# Arteriovenous Malformation Of The Brain

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# Arteriovenous malformations(AVMs)

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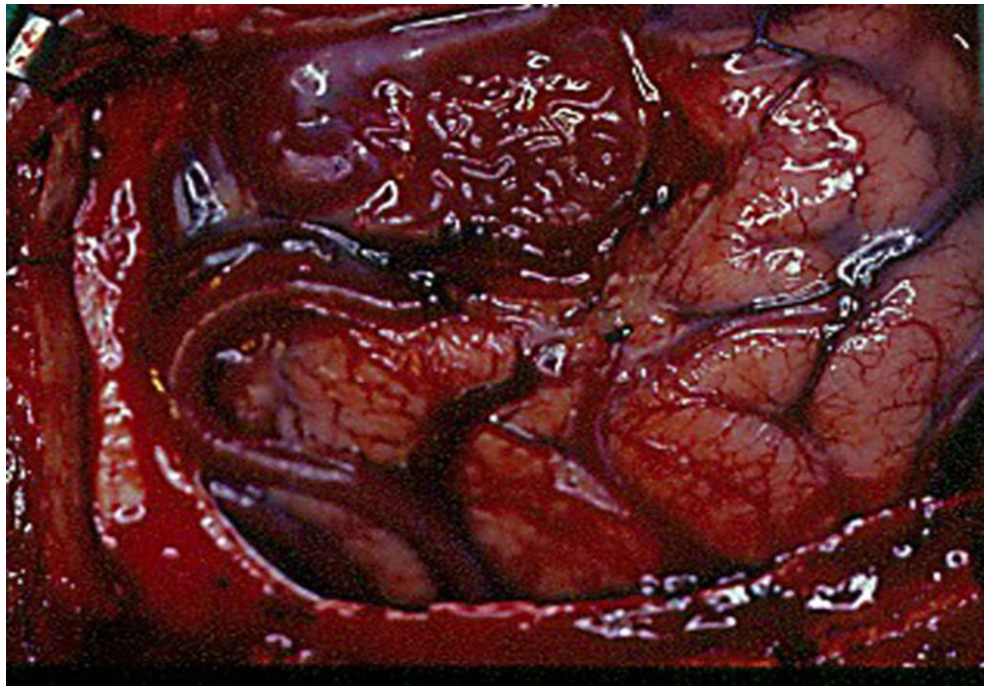
- History and Physical
- Evaluation
- Treatment/Management
- Prognosis
- Complication

# Introduction



- A developmental anomaly of the vascular system(tangles of poorly formed bloody vessels)

  feeding arteries direct to venous drainage, without capillary system

- Brain AVMs : the inherent high risk of bleeding: neurological damage

# Etiology

- Not much is known.  possibly multifactorial
- genetic mutation and angiogenic stimulation playing roles in AVM development.

  Some believe that AVMs develop in utero.
- others advocate an angiopathic reaction.

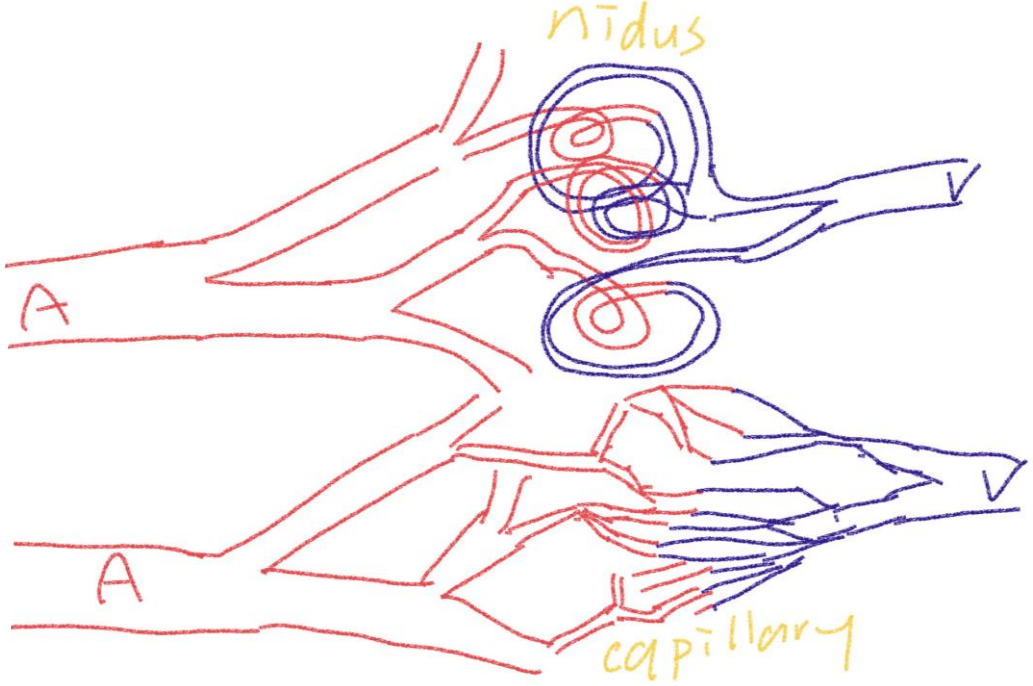
  following either a cerebral ischemic or hemorrhagic event

# Epidemiology

- The incidence in the United States is 1.34 per 100,000 person-years.
- Hemorrhage

  The mortality rate is 10-15% and morbidity rate is 30-50%.
- There is no sex predilection.
- Despite the considered congenital origin of AVMs, the clinical presentation most commonly occurs in young adults.

# Pathophysiology

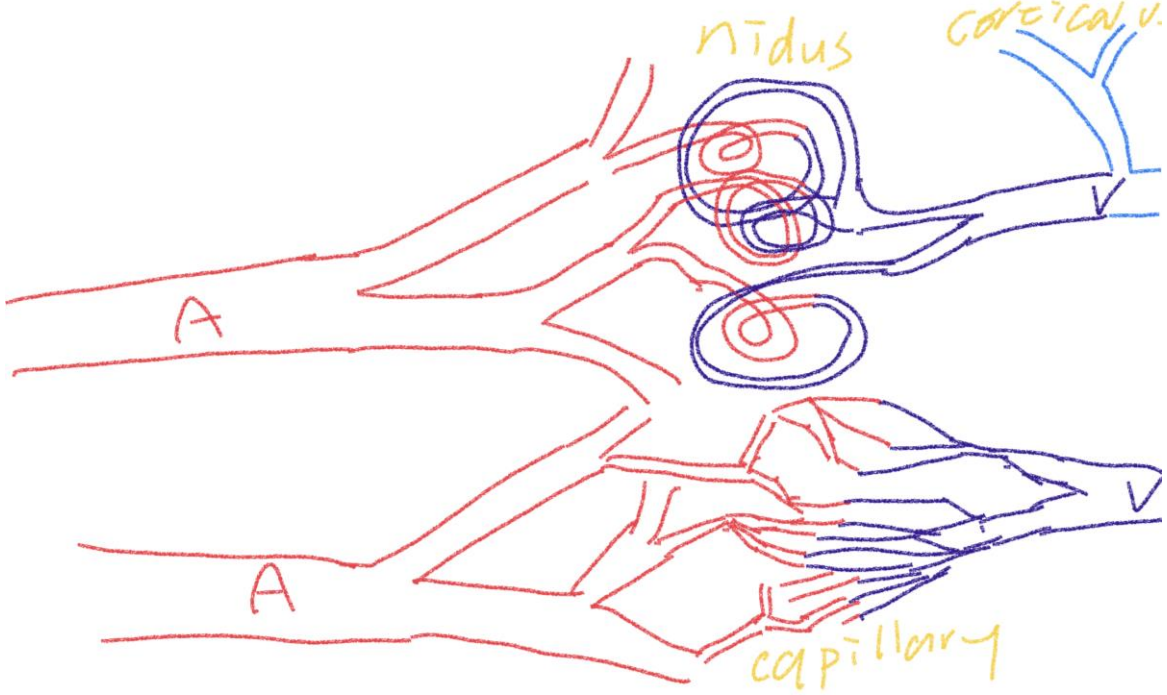



- AVMs:a central vascular nidus which is a conglomerate of arteries and veins.

  **There is no intervening capillary bed

- the feeding arteries drain directly into the draining veins by one or multiple fistulae.
- These arteries lack the normal muscularis layer and the draining veins often appear dilated

# Pathophysiology-Neurological dysfunction




- Firstly, the abnormal blood vessels have a propensity to bleed resulting in diffuse SAH, IVH and ICH.
- Secondly, seizures may occur as a consequence of the mass effect of AVM or venous hypertension in draining veins.
- The third important cause of slowly progressive neurological deficits is attributed to the“ steal phenomenon"

# History and Physical-hemorrhage

- Clinically asymptomatic in 15% of cases until the presenting event occurs.
- 41 to 79 % present with intracranial hemorrhage(mainly intraparenchymal).

  10-15% of all SAH (the second most common cause of SAH after aneurysms)
- Children are more likely to present with hemorrhage than adults.
- S/S: loss of consciousness, severe headache, nausea/vomiting, seizure, hemiparesis, a loss of sensation of the body, and deficits in language.

# History and Physical- seizure, steal syndrome

- Seizure:15 to 40%. The higher risk: cortically-located, large, multiple, and superficial-draining AVMs.
- Steal syndrome : Progressive neurological:6-12% of patients over a few months to several years.
- Headache: no specific headache features

# Complication-(Neurological deficits)

- Intracranial bleed
- Mass effect
- Seizures
- Steal phenomenon

# Evaluation-Computed tomography (CT)

- Non-contrast CT:

  the nidus is blood density: usually hyperdense, enlarged draining veins, and calcification.
- Postcontrast CT with CT angiogram(CTA):

  visible feeding arteries, nidus, and draining veins :"bag of worm"
- The sensitivity of CT to identify brain AVMs decreased  in the acute hemorrahge stage

# Evaluation-Computed tomography (CT)

# Evaluation-Magnetic resonance imaging(MRI)

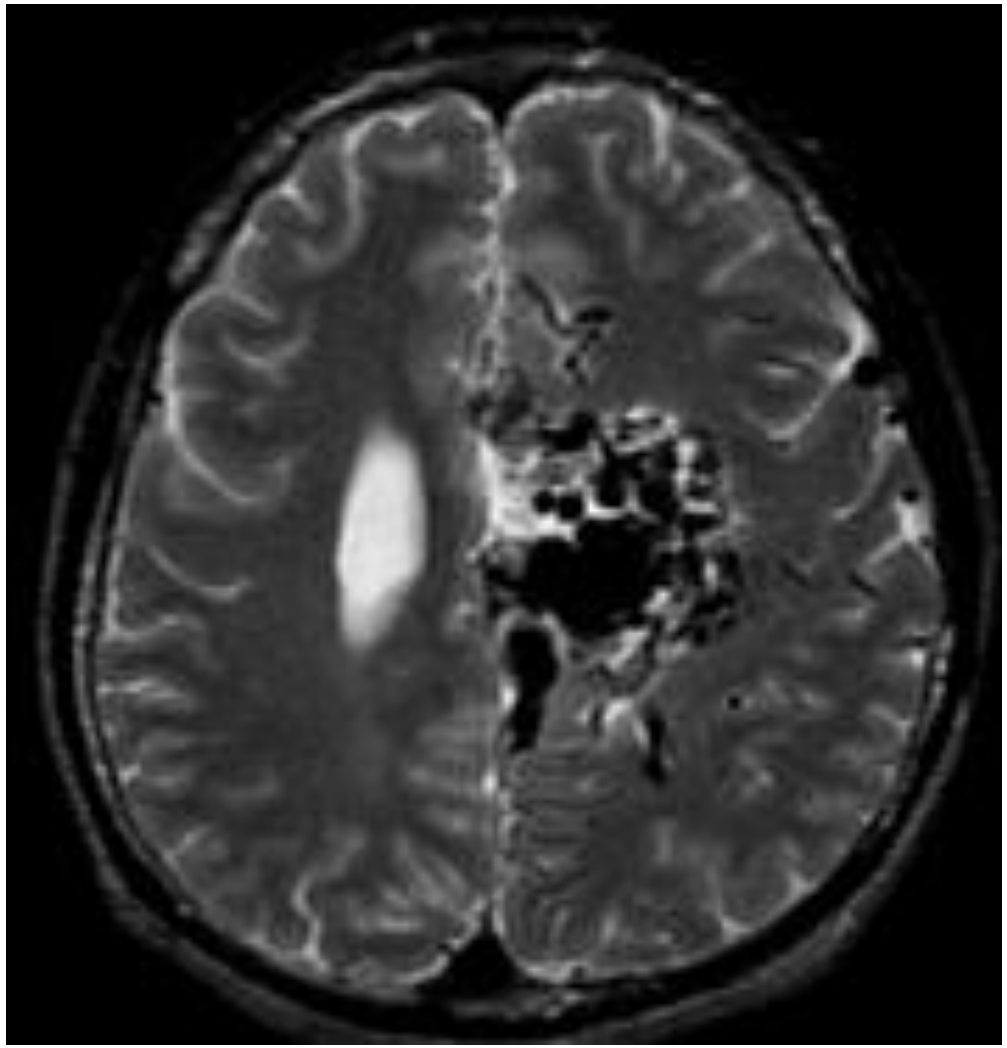
- MRI: very sensitive for nidus and draining vein or any distant bleeding.

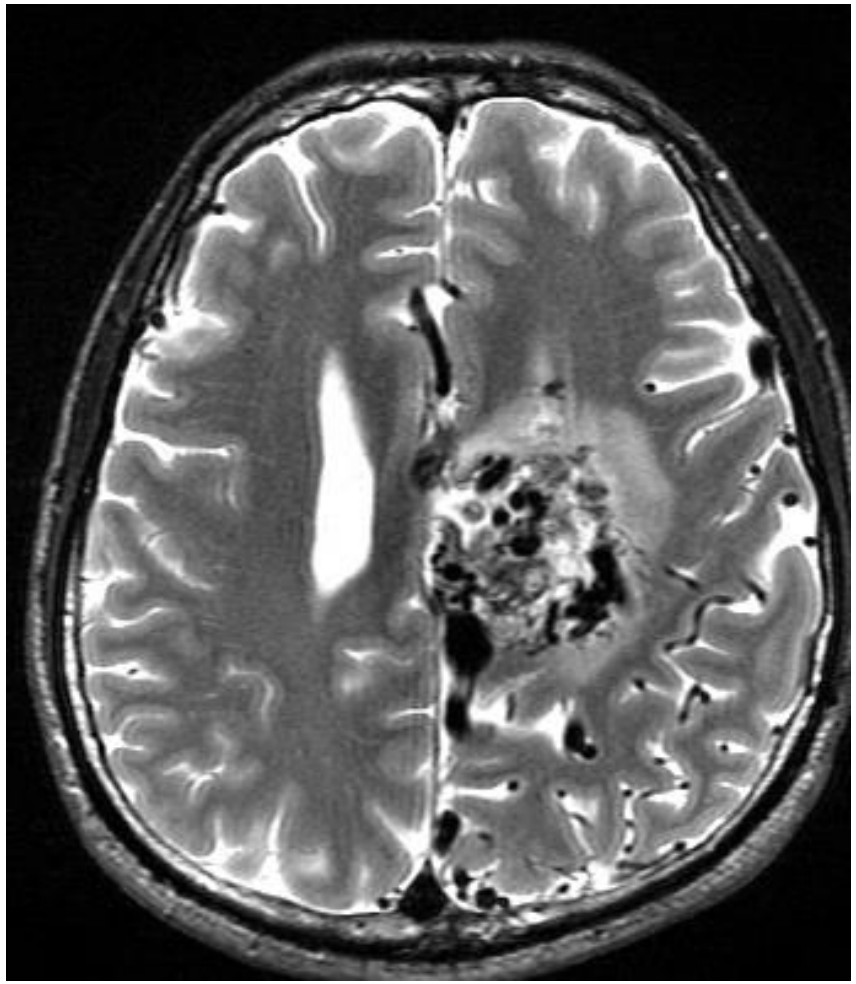
  **Flow voids seen on bothT1 and T2(mostly evident on T2 weighted):

  fast flow in a conglomerate of tangled blood vessels

- Previous hemorrhage, adjacent brain edema, and atrophy may be seen.
- After radiosurgery, MRI can evaluate the nidus volume, post-therapy edema, radiation necrosis.

# Evaluation-Magnetic resonance imaging(MRI)

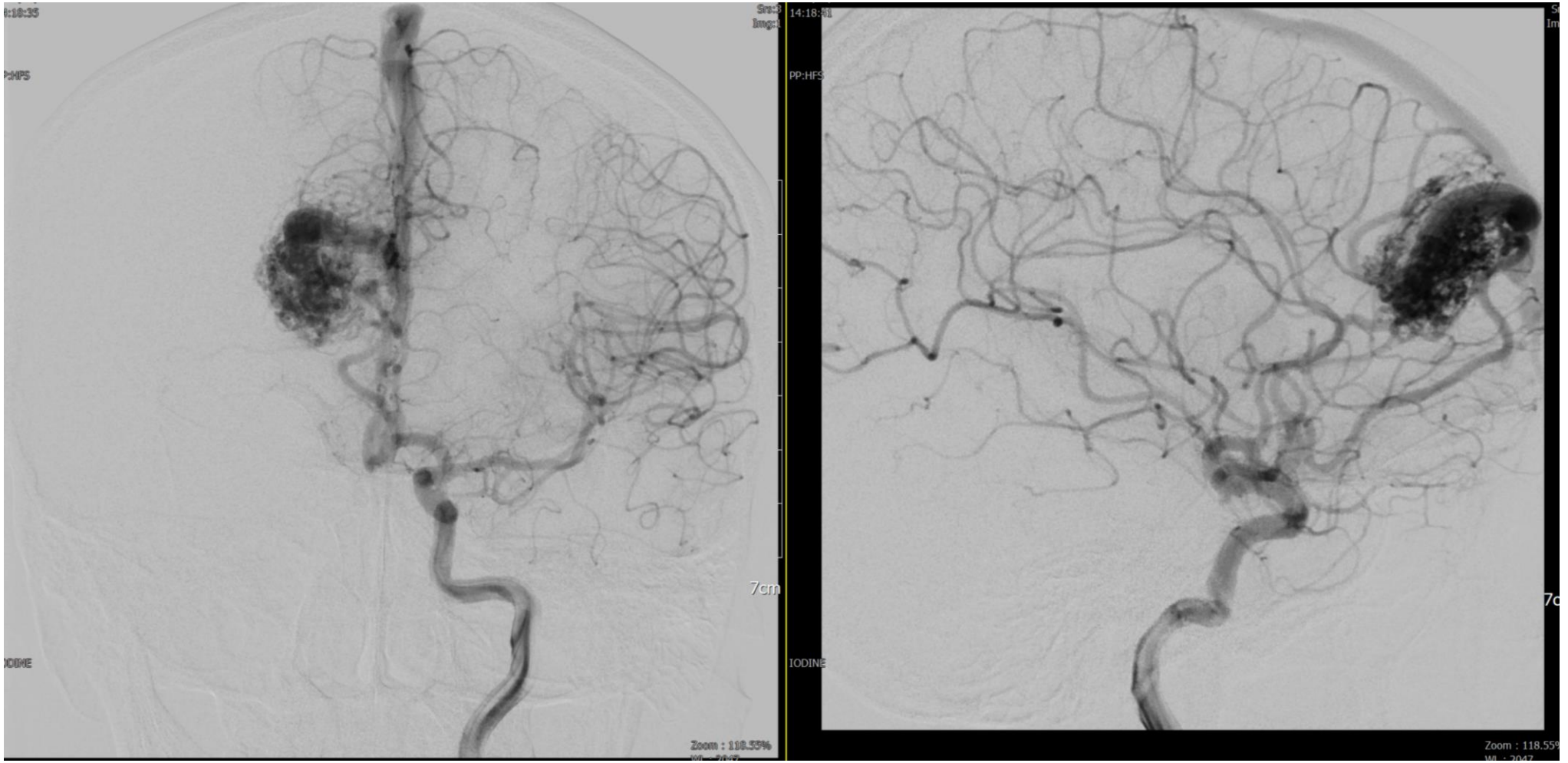




# Evaluation-Digital subtraction angiography (DSA)

- DSA :the gold standard for diagnosis and treatment planning.
- Nidus configuration, its relationship, and drainage to surrounding vessels are precisely evaluated.
- The presence of an associated aneurysm suggests a higher risk for hemorrhage.
- Contrast transit time, which relates to the flow state of the lesion, can provide critical information for endovascular treatment planning.

# Evaluation-Digital subtraction angiography (DSA)



# Treatment/Management

- Invasive management: younger patients with high-risk features for an AVM rupture

  *hx of previous rupture, AVM size, location, presence of aneurysms, intractable epilepsy
- Medical management : older individuals with no high-risk features

  *anticonvulsants for seizure control and pertinent analgesia for headaches

# Treatment/Management

- Open microsurgical excision (surgical excision): offers the cure for patients with high risk of hemorrhage.
- Radiotherapy and endovascular: alternatives to surgical treatment in patients at high risk for surgical therapy but can also be useful adjuncts to the main surgical management.

# AVM scoring systems

- There are different scoring systems in order to identify the morbidity and mortality associated with observation vs intervention in different types of cerebral AVMs. The main ones are:

1. Spetzler-Martin scale - for microsurgery
2. Supplementary Spetzler-Martin scale - for microsurgery
3. Pittsburgh radiosurgery-based AVM grading scale
4. Toronto score - for microsurgery
5. Buffalo Score - for endovascular treatment

# Spetzler-Martin Grade (SMG) scale

- Assess the risk of surgical morbidity and mortality with brain AVMs.

  **nidus size (<3 cm, 3-6 cm, >6 cm; 1-3 points),

  **the eloquence of adjacent brain (1 point for lesions located in eloquence region)

  **venous drainage (1 point if venous drainage is via deep veins).

# Spetzler-Martin Grade (SMG) scale

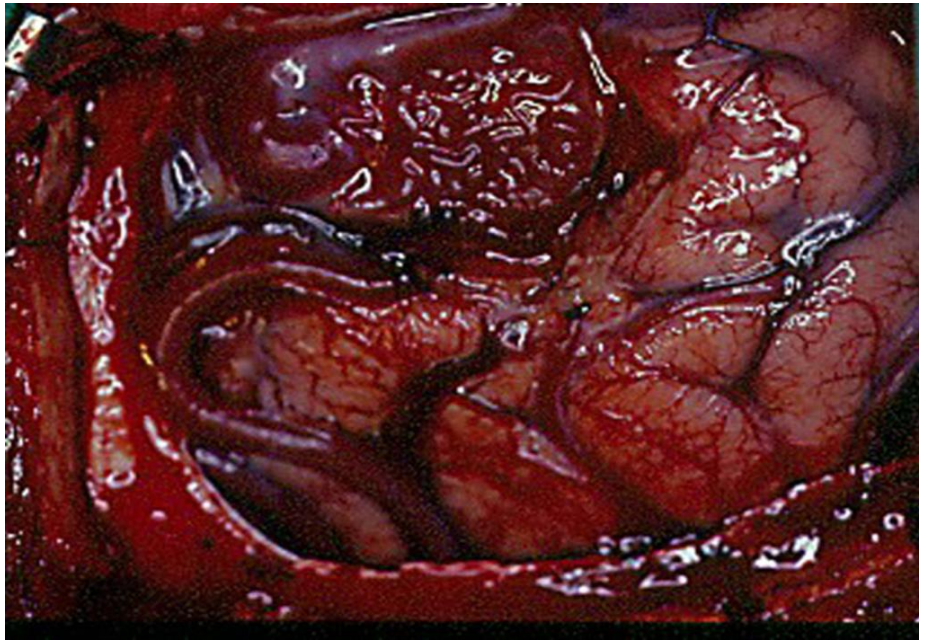
| Criteria | Score |
|---|---|
| ***Size of Nidus*** | |
| Small (,3 cm) | 1 |
| Medium (3-6 cm) | 2 |
| Large (>6 cm) | 3 |
| ***Eloquence of Adjacent Brain*** | |
| No | 0 |
| Yes | 1 |
| ***Deep Vascular Component*** | |
| No (superficial) | 0 |
| Yes (deep) | 1 |
| ***Sum of Scores*** | |
| Size + eloquence + depth = AVM score | |

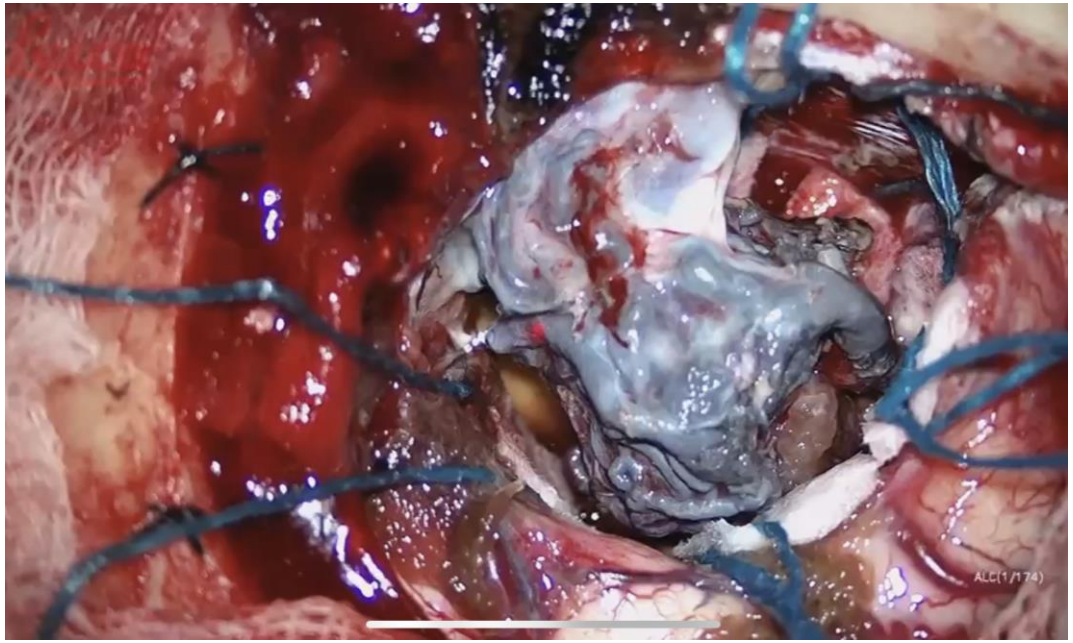
# Surgical intervention

# 46F, Right F AVM

- (1) perform a craniotomy to obtain adequate exposure to the AVM, including its arterial feeders and venous outflow
- (2) isolate and divide its arterial feeders
- (3) circumferentially dissect the nidus from the adjacent brain parenchyma and surrounding neurovascular structures
- (4) disconnect the venous outflow
- (5) close the wound.

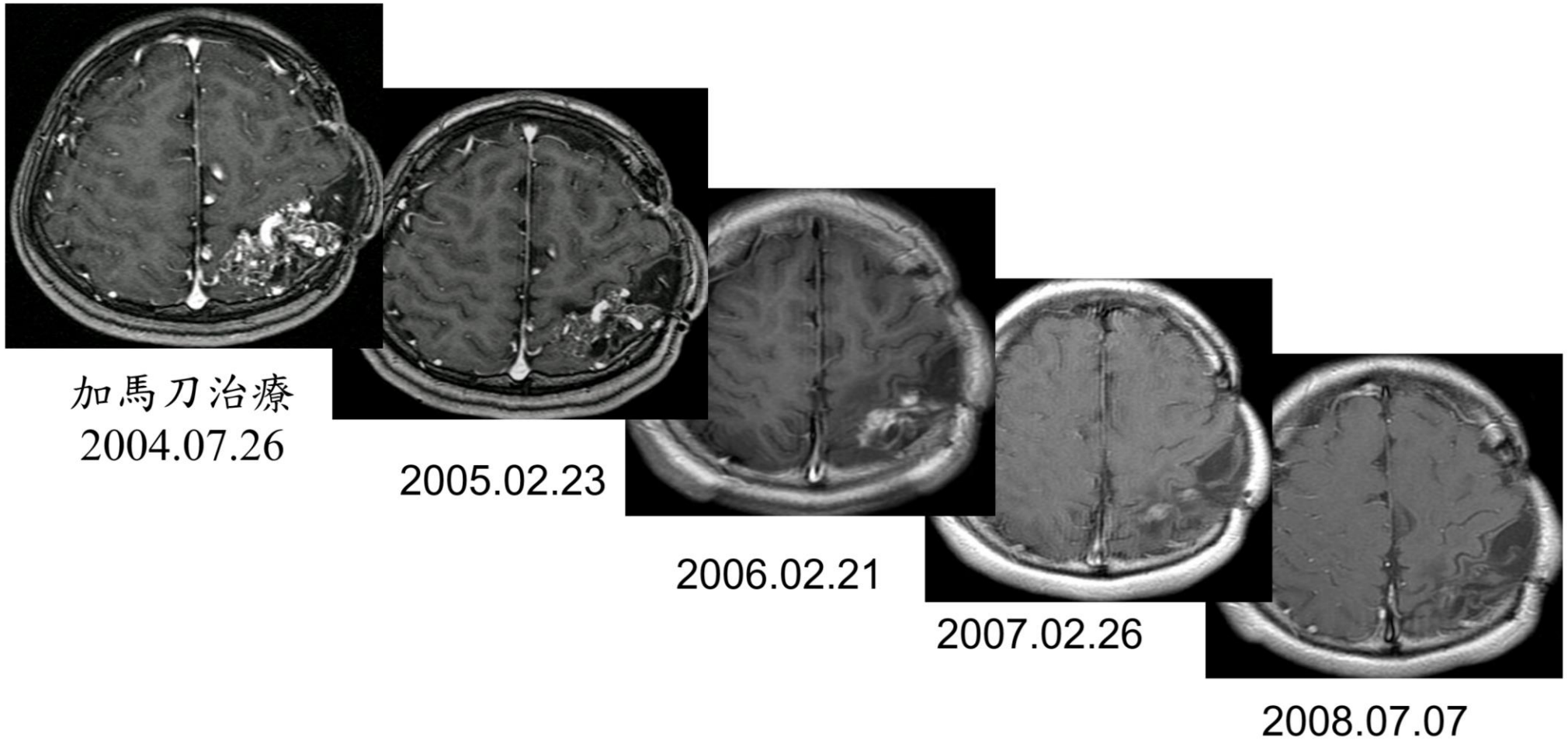
# 46F, Right F AVM





# Radiosurgery

# 14 y/o boy, ICH, left PO AVM



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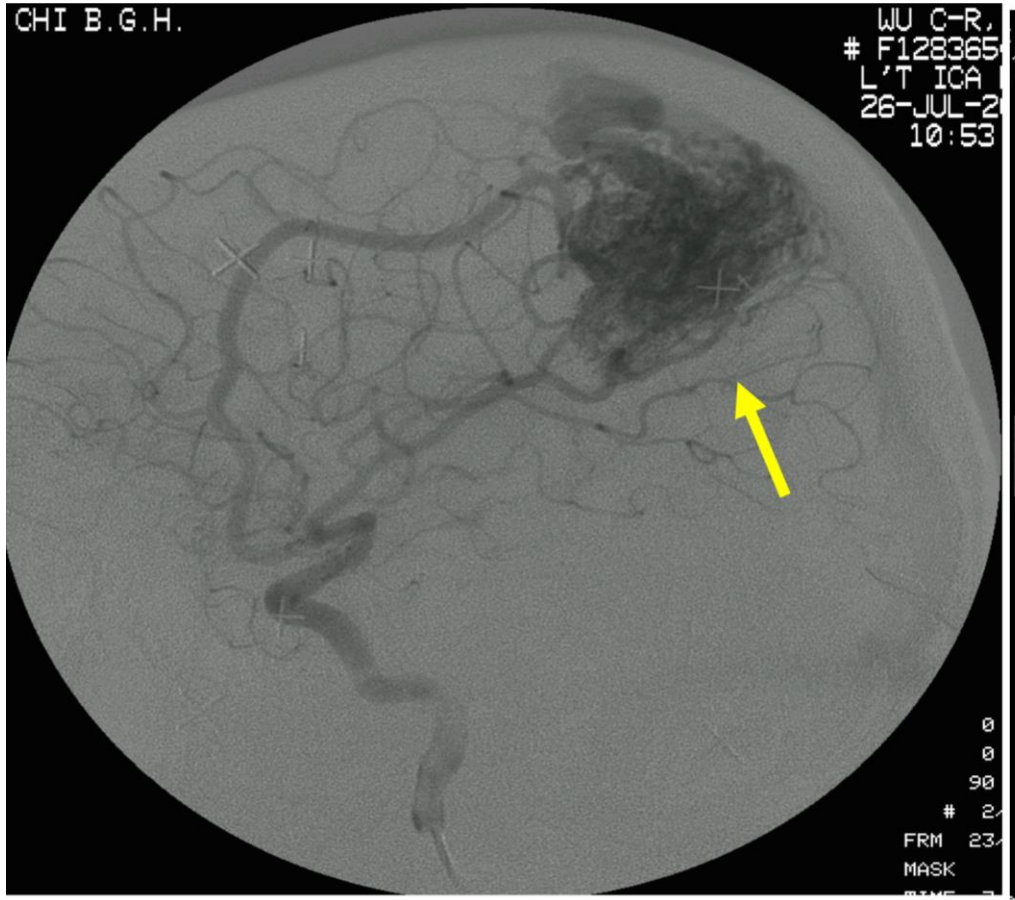
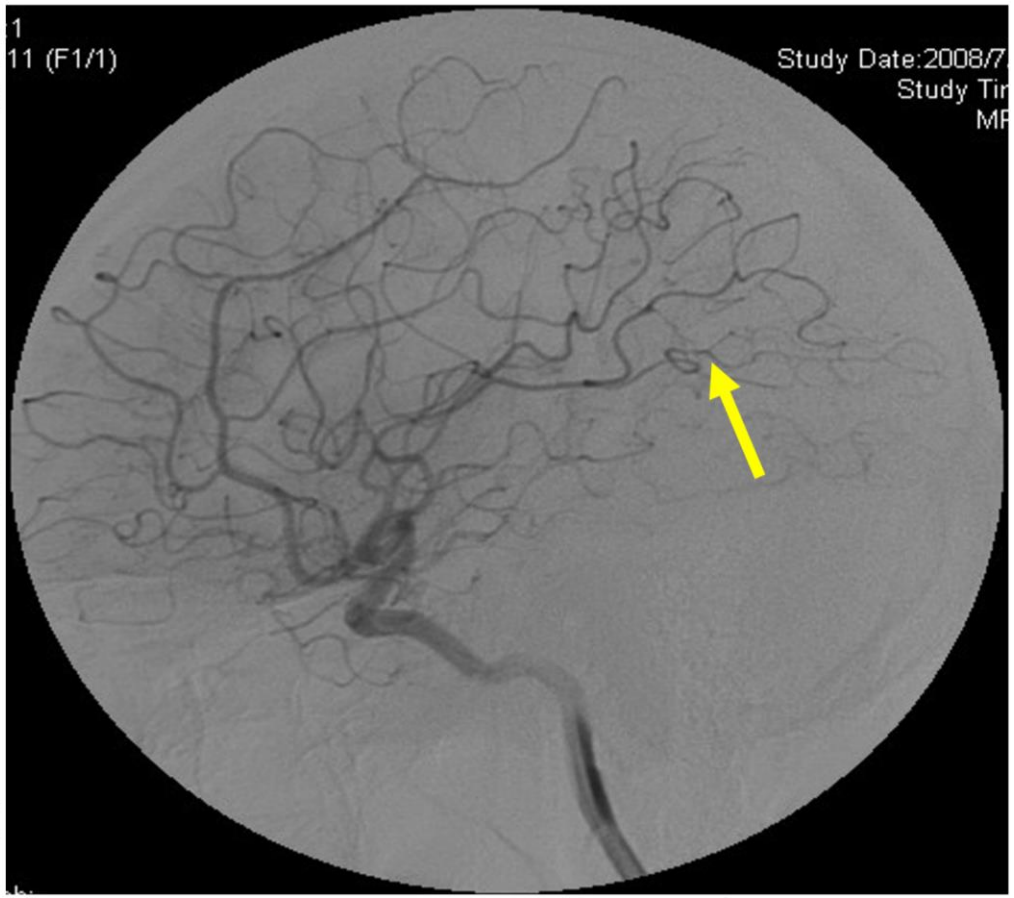



加馬刀治療
2004.07.26




加馬刀治療後4年
2008.07.22

# 14 y/o boy, ICH, left PO AVM


加馬刀治療
2004.07.26


(治療後)
2008.07.22

# Endovascular surgery

# Goal of embolization of AVM

- To permanent reduce the size and diminish the anomalous flow of the AVM

  **reduce the risk of hemorrhage
- Reduce the risk of the curative procedures

  **surgical resection or radiosurgery

# Indication/ recommendation for treatment of AVMs

- An associated arterial aneurysm in the feeding pedicle/ nidus.
- Venous thrombosis, outflow restriction, venous hyper-pressure, venous pouches or dilatation, or venous pseudoaneurysm.

# Technique of embolization of AVM

- General anesthesia.
- 6 or 7F guiding catheter
- Flow-guided or over-the-wire microcatheter
- Selection of embolic agents

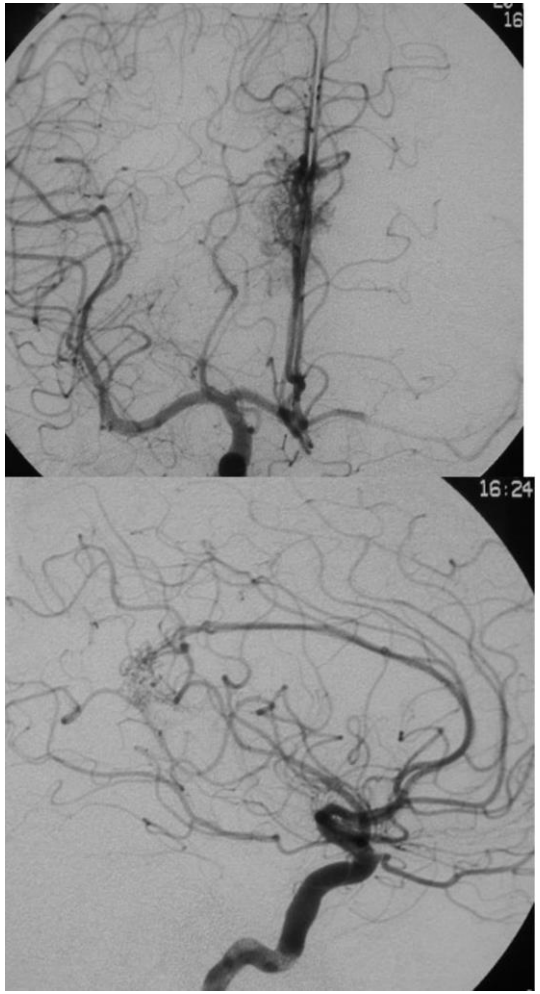
# Common embolic agents for AVM embolization

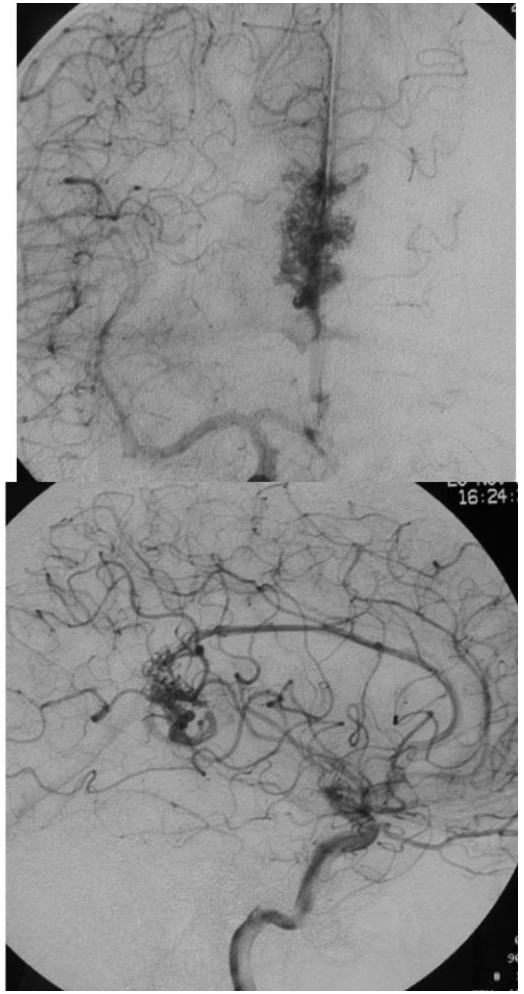
- Liquid adhesives (NBCA)
- Particles
- Absolute alcohol
- Proximal coil occlusion of feeders, liquid coils
- Other agents: hydrogels, oils, sclerosing agents, plastics…etc.
- Non-adhesives agent: Onyx.

# Curative embolization of BAVMs

- Complete anatomical obliteration of the BAVM by endovascular route.
- Confirmation by immediate post-embolization is not sufficient, F/U at least 6 months later, preferably 1-2 year later.
- Anatomical cure in 71% of AVM with a volume between 4-8mL (13.3% for all).
- Only in a limited number of patients: in 5-15% of BAVMs.

# 32F, seizure, right TP AVM



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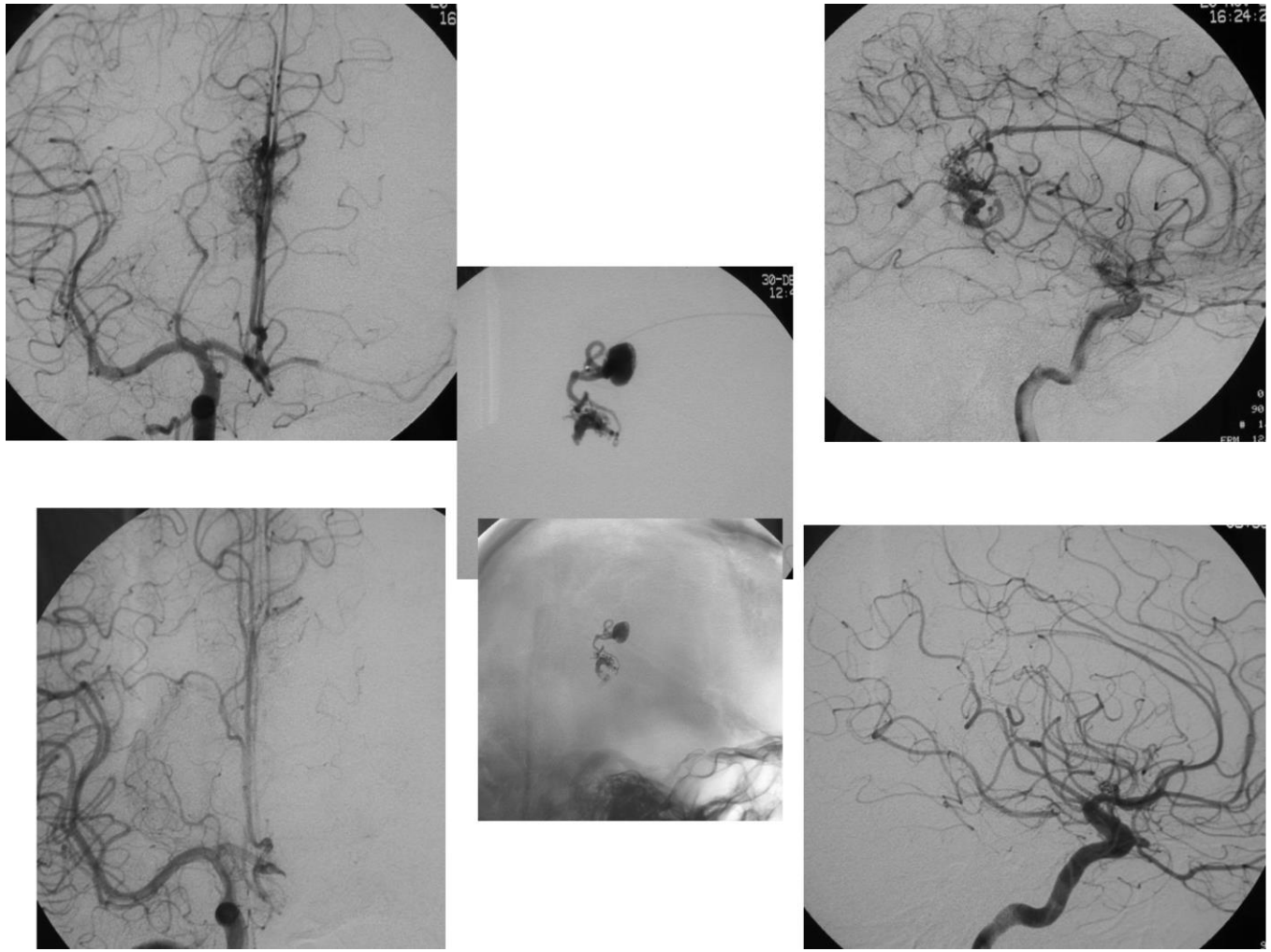

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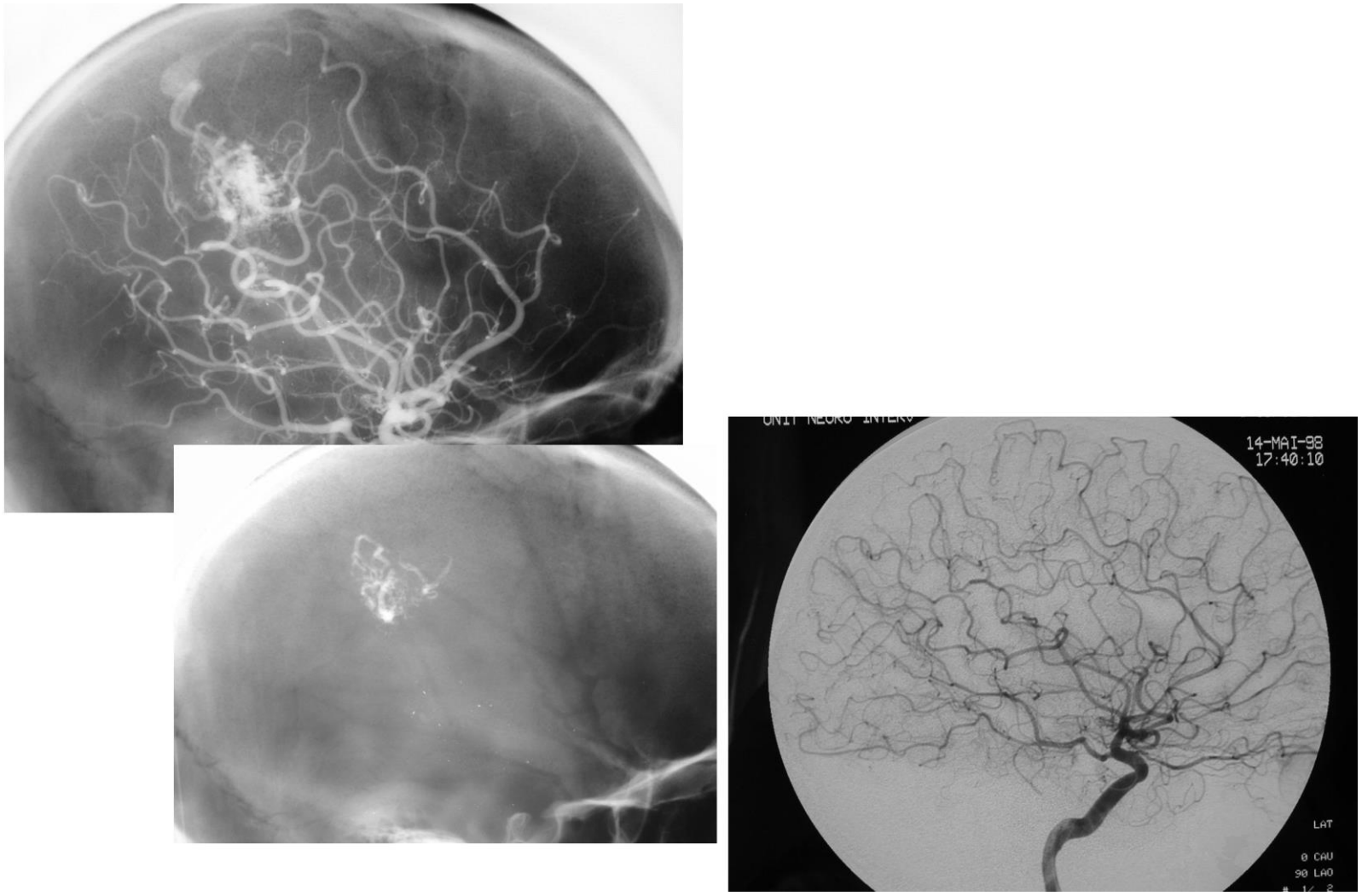
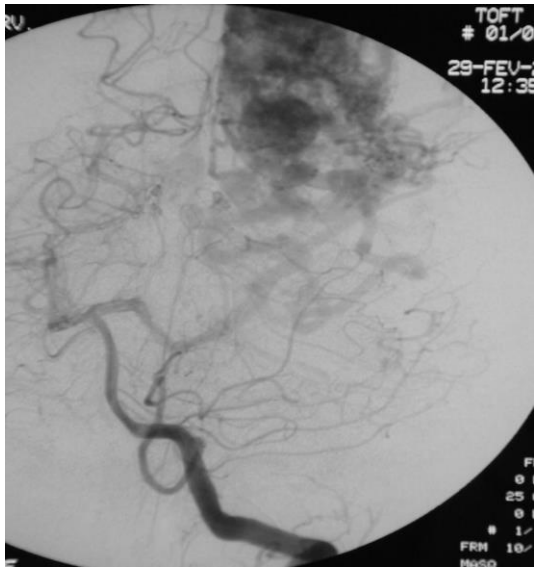
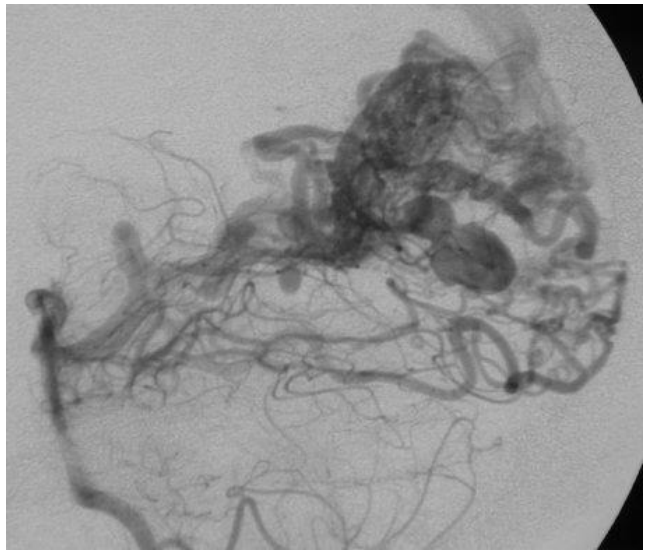


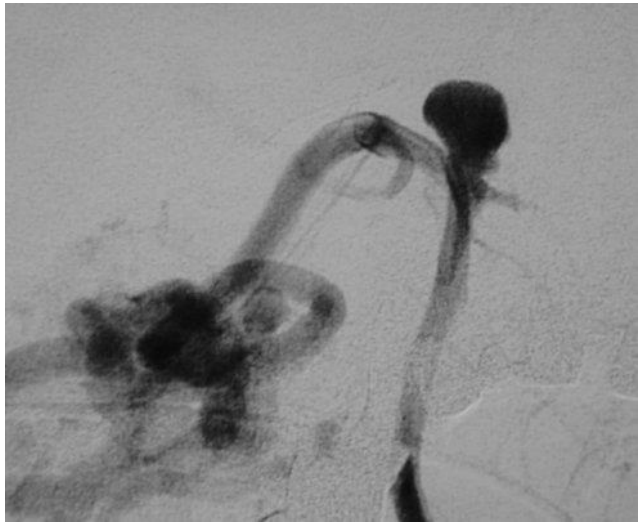
# Partial, palliative embolization of AVMs

- Clinical cure.
- To stabilize the weakness in the angio-architecture (e.g. intranidus aneurysm, aneurysms in the arterial or venous side of malformation).
- To close aneurysms in the pedicle to a AVM.
- Occlusion of the high-flow fistulas, to decrease the flow and/or pressure in draining veins or to improve tissue perfusion by decreasing venous hypertension.

# 43 y/o F , left O BAVM





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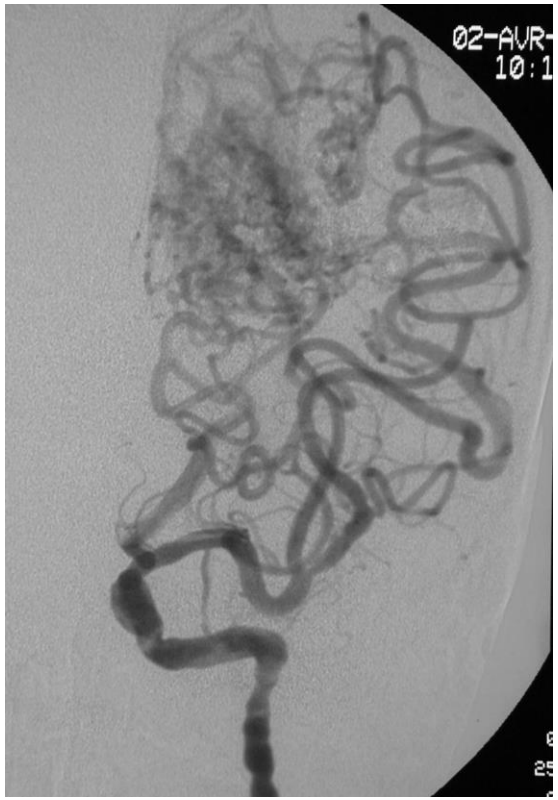
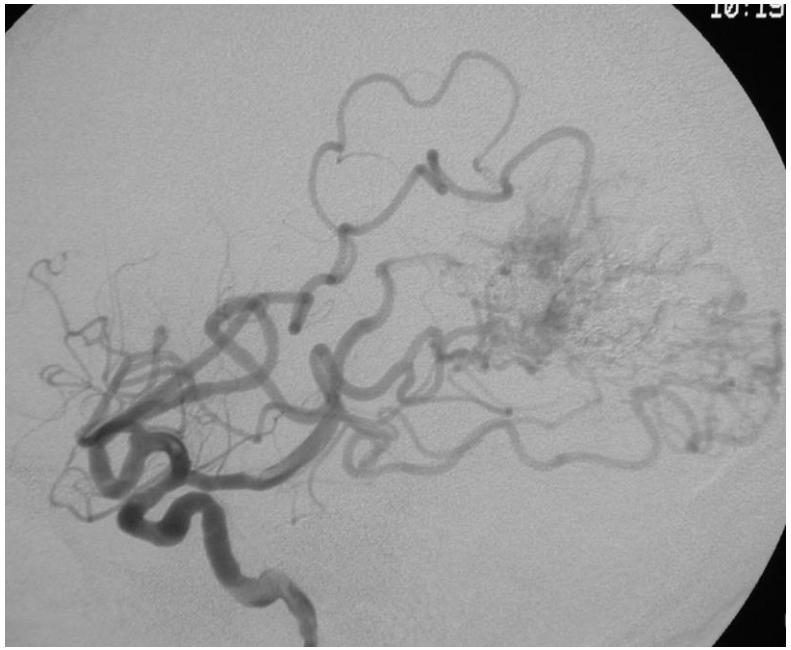
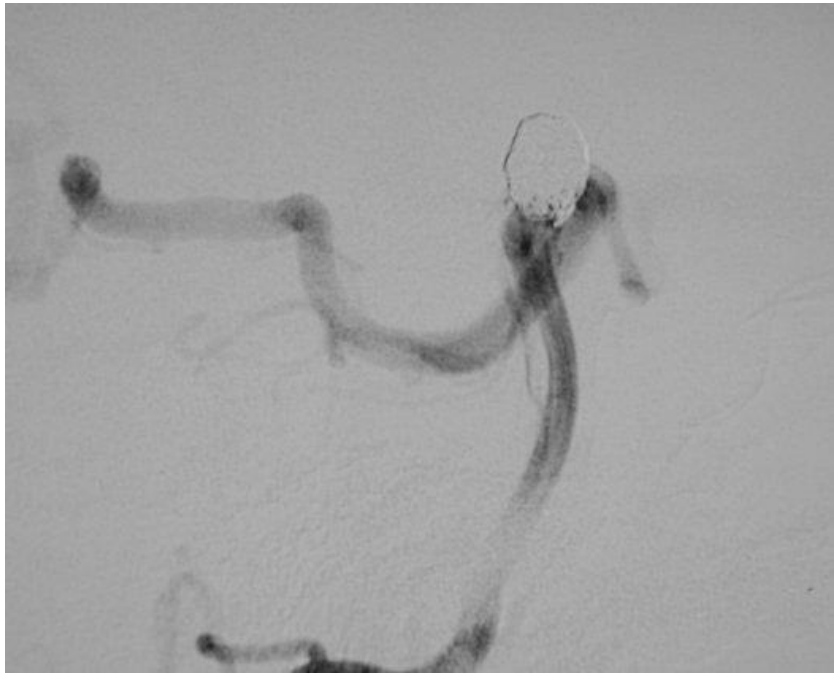


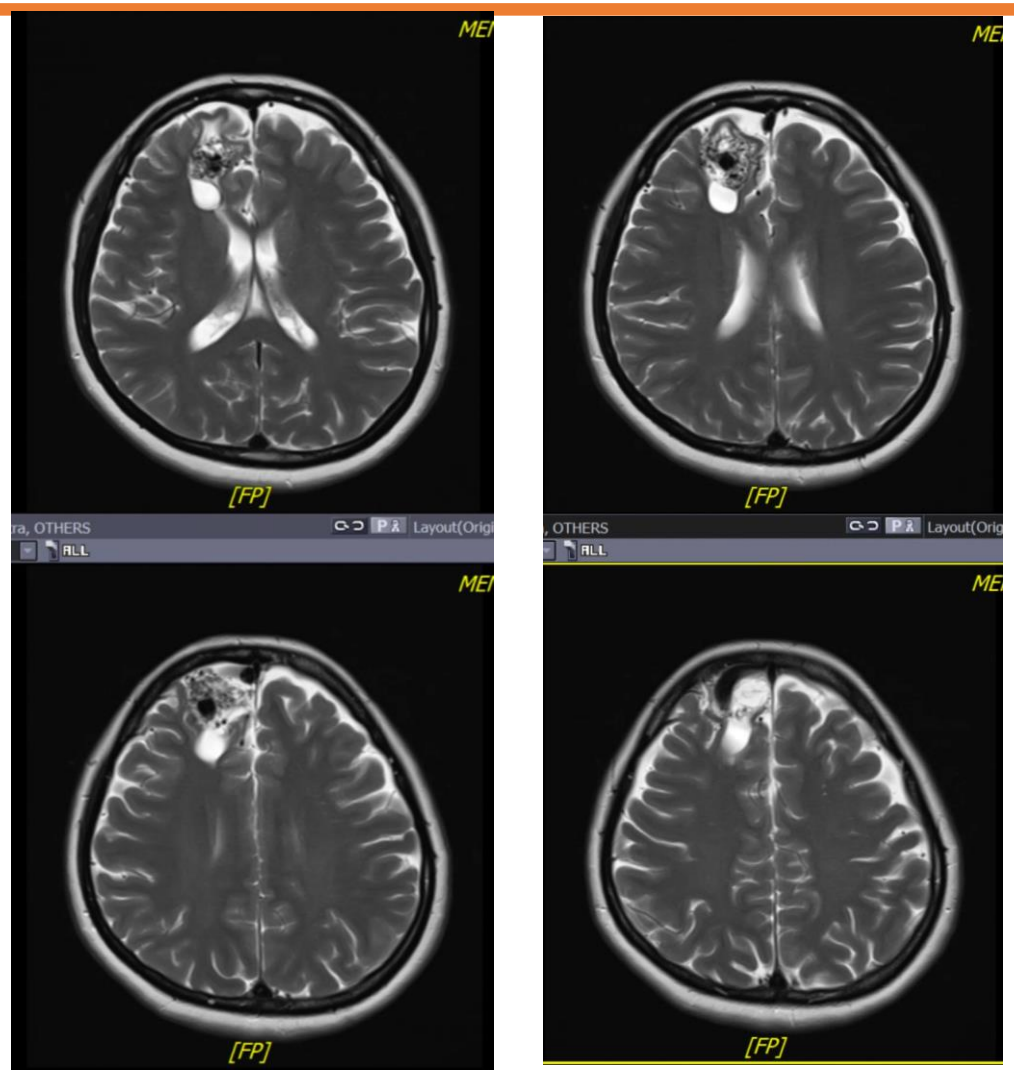





# Pre-surgical embolization of AVM

- The goal of preoperative embolization is the overall reduction in treatment risk by increasing the ease and safety of surgical resection.
- Embolization of AVM is targeted at a reduction in the size and shunt volume of the AVM, occlusion of less accessible arterial feeders (perforator and transcortical), and occlusion of intracranial aneurysms, pseudoaneurysms, and large fistulae.
- Decrease bleeding, shortening operative time, minimize the hemodynamic changes at the time of excision.

# 46F, Right F AVM



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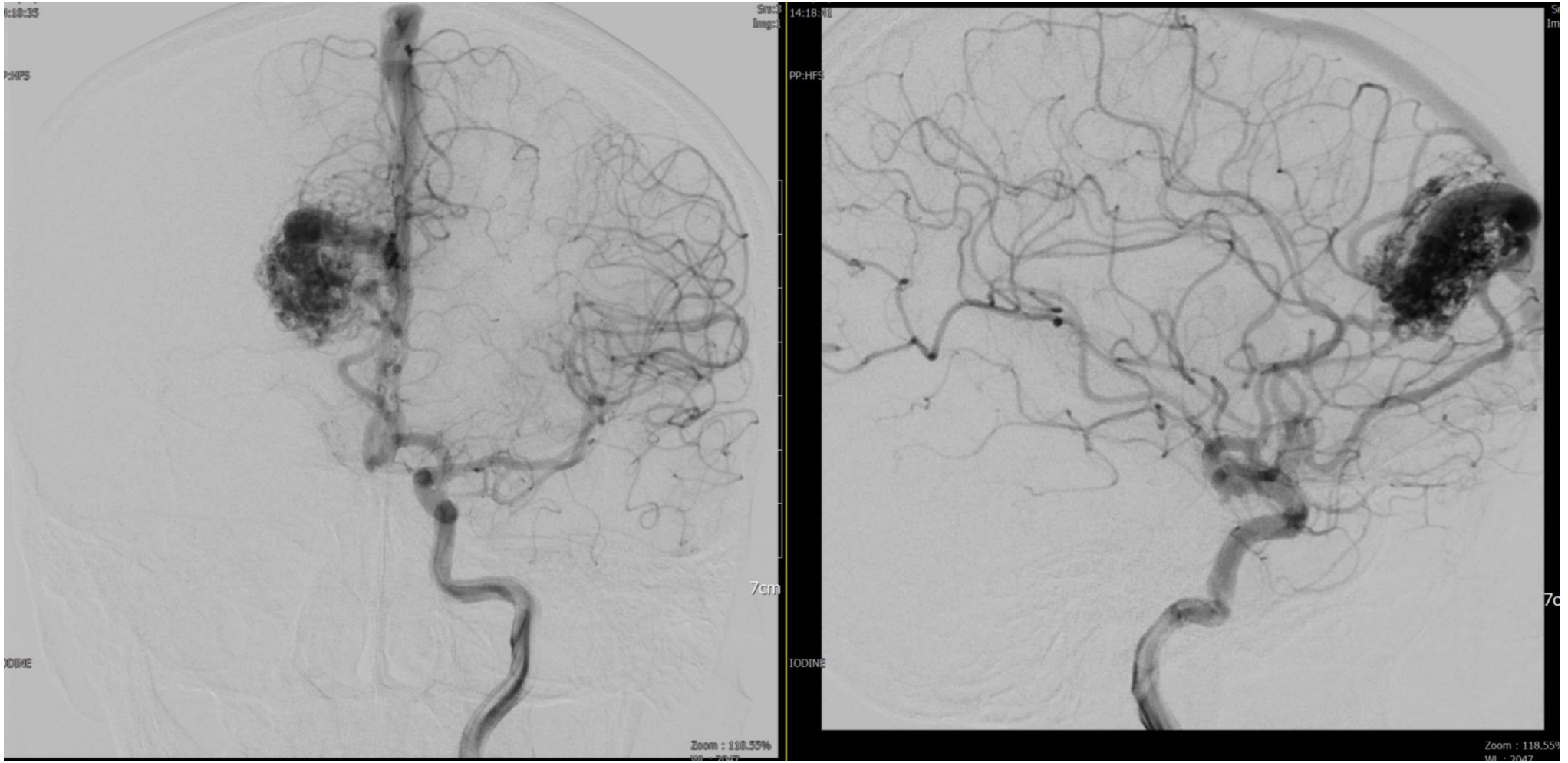

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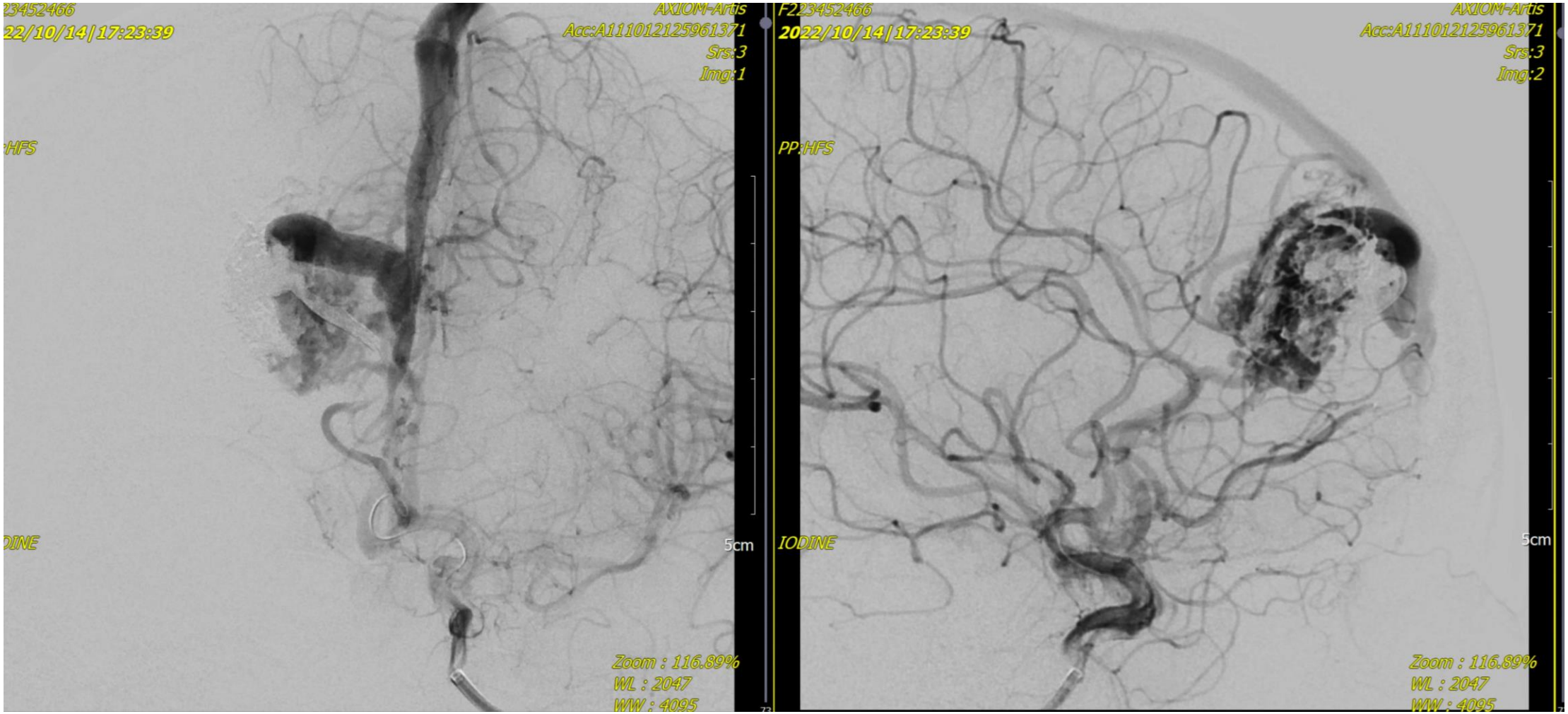

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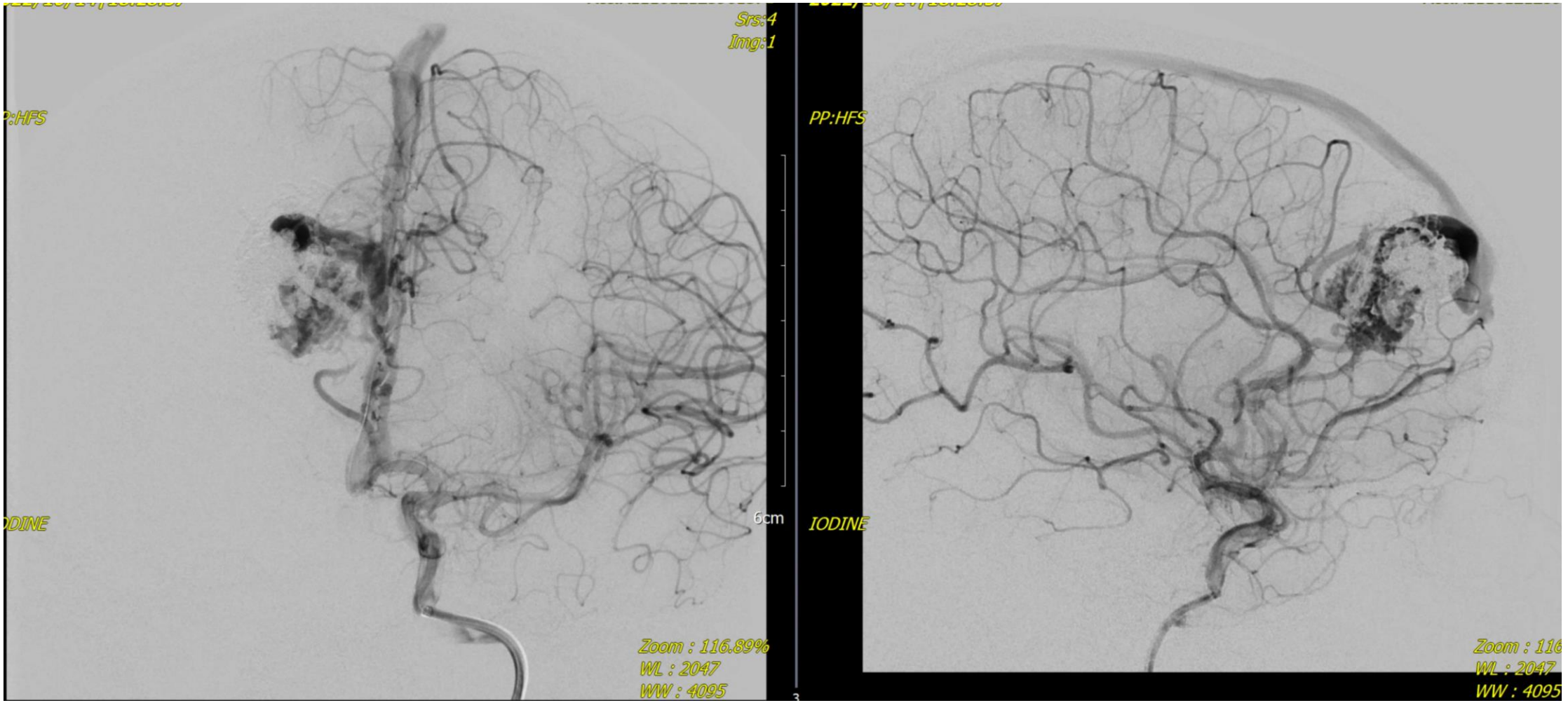

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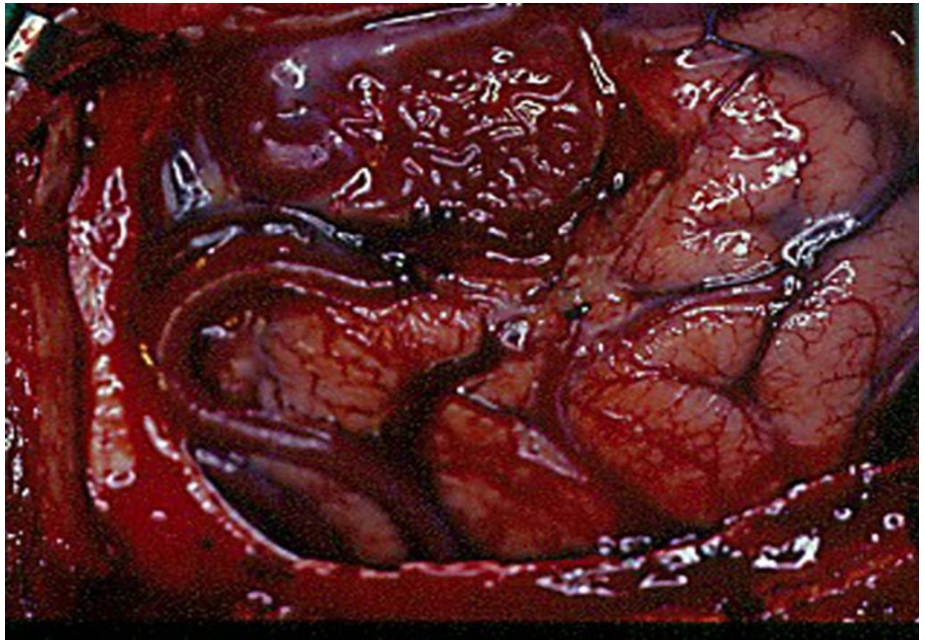

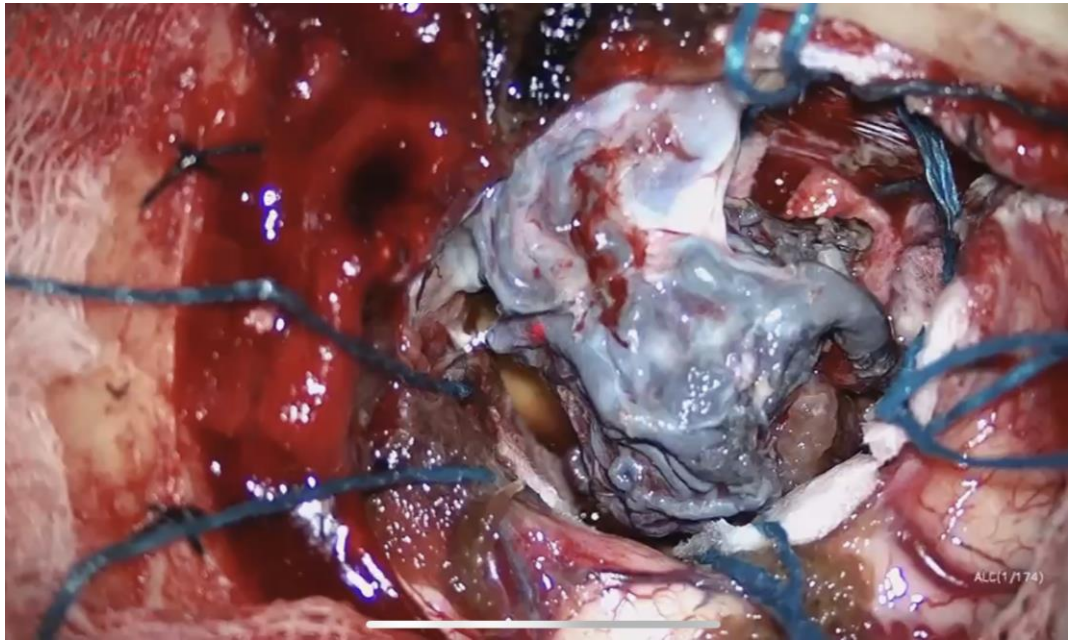

# Conclusion

- Nature course of disease、 angioarchiecture、 technique
- Clinical cure vs. morphological cure
- Invasive management: younger patients with high-risk features for an AVM rupture
- Medical management : older individuals with no high-risk features

# Thanks for your listening