CASE CONFERENCE

Extern
Natwara Khonsoong

IDENTIFICATION DATA

- male 33 years old
- เชื้อชาติ พม่า
- Visit date 27/10/63

Chif complaint

ชักเกร็ง 2 ชั่วโมง ก่อนมาโรงพยาบาล

Triage

EMERGENCY

Primary Survey

Primary survey

- A : can talk , no stridor , no hoarseness , no secretion or foreign body , can active neck flexion
- B : RR 20/min , spO2 98 % RA , clear and equal breath sound both lungs , symmetrical chest movement , trachea in midline
- C : BP 134/94 mmHg , PR 106 bpm , no external bleeding , no abdominal tenderness
- D: E4V5M6, pupil 3 mm RTLBE

Adjunct to primary survey

- - CBG = 134 mg %

Secondary Survey

Secondary survey

- **A:** No history of drug or food allergy
- **M**: No current medication
- **P**: No Underlying disease
- Chronic alcohol drinking ดื่มสุราทุกวันวันละ 1 ขวด มีประวัติดื่มสุราล่าสุด 20.00 น.1วันก่อนมาโรงพยาบาล
- ไม่สูบบุหรี่
- **L**: Last meal 3 hour PTA
- **E**: ชักเกร็ง 2 hr PTA

EVENT

2 ชั่วโมงก่อนมาโรงพยาบาล (09.50 น.) ขณะทำงานอยู่เพื่อนร่วมงานเห็นชักเกร็งทั้งตัวไม่กระตุกประมาณ 2 นาทีแล้วล้มลง ตาเหลือก เรียกไม่รู้สึกตัวขณะชัก ศีรษะไม่กระแทก หลังจากนั้นหยุดชักเอง หลังชักซึม at ER รพช. ตื่นรู้ตัว ถามตอบได้ ขณะอยู่ที่ ER รพช. ผู้ป่วยมีชักเกร็งกระตุกทั้งตัวประมาณ 2 นาที จากนั้นหยุดเอง ถามตอบรู้เรื่อง มีปวดศีรษะ ไม่มีคลื่นไส้อาเจียน ไม่เคยชักมาก่อน ไม่มีไข้ ไม่ไอเสมหะ ไม่มีท้องเสีย ไม่มีอ่อนแรง ไม่ชา ไม่มีปากเบี้ยวหน้าเบี้ยว ไม่เจ็บอก/ใจสั่น
At ER CRH มีชักซ้ำแขนซ้ายกระตุก ประมาณ 4 นาที มีทั้งช่วงที่เรียกรู้ตัวและเรียกไม่รู้สึกตัวตาลอย

Physical examination

Vital sign : BT 37.0 °C PR = 106 bpm RR = 20 /min BP = 134/94mmHg
GA : A man with good consciousness
HEENT : no pale conjunctiva, no icteric sclera, no lateral tongue bite
Lymph node : can't be palpated
Skin : no rash , no petechiae
CVS : regular rhythm, normal S1S2, no murmur
RS : clear&equal breath sound both lungs, no adventitious sound
Abdomen : soft, not tender, normoactive bowel sound
Neuro : E4V5M6,pupil 2 mm RTLBE, Motor power grade V all except left arm grade IV, Decrease sensation at left arm, no facial palsy ,Reflex 2+ all , Left pronator drift, BBK negative, Finger to nose not sway,
Extermities : CRT <2 sec , pulse full , no pitting edema

Problem Lists

Differential diagnosis

Adjunct to secondary survey

- **CBC**

ผลวันที่ 27/10/2563				
WBC count	5000 - 10000	7300	cell/cu.mm	
Hb	12.9 - 17.1	17.9	g/dL	
Hct	40 - 50	54.7	%	
Neutrophil	55 - 65	79.4	%	
Eosinophil	1 - 3	0.1	%	
Basophil	0 - 1	0.0	%	
Lymphocyte	25 - 35	13.0	%	
Monocyte	2 - 7	7.5	%	
Platelet	#	Adequate		
Platelet count	140000 - 400000	148000	cell/cu.mm	
RBC	4.3 - 6.1	6.03	M/ul	
MCV	80 - 100	90.8	fl	
Slide NO.	#	2187		
RDW	11.2 - 14.8	16.5	%	
MCH	26 - 33	29.7	pg	
MCHC	31 - 36	32.7	g/dL	

Adjunct to secondary survey

- **PT,PTT,INR**

ผลวันที่ 27/10/2563			
PT	9.2 - 13.1	11.5	sec
Control	#	11.1	sec
INR	#	1.03	
PTT	23.2 - 36.9	32.5	sec
CONTROL	#	29.8	sec

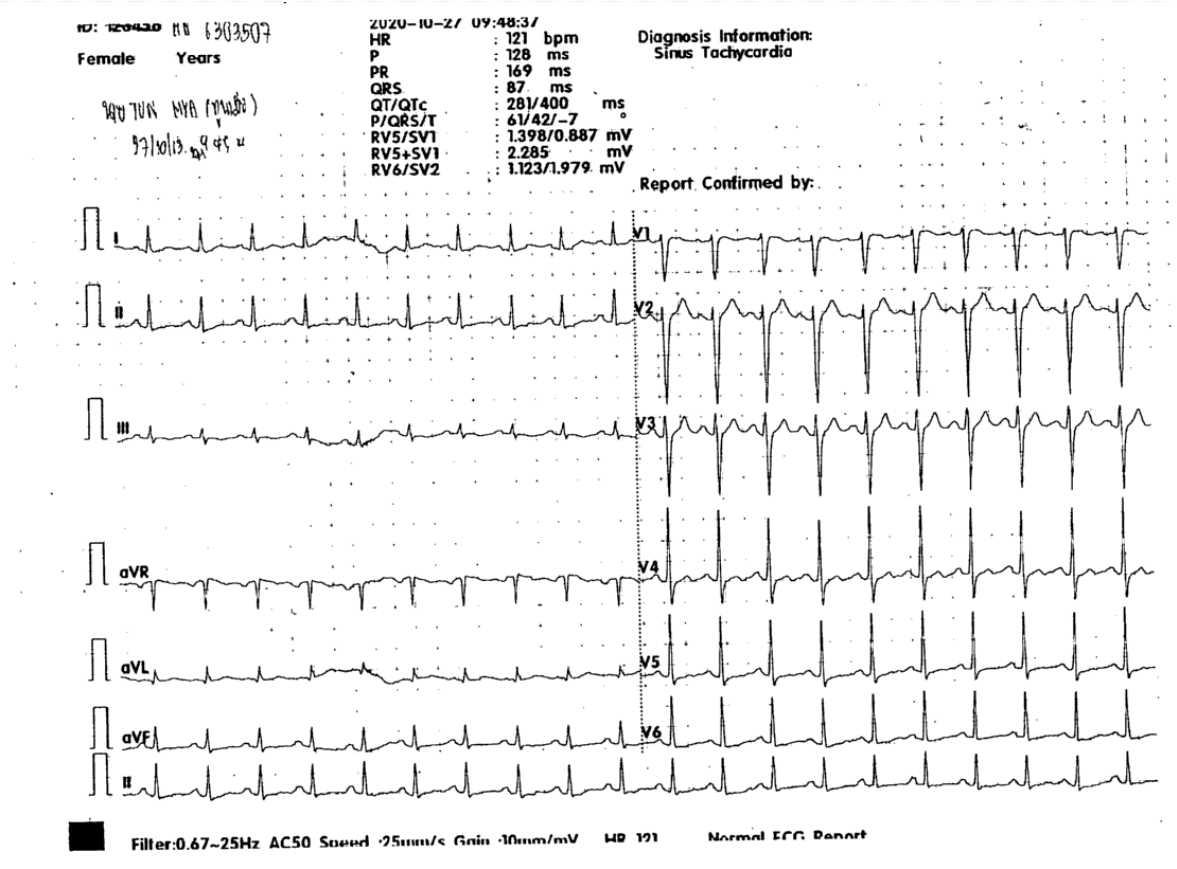
Adjunct to secondary survey

- BUN,Cr
- Electrolyte
- Ca,Mg,PO4
- LFT

ผลวันที่ 27/10/2563			
ALK.PHOSPHATASE	30 - 120	102	U/L
ALT(SGPT)	0 - 55	48	U/L
AST(SGOT)	5 - 34	46	U/L
ALBUMIN	3.5 - 5.2	3.8	g/dl
BUN	8.9 - 20.6	9	mg/dl
CHLORIDE(CL)	101 - 109	106	mmol/l
CARBONDIOXIDE(CO2)	21 - 31	25	mmol/l
CREATININE	0.78 - 1.18	1.09	mg/dl
CALCIUM	8.8 - 10.6	8.8	mg/dl
DIRECT BILIRUBIN	0 - 0.2	0.4	mg/dl
GLOBULIN	#	2.8	gm/dl
POTASSIUM(K)	3.5 - 5.1	3.6	mmol/l
MAGNESIUM	1.6 - 2.6	1.5	mg/dl
SODIUM(NA)	136 - 146	141	mmol/l
PHOSPHOUROUS(PO4)	2.5 - 4.5	1.8	mg/dl
TOTAL BILIRUBIN	0.3 - 1.2	1.3	mg/dl
TOTAL PROTEIN	6.6 - 8.3	6.6	gm/dl
eGFR	60 - 120	89	

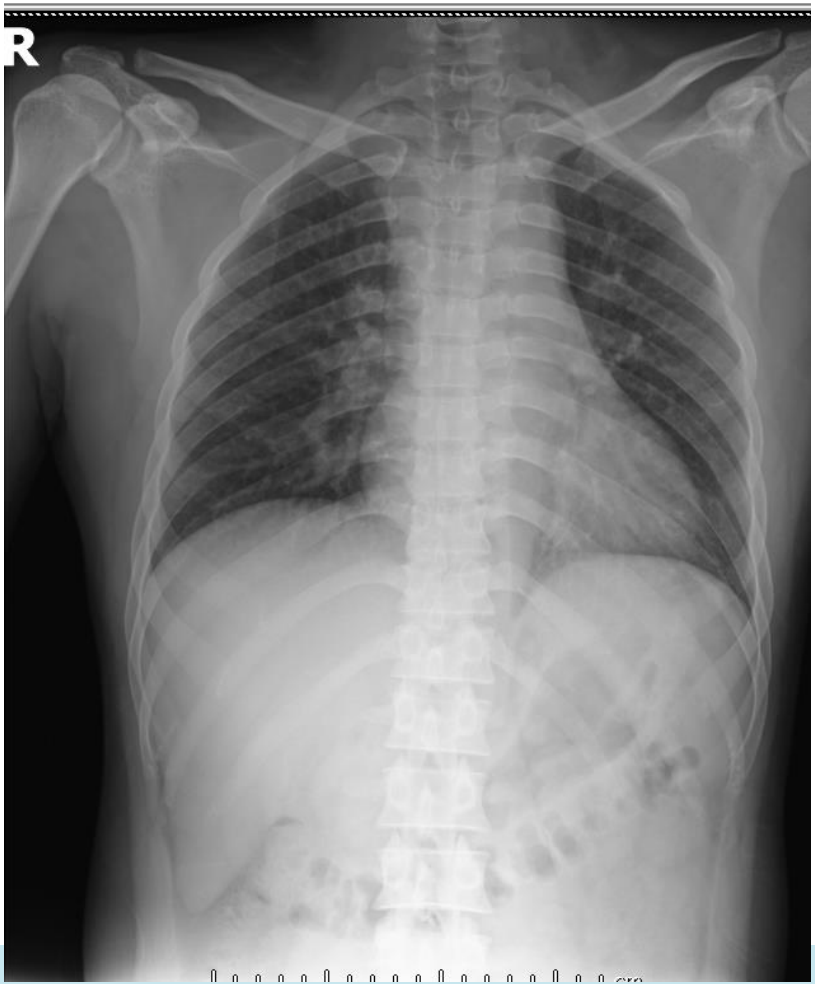
Adjunct to secondary survey

- **EKG 12 leads**



Adjunct to secondary survey

- CXR



Adjunct to secondary survey

- **CT brain NC**

CT BRAIN: Plain axial cranial MDCT scan.
HISTORY: Case 1st episode seizure
FINDINGS: Limitation of the study due to noncontrast enhanced study. The study shows
- Hyperdensity of the cortical veins in bilateral frontal and right parietal lobes and anterior superior sagittal sinus. Faint hypodense area with effacement of the cortical sulci of the right parietal lobe, compatible with brain edema.
- The remaining visualized brain parenchyma is unremarkable.
- No hydrocephalus is seen. There is no shift of the midline structures.
- The PNS and bilateral mastoid air cells are clear. The bony calvaria is intact.
IMPRESSION:
- Hyperdensity of the cortical veins in bilateral frontal and right parietal lobes and anterior superior sagittal sinus with focal brain edema at right parietal lobe, dural venous sinus thrombosis is suspected. Please correlate with clinical context and CTV brain is suggested.

Adjunct to secondary survey

- ## CTV brain

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CTV BRAIN
FINDINGS: The study shows
- Filling defect in superior sagittal sinus and cortical veins.
- Vasogenic brain edema in with a 0.8 cm hematoma at right high parietal lobe.
- The remaining visualized brain parenchyma is unremarkable.
- No hydrocephalus is seen. There is no shift of the midline structures.
- The PNS and bilateral mastoid air cells are clear. The bony calvaria is intact.
- The remaining brain parenchyma is unremarkable. No brain herniation, hydrocephalus or shifting of midline structures.
- Intact bony calvaria.
- Both orbits appear normal.
IMPRESSION:
-Thrombosis in cortical vein and superior sagittal sinus. Venous infarction and hemorrhage in right pareital lobe.
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Difinitive diagnosis

CEREBRAL VENOUS SINUS THROMBOSIS

Management at ER

- at ER ผู้ป่วยมีชักซ้ำแขนซ้ายกระตุก ประมาณ 4 นาที
- ได้ valium 10 mg IV

Investigation

- protein c , protein s
- Antithrombin III
- Anti lupus anti coagulant
- Beta2 glycoprotein

} pending

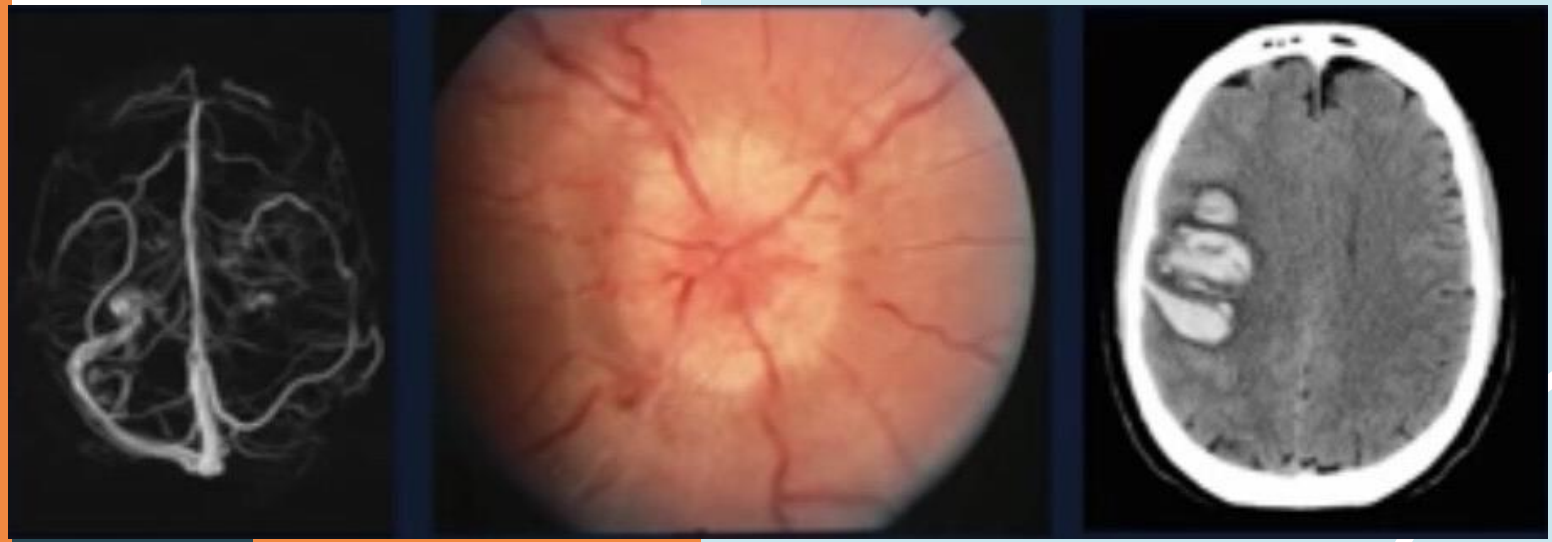
TREATMENT

- Enoxaparin 0.6 ml SC q 12 hr
- Bridging to warfarin (3) 1x1 PO PC (Total week dose = 21 mg/wk)

Progression

- Motor power grade V all except left arm grade IV, Decrease sensation at left arm

CEREBRAL VENOUS SINUS THROMBOSIS

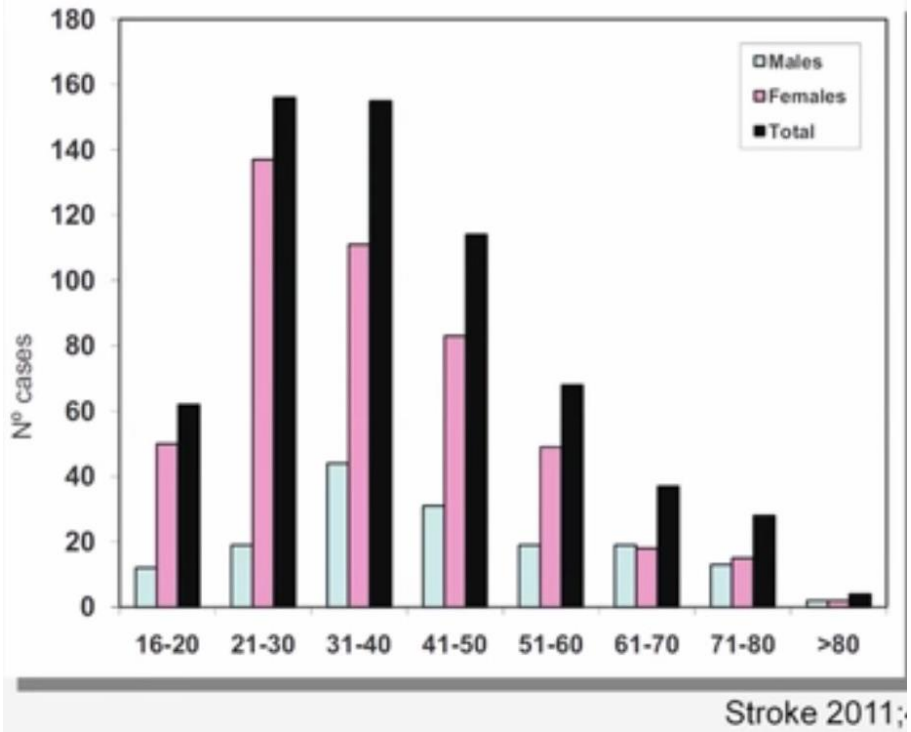


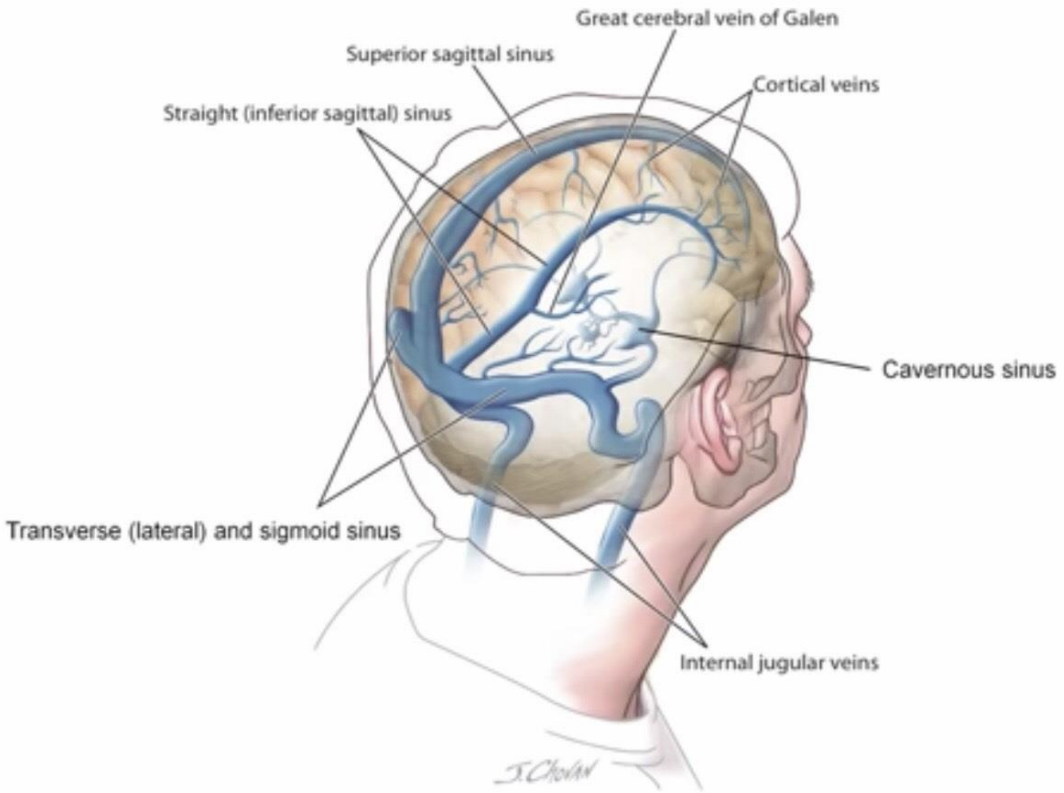
Overview

- Cerebral venous thrombosis, including thrombosis of cerebral veins and major dural sinuses, is an uncommon disorder in the general population

- However, it has a higher frequency among patients younger than 40 years old, patients with thrombophilia, and women who are pregnant or receiving hormonal contraception.

Cerebral Venous Thrombosis: Age and Sex Distribution





http://patientblog.clotconnect.org/2011/02/07/sinus-and-cerebral-vein-thrombosis/

Clinical features

- Young to middle age Pt + secondary headache + stroke like symptom +/- seizure + No vascular risk factor

TABLE 1: Signs and Symptoms of Cerebral Venous Thrombosis

Presentation	Frequency (%)
Headache	75
Papilledema	49
Seizures	37
Motor or sensory deficit	34
Mental status changes	30
Dysphasia	12
Cranial nerve palsies	12
Cerebellar incoordination	3
Bilateral or alternating cortical signs	3
Nystagmus	2
Hearing loss	2

Note—Percentages total > 100% because patients may have multiple presentations.
Adapted from [1].

Risk Factors

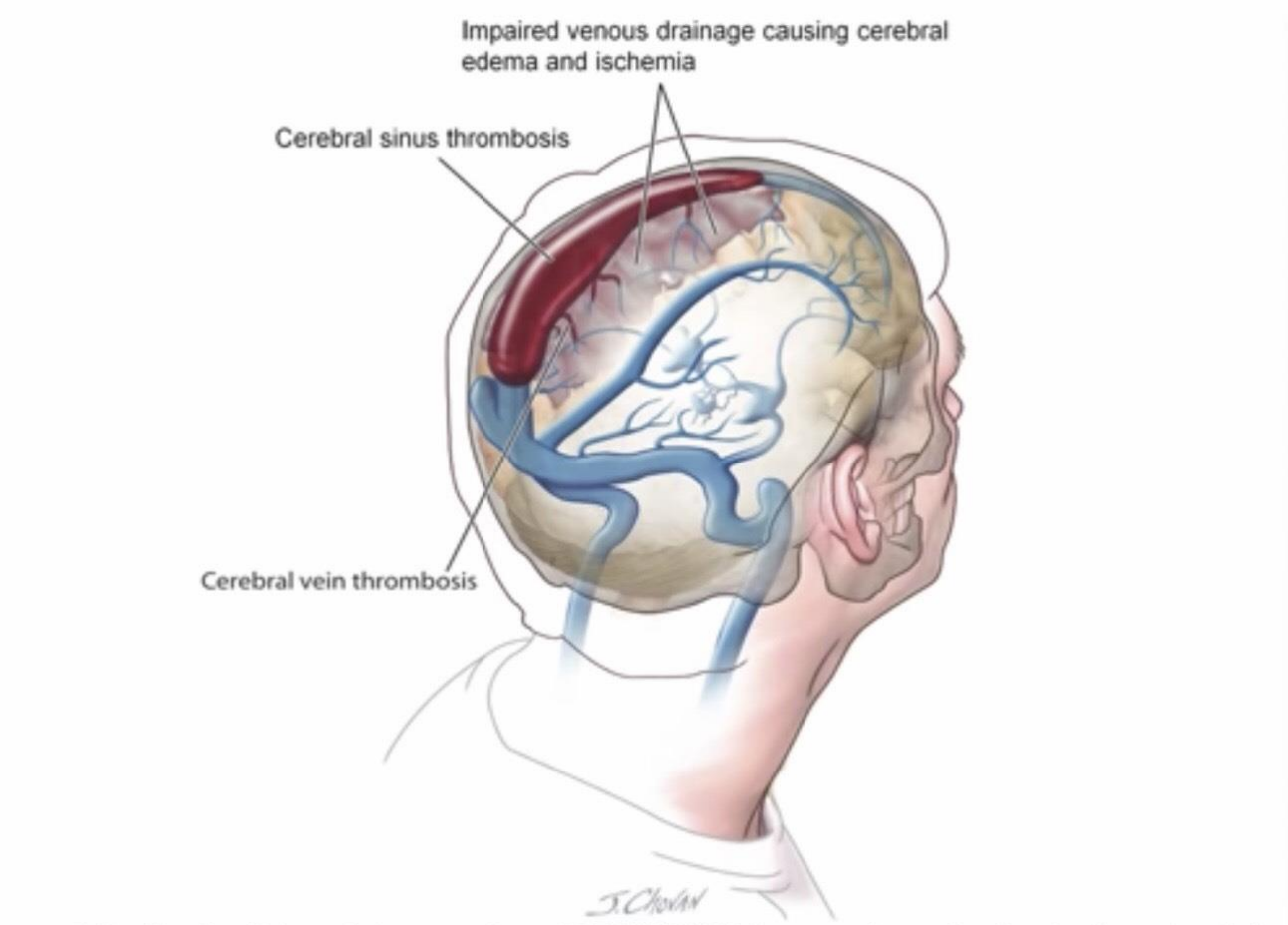
Thrombophilia	• Deficiencies of antithrombin,protein C and protein S • Factor V Leiden/prothrombin gene mutation 20210 • Antiphospholipid antibodies
Women's Health	• Pregnancy • Postpartum state • Hormonal contraception
Infection and Inflammation	• Localized (otitis,sinusitis,meningitis) and systemic infections • Vasculitis • Inflammatory bowel disease
Hematologic – Oncologic Disorders	• Cancer • Polycythemia/essential thrombocytosis • Paroxysmal nocturnal hemoglobinuria
Trauma	• Head trauma/neurological procedures • Local injury to the jugular vein or cerebral sinuses/veins • Lumbar puncture
Other	• Nephrotic syndrome

Pathophysiology

- 2 Major pathophysiologic mechanisms contribute to the clinical presentation of cerebral venous thrombosis

1.) Increased venular and capillary pressure

2.) Decreased cerebrospinal fluid absorption



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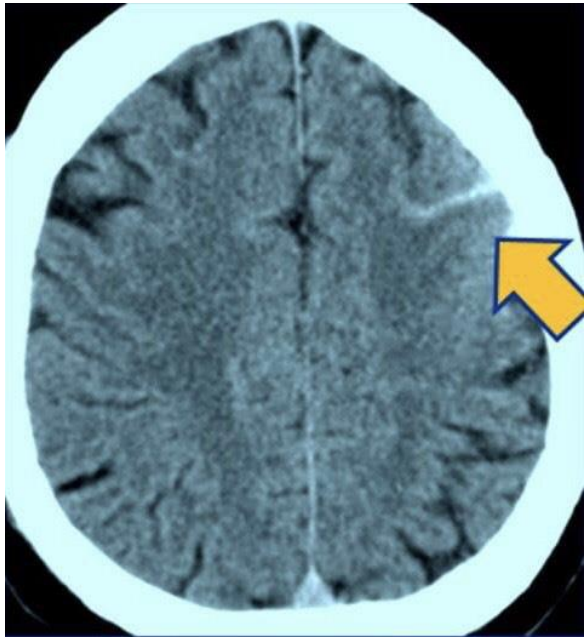
4 Major Clinical Syndromes

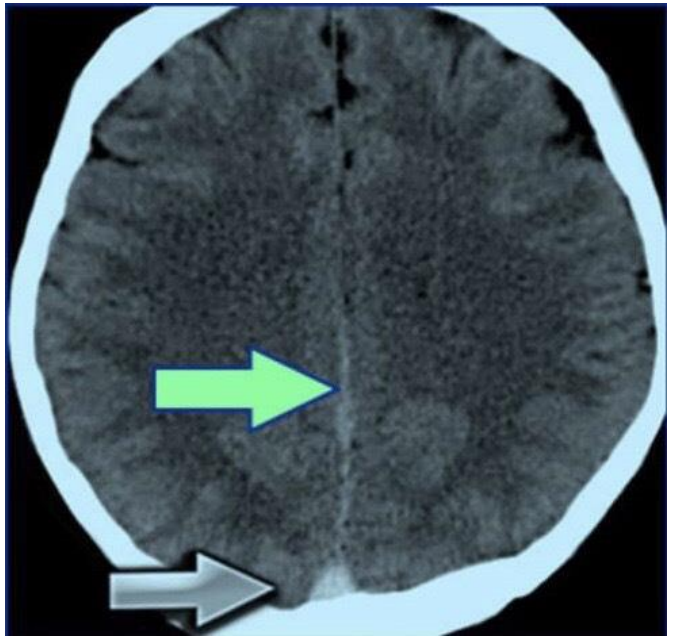
Intracranial Hypertension	• Presents as headache (90%): frequently misdiagnosed as migraine • Headache maybe generalized or localized and may worsen with Valsava or position change. • Other findings include papilledema and visual complaints
Focal Deficits	• Focal neurological deficits are noted in 44% of patients • Motor weakness (hemiparesis) is the most common finding • Fluent aphasia may result from left transverse sinus thrombosis
Seizures	• Focal or generalized seizures are observed in 30-40% of patients • Cerebral venous sinus thrombosis should be considered in any patient with seizures and other focal findings consistent with stroke • Most commonly noted in sagittal sinus and cortical vein thrombosis
Encephalopathy	• Often results from thrombosis of the straight sinus and branches • May result from any cerebral venous thrombosis with cerebral edema, venous infarction, or parenchymal hemorrhage leading to herniation • More common in elderly patients

Non contrast CT

Dense clot sign : 25% of cases

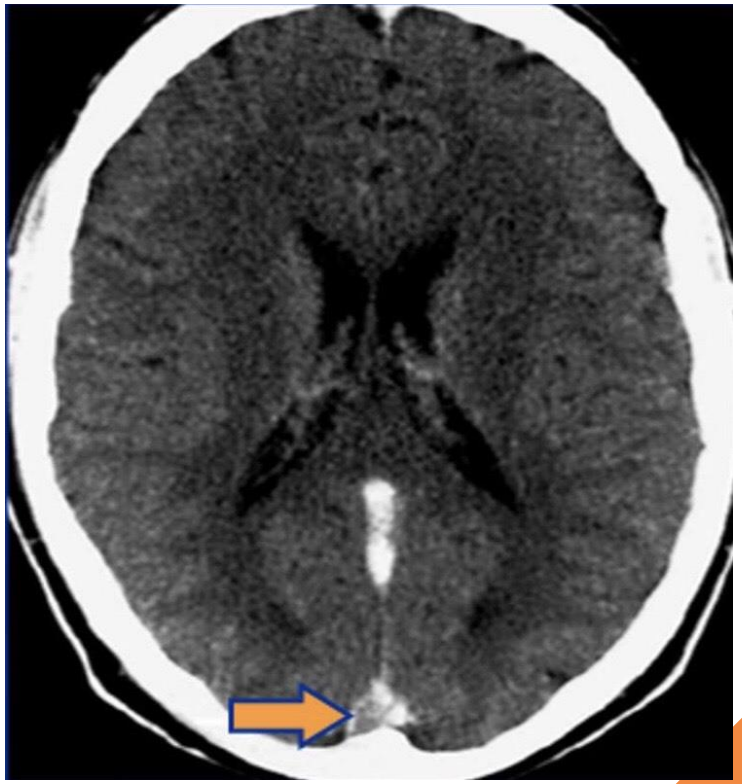
- Hyperattenuating thrombus in the occluded sinus (Linear hyperdensity)



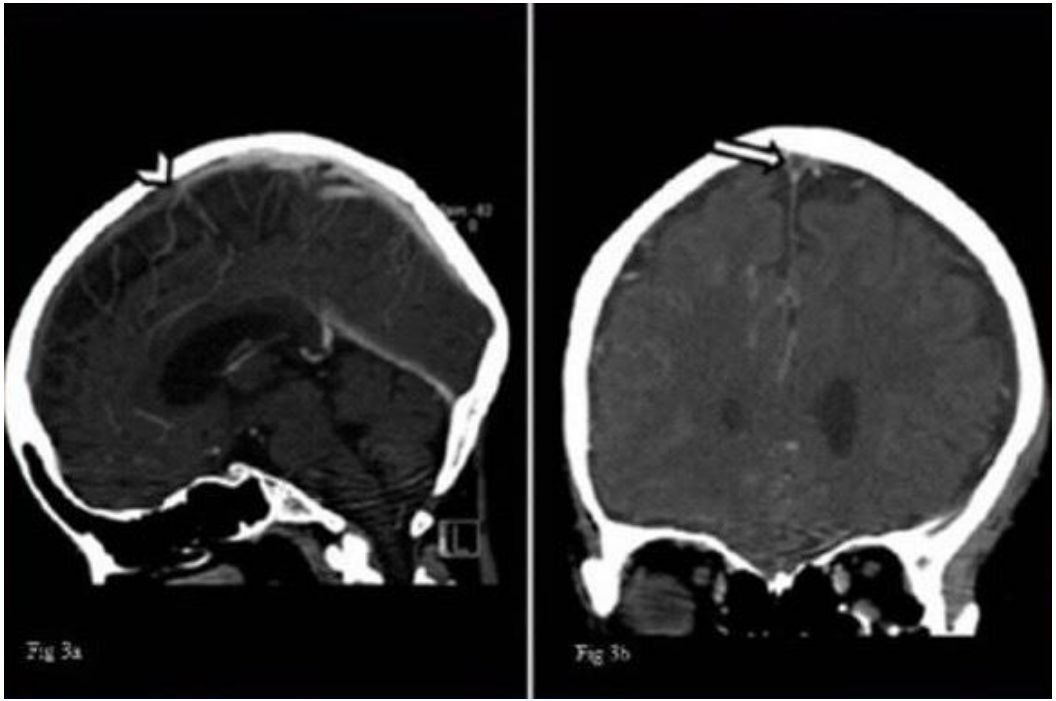


Contrast-enhanced CT

- Empty delta sign
- May seen 5 days to 2 months from onset

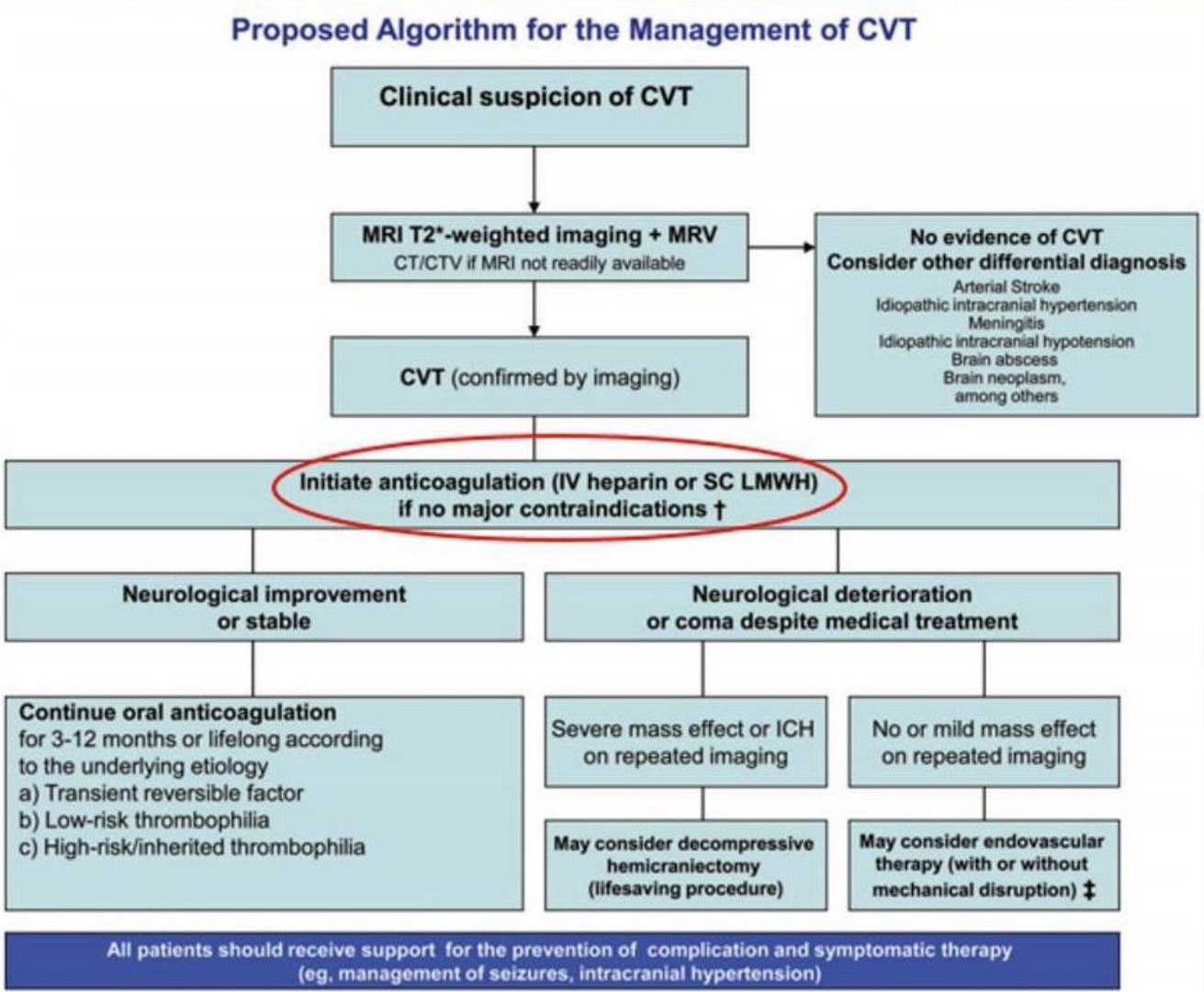


CTV



- Scan like CTA and 45-50 seconds delay after the start of contrast injection
- At least 70 ml of contrast
- Visualization of thrombus filling defect

Management


Proposed Algorithm for the Management of CVT
Clinical suspicion of CVT
MRI T2*-weighted imaging + MRV
CT/CTV if MRI not readily available
No evidence of CVT
Consider other differential diagnosis
Arterial Stroke
Idiopathic intracranial hypertension
Meningitis
Idiopathic intracranial hypotension
Brain abscess
Brain neoplasm,
among others
CVT (confirmed by imaging)
Initiate anticoagulation (IV heparin or SC LMWH)
if no major contraindications †
Neurological improvement
or stable
Neurological deterioration
or coma despite medical treatment
Continue oral anticoagulation
for 3-12 months or lifelong according
to the underlying etiology
a) Transient reversible factor
b) Low-risk thrombophilia
c) High-risk/inherited thrombophilia
Severe mass effect or ICH
on repeated imaging
No or mild mass effect
on repeated imaging
May consider decompressive
hemicraniectomy
(lifesaving procedure)
May consider endovascular
therapy (with or without
mechanical disruption) ‡
All patients should receive support for the prevention of complication and symptomatic therapy
(eg, management of seizures, intracranial hypertension)

THANK YOU