

대한뇌혈관외과학회 | 대한뇌혈관내수술학회

KSCVS-SKEN

합동 연수강좌

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주최 | 대한뇌혈관외과학회, 대한뇌혈관내수술학회

주관 | 대한신경외과학연구재단

평점 | 6점



Korean Society
of Cerebrovascular Surgeons
대한뇌혈관외과학회



2018 대한뇌혈관외과학회 임원진

실행이사회

직 위	성 명	소 속
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방사선수술	임용철	아주대학교병원
여성분과	박문선	울지대학교병원
뇌혈관해부연구회	최찬영	인제대학교 일산백병원
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특별이사(News Letter)	이상원	양산부산대학교병원
감 사	권순찬	울산대학교병원
감 사	현동근	인하대학교병원
간 사	이재일	부산대학교병원

상임이사회

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전임회장	오창완	분당서울대학교병원
전임회장	박현선	인하대학교병원

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직 위	성 명	소 속
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명예회장	김달수	가톨릭의대
명예회장	김승래	경북의대
명예회장	이제혁	전남의대
명예회장	한대희	서울의대
명예회장	이동열	침례병원
명예회장	최병연	영남의대
명예회장	권병덕	경희대학교병원
명예회장	허승곤	연세대학교 세브란스병원
명예회장	김한규	차의과학대학교 분당차병원

2018 대한뇌혈관내수술학회 임원진

명예회장

직 위	성 명	소 속
명예회장	백민우	인봉의료재단 뉴고려병원

회장

직 위	성 명	소 속
회장	고준석	강동경희대학교병원

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제4, 5대	권도훈	울산대학교 서울아산병원
제6대	안성기(작고)	(전) 한림대학교 성심병원
제7대	신용삼	가톨릭대학교 서울성모병원
제8대	권오기	분당서울대학교병원
제9대	김범태	순천향대학교 부천병원
제10대	성재훈	가톨릭대학교 성빈센트병원

인사말

올해도 대한뇌혈관내수술학회와 연합하여 합동연수강좌를 시행하게 되었습니다.

무더웠던 여름이 지나고 이제 9월 중순에 새로운 바람을 타고 온 지식들로

머리 안을 시원하게 해줄 멋진 강사 분들이 우리를 위해 열성적으로,

정성껏 준비한 내용들을 즐길 시간입니다.

최근 일본에서 venous approach를 주로 하시면서 뇌정맥계의 해부학에 대해 멋진 발표들을 해 오신

오사카 시립병원의 신경중재 분과에 근무하시는 마사키 코미야마 선생님을 통해 뇌정맥계 해부학을,

분당 서울대 병원의 권오기 교수를 통해서도 뇌동맥 해부학을

새로이 고찰하는 시간을 가지게 되어 기쁩니다.

강의를 수락해 주신 두 분 선생님께 깊은 감사를 드립니다.

총무이사이신 김정은 교수의 장소섭외 및 행사준비와 진행, 학술이사이신 이재환 교수의 세심한 배려로

beginner를 위한 뇌동맥류 접근 anatomy에 대한 비디오 강연을 준비하였습니다.

또한 급성기 허혈성 뇌졸중의 최근 치료의 update 한 내용들과,

해면상 혈관종에 대한 치료를 고찰하는 시간을 가지게 되었습니다.

힘들고 바쁜 와중이지만 분당에 오셔서 동병상련의 정을 나누시고,

머릿속을 새롭게 할 신선한 강의들을 접하고 즐기시길 바랍니다.

준비해주시는 강사님들과 여러분께 감사드리고

또한 저희 대한뇌혈관외과학회와 대한뇌혈관내수술학회가 다같이 발전하는 계기의 한 장에

동참해 주시고 격려를 부탁드립니다.

강건하시고 늘 승리하는 삶을 기원합니다. 감사합니다.



대한뇌혈관외과학회 회장 정영균

인사말

유난히도 무더웠던 올 여름,

끝없이 우리들을 힘들게 했던 폭염도 점차 지나가고 모든 것이 풍성해지는 가을이 오고 있습니다.

먼저 이번 2018년 합동연수강좌를 꼼꼼히 준비해주신 대한뇌혈관외과학회 정영균 회장님과
학회 이사진 여러분의 노고에 깊이 감사 드립니다.

뇌혈관질환을 전문적으로 다루는 대한뇌혈관외과학회와 대한뇌혈관내수술학회가

서로의 경험을 공유하고 새로운 술기와 지식을 습득하는 것은 양 학회 회원들의 질적 향상 뿐 아니라

아직까지도 국내에서 높은 사망률을 보이는 뇌혈관질환 관련 환자들에게

최선의 진료를 제공할 수 있는 필수적인 과정이라 판단됩니다.

갈수록 척박해지고 경쟁으로 내몰리는 의료 환경 속에서 뇌혈관질환을 전문적으로 다루는

신경외과의사로서의 역량을 다지고 양 학회 회원간의 친교를 다질 수 있는

뜻 깊은 합동연수강좌가 되기를 기대합니다.

다시 한번 이번 2018 대한뇌혈관외과학회/대한뇌혈관내수술학회 합동연수강좌를 위해

수고해주신 모든 회원 여러분께 감사 드립니다.



대한뇌혈관내수술학회 회장 **고 준 석**

Invited Speakers

Masaki Komiyama, M.D.

Director, Department of Neuro-Intervention, Osaka City General Hospital, Osaka



1979 Keio University of Medicine, Tokyo

Speciality: Neurosurgery, Neuro-intervention

Subspeciality: Cerebro-vascular diseases, Pediatric vascular diseases, Hereditary hemorrhagic telangiectasia, Head & Neck vascular diseases, Neurovascular anatomy

1979 Resident, Department of Plastic and Reconstructive Surgery, Keio University Hospital

1980 Resident, Departments of Surgery and Neurosurgery, the 2nd Tokyo National Hospital

1982 Senior Resident, Department of Neurosurgery, Osaka City University Hospital

1987-1988 Fellow, Department of Neurosurgery, University of Vienna, Austria

1984-1987, 1988-1993 Staff, Department of Neurosurgery, Baba Memorial Hospital, Osaka

1993-2000 Staff, Department of Neurosurgery, Osaka City General Hospital

Journals

2007-2010 Editor-in-Chief, Journal of Neuroendovascular Therapy

2011-2016 Section Editor (Intervention, Japan), Neuroradiology

2011- Section Editor (Intervention), Neurologica Medico-Chirurgica (Tokyo)

2010-2014 Reviewer, Journal of Neuroendovascular Therapy

2013-2017 Reviewer, Surgery for Cerebral Stroke

2018-present Editor-in-Chief, Interventional Neuroradiology

Memberships

The Japanese Society for Neuroendovascular Therapy

The Japan Neurosurgical Society

The Japanese Congress of Neurological Surgeons

Japanese Society on Surgery for Cerebral Stroke

World Federation of Interventional and Therapeutic Neuroradiology

Asian-Australasian Federation of Interventional and Therapeutic Neuroradiology

Global Research and Medical Advisory Board for cure HHT (2016-)

Niche Neuro-Angiology Conference

HHT JAPAN

Invited Speakers Masaki Komiyama, M.D.

Publications in English as a head author

1. **Komiyama M**, Yagura H, Baba M, Yasui T, Hakuba A, Nishimura S, Inoue Y: MR imaging: Possibility of tissue characterization of brain tumors using T1 and T2 values. *AJNR* 8:65-70, 1987
2. **Komiyama M**, Yasui T, Yagura H, Baba M, Hakuba A, Nishimura S, Inoue Y: Pitfalls in spinal MR imaging. *Neuro-Orthopedics* 3:90-98, 1987
3. **Komiyama M**, Hakuba A, Inoue Y, Yasui T, Yagura H, Baba M, Nishimura S: Magnetic resonance imaging: Lumbosacral lipoma. *Surg Neurol* 28:259-264, 1987
4. **Komiyama M**, Baba M, Hakuba A, Nishimura S, Inoue Y: MR imaging of brainstem hemorrhage. *AJNR* 9:261-268, 1988
5. **Komiyama M**, Yasui T, Yagura H, Fu Y, Sugata H, Kiyama M, Baba M: Chemical shift misregistration effect in spinal magnetic resonance imaging. *Neurol Med Chir (Tokyo)* 28:642-644, 1988
6. **Komiyama M**, Yasui T, Yagura H, Fu Y, Sugata H, Baba M: Magnetic resonance imaging of traumatic pneumocephalus. Case report. *Neurol Med Chir (Tokyo)* 28:677-680, 1988
7. **Komiyama M**, Yasui T, Baba M, Hakuba A, Nishimura S, Inoue Y: MR imaging of blood flow in the cavernous sinus. *Radia Med* 6:124-129, 1988
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102. **Komiyama M**: Functional venous anatomy of the brain for neurosurgeons. *Jpn J Neurosurg* 26:488-495, 2017
103. **Komiyama M**: Cardio-cephalic neural crest syndrome: a novel hypothesis of vascular neurocristopathy. *Interv Neuroradiol* 6:572-576, 2017

Invited Speakers

권오기

분당서울대병원 신경외과



1983-1989	Seoul National University, Medical School
1989-1990	Internship in Seoul National University Hospital
1990-1993	Military Doctor
1993-1997	Residency in Department of Neurosurgery, Seoul National University Hospital
1997-1998	Fellowship, Cerebrovascular section, Department of Neurosurgery, Seoul National University Hospital
1999-2000	Fellowship, Neurointerventional section, Department of Diagnostic Radiology, Seoul National University Hospital
2001.	Ph.D. Neurosurgery
2000-2002	Assistant Professor, Neurosurgery, Seoul Paik Hospital, Inje University
2003-	Professor, Neurosurgery, Bundang Seoul National University Hospital.
2012-2013	President of Society for Korean Endovascular Neurosurgeons.

대한뇌혈관외과학회 | 대한뇌혈관내수술학회

KSCVS-SKEN 합동 연수강좌

08:30	Registration		
08:40-08:50	Opening Remarks	정영균 (대한뇌혈관외과학회 회장) 고준석 (대한뇌혈관내수술학회 회장)	
08:50-09:00	Congratulatory Address	강성돈 (대한신경외과학회 회장)	
09:00-10:20	Scientific Session I: Cavernous Malformation	좌장: 성재훈 (가톨릭대), 강현승 (서울대)	
	1. Overview	연제영 (성균관대)	• 17
	2. Microsurgery of supratentorial CM	김용배 (연세대)	• 21
	3. Microsurgery of infratentorial CM	안재성 (울산대)	• 22
	4. Radiosurgery	박성현 (경북대)	• 33
10:20-10:40	Coffee Break		
10:40-12:10	Special Lecture	좌장: 정영균 (인제대), 고준석 (경희대)	
	1. Functional Venous Anatomy of the Brain	Masaki Komiyama (Osaka City General Hospital, Japan)	• 37
	2. Cerebral arterial anatomy	권오기 (서울대)	• 38
12:10-13:10	Lunch		
13:10-14:30	Scientific Session II: Dural AVF	좌장: 임동준 (고려대), 장철훈 (영남대)	
	1. Overview	조원상 (서울대)	• 49
	2. Transarterial approach	임용철 (아주대)	• 54
	3. Transvenous approach	박중철 (울산대)	• 60
	4. Direct approach; SOV, burr hole	김동준 (연세대 영상의학과)	• 63
14:30-14:50	Coffee Break		
14:50-16:30	Workshop by Video for Beginner (Surgical Anatomy and Basic Technique)	좌장: 김태선 (전남대), 김정은 (서울대)	
	1. ACOM, Distal ACA aneurysm	정준호 (연세대)	• 79
	2. MCA aneurysm	방재승 (서울대)	• 80
	3. PCOM, Anterior choroidal artery aneurysm	이상원 (부산대)	• 88
	4. CEA	주성필 (전남대)	• 91
	5. STA-MCA bypass	박익성 (가톨릭대)	• 96
16:30-17:30	Scientific Session III: SAH, Vasospasm	좌장: 고현승 (충남대), 김성림 (가톨릭대)	
	1. VSP, Basic research update	박동혁 (고려대)	• 103
	2. Intervention for VSP	신승훈 (분당제생병원)	• 104
	3. Medical complication & treatment	유찬중 (가천대)	• 105
17:30	Closing Remarks	정영균 (대한뇌혈관외과학회 회장) 고준석 (대한뇌혈관내수술학회 회장)	

대한뇌혈관외과학회 | 대한뇌혈관내수술학회

KSCVS-SKEN 합동 연수강좌

Scientific Session I: Cavernous Malformation

좌장: 성재훈 (가톨릭대), 강현승 (서울대)

1. Overview

연제영 (성균관대)

2. Microsurgery of supratentorial CM

김용배 (연세대)

3. Microsurgery of infratentorial CM

안재성 (울산대)

4. Radiosurgery

박성현 (경북대)





1. Overview

연제영

성균관대학교 의과대학 삼성서울병원 신경외과

KSCVS-SKEN 합동 연수강좌
2018.9.15.

Overview of Cerebral Cavernous Malformation (CCM)

cavernous angioma, cavernoma, cavernous hemangioma

Je Young Yeon

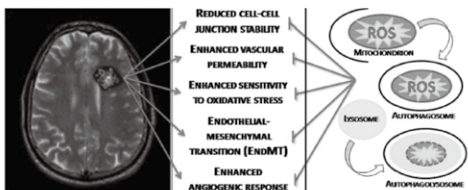
Department of Neurosurgery
Samsung Medical Center
Sungkyunkwan University School of Medicine

Epidemiology

- Prevalence
 - Retrospective analysis of 24,535 autopsies : **0.53%**
Otten et al. Neurochirurgie, 1989
 - Prospective 3T MR imaging of 2,715 participants : **0.44%**
Flemming et al. JAMA Neurol, 2017
- Symptomatic lesions
 - **Seizures (50%)**
 - Intracranial hemorrhage (25%)
 - Focal neurological deficits without radiographic evidence of recent hemorrhage (25%)
Al-Shahi et al., Lancet Neurol, 2012

Pathophysiology

- Enlarged, blood-filled endothelial compartments ('caverns') within the venous-capillary vascular bed (low flow)
- Multilobulated, often calcified vascular balls are characterized by a dysfunctional blood-brain barrier and leakiness
- Lined by **endothelium with defective intercellular junctions**
- Lack of pericytes and vascular smooth muscle cells



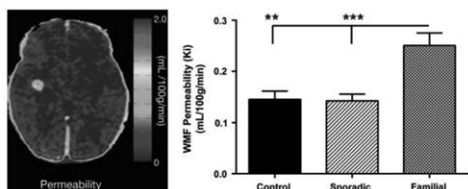
Pathophysiology

- Multiple familial form
 - **autosomal dominant germline mutations in CCM1 (KRIT1), CCM2, or CCM3 (PDCD10) genes**
 - numerous signaling pathways (including Rho, SMAD and Wnt/ β -catenin) and processes such as endothelial-mesenchymal transition (EndMT)
 - MEKK3 signaling and KLF2/4 function
 - Endothelial Toll-like receptor 4 (TLR4) and the gut microbiome
- Single sporadic form
 - **somatic mutations within the three CCM genes**
 - share common pathogenesis

McDonald et al., Hum Mol genet 23(16):4357-70, 2014

Pathophysiology

- Hyperpermeability associated with RhoA kinase activation
 - Potential therapy : RhoA kinase inhibition (Fasudil, Statins)
 - dynamic contrast-enhanced quantitative perfusion MRI
 - Permeability in white matter far (WMF) from lesions was significantly greater in familial than in sporadic cases, but was similar in CCM lesions.



Mitaki et al., JCBFM 35(10):1632-9, 2015

Natural history

- A meta-analysis of individual patient data (n=1620) from six studies
- to estimate the **5-year risk of symptomatic hemorrhage**
- independently associated with **clinical presentation** and location

	Number of people (%)	Number of outcome events during 5-year follow-up	Hazard ratio (95% CI)*	Estimated 5-year risk (95% CI)
Primary outcome: ICH (n=1620)				
ICH or FND presentation, brainstem location	495 (31%)	135	10.2 (5.0-23.9)	30.8% (26.3-35.2)
ICH or FND presentation, other CCM location	327 (20%)	45	5.6 (3.7-9.4)	18.4% (13.3-23.5)
Other presentation, brainstem CCM location	80 (5%)	4	1.8 (1.3-2.6)	8.0% (0.1-15.9)
Other presentation, other CCM location	718 (44%)	20	Reference	3.8% (2.1-5.5)

Horne et al., Lancet Neurol 15:166-73, 2016

Natural history

- A meta-analysis of 25 studies
- to estimate the incidence rate of **rehemorrhage**

Incidence rate	No. of studies (no. of subjects)	Bleeding	Model used	p Value for heterogeneity (I ²)	Overall pooled estimate (95% CI)
Median time of rehemorrhage	12 (—)	385	Weighted mean ^b	—	10.5 mo
Brainstem hemorrhage (person-years)	12 (875)	351	Meta-regression	0.9 (0%)	2.8 (2.5-3.3)
Nonbrainstem hemorrhage (person-years)	12 (875)	351	Meta-regression	0.9 (0%)	0.3 (0.1-0.5)
Overall brainstem rehemorrhage rate ^a	17 (1295)	971	Meta-regression	<0.001 (92%)	32.3 (19.8-52.7)
Overall nonbrainstem rehemorrhage rate ^a	17 (1295)	971	Meta-regression	<0.001 (92%)	6.3 (3.0-13.2)

- Rehemorrhage within 2 years vs thereafter (ratio 1.8, p=0.042)

Taslimi et al., Neurology 86:1984-91, 2016

Natural history

- Symptomatic CCM
 - The incidence of symptomatic hemorrhage or rehemorrhage is higher in brainstem lesions because little hemorrhage is more likely to cause symptoms in the brainstem.
 - Symptomatic hemorrhage and rehemorrhage rate in highly eloquent cortical locations : **unknown**
- Incidental CCM
 - 107 patients with a mean f/u period of 12 years
 - Definite hemorrhage related to CCM in only one patient
 - Annual bleeding rate 0.1% per patient**
 - No new seizures in any of the patients

Moore et al., J Neurosurg 120:1188-92, 2014

Variable definitions of CCM hemorrhage

Table 1. Definitions of CM Hemorrhage in Studies of the Untreated Clinical Course of ≥20 Participants (Organized Chronologically)

Study	Symptoms/Signs?	Imaging Evidence?		Acute Blood?		Growth	Comments
		CT	MRI	Within CM	Outside CM		
Del Gaudio et al ¹	■	...	?	...	...	...	Clinically significant, radiographically identifiable hemorrhage
Robinson et al ²	■	...	...	□	□	□	Or prior hemorrhage on lumbar puncture/fresh distal viable CM at surgery
Fritsch et al ³	■	■	■	■	■	□	Or prior hemorrhage on lumbar puncture/fresh distal viable CM at surgery
Konczak et al ⁴	...	■	■	...	...	...	Established by referring physicians
Alta et al ⁵	...	■	□	■	■	□	Or fresh distal viable CM at surgery, or extra-ventricular CM distal density on CT that decreased in size and density on follow-up CT
Kim et al ⁶	...	...	...	...	...	...	No hemorrhage definition given
Porter et al ⁷	■	■	■	□	■	■	Increase in CM size by 25% or more in diameter with associated mass effect or edema and relevant symptoms also qualified
Abdelrazek et al ⁸	■	■	■	...	...	□	Same criteria as Fritsch et al ³
Porter et al ⁹	■	■	■	■	■	□	
Merritt et al ¹⁰	■	...	...	□	■	□	
Labauge et al ¹¹	...	...	■	■	...	...	Hemorrhage was distinguished from mass change in size
Barker et al ¹²	■	■	■	...	...	■	Some events without contemporaneous imaging classified as hemorrhages. Lesion growth was a hemorrhage if accompanied by clinical deterioration
Kapoor et al ¹³	■	...	■	■	■	□	
Ceballos et al ¹⁴	■	□	■	...	...	...	

■ indicates contributed to hemorrhage definition; □, not required for hemorrhage; ?, unclear; ... not specified.

Al-Shahi et al., Stroke 39:3222-30, 2008

Risk factors

- Retrospective analysis of 154 patients
- General predictor of hemorrhage
 - young age (<45 years, OR 2.2), infratentorial location (OR 2.9), and existence of a DVA (OR 4.7)
- Stratified by location – supratentorial (98 patients)

A. Results of risk factors for hemorrhage in patients with CM			
Patient characteristics	Ruptured (n = 51)	Unruptured (n = 47)	p Value ^a
Female	31 (61)	27 (57)	NS
Age <45 y	34 (67)	22 (47)	NS
Positive family history	2 (4)	1 (2)	NS
Hypertension	5 (10)	8 (17)	NS
Antiplatelet therapy	0 (0)	1 (2)	NS
Multiple CMs	6 (12)	7 (15)	NS
CM size ≥1 cm ^{3b}	49 (96)	44 (94)	NS
DVA ^c	27 (53)	10 (21)	<0.01, ^c OR 4.16 (95% CI 1.7-10.13)

Kashefiolasi et al., Neurology 90:e856-63, 2018



Risk factors

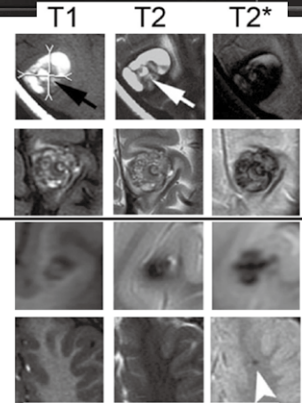
- Retrospective analysis of 154 patients
- General predictor of hemorrhage
 - young age (<45 years, OR 2.2), infratentorial location (OR 2.9), and existence of a DVA (OR 4.7)
- Stratified by location – infratentorial (47 patients)

A. Results of risk factors for hemorrhage in patients with CM			
Patient characteristics	Ruptured (n = 33)	Unruptured (n = 14)	p Value*
Female	23 (70)	7 (50)	NS
Age <45 y	14 (42)	6 (43)	NS
Positive family history	1 (3)	0 (0)	NS
Hypertension	8 (24)	2 (14)	NS
Antiplatelet therapy	2 (6)	1 (7)	NS
Multiple CMs	6 (18)	0 (0)	NS
CM size ≥1 cm ³	30 (91)	4 (29)	<0.0001,* OR 35 (95% CI 6.9–178)
DVA*	12 (36)	2 (14)	NS

Kashefiolasi et al., *Neurology* 90:e856-63, 2018

MRI classification

- Type I (acute or subacute)
 - T1 high / T2 high or low
- Type II (minor repetitive)
 - T1 mixed / T2 mixed
- Type III (chronic inactive)
 - T1 hypo / T2 hypo
- Type IV (suspicious)
 - Microbleed visible on T2*
 - Familial or cavernomatosis



Zabramski et al., *J Neurosurg* 80:422-32, 1994 Nikoubashman et al., *AJNR* 36:2177-83, 2015

MRI classification

- Retrospective review of 326 patients with 410 cavernomas
- Zabramski's classification
 - Type I (27%) / Type II (35%) / Type III (38%)
- During a follow-up of 34.7 months, 53 lesions (13%) bled
- Prior hemorrhage (adjusted HR, 7.13) / MRI appearance
- Annual bleeding risk : type I > type II >>> Type III

Table 3 Prospective haemorrhage rate according to MR appearance in adult patients with cavernous malformations

Interval	Type I	Type II	Type III
0-≤1 year	10.35 (5.73 to 18.69)	5.01 (2.39 to 10.51)	1.31 (0.33 to 5.26)
<1-≤2 years	11.89 (6.19 to 22.86)	5.75 (2.58 to 12.80)	1.69 (0.42 to 6.76)
<2-≤3 years	9.21 (3.46 to 24.55)	5.77 (2.17 to 15.37)	2.51 (0.63 to 10.04)
<3 years	4.05 (1.01 to 16.21)	2.75 (0.89 to 8.53)	0.71 (0.10 to 5.07)
Overall	9.47 (6.45 to 13.90)	4.74 (3.06 to 7.34)	1.43 (0.68 to 3.00)

Jeon et al., *J Neurol Neurosurg Psychiatry* 85:1366-70, 2014

Medical treatment (β-blocker, Propranolol)

- Propranolol treatment for a giant infantile brain cavernoma. *J Child Neurol* 25:653–655, 2010 (1 case)
- Propranolol for cerebral cavernous angiomas: a magic bullet. *Clin Pediatr (Phila)* 53:189–190, 2014 (1 case)
- Successful treatment of multifocal intracerebral and spinal hemangiomas with propranolol. *J Am Acad Dermatol* 70:e83–e84, 2014 (1 case)
- Propranolol stops progressive multiple cerebral cavernoma in an adult patient. *J Neurol Sci* 367:15–17, 2016 (1 case)
- Propranolol treatment of cavernous malformations with symptomatic hemorrhage. *World Neurosurg* 88:631–639, 2016 (2 cases)

Medical treatment (β-blocker, Propranolol)

- Retrospective review of 408 patients
- Descriptive and survival analyses of patients on long-term β-blocker medication vs those who were not

TABLE 5. Multivariate analysis of factors that influence follow-up hemorrhage among 496 CCMs

Factor	p Value* (HR, 95% CI)
Any β-blocker	0.53 (1.36, 0.52–3.56)
Brainstem location	0.03 (2.40, 1.09–5.29)
Hemorrhage at diagnosis	0.69 (1.15, 0.57–2.34)
Younger age	0.01 (0.98, 0.96–0.99)

* Cox regression.

- β-blocker does not seem to be associated with a decreased hemorrhage risk at presentation or during follow-up.

Goldberg et al., *J Neurosurg (Epub)*, 2018

Candidates for medical therapy

Targeted pathway/function	Drug(s)	Testing/proof of concept status	Development status
RhoA/ROCK	Fasudil ⁴⁴	Predclinical ⁴⁴	No current pathway for clinical development (side effects and expiration of patent)
	Statins ⁴⁵	Predclinical ⁴⁵	Ready for repurposing with IND exemption, Phase I/IIa proof of concept trial proposed
	ROCK-2 specific inhibitors (BA-1049)	Drug development stage (SBR), predclinical ⁴⁶ in progress	BioAzone committed to development for CASH indications in humans
Inflammation	Tempol ⁴⁷	Predclinical effect in acute models	Incursion Pharmaceuticals received Orphan Drug status, committed to development for CASH
	Immuno-modulators (B-cell depletion) ^{27,48}	Predclinical ⁴⁸	Potential repurposing of B-cell depletion biologics developed by several pharma companies for multiple sclerosis and other indications
	N-acetylcysteine ⁴⁹	Mechanistic Studies	
	Arenanthramide ⁵⁰	Mechanistic Studies	
	Platinum nanocages ⁵¹	Mechanistic Studies	
	Vitamin D ^{52,53}	Cohort studies showing aggressive CA disease in Vit D deficient subjects ⁵²	Clinical trials not likely because of promiscuous use of Vit D in the population
Angiogenesis/VEGF	Propranolol ^{45,52}	Empiric case studies	Ready for repurposing with IND exemption, Plans for pragmatic exploratory trial
	VEGF receptor inhibitor (SU5416 semaxanib) ⁵³	Predclinical	Not likely to be pursued because of systemic side effects
Endothelial mesenchymal transition/β-catenin	Inhibitors of TGF-β and β-catenin (ie, Solixadec metabolites) ^{54–56}	Predclinical	Solixadec proposal to the European Commission
Autophagy	mTOR inhibitors, autophagy inducers (ie, Torin 1, Rapamycin) ⁵⁷	Predclinical	
DELTA-NOTCH	Recombinant DLL4 ⁵⁸ Sorafenib ⁵⁹	Predclinical	
MEKK1/ERK1/2	B000289 (anti-MEKs), ⁶⁰ XMCD-109 (anti-ERKs) ⁶¹	Predclinical	

Polster et al., *Neurosurgery (Epub)*, 2018

Antithrombotics

- Retrospective review of 16 patients (among 87 patients)
 - 11 antiplatelet alone, 3 anticoagulant alone, 2 both
 - 0 of 16 initially presented with hemorrhage
 - None developed hemorrhage** over the 82 person-years
- Schneble et al., Stroke 43:3196–9, 2012*
- Retrospective review of 40 patients (among 292 patients)
 - 32 antiplatelet alone, 6 anticoagulant alone, 2 both
 - 5 of 40 (13%) initially presented with hemorrhage
 - 1 patient developed hemorrhage** over the 258 person-years
 - Annual hemorrhage rate 0.41%**

Flemming et al., J Neurosurg, 2013

Antithrombotics

- Retrospective review of 82 patients (among 408 patients)
 - 81% antiplatelet, 19% anticoagulant
 - 19 of 91 (21%) lesions initially presented with hemorrhage
 - 1 lesion developed hemorrhage** over the 143.9 lesion-years
 - Annual hemorrhage rate 0.7%**

Variable	No. of CCMs (%)	% of Group Developing De Novo CCM-Related Hemorrhage (no.)	Survival Analysis			
			p Value (Cox univariate)	p Value (Cox multivariate)	HR	95% CI
Age (continuous), yrs	48 ± 19	38 ± 17	0.035	0.019	0.98	0.959–0.998
Sex			0.249	—	—	—
Male	243 (49.4)	38.8 (14)	—	—	—	—
Female	249 (50.6)	61.1 (22)	—	—	—	—
High blood pressure	141 (28.7)	19.4 (7)	0.634	—	—	—
ATM	78 (15.8)	2.8 (1)	0.145	—	—	—
Antiplatelet medication	63 (12.8)	0 (0)	0.231	—	—	—
Anticoagulant medication	15 (3.0)	2.8 (1)	0.903	—	—	—
Brainstem location	61 (12.4)	25.0 (9)	0.017	0.008	2.83	1.318–6.065
CCM-related hemorrhage at presentation	128 (26.0)	30.5 (11)	0.239	—	—	—
Multiple CCMs	181 (36.8)	41.7 (15)	0.112	—	—	—
Association with DVA	90 (18.3)	25.0 (9)	0.125	—	—	—

Bervini et al., J Neurosurg (online), 2018

Research priorities (non-surgical)

- How do cavernomas start and develop?
- Could drugs targeted at cavernomas improve outcome?
- What mechanisms trigger bleeding or epileptic seizures?
- Does regular monitoring of cavernomas improve outcome?
- Do any specific activities provoke bleeds or other symptoms?



2. Microsurgery of Supratentoria CM

김용배

연세대학교 의과대학 강남세브란스병원 신경외과

Cavernous malformations(CMs) are common vascular anomaly of the central nervous system, potentially causing hemorrhagic stroke and various spectrum of neurological morbidity. To select the best strategies for each clinical scenario of CMs, one must be requested to weigh the treatment risk against the best estimate of the cumulative natural risk. To date, there has been a notable progress on understanding of genetic background of CMs, imaging techniques, and treatment modalities.

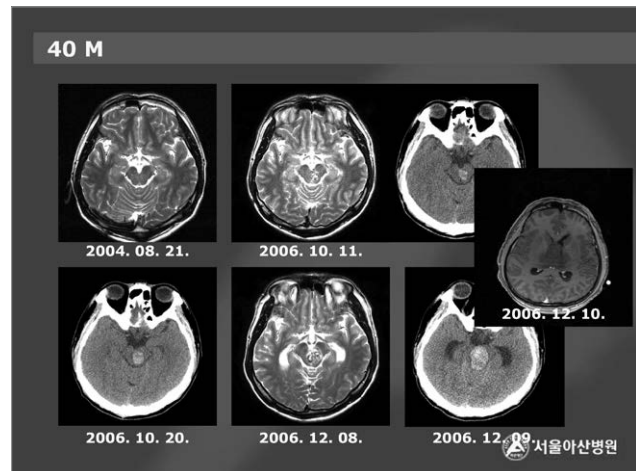
According to previous study, CMs showed the bleeding rate of 2.3%/person/year (1.4%/lesion/year), and recurrent bleeding rate of 7.8%/person/year. Follow-up study with MR imaging revealed that the CMs showing mixture of subacute and chronic hemorrhage with hemosiderin rim had a higher frequency to rebleed than other types of CMs. Developmental venous anomaly seemed to affect concurrent sporadic CM by its angioarchitectural flow dynamics.

Microsurgical treatments of CMs are primarily aiming to minimize neural tissue trauma and maximize the capability of elimination of the target. Recent advances in microsurgical armamentarium including sophisticated monitoring, neuro-navigations, and functional imaging techniques could shed us a valuable light to reach the goal of microsurgical options. In this review, the experiences mainly focusing on microsurgery of supratentorial CMs will be presented. A concise consolidation of the growing body of knowledge regarding CMs will be provided by referencing literatures as well.

3. Microsurgery of infratentorial CM

안재성

울산대학교 의과대학 서울아산병원 신경외과



Introduction

Brain stem Cavernous Malformations (CMs)

- 9~35% of brain CMs
- Benign disease – curable, clinically malignant
- Surgery : relatively easy / high morbidity
 - no interstitial brain tissue, gliotic capsule
 - skull base techniques
 - navigation
 - intraoperative monitoring
- MRI : revolutionary
 - asymptomatic CMs
- natural history

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Introduction - Brain stem CMs

Clinical characteristics

- repeated hemorrhage
 - : rate increases after one hemorrhage
- young patients, normotensive, repeated sx attack

Risk of symptomatic hemorrhage

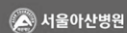
Author & year	No of patients	Follow up (mos)	Annual hemorrhage rate (%)
Kondziolka, et al. 1995	47	43	32
Amin, et al. 1998	73	65	17.4
Kida et al. 1999	80	27	31.8
Pollock et al. 2000	17	51	24.8

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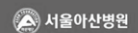
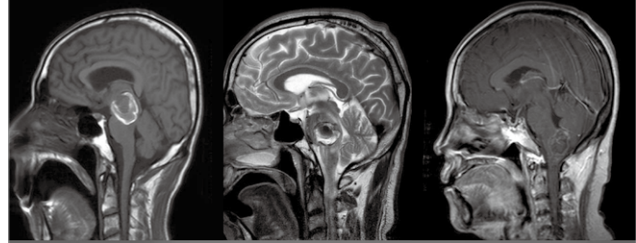


Surgical indications

- symptomatic hemorrhage who harbor lesions that approach the pial surface
 - abrupt or progressive neurological deficits
 - acute hemorrhage beyond the capsule
 - severe mass effect
 - Repeated, multiple hemorrhage that causing deterioration of clinical course
- first hemorrhage - conservative
 - delay surgery in acute hemorrhage for 3~5 days
 - more conservative in children

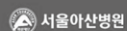


Brain stem



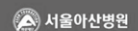
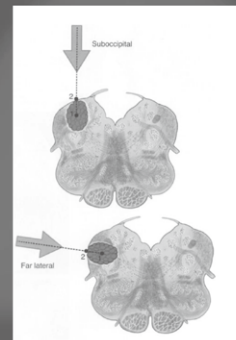
Clinical presentations

Presentation	No	%
Headache/dizziness, nausea	9	27.3
Cranial nerve symptoms	22	66.7
III, IV, VI	12	36.4
V	3	9.1
VII	3	9.1
IX, X, XI	2	6.1
XII	2	6.1
Cerebellar symptoms	16	48.5
Hemiparesis	7	21.2
Hemiparesthesia or hemihypesthesia	6	18.2
Horner syndrome	1	3.0

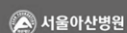
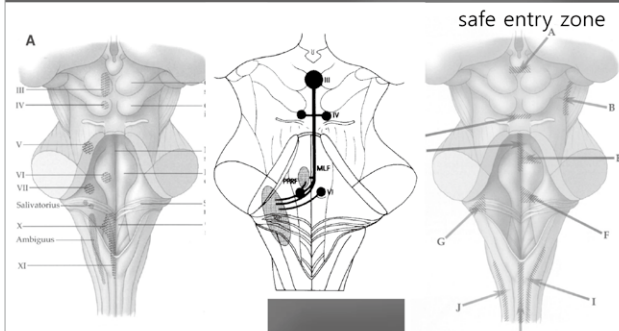


Surgical Treatment

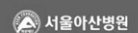
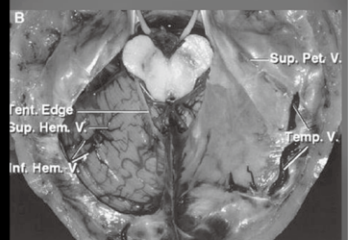
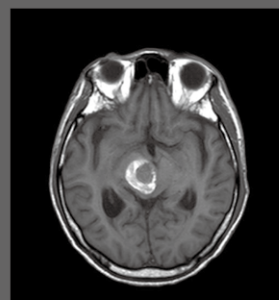
- Approach
- Midline
 - Retromastoid
- Two point method



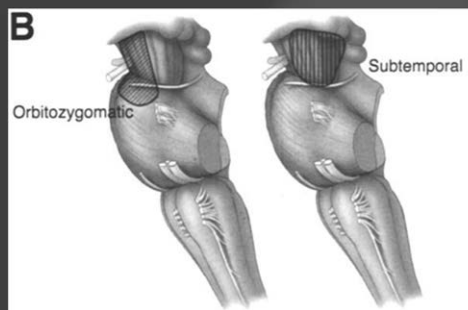
Surgical Treatment



Surgical approach – midbrain



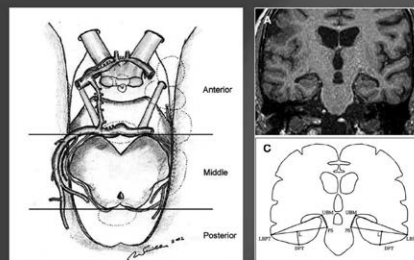
Surgical approach – midbrain



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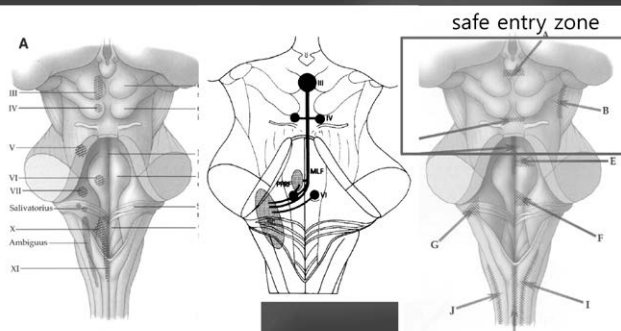
Surgical approach – midbrain

• Subtemporal approach



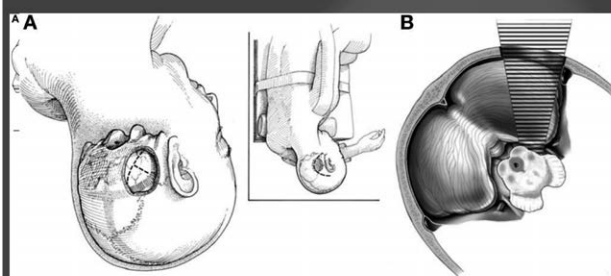
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Surgical Treatment



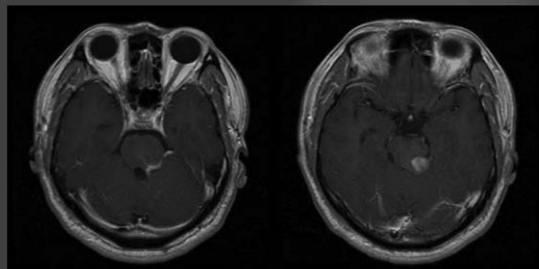
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Surgical approach – midbrain

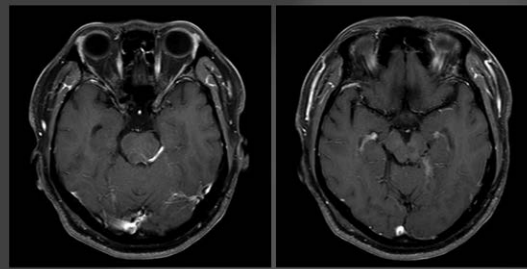


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48M, diplopia, ataxia, sensory change



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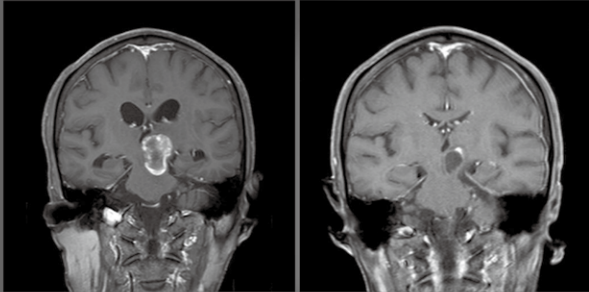


1 year F/U, postop

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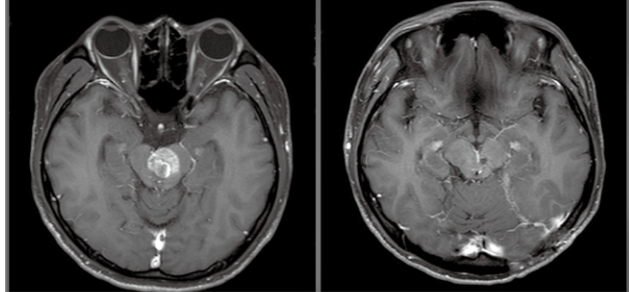
61M, diplopia, hemiparesis, facial palsy



3 week F/U, postop

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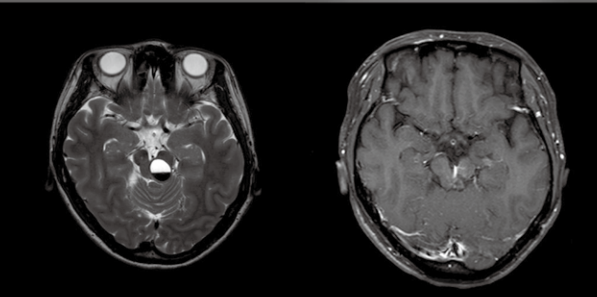
30M, diplopia, hemiparesis, confusion



0.5 year F/U, postop

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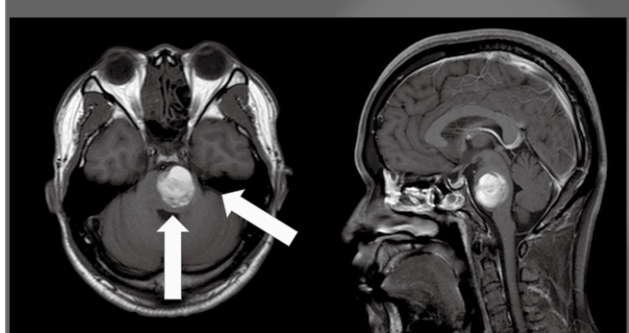
49F, hemiparesis, ataxia



1 year F/U, postop

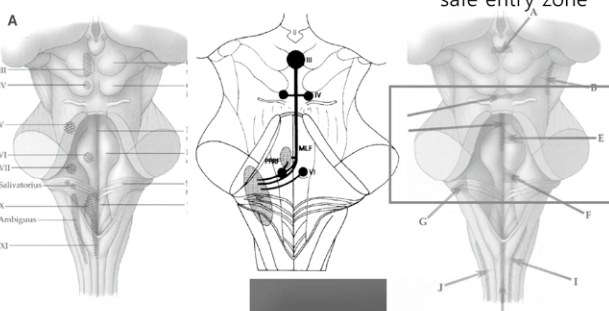
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Surgical approach - pons



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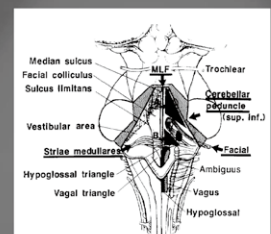
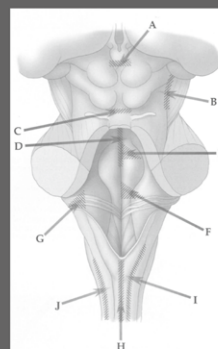
Surgical Treatment



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Surgical approach

• Safe entry zone – fourth ventricle floor

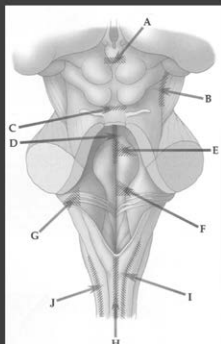


D. median sulcus above the facial colliculus
E. suprafacial
F. infrafacial

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Surgical approach

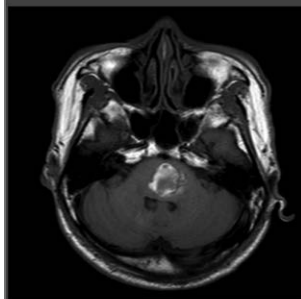
• Safe entry zone



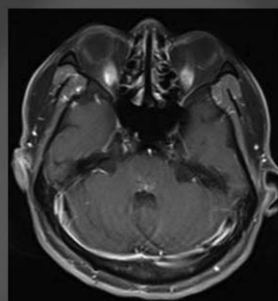
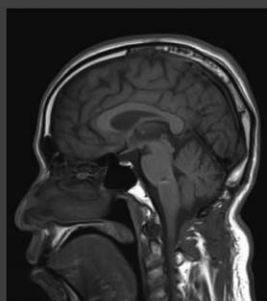
- A. Supracollicular
- B. lateral mesencephalic sulcus
- C. infracollicular
- D. median sulcus above the facial colliculus
- E. suprafacial
- F. infrafacial
- G. area acustica
- H. Posterior median fissure below the obex
- I. posterior intermediate sulcus
- J. posterior lateral sulcus

서울아산병원

57M, diplopia, ataxia



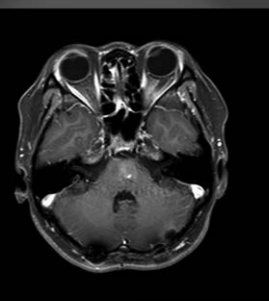
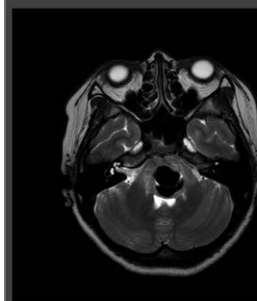
서울아산병원



1 year F/U, postop

서울아산병원

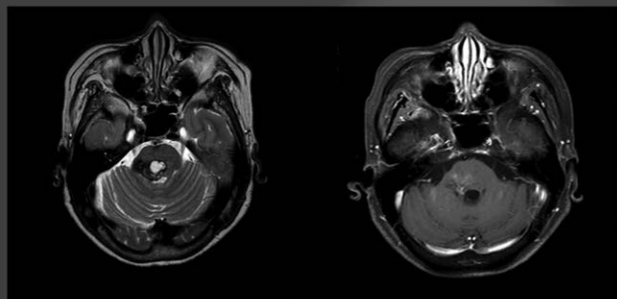
33F, diplopia, hemiparesis



6 month F/U, postop

서울아산병원

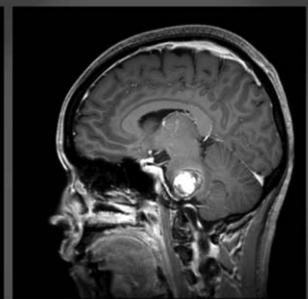
55F, facial numbness, ataxia



2 year F/U, postop

서울아산병원

41F, dizziness, facial paresthesia



2016 Aug 01

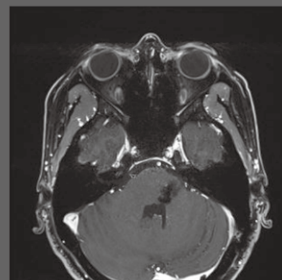
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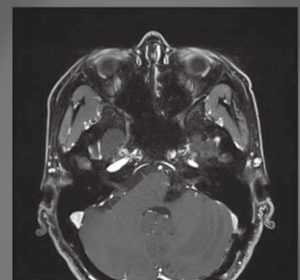
2016 Aug 15



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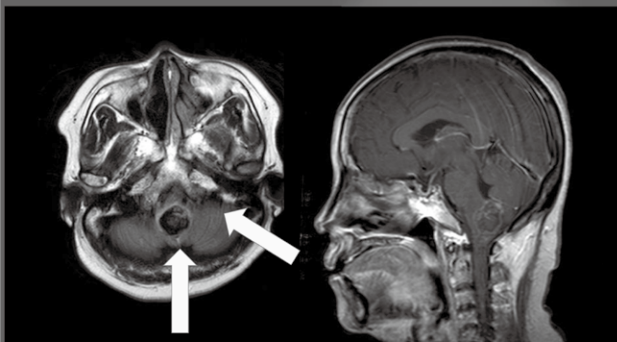


2017 Mar 23



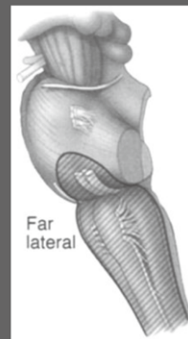
서울아산병원

Surgical approach - medulla

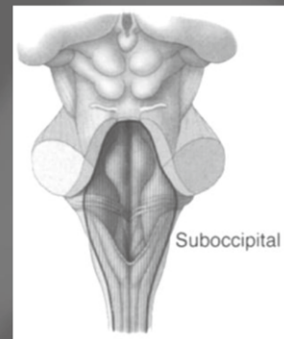


서울아산병원

Surgical approach - Medulla



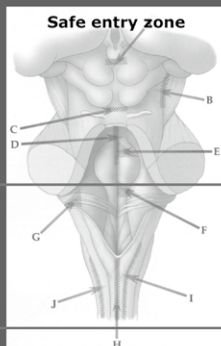
Far lateral



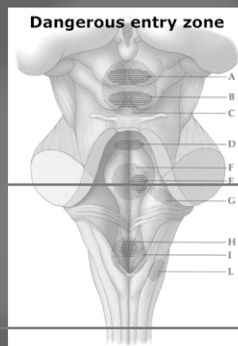
Suboccipital

서울아산병원

Surgical approach - medulla



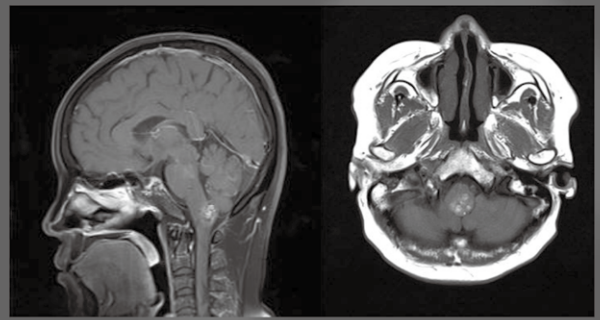
Safe entry zone



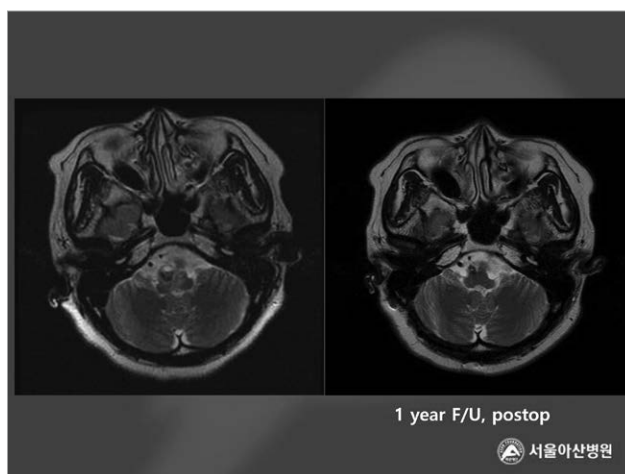
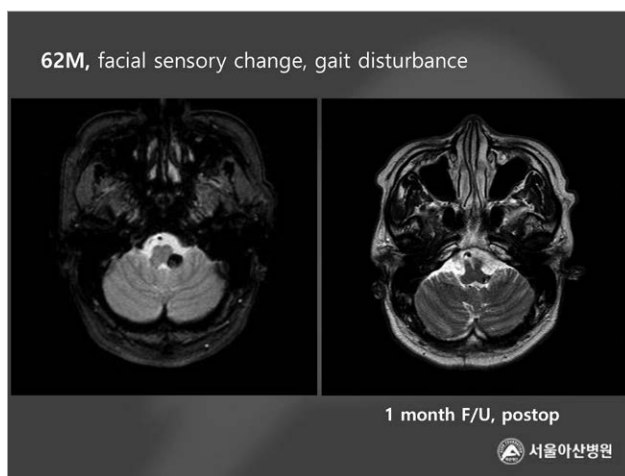
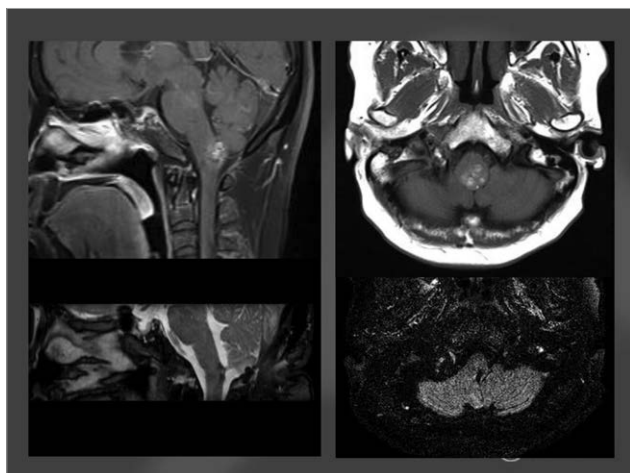
Dangerous entry zone

서울아산병원

57/F, dysarthria



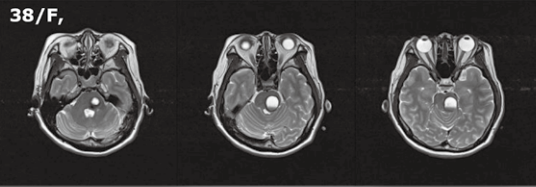
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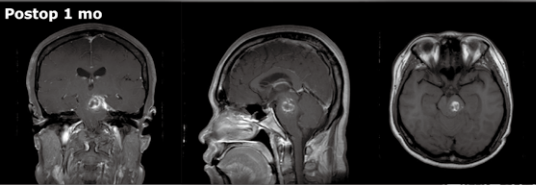


Postop rebleeding - midbrain

38/F,



Postop 1 mo

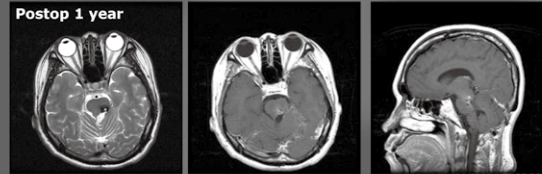


Postop rebleeding - midbrain

Postop 3 mo

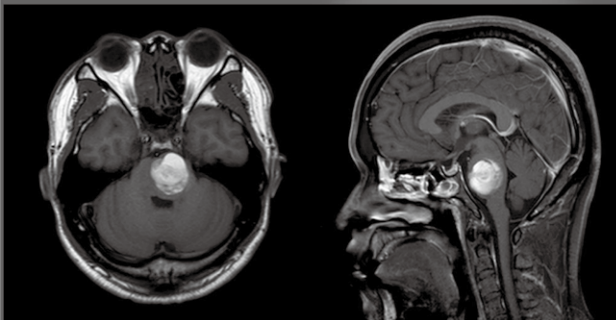


Postop 1 year

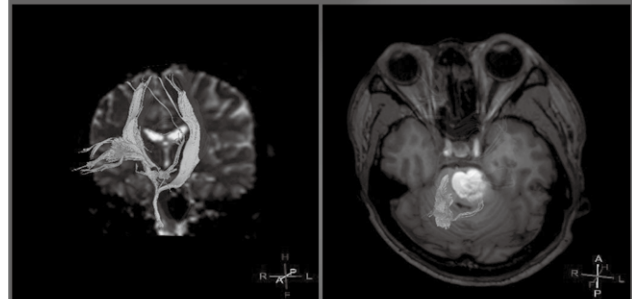


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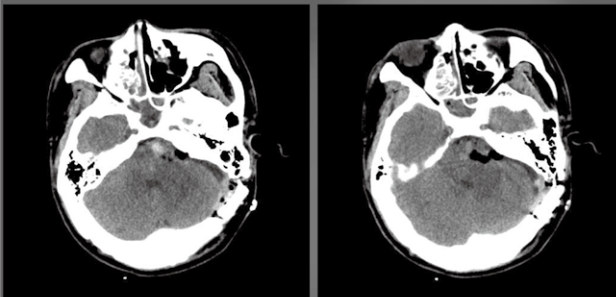
28M, dysarthria, hemiparesis



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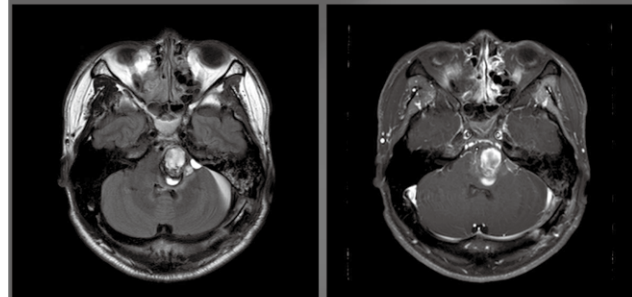


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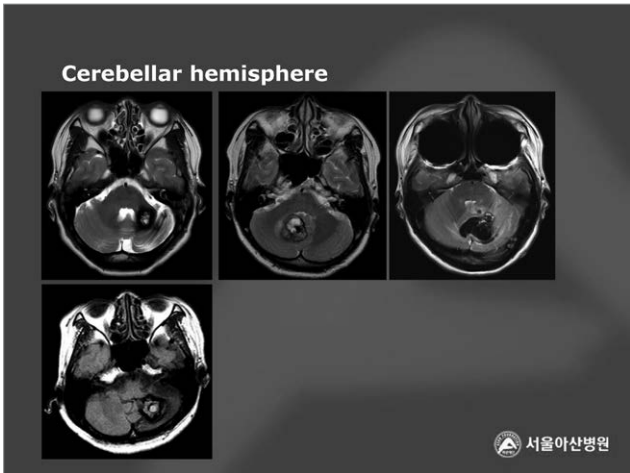
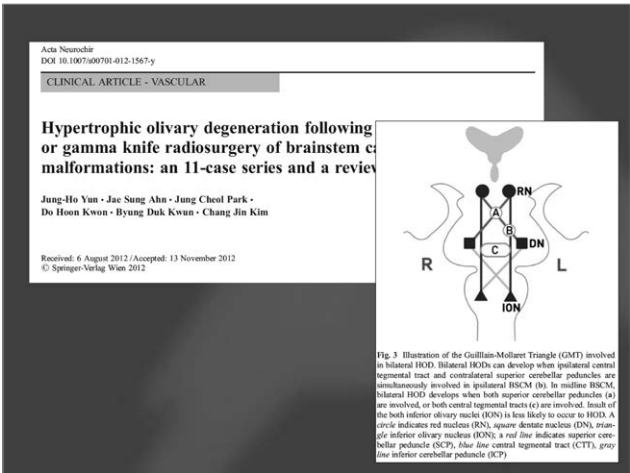
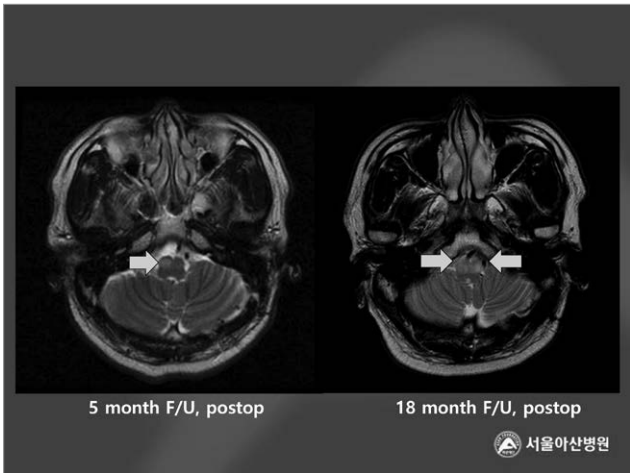
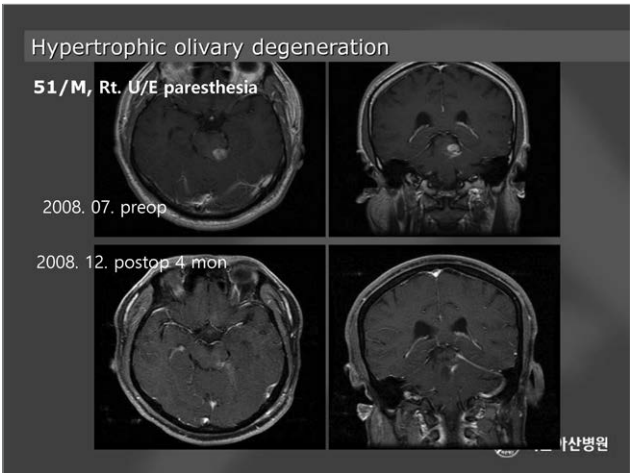
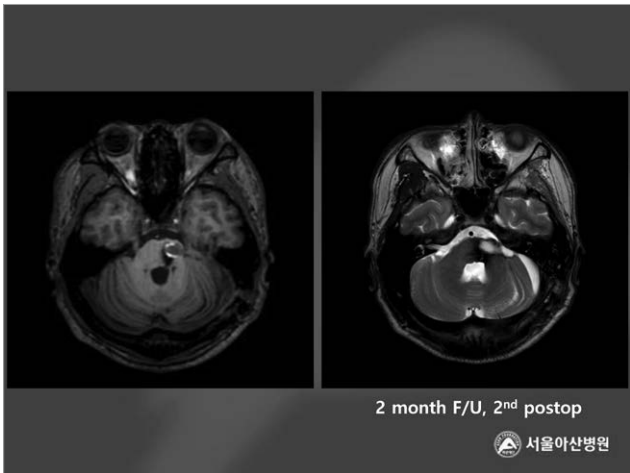
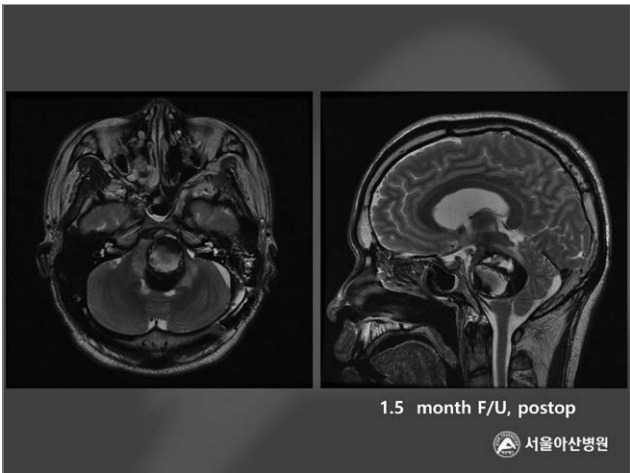
2 days F/U, postop

서울아산병원



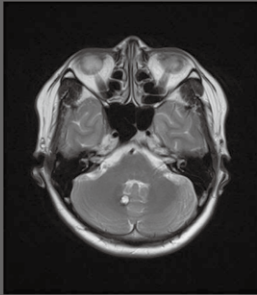
1 month F/U, postop

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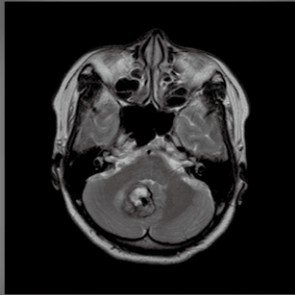




58F, dizziness, 균형을 잡기 어렵다

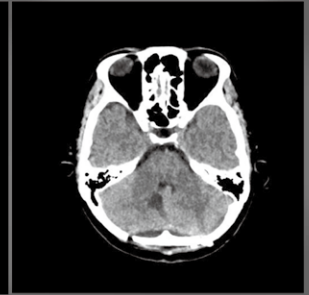
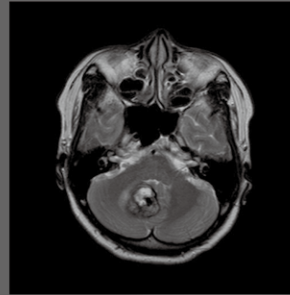


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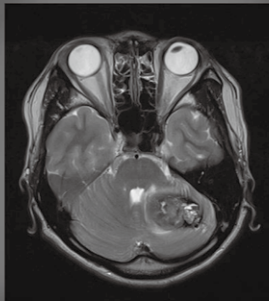
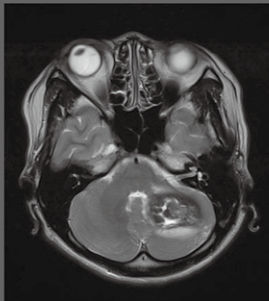
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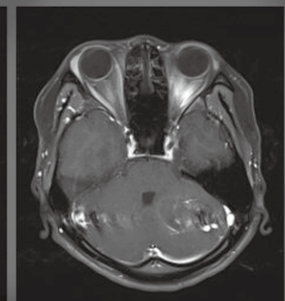
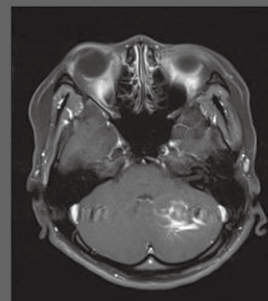


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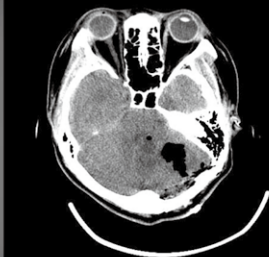
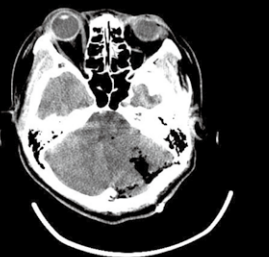
24/F Headache, dizziness, ataxia



서울아산병원

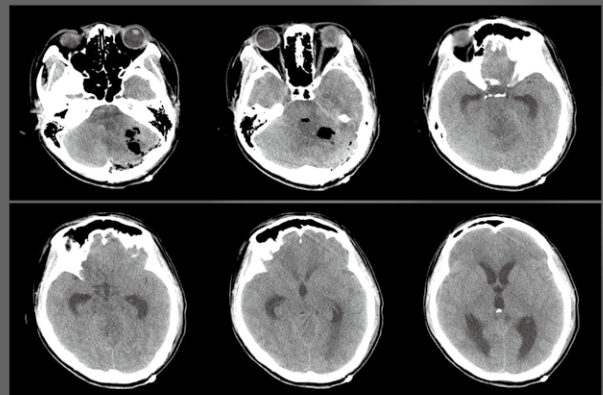


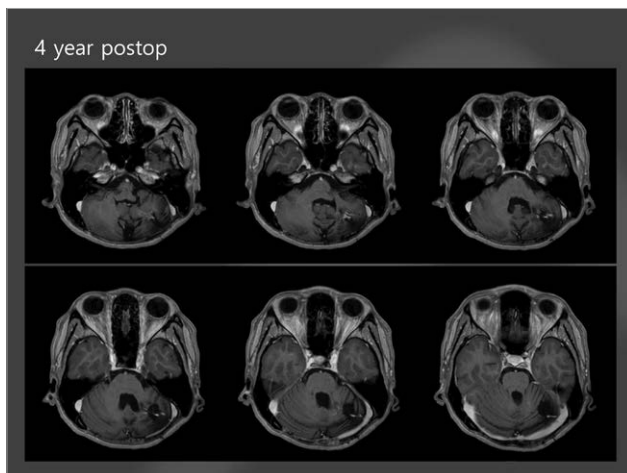
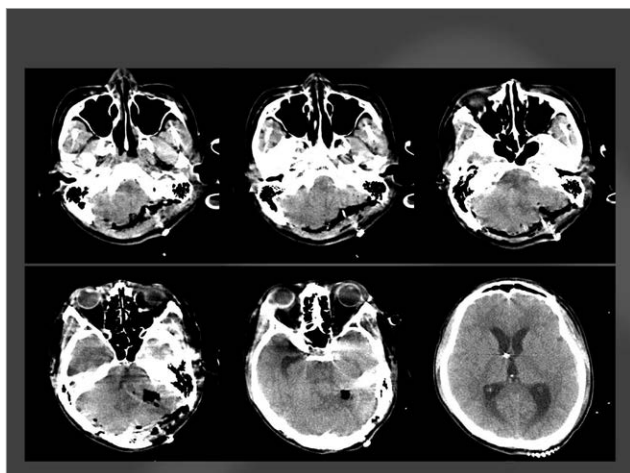
서울아산병원



서울아산병원

1st postop day





Conclusions

Brain stem Cavernous Malformations (CMs)

- Complete surgical resection could be possible in about 80% of patients.
- Rebleeding rate was lower in cases with complete surgical resection than in cases with incomplete resection
- Surgery is highly recommended for patients with symptomatic hemorrhage who harbor lesions that approach the pial surface, and have abrupt or progressive neurological deficits, and have severe mass effect
- Surgical exposure and knowledge of the anatomy are very important for the safe, total resection of the mass

Conclusions

Approaches

- Exposure is Everything!
- Preserve Venous Anomaly
- Image Guidance
- SSEP's & MEP's
- Sharp Dissection
- EEG - Burst Suppression
- Know Your Anatomy

2 point method



4. Radiosurgery

박성현

경북대학교병원 신경외과

Improvements in surgical techniques have made possible resection of some cavernous malformations (CMs) in previously unresectable locations such as the brainstem, thalamus, and basal ganglia. Despite these advances, some CMs are located in regions that pose an unacceptable surgical risk. For these patients and for those with medical contraindications to surgery, stereotactic radiosurgery (SRS) has been advocated as an alternative treatment. Our understanding of natural history of CMs and the role of SRS in the treatment of these lesions is controversial. SRS has been introduced for the treatment of CMs, assuming that their pathological vessels will respond to radiosurgery with a response similar to AVMs. CMs are included as accepted indications for SRS, not established indications. Established indications for SRS have withstood the test to time, and patient outcomes are comparable to, if not better than, other treatment options, such as critically located AVMs, brain metastases, and recurrent vestibular schwannomas. Accepted indications for SRS include dural arteriovenous fistulas, high-grade gliomas, and CMs. SRS for accepted indications is frequently performed, but the available data are not conclusive at this point as to whether patient outcomes are superior to alternative management strategies. When treatment for CMs is considered, bleeding and seizure are two major clinical factors. If left untreated, the bleeding will repeat and can cause neurological deterioration, especially from deep-seated lesions. Therefore, minimally invasive treatment with low morbidity such as SRS should be considered even if long-term benefits have not yet been identified. The Author presents the efficacy of SRS for CMs and reports long-term effects on bleeding rate and epilepsy control. SRS seems to be relatively effective and safe for reducing the rebleeding rate to deep-seated CMs. Careful selection of a low marginal dose and an optimal radiosurgical technique are helpful to achieve good outcomes.

Key Words: Cavernous malformation · Stereotactic radiosurgery

대한뇌혈관외과학회 | 대한뇌혈관내수술학회

KSCVS-SKEN 합동 연수강좌

Special Lecture

좌장: 정영균 (인제대), 고준석 (경희대)

1. Functional Venous Anatomy of the Brain

Masaki Komiyama (Osaka City General Hospital, Japan)

2. Cerebral arterial anatomy

권오기 (서울대)





1. Functional Venous Anatomy of the Brain

Masaki Komiyama

Osaka City General Hospital, Japan

Knowledge of the functional vascular anatomy of the brain is essential in the neuro-interventional procedures. Similarly, this knowledge is also required in neurosurgery, especially when a particular artery or vein is to be sacrificed during surgery. Permanent occlusion of the major artery is challenged by balloon occlusion test, but for venous sacrifice, such a procedure is practically not applicable. Up to now, reliable methods to judge safety of such destructive procedures are lacking. Knowledge of the basic angioarchitecture of the cerebral veins, in other words, “functional venous anatomy” may help to understand safety or risk of sacrificing cerebral veins.

Development of the cerebral veins does not parallel to that of the cerebral arteries. Paired primary head veins developed at both sides of the neural tube collect blood from the superficial primitive dural plexuses (anterior, middle, and posterior). After the closure of the neural tube, the initial choroidal drainage to the median vein of prosencephalon is transferred to the paired internal cerebral veins and the great vein of Galen. Dorsolateral enlargement of the cerebrum requires development of tentorium cerebelli. Tentorial sinus within the tentorium initially receives blood transversely from telencephalon, diencephalon, and mesencephalon, and transfers its role to the longitudinally formed basal vein (1st, 2nd and 3rd portions, respectively). Superficial cerebral veins (Sylvian, Trolard, and Labbé) establish enough cortical anastomoses each other. Subsequently, the superficial Sylvian vein draining to the tentorial sinus is captured to the cavernous sinus (cavernous sinus capture) to a variable degree following regression of the distal tentorial sinus. Variations of drainage patterns of the basal vein, cavernous sinus capture, collaterals at the base of the brain (Trolard venous circle), development of transcerebral (transmedullary) veins may provide various clinical pictures in cerebral venous pathologies. Understanding of the functional venous anatomy is essential for the interpretation of the pathophysiology of the cerebral venous diseases and for the safer treatment.

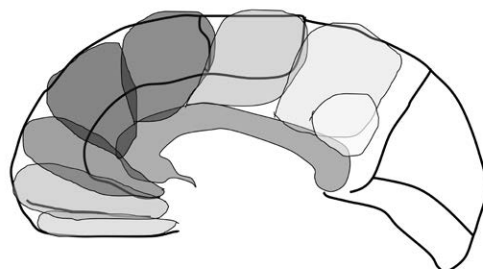
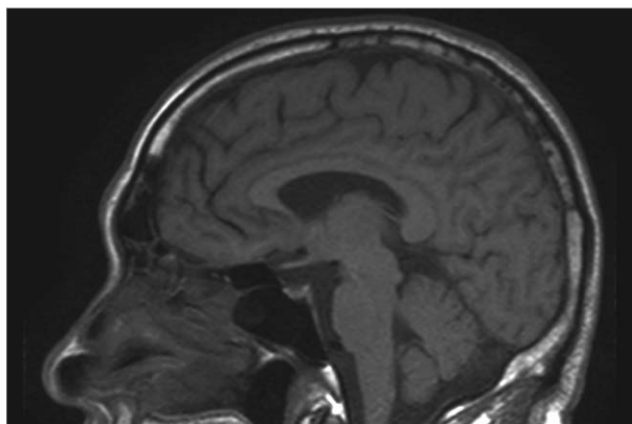
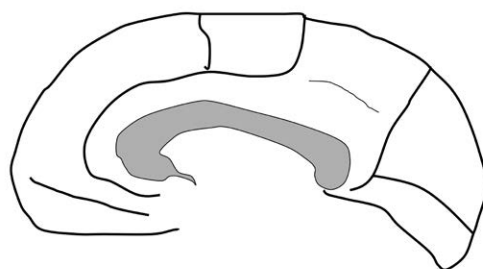
Key Words: cerebral vein, dangerous vessel, embryology, functional anatomy

2. Cerebral arterial anatomy

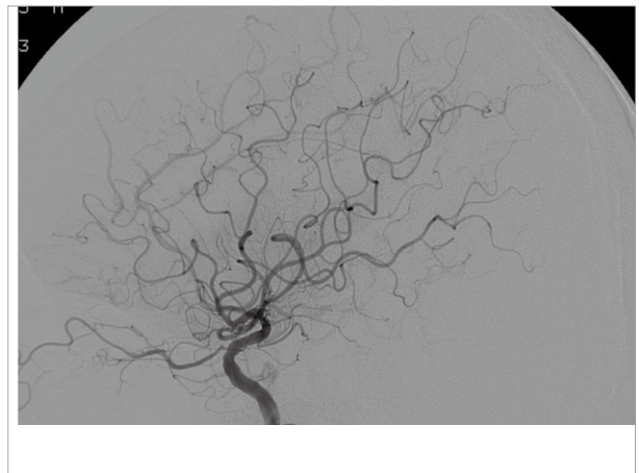
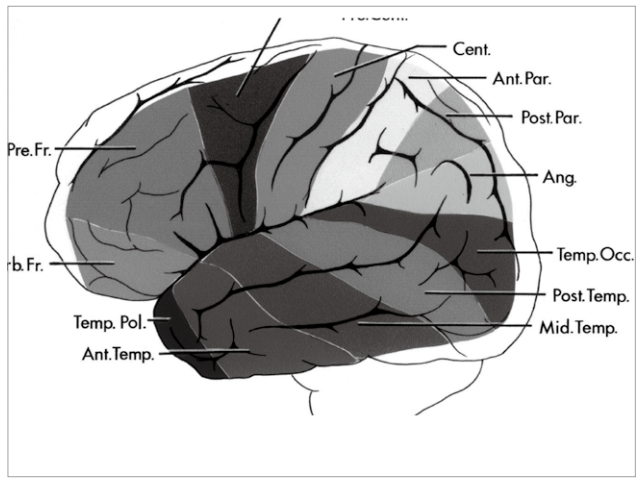
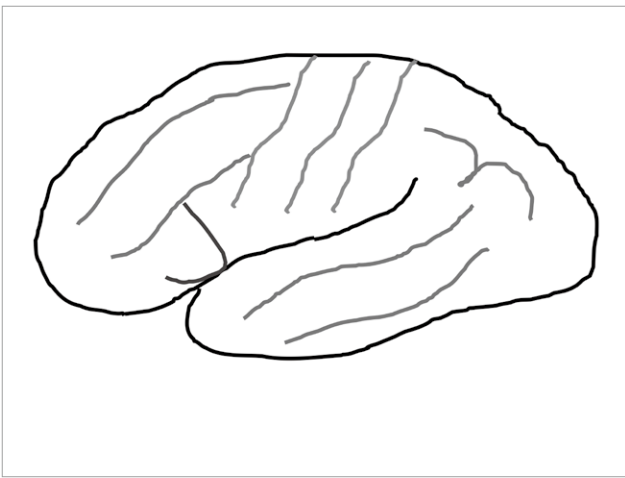
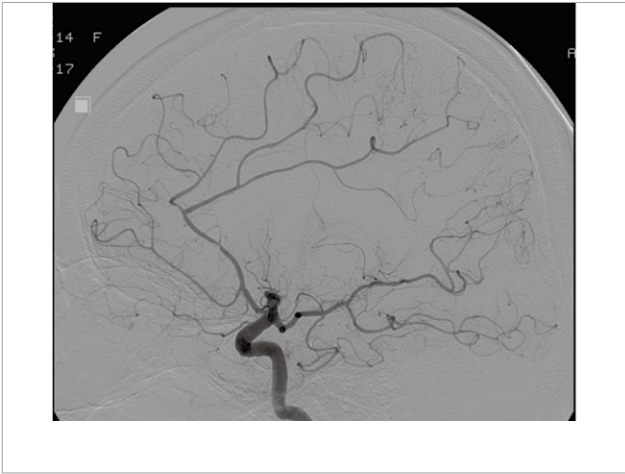
권오기
분당서울대학교병원 신경외과

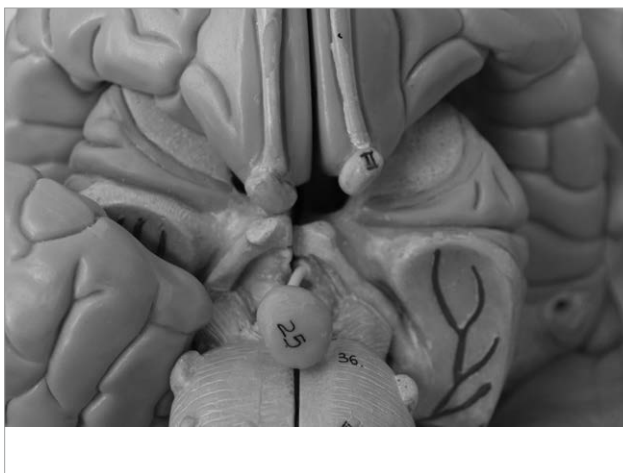
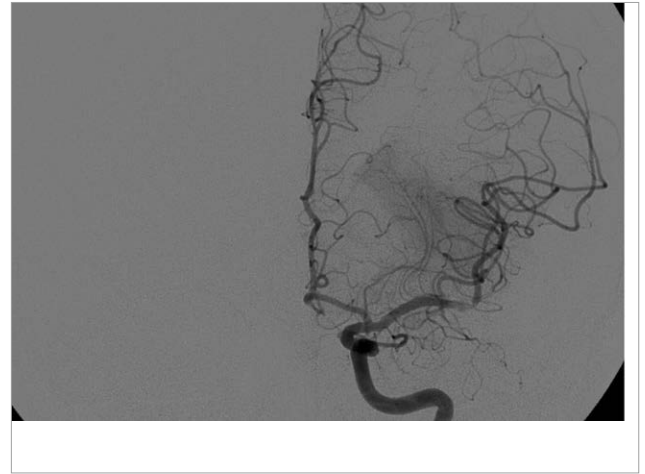
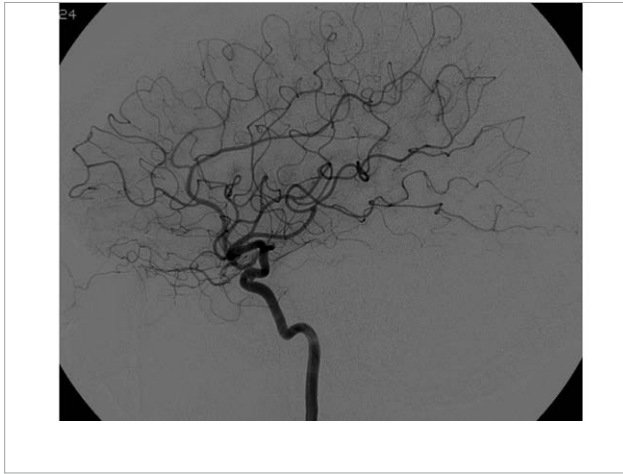
연수강좌
cerebral arterial anatomy
supratentorial arteries

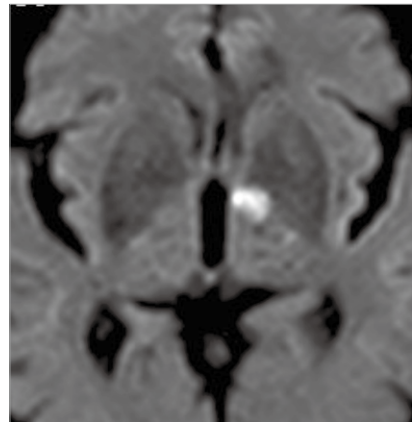
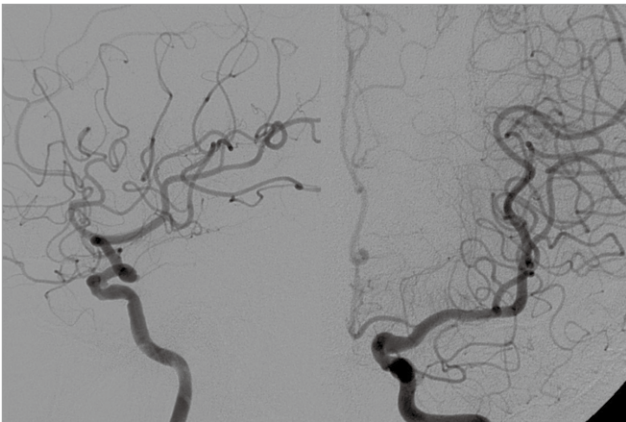
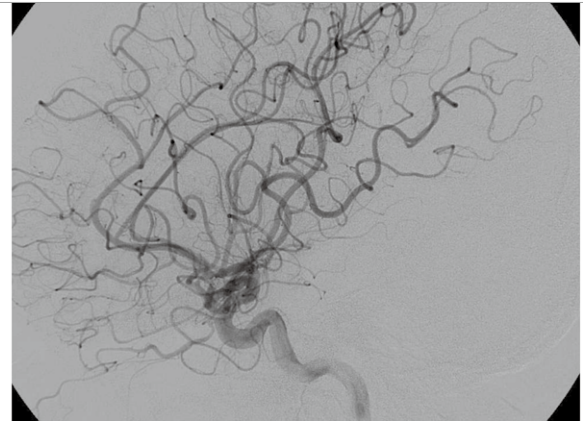
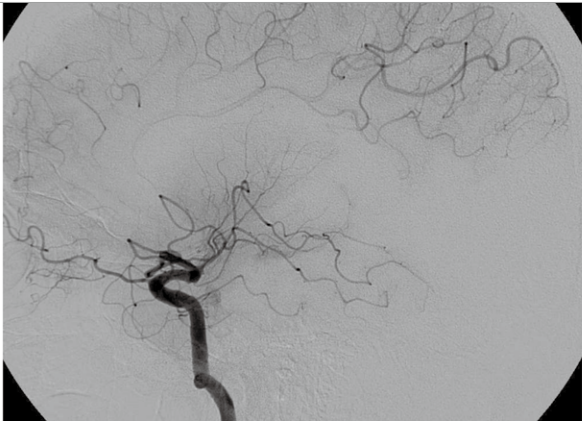
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분당서울대학교병원 신경외과



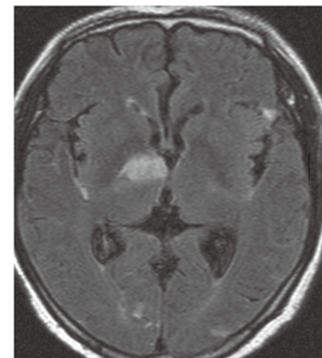
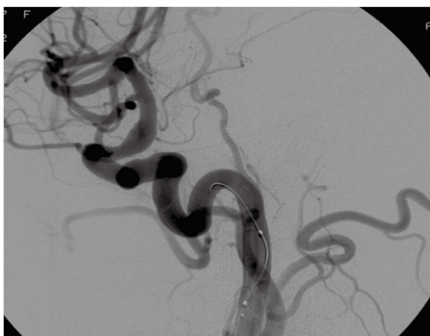
paracentral artery along marginal sulcus or paracentral sulcus

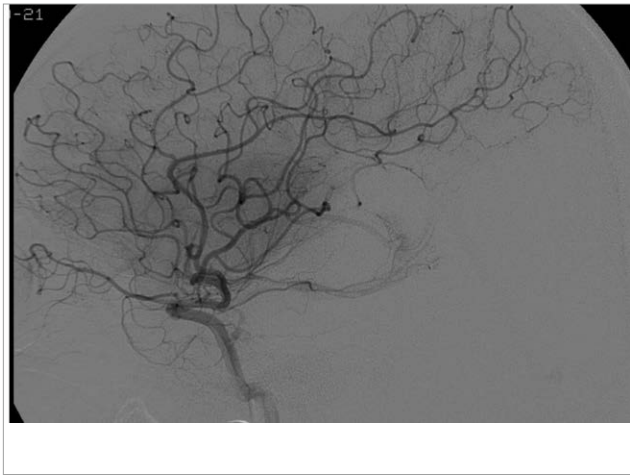
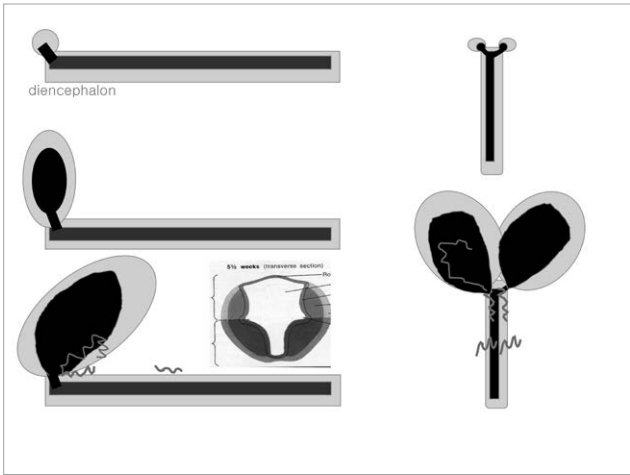
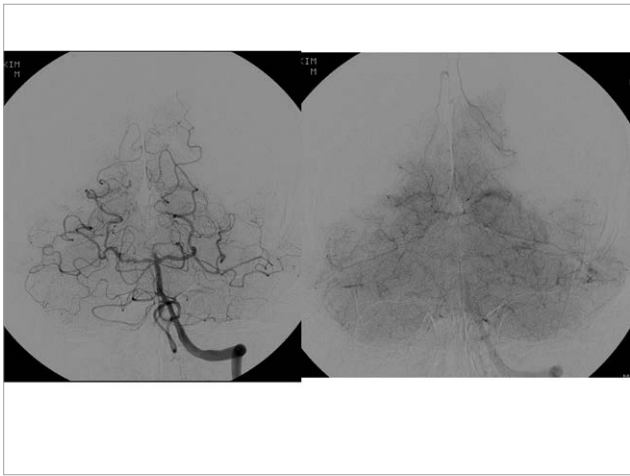
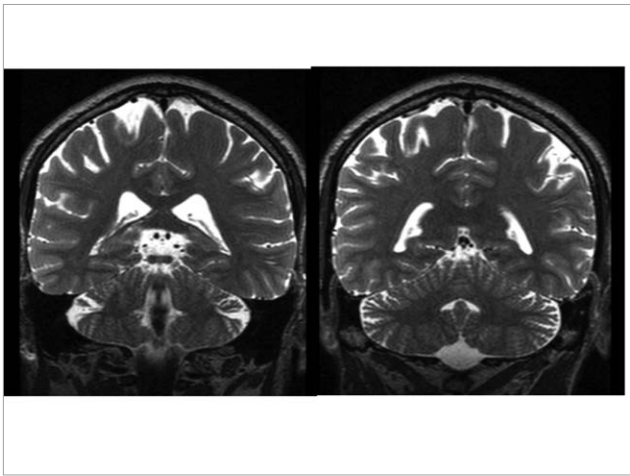
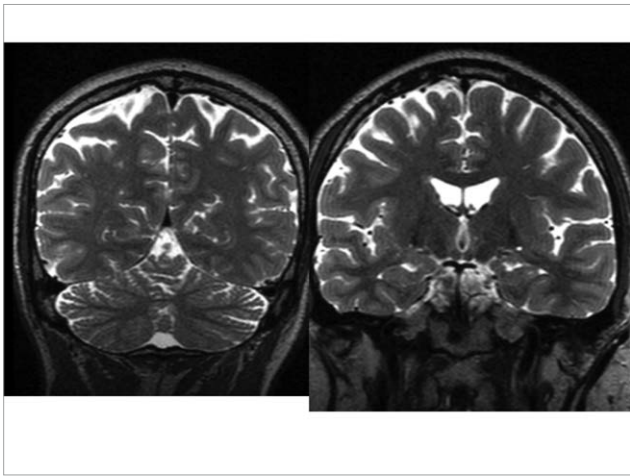






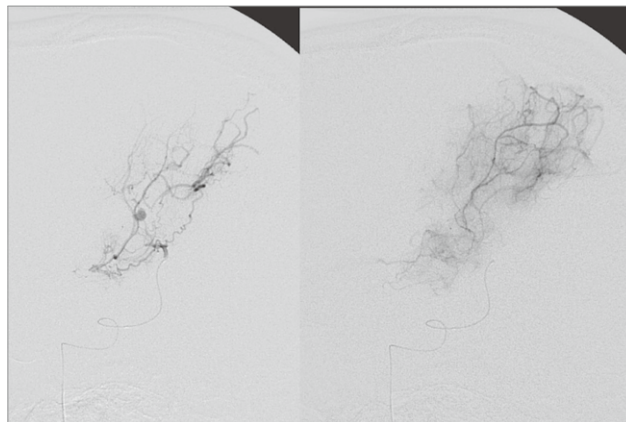
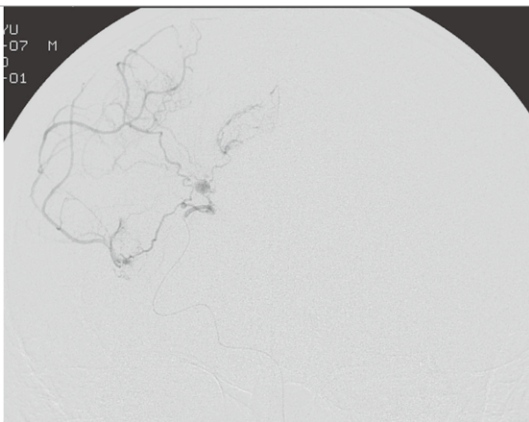
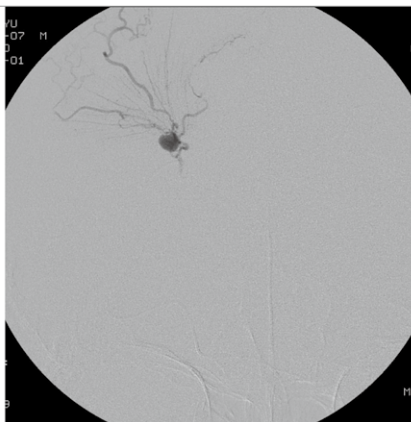
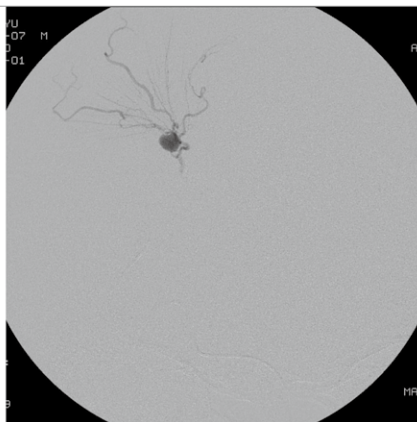
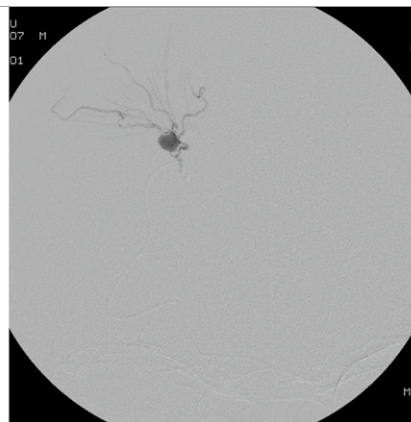
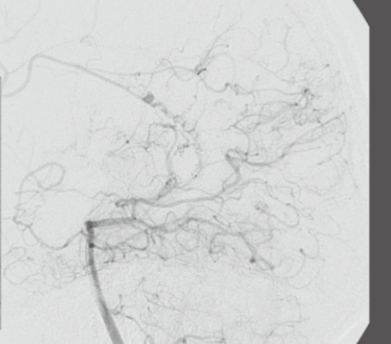
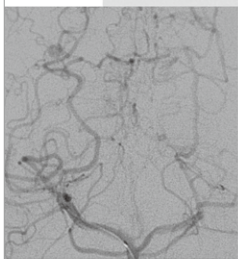
1. lethargy
2. hemiparesis
3. aphasia

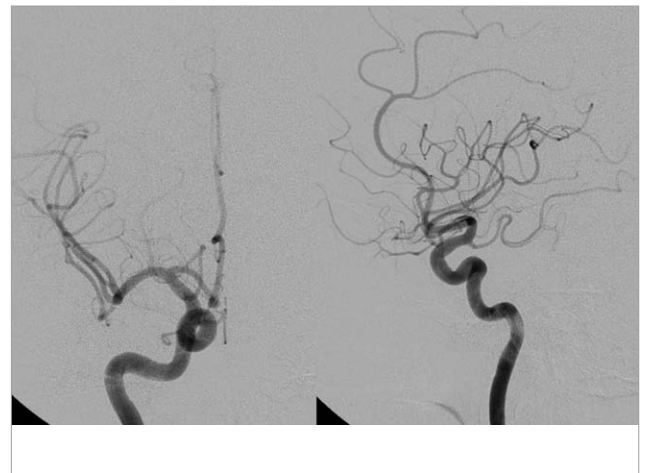
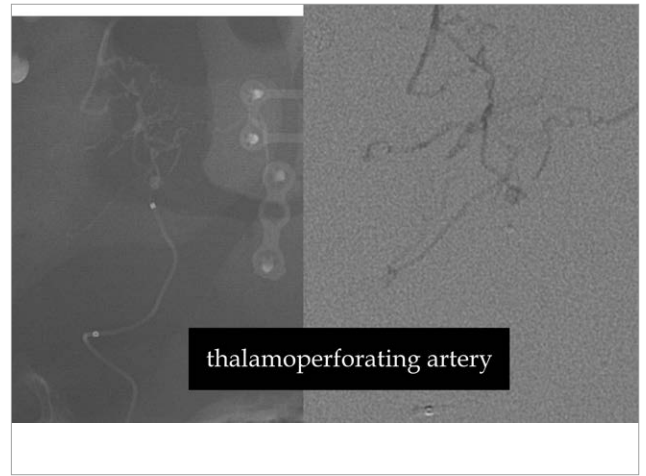
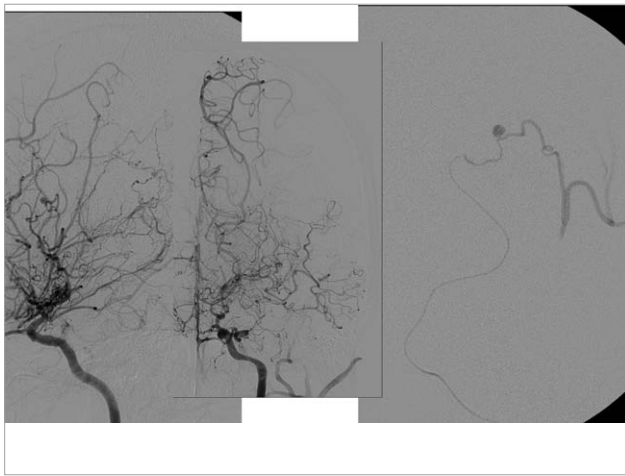
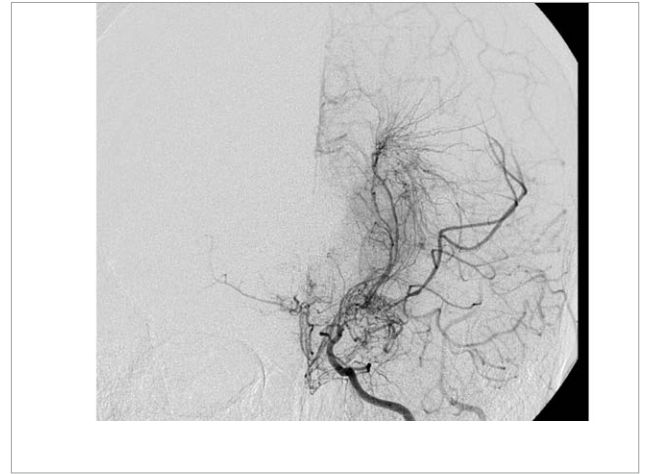
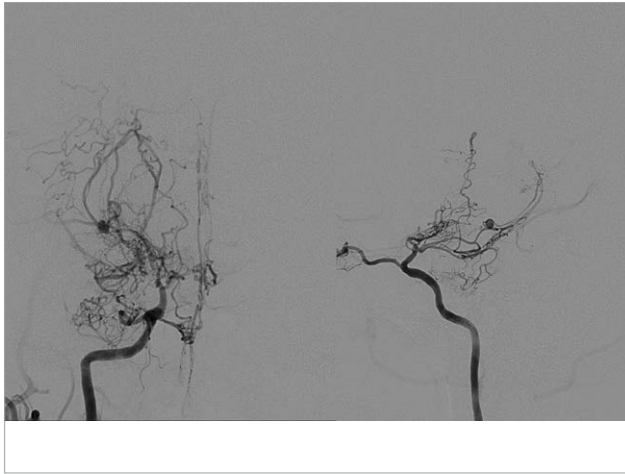


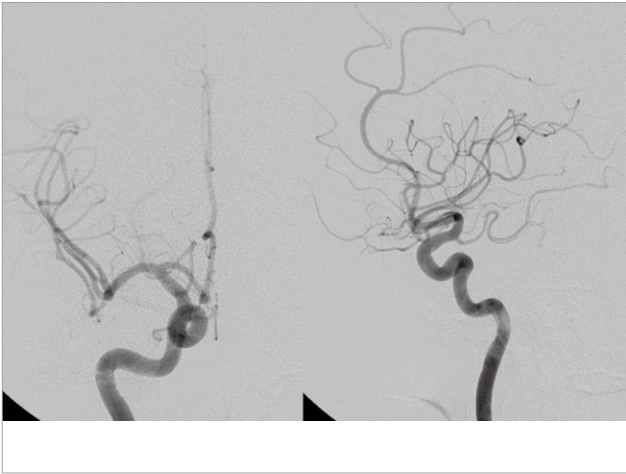




an aneurysm at collaterals from LPCA







감사합니다

대한뇌혈관외과학회 | 대한뇌혈관내수술학회

KSCVS-SKEN 합동 연수강좌

Scientific Session II: Dural AVF

좌장: 임동준 (고려대), 장철훈 (영남대)

1. Overview

조원상 (서울대)

2. Transarterial approach

임용철 (아주대)

3. Transvenous approach

박중철 (울산대)

4. Direct approach; SOV, burr hole

김동준 (연세대 영상의학과)





1. Overview

조원상
서울대학교병원 신경외과

2018.09.15 합동연수강좌

Overview of Cranial Dural ArterioVenous Fistulas

Won-Sang CHO

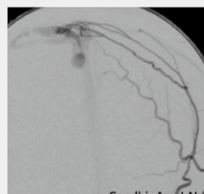
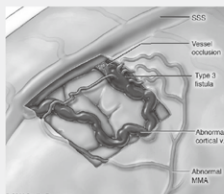
Department of Neurosurgery, SEUL NATIONAL UNIVERSITY
Seoul National University Hospital

Contents

- General perspectives
- Special considerations in some subtypes
- Treatment experience in SNUH

Definition

- Pathological anastomosis between meningeal arteries and dural/cortical veins/sinuses

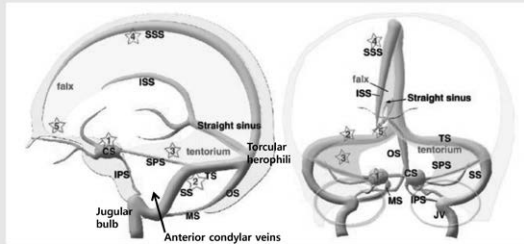


Gandhi, Am J Neuroradiol 2012

Characteristics

- 10%-15% of intracranial vascular malformations
- **Locations:** TSS, CS, SSS, ACF, tentorium, others
- **Onset:** 50-60 years > younger ages
- No predilection of sex
- Various clinical manifestations related to the locations
 - ✓ Hemorrhage
 - ✓ Non-hemorrhagic neurological deficits
 - ✓ Benign symptoms
 - ✓ Incidental finding

Locations of DAVFs



Kiyosu, Radiographics 2004

Locations of Feeders

- **Trans-osseous feeders**
OA, STA
- **ECA feeders**
MMA, AMA, artery of foramen rotundum, deep temporal artery, ascending pharyngeal artery
- **ICA feeders**
Ethmoidal artery, inferolateral trunk (ILT), capsular artery, meningohypophyseal trunk (MHT)

Pathophysiology

- **Causes**
 - ✓ Idiopathic
 - ✓ Others: trauma, infection, previous surgery, tumor, venous thrombosis
- **Hypothesis**
Progressive sinus steno-occlusion → Venous hypertension → Formation of fistulous connection between meningeal artery and sinus
- Cause of clinical manifestations
Venous hypertension, global and focal

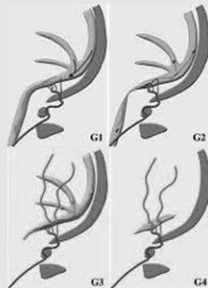
Djindjian-Merland Classification, in 1978

- **Type 1:** feeder to sinus or meningeal veins
- **Type 2:** type 1 + cortical venous reflux
- **Type 3:** feeder to cortical veins
- **Type 4:** feeder to dural or venous lakes

Lalwani Classification, JNS 1993

Kiyosu, Radiographics 2004

- **Grade 1:** feeder to sinus (antegrade)
- **Grade 2:** grade 1 + sinus reflux ± cortical reflux
- **Grade 3:** feeder to sinus (retrograde) and cortical veins
- **Grade 4:** feeder to cortical veins



Borden-Schucart Classification, J Neurosurg 1995

- **Borden type I:** feeder to sinus (antegrade)
- **Borden type II:** feeder to sinus (antegrade) and cortical veins (retrograde)
- **Borden type III:** feeder to cortical veins (retrograde)
- **Subtype a:** single feeder
- **Subtype b:** multiple feeder



Cognard Classification, Radiology 1995

- **Type I:** feeder to sinus (antegrade)
 - **Type IIa:** type I + sinus reflux
 - **Type IIb:** type I + cortical reflux
 - **Type IIa+b:** type I + sinus reflux + cortical reflux
 - **Type III:** feeder to cortical veins
 - **Type IV:** type III + venous ectasia
 - **Type V:** feeder to spinal perimedullary veins
- ↓
- ✓ Bleeding ↑
✓ ICP ↑
✓ Myelopathy ↑

Zipfel Classification, Neurosurgical Focus 2009

TABLE 2: Proposed modification to angiographic classification systems for DAVFs*

Modified Type	Borden-Shucart Type	Cognard Type	Venous Drainage	CVD	Annual Risk (%)			Treatment Recommendation
					ICH	Death		
1	I	I, IIa	dural sinus	no	<1†	0†		elective treatment for intractable symptoms
2 w/ aCVD	II	IIb, IIa + b	dural sinus	yes	1.4–1.5‡§	0§		elective treatment to prevent ICH/NHND
2 w/ sCVD	II	IIb, IIa + b	dural sinus	yes	7.4–7.6‡§	3.8§		immediate treatment to prevent ICH/NHND
3 w/ aCVD	III	III, IV, V	CVD	yes	1.4–1.5‡§	0§		elective treatment to prevent ICH/NHND
3 w/ sCVD	III	III, IV, V	CVD	yes	7.4–7.6‡§	3.8§		immediate treatment to prevent ICH/NHND

* aCVD = asymptomatic CVD; sCVD = symptomatic CVD.

Zipfel, Neurosurg Focus 2009

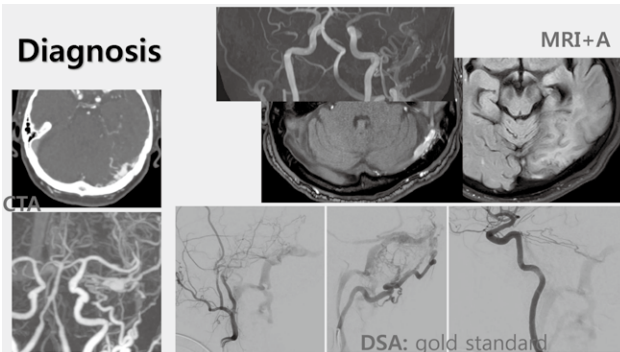
Clinical Symptoms

- **Clinical course**
 - ✓ **Asymptomatic**
 - ✓ **Benign (non-aggressive):** ophthalmologic symptoms (chemosis, proptosis, blurred vision), tinnitus, bruit
 - ✓ **Aggressive:** bleeding, neurological deficit w/ venous hypertension
- **Hemorrhage**
- **Non-hemorrhagic neurological deficits (NHND)**
 - Seizure, cranial nerve palsy, trigeminal neuralgia, dementia, Parkinsonism, cerebellar dysfunction, motor weakness, sensory disturbance, myelopathy, aphasia, apathy, apraxia, IICP signs (HA, N/V, papilledema)

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

Kiyosue, Radiographics 2004

Diagnosis



Treatments

- **Conservative Management**
 - ✓ Benign or asymptomatic lesions with low grade
 - ✓ Manual compression: carotid or ophthalmic areas
- **Surgery**
- **Endovascular intervention**
 - ✓ Approaches
 - Trans-arterial embolization (TAE)
 - Trans-venous embolization (TVE)
 - Combined embolization
 - ✓ Materials: NBCA, PVA, Onyx, PHIL
- **Radiosurgery**

Surgery

- **Indication**
 - ✓ Difficulty in endovascular app
 - ✓ No or low global venous hypertension
 - ✓ ACF DAVF, isolated/occluded CS/TSS/SSS/torcular DAVF
- **Techniques**
 - ✓ Obliteration or disconnection
 - ✓ Skeletonization
 - ✓ Exposure and direct puncture of dura or draining veins

TAE

- Superselection of feeding arteries and microcatheter wedging
- Embolization materials: coils, particles, glue, nonadhesive agents (Onyx, PHIL)
- Be able to preserve venous drainage
- Risk of infarction (brain, cranial nerve), skin necrosis, hemorrhage by vessel injury

TVE

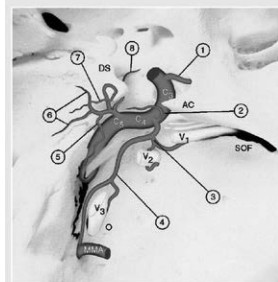
- Retrograde access into sinus or vein, even occluded routes
- Embolization materials: coils, liquid agent
- Advantageous for lesions with multiple arterial feeders with small caliber and tortuosity: CS DAVF, TS DAVF
- Important to maintain the normal venous drainage
→ unless, venous infarction or hemorrhage can occur

radiosurgery

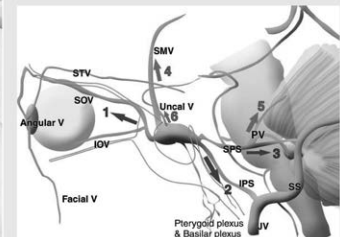
- **Indication**
 - ✓ Lesions not amenable to endovascular or surgical treatment
 - ✓ Lesions w/o CVD
 - ✓ Nonaggressive lesions w/ CVD
- **Results**
 - ✓ Still preliminary
 - ✓ Tendency of high obliteration and low complication/bleeding
 - ✓ DAVF w/o CVD > DAVF w/ CVD
 - ✓ Low grade DAVF > high grade
 - ✓ Nonaggressive DAVF w/ CVD > aggressive DAVF w/ CVS

CS DAVF

- Most common with TSS DAVF
- **Symptoms:** orbital, ocular, cavernous, cerebral Sx
- **Clinical course:** benign
Spontaneous regression (20%-50%)
- **Treatment:**
 - ✓ TVE > TAE, SRS
 - ✓ Complete obliteration rate = 80%-100%
- **Cranial nerve palsy**
 - ✓ New onset
 - ✓ Paradoxical worsening
 - ✓ Delayed occurrence



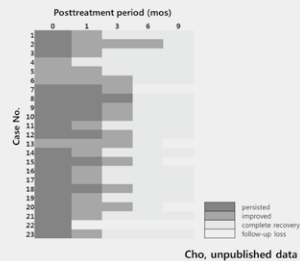
Robinson, Am J Neuroradiol 1999





Cranial Nerve Palsy After Treatment

- TVE >> TAE
- New occurrence or worsening
 - ✓ 7%-22%
 - ✓ CN6 >> CN3 and 5
- Complete recovery
 - ✓ 83%-100%
 - ✓ Within 6 months

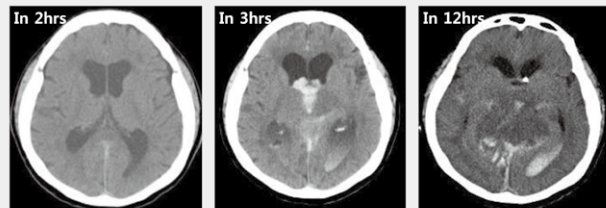


TSS DAVF

- Most common with CS DAVF
- Symptoms: tinnitus, HA, hemorrhage, NHND
- Clinical course: more aggressive than CS DAVF
- Treatment:
 - ✓ Incidence: TVE > TAE > SRS > surgery
 - ✓ Success rate: surgery > TAE, TVE > SRS
Important: preservation of normal venous drainage
 - ✓ Complete obliteration rate: 60%-100%

Compromise of Deep Venous Drainage

- ✓ F/69
- ✓ Bilateral CS DAVFs, Rt TSS DAVF, Rt jugular bulb DAVF
- ✓ Treatment history
 - TVE+TAE (2 sessions) for bilateral CS DAVF: complete oblit.
 - TVE (3 session) for recurred Rt TSS DAVF: complete oblit.
- ✓ Complication after 5th TVE
 - Severe HA and HCP in 2 hours
 - Coma in 3 hours



Anterior Condylar Vein or Hypoglossal Canal DAVF

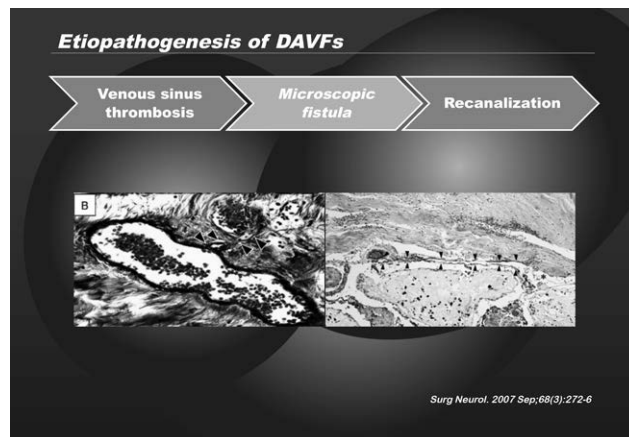
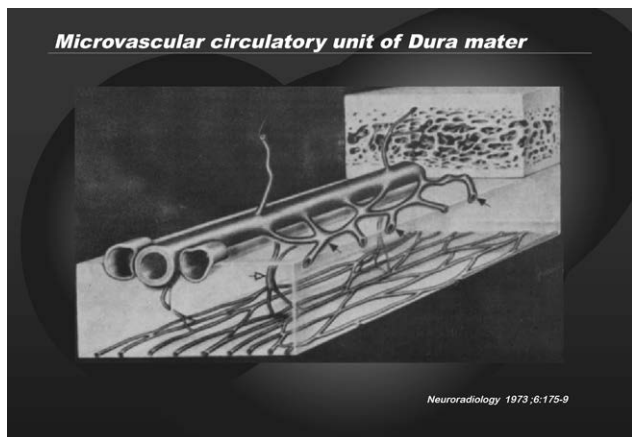
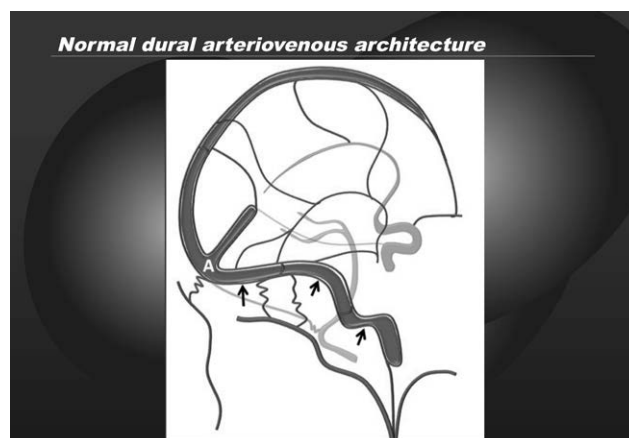
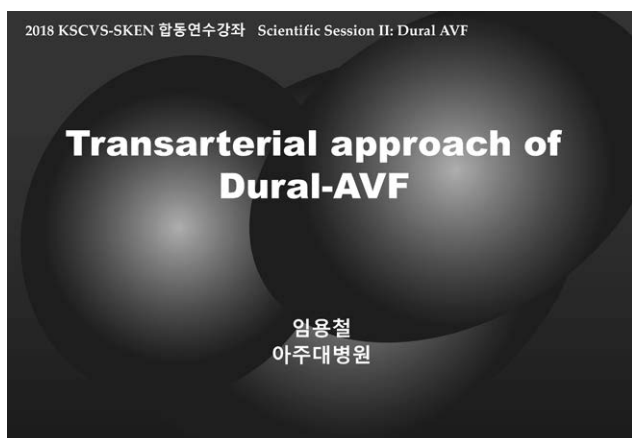
- Symptoms
 - Tinnitus, cavernous sinus syndrome, CN XII palsy
- Fistula
 - Osteolytic lesions near or at hypoglossal canal
 - DDx: Jugular bulb DAVF
- Treatment
 - TVE (venous pouch) >> TAE (APA)

SNUH data: 2004-2017

- Endovascular intervention
 - 287 patients with 331 lesions, 349 sessions
- Surgery
 - 12 patients
- Radiosurgery
 - 16 patients

2. Transarterial approach

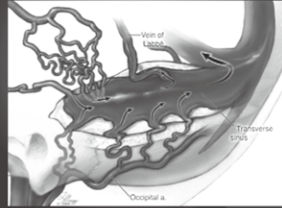
임용철
아주대학교병원 신경외과





Definition of DAVFs

- Abnormal AV shunts within the leaflets of the dura mater, usually within or near the wall of the dural sinus.
- Arterial supply mainly from meningeal / dural arteries.



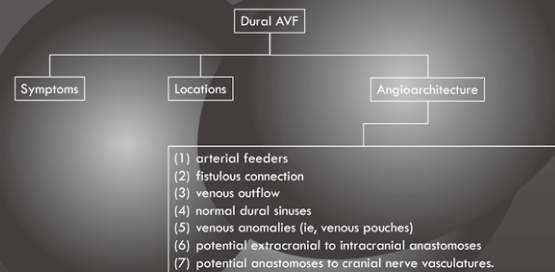
Goal of embolization of DAVFs

- An angiographic cure by obliteration of all feeders and proximal draining veins with preservation of the patency of the affected sinus
- In complex and difficult DAVFs
 - to achieve partial treatment with reversal of the aggressive type of DAVF to benign type
 - to facilitate subsequent radiosurgery or neurosurgery
- Tailored individual treatment strategy
 - Angioarchitecture
 - Natural history of the DAVFs
 - The severity of symptoms

Requirements of embolization of DAVFs

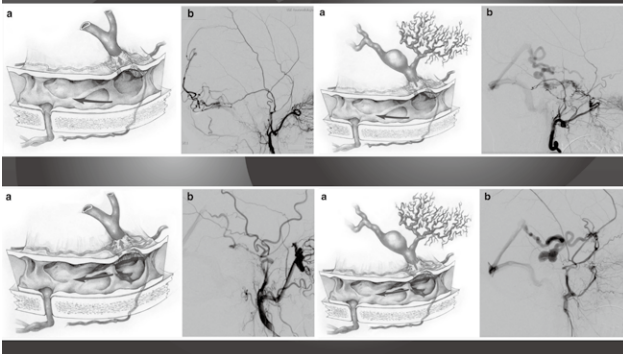
- If a sinus is occluded or severely stenosed, treatment should be directed to reestablish sinus patency
 - if that fails, sinus occlusion with angiographic cure of DAVF is considered
- If there is a functional venous pathway that was not previously occluded, it should not be sacrificed
- Treatment should not induce redirection of flow to other cortical veins

Tx. Algorithm



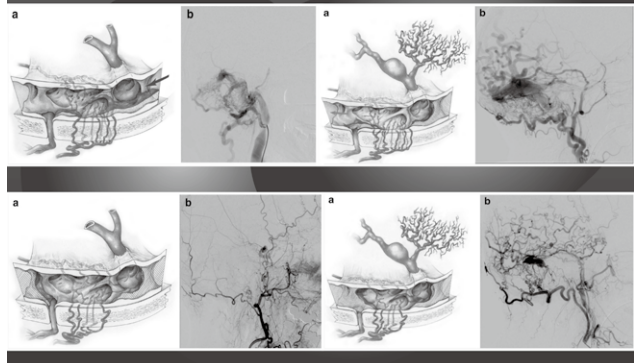
Bridging Vein Shunt

Neurosurg Rev (2015) 38:273–281

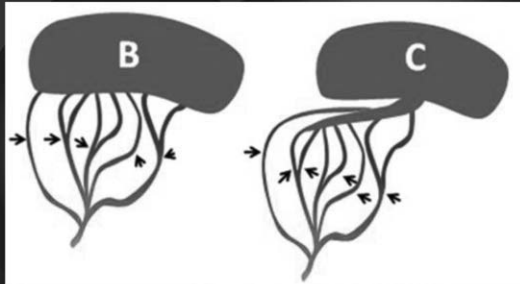


Dural Sinus Shunt

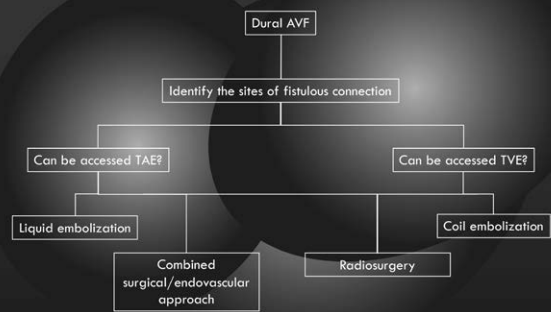
Neurosurg Rev (2015) 38:273–281



Dural fistula angioarchitecture



Tx. Algorithm



Requirements of selective TAE of DAVFs

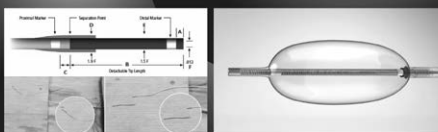
- The microcatheter be positioned into the ideal distal aspect of the feeding artery
- Delivery of the embolic material through a feeding artery to the shunt and then into the most proximal venous outlet
- Challenges
 - Multiple, small, and tortuous feeders
 - Proximal occlusion of feeding artery
 - The persistent arterial flow and recruitment of collateral blood supply
 - Dangerous anastomosis of the ECA-ICA or VA anastomoses
 - Embolization of feeders of cranial nerves
 - Embolization of material migration to the venous site - venous occlusion
 - Exacerbating venous hypertension

Liquid embolics

	PHIL TM (MICROVENTION)	Onyx TM (Covidien/Medtronic)	nBCA (various manufacturers)
Mode of Action	Precipitating Co-polymer Dissolved in DMSO	Precipitating Co-polymer Dissolved in DMSO	Polymerizing Agent
Radiopaque and Density	Iodine covalently bonded to the copolymer Moderate/Homogenous	Tantalum in Suspension High	Lipiodol Tantalum can be added
Embolitic Material	Hydroxyethyl methacrylate (nHMA)	Ethylene Vinyl Alcohol (EVOH)	N-Butyl CyanoAcrylate (nBCA)
Viscosity Range In Centistoke (cSt)	25% = 16 cSt 30% = 36 cSt 35% = 72 cSt	18 cSt 34 cSt 300 cSt (For aneurysm treatment)	Viscosity is determined by pre-procedural mixing with Lipiodol
MR or CT Artifact	Minimal MR or CT Artifact	Significant Artifact with MR or CT	No MR or CT Artifact if no tantalum
Pre-Procedure Preparation	No need for shaking Ready to use Sterile preloaded syringe	Must be shaken at least for 20 minutes Transfer from vial to syringe	No need for shaking Must be mixed/diluted
Specialized Catheter Required?	Yes (DMSO compatible)	Yes (DMSO compatible)	No
Risk of Retained Catheter	Moderate	Moderate	High

DMSO Compatible microcatheters

Microcatheter	MicroVent	Medtronic	Stryker
Detachable Tip Catheter		Apollo TM	
Dual Lumen Balloon	Scepter TM C Scepter TM XC		
Wire Directed Catheter	Headway [®] Headway [®] DUO	Marathon TM Echelon TM Rebar TM UltraFlow TM	Excelsior TM XT-17



NBCA

- a cheap, fast embolic material
- a liquid adhesive, thrombogenic
- requires a highly experienced operator with the ability to determine the appropriate concentration of NBCA (about 20–25%)
- Major disadvantages
 - short injection time (within minutes) and limited amount of NBCA mixture injection → insufficient embolization and lower cure rate (30–50%)
 - difficult to control NBCA flow after injection
 - proximal arterial occlusion in highly concentrated NBCA
 - migration to a draining vein in low-concentrated NBCA
- Advantages
 - shorter procedure lengths
 - decreased radiation exposure
 - lower cost compared with Onyx-based embolization



TAE of DAVFs with NBCA

- Candidates
 - direct RLVD : ideal candidate
 - Less commonly, RLVD + sinus (parallel venous pouch or compartmentalization)
- Standard procedure
 - transfemoral arterial route
 - NBCA-compatible microcatheter is navigated as close as possible to the fistulous connection ("wedged")
 - ✓ establishing flow arrest in the vessel
 - the microcatheter is flushed with 5% dextrose solution
 - ✓ to avoid glue precipitation within the microcatheter
 - The injection dynamic should be predicated on the catheter's distance from the fistula, flow rate, the size of the feeding a. engaged
 - Low-concentration NBCA is used to achieve distal penetration
 - concurrent injection of dextrose solution via the guide catheter during embolization can prevent proximal polymerization and premature proximal occlusion

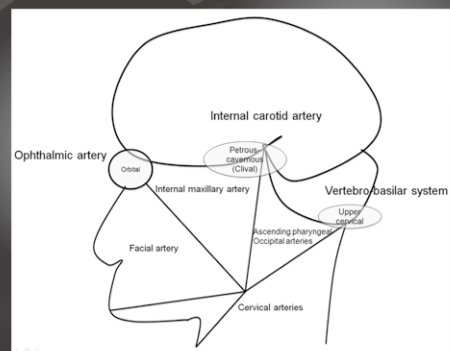
ONYX

- a nonadhesive liquid embolic system
- Consists
 - ethylene vinyl alcohol (embolic material)
 - dimethyl sulfoxide (solvent) - deliver ethylene vinyl alcohol
 - tantalum powder (radiographic visualization)
- initially introduced for AVM in 1999, DAVF in 2006 (Carlson et al.)
- advantages
 - slow, controlled injection
 - Injection of larger amounts
 - ✓ can result in filling of the fistulous network and allow for reflux into other arterial feeders in a single feeder embolization
 - ✓ relatively high morphologic cure rate of 62.5–80%
- disadvantages
 - Unknown long-term patency
 - Onyx itself may leave microchannels within the cast - allow a small fistula of flow - recurrence
 - cause significant inflammation within the vasculature (CCF)
 - ✓ Cranial nerve irritation – cranial N. palsy

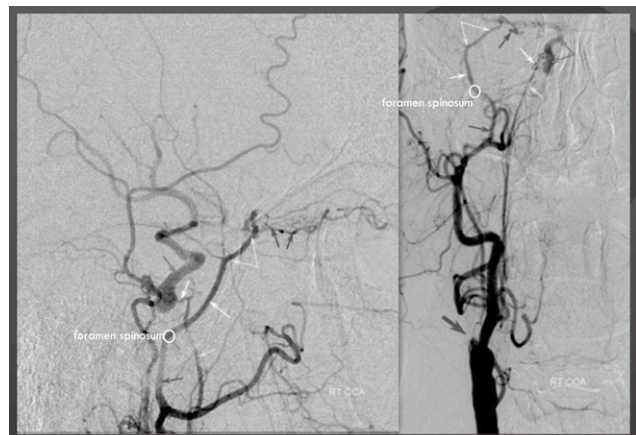
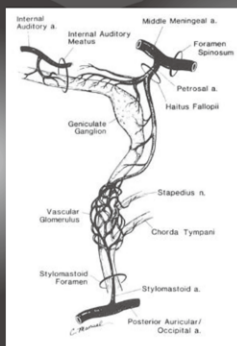
TAE of DAVFs with ONYX

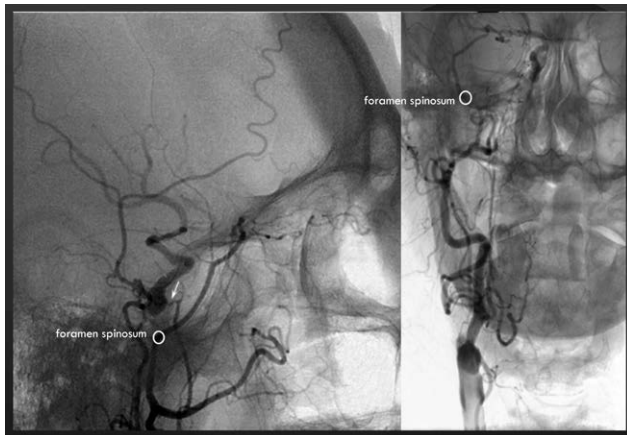
- Candidates
 - direct RLVD : ideal candidate
 - RLVD + sinus (parallel venous pouch or compartmentalization)
- Standard procedure
 - transfemoral arterial route
 - Onyx-compatible microcatheter is navigated as close as possible to the fistulous connection ("wedged")
 - the microcatheter is filled initially with the solvent DMSO
 - Proximal plug of Onyx
 - ✓ proximal flow arrest / allow distal penetration of Onyx into the fistula
 - If excessive reflux, inadvertent distal migration of Onyx
 - ✓ the injection can be halted, the roadmap renewed, and the injection restarted after 1 to 2 minutes
 - reflux should not be allowed in proximity to the level of the foramen spinosum → to avoid inadvertent compromise of the arterial supply to the trigeminal and facial nerves

Dangerous EC-IC Anastomoses & Supply to the Cranial Nerves



Facial Nerve (Geniculate Ganglion): MMA (foramen spinosum)





Cranial Nerve VI, IX ~ XII : Ascending Pharyngeal A.

- Neuromeningeal trunk is a major branch of the ascending pharyngeal that continues cephalad, but angling gently to the posterior. It has several important branches:
 - Jugular Branch:
 - ✓ Often the largest branch
 - ✓ Supplies the CN IX ~ XI
 - ✓ Also ascends along the clivus supplying the CN VI
 - Hypoglossal Branch:
 - ✓ Supply to CN XII



TAE of DAVFs with ONYX

- The most common cause of inadequate fistula obliteration
 - failure to reach the venous side
 - failure to overfill the proximal venous side
 - Onyx will initially coat the endothelial surface circumferentially before obliterating the lumen of the embolized vessel
- Disadvantage
 - prolonged fluoroscopy : Alopecia, cutaneous burn
 - High cost
 - Postprocedural pain d/t dural ischemia
- Procedural complications
 - Inadvertent microcatheter retention
 - Pulmonary embolism
 - Bradycardia d/t trigeminocardiac reflex
 - CN palsy

Transverse-sigmoid sinus DAVFs

- arterial supply
 - Dural branches of occipital artery
 - middle meningeal a.
 - ascending pharyngeal a.
 - Dural branches of vertebral a.
 - Tentorial branches of meningohypophyseal trunk
- benign DAVFs + functional antegrade TSS drainage
 - TAE with preservation of the patency of the TSS and symptomatic Tx.
- aggressive TSS DAVFs + leptomeningeal venous drainage/ reflux
 - total occlusion of the DAVFs by TAE or TVE
 - normal antegrade cortical venous drainage should be avoided
- Techniques
 - TAE of DAVFs with Onyx or NBCA
 - Transvenous balloon-assisted sinus protection during TAE
 - Double-lumen balloon microcatheter of TAE of dAVFs with Onyx
 - Transarterial balloon-assisted embolization combined with transvenous balloon protection





Cavernous sinus DAVFs

- Arterial supply
 - multiple small dural branches of the ICA and/or ECA
- Venous drainage
 - Anterior drainage → ocular symptoms (e.g., chemosis, exophthalmos)
 - Posterior drainage (Sup/Inf. Petrosal sinus) → bruit, CN palsy, reflux to cerebellar veins
 - Superior drainage (Superficial middle cerebral v.)
 - Inferior drainage (pterygoid plexus) → bruit, nasal congestion
- Tx. Strategies
 - mild clinical symptoms + no angiographic risk of hemorrhagic or nonhemorrhagic neurologic deficits → observation, manual compression, radiosurgery
 - Pial reflux + gradual loss of visual acuity / CN palsy → embolization
 - TVE : most effective
 - TAE, transorbital approach
- Concerns of TAE with Onyx
 - occlusion of blood flow to facial or trigeminal nerve
 - migration of Onyx cast to the pial artery via dangerous anastomoses → ischemic stroke, blindness

Tentorial DAVFs

- Arterial supply
 - Tentorial a. from
 - ✓ Dural branches of meningohypophyseal a. / MMA
- Venous drainage
 - drain only via the leptomeningeal vein
 - ✓ Sup. Petrosal sinus
 - ✓ pontine, perimesencephalic, and basal veins into the Galenic system
 - high risk of aggressive neurologic symptoms
 - ✓ Aggressive clinical behavior : 79~92%
 - ✓ Intracranial hemorrhage : 70% → fatal bleeding in posterior fossa
 - Tx. Goal : total fistula occlusion
- Tx. Strategies
 - TAE with Onyx / NBCA

Superior sagittal sinus DAVFs

- Arterial supply
 - dural branches of bilateral MMA
 - Anterior falcine a. of ophthalmic a.
- Venous drainage
 - frequently associated with restrictive drainage of the SSS and retrograde cortical venous drainage
 - ✓ aggressive neurologic Sx. (50%)
 - ✓ Bleeding (15~40%)
 - ✓ Dementia d/t frontal lobe venous congestion, ischemia
- Tx. Strategies
 - similar to TSS DAVFs
 - TVE with coil packing of affected SSS in nonfunctioning SSS
 - if the SSS has participated in normal venous drainage → TAE with NBCA/Onyx
- Concerns of TAE
 - usually distally and tortuously feeding a. → difficulty in navigating the microcatheter to the fistula site → insufficient embolization → recurrence
- Techniques
 - TAE of DAVFs with Onyx or NBCA
 - Transvenous balloon-assisted sinus protection during TAE
 - Double-lumen balloon microcatheter of TAE of dAVFs with Onyx
 - Transarterial balloon-assisted embolization combined with transvenous balloon protection

Anterior cranial fossa DAVFs

- Arterial supply
 - posterior / anterior ethmoidal branches of the ophthalmic arteries
 - ethmoidal branches of the ECA
- Venous drainage
 - There is no dural sinus
 - retrograde leptomeningeal - cortical venous drainage system (frontal cortical v.) only → high risk of aggressive neurologic symptoms and hemorrhage (84%)
 - Tx. Goal : complete angiographic cure
- Tx. Strategies
 - Surgical approach
 - TAE through ophthalmic a.
 - TVE
- Concerns of TAE
 - microcatheter tip is located beyond the origin of the central retina artery → avoid stroke and blindness

SUMMARY

- Endovascular embolization is the mainstay of treatment of most intracranial DAVF.
- Transarterial, transvenous, combined, or direct percutaneous approaches can be used based on the angioarchitecture, clinical presentation, location, and operator preference.
- With close examination of the angioarchitecture and individualized treatment, effective obliteration can be achieved with an acceptably low complication rate.

감사합니다.



3. Transvenous approach

박중철

울산대학교 의과대학 서울아산병원 신경외과

Transvenous approach of cranial dAVF

Jung Cheol Park

Department of Neurosurgery

Asan Medical Center

University of Ulsan College of Medicine, Seoul, Korea

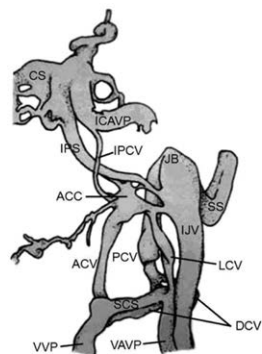
Management of cranial dAVF

✓ Understanding

- The anatomy
- The angioarchitecture
- The hemodynamic condition
- Device including the embolic material

Localization

- Cavernous sinus
- Lateral sinus
- Superior sagittal sinus
- Fronto-ethmoidal
- Tentorial
- Skull base



Hiramatsu H et al. Clin Neuroradiol 2014

Transverse-Sigmoid Sinus dAVFs

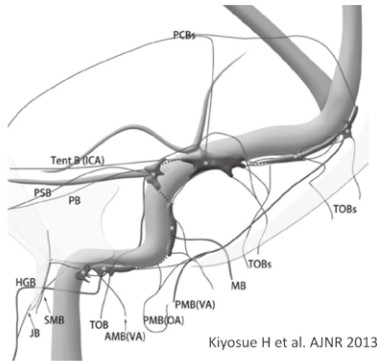
✓ transvenous embolization

- Suitable venous anatomy
 - compromised, no longer contribute to drainage of normal tissues
 - parallel venous channel
 - separate from, but in communication with the TS or SS
- Approaches
 - femoral venous access
 - direct puncture of the IJV
 - craniotomy with direct punctures of a venous sinus

✓ venous sinus angioplasty and stenting

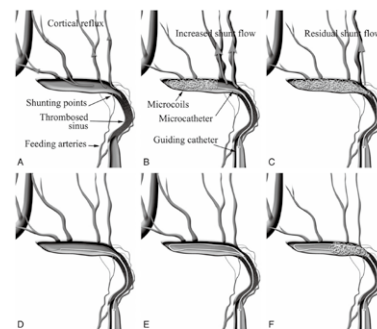


Transverse-Sigmoid Sinus dAVFs



Kiyosue H et al. AJNR 2013

Transverse-Sigmoid Sinus dAVFs

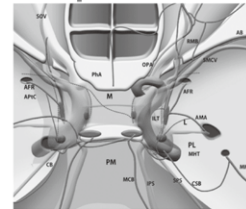


Tanoue S et al. AJNR 2012

Cavernous Sinus dAVFs

- ✓ **meta-analysis (1997)**
 - Overall success rates: TVE 78% vs. TAE 62%
- ✓ **IPS route**
 - Successful embolization: 64%
 - Thrombosed IPS can be traversed in some cases: 0.035" guidewire
- ✓ **SOV route**
 - Surgical exposure, success rate: 92-100%
 - Percutaneous puncture of the SOV
- ✓ **Alternative techniques for access**
 - Direct puncture of the IJV
 - Transfemoral facial v. approach
 - Superior petrosal sinus
 - Sylvian vein, pterygoid plexus, frontal vein

Pin-point the shunt



Kiyosue H et al. Neuroradiol 2015

Shunted pouches		Number of cases	Techniques		Immediate angiographic results	
Types	Number (average)		Selective TVE	Sinus packing	Complete occlusion	Marked regression
PM alone	1-2 (1.2)	6	5	1	6	0
PL alone	1	3	0	3	3	0
PM and PL	2-3 (2.4)	5	1	4	5	0
PM and PL and L	3	2	1	1	1	1
PM and PL and L and M	3-4 (3.7)	3	1	2	1	2
Totals		19	8	11	16	3

Tentorial (superior petrosal) dAVFs

- ✓ **Transvenous embolization**
 - Difficult or impossible
 - tortuous leptomeningeal drainage is not connected to a venous sinus

Superior Sagittal Sinus dAVFs

- ✓ **Transvenous embolization**
 - Angiographically remote

Anterior Cranial fossa dAVFs

- ✓ **Transvenous embolization**
 - Angiographically remote

Treatment strategy

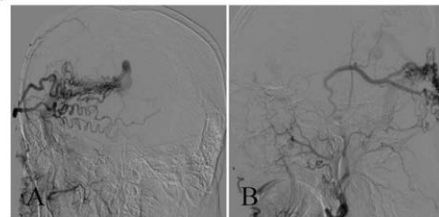
- ✓ **Treatment indications**
- ✓ **Endovascular approach first**
- ✓ **Route**
 - Location and angio-architecture
 - Transvenous approach to interrupt the fistula
 - Transarterial approach via the most accessible feeder for Onyx use
 - Combined approach
- ✓ **Palliative arterial embolization if curative embolization is not possible**
- ✓ **Surgery or GKRS**

Complications

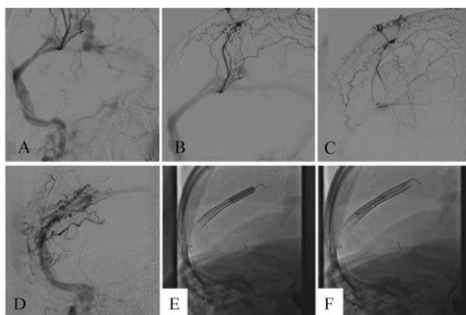
- ✓ **Inadvertent occlusion of the venous side**
- ✓ **Change of pattern of the venous drainage in partially treated cases**
- ✓ **Cranial nerve palsy**
 - Direct injury to the vasa nervorum
 - Mass effect of the embolic materials
- ✓ **Arterial embolism**
- ✓ **Trigemino-cardiac reflex**
- ✓ **Device-related complications**

Innovative approach

- ✓ **Stenting of the stenotic sinus, Use of stent-assisted angioplasty**
 - To open the sinus
 - To collapse the mural fistulae by the outward expansile force of the stent



Innovative approach



Summary

- ✓ **Cranial dAVF shows a diverse and dynamic clinical pictures**
- ✓ **Endovascular treatment strategy has changed a lot due to recent advance in imaging and intervention**
- ✓ **Understanding the angioarchitecture in depth is the most important step in the intervention procedure**
- ✓ **Both transvenous and trasarterial approaches are feasible and the choice is solely dependent on the lesion itself**



4. Direct approach; SOV, burr hole

김동준

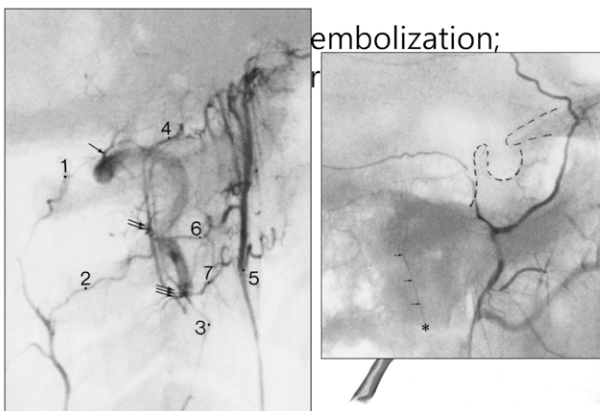
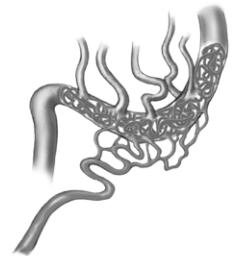
연세대학교 의과대학 세브란스병원 영상의학과

Direct puncture for treatment of DAVS; the last choice?

Dong Joon Kim
Dept of Radiology
Severance Hospital
Yonsei University College of Medicine

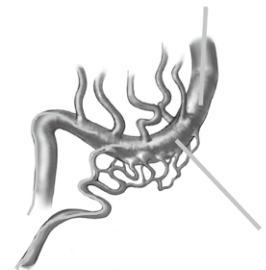
Endovascular embolization ; transvenous

- Block the initial vein / dural sinus
- Thrombogenic coils/liquid embolics
- Principles
 - No antegrade normal draining vein (nonfunctioning)
 - Arterialized pial veins
 - Complete occlusion



DAVF accessible routes

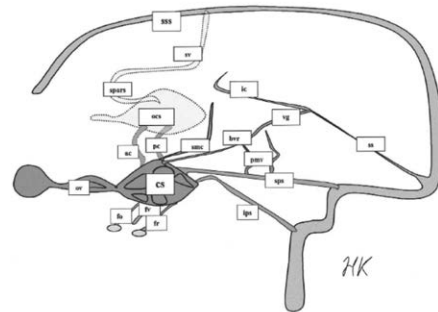
1. TVE through intact or occluded sinus
2. Collateral routes
3. Transarterial intrasinus embolization
4. Direct puncture ; percutaneous, burr hole



Direct venous access

- Indications
 - High risk DAVS
 - No accessible route
 - High risk of complications
 - Cranial nerve palsy
 - Collateral
- Methods
 - Direct percutaneous access
 - Direct surgical access

Cavernous sinus

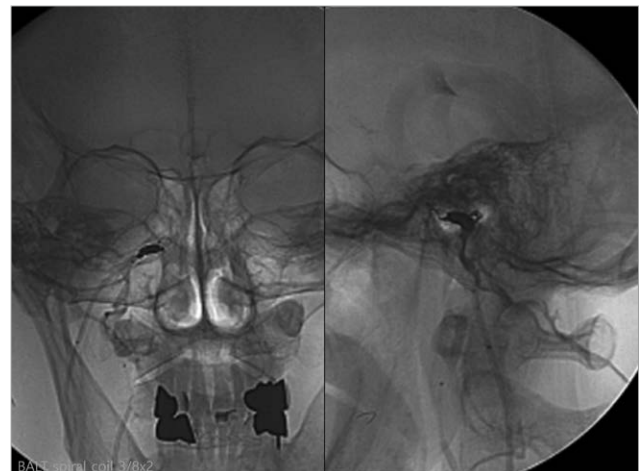
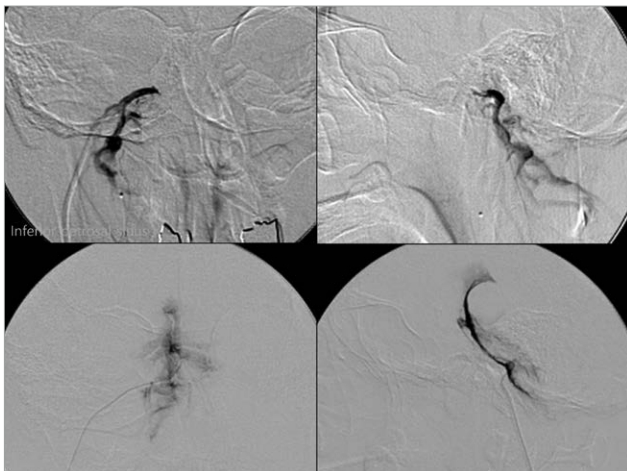
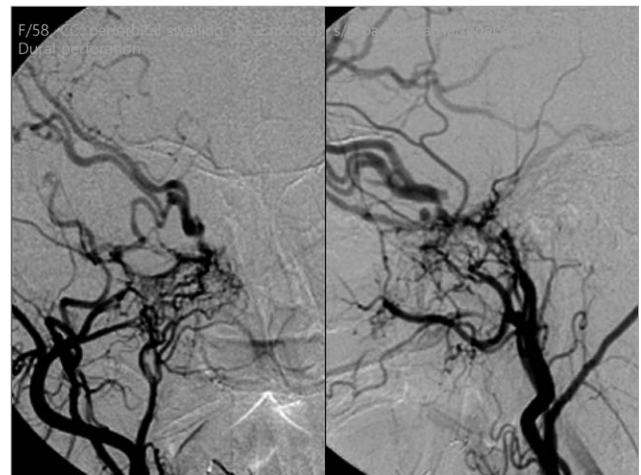


Stiebel-Kalisch Ophthalmology 2002

Cavernous sinus

- Occluded IPS (n=49)
 - Initial route
 - Transoccluded IPS
 - 34/49 cases, Selection success; 54.3% (19/34 cases)
 - Transfacial vein
 - 4/49 cases
 - Transarterial
 - 11/49 cases
 - Failed initial route
 - Radiosurgery
 - 4/49 cases
 - Direct puncture of SOV
 - 3/49 cases

RhimJK NS 2015





Dura perforation during occluded IPS catheterization

Case	Sex	Age	Duration of sx	Perforation site	Sinus patency	Cause of perforation	Tx	Outcome
1	F	55	3 mo	IPS	Occluded	Microwire	Coil	Good
2	F	68	1 mo	CS	Occluded	Microwire	Coil	Good
3	F	58	2 mo	IPS	Occluded	Microwire	Coil	Good

Sx: symptoms, mo: months, IPS: inferior petrosal sinus, CS: cavernous sinus, Tx: treatment

Direct surgical SOV access

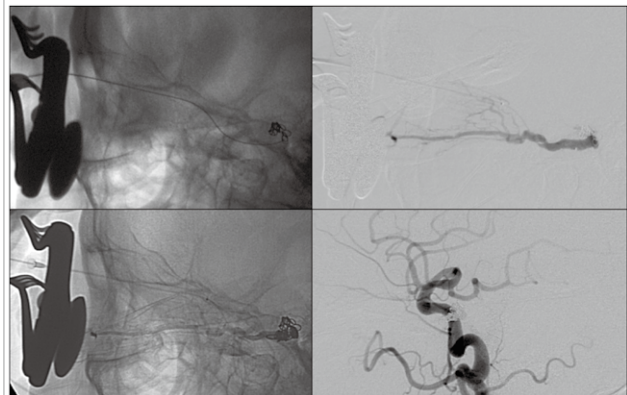
- Technique
 - Eyelid cutdown, SOV exposure
 - 5F micropuncture system/ coaxial dilator catheter; *fixation of external catheter under roadmap guide*
 - Microcatheter access
- Indication
 - Facial vein, IPS, IJV thrombosis
- Contraindication
 - No dilated SOV
 - location deep in orbit
- Risks
 - Retro-orbital hematoma
 - weak venous wall related to age of shunt
 - Damage to anterior orbital structures
 - Cranial nerve, Intraocular muscles, Globe

Quinones AJNR 1997
LeeJW ActaNeurochir 2008
Jiang JNIS 2012
Uflacker Radiology 1986

Direct surgical SOV access

- Quinones AJNR 1997
 - N=13
 - Coil embolization
 - Complete occlusion & symptom improvement (12/13 cases)
 - Transient abducens palsy (n=1), *Silk suture granuloma* (n=1)
- LeeJH ActaNeurochir 2008
 - N=7, no access fail
 - Coil embolization
 - Complete occlusion & symptom improvement (n=7)
 - Transient neuralgia (n=2), transient brainstem congestion (n=1)
- Jiang JNIS 2012
 - N=9, no access fail
 - *Onyx & coils*
 - Complete occlusion & symptom improvement (n=9)

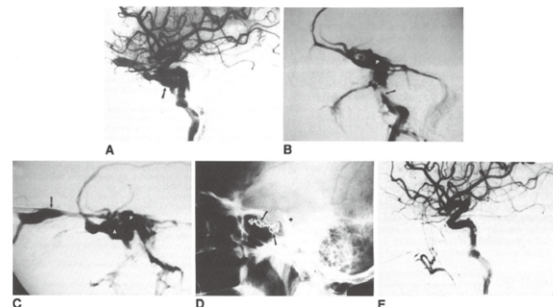
F/65 cDAVF direct puncture of SOV



Direct percutaneous SOV access

- Technique
 - Small incision on upper eyelid
 - 16G needle puncture under roadmap or real time DSA
 - Blood return
 - 035 wire
 - 5F catheter
- Advantage
 - No surgical cutdown
 - Cosmetic
- Risk
 - Intraorbital/eyelid hemorrhage

Bendorff Neurorad 2001
TengM AJNR 1988

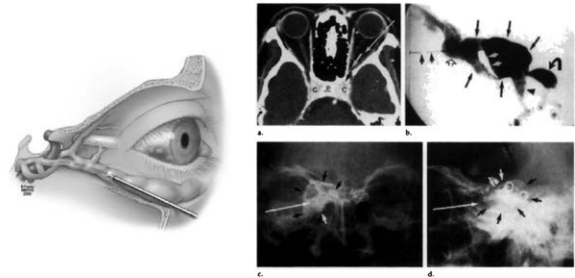


TengM AJNR 1988

Direct percutaneous IOV/CS access

- Technique
 - 6 inch 18G radial artery-line catheter inferolateral to globe under roadmap or fluoroscopic guidance
 - Advance along inferior orbital rim
 - Target SOF
 - Oblique projection for AP tube; separate SOF from optic canal
 - Blood return
 - Microcatheter & microwire
- Risk
 - Orbital hemorrhage
 - Nerve damage
 - ICA laceration
 - Globe puncture

TengM Radiology 1995
White AJNR 2007

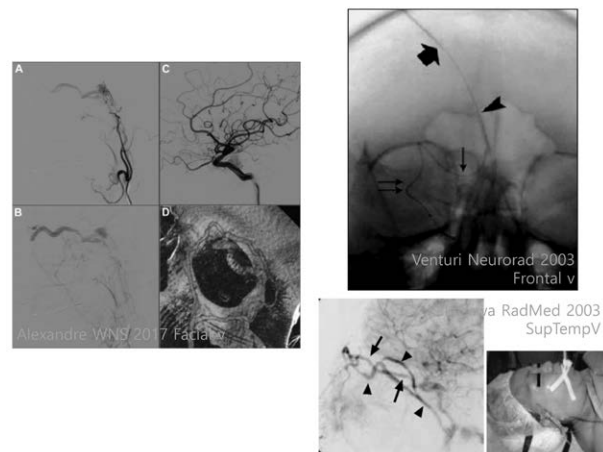


TengM Radiology 1995
White AJNR 2007

Percutaneous superficial vein access

- Transfacial vein
 - US guide, 18G cannula, 010 microcatheter
 - Less risk related to venous rupture
- Transfrontal vein
 - 18G cannula, 010 microcatheter
 - Maximal posterior flexion/compression of jugular vein for venous distension
- Trans-superficial temporal vein
 - 18G IV needle, microcatheter
 - Percutaneous

Alexandre WNS 2017
Venturi Neurorad 2003
Kakezawa RadMed 2003



Transcranial burr hole access

- Technique @ Severance
 - Landmark for involved sinus & puncture site under DSA guide
 - Supine position for SSS, lateral decubitus for TS
 - Skin incision & burr hole @ puncture site
 - Target sinus puncture
 - (1 week after craniectomy to allow healing of subQ fascia)
 - 18G angiocatheter (micropuncture set)
 - Sinography
 - Fix sheath
 - Advance microcatheter & wire to target under roadmap or DSA guide
 - Embolization with coils (liquid embolics)

Houdart JNS 2002

Transcranial burr hole access

- Indication
 - Isolated DAVS with CVR
 - No other access route available
- Risks
 - Hemorrhage
 - Patient logistics; hybrid OR

ParkSH JCBVascEndoVascNS 2017
Houdart JNS 2002
Ushikoshi SurgNeurol 2002
Endo JNS 1998



Transcranial burr hole access

*Clinical and radiological characteristics of patients treated by transcranial approach**

Case No.	Age (yrs), Sex	Symptoms	Location of Fistula	Cause of IIV Disconnection	Failed Interventions Before TCA	Embolic Agent(s) Used in TCA	Last Angio Follow Up (mos)
1	21, F	CI & headaches	SSS	spontaneous thrombosis	1 TVA	coils	14
2	70, M	temporal hematoma	rt TS	spontaneous thrombosis	2 TAA, 1 TVA	coils & glue	18
3	70, M	CI & seizures	SSS	spontaneous thrombosis	1 TVA	coils	15
4	32, M	CS & headaches	lt TS	previous coil embolization	1 TVA, 1 TAA	glue	9
5	46, M	CS & headaches	rt TS	previous coil embolization	1 TVA, 1 TAA	glue & coils	3
6	29, F	CI & seizures	SSS	spontaneous thrombosis	1 TAA, 1 surgery	glue	—
7	64, F	cerebellar infarction	rt SPS	spontaneous thrombosis	1 TAA	glue	—
8	58, F	headaches & tinnitus	lt TS	spontaneous thrombosis	1 TVA	coils	—
9	47, F	tinnitus	rt TS	spontaneous thrombosis	1 TVA	coils & glue	—
10	61, F	CI	SSS	spontaneous thrombosis	1 TAA, 1 TVA	coils	—

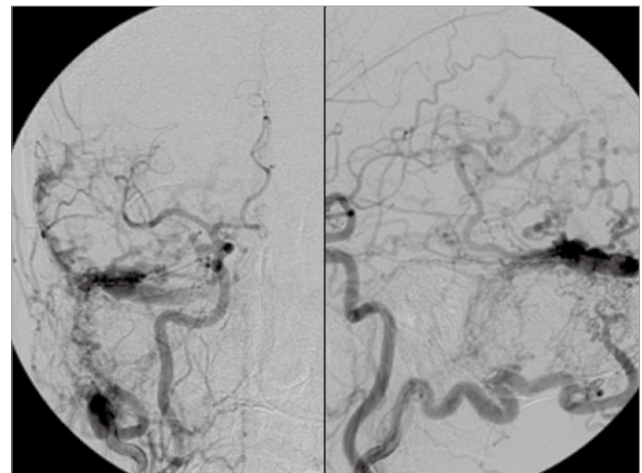
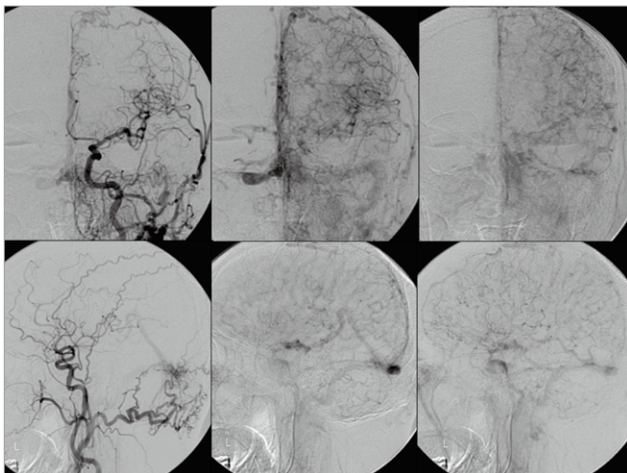
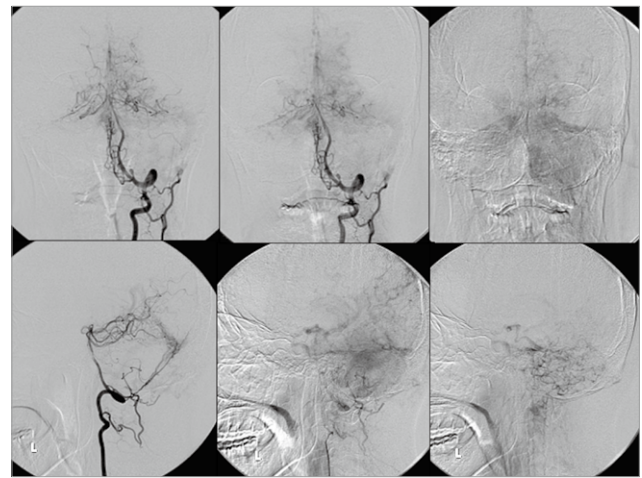
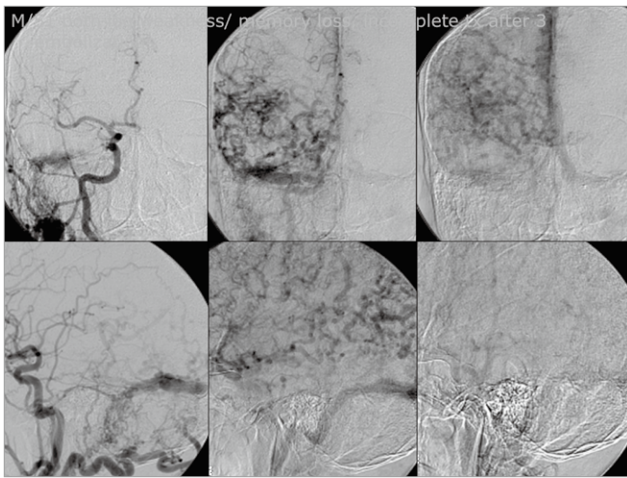
* Angio = angiographic; CI = cognitive impairment; CS = cerebellar syndrome; TAA = transarterial approach; TCA = transcranial approach; TVA = transvenous approach; — = not applicable when paper was written.

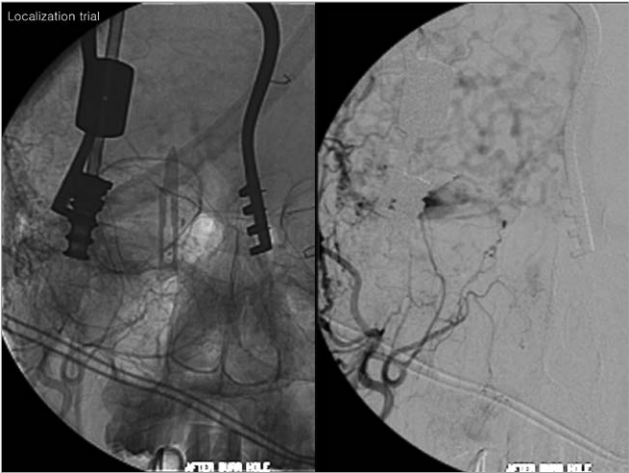
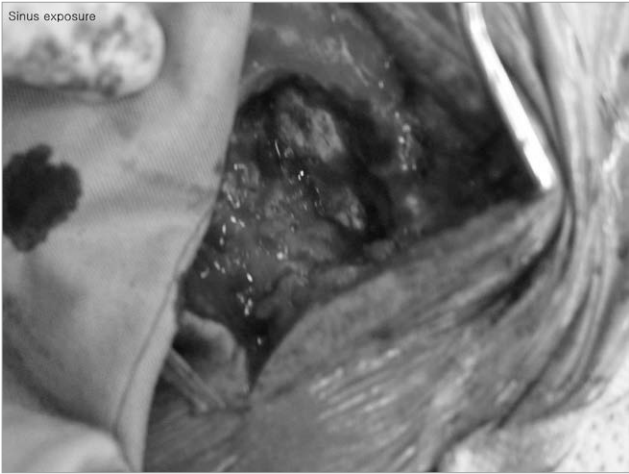
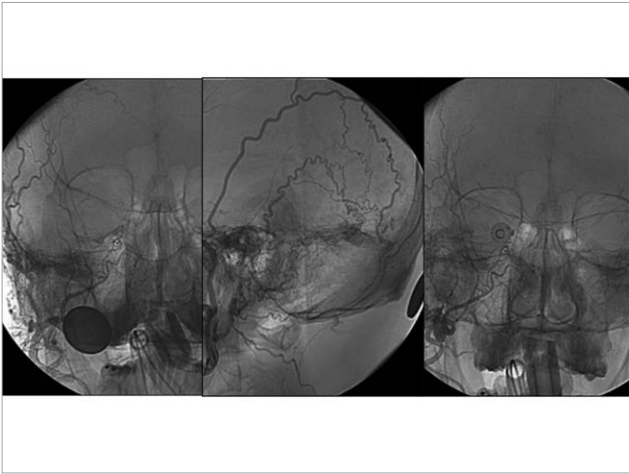
- Craniectomy in OR, Embolization in DSA suite
- Enlargement of craniectomy; 7/10 cases
- Complication (n=1); transient hemiparesis from parasagittal SDH after failed sinus puncture
- Complete occlusion (n=10)

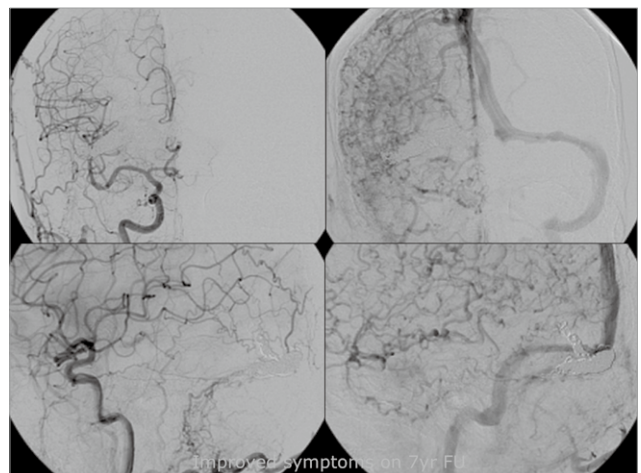
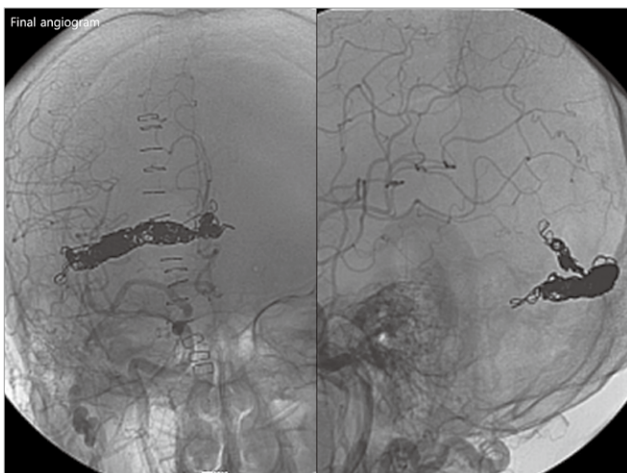
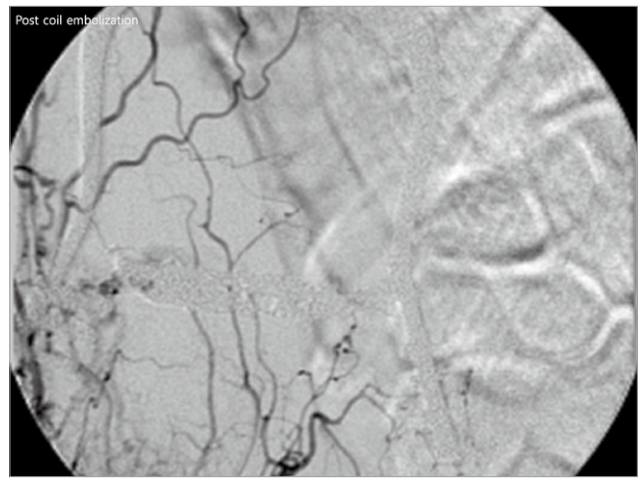
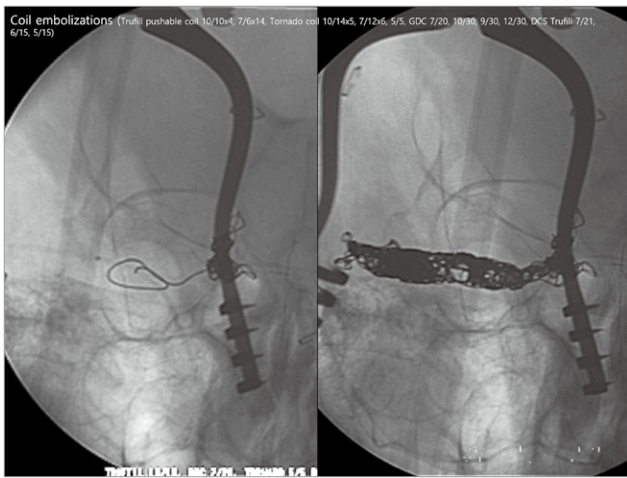
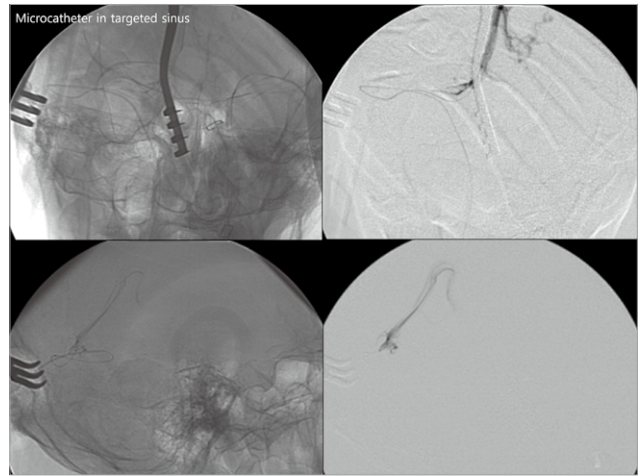
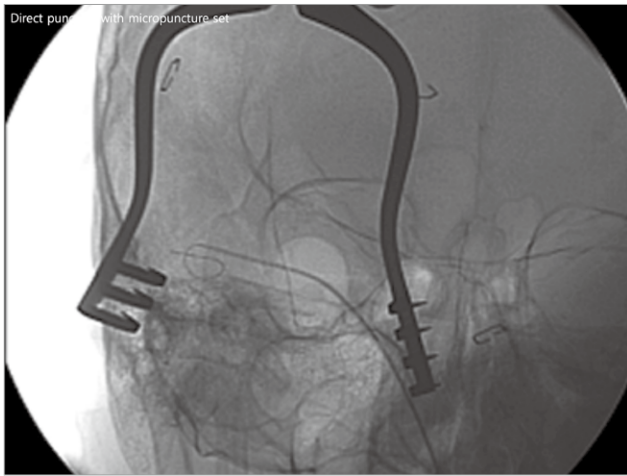
Houdart JNS 2002

Transcranial burr hole access Severance experiences

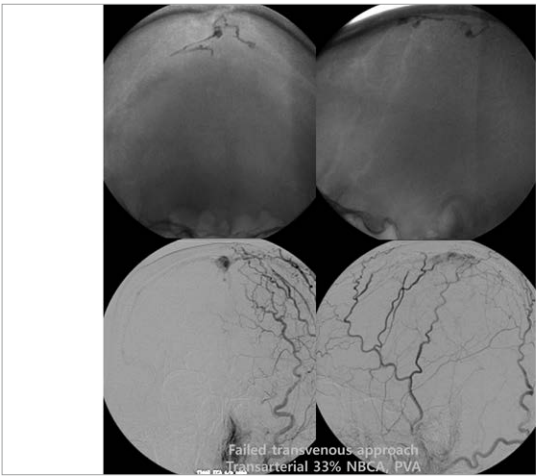
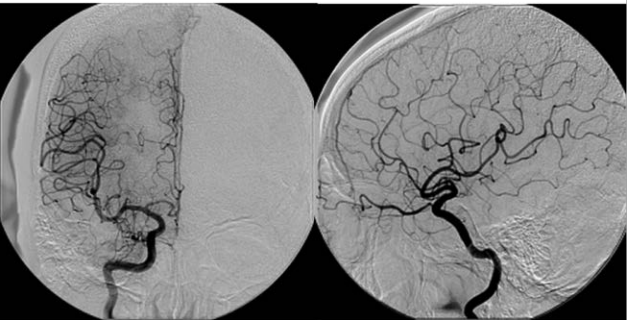
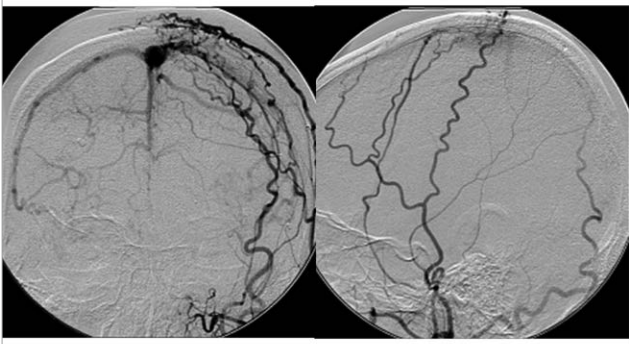
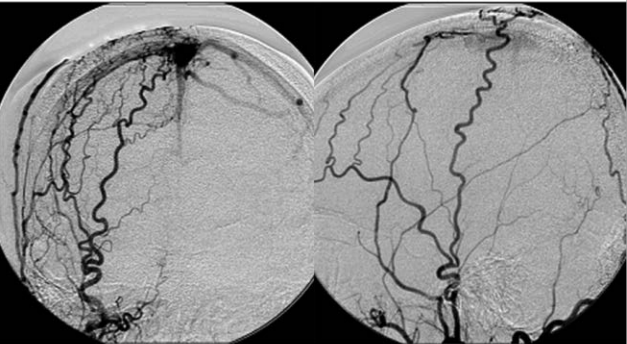
Case	Sex	Age	Presentation	DAVF Location	Direct puncture lesion	Embolization Materials	Angiographic outcome	Clinical outcome
1	M	50	Gait disturbance, memory impairment	Rt TS, torcula	Right TS, torcula	Coils	Complete	Complete resolution
2	F	68	Mental change, Headache, Dysarthria	Left TS, Cavernous sinus	Left TS	Coils	Complete	Complete resolution
3	M	60	Headache	SSS, Right TS	SSS	Coils	Near complete	Complete resolution
4	M	61	Left side weakness, Headache, Dementia	SSS	SSS	Coils	Near complete	Complete resolution
5	F	53	Aphasia s/p TAE outside hospital	Left TS	Left TS	Coils	Complete	Mortality
6	F	2	Exophthalmos, hydrocephalus, scalp lipoma	Left TS, SSS	Left TS	Coils	Near complete	Symptom resolution



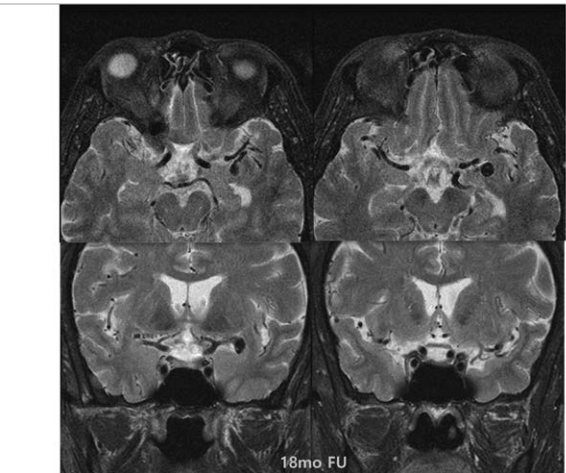




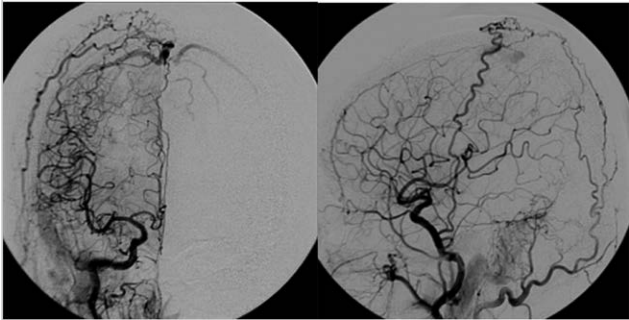
M/60 Gait disturbance, memory impairment for 6mo

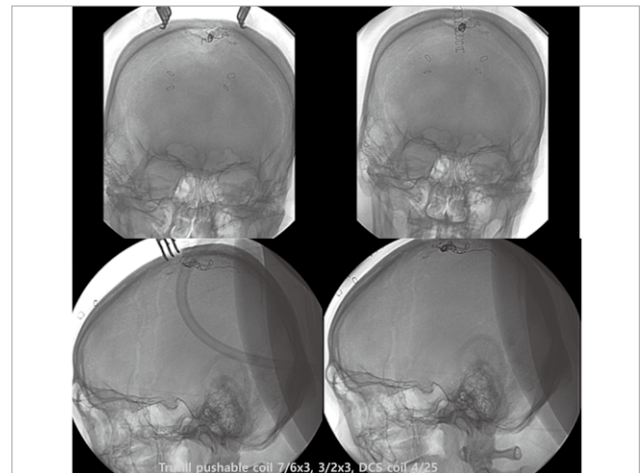
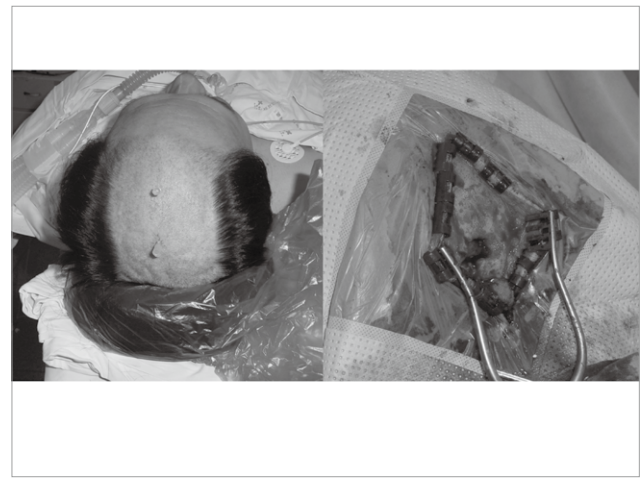
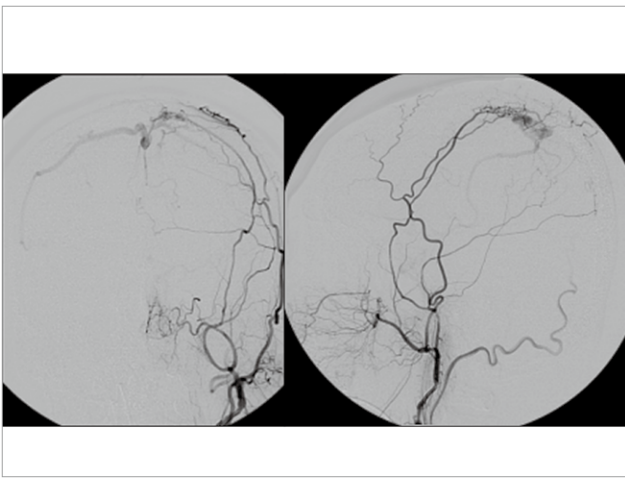
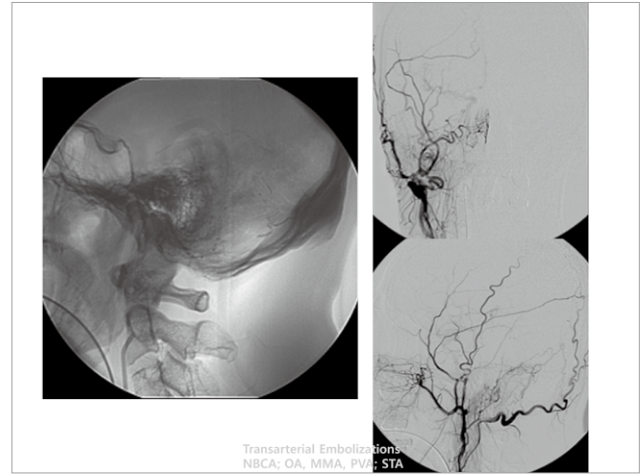
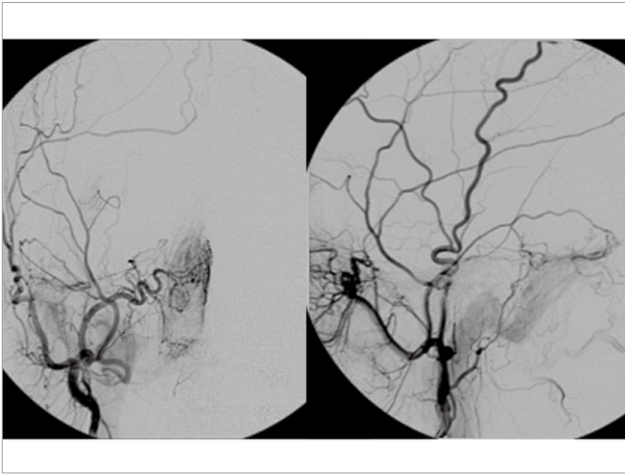


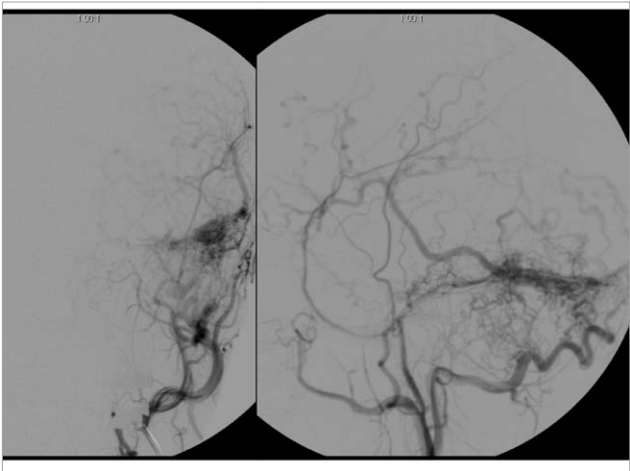
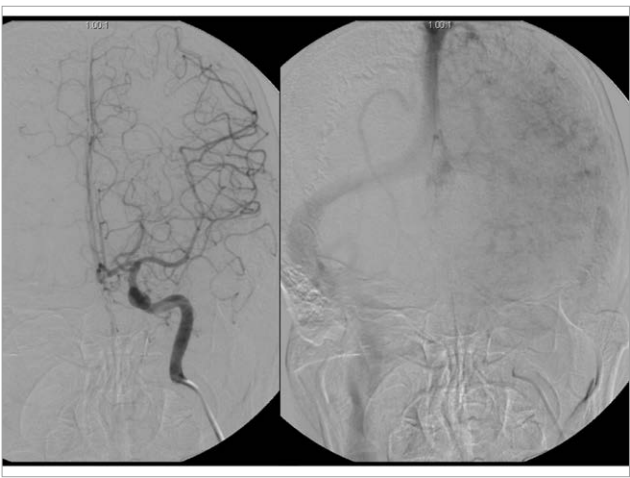
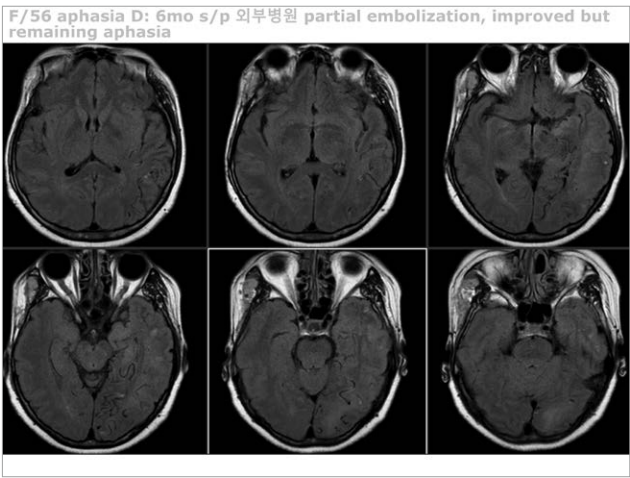
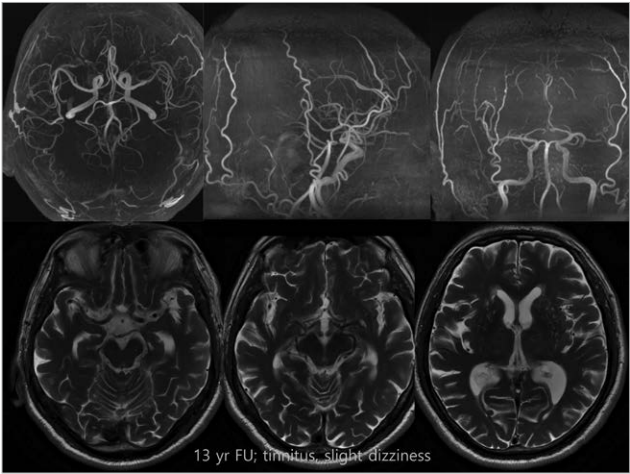
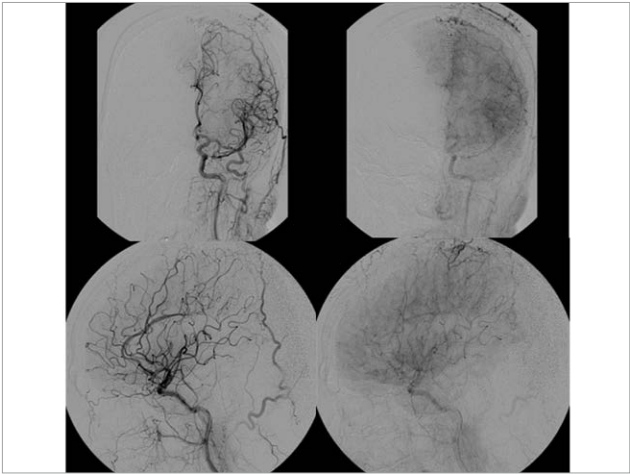
Failed transvenous approach
Transarterial 33% NBCA, PVA

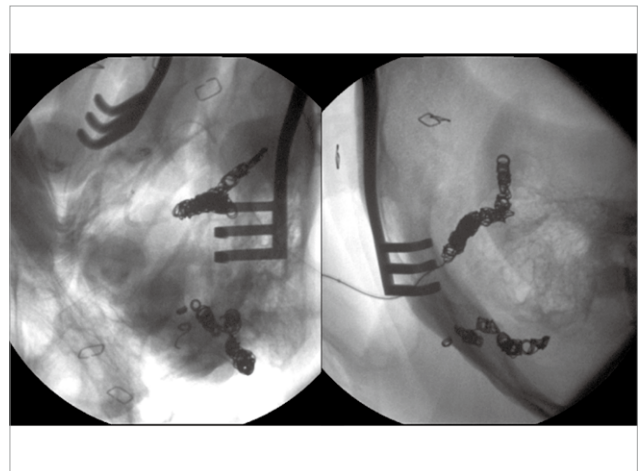
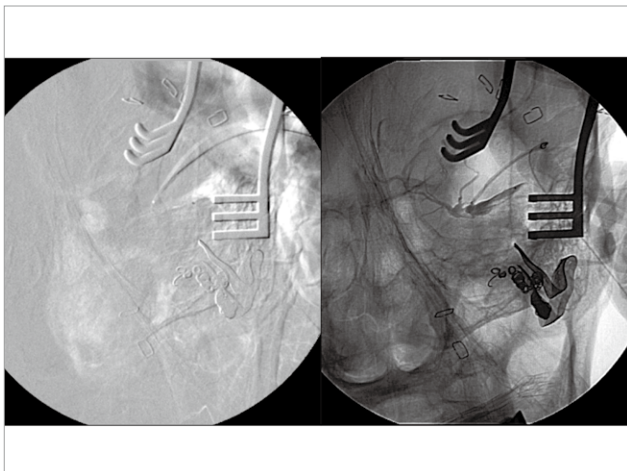
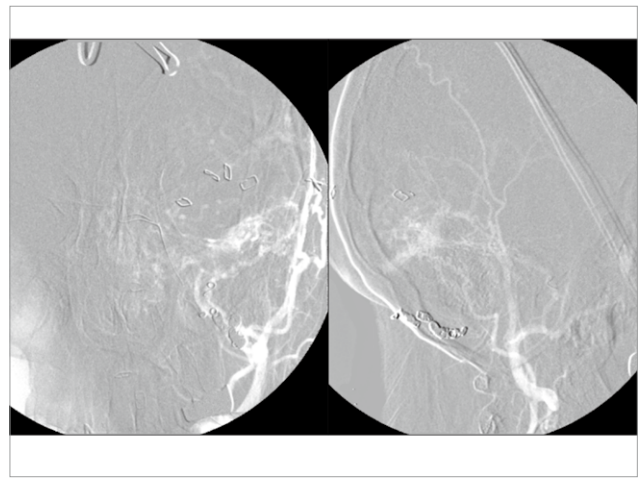
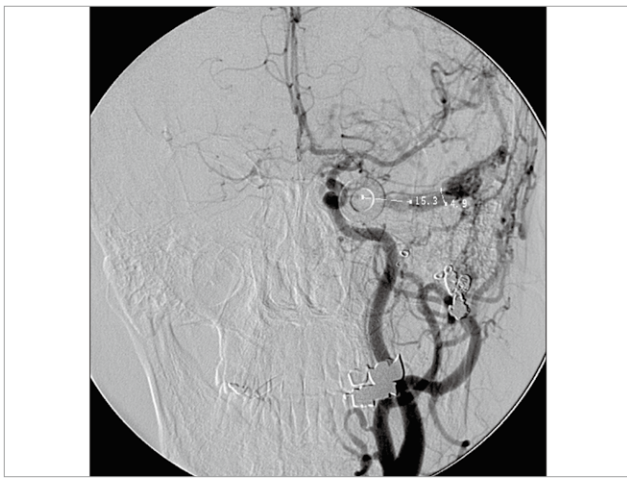
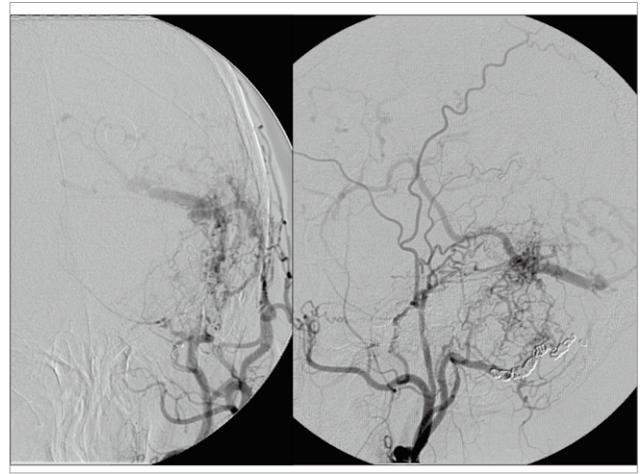
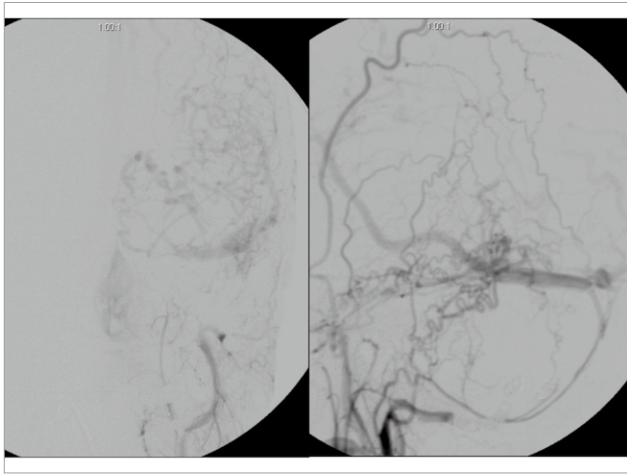


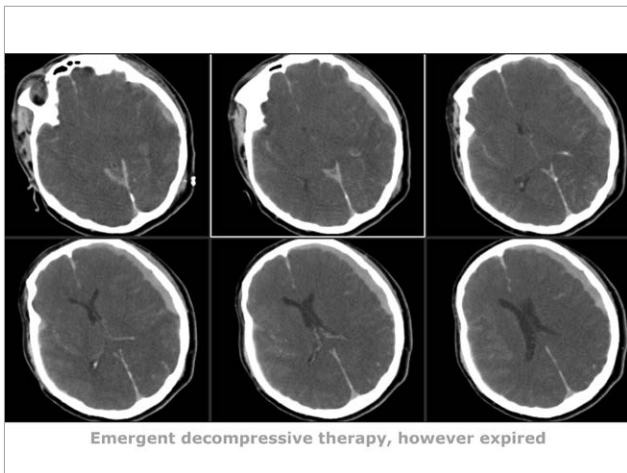
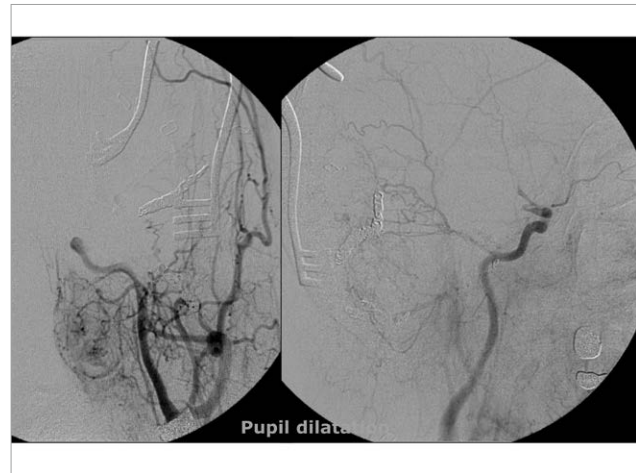
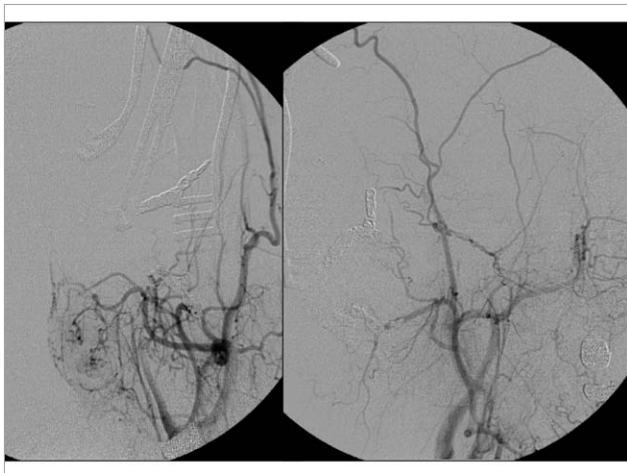
18mo FU









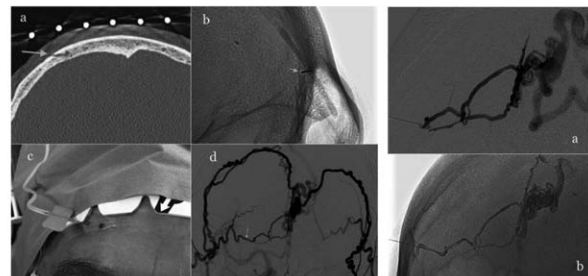


- Early detection of hemorrhage
 - Only minimal BP fluctuation
 - BP during procedure 90-110/50-70, PR 60-80
 - Single peak 140/80, 71
 - IICP sign check
- Exact localization
 - Larger FOV
 - Puncture
 - Subdural hematoma detection
 - Too small target? 4.9mm?
 - More difficult on left side?
 - Working space; detector & anesthesiology table
 - Patient position
- Other treatment modality?

Transcranial foraminal access

- Technique
 - Identification of transcranial (transdiploic) meningeal branch
 - Large enough diameter of foramen
 - Visualization of foramen on 2d x-ray
 - AP view; puncture location
 - Lat view; puncture depth (outside inner table of skull)
 - 25G venous system butterfly needle with 30cm microbore (AbbottLab) or 18G IV catheter with microcatheter
 - Onyx/glue injection

Chapot JNS 2007
Saura JNIS 2013

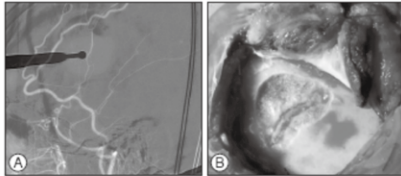


Saura JNIS 2013



Transcranial MMA access

- Technique
 - Identification of target MMA branch
 - *Cranial decortication*
 - Meningeal artery vasospasm prevention
 - 10mg verapamil IA

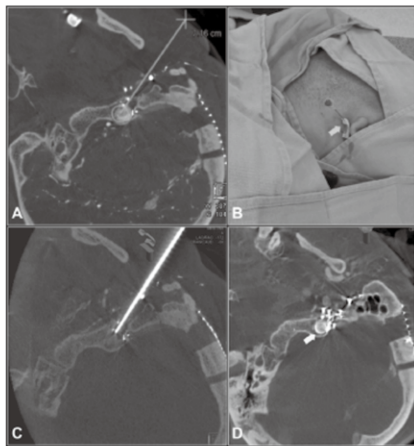


OhJS JKNS 2015

Transcranial navigation assisted access

- Hypoglossal canal DAVS
 - 3D DSA; exact localization of shunt
 - Multiplanar & volume rendered CBCT; ideal virtual needle path & depth
 - *Navigation software* (Syngo iGuide needle guidance, Siemens); Bull's eye & needle progression views

Diaz JNIS 2018



Diaz JNIS 2018

Conclusions

- Direct access of the target sinus through various routes may be a viable option for embolization of DAVFs when other conventional transvenous and transarterial routes are unavailable or not safe
- Techniques, devices, vascular anatomy, patient position, and logistics should be meticulously studied and planned before the procedure
- Multidisciplinary team with state of the art equipment is necessary for safe and effective treatment

대한뇌혈관외과학회 | 대한뇌혈관내수술학회

KSCVS-SKEN 합동 연수강좌

Workshop by Video for Beginner (Surgical Anatomy and Basic Technique)

좌장: 김태선 (전남대), 김정은 (서울대)

- | | |
|---|------------|
| 1. ACOM, Distal ACA aneurysm | 정준호 (연세대) |
| 2. MCA aneurysm | 방재승 (서울대) |
| 3. PCOM, Anterior choroidal artery aneurysm | 이상원 (부산대) |
| 4. CEA | 주성필 (전남대) |
| 5. STA-MCA bypass | 박익성 (가톨릭대) |





1. ACOM, Distal ACA aneurysm

정준호

연세대학교 의과대학 세브란스병원 신경외과

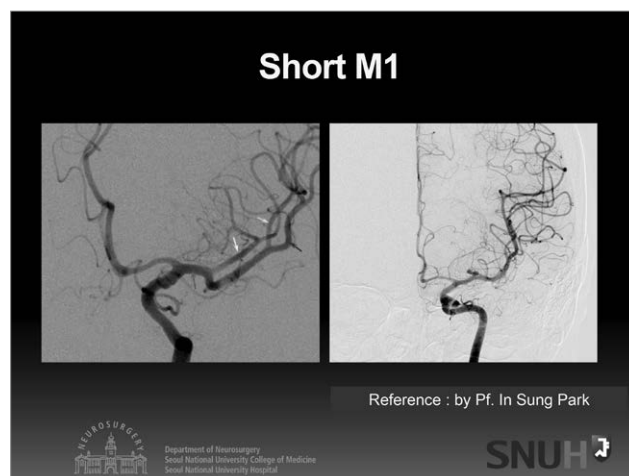
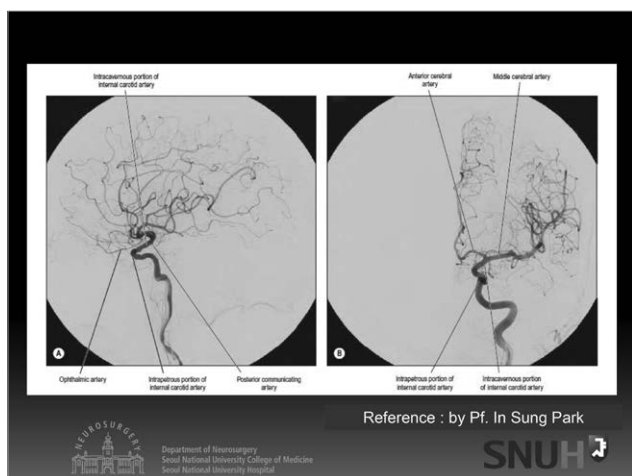
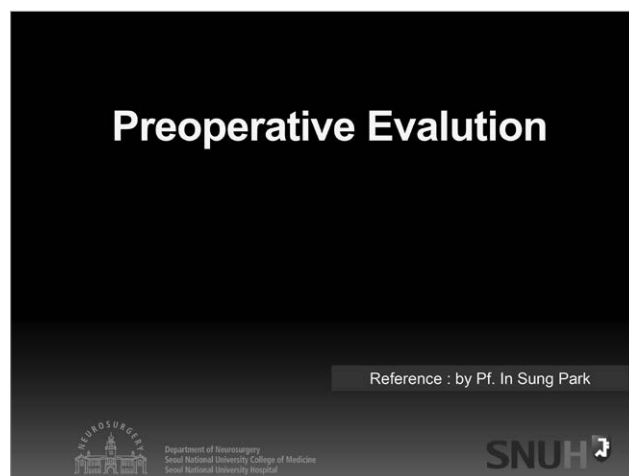
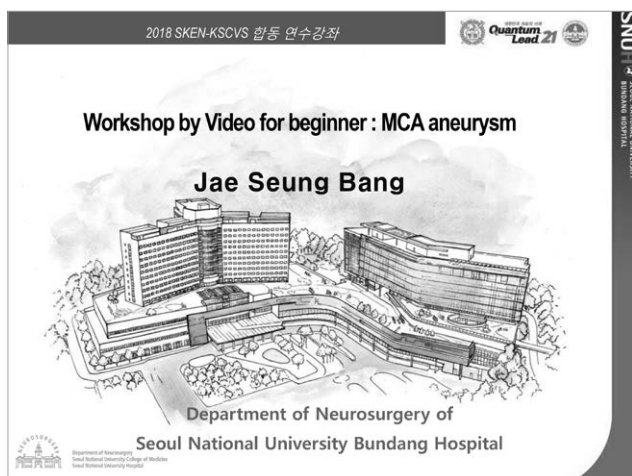
The anterior communicating artery (Acom) region is one of the most common sites for ruptured intracranial aneurysms. When performing microsurgical clipping of the Acom aneurysms, it should be concerned that the Acom aneurysms are made complex by the frequently associated variants of anatomy and the difficulties in fully visualizing the major arterial trunks and perforating arteries. Cerebrovascular surgeons should be aware of microsurgical orientation, such as the dominance of A1 segments, the direction in which the fundus points, main parent arteries with their branches, and surrounding structures, to perform microsurgical clipping safely. In addition, surgically challenging characteristics of the Acom aneurysms are their bilateral antegrade arterial supply, their deep & midline location, and their intimate relationship to 11 critical arteries.

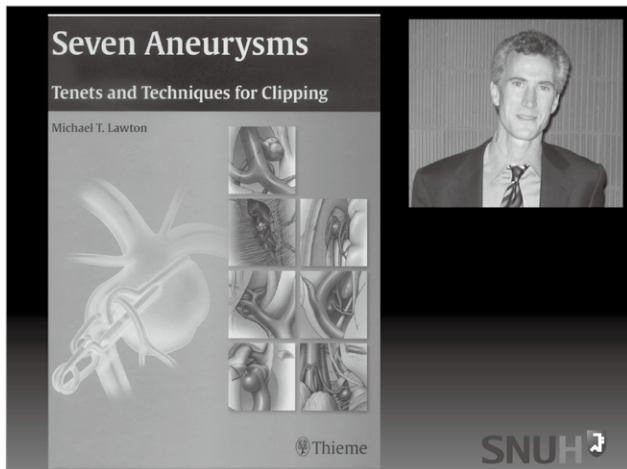
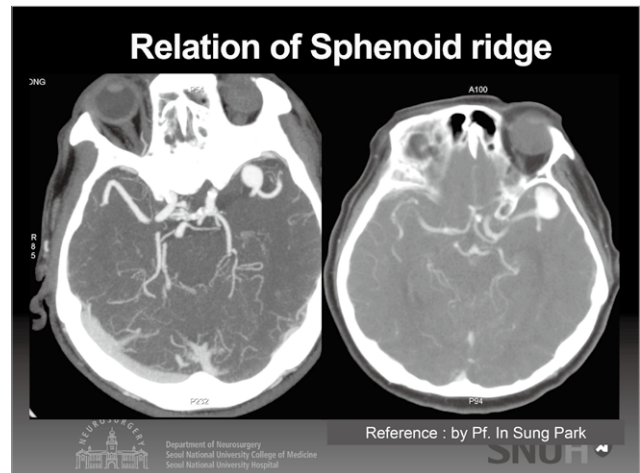
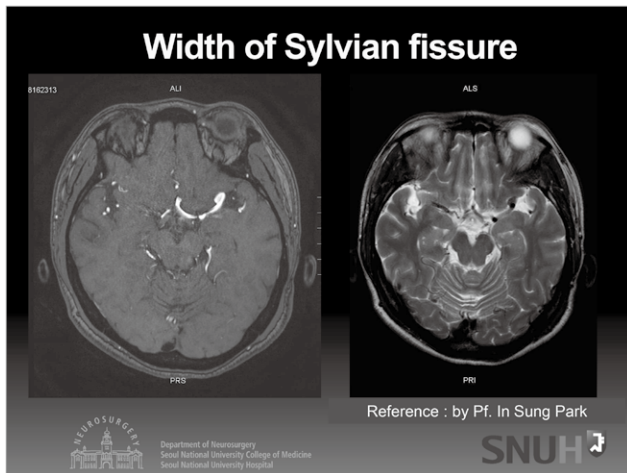
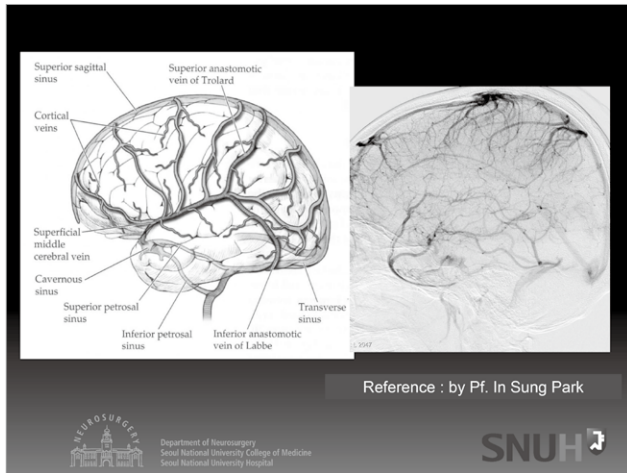
The most common aneurysm site on the distal anterior cerebral artery (DACA) is at the level of origin of the callosomarginal artery from the pericallosal artery, usually in close proximity to the anterior part of the corpus callosum, near the point of greatest angulation of the artery at the genu. The reasons why those aneurysms are treated microsurgically are their peripheral location, small size, unfavorable neck-to-parent artery ratio, and tendency to “blow out” the bifurcation at which they occur. The amount of brain retraction and muscle dissection needed to clip these lesions is comparatively minimal relative to other aneurysms.

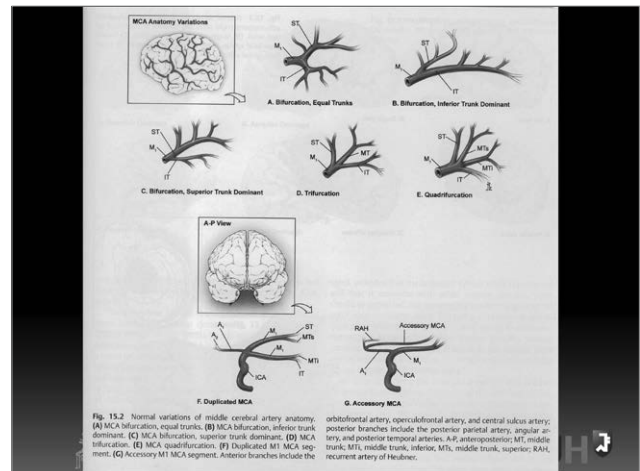
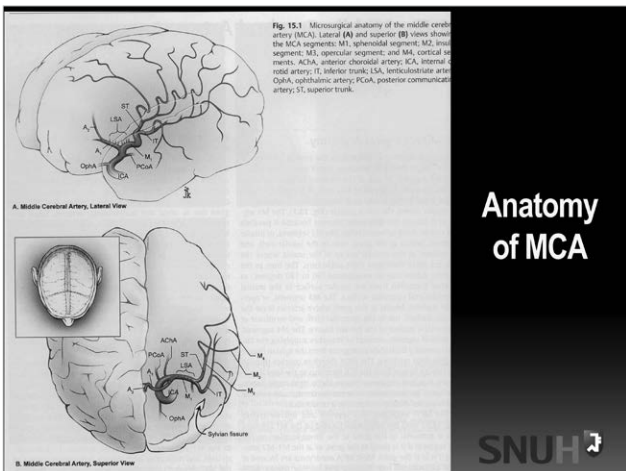
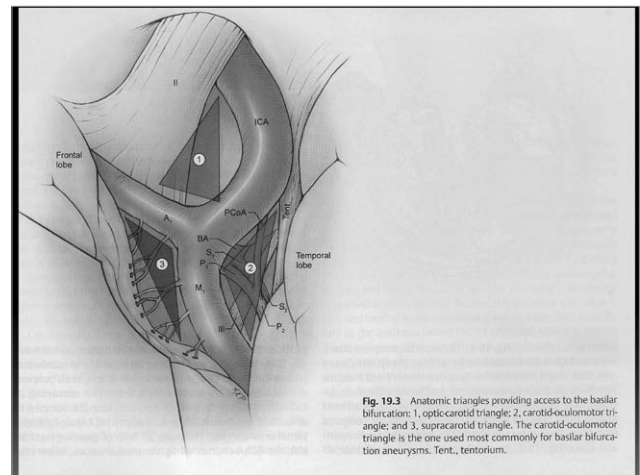
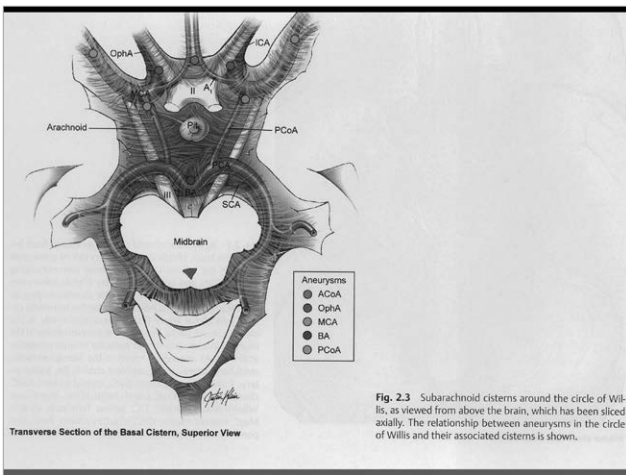
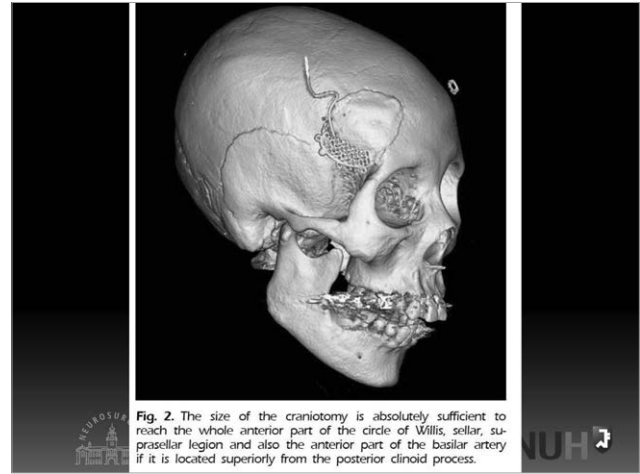
Here, we discuss surgical adjuncts, choice of the side or location of the craniotomy, head position, sylvian or interhemispheric fissure dissection, microsurgical anatomy under microscope, identification of the anatomical structures, and clip selection by showing the operative video.

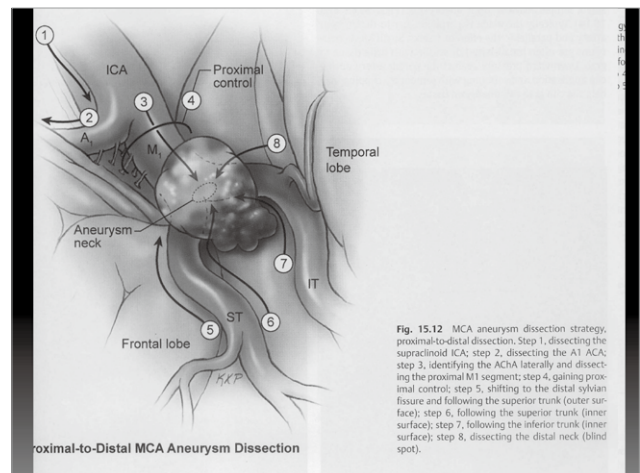
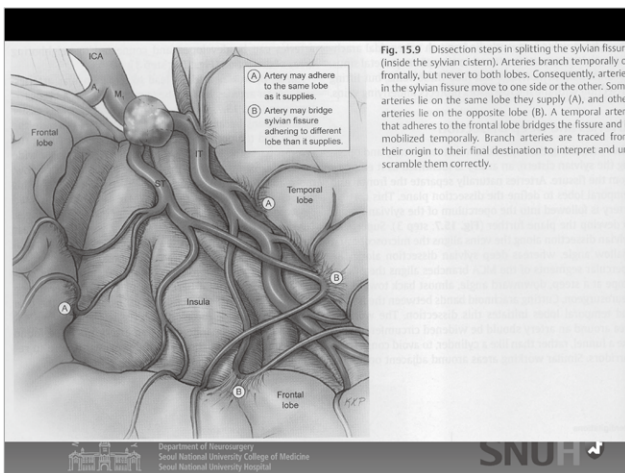
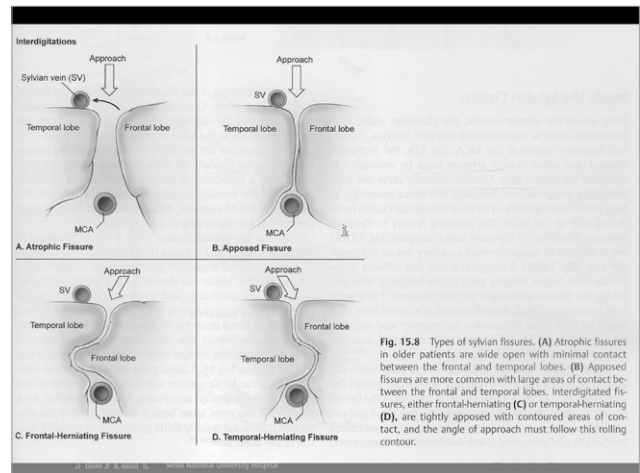
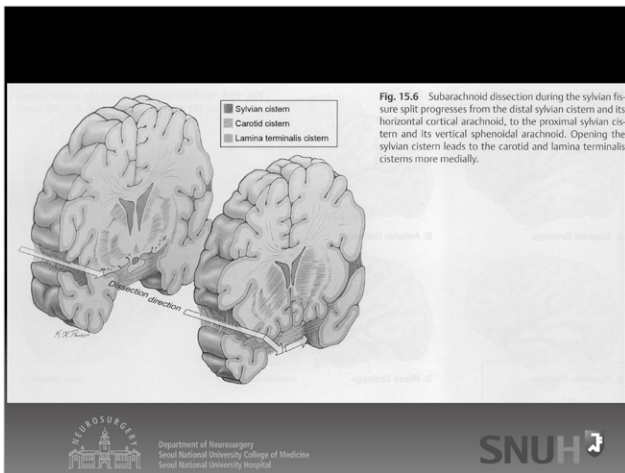
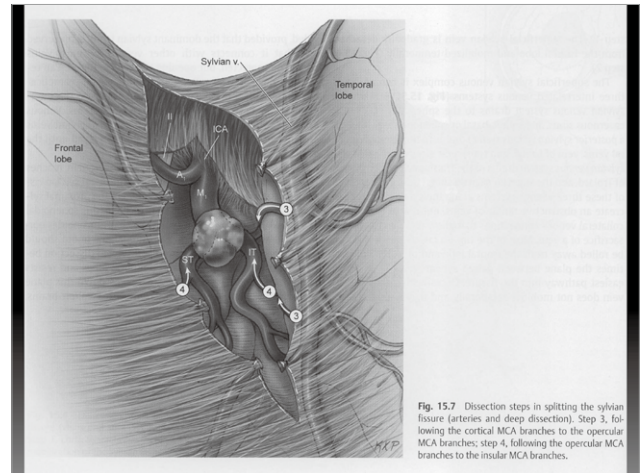
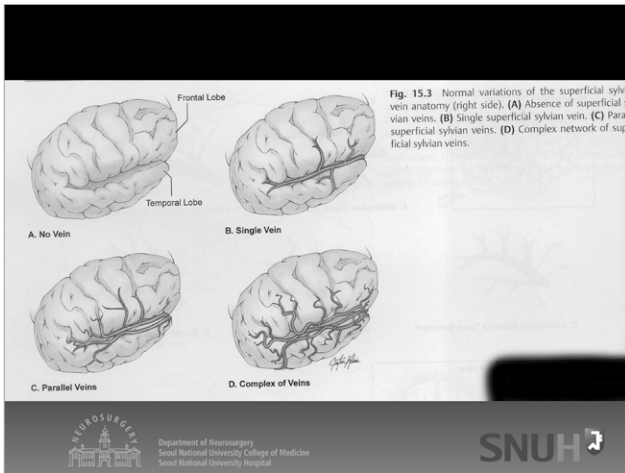
2. MCA aneurysm

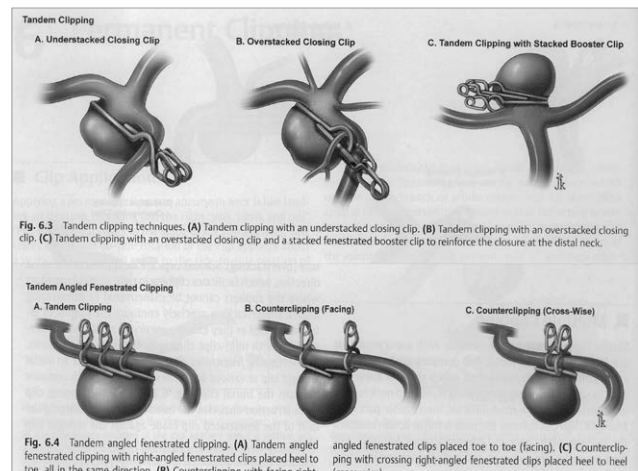
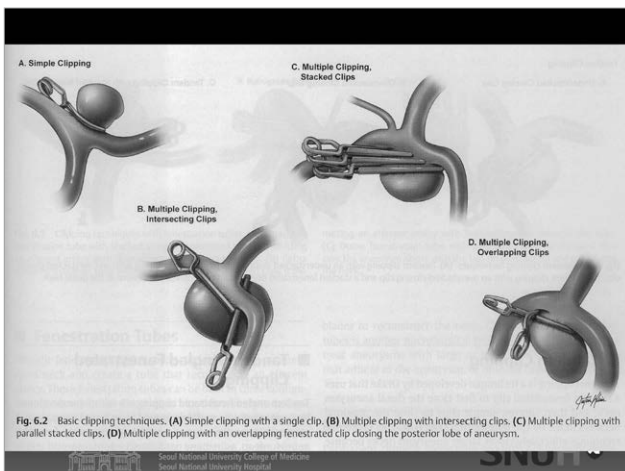
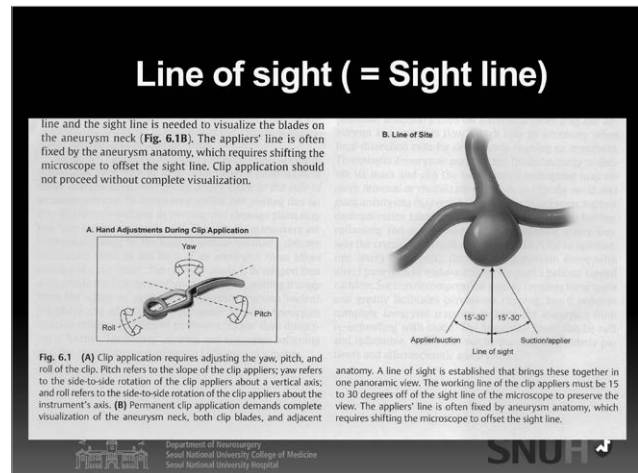
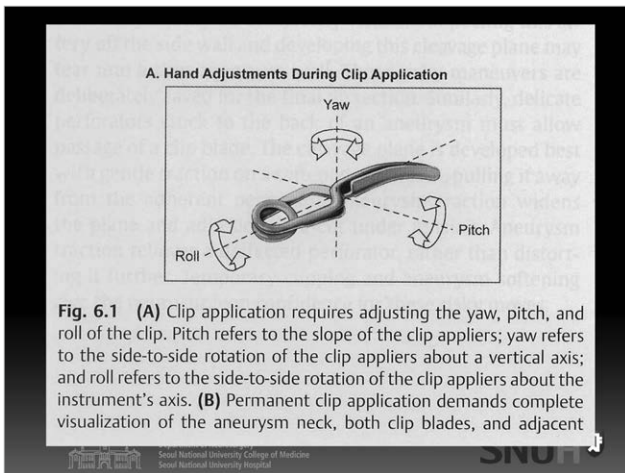
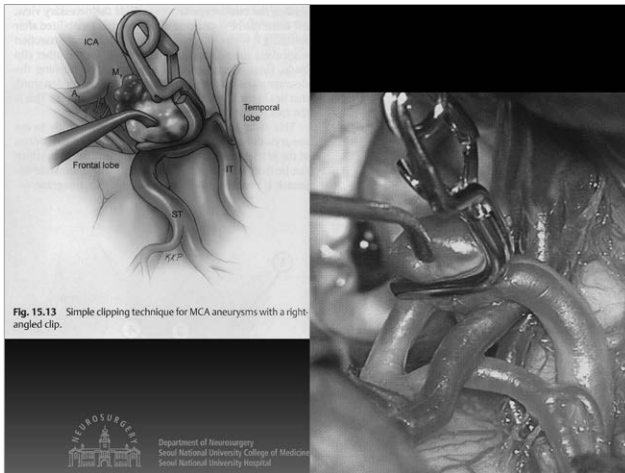
방재승
분당서울대학교병원 신경외과

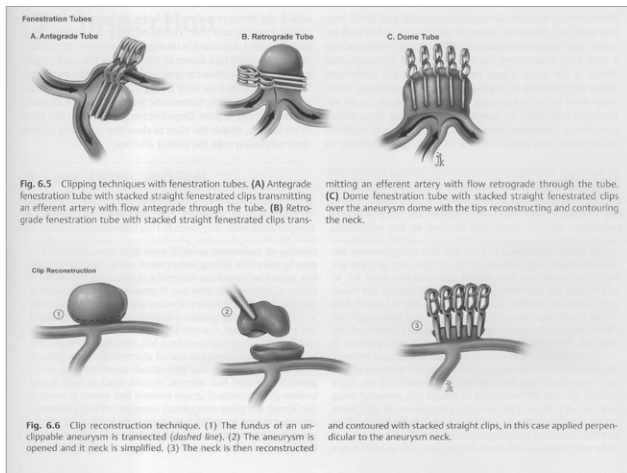








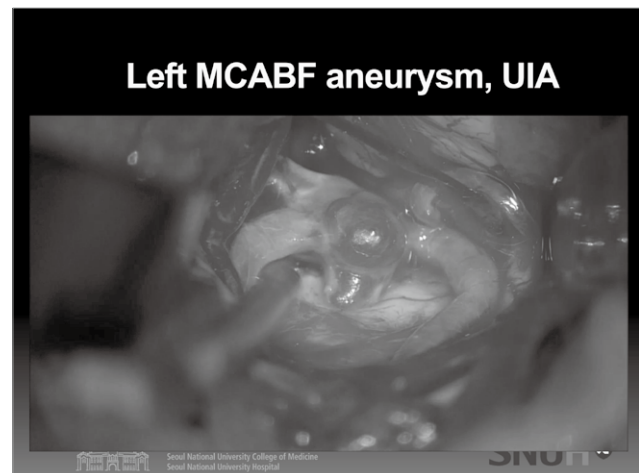
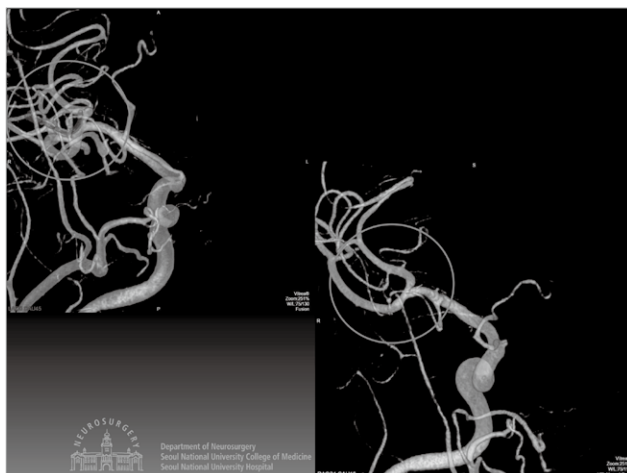
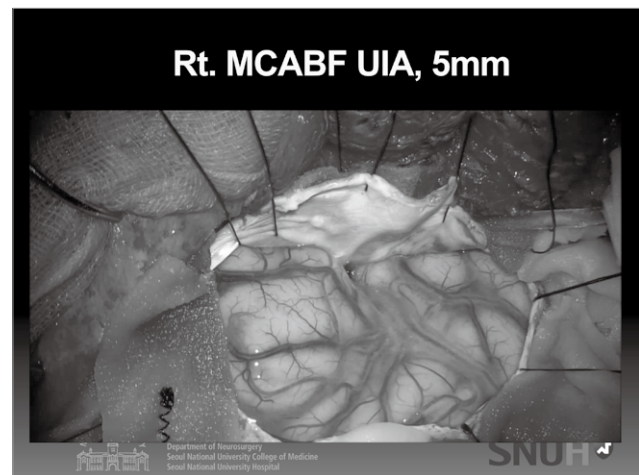
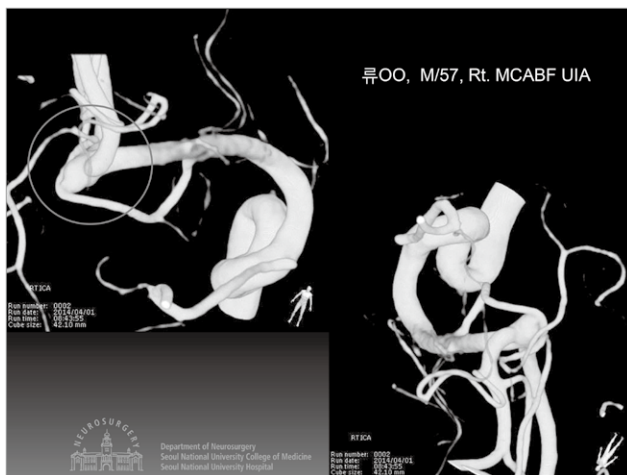


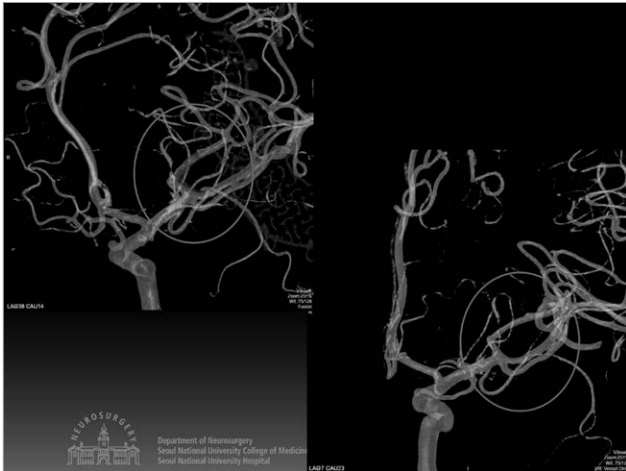


Principle of Arachnoid dissection

□ 3 basic maneuver with 3 simple instruments

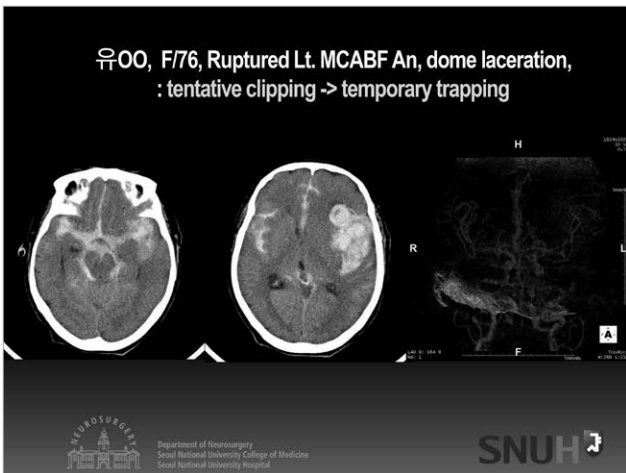
- 1) Cutting with microscissor
- 2) Spreading with bipolar forcep
- 3) Probing with a slightly curved dissector
(Rhoton No. 6 dissector; Codman; Raynham, MA)





Coping with crisis during clipping

- Aneurysmal dome laceration
 - Try proximal control!
 - Try tentative clipping!
 - Try temporary trapping!
- Aneurysmal neck laceration
 - Clipping with Tachocom®/Bemsheet®!
 - Clipping after direct suture!
 - Trapping and then do bypass surgery!



Conclusion : Surgical management of MCA aneurysm

- Routine pterional craniotomy, especially to beginners!
- Sylvian dissection with calm mind
 - protection of important v. is very important!
 - long-time retraction must be avoided!
 - M1 perforators must be saved!
- Iatrogenic stenosis by clipping must be considered, especially in atherosclerosis.



Thank you for your attention!



3. PCOM, Anterior choroidal artery aneurysm

이상원
양산부산대학교병원 신경외과

Post. Communicating Artery Aneurysm & Ant. Choroidal Artery Aneurysm

양산부산대학교병원
이 상 원

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Post. Communicating Artery

- Origin
Posteromedial aspect of the ICA
halfway between the ophthalmic artery & the bifurcation
- Course
Run medially & posteriorly above & medial to the 3rd n.
- Type
Normal type(54%): > 1mm, < P1
Hypoplastic type(24%): < 1mm
Fetal type(18%): > P1
- Anterior thalamo-perforating artery
Average of eight (4 to 14) perforating branches

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Premamillary Artery (Tuberothalamic Artery)

The largest branch and commonly originates at the middle of the PCoA.
Enters the floor of the third ventricle between the mammillary bodies and the optic tract

Supply:

posterior hypothalamus
ventral thalamus
post. limb of internal capsule

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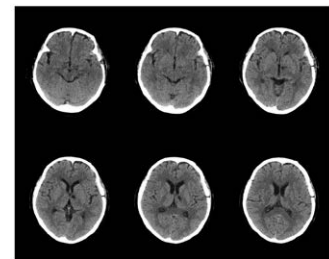
Premamillary Artery (Tuberothalamic Artery)

Supply

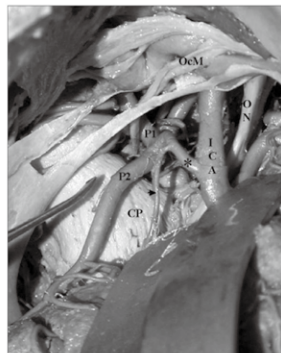
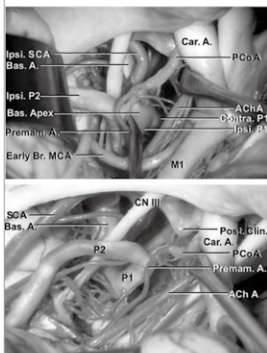
Hypothalamus
Ventral/Anterior thalamus
Optic tract
Posterior limb of the internal capsule
Posterior perforated substance
Subthalamic nucleus

Symptom

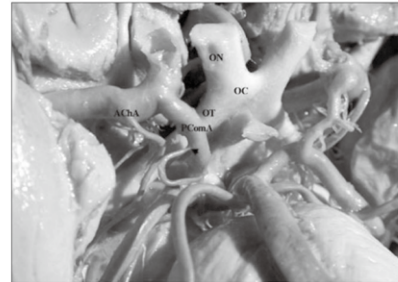
Language and memory disturbances
Contralateral motor weakness
Changes in the superficial sensation
Apathy
Lack of spontaneity
Disorientation



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Ant. Choroidal Artery

- Origin
 - Posterior wall of the ICA
 - Between P-com and ICA bifurcation
 - Commonly single artery(70%) (infrequently two or more)
- Course
 - posteromedial direction behind the ICA
 - passes below or along the medial side of the optic tract
 - It enters the crural cistern (between the cerebral peduncle and uncus), and then it passes through the choroidal fissure into the choroid plexus within the temporal horn.

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Ant. Choroidal Artery

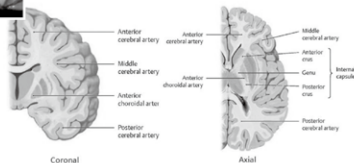
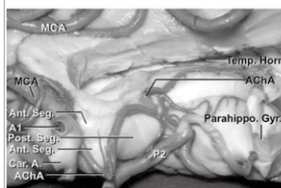
Segments

- Cisternal segment : extends from its origin until the choroidal fissure
- Plexal point
- Intraventricular segment : After entering the choroidal fissure
 - Anastomoses with branches of the medial posterior choroidal artery

Vascular territory

- Cisternal segment
 - Optic tract
 - Lateral geniculate nucleus and lateral aspect of thalamus
 - Posterior limb of the internal capsule
 - Lateral aspect of the midbrain
- Intraventricular(choroidal) segment
 - Choroid plexus of the lateral ventricle(temporal horns)

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PComA Aneurysm

Pterional craniotomy

Sylvian fissure open and distal neck dissection, identification of AChA

Identification of PComA

Proximal control (tent. Incision, ACP removal, cervical ICA, retrograde suction decompression)

Proximal neck dissection

Clipping (straight clip, bayonet clip, curved clip, fenestrated clip)

AChA, PComA, perforators, 3rd nerve

Ruptured aneurysm : avoid medial temporal lobe retraction

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Dome Projection of PComA Aneurysm

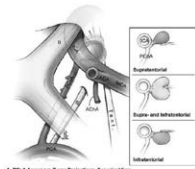
Anterolateral

Superolateral

Posterolateral superior : (supratentorial) :temporal lobe

Posterolateral inferior : (infratentorial) : 3rd n

Posteromedial inferior : inferior wall aneurysms.



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AChOA Aneurysm

Pterional craniotomy

Sylvian fissure opening

Identification of PComA & proximal control

Identification of ICA bifurcation

Identification of AChOA

Neck dissection

Clip application

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Summary

- Pterional transsylvian approach
- Avoid medial temporal retraction in laterally & superiorly projecting aneurysm (ruptured)
- Identify & preserve PComA, AChOA, Perforators, 3rd nerve
- Avoid long clip
- MEP monitoring & ICG videoangiography (AChOA aneurysm)
- PComA An : ACP removal, Cervical ICA dissection, Retrograde suction decompression
- Subpial resection of temporal lobe (uncus)

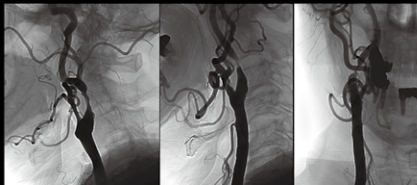
Thank You for Your Attention !



4. CEA

주성필
전남대학교병원 신경외과

Carotid Endarterectomy



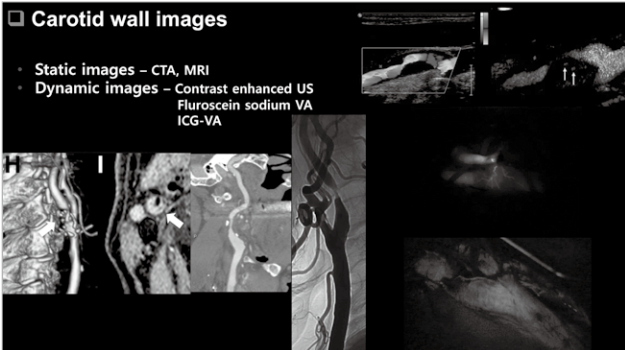
Sung-Pil Joo, Han-Young Han, Tae-Wook Song, Yoo-Sub Kim, Tae-Sun Kim
DEPARTMENT OF NEUROSURGERY,
CHONNAM NATIONAL UNIVERSITY, GWANGJU, KOREA

Op. indication

- **Symptomatic carotid stenosis (2 weeks of the last symptom)**
70% to 99% in patients with a life expectancy of at least 5 years
+ perioperative risk <6%
- **Symptomatic carotid stenosis**
50% to 69% with the same caveats as above
Women : no benefit from CEA and should probably be managed medically
- **Asymptomatic patients**
between the ages of 40 and 79 years with greater than 60% stenosis
: no significant comorbid conditions that might increase their perioperative risk or result in a life expectancy of less than 5 years, The overall perioperative risk <3%,
The benefit to women is controversial

□ Carotid wall images

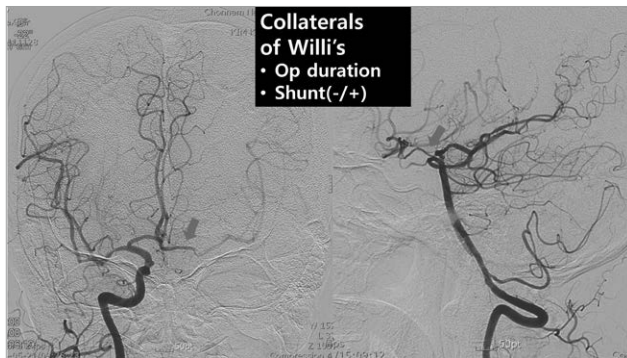
- Static images – CTA, MRI
- Dynamic images – Contrast enhanced US
Fluorescein sodium VA
ICG-VA



Anesthesia

General vs Local

Advantages	Disadvantages
General anaesthesia	
Immobility	Lack of direct neurological monitoring during surgery
Potential for neuroprotection	Intraoperative hypotension
Controlled ventilation and CO ₂	Postoperative hypertension
Attenuated stress response	Increased rate of shunt use
	Delayed recovery from GA may mask postoperative neurological complications
Regional anaesthesia	
Allows direct real-time neurological monitoring	Risks associated with sitting blocks (deep cervical plexus blockade)
Avoids the risks of airway intervention	Patient stress/pain causing increased risk of myocardial ischaemia
Reduced shunt rate	Restricted access to airway during surgery
Reduced hospital stay	Requires co-operative patient, able to lie flat
Allows arterial closure at "normal" arterial pressure: may reduce risk of postoperative haematoma	Risk of requirement to convert to GA during surgery



Monitoring

- Transcranial doppler
- EEG
- SSEP/MEP
- Stump pressure
- Oxygen Monitoring
 - SOMA(oxygen monitoring)
 - NIRS (Near-Infrared Spectroscopy)

Monitoring - IOM(SSEP/MEP)

Stump pressure

- Pressure distal to carotid
- Represents pressure transmitted from contralateral side via the circle of willis
- Cerebral ischemia rarely occurs at stump pressure above 60 mmHg

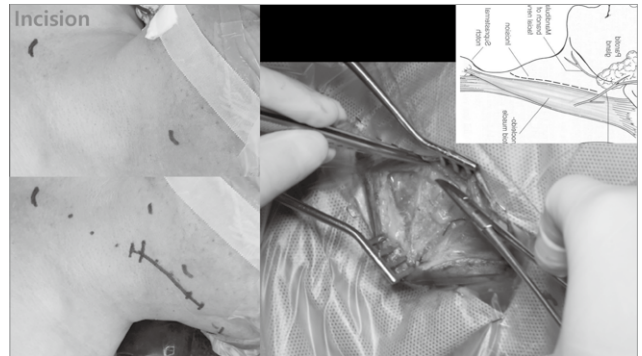
Carotid stump

NIRS (near-infrared spectroscopy)

Real-time non-invasive detection of tissue oxygen saturation using transillumination spectroscopy

NIR light wavelengths (690-880nm) can be used to measure regional tissue oxygen saturation

SOMA(oxygen monitoring)

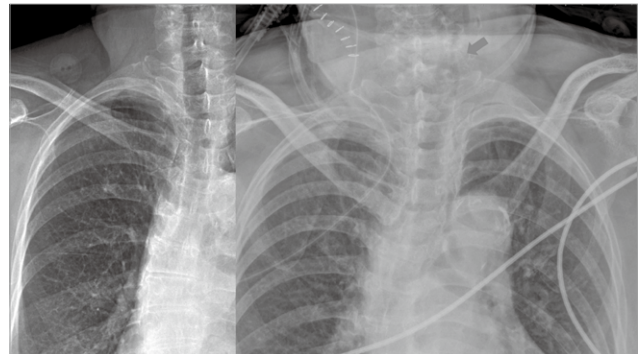
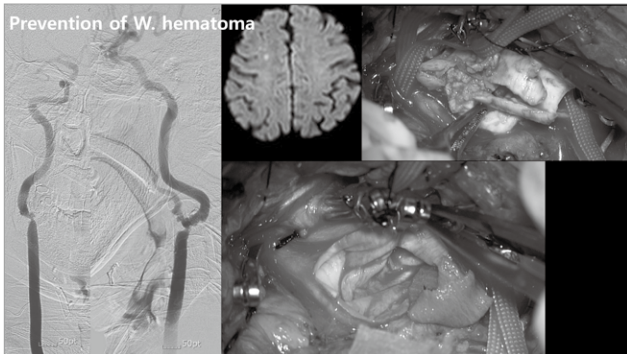


How to reduce post-CEA complications?

1. Wound hematoma
2. Post-CEA cerebral infarction
3. Hyper-perfusion syndrome

How to prevent and treat wound hematoma

- Change the pattern of suture (*interlocking suture*)
→ Marginal suture with muscle string, 2-3 min necessary.
- Postoperative BP control and check the PT and APTT
- Aggressive treatment of wound hematoma



How to prevent Post Cb. infarction

1. Short op time
2. Shunt
3. early ECA opening
4. pre-OP angiographic evaluation

High-risk Features for CEA

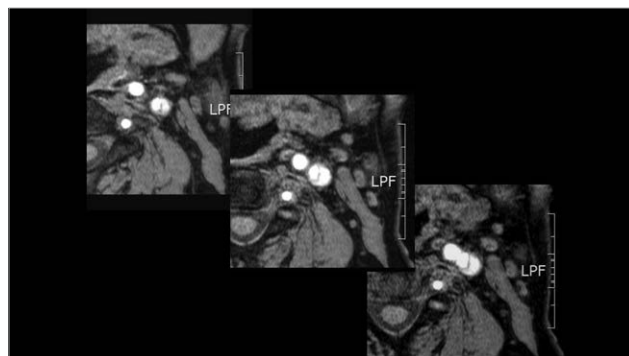
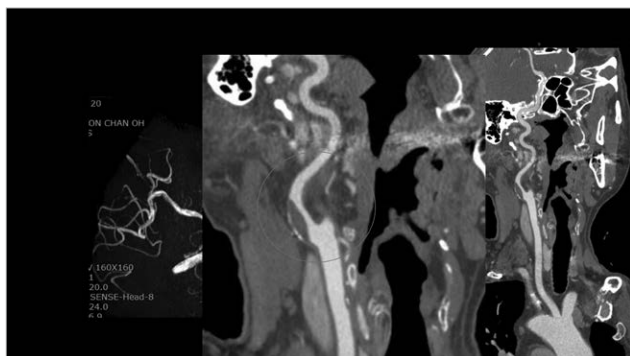
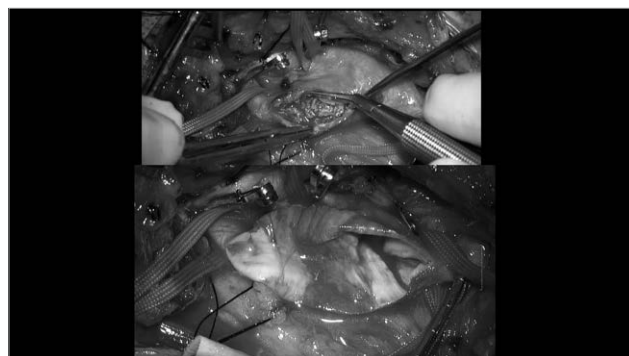
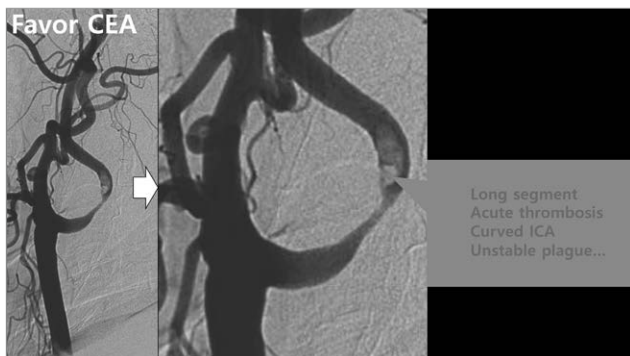
Anatomic

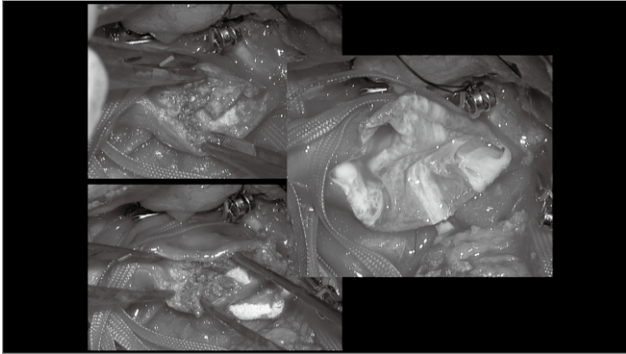
- Restenosis after endarterectomy
- Bilateral stenosis
- Contralateral carotid occlusion, laryngeal nerve palsy
- Previous radiation therapy or surgery on the neck
- Lesion inaccessible by surgery
- Neck immobility
- Tracheostomy or tracheostoma
- Severe intracranial stenosis

Comorbid Conditions

- Unstable angina
- Left ventricular ejection fraction <30%
- Congestive heart failure
- Planned coronary artery bypass or valve replacement
- Renal failure
- Chronic obstructive pulmonary disease
- Coronary artery disease with $\geq 70\%$ stenosis
- Planned peripheral vascular surgery
- Myocardial infarction within 6 weeks of the procedure
- Age older than 80 years

Favor CEA





5. STA-MCA bypass

박익성

가톨릭대 부천성모병원 신경외과

STA-MCA anastomosis technique
for beginner : Video presentation

박익성

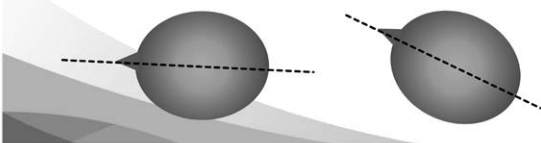
가톨릭대학교 부천성모병원

Overview

- Position and anesthesia
- Flap design
- Donor vessel harvest and preparation
- Craniotomy
- Recipient selection and preparation
- Anastomosis
- Closure
- Some tips for beginners

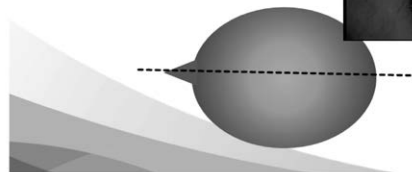
Position and anesthesia

- General anesthesia with inhalation or i.v. anesthetics
- Avoiding hypotension, hyperventilation
- Supine position without pinning
- Head rotation



Flat (full lateral)

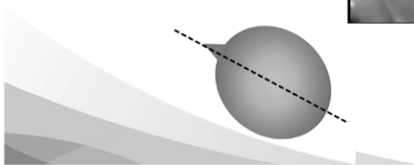
- Working under CSF
- Minimal brain shifting
- Difficulty in handling nylon
- Unclear Operation field





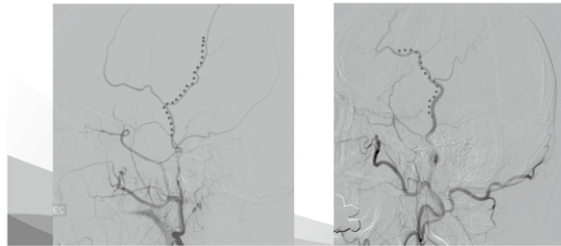
Natural (Tilting)

- Clear operation field
- Comfortable in handling nylon
- More brain shift
- Dry up : Thrombogenic risk



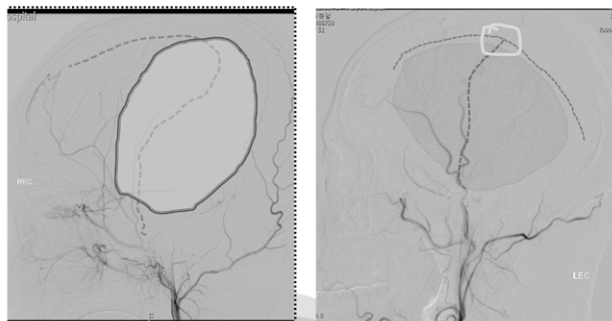
Flap design

- Depends on operation strategy and STA status



Flap design

- Depends on operation strategy and STA status

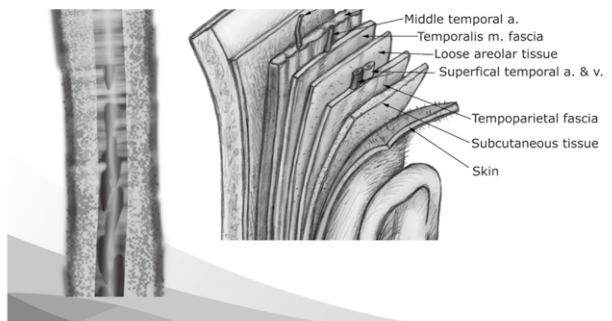


Donor vessel harvest and preparation

- Atraumatic dissection of STA with preserved perivascular soft tissue
- Complete hemostasis with microbipolar coagulator
- Temporary occlusion – just distal to bifurcation, complete secure
- Trimming distal tip without intimal dissection



Location of STA



Donor vessel harvest and preparation



Craniotomy

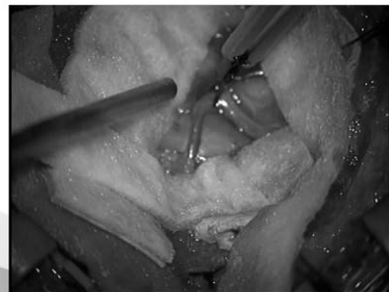
- Depends on the purpose of surgery
 - Single vessel direct anastomosis –Chater' s point
 - Double vessel direct anastomosis – along sylvian cistern
 - Moyamoya disease : as wide as possible

Recipient selection and preparation

- Location : cortical M₄
- Size : more than 1 mm (0.7 -0.8 mm)
- Shape : no atherosclerotic change
- Morphologic consideration :Red >> gray
- Hemodynamic consideration
 - Flow direction: antegrade vs retrograde
 - Flow amount
- Dominancy consideration

Recipient preparation

- Cauterization and cutting of small branch
- Apply rubber dam under the vessel
- Continuous suction or natural drain of CSF
- Papaverine for constricted vessel





Anastomosis

- Temporary clipping of recipient vessel, complete secure
- Make small hole and extend – avoid intimal dissection
- Linear incision along vessel axis, twice longer than vessel diameter
- Same incision length : donor/recipient
- Frequent heparin saline irrigation
- Avoid intimal injury during suture by forcep

Anastomosis

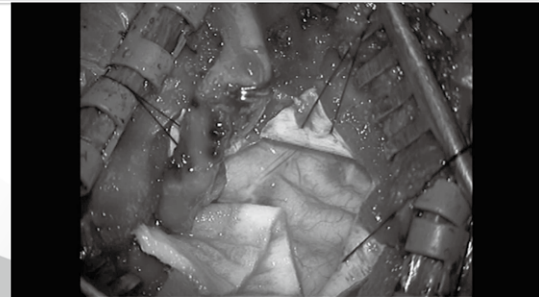
- First stitch : heel or toe
- Running or interrupted suture
- Even stitch interval
- Avoid dog ear
- Avoid inversion of adventitia
- Temporary opening of occluded vessel before final tie
- Flow confirmation with doppler

Closure

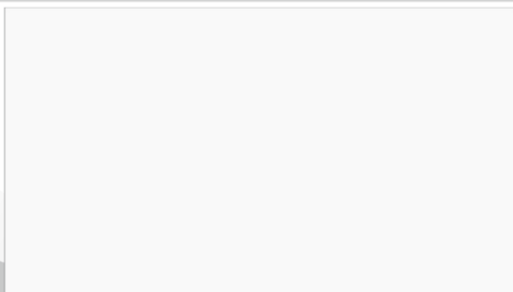
- Dura, Muscle, Bone : make a room for the vessel
- Skin : avoid tension
- Dressing : avoid compression



Some useful tips



Some useful tips



- 경청해 주셔서 감사합니다
- You Tube : sta-mca, 박익성

대한뇌혈관외과학회 | 대한뇌혈관내수술학회

KSCVS-SKEN 합동 연수강좌

Scientific Session III: SAH, Vasospasm

좌장: 고현송 (충남대), 김성림 (가톨릭대)

1. VSP, Basic research update

박동혁 (고려대)

2. Intervention for VSP

신승훈 (분당제생병원)

3. Medical complication & treatment

유찬중 (가천대)





1. VSP, Basic research update

박동혁

고려대학교 의과대학 안암병원 신경외과

Delayed cerebral vasospasm has long been recognized as an important cause of poor outcome after an otherwise successful treatment of a ruptured intracranial aneurysm, but it remains a pathophysiological enigma despite intensive research for more than half a century. Cerebral vasospasm has traditionally been regarded as an important cause of delayed cerebral ischemia (DCI) which occurs after aneurysmal subarachnoid hemorrhage, and often leads to cerebral infarction and poor neurological outcome. However, data from recent studies argue against a pure focus on vasospasm as the cause of delayed ischemic complications. Findings that marked reduction in the incidence of vasospasm does not translate to a reduction in DCI, or better outcomes has intensified research into other possible mechanisms which may promote ischemic complications. Early brain injury and cell death, blood-brain barrier disruption and initiation of an inflammatory cascade, microvascular spasm, microthrombosis, cortical spreading depolarizations and failure of cerebral autoregulation, have all been implicated in the pathophysiology of DCI. Summarized in this presentation are highlights of research reflecting recent advances in the understanding of delayed ischemic deficit. It will focus on current accepted mechanisms and on new frontiers in vasospasm research.

2. Intervention for VSP

신승훈
분당제생병원 신경외과



3. Medical complication & treatment

유찬중

가천대학교 의과대학 길병원 신경외과

지주막하 출혈의 병태생리

1) 지주막하출혈

지주막하 출혈 후 적혈구가 파괴되면서 유리되는 K⁺ 나 혈색소, 또는 백혈구나 혈소판에서 유리되는 prostaglandins, leukotrienes, bradykinin에 의해 생화학적 염증성 연쇄 반응을 일으켜 뇌의 손상을 줄 수 있다. 파열후 두시간 이내에 염증성 반응이 일어날 수 있고 다형성 백혈구의 지주막 아래의 유입도 관찰된다. 이러한 다형성 백혈구는 림프구와 단핵구가 되어 파열후 48시간 이내에 식세포 작용이 일어나 국소적으로 연막의 섬유화와 지주막아래 공간의 파열이 일어날 수 있다. 이로인해 뇌동맥류 파열 후 경부각직과 열이 나는 것으로 사료된다. 뿐만 아니라 염증성 반응으로 인해 수두증과 혈관 연축도 일어날 수 있다.

2) 뇌실질내 출혈

뇌동맥류 파열의 60%에서 발생하며 중대뇌동맥이나 전교통동맥 파열에서 주로 흔히 일어난다.

중대뇌동맥 - 실비우스열 또는 직회, 전두부로 일어나고 뇌량주위 동맥류는 대상회로의 뇌실질내 출혈이 일어나는 반면에 후교통동맥의 경우 측두부 뇌실질내 일어난다.

후순환계 동맥류는 뇌실질내 출혈이 드문 반면 원위부 후대뇌동맥의 경우 후두엽 뇌실질내 출혈을 일으킬 수 있다.

3) 뇌실질내 출혈

뇌동맥류 파열의 25%에서 관찰되며 많은 경우 불량등급에서 발생 가능성이 높으며 급성 또는 만성 수두증, 뇌압상승, 뇌혈류 감소, 혈관 연축의 발생과도 관련이 있다. 뇌동맥류 파열 후 갑작스런 사망과도 관련이 있어 급사의 90%에서 뇌실질내 출혈이 관찰된다.

4) 경막하 혈종

약 1%에서 관찰되고 발생한 경우 불량한 등급에서 관찰되며 뇌동맥류의 dome이 지주막과 근접해 있는 내경동맥과 중대뇌동맥 동맥류 파열 때 가장 잘 발생한다. 종괴 효과로 뇌압 상승 유발한다.

5) 수두증

급성수두증은 20%에서 발생되며 급성 뇌실 확장은 뇌실내 출혈과 연관되어 나타난다.

만성 또는 지연성 수두증은 지주막 출혈 후 1-3주사이에 바렛 o되며 약 20%에서 보인다. 급성의 경우 뇌실이나 지주막에서 뇌척수액의 배출이 막힌 경우 발생하는 반면에 만성 수두증은 뇌실막, 뇌실막하, 지주막용모의 손상, 섬유화, 척수액 흡수의 장애로 발생한다. 급성 수두증은 두개강내압의 상승, 혈관 연축과 연관되는 반면 만성 수두증의 경우 두개강내압은 정상이다.

6) 재출혈

이미 뇌 손상이 있는 상태에서 출혈이 다시 일어나기 때문에 예후가 나쁘며 파열을 치료하지 않으면 약 50%에서 6개월 이내에 재출혈이 일어난다.

재출혈의 발생율은 지주막출혈이 발생한 후 첫 24시간 이내에 가장 높고(4.1%), 첫 13일 간 매일 1.5% 정도씩 감소한다. 첫 2주간 재발율은 195이다.

7) 뇌부종과 경색

뇌부종은 직접적으로 뇌의 기능을 방해하거나 두개강내 내압을 높일 수 있다. 혈관인성, 세포독성, 간질성 또는 삼투성 부종이 모두 발생 가능하다. 출혈 후 첫 몇시간 동안 뇌의 체액과 나트륨의 확연한 증가로 혈관인성 부종이 되거나 세포독성 부종이 나타나며, 포타슘의 변화도 관찰되는데 이는 혈관인성 부종이 지주막하 출혈의 주된 부종이라는 것을 시사한다. 출혈과 직접 접한 뇌 부분에서는 하루 안에 모세 혈관의 누출이 보이며 이는 지주막하 출혈후 부종 형성에 중요한 역할을 하는 것을 시사한다.

뇌경색은 지주막출혈의 양과는 연관이 있으며 저혈압이나 뇌실질내 출혈이 있을 때 뇌경색의 위험도가 세배 정도 된다. 또한 뇌경색은 뇌탈출로 인해 후대뇌동맥의 감돈으로도 일어날 수 있다.

지연성 뇌경색은 나치성 두개강내 고혈압, 수두증, 혈관 연축 등에서 발생할 수 있으며 혈관 조영술에서 보이는 혈관 연축 환자의 30%에서 지연성 뇌경색이 발생한다.

8) 대뇌탈출 증후군 뇌실질내 출혈, 대뇌부종, 급성 수두증, 두개강내압의 상승에 의한다.

지주막하 출혈의 병태 생리

지주막하 출혈은 두개강내압 상승, 뇌혈류량 감소, 뇌조직 산소공급의 감소 및 자동조절이나 이산화탄소 방응성의 이상, 대사이상, 전신 순환 용적의감소등 여러 요인으로 인해 이차적 뇌손상의 가능성이 높다.



1) 두개강내압

뇌동맥류 파열 후 두개강내압의 증가가 심해 단기간의 뇌순환 정지가 발생하기도 한다. 150ml의 대량출혈은 나쁜 예후를 보이고 양호한 경우는 두개강내압의 증가가 후에도 빠른 감소를 보인다. 일반적으로 두개강 내압은 20mmHg이상 이며 30mmHg이상의 경우 불량한 예후를 갖고 15mmHg 이하의 경우 80% 이상에서 양호한 예후를 15mmHg이상의 경우 15%에서만 양호한 결과를 보인다. 두개강내압의 상승의 원인은 명확하지 않으나 지주막하 출혈의 용적, 뇌척수액 유출로의 폐쇄, 전반적인 뇌혈관마비, 말초 뇌동맥혈관의 확장 등이 가능한 원인으로 거론되고 있다.

2) 동맥류 출혈의 정지

급격한 두개강내압의 상승은 평균 동맥압을 높이며 관류정지를 일으켜서 파열 뇌동맥류의 출혈에 병적 압박을 일으킨다. 그러나 최저 관류압과 동맥류 파열에 의한 출혈량과는 아무런 연관성이 없고 뇌동맥류 파열의 절반이상에서 의식을 잃지 않으므로 관류 정지가 뇌출혈의 정지에 결정적인 반응으로 볼 수는 없다. 많은 경우에 파열부위의 혈류의 감소하여 섬유소가 응고되어 침착을 잘 일으키는 상태가 되면 출혈이 멈추는 것으로 되어 있다. 뇌동맥류 파열 후의 출혈량, 임상적 결과, 혈전 형성등은 지주막하 출혈의 초기 혈류 속도와 연관이 있다.

3) 뇌혈류

뇌동맥류 파열후 뇌혈류는 30 - 40 %정도 감소한다. 일반적으로 뇌혈류량의 감소는 초기(1-3일)와 후기(10-14일)가 많고 첫 14일에 최하점이 이르게 되며 초기 감소는 급성 대뇌손상(염증, 뇌압증가, 수두증, 뇌부종)에 의한 것이며 아급성 감소는 지연성 뇌혈관 연축때문으로 생각된다. 뇌혈류량 감소는 임상적 상태가 좋은 환자에서 호전되나 불량한 예후 환자에서는 그렇지 못하다. 뇌혈류량 감소는 두개강내압의 증가와 연관 되고 지주막 출혈 이후 수주 동안 뇌혈류량 변화가 일어나며 뇌혈류는 입원시 Fisher등급, Hunt-Hess등급과도 연관이 있다. 혈류량 감소에 미칠 수 있는 인자는 뇌출혈, 뇌실내출혈, 수두증, 뇌부종, 혈관연축, 전신적 저혈압, 대사율 등이다.

4) 뇌혈류량

뇌동맥류 파열 후 뇌혈류량은 증가하며 뇌혈관을 동반하면서 신경학적 결손이 있는 환자에게서 혈류량이 증가를 일으킨다. 명확한 기전은 확인되지 않으나 근위부 기저혈관의 연축 및 허혈을 보상하기 위해 원위부 미세 순환의 혈관이 확장이 일 가능성이 있다. 원위부 미세 혈관의 확장은 혈관이 탄성도와 자가 조절 능력의 감소로 혈관 예비능의 감소가 일어나며 임상적으로 불량한 등급의환자들이나 뇌혈관 연축이 동반된 환자에서 지연성 뇌허혈을 예측할 수 있다.

5) 뇌대사

지주막하 출혈은 뇌대사를 감소가 일어나는데 이는 지주막하 출혈의 직접적인 독성으로 인하여

산소 대사율의 감소, 이차적인 뇌혈류 속도가 감소가 일어나게 된다.

6) 대뇌의 자동조절 및 이산화탄소 반응성

대뇌순환은 정상적으로 뇌혈류 속도와 뇌관류압의 정도를 일정하게 유지 할 수 있도록 자동조절이 이루어지는데 관여 인자중 동맥의 평활근세포가 관여하고 출혈후 3-7일에 자동능력이 비정상적으로 보인다. 자동조절의 곡선은 우측으로 이동하고 결과적으로 작은 혈압의감소도 뇌부종 두개강내압력의 증가, 대뇌허혈이 일어나게 된다. 지주막하출혈 후 첫 일주일제 자동조절 능력의 장애가 보이고 뇌혈관 연축에 의해 신경학적 증상이 더 악화될 수 있다. 뇌지주막하 출혈로 인해 이산화 탄소의 반응성의 저하가 약 50%까지 일어난다. 자동 조절 능력의 장애는 고혈압, 고혈량적 치료를 통해 혈관 연축을 줄이는 이론적 근거를 제시한다. 자동조절이 잘 되지 않을 때 전신 동맥압의 증가는 뇌혈류속도와 측부 순환의수동적인 증가를 일으킨다. 그러나 과도한 전신동맥압의 증가는 유해하며 과도한 관류, 뇌부종, 심지어 뇌출혈까지 유발할 수 있다.

7) 뇌의 수분 전해질 균형

뇌는 체액의 균형과는 독립적으로 수분, 전해질 균형을 조절할 뿐만 아니라 전신의 수분, 전해질 조절하는 호르몬이 생성되는데 심방나트륨이뇨펩티드의 증가가 오게 되어 물 축적이지연되게 된다.

8) 생화학적 변화

뇌손상의 대부분은 뇌혈류 속도의 감소에 의한 뇌손상이다. 젖산증, free radical 생성, 세포 내 산화-환원 전위 변화, 세포자멸사 단계의 활성화, 교세포기능 및 이온항상성의 변화, 세포 내 에너지 물질의 운반과 사용의 장애

, 흥분독성등은 뇌에 손상을 줄 수 있다.



뇌지주막하출혈의 수술 전, 후치료

1. 진단

1) 컴퓨터 단층 촬영(CT)

첫 3일 이내에 거의 100%에서 지주막하출혈을 진단

그러나 출혈 5일후 85%, 7일후 50%, 2주후 30%정도 진단

수두증 : 21%에서 뇌동맥류파열 후 뇌실 크기의 증가

Talbe.1. 뇌단층 촬영에서의 뇌동맥류 파열에 따른 뇌출혈 소견

뇌 동 맥 류 뇌 출 혈 위 치	중대뇌동맥 (MCA)	전대뇌동맥 (distal ACA)	전교통동맥 (A-COM)	내경동맥 분지부 (ICA Bifurcation)	후교통동맥 (P-com)	뇌기저동맥 (Basilar artery)	후하소뇌동맥
	ICH	ICH	ICH	ICH			
	Sylvian fissure, temporal lobe	Anterior interhemispheric fissure, septum pelucidum, Cingulate gyrus	Gyrus rectus	Basal ganglia	Medial Temporal lobe, Uncus	Interpeduncular cistern	CP angle
	IVH(ICH 연장선상으로 발생 가능)	IVH(ICH 연장선상으로 발생 가능)	IVH (Lamina terminalis)			IVH(3 rd ventricle floor)	

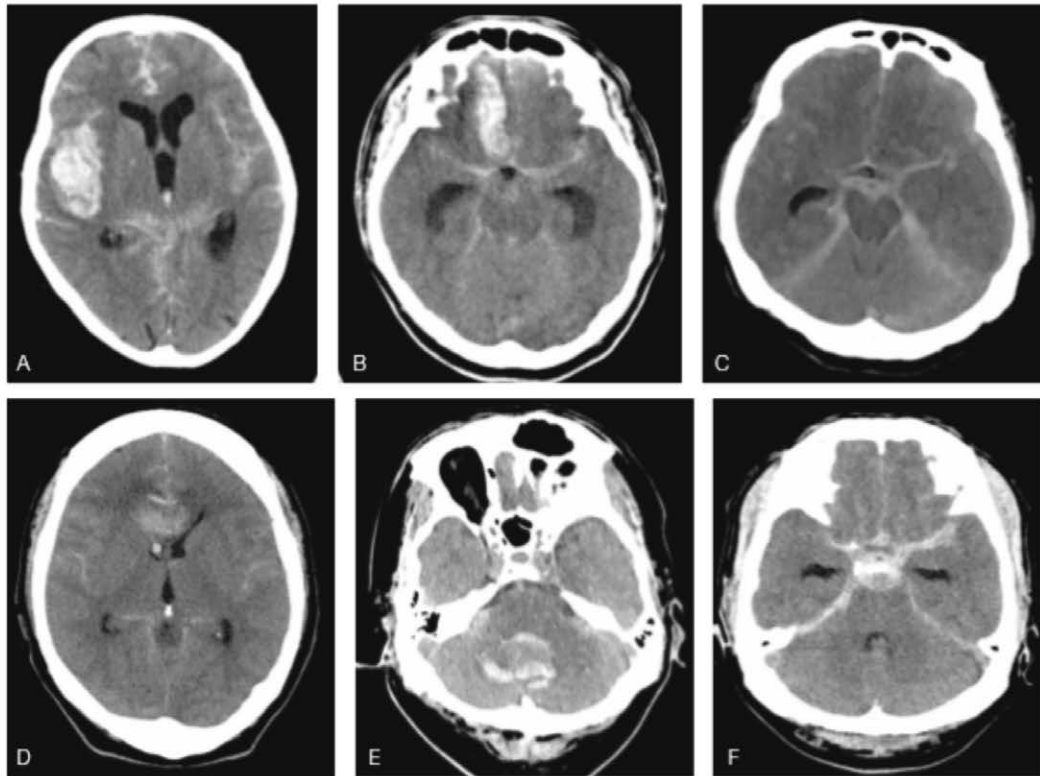


그림 35-1. 파열된 동맥류의 위치에 따른 CT 양상. (A) 우측 중대뇌동맥류 파열로 인한 측두엽의 뇌실내출혈, (B) 전교통동맥류 파열로 인한 전두엽의 뇌실내출혈, (C) 기저동맥 분지부 동맥류 파열로 인한 다리사이수조(interpeduncular cistern)에 우세한 지주막하출혈, (D) 원위부 전대뇌동맥류 파열에 의한 전두엽의 뇌실내출혈, (E) 후하소뇌동맥류 파열에 의한 소뇌 및 제4뇌실 내 출혈, (F) 후교통동맥류 파열에 의한 기저수조(basal cistern)에 우세한 지주막하출혈

2) 요추 천자

뇌지주막하 출혈에 대한 가장 민감한 검사

임상 증상으로 볼 때 뇌지주막하출혈이 의심되나 단층 촬영에서 보이지 않는 경우 시행

금기: 혈액응고이상, 두개강내압상진, 척추동정맥기형의심, 요추 천자부위 감염등

합병증: 뇌압의 감소에 따른 뇌동맥류 재출혈 또는 뇌탈출로 인한 신경학적 악화 소견

진단 ; 뇌척수액 황색변조증(xanthochromia) Hb 에서 유래한 빌리루빈에서 기인

적혈구가 검출

* 드물게는 뇌실질내 뇌동맥류 파열이나 소엽으로 갈라진 지주막 공간 일 때는 뇌척수액 에서 적혈구가 검출되지 않을 수도 있다.

* CTA or MRA : CT가 정상이면서 적혈구는 검출되지만 황색 변조증이 없으나 증상 발생 후 12시



간이 경과한 경우

지주막하 출혈과 외상성 천자(Traumatic tap)과의 감별

감별점	외상성 천자	뇌지주막하 출혈
적혈구 수	연속된 관에서 감소	연속된 관에서 일정
혈전	있음	없음
황색 변조증	없음	있음
적혈구/백혈구 비	정상	감소
단백질	정상	증가
뇌척수압	정상	상승
다른부위에서의 반복 천자	정상	지주막하 출혈 존재

3) 자기공명 영상 (MRI)

지주막하 출혈이 5-7이 지난 경우나 뇌단층 촬영에서 음성이 나오는 경우

자기공명 영상 민감도: FLAIR 급성기: 81%, 아급성기: 87%

T2 gradient echo 급성기: 94%, 아급성기: 100%

이외에, proton density, DWI

출혈 후 24-48시간 이내에는 출혈과 뇌조직과 구별되지 않으므로 부적합한 검사이나 두경부 경계, 뇌실질 출혈등 비전형적인 출혈 양상을 보이거나 증상 발생후 시일이 경과한 경우 유용함.

혈관 연속시 뇌허혈상태 일찍 확인가능

다발성 뇌동맥류인 경우 파열된 동맥류 확인에 도움

4) 뇌혈관 조영술

뇌동맥류 진단에 가장 정확한 방법 (gold standard)

위치, 형태, 크기, 돌출 방향 및 동반질환 확인

뇌혈관 조영술이 절대적으로 필요한 경우:

복잡 뇌동맥류,

동반 혈관 병변(AVM, dural AV fistular),

혈관내 수술을 고려하는 경우, CT에서 SAH가 넓게 퍼져 있거나 출혈 후 의식소실이

관찰되나 CTA에서 뇌혈관이상이 확인되지 않는 경우

뇌혈관 조영술 시행 가이드

가장 의심되는 혈관부터 조영술을 먼저 시행하는 것이 원칙임.

Both ICA, VA, ECA 총 6개 혈관 조영술 시행하여 추가적인 동맥류 또는 측부순환 확인 필요

한쪽 V.A. 조영제 주입시 반대측 VA 혈류 역류로 양측 PICA 조영이 되지 않는 경우 꼭 반대측 VA 조영하여야 한다.

혈관 조영술 음성인 경우(9-30%) 2주후 혈관 조영술 다시 시행 - 전교통 동맥류가 가장 흔함.

중뇌주위 지주막하 출혈(Perimesencephalic SAH)

혈관 조영술에서 음성으로 보이며 대부분 양성의 경과를 보임.

심한 두통이 있고 출혈의 양상은 대칭적임.

각간조(Interpeduncular cistern), 전뇌교조(prepontine), 뇌각조(crural), 중간뇌주위수조(ambient cistern)에 국한 됨.

명백한 뇌실질내 출혈은 존재하지 않으며 실비우스 지주막출혈도 잘 관찰되지 않음.

증상에 따른 치료를 하면되며 칼슘길항제와 항경련제 치료는 필요 없다.

반복적인 혈관 조영술은 필요 없다.

다발성 뇌동맥류에서 파열된 뇌동맥류 가능성 선택 고려점

지주막하 출혈이 많은 곳

종괴 효과를 보이거나 국소적으로 뇌혈관 연축이 보이는 곳

동맥류가 더 크고 모양이 불규칙한 것

국소 신경학적 증상과 일치하는 곳



근위부에 위치한 것

반복적인 검사에서 크기가 변하는 것

전교통동맥 동맥류가 파열이 더 많음.

경막외 동맥류는 배제 한다.

5) 자기공명 혈관 조영술

90%에서 뇌동맥류 진단 가능

6) 단층 혈관 조영술(CTA)

민감도: 77-97% 특이도: 87%-100%

뇌동맥류크기가 3mm이하에서의 민감도 : 40-90%

고령의 환자나 심한 사지 혹은 대동맥 죽종환자에서 혈관 조영술 대신 사용

이웃한 골굴조와 관계확인하여 수술하는데 도움 줄 수 있음.

두개저부위 골의 인공음영이나 재구성 기법상의 문제로 뇌동맥류 발견 안 되는 경우도 있음.

2. 임상 등급

G.C.S : 두통의 정도와 의식 손상 정도를 반영하는데 다소 주관적

WFNS ; 신경학적 결손을 기반

Fisher : CT에서 지주막하 출혈의 양상을 기술한 것으로 뇌혈관 연속 가능성 예측 위해 고안됨

Modified Fisher : Fisher의 주관적이라는 문제를 해결하기 위해 고안됨.

표 35-2. Hunt-Hess Grading System

Category	Criteria
I	Asymptomatic, or minimal headache and slight nuchal rigidity
II	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
III	Drowsiness, confusion or mild focal deficit
IV	Stupor, moderate to severe hemiparesis, possibly early decerebrate rigidity and vegetative disturbance
V	Deep coma, decerebrate rigidity, moribund appearance

표 35-3. WFNS Grading system

Grade	GCS score	focal deficit
I	15	Absent
II	13~14	Absent
III	13~14	Present
IV	7~12	Present or Absent
V	3~6	Present or Absent

WFNS: World Federation of Neurological Surgeons, GCS: Glasgow coma scale

표 35-4. Fisher scale & modified Fisher scale (radiological scales for the estimation of delayed ischemia in aneurysmal SAH)

Grade	Fisher Scale	Modified Fisher Scale
0	-	No SAH or IVH
1	No SAH or IVH	Minimal/thin SAH, no IVH in both lateral ventricles
2	Diffuse, thin SAH, no clot > 1 mm in thickness	Minimal/thin SAH with IVH in both lateral ventricles
3	Thick layer of SAH or localized subarachnoid clot > 1 mm in thickness	Thick SAH, no IVH in both lateral ventricles
4	Predominant IVH or intracerebral hemorrhage without thick SAH	Thick SAH with IVH in both lateral ventricles

IVH: intraventricular hemorrhage, SAH: subarachnoid hemorrhage

3. 기본적인 치료

1) 일반적인 처치

가족력, 과거력의 확실한 문진

기본적인치료와 함께 재출혈 및 혈관 연축을 막는 치료가 병행되어야 함.



수술 전까지 중환자실에서 집중관찰 및 시행하여야 할 것

지속적인 심전도

최소 한시간 간격으로 GCS, N/Ex, BP, Temp. L/R, I/O

I/O 확인위한 Foley catheter삽입.

하루 3L 이상의 수분 섭취 -뇌허혈 위험 방지, 저나트륨혈증이나 발열환자에서도 정상 혈량유지

Hematocrit : 뇌로의 산소 운반 능력 유지위해 30%이상 유지

혈관 연축시 고체온증, 저혈량, 뇌압상승, 저마그네슘혈증, 저나트륨혈증 피하고 경구로 니모디핀 60mg 4시간 간격으로 경구 투여

니모디핀 유지기간은 지주막 출혈 후 21일간 유지 권장

니모디핀 부작용 : 두통, 저혈압, 장내 가성 폐색 등

저혈압발생 시 용량은 적절히 줄여줌

대변 완화제 및 구토 억제제 사용

아세트아미노펜 및 모르핀등으로 통증 조절

위장관 출혈을 막기위한 제산제, 히이스타민 수용체 길항제, 프로토펙트릭억제제 사용

2) 혈압조절

뇌관류압을 유지하기 위해 평균동맥압은 적어도 90mmHg이상 유지

수축기혈압을 160-180 mmHg보다 낮게 유지하는 것을 목표로 함.

항고혈압제 사용은 적정 가능한 정맥투여를 통하여 시행.

4. 지주막하 출혈에 특이적인 합병증

1) 재출혈

재출혈시 사망률 : 70-90%

재출혈 위험률 : 첫 2-12시간에서 가장 높고 첫 24시간 이내에 4-13.6% 발생

재출혈 환자의 1/3 이상의 환자에서 3시간 이내에 재출혈 발생 거의 절반정도는 6시간 이내에 발생하며 이후 재출혈 위험율은 하루 평균 1.5%씩 증가하여 재출혈 누적 위험율은 35-40%정도임.

재출혈이 잘 일어나는 경우

내원시 임상 등급이 나쁜 환자

첫 출혈 시 의식 소실

파수뚫두통의 경험(sentinel headache)

큰동맥류

수축기 혈압이 160mmHg이상인 경우

수술 시기

조기수술은 뇌지주막하 출혈후 72시간 내 지연수술은 1-2주 경과 후에 시행

수술 시기의 선택 논란은 있으나 출혈량이 많은 경우 응급수술 고려

조기 수술의 장점 ; 재출혈 감소, 혈관 연축 빈도 감소, 혈관 연축에 대한 치료유리, 수술시 혈종과 동맥류박리와 혈종제거가 상대적으로 쉽고 환자를 조기에 활동할 수 있음.

조기수술의 단점; 수술시 뇌의 견인이 힘들고 동맥류 박리시 조기 파열의 위험이 있음.

약물적 치료

항피브린용해제 사용은 진단후 수술전까지만 사용하는 것이 일반적임.

2) 뇌혈관 연축

뇌혈관의 직경이 원인이 불분명하게 감소하는 현상

사망 및 신경학적 장애의 주원인임.

뇌동맥류 파열에 의한 뇌지주막하 출혈 후 뇌혈관 연축:

파열후 4-14일 사이에 발생 주로 6-10일 사이에 가장 많이 발생, 21일 이후에 저절로 호전

환자의 2/3에서 혈관 검사에서 혈관 연축이 있고 이중 절반에서 혈관 연축의 증상이 보임.



뇌혈관 연축의 위험인자(risk factor for vasospasm)

Thick subarachnoid clots
Intraventricular hemorrhage
Persistent subarachnoid clots(slow clearance)
Poor neurological condition on admission
Loss of consciousness associated with rupture
History of cigarette smoking
Preexisting hypertension
Diabetes mellitus
Cocaine use

1. 혈관 연축의 병리 및 병태 생리

1) 구조적인 변화 : 평활근 수축

평활근 수축으로 동맥 중간막의 비후 및 내피와 내부 탄성층에 주름이 잡히는 조직학적 변화

연축은 지주막 출혈시 Hb에 의해 촉발되며 칼슘과 ATP에 의존하는 평활근 수축이고 만성 혈관연축은 Protein kinase C, Rho kinase, Protein tyrosine kinase이 관여한다.

2) 혈액 부산물

산화 혈색소: 혈관 연축을 일으키는 정확한 기전은 알려지지 않았음.

혈관 벽에서 에이코사노이드의 합성을 변화시켜 혈관 수축시키는 PGE2증가 유발

스스로 산화되어 metHb으로되면서 superoxide발생 시키고 지질과산화와 혈관 수축 유발

3) 혈관 내피 손상, 이산화질소(NO), Endothelin-1(ET-1)

혈관 내피 손상; 혈관 확장제인 endothelial NO 합성의 소실 또는 강력한 혈관 수축 물질인 ET-1의 과생성을 통해 혈관 연축을 발생

4) 염증

직접적인 상관 관계를 확신할 수 없으나 뇌척수액내 호주의 비율(>62%)이 혈관 연축을 예측할 수 있는 독립적인자라고 보고됨.

2. 지연성 뇌허혈의 병태 생리

1) 혈관 연축

자연성 뇌경색의 25%정도는 혈관 연축과 관련없이 유발되는 경향이 있음.

그러나 혈관 연축이 자연성 뇌허혈의 주요 원인임.

2) 초기의 뇌손상

출혈 후 첫 72시간 중요시기로 뇌압 조절위해 고혈압 조절이 필요

3) 미세 혈전증

혈액응고 촉진인자 활성화는 자연성 뇌허혈에 선행되고 혈소판 활성화 인자의 증가는 뇌지주막하 출혈 후 4일째 관찰

항섬유소용해치료는 재출혈을 막을 수 있는 인자이지만 자연성 뇌출혈을 일으킬 수 있음.

4) 뇌피질 확산 탈분극

피질 신경부종, 가지돌기가시 변형, 뇌전위 변화, 뇌전기활동도 감소등을 반영하고 출혈반응과 연관이 있다.

5) 혈뇌장벽손상

6) 염증

7) 뇌자동조절

3. 진단

증상 - 점진적일 발병하는 양상 두통의 증가, 졸음 및 흥분 등의 전조 증상 보임.

중대뇌동맥 영역: 단사지마비나 편측 마비 우성 반구 침범시 실어증

전대뇌동맥 영역: 하지 위약감

혼돈, 기면, 언어감소, 의지결여

후방순환계 : 의식 저하 먼저 보이고 위약감 발생

악화 요인 : 부종증가, 재출혈, 수두증, 폐혈증, 저나트륨혈증, 저산소증, 저혈압

진단 검사

1) 경두개 초음파



중대뇌동맥 혈류 속도: 120cm/s이상이면 혈관 연축의미 200cm/s이상이면 심한 혈관 연축

진단적 민감도: 90% 특이도: 71%, 양성 예측도: 57%, 음성 예측도 : 92%로 초기 선별검사로 매우 유용함.

2) 뇌혈류 및 관류

SPECT, Xenon-CT, PET등은 뇌허혈 진단 가능

임상적으로 유용하지 않음.

Perfusion CT : 뇌혈류의 감소와 MTT연장, Cerebral blood volume의 상태를 초기에 제공하여 혈관 연축의 민감도는 74%, 특이도는 93%임.

3) 혈관 영상

CTA: 민감도 특이도 80%이상에서 연축 확인됨.

4) 치료

a. adequate intravascular volume(euvolemia)

b. Induced hypertension is the most effective

c. Pharmacological management

Calcium-channel blockade with nimodipine is given to all patients.

The role of oral nimodipine in the prevention or treatment of delayed ischemic events

little effect on the incidence and severity of angiographic vasospasm.

Anti-ischemic effects of nimodipine and nicardipine may occur by inhibiting calcium entry into smooth muscle cells and vasoactive substance release from platelets and endothelial cells. In addition, calcium antagonists may favor the development of collateral circulation. They also may be neuroprotective.

Nimodipine, administered orally at a dose of 60 mg every 4 h and maintained for 3 weeks, usually is well tolerated, even if its use might be associated with systemic hypotension especially when intravenously administered.

Other pharmacological management - Lack of evidenced base

1. Magnesium

based on its action as a noncompetitive calcium antagonist via binding to voltage-dependent calcium channels

SAH hypomagnesemia occurs in more than 50% of the patients,

investigated as a possible neuroprotective agent

several preclinical studies have demonstrated that magnesium can provide vasodilation by inhibiting the cellular calcium influx, inhibiting vascular smooth muscle contraction, and decreasing glutamate release.

***current recommendation** is to administer magnesium as necessary to prevent hypomagnesium in SAH-affected patients.

2. Statins

Cerebral arterial blood vessel tone is balanced by vasoconstrictor and dilator systems designed to achieve equilibrium

in physiological conditions, there is a balance between vasodilator and constrictor mechanisms in the control of the cerebrovascular tone. It has been suggested that statins upregulate endothelial nitric oxide synthase and the availability of endogenous nitric oxide by inhibition of 3-hydroxy-3-methylglutaryl coenzyme A reductase. By this mechanism, statins would correct the imbalance between the nitric oxide and endothelin pathways, which is believed to be a main contributor to the pathophysiology of cerebral vasospasm.

neuroprotective action by decreasing the glutamate-mediated excitotoxicity, reducing the production of reactive oxygen species, and modulating the inflammatory response

phase II randomized, double-blind, placebo-controlled study did not show significant differences in the outcome between simvastatin and placebo-treated patients.

3. Endothelin receptor antagonists

found to be effective in reducing the incidence of vasospasm

clazosentan produced dose-dependent reductions in angiographic vasospasm.

in the CONSCIOUS-2 study, poor functional outcomes occurred more frequently in the clazosentan-treated patients, whereas the mortality between the groups was the same. Furthermore, the occurrence of adverse events in the clazosentan group (i.e., pulmonary edema, hypotension, cerebrovascular spasm, pleural effusion, and cerebral infarction) halted the study and prevented the CONSCIOUS-3 to be further continued.

4. Tirilizad

Tirilizad is a nonglucocorticoid 21-aminosteroid that functions as a free radical scavenger by inhibiting lipid peroxidation. This antioxidant effect is believed to provide neuroprotection also during SAH.

reduce angiographic vasospasm following SAH.

Although it has been shown to reduce the frequency of secondary symptomatic vasospasm in patients with aneurysmal SAH, its role in the incidence of cerebral infarction as the result of symptomatic vasospasm still remains unresolved. Tirilizad can be administered without risk of cardiovascular and mental adverse effects. The most common adverse event of tirilizad reported



was phlebitis.

5. Erythropoietin

a 165 amino acid (~30 kDa) glycoprotein, member of the type I cytokine superfamily, which regulates the differentiation and proliferation of immature erythroid cells.

erythropoietin mediates neuroprotective effects by different mechanisms of action including maintaining normal vascular autoregulation

As a result, although no differences were demonstrated in the incidence of vasospasm and adverse events between the two groups, patients receiving erythropoietin had a decreased incidence of severe vasospasm, reduced DIDs, a shortened duration of impaired autoregulation, and more favorable outcome at discharge.

Potential adverse effects, however, should be considered. Several lines of evidence suggest that chronic erythropoietin administration can produce hypertension, hypertensive encephalopathy, accelerated atherosclerosis, seizures, and thrombotic/vascular events. acute ST-segment elevation myocardial infarction (STEMI) was associated with higher rates of adverse cardiovascular events among older patients.

the dosage used in the clinical setting is the lowest dose considered to be effective following SAH

6. Glyburide

oral anti-diabetic drug that has recently shown neuroprotective properties.

glyburide can act through the inhibition of SUR1, a membrane protein that co-associates with heterologous pore-forming subunits to form ion channels.

Opening of SUR1-Trpm4 channels is associated with excess influx of Na⁺, which is accompanied by influx of Cl⁻ and H₂O, resulting in cytotoxic edema or cell death.

Recently, patients with type 2 diabetes affected by ischemic stroke and taking a sulfonylurea drug for glycemic control presented with significantly fewer deaths and a significantly lower rate of symptomatic hemorrhagic transformation. These findings have been recently confirmed by the preliminary results of a multicenter, randomized, double-blind, phase II trial that examined the efficacy of glyburide in the prevention of malignant edema in severe anterior circulation ischemic stroke

3) 수두증

급성 수두증 발생 빈도; 12-87%

만성 단락 의존성 수두증 빈도 : 8.9 – 48%

급성 수두증 예측 인자 : 뇌실내 출혈과 지주막하 출혈이 많은 경우

급성 수두증의 치료

EVD 또는 Lumbar drainage

EVD후 뇌압 유지 ; 재출혈 방지위해 15-25mmHg범위로 유지

수두증의 증상 ; 1/3에서는 무증상, 초기 수두증과 의식 저하가 있는 환자 1/2에서는 자연적으로 호전되기도 함.

EVD제거는 24시간 이상 걸쳐 서서히 떼는 것이 필요

수술시 Lamina Terminalis 개통: 단락 의존성 수두증 발생을 감소 도움 안되므로 시행하지말 것

증상이 있는 만성 수두증의 치료

뇌실 단락술 등의 영구적인 뇌척수액 전환

4) 발작 (Seizure)

발작 빈도 : 지주막하 출혈의 20%

출혈 직후 7%, 출혈후 수주 이내에 105정도

경련성 간질지속증 : 0.2%

발작이 발생한 경우 : 재출혈, 뇌허혈, 수두증 또는 저나트륨 혈증 고려

발작의 효과 ; 뇌의 산소 소비를 증가 시키고 저산소증 유발

고탄산혈증, 산증,흡인성 폐렴 유발, 혈압 상승에 따른 재출혈

간질의 정의 : 지주막하 출혈 후 적어도 1주 이내에 2번이상 발작이 발생한 경우 (3-10%)

발작의 발생률 : 6-8% 지연성 발작: 3-7%

발작의 발생률에 영향을 주는 인자 : 뇌동맥류 결찰술, 젊은나이. 수술후 신경학적 결손, 이전 과거력, 단락의존성 수두증. 임상 등급이 불량한 경우, 재출혈, 중대뇌동맥 동맥류, 뇌실질내 혈종 , 경막하 혈종, 대뇌피질의 경색, 수술중 내측 측두엽 견인.

항경련제 유지: 이전 간질 과거력, 뇌내 혈종, 피질 경색, 동정맥기형을 동반한 경우

5. 내과적 합병증

1) 발열



나쁜 임상 결과에 영향을 미치는 독립적 위험인자 - 인지 및 신경학적 결손 관여

비감염성 발열의 원인 ; 손상의 정도, 출혈량, 혈관 연축과 관련이 있고 혈액과 그부산물에 의한 전신적인 염증 상태

2) 빈혈

뇌로의 산소 전달을 떨어뜨릴 수 있으므로 혈관 연축이 없는 환자에게는 8mg/Dl, 혈관 연축이 있는 환자의 경우 10mg/dL이상 유지 이하인 경우 수혈을 고려

3) 고혈당

지주막 출혈의 1/3정도에서 고혈당증(>200g/dL) 발생

나쁜 임상 상태와 관련있고 허혈성 뇌손상과 관련

4) 호흡기 합병증

환자가 나이가 많거나 임상등급이 나쁜 경우 흔하게 보임.

지주막 출혈 사망 환자의 50%에서 폐합병증으로 사망함.

호흡기 합병증 종류: 폐부종, 폐렴, 무기폐, 흡인, 기흉, 천식, 폐색전증

폐부종 ; 심인성 (좌측 심장 부전에 의한 폐정맥압의 상승)

비심인성(신경인성 또는 흡인이나 폐렴에 이차적으로 발생)

폐부종 발생 가설 :

1. 갑작스런 뇌압 상승이 교감 신경계의 과활성화를 일으켜 폐동맥의 혈관 수축과 전신적인 혈압상승을 유도하고 혈관 수축이 폐정맥까지 발생할 경우 폐 모세혈관의 압력이 증가하고 그로 인해 정수압의 차이가 발생함. 이로 인해 저단백 체액이 폐로 누출됨.
2. 중추 신경계의 신경전달이나 폐 압력의 변화로 인해 폐 모세혈관의 투과성이 증가하여 단백질의 풍부한 혈장이 폐로 새어 들어간다는 것

신경인성 폐부종: 갑자기 발생하고 시상하부를 포함하는 심한 신경학적 손상과 관련이 있고 아드레날린 차단제의 의해 억제되고 부종액에 단백질이 많이 함유되어 있는 것이 특징임.

폐렴의 치료

기도 삽관의 경우가 많으므로 가능하면 비위관과 기도내관을 빨리 제거

손씻기 및 반좌위 자세

적절한 영양공급

적절한 항생제 사용

기관 절개술 시기 : 기도 삽관 10-14일 지나지 않거나 기관 제거가 바로 가능하지 않는 경우

5) 심혈관계 합병증

지주막하출혈 후 심전도 이상 소견 : QT간격 연장, U파형성, ST상승 또는 하강, T파 평탄화등

Neurogenic stunned myocardium : 가역적인 심장 손상으로 수축 밴드의 괴사 소견을 보이는 것

과도한 카테콜라민과 세포내 칼슘 분비로 심장의 과도한 수축

심장초음파 소견 : 심박출량 감소 정도는 8%정도

심박출량 감소 경우: 임상 등급이 나쁜 경우, 폐부종 동반 경우, 심한 저혈압, 심근 효소 상승

대부분 별치료 없이 호전

단, 대칭적인 T파 전위, 심한 QT연장이 있는 경우 심실의 기능이상 의심되므로 추적 심초음파

트로포닌 I 상승: 68%환자에서 보이며 신경학적 장애와 사망에 관련 있음.

출혈후 2일 경과시 최고조

좌심실벽 운동장애가 동반되는 경우 더 상승

심전도, 전해질, 특히 칼륨의 지속적 관찰 필요함.

6)정맥 혈전색전증 (DVT)

2%에서 발생

무증상의 심부정맥 혈전증 : 19-50%에서 발생

정맥혈전증의 약 50%에서 폐색전증이 동반되며 50%는 치명적임.



심부정맥혈전증 위험인자 :고령, 심부전, 이전 정맥혈전 색전증 과거력, 외상, 비만, 감염, 악성종양, 임신, 산욕기, 하지정맥류, 경구피임약, 4시간 이상의 수술, 상지 또는 하지 마비,

치료: 간헐적 사지 압박 , 저용량의 헤파린, 저분자량 헤파린. 항응고제 경구투여

7)수분 및 전해질 이상

정상 혈장량을 목표로 치료

하루수분 공급량이 배출량보다 500mL이상이 되지 않도록 조절

생리식염수 또는 lactated Ringer's solution 주입 : 1.5ml/kg/hr

전해질 이상 종류 : 저나트륨 혈증(14-40%) – SIADH, CSWS

저마그네슘혈증(37%)

저칼륨혈증(27%)

고나트륨혈증(20%)

저나트륨 혈증

증상 : 의식저하. 국소적 신경학적 이상의 발생 또는 악화, 발작, 실조증

SIADH와 CSWS 차이점 : 혈장량의 차이

: SIADH 항이뇨호르몬 부적절한 증가로 혈장량이 증가하는 것이고 CSWS는 혈중 심방 나트륨이

인자 (Atrial natriuretic factor)의 증가로 인한 나트륨 배설 증가로 염분이 소변으로 빠져나가

혈장량 감소

혈장량 변화 측정 방법 : Hct 변화, BUN/Cr비, 심장충만압의 측정

CSWS: 임상 등급이 나쁜 경우, 전교통동맥의 파열, 수두증환자에서 흔히 발생

치료는 적절한 수분과 염분을 보충하여 정상 혈장량과 정상 나트륨 수치를 유지

SIADH: 수분 억제제가 치료 원칙, 혈관 연축의 경우 고장 식염수와 염분 보충으로 치료

교정은 천천히 시행

표 35-5. 항이뇨호르몬부적절분비증후군(SIADH), 대뇌염류유출증후군(CSW), 요붕증(DI)의 특징

특징	SIADH	CSWS	DI
혈청 나트륨	< 135 mEq/L	< 135 mEq/L	다양(주로 상승)
혈장 오스몰농도	< 280 mOsm/L	< 280 mOsm/L	다양(주로 상승)
소변 나트륨	> 20 mEq/L	> 20 mEq/L	다양
소변 오스몰농도	> 혈장 오스몰농도	> 혈장 오스몰농도	50~150 mOsm/L
혈청 칼륨	감소 또는 정상	증가 또는 정상	정상
혈액량	증가	감소	정상 또는 감소
나트륨 균형	다양	감소(negative)	다양
체중	증가	감소	정상 또는 감소
심장충만압	증가 또는 정상	감소	정상 또는 감소
헤마토크리트	감소	증가	정상 또는 감소
Blood urea nitrogen/ creatinine	감소 또는 정상	증가	정상 또는 감소
혈압	정상	체위저혈압(postural hypotension)	정상 또는 감소
심박수	정상	빈맥	정상 또는 빈맥
기타	갑상선, 부신, 신기능 정상; 말초 부종 없음; 탈수 없음	혈장량 부족의 증상, 징후	소변량 > 3 L/일

SIADH: syndrome of inappropriate diuretic hormone, CSWS: cerebral salt wasting syndrome, DI: diabetes insipidus

요붕증(DI): 지주막하 출혈환자의 0.04%정도에서 발생, 전교통동맥 동맥류 파열과 관련

적절한 수분공급 만성기에는 항이뇨 호르몬 사용

8) 위장관계 합병증

뇌지주막하 출혈후 첫 3일 이내에는 교감 신경계 흥분으로 과다 대사 상태의 이화 반응이 주를

이루고 음성 질소 균형을 이루는데 이들은 체중감소, 감염의 위험성 증가, 상처 회복을 방해

일차적인 치료는 적절한 영양 공급이 중요 - 장관 영양이 좋다.

위와 상부 위장관 스트레스성 궤양 예방 위한 제산제와 라니티딘, 오메프라졸 사용

대변 완화제 사용 - 배변 힘주, 복통, 대변 막힘으로 인해 뇌압 상승 예방

니모디핀 합병증 - 위장관의 가성 폐색, 장마비등의 부작용 발생할 수 있고 장천공도 유발함.

9) 수술후 증상의 악화



악화 원인.: 혈관 연축이 가장 흔한 원인

내과적 합병증 및 신경학적 합병증 관리가 필요

7. 예후인자

중요 예후 인자: 환자의 나이, 신경학적 등급, CT에서의 출혈량, 고혈압 병력, 혈관 연축, 동반된 뇌실질내 출혈, 뇌실내출혈, 뇌동맥류의 위치

Table 1 Intensive care unit management of aneurysmal subarachnoid hemorrhage

Complication	Therapeutic measures
Before aneurysm treatment	
Hydrocephalus	Place external ventricular or lumbar drain
Aneurysmal rebleeding	Early aneurysm repair Tranexamic acid 1 g intravenously every 6 h not exceeding 48 to 72 h
Increased intracranial pressure	Elevate head of bed to 30° Control fever and agitation Short-term hyperventilation Osmotherapy (20% mannitol, hypertonic saline) Surgical evacuation/decompression for lesions with mass effect and clinical deterioration
Neurogenic stunned myocardium	β blockers
Neurogenic pulmonary edema	Increase level of positive end-expiratory pressure
After aneurysm treatment	
Delayed cerebral ischemia	Nimodipine 60 mg every 4 h
Vasospasm	Maintain euvolemia with crystalloids or colloids If symptomatic, induce hypertension with phenylephrine, norepinephrine, or dopamine If refractory, provide endovascular treatment (transluminal angioplasty or intra-arterial vasodilators)
Hyponatremia	Strict avoidance of free water Hydration with hypertonic saline solutions (1.5% or 3%) Fludrocortisone acetate up to 0.2 mg twice daily
Hyperglycemia	Use insulin to maintain normoglycemia (5.6–10 mmol/L)
Fever	Maintain normothermia with acetaminophen, cooling blankets, or surface cooling devices

참고 문헌

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KSCVS-SKEN 합동 연수강좌

인 쇄 2018년 9월 13일

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Reference 1. Finnerup, Nanna B., et al. "Pharmacotherapy for neuropathic pain in adults: a systematic review and meta-analysis." The Lancet Neurology 14.2 (2015):162-173

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