

DURAL AVF – CLASSIFICATION AND MANAGEMENT

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BRIEF ANATOMY OF DURA MATER

- *Dura mater*, means "tough mother" (Latin - Toughest tissue in the body).
- Embryologically develops from the mesoderm surrounding the neural tube.
- Forms a tough and fibrous outer covering of CNS
- Consists of two layers :
 - Outer or endosteal layer (Endocranium)
 - Inner or meningeal layer – continuous with spinal dura mater

Blood supply of cerebral durameter

A - Vault – Middle meningeal artery

B - Ant. cranial fossa - Ant. Ethmoidal artery

- Post. Ethmoidal artery

- Ophthalmic artery

C - Middle cranial fossa - Middle meningeal artery

- Accessory meningeal artery

- ICA

- Ascending pharyngeal artery

D – Post. cranial fossa - Vertebral artery

- Occipital arteries

- Ascending pharyngeal artery

DURAL AVFs

:INTRODUCTION:

- Abnormal direct connection (fistula) between meningeal artery and a meningeal vein or dural venous sinus within dural leaflets.
- Nidus of AV shunting is contained solely within the dural leaflets, and is distinguished from pial AVM.
- Arterial supply is usually derived from dural arteries and less frequently from osseous branches.

- May drain through an adjacent dural sinus and/or other dural and leptomeningeal venous channels.

- Retrograde leptomeningeal venous drainage is often tortuous and variceal, and may be frankly aneurysmal.

- Broadly divided into –
 - 1 – Intracranial dural AVFs
 - 2 – Spinal dural AVFs

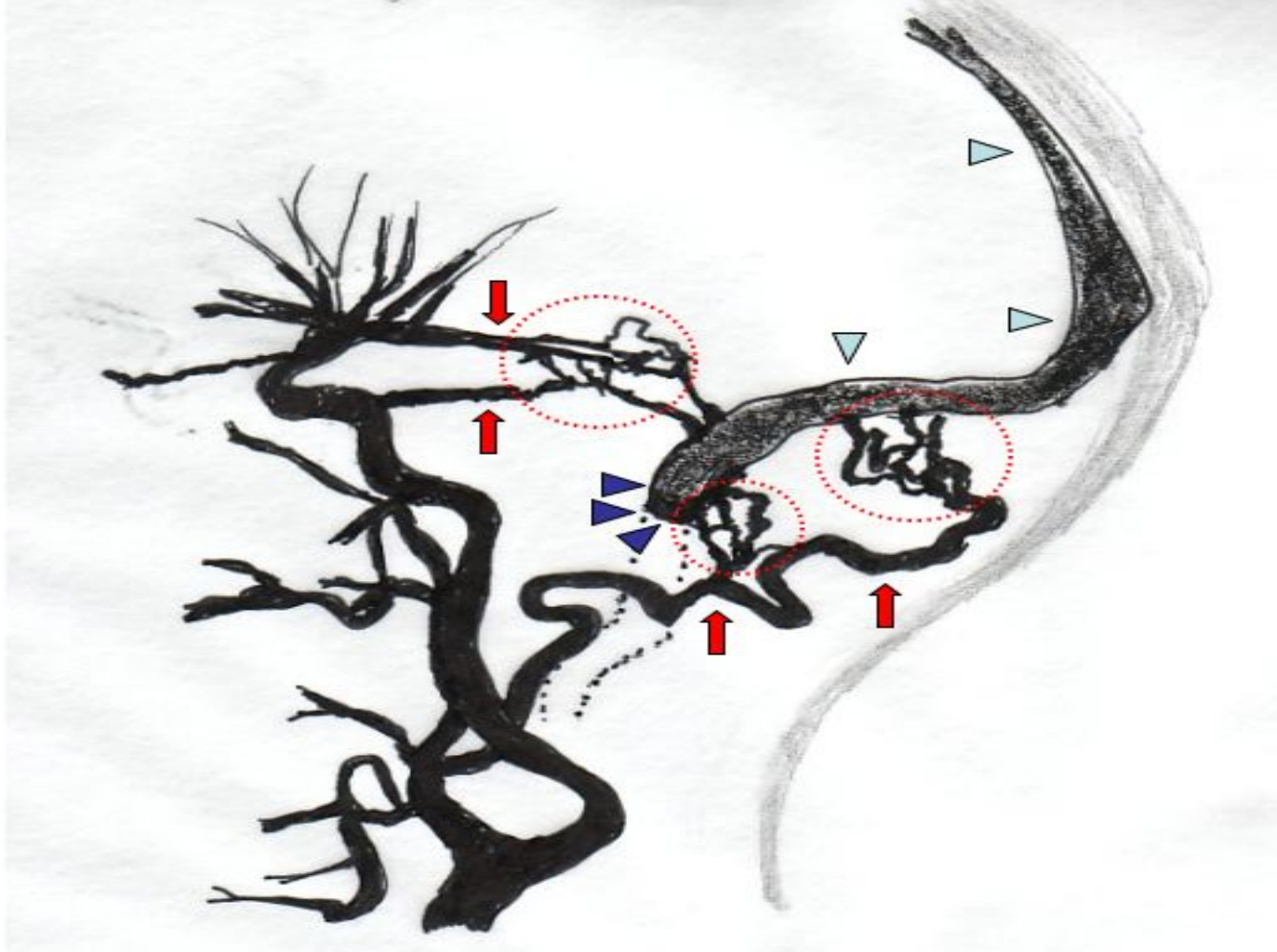


Figure shows a **cranial DAVF** as seen on a side-on or lateral view of a **cerebral angiogram**. The small fistulae are in the **dura** (red circles). The **arterial supply** is marked by the red arrows (branches of the external and internal carotid arteries). In this case, there is no distinct **draining vein**, but there is a **thrombosed dural venous sinus** in the vicinity of the fistulae. The blocked or thrombosed portion of the sinus is shown by dark-blue arrow heads, the normal open portion is shown by light-blue arrow heads.

Intracranial Dural AV F

:EPIDEMIOLOGY: (Brown RJ et al, 1996).

- Incidence= 0.17 per 100,000 persons
- Comprise 10-15% of intracranial vascular malformation.
- Found in 1.1% of consecutive angiograms and one fifth as common as AVMs.

- Usually solitary (Multiple in $< 7\%$ cases, Malik et al, 1988)
May occur anywhere in the dura mater covering the brain (M.C. site - CS and T-S sinuses).
- Usually located within or near the wall of a dural venous sinus, which is often narrowed or obstructed.
- Most commonly involve occipital and meningeal arteries.

ETIOLOGY

- May be due to :
 - Idiopathic – Most common
 - Sinus thrombosis
 - Head trauma : linear # across T.-sinus
 - Cranial surgery
 - Hormonal influence
 - Sinusitis : in CCF
 - Congenital (rare)

- May associated with:
 - Meningioma
 - Ehlers-Danlos syndrome
 - Fibromuscular dysplasia
 - NF-1

NATURAL HISTORY

Highly variable

- No or benign symptoms for many years.
- May be incidental finding on angiogram.
- May exhibit aggressive behaviours.
- Benign DAVFs may spontaneously thrombose, occasionally after angiography or compression.
- Acute or gradual shift of grade may occur.

- Overall risk of hemorrhage – 1.6% / yr (Brown et al.)
- Annual morbidity and mortality rates of with an aggressive presentation differ widely (1.8% to 20% per year).
- Risk of re bleed – 35% within 2 weeks (Duffau et at.1999).

A largest prospective natural history study:

(Van Dijk et al, 2002)

- Total pt.= 236
- Two groups :

A – Pt. with CVR :

N = 119

- Successful curative treatment = 96
- Lost follow up = 3

➤ Rest 20 pt.:

- Refused to treatment = 14
- Partial treatment = 6

Mean follow up = 4.3 yr

➤ **RESULT :**

- Annual risk of ICH and NHND = 15 % (8.1+6.9)
- Annual mortality rate = 10.4%

B – Pt. without CVR :

N =117

- Lost follow up = 5
- Rest 112 pts. median follow up = 27.9 months
- Only observation = 68
- Palliative treatment =44
(Endovascular = 43,Surgery=1)

➤ **RESULT:**

- Pts. with benign and well tolerated clinical course without any ICH or NHND =98%
- Long term angiographic follow up (N=50) shows 2-3% of risk of developing CVR

Pathophysiology

- Mostly acquired, idiopathic lesion.
- Varies in complexity from a single shunt in small region of the dura to an extensive malformation.
- **Two theories for origin :**
 - 1- Opening of preexisting microscopic vascular channels within duramater after venous hypertension secondary to sinus thrombosis.

2 - Formation of new vascular channels within duramater, a process stimulated and regulated by angiogenic factors.

(Pathology specimen contain- Angiogenic growth factor and basic fibroblast growth factor)

- With aggressive symptoms, lesions seem to grow by recruitment of arterial feeders into nidus, favored by the AV shunting (Sump effect . Awad et al,1990).
- Hypertrophy of dural arteries and reappearance of involuted embryonic arteries occur.

- Formed as a consequence of thrombosis and subsequent recanalization of dural venous sinuses.
- Sinus thrombosis seems to be related to either initiating or healing phases of the disease progression.
- Postmenopausal and pregnant females more commonly experience CS and TS DAVF - thus possibility of a hormonal influence can not be ruled out.

- Progressive arterialization of the pathologic dural leaflet results in hypertension in adjacent leptomeningeal channels.
- Thus retrograde leptomeningeal venous drainage may occur.
- Under arterialized pressure, these venous channels may become tortuous and eventually, varicose or aneurysmal.
- Demyelination may occur around leptomeningeal veins due to venous hypertension.

LOCATION

➤ Common locations of dural AVFs:

1 = Cavernous sinus (*CS*) (20%-40% of cases)

2 = Transverse-sigmoid sinus (*TS, SS*) (20%-60%)

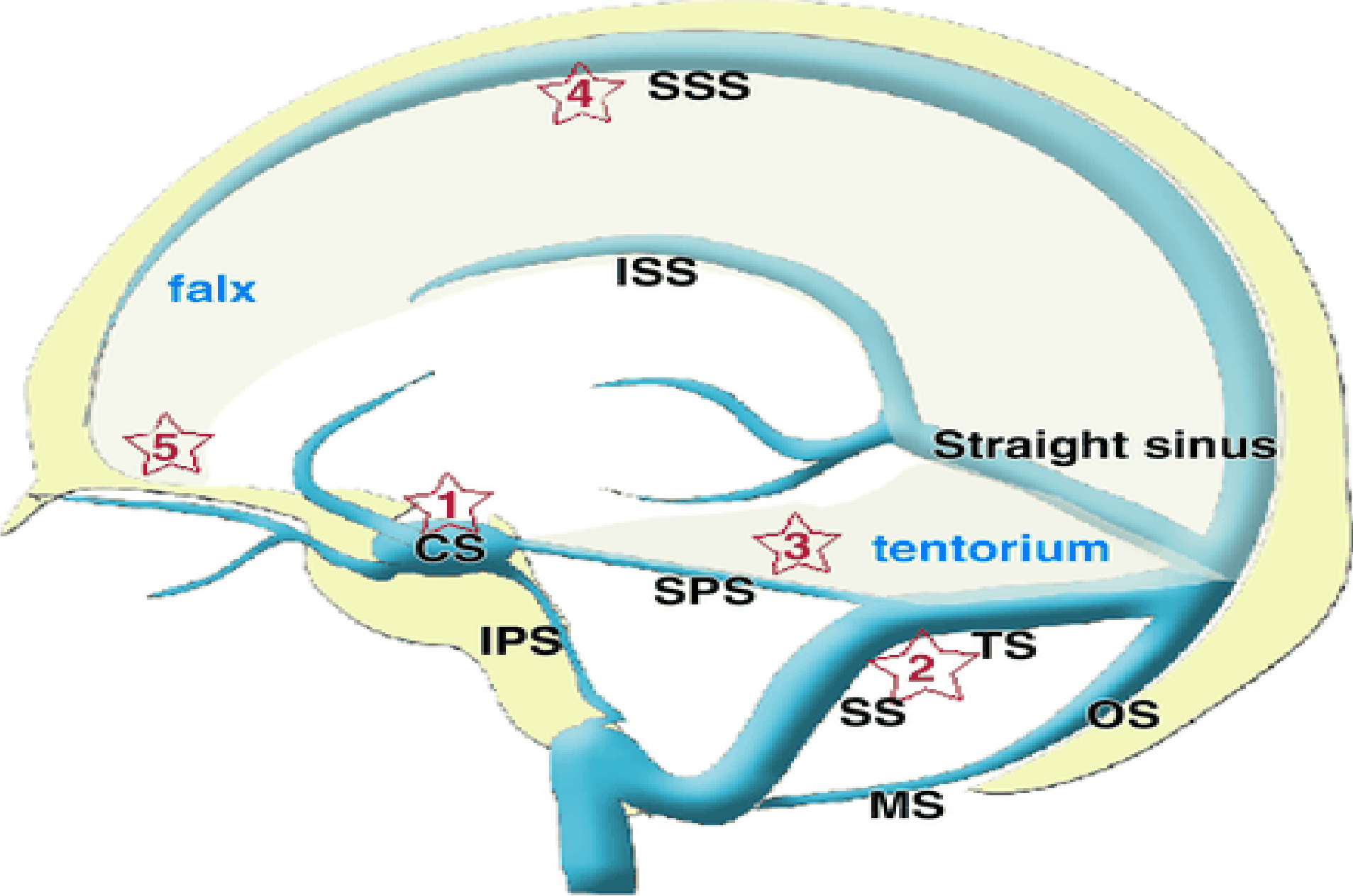
3 = Tentorium (12%-14%),

4 = Superior sagittal sinus (*SSS*) (8%), and

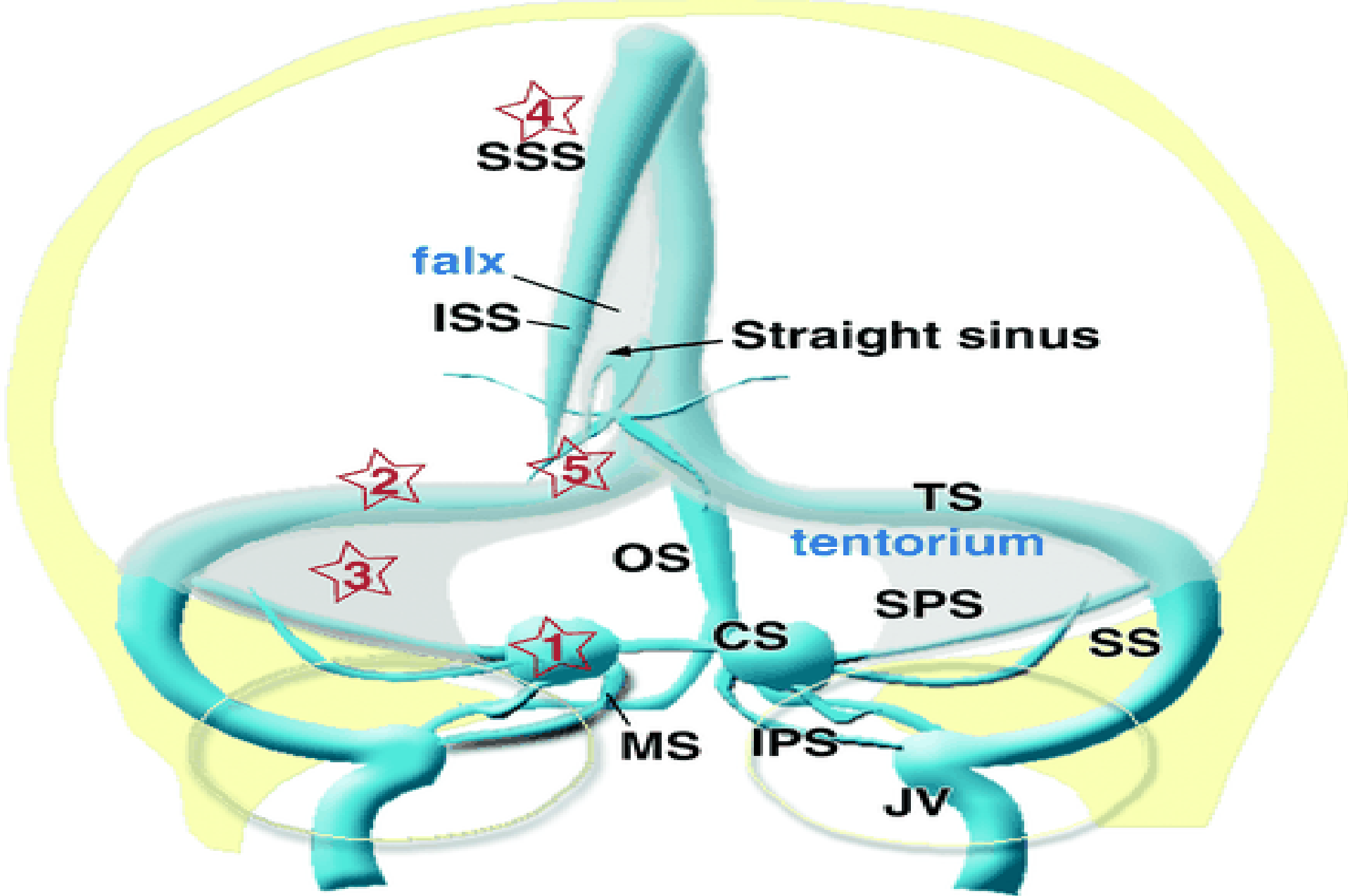
5 = Anterior fossa (2%-3%).

➤ CCF and T-S D-AVFs more common in female.

➤ Ant. Cranial fossa and Tentorial D-AVFs more in males.



(IPS = inferior petrosal sinus, ISS = inferior sagittal sinus, JV = jugular vein, MS = marginal sinus, OS = occipital sinus, SPS = superior petrosal sinus.)



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Classification

➤ Most classification systems are based on :

- 1- Presence of venous stasis or occlusion
- 2- Direction of flow
- 3- Presence of leptomeningeal venous drainage, aneurysm and single or multiple fistulas.

Classification of Intracranial Dural Arteriovenous Malformations

Bases of Classification	References	Categories	Characteristics
Sinus location	Aminoff (1)	Anterior-inferior group Superior-posterior group	Drain into cavernous, sphenoparietal, sup. and inf. petrosal ss. Drain into sup. and inf. sagittal, straight, lateral, sigmoid, occipital ss.
	Awad et al. (4)	Lateral-sigmoid sinus	Most common and most benign of dural AVMs. Present with bruit in 70%.
	Lasjaunias et al. (31) Mullan (36)	Cavernous sinus Base of anterior cranial fossa Superior sagittal sinus Tentorial incisura-straight sinus Other regions (uncommon)	Discussed elsewhere under carotid cavernous fistulas. Often present with hemorrhage. Treatment is surgical. Often associated with sinus injury. Venous hypertension is common. Virtually all present with hemorrhage or focal neurologic deficit, e.g., sup. and inf. petrosal ss., sylvian, torcular, and foramen magnum.
Pattern of venous drainage	Djindjian and Merland (10)	Type I Type II Type III Type IV	Drain into a sinus with flow in the normal anterograde direction. Drain into a sinus. Flow is retrograde from sinus to pial veins. Drain directly into leptomeningeal veins. Drain into a dural venous ectasia.
	Piton et al. (38)	Type (or stage) I Type II Type III	Venous outflow is anterograde. Venous outflow is retrograde, but there is no pial venous efflux. Venous outflow is retrograde with efflux into pial veins.
Arterial supply	Kühner et al. (29)	External Intermediate Internal	Supplied by extracranial branches of external carotid and cervical vertebral aa. Supplied by dural (middle and posterior meningeal) aa. Supplied by dural branches of cerebral aa. (e.g., tentorial artery).

TABLE 127-4 ■ Classification and Grading of Dural Arteriovenous Fistulas

GRADE	DESCRIPTION	NUMBER	HEMORRHAGE
Cognard²⁵⁸			
1	Dural venous sinus and meningeal vein outflow, anterograde	84	1 (1.2%)*
2a	Dural venous sinus and meningeal vein outflow, retrograde	27	10 (37%)*
2b	Dural venous sinus and meningeal vein outflow (anterograde) and retrograde leptomeningeal venous drainage	10	3 (30%)*
2a + b	Dural venous sinus and meningeal vein outflow (retrograde) and retrograde leptomeningeal venous drainage	18	12 (67%)*
3	Retrograde leptomeningeal venous drainage only (no ectasia)	25	19 (76%)*
4	Retrograde leptomeningeal venous drainage only (ectasia)	29	28 (97%)*
5	Retrograde leptomeningeal venous drainage only (spinal)	12	12 (100%)*
Border²⁷³			
1	Dural venous sinus and meningeal vein outflow	55	0 (0%)
2	Dural venous sinus and meningeal vein outflow and retrograde leptomeningeal venous drainage	18	2 (11%)
3	Retrograde leptomeningeal venous drainage	29	14 (48%)
Lalwani²⁷¹			
1	Anterograde sinus drainage; no venous restrictive disease; no retrograde or cortical drainage	—	0
2	Anterograde and retrograde venous sinus drainage with or without cortical drainage	—	0
3	Retrograde and cortical drainage without anterograde sinus drainage	—	31%
4	Cortical venous drainage only	—	100%

CLINICAL FEATURES

- More common in adults (Age= 40-60 yr).
- May be asymptomatic or benign symptoms to fatal hemorrhage.
- Two groups of symptoms –
 - A- Non aggressive : -Tinnitus (pulsatile)- M.C.
- Headache
 - B- Aggressive : -Intracranial hemorrhage
-Non hemorrhagic neurological deficits

Symptom	Location				
	Cavernous Sinus (%)	Transverse-Sigmoid Sinus (%)	Tentorium (%)	Superior Sagittal Sinus (%)	Anterior Fossa (%)
Ocular symptoms	80-97
Cranial nerve deficits	44-77	7-12	14-17
Bruit, tinnitus	40-50	40-42	70-88
Headache	...	46-76	8-24	50	12-15
Visual symptoms	28-38	12-28
Central nerve deficits	3	10-20	23-42	29	5-33
Intracranial hemorrhage	Rare	15-28	60-74	23	44-84
Dementia	...	Rare	...	5	...

Symptoms of cranial dural AVF

➤ **Tinnitus :**

- Pulsatile
- With high flow fistula in TS or SS location
- Due to recruitment of arterial feeders and close proximity to middle ear
- Bruit can be auscultated
- Compression of carotid artery, jugular vein or occipital artery may decrease the bruit intensity.

➤ **Headache :**

- U/L or generalized
- Increase on physical activity or position change
- Due to .. Engorgement of venous collaterals and dural sinus distention, edema, compression of 5th nerve or inflammation d/t venous thrombosis.

➤ Hemorrhage :

- May be
 - Intra ventricular
 - Intraparenchymal
 - Subarachnoid
 - Subdural

- May be distant from the fistula site.

➤ Most predictive predisposing factors for aggressive symptom are :

1 - Venous drainage pattern

- Cortical venous drainage
- Venous ecstasies
- Galenic venous drainage

2- Location

- Tentorial incisura (I.=8.4%)
- Ant. Cranial fossa

➤ At the transverse-sigmoid sinuses and cavernous sinuses
- least likely associated with aggressive symptoms.

➤ Any change in tinnitus or bruit warrants re-evaluation.

INVESTIGATION:

1- CT :

- N
- Hemorrhage
- Hcp

2- MRI and MRA :

- N
- Dilated pial vessels
- White matter edema
- Stenosis or occlusion of dural sinuses

3- Intra arterial catheter angiography

- Gold standard
- Premature visualization of intracranial veins or venous sinuses during arterial phase- Characteristic
- Best views of nidus are often obtained during the ultra early arterial phase and by injections of distant arterial feeders.
- Exact site of CVR must be determined

MANAGEMENT OPTIONS

- 1 - Observation
- 2 – Transarterial Embolisation
- 3 – Transvenous Embolisation
- 4 – Surgery:
- 5 - Stereo tactic Radio surgery

➤ ***OBSERVATION:***

- In lesions without CVR
- Asymptomatic or tolerable symptoms
- Follow up with serial MRI, MRA and DSA after 3 yr
- 2-3% chance of developing CVR
- Any change in symptoms needs evaluation.

➤ ***TRANSARTERIAL EMBOLIZATION :***

- Rarely curative
- Best agent : n-BCA
- Commonly used as an adjuvant before surgery or TVE
- Palliative treatment for benign lesions with intolerable symptoms

Complications:

- Stroke
- conversion of benign fistula into aggressive one

➤ ***TRANSVENOUS EMBOLIZATION :***

- Through transfemoral route
- Sacrifice the involve dural venous sinus
- Only done when venous phase of DSA shows that the involved sinus is not used by brain and alternate pathway for venous drainage is developed

Complications :

- Venous hypertension and infarction
- SAH

➤ **SURGERY:**

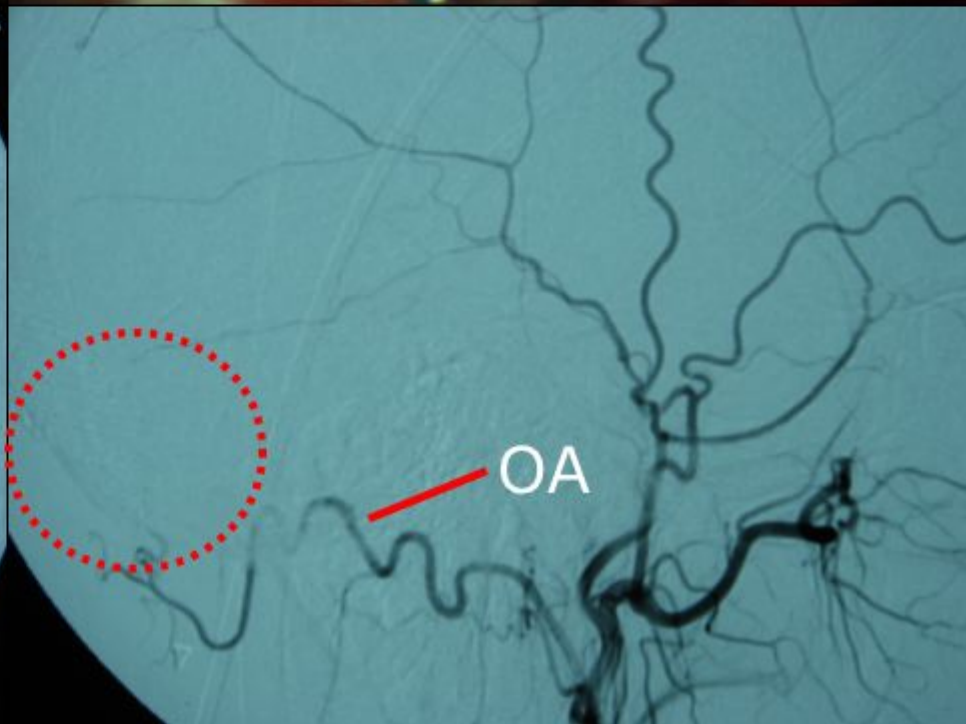
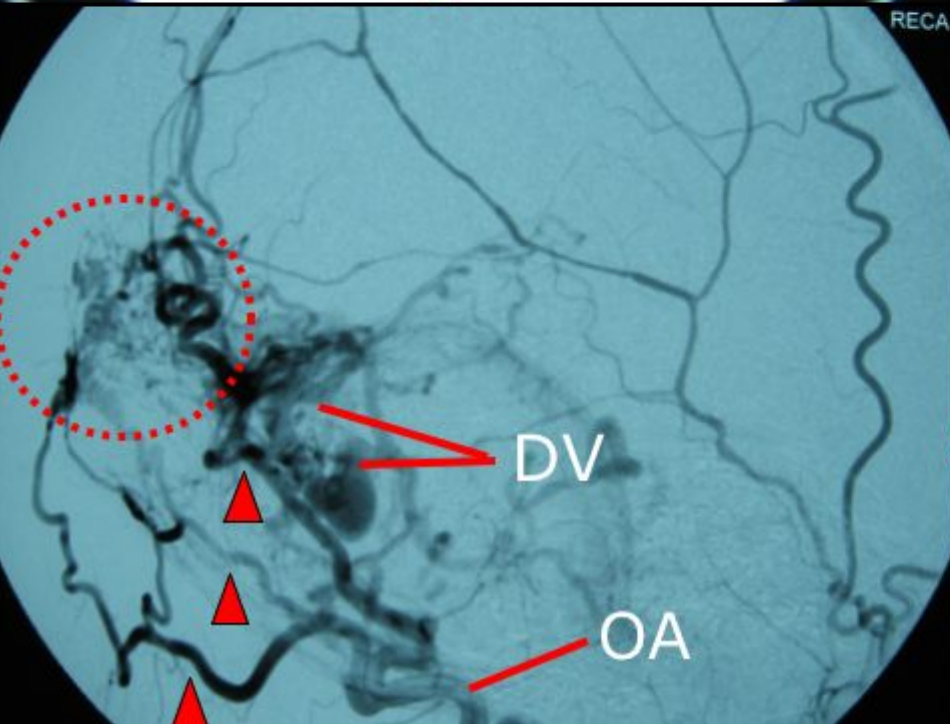
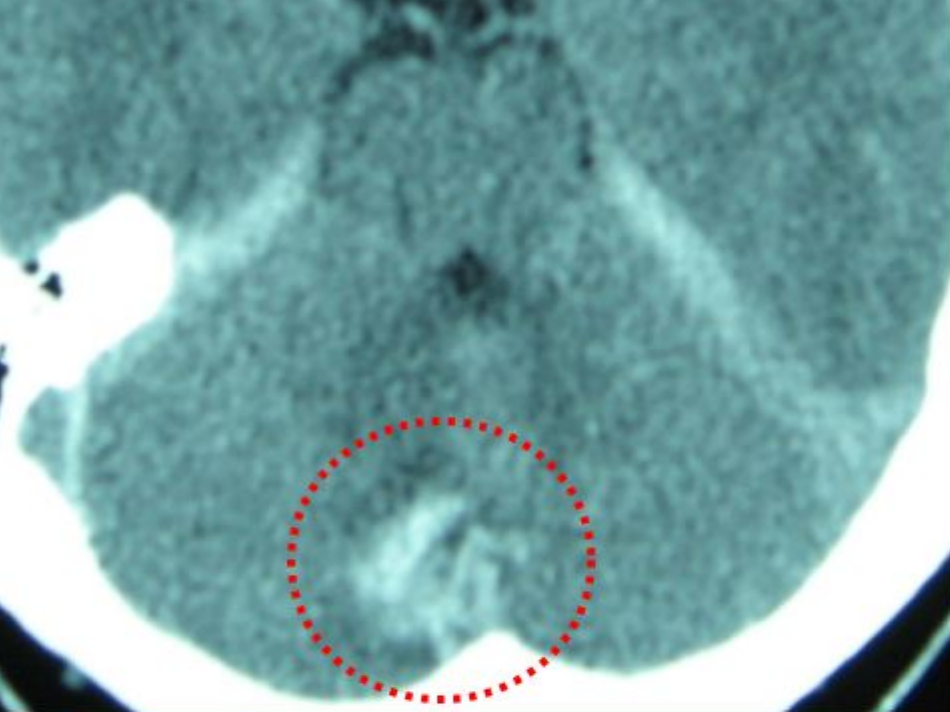
Indication –

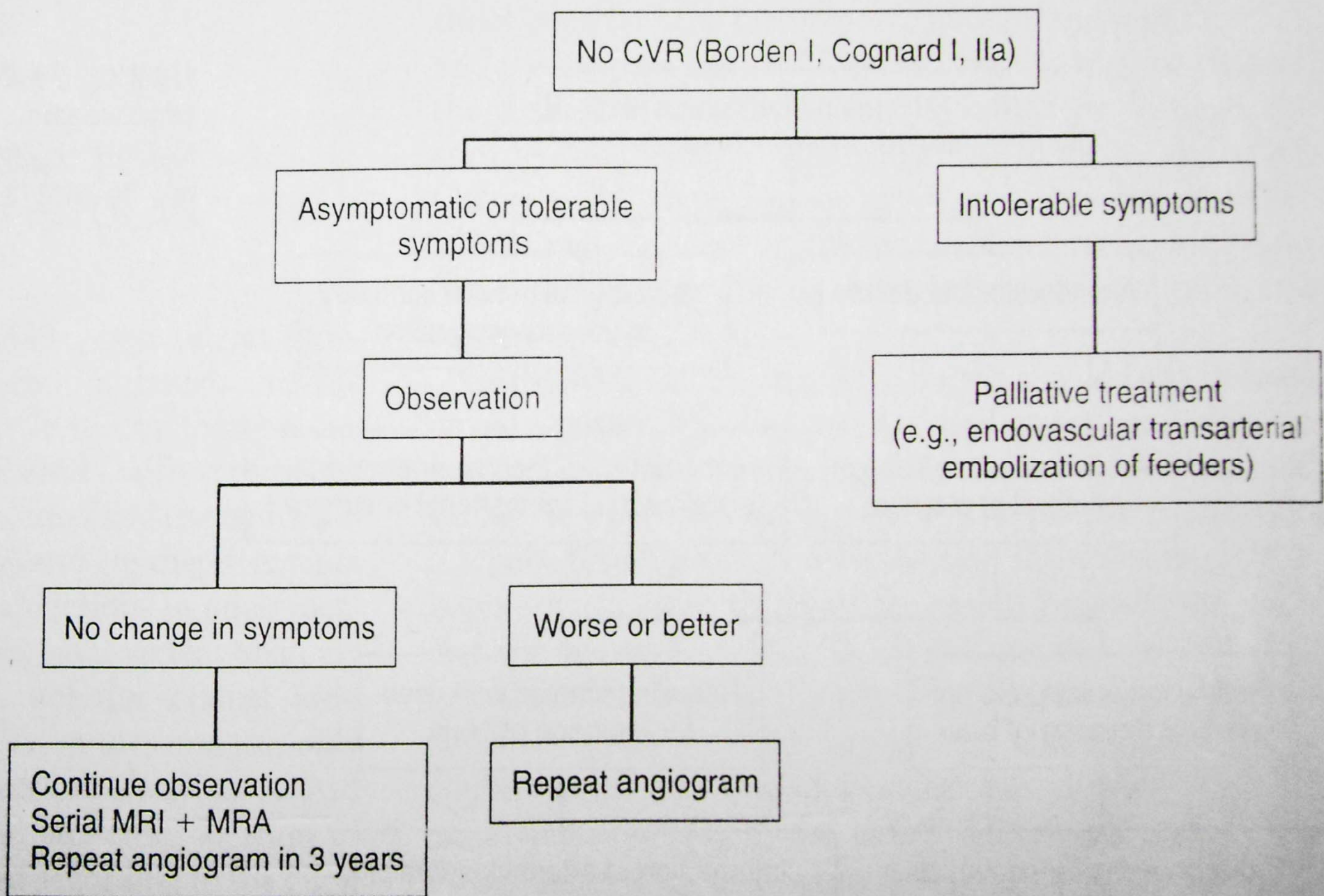
- When endovascular therapy fails
- When endovascular therapy not feasible

- **3 strategies :**
 - 1- Obtain venous access for direct packing
 - 2- Complete excision of AVF
 - 3- Disconnection of CVR alone

Complications:

- 1- Technically difficult
- 2- Risk of exsanguination
- 2- Risk of venous infarction





Management Strategy for D-AVF without CVR

CVR and sinosal drainage (Borden II, Cognard IIb, IIa+b)

No neurological deficit

Neurological deficit secondary
to venous congestion

Assess venous phase
of cerebral angiogram

Interruption of feeding arteries only
(endovascular transarterial or surgical)

Sinus not used by brain

Sinus used by brain

Complete obliteration or excision
of lesion including sinus sacrifice
OR
CVR disconnection only
(endovascular transvenous or surgical)

CVR disconnection only
(endovascular transvenous or surgical)

Management Strategy for D-AVF with CVR

CVR only (Borden III, Cognard III, IV, V)

No neurological deficit

Neurological deficit secondary to venous congestion

Assess venous phase of cerebral angiogram

Interruption of feeding arteries only (endovascular transarterial or surgical)

Refluxing cortical vein not used for drainage of brain

Refluxing cortical vein used for drainage of brain

CVR disconnection (endovascular transvenous or surgical)

Interruption of arterial feeders only (endovascular transarterial or surgical)

Management Strategy for D-AVF with CVR and sinus drainage

TABLE 250-2 Treatment Approaches to Intracranial DAVMs.

Expectant or Symptomatic Treatment	Transarterial Embolization	Transvenous Obliteration	Open Surgery	Stereotactic Radiosurgery
Lesions with nondisabling symptomatology; incidentally discovered lesions	Accessible arterial feeders; may use chemical/sclerosing agents	Accessible venous drainage (via patent sinus or by direct puncture of venous varices)	Lesions with leptomeningeal venous drainage and/or recent aggressive neurological symptoms	Lesions near noneloquent brain; no recent hemorrhage
Lesions without leptomeningeal venous drainage	Rarely "curative"; may palliate disabling flow-related symptoms	Curative with occlusion of associated sinus	Typically curative; enhanced by preparatory embolization or open transvenous obliteration	Delayed effectiveness (does not protect against early rebleeding)
Close follow-up of lesions to exclude development of leptomeningeal venous drainage	Close follow-up of residual DAVM as in untreated lesions	Sudden obliteration may be associated with hemodynamic or thrombotic complications	Maximal resection and/or coagulation of pathologic dural leaflet; interruption of leptomeningeal arterialized veins	Close follow-up to verify effectiveness
Common approach to most transverse-sigmoid or cavernous sinus DAVMs	Useful as preparatory adjunct before transvenous, open, or radiosurgical treatment	Useful in cavernous sinus and transverse-sigmoid DAVMs	Useful for incisural, anterior fossa, and foramen magnum DAVMs	Useful for lateral tentorial, convexity, and middle fossa DAVMs

STEREOTACTIC RADIOSURGERY

- Recent studies have demonstrated promising results in selected pts.
- Pan and colleagues (2002) demonstrated complete angiographic resolution of the nidus in 47% of 19 T-S sinus DAVFs (Median follow up=19 month).
- O,leary S et al (2002) in 17 pts of various sites lesion, demonstrated complete obliteration in 10 pts.
 - 2 pts showed considerable reduction in size.
 - 1 pts showed deterioration.
 - In 4 pts, no angiographic follow up available

- Koebbe CJ et al, (2005) in 18 pts (10 pt with embo) with mean follow up of 43 months and dose 20 GY showed-
 - Angiographic obliteration in all 8 cases
 - No e/o lesion in 4 pts and decreased size in 3 pts assessed by MRA/CTA.
 - 2 pts developed neurological deficits after embolization before radiosurgery.
 - Temporary hemiparesis in 1 pts.

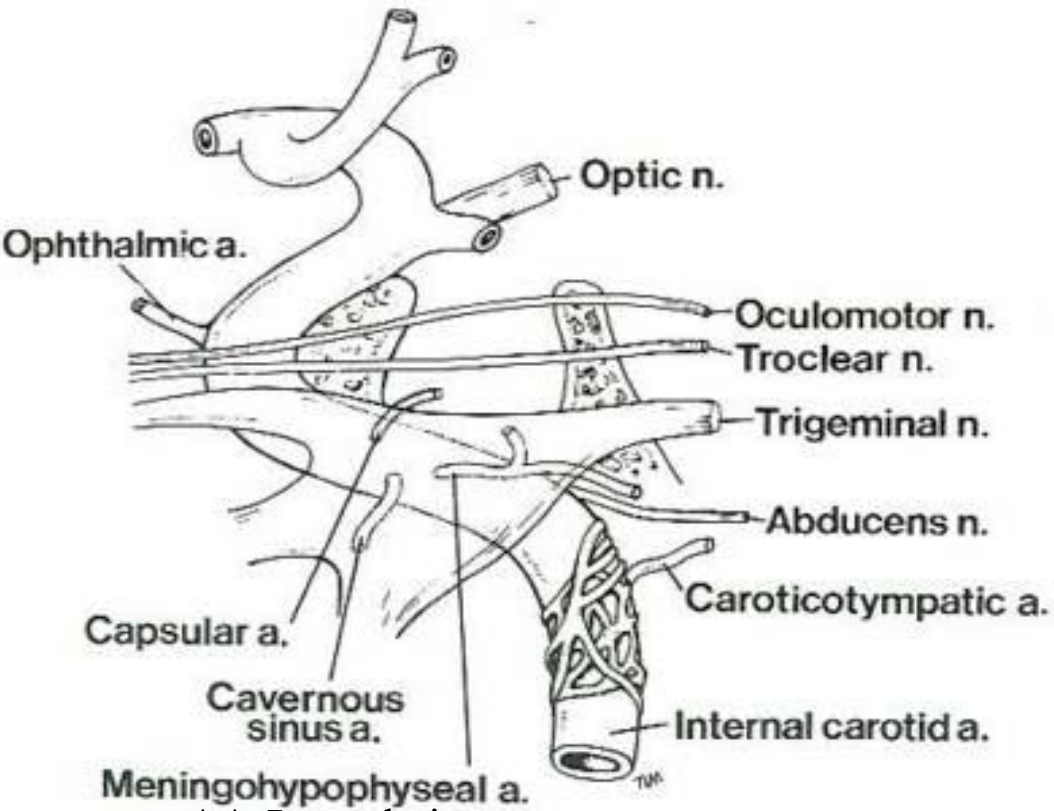
➤ ***LIMITATIONS:***

- 1- Long interval b/w treatment and therapeutic effect
 - Not acceptable in pts with CVR
- 2- Without CVR, most pts can be managed with observation alone or palliative transarterial embolization.

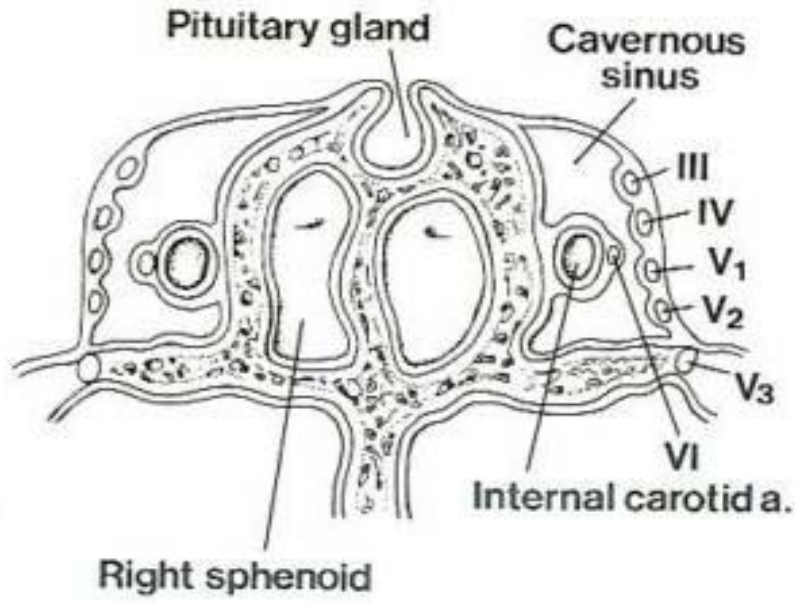
CAVERNOUS SINUS DURAL AVFs

- Cavernous Sinus is the 2nd m. c. involved region.
- First recorded treatment by Travers in 1809.
(Ligation of CCA)
- Dandy (1935) performed a craniotomy and Clipped the ICA just proximal to p-com artery.
- Nunnally (1865) and Rivington (1874) described the pathology and site of lesion.

- Parkinson (1965) opened the CS and directly occlude the fistula.
- Serbinenko (1974) developed and used a detachable balloon for vascular occlusion.
- Guglielmi (1991) introduced soft detachable and retrieval platinum coils.



AA-Lateral view



Relation of structures in cavernous sinus

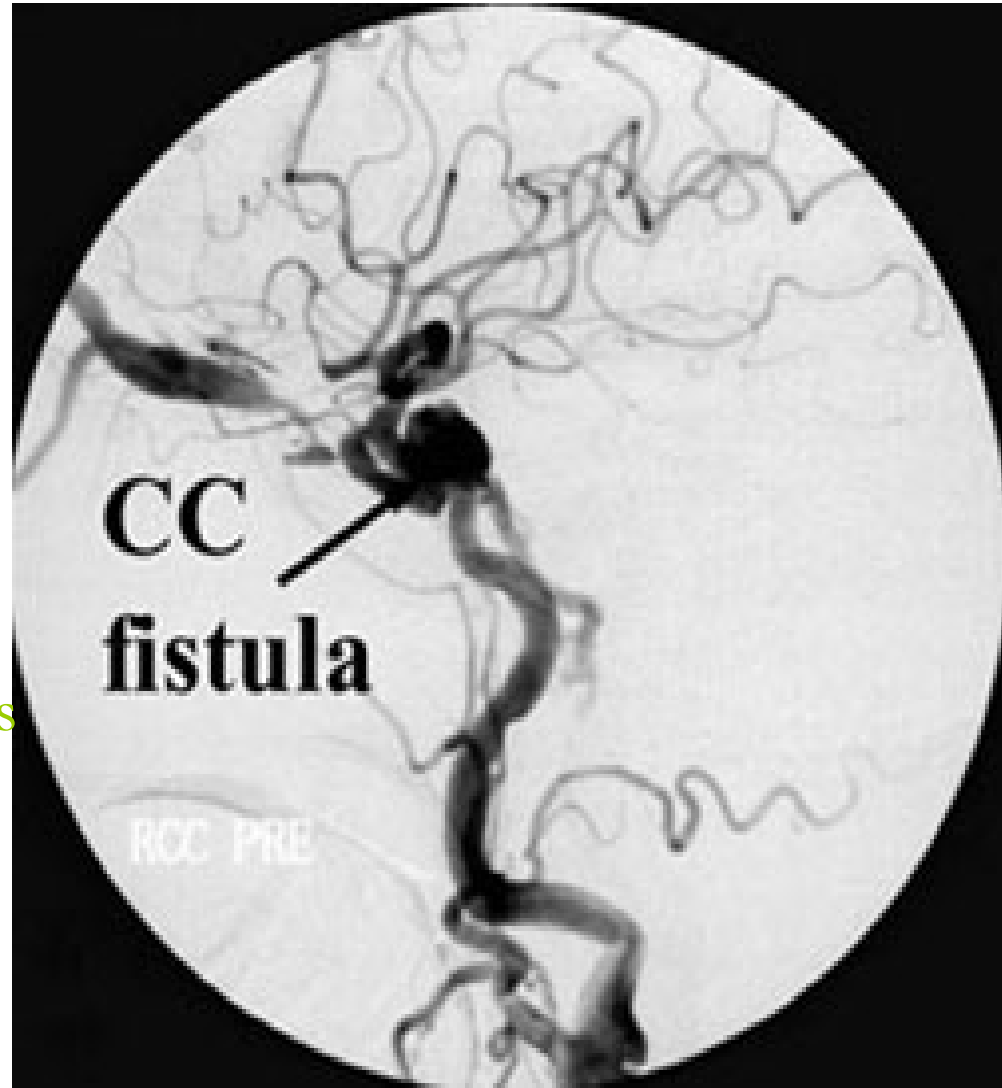
Classification of CCF

Direct CCF (High-flow)

Most common (70-90%)
75% traumatic (assoc. basal skull fx)
Defect in intra-cavernous ICA
Spontaneous: mid-age, HTN, post-menopausal female

Indirect CCF (Low-flow)

Dural Shunt: Meningeal arteries (ICA, ECA or combo)
Can be spontaneous or traumatic also
More insidious onset of symptoms



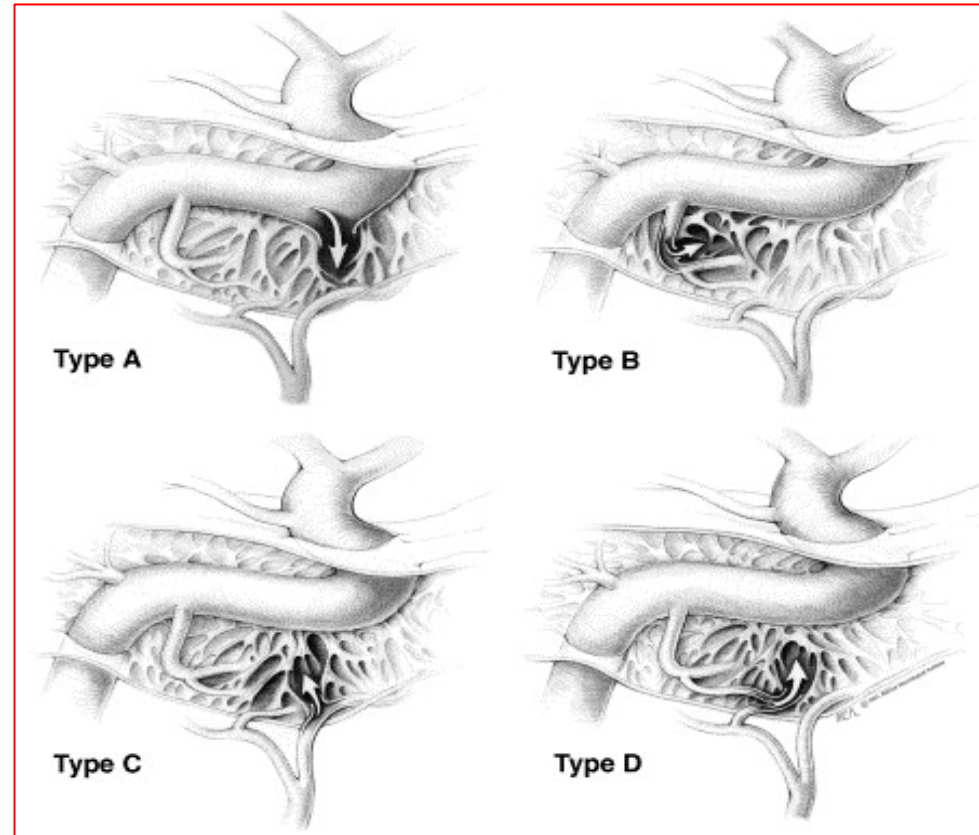
Barrow's Classification (1985)

Type A: Direct between ICA and CS

Type B: Dural ICA branches to CS (uncommon)

Type C: Dural ECA branches to CS

Type D: Dural ICA & ECA branches to CS



Angiographic Pattern Classification

(Dae Chul Suh et al. : 2005)

- Categorized by the degrees and patterns of prominent arteriovenous shunt as well as venous flow.

Type – 1: Proliferative type (40%)

Type – 2: Restrictive type (40%)

Type – 3: Late restrictive type (20%)

- Principle ECA feeders are :
 - 1- Int. maxillary artery – distal branches
 - 2- MMA – meningeal branch
 - 3- Accessory meningeal artery – meningeal branch
 - 4- Ascending pharyngeal artery- meningeal branch

- ICA feeders – Dural branches from cavernous segment-C5 branches and the inferolateral trunk.

Carotid Cavernous Sinus Fistula

- Symptoms:

- Double vision
- eyelid droop
- facial pain/numbness

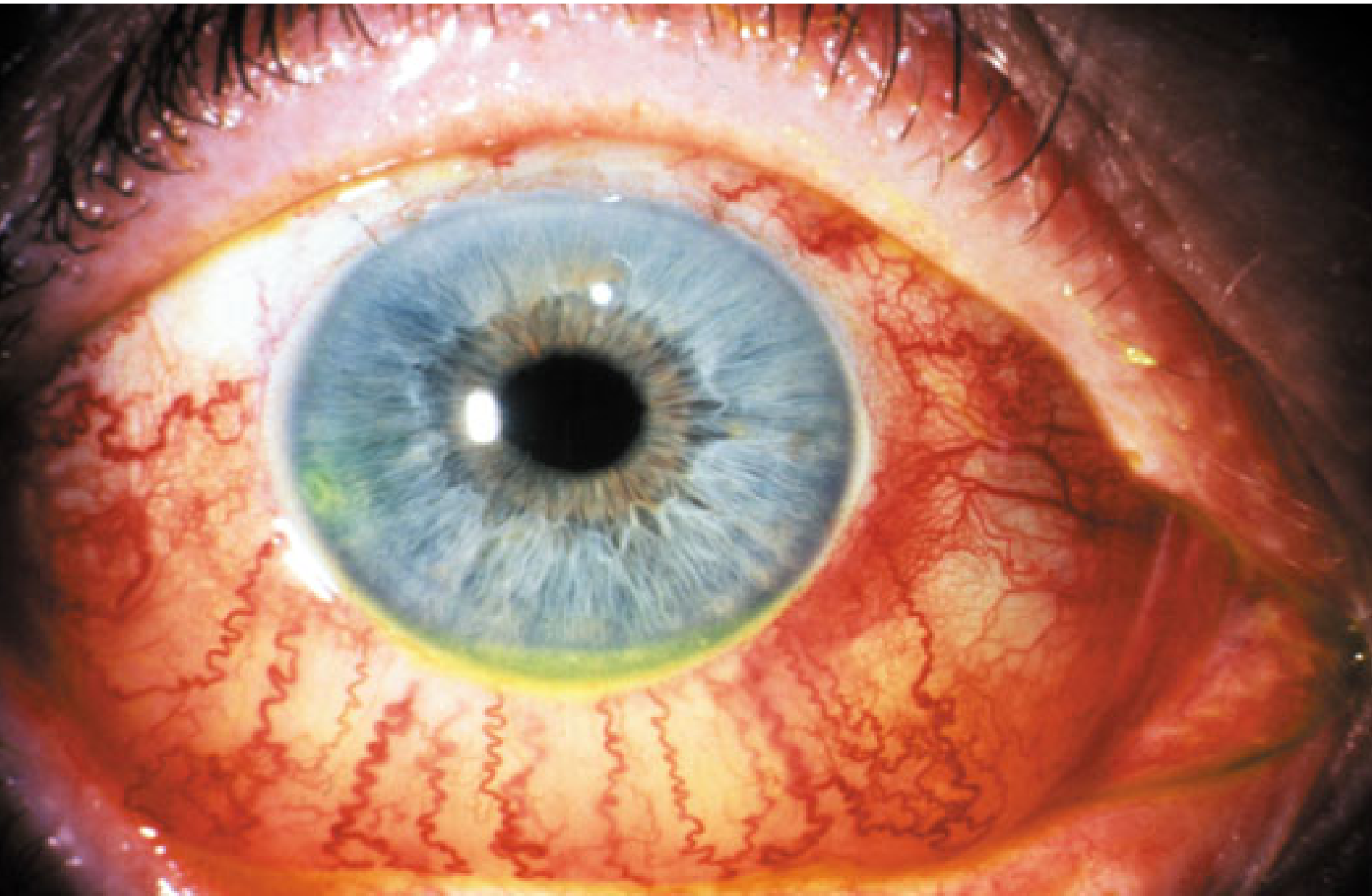
- Signs:

- Proptosis
- chemosis
- ↑ IOP
- ocular pulse pressure
- orbital/temple bruit
- ptosis
- miosis
- ophthalmoplegia (most commonly CN VI)
- facial hypoesthesia
- optic disk swelling
- retinal venous dilatation
- intraretinal hemorrhage



Classic Sign:

Limbal injection with arterialized conjunctival & episcleral vessels



- Symptoms and signs are usually benign because CS has sufficient venous drainage routes.
- Because the symptoms are very diverse and they fluctuate, analysis of the symptomatology related to the angiographic findings does not always correspond to the disease status.
- Symptoms depend on the direction of venous drainage.

- Severity depends on the rate of blood flow through shunt.
- C/L eye symptoms in 11% of patients.
- More common in
 - Postmenopausal elderly women
 - Pregnancy
 - Sphenoid sinusitis
 - Trauma
- Over all mortality = 1-2%

Pathophysiology

➤ Retrograde venous drainage into orbit :

–Venous HTN

→ Enlarged EOM's

→ Restriction → Diplopia

→ Proptosis → Exposure keratopathy

→ Chemosis & Injection → Red Eye

→ Increased episcleral & vortex venous pressure

→ Increased IOP → Secondary Glaucoma

–Venous & Arterial Stasis

→ Decreased ocular/retinal perfusion

→ Decreased visual acuity

→ Anterior Segment Ischemia

→ Decreased perfusion to intra CS cranial nerves

→ Ophthalmoplegias → Diplopia

Pathophysiology

- Venous HTN in other directions may also occur -
 - Retrograde cortical venous drainage
 - 10-55% of CCF cases
 - Severe HA
 - Contralateral neurological deficits
 - 30-40% risk of intracerebral hemorrhage
 - May be fatal

Diagnostic Studies

Orbital color Doppler U/S:

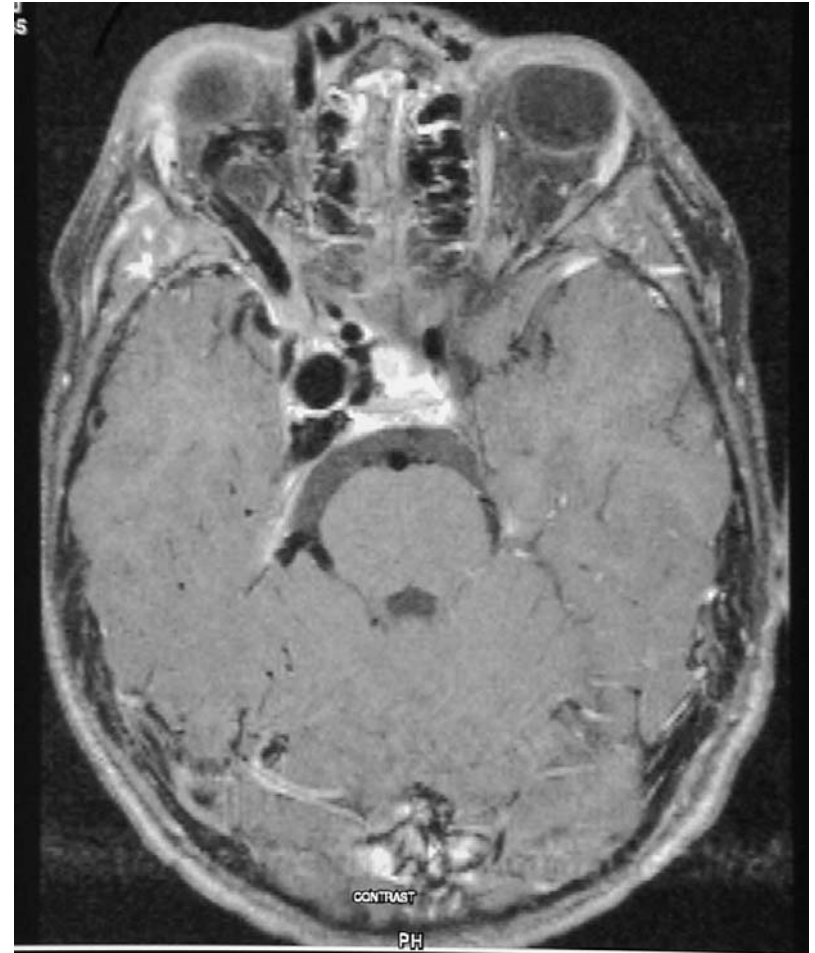
- Reversed, arterialized flow in S. Ophthalmic v.

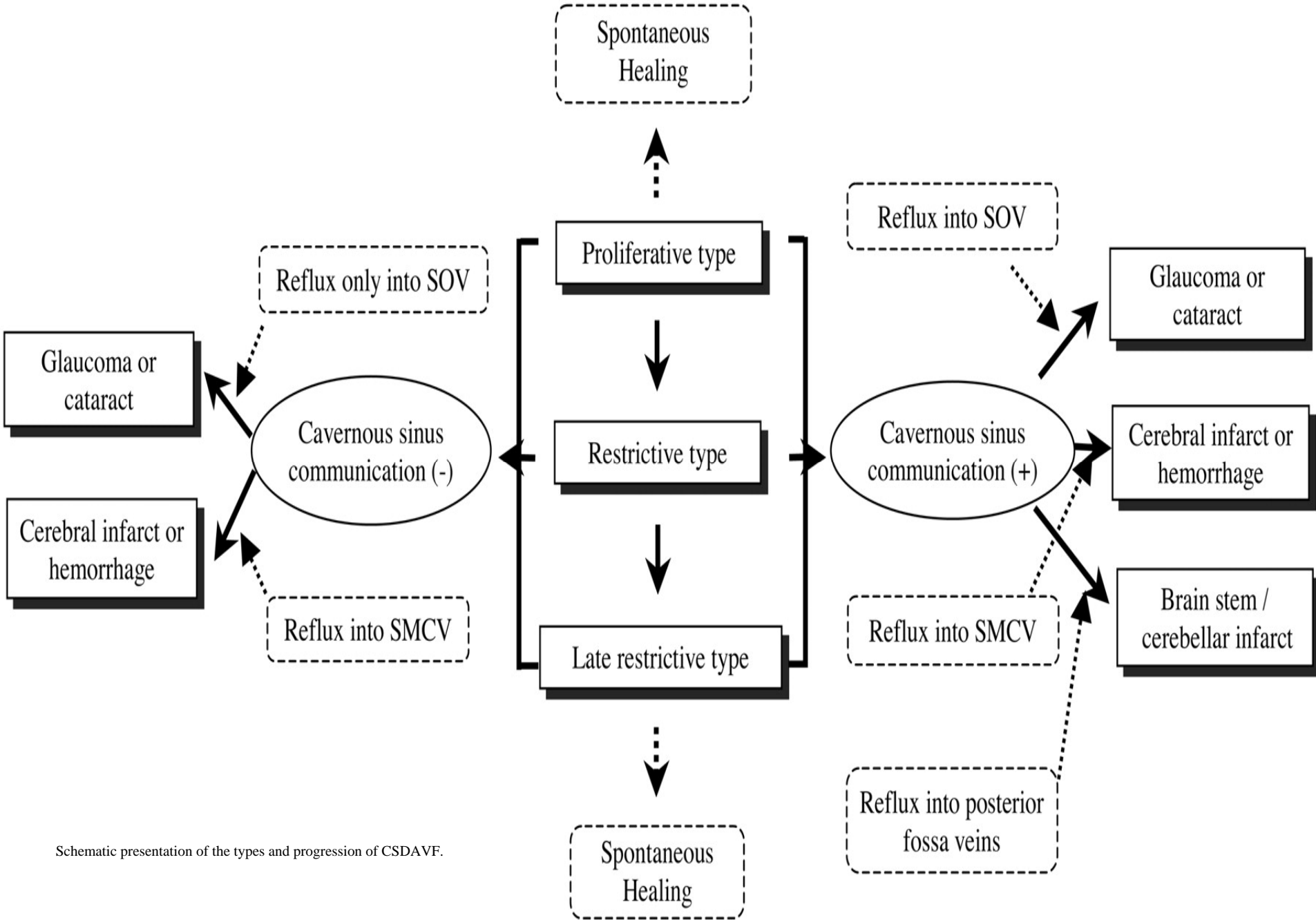
CT/MRI:

- Enlarged S. Ophthalmic v.
- Enlarged EOM
- Proptosis

Cerebral Angiography

- “Gold Standard” diagnosis
- View ICA, ECA, & vertebral circulations





Schematic presentation of the types and progression of CSDAVF.

Schematic presentation of the types and progression of CSDAVF

Treatment Option*	Results†
Conservative treatment	Regression of symptoms (20–50)
Radiation therapy	Complete occlusion (70–87)
Intervention	
TAE with particles	Complete occlusion (<50)
TVE	Complete occlusion (80–100)
TAE with <i>n</i> -butyl-2-cyanoacrylate	Complete occlusion (90–100)

Treatment Options for Cavernous Sinus Dural AVFs

(Listed in increasing order of potential risk and technical difficulty)

Management

➤ *Indications for Tx:*

- Lack of spontaneous closure
- Risk to eye/vision,
- Intolerable symptoms
- “High-risk” for stroke,
- Venous thrombosis,
- Mental status changes

Surgical closure

- Rare in last 30 years
- Can be salvage option in : In failed embolization, contraindication of embo, in occluded ICA with patent fistula d/t previous intervention.

Interventional Radiology :

(balloon occlusion/embolization)

- Primary treatment modality.
- Trans-arterial route directly through tear or embolization of feeding vessels.
- Trans-venous through sup. ophthal. v. or inferior petrosal sinus.

Endovascular Management

- Meyers, et al. Am J Ophthalmology, 2002

- Retrospective interventional case series

- 121 (90%) patients were cured clinically (mean f/u 56 mos)
- 4% patients with moderate/severe disability
- 6% with symptomatic complications
- 133/135 consecutive cases had tx
 - Cerebral infarction, Decreased VA (2), Diabetes Insipidus, orbital ecchymosis, retroperitoneal hematoma, DVT's (2)
- No operative mortality

Conclusions:

- High success rate
- Low complication/morbidity rate
- Patient's ocular symptoms may be transiently worsened post procedure

Superior Ophthalmic Vein Approach

- First proposed by Hanneken, et al. in 1989.
 - Direct access to cavernous sinus

Potential complications: puncture of S. ophthal v., orbital hemorrhage, infection, trochlea or other structure damage

Conclusions

- Especially effective with significant ICA contribution to CCF
- “technically straightforward, safe, and effective treatment”

➤ **RADIORURGERY :**

- May be effective in treating some indirect CCF.
(Type –B,C or D)

Dose : 20 Gy

➤ **LIMITATIONS** are –

- Takes a mean of 7.5 month to show effect on lesion.
- Inappropriate for pts. with progressive visual loss, neurological deficit, or cortical venous drainage.
- 15% recurrence rate (Pollock et al.)
- Risk of radiation induced malignancies.

➤ Thus precise role remains to be determined.

Treatment of Choice in CCF

Type – A : TA detachable balloon (Silicon balloons filled with HEMA)

Type – B : TVE

Type – C : TAE

Type – D : TAE, if fail then – TVE or Surgery

Prognosis

➤ Direct CCF:

- Poor visual prognosis (90% with severe vision loss)
- . -Ocular & optic nerve damage,
 - Exposure keratopathy, secondary glaucoma,
 - Ant segment ischemia,
 - CRVO
 - Ischemic ON
 - Concern for intra-cerebral hemorrhage

➤ Indirect CCF:

- Prognosis less severe
- Also concern for intra-cerebral hemorrhage
- Exacerbation & remission is the hallmark
- May close spontaneously (10-60%)

Transverse-Sigmoid Sinus Dural AVFs

- Most common location of IC dural AVFs.
- Most common symptoms are benign (pulsatile tinnitus and headache).
- More frequently associated with hemorrhagic and non hemorrhagic aggressive neurologic symptoms than CS dural AVFs.
- Spontaneous regression is relatively rare (approx.5%) and usually occurs following hemorrhagic events.

- Epicenter of fistula most often found at junction between the transverse and sigmoid sinus.

- Arterial feeders :
 - 1- Occipital artery branches
 - 2- MMA – posterior and petrosal branches
 - 3- ICA- marginal tentorial branch
 - 4- Post. auricular artery
 - 5- Vertebral artery – post. meningeal branch
 - 6- A. pharyngeal artery – meningeal branch

- Require treatment because of :
 - Low rate of spontaneous regression without symptomatic events.
 - Relatively high rate of aggressive symptoms.

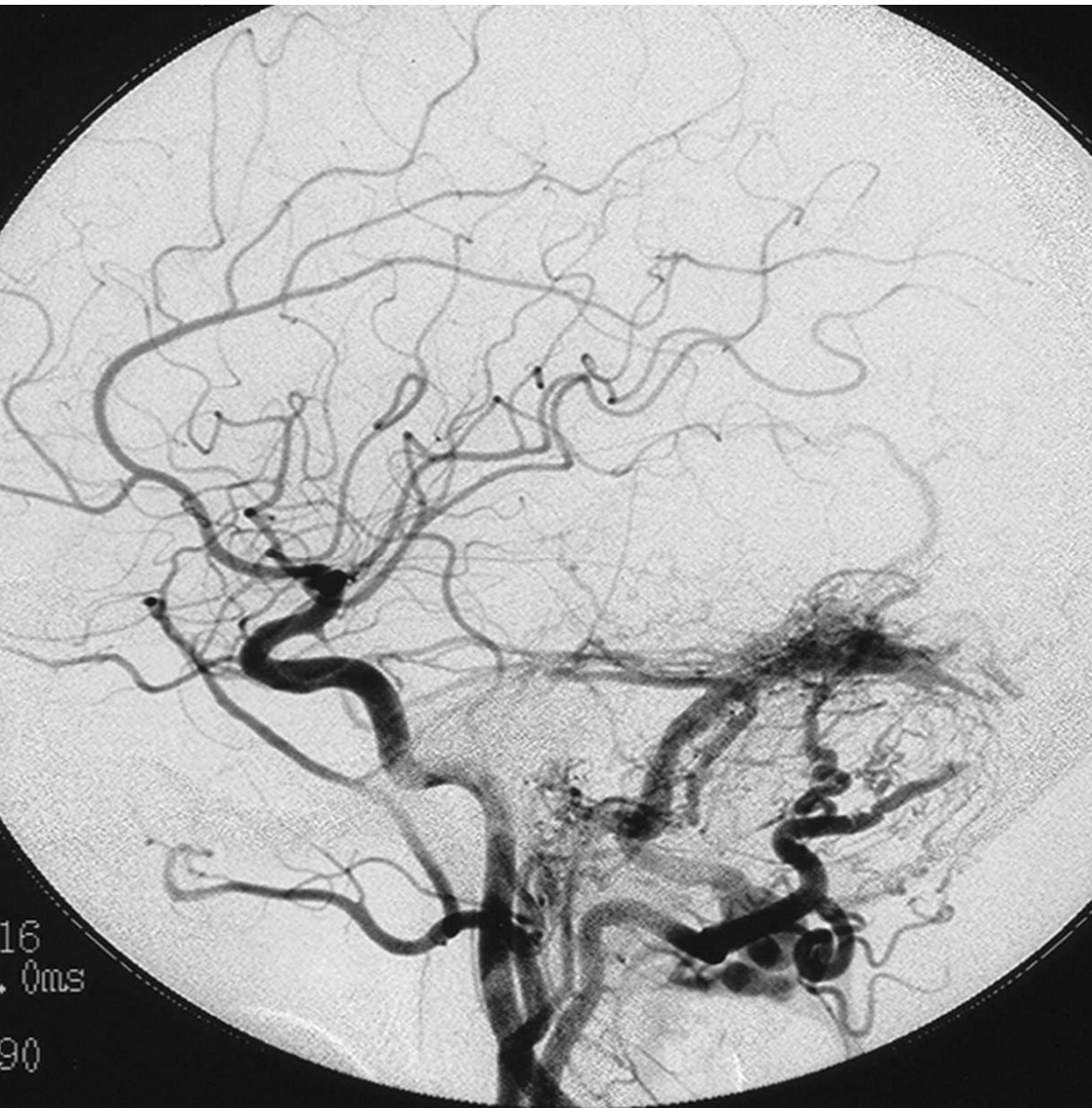
- Recent studies of stereo tactic radiation therapy for transverse-sigmoid sinus dural AVFs showed a relatively high occlusion rate of the AVF (approximately 60% of cases) several months after treatment without significant complications.

- Although TVE showed higher occlusion rates (80%–100% of cases), this procedure requires sacrifice of sinus flow and may cause venous infarction if the sinus contributes to the drainage of normal cerebral tissue.
- The rate of permanent complications in TVE is approximately 4%.

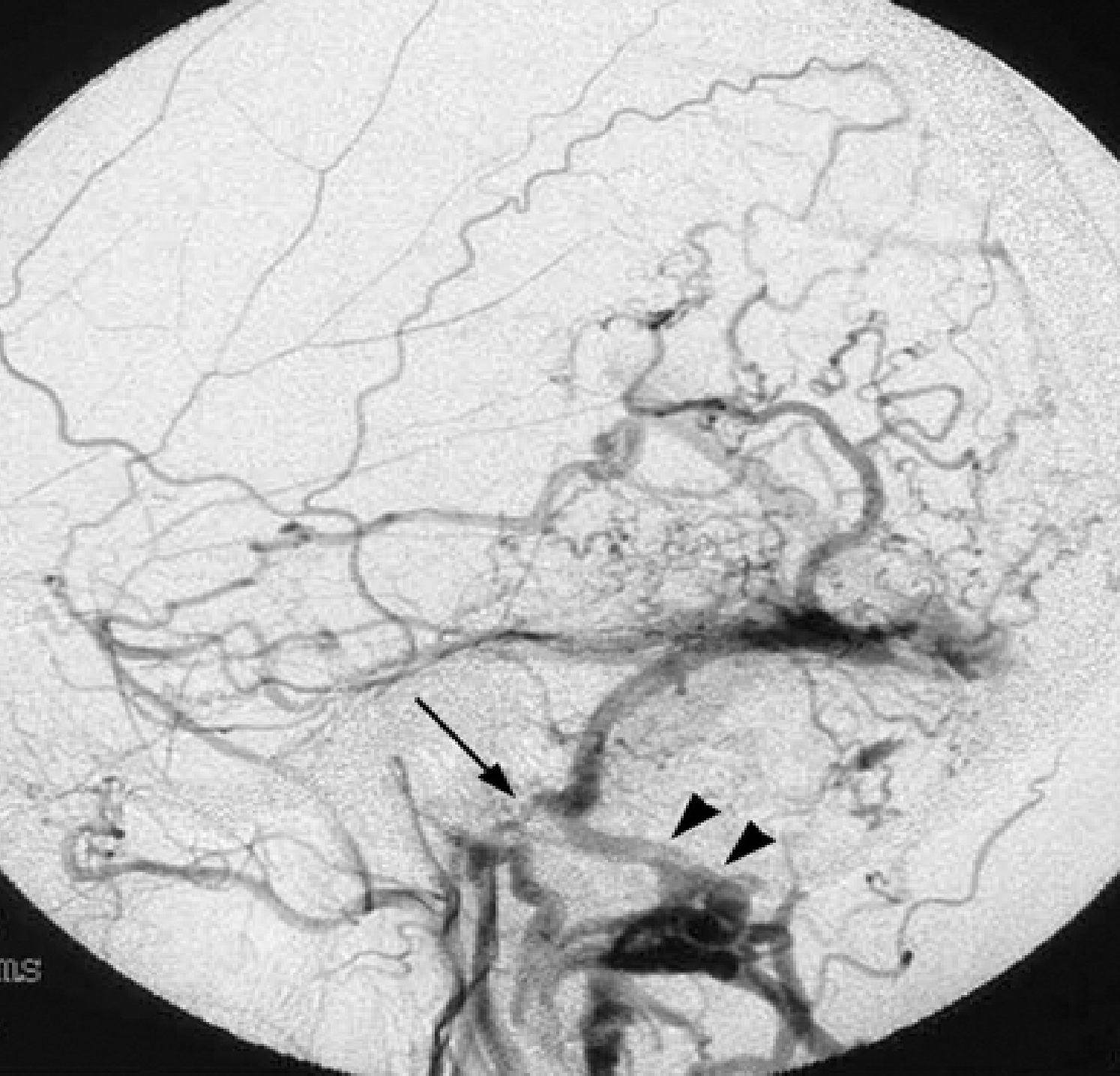
- In the treatment of Grade 2 lesions, occlusion of the normal cortical venous drainage system should be avoided.
- When there is a high risk of normal cortical venous drainage sacrifice at TVE, other treatments such as radiation therapy should be applied.
- Surgical isolation of the sinus with preservation of normal cortical venous drainage may also be performed but is more invasive.

- Grade 3 lesions can be treated with TVE, during which time the affected sinus and retrograde cortical drainage outlet should be tightly packed with coils.
- Loose packing might cause recanalization, resulting in delayed hemorrhagic infarction after embolization.

- Grade 4 lesions are the most difficult type of dural AVF to treat.
- The standard techniques combine endovascular and neurosurgical elements (eg, TVE combined with a surgical approach).
- In patients in poor general condition, other techniques (eg, TVE combined with other approaches, TAE with *n*-butyl-2-cyanoacrylate) may be used; however, they require more skill.



Recanalization
of a grade 3
transverse-
sigmoid sinus
dural AVF after
TVE. **(a)** Early
arterial phase
left common
carotid
angiogram
shows a Grade 3
transverse-
sigmoid sinus
dural AVF.
outlet (arrows).



Recanalization of a grade 3 transverse-sigmoid sinus dural AVF after TVE.

(b) Late arterial phase left common carotid angiogram shows that the left sigmoid sinus is occluded (arrow) and the dural AVF drains mainly into cortical veins and the posterior condylar vein (arrowheads).



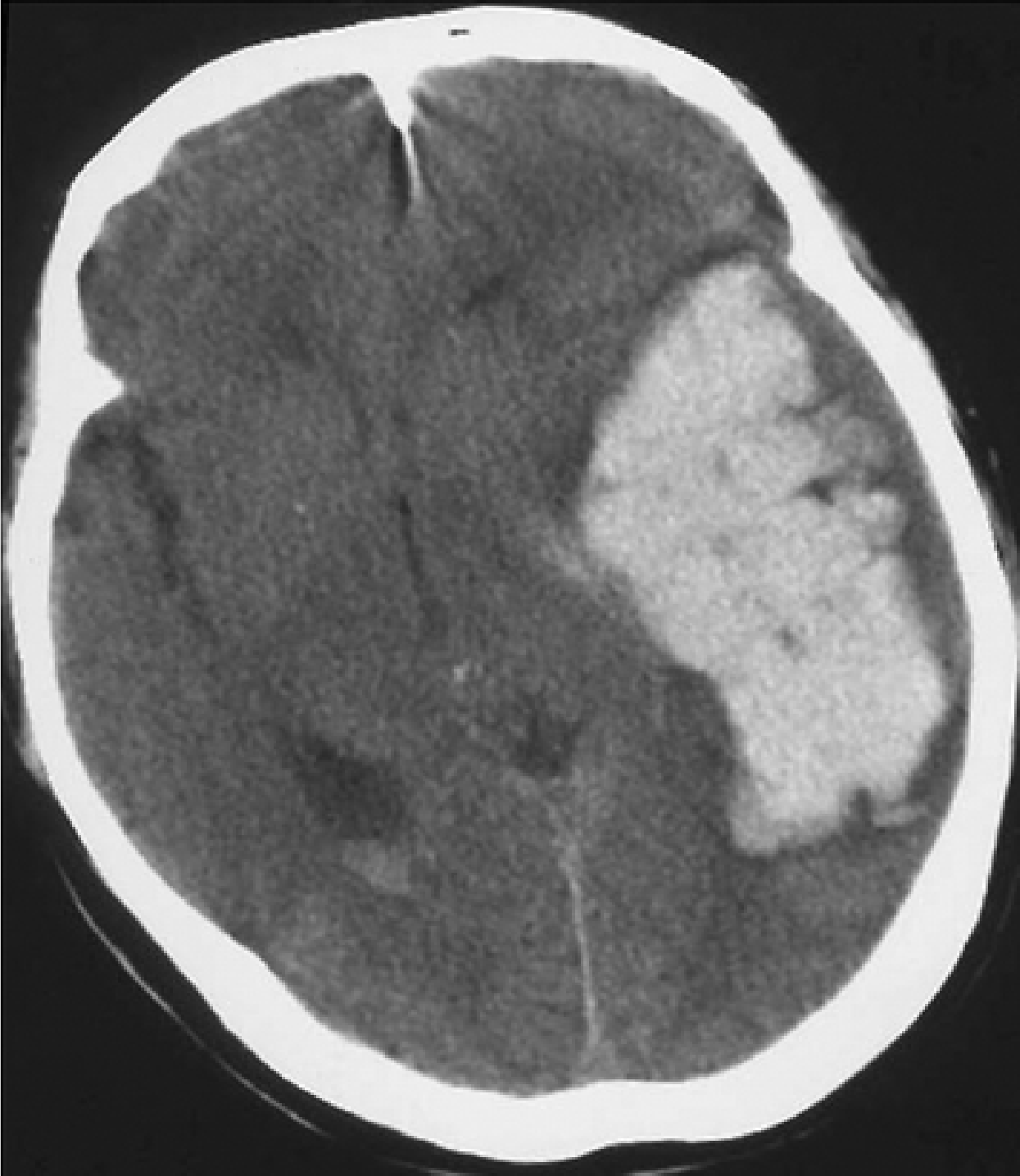
Recanalization
of a grade 3
transverse-
sigmoid sinus
dural AVF
after TVE.

(c) Superselective
venogram shows
a microcatheter
that has been
advanced via the
posterior condylar
vein (arrowheads)
into the affected
sinus.



Recanalization of a grade 3 transverse-sigmoid sinus dural AVF after TVE.

d) Left common carotid angiogram obtained after TVE shows disappearance of the AVF.



Recanalization
of a grade 3
transverse-
sigmoid sinus
dural AVF after
TVE.

(e) CT scan
obtained 2
months after
TVE shows a
massive
hemorrhage in
the left temporal
lobe.



Recanalization of a grade 3 transverse-sigmoid sinus dural AVF after TVE.

f) Left common carotid angiogram shows recanalization of the dural AVF at the retrograde cortical drainage outlet (arrows).

Treatment Options for Transverse-Sigmoid Sinus Dural AVFs

Treatment Option*	Results†
Radiation therapy	Complete occlusion (50–70)
Intervention	
TAE with particles	Complete occlusion (rare)
TVE	Complete occlusion (80–100)
TAE with <i>n</i> -butyl-2-cyanoacrylate	Complete occlusion (90–100 in cases without sinus drainage)
Stent placement	Reconstruction of antegrade sinus drainage
Surgery (sinus isolation or resection) combined with intervention	Complete occlusion (100)

Treatment options are listed in increasing order of potential risk and technical difficulty.

(Numbers in parentheses indicate percentages of cases)

Recommended Treatment Strategies for Transverse-Sigmoid Sinus Dural AVFs

Lesion Grade	Strategy
1	Radiation therapy combined with TAE with particles
2	(a) TVE with or without preparatory transarterial feeder embolization, (b) radiation therapy combined with TAE with particles, or (c) stent placement with transarterial feeder embolization and radiation therapy
3	(a) TVE with or without preparatory transarterial feeder embolization, or (b) stent placement
4	(a) TVE with either a surgical approach, an approach through the occluded sinus, or a transcortical venous approach; (b) TAE with <i>n</i> -butyl-2-cyanoacrylate; or (c) surgical resection or resection with preparatory TAE

Comparison of Various Treatment Options for Dural AVMs of the Lateral and Sigmoid Sinuses

Surgical resection	High cure rate	Technically difficult Risk of exsanguination Risk of venous infarction if cortical drainage is predominantly anterograde
Surgical "skeletonization" of the sinus	Preserves cortical venous outflow when cortical drainage is anterograde	Potential for recurrence due to collateral arterial recruitment
Transarterial embolization	Safe and effective when few exclusively dural arterial pedicles feed the malformation	Low cure rate when many arterial feeders are present Requires surgical exposure if proximal arterial pathways are occluded Risk of parenchymal embolization if arterial supply arises from internal carotid or vertebral artery branches
Transvenous embolization	Direct access to nidus of malformation Higher efficacy than transarterial route	Risk of parenchymal embolization through retrogradely draining bridging veins Percutaneous access may be limited by sinus thrombosis, requiring surgical exposure Inadequate embolization may exacerbate venous hypertension

Tentorial Dural AVFs

- Incidence of intracranial hemorrhage – 60% to 74%.
- May cause fatal bleeding in the posterior fossa.
- Tentorial dural AVFs drain through the retrograde leptomeningeal-cortical venous drainage system only (Cognard types III and IV, Borden type III), resulting in a high risk of hemorrhagic or nonhemorrhagic aggressive symptoms (19% and 10% of cases per year, respectively).

- Complete cure requires aggressive treatment.
- Interventional and surgical procedures are both used to disconnect the venous drainage system.
- Because of the deep-seated location of such lesions, the difficult access route, and the need for *n*-butyl- 2-cyanoacrylate, these techniques require a high level of skill.

- Treatment selection depends on :
 - Skill of the neurosurgeon and interventional radiologist
 - On lesion accessibility

- Stereotactic radiosurgery should be considered an option, especially in older patients or in those in poor general condition.

Treatment Options for Tentorial Dural AVFs

Treatment Option*	Results†
Radiation therapy	Complete occlusion (50–60)
Intervention	
TAE with <i>n</i> -butyl-2-cyanoacrylate	Complete occlusion (50–100)
TVE	Complete occlusion (90–100 in a few case reports)
Surgery (disconnection of leptomeningeal venous drainage)	Complete occlusion (100)



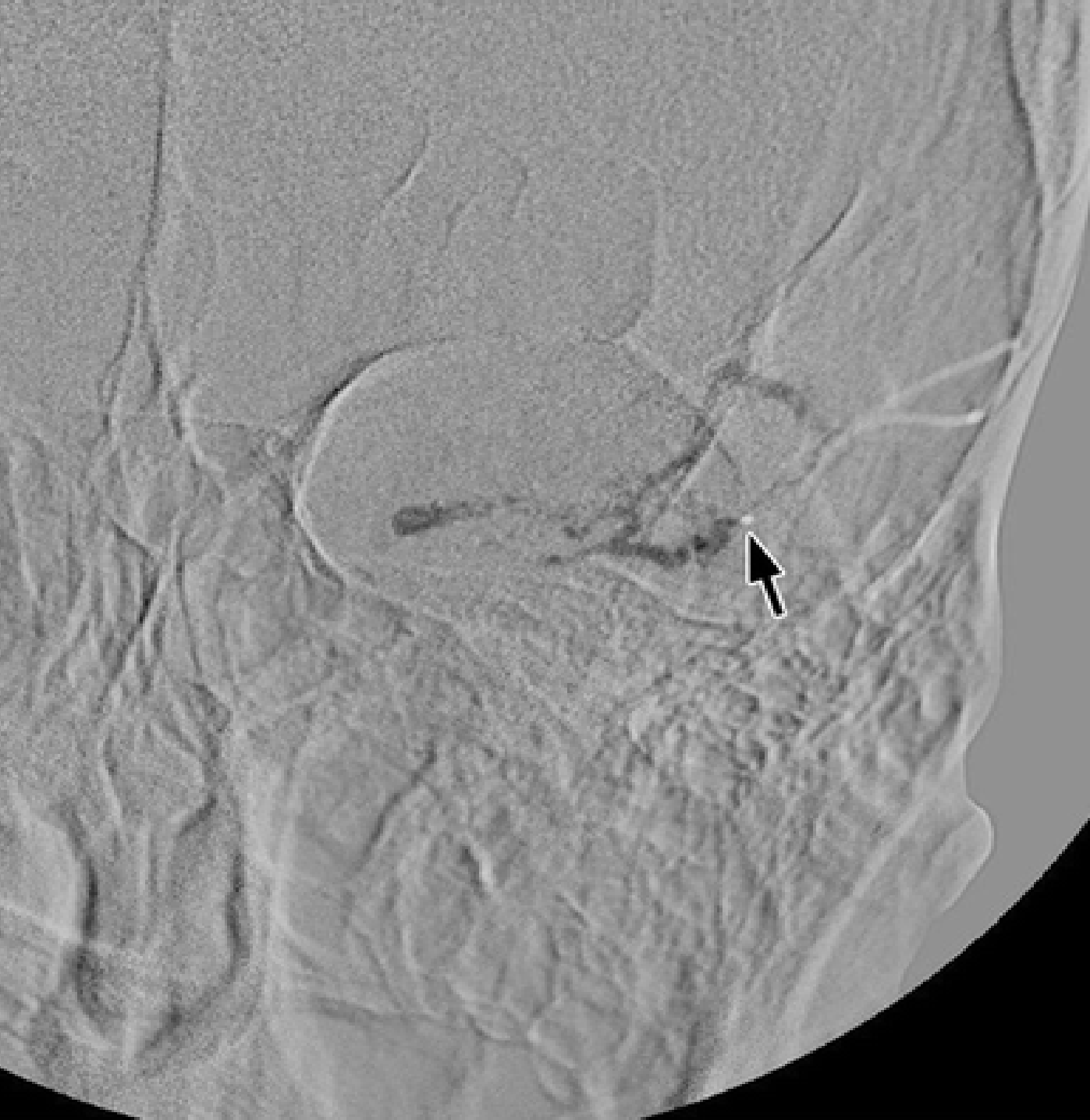
Type IV
tentorial dural
AVF with
intracranial
hemorrhage.

(a)
Unenhanced
CT scan
shows
intracranial
hemorrhage in
the left
occipital lobe
and the lateral
ventricle.



Type IV
tentorial dural
AVF with
intracranial
hemorrhage.

(b) Left external
carotid
angiogram shows
a tentorial dural
AVF
(arrowheads)
with
leptomeningeal-
cortical venous
drainage and
venous ectasia
(arrow).



Type IV tentorial
dural AVF with
intracranial
hemorrhage

(c) Digital
subtraction
angiogram
obtained during
the injection of
diluted *n*-butyl-2
cyanoacrylate
demonstrates the
tip of a
microcatheter
(arrow).



Type IV
tentorial
dural AVF
with
intracranial
hemorrhage

(d) Left
common
carotid
angiogram
obtained after
TAE shows
complete
obliteration of
the AVF.

SDAVFs

Arterial Supply of the Spinal Cord

- By the vertebral arteries.
- Also from the branches ultimately from the thoracic and abdominal aorta (Radicular arteries).
- Each vertebral artery (or PICA) gives rise to a posterior spinal artery, which proceeds along the line of attachment of the dorsal roots.
- Each vertebral artery also gives rise to an anterior spinal artery.

- The two ant. spinal arteries fuse to form a single midline vessel along the anterior median fissure.
- The posterior spinal arteries and the anterior spinal artery supply upper cervical levels with from the vertebral arteries.
- Below this, all 3 spinal arteries form a more or less continuous series of anastomoses with radicular arteries.

- Great radicular artery (of Adamkiewicz), present at the spinal cord level T12-L2 may provide the entire arterial supply for the caudal 2/3 of the spinal cord.
- Very long anterior spinal artery is usually a continuous vessel for the length of the spinal cord.
- It gives rise to hundreds of central and circumferential branches.
- These supply the anterior 2/3 of the spinal cord.
(*posterior horn and a variable portion of the lateral corticospinal tract*)

- The posterior spinal arteries are really more of a plexiform network of small arteries.
- They supply the posterior columns, substantia gelatinosa, dorsal root entry zone, and a variable portion of the lateral corticospinal tract.

Venous Drainage of the Spinal Cord

- By 6 irregular, plexiform channels.
- There is one along:
 - The anterior and posterior midlines;
 - Along the line of attachment of the dorsal roots of each side;
 - Along the line of attachment of the ventral roots of each side.
- These are drained by the radicular veins.
- Each, in turn empty into the epidural venous plexus.

Spinal Dural AVFs

INTRODUCTION :

- Rare and enigmatic disease entity
- Also known as
 - Long dorsal AVM
 - Angioma racemosum venosum (Wyburn-Mason)
 - Malformation retromedullaire (Djindjian)
 - Dorsal extra medullary AVMs
- Clinical features and structural changes **have** been recognized since 1926 (Foix and Alajouanine), and the pathophysiology and the essentials of treatment since 1974.
- Etiology unknown

- Probably the first successful operation on an SDAVF was performed by Elsberg in 1916.
- He ligated and excised an enlarged and thickened vein at the level of T8 in a patient with a sensory level at T9.
- The patient made a full recovery
- Kendall and Logue (1977) showed that the site of the fistula was not located in the spinal cord but on or in the **dural root sleeve**.

- Caudal end of the spinal -first affected by congestive edema and ultimately infarction, regardless of the level of the fistula.
- Thus initial clinical features often consist of sensory and motor symptoms ascending from the feet, suggesting a polyneuropathy or polyradiculopathy

Epidemiology

- SDAVFs are rare, but still make up the most common vascular anomaly of the spine, with a proportion of 60–80%.
- 5–10/million/year in the general population
- Underdiagnosed disease.
- Mostly in middle aged men.

- Mean age at the time of diagnosis is 55–60 years.
- Patients under the age of 30 are rarely reported
- M:F = 5:1
- M. C. location - Thoracolumbar region
- Cervical and sacral SDAVF constitute just under 6% of patients with SDAVF.
- Multiple SDAVFs are uncommon (0.5-4%).

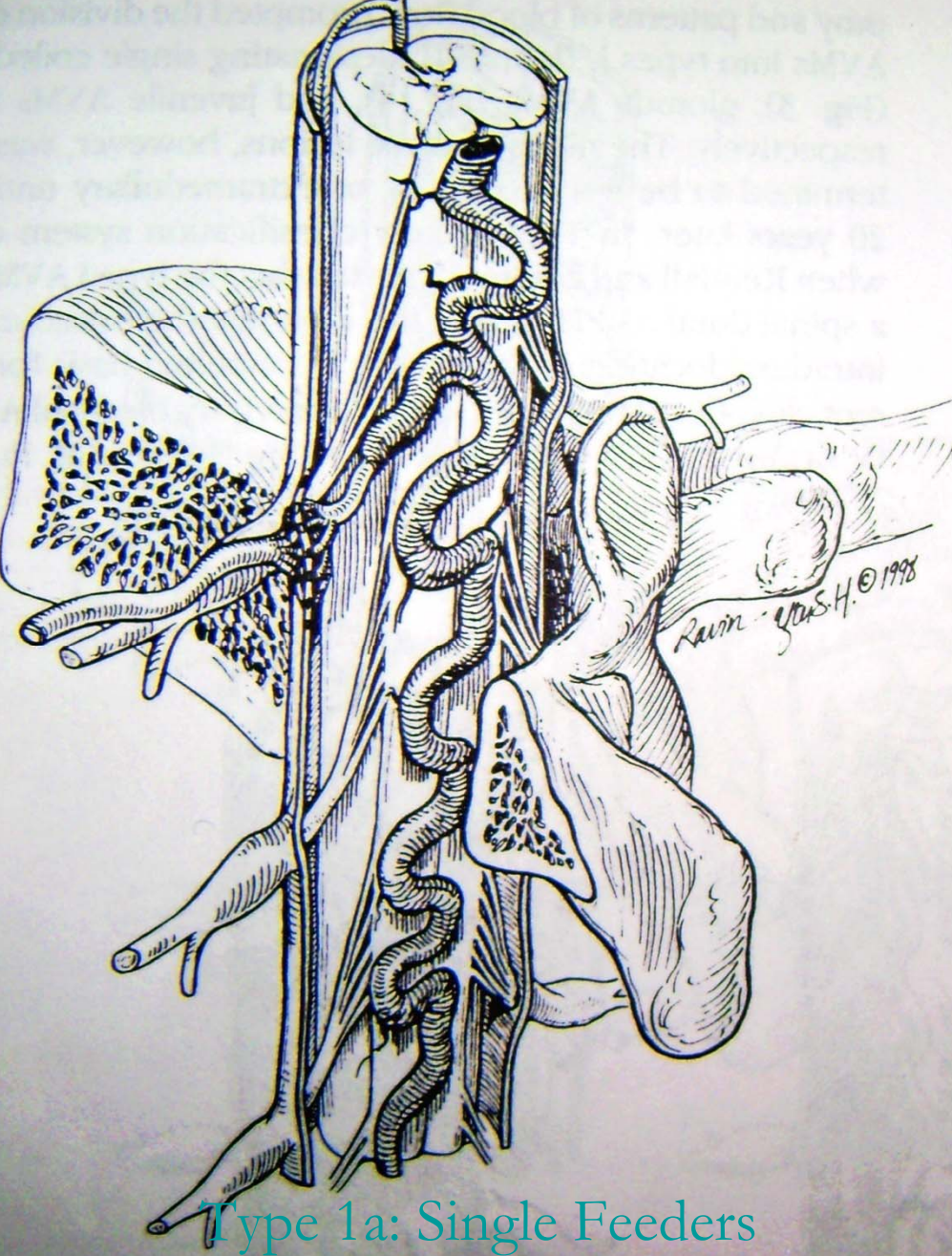
Classification of spinal vascular lesions (Spetzler et al,2002)

- Neoplastic vascular lesions
 - Haemangioblastoma
 - Cavernous malformations
- Aneurysms
- Arteriovenous lesions
 - Arteriovenous malformations
 - Arteriovenous fistulas
 - : Extradural
 - : Intradural
 - Ventral - A: Small, B: Medium, C: Large
 - Dorsal - A: Single arterial feeder
 - B: Multiple arterial feeder

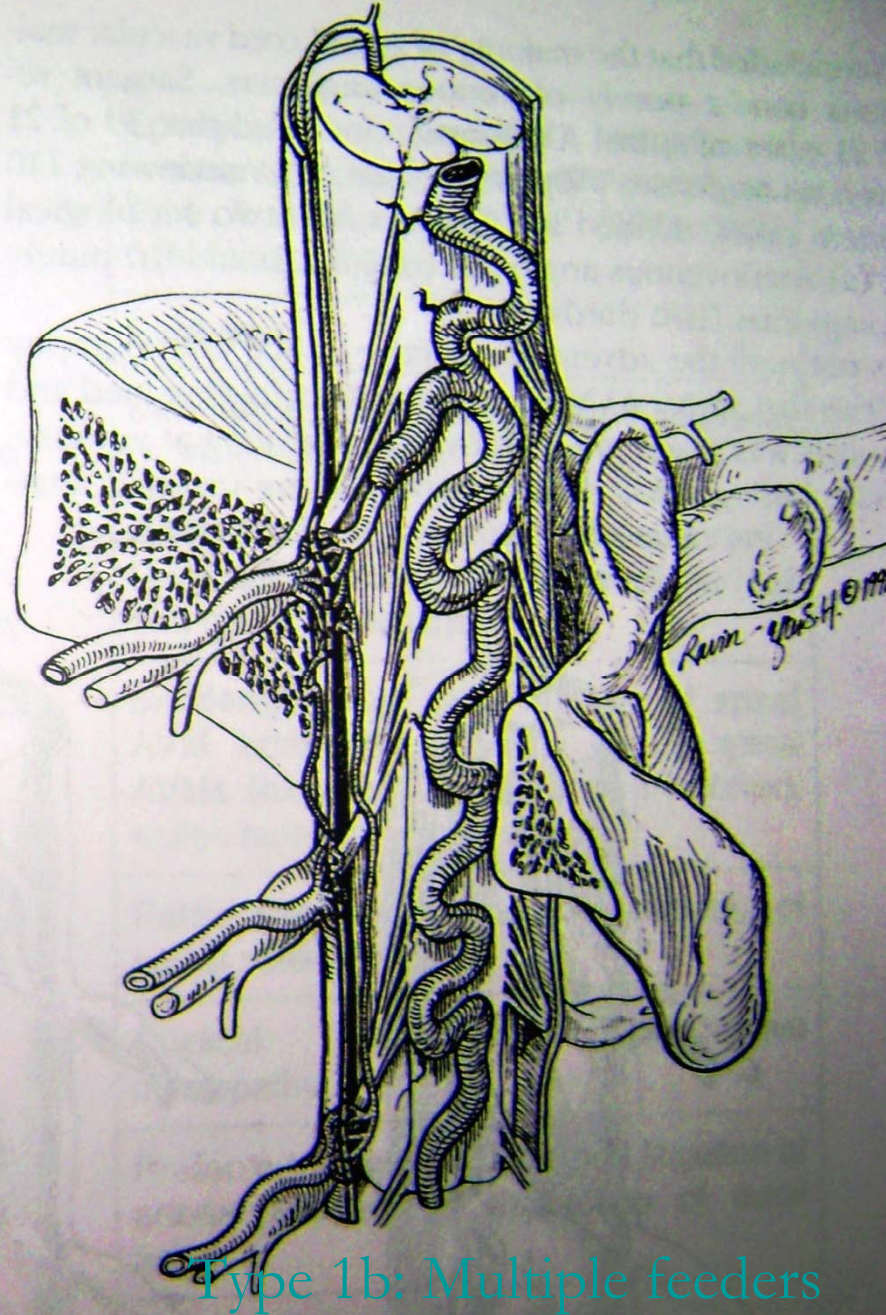
TABLE 141-2 ■ Classification of Spinal Arteriovenous Malformations

TYPE	DESCRIPTION
I	Arteriovenous fistula located between a dural branch of the spinal ramus of a radicular artery and an intradural medullary vein
II	Intramedullary glomus malformation with a compact nidus within the substance of the spinal cord
III	Extensive arteriovenous malformation often extending into the vertebral body and paraspinal tissues
IV	Intradural perimedullary arteriovenous fistula
IV = A	Simple perimedullary fistula fed by a single arterial branch
IV = B	Intermediate-sized fistula with multiple dilated arterial feeders
IV = C	Large perimedullary fistula with multiple giant arterial feeders

From Anson JA, Spetzler RF: Classification of spinal arteriovenous malformations and implications for treatment. BNI Q 8:2-8, 1992.



Type 1a: Single Feeders



Type 1b: Multiple feeders

Spinal Dural AVF, Extradural type

Causal factors and pathophysiology

- An acquired condition – Onset in middle age.
- Venous hypertension - the main pathophysiological factor.
- The shunt is most often formed within the dorsal surface of the dural root sleeve in the intervertebral foramen, where the radicular vein pierces the dura, together with one or more dural branches of the radicular artery.

- Shunt is sometimes situated along the dura between two adjacent nerve roots.
- Increased pressure causes the venous system to ‘arterialize’.
- The radicular feeding artery is often a dural branch and in a minority, the medullary artery.
- The shunt (low flow) results in venous hypertension in the spinal cord, because the intramedullary veins and the radicular vein share a common venous outflow.

➤ Reduced arteriovenous pressure gradient:



- Decrease in tissue perfusion and venous infarction.

➤ Apart from the increased pressure caused by the shunt, the venous outflow may be less efficient to start with than is the case in healthy individuals.

➤ Lower thoracic region has relatively fewer venous outflow channels at a segmental level than the cervical or lumbosacral region.

- These differences in segmental outflow probably contribute to the phenomenon that venous congestion is transmitted in a caudo-cranial direction throughout the spinal cord.
- Thus, first symptoms of myelopathy tend to reflect dysfunction of the lowest part of the cord, (conus medullaris), even though the shunt is at the higher level, or in some cases even near the skull base.
- Venous outflow through the medullary vein and venous plexus is dorsal from the cord in 80–90%, and combined ventral and dorsal in 10–20%.

Table 3 Proportion of patients with symptoms present at the onset of the disease and symptoms present at the time of diagnosis

	Initial symptoms (%)	Symptoms at diagnosis (%)
Gait difficulties and motor disturbances	50–81	78–100
Pain (either pain in the back or radicular pain)	13–64	17–86
Micturition difficulties	4–75	62–91
Defaecation problems	0–38	30–100
Sexual dysfunction	0–17	11–80

Clinical diagnosis

- SDAVF is notoriously hard to diagnose, because of the misleading nature of the initial symptoms and the rarity of the disease.
- The median time between onset and diagnosis ranges between 12 and 44 months.

- Erroneous diagnoses often made initially are -
 - Sensory polyneuropathy,
 - Acute or chronic inflammatory demyelinating polyneuropathy
 - Spinal muscular atrophy
 - Medullary tumour

- Not infrequently patients are unsuccessfully operated for a lumbar disc prolapse.

- At the time of diagnosis, two-thirds of patients show a combination of gait difficulties, sensory disturbances and involvement of sacral segments (micturition, defaecation or sexual dysfunction).
- Bowel and micturition problems frequently occur, mostly later in the course of the disease .
- Micturition disturbances consist of urinary retention.
- Erectile dysfunction exists in 11–80% of men patients, and unwanted and involuntary ejaculations may occur after exercise.

- In the majority of patients (40–63%) progression lasts for 1–3 years before the diagnosis is made, but a protracted course with a duration of >3 years occurs in 10–34%.
- A gradually progressive course with stepwise deterioration is recorded in 11–32% of patients.
- A spontaneous and complete disappearance of the fistula has occasionally been described, though the clinical deficits remained unchanged in these patients.

- If symptoms develop within minutes to hours they can mimic an anterior spinal artery syndrome.
- The sudden episodes mostly occur after exercise, prolonged standing and even singing and may disappear after rest.
- Acute worsening of symptoms may also be related to changes in posture such as bending over; even eating has been related with worsening of symptoms

TABLE 142-2 ■ Comparison of Clinical Syndromes of Dural Arteriovenous Fistulas and Intradural Arteriovenous Malformations

	DURAL AVF (N = 27)	INTRADURAL AVM* (N = 54)
Gender	Predominantly male	Male or female
Mean age (yr) at diagnosis	46	24
Onset of symptoms	Gradual (85%)	Acute (37%)
SAH	0	50%
First symptom	Paresis (44%)	SAH (32%)
Spinal bruit	0	6%
Exacerbation of symptoms by activity	70%	15%
Arms affected	0	11%

INVESTIGATION

- The essential investigations to establish the diagnosis are MRI and catheter angiography.
- **MRI findings** - Hypointens -T₁WI
 - Hyperintens-T₂-WI
 - Increased signal intensity in the centre of the spinal cord and peripheral sparing on T₂-WI is found in 67–100% of patients.
- Hyper intensities extend over an average level of 5–7 vertebrae, with conus involvement over 80%, and are typically homogeneous.



MRI and angiography of SDAVF. (A) T₂-weighted MRI image of a 66-year-old man with SDAVF. Multiple flow voids resembling an enlarged medullary draining vein can be seen. (B) Angiogram of a 57-year-old man showing an SDAVF at T7 on the right.



Increased signal intensity in the centre of the **spinal** cord with peripheral sparing on a T₂-weighted MRI image of a 60-year-old male with a left-sided SDAVF at T8

- Abnormal blood vessels may be seen on either the ventral or the dorsal side of the spinal cord (flow void phenomena in 35-91% patients).
- Marked enhancement occurs some 40–45 min after contrast injection.
- **MR Angiography:**
 - reveals flow in serpentine perimedullary structures in up to 100% of patients.

Catheter angiography:

- Gold standard in the diagnosis of SDAVF
- Not only the intercostal and lumbar arteries should be visualized as potential feeding arteries of an abnormal shunt but also the median and lateral sacral artery, the deep cervical and ascending cervical arteries.

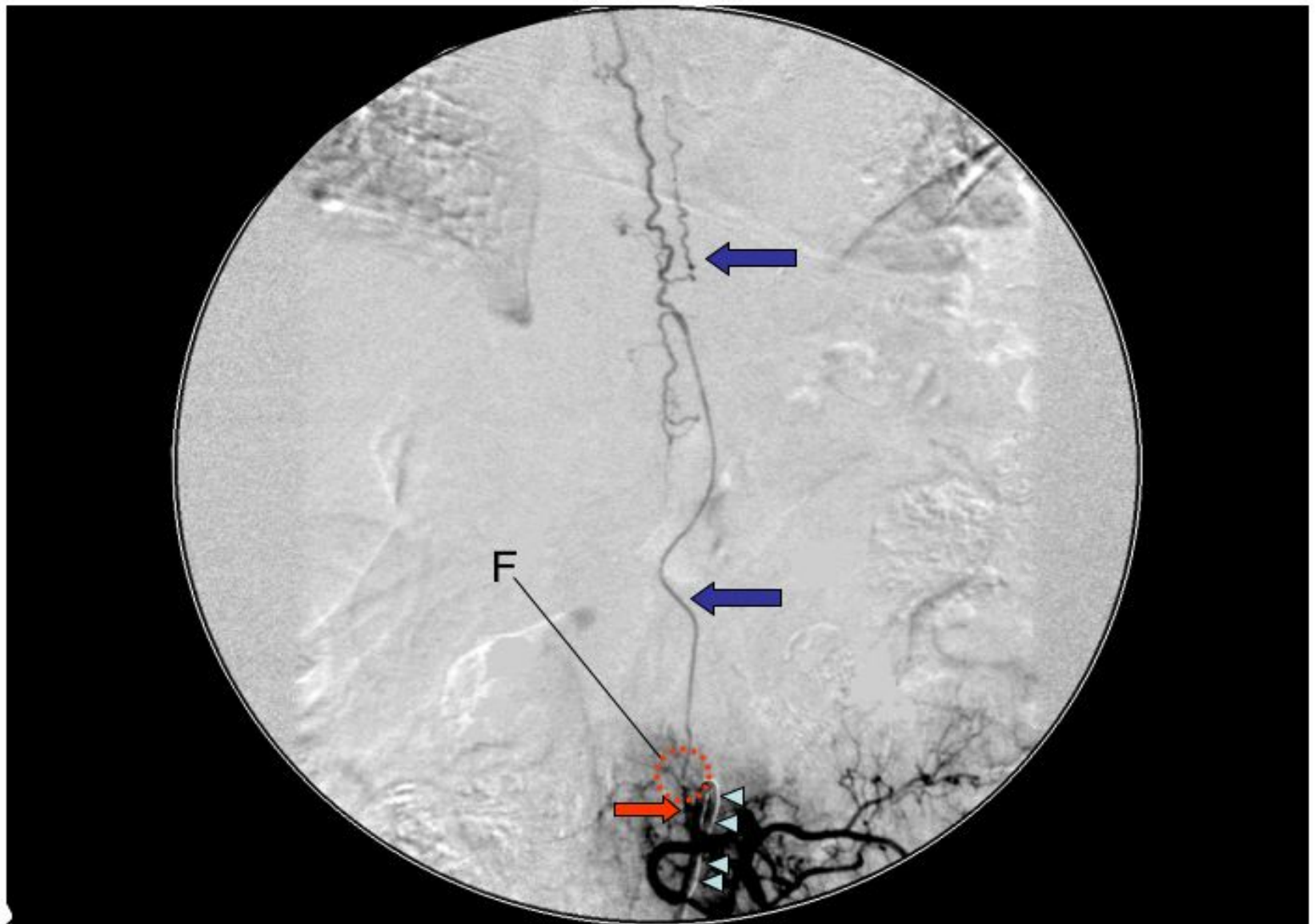


Figure 3 (above) shows a spinal DAVF during selective spinal angiography. The catheter through which the contrast is injected is marked by the light-blue arrow heads. The fistula itself (F; red circle) is in the spinal nerve root dural sleeve. The arterial supply is marked by the red arrow and the draining vein is marked by the dark-blue arrows.

Methods of treatment

- The choice of treatment is between endovascular embolization and surgical ligation of the fistula.

Embolization :

- Liquid polymers [such as isobutyl 2-cyanoacrylate (IBCA), *n*-butyl 2-cyanoacrylate (NBCA)] is preferred over particles such as polyvinyl alcohol (PVA), because the use of particles leads to a recurrence rate as high as 30–93% (Nichols *et al.*,1992). In contrast, occlusion is successful with liquid polymers in 44–100%.

➤ Embolization of SDAVF is not possible in -.

- 1- If the arterial feeder of the fistula is a segmental medullary artery.
(high risk of spinal cord ischemia)
- 2- In technical difficulties such as arterial wall dissection of the feeding vessels during the embolization procedure may prohibit introduction of the micro catheter close enough to the fistula.

- If embolization is possible, then the success of treatment depends on endovascular occlusion of the draining vein, which means that recanalization will rarely occur when the draining vein is filled with glue (Jellema *et al.*, 2005).
- The reason is probably that the fistula is often made up of several small feeding arteries and a single draining vein, so that occlusion of only (one of) the arterial feeder(s) to the fistula will generally lead to development of new arterial feeders.

Surgical treatment

- Multi-level laminectomy with stripping of the draining vein and decompression of the spinal cord (when pathophysiology of SDAVF was not clear)
- Presently an intradural interruption of the vein draining the fistula is advocated (Huffmann *et al.*, 1995).
- As effective as total removal of the draining vein.

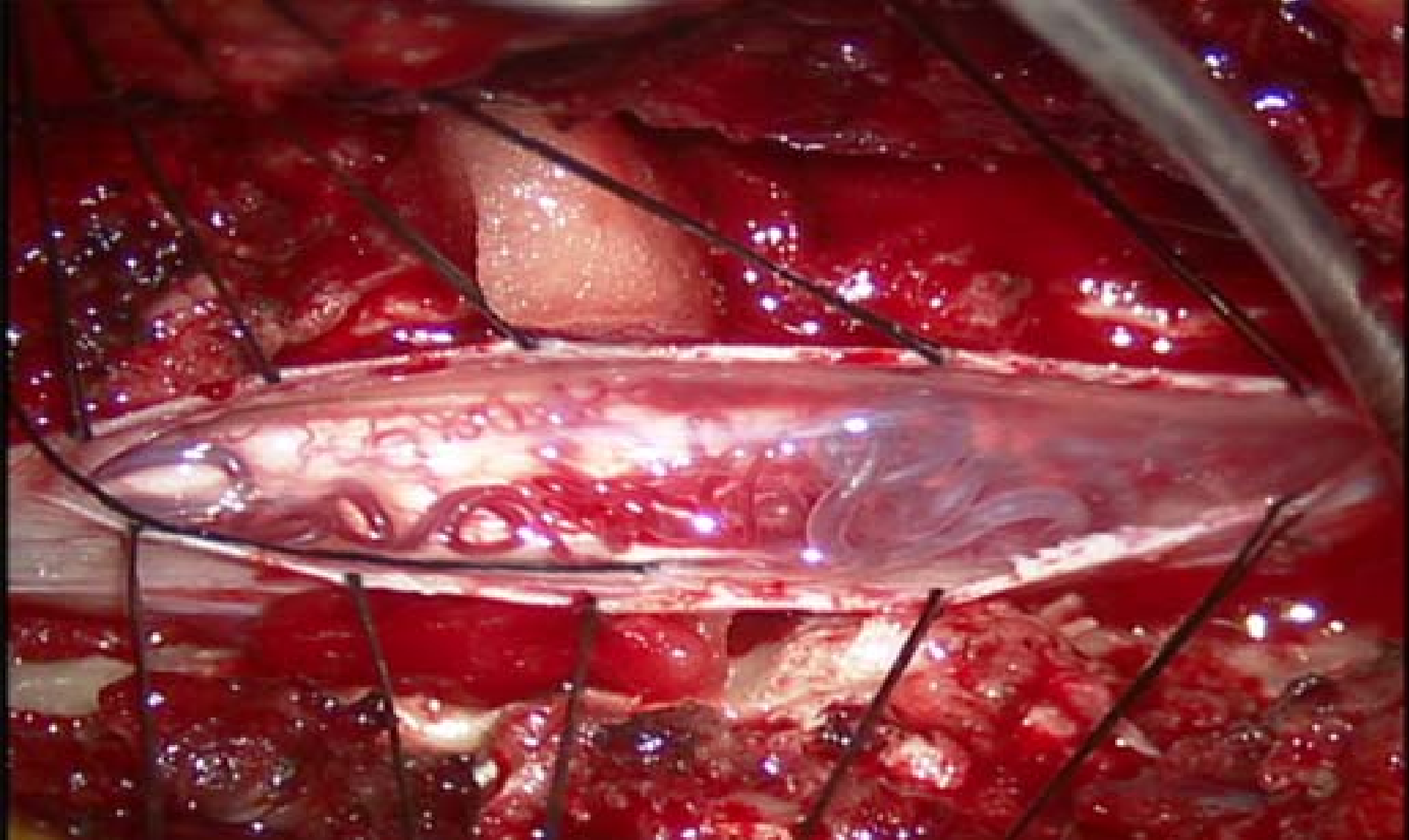


Figure shows the draining vein complex of a spinal DAVF immediately following open surgical disconnection of the fistula. The draining vein complex, a tangle of red-purple veins, can be seen. Normally, there should be no such tangle of veins on the spinal cord surface.

- A meta-analysis of patients with SDAVF who were treated with either embolization or operation showed that almost 98% of surgical procedures were technically successful; this stands in contrast to the 46% of patients who were successfully treated with embolization (Steinmetz *et al.*, 2004).

Outcome after treatment

- Depends on several factors:
 - Duration of symptoms
 - Pre-treatment disability
 - Success of the procedure to close the fistula
- Treatment is directed at halting the progression of symptoms or even reversing them.
- Symptoms that generally respond well to treatment are :
 - Gait difficulties
 - Muscle strength(resulting in less disability and dependence)

➤ Symptoms that often respond less well to treatment are :

- Micturition,
- Pain
- Muscle spasms

Aminoff–Logue disability scales for gait and micturition (Aminoff and Logue, 1974*b*)

Gait

- 0 Normal
- 1 Leg weakness, abnormal gait or stance, but no restriction of activity
- 2 Restricted activity but not requiring support
- 3 Requiring one stick for walking
- 4 Requiring two sticks, crutches or walker
- 5 Confined to wheelchair

Micturition

- 0 Normal
 - 1 Hesitancy, urgency, frequency, altered sensation, but continent
 - 2 Occasional urinary incontinence or retention
 - 3 Total incontinence or persistent retention
-

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. Images of a brain dural arteriovenous fi

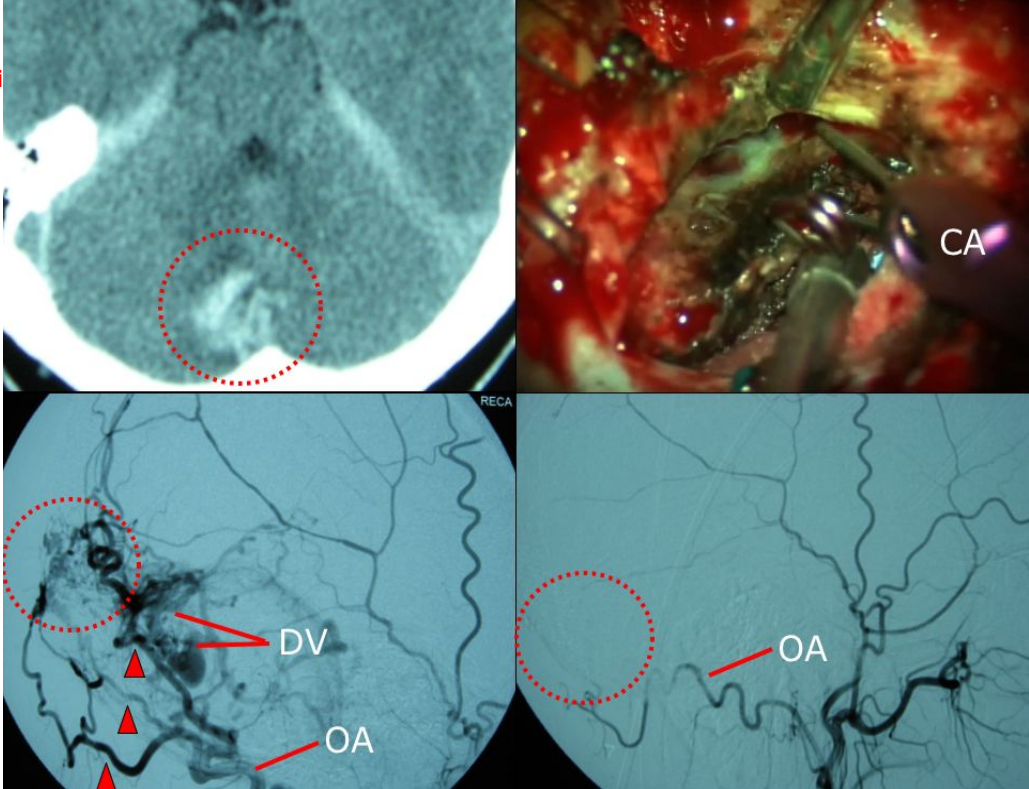


Figure 2 (above collage) shows a cranial (brain) DAVF. Top left: Axial CT scan shows a **ruptured DAVF** (circled in red) located in the right paramedial cerebellum and located in the dura surrounding a very high-risk venous structure known as the *torcula herophili*. The patient presented with **abrupt-onset headache** and **impaired consciousness**. Bottom left: Preoperative cerebral angiogram (right external carotid artery injection) showing the actual **fistula** (circled in red) with its **blood supply** arising mainly from the **occipital artery** (OA) via many **arterial channels** (red arrow heads). The fistula is drained by an abnormal sac-like **draining vein** (DV). Top right: Intraoperative photograph showing the fistula being **surgically disconnected** by the placement of **titanium microclips** via a neurovascular clip applicator (CA) across the draining vein. Bottom right: Postoperative cerebral angiogram (right external carotid artery injection) following surgical disconnection of the fistula. The angiogram shows complete obliteration of the fistula (nothing left to see in the red circled area!). The patient was discharged from hospital neurologically intact

External layer- derived from the internal layer of periosteum and adherent to the inner surface of the skull.

Continuous with the ectocranial periosteum through the foramina of the skull.

Internal layer - True meningeal duramater (cranial duramater) and adheres in most places to the external layer.

Separations of the two layers create the dural sinuses, the dural reflections, and the trigeminal cave.

Internal layer continues through the foramen magnum to enclose the spinal cord ending at S2 level.

Internal layer forms the five dural reflections :

- 1 - Falx cerebri
- 2 - Falx cerebelli
- 3 - Tentorium cerebelli
- 4 - Diaphragma sellae
- 5 - Tentorium bulbus

olfactorium

Cranial duramater contains :

- 8 paired venous sinuses
- 7 unpaired venous sinuses

A - Valt – Ophthalmic branch of trigeminal nerve

B - Ant. cranial fossa :

- Ant. Ethmoidal nerve
- Maxillary nerve

C – Middle cranial fossa :

Ant. half - Maxillary

nerve

- Mandibular

nerve

Post. half- Trigeminal

ganglion

D – Posterior cranial fossa :

- C1, C2, C3
- 9th and 10th cranial

nerves

ANATOMY OF CS

Plexus of veins encased in a double layer of dura.

Located on either side of the body of sphenoid sinus.

Length = 2 cm, Width = 1 cm.

Extends from SOF to apex of petrous temporal bone

ICA given of 3 branches in the CS :

- 1 – Meningohypophyseal trunk
- 2 – A. of inf. CS
- 3 – McConnell,s capsular arteries (30-50%)

Venous drainage of CS include:

- Superior ophthalmic vein (SOV)
- Superior petrosal sinus (IPS)
- Inferior petrosal sinus
- Superficial middle cerebral vein (SMCV)
- Coronary sinus to the opposite side of CS.

CS and IPS drain veins of the brain after birth.

All the cerebral veins converge toward the posterior

sinuses, depending on the rapidity with which CS matures

and venous communication between CS and cerebral veins takes place.

Djindjian Classification (1977)

Type -1: Immediate drainage into a sinus with antegrade flow.

Type -2 : Initial drainage into a sinus with reflux into other sinuses or cortical veins.

Type -3 : Initial drainage into cortical vein.

Type -4 : Initial drainage into cortical vein with a venous pouch.

Borden Classification

- Three types: based upon their venous drainage:

Type I: Dural arterial supply drains anterograde into venous sinus.

Type II: Dural arterial supply drains into venous sinus.

High pressure in sinus results in both anterograde drainage and retrograde drainage via subarachnoid veins.

Type III: Dural arterial supply drains retrograde into subarachnoid veins.

Type I :

- Supplied by meningeal arteries and drain into a meningeal vein or dural venous sinus. The flow within the draining vein or venous sinus is anterograde.

Type Ia -Simple dural arteriovenous fistula have a single meningeal arterial supply.

Type Ib -More complex arteriovenous fistulas are supplied by multiple meningeal arteries.

Type II

The high pressure causes blood to flow in a retrograde fashion into subarachnoid veins which normally drain into the sinus.

Typically this is because the sinus has outflow obstruction.

Such draining veins form venous varices or aneurysms which can bleed.

Type II fistulas need to be treated to prevent hemorrhage.

Type III

Drain directly into subarachnoid veins.

These veins can form aneurysms and bleed.

Type III dural fistulas need to be treated to prevent hemorrhage.

Classification System

Type	Cognard et al (3)	Borden et al (8)
I	Antegrade sinus drainage	Sinus or meningeal venous drainage
II	Insufficient antegrade sinus drainage	Sinus drainage with CVR
IIa	Retrograde sinus drainage only	...
IIb	Retrograde CVR only	...
IIa + IIb	Retrograde sinus drainage and CVR	...
III	CVR only without venous ectasia	CVR only
IV	CVR only with venous ectasia	...
V	Spinal venous drainage	...