Cerebral Venous Thrombosis in Tropical Areas

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Disclosures

None

Due to copyright reasons, more images will be included in the final presentation.

Learning Objectives

- Risk Factors of CVT
- Clinical Manifestations of CVT
- Diagnosis of CVT
- Treatment of CVT

Why CVT is important

- Heterogeneous condition
- Varied clinical manifestation
- Change in pattern
- Different predisposing factors
- Good prognosis

CVT – Age and Sex

Country	Author	Year	N=	M %	F %	Mean Age
Brazil	Christo P	2010	15	27	73	36
Chile	Truzillo O	2016	62	13	87	37
Morocco	Souirti Z	2014	30	30	70	29
Saudi Arabia	Rizwana S	2019	26	42	58	29
Turkey	Duman T	2017	1144	32	68	38
Tunisia	Sassi S	2017	160	17	83	37
Pakisthan UAE	Khealani	2008	109	47	53	35
India	Narayan	2012	428	54	46	31
India	Pai	2013	573	62	38	34
India	Meshram	2019	894	68	32	32
ISCVT-21C	Ferro J	2004	624	26	74	39
112 Studies	Zuurbier	2016	23638	35	65	37

Cerebral Venous Thrombosis

Risk Factors (Genetic)

- Deficiency of anticoagulants
  - Antithrombin III
  - Protein C , Protein S
- Abnormal Proteins
  - Factor V Leiden
  - Dysfibrinogen
- Increased Procoagulants
  - Prothrombin , Factor VIII
- Abnormal Metabolism
  - Homocysteinaemia

Cerebral Venous Thrombosis
Risk Factors – Acquired

Tissue trauma – surgery
Pregnancy & Puerperium
Malignancy
Sepsis
Nephrotic syndrome
Hyperviscosity state, Dehydration
APLA , PNH
Myeloproliferatus disorders
Hyperhomocysteinaemia
Oral Contraceptive
Inflammatory diseases- Crohns, Behcets, ulc colitis
Drug Abuse,Alcohol

Balance between Procoagulants & anticoagulants keeps the blood fluid & flowing.

Increased Procoagulants &/or decreased anticoagulants can lead to thrombosis.

Why CVT common in Tropical areas

- Anemia
- B12 Deficiency
- Hyperhomocysteinaemia
- Poor obstetrics care
- Dehydration
- Raised Hematocrit
- Infection
- Genetic
- Other environmental factors

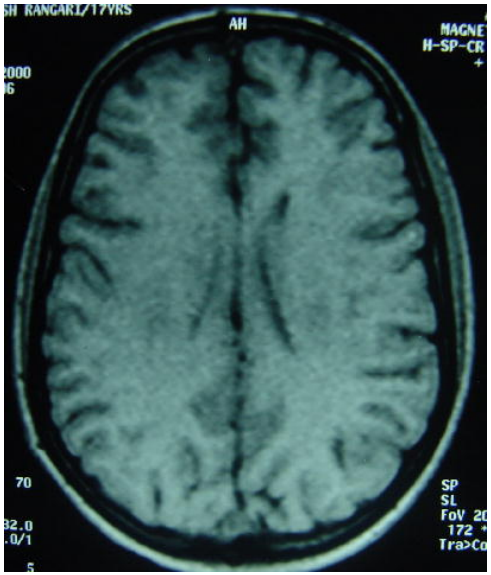
Clinical Presentations

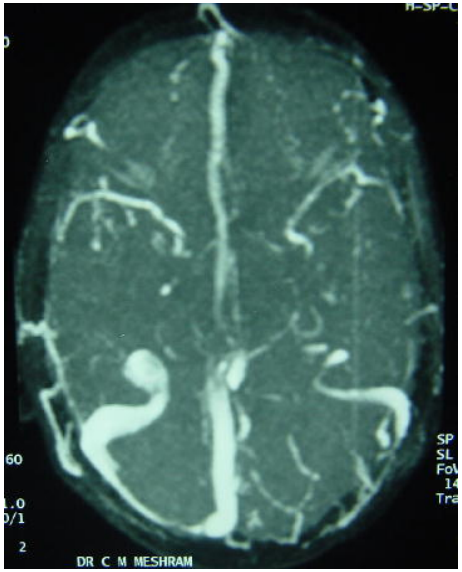
four main syndromes

- Raised intracranial Pressure
- Presentation with Seizures
- Stroke like Presentation with Focal Deficit
- Encephalopathy

GR 17 M

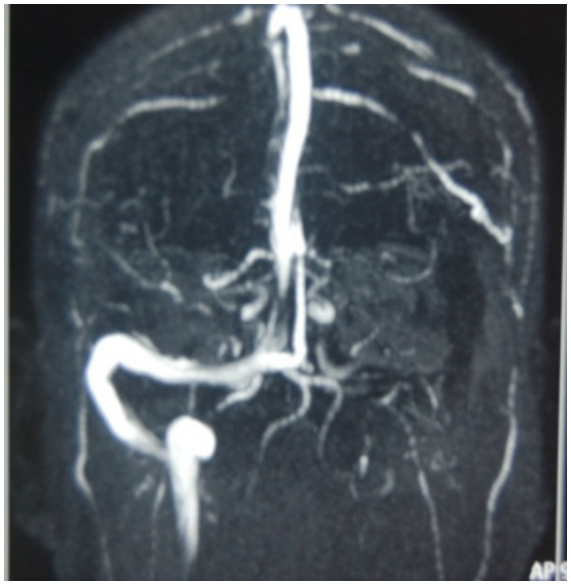
- Headache 3 wks
- Vomiting
- Papilloedema





DT 42 M

- Swelling Lt side of neck
- Headache
- Diplopia – VI nerve palsy
- Papilledema

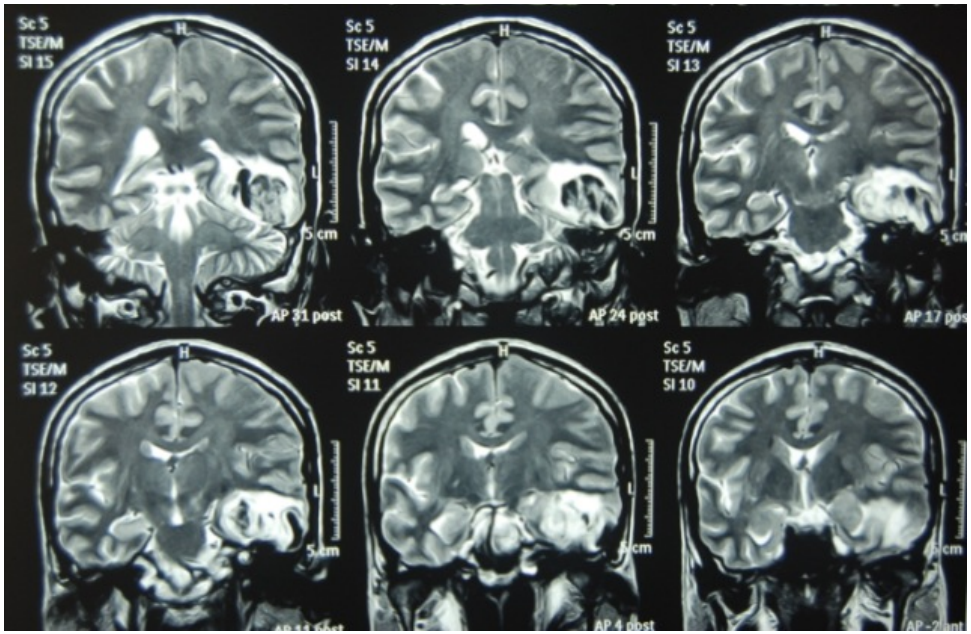


VK 36 m

- Headache – 5 days
- Seizures
- No focal deficit

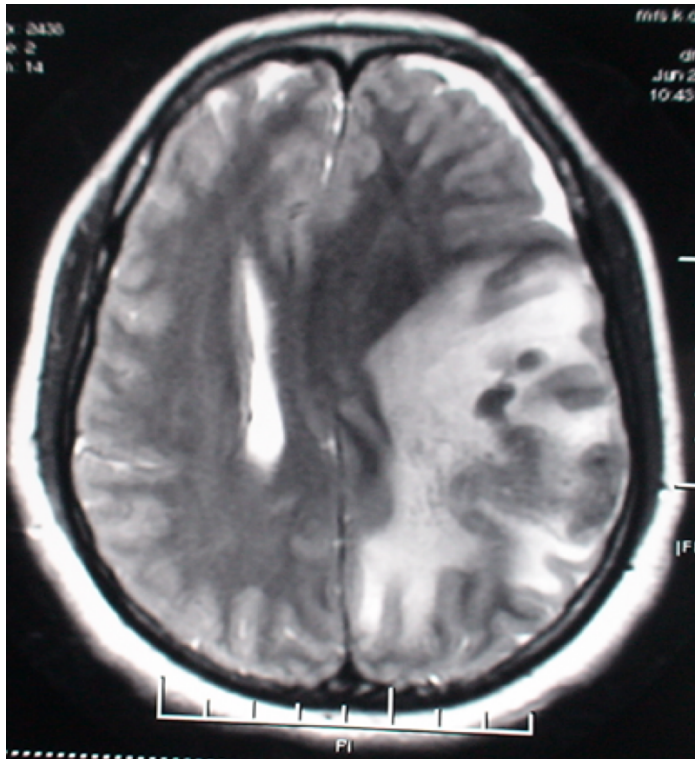
NM 26 M

- Headache – 7 days
- Seizures – 4 days
- Rt. Hemiparesis – 1 day



PU 40 M

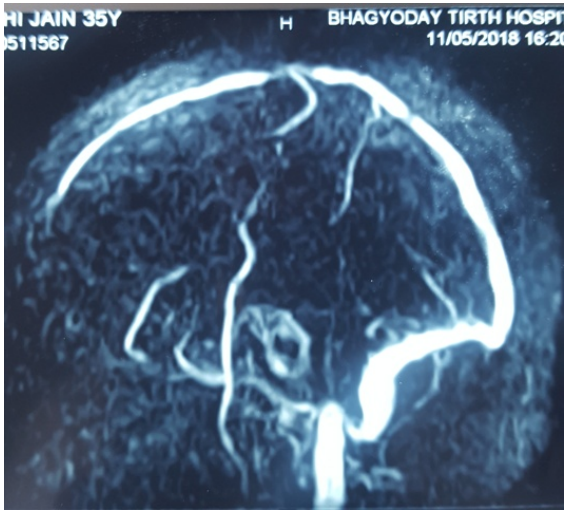
- Headache
- Seizures
- Rt. Hemiparesis
- Altered Sensorium

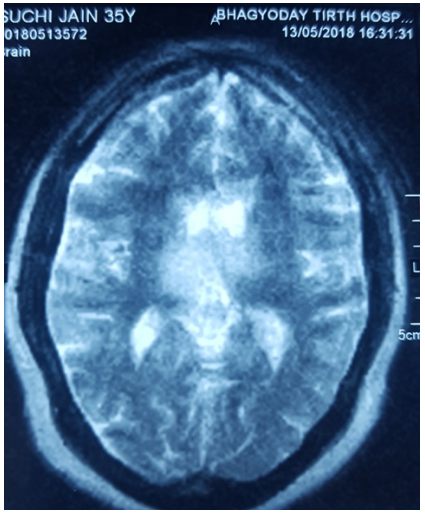


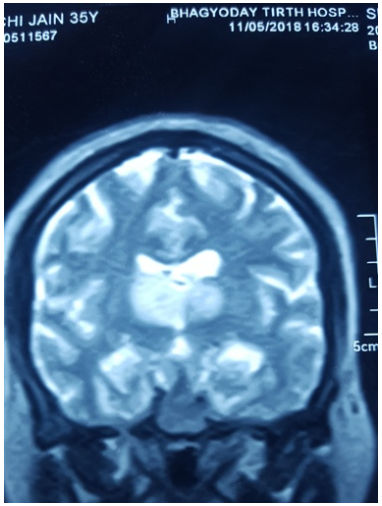
Uncommon Presentation

- Subarachnoid Hemorrhage
- Cavernous Sinus Thrombosis
- Multiple cranial nerve palsies
- Deep venous Thrombosis
- Psychological Symptoms
- Recurrent Syncope
- Hearing Impairment

- SJ 35 F
- Headache
- Altered Sensorium

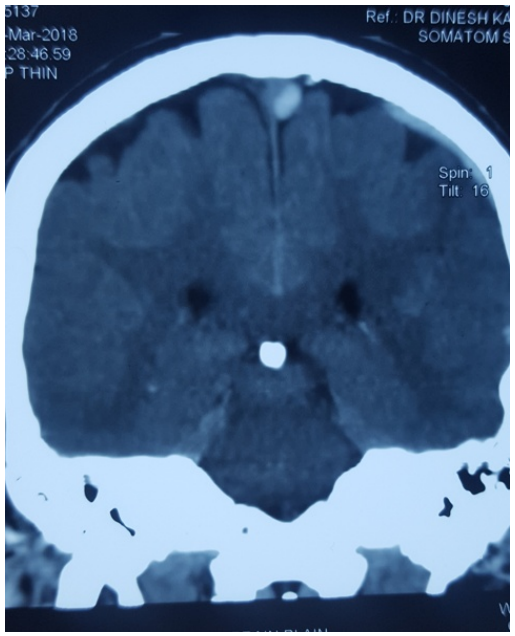


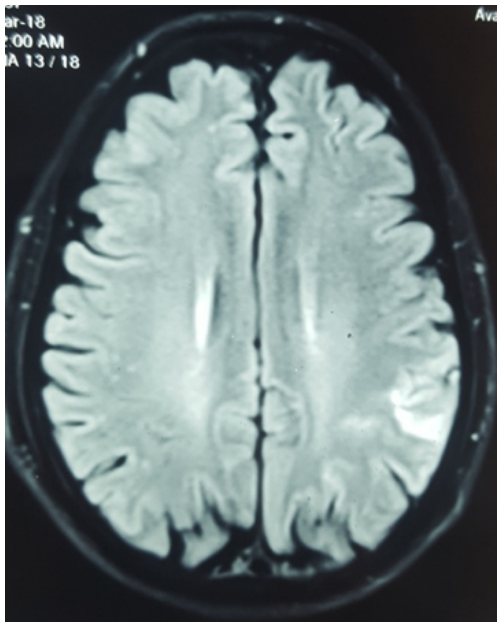


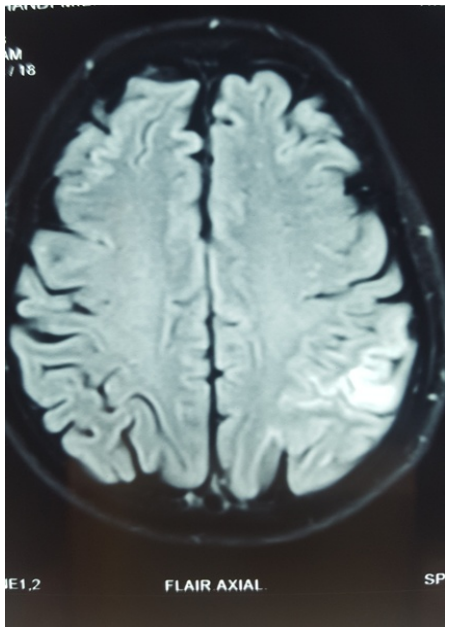


M B 56 M

- Loss of memory and irrelevant Talk
- Wernicke Aphasia
- DM, HT







CVT Suspect if

- Young Patient
- First time headache
- Occipital Headache U/ L → B/L
- Neck Pain
- Headache - seizures
- Papilledema
- H/o Blackouts , Diplopia
- Insidious onset with indolent course
- Altered sensorium

Diagnosis

CVT- Neuroimaging Studies MRI with MRV

Duration	T1	T2
Upto 5 days	Isointense	Hypointense
6- 9 days	Hyperintense	Isointense
10-15 days	Hyperintense	Hyperintense
After 15 days	Hypointense	Hypointense

- Parenchymal Changes
- MRI using gradient T2* Susceptibility weighted sequences
- MRV – TOF –Time of flight technique
- Contrast enhanced MRV

CVT – Neuroimaging Study
CT Scan

- Dense Triangle Sign
- Cord Sign
- Parenchymal Abnormality
- CECT – Empty Delta Sign
- CT Venography

Lab Tests- D Dimer

- Meta-analysis of 14 studies

- Sensitivity – 93.9 %
- Sensitivity - 89.7 %

- Normal D Dimer does not exclude presence of CVT

CVT - Investigations

1. Hereditary Abnormalities
   Activated Protein C Resistance
   (Factor V Leiden)
   Antithrombin Functional Assay
   Protein C functional assay
   Protein S Functional Assay
   Fibrinogen
2. Aquired Abnormalities
   APLA
   Fasting Plasma Homocystein Level

CVT – When to do tests for Thrombophilic factors

- During Stable State
- Not During Acute Thrombosis
- Not When on Anticoagulants

CVT - Treatment

- Symptomatic
- Treatment of Underlying cause
- Treatment of thrombotic Process

Treatment

- Anticoagulation Aim
  to recanalise occluded Sinus
  Prevent Propagation of Thrombus
  Treat underlying Prothrombotic state

CVT- Treatment Anticoagulants

- Heparin
  I.V. unfractionated heparin
  Bolus 80u/kg followed by 18u/kg/hr
- LMW Heparin

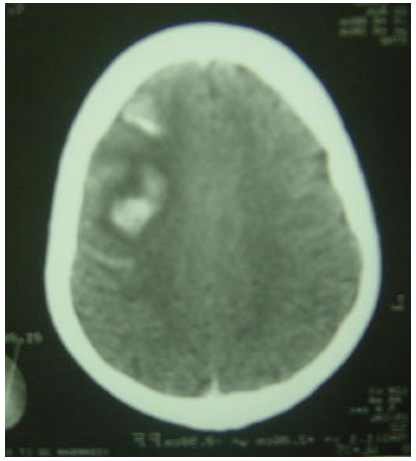
Chronic CVT- Dural AVM

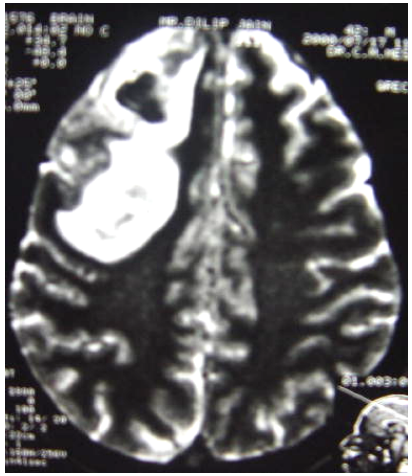
CVT Treatment

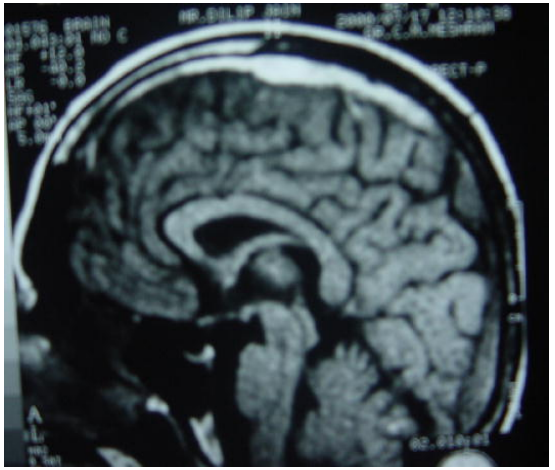
- Oral anticoagulants
- How long to continue

DJ 42 M

- Lt. Hemiplegia, Seizures
- Lupus anticoagulant +ve

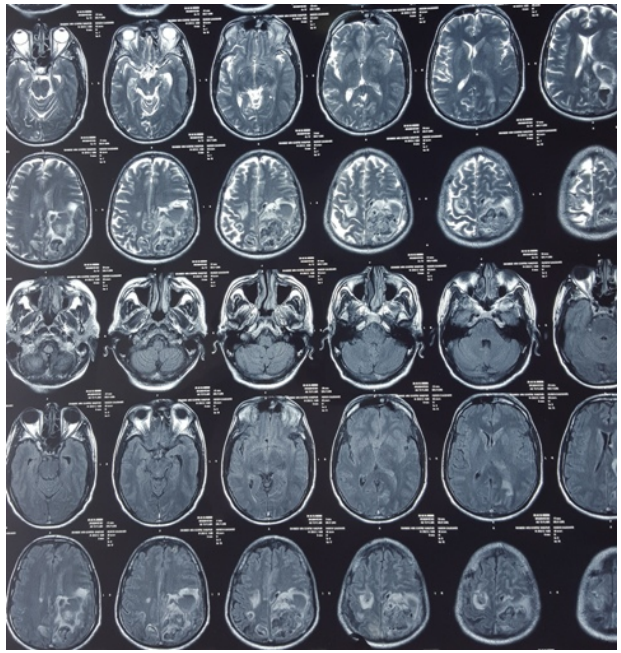






IG 39 M

- CVT-On oral Anticoagulants for 2 years
- Asymptomatic
- Anticoagulants stopped
- After 2 weeks



Genetic Thrombophilia

- Lifelong anticoagulants

Evidence based medicine to experience based medicine

CVT – Treatment
Endovascular Thrombolysis

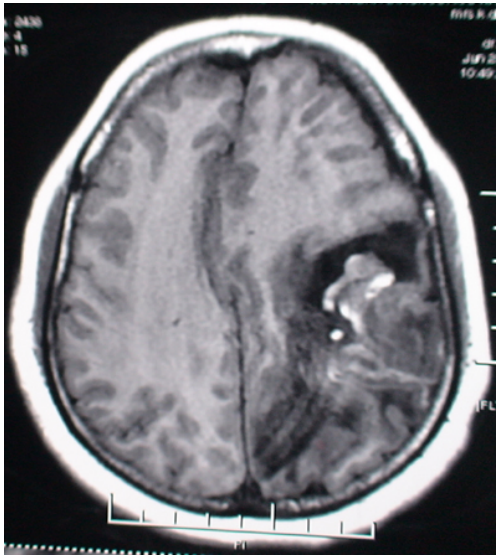
- Urokinase
- rtPA

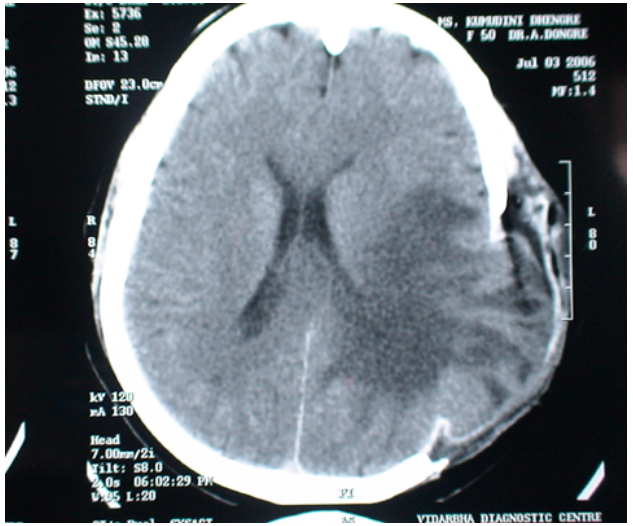
CVT – Treatment

- Mechanical Thrombolysis

CVT – Treatment

- Decompressive Craniotomy
- Life saving measure in case of large venous infarction .





CVT – Prognosis

- Mortality
- Functional recovery is much better as compared to arterial stroke.

CVT – Key Message

- Key to diagnosis is – High index of suspicion.
- Disease of the young
- MRI with MRV is investigation of choice
- Investigate for predisposing conditions
- Early diagnosis would lead to early start of treatment and better prognosis
- Anticoagulation is the treatment of choice even in the patients with hemorrhagic lesions

CVT – References

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