LII

#SENR2024

Reunión Anual

SOCIEDAD ESPAÑOLA DE NEURORRADIOLOGÍA

7 - 9 de noviembre de 2024 • SAN SEBASTIÁN

SEDE: Hotel Barceló Costa Vasca

S.E.N.R
Sociedad Española de Neurorradiología

MALFORMACIONES VASCULARES DEL SNC DIAGNÓSTICO Y CLASIFICACIÓN

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INTRODUCCIÓN

1887 primera observación clínica de una MAVc. Pfannenstiel --lesión varicosa autopsia embarazada

1889 primera intervención quirúrgica de una MAVc. Giordano

1889 primera resección completa de una MAVc. Pean

1932-1957 500 intervenciones en pacientes con MAVc

FISIOLOGIA

Egas Moniz 1934, angiograma 3 fases (al observar el paso de contraste por el encéfalo):

*Fase arterial:1,5 sg (mas de 2,5 sg es patológico)

*Fase capilar: 0,5-1 sg

*Fase venosa: 4-5 sg

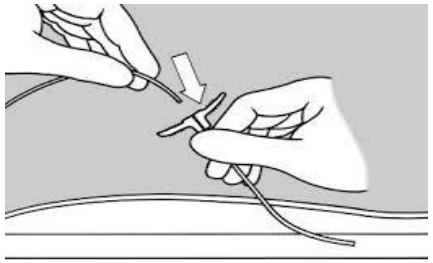
El estudio angiográfico cerebral:

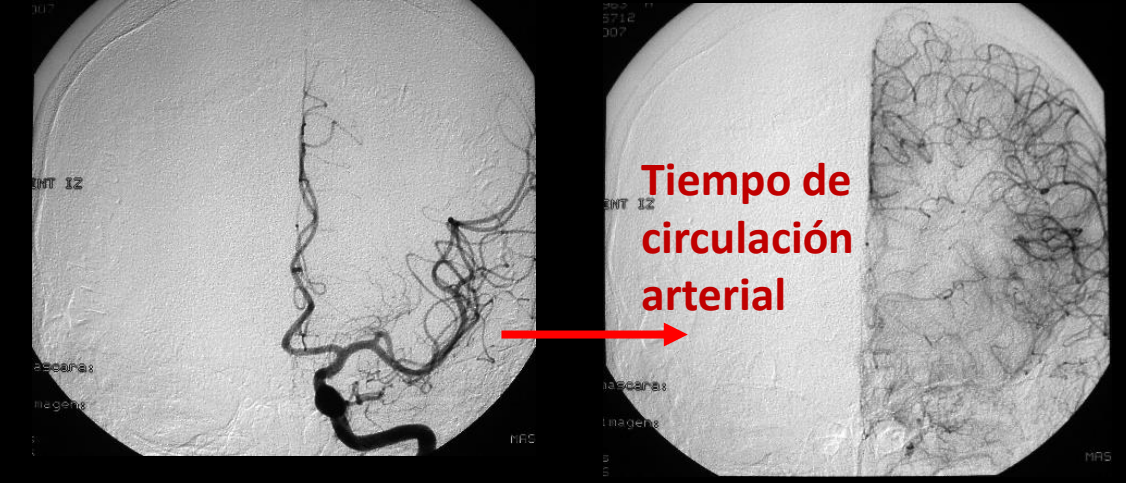
* Analisis morfológico de los vasos
* Estudio dinámico de la circulación cerebral

Egas Moniz

(António Caetano de Abreu Freire Egas Moniz; Avança, 1874 - Lisboa, 1955) Médico, político y diplomático portugués. Estudió en la Universidad de Coimbra y en 1911 obtuvo la primera cátedra de neurología en la de Lisboa. Paralelamente inició una carrera política, llegando a ser diputado de la República portuguesa, embajador de Portugal en Madrid en 1917 y presidente de la delegación lusa que asistió a la conferencia de la paz en Versalles. En 1921 volvió a dedicarse por completo a la medicina, y en 1927 estableció la técnica diagnóstica de la angiografía cerebral para la detección de tumores. También realizó las primeras arteriografías e introdujo la leucotomía prefrontal en el tratamiento de algunas enfermedades mentales.

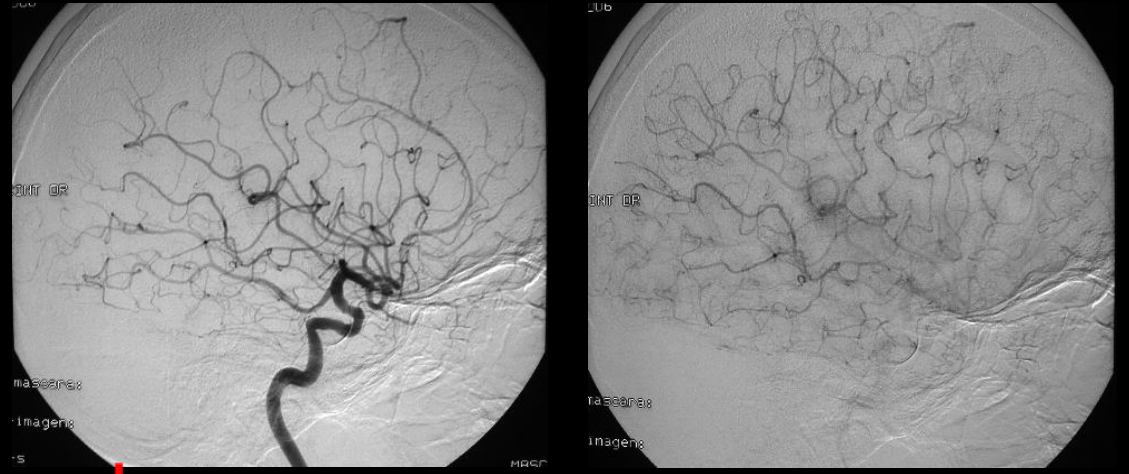
1953 **Seldinger** (punción-cateterismo con guía en la arteria femoral) facilita la cateterización de los vasos cerebrales y permite mejorar el tratamiento endovascular de las MAVc.

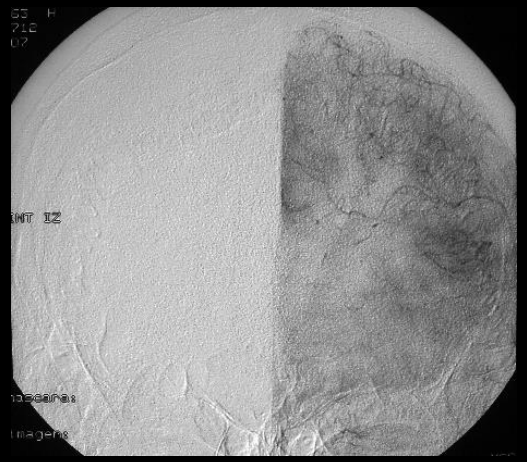




Fase arterial

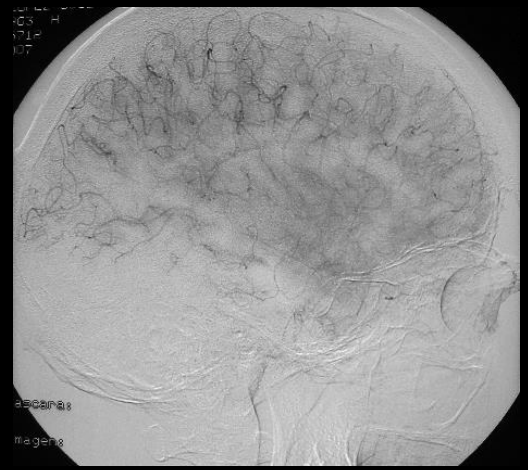
Tiempo de circulación arterial

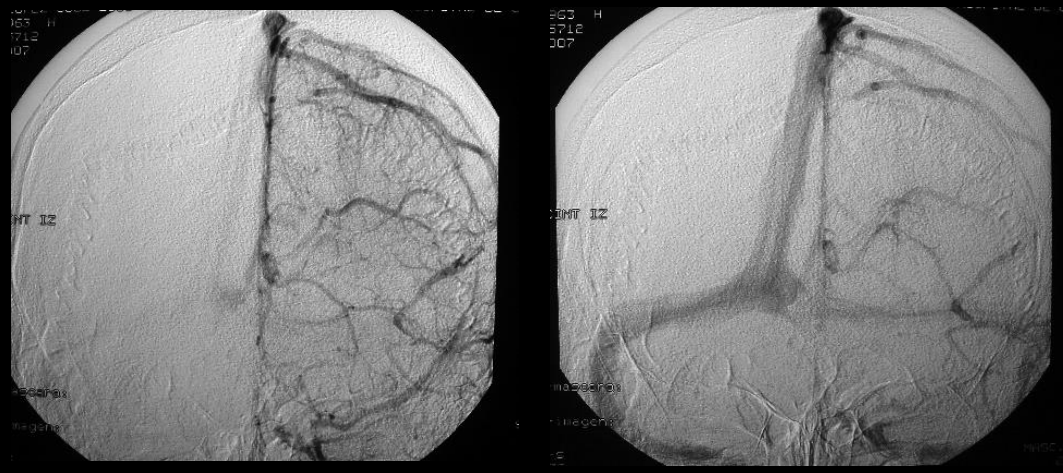




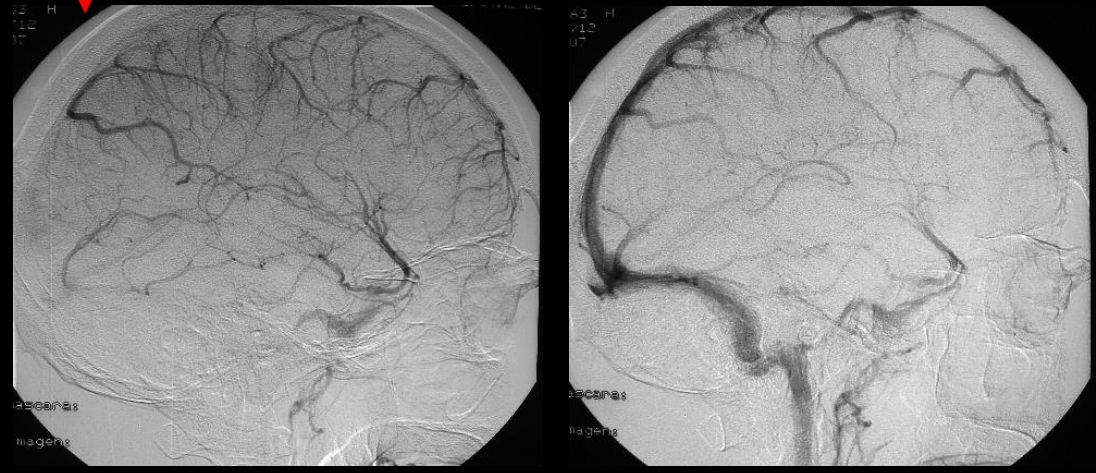
Fase capilar

Tiempo de circulación arteriovenosa





Fase venosa

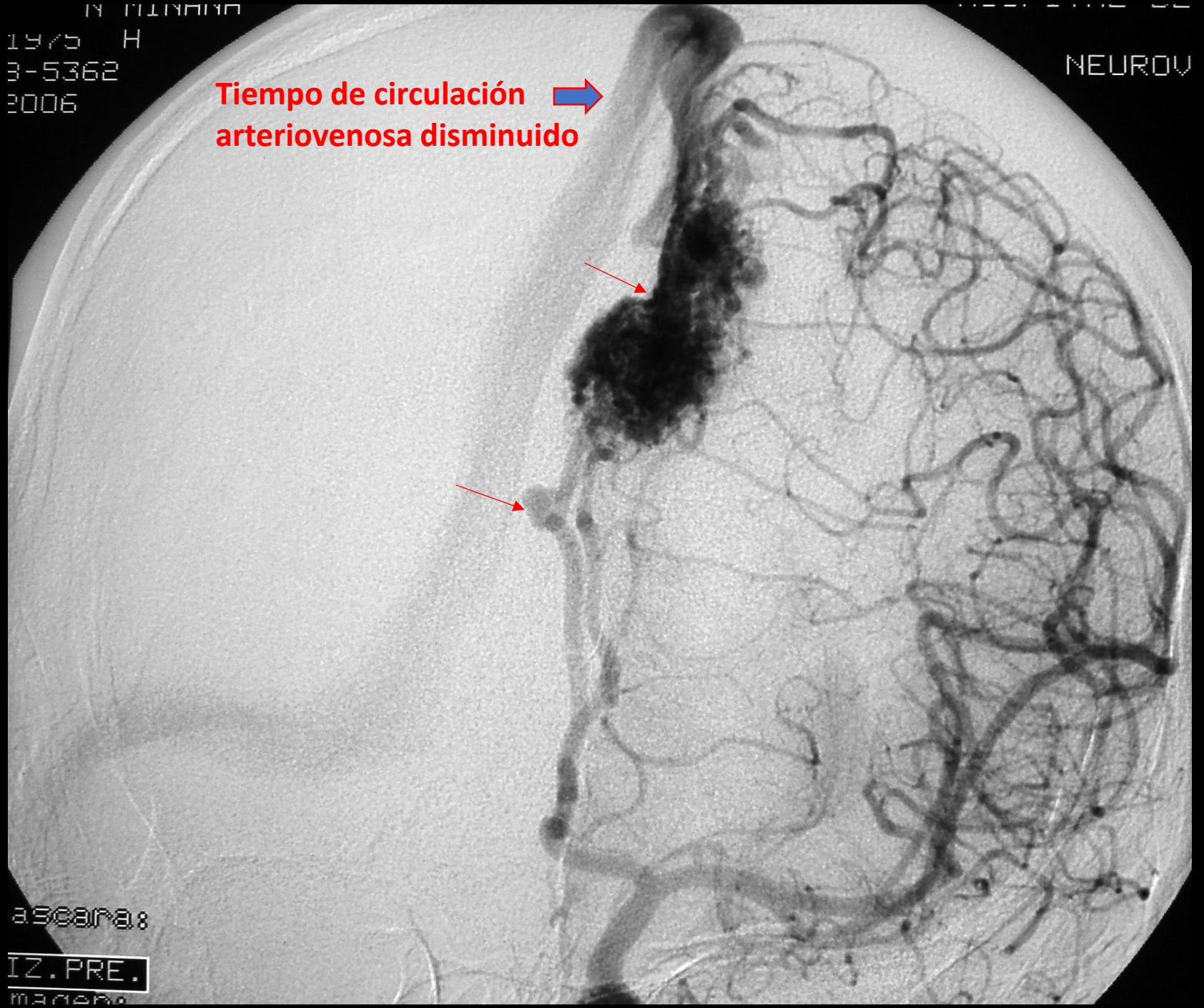


FISIOLOGIA

***__**Tiempo de circulación arterial**__: concentración de contraste en el sifón carotídeo---llenado de las ramas arteriales terminales = 1,5 segundos

***__**Tiempo de circulación arteriovenosa**__: concentración máxima de contraste en el sifón carotídeo---concentración máxima en venas parietales= 4,37+-0,83 segundos

- Aumenta el tiempo: Hipertensión, trombosis arterial, LOE
- Disminuye el tiempo: Fistula o MAV, infarto-perfusión de lujo, vasodilatación

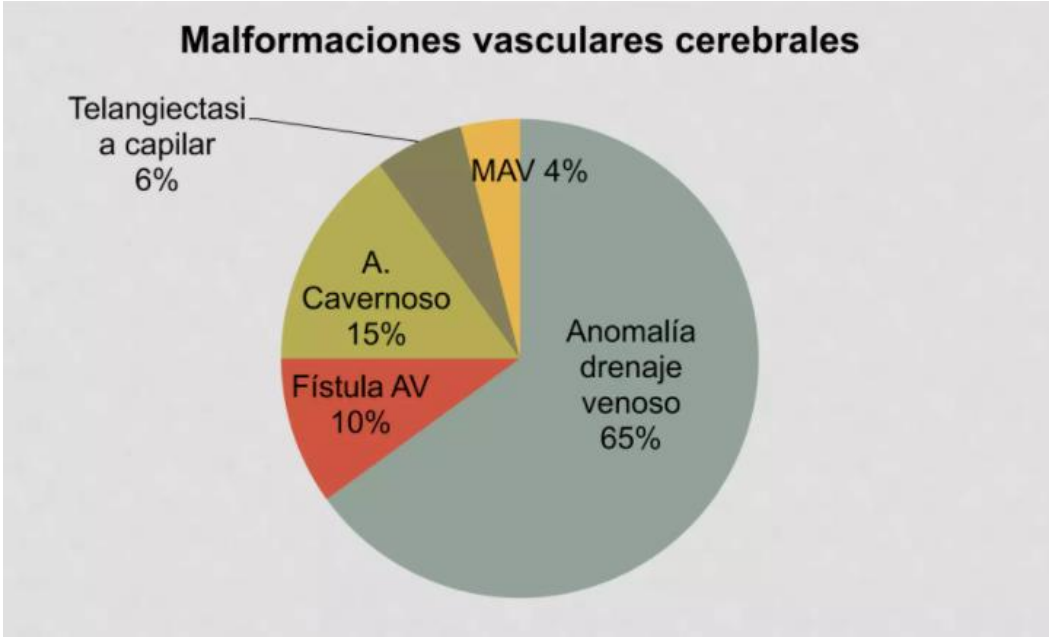

Tiempo de circulación
arteriovenosa disminuido
NEUROV

CLASIFICACIÓN

Mc Cormick 1960, modificada 1984

Sin shunt arteriovenoso y bajo flujo:

- ***Angioma Venoso (anomalía del desarrollo venoso)**
- ***Angioma Cavernoso (cavernoma)**
- ***Angioma Capilar (telangiectasia capilar)**
- ***Sinus pericranii**



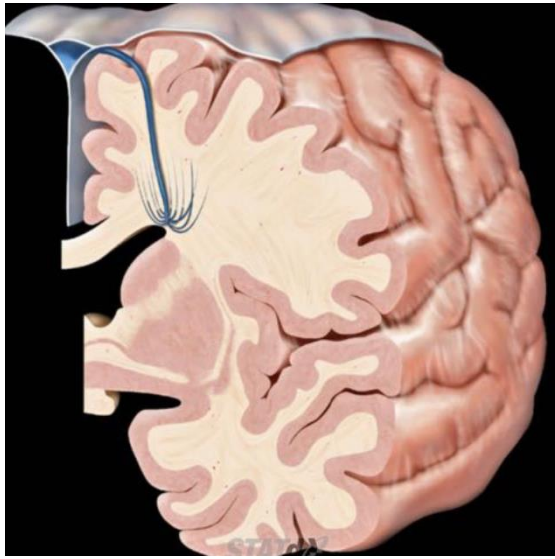
Con shunt arteriovenoso y alto flujo:

- ***MAV parenquimatosas**
- ***Fistulas AV piales y durales(DAVF)**
- ***Angiopatía cerebral proliferativa**

Anomalía venosa del desarrollo. Angioma Venoso 60%

- Grupo de venas medulares aumentadas de tamaño de disposición radial, separadas por parénquima normal, que convergen en una vena colectora dilatada

"Cabeza de Medusa"



- Asintomáticos, salvo si asocian otras malformaciones

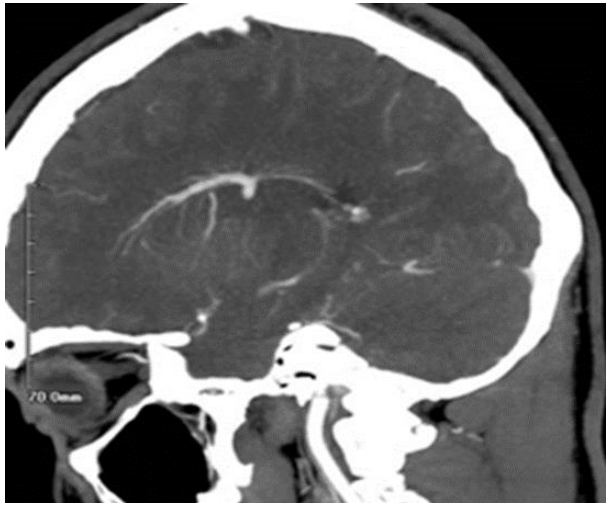
- Localización: sustancia blanca profunda cerebral/cerebelosa, cerca del ángulo ventricular.

Anomalía venosa del desarrollo. Angioma Venoso

TC

SinC: normal

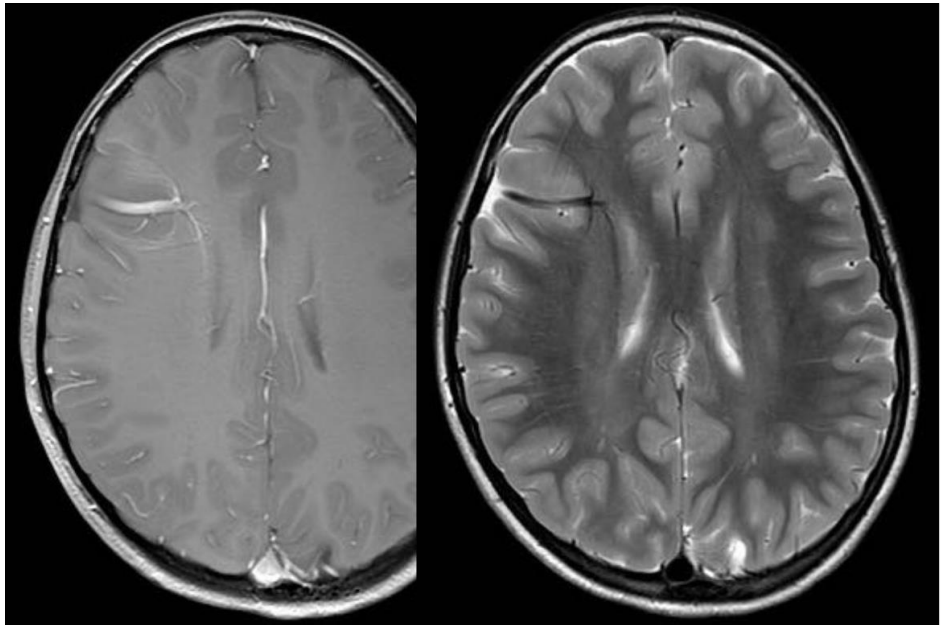
ConC: focos lineales de realce convergiendo en una vena de drenaje dilatada



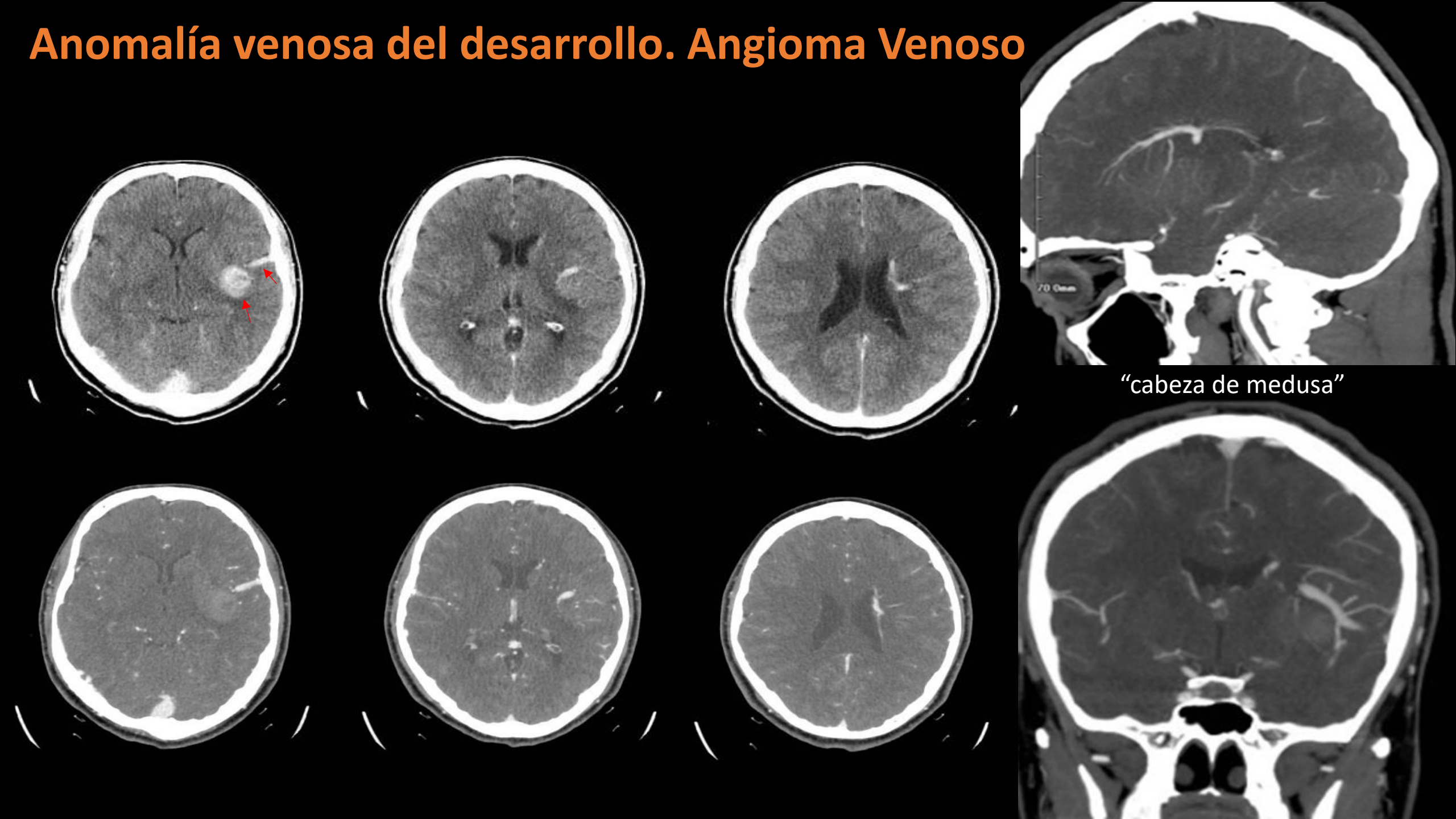
RM

T2 vacío de señal

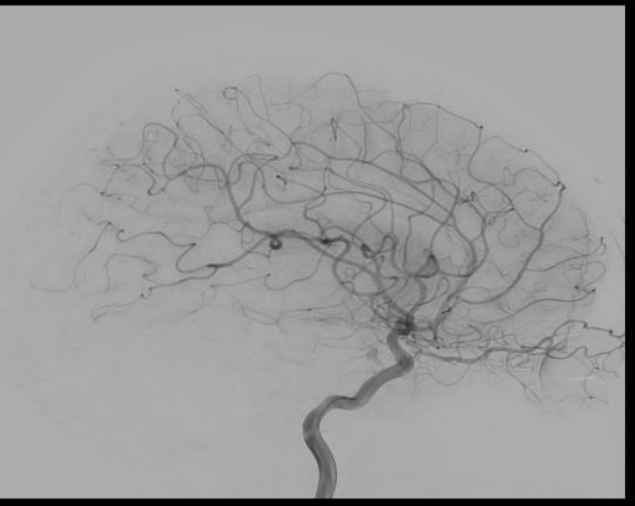
Realce intenso con Gadolinio

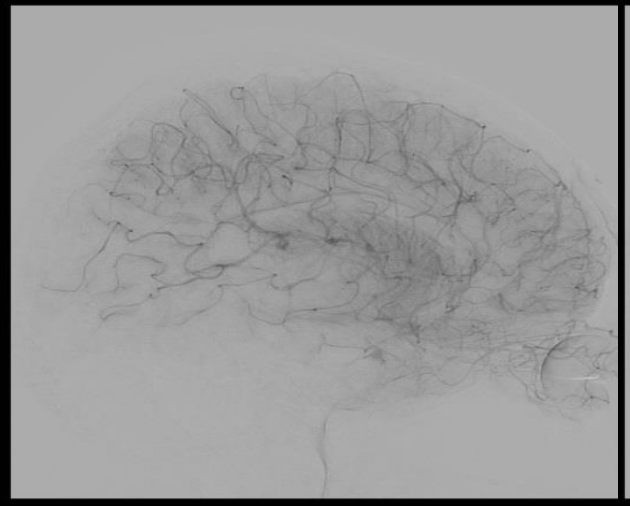


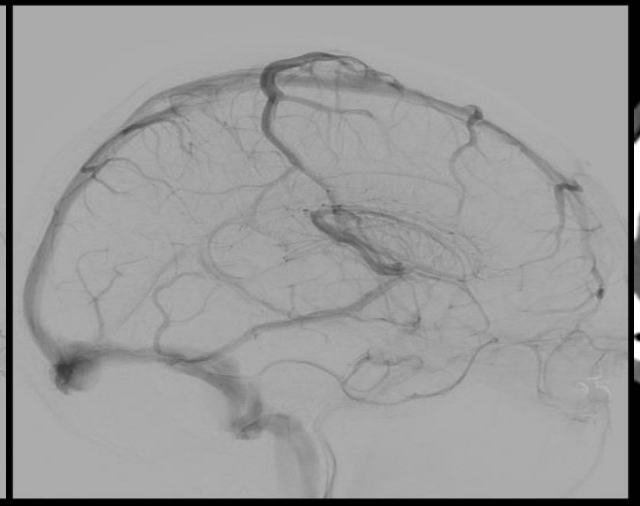
Anomalía venosa del desarrollo. Angioma Venoso

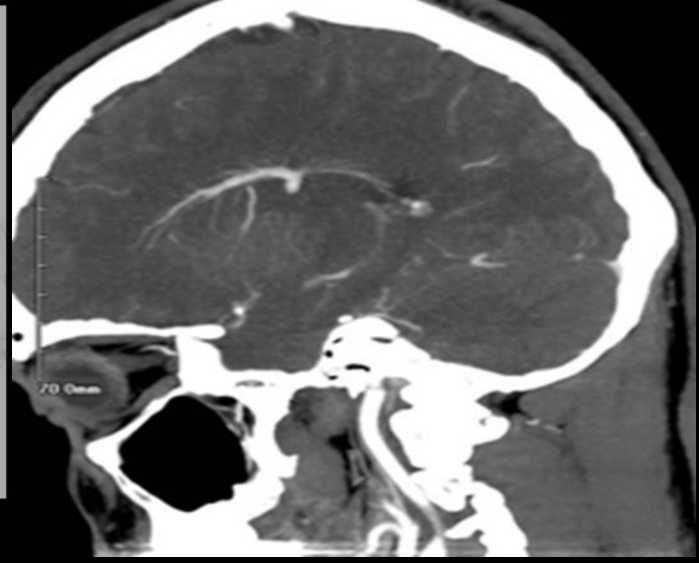


"cabeza de medusa"







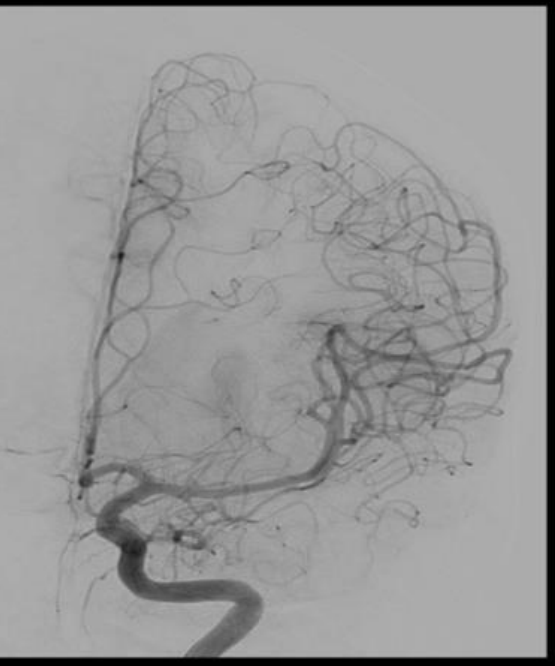


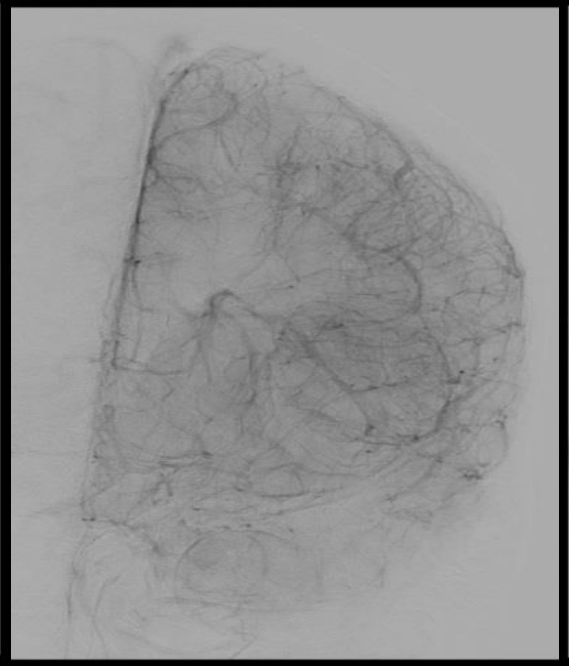
Fase arterial

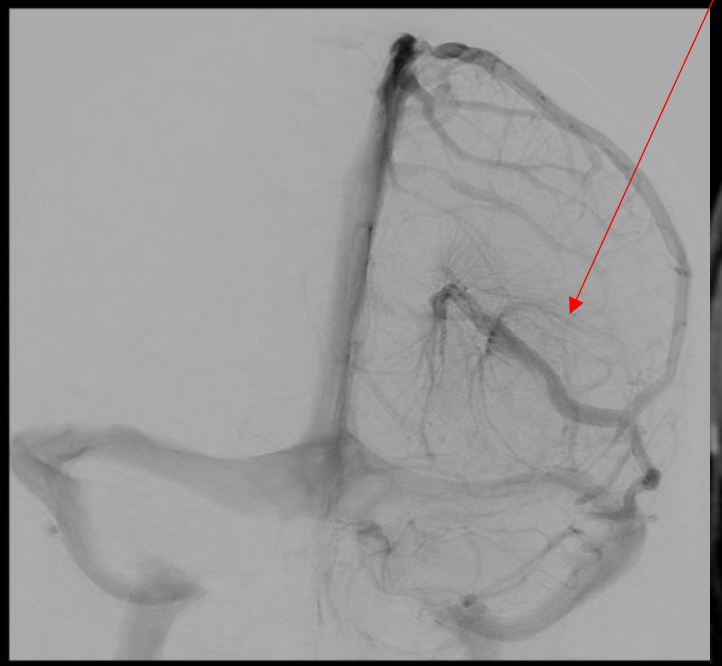
Fase capilar

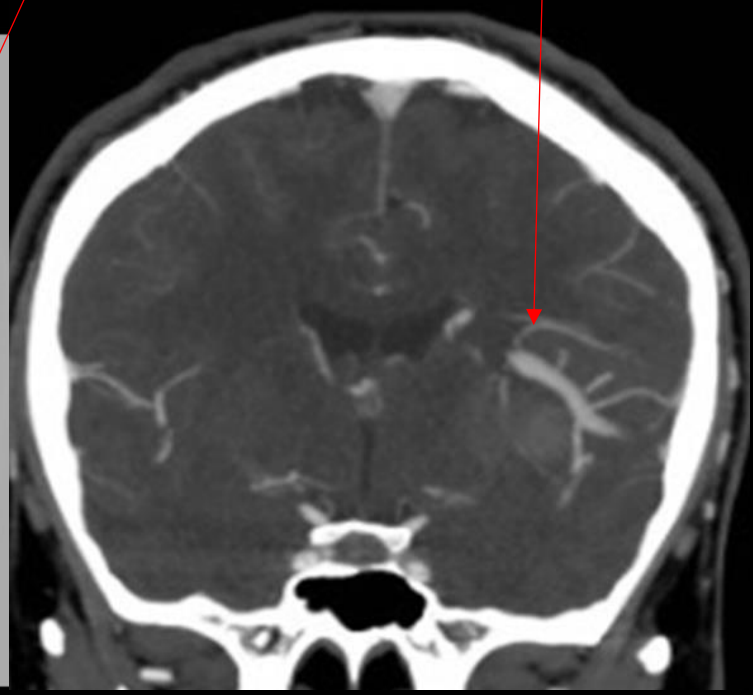
Fase venosa

Venas medulares dilatadas
Vena colectora ensanchada

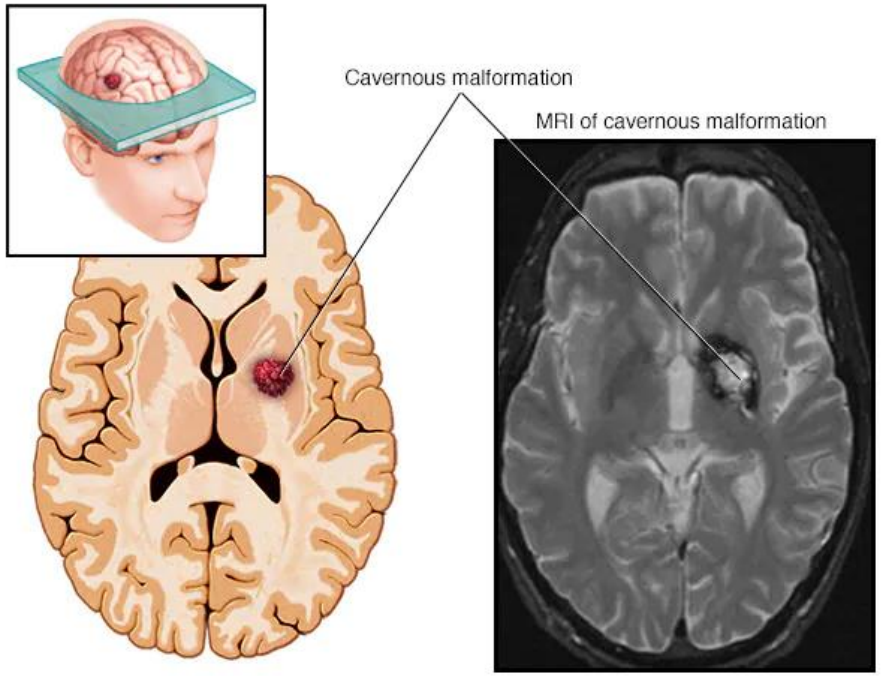






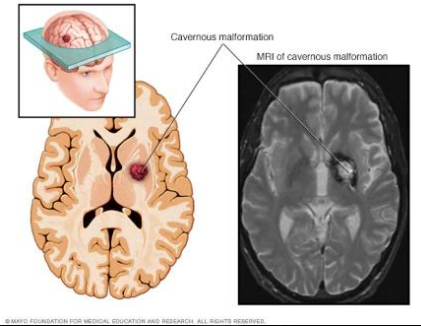


Cavernoma-Angioma Cavernoso 8-15%



- Espacios sinusoidales tapizados de endotelio sin encéfalo intercalado, morfología de morula
- Angiográficamente ocultos
- Tamaño, 2 mm----varios cm
- Clínica hemorragia (0,5-1% anual), convulsiones, DNF, cefalea

Cavernoma-Angioma Cavernoso 8-15%

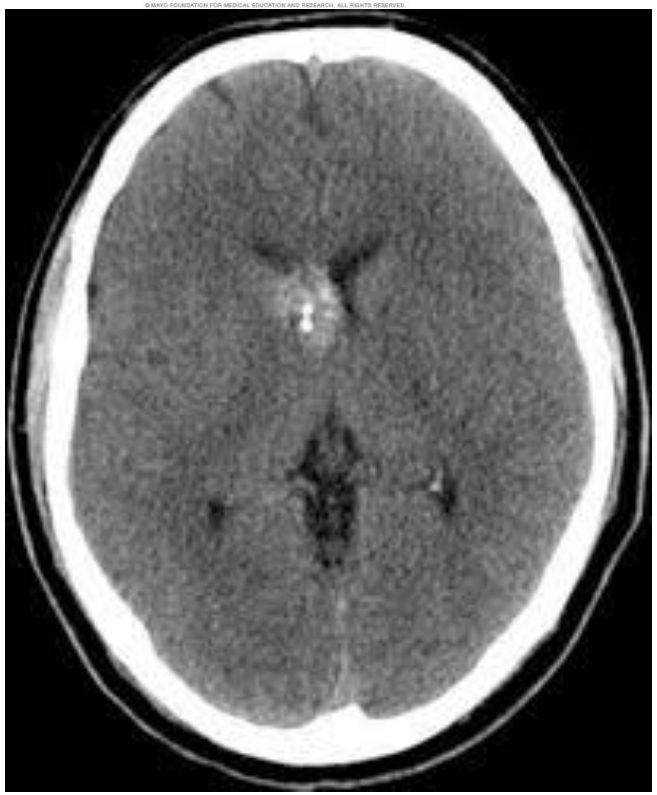


•TC SinC:

Lesiones focales ovoideas.

Iso/moderadamente hiperdensos, calcificaciones frecuentes.

Sin efecto masa salvo hemorragia reciente.



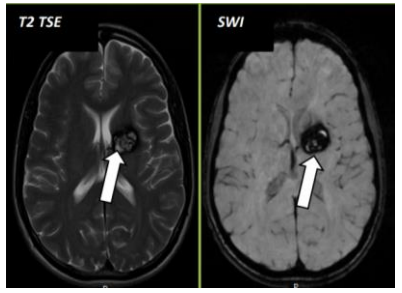
•TC ConC:

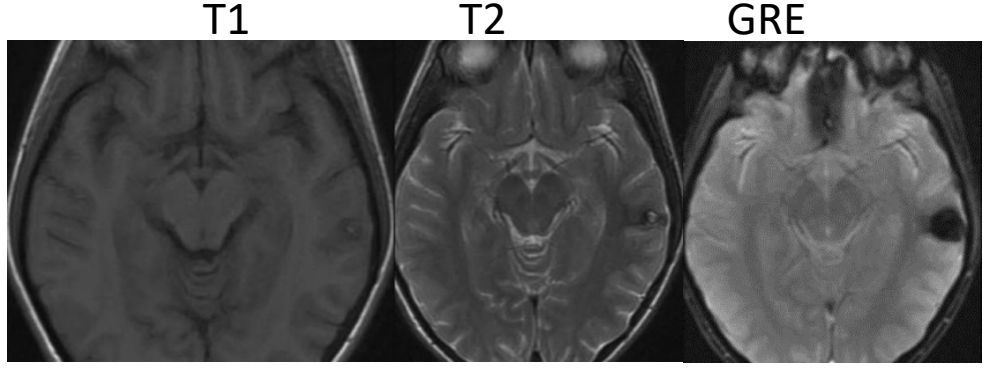
Variable (nulo - escaso - intenso realce)

Cavernoma-Angioma Cavernoso 8-15%

RM

•Varia, dependiendo de hemorragia aguda/crónica, depósito periférico de hemosiderina

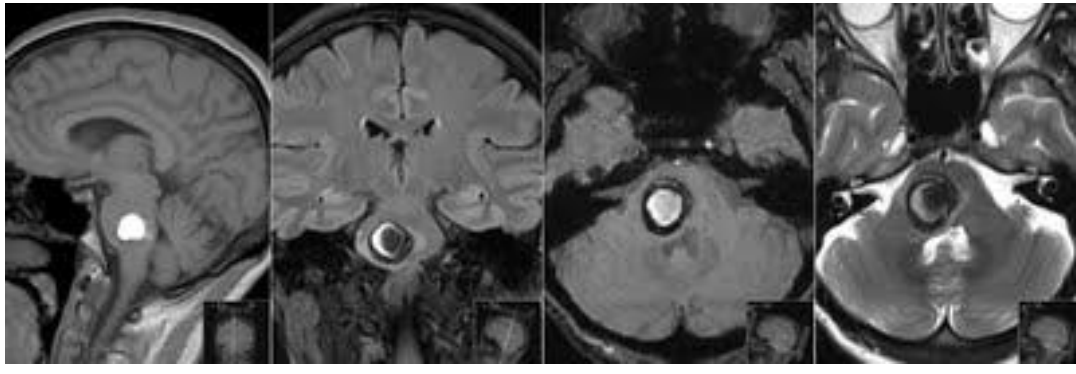




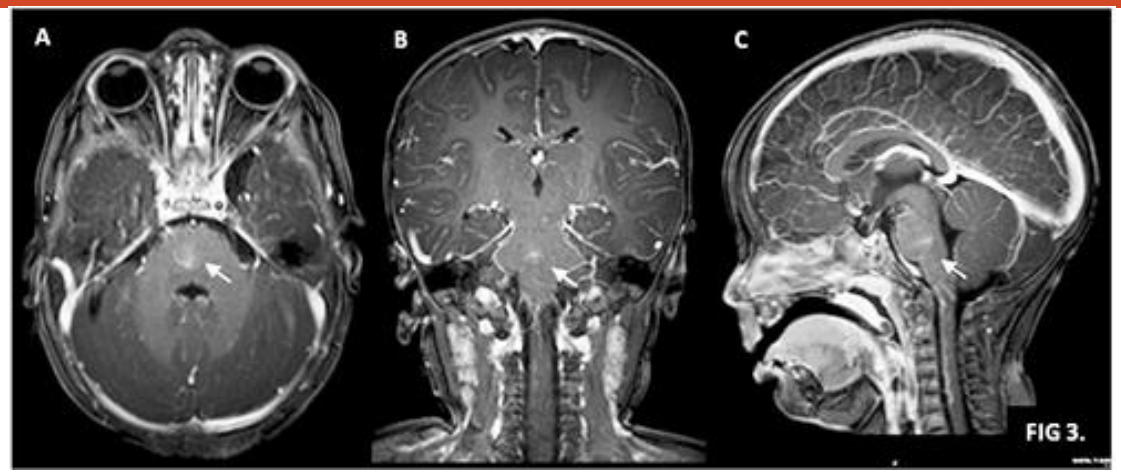
•Lesión “en palomitas de maíz”:

- ✓Márgenes precisos, anillo hipointenso
- ✓Núcleo de intensidad mixta heterogéneo
- ✓Loculaciones de sangre con niveles líquido-líquido

•Realce mínimo o ausente



Telangiectasias capilares 15-20%



- Asintomática, hallazgo incidental en autopsias.

- Colección de capilares dilatados sin fibras musculares lisas, con encéfalo normal intercalado.

- Mesencéfalo, Protuberancia, Bulbo, Médula espinal

- Hasta 2/3 partes asocia una vena colectora dilatada

Telangiectasias capilares15-20%

Pasa desapercibida en TC y RM sin contraste

•TC C

Normal. A veces débil realce.

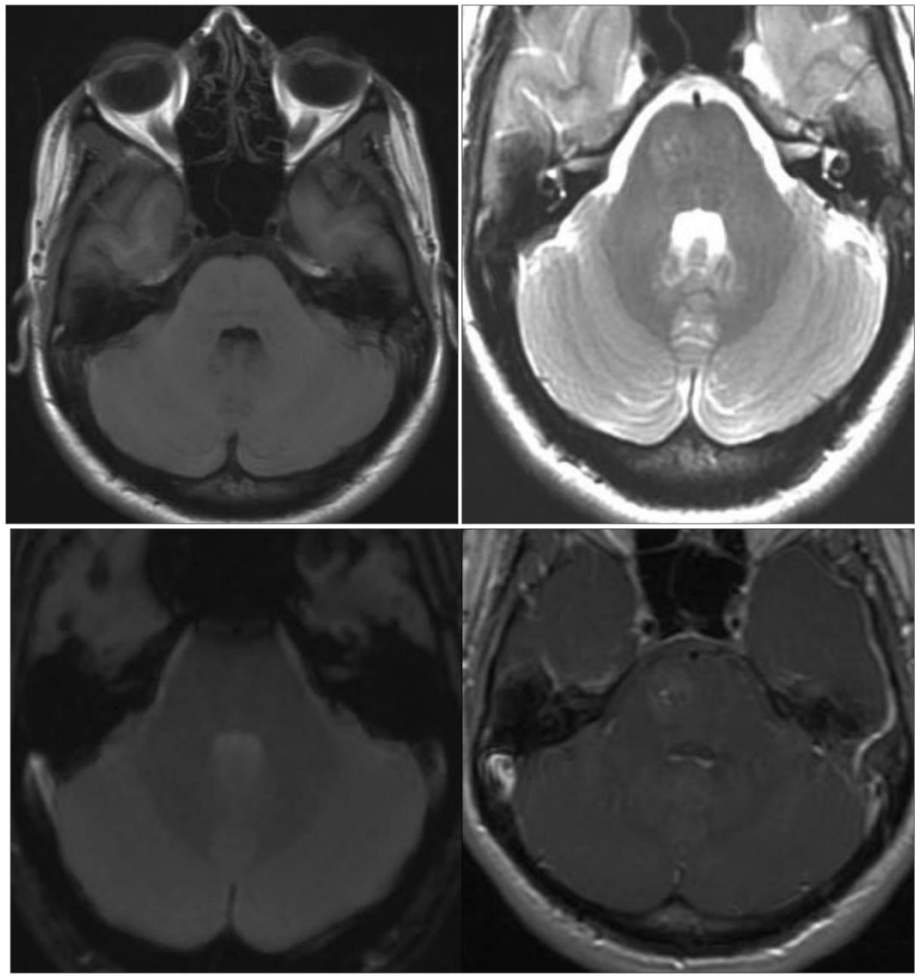
•RM

T1 normal

T2 y FLAIR 50% hiperintensas

T2*GRE pueden ser hipointensas (por desoxihemoglobina, flujo lento)

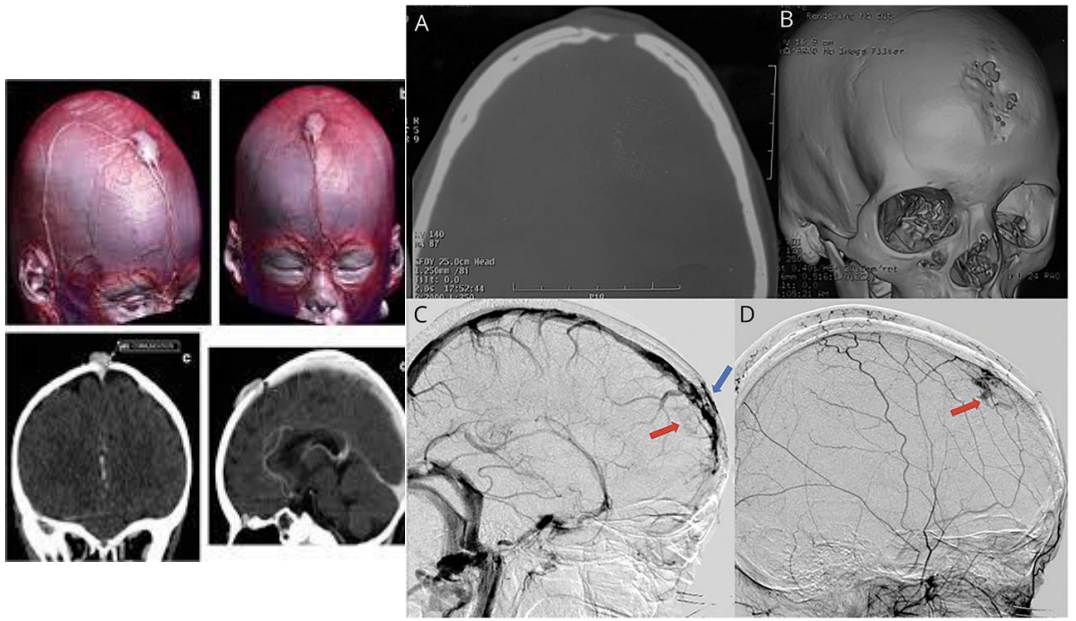
Con Gd tenue realce en forma de “cepillo”, pequeños vasos lineales ramificados.



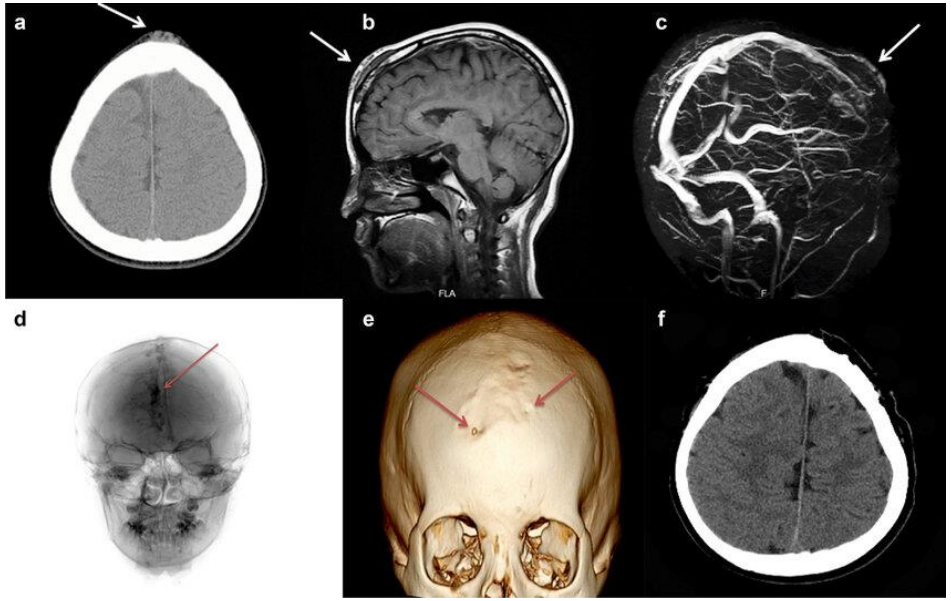
Sinus Pericranii

Comunicación venosa directa entre senos durales y venas epicraneales dilatadas que fluctuan con Valsalva

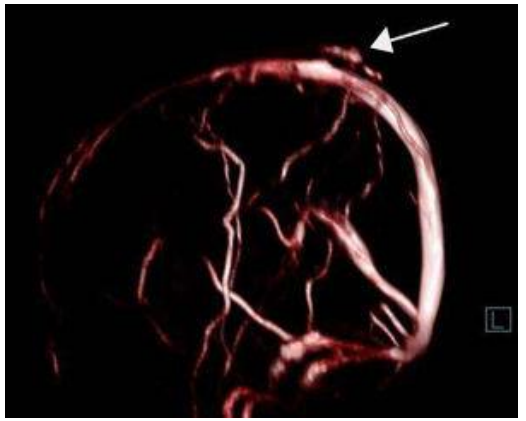
Inflamación de tejidos blandos con defecto óseo



Lesión subgaleal en comunicación con el senoSS



Se recomienda tratamiento conservador



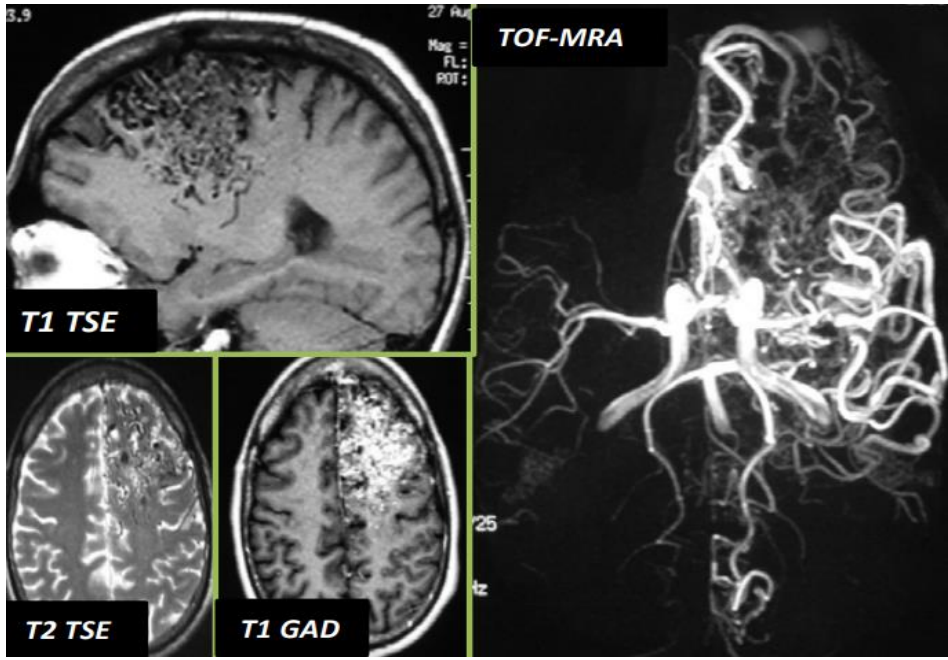
Angiopatía cerebral proliferativa

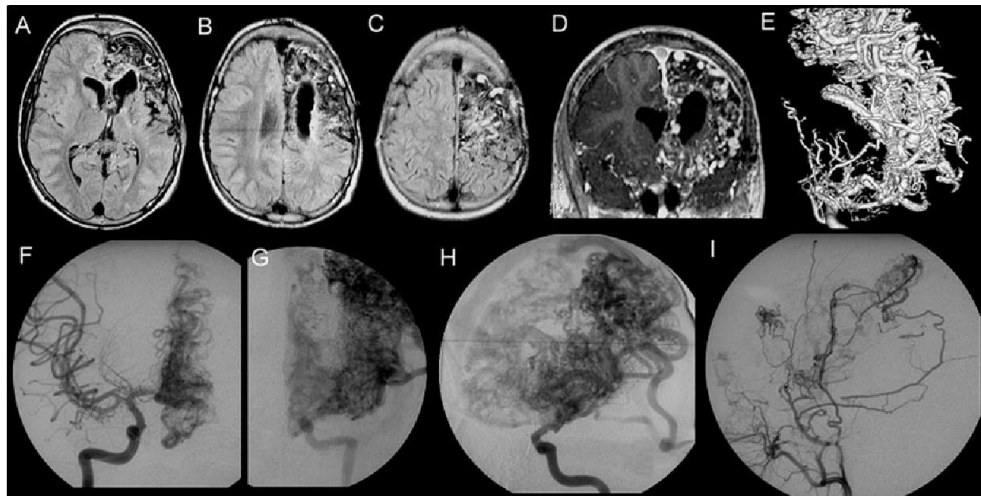
- Mujeres jóvenes con convulsiones, dolor de cabeza y DNF

- Red difusa de espacios vasculares realzados, con parénquima cerebral normal entrelazado (Dº diferencial MAVc), cubriendo un hemisferio o varios lóbulos.

La aferencia transdural es frecuente.

- El término "proliferativa“, continuo crecimiento---"formación de nuevos vasos sanguíneos“, que agravan los síntomas



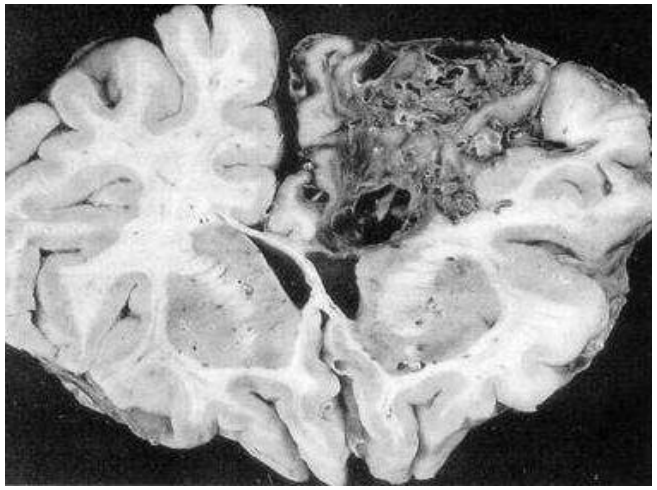


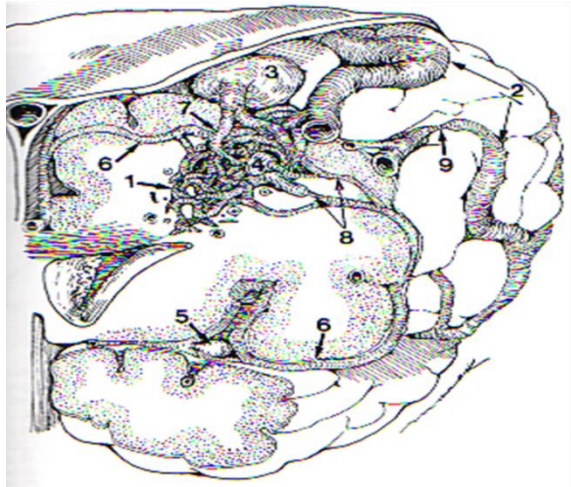
MAV PARENQUIMATOSAS

- Conexiones anómalas directas arteriovenosas, arteriolovenosas o arteriolovenulares, sin lecho capilar, conformando un nido de alto flujo epicéntrico con reclutamiento vascular, arterialización de los drenajes venosos, gliosis, angiopatía de flujo

- Entre los vasos anómalos no hay parénquima cerebral normal

- Susceptibles de hemorragia por rotura de vasos nidales, aneurismas u obstrucción venosa





ETIOPATOGENIA Y EPIDEMIOLOGÍA

La anomalía desencadenante de la MAVc sigue siendo controvertida.

- Errores congénitos, desarrollo anómalo embriológico del sistema vascular
- MAVc de novo, observado angiográficamente

Incidencia 1-2/100.000 habit

Prevalencia 10-18/100.000 habit

Sexo sin predilección

Edad pico 20-40 años

LOCALIZACION

HEMISFÉRICAS

-PARIETAL	27%
-FRONTAL	22%
-TEMPORAL	18%
-OCCIPITAL	5%

FOSA POSTERIOR

-CEREBELOSAS	5%
-TE	2%

INTRAVENTRICULARES	18%
OTRAS	3%

CLÍNICA

Asintomáticas 15%

El 80% se hacen sintomáticas entre los 20-40 años

Convulsiones 18%-40%

Crisis generalizadas +.Tratamiento anticomicial.

Déficit neurológico 1%-40%

Fenómeno de robo, HT venosa.

Cefalea 5-14%

CLÍNICA

Hemorragia intracraneal 38%-71% (6% de los ACV hemorrágicos)

75% <50 años. Parenquimatosa 41%, Subaracnoidea 24%, Intraventricular 12%

Mortalidad 15-20%, morbilidad 45-80%

Factor predictor independiente de futura hemorragia, 18%(1º año)---3,2% disminuye el riesgo

Riesgo anual de hemorragia 2-4%. 1-33% si factores de riesgo:

-Rotas 3,2% (18% 1º año)
-Profundas 2,4%
-Drenaje venoso profundo 2,4%
-Estenosis venosa
-Aneurisma extranidal(5,3%)
-Aneurisma intranidal (9,8%)
-Infratentorial
-Periventricular

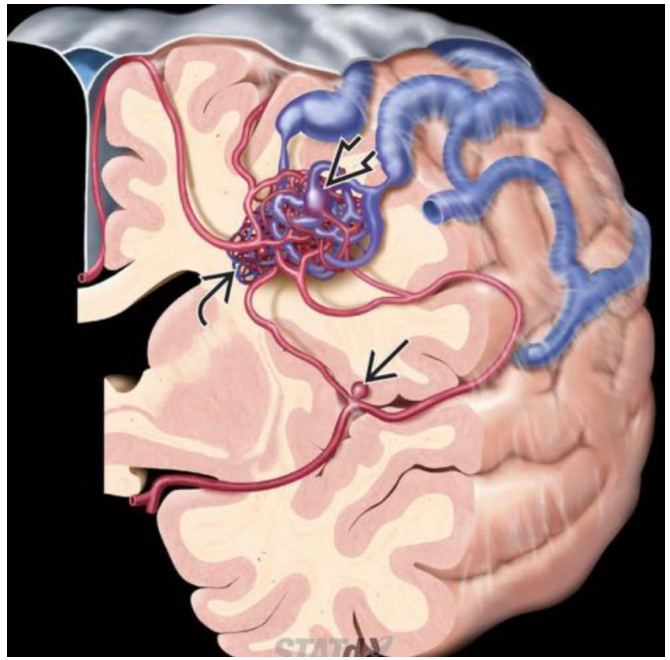
1FR 3-5%
2FR 8-15%
3FR >30%

ANEURISMAS Y MAV

Formados por stress hemodinámico

Intranidales(nido)---HP

Extranidales(arterias aferentes)---HSA



Empeoran el <u>pronóstico</u>

Se correlacionan de manera significativa con la <u>presentación hemorrágica</u>

CLASIFICACIÓN Spetzler y Martin 1986
ESCALA DE GRADACIÓN Y RIESGO DE TRATAMIENTO QUIRÚRGICO

Clasificación de Spetzler-Martin pondera 3 elementos (tamaño, elocuencia y drenaje venoso), estableciendo 5 grados de morbimortalidad neuroquirúrgica

Características		Puntos asignados
Tamaño de la MAV	Pequeña (< 3 cm)	1
	Mediana (3-6 cm)	2
	Grande (> 6 cm)	3
Localización	No elocuente	0
	Elocuente	1
Patrón de drenaje venoso	Superficial únicamente	0
	Profundo	1

***Cerebro elocuente**: sensitivomotora, lenguaje y corteza visual; hipotálamo y tálamo; cápsula interna; tronco cerebral; pedúnculos cerebelosos; núcleo cerebeloso profundo*

Se establecen 5 grados según riesgo quirúrgico

- **Grado I y II** morbimortalidad 0-5%
- **Grado III** resultados dispares 15-25%
  - **IIIa** corticales
  - **IIIb** profundas o **VI**
- Grado **IV y V** morbimortalidad 30-50%. Se recomienda tratamiento conservador.

Mayor grado mayor riesgo de tratamiento quirúrgico(no valora el riesgo de tratamiento endovascular o radiocirugía)

DIAGNOSTICO MAVc

Gold standard --- Angiografía Cerebral

La evolución de técnicas de imagen no invasivas mejoran el diagnóstico

CT (angio-CT)

RM (angio-RM)

Angiografía (DSA)

Tamaño	Localización	Aneurismas intranidales o de flujo	Drenaje venoso	Estenosis en venas de drenaje

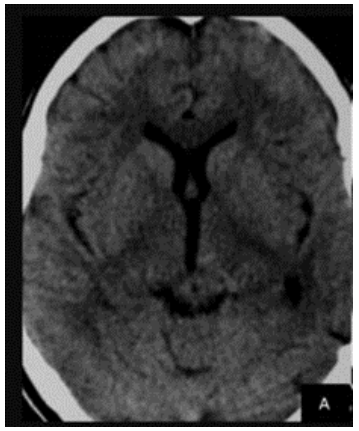
TAC MAVc

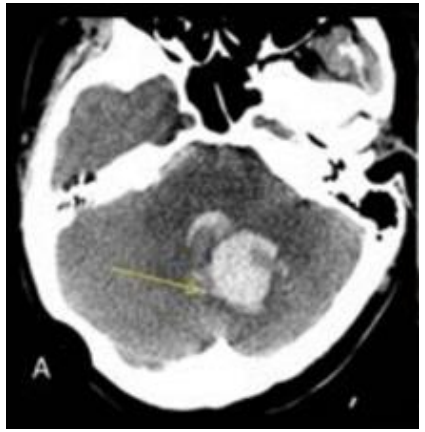
- **TC sin contraste**:

- Vasos iso/ligeramente hiperdensos

- 25-30% Ca++

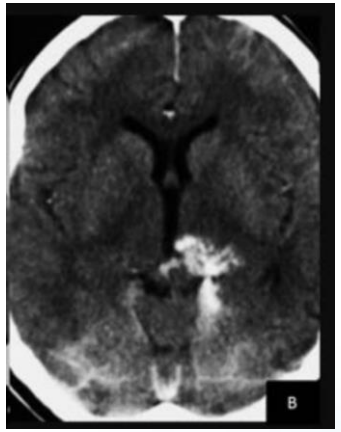
- Sangrado intraparenquimatoso, intraventricular, HSA

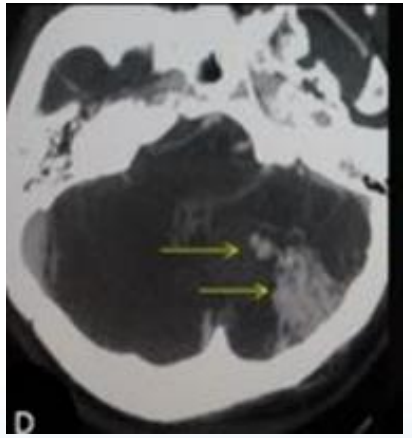


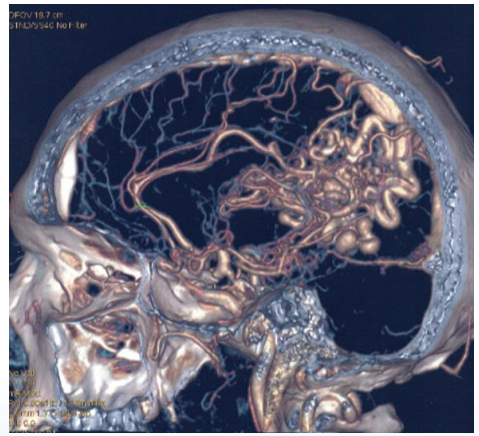


- **TC con contraste**:

- Vasos serpiginosos hipercaptantes





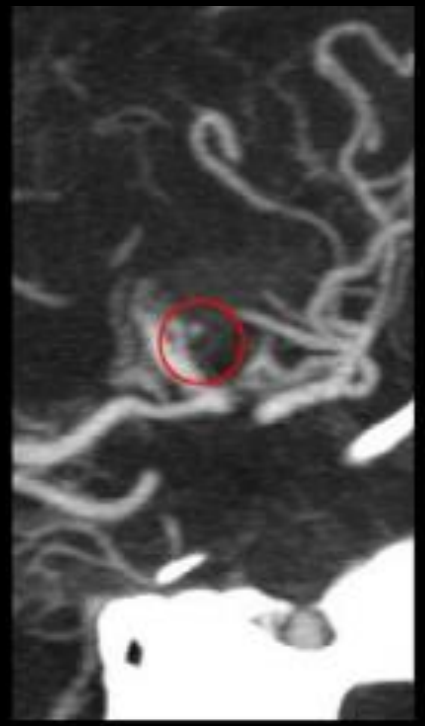


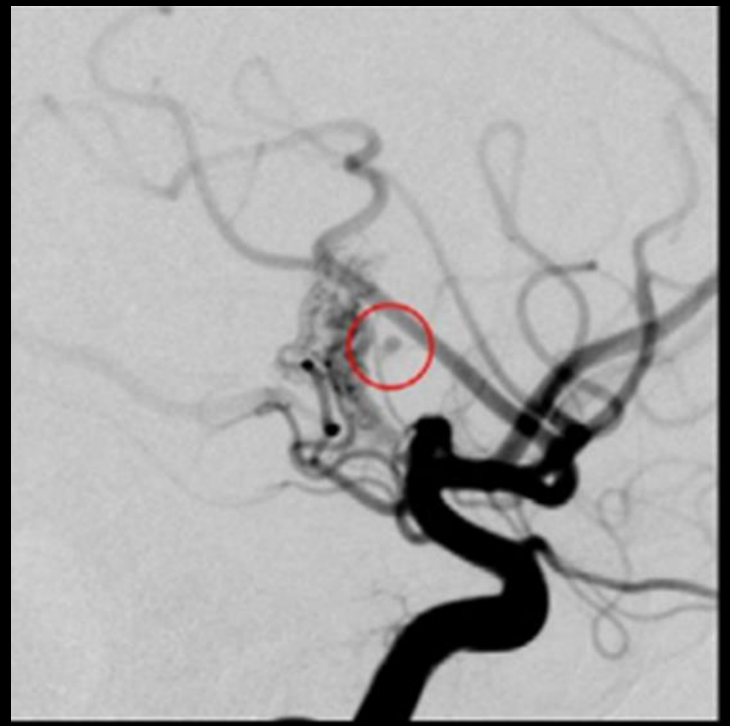
- **Angio TC** :

- Reconstrucciones tridimensionales

- Aneurismas intranidales o de flujo

- Aporte arterial, nidus, drenaje venoso





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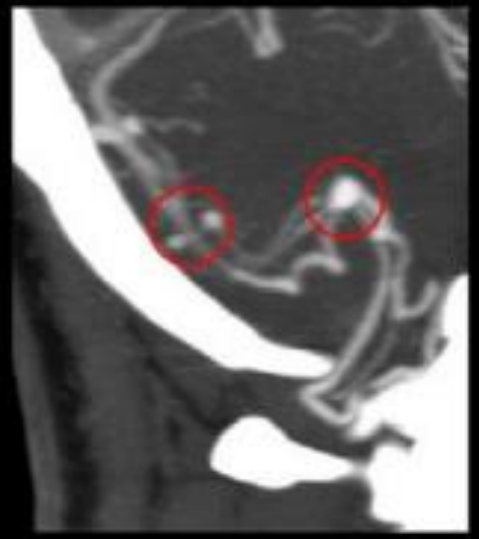

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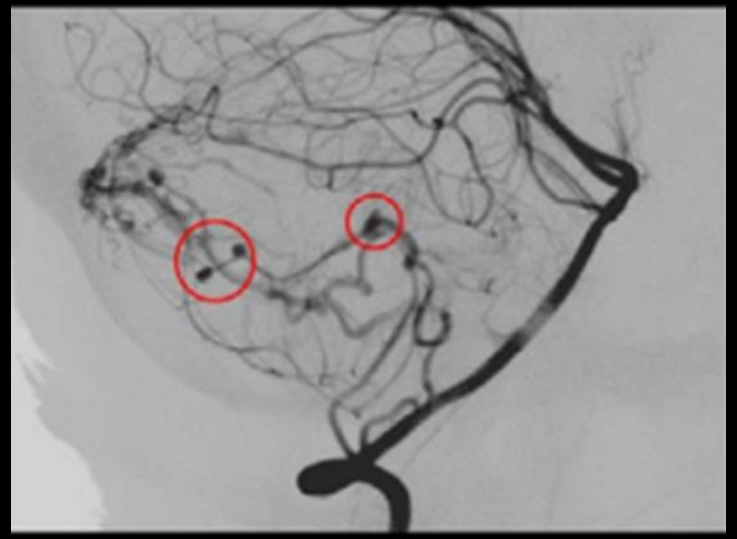

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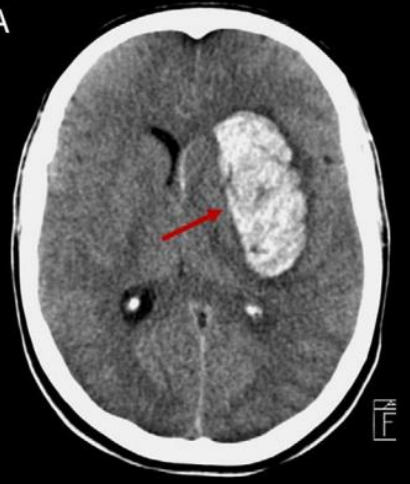

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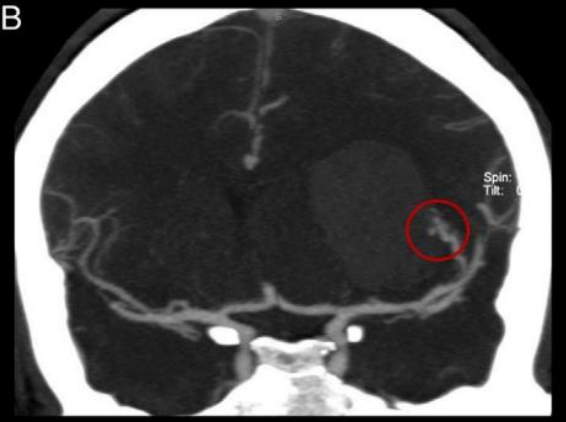

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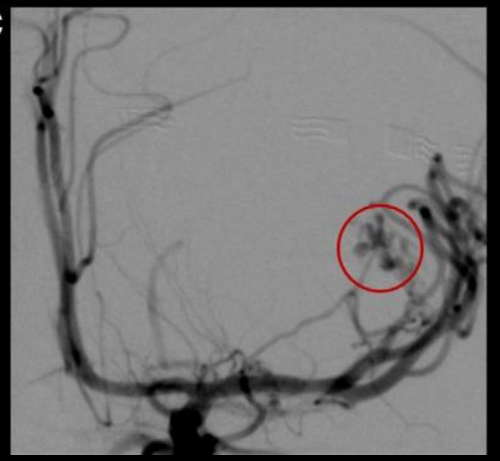

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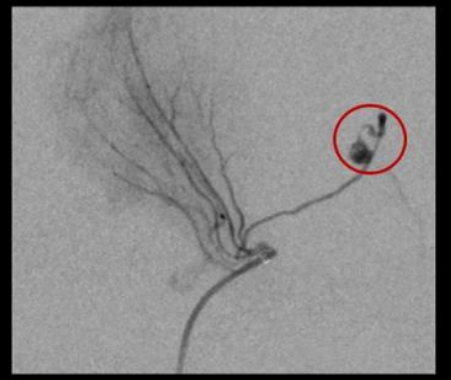

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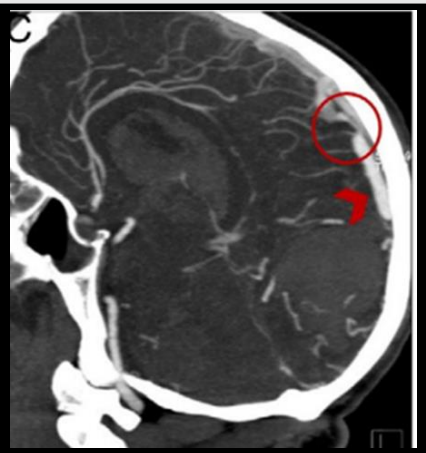
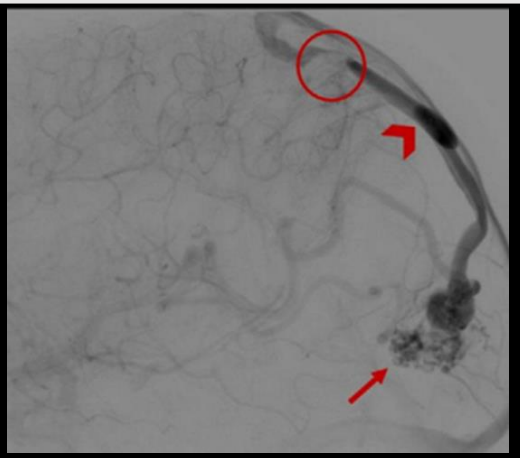
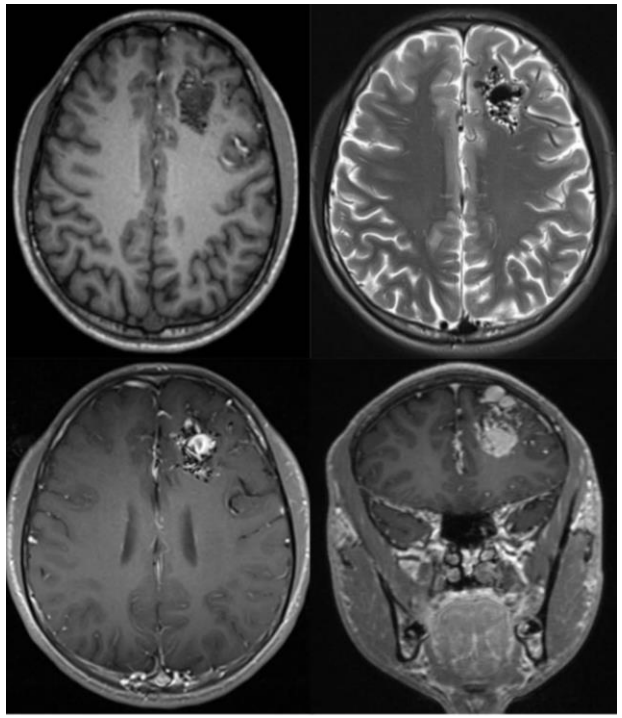
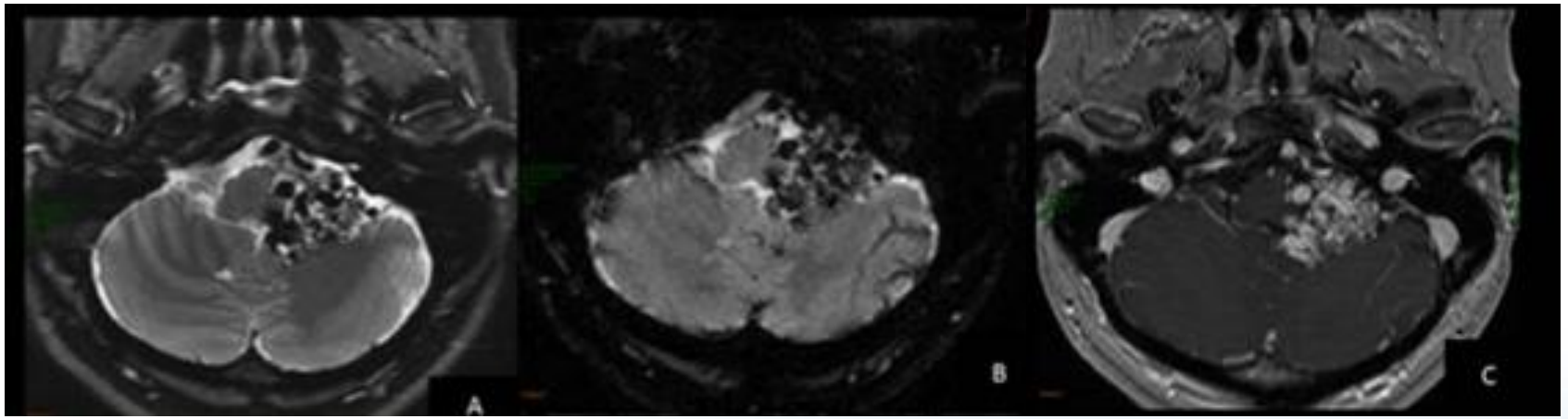




RM MAVc

- **T1 y T2**: imágenes serpiginosas hipointensas debido a la pérdida de señal de alta velocidad "nido de gusanos", que realzan con contraste. Aumento de señal en vasos trombosados o con flujo lento turbulento.
- **T2, Flair:** aumento de señal por gliosis, edema o isquemia parenquimatosa adyacente. Efecto masa mínimo o ausente
- Adecuada visualización de las estructuras parenquimatosas adyacentes al nidus, importante para el tratamiento

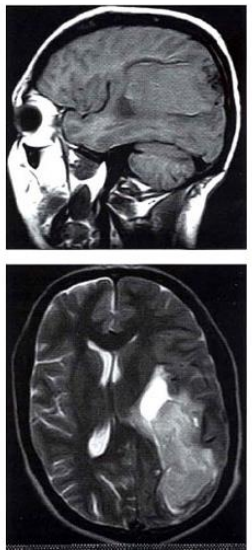




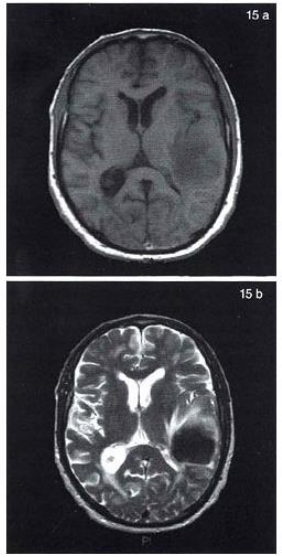
RM MAVc

- **T2* GRE, SWI** (susceptibilidad paramagnética): hemorragia, evolución aguda/subaguda, hemosiderina, hemorragia vs calcio(**SWI**)

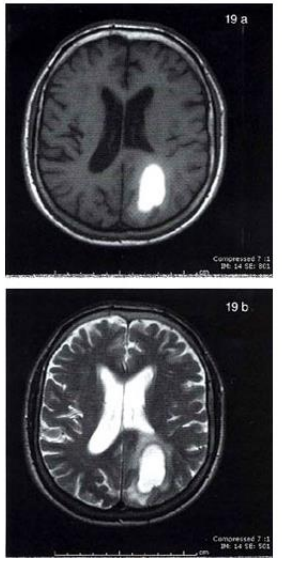
Hematoma	Edad	Imagen T1	Imagen T2
Hematoma hiperagudo	<1° día	Isointenso	Hiperintenso
Hematoma agudo	1er a 3er día	Hipo-isointenso	Hipointenso
Hematoma subagudo precoz	4° a 7° día	Hiperintenso	Hipointenso
Hematoma subagudo tardío	8° a 14° día	Hiperintenso	Hiperintenso
Hematoma crónico	>14° día	Hipointenso	Hipointenso



HEMATOMA HIPERAGUDO



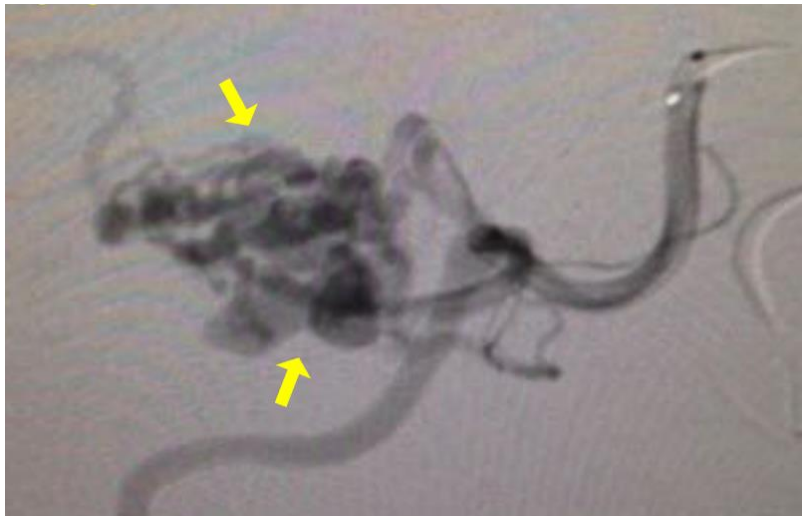
HEMATOMA AGUDO



HEMATOMA SUBAGUDO

- **Técnicas Angiográficas 2D y 3D TOF** sin y con Gad y **Técnicas con resolución de tiempo de cinética de contraste**, extraen distintas fases vasculares

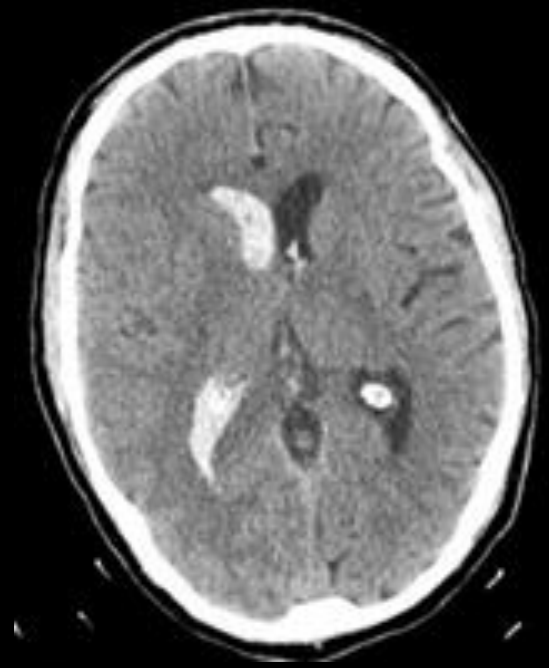
ANGIOGRAFÍA CEREBRAL MAVc

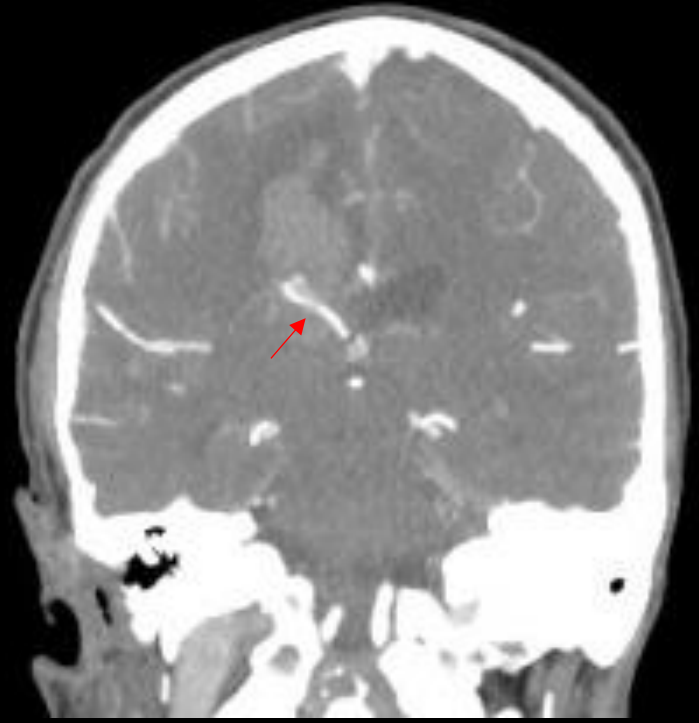


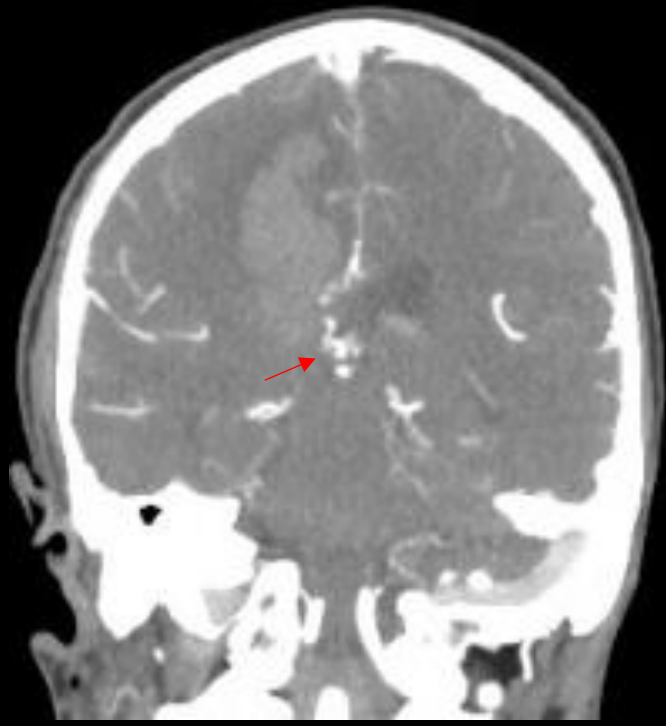
Angiografía cerebral Gold Standar

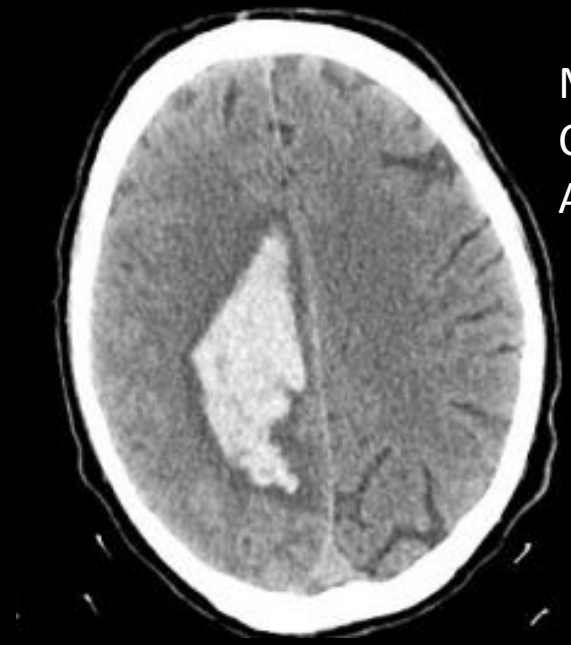
- Aferencias CI, CE, durales y vertebrales
- Nido
- Drenaje venoso y patrón venoso cerebral
- Aneurismas intra/extranidales
- Cambios angiopáticos secundarios al alto flujo: estenosis, dilataciones
- Hemodinámica de tiempo arteriovenoso
- Fístulas AV directas

Planificación del tratamiento

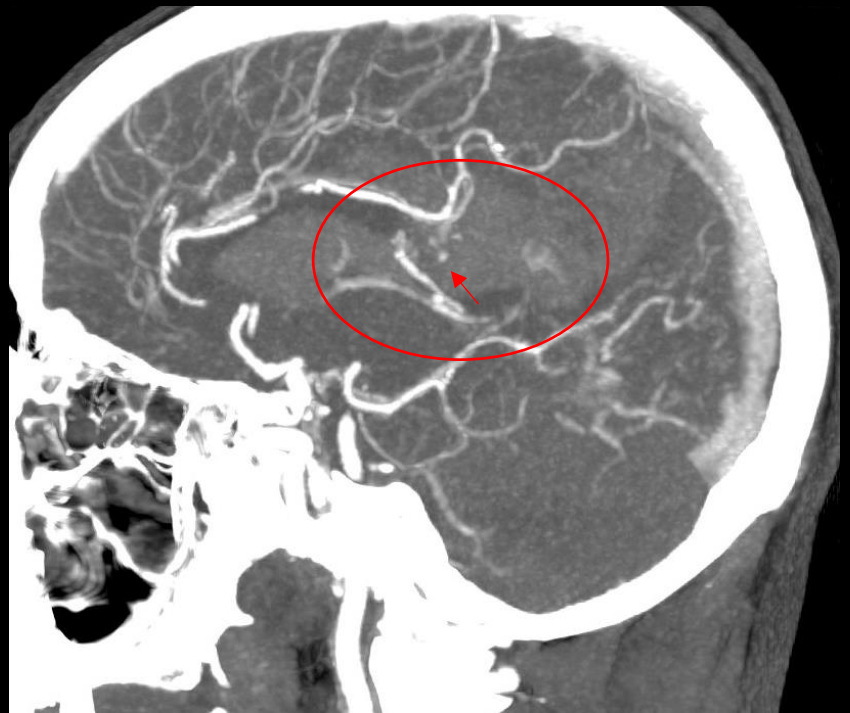


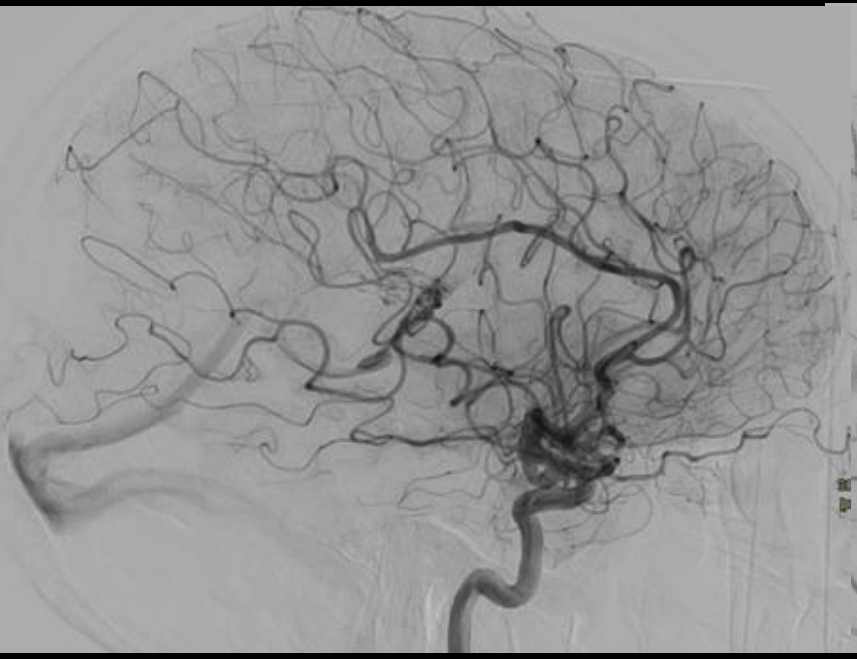


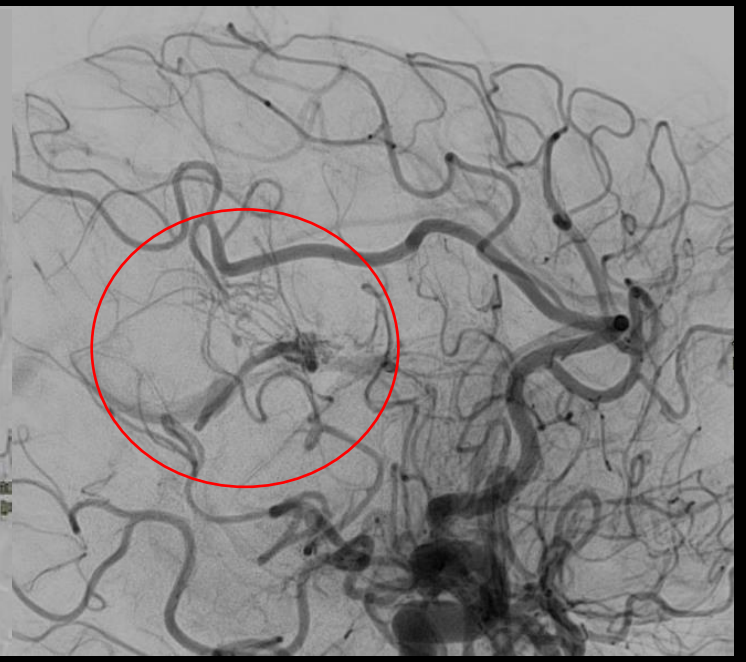


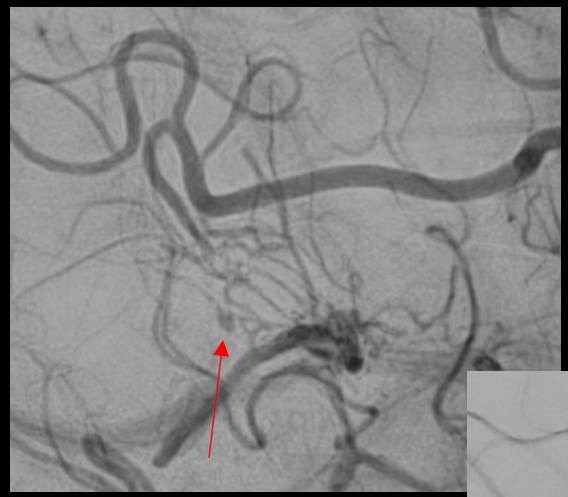


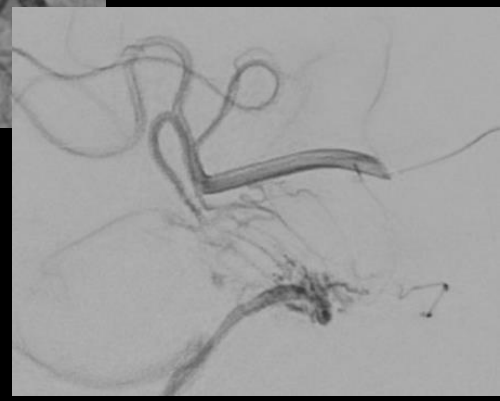
MAV HEMATOMA PARENQUIMATOSO
CON EXTENSIÓN INTRAVENTRICULAR
ANEURISMA INTRANIDAL

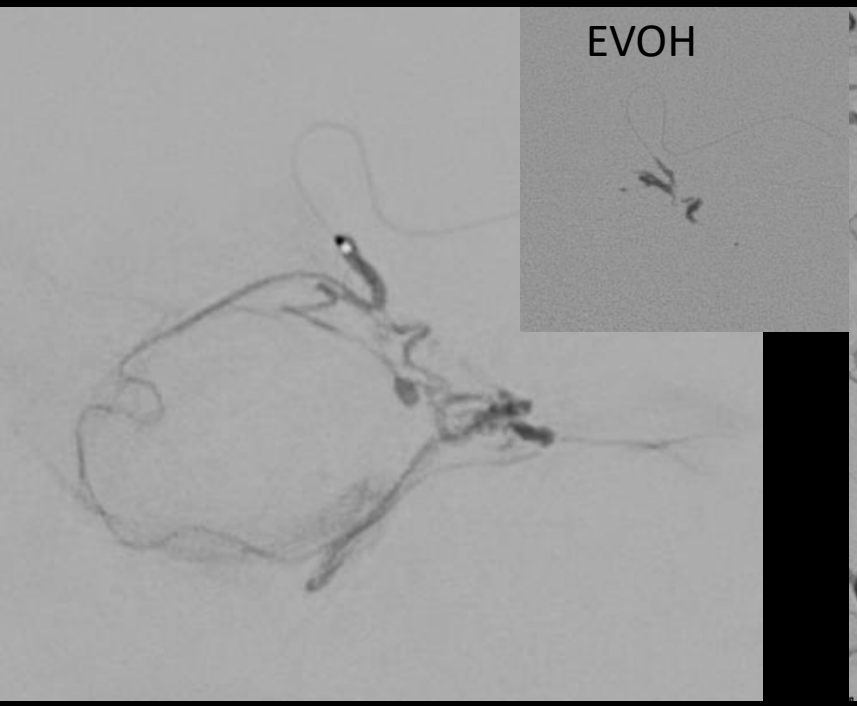




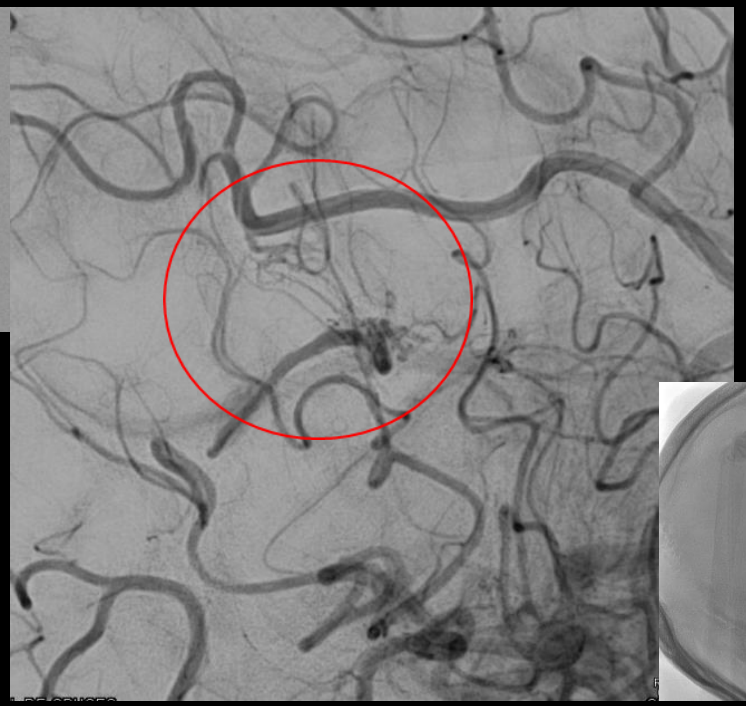


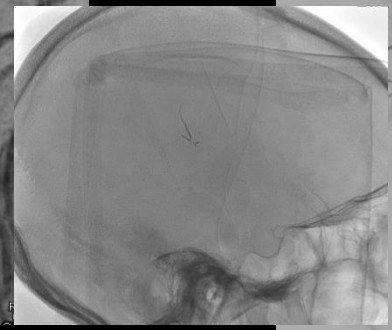


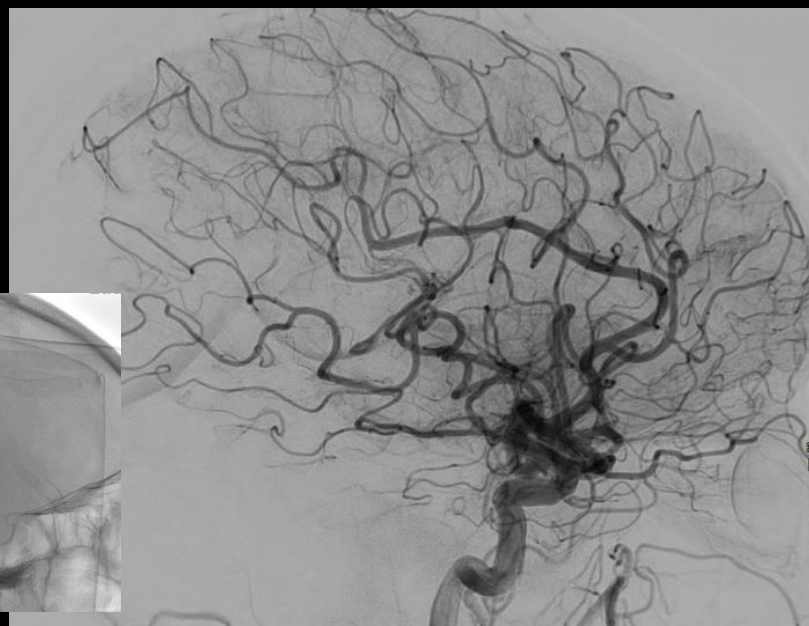




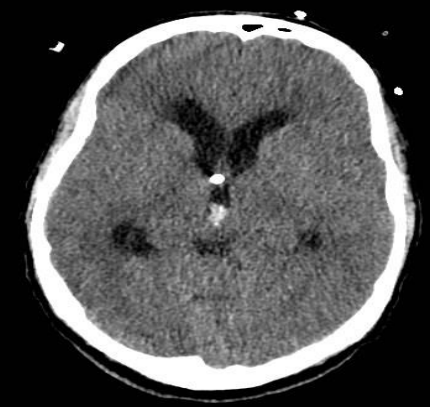
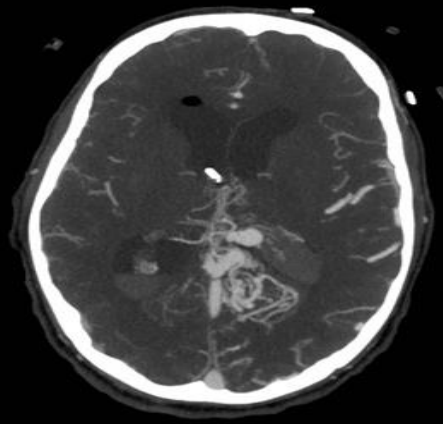
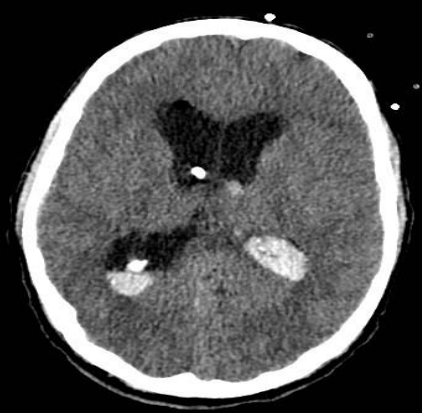
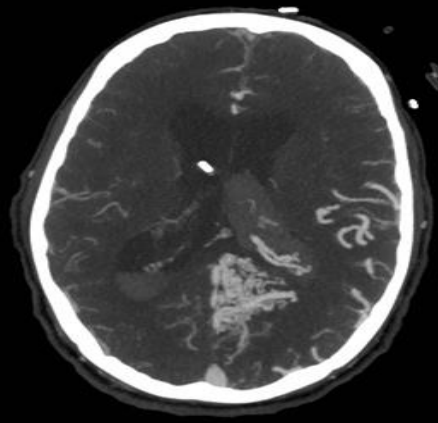
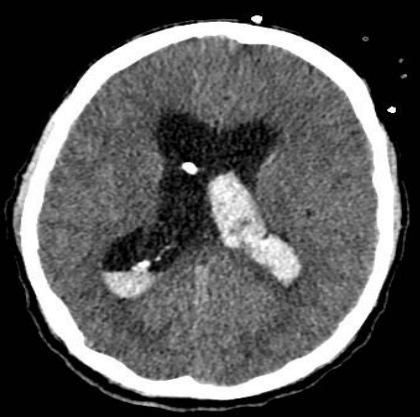
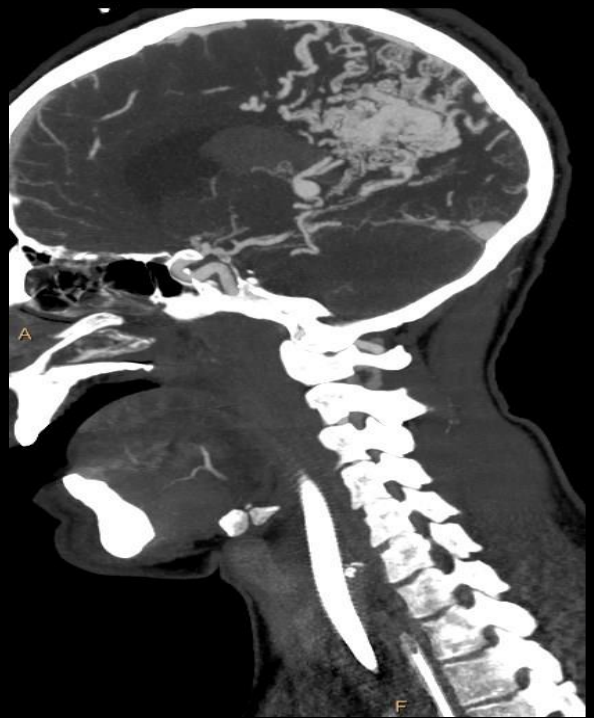
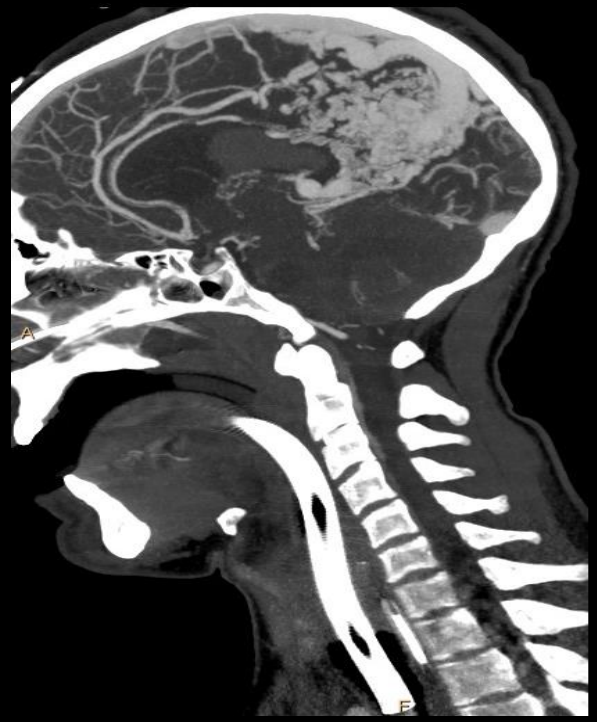
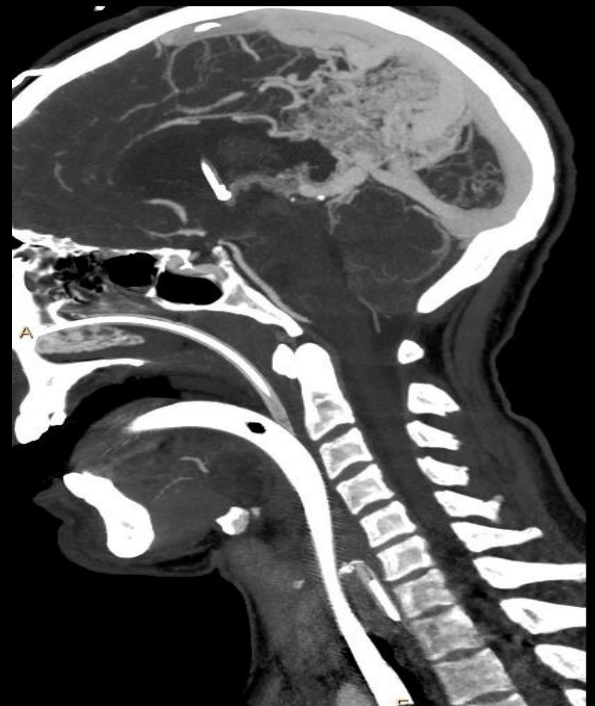
EVOH

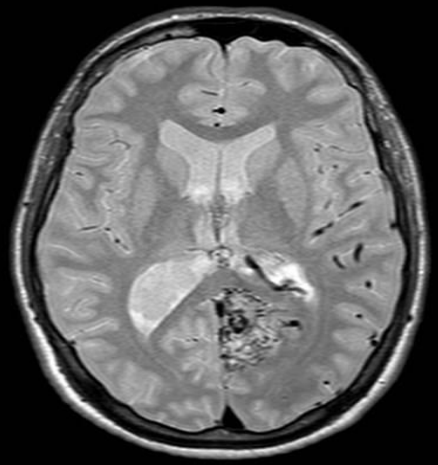
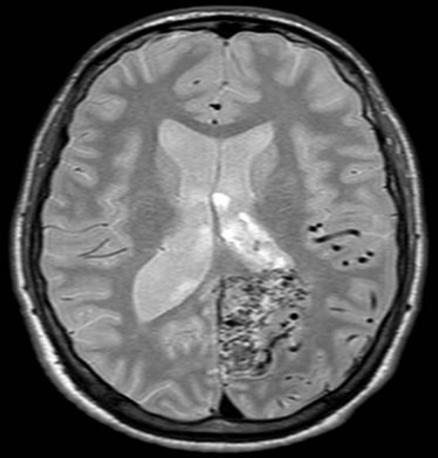
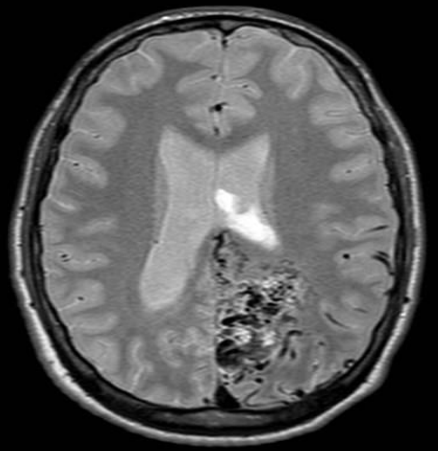
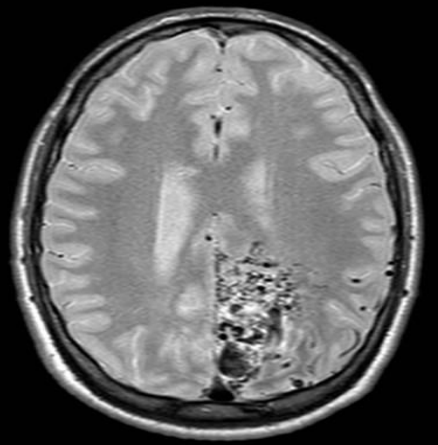
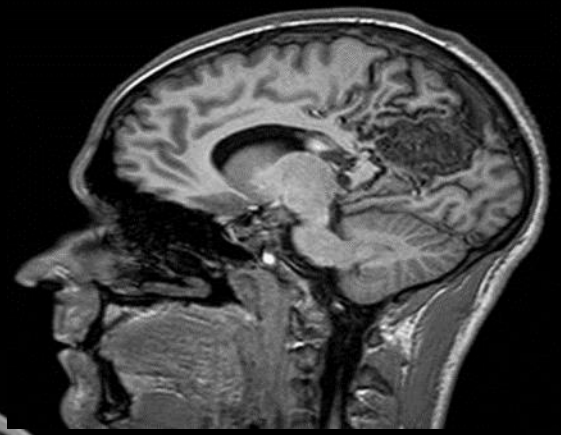
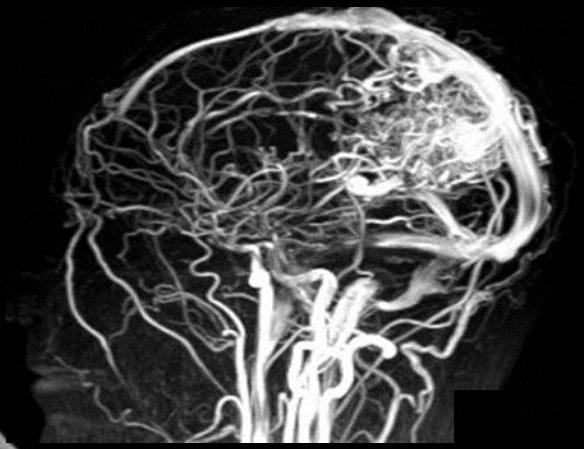
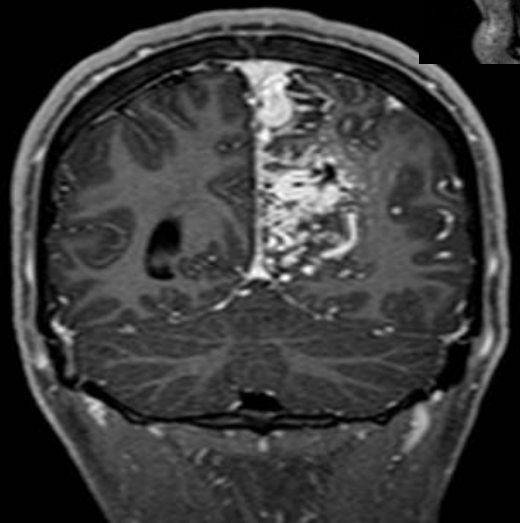
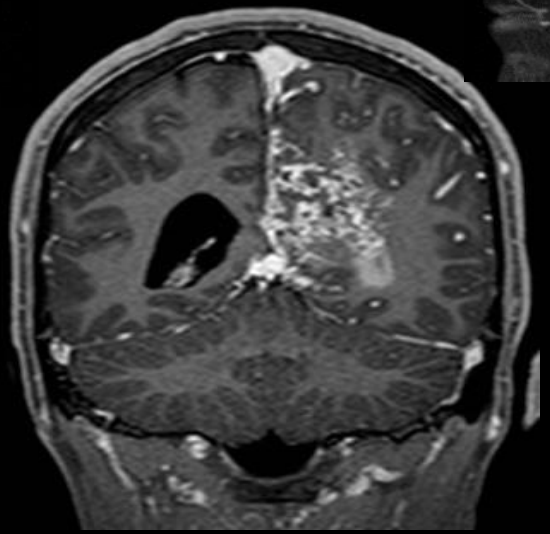
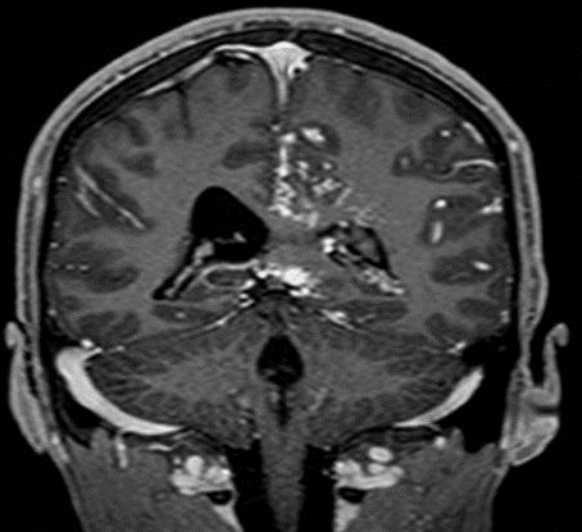


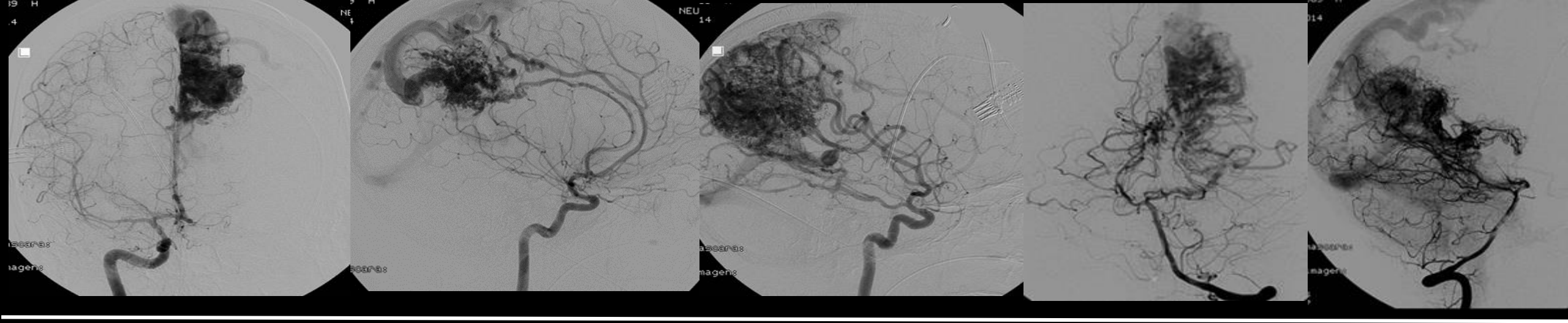




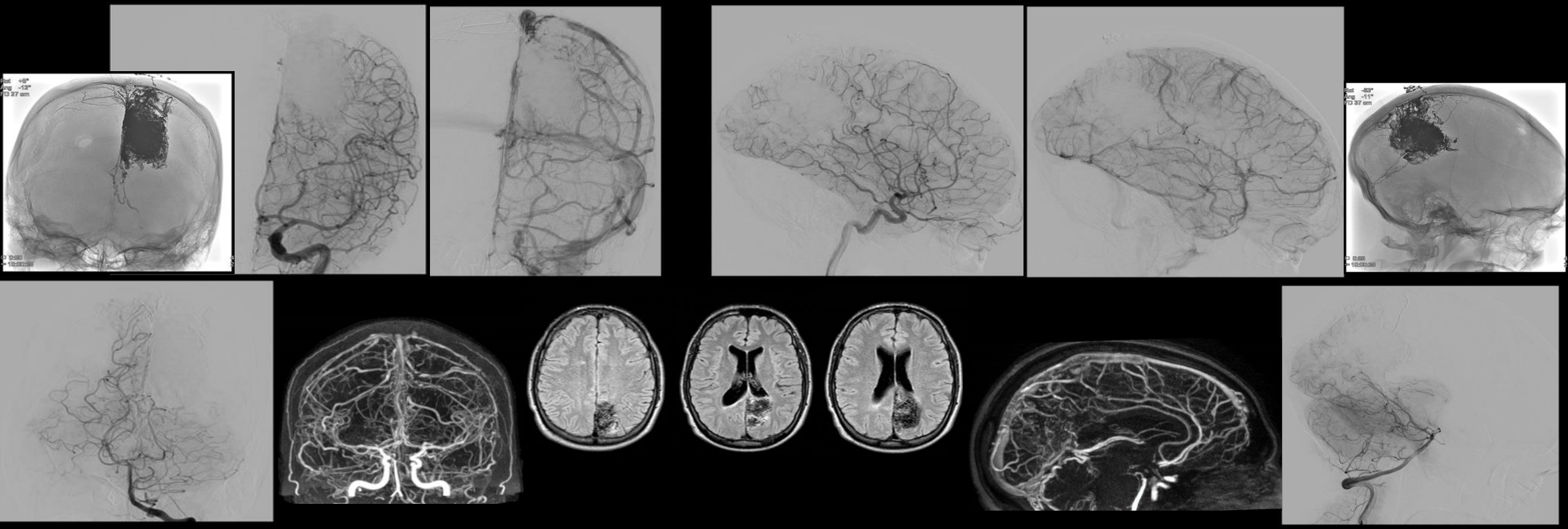
VARON 25 AÑOS
HEMORRAGIA IV

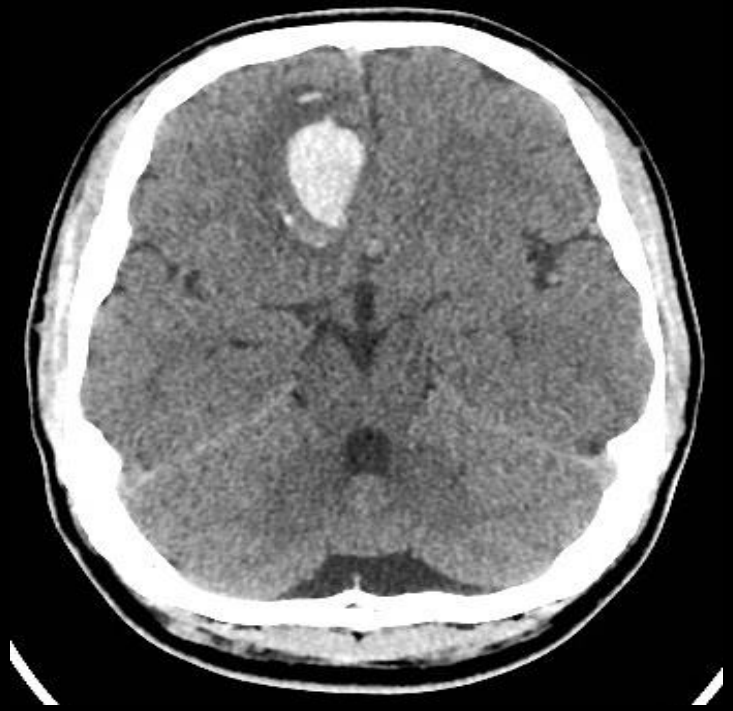









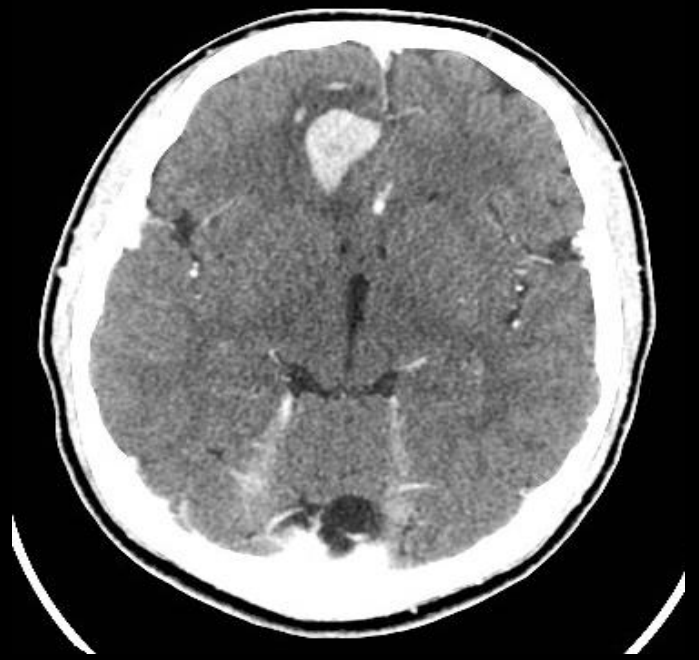










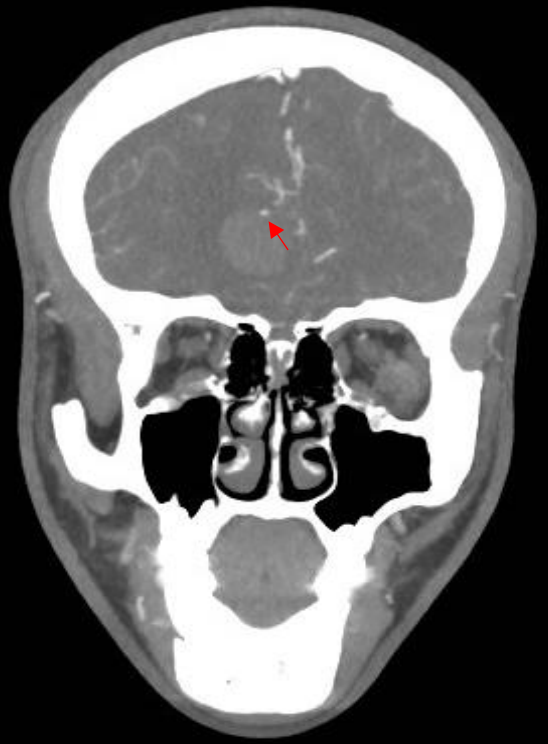


Control post tratamiento

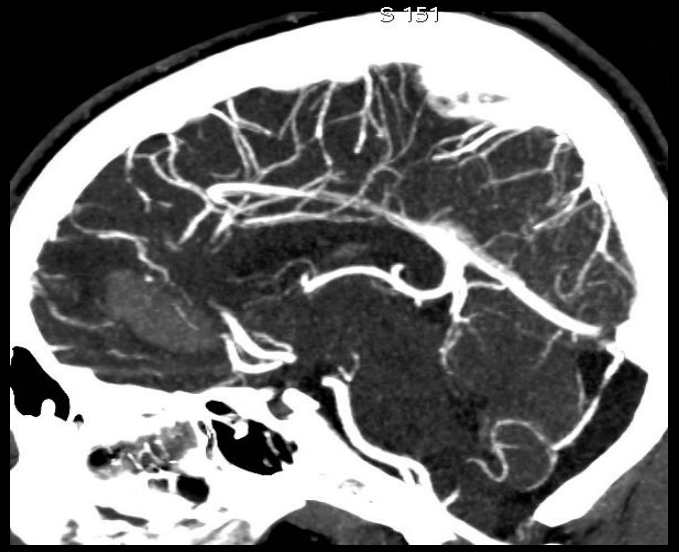


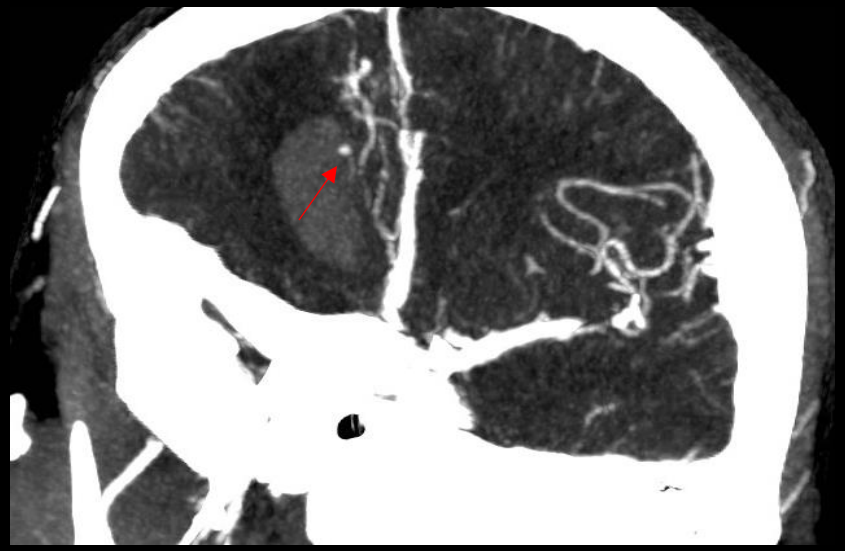


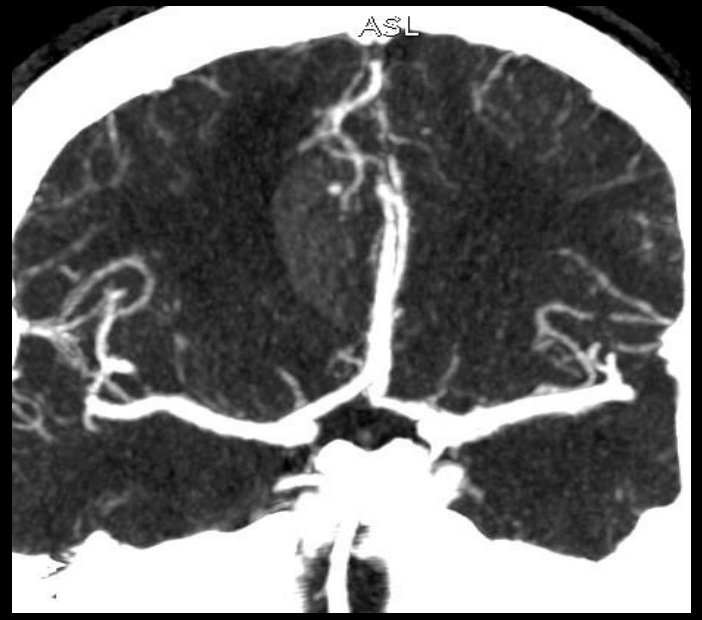


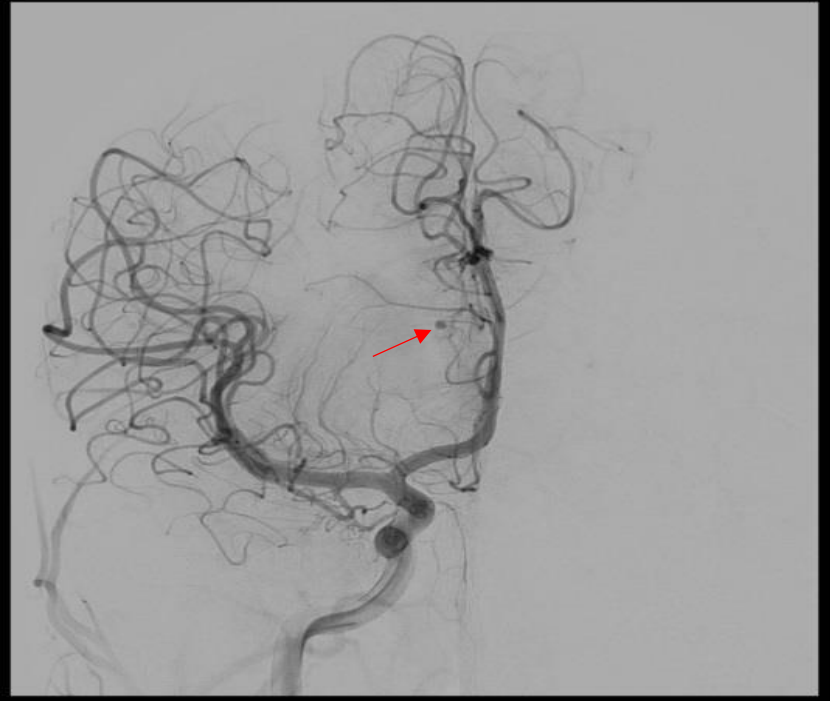
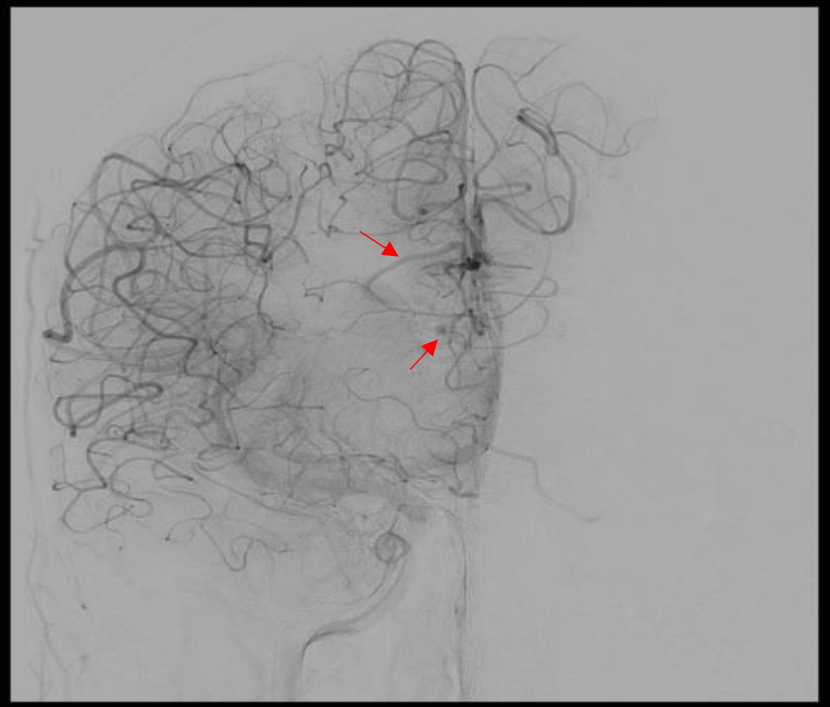
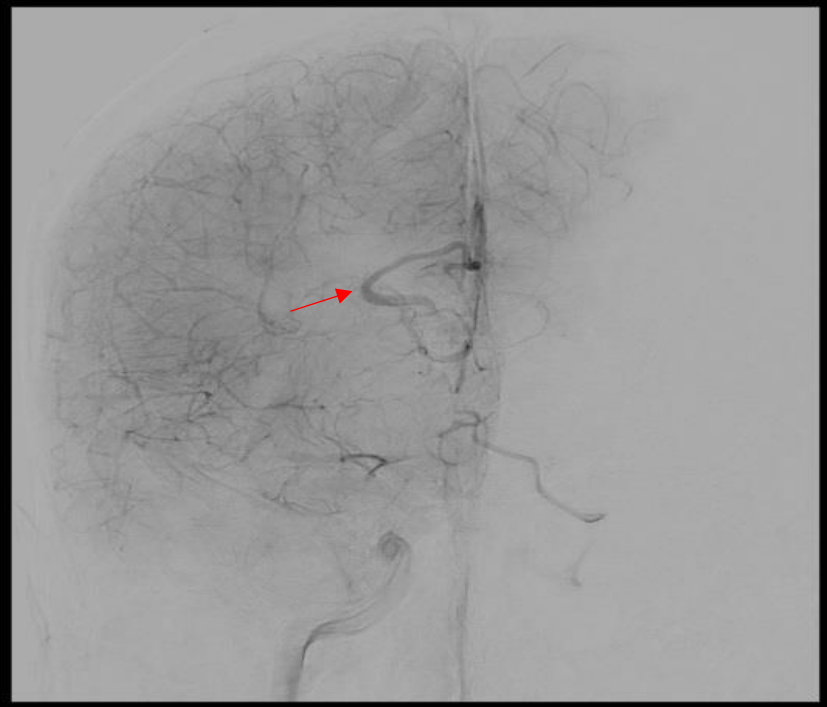
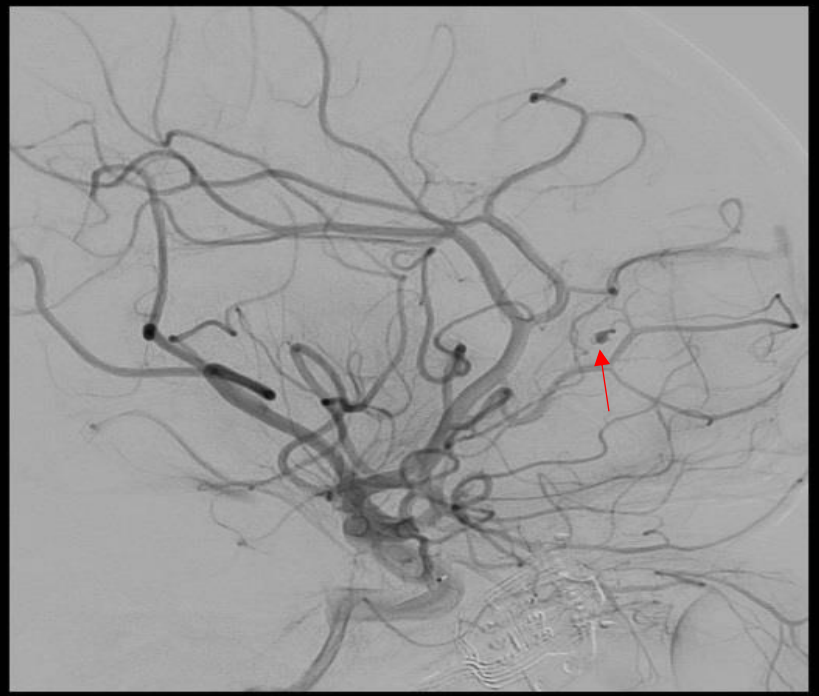
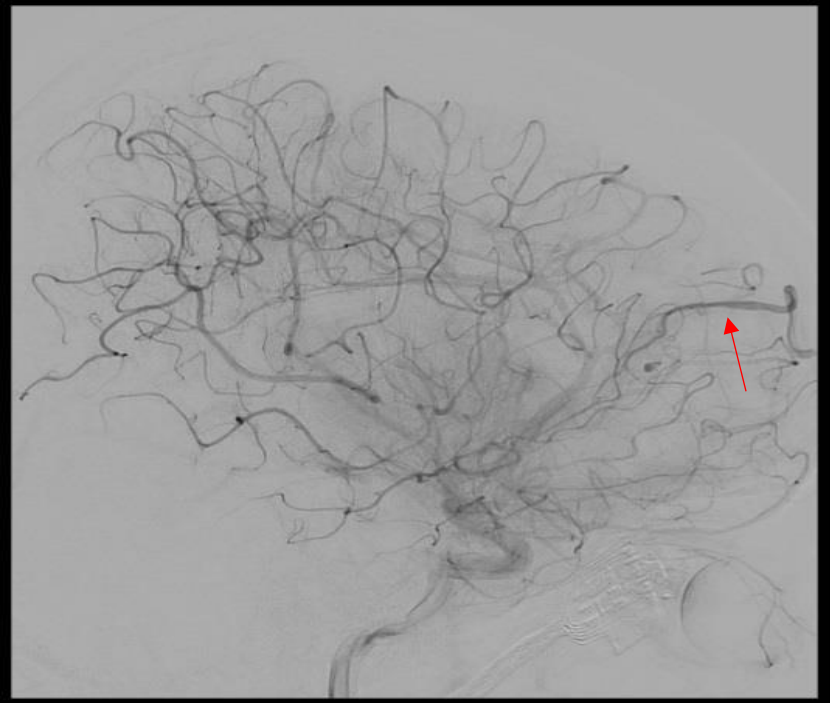
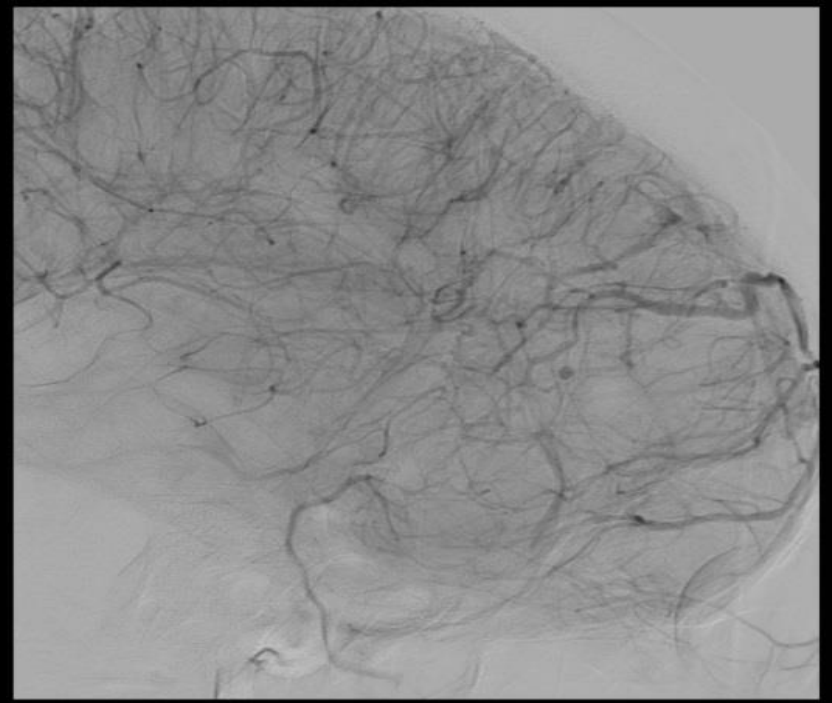


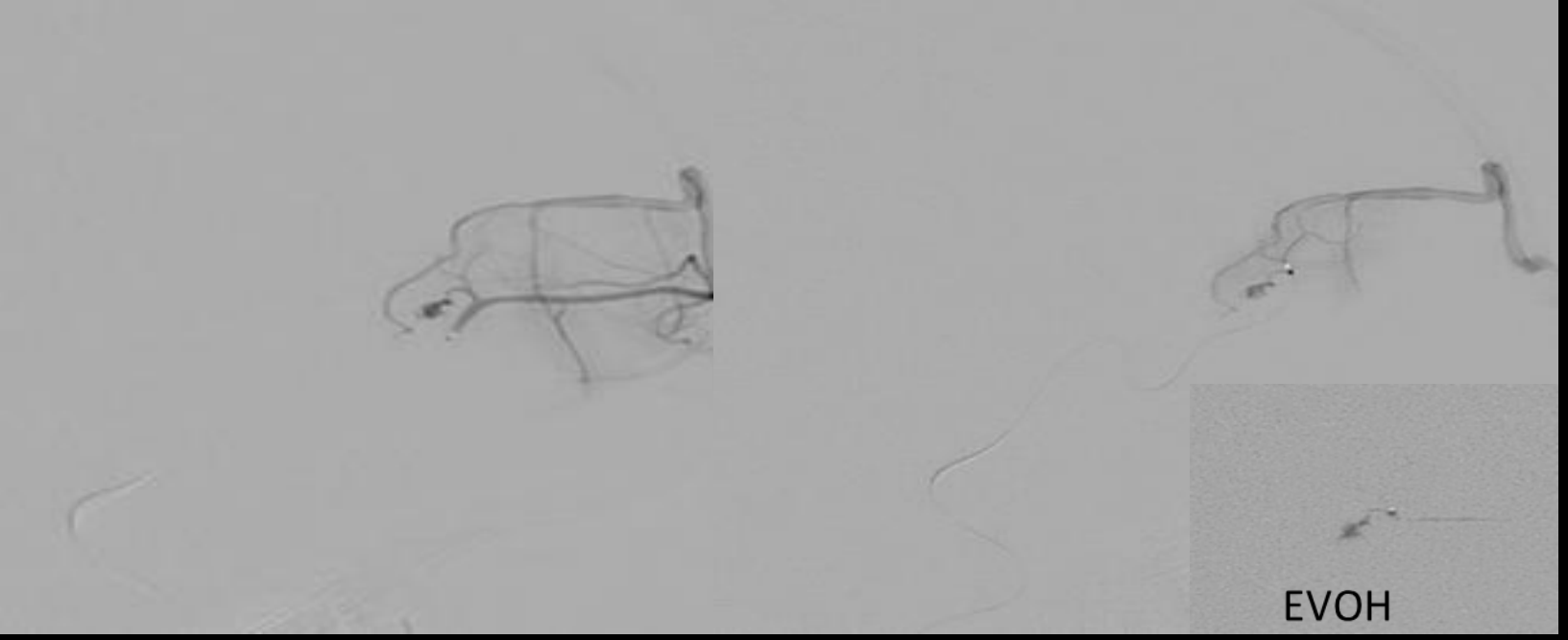
MAV CON HEMORRAGIA POR ANEURISMA DE FLUJO





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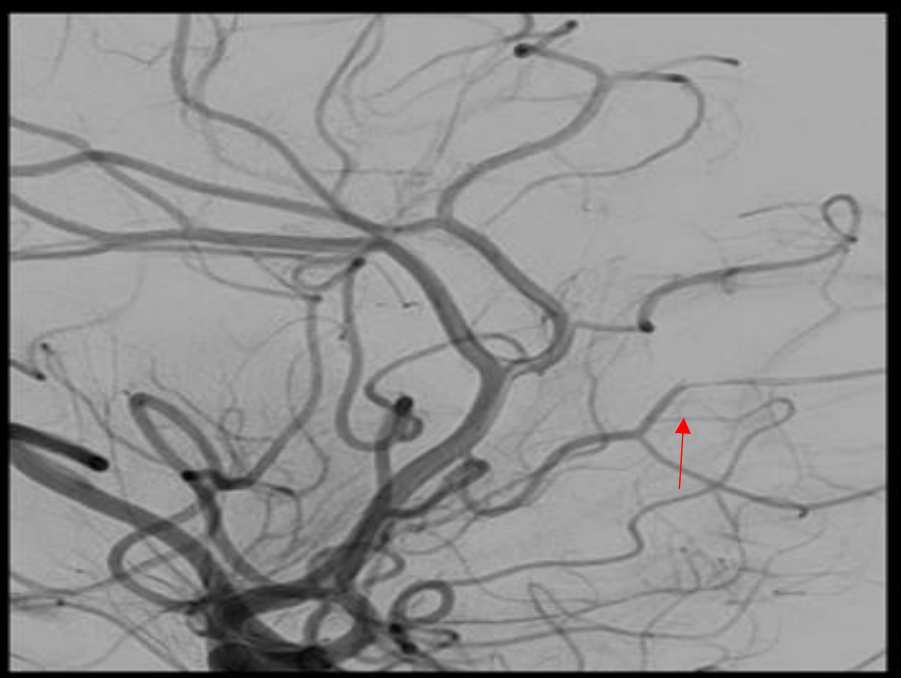

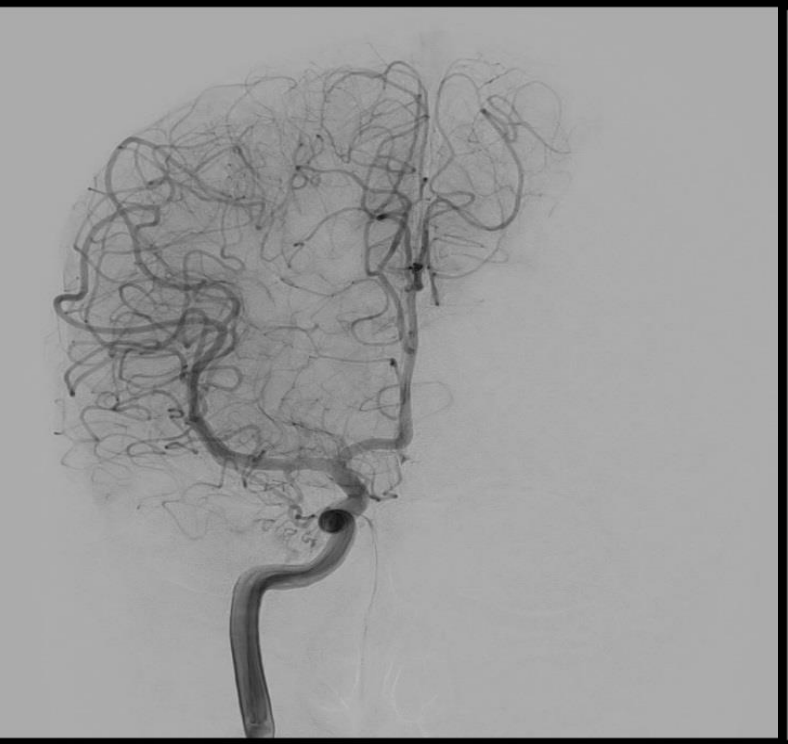

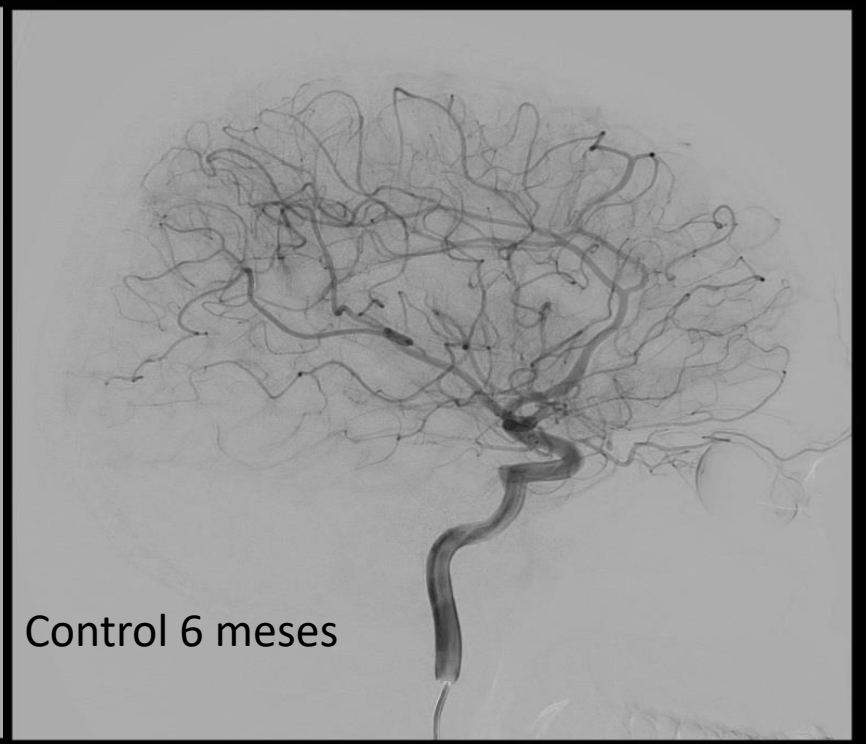
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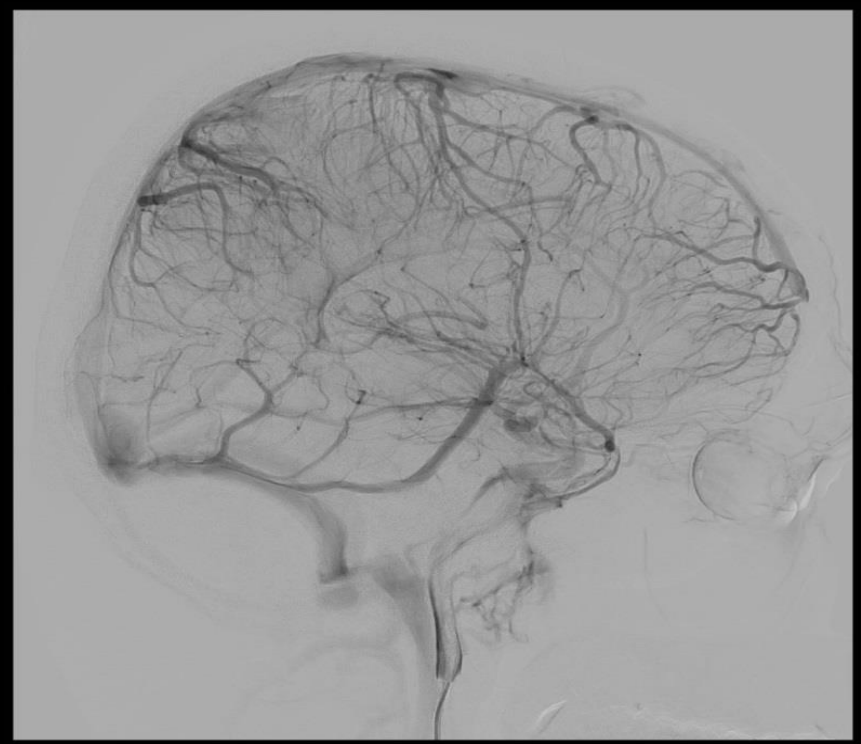
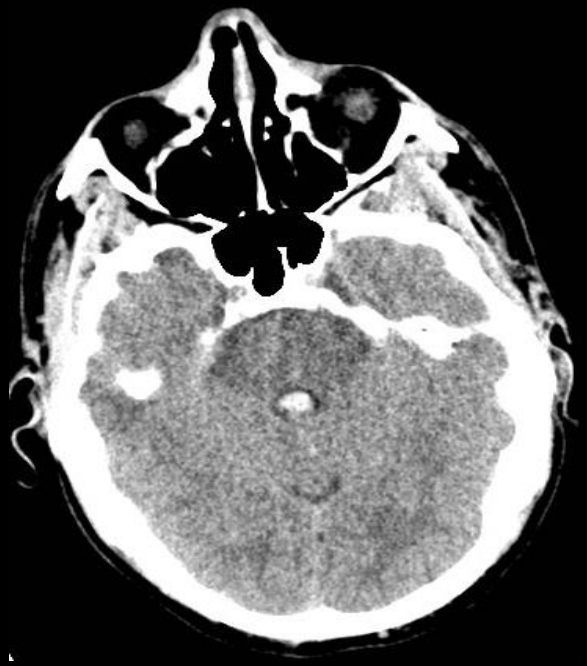
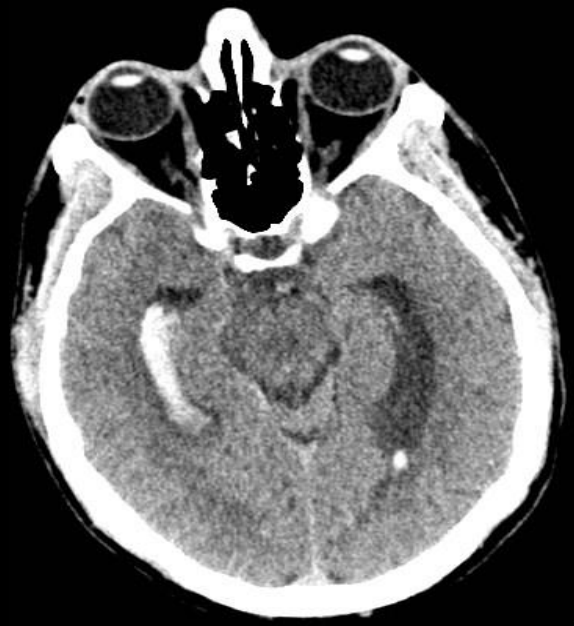
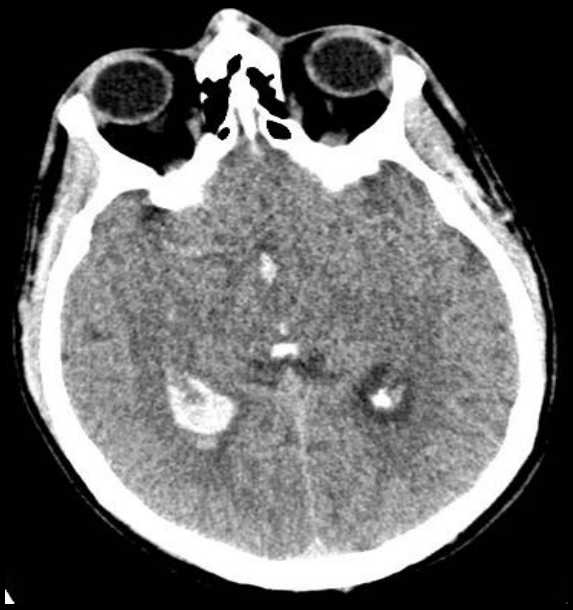
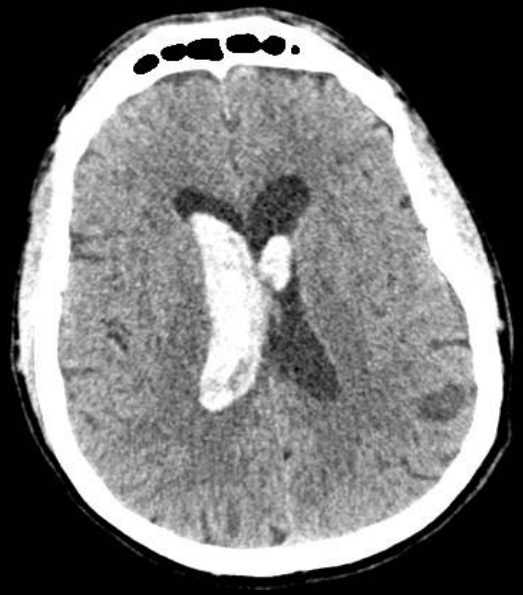
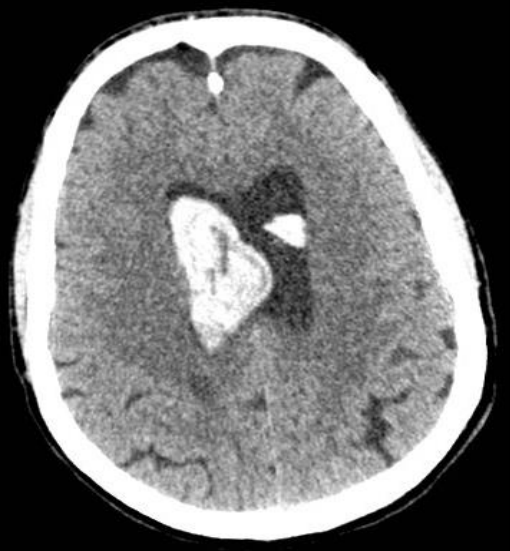
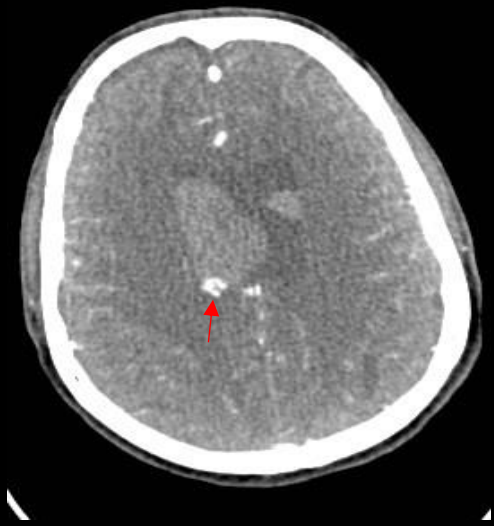
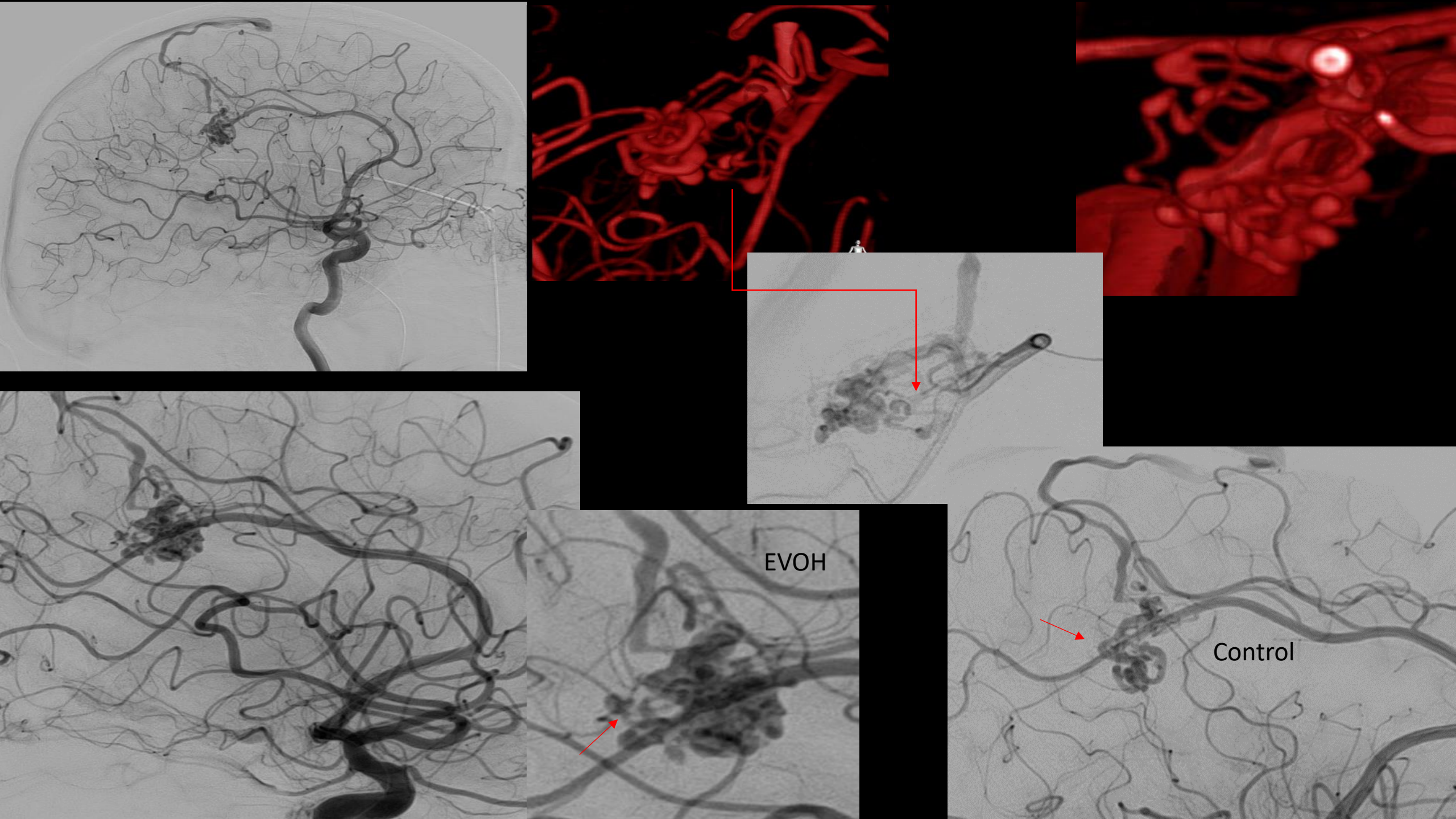
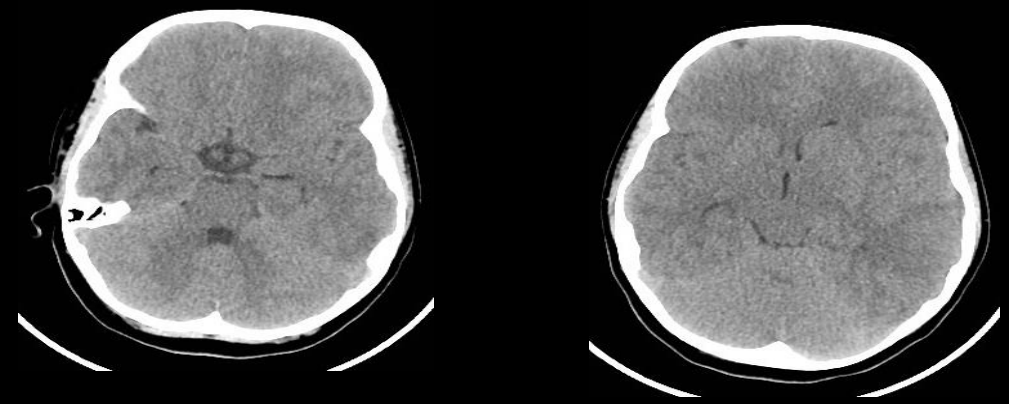


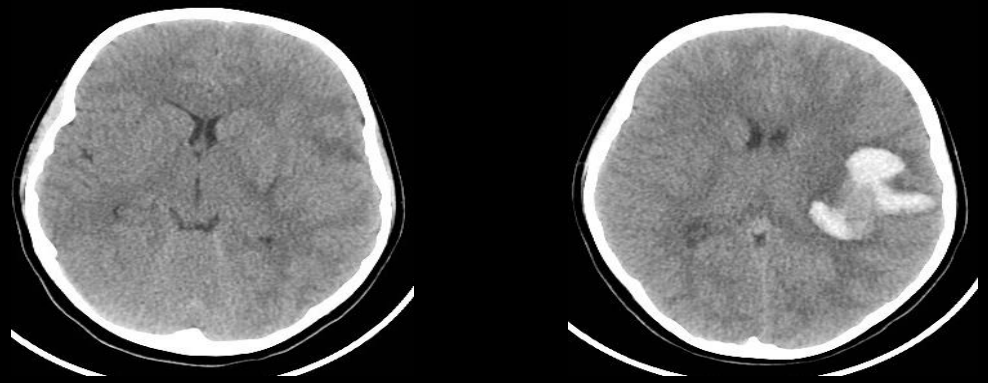




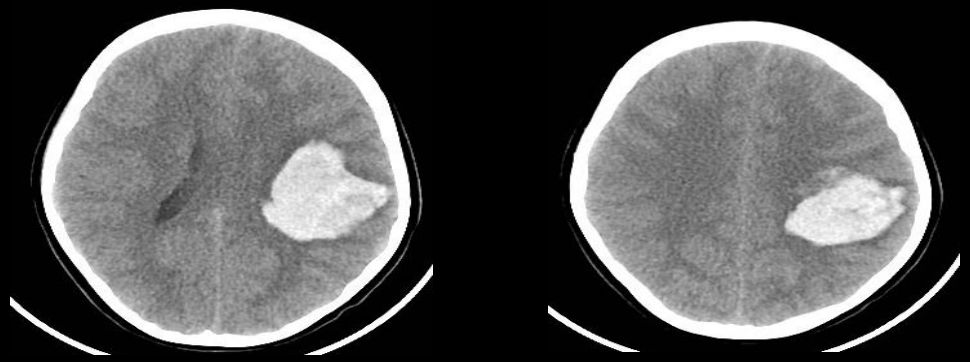
MAV CON HEMORRAGIA POR MICROANEURISMAS INTRANIDALES PERIVENTRICULARES


EVOH
Control







VARÓN 8 AÑOS
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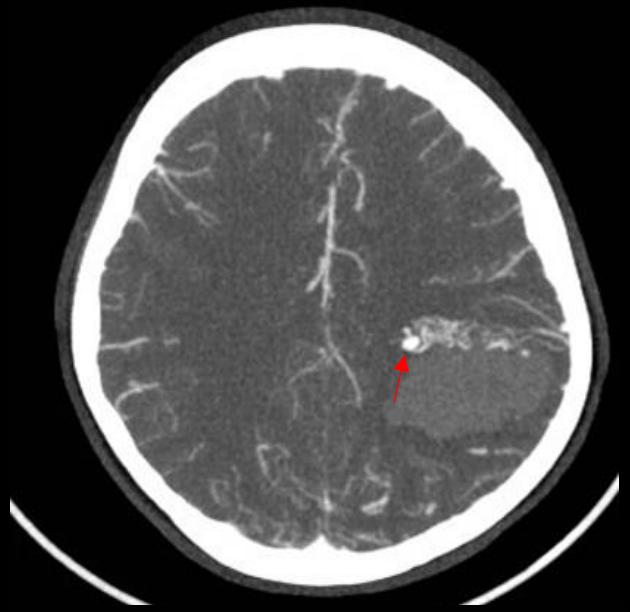
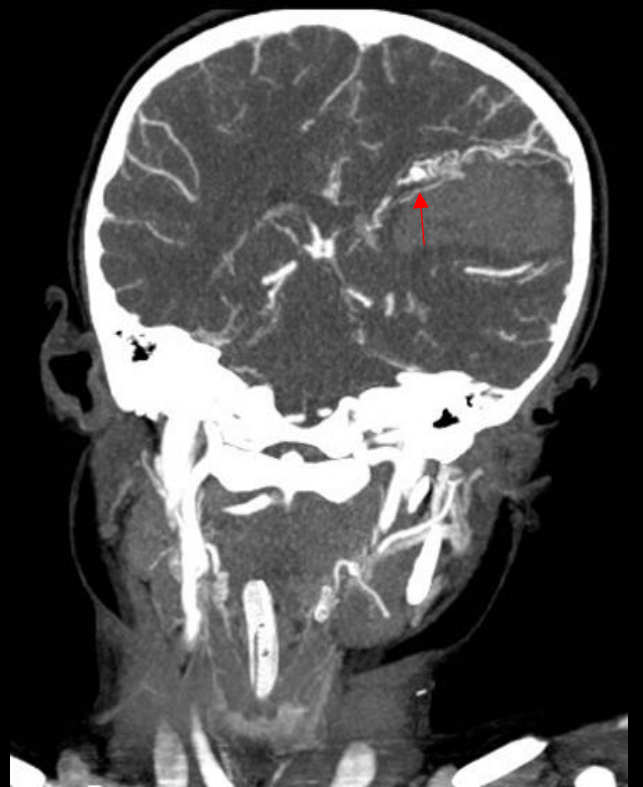
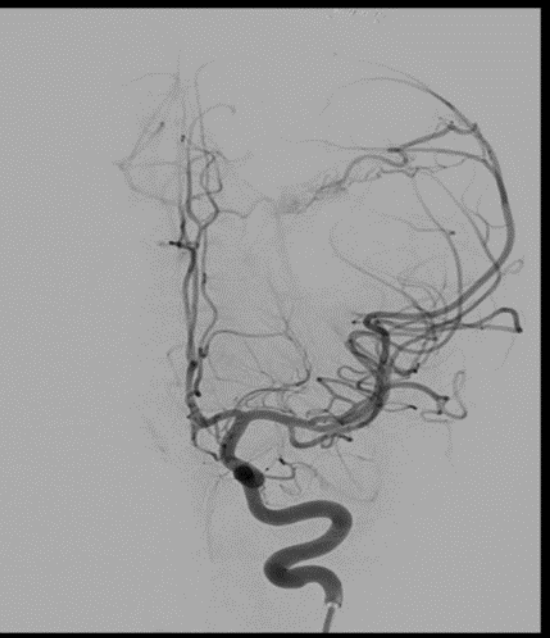
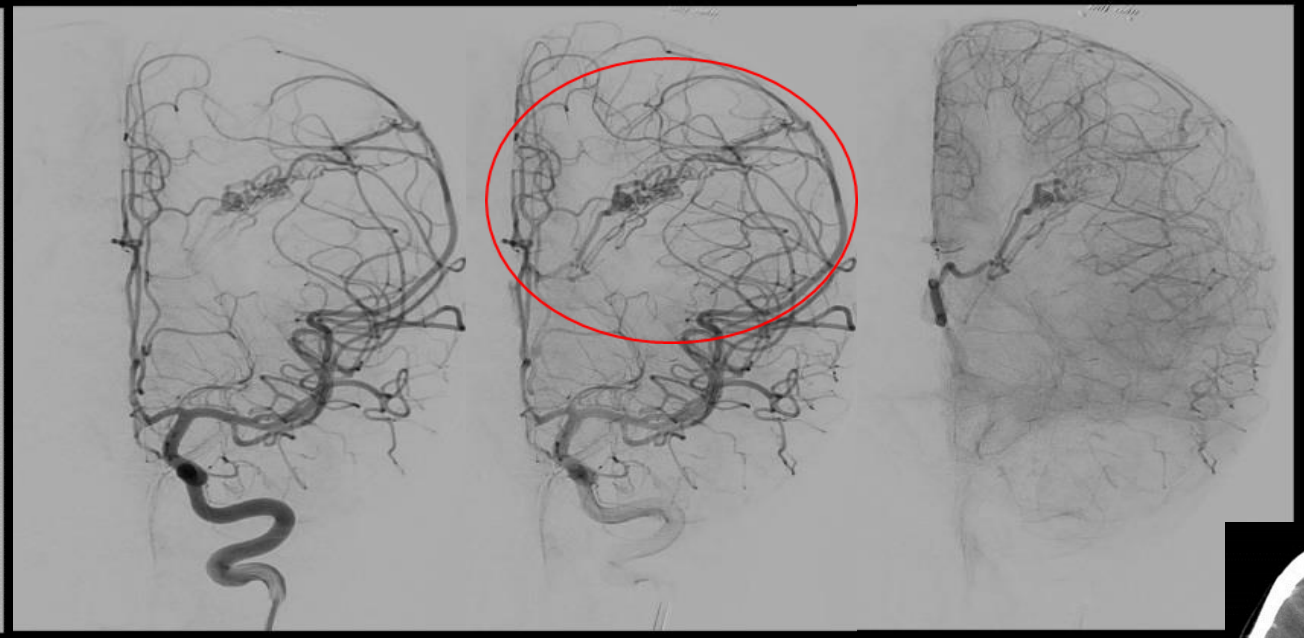
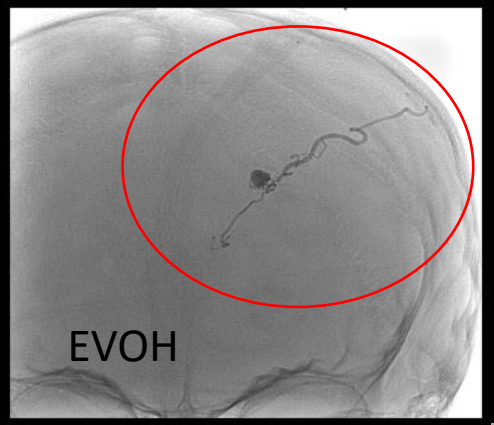
ANEURISMA INTRANIDAL







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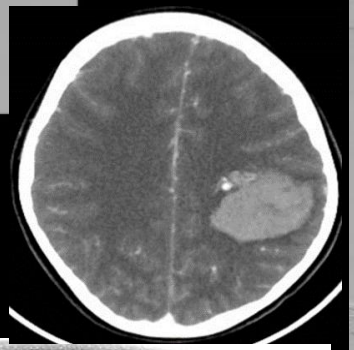

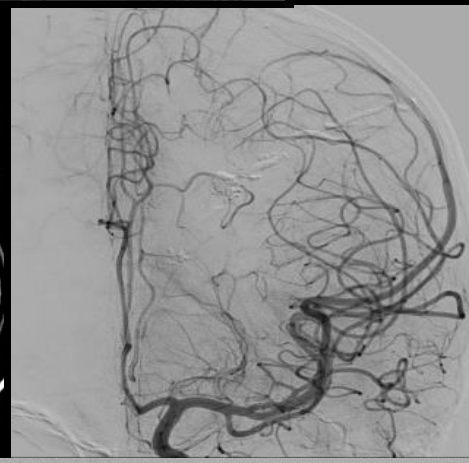

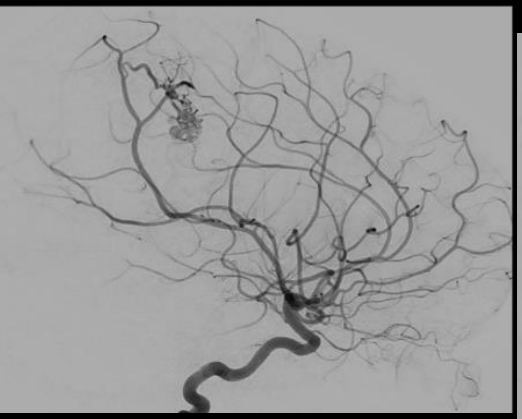

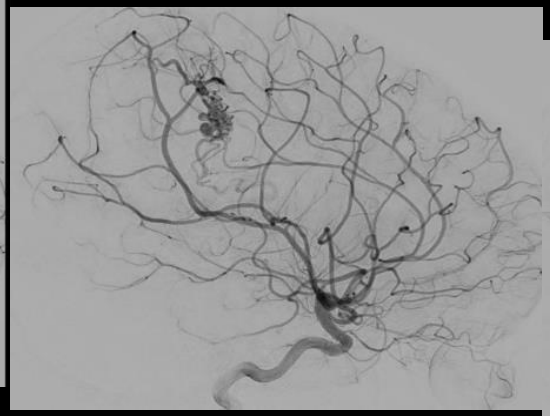

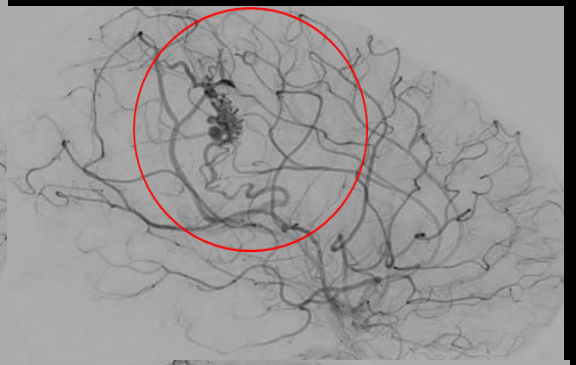

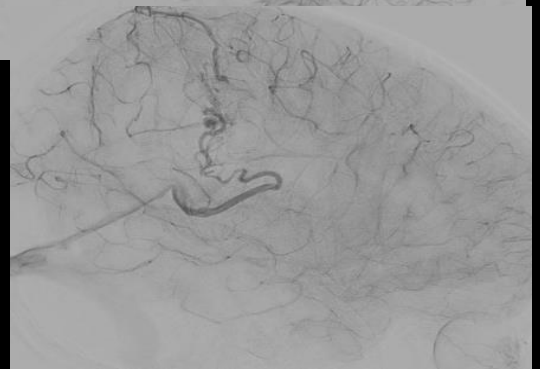

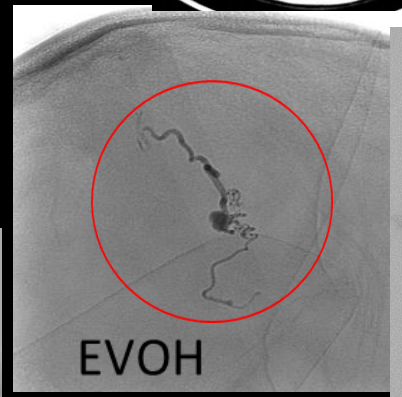
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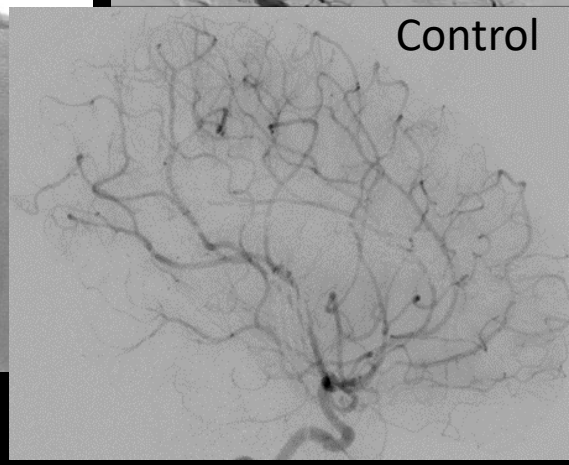
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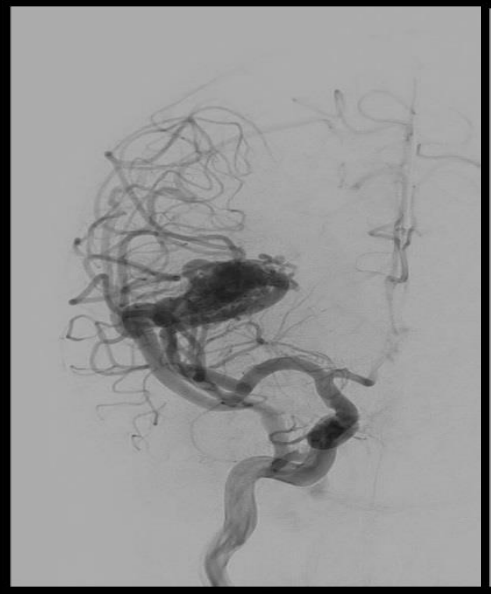
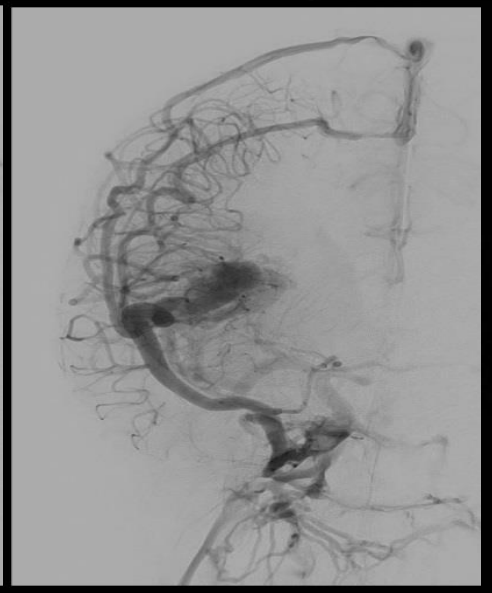
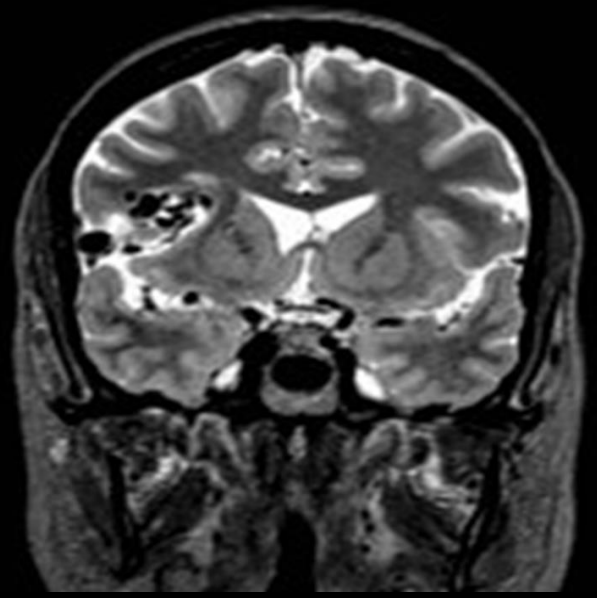
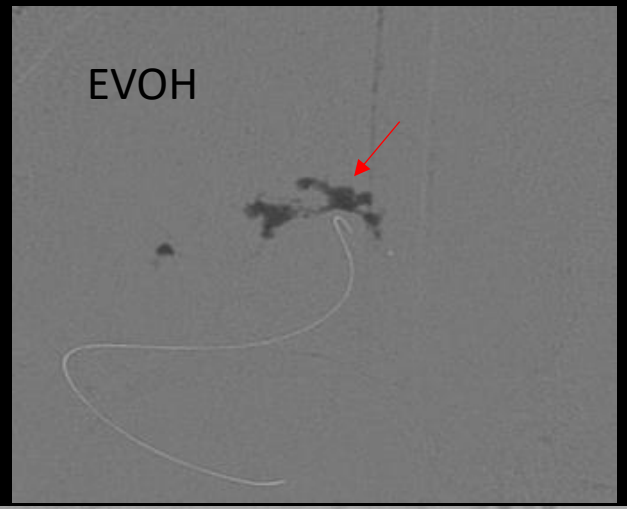
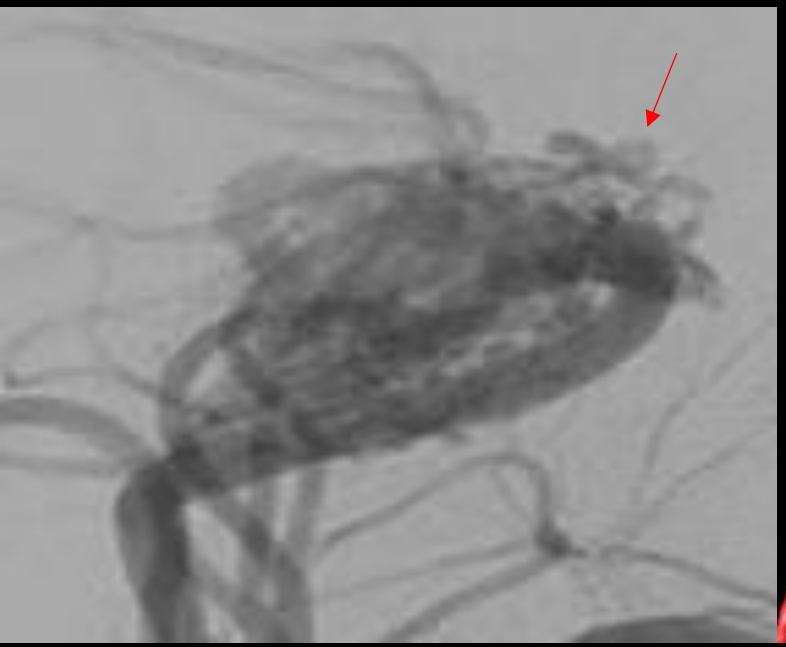


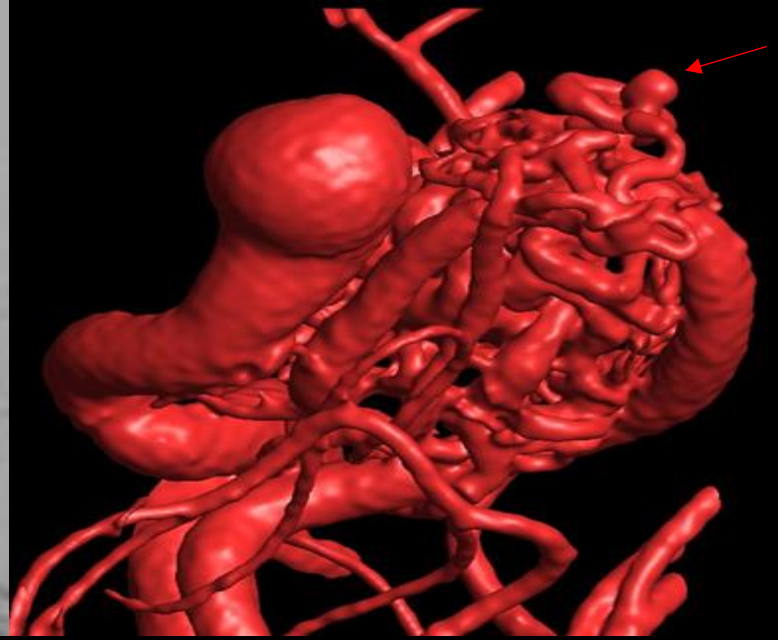


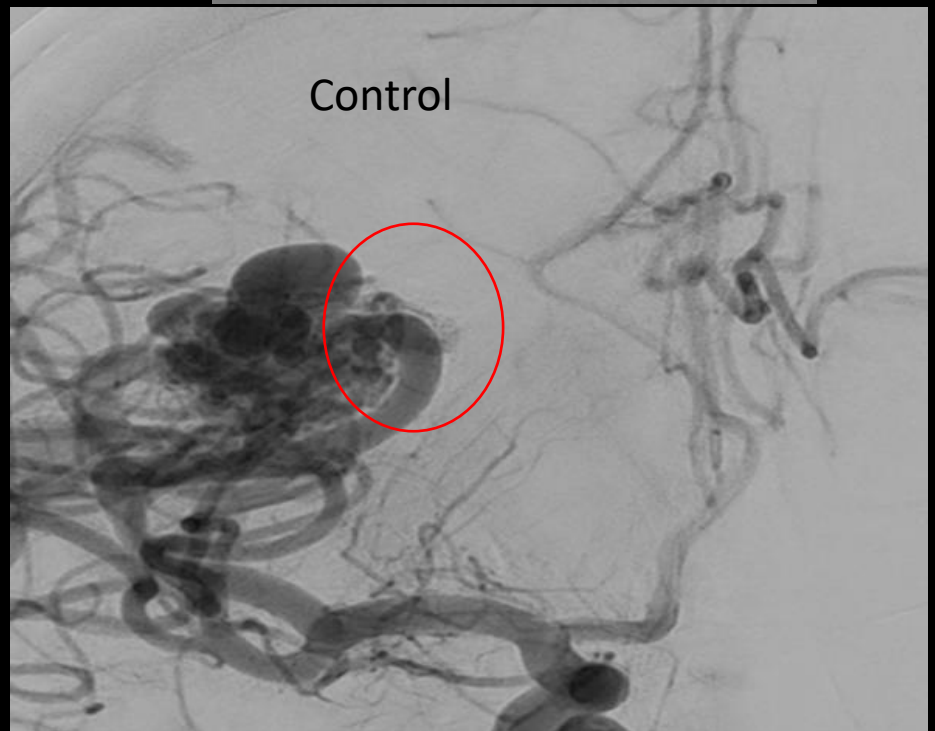


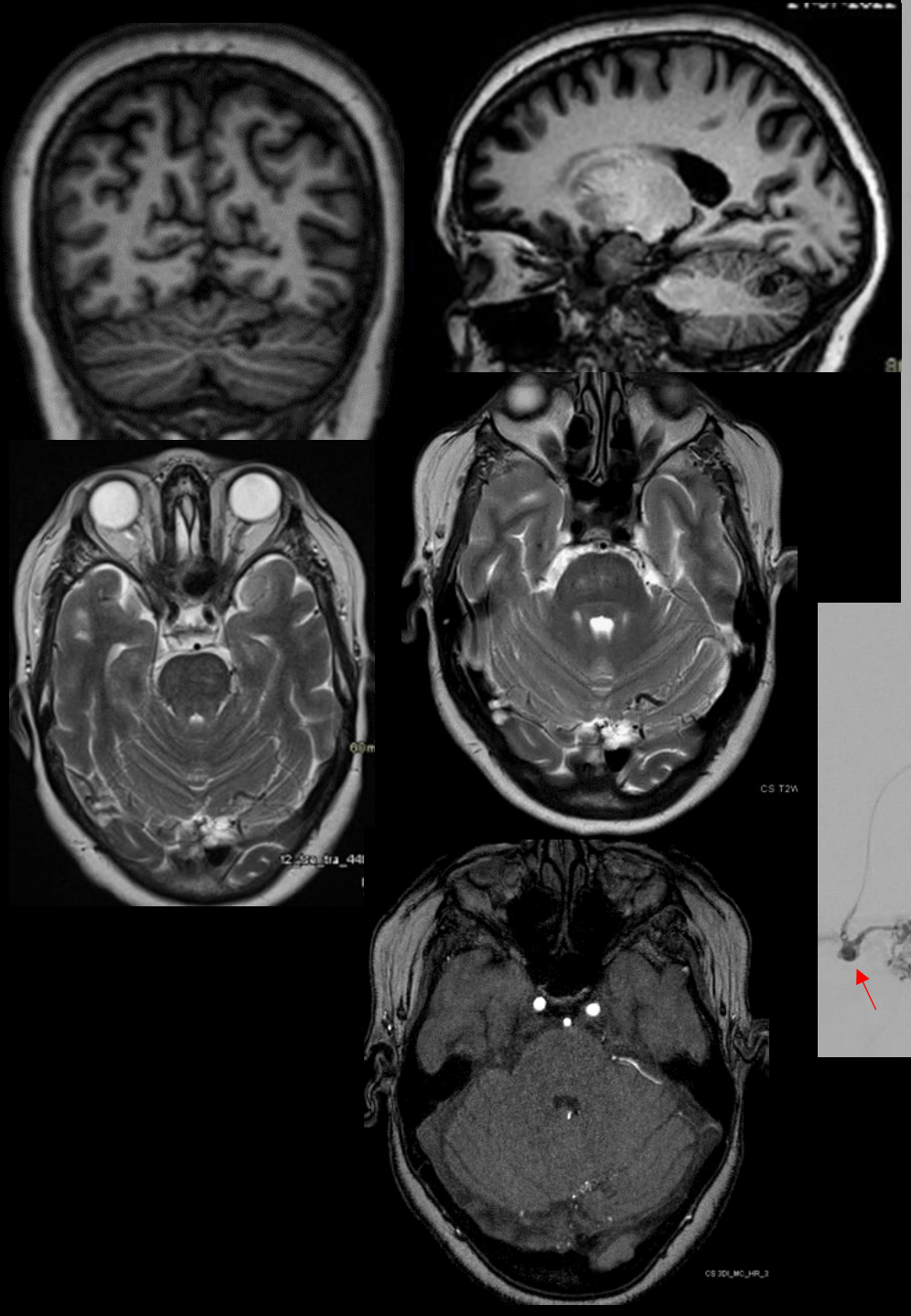
MAV INCIDENTAL
ANEURISMA INTRANIDAL

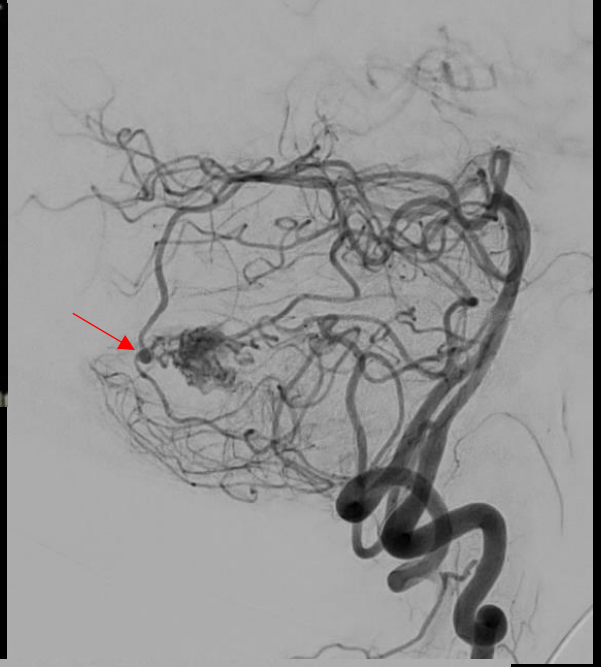


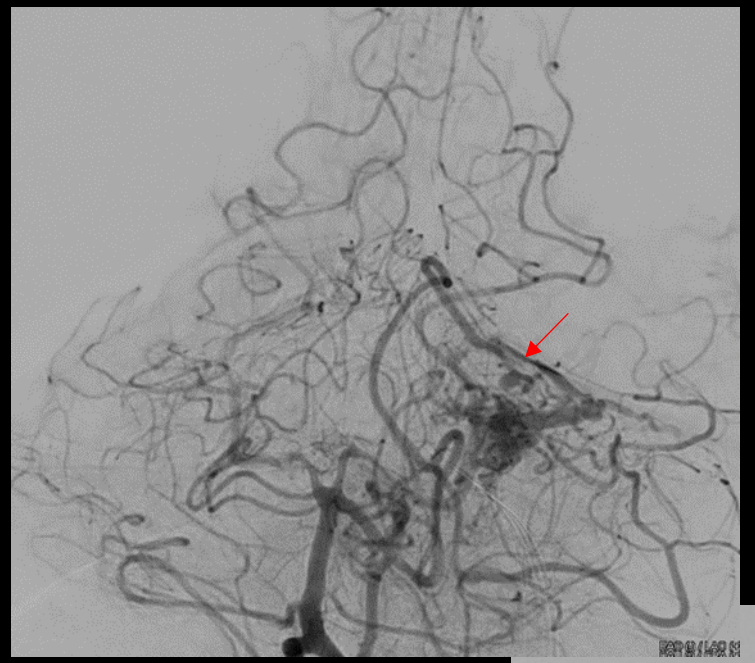




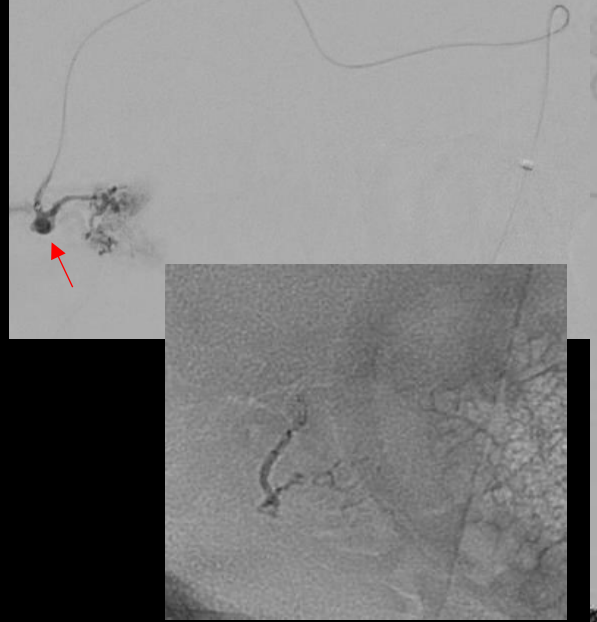


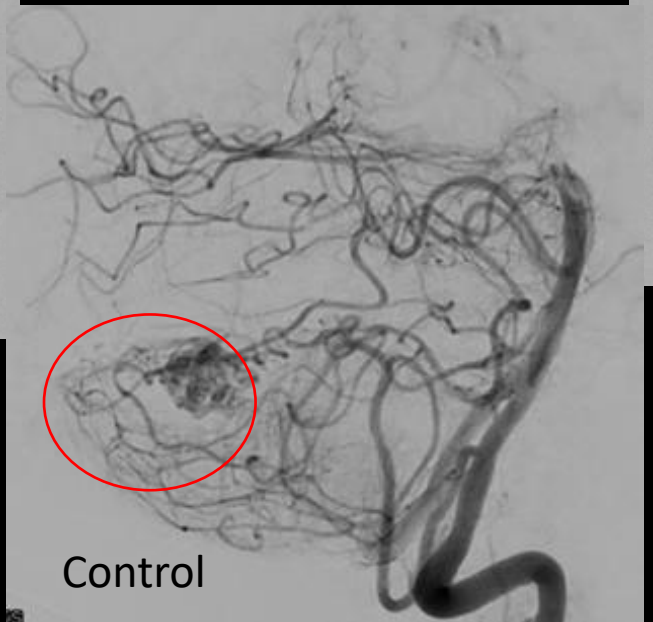




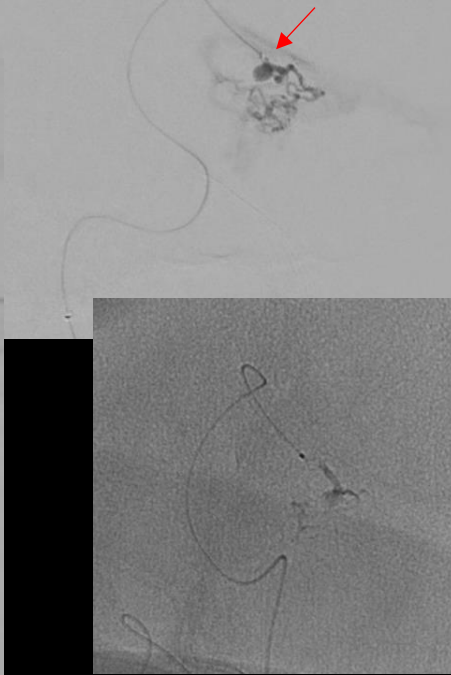


MAV INCIDENTAL
INFRATENTORIAL
ANEURISMA NIDAL





Control



FISTULA AV CEREBRAL

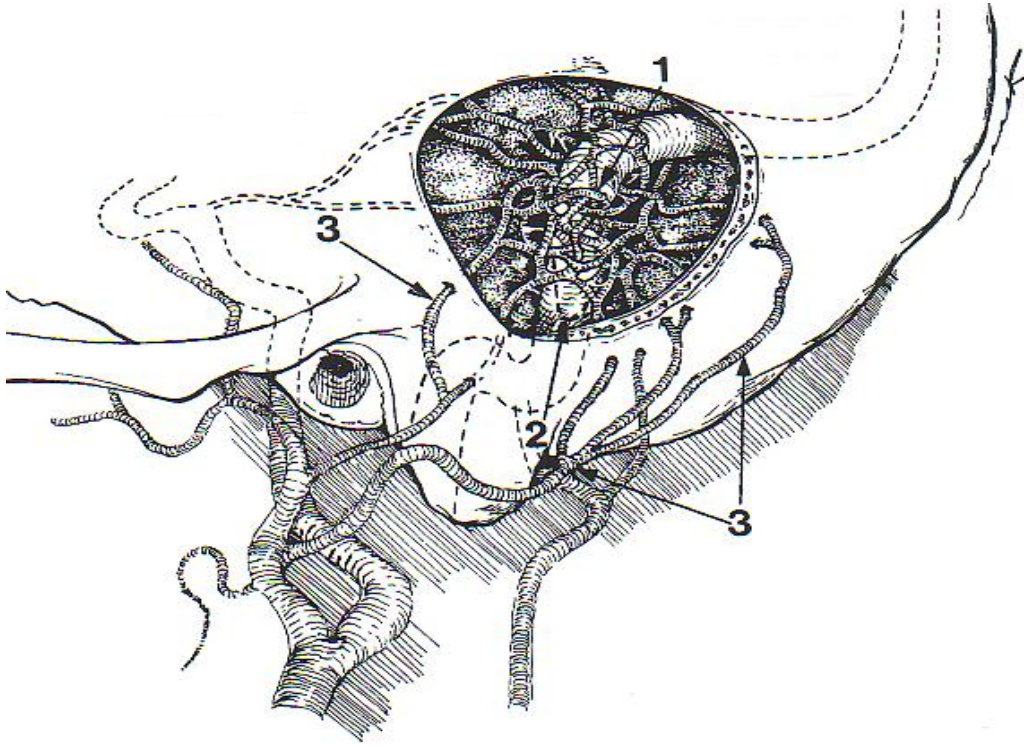
FISTULA ARTERIOVENOSA DURAL(FAVD)

FISTULA ARTERIOVENOSA PIAL O PARENQUIMATOSA

FAVD 10-15%

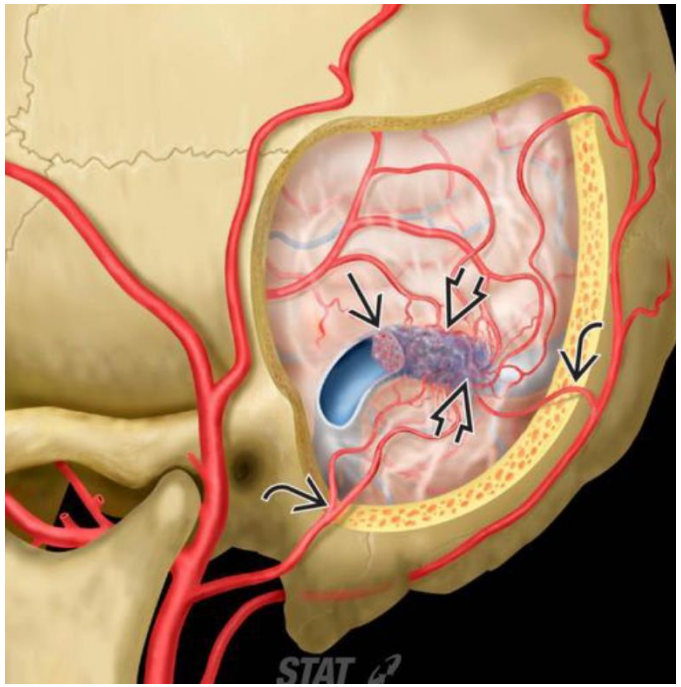
Conexiones anómalas entre arterias durales (ACE, ACI, AV) y senos venosos durales, venas meníngeas o venas corticales, sin nidus ni lecho capilar.

- 50-60 años.



ETIOPATOGENIA. FAVD

- Trombosis venosa
- Traumatismo
- Craneotomía previa



CLINICA. FAVD

Depende de la localización y del patrón de drenaje venoso.

- Tínitus pulsatil (ST y SS)
- Proptosis, quemosis, oftalmoplejia, pérdida de agudeza visual (SC)
- Convulsiones, cefalea, demencia (hiperT venosa)
- Hemorragia FAVD con reflujo venoso cortical(RVC)

DIAGNÓSTICO. FAVD

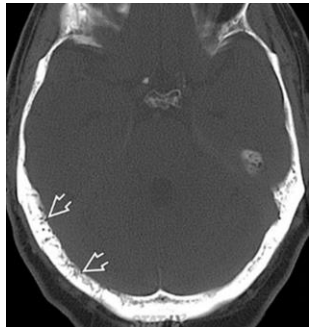
TC y RM evaluación diagnóstica primaria, relación de la FAVc con las estructuras intracraneales y evaluación no invasiva de las características dependientes del tiempo (fase arterial, venosa)

Sin embargo **DSA** es el **gold standard** para el estudio de la angioarquitectura, clasificación y planificación del tratamiento de las FAVc

DIAGNÓSTICO-TC . FAVD

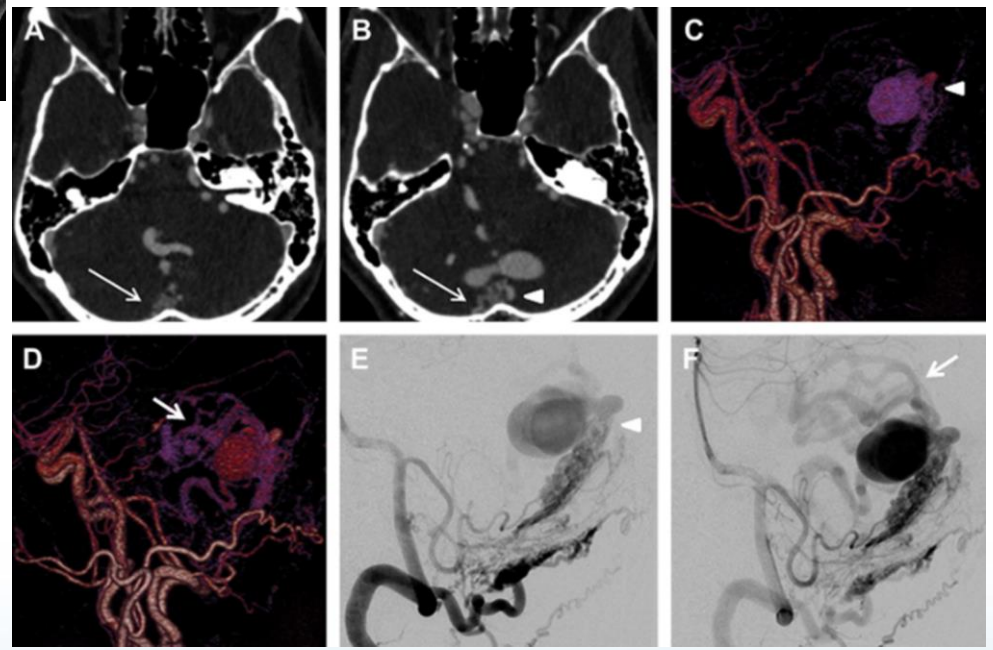
TC sinC:

- Canales vasculares transóseos
- Edema vasogénico por congestión venosa
- Hemorragia



TC conC:

- “Clusters” de vasos rodeando el seno dural
- Senos venosos/v.drenaje ensanchados
- Vena oftálmica superior dilatada(FCC)



Angio CT:

- Realce asimétrico de los senos venosos (92%)
- Arterias de ACE dilatadas (79%)
- Realce de los vasos transóseos (79%)
- 4D CTA dinámico, resolución espacial insuficiente para reemplazar a la DSA en la planificación del tratamiento.

DIAGNÓSTICO-RM. FAVD

RM

- Vacíos de señal por “microfístulas” en senos transverso/sigmoide trombosados

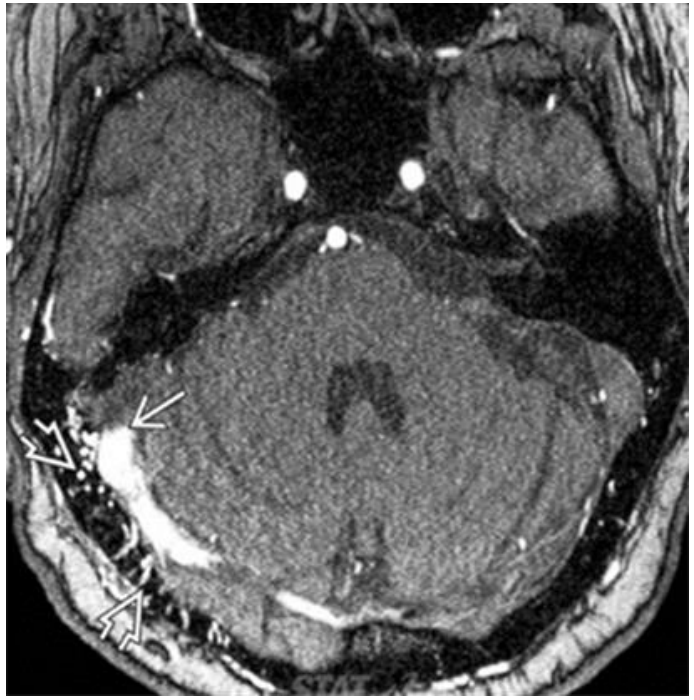
- Venas corticales dilatadas sin nido intraparenquimatoso

- Hiperintensidad en sustancia blanca por Hipertensión venosa, Infarto venoso, Hemorragia

- SWI puede detectar shunt AV en FAVD, representada como una señal venosa hiperintensa

- Venas oftálmicas superiores ensanchadas(FCC).

- Congestión venosa medular (FAVD grado V)



ARM

- Detección de vasos nutricios arteriales, dirección del flujo en las venas de drenaje y en los senos venosos.

- La ARM dinámica 3T, identifica las fases arterial precoz, arterial, parenquimatosa y venosa temprana, y las arterias nutricias, las venas de drenaje o la presencia de RVC, siendo adecuada para el diagnóstico y el seguimiento de las FAVD.

DIAGNÓSTICO-ARTERIOGRAFÍA CEREBRAL. FAVD

- Gold standard para la detección y clasificación de FAV

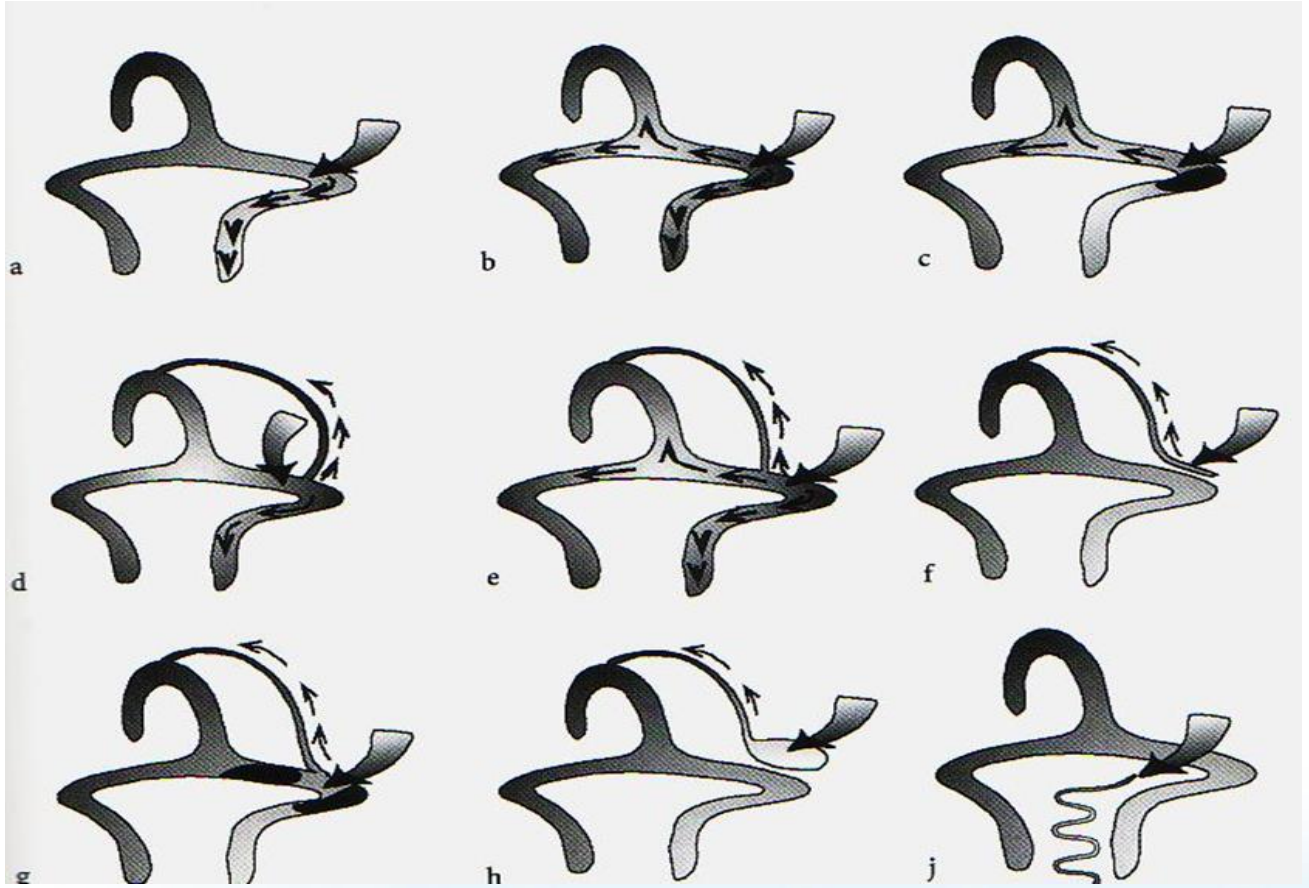
- Resolución espacial submilimétrica

- Planificación previa al tratamiento endovascular, primera línea de tratamiento de las FAVD.

- Inyecciones selectivas, con un estudio detallado de los puntos fistulosos, aportes arteriales, drenajes venosos, llenado temprano del seno venoso dural y reflujo venoso cortical.

FAVD.CLASIFICACIÓN SEGÚN DRENAJE VENOSO

Cognard Classification of DAVF	
TYPE	**VENOUS DRAINAGE**
I	Anterograde sinus drainage
II a	Retrograde sinus drainage
II b	Retrograde CVR
II a+b	Retrograde sinus drainage and CVR
III	CVR only without venous ectasia
IV	CVR only with venous ectasia
V	Spinal venous drainage



FAVD. PRESENTACIÓN CLÍNICA

-**FAVD TIPO I**

Acúfeno pulsatil

-**FAVD TIPO IIa**

Hipertensión intracraneal (cefalea, deficit visual, diplopia, papiledema)

RVC

-**FAVD TIPO IIb-IIa+b**

Hipertensión intracraneal. Riesgo de hemorragía 10%

-**FAVD III Y IV**

Tentoriales o fosa craneal anterior.

Hipertensión intracraneal. Riesgo de hemorragía 40-60%

-**FAVD V**

Mielopatía por hipertensión venosa o hemorragias medulares

FAVD. CLASIFICACIÓN SEGÚN LOCALIZACIÓN

TENTORIO 14%

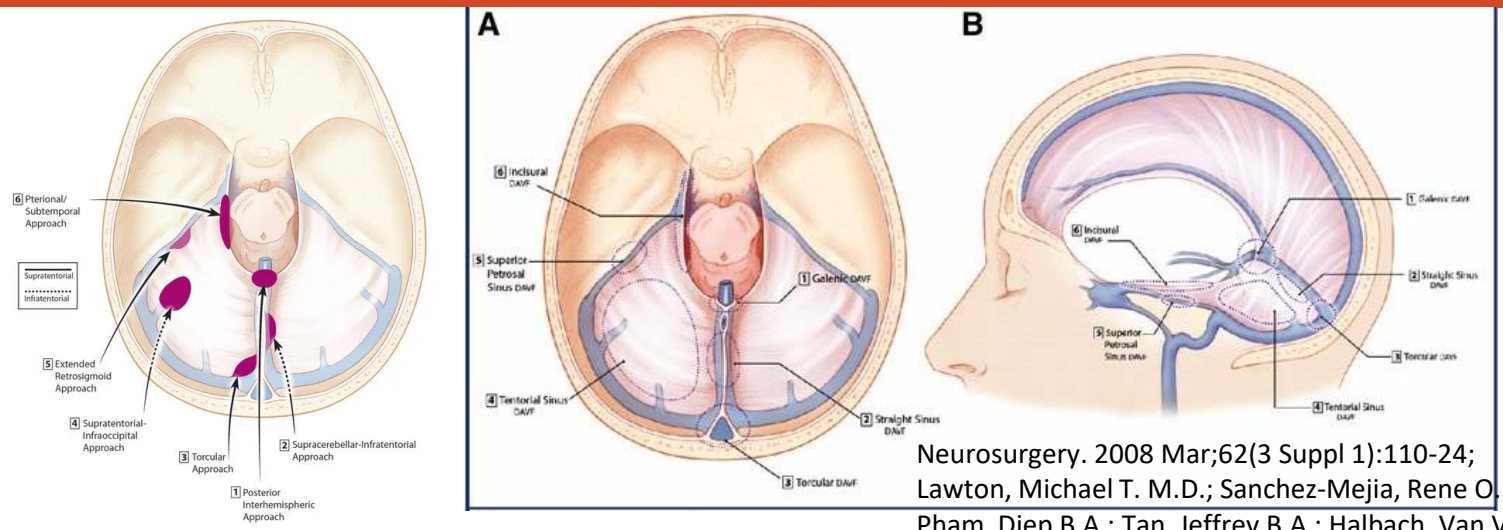
SENO CAVERNOSO 20%

SENO TRANSVERSO-SIGMOIDEO 55%

SENO LONGITUDINAL SUPERIOR 8%

FOSA CRANEAL ANTERIOR 3%

DAVF TENTORIALES




VASCULAR
Surgical Anatomy and Technique

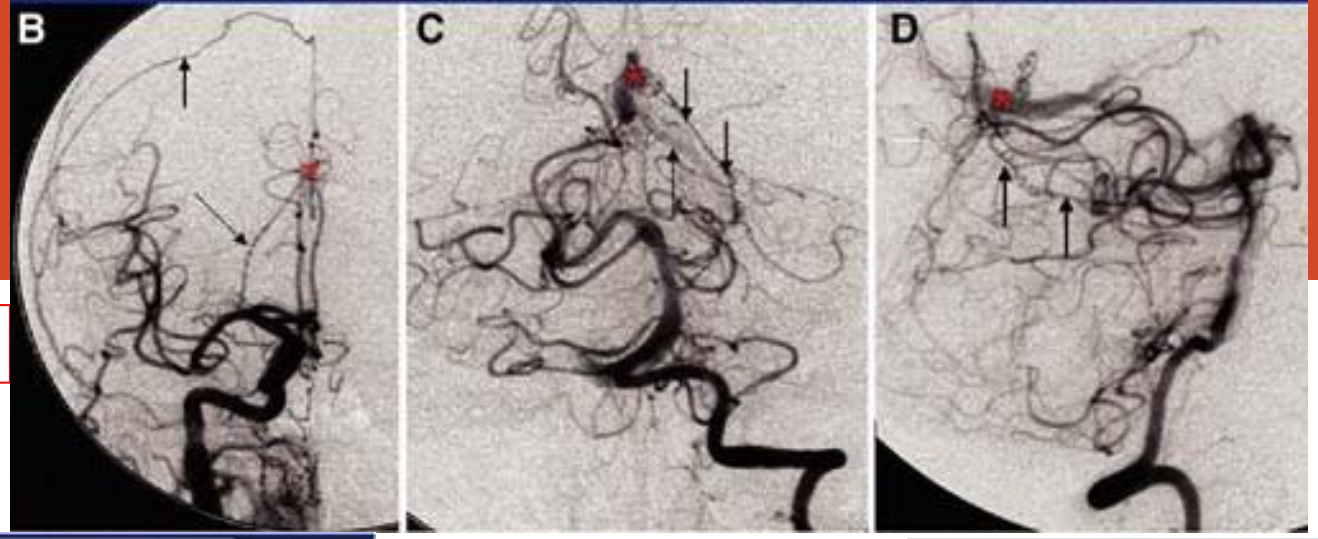
TENTORIAL DURAL ARTERIOVENOUS FISTULAE: OPERATIVE STRATEGIES AND MICROSURGICAL RESULTS FOR SIX TYPES

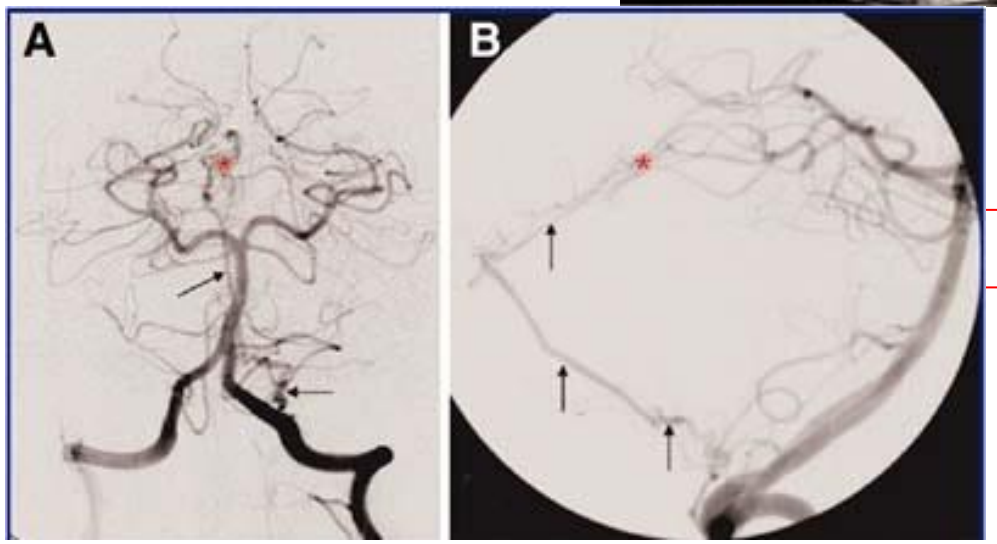
Neurosurgery. 2008 Mar;62(3 Suppl 1):110-24;
Lawton, Michael T. M.D.; Sanchez-Mejia, Rene O. M.D.;
Pham, Diep B.A.; Tan, Jeffrey B.A.; Halbach, Van V. M.D.

TABLE 1. Types of tentorial dural arteriovenous fistulae

Dural arterio-venous fistulae	Type	Patients, no. (percentage)	Location	Dural base	Venous sinus	Venous drainage
Galenic	1	7 (23%)	Midline	Anterior falcotentorial junction	Vein of Galen	Supra- and infra-tentorial
Straight sinus	2	8 (26%)	Midline	Middle falcotentorial junction	Straight sinus	Infratentorial
Torcular	3	3 (10%)	Midline	Posterior falcotentorial junction	Torcula	Supratentorial
Tentorial sinus	4	3 (10%)	Paramedian	Tentorium	Tentorial sinus	Supratentorial
Superior petrosal sinus	5	8 (26%)	Lateral	Petrotentorial junction	Superior petrosal sinus	Infratentorial
Incisural	6	2 (6%)	Paramedian	Tentorial incisura	None	Supratentorial

Tipo 1





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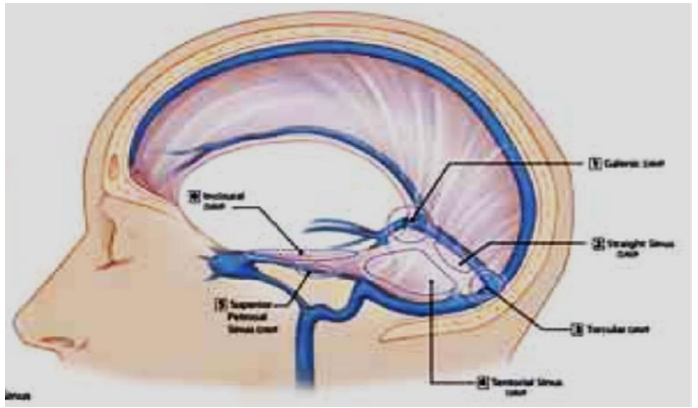

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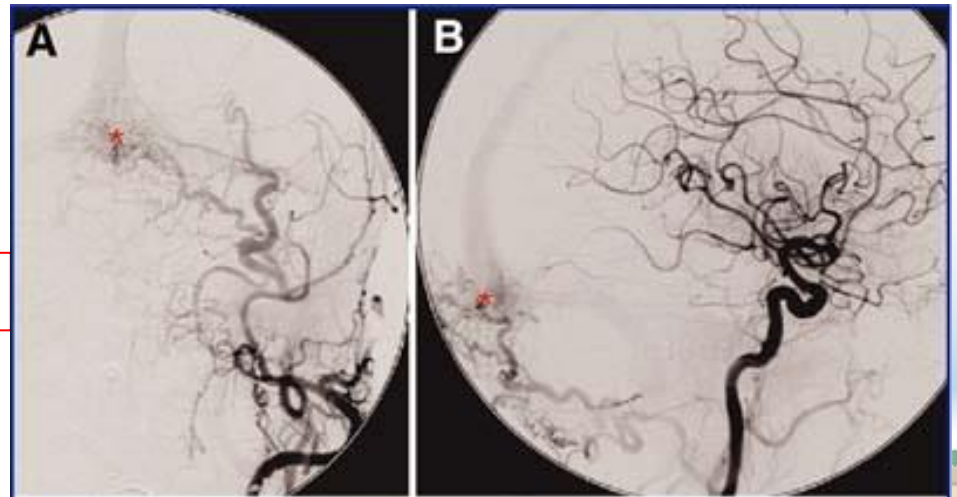

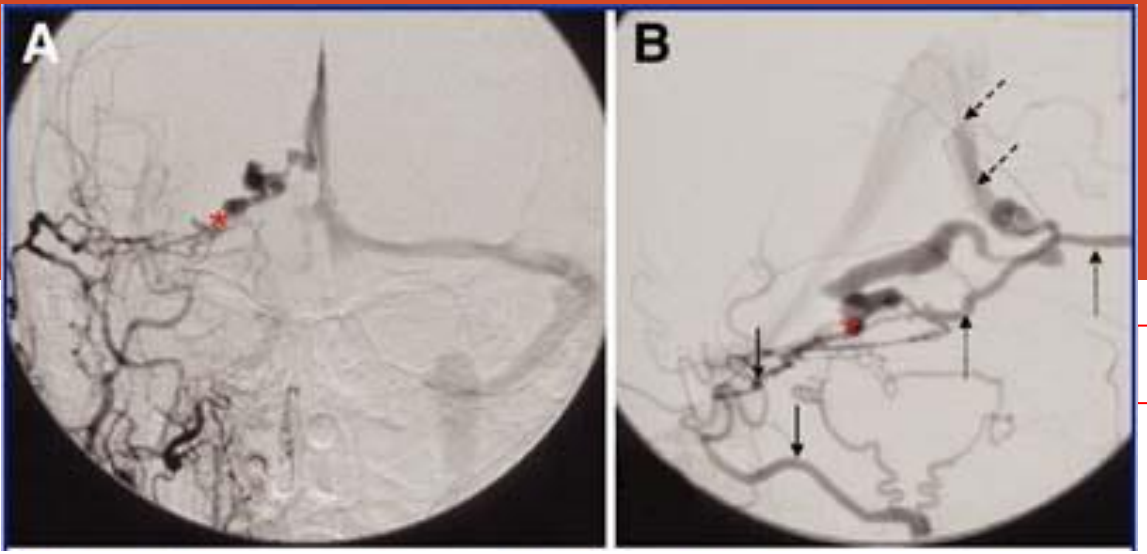

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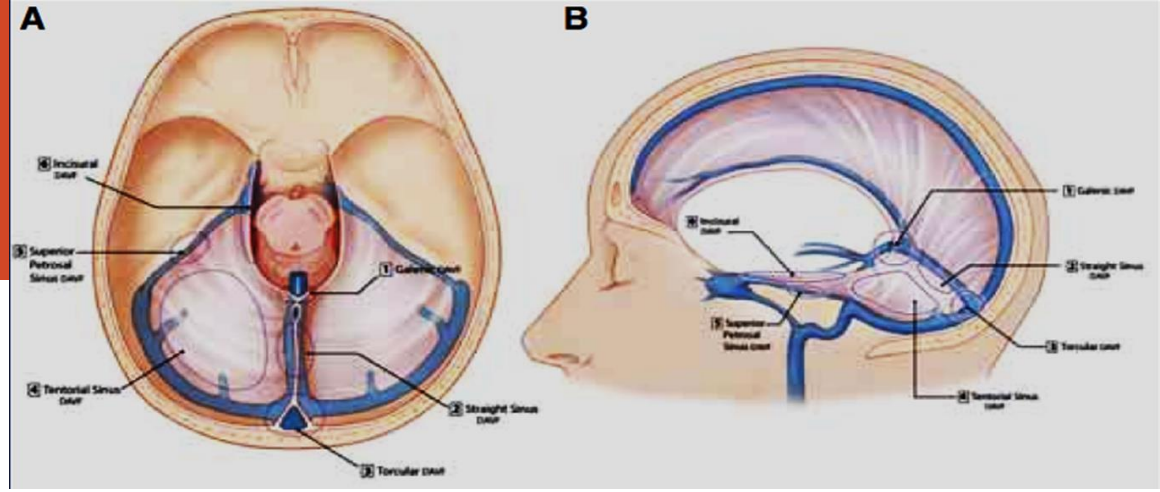
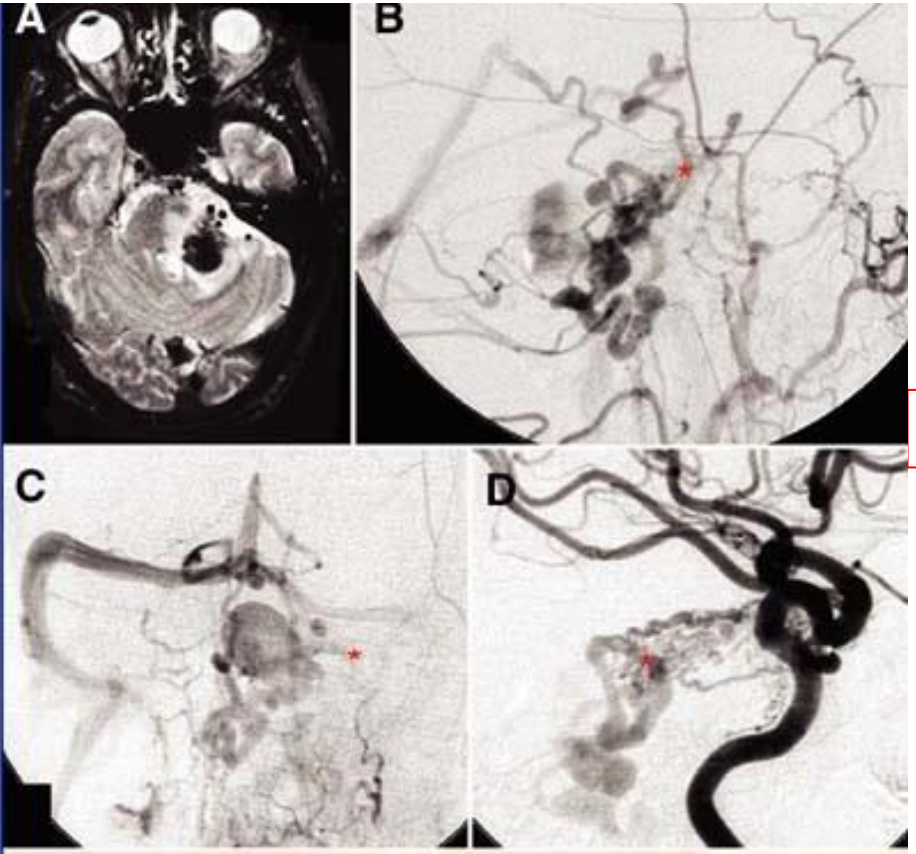
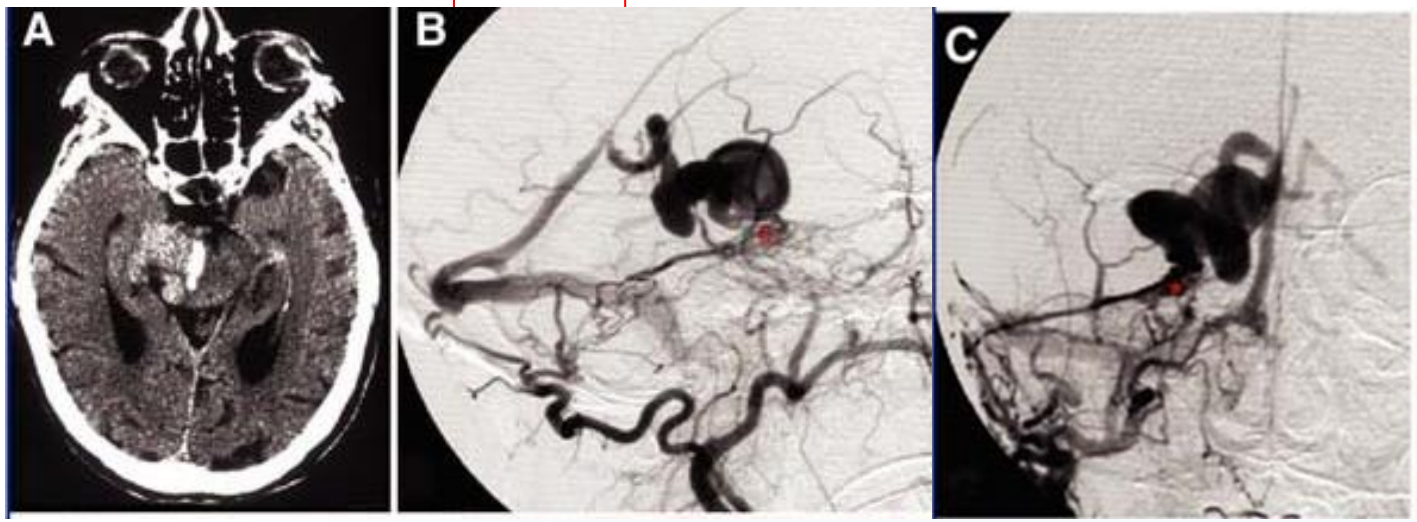




Tipo 5

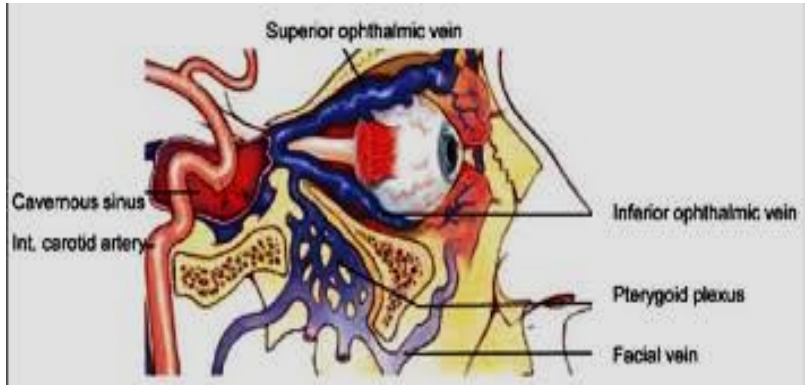
Tipo 6



FAVD. FISTULAS CAROTIDO CAVERNOSAS(FCC)

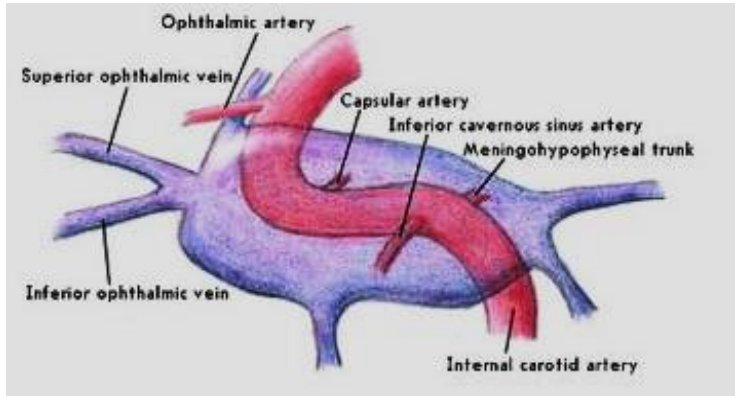
FCC: conexiones anómalas entre ACI y/o ACE y el seno cavernoso

El seno cavernoso recibe el drenaje de VOS, VOI, VCM superficial, seno esfenoparietal y drena a SPS, SPI y plexo pterigoideo.



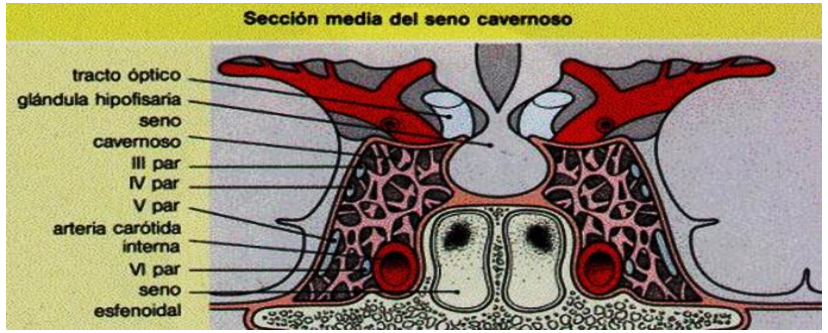
Barrow las clasificó en 4 tipos:

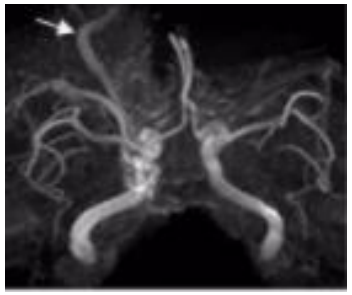
- Tipo A: Directas.
- Tipo B: Dural con aporte de la ACI.
- Tipo C: Dural con aporte de la ACE.
- Tipo D: Dural con aporte de ambas.

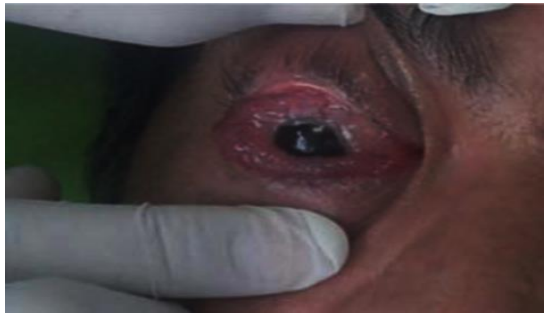


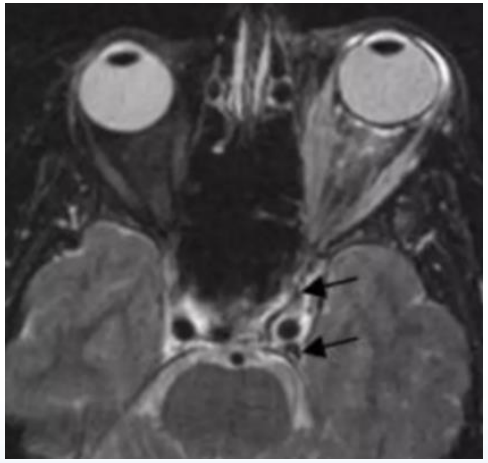
CLINICA. FCC

- Parálisis del VI par u oftalmolejía completa III,IV y VI par, por dilatación del seno cavernoso y compresión.









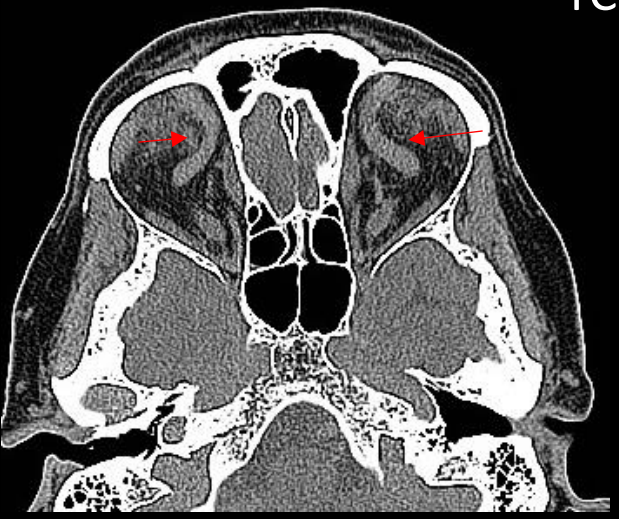
- Exoftalmos, quemosis, edema de MOE

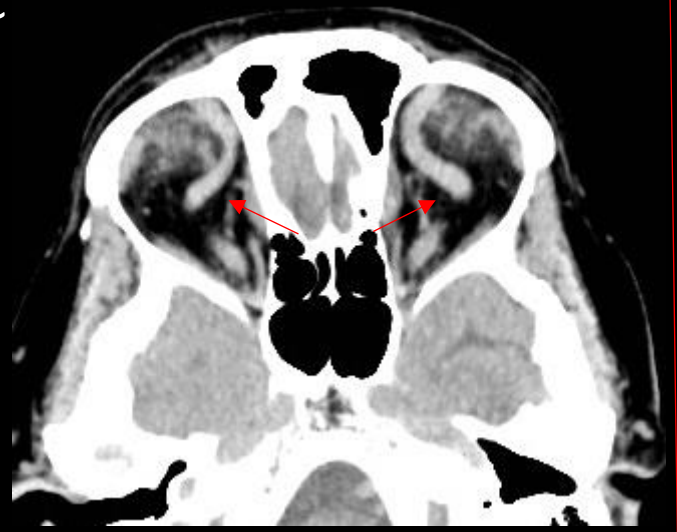
- HiperT venosa orbitaria, atrofia del nervio óptico, alteraciones visuales.

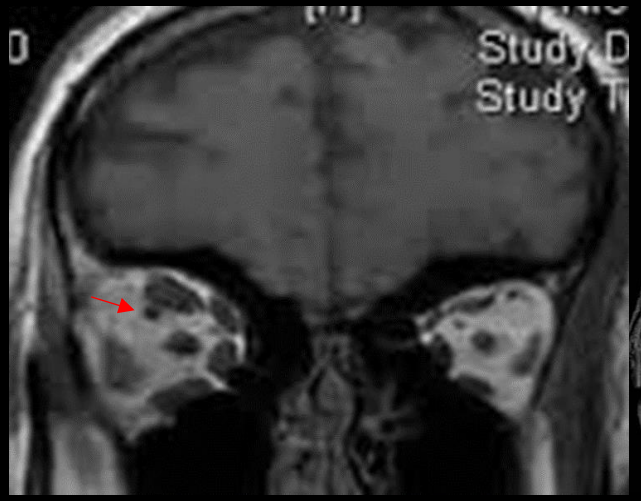
- HiperT venosa cerebral y edema, si RVC

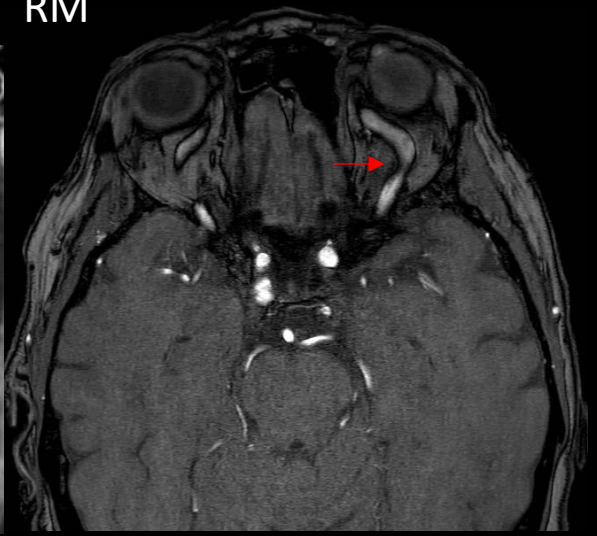
TC

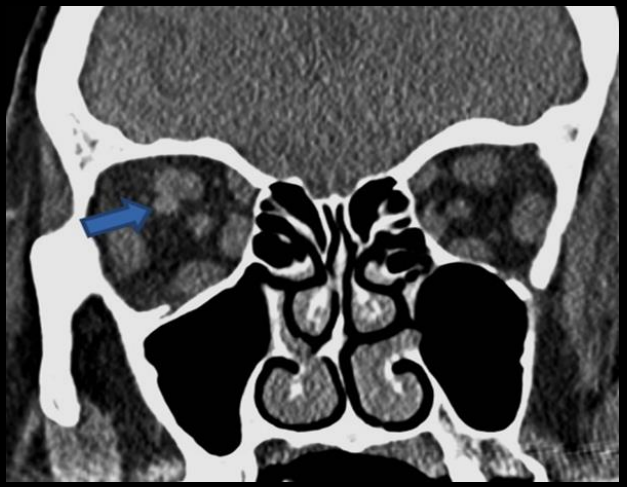
RM

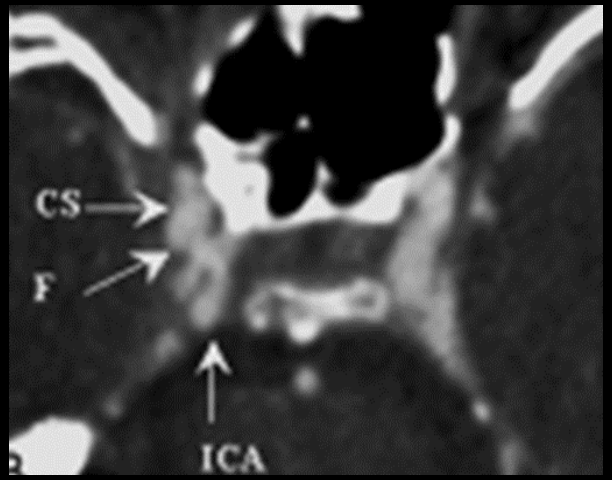


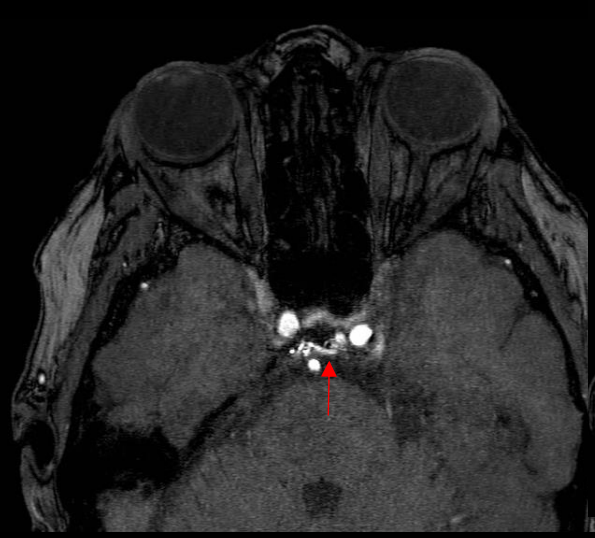


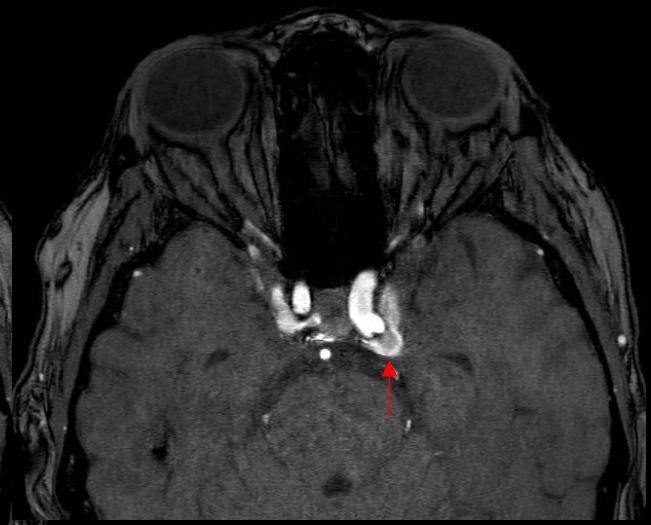








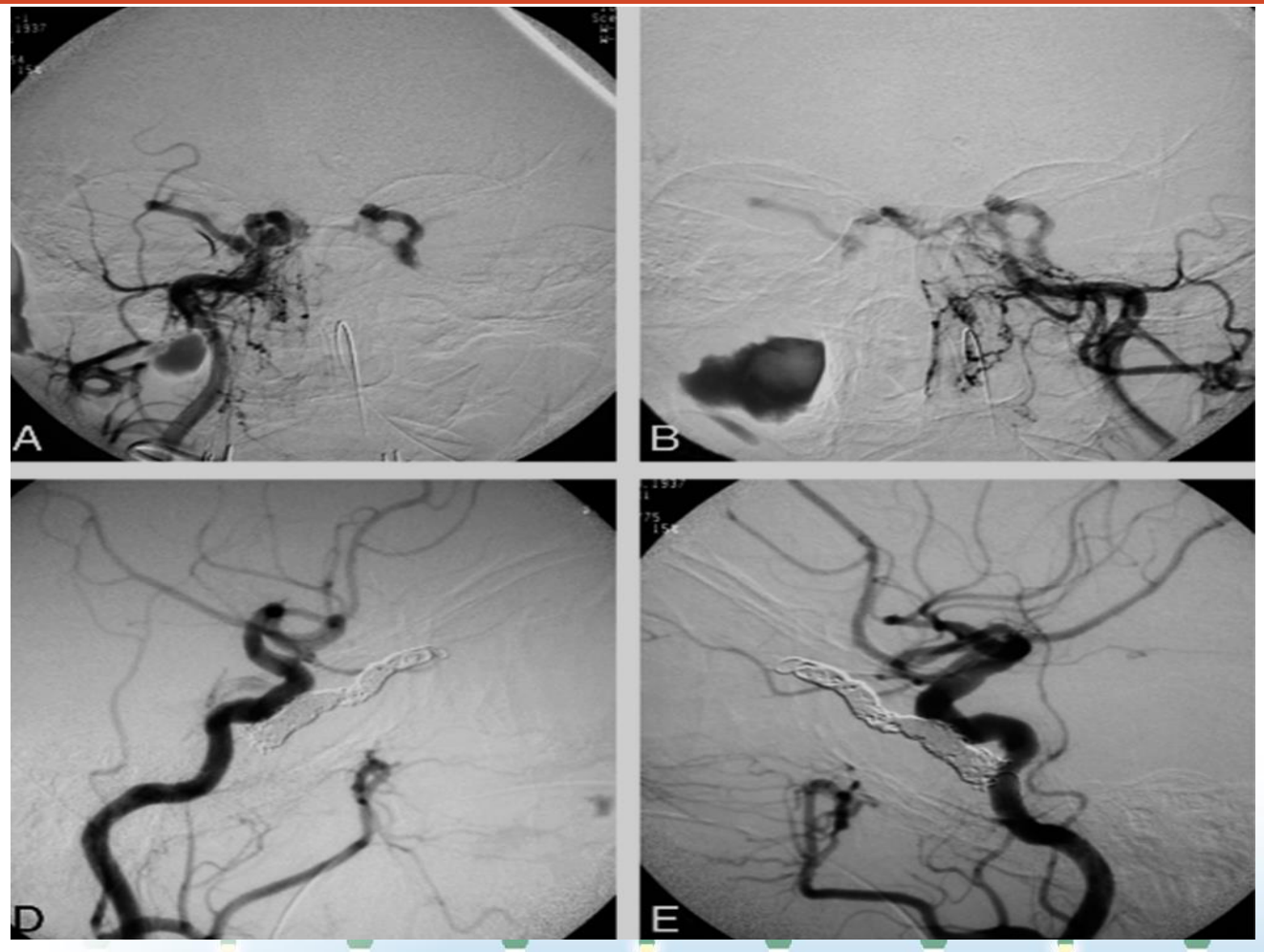


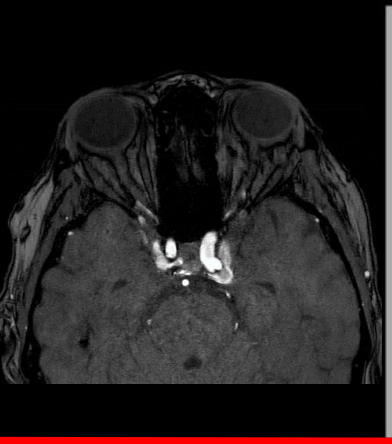
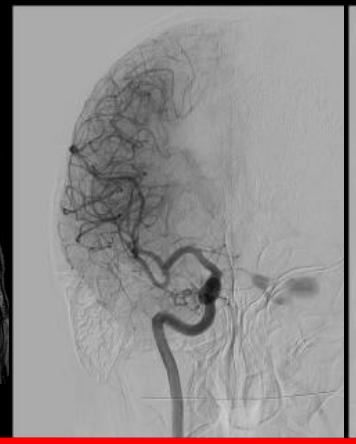
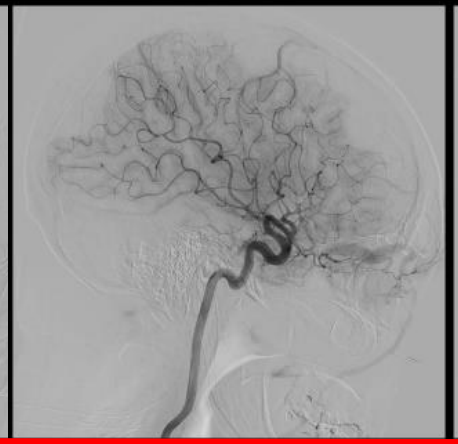
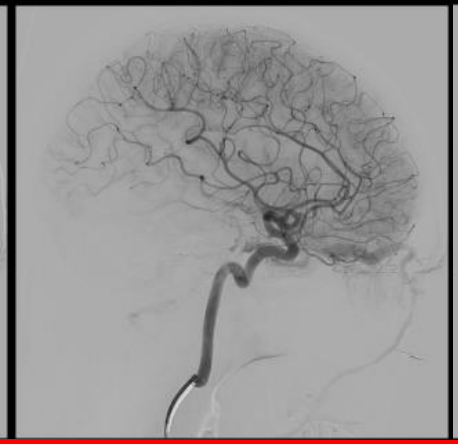
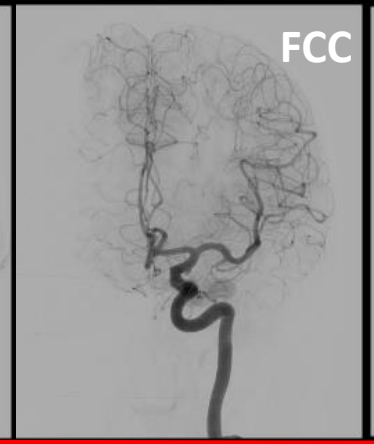
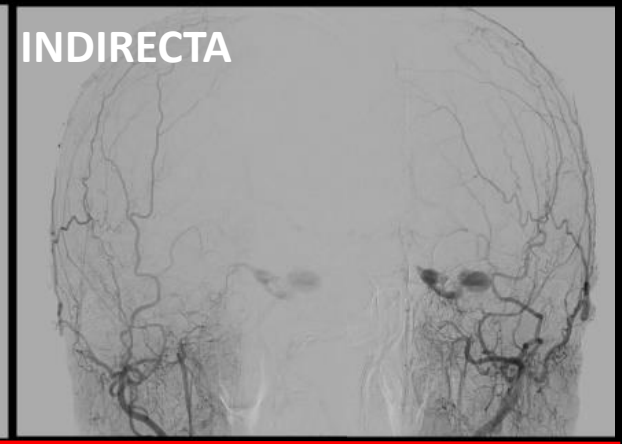


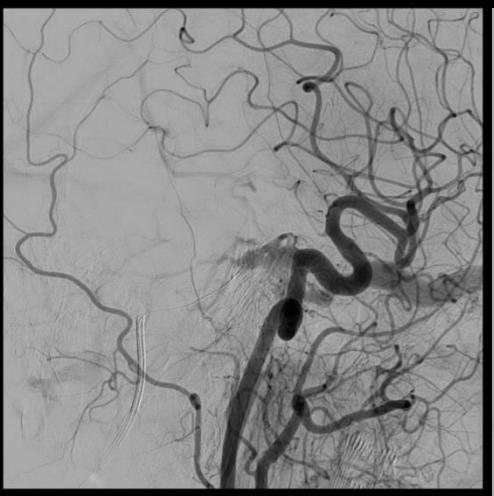
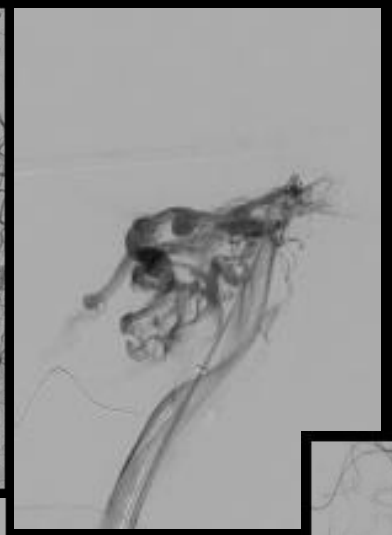
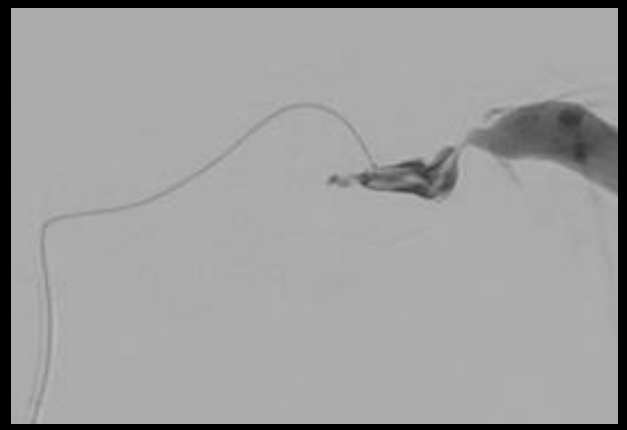
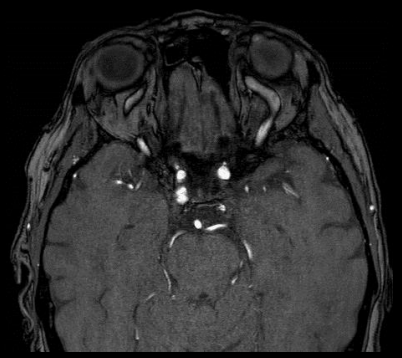
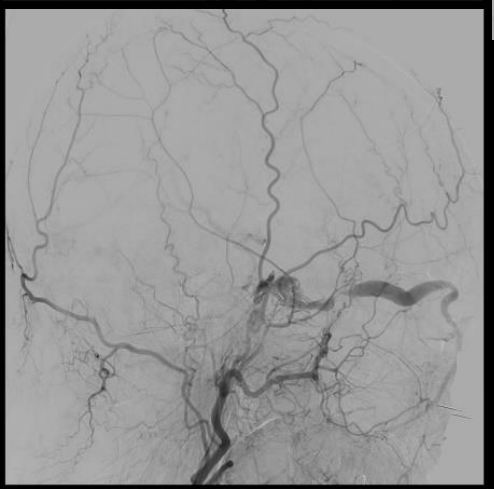
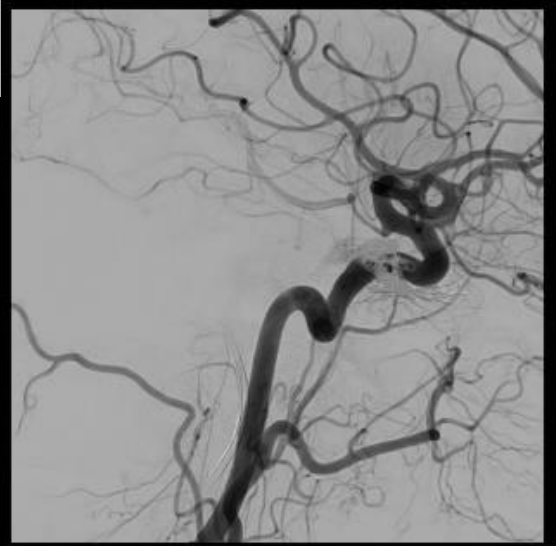
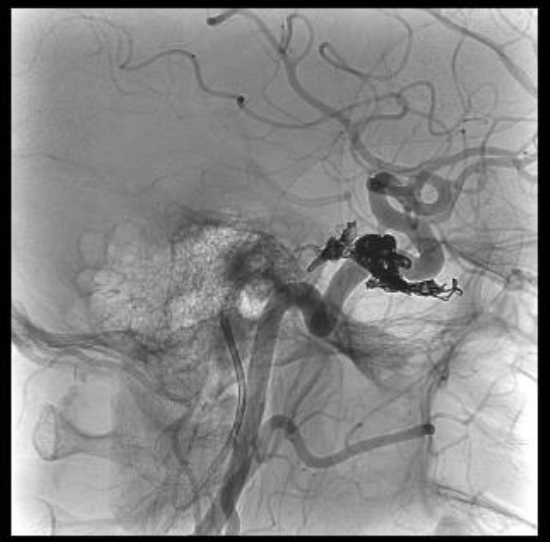
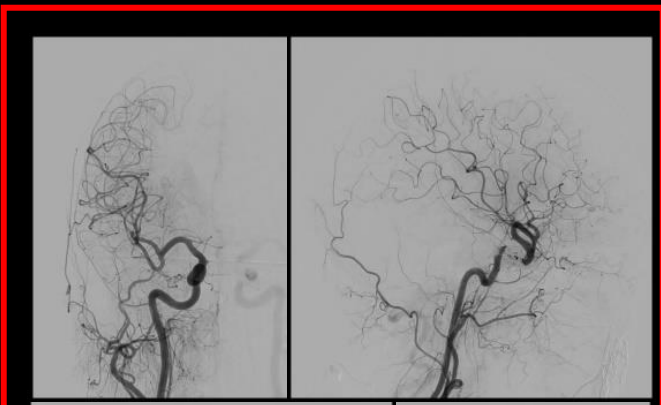
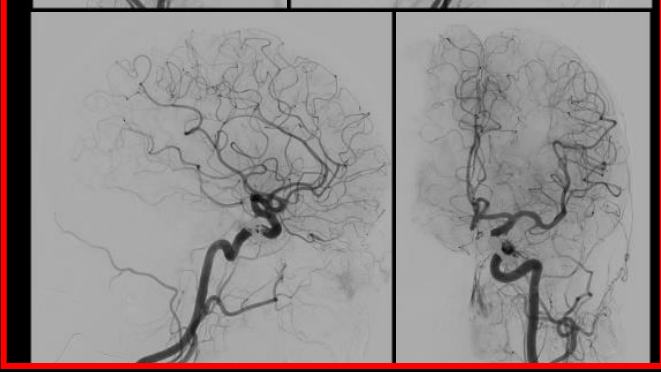
FCC INDIRECTAS

- Traumatismo
- Cirugía intracraneal
- Trombosis venosa

Embolización transvenosa con coils es el tratamiento de elección



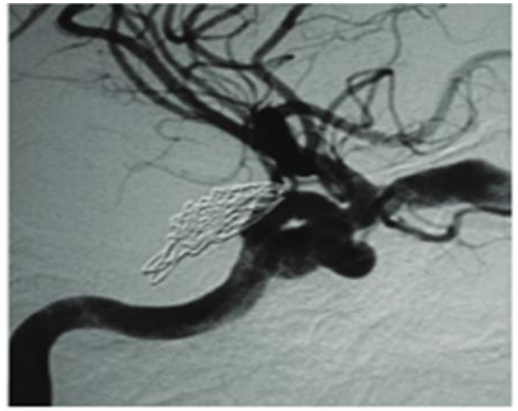











Proptosis y quemosis
ojo izquierdo






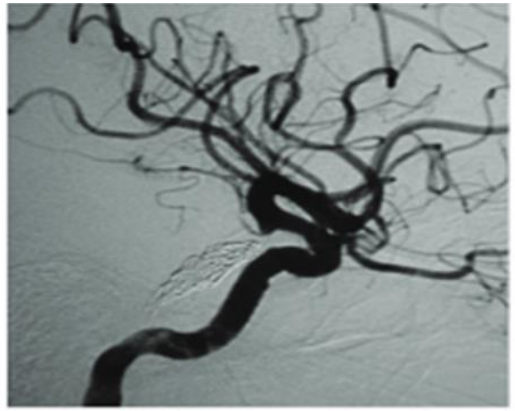
FCC DIRECTA

- Traumática
- Rotura de un aneurisma

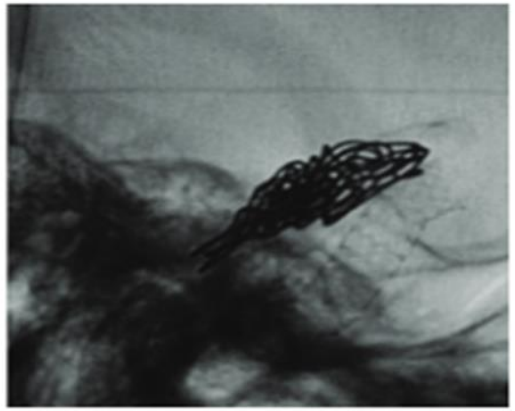
Tratamiento con coils, sacrificio de la ACI, stent tipo FD

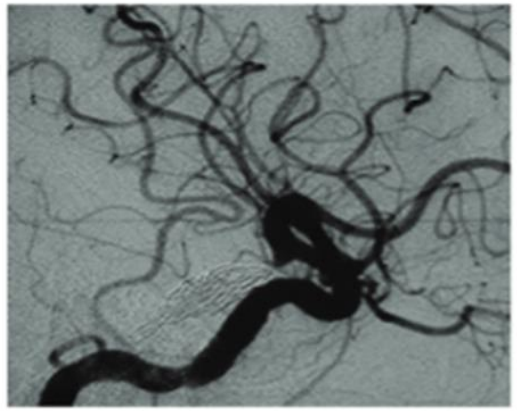


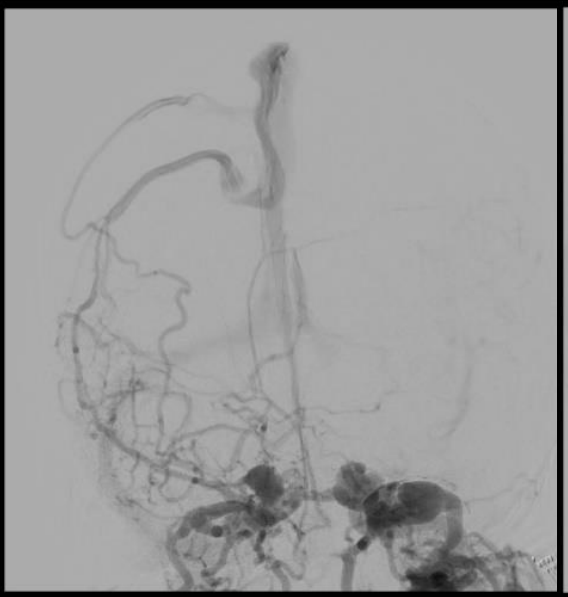
A

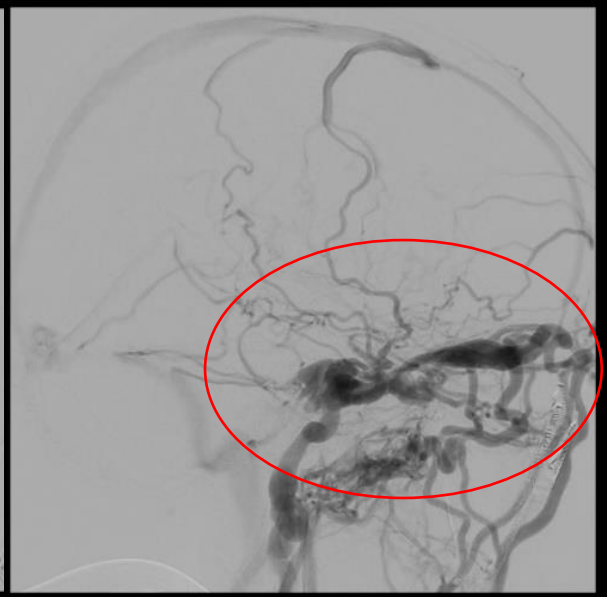


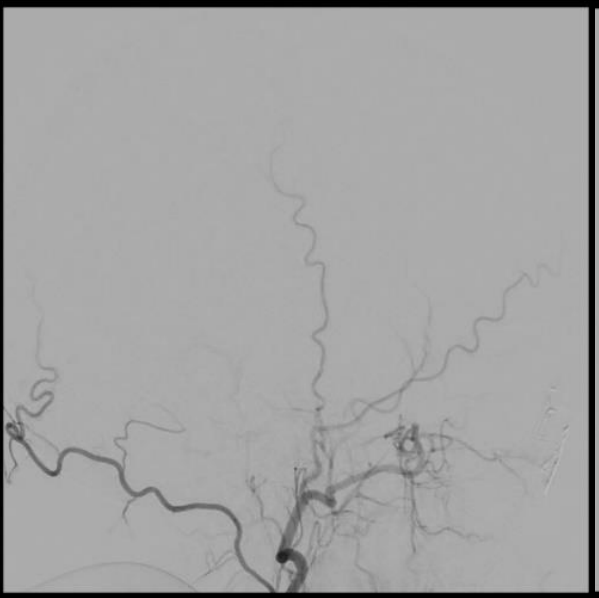
B

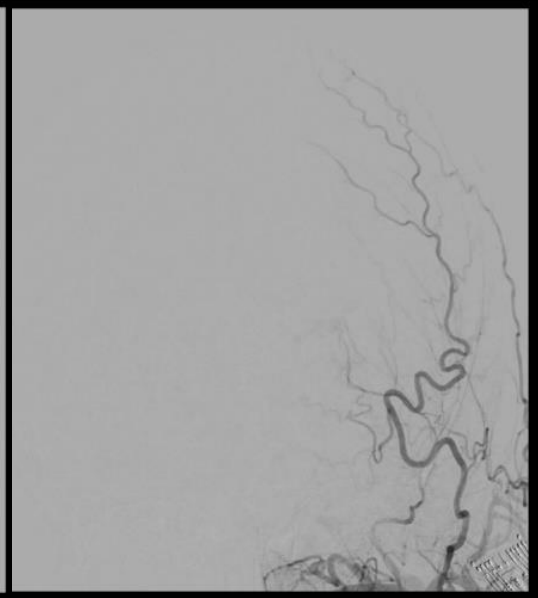












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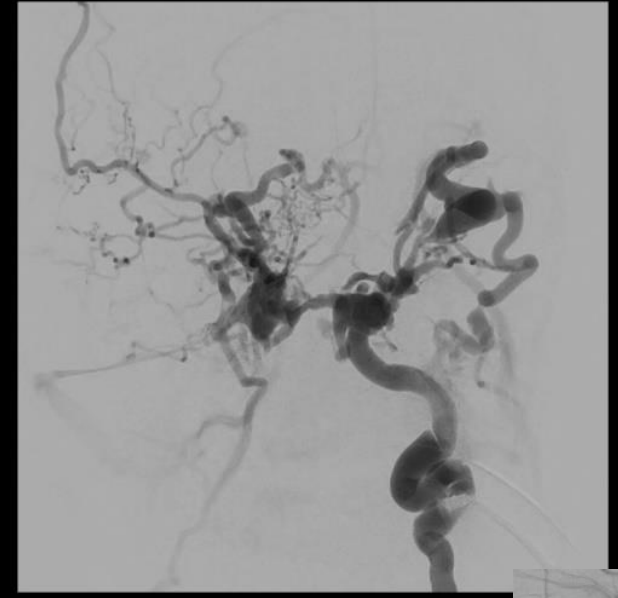

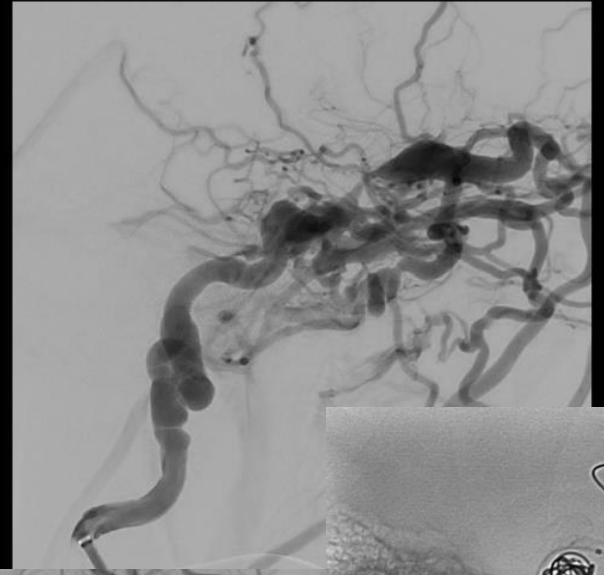

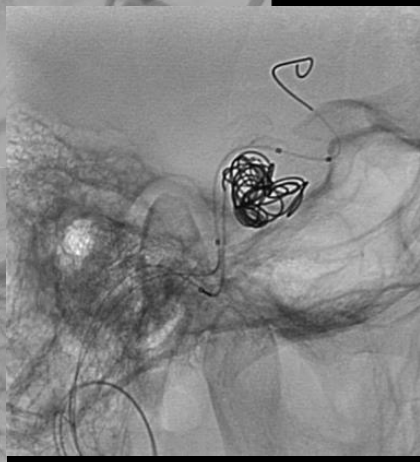

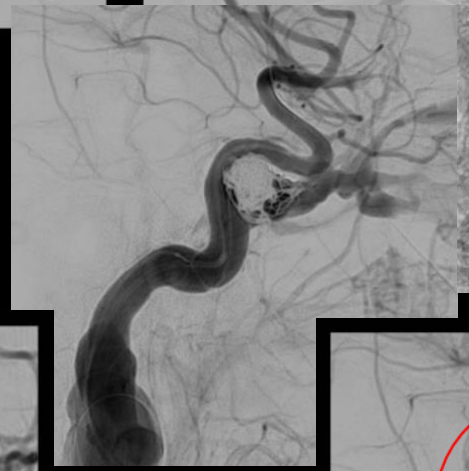

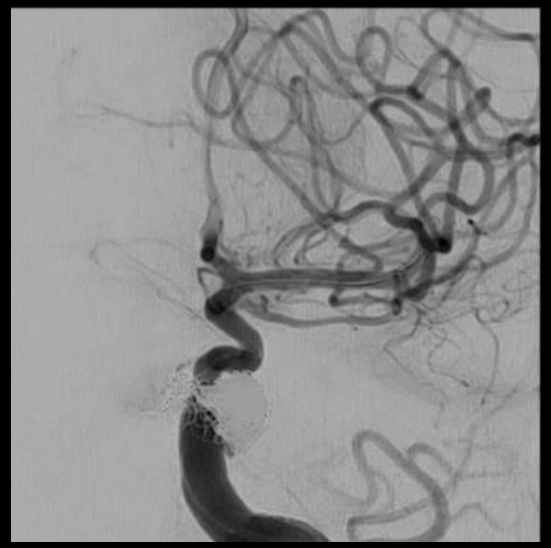

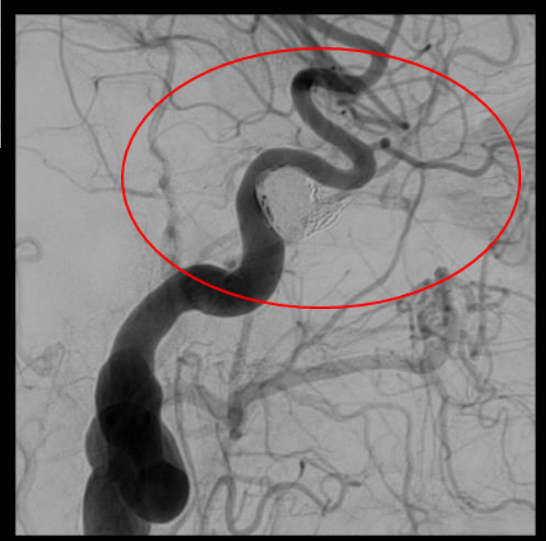

FAVD. TRATAMIENTO

TRATAMIENTO ENDOVASCULAR

RADIOCIRUGIA

CIRUGIA

El tratamiento de primera línea es el tratamiento endovascular asociado o no a cirugía

Standards

Standard and Guidelines: Intracranial Dural Arteriovenous Shunts

Seon-Kyu Lee,[1] Steven W Hetts,[2] Van Halbach,[2] Karel terBrugge,[3] Sameer A Ansari,[4] Barb Albani,[5] Todd Abruzzo,[6] Adam Arthur,[7] Michael J Alexander,[7] Felipe C Albuquerque,[8] Blaise Baxter,[9] Ketan R Bulsara,[10] Michael Chen,[11] Josser E Delgado Almandoz,[12] Justin F Fraser,[13] Don Frei,[14] Chirag D Gandhi,[15] Don Heck,[16] Muhammad Shazam Hussain,[17] Michael Kelly,[18] Richard Klucznik,[19] Thabele Leslie-Mazwi,[20] Ryan A McTaggart,[21] Philip M Meyers,[22] Athos Patsalides,[23] Charles Prestigiacomo,[24] G Lee Pride,[25] Robert Starke,[26] Peter Sunenshine,[27] Peter Rasmussen,[28] Mahesh V Jayaraman,[29] on behalf of the Standard and Guidelines Committee for the Society of Neurointerventional Surgery

Lee S-K, et al. J Neurointervent Surg 2015

RECOMMENDATIONS

1. All patients with suspected intracranial DAVS based on clinical presentation and/or non-invasive imaging findings should receive complete and high quality DSA in order to confirm and risk stratify their disease. (Class I; level of evidence C)
2. DAVS with high risk features (eg, CVR) should be treated promptly to reduce the potential risk of intracerebral hemorrhage, venous hypertensive encephalopathy, or other neurologic events. Endovascular treatment is considered as the preferred first-line treatment option with favorable anatomy. Open surgical treatment alone or combined endovascular and open surgical treatment should be considered for high-risk fistulas not curable by endovascular means alone. SRS should be reserved as an adjunctive and/or complementary option for aggressive and symptomatic DAVS. (Class I; level of evidence C)
3. Non-aggressive but symptomatic DAVS can be considered for definitive treatment. Endovascular treatment, open surgery, and SRS can be considered for this type of DAVS, but only if associated with very low treatment-related risk in view of the benign natural history of these lesions. (Class IIb; level of evidence C)
4. Non-aggressive asymptomatic (ie, incidental) DAVS lesions without CVR do not warrant active intervention and, if treatment is considered, treatment-related risk versus the natural history of the disease should be thoroughly discussed between the practitioner and patient. Nonetheless, these patients should be followed both clinically and with non-invasive imaging studies in regular fashion. An exception to this recommendation would be a patient who has become asymptomatic who was previously symptomatic, as a change in symptoms can portend a venous outflow thrombosis and, hence, potential change in fistula angioarchitecture and venous drainage pattern that would warrant re-evaluation with DSA. (Class I; level of evidence C)
5. SRS is a reasonably effective and safe treatment option. Thus, it could be considered as a viable option for DAVS that have a small compact shunt zone in patients who are not good candidates for endovascular or open surgical treatment or those who prefer a less invasive approach. (Class I; Level of Evidence C)
6. As a rare and incompletely understood disease, intracranial DAVS warrants further scientific investigation both with regard to natural history and clinical course following treatment. Standardized reporting of angiographic and clinical features and development of multi-institutional data collection consortia would benefit our understanding and may improve clinical and surgical outcomes in the future.

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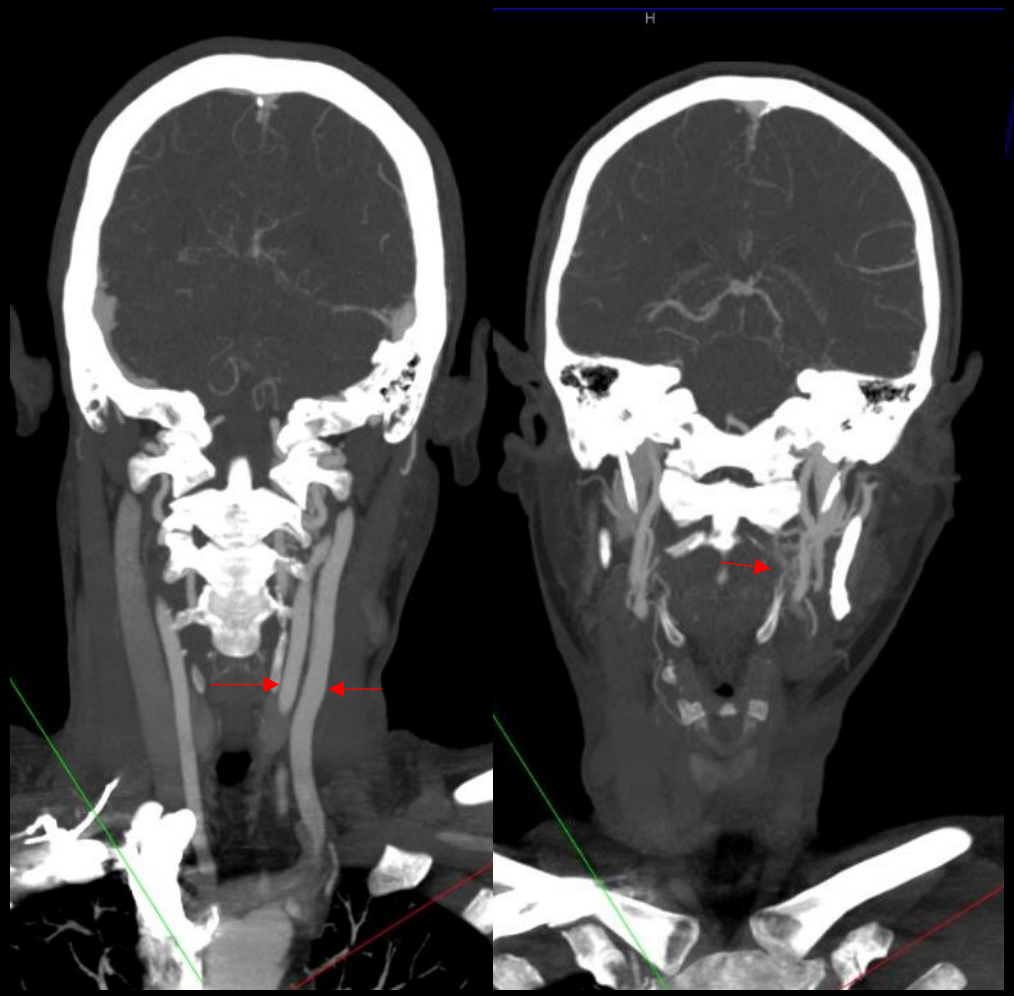

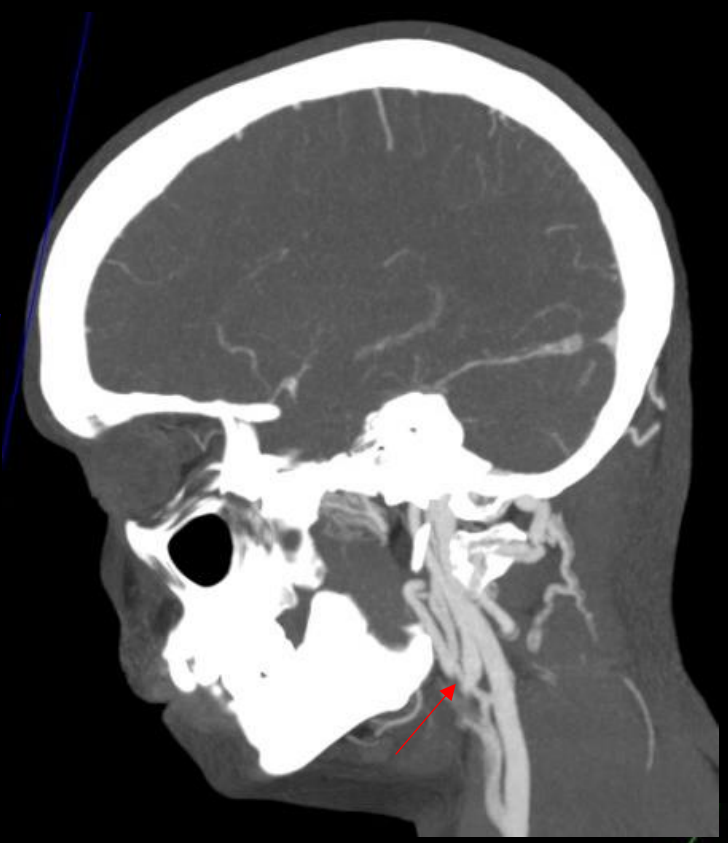

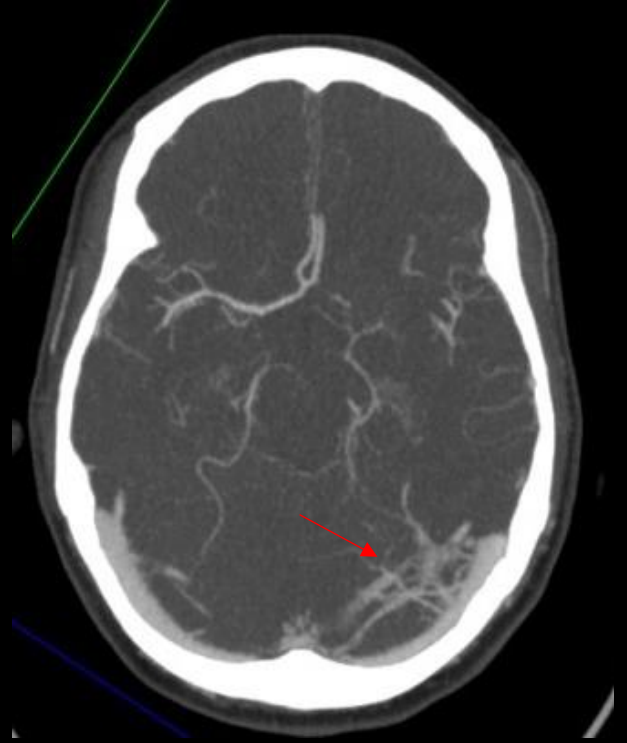

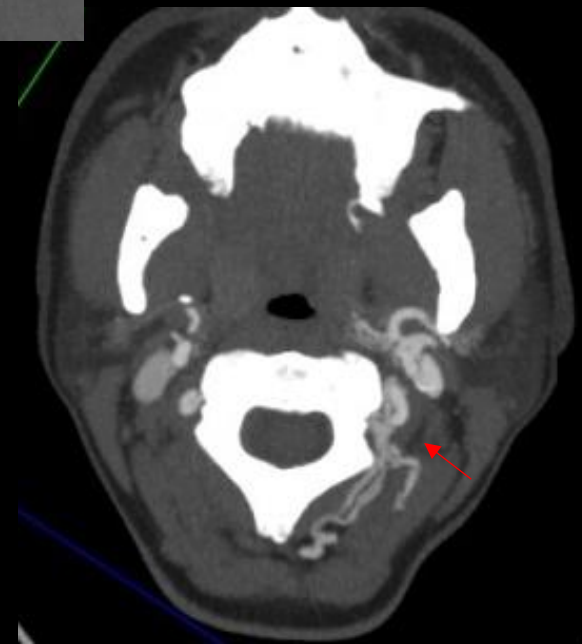

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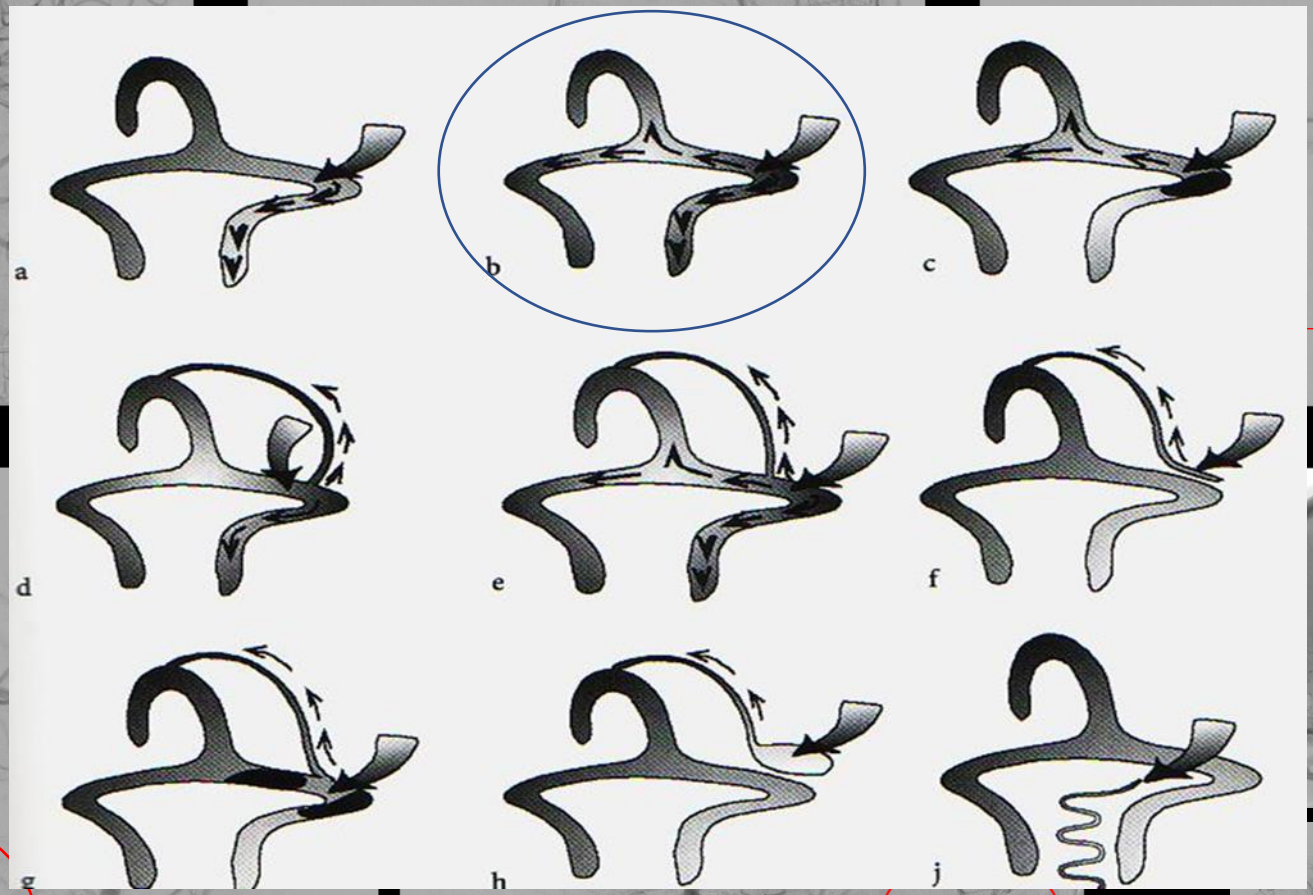

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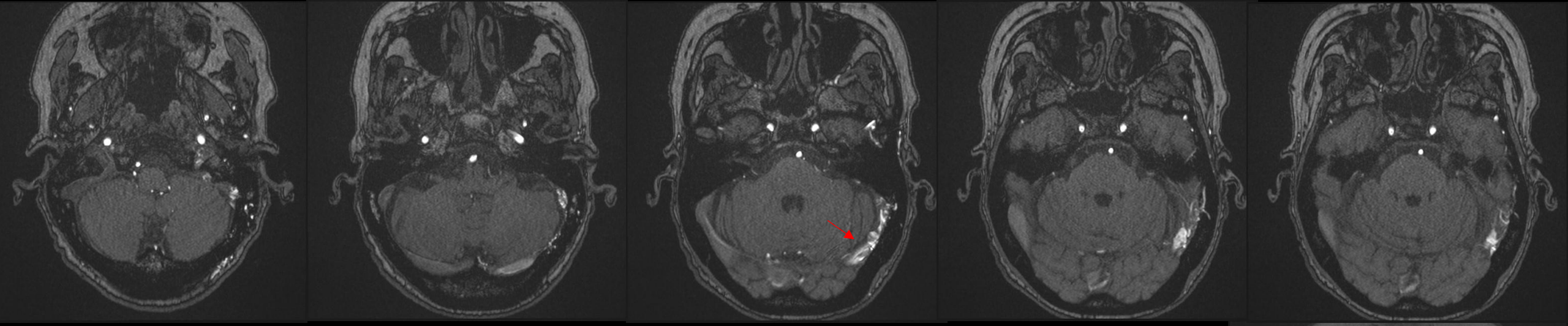

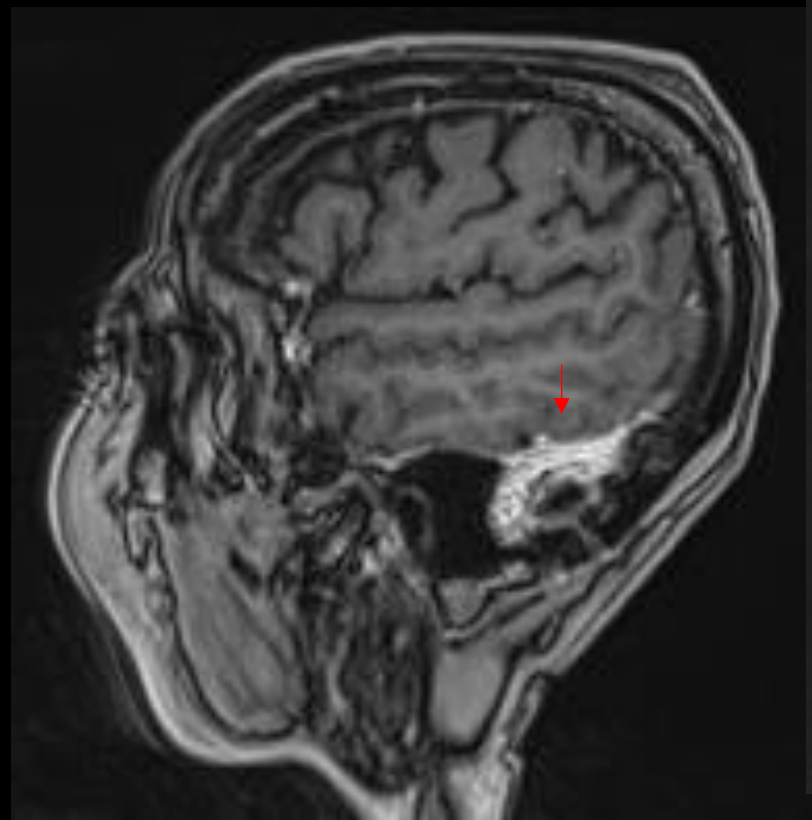

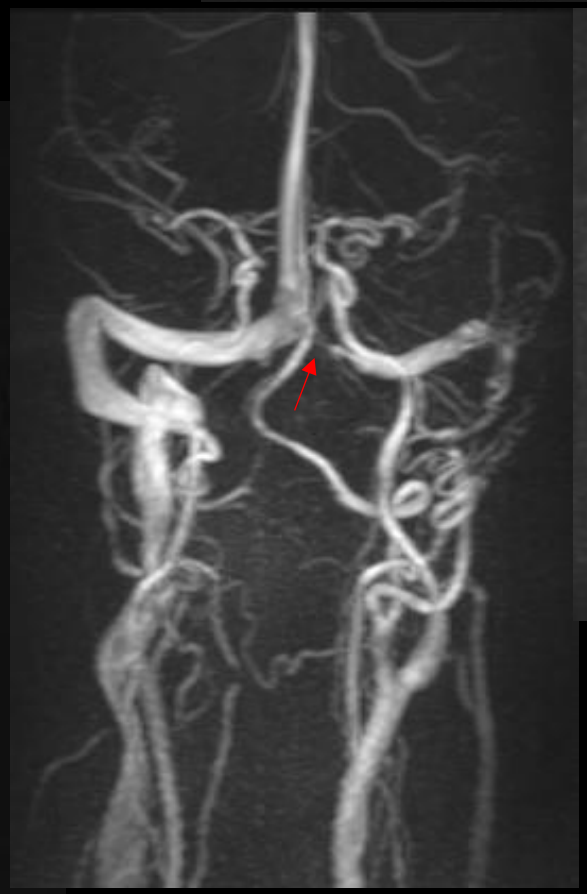

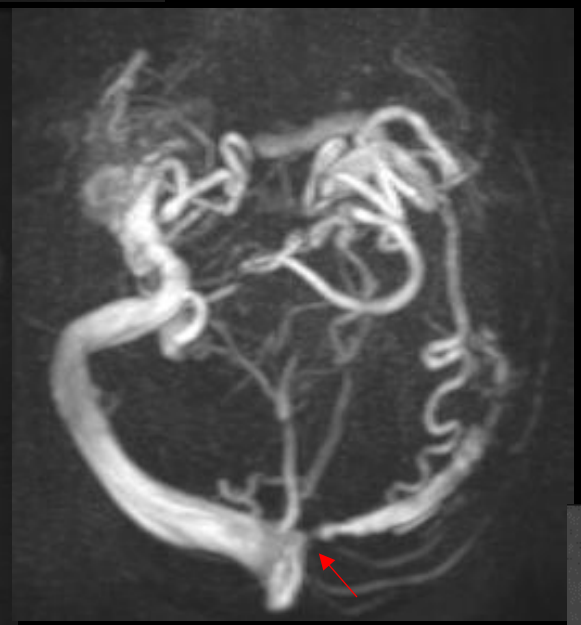

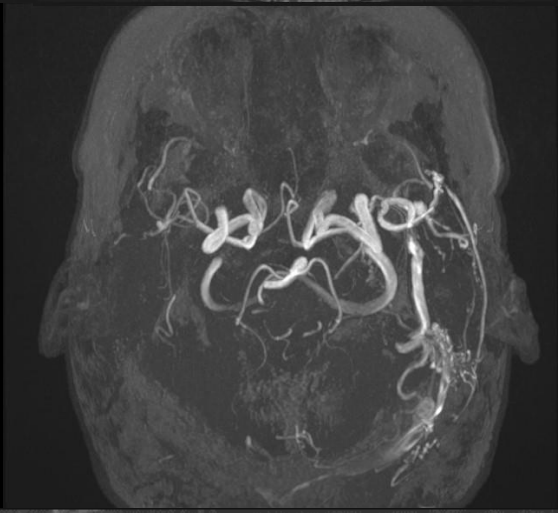

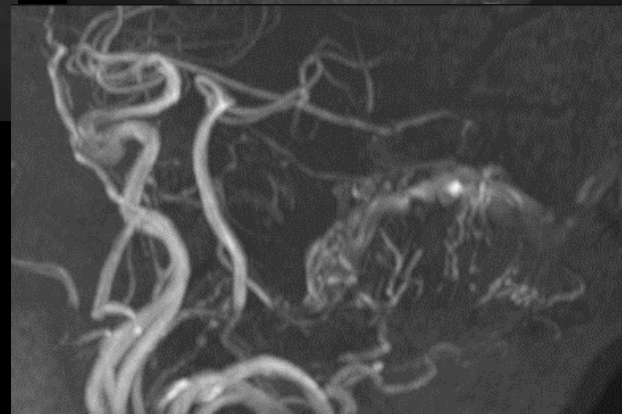

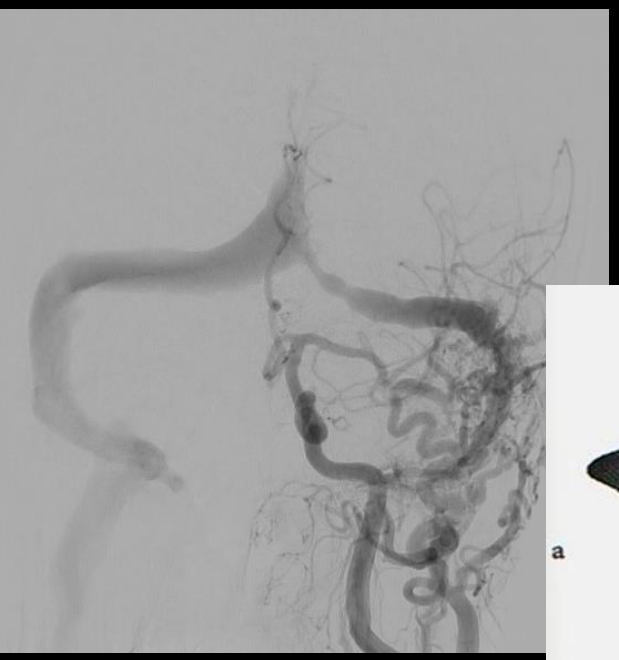

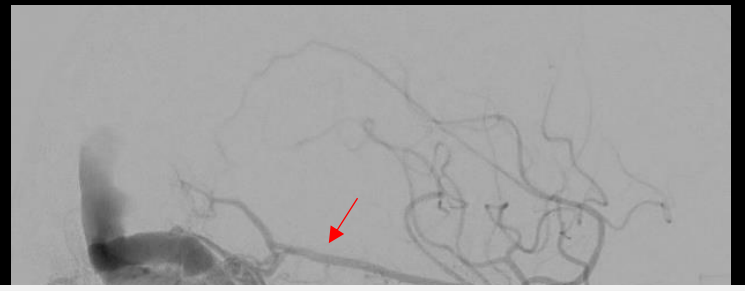

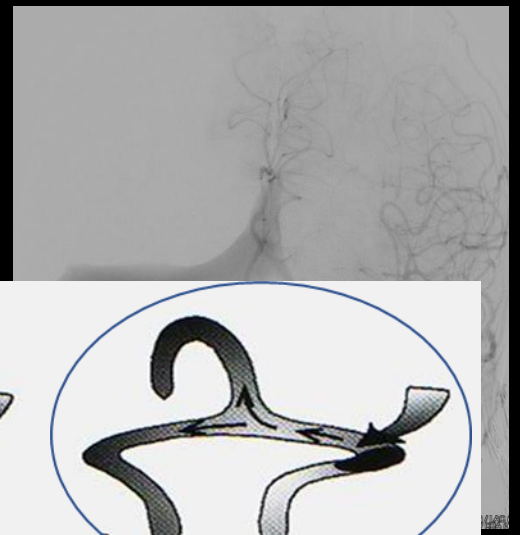

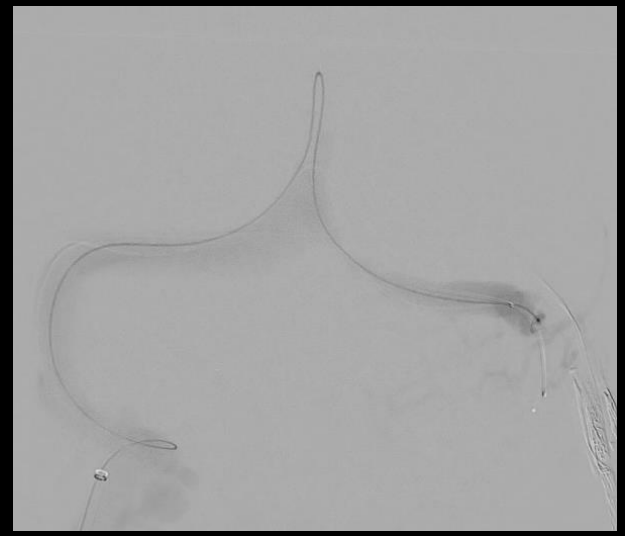

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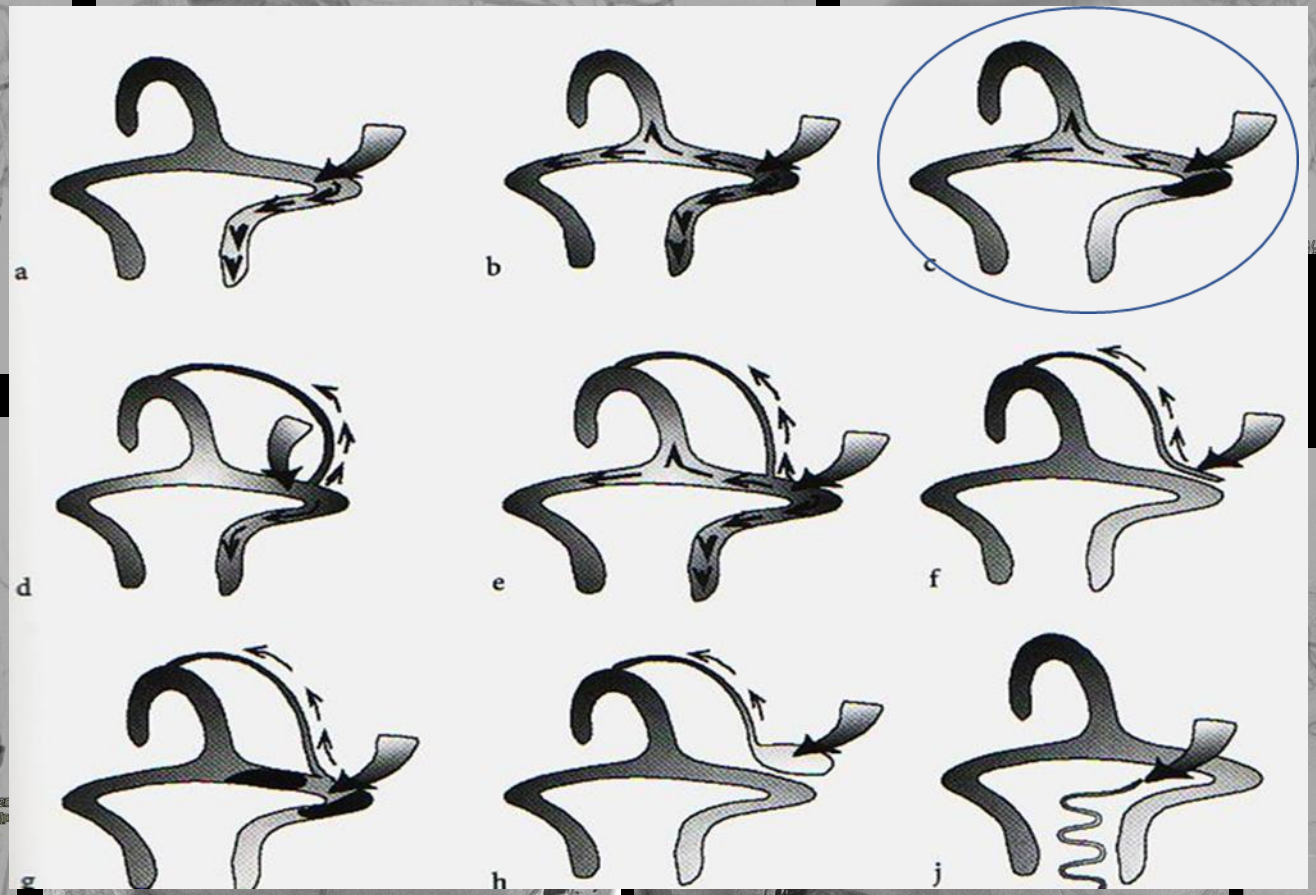

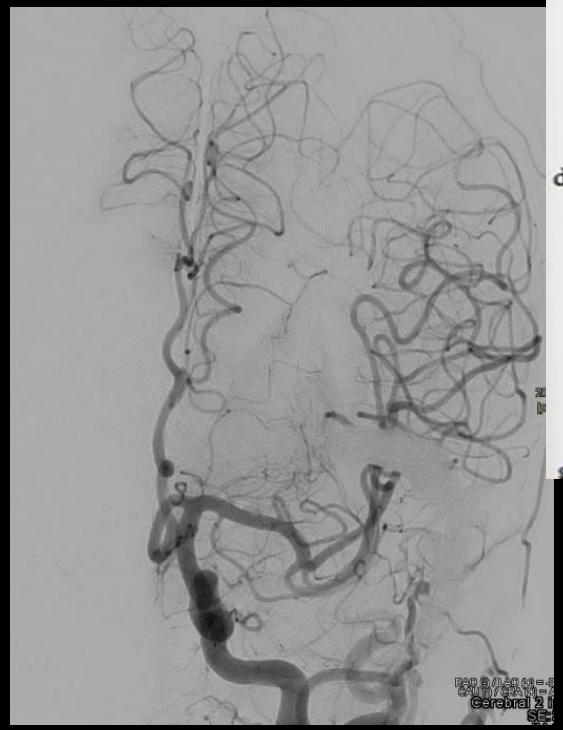

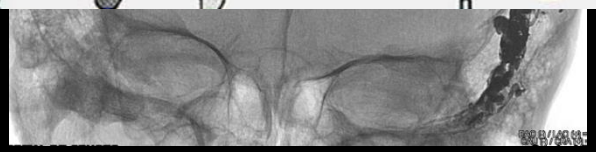

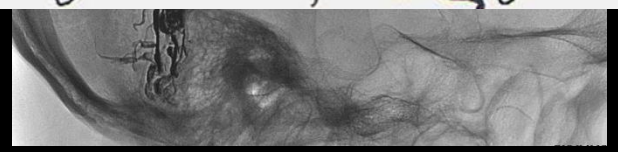

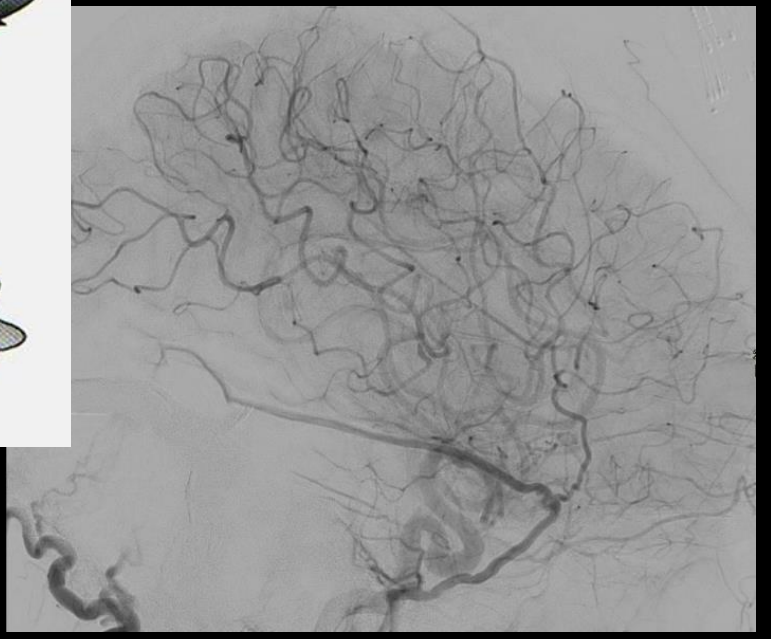

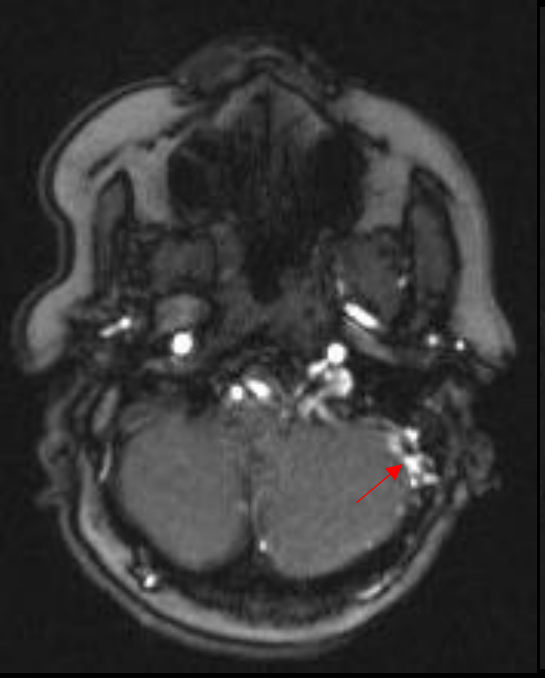

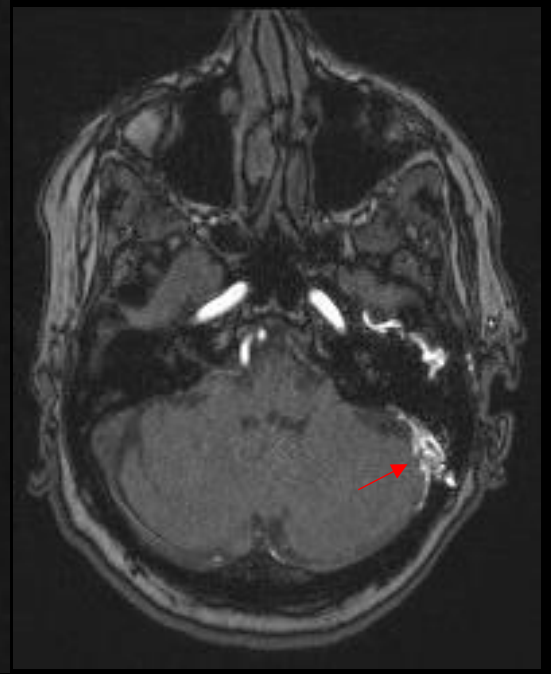

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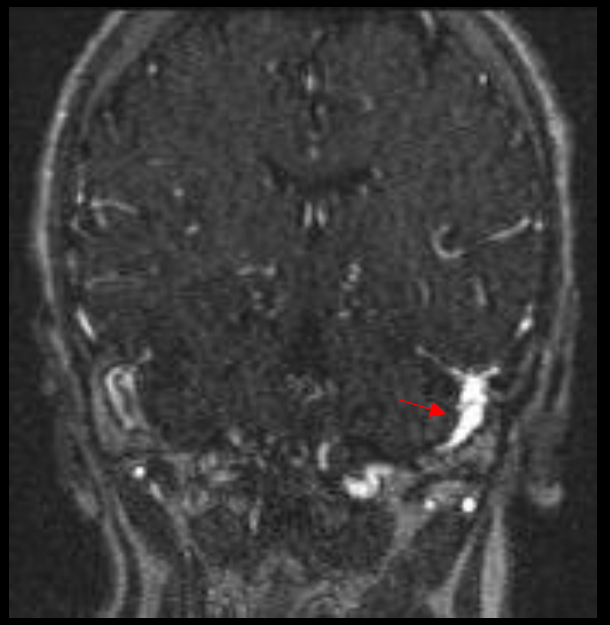

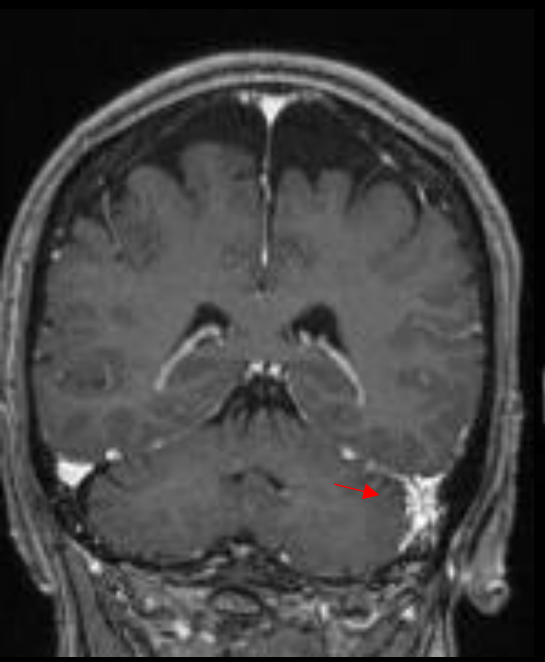

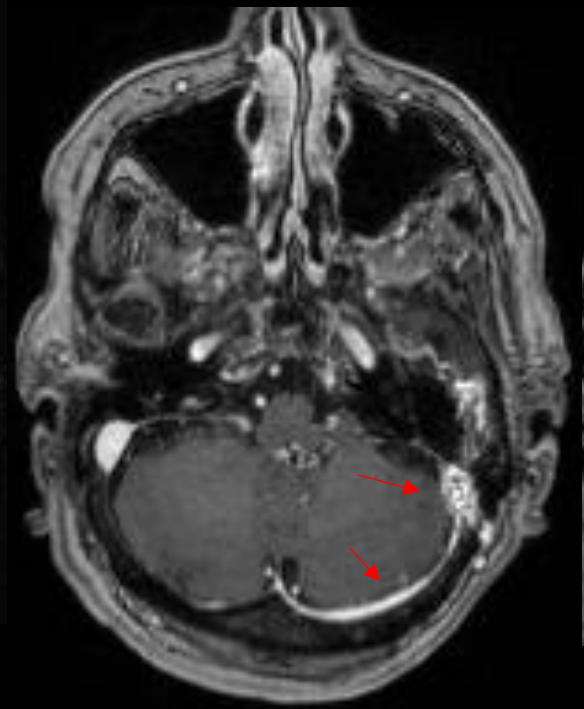

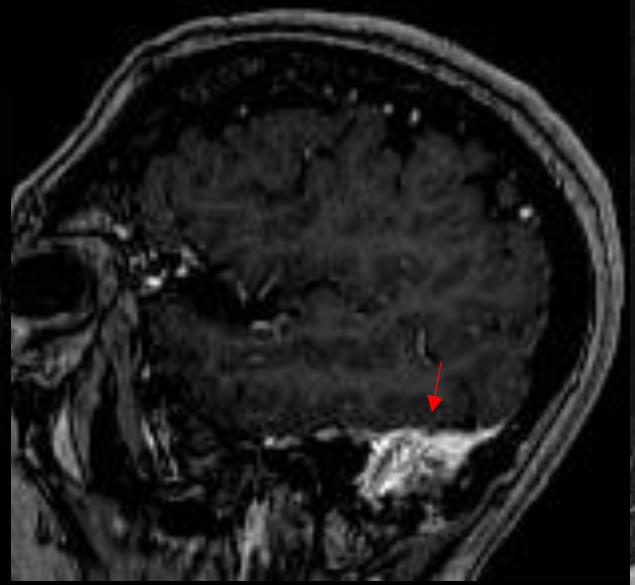

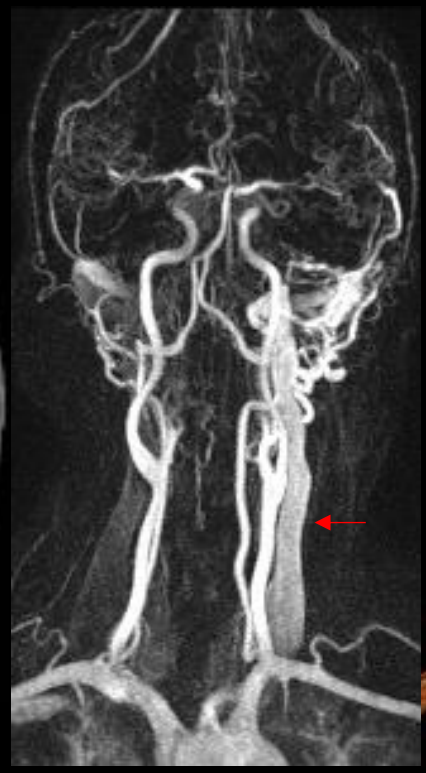

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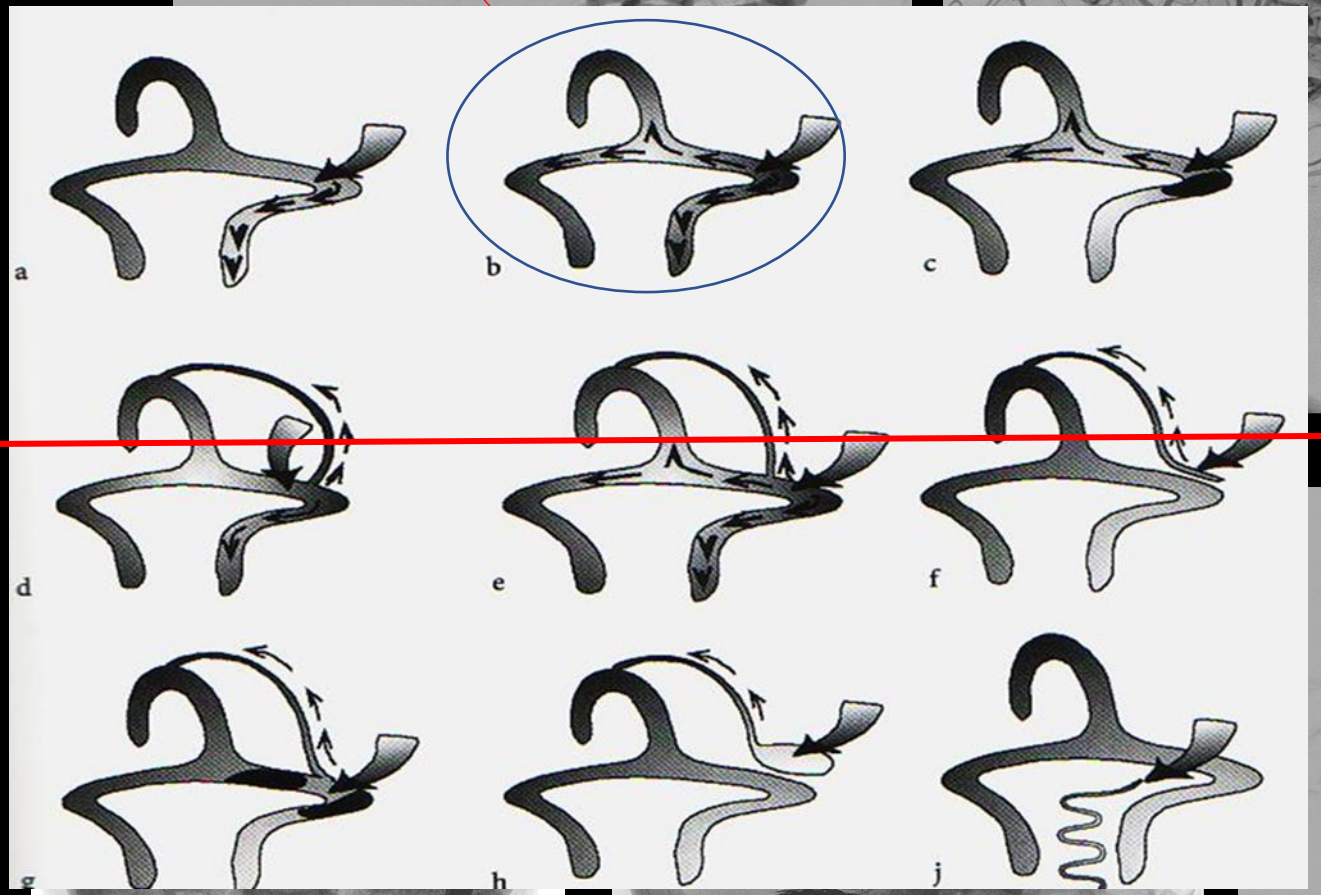

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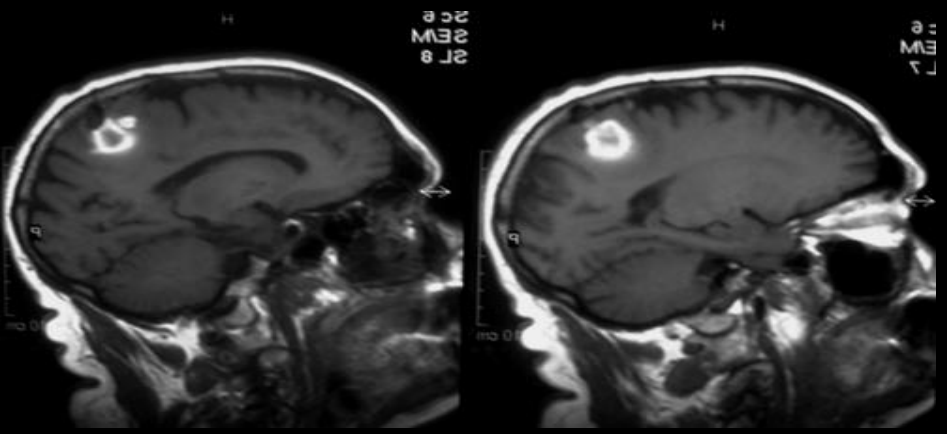
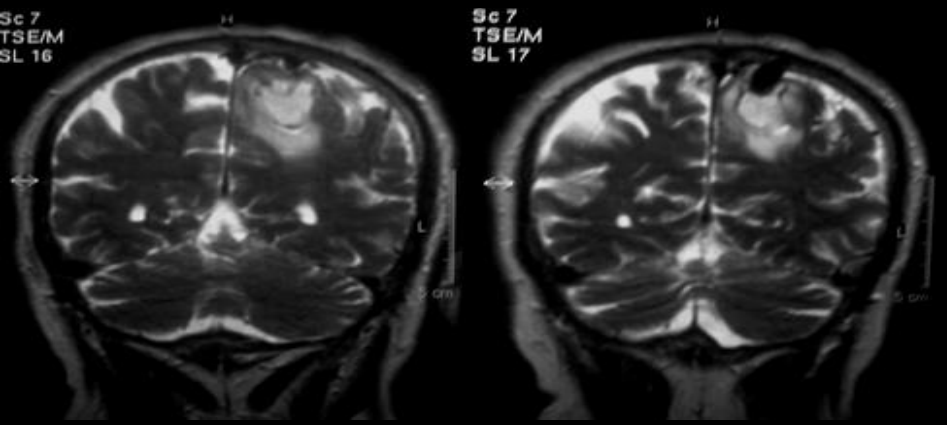
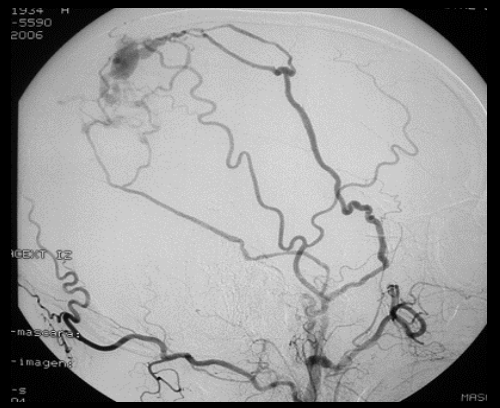
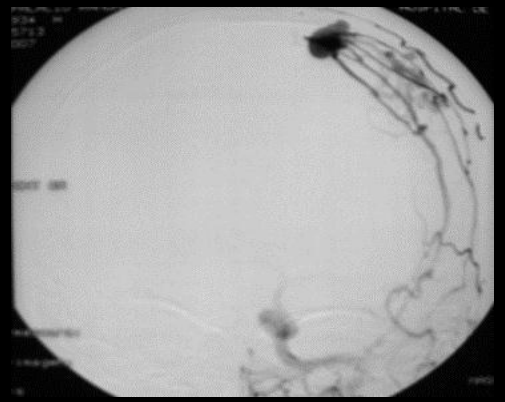
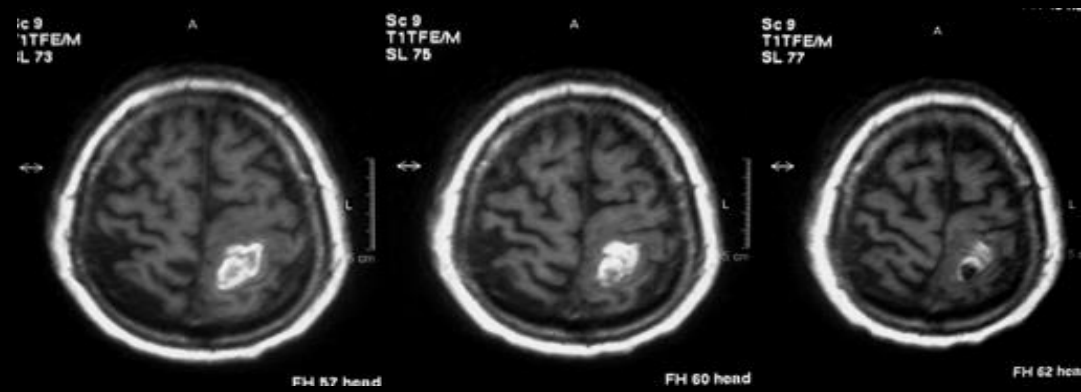
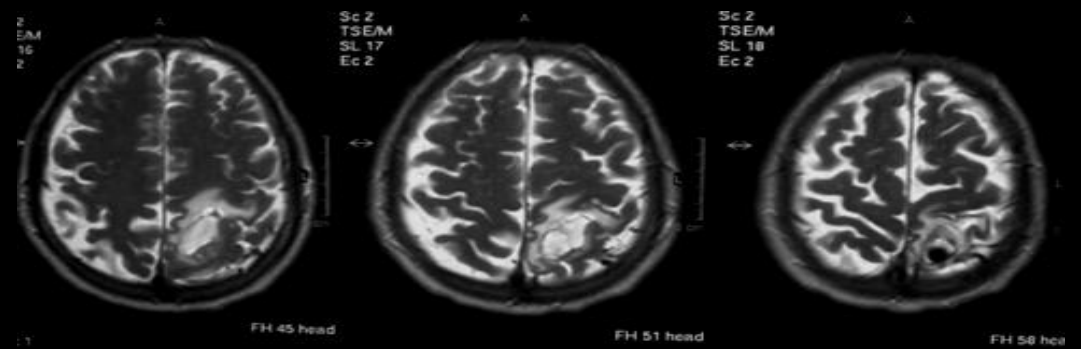


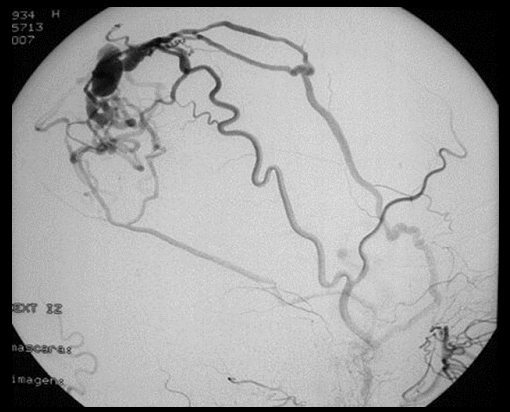


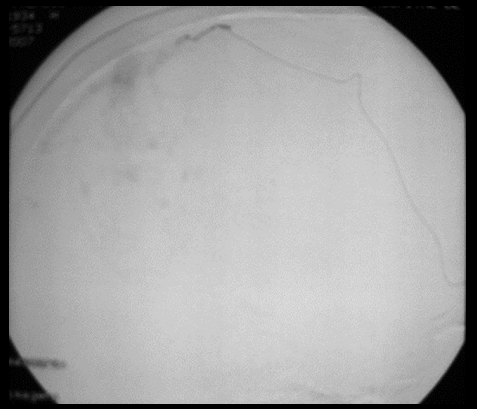


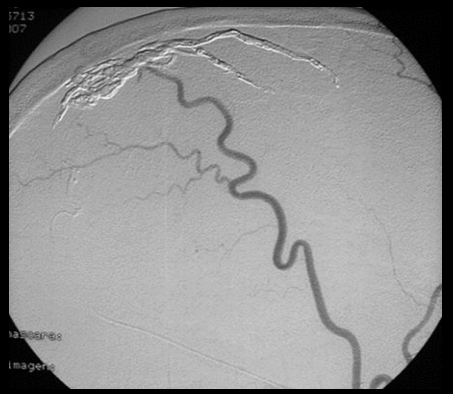


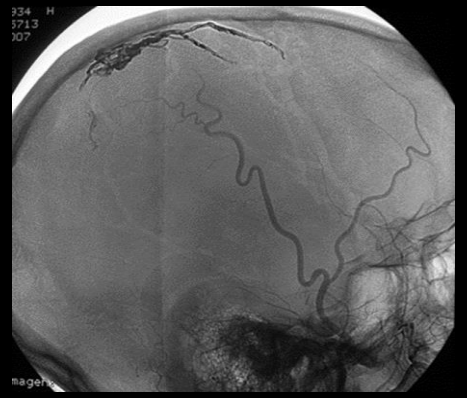


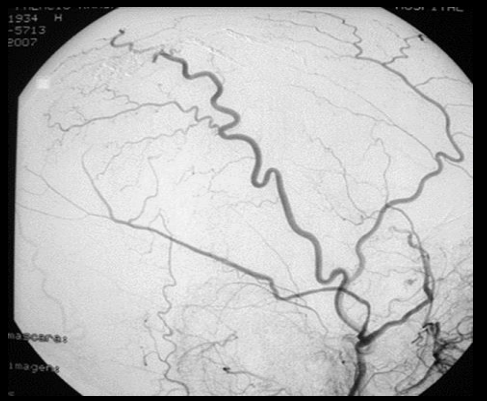


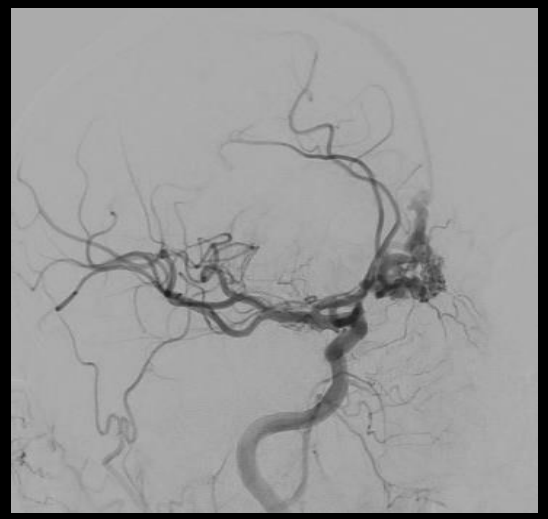
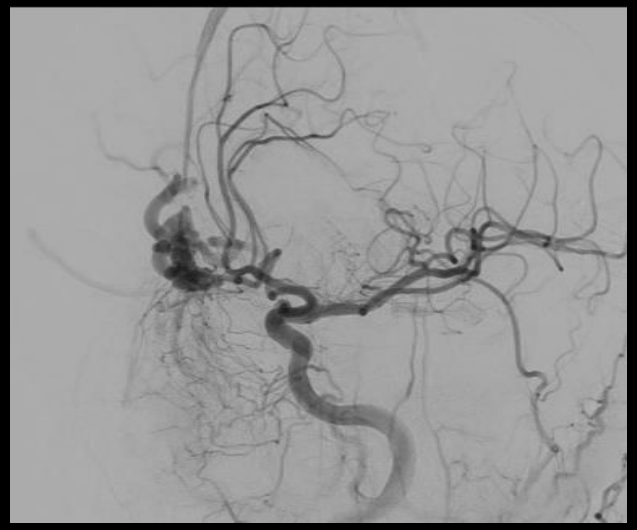
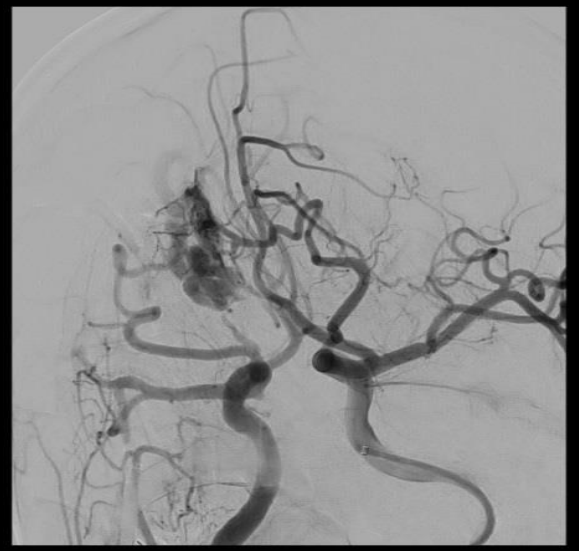
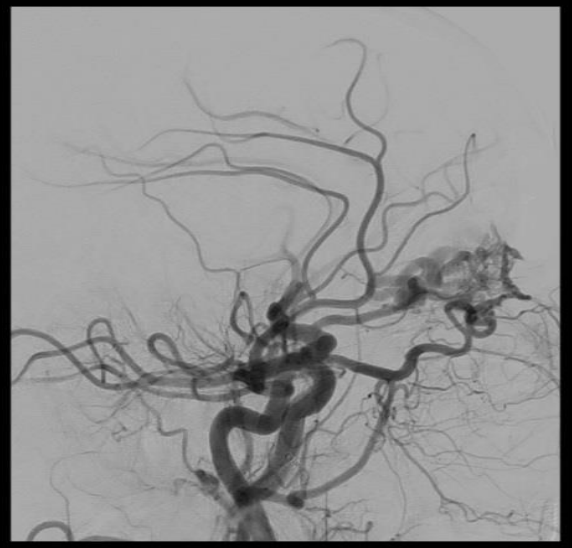
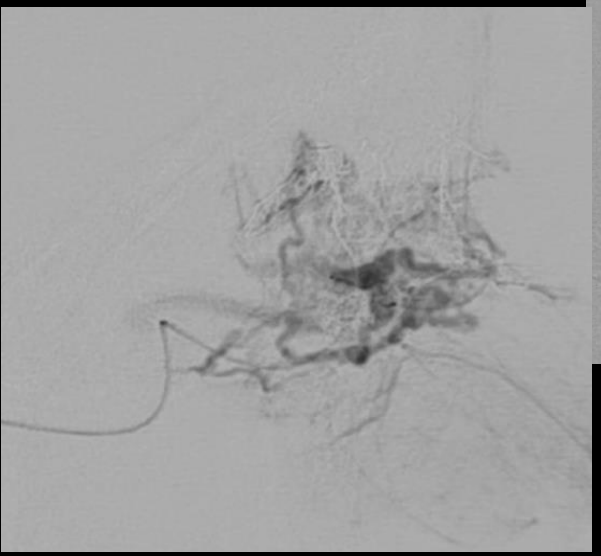
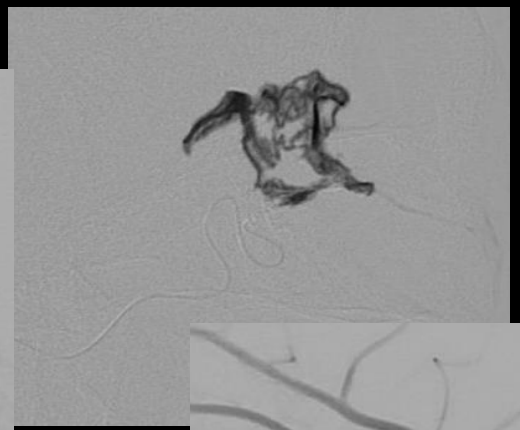
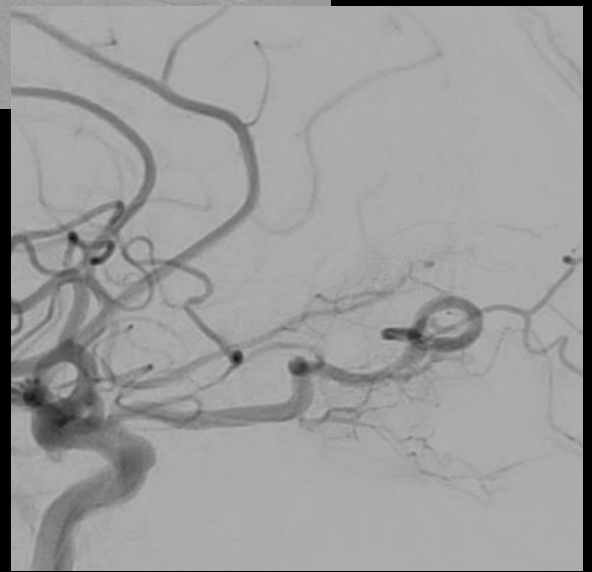




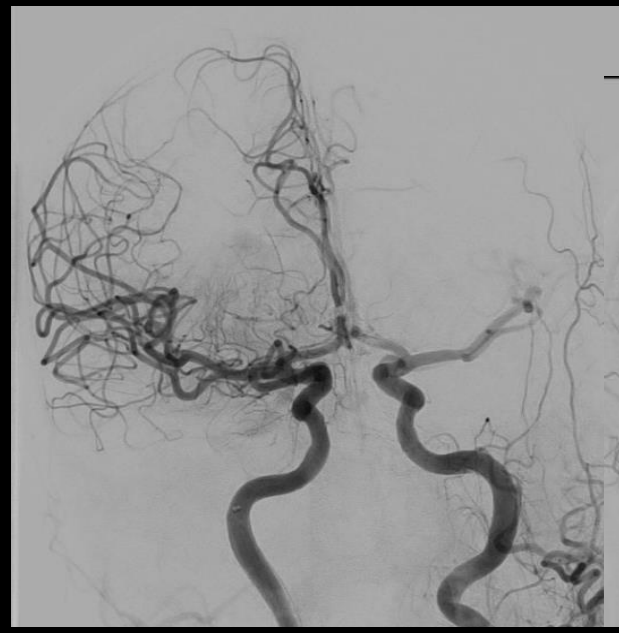
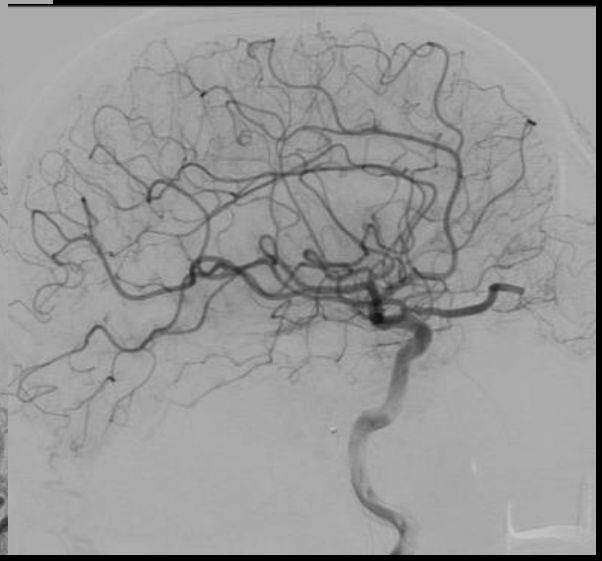


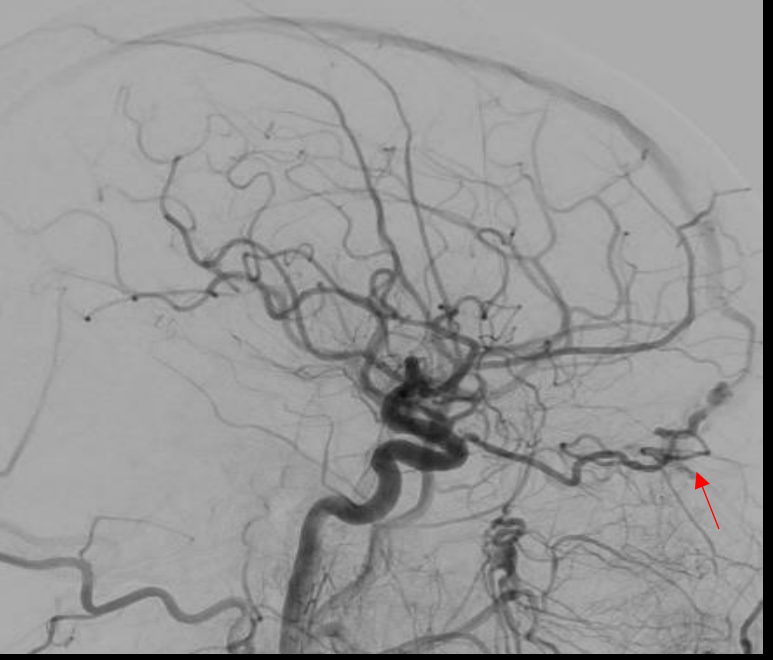


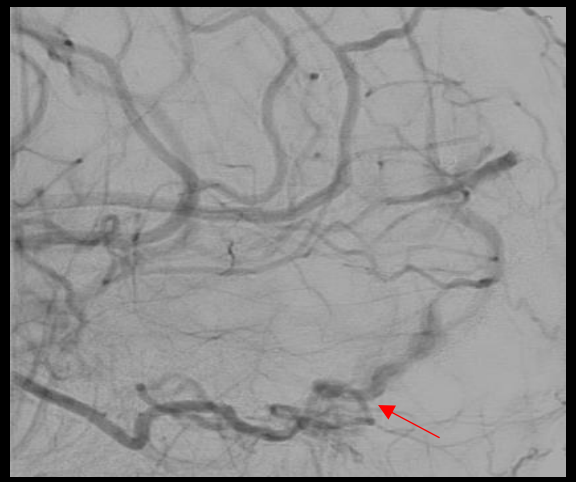


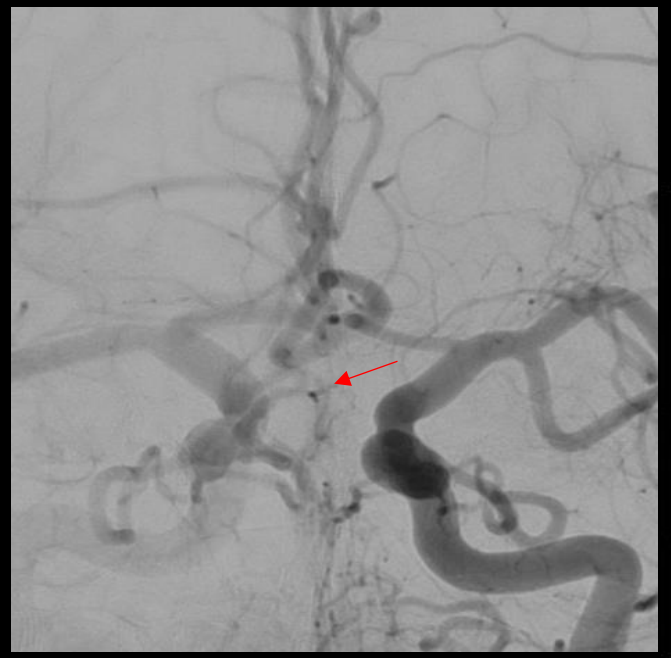








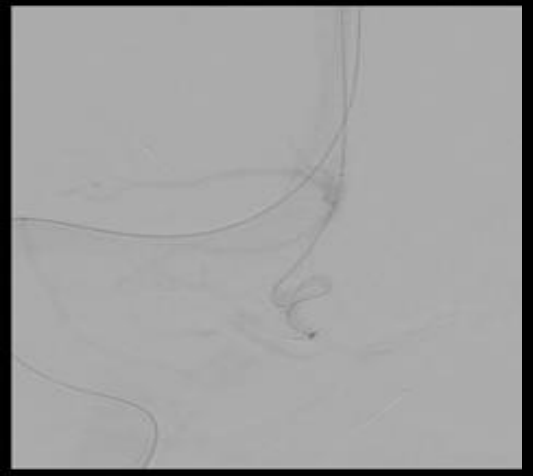
FAVD grado IV fosa craneal anterior
Embolización vía arterial

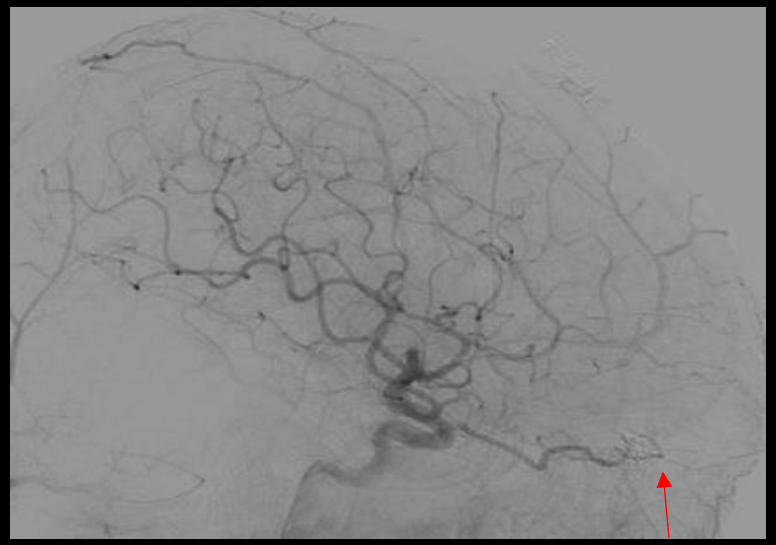


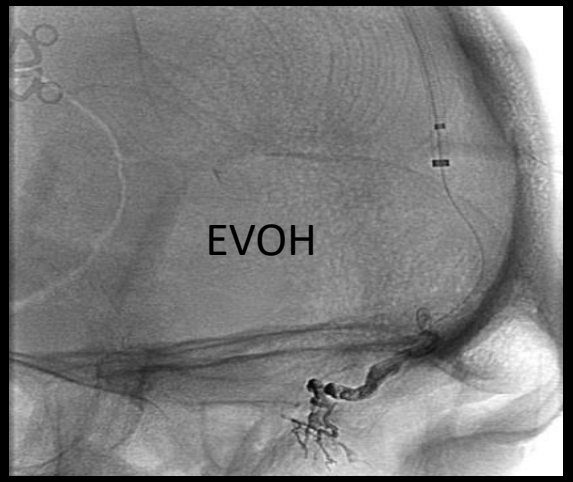


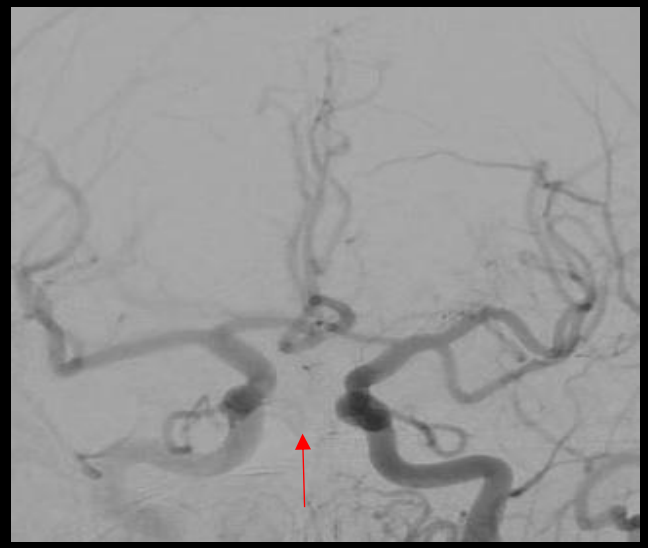


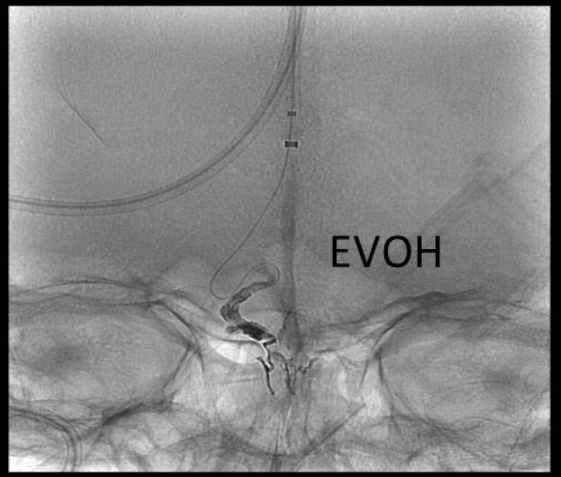


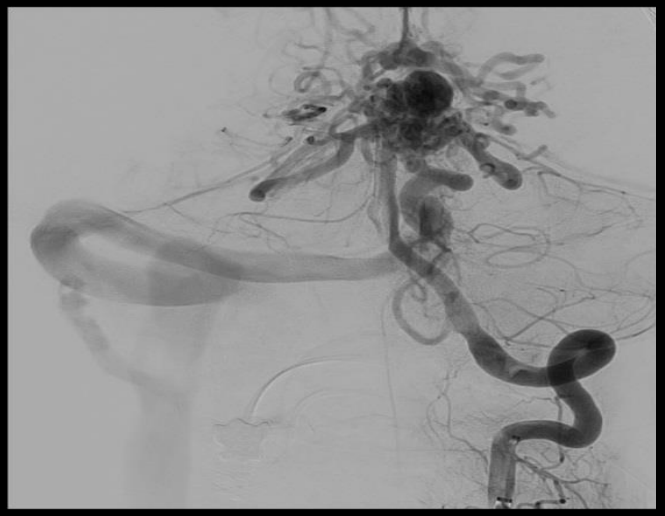
FAVD grado IV fosa craneal anterior
Embolización vía venosa

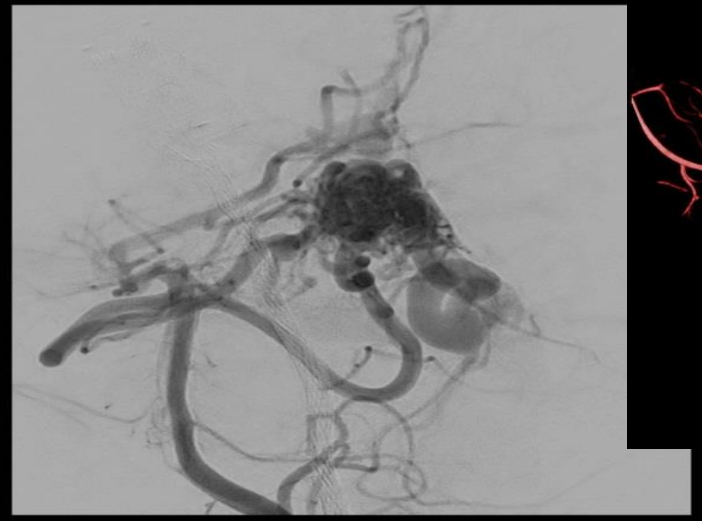


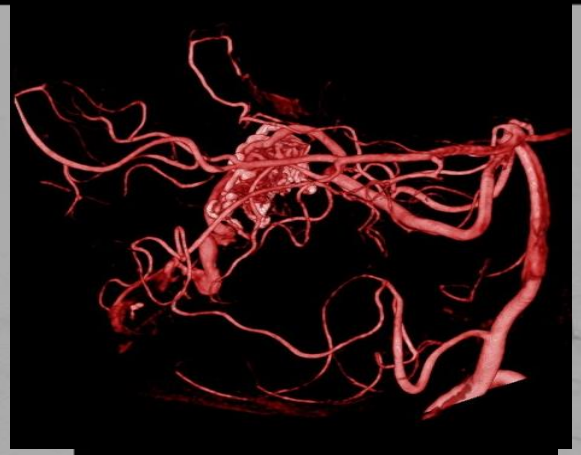


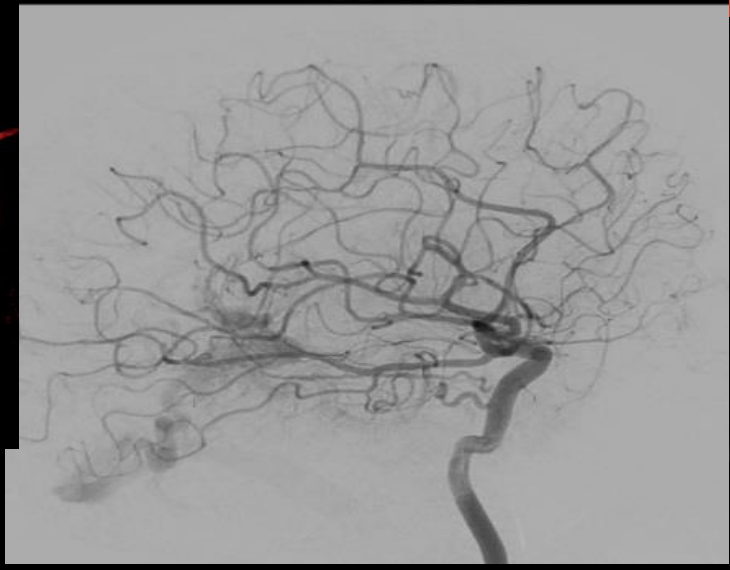


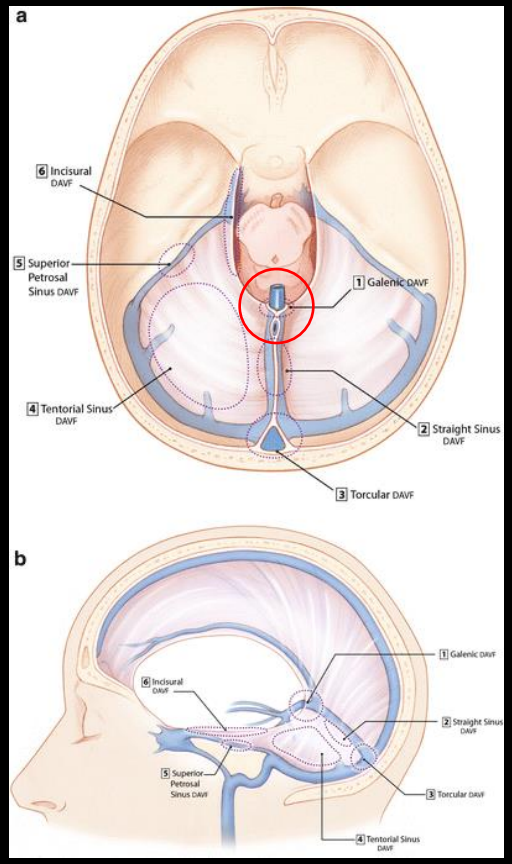




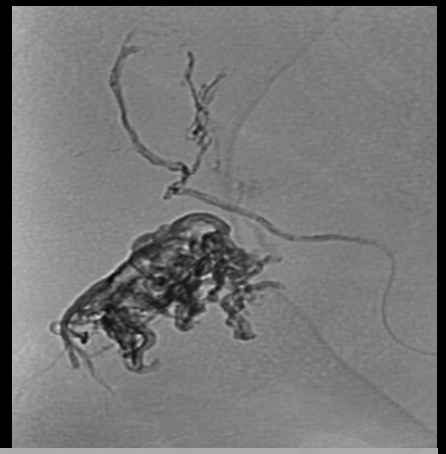


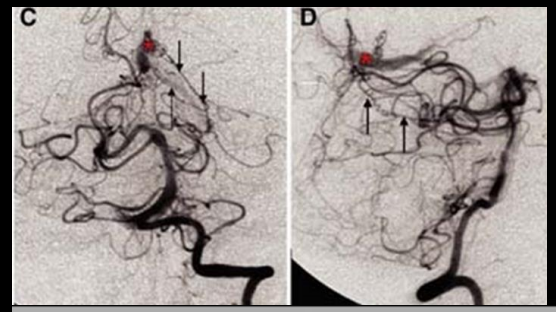


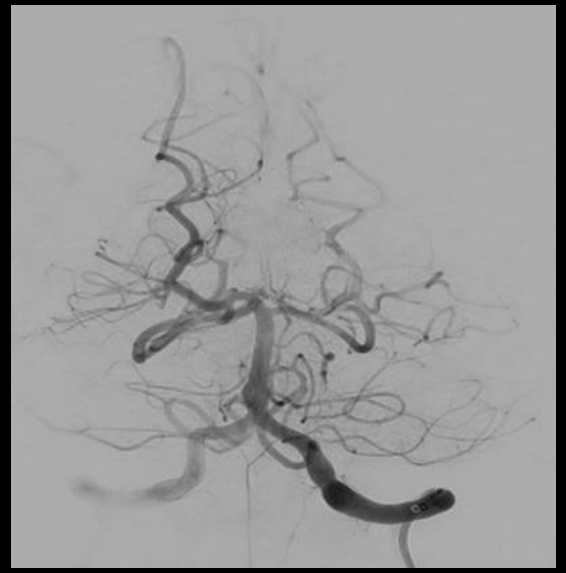


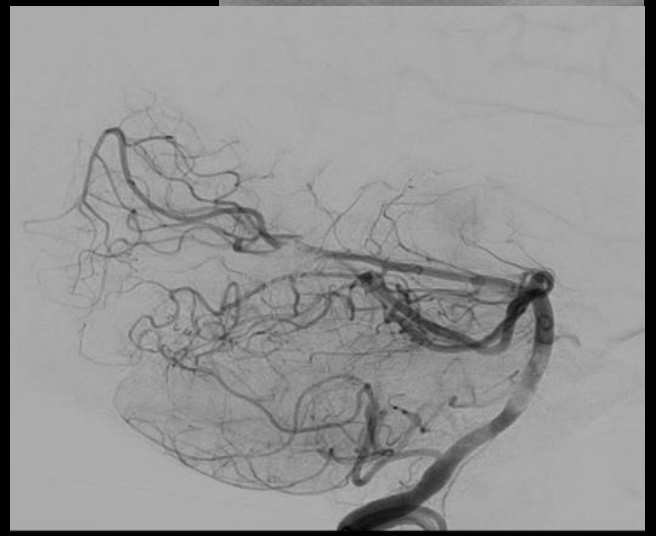


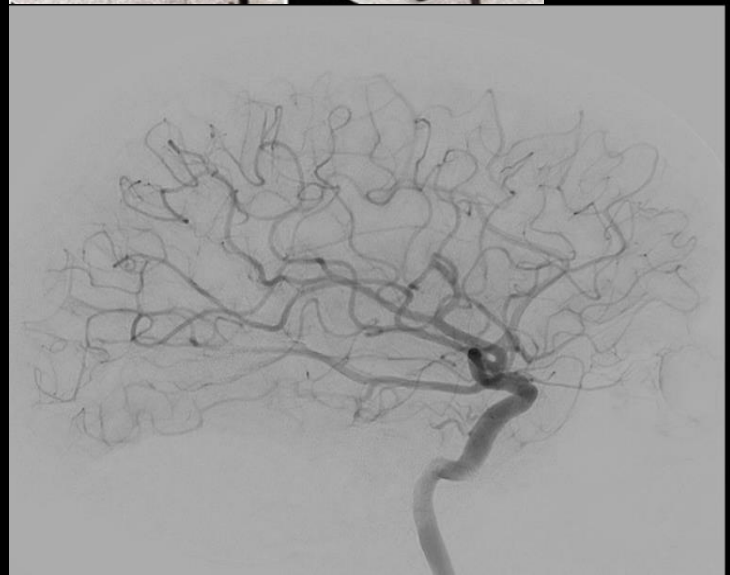
FAVD tentorial tipo I
Embolización vía arterial

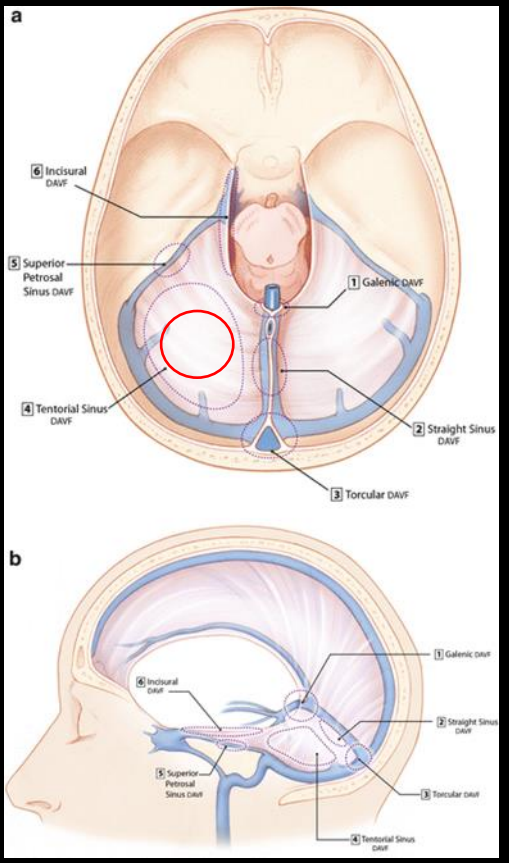




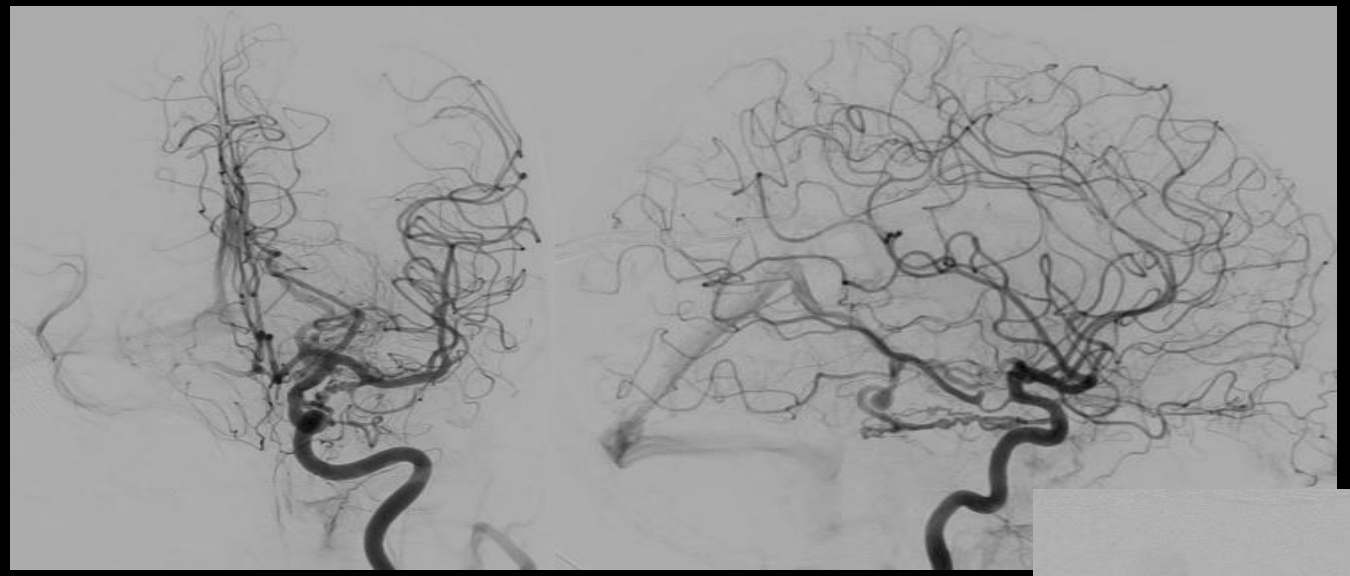


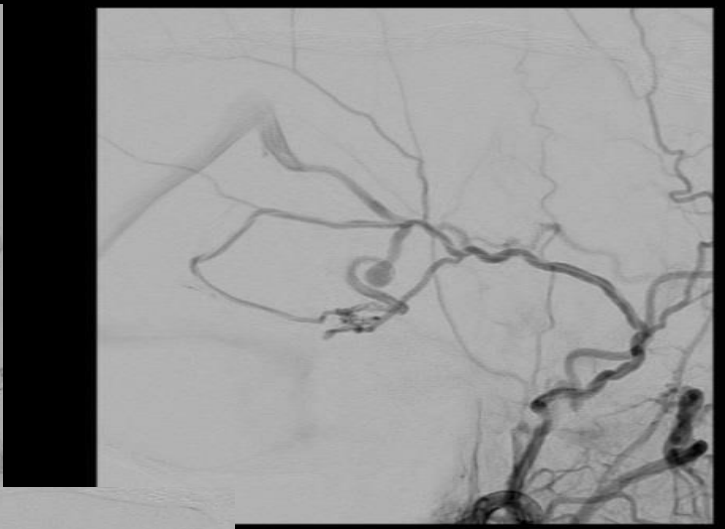


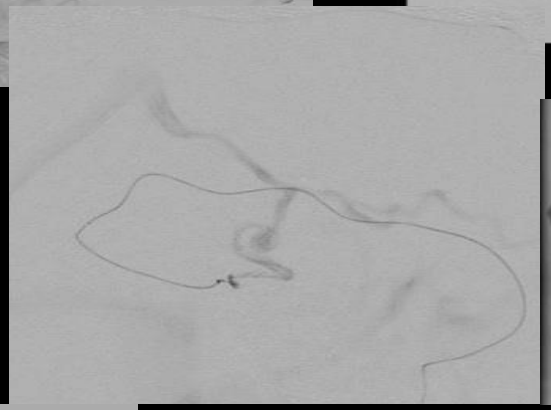


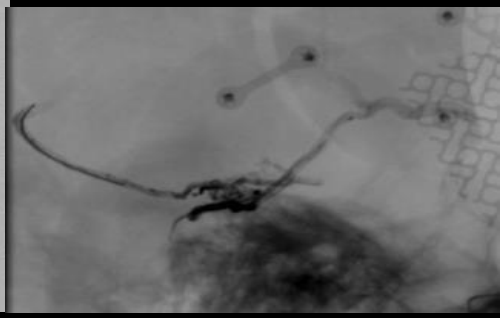


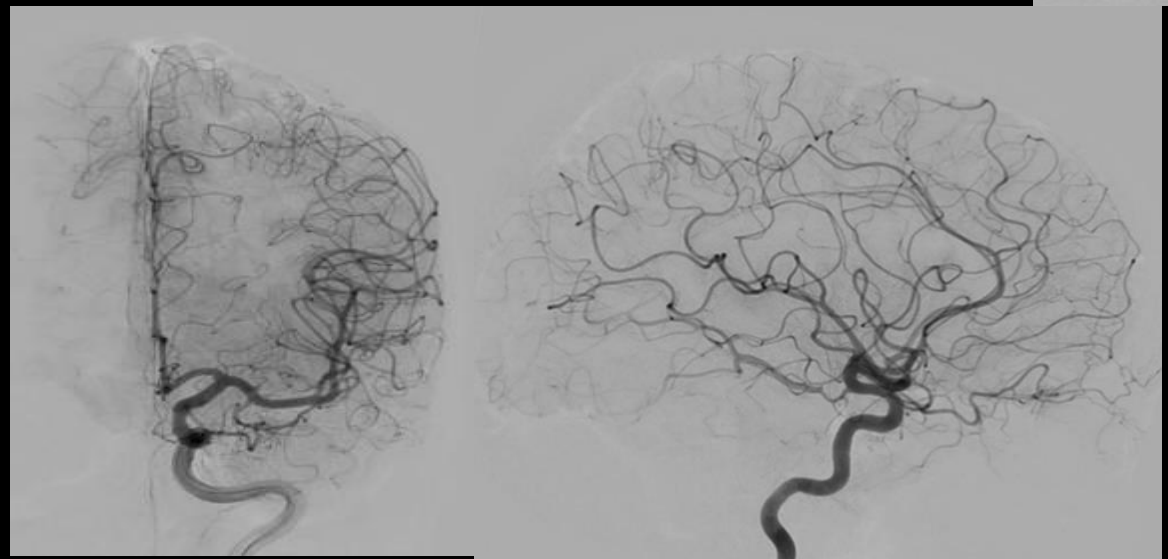
FAVD tentorial Tipo 4

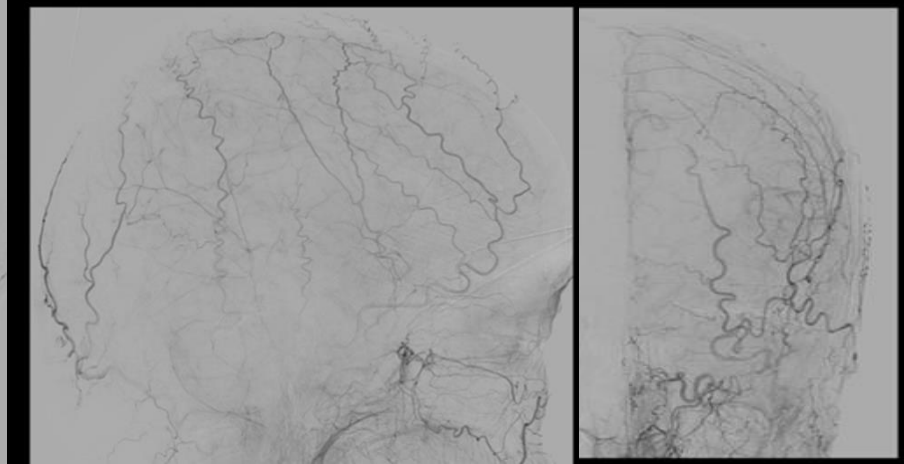




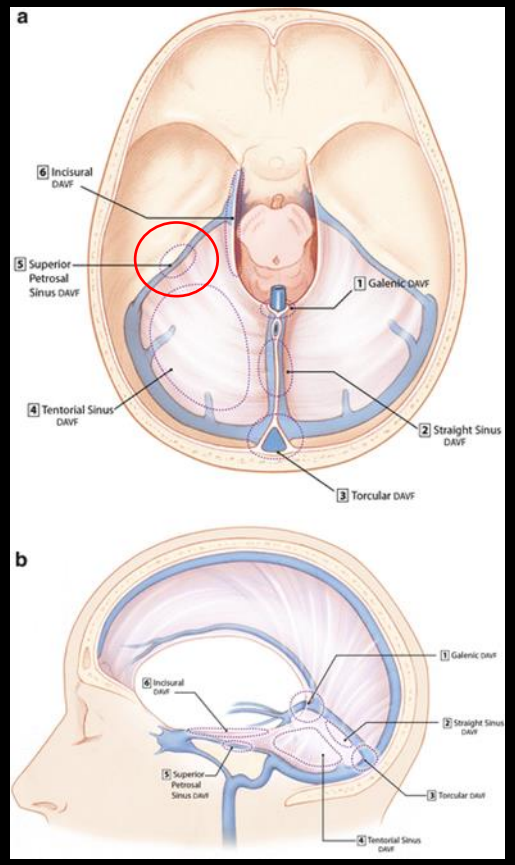


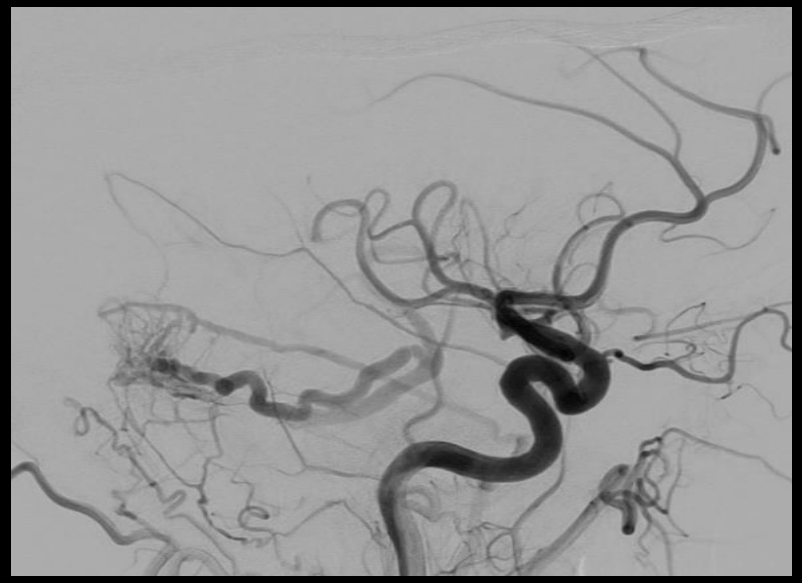


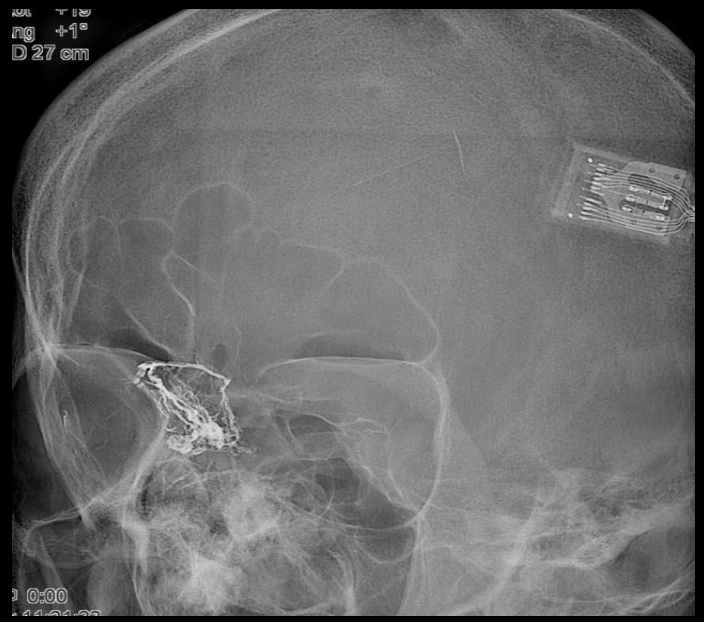


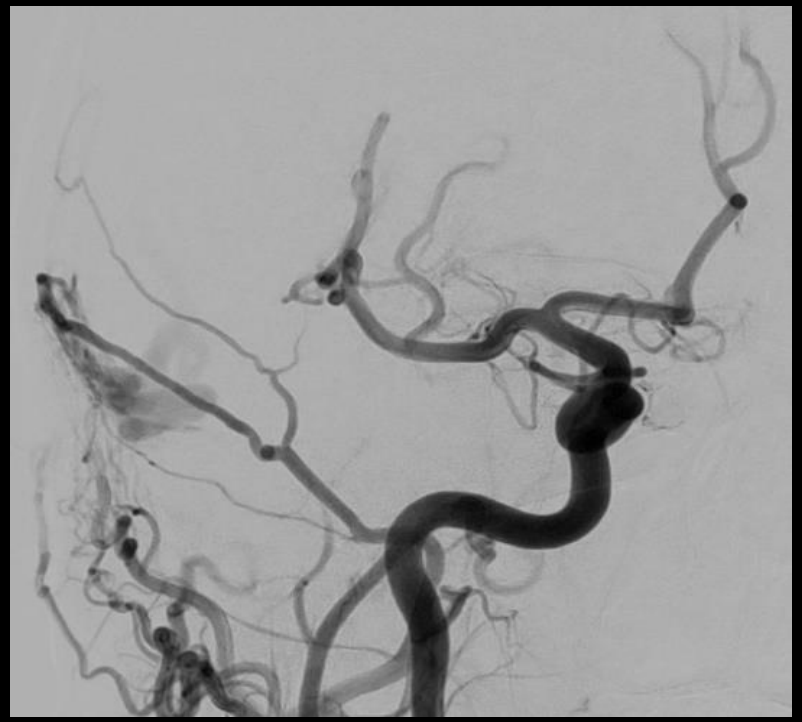


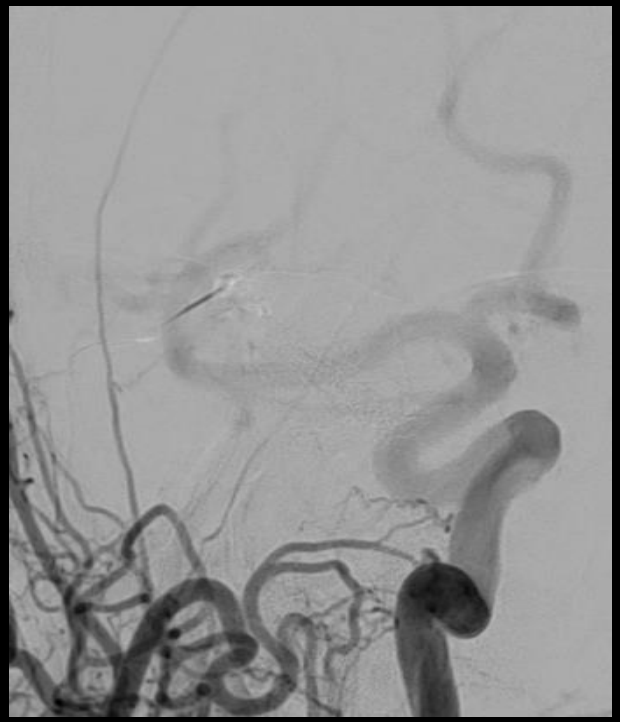
FAVD tentorial Tipo 5



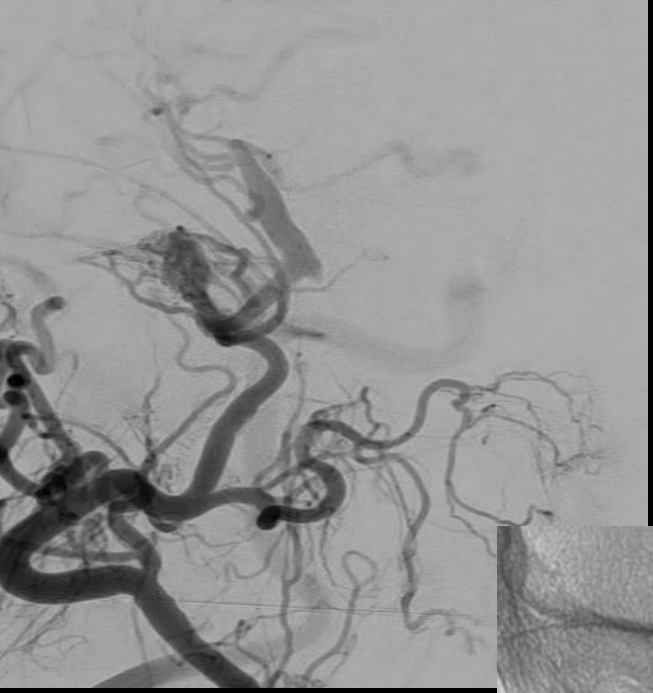


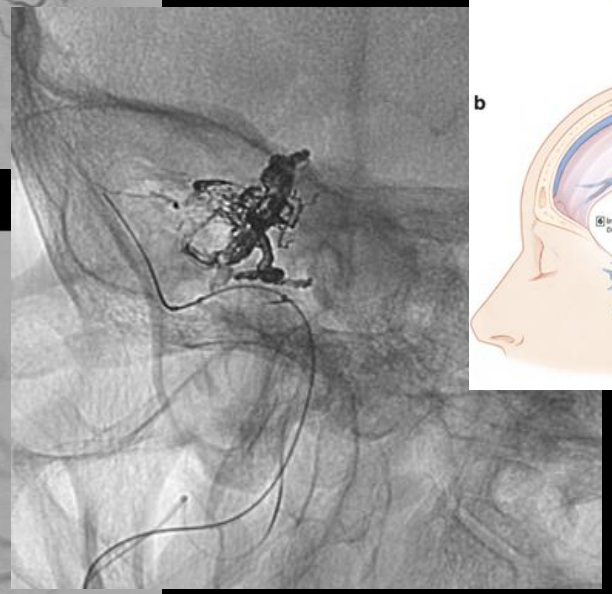


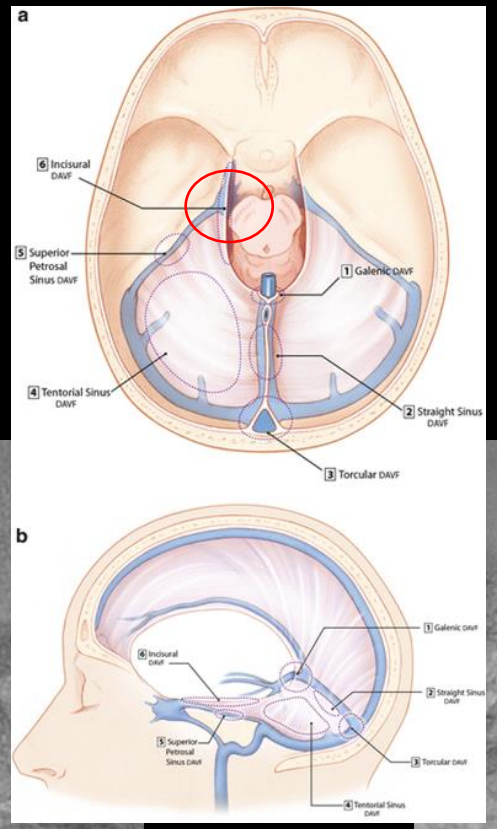


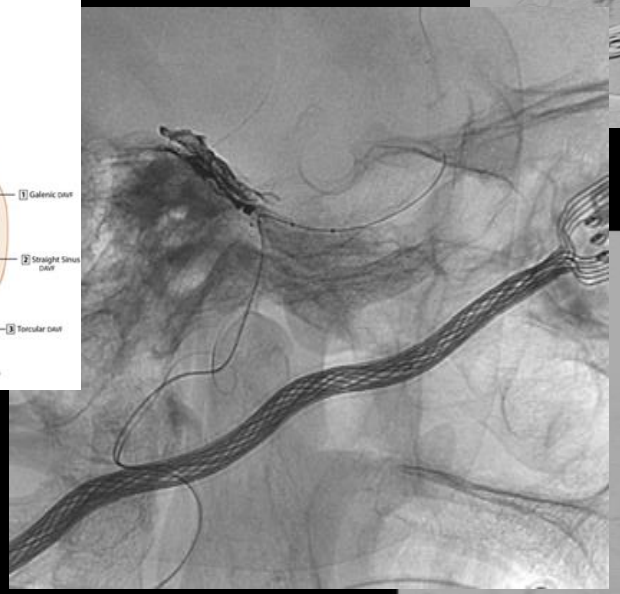


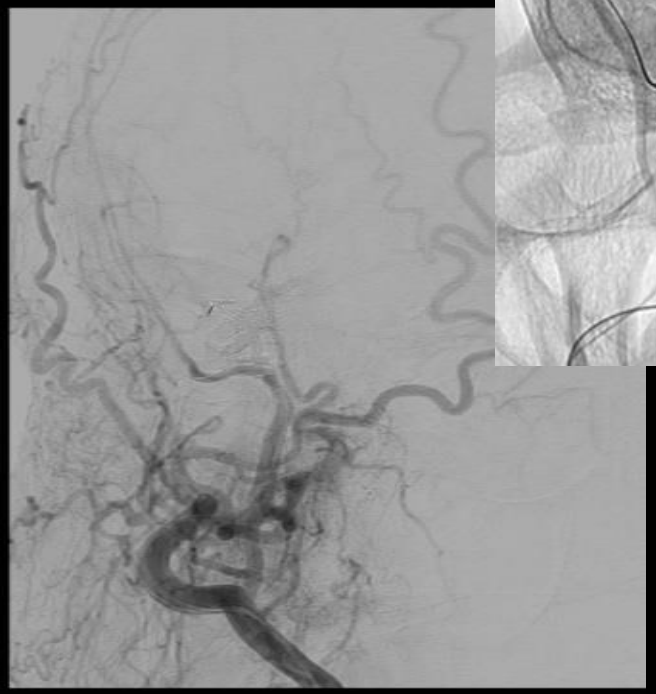
FAVD tentorial Tipo 6

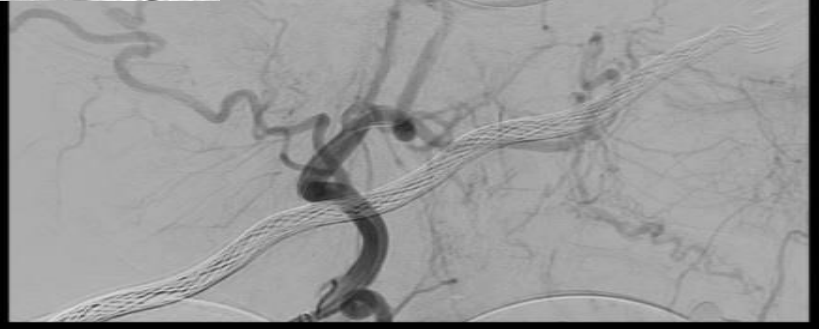


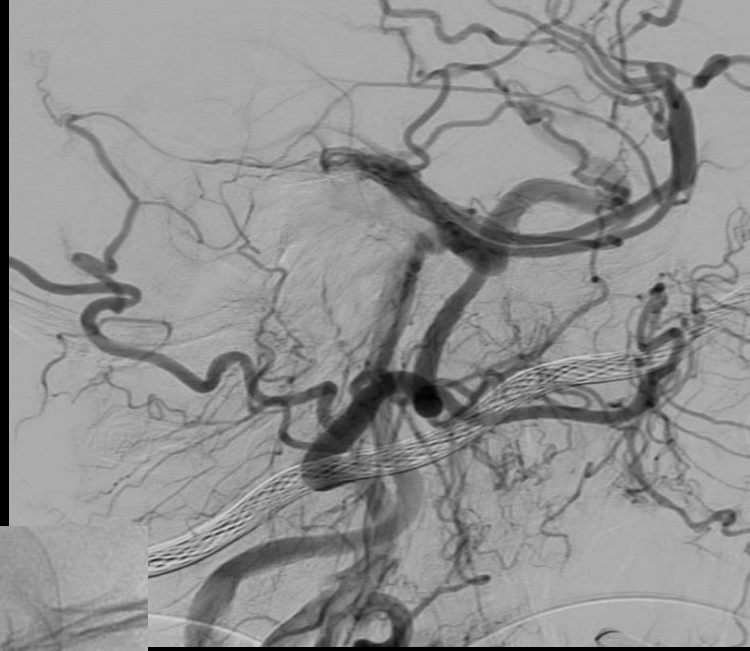


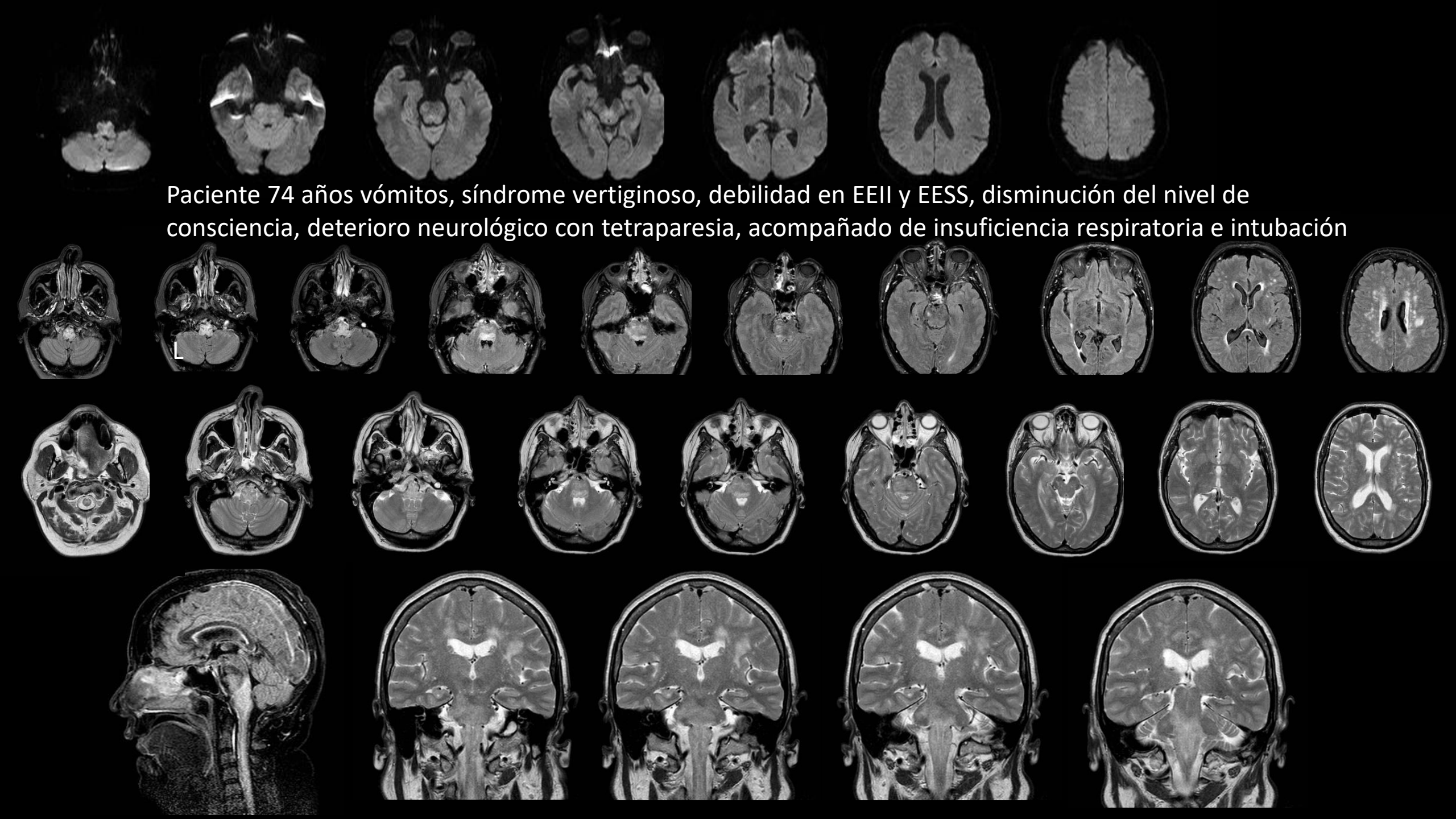


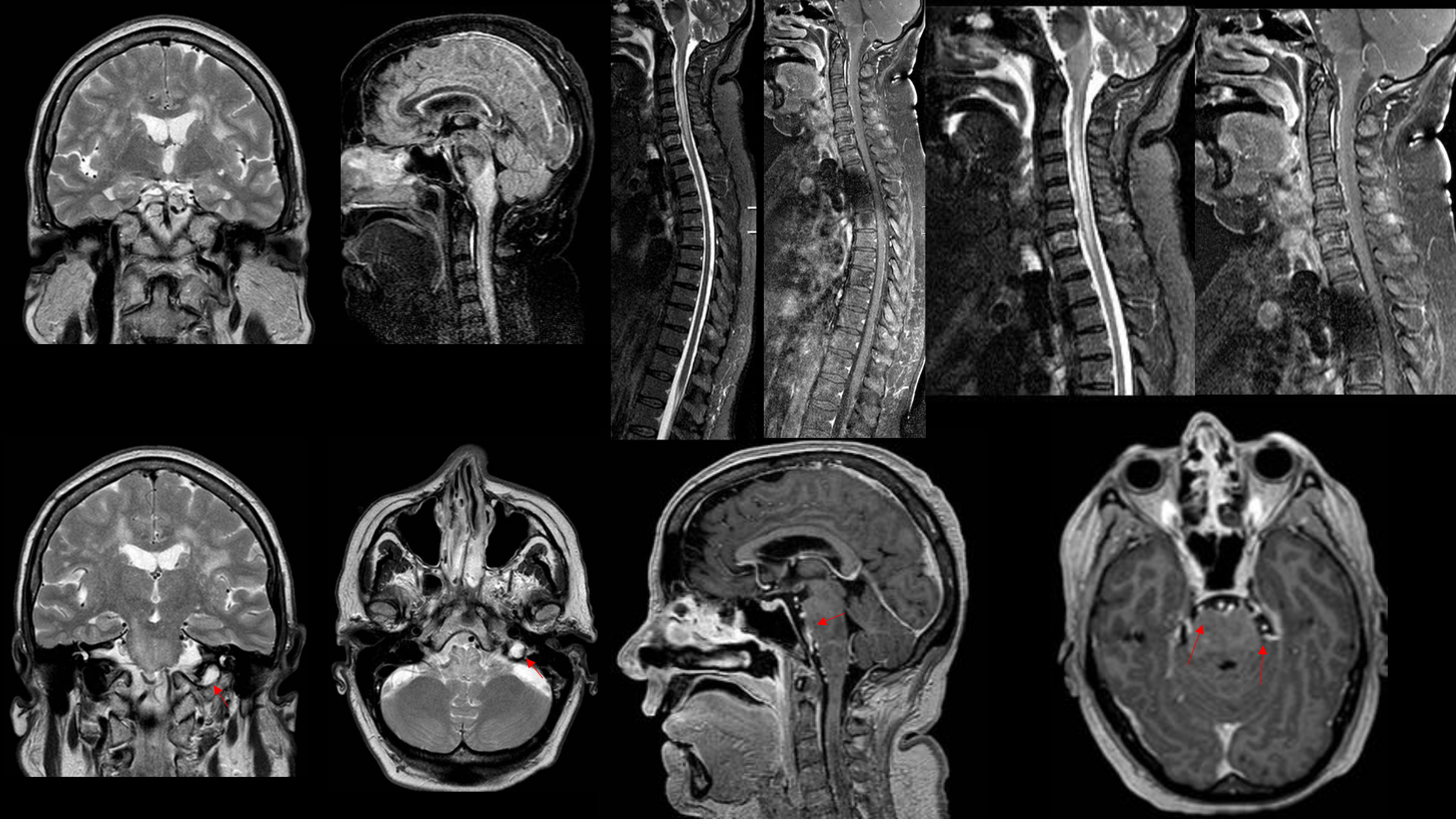


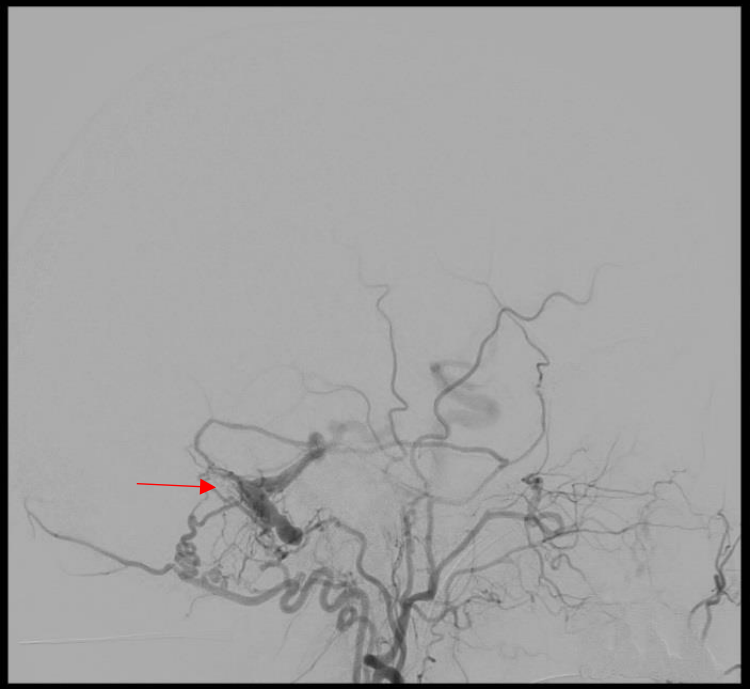


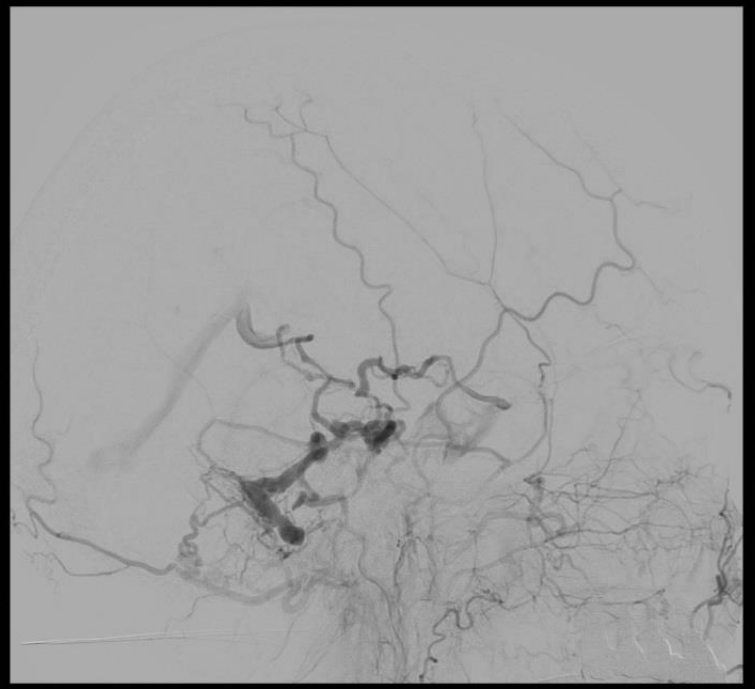


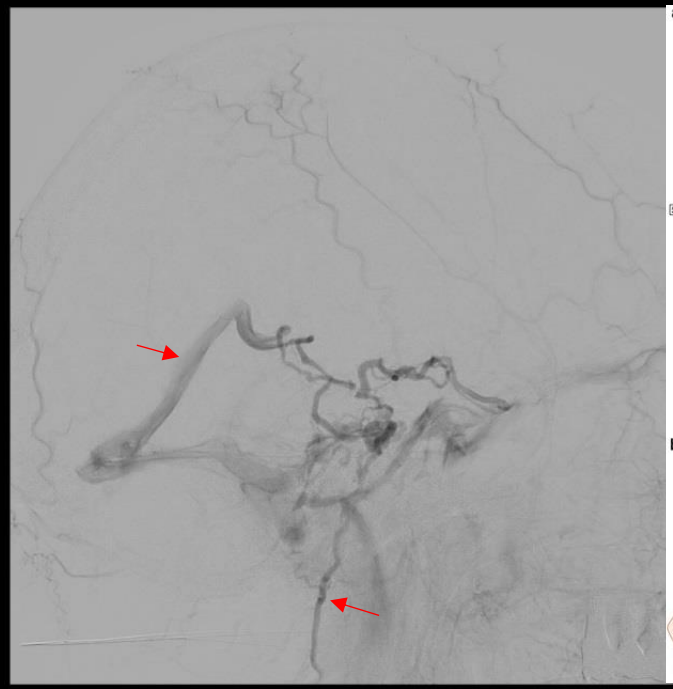


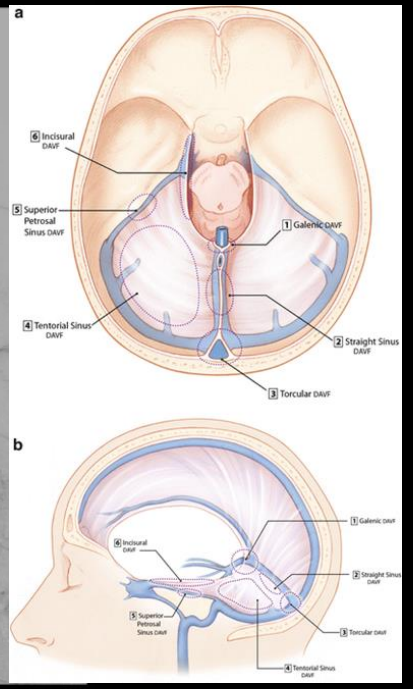

Paciente 74 años vómitos, síndrome vertiginoso, debilidad en EEII y EESS, disminución del nivel de consciencia, deterioro neurológico con tetraparesia, acompañado de insuficiencia respiratoria e intubación
L



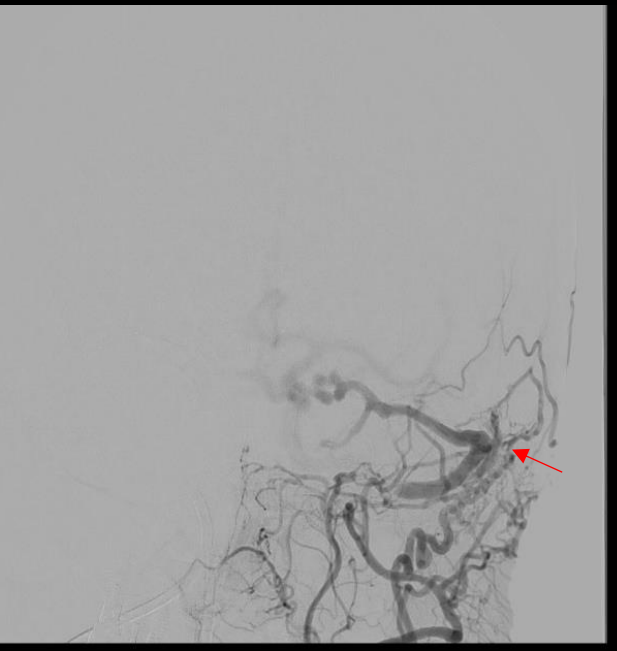


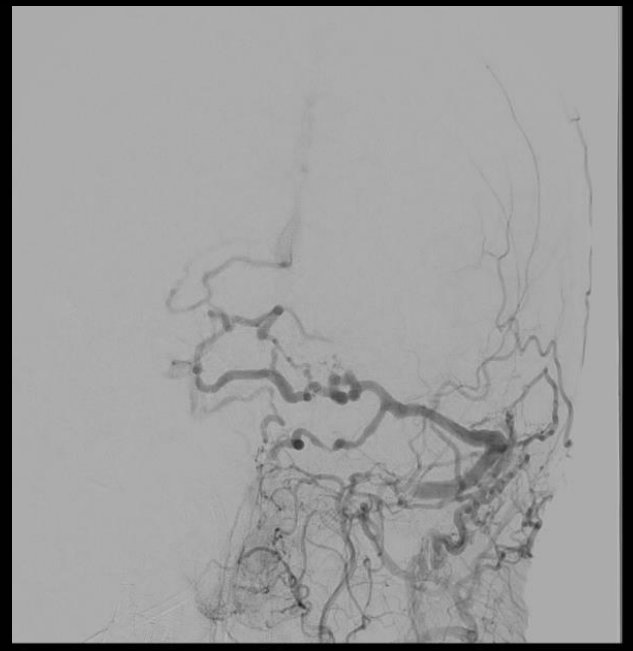


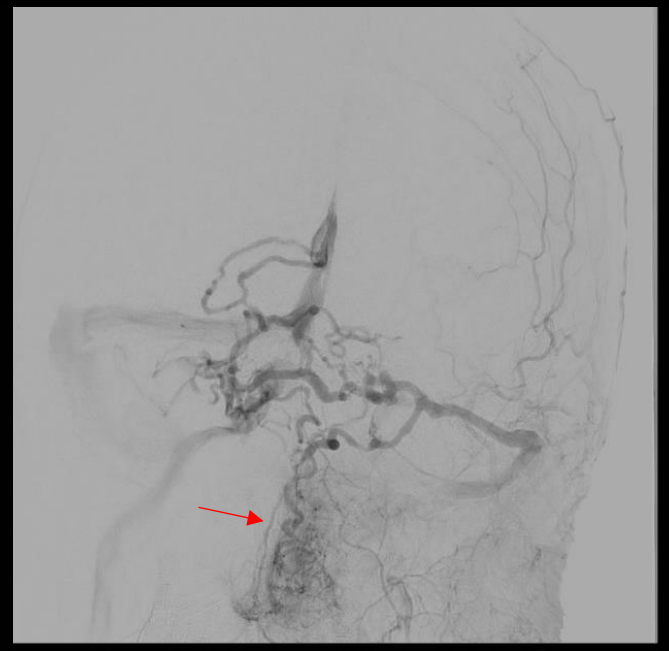


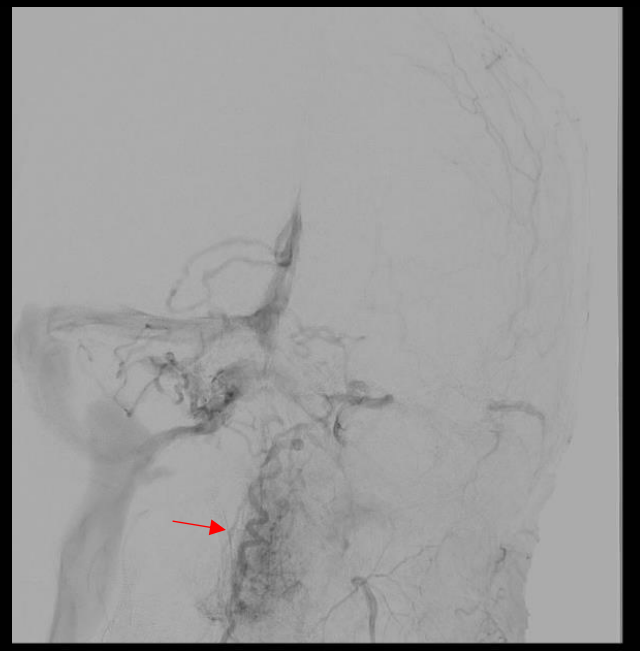


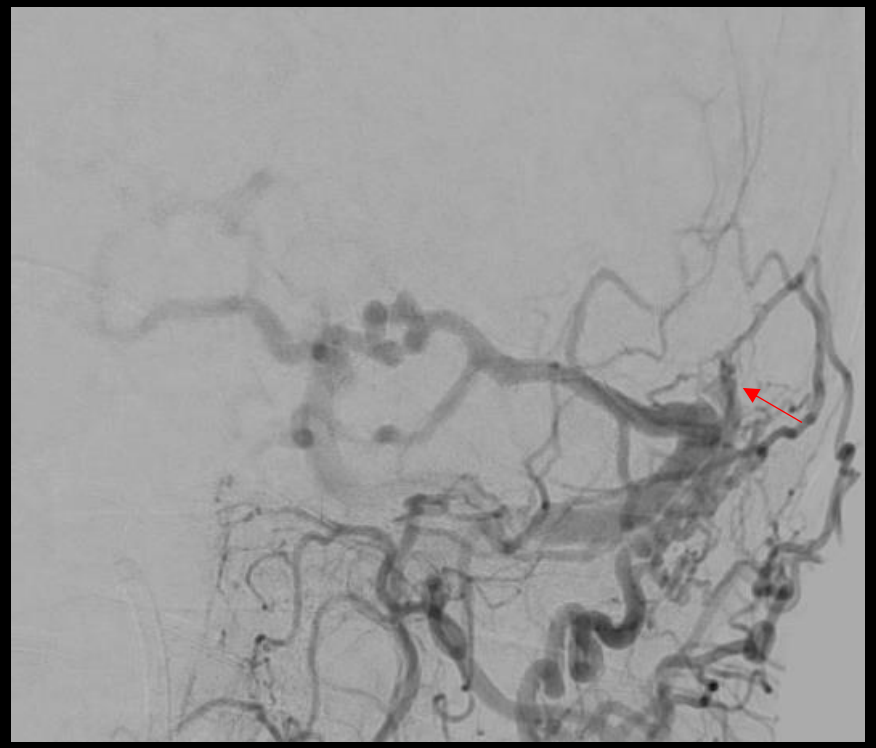
FAVD tentorial tipo 5

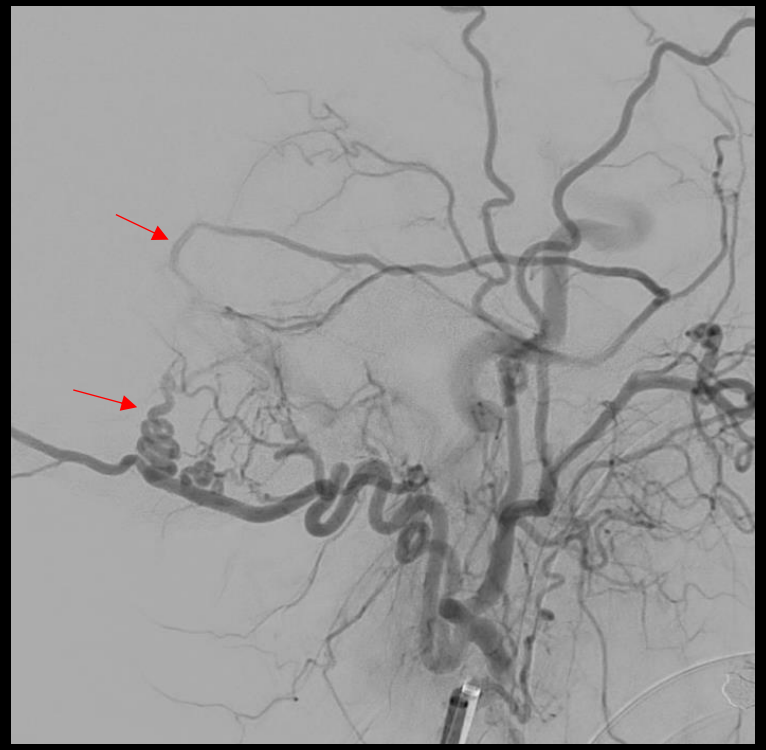


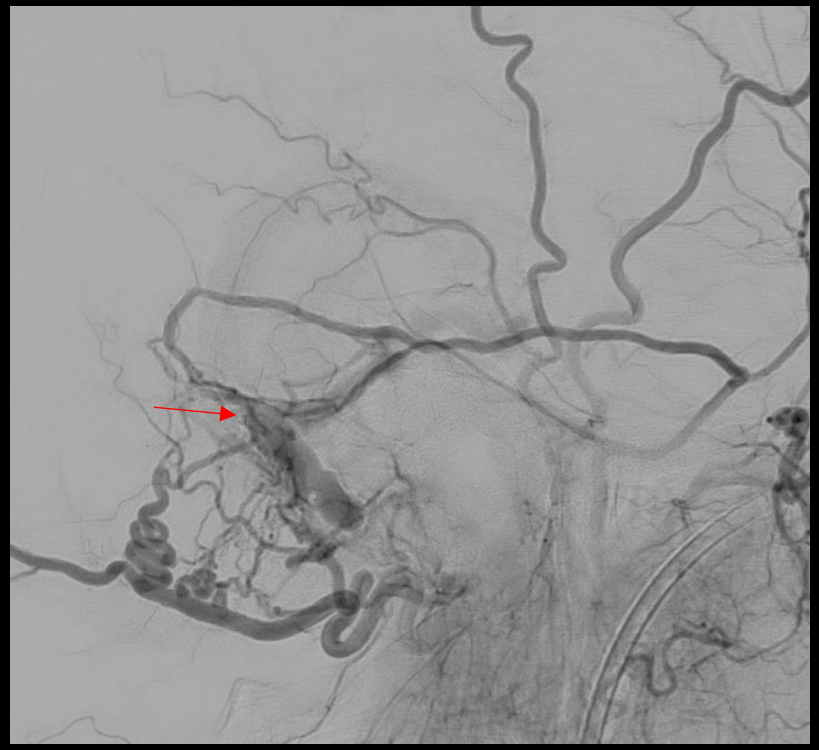


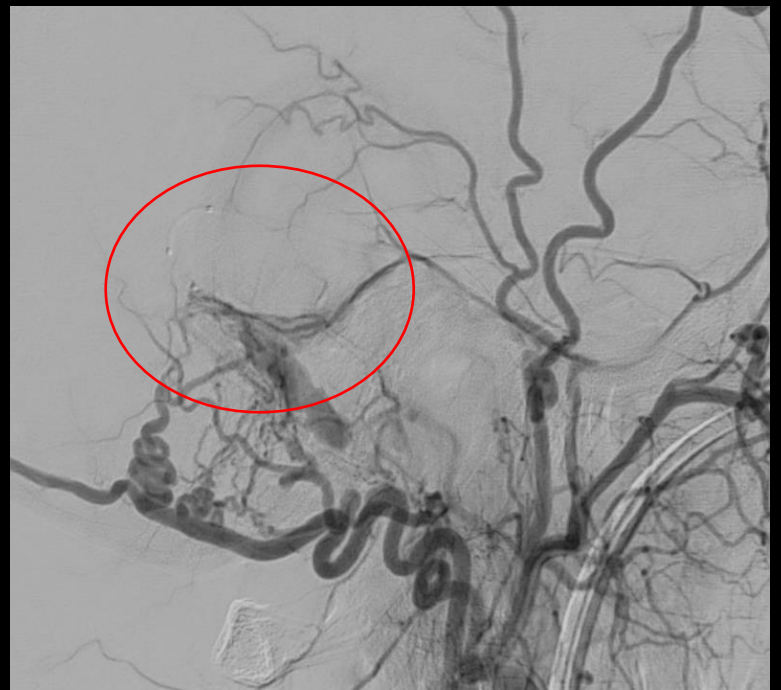




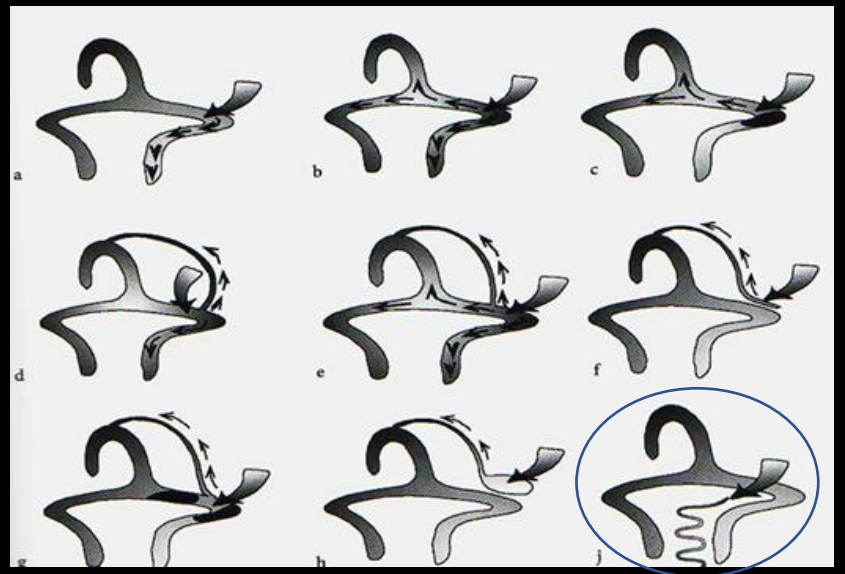


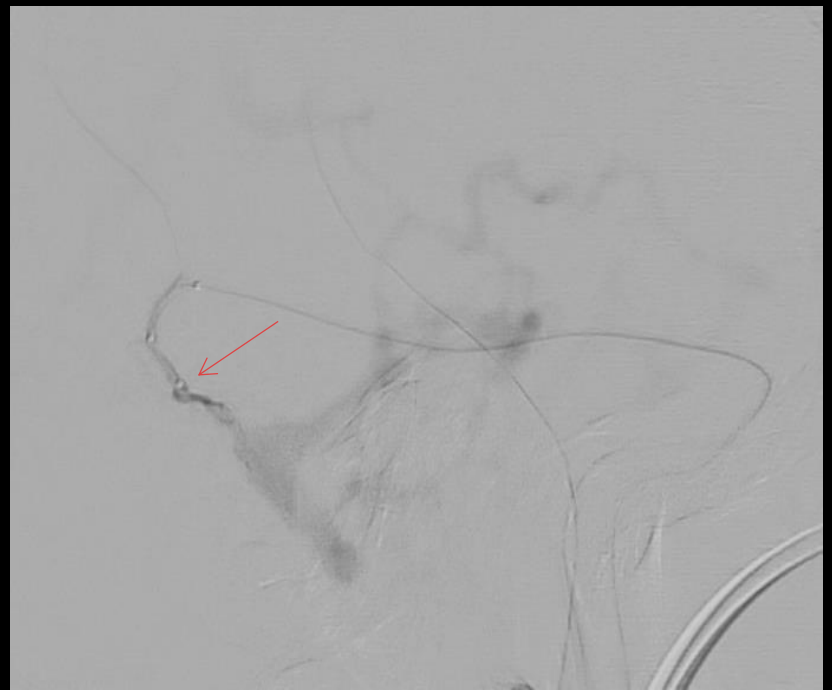


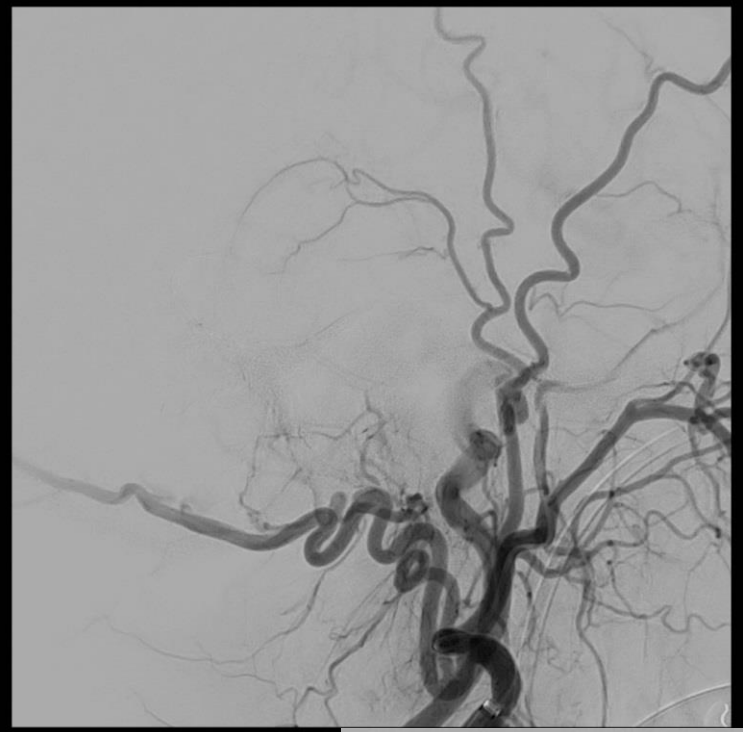


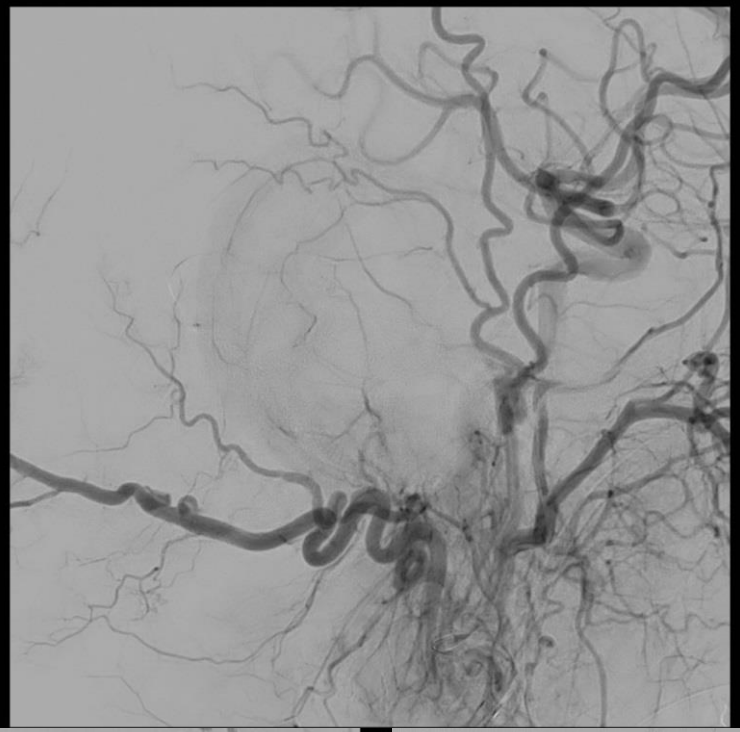


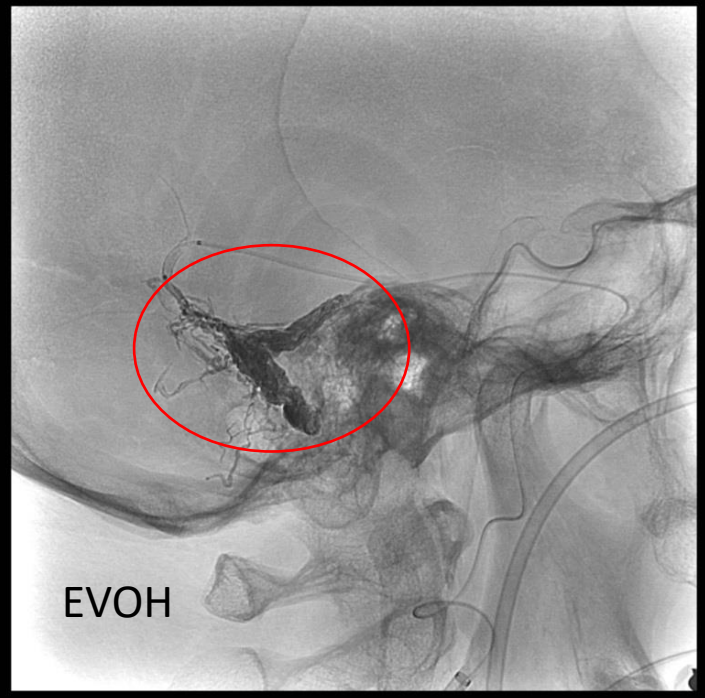
FAVD grado V

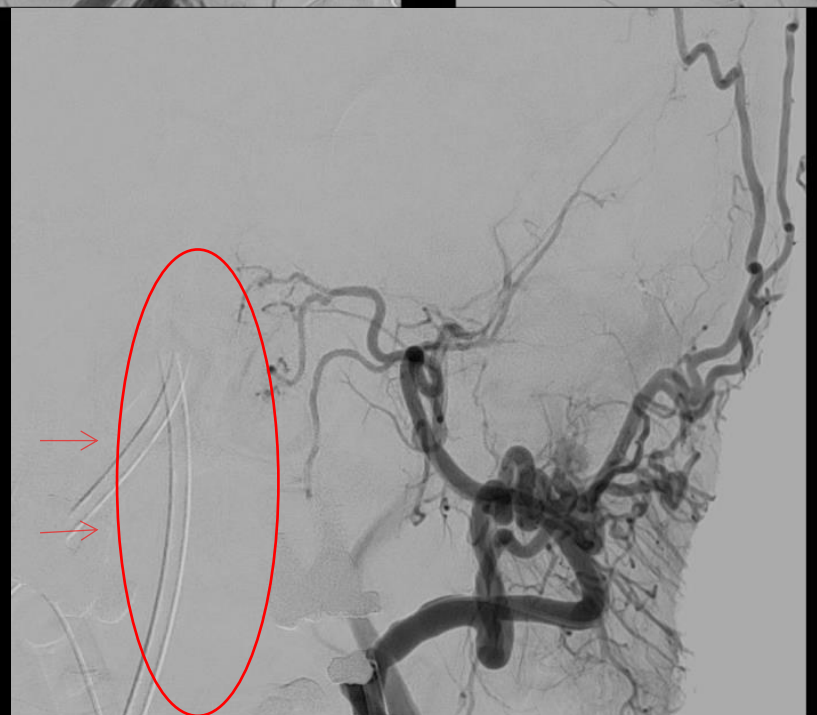


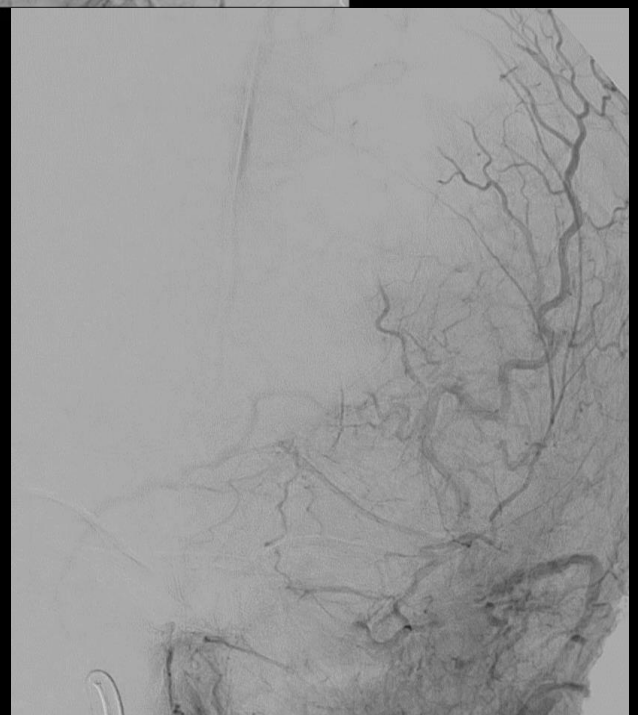


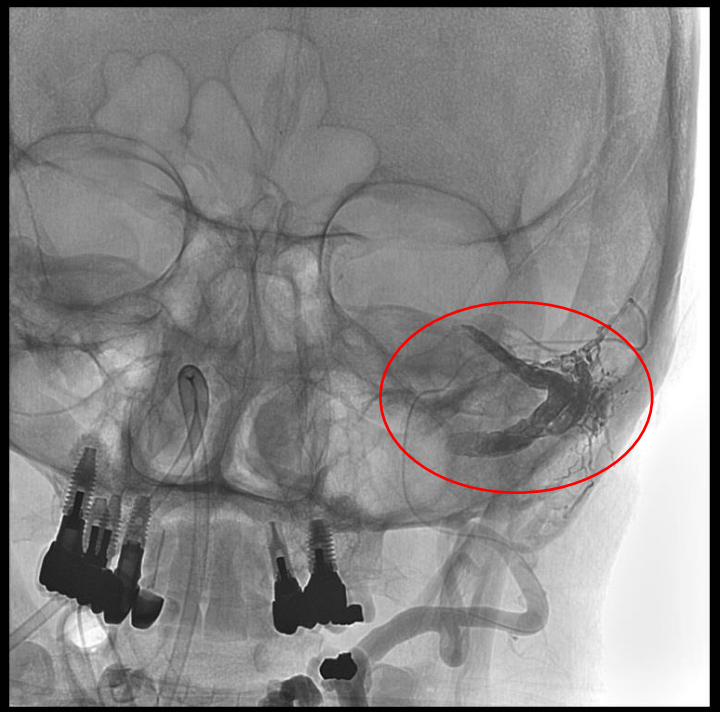





EVOH

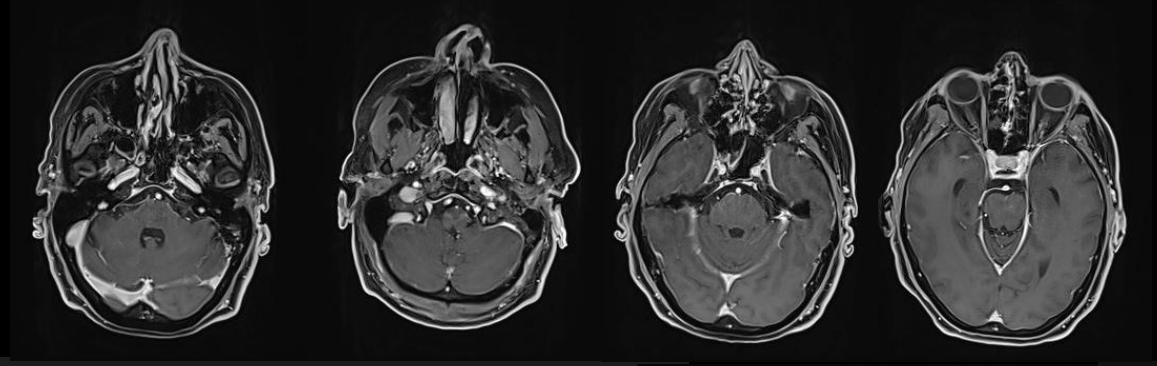


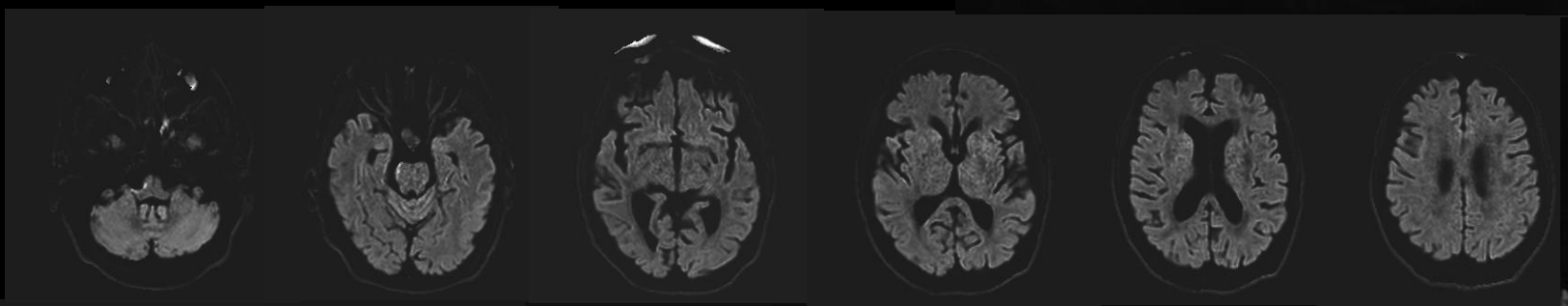


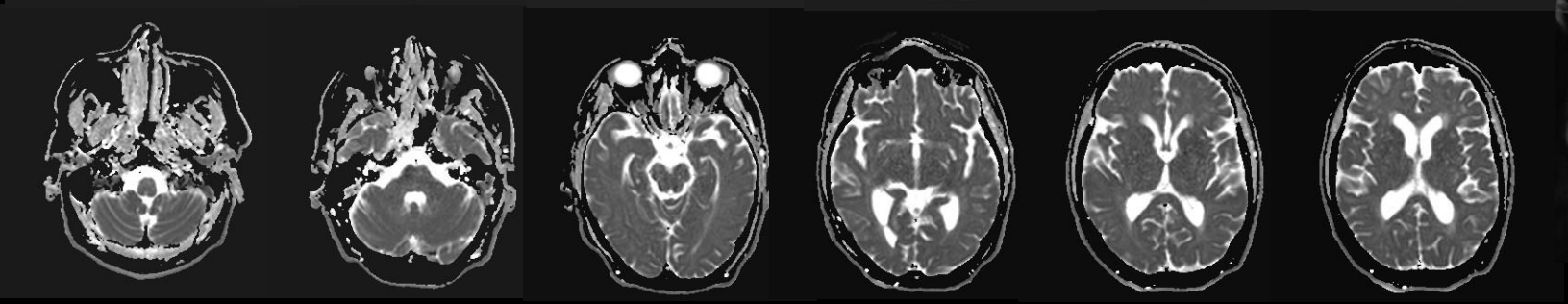


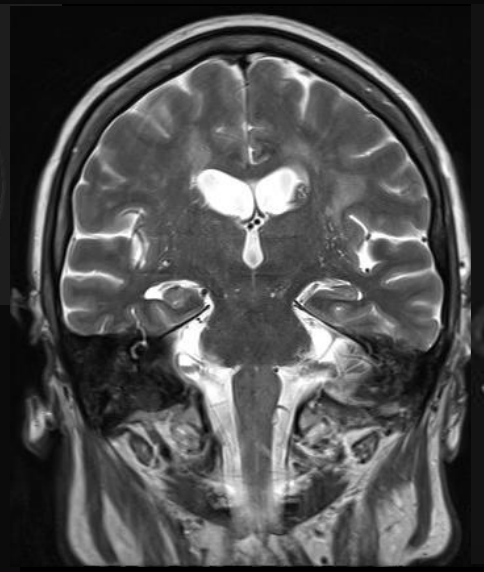
RM CONTROL 7 DÍAS

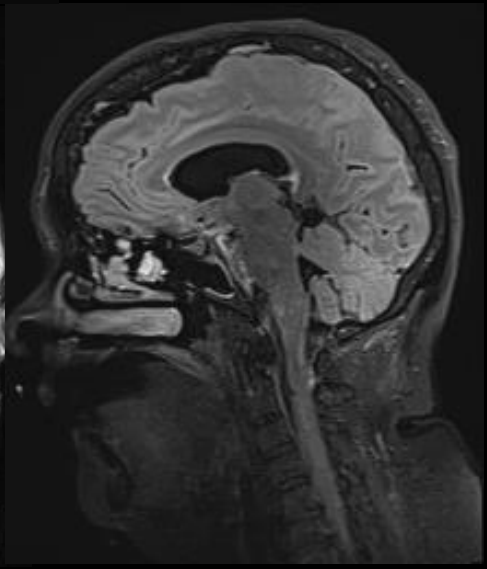
Ausencia de prominencia de vasos perimesencefálicos y bulboprotuberanciales.



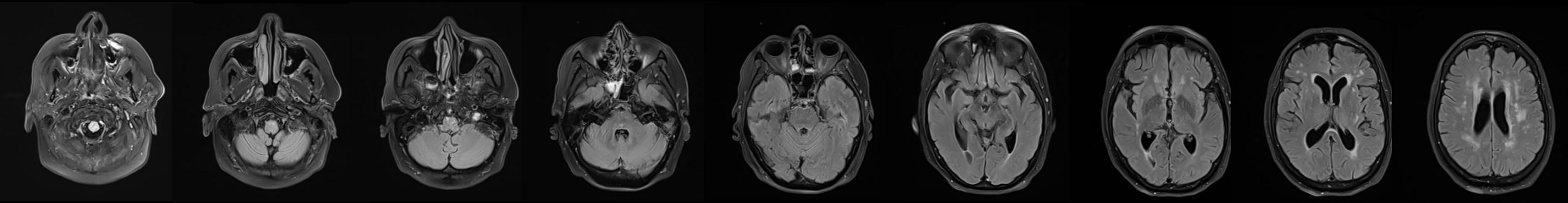


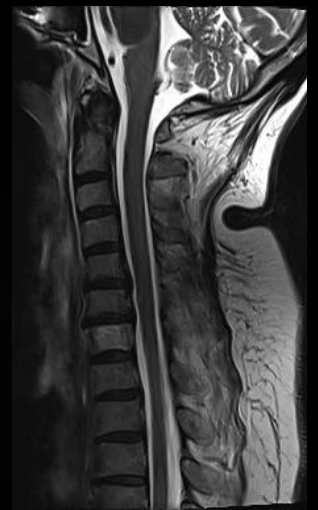




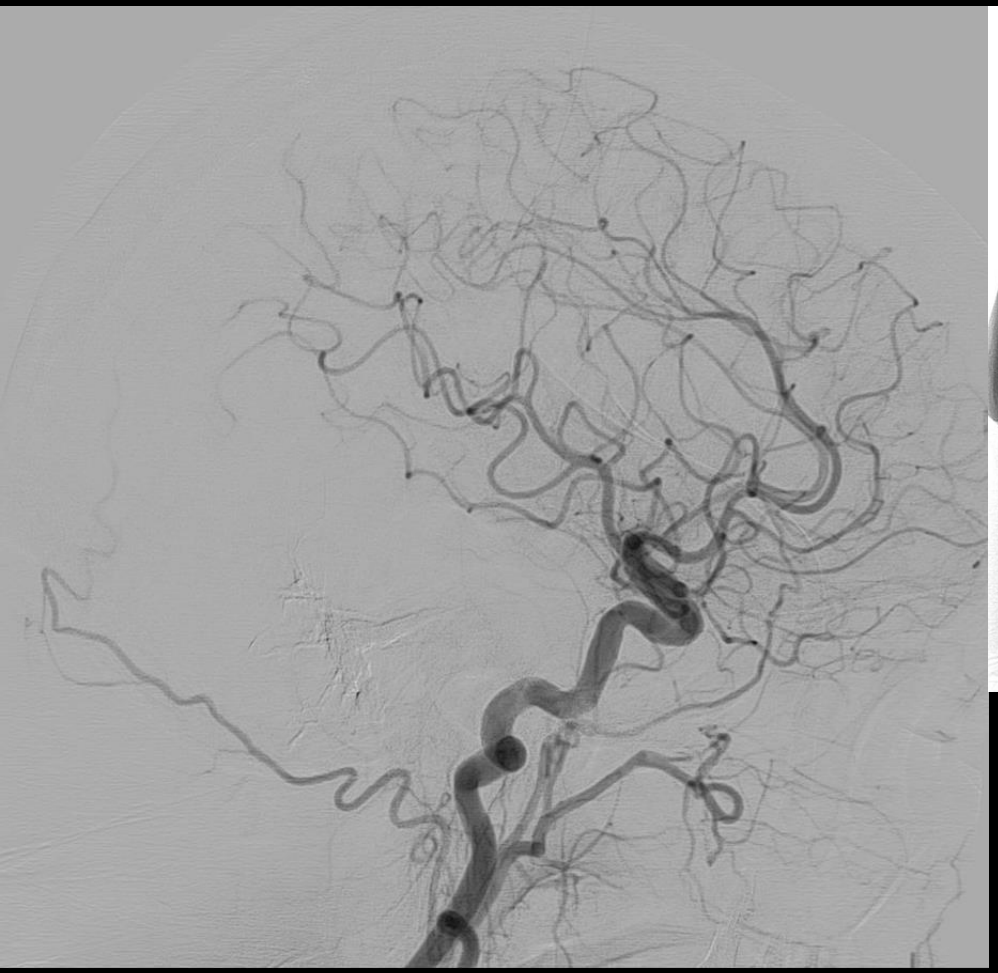


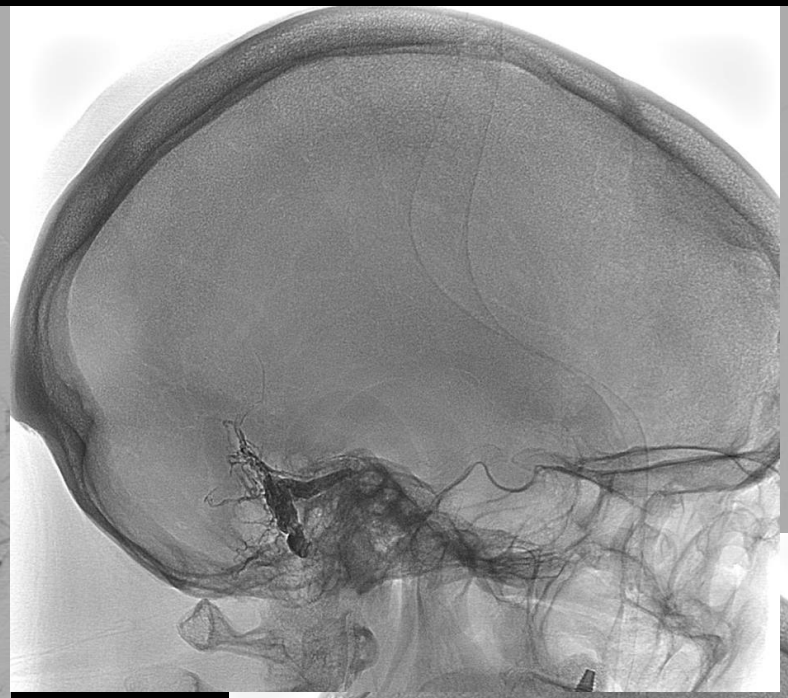
Disminución de la alteración señal en tronco encéfalo y médula, tenue afectación a nivel bulbar y médula cervical

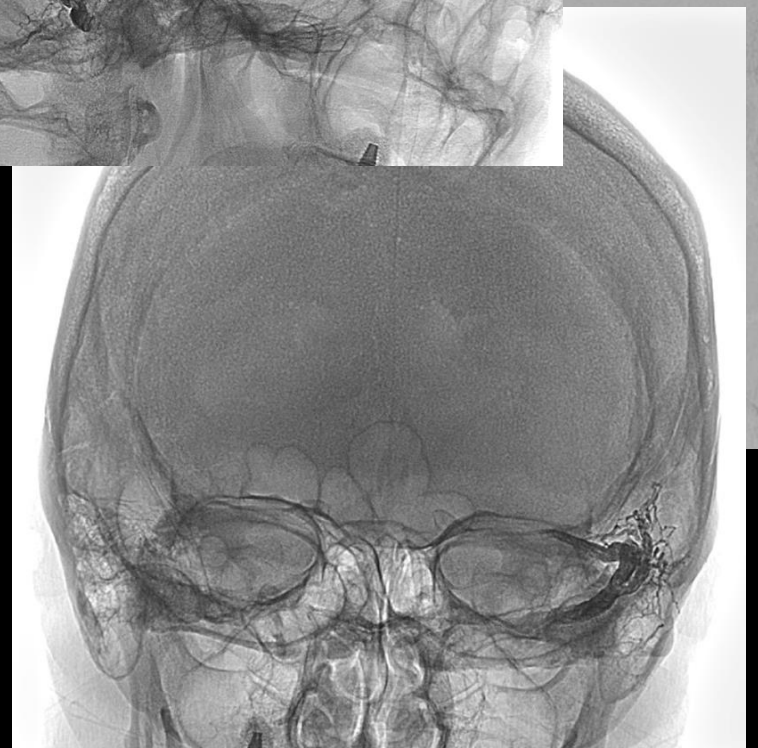


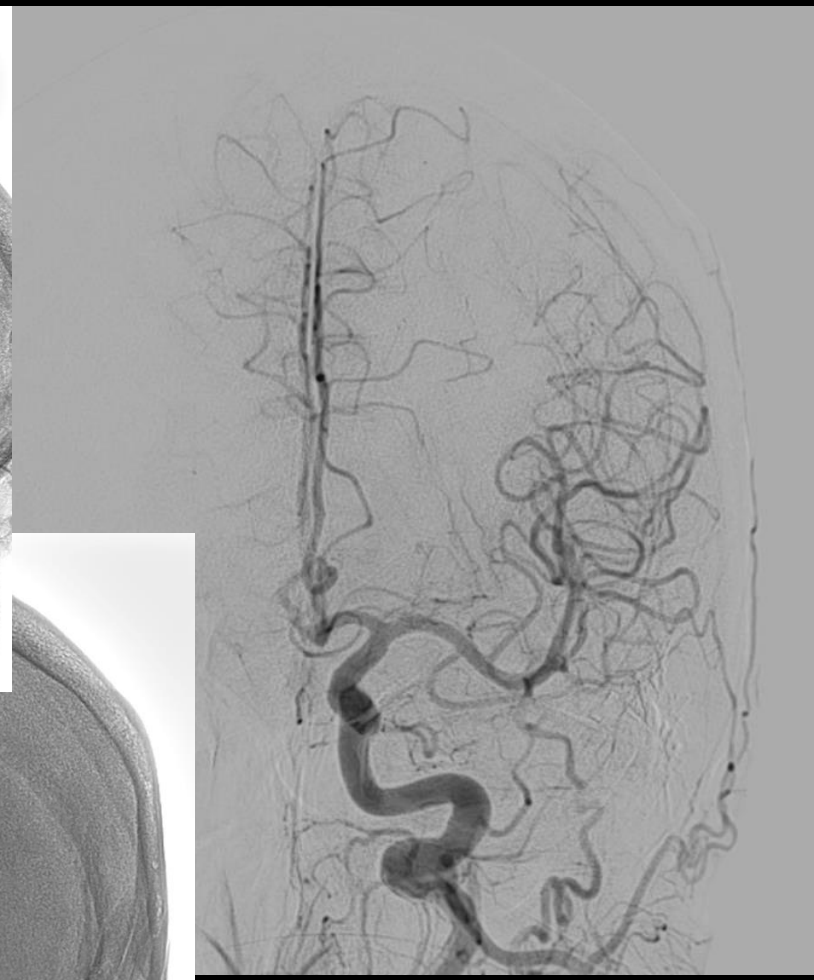


ARTERIOGRAFIA CONTROL 6 MESES









Refiere encontrarse muy bien.
Camina sin ayudas técnicas 5 km
Micción espontanea

FISTULAS AV PIALES

- Comunicaciones directas entre arterias y venas
- Congénitas
- Grandes varices que comprimen el parénquima cerebral

Clasificación topográfica:

* FAVC Galénicas
* FAVC no Galénicas

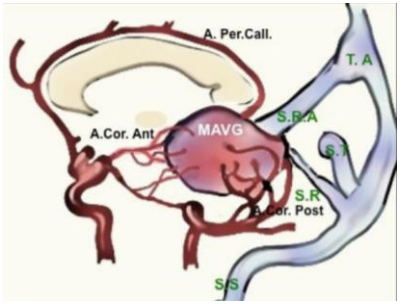
FAV PIAL GALENICA

- Congénita. Edad pediátrica

- Se encuentra en línea media, en la pared de la vena de Galeno dilatada.

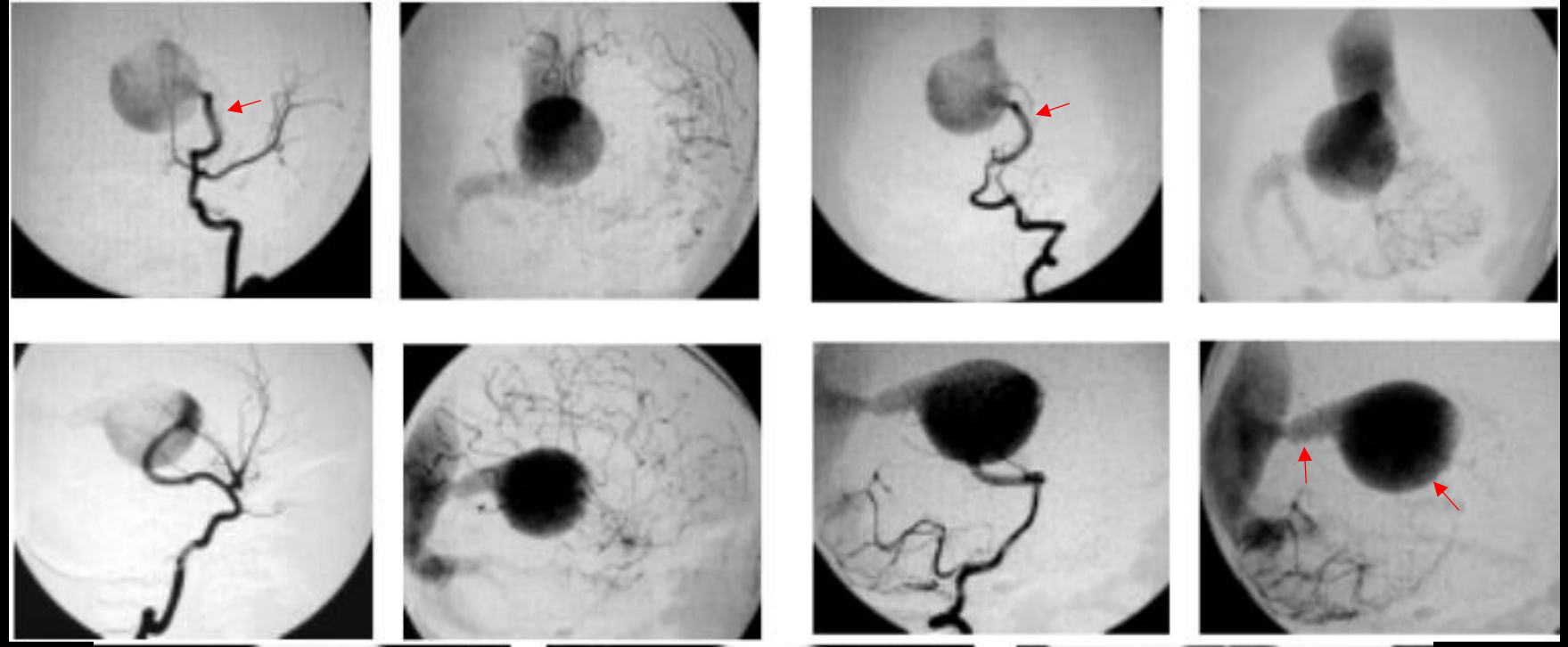
Aferencias coroideas anteriores y posteriores, pericallosa, perforantes transmesencefálicas

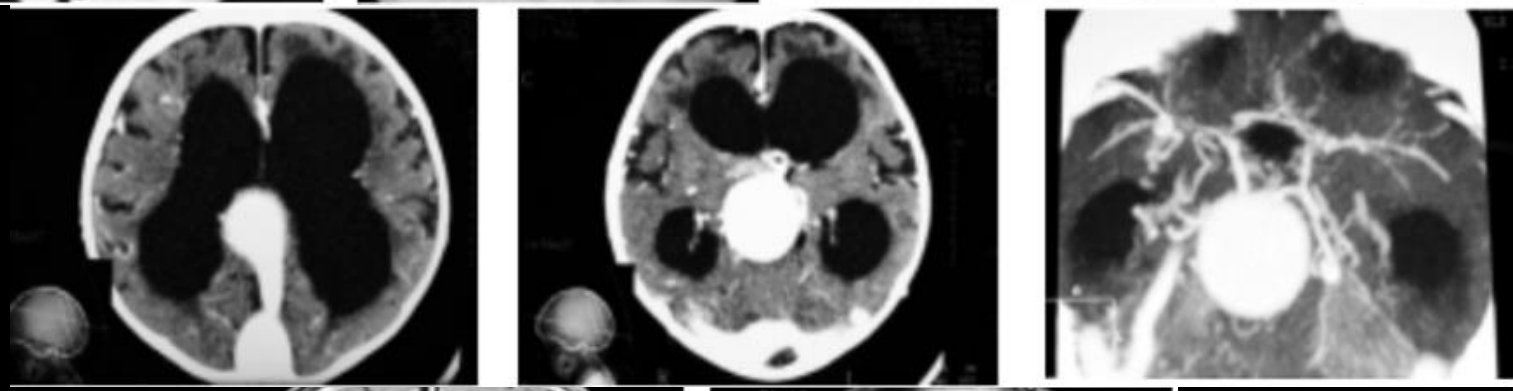
Drenajes a la vena prosencefálica media de Markowski (precursora embrionaria de la vena de Galeno) y seno falcino que drena en el tercio posterior del seno longitudinal

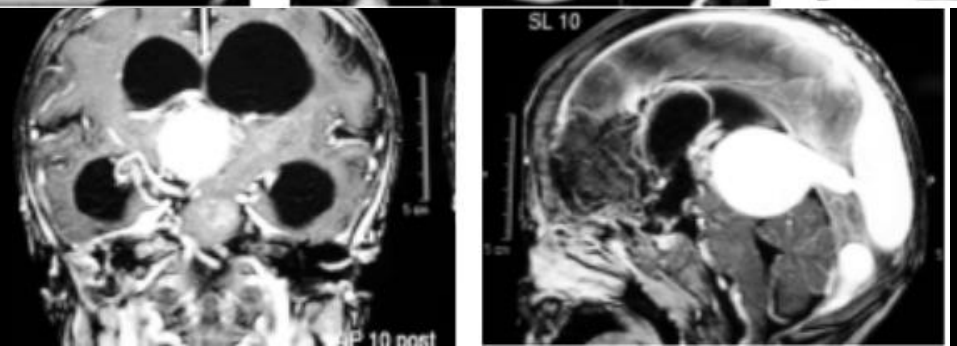


- Clínica: ICC, hipertensión venosa cerebral, hidrocefalia

- El tratamiento: oclusión pedicular selectiva con líquido embólico






SL 10

FAV PIAL NO GALENICA

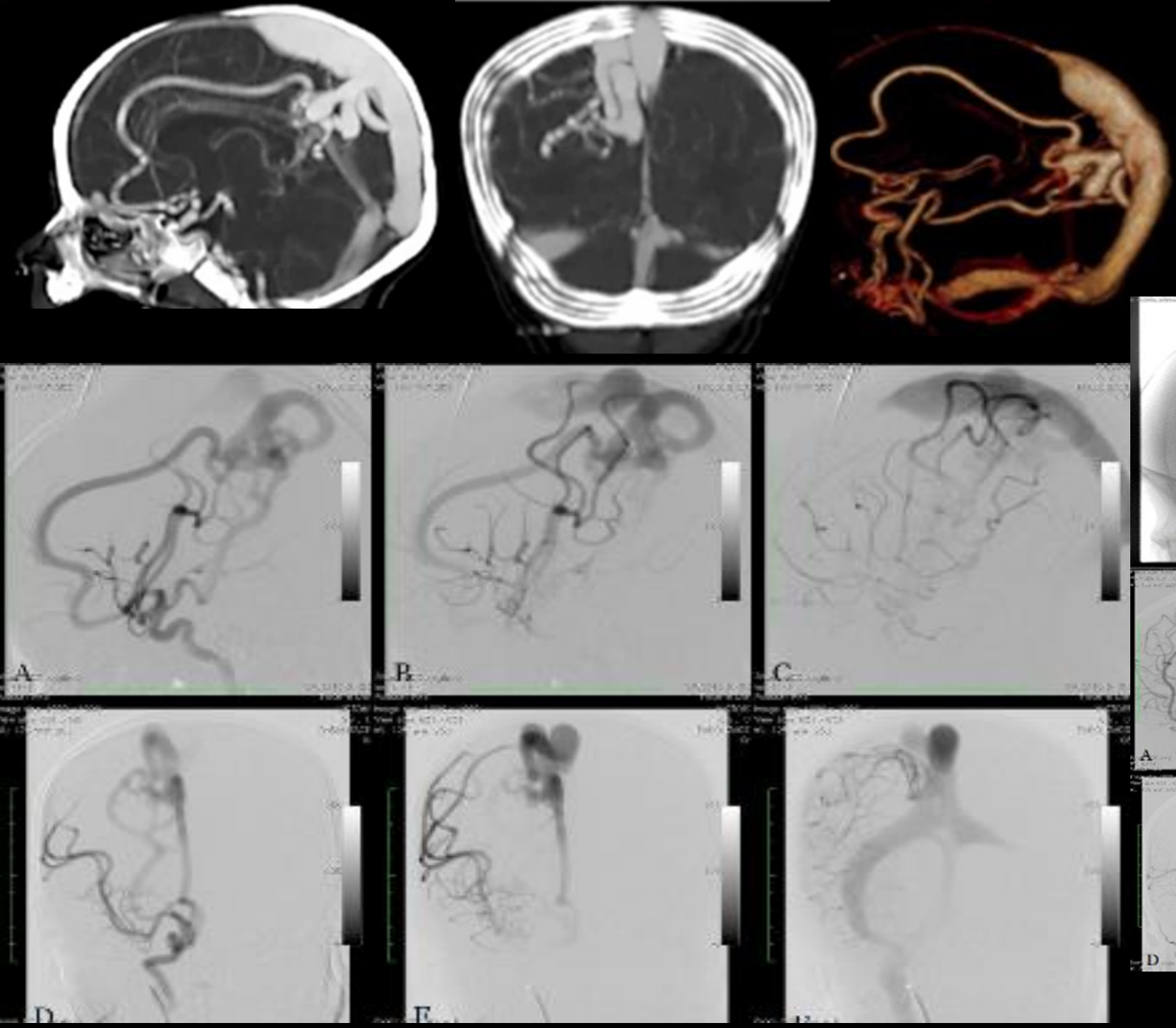
- Neonatos, infantes, adolescentes, adultos

- Supra o infratentoriales

- Conexión directa de una o mas arterias cerebrales, a una o mas varices dilatadas, generalmente extraparenquimatosas, comprimiendo estructuras

- Efecto masa, convulsiones, hidrocefalia, ICC

- El tratamiento es la oclusión del pie de vena


A
B
C
D
E
F

MAV y FAVD Cerebrales

HOSPITAL UNIVERSITARIO DE CRUCES

NUESTRA EXPERIENCIA