ECMINT 3.1 December 2018 – Cerebral Venous Sinus Thrombosis

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Overview

1. Introduction
2. Epidemiology
3. Anatomy and Pathophysiology
4. Clinical picture and risk factors
5. Diagnosis
6. Treatment
7. Prognosis

1. Introduction

- Occlusion of dural sinuses ± cerebral veins – 0.5-1% of all strokes
- Venous hypertension
- ↓ drainage of blood and CSF
- Swelling – oedema → mild / severe symptoms
- ≈ 50% venous infarcts – some haemorrhagic
- Stroke in young - females

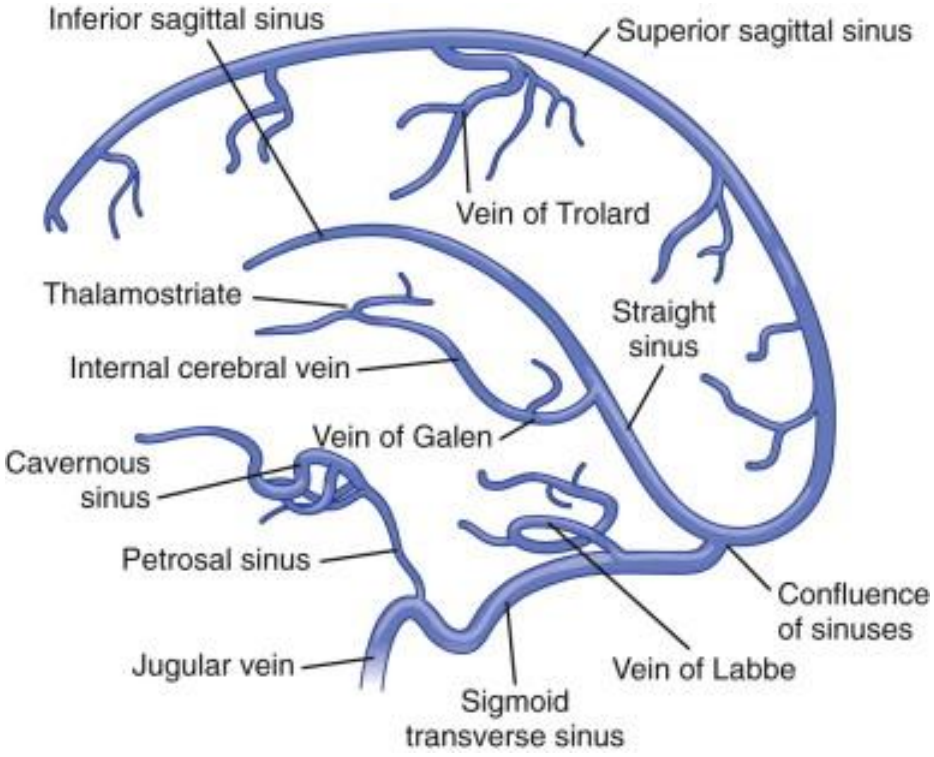
⬇

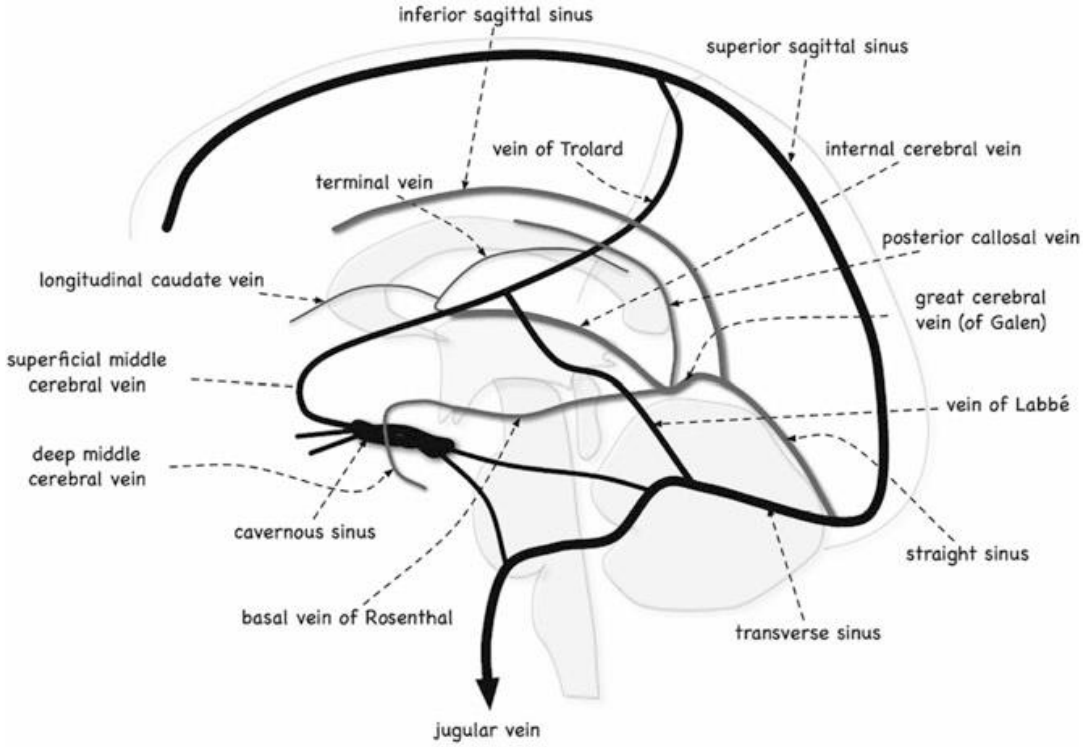
2. Epidemiology

- Incidence 1,3/100.000/year
- Women 31-50yo - 2,8
- Children – 0,7
- Uncommon in elderly – 78% in pts < 50yo

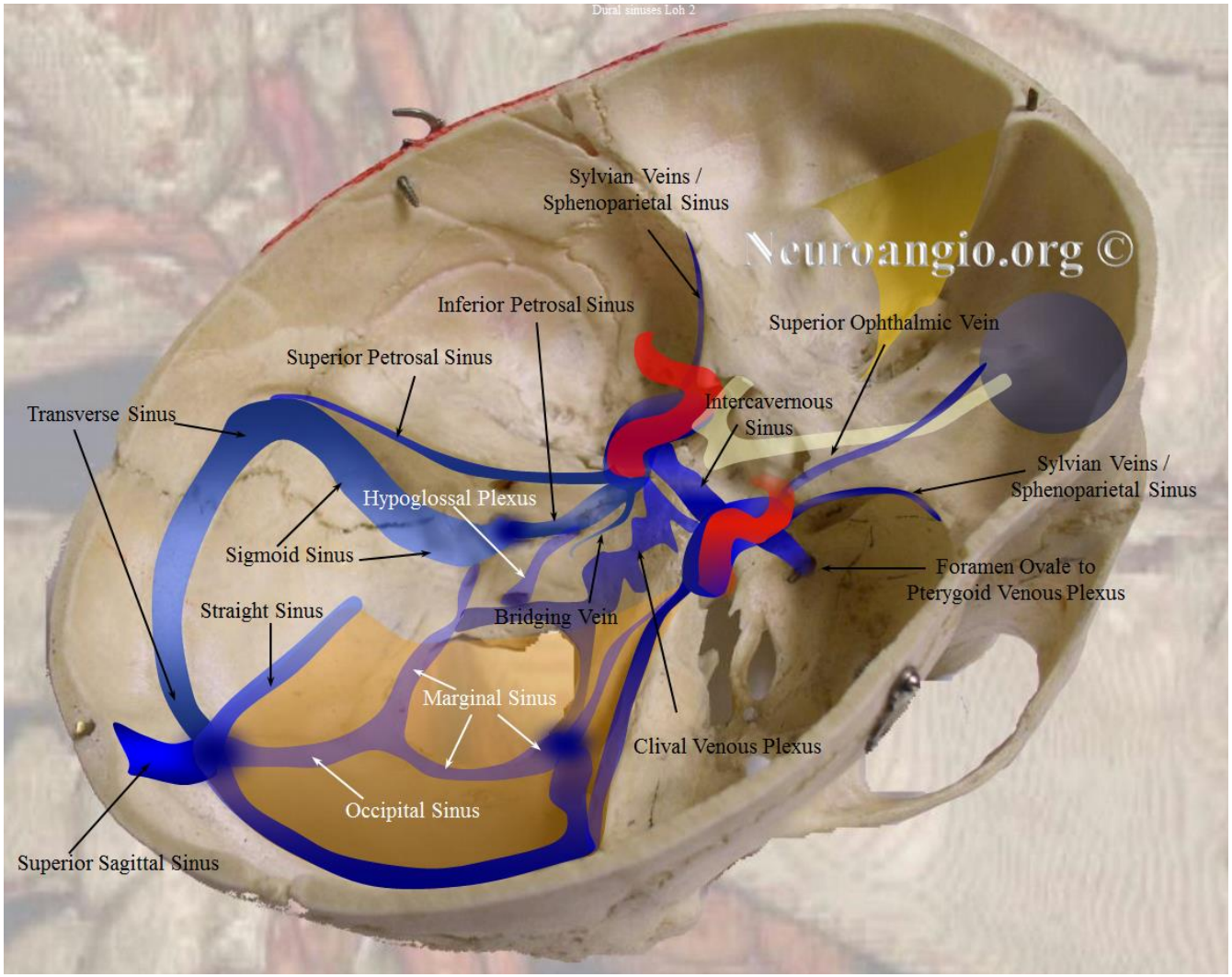
3. Anatomy

Major veins and sinuses





Cerebral venous sinuses



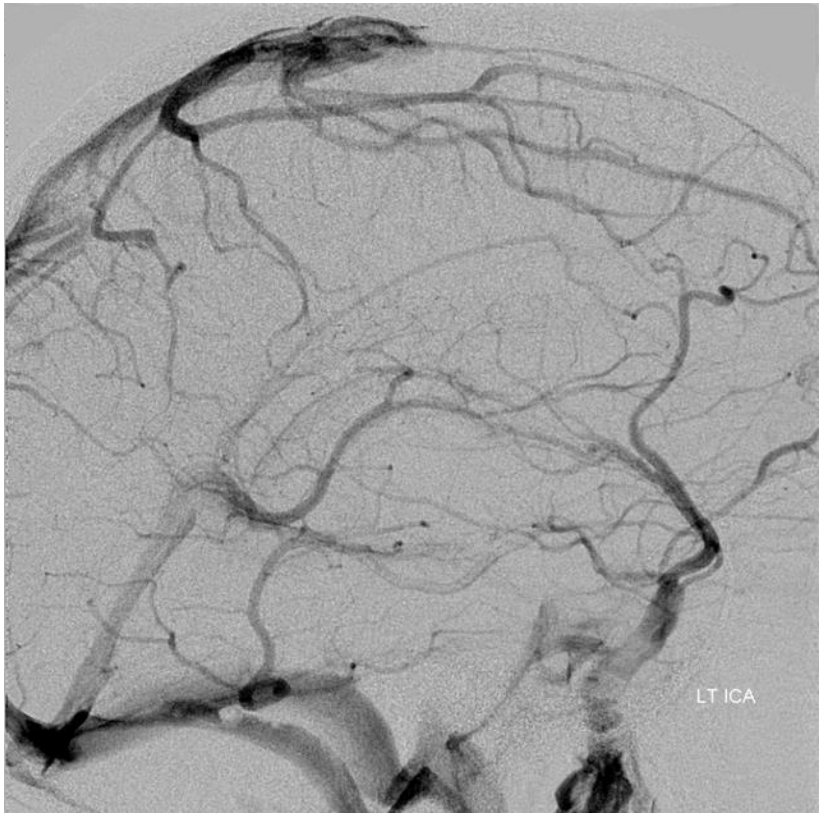
Cerebral venous sinuses

- Transverse sinus
  - R>L- check jugular foramen
- Sigmoid sinus
  - May be larger than TS
- Occipital – marginal sinus(es)
  - More likely present in children

Cerebral venous sinuses

- Straight sinus
  - Vein of Galen → Torcula
- Inferior petrosal sinus
  - From Cav Sin along petrous pyramid → jugular foramen
- Superior petrosal sinus
  - From CS along petrous ridge → sigmoid sinus
- Vein of Labbé
  - Superficial vein - crosses the temporal lobe – connects to the superficial middle cerebral vein → transverse sinus

Cerebral venous sinuses



Vein of Galen

- Short trunk formed by the union of the two internal cerebral veins and basal veins of Rosenthal

- In the quadrigeminal cistern

- Curves backward and upward around the posterior border of the splenium of the corpus callosum → confluence of the inferior saggital sinus and the straight sinus

Basal vein of Rosenthal

- Paired, paramedian veins
- From medial surface of the temporal lobe → posteriorly and medially → lateral to the midbrain through the ambient cistern → drain into the vein of Galen with the internal cerebral veins
- Closely related to the PCA

3. Pathophysiology CVST

Principle 1: Two parts of human venous system:

1. Superficial
2. Deep

1. Cortex + outer WM → Cortical veins → SSS → Transverse → Sigmoid → Jugular / Vein of Labbé → Sigmoid

2. Deep WM + BG → ISS + ICV + BVR → VOG → Straight → Transverse at Torcula

Principle 2: Pathological mechanisms in relation to location

1. Cortical veins: Breakdown of BBB due to ↑ pressure → venous infarction → haemorrhagic → focal deficits/epilepsy

2. Dural sinuses: Restricted outflow CSF/Venous blood → IC hypertension/vasogenic oedema → ICP ↑ - CPP ↓ → headache/↓visual acuity --- → *possibly* ↓ consciousness + herniation

4. Clinical Picture

- Diffuse → delay up to 7 days

- Symptoms
  - Headache (90%) – mostly diffuse – related to venous outflow obstruction / cortical veins → absent
  - Papilledema
  - Focal neurological symptoms: ≈ 50% - related to venous infarct / haemorrhage
  - Seizures: slightly less frequent (37% adults), also related to Vinf / Haem

- "Malignant" development – minority but ≈ 10-15% may present comatose and 9-13% have poor outcomes despite anticoagulation

  - Venous outflow↓ → central oedema /large venous infarcts + decreased level of consciousness → herniation

4. Risk factors

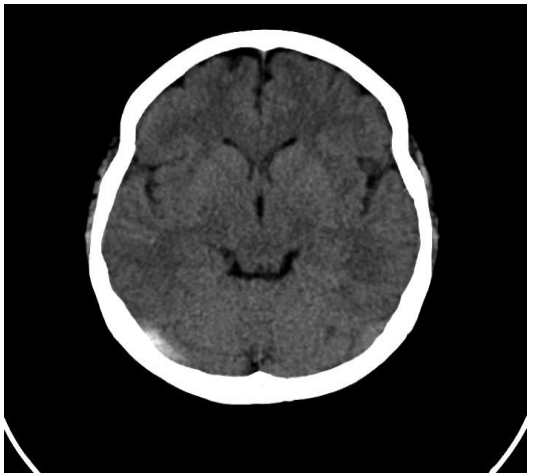
- Young and middle-aged women:
  - Oral contraceptives
  - Hormone therapy
  - Pregnancy/puerperium
- Thrombophilia:
  - Factor-V Leiden
  - Protein-S and –C deficiencies
- H&N infections
- Inflammatory bowel disease / Anaemia

Other causes

- May also include
  - Trauma: Head and Neck
  - Surgery
  - Neoplasms
  - Dehydration
  - …and more…
- Cause not found in 20-30%

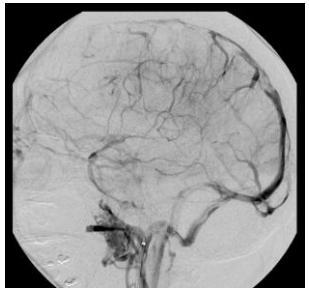
5. Diagnosis

NCCT: 1/3 hyperdense sinus, HFU>70



- NCCT
  - Hyperattenuating thrombus in sinuses and CVs
  - Oedema
  - Venous infarcts and haemorrhages
- MRI
  - Same info by SWI, DWI, T2/FLAIR

- CTV or MRV (2D TOF or Gd+) – confirm diagnosis
- DSA
  - not for diagnosis - treatment

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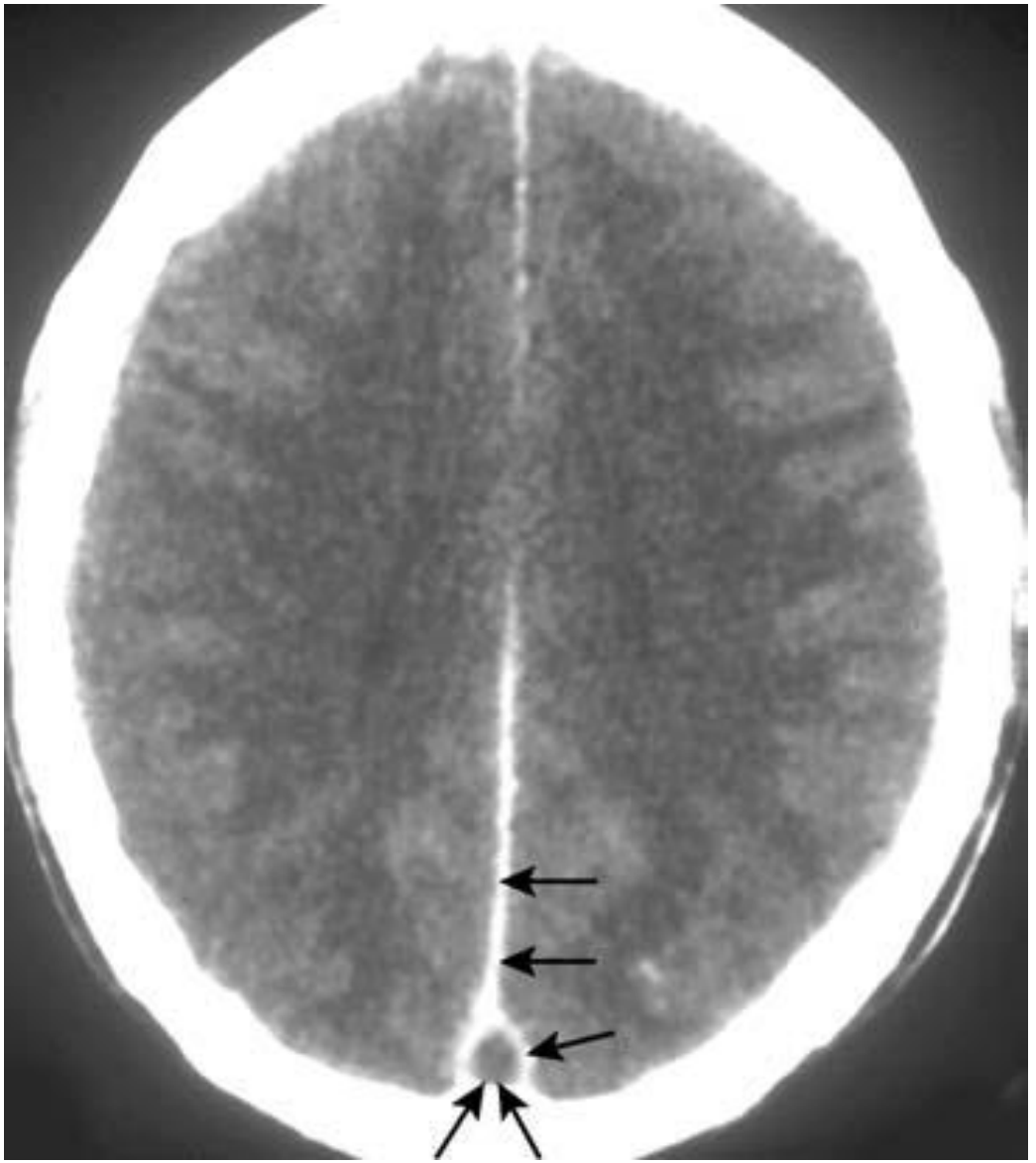
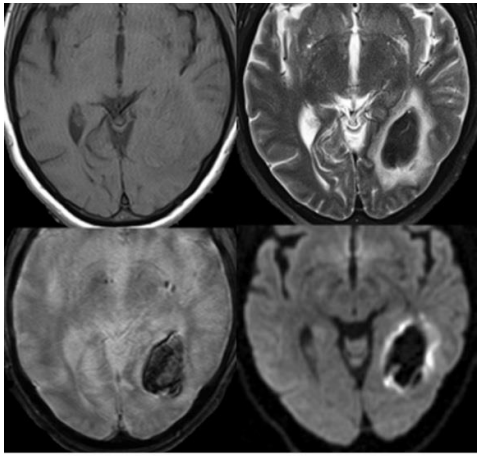


MRI

- Thrombus signal varies according time onset → imaging

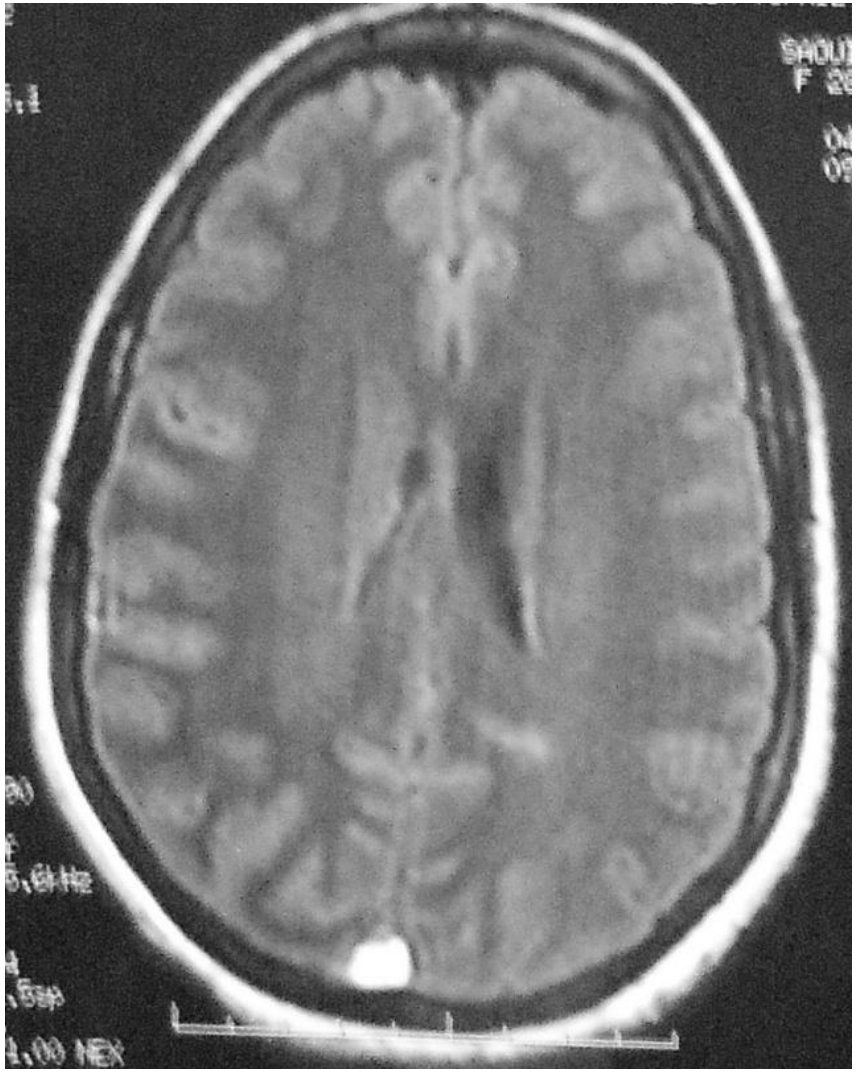
- 1st week: T1 – isointense + T2 – hypointense due to deoxyhemoglobin

- 2nd week: T1+T2 hyperintense due to methemoglobin

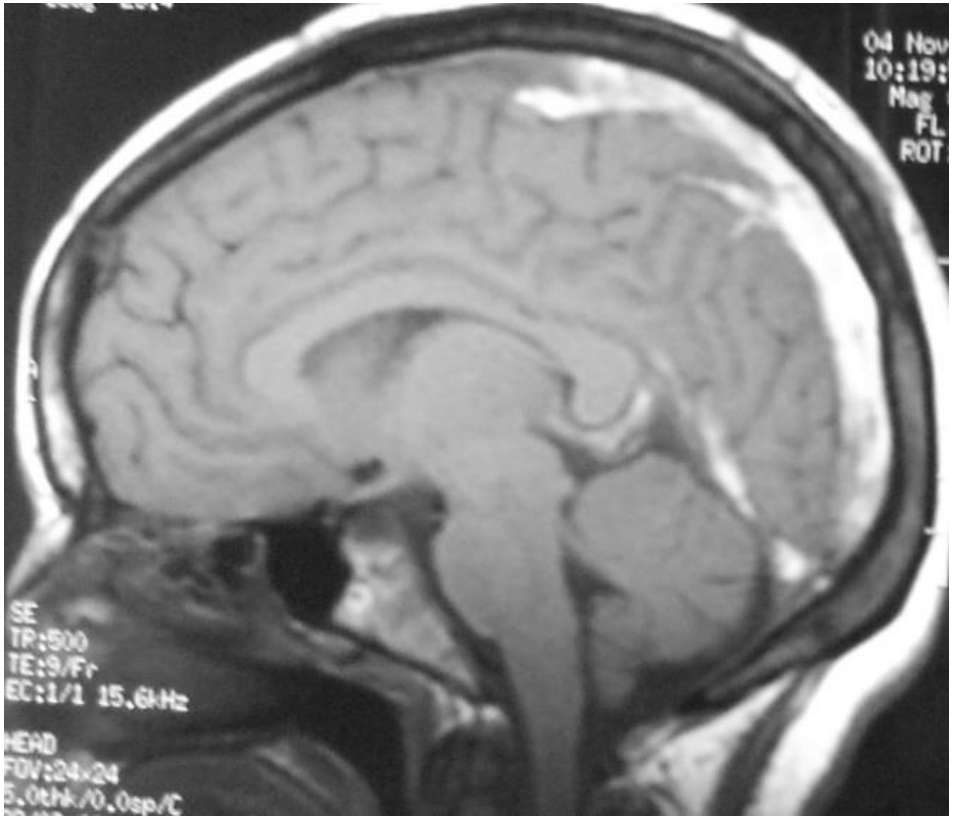


Deoxyhemoglobin

Acute hemorrhage (12 hr - 2 d)
4 unpaired electrons - Paramagnetic
T1 (iso), T2/FLAIR (dark)
GRE (dark), DWI (dark)

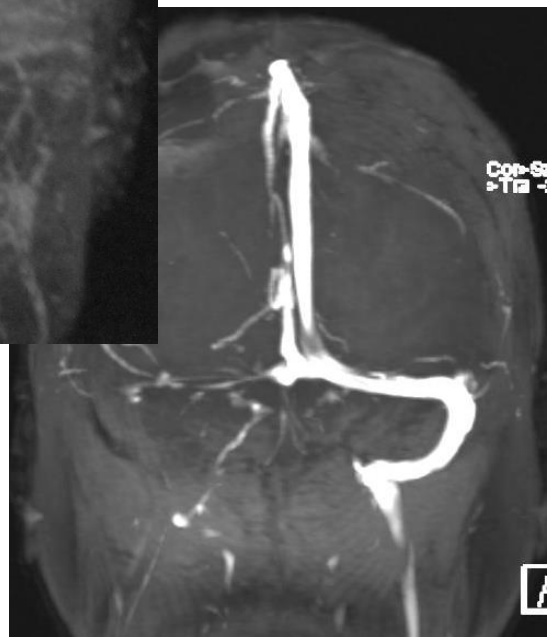


Flair WI Axial



T1 WI sagittal

Diagnosis by CTV/MRV



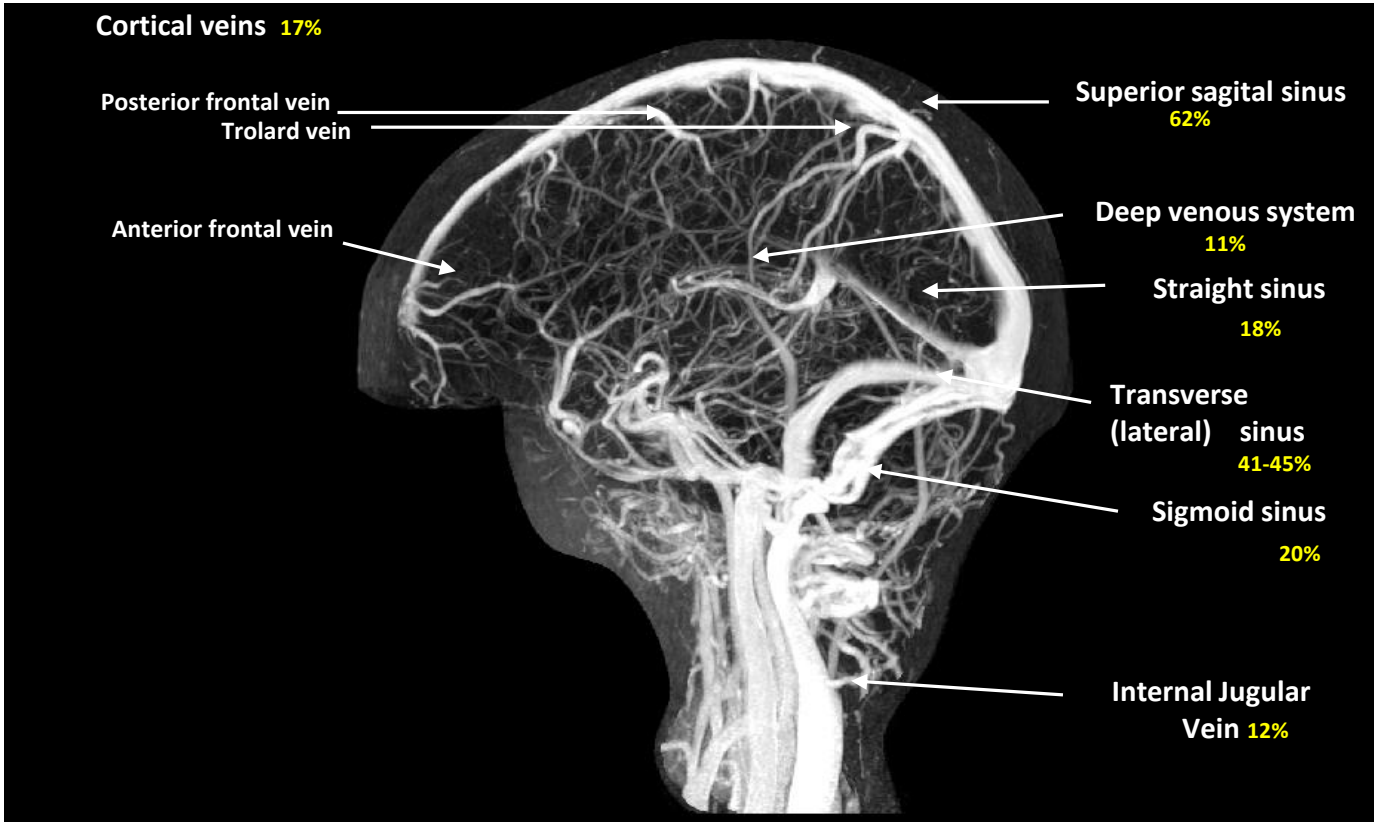
Analyses

- Blood counts / electrolytes
- Extended coagulation panel
  - Prothrombin time
  - Activated partial thromboplastin time
- Screening for thrombophilia
  - Not in the acute phase

Locations

- SSS – affected in ≈ 60%
- TS – ≈ 40%
- SS – ≈ 20%

- Parenchymal lesions (oedema, infarcts, bleeds): ≈ 50%
- Largest infarcts / bleeds in relation to VO Labbé


Cortical veins 17%
Posterior frontal vein
Trolard vein
Anterior frontal vein
Superior sagital sinus
62%
Deep venous system
11%
Straight sinus
18%
Transverse
(lateral) sinus
41-45%
Sigmoid sinus
20%
Internal Jugular
Vein 12%

6. Treatment

- Anticoagulation
  - Unfractionated
  - LMWH
  - Oral anticoagulation in the rehab phase (Coumadin)
    - 6-12 months – sometimes shorter / sometimes indefinite
  - NOACs?

Acute heparinization *does not* increase the haemorrhage risk – may be adm also with a haemorrhagic Vinf

EVT - Surgery

Some neurologic worsening may occur in ≈ 20-25% of the patients

EVT: Pts that deteriorate despite medical therapy (≈10%)

LOC

Focal neurological symptoms

- rt-PA or Urokinase to create a channel
- Continuous IC infusion (≈ 1mg/hr) via retained MC: ICU, control of fibrinogen, level of consciousness, papilledema

- ICH occurred in 17% of patients after thrombolysis - associated with clinical worsening in 5%
- Mechanical thrombectomy – aspiration - ≈ 50 % success
- Hemicraniectomy

Adjunctives

- Anti-epileptics – All patients? - Kept 6-12 months
- EVD only if acute obstructive HC due to oedema
  - Risk because of need for heparin
  - Risk for increased swelling?
- Lumbar drain?
- Acetazolamide – limited effect

7. Prognosis

- Majority recover – obstruction recanalize
- ISCVT: Complete recovery (Md 16 mo) 79% - (57% full recovery?) – HA may persist – back to work?
- ≈ 10% recurrence
- Mortality 5-15% - higher with older age – ISCVT 8.3% over 16 months
- ISCVT: 79% mRS 0-1, 10.4% mRS 2-3 and 2.2% mRS 4-5
- Negative predictors
  - Male gender
  - Older age (>37!)
  - Thrombosis of deep venous system
  - ICH on admission
  - GCS <9 – "mental status disturbance"
  - CNS infection
  - Malignancy

International Study on Cerebral Venous and Dural Sinuses Thrombosis

Ferro JM et al, *Stroke.* 2004;35(3):664-670

Two groups of CVT patients

Group 1

- Majority
- "Limited" symptomatology
- Recover on anticoag
- Good venous back-up

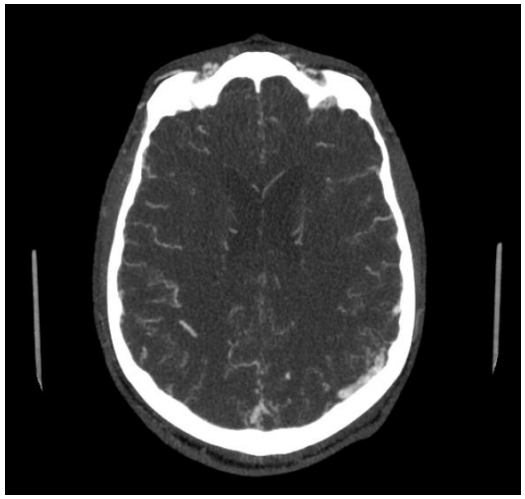
Group 2

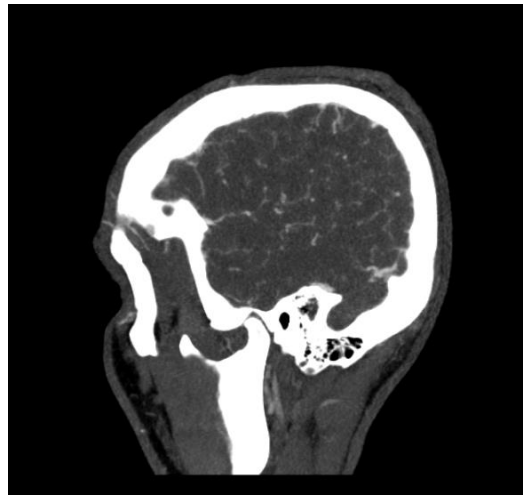
- Minority
- Deteriorate acutely
- ICU
- Advanced therapy
- Severe clinical impairment

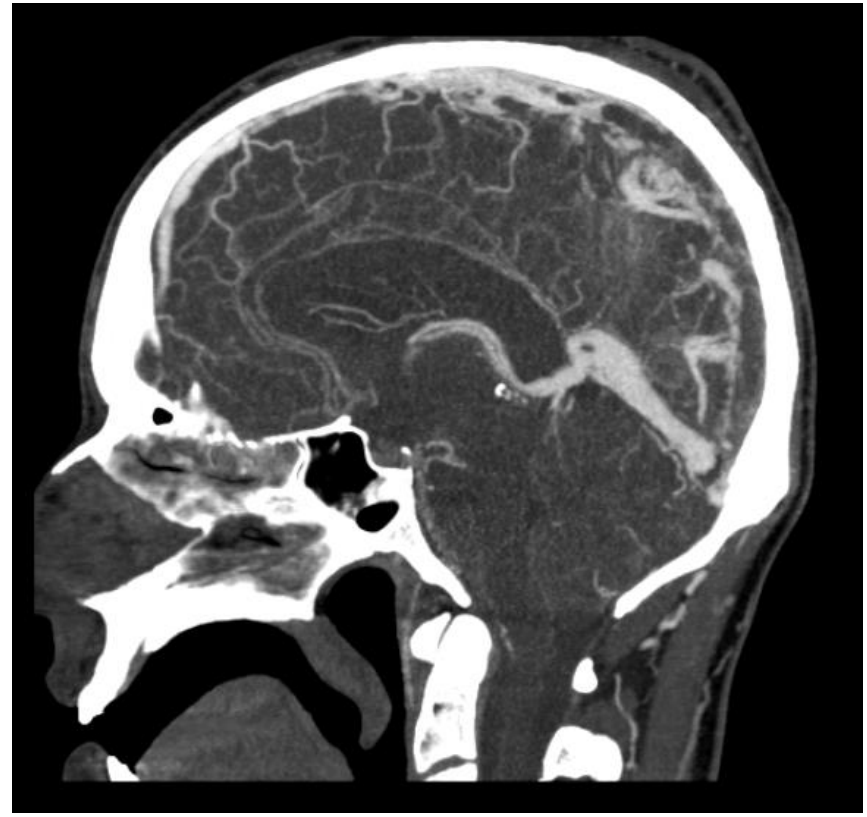
Key point to find patients at risk – "Group 2!"

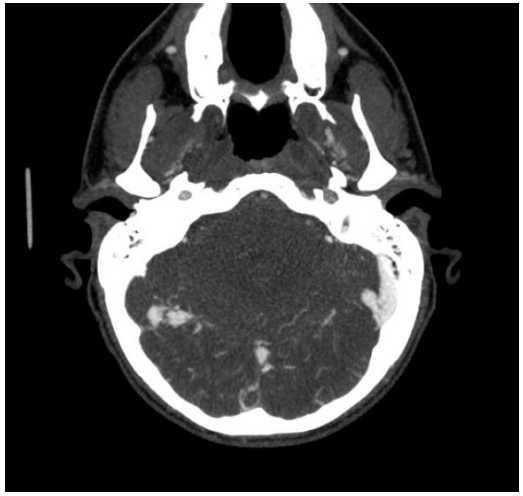
33 yo male

- Healthy (Protein-C deficiency)
- Physical activity – neck trauma R
- w/u for trauma
- Headache
- Seizure

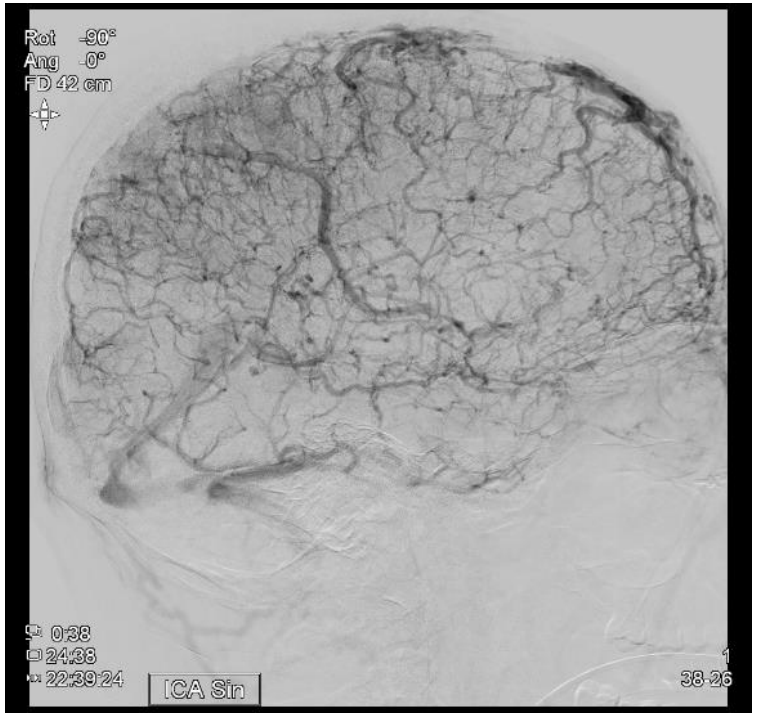









Rot -90°
Ang -0°
FD 42 cm
0:38
10:38
22:39:24
ICA Sin
38-17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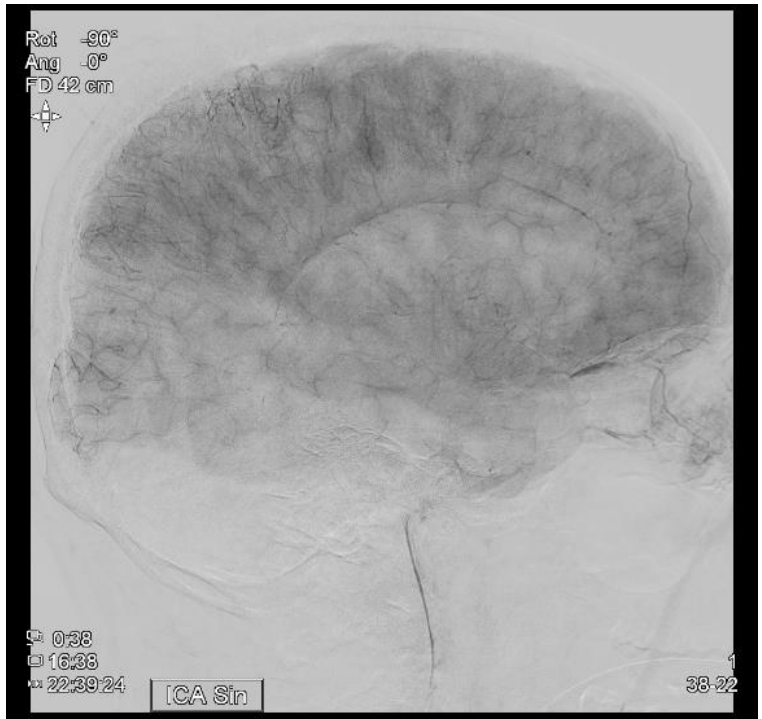
ICA Sin
38-26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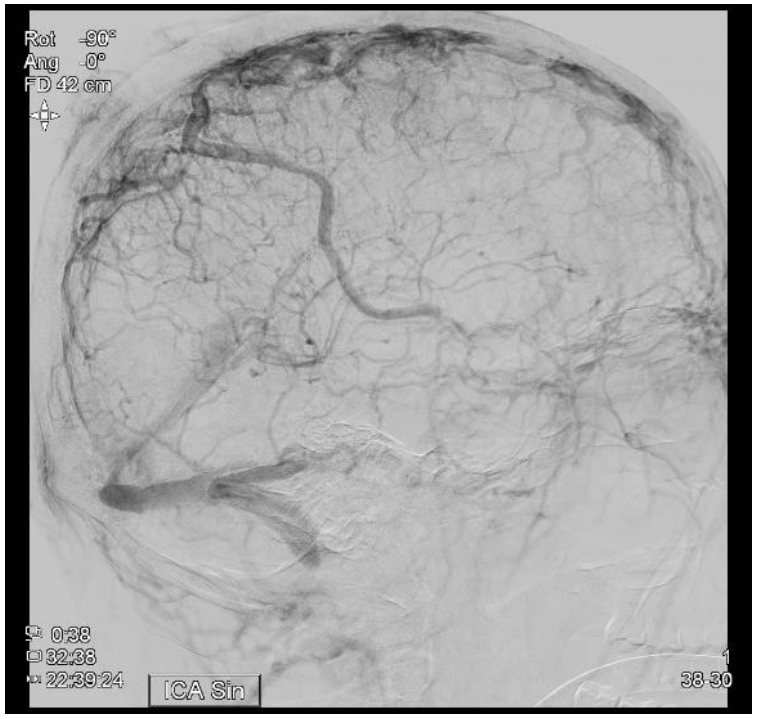
ICA Sin
38-22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32:38
22:39:24
ICA Sin
38-30


Rot +0°
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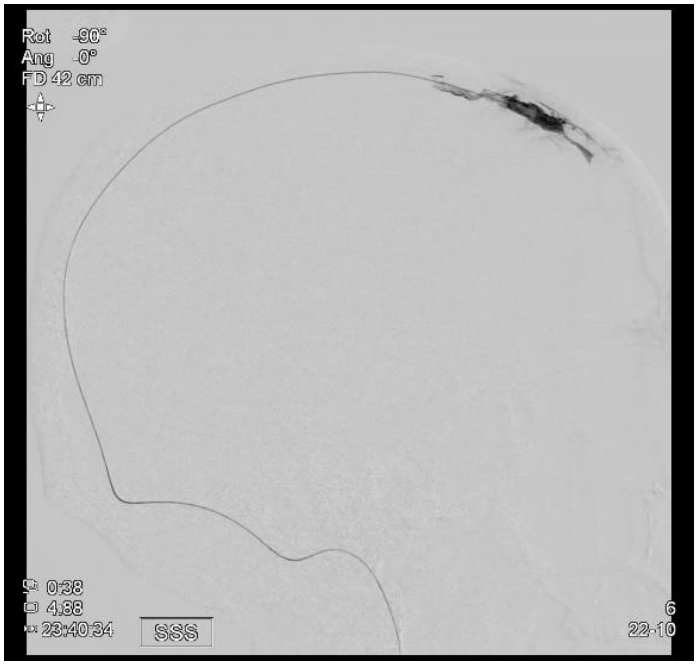
v jugularis int dx
2
2


Rot -90°
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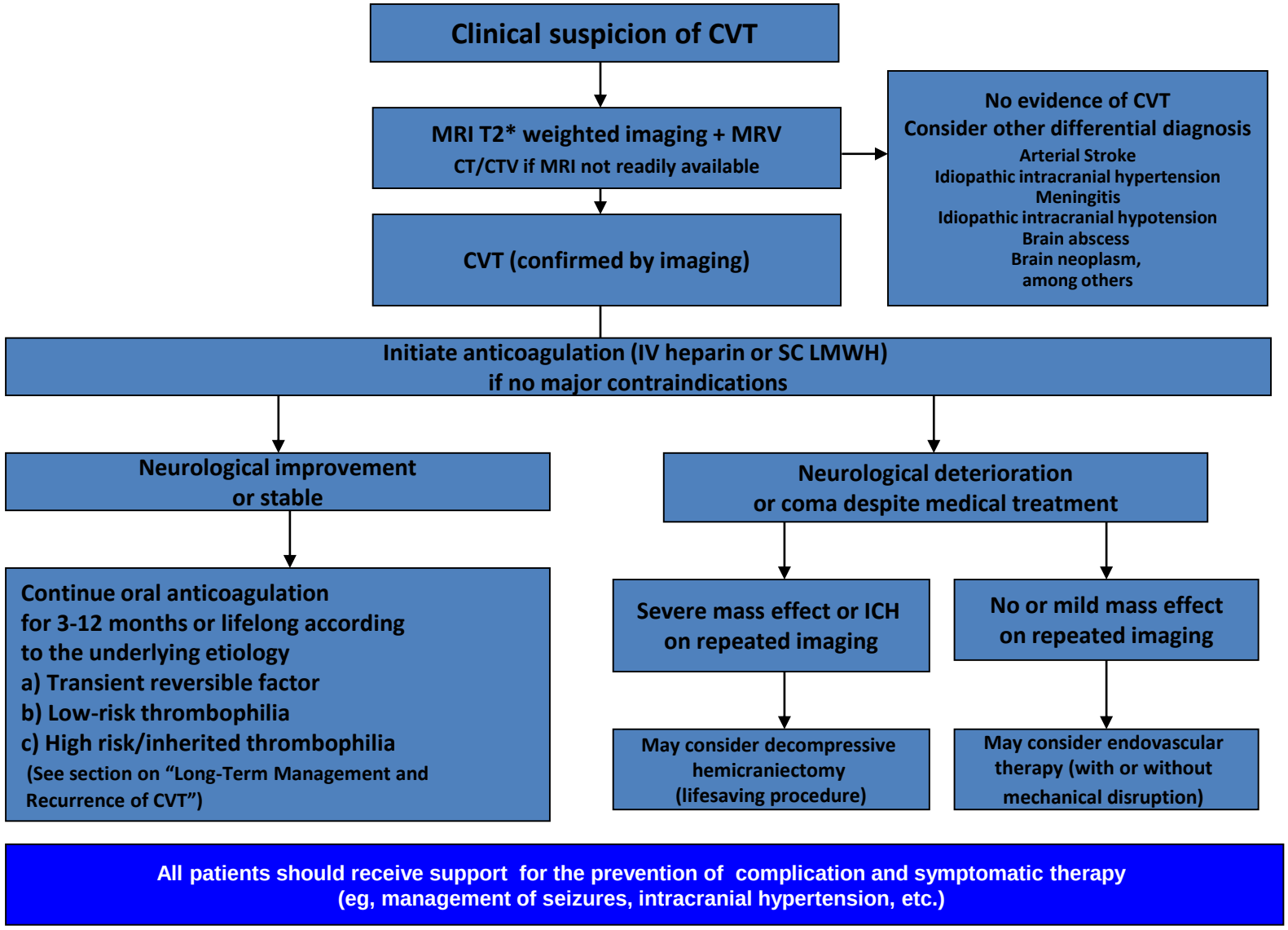
0:38
4.88
23:40:34
SSS
6
22-10


Rot -90°
Ang -0°
FD 42 cm
7:38
23:40:34
SSS
6
14

American Heart Association
Proposed Algorithm for the Management of CVT



Thank you!!!