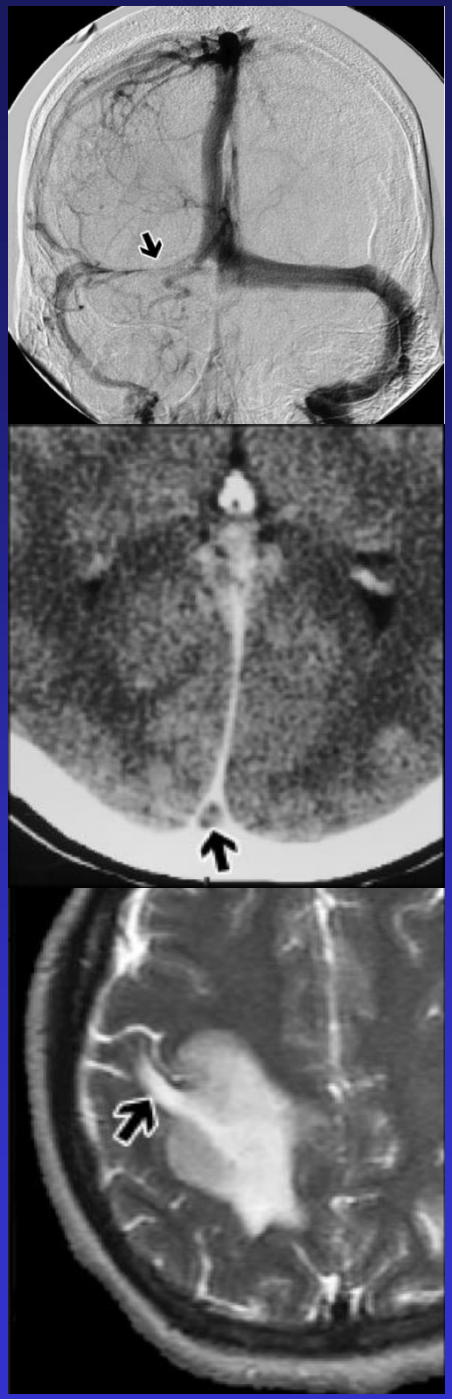


Thrombophlébites cérébrales

Diagnostic et prise en charge

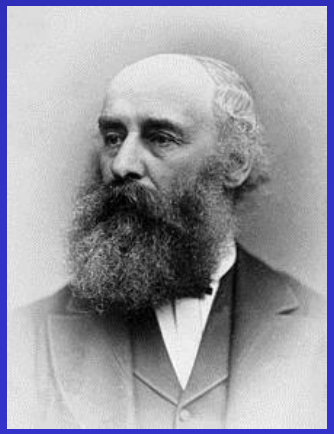
Thomas Ronzière

CHU Rennes

Il s'agit d'une jeune femme de 23 ans qui, ainsi que devait le montrer plus tard l'autopsie, était au début d'une grossesse non avouée, quand, le 25 mai 1887 s'est installée une céphalée intense accompagnée de vomissements, l'obligeant bientôt à s'aliter puis à être hospitalisée le 6 juin. On note une somnolence et un œdème papillaire. Température normale. Urine : ni sucre, ni albumine. Le 8 juin s'installe une paralysie du membre supérieur et une parésie du membre inférieur droit ; l'œdème papillaire augmente. La malade ne répond plus aux questions. Survient ensuite une amélioration progressive, avec reprise de la force et retour de la parole. Le fond de l'œil reste inchangé ; les céphalées, quoique diminuées, continuent. Puis, pendant la nuit du 19 juin, changement brusque de l'état de la malade : elle s'assied, déviation de la tête et des yeux vers la gauche, suivie d'une convulsion tonique en extension avec vomissements. Elle se recouche sur le dos, semble être consciente et suit l'infirmière des yeux mais ne parle plus. Pouls faible, figure pâle. Décès 1 h 1 /2 après.

A l'autopsie générale : utérus contenant un fœtus d'environ 6 semaines ; ovaire droit avec corpus luteum. Par ailleurs rien à signaler.

A l'autopsie du cerveau : toutes les veines superficielles sont distendues et remplies de thrombus adhérents jusque dans l'entrée dans le sinus longitudinal. Deux petites hémorragies symétriques dans la partie antéro-supérieure de la fissure pariéto-occipitale. Les veines de Galien (veines cérébrales internes) sont également thrombosées ainsi que les sinus droit et latéral gauches et la veine jugulaire gauche.



Bristowe, Traité de Neurologie 1888
Trad : L.J. Endtz (La Haye)

John Syer Bristowe (1827–1895) was an English physician.

Jeanne Cl..., 35 ans, n'a jamais été malade. Elle a eu trois grossesses qui se sont terminées par l'accouchement de trois enfants bien portants ; elle n'a pas fait de fausses couches. Elle est actuellement enceinte de sept mois.

Il y a huit jours apparurent brusquement des maux de tête dont elle s'est plainte à son entourage. Les deux jours suivants, ces céphalées augmentèrent d'intensité. Et la nuit, deux jours après le début des troubles, la malade eut trois crises nerveuses suivies de perte de connaissance. Le matin, au réveil, elle présentait une hémiplégie droite avec troubles du langage. Survint ensuite une phase d'amélioration légère qui dura trois jours. Puis apparurent deux nouvelles crises suivies d'une aggravation des phénomènes paralytiques.

Elle entre dans notre service sept jours après le début de ses troubles.

Deux jours après son entrée dans le service, donc dix jours après le début de ses premiers malaises, la malade fait, sous nos yeux, une nouvelle crise. Elle était assise sur le bord de son lit quand elle pâlit, puis tombe, et presqu'immédiatement se cyanose. Elle n'a pas de mouvements cloniques. Elle perd connaissance pendant une minute puis elle reprend conscience. Mais la cyanose s'accentue de plus en plus ; la respiration s'accélère et devient irrégulière. La malade a certainement conscience et ses yeux expriment l'angoisse la plus vive. Enfin la cyanose augmente encore, la respiration devient de plus en plus irrégulière, elle se ralentit et la malade meurt sous nos yeux. Nous tentons immédiatement une extraction de l'enfant par césarienne. L'aspect correspond bien à celui d'un enfant de sept mois ; son cœur bat encore dès l'extraction mais il s'arrête presqu'aussitôt.

SÉANCE DU 11 MARS 1955

Thrombo-phlébite cérébrale de la grossesse. Etude clinique et anatomique (*),

par MM. **J. Dereux** et **J.-F. Dereux**.

Journal of Neurology, Neurosurgery, and Psychiatry, 1978, **41**, 726–729

Intracranial venous thrombosis in the first trimester of pregnancy

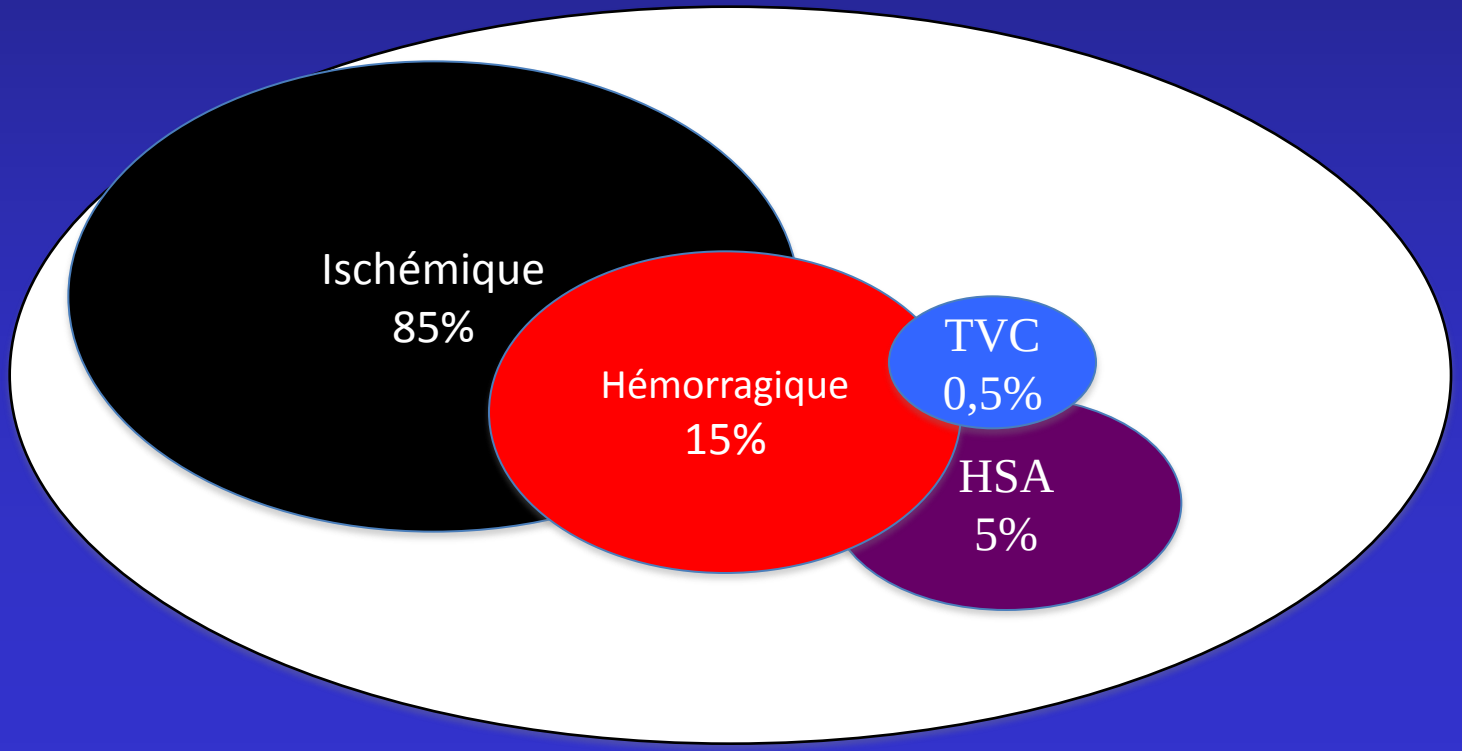
P. J. M. LAVIN, I. BONE, J. T. LAMB, AND L. M. SWINBURNE

From the Departments of Neurology, Pathology, and Neuroradiology, St James's University Hospital and Chapel Allerton Hospital, Leeds

SUMMARY We describe a fatal case of intracranial venous thrombosis occurring in early pregnancy. Such thrombosis usually occurs in late pregnancy or the puerperium but rarely during the first trimester of pregnancy. Computerised axial tomography suggested massive cerebral venous infarction. Necropsy findings showed not only cerebral venous thrombosis but also extensive pelvic and iliac vein thromboses. The relationship of cerebral venous thrombosis and pregnancy is discussed and the literature reviewed.

Thrombophlébite cérébrale

Occlusion d'une veine ou d'un sinus au niveau cérébrale



Thrombophlébite cérébrale

European Journal of Neurology 2010, **17:** 1229–1235 doi:10.1111/j.1468-1331.2010.03011.x

EFNS GUIDELINES

EFNS guideline on the treatment of cerebral venous and sinus thrombosis in adult patients

K. Einhäupl[a], J. Stam[b], M. -G. Bousser[c], S. F. T. M. de Bruijn[d], J. M. Ferro[e], I. Martinelli[f] and F. Masuhr[a]

[a]*Department of Neurology, Charité-Universitätsmedizin Berlin, Berlin, Germany;* [b]*Department of Neurology, Academic Medical Centre Amsterdam, Amsterdam, The Netherlands;* [c]*Department of Neurology, Hôpital Lariboisière, Paris, France;* [d]*Department of Neurology, Haga Hospital The Hague and LUMC, Leiden, The Netherlands;* [e]*Department of Neurology, Hospital Santa Maria, Lisboa, Portugal; and* [f]*A. Bianchi Bonomi Hemophilia and Thrombosis Center, IRCCS Maggiore Hospital, University of Milan, Milan, Italy*

Diagnosis and Management of Cerebral Venous Thrombosis

A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association

Conclusions—Evidence-based recommendations are provided for the diagnosis, management, and prevention of recurrence of cerebral venous thrombosis. Recommendations on the evaluation and management of cerebral venous thrombosis during pregnancy and in the pediatric population are provided. Considerations for the management of clinical complications (seizures, hydrocephalus, intracranial hypertension, and neurological deterioration) are also summarized. An algorithm for diagnosis and management of patients with cerebral venous sinus thrombosis is described. **(*Stroke*. 2011;42:1158-1192.)**

Traitement anticoagulant des thromboses veineuses cérébrales de l'enfant et du nouveau-né. Les recommandations de la Société française de neurologie pédiatrique (SFNP)

GUIDELINES

TVC : une affection rare

- **Pas d'études en population**
  - **Incidence en France : 0.5% des AVC ; 500 cas/an**
  - **Adultes 3-4 pers/1 million - Enfants 7 pers/1 million surtout <6mois**
- **Plus fréquentes que dans les données classiques**
  - **Séries récentes > 100 cas** (Lariboisière: 450)
  - **ISCVT : 624 cas (98 -2001), 89 centres, 21 pays**
  - **probable sous estimation** (formes sans AVC)
- **Tous âges : du nouveau-né au vieillard (m=39)**
- **Prépondérance féminine : F/M : 2-3 / 1**

=> pic d'incidence chez la femme jeune

Contraceptifs OP, grossesse
12 pts / 100.000 accouchements

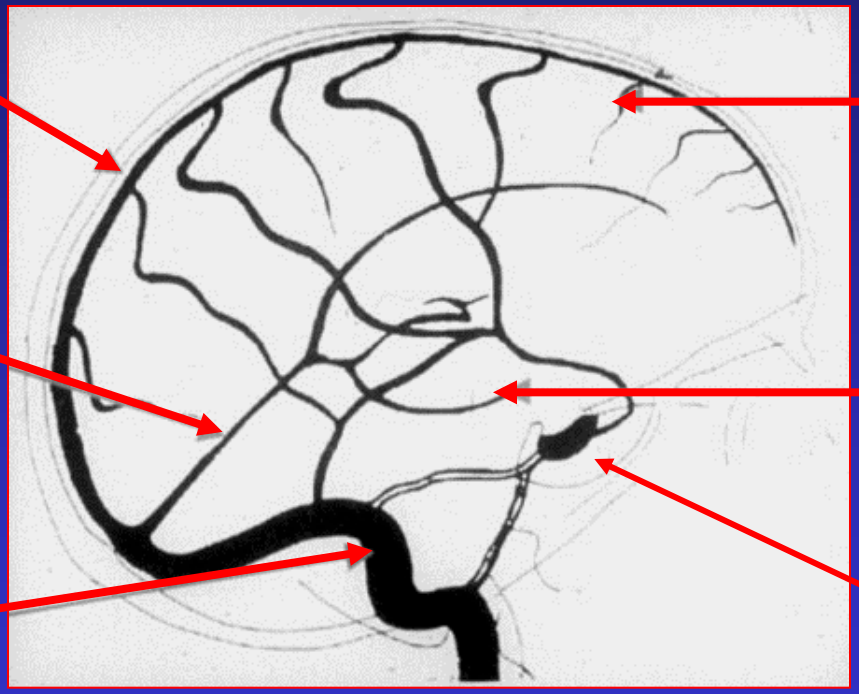
Tous les sinus et veines peuvent être thrombosés souvent en association (60%)

ISCVT (624) Lariboisière (450)

SSS
62-52%

S droit
11-18%

S Latéral
(D, G, les 2)
79%



Veines Corticales
60%

Veines profondes
7-17%

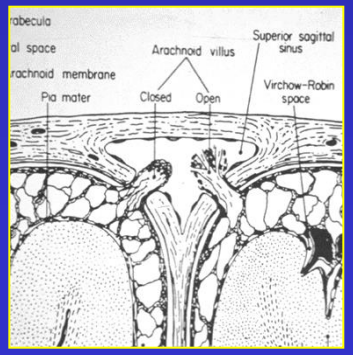
Sinus caverneux
2%

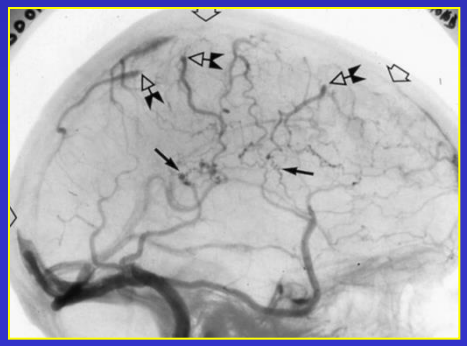
=> Pas de syndromes anatomo-cliniques bien définis

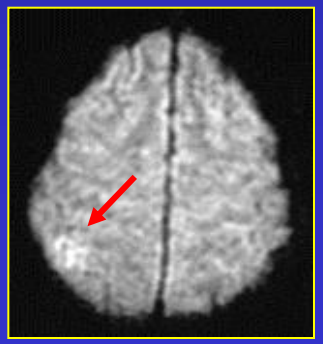
Environnement veineux ≠ artères

- **Sinus**
  - villosités arachnoïdiennes (résorption LCS) => hypertension intracrânienne
  - paroi dure-mère très innervée => céphalées
  - variations anatomiques (torcular, S Lat) => drainage variable
- **Veines corticales**
  - variables en nombre et siège => pas de territoire bien défini
  - parois fines sans fibres musculaires => dilatations , saignement
  - absence de valves => inversion du flux
- **Sinus et veines**
  - Nombreuses anastomoses => circulation collatérale
  - drainage veineux => œdème, hémorragies, peu d'hypoxie/ischémie

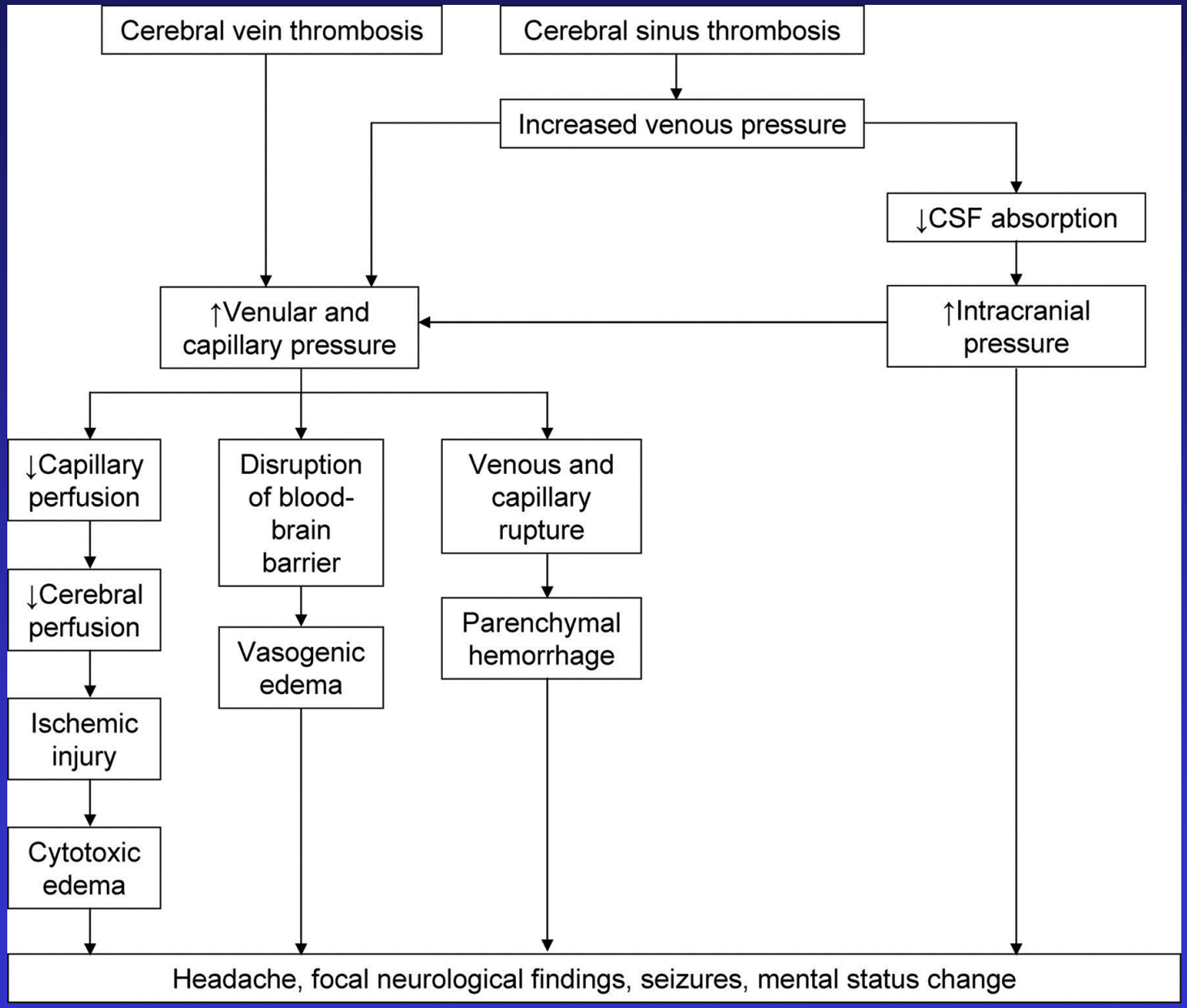
=> Remarquables capacités de récupération







Pathophysiology of cerebral venous thrombosis



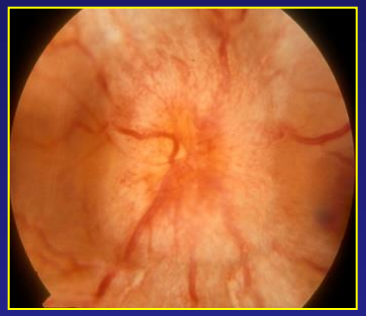
Piazza G Circulation. 2012;125:1704-1709

Polymorphisme clinique

	Lariboisière (450)	ISCVT (624)
Céphalées	96%	89%
Oedème papillaire	36%	28%
Epilepsie	35%	39%
Déficit focal	41%	37%
Troubles de conscience	20%	22%
Paralysie nerfs crâniens	13%	
Syndrome cérébelleux	3%	

Mode d'installation variable :

< 48h : 25% ; 3 - 30j : 60% ; > 30j : 15%



4 grands syndromes

- HTIC isolée : 10-40%
- Syndrome focal : 50-80%
- Encéphalopathie diffuse :10-20%
- Syndrome du sinus caverneux

Formes inhabituelles: 15 à 20%

Présentations inhabituelles

- **Confusion ou trouble psychiatrique isolé**
- **Atteinte isolée d'un nerf crânien (VII, VIII…)**
- **« Accidents ischémiques transitoires »**
- **Céphalées isolées et/ou inhabituelles (15% dans série Lariboisière)**
  - **après hypotension LCS : posturale puis permanente**
  - **"en coup de tonnerre", avec ou sans HSA (3.6%)**
  - **migraineuse, avec ou sans aura**
  - **localisée, rétromastoïdienne**
  - **Isolée, localisée,**
    - **avec scanner et LCS normaux (Pression ?) : 10%**

TOUJOURS PENSER aux TVC

TVC sans céphalée

Cerebral Venous Thrombosis in the Absence of Headache

Jonathan M. Coutinho, MD, PhD; Jan Stam, MD, PhD; Patricia Canhão, MD, PhD; Fernando Barinagarrementeria, MD; Marie-Germaine Bousser, MD, PhD; José M. Ferro, MD, PhD; on behalf of the ISCVT Investigators

Background and Purpose—Although headache is the most common symptom in cerebral venous thrombosis, 5% to 30% of patients do not report headache at baseline. Characteristics of these patients have not been investigated.

Methods—In post hoc analysis of the International Study on Cerebral Vein and Dural Sinus Thrombosis study, patients who might not have been able to report headache (aphasia, stupor, coma, or mental status disorder) were excluded.

Results—Three hundred eighty-two of the original 624 patients (61%) were included, of whom 38 (10%) did not report headache at baseline. Patients without headache were older (mean age, 45 versus 37; P=0.001) and less often female (63% versus 77%; P=0.06). Paresis (42% versus 27%; P=0.05) and seizures (58% versus 32%; P=0.001) were more common in patients without headache, whereas papilledema was less common (8% versus 35%; P=0.001). Isolated cortical vein thrombosis (16% versus 2%; P=0.001), brain parenchymal lesions (66% versus 46%; P=0.02), and malignancies (18% versus 6%; P=0.009) were more common among patients without headache. Outcome at last follow-up was worse in patients without headache (modified Rankin Scale, 0–1; 76% versus 89%; P=0.04; mortality, 13% versus 5%; P=0.05), but after adjustment for prognostic variables, headache was not an independent predictor of outcome.

Conclusions—Patients with cerebral venous thrombosis but without headache are a heterogeneous subgroup, in which older patients, men, and some associated conditions are over-represented. Patients without headache had a worse clinical outcome, but after adjustment for imbalances, headache was not an independent predictor of outcome.
(***Stroke***. **2015;46:245-247. DOI: 10.1161/STROKEAHA.114.007584.**)

Bilan étiologique

FR retrouvé dans 85%, non trouvé 15%
Multiples 44 %
22% un trouble héréditaire de l'hémostase

Transient risk factors

- Infections
  - Central nervous system (empyema, meningitis)
  - Ear, sinus, mouth, face and neck (otitis, mastoiditis, tonsillitis, stomatitis, sinusitis, cellulitis)
  - Systemic infections (sepsis, endocarditis, tuberculosis, human immunodeficiency virus, malaria)
- Pregnancy and puerperium
- Physical precipitants
  - Head trauma
  - Lumbar puncture, myelography, intrathecal medications, spinal anesthesia
  - Radical neck surgery
  - Neurosurgical procedures
  - Jugular and subclavian catheters
- Drugs with prothrombotic action
  - Oral contraceptives, hormone replacement therapy, androgens, medroxyprogesterone acetate, L-asparaginase, cyclosporine, tamoxifen, steroids, lithium, thalidomide, ecstasy, sildenafil
- Other conditions
  - Dehydration
  - Diabetic metoacidosis

Cerebral Venous Sinus Thrombosis: Update on Diagnosis and Management

José M. Ferro · Patrícia Canhão

Curr Cardiol Rep (2014) 16:523

Permanent risk factors

- Prothrombotic conditions
  - Genetic (Protein S, C, antithrombin deficiencies, factor V Leiden and prothrombin mutations)
  - Acquired (antiphospholipid syndrome, nephrotic syndrome, cyanotic congenital heart disease)
- Malignancy
  - Central nervous system (meningioma)
  - Solid tumour outside central nervous system
  - Haematological (leukemias, lymphomas)
- Haematological condition
  - Anaemias (sickle cell disease and trait, iron deficiency, folic acid deficiency)
  - Paroxysmal nocturnal hemoglobinuria
  - Polycythemia (primary or secondary)
  - Thrombocythemia (primary or secondary)
- CNS disorders
  - Dural fistulae
- Inflammatory diseases
  - Behçet's disease
  - Systemic lupus erythematosus
  - Sjögren's syndrome
  - Wegener's granulomatosis
  - Temporal arteritis
  - Thromboangiitis obliterans
  - Inflammatory bowel disease
  - Sarcoidosis
- Other disorders
  - Thyroid disease (hyper and hypothyroidism)

Bilan étiologique : grave ?

- Transient risk factors
  - Infections
    - Central nervous system (empyema, meningitis)
    - Ear, sinus, mouth, face and neck (otitis, mastoiditis, tonsillitis, stomatitis, sinusitis, cellulitis)
    - Systemic infections (sepsis, endocarditis, tuberculosis, human immunodeficiency virus, malaria)
  - Pregnancy and puerperium
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  - Other conditions
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  - Other disorders
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Bilan étiologique : traitement spécifique ?

Transient risk factors

- Infections
  - Central nervous system (empyema, meningitis)
  - Ear, sinus, mouth, face and neck (otitis, mastoiditis, tonsillitis, stomatitis, sinusitis, cellulitis)
  - Systemic infections (sepsis, endocarditis, tuberculosis, human immunodeficiency virus, malaria)
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José M. Ferro · Patrícia Canhão

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- Other disorders
  - Thyroid disease (hyper and hypothyroidism)

Etiologies des TVC des enfants et des nouveaux-nés

Paediatric cerebral sinovenous thrombosis: findings of the International Paediatric Stroke Study

Ichord RN, *et al*. *Arch Dis Child* 2015;**100**:174–179. doi:10.1136/archdischild-2014-306382

Risk factors*	
Acute provoking illness	
Acute head/neck infection†	78/169 (46)
Meningitis	14/169 (8)
Acute systemic illness or injury	
Dehydration	34/165 (17)
Acidosis	4/165 (2)
Anoxia	6/165 (4)
Shock	4/165 (2)
Trauma	19/169(11)
Central venous line	2/169 (1)
Underlying chronic disease condition	
Prothrombotic state*	34/170 (20)
Protein C or S deficiency	2
Antithrombin deficiency	0
Factor V Leiden	5
Prothrombin mutation	4
Lipoprotein (a) elevation	9
Antiphospholipid antibody	2
Lupus anticoagulant	2
MTHFR mutation	12
Factor VIII elevation	1
PA1 mutation	3
Thrombocytosis	1
Other	0
Haematological disorder	32/170 (19)
Iron deficiency anaemia	15/170 (9)
Haemoglobinopathy	1/170 (1)
Haematological malignancy	16/170 (10)
Immunological disease‡	6/170 (4)
Cardiac disease	5/167 (3)
Extracranial solid tumour	2/170 (2)
Other§	8/170 (5)

Data are given as N (%) unless otherwise specified.
*Risk factors and prothrombotic factors are not mutually exclusive. Multiple risk factors coexist in many patients.
†Otitis, sinusitis and mastoiditis.
‡Inflammatory bowel disease, Kawasaki syndrome and macrophage activation syndrome.
§Osteogenesis imperfecta, brain tumour, vein of Galen malformation and oral contraceptive use.

Imagerie TVC

- Si suspicion de TVC :

  - IRM = examen de référence

  - Séquences classiques et angio-IRM (3D gado ou 2D TOF)

- Angio-TDM

  - Si contre indication à l'IRM

  - En cas de difficulté d'accès à l'IRM

  - Supériorité de l'IRM pour l'atteinte parenchymateuse

Pas de parallélisme entre l'importance des lésions parenchymateuses et l'étendue de la thrombose sinusienne…

TVC : IRM - Evolution du thrombus

	Sinus Normal	Thrombus <J5	Thrombus J5-J30	Thrombus <1mois
T1	Hypo	Iso	Hyper	Iso/Hyper
T2	Hypo	Hypo/Iso	Iso/Hyper	Iso/Hyper
T1 gado	Homogène	Signe du Delta		

T2 EG : thrombus = artefacts hyposignal

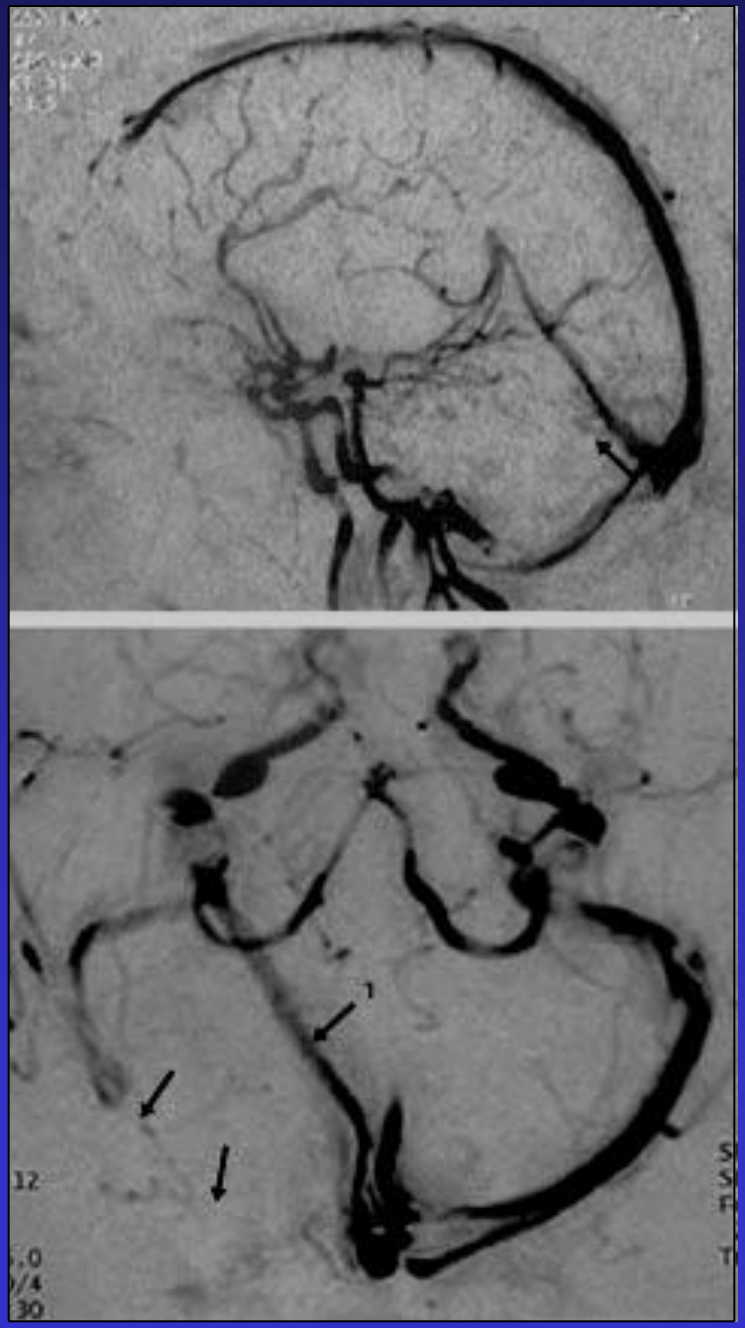
Images pièges (1)

- **Asymétrie de drainage**
  . Sinus latéral droit habituellement dominant
  . Aplasie complète ou partielle du SL possible
  . Si hypoplasie :
    gouttière du SL
    trou déchiré post
    veine jugulaire int
    ⎫
    ⎬ hypoplasiques
    ⎭

  . Hypertrophie controlatérale
  . Mieux visualisée sur TDM ++

Variante anatomique

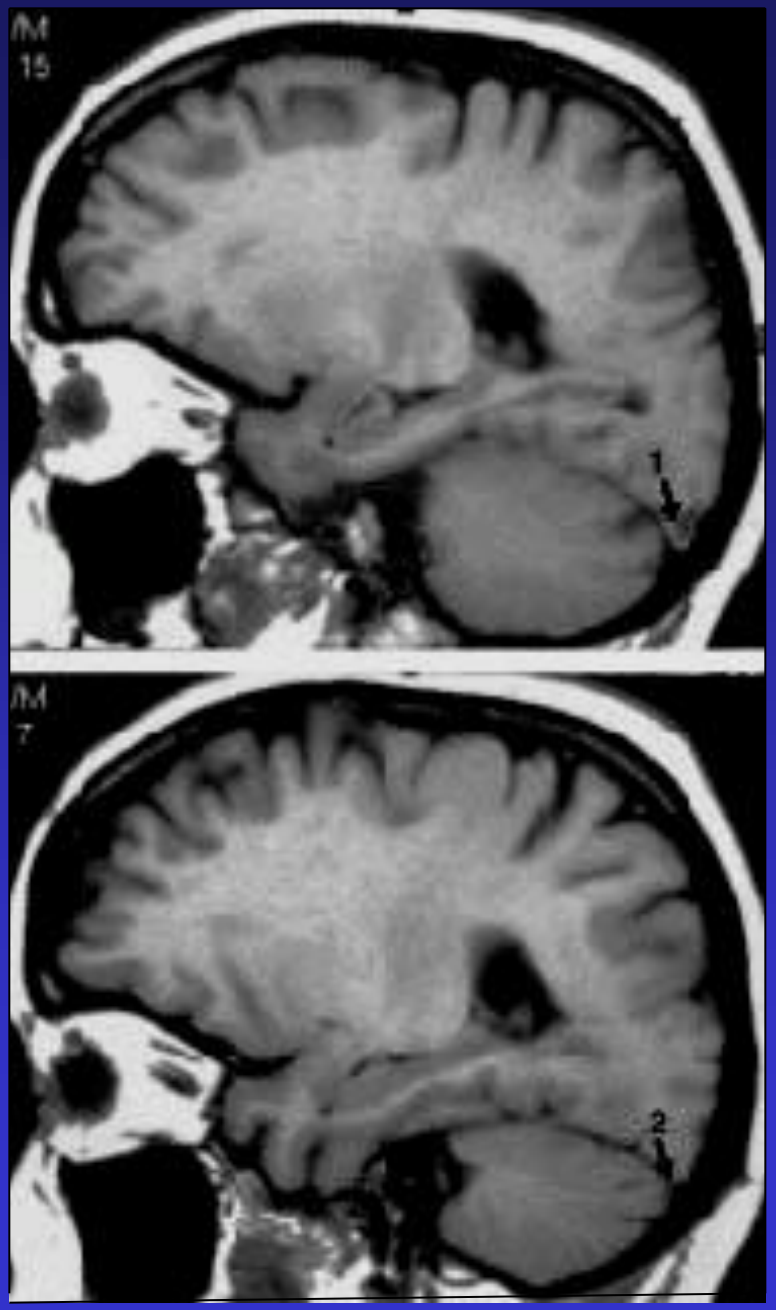
Angio-IRM en 2DPC



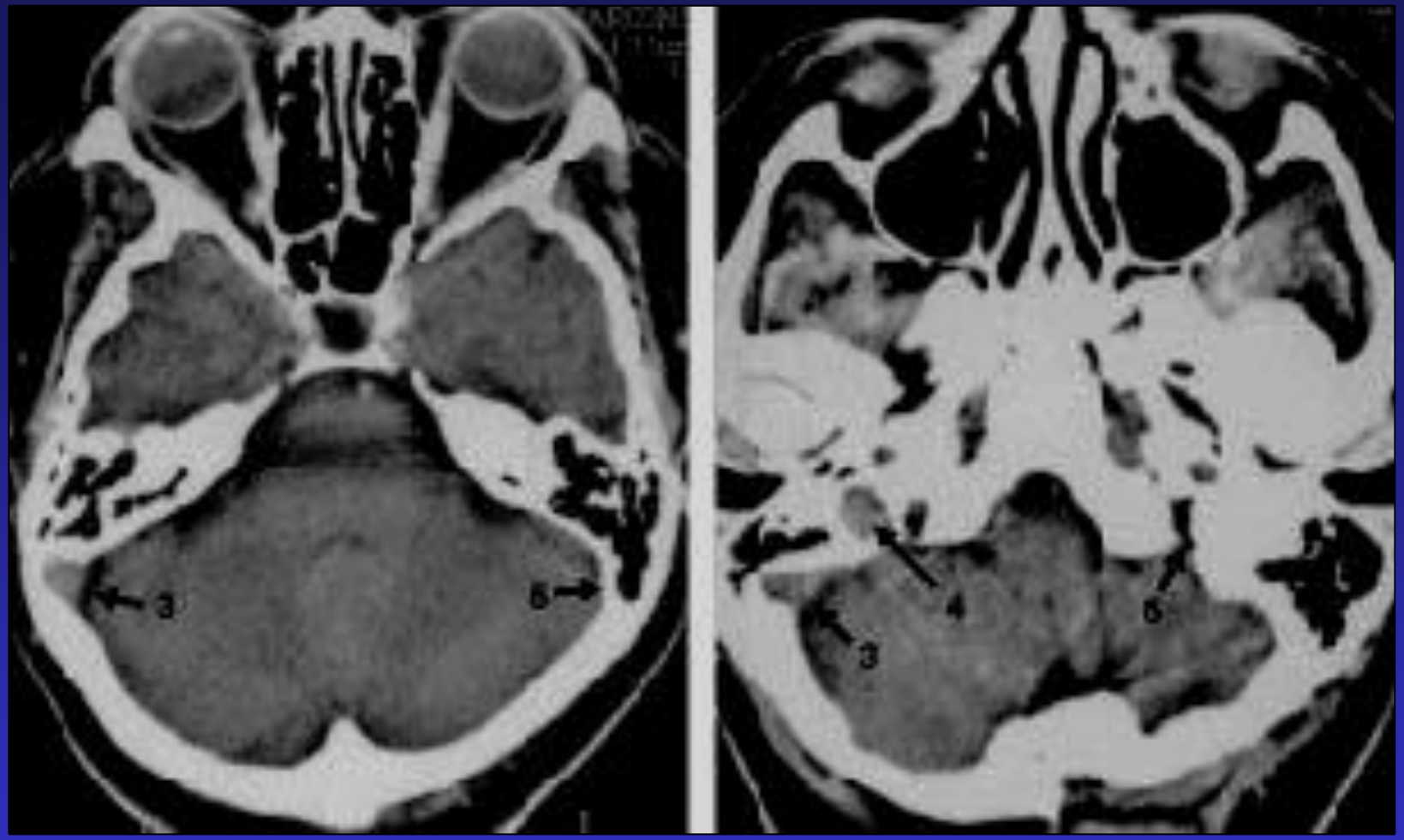
- Sinus latéral droit aplasique
- Sinus latéral gauche large
- Drainage veineux par sinus occipital

Variante anatomique

Coupes sagittales T1



→ Aplasie du SL gauche



TDM après injection

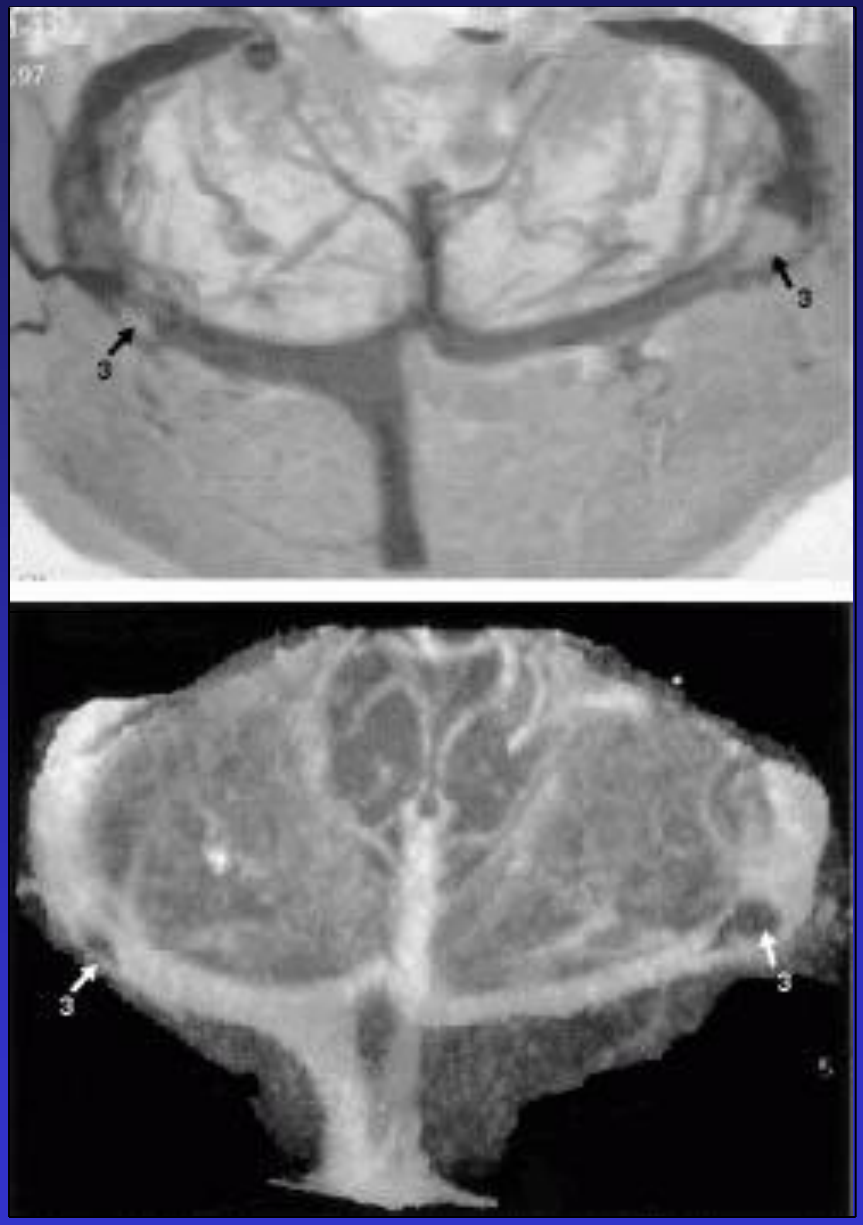
- Sinus latéral + golfe de la jugulaire interne larges à droite
- Pas de sinus latéral en arrière de la mastoide gauche
- Golfe de la veine jugulaire interne gauche hypoplasique

Images pièges (2)

- **Granulations arachnoidiennes de Pacchioni**

. Défect intra sinusien, bien limité
. Densité ou signal liquidien
. Correspond à des invaginations des leptoméninges dans les sinus duraux
. Surtout dans sinus transverse (90%)
. Aucune prise de contraste
. Prévalence augmente avec l'âge

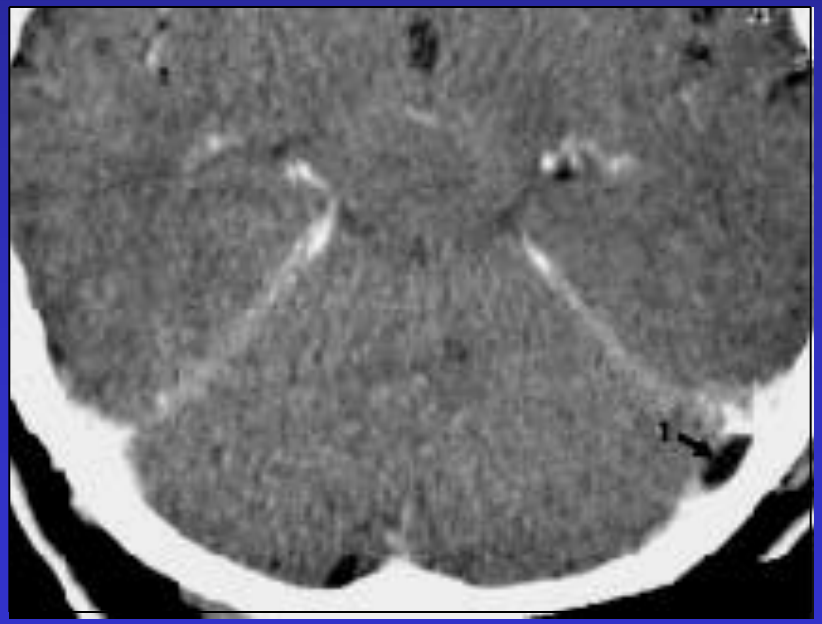
Angio-IRM 3D gado



Angio-scanner en projection MIP

Granulations de Pacchioni

Peuvent simuler une thrombose localisée



TDM après injection

Images pièges (3)

- **Variations anatomiques**

  . Terminaison du SSS au dessus de la protubérance occipitale interne en 2 sinus latéraux

→ Provoque un faux signe du delta

Images pièges (4)

- **Artéfact de flux**
  . Hypersignaux au sein des SL et SSS

- **Artéfact technique**
  . Hyperdensité des sinus chez nourrisson
  . Densité sinusienne élevée si taux d'hématocrite augmenté

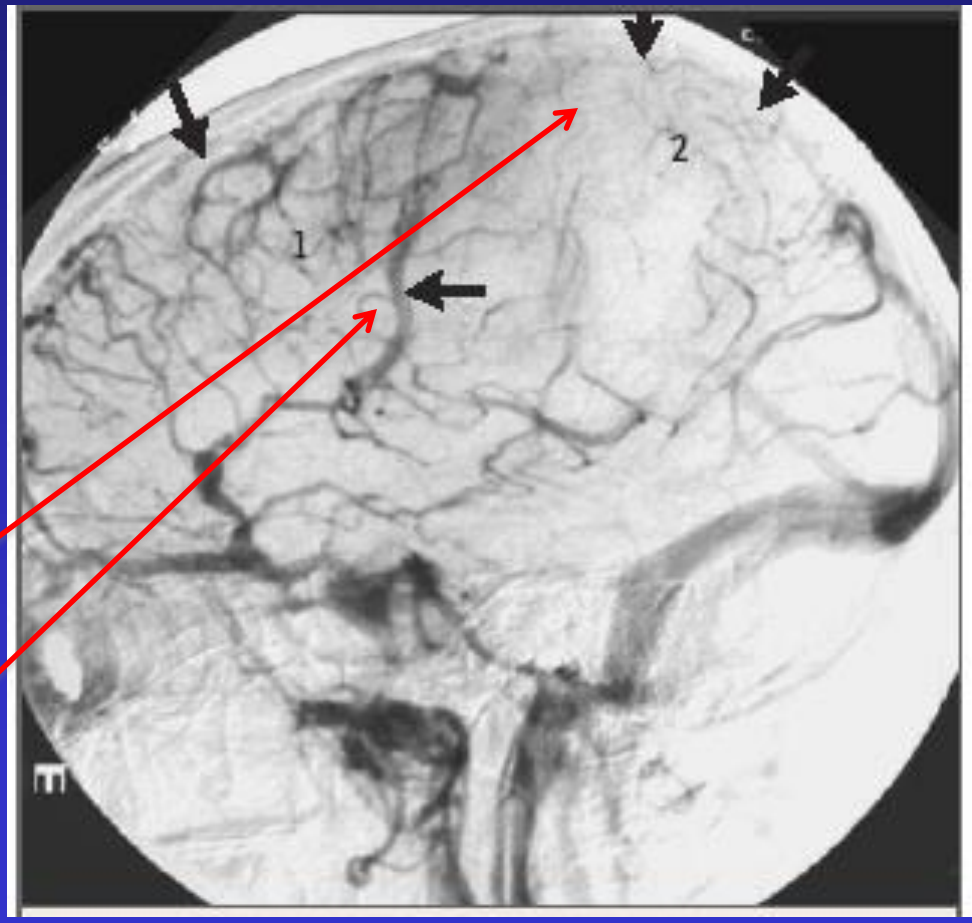
- **Septa fibreux intrasinusien**

Angiographie cérébrale

Examen de référence

-Si CI à l'IRM et scanner normal
-Intérêt dans T. corticale isolée

Retard circulatoire:
stagnation du produit de contraste dans les veines cérébrales, en amont de l'occlusion.
Occlusions veineuses:
absence d'opacification
Voies de suppléance:
dilatation d'un réseau collatéral



Imagerie TVC Rennes

- Pas de diagnostic de thrombophlébite sans imagerie cérébrale
- 200 demandes de recherche de TVC à Rennes par an (TDM + IRM)
- 5 à 10 cas de TVC diagnostiqués…

Autres examens complémentaires (à visée étiologique)

- **Ponction lombaire : A faire**
  - ***- en l'absence de contre-indication***
  - ***- avant l'héparinothérapie***
  - ***- après bilan d'hémostase***
  - **- avec prise de pression du LCS si HIC isolée**

=> double intérêt :

- étiologique : méningites de tous types

- thérapeutique : HTIC isolée => amélioration rapide des céphalées et de la vision

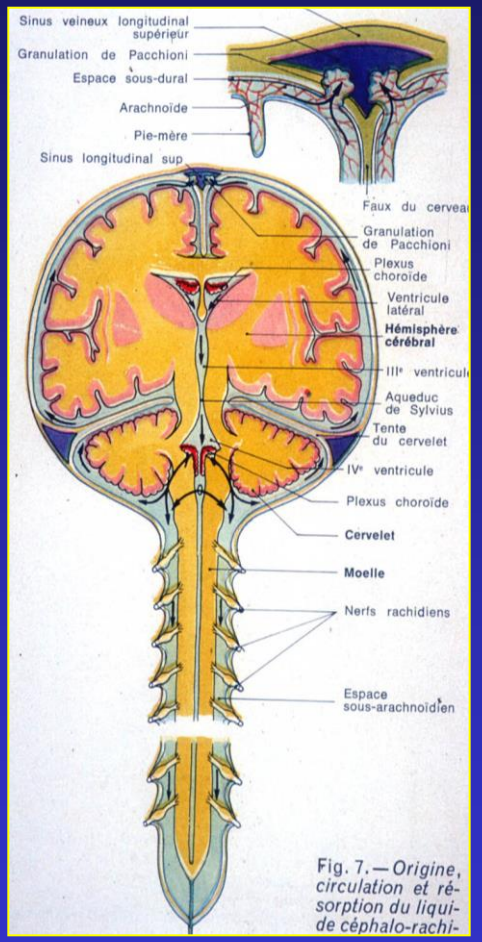


Fig. 7.—*Origine, circulation et résorption du liquide céphalo-rachi-*

- **D-Dimères** : **=> D-Dimères normaux excluent le diagnostic de TVC récente sauf en cas de céphalée isolée** (D-Dimères normaux: 25%) ***Crassard et al Stroke2005***

Place des D-Dimères

Stroke
Journal of the American Heart Association
American Stroke Association
A Division of American Heart Association

2004

Do Normal D-dimeres Levels Reliably Exclude Cerebral Sinus Thrombosis
Kosinski & al.

343 pts =>35 TPC
étude prospective multicentrique
Cutt off 500

Se= 97,1% ; Sp 91,2%,
VPN 99,6%, VPP 56%

D-Dimères normaux =>TPC improbable

2005

A Negative D-Dimer Assay Does Not Rule Out Cerebral Venous Thrombosis Crassard & al.

73 pts
Cutt-off à 500 ng/ml
Normaux :

- 10 % des TPC à la phase aigue de l'ensemble de la cohorte
- 26 % des patients présentant des céphalées isolées

1 seul patients avec des signes focaux avait des D Dimères négatifs

Des D-Dimères normaux n'excluent pas le diagnostic

D-Dimères rarement négatifs si dosés après 7j et/ou signes focaux, convulsions...
D-dimères négatifs n'excluent pas le diagnostic surtout si céphalées isolées ou premier symptôme < 7 j

Pronostic

Prognosis of Cerebral Vein and Dural Sinus Thrombosis: Results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT)

José M. Ferro, et al. 2004

TABLE 3. Outcome at Discharge, 6 Months, and Last Follow-Up

	Outcome at Discharge (n=624)		Outcome at 6 Months (n=616)		Outcome at Last Follow-Up (n=624)	
	No. of Cases	%	No. of Cases	%	No. of Cases	%
Modified Rankin Scale						
0	170	27.2	284	46.1	356	57.1
1	240	38.5	197	32	137	22
2	96	15.4	49	8	47	7.5
3	43	6.9	24	3.9	18	2.9
4	33	5.3	16	2.6	10	1.6
5	15	2.4	4	0.6	4	0.6
Death	27	4.3	42	6.8	52	8.3
Complete recovery	410	65.7	481	78.1	493	79
Death or dependency	118	18.9	86	14.0	84	13.4

79% bon pronostic

7,5% Handicap mineur

5% Handicap sévère

8% décès

Pronostic

- Récidive TVC :
  - ISCVT 2,2 % de TVC
  - 3,7 % autres veineux

Recurrences in untreated patients with isolated cerebral venous thrombosis

Julia Petrova[1], Victor Manolov[2]*, Borislav Milev[3], and Vasil Vasilev[4]

We suggest that the risk of recurrences in untreated patients with monosymptomatic focal cerebral signs is associated with insufficient treatment.

Int J Stroke 2015

Complication à long terme des TVC

- **Fistule artério-veineuse Durale**
  - Ouverture de shunt artério-veineux lié à l'inflammation post-thrombotique
  - Connexion entre
    - branche artérielle durale souvent issue d'une collatérale de la carotide externe
      - artère méningée moyenne
      - artère occipitale
    - veines durales non thrombosées (issues du réseau de collatéralité)
- **Récidive :** 12% des cas (essentiellement dans la première année)

Fistules Durales: *Classification de Djindjian et Merland revisité en 1998*

- **Type I** : Drainage direct dans un **sinus veineux**
- **Type II**: Drainage dans un **sinus veineux mais reflux rétrograde…**
  - **IIa :** dans d'autres sinus veineux
  - **IIb:** dans des veines corticales
  - **II a+b**: dans d'autres sinus veineux et des veines corticales
- **Type III** : Drainage direct dans une **veine corticale**
- **Type IV** : Drainage direct dans une **veine corticale dilatée**
- **Type V** : Drainage veineux **péri-médullaire**
  - **Myélopathie ischémique+++**

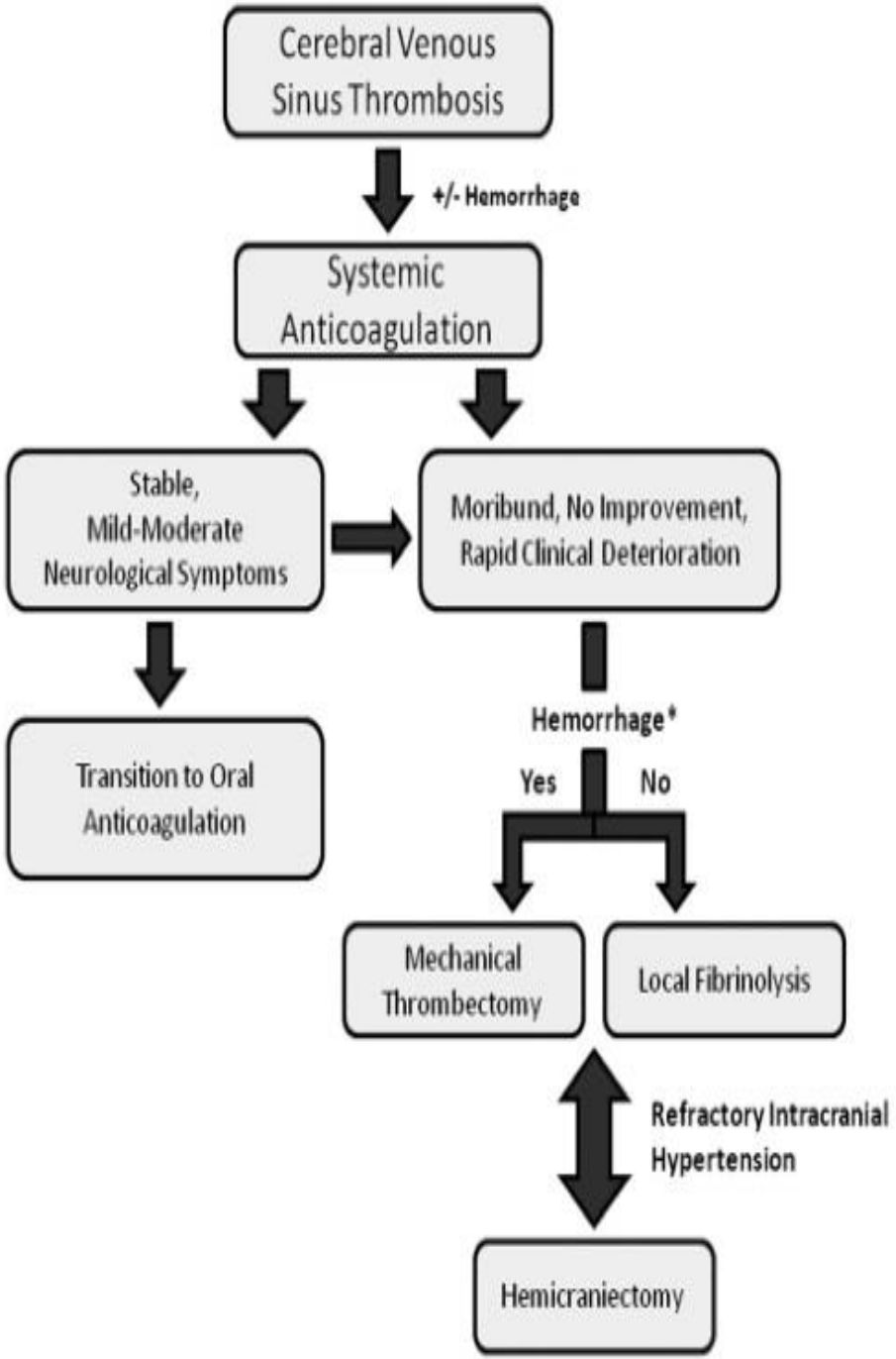
Risque Hémorragique

10%

40%

80%

Traitement



Neurosurg Focus 2009 Rahman & al

Table 2 CVT treatment: adapted from EFNS guidelines

Acute phase

1. Treatment of the permanent or transient risk factors, if applicable
2. Antithrombotic treatment
   - i. Sc. Low molecular weight heparin or IV heparin in therapeutical dosages
   - ii. Local IV thrombolysis and/or mechanical thrombectomy is an option if neurological worsening occurs despite best medical treatment, is not explained by an herniating lesion, occurs despite best medical treatment and other causes of neurological deterioration (e.g. non convulsive epileptic seizures, meningitis) are excluded
3. Symptomatic treatment
   - a. Antiepileptics
     - i. In patients with acute seizures and supratentorial lesions
     - ii. Consider as an option also for patients with acute seizures without supratentorial lesions
   - b. Intracranial hypertension
     - i. Headache.
       1. Analgesics
       2. Lumbar puncture, if there are no parenchymal lesions; perform before starting anticoagulation
       3. Acetazolamide.
     - ii. Impairment of consciousness or herniation
       1. Osmotic therapy
       2. Sedation and hyperventilation
       3. Decompressive surgery (hemicraniectomy and hematoma evacuation)
     - iii. Threatened vision
       1. Lumbar puncture, if there are no parenchymal lesions; perform before starting anticoagulation.
       2. Acetazolamide
       3. Lumboperitoneal shunt
       4. Optic nerve fenestration

Post-acute phase

1. Treatment of the permanent or transient risk factor, if applicable
2. Antithrombotic treatment
   a. Oral anticoagulants
      i. For 3-6 months, if CVT is associated with a transient risk factor
      ii. For 6-12 months, if CVT is idiopathic or associated with a "mild" hereditary thrombophilia
      iii. Permanent, if CVT is recurrent or associated with a "combined" or "severe" hereditary thrombophilia
3. Symptomatic treatment
   a. Antiepileptics
      i. In patients with acute seizures or seizures in the post-acute phase and supratentorial lesions
      ii. Consider as an option also for patients with acute seizures or seizures in the post-acute phase without supratentorial lesions
   b. Headache
      i. Paracetamol
      ii. Acetazolamide
      iii. Lumbar puncture (s)
      iv. Lumboperitoneal shunt
   c. Threatened vision
      i. Acetazolamide
      ii. Lumbar puncture (s)
      iii. Optic nerve fenestration
      iv. Lumboperitoneal shunt

Traitement

Etiologique
Septique+++
Arrêt des Oestro-Progestatifs

Symptomatique
Antalgiques
Anti épileptiques
HTIC

Antithrombotique
Héparines
Fibrinolyse
Geste endovasculaire

40 % crises convulsives avant le Dg
TT recommandé chez patients ayant présenté des convulsions et ayant des lésions supratentorielles

Stroke 2008 FERRO & al.

+ patient sévère, coma..

HTIC
Acetazolamide, PL répétés, fenestration du nerf optique
Osmothérapie, VM, chirurgie : neuroréa
Craniectomie de décompression, résection des zones infarcies

Neurosurgery 1999 STEFINI & al.
Neurosurg Focus 2008 LANTERNA & al.

Une stratégie simple est efficace et suffisante dans 90% des cas

Cerebral Venous Thrombosis — A Review of 38 Cases

MARIE-GERMAINE BOUSSER, M.D.,* JACQUES CHIRAS, M.D.,†
JACQUES BORIES, M.D.,† AND PAUL CASTAIGNE, M.D.*

SUMMARY **A series of 38 patients with angiographically proven cerebral venous thrombosis (CVT) affecting dural sinuses is reported. This study shows that CVT is not rare, that the clinical diagnosis is extremely difficult because of the variable modes of onset and groupings of symptoms, that most CT findings are non specific and that angiography remains the best diagnostic tool. Only 4 patients died, which suggests a more benign outcome than classically described. None of the 23 heparin treated patients died, which indicates that anticoagulants were not harmful in this series.**

Stroke Vol 16, No 2, 1985

Anticoagulants — first used in 1942 by Stansfield[73] — are said by some to be dangerous because they promote hemorrhage into an already hemorrhagic infarct[7, 17, 27, 29] and by others to be highly beneficial, probably by preventing extension of thrombosis and allowing collateral circulation to develop.[9, 10, 16, 18, 28, 74] The only therapeutic conclusion that can be drawn from the present study is that anticoagulants were not harmful since among the 23 heparin treated patients, there was not a single death and 19 made a complete recovery. Furthermore, the dramatic improvement seen in 3 patients just after initiation of heparin does suggest that anticoagulants may be beneficial;[9, 10, 28] this seemed to be the case even in the patient who had a CT scan suggestive of an hemorrhagic infarct.

ISCVT
624 pts
39 % hémorragie intracérébrale avant traitement
83% mis sous héparine
Mortalité à j30 : seulement 3,4%

Diagnosis and Management of Cerebral Venous Thrombosis : A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association

Gustavo Saposnik, Fernando Barinagarrementeria, Robert D. Brown, Jr, Cheryl D. Bushnell, Brett Cucchiara, Mary Cushman, Gabrielle deVeber, Jose M. Ferro and Fong Y. Tsai

6. For patients with CVT, initial anticoagulation with adjusted-dose UFH or weight-based LMWH in full anticoagulant doses is reasonable, followed by vitamin K antagonists, regardless of the presence of ICH[161,171,172,175,181,183] *(Class IIa; Level of Evidence B)*. (For further details, refer to "Acute Management and Treatment of CVT: Initial Anticoagulation.")

EFNS guideline on the treatment of cerebral venous and sinus thrombosis in adult patients

K. Einhäupl[a], J. Stam[b], M. -G. Bousser[c], S. F. T. M. de Bruijn[d], J. M. Ferro[e], I. Martinelli[f] and F. Masuhr[a]

Current evidence shows that patients with CVST without contraindications for AC should be treated

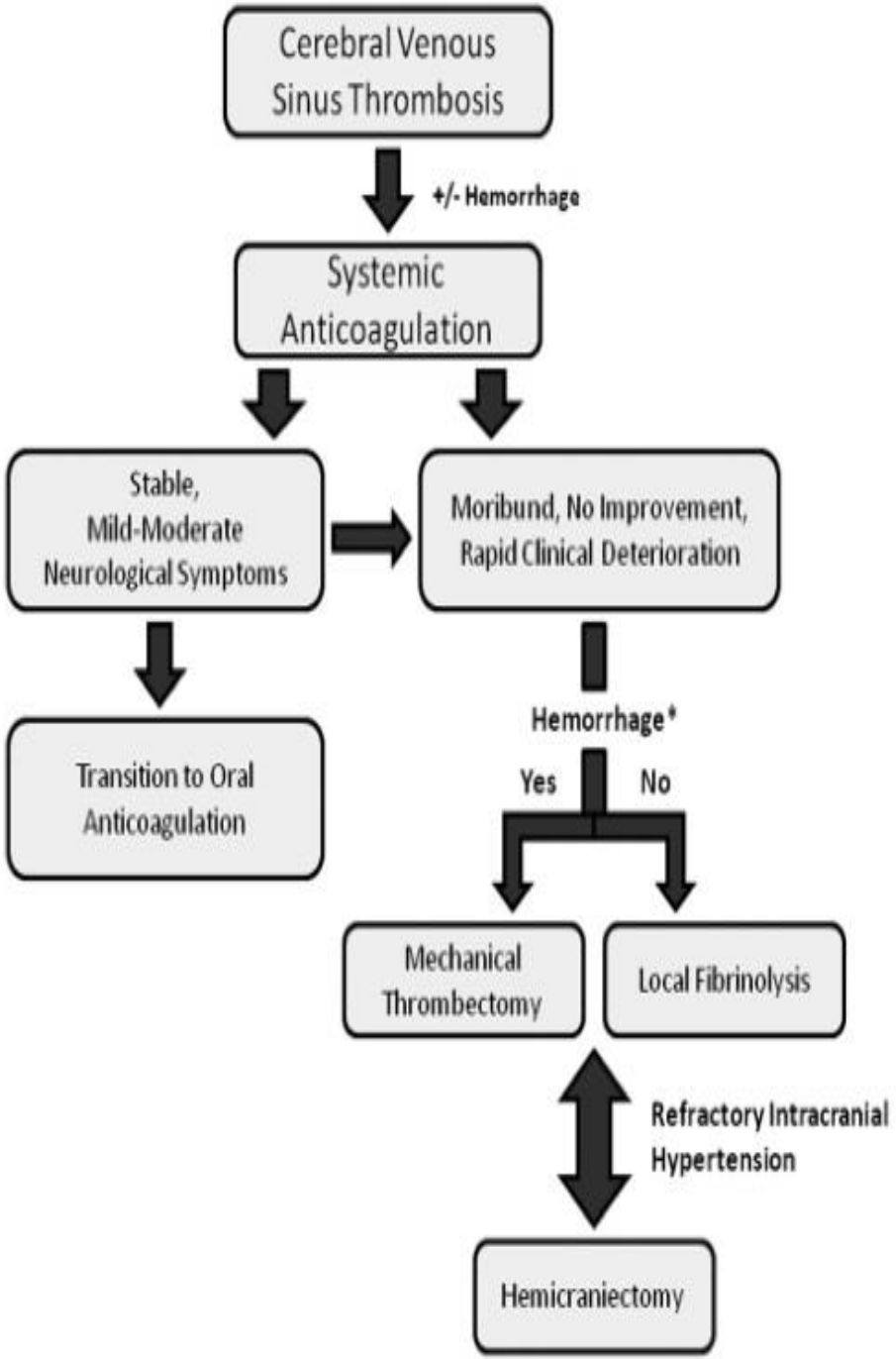
either with body weight-adjusted subcutaneous LMWH or with dose-adjusted intravenous heparin with an at least doubled activated partial thromboplastin time (level B recommendation). Concomitant ICH related to CVST is not a contraindication for heparin therapy. For the reasons mentioned elsewhere, LMWH should be preferred in uncomplicated CVST cases.

NB : HBPM

AOD et TVC

- Dabigatran
- Rivaroxaban

Traitement



Neurosurg Focus 2009 Rahman & al

Thrombectomie

Mechanical Thrombectomy in Cerebral Venous Thrombosis

Systematic Review of 185 Cases

Fazeel M. Siddiqui, MD; Sudeepta Dandapat, MD; Chirantan Banerjee, MBBS;
Susanna M. Zuurbier, MD; Mark Johnson, MD; Jan Stam, MD, PhD; Jonathan M. Coutinho, MD, PhD

Background and Purpose—Cerebral venous thrombosis is generally treated with anticoagulation. However, some patients do not respond to medical therapy and these might benefit from mechanical thrombectomy. The aim of this study was to gain a better understanding of the efficacy and safety of mechanical thrombectomy in patients with cerebral venous thrombosis, by performing a systematic review of the literature.

Methods—We identified studies published between January 1995 and February 2014 from PubMed and Ovid. We included all cases of cerebral venous thrombosis in whom mechanical thrombectomy was performed with or without intrasinus thrombolysis. Good outcome was defined as normal or mild neurological deficits at discharge (modified Rankin Scale, 0–2). Secondary outcome variables included periprocedural complications and recanalization rates.

Results—Our study included 42 studies (185 patients). Sixty percent of patient had a pretreatment intracerebral hemorrhage and 47% were stuporous or comatose. AngioJet was the most commonly used device (40%). Intrasinus thrombolysis was used in 131 patients (71%). Overall, 156 (84%) patients had a good outcome and 22 (12%) died. Nine (5%) patients had no recanalization, 38 (21%) had partial, and 137 (74%) had near to complete recanalization. The major periprocedural complication was new or increased intracerebral hemorrhage (10%). The use of AngioJet was associated with lower rate of complete recanalization (odds ratio, 0.2; 95% confidence interval, 0.09–0.4) and lower chance of good outcome (odds ratio, 0.5; 95% confidence interval, 0.2–1.0).

Conclusions—Our systematic review suggests that mechanical thrombectomy is reasonably safe but controlled studies are required to provide a definitive answer on its efficacy and safety in patients with cerebral venous thrombosis.
(*Stroke*. 2015;46:1263-1268. DOI: 10.1161/STROKEAHA.114.007465.)

Thrombolyse intraveineuse

Systemic Thrombolysis for Cerebral Venous and Dural Sinus Thrombosis: A Systematic Review

Cerebrovasc Dis 2014;37:43–50

L.D. Viegas[a] E. Stolz[b] P. Canhão[a] J.M. Ferro[a]

Conclusions: In all, 88% of the CVT patients treated with systemic thrombolysis regained their independency, but 2 deaths associated with intracranial hemorrhage occurred. The mortality rate and disability at the last available follow-up were similar to those found in 2 previous systematic reviews concerning the use of thrombolytics in CVT. Due to the small sample size and lack of controls, the efficacy of systemic thrombolysis in acute CVT cannot be assessed from the published information. Concerning safety, a nonnegligible proportion of bleedings was reported.

Hémicrâniectomie

1 - Decompressive hemicraniectomy in severe cerebral venous thrombosis: a prospective case series.

Zuurbier et al J.Neurol. 2012 ; 259(6) : 1099-105

Ten patients (8 women) with a median age of 41 years (range 26-52 years). Unilateral hemicraniectomy in 9 patients and bilateral hemicraniectomy in one.

Two patients died . Five patients recovered without disability at 12 months (mRS 0-1). Two patients had some residual handicap (one minor, mRS 2; one moderate, mRS 3). One patient was severely handicapped (mRS 5).

Decompressive hemicraniectomy in the most severe cases of cerebral venous thrombosis was probably life saving in 8/10 patients, with a good clinical outcome in six. In 2 patients death was caused by enlarging hemorrhagic infarcts.

2. Decompressive craniectomy in cerebral venous thrombosis: a single centre experience

Aaron et al.J Neurol Neurosurg Psychiatry. 2013;84(9):995-1000

Over 10 years (2002-2011), 44/587 (7.4%) patients with CVT underwent decompressive craniectomy. Hemicraniectomy was done in 38/44 (86%) and bifrontal craniectomy in 6/44 (13.6%). Mortality was 9/44 (20%). On multivariate analysis (5% level of significance) age <40 years and surgery within 12 h significantly increased survival. Mean follow-up was 25.5 months (range 3-66 months), 26/35 (74%) had 1 year follow-up. Modified Rankin Scale (mRs) continued to improve even after 6 months with 27/35 (77%) of survivors achieving mRs of ≤ 2.

Decompressive craniotomy should be considered as a treatment option in large venous infarcts. Very good outcomes can be expected especially if done early and in those below 40 years.

3. Decompressive surgery for malignant cerebral venous sinus thrombosis: a retrospective case series from Pakistan and comparative literature review.

Raza et al J Stroke Cerebrovasc Dis. 2014 ;23:e13-22.

134 patients were diagnosed with CVST. Of these, 7 received intervention.

Four of the 7 patients had an excellent outcome, 2 of 7 died, and 1 of 7 left against medical advice (in a comatose state) and was lost to follow-up. All those patients who had preoperative reactive pupils with low Glasgow Coma Scale scores made a complete neurologic recovery, and patients with fixed, dilated, and nonreactive pupils preoperatively died in the first postoperative week (P = .05).

Patients who received decompressive hemicraniectomy had excellent outcomes in all cases when intervention was performed with intact preoperative pupillary reflexes. Of the data reviewed, most reported (two-third) patients show the same prognosticators; however, one third show that even with nonreactive pupils complete recovery is possible.

Conclusion

Cerebral venous sinus thrombosis (CVST) rarely induces cerebral hemorrhage, and CVST with cerebral hemorrhage during early pregnancy is extremely rare. Upon literature review, we are able to find only one case of CVST with cerebral hemorrhage in early pregnancy. In this paper, we report another case of a 27-year-old patient who developed CVST with cerebral hemorrhage in her fifth week of pregnancy. Although the optimal treatment for this infrequent condition remains controversial, we adopted anticoagulation as the first choice of treatment and obtained favorable results.

Neurosciences 2015; Vol. 20 (1): 48-51