MAV ET THROMBOSE VEINEUSE CEREBRALE.
SIGNES IRM ET IMPLICATIONS THERAPEUTIQUES.

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MAV et hémorragie cérébrale

- Facteurs de risque hémorragique connus :
  - Type de drainage
  - Loc. Profonde de la MAV
  - Début hémorragique
  - Fosse post

  - *Stapf Neurology 2006*
  - *Hernesniemi 2008*

Insatisfaisant pour de nombreux cas

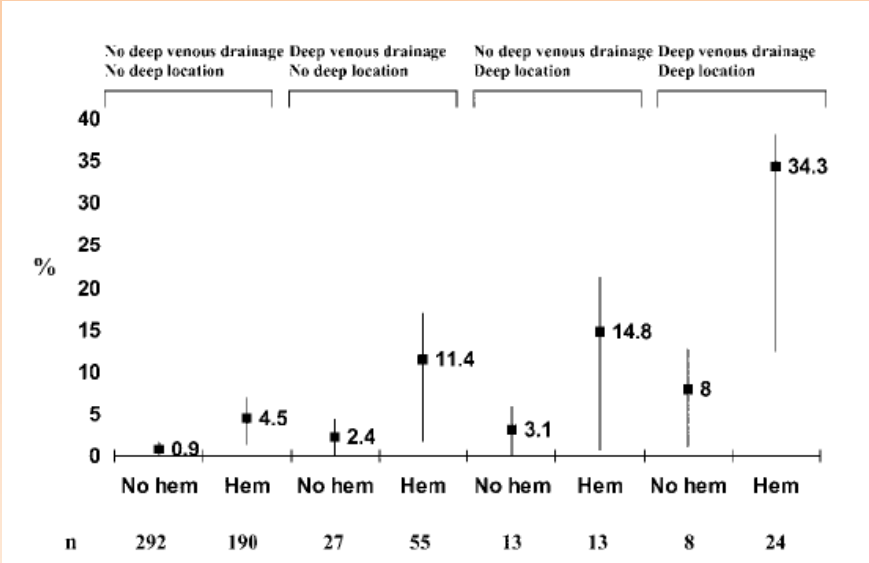


TABLE 2. Annual and cumulative rupture rates in relation to previous rupture, supra- or infratentorial location, superficial or deep location, arteriovenous malformation size, and pattern of venous drainage[a]

Characteristic	No. of patients	Annual rupture rates (%)			Cumulative rupture rates, % (95% CI)		Log-rank *P* values	
		0–5 years after admission	> 5 years after admission	Whole follow-up period	5 years after admission	20 years after admission	First 5 years after admission	Entire follow-up period
All patients	238	4.7	1.6	2.4	21 (15–27)	39 (32–47)		
Sex							0.265	0.250
Male	141	4.0	1.5	2.1	18 (11–25)	37 (27–47)		
Female	97	5.8	1.7	2.8	25 (15–35)	43 (31–66)		
Previous rupture							0.011	0.016
Ruptured	139	6.2	1.7	2.8	26 (19–34)	45 (27–63)		
Unruptured	99	2.3	1.3	1.6	10 (3–17)	29 (16–42)		
Supra- or infratentorial AVM							0.023	0.008
Supratentorial	218	4.3	1.5	2.2	19 (13–25)	37 (29–45)		
Infratentorial	18	11.6	3.6	6.7	45 (18–72)	76 (51–100)		
Superficial or deep AVM							0.003	0.003
Superficial	170	3.5	1.4	1.9	16 (10–22)	35 (27–44)		
Deep	66	8.9	2.2	4.1	35 (22–49)	53 (38–67)		
AVM size							0.807	0.220
Small	88	5.0	1.0	1.9	22 (12–32)	33 (21–45)		
Medium	96	4.2	1.6	2.3	17 (9–26)	38 (25–51)		
Large	47	5.5	2.7	3.5	24 (11–36)	52 (35–69)		
Venous drainage							0.013	0.111
Cortical and deep	42	1.2	1.9	1.7	5 (0–13)	18 (3–33)		
Cortical	122	4.5	1.4	2.1	20 (12–28)	38 (29–47)		
Deep	64	8.1	1.6	3.4	34 (20–48)	52 (37–68)		

[a] AVM, arteriovenous malformation; CI, confidence interval.

MAV et hémorragie cérébrale

World Neurosurg. 2013 Feb 5. pii: S1878-8750(13)00270-2. doi: 10.1016/j.wneu.2013.02.005. [Epub ahead of print]

Venous flow rearrangement following the treatment of cerebral AVMs: A novel approach to evaluate the risks of treatment.

D'Aliberti G, Talamonti G, Piparo M, Debernardi A, Zella S, Boccardi E, Valvassori L, Nichelatti M.

Department of Neurosurgery, Niguarda CA' Granda Hospital, Milan Italy.

Results

An overall number of 245 patients (61.2%) achieved favourable outcomes. Hyperemic complications occurred in a total of 28 patients (7%): no patient belonged to the *Group 1* so that these adverse effects were reported in 30.7% of *Group 2* patients. The presence of deep drainage and the number of recruited veins resulted to have statistically different impacts on the risk of the different grades.

- Origine veineuse des complications hemorragiques ..
  - Loc. profonde
  - Ralentissement veineux arteriographiques ..(?)

Action de proteines inflammatoires « parieto-aggressive »

- **Li Xiong et al. in Zhonghua Yi Xue Za Zhi China.**
- Z HIRESULTS: The blood levels of IL-6 in the ruptured group were significantly higher than those in the non-ruptured and control groups (33.2 +/- 4.8 vs 23.8 +/- 1.2 ng/L, $P < 0.05$; 33.4 +/- 4.8 vs 15.6 +/- 1.0 ng/L, $P < 0.01$). Protein expression in the non-ruptured group was greater than that in the normal and ruptured groups (1.20 +/- 0.35 vs 0.34 +/- 0.07; 1.20 +/- 0.35 vs 0.31 +/- 0.09, unit:molecular weight ratio of MMP-9 and beta-actin), and gelatin zymography showed that the activity of MMP-9 was significantly higher in the ruptured than the non-ruptured and control groups (0.98 +/- 0.07 vs 0.40 +/- 0.09; 0.98 +/- 0.07 vs 0.30 +/- 0.07, unit: ratio of MMP-9 and standard). In the ruptured group, active MMP-2 expression was significantly higher than that in the other groups.
- CONCLUSION: IL-6 may stimulate the transformation of MMP-9 into activated form in ruptured AVM tissues and thus lead to an elevated hemorrhage risk of AVM

		MMP 9 expression	IL 6	MMP 9 activée
Rompues	14	0,31	33	0,98
Non rompues	17	1,2	23	0,3
Control	30	0,34	15	0,4

Lesions parietales veineuses ????

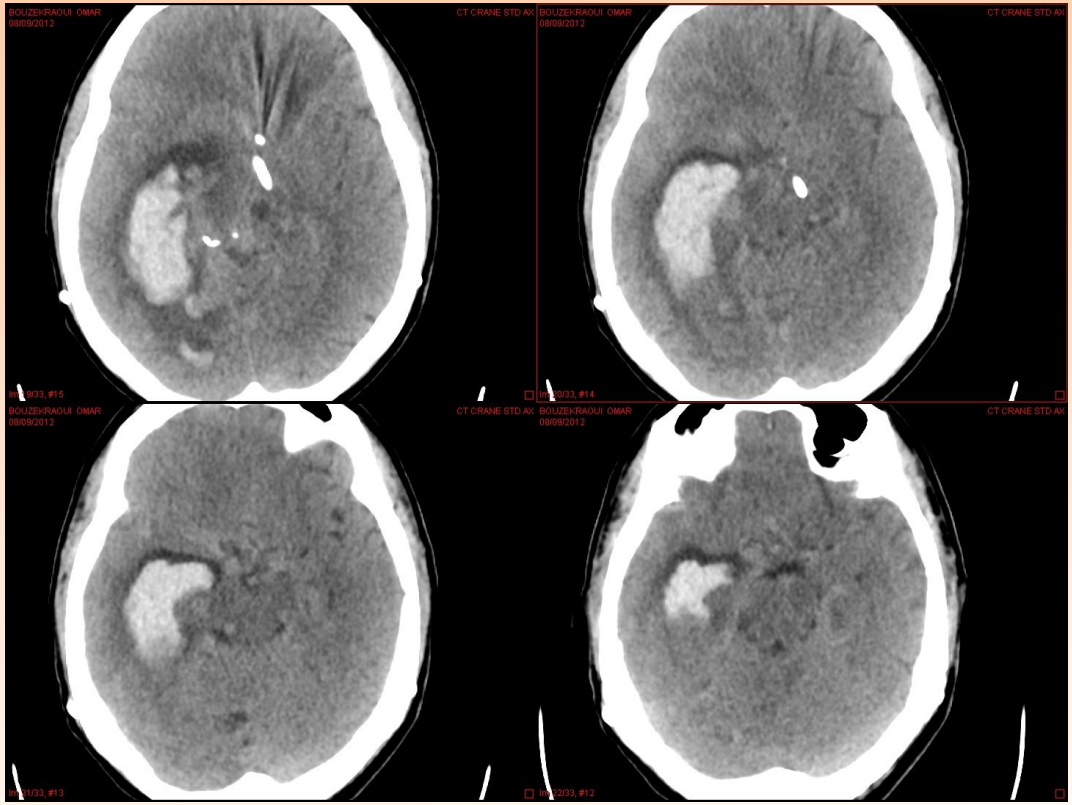
Analyse de Cas

- 4 cas de MAV
- 1 rompu
- 4 symptomatiques
- 4 thrombophlebites authentifiées

- Suivi pré post TT
- TDM
- Arteriograghie
- IRM

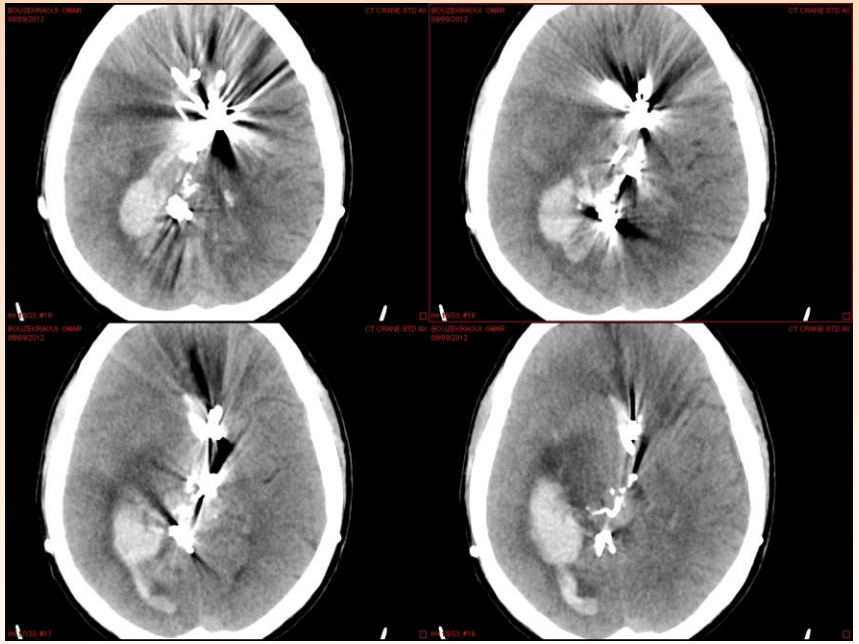
Cas 1 thrombose profonde hemorragique

- Cas 1
- Homme 42 ans
- MAV traitée
- 4 e hémorragie

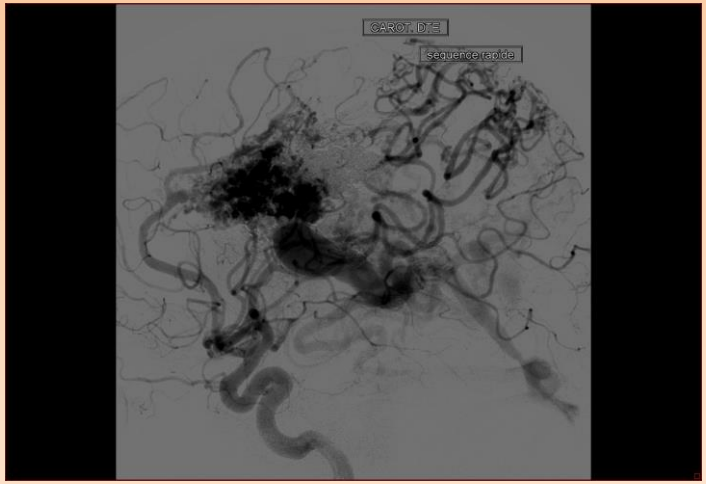


Cas 1 thrombose profonde hémorragique

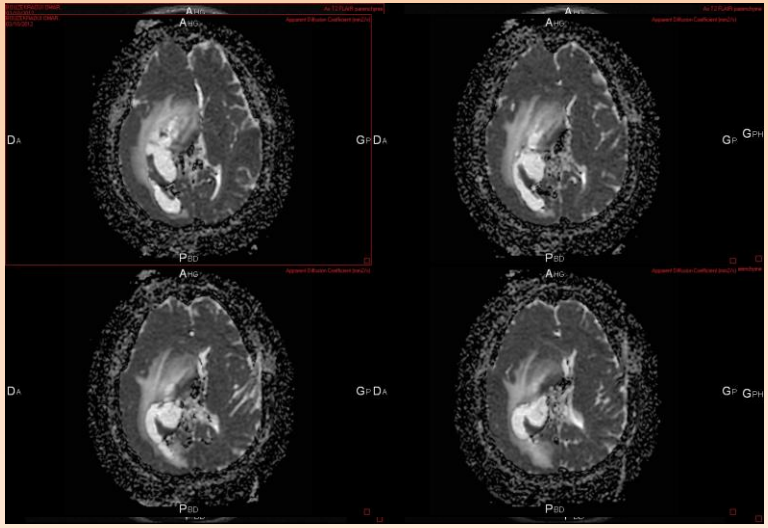
- Transfert USI
- Etat neurologique stable G13
- Hemiparesie gauche



Cas 1 thrombose profonde hemorragique

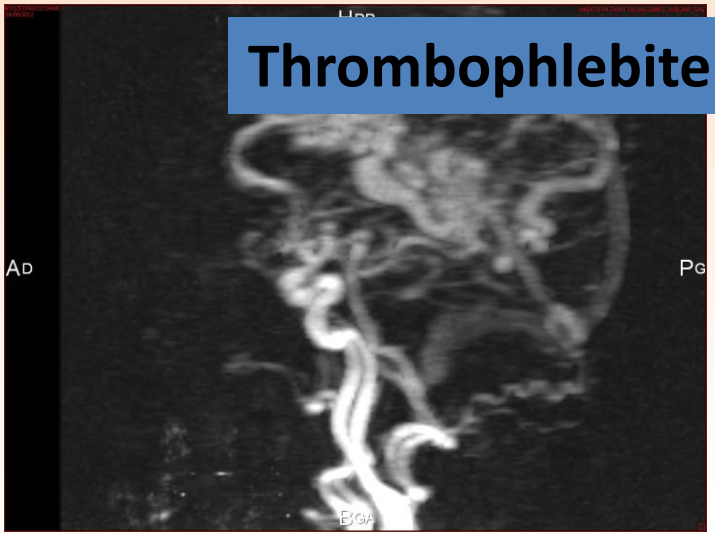


J2

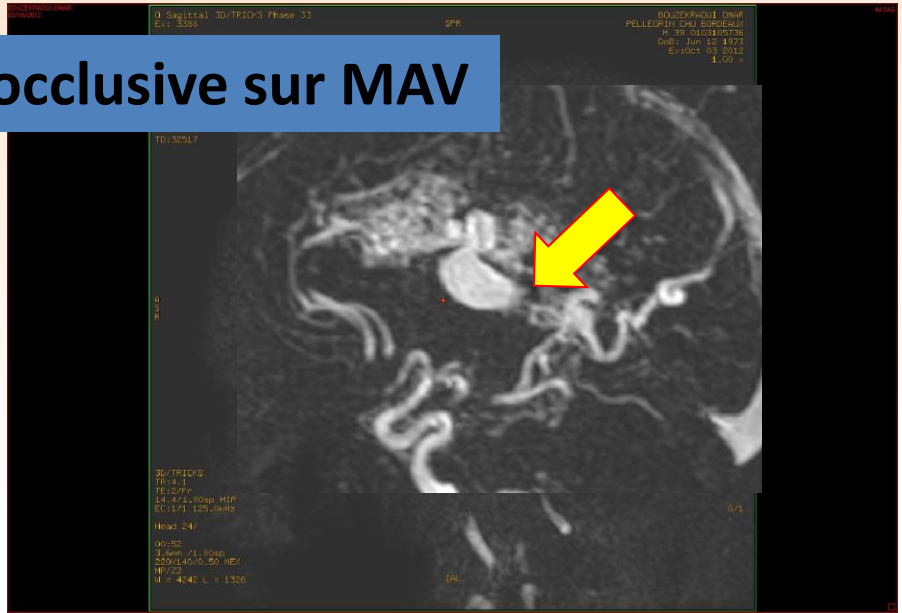


Aggravation J7

Thrombophlebite sub occlusive sur MAV

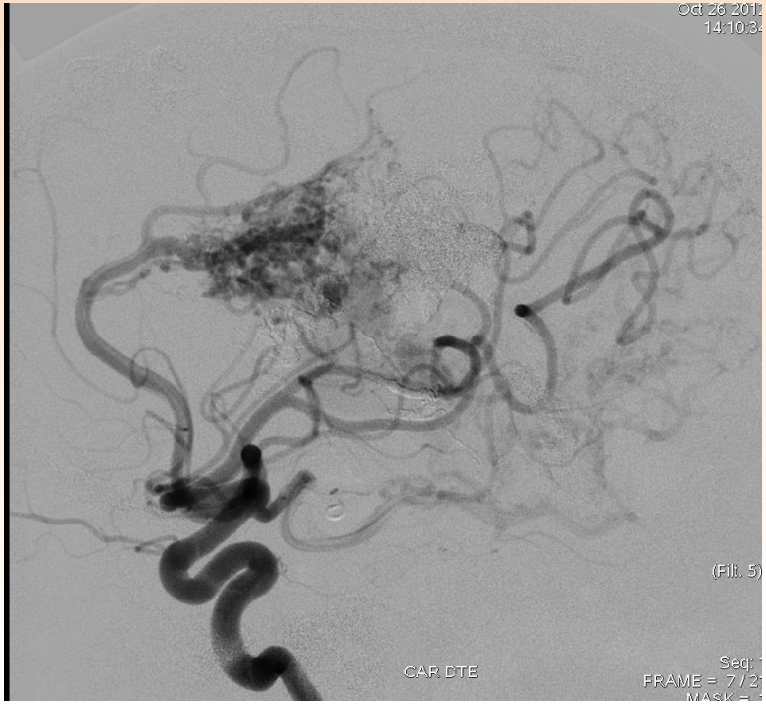


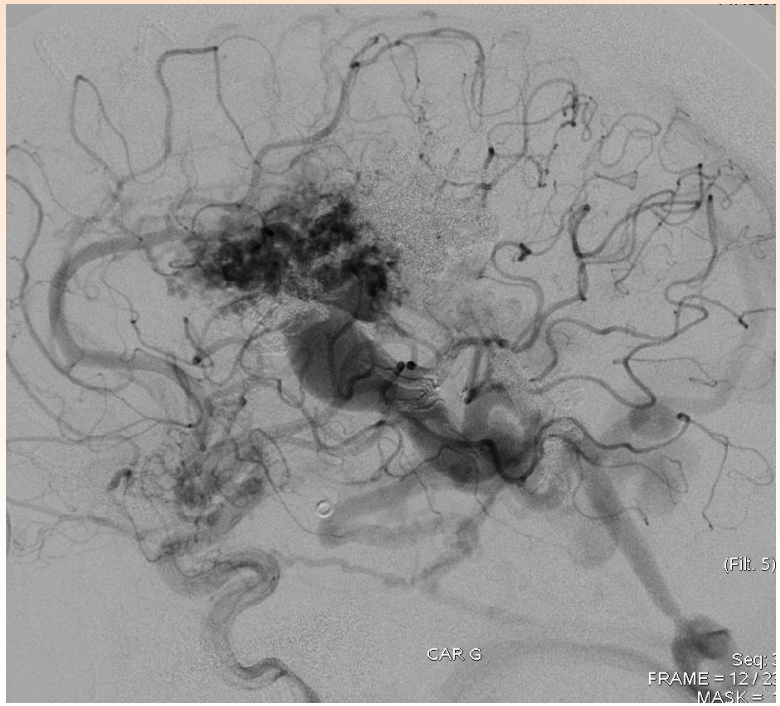
IRM J1



Cas 1 thrombose profonde hemorragique

- Anticoagulation efficace
- TCA cible 2T
- Pas d’embolisation

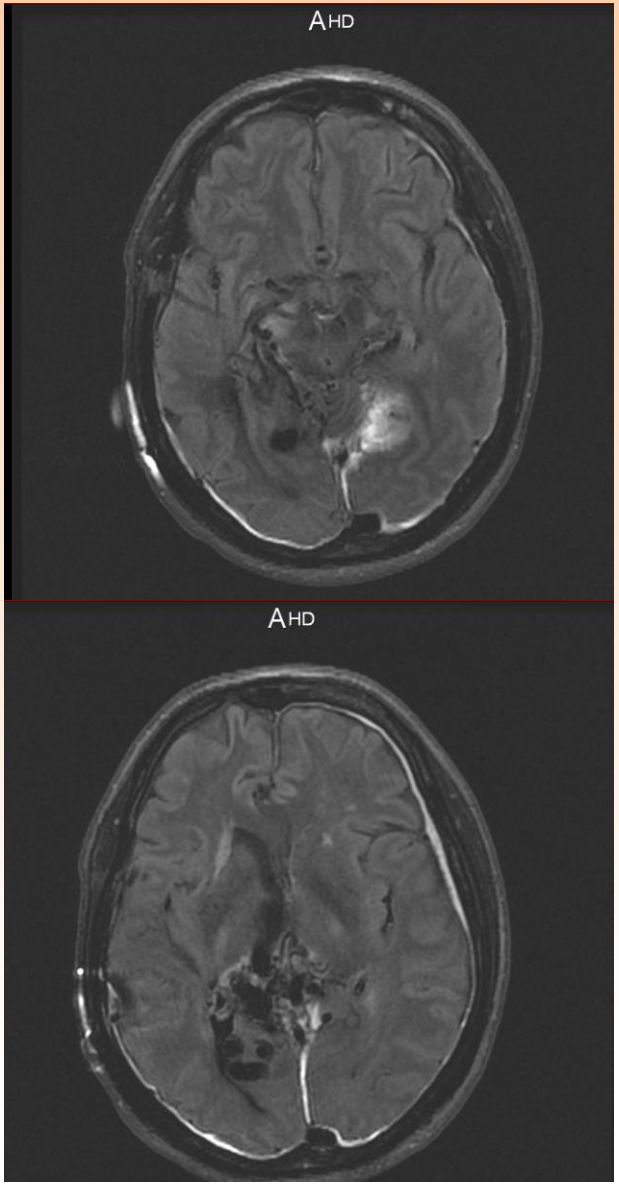




Cas 1 thrombose profonde hemorragique

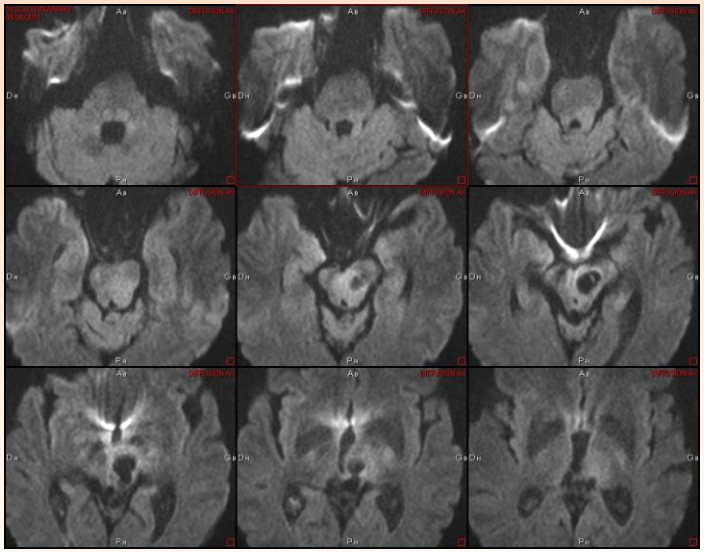
IRM J30
Normalisation oedeme
Regression de
l'hemiparesie gauche

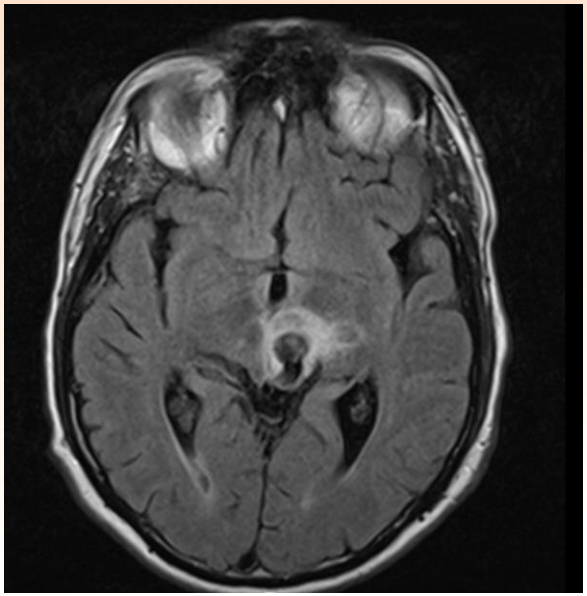
REGRESSION OEDEME
VEINUEUX



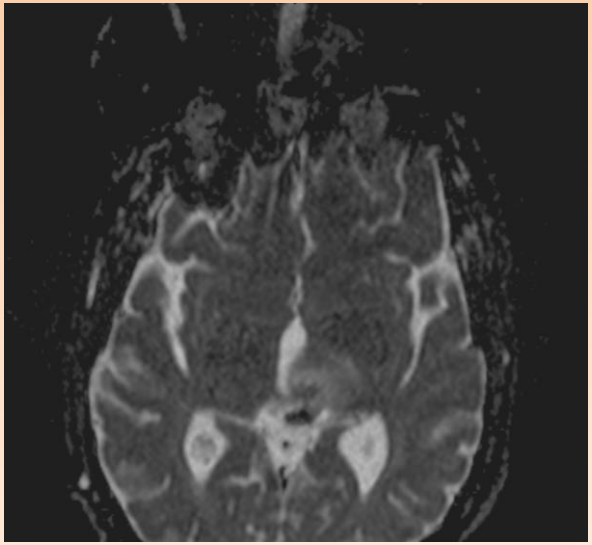
Cas 2 thrombose profonde non hemorragique

- Homme 58 ans
- Diplopie et cephalées depuis 8 j
- Aggravation par hemiparesie droite

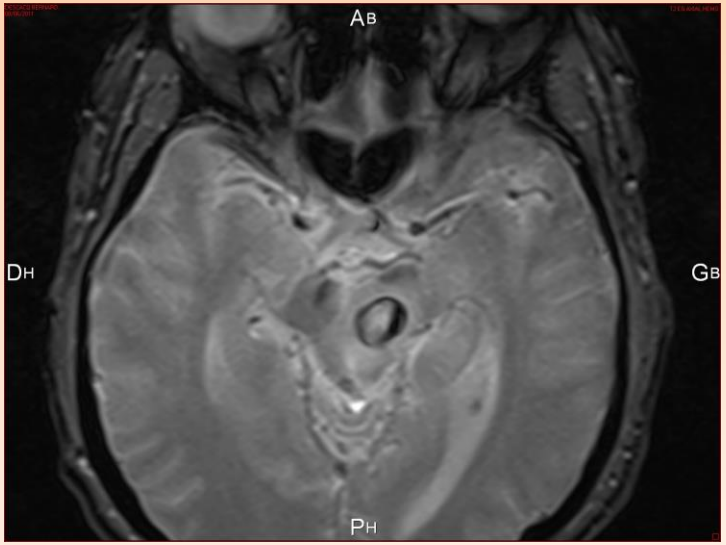




Cas 2 thrombose profonde non hemorragique

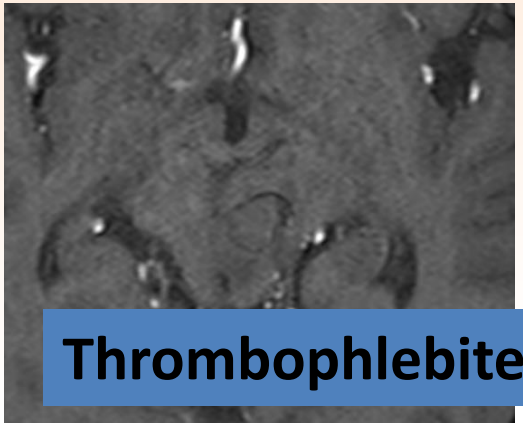


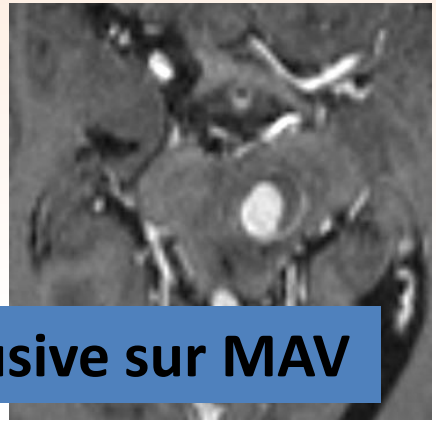
ADC augmenté



Thrombus veineux en T2*

TOF

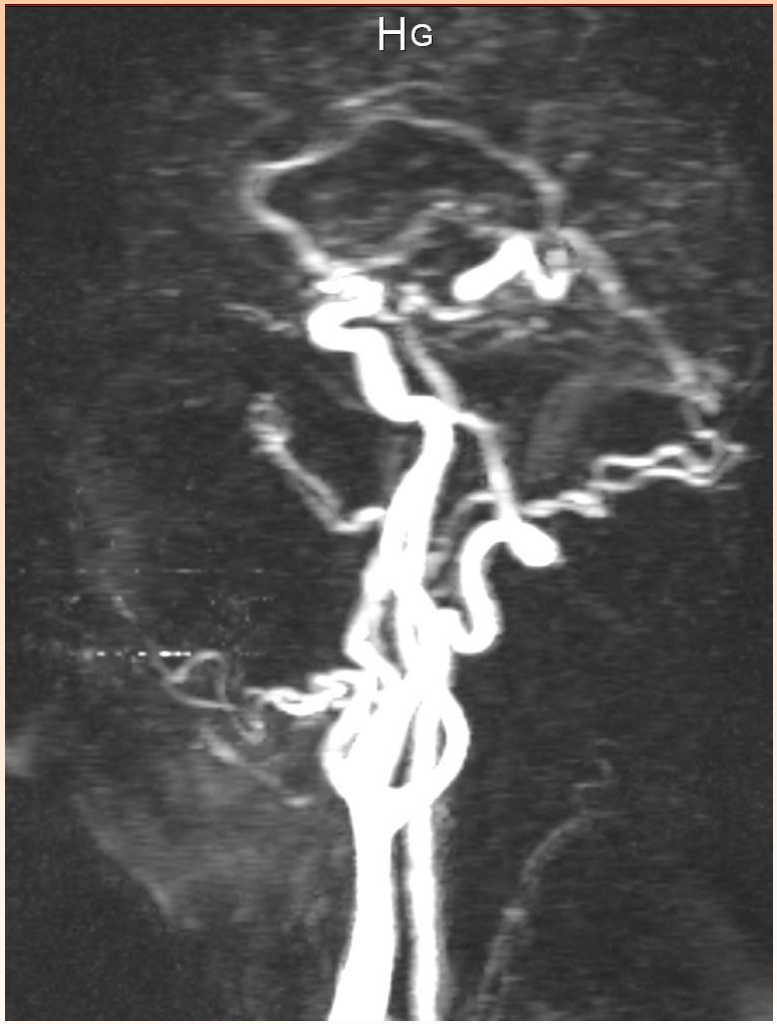


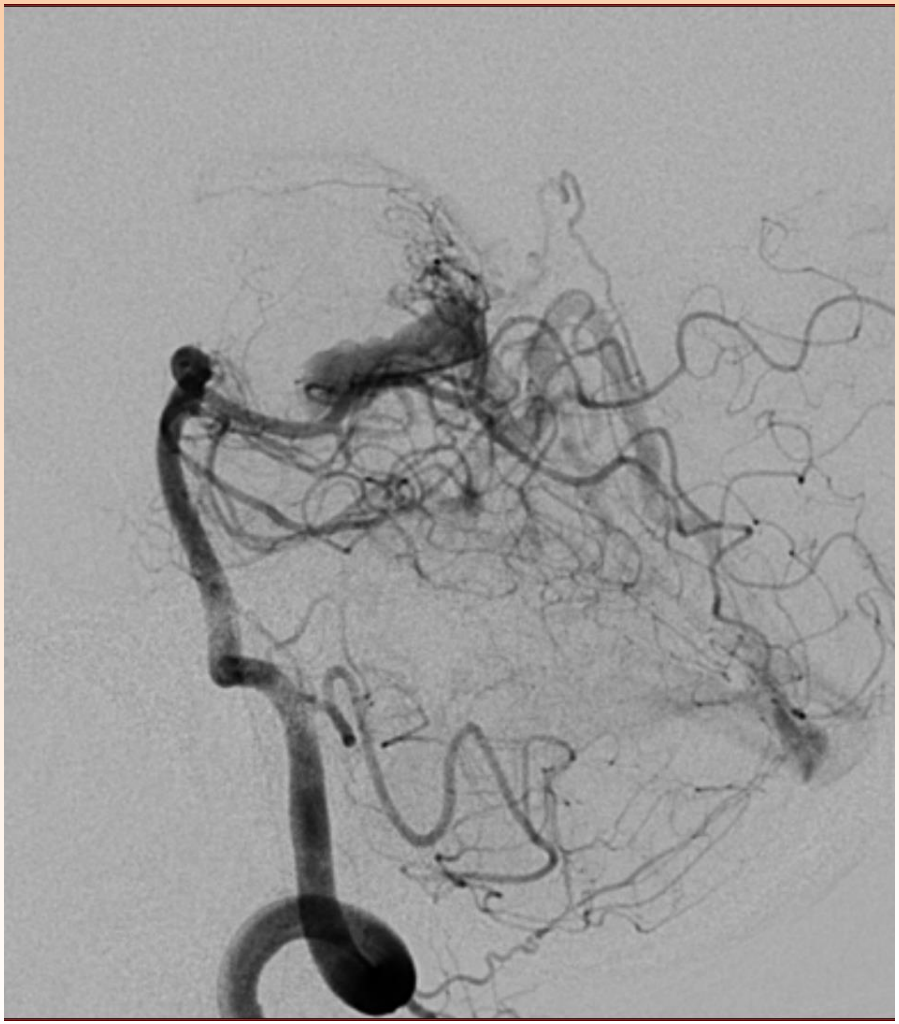


TOF injecté

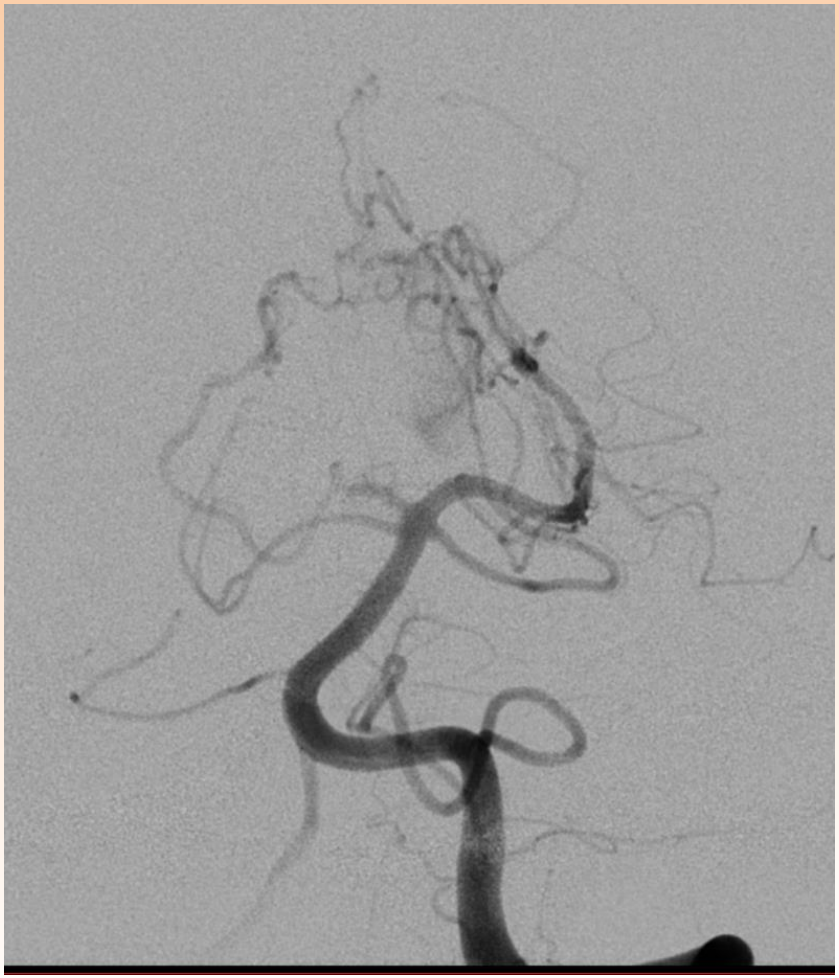
Thrombophlebite non occlusive sur MAV

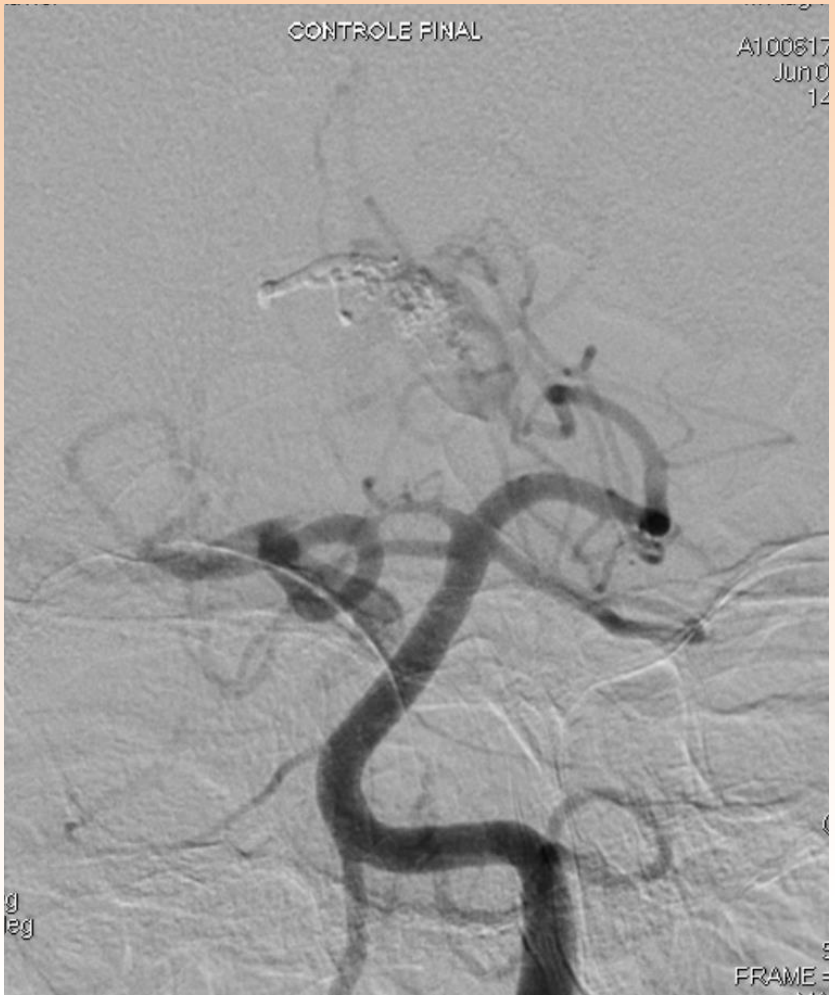
Cas 2 thrombose profonde non hémorragique





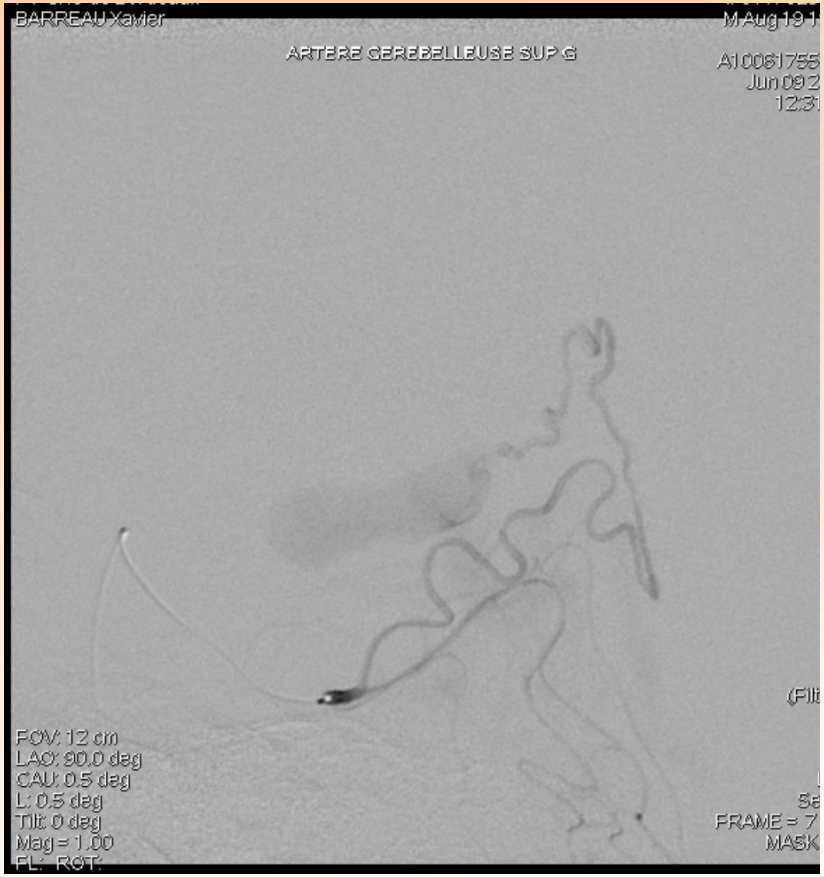
Cas 2 thrombose profonde non hemorragique



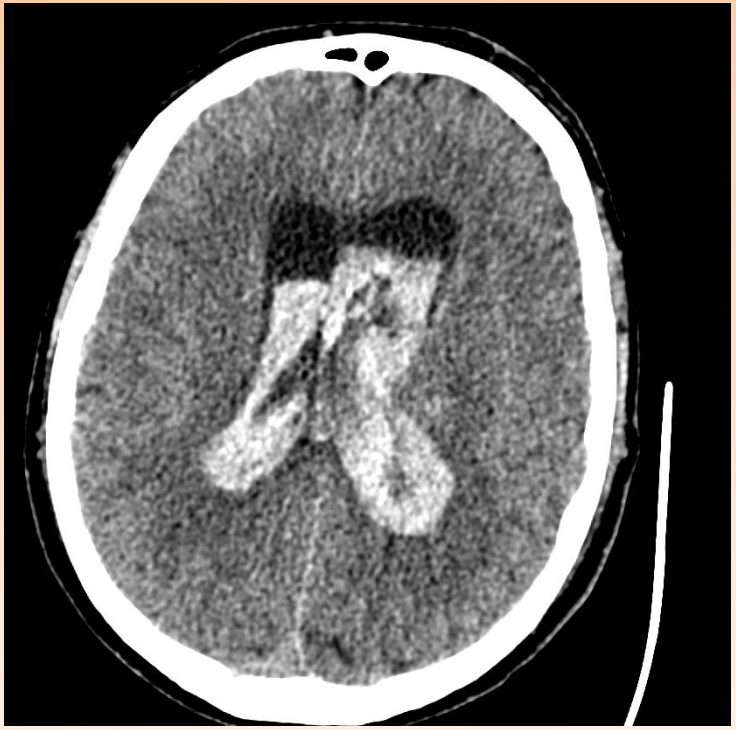


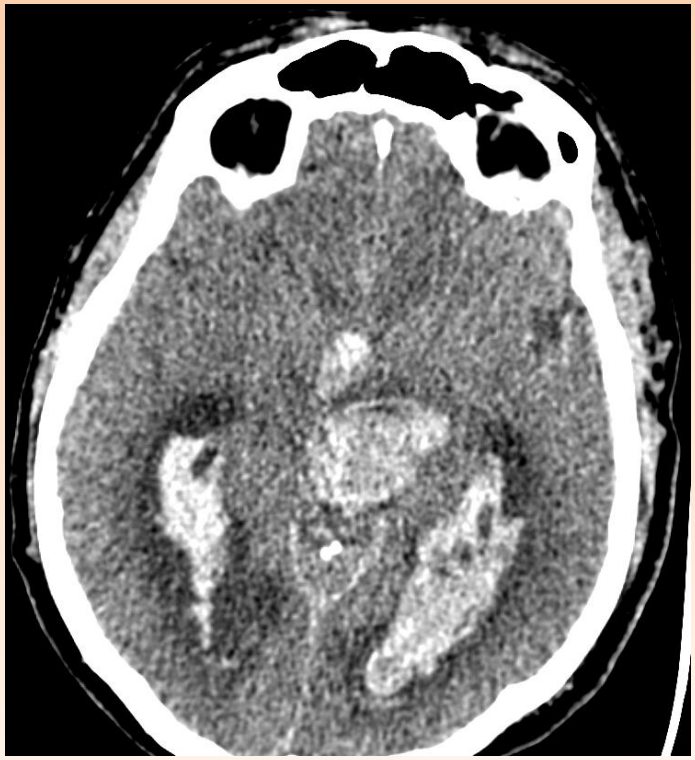
Cas 2 thrombose profonde non hemorragique

- Catheterisme distal complexe
- Embol incomplete
- Anticoagulation effcicace
- Decision de reprise



Cas 2 thrombose profonde non hemorragique

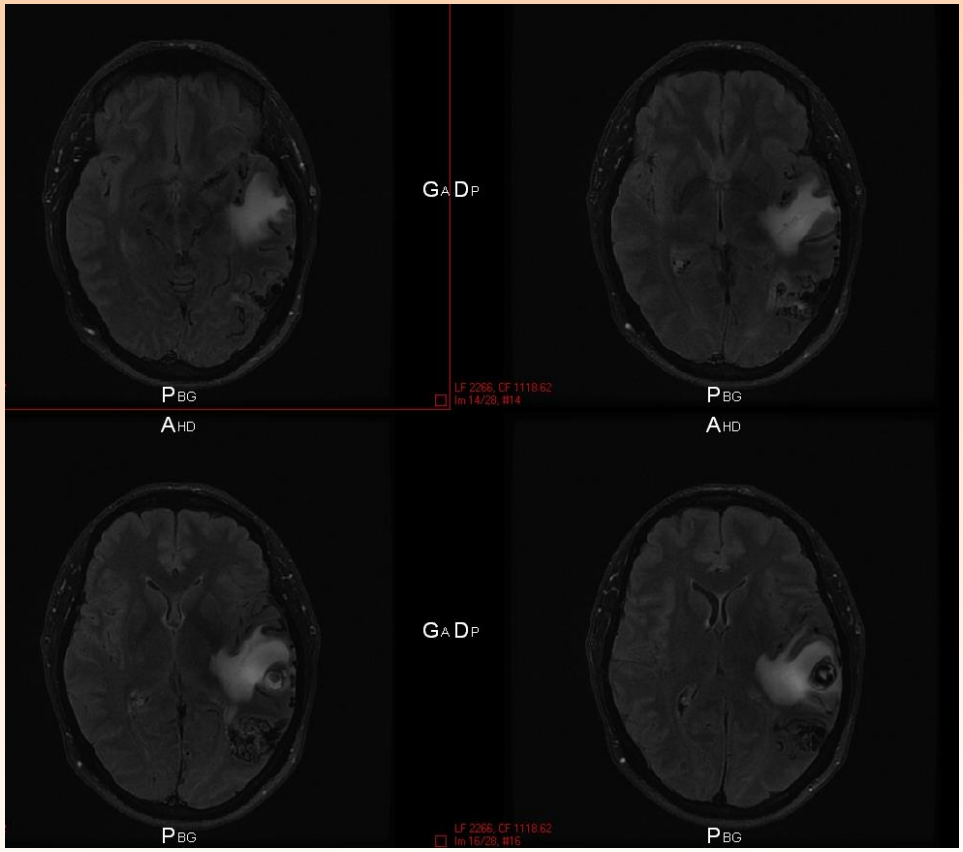




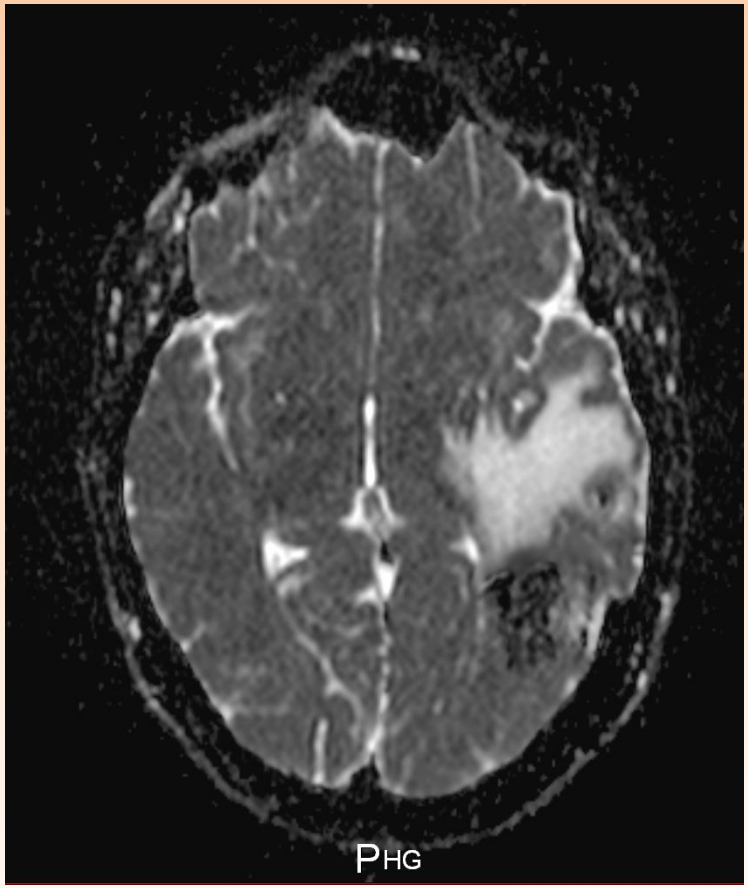
Evolution fatale a J3
Pas de bilan IRM ou TDM
Occlusion MAV ???

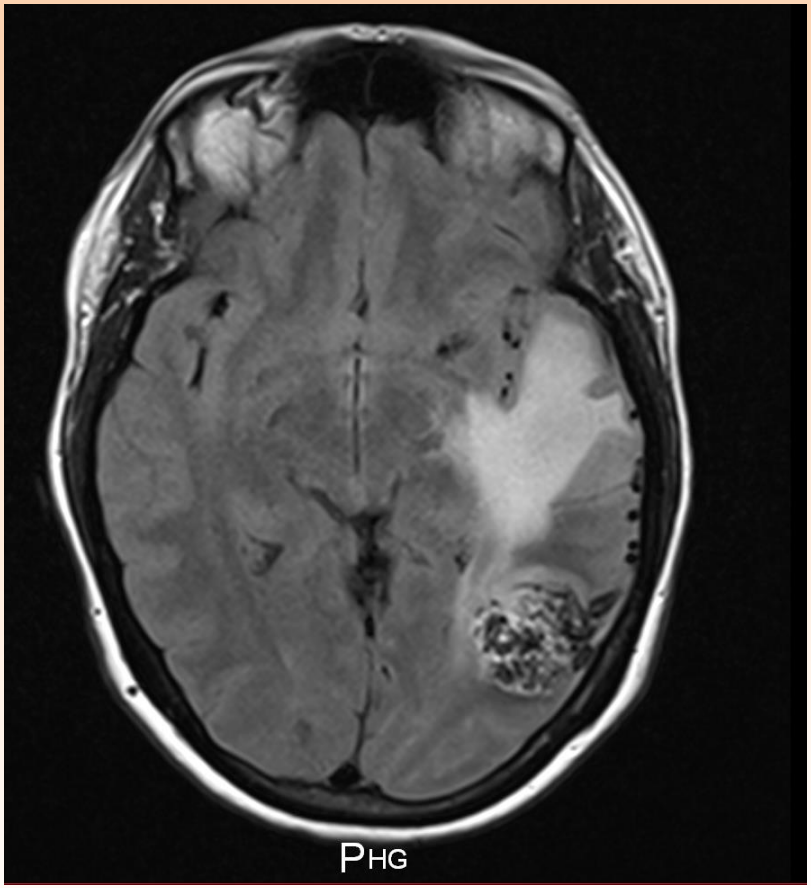
Cas 3 Thrombophlebite corticale non hémorragique

- Femme 53 ans
- Confusion aigue
- Troubles phasiques



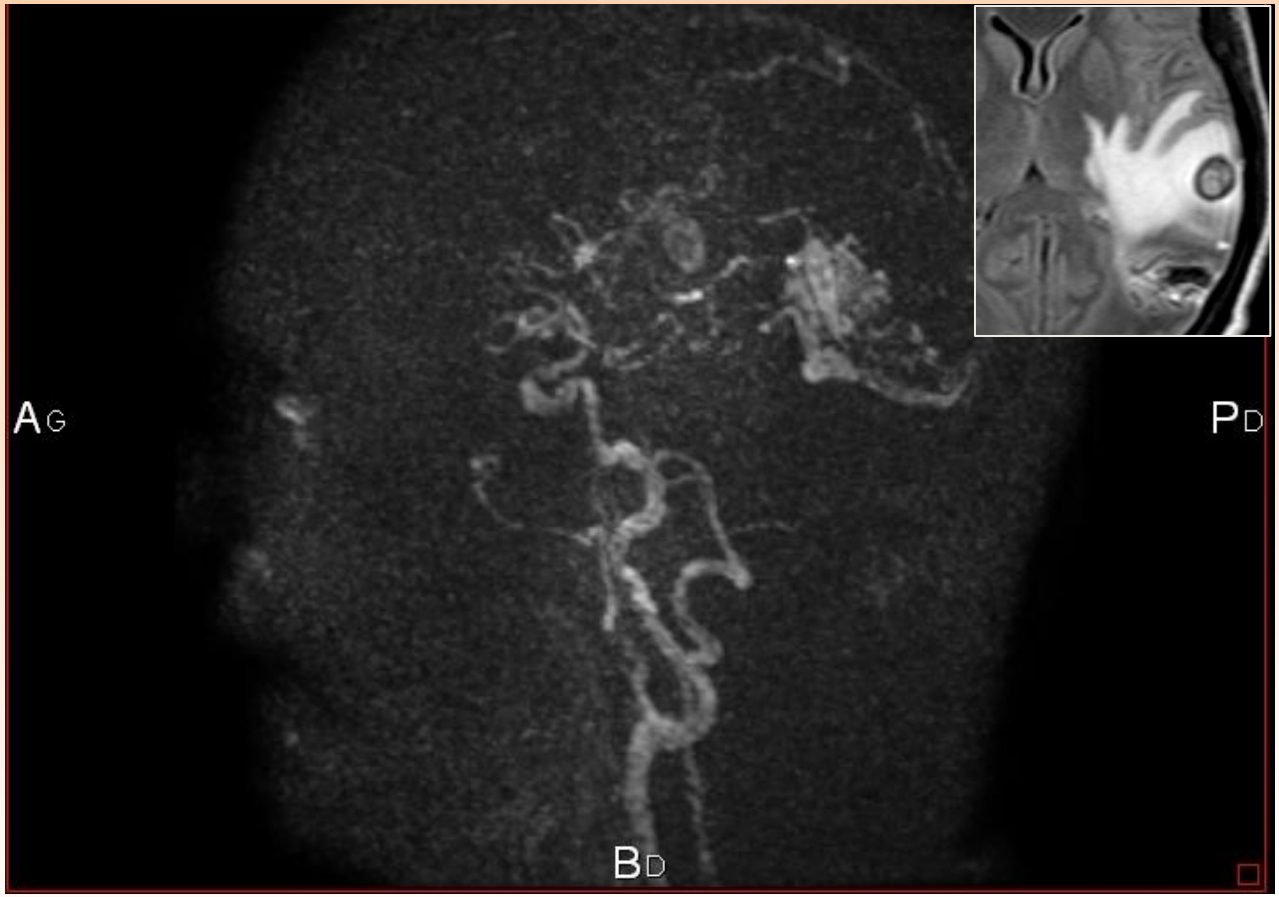
Cas 3 Thrombophlebite corticale non hémorragique



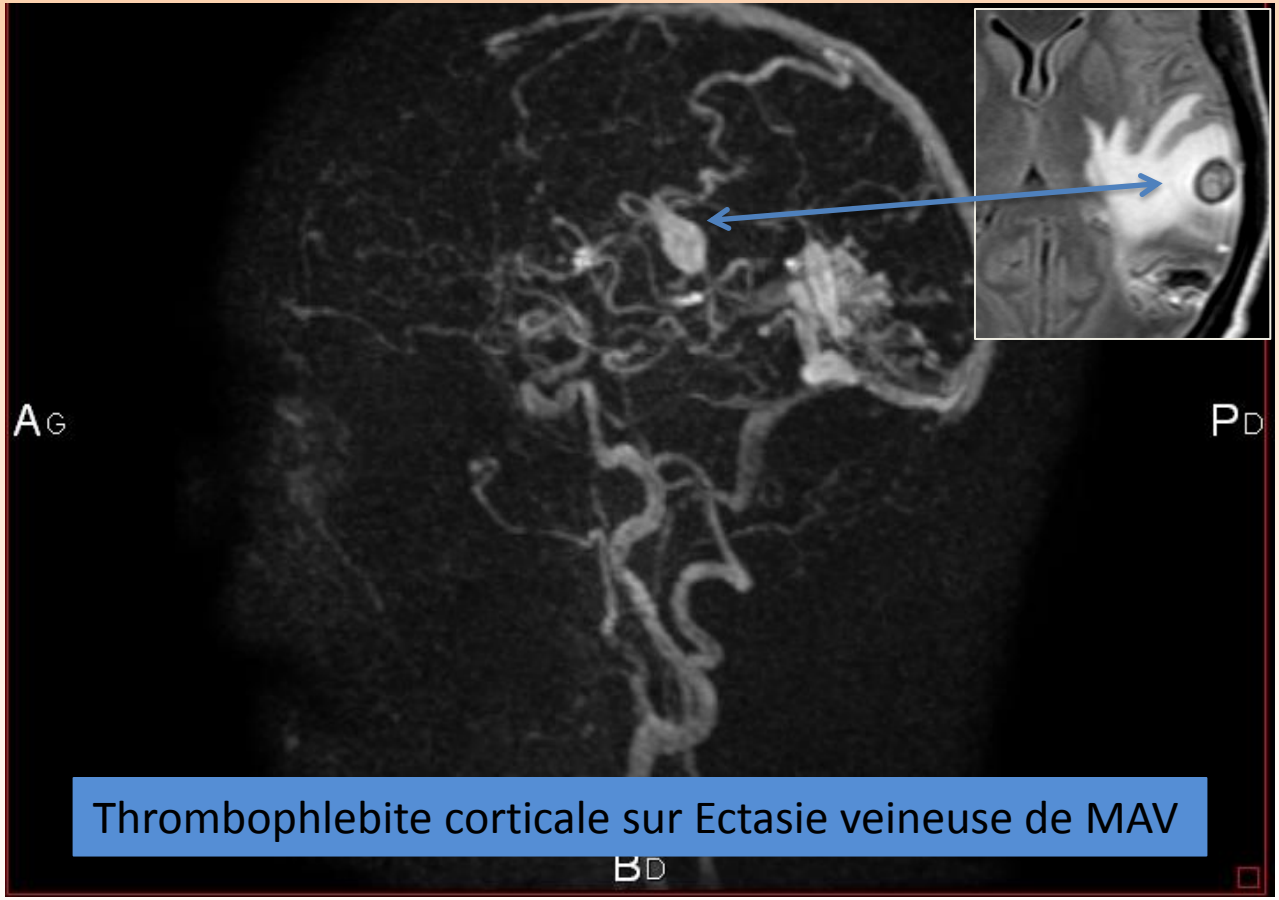


ADC augmenté

Cas 3 Thrombophlebite corticale non hémorragique

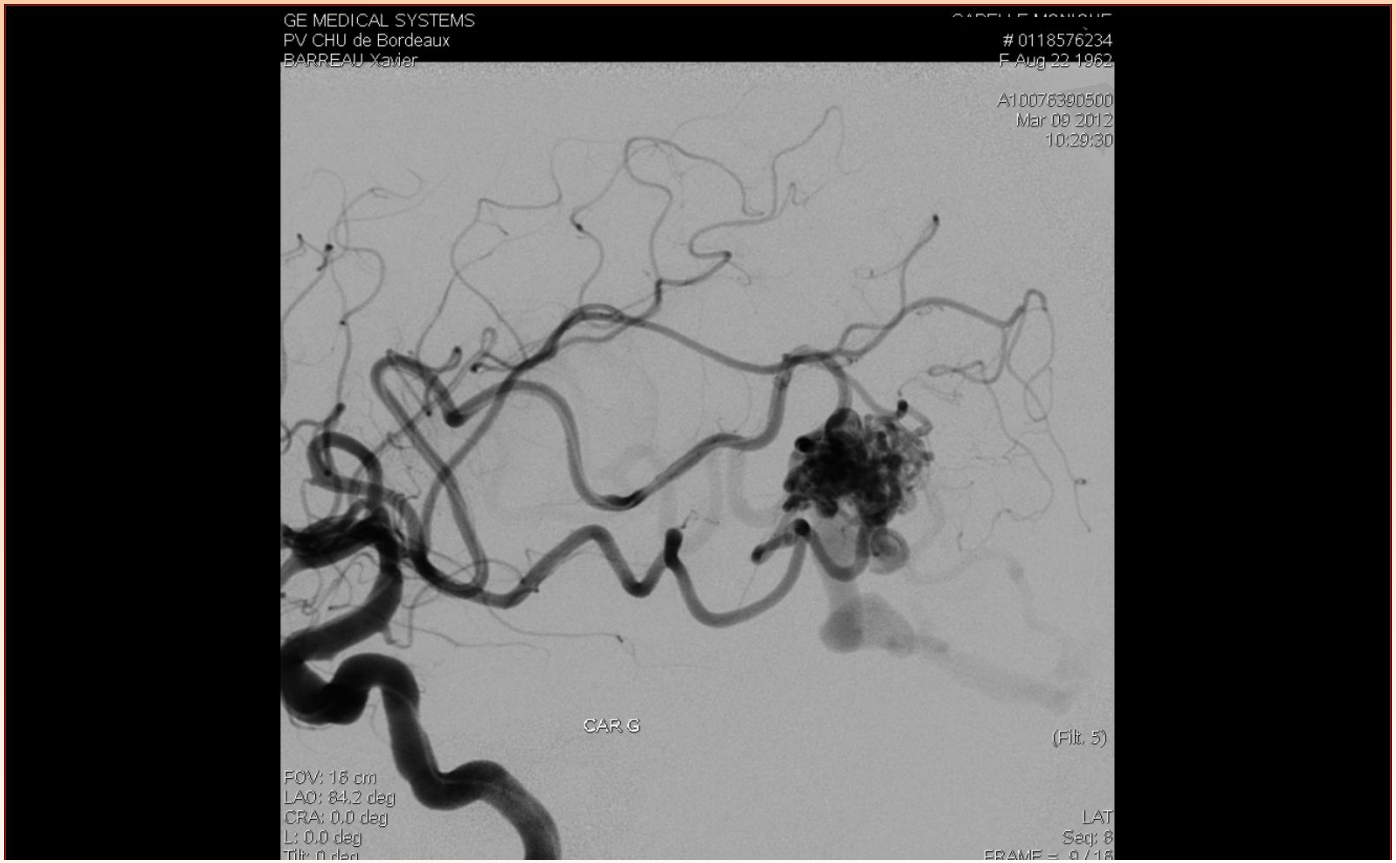


Cas 3 Thrombophlebite corticale non hémorragique



Thrombophlebite corticale sur Ectasie veineuse de MAV

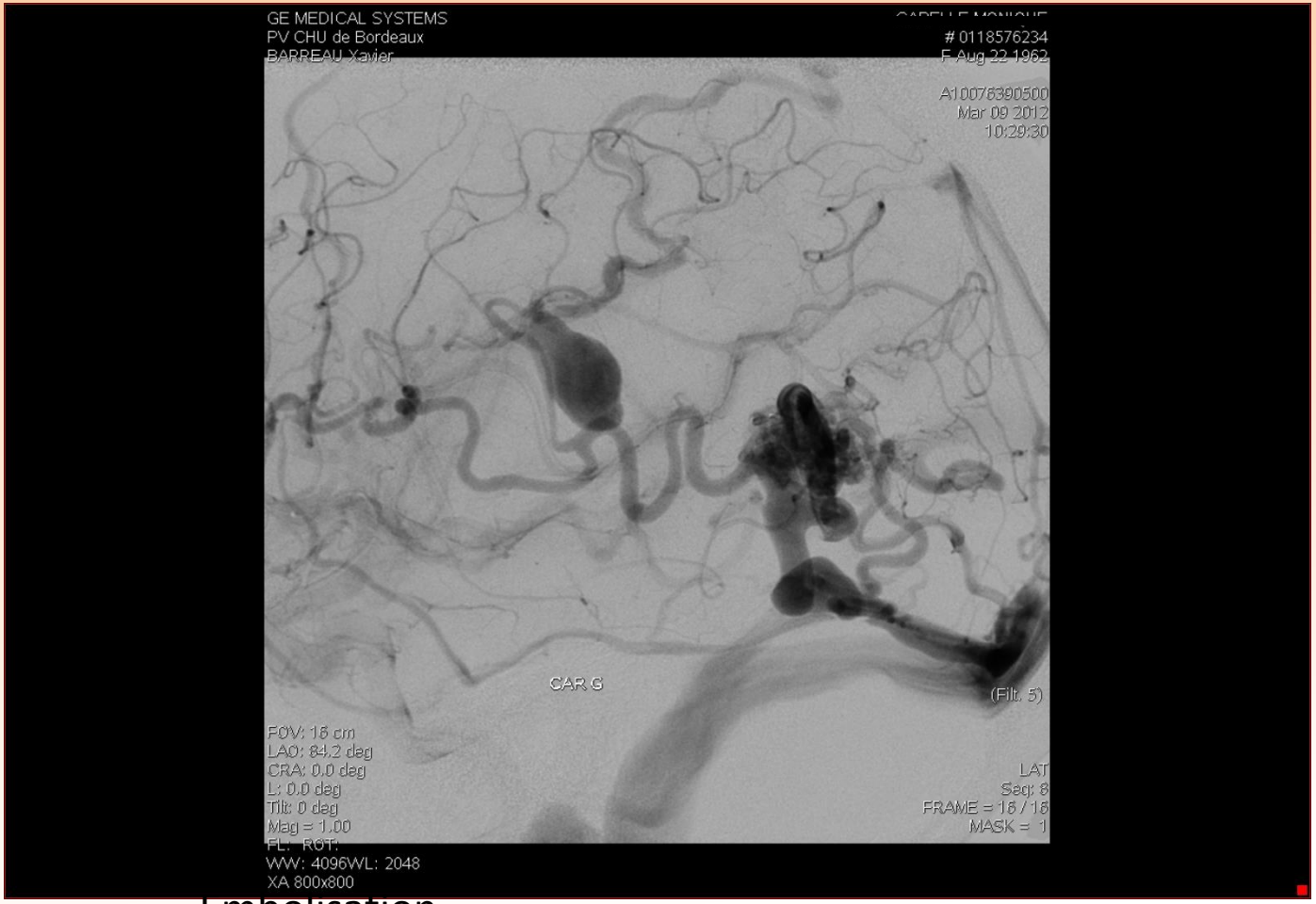
Cas 3 Thrombophlebite corticale non hémorragique



Embolisation

But: reduire débit veineux / limiter stress parietal

Cas 3 Thrombophlebite corticale non hémorragique



Embolisation

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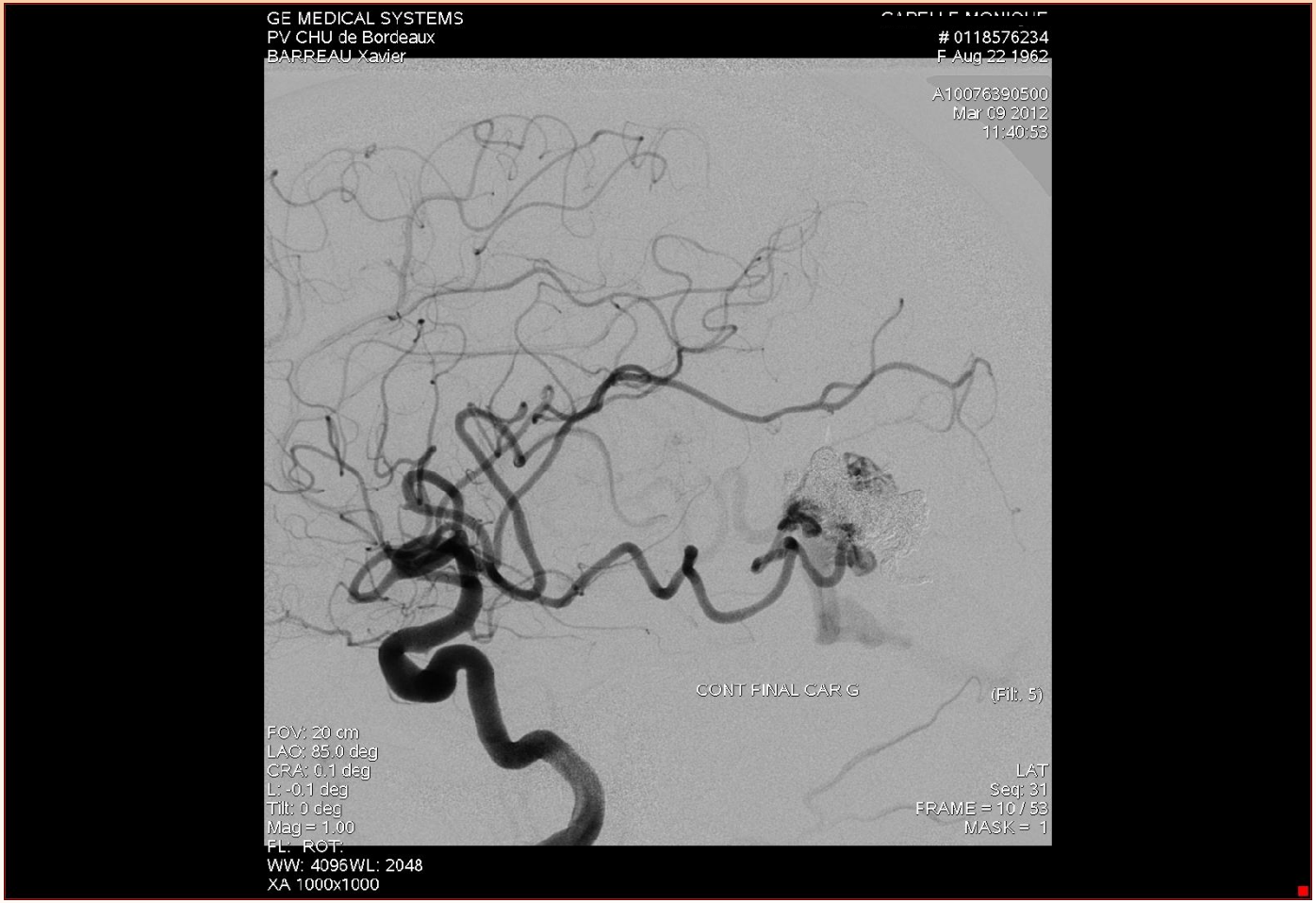
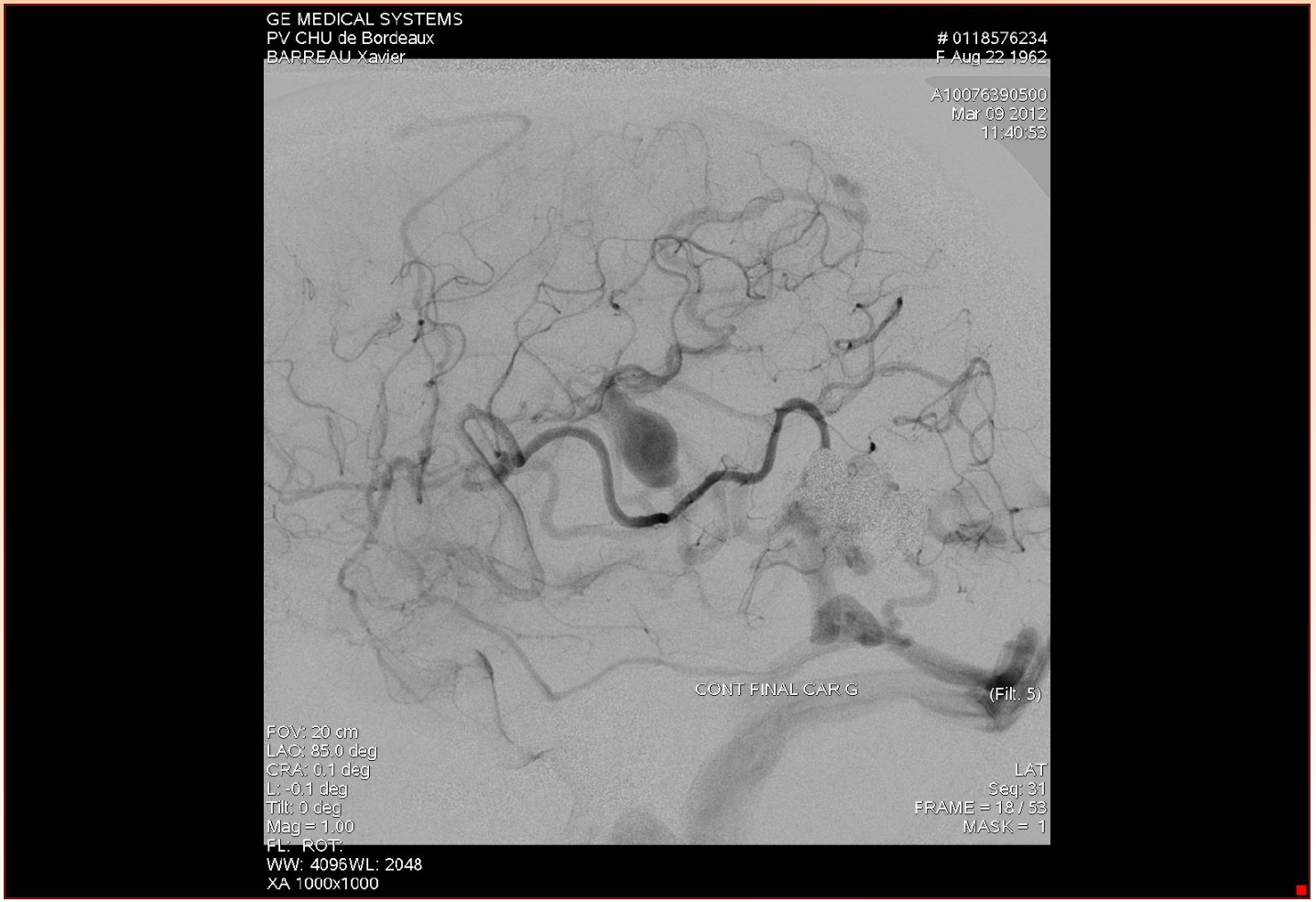
Cas 3 Thrombophlebite corticale non hémorragique



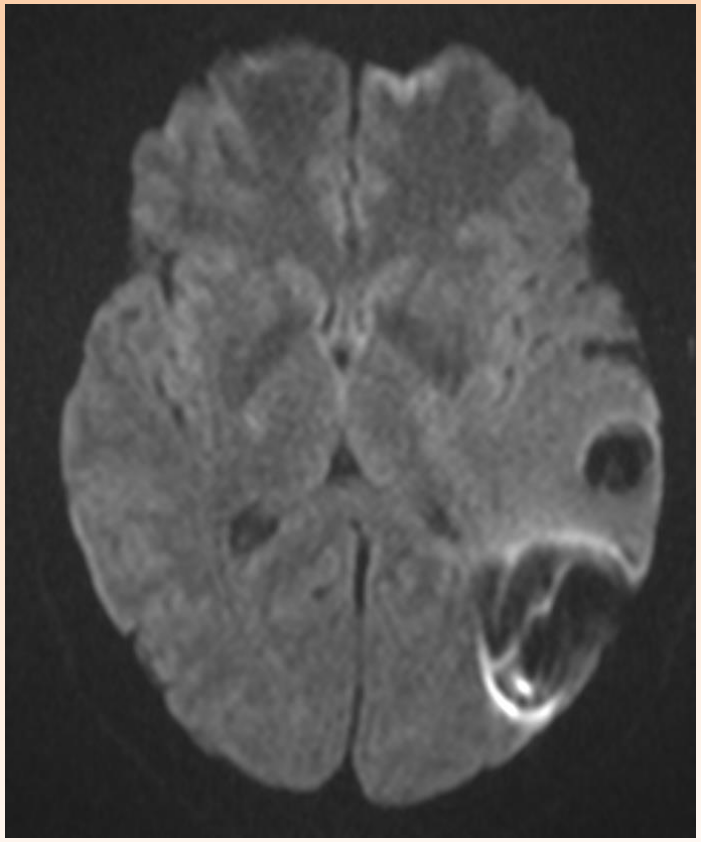
Embol 50 % - Anticoagulation efficace pendant 5 jours

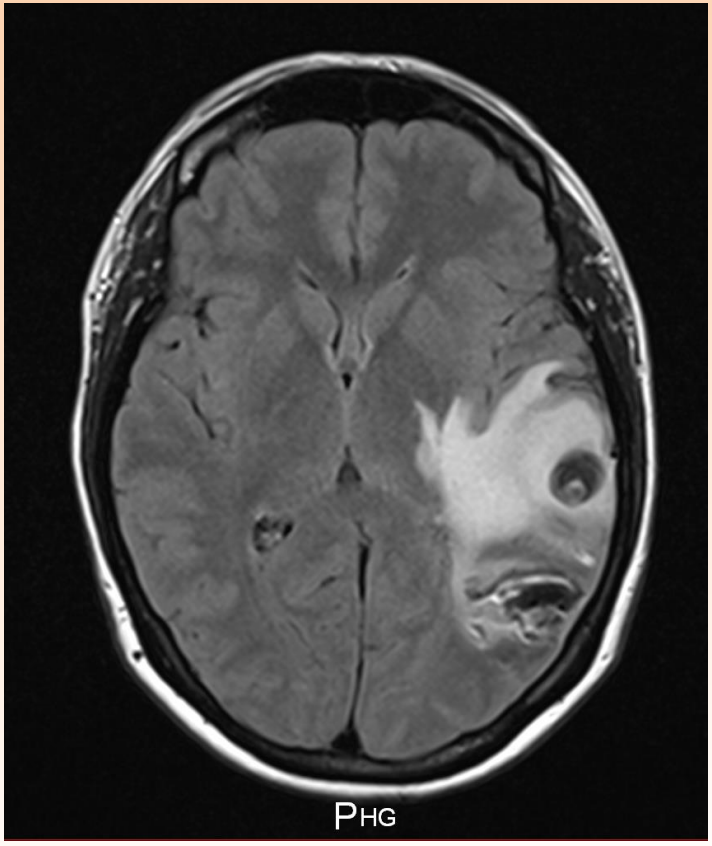
Cas 3 Thrombophlebite corticale non hémorragique



Embol 50 % - Anticoagulation efficace pendant 5 jours

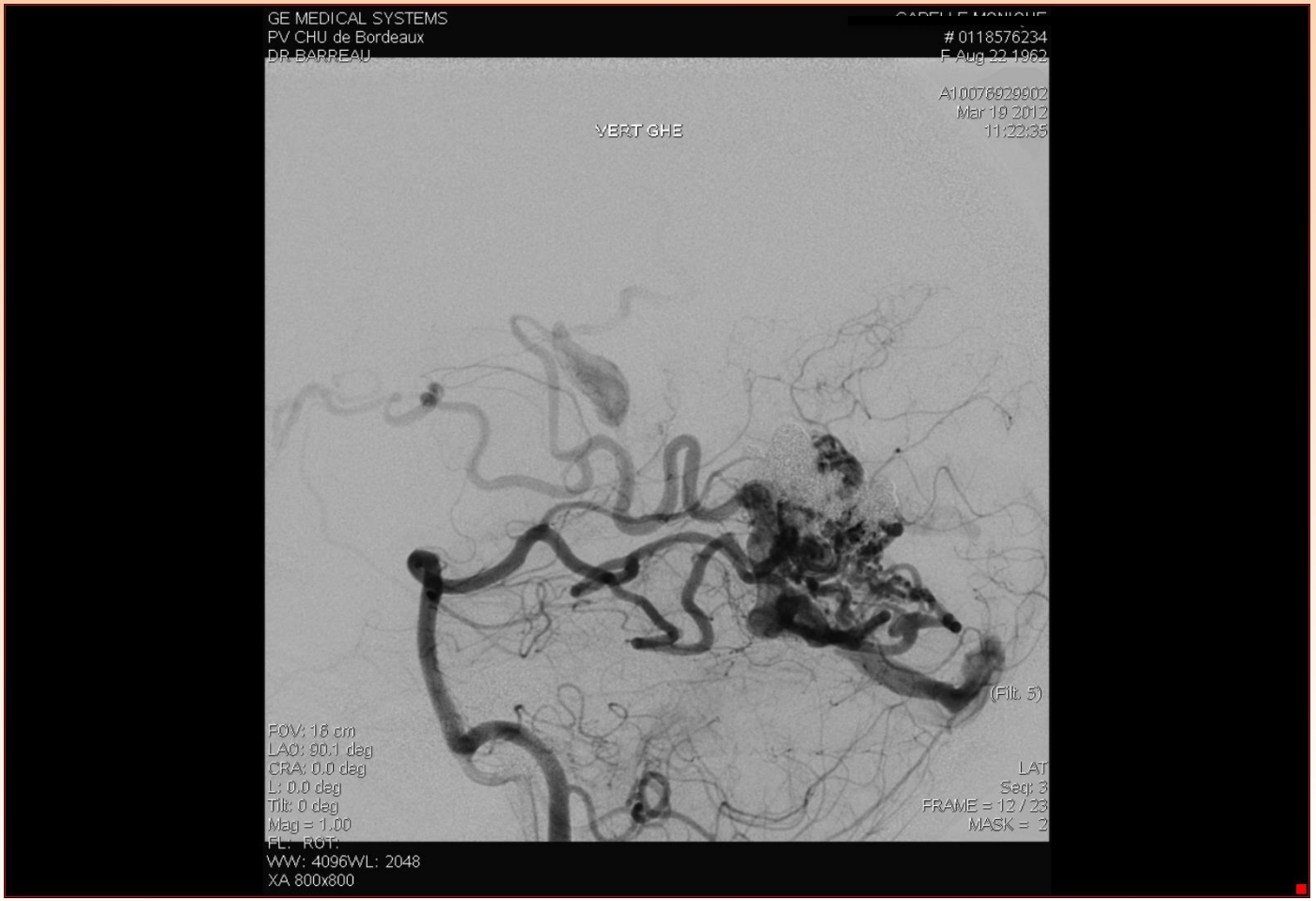
Cas 3 Thrombophlebite corticale non hémorragique





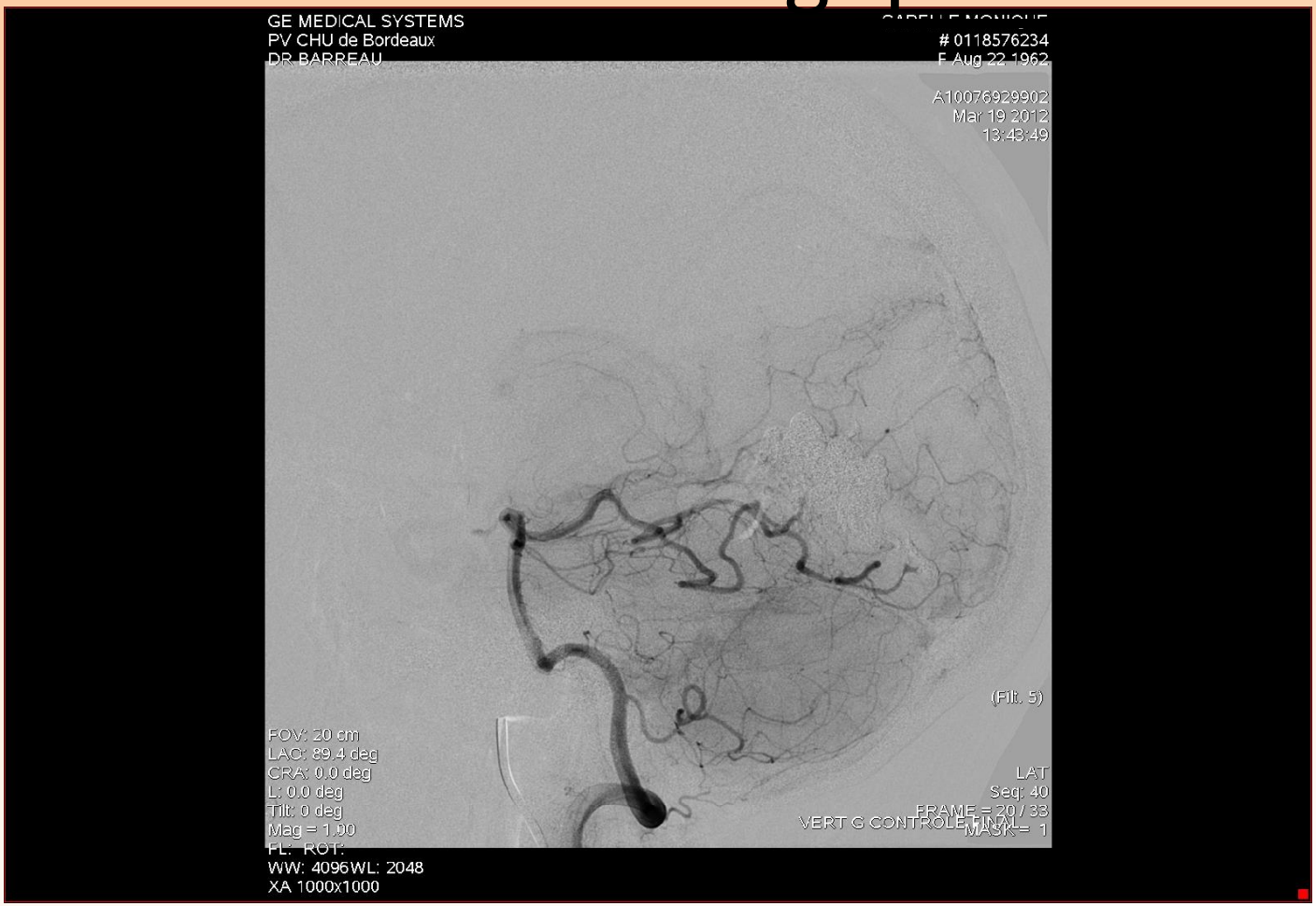
IRM J5, amelioration clinique, qq cépalées.

Cas 3 Thrombophlebite corticale non hémorragique



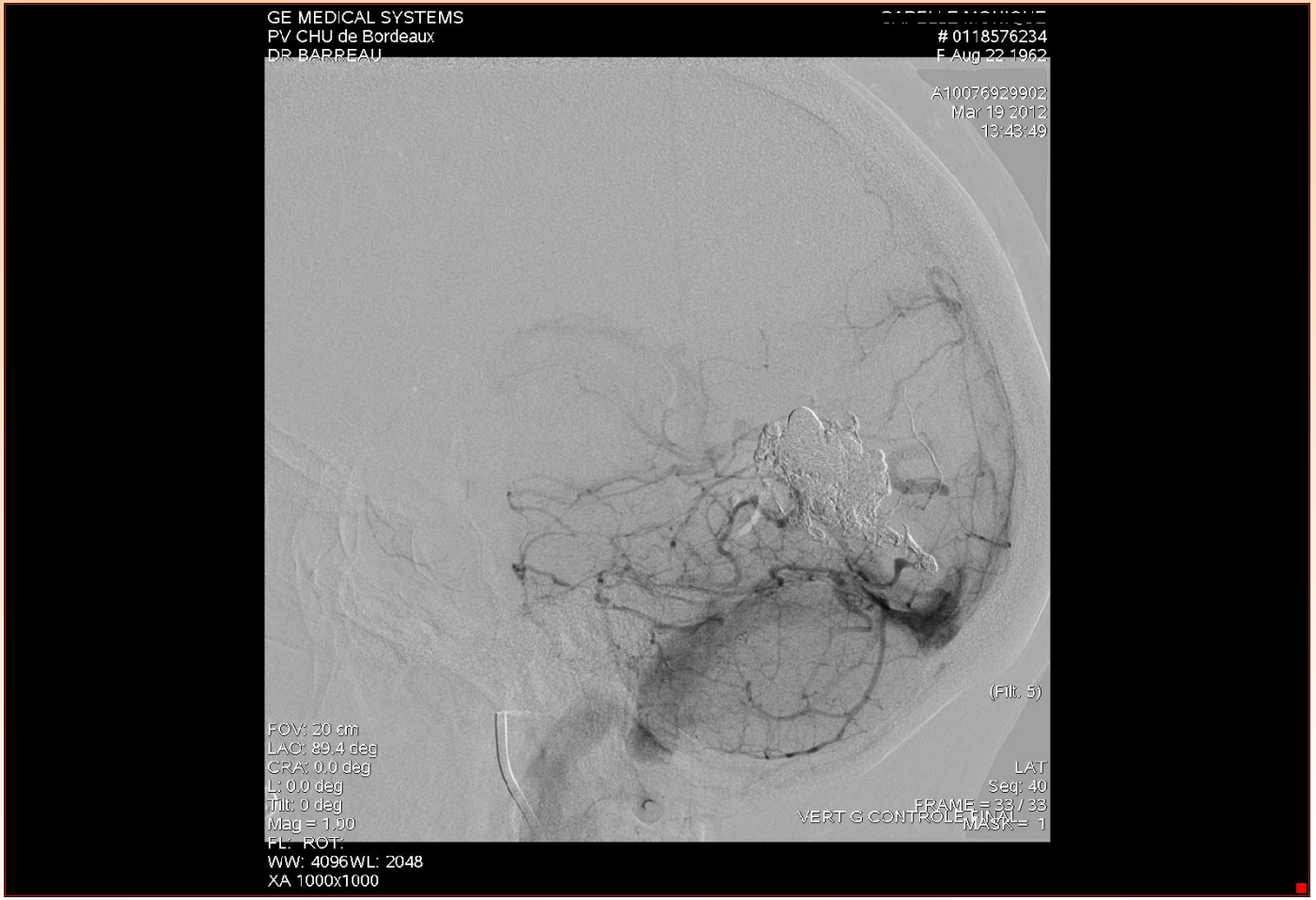
Embol 2 à J 10, régression ectasie veineuse

Cas 3 Thrombophlebite corticale non hémorragique



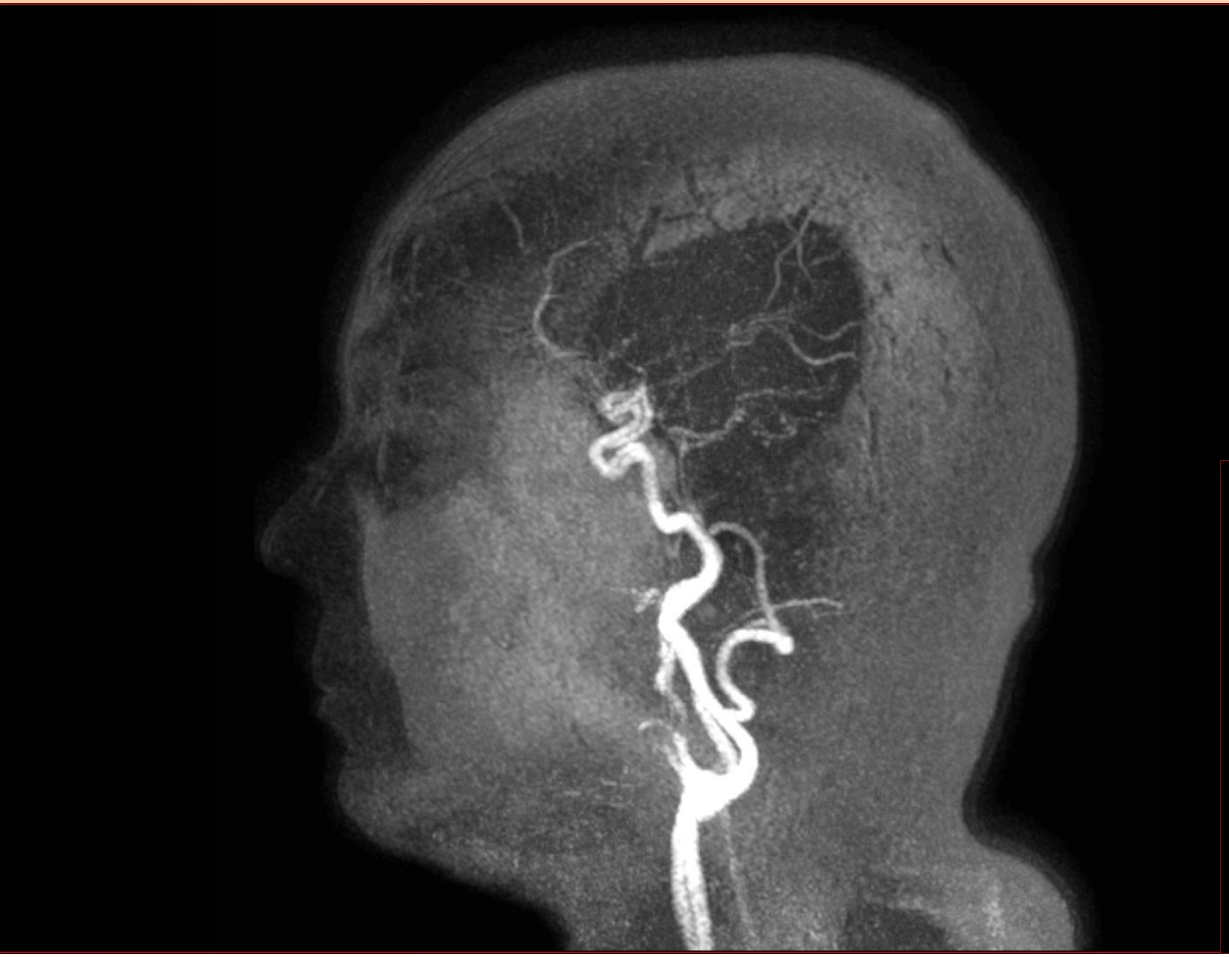
Embol 2 à J 10, régression ectasie veineuse

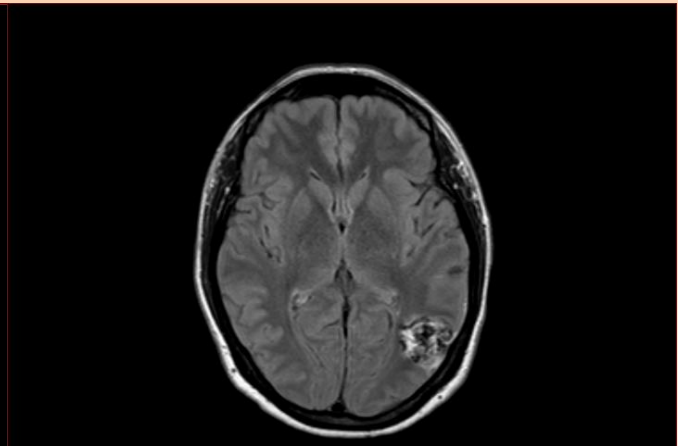
Cas 3 Thrombophlebite corticale non hémorragique

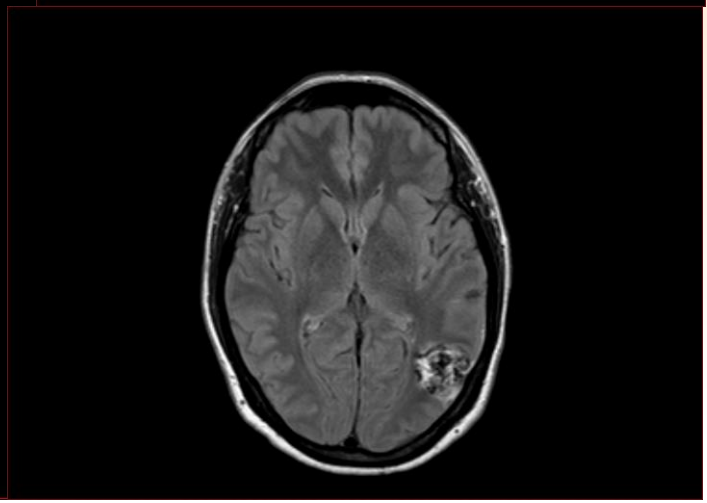


Embol 2 à J 10, Occlusion MAV

Cas 3 Thrombophlebite corticale non hémorragique

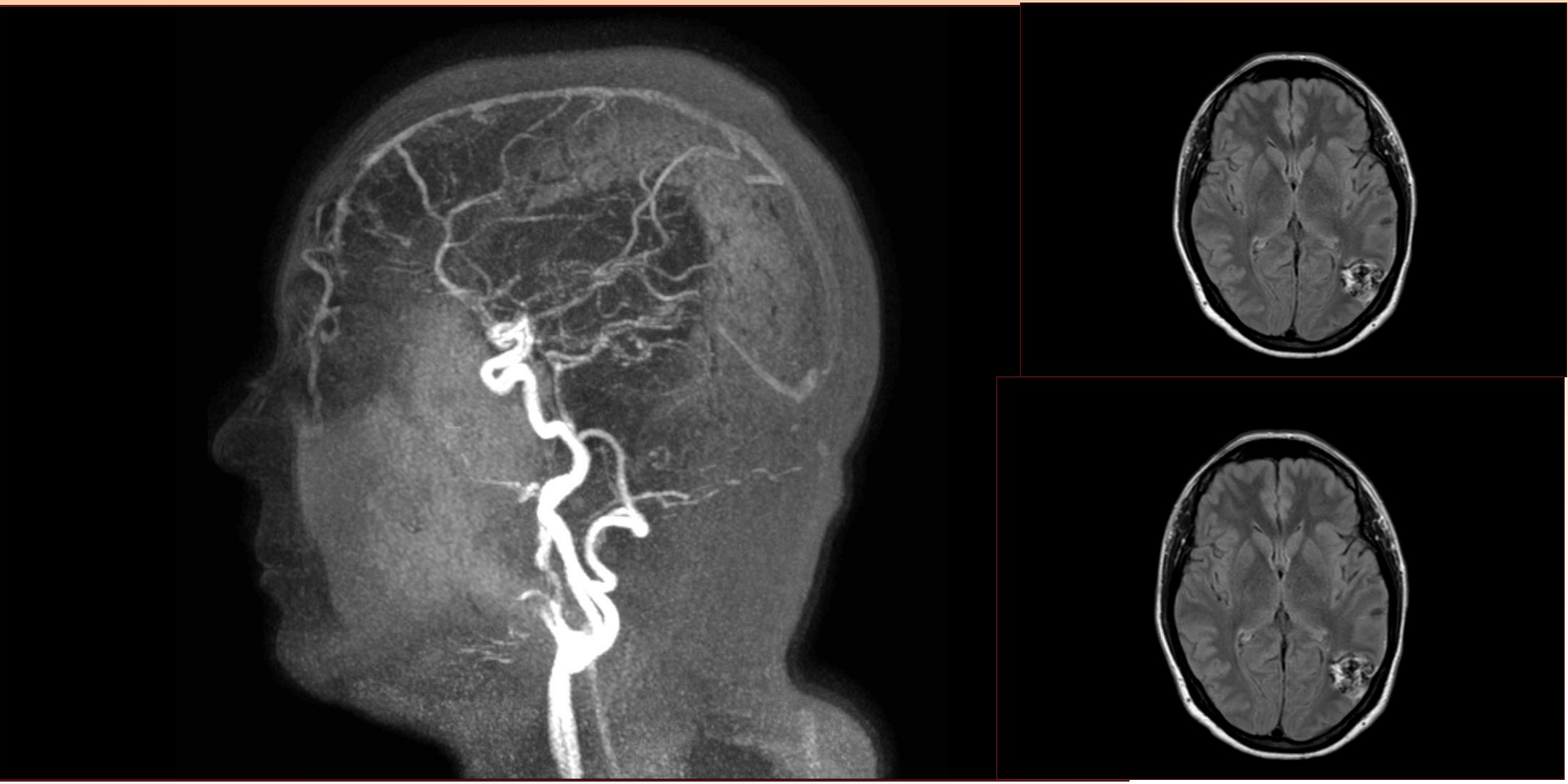






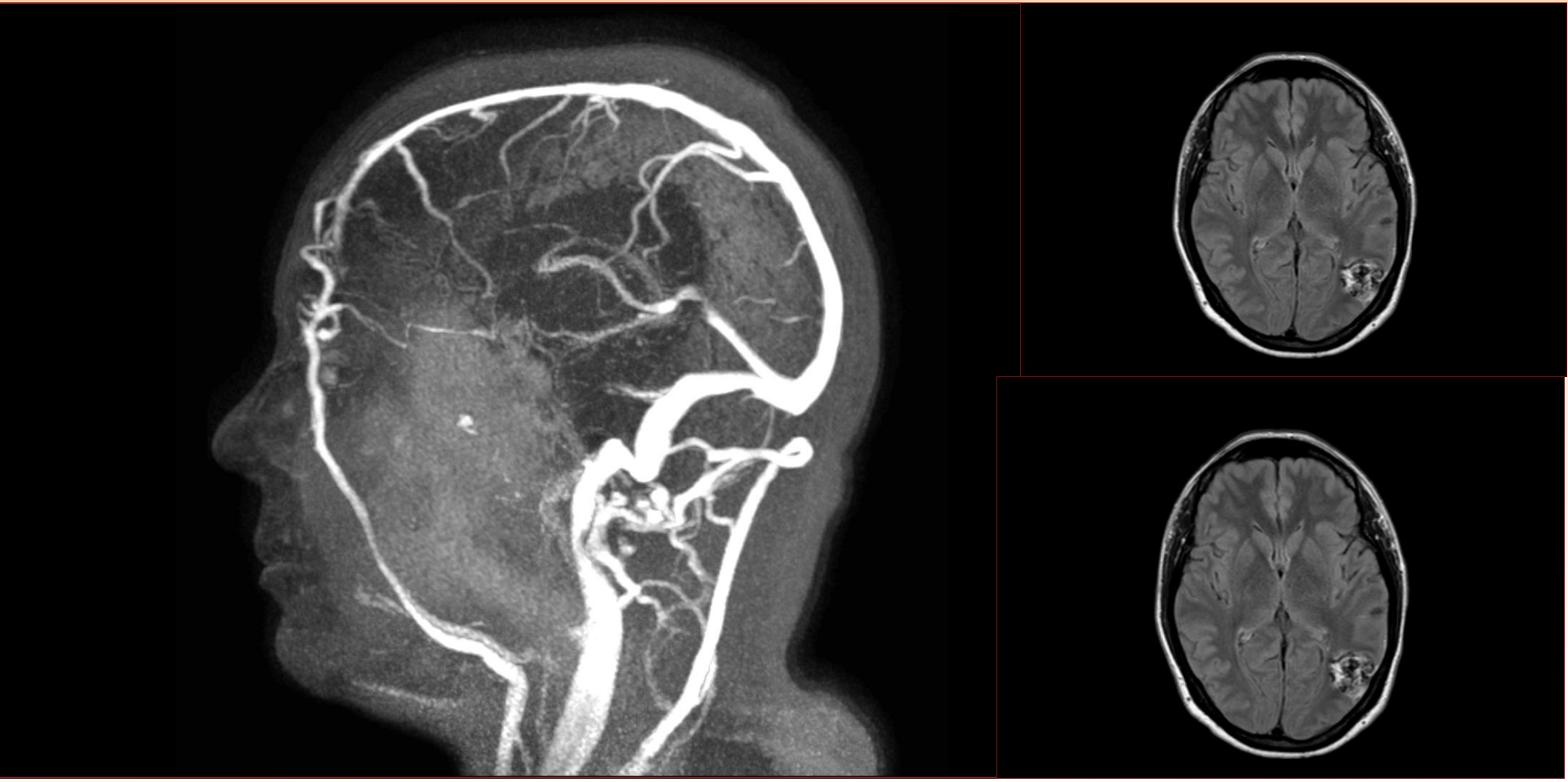
Contrôle M3

Cas 3 Thrombophlebite corticale non hémorragique



Contrôle M3

Cas 3 Thrombophlebite corticale non hémorragique

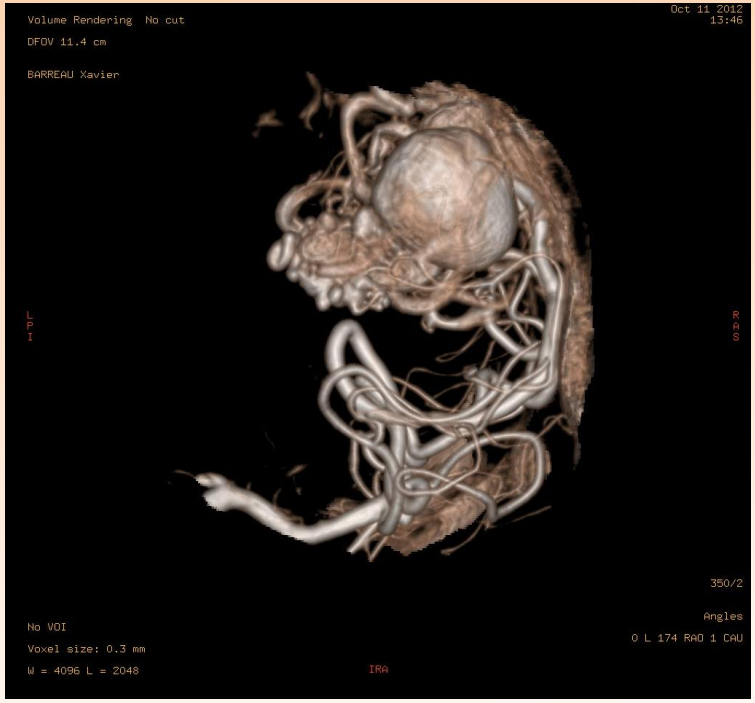


Contrôle M3

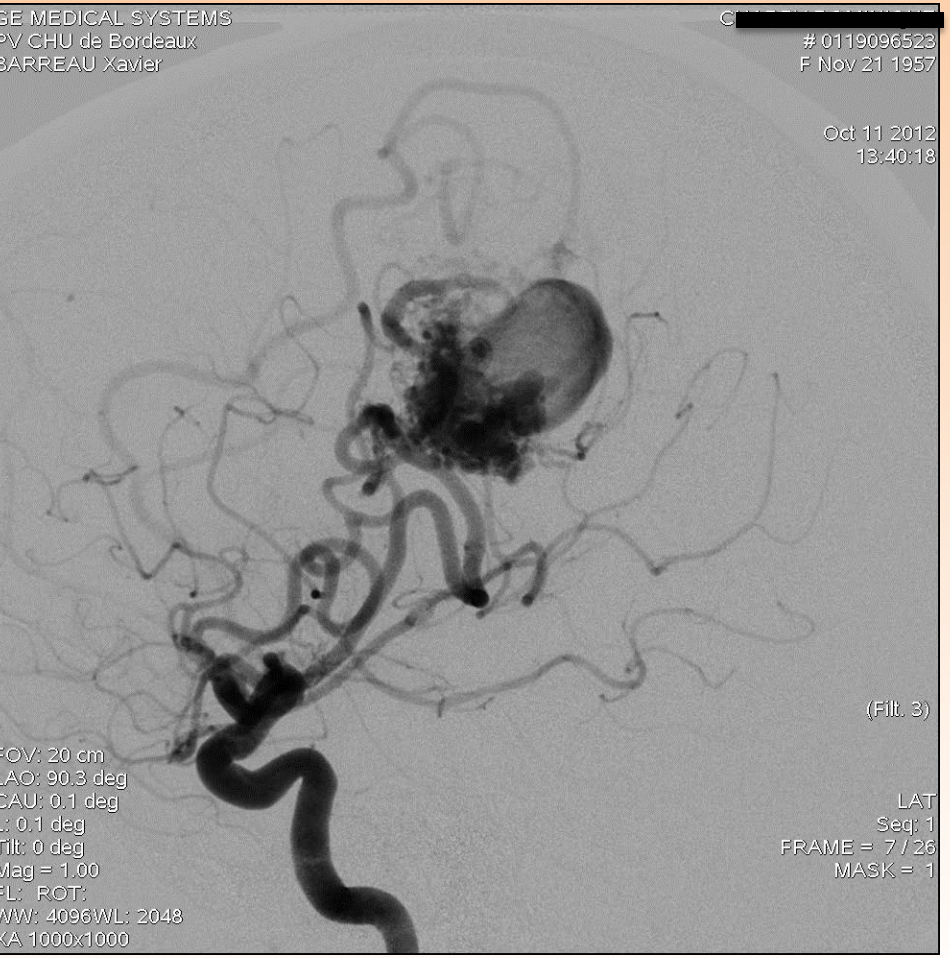
Cas 4 Thrombophlebite post embolisation

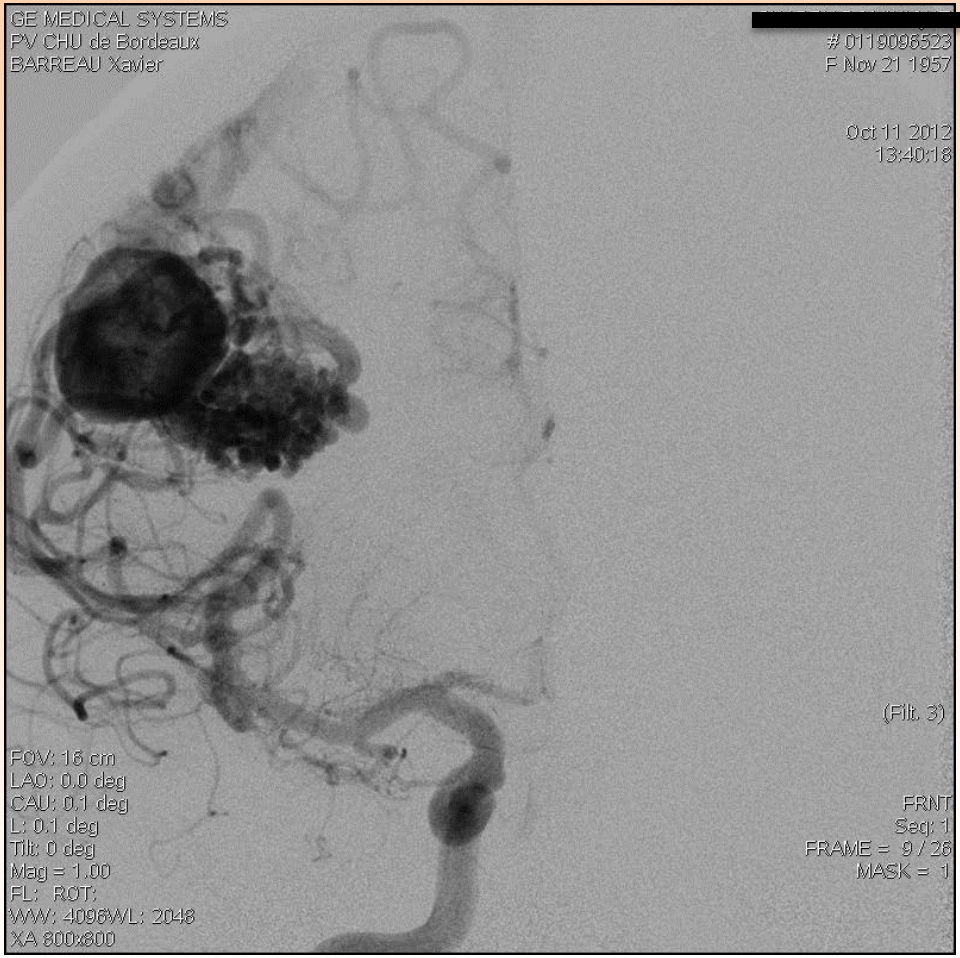
Patient 53 ans
Deficit transitoire MSG
ATCD de petit mal enfance

MAV corticale avec ectasie veineuse++

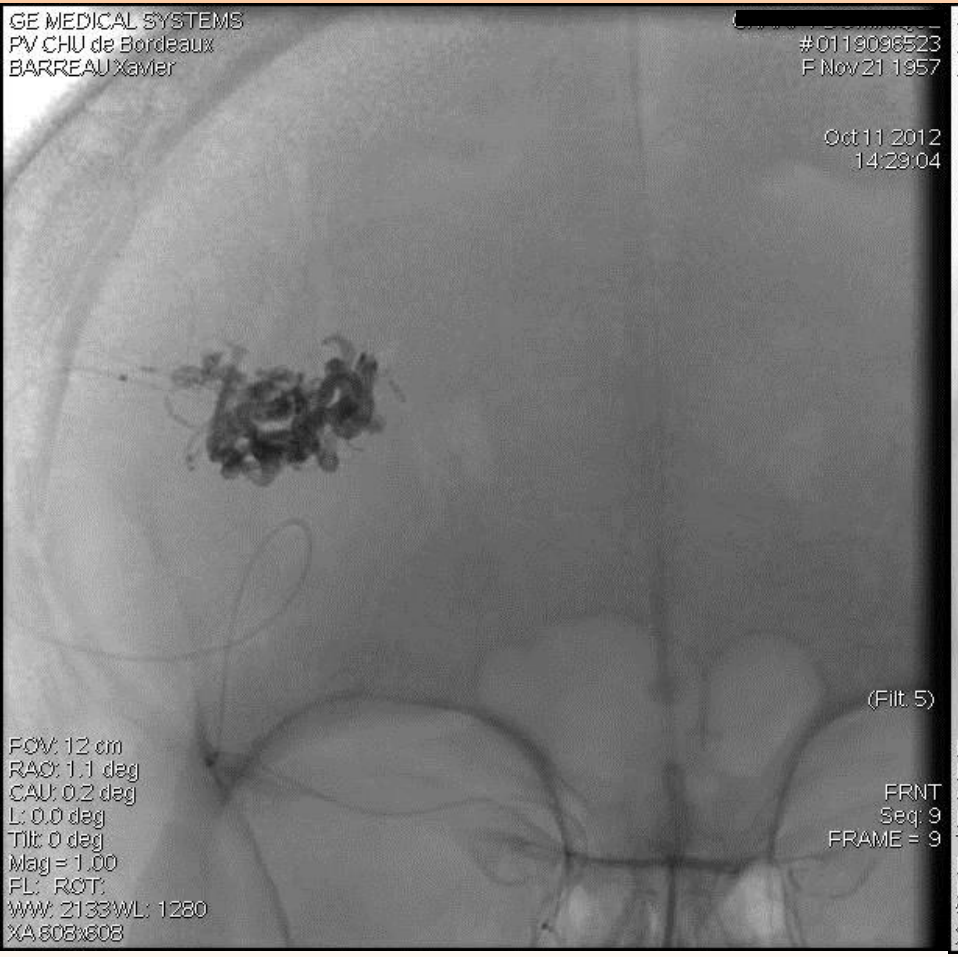


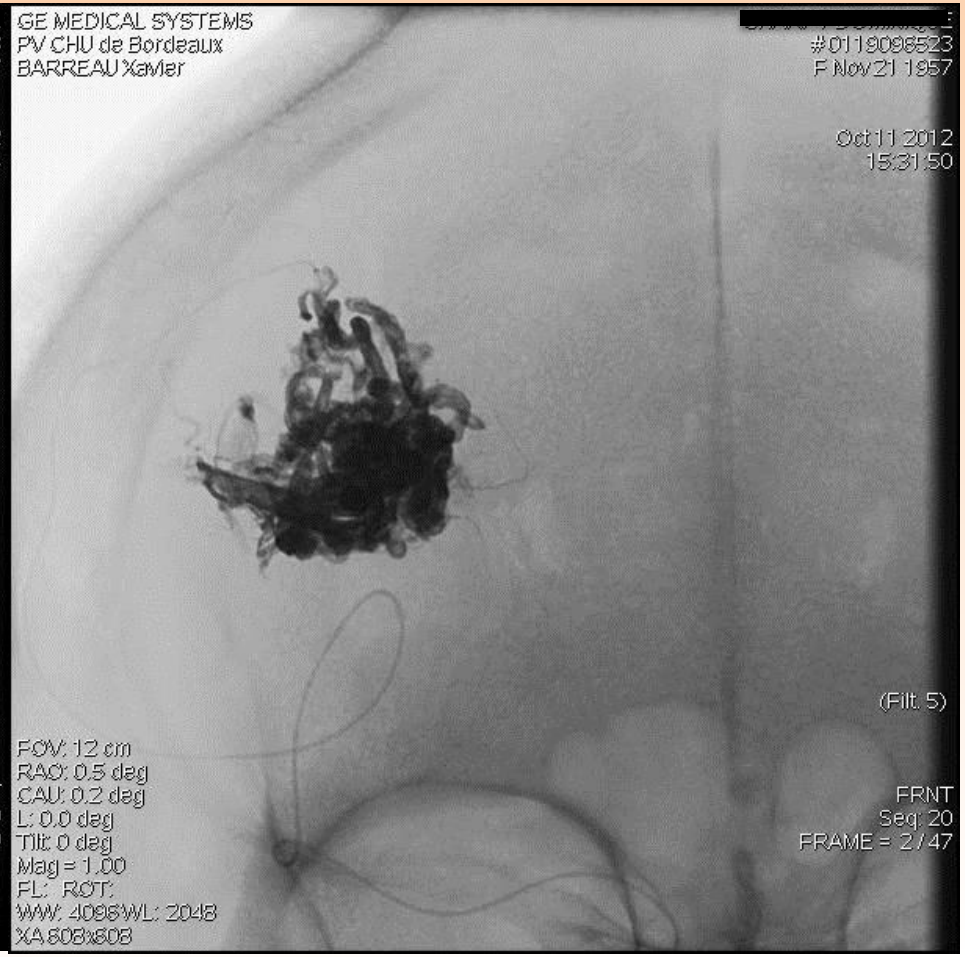
Cas 4 Thrombophlebite post embolisation



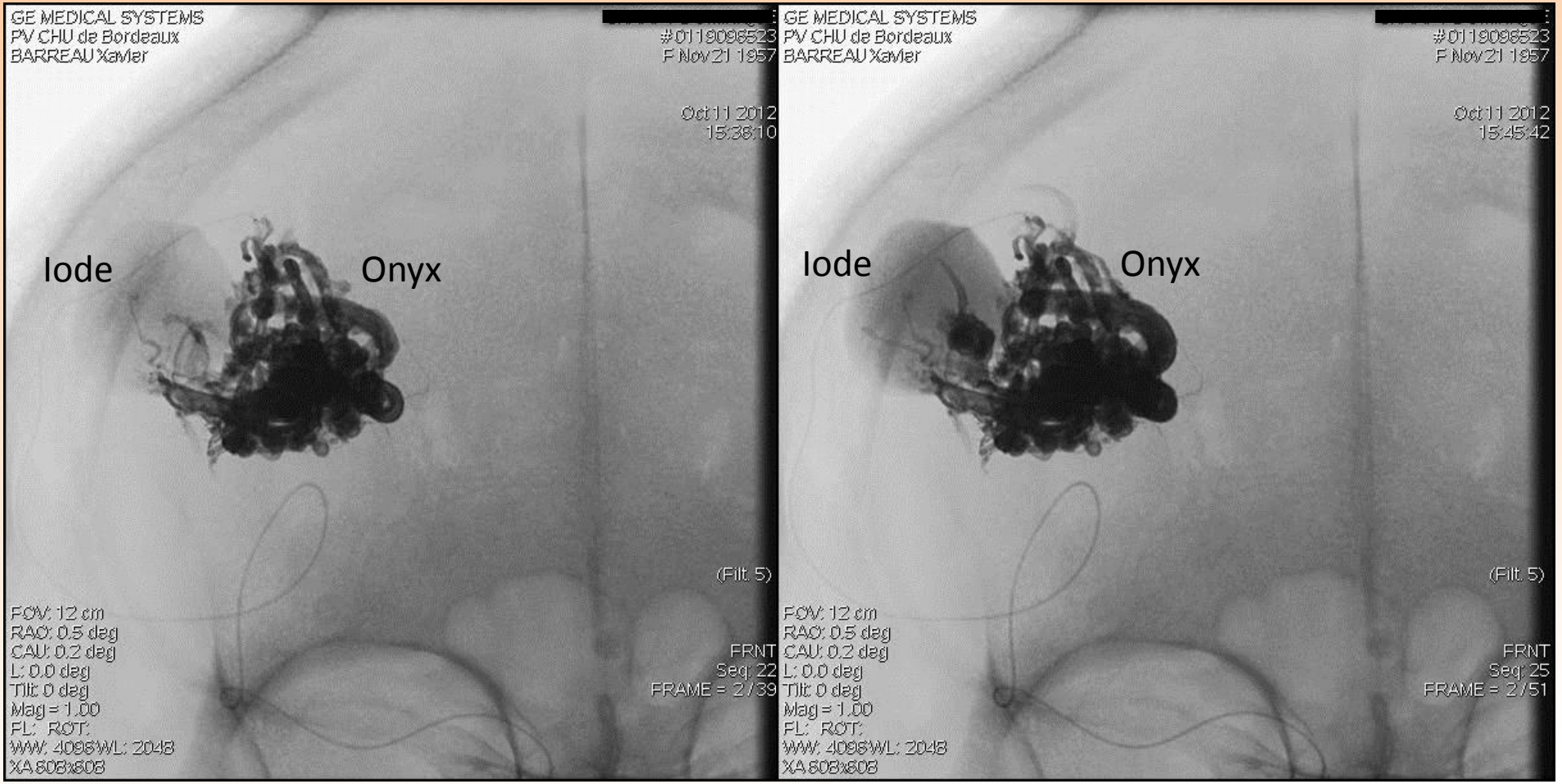


Cas 4 Thrombophlebite post embolisation



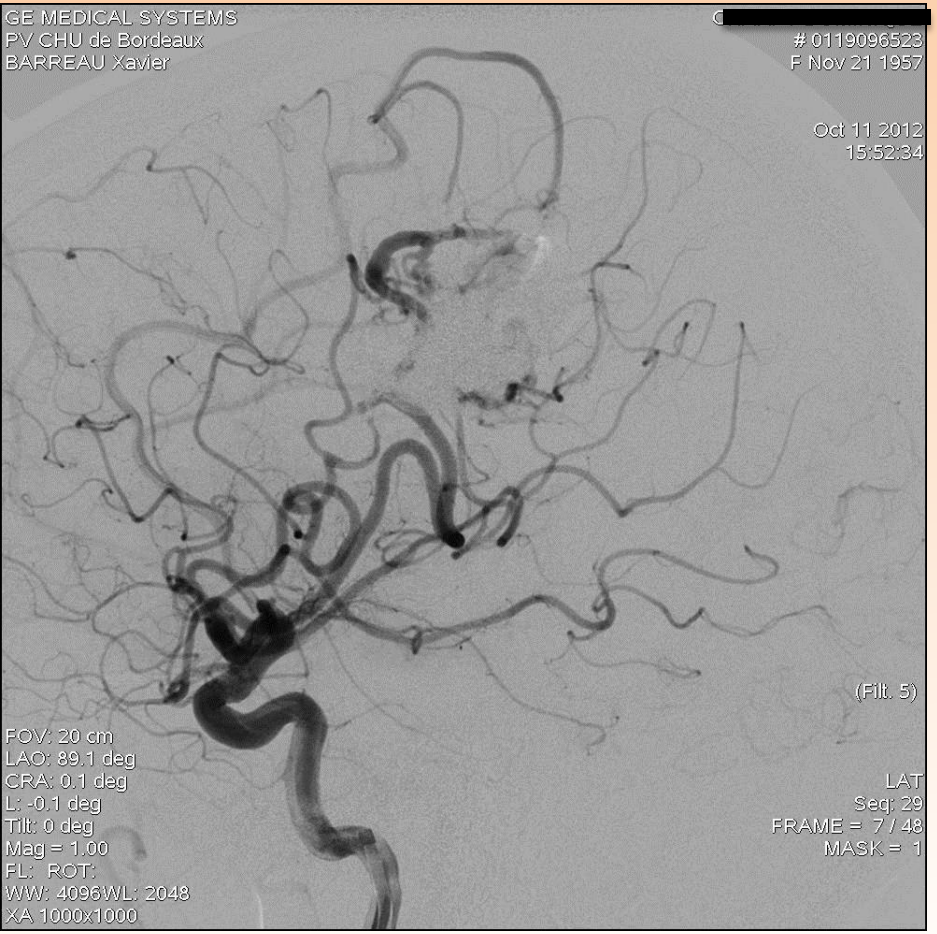


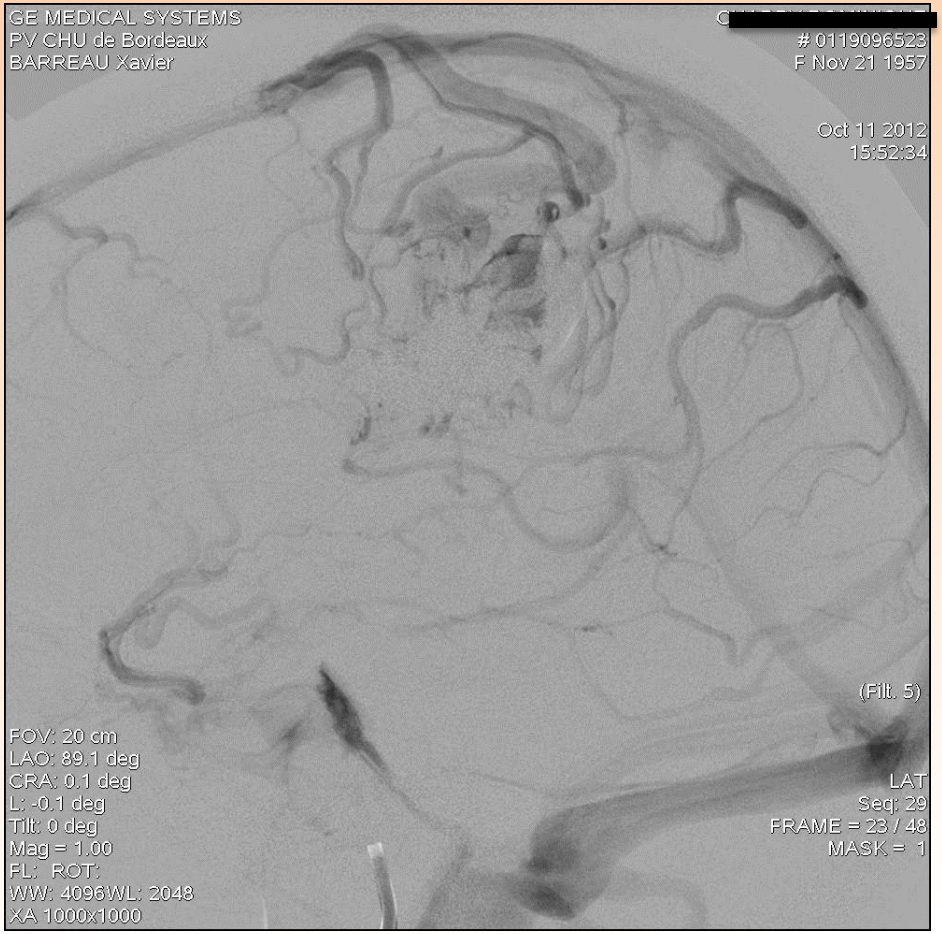
Cas 4 Thrombophlebite post embolisation



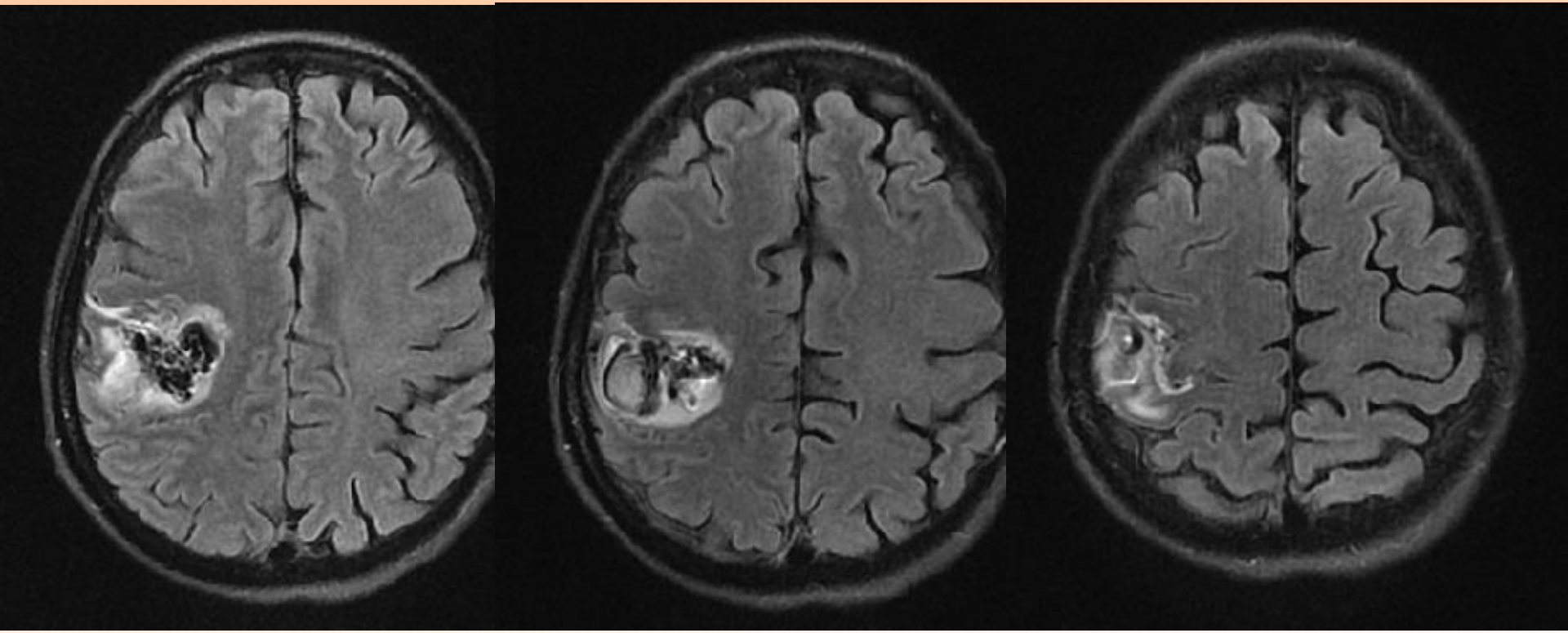
Stagnation post embolisation….

Cas 4 Thrombophlebite post embolisation



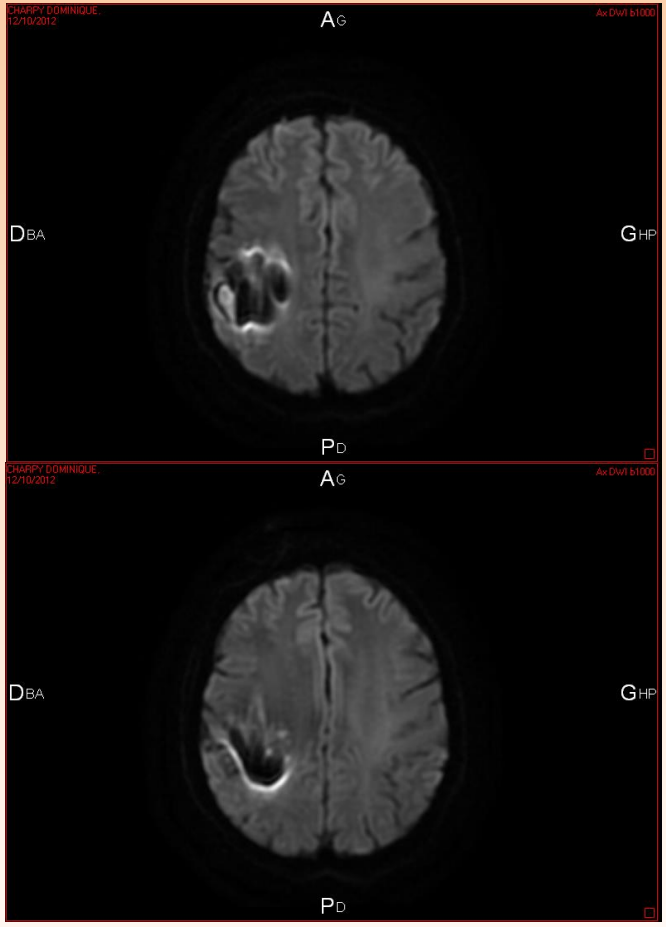


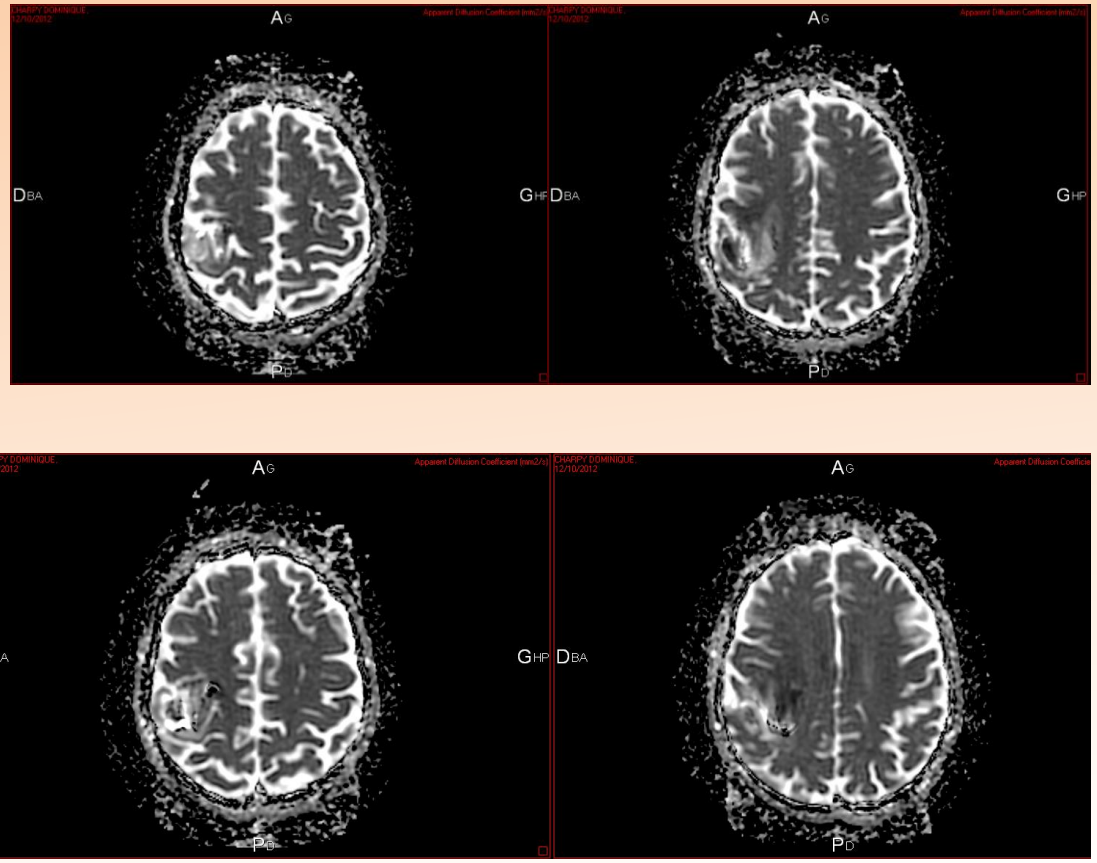
Cas 4 Thrombophlebite post embolisation



IRM J1 post embol
Deficit MSG 2/5

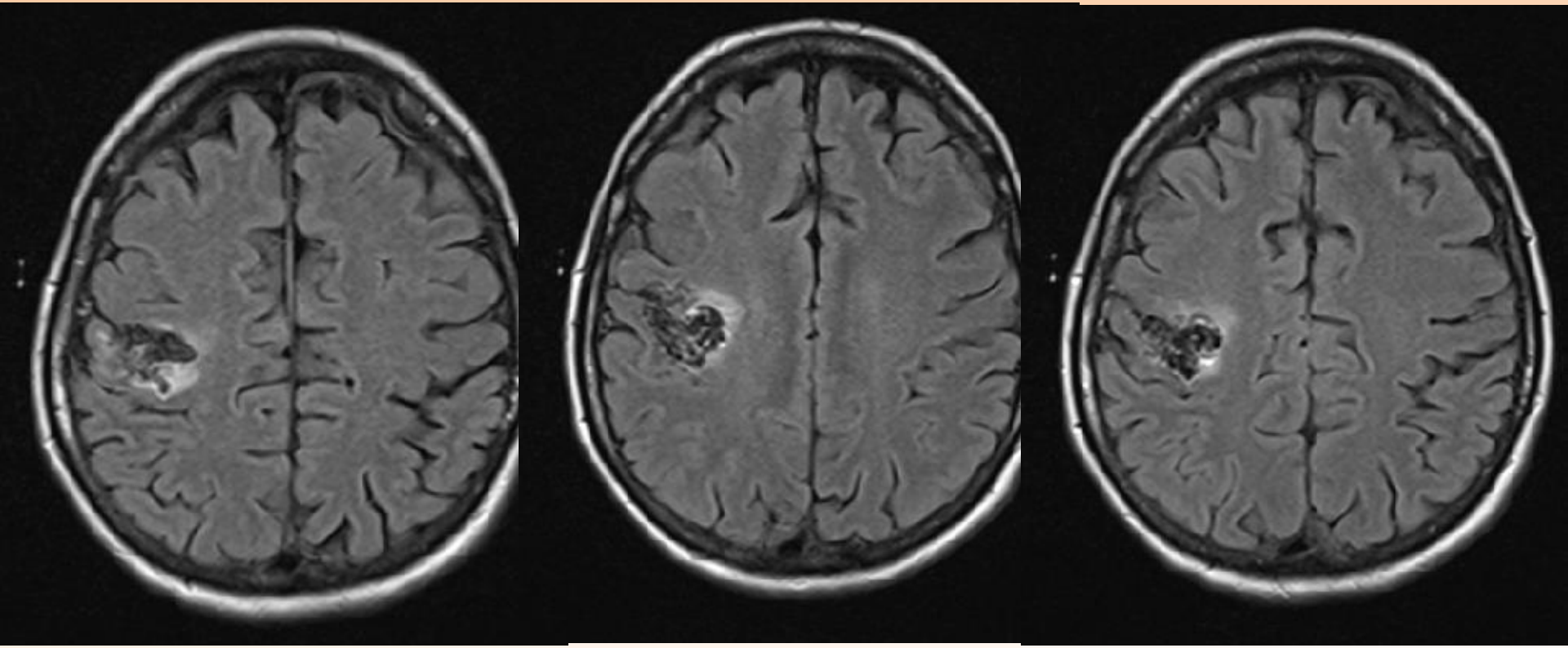
Cas 4 Thrombophlebite post embolisation





IRM J1 post embol
Deficit MSG 2/5

Cas 4 Thrombophlebite post embolisation



IRM M3 post embol
Recup total (dés J30)

DISCUSSION

- Thrombophlebite peut faire partie integrante du proccessus evolutif des MAV
- Littérature :
  - Très rare données sur thrombose veineuse.
  - Seulement sous l'angle regression spontanée des MAV (1,5 %) ou évolution post RTT.

DISCUSSION

The Relationship between Occlusive Hyperemia and Complications Associated with the Radiosurgical Treatment of Arteriovenous Malformations: Report of Two Cases

OBJECTIVE AND IMPORTANCE: It has been suggested that impaired venous drainage of normal brain after surgical removal of an arteriovenous malformation (AVM) may cause perinidal edema and hemorrhage. The term *occlusive hyperemia* has been proposed for this phenomenon. There is evidence that occlusive hyperemia also may occur after radiosurgical treatment of AVMs. The purpose of this article is to lend further support to the concept that venous occlusion may be responsible for some complications observed after AVM radiosurgery.

CLINICAL PRESENTATION: We report two patients with unusual radiosurgery-associated complications, and we examine the evidence for venous occlusion as the mechanism underlying the observed clinical sequelae in each patient.

INTERVENTION: Patient 1 had a large parietal venous infarct remote from her frontal AVM site 11 months after radiosurgery. At that time, the AVM was confirmed by angiography to have been obliterated. During the next 4 years, the patient experienced persistent posterior hemispheric edema with recurrent focal hemorrhages until the patient's death from massive swelling and uncal herniation. During this period, radiographic studies, including repeat angiography, demonstrated sequential cortical venous occlusions and findings most consistent with venous insufficiency. Postmortem examination revealed no evidence of radionecrosis. Patient 2 exhibited a biphasic pattern of neurological deterioration at 3 and 6 years after radiosurgery. Associated with this unusual phenomenon, there was radiographic evidence of venous outflow obstruction of her thalamic AVM with prominent perinidal edema and progressive occlusion of the nidus.

CONCLUSION: We conclude that occlusive hyperemia is responsible for some cases of neurological deterioration after AVM radiosurgery, especially in a setting for which the time course or other clinical features are not as might be expected from a radiobiological perspective. The two patients we describe in this report suggest that manifestations may vary.

KEY WORDS: Arteriovenous malformation, Cerebral veins, Complications, Radiosurgery

Neurosurgery 55:228–234, 2004 DOI: 10.1227/01.NEU.0000126950.67029.F8 www.neurosurgery-online.com

Early Draining Vein Occlusion After Gamma Knife Surgery for Arteriovenous Malformations

BACKGROUND: Increased signals on T2-weighted magnetic resonance imaging usually interpreted as radiation-induced changes or brain edema is a common short- to mid-term complication after Gamma Knife surgery (GKS) for intracranial arteriovenous malformations (AVMs), although its nature remains to be clarified. Early draining vein occlusion with resultant brain edema or hemorrhage, although well established in surgical series, was not described in radiosurgical literature until recently.

OBJECTIVE: To outline the incidence, clinical manifestations, and outcomes of this unusual complication in our series of 1256 AVM patients treated with GKS.

METHODS: From 1989 to 2008, 1400 patients underwent GKS for cerebral AVMs or dural arteriovenous fistulae at the University of Virginia. In 1256 patients, magnetic resonance imaging after GKS was available for analysis of radiation-induced changes and early draining vein occlusion.

RESULTS: After GKS, 456 patients (36%) developed radiation-induced changes surrounding the treated nidi. Among these patients, 12 had early thro[illegible]sis of the draining vein accompanied by radiation-induced changes. Venous th[illegible]occurred 6 to 25 months (median 11.6 months) after GKS. Three patients w[illegible]matic on the image findings of venous occlusion and brain edema, 3 expe[illegible]adache, 1 had seizure and headache, and neurological deficits developed in s[illegible]nts with neurological deficits were treated with corticosteroids; 2 of the patients recovered completely, 1 still had slight hemiparesis, 1 had short-term memory deficits, and 1 died of massive intracerebral hemorrhage.

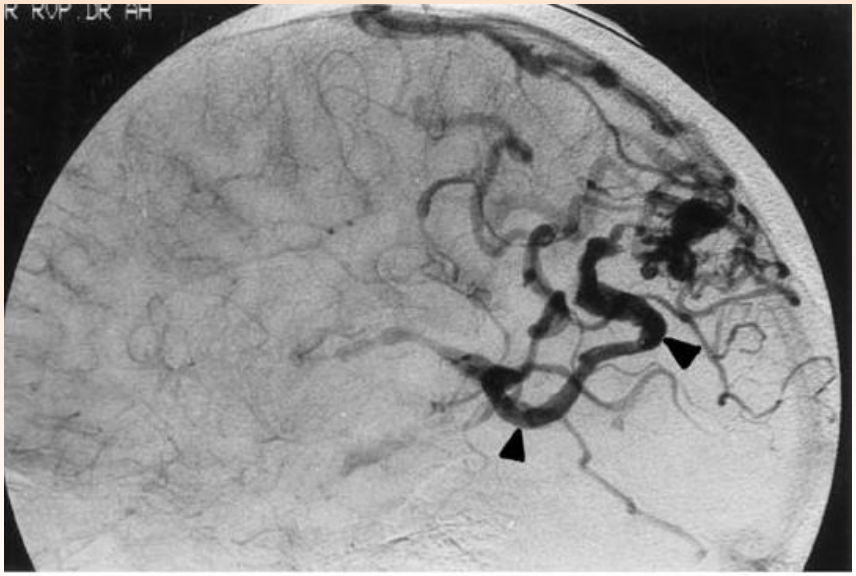
CONCLUSION: Although venous structures are considered more radioresistant, endothelial damage accompanied by venous flow stasis might cause early venous thrombosis and premature venous occlusion after radiosurgery for AVMs. In our series, all patients had a favorable outcome except 1 with a fatal hemorrhage.

KEY WORDS: Arteriovenous malformation, Complication, Gamma Knife, Radiation induced changes, Venous infarction, Venous thrombosis

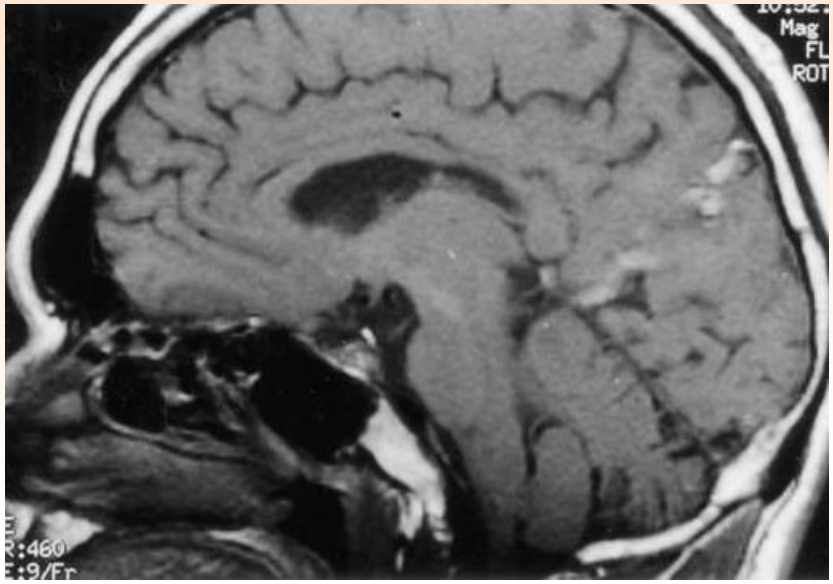
Neurosurgery 67:1293–1302, 2010 DOI: 10.1227/NEU.0b013e3181f2b396 www.neurosurgery-online.com

DISCUSSION

- Sawlani *et al. 2004 J of Neur Science*
- 1 cas de thrombose spontanée , Epilepsie.
- Anticomitiaux avant embolisation a M3
- Mécanisme thrombose évoqué mais pas à la phase initiale
- Pas d'anticoagulation



Hors , à J0, Phlébite deja presente !!!



IRM M3

DISCUSSION

- Notre experience :

1. Facteur déterminant de l'évolutivité des MAV
2. A permis dans 3/4 cas de modifier l'attitude thérapeutique pour aboutir à une réduction des symptômes
3. Signes IRM montrent systematiquement un ADC augmenté en faveur d'une congestion veineuse sur le territoire de drainage de la veine occluse.
4. Utilisation des anticoagulants est un élément du traitement de la MAV
    1. Doses curatives
    2. 8-10j
    3. Contrôle IRM et clinique

DISCUSSION

- **70 % des thromboses spontanées se font sur hemorragie ou HSA .** ***Abdulrauf Neurosurgery 1999***
  - **<u>Hypothese : Saignement due à Thrombose et pas l'inverse !!!</u>**

Suivi évolutif des MAV ?

Répéter les IRM en fonction de la clinique

Détection des thromboses débutantes

Conclusion

- La thrombophlébite est une modalité evolutive des MAV
- Sa fréquence est sous estimé car IRM phase aigue est rare
- L’ anticoagulation doit alors être mené parallèlement au traitement de la MAV