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Review Article

Cavernous Sinus Dural Arteriovenous Fistulas: General Aspects

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Cavernous sinus (CS) dural arteriovenous fistulas (AVF), which are most common in middle-aged females, present with benign symptoms such as exophthalmos, chemosis, and orbital bruit. Benign CS dural AVF without cortical venous drainage (CVD) have the rare potential for development of CVD with neurological symptoms, even without treatment. On the other hand, aggressive type AVF with CVD can cause more aggressive symptoms such as cerebral hemorrhage. As symptoms are highly related to the drainage pattern, it is important to understand the anatomy of the CS itself, shunt point, and draining vein when treating the lesion. In general, the drainage route is gradually diminished by thrombosis and compartmentalization within the CS according to progression of the angiographical stage. At the restrictive stage, the disease is usually treated by endovascular treatment, particularly transvenous embolization.

Keywords ▶ cavernous sinus, dural arteriovenous fistula, general aspects, natural history

Introduction

Cavernous sinus (CS) dural arteriovenous fistulas (AVF) are the most common intracranial dural AVF in Japan.[1,2] Postmenopausal females more commonly develop CS dural AVF, suggesting hormonal influence.[3] Clinical symptoms are generally benign because the CS has sufficient venous drainage routes such as the superior ophthalmic vein (SOV), inferior petrosal sinus (IPS), superior petrosal sinus (SPS), superficial middle cerebral vein (SMCV), and the intercavernous sinus connecting to the opposite side of the CS. However, in some cases, aggressive features are expressed when some extracranial drainage routes are occluded.

In this article, the etiology, epidemiology, natural history, anatomy, diagnosis, and classification of CS dural AVF based on recent studies, respectively, are reviewed.

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Etiology of CS Dural AVF

Dural AVFs are generally thought to be acquired lesion except for rare cases of congenital dural AVFs in infant. Several factors can be associated with development of dural AVFs. Houser and Chaudhary et al.[4,5] suggested that thrombosis of the dural sinus and/or vein is a potential cause in formation of sural AVFs. Experimentally, Terada et al.[6] reported in 1994 that dural AVF was developed in venous hypertensive rats. Factors that trigger the formation of dural AVF include trauma, abnormal hormone balance, sinus thrombosis, abnormal coagulation, venous hypertension, and so on. Especially in CS dural AVF, since it is apparently common in postmenopausal women, estrogen, and eventually sinus thrombosis associated with the imbalance of estrogen is considered as a trigger.[3,7] In histological examination of dural AVF, it was confirmed that angiogenic factors such as basic fibroblast growth factor and vascular endothelial growth factor were expressed.[8,9] However, such angiogenic factors generally amplify capillary vessel rather than large blood vessels with a diameter of about 200 μm which constitutes dural AVF. Recent studies have suggested the involvement of transforming growth factor-β and its receptor-like activin receptor-like kinase type 1, in the pathogenesis of dural AVF.[10] Therefore, multiple factors appear to involve the formation of dural AVF, and further research will be awaited.

Epidemiology of CS Dural AVF

Japanese nationwide survey

A specialist-based nationwide questionnaire survey about the treatment of dural AVF was conducted in 2007.[1] The survey data were collected from 388 specialists (268 facilities) certified in 2006 by the Society of Neuroendovascular Therapy. The assessed diseases were intracranial and spinal dural AVF, which were newly treated between January 2005 and December 2006.

In all, 396 cases of CS dural AVF were included in the study. The average age was 66.8 ± 11.0 years, and 76% cases were in women. Regarding the location of dural AVF, the CS was the most dominant (45.9%), followed by the transverse-sigmoid sinus (TS-SS) (26.7%). In Western countries, TS-SS dural AVF are more common than CS lesions. Therefore, incidence rate may depend on race. The average age was as high as 66.8 years for CS dural AVF and as low as 55.4 years for the cranial vault dural AVF.

JRNET 2

In the Japanese Registry of Neuroendovascular Therapy (JR-NET) 2 database, 1520 attempted or completed endovascular procedures for dural AVF in 1075 patients were recorded between January 2007 and December 2009.[2] In this series, 469 patients (43.6%) had CS dural AVF. Similar to the previous report, the CS (43.6%) was reported as the most frequent location, followed by the TS-SS (33.4%).

JRNET 3

The JRNET 3 database was created retrospectively from a multicenter observational study between January 2010 and December 2014.[11] Hiramatsu et al. analyzed 1940 attempted or completed procedures in 1458 patients with cranial dural AVF.[11] One-third of the dural AVF was located in the TS-SS (40%), followed by the CS (34%), tentorium (5%), condylar marginal sinus (CMS; 4%), superior sagittal sinus (SSS; 3%), cranio-cervical junction (CCJ; 2%), anterior cranial fossa (ACF; 2.5%), and SPS (0.5%).

They analyzed complication details and risk factors in this study. Regarding the complication rate of the overall procedure for CS dural AVF, the mortality rate was 0.5% (3 patients) and morbidity rate was 3.1% (18 patients). The overall complication rate was 8.7% (5 patients).

Natural History of CS Dural AVF

Natural history of benign CS dural AVF

In 2002, Satomi et al. reviewed clinical and angiographical data of cranial dural AVF without cortical venous drainage (CVD) to evaluate the behavior of these shunts and to assess the risk of aggressive conversion during conservative management.[12]

In their study, 117 patients with cranial dural AVF were categorized as having benign lesions based on the absence of CVD (Borden Type 1/Cognard Type I or IIa). Benign CS dural AVF were observed in 50 (42.7%) patients in their series. Among these 50, angiography revealed aggressive conversion into a lesion with CVD in only one conservatively managed patient during a median follow-up period of 27.9 months (range: 1 month–17.5 years). This conversion was related to symptomatic aggravation. This study demonstrated that benign CS dural AVF has a 2% (1/50 cases) risk of angiographically aggressive conversion.

Natural history of aggressive CS dural AVF

Van Dijk et al. reported the natural history of aggressive (Borden 2 and 3) cranial dural AVF in 2002. In their series, 14 non-treated patients (11.9%) and 6 partially treated patients were observed clinically and angiographically over time.[13]

These 20 patients with aggressive dural AVF with CVR had a poor prognosis. The annual mortality rate was 10.4% and the annual incidence of serious adverse events due to central nervous system disorder was 15%. This study included three CS lesions. In 1 patient without treatment, the fistula disappeared spontaneously after 14.5 years of follow-up. On the other hand, 1 patient with partial TAE died and another without treatment developed moderate disability during follow-up. The cause of these 2 adverse events was not identified from their report.

Similarly, Söderman et al. reported the natural history of 85 lesions with CVD. Five CS dural AVF were included in this study, among which four were classified as Borden 2 and 1 as Borden 3.[14] The annual hemorrhage rate was 7.4% in cases of hemorrhage-onset and 1.5% in cases of non-hemorrhage-onset. However, location-specific study was not performed; therefore, the clinical details of these 5 CS dural AVF with CVD are unknown.

Angiographical change in CS dural AVF

Satomi et al. analyzed 65 cases of CS dural AVF angiographically between 1974 and 2003.[15] The study consisted

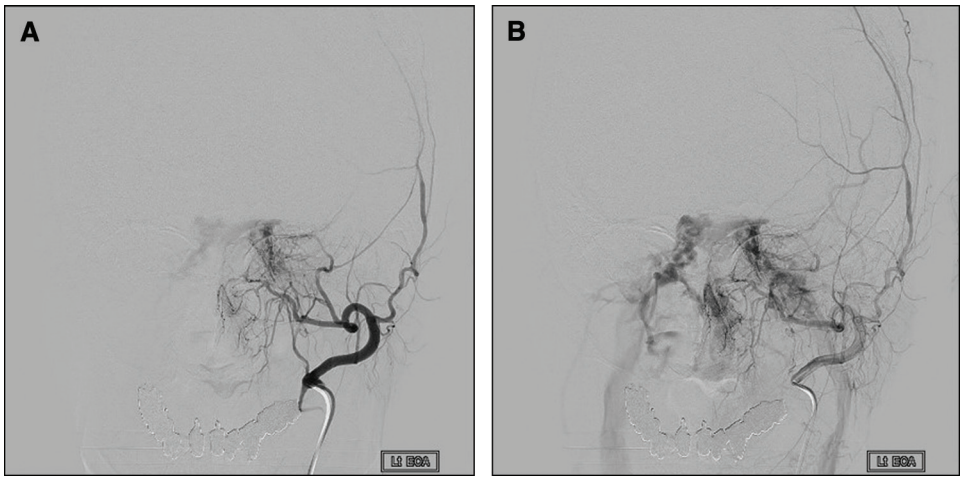

Fig. 1 (**A** and **B**) Left external carotid angiography (AP view, **A**: early phase, **B**: late phase). Representative CS dural AVF in the early stage such as type 1 and proliferative type. Angiography shows the cavernous dural AVF fed from the left external carotid arteries and drained to the bilateral IPS. Each arterial feeder converging to the wall of the dilated CS cannot be delineated. AVF: arteriovenous fistulas; CS: cavernous sinus

of 51 women and 14 men, and the mean age was 65.5 years (range: 31–85 year). CS dural AVF were divided into three groups. Type 1 dural AVF had patent anterior and posterior drainage routes. In type 2 lesions, the posterior route was closed, but the anterior drainage route was still open. In type 3 lesions, posterior and anterior drainage routes were both closed regardless of CVD (**Figs. 1** and **2**).

During the follow-up, 17 CS dural AVF, which were treated by transarterial embolization (TAE) (n = 11) or observation (n = 6), demonstrated angiographical change. In 11 of 17 cases, angiographical change was recognized in the venous drainage pattern. Type 1 to 2 conversion was observed in 5 cases, type 2 to 3 in 3, and type 1 to 3 in 3. One of 11 cases exhibited aggressive conversion with CVD. The remaining 6 CS AVF (4 cases with observation, 2 with TAE) disappeared. In 5 of these 6 cases, the affected CS was not visualized on follow-up angiography.

Another study was reported by Suh et al. in 2005.[16] They retrospectively analyzed 58 patients with CS dural AVF by reviewing their angiographical findings and medical records during follow-up (23 month [mean]). They classified dural AVF into three groups. The proliferative type (PT) exhibited numerous arterial feeders to the CS. The restrictive type (RT) had multiple feeding arteries, but fewer than those in the PT. The late restrictive type (LRT) had only a few arteries with sluggish retrograde venous flow (**Figs. 1** and **2**).

In this series, out of 58 patients with the PT (n = 23), RT (n = 23), or LRT (n = 12), they were able to perform 16 follow-up angiograms for 11 patients during 1 month to 6 years. In 7 of 11 patients, the change from PT to RT

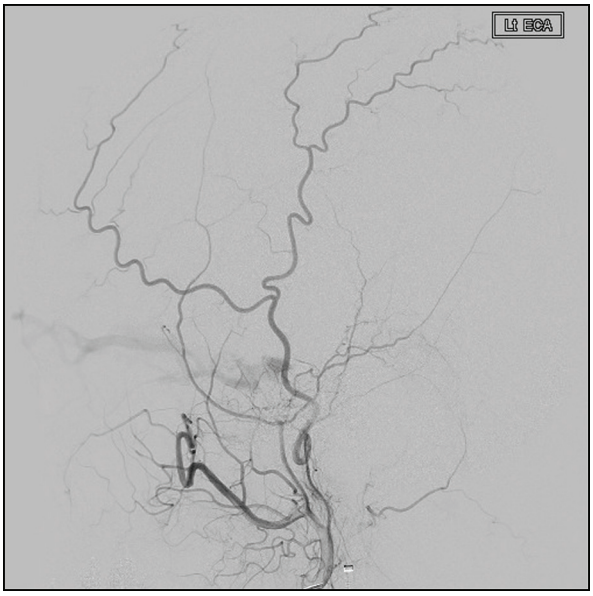

Fig. 2 Left external carotid angiography (lateral view). Representative CS AVF in the late stage such as type 2 and the late restrictive type. Angiography shows the CS dural AVF fed from the left external carotid arteries and drained slowly to the left superior ophthalmic vein only. There is no drainage to the bilateral IPS. AVF: arteriovenous fistulas; CS: cavernous sinus; IPS: inferior petrosal sinus

was noted in 3 (4, 5, and 7 months later in each respective patient), whereas PT to LRT was observed in the other 3 patients. Spontaneous cure was noted in a patient with a LRT lesion at the time of attempted embolization 4 days after her first angiogram. On the other hand, there was no change in 4 patients with RT lesions (mean 2 months; range from 1 to 4 months).

Clinical Symptoms and Venous Drainage

As CS dural AVF frequently exhibit extracranial superficial symptoms, it is possible to clarify the clinical course and timing of symptomatic change. CS dural AVF at the early stage have similar drainage to a normal venous pattern. The shunt flows into the CS and goes throughout all drainage routes; therefore, typical symptoms are diplopia, tinnitus, and mild conjunctival congestion. At this early stage, therapeutic indications are limited because symptoms are mild and it is difficult to identify the exact shunt point angiographically and embolize it by transvenous embolization.

As the lesion progresses, the drainage route is gradually limited by thrombosis and compartmentalization within the CS. Obstruction usually begins from the lower channel, such as the IPS, and the shunt flow is concentrated toward anterior drainage such as the SOV.

When the anterior outflow from the SOV to the facial vein and/or the superficial temporal vein is occluded, high-pressure stagnation of the ocular vein develops and the intraocular pressure rapidly increases, which causes visual impairment. On angiography during this period, the shunt flow is slow and poorly visualized due to the nearly closed outflow, and the shunt appears to be almost cured, representing "paradoxical worsening."

Beyond this period, spontaneous thrombosis and clinical cure may be observed. However, prior to this change, irreversible glaucoma or retinal hemorrhage may cause visual loss. Therefore, urgent treatment is required. On the other hand, when the SOV is occluded, intracranial hemorrhage may develop if the sole route of intracranial reflux, such as the SMCV and uncal vein, is remained.

According to the Japanese nationwide survey, clinical symptoms in CS dural AVF were mostly benign at onset compared with other sites, and aggressive symptoms were only noted in 5.6% (1.8% intracranial hemorrhage, 1.3% venous infarction alone, 1.8% chronic intracranial hypertension alone, 0.5% convulsions alone, and 0.3% consciousness disorder alone).

Diagnosis

MRI/MRA

Identification of the shunt point is the most important factor for successful TVE against dural AVF. MRI is the first modality used for the diagnosis of CS dural AVF, similar to other lesions.

On conventional MRI, important findings are not only substantial change, such as edema and venous infarction, but also venous dilation. 3D time-of-flight (3D-TOF) MRA is also a common screening method for cerebral vascular disease, and it has high detection capability even for CS dural AVF. Diagnosis by MRA is usually based on findings such as dilated dural arteries and draining early veins or sinuses. In particular, original TOF MRA images, which delineate the affected sinuses receiving the shunt flow as high-signal-intensity regions, are of diagnostic value.

However, even in normal cases, the jugular bulb, the IPS, and SS are often visualized as high signal, and it is sometimes troublesome to distinguish it from the CS dural AVF. In such cases, the MRA TOF original image is useful. In the CS dural AVF, not only the venous sinus but also fine high signal that seems to be enlarged arteries is often accompanied.[17] Moreover, recent 4D MRA is more reliable because detailed hemodynamic evaluation is possible.

Ultrasound echography

SOV color Doppler flow imaging obtains information about the flow direction of the SOV along with the two-dimensional image in B mode, and can visualize the SOV, blood flow direction, and waveform.[18]

In CS dural AVF, enlargement of the SOV is observed first, and then the arterial pulse wave affects the original waveform in the SOV (**Fig. 3**). Furthermore, reflux of the SOV is observed according to the increased arterial shunt flow to the SOV. Therefore, SOV echography is simple and useful, can be performed in a short time, and can be repeated.

Angiography and cone-beam CT technology

MRA has diagnostic limitations. Identification of the shunt point is limited due to insufficiency of spatial resolution, and the affected sinus is less visualized when the shunt flow is slow. In these situations, cerebral angiography is still valuable for the identification of CS dural AVF because of its higher spatial resolution.

Moreover, recent modern angiographic systems use a flat panel detector, enabling cone-beam CT, which provides CT-like images using an angiographic system.[19] At present, after insertion of the microcatheter up to the shunt point, we routinely perform scanning for 20 seconds with contrast enhancement using a twofold or threefold dilution and send the data to the workstation for 3D reconstitution. By observing serial images from the preferred direction on

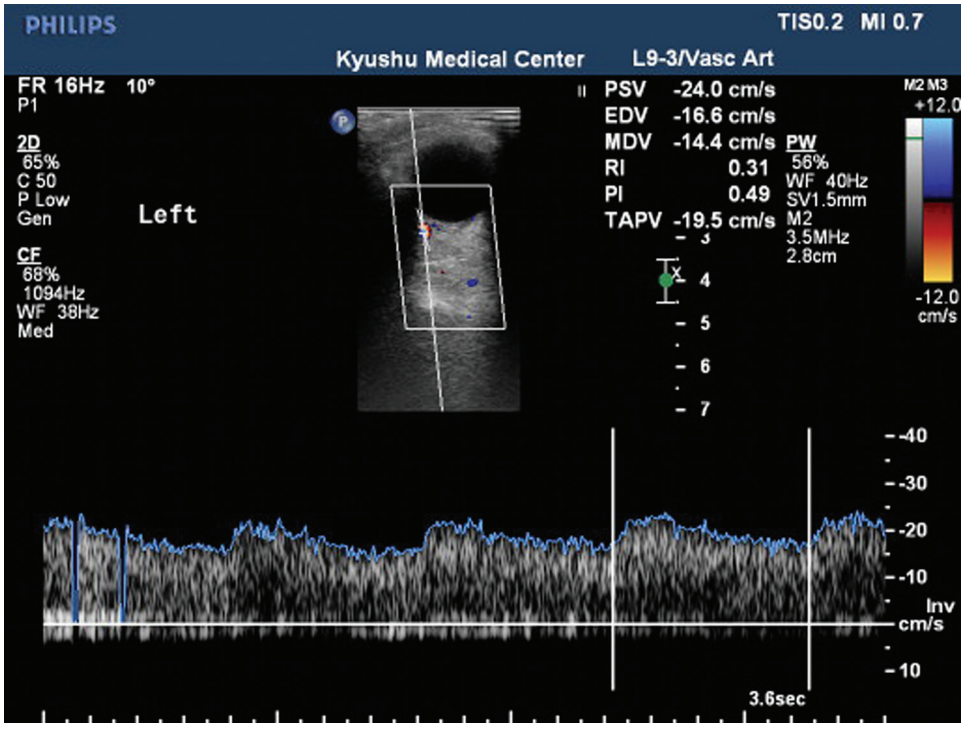

Fig. 3 Superior orbital vein color Doppler flow image. The arterial pulse wave pattern is recognized in the original wave in the SOV. SOV: superior ophthalmic vein

the 3D workstation, the site of connection of the feeding artery, the shunt point, and the position of the catheter can be identified (**Fig. 4**).

Feeding Artery and Shunt Point in CS Dural AVF

From the internal carotid artery, the inferolateral trunk (ILT), meningohypophyseal trunk (MHT), and recurrent meningeal and recurrent deep ophthalmic artery originated from ophthalmic artery may feed the shunt. The MHT usually terminates into the medial aspect of the CS, and the ILT can supply both medial and lateral aspects of the CS. On the other hand, from the external carotid artery (ECA), several arteries feed the CS. First, there are middle meningeal arteries (MMA), which include the anterior branch and CS branch. From the distal portion of the ECA, the accessory meningeal artery, artery of the foramen rotundum, and artery of the superior orbital fissure may supply the shunt. Furthermore, ascending pharyngeal arteries (APA), such as the neuromeningeal branch and pharyngeal branch, supply the posterior aspect of the CS.

Regarding the shunt point in the CS, Kiyosue et al.[20] retrospectively analyzed 19 consecutive patients with CS dural AVF.

They identified a total of 41 shunted pouches in 19 patients. The number of pouches ranged from 1 to 4 (2.2 [mean]) in each patient. In 16 patients, the pouch was located posteromedially (PM) to the CS, where it was in or adjacent to the clivus. In 13 patients, the pouch was located posterolaterally (PL) to the CS. The pouch was lateral to the CS (lateral wall of the CS, laterocavernous sinus, or sphenoparietal sinus) in 6 patients and medial to the CS (medial wall of the CS) in 3 patients.

They also reported a significant relationship between the location of the pouch and the feeding artery. The bilateral branches of the APA and the pharyngeal artery were significantly related to the PM pouch, the ipsilateral accessory meningeal artery was related to the PL pouch, the anterior branch of the MMA was related to the lateral side, and the artery of the pterygoid canal was related to the medial side.

Drainage Route of CS Dural AVF

Anterior drainage is observed into the SOV and IOV, which can cause ocular symptom such as exophthalmos and chemosis. Posteroinferior drainage is into the IPS, basilar plexus, and pterygoid plexus, which may result in bruit and cranial nerve deficit. Posterior drainage into the SPS may lead to bruit. The CVD through the sphenopari-

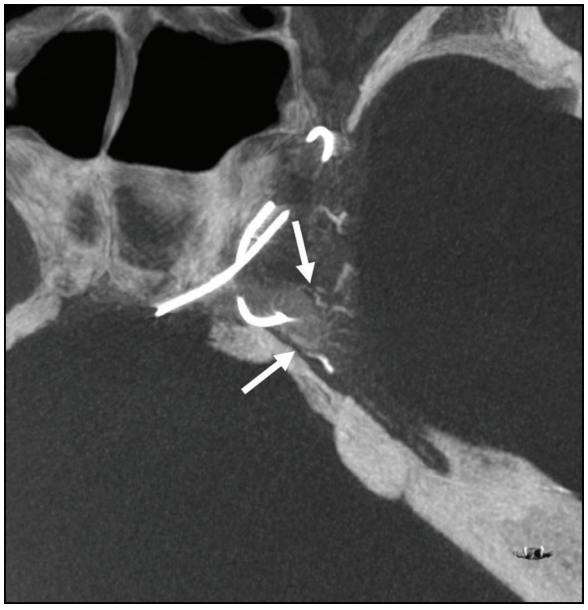

Fig. 4 HRCBCT. The tip of the catheter is identified around the shunt point (arrows). HRCBCT: High-resolution cone-beam CT

etal sinus and SMCV can cause venous infarction and hemorrhage. Cerebellar drainage into the petrous vein (PV) via the SPS may lead to cerebellar ataxia and hemorrhage. Deep drainage into the deep middle cerebral vein, prepontine bridging vein, and uncal vein can also lead to hemorrhage.

Classification of CS Dural AVF

Classification of CS dural AVF from the viewpoint of embryology

Geibprasert and Lasjaunias et al.[21] classified dural AVF into three groups such as the ventral epidural, dorsal epidural, and lateral groups. According to their classification based on embryology, CS dural AVF belong to the ventral epidural group.

The ventral epidural group was dominant in female and with more benign clinical presentation. Also, in this group, lower rate of CVD and spinal venous reflux was observed.

Classification of CS dural AVF from the arterial side

Barrow classification is known as the classic classification of CS dural AVF. This is composed only of information on the arterial side.[22] As TVE has recently become more popular as treatment for CS dural AVF, this classification is used less often. According to this classification, many CS dural AVF are classified as Type D.

Classification of CS dural AVF from the venous drainage side

The Cognard and Borden classifications focus on venous drainage, and are often used when considering therapeutic indications and methods.[23]

In the Cognard classification, venous drainage patterns of dural AVF are correlated with increasingly aggressive neurological clinical courses. It was first described in 1995 and may be the most widely used classification system for dural AVF.

The Cognard classification is based on the direction of drainage, the presence of CVD, and venous outflow architecture such as non-ectatic cortical veins, ectasia cortical veins, and spinal perimedullary veins. Type I lesions drain into the dural sinus, have an antegrade flow direction, and lack CVD. Type II lesions are subcategorized into 3 types: type IIa lesions drain retrogradely into the sinus without CVD, type IIb lesions drain antegradely into the sinus with CVD, and type IIa + b lesions drain retrogradely into the sinus with CVD. Type III, IV, and V lesions all have CVD without dural venous drainage, and variable cortical venous outflow architecture.

The Borden classification depends on the type of venous drainage: type I, drainage into the sinus without cortical drainage; type II, drainage into the sinus with retrograde CVD; and type III, retrograde CVD alone.[24]

Recently, Thomas et al.[25] proposed an updated classification system using venous drainage. Their classification system was easily applicable in clinical practice, and demonstrates significant correlations with symptomatology, treatment approach, and outcome. They defined posterior/inferior drainage primarily as that through the SPS and IPS, pterygoid, and parapharyngeal plexus. Anterior drainage was through the SOV and the inferior ophthalmic vein. CVD was defined as filling of the SMCV, perimesencephalic, and cerebellar venous system.

In their classification, type 2 lesions, which have posterior/inferior and anterior drainage, are significantly correlated with ocular/orbital symptoms and cavernous symptoms only. Type 3 lesions, which have anterior drainage only, are significantly correlated with ocular/orbital symptoms only, and type 4 lesions, which have retrograde drainage into cortical veins with cortical symptoms (e.g., intracranial hemorrhage, seizure, or focal neurological deficits) with or without ocular/orbital and cavernous symptoms.

Disclosure Statement

The author has declared no conflicts of interest related to this article.

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