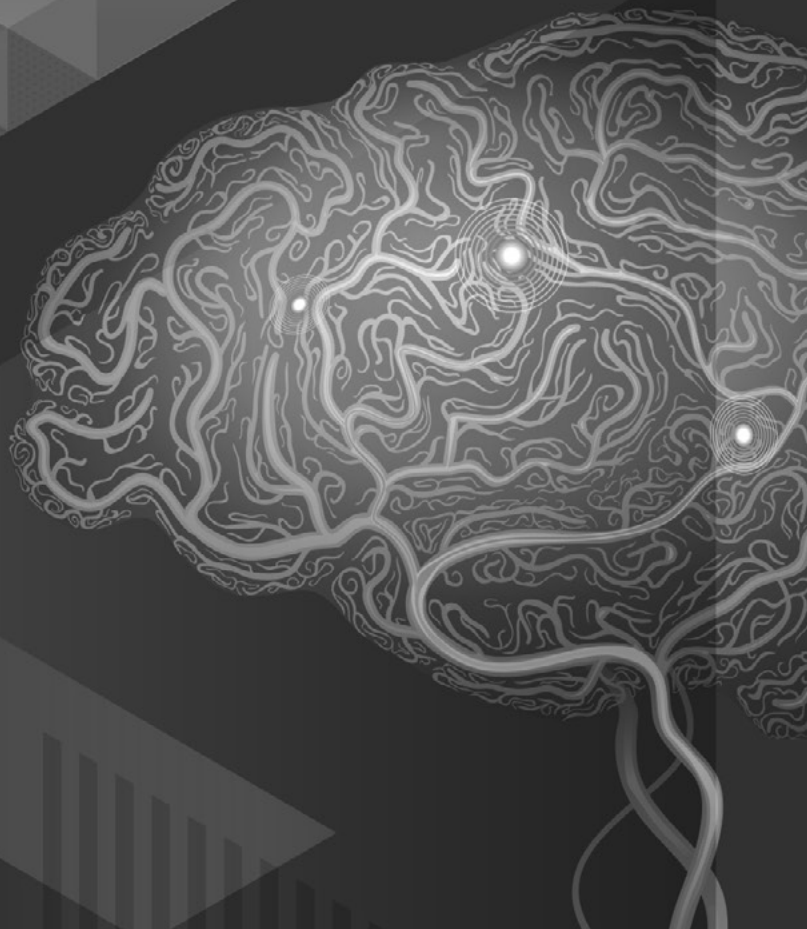


2021 대한뇌혈관내 치료의학회 춘계보수교육

일시 | 2021년 2월 27일(토)

장소 | 판교 차바이오컴플렉스 지하1층 국제회의실



모시는 글

존경하는 대한뇌혈관내치료의학회 회원 여러분!

안녕하십니까? 신축년 흰 소띠의 해를 맞아 회원 여러분과 가정에 건강과 행복이 가득하기를 기원합니다. 작년 한 해는 코로나-19 대유행으로 평범했던 일상조차 얼마나 소중한지를 느끼는 한 해였습니다. 이렇게 특별한 상황에서 환자 진료와 교육, 연구에 매진하고 계신 회원 여러분께 존경과 경의를 표합니다. 새해를 맞아 정부의 방역수칙 강화에도 불구하고 아직 어려운 상황이 지속되고 있으나 수련 교육을 게을리할 수는 없기에 매년 해오던 춘계 보수교육을 온라인을 병행하여 시행하고자 합니다.

이번 춘계 보수교육의 주제는 Dural AVF입니다. 경막동정맥루는 발생 빈도가 드문 질환으로 환자 치료 경험이 제한적일 수밖에 없기에 병변을 이해하고 치료 술기를 습득하는데 적지 않은 어려움이 있을 것입니다. 이러한 어려움을 극복하는데 도움이 되고자 이번 보수교육에서는 경막동정맥루의 발생 기전과 치료 시 고려해야 할 측부순환, 그리고 병변 위치에 따른 치료의 특성 등 동정맥루와 관련된 전반적인 사항을 다루는 프로그램을 준비하였습니다. 그리고, 이 분야의 저명한 두 분 교수님을 초청 연자로 모셔서 특강을 듣는 “Meet the Experts” 세션을 마련하였습니다. 우리나라 뇌혈관내치료 분야의 선구자이신 한문희 교수님의 명강의를 들을 수 있을 것이며, 동정맥루에 대한 해박한 지식과 풍부한 경험을 갖춘 일본 Toyama 대학의 Kuwayama 교수님의 온라인 강의를 흥미로울 것입니다. 이를 통해 동정맥루를 보다 더 잘 이해하게 될 것이며, 치료의 지평을 넓힐 수 있는 기회가 될 것입니다.

또한 뇌졸중 시술 인증제를 어떻게 국가에서 필요로 하는 시스템으로 발전시킬 수 있을지 생각해 볼 것이며, 향후 추진할 가능성이 높은 지역심뇌혈관센터 지정 및 육성 사업과 관련하여 의료 현장의 현실을 반영한 우리 학회안을 구상해 보는 시간을 마련 하였습니다. 회원 여러분의 관심과 많은 참여 부탁드립니다.

마지막으로 이번 춘계 보수교육을 준비하느라 수고해 주신 임원 여러분과 어려운 상황에서도 항상 우리 학회에 관심과 성원을 보내 주시는 기업 관계자 여러분께 감사드립니다.

모쪼록 건강에 유의하시고 건강한 모습으로 2월 27일에 뵙기를 고대합니다. 감사합니다.

대한뇌혈관내치료의학회 회장 윤석만 드림

2020~2021 대한뇌혈관내치료의학회 임원진

명예회장

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명예회장	백민우	인봉의료재단 뉴고려병원
	권도훈	울산대학교 서울아산병원

회장

직위	성명	소속
회장	윤석만	순천향대학교 천안병원
부회장	장철훈	영남대학교병원

상임이사

직위	성명	소속
총무	박석규	순천향대학교 서울병원
학술	권순찬	울산대학교병원
정책	신승훈	차의과대학교 분당차병원
재무	김영우	가톨릭대학교 의정부성모병원
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간사	오재상	순천향대학교 천안병원

전임회장단

직위	성명	소속
초대, 제2대	백민우	인봉의료재단 뉴고려병원
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제6대	안성기(작고)	(전) 한림대학교 성심병원
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제8대	권오기	분당서울대학교병원
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제11대	고준석	강동경희대학교병원

운영위원

직위	성명	소속
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	조병래	가톨릭대학교 은평성모병원
	문종현	광주기독병원
	전효섭	강원대학교병원
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인증관리위원회	이호준	가톨릭대학교 성빈센트병원
	신동성	순천향대학교 부천병원
	신희섭	경희대학교 강동병원
	전홍준	한림대학교 강동성심병원
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	김태곤	차의과대학교 분당차병원
	황교준	한림대학교 한강성심병원
	정준호	연세대학교 세브란스병원
	오재상	순천향대학교 천안병원
국제교류위원회	남택균	중앙대학교병원
	박정수	전북대학교병원
	신희섭	경희대학교 강동병원
	이동훈	가톨릭대학교 성빈센트병원
	정영진	영남대학교병원



Naoya Kuwayama, M.D., Ph.D.

Title

Professor, Division of Neuroendovascular Therapy

Associate Professor, Department of Neurosurgery, University of Toyama, Japan

Education

1976 - 1981 Medical Faculty, University of Tsukuba, Japan

1982 Medical Doctor

1988 Certified neurosurgeon (specialist of neurosurgery)

2001 Certified endovascular neurosurgeon (senior instructor)

Academic Appointments

1985 - 1994 Assistant professor, Dept. of Neurosurgery, University of Toyama

1994 - 2006 Lecturer, Dept. of Neurosurgery, University of Toyama

2006 - Present Associate professor, Dept. of Neurosurgery, University of Toyama

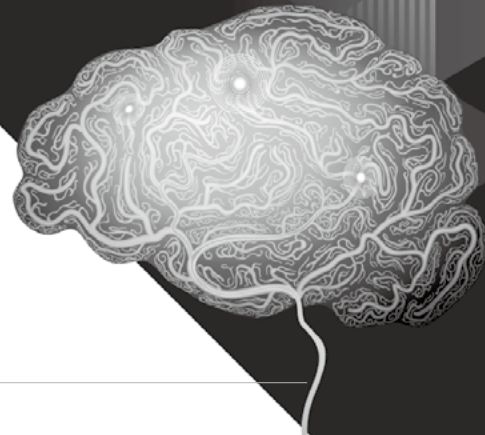
2007 - Present Professor, Div. of Neuroendovascular Therapy, University of Toyama

Academic Society

2001 - Present Board member of The Japanese Society of Neuroendovascular Therapy

2008 - 2009 President of Japanese Society of Neuroendovascular Therapy (JSNET)

[PROGRAM]



08:30-09:15	Registration		
09:15-09:30	Course Introduction	김태곤 (대한뇌혈관내치료의학회 수련교육이사)	
	Opening Remark	윤석만 (대한뇌혈관내치료의학회 회장)	
	Welcome Address	이일우 (대한신경외과학회 회장)	
09:30-11:00	Session I. Anatomy and pathophysiology of dural AVF 좌장: 성재훈 (가톨릭대), 장인복 (한림대)		
09:30-10:00	Pathophysiology of dural AVF, classification	심숙영 (인제대)	11
10:00-10:30	Pathological vascular anatomy for dural AVF	조수희 (울산대)	19
10:30-11:00	Clinical features and natural history, treatment indication	송지혜 (아주대)	30
11:00-11:20	Coffee break		
11:20 - 12:10	Special symposium 좌장: 윤석만 (순천향대), 고준석 (경희대)		
11:20 - 11:40	KoNES 뇌졸중 시술 인증제의 발전 방향	신희섭 (경희대)	43
11:40 - 12:10	KoNES 뇌혈관센터 구성안	신승훈 (차의과학대)	50
12:10 - 13:10	Photo time & Lunch		
13:10-14:40	Session II. Endovascular treatment of dural AVF 좌장: 신용삼 (가톨릭대), 하성곤 (고려대)		
13:10-13:40	Dural AVF of the cavernous sinus	권순찬 (울산대)	53
13:40-14:10	Dural AVF of transverse-sigmoid sinus, tentorial, clival, etc	이창영 (계명대)	54
14:10-14:40	Spinal AVF	강현승 (서울대)	55
14:40-15:00	Coffee break		
15:00-16:30	Session III. "Meet-the-Expert" session 좌장: 백민우 (뉴고려병원), 김범태 (순천향대)		
15:00-15:45	Endovascular treatment of dural AVF	한문희 (중앙보훈병원)	63
15:45-16:30	Endovascular treatment of dural AVF	Naoya Kuwayama (University of Toyama, Japan)	70
16:30-17:30	Session V. Interesting or complicated cases presentation of dural AVF 좌장: 권오기 (서울대), 허준 (명지성모병원)		
16:30-16:45	Case Presentation 1	박근영 (연세대)	81
16:45-17:00	Case Presentation 2	임종국 (제주대)	86
17:00-17:15	Case Presentation 3	고정호 (단국대)	87
17:15-17:30	Case Presentation 4	신희섭 (경희대)	96
17:30	Closing Remark	김태곤 (대한뇌혈관내치료의학회 수련교육이사)	

2021 대한뇌혈관내 치료의학회 춘계보수교육

Session I. Anatomy and pathophysiology of dural AVF

좌장: 성재훈 (가톨릭대), 장인복 (한림대)

Pathophysiology of dural AVF, classification

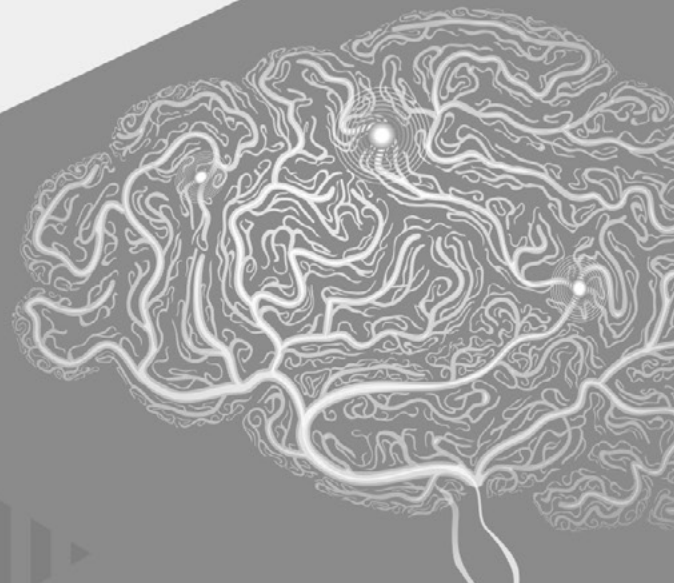
Pathological vascular anatomy for dural AVF

Clinical features and natural history, treatment indication

심숙영 (인제대)

조수희 (울산대)

송지혜 (아주대)



Pathophysiology and Classification of Dural AVF

심숙영

인제대 일산백병원 신경과

Intracranial Dural AVF

Incidence

Dural arteriovenous fistulas (DAVFs) are pathologic shunts between pachymeningeal arteries and dural venous channel. DAVFs are distinguished from parenchymal or pial arteriovenous malformations by the presence of a dural arterial supply and the absence of a parenchymal nidus [1]. The incidence of cranial DAVFs has been estimated 0.15~0.29/100,000 per year [2] and the intracranial location is reported as transverse sinus (50%), cavernous sinus (16%), tentorium cerebelli (12%), and superior sagittal sinus (8%) [3]. DAVFs are relatively rare, accounting for 5–15% of all cranial vascular malformation. However, DAVFs can lead to significant morbidity and mortality due to intracranial hemorrhage and non-hemorrhagic neurologic deterioration related to leptomeningeal venous drainage [3, 4].

Etiology

The etiology of DAVFs is not fully understood, but the majority of DAVFs in adulthood appear to be acquired. Different etiologies have been implicated such as trauma, previous craniotomy, and a hypercoagulable state including malignancy, pregnancy, infection, and prothrombotic genes [5–7]. DAVFs in the pediatric population are thought to be associated with congenital venous anomalies of the developing venous system or a result of birth trauma, infection, in utero venous thrombosis, or maternal hormones [8].

Angioarchitecture associated with DAVFs

A meningeal branch of each artery supplying the brain or spinal cord consistently pierce the dura through a venous sinus or else are surrounded by a venous plexus. When meningeal arteries running outside the dural layers penetrate the dura, they come much closer to meningeal veins or venous sinuses running between outer periosteal and inner meningeal layer of dura.

In normal dural sinus [9], dural vessels were more numerous at angles formed by a fusion of two dural leaves, which were rich in veins of varying sizes communicating with one another. Dural arteries were not observed inside of the sinuses, but branches of veins occasionally penetrated through the sinus wall and communicated with their lumens. Even though there is no arteriovenous anastomosis among the dural vessel, the dural arteries and veins were located close to one another. This intimate relationship between the meningeal branches and the venous sinuses offers anatomical opportunity for the development of fistulas [10, 11].

In histologic specimen of experimental DAVF models [9], the affected sinuses were remarkably

narrowed by thickening of the intima and organized thrombi. Dural veins were markedly dilated and had communication with one another and with the lumens of the venous sinuses. No direct communication was observed between dural arteries and venous sinuses. However, fistulas were seen between arteries with a diameter of about 200um and dilated dural veins near the venous sinuses.

Pathophysiology of Development of DAVFs

While the exact mechanisms are still controversial, venous sinus thrombosis and venous hypertension have been believed to be associated with the genesis of DAVFs [5, 12-14]. Two etiologic hypotheses regarding to sinus thrombosis and venous hypertension have been suggested. The first is that physiologic arteriovenous shunts that are normally present between meningeal arteries and dural venous sinuses expand to form a pathologic shunt in response to elevated local venous pressure as a result of sinus thrombosis [15, 16]. The second is that venous hypertension due to outflow obstruction causes decreased cerebral perfusion and promotes neoangiogenesis [14, 15, 17]. Angiogenic growth factors, produced as a result of sinus thrombosis, might contribute to the development of DAVFs [14]. Kultulk et al. [18] proposed a three stage hypothesis for the pathogenesis of DAVFs as initial stage of venous sinus thrombosis, second stage of angiogenic factor induced microscopic fistula development within the wall of the thrombosed sinus, and final stage of recanalization of the thrombosed sinus. Whether the cause or the result of hemodynamic disturbance, venous hypertension and thrombophilic condition such as venous thrombosis clearly plays a role in the development of DAVFs [7, 19]. Once triggered by venous hypertension and venous thrombosis, arterial feeder recruitment and development of DAVFs via neoangiogenesis might proceed. This process is associated with upregulation of hypoxia-inducible factor-1, vascular endothelial growth factor, stroma-cell derived factor alpha, and MMP-9 [19-21].

Classification

The relationship between the pattern of venous drainage and the natural history of DAVFs leads to proposal of classifications based on the venous drainage pattern of the shunts [10, 22]. Borden et al. proposed a classification system incorporating spinal DAVFs on the basis of the site of venous drainage and the presence of cortical venous drainage (CVD) [10]. Spinal epidural venous plexus corresponds to an intradural sinus and the spinal perimedullary veins are subarachnoid veins. Borden type I DAVFs exhibit normal antegrade flow into the dural venous sinus or meningeal vein. Type II DAVFs are those that drain into the dural sinus with retrograde flow into the subarachnoid veins. Type III DAVFs have retrograde drainage directly into the subarachnoid veins or an isolated sinus that is completely thrombosed on both sides of the arterialized segment.

Cognard et al. have classified DAVFs into five types based on the direction of dural sinus drainage, the presence of CVD, and venous outflow architecture. Cognard Type I DAVFs have an antegrade drainage into the dural sinus without CVD. Type II DAVFs are subdivided into type IIa (retrograde drainage into a dural sinus without CVD), IIb (antegrade drainage into a dural sinus with CVD), and IIa+b (retrograde drainage into a dural sinus with CVD). Type III, IV, and V DAVFs have retrograde drainage directly into subarachnoid vein, ectatic subarachnoid vein, and spinal perimedullary vein, respectively (Table 1).

Table 1. Classification of Dural Arteriovenous Fistulas

Borden		Cognard	
I	Drainage into sinus or meningeal vein	I	Drainage into a sinus, with antegrade flow
II	Drainage into sinus/meningeal vein with retrograde flow into subarachnoid vein	II	Drainage into a sinus, with insufficient antegrade sinus drainage and reflux

		IIa	Drainage into a sinus, sinus reflux only
		IIb	Drainage into a sinus, cortical vein reflux only
		IIa+IIb	Drainage into a sinus, sinus and cortical vein reflux
III	Drainage into subarachnoid vein only	III	Direct drainage into a cortical vein without venous ectasia
		IV	Direct drainage into a cortical vein with venous ectasia
		V	Drainage into spinal perimedullary veins

In either classification scheme, lack or presence of CVD can allow appropriate risk prediction for therapeutic decision-making. DAVFs without CVD (Borden type I, Cognard type I, IIa) have benign natural history with mild symptoms. Annual risk of hemorrhage has been reported low [23, 24]. The presence of CVD (Borden types II and III, Cognard types IIb-V) indicates an aggressive feature with an annual incidence of hemorrhage of 8% and non-hemorrhagic neurological deficit of 6~15% [25, 26]. Rebleeding rate in DAVFs with CVD reaches up to 35% [27]. Subdividing DAVFs with CVD according to presence of symptoms may have more predictive value in risk stratification [24]. Asymptomatic DAVFs patients with CVD have a considerably lower annual hemorrhage risk of 1.5%, while symptomatic patients with CVD have 7.4% of annual risk. However, DAVFs has been considered to have a dynamic nature. DAVFs without CVD can develop CVD in the future with ongoing venous stenosis, thrombosis, or increased arterial flow [28, 29]. Spontaneous thrombosis and resolution of DAVFs have also been reported [30].

Spinal Dural AVF

History and Classification of Spinal Arteriovenous Lesions

Since Foix and Alajouanine first described of spinal DAVFs (1926), the evolution of diagnostic tools has led to examine the angioarchitecture of these spinal vascular lesions better. However, there is still a lack of consensus in the classification and terminology of the spinal arteriovenous lesions [12]. Spinal vascular lesions have been described by numerous names such as spinal AV shunt (Di chiro, 1967), extramedullary and intramedullary vascular malformation (Yasargil, 1975), a spinal epidural angiomatous malformation draining into intrathecal veins (Kendall, 1977), and spinal arteriovenous malformation (AVM) and divided into various classifications according to clinical feature, anatomical location and angioarchitecture [31]. In general, spinal vascular lesions are divided into two categories; arteriovenous shunting lesions including spinal arteriovenous malformation (AVM) and arteriovenous fistulas (AVF), and non-shunting lesions including capillary telangiectasia and cavernous malformations. In this chapter, spinal arteriovenous shunting lesions will be further discussed.

In 1971, Di chiro et al. described the first classification of spinal AV shunts as type I (single coiled vessel), type II (glomus), and type III (juvenile). The majority of type I fistula fit current concept of spinal DAVFs and the others correspond to intramedullary AVMs. In 1986, a direct spinal AVF (perimedullary fistula in current terminology) was added as type IV (a direct AV shunt between the anterior spinal artery and vein that was located ventral to the spinal cord). Rosenblum (1987) established most widely accepted classification: type I (dural AVF), type II (intradural intramedullary glomus AVM), type III (intradural intramedullary juvenile AVM), and type IV (intradural direct AVF) [32]. Since 1993, type IV intradural direct AVFs were described as perimedullary AVFs and perimedullary AVFs were further classified according to the number of feeding arteries and size of AVF; type I with a single feeder and single small AVF, type II with multiple feeders and multiple medium AVFs, and type III with multiple feeder and a single giant AVF

[33]. Now, extradural AVFs were added in this system and subdivided into three types according to the presence of intradural venous drainage and neurological deficits [34]. Recently, Takai [35] proposed a revised version of previous classifications (Table 2. Fig. 1). In 2008, Geibprasert and Lasjaunias reported a classification including intracranial and spinal AVFs in which AV shunts were divided into 3 groups based on the embryologic development of the venous drainage; the ventral, lateral, and dorsal epidural groups [36]. Spinal DAVFs are the lateral epidural AVFs.

Table 2. Revised classification of spinal AV shunt by Takai (2017)

Type	Academic name	Subtype	Feeder, AVF, and venous drainage
I	Dural AVF		
II	Intramedullary glomus AVM		
III	Intramedullary juvenile AVM		
IV	Perimedullary AVF	IVa	A single feeder and small AVF
		IVb	Multiple feeders and medium AVF
		IVc	Multiple feeders and a giant AVF
V	Extradural AVF	Va	With intradural venous drainage
		Vb	Without intradural venous drainage

Incidence and Clinical Characteristics

Spinal DAVFs are defined by pathologic shunt between a radicular artery and coronal venous plexus of the spine without an intervening capillary bed. They are the most common type of spinal cord vascular malformation, accounting for 50% to 85% of all spinal vascular lesions [37]. Spinal DAVFs are reported to affect 0.5~1/100,000 per year [38, 39] and precise incidence of the rest of spinal vascular lesions is lacking. Spinal DAVFs are thought to be acquired lesions and preferentially affect middle-aged men. At least 80% of patients in most series are male [39]. The most frequent location is in the lower thoracic and upper lumbar area [40] and patients most commonly present with gait abnormality or lower-extremity weakness and sensory disturbances. Spinal AVM including intramedullary AVMs and perimedullary fistulas are believed to be congenital and become symptomatic in childhood or early adulthood by steal or hematoma, whereas spinal DAVF typically present in the fifth and sixth decades [32]. Perimedullary fistula is a single shunt without a nidus between the spinal artery and the spinal vein and constitutes 20% of all spinal vascular lesions. In contrast with DAVF, these lesions are located ventrally or dorsally on the spinal cord and are located intradurally but extramedullarily. Spinal subarachnoid hemorrhage is one of the occurring signs because of its intradural location, while spinal DAVF seldom presents subarachnoid hemorrhage. Intramedullary AVM presents compression effects from dilated vessels, steal phenomena, and catastrophic intramedullary hematoma in young aged patients.

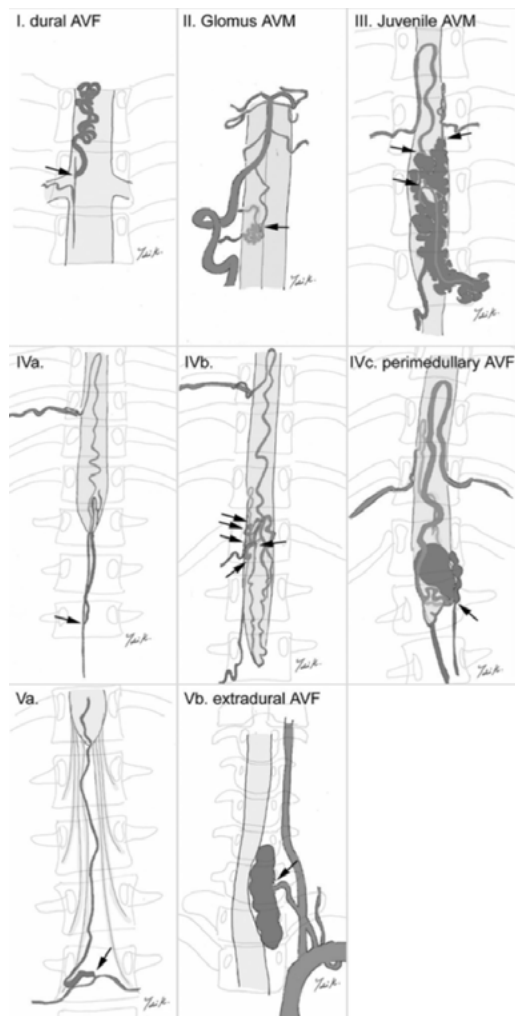
Pathophysiology

The pathophysiology of development of lesions and neurological deterioration is different in patients with DAVF and those with intradural spinal AVMs. Spinal DAVFs are probably acquired lesions while medullary AVM's are congenital in nature.

Although complex pathophysiologic mechanisms underlying the development of spinal DAVFs are yet to be clarified, meningeal venous thrombosis and trauma are implicated in the development of spinal DAVFs [38]. Conditions associated with vascular fragility such as fibromuscular dysplasia [41] and neurofibromatosis type 1 [42] are associated with spinal DAVFs. Histopathological examination supported the theory of venous hypertension as a pathophysiological mechanism in the development of DAVFs and increased venous congestion as a cause of neurological deterioration [43, 44].

Segmental arteries from each level of the spinal column gives rise to spinal radicular arteries supplying the dura (radiculomeningeal arteries) and accompanies the corresponding nerve root in its dural sleeve. These arteries may continue as a radiculomedullary artery, which accompanies the dorsal and ventral rami of the nerve root into the thecal sac to supply the anterior and posterior spinal arteries. An extramedullary, circumferential network of vein known as the coronal venous plexus receives spinal cord venous outflow and drains into the epidural vein via the radicular/radiculomedullary veins [12, 37]. In spinal DAVFs, the radiculomeningeal artery within the dural sleeve of the nerve root fistulizes with the radicular vein and this leads to arterialize the valveless perimedullary venous plexus. An increased venous pressure, venous congestion, edema, and progressive ascending myelopathy are caused by obstruction of venous outflow [39]. An increased venous pressure results in a reduced arteriovenous pressure gradient, intramedullary impairment of autoregulation, and hypoxia [37, 44]. In patients with intradural AVMs, neurological presentations are probably caused by high-pressure, high-volume, turbulent blood flow through the intradural nidus and its feeding and draining channels. Increased venous pressure as well as arterial steal attributes spinal cord ischemia due to reduction of tissue perfusion pressure [45]. Spinal epidural (extradural) AVFs are very rare, but may cause a compressive myelopathy due to a presence of a venous pouch in the epidural venous plexus. It may or may not develop venous congestion due to retrograde intradural venous drainage [46].

Fig. 1. Schematic drawing of spinal AV shunt



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Pathological vascular anatomy for dural AVF

조수희

울산대 강릉아산병원 신경외과

Pathological vascular anatomy for dural AVF

울산대학교 강릉아산병원 조수희

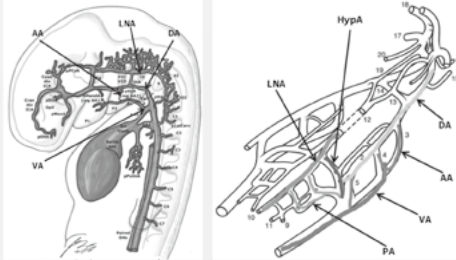
- Clinical anatomy for safe and effective endovascular treatment of dural AVF
 - Dangerous EC-IC anastomoses
 - Arterial supply of the cranial nerve

Dangerous EC-IC anastomoses

clinical significance

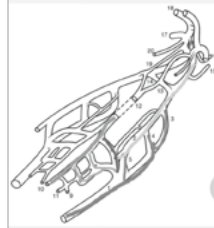
- Cause of major complication
 - Migration of embolic materials into anastomotic channels can result in embolic strokes or cranial nerve palsies.
 - Most of these anastomotic channels follow the CN along the neural foramen.
- "Potential" anastomoses
 - Although they may not be visualized on routine catheter angiographies, they are always present.
 - They will necessarily open under following circumstances
 - 1) increased intra-arterial pressure
(during embolization procedures or superselective injection)
 - 2) in the presence of high-flow shunts
 - 3) major intracranial artery occlusion

Embryology



- ventral aorta(VA) -> regression -> proximal remnant -> ECA trunk
- dorsal aorta(DA) -> ICA trunk
- longitudinal neural artery(LNA) -> VB system
- Primitive artery that link the VA and DA
- aortic arch(AA) -> regression -> dorsal remnant -> ICA br, ECA br
- Primitive artery that link the prox IIIAA or VA
- hypoglossal artery(HypA) -> regression -> ascending pharyngeal a(aphA)
- proatlantal artery(PA) -> regression -> occipital artery(OA), C1,2 segmental artery

Ref 1),2)



AA
 VA - DA
 Embryonic artery
 (ECA and ICA branch)
 ECA - ICA

HypA

LNA - VA , prox IIIAA

AphA

Vertebral artery - ECA , pICA

PA

LNA - VA , prox IIIAA

OA

Vertebral artery - ECA , pICA

EC-IC anastomoses

• Orbital region

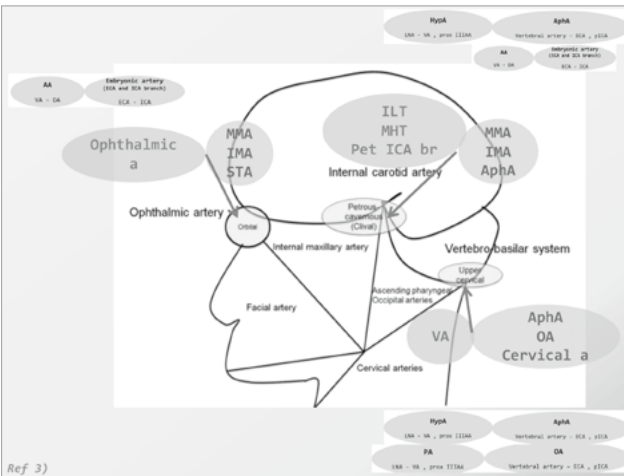
- 1)proximal internal maxillary collaterals
- 2)distal internal maxillary collaterals
- 3)cutaneous collaterals

• Cavernous-petrous region

- 1)ascending pharyngeal collaterals
- 2)proximal internal maxillary collaterals
- 3)distal internal maxillary collaterals

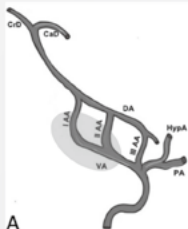
• Upper cervical region

- 1)occipital collaterals
- 2)ascending pharyngeal collaterals
- 3)cervical collaterals



Ref 3)

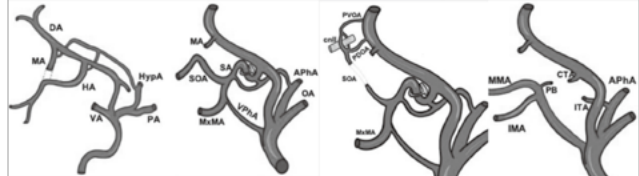
Embryology



- Primitive aortic arch(AA) that linked ventral aorta(VA) and dorsal aorta(DA).
- VA regresses together with the ventral part of AA.
- IIIAA,DA -> ICA trunk
- Dorsal remnants of AA persist as embryonic arteries.
- -IIAA dorsal remnant -> hyoid artery(HA) -> SA -> MMA/IMA(collaterals with opha)
- -IAA dorsal remnant -> mandibular artery(MA)
- VA prox remnant -> (ventral pharyngeal a.) -> ECA trunk

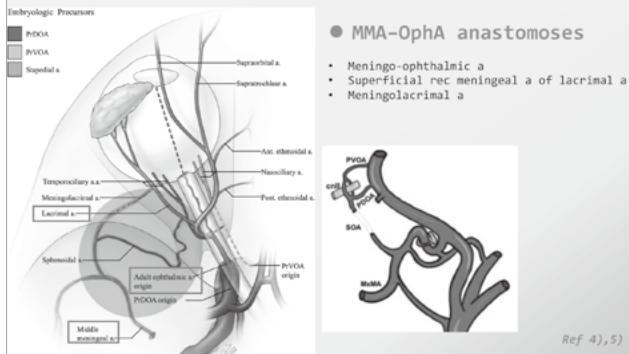
Ref 4)

Development of stapedial artery

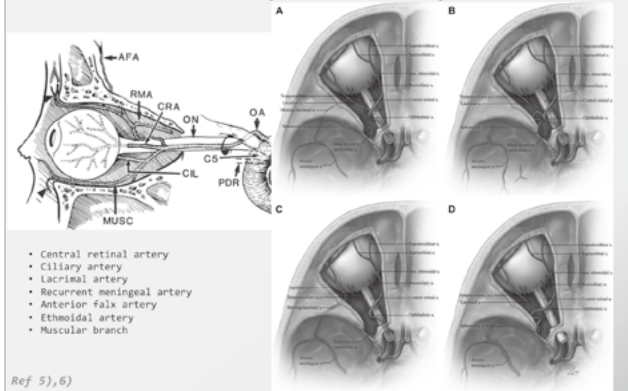


- Elongation of HA
- Annexation of PA territory by the HA
- Cranial growing of SA (from HA) passing into the middle ear(crus of the stapes)
- Extension of the two branches of the SA: supraorbital(SOA) and maxillomandibular(MxMA) branches
- SOA -> orbital br(supraorbital,lacrima,ethmoid,frontal a) and MMA
- Extracranially, Vpha annexes MxMA of SA, forming the proximal stem of the MMA and becoming the ECA
- Annexation of the SOA by the ophthalmic artery(POOA,PVOA)
- Regression of the trans-ossous(sup orbital fissure) segment of the SOA
- ->leaving anastomotic artery between MMA and lacrimal a(sphenoid a) which penetrate the orbit through sup orbital fissure
- Regression of the intratympanic segment of the SA
- Remnant of SA : caroticotympanic artery and sup tympanic a

MMA-OphA anastomoses

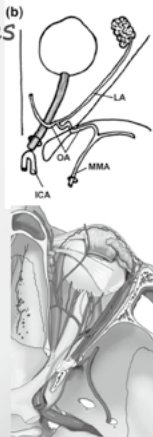


Anatomy of the OphA

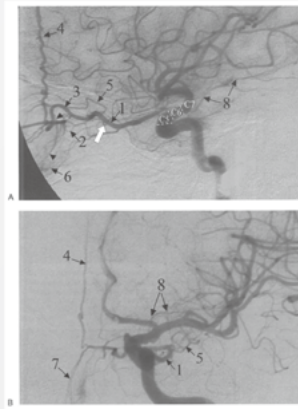


MMA-OphA anastomoses

- Meningo-ophthalmic artery**
 - Extreme variation of remnant of the embryologic stapedial artery
 - MMA takes over the entire orbital supply (including central retinal a, ciliary a.)
- Superficial recurrent meningeal branch of the lacrimal artery**
 - Anastomoses with orbital br through the SOF and ant falx artery
- Meningeal branch arising from distal Oph**
 - supply the anterior falx
 - Anastomosing indirectly with the ant branch of the MMA through the meninges
- These are always present except for meningo-ophthalmic a. They are usually not visualized on global injection.

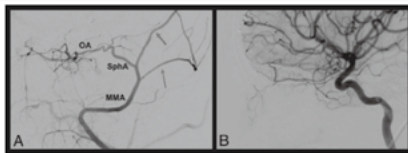


Ref 7)

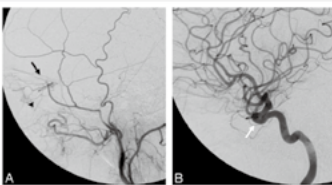


- Ophthalmic a
 - ocular OA br
 - Orbital OA br
 - Ant falx a
 - Lacrimal a
 - Muscular br
 - Ant ethmoidal a
 - Acho a
- Arrow heads : choroid blush

Ref 6)

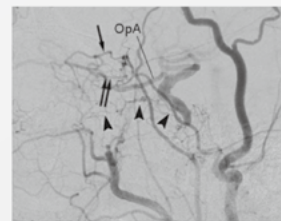


- Meningo-ophthalmic artery**
 - Selective injection of the MMA(A) and ICA(B)
 - Sphenoidal artery arises from the MMA
 - It courses along the inner surface of the temporal and sphenoidal bone and enters the orbit through the superior orbital fissure.

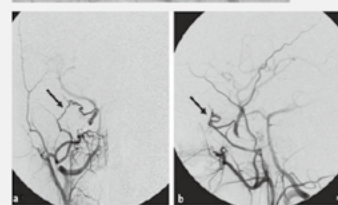


- Meningo-ophthalmic artery**
 - Left ECA and ICA angiography
 - meningo-ophthalmic a. (black arrow)
 - choroidal blush (arrowhead)
 - contributing supply to the entire orbit with the absence of the opha from the ICA (white arrow)
 - In this setting, even proximal occlusion of the MMA carries a high risk because the MMA is the sole supply to the distal opha and must be avoided.

Ref 4),3)



- Meningolacrimal artery (single arrow)**
- Recurrent meningeal artery (double arrows)**
- Left CCA angiography in case of ipsilateral ICA occlusion
- OphA supplied by MLA and RMA



- Meningolacrimal artery**
 - Rt ECA angiography

Ref 8),9)

• **Orbital region**

- 1) proximal internal maxillary collaterals
- 2) distal internal maxillary collaterals
- 3) cutaneous collaterals

• **Cavernous-petrous region**

- 1) ascending pharyngeal collaterals
- 2) proximal internal maxillary collaterals
- 3) distal internal maxillary collaterals

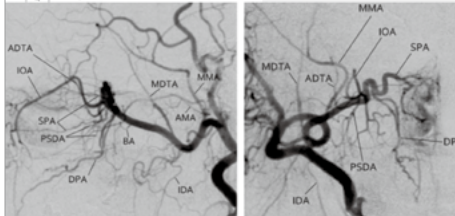
• **Upper cervical region**

- 1) occipital collaterals
- 2) ascending pharyngeal collaterals
- 3) cervical collaterals

Branches of IMA



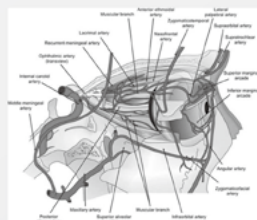
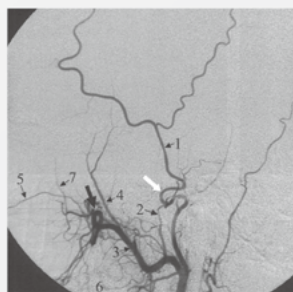
ADTA: ant deep temporal a
 BA: buccal a
 DPA: descending palatine a
 IOA: infraorbital a
 MDTA: mid deep temporal a
 PSDA: posterior superior dental a
 SPA: sphenopalatine a



Ref 8)

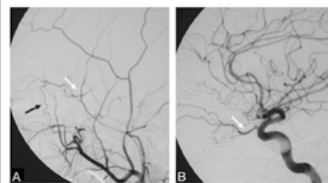
**Collaterals between
 Distal IMA br - OphA**

- Ant deep temporal a, infraorbital a. - Inferior br of lacrimal artery



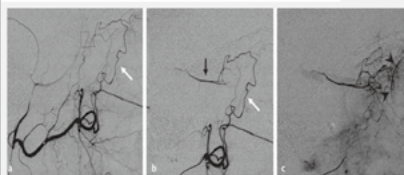
1. Superficial temporal artery
2. Middle meningeal artery
3. Maxillary artery
4. Middle deep temporal artery
5. Infraorbital artery
6. Buccal arteries
7. Anterior deep temporal artery

Ref 6)



• **ADTA-lacrimal a**

- Left ECA and ICA angiography
- Pt with balloon embolization of a traumatic ccf
- Anastomosis between the ADTA (black arrow) through the LA to OphA (white arrow)



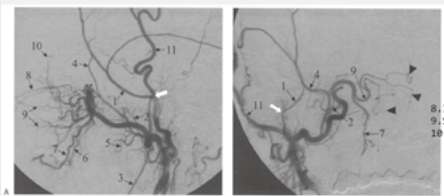
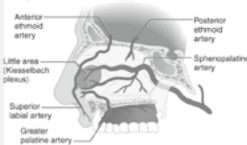
• **ADTP-lacrimal a**

- ECA angiography
- Occlusion of ipsilateral ICA
- anastomosis between ADTA (white arrow) through lacrimal a to ophA (black arrow)
- Choroidal blush (arrowhead)

Ref 3), 9)

**Collaterals between
 Distal IMA br - OphA**

- Sphenopalatine a from IMA
 - ant/post ethmoidal a. of OphA
 (through septal arteries)



8. Infraorbital a
9. Sphenopalatine a with nasal br
10. ADTA

Ref 6)

• **Orbital region**

- 1) proximal internal maxillary collaterals
- 2) distal internal maxillary collaterals
- 3) cutaneous collaterals

• **Cavernous-petrous region**

- 1) ascending pharyngeal collaterals
- 2) proximal internal maxillary collaterals
- 3) distal internal maxillary collaterals

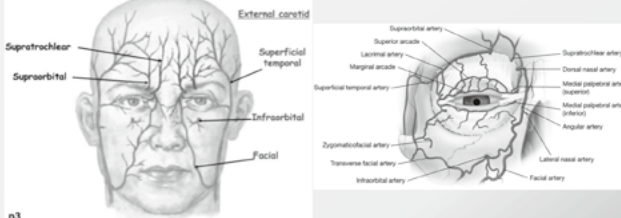
• **Upper cervical region**

- 1) occipital collaterals
- 2) ascending pharyngeal collaterals
- 3) cervical collaterals

Collaterals between STA, FA - ophA

- Both facial artery and superficial temporal artery branches thereby classically connect to the distal(third) portion of ophA.
- Frontal br of STA - ophA (through supraorbital a.)
- Distal end of FA - dorsal nasal artery

Major arteries of the face



p3

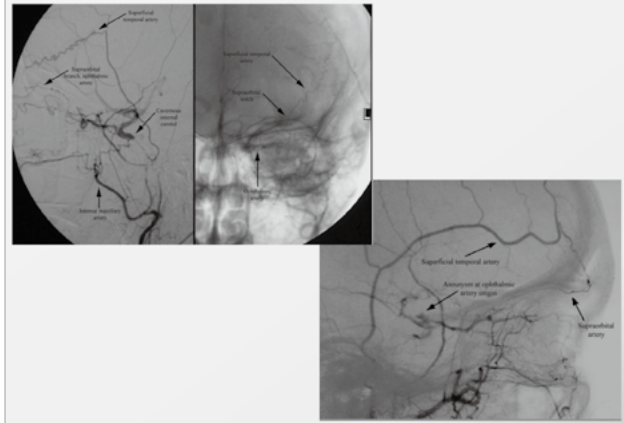


Table 2: Summary of the branches of the ophthalmic artery and their anastomoses to the ECA

Ophthalmic Artery Branch	Origin of the Ophthalmic Branch	ECA Branch
Proximal lacrimal artery (main branch)	Second portion of OA	MMA (through superior orbital fissure)
Distal lacrimal artery (inferior branch)	Second portion of OA	Anterior deep temporal artery and infraorbital artery (IMA)
Anterior ethmoidal arteries	Third portion of OA	Septal arteries: sphenoparietal artery (IMA), MMA, Sphenoparietal artery, greater palatine artery (IMA), MMA, STA
Posterior ethmoidal arteries	Second or third portion of OA	STA
Supraorbital artery	Third portion of OA	Angular termination of FA, infraorbital artery
Dorsal nasal artery	Terminal branch of OA	

Table 1: Main anastomoses between the ECA and the OA

Name	OA branch	ECA branch	Anat. F.	Ang. F.	VI
Meningo-ophthalmic a.	Lacrimal a.	MMA	47.14% [12]	37.7% [10]	0.80
	OA itself	MMA	ND	31.1% [10]	—
A. of the superior orbital fissure	Lateral muscular a.	MMA	5.71% [12]	ND	—
	Deep recurrent OA	Maxillary a.	ND	3% [32]	—
	Lacrimal a.	Anterior deep temporal a.	ND	33.3% [10]	—
	Lacrimal a.	Transverse facial a.	ND	ND	—
	Lacrimal a.	Orbital branch of the infraorbital a.	ND	ND	—
	Lacrimal a.	Zygomatico-orbital a.	ND	2.22% [10]	—
	Dorsal nasal a.	Facial a.	60% [7]	8.9% [10]	0.15
	Dorsal nasal a.	Orbital branch of the infraorbital a.	27% [7]	6.6% [10]	0.24
	Supraorbital a.	Superficial temporal a.	33% [7]	2.22% [10]	0.07
	Supraorbital a.	Zygomatico-orbital a.	ND	2.22% [10]	—
	Supratrochlear a.	Superficial temporal a.	ND	2.22% [10]	—
	Anterior ethmoidal a.	Sphenoparietal a.	ND	ND	—
	Posterior ethmoidal a.	Sphenoparietal a.	ND	ND	—

Anat. F. anatomic frequency, Ang. F. angiographic frequency, ND not determined

Ref. 33, 7)

Table 1: Different dural branches of the OA with their respective supply and anastomoses

OA Branches	Origin from the OA	Foramen	Dural Territory	Possible Anastomosis	Clinical Consequences in Case of Embolism
Deep recurrent OA	First segment	Superior orbital fissure	Superior orbital fissure (lateral part), sphenoid wing	Inferolateral trunk (ICA)	Cerebrovascular accident
Superficial recurrent OA	Second segment	Superior orbital fissure	Anterior clinoid process, Lesser sphenoid wing, Middle fossa (anteromedial portion)	Posterior ethmoidal artery, MMA (anterior division), Medial tentorial artery (ICA)	Cerebrovascular accident, loss of vision
Anterior ethmoidal artery	Third segment	Anterior ethmoidal canal	Anterior convexity (anterior meningeal artery), Anterior cranial fossa (medial third), Anterior falx cerebri (anterior falcine artery)	Contralateral anterior ethmoidal artery, Bilateral MMAs, Posterior ethmoidal artery, Olfactory branch (ACA)	
Posterior ethmoidal artery	Third segment	Posterior ethmoidal canal	Anterior cranial fossa (medial third), Anterior clinoid process, Chiasmatic groove	Contralateral posterior ethmoidal artery, Anterior ethmoidal artery, MMA (anterior division)	

◆ OphA anastomoses with branches from ECA and ICA.

Ref. 10)

Orbital region

- 1) Proximal internal maxillary collaterals
- 2) distal internal maxillary collaterals
- 3) cutaneous collaterals

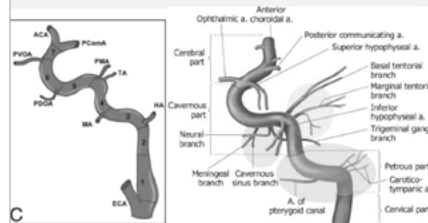
Cavernous-petrous region

- 1) ascending pharyngeal collaterals
- 2) proximal internal maxillary collaterals
- 3) distal internal maxillary collaterals

Upper cervical region

- 1) occipital collaterals
- 2) ascending pharyngeal collaterals
- 3) cervical collaterals

Anastomoses in Cav-Pet region



- Embryonic arteries:
- Cav: PDOA -> ILT
- Cliv: PMA -> PHT
- TA -> lat clival a.
- Pet: PA -> PA
- HA -> CTA

Petrous

Mandibular a,
carotid-tympanic a
~
AphA
IMA br

Clival

Meningohypophyseal
trunk
Lateral clival a
~
AphA
IMA br

Cavernous

Inferolateral
Trunk
~
PMA
IMA
AphA

• Orbital region

- 1) Proximal internal maxillary collaterals
- 2) distal internal maxillary collaterals
- 3) cutaneous collaterals

• Cavernous-petrous region

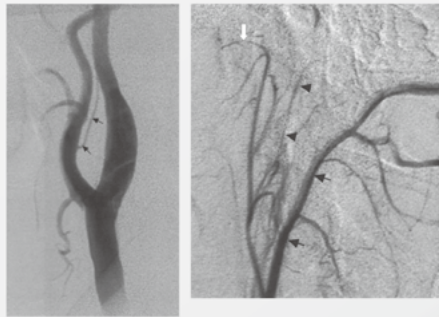
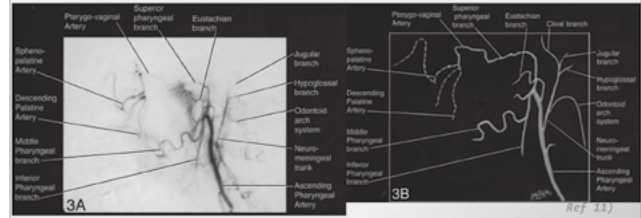
- 1) ascending pharyngeal collaterals
- 2) proximal internal maxillary collaterals
- 3) distal internal maxillary collaterals

• Upper cervical region

- 1) occipital collaterals
- 2) ascending pharyngeal collaterals
- 3) cervical collaterals

Ascending pharyngeal artery

- After a short common trunk, apha divides into two major trunks.
 - ant : pharyngeal trunk(extracranial br)
 - Post : neuromeningeal trunk(intracranial br) enters the posterior fossa through the foramen magnum
- Inf tympanic br
- Musculospinal artery



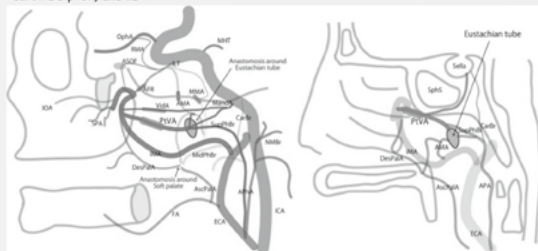
Ref 6)

Collaterals between Pharyngeal trunk of Apha - pet/cav ICA br

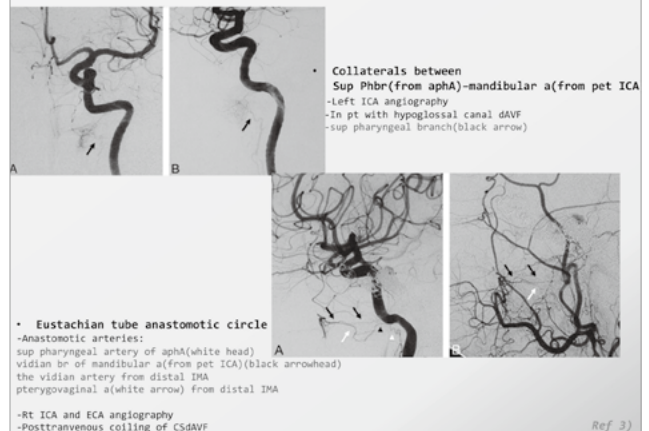
- Pharyngeal trunk
 - Anteriorly located branches from apha common trunk
 - 3 branches : inf/mid/sup pharynx a.
 - These supply the pharyngeal submucosal spaces
 - Abundant arterial anastomoses exist with the sphenopalatine system.
- Sup br has most important anastomotic routes.
 - collaterals with AMA, pterygogingival a. from IMA
 - collaterals with rec a of foramen lacerum(from lat clival a.), br of ILT

*eustachian tube anastomotic circle

- Sup phbr
 - connects with mandibular artery from petrous ICA
 - anastomosis with AMA and pterygogingival artery from distal IMA
- Carotid canal branch from Sup phbr
 - enters to carotid canal through foramen lacerum
 - join with recurrent artery of foramen lacerum(cbr of lat clival a, arises from C5 of carotid siphon) and ILT



Ref 12)



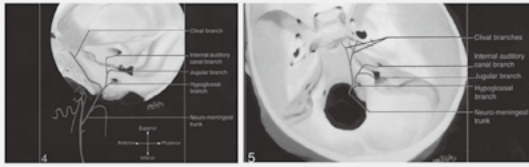
• Eustachian tube anastomotic circle

- Anastomotic arteries:
 - sup pharyngeal artery of apha(white head)
 - vidian br of mandibular a(from pet ICA)(black arrowhead)
 - the vidian artery from distal IMA
 - pterygogingival a(white arrow) from distal IMA

- Rt ICA and ECA angiography
- Posttransvenous coiling of C5AVF

Ref 3)

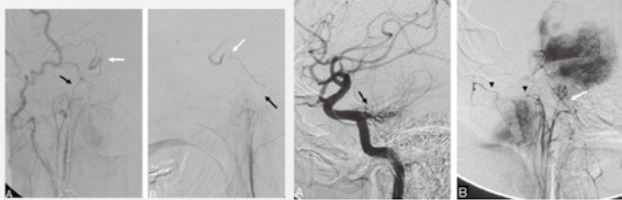
Collaterals between NM trunk of aphA - LCA, MHT



- 2 branches : jugular br and Hypoglossal br
- Rarely, NM trunk may arise from occipital artery or post auricular artery
- Both branches give off medial and lat clival br immediately after exiting the hypoglossal and jugular foramen
- These branches anastomoses with clival branches from lateral clival artery (from cav ICA) and branches from meningohypophyseal trunk.

Ref 11)

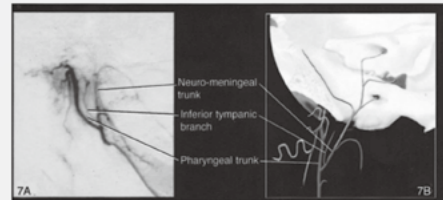
- Hypoglossal br
: extends to the posterior fossa to the hypoglossal canal
: supplies the meninges of post fossa and vasa nervorum of CN XII
- Jugular br
: extends to the post fossa to the jugular foramen
: supplies vasa nervorum of CN IX,X,XI
: leaves the jugular foramen to supply
~ (superiorly) meninges of the int auditory canal
(medially) dura of inf petrosal sinus and vasa nervorum to CN VI proximal to Dorello's canal
(laterally) dura of sigmoid sinus



- Collaterals between NM trunk - Lat clival a of cav ICA
- Rt aphA angiography
- NM trunk (black arrow), LCA (white arrow)
- Collaterals between NM trunk - MHT
- Rt ICA and aphA angiography
- In patient with clival MCA
- MHT (black arrow), NM trunk from aphA (white arrow)
- Retrograde filling of PVA (arrowhead) of dINA from sup pha through eustachian tube

Ref 3)

- Inf tympanic artery
- Arise from main trunk of asc pha or one of its major br
- Most commonly arises from the prox aspect of NM trunk
- Enters the tympanic cavity through inf tympanic foramen with Jacobson's nerve
- It is close to CN IX and provide rami to the caroticotympanic branch of ICA, vasa nervorum of CN XI, vasa vasorum of ICA
- Retains the embryologic connection of the artery of the third brachial arch (Asc pha) and the hyoid artery (or caroticotympanic a) from the petrous ICA
- Inside middle ear, anastomoses with sup tympanic a (arise from petrous br of MMA), ant tympanic a (from prox IMA), stylomastoid a (from post auricular-OA), mandibular br (of ICA)



Ref 11)

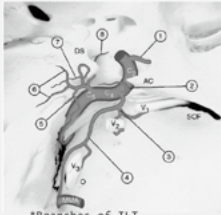
Collaterals of aphA



Ref 11)

- Orbital region
1) Proximal internal maxillary collaterals
2) distal internal maxillary collaterals
3) cutaneous collaterals
- Cavernous-petrous region
1) ascending pharyngeal collaterals
2) proximal internal maxillary collaterals
3) distal internal maxillary collaterals
- Upper cervical region
1) occipital collaterals
2) ascending pharyngeal collaterals
3) cervical collaterals

ILT/MHT branches



*Branches of IMA

2. sup br : supplying the roof of cav sinus - collateral with cav br of MMA
3. ant br : a. to SOF - collateral with orbital br of MMA, rec meningeal br of OphA
a. to fo rotundum - collateral with fo rotundum br of IMA
4. post br : a. to fo ovale - collateral with AMA, carotid br of apha
a. to fo spinosum

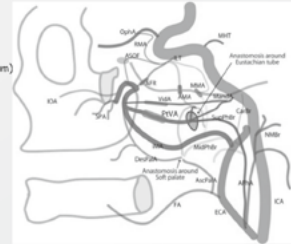
*Branches of MHT

5. recurrent artery of fo lacerum
6. mid and lat dorsal clival arteries : collateral with jugular br of apha
7. tentorial marginal artery : collateral with petrosquamosal br of MMA
8. inf hypophyseal artery : hypophyseal br
: inf clival br - collateral with asc clival br of aph

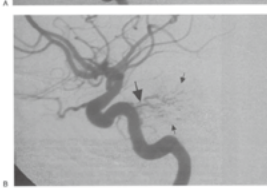
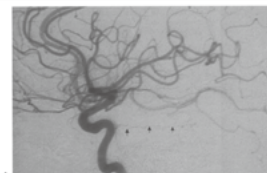
Ref 13)

Collaterals between IMA branches - IMA, MHT

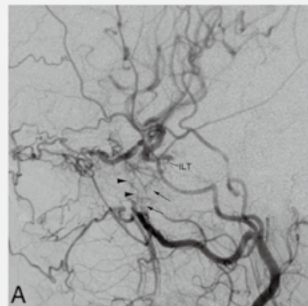
- Prox IMA
 - MMA :
 - Cav br - sup br of IMA
 - Orbital br - antmed br of IMA (SOF)
 - Petrosquamosal or post br - marginal a of tentorial a from IMA or MHT
 - AMA - Posteromedial br of IMA (fo ovale)
- Distal IMA
 - artery of foramen rotundum
 - inferolateral br of IMA or lat clival a.
 - Vidian artery
 - vidian br of mandibular a. from pet ICA (foramen lacerum)
 - Pterygogingival artery
 - ending at anastomotic circle around the eustachian tube



Ref 12)

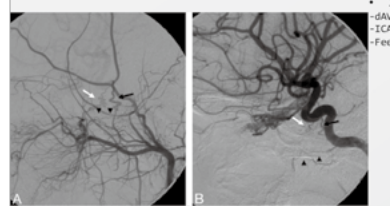
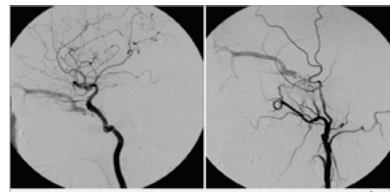


- Meningohypophyseal trunk
 - a. Normal patient
 - b. Clival MGH



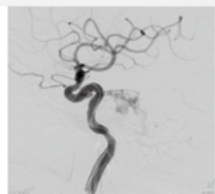
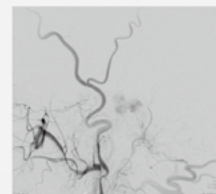
- Anastomoses between IMA and IMA, OphA
 - Lt CCA angiography
 - Case of left ICA occlusion
 - a of fo rotundum (arrow), a of SOF from IMA (arrowhead)

Ref 6), 15)



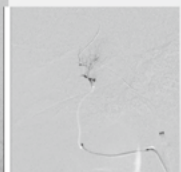
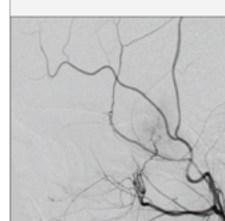
- Inferolateral trunk and collaterals
 - dAVF of oph V
 - ICA and ECA angiography
 - feeder : IMA, AMA, MMA, dIMA (a of fo rotundum)
- Anastomosis between
 - A of fo ovale (from AMA) and a of fo rotundum (from dIMA) to IMA
 - Vidian a (from IMA) and vidian branch of mandibular a
 - Left ECA and ICA angiography
 - Patient with cerebrofacial arteriovenous metamerism, AVM of optic nerve
 - a of fo ovale (black arrow), a of fo rotundum (white arrow), Vidian a (arrow head) from IMA

Ref 3), 14)



- Anastomoses between IMA and IMA
 - ICA and ECA angiography
 - Case of dAVF in cbll v
 - feeders: MHT (marginal tentorial a), pet br of MMA, AMA, a of foR, post auricular a, OA
 - Balloon occlusion of ICA :
reflux into deep rec oph a (double arrow), a of foR (white arrow), AMA (black arrow)

Ref 16)



- R16
 - Anastomoses between IMA and IMA
 - Lt ECA and ICA angiography
 - Tumor staining by a of foR and IMA
 - ICA balloon occlusion at IMA os - anastomoses by a of foR

Ref 16)

• Orbital region

- 1)proximal internal maxillary collaterals
- 2)distal internal maxillary collaterals
- 3)cutaneous collaterals

• Cavernous-petrous region

- 1)ascending pharyngeal collaterals
- 2)proximal internal maxillary collaterals
- 3)distal internal maxillary collaterals

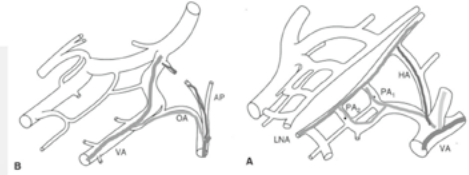
• Upper cervical region

- 1)occipital collaterals
- 2)ascending pharyngeal collaterals
- 3)cervical collaterals

Embryology



- dorsal aorta -> ICA
- ventral aorta -> ECA
- longitudinal neural artery -> vertebral a
- Aortic arch -> ECA, ICA br
- Hypoglossal artery -> apha
- Proatlantal artery -> occipital a



Ref 2)

Persistent primitive artery carotid-vertebrovascular anastomoses

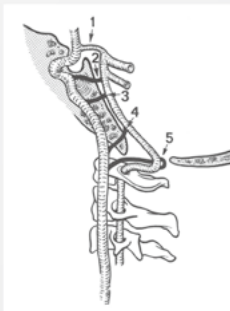


FIG. 3-14. Anatomic diagram depicts the embryonic carotid-basilar and carotid-vertebral anastomoses. The posterior communicating artery is the only vessel that normally persists; the other four, shown in black, usually regress completely.

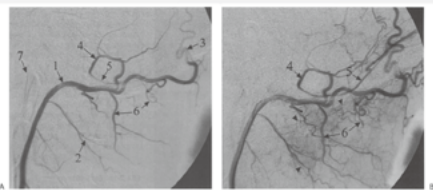
1. Posterior communicating artery
2. Trigeminal artery
3. Cili artery
4. Hypoglossal artery
5. Proatlantal intersegmental artery

Ref 6)

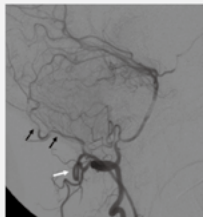
1)Occipital collaterals

- OA is a remnant of the embryologic type I and II proatlantal arteries(=correspond to C1 and C2 segmental arteries)
- In the fetal period, both embryologic arteries serve as a connection routes between the carotid and VB systems.
- Full persistence of these segmental a. is extremely rare.
- posterior anastomotic radicular branches(arise from the horizontal portion of the OA at both C1 and C2 level)
 - These are the major collaterals from the VA to the carotid system
 - Often seen in case of CCA ligation or occlusion

- Stylomastoid artery
 - Arise from either the post auricular or occipital artery
 - Supply the meninges of the posterior fossa and forms anastomoses with other meningeal branches from MMA, apha, ILT, MHT, post meningeal artery of VA

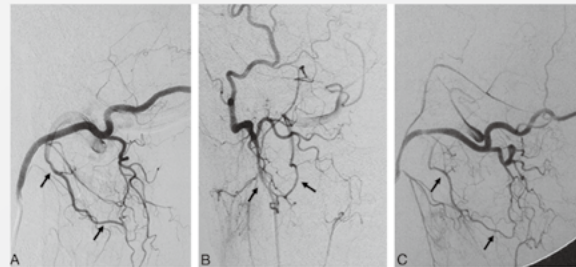


- 1.OA
- 2.Muscular br
- 3.Scalp br
- 4.Transosseous br
- 5.Posterior meningeal a
- 6.Muscular br
- 7.Stylomastoid br



- Anastomoses between OA and VA
- VA angiography
- OA(black arrow), post radicular br(white arrow) at C1

Ref 6),3)



- Anastomoses between OA and VA
- OA angiography
- post radicular br(arrow) at C2

Ref 3)

2) Ascending pharyngeal collaterals

- Remnant of the embryologic hypoglossal artery
- Keeps connection with the VA through prox br.

• Musculosplinal br

:Lowest, usually most prox br of apha

:anastomoses laterally with C3 radicular anastomotic artery of VA

- Anastomoses with VA at level of third intervertebral space(C2-3)

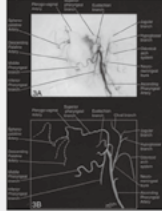
- Anastomoses with most superior br of asc cervical artery at C3 and with deep cervical a
- Supply the vasa nervorum to CN XI and sup sympathetic ganglion

• Prevertebral br

:located on the ventral surface of the C1-2 vertebrae

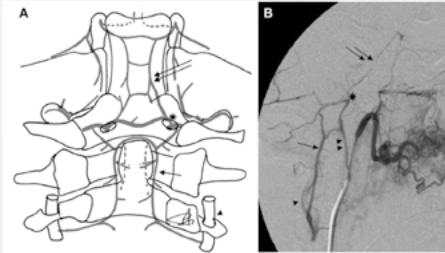
:typically arises from the neuromeningeal trunk, but may also arise directly from the main apha anastomoses with the odontoid arch

:characteristic U shaped curve before anastomosing medially on the surface of the dens with the C3 collaterals from VA



*odontoid arch system

arterial arcade surrounds the dens and has transverse br that anastomoses with ant/post asc arteries of dens)



• Odontoid arch system

epidural clival network(double arrow)

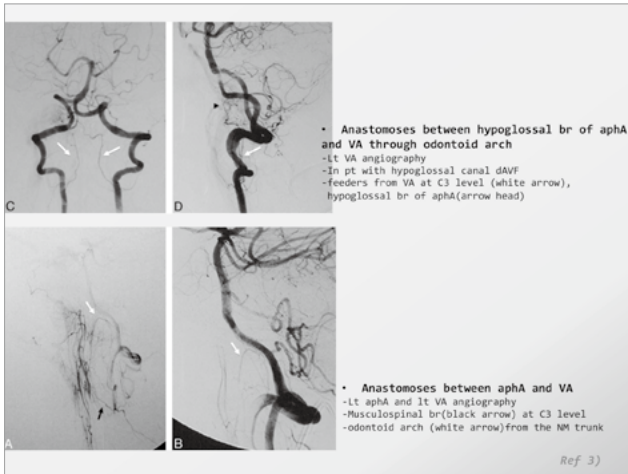
hypoglossal branch(asterisk) of apha

odontoid arch(arrow)

NM trunk(double arrowhead)

VA(arrowhead)

Ref 17)



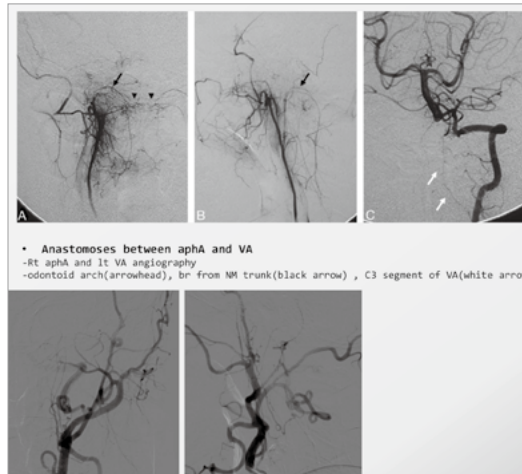
• Anastomoses between hypoglossal br of apha and VA through odontoid arch

- In pt with hypoglossal canal dAVF
- feeders from VA at C3 level (white arrow), hypoglossal br of apha(arrow head)

• Anastomoses between apha and VA

- lt apha and lt VA angiography
- Musculosplinal br(black arrow) at C3 level
- odontoid arch (white arrow)from the NM trunk

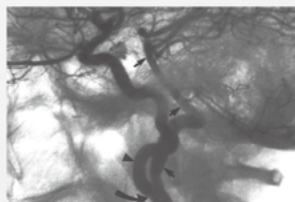
Ref 3)



• Anastomoses between apha and VA

- Rt apha and lt VA angiography
- odontoid arch(arrowhead), br from NM trunk(black arrow), C3 segment of VA(white arrow)

Ref 3)



Typical persistent hypoglossal artery(arrow)

- Left CCA angiography
- Cervical ICA(arrowhead)



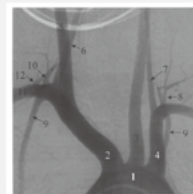
Persistent hypoglossal artery variant

- Rt ECA angiography
- hypoglossal br of NMT(black arrow), PICA(arrow head)

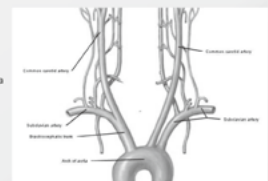
Ref 6), 18)

3) Cervical collaterals

- Asc cervical a, deep cervical a are major anastomotic channels of VA at C2-4 levels.
- Both arise from the subclavian a.
- AscCA typically originates from thyrocervical trunk
- DeepCA from costocervical trunk



- 5 rt CCA
- 6 rt VA
- 7 lt VA
- 8 lt thyrocervical
- 9 rt and let int thoraci a
- 10 rt thyrocervical
- 12 rt costocervical



Ref 6)



Summary of the major EC-IC anastomoses

Table 1: Summary of the major extra- and intracranial anastomoses

Major artery	Extracranial		Intracranial	
	Location	Branch	Branch	Artery
Internal maxillary artery	Proximal	MMA	Orbital branches, anterior branch (anterior labial artery)	Ophthalmic artery
			Cavernous branches	ILT
			Petrous branch	CN VII supply
	Proximal	AMA	Artery of foramen ovale	ILT
	Distal	Vidian artery		Petrous ICA
	Distal	Artery of foramen rotundum		ILT
	Distal	Anterior deep temporal artery		Ophthalmic artery
Superficial temporal artery	Frontal branch		Supraorbital branch	Ophthalmic artery
Ascending pharyngeal artery	Pharyngeal trunk	Superior pharyngeal artery	Carotid branch (foramen lacunum)	Lateral clival artery
	Neuromeningeal trunk			Vertebral artery (C1)
	Occipital arch			Meningopharyngeal trunk of ICA
	Hypoglossal and jugular branch			CN VII supply
Posterior auricular-occipital artery	Stylomastoid branch			Vertebral artery (C1-C2)
Occipital artery	Muscular branches			Vertebral artery (C3-C7)
Ascending and deep cervical arteries				

Note: MMA indicates middle meningeal artery; AMA, accessory meningeal artery; ILT, inferolateral trunk; ICA, internal carotid artery; CN, cranial nerve.

Ref 3)

Arterial supply of the Cranial nerves

Table 1: Arterial contribution to the vascularization of the cranial nerves and skull base foramina

Cranial Nerves	Intracranial Feeders		Skull Base Foramen	External Carotid Arteries Feeders
	Intracranial Vertebral-basilar Origin	Episternal Internal Carotid Artery (ICA) Origin		
III	PCA	MAT	Superior orbital fissure	Ophthalmic, or lacrimal, or MMA tentorial branches
IV	ASCA	Anteromedial branch of the ILT	Superior orbital fissure	Ophthalmic, or lacrimal, or MMA tentorial branches
V ₁	Remnant of the trigeminal artery	MAT	Superior orbital fissure	Ophthalmic, or lacrimal, or MMA tentorial branches
V ₂	Remnant of the trigeminal artery	Anteromedial branch of the ILT	Superior orbital fissure	Ophthalmic, or lacrimal, or MMA tentorial branches
V ₃ and V ₄	Remnant of the trigeminal artery	Medial branch of the ILT to the artery of the foramen rotundum	Foramen rotundum	Artery of the foramen rotundum (distal maxillary artery)
VI	Remnant of the trigeminal artery	Posterior branch of the ILT, to the accessory meningeal artery	Foramen ovale	Accessory meningeal artery (proximal maxillary artery)
		Clival arteries from ICA	Superior orbital fissure	Clival arteries from neuromeningeal trunk of APA
VII	Auditory artery, branch of the AICA	MAT and anteromedial branch of the ILT	Facial Canal	Petrous branch of MMA
			Facial Canal	Stylomastoid artery from posterior auricular artery or Occipital artery
VIII	Auditory artery, branch of the AICA		Stylomastoid foramen	
IX	Vertebral artery		Jugular Foramen	Neuromeningeal trunk of APA (jugular branch)
X	Vertebral artery		Jugular Foramen (Par nervosa)	Neuromeningeal trunk of APA (jugular branch)
XI	Vertebral artery		Jugular Foramen (Par nervosa)	Neuromeningeal trunk of APA (jugular branch)
XII	Vertebral artery		Hypoglossal canal	Neuromeningeal trunk of APA (hypoglossal branch)

Abbreviations: AICA, anterior internal carotid artery; APA, ascending pharyngeal artery; ASCA, anterosuperior cerebellar artery; ICA, internal carotid artery; ILT, inferolateral trunk; MAT, marginal artery of the tentorium; MMA, middle meningeal artery; PCA, posterior cerebral artery.

Ref 17)

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Clinical features and natural history, treatment indication

송지혜

아주대병원 신경외과

2021 대한뇌혈관내치료의학회 춘계 보수 교육
2021년 2월 27일 (토) 차바이오 콘클라스트

SESSION I. ANATOMY AND PATHOPHYSIOLOGY OF DURAL ARTERIOVENOUS FISTULA

1. PATHOPHYSIOLOGY
2. VASCULAR ANATOMY
3. CLINICAL FEATURES, NATURAL HISTORY, AND TREATMENT INDICATION

Jihye Song, MD., PhD.

Associate Professor, Department of Neurosurgery
Ajou University School of Medicine, Suwon, Republic of Korea

Dura arteriovenous fistula (dAVF)

- dAVF can occur anywhere within dura mater
- Rare disease → limited the sizes of case series examining the untreated clinical course of dAVF
 - Incidence: 0.16 - 0.29/100 000 adults/year
 - 10-15% of intracranial vascular malformations
- Age; fifth and sixth decades
- Female predominance, dAVF men are more likely to display aggressive NEx and present hemorrhage
- Location & venous drainage pattern determines their clinical presentation and potential for serious sequelae

Stroke, 2015, 46:2017-2025, Miller et al

Clinical Features, Natural History and Treatment Indication

Clinical features

- Clinical features: variable
 - dAVF can occur anywhere within dura mater
 - Symptom (Sx) : depending on their location, arterial supply, degree of arteriovenous shunting, magnitude of flow, and most importantly, their venous drainage pattern.
- Sx (-)
 - incidental finding on angiography
 - generally small or located near the occiput and are not a/w CVD
- Sx (+)
 - Mild symptoms
 - headache, pulsatile tinnitus
 - ocular symptoms

Yumans, 7th edition, p5514.

Clinical Features, Natural History and Treatment Indication

Clinical features

- Non-hemorrhagic neurologic deficit (NHND)
 - seizure, dementia, parkinsonism, cerebellar symptoms, apathy, cranial nerve abnormalities, rarely trigeminal neuralgia, cervical myelopathy.
 - global or focal (or both) neurological deficits that may be transient or progressive
 - Related to the presence of associated CVD and venous congestion in the affected vascular territory
- Hemorrhage
 - Overall hemorrhage presentation: 10-34%
 - Risk factor : cortical venous drainage (CVD), presentation, location (posterior fossa), male sex, and increasing age.

Clinical Features, Natural History and Treatment Indication

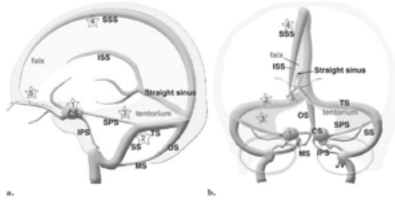


TABLE 401-8 Nonhemorrhagic Symptoms of Dural Arteriovenous Fistulas Based on Location of Venous Outflow

Venous Drainage	Potential Symptoms
Cavernous sinus (e.g., anterior orbital fossa or carotid-cavernous fistula)	Proptosis Chemosis Ophthalmoplegia Retro-orbital pain Visual acuity decline Pulsatile tinnitus Hydrocephalus Papilloedema Seizures
Transverse-sigmoid sinus	Dementia Cerebral neuropathies Coma Cerebral myoclonus
Superior sagittal sinus or deep venous system	Hemorrhage (intracerebral, subarachnoid, rarely subdural)
Basilar venous plexus	
Petrosal veins	
Cortical veins	

Younans, 7th edition, p5514-
RSNA, 2004 H.K. et al

Clinical Features, Natural History and Treatment Indication

Headache

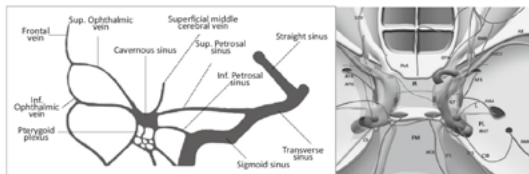
- Common initial symptom
- Unilateral on the side of the dAVF or generalized
- frequently exacerbated by physical activity or changes in position
- Mechanism
 - engorgement of innervated venous collaterals with dural sinus distention
 - brain edema secondary to venous hypertension
 - compression of the trigeminal nerve
 - inflammation as a result of venous thrombosis
- More worrisome causes of headaches include associated hemorrhage, hydrocephalus, or pseudotumor secondary to generalized venous hypertension

Younans, 7th edition, p5514

Clinical Features, Natural History and Treatment Indication

Ocular symptom

- M/C Sx of cavernous sinus(CS) dAVF are ocular symptoms caused by anterior venous drainage

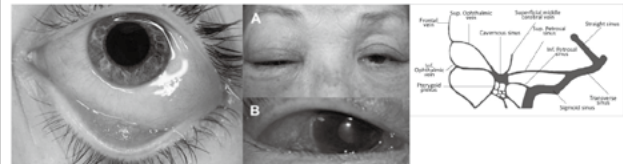


Stroke 2005 Suh et al.

Clinical Features, Natural History and Treatment Indication

Ocular symptom

- 1) Orbital Sx
 - 기전: CS 로 후터의 retrograde venous flow and/or pressure from CS
 - 증상: Chemosis, exophthalmos, periorbital pain, eyelid swelling



Chemosis (swelling of conjunctiva)

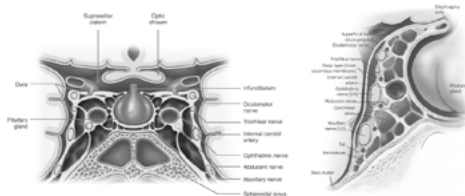
Exophthalmos (bulging eye)

Stroke 2005 Suh et al.

Clinical Features, Natural History and Treatment Indication

Ocular symptom

- 2) Cavernous Sx
 - 기전: Cranial nerve deficit due to bulging or elevated pressure of CS and/or to the steal phenomenon of blood supply to the cranial nerve
 - 증상: diplopia, ptosis, anisocoria, ophthalmoplegia



Stroke 2005 Suh et al.

Clinical Features, Natural History and Treatment Indication

Ocular symptom

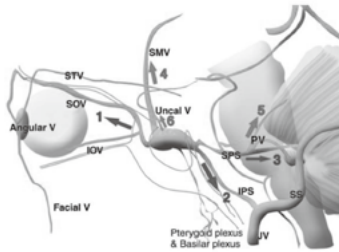
- 3) Ocular Sx
 - 기전: increased venous pressure draining the eyeball
 - 증상
 - decreased vision (subjective symptom or interocular difference of >0.2)
 - increased intraocular pressure (>20mmHg or interocular difference of >5mmHg)
 - eyeball pain, retinal hemorrhage
- Visual loss - one of the most serious consequences of CSdAVF
 - retinal ischemia
 - intaorbital venous pressure 증가
 - intaocular pressure 증가

Stroke 2005 Suh et al.

Clinical Features, Natural History and Treatment Indication

Ocular symptom

- 4) Cerebral Sx
 - 기전: reflux of A-V shunt into superficial MCV or petrosal vein in to the perimesencephalic vein and cerebellar vein → Infarction or venous congestion in the basal ganglia, stem, cerebellum
 - 증상: seizure, hemorrhage

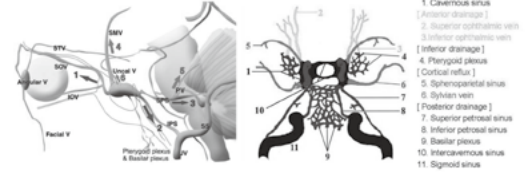


RSNA, 2004, 24:1637-1653 H.K. et al
 Neurology, 2009, Mar;51(3):175-81 H.K. et al

Clinical Features, Natural History and Treatment Indication

Ocular symptom

- CS dAVF가 다른 dAVF 와 다른점
 - extradural location (others; between 2 dural walls in the cranial cavity)
 - postmenopausal female (others; males experience transverse sigmoid sinus); suggesting the possibility of hormonal influence
 - benign clinical symptoms
 - due to various venous drainage route : superior ophthalmic vein, superficial MCV, IPS, coronary sinus to contralateral CS
 - spontaneous regression : 10%~50% of cases



Stroke 2005 Suh et al
 RSNA, 2004 H.K. et al

Clinical Features, Natural History and Treatment Indication

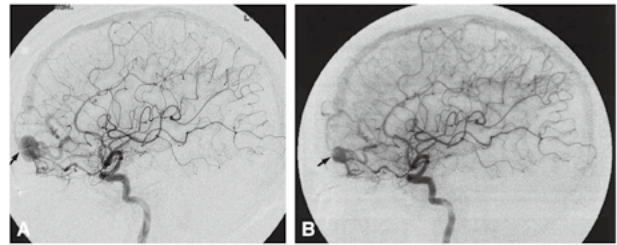
Ocular symptom

- Aggressive neurologic symptoms
 - intracranial hemorrhage are extremely rare but can occur in association with dangerous venous drainage patterns
 - a) cortical venous reflux without other venous drainages (hemorrhagic infarction)
 - b) dominant deep venous drainage (hemorrhage, edema)
 - c) thrombosis of the central retinal vein (blindness)

Stroke 2005 Suh et al
 RSNA, 2004 H.K. et al

Clinical Features, Natural History and Treatment Indication

Ocular symptom

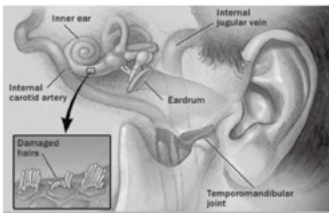


Surg Neurol, 2004; 62:72-75, Han et al

Clinical Features, Natural History and Treatment Indication

Pulsatile tinnitus

- M/C initial Sx in the transverse-sigmoid (Tv-S) sinus location.
- Recruitment of arterial feeders by the dAVF and its close proximity to the middle ear.
- 청진 가능 (bruit can often be auscultated over the cranium)
- ICA/jugular vein/occipital artery compression 시 소리가 줄어든다. (clue to Dx)
- CVD가 dominant drainage 인 환자에서는 드물다. (3% 내외)



- increased flow volume or stenosis of the vessel lumen → result of turbulence in the blood flow → the turbulent flow is transmitted directly to the inner ear and hence causes the pulsatile tinnitus

Yumans, 7th edition, p5514-

Clinical Features, Natural History and Treatment Indication

Pulsatile tinnitus: Cause

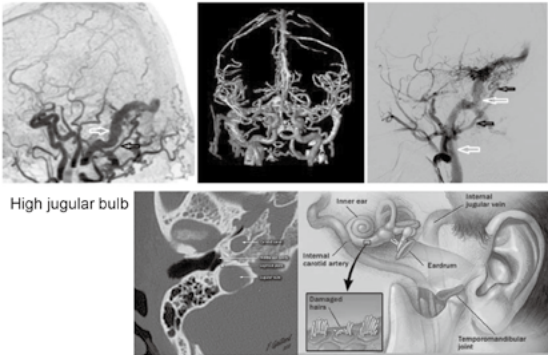
	Congenital	Acquired
Arterial	- Aberrant ICA - Persistent stapedial artery - Lateralized ICA - Fibromuscular dysplasia of the carotid artery	- Intracranial aneurysm (petrous) - Carotid artery dissection/stenosis/atherosclerosis - AVM
Venous	Jugular bulb abnormalities (high riding / diverticula/Dilataciones)	- Transverse sigmoid d AVF - Cavernous sinus d AVF/CCF - Laterally placed sigmoid sinus - Abnormal mastoid emissary veins
Vascular Tumor	Paraganglioma (glomus tympanicum or glomus jugulare)	
Miscellaneous	- Superior semicircular canal dehiscence - Otospongiosis - Inflammatory hyperemia disease (mastoiditis- paget's disease)	- Narrowing of the transverse sinus (transverse sinus / jugular thrombosis) - Benign intracranial hypertension (M/C Pseudotumor cerebri)

Journal of Laryngology&Otolaryngology 2006,120:93-97, Liyanage et al

Clinical Features, Natural History and Treatment Indication

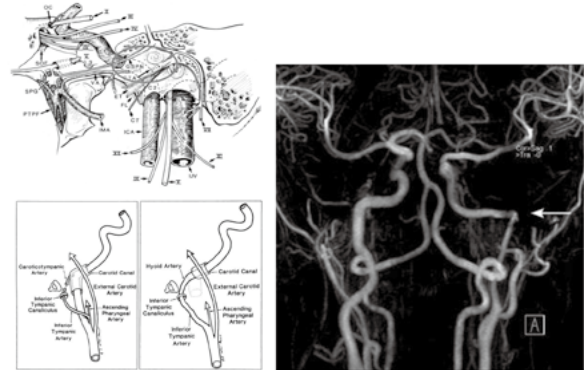
Pulsatile tinnitus

Tv-S dAVF



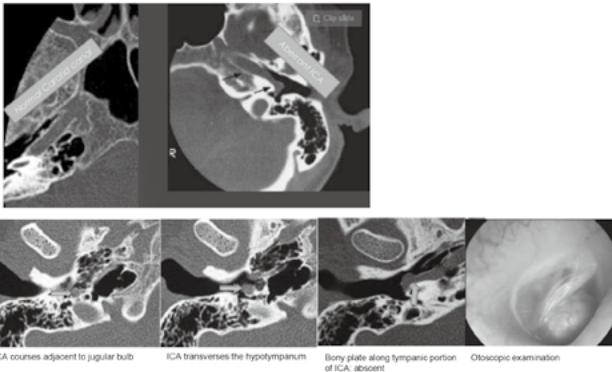
Clinical Features, Natural History and Treatment Indication

Aberrant ICA



Clinical Features, Natural History and Treatment Indication

Aberrant ICA



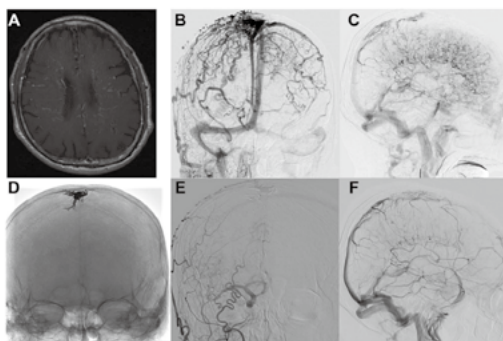
Clinical Features, Natural History and Treatment Indication

NHND

- seizure, dementia, parkinsonism, cerebellar symptoms, apathy, cranial nerve abnormalities, rarely trigeminal neuralgia, cervical myelopathy
- global or focal (or both) neurological deficits that may be transient or progressive
- related to the presence of associated CVD and venous congestion in the affected vascular territory
- related to cerebrospinal fluid malabsorption
- cranial nerve palsies
 - because of an arterial steal phenomenon or occasionally mass effect from an enlarged arterial feeder
- extra-axial hemorrhage in the cervical spine, as well as cervical and upper thoracic myelopathy

Clinical Features, Natural History and Treatment Indication

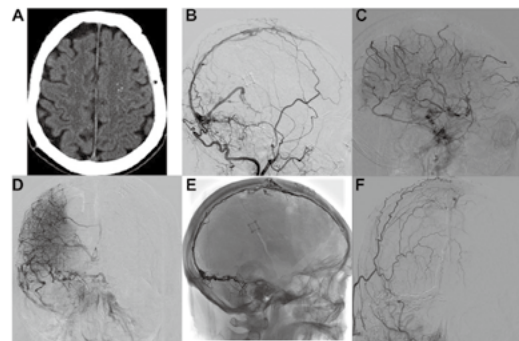
NHND



WNS, 2019, 121.E543-553, Brito et al

Clinical Features, Natural History and Treatment Indication

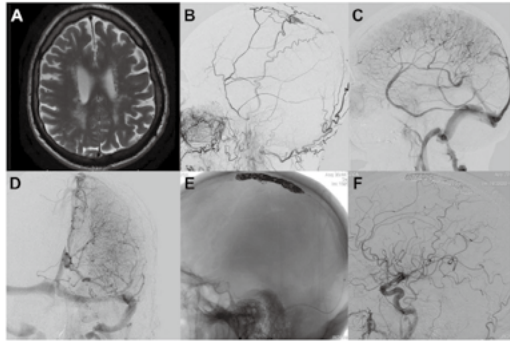
NHND



WNS, 2019, 121.E543-553, Brito et al

Clinical Features, Natural History and Treatment Indication

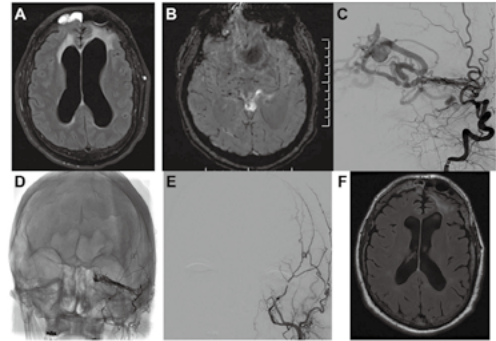
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WNS, 2019, 121 E543-553, Brito et al

Clinical Features, Natural History and Treatment Indication

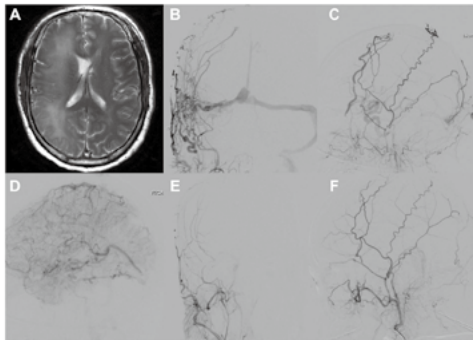
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WNS, 2019, 121 E543-553, Brito et al

Clinical Features, Natural History and Treatment Indication

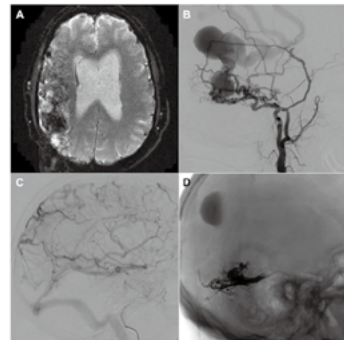
NHND



WNS, 2019, 121 E543-553, Brito et al

Clinical Features, Natural History and Treatment Indication

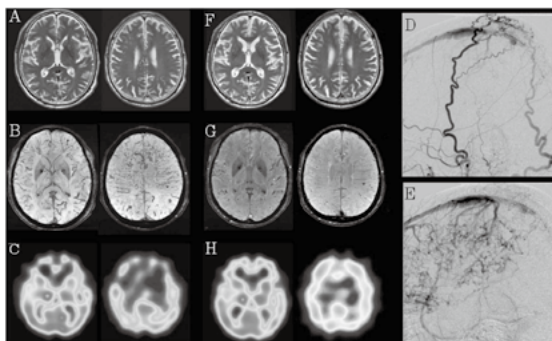
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WNS, 2019, 121 E543-553, Brito et al

Clinical Features, Natural History and Treatment Indication

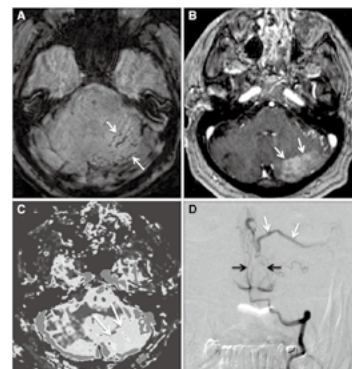
NHND



BMJ case rep, 2014; Fujii et al

Clinical Features, Natural History and Treatment Indication

NHND



Stroke, 2015, 46 2017-2025, Miller et al

Clinical Features, Natural History and Treatment Indication

Hemorrhage

- ICH (Intracerebral hemorrhage), SAH, rarely SDH
- Hemorrhage is the initial presentation of DAVF in up to 34% of patients, most commonly occurring in men between the ages of 50 and 70 years.
- Patients with retrograde cortical venous drainage have a higher likelihood of initially presenting with hemorrhage

Youmans, 7th edition, p5514.

Clinical Features, Natural History and Treatment Indication

Natural History

- Rare disease → limited the sizes of case series examining the untreated clinical course of dAVF
 - Incidence: 0.16 - 0.29/100 000 adults/year
 - 7% -15% of intracranial vascular malformations
 - Overall annual hemorrhage risk for unruptured dAVF : 1.5% to 2%/yr
 - Individual patient risk depends on several factors
 - Venous drainage pattern (cortical venous drainage(CVD))
 - Presenting symptom
 - Venous ectasia
 - Location
- Youssef, 7th edition, p554

Youmans, 7th edition, p5514.
Neurosurgery, 2012;71:594-602. B.A.Gross et al
Neurosurgical Focus, 2009;26:E14 Zipfel et al

Clinical Features, Natural History and Treatment Indication

Venous drainage pattern

TABLE 1. Borden Classification Compared With the Cognard Classification*

Borden		Cognard	
I	Drainage into a dural venous sinus without CVD	I	Drainage into a dural venous sinus with antegrade sinus flow
		IIa	Drainage into a dural venous sinus with retrograde sinus flow
II	Drainage into a dural venous sinus with cortical venous reflux	IIb	Drainage into a dural venous sinus with antegrade sinus flow and cortical venous reflux
		IIa + b	Drainage into a dural venous sinus with retrograde sinus flow and cortical venous reflux
III	Direct CVD	Direct CVD	Direct CVD
		IV	Direct CVD with vein ectasia
		V	Spinal perimedullary venous drainage

Clinical Features, Natural History and Treatment Indication

Venous drainage pattern

- **Borden type I; benign**
- Satomi *et al* (68 observation <112 Pt with Borden type I dAVF)
 - Observation :68/112 (60.7%)
 - Tolerable/stable : 67/68 (98.5%) & Seizure as a result of ICH after CVD development : 1(1.5%)
 - Long term f/u in 50 Patients: Change in venous drainage pattern: 5 (2; CVD (+) → Tx)
 - Palliative treatment : 44/112 (39.3%) (due to unbearable Sx or pressing ophthalmological Sx)
- No hemorrhage & no NHND over mean f/u of 27.9 mo
- Progressed to CVD : 4% (2/50)
- New NHND: 1.5% (1/68)

Clinical Features, Natural History and Treatment Indication

Venous drainage pattern

- **Borden type I; benign**
- Gross *et al* (16 observation < 24 Borden type I < 56 patients with 70 dAVF)
 - No hemorrhage for 409 lesion- years
 - Progressed to CVD : 0
 - Meta-analysis) CVR developed: 1.4%
 - spontaneous resolution; 3/31 (3/24, 13%)

TABLE 2. Demographic and Hemorrhage Data for Patients in Our Cohort Stratified by Borden Type^a

Borden Type	DAVF	M:F Ratio	Mean Age, y	Location	Presentation	Lesion-Years	NHND	Annual NHND Rate	Bleeds	Annual Bleed Rate
I	24	15:9	48	12 TS 5 SSS 4 Other 3 Cav	0 NHND 0 Hem	60.9	0	0	0	0

Neurosurgery, 2012;71:594-602, B.A. Gross et al

Clinical Features, Natural History and Treatment Indication

Venous drainage pattern

- **Borden Type II or III dAVF**
 - CVD (+) few natural history studies

Table 1. Epidemiology and Natural History of Bursitis Type I and II Cardiac Dural Arteriovenous Fistulae

Study	Year	RR (95% CI)	Number of cases	Location	Exposure	Number of cases	Exposure level	RR (95% CI)	RR (95% CI)	Notes
Salmon <i>et al.</i> ¹⁰	1975	1.7 (1.2, 2.3)	12	USA	Occupational	12	High	1.7 (1.2, 2.3)	1.7 (1.2, 2.3)	
					1 case					
					2 cases					
					3 cases					
					4 cases					
Goldman <i>et al.</i> ¹¹	1978	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	1.0 (0.8, 1.2)	
					1 case					
					2 cases					
					3 cases					
					4 cases					
Hess <i>et al.</i> ¹²	1978	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	1.0 (0.8, 1.2)	
					1 case					
					2 cases					
					3 cases					
					4 cases					
Hess <i>et al.</i> ¹³	1978	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	1.0 (0.8, 1.2)	
					1 case					
					2 cases					
					3 cases					
					4 cases					
Hess <i>et al.</i> ¹⁴	1978	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	1.0 (0.8, 1.2)	
					1 case					
					2 cases					
					3 cases					
					4 cases					
Hess <i>et al.</i> ¹⁵	1978	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	1.0 (0.8, 1.2)	
					1 case					
					2 cases					
					3 cases					
					4 cases					
Carter <i>et al.</i> ¹⁶	1978	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	1.0 (0.8, 1.2)	
					1 case					
					2 cases					
					3 cases					
					4 cases					
Carter <i>et al.</i> ¹⁷	1978	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	1.0 (0.8, 1.2)	
					1 case					
					2 cases					
					3 cases					
					4 cases					
Total	1978	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	1.0 (0.8, 1.2)	
					1 case					
					2 cases					
					3 cases					
					4 cases					
Summary estimate										
		RR (95% CI)	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	
		RR (95% CI)	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	
		RR (95% CI)	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	
		RR (95% CI)	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	
		RR (95% CI)	1.0 (0.8, 1.2)	10	USA	Occupational	10	High	1.0 (0.8, 1.2)	

Neurosurgery, 2012;71:594-602, B.A. Gross et al

Clinical Features, Natural History and Treatment Indication

Location

- Cavernous dAVF
 - 84% were type I
 - very low overall risk of hemorrhage
- Anterior fossa, tentorial, petrosal dAVF
 - Vast majority were type III
 - Highly aggressive
 - Lawton et al
 - hemorrhagic presentation in 55% (17 of 31) for tentorial dAVF

TABLE 7. Borden Type as a Function of Dural Arteriovenous Fistula Location

Location	Total No.	Type I, No. (%)	Type II, No. (%)	Type III, No. (%)
Transverse-sigmoid	125	71 (57)	44 (35)	10 (8)
Cavernous	63	53 (84)	7 (11)	3 (5)
Tentorial	35	0	5 (14)	30 (86)
Petrosal	18	1 (6)	3 (17)	14 (78)
Superior sagittal sinus	17	6 (35)	4 (24)	7 (41)
Anterior cranial fossa	10	1 (10)	0	9 (90)
Torcular	7	1 (14)	3 (43)	3 (43)

Neurosurgery, 2012;71:594-602, B.A. Gross et al
Stroke, 2015, 46:2017-2025, Miller et al

Clinical Features, Natural History and Treatment Indication

Treatment indication

- Based on patient characteristics, symptom severity, and risk of serious sequelae (CVD/presentation/venous ectasia/location)
- Low-grade lesions with severe debilitating symptoms (eg, severe tinnitus or visual symptoms resulting in poor quality of life) are candidates for treatment
- Conservative treatment / Endovascular therapy (transvenous /transarterial) Radiation therapy / Surgery

Clinical Features, Natural History and Treatment Indication

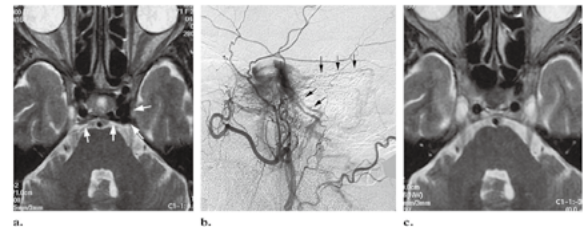
Cavernous sinus dAVF

- Benign clinical course
- Spontaneous regression in 10-50%
- Conservative management for 1-3 months with close clinical Sx and image f/u
 - New Sx or progression of existing Sx
 - Dangerous drainage patterns
 - Should be aware that the low-risk drainage patterns of dural AVFs develop into high-risk patterns with progressive thrombosis or restriction of the cavernous sinus outlet without symptom change
- CS dAVF 에서 즉각적 치료 필요한 경우
 - 1) increased ICP
 - 2) rapidly progressing proptosis
 - 3) diminished visual acuity
 - 4) hemorrhage, TIAs
 - 5) dangerous venous patterns
 - (a) cortical venous reflux without other venous drainages (hemorrhagic infarction)
 - (b) dominant deep venous drainage (hemorrhage, edema)
 - (c) thrombosis of the central retinal vein (blindness)

RSNA, 2004 H.K. et al

Clinical Features, Natural History and Treatment Indication

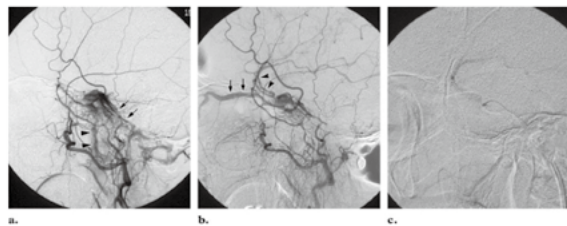
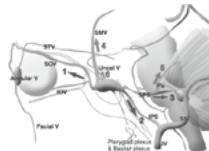
Cavernous sinus dAVF



Clinical Features, Natural History and Treatment Indication

Cavernous sinus dAVF

- M/56
- Sx) mild chemosis, proptosis, diplopia



RSNA, 2004 H.K. et al

Clinical Features, Natural History and Treatment Indication

Cavernous sinus dAVF

- F/56
- Sx) headache, possible ataxia



RSNA, 2004 H.K. et al

Clinical Features, Natural History and Treatment Indication

Transverse sigmoid dAVF

- Most common symptoms are benign (pulsatile tinnitus and headache)
- Transverse-sigmoid sinus dural AVFs are more frequently associated with hemorrhagic and NHND than are CS dural AVFs
- All transverse-sigmoid sinus dural AVFs are considered to require treatment because of the low rate of spontaneous regression without symptomatic events and the relatively high rate of aggressive symptoms

Clinical Features, Natural History and Treatment Indication

Tentorial dAVF

- Retrograde leptomeningeal venous drainage system only
- high risk of aggressive neurologic symptoms
 - Hemorrhage rate: 60% to 74% (19%/yr)
 - NHND: (10%/yr)

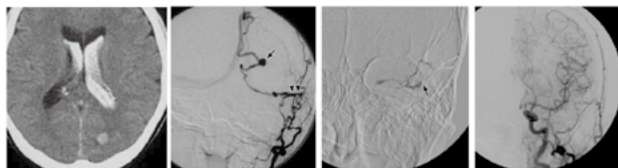
Table 1
Symptoms of Intracranial Dural AVFs

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	...	...
Head, tinnitus	40-50	40-42	70-88	...	...
Headache	...	46-76	8-24	50	12-15
Visual symptoms	28-38	12-28	...	...	...
Cerebral nerve deficits	3	10-20	23-42	29	5-33
Intracranial hemorrhage	Rare	15-28	60-74	23	44-84
Dementia	...	Rare	...	5	...

RSNA, 2004 H.K. et al

Clinical Features, Natural History and Treatment Indication

Tentorial dAVF



Clinical Features, Natural History and Treatment Indication

Superior sagittal sinus dAVF

- frequently associated with restrictive change of the superior sagittal sinus and retrograde cortical venous drainage, aggressive neurologic symptoms are seen in one-half of cases
- Venous congestion of the bilateral frontal lobes due to a superior sagittal sinus dural AVF can cause dementia
- The dementia can be misdiagnosed as a psychogenic or degenerative disorder but can be cured after treatment of the dural AVF

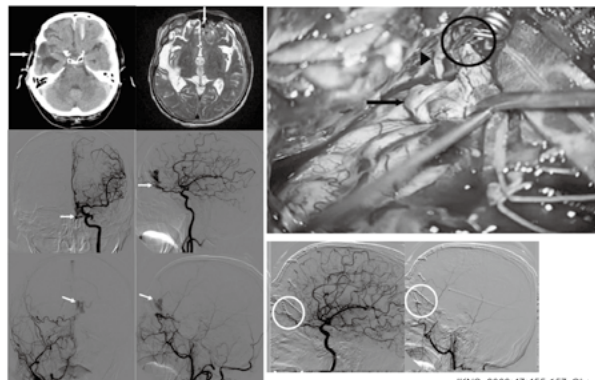
Clinical Features, Natural History and Treatment Indication

Anterior fossa dAVF

- venous drainage pattern similar to that of tentorial dural AVFs with retrograde leptomeningeal drainage and are frequently associated with hemorrhage or NHND

Clinical Features, Natural History and Treatment Indication

Anterior fossa dAVF



JKNVS, 2009;47:455-157, Cho et al

THANK YOU FOR YOUR
ATTENTION

2021 대한뇌혈관내 치료의학회 춘계보수교육

Special symposium

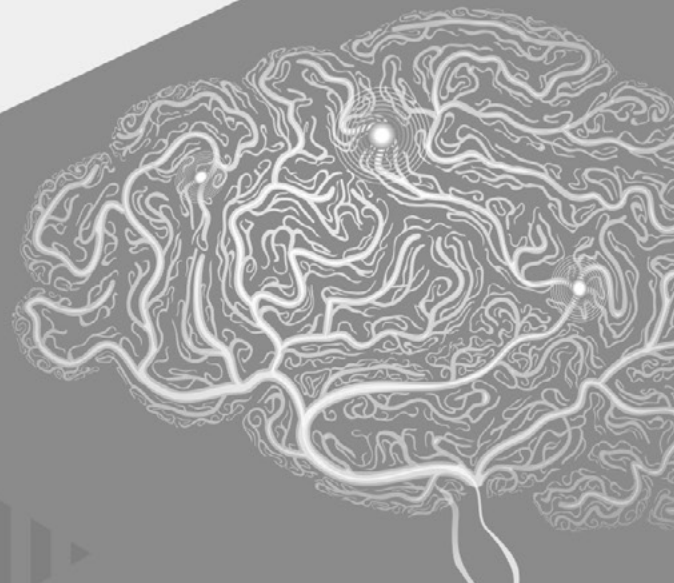
좌장: 윤석만 (순천향대), 고준석 (경희대)

KoNES 뇌졸중 시술 인증제의 발전 방향

KoNES 뇌혈관센터 구성안

신희섭 (경희대)

신승훈 (차의과학대)



KoNES 뇌졸중 시술 인증제의 발전 방향

신희섭

강동경희대병원 신경외과



KoNES 뇌졸중시술 인증제의 발전방향

대한뇌혈관내치료의학회
뇌졸중시술 인증관리위원회

목차

- I. 인증제 연혁
- II. 인증제 현황 및 개요
- III. 인증제 발전 방향



인증제 태동 2010-2012



- 2010년 8월 _ 뇌혈관내수술 인증제 TFT 구성 (이사: 김병태)
_ TFT 회의, 전문가 공청회, 워크숍
- 2012년 7월 _ '우리나라 뇌혈관내수술 인증의 인증기관 수련기관 규정 연구결과 보고서' 발간
- 2012년 10월 _ 대한신경외과학회 제52차 추계학술대회 연설회미나
_ 뇌혈관내수술 인증제도, 의의 및 세부사항: 백인우 (가톨릭대)
_ 다과 전문분야의 인증제도 소개 (심혈관중재술): 전은선 (경희대)
_ 우리나라 뇌혈관내수술 인증제도, 준비 및 경과보고: 김병태 (순천향대)
- 2012년 11월 _ '대한뇌혈관내수술학회 산하 뇌혈관내수술 인증위원회' 구성 (인증위원장: 김병태)

인증제 1기 2013-2014



- 2013년 3월 _ 제1회 대한뇌혈관내수술학회 뇌혈관내수술 인증제 공고

뇌혈관내수술인증제를 도입하면서

뇌혈관내수술인증제는 뇌혈관내수술을 시행하는 의료인의 질적 표준을 수립하여, 뇌졸중 환자 건강 및 안전을 위한 최선의 노력을 기울이고자 합니다. 인증제 도입을 통해, 뇌혈관내수술 인증제 도입을 통해, 뇌졸중 환자 건강 및 안전을 위한 최선의 노력을 기울이고자 합니다.

뇌혈관내수술인증제는 뇌혈관내수술을 시행하는 의료인의 질적 표준을 수립하여, 뇌졸중 환자 건강 및 안전을 위한 최선의 노력을 기울이고자 합니다. 인증제 도입을 통해, 뇌혈관내수술 인증제 도입을 통해, 뇌졸중 환자 건강 및 안전을 위한 최선의 노력을 기울이고자 합니다.

공급적으로 환자의 건강 및 안전뿐만 아니라 수술자가 근무하는 뇌혈관내수술실/뇌혈관중재실의 환경, 즉, 시설장비 및 인력 등의 필수적인 근무여건을 개선하는 것을 목적으로 하고 있습니다.

뇌혈관질환을 가진 우리나라 국민들과 그 가족들이 신뢰할 수 있는 뇌혈관내수술 전문가를 배출하는 좋은 제도로 자리매김

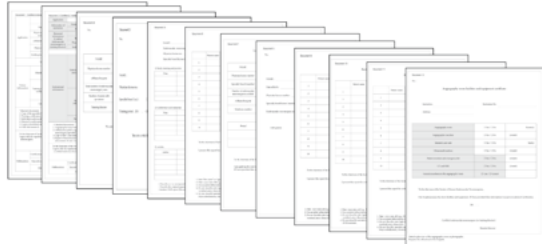
2013년 3월

대한뇌혈관내수술학회 인증제 1기 인증제

인증제 1기 2013-2014



- 2013년 3월 ~ 6월 _ 제1회 대한뇌혈관내수술학회 뇌혈관내수술 인증제 접수 / 심사



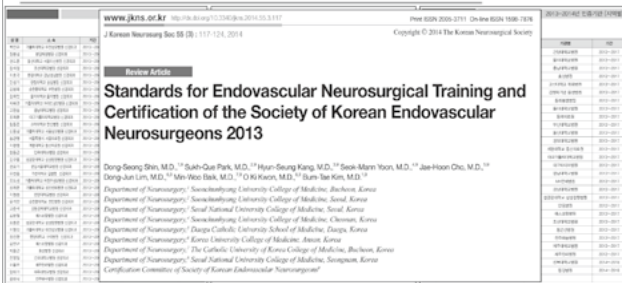
인증제 1기 2013-2014



- 2013년 6월 _ 제1회 대한뇌혈관내수술학회 뇌혈관내수술 인증제 합격 발표
 - _ 인증의 127명 / 인증기관 55개소 합격
 - _ 뇌혈관내치료 유관 학회 최초 인증제 / 최초 인증의 / 최초 인증기관
- 2012년 10월 _ 대한신경외과학회 제53차 추계학술대회 연설회
 - 일본뇌혈관내수술의 현황과 전망: Tamaoki Terrada (Wakayama Medical College)
 - 일본뇌혈관내치료학의 인증의 제도의 현황: Tamaoki Terrada (Wakayama Medical College)
 - 대한뇌혈관내수술학회 인증의 제도의 현황: 김병태 (순천향대)
- 2014년 3월 _ 제2회 대한뇌혈관내수술학회 뇌혈관내수술 인증제 시행
 - 6월 _ 인증의 10명 / 인증기관 2개소 합격

인증제 1기 2013-2014

인증의 137명 / 인증기관 57개소



인증제 2기 2015-2020



- 2014년 11월 _ 뇌혈관내수술 인증위원회 신임위원장 이호국
- 2015년 3월 _ 제3회 대한뇌혈관내수술학회 뇌혈관내수술 인증제 시행
 - _ 재인증 시행 (제1회 인증의/인증기관 중 일부)
 - 6월 _ 인증의 13명 (재인증 32명) / 인증기관 6개소 (재인증 10개소) 합격
- 2016년 6월 _ 제4회 인증제 _ 인증의 9명 (재인증 20명) / 인증기관 1개소 (재인증 9개소) 합격
- 2017년 6월 _ 제5회 인증제 _ 인증의 6명 (재인증 22명) / 인증기관 2개소 합격
- 2018년 6월 _ 제6회 인증제 _ 인증의 20명 (재인증 36명) / 인증기관 3개소 (재인증 29개소) 합격

인증 지원 / 재인증 증가 + 지원/심사의 편의성 증대
자료의 효율적 보관 인증제도의 연속성 확보 = 온라인 시스템 구축

인증제 2기 2015-2020



- 2019년 3월 _ 인증제 (인증의 부분) 온라인 접수/심사 시스템 개발 완료 (유관기관 최초)
- 2019년 6월 _ 제7회 인증제 _ 인증의 20명 (재인증 10명) / 인증기관 2개소 (재인증 3개소) 합격
- 2020년 3월 _ 인증제 (인증기관 부분) 온라인
접수/심사 시스템 개발 완료
- 2020년 1월 _ '뇌졸중수술 인증제'로 명칭 변경
 - 6월 _ 제8회 인증제
 - _ 인증의 22명 (재인증 38명)
 - _ 인증기관 11개소 (재인증 14개소)



인증제 연혁 2013-2021



연도	인증제	인증의 (명)		인증기관 (개소)	
		신규인증	재인증	신규인증	재인증
2013	제1회 인증제	127	-	55	-
2014	제2회 인증제	10	-	2	-
2015	제3회 인증제	13	32	6	10
2016	제4회 인증제	9	20	1	9
2017	제5회 인증제	6	22	2	-
2018	제6회 인증제	20	36	3	29
2019	제7회 인증제	20	10	2	3
2020	제8회 인증제	22	38	11	14

목차



- I. 인증제 연혁
- II. 인증제 현황 및 개요
- III. 인증제 발전 방향

인증제 현황 2021.02



- | | |
|---|--|
| • 인증의
_ 인증 승인 (2013-2020) : 227 명
_ 인증 만료 및 유고 : 10 명
_ 2021년 2월 현재 인증의 : 217 명 | • 인증기관
_ 인증 승인 (2013-2020) : 82 개소
_ 인증 만료 및 기관 폐쇄 : 6 개소
_ 2021년 2월 현재 인증기관 : 76 개소 |
|---|--|

인증제 목적



<제1회 인증제 공고문 발췌>

- 뇌혈관내수술인증제는 뇌혈관내수술을 시행하는 의료인의 질적 표준을 수립
- 궁극적으로 환자의 건강 및 안전 뿐 만 아니라
- 수술자가 근무하는 뇌혈관내수술실/뇌혈관조영실의 환경, 즉, 시설장비 및 인력 등의 필수적인 근무여건을 개선하는 것을 목적

인증제 평가 항목 _ 인증의 지원 자격



- 인증의 _ 수련프로그램 수련의
 - _ 대한민국 법정전문과목 전문의 자격을 취득한 후
 - _ 뇌졸중시술 및 뇌혈관내치료를 시행하는 전문의가 상근하는 병원에서 해당 의료기관이 지정한 수련프로그램에 따라 뇌졸중시술 및 뇌혈관내치료에 대한 수련을 1년 이상 받은 자
- 인증의 _ 단독 시술의
 - _ 대한민국 법정전문과목 전문의 자격을 취득한 후
 - _ 수련프로그램을 수료하였거나 이에 준하는 수련을 받은 후, 1년 이상 주수술자로 뇌졸중시술 및 뇌혈관내치료를 단독으로 시행하고 있는 자

인증제 평가 항목 _ 인증의 진료 실적 요건



- 뇌혈관 조영술
 - _ 주수술자로 150례 이상의 진단적 뇌혈관 조영술을 시행
- 뇌졸중 시술 및 뇌혈관내 치료
 - _ 주수술자 또는 제1조수로 뇌졸중시술 및 뇌혈관내치료를 80례 이상의 증례를 시행
 - _ 80례 중 최소 40례 이상은 다음의 수술이 포함
 - 뇌동맥류 색전술
 - 뇌혈관기형(뇌동정맥기형 및 동정맥류) 색전술
 - 두개강내의 혈관성형술 및 스텐트 설치술
 - 동맥내 색전용배출 및 색전제거술

인증제 평가 항목 _ 인증의 학술 활동 요건



- 연수 평가
 - _ 대한뇌혈관내치료의학회가 인정하는 연수강좌 또는 학술대회에 참가하여 '인증제 평정 인정 규정'에 따라 10점 이상의 연수평점을 이수
- 논문 및 학술 발표 (아래 내용중 1개 이상 충족)
 - _ JCN 또는 타 학술지에 뇌졸중시술 및 뇌혈관내치료에 관련된 내용으로 1편 이상의 논문을 발표
 - _ 대한뇌혈관내치료의학회가 인정하는 학술대회에서 뇌졸중시술 및 뇌혈관내치료에 관련된 내용으로 1편 이상의 연제를 구연 또는 포스터 발표

인증제 평가 항목 _ 인증기관 지원 자격



• 인증기관

- _ 대한뇌혈관내치료의학회에서 인정한 뇌졸중시술 인증의가 상시 근무
- _ 대한뇌혈관내치료의학회에서 요구하는 진료실적, 시설과 장비 및 인력 등을 갖추어야 한다.

현제	비준 2020.12.28
제 4 조 (인증기관 지정의 요건) 인증기관으로 인증 받기 위한 요건은 다음과 같으며, 인증관리위원회는 필요에 따라 신청 기관에 대한 현장조사를 실시할 수 있다. 1. 인증기관으로 지정 받을 수 있는 의료기관은 상급종합병원 혹은 종합병원에서 일정한 중합병원이어야 한다. 해당 기관은 대한뇌혈관내치료학회에서 인정한 뇌졸중시술 인증의(이하 '인증의')가 상시 근무하여야 하며 대한뇌혈관내치료학회에서 요구하는 진료실적, 시설과 장비 및 인력 등을 갖추어야 한다.	제 4 조 (인증기관 지정의 요건) 인증기관으로 인증 받기 위한 요건은 다음과 같으며, 인증관리위원회는 필요에 따라 신청 기관에 대한 현장조사를 실시할 수 있다. 2. 인증기관으로 지정 받을 수 있는 의료기관은 병명(중합)을 전문으로 진료하는 병원 또는 종합병원이어야 하며, 해당 기관은 대한뇌혈관내치료학회에서 인정한 뇌졸중시술 인증의(이하 '인증의')가 상시 근무하여야 하며 대한뇌혈관내치료학회에서 요구하는 진료실적, 시설과 장비 및 인력 등을 갖추어야 한다.

인증제 평가 항목 _ 인증기관 시설/인력/실적 요건



• 인증기관 시설 및 장비

- _ 1개소 이상의 혈관조영실 및 1개 이상의 혈관조영장비가 설치
- _ 뇌혈관질환 개두술이 가능한 수술실 및 뇌혈관질환 환자를 치료할 수 있는 중환자실
- _ 1개 이상의 CT/MRI 보유

• 인증기관 인력

- _ 인증의 자격을 갖춘 전문의가 1명 이상 상시 근무
- _ 뇌혈관질환 개두술이 가능한 신경의과 전문의가 1명 이상 상시 근무
- _ 1개소의 혈관조영실 당, 혈관조영실 전담 방사선사 1명 이상, 간호사가 1명 이상 상시 근무
- 인증기관 진료실적
_ 연간 40례 이상의 뇌졸중시술 및 뇌혈관내치료를 시행

타 유관 학회 비교 _ 대한뇌졸중학회



인증제 평가 항목	대한뇌졸중학회
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- 대한뇌졸중학회 뇌졸중센터 인증기관
_ 2021년 2월 현재 인증기관: 65개소

타 유관 학회 비교 _ KSIN



- KSIN 신경중재치료 인증의
_ 2021년 2월 현재 인증의: 117명
- KSIN 신경중재치료 인증병원
_ 2021년 2월 현재 인증병원: 45명

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인증제 목표



인증제 지향점

- 뇌졸중기술 - 뇌혈관내치료

OR

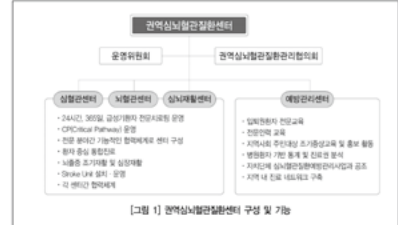
- 뇌졸중치료 - 급성기 치료 전반 (약물, 시술, 수술, 치료 프로토콜)
 - 재활, 예방
 - 환자 교육



2020년 권역심내혈관질환센터 운영지침/평가지표



권역심내혈관질환센터 2020년 운영지침



2. 뇌졸중센터

구분	항목	목표치	현황
1. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
2. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
3. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
4. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
5. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
6. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
7. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
8. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
9. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
10. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개

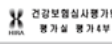


2020년 급성뇌졸중 적정성평가



2020년(9월) 급성뇌졸중 적정성평가 세부시행계획

2020. 7.



1. 평가대상	2020년 7월 1일부터 2020년 9월 30일까지 평가 대상
2. 평가기준	2020년 7월 1일부터 2020년 9월 30일까지 평가 대상
3. 평가결과	2020년 7월 1일부터 2020년 9월 30일까지 평가 대상
4. 평가결과	2020년 7월 1일부터 2020년 9월 30일까지 평가 대상
5. 평가결과	2020년 7월 1일부터 2020년 9월 30일까지 평가 대상
6. 평가결과	2020년 7월 1일부터 2020년 9월 30일까지 평가 대상
7. 평가결과	2020년 7월 1일부터 2020년 9월 30일까지 평가 대상
8. 평가결과	2020년 7월 1일부터 2020년 9월 30일까지 평가 대상
9. 평가결과	2020년 7월 1일부터 2020년 9월 30일까지 평가 대상
10. 평가결과	2020년 7월 1일부터 2020년 9월 30일까지 평가 대상

2020년 급성뇌졸중 적정성평가



구분	항목	목표치	현황
1. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
2. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
3. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
4. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
5. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
6. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
7. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
8. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
9. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개
10. 뇌졸중센터 운영	뇌졸중센터 운영	2020년 12월 31일 현재, 10개 권역심내혈관질환센터가 운영 중임	10개

I. 인증기관 수 증대 및 지역 편중 해결



- 인증의/인증기관

• 유관 학회 확대

• 인증의 : 217 명 / 인증기관 : 76개소

- 인증의 근무 병원 중 비인증기관: 34개소

- 주요 수련 병원 중 비인증기관: 19개소

• 인증병원 신청 폭리

- 지역별

지역	인증병원
서울	12
경기	13
인천	6
강원	3
충북	4
대전, 충남	6
대구, 경북	11
부산, 울산, 경남	14
전북	4
광주, 전남	2
제주	1

I. 인증기관 수 증대 및 지역 편중 해결



• 인증기관

– 뇌혈관질환 전문병원 또는 이에 준하는 병원의 인증기관 신장 독려

제 4 조 (인증기관 지칭의 요건)	제 4 조 (인증기관 지칭의 요건)
인증기관으로 인증 받기 위한 요건은 다음과 같으며, 인증관리위원회는 필요에 따라 신청 기관에 대한 현장조사를 실시할 수 있다.	인증기관으로 인증 받기 위한 요건은 다음과 같으며, 인증관리위원회는 필요에 따라 신청 기관에 대한 현장조사를 실시할 수 있다.
1. 인증기관으로 지칭 받을 수 있는 의료기관은 상급종합병원 혹은 인증위원회에서 인정한 종합병원이어야 한다. 해당 기관은 대한뇌혈관내치료학회에서 인정한 뇌졸중시술 인증의(이하 '인증의')가 상시 근무하여야 하며 대한뇌혈관내치료학회에서 요구하는 진료실적, 시설과 장비 및 인력 등을 갖추어야 한다.	1. 인증기관으로 지칭 받을 수 있는 의료기관은 상급종합병원-특수 인증위원회에서-인증의-인증기관이어야 한다. 해당 기관은 (식재)대한뇌혈관내치료학회에서 인정한 뇌졸중시술 인증의(이하 '인증의')가 상시 근무하여야 하며 대한뇌혈관내치료학회에서 요구하는 진료실적, 시설과 장비 및 인력 등을 갖추어야 한다.

II. 평가 항목 개선 - 시술 항목 조정



• 인증의

– 150례 이상의 뇌혈관조영술

– 80례 이상의 뇌졸중시술/뇌혈관내치료

(40례 이상은 뇌동맥류 색전술, 뇌혈관기형 색전술, 두개강내의 혈관성형술 및 스텐트 설치술, 동맥내 적전용해술 및 색전제거술)

• 인증기관

– 연간 40례 이상의 뇌졸중시술/뇌혈관내치료

– 재인증은 5년간 연간 평균 40례

- ⇒ 1) 연간 시술 증례 수 상향
2) 40례 중 뇌동맥류 색전술, 동맥내 색전제거술의 최소 증례 전수 지정

III. 평가 항목 개선 - 치료 대상 (뇌졸중) 질환 규정



• 뇌졸중

– 국가 기관의 기준은 지나치게 뇌경색 위주

– 뇌졸중 치료의 중요성에 대한 인식 개선

: 뇌지주막하출혈 / 뇌동맥류

: 뇌내출혈에 대한 개두술

• 인증기관

– 치료 대상 질환의 규정 없음

⇒ 1) 질병코드 규정 (I60, I61, I62, I63, I65, I66, I67)

2) 뇌출혈, 뇌경색의 질환 구분

뇌출혈 치료 최소 증례 수 규정

3) 뇌혈관질환 개두술 가능 신경외과 전문의

뇌혈관질환 개두술 증례 수 지정

IV. 평가 방법 개선 - 점수제



• 현재 평가 방법

– 4개 항목, 14개 세부 지표에 대한

pass or fail

– 세부 지표별로 점수화

ex) 조영술 증례 수, 혈관내치료 증례 수,

AngioMachine 대수 (뇌혈관내치료 전용),

CT/MR 대수, 중환자실 전용병상수,

상간 인증대 수, 전담방사선사, 간호사 수 등

뇌졸중시술 전문 인증기관 평가지표	
구분	평가 항목
1. 인증 대상	1.1.1. 인증 대상 기관의 인증 대상
	1.1.2. 인증 대상 기관의 인증 대상
	1.1.3. 인증 대상 기관의 인증 대상
	1.1.4. 인증 대상 기관의 인증 대상
	1.1.5. 인증 대상 기관의 인증 대상
	1.1.6. 인증 대상 기관의 인증 대상
	1.1.7. 인증 대상 기관의 인증 대상
	1.1.8. 인증 대상 기관의 인증 대상
	1.1.9. 인증 대상 기관의 인증 대상
	1.1.10. 인증 대상 기관의 인증 대상
2. 인증 기준	2.1.1. 인증 기준
	2.1.2. 인증 기준
	2.1.3. 인증 기준
	2.1.4. 인증 기준
	2.1.5. 인증 기준
	2.1.6. 인증 기준
	2.1.7. 인증 기준
	2.1.8. 인증 기준
	2.1.9. 인증 기준
	2.1.10. 인증 기준
3. 인증 절차	3.1.1. 인증 절차
	3.1.2. 인증 절차
	3.1.3. 인증 절차
	3.1.4. 인증 절차
	3.1.5. 인증 절차
	3.1.6. 인증 절차
	3.1.7. 인증 절차
	3.1.8. 인증 절차
	3.1.9. 인증 절차
	3.1.10. 인증 절차

V. 인증제 세분화



• 인증의 수련지도 전문의

– “뇌졸중시술 인증의 수련지도 전문의”

– 인증의 취득 후 일정 기간 경과

– 일정기간 뇌졸중시술 증례 수 기준

– 근무하는 기관이 인증기관

⇒ 향후 “수련지도 전문의”에게

수련을 받아야 인증의 지원이 가능하도록

• 인증기관 세분화

– 시술 항목/치료질환에 따라

: 뇌출혈 특화센터 or 급성 뇌경색 특화센터

– 뇌졸중시술 인증의 수련 기관

– 점수제에 따라

: 권역, 지역 인증기관

⇒ 신중히 접근하여야 (인증기관의 서열화 후)

지나친 세분화에 따른 인증제 최소성 저하

VI. 교육/수련 강화 - 의료인, 환자



• 인증의 수련 및 교육

– 연간 이수 평점 강화 (현재 10점)

– KoNES 학회, 연수교육, training course

필수 참여 필수 규정

– KoNES CME 필수 교육 이수

• 전담 방사선사/간호사

– 필수 이수 교육

– 교육 프로그램 개발 (CME)

• 전담 방사선사/간호사

– 환자 대상 교육 프로그램 개발

VII. 진료 환경/안전 개선



• 안전요 기계

- _ 진단용 방사선 발생장치 신고증명서 제출
- _ 특수의료장비 등록 증명서 제출
- _ 영상장비 유지보수 및 품질관리 기록서 제출

• 의료인 근무 시간

- _ 4주간 평균 근무 시간
- _ 휴일 및 야간 일수
- _ 연간 응급 시술 건수

• 방사선 관리

- _ 의료인 연간방사선 피폭선량 (TLD) 제출
- _ 방사선 피폭량 초과시 적절 대처 여부

맺음말

• KoNES 인증제

- _ 최초/최다/최대/유일

• 다학제/의학회

- _ 뇌졸중시술 인증제 or 뇌졸중치료 인증제

• 양적 확대 + 질적 내실

- _ 정책/교육/학술/연구/인증

- _ 모든 역량 집중

많은 관심과 조언 부탁드립니다.

환자의 건강 / 안전한 뇌졸중 치료가 궁극적 목적



KoNES 뇌혈관센터 구성안

신승훈

차의과학대 분당차병원 신경외과

최근 코로나-19 사태 이후로 공공의료의 중요성이 더욱 더 대두되고 있는 상황입니다. 특히 공공의료의 한 축이 되는 필수보건의료 분야에 대한 국가의 관심이 높아지면서, 정부는 2017년 제정된 “심뇌혈관질환의 예방 및 관리에 관한 법률의 제정 및 시행”, 공공보건의료 발전 종합대책(‘18.10.), 지역의료 강화대책(‘19.11.)을 통해 지역 간 의료격차 해소 및 필수의료 서비스의 지역균형발전을 도모하기 위한 전달 체계 확립 필요성을 강조하고 있는 상황입니다.

그간 신경외과 의사들의 국가정책에 대한 관심도는 매우 낮았고, 그에 따른 일정 부분의 피해도 있는 것이 사실입니다. 이에 현재 뇌혈관질환에 대한 국가정책의 흐름 및 현 상태를 되돌아 보고, 신경외과 의사, 특히 뇌혈관질환의 큰 축을 담당하고 있는 대한뇌혈관내치료의학회의 뇌혈관센터 구성안을 살펴봄으로써 국가뇌혈관질환 정책에 이바지하고자 합니다.

2021 대한뇌혈관내 치료의학회 춘계보수교육

Session II. Endovascular treatment of dural AVF

좌장: 신용삼 (가톨릭대), 하성곤 (고려대)

Dural AVF of the cavernous sinus

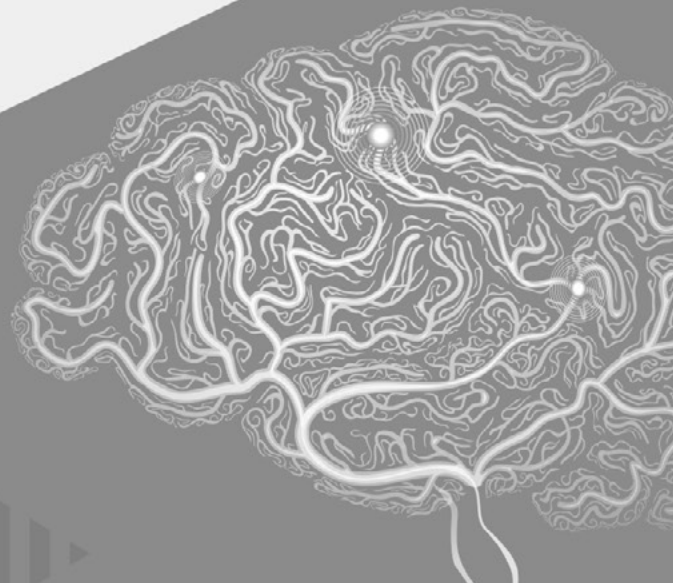
Dural AVF of transverse-sigmoid sinus, tentorial, clival, etc

Spinal AVF

권순찬 (울산대)

이창영 (계명대)

강현승 (서울대)



Endovascular Treatment for Dural AVF of the Cavernous Sinus

권순찬

울산대병원 신경외과

Endovascular treatment is the first-line treatment modality for dural arteriovenous shunts (DAVSs) with the low complication rate and the favorable long-term outcome. With advancements of endovascular techniques and materials, numerous routes of access are available. Endovascular strategies must be carefully tailored to the individual patient. The approach to an individual DAVSs is dictated by a variety of factors including operator's preference for a specific embolic agent, angio-architecture of the fistula, clinical symptoms, and routes of access available.

Endovascular treatment strategies for dural AVF of the cavernous sinus (CS) can be divided into trans-arterial and trans-venous approach. Generally trans-arterial embolization alone has limited effectiveness, more often palliative or preparative treatment before more definite therapy. But, with the availability of Onyx as new highly penetrable embolic agent, there has been a resurgence of interest in trans-arterial approaches. Under appropriate circumstances, trans-venous embolization is highly effective to achieve curative obliteration of high-risk DAVSs.

I would like to review endovascular approaches for DAVSs of CS by Topology, and share our limited experiences during the last 5 years.

Endovascular treatment of dural arteriovenous fistula: dural AVF of the transverse-sigmoid, tentorial, clival, etc.

이창영

계명대 동산병원 신경외과

Currently, treatments for dAVFs include surgical resection, endovascular treatment, stereotactic radiosurgery or a combination of these treatment. Among them, endovascular therapy is generally the first-line treatment for dAVF. The mainstay for endovascular treatment involves embolization of the fistulous connection and its venous components. Either a transarterial, transvenous, or combined accesses are currently being used with their respective pros and cons. The selection of treatment approach depends on the angioarchitecture of the dAVF, the location, the direction of venous flow. The author presents personal experience in endovascular treatment of various dAVFs and reviews the literature focusing on technical and safety aspects of the procedure

Spinal AVF

강현승

서울대병원 신경외과

Spinal DAVF: Conceptual Understanding

강현승

서울대학교병원 서울의대 신경외과

Spinal DAVF: Conceptual Understanding

- Spinal DAVF **homologs**
- Spinal DAVF in general
- Spinal DAVF **patho-anatomy**
- Myth of **Foix-Alajouanine syndrome**
- **Aminoff-Logue** disability scale
- Spinal DAVF treatment outcome: **Curable Cause of Paraplegia**

Dural arteriovenous shunts: a new classification of *craniospinal epidural venous anatomical bases*

- **Ventral** epidural group (n=150): a female predominance, more benign clinical presentations, lower rate of cortical and spinal venous reflux, and no cortical and spinal venous reflux without restriction of the venous outflow.
- **Dorsal** epidural group (n=67): a lower mean age and a higher rate of multiplicity.
- **Lateral epidural** group (n=63): presented later in life with a **male** predominance, more **aggressive** clinical presentations, and **cortical** and **spinal** venous reflux without evidence of venous outflow restriction.

Pierre Lasjaunias 2008 Stroke

Spinal dural arteriovenous fistulas: A congestive myelopathy...

- Spinal dural arteriovenous fistula is a **rare** and **enigmatic** disease entity.
 - The clinical features and structural changes have been recognized since 1926.
 - The pathophysiology and the essentials of treatment, since 1974.
 - Up to the present day it is **unknown why** these fistulas **develop**.
- The fistula between a radicular artery and the corresponding radicular vein within the dural **root sleeve** leads to congestion of the venous outflow of the spinal cord and eventually ischaemia.
- Patients, who are mostly **middle-aged men**, develop a progressive myelopathy, which at the early stages of the disease often mimics polyradiculopathy or anterior horn cell disorder.
- By the time involvement of upper motoneurons or sacral segments makes the diagnosis of SDAVF inescapable, patients suffer from considerable neurological deficits.

2006 Brain

Spinal dural arteriovenous fistulas: A congestive myelopathy...

- The diagnosis relies on MRI.
 - **swelling of the spinal cord**: a centrally located hyperintense signal on T2WI
 - **enlarged and tortuous veins**: hypointense 'flow void' phenomena dorsal to the cord
 - Catheter angiography, to determine the exact location of the fistula and the angio-architecture, on which the mode of treatment depends.
- If the arterial feeder of the fistula is a tributary of the anterior spinal artery, embolization is not possible.
- After embolization recanalization may occur, but this is rarely seen after **filling of the draining vein with glue**.
- Alternatively, operation is a safe and permanent mode of treatment.

2006 Brain

Spinal dural arteriovenous fistulas: A congestive myelopathy...

- No prognostic factors have been reliably established.
- **Muscle** strength and **gait** disturbances respond better to treatment than pain and symptoms related to damage of sacral segments.
- In any middle aged male patient with ascending motor or sensory deficits in the legs, **SDAVF should be considered** in order to prevent irreversible handicap.

2006 Brain

Spinal DAVF: Where is the shunt?

SpDAVF: Microangiographic Study

- In all of the lesions the artery split into daughter vessels that coalesced one to three times to form a skein of arterial loops **in the dura** that invariably emptied into a **medullary vein** without an intervening capillary plexus.
- Several medium-to-small collateral vessels arising from adjacent intercostal or lumbar arteries were commonly present in the dura and **converged** at the site of the AVF to join a **single medullary vein**.
- These results show that spinal dural AVMs are **direct AVFs** that link the dural branch of the radiculo-medullary-dural artery with the intradural medullary vein.

1996 J Neurosurg

Legacy and current understanding of the *often-misunderstood* Foix-Alajouanine syndrome

- The traditional understanding of this clinical syndrome is as a **progressive spinal cord venous thrombosis** related to a **spinal vascular lesion**, resulting in **neurotic myelopathy**. However, spinal venous thrombosis is extremely rare and not a feature of any common spinal vascular syndrome.
- A translation and review of the original 42-page French report revealed 2 young men who had presented with progressive and unrelenting myelopathy ultimately leading to their deaths.
- Pathological analysis demonstrated endomesovascularitis of unknown origin, including vessel wall thickening **without** evidence of luminal narrowing, obliteration of cord vessels, or **thrombosis**.
- Foix and Alajouanine also excluded the presence of intramedullary AVM.

2009 J Neurosurg

Legacy and current understanding of the *often-misunderstood* Foix-Alajouanine syndrome

- At the time, DAVFs had not been described, and therefore this type of lesion was not specifically sought.
- In retrospect, it seems possible that both patients had progressive myelopathy due to Type I DAVFs.
- In the decades since that original report, numerous authors have included **spinal cord venous thrombosis** as a central feature of Foix-Alajouanine syndrome.
- The inclusion of thrombosis in the clinical picture of this syndrome is not only **incorrect** but may leave one with the impression of therapeutic futility, thus possibly preventing successful surgical or endovascular therapy.

2009 J Neurosurg

Legacy and current understanding of the often-misunderstood Foix-Alajouanine syndrome

• Summary of Cases

- Case 1 (M29), Case 2 (M27)
- Pathological Findings

• Extensive pathological analysis in both cases revealed **myelitis** and **spinal cord necrosis**, which were most prominent at the level of the lumbosacral prominence. The spinal cord abnormalities primarily involved the gray matter but also included the white matter. These findings progressively diminished in a rostral direction and disappeared in the region of the midthoracic cord. **Extensive hypertrophy of the intradural vessels** predominantly involved **veins on the surface** of the cord as well as both extra- and intramedullary veins, and to a lesser extent some arteries. Histological analysis of the affected vessels showed "endo-meso-vasculitis with necrotizing tendencies" and considerable hypertrophy of the media. The **vessel lumens** were **widely patent**.

2009 J Neurosurg

Legacy and current understanding of the often-misunderstood Foix-Alajouanine syndrome

- **Thrombosis** as a feature of Foix-Alajouanine syndrome is a **myth** that has been perpetuated most likely because the original report was written in French.
- Foix and Alajouanine *excluded* the presence of vascular malformations within the cord (page 31). They also stated that **no thrombosis** was identified: "Nous avons enfin que . . . il n'y a pas thrombose dans nos cas" (page 12).

2009 J Neurosurg

TABLE 1: Aminoff-Logue disability scales for gait and micturition

Description	Score
gait	
leg weakness, abnormal stance or gait w/o restriction of local motor activity	1
restricted exercise tolerance	2
need for a cane or some support for walking	3
need for 2 canes or crutches for walking	4
unable to stand, confined to bed or wheelchair	5
micturition	
normal	0
urinary hesitancy, urgency, increased frequency, or altered sensation	1
occasional urinary incontinence or retention	2
total urinary incontinence or persistent retention	3

Natural History of Spinal DAVF

- No prospective studies of the natural history of untreated SDAVFs.
 - Such studies, difficult to justify: Treatments are simple, safe, and effective.
- To deduce the natural history through retrospective analysis of patients with spinal vascular malformations who were studied before the introduction of selective spinal arteriography (and thus before a diagnosis or treatment of SDAVF).
- In 1974 **Aminoff** and **Logue** reported on a series of 60 cases with a clinical diagnosis of spinal vascular malformations
 - 1/5, nonambulatory or ambulatory with crutches after 6 months
 - 1/2, severely disabled within 3 years of onset of gait impairment
 - 91%, restricted in their activities within 3 years of symptom onset
- General agreement:
 - Myelopathy due to SDAVF is **progressive** without fistula treatment.

2012 Neurosurg Focus

Brain (1974) 97, 197-210

CLINICAL FEATURES OF SPINAL VASCULAR MALFORMATIONS

BY

M. J. AMINOFF AND VALENTINE LOGUE

(From the National Hospital for Nervous Diseases, Maida Vale Hospital, London W9, and the Institute of Neurology, Queen Square, London WC1)

TABLE IX.—DEVELOPMENT OF NEUROLOGICAL DISABILITY

(1) <i>Progressive course</i>	
Gradual onset, steadily progressive course	36
As above, but previous transient neurological incident (other than pain)	7
Gradual onset, progressive course interrupted by acute deterioration	5
Acute onset, steadily progressive course	5
(2) <i>Non-progressive course</i>	
Acute onset	7
	60

Brain (1974) 97, 211-218

THE PROGNOSIS OF PATIENTS WITH SPINAL VASCULAR MALFORMATIONS

BY

M. J. AMINOFF AND VALENTINE LOGUE

(From the National Hospital for Nervous Diseases, Maida Vale Hospital, London W9, and the Institute of Neurology, Queen Square, London WC1)

In making this study, we have assumed for practical purposes that the degree of disability provides a useful index of the course of the disease, so we have assessed the degree of disability in our patients on two or more occasions. Disturbances of gait were common, due to weakness and/or loss of balance. We have graded these as (1) onset of leg weakness, abnormal stance or gait, without restriction of locomotory activity; (2) restricted exercise tolerance; (3) requires one stick or some support for walking; (4) requires crutches or 2 sticks for walking; (5) unable to stand, confined to bed or wheelchair. Disturbances of micturition have been classified as mild—hesitancy, urgency or frequency; moderate—occasional urinary incontinence or retention; severe—total urinary incontinence or persistent retention. Disordered control of defecation has been similarly classified as mild—constipation; moderate—occasional fecal incontinence or severe intractable constipation; severe—fecal incontinence.

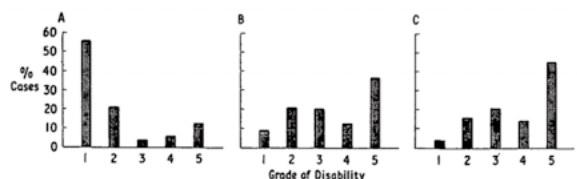


FIG. 1.—Severity of gait disorder. A, Six months after its onset; B, At or within three years of its onset; C, Within the period of follow-up.

Prognosis/Outcome of Spinal DAVF

- Following successful surgical or endovascular treatment of SDAVFs, almost 90% of patients experience **stabilization** of or improvement in their symptoms.
 - 55% of patients had symptom improvement after treatment, 34% had no change, and 11% had neurological deterioration.
- When improvement occurs, patients gain 1–2 points on average on the Aminoff-Logue disability scale for ambulation.
 - This is largely accounted for by an improvement in **motor** symptoms, the symptoms that tend to respond best to treatment, which occurs in approximately two-thirds of treated patients.
- Sensory** symptoms such as numbness, dysesthesia, or burning pain tend to improve less than motor symptoms, but improvement has been reported in 12%–43% of patients.
 - While most patients at least have stabilization of sensory symptoms, these worsen in 14%–22% of patients, and can be a cause of significant pain.
- Sphincter** disturbances tend to recover less well, remaining impaired in up to 75% of those affected and improving in only 15%.

2012 Neurosurg Focus

Outcome Predictors of Spinal DAVF

- One consistent finding in the literature is that patients with severe preoperative neurological deficits have worse functional outcomes when compared with those with mild or moderate preoperative impairment.
 - Patients with preoperative gait disability scores of 2–3 improve more than those in other grade categories.
 - Only **11%** of patients who have severe preoperative disability (defined by a total score of **6–8** on the combined Aminoff-Logue disability scale for gait and micturition) have substantial improvement postoperatively, while **78%** of those with mild disability (score **0–3**) improve, and **29%** of those with moderate disability (score of **4–5**) improve.
- Nevertheless, clinical recovery is possible even for patients with severe deficits, including paraplegia.
- Treatments should not be withheld** from patients who are severely affected, because they still may benefit from surgery.

2012 Neurosurg Focus

Curable Cause of Paraplegia: Spinal DAVF

- Of 42 patients with spinal dural arteriovenous fistulae treated in our institution (surgery or endovascular treatment), 6 were **paraplegic** preoperatively (Grade IV on the McCormick scale and **Grade V** on the **Aminoff scale**, Grade 5 of modified Rankin Scale with motor ASIA between 0 and 10 for both lower limbs). Their clinical history revealed that paraplegia appeared progressively within a period of **<3 months**.
- 3 patients showed **almost normal walking** (Grade I on the McCormick scale, **I** on the Aminoff scale, Grade 1 of modified Rankin Scale) and 3 were **able to walk with a cane** (Grade II on McCormick, Grade **III** on Aminoff scale, Grade 2 of modified Rankin Scale).

2008 Stroke

Outcome Predictors of Spinal DAVF

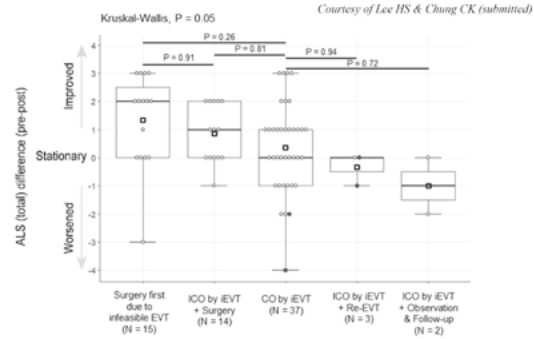
- Success of the treatment procedure undoubtedly influences outcomes.
 - Complete and permanent fistula obliteration provides the best chance for symptomatic improvement and a favorable outcome.
- In the past, endovascular treatment has been criticized because of low initial success rates and high fistula recurrence rates (up to 83% with the use of polyvinyl alcohol).
- More recent data indicate a higher success rate and lower recanalization rates with the use of **liquid adhesive** embolization.
 - The initial success rate of embolization was **69%** in 1 recent study and has ranged from 30% to 90% depending on whether penetration of the proximal vein is required.
- Recanalization—a complication more often seen in endovascular therapy compared with microsurgical clipping—is another consideration.
 - In 1 recent study of 26 patients treated endovascularly, **19%** had recanalization and all had accompanying worsening neurological symptoms.

2012 Neurosurg Focus

SpDAVF: SNUH Experiences (2004 to 2019)

- Of the 71 patients, 56 underwent initial EVT. Complete occlusion was achieved by initial EVT in 37 of 56 patients (**66.1%**).
 - ASpA/PSpA (n=6); Too small/tortuous feeder (n=9)
 - Multiple feeders, more frequently observed in patients with incomplete occlusion than complete occlusion after initial EVT (73.7% vs. 27%, p<0.001).
- The remaining 15 patients underwent surgery as initial treatment.
- Among 19 patients with incomplete occlusion upon initial EVT, 14 underwent additional surgery, 13 of whom (92.9%) obtained improved or stationary functional outcomes.
- Recurrence was observed in 8 of 37 (**21.6%**) patients with complete occlusion upon initial EVT.

Courtesy of Lee HS & Chung CK (submitted)



If EVT is the first-line choice...

- If endovascular treatment is the first-line choice, it should be planned in combination with preparation for **open microsurgery** in the same institution for patients in whom embolization fails.
- Because angiographic evaluations alone cannot predict the risk for recanalization, the embolization cast position must be evaluated by **CT** immediately after the intervention to determine whether it is in the intradural proximal radiculomedullary vein as well as within the dura mater.
- If the embolic material does not penetrate the intradural proximal vein, surgical treatment should be performed as soon as possible to try and block an AVF **permanently** before the progression of incurable neurological deficits.

2015 J Neurosurg

2021 대한뇌혈관내 치료의학회 춘계보수교육

Session III. "Meet-the-Expert" session

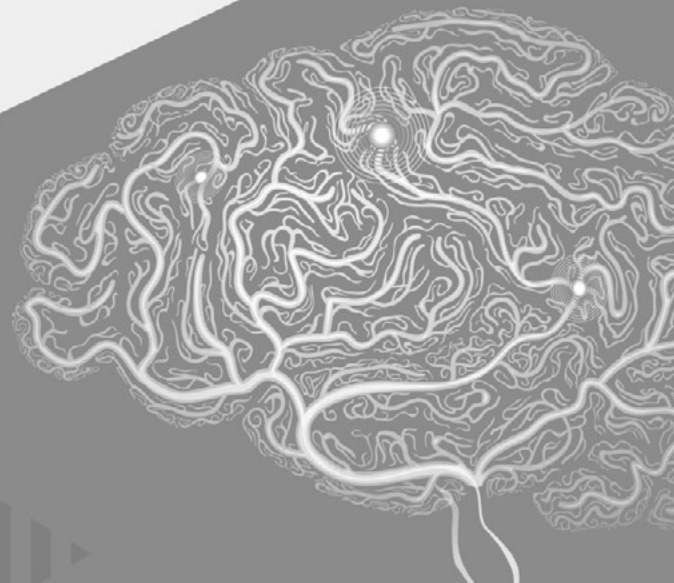
좌장: 백민우 (뉴고려병원), 김범태 (순천향대)

Endovascular treatment of dural AVF

Endovascular treatment of dural AVF

한문희 (중앙보훈병원)

Naoya Kuwayama (University of Toyama, Japan)



Endovascular treatment of dural AVF

한문희

중앙보훈병원 영상의학과



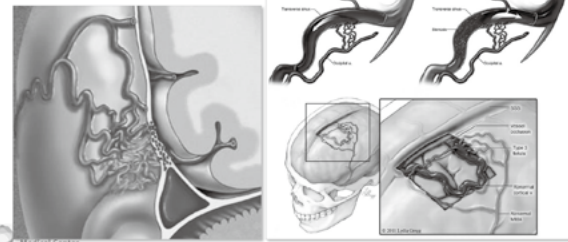
Cranial Dural AVF: Endovascular Treatment

Moon Hee HAN, MD, PhD

Department of Neurosurgery,
VHS Medical Center, Seoul, Korea

Cranial Dural AV Fistula

- Abnormal connection between dural arteries and veins within the dura.



Cranial Dural AV Fistula

A-V communication



Local symptoms by
Venous dilatation
Venous congestion
Increased blood flow

Pathophysiology

Neurological symptoms by
Venous hypert. / infarction
Increased IC pressure
Cerebral hemorrhage

Treatment Goal



Cranial Dural AV Fistula

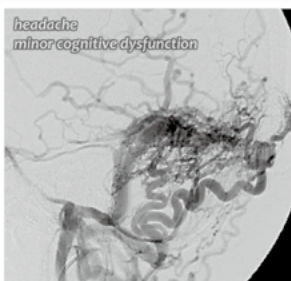
A-V communication



Local symptoms by
Venous dilatation
Venous congestion
Increased blood flow



Cranial Dural AV Fistula



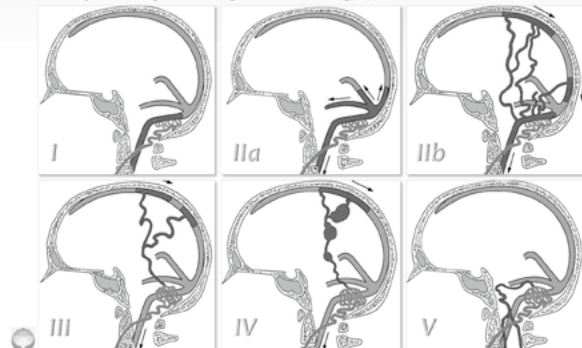
Pathophysiology

Neurological symptoms by
 Venous hypert. / infarction
 Increased IC pressure
 Cerebral hemorrhage

Treatment Goal

Veterans Health Service
 Medical Center

Classification of DAVF - Cognard et al. Radiology 1995;194:671



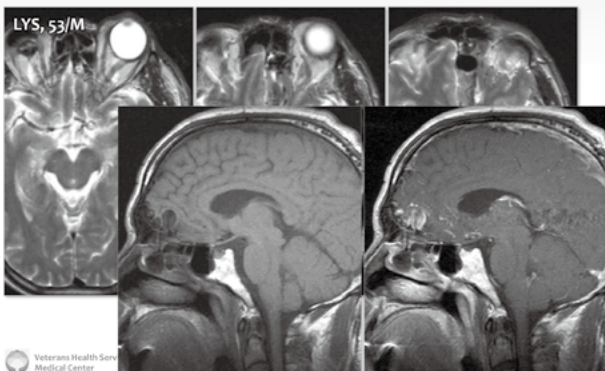
Cranial Dural AV Fistula

Locations

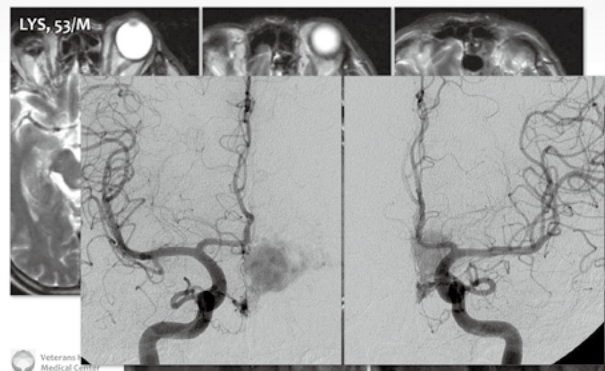
- Cavernous sinus - most common
- Transverse and sigmoid sinuses
- Superior sagittal sinus and Falx cerebri
- Straight sinus and Torcula herophili
- Anterior and posterior cranial bases
- Marginal s., SPS, condylar v., convexity

Veterans Health Service
 Medical Center

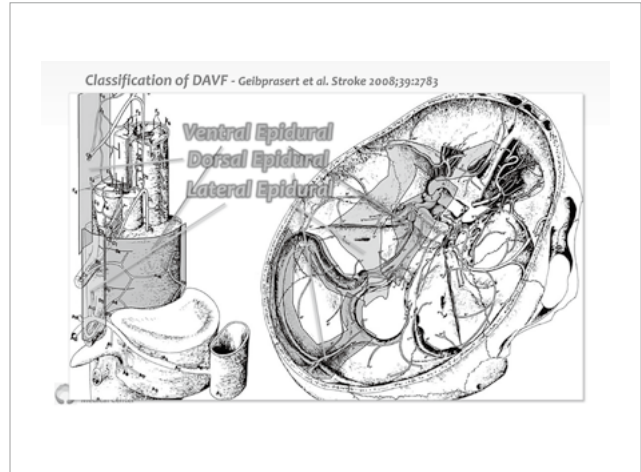
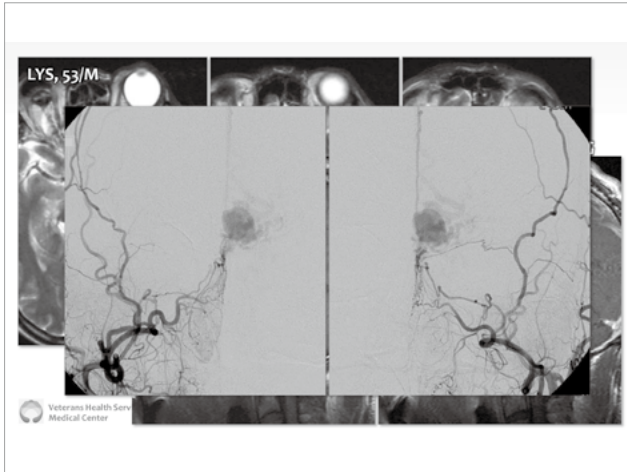
Cranial Dural AV Fistula



Veterans Health Service
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Veterans Health Service
 Medical Center



Cranial Dural AV Fistula

Endovascular Treatment

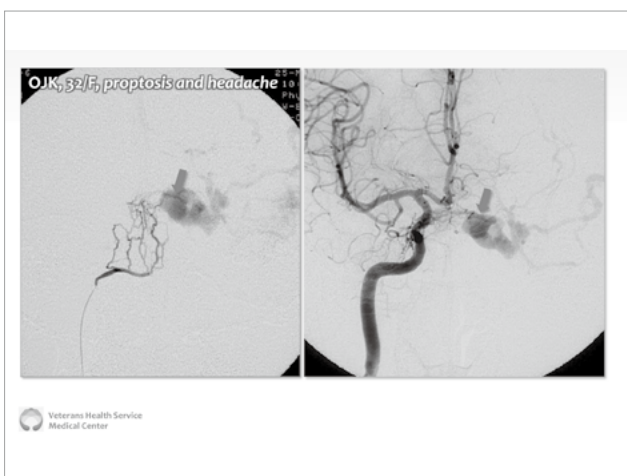
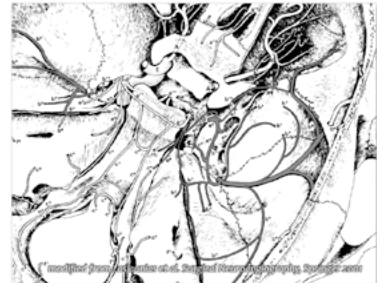
- Transarterial routes
 - Liquids (co-EVA, n-BCA), Particles
- Transvenous routes
 - Coils (detachable, pushable), Liquids

Veterans Health Service Medical Center

Cavernous Dural AV Fistula

Arterial Feeders of Cavernous Sinus

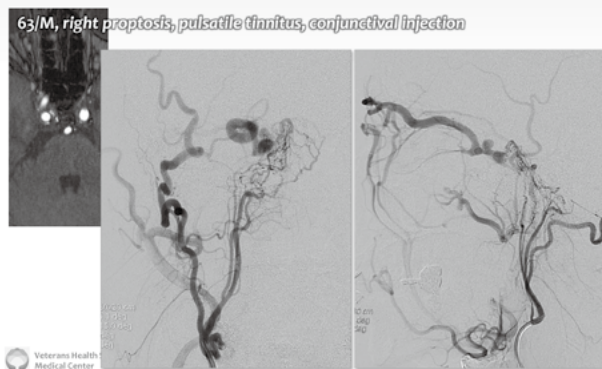
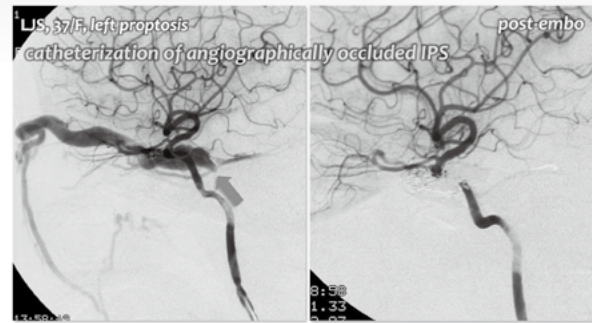
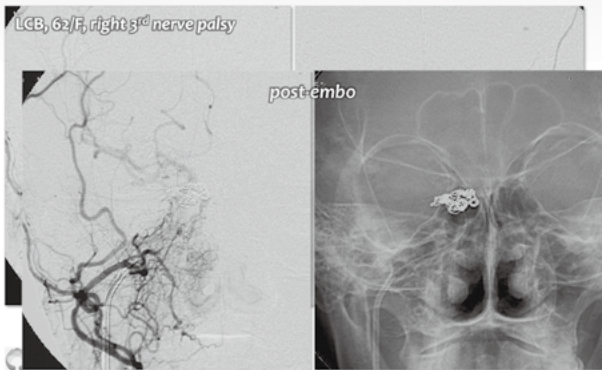
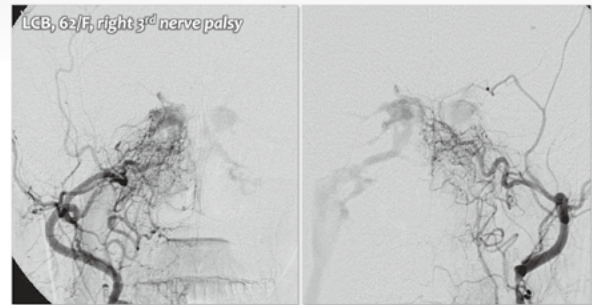
- Middle meningeal A.
- Sphenopalatine A.
- Acc. Meningeal A.
- Inferolateral trunk
- Mening. hypophy. T.
- Asc. Pharyngeal A.

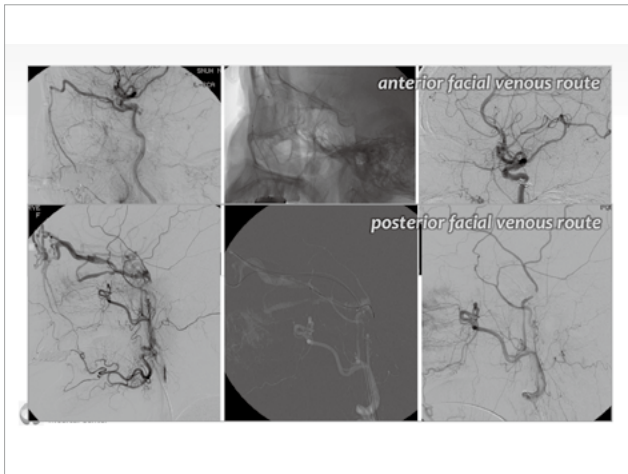


Cavernous Dural AV Fistula

Venous Endovascular Routes

- *Inferior petrosal sinus*
 - ipsilateral or contralateral
- *Superior ophthalmic vein*
 - direct or trans-facial





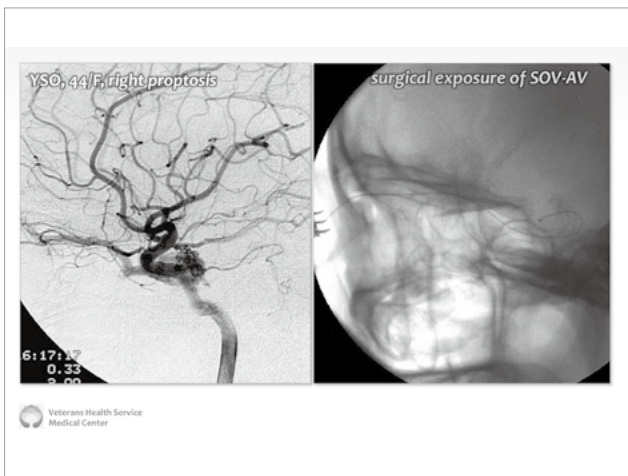
Cavernous Dural AV Fistula

Venous Endovascular Routes

White JB et al. AJNR 2007;28:1415

- Inferior petrosal
 - ipsilateral
- Superior ophthalmic
 - direct or

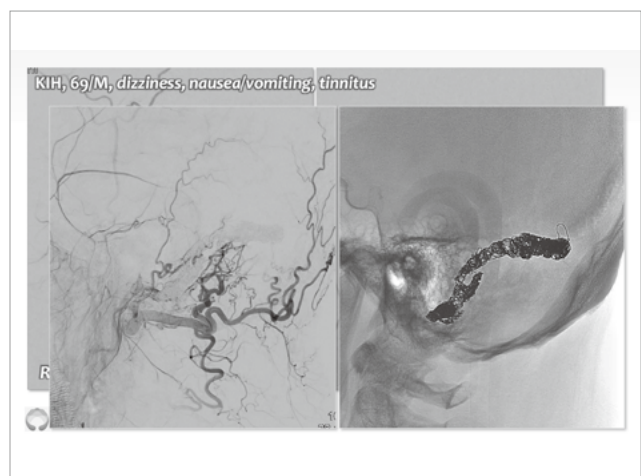
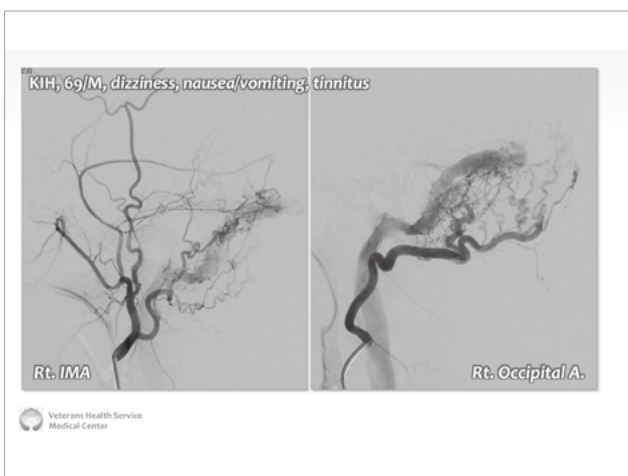
Veterans Health Service Medical Center



Cranial Dural AV Fistula

- Cavernous s...
- Transverse
- Superior sa...
- Straight sin...
- Anterior an...
- Marginal s...

Veterans Health Service Medical Center



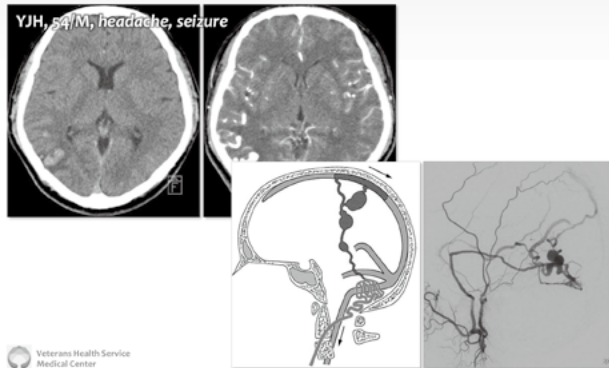
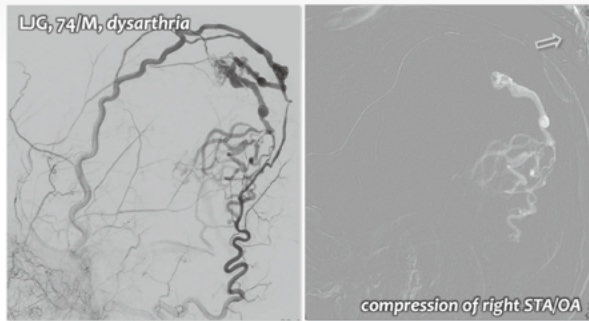
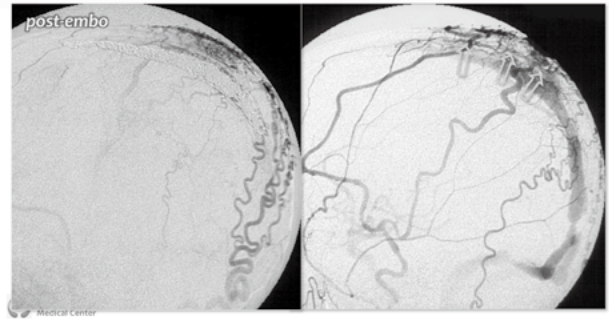
Cranial Dural AV Fistula

Target Points of Occlusion

- Location and extent of A-V connection
- Cerebral circulation and ven. hypertension
- Major clinical problems

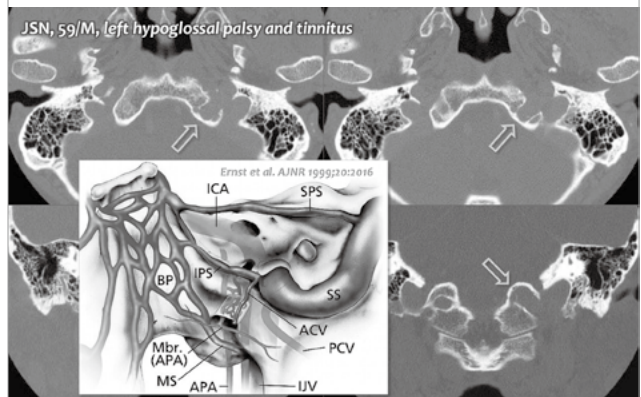


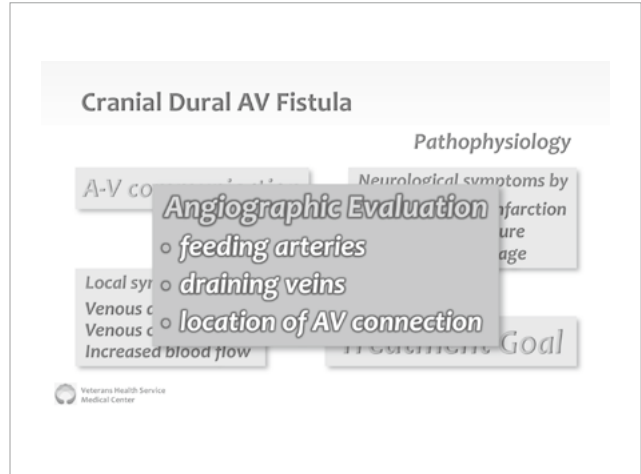
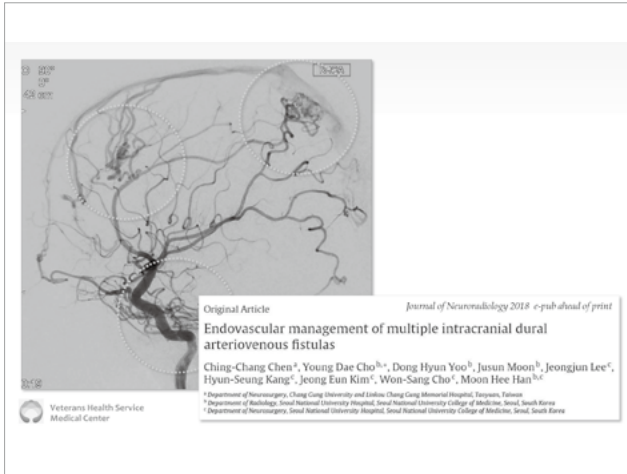
Cranial Dural AV Fistula



Cranial Dural AV Fistula

- Cavernous
- Transverse
- Superior
- Straight s
- Anterior
- Marginal

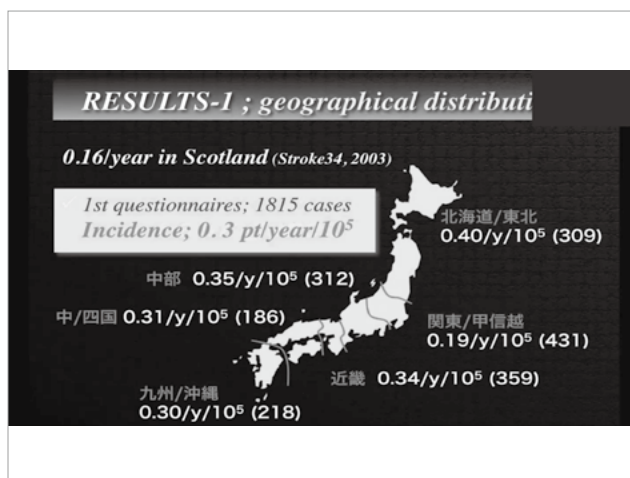
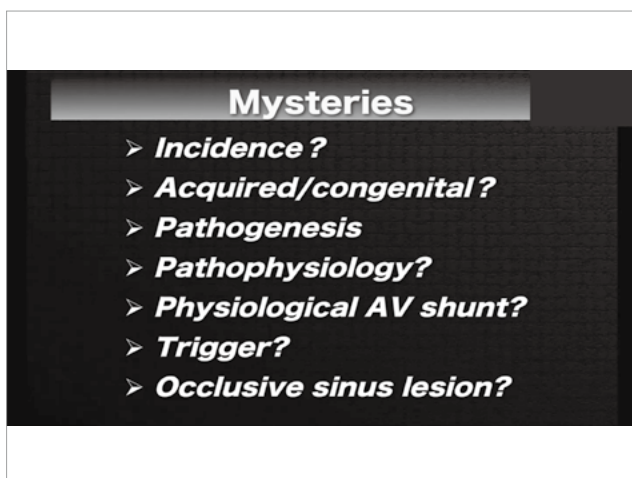
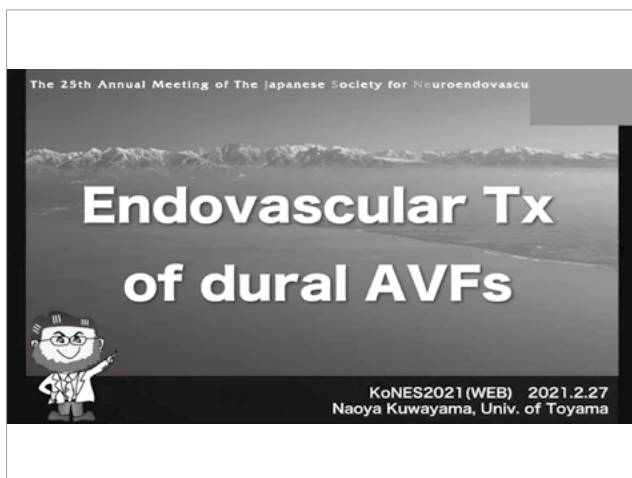




Endovascular treatment of dural AVF

Naoya Kuwayama

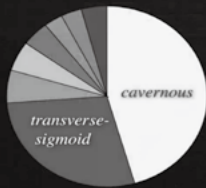
University of Toyama, Japan



Nation-wide survey in Japan

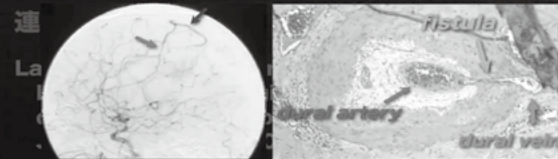
1815 cases were detected (1998-2002)

Incidence: 0.29 patients / 100,000 population / year



CS	46%
TS	28%
spinal	6%
SSS	5%
ACB	4%
CCj	3%
tent	3%
others	4%

Histopathology



Cranial dura: dural artery ⇄ dural vein
 Hori E, Kuwayama N: Histopathological aspect of the non-sinal type dAVF.
 Neurologia Medico Chirurgica, 2007

Pathophysiology

AV fistula between
dural artery & dural vein



Principle of Tx

Feeder occlusion
does not treat AVF itself



Principle of Tx

Sinus occlusion
does not treat AVF itself



Principle of Tx

Glue/Onyx penetration
can treat AVF itself



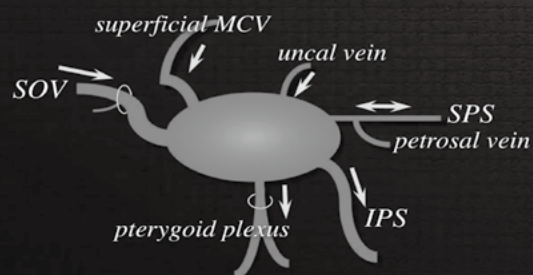
Endovascular treatment

Cavernous sinus

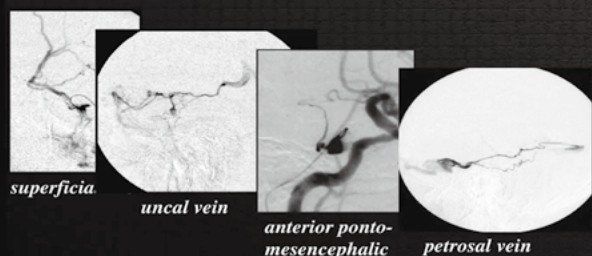
Cavernous sinus

- **Consensus: TVE standard**
 - ⇒ **Outflow occlusion**
 - ⇒ **Selective shunt occlusion**
 - ⇒ **Superselective occlusion**
- **Onyx is not indicated**

Cavernous sinus : in/o

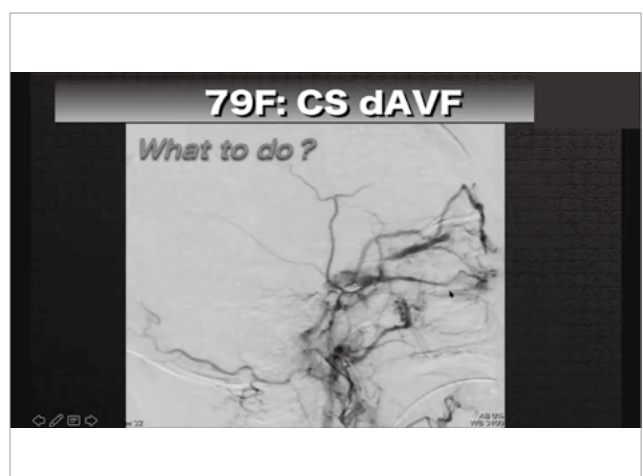
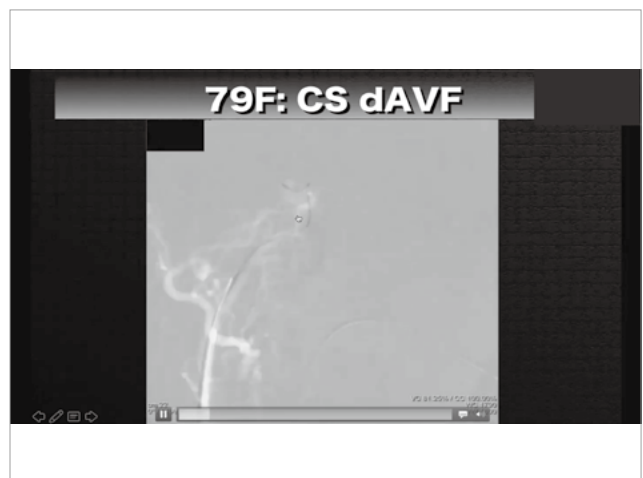
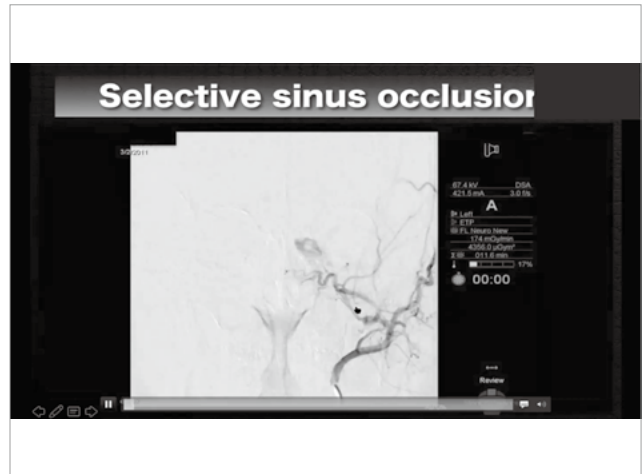


Dangerous drainage



Cavernous sinus

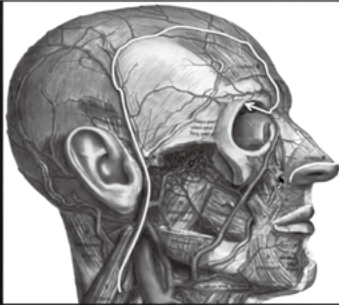
- **Consensus: TVE standard**
 - ⇒ **Outflow occlusion**
 - ⇒ **Selective shunt occlusion**
 - ⇒ **Superselective occlusion**
- **Onyx is not indicated**



79F: CS dAVF

EJV

- STV
- frontal V
- Angular V
- SOV



Rayner Kapsch Band 1.55



Tips of catheterization

Tri-axial catheter

- ⇒ 6/7F + DAC + micro (Echelon)
- ⇒ short connector



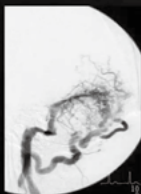
5F DAC
 Echelon-14
 Chikai-14

Hemostatic valve
 T conector

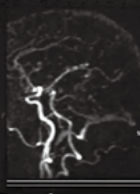
Transverse-sigmoid sinus

TAE or TVE

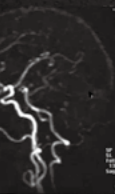
Type-I dAVF: 65M (tinnitus)



initial

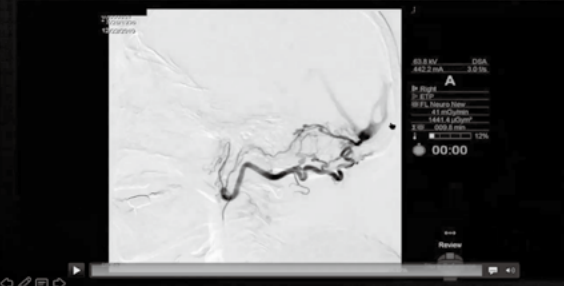


5y



7y

74M (dementia): isolated TS→S



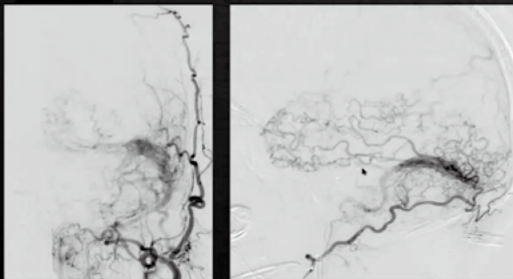
Transverse-sigmoid sinus

Current stream
TVE $\Rightarrow$ Onyx TAE

Onyx

*Approved for dural AVF
in Japan, 2019*

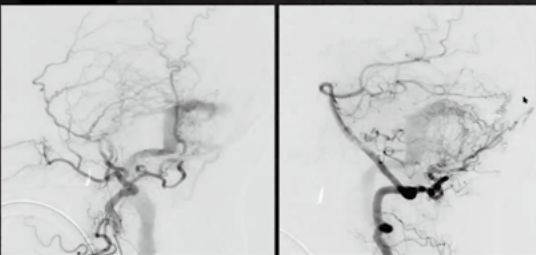
Isolated TSS : 84F



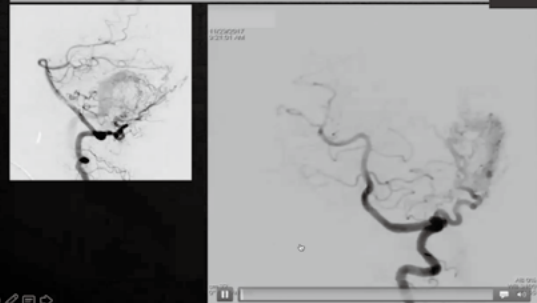
Isolated TSS : 84F



Type-1 TSSdAVF: 84F

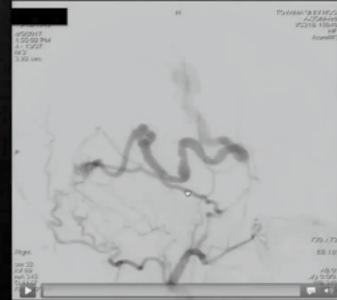


Type-1 TSSdAVF: 84F



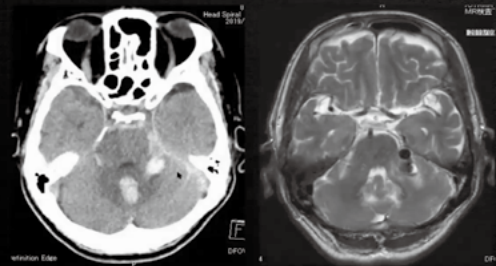
On the wall

On the wall (type-III) : 69

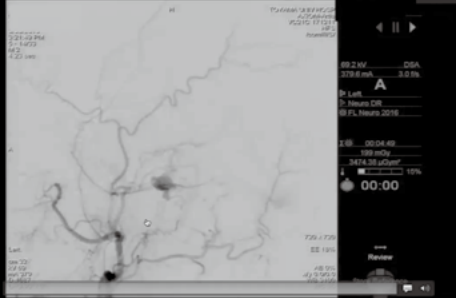


Lateral
tentorium

Tentorial (lateral type): 58

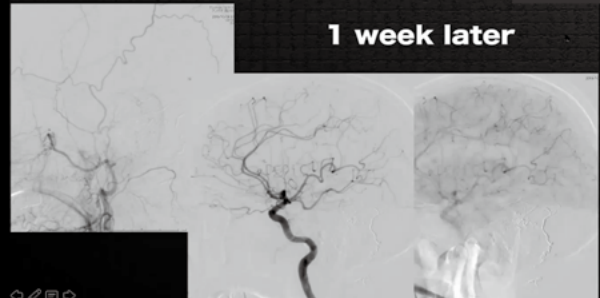


Tentorial (lateral type): 58

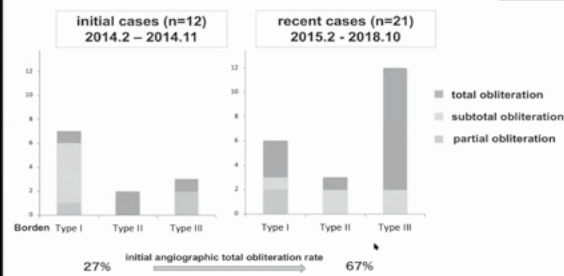


Tentorial (lateral type): 58

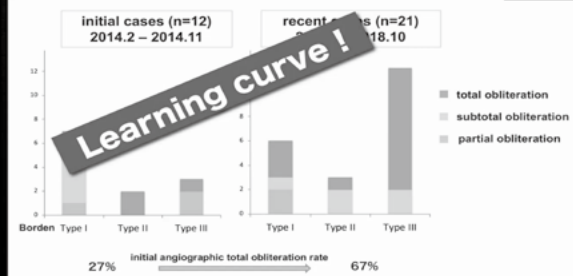
1 week later



Onyx : Results



Onyx : Results



Before & after Onyx

Evolution of treatment and a detailed analysis of occlusion, recurrence, and clinical outcomes in an endovascular library of 260 dural arteriovenous fistulas

Bradley A. Gross, MD, Felipe C. Albuquerque, MD, Karam Moon, MD, and Cameron G. McDougall, MD

Department of Neurosurgery, Barrow Neurological Institute, St. Joseph's Hospital and Medical Center, Phoenix, Arizona

OBJECTIVE Many small series and technical reports chronicle the evolution of endovascular techniques for cranial dural arteriovenous fistulas (dAVFs) over the past 3 decades, but reports of large patient series are lacking. The authors provide a thorough analysis of clinical and angiographic outcomes across a large patient cohort.

METHODS The authors reviewed their endovascular database from January 1996 to September 2015 to identify patients harboring cranial dAVFs who were treated initially with endovascular approaches. They extracted demographic, presentation, angiographic, detailed treatment, and long-term follow-up data, and they evaluated natural history, initial angiographic occlusion, complications, recurrence, and symptomatic resolution rates.

RESULTS Across a cohort of 251 patients with 260 distinct dAVFs, the overall initial angiographic occlusion rate was 70%; recurrence or occult residual lesions were seen on subsequent angiography in 3% of cases. The overall complication rate was 8%, with permanent neurological complications occurring in 3% of cases. Among 102 patients with dAVFs

Gross BA, JNS 126:1884, 2017 (from Barrow Institute)

Before & after Onyx

TABLE 3. Treatment approaches and results before and during the Onyx era

Treatment Approach & Results	Before Onyx (n = 87)	Onyx Era (n = 173)	p Value*
Treatment approach			
Transarterial only	37 (43)	108 (62)	0.008
Cure via transarterial-only approach	20 (23)	74 (43)	0.002
Cure via single arterial-juxtineural embolization	10 (11)	50 (29)	0.002
Initial angiographic occlusion rate			
Overall	52 (60)	131 (76)	0.01
Type I dAVF	27 (42)	44 (59)	0.28
Type II-IV dAVF	25 (44)	87 (50)	0.02
Superior sagittal sinus dAVF	6 (10)	19 (22)	0.22
Cavernous sinus dAVF	16 (27)	27 (34)	0.12
Tentorial/parietal dAVF	10 (17)	31 (34)	0.33
Transverse sigmoid sinus dAVF	1 (2)	38 (57)	0.16
Tentorial dAVF	2 (3)	6 (7)	0.47
Marginal sinus dAVF	3 (5)	7 (10)	0.36
Additional treatment			
Embolization	4 (5)	16 (9)	0.22
Surgery	12 (14)	14 (8)	0.79
Radiotherapy	10 (11)	2 (1)	0.004
Complication rates			
All complications	6 (7)	16 (9)	0.64
Permanent complications	2 (3)	6 (3)	0.72

* Bonferroni type indicates statistical significance.

NBCA Onyx

TAE only: 43% vs 61%

TAE cure: 23% vs 43%

single TAE: 11% vs 29%

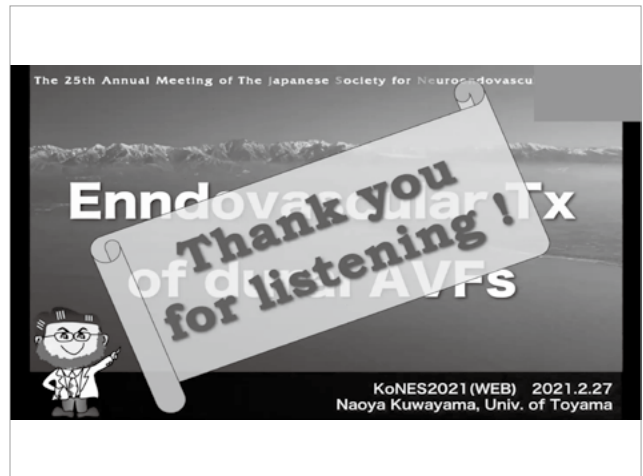
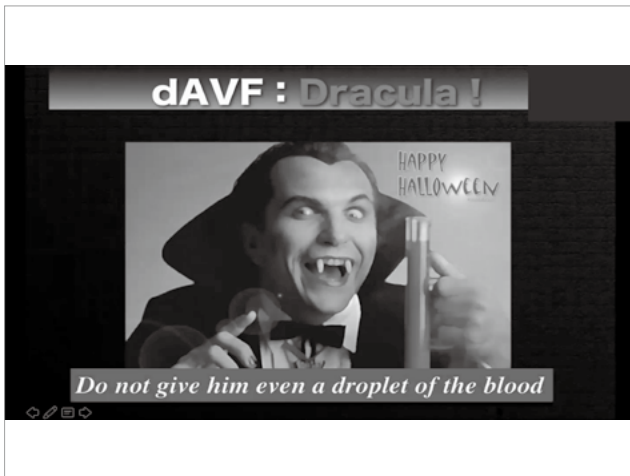
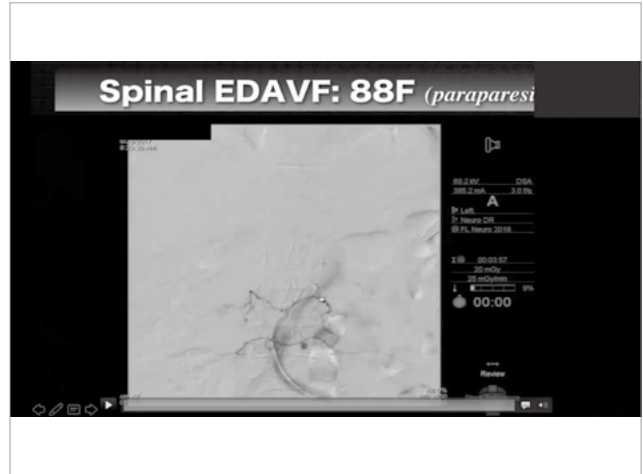
occlusion: 60% vs 76%

SRS: 11% vs 1%

Conclusions : Onyx Tx

- ✓ Onyx is a completely different material from NBCA
- ✓ It goes into other feeders and draining veins
- ✓ Need full anatomic understanding & skillful technique
- ✓ Best indication in type-IIb, III in TSS, SSS, tentorial sinus
- ✓ Type-I / IIa can be treated by skillful hands
- ✓ Can be injected from distal branches of OA and STA
- ✓ Cavernous sinus, CCJ, ACB and spinal lesions are out of indication

Spinal dural/epidural



2021 대한뇌혈관내 치료의학회 춘계보수교육

Session V. Interesting or complicated cases presentation of dural AVF

좌장: 권오기 (서울대), 허준 (명지성모병원)

Case Presentation 1

Case Presentation 2

Case Presentation 3

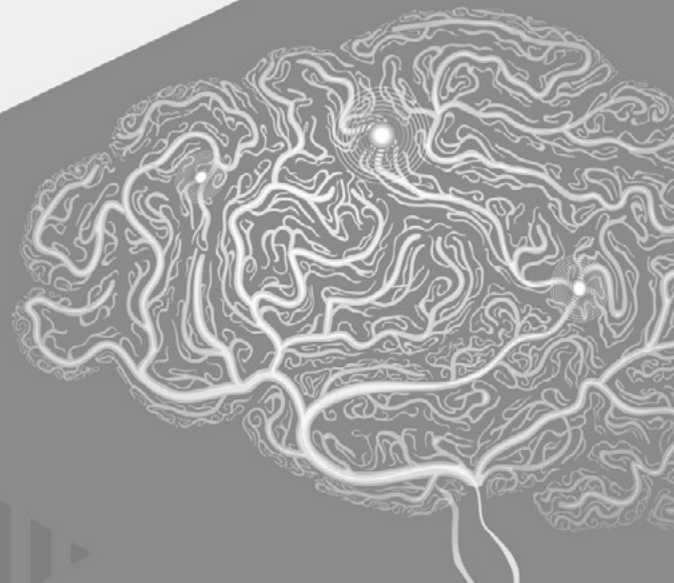
Case Presentation 4

박근영 (연세대)

임종국 (제주대)

고정호 (단국대)

신희섭 (경희대)



Case of Dural Arteriovenous Fistula

박근영

연세대 세브란스병원 신경외과

YONSEI UNIVERSITY

Case of Dural Arteriovenous Fistula

KEUN YOUNG PARK, MD. PhD.
Dept. of Neurosurgery, Severance Hospital
Yonsei University College of Medicine

Severance

Background

Pathogenesis of dAVF

- ✓ Opening of dormant channel between meningeal A & V by venous thrombosis / hypertension
Kerber (1973), Brainin (1983)
- ✓ Neovascularization within the dura mater by angiogenetic factors (VEGF & bFGF)
Rothbart (1996), Uranishi (1999)

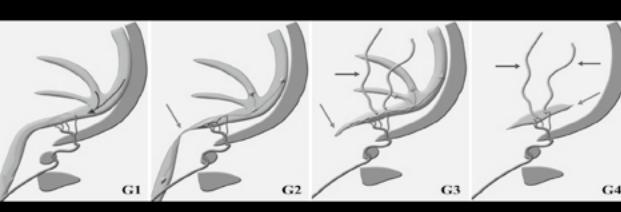
Background



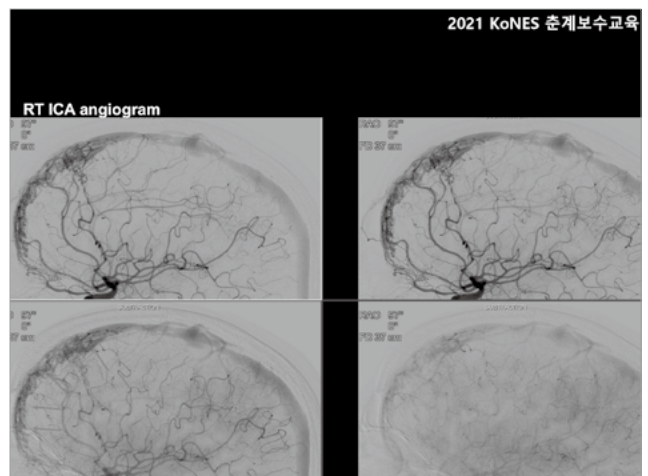
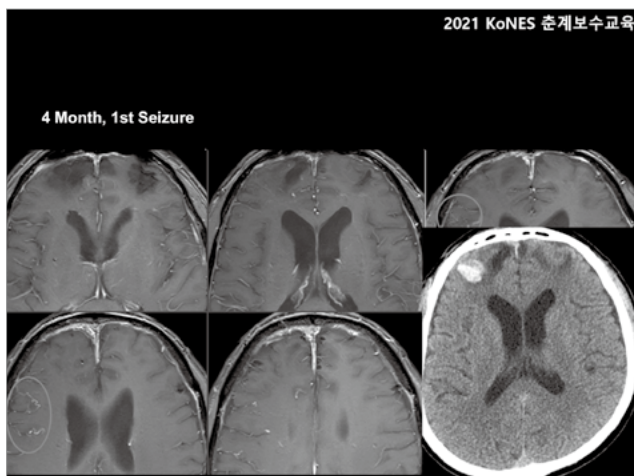
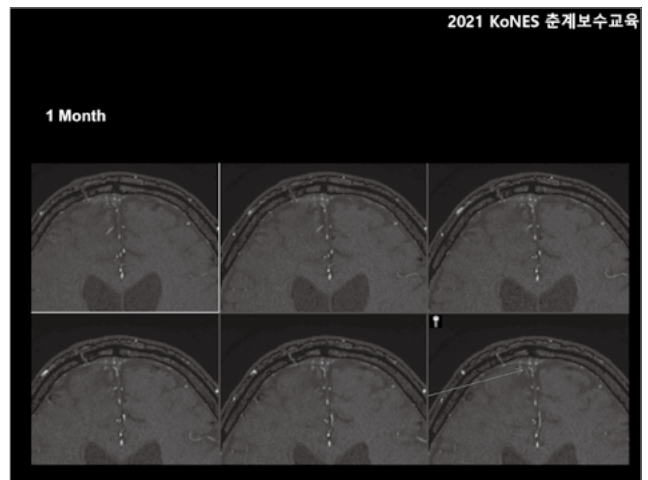
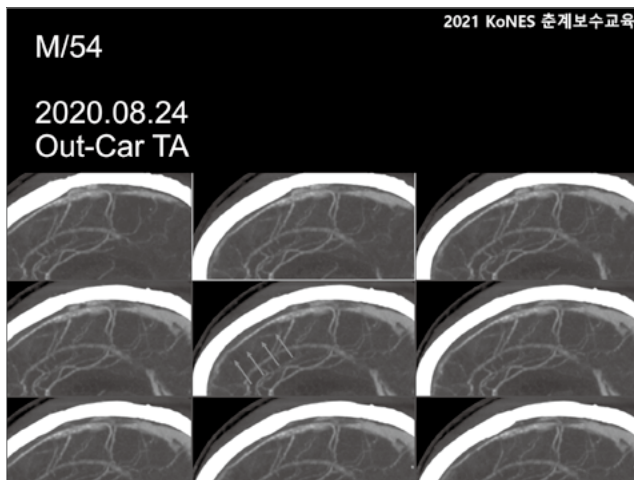
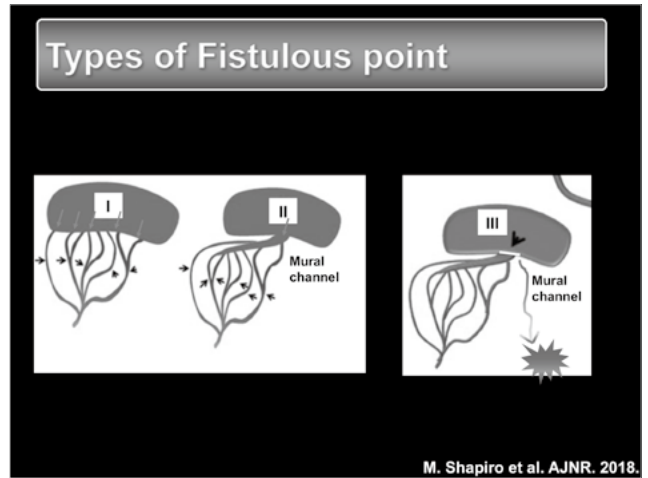
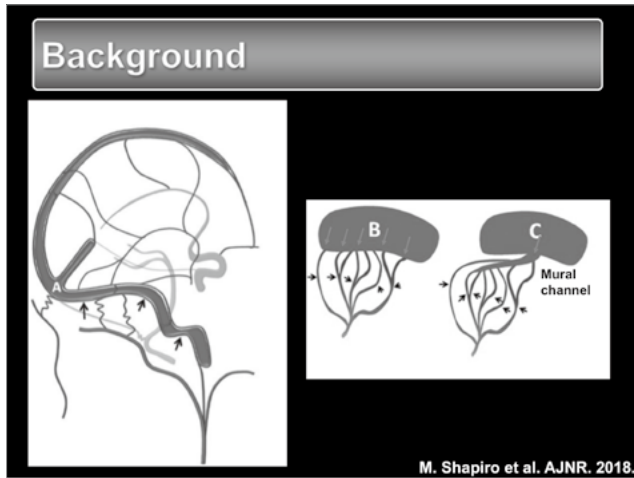
- Venous congestion
- Orbital/Ocular Sx.
- Seizure
- Hemorrhage

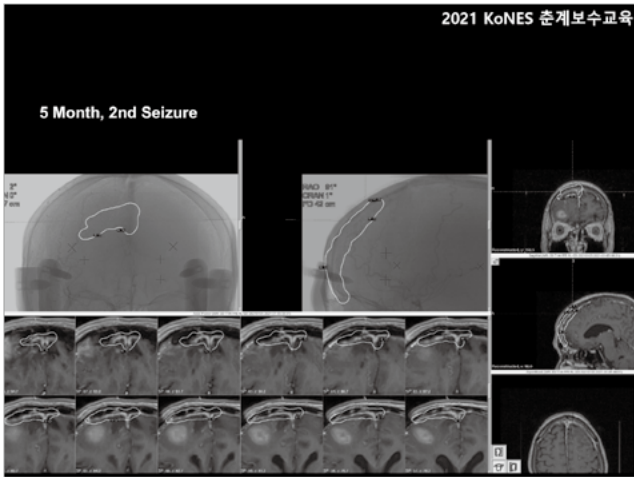
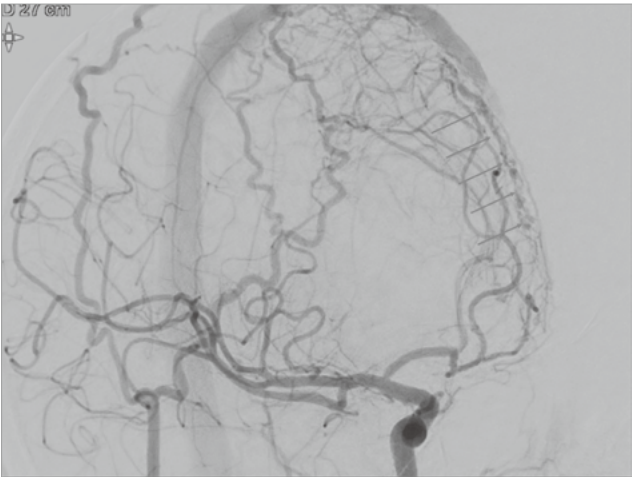
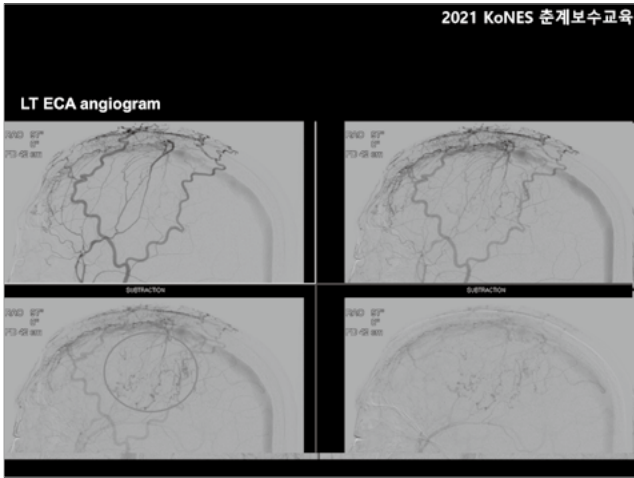
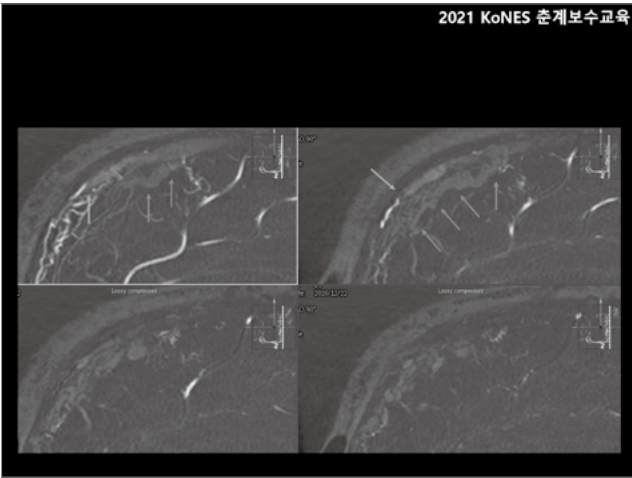
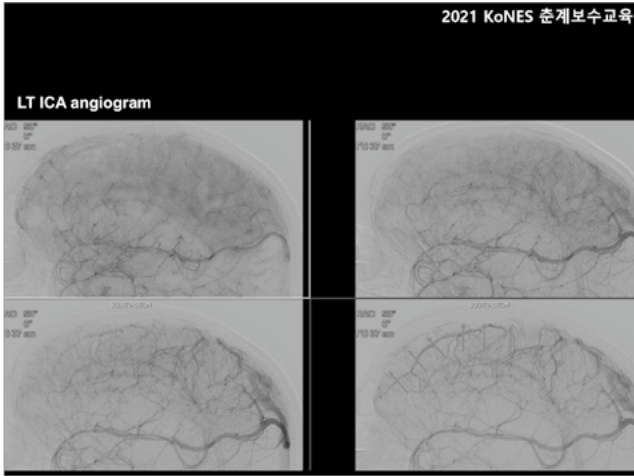
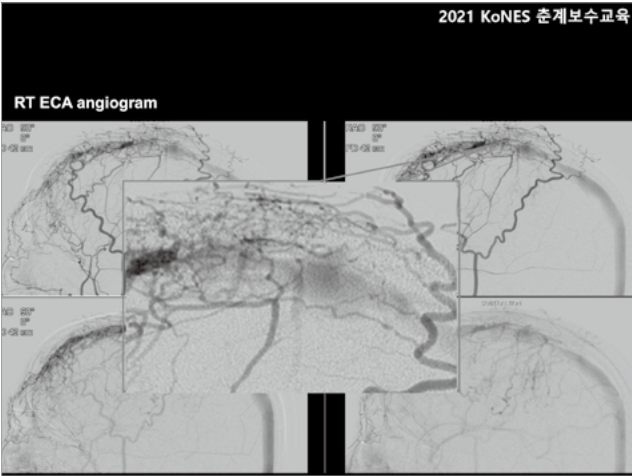
Pathogenesis of dAVF
dAVF ≈ Venous disease

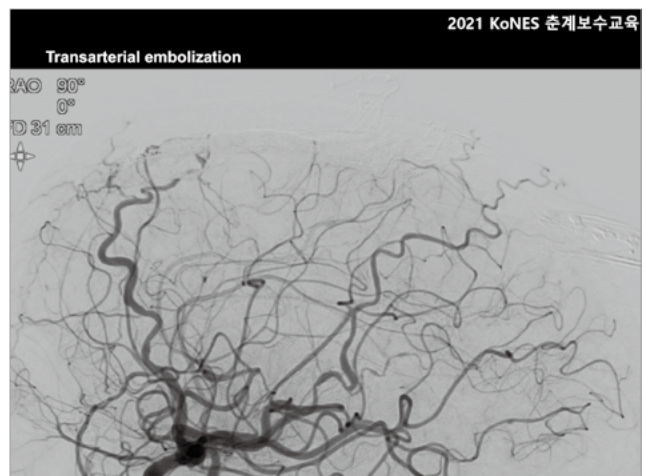
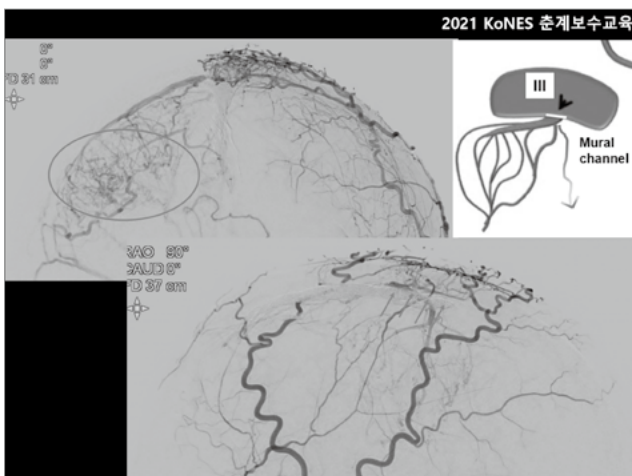
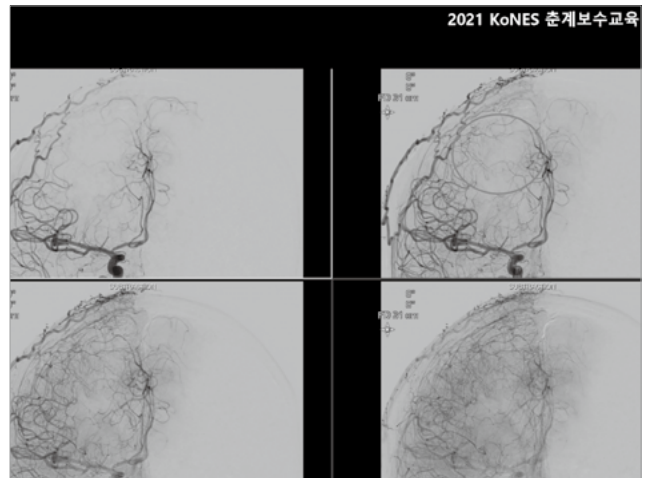
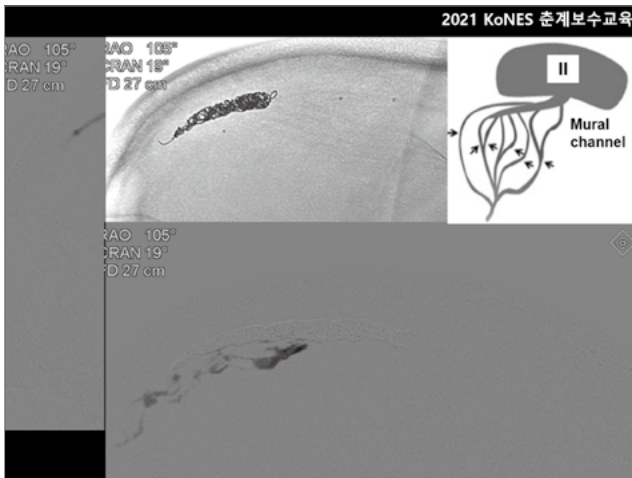
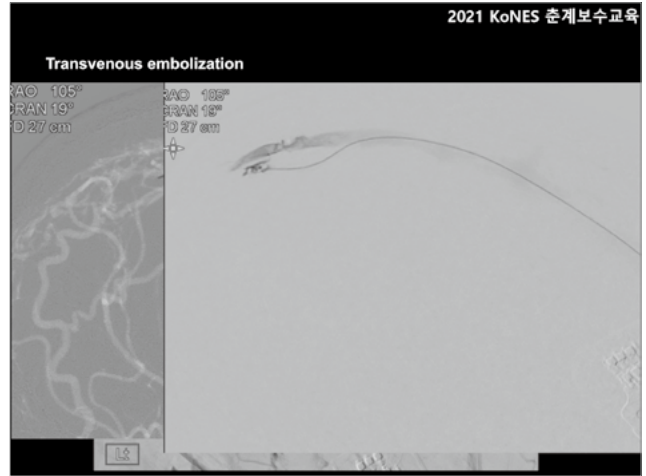
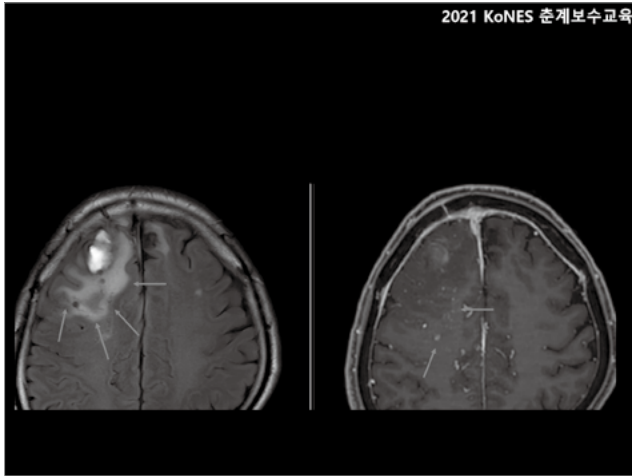
Background



G1 G2 G3 G4







2021 KoNES 춘계보수교육

Conclusions

- Understanding of pathogenesis of dAVF
- Understanding of anatomy of dAVF
- Fistulous point and Mural channel

Multiple fistula points in cavernous sinus dural arteriovenous fistula

임종국

제주대병원 신경외과

The patient visited the neurology department of our hospital with typical cavernous sinus (CS) dural arteriovenous fistula (dAVF) symptoms. Routine transfemoral cerebral angiography (TFCA) was performed and three major fistula points were presented. This CS dAVF occurred around the cavernous sinus, resulting in bilateral lesions, and the posterior compartment of CS is the main drainage sinus. Symptoms appear only on the left side with a drainage pattern. First of all, I planned a transvenous embolization through inferior petrosal sinus (IPS). Navigating through IPS was not difficult, but several trials with microwires found a main fistula point. The microcatheter reached the main lesion and a detachable coil was placed in the main hole of the lesion. Follow-up images showed almost occlusion on right external carotid angiography (ECAG). The left ECAG showed another unexpected lesion on top of the problem I was thinking of. That point was unreachable with the microcatheter and microwire, and the posterior compartment of the CS was filled with pushable coils. Final angiography showed complete occlusion of CS dAVF. The patient was satisfied with the improvement in symptoms. Paradoxical exacerbation was seen 2 days after the procedure, but all symptoms improved 1 month later. IPS was a good therapeutic route for CS dAVF, and the angio-anatomical features were thoroughly evaluated on magnetic resonance imaging and TFCA. It is too difficult to return to the initial lesion after winding the coil. We need to consider the pattern/pattern of the draining vein and the caliber/path of the feeding artery.

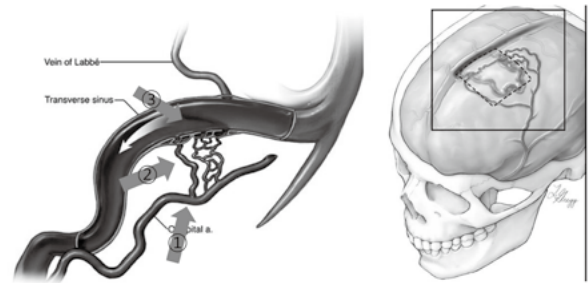
My experiences of PHIL In Dural AVFs

고정호

단국대병원 신경외과

My experiences of PHIL In Dural AVFs

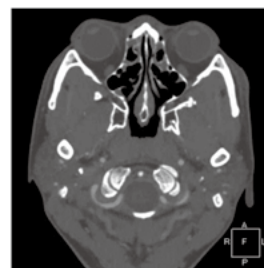
단국대학교 고정호



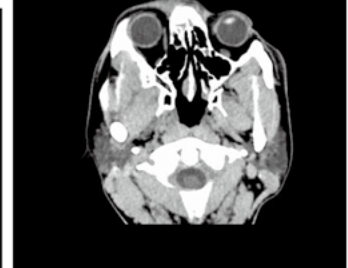
Several concerns about embolization using Phil for DAVF

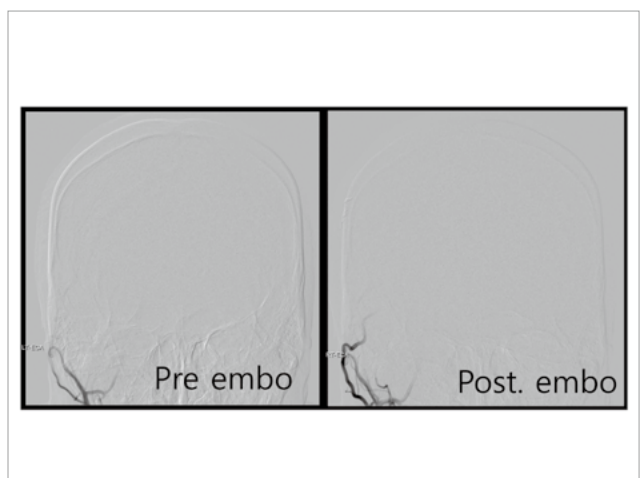
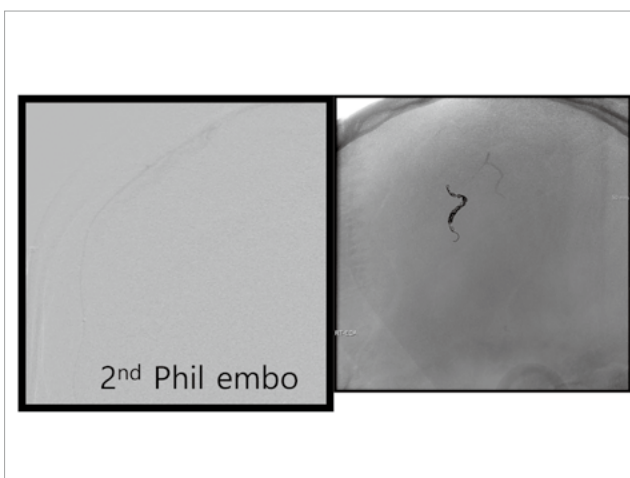
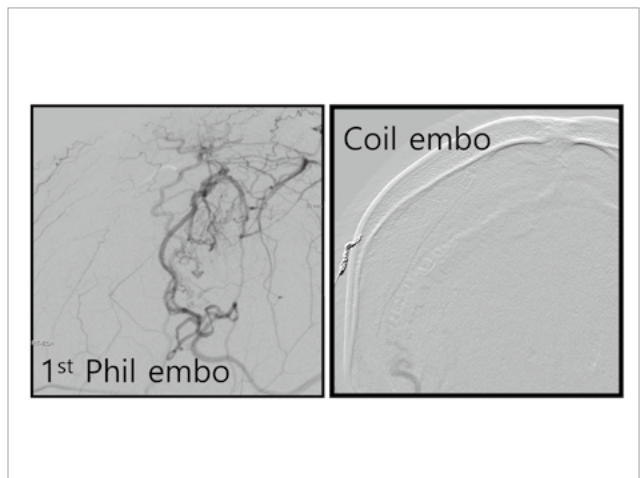
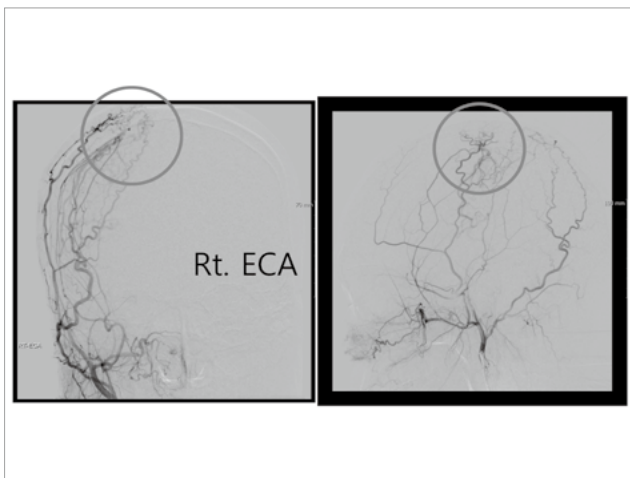
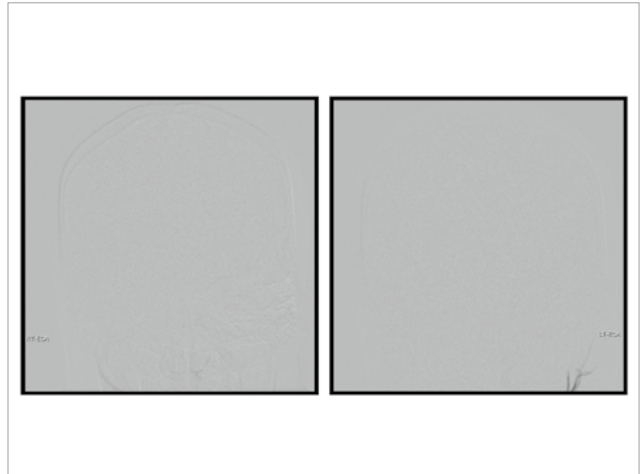
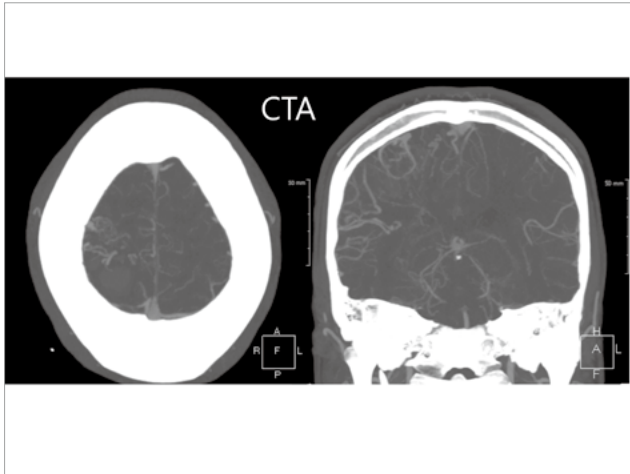
1. Venous obstruction due to poor control
2. Poor visualization
3. Poor penetration via AVF network
4. Attachment of a microcatheter on a feeder

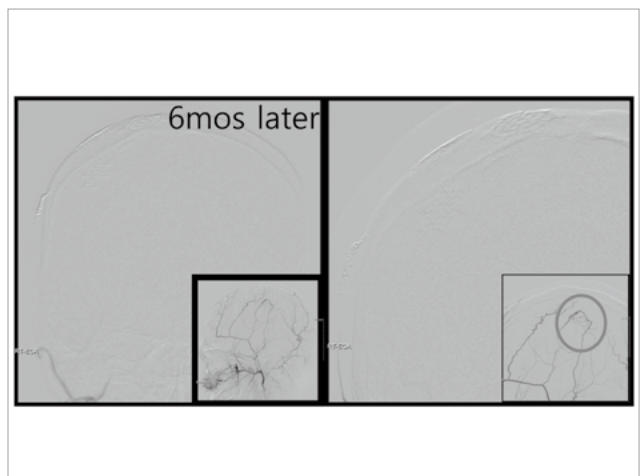
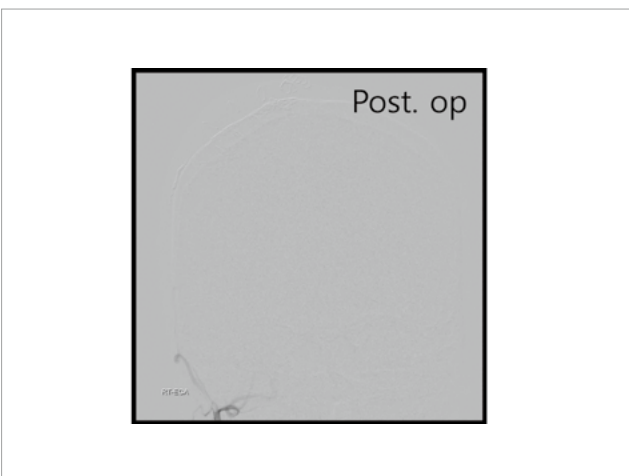
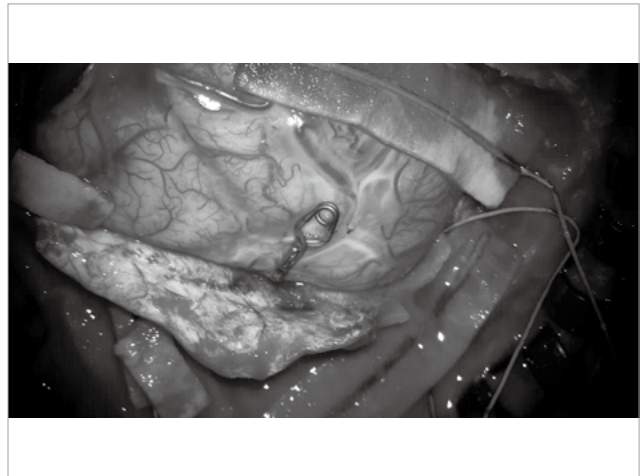
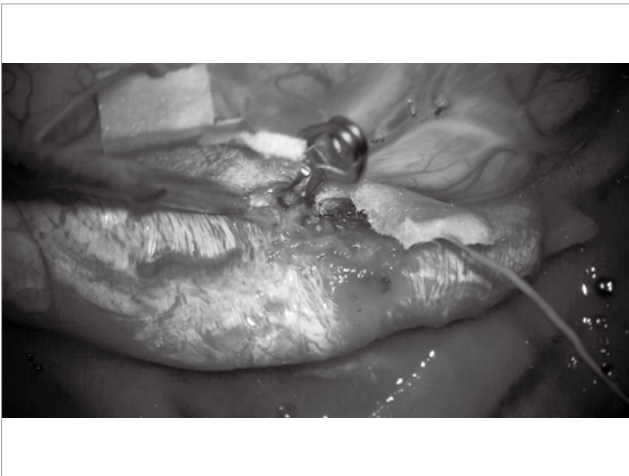
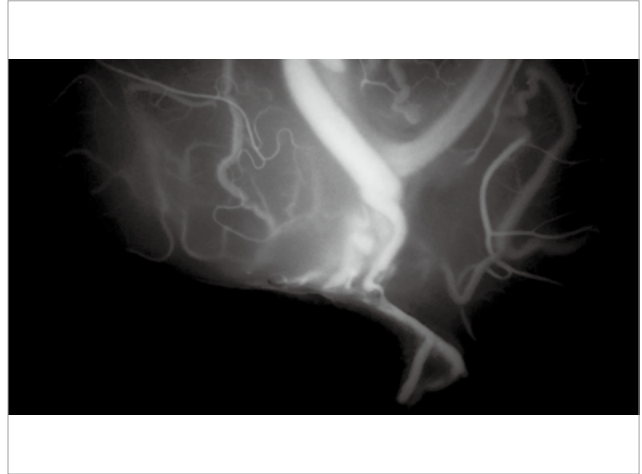
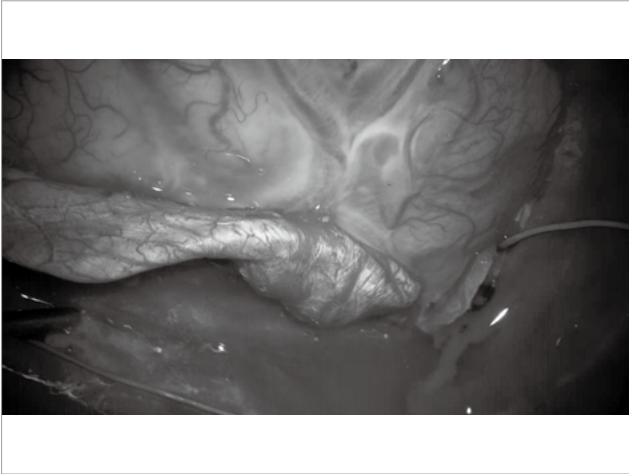
M/25 S-ICH

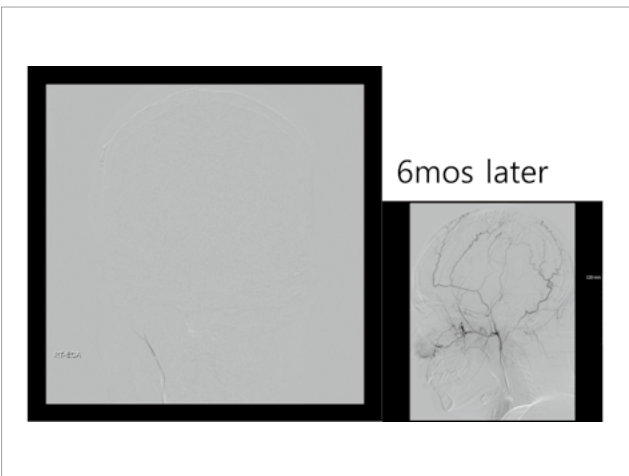
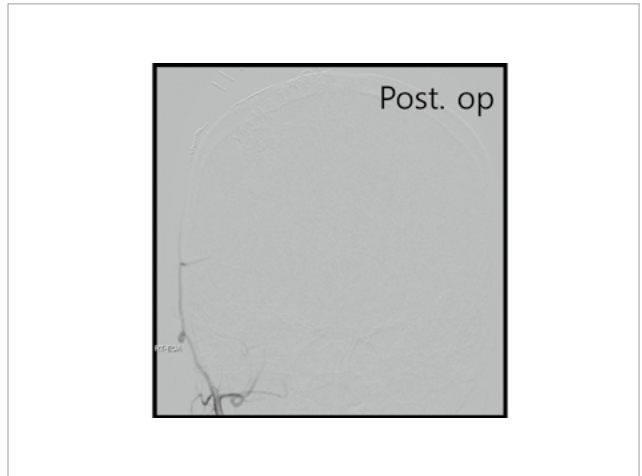
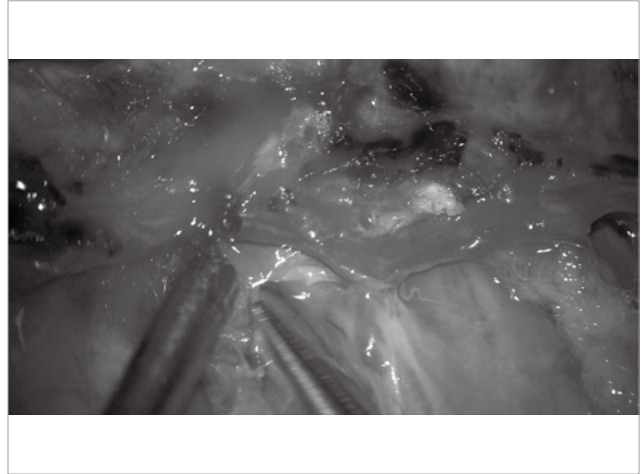


Brain CT

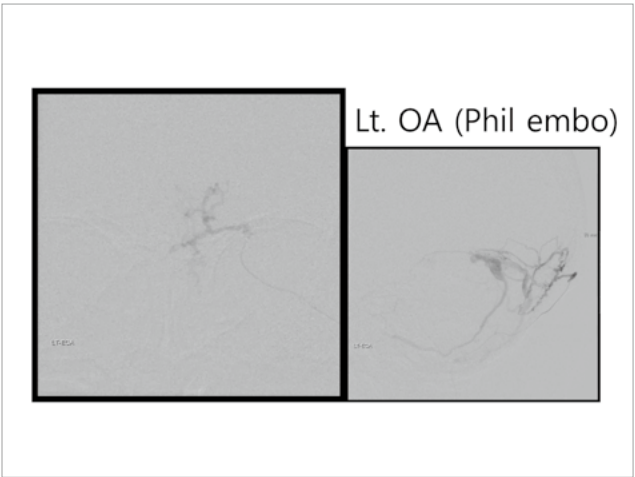
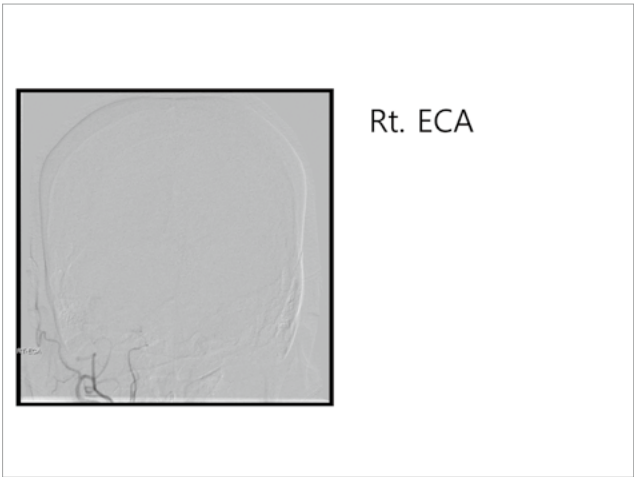
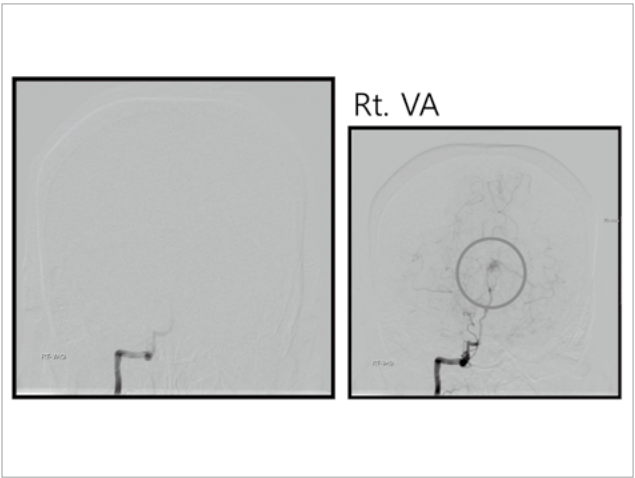
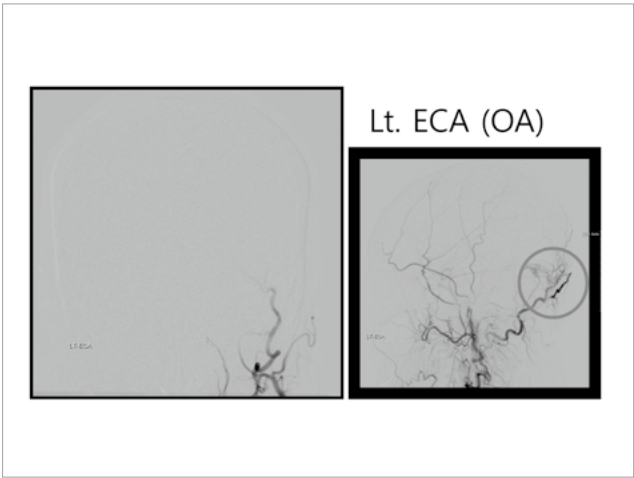
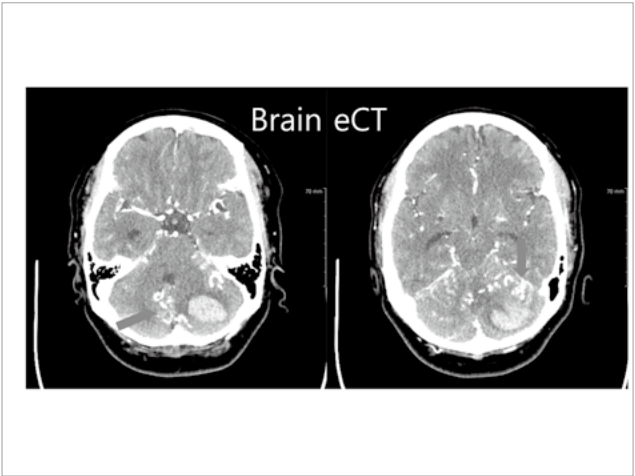
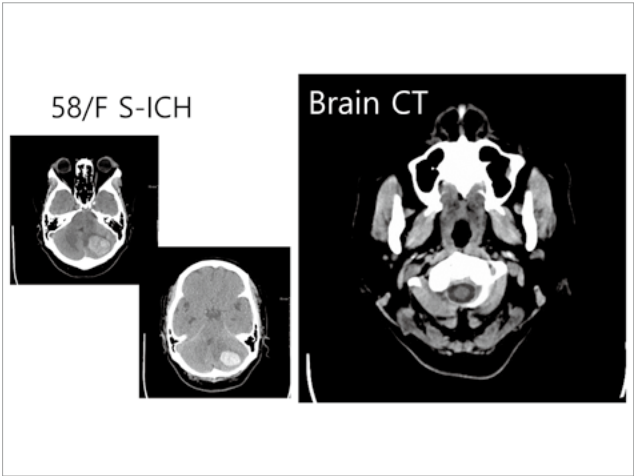


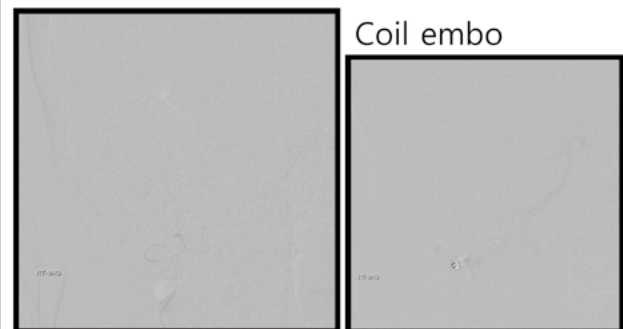
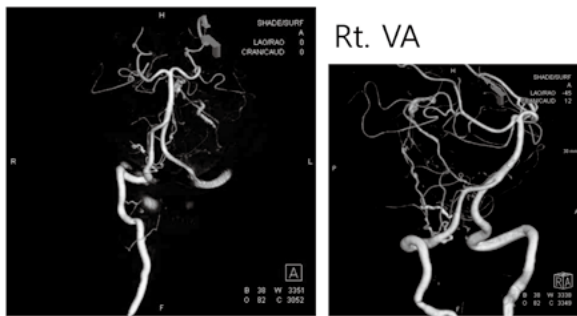
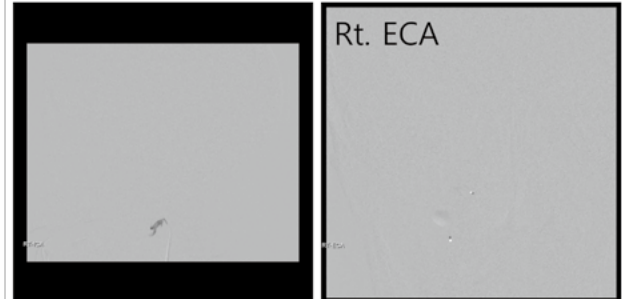
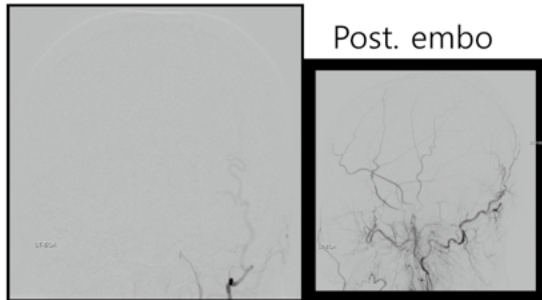






In my experience

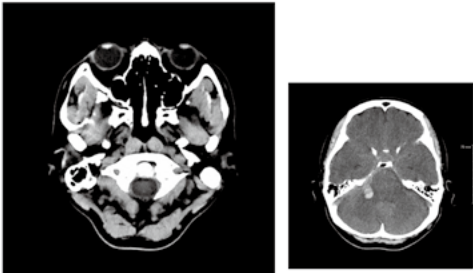




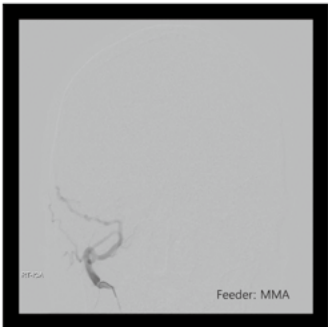
In my experience

- Phil Injection(DMSO) into the scalp vessels make a severe pain.
- So general anesthesia is needed.
- It is difficult to penetrate through skull.

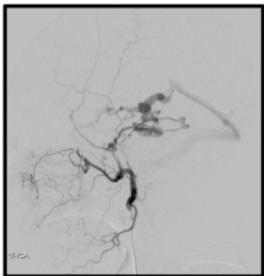
45/M S-ICH around C-P angle



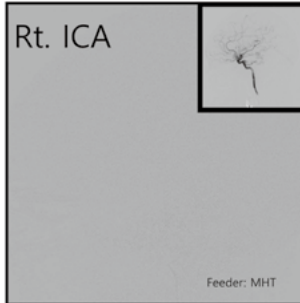
CTA



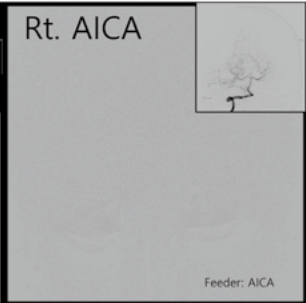
Rt. ECA



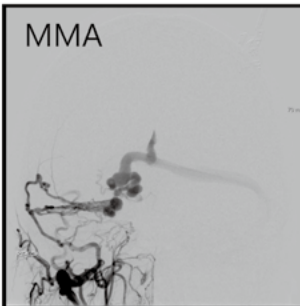
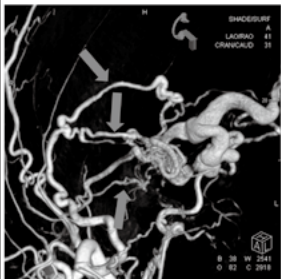
Rt. ICA



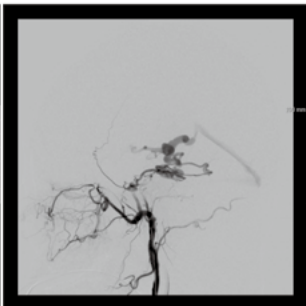
Rt. AICA

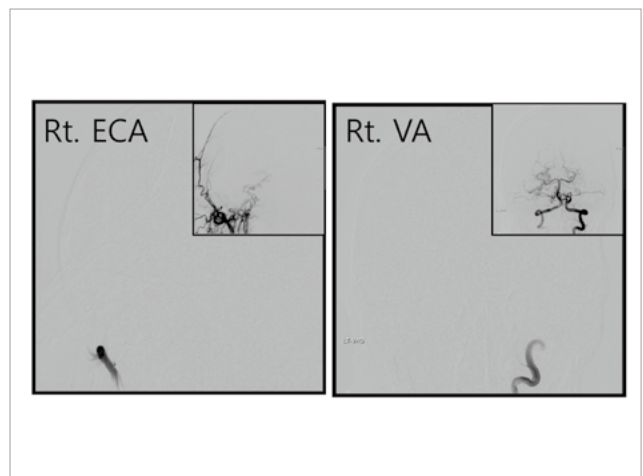
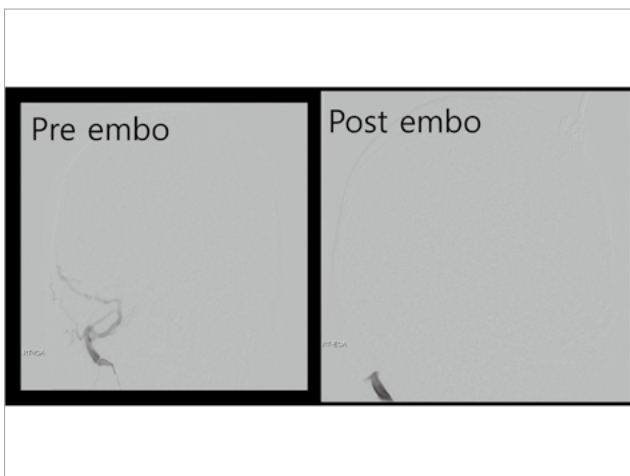
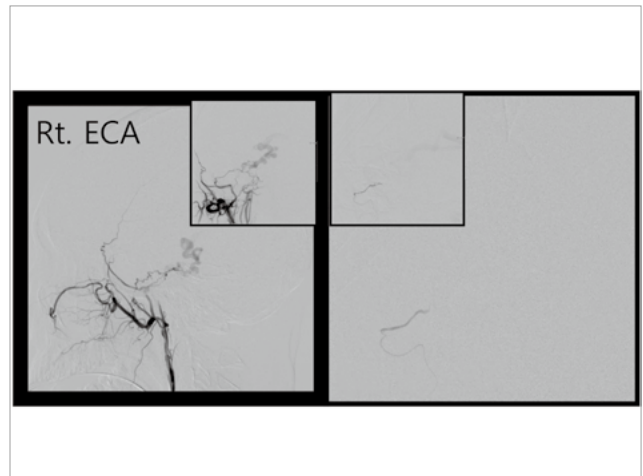
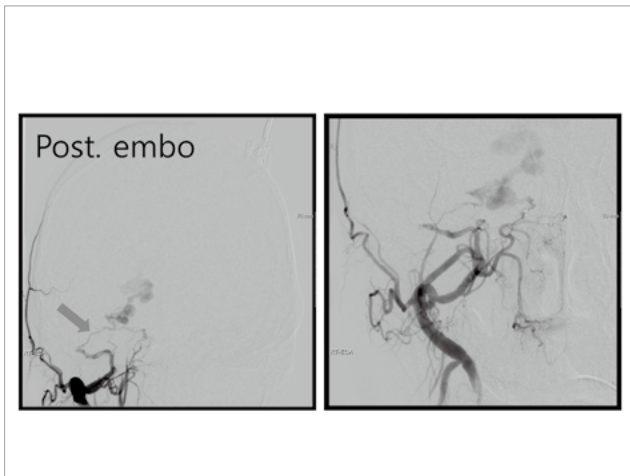
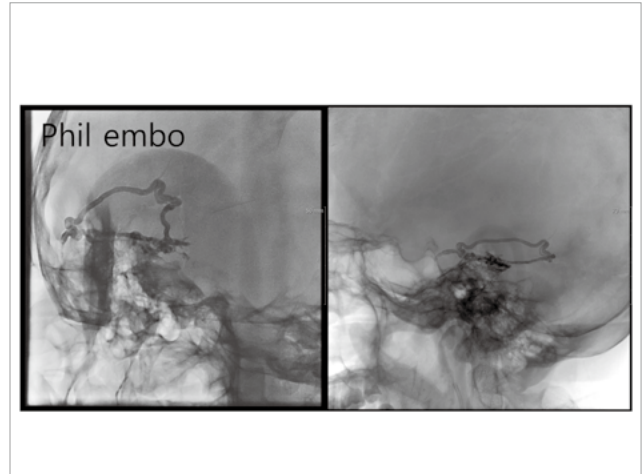
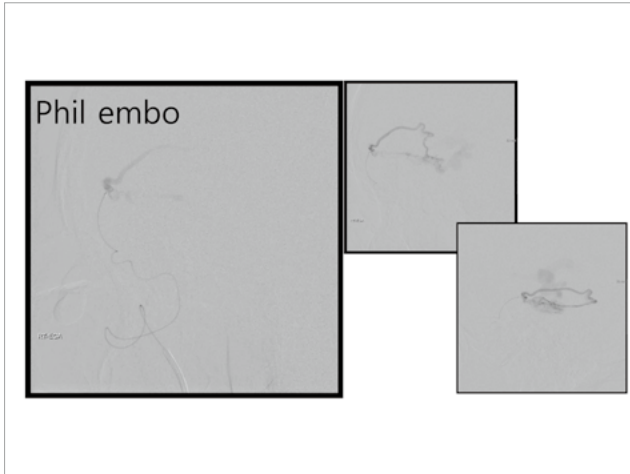


MMA



MMA





In my experience

DMSO allergy

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Adverse reactions of dimethyl sulfoxide in humans: a systematic review [version 2; peer review: 2 approved]
Benedikte Kollup Madsen ¹, Maria Hilscher, Dennis Zetner ², Jacob Rosenberg
¹Department of Surgery, Center for Peroperative Optimization (CPO), Hvidovre Hospital, Hvidovre, 2730, Denmark

Abstract

Background: Dimethyl sulfoxide (DMSO) has been used for medical treatment and as a pharmacological agent in humans since the 1960s. Today, DMSO is used mostly for cryopreservation of stem cells, treatment of interstitial cystitis, and as a penetrating vehicle for various drugs. Many adverse reactions have been described in relation to the use of DMSO, but to our knowledge, no overview of the existing literature has been made. Our aim was to conduct a systematic review describing the adverse reactions observed in humans in relation to the use of DMSO.

Methods: This systematic review was reported according to the PRISMA-Narrative (Preferred Reporting Items for Systematic reviews and Meta-Analyses) guidelines. The primary outcome was any adverse reactions occurring in humans in relation to the use of DMSO. We included all original studies that reported adverse events due to the administration of DMSO, and that had a population of five or more.

Results: We included a total of 109 studies. Gastrointestinal and skin reactions were the commonest reported adverse reactions to DMSO. Most reactions were transient without need for intervention. A relationship between the dose of DMSO given and the occurrence of adverse reactions was seen.

Conclusions: DMSO may cause a variety of adverse reactions that are mostly transient and mild. The dose of DMSO plays an important role in the occurrence of adverse reactions. DMSO seems to be safe to use in small doses.

Registration: PROSPERO CRD42018036117.

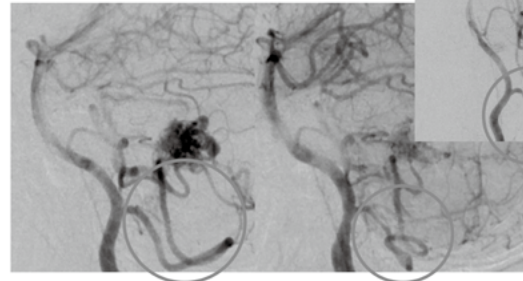
Patients characteristics

No	나이	성별	Sx	ICH	Feeder(embo)	Other treatment	complication	Outcome
1	25	M	Headache, Lt. motor weakness	O	Rt. MMA(Phil) Rt. STA(Coil) Lt. STA(OP)	Craniotomy And clipping	X	Good
2	58	F	Dizziness Headache	O	Lt. OA (Phil) Rt. VA (Coil)		X	Good
3	45	M	Headache, dizziness	O	Rt. MMA(Phil) Rt. ICA Rt. AICA	radiosurgery	X	Good

Several concerns about embolization using Phil for DAVF

1. Venous obstruction due to poor control
2. Poor visualization
3. Poor penetration via AVF network
4. Attachment of a microcatheter on a feeder

Catheter attachment



DMSO COMPATIBLE MICROCATHERETERS

Microcatheter	MicroVenton	Medtronic	Balt	Stryker
Detachable Tip Catheter		Apollo™	Sonic	
Dual Lumen Balloon	Scepter™C Scepter™ XC		Eclipse 2L, Copernic 2L	
Wire Directed Catheter	Headway® Headway® DUO	Marathon™ Echelon™ Rabus™ UltraFlow™		Excelsior™ XT-17

Thanks for your attention

Case Presentation of dAVF

신희섭

강동경희대병원 신경외과



Case Presentation of dAVF

경희대학교 의과대학 신경외과학교실
 강동경희대학교병원 신경외과
 신 희 섭

2021 대한뇌혈관내치료의학회
 춘계보수교육



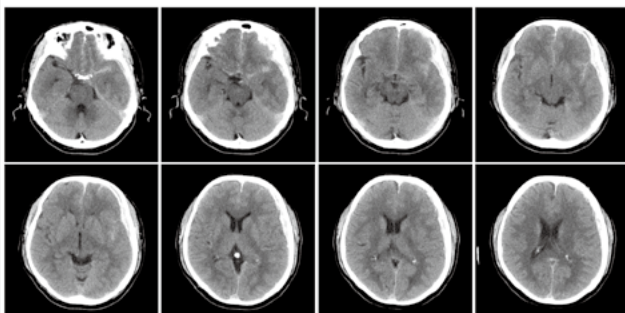
61/M

갑작스럽게 발생한 두통, 구역, 구토
 신경과 입원 후, CT 참고 refer

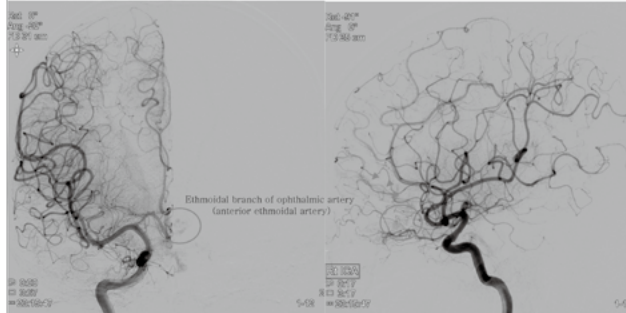
temporal lobe seizure: 10년 전부터 신경과 외래 t/u
 음주: 1주에 4일 정도
 운동: 자전거

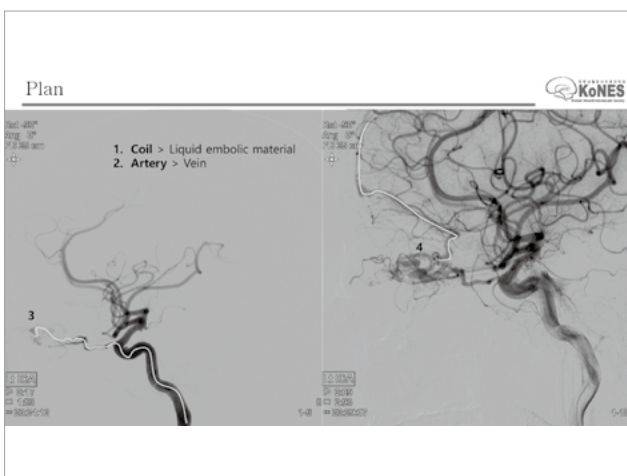
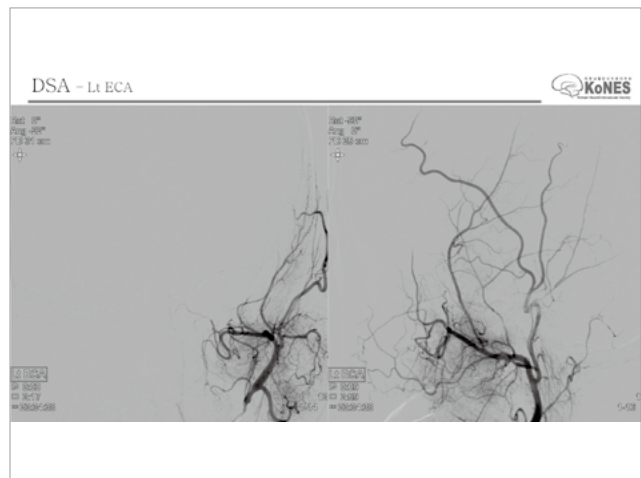
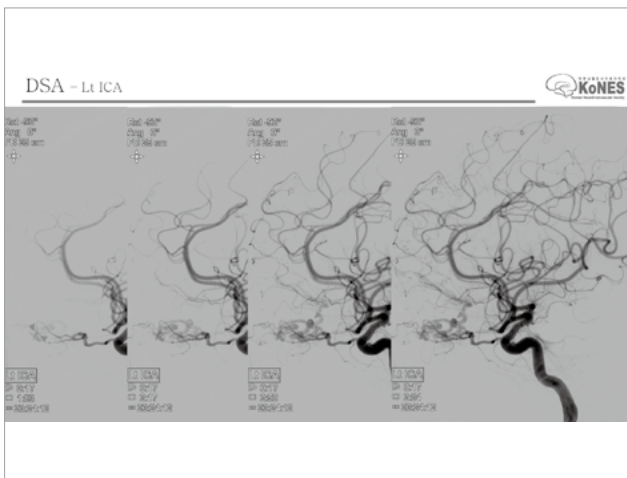
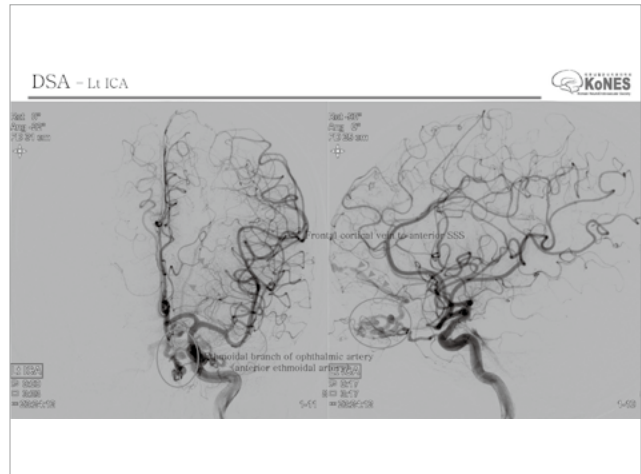
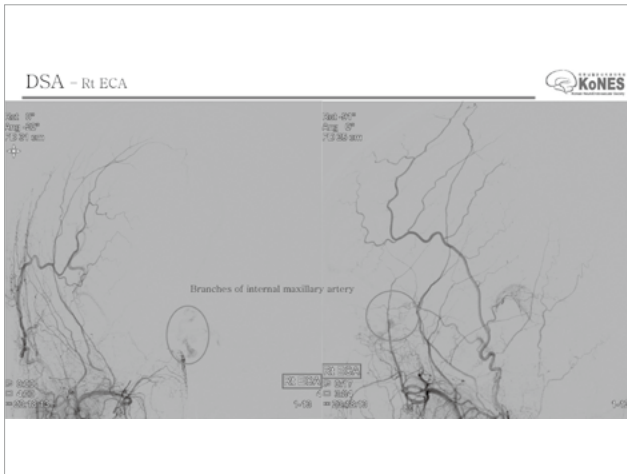
2021 대한뇌혈관내치료의학회
 춘계보수교육

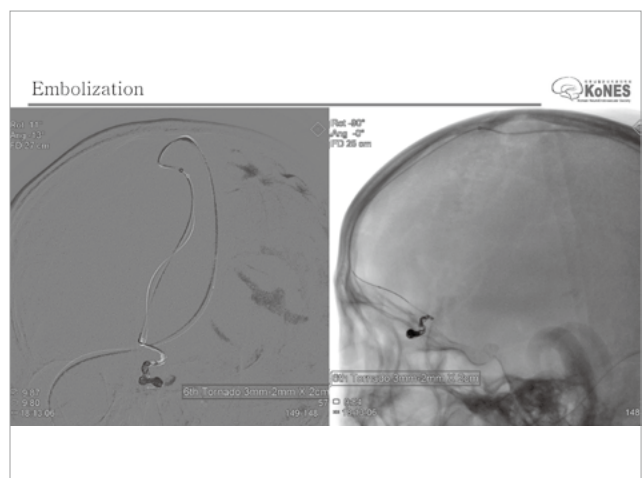
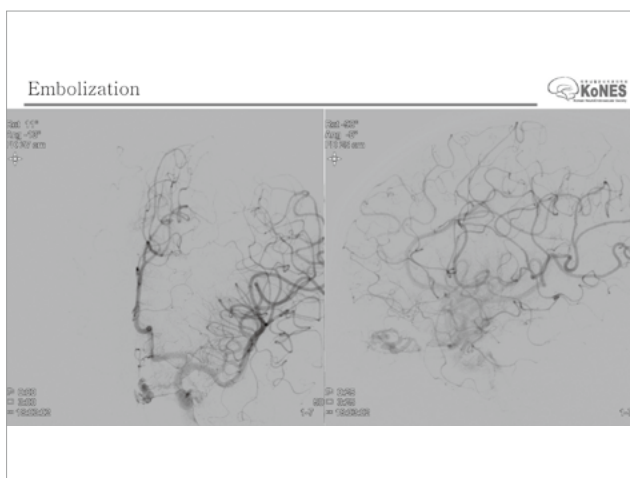
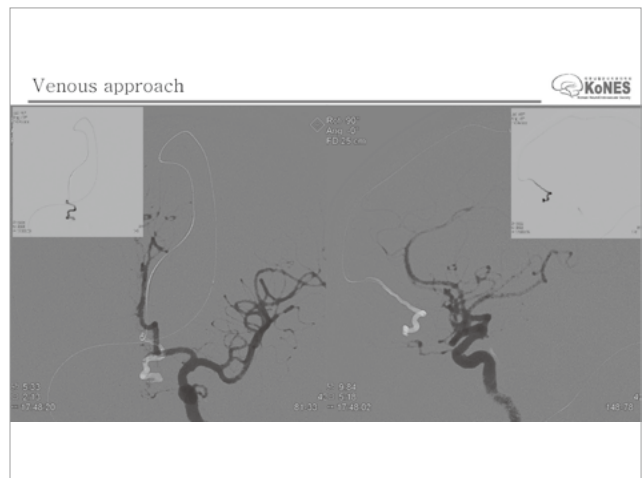
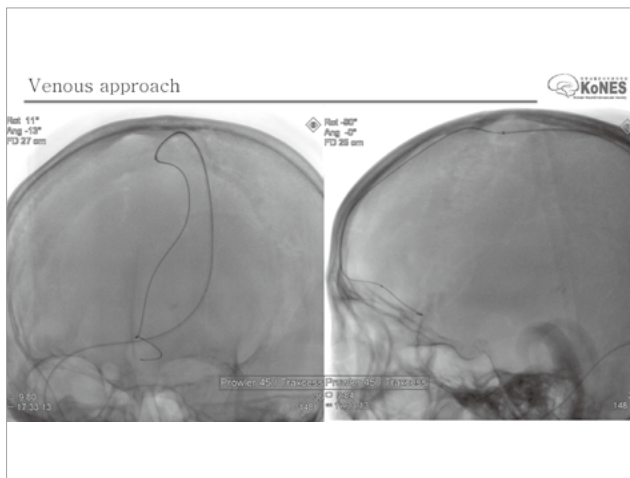
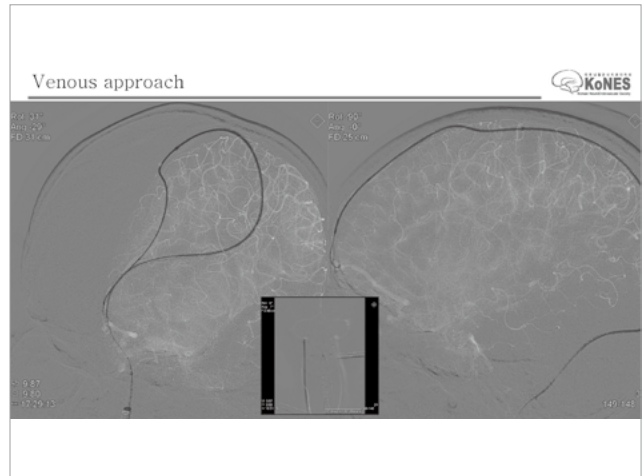
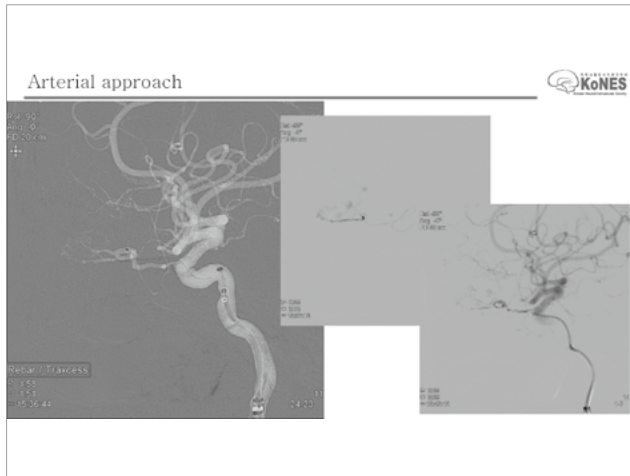
Brain CT

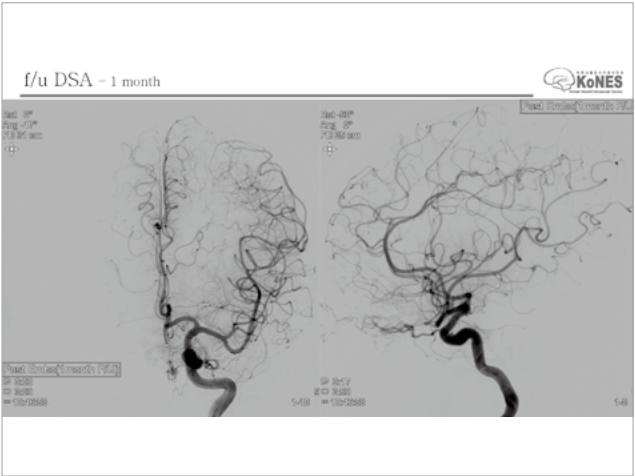
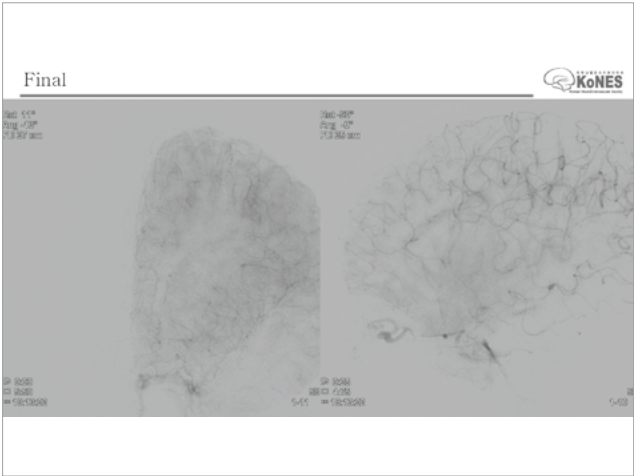


DSA - Rt ICA









2021 대한뇌혈관내치료의학회 춘계보수교육

인 쇄 2021년 2월 25일

발 행 2021년 2월 27일

발 행 처 대한뇌혈관내치료의학회

회 장 윤 석 만

총무이사 박 석 규

수련교육이사 김태곤, 유승훈

주 소 서울시 서초구 서초대로 350 (서초동 동아빌라트 2타운) 407호

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제 품 명 Q-Guard / 큐가드
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가 격 법정비급여
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2 x 2
Hemostatic Dressing



4 x 4
Hemostatic Dressing



Z-Fold
Hemostatic Dressing



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Hemostatic Dressing



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(Choline alfoscerate)

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알포아티린®리드캡슐(클린알포세레이트)

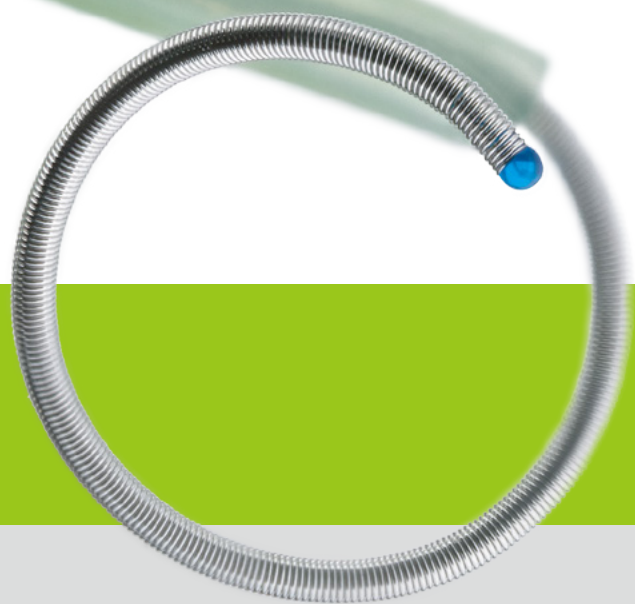
【원료약품 및 분량】 1캡슐 중 클린알포세레이트(KP) 400mg **【성상】** 무색 투명한 액체가 충전된 빨간색의 띠로 감싸진 산하부 무색 투명한 캡슐체 **【효능·효과】** - 뇌혈관 결손에 의한 2차 중상 및 변성 또는 퇴행성 뇌기질성 정신증후군: 기억력저하와 착란, 의욕 및 자발성저하로 인한 방향감각장애, 의욕 및 자발성 저하, 집중력감소 - 감정 및 행동변화: 정서불안, 자각과민성, 주위무관심 - 노인성 가성우울증 **【용법·용량】** 클린알포세레이트로서 1회 400 mg을 1일 2~3회 경구투여한다. 중상에 따라 적절히 증감한다. **【사용상의 주의사항】** 1. 금기 1) 이 약 및 이 약의 구성성분에 과민반응 환자 2) 임부 또는 임신하고 있을 가능성이 있는 여성 **【저장방법】** 기밀용기, 실온(1~30℃)보관 **【포장단위】** 30캡슐/병, 60캡슐/병 **【첨부문서개장일】** 2018.10.2 **【제조·의뢰·판매자】** (주)유한양행 서울시 동작구 노량진로 74 www.yuhan.co.kr 소비자상담실: 080-024-1188(수신자 요금부담) ※ 보다 자세한 사항은 회사의 제품설명서 전문을 참고하시기 바랍니다.

알포아티린®정(클린알포세레이트)

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【원료약품 및 분량】 1캡슐 중 클린알포세레이트(KP) 400mg **【성상】** 무색투명한 액을 넣은 담황색을 띤 타원형의 연질캡슐체 **【효능·효과】** - 뇌혈관 결손에 의한 2차 중상 및 변성 또는 퇴행성 뇌기질성 정신증후군: 기억력저하와 착란, 의욕 및 자발성저하로 인한 방향감각장애, 의욕 및 자발성 저하, 집중력감소 - 감정 및 행동변화: 정서불안, 자각과민성, 주위무관심 - 노인성 가성우울증 **【용법·용량】** 클린알포세레이트로서 1회 400 mg을 1일 2~3회 경구투여한다. 중상에 따라 적절히 증감한다. **【사용상의 주의사항】** 1. 금기 1) 이 약 및 이 약의 구성성분에 과민반응 환자 2) 임부 또는 임신하고 있을 가능성이 있는 여성 **【저장방법】** 기밀용기, 실온(1~30℃)보관 **【포장단위】** 100캡슐(10캡슐/PTPx10) **【첨부문서개장일】** 2017.10.10 **【제조·의뢰·판매자】** (주)유한양행 서울시 동작구 노량진로 74 www.yuhan.co.kr 소비자상담실: 080-024-1188(수신자 요금부담) ※ 보다 자세한 사항은 회사의 제품설명서 전문을 참고하시기 바랍니다.

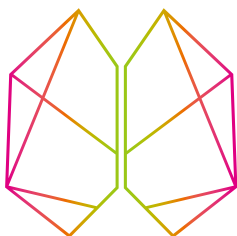


- Random Complex Shape Technology
- Low Coil Profile — 0.009" Primary Wind
- Complete Product Offering

ORDERING INFORMATION

GALAXY G3 Mini Microcoil

Catalog Number	Secondary Diameter (mm)	Coil Length (cm)
GLM910010	1	1
GLM910015	1	1.5
GLM910020	1	2
GLM910025	1	2.5
GLM910030	1	3
GLM910040	1	4
GLM915020	1.5	2
GLM915025	1.5	2.5
GLM915030	1.5	3
GLM915040	1.5	4
GLM920030	2	3
GLM920040	2	4
GLM920060	2	6
GLM925035	2.5	3.5
GLM925045	2.5	4.5
GLM925055	2.5	5.5
GLM930040	3	4
GLM930060	3	6



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Perclose ProGlide™

Suture-Mediated Closure System



**10 MILLION+
REPAIRS†**

**DON'T
JUST CLOSE.
REPAIR.**



†January 2020 Finance Report. Data on file at Abbott

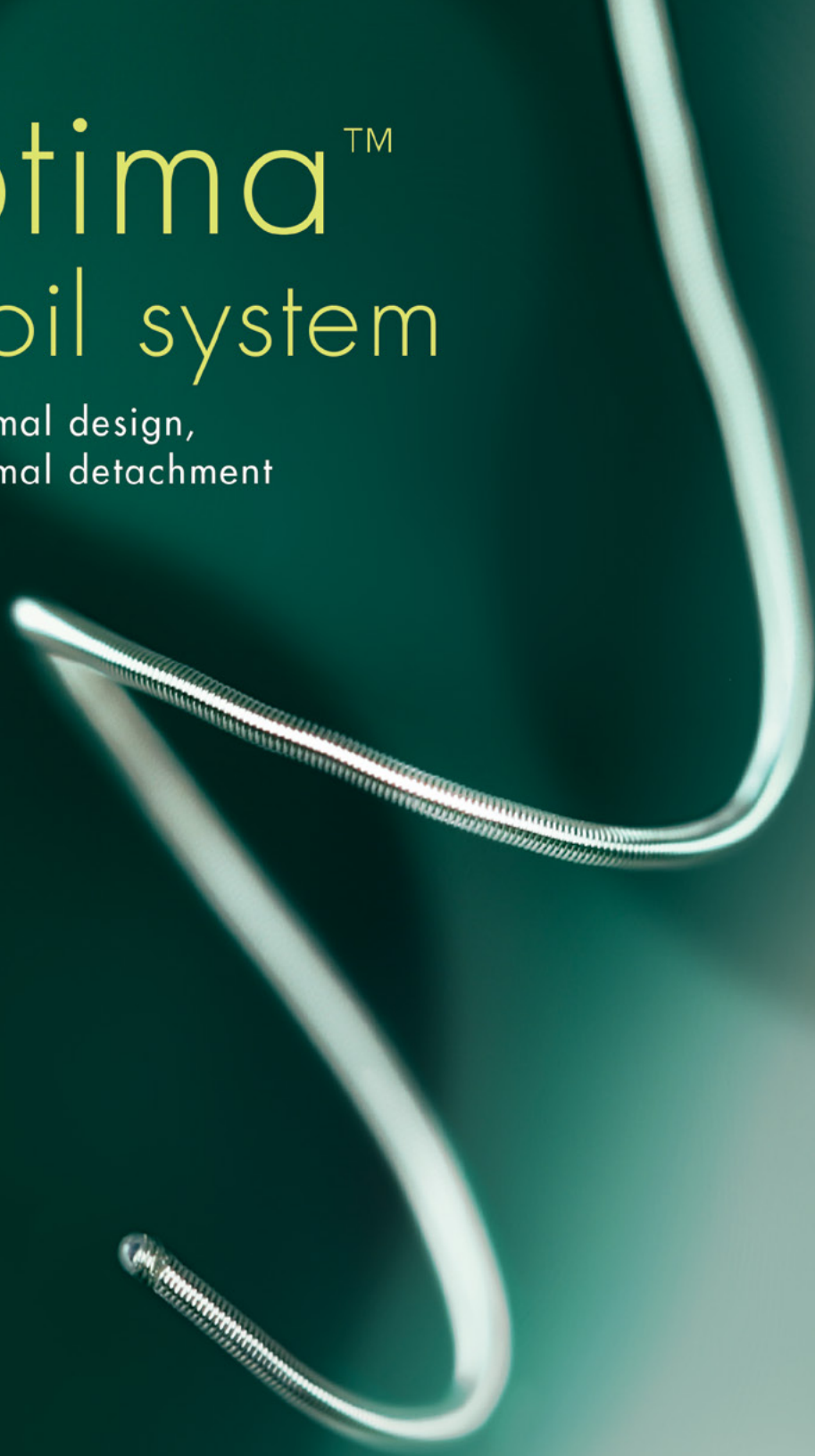
*On Nov. 8, 1993, the first (Perclose) patent was filed for the percutaneous suture vascular closure device. The Perclose portfolio includes all percutaneous suture closure devices. Data on file at Abbott.

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optima™ coil system

optimal design,
optimal detachment



Optima Coil System is manufactured by Balt USA, 29 Parker, Irvine, CA 92618. The Optima Coil System is intended for the endovascular embolization of intracranial aneurysms and other neurovascular abnormalities such as arteriovenous malformations and arteriovenous fistulae. The Optima Coil System is also intended for vascular occlusion of blood vessels within the neurovascular system to permanently obstruct blood flow to an aneurysm or other vascular malformation and for arterial and venous embolizations in the peripheral vasculature. The content of this document, in particular data, information, trademarks and logos are BALT S.A.S and affiliates' sole property. Consequently, all representation and/or reproduction, whether in part or in full, is forbidden and would be considered a violation of BALT S.A.S and affiliates' copyrights and other intellectual proprietary rights ©2018 BALT S.A.S and affiliates all rights reserved. This document with associated pictures are non-contractual and are solely dedicated to healthcare professionals and BALT S.A.S and affiliates' distributors. The products commercialized by BALT S.A.S and affiliates shall exclusively be used in accordance with the package inserts which have been updated and included in the boxes. Optima Coil System is Class III CE marked (DQS CE0297) according to the Medical Device Directive 93/42/EEC Annex II Section 4 since July 2017 (535003 W). (05/2018).





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#Neurorecovery

#Motor and cognitive improvement

뉴로리진주

(돼지뇌펩티드)

Cerebroprotein hydrolysaate 10 mL / 20 mL



뉴로리진주 10 mL, 20 mL

【효능·효과】 알츠하이머형 노인성치매, 뇌졸중 후 뇌기능장애, 두개골의 외상(뇌진탕, 뇌좌상, 수술 후 외상) 【용법·용량】 일반적으로 하기 환자의 1일 투여용량은 다음과 같다. ·알츠하이머형 노인성치매, 뇌졸중 후 뇌기능장애 : 5~20mL · 두개골의 외상(뇌진탕, 뇌좌상, 수술 후 외상) : 10~50mL · 소아 : 1~2mL 투여기간은 매일 투여로 10~20일이나, 통상 계속 투여해도 더 이상 진전이 없을 때까지 투여하며, 투여 횟수를 매일 투여에서 주 2~3회 투여로 변경할 수 있다. 각 치료주기 사이에 치료기간만큼의 휴약기를 갖는다. 단, 투여량, 투여횟수, 투여기간은 연령 및 증상에 따라 적절히 증감할 수 있다. 5mL까지는 근육주사, 10mL까지는 정맥주사하고, 10mL에서 최고 30mL까지는 0.9%생리식염주사액, 링겔액, 5%포도당 주사액, 또는 덱스트란 40에 무균적으로 혼합하여 약 60분간에 걸쳐서 천천히 정맥 주입하며 필요할 경우 심혈관계 작용약 또는 비타민제와 동시투여할 수 있으나 용액에 직접 혼합해서는 안 된다. 【이상반응】 1) 과민반응 : 극히 드물게 알레르기 반응(피부반응, 국소혈관반응, 두통, 목의 통증, 사지통증, 발열, 하부요통, 호흡곤란, 오한, 속 유사 증상)이 관찰되었다. 2) 정신신경계 : 드물게 심리적 불안감(공격적, 혼란, 불면증)이 나타날 수 있다. 3) 소화기계 : 드물게 식욕감퇴, 소화불량, 설사, 변비, 구역, 구토 등이 나타날 수 있다. 4) 투여부위 : 피부발적, 가려움증, 작열감이 보고되었다. 5) 기타 : 드물게 과다호흡, 과다호흡긴장증, 근육긴장저하, 피로, 떨림, 우울증, 냉담, 어지러움, 의식의 혼미, 인플루엔자의 증상(예 오한, 기침, 호흡기 감염) 등이 보고되었으며, 대발작과 경련이 단 한건 보고되었다.