Literature Review

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Anesthetic management in malignant dural intracranial arteriovenous fistula. Literature review

Abstract

Arteriovenous fistulas correspond to a rare type of cerebral vascular malformation, consisting of a direct connection between the meningeal arteries and the dural venous sinuses or leptomeningeal veins. They represent between 10-15% of cerebral vascular malformations. The main clinical picture of presentation is incidental, however, the symptoms can vary from mild symptoms such as tinnitus to cerebral hemorrhage. The formation of this systemic entity is attributable to different factors, among which are the release of proangiogenic factors, increased microthrombotic activity, increased expression of fibroblast growth factor and endothelial growth factor. The first-line diagnostic method is computed tomography, the gold standard is digital subtraction angiography.

Keywords: Anesthesiology, Neurology, Neurosurgery.

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Abbreviations: icp: intracranial pressure, cvd: cortical venous drainage, evt: endovascular therapy, tae: transarterial embolization, davf: dural arteriovenous fistulas

Introduction and background

Dural arteriovenous fistulas (dAVFs) are a rare cerebral vascular malformation.[1] They involve a rare subset of cerebrovascular malformations defined by direct arteriovenous connections between the meningeal arteries and the dural venous sinuses or leptomeningeal veins without a true intervening nidus.[2] They are exclusive to the neuraxis, anywhere there is a dural-meningeal covering since the shunt site is contained within the dural leaflets.[3,4] The fistulous point is within the dural leaflets near a venous sinus, adding arteries, which usually perfuse the dura mater. They profoundly alter the architecture of local and global cerebral venous drainage.[2,4] Cerebral vascular neurodevelopment is the junction of multiple and coordinated steps, which are driven by local metabolic demands during embryogenesis The classification is based on venous drainage with an assessment of bleeding risk.[3,5,6]

Classification

The first classification reported is Aminoff, who based it on topography, dividing dAVF into two: anteroinferior and posterosuperior. In 1976 Castaigne classified them according to cortical venous drainage (CVD).[5] In 1977 Djindjian based himself on angioarchitecture to propose the first classification of this type.[7] The classifications currently used are based on the venous drainage pattern, the most widely used are the Cognard classifications (based on the angioarchitecture of venous flow)[8] and Borden (based on the venous drainage pattern).[9] The most common locations for this type of malformation are the cavernous sinus, the cribriform plate, the transverse sigmoid sinus and the tentorium. The exact etiology of dAVF is not entirely clear. It is attributed to anomalies of the venous system, occlusion or thrombosis of the venous sinuses (release of angiogenic factors by the thrombus in formation), increased systemic thrombotic activity, head trauma, a history of cranial surgery. Venous hypertension causes tissue hypoxia, which leads to an increase in the production of angiogenic factors, which promote endothelial proliferation and neoangiogenesis.[9-13] The principle of endovascular treatment is the occlusion of the pathological vessels of the malformation using a selective microcatheter. Currently the embolic materials used for cerebral arteriovenous malformations and dural arteriovenous fistulas are liquid agents.[11,12,14-16] They represent between 10 - 15% of all intracranial vascular malformations.[17-20] Although dAVFs are usually asymptomatic and their diagnosis is often incidental, mild symptoms related to increased sinus drainage are found to be associated with much more serious symptoms secondary to increased cortical venous pressures such as hemorrhage or non-hemorrhagic neurological deficits.[1,2]

Diagnosis

Non-invasive studies such as computed tomography (CT), CT angiography, CT venography, and magnetic resonance imaging are the first instance tests for the diagnostic approach of this type of malformation. Digital subtraction angiography is considered the gold standard in case of suspected dAVF, with 6-vessel assessment, it provides us with precise and very valuable information, such as the origin of the arterial supply, the drainage pattern, as well as sinus involvement. venous. The presence of early venous drainage is indicative of a dAVF.[1]

Case presentation

24-year-old female with no chronic-degenerative history, complete immunizations. Presenting a clinical picture of 8 years of evolution with pulsatile and oppressive left hemicranial headache, of intermittent duration with a score of 10/10 on an analogue scale of pain, during exacerbations, which had partial improvement with the administration of NSAIDs, he was evaluated by neurology, who started treatment with magnesium valproate, amitriptyline and topiramate, which partially improved the symptoms. In 2020, paresthesias were added in both thoracic extremities, without having another neurological deficit, diagnostic angiography was performed with findings of left dural arteriovenous fistula (AVFd) Cognard II (a + b), Borden II; for which she initiates a treatment protocol in neurological endovascular therapy. Endovascular procedure was performed by triple approach (bifemoral and left carotid), under intravenous general anesthesia.

Transanesthetic: type I monitoring (electrocardiogram systemic blood pressure, oxygen saturation) and monitoring of anesthetic depth with entropy.

Conventional anesthetic induction based on fentanyl at 0.045 mcg/kg/min, propofol TCI (Schnider) 3 mcg/ml, lidocaine 1 mg/kg, rocuronium 50 mg. A 20-gaus right radial artery was cannulated for type II monitoring. Volume controlled mechanical ventilation.

Maintenance: Fentanyl infusion 0.026 mcg/kg/min, Propofol (Schinder) 2.0 - 5.0 mcg/ml, guided by entropy RE/SE 50-55, BSR 0%.

Bilateral femoral and left carotid access were used to approach the fistula. As embolizing agents, coils and embolic fluid were used. Mean arterial pressure control was started at <70 mm/hg using nitroprusside at 0.5 mcg/kg/min.

Post-anesthetic: The patient was extubated, without complications, and the patient was discharged to the post-anesthetic recovery unit with vital signs within goals.

Discharged at home 96 hours after embolization, without neurological deficits.

Technical considerations

Anesthetic management for neurological endovascular therapy (TEV) procedures implies knowledge of multiple considerations, potential difficulties related to access and manipulation by the anesthesiologist in this type of procedure. In our center, the VTE endosuite is a surgical room with a large space, multiple instruments, and many people simultaneously caring for the patient. Regardless of the fact that it is a large room, in general, the space assigned to the anesthesiologist and his team is very limited, due to the mobility that the surgical table must have, the rotation of the angiographer for the different angles and projections necessary for the approach. Our endosuite is equipped with an advanced anesthesia machine and anesthetic depth monitoring with entropy, conveniently for the interventional team the entropy sensor, unlike other options such as BIS®, SedLine®, electroencephalogram, is the best option, due to that does not interfere with the visibility of structures nor does it interfere with their radiological visibility in angiographic controls.

Airway and vascular access

It is known that the endosuite surgical table is difficult to manipulate, speaking specifically in relation to the airway, it is designed with lateral supports for the head, which makes it difficult for us to manipulate when aligning the axes and achieving control of the airway on the first attempt, avoiding multiple manipulations and the painful stimulus that they can cause is essential in vascular pathologies such as unruptured aneurysms, which could rupture upon repetitive algic stimulation. These same obstacles are found when trying a central vascular access, in the procedures that are required, the subclavian vein access technique requires a discreet turn of the head to the contralateral side of the access, to improve the visualization of the structures, which is practically impossible on this table. In our center, the central vascular accesses are performed under ultrasound guidance, thereby reducing potential complications; however, due to the conformation of the table, space is very limited for both aseptic and antiseptic techniques, placement of sterile fields, and material with this. Also the setup of the ultrasound transducer.

Track and circuit control

The movement of the surgical table in the cephalad - caudal direction should be considered, as well as that of the angiographer in the 360° turn, to avoid accidentally pulling or extracting some venous or arterial lines or even the anesthetic circuit, because it must be done. Anticipate the length of these and that our equipment (machine, perfusers, TCI equipment,) hinder the rotation of the angiographer or even that this equipment is damaged.

Discussion

Coils are used as a complement to liquid agents in specific techniques. Currently available liquid embolic agents are divided into two cyanoacrylates or adhesive (glue-like) embolic agents and copolymers or nonadhesive embolic agents.[21,22] Histological studies suggest that microscopic thrombosis plays an important role in dAVF formation. Immunohistochemical studies have detected the expression of fibroblast growth factor and endothelial growth factor in the dural sinus wall in patients with dAVF.[23,24] The general principles of neuroanesthesia are important in the management of these cases and particularly in the challenging environment of procedures outside the operating room such as the neurological endovascular therapy room. Special care is required to avoid increases in intracranial pressure (ICP), sudden changes in the patient's hemodynamics, as well as maintaining adequate cerebral perfusion, so invasive blood pressure monitoring is imperative in these cases. At present there is no scientific evidence that supports one anesthetic technique or another. In the current trend, total intravenous anesthesia is preferred, due to its pharmacokinetic and pharmacodynamic characteristics, within which cerebral vasomotor tone is preserved, better coupling flow-metabolism, vasoreactivity to carbon dioxide and less vasodilation.[25] It is considered that controlled ventilation in general anesthesia procedures reduces the risk of potential movement of the patient in the critical periods of the procedure, this in turn facilitates visualization of the cerebral vessels and control in the injection of embolic material. In addition to this, controlled hypotension would reduce the flow through the fistula. Avoiding periods of hypertension is critical to reduce episodes of intraoperative rupture and thus avoid intracerebral edema and hemorrhage.[25,26] As a perioperative objective in these procedures, maintenance of normotension or controlled hypotension is recommended, maintaining between 15-20% below the patient's initial blood pressure, although a high level of evidence has been found, this suggestion is supported by current evidence,[25,27-30] so routine monitoring should be complemented with invasive monitoring of systemic blood pressure. The use of a central venous catheter is considered in case of comorbidities and its placement is justified for the use of vasopressor infusions during the procedure.[28,29]

Catastrophes in neurological endovascular therapy

The first line of action will always be an adequate initial resuscitation. Communication with the intervention team, if necessary, request support from more expert personnel in neuroanesthesiology, ensure the airway if it is not available, determine the origin (hemorrhagic or ischemic). The use of embolic agents may present complications such as delayed parenchymal edema (1 to 90 hours after embolization) or hemorrhage adjacent to the malformation, perforation or rupture of blood vessels, retention of the microcatheter, and embolization of material into the normal brain parenchyma resulting in a stroke. There is also a potential risk of embolization of material beyond the brain.[28] Another important factor to consider and generally overlooked is the administration of intravenous fluids (to rinse the catheter), which makes it essential to place a urinary catheter, considering that the use of hyperosmotic contrast media produces significant diuresis, possible kidney injury.[28,29] In these situations, the most important thing in these events is prompt coordination with the neurosurgical team, and achieving immediate intervention with this, achieving a change in the patient's prognosis. In the event of a hemorrhagic event, it is essential to use protamine a (protamine 1 mg per 100 IU of heparin) for heparin antagonism, and maintenance of perfusion stockings according to the underlying pathology, simultaneously with the occlusion by the interventionist. Some other recommendations have been documented, such as maintaining normocapnia, use of osmolar therapy (mannitol 0.5g/kg, hypertonic solutions), slight elevation of the head, convulsive prophylaxis, titrating the inducer and inducing a suppression flare, thermal control (33 °- 34°) may even require decompressive ventriculostomy and control of intracranial pressure.[30-34]

Conclusion

Complex neurosurgical pathologies require a multidisciplinary approach for their approach and treatment. There must be an excellent surgical team and a neuroanesthesiology team, who must have continuous communication to successfully treat patients.

Appendices

Table 1 Cognard classification of intracranial dural arteriovenous fistulas[7]

Type		Description	Malignancy
I		Antegrade drainage in sinus venosus	
II			Low grade
	IIA	Main sinus drainage with secondary sinus reflux (retrograde sinus drainage)	
	IIB	Main sinus drainage with reflux into cortical veins	
	IIA + IIB	Drainage in the main sinus with reflux in the secondary sinus and cortical veins	
III		Direct cortical venous drainage without ectasia	High grade
IV		Direct cortical venous drainage with venous ectasia (>5 mm and 3_ greater than the diameter of the draining vein)	
V		Drainage into the perimedullary spinal veins	

Table 2 Borden's classification of intracranial dural arteriovenous fistulas[8]

TYPE	DESCRIPTION
I	Antegrade drainage in the dural sinus/meningeal vein
II	Antegrade drainage in the dural sinus and retrograde drainage in the cortical veins
III	Isolated retrograde drainage: — Drains directly into cortical veins — Entrapped segment of sinus with reflux in cortical veins —Variceal veins/dural lake with reflux into cortical veins

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