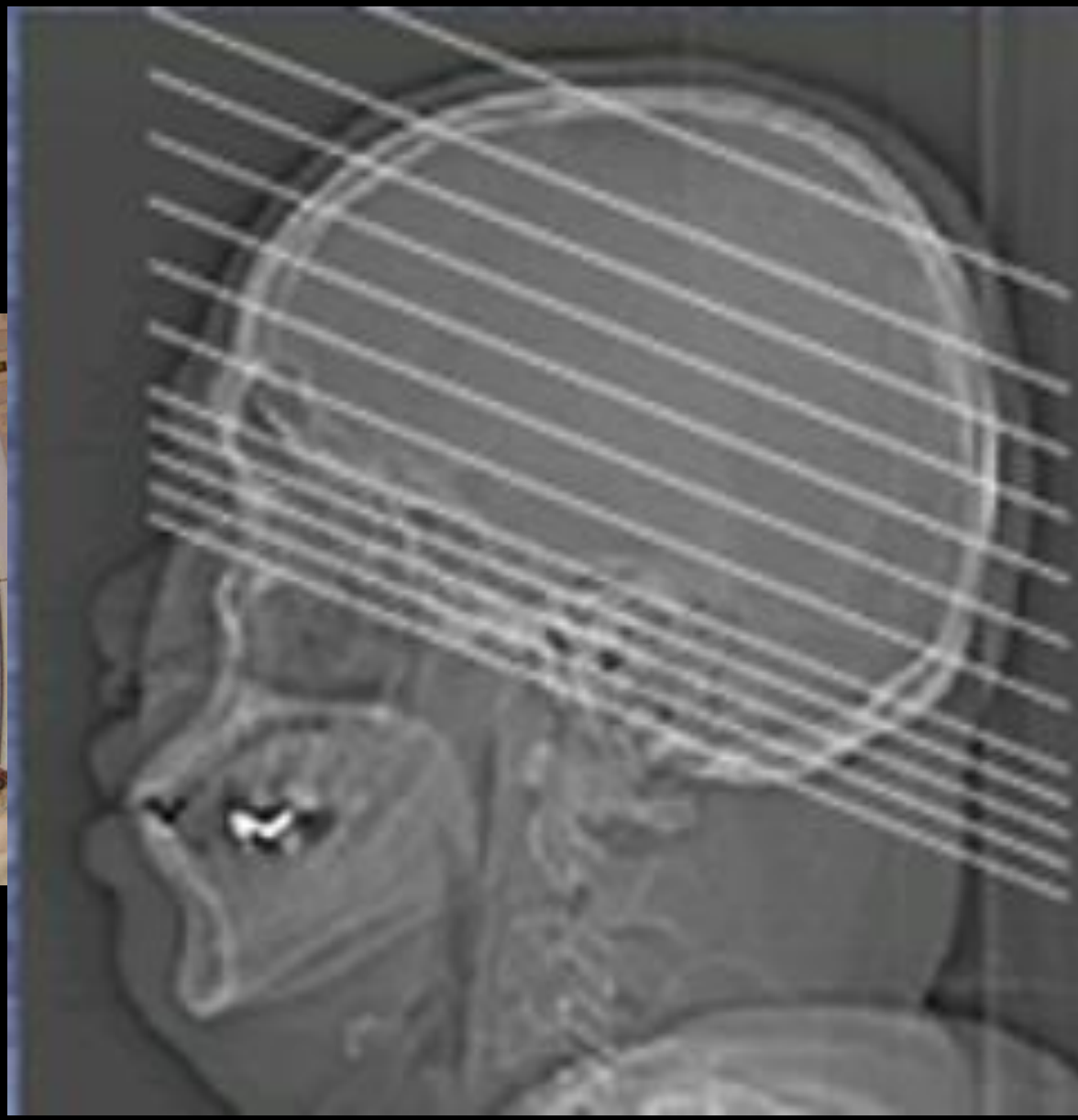
Introduction to Head CT Imaging




Ring of fixed detectors
Rotating X-ray tube and
a fan beam of X-rays
Fan angle
Patient

Multidetector CT scan



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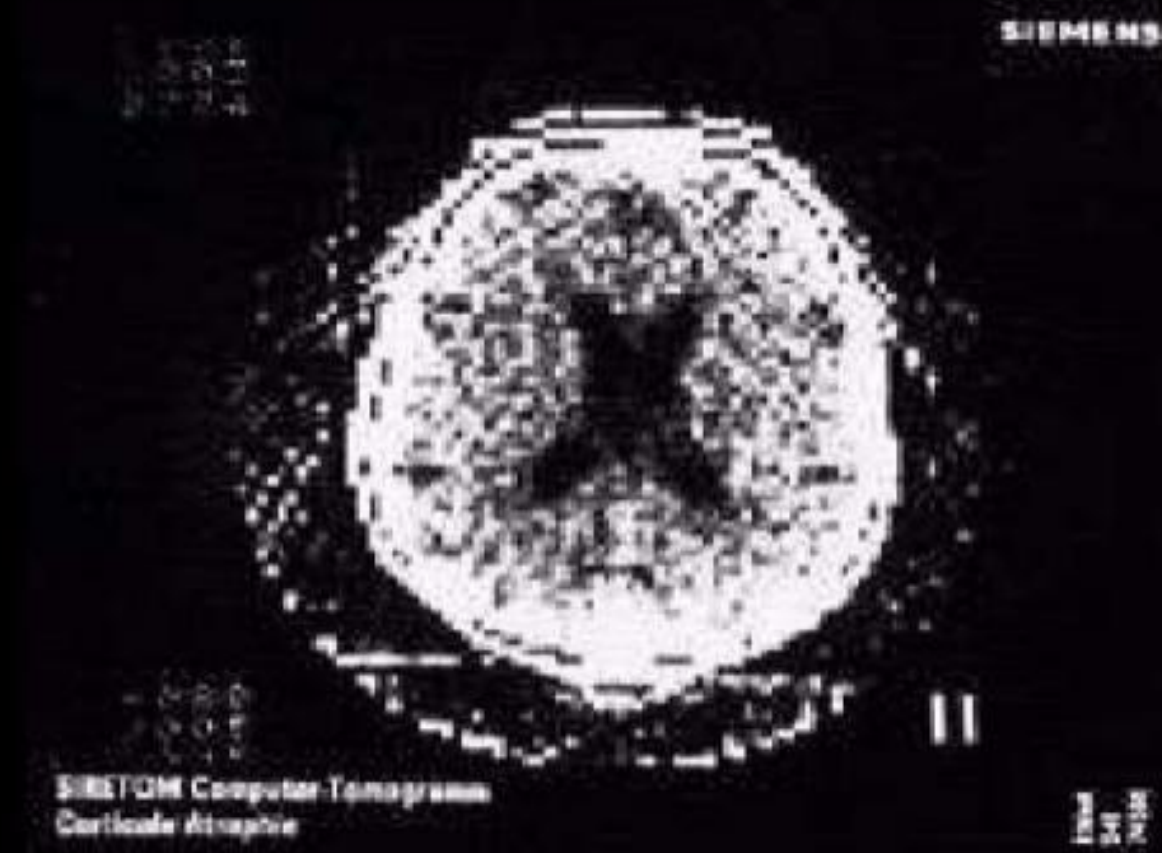
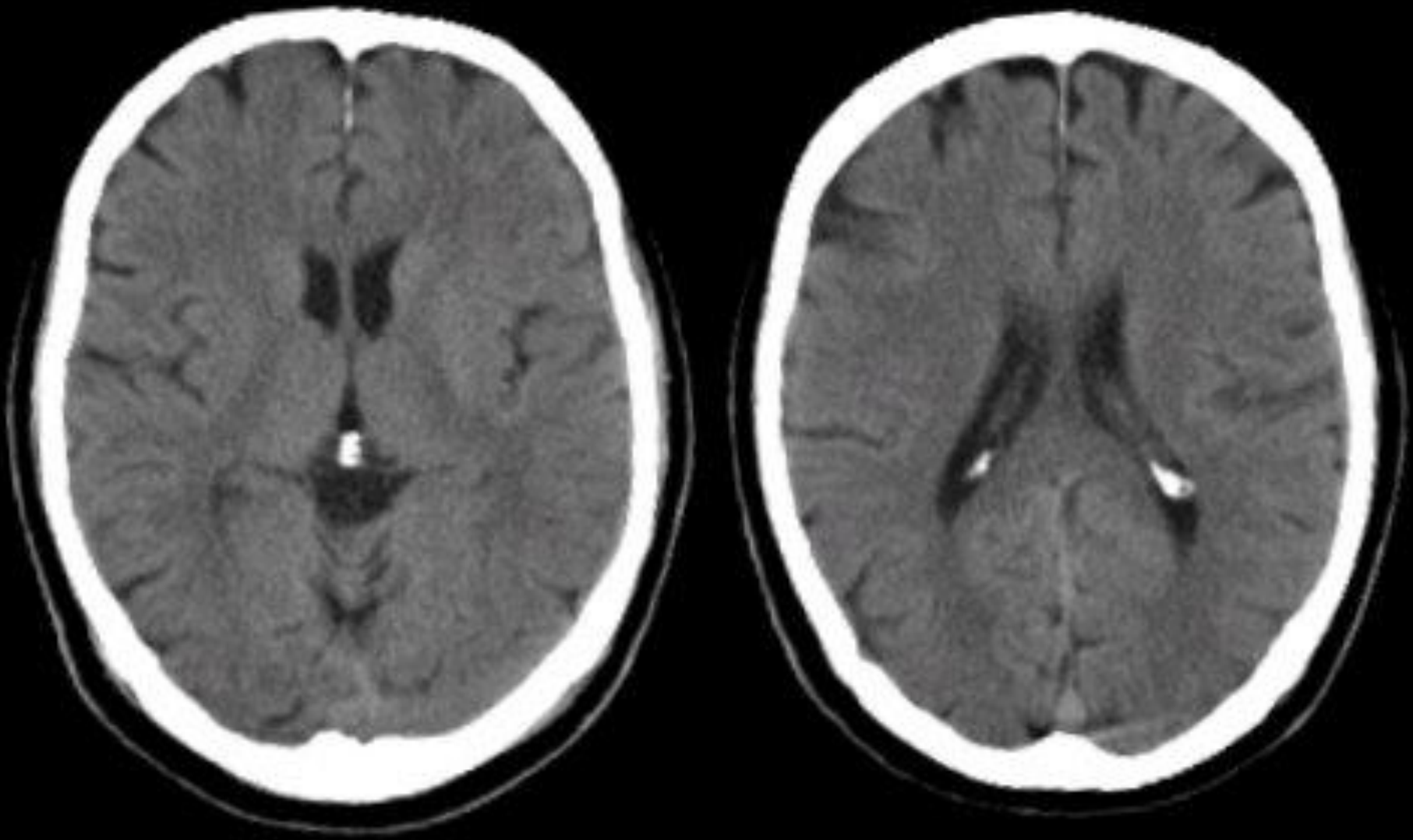
W 392
C 69



Original axial CT image from the dedicated Siretom CT scanner circa 1975. This image is a coarse 128 x 128 matrix

High resolution brain CT



CT vs. MRI

	CT	MRI
Obtained	X-ray beam	Magnetic fld
Bone	Bright	Dark
Cost	$330	$900
Plane	Axial	3-D
Technique	Adjust window	T1, T2, Pd
Length	10-20 minutes	30-60 min
Opening	Wide doughnut	Long, narrow

Advantages to CT

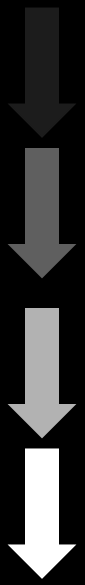
- Costs less than MRI
- Better access
- Shows up acute bleed
- A good quick screen
- Good visualization of bony structures and calcified lesions

Disadvantages to CT

- Resolution
- Beam-hardening artifact
- Limited views of the posterior fossa and poor visualization of white-matter disease

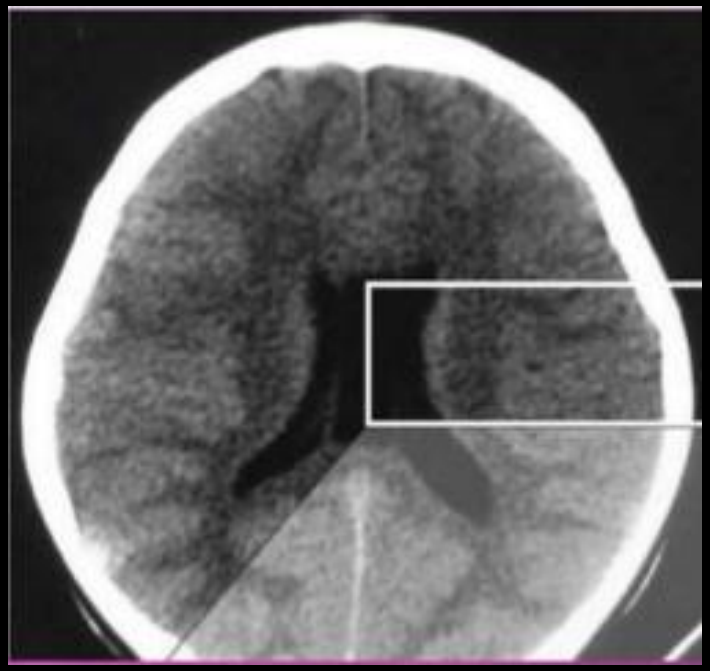
CT density

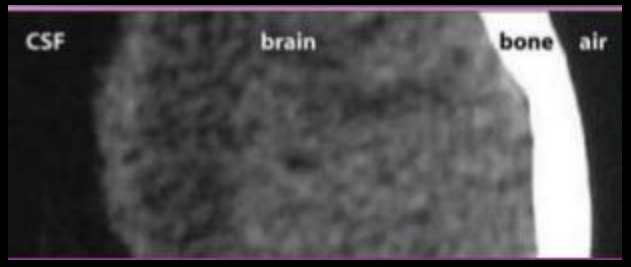
Black



White

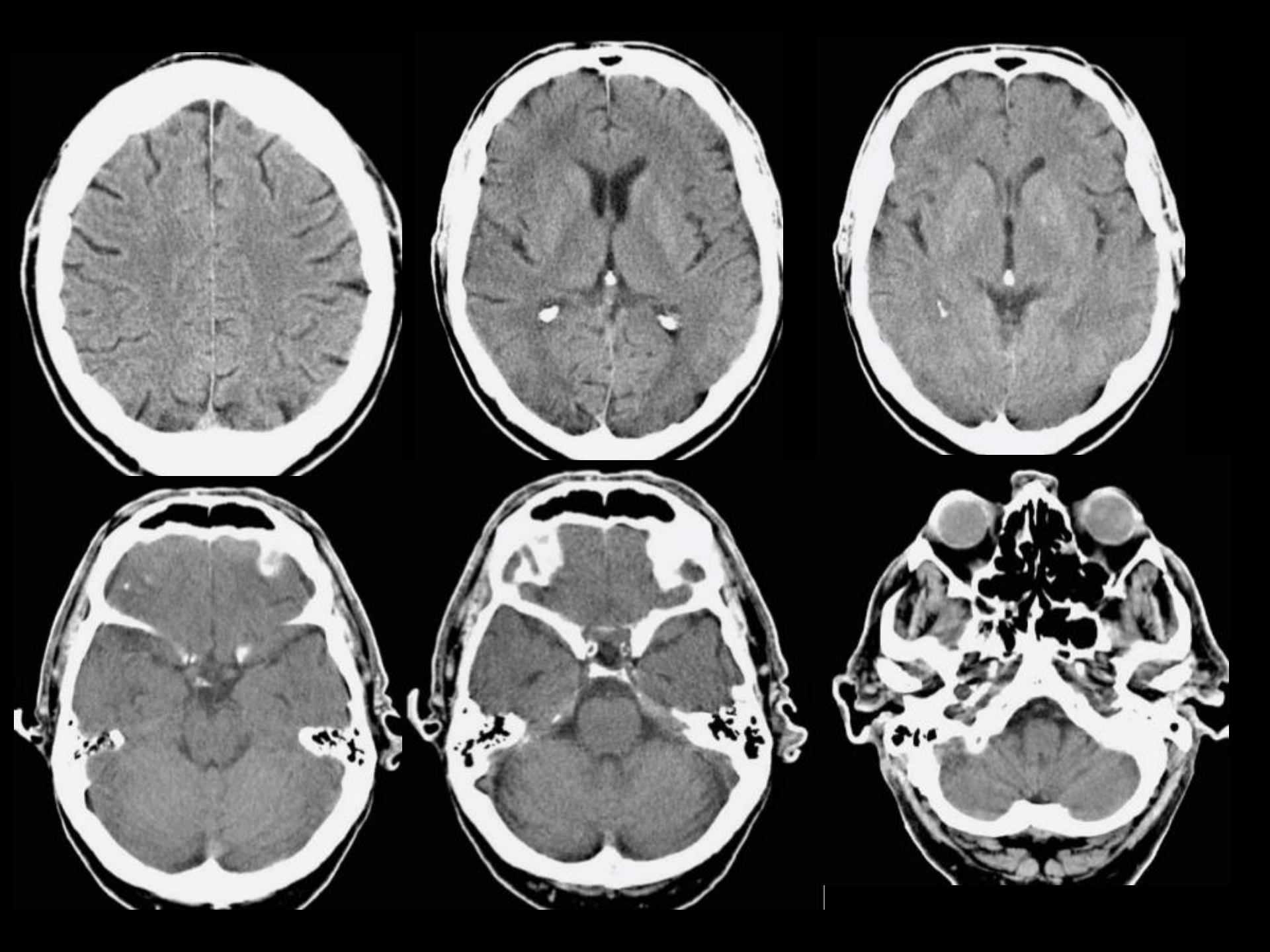
Structure/ Tissue	Hounsfield units
Air	-1000 to -600
Fat	-100 to -60
Water	0
CSF	+8 to 18
White matter	+30 to 41
Gray matter	+37 to 41
Acute blood	+50 to 100
Calcification	+140 to 200
Bone	+600 to 2000
metal	**+1000-+10000**



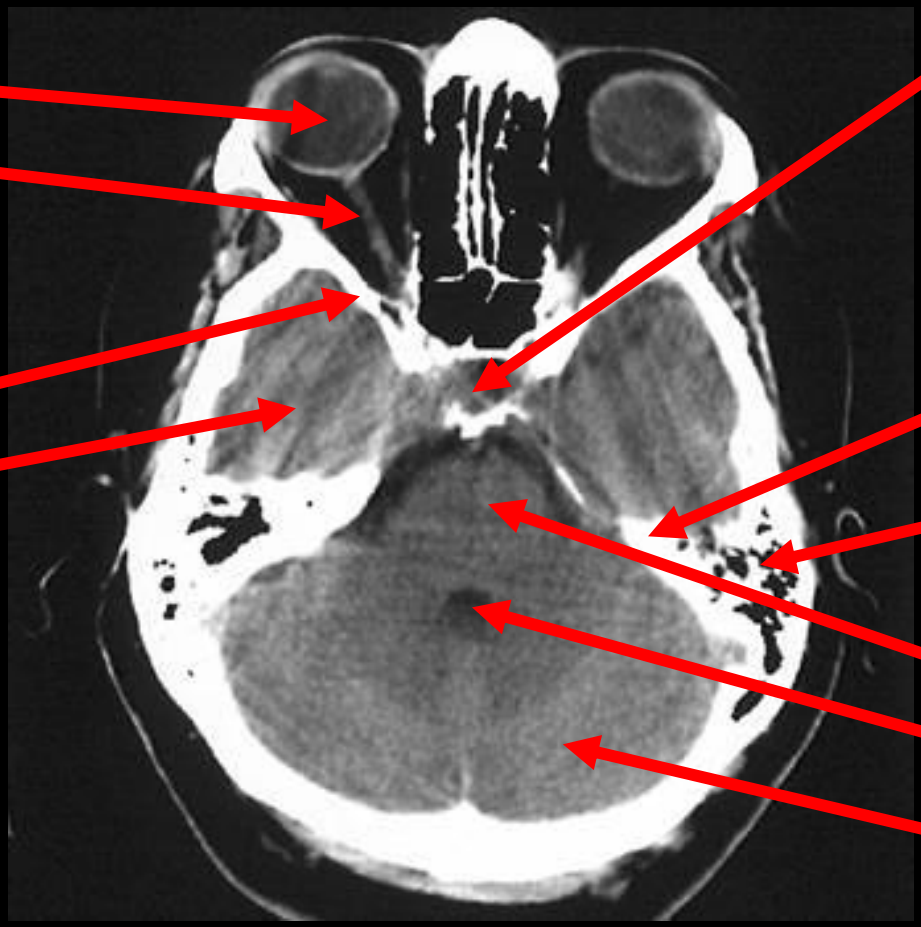

CSF
brain
bone
air

Normal brain CT scan report :

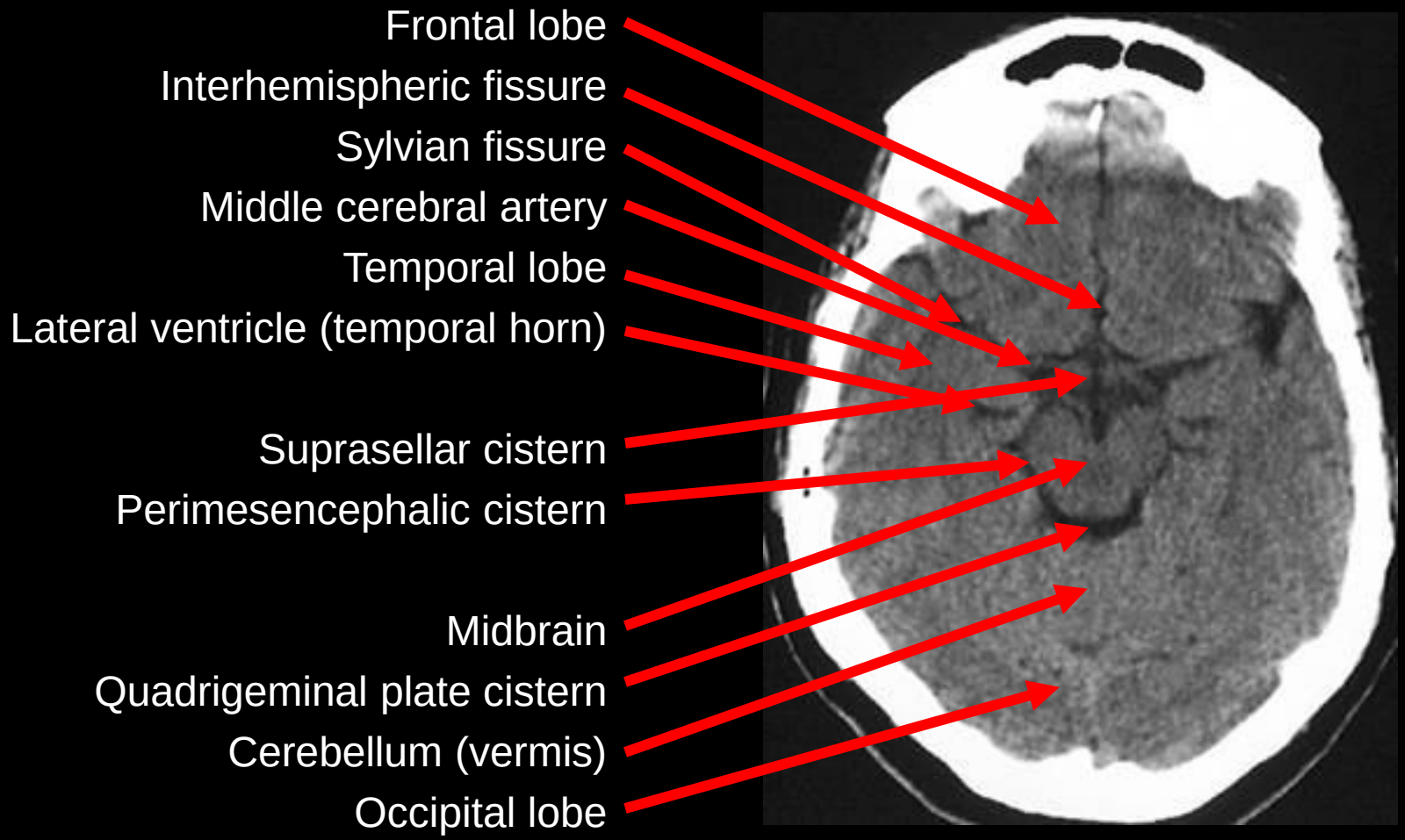
No brain focal lesion
No midline shift
No hydrocephalus



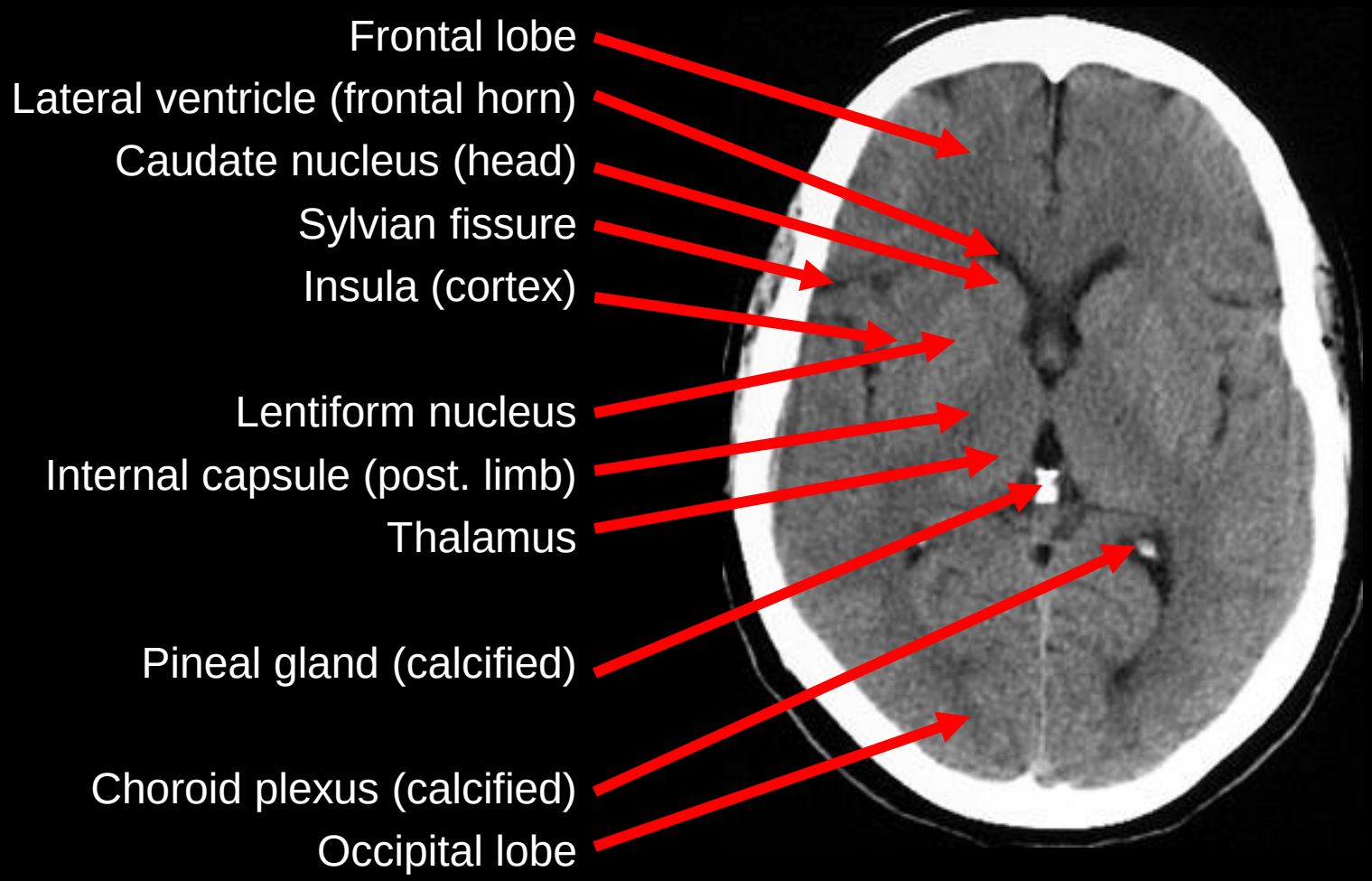
Normal Brain anatomy



Normal Brain Anatomy



Normal Brain Anatomy



Brain Radiology Report

Look for:

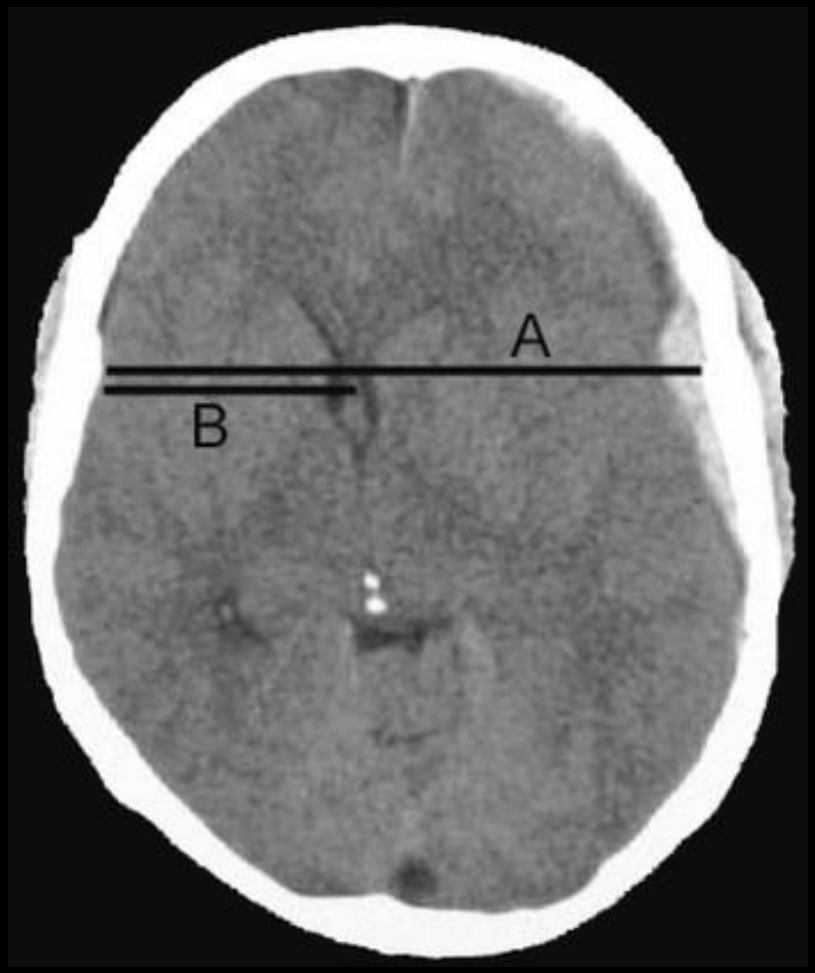
Brain focal lesions

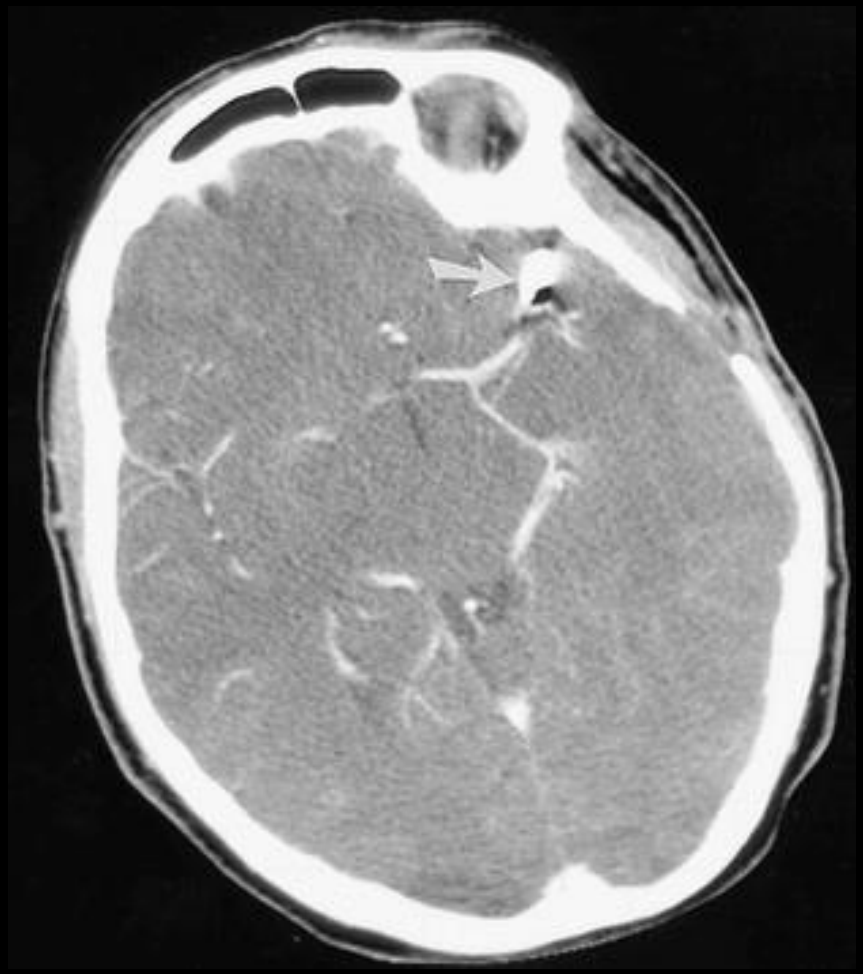
Midline shift

Dilated ventricular system (Hydrocephalus)

Midline shift

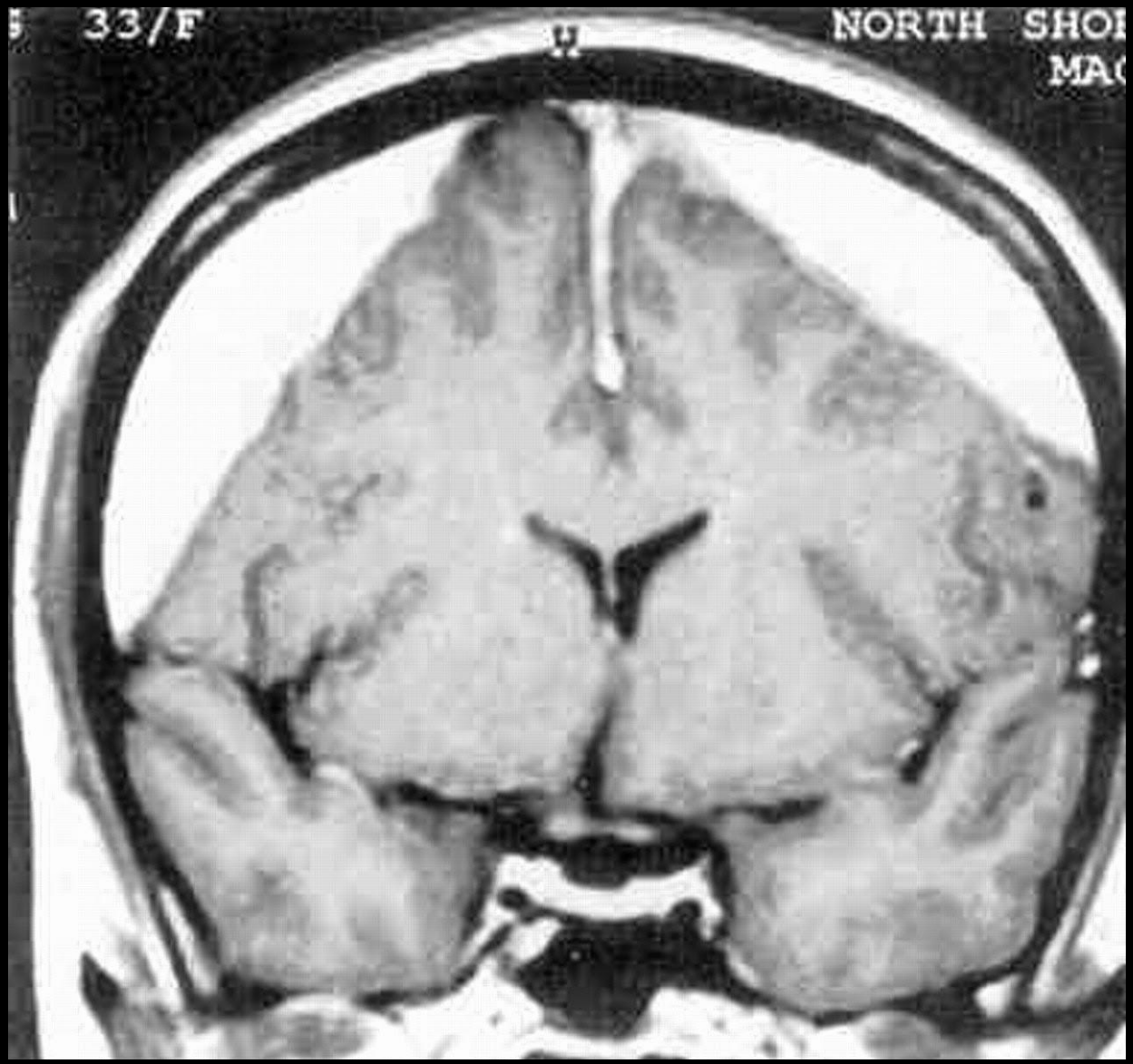
Midline shift v/s positional tilt



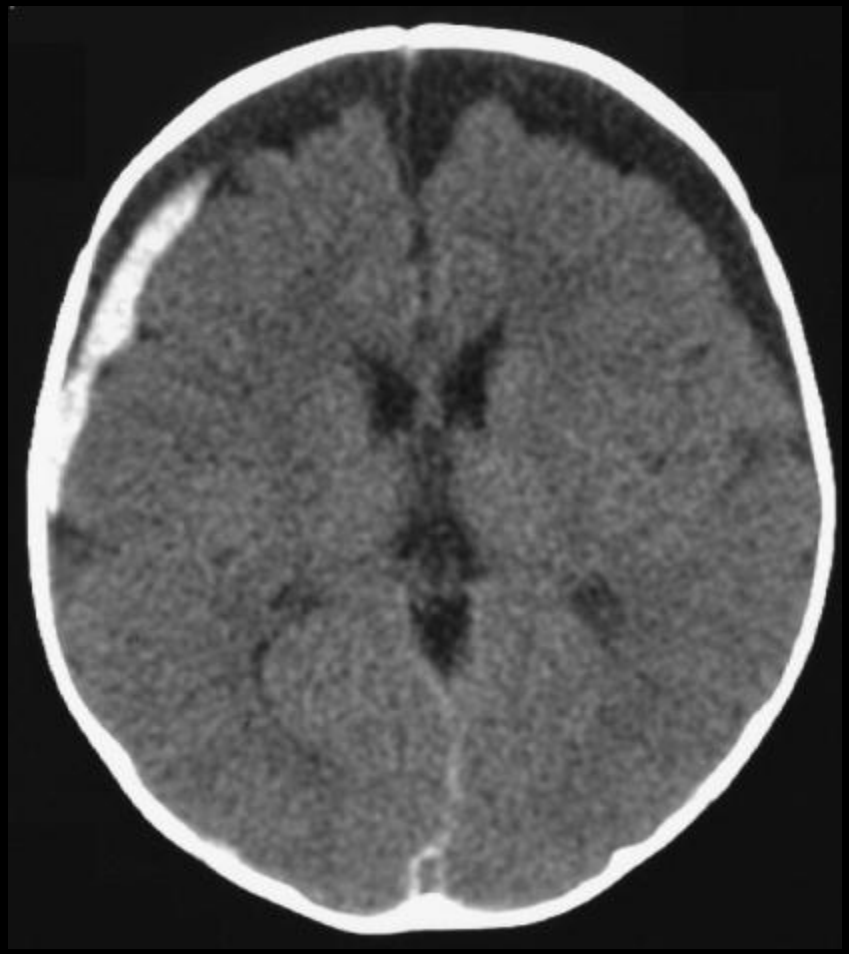


Not always there is midline shift (MLS) , there could be pathologies that don’t cause MLS

1) Bilateral



2) Small



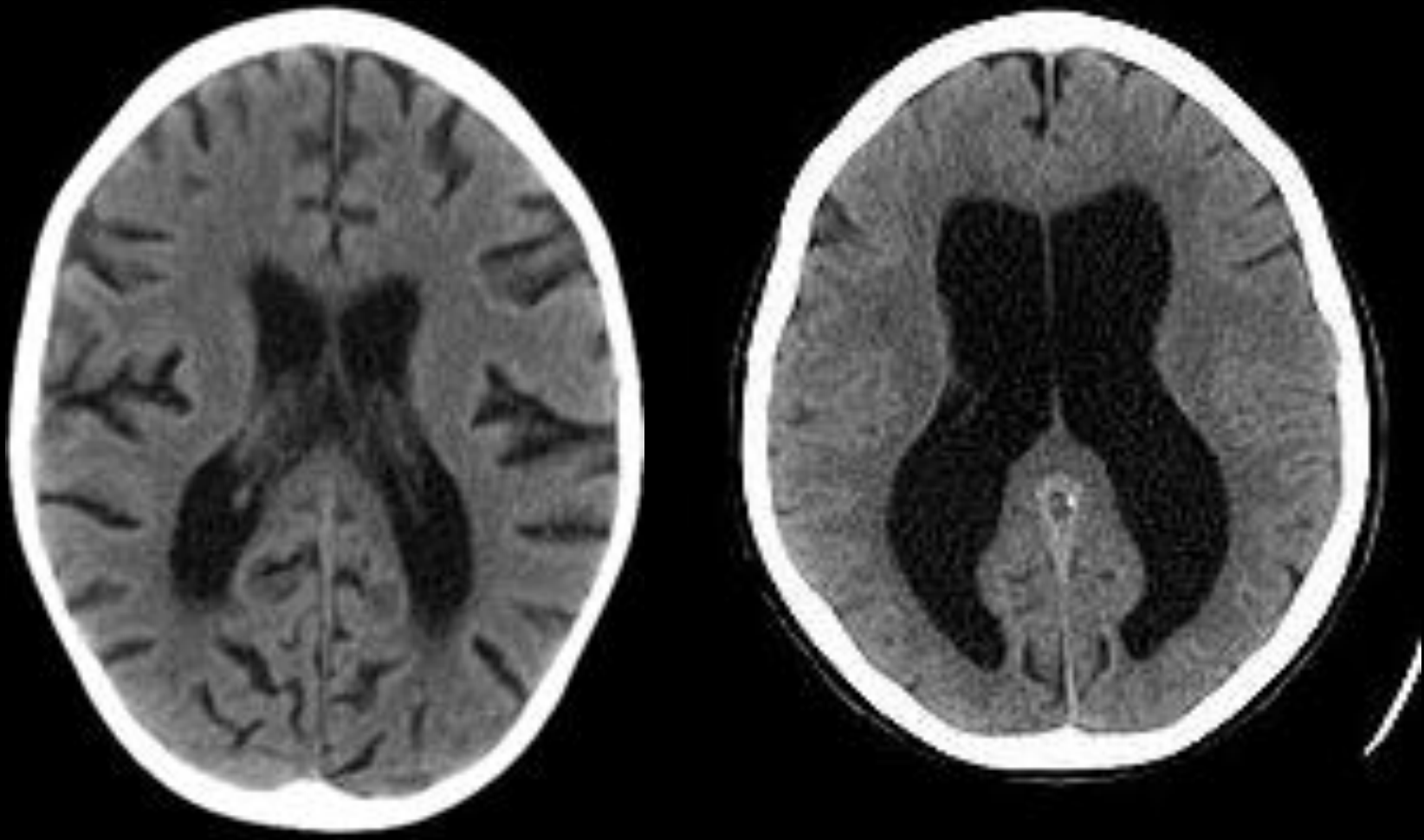
3) Lesion that are high up near the vertex

Ventricular Dilatation

Ex vacuo dilatation: due to diffuse brain atrophy

Hydrocephalus : communicating and non communicating

Brain atrophy v/s hydrocephalus



Brain focal leison

Abnormalities divided into :

Intraventricular

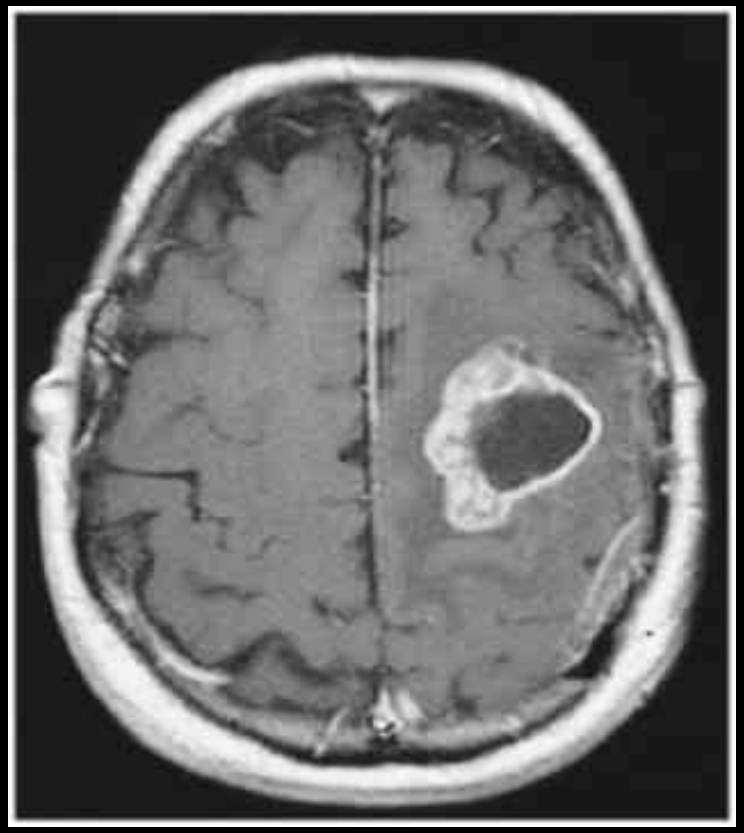
Intra-axial

Extra-axial

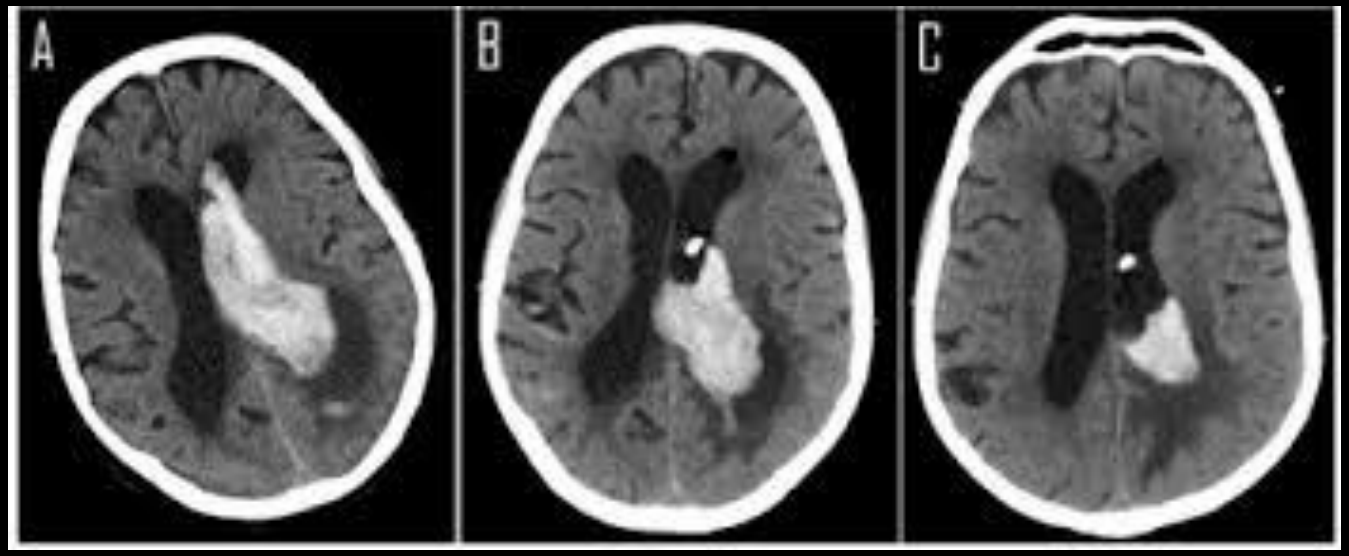
Intra-osseous

Scalp

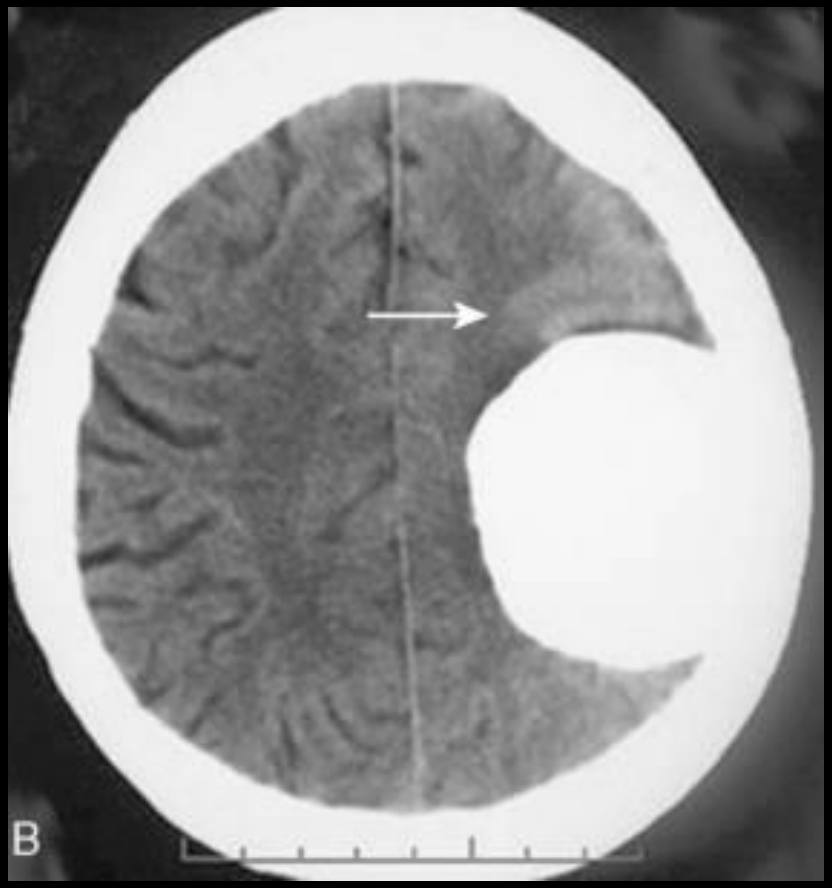
Intra-axial



Intraventricular

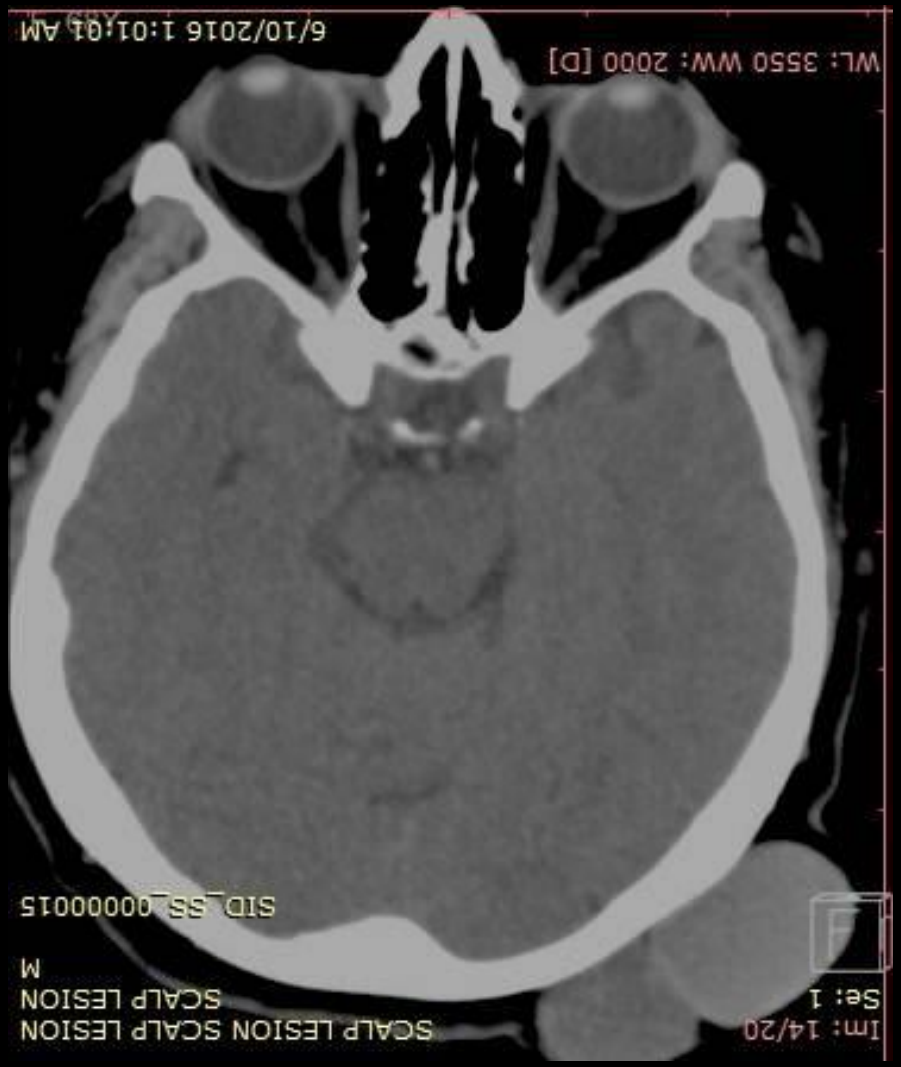


Extra-axial



Intra-osseous

Scalp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Brain Pathology - CT

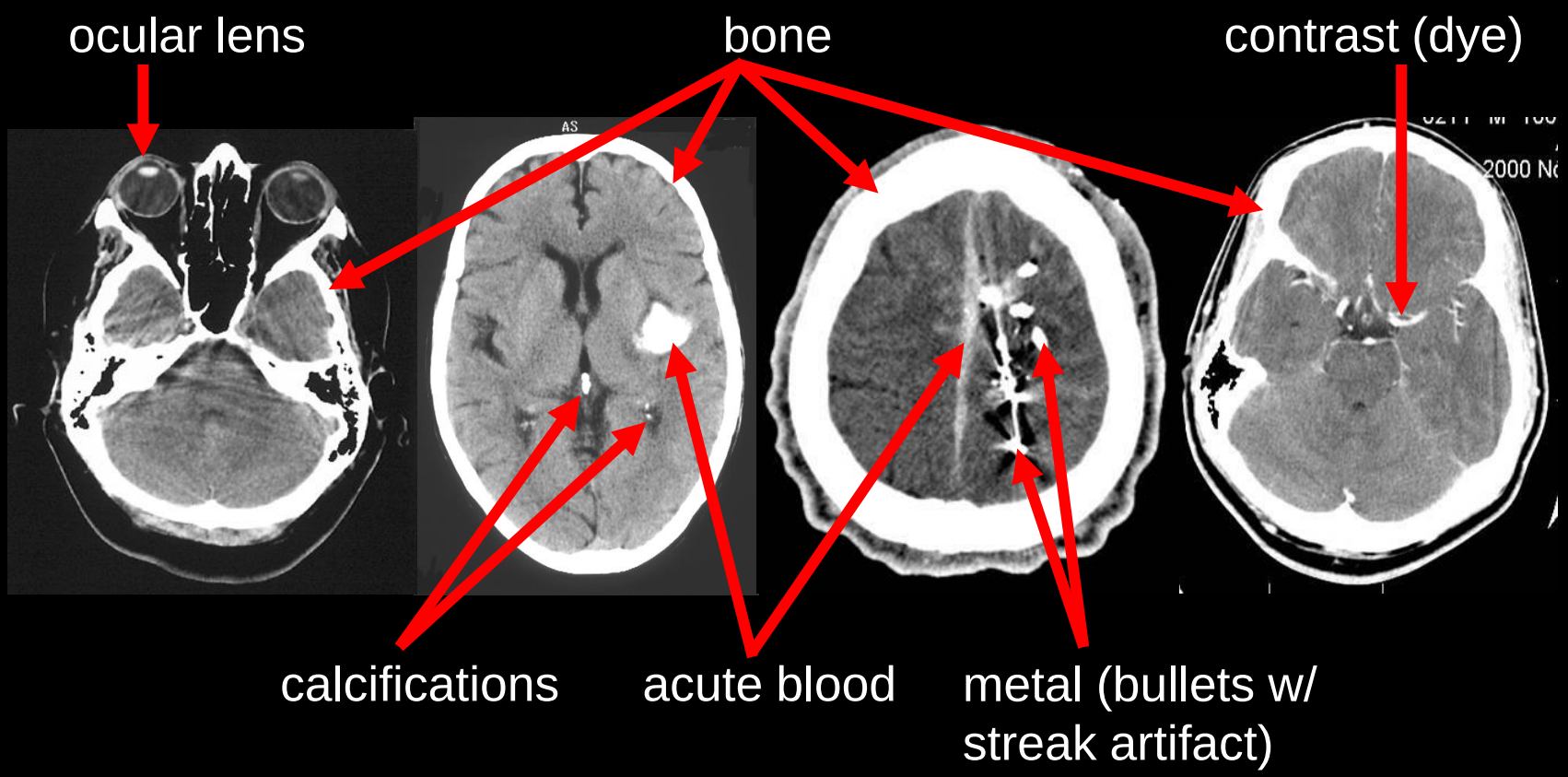
Brain pathology could be divided to two type according to their density on CT:

1- Hyperdense lesions

2- Isodense lesions

3- Hypodense lesions

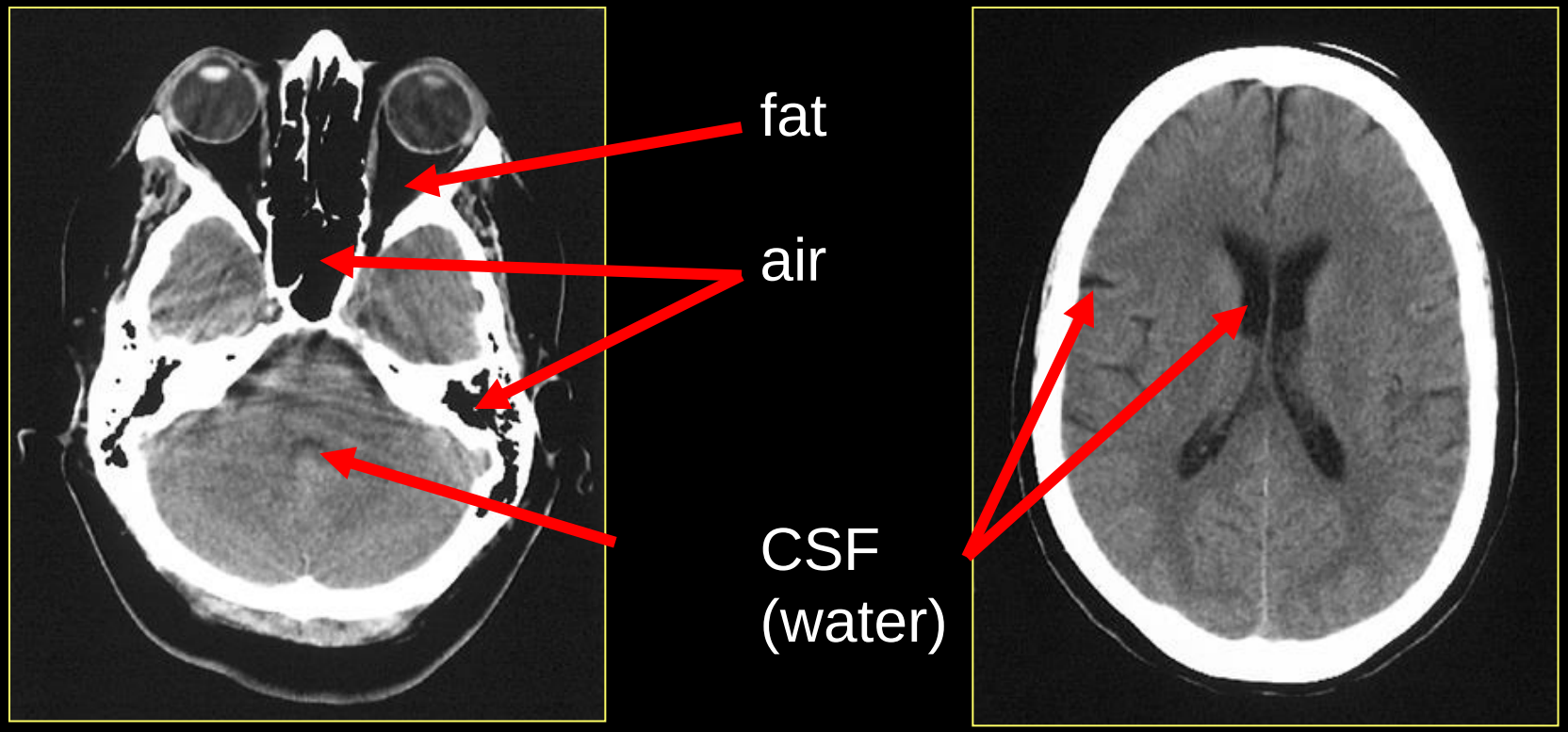
Hyperdense things on CT



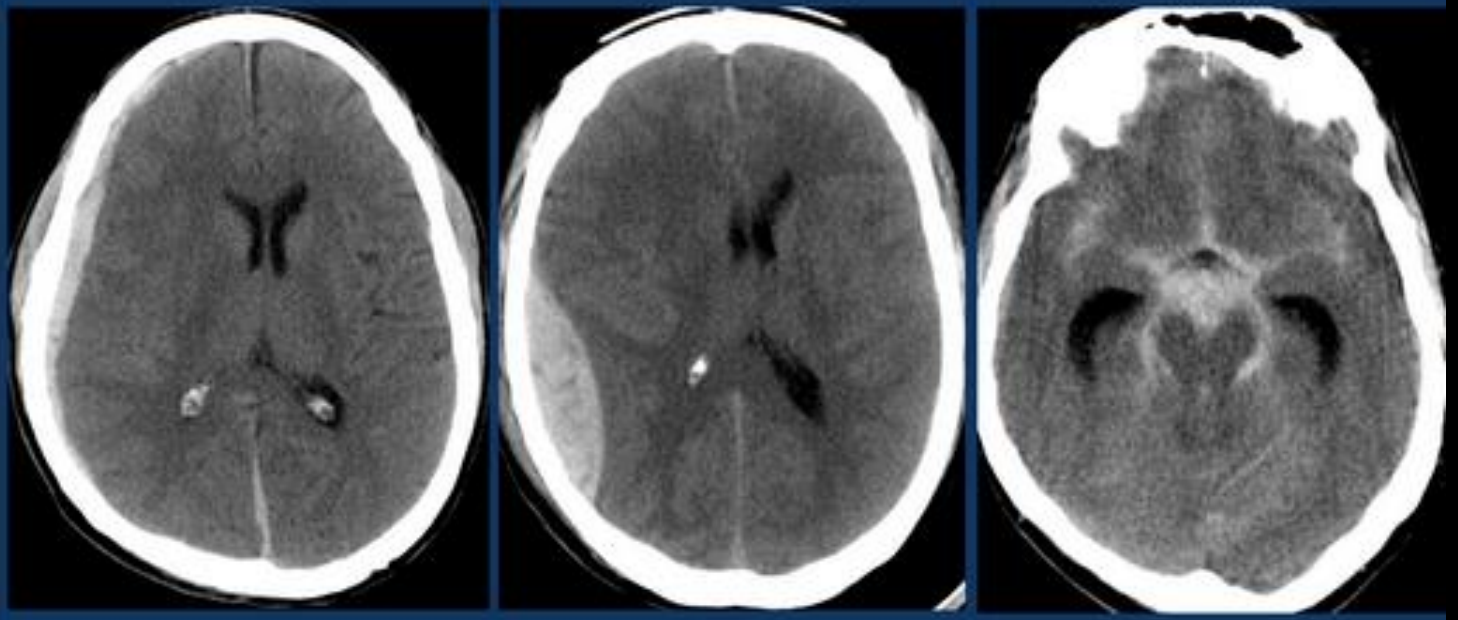
Isodense things on CT

- *Note that white matter is less dense than gray matter and therefore: white matter is darker than gray matter*

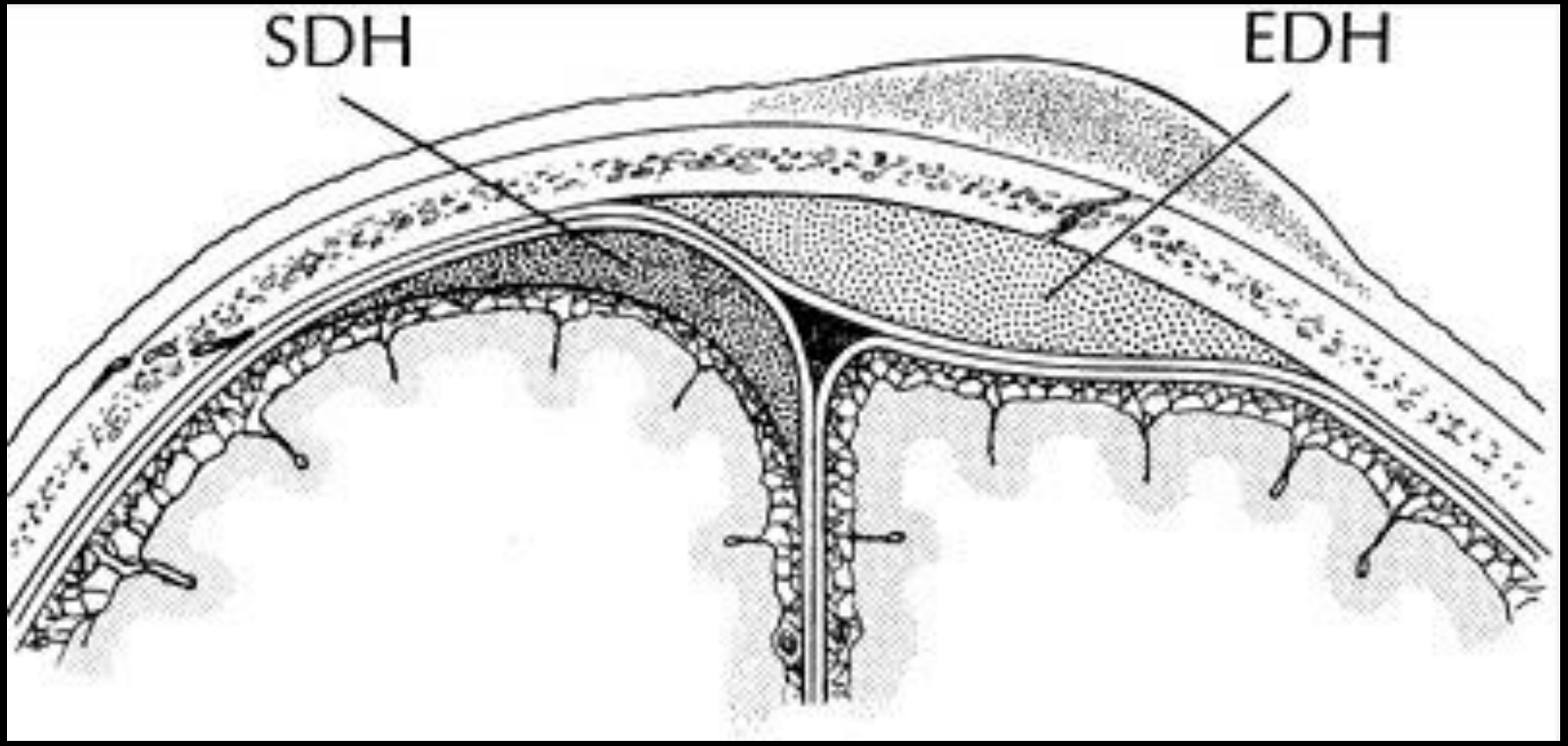
Hypodense things on CT

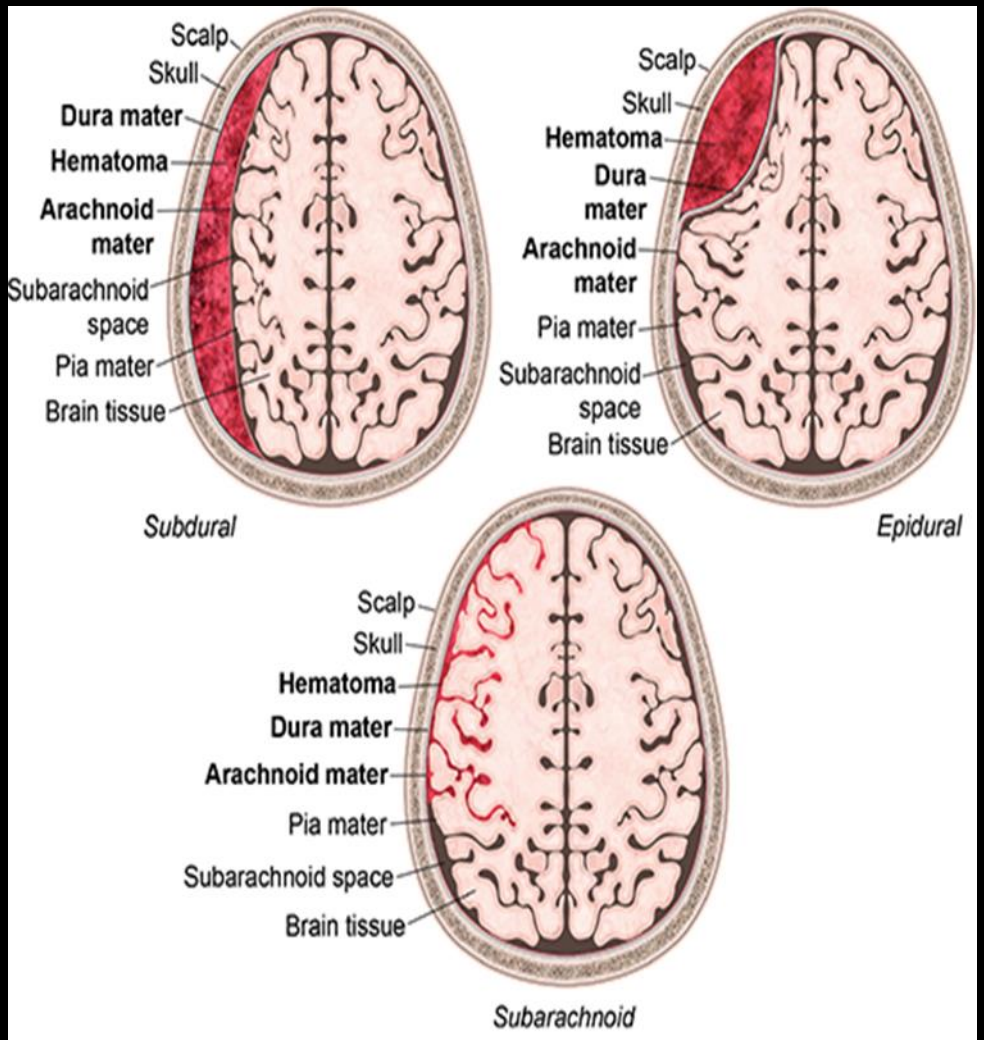


Extra-axial Hemorrhage



Subdural | Epidural | Subarachnoid


SDH
EDH



Epidural hematoma (EDH)

- **Mechanism:** low-velocity blunt trauma to the head
- **Types:**
- • Arterial EDH, 90% (middle meningeal artery)
- • Venous EDH, 10% (sinus laceration, meningeal vein)
  - Posterior fossa: transverse or sigmoid sinus laceration (common)
  - Parasagittal: tear of superior sagittal sinus
- Large EDHs are neurosurgical emergencies.
- Small (<5 mm thick) EDHs adjacent to fractures are common and do not represent a clinical emergency.
- 95% of all EDHs are associated with fractures.

Epidural hematoma

Imaging Features

Arterial EDH:

• 95% are unilateral, **temporoparietal**

• Biconvex, lenticular shape

• Does not cross suture lines

• May cross dural reflections (falx tentorium), in contradistinction to subdural hematoma (SDH)

• Commonly associated with skull fractures

.

Venous EDH

• More variable in shape (low-pressure bleed)

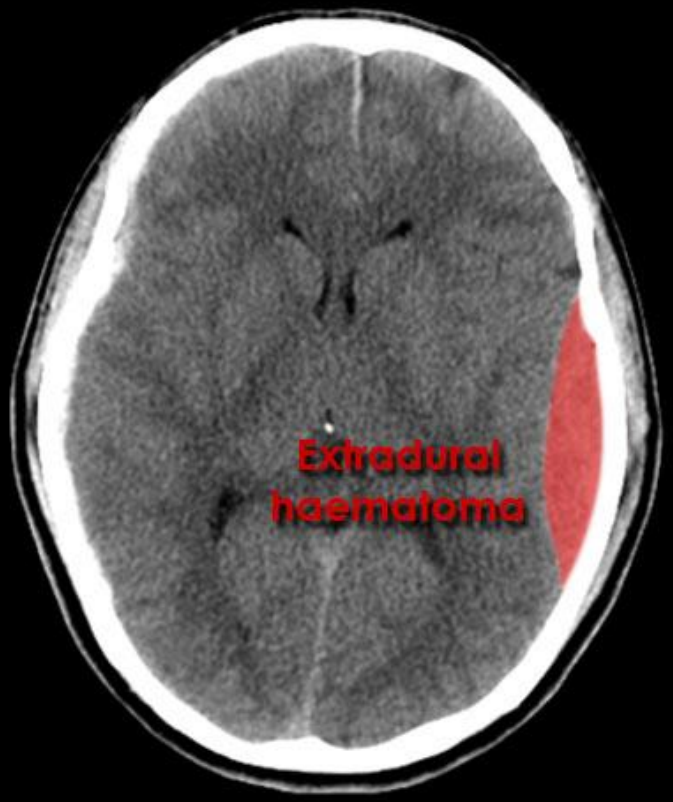
• Often requires delayed imaging because of delayed onset of bleed after trauma

Epidural hemorrhage

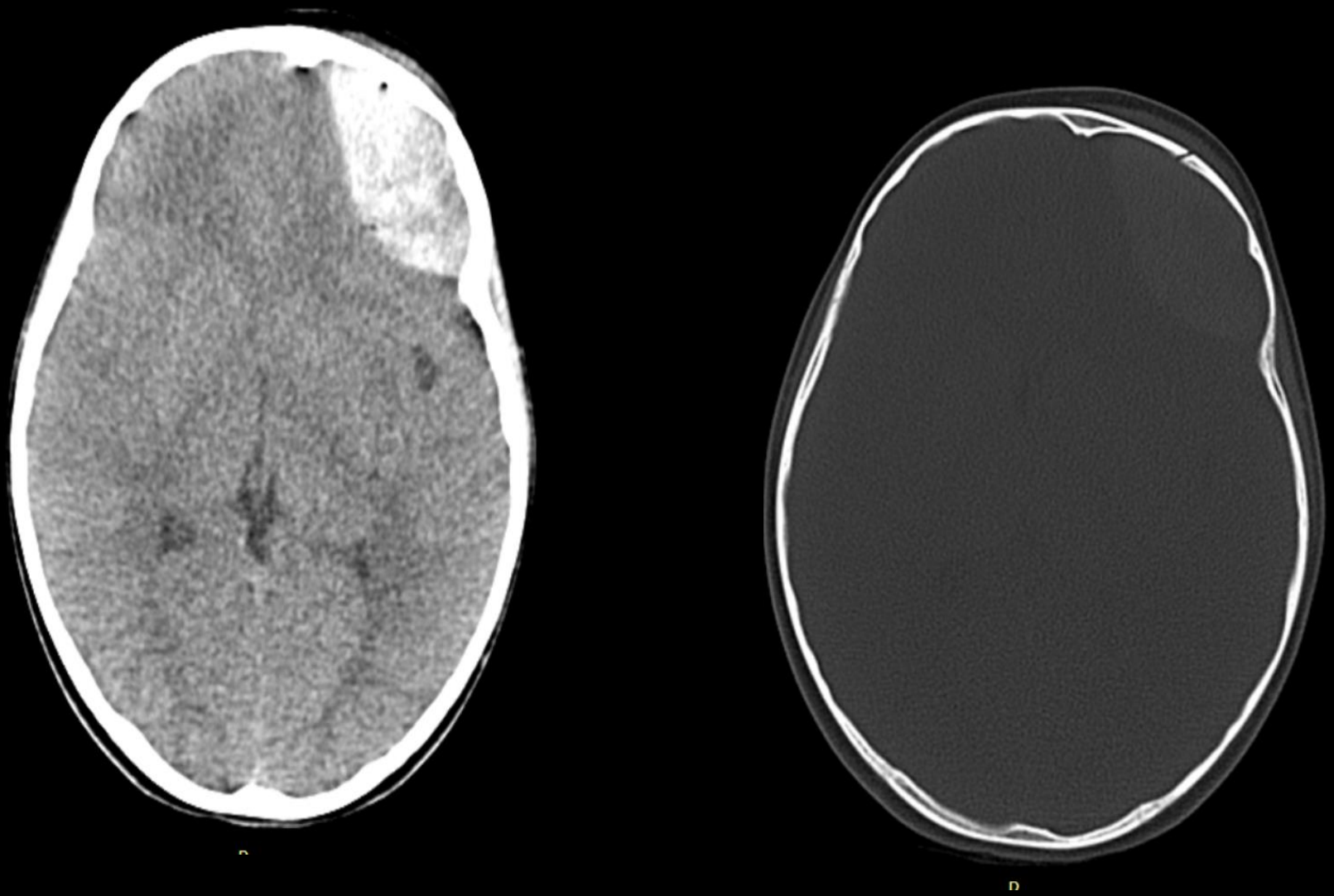
Origin: Arterial (middle meningeal artery) – associated with skull fracture

Lens in shape

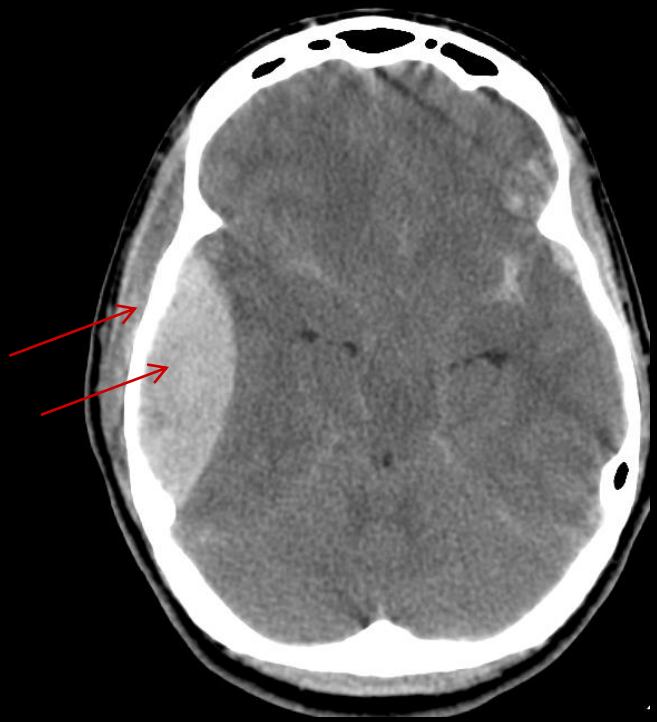
Treatment: surgical - Craniotomy



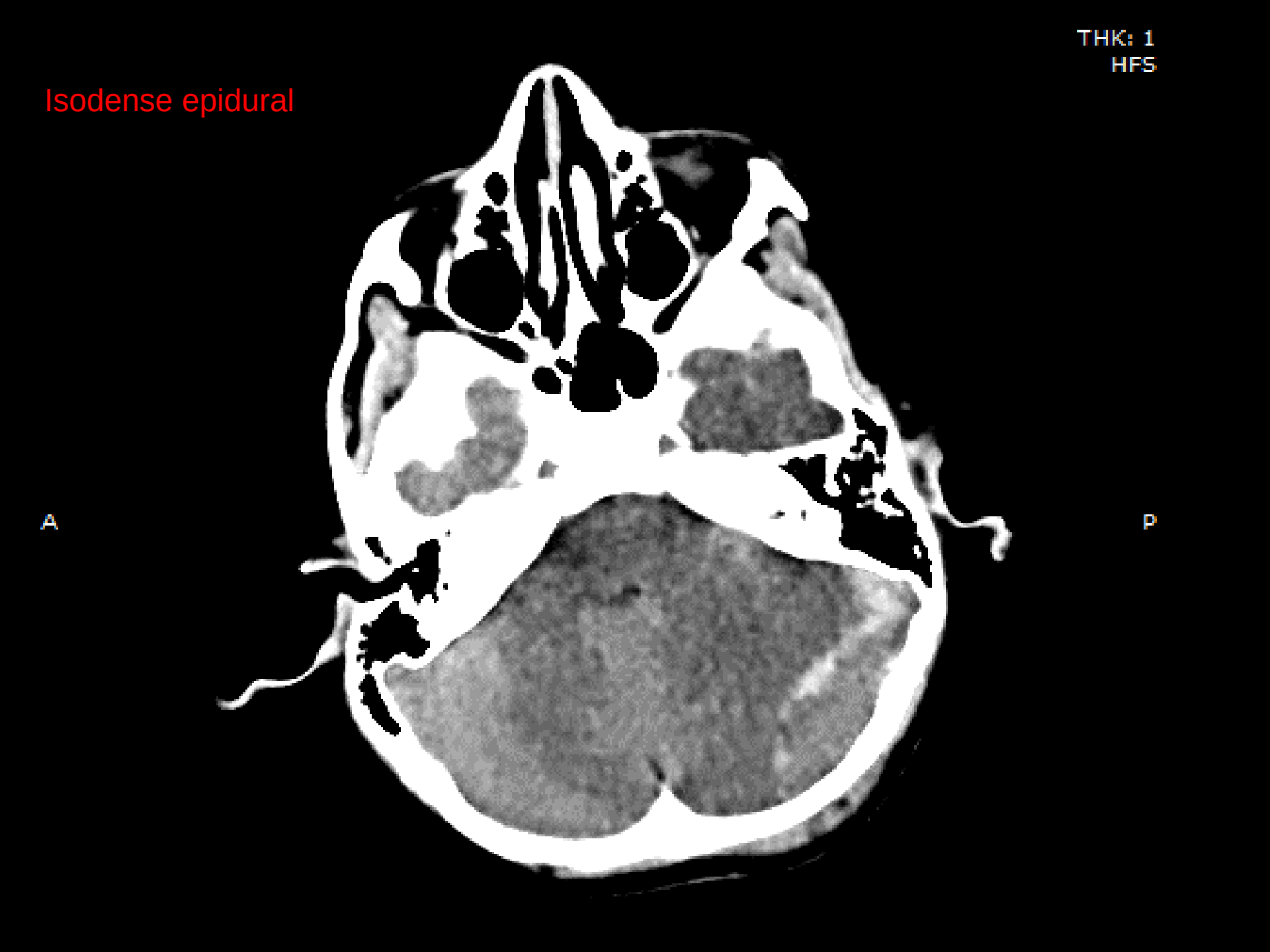
Acute epidural hemorrhage



Acute epidural hemorrhage with overlying fracture



Note the soft tissue swelling adjacent to the hematoma explaining the mechanism of the injury


Isodense epidural
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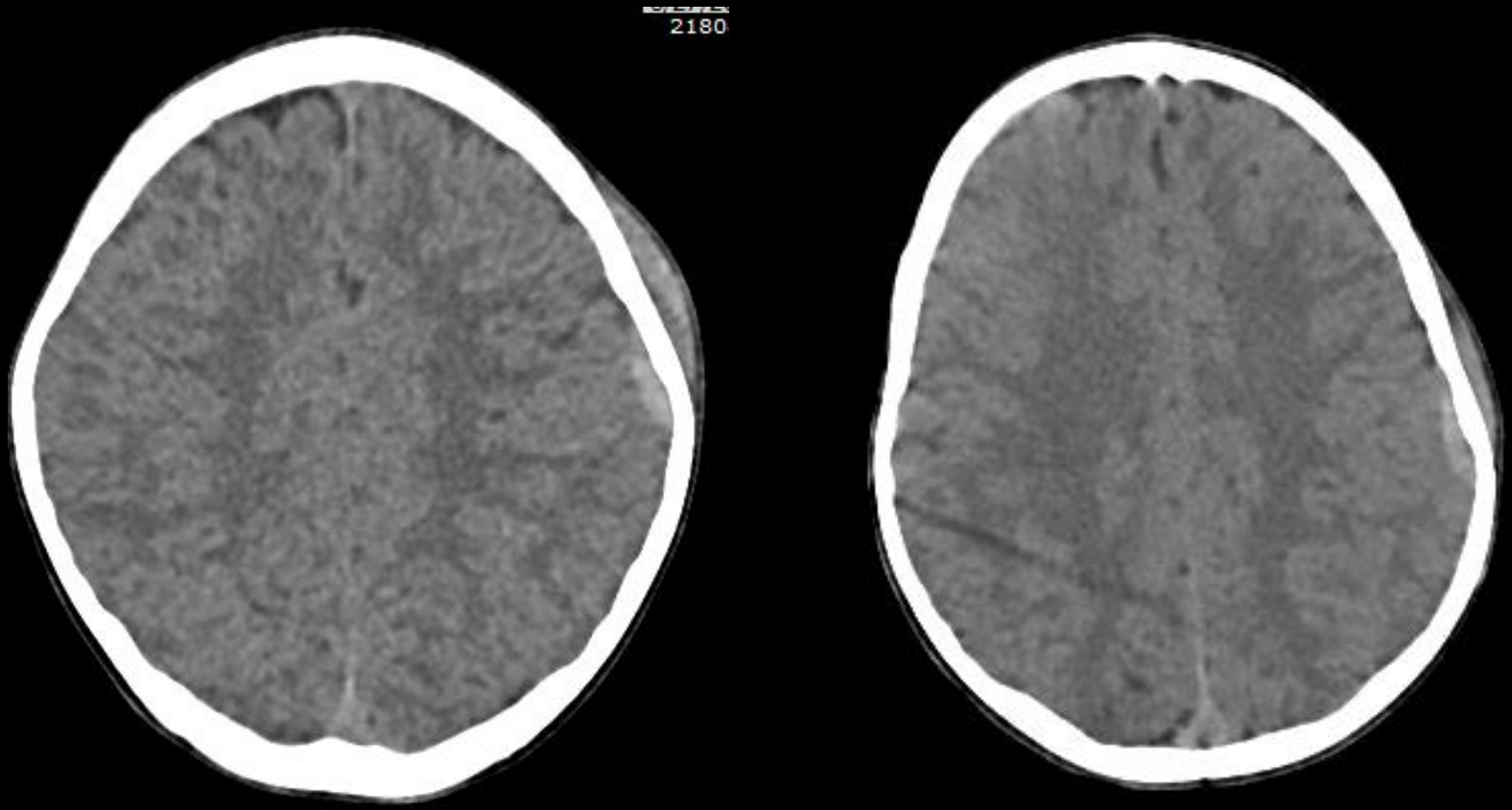


After 3 days follow up

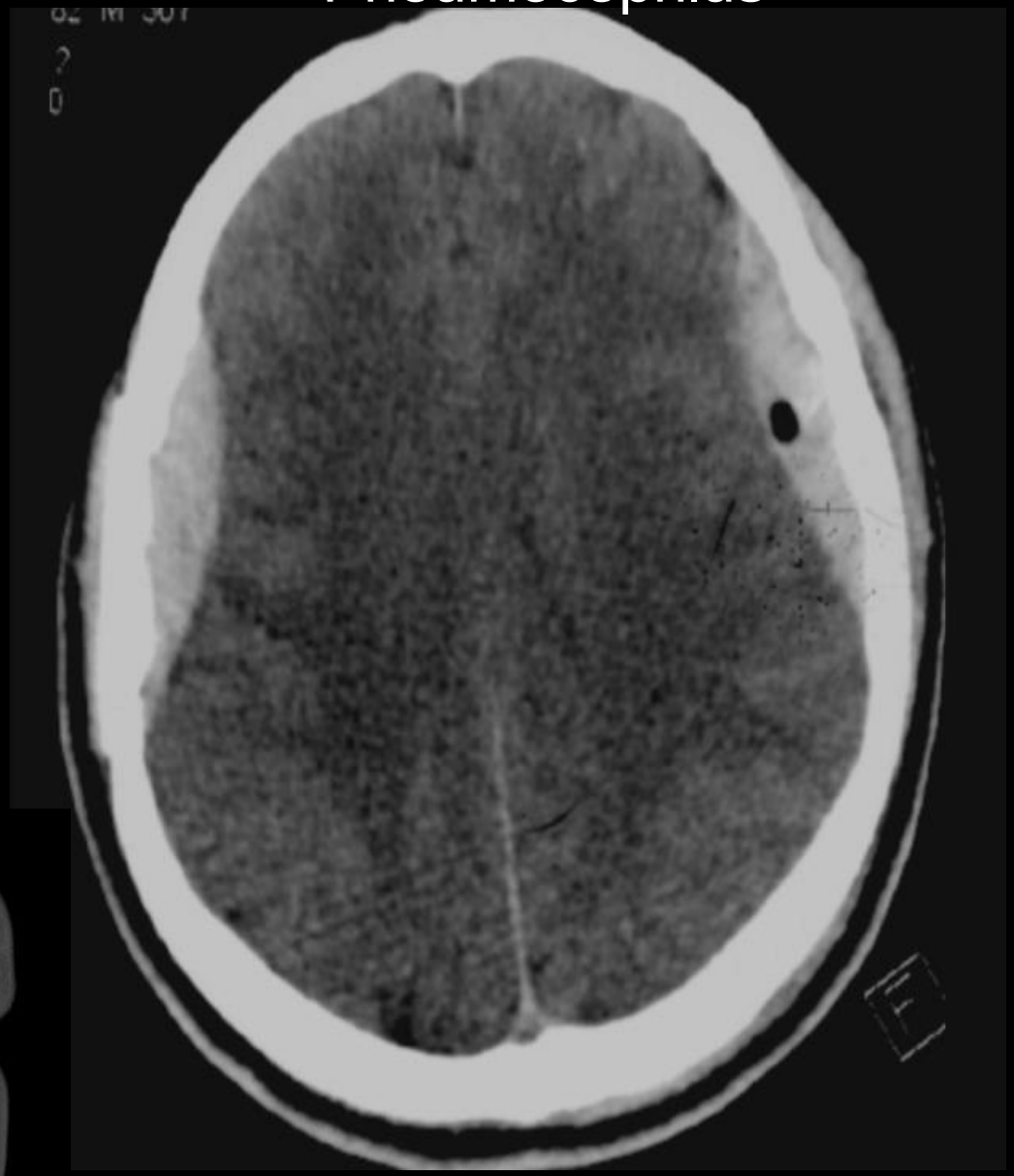
The epidural with hypodensity :

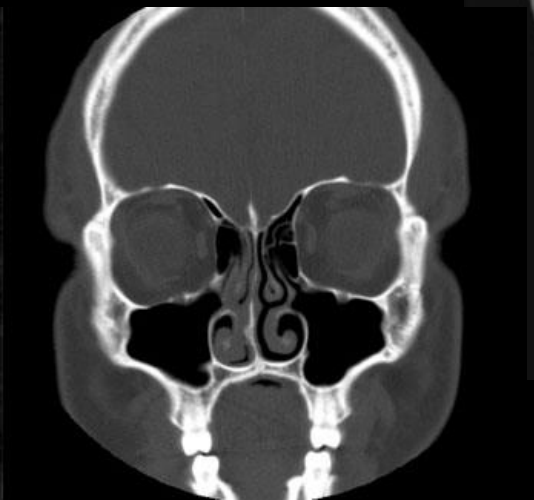
Clotting

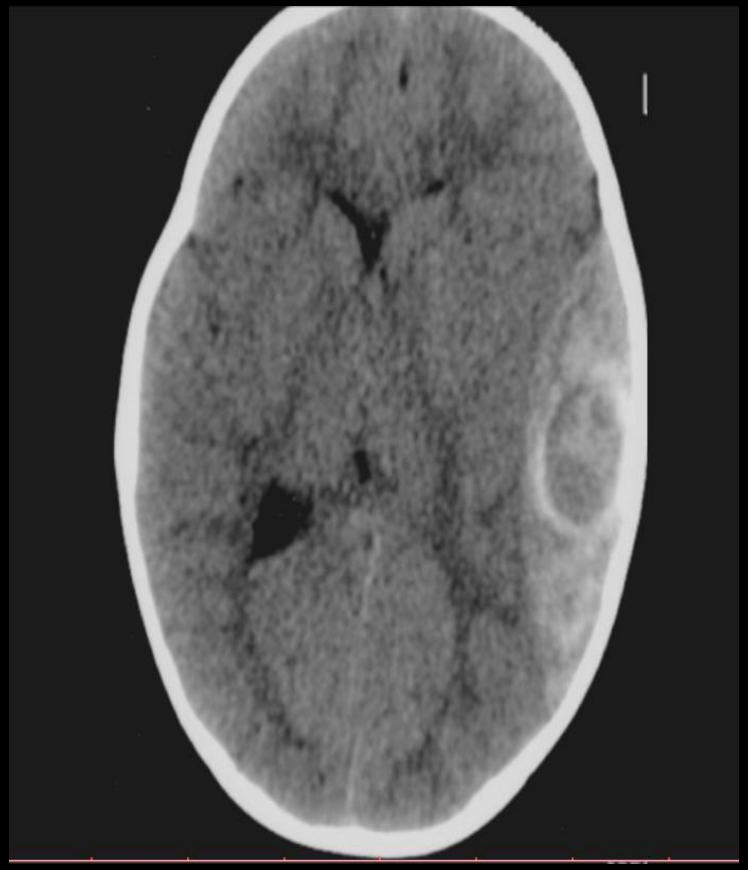
Pneumocephlus

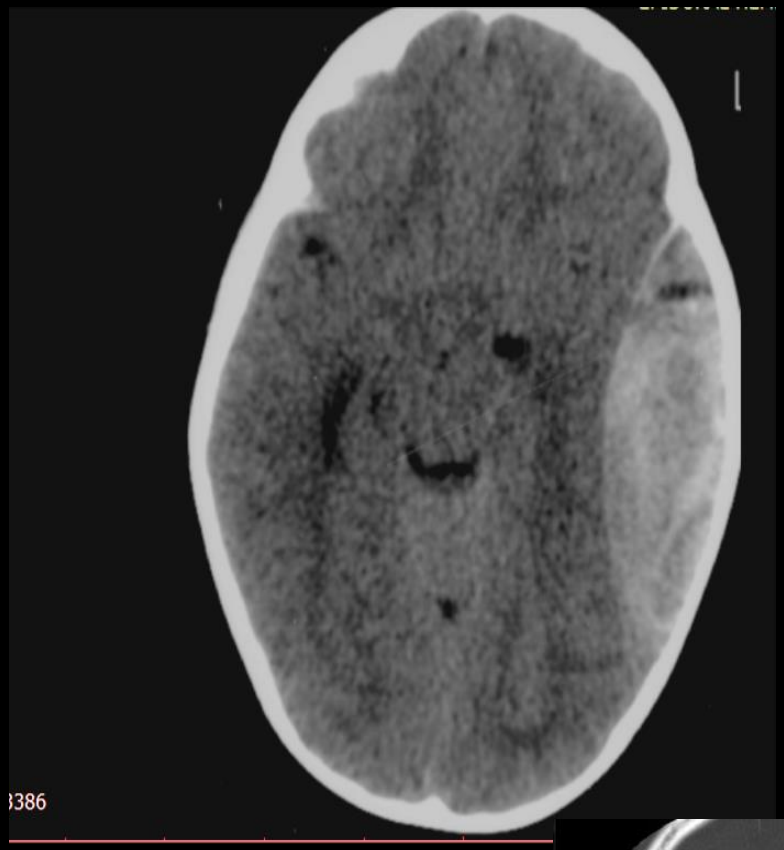
whirl sign

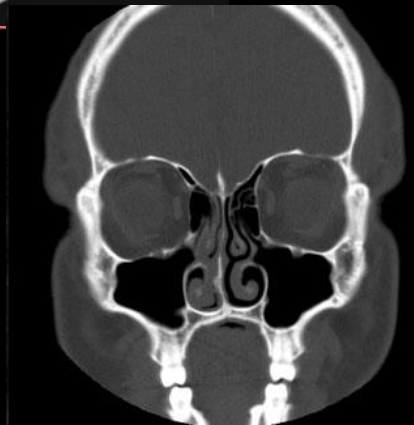
Pneumocephlus



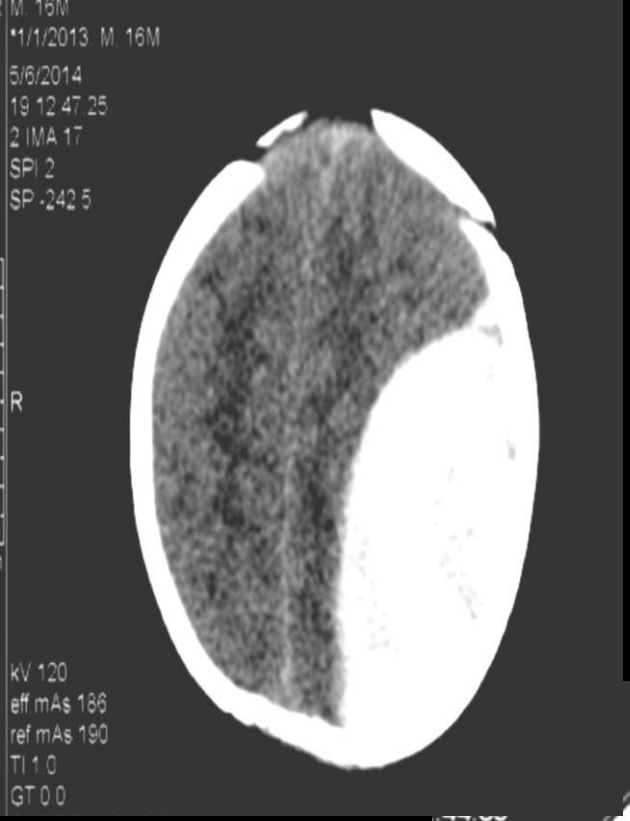








Clotting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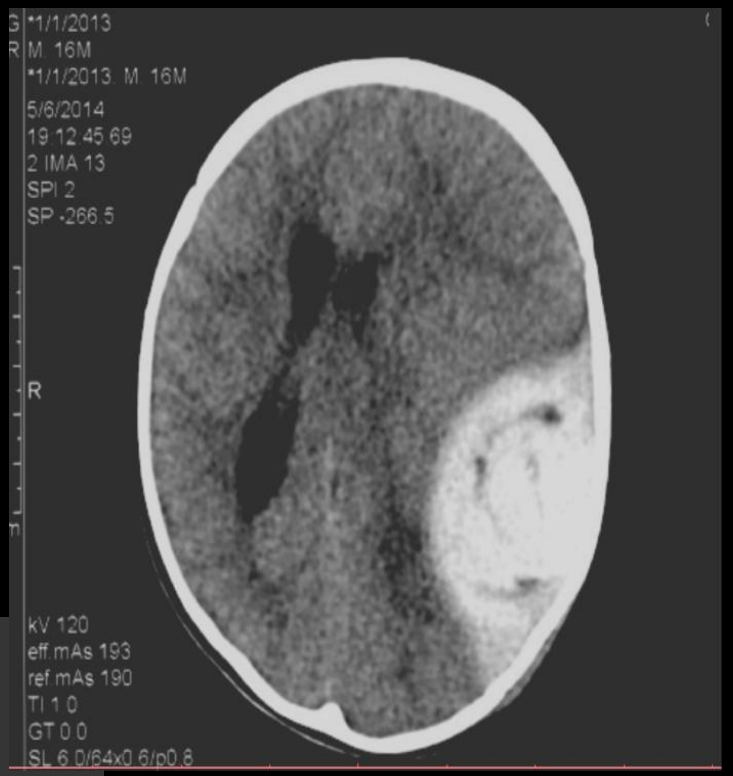
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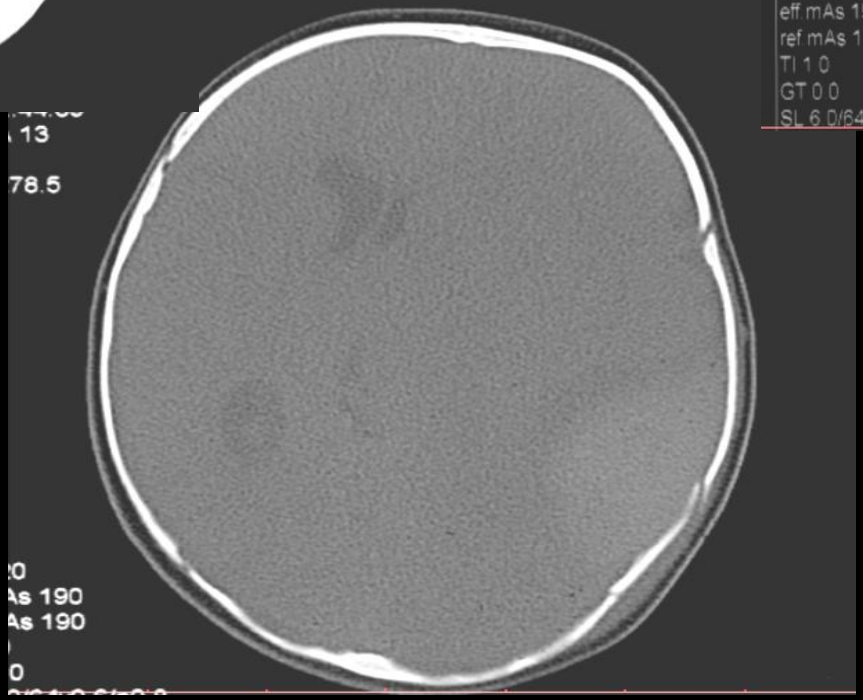
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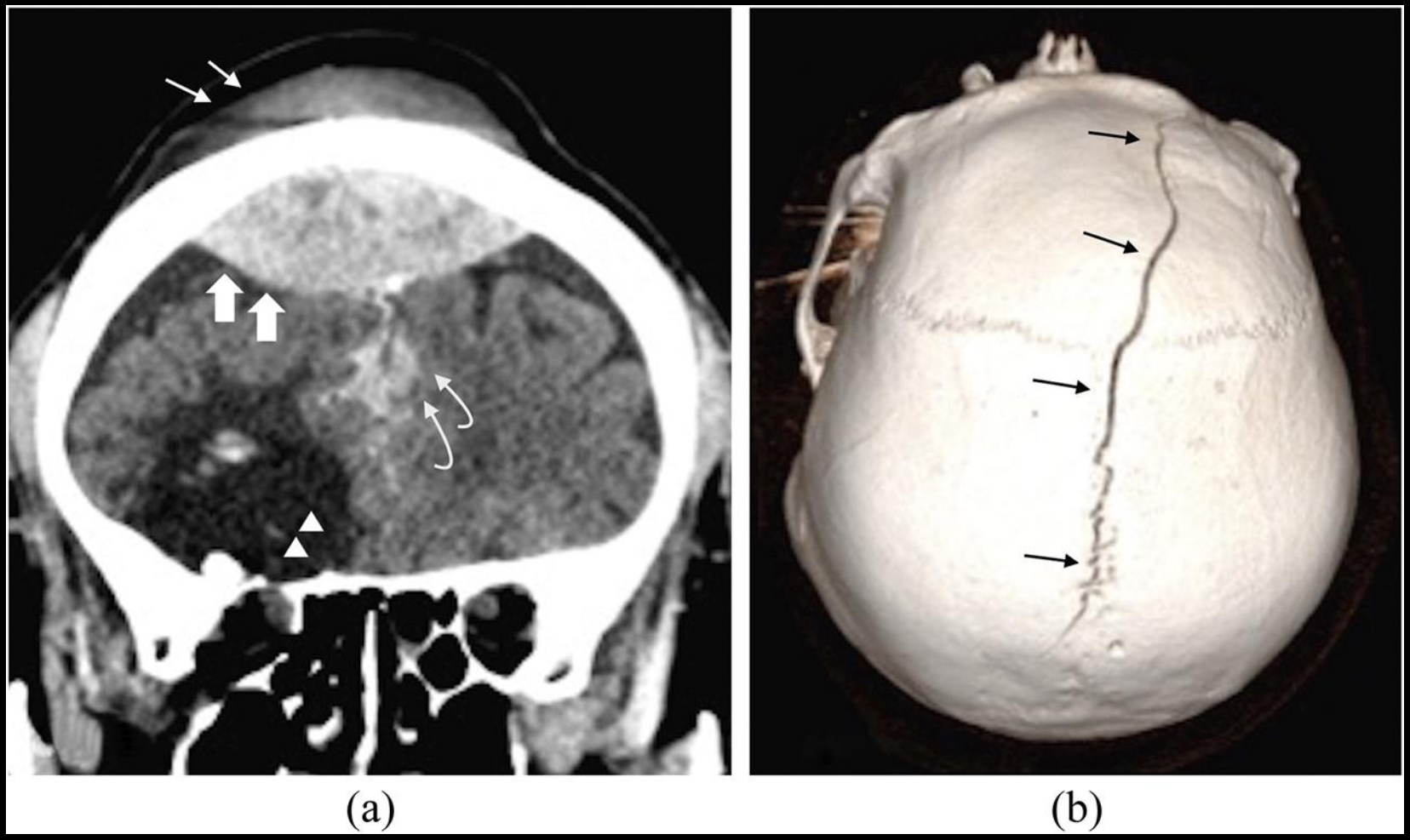

(a)
(b)

Subdural hematoma (SDH)

Mechanism: caused by traumatic tear of bridging veins (rarely arteries).

In contradistinction to EDH, of skull fractures.

Common in infants (child abuse; 80% are bilateral or interhemispheric) and elderly patients (20% are bilateral).

Subdural hematoma (SDH)

Imaging Features

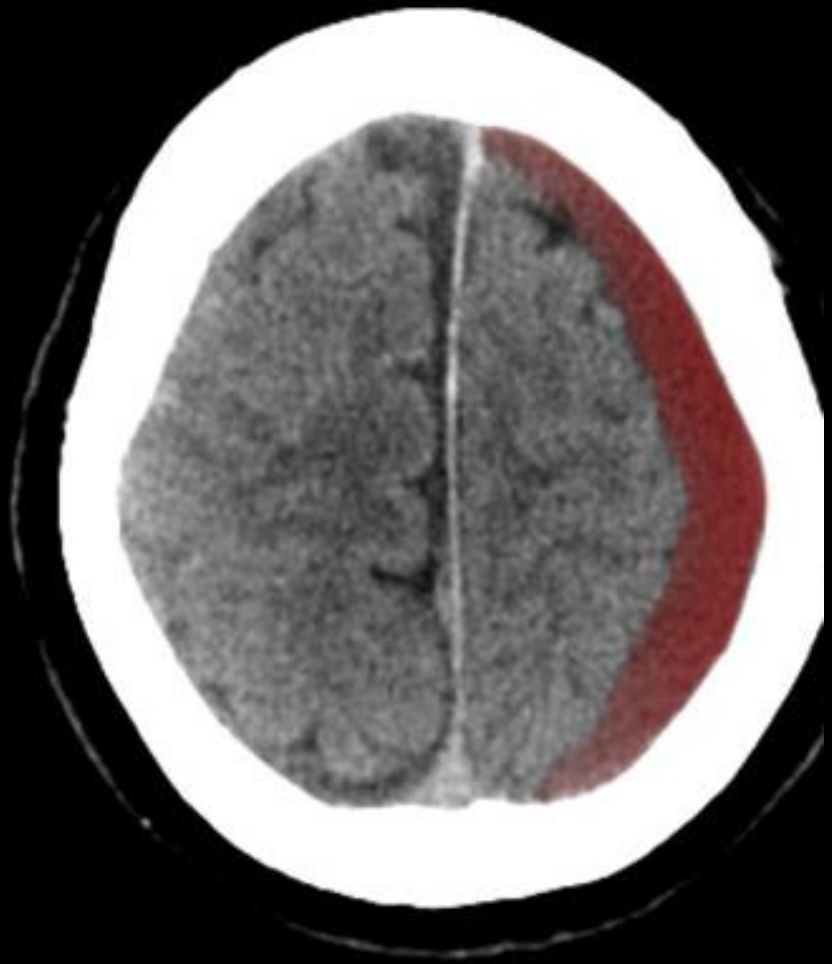
- 95% supratentorial
- Crescentic shape along brain surface
- Crosses suture lines
- Does not cross dural reflections (falx, tentorium)
- MRI > CT particularly for:
  - Bilateral hematomas
  - Interhemispheric hematomas
  - Hematomas along tentorium
  - Subacute SDH

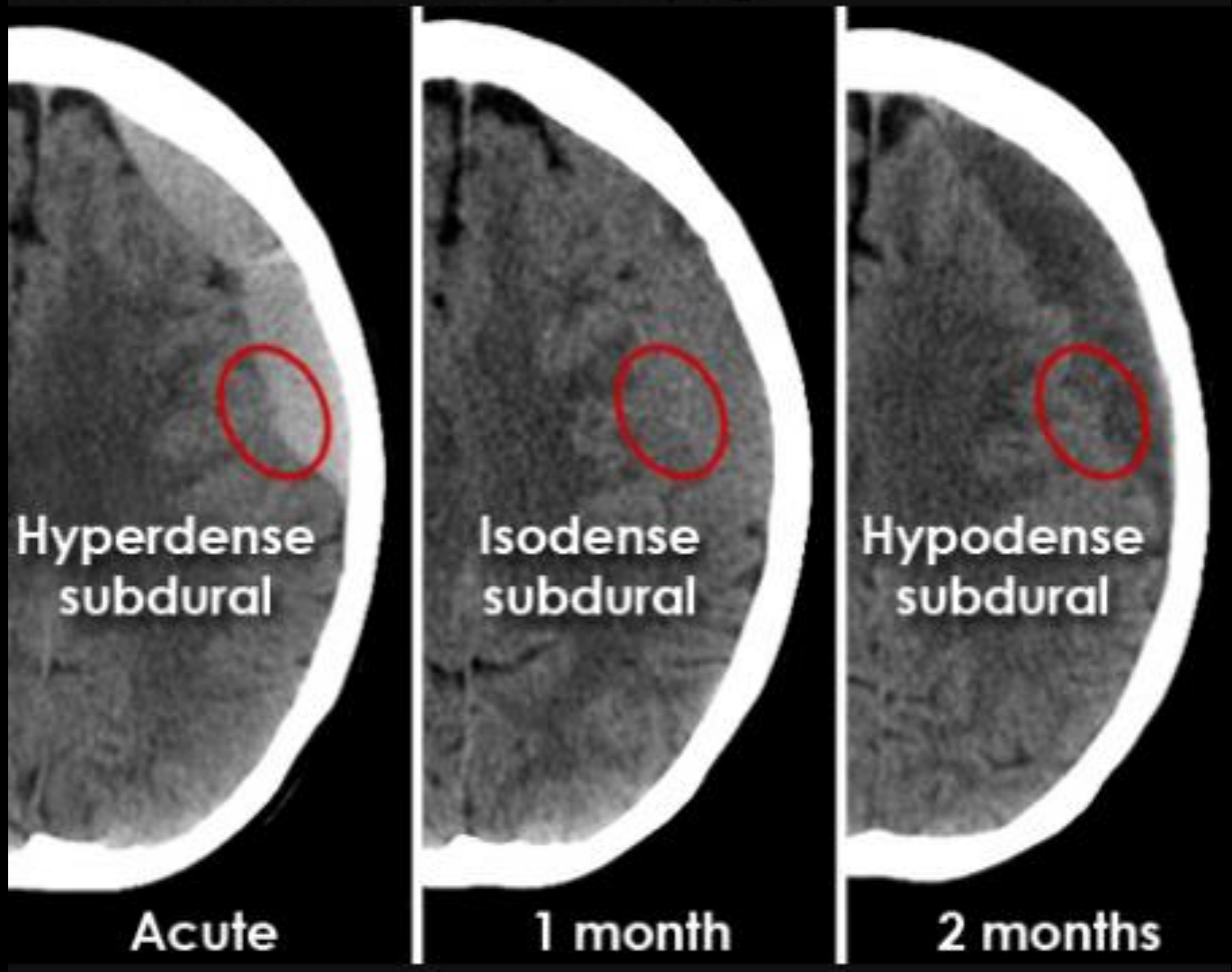
Subdural hemorrhage

Origin: Venous

Treatment: Burr hole

Cresent (semilunar) in shape

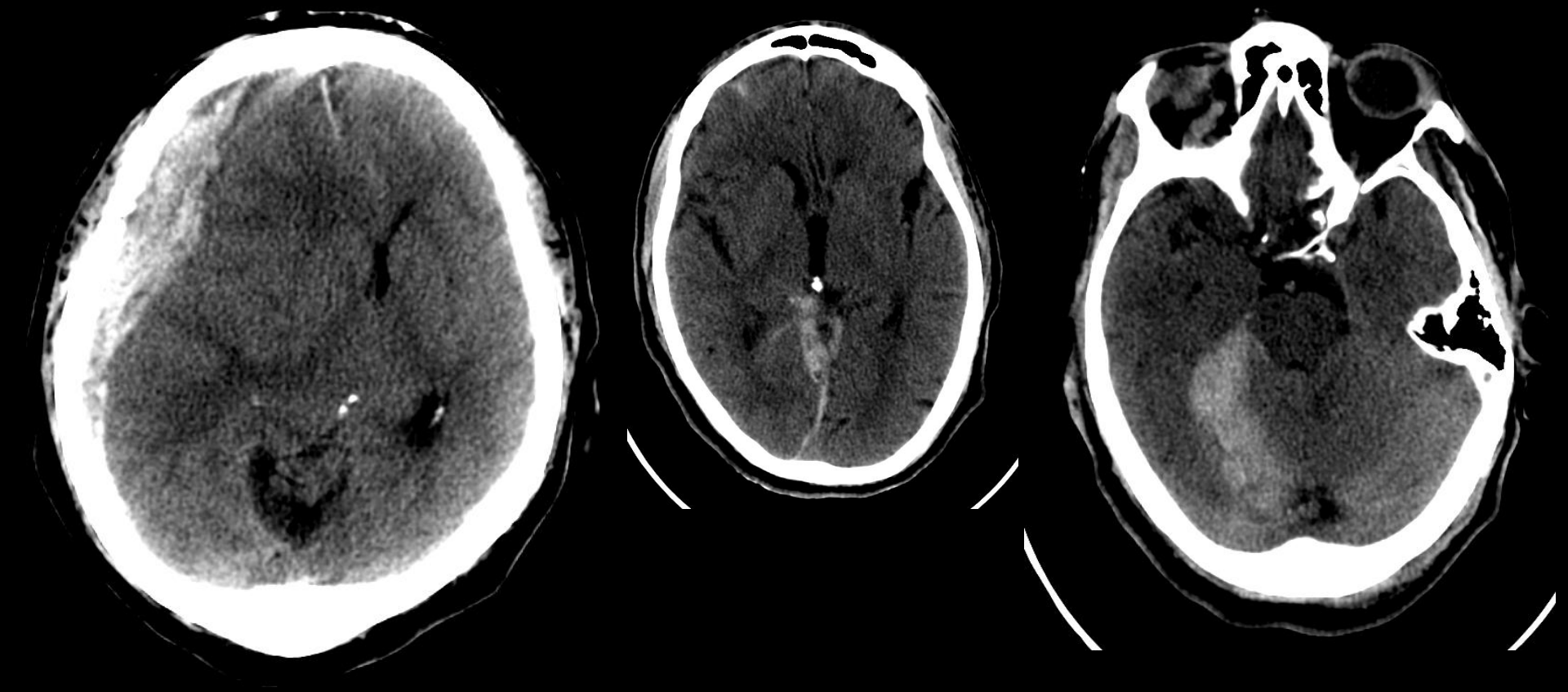



Hyperdense subdural
Isodense subdural
Hypodense subdural
Acute
1 month
2 months

Stages of Subdural hemorrhage

Stage of hemorrhage	Appearance	Blood product
Acute (days)	Hyperdense	Oxyhemoglobin Deoxyhemoglobin
Subacute (Weeks)	Isodense	Methemoglobin (Intracellular and extracellular)
Chronic (months)	Hypodense	Ferritin Hemosiderin

Acute Subdural Hematoma

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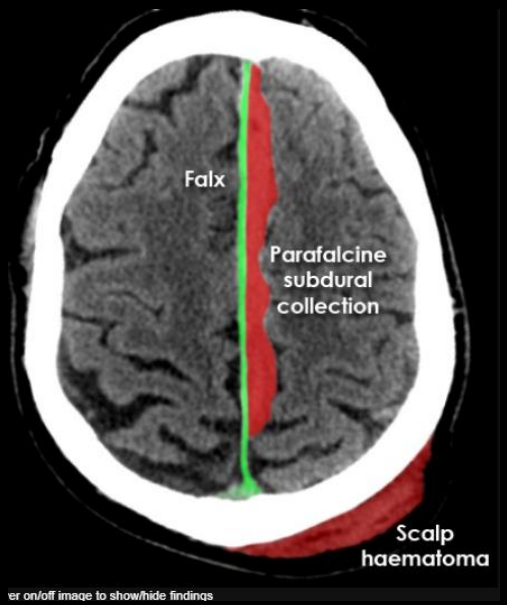
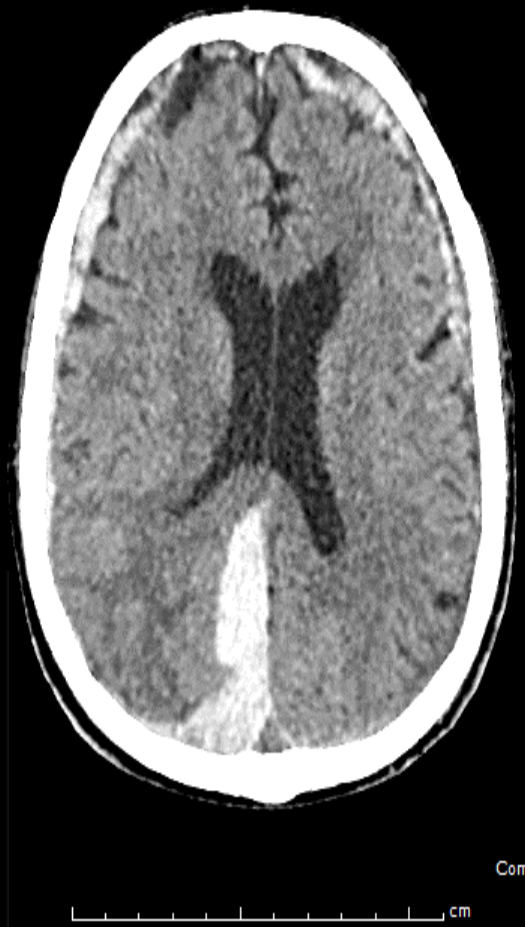


er on/off image to show/hide findings

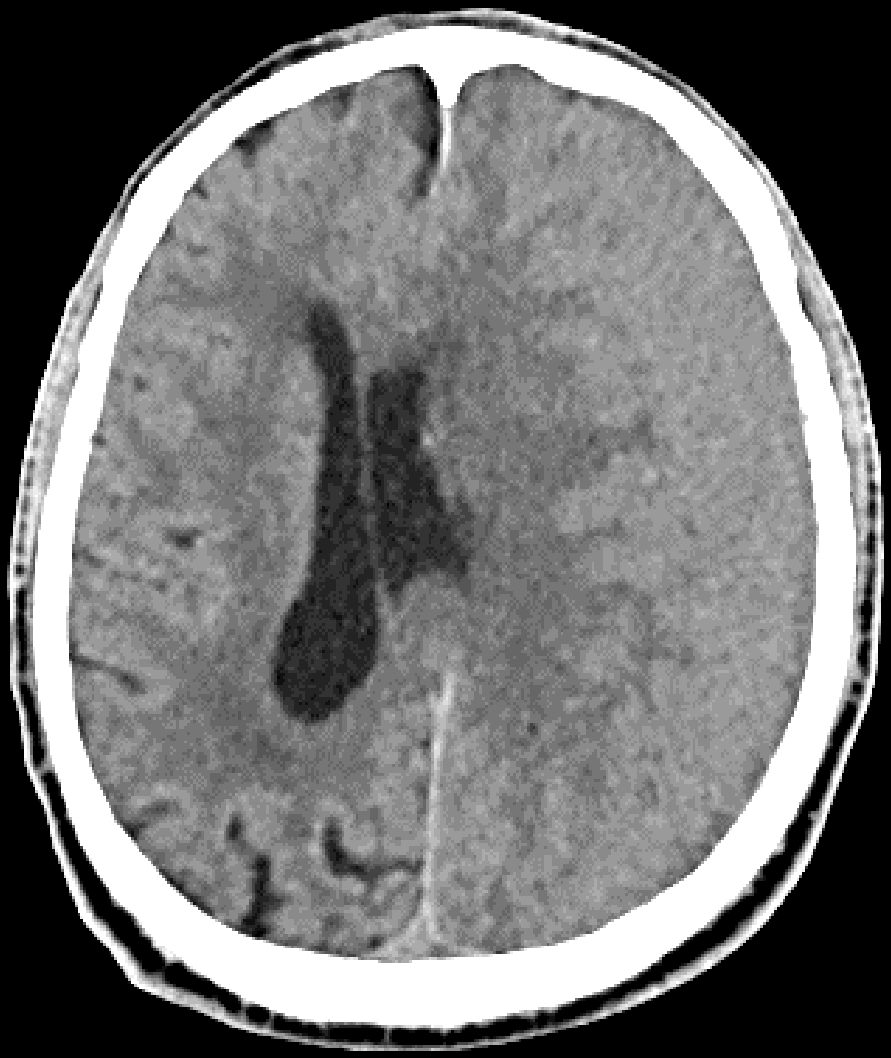


Compressed 11:1
IM: 63 SE: 2

cm

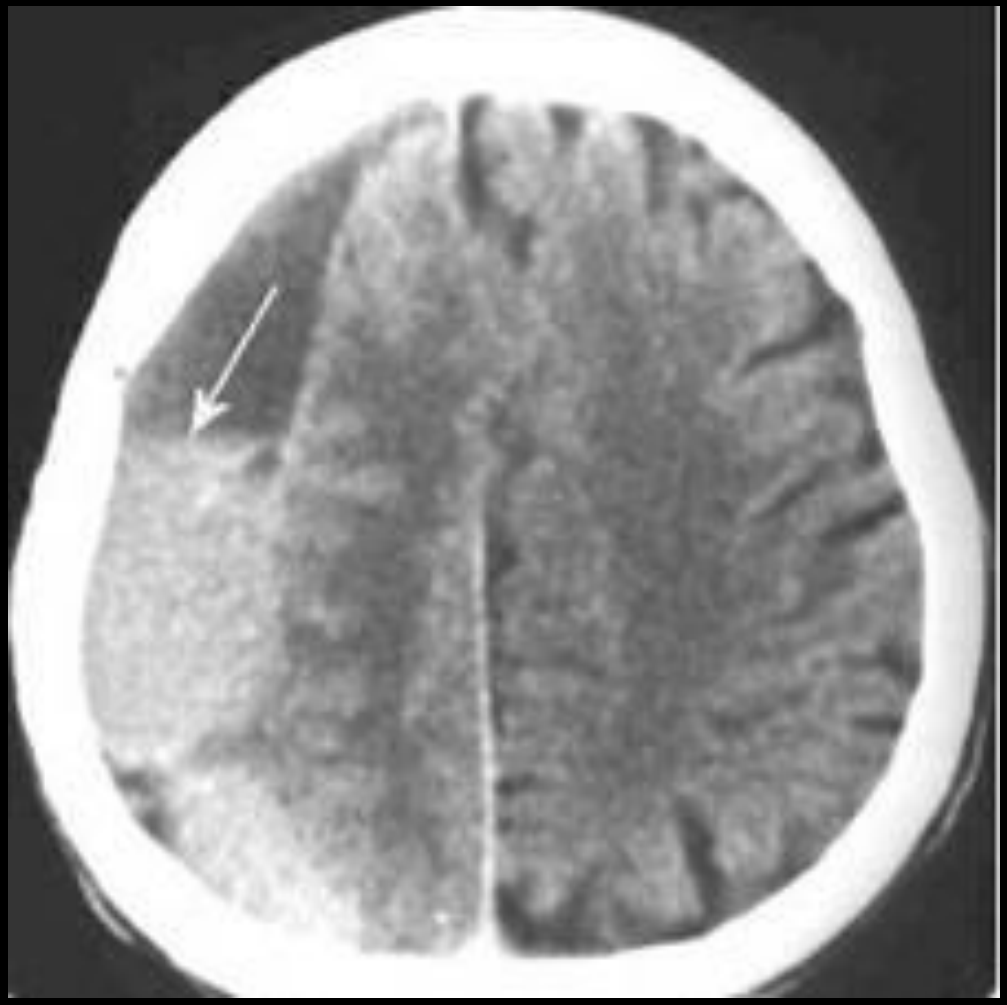

a

Subacute Subdural Hemorrhage



Chronic SDH with rebleeding

Acute on top of chronic Subdural Hemorrhage



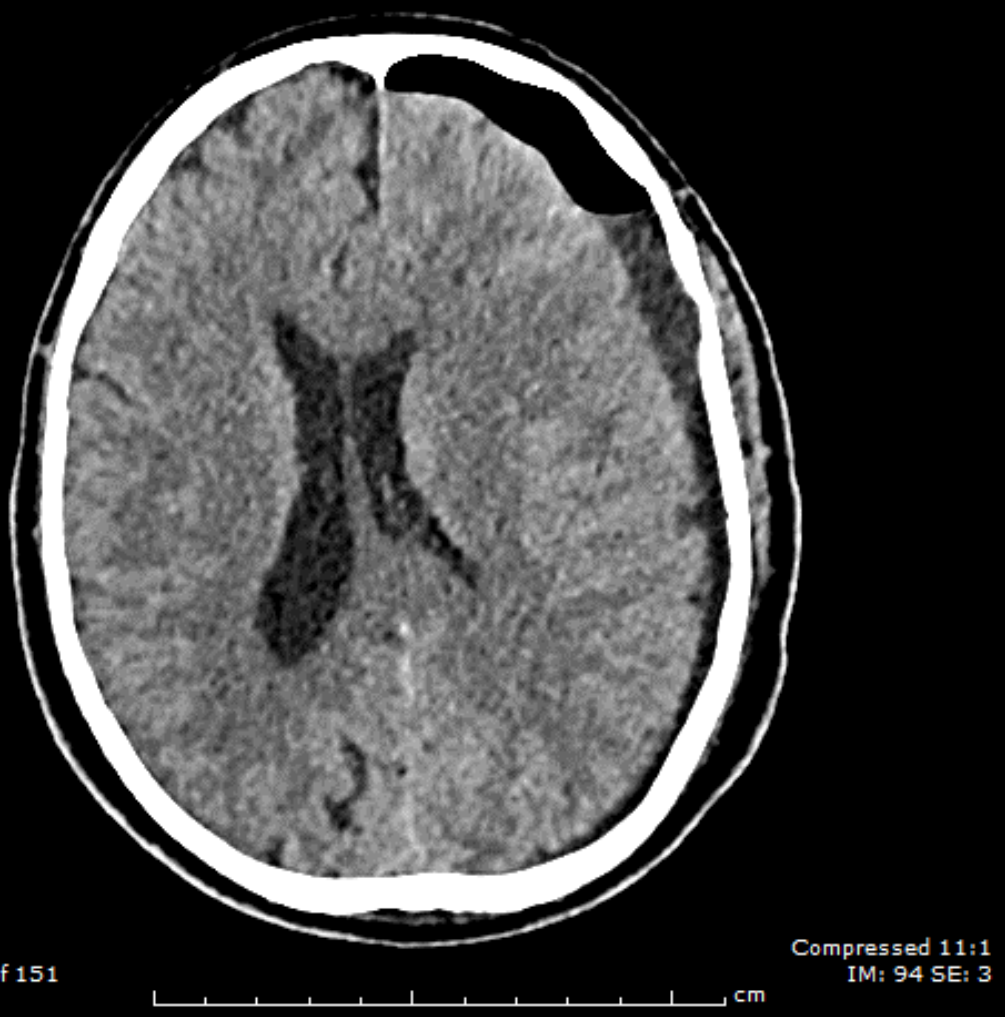

SUBDURAL HEMMO
D

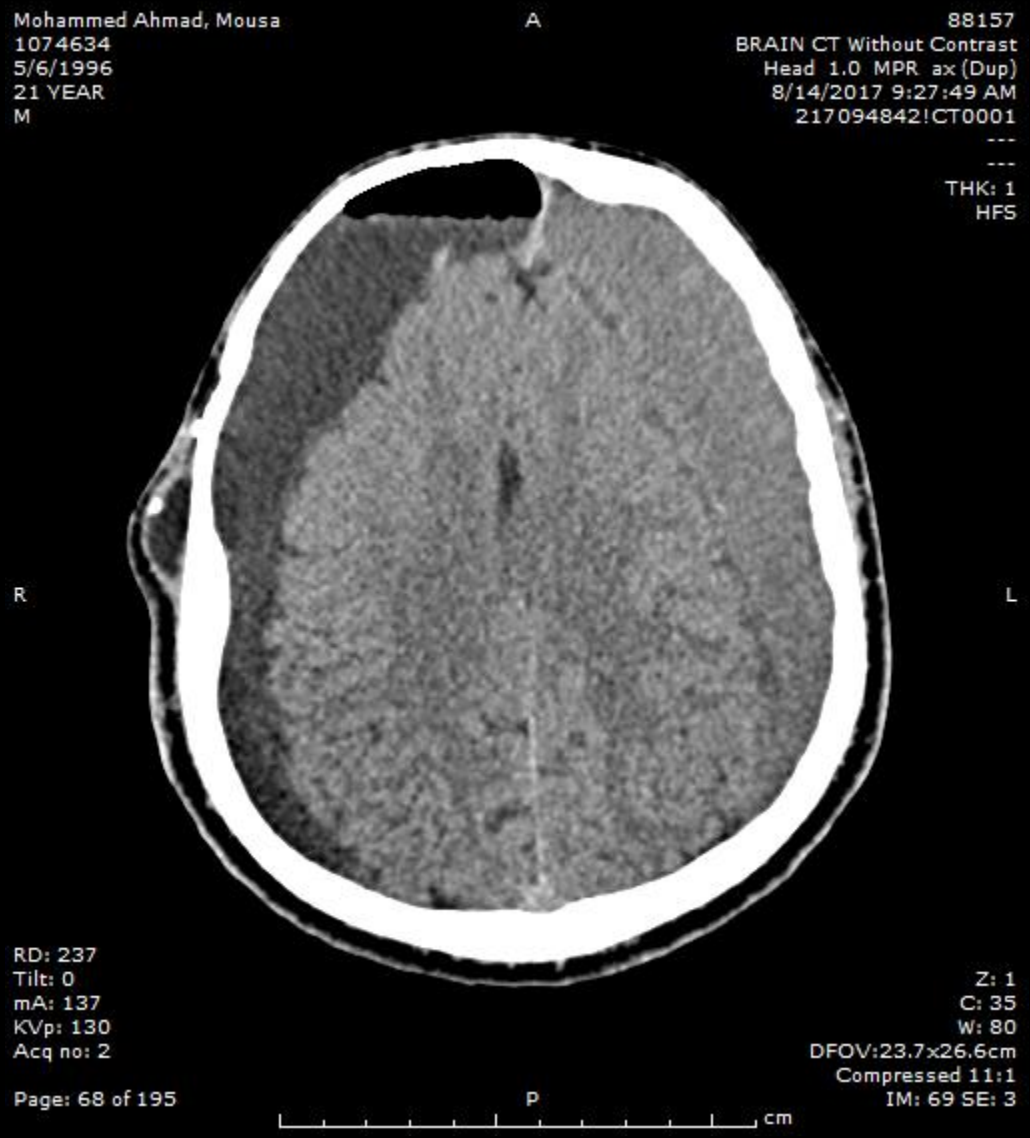
Hyperacute component

The less dense blood (grey) is not due to a chronic haematoma, it is hyperacute blood which has not yet had time to clot


LOC: -29.10
THK: 1
HFS
L
C: 57
W: 80
DFOV:22.4x22.4cm
Compressed 11:1
IM: 114 SE: 4
P
cm

Subdural hematoma with pnemocephlus




Mohammed Ahmad, Mousa
1074634
5/6/1996
21 YEAR
M
A
88157
BRAIN CT Without Contrast
Head 1.0 MPR ax (Dup)
8/14/2017 9:27:49 AM
217094842!CT0001

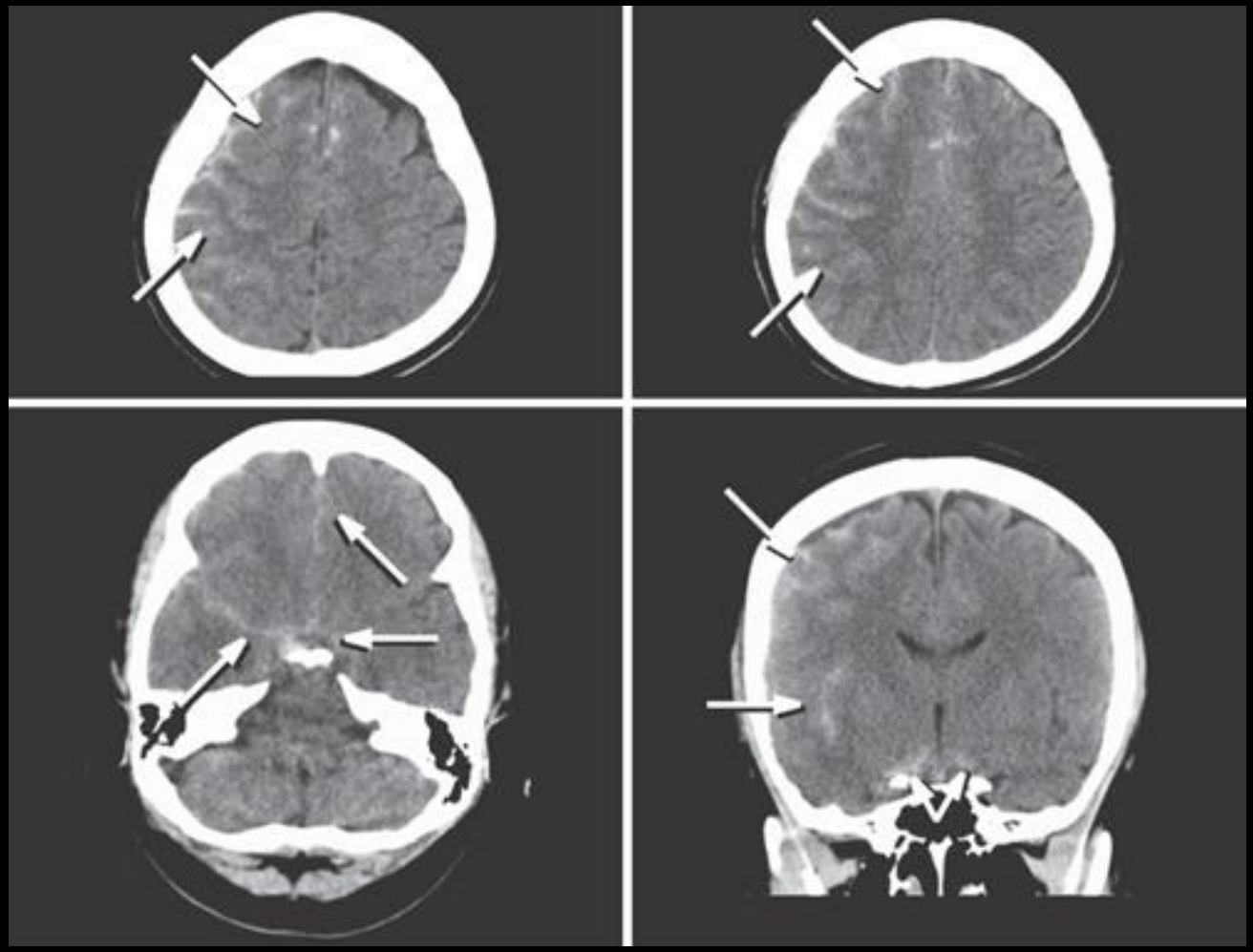
THK: 1
HFS
R
L
RD: 237
Tilt: 0
mA: 137
KVp: 130
Acq no: 2
Page: 68 of 195
P
cm
Z: 1
C: 35
W: 80
DFOV:23.7x26.6cm
Compressed 11:1
IM: 69 SE: 3

Subarachnoid hemorrhage

Mechanism:results from injury to small subarachnoid vessels or extension of intraparenchymal hemorrhage beyond the pial limiting membrane and into the subarachnoid space.

CT: SAH appears as areas of high-density conforming to the shape of the cerebral sulci and basal cisterns.

- Subarachnoid Hemorrhage : post trauma or ruptured aneurysm




AY-2008
4:56.83
SUBARCHNOID HEMMORAGE SUB
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AY-2008
4:53.43
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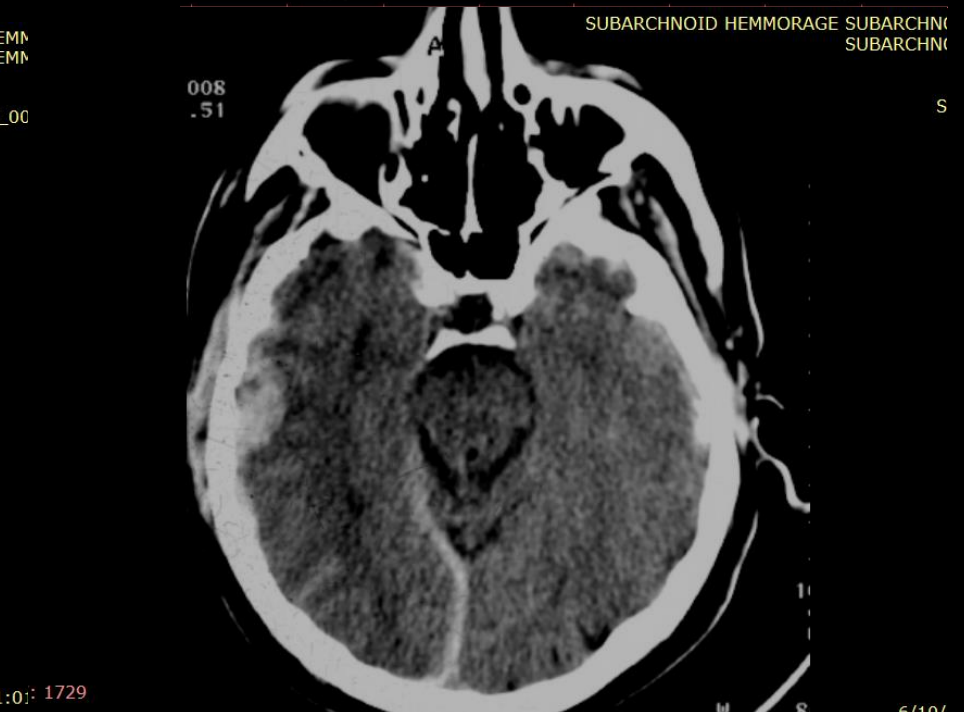
6/10/2017 1:0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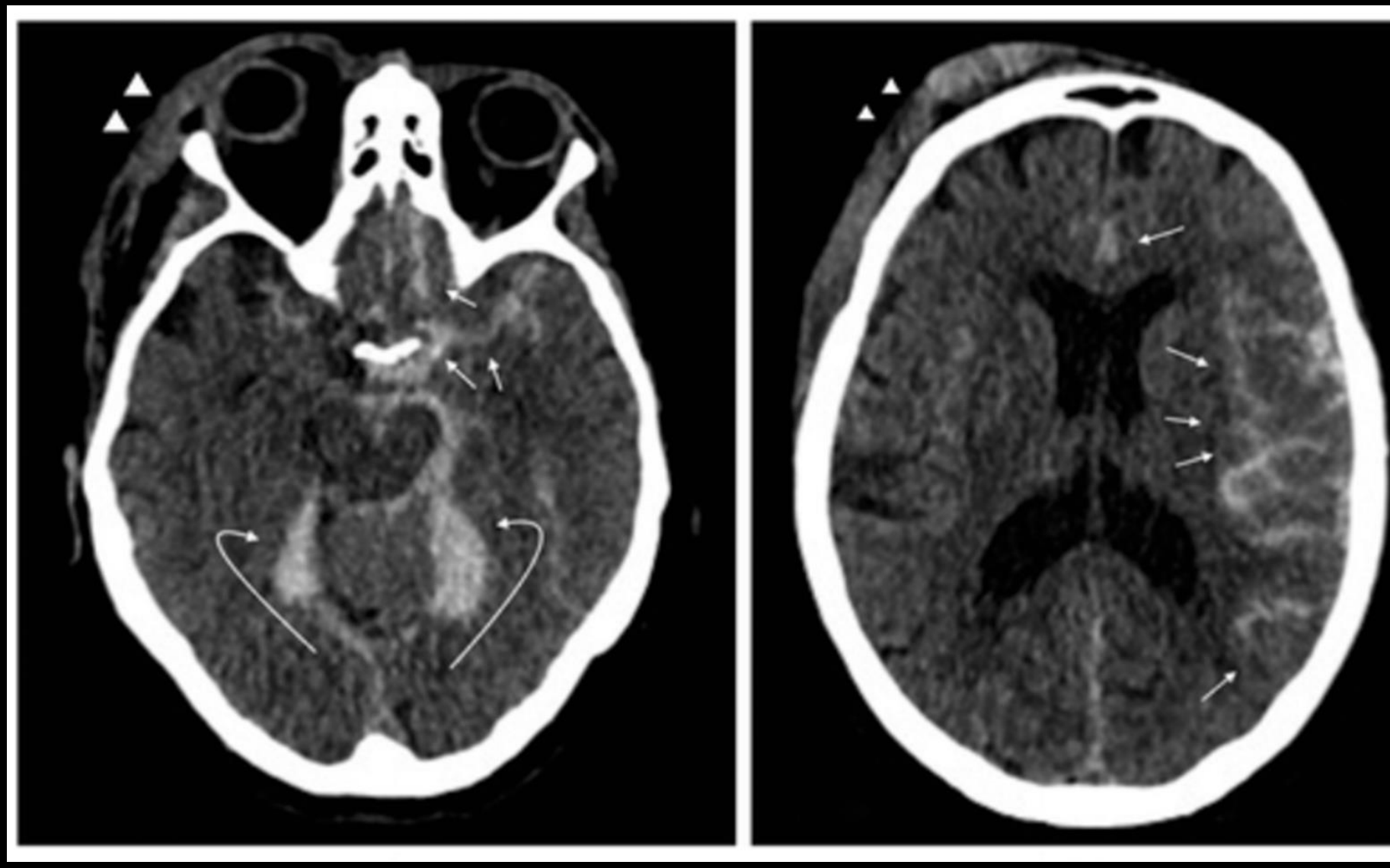



-2008
:50.13
546.6
W

Intraventricular hemorrhage

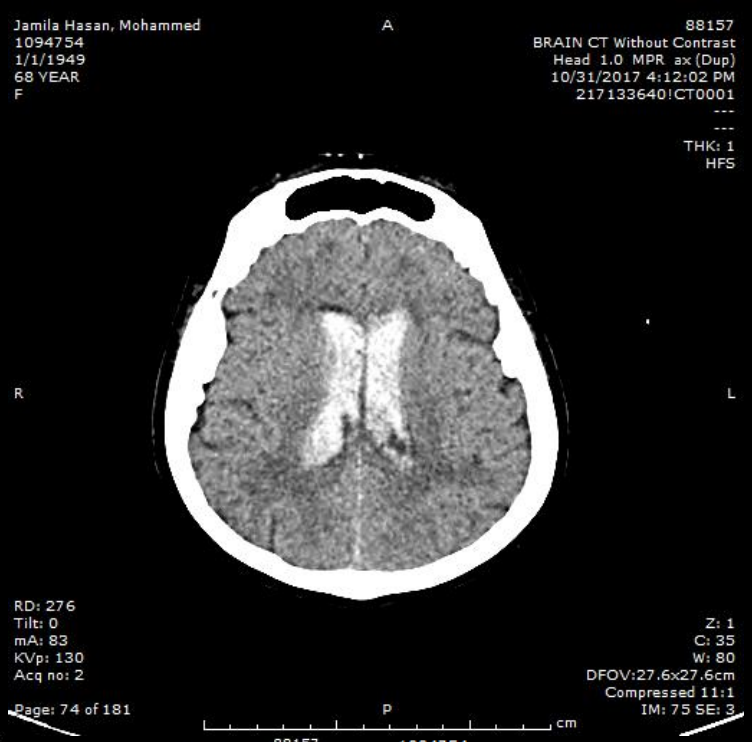
Mechanism:

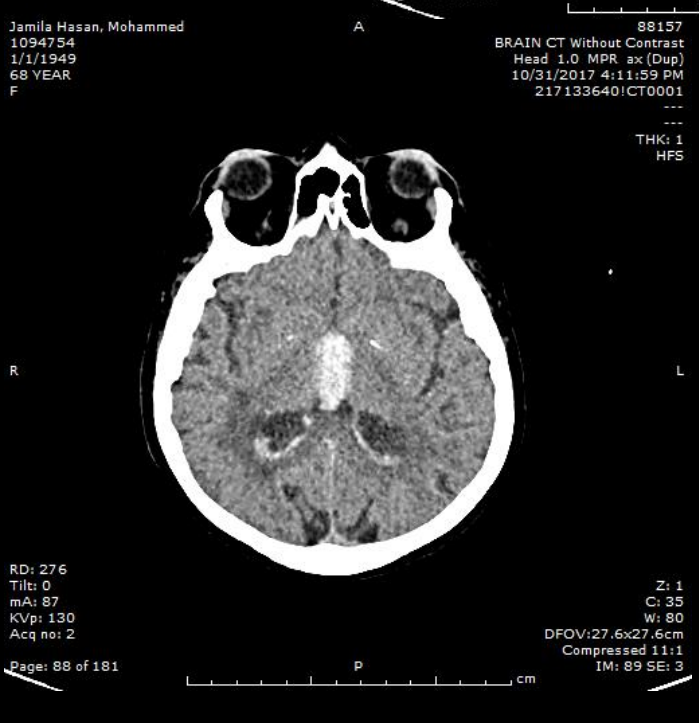
- result from shearing of the choroid plexus or subependymal veins along the surface of the ventricles,
- an extension of a parenchymal hematoma into the ventricles,
- by retrograde flow of blood from a SAH into the ventricular system .

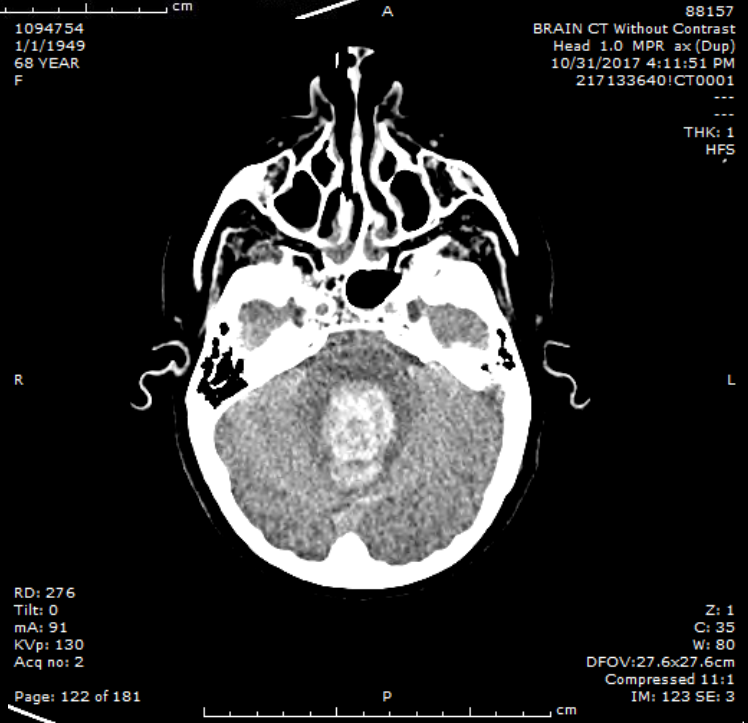
Patients with IVH can develop complications including hydrocephalus and even ependymitis

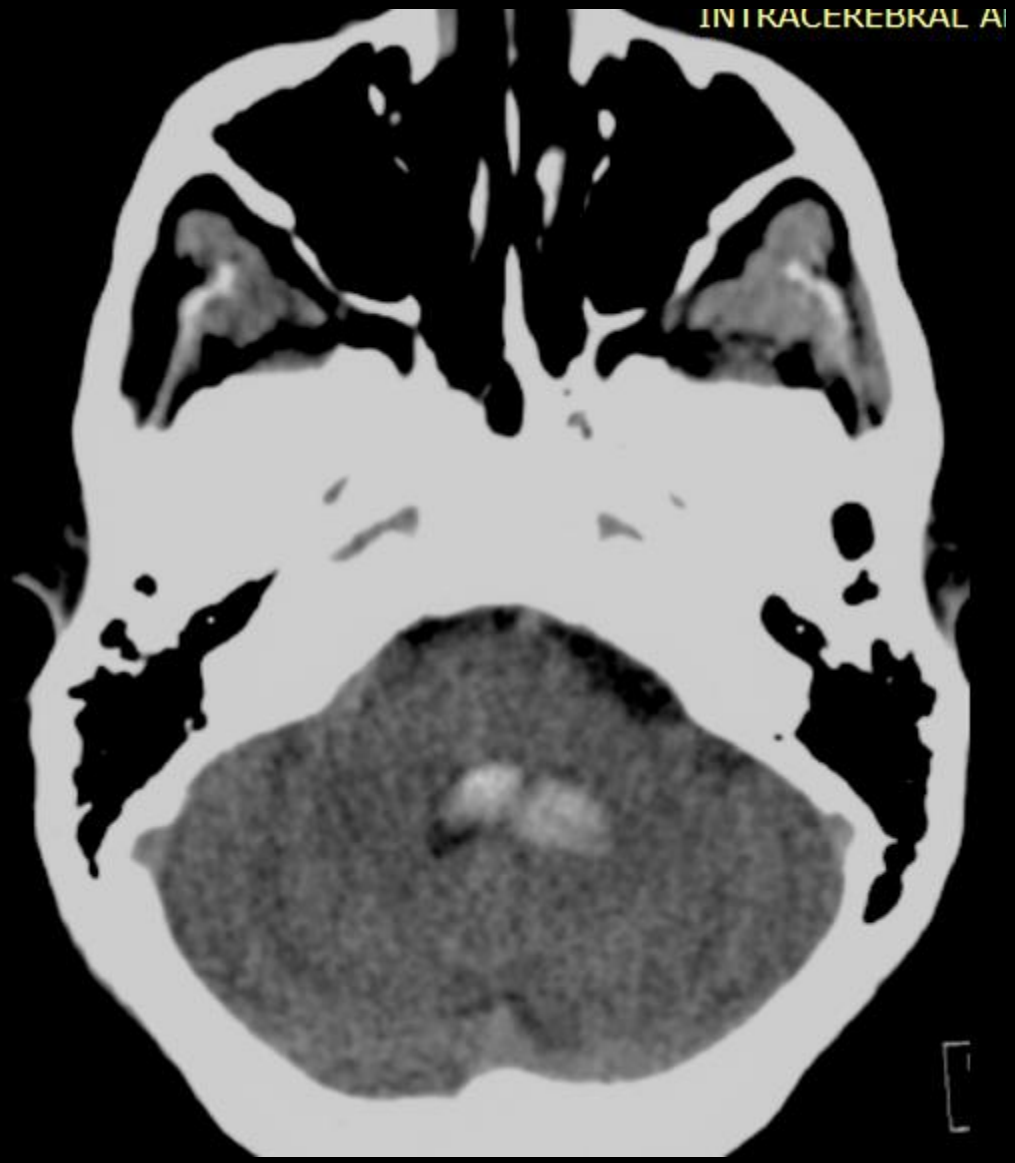
Imaging features:
On CT, IVH is most commonly seen as hyperdense collections that layer within the occipital horns .

Intraventricular hemorrhage








INTRACEREBRAL A


INTRACEREBRAL AND INTRAVENTRICULAR HEMMORAGE
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SID_II_00000014
WL: 3436 WW: 2006 [D]
13
6/10/2016 1:01:01 AM


M
-FEB-2009
:40:56.12
SID_II_00000014
: 3436 WW: 2006 [D]
12
6/10/2016 1:01:01 AM

Primary intra-axial injuries

Includes:

Diffuse axonal injury

Cortical contusion

Intracerebral hematoma

Brain stem injury

Cortical contusion

Mechanism: when the brain forcibly impacts the irregular surface of the overlying skull, which typically occurs at (coup injury) or opposite (contrecoup) the site of blunt trauma.

Contusions frequently contain hemorrhagic foci ranging in size from punctate cortical surface petechiae to much larger confluent regions of hemorrhage occupying an entire lobe.

Cortical contusion

Imaging Features

Multifocal and bilateral, usually involving the superficial grey matter.

Location:

Anterior temporal lobes, 50% (adjacent the petrous bone and posterior to the greater *sphenoid wing)*

Frontal lobes, 30% (superior to the cribriform plate, orbit roof and lesser sphenoid wing)

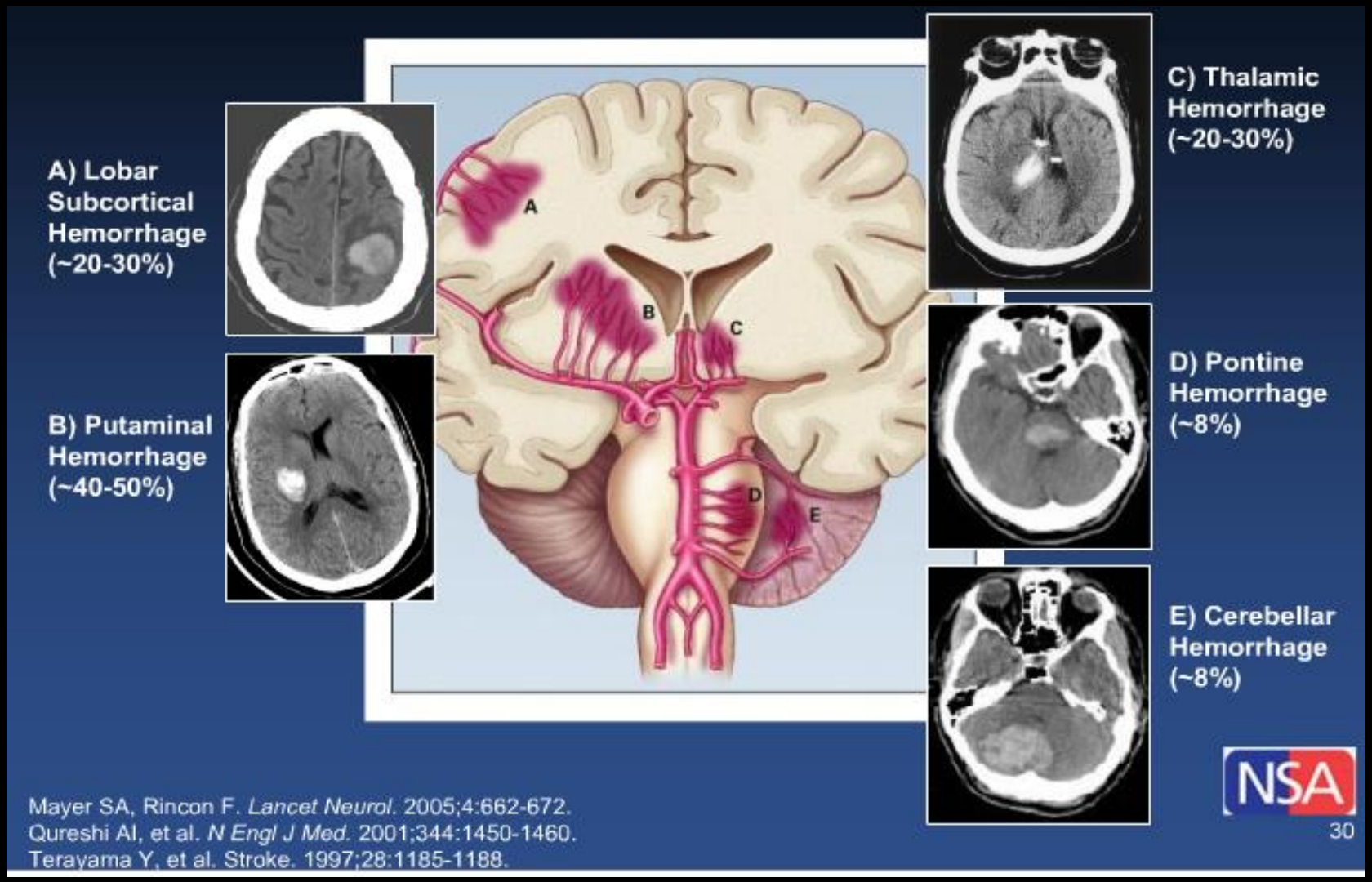
CT appeared as irregular hyperdense foci at gyral surfaces with associated areas of surrounding vasogenic edema

Intraparenchymal hematomas

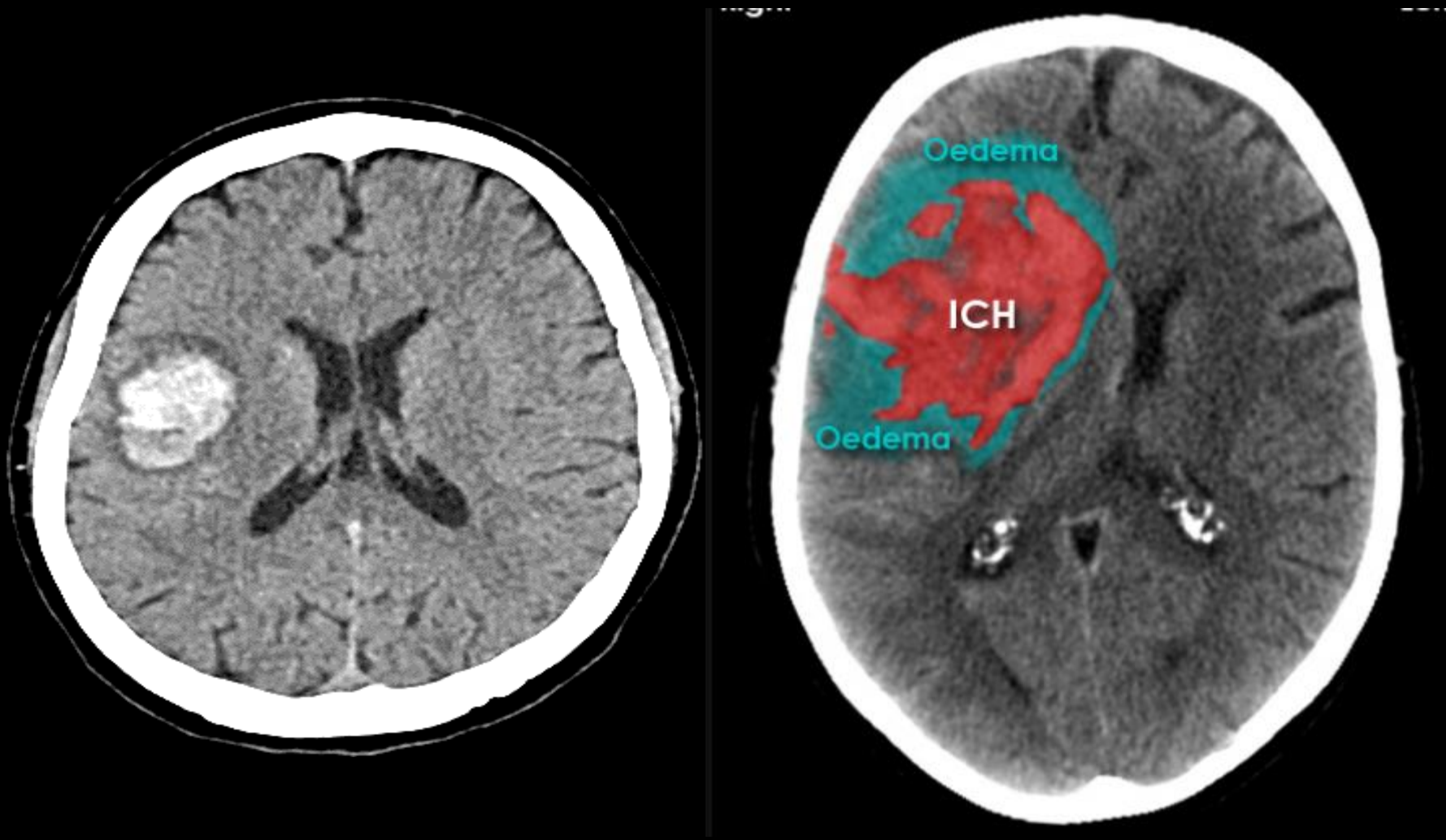
Mechanism: result from injury to intraparenchymal arteries or veins secondary to rotational strain or penetrating trauma .

Intraparenchymal hematomas are usually located **deeper** in the brain parenchyma compared with cerebral contusions, but hematomas may also develop from a superficial cortical contusion.

intracerebral hemorrhage



Mayer SA, Rincon F. *Lancet Neurol.* 2005;4:662-672.
Qureshi AI, et al. *N Engl J Med.* 2001;344:1450-1460.
Terayama Y, et al. Stroke. 1997;28:1185-1188.



acute putamenal haematoma


Se: 1 INTRACEREBRAL AND INTRAVENTICULAR HEMMORAGE
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SID_II_00000014
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6/10/2016 1:01:01 AM


JUL-2007
36:56.98
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SID_II_00000014
173.5
WL: 3036 WW: 1721 [D]
W
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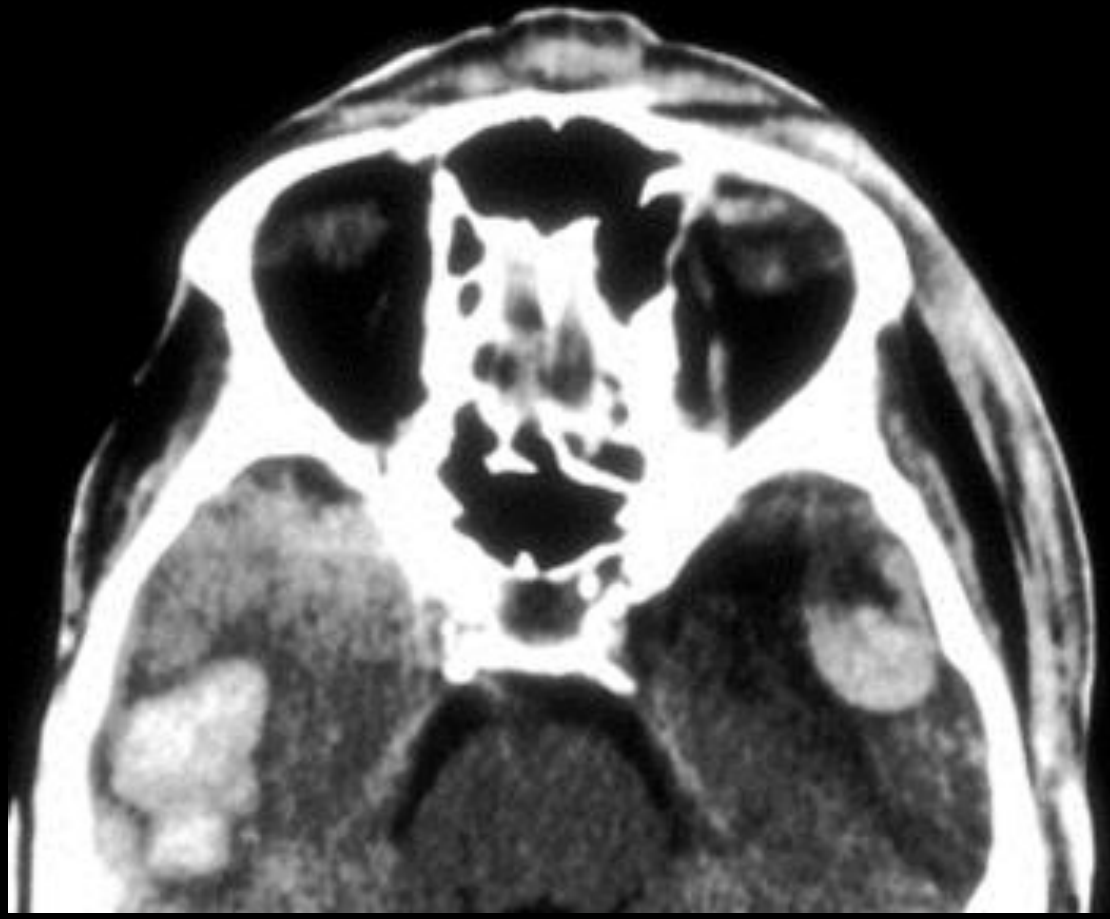
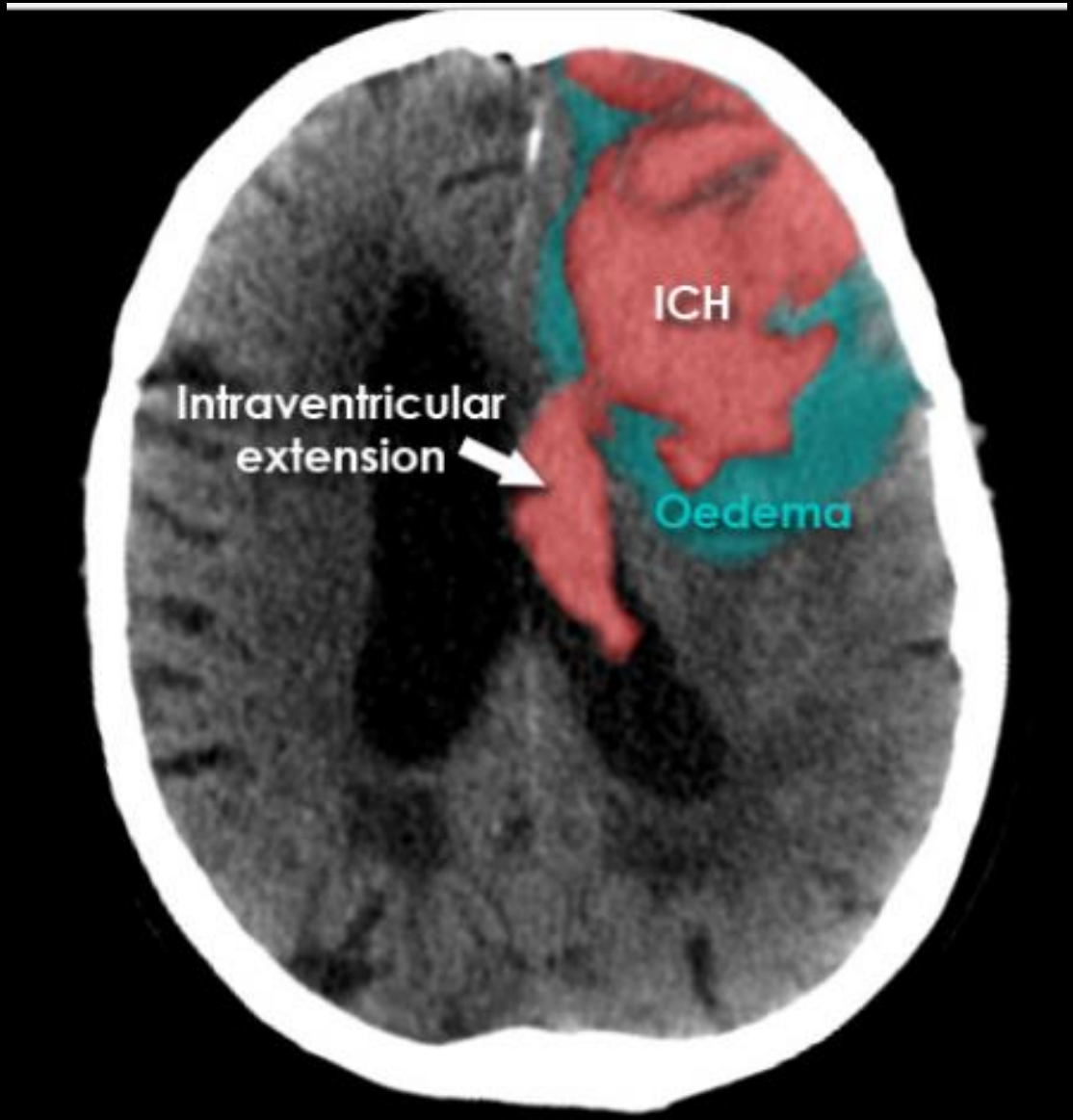
6/10/2016 1:01:01 AM




ICH
Intraventricular extension
Oedema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SID_II_00000014
WL: 3121 WW: 2005 [D]
6/10/2016 1:01:01 AM


Se: 1INTRACEREBRAL AND INTRAVENTICULAR HEMMORAGE
M
SID_II_00000014
WL: 3121 WW: 2005 [D]
6/10/2016 1:01:01 AM


Se: 1INTRACEREBRAL AND INTRAVENTICULAR HEMMORAGE
M
SID_II_00000014
WL: 3121 WW: 2005 [D]
6/10/2016 1:01:01 AM


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218003775!CT00
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R
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Tilt: 0
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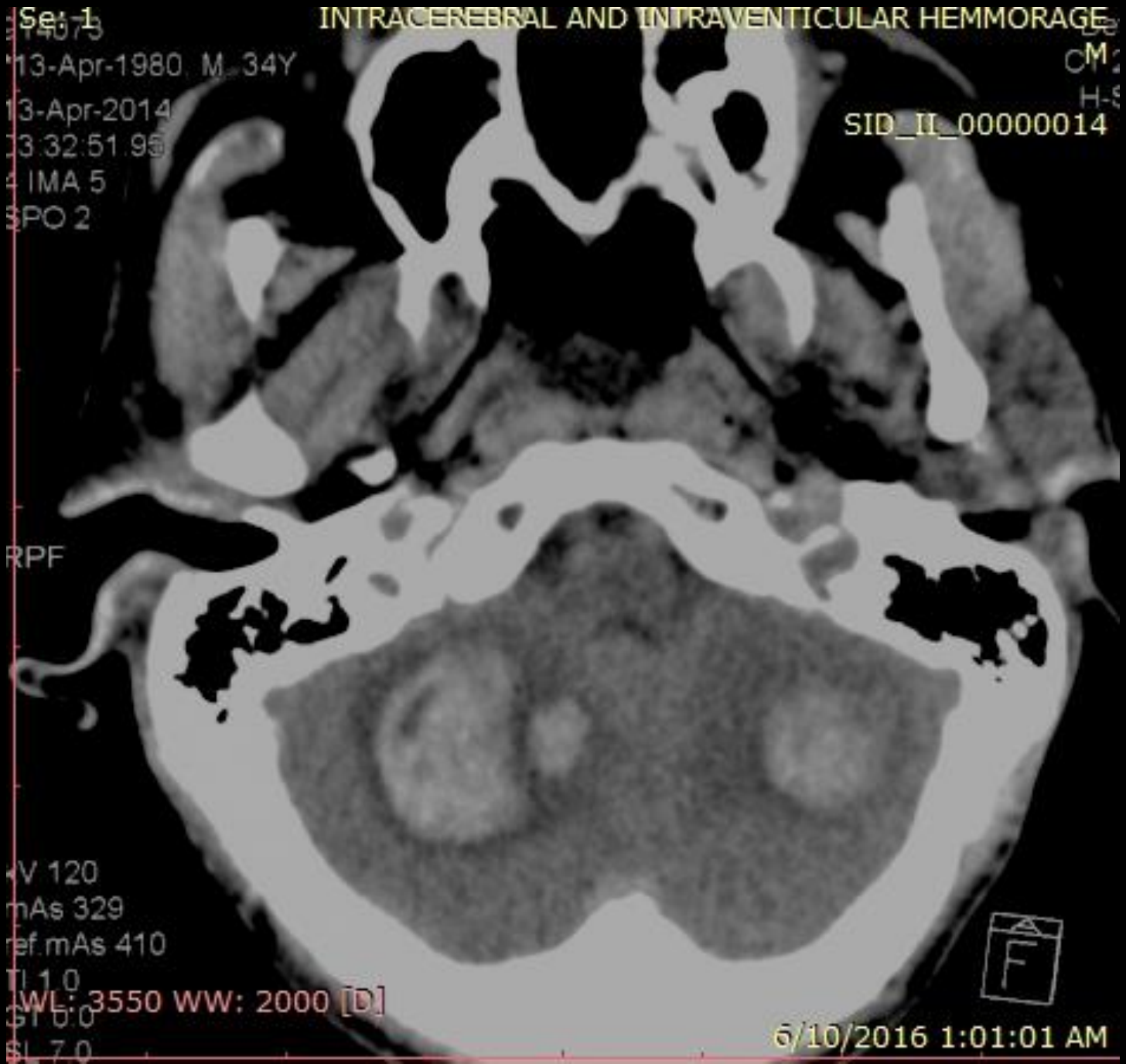
KVp: 130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13-Apr-2014
SID_II_00000014
IMA 5
PO 2
RPF
V 120
mAs 329
ef.mAs 410
WL: 3550 WW: 2000 [D]
6/10/2016 1:01:01 AM


Im: INTRACEREBRAL INTRACEREBRAL AND INTRAVENTICULAR HEMMORAGE
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INTRACEREBRAL AND INTRAVENTICULAR HEMMORAGE
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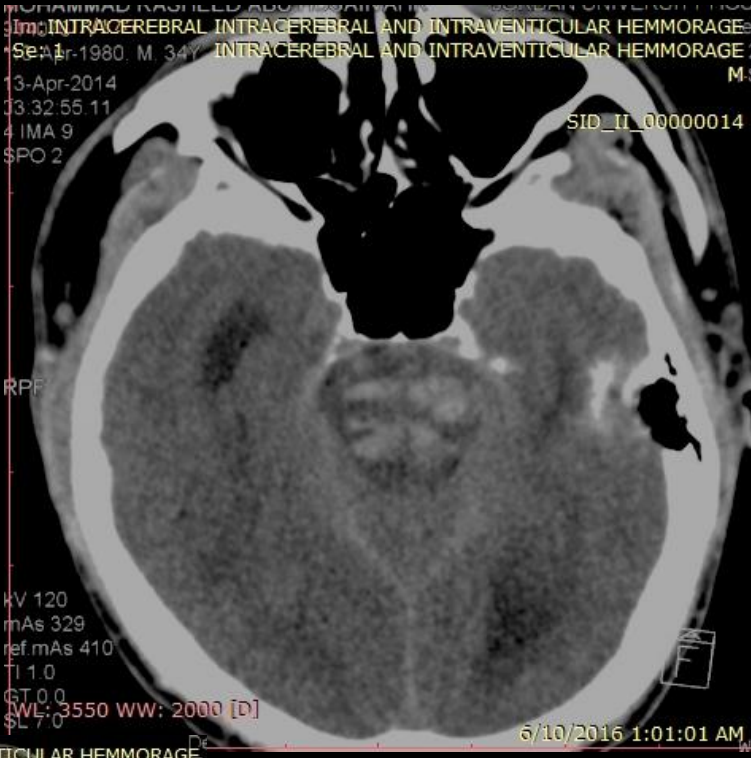
6/10/2016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Se: 1
INTRACEREBRAL AND INTRAVENTICULAR HEMMORAGE
M
SID_II_00000014
RPF
kV 120
mAs 329
ref.mAs 410
WL: 3550 WW: 2000 [D]
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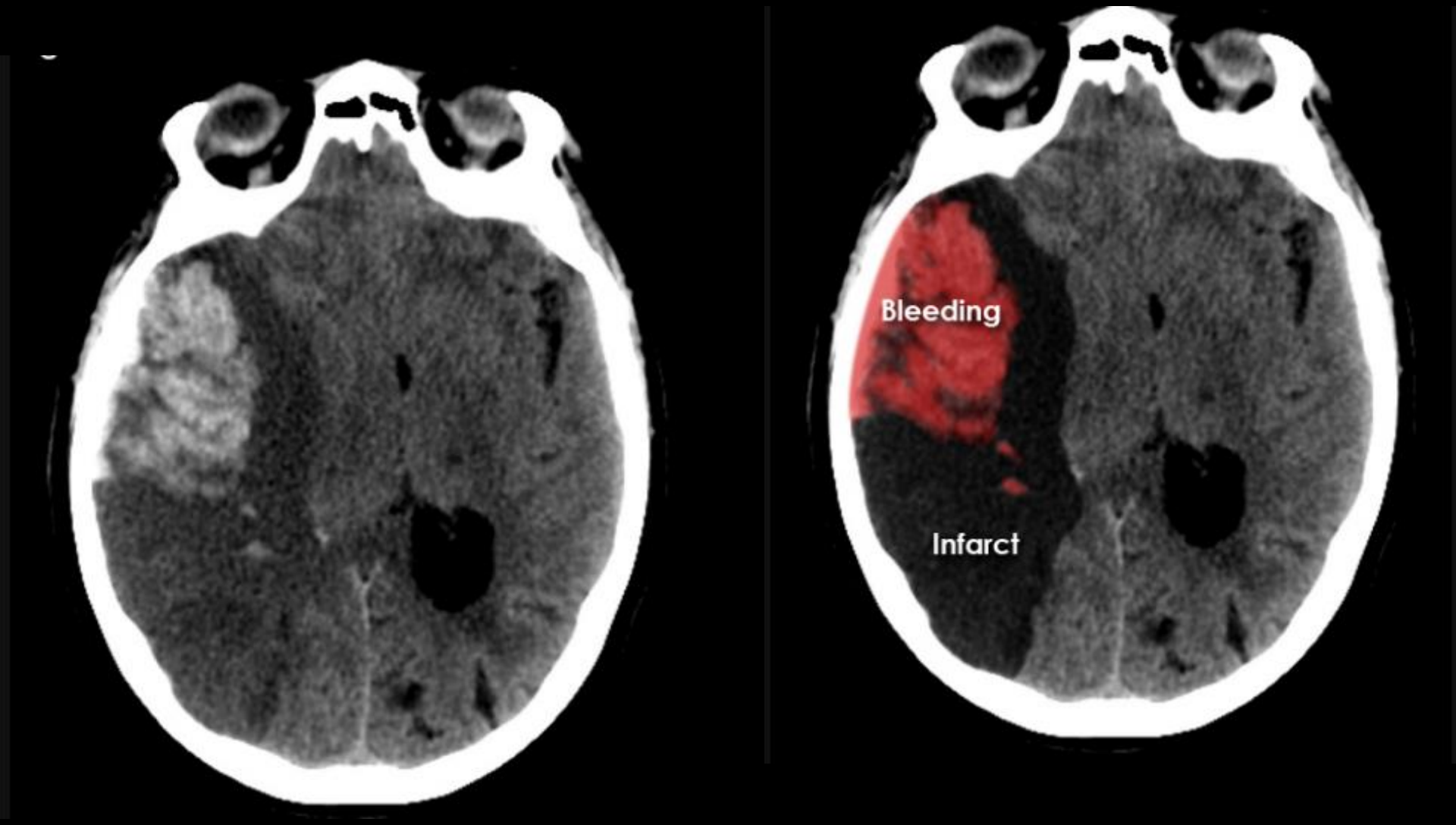
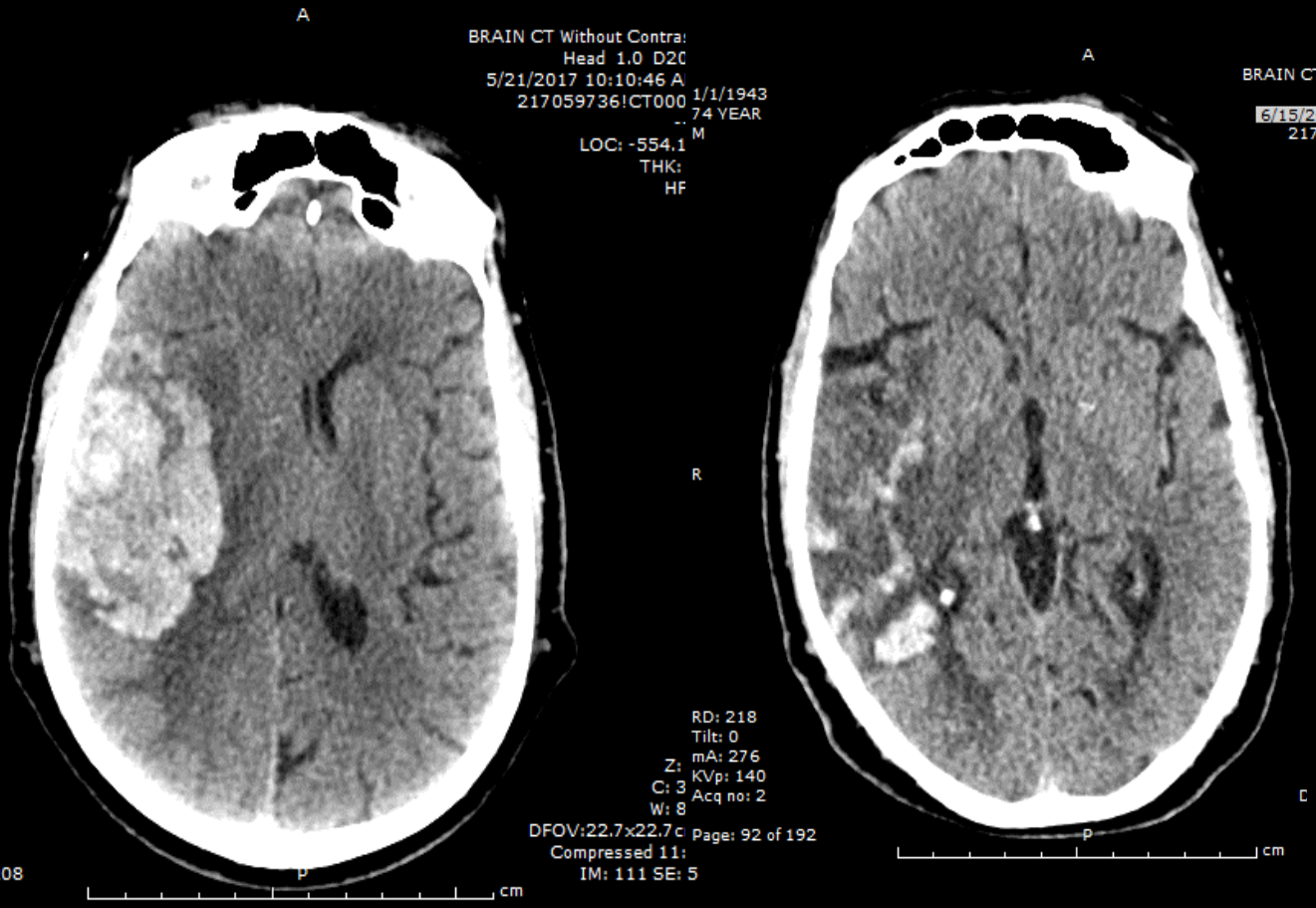
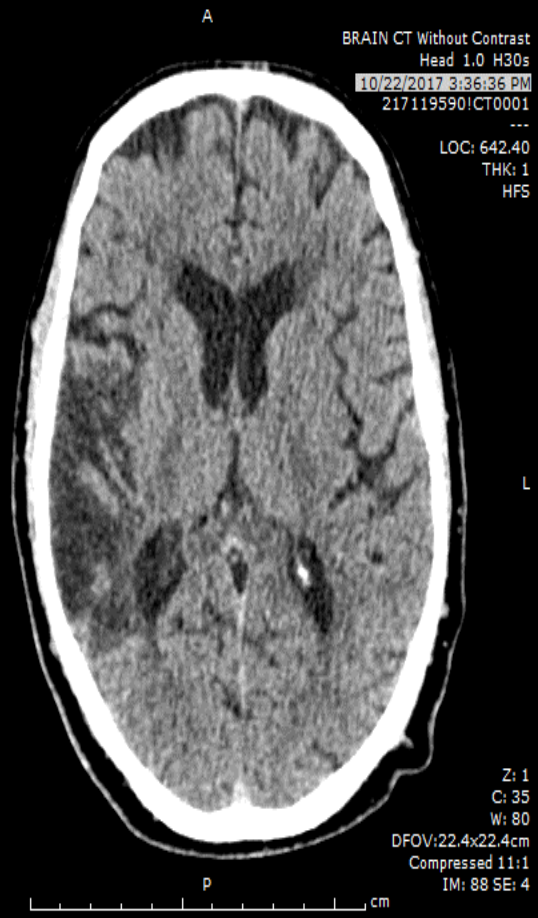
Im: INTRACEREBRAL INTRACEREBRAL AND INTRAVENTICULAR HEMMORAGE
Se: 1
INTRACEREBRAL AND INTRAVENTICULAR HEMMORAGE
M
SID_II_00000014
RPF
kV 120
mAs 329
ref.mAs 410
WL: 3550 WW: 2000 [D]
6/10/2016 1:01:01 AM

Infarct with haemorrhagic transformation

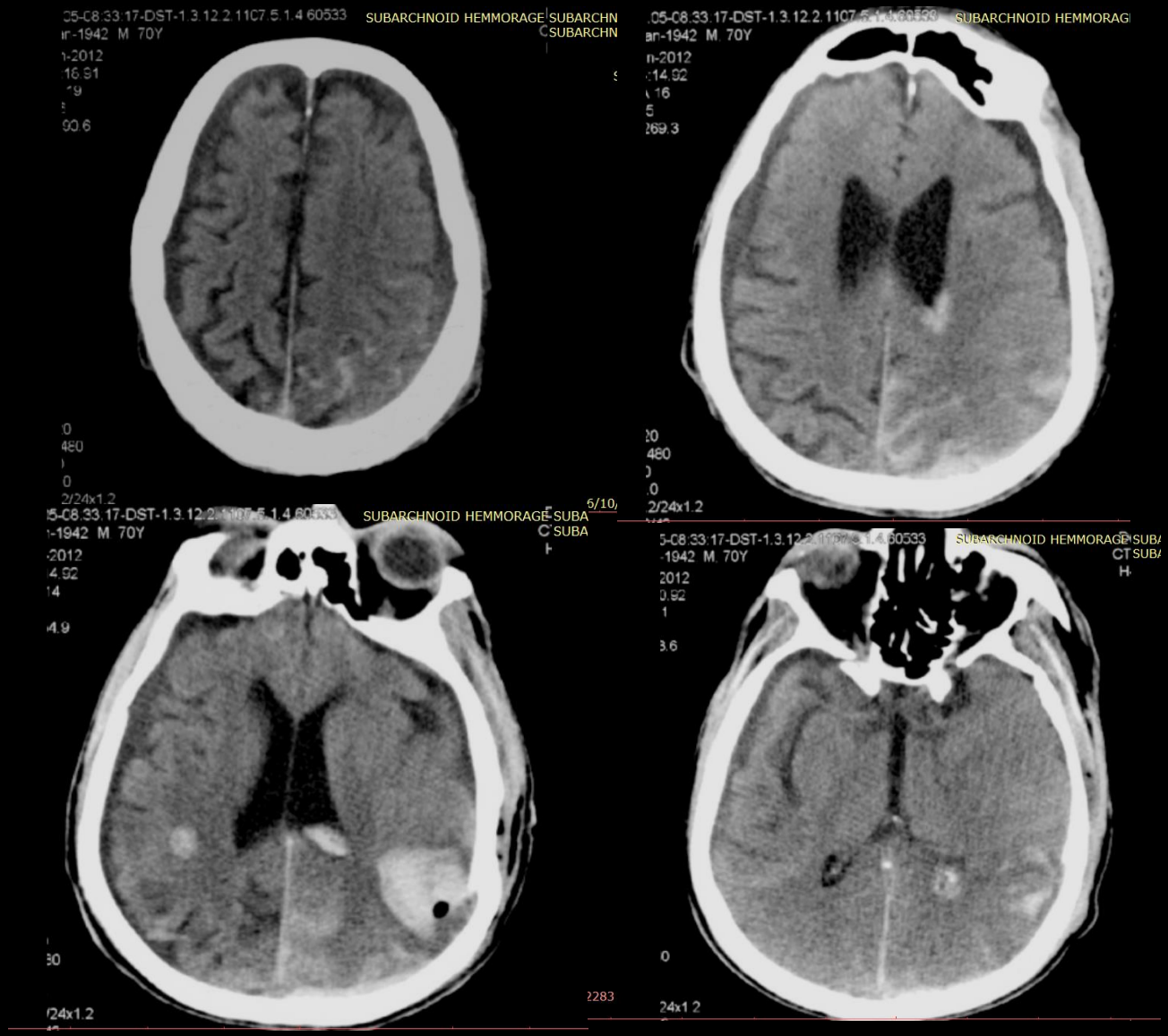




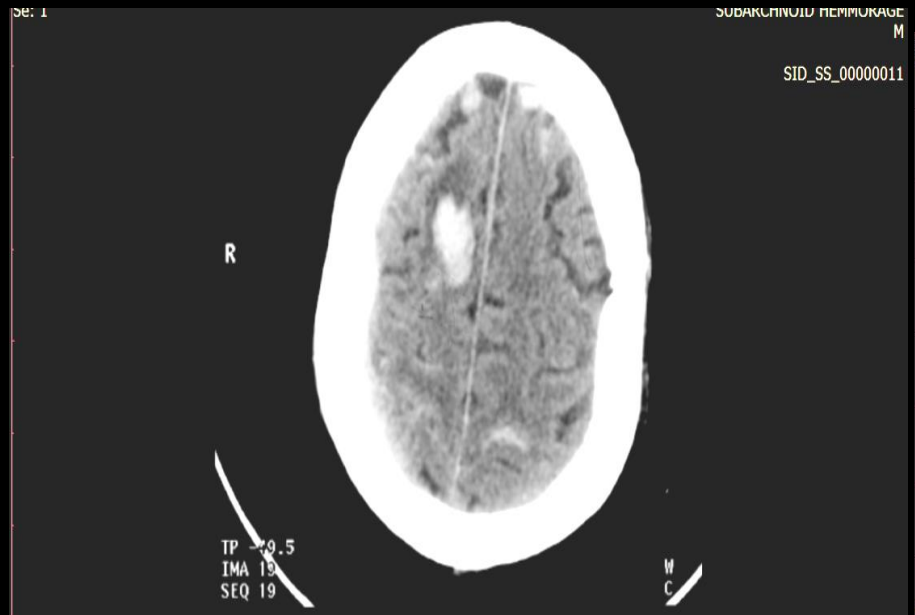


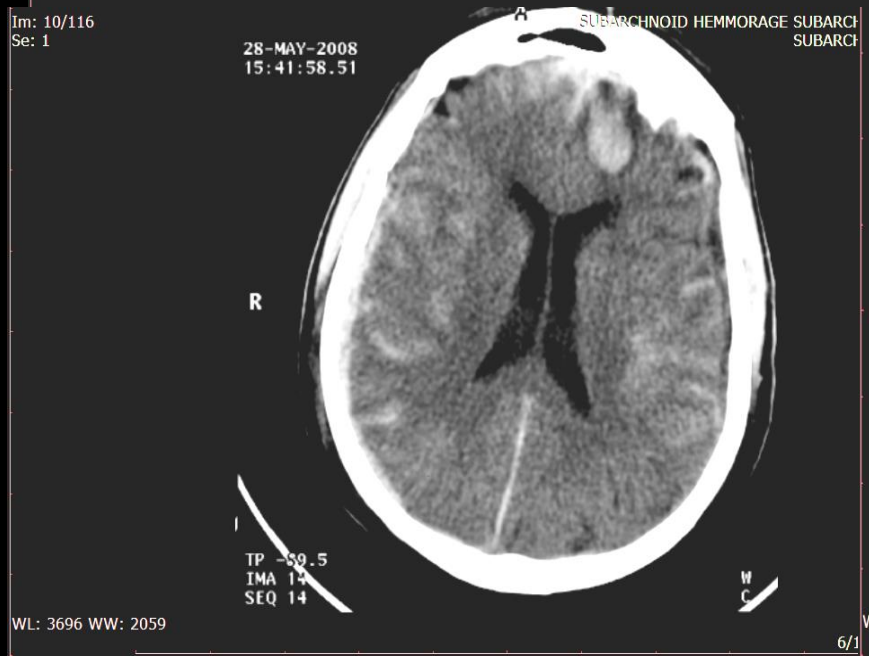
Degradation of blood products with time

Different types of hemorrhage in the same patient



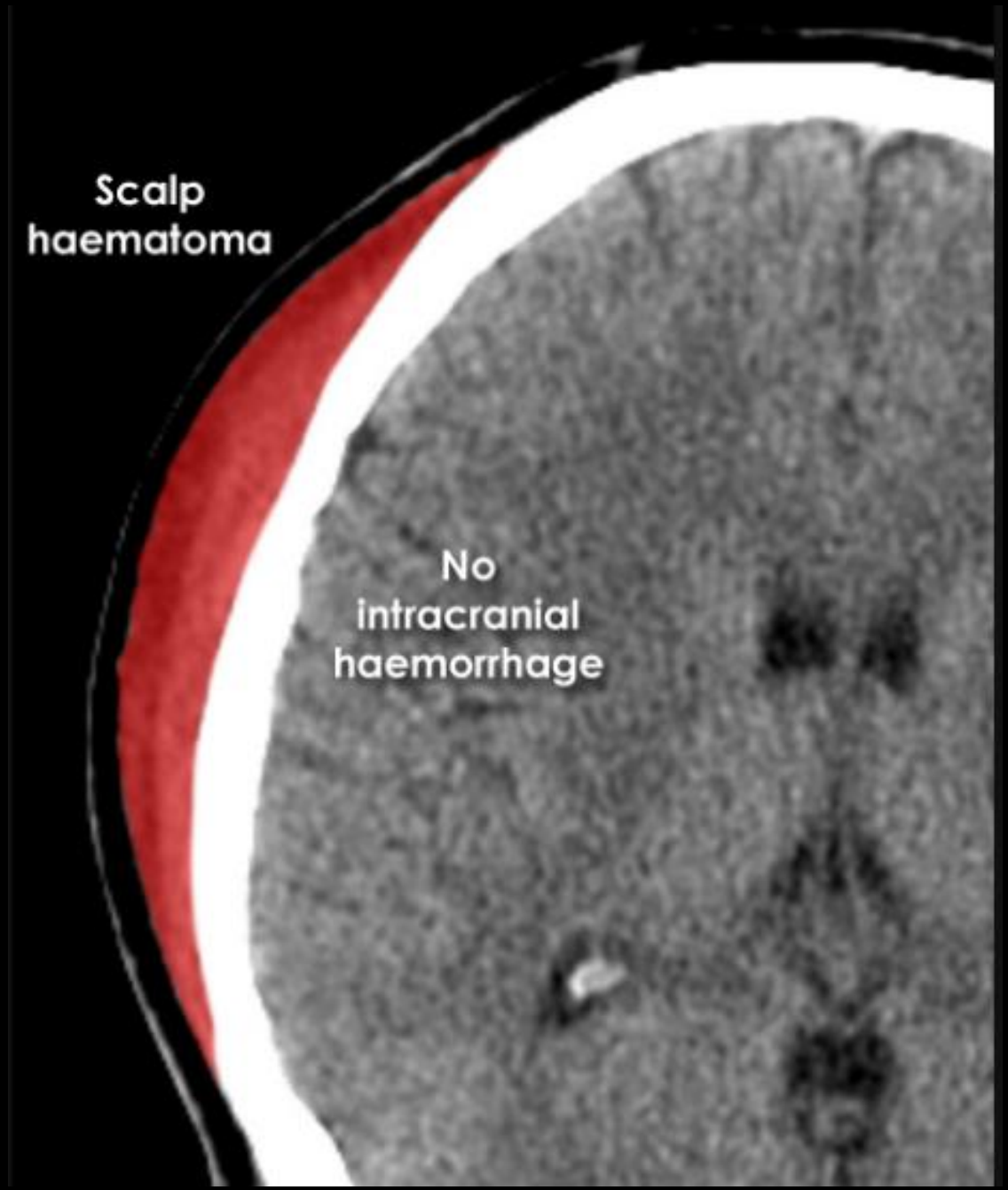
Another example



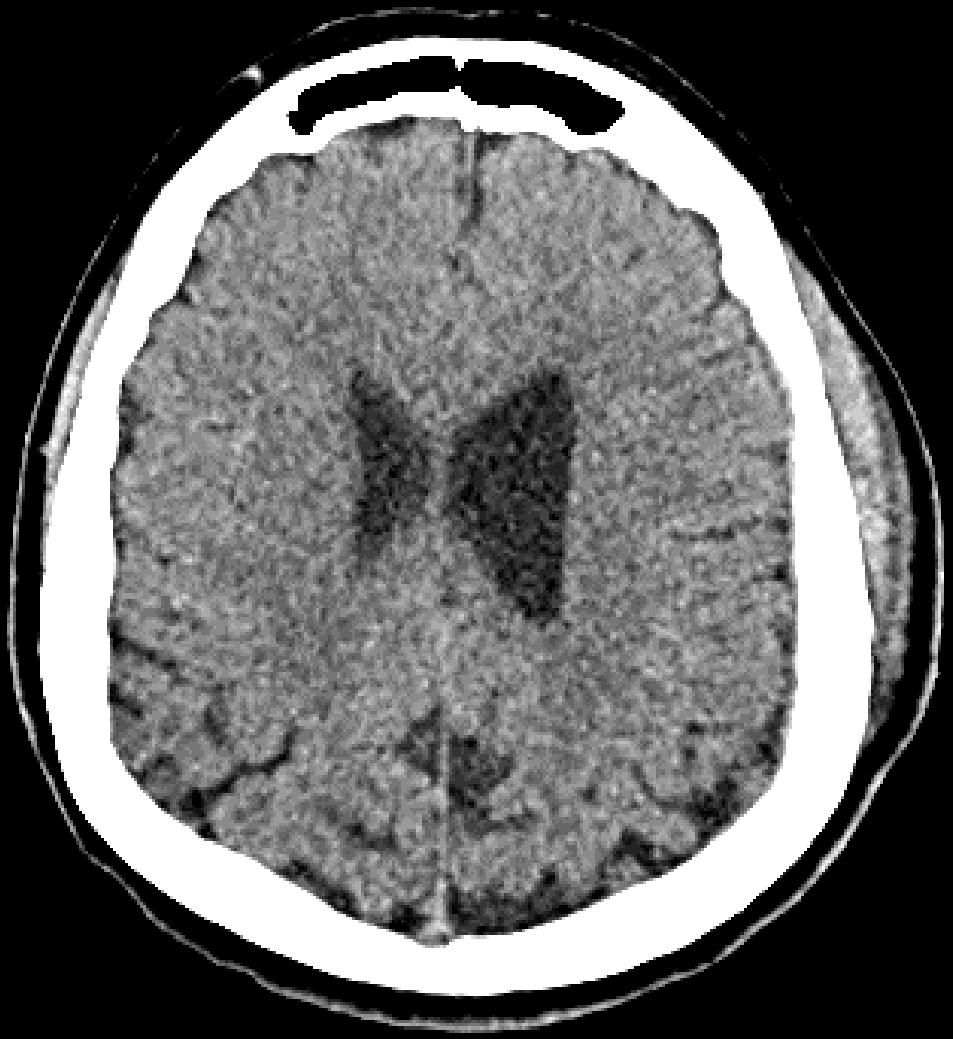


Cephalohematoma

Traumatic subperiosteal haematomas of the skull that are usually caused by birth injury. They are bound between the periosteum and cranium, and therefore cannot cross sutures. Being bound by a suture line distinguishes them from subgaleal haematoma, which can cross sutures.


Scalp
haematoma
No
intracranial
haemorrhage

Cephalo-hematoma

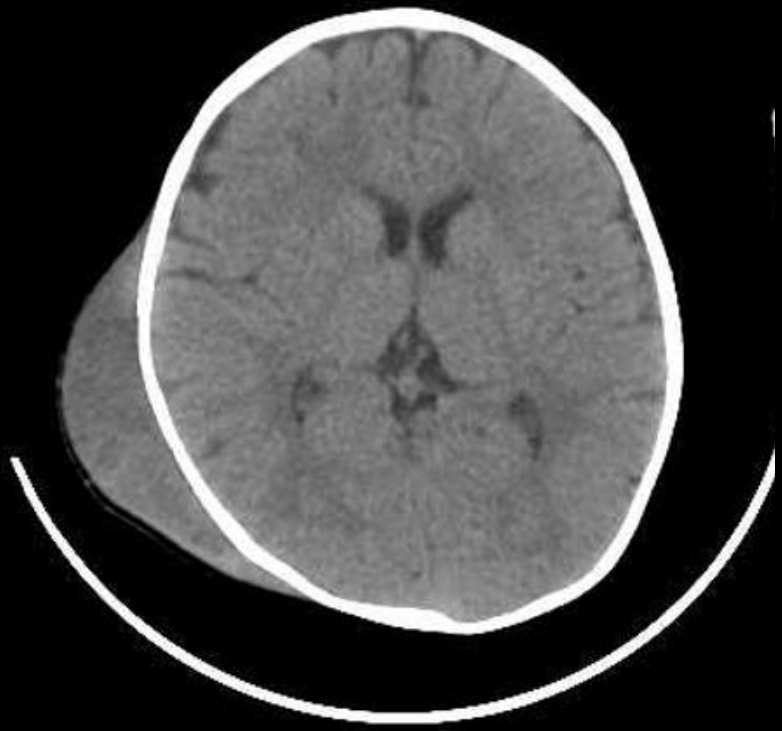


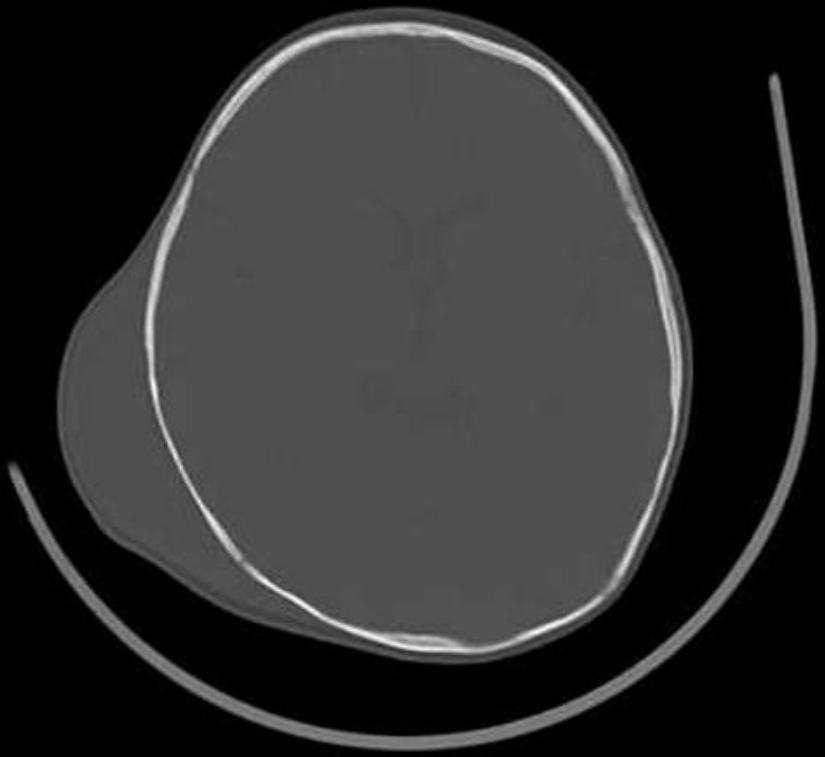
Subgaleal hematoma

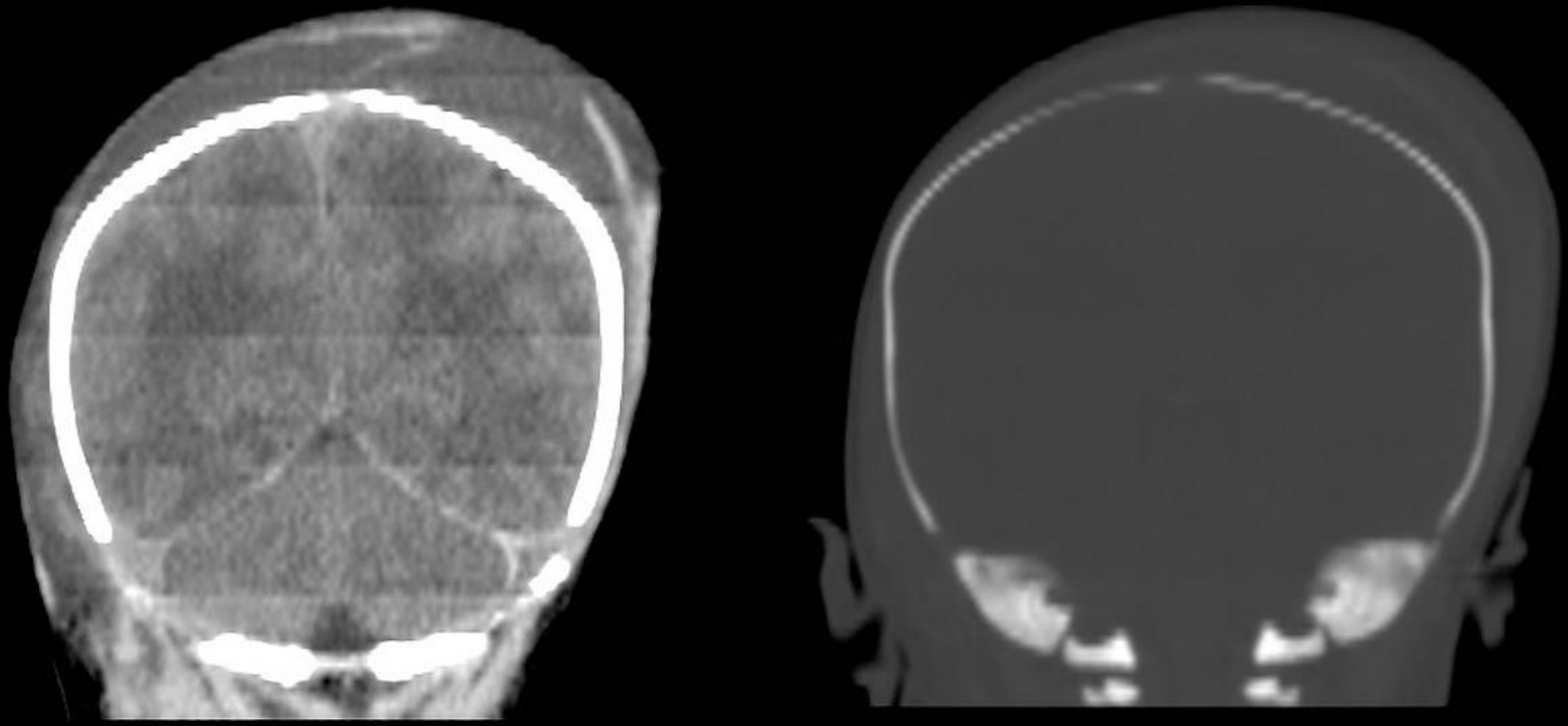
Describes scalp bleeding in the potential space between the periosteum and the galeal aponeurosis. It is a rare but possibly lethal emergency.

Bleeding occurs as a result of rupture to emissary veins which drain the scalp veins into the dural sinuses

Due to being superficial to the periosteum, subgaleal haematomas are able to cross suture lines and surround the entire skull.







Calcification

- Normal variation
- Pathological :

1- AVM

2- Infection (congenital in pediatrics)

3- Tumors

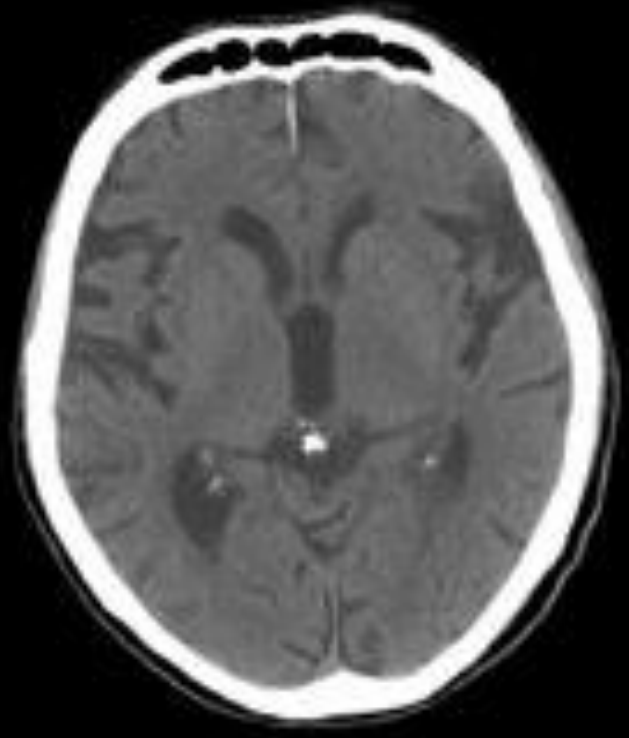
4 – metabolic

Calcification

1- Normal variation/ normal aging

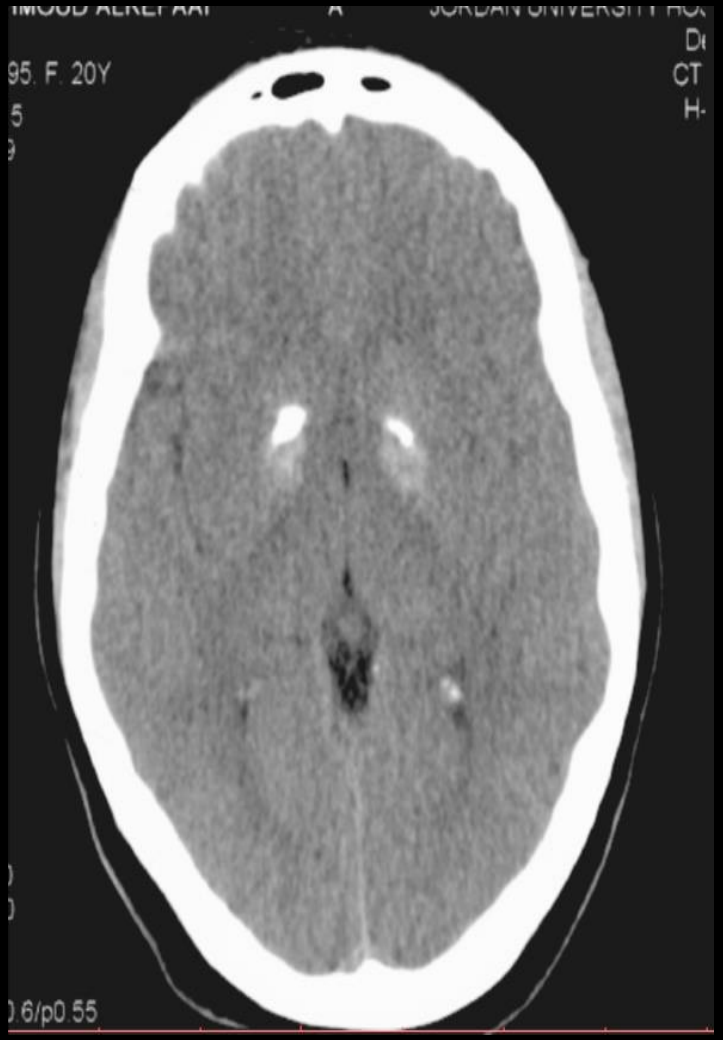
basal ganglia , vascular calcifications ,Choroid plexus , Pineal gland , dentate nuclues , calcified falx .

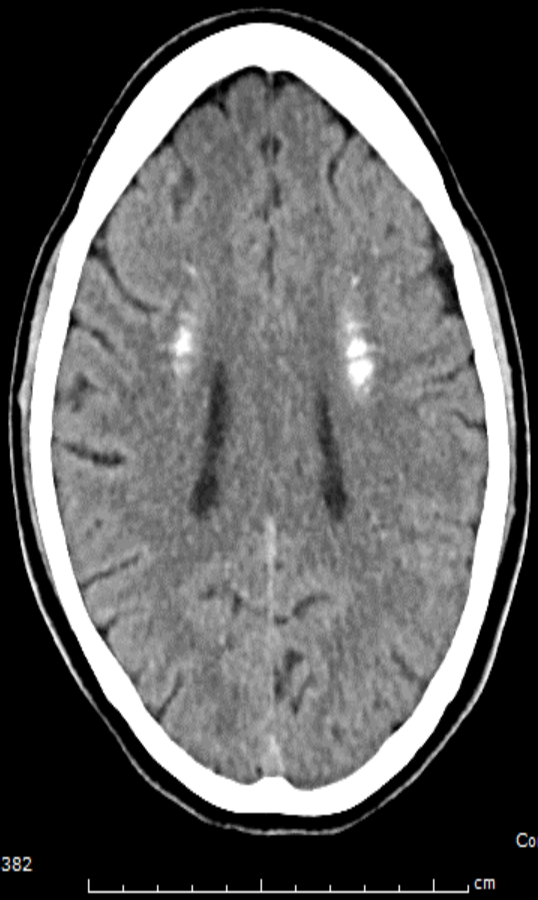
Choroid plexus and Pineal gland



Basal ganglia

Basal ganglia

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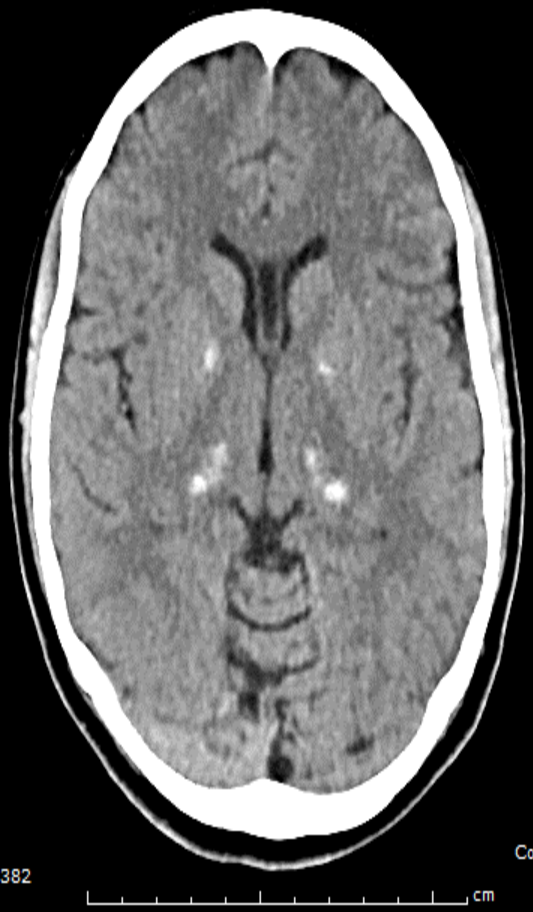
Compressed 11:1
IM: 65 SE: 3
cm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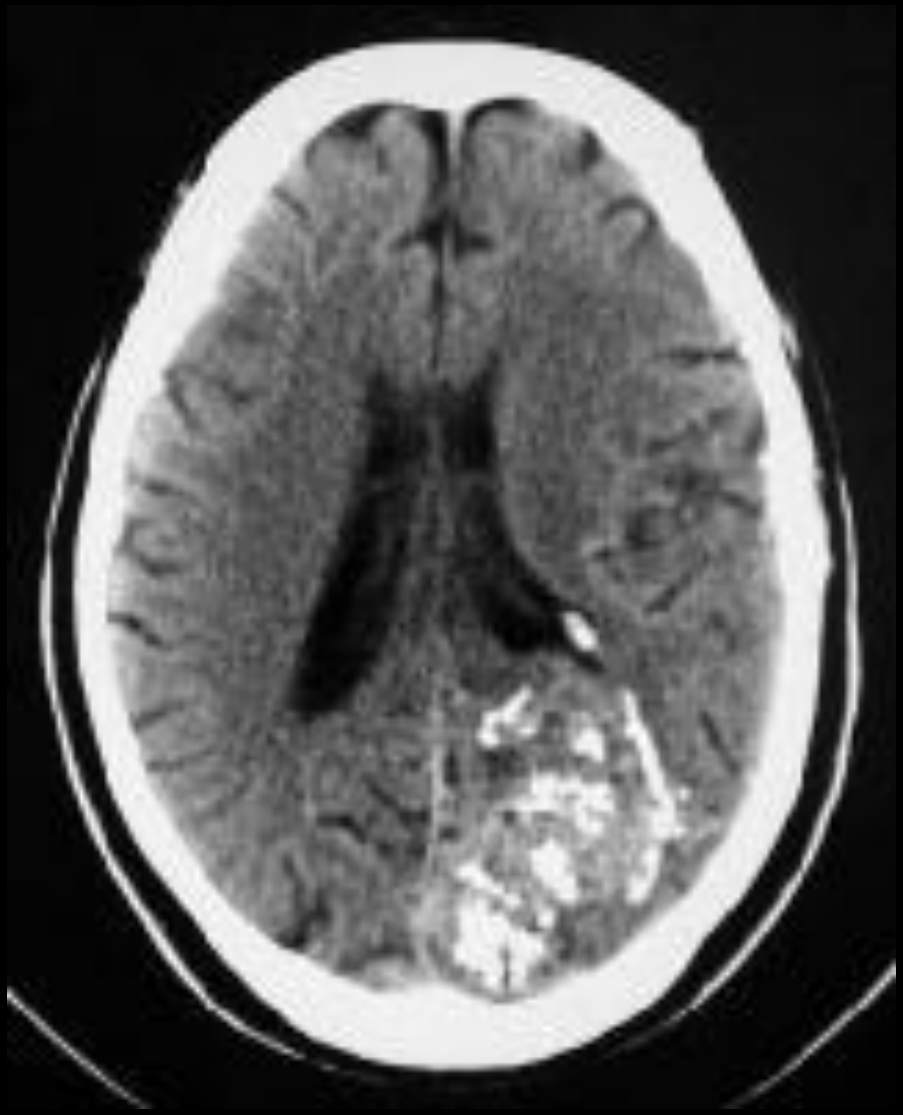
Compressed 11:1
IM: 91 SE: 3
cm

Dentate nucleus

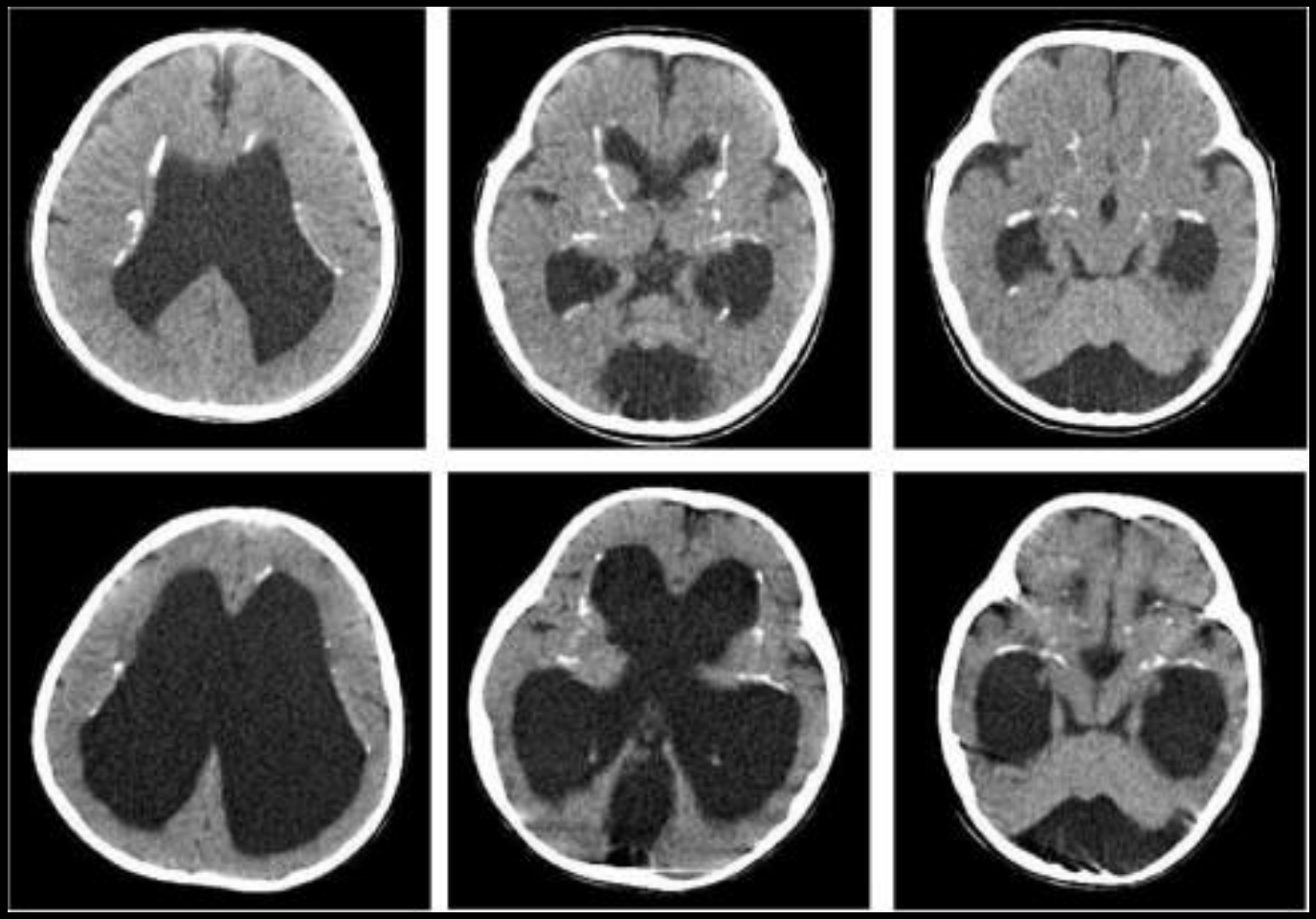
vascular calcification

Pathological Calcification

AVM



Infection (congenital) : TORCH




Im: 49/180
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WL: 3550 WW: 2000 [D]
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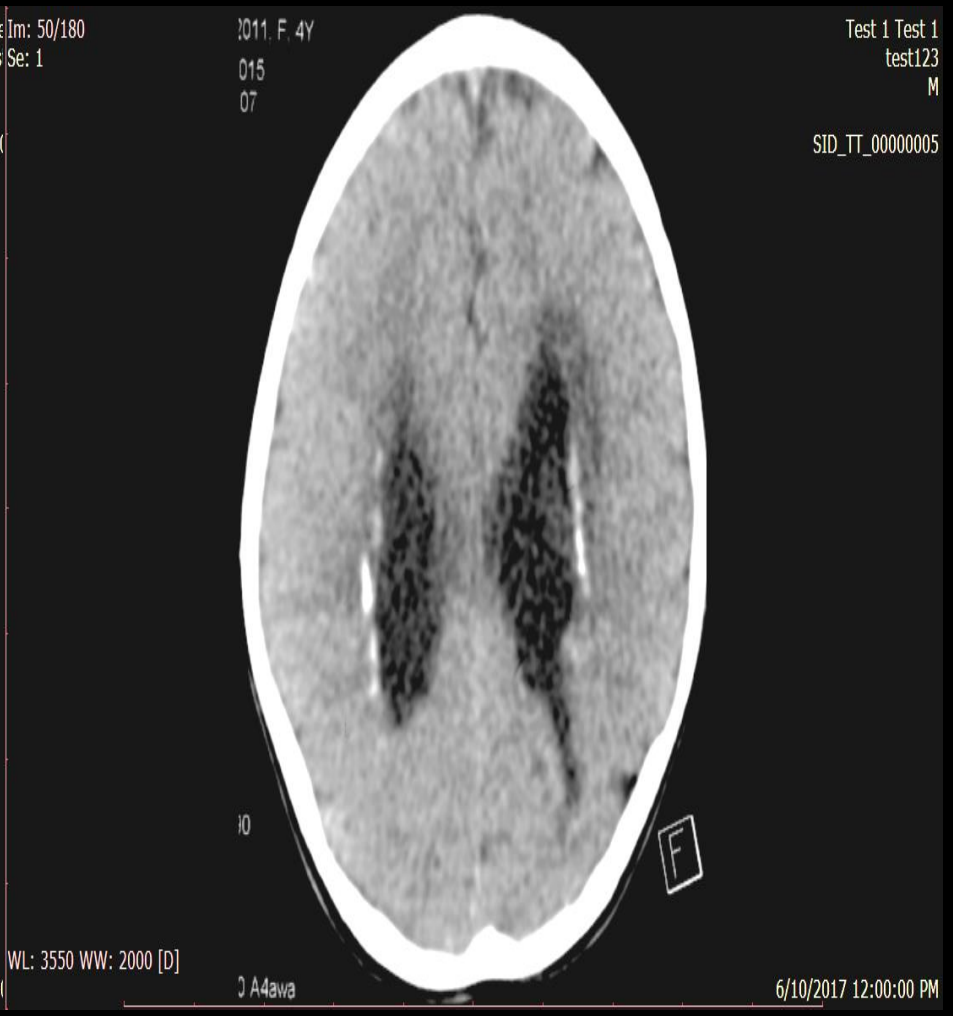
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Test 1 Test 1
test123
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WL: 3550 WW: 2000 [D]
6/10/2017 12:00:00 PM

metabolic

Sturge weber

Tumor

Tumors that usually calcifies :

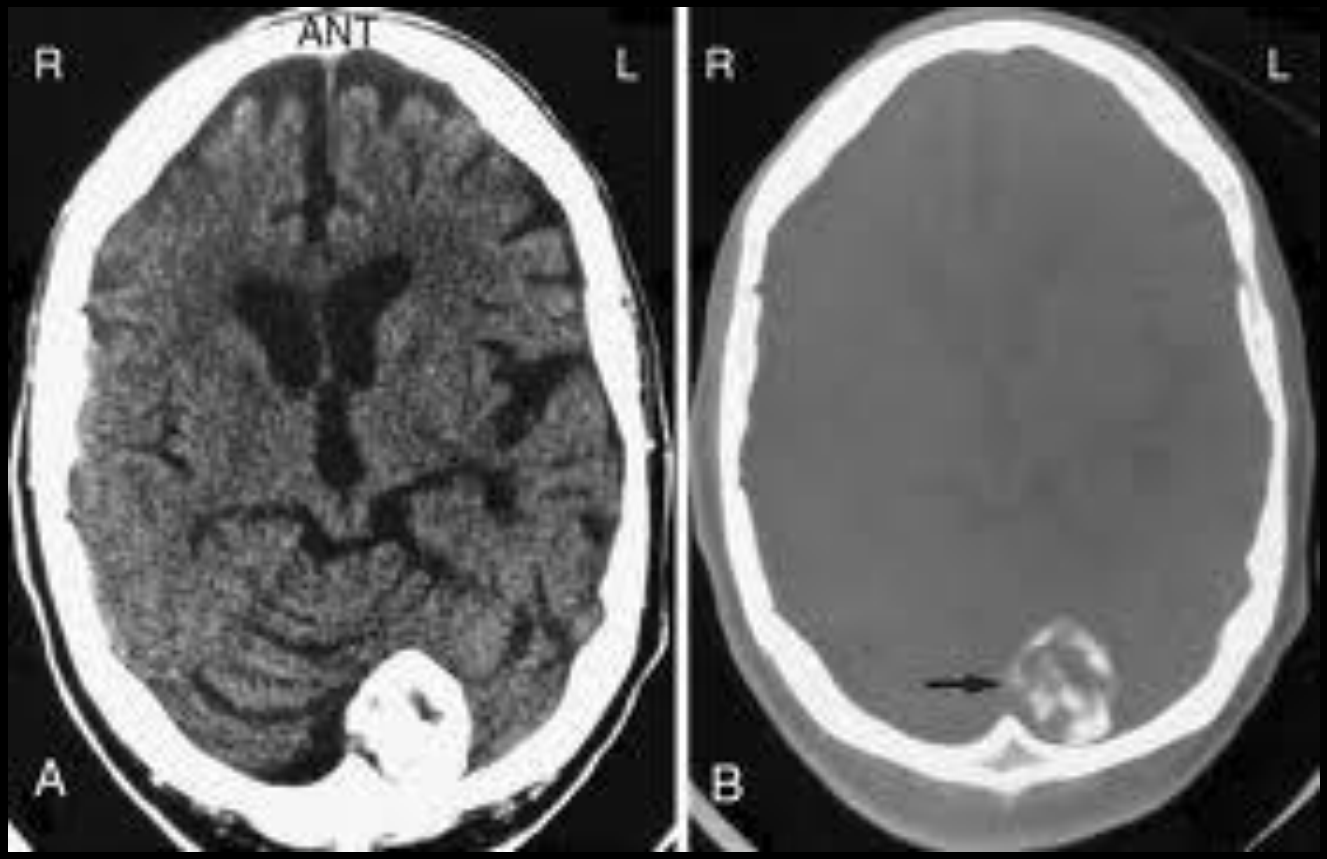
Meningioma

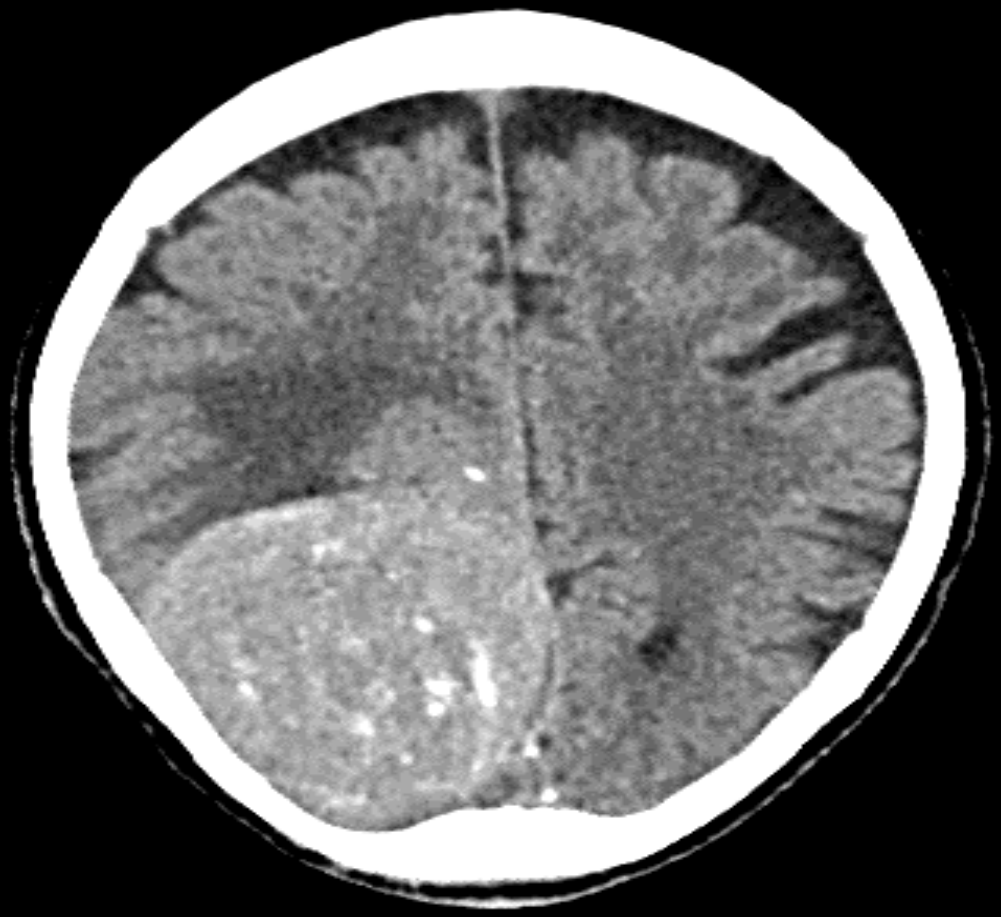
Craniopharangioma

Low grade astrocytoma

Oligodendroglioma

Meningioma




219
P

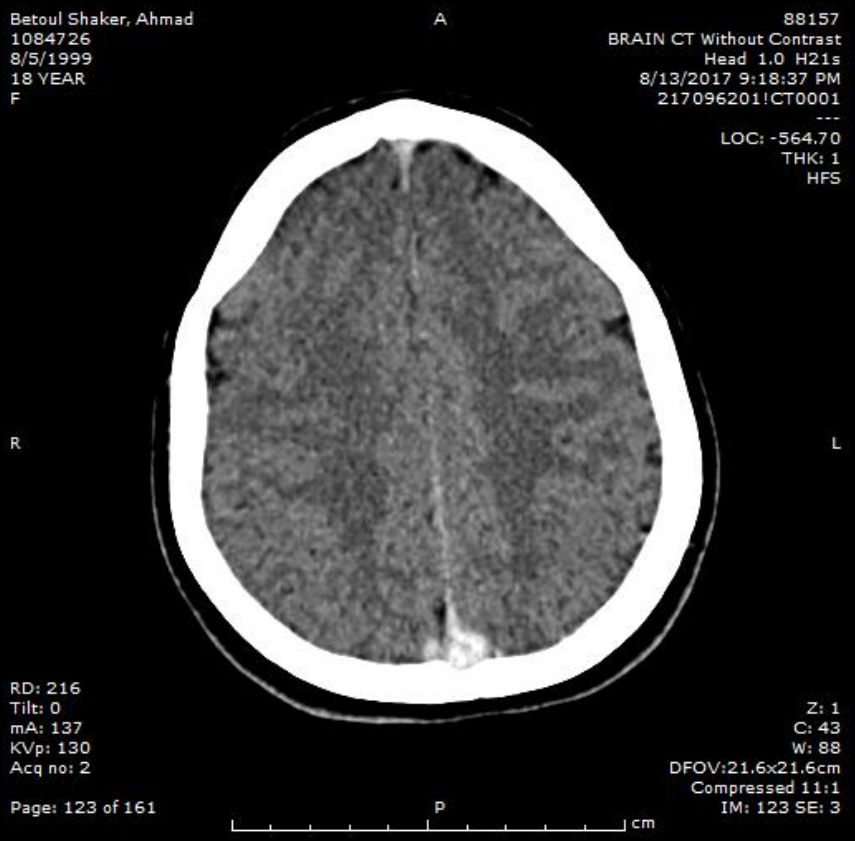
Craniopharangioma

Low grade Astrocytoma

Oligodendroglioma

Thrombosed cerebral venous sinuses

Thrombosed cerebral venous sinuses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1084726
8/5/1999
18 YEAR
F
A
88157
BRAIN CT Without Contrast
Head 1.0 H21s
8/13/2017 9:18:37 PM
217096201!CT0001

LOC: -564.70
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HFS
R
L
RD: 216
Tilt: 0
mA: 137
KVp: 130
Acq no: 2
Page: 123 of 161
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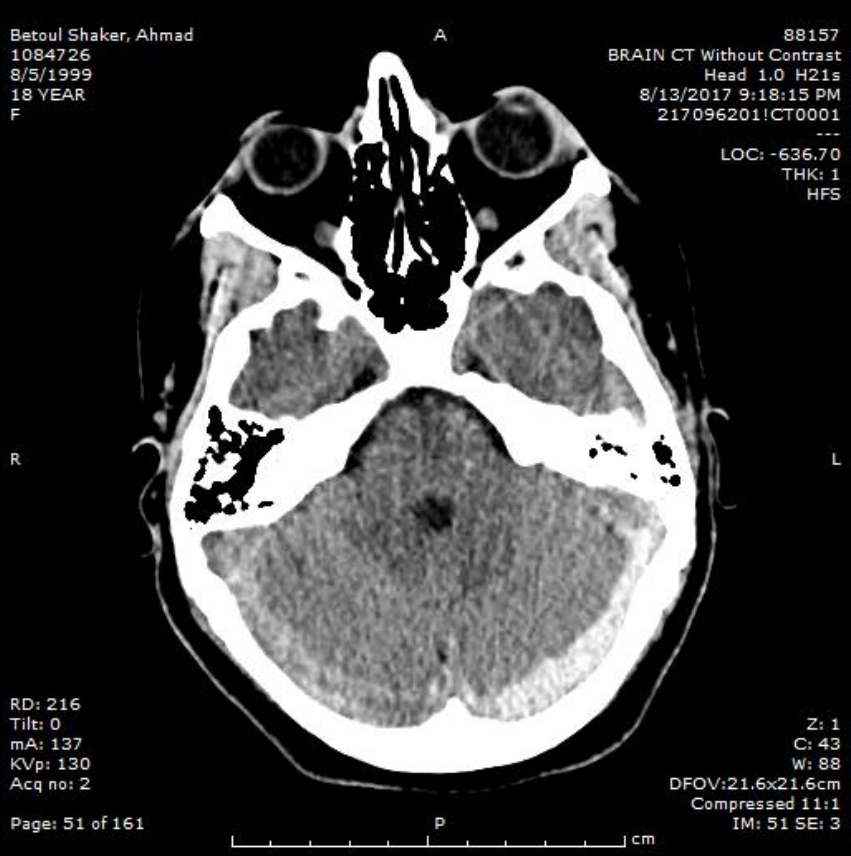
C: 43
W: 88
DFOV:21.6x21.6cm
Compressed 11:1
IM: 123 SE: 3
P
cm


Betoul Shaker, Ahmad
1084726
8/5/1999
18 YEAR
F
A
88157
BRAIN CT Without Contrast
Head 1.0 H21s
8/13/2017 9:18:15 PM
217096201!CT0001

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HFS
R
L
RD: 216
Tilt: 0
mA: 137
KVp: 130
Acq no: 2
Page: 51 of 161
Z: 1
C: 43
W: 88
DFOV:21.6x21.6cm
Compressed 11:1
IM: 51 SE: 3
P
cm

Fetal HB


Se: 1
A
FETAL HB
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5-NOV-2009
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WL: 2850 WW: 1909 [D]
P -130.5
10
6/10/201


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6/10/2017 1:01:01 AM
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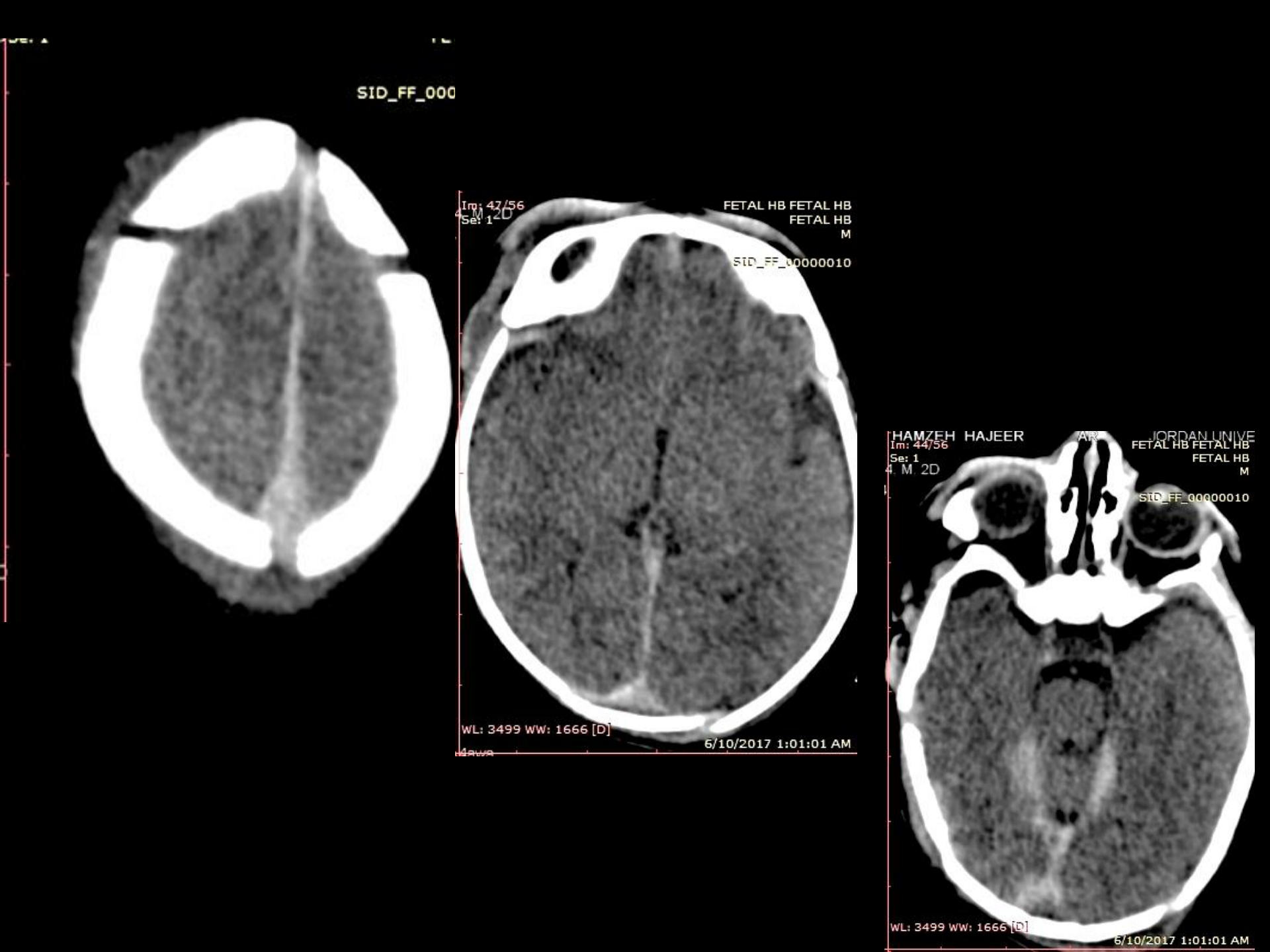
17
6/10/2017 1:01:01 AM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Se: 1
4 M 2D
FETAL HB FETAL HB
FETAL HB
M
SID_FF_00000010
WL: 3499 WW: 1666 [D]
6/10/2017 1:01:01 AM
HAMZEH HAJEER
Im: 44/56
Se: 1
4 M 2D
JORDAN UNIVE
FETAL HB FETAL HB
FETAL HB
M
SID_FF_00000010
WL: 3499 WW: 1666 [D]
6/10/2017 1:01:01 AM

Contrast enhancing lesions

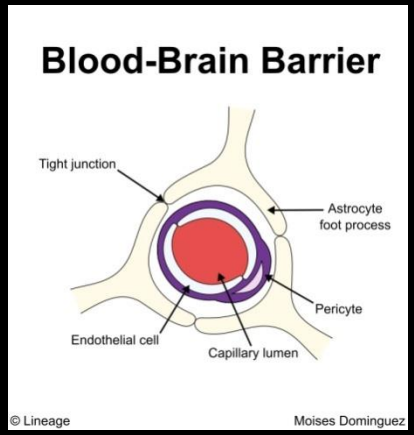
Benign

Malignant

When there is breakage in BBB , there will be enhancement

Structures that normally enhance(no BBB) :

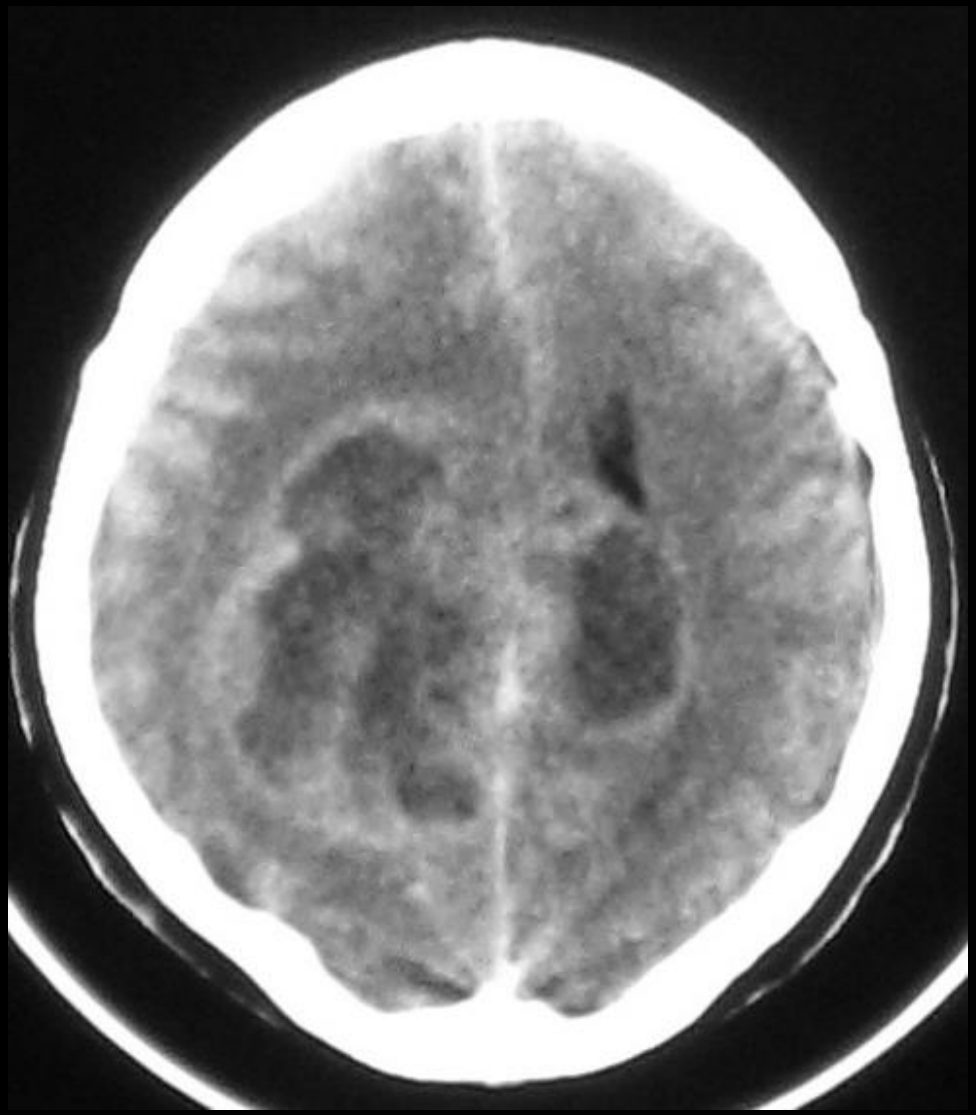
pineal gland , pituitary gland and choroid plexus

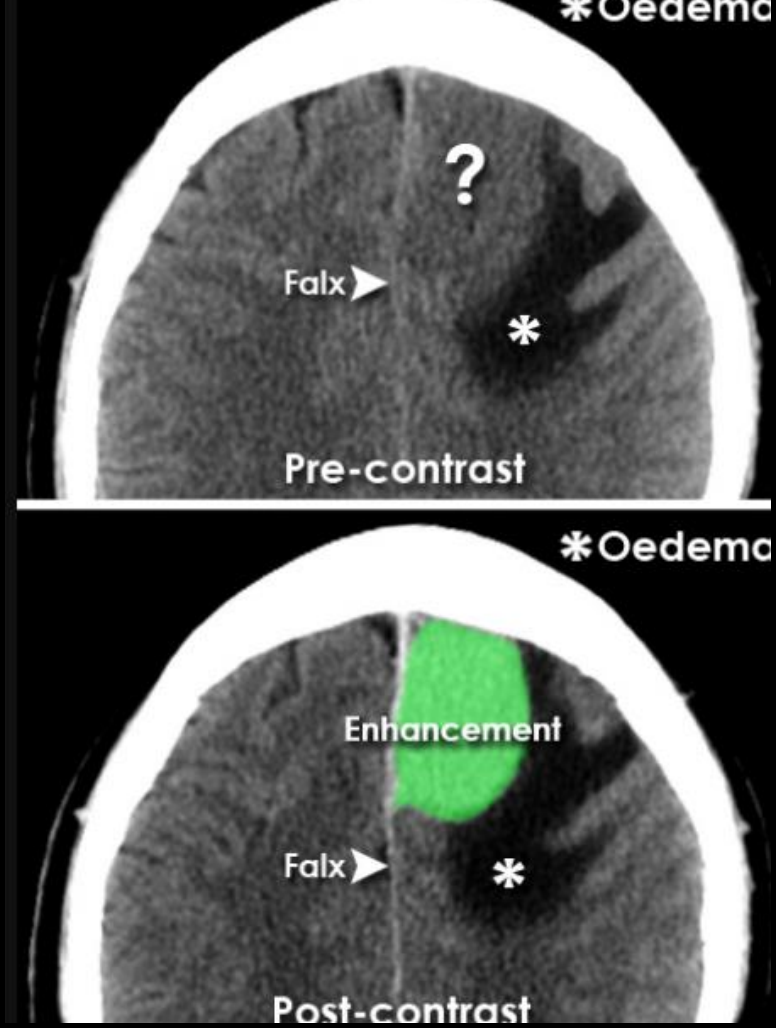


Benign: Meningioma

Benign: Abscess

Malignant : GBM





Before contrast is given this meningioma is barely visible
Post-contrast it enhances brightly and its location next to the meningeal surface (falx) is clearly seen
Cerebral oedema - black area next to the meningioma (**asterisk**) - is a finding often associated with a large meningioma

Hypodense Lesions:

1- Fluid

2- air

3- Fat

Hypodense Lesions:

Fluid:

CSF: normal v/s hydrocephalus

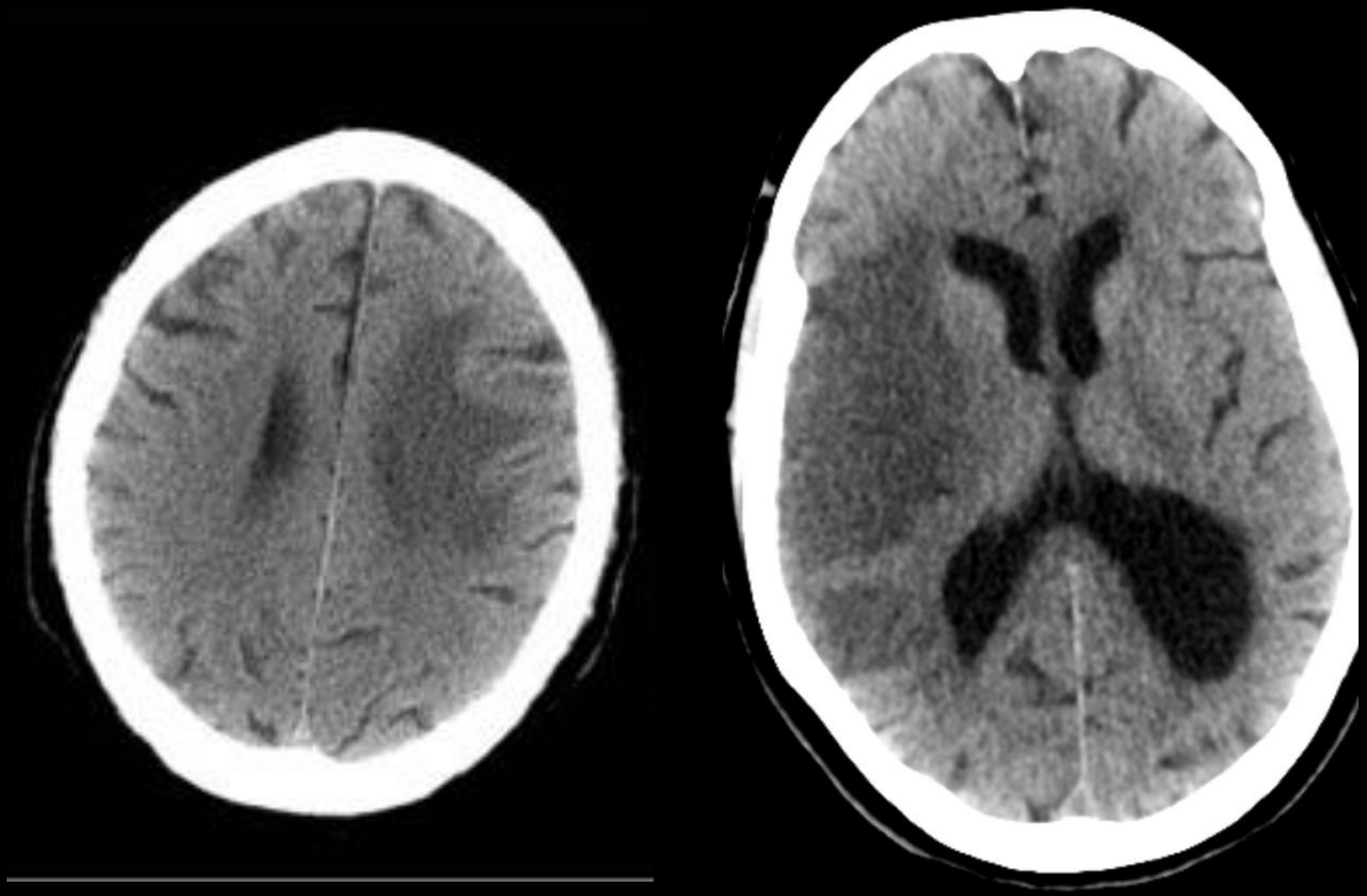
Edema: vasogenic v/s cytotoxic

Diffuse brain edema

Necrotic tissue: tumor v/s abscess

CSF: normal v/s hydrocephalus

Edema: vasogenic v/s cytotoxic
Mass lesion v/s infarction



Vasogenic edema

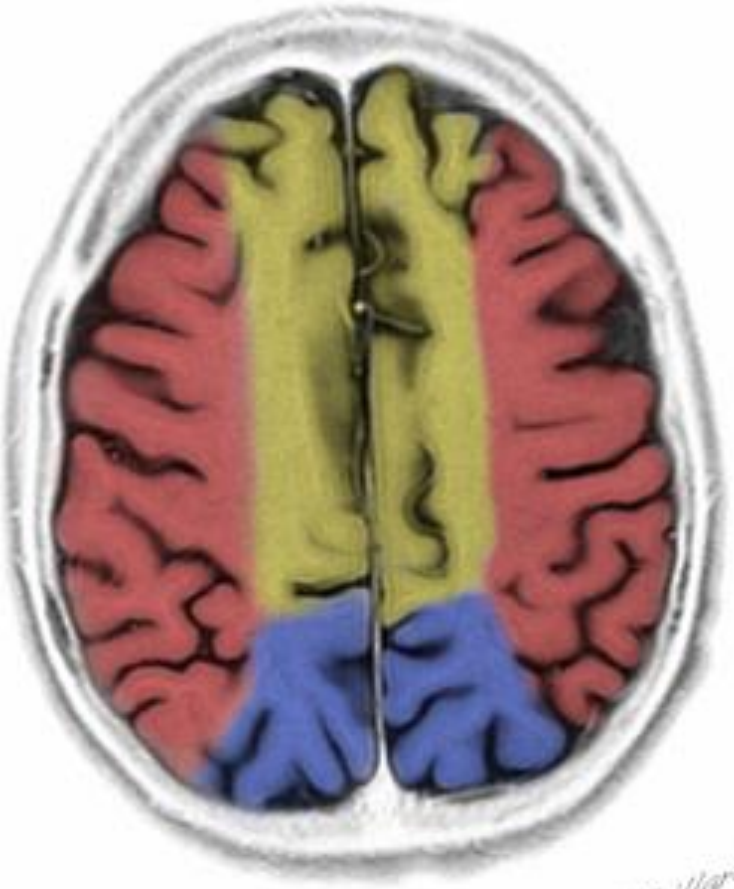
Cytotoxic edema

Cerebral Vascular Territories

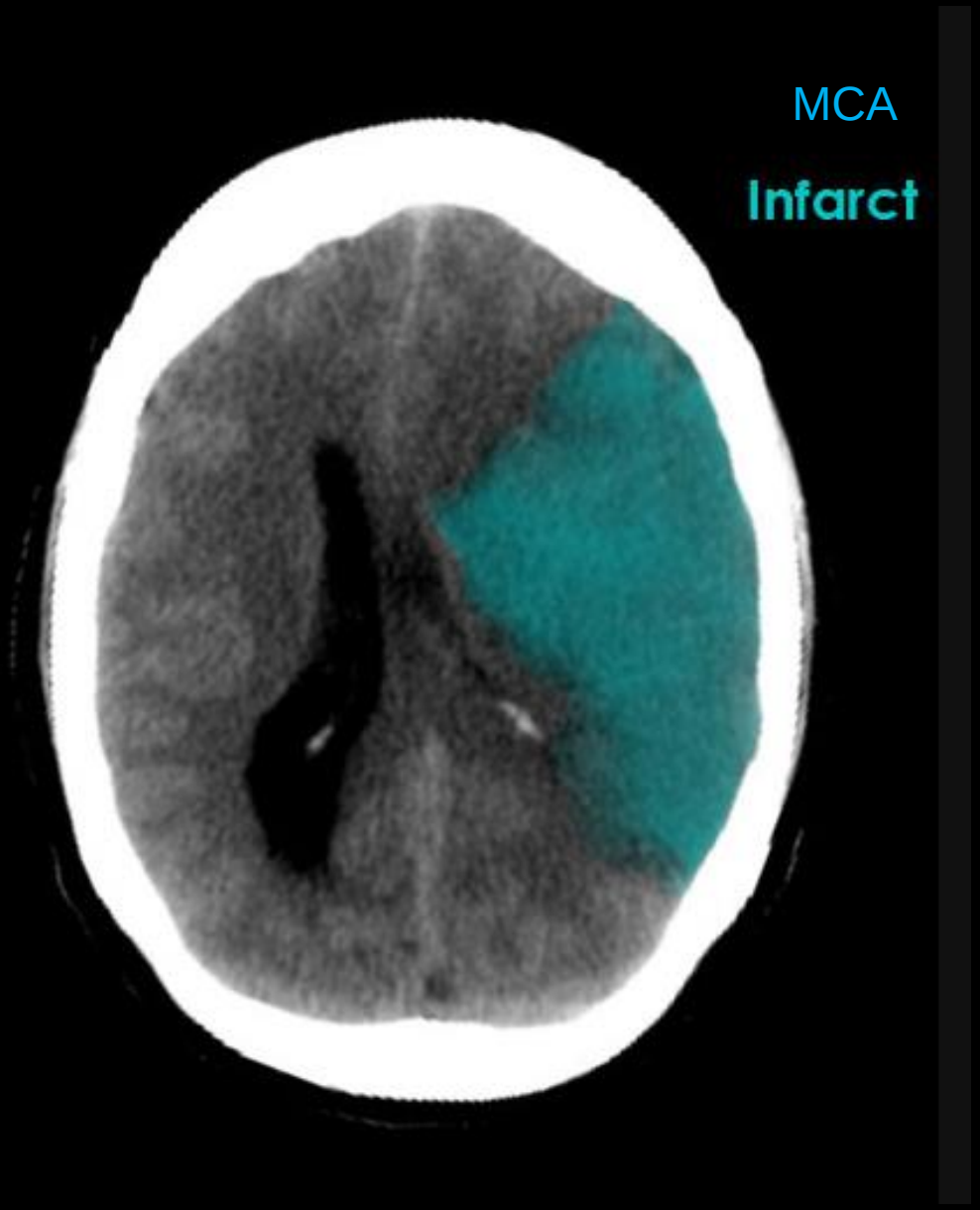
Anterior cerebral artery (ACA)

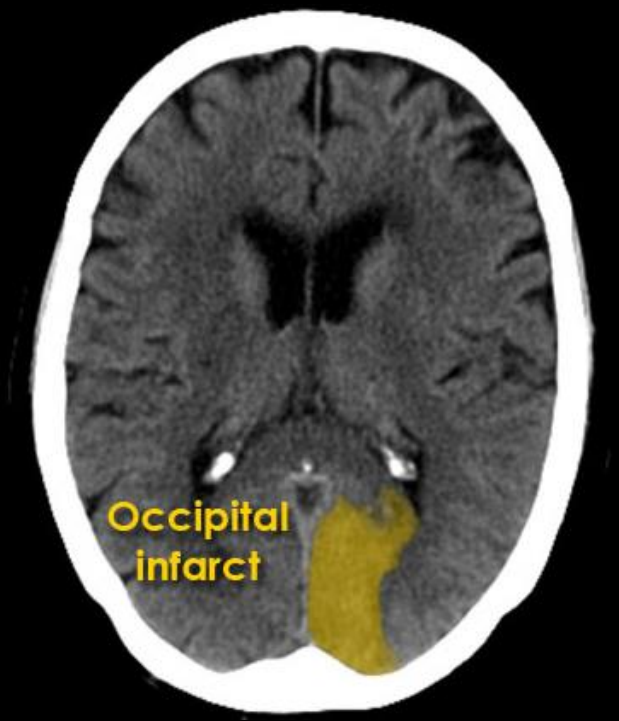
Middle cerebral artery (MCA)

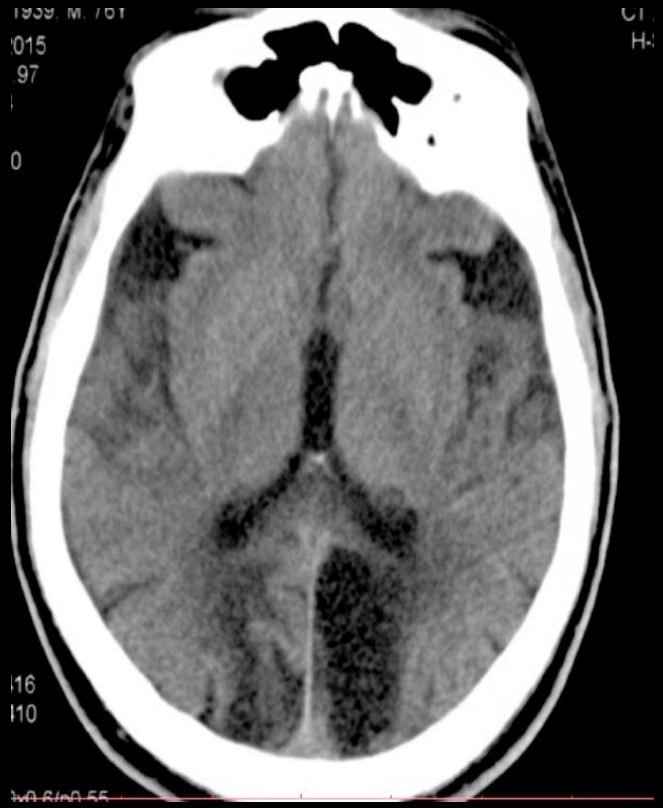
Posterior cerebral artery (PCA)



ACA INFARCT

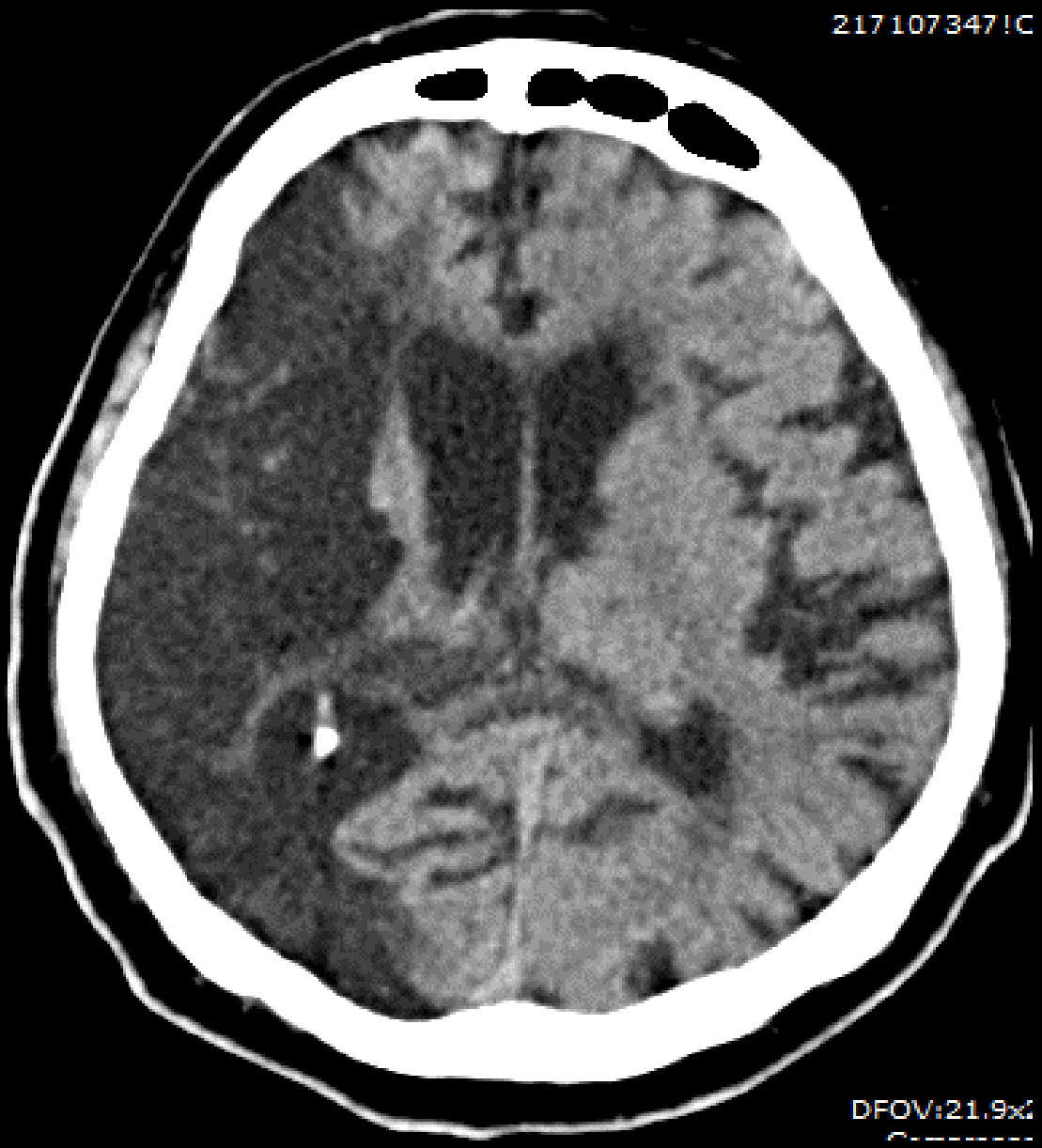

MCA
Infarct





PCA infarct

977


217107347!C
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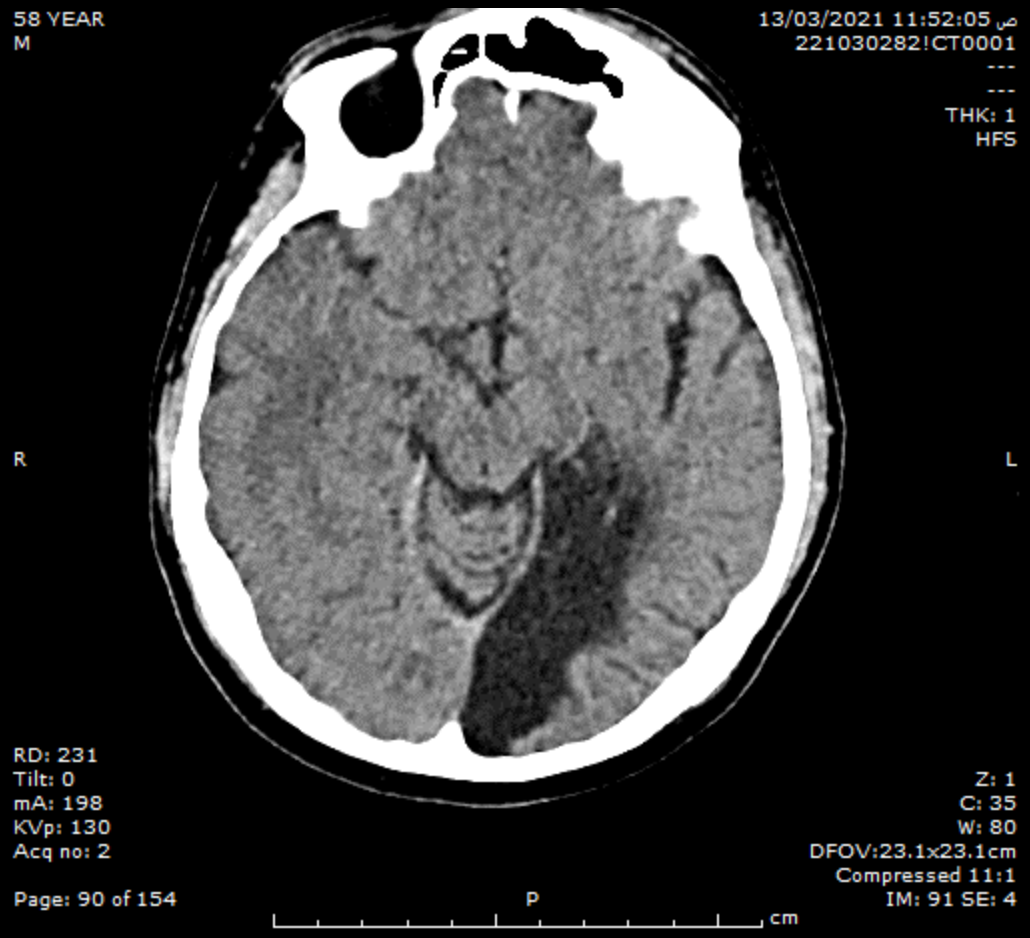
1.10
-173.0
WL: 3030 WW: 1977
10


58 YEAR
M
13/03/2021 11:52:05 ص
221030282!CT0001

THK: 1
HFS
R
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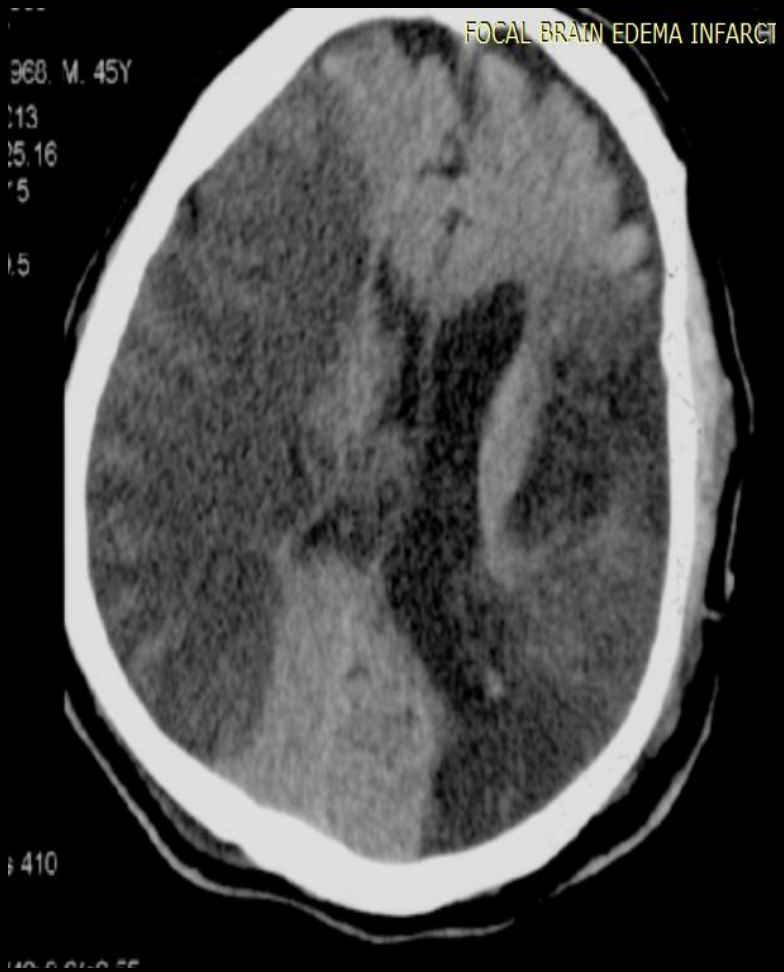
RD: 231
Tilt: 0
mA: 198
KVp: 130
Acq no: 2
Page: 90 of 154
P
cm
Z: 1
C: 35
W: 80
DFOV:23.1x23.1cm
Compressed 11:1
IM: 91 SE: 4

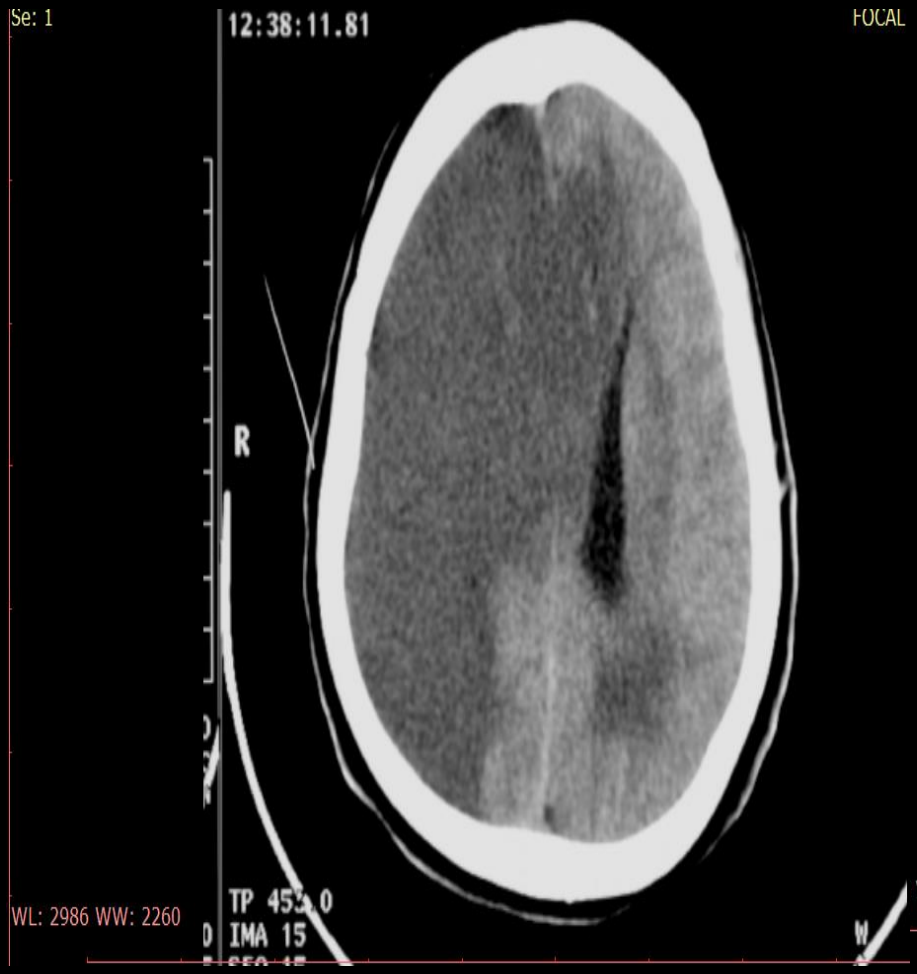

FOCAL BRAIN EDEMA INFARCT
120
mAs 410
1.0
0.0


FOCAL BRAIN EDEMA INFARCT


WL: 3019 WW: 1602 [D]

Right

Left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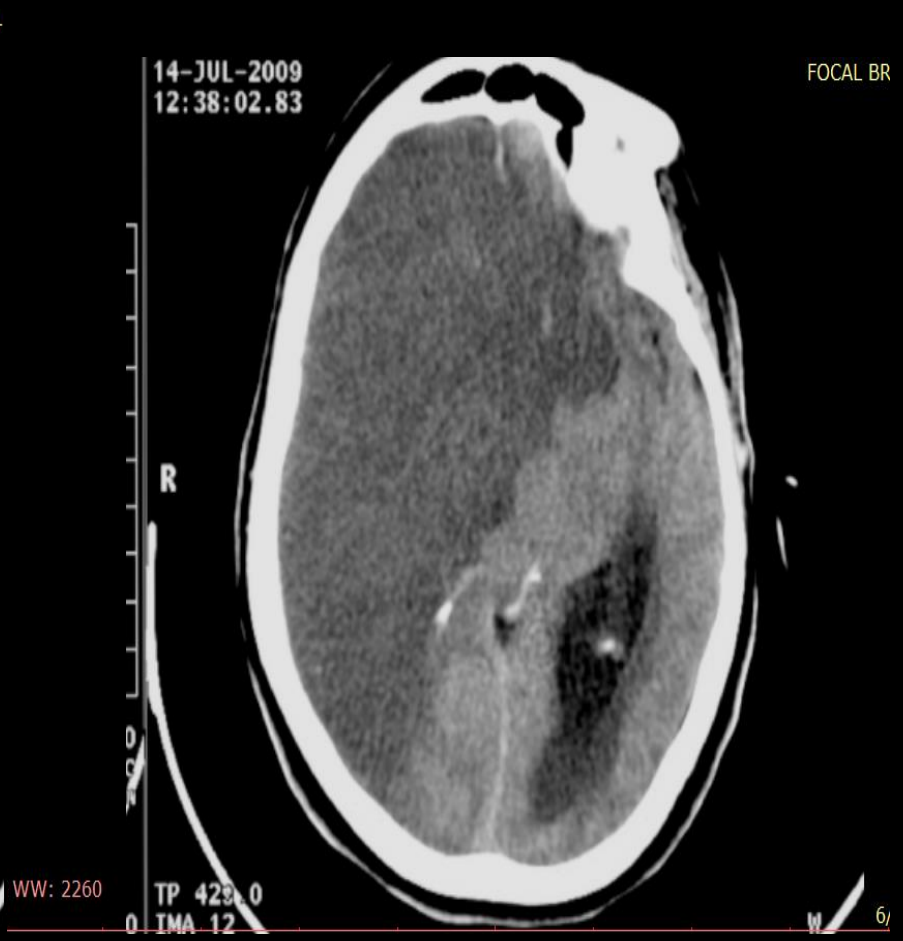
FOCAL
R
WL: 2986 WW: 2260
TP 453.0
IMA 15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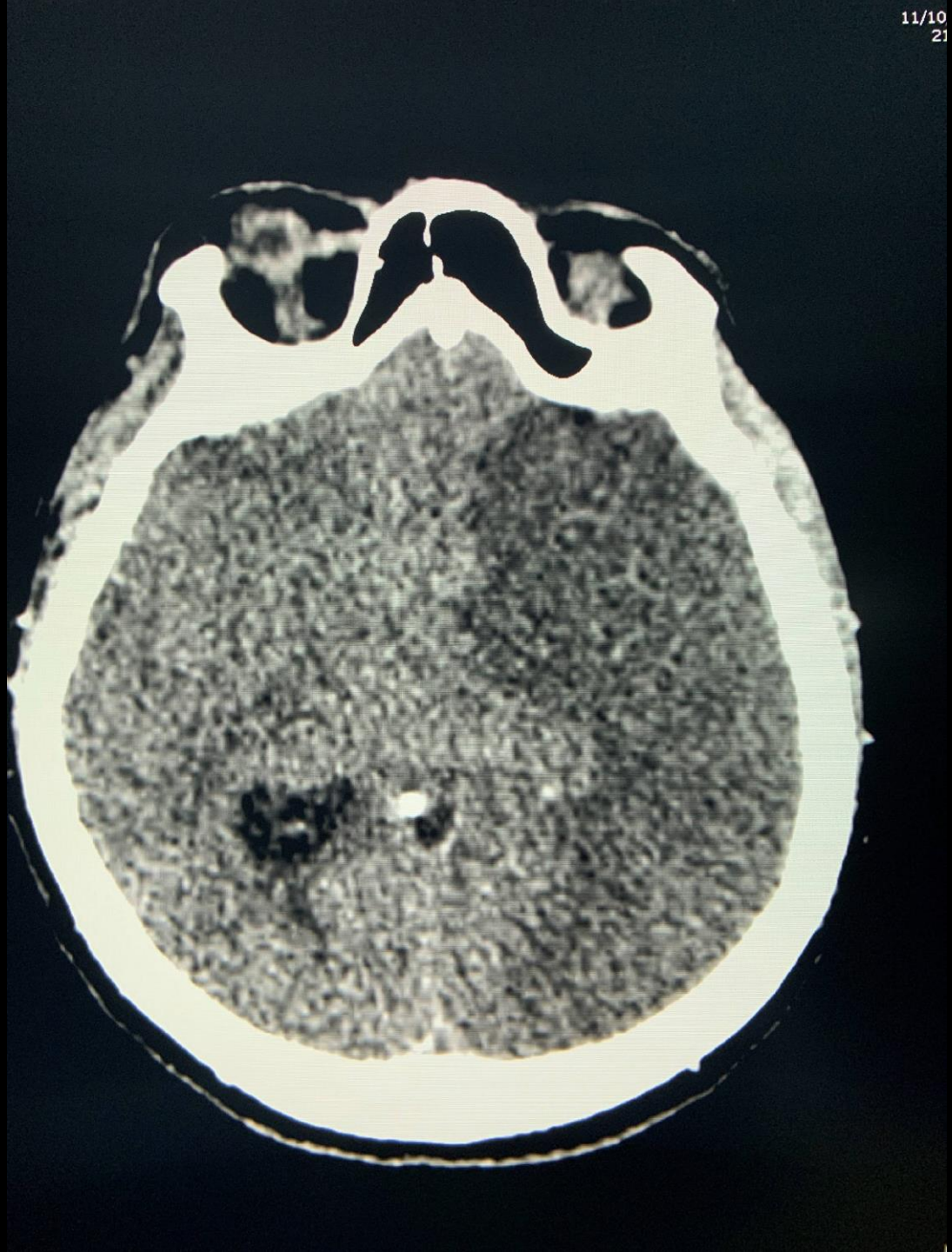
FOCAL BR
R
WW: 2260
TP 429.0
IMA 12


11/10
21


ahmoud Marawah, Saadah
180834
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3 YEAR
A
BRAIN CT Withou
Head 1.0 MPR
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2191397
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RD: 222
Tilt: 0
mA: 189
kVp: 130
Acq no: 2
Page: 130 of 184
P
cm
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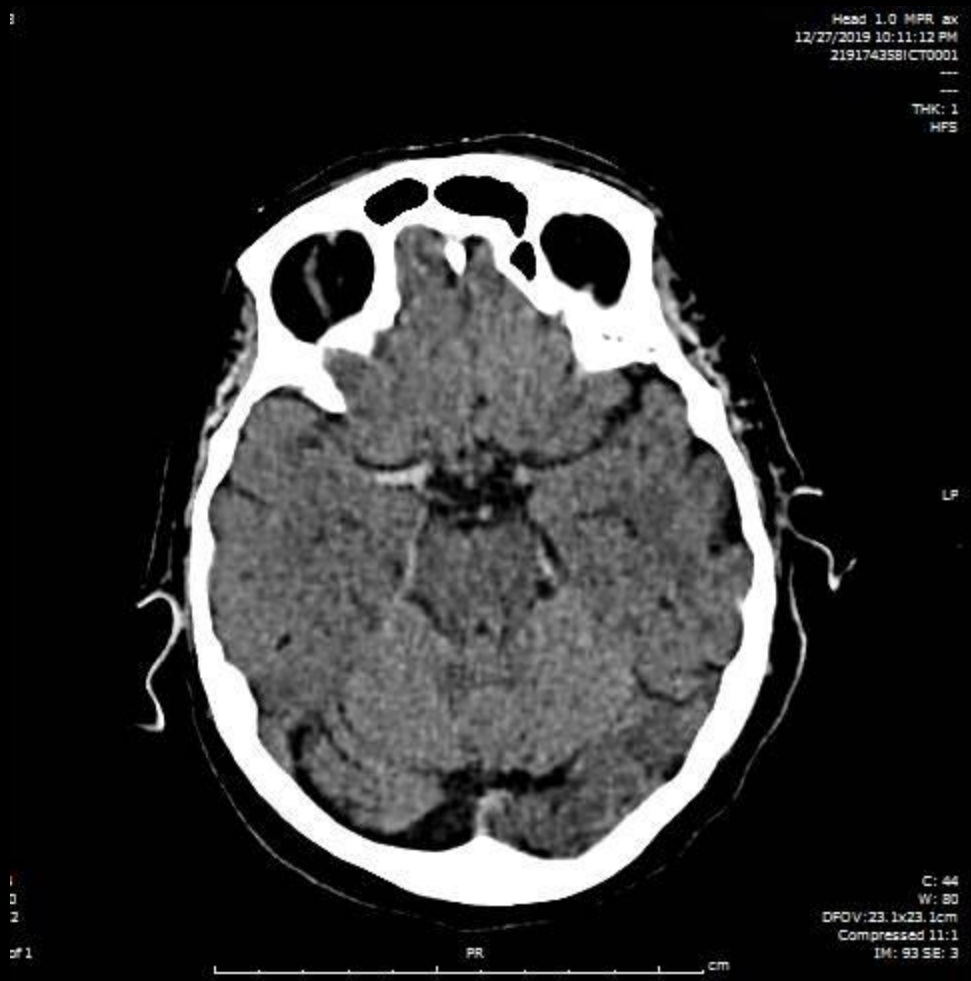
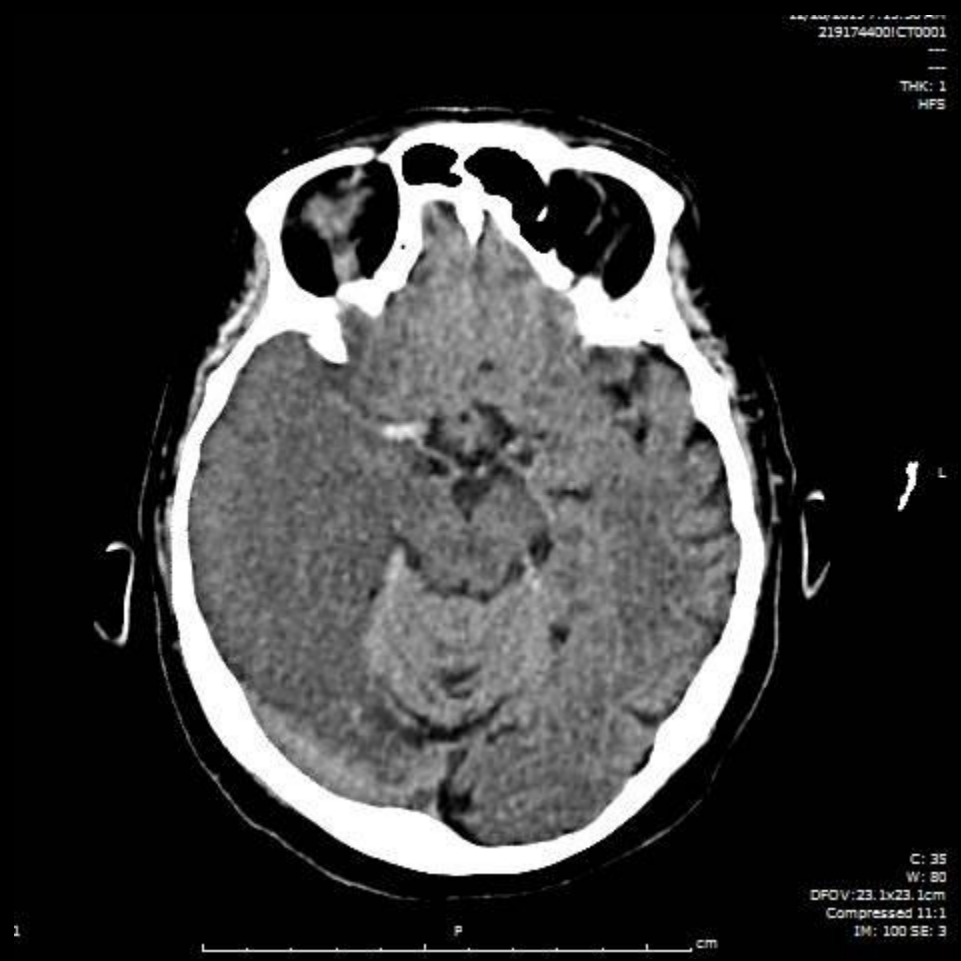
DFOV:22.2x26.
Compressed
IM: 131

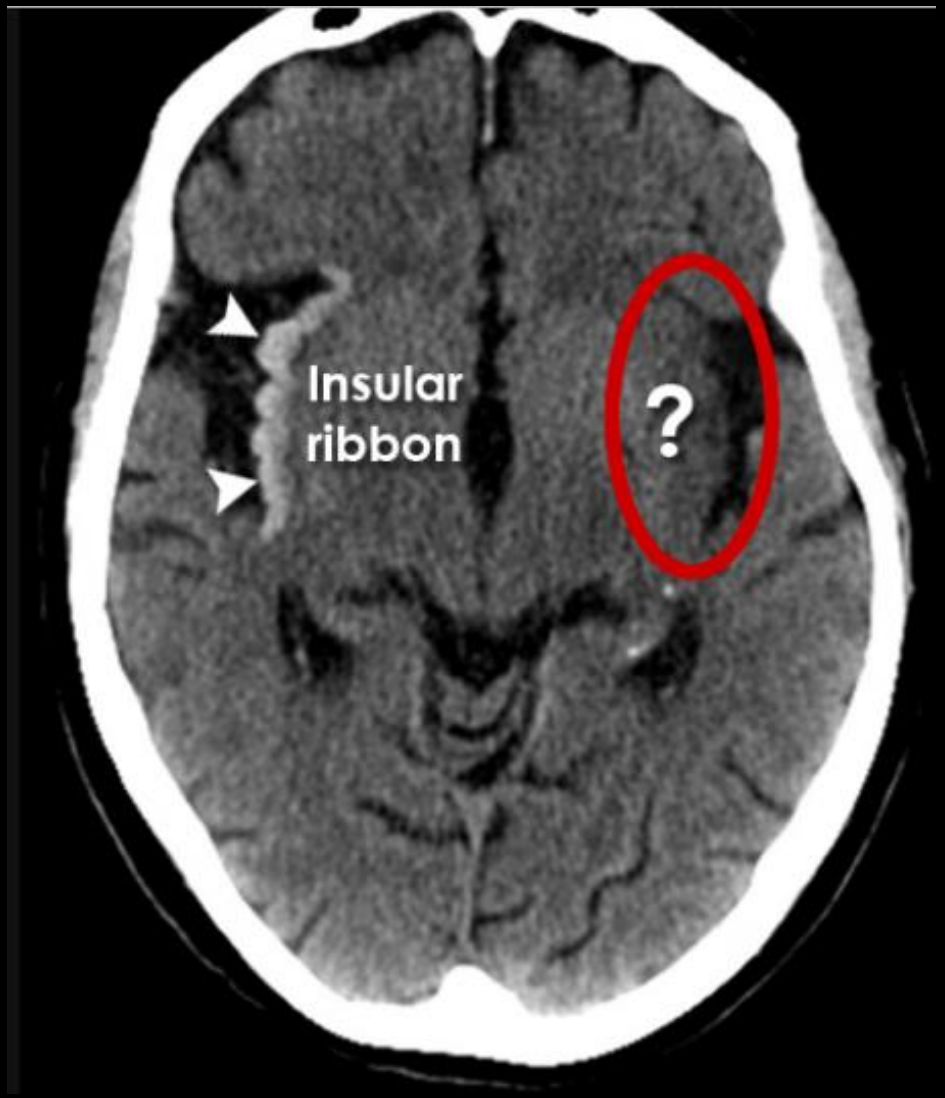
Hyperdense MCA sign

Hyperdense MCA sign



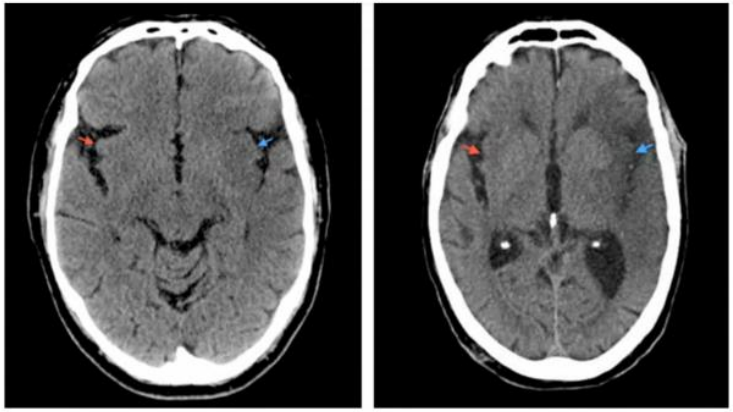
After 24 hours of the hyperdense MCA




Insular
ribbon
?

Insular Ribbon Sign on Left

- Loss of the normal insular cortex grey-white differentiation



<4 hours >4 hours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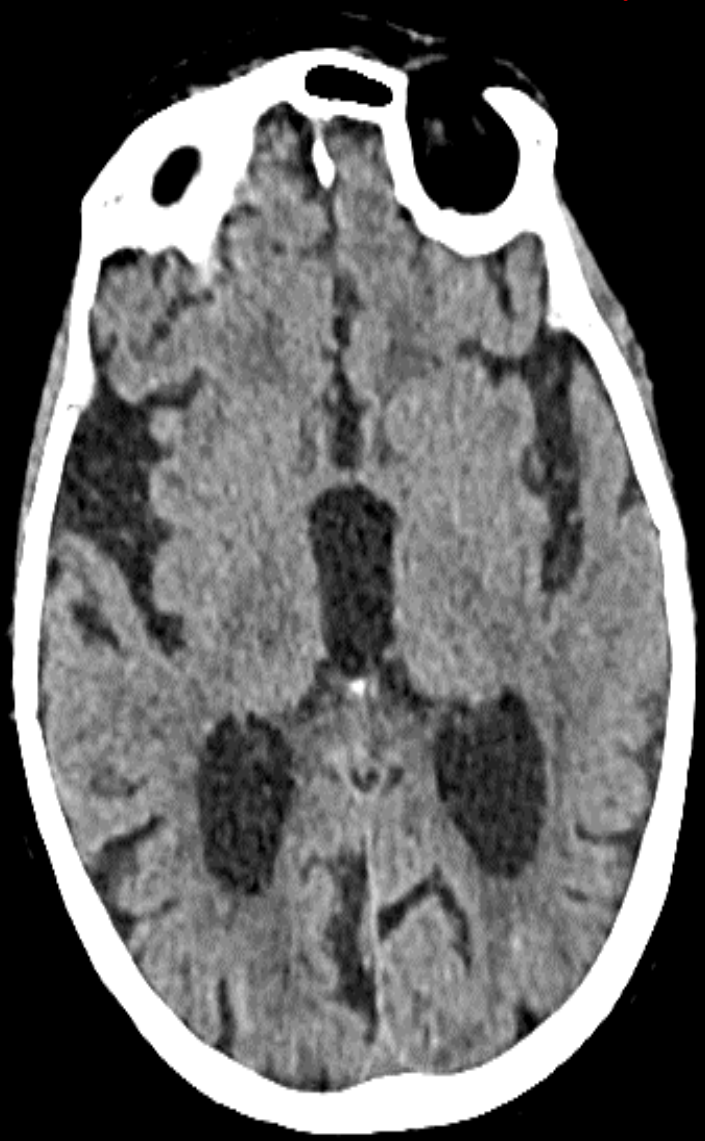
Compres

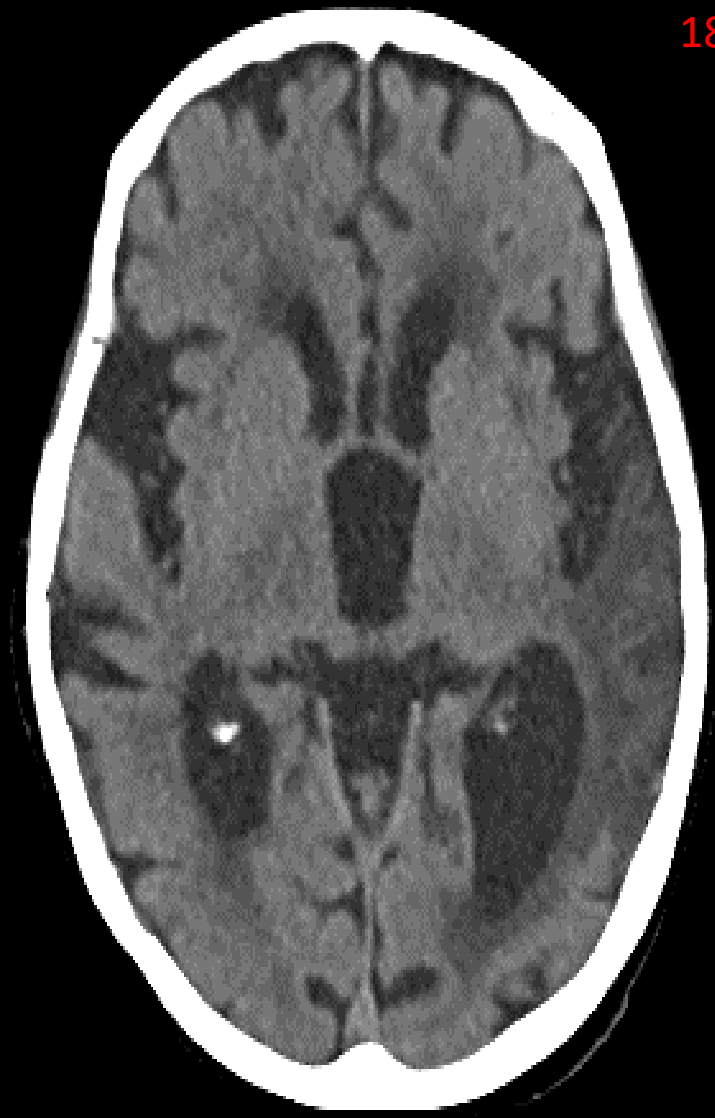

4
BRAIN CT Without C
Head 1.0 MPF
3/5/2018 12:32
218031926!C
175
P
DFOV:23
Compresse
IM: 8

Insular ribbon sign

16/5

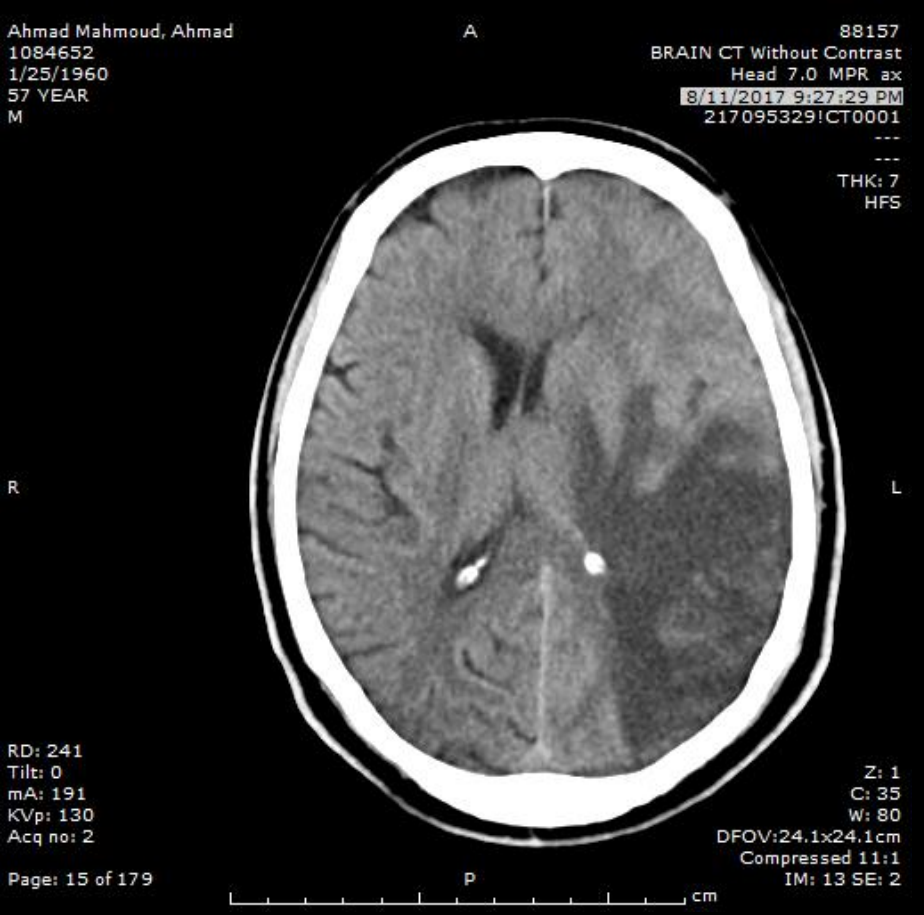


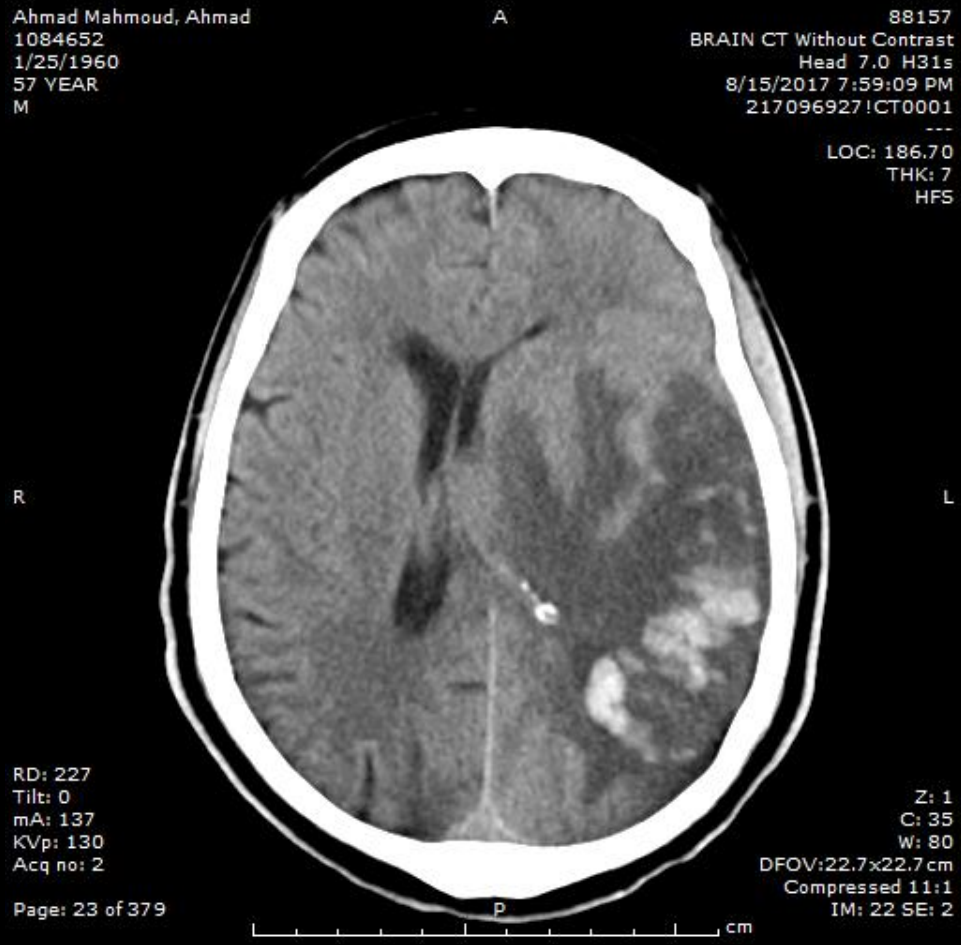
18/5



Extensive edema with midline shift

Hemorrhagic transformation





Vasogenic edema

Necrotic tissue: tumor v/s abscess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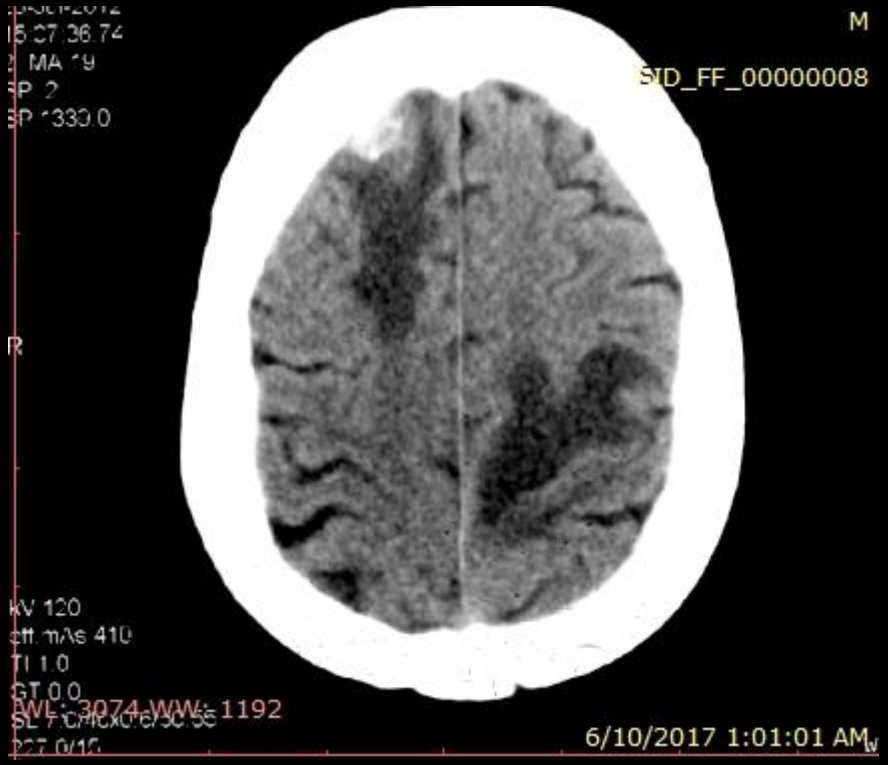
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6/10/2017 1:01:01 AM


FOCAL BRAIN EDEMA METS MASS ABSCESS
FOCAL BRAIN EDEMA METS MASS ABSCESS
M
SID_FF_00000008
WL: 3074 WW: 1192
6/10/2017 1:01:01 AM


FOCAL BRAIN EDEMA METS MASS ABSCESS
FOCAL BRAIN EDEMA METS MASS ABSCESS
M
SID_FF_00000008
WL: 3074 WW: 1192
6/10/2017 1:01:01 AM


SID_FF_00000008
WL: 3074 WW: 1192
6/10/2017 1:01:01 AM


M
SID_FF_00000008
WL: 3074 WW: 1192
6/10/2017 1:01:01 AM


FOCAL BRAIN EDEMA METS MASS ABSCESS
FOCAL BRAIN EDEMA METS MASS ABSCESS
M
SID_FF_00000008
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WL: 3074 WW: 1192
I.V.CONTRAST
6/10/2017 1:01:01 AM


JORDAN UNIVERSIT
FOCAL BRAIN EDEMA METS MASS ABSCESS
FOCAL BRAIN EDEMA METS MASS ABSCESS
M
SID_FF_00000008
WL: 3074 WW: 1192
6/10/2017 1:01:01 AM


THK: 1
HFS
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RD: 231
Tilt: 0
mA: 163
KVp: 130
Acq no: 2
Page: 49 of 165
P
cm
Z: 1
C: 35
W: 80
DFOV:23.1x23.1cm
Compressed 11:1
IM: 50 SE: 5


FOCAL BRAIN ED


FOCAL BRAIN EDEMA


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)9-NOV-2008
22:37:20.63
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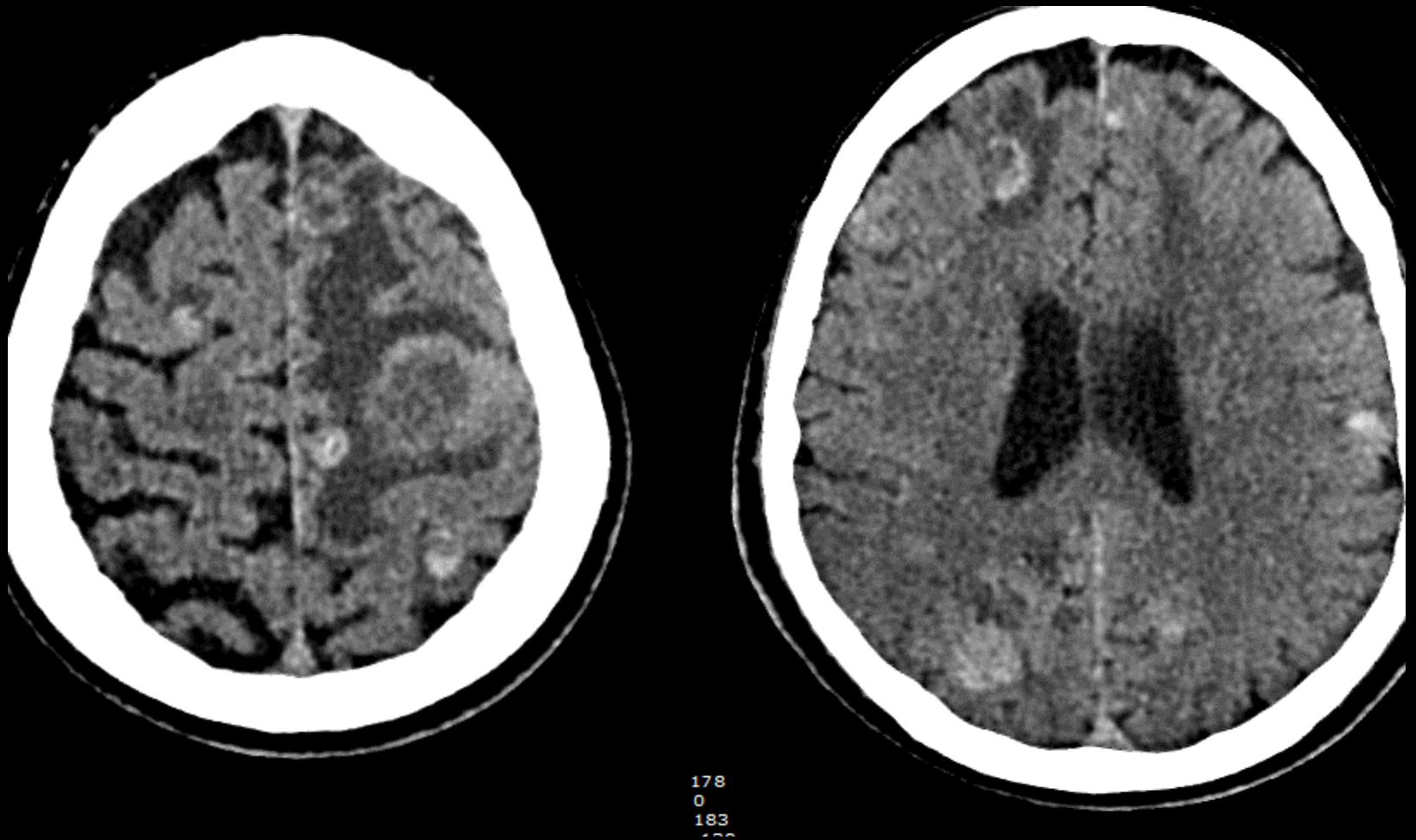
WL: 3133 WW: 1193
P -609.0
5/10/2017 1:01:01 AM

Heamorrahgic metastasis

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IMA 13
SEQ 13
R
kV 140
mA 206
TI 1.5
GT 0.0
SL 8.0
213 -18/-50
WL: 2639 WW: 1268


SID_F
':32.52
'5.5
31
31
40
06
.5
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-27/-53
LO
6/10/2017 1:01:01 AM

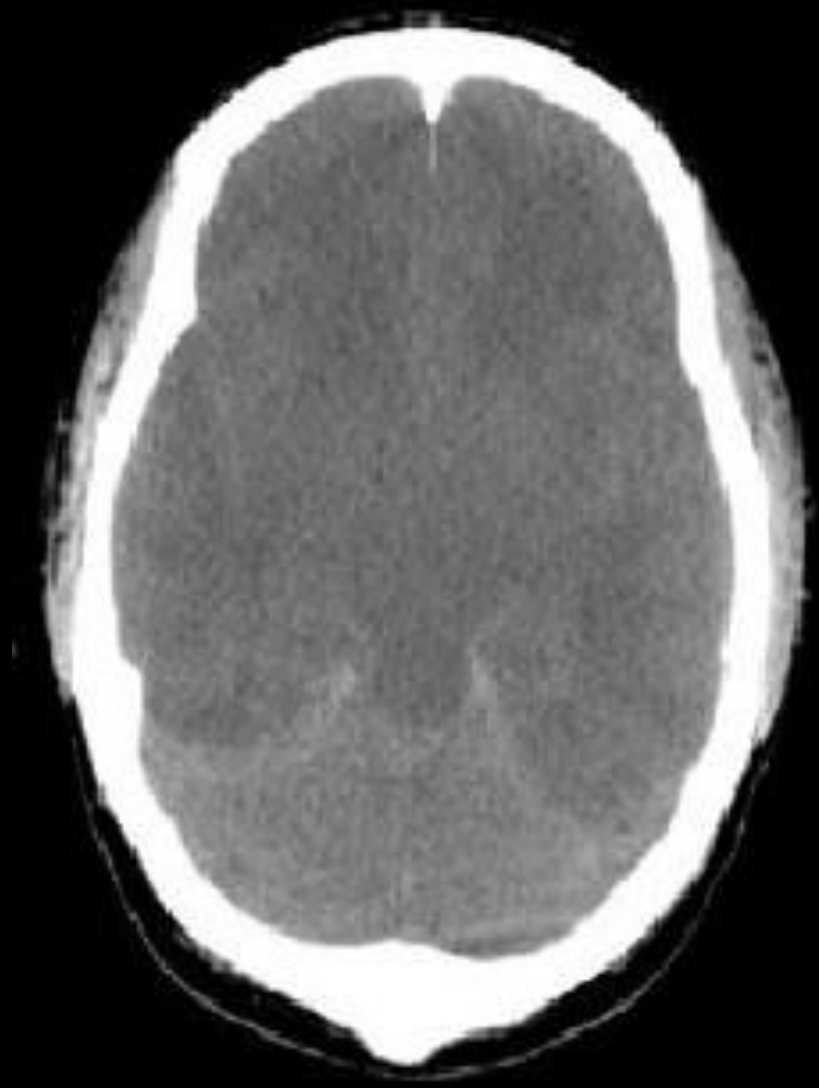
Diffuse brain edema

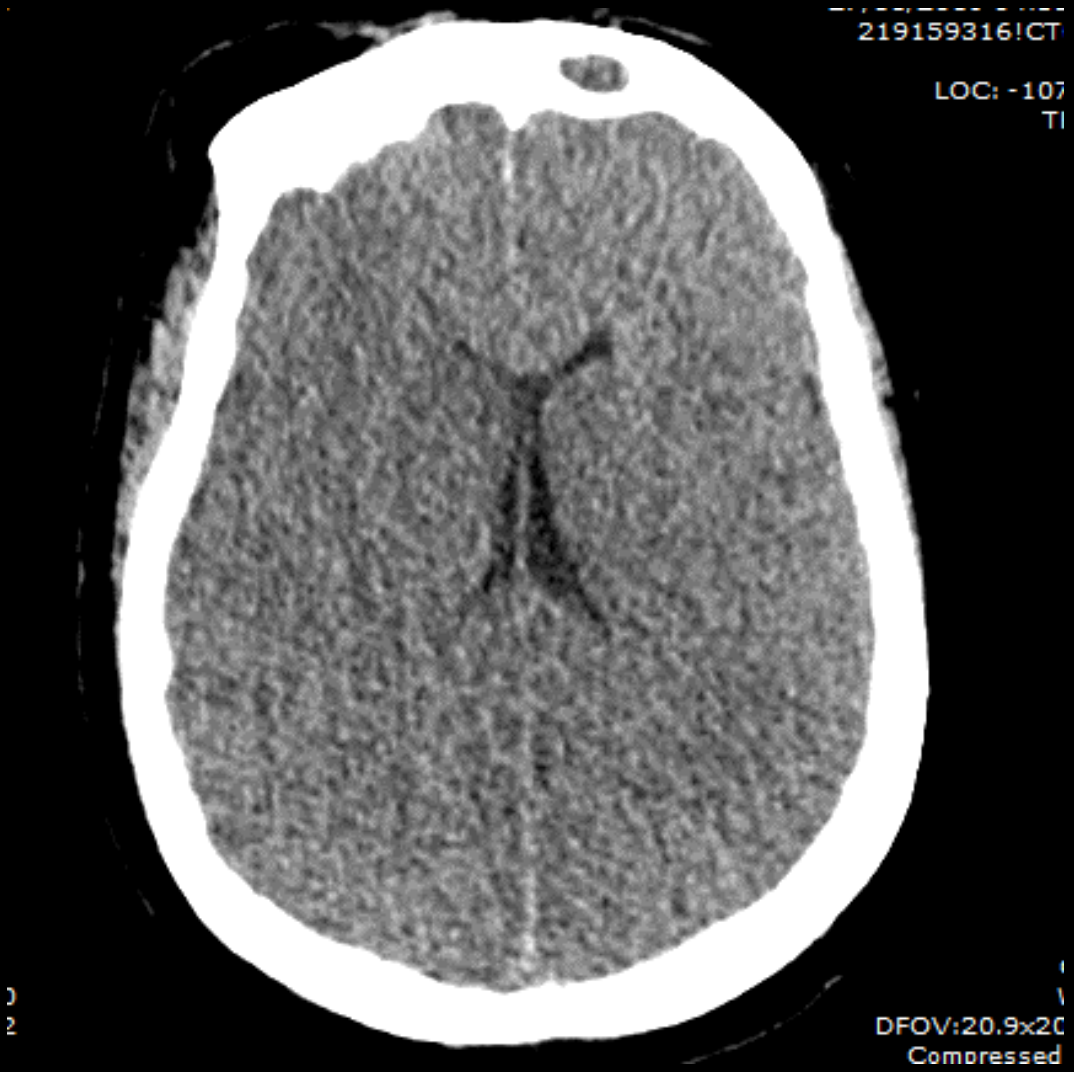
Signs of diffuse brain edema due to medical causes :

Diffuse brain hypodensity
Diffuse loss of grey white matter differentiation
Effacement of sulci
Small ventricles
Effacement of basal cistern
Pseudo SA sign

Diffuse brain edema

- increase brain hypodensity
- loss of gray white matter differentiation
- Effacement of the sulci
- Effacement of basal cisterns




219159316!CT
LOC: -107
TI
DFOV:20.9x20
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ROBA JEHAD HERZALLA
JORDAN UNIVERSITY HOS
DIFFUSE
DIFFUSE
Im: 9/108
Se: 1
WL: 3496 WW: 1960 [D]
6/10/2(


OBA JEHAD HERZALLA
JORDAN UNIVERSITY HOSI
DIFFUSE BRAIN EDEMA
DIFFUSE BRAIN EDEMA
SID_D_00000006
6/10/2017 1:01:01 AM


-SEP-2009
:14:44.30
WL: 3285 WW: 1684 [D]
-59.0
A 13
6/10/2017 1:


M
:14:56.30
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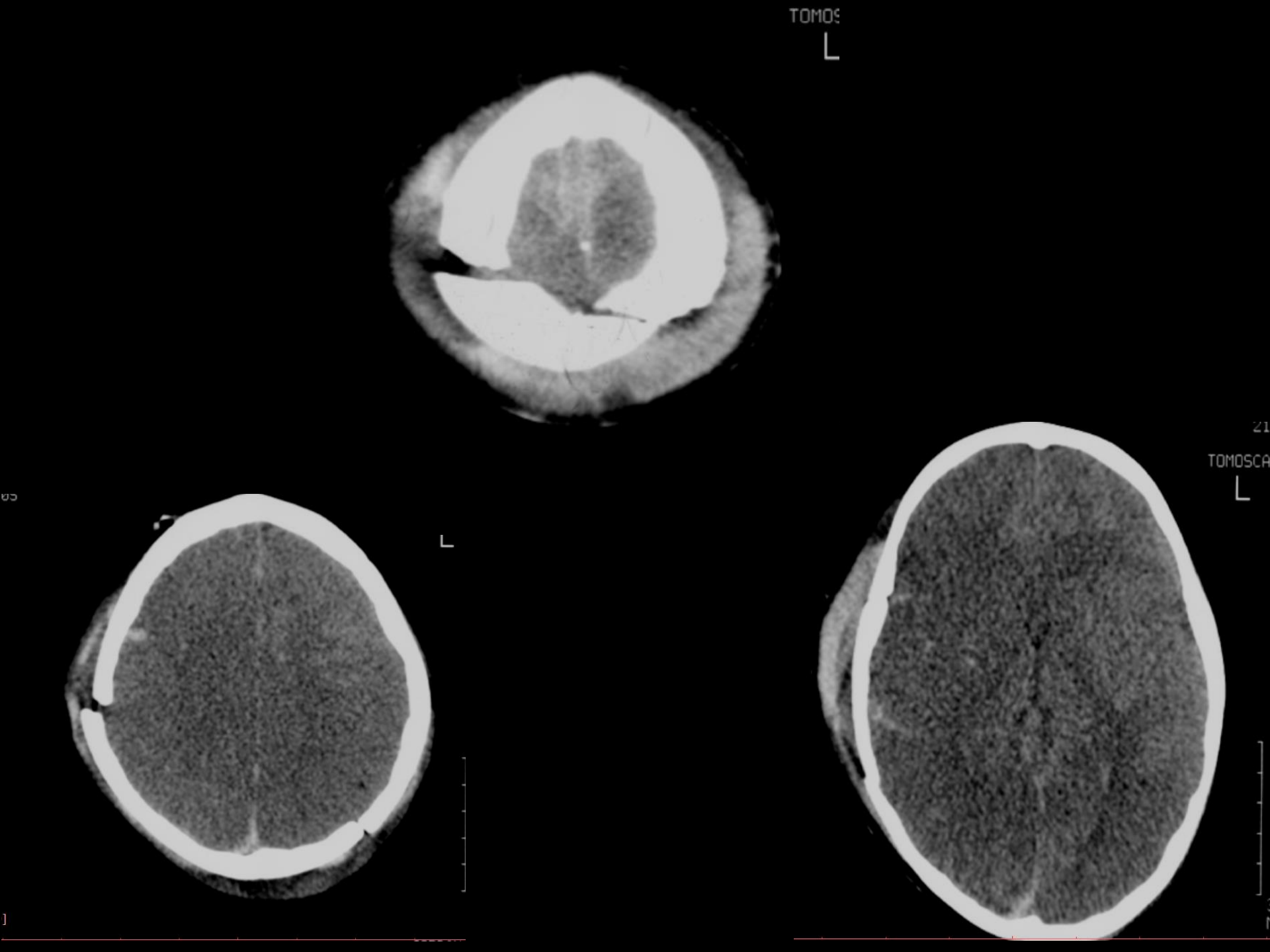
A 17
6/10/2017 1:01:01 AM


TOMOS
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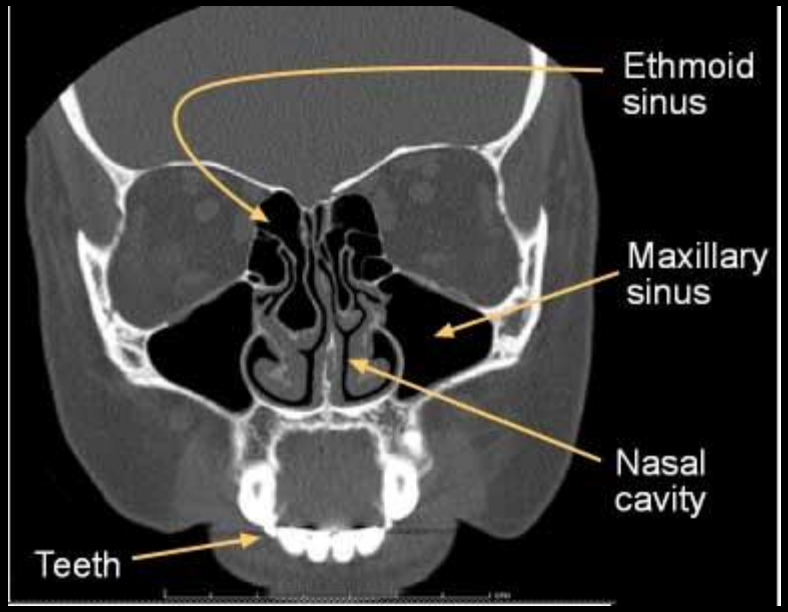
L

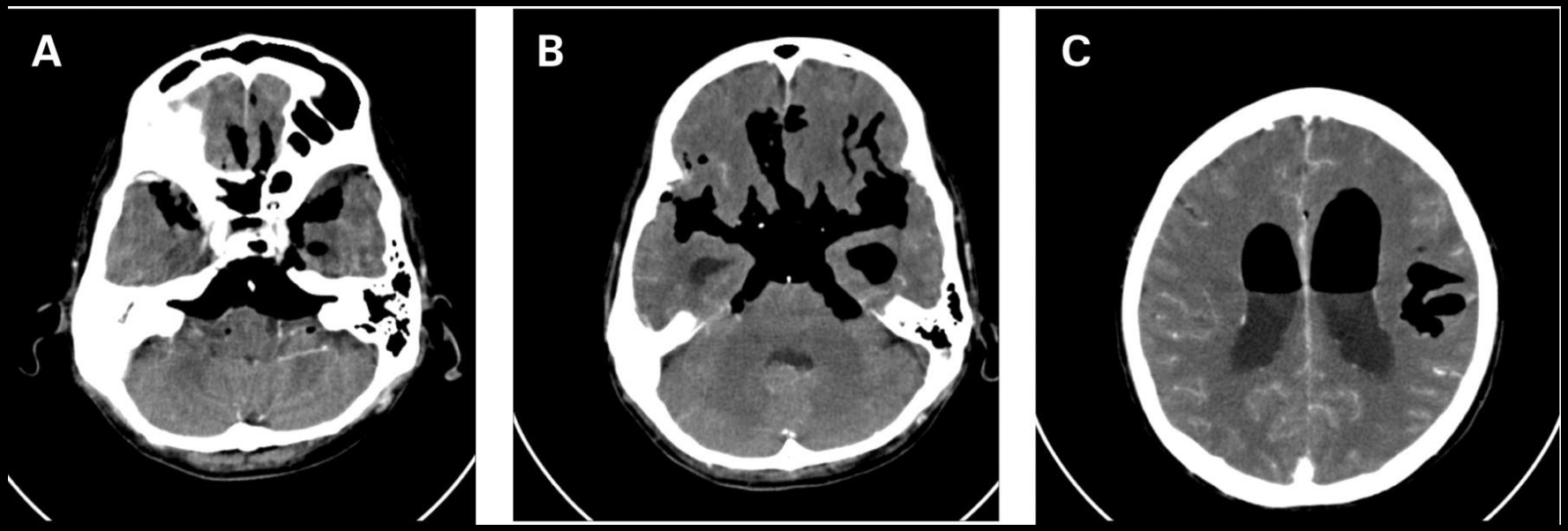
Hypodense Lesions:

Air:

Normal : sinuses



Abnormal: Pneumocephalus (post Sx or post trauma)




F
Page: 218 of 339
cm
Compressed 11:1
IM: 51 SE: 4

Compressed 11:1
IM: 89 SE: 3

Hypodense Lesions:

Fat:

Lipoma, dermoid cyst

Dermoid Cyst

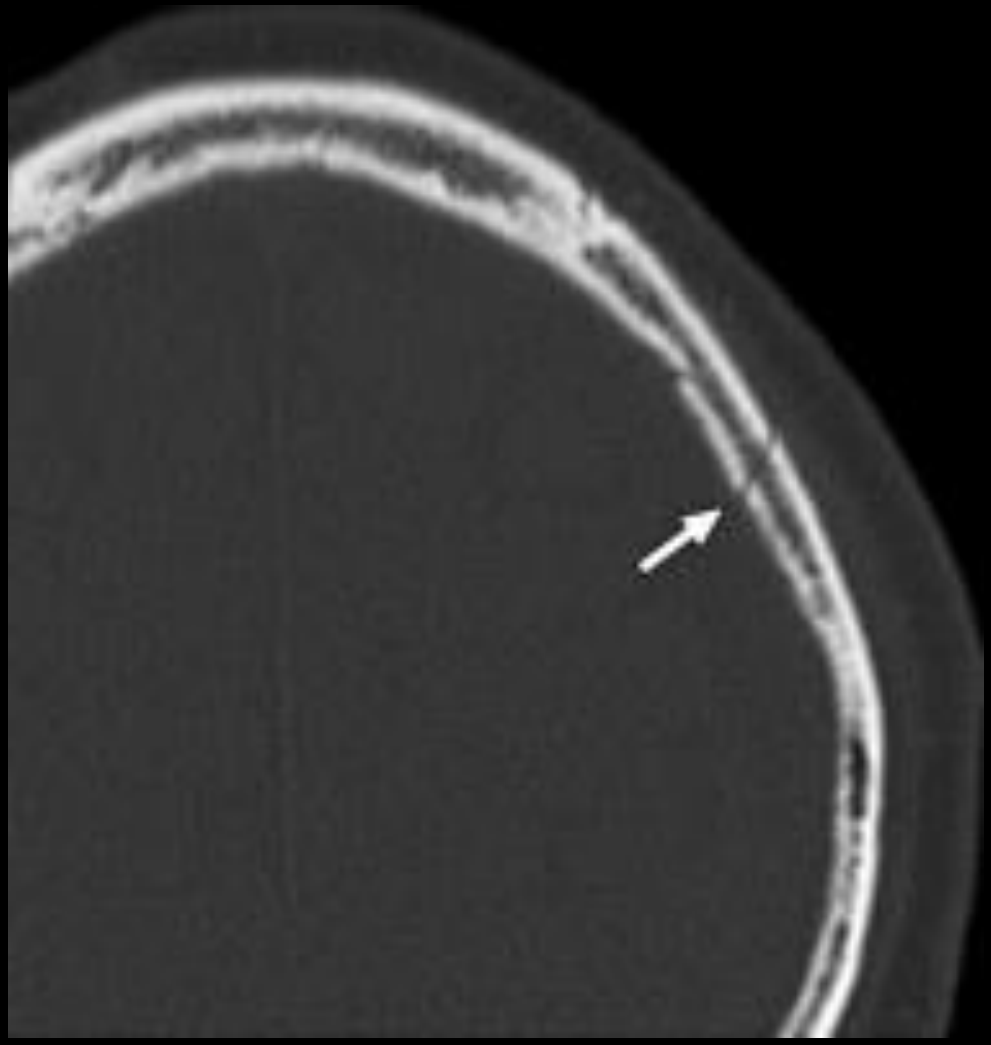
Skull Fractures

Linear

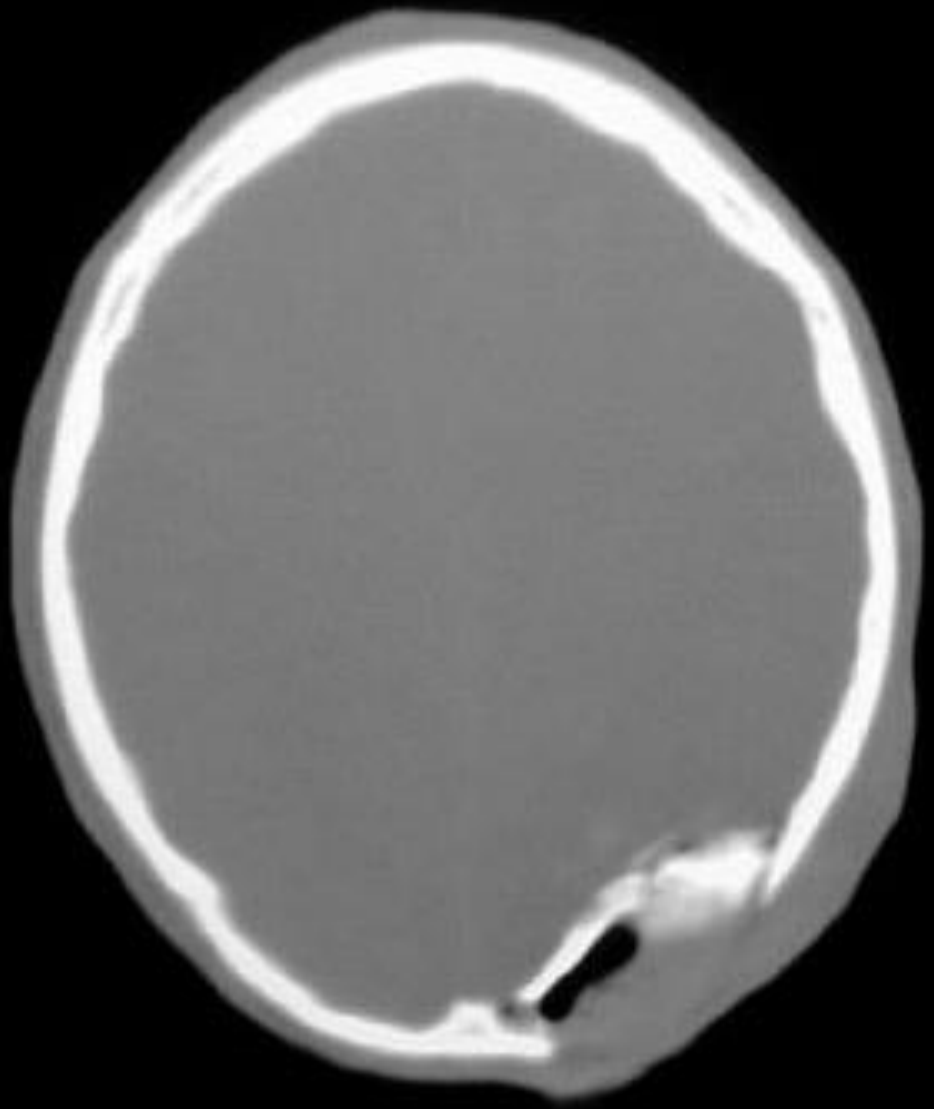
Depressed

Growing Fracture

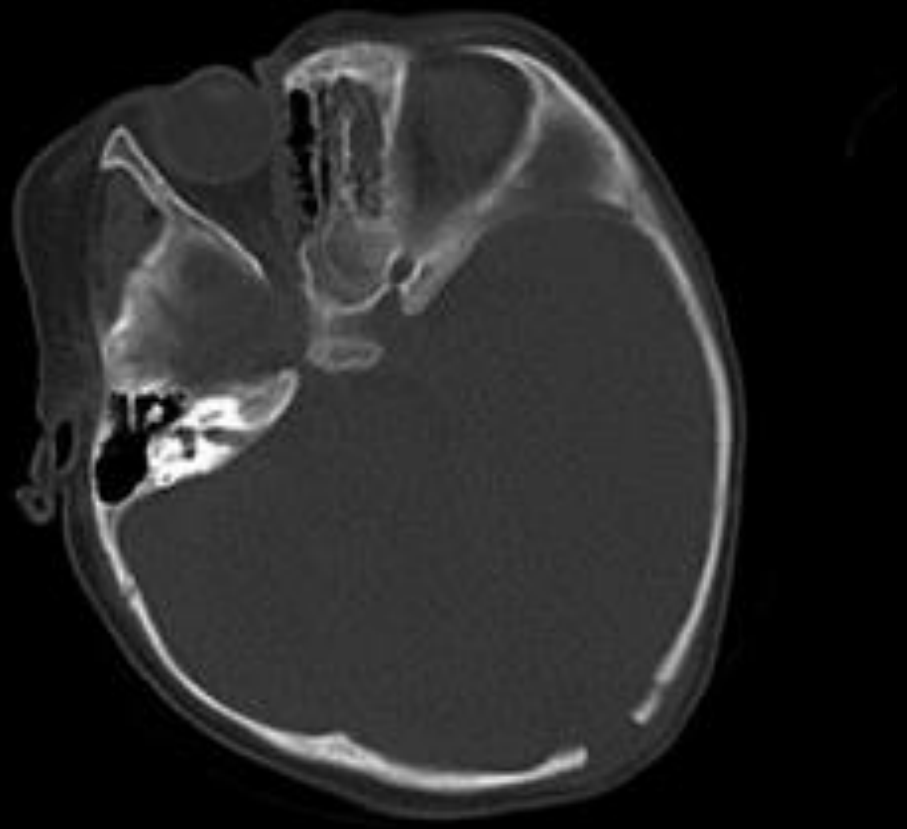
Linear Fracture



Depressed Fracture



Growing Fracture

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C 35


Im: 5/9
SID_dd_00000022
WL: 1920 WW: 1632


Im: 3/9
Se: 1
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H-SP-CR
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SEQ 10
R
WL: 2892 WW: 1480
1/1/2011 1:01:01 AM


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VC10B
H-SP-CR
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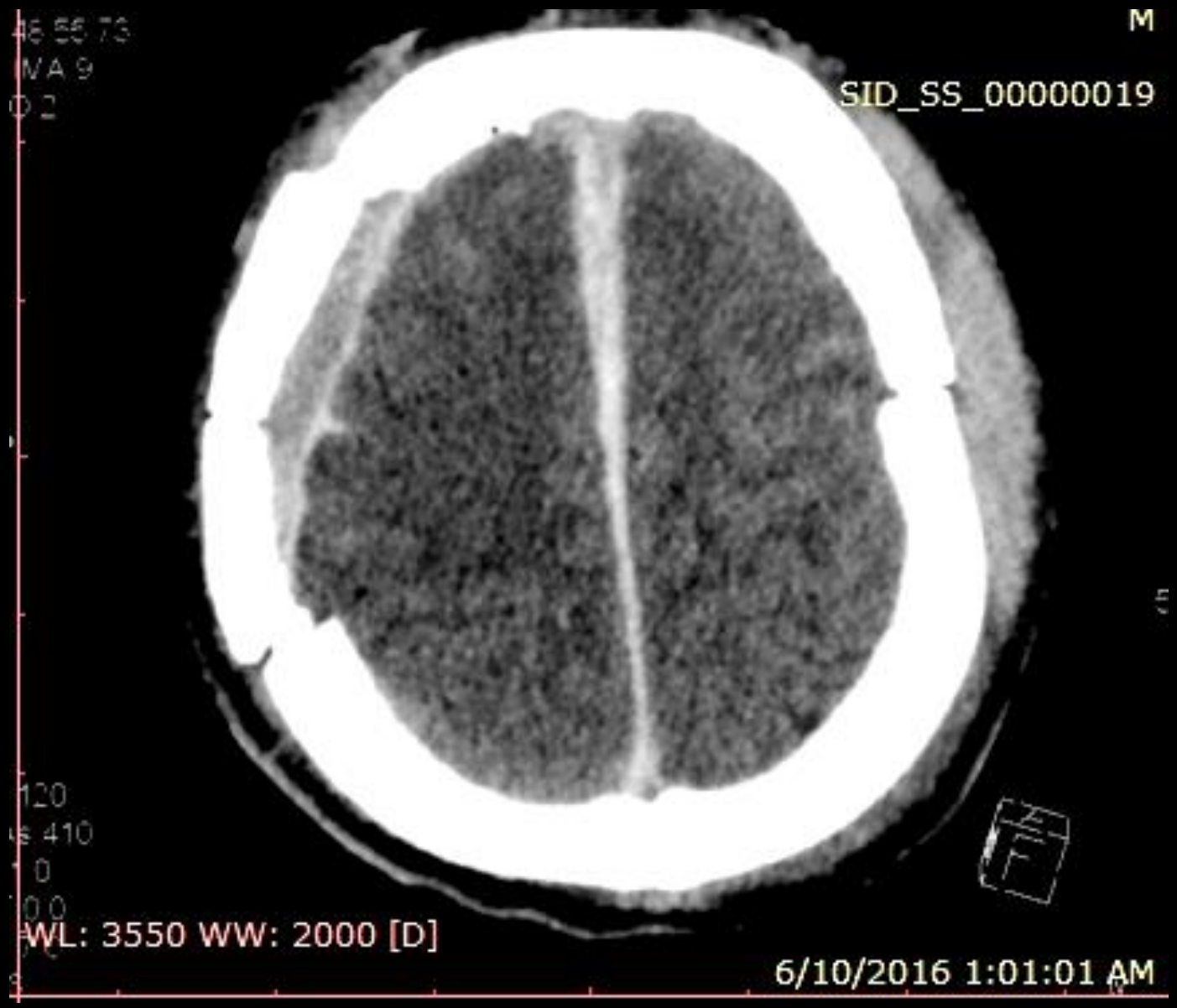
R
WL: 2828 WW: 1740
1/1/2011 1:01:01 AM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6/10/2016 1:01:01 AM


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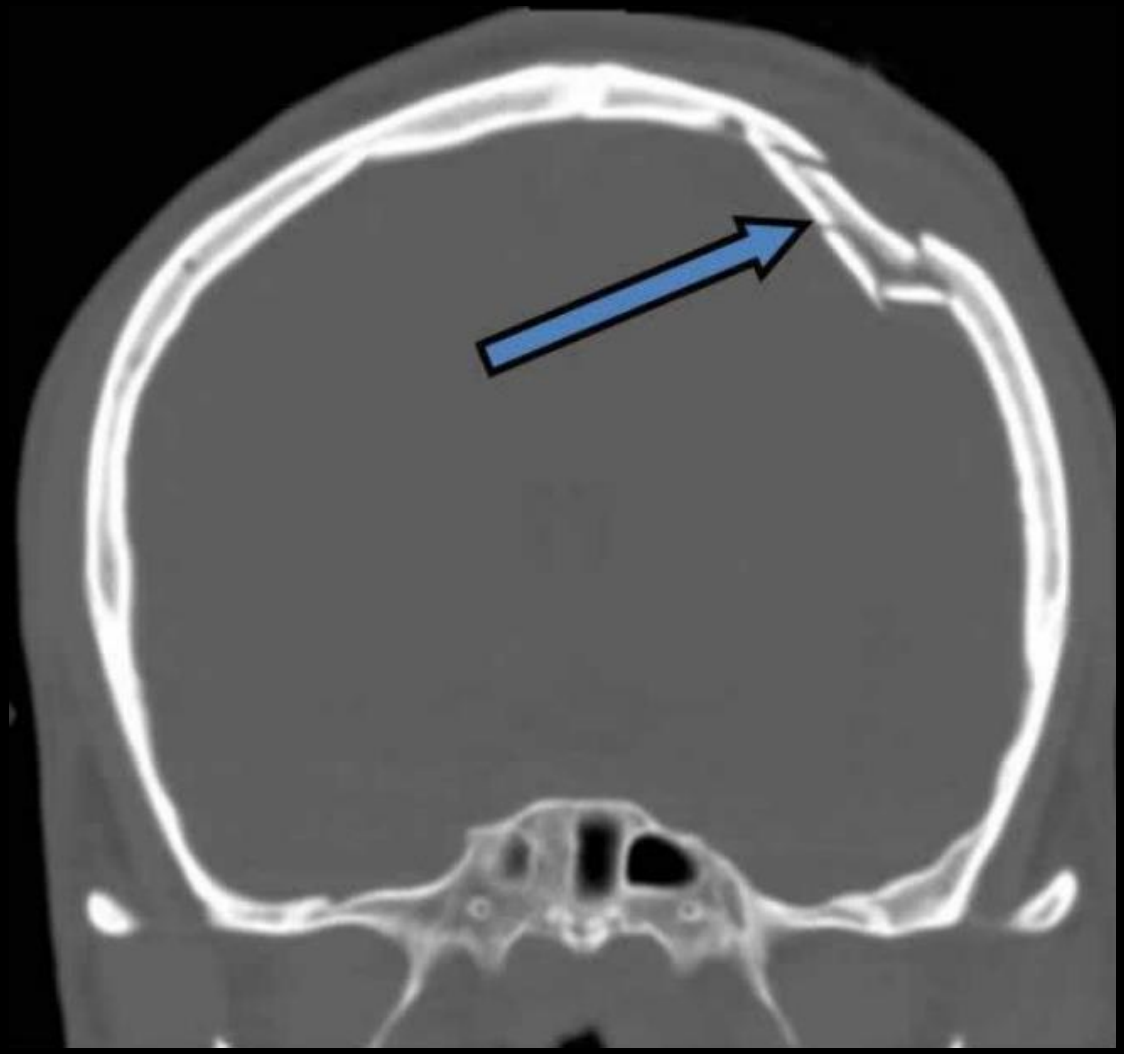
WL: 2822 WW: 1719 [D]
60s S9C0 4awa
6/10/2016 1:01:01 AM

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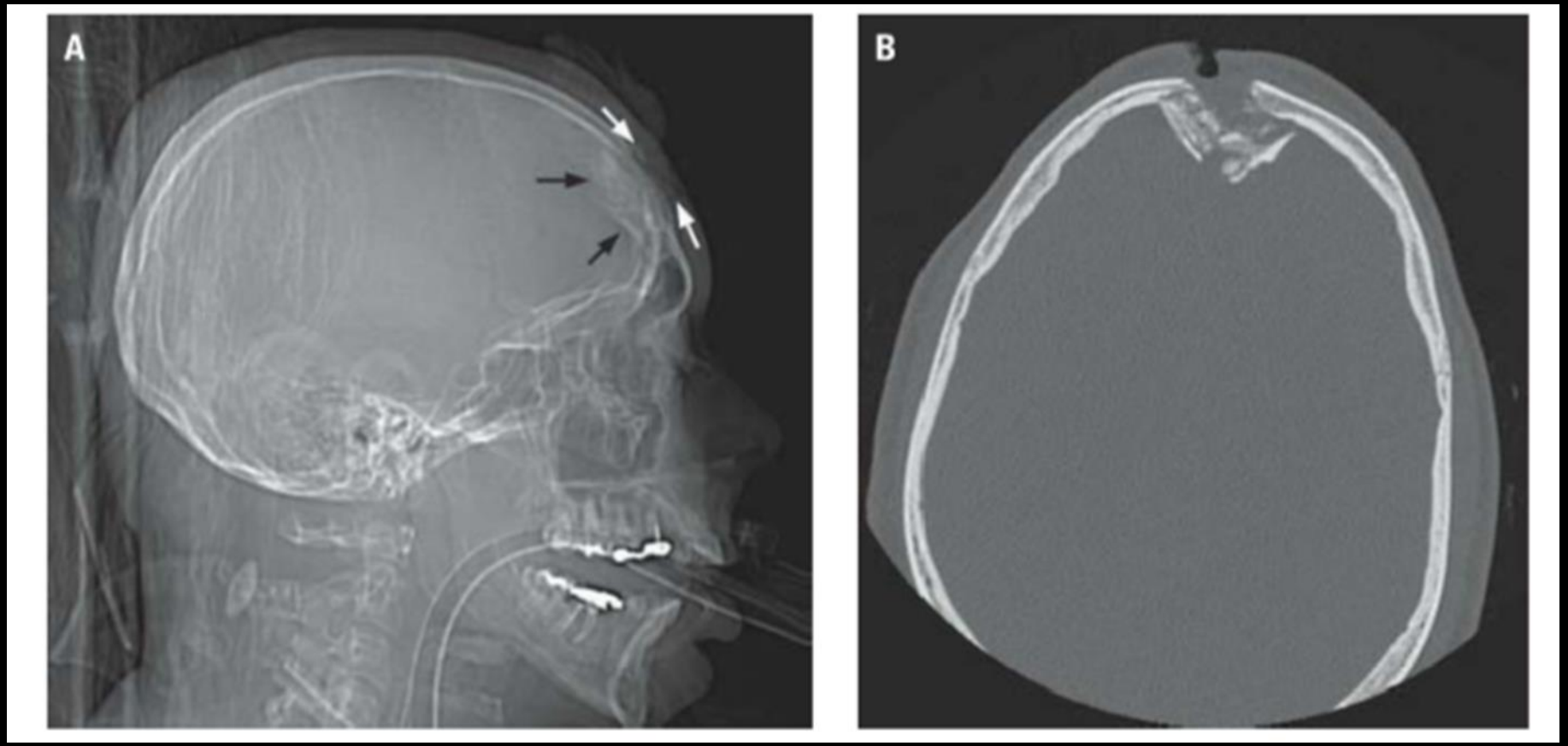
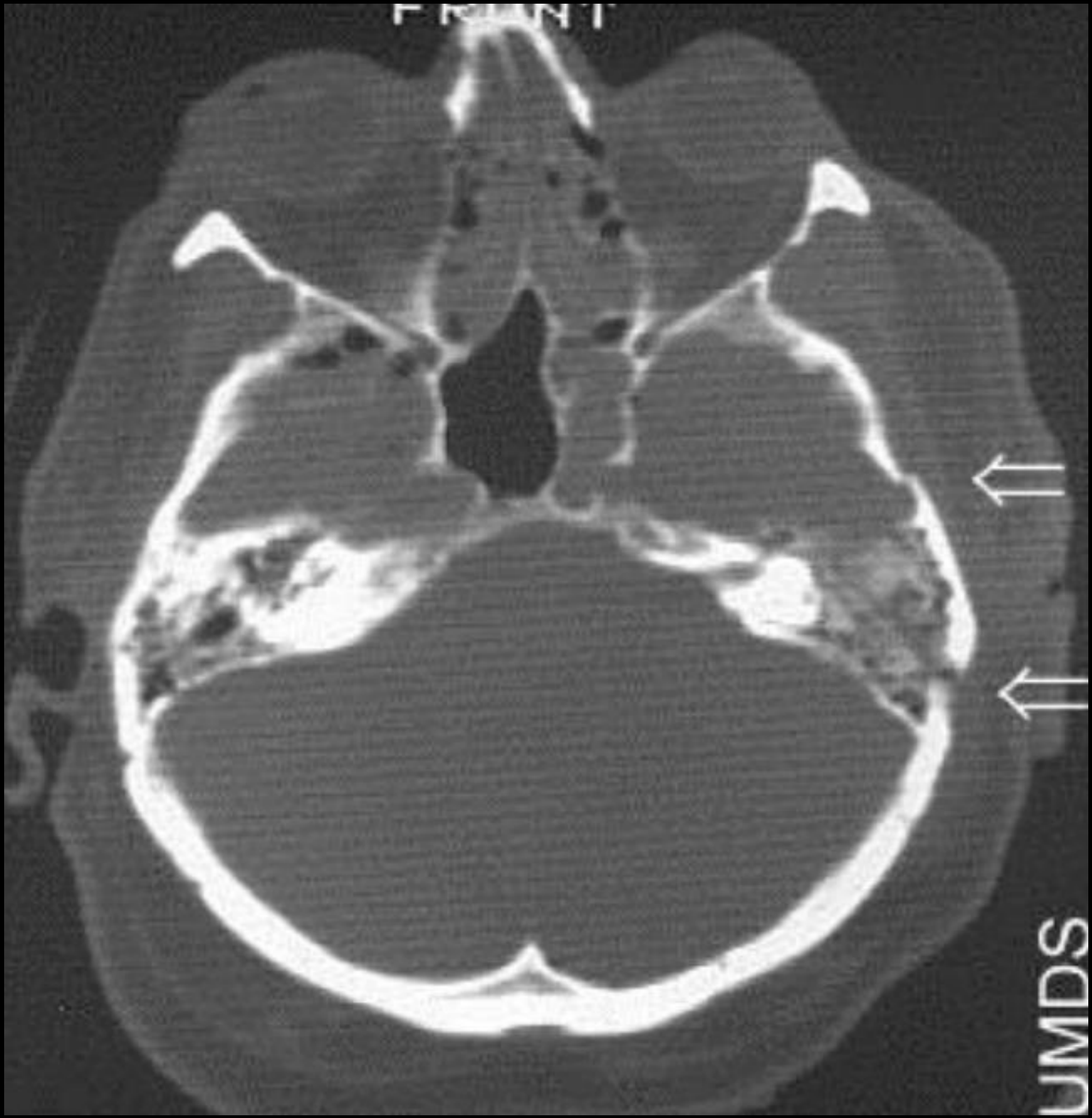
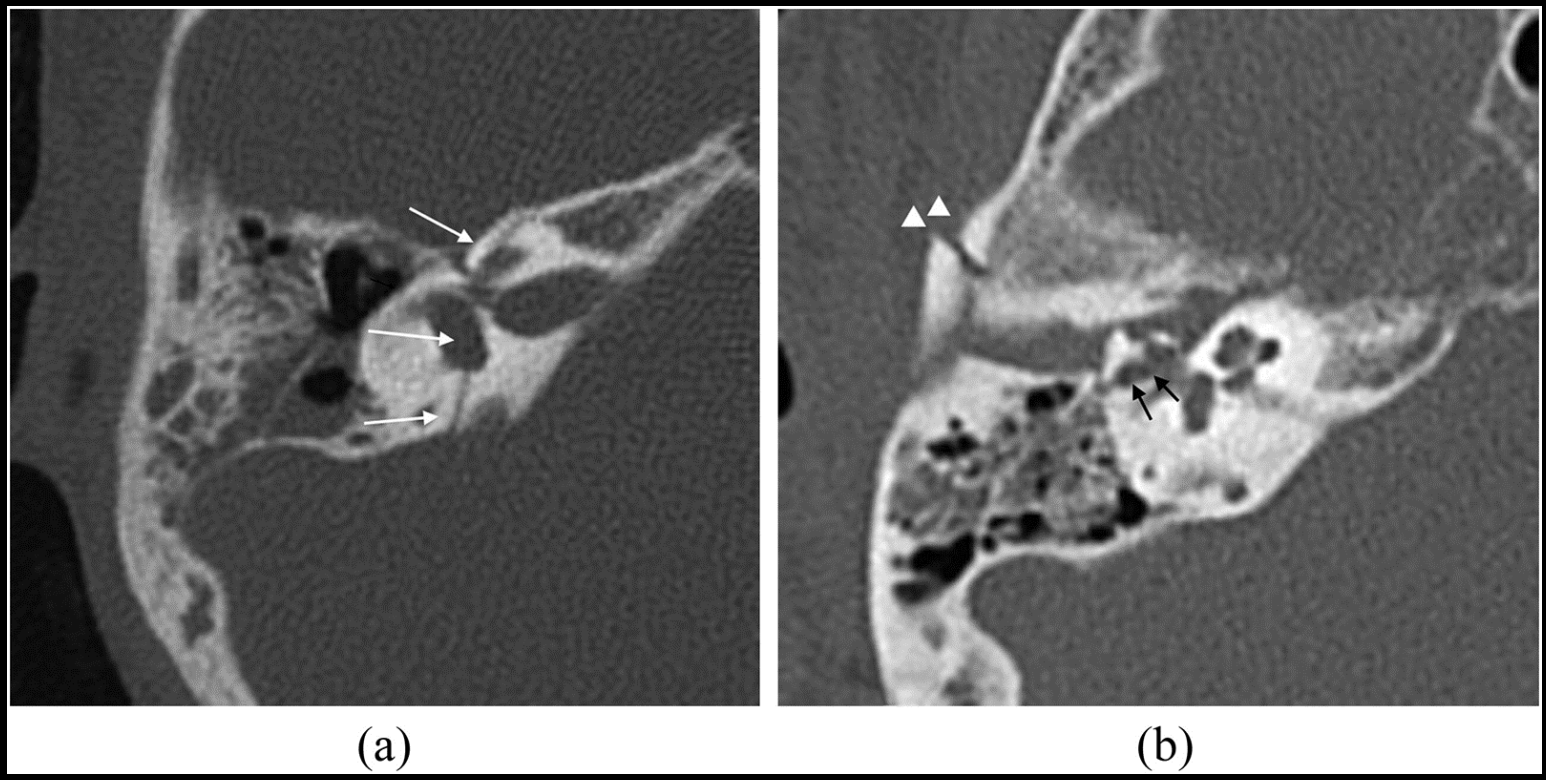
HFS
R
L
RD: 222
Tilt: 0
mA: 143
KVp: 130
Acq no: 3
Page: 68 of 211
Z: 1
C: 450
W: 1500
DFOV:22.2x26.3cm
Compressed 11:1
IM: 69 SE: 3
P
cm

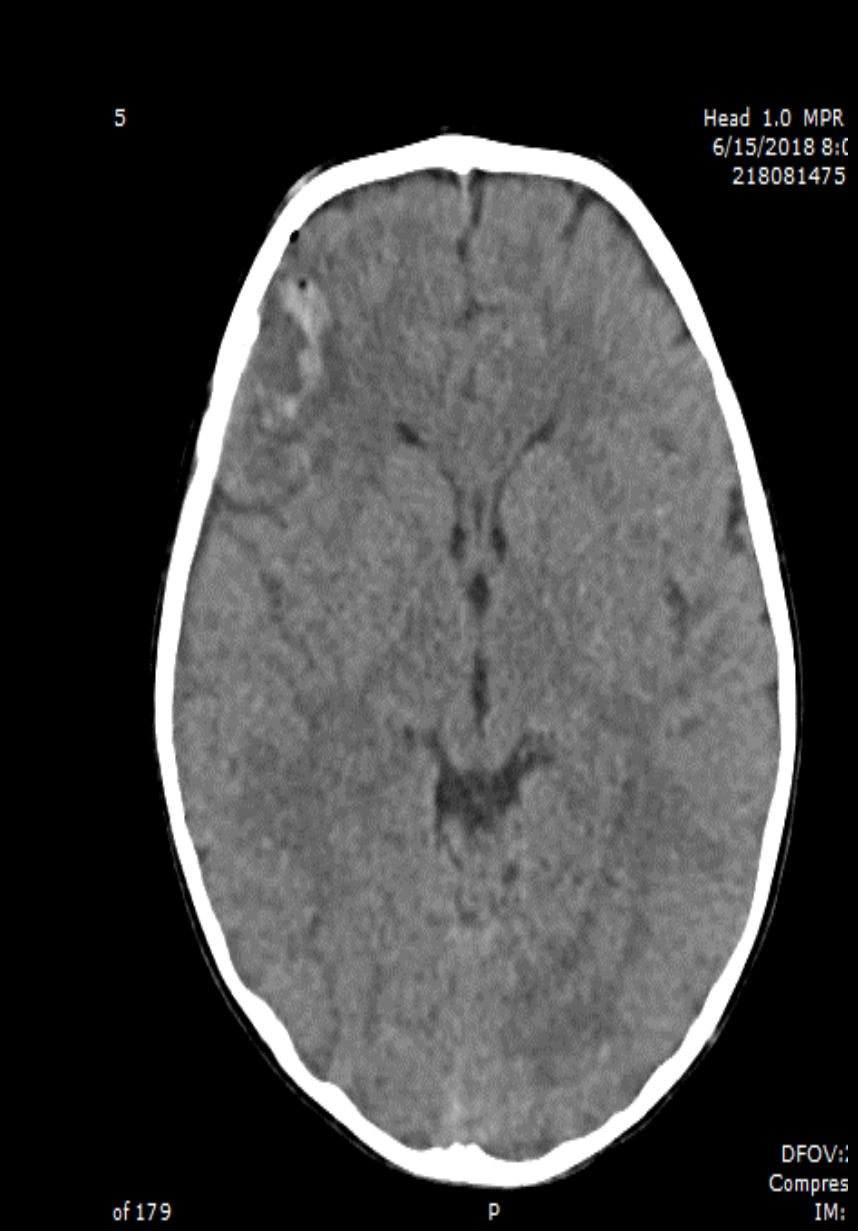

HFS
R
L
RD: 222
Tilt: 0
mA: 143
KVp: 130
Acq no: 3
Page: 66 of 211
Z: 1
C: 35
W: 80
DFOV:22.2x26.3cm
Compressed 11:1
IM: 67 SE: 2
P
cm


A
B

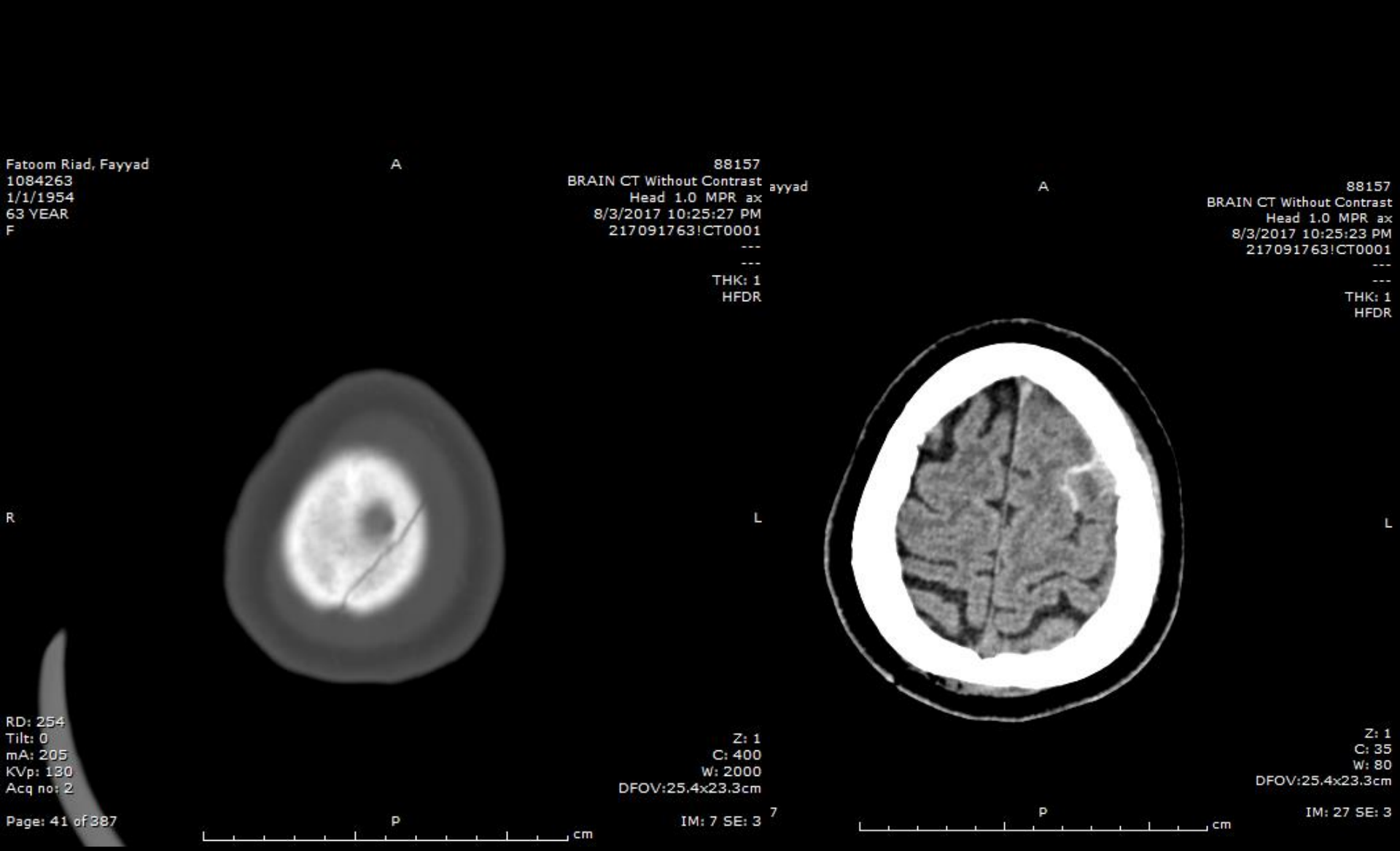

FRONT
UMDS



(a) (b)


5
Head 1.0 MPR
6/15/2018 8:(
218081475
DFOV:
Compres
IM:
of 179
P


5
Head 1.0 MPR
6/15/2018 8:0
218081475!
DFOV:2
Compres
IM:
of 179
P


Fatoom Riad, Fayyad
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BRAIN CT Without Contrast
Head 1.0 MPR ax
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THK: 1
HFDR
ayyad
R
L
RD: 254
Tilt: 0
mA: 205
KVp: 130
Acq no: 2
Page: 41 of 387
P
cm
Z: 1
C: 400
W: 2000
DFOV:25.4x23.3cm
IM: 7 SE: 3
7
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BRAIN CT Without Contrast
Head 1.0 MPR ax
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THK: 1
HFDR
L
P
cm
Z: 1
C: 35
W: 80
DFOV:25.4x23.3cm
IM: 27 SE: 3


THANK YOU