Falcotentorial Dural Arteriovenous Shunts

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Acknowledgements

- **Philippe Mercier – dissections**
- **Paul Bhogal - research**

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Content

- Update on the patophysiology of DAVS
- Indirect and direct pial arterial supply
- Cases

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Mironov's etiological DAVS classification

- 96 patients with DAVS
- Classification according to the supposed pathogenesis of the DAVS
- The hypotheses were:
  - The angiomorphology depends on the location of the venous recipient
  - A territorial classification can be created based on the acquired venous characteristics
- Flow physics (Starling-resistor and Venturi effects) were applied to explain how DAVS emerge and why the differ in different locations

Pathogenetical Consideration of Spontaneous Dural Arteriovenous Fistulas (DAVFs), A. Mironov; Acta Neurochir (1994)

- **Type1: DAVS of the dural sinuses**
- **Type 2: DAVS of the cavernous sinuses**
- **Type 3: DAVS of the Galen's system**
- **Type 4: DAVS of the venous plexuses at the base of the skull**
- **Type 5. DAVS of the cortical veins situated near the dural sinuses**

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Thrombotic phenomenon

- **Thrombosis was frequently found in large sinuses**
  - **72% in type 1 and 62% in type 2**
- **Could not image thrombosis in basal plexus or small veins**
- **Almost all patients without proven thrombosis had other conditions such as hypercoagulopathy, diabetes mellitus, otitis media, other infections**

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Venous hypertension

- **46 rats**
  - **22 anastomosis CCA-EJV**
  - **13 anastomosis CCA-EJV and occlusion contralateral posterior facial vein**
  - **11 controls – only unilateral facial vein occlusion**
  - **Ligation of shunts and follow up angiography after 2-3 months**

Development of acquired arteriovenous fistulas in rats due to venous hypertension , Terada, T; J Neurosurg 1994

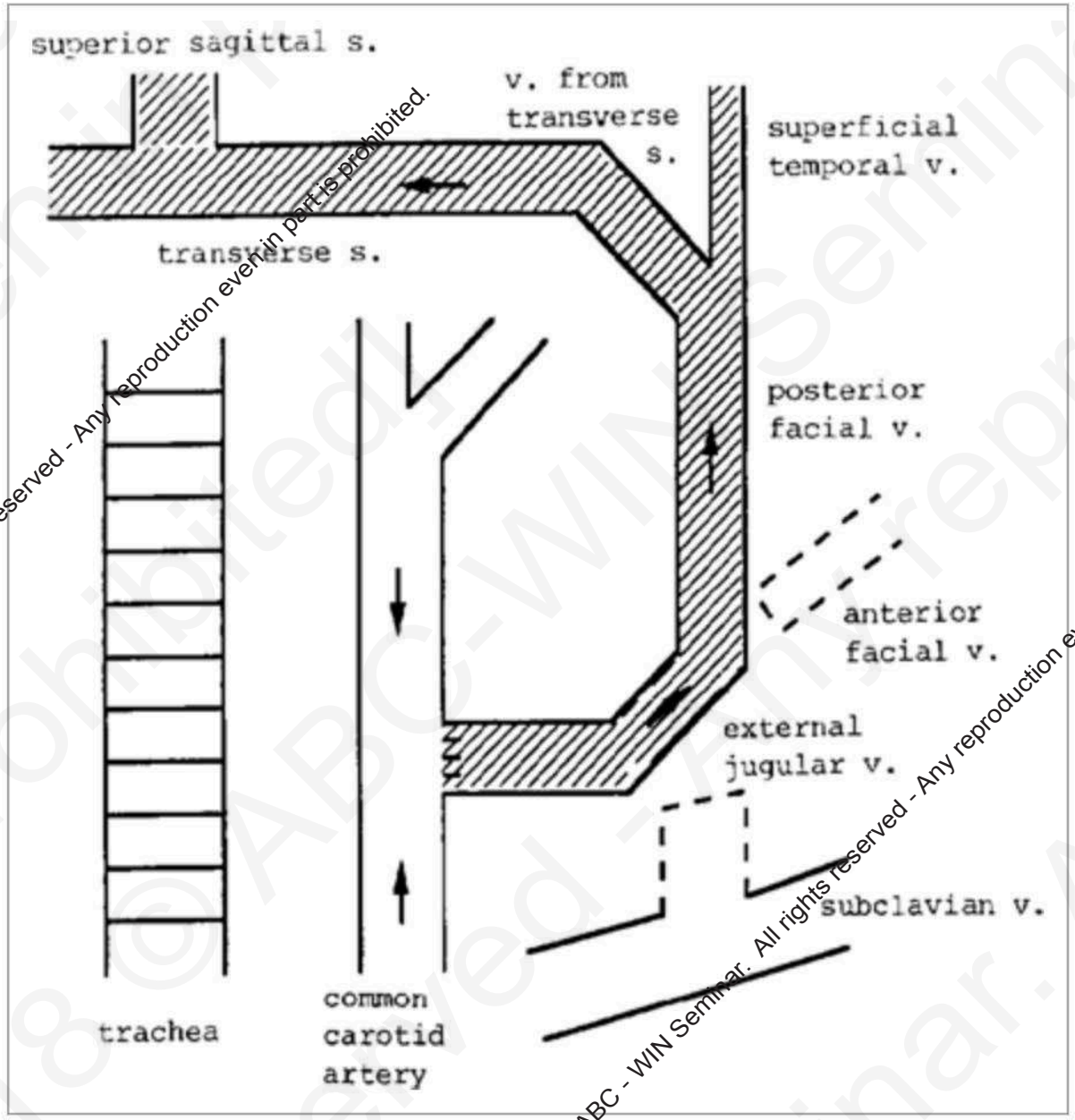

FIG. 1.

Diagram of shunt surgery. Abbreviations: s. = sinus; v. = vein. *Arrows* show the direction of blood flow.

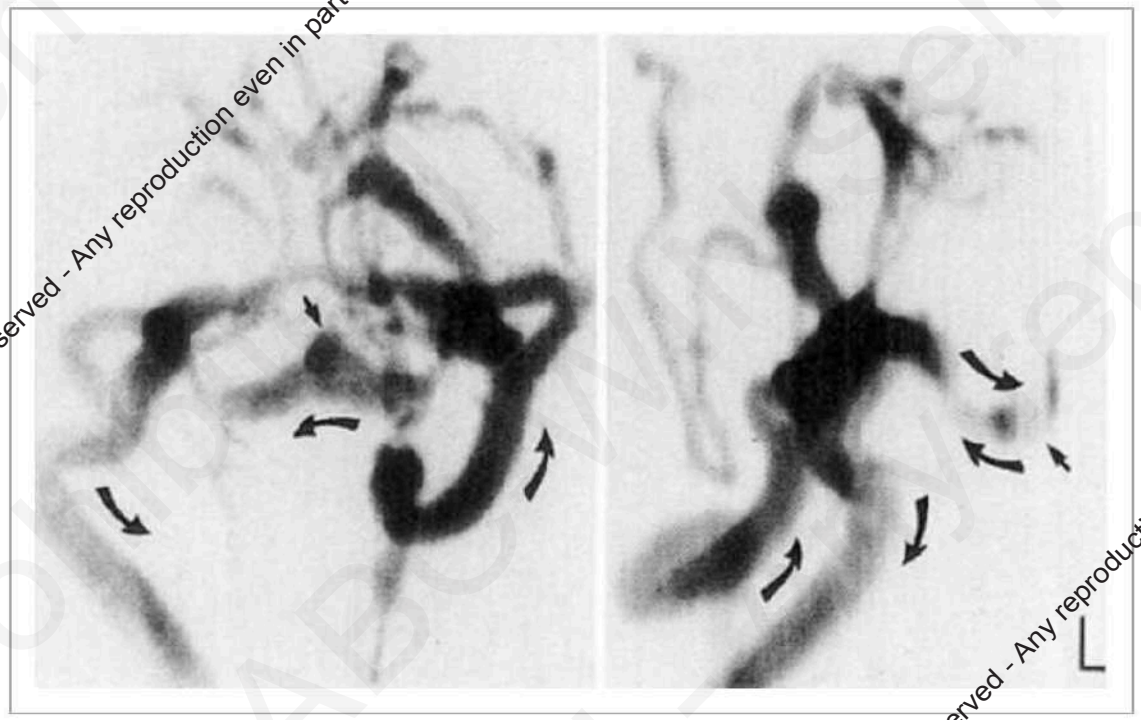

FIG. 2.

Left common carotid angiograms, frontal *(left)* and lateral *(right)* views, in a Group I rat showing dilated veins, the transverse sinus, and the posterior part of the superior sagittal sinus *(straight arrows)*. These veins drain into the right external jugular vein. Arterial structures are not well shown because of the steal phenomenon of the large arteriovenous shunt. The direction of blood flow is shown by *curved arrows*.

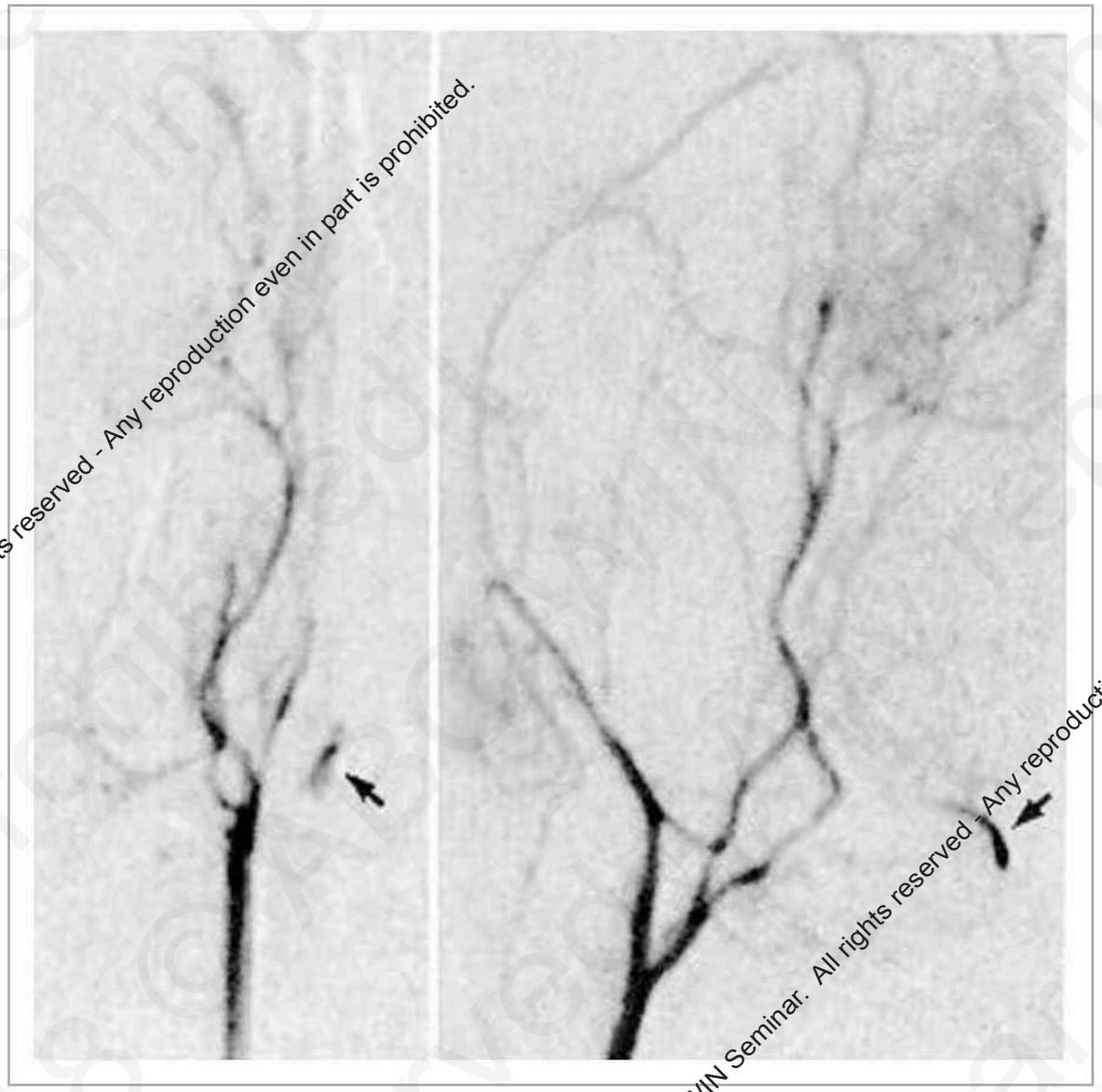

FIG. 3.

Right common carotid angiograms, frontal *(left)* and lateral *(right)* views, arterial phase, in a rat with a new dural arteriovenous fistula. Opacification of the posterior part of the superior sagittal sinus *(arrows)* can be seen.

2-3 months follow up

- **AVF's developed in**
  - **three rats (14%) in Group I**
  - **three rats (23%) in Group II**
  - **no rats in Group III**
- **One of these newly formed fistulas was located at the dural sinus, analogous to the human dural AVF and was still present at one week follow up angiography**
- **The other five were located in the subcutaneous tissue**

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Conclusion

- **Chronic venous hypertension of 2 to 3 months' duration, without associated venous or sinus thrombosis, can induce new AVF's affecting the dural sinuses or the subcutaneous tissue.**

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bFGF in DAVS

- Samples from four human sinuses with DAVS and one control
- Immunohistochemistry for bFGF
- Immunohistochemistry for alpha smooth muscle actin, factor VIII related antigen, and macrophages to identify the bFGF positive cell types
- Control normal dural sinus showed negative staining by bFGF immunohistochemistry
- In sinuses of the dAVF patients, smooth muscle cells, endothelial cells, and meningeal cells were stained positively in various degrees by bFGF immunohistochemistry

The Role of Angiogenic Factor bFGF in the Development of Dural AVFs , T. Terada et al; Acta Neurochir 1996

Conclusion

- bFGF-like immunoreactivity was strongly shown in the sinus walls of dural AVF patients.
- The bFGF immunoreactive cells were mainly smooth muscle cells and endothelial cells.
- bFGF may play an important role in the development of dAVFs.

The Role of Angiogenic Factor bFGF in the Development of Dural AVFs , T. Terada et al; Acta Neurochir 1996

Venous hypertension II

- **40 rats underwent a surgical procedure to induce venous hypertension, venous outflow occlusion, and sagittal sinus thrombosis**
  - **CCA-EJV-anastomosis**
  - **Ligation of contralateral jugular vein**
  - **Thrombosis of SSS**
- **15 rats same procedure without AV shunt**
  - **CCA-EJV-coagulation**
  - **Ligation of contralateral jugular vein**
  - **Thrombosis of SSS**

Redefined role of angiogenesis in the pathogenesis of dural arteriovenous malformations, Lawton M et al; J Neurosurg 1997

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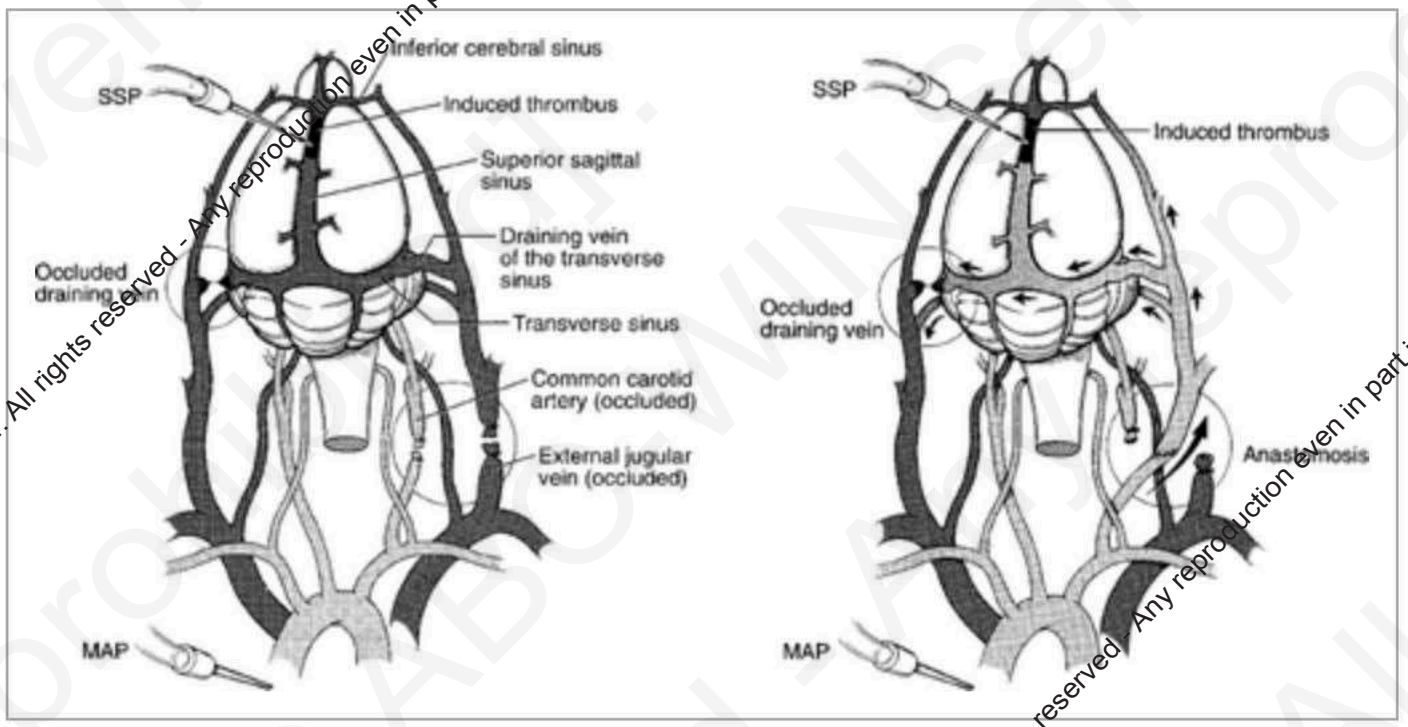

FIG. 1.

Illustrations showing the surgical procedures that induced *(left)* or did not induce *(right)* venous hypertension in rats.

Sampling

- After 1, 2, or 3 weeks, dura mater was obtained from one group of hypertensive rats and from one group of nonhypertensive rats
- Assayed for angiogenic activity (rabbit cornea bioassay)
- 10 hypertensive rats was not assayed to determine if sampling affected dural AVM formation

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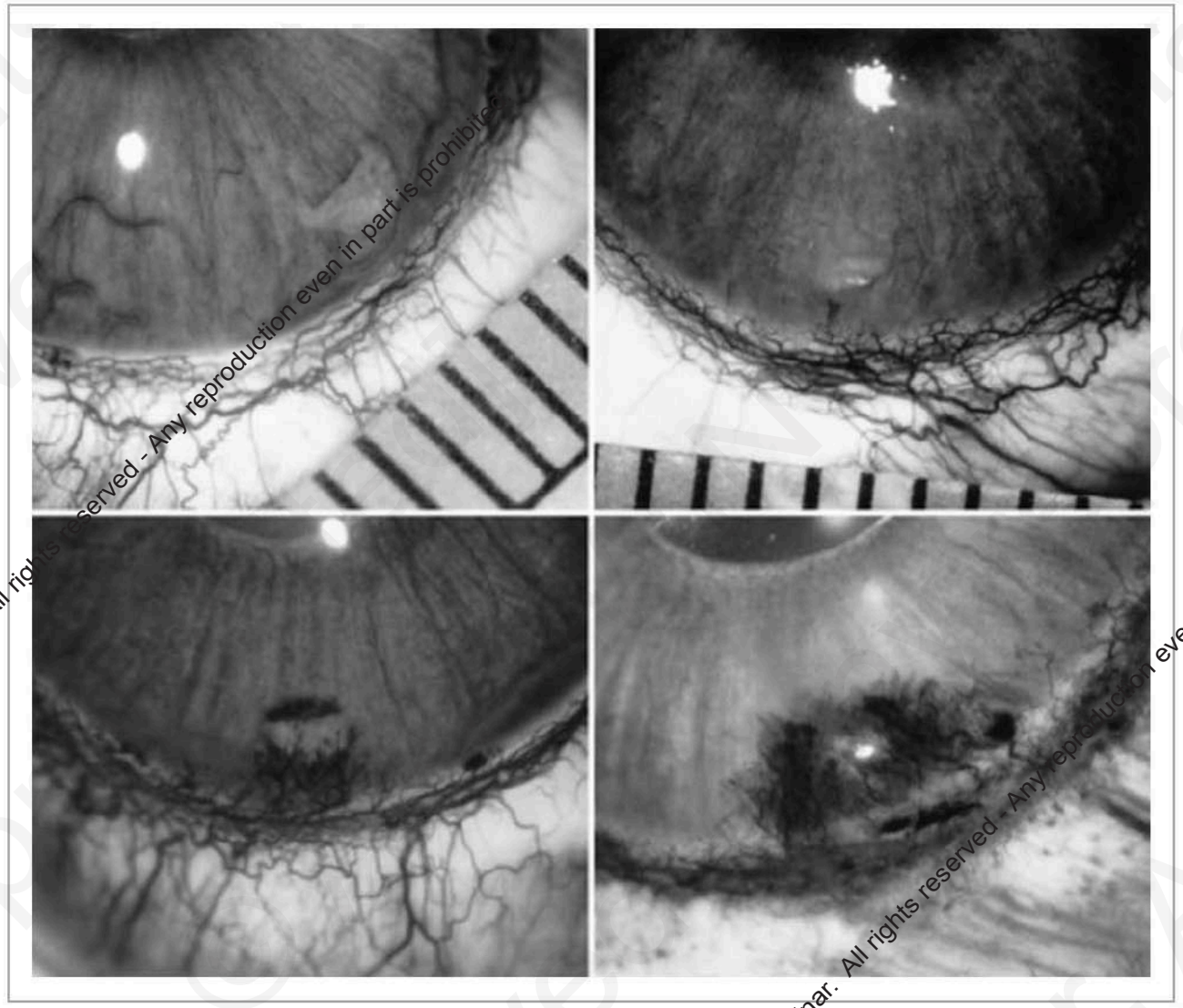


FIG. 2.

Photographs showing angiogenic activity of rat dura mater assayed in the rabbit cornea. Blood vessels grow from the cornea—sclera junction toward the implant in the corneal pocket. *Upper Left:* No vessels (angiogenesis index of 0). *Upper Right:* One to 10 vessels (angiogenesis index of 1). *Lower Left:* More than 10 vessels, loosely packed with the iris visible through gaps between the vessels (angiogenesis index of 2). *Lower Right:* More than 10 vessels, tightly packed with no gaps between the vessels (angiogenesis index of 3). Original magnification × 10.

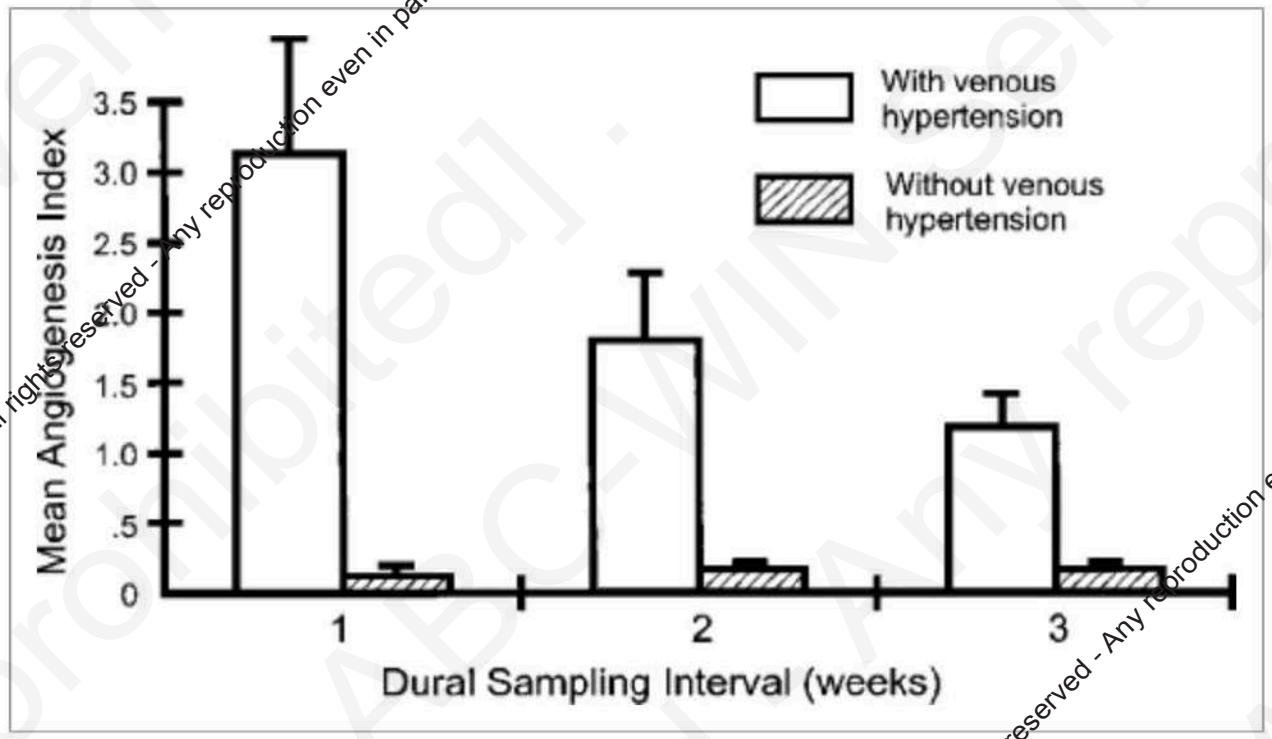


FIG. 3.

Bar graph displaying angiogenic activity (average angiogenesis index) of dura sampled from rats with and without venous hypertension at 1, 2, and 3 weeks. *Error bars* depict the standard error of the mean.

Conclusion

- **Development of dural AVMs correlated positively with both venous hypertension and angiogenic activity**
- **Venous hypertension may induce angiogenic activity either directly or indirectly by decreasing cerebral perfusion and increasing ischemia**
- **Dural AVM formation may be the result of aberrant angiogenesis**

- **Interestingly DAVS regressed after ligation of the shunt**

Experimental DAVS in a rat model

- **120 rats, 70 with occipital venous occlusion**
- **Venous hypoperfusion for 12 weeks**
- **High expression of VEGF and metalloproteinase-9 in occipital brain and adjacent dura mater**
- **Developed micro AV shunts in dura mater**

Local chronic hypoperfusion secondary to sinus high pressure seems to be mainly responsible for the formation of intracranial dural arteriovenous fistula. Chen, L. et al, Neurosurgery, 2009.

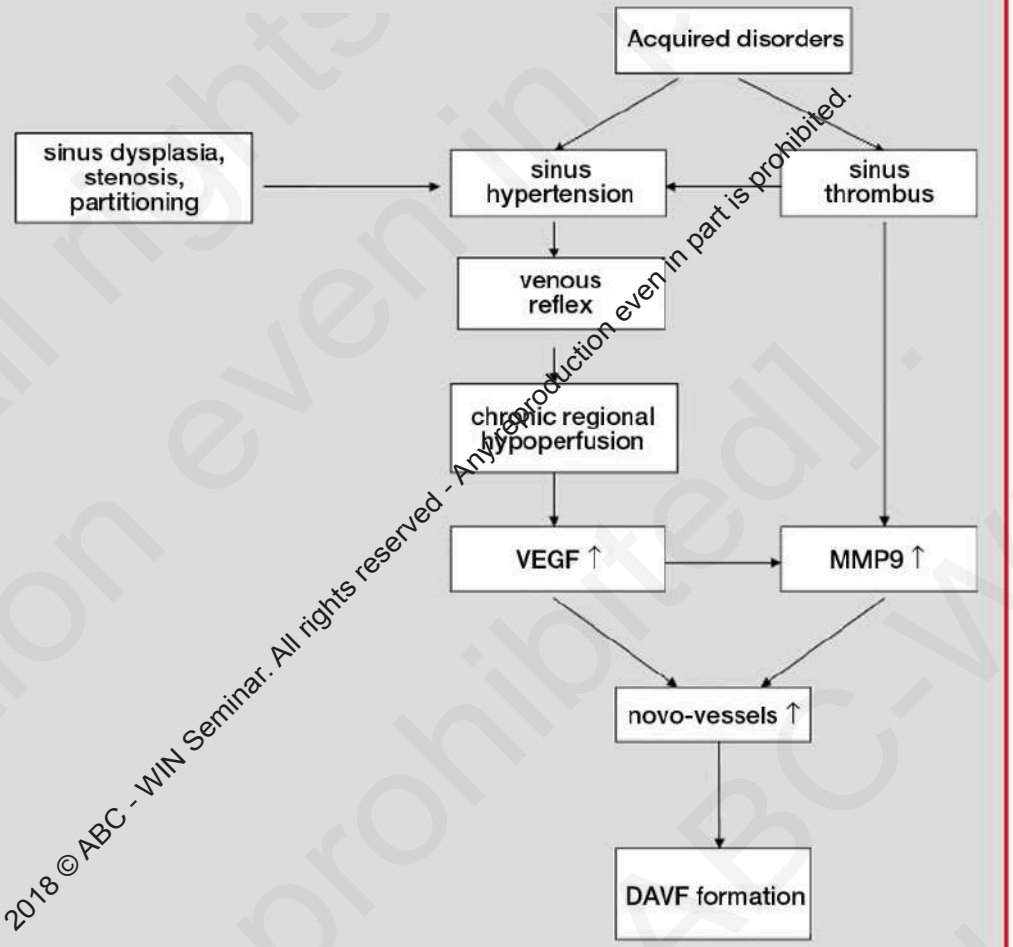


FIGURE 8. *Hypothesis of the mechanism of dural arteriovenous fistula (DAVF) formation. In this hypothesis, sinus high pressure is the main cause of angiogenesis of the dura mater and is critical for DAVF formation. Sinus thrombosis is a risk factor for elevation of sinus pressure. Chronic brain hypoperfusion is an early sign of venous hypertension. It will promote the expression of vascular endothelial growth factor (VEGF) and matrix metalloproteinase (MMP)-9 and enhance the abnormal angiogenesis of the dura mater. Chronic brain hypoperfusion is an important step from the progression of venous hypertension to DAVF formation.*

LOCAL CHRONIC HYPOPERFUSION SECONDARY TO SINUS HIGH PRESSURE SEEMS TO BE MAINLY RESPONSIBLE FOR THE FORMATION OF INTRACRANIAL DURAL ARTERIOVENOUS FISTULA.

Chen, Liang; Mao, Ying; Zhou, Liang-Fu

Neurosurgery. 64(5):973-983, May 2009.
DOI: 10.1227/01.NEU.0000341908.48173.EB

Quang Li et al 2104

- 216 rats
- Following transfection with VEGF recombinant adenovirus, angiogenesis in the dura mater of venous hypertensive rats was increased subsequent to the increase in the VEGF expression levels of the brain and dura mater.
- The rate of DAVF induction by venous hypertension was significantly reduced by the VEGFR antagonist due to reduced angiogenesis in the dura mater.
- In conclusion, VEGF and its receptor may be important in the formation of venous hypertension-induced DAVFs.

Activation of the VEGF/VEGFR signaling pathway is important in the formation of venous hypertension-induced DAVFs

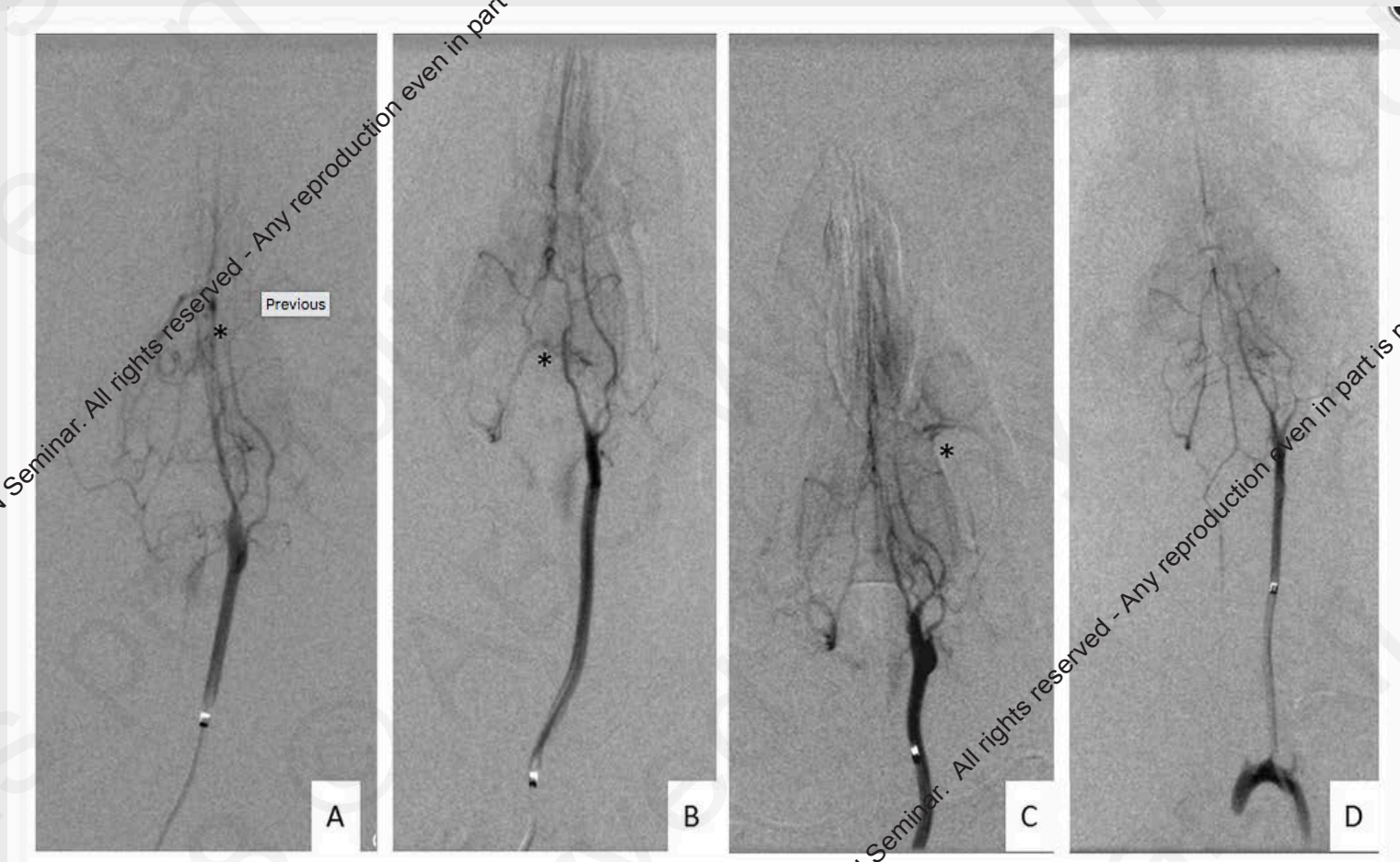


Figure 5 - Angiography of the left common carotid artery. Asterisks show the locations of dural arteriovenous fistulas (DAVFs). (A) DAVF in the sagittal sinus. (B) DAVF in the transverse sinus. (C) DAVF in the basis cranii. (D) Angiography of normal rats.

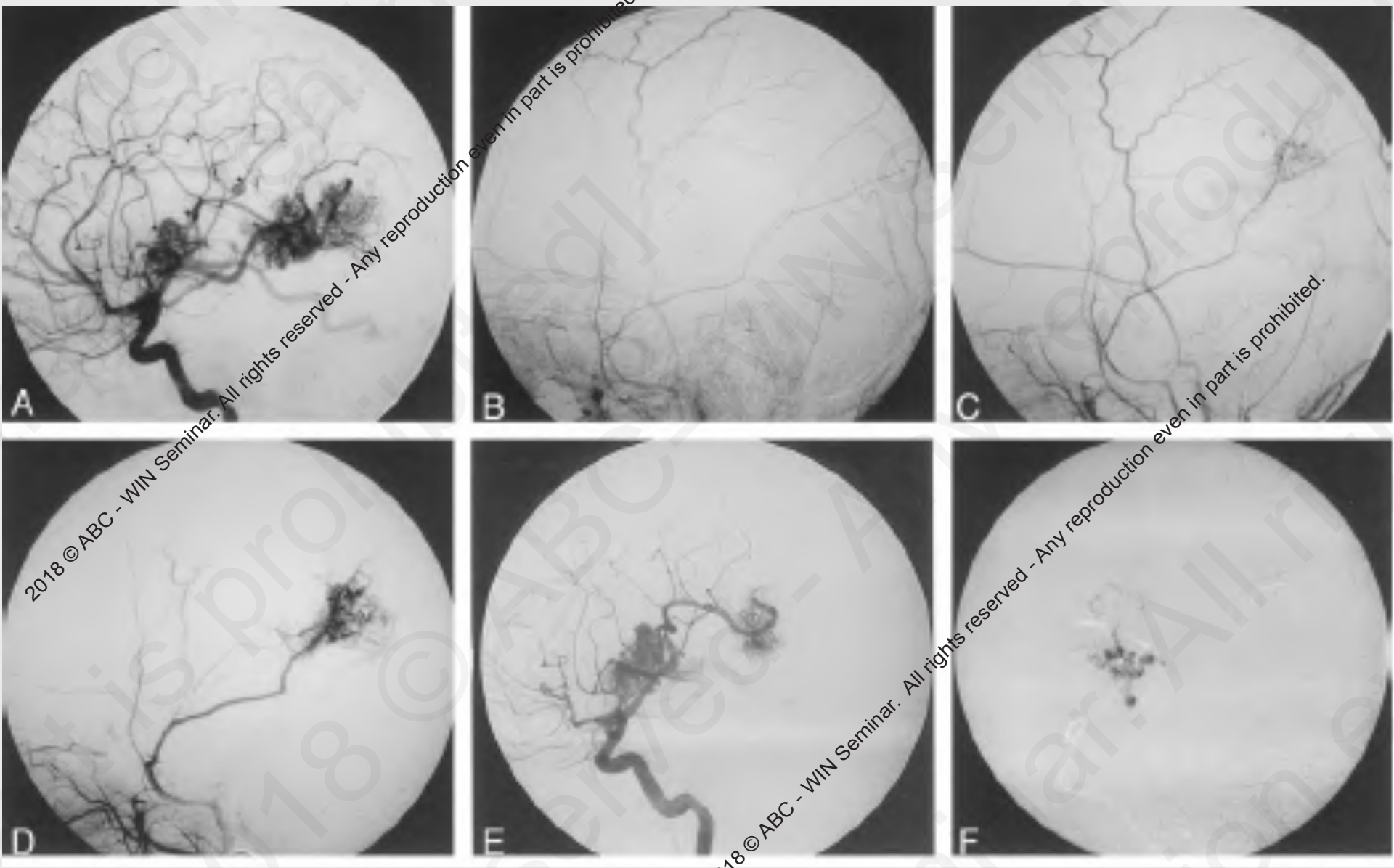
A pivotal role of the vascular endothelial growth factor signaling pathway in the formation of venous hypertension-induced dural arteriovenous fistulas, Qiang Li et al

Piodural arterial connections

- **Pial arteries crossing the subarachnoid space and penetrating the dura mater, anastomosing with a dural artery (normal finding or induced by AV shunt)**
- **Pial arteries crossing the subarachnoid space and penetrating the dura mater, supplying directly a DAVF**
- **Dural arteries crossing the subarachnoid space and supplying directly the brain or a BAVM**

“De novo” piodural vessels

- Vessels penetrating the innermost dural layer and crossing the arachnoid space to connect the pial and the dural arterial systems
- Develop as a response to ischemia?
- Frequent in Moyamoya, proliferating angiopathy etc
- May be found in chronic brain ischemia
- May appear as a late sequel after radiation treatment
- Not infrequent supply to brain AVM
  - Can be induced by partial embolisation
- May supply dural arteriovenous shunts



Transdural blood supply to cerebral arteriovenous malformations adjacent to the dura mater; Söderman, M., G. Rodesch, and P. Lasjaunias, AJNR 2002

Extremely brief embryology

- Crown-rump length 12-20mm (7.5-9.0 weeks)
- Differentiation cranium-dura-arachnoid-pia
- Separation of vasculature into extradural, dural and cerebral layers
- Closing of anastomosing channels
- Separation of the arteries that supply the cranium/dura and those that supply the brain (pial arteries)

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Arterial vascularization of the dura mater

- **Major meningeal branches**
  - **Located on the periostal surface of the calvarium**
  - **400-800 micron**
  - **Visible on angiography**

- **Primary anastomic arteries**
  - **Located on the periostal surface of the calvarium**
  - **100-400 micron**
  - **Often visible on angiography**
  - **Rich anastomotic network**
  - **Crosses the midline**

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- **Secondary anastomic arteries**
  - Located on the periostal surface of the calvarium
  - 50-100 microns
  - Generally short straight vessels

- **Arterial supply to the skull**
  - Penetrates the bone
  - 40-80 microns

- **Normal microscopic arteriovenous shunts**
  - Midportion of the dura mater

- **Penetrating vessels (arterioles)**
  - Penetrate the dura mater
  - Form an extremely rich anastomotic network close to the inner surface of the dura mater

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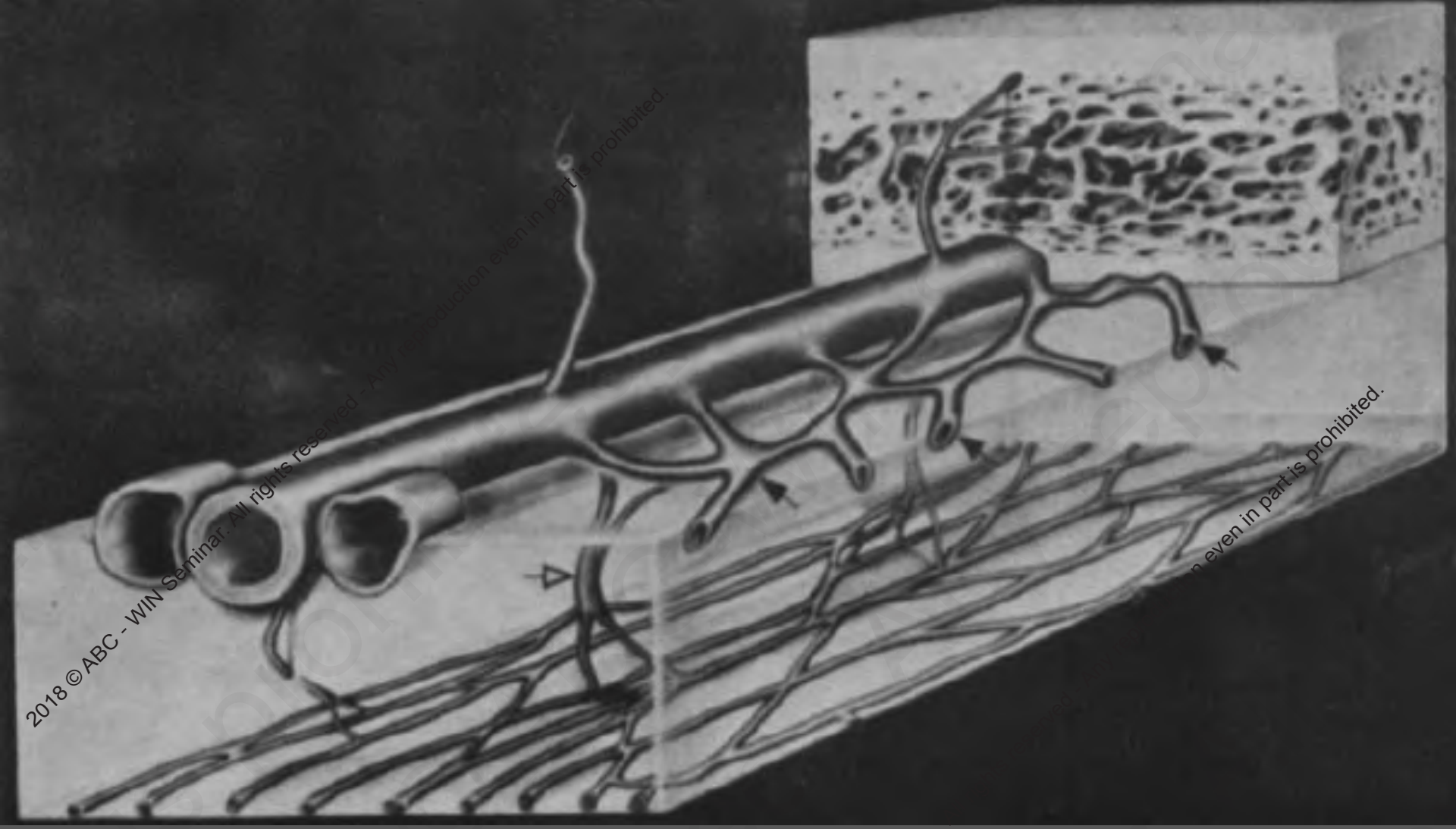
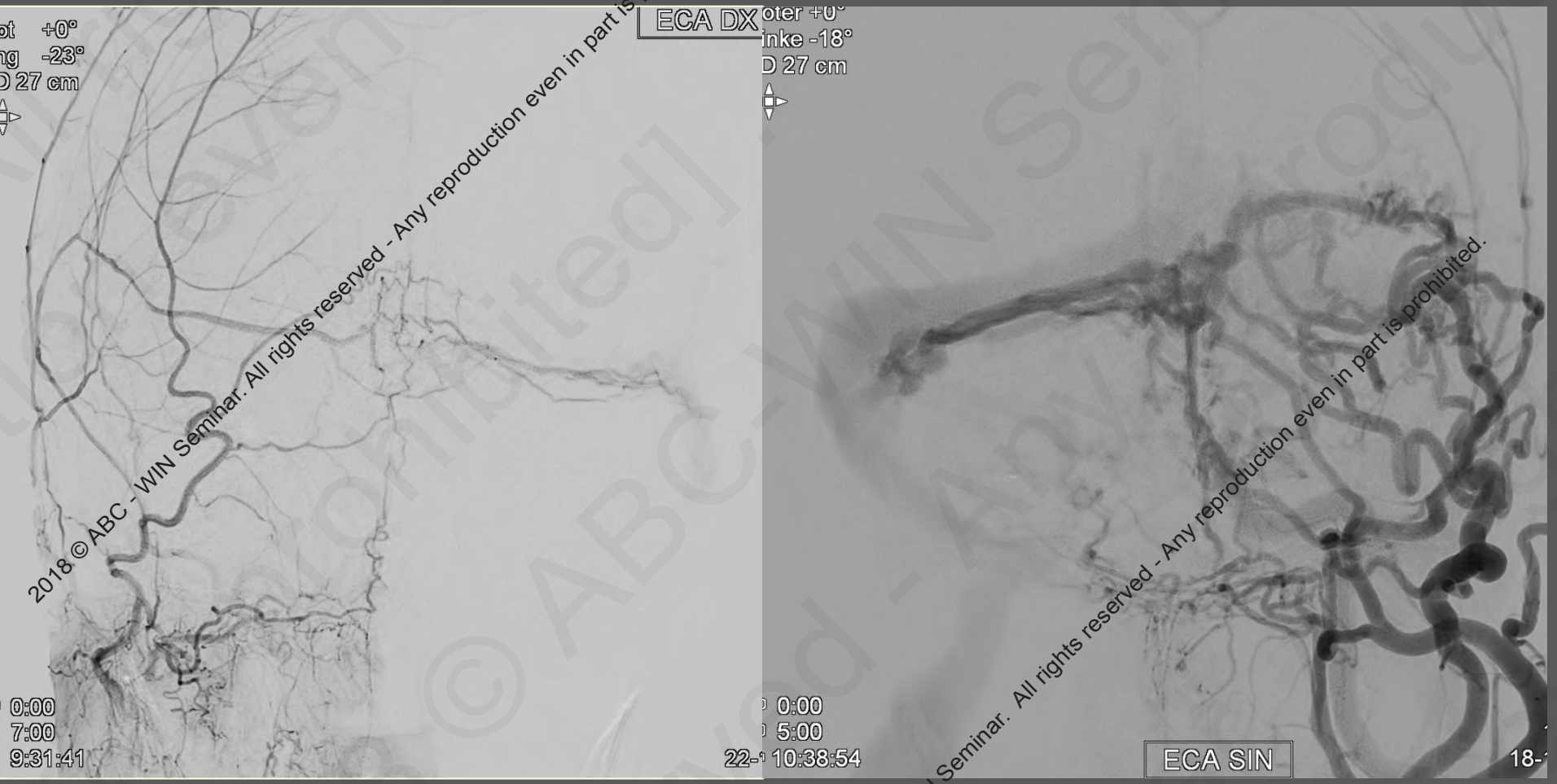


The Macro and Microvasculature of the Dura Mater
CW Kerber and TH Newton, Neuroradiology 1973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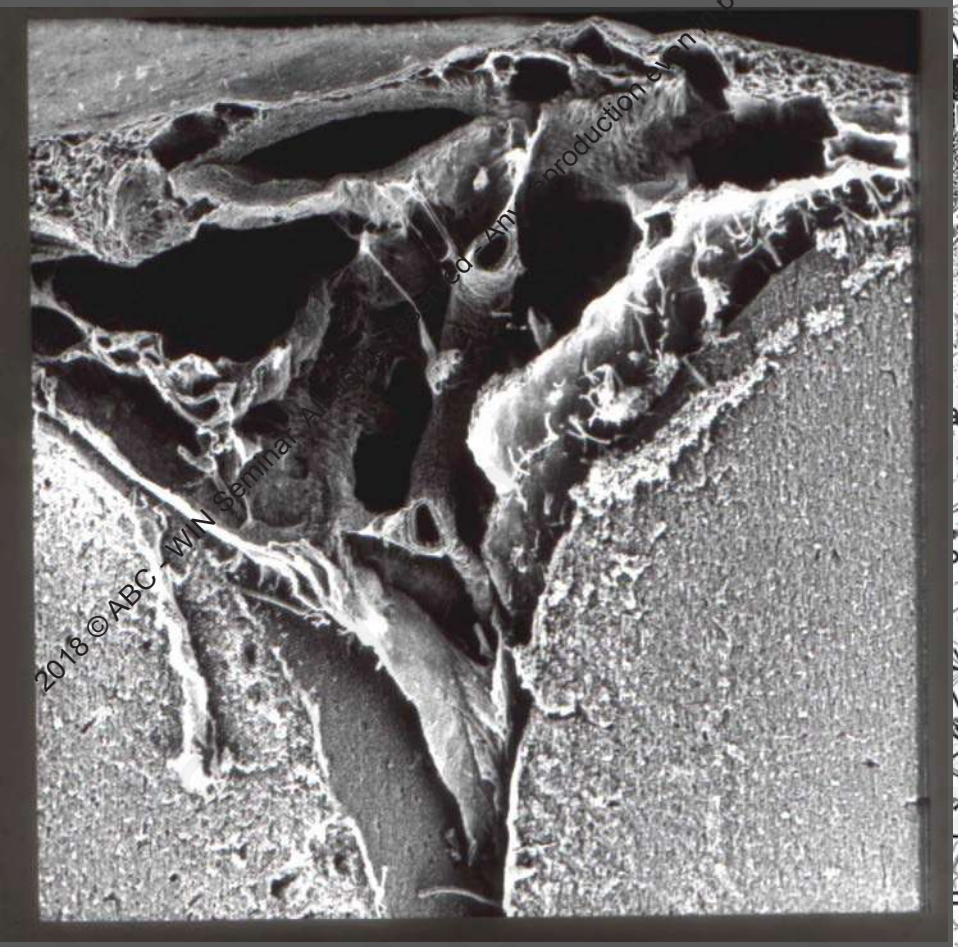
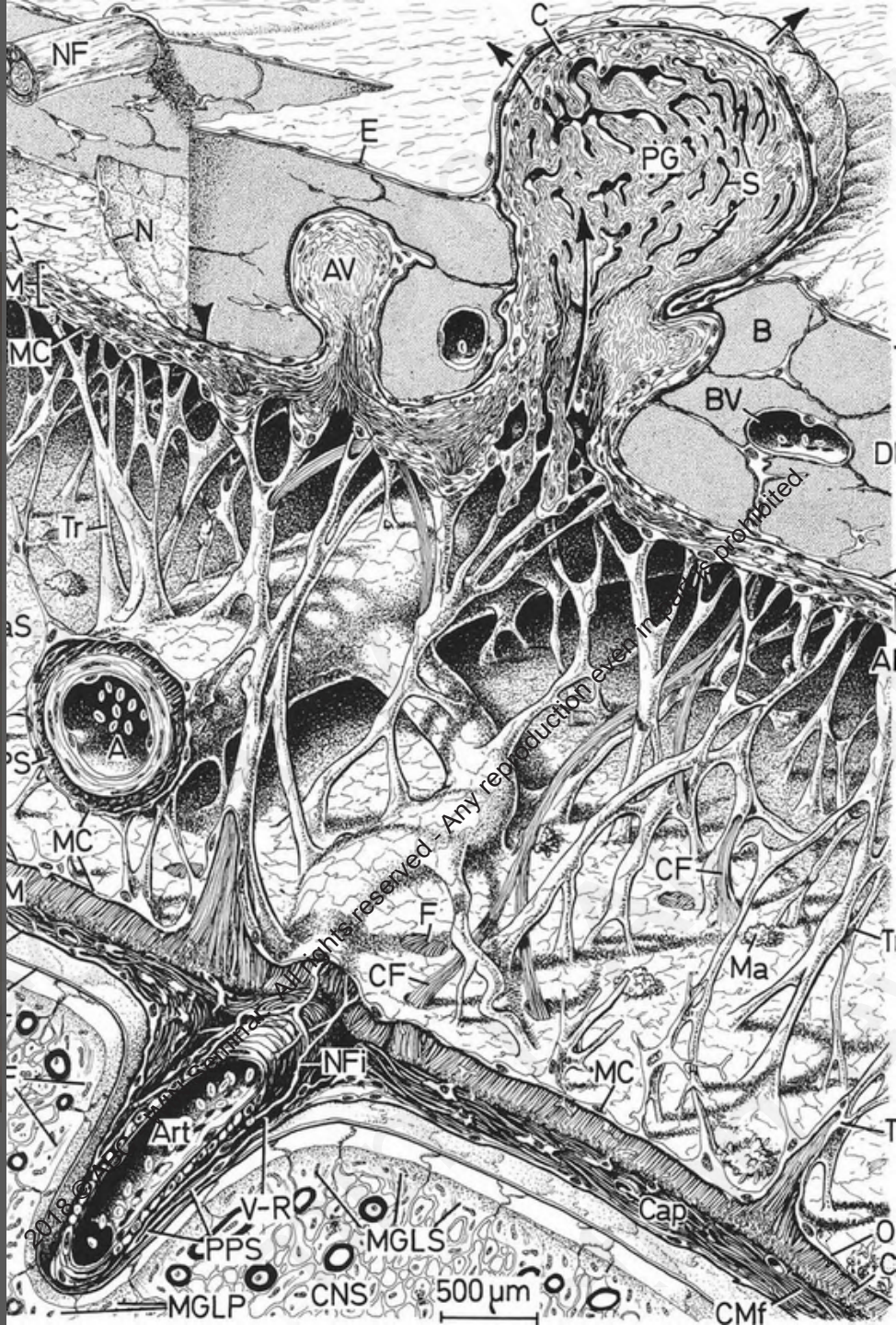
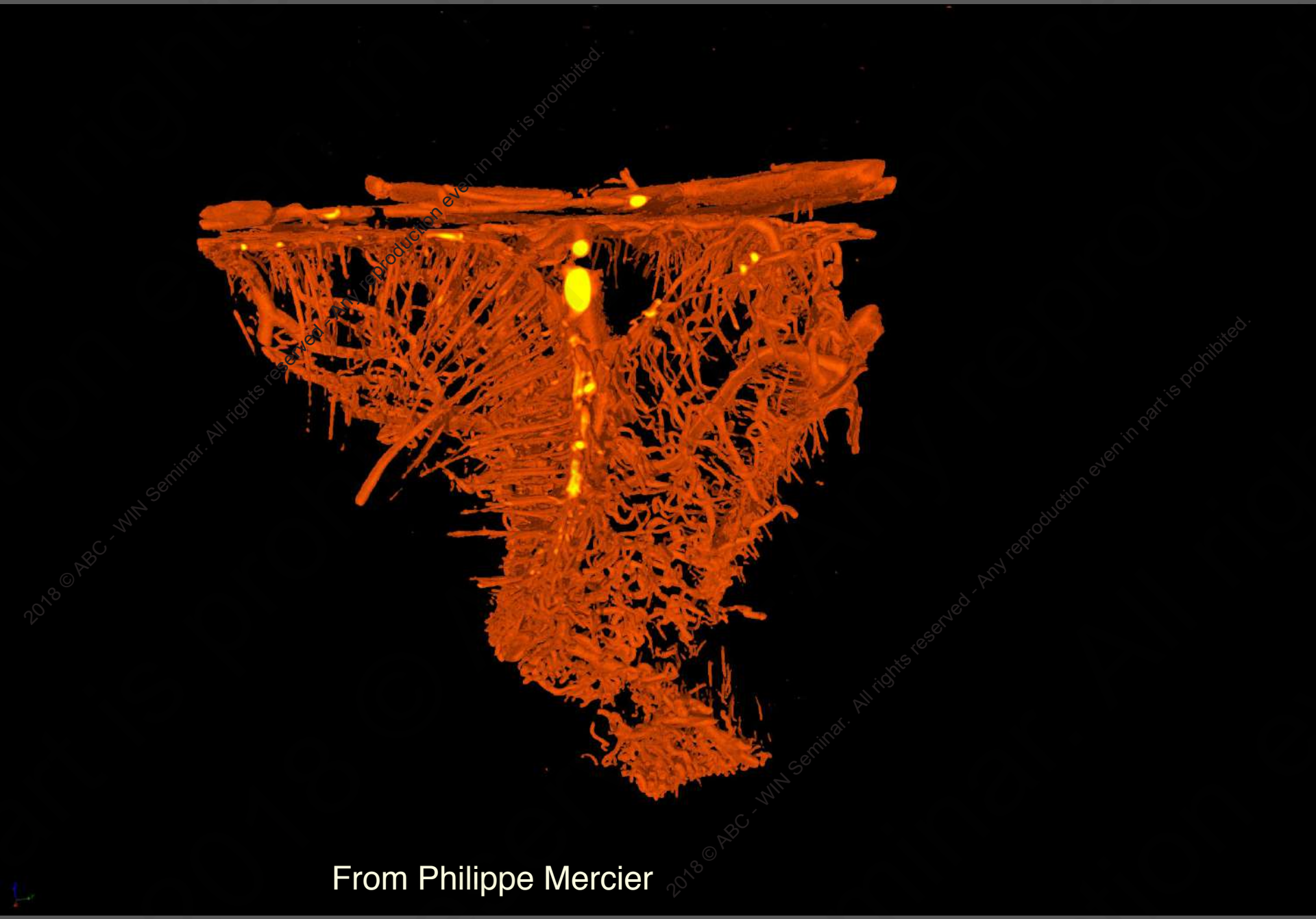
Subdural and subarachnoid spaces



Anatomical relationship: Hutchings M. JNS 1986

Human Microscopic Anatomy
Radivoj V. Krstic





From Philippe Mercier

“Normal” (common) piodural arterial connections

- ACA - Callosomarginal artery-anterior falcine artery
- ACA – Primitive Olfactory artery
- Post Cerebral A - artery of Davidoff and Schechter
- Circumferential arteries - artery of Davidoff and Schechter?
- Sup Cerebellar artery - medial dural tentorial branch
- AICA - the subarcuate artery
- PICA - posterior meningeal artery

- Not from the MCA

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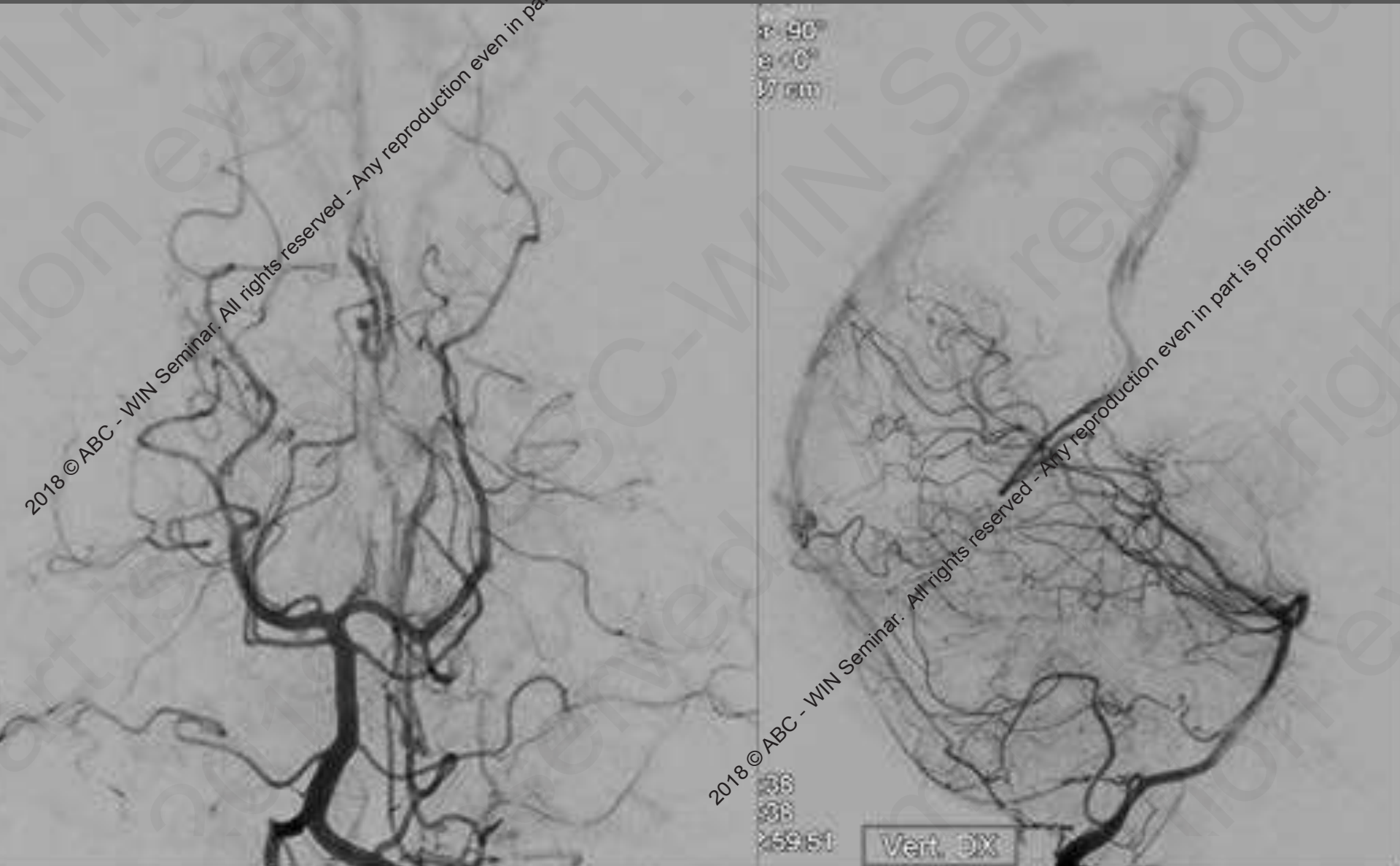
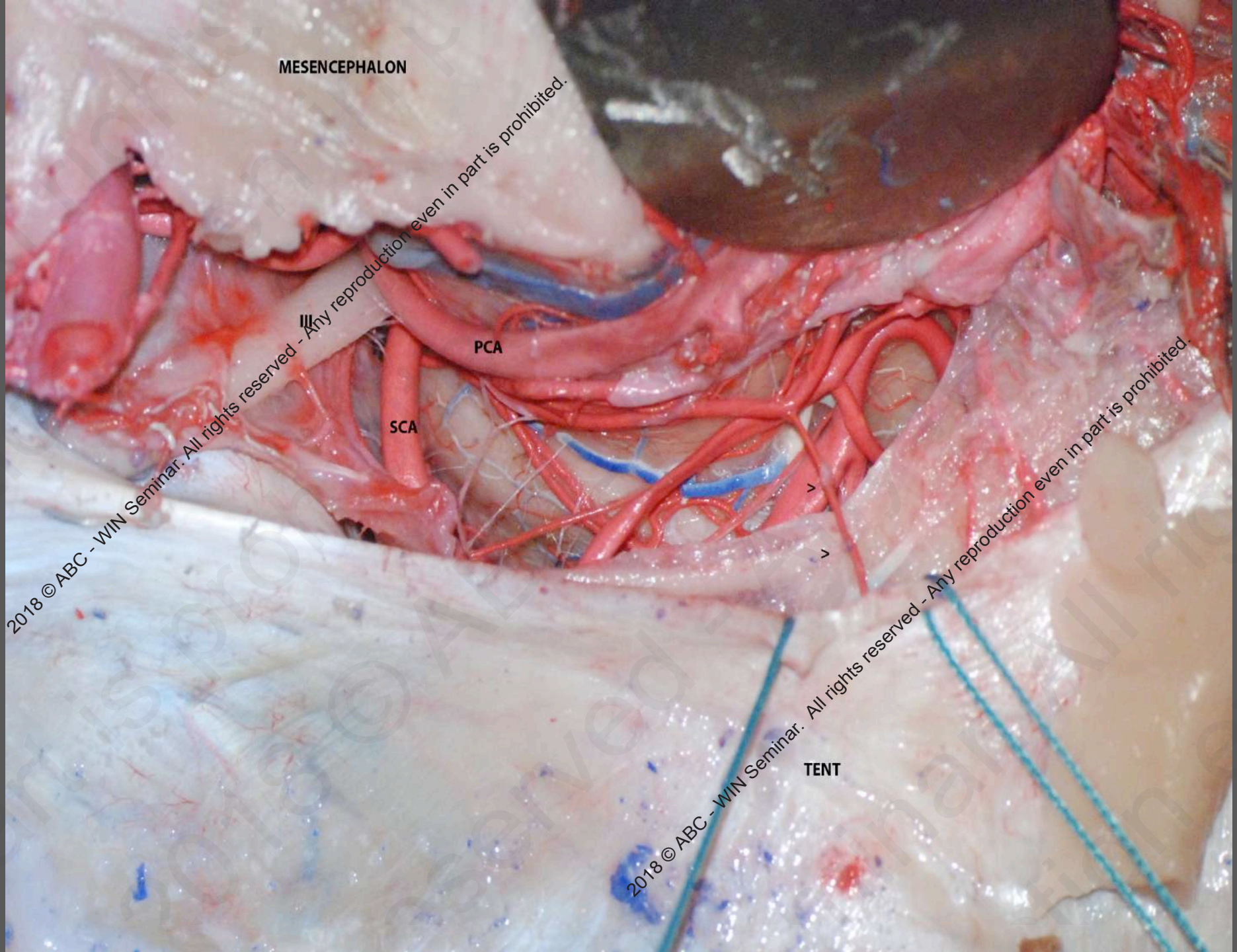
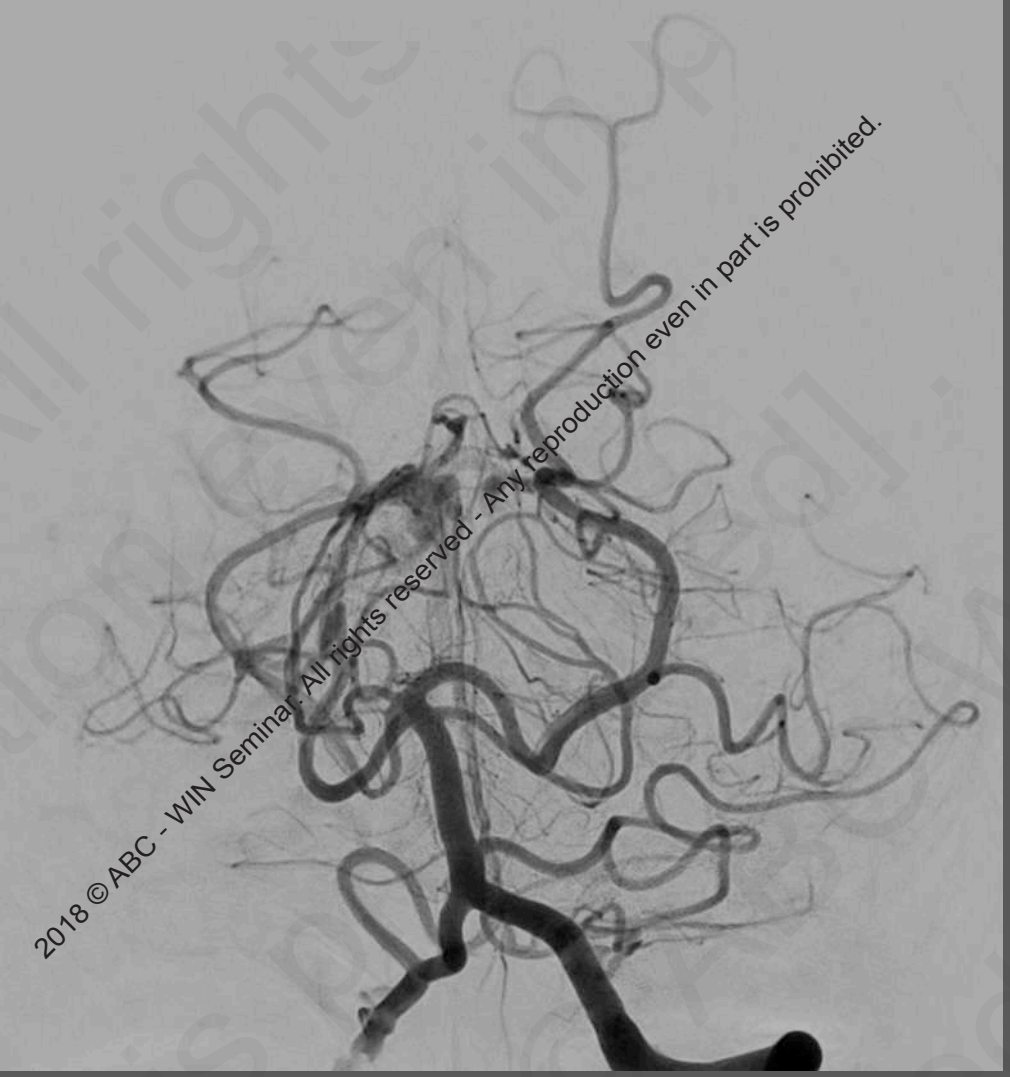
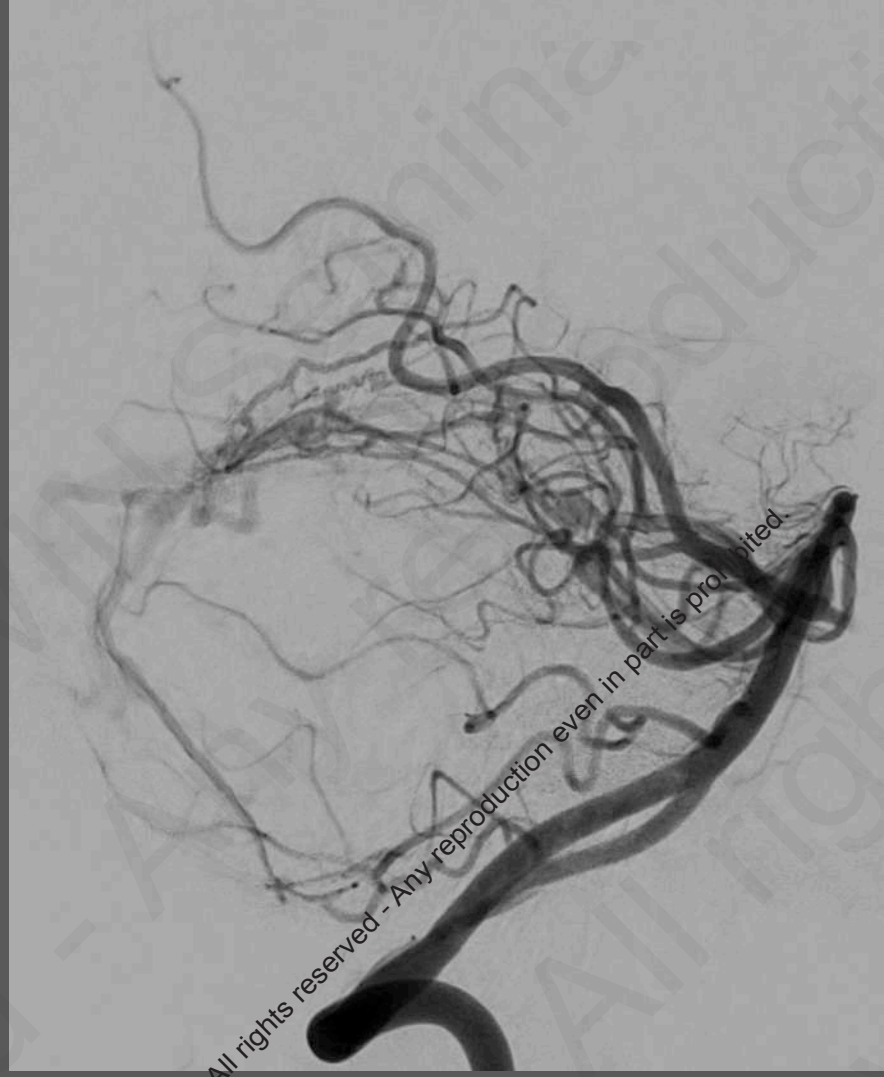
Post Cer Art – Falx Cerebri

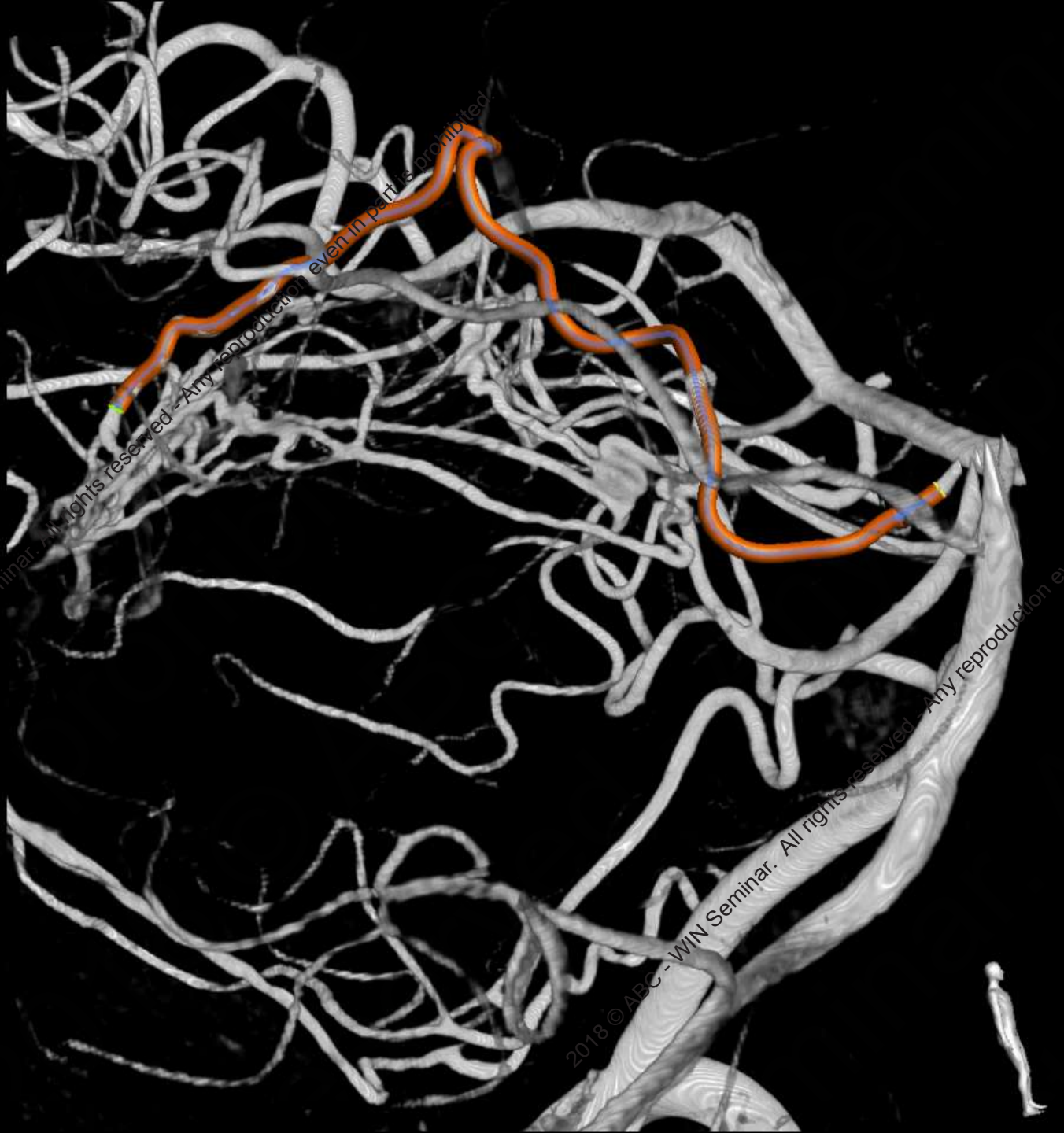
Artery of Davidoff and Schekter











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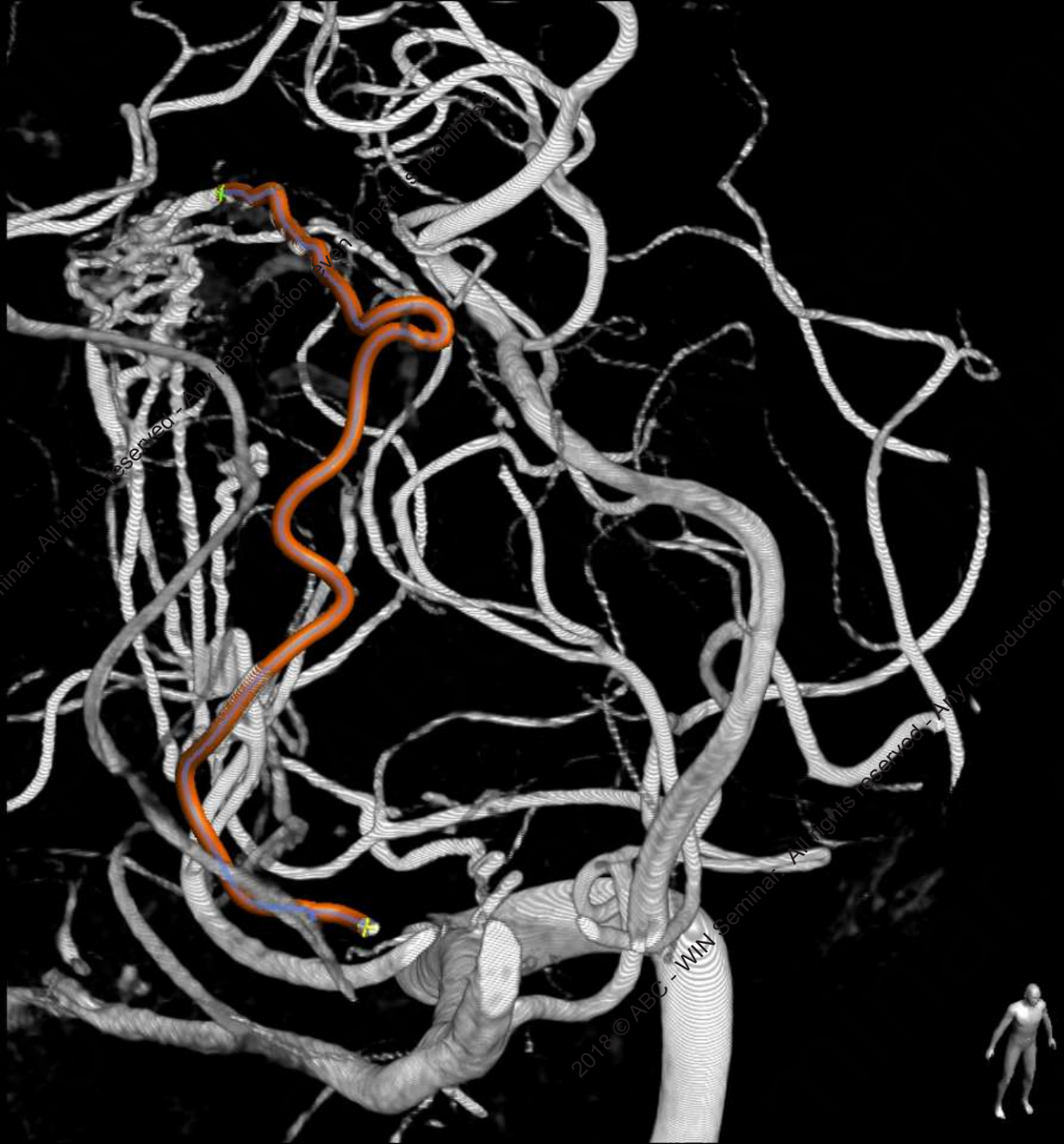

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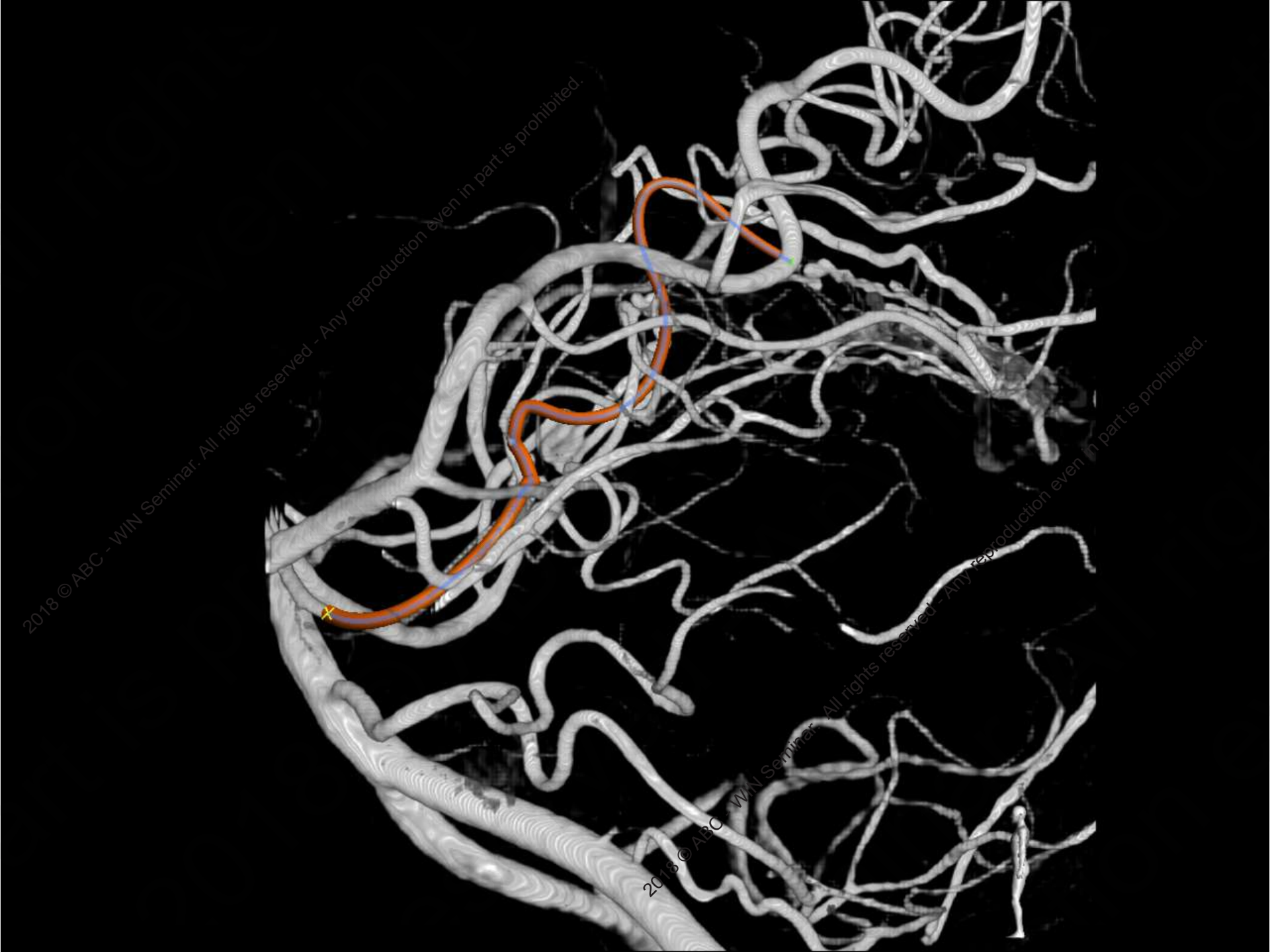

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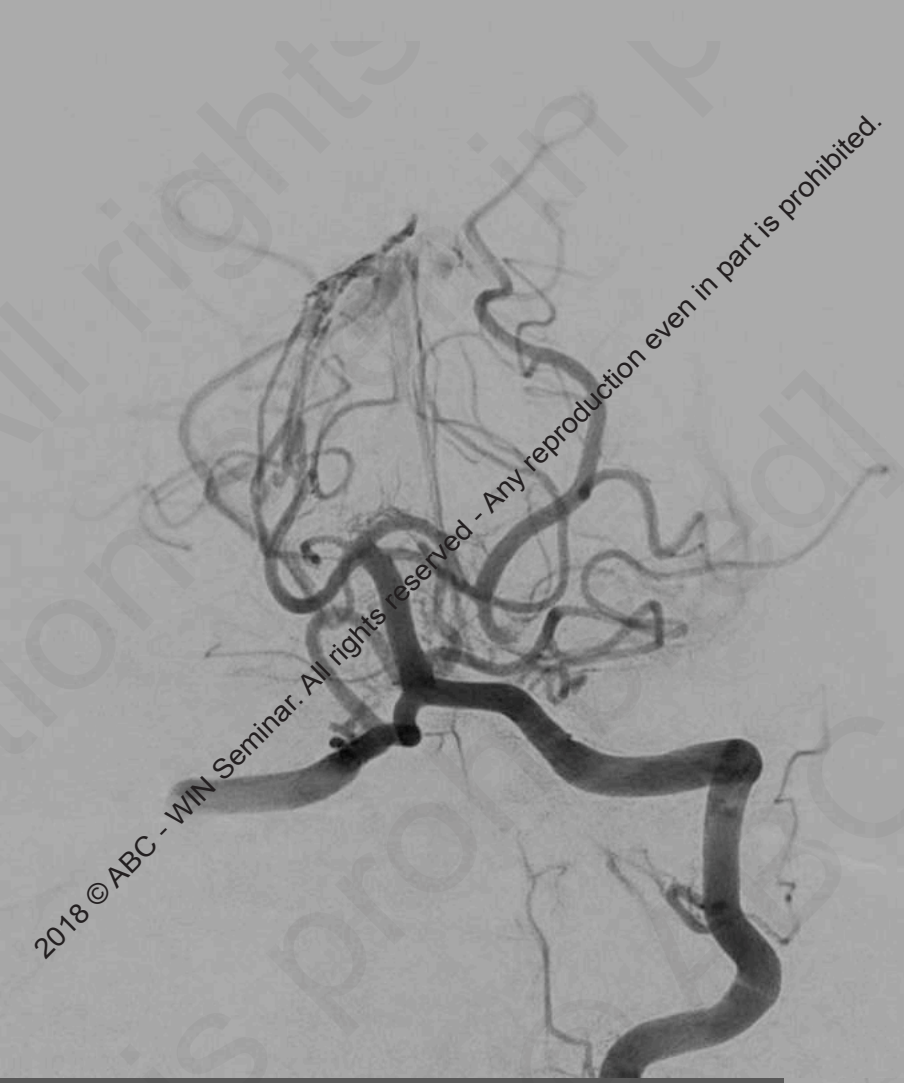
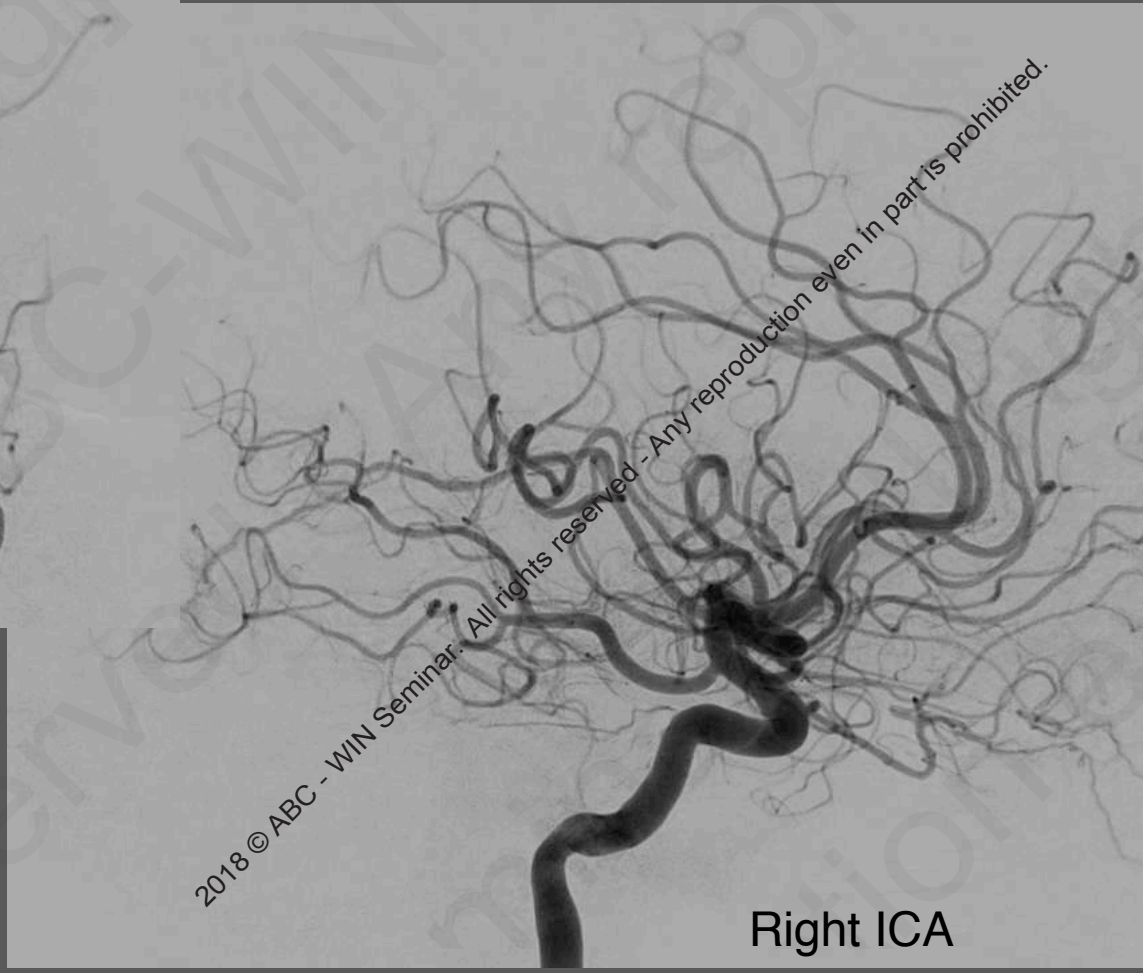
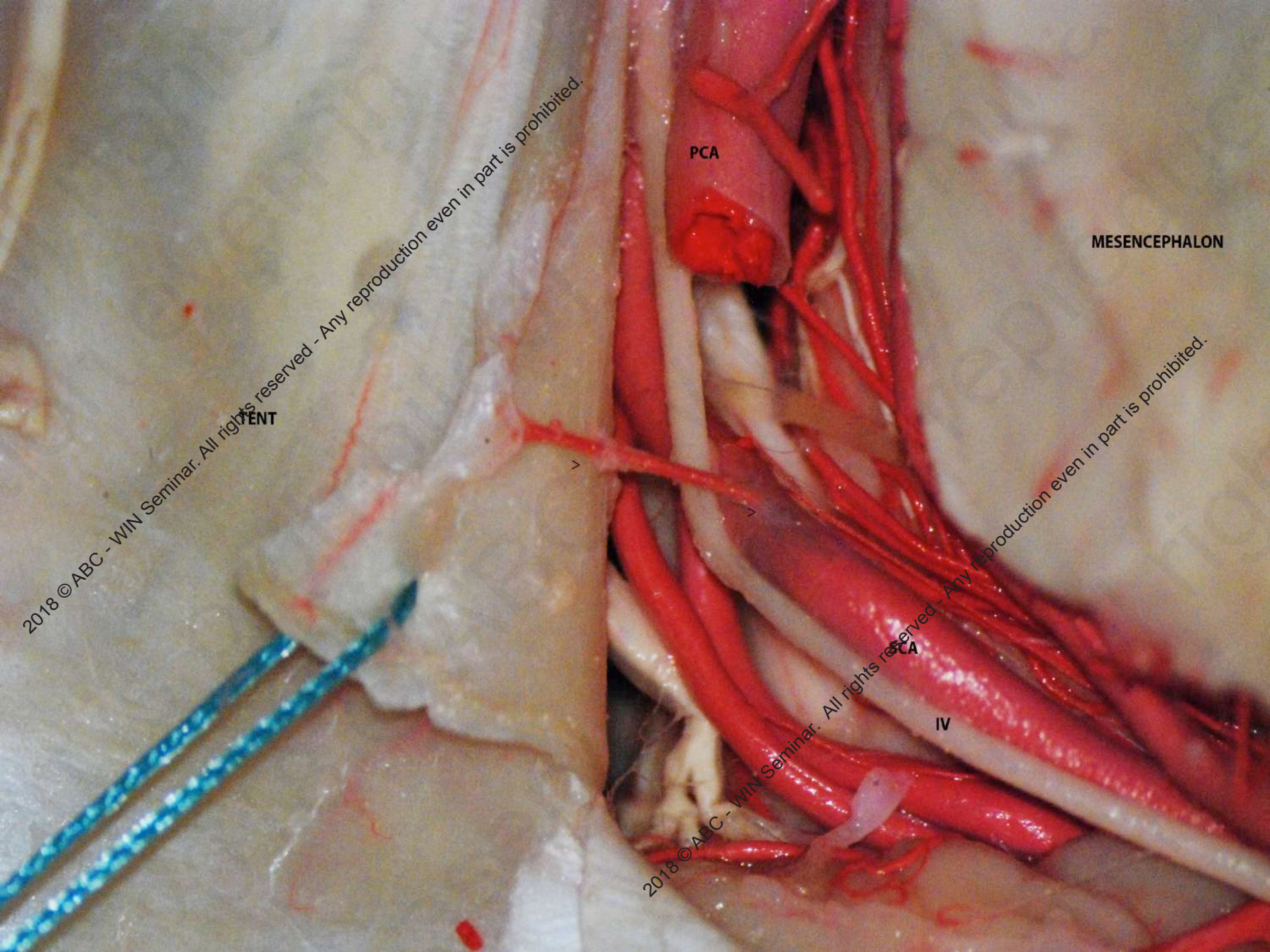




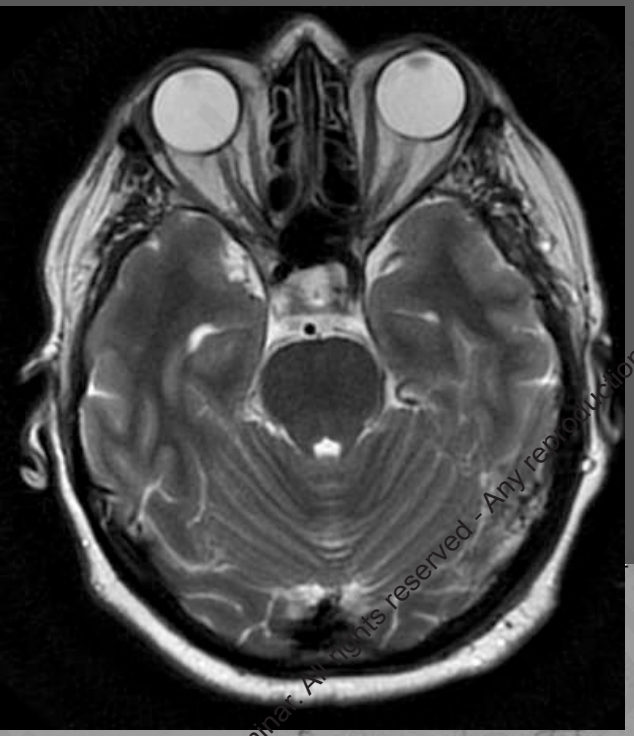
Right ICA

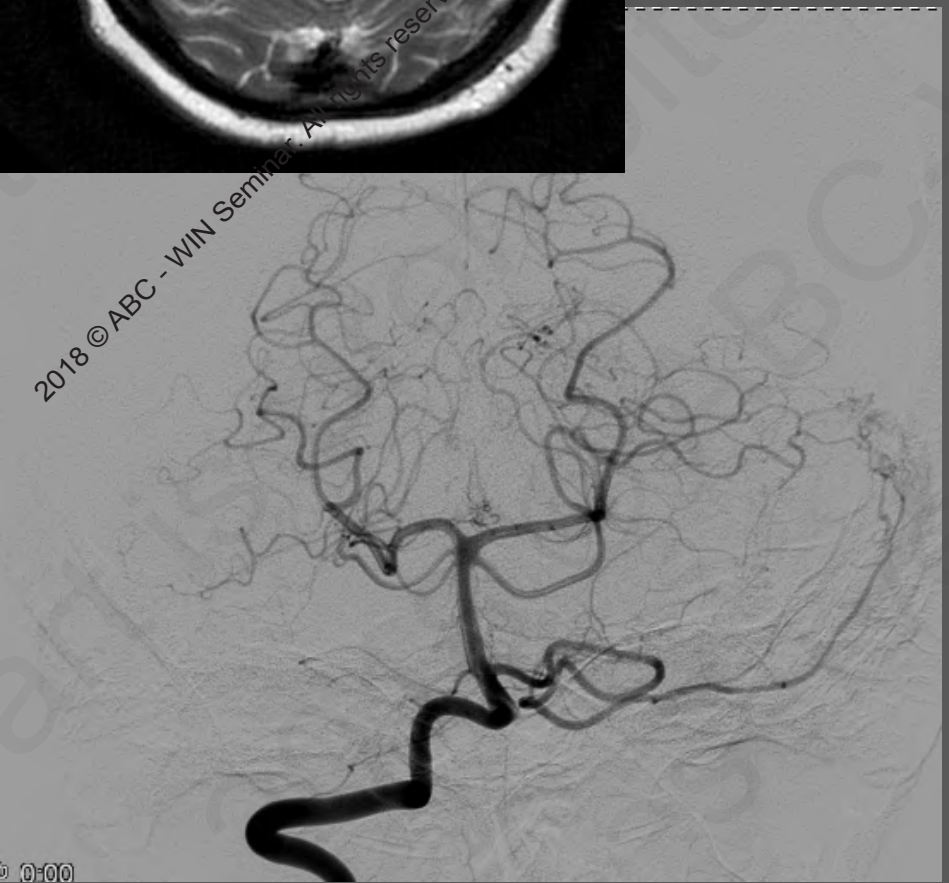


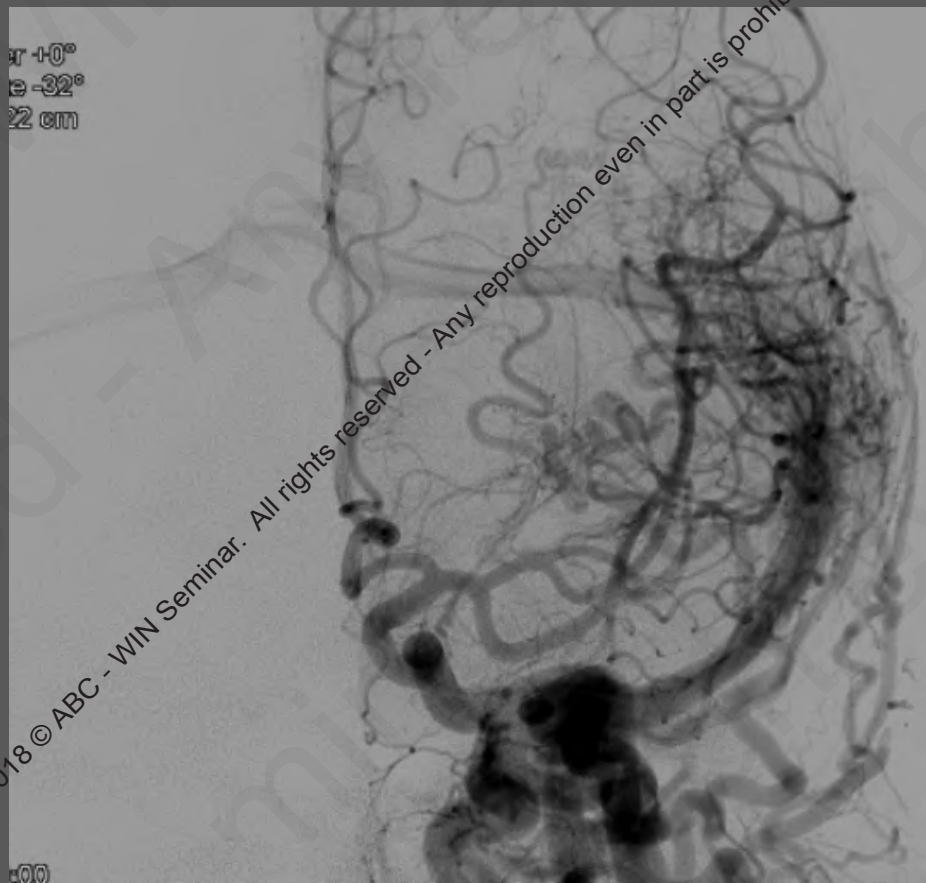
Piodural connections

- Always present in certain locations
- Direct connections may be induced by venous or arterial ischemia
- Local release of angiogenetic factors
  - VEGF, FGF, bTGF, etc
- Angiogenesis with formation of transdural, transarachnoid vessels, or
- Vasculogenesis with enlargement of already present arteries
- Genetic disposition may play a role
- Does have clinical relevance

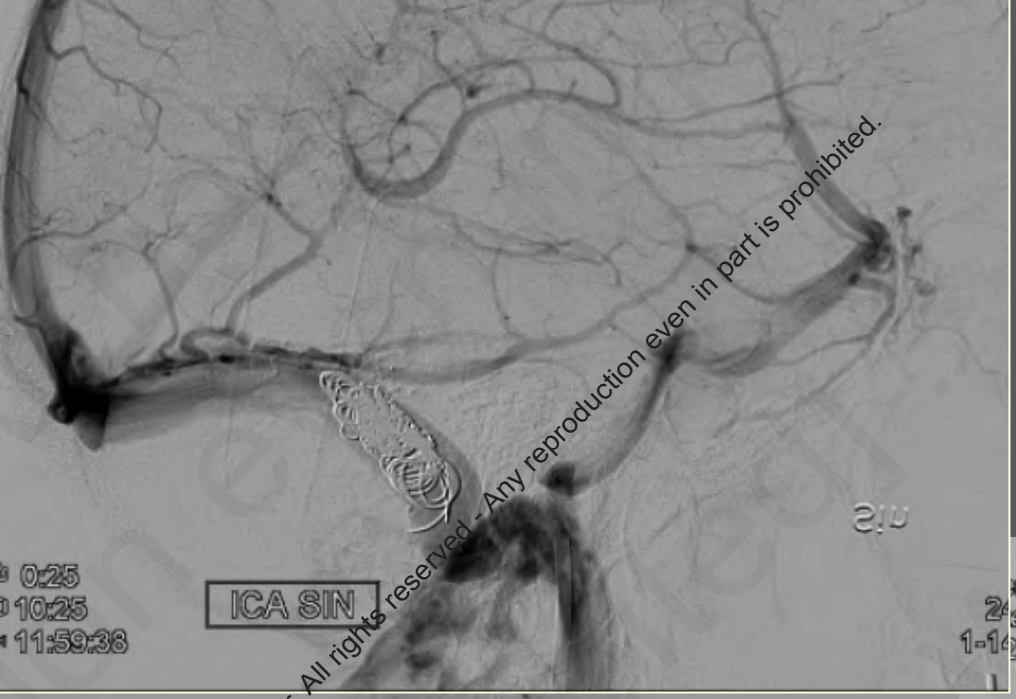
Transdural vessels in dural arteriovenous shunts

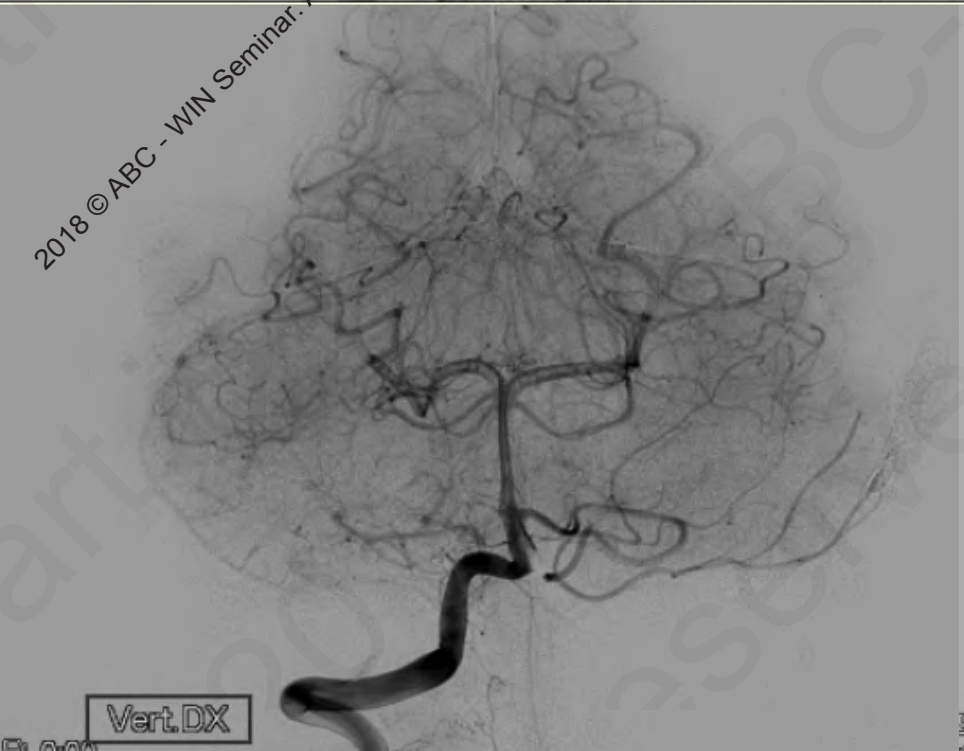


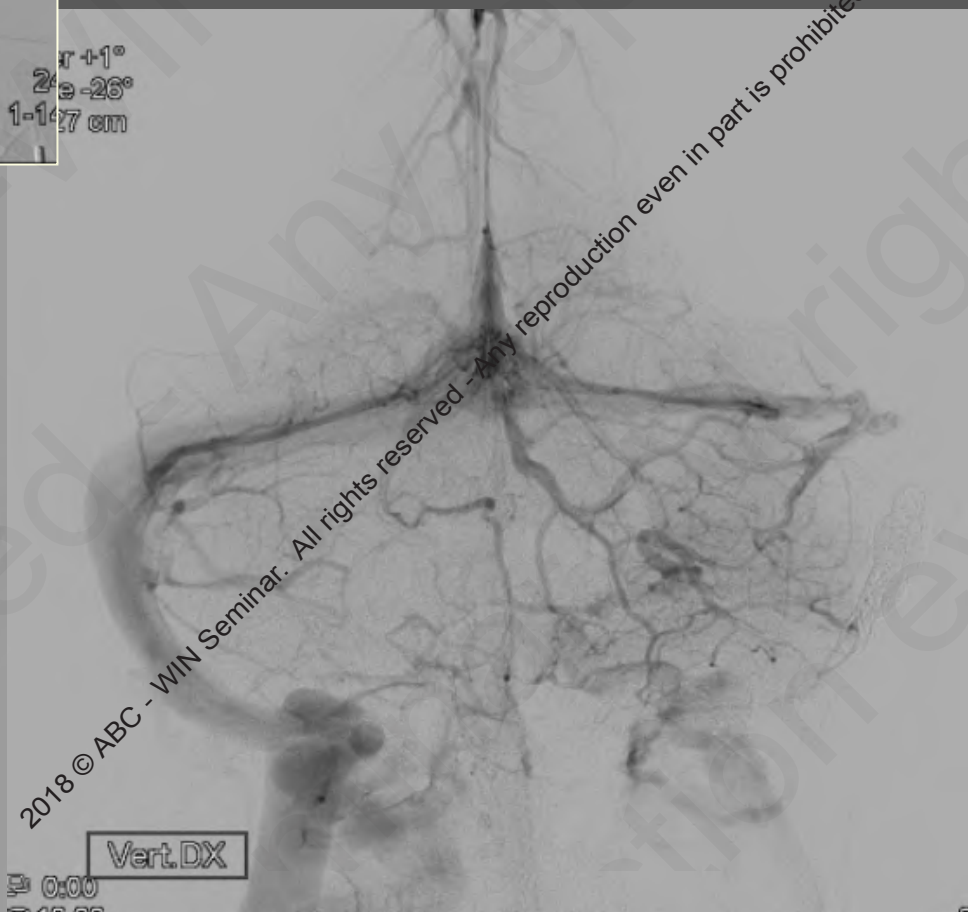


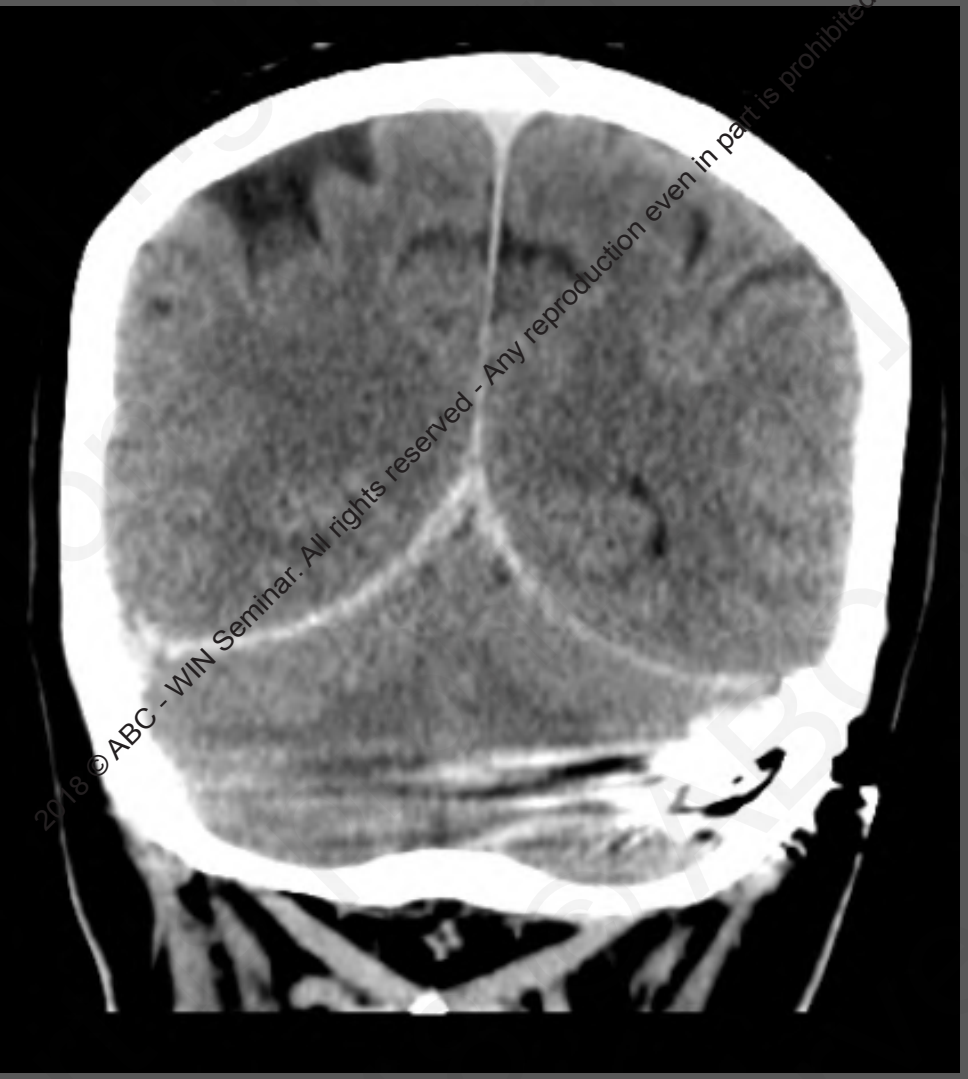


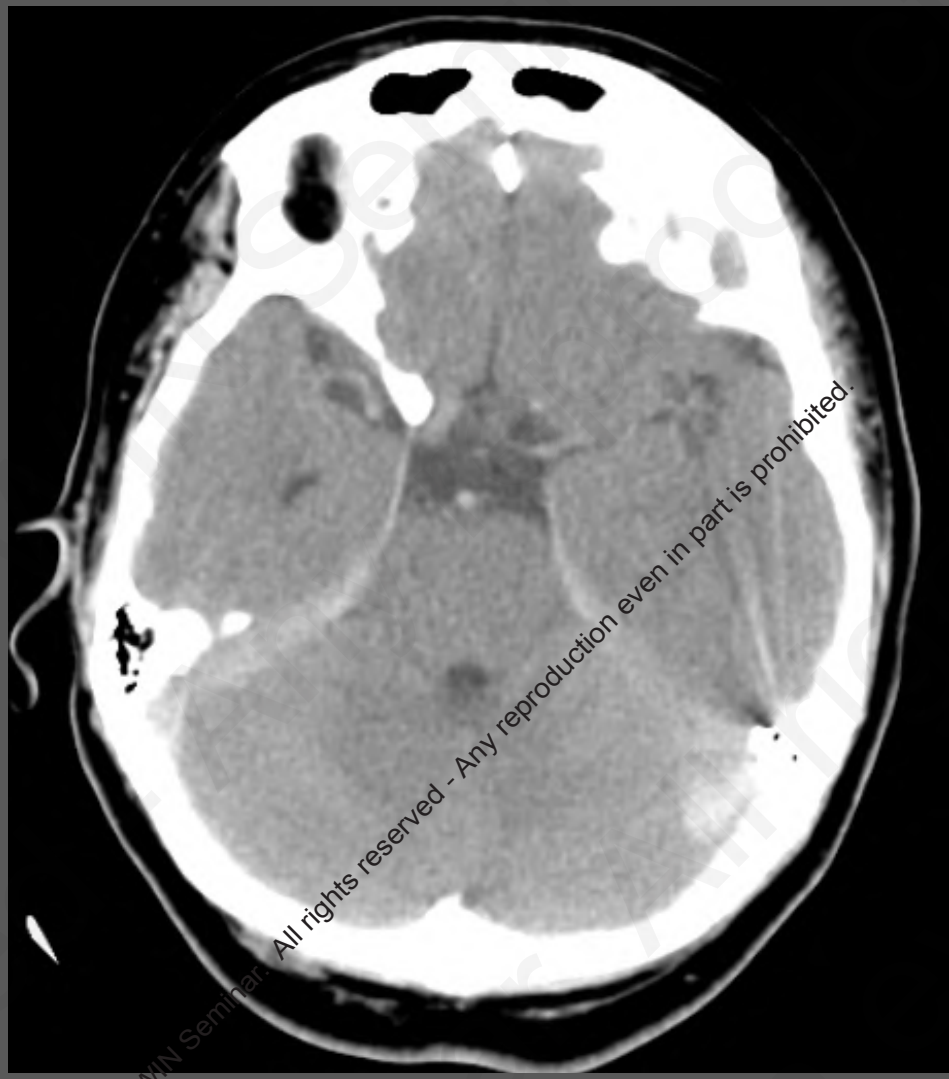
Post sinus venous occlusion

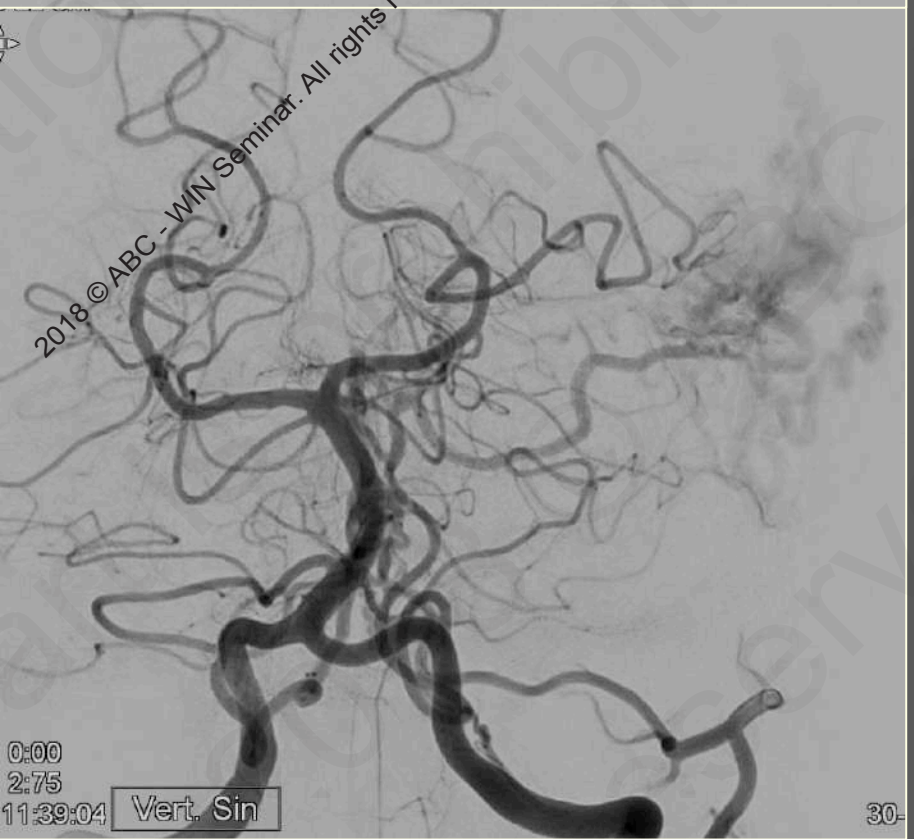


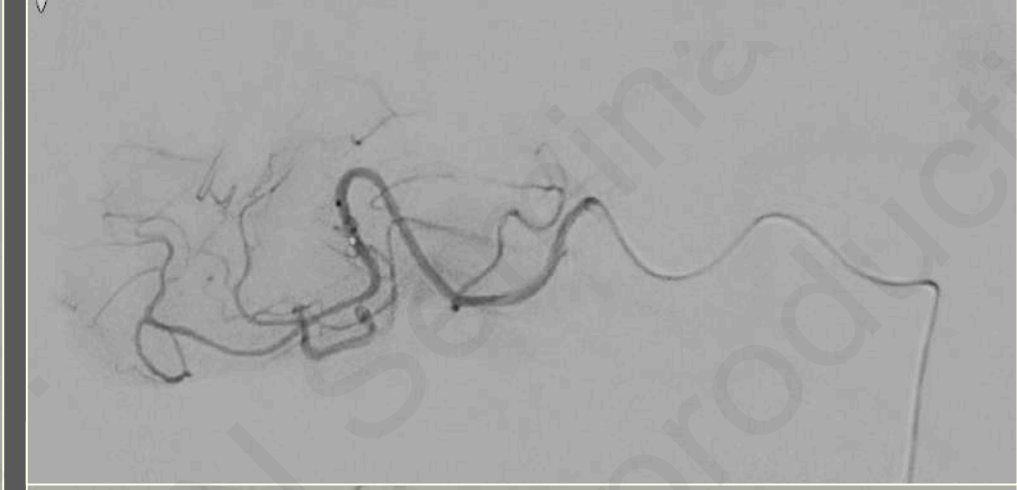


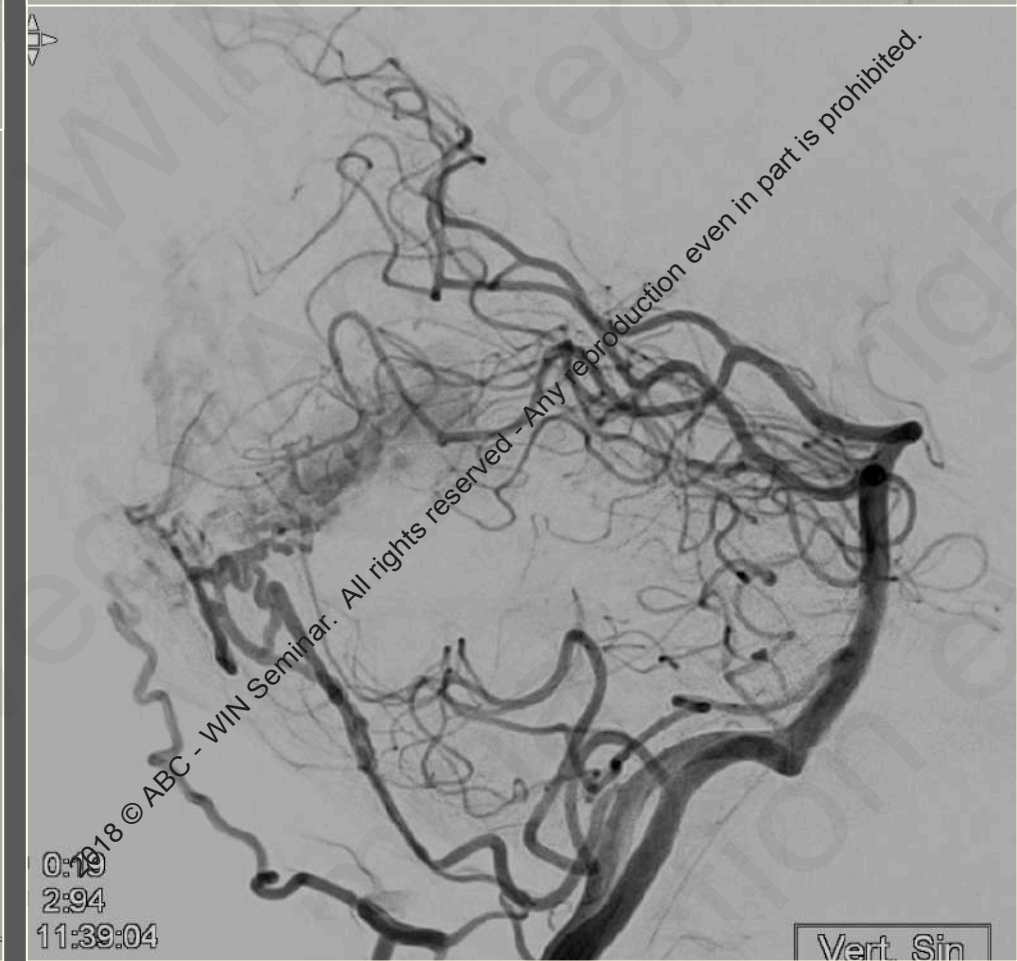


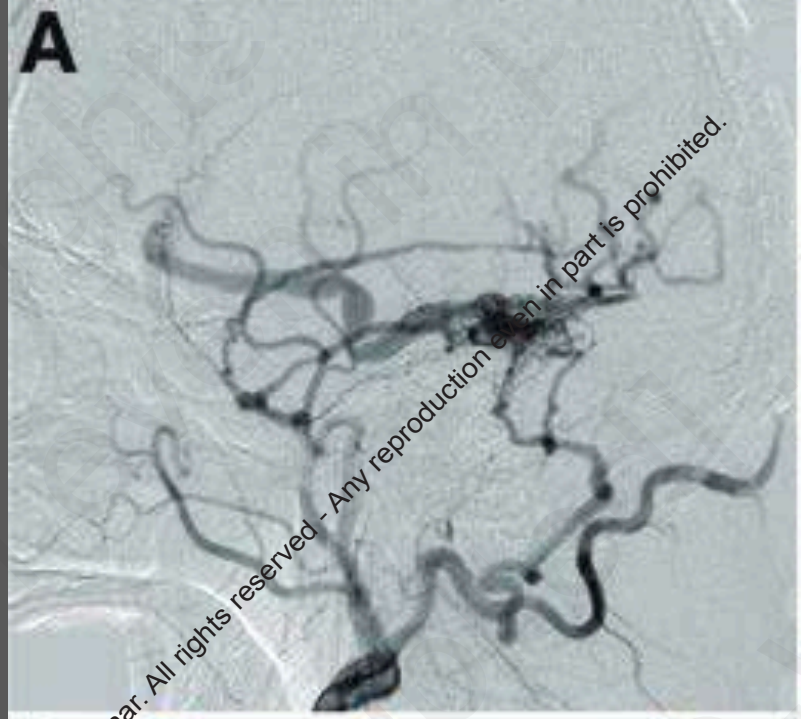


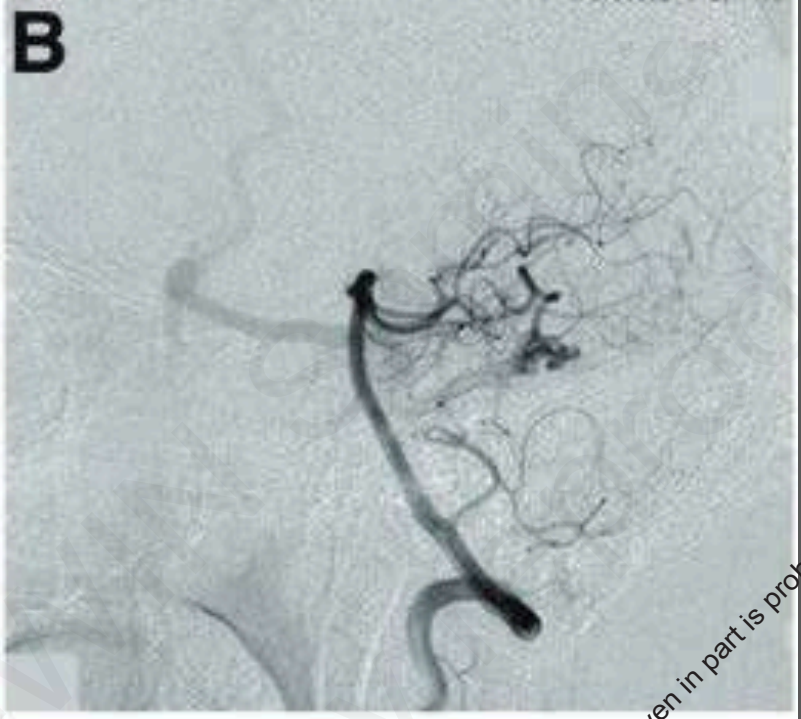


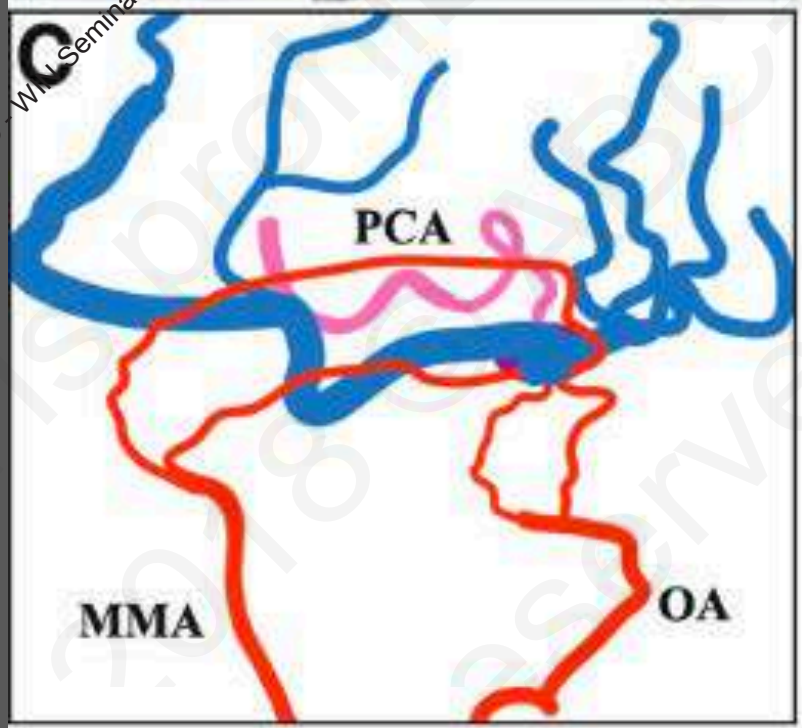




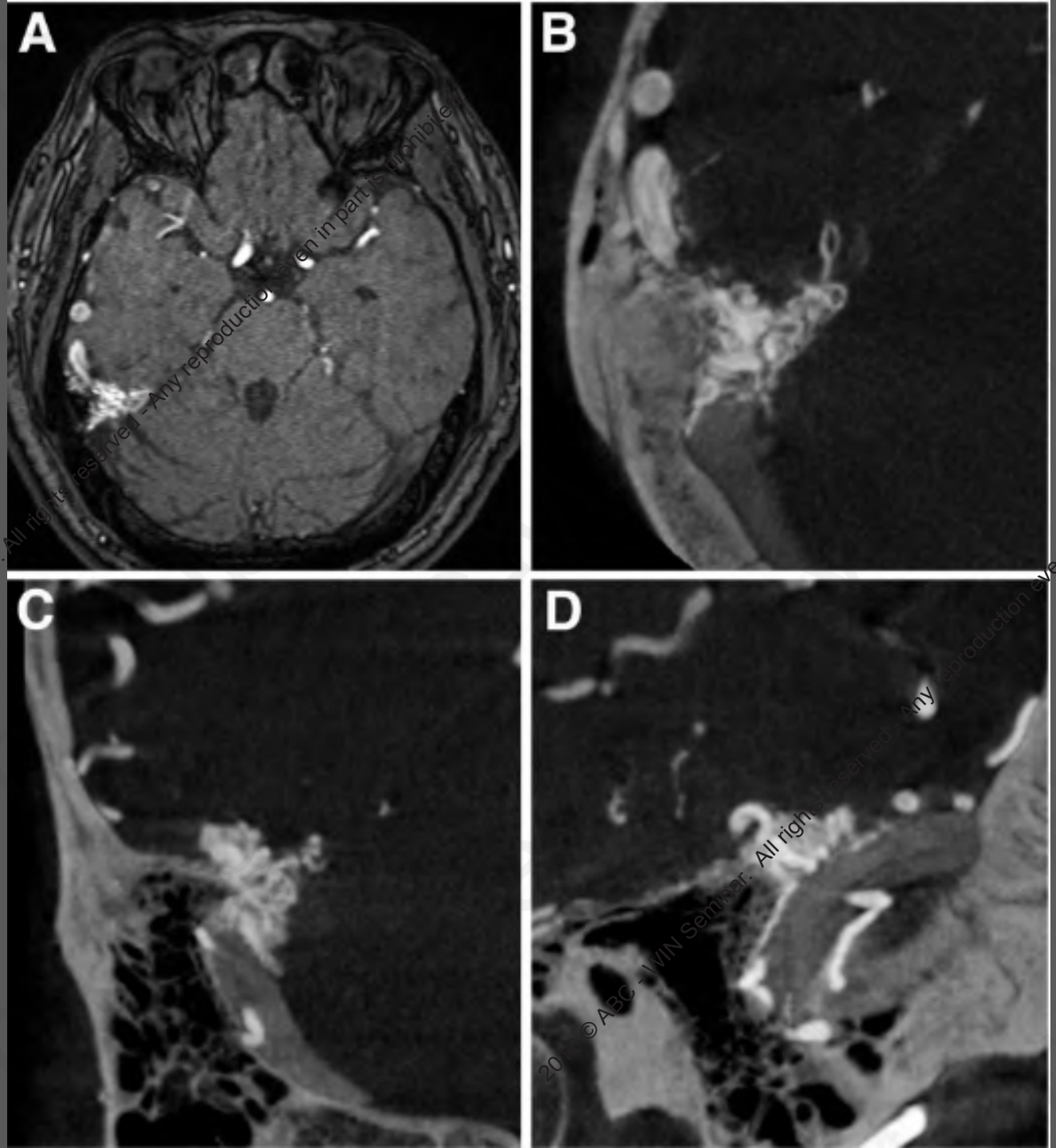


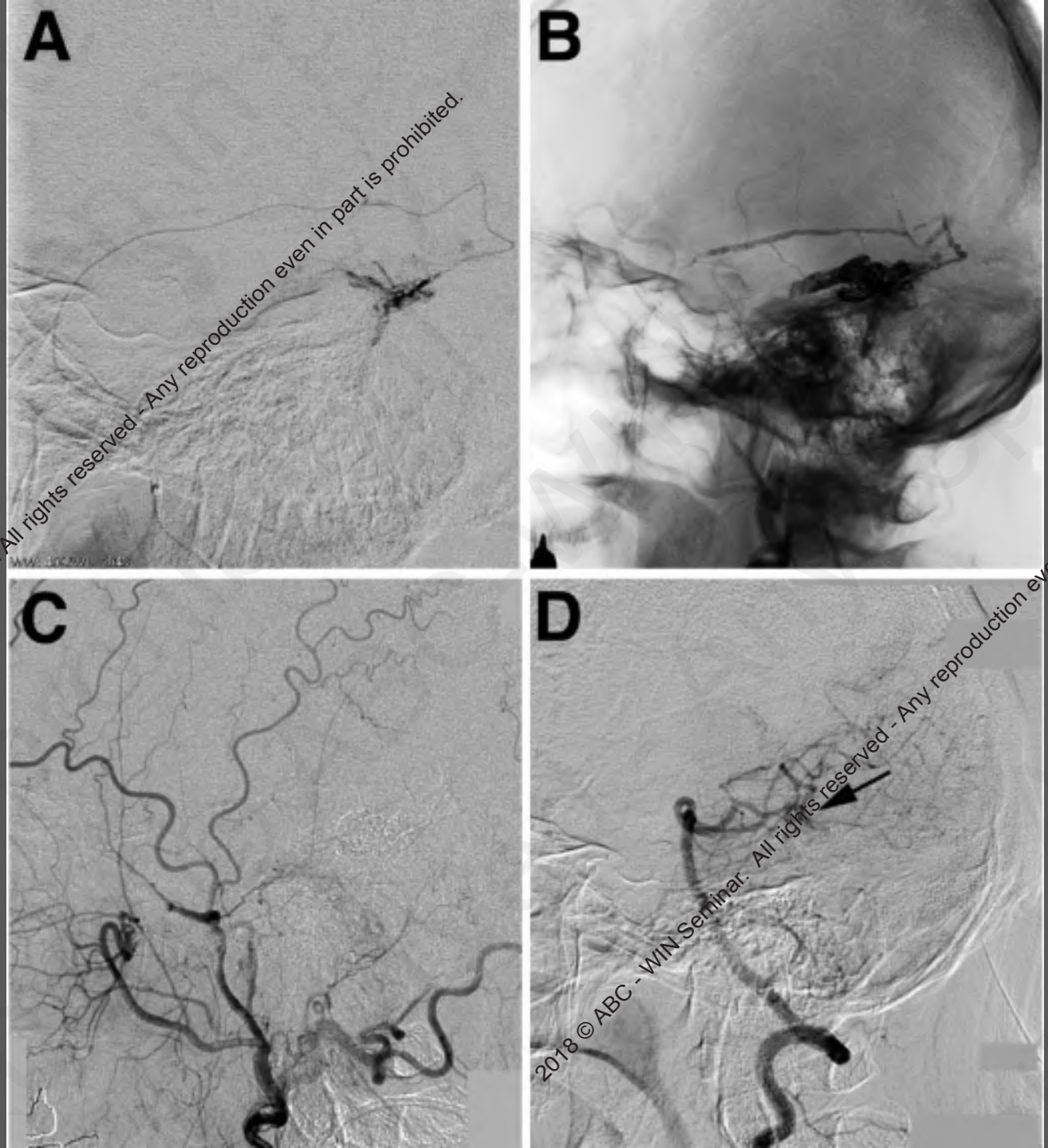


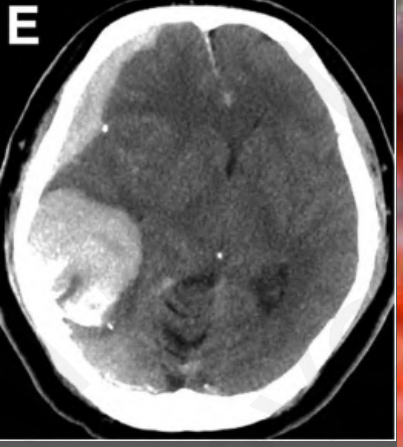


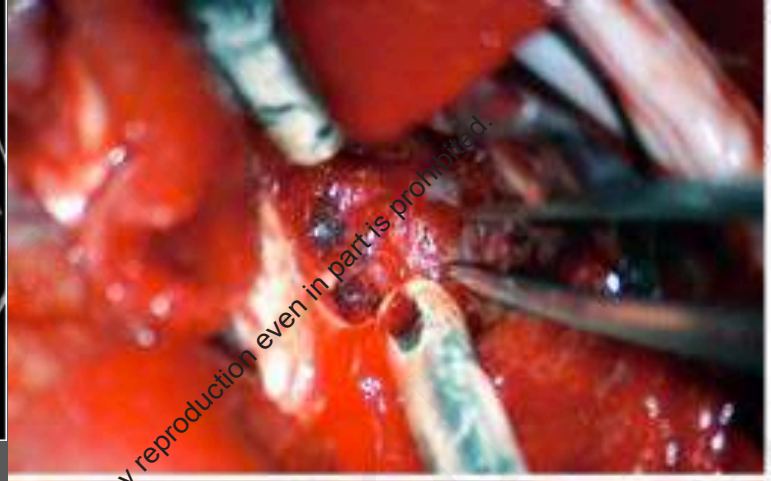


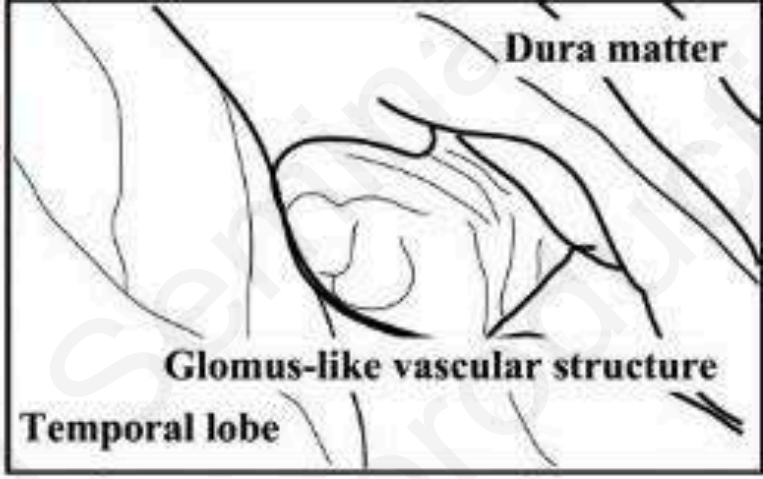
A hemorrhagic complication after Onyx embolization of a tentorial dural arteriovenous
fistula: A caution about subdural extension with pial arterial supply
Kenichi Sato et al, Interv Neurorad 2017

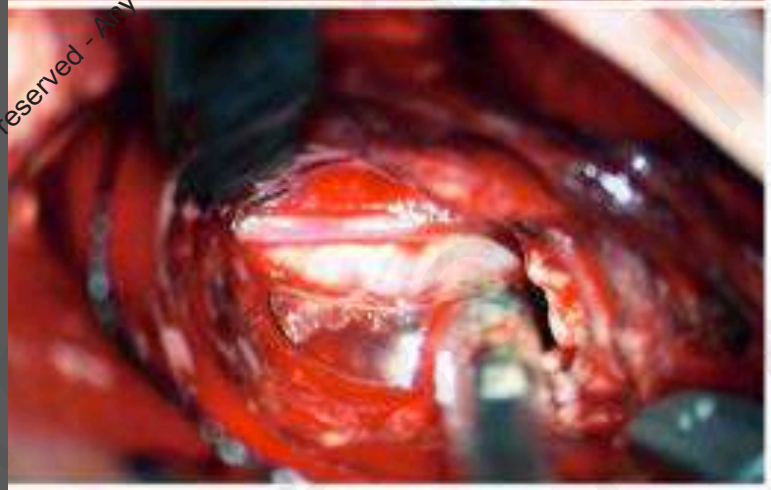


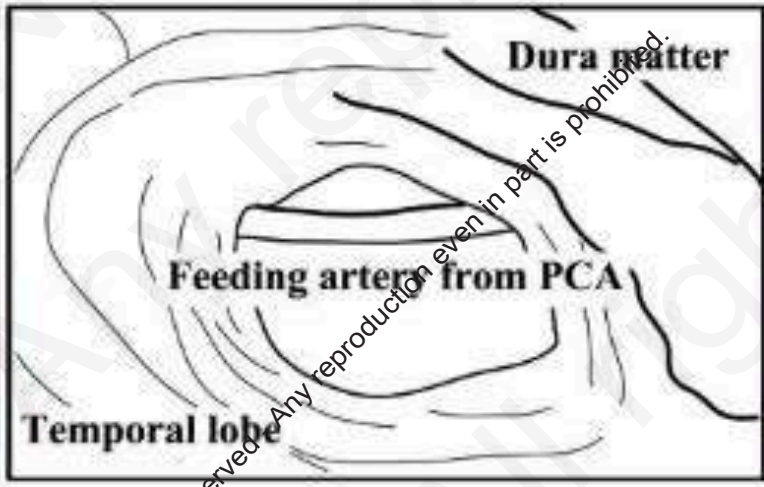


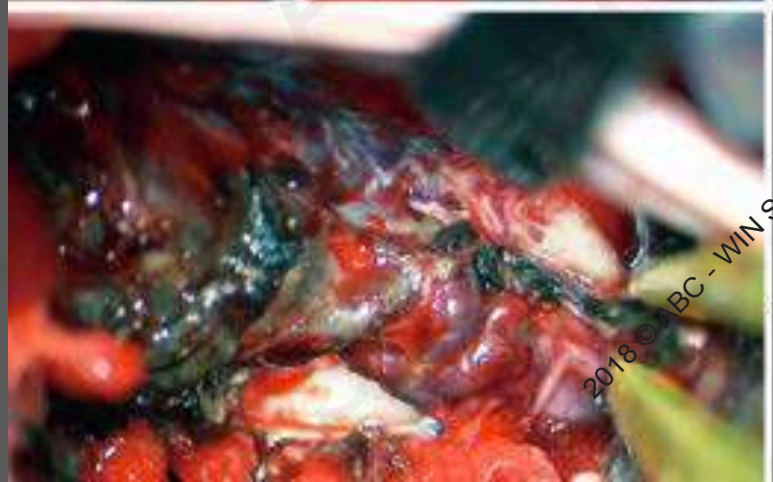


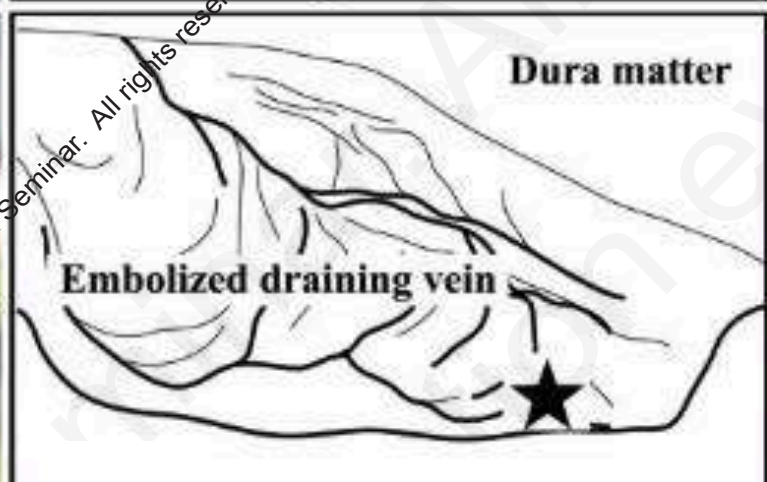












Male 61 y.o.

- TIA
- Incidental finding
- Asymptomatic
- No bruit
- For embolisation

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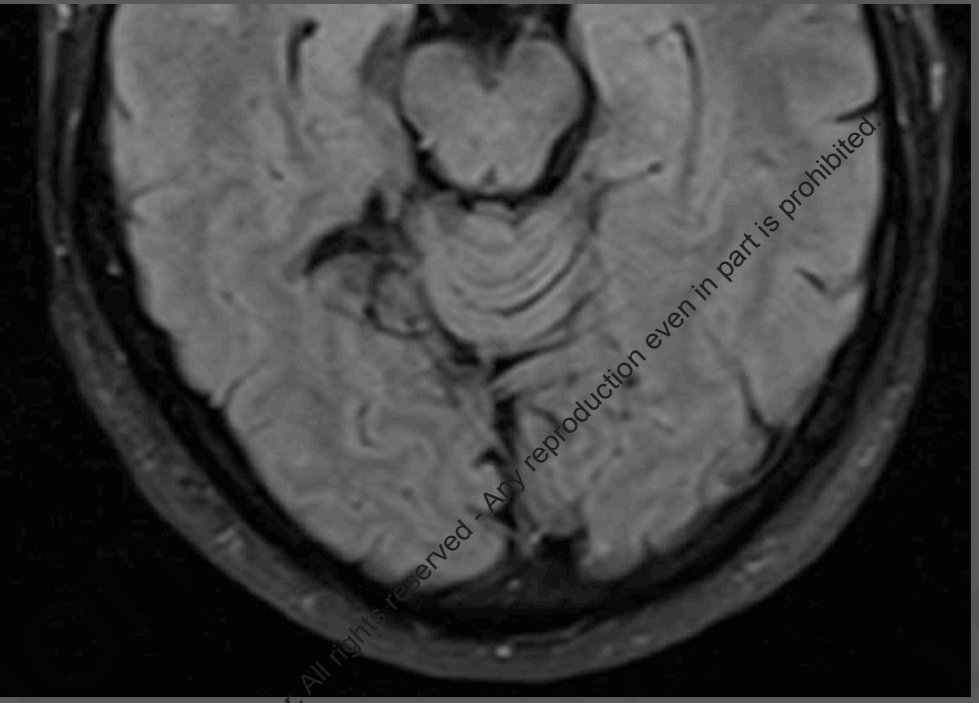
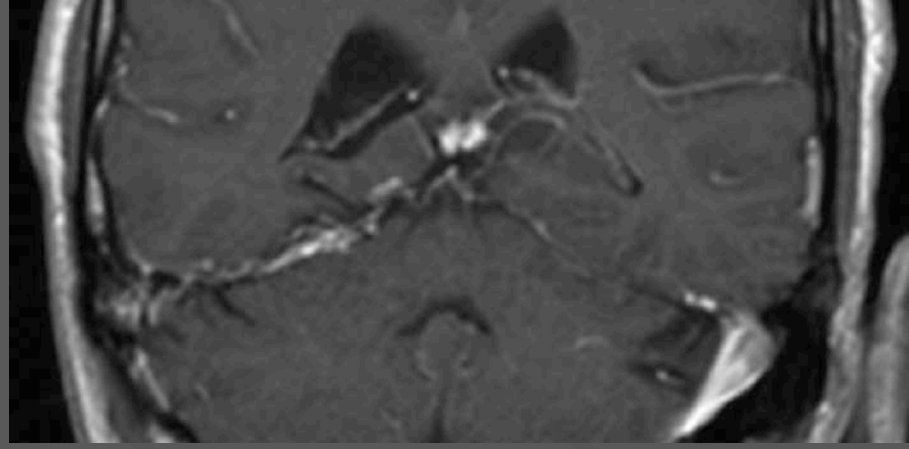
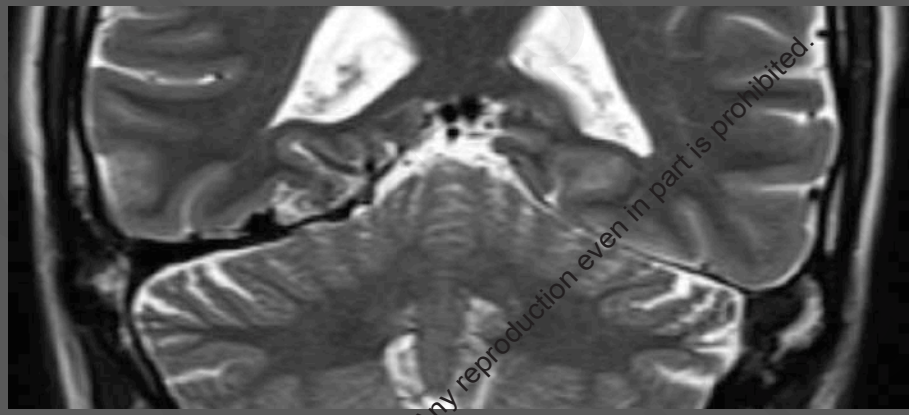
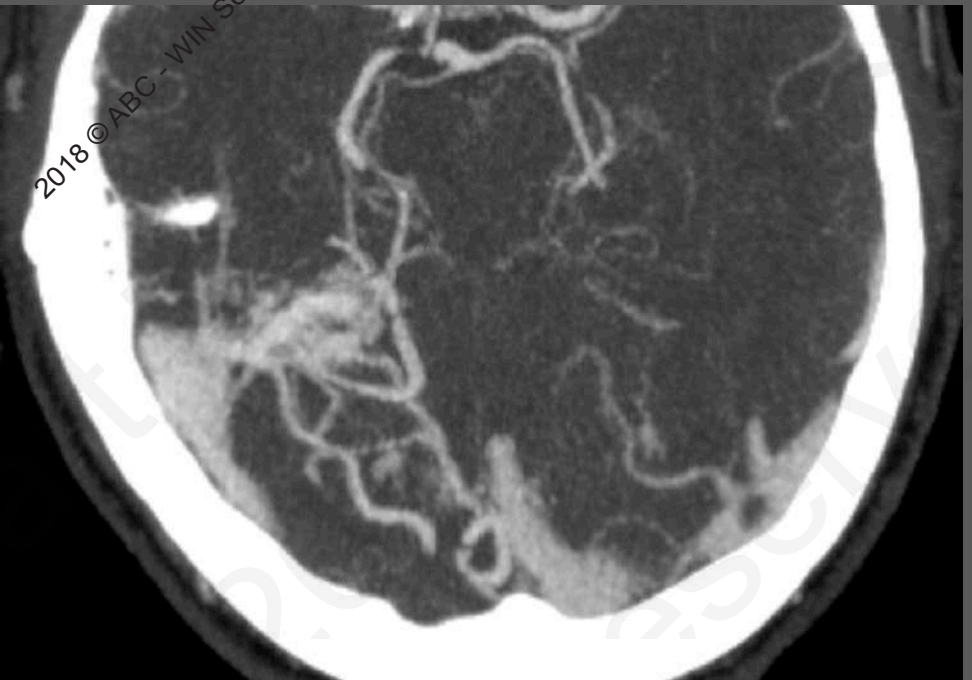
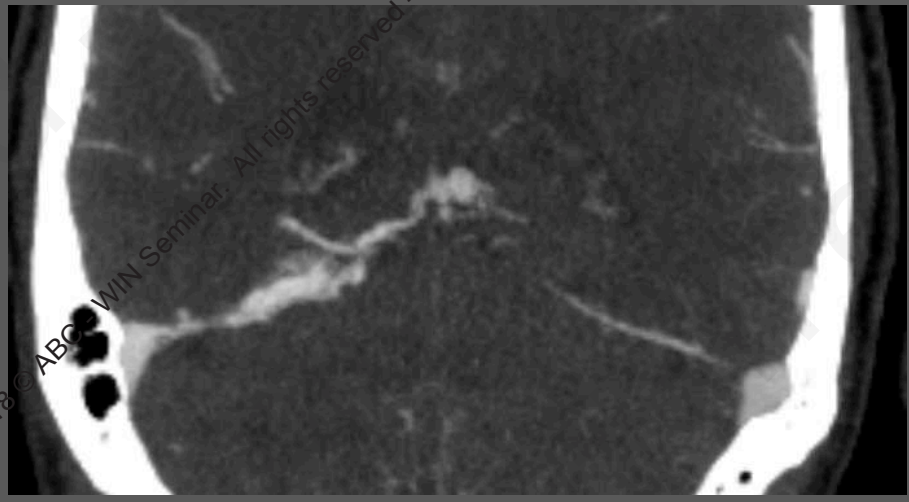
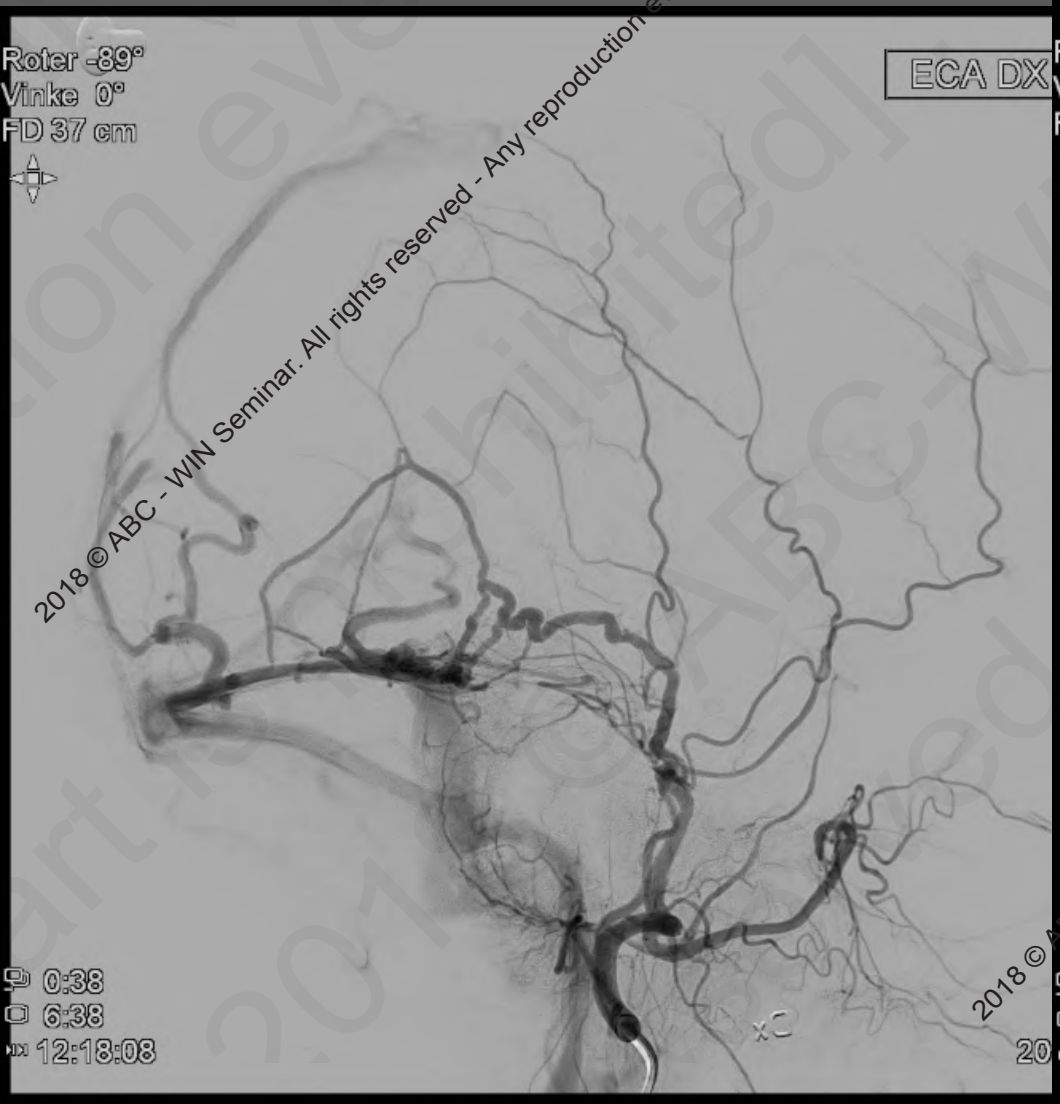


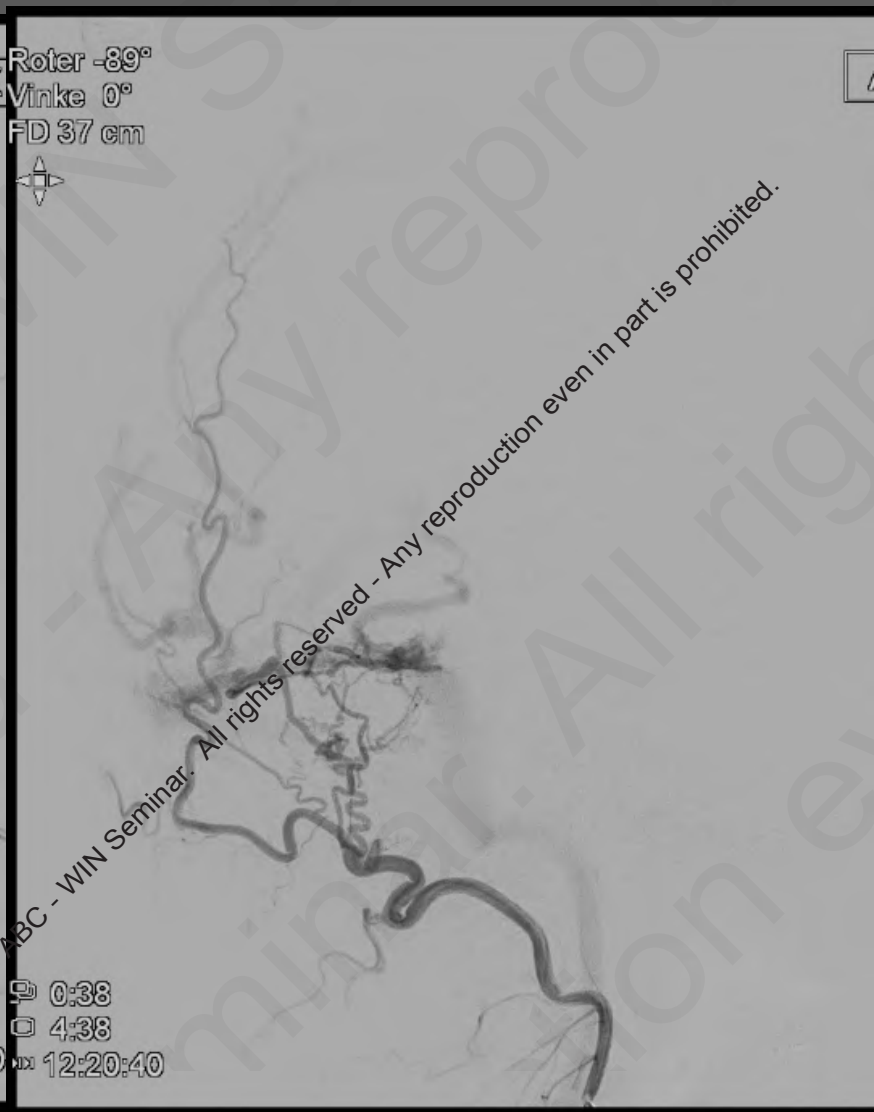


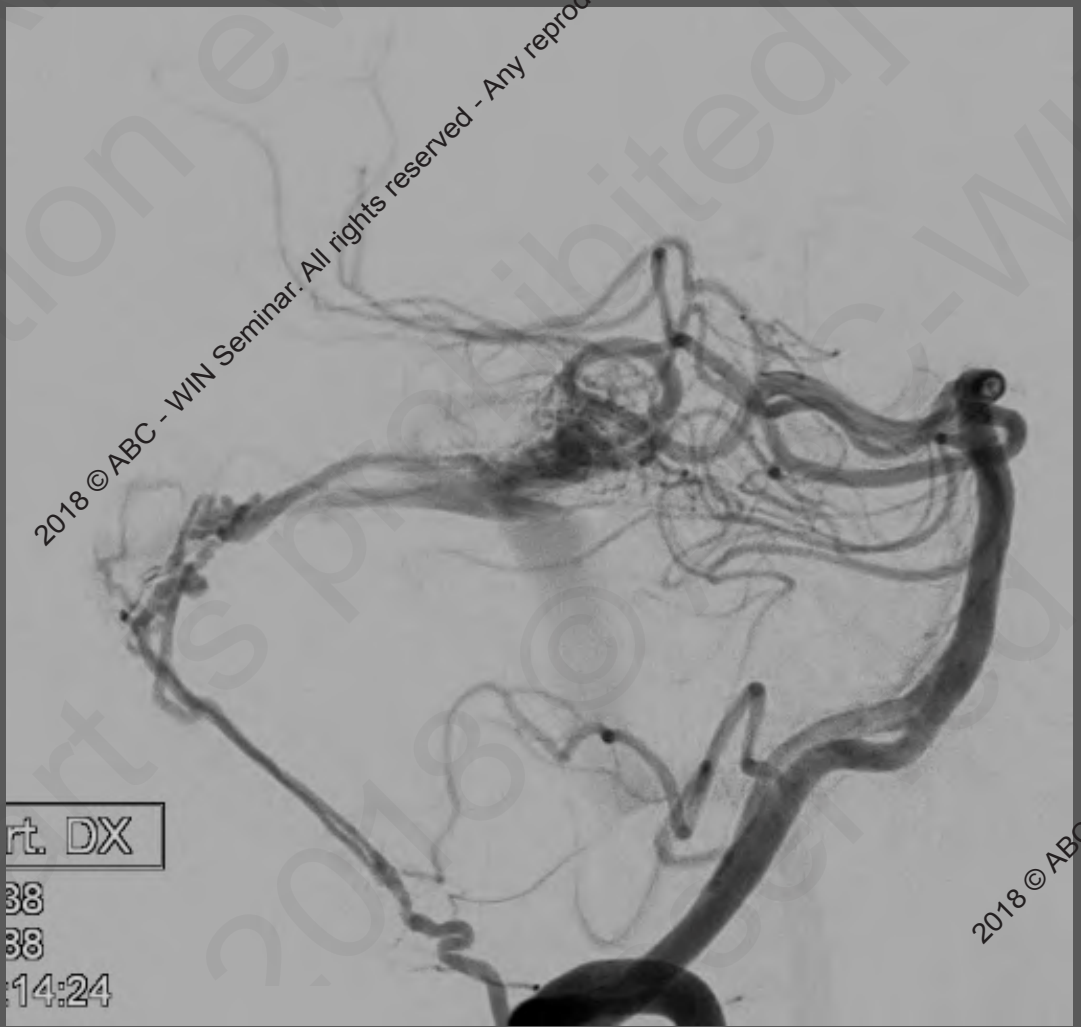


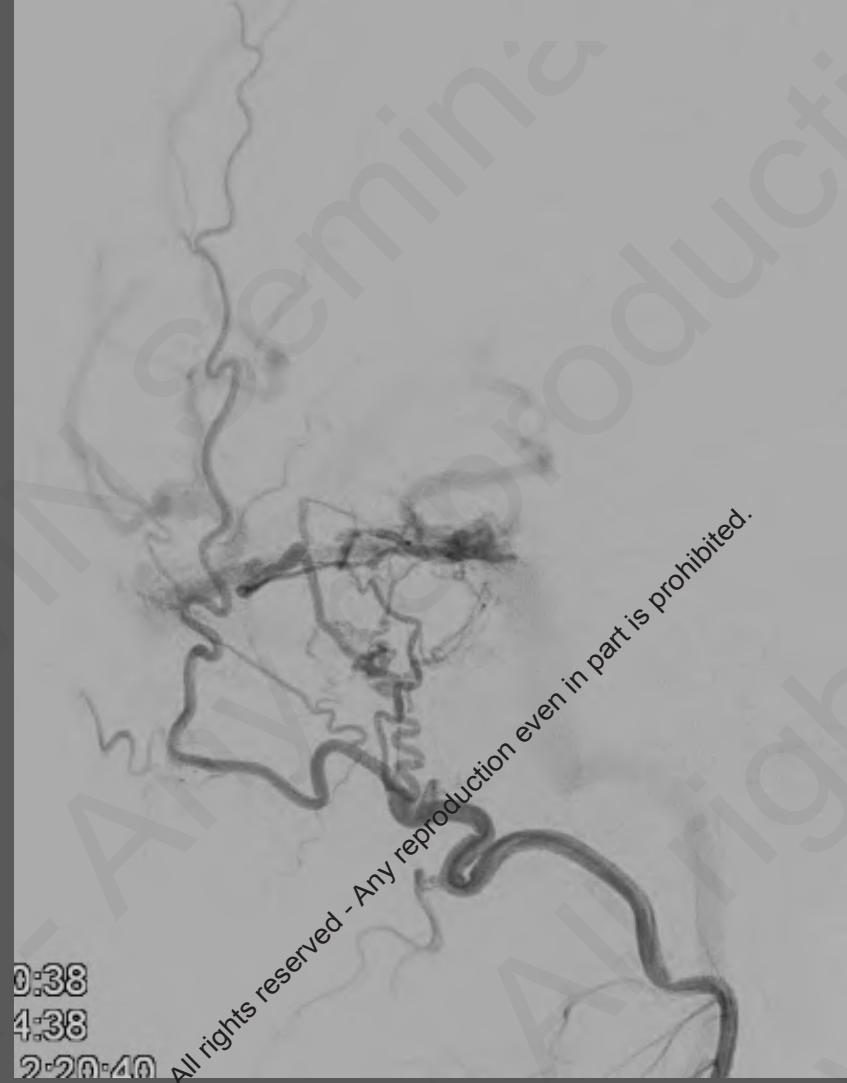


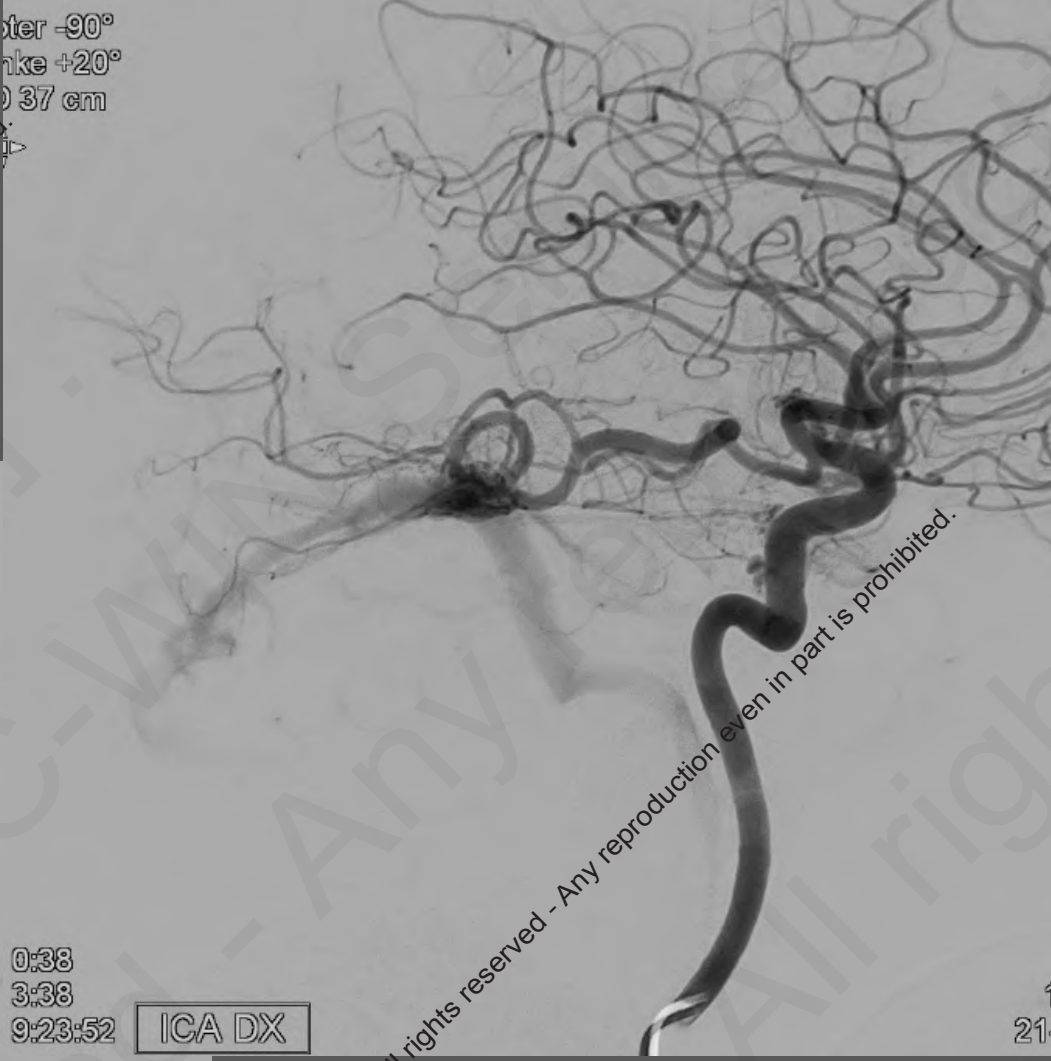


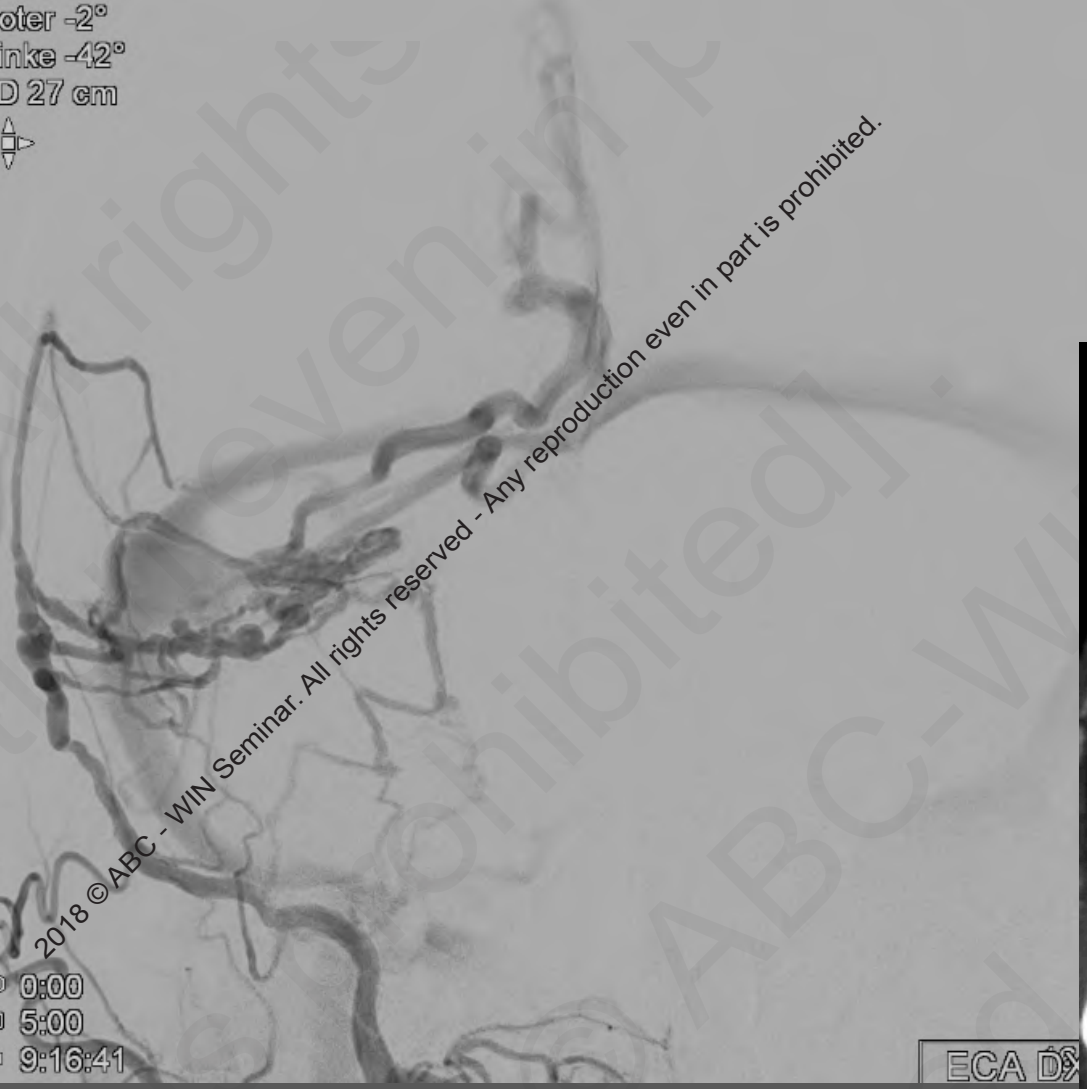


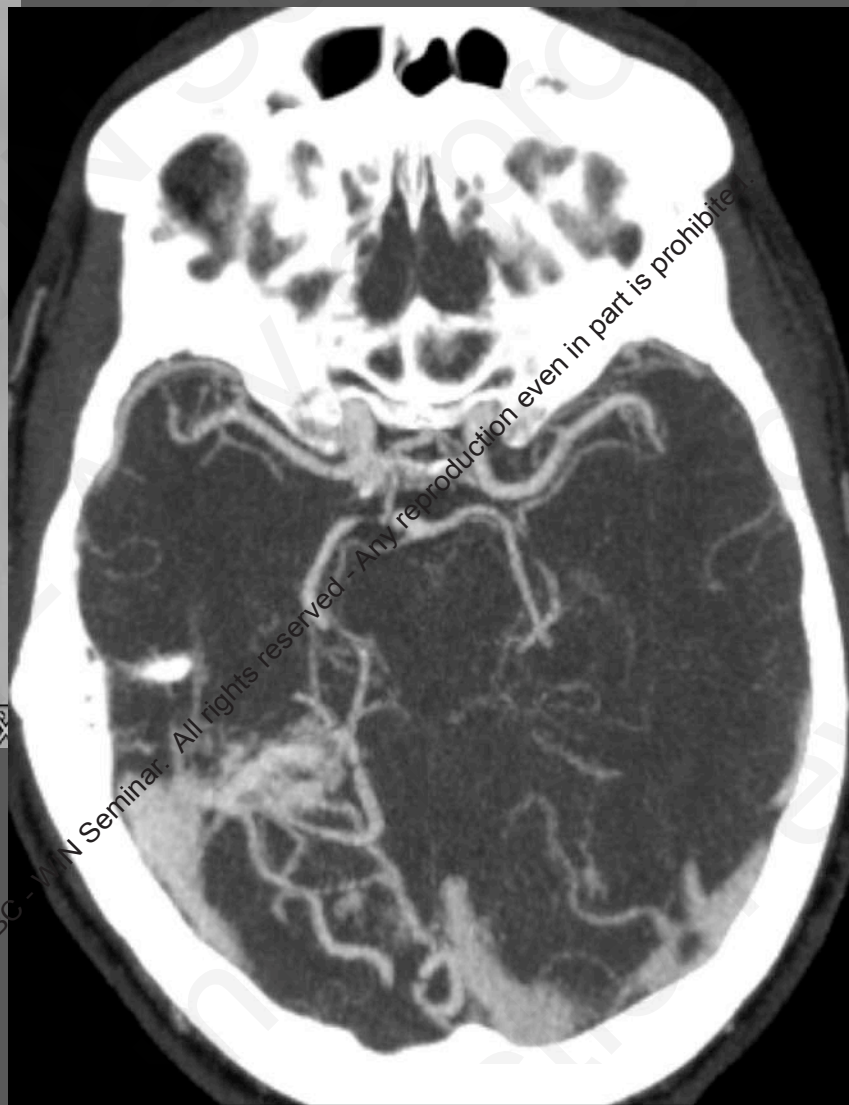


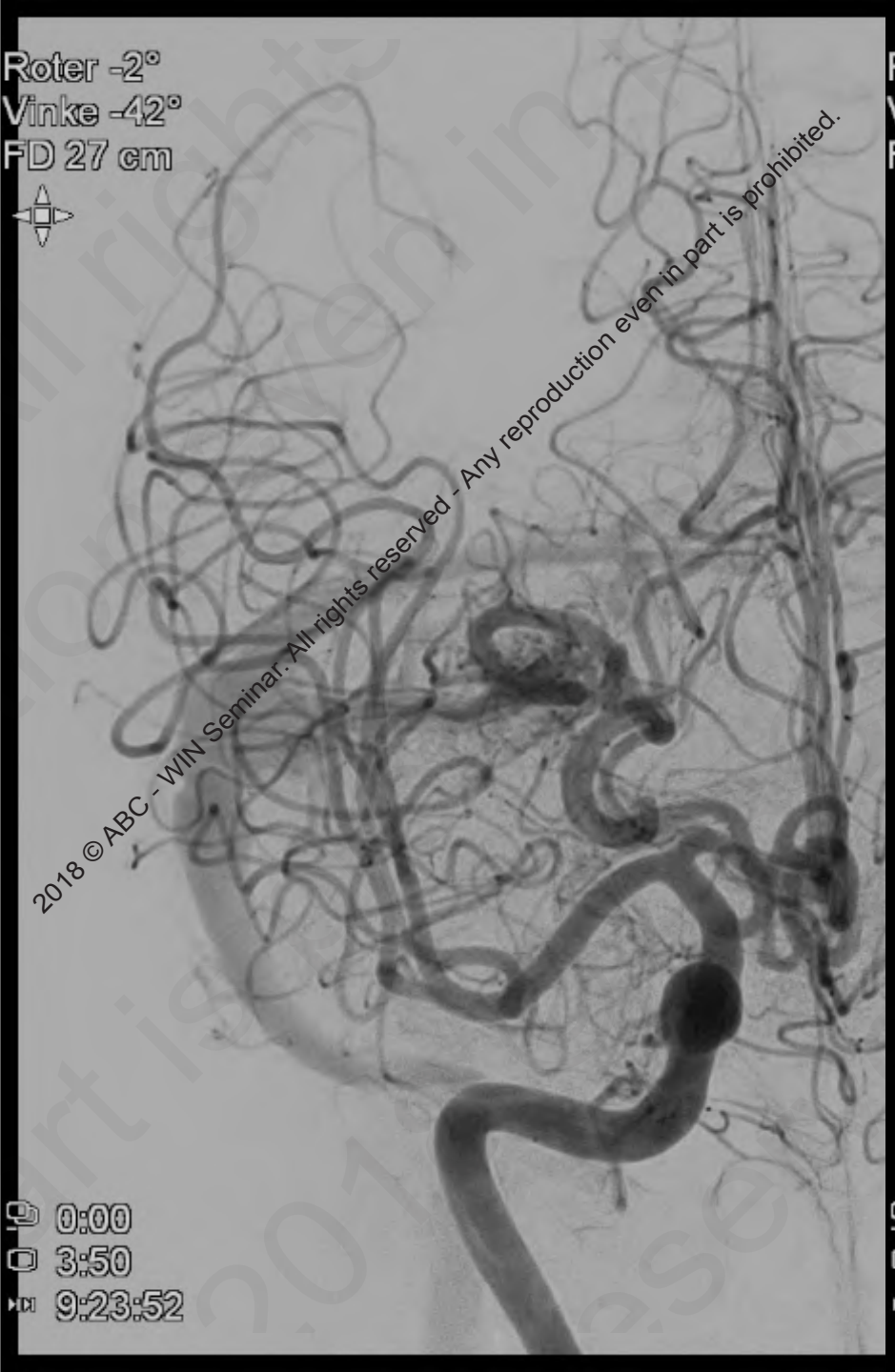


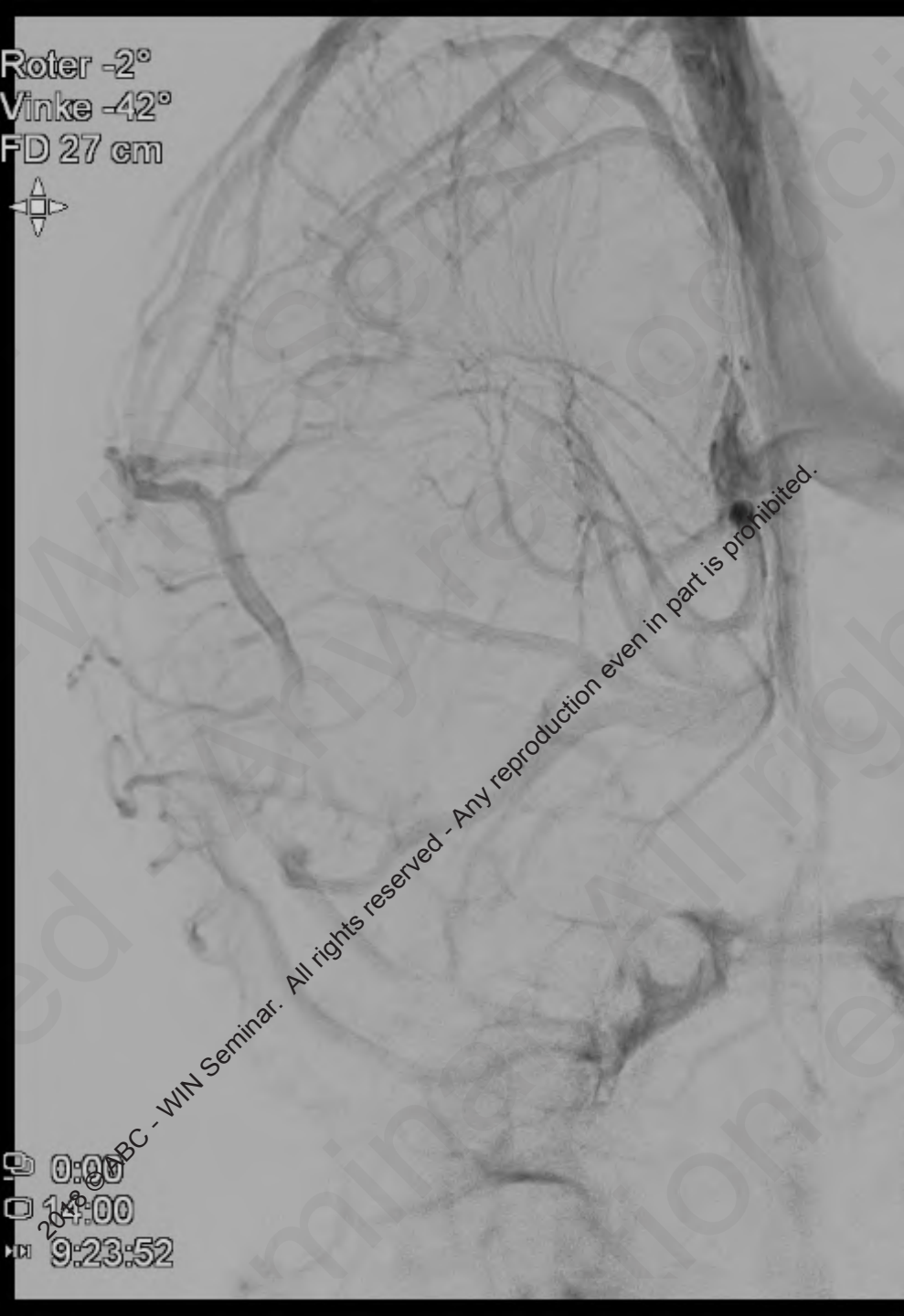


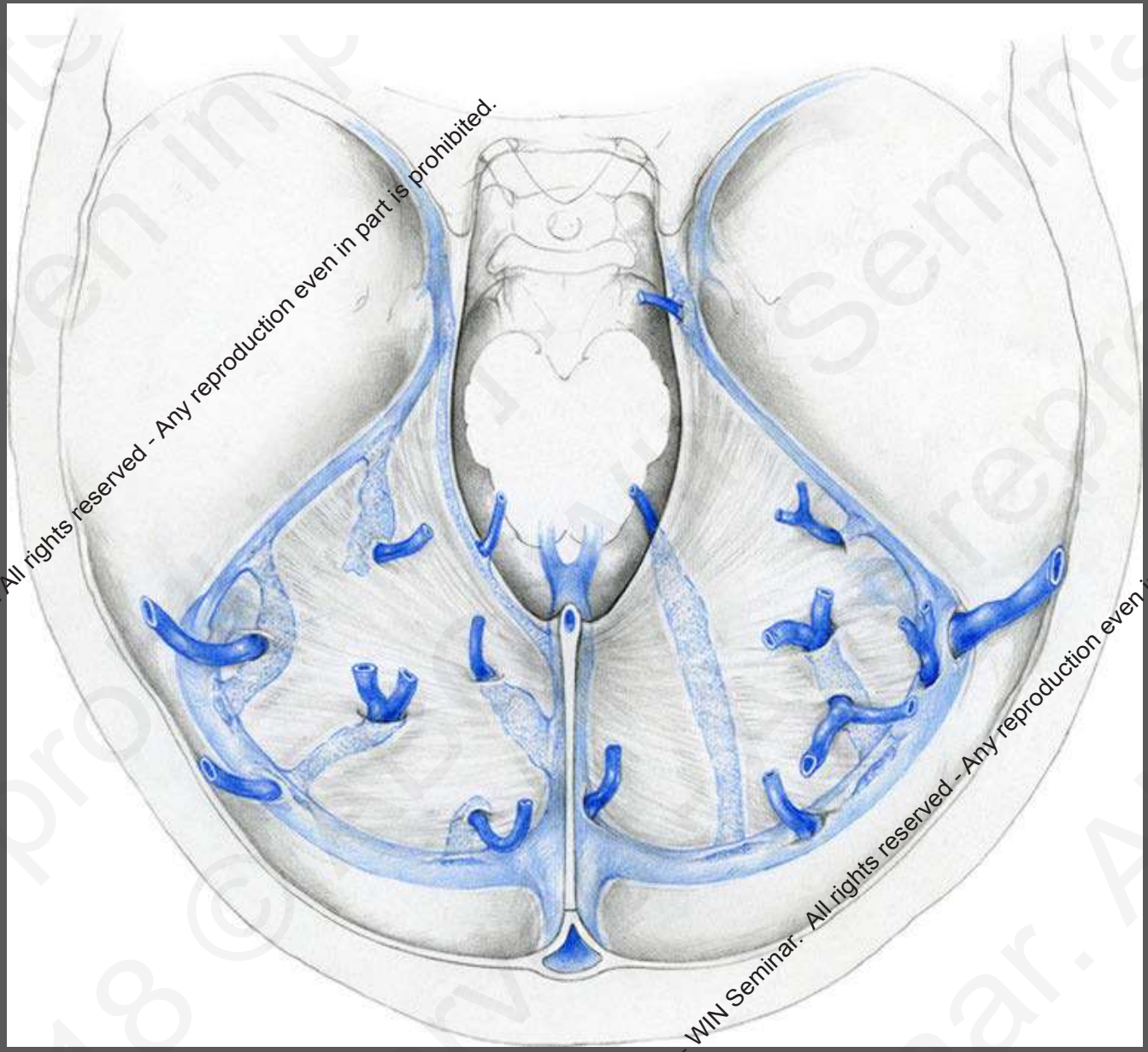




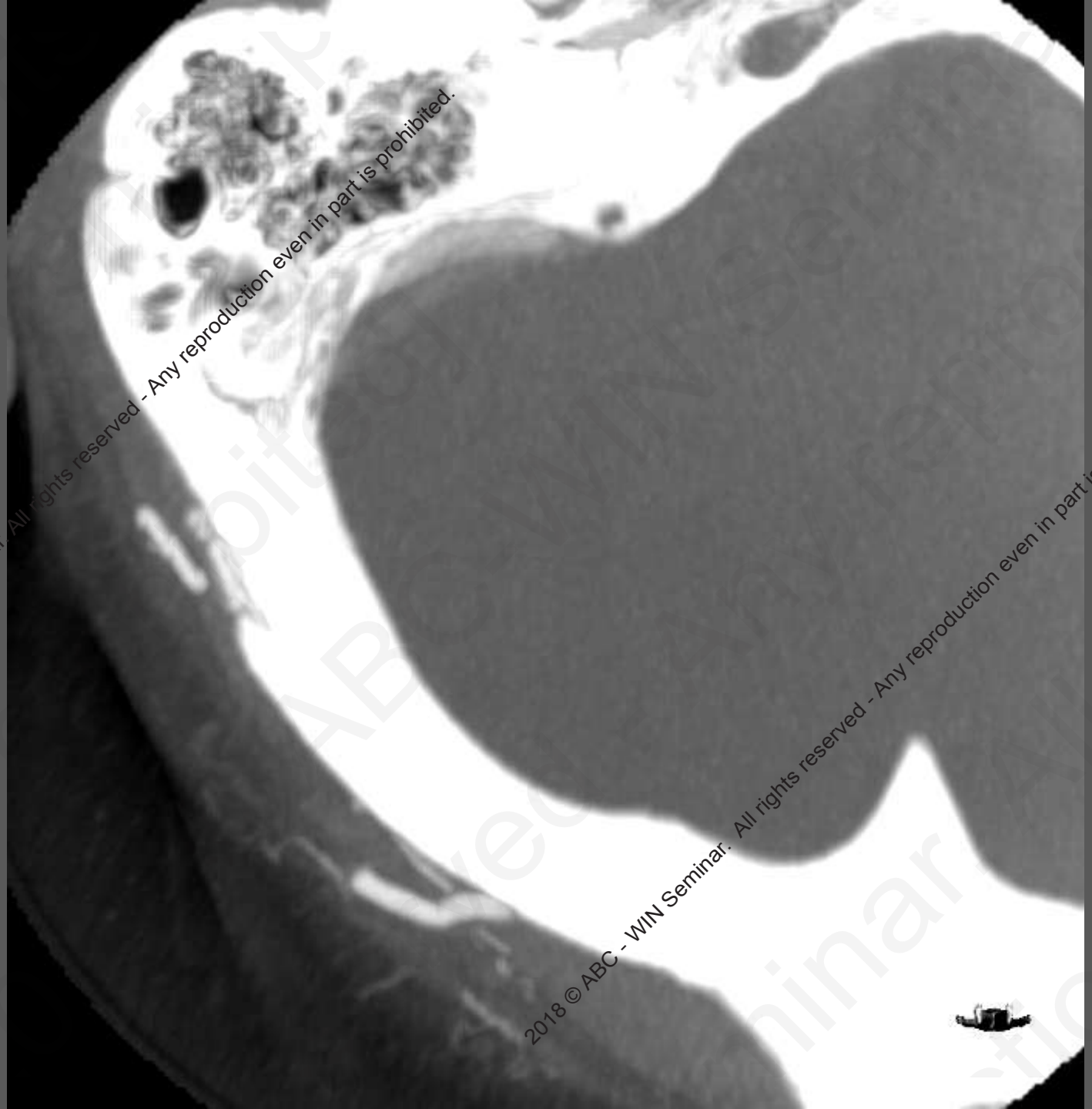


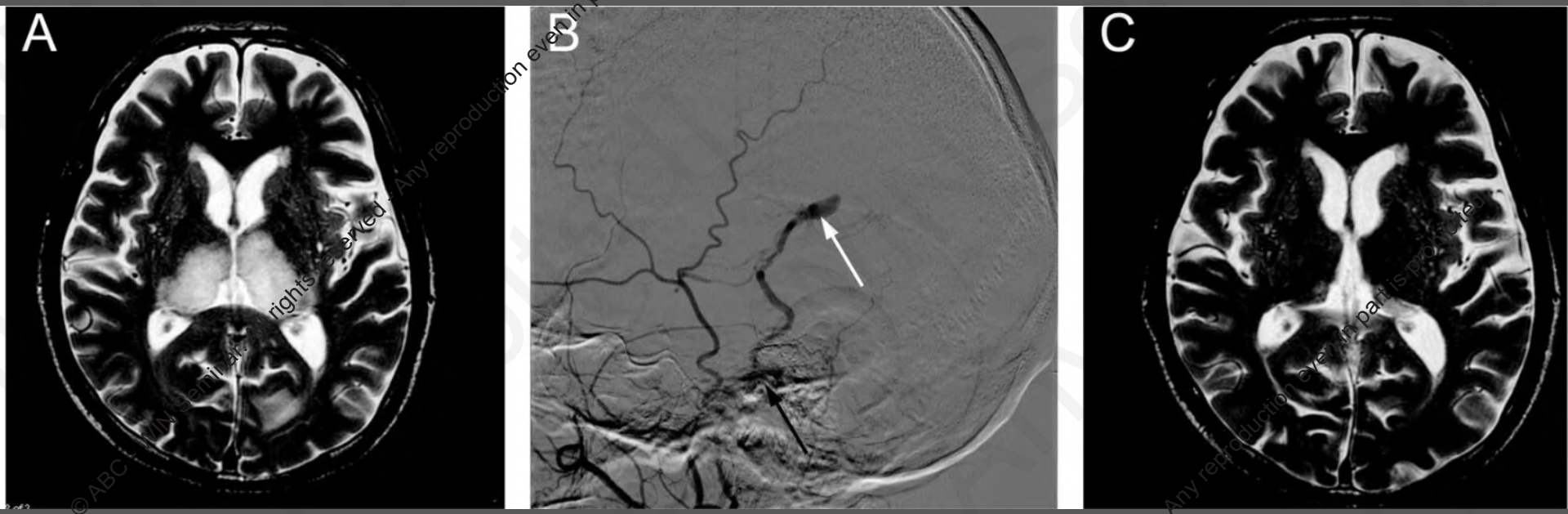






Cranial dural arteriovenous shunts, Gerasimos Baltsavias; Neurosurg Rev (2015)





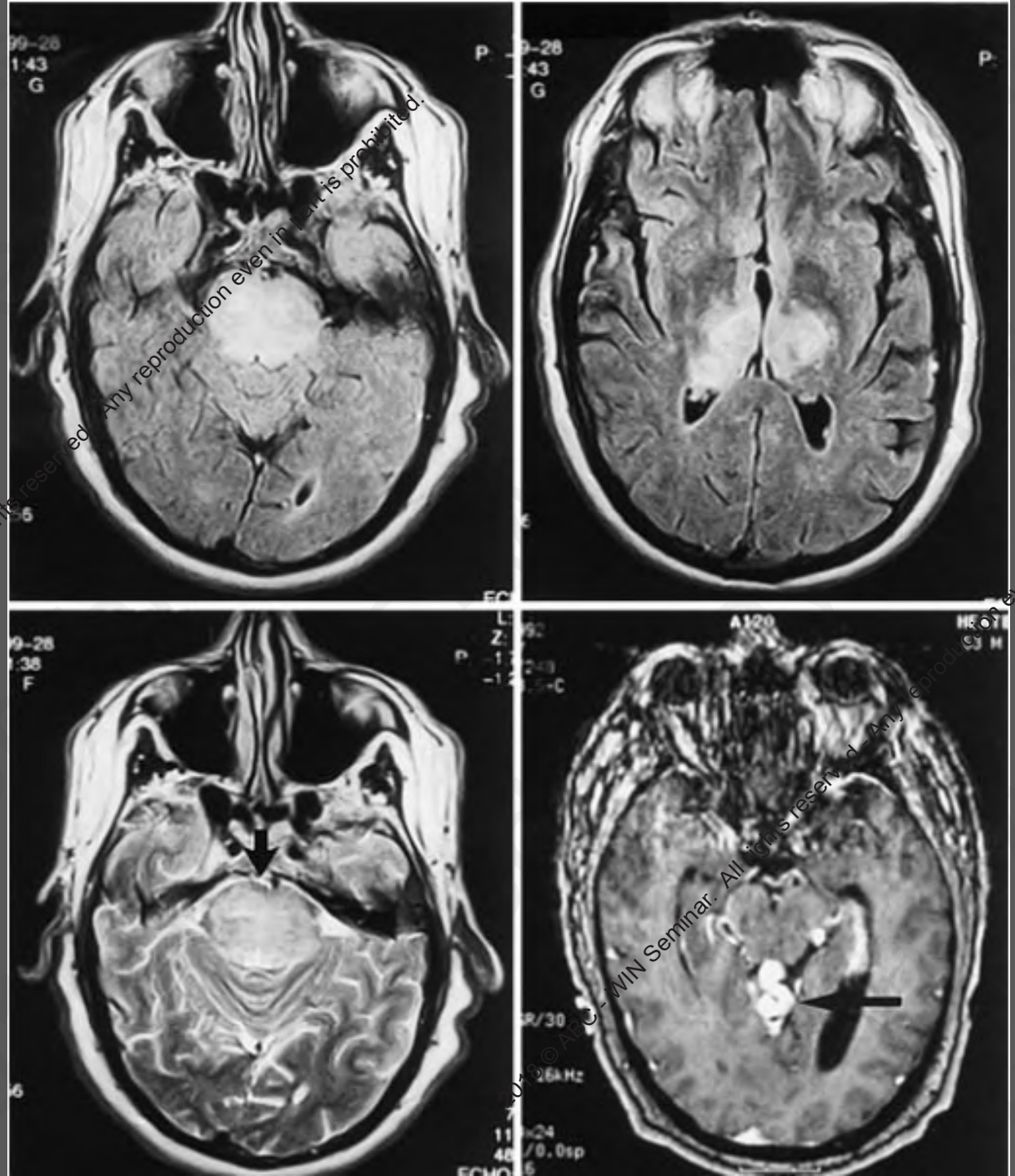
Dural arteriovenous fistula in a case of dementia with bithalamic MR lesions, A. Matsumura et al; Neuroimages

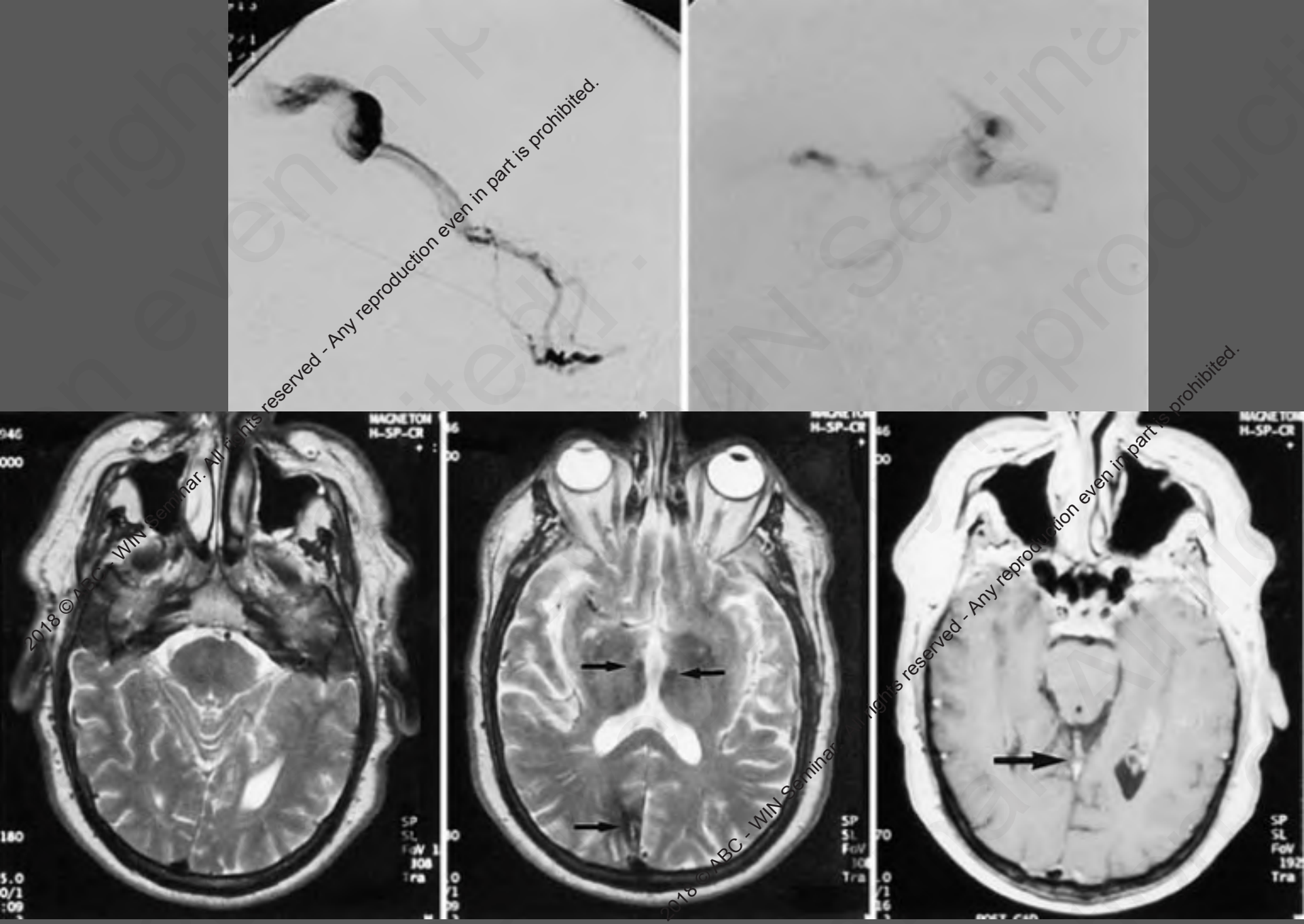
Case	Country	Age/sex	Underlying condition	Tremor	Rigidity	Brady-kinesia	Gait disturbance	Dementia	Time from onset to treatment	Hyperintense lesion on T2	The region of DAVF	Treatment	Outcome
1[14]	China	50/M	Hypertension diabetes	–	+	+	+	–	1 y	Bilateral frontal lobe	Superior sagittal sinus	TAE	Improved
2[15]	China	>60/M	NR	+	+	+	+	+	NR	NR	NR	L-dopa	Unimproved
3[15]	China	>60/M	Head trauma	–	+	+	+	+	NR	Bilateral cerebral	Left transverse sinus	L-dopa	Unimproved
4[16]	Japan	81/M	NR	–	+	–	+	+	6 mo	Bilateral cerebral white matter	Right transverse sinus	TAE	Improved
5[16]	Japan	55/M	–	–	–	+	+	+	8 mo	Bilateral deep and subcortical white	Right sigmoid sinus	TAE	Improved
6[16]	Japan	78/M	NR	–	+	+	+	+	9 mo	NL	Right sigmoid sinus	TAE	Improved
7[12]	Japan	69/F	–	–	+	+	–	+	Several years	Bilateral cerebral white matter	Left sigmoid sinus	TAE	Unimproved
8[17]	Japan	44/M	NR	–	–	+	+	–	1 wk	NL	Anterior cranial fossa	TAE	Improved
9[10]	Europe	60/F	–	Bilateral action tremor	+	+	+	+	18 mo	Bilateral subcortical white matter	Superior sagittal sinus	TAE	Improved
10[6]	Japan	75/M	–	Bilateral postural tremor	+	+	+	+	14 wk	NL	Superior sagittal sinus and right transverse-sigmoid sinus	TAE	Improved
11[18]	Japan	65/M	NR	–	+	+	+	+	11 mo	Basal ganglia and deep white matter	Left transverse- sigmoid sinus	TAE	Improved
12[19]	US	79/M	Tongue- base cancer	Bilateral postural tremor	+	+	+	–	2 y	NL	Left transverse sinus	TAE	Normalized
13[20]	India	54/M	Diffuse headache	Bilateral postural tremor	+	+	+	+	3 y	Bilateral thalamus and globus pallidus	Bilateral transverse sinuses and straight sinus	TAE	Only minimal change
14[20]	India	40/M	NR	–	+	–	–	+	3 mo	NE	Superior sagittal sinus	TAE	Unchanged
15[21]	Japan	52/F	NR	–	–		+	+	3 mo	Basal ganglia and deep white matter	Right transverse- sigmoid sinus	TAE	Improved
16[12]	France	69/M	Hypertension and diabetes	Right hand postural tremor	+	+	+	+	2 y	NL	Superior sagittal sinus	TAE	Improved
17[22]	Canada	62/M	On warfarin for a remote history	Bilateral rest, movement, posture tremor	+	+	+	+	2 mo	Bilateral cerebral hemispheric white matter	Superior sagittal sinus, both transverse sinuses, torcula, and right sigmoid sinus	TAE	Improved
18[22]	Canada	65/F	NR	Bilateral rest, movement, posture tremor	+	+	+	+	5 y	Bilateral cerebral hemispheric white matter	Transverse-sigmoid	TAE	Improved
19 (present case)	China	51/M	Hypertension hyperlipemia	–	–	+	–	+	1 mo	Bilateral lenticular nuclei	Straight sinus	TAE	Improved

F = female, M = male, NE = not examined, NL = normal, NR = not report, TAE = transarterial embolization therapy.

Straight sinus dural arteriovenous fistula presenting with reversible parkinsonism: A case report and literature review
Pu, Jiali; Medicine

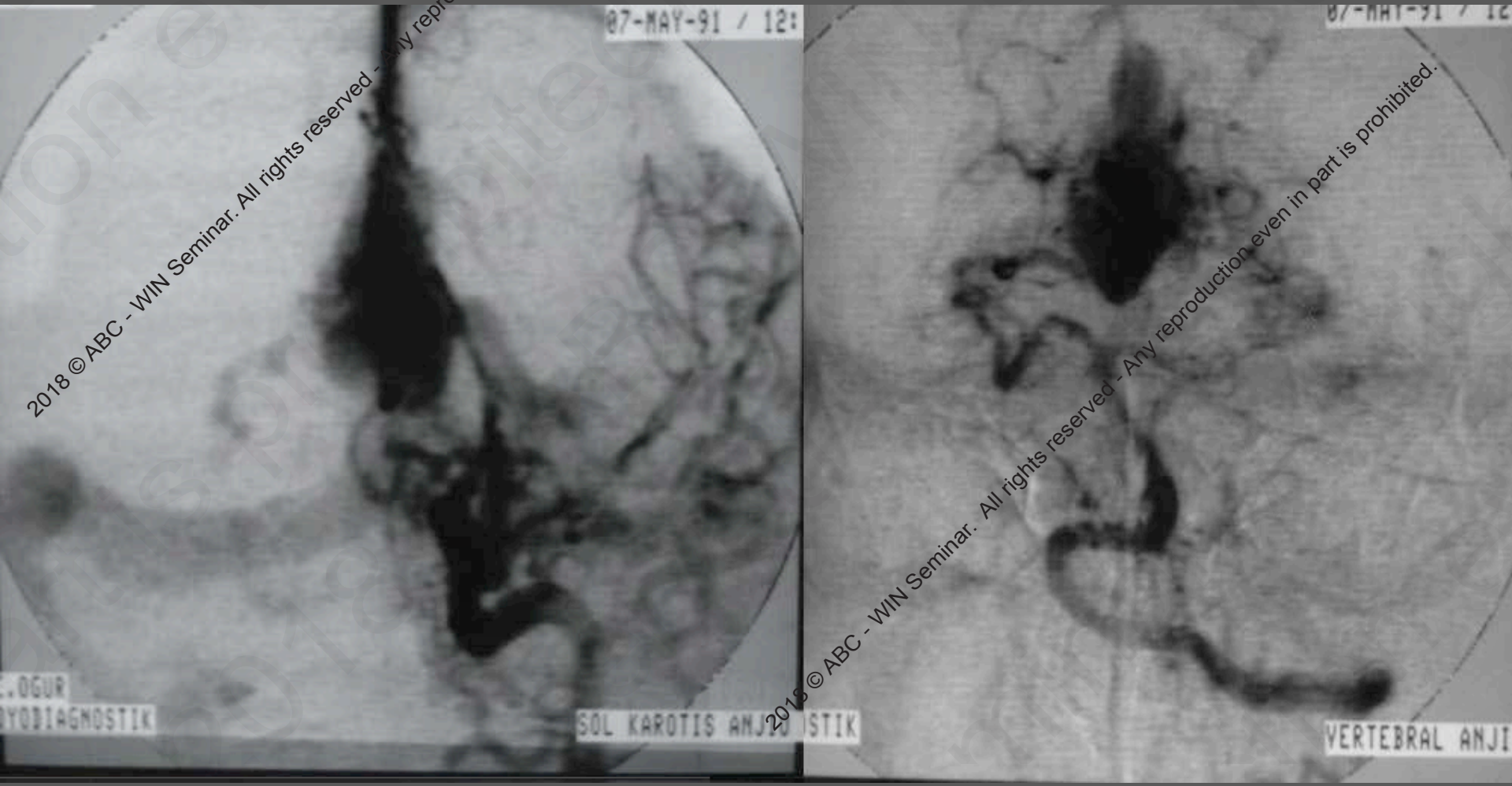
Neurological presentation and imaging findings of DAVF presented with progressive dementia and Parkinsonism.



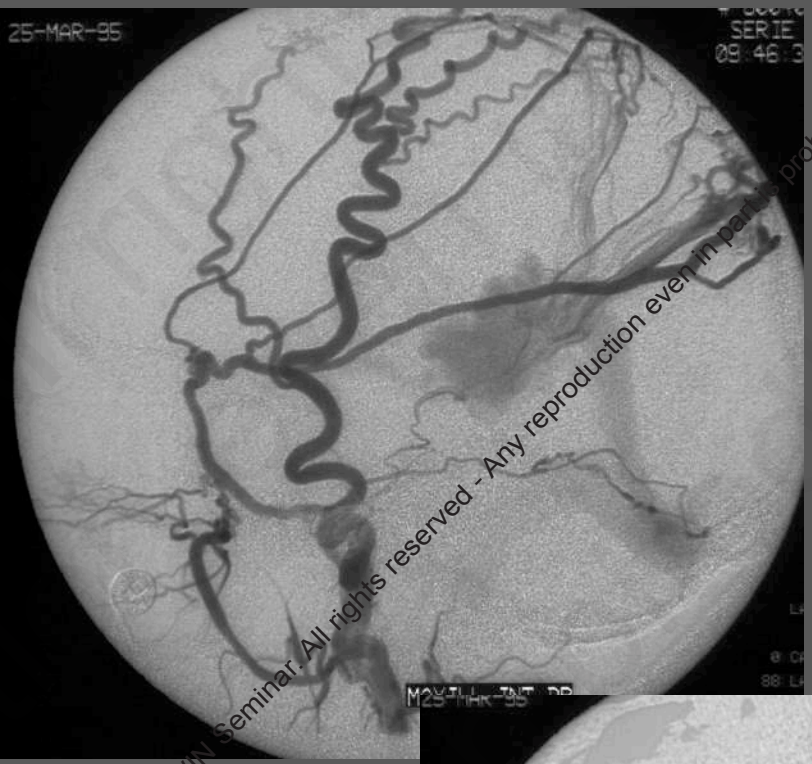


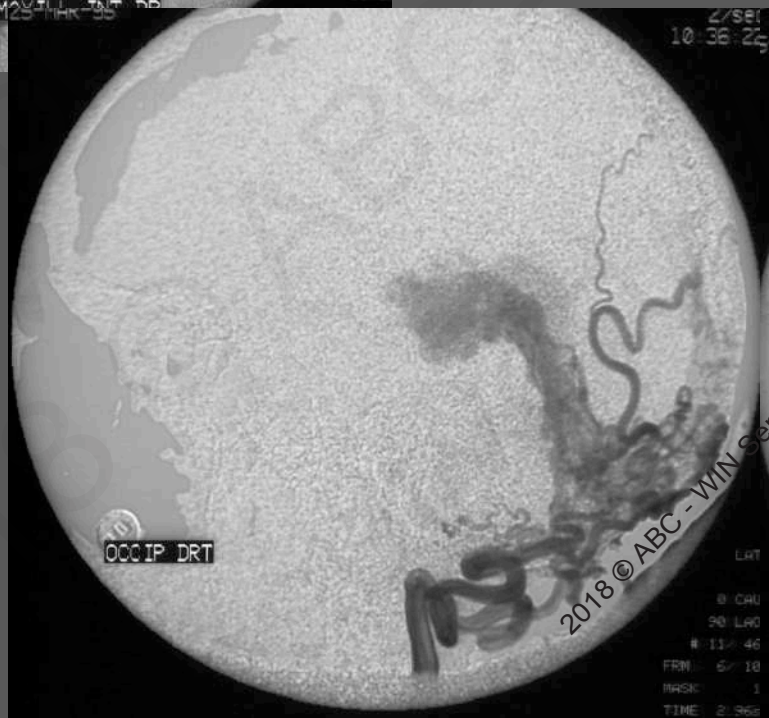
Galenic dural arteriovenous fistula: unusual clinical presentation and successful endovascular therapy, J. Eigele, J Neurosurg 2002

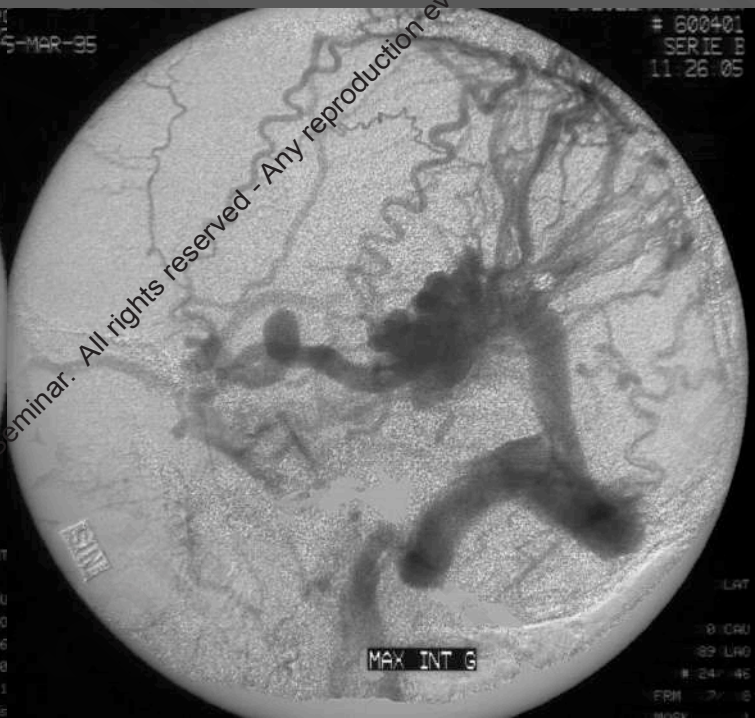
Male 31 y.o.
Since a few years progressive brainstem syptoms



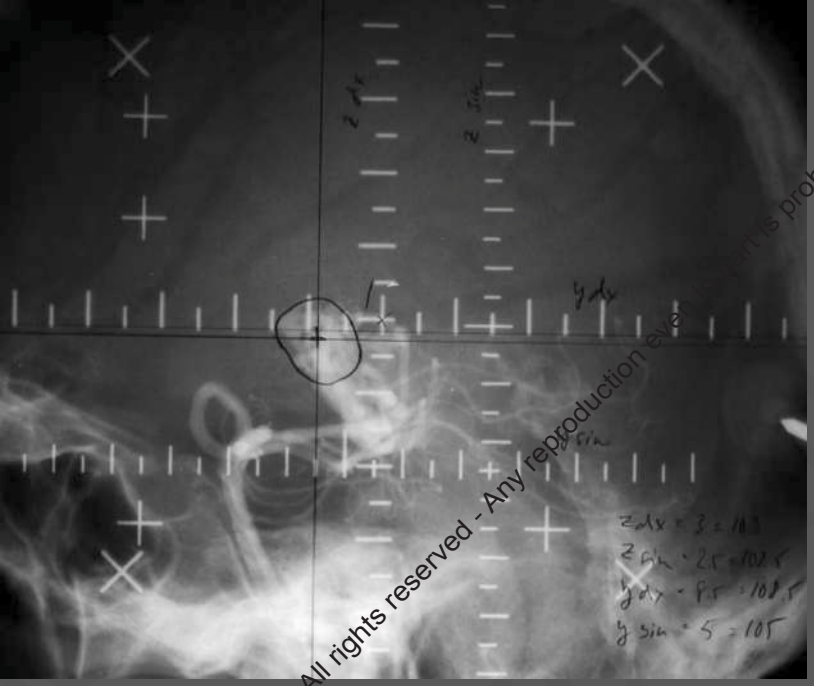
Transarterial embo with NBCA Limited flow reduction

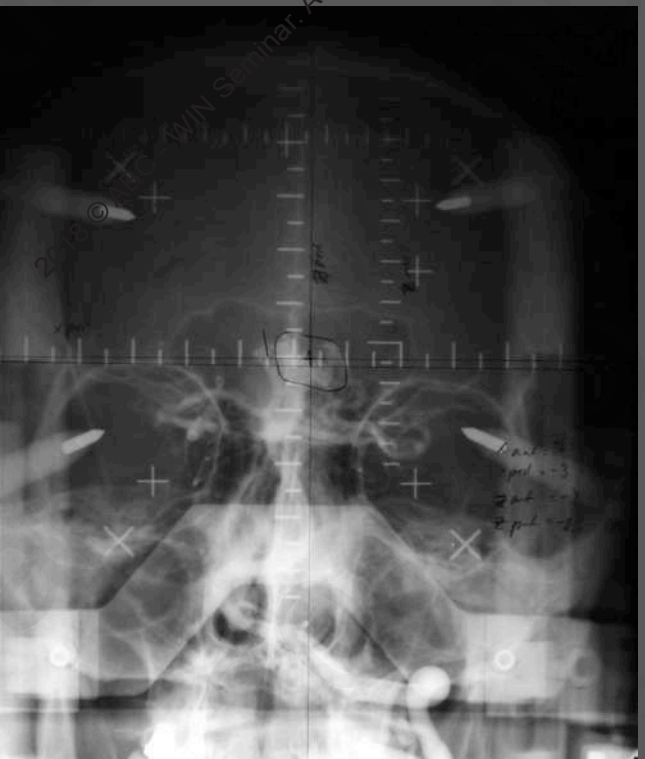


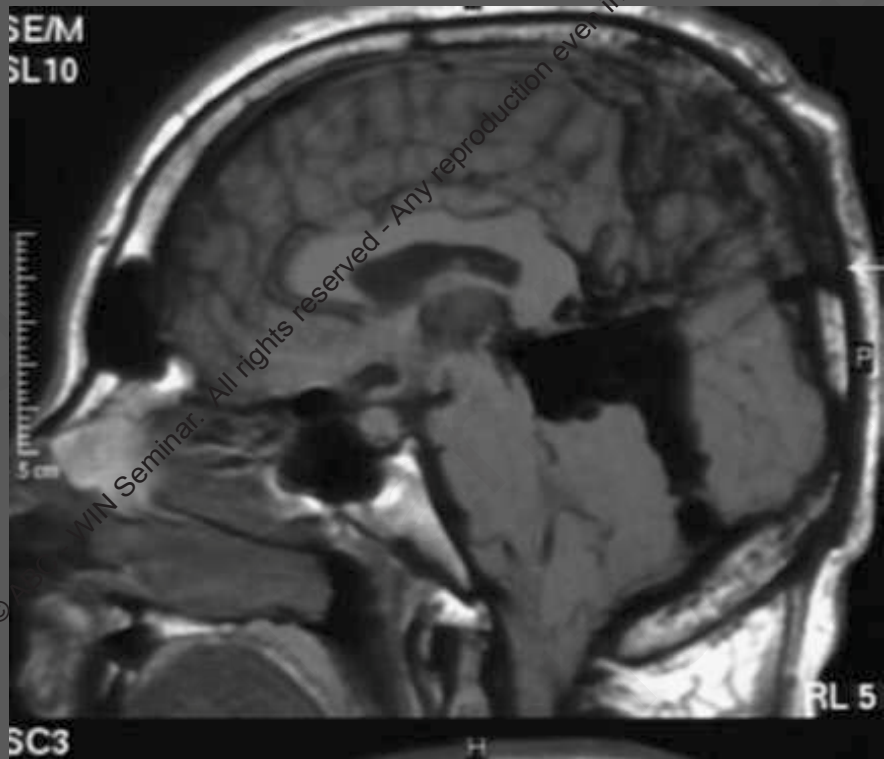


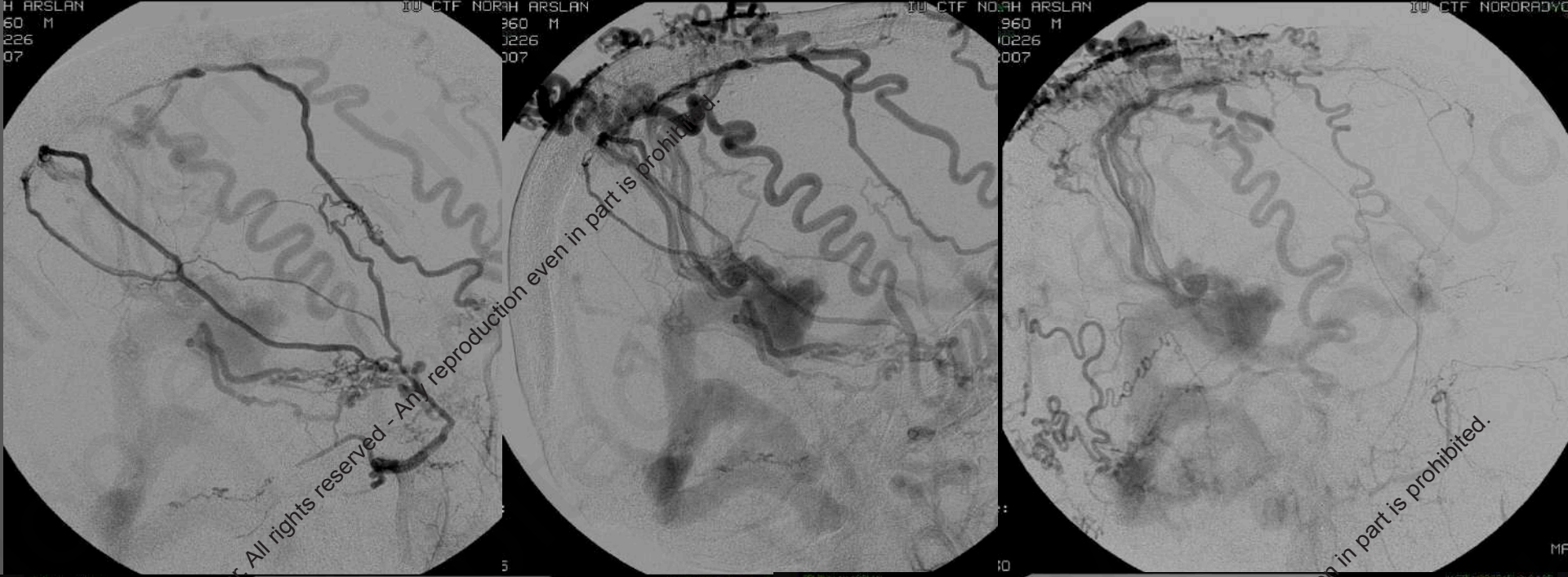


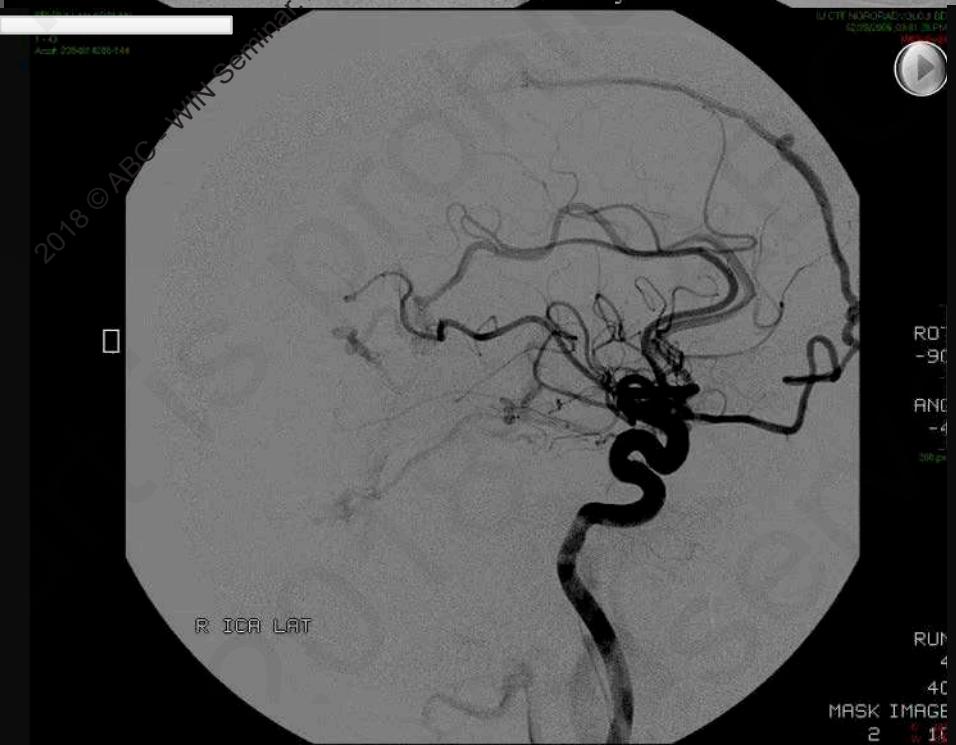
Gamma knife 25 Gy and two years later

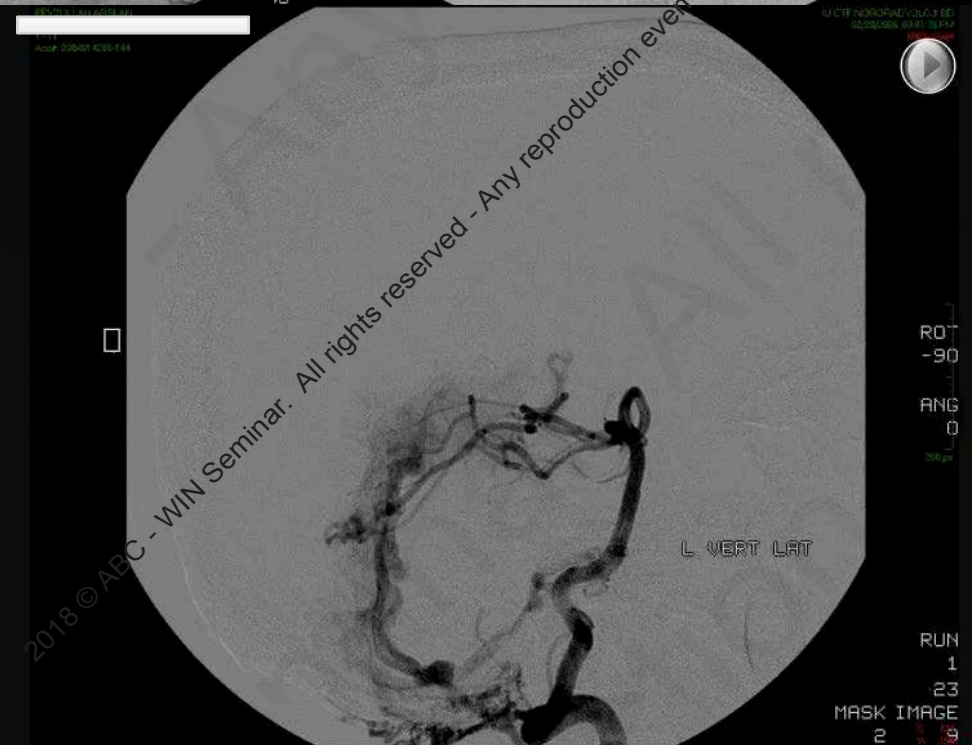


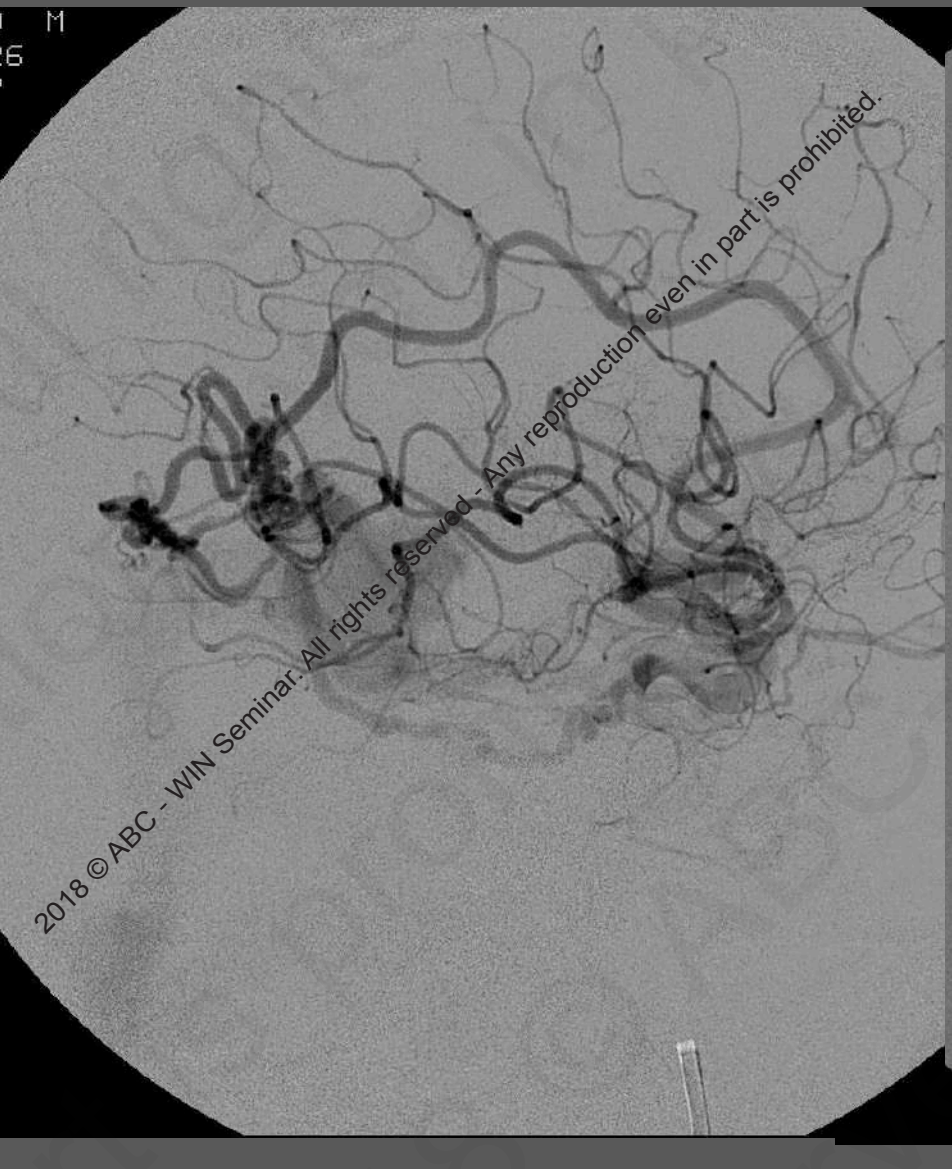


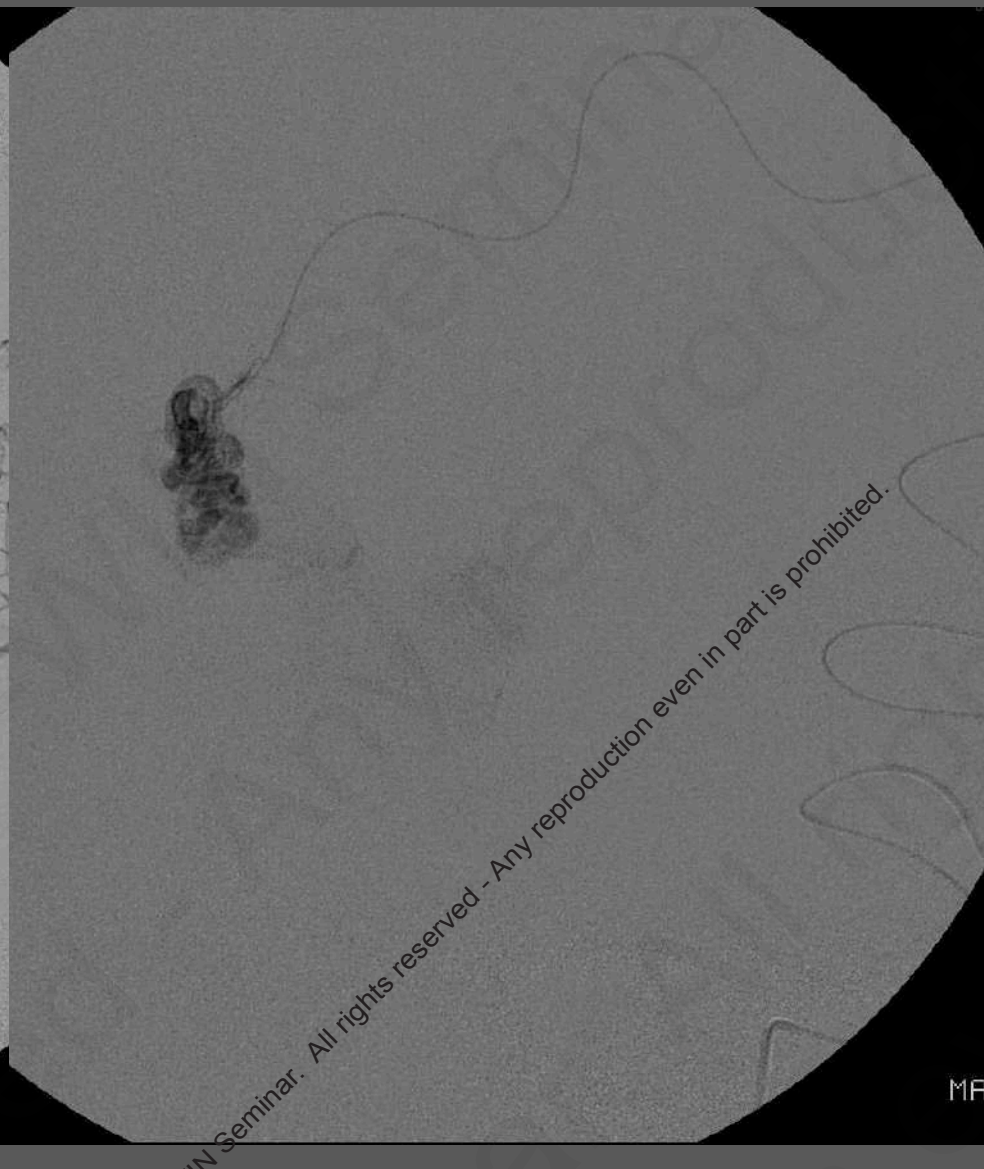


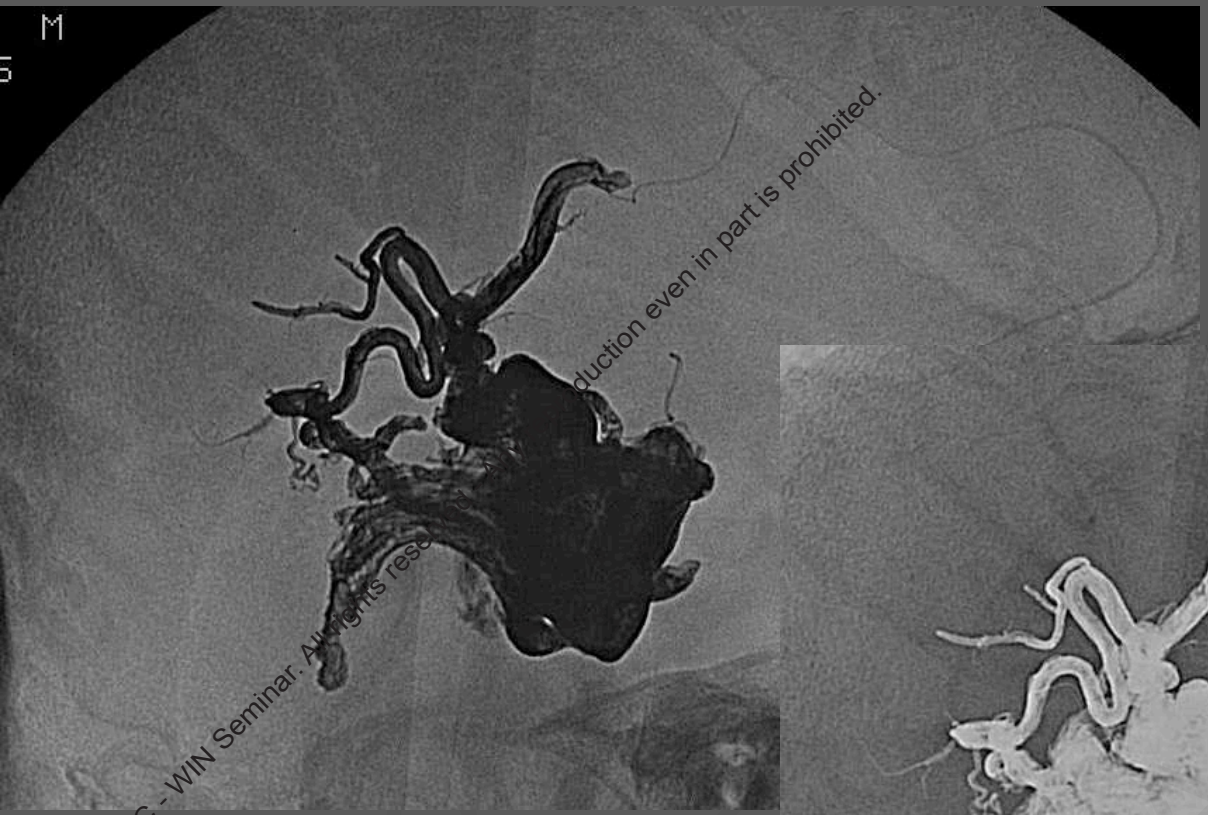


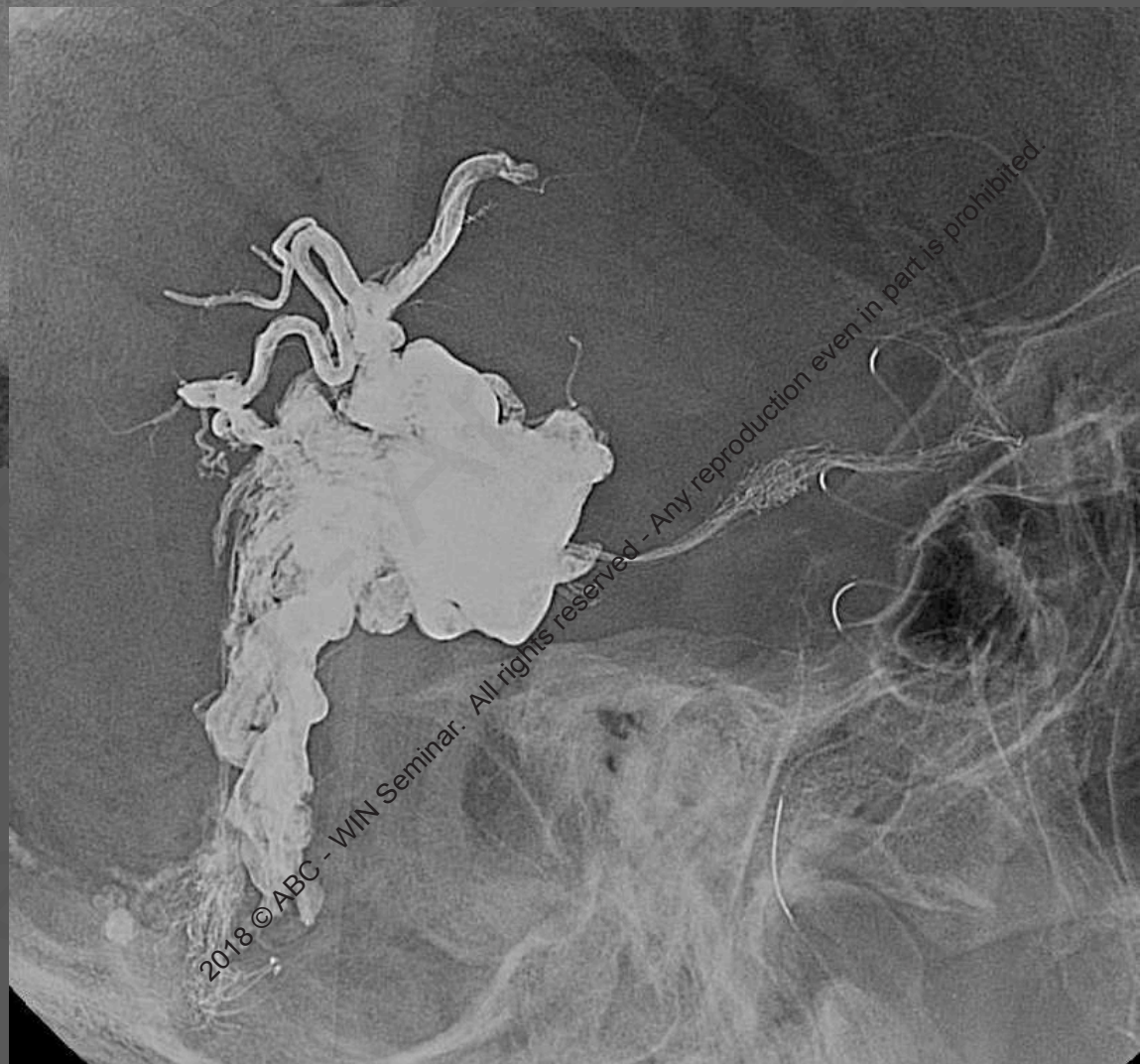


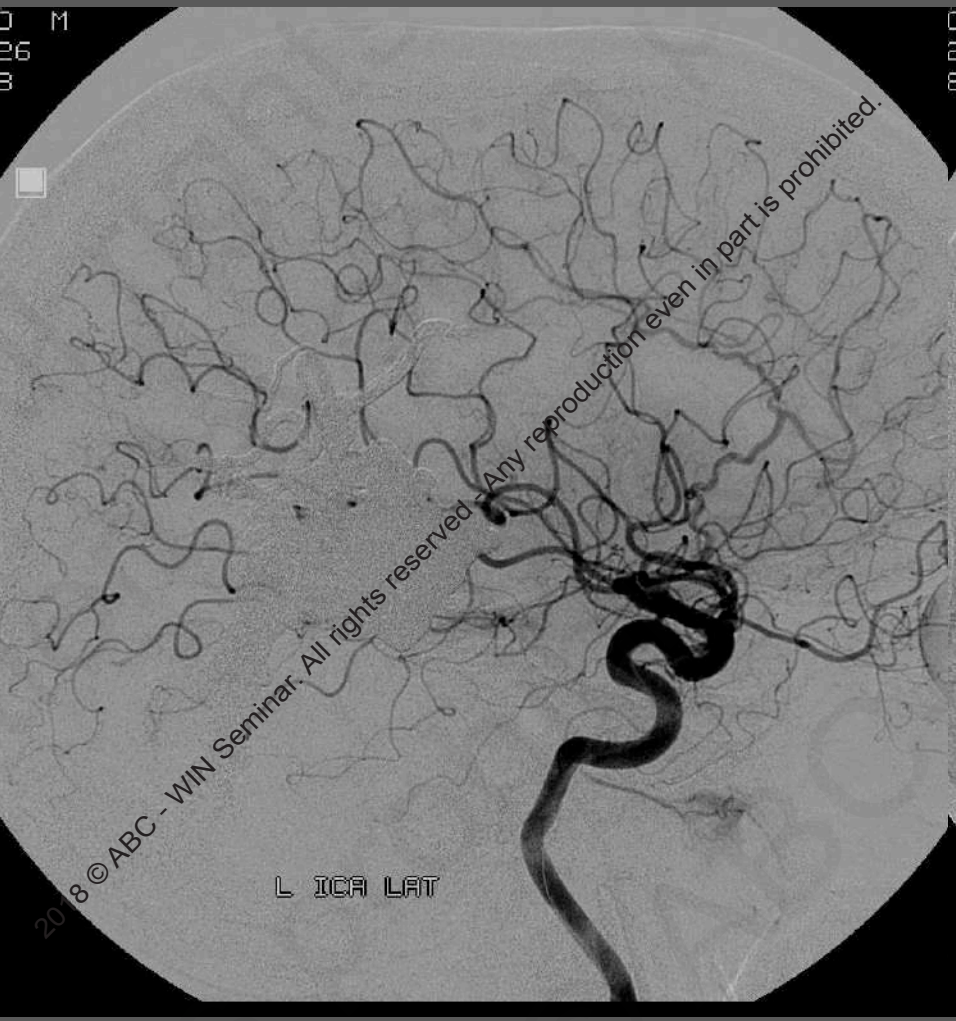


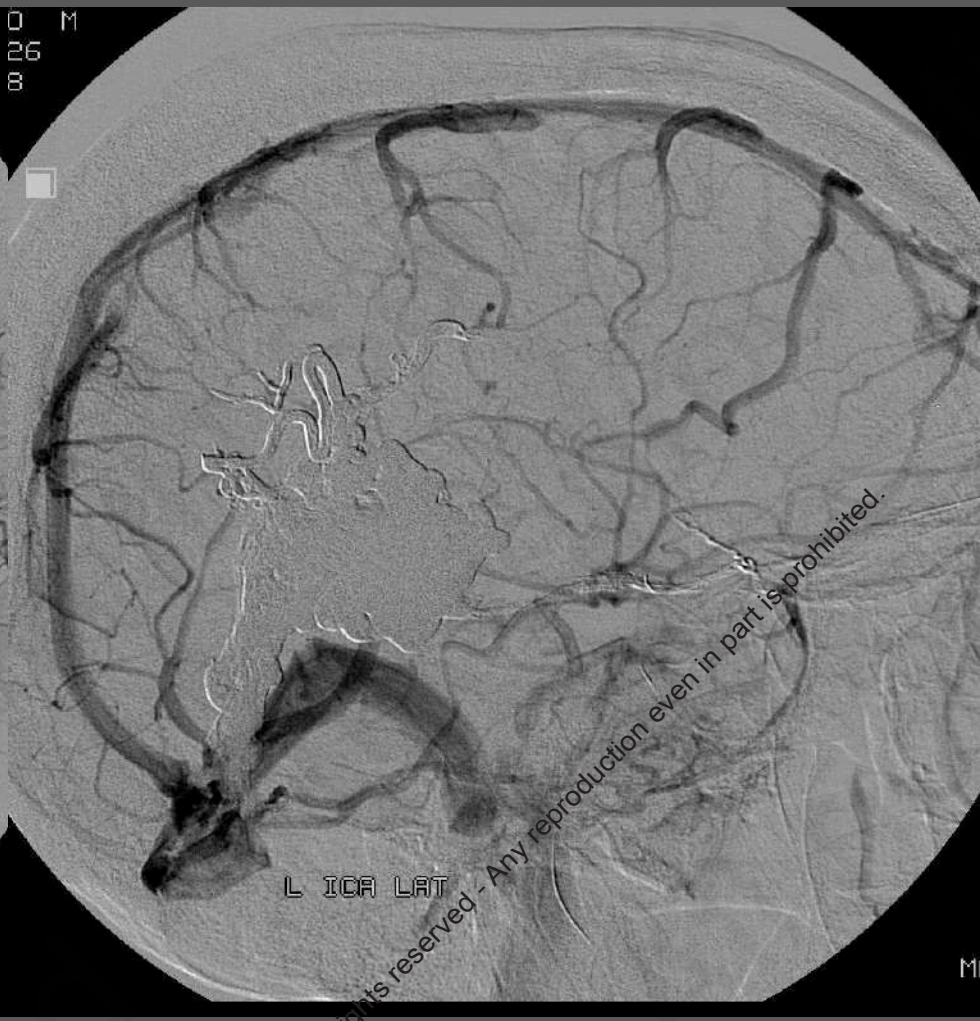












Conclusion

- **DAVS are assocated with increase in several angiogenic factors that can be induced by local venous hypertension or ischemia**
  - **Thrombosis may be a triggering factor**
  - **In cases without thrombosis the mechanims is unclear**
- **Transdural blood flow can also be induced by the same angiogenetic factors**
  - **Ischemia is a triggering factor**
- **DAVS in the falcotentorial area may develop symptom secondary to elevated venous pressure in central structures**