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Original Article

Central retinal artery occlusion: a retrospective study of disease presentation, treatment, and outcomes

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Objective: A central retinal artery occlusion (CRAO) represents a form of ocular stroke with poor visual prognosis. CRAO shares a common pathophysiology with cerebral ischemic stroke but presents unique diagnostic and management challenges leading to variability in clinical practice. This study aims to assess the presentation, treatment, and outcomes of CRAO at a tertiary care centre in Canada over 2 years and elucidate potential areas for improvement in the care of these patients.

Methods: Single-institution retrospective review including 27 patients diagnosed with CRAO from March 2018 to March 2020 in Edmonton, Alberta.

Results: Most patients with CRAO presented to eye care providers (14 of 27); others presented to the emergency department (10 of 27) or family physicians (2 of 27). Three patients (11.1%) presented within 4.5 hours of symptom onset. At presentation, 81% of patients had visual acuity of 20/400 or worse in the affected eye. No patients received thrombolysis. The majority of CRAO cases had a nonarteritic etiology (92.6%). All patients had at least one pre-existing vascular risk factor. Forty-eight percent of patients received escalated medical therapy. Ipsilateral carotid stenosis was identified in 5 patients (18.5%); 3 patients required carotid endarterectomy. Two patients were diagnosed with atrial fibrillation. Two patients experienced symptomatic cerebral ischemia within 6 weeks of CRAO.

Conclusions: The majority of patients with CRAO presented to eye care providers, and few present within the potential window for thrombolysis of 4.5 hours, highlighting the need for public awareness strategies. Our cohort highlights the significant rate of systemic comorbidity that exists in these patients.

Objectif: L'occlusion de l'artère centrale de la rétine (OACR) constitue en fait une forme d'accident vasculaire cérébral (AVC) oculaire au pronostic visuel médiocre. Si la physiopathologie de l'OACR est semblable à celle de l'AVC ischémique, l'OACR s'accompagne de défis uniques en matière de diagnostic et de prise en charge, ce qui donne lieu à une grande variabilité en pratique clinique. Notre étude a pour objectif d'évaluer les résultats de l'examen initial, le traitement et le bilan de l'OACR dans un centre de soins tertiaires du Canada sur une période de 2 ans, et de proposer des axes d'amélioration potentielle de la prise en charge de ces patients.

Méthodes: Revue rétrospective réalisée dans un établissement unique qui regroupait 27 patients chez lesquels on a établi un diagnostic d'OACR entre mars 2018 et mars 2020 à Edmonton, en Alberta.

Résultats: La plupart des patients qui ont subi une OACR ont d'abord consulté un spécialiste des soins oculaires (14 sur 27); d'autres se sont rendus au service des urgences (10 sur 27) ou ont consulté leur médecin de famille (2 sur 27). Trois patients (11,1 %) ont consulté dans les 4,5 heures suivant l'apparition des symptômes. Lors de l'examen initial, l'acuité visuelle de 81 % des patients était de 20/400 ou pire dans l'úil atteint. Aucun patient n'a fait l'objet d'une thrombolyse. La majorité des cas d'OACR était d'origine non artéritique (92,6 %). Tous les patients présentaient déjà au moins un facteur de risque vasculaire, et 48 % des patients ont fait l'objet d'une intensification du traitement médicamenteux. Une sténose de la carotide homolatérale a été diagnostiquée chez 5 patients (18,5 %); d'ailleurs, 3 patients ont dû subir une endartériectomie de la carotide. On a noté la présence d'une fibrillation auriculaire chez 2 patients. Enfin, il s'est produit une ischémie cérébrale symptomatique chez 2 patients dans les 6 semaines suivant l'OACR.

Conclusions: La majorité des patients qui ont subi une OACR ont consulté un spécialiste des soins oculaires, mais très peu l'ont fait à l'intérieur de la fenêtre pendant laquelle une thrombolyse peut être réalisée, soit dans les 4,5 heures suivant l'épisode. Voilà qui met en lumière l'importance des stratégies de sensibilisation du public. Notre étude fait également ressortir le taux significatif de comorbidité générale présente chez ce type de patients.

A central retinal artery occlusion (CRAO) is a form of ocular stroke that occurs when there is a blockage in the central retinal artery, a branch of the ophthalmic artery, resulting in acute retinal ischemia. The estimated incidence ranges from 1−10 per 100 000 and increases with age, with a peak around 80 years of age.[1−3] CRAO is classified as arteritic (resulting from giant cell arteritis) or nonarteritic. Among those with nonarteritic CRAO, emboli resulting from ipsilateral carotid stenosis, cardiac valvular disease, or arrhythmia remain the most common causes.

CRAO is cause of significant visual disability. Visual outcomes are poor, with most patients experiencing severe and permanent vision loss of counting fingers or less[4]; only 17% of patients experience spontaneous improvement in vision.[5] Less than 10% report meaningful visual recovery.[6] CRAO increases the risk of falls and subsequent hip fractures and

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ultimately threatens the ability of patients to live independently.[7] Greater dependency on others, limitations in social functioning, and mental health symptoms contribute to a reduced vision-related quality of life with a deleterious impact on general and mental health.[8–10]

CRAO represents acute retinal ischemia and is a form of cerebral infarction. The American Academy of Ophthalmology Preferred Practice Patterns 2019 for Retinal and Ophthalmic Artery Occlusions states that "acute, symptomatic, posterior segment arterial occlusions represent an emergent ophthalmic condition and require prompt evaluation."[11] It is recommended that affected patients have an "immediate referral to a stroke center for a medical evaluation." Despite the commonalities in pathophysiology between retinal and cerebral ischemia, the management of CRAO is much more variable in practice because of multiple challenges. Although 75% of neurologists surveyed believed that acute CRAO requires urgent referral to a stroke centre, only 18% of retina specialists agreed.[12] Patients do not necessarily recognize acute vision loss as a form of stroke and may seek care in a delayed fashion; they also may present to an eye care provider instead of the emergency department. To clarify the cause for monocular vision loss, the patient may be referred to an eye care professional, leading to further delay in management. Ocular manoeuvres may be offered acutely, while vascular imaging and stroke risk factor modification may be delayed or organized on an outpatient basis. Despite a growing body of evidence and ongoing clinical trials evaluating the use of intravenous thrombolytic therapy for CRAO, few patients present within the window for treatment, and institutional protocols do not exist.[13]

This study aims to assess the presentation, early management, and outcomes of CRAOs in the Edmonton catchment area between 2018 and 2020, with the goal of identifying potential areas for improvement to facilitate early diagnosis and intervention in the hope of improving the visual and systemic outcomes in patients with CRAO.

Methods

Study design and patient selection

Research ethics board approval was obtained. A retrospective review of patients diagnosed with CRAO from March 1, 2018, to March 1, 2020, was performed at a major retina practice serving northern Alberta. Eligible patients were identified via electronic medical records using the search terms "central retinal artery occlusion," "retinal artery occlusion," and "CRAO." This search yielded 138 charts, which were reviewed for accuracy of the diagnosis (Fig. 1). Alternate diagnoses were identified in 97 of 138 charts, and these patients were excluded from the study. Alternate diagnoses included branch retinal vein occlusion, central retinal vein occlusion, branch retinal artery occlusion, old central retinal artery occlusion, and old cilioretinal artery occlusion (*old* was defined as occurring prior to March 1, 2018, outside the study window). Forty-one patients were eligible for the study.

Participant recruitment

Eligible patients were contacted by a research coordinator, and verbal consent was obtained over the phone with electronic confirmation via email. Twenty-six patients

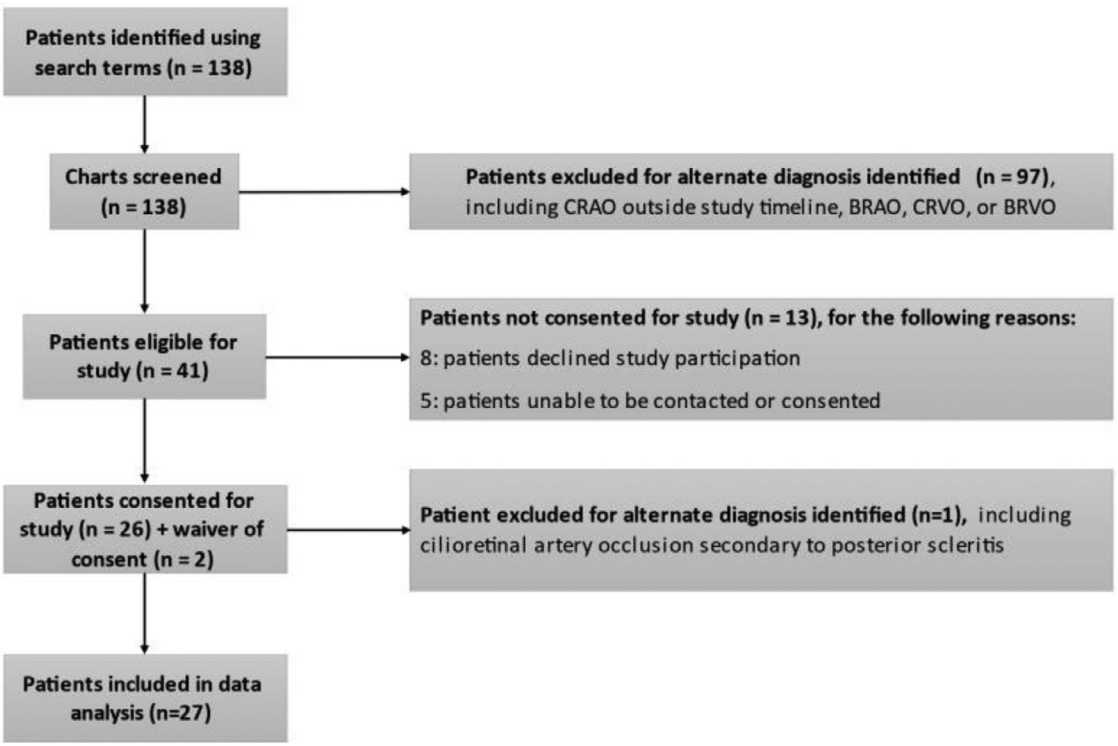

Fig. 1—Flowchart of inclusion or exclusion of patients.

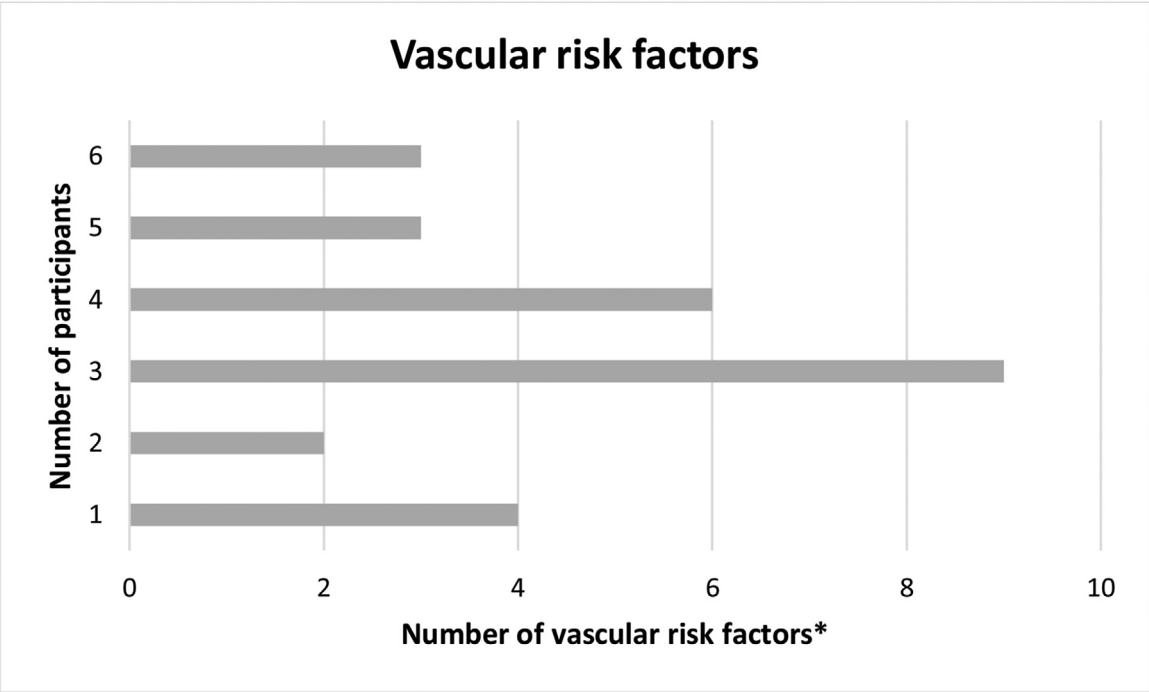

Fig. 2—*Vascular risk factors included a history of smoking, hypertension, diabetes mellitus, dyslipidemia, prior stroke, transient ischemic attack, or amaurosis fugax, cardiovascular disease, or atrial fibrillation.

consented, 8 declined study participation, and 5 could not be reached. A waiver of consent was issued for 2 patients who are deceased. One patient was subsequently excluded for an alternate diagnosis (cilioretinal artery occlusion). In total, 27 patients were included in the study, and their charts were further reviewed.

Outcome measures

Demographics, pre-existing medical conditions, and medications were recorded. Primary outcome measures included time to presentation (defined as <4.5 hours, 4.5–24 hours, and >24 hours), initial health care provider (emergency department, ophthalmology, primary care physician, or optometry), presenting visual acuity in event eye, time to diagnosis by an ophthalmologist, acute intervention offered (anterior-chamber paracentesis, ocular hypotensive agents, oral hypotensive agents, ocular massage, or thrombolysis), time to investigations (inflammatory markers, neuroimaging, carotid imaging, Holter monitor, and echocardiogram), results of investigations, and final visual acuity in event eye.

Table 1—Patient characteristics

Characteristics	Total number of patients (n=27) (%)
Mean age, years	73.3 (range, 37 – 93)
Sex	
Male	12 (44.4%)
Female	15 (55.6%)
Affected eye	
Left	1 (4%)
Right	26 (96%)
Stroke risk factors	27 (100%)
Hypertension	23 (85.2%)
Dyslipidemia	22 (81.5%)
Diabetes mellitus	14 (51.9%)
Coronary Artery Disease	11 (40.7%)
Atrial fibrillation	3 (11.1%)
Prior Stroke, TIA, or amaurosis fugax	9 (33.3%)
History of smoking	10 (37.4%)
Prior antiplatelet/anticoagulation therapy	20 (74.1%)
Single antiplatelet agent	13 (48.1%)
Dual antiplatelet agents	3 (11.1%)
Anticoagulant	4 (14.8%)

TIA = transient ischemic attack

Table 2—Features at presentation of central retinal artery occlusion

Characteristics	Total number of patients (n=27)
Initial presentation	
Emergency physician	10 (37.0%)
Ophthalmologist	8 (29.6%)
Optometrist	6 (22.2%)
Family physician	2 (7.4%)
Unknown	1 (3.7%)
Time to presentation	
< 4.5 hours	3 (11.1%)
4.5 – 24 hours	9 (33.3%)
>24 hours	15 (55.6%)
Presenting Snellen visual acuity	
No light perception	1 (3.7%)
Light perception	4 (14.8%)
Hand motions	8 (29.6%)
Count fingers	9 (33.3%)
20/200	3 (11.1%)
20/100	1 (3.7%)
20/40	1 (3.7%)
Etiology of CRAO	
Non-arteritic	25 (92.6%)
Arteritic (GCA, small vessel vasculitis)	2 (7.4%)

Table 3—Outcomes of patients with central retinal artery occlusion

	Total number of patients (n=27) (%)
Final Snellen visual acuity	
No light perception	1 (3.7%)
Light perception	2 (7.4%)
Hand motions	11 (40.7%)
Count fingers	5 (18.5%)
20/400	2 (7.4%)
20/100	1 (3.7%)
20/60	1 (3.7%)
20/40	1 (3.7%)
20/30	1 (3.7%)
20/20	1 (3.7%)
No final VA	1 (3.7%)
Improved visual acuity*	
With ocular intervention (n=5)	1 (20%)
Without ocular intervention (n=22)	6 (29%)
Comorbid events within 3 months	
TIA or stroke	2 (7.4%)

*Defined as improvement of 2 or more Snellen visual acuity lines.

Table 4—Investigations and results of patients with central retinal artery occlusion

	Total number of patients (%)
Investigation	
Computer Tomography (CT) or CT Angiogram (CTA)	3 (11.1%)
Magnetic Resonance Imaging (MRI)	0 (0%)
Carotid Dopplers	20 (74.1%)
Transthoracic Echocardiogram	20 (74.1%)
Inflammatory markers	23 (85.2%)
Temporal artery biopsy	4 (14.8%)
Carotid Doppler Ultrasound	**(n = 20)**
>70% stenosis	2 (10%)
50-70% stenosis	3 (15%)
Echocardiogram	**(n = 23)**
Normal	6 (73.9%)
Abnormal*	17 (26.1%)
Acute stroke on initial head imaging	**(n=3)**
Acute stroke present	0 (0%)
Acute stroke absent	3 (100%)

*Abnormal echocardiogram included mitral valve calcification, other mitral valve abnormalities, aortic valve calcification, or other aortic valve abnormalities.

Statistical analysis

Data were collected from charts and recorded in an Excel (Microsoft, Redmond, Wash.) spreadsheet. Basic summary statistics were performed to quantify average age at onset of symptoms and timing of presentation, assessment, referrals, investigations, and interventions.

Results

Patient characteristics and presentation

Twenty-seven patients (mean age 73.3 years; range, 37–93 years) were included; 55.6% were female (Table 1). The right eye was more frequently involved (96%, n = 26 of 27). At presentation, 81.5% of patients (22 of 27) had visual acuity of 20/400 or worse in the affected eye (range, 20/40 to no light perception; median: counting fingers; Table 2). Twenty-one patients (77.7%) were left with visual acuity of 20/400 or worse (range, 20/30 to no light perception; median: hand motions) at follow-up (range, 7 days to 33 months after CRAO; Table 3).

Ten patients (37%) presented initially to the emergency department, whereas the remainder presented to an ophthalmologist (8 of 27, 29.2%), optometrist (6 of 27, 22.2%), family physician (2 of 27, 7.4%), or unknown (1 of 27; Table 2). Three patients (11.1%) presented within 4.5 hours of symptom onset, 33.3% (9 of 27) presented within 24 hours, and 55.6% (15 of 27) presented after 24 hours. For those presenting within 4.5 hours, average additional time before an ophthalmology assessment was 2.83 hours (2 hours 50 minutes). Twenty-six percent (7 of 27) had documented visible emboli on fundus examination. Eighty-nine percent (24 of 27) had previously diagnosed ocular pathology in the event eye, including branch retinal artery occlusion (1 of 27), ocular ischemic syndrome (2 of 27), diabetic retinopathy with and without neovascularization (4 of 27), wet (1 of 27) and dry (2 of 27) age-related macular degeneration, glaucoma (3 of 27), prior cataract extraction and lens implantation (10 of 27), cataract (10 of 27), prior retinal detachment with repair (1 of 27), epiretinal membrane (1 of 27), lattice degeneration (1 of 27), and amblyopia (1 of 27).

Additional testing included inflammatory markers (23 of 27, 85%) and computed tomography scan of the head (2 of 27, 7.4%) performed acutely; none showed evidence of acute ischemic stroke. No patients underwent dedicated vascular imaging or magnetic resonance imaging with diffusion-weighted sequences. Outpatient investigations included carotid Doppler imaging (20 of 27, 74.1%), echocardiogram (20 of 27, 74.1%), and referral to stroke neurology (21 of 27, 77.8%; Table 4). Average time until carotid imaging was performed was 12 days (range, 1–42 days), and average time until echocardiography was 75 days (range, 4–480 days). Three patients had an echocardiogram performed in the weeks preceding CRAO for alternate presentations, including transient ischemic attack, endocarditis, and a renal transplant work-up, so echocardiography following CRAO was not performed.

Etiology and comorbidity

The underlying etiology for CRAO was attributed to systemic vasculitis including giant cell arteritis (n = 1) and small vessel vasculitis (n = 1) in 7.4%, whereas 92.6% had a presumed or confirmed embolic etiology (Table 2). One patient had a pacemaker insertion 12 days earlier, and 1 patient had an aortic valve replacement 3 days prior to CRAO. All patients with embolic CRAOs had pre-existing vascular risk factors, including hypertension (23 of 25, 92%), dyslipidemia (20 of 25, 80%), diabetes mellitus (14 of 25, 56%), and cardiovascular disease (11 of 25, 44%; Table 1, Fig. 2). Of the patients with embolic CRAOs, 72% (18 of 25) were already on antiplatelet/anticoagulant therapy prior to presentation (Table 1). Five patients (18.5%)

Table 5—Management of patients presenting with central retinal artery occlusion

Management	Total number of patients (n = 27) (%)
Ocular massage, AC paracentesis, topical anti-glaucoma drops	5 (18.5%)
Thrombolysis	0 (0%)
Escalation of medical therapy*	13 (48.1%)
Ipsilateral carotid endarterectomy (CEA)	1 (3.7%)
Contralateral CEA	1 (3.7%)
None	10 (37%)

*Escalation of medical therapy included initiating antiplatelet or anticoagulant therapy, initiating or increasing the dose of a cholesterol-lowering agent, or antihypertensive medication.

were found to have ipsilateral carotid stenosis >50%, and 3 patients required carotid endarterectomy for severe carotid stenosis (>70%). Of the 8 patients with available data, 2 were newly diagnosed with atrial fibrillation (Table 4). Two patients (7.4%) experienced symptomatic transient ischemic attack or cerebral stroke within 6 weeks of CRAO.

Acute management and outcomes

Five patients (18.5%) were treated with ocular massage, anterior-chamber paracentesis, or topical intraocular pressure–lowering medications within 24 hours of developing CRAO (Table 5). In this group, 20% (1 of 5) had an improvement in vision of 2 lines, and 80% (4 of 5) stayed the same. No patients received thrombolysis. Of those who received no ocular treatments, 6 patients (29%) experienced improvements in Snellen visual acuity of 2 or more lines from presentation to follow-up visit. Forty-eight percent of patients (13 of 27) were managed with escalation of medical therapy, including additional anticoagulants, cholesterol-lowering agents, or blood pressure medications (Table 5) either at the time of presentation or at the outpatient encounter with a stroke neurologist.

Discussion

We present a retrospective review of the outcomes of patients presenting with CRAO who were able to attend follow-up at an outpatient retina clinic in Edmonton. The results of this study show that 63% of patients with CRAO presented initially to eye care providers or primary care physicians. Eighty-nine percent presented more than 4.5 hours after symptom onset which is outside the window to receive thrombolytic treatment, a finding that has been reported previously.[5,14–16] These findings highlight the need for greater public health strategies to enhance awareness that acute monocular vision loss is a medical emergency that should be evaluated immediately at the nearest stroke treatment centre.

Obtaining an in-person ophthalmic examination for diagnostic confirmation adds to the complexity of CRAO management. In our study, nearly 3 hours had elapsed before an ophthalmic examination was performed. This delay would prevent otherwise eligible patients from accessing thrombolysis within the therapeutic window. Ocular manoeuvres such as intraocular pressure–lowering medications and anterior-chamber paracentesis were attempted in 5 patients, and only 1 (20%) experienced an improvement in visual acuity.

Acute retinal ischemia is a form of acute cerebral ischemia, but our study demonstrates major differences in management of patients with CRAO. All of our patient cohort had at least 1 vascular risk factor, and 72% were already taking antiplatelet therapy or anticoagulation. Eighteen percent had ipsilateral carotid stenosis of >50%, and 3 patients required carotid endarterectomy for severe carotid stenosis. Two patients with available data had newly diagnosed atrial fibrillation, and 2 (7.4%) experienced a second episode of cerebral ischemia within 6 weeks. However, no patients received dedicated vascular imaging or stroke neurology consultation at presentation, and none were offered thrombolysis. Instead, vascular imaging, investigations, and neurology consultation were organized on an outpatient basis.

We recognize that this study has several limitations. First, the retrospective nature of the study contributed to missing information. Time from symptom onset to presentation was based on patient recollection and often incomplete documentation. Our cohort was limited to a single institution, and 15 eligible patients (37%) declined participation, were unable to participate, or could not be reached. Patients with CRAO and concomitant cerebral ischemia were less likely to be captured, and those with serious complications such as myocardial infarction, stroke, or death following CRAO would have been missed because of inability to attend follow-up. For these reasons, we believe that our study population likely represents those with better outcomes and milder underlying vascular disease.

Our study highlights the need for institutional reflection in CRAO management within Canadian centres. Whereas patients with signs of transient or manifest cerebral ischemia almost uniformly receive admission for stroke neurology consultation and expedited inpatient investigations, CRAO was managed on an outpatient basis. Current guidelines on the management of CRAO recommend urgent vascular imaging, stroke neurology consultation, and consideration of thrombolysis if the patient presents within the therapeutic window of 4.5 hours. In a meta-analysis published in 2015, Schrag et al.[5] found that intravenous thrombolysis administered within 4.5 hours provided a 50% chance of

visual recovery to 20/100 or better with thrombolysis, compared with 17.7% in the natural history group, with a number needed to treat of 4.0. In a recent systematic review, most studies demonstrated that very early administration of intravenous thrombolytics improved vision in patients with CRAO.[13] A survey conducted in 2018 revealed that more than half of academic centres in the United States offer intravenous thrombolytics to CRAO patients who present within the therapeutic window.[17] A recently published scientific statement from the American Heart Association underscored the variability in management of CRAO and emphasized the need for urgent assessment and treatment of vascular risk factors.[18] This same review highlighted the trend in the literature toward intravenous tissue plasminogen activator as a potentially effective treatment option for acute CRAO. In our cohort, 3 patients (11.1%) would have been eligible for fibrinolytic therapy, but local practice patterns have not evolved to actualize these recommendations.

The results of this study have been the impetus for the development of an interdisciplinary eye stroke pathway in Edmonton. We urge other Canadian ophthalmology departments to form strong relationships with emergency physicians and stroke neurologists and develop similar pathways that will optimize the care of patients with CRAO.

Conclusions

This study highlights multiple areas for improvement in the current management of ocular stroke at a Canadian centre. We demonstrate a significant burden of pre-existing and modifiable vascular disease in patients presenting with CRAO. We emphasize the need for public health awareness for patients and providers to ensure that acute monocular vision loss is referred immediately to an emergency department and not to eye care providers. Treatment algorithms in the acute care setting that involve emergency physicians, ophthalmologists, and stroke neurologists and incorporate fundus photography to enable early diagnosis may expedite care and improve visual and systemic outcomes for patients with CRAO.

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Footnotes and Disclosures

The authors have no proprietary or commercial interest in any materials discussed in this article.

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