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Clinical ophthalmic evaluation of post-traumatic carotid-cavernous fistula

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创伤后颈内动脉海绵窦瘘患者眼部临床评估

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摘要

目的:明确创伤后颈内动脉海绵窦瘘(CCF)患者视力预后的临床及眼底预测指标,并报告诊断延迟、血管造影闭塞率与视功能预后之间的关联。

方法:本回顾性病例系列研究纳入 2024 年 1 月至 2025 年 10 月期间经数字减影血管造影确诊的创伤后颈内动脉海绵窦瘘患者。全套眼科检查包括视力、眼压(IOP)、眼底镜检查、相对性传入性瞳孔障碍(RAPD)和色觉。对临床表现模式、眼底镜下表现及视觉预后进行系统记录和描述性分析。

结果:共纳入 8 例患者,男 5 例、女 3 例,平均年龄 28.5±13.2 岁;其中 7 例(88%)由机动车事故致伤。患者平均诊断延迟时间为 2.6±1.6 mo,7 例(88%)就诊时病程已超过 6 wk,5 例(63%)继发青光眼;尽管均为单侧瘘管,仍有 3 例(38%)出现双侧体征与体征。瘘管分型以 A 型直接型 CCF 为主(7 例,88%)。眼底镜下表现分为三类:视盘正常(2 例,25%)、视盘水肿(3 例,38%)、视神经萎缩(3 例,38%)。仅 3 例(38%)实现血管造影下瘘口完全闭塞,但患者视力预后与基线眼底表现的相关性远高于瘘口闭塞程度。3 例视神经萎缩患者视力预后差(末次视力从无光感至 20/30);2 例眼底无异常者视力恢复理想(末次视力达 20/20 至 20/25);3 例视盘水肿患者预后因疾病严重程度而异,从完全恢复到持续性视力丧失不等。1 例患者行部分栓塞术后,对侧眼视力下降(从 20/20 降至 20/150)。

结论:本研究中 88%患者就诊时已超出 6 wk 关键治疗窗口期,在医疗资源有限地区,创伤后 CCF 的早期识别仍是一大挑战。患者视力预后主要由基线眼底镜所见相关,而非血管造影瘘口闭塞程度。已发生视神经萎缩的患者(38%),无论手术是否成功,视功能恢复极为有限;而视盘结构完好者预后更佳。在缺乏先进影像学检查手段的情况下,系统的神经眼科检查——尤其是 RAPD 检测和眼底镜检查——可实现早期诊断和预后评估。

关键词:颈内动脉海绵窦瘘;创伤后;诊断延迟;眼底检查;预后因素;视神经萎缩;继发性青光眼

Abstract

• AIM: To identify clinical and fundoscopic predictors of visual outcomes in post-traumatic carotid-cavernous fistula (CCF) and report the relationship between diagnostic delay, angiographic closure rates, and functional prognosis.

• METHODS: This retrospective case series reports patients with post-traumatic CCF confirmed by digital subtraction angiography between January 2024 and October 2025. Comprehensive ophthalmic evaluation included visual acuity, intraocular pressure (IOP), fundoscopy, relative afferent pupillary defect (RAPD), and color vision. Clinical presentation patterns, fundoscopic findings, and visual outcomes were systematically documented and analyzed descriptively.

• RESULTS: Eight patients (5 males, 3 females, mean

age 28.5±13.2 y) were included; 7 cases (88%) from motor vehicle accidents. Mean diagnostic delay was 2.6±1.6 mo; 7 cases (88%) presented beyond 6 wk. Secondary glaucoma developed in 5 cases (63%). Bilateral signs and symptoms occurred in 3 cases (38%) despite unilateral fistulas. Type A direct CCF predominated (7 cases, 88%). Fundoscopic examination revealed three presentations: normal disc (2 cases, 25%), optic disc edema (3 cases, 38%), and optic atrophy (3 cases, 38%). Despite complete angiographic closure in only 3 cases (38%), visual outcomes seemed more closely associated with baseline fundoscopic findings than closure rates. Patients with optic atrophy (n=3) demonstrated poor visual prognosis (final visual acuity ranging no light perception to 20/30), while those with normal fundus (n=2) achieved excellent recovery (final visual acuity 20/20-20/25). Patients with disc edema (n=3) showed variable outcomes depending on disease severity, ranging from complete recovery to persistent visual loss. One patient experienced contralateral visual decline (20/20 to 20/150) following partial embolization.

• CONCLUSION: With 88% of patients presenting beyond the critical six-week therapeutic window, early recognition of post-traumatic CCF remains a major challenge in resource-limited settings. Visual outcomes corresponded with baseline fundoscopic findings rather than angiographic closure rates. Patients with established optic atrophy (38%) demonstrated minimal visual recovery regardless of treatment success, while those with preserved disc architecture achieved favorable outcomes. In settings where advanced imaging is unavailable, systematic neuro-ophthalmic examination—particularly RAPD testing and fundoscopy—enables both early diagnosis and prognostic assessment.

• KEYWORDS: carotid-cavernous fistula; post-traumatic; diagnostic delay; fundoscopy; prognostic factors; optic atrophy; secondary glaucoma
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INTRODUCTION

Carotid-cavernous fistula (CCF) is defined as any abnormal arteriovenous connection between the carotid artery system and the cavernous venous system[1]. These abnormal arteriovenous malformations can occur either following trauma or spontaneously, representing a rare but potentially sight- and life-threatening vascular complication[2]. While CCF is reported to occur in only 0.2% of patients with cranial trauma, its clinical significance is profound, and post-traumatic CCF, particularly following head trauma, poses unique diagnostic challenges that can lead to delayed recognition and treatment, with serious implications for patient outcomes[3].

CCF is classified into direct and indirect types based on the anatomical origin of the fistula. Direct CCFs arise directly from the carotid artery itself, while indirect CCFs originate from branch vessels of the carotid artery[4]. The cavernous sinus, a structure composed of a group of thin-walled veins through which the carotid artery and its branches pass, becomes the site of pathological arteriovenous shunting. This abnormal communication between the high-pressure arterial system and the low-pressure venous system causes blood pressure elevation in the ocular veins and exerts pressure on the surrounding cranial nerves (III, IV, V, and VI) that traverse the cavernous sinus. Consequently, patients may present with a constellation of signs and symptoms including diplopia, headache, conjunctival injection, proptosis, and ophthalmoplegia[5].

The diagnostic complexity of CCF is particularly pronounced in the trauma setting, where the clinical manifestations often closely resemble those of skull base fractures[3]. This symptomatic overlap, combined with the uncommon nature of CCF, creates a diagnostic dilemma that can result in missed or delayed diagnosis[6-10]. In Indonesia, particularly in the eastern regions, these inherent diagnostic challenges are further compounded by systemic healthcare limitations. The scarcity of ophthalmologists and limited availability of advanced diagnostic imaging modalities in peripheral healthcare facilities necessitate multiple referrals before patients reach tertiary centers equipped with comprehensive diagnostic and therapeutic capabilities. This fragmented referral chain often results in delayed presentation, during which irreversible ocular and neurological complications may develop.

The complexity of CCF diagnosis is further amplified in the acute trauma setting. Patients presenting with severe head trauma often exhibit multiple life-threatening injuries that understandably take precedence in the initial management phase. In this context, the subtle or evolving ocular manifestations of CCF—such as conjunctival injection, chemosis, or progressive proptosis—may be overlooked or attributed to direct orbital trauma or skull base injury[10]. The initial focus on stabilizing potentially fatal injuries can inadvertently delay the recognition of this vascular complication until more pronounced symptoms emerge, sometimes days, weeks, or even months after the initial trauma[5].

Adding to these challenges is the nature of CCF presentations, which frequently mimic more common and benign ocular conditions. The characteristic conjunctival injection and "red eye" presentation can easily lead to initial misdiagnosis as conjunctivitis, orbital cellulitis, episcleritis, or thyroid eye disease[4,9]. Studies have showed that up to 45% of CCF patients are misdiagnosed by their first healthcare provider, with infectious conjunctivitis being the most common diagnosis. This leads to inappropriate treatment and further diagnostic delay until classic signs such as orbital bruit, pulsatile proptosis, or corkscrew episcleral vessels become apparent or are specifically sought through detailed neuro-

ophthalmic examination[5].

Moreover, trauma patients in Indonesia typically present first to emergency physicians, general surgeons, or neurosurgeons rather than ophthalmologists. These initial providers, while highly skilled in their respective domains and focused on life-threatening injuries, may not be familiar with the subtle neuro-ophthalmic manifestations of CCF or may lack the specialized examination techniques required to detect early signs. The pressure effects on cranial nerves III, IV, and VI within the cavernous sinus may be attributed to direct trauma rather than recognized as secondary to vascular abnormality. This initial point of contact outside ophthalmology increases the risk of missed or delayed diagnosis, as CCF may not be considered in the differential diagnosis until persistent or worsening ocular symptoms prompt specialist referral[11-12].

Understanding these multifactorial diagnostic challenges and the factors contributing to delayed presentation is crucial for improving outcomes in post-traumatic CCF. This case report aims to report the patterns of delayed diagnosis, the imaging challenges encountered in resource-limited settings, and identify prognostic factors associated with advanced presentations of post-traumatic CCF at our tertiary referral center in Indonesia. By characterizing these challenges and outcomes, we hope to raise awareness among first-line providers, contribute to the development of improved referral pathways and early detection strategies, and ultimately reduce the morbidity associated with this potentially devastating but treatable condition.

PARTICIPANTS AND METHODS

Ethical Approval This study was conducted in accordance with the Declaration of Helsinki and institutional ethical standards. Patient data were anonymized and the retrospective design precluded the need for individual informed consent.

Participants A total of 19 patients with CCF confirmed by digital subtraction angiography (DSA) were identified at our tertiary referral center between January 2024 and October 2025. Exclusion criteria were applied as follows: 1) patients without a documented history of head trauma; 2) patients who did not complete a comprehensive ophthalmic examination; 3) patients who declined to undergo endovascular coiling procedures; 4) patients with incomplete or inadequate follow-up data. After applying these exclusion criteria, a final cohort of eight patients met the inclusion criteria and were enrolled in this retrospective case series.

Methods Comprehensive ophthalmic evaluation included visual acuity, intraocular pressure (IOP), fundoscopy, relative afferent pupillary defect (RAPD), and color vision. Clinical presentation patterns, fundoscopic findings, and visual outcomes were systematically documented and analyzed descriptively.

RESULTS

Study Population and Demographics Eight consecutive patients with post-traumatic CCF confirmed by DSA were treated at our center between January 2024 and October 2025 (Table 1). The cohort comprised five males (75%) and three females (25%), with a mean age of 28.5±13.2 y (range 15-54 y). Motor vehicle accidents accounted for 88% of cases (7/8), while one patient (Case 4) developed CCF following repetitive minor head trauma.

Presentation Timing and Referral Pathway The mean interval from initial trauma to definitive CCF diagnosis was 2.6±1.6 mo (range 1 to 6 mo). Seven patients (88%) presented beyond six weeks post-trauma. Three patients travelled over 300 km from rural Eastern Indonesia, requiring referral through district and provincial facilities before reaching our tertiary center. Case 4 experienced the longest delay (6 mo), having been initially misdiagnosed with chronic sinusitis and tension headaches at multiple healthcare facilities.

Clinical Manifestations Proptosis was present in all patients (8/8, 100%) at initial presentation, ranging from mild (2-3 mm) to severe (>8 mm). Bilateral proptosis occurred in three patients (38%) despite unilateral fistula location, attributed to intercavernous venous communication. Orbital bruit, while pathognomonic for CCF, was documented in seven patients (88%). Secondary glaucoma developed in five patients (63%), with intraocular pressure (IOP) ranging from 27 to 40 mmHg in affected eyes. Case 2 presented with IOP of 40 mmHg requiring urgent medical management. Two patients (Cases 4 and 6) maintained normal IOP despite high-flow fistulas.

Neuro-ophthalmic Assessment Baseline visual acuity varied widely (Table 1). Three patients (38%) maintained vision of 20/60 or better, while five patients (63%) presented with severe visual impairment. One patient (Case 5) had no light perception in one eye at presentation. Relative afferent pupillary defect (RAPD) was present in four patients (50%) and four patients (50%) with bilateral afferent pupillary defect (APD) despite unilateral fistula location in Case 5. Color vision testing using Ishihara plates revealed deficits in five of the seven patients tested (71%). Severity ranged from complete color blindness (0/17 plates in Cases 1 and 5) to mild red-green deficiency (14/17 plates in Case 8). Case 2 demonstrated normal color vision (17/17) despite two months of symptoms.

Fundoscopic Findings Fundoscopic examination at presentation revealed three distinct patterns associated with disease severity. Normal optic disc appearance: two patients (Cases 2 and 3, 25%) presented with normal fundoscopic findings despite prolonged symptom duration. Optic disc edema: three patients (Cases 1, 4, and 8, 38%) demonstrated disc edema with preserved disc perfusion. Case 8 exhibited features suggestive of central retinal vein occlusion, including disc edema, vascular tortuosity, and flame hemorrhages. Established optic atrophy: three patients (Cases 5, 6, and 7, 38%) presented with optic disc pallor indicating established optic nerve atrophy.

Table 1 Patient demographics, clinical characteristics, and visual outcomes

Case	Age(y)/sex	Trauma mechanism	Associated injuries	Time to CCF diagnosis	Presenting signs and symptoms	Audible bruit	EOM status	Initial VA (OD/OS)	Final VA (OD/OS)	Initial IOP (mmHg) OD/OS	Final IOP (mmHg) (OD/OS)	RAPD	Fundoscopic findings	Kestenbaum index (OD/OS)	Outcome
1	21/M	Motor accident	Orbital rim fracture	3 mo	Pain, diplopia, headache	Subtle (+)	Mild restriction OD (superotemporal-1)	20/400 20/70	NLP 20/70	17/13	24/13	APD(+)	Normal→edema	4/10	Advanced optic neuropathy, persistent glaucoma
2	18/M	Motor vehicle accident; mandibular fracture (post ORIF)	Mandibular fracture, ICH frontalis	2 mo (symptoms 2 wk post-ORIF)	Proptosis, diplopia, headache	OD(+)	Restricted EOM (superolateral, lateral, inferolateral limitations)	20/25 20/20	20/25 20/20	40/22	14/18	OD (+)	Normal	5/10	IOP controlled with dual medical therapy
3	21/M	Motor accident, head impact right side	Maxillofacial fractures, hematosinus	2 mo	Blurred vision, OD, proptosis, headache	Subtle (+)	Restricted EOM (inferotemporal-4)	20/20 20/20	20/150 20/20	32/13	21/13	OD (+)	Normal, CDR 0.3	5/10	IOP maintained post-coiling
4	44/F	Repeated door impact while sweeping	Not Applicable	6 mo	Pain OD, headache, vertical diplopia	None	Restricted EOM (inferior oblique paresis)	20/20 20/20	20/20 20/20	16/13	12/13	OD (+)	Optic disc edema (+)	4/6	Complete recovery and symptom resolution

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Table 1 Patient demographics, clinical characteristics, and visual outcomes (continued)

Case	Age(y)/sex	Trauma mechanism	Associated injuries	Time to CCF diagnosis	Presenting signs and symptoms	Audible bruit	EOM status	Initial VA (OD/OS)	Final VA (OD/OS)	Initial IOP (mmHg) OD/OS	Final IOP (mmHg) (OD/OS)	RAPD	Fundoscopic findings	Kestenbaum index (OD/OS)	Outcome
5	54/F	Motor vehicle accident; blood from ears/nose/mouth at scene	Severe head trauma	1 mo	Bilateral lid closure (progressive), bilateral proptosis, visual loss, diplopia	Bilateral (+)	Complete bilateral Ophthalmoplegia	NLP 1/60	NLP 1/60	27/33	29/33	APD (+)	Optic atrophy bilateral	3/3	IOP worsened despite complete closure (paradoxical worsening)
6	19/M	Motor vehicle accident; prolonged unconsciousness (21 d ICU)	Multiple cranial fractures, severe TBI	2 mo	Bilateral proptosis, chemosis, ophthalmoplegia, headache (left)	Bilateral (+)	Bilateral Ophthalmoplegia	NLP/20/60	NLP 20/25	16/19	15/19	APD(+)	OD: optic atrophy; OS: slight edematous	1/3	IOP normalized post-embolization
7	15/F	Motor accident, LOC 30 min	Head trauma with brief unconsciousness	2 mo	Pain and swelling OS, red eye, headache	OS(+)	Superior gaze restriction OS (-3)	20/200 20/200	20/200 20/200	28/24	18/20	OD(+)	OD: atrophy; OS: edema, hemorrhages, tortuous vessels (CRVO pattern)	2/6	IOP stabilized after emergent endovascular intervention
8	36/M	Motor vehicle accident; fall with LOC	Head trauma, LOC	2-3 mo	Bilateral proptosis, bilateral bruit, diplopia, tinnitus (right)	Bilateral (+)	EOM restriction OD	20/50 20/30	20/50 20/30	15/19	14/16	APD(+)	Blurred, tortuous	3/8	IOP remained within normal limits

M: Male; F: Female; CCF: Carotid-cavernous fistula; EOM: Extraocular muscles; VA: Visual acuity; OD: Right eye; OS: Left eye; IOP: Intraocular pressure; NLP: No light perception; APD: Afferent pupillary defect; RAPD: Relative afferent pupillary defect; ICU: Intensive care unit; ICH: Intracerebral hemorrhage; ORIF: Open reduction internal fixation; TBI: Traumatic brain injury; LOC: Loss of consciousness; CRVO: Central retinal vein occlusion; CDR: Cup-disc ratio.

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Representative Case Illustration Case 2 (Figure 1) exemplifies the prognostic significance of baseline fundoscopic examination in post-traumatic CCF, even in the context of prolonged diagnostic delay. An 18-year-old male presented with progressive right eye proptosis, diplopia, and conjunctival injection two months following a motor vehicle accident that resulted in mandibular fracture requiring surgical fixation. Symptoms developed two weeks post-operatively and progressed gradually over the subsequent six-month period before definitive diagnosis. At presentation, visual acuity was 20/25 in the right eye and 20/20 in the left eye, with markedly elevated IOP of 40 mmHg in the affected right eye. Clinical examination revealed unilateral proptosis (2.41 cm anterior to interzygomatic line), orbital bruit, conjunctival hyperemia with arterialized episcleral vessels, and restrictive ophthalmoplegia affecting superolateral, lateral, and inferolateral movements. Notably, fundoscopic examination demonstrated normal optic disc appearance bilaterally despite the prolonged symptom duration and severe secondary glaucoma.

DSA confirmed a Type A direct CCF arising from the C4 segment of the right internal carotid artery with complex venous drainage involving the superior ophthalmic vein, inferior ophthalmic vein, sphenoparietal vein, and inferior petrosal sinus. The patient underwent endovascular coil embolization using three Axium coils (14 mm × 40 cm, 14 mm×30 cm, 12 mm×30 cm), achieving partial fistula closure with significant reduction in superior ophthalmic vein reflux and restoration of intracranial flow. At follow-up examination, visual acuity improved to 20/25 in the right eye with complete resolution of proptosis, orbital bruit, and conjunctival hyperemia. IOP normalized to 21 mmHg without medication. Fundoscopic examination continued to show normal optic disc appearance bilaterally. This case demonstrates

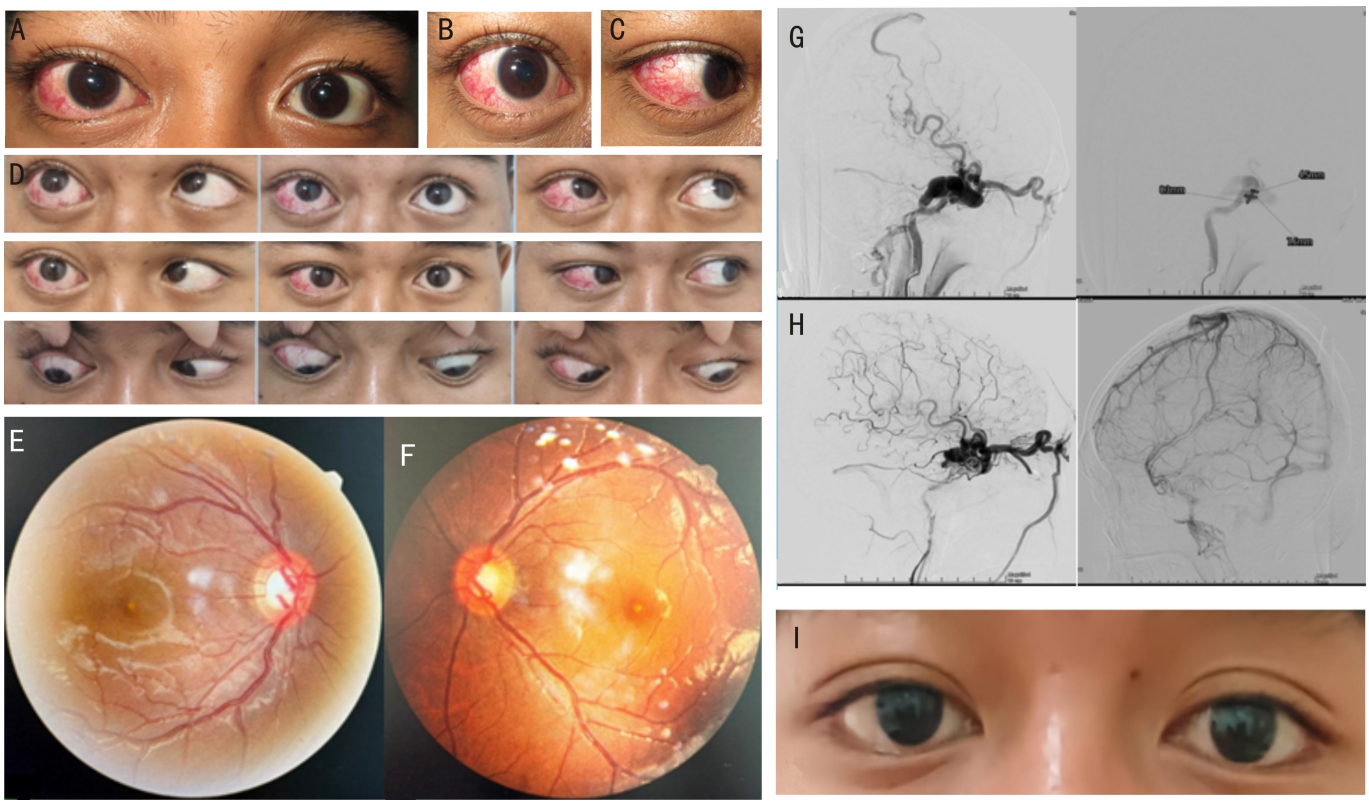

Figure 1 **Diagnostic progression, endovascular intervention, and visual outcome in a high-flow CCF presenting as refractory glaucoma (18 y/male)** A case initially misdiagnosed as secondary glaucoma that achieved excellent visual recovery due to preserved optic nerve integrity at baseline. Fundus photographs were obtained using a non-mydriatic auto fundus camera (AFC-330, Nidek Co., Ltd., Gamagori, Japan) with a 45° field of view. A-C: Initial presentation and misdiagnosis. Clinical photographs detailing the severe right-sided presentation 2 mo post-trauma, characterized by prominent proptosis, dense conjunctival injection, and severely elevated IOP (40 mmHg OD). This led to an initial misdiagnosis of secondary glaucoma. D: Ophthalmic findings. Restricted EOMs, specifically superolateral, lateral, and inferolateral limitations. E, F: Prognostic baseline. Fundoscopic images of the right eye (E) and left eye (F) showing normal optic disc appearance bilaterally, indicating potentially reversible pathology and a favorable prognosis regardless of the degree of final angiographic closure. G: Definitive diagnosis. DSA confirming a Barrow Type A direct CCF at the right ICA C4 segment. H: Treatment outcome (angiographic). DSA image post-embolization showing partial fistula closure achieved via endovascular coiling (three Axium coils) and reduced venous reflux. I: Functional outcome. Follow-up clinical photograph demonstrating complete resolution of orbital congestion and proptosis, stabilization of visual acuity at 20/25 OD, and successful IOP control using dual medical therapy. CCF: Carotid-cavernous fistula; IOP: Intraocular pressure; OD: Right eye; EOM: Extraocular movement; ICA: Internal carotid artery; DAS: Digital subtraction angiography.

that preserved optic disc architecture at baseline, despite prolonged diagnostic delay and severe secondary glaucoma, reliably predicts favorable visual recovery potential following treatment. The favorable outcome achieved despite six-month symptom duration and incomplete angiographic closure underscores that baseline fundoscopic findings provide superior prognostic value compared to either symptom duration or completeness of fistula occlusion.

Angiographic Classification and Vascular Anatomy DSA revealed Type A direct CCF in seven patients (88%), with fistulas originating from the intracavernous internal carotid artery at the C4 segment in six cases (75%) and C5 segment in two cases (25%). One patient (Case 4, 13%) presented with Type D indirect CCF arising from dural branches (inferolateral trunk and middle meningeal artery) following repetitive minor trauma (repeated door impact while sweeping). Complex venous drainage patterns were identified in six patients (75%), involving combinations of anterior (superior ophthalmic vein), posterior (inferior petrosal sinus), and inferior (inferior ophthalmic vein) routes. Five patients (63%) demonstrated decreased or absent internal carotid artery flow distal to the fistula site. Only two patients maintained normal distal ICA perfusion.

Table 2 summarizes the detailed angiographic characteristics, including arterial supply, venous drainage patterns, and ICA flow status for each patient.

Treatment Modalities and Angiographic Outcomes All eight patients underwent endovascular treatment. Coil embolization was used in six patients (75%), while liquid embolic agents (Onyx-18 or Squid-12) were used in two patients (25%), either alone or in combination with coils. Complete angiographic closure was achieved in four patients (50%), partial closure (40%-70% reduction in flow) in three patients (38%), and minimal closure in one patient (13%). Case 3 received partial embolization only due to concerns regarding pre-existing severe cerebrovascular disease (70% C7 stenosis and complete M1 middle cerebral artery occlusion). Case 6 achieved bilateral fistula closure but developed new contralateral symptoms requiring additional intervention

Visual and Functional Outcomes Final visual outcomes are presented in Table 1. Visual acuity remained stable or improved in three patients (38%), while five patients (63%) retained severe visual impairment despite treatment. Among patients presenting with normal optic disc appearance ($n=2$), both maintained or achieved good visual outcomes (20/20 and 20/150) regardless of completeness of angiographic closure. Among patients with optic disc edema at presentation ($n=3$), two achieved stable visual function while one experienced decline. All patients with established optic atrophy at presentation ($n=3$) showed no meaningful visual recovery despite intervention. Secondary glaucoma improved following fistula treatment in most cases, though two patients (Cases 1 and 7) required ongoing IOP-lowering therapy despite successful fistula closure.

Complications Three patients experienced adverse outcomes warranting special consideration. Case 3 experienced contralateral visual decline (OS: 20/20 to 20/150) following partial embolization, developing new retinal hemorrhages and cotton-wool spots consistent with acute hypoperfusion in the context of pre-existing cerebrovascular disease. Cases 1 and 7 developed persistent elevation of IOP following fistula closure, attributed to irreversible trabecular meshwork damage from

Table 2 Angiographic classification, anatomical features, and endovascular intervention

Case	Laterality	Barrow classification	API-ACE classification[13]	Fistula location (segment/zone)	Arterial supply	Venous drainage pattern	Contralateral CS	Embolization approach/material	Closure
1	Unilateral	A	AP±I-A1C1E0	C4 (Z1)	Direct ICA	AP±I	-	TA, Coils	Partial (40%)
2	Unilateral	A	AP±I-A1C1E0	C4 (Z1)	Direct ICA	AP	-	TA, Onyx-18	Complete (100%)
3	Unilateral	A	A-A1C0E0	C4 (Z1)	Direct ICA	AP	-	TA, Coils	Partial (50%)
4	Unilateral	D	P±I-A0C0E1	C4 (Z1)	Indirect ICA	P±I	-	TA, Coils	Complete (100%)
5	Bilateral	A	AP±I-A1C1E0	C5 (Z1)	Direct ICA	AP±I	+	TA, Coils	Partial/ minimal
6	Bilateral	A	AP±I-A1C1E0	C4 Bilateral	Direct ICA	AP±I	+	TA, Onyx-18+ Squid-12	Complete (100%)
7	Bilateral	A	AP±I-A1C1E0	C5 (Z2)	Direct ICA	AP±I (*via* facial vein)	+	TA, Coils	Complete (100%)
8	Unilateral	A	AP±I-A1C1E0	C4 (Z1)	Direct ICA	AP±I	-	TA, Coils	Partial (50%)

ICA: Internal carotid artery; CS: Cavernous sinus; TA: Trans-arterial. A±I: Anterior drainage (superior and inferior ophthalmic veins)±inferior (pterygoid and/or parapharyngeal plexus); P±I: Posteriorly: (inferior and/or superior petrosal sinuses)±inferior (pterygoid and/or parapharyngeal plexus); AP: Anterior+posterior.

prolonged episcleral venous hypertension. No patients developed intracranial hemorrhage or cortical venous infarction during the follow-up period (mean 6 mo, range 3-12 mo).

DISCUSSION

Our series of eight patients with post-traumatic CCF presenting to an Indonesian tertiary referral center reveals critical insights into the consequences of delayed diagnosis and identifies baseline fundoscopic examination as the most reliable predictor of visual outcomes. The cohort exhibited typical epidemiologic features of traumatic CCF, with male predominance and young mean age reflecting the demographics of high-velocity motor vehicle trauma in our region. However, the extended interval to diagnosis, coupled with advanced presentations characterized by irreversible optic nerve damage and secondary glaucoma in the majority of cases, underscores the profound impact of healthcare system barriers on patient outcomes. Despite advances in imaging and intervention, late diagnosis of post-traumatic CCF remains a critical barrier to optimal outcomes, particularly in resource-limited settings. Our cohort's mean diagnostic delay of 2.6 mo substantially exceeds the established six-week therapeutic window[7], directly compromising visual prognosis while substantially increasing treatment complexity[12,14-15]. Delayed diagnosis in our series reflects multiple interconnected challenges.

First, CCF manifestations often mimic benign conditions such as conjunctivitis, episcleritis, thyroid-related orbitopathy, or simple post-traumatic red eye. The classical triad of pulsatile proptosis, orbital bruit, and corkscrew conjunctival vessels is rarely complete at initial presentation. Initial misdiagnosis occurs in up to 45% of reported cases and in 38% of our series[10]. Many patients receive multiple courses of treatment for presumed infection or inflammation before CCF is considered.

Second, some cases develop insidiously with mild symptoms and may remain visually asymptomatic for extended periods. Case 2 exemplified this challenge, maintaining 20/25 vision with minimal conjunctival hyperemia despite two-month symptom duration. His normal CT and magnetic resonance imaging (MRI)/magnetic resonance angiography (MRA) findings demonstrate that high clinical suspicion must override negative imaging, as only DSA ultimately revealed his Type A direct CCF. This case confirms that absence of severe symptoms or positive cross-sectional imaging cannot exclude CCF when clinical context suggests vascular pathology[14-15].

Third, fragmented referral pathways create substantial barriers to timely diagnosis. Patients in rural settings frequently present first to non-ophthalmologists whose triage priorities focus on life-threatening injuries. Subtle neuro-ophthalmic signs are easily overlooked or attributed to direct trauma[14]. Three of our patients travelled over 300 km through mandatory tiered referral systems, requiring up to 12 h of transport. Such distances create both logistical obstacles and financial burdens that delay or prevent patients from seeking specialized care.

Fourth, limited access to definitive imaging represents a critical challenge. DSA remains the gold standard for definitive diagnosis but is typically limited to tertiary facilities, resulting in delays of weeks to months after initial symptom onset[14].

Finally, the sequential nature of trauma management contributes significantly to diagnostic delays. Post-trauma patients are typically managed first by trauma surgeons or emergency physicians whose appropriate focus on life-threatening injuries inadvertently leads to overlooking of ocular symptoms. Given CCF's rarity (estimated incidence 1-3 per million), clinical recognition remains challenging in acute trauma settings. The temporal gap between trauma and symptom presentation, often weeks to months, further obscures the connection between injury and ophthalmic complaints. The consequences of diagnostic delay in our series were substantial and largely irreversible. Our data demonstrate that delayed recognition allows reversible optic disc congestion to progress to established atrophy, an irreversible state after which embolization or fistula closure yields minimal functional visual recovery[11,15].

Our cases demonstrated clear temporal progression, normal disc appearance transitioning through optic disc edema to irreversible optic atrophy. Patients may transition from a potentially recoverable to a definitive vision-impaired state over weeks of missed diagnostic opportunity—a preventable outcome with earlier recognition. Our case series reveals that diagnostic delay profoundly impacts outcomes in post-traumatic CCF, with 88% presenting beyond the critical six-week window. During weeks 0-2, reversible venous congestion and optic disc edema allow potential recovery, but by weeks 3-6, axonal transport failure initiates ganglion cell apoptosis, marking the final opportunity for intervention[15-17]. Beyond six weeks, completed axonal degeneration establishes irreversible optic atrophy, as observed in 38% of our cohort. This temporal progression explains why baseline fundoscopic findings proved more predictive of visual outcomes than angiographic closure rates[5,7]. This disconnect between technical and functional success was evident: patients with preserved disc architecture achieved favorable outcomes even with partial fistula closure, whereas those with established atrophy showed no recovery despite complete angiographic occlusion.

Sustained venous hypertension caused secondary glaucoma in 63% of cases, reflecting impaired aqueous humor outflow from elevated episcleral venous pressure[1,4]. Advanced presentations featured complex multi-compartmental drainage in 50%, making complete closure technically challenging[16]. In such contexts, systematic neuro-ophthalmic examination—including RAPD testing and fundoscopy—provides accessible prognostic tools that supersede imaging-based endpoints. These findings align with reports emphasizing that prompt identification of visual impairment is crucial to avoid permanent damage, and that treatment decisions should prioritize clinical improvement over angiographic perfection.

Sustained venous hypertension from ongoing arteriovenous shunting in untreated CCF causes intractable secondary glaucoma, observed in 63% of our cases. Elevated episcleral venous pressure impairs aqueous humor outflow and renders even surgical interventions often ineffective without addressing the underlying vascular lesion. Case 5 demonstrated that prolonged venous hypertension causes irreversible trabecular damage, with IOP paradoxically worsening despite complete fistula closure. Even successful endovascular treatment may not reverse established glaucoma, necessitating lifelong management in affected patients[7,14].

Advanced CCFs with multi-compartmental or bilateral venous drainage, observed in 50% of our cases, complicate planning and execution of endovascular procedures[16]. The formation of collateral venous channels during the prolonged pre-diagnosis period makes achieving complete closure technically more challenging and increases the risk of paradoxical worsening or new neurological deficits post-procedure, especially in cases with cortical venous reflux[17]. DSA in our series revealed complex venous drainage patterns, including involvement of inferior ophthalmic vein and petrosal sinuses in 75% of cases, which occurred alongside more severe clinical presentations and lower rates of complete closure[18-19]. The challenges observed in our series reflect broader patterns related to healthcare infrastructure and resource availability. In advanced economies with streamlined trauma care pathways, median time to diagnosis ranges from one to two weeks, with consequently lower rates of irreversible visual loss[14,20]. In contrast, reports from low- and middle-income countries where diagnostic delays of three to six months are typical reveal substantially higher rates of secondary glaucomatous optic atrophy, central retinal vein occlusion (CRVO)-pattern retinopathy, bilateral involvement, and persistent motility deficits[14].

Our series aligns with these international patterns from resource-limited settings. This disparity in outcomes is not attributable to differences in injury severity or CCF characteristics, but rather to elapsed time between injury and definitive intervention—a modifiable system-level factor with profound impact on individual patient outcomes[21-23]. Fundoscopic examination proved to be a reliable prognostic indicator in our series, more predictive than both angiographic closure rates and initial visual acuity. This progression matched outcomes more consistently than any other clinical or angiographic parameter, a finding reported in other series[7,14]. Patients presenting with normal or near-normal disc appearance retained potential for visual recovery with prompt intervention. Cases 2 and 3 maintained stable outcomes despite achieving only partial fistula closure, suggesting that preserved optic nerve architecture at baseline permits functional compensation even when vascular anatomy cannot be fully normalized. Conversely, established optic atrophy consistently predicted poor visual prognosis regardless of treatment success. Case 5 achieved complete angiographic closure yet remained at no light perception and 1/60 bilaterally due to baseline bilateral disc pallor, demonstrating that technical procedural success does not reverse pre-existing irreversible nerve damage. Case 6 provided a useful within-patient comparison that isolated the prognostic value of fundoscopic findings while controlling for all other variables. His left eye presented with baseline disc edema and improved from 20/60 to 20/25 following bilateral embolization, while his right eye with established atrophy remained at no light perception despite identical treatment. This comparison demonstrates that baseline optic nerve status, not treatment efficacy or fistula characteristics, determines visual outcomes. Optic disc edema without atrophy represented an important window of potentially reversible pathology. Case 2, despite two-month delay and normal cross-sectional imaging, maintained preserved disc perfusion that predicted favorable recovery. Following partial embolization, he maintained 20/25 vision with full symptom resolution. Case 7's presentation with CRVO-like features including disc edema, vascular tortuosity, and flame hemorrhages illustrated secondary venous occlusive disease requiring prompt intervention to prevent irreversible retinal damage. Her timely treatment resulted in stabilized visual acuity, consistent with the concept that acute congestive changes remain reversible if addressed before atrophic transformation[24]. These findings have practical implications for treatment planning. Patients presenting with severe posterior segment findings including optic atrophy should be counseled that vision recovery may be limited even with successful fistula closure, allowing realistic goal-setting. Conversely, patients with normal or minimal fundoscopic changes can be informed of favorable prognosis if treatment is initiated promptly. Fundoscopic findings may also guide treatment intensity. Patients with severe findings may warrant more aggressive multi-modality approaches, whereas those with minimal findings might be managed with more conservative strategies[16,24].

DSA revealed Type A direct CCF in 88% of cases, predominantly at the C4 segment. The high prevalence of direct fistulas aligns with traumatic etiology, as high-velocity impact causes direct carotid artery laceration into the cavernous sinus. An unexpected finding was Case 4's Type D indirect CCF following repetitive minor head trauma, suggesting alternative mechanisms whereby trauma causes secondary dural-arterial recruitment rather than acute laceration. Endovascular treatment achieved complete angiographic closure in only 38% of cases, substantially lower than the 70%-90% success rates reported by high-volume centers. This discrepancy likely reflects both the complexity of direct fistulas with extensive collateral drainage in delayed-presenting cases and our center's evolving experience with these rare lesions. Liquid embolic agents (Onyx-18, Squid-12) demonstrated superiority over coiling alone for achieving durable closure, consistent with contemporary literature[13]. However, the most striking finding was the marked disconnect

between angiographic success and functional visual outcomes. Baseline optic nerve status, not degree of fistula closure, determined visual prognosis[14]. Cases 2 and 3 maintained stable or improved visual function despite achieving only partial fistula closure, experiencing significant symptomatic improvement with resolution of chemosis, reduction in proptosis, and IOP control. Case 2's IOP decreased from 40 to 14 mmHg following treatment, and orbital bruit resolved completely. These cases demonstrate that subtotal closure may provide sufficient clinical benefit when complete closure proves technically challenging or carries high procedural risk, challenging traditional emphasis on complete angiographic cure as the sole measure of treatment success. This pragmatic approach recognizes that treatment goals are clinical improvement and quality of life restoration rather than angiographic perfection. We generally accepted partial closure when patients achieved symptomatic relief, IOP control, and stable or improved visual function, particularly if further treatment would require high-risk approaches or if venous anatomy precluded safe additional access[13,25].

Collective findings from our series inform several evidence-based recommendations for clinical practice. First, clinicians should maintain high clinical suspicion for CCF in any patient presenting with persistent unilateral red eye following head trauma, particularly when accompanied by elevated IOP, corkscrew episcleral vessels, proptosis, or RAPD. Second, systematic documentation is essential for all patients presenting with red eye in the post-traumatic setting. At minimum, examination should include orbital auscultation, IOP measurement, RAPD testing, and fundoscopic examination with documentation of optic disc appearance and retinal vascular status. Third, management of secondary glaucoma requires a proactive approach. Prompt IOP-lowering therapy should be initiated immediately upon CCF diagnosis, even before definitive endovascular treatment, to prevent irreversible glaucomatous damage. Fourth, treatment endpoints should be defined based on clinical improvement rather than solely angiographic criteria. Subtotal closure should be considered acceptable when complete closure proves technically challenging or carries high procedural risk, provided that meaningful clinical improvement has been achieved. Finally, fundoscopic examination at presentation should be thoroughly documented and used to guide patient counseling regarding prognosis. The presence of optic atrophy warrants candid discussion about limited visual recovery potential, while preserved disc architecture should emphasize the favorable prognosis achievable with prompt treatment[1-4].

This study has inherent limitations as a retrospective case series from a single tertiary center. The small sample size (n=8) precludes statistical analysis of prognostic factors and limits identification of independent predictors of outcome. Selection bias toward advanced presentations is inevitable given our role as a tertiary referral center, limiting generalizability to settings with more immediate access to specialized care. Treatment heterogeneity reflects evolving institutional practices and case complexity rather than standardized protocols. Variable follow-up duration and incomplete documentation of certain parameters constrain comprehensive outcome analysis. Despite these limitations, our series provides valuable insights into the unique challenges of managing post-traumatic CCF in resource-limited settings, where geographic barriers and diagnostic delays result in presentation beyond the critical therapeutic window in the majority of patients. The consistent patterns observed across our cases, particularly regarding prognostic value of fundoscopic findings and the disconnect between angiographic and clinical outcomes, align with and extend findings from larger series, suggesting that our observations have broader applicability despite the small sample size.

In conclusion, this case series from an Indonesian tertiary referral center demonstrates that baseline fundoscopic examination provides the most reliable prognostic indicator for visual outcomes in post-traumatic CCF, superseding both angiographic closure rates and initial clinical severity. The identification of three distinct fundoscopic presentation patterns—normal optic disc, edema, and established optic atrophy—enables accurate prognostic stratification at initial evaluation. Patients presenting with established optic atrophy demonstrated no meaningful visual recovery regardless of treatment success or completeness of angiographic closure, while those with preserved disc architecture achieved favorable outcomes even with incomplete fistula occlusion. This disconnect between angiographic and functional outcomes challenges traditional treatment paradigms that prioritize complete fistula closure as the sole measure of success.

The mean diagnostic delay of 2.6 mo in our cohort, with 88% of patients presenting beyond the critical six-week therapeutic window, resulted in 38% developing irreversible optic nerve damage. This delay reflects multifactorial barriers including diagnostic mimicry of benign conditions, fragmented referral pathways, limited access to definitive imaging, and the sequential nature of trauma management where ocular symptoms may be overlooked during initial stabilization. The high prevalence of secondary glaucoma (63%) and complex venous drainage patterns (75%) in delayed presentations further complicates treatment and compromises outcomes.

In resource-limited settings where advanced imaging availability is restricted and diagnostic delays are common, systematic neuro-ophthalmic examination—particularly RAPD testing and fundoscopic evaluation—provides accessible and reliable tools for early diagnosis and prognostic assessment. Recognition of post-traumatic CCF within the six-week therapeutic window, guided by high clinical suspicion for any post-traumatic red eye with elevated IOP, proptosis, or RAPD, remains critical for preventing permanent visual loss. Treatment decisions should be guided by baseline fundoscopic findings and clinical improvement rather than pursuit of complete angiographic closure in all cases, particularly when

patients present with established optic atrophy or when aggressive intervention carries substantial procedural risk.

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