

# Choroidal Detachment and Dural Carotid-Cavernous Sinus Fistula

## —Case Report—

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### Abstract

A 71-year-old female presented with the rare complication of choroidal detachment after endovascular treatment of a dural carotid-cavernous sinus fistula. Since the residual arteriovenous shunt flow was minimal and intraocular pressure was normal, the choroidal detachment was treated conservatively and disappeared within one month. The possibility of choroidal detachment during the clinical course of a dural carotid-cavernous sinus fistula should be recognized.

Key words: choroidal detachment, dural carotid-cavernous sinus fistula, embolization, intraocular pressure

### Introduction

Choroidal detachment is not a distinct clinical entity but rather a state of accumulation of fluid in the suprachoroidal space.<sup>1)</sup> Therefore, a number of different conditions may cause a clinical picture of choroidal detachment. These conditions have been classified as follows: a) *inflammatory*, which may result from trauma, intraocular surgery, scleritis, cryocoagulation or photocoagulation, or chronic uveitis; b) *hydrostatic*, including dural carotid-cavernous sinus fistula (CCF), hypotony, wound leak, and abnormally thick sclera; and c) *idiopathic*.<sup>1)</sup> Choroidal detachment is most commonly associated with inflammatory conditions.

Choroidal detachment in association with a dural CCF is rare. Ocular manifestations of dural CCFs include proptosis, chemosis, conjunctival injection, diplopia due to paresis of the extraocular nerves and/or swelling of the extraocular muscles, ptosis, pain, and visual decline. Visual decline is usually associated with ischemic retinopathy and/or retinal hemorrhage due to central retinal vein occlusion. However, choroidal detachment may also cause visual decline. Fifteen such cases have been reported in the ophthalmological literature, but none in the neuroradiological or neurosurgical

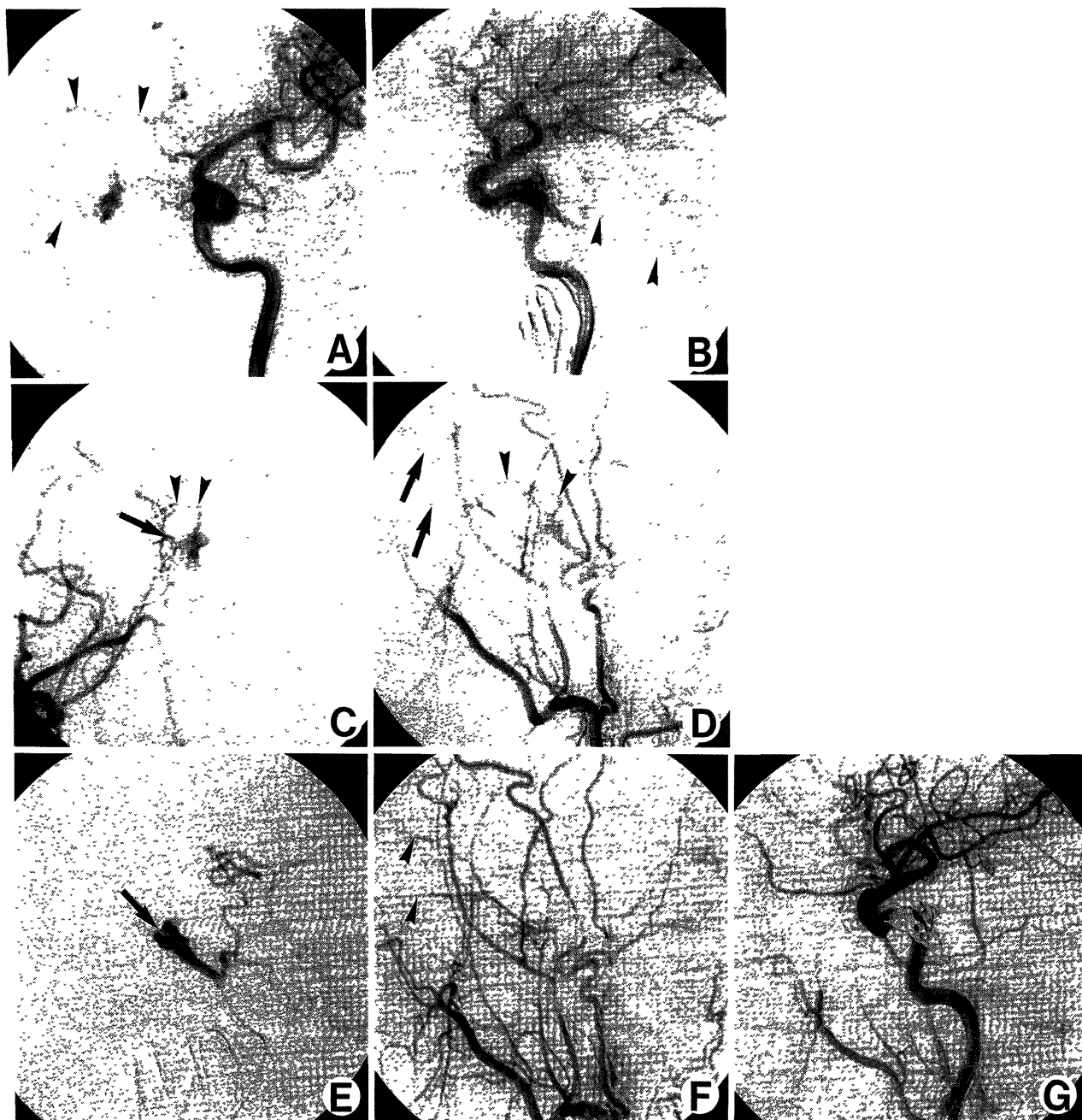
literature.<sup>2,5-8,10,13,14)</sup> We present a case of visual decline due to choroidal detachment associated with dural CCF to further the recognition of this ocular complication.

### Case Report

A 71-year-old female experienced chemosis, proptosis, and abducens nerve palsy on the right in September 1994. Angiography at another hospital revealed a right dural CCF. The patient's medical history was otherwise unremarkable, except for uterine cancer, which was managed by local irradiation in 1990. In November 1994, the patient twice underwent transarterial particulate embolization. Her ocular signs improved gradually, but in early January 1995, the proptosis and conjunctival injection on the right side deteriorated.

The patient was referred to us in March 1995. She had mild chemosis, mild proptosis, and a corkscrew-like vein in the right eye. Visual acuity was 0.6 (1.2) on the right side and 0.4 (1.0) on the left, and the intraocular pressure was 13 mmHg on the right side and 8 mmHg on the left. Funduscopic examination revealed venous congestion and moderate retinal hemorrhage in the right eye.

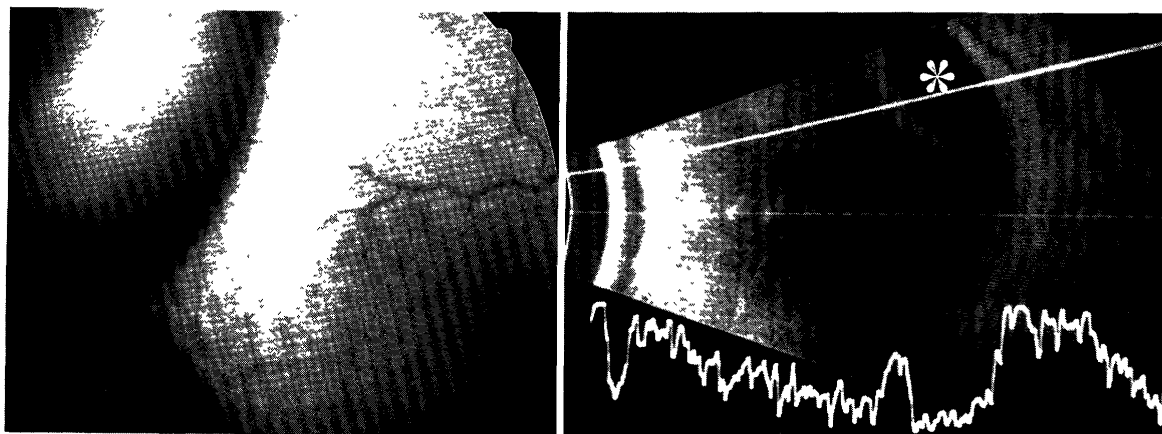
Left carotid angiography revealed that the left meningohypophyseal trunk fed the right dural CCF. The right internal carotid artery contributed minimally to the CCF. The right middle meningeal



**Fig. 1** Left internal carotid angiograms, anteroposterior view (A) and lateral view (B), performed in March 1995, showing the dural carotid-cavernous sinus fistula (CCF) at the right cavernous sinus, fed by the left meningohypophyseal trunk, and with cortical venous drainage (arrowheads) in the posterior fossa. Right external carotid angiograms, anteroposterior view (C) and lateral view (D), showing the dural CCF at the right cavernous sinus and the venous drainage to the superior and inferior orbital veins (arrows) and cortical veins (arrowheads). Right cavernous sinogram, lateral view (E), showing the cortical venous drainage and the tip of the microcatheter (arrow). Right external carotid angiogram, lateral view (F), and left internal carotid angiogram, lateral view (G), after transvenous occlusion of the right posterior cavernous sinus, showing the cortical drainage is almost obliterated, but a small shunt from the anterior cavernous sinus to the orbital veins persists (arrowheads).

artery, the accessory meningeal artery, and the artery of the foramen rotundum also supplied the CCF. The shunt was located in the right cavernous sinus,

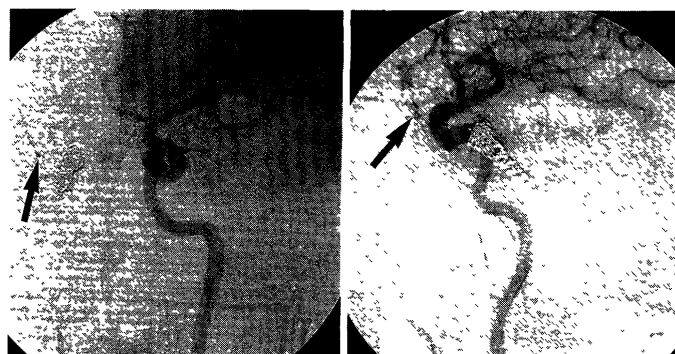
predominantly in the posterior portion, which drained to the cortical veins and to the right superior and inferior orbital veins (Fig. 1A-D).



**Fig. 2** Right funduscopy (left), performed in December 1995, disclosing choroidal detachment at the temporal half of the right eye. Echogram (right) also disclosing the choroidal detachment and fluid collection (asterisk) in the suprachoroidal space.

Transfemoral transvenous embolization through the right inferior petrosal sinus to the right posterior cavernous sinus was performed using interlocking detachable coils (Target Therapeutics, Inc., Fremont, Cal., U.S.A.) (Fig. 1E). The tip of the microcatheter could not be introduced into the anterior cavernous sinus. The right posterior cavernous sinus was packed with coils. Although a small shunt remained in the anterior cavernous sinus draining to the orbital veins, the cortical drainage was almost obliterated (Fig. 1F,G). In June 1995, follow-up angiography revealed no cortical drainage. However, the right accessory meningeal artery was feeding the small shunt at the right anterior cavernous sinus. Following transarterial embolization with polyvinyl alcohol particles, the dural CCF disappeared completely.

In December 1995, the patient complained of blurred vision in the right eye. There was no chemosis or proptosis, but mild corkscrew-like veins were noted in the right eye. Visual acuity was 0.6 (1.0) on the right and 0.8 (1.0) on the left, with intraocular pressure of 9 mmHg on the right and 10 mmHg on the left. Ophthalmological examination revealed choroidal detachment at the temporal half of the right fundus and minimal retinal hemorrhage (Fig. 2 left). Echography also revealed choroidal detachment and fluid collection at this location (Fig. 2 right). Angiography revealed that only the left meningohypophyseal artery contributed minimally to the right dural CCF (Fig. 3). The choroidal detachment was not extensive and did not affect visual acuity, and the arteriovenous shunt was minimal, so she was treated conservatively. Follow-up examination one month later revealed that the choroidal detachment had spontaneously improved. The most recent



**Fig. 3** Left internal carotid angiograms, anteroposterior view (left) and lateral view (right), performed in January 1996, showing minimal arteriovenous shunt flow draining to the right orbital veins (arrow).

follow-up examination in June 1996 found there was a minimal presence of corkscrew-like veins in the right eye, and no choroidal detachment. Visual acuity was 0.5 (1.0) on the right and 0.4 (1.0) on the left. Intraocular pressure was 12 mmHg bilaterally.

## Discussion

Choroidal detachment associated with a dural CCF has previously been reported only in the ophthalmological literature. Our review revealed 16 reported cases, including ours, in five males and nine females aged 43 to 87 years (mean 70 years)<sup>2,5-8,10,13,14</sup> (Table 1). The symptomatic side of the dural CCF was the right in six cases, left in five, and bilateral in three cases. Choroidal detachment occurred on the right in six cases, on the left in six, and bilaterally in two cases. Choroidal detachment was always observed

**Table 1** Cases of choroidal detachment (CD) associated with dural carotid-cavernous sinus fistula (CCF)

Case No.	Author (Year)	Age/ Sex	Side of CD	Symptom of dural CCF	IOP (mmHg)		Treatment of dural CCF	Treatment of CD or increased IOP	Outcome
					Rt	Lt			
1	Harbison et al. (1978) <sup>8)</sup>	70/M	bil	bil	46	46	none	orbital decompression, acetazolamide	GR
2	Klein et al. (1978) <sup>8)</sup>	76/F	lt	lt	16	16	none	mydriatic	GR
3	Mazzeo et al. (1985) <sup>10)</sup>	78/F	bil	rt	30	12	none	medical	GR
4		43/M	lt	lt	N	31	carotid compression	none	GR
5		59/M	lt	bil	N	N	general steroid	local steroid	GR
6		59/M	rt	bil	43	33	none	$\beta$ -blocker, acetazolamide	GR
7	Jorgensen and Guthoff (1988) <sup>6)</sup>	83/F	lt	lt	12	14	none	none	GR
8		76/M	rt	rt	32	12	none	none	GR
9		80/?	?	?	?	?	?	?	?
10		72/?	?	?	?	?	?	?	?
11	Fiore et al. (1990) <sup>2)</sup>	66/F	rt	rt	28	19	embolization	miotic, $\beta$ -blocker	poor
12		73/F	lt	lt	10	20	none	$\beta$ -blocker, mydriatic, acetazolamide, steroid, canthotomy, iridotomy	GR
13	Yamada et al. (1991) <sup>14)</sup>	87/F	lt	lt	11	18	carotid compression	none	GR
14	Kitazawa et al. (1994) <sup>7)</sup>	77/F	rt	rt	19	15	carotid compression	$\beta$ -blocker, acetazolamide	GR
15	Shirataki and Shimakawa (1994) <sup>13)</sup>	52/F	rt	rt	20	16	embolization	none	GR
16	Present case	71/F	rt	rt	9	10	embolization	none	GR

GR: good recovery, IOP: intraocular pressure when CD occurred, N: normal.

on the same side as the dural CCF. Elevated intraocular pressure ( $\geq 20$  mmHg) was observed in eight patients whereas intraocular pressure was  $\leq 18$  mmHg in four patients. Therefore, elevated intraocular pressure was not always observed in cases of choroidal detachment associated with a dural CCF. The dural CCFs were treated by carotid compression in three cases, embolization in three, steroid administration in one, and no treatment was given in seven cases. Choroidal detachment and/or increased intraocular pressure was treated locally or by general medical treatment in eight cases, by orbital decompression, canthotomy, and iridotomy in two, and six patients received no treatment. Good recovery was observed in all patients but one, who had decreased visual acuity.

Under normal conditions, intraocular pressure, intracapsular blood pressure in the choroid, and colloid osmotic pressure are balanced.<sup>5)</sup> The intraocular pressure may remain normal in cases of choroidal detachment caused by inflammatory conditions, but is often reduced because of lowered aqueous humor production. However, intraocular pressure is frequently increased in cases of dural CCF. Choroidal detachment associated with a dural CCF may occur due to increased blood pressure in the orbital veins and choriocapillaries of the choroid. Arteriovenous shunting and venous thrombosis cause venous

hypertension and increased intracapsular pressure in the choroid. This induces transudation into the suprachoroidal space, resulting in choroidal separation.

Perfusion pressure in the eye (the arteriovenous pressure gradient) decreases due to increased venous pressure caused by arteriovenous shunting. Increased intraocular pressure and decreased perfusion pressure results in ischemia of the retina and the choroid.<sup>11)</sup> If the blood flow falls below the level required to satisfy the local metabolic demands of the retina, the retina becomes hypoxic and functional failure begins.<sup>11)</sup> When a patient with dural CCF complains of visual decline, the possibilities of ischemic retinopathy, central retinal vein occlusion,<sup>9)</sup> and choroidal detachment must be considered. The prognosis for choroidal detachment is better than that of the former two clinical situations, so appropriate diagnosis and treatment are essential.

The development of venous thrombosis in the superior and/or inferior orbital veins is well known to result in a temporary worsening of ocular signs, which then gradually improve. This is called "paradoxical worsening" of the ocular signs,<sup>12)</sup> and has resulted from central retinal vein occlusion in some cases,<sup>9)</sup> but choroidal detachment may also be a cause. We believe that choroidal detachment associated with a dural CCF occurs more frequently than

expected from the frequency of reports in the literature. Underreporting may result from failure of ocular signs to manifest abnormally at the clinical level, or from poor recognition of this clinical condition.

Since many different conditions may create a clinical picture of choroidal detachment, treatment should be directed to the specific pathogenic condition. Treatment of choroidal detachment associated with a dural CCF should be directed toward the causative arteriovenous fistula. We treated our patient conservatively because of the minimal shunt flow and the relative mildness of the clinical symptoms. However, when choroidal detachment is extensive, whether intraocular pressure is elevated or not, aggressive treatment to reduce venous pressure in the orbital veins is necessary. Medical treatment to reduce the intraocular pressure is necessary when intraocular pressure is high enough to cause deterioration of choroidal and retinal circulation. Dural CCFs require conservative treatment, manual carotid-jugular compression, or endovascular surgery.<sup>4)</sup> When the clinical condition requires emergency treatment, endovascular surgery is the treatment of choice.<sup>3)</sup>

Interventionalists must recognize the possibility of choroidal detachment in the management of dural CCFs. Whatever treatment is undertaken, close cooperation between neurointerventionalists and ophthalmologists is essential.

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