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REVIEW ARTICLE

Intracranial dural arteriovenous fistula: a comprehensive review of the history, management, and future prospective

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Abstract

Dural arteriovenous fistulas (DAVF) are abnormal acquired intracranial vascular malformations consisting of pathological connections located within the dura between the pial arteries and the veno vasora, comprising the walls of the dural sinuses, bridging veins, or transosseous emissary veins. Dural arteriovenous fistulas are distinguished from arteriovenous malformations by their arterial supply from the vessels that perfuse the dura mater and lack of a parenchymal nidus. They are most commonly situated at the transverse and cavernous sinuses. The mechanism of development behind dural arteriovenous fistula can be explained by the molecular and anatomical factors. Multiple classification systems have been proposed throughout history including; Djindjian and Merland, Cognard, and Borden classification systems. The aggressiveness of the clinical course in intracranial dural arteriovenous fistula can be predicted through the angiographic patterns of venous drainage, more specifically, the presence of cortical venous drainage, the presence of venous ectasia, and the aggressiveness of clinical presentation. Intracranial dural arteriovenous fistulas might be discovered incidentally. However, if symptomatic, the clinical presentation ranges from mild neurological deficits to severe, lethal intracranial hemorrhage. Angiography is the imaging of choice to investigate, diagnose, and plan treatment for intracranial dural arteriovenous fistula. The management algorithm of intracranial dural arteriovenous fistula can be broadly divided into conservative, surgical, endovascular, and/or radiosurgical options. With the advent of endovascular therapies, surgery has fallen out of favor for managing intracranial dural arteriovenous fistulas. In the present article, the pathophysiology, classifications, natural history, clinical manifestations, radiological features, management, and complications are comprehensively reviewed.

Keywords Connection · Endovascular · Review · Shunt · Vascular

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Introduction

Dural arteriovenous fistulas (DAVF), also known as dural arteriovenous shunts, are abnormal acquired intracranial vascular malformations consisting of pathological connections located within the dura between the pial arteries (occasionally) and the veno vasora, comprising the walls of the dural sinuses, bridging veins, or transosseous emissary veins [1]. It was described for the first time in 1881 by Rizzoli, and the first angiographic description was reported by Sachs in 1931 [2].

The estimated true incidence of intracranial dural arteriovenous fistulas remains undetermined [3]. However, the reported incidence ranges between 10 and 15% of all diagnosed intracranial vascular malformations [2]. Of note, some intracranial dural arteriovenous fistulas remain asymptomatic or regress spontaneously [4]. Therefore, the true incidence might have been underreported [4].

Dural arteriovenous fistulas are distinguished from arteriovenous malformations by their arterial supply from the vessels that perfuse the dura mater and lack of a parenchymal nidus [5]. They affect patients between 50 and 60 years of age and are less common in younger age groups, especially children [5]. There is no clear sex predilection nor a genetic component to these lesions [5]. Most dural arteriovenous fistulas are idiopathic [5]. Their clinical manifestations vary greatly and depend on both the hemodynamic characteristics and the location of the fistula [5]. Patients with symptoms of cortical venous hypertension have a significantly higher risk of new neurological events [6]. They have been considered benign, but it appears that some behave more aggressively, causing intracerebral hemorrhage and progressive neurological symptoms [7].

Location

Despite their occurrence at any location within the intracranial dura mater, cranial dural arteriovenous fistulas are most commonly situated at the transverse and cavernous sinuses [2]. In a cohort with pooled data, 90% of Borden grade III dural arteriovenous fistulas were located in the anterior cranial fossa, as opposed to grade I (10%) and II (0%) [8]. However, the majority (84%) of cavernous dural arteriovenous fistulas were of type I, as opposed to type II (11%) and III (5%) [8].

Pathophysiology

Although several theories have been postulated to describe the pathogenesis of intracranial dural arteriovenous fistulas, the true cause behind their development is still controversial. The mechanism of development behind dural arteriovenous fistula can be explained by the molecular and anatomical factors.

Molecular factors

The first major factors attributed to the formation of dural arteriovenous fistula are venous thrombosis and venous hypertension [9–11]. The development of angiogenesis, evidenced by the enhancement of vascular endothelial growth factor (VEGF) in the dura, results in new shunt formation and/or microshunt opening within the dura [2].

Hypoxia-inducible factor-1 (HIF-1) expression was observed in the pro-angiogenic states in non-ischemic venous hypertension. HIF-1 enhances the expression of VEGF in the surrounding tissue, stroma-cell derived factor alpha (SDF-1) in the macrophages, and MMP-9 matrix metalloproteinase in the neutrophils [12, 13]. After arteriovenous shunt formation, the endothelial shear stress and nitric oxide maintain VEGF [12, 13]. Furthermore, in dural arteriovenous fistula, elevated D-dimer levels might explain the thrombotic abnormalities [14]. All these factors can play a role in the formation of multiple dural arteriovenous fistula. The multiplicity of dural arteriovenous fistula might be explained by the presence of cortical venous drainage [15].

The occurrence of venous thrombosis and hypertension in expected locations within the cerebral venous system has not been explained. Formation of dural arteriovenous fistula has been proposed to form in three stages [16]. Stage one: venous sinus thrombosis formation. Stage two: development of microscopic venous fistulas within the wall of the thrombosed venous sinus linking vasa vasorum to the small venous tributaries. Stage three: recanalization of the thrombosed veins. Stage two is dependent on the angiogenic factors.

Anatomical factors

The anatomical factors classification of dural arteriovenous fistula, based on the embryological origin of three discrete epidural venous systems developed by Geibprasert et al. [17], can assist in the understanding of dural arteriovenous fistula locations in the venous drainage pathways [17].

The ventral epidural space, in which originates more than half of all dural arteriovenous fistulas, is in direct contact with the spongious bony structures of the petrous bone, basiocciput, basisphenoid, and sphenoid wings [17]. Dural arteriovenous fistula in this region may invade and/or derive blood supply from these adjacent bony structures [17]. The fistulas in this region usually involve the epidural space [17].

The dorsal epidural space, in which originates 22% of all dural arteriovenous fistulas, is formed by the connection of calvarial osseous and cerebral leptomeningeal veins [17]. It composes the superior sagittal sinus, transverse sinus,

sigmoid sinus, medial occipital sinus, posterior marginal sinus, and torcular Herophili [17].

The lateral epidural space, originating 21% of all dural arteriovenous fistulas, comprises all emissary bridging veins and forms the marginal sinus at the level of foramen magnum, emissary veins at the level of anterior and posterior condylar confluences, the vein of Galen, the cavernous, basitentorial, petrosal, sphenoparietal sinuses, intraorbital, and cribriform plate veins [17].

Classification systems

In 1977, Djindjian and Merland first described the connection between angioarchitecture and hemorrhage risk in dural arteriovenous fistula, from which the two most utilized grading systems were derived [18]. Cognard [19] and Borden [20] classification systems both associate the pattern of venous drainage with hemorrhage risk and neurological deficit (Table 1) [19, 20].

The defining angiographic feature that differentiates benign from aggressive lesions is the cortical venous drainage (CVD). The annual risk of hemorrhage in all grading systems is 0, 6 and 10% in low-grade, intermediate-grade, and high-grade dural arteriovenous fistula, respectively [8]. The incidence of aggressive presentation in low-grade lesions is 10%, intermediate-grade lesions is 54%, and high-grade lesions is 89% [19]. Poor long-term prognosis of intermediate and high-grade lesions is determined by the presence of intracranial venous hypertension [6, 19]. Angiographic evidence of cortical venous drainage does not indicate the presence of intracranial venous hypertension [6].

Söderman et al. [21] reported annual rates of hemorrhage of 1.5 and 7.4% in unruptured and ruptured high-grade dural arteriovenous fistula, respectively [21]. Comparably, Strom et al. [22] reported annual rates of hemorrhage of 1.4% and 7.6% in unruptured and ruptured high-grade lesions, respectively [22]. Detailed analysis of seven studies by Gross and Du, including their own cohort group, concluded that the annual hemorrhage rates for Borden Types II and III dural arteriovenous fistula were 6 and 10%, respectively [8]. The risk of recurrent hemorrhage in the first two weeks of the initial event can be as high as 35% [8]. The rate of recurrent hemorrhage has been reported to increase up to 46% per year [8].

Natural history

The aggressiveness of the clinical course in intracranial dural arteriovenous fistula can be predicted through the angiographic patterns of venous drainage, more specifically, the presence of cortical venous drainage, the presence of venous ectasia, and the aggressiveness of clinical presentation [8]. In 1972, Houser et al. [23] were the first to report the relationship of cortical venous drainage with intracranial hemorrhage [23].

The foundation of all classification systems, discussed previously, is the pattern of venous drainage [19]. Low-grade fistulas are characterized by the absence of cortical venous drainage and have an annual risk of hemorrhage and non-hemorrhagic neurological deficit of 0%. For intermediate-grade fistulas with indirect cortical venous drainage, the reported annual incidence of hemorrhage is 6%. The incidence of intracranial hemorrhage and

Table 1 Classifications of dural arteriovenous fistula based on the pattern of venous drainage

Type	Djindjian and Merland 1977	Cognard 1995	Borden 1995
I	Dural sinus or meningeal vein	Dural sinus with normal antegrade flow	Antegrade flow into dural sinus
Ia			Single meningeal feeder
Ib			Multiple meningeal feeders
II	Dural sinus with cortical venous reflux	Dural sinus with reflux	Antegrade into dural sinus and retrograde into cortical vein
IIa		Retrograde into dural sinus only	
IIb		Retrograde into cortical vein(s) only	
IIc		Retrograde into dural sinus and cortical vein(s)	
III	Purely into cortical vein	Cortical vein only without venous ectasia	Retrograde drainage into cortical veins which may be dilated
IV	Cortical vein with supra or infratentorial venous lake	Cortical vein only with venous ectasia	
V		Intracranial DAVF drainage into spinal perimedullary veins	

DAVF Dural arteriovenous fistula

non-hemorrhagic neurological deficit increases up to 10% in high-grade fistulas with exclusive direct drainage to cortical veins. The reported annual rate of non-hemorrhagic neurological deficit for both high and intermediate-grade dural arteriovenous fistula is 4%. Angiographic evidence of venous ectasia increases the annual rate of hemorrhage to 21% [8, 20].

Upgrading of low-grade fistula, without cortical venous drainage, is a potential [8]. This can be caused by the progression of venous stenosis, the emergence of additional feeders, or an insidious increase in arterial flow [5]. The reported rate of upconversion of low-grade fistulas is 1.4% [8]. For instance, dural arteriovenous fistula without cortical venous drainage by themselves do not necessarily pose a significant risk of hemorrhage [8]. However, they should be monitored and followed-up clinicoradiologically to assess progression and development of cortical venous drainage [8].

Contrarily, spontaneous resolution of dural arteriovenous fistula can occur. In a cohort study with incorporated data from the literature, the reported rate of spontaneous resolution was 13% for low-grade fistulas and 3% for intermediate and high-grade fistulas [8]. In fistulas with cortical venous drainage, the clinical presentation is a predictor for the future development of intracranial hemorrhage. In Gross et al. cohort, the hemorrhagic presentation was a significant predictor of subsequent hemorrhage and non-hemorrhagic neurological deficits [8].

Spontaneous resolution of low and high-grade intracranial dural arteriovenous fistula has been reported in the literature, of which, the underlying mechanisms are still unclear [8, 24, 25]. Additionally, the natural history of incidental intracranial dural arteriovenous fistula is not known, given that many of the cases presented in the literature are of symptomatic patients [8, 26].

Clinical manifestations

If symptomatic, patients with intracranial dural arteriovenous fistula may present with mild neurological deficits to severe, lethal hemorrhage [2]. The constellation of symptoms primarily depends upon the location and the pattern of venous drainage. [2]. The presence of impaired venous drainage can manifest as increased intracranial pressure, papilledema, or persistent/transient neurological deficits [27]. Pulsatile tinnitus, without or with bruit, can occur if the dural arteriovenous fistula is involving the petrous region and draining into the sigmoid or transverse sinuses [2]. Aggressive symptoms include intracerebral hemorrhage, subdural hemorrhage, and subarachnoid hemorrhage [19].

Radiological features

Computed tomography

Initially and before any intervention, computed tomography (CT) scan without contrast must be performed to rule out sequelae of intracranial dural arteriovenous fistula, such as intracranial hemorrhage, subarachnoid hemorrhage, hydrocephalus, and vasogenic edema caused by cortical venous hypertension [2, 28]. Chronic venous congestion produces an area of hypodensity on CT scan due to edema [2]. CT scan may sometimes demonstrate nonspecific findings, such as asymmetry of the dural venous sinuses or mild enlargement of the cavernous sinus/superior ophthalmic vein [5, 28]. Furthermore, a non-enhanced CT scan can delineate bony defects, resulting from enlargement of emissary veins [2]. However, a brain CT scan is usually unable to detect intracranial dural arteriovenous fistulas alone [5, 28].

Magnetic resonance imaging

Magnetic resonance imaging (MRI) can display features of dural thickening, hypertrophied arteries, thrombosed, and/or stenosed venous sinuses [2]. Furthermore, MRI in intracranial dural arteriovenous fistulas demonstrate dilated cortical veins without a parenchymal nidus [2]. The arterial supply to the dural arteriovenous fistula is not typically demonstrated on MRI [2]. The common findings of dural arteriovenous fistula on MRI include the visualization of transosseous collaterals, increase in the number and size of extracranial vessels, stenosis, occlusion, or abnormal dural sinuses flow [28, 29].

Aggressive dural arteriovenous fistulas demonstrate diffuse white matter edema secondary to venous congestion, diffuse cortical enhancement, dilated pial vessels, and possible sequelae such as infarction and hemorrhage [28]. The actual fistulous channels may not be detected using MRI because of their adjacent location to the dura or their small size and the lack of contrast between the signal intensity void within the fistula and adjacent bony structures [28]. Flow voids from large, arterialized draining veins and varices may be visualized on T2-weighted MRI. Fistula-related microhemorrhages are often more pronounced on T2*/gradient echo MR sequences.

Post-gadolinium T1-weighted MRI may show venous ectasia, dilated medullary and leptomeningeal vessels, venous sinus occlusion/thrombosis and parenchymal enhancement. Hyperintensities on T2-weighted and fluid-attenuated inversion recovery sequences suggest vasogenic edema because of venous hypertension [5].

It is possible that some patients with a provisional clinical diagnosis of dural arteriovenous fistula but negative findings on MR imaging are eventually provided with an alternate diagnosis and thus are not offered conventional angiography, despite being more sensitive in detecting dural arteriovenous fistulas [28].

Angiography

Angiography is the imaging of choice to investigate, diagnose, and plan treatment for intracranial dural arteriovenous fistula [2]. It can be utilized to identify the site of the fistula, presence of arterial feeders, and the direction of venous drainage [2]. Some of the well-defined features of intracranial dural arteriovenous fistula are venous ectasia, calcification, stenosis, arteriovenous shunting, impaired drainage of cerebral parenchyma with delayed venous drainage, and cortical venous collaterals [2].

Selective studies of the external carotid, internal carotid, and vertebral arteries allow assessment of the morphology of draining veins, cortical veins, dural sinuses and feeding arteries [2]. Advantages of conventional angiography include improved identification of small dural arteriovenous fistulas, the opportunity for endovascular embolization, and follow-up of previously embolized dural arteriovenous fistulas [28].

Management

The management algorithm of intracranial dural arteriovenous fistula can be divided into conservative, surgical, endovascular, and/or radiosurgical (Fig. 1).

Conservative management

The choice of conservative management is preserved for patients without cortical venous drainage on angiographic studies or debilitating symptoms [1]. Since dural arteriovenous fistulas could regress spontaneously, the management includes interval imaging and clinical follow-up along with symptomatic medical treatment [1, 30] Although angiography is not commonly performed for routine follow-up, it is required for any symptoms alteration [1, 30] Dural cavernous sinus fistulas necessitate frequent ophthalmologic evaluation with fundoscopic and visual field examination along with intraocular pressure measurement [31]. Intermittent external manual compression of the external carotid artery, or its branches, is a potential management option for transverse, sigmoid, or cavernous sinus dural arteriovenous fistulas [1, 32].

Surgical management

With the advent of endovascular therapies, surgery has fallen out of favor for managing intracranial dural arteriovenous

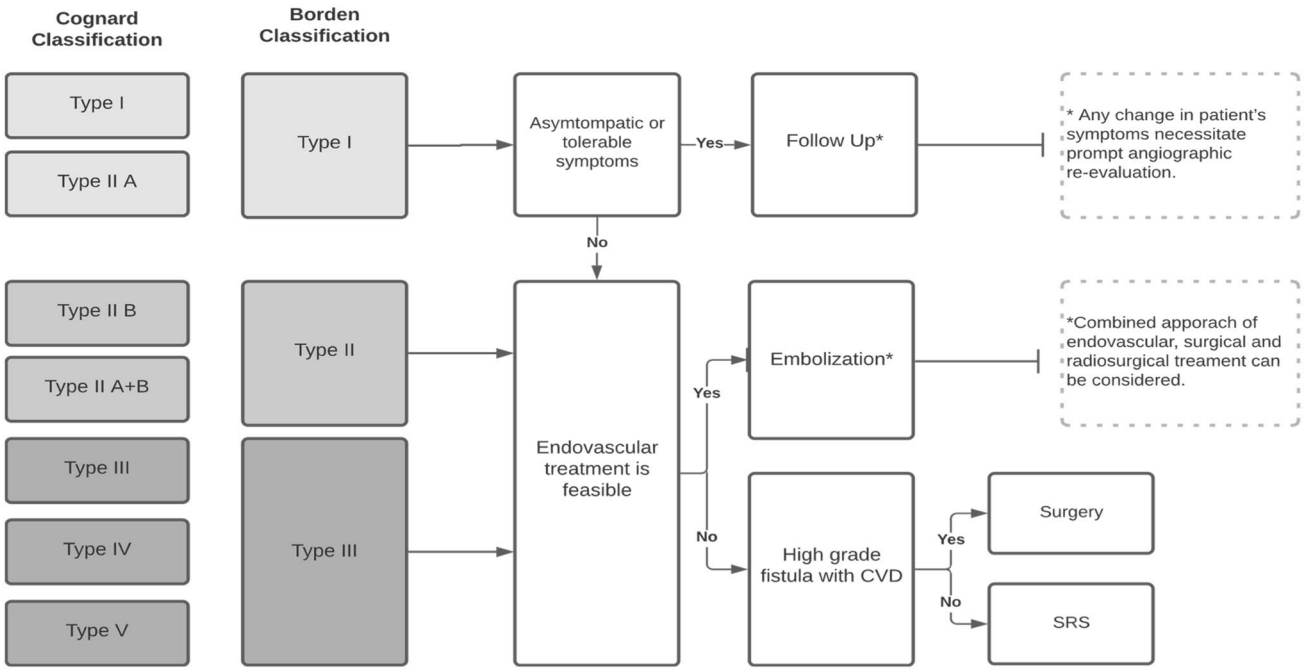

Fig. 1 Proposed management algorithm of intracranial dural arteriovenous fistula based on the available evidence

fistulas [2]. However, some dural arteriovenous fistulas, especially those located in the anterior cranial fossa, can be safely managed with surgical disconnection of the venous drainage [2, 33]. Open surgery is needed for fistulas with inaccessible anatomical location trans-vascularly [34]. It can be performed for the definitive treatment or as an adjunct to optimize endovascular treatment, especially in the case of fistulas of the cavernous sinus [34]. Additionally, presentation with hemorrhage or severe symptoms, as seizures or paresis, could prompt surgical intervention with excellent post-operative outcomes [35]. Surgery could be complemented with endovascular treatment for remaining residuals or partially treated fistulas [35].

Endovascular management

Endovascular management is the preferred treatment of choice in most cases of intracranial dural arteriovenous fistulas [1]. It could be achieved through trans-arterial, trans-venous, or direct puncture in case of inaccessibility which is less frequently used [1]. Trans-arterial embolization of the branches of the external carotid artery with particles can be safely performed in order to reduce the flow of the shunt [2]. As feeding arteries are generally difficult to catheterize, complete obliteration is not usually achievable [2]. As a result, trans-arterial embolization can serve as a temporary measure before other procedures are attempted, e.g., surgery and radiation [2].

Other adjunct treatment options include Onyx injection, coiling, and venous stenting [2]. On the other hand, when accessible, trans-venous embolization is more curative with higher rates of complete resolution, rendering it to be the preferred treatment of choice for most cases, especially for dural arteriovenous fistulas of the cavernous sinus, superior petrosal sinus, sigmoid sinus, and transverse sinus [1, 36–38]. In contrast to the trans-arterial approach, coiling is the preferred embolic agent of choice in transvenous embolization with higher accuracy and lower complication rates than polyvinyl alcohol or liquid embolic agents such as ethylene–vinyl alcohol copolymer and n-butyl cyanoacrylate [1, 36].

Radiosurgery

Radiosurgery has been shown to be an effective adjuvant therapy in lesions with persistent flow after surgery or embolization [39]. Stereotactic radiosurgery, via linear accelerator or gamma knife radiosurgery, is reasonable for patients with low risk of hemorrhage since it might take several months for effective obliteration of the dural arteriovenous fistula [1]. Fistulas with cortical venous drainage are considered a good candidate for radiosurgery as they have a lower risk of hemorrhage and have shown a higher resolution rate following radiosurgery [40]. In addition, radiosurgery can be safely performed as an alternative for patients with contraindications to endovascular treatment, surgery, or anticipated subsequent morbidity [41]. Combination with endovascular treatment could optimize resolution rates [42].

Multiple reports have shown that radiosurgery is a safe therapeutic intervention with low complication rates [39, 40, 43, 44]. Successful obliteration rates after radiosurgery have been reported to range between 54 and 65% [45–52]. Chen et al. systematically reviewed the rate of obliteration in intracranial dural arteriovenous fistula after radiosurgery [40]. Complete obliteration after radiosurgery was significantly observed in 75 and 56% of lesions without and with cortical venous drainage, respectively [40]. The mean obliteration rate of dural arteriovenous fistula, treated with radiosurgery, in nineteen studies was 63% [40]. Sardana et al. reported complete obliteration after radiosurgery in all lesions without cortical venous drainage [41]. Clinical improvement after radiosurgery was reported to range from 66 to 86% [39, 43]. Lesions with persistent pial venous draining vessels are less favorably treated with gamma knife radiosurgery (GKRS) [39, 47].

The mechanism of efficaciousness of radiosurgery in treating dural arteriovenous fistula remains unclear. However, ionizing radiation has been postulated to exert its effect on the angiogenic factors, although not addressed in the literature, because it is less likely to affect the venous obstruction [39]. The evaluation of radiosurgery efficaciousness is mainly based on pre and post-treatment angiography, together with clinical symptoms [39]. Although expensive and invasive, post-treatment angiography has been shown to be superior to magnetic resonance imaging due to the limited resolution of dural arteriovenous fistulas on MRI [39]. Gamma Knife radiosurgery may be effective in deep dural arteriovenous fistulas and in lesions inaccessible by endovascular treatment [39].

Complications

The complications from endovascular procedures have decreased drastically with the development of more advanced techniques and a better understanding of venous and arterial anatomical variations. Nevertheless, detailed complications reporting is scarce in the literature [53]. In a recent meta-analysis of endovascular treatment of cavernous sinus dural arteriovenous fistulas, the treatment-related complication rate was found to be 7.75%, most commonly due to transient cranial nerve palsies [36].

While transvenous coiling has the lowest complication rate compared to other treatment options, it could be complicated by venous perforation or venous congestion, which could lead to significant morbidity [53]. The overall rate of recanalization and recurrence of treated fistulas is 4.5% with a 3-year predicted recurrence rate of 11% [54]. Recurrence risk factors include the presence of cortical venous drainage, deep cerebral

venous drainage, and tentorial location [54, 55]. Moreover, endovascular treatment and radiosurgery correlate with higher recurrence rates than surgery [54].

Conclusion

The dynamic nature of dural arteriovenous fistula formation challenges the accurate prediction of its course. The predilection of certain locations to have cortical venous drainage suggests the possible importance of the lesion location in the treatment plan. Despite the progress in our understanding of intracranial and spinal dural arteriovenous fistula, there is still a vast gap of knowledge in our understanding of its etiology and pathogenesis. The clinical presentation of these lesions can be unspecific and unpredictable. Thus, the treatment decision for asymptomatic and mildly symptomatic intracranial dural arteriovenous fistula is largely challenged by the uncertainty of their course. Future prospective, follow-up studies, and pooled literature data can bridge gaps in evidence and refine practice.

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