Optic Nerve Sheath Dural Arteriovenous Fistula Misdiagnosed As A Carotid Cavernous Fistula

Elena Downs[1*]

1. Department of Neurosciences, Royal Brisbane and Women's Hospital, Brisbane, Australia

* **Correspondence:** Elena Downs, Department of Neurosciences, Level 7 Ned Hanlon Building, Royal Brisbane and Women's Hospital, Butterfield Rd, Herston QLD 4029, Brisbane, Australia
(*elena.downs@health.qld.gov.au*)

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ABSTRACT

Dural arteriovenous fistulas (dAVf) are uncommon, acquired vascular lesions. Their location and vascular anatomy produces varied signs and symptoms and are misdiagnosed as other vascular anomalies. We present an atypical case of a dAVf identified along the left optic nerve sheath. The patient presented with progressive ophthalmological signs and was initially misdiagnosed with a left carotid-cavernous fistula. The aetiology, classification and treatment options are discussed.

CASE REPORT

CASE REPORT

A 78 year old female was referred from a peripheral hospital with a presumed left carotid-cavernous fistula (CCF). Her past medical history included paroxysmal atrial fibrillation, protein C deficiency with recurrent venous thromboembolism for which she was anticoagulated (Warfarin), chronic obstructive pulmonary disease (COPD) and non-small cell lung cancer. Approximately 3 months prior to presentation she first noticed double vision in her left eye. The precipitating event for her hospital presentation was a mechanical fall. Following the fall her diplopia became progressively worse and she noted new proptosis and epiphora (see fig 1). During her admission she also complained of decreasing visual acuity and new onset headaches. To further investigate the cause for her symptoms, she underwent a Computer Tomography Angiogram (CTA) and Magnetic Resonance Imaging and Angiogram (MRI/A) brain with Gadolinium. On CTA enlarged extra-ocular muscles and proptosis of the left eye were observed but internal carotid arteries, ophthalmic arteries and the cavernous sinus were reported as normal. The MRI/A added that the left superior ophthalmic vein was engorged and tortuous but again no clear abnormality of the carotids or cavernous sinus was identified. Despite reportedly normal carotid-cavernous anatomy, the other radiological findings were still thought to be concerning for a carotid-cavernous fistula and a cerebral angiogram was recommended.

The patient was subsequently transferred to our facility under the Neurosurgical team. On arrival she had a formal Neuro-Ophthalmology review and was found to have reduced but stable visual acuity, increased intraocular pressure in her left eye (27mmHg vs 9mmHg in the right eye) and evidence of subtle left optic nerve compression on visual field testing.

She then underwent conventional cerebral angiography which revealed a small left optic nerve sheath dural arteriovenous fistula (dAVf) at the superio-medial aspect of the orbit (Fig 2-5). Arteriovenous shunting from the left middle meningeal and ophthalmic artery into a partially thrombosed superior ophthalmic vein (SOV), angular vein and a smaller accessory vein were observed. Venous drainage also extended across to the right SOV and cavernous sinus. A venous stricture in the left SOV near the orbital roof was also noted (Fig 4) as well as bilateral small cavernous internal carotid artery (ICA) aneurysms. In retrospect, the cavernous ICA aneurysms were identifiable on CTA (Fig 6) as well as an abnormal vascular network around the optic nerve on MRI/A (Fig 8).

After multidisciplinary discussions, conservative management was recommended. Endovascular and surgical intervention was deemed to be both technically challenging and high risk for complications particularly permanent visual loss. The Neuro-Ophthalmology opinion was that risk of blindness was not significantly elevated however frequent

visual assessment and tonometry with her local Ophthalmologist were arranged. If her vision rapidly deteriorated in the future and intervention for the dAVf was required, the provisional plan would be for an orbital approach involving catheterization of either the SOV or inferior ophthalmic vein to attempt fistula embolization.

DISCUSSION

Etiology & Demographics:

Dural arteriovenous fistulas are acquired dural lesions resulting from an anomalous vascular communication between meningeal arteries and their associated draining veins [1]. They can occur anywhere throughout the neural axis. Their presentation and symptomatology is diverse and dependent on location and vascular supply. Optic arteriovenous fistulas have previously been reported [2,3,4] however a dAVf located on an optic nerve sheath is a particularly rare occurrence with only one previously published case report identified in the literature [5]. Gender and age predilection is unknown.

The optic nerve sheath is an extension of the intracranial dura from the superior orbital fissure to the back of the eye [6]. The arterial supply comes from two embryological sources; the ophthalmic and orbital arteries [7]. The orbital artery, from the external carotid forms the future middle meningeal artery which anastomoses initially with the primitive ophthalmic artery around the optic nerve sheath. Usually this connection is obliterated in later development however if persists, can form a dAVf at the level of the optic nerve sheath; as in this case (see fig 5 and 8) and previous case report [5].

Dural arteriovenous fistulas account for 10-15% of all intracranial vascular malformations [8]. Whilst their pathogenesis is controversial, recent studies and case reports of typical intracranial or spinal dAVfs suggest that they are acquired lesions initiated by dural or venous sinus thrombosis [9]. It is postulated that a clot precipitates venous congestion and hypertension therefore causing small capillaries to dilate, subsequently opening channels between small dural arteries and veins [8]. Various aetiologies causing or contributing to venous occlusion have been documented in the literature including trauma, surgery, infection and hypercoagulable states [10]. While this case is atypical given its rare extracranial location, the patient did have a number of risk factors increasing her risk for a thromboembolic event; AF, protein C deficiency and malignancy. She also suffered from COPD which would also increase her intravenous pressure. These may have contributed to the development of her dAVf.

AVFs can theoretically occur between any artery and vein and thus have been observed throughout the body with some locations more common than others e.g. the pulmonary circulation (incidence 2-3 per 100 000 people [11]). DAVfs should be considered as a separate entity or subtype of arteriovenous fistula because of their relationship to a separate tissue layer; the dura. Other types of intracranial AVFs have been observed such as non-galenic pial arteriorvenous fistulas (NGPAVF) [12].

Clinical & Imaging findings:

Intracranial and spinal dAVfs typically present with haemorrhage or symptoms due to venous occlusion / hypertension causing mass effect [8]. Their location dictates their signs and symptoms. In the present case arterial shunting to the SOV and its branches induced high intravenous pressure and dilatation leading to exophthalmos/proptosis [4]. Distal capillary dilatation around the eyeball itself can also result in glaucoma, chemosis and epiphora [4]. Orbital venous congestion can further induce retinal haemorrhages and motility disturbances due to swollen ocular muscles, direct nerve compression from cavernous sinus dilation and/or by arterial steal to nerve vascular supply [4]. Epiphora and raised intraocular pressure reported in both cases of optic nerve sheath dAVFs; however, our patient also presented with protosis, diplopia, visual failure and headaches. All these signs and symptoms are found commonly in patients with CCFs [4]. Therefore, it was reasonable that this more common alternative diagnosis was initially considered. Furthermore, if her bilateral cavernous aneurysms had been noted on the initial CTA, this may have lent support for a diagnosis of CCF as ruptured cavernous ICA aneurysm can be a precipitant for a spontaneous CCF [13]. While CCFs can also present with very similar radiological findings on CT and MRI to this case (ie proptosis, extraocular muscle enlargement, SOV dilation); typically a cavernous anomaly i.e. ipsilateral engorgement and/or associated skull fracture, is evident [13]. The fact that the cavernous sinus was essentially normal and that no bony trauma was evident should have contradicted this initial diagnosis (see figure 7). Orbital ultrasound was not considered in this case as its use has been superseded by other imaging modality to investigate orbital lesions. However, had it been undertaken it is likely that an enlarged SOV would have been detected and abnormal flow velocities/arterialised veins would be observable on Doppler flow studies [4].

Findings on non-contrast intracranial imaging modalities (CT, MRI) can be subtle and even with fine slice sequences defining orbital vascular lesions can be difficult. Therefore, vascular studies with catheter angiography of the internal and external carotid vessels is required and remains the gold standard for evaluating vascular anomalies throughout the neural axis. It is possible that other cases have gone under-diagnosed/misdiagnosed when catheter angiography was not undertaken or not available.

Treatment & Prognosis:

Treatment of a dAVf involves occlusion or disconnection of the fistula, specifically obliteration of the venous outlet [5]. The three broad treatment modalities available are embolization, radiation therapy, surgery or a combination of these [10]. They may also spontaneously occlude leading to regression or resolution of symptoms [10]. In this case, trans-arterial embolization or surgery were not viable options given the location and size of fistula on the optic sheath. Retrograde cannulation of the SOV was considered. This approach was first proposed by Hanneken et al [14] in 1989 in the setting of treatment of CCFs. However, it cannot be performed if the SOV is thrombosed, an issue in this case, and carries the risk of several serious complications including orbital

haemorrhage, infection, trochlear nerve damage; and thrombosis of ophthalmic veins leading to acute orbital congestion and vision loss, cerebral infarction and aggravation of symptoms [15]. Isolation and cannulation of the SOV is also technically challenging [15]. A number of authors [3,16] have described a successful inferior orbital vein approach but again in the setting of a CCF.

Radiotherapy has been utilised for treatment of intracranial dAVfs and for CCFs with some studies reporting >70% success rates [17]. However, radiation treatment is not recommended in patients with visual impairment or when risk to the optic nerve or chiasm is high [18]. Therefore, radiotherapy was not undertaken in this case.

In a case series of ten orbital arteriovenous shunts by de Keizer [4], nine of ten patients were managed conservatively. Of these, four patients had spontaneous occlusion of the fistula and a further three had improvement in their symptoms long-term. The length of follow up was not clearly specified but was greater than one year. One patient had progressive deterioration in their vision despite angiographic evidence of shunt occlusion. While in this case the pathology is different, some reassurance from this data regarding conservative management may be obtained.

The success of these approaches for treatment of an optic nerve sheath dAVf is difficult to quantify given that, to our knowledge, only one previous case has been reported in the literature and it was also conservatively managed [5]. Avoidance of permanent visual loss was the key concern for our patient. Therefore, in light of her medical co-morbidities and potential risks of treatment, a conservative approach with close surveillance was adopted.

Several classification schemes based on venous drainage exist to help stratify the risk of typical dAVf haemorrhage [1,19]; likelihood of symptomatic due to venous congestion [1,19]; and therefore recommendation for treatment [1,19]. Borden et al [1] propose arguably the most simplistic system of classification and Cognard et. al. the most complex [19](tables 3 and 4). Both have proven robust with a significant correlation between type and clinical presentation, as demonstrated by Davies et al [21]. However, given the location of the dAVf along the optic sheath described in this case, the venous drainage is primarily to the SOV and some other smaller orbital veins and not to a dural sinus, cortical, meningeal or subarachnoid vein as described by existing classification systems [1,19]. The lesion is therefore unable to be graded using these schemes.

Differential Diagnoses:

The main differential diagnosis for an optic nerve sheath dAVf include a CCF, orbital AVF or arteriovenous malformation (AVM). A CCF cannot be considered without communication with the cavernous sinus being established. Distinction between an orbital AVF or AVM can be more difficult. AVFs are felt to be acquired lesions generally with single or simple communication between an artery and vein without capillary vessels whereas AVMs are congenital and involve a complex arterio-venous communication or nidus [4]. As previously discussed, catheter angiography is the modality of choice for detailed evaluation of orbital vascular structures. Other mass lesions including an orbital tumour, osteoma, fibrous dysplasia, frontal sinus mucocele or encephalocele causing proptosis; can be excluded often by plain radiograph but certainly by cross-sectional imaging.

A cavernous sinus thrombosis may present with chemosis, proptosis and cranial nerve 3-6 deficit, therefore localizing the pathology. Most patients will have a headache and history of infection typically involving the midface. Filling defects /absence of flow through the region together with evidence of facial infection are considered diagnostic [22].

An inflammatory condition of the eye such as peri-orbital cellulitis, could produce swelling and erythema of the region and surrounding soft tissues. However, radiological changes are extra-ocular.

Thyroid ophthalmopathy /orbitopathy can cause thickening of the extraocular muscles usually with tendon sparing. However, epiphora and protosis typically affects the eyes symmetrically and other cutaneous manifestations are often observed [23]. Clinical examination and hormone studies would exclude this differential.

Conclusion:

This case highlights the importance of high quality catheter angiography in helping make the final diagnosis and should be considered in all patients with an arteriovenous fistula. Unfortunately, the existing classification systems for dAVfs are not clearly applicable in this case making prognostication difficult. Given that only one other reported case exists within the literature to date; more information and research is required to better understand the natural history of this condition and for treatment recommendations. This case also demonstrates how non-specific radiological signs biased by clinical suspicion can lead to a misdiagnosis when uncommon and infrequent entities are present.

TEACHING POINT

Optic nerve sheath dural arteriorvenous fistulas are acquired, atypical vascular malformations that can present with symptoms similar to carotid-cavernous fistulas and are often initially misdiagnosed. While they can be assessed with Computer Tomography (CT) and Magnetic Reasonance Imaging (MRI); catheter angiography is the gold standard for evaluating their vascular anatomy and can help guide treatment rationalisation.

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FIGURES

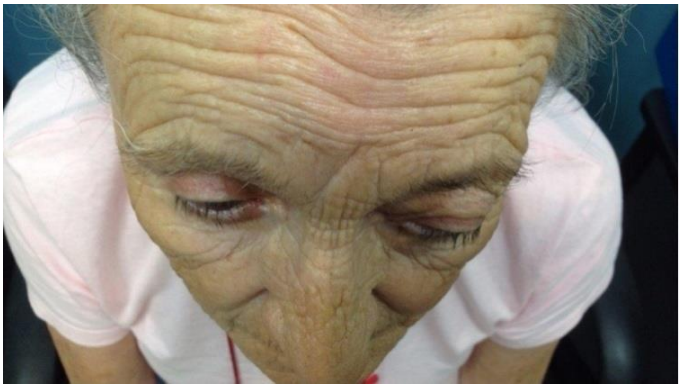

Figure 1: A 78 year old female presents with progressive diplopia, proptosis, epiphora, decreased visual acuity in the left eye and headaches secondary to a left optic nerve sheath dural arteriovenous fistula
Clinical photo
FINDINGS: Left eye proptosis

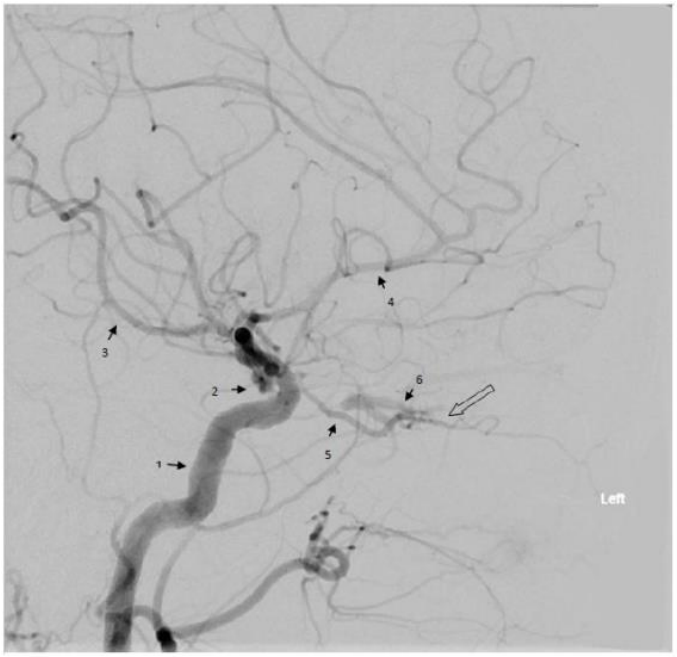

Figure 2: A 78 year old female presents with progressive diplopia, proptosis, epiphora, decreased visual acuity in the left eye and headaches secondary to a left optic nerve sheath dural arteriovenous fistula.
Angiography, Interventional
FINDINGS: Left common carotid artery angiogram (sagittal view) demonstrates contrast predominantly through the left internal carotid with arteriovenous shunting from meningeal branches of left ophthalmic artery into partially thrombosed, dilated superior ophthalmic vein - early phase (large arrow)
1. Internal carotid artery 2. Cavernous internal carotid aneurysm 3. Anterior cerebral artery 4. Middle cerebral artery 5. Ophthalmic artery 6. Superior ophthalmic vein
TECHNIQUE: Siemens Axiom-Artis, left common carotid digital subtraction angiogram. 10ml Omnipaque 300

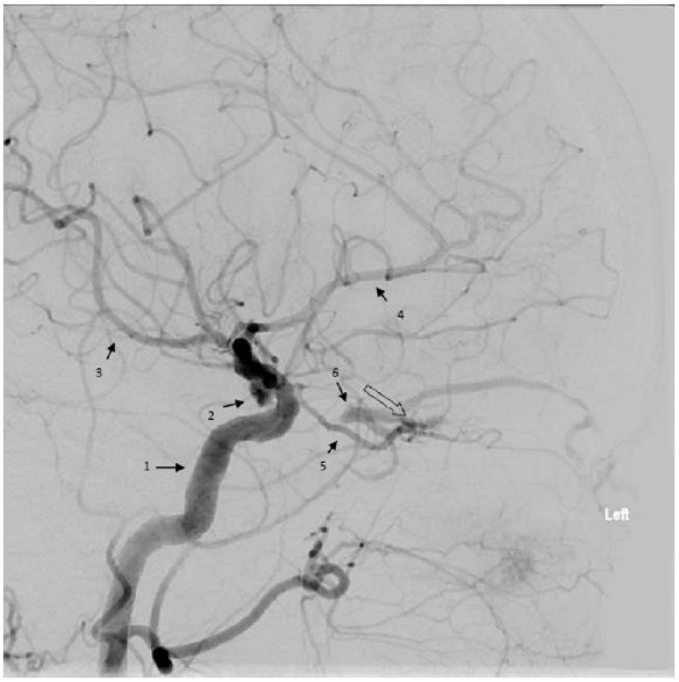

Figure 3: A 78 year old female presents with progressive diplopia, proptosis, epiphora, decreased visual acuity in the left eye and headaches secondary to a left optic nerve sheath dural arteriovenous fistula.
Angiography, Interventional
FINDINGS: Left common carotid artery angiogram (sagittal view) demonstrates contrast predominantly through the left internal carotid with arteriovenous shunting from meningeal branches of left ophthalmic artery into partially thrombosed, dilated superior ophthalmic vein and cranial accessory vein - mid phase (large arrow)
1. Internal carotid artery 2. Cavernous internal carotid aneurysm 3. Anterior cerebral artery 4. Middle cerebral artery 5. Ophthalmic artery 6. Superior ophthalmic vein
TECHNIQUE: Siemens Axiom-Artis, left common carotid digital subtraction angiogram. 10ml Omnipaque 300

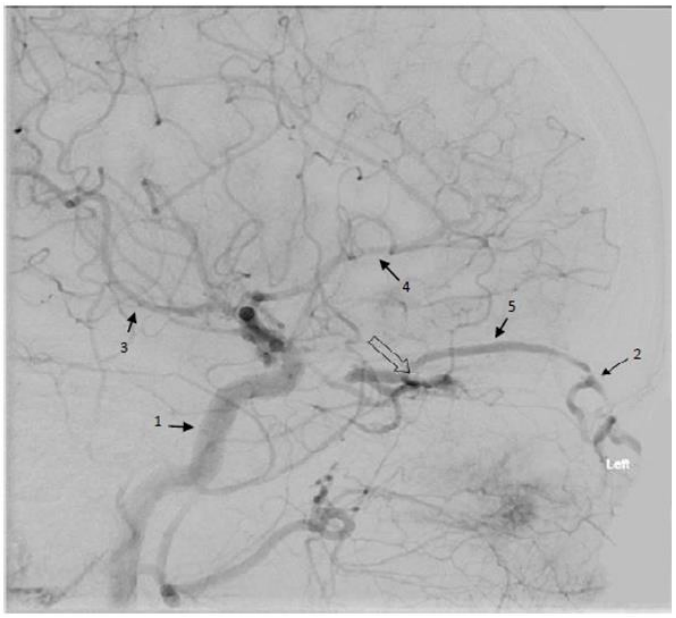

Figure 4: A 78 year old female presents with progressive diplopia, proptosis, epiphora, decreased visual acuity in the left eye and headaches secondary to a left optic nerve sheath dural arteriovenous fistula.
Angiography, Interventional
FINDINGS: Left common carotid artery angiogram (sagittal view) demonstrates contrast predominantly through the left internal carotid with arteriovenous shunting from left ophthalmic artery into partially thrombosed, dilated superior ophthalmic vein and cranial accessory vein - late phase (large arrow)
1. Internal carotid artery 2. Venous stricture in superior ophthalmic vein 3. Anterior cerebral artery 4. Middle cerebral artery 5. Superior ophthalmic vein
TECHNIQUE: Siemens Axiom-Artis, left common carotid digital subtraction angiogram. 10ml Omnipaque 300

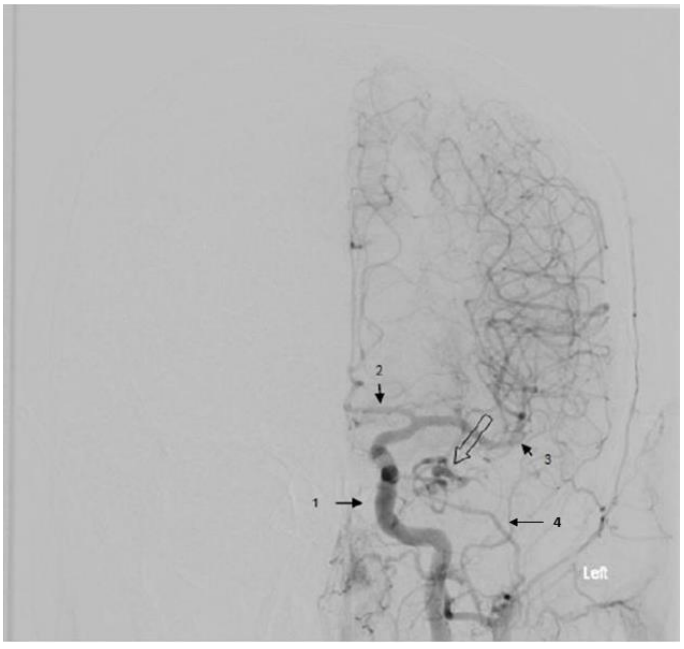

Figure 5: A 78 year old female presents with progressive diplopia, proptosis, epiphora, decreased visual acuity in the left eye and headaches secondary to a left optic nerve sheath dural arteriovenous fistula.
Angiography, Interventional
FINDINGS: Left common carotid artery angiogram (coronal view) demonstrates arteriovenous shunting from the left middle meningeal artery into partially thrombosed superior ophthalmic vein and cranial accessory vein - mid phase (large arrow).
1. Internal carotid artery 2. Anterior cerebral artery 3. Middle cerebral artery 4. Middle meningeal artery
TECHNIQUE: Siemens Axiom-Artis, left common carotid digital subtraction angiogram. 10ml Omnipaque 300

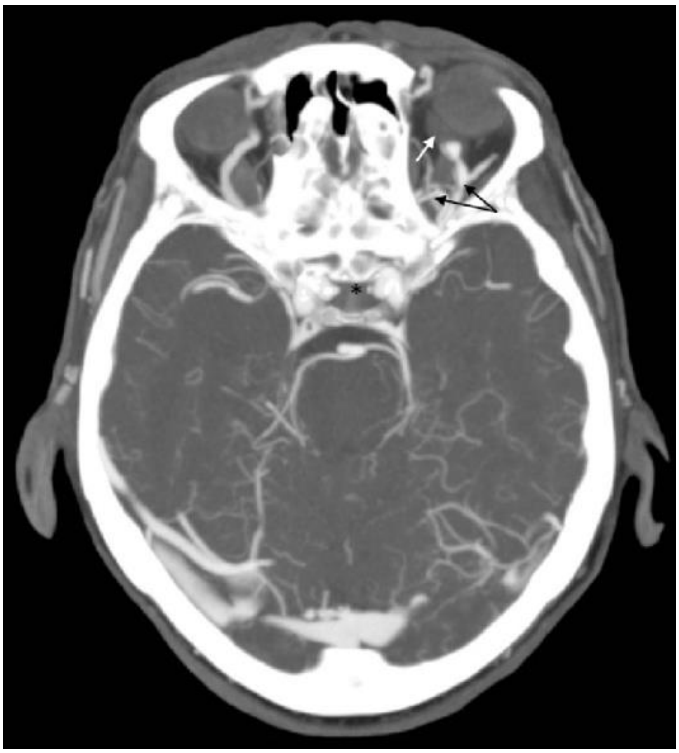

Figure 6: A 78 year old female presents with progressive diplopia, proptosis, epiphora, decreased visual acuity in the left eye and headaches secondary to a left optic nerve sheath dural arteriovenous fistula.
Computer Tomography
FINDINGS: Axial CT-A demonstrates abnormal vessels left orbit suggestive of a vascular anomaly
* bilateral cavernous internal carotid aneursyms, black arrows: enhancing dilated orbital vessels and superior ophthalmic vein with small area of stricture, white arrow: enlarged intra-conal retrobulbar soft tissue
TECHNIQUE: Toshiba Activion 16, axial CT-A 150mAs. 120 kv. 4.1mm slice thickness. 120ml of Omnipaque 240 intravenous contrast.

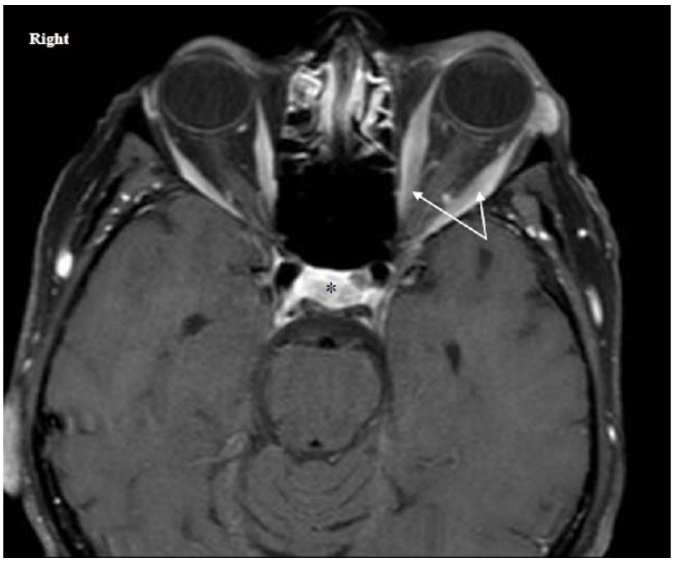

Figure 7: A 78 year old female presents with progressive diplopia, proptosis, epiphora, decreased visual acuity in the left eye and headaches secondary to a left optic nerve sheath dural arteriovenous fistula.
Magnetic Resonance Imaging
FINDINGS: Axial MRI T1W FSAT post contrast image demonstrating fat stranding /engorgement of recti secondary to venous congestion from dAVf (arrows), * early filling left intracavernous internal carotid aneurysm but otherwise normal cavernous sinus
TECHNIQUE: Philips Integra 1.5 Tesla, Post Contrast T1W FSAT, TR/TE: 588.516/12, 3mm slice thickness. 12ml Gadolinium intravenous contrast.

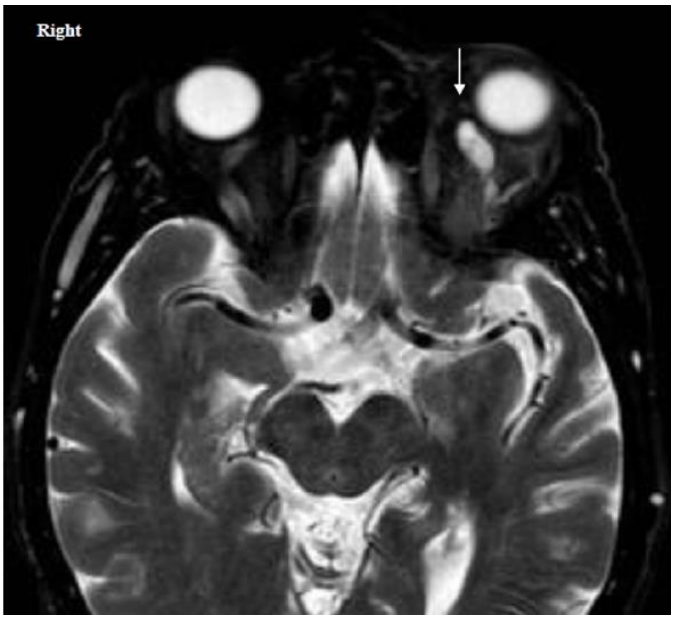

Figure 9: A 78 year old female presents with progressive diplopia, proptosis, epiphora, decreased visual acuity in the left eye and headaches secondary to a left optic nerve sheath dural arteriovenous fistula.
Magnetic Resonance Imaging
FINDINGS: Axial MRI T2W FSAT post contrast images demonstrating flow void in a dilated left superior ophthalmic vein (arrow)
TECHNIQUE: T2W FSAT TR/TE: 3216.349/90, 3.2mm thickness. 12ml Gadolinium intravenous contrast.

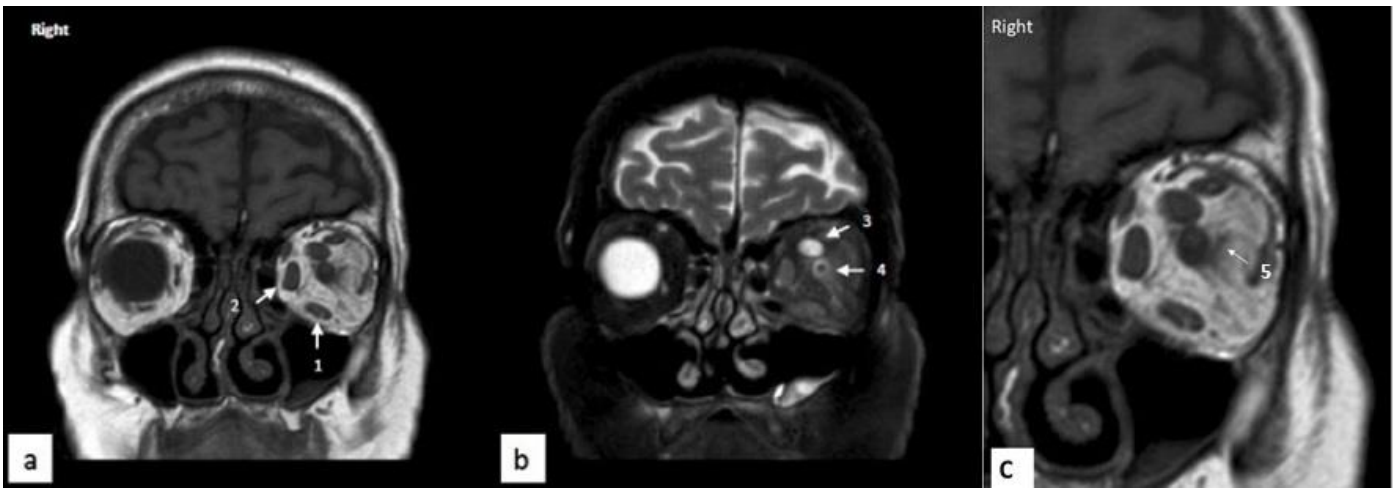

Figure 8: A 78 year old female presents with progressive diplopia, proptosis, epiphora, decreased visual acuity in the left eye and headaches secondary to a left optic nerve sheath dural arteriovenous fistula.
Magnetic Resonance Imaging
FINDINGS: Coronal MRI (a,c)T1W SE post contrast and (b) T2W FSAT images demonstrating engorged recti and vessels in left orbit
1. Enlarged inferior rectus muscle 2. Enlarged medial rectus muscle 3. Dilated superior ophthalmic vein 4. Optic nerve 5. Abnormal arterial network around optic nerve
TECHNIQUE: Philips Integra 1.5 Tesla (a) T1W SE+ C TR/TE: 522.582/12, 3.2mm slice thickness (b) T2W FSAT TR/TE: 3216.349/90, 3.2mm thickness. 12ml Gadolinium intravenous contrast.

Aetiology	Acquired
Incidence	Unknown but rare (one previously reported optic nerve sheath dural arteriovenous fistula (dAVF) in literature)
Gender ratio	Unknown
Age Predilection	Unknown. Both reported cases patient 60 years or older
Risk factors	Unknown but likely similar to typical dAVFs ; trauma, surgery, infection, hypercoagulable state
Treatment	Closure of fistula; Embolization, radiation or surgical disconnection. Can occur spontaneously
Prognosis	Uncertain. Venous congestion is more likely to be symptomatic. Increased risk of visual failure with raised intra-ocular pressure
Findings on imaging	Absence of a mass lesion. Dilated orbital vessels on Computer Tomography Angiography (CTA) and Magnetic Resonance Angiography (MRA). Swelling of extra-ocular muscles on T1 weighted imaging (T1WI). Fistula between dural artery and vein not to a venous or dural sinus, cortical / meningeal or subarachnoid vein on internal and external carotid angiogram. Absence of cavernous sinus abnormality. Likely to observe dilated superior ophthalmic vein on orbital ultrasound and possible arterialised veins with Doppler studies

Table 1: Summary table for Optic Nerve Sheath Dural Arteriovenous Fistula.

Type	Description
I	Drain directly into dural venous sinuses or meningeal veins
II	Drain into dural sinuses or meningeal veins and also have retrograde drainage into subarachnoid veins
III	Malformations drain into subarachnoid veins and do not have dural sinus or meningeal venous drainage

Table 2: Borden Classification System for Spinal and Cranial Dural Arteriorvenous Fistulous Malformations [1].

Type	Description
I	Located in the main sinus / venous drainage into dural venous sinus, with antegrade flow
II	Located in the main sinus / venous drainage into dural venous sinus with retrograde flow (IIa), antegrade flow into cortical veins (IIb), or both (IIa + b)
III	Direct cortical venous drainage without venous ectasia
IV	Direct cortical venous drainage with venous ectasia;
V	Spinal perimedullary venous drainage

Table 3: Cognard et al Classification System for Spinal and Cranial Dural Arteriovenous Fistulas [16,19]

	Radiography	US	CT/CTA	MRI/A	Angiography
Optic nerve sheath dAVF		-Enlarged superior ophthalmic vein (SOV) -likely abnormal Doppler flow velocities	Non-specific May show; -Enlarged or abnormal orbital vessels -Engorged extra-ocular muscles -Globe proptosis -Haemorrhage	Non-specific -Same as CTA -Fat stranding and oedema in extra-ocular muscles on T1WI -Flow voids in SOV on T1 and T2 weighted imaging	Communication between dural artery and vein without capillary vessels
Carotid-cavernous fistula	Evidence of trauma / facial fractures	-Enlarged SOV -Abnormal Doppler flow velocities	-Same as dAVF -Engorgement of cavernous sinus -Evidence of trauma/skull fracture -Cavernous internal carotid artery aneurysm	-Same as dAVF -May show communication with cavernous sinus -Cavernous internal carotid artery aneurysm	Communication between the internal or external carotid artery or their branches and the cavernous sinus
Orbital Arteriovenous Malformation		-Abnormal cluster of vessels -Abnormal Doppler flow velocities	-Same as dAVF -Calcification	-Same as dAVF -May demonstrate nidus -Calcification (susceptibility weighted imaging SWI)	Communication between an artery and vein via a complex or nidus without a capillary system
Orbital tumour	Distortion or destruction of bony orbit	Discrete mass	-Intra-orbital mass -Bony involvement -Soft tissue involvement -Abnormal or increased vasculature of lesion -Other lesions/ metastasis -Proptosis -Neural compression	-Same as CT findings but more defined	-Distortion of orbital vessels -increased vasculature to lesion
Osteoma	Calcified growth, usually involving paranasal sinuses		-Well circumscribed calcified growth usually involving paranasal sinuses -Mass effect	-Well circumscribed calcified growth usually involving paranasal sinuses -No or low signal change	
Fibrous Dysplasia	-Smooth expansion of affected bone -Well circumscribed -No periosteal reaction -Rind sign		-Bony expansion -Ground glass matrix -Mass effect -Proptosis	-Bony extra-conal lesion -Mass effect -Proptosis -Heterogenous signal change on T1 and T2 sequences and heterogenous enhancement post contrast	
Frontal sinus mucocele / encephalocele	Opacification and expansion with possible defect in sinus		-Opacification of sinus -Bony defect -Parenchymal involvement -May have peripheral enhancement -Mass effect -Proptosis	-Extra-orbital lesion -Parenchymal involvement -Heterogenous signal change -May have peripheral enhancement -Proptosis	
Cavernous sinus thrombosis		-Enlarged orbital veins	-Hyperdense cavernous sinus -Reduced or absent flow through region -dilated orbital veins -Proptosis -Evidence of midface infection i.e. sinusitis or periorbital soft tissue swelling	-Absence of flow void in sinus -May have post contrast enhancement -Dilation or orbital veins -Extra-ocular muscle oedema -Proptosis -Evidence of midface infection	-Absence of venous flow through region -dilated orbital veins
Orbital cellulitis		Periorbital soft tissue oedema	Periorbital soft tissue oedema and fat stranding	-Periorbital soft tissue oedema and fat stranding -Contrast enhancement of surrounding tissues	
Thyroid Ophthalmopathy/ Orbitopathy			-Enlargement of recti with sparing of muscle tendons 'coke bottle sign' -Typically, bilateral and symmetrical -proptosis -increased retro-ocular orbital fat	-Enlargement of recti with sparing of muscle tendons -Typically, bilateral and symmetrical -Proptosis -increased retro-ocular orbital fat -increased signal recti T2 -post contrast enhancement of recti	

Table 4: Differential diagnosis table for Optic Nerve Sheath Dural Arteriovenous Fistula.

ABBREVIATIONS

AF = Atrial fibrillation
AVM = Arteriovenous Malformation
CCF = carotid-cavernous fistula
COPD = Chronic Obstructive Pulmonary Disease
CT = Computer tomography
CTA = CT angiogram
dAVF = dural arteriovenous fistula
FSAT = Fat suppression sequence
IOP = intraocular pressure
MRI = Magnetic Resonance Imaging
SE = Spin Echo
SOV = Superior Ophthalmic Vein
T1W = T1 weighted image
T2W = T2 weighted image
US = Ultrasound

KEYWORDS

Dural Arteriovenous fistula; Optic nerve sheath; Carotid Cavernous fistula; Angiography

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