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Case report

EFFECTIVENESS OF INTRA-ARTERIAL NIMODIPINE ON CENTRAL RETINAL ARTERY OCCLUSION

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ABSTRACT

Background: *Central Retinal Artery Occlusion (CRAO) is a rare but severe ophthalmic emergency characterized by sudden, painless vision loss. Standard treatments often have limited success. This study explores the use of intra-arterial nimodipine as a potential treatment for CRAO.*

Case report: *We present a case series of three patients diagnosed with CRAO at Hue Central Hospital. All patients had visual acuity of 1/10 and failed to respond to standard treatments including ocular massage, anterior chamber paracentesis, and hyperbaric oxygen therapy. Intra-arterial nimodipine (20 mg) was administered via the internal carotid artery. Visual acuity was assessed at baseline, immediately after treatment, at 3 days, and 1 month post-treatment. Digital Subtraction Angiography (DSA) images were obtained before and after treatment. All three patients showed immediate improvement in visual acuity following intra-arterial nimodipine treatment, with increases to 4/10 or 5/10. These improvements were sustained at 3 days and 1 month follow-up. DSA images demonstrated notable vasodilation with improved blood flow to the retinal arteries and posterior ciliary artery. The procedure was well-tolerated, with only mild and transient side effects reported.*

Conclusion: *Intra-arterial nimodipine shows promise as a treatment for CRAO, demonstrating significant improvements in visual acuity even after the failure of standard treatments. However, larger, controlled studies with longer follow-up periods are necessary to confirm its efficacy and safety before it can be considered as a standard treatment option.*

Keywords: *Central Retinal Artery Occlusion, intra-arterial nimodipine, visual acuity, vasodilator, case report.*

I. BACKGROUND

Central Retinal Artery Occlusion (CRAO) is a rare but severe ophthalmic emergency characterized by sudden, painless vision loss due to blockage of the central retinal artery. This condition leads to retinal ischemia and, if left untreated, can result in permanent vision loss [1, 2]. CRAO is often likened to a "stroke of the eye" due to the similarity in its abrupt onset and the ischemic nature of the damage [3, 4].

The incidence of CRAO is approximately 1.9 per 100,000 individuals annually in the United States, with a higher prevalence in older adults, particularly those over 80 years of age [2, 5]. Risk factors for CRAO include hypertension, diabetes, hyperlipidemia, and smoking, highlighting its association with systemic vascular diseases [6]. The condition is more common in men and is often indicative of underlying atherosclerosis, serving as a potential marker for ischemic heart disease and cerebral stroke [7].

The pathophysiology of CRAO involves the sudden blockage of the central retinal artery, which causes ischemia primarily in the inner retinal layers [8, 9]. In some cases, the presence of a cilioretinal artery can help preserve central vision by providing alternate blood flow [10]. However, without prompt intervention to restore blood flow, permanent vision loss can occur within hours.

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Standard treatments include thrombolysis, hyperbaric oxygen therapy (HBOT), and vasodilators, but the prognosis for vision recovery remains poor [5, 11]. Thrombolysis involves dissolving the clot in the central retinal artery with agents like tissue plasminogen activator (tPA), but it is controversial due to the narrow therapeutic window [12]. HBOT increases oxygen delivery to ischemic tissues but faces logistical challenges [13], while vasodilators aim to improve blood flow but are limited by the anatomy of the central retinal artery. These limitations highlight the need for new treatments.

Nimodipine, a calcium channel blocker with vasodilatory properties, is commonly used for cerebral vasospasm after subarachnoid hemorrhage [14]. Its ability to dilate blood vessels and improve microcirculation makes it a potential treatment for CRAO. Intra-arterial nimodipine can directly target the occluded retinal artery, offering a promising approach to restoring blood flow and improving visual outcome. In this report, we describe three cases of CRAO treated with intra-arterial nimodipine that demonstrated improved visual acuity and no adverse effects during treatment and follow-up.

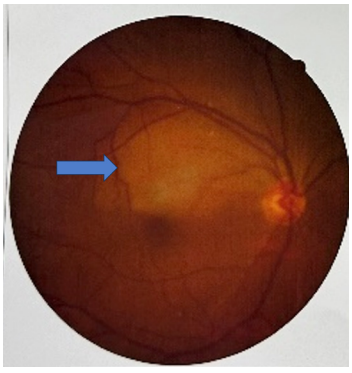
II. CASE PRESENTATION

In this report, we present three cases of central retinal artery occlusion (CRAO) that demonstrated recovery following intra-arterial nimodipine treatment.

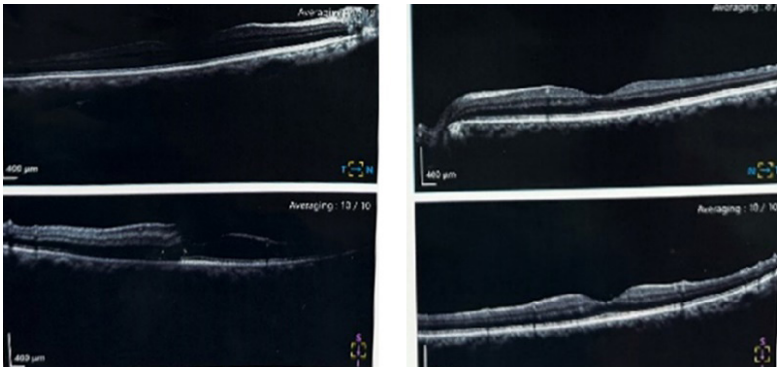
Case 1: An 81-year-old female presented with acute onset of blurred vision and vision loss in her right eye, occurring six hours prior to admission. The patient had a history of hypertension but no prior visual complaints. On examination, visual acuity in the right eye was 1/10 (decimal chart), with significant visual field loss in the lower half (hand motion only). Fundoscopic examination revealed retinal pallor in the upper temporal region (A), and optical coherence tomography (OCT) indicated para-retinal macular edema in the right eye (B). Visual acuity in the left eye was 8/10 with no abnormalities detected. Laboratory tests showed dyslipidemia with elevated triglycerides, while other parameters were within normal limits. MRI of the brain showed no lesions related to the visual areas.

Diagnosis: Incomplete central retinal artery occlusion (CRAO) in the right eye.

Standard treatments including ocular massage, anterior chamber paracentesis, and hyperbaric oxygen therapy were administered, but no improvement in visual acuity was observed after 10 hours. Therefore, intra-arterial infusion of nimodipine was initiated. Nimodipine at a concentration of 0.02 mg/mL was infused at the rate of 10 mg in 20 minutes via catheter into the right internal carotid artery. A total dose of 20 mg was administered in 40 minutes.



A



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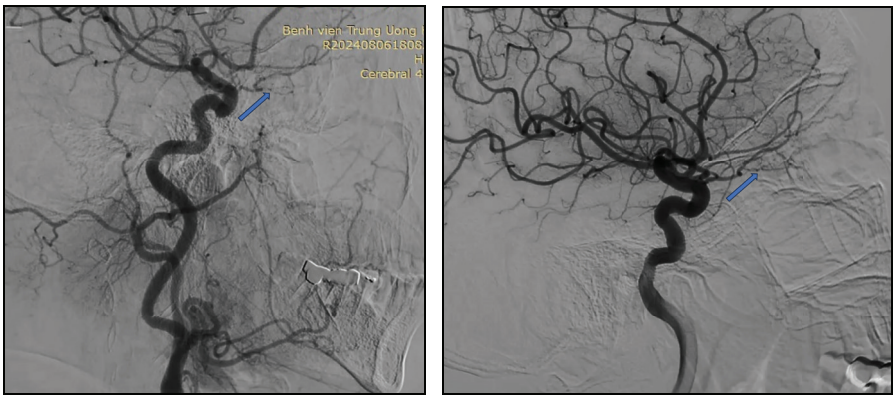


C D

Figure 1: (A) Fundoscopic; (B) OCT; (C) Before infusion: atherosclerosis of the central retinal artery, consistent with the previous diagnosis of incomplete central retinal artery occlusion; (D) Post-treatment angiography demonstrated improved visualization of the ophthalmic, central retinal arteries and posterior ciliary artery, an important collateral branch to the retina

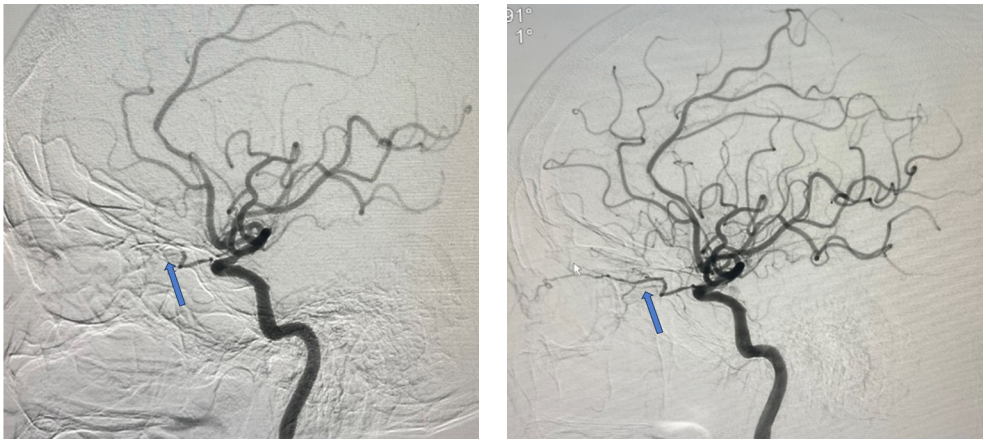
The patient reported no headache during the procedure, and blood pressure remained within normal limits despite a slight reduction.

Outcome: Immediate improvement in visual acuity to 4/10 with the ability to count fingers (CF). At three days post-treatment, visual acuity remained at 4/10. At 1 month post-treatment, visual acuity remained at 4/10.

Case 2: A 61-year-old female was admitted with sudden vision loss in her left eye, occurring 12 hours prior to presentation. She had a history of hypertension and dyslipidemia. Examination revealed a visual acuity of 1/10 in the left eye (hand motion only) with near-complete visual field loss. Fundoscopic examination showed retinal pallor in the affected eye. Visual acuity in the right eye was 9/10, and the examination was unremarkable. Laboratory tests showed elevated LDL-C levels, with other results within normal limits. MRI of the brain showed no lesions related to the visual areas.

Diagnosis: Subtotal central retinal artery occlusion (CRAO) in the left eye.

Standard treatments including ocular massage, anterior chamber paracentesis, and hyperbaric oxygen therapy were administered, but no improvement in visual acuity was observed after 12 hours. Therefore, intra-arterial infusion of nimodipine was initiated. Nimodipine at a concentration of 0.02 mg/mL was infused at the rate of 10 mg in 15 minutes via catheter into the left internal carotid artery. A total dose of 20 mg was administered in 30 minutes.



A B

Figure 2: (A) Before infusion: Image showed the central retinal artery not clearly, consistent with the previous diagnosis of incomplete central retinal artery occlusion; (B) Post-treatment imaging revealed

clearer visualization of the central retinal artery and ophthalmic artery, especially increased perfusion from the posterior ciliary artery, an important collateral branch to the retina.

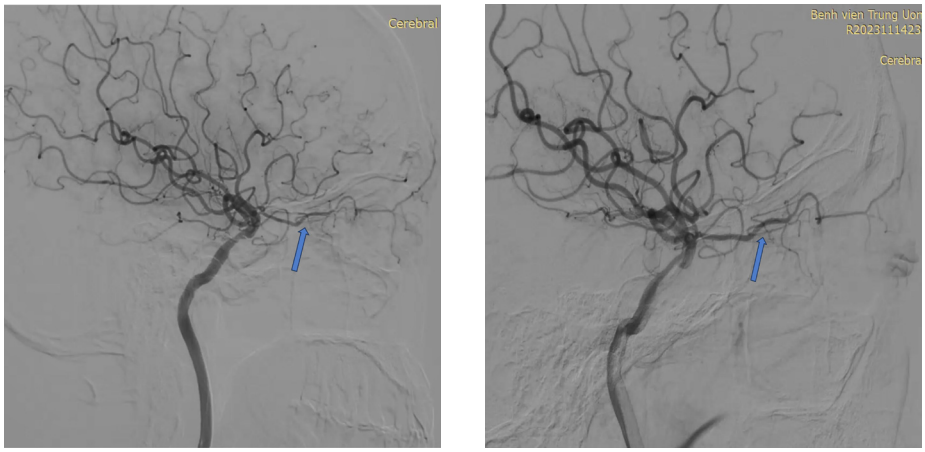
The patient experienced a mild headache during the procedure, and blood pressure remained stable.

Outcome: Visual acuity improved to 5/10 with the ability to count fingers. At three days post-treatment, visual acuity remained at 5/10. At 1 month post-treatment, visual acuity remained at 5/10.

Case 3: A 69-year-old male presented with sudden blurred vision and vision loss in the right eye, eight hours prior to admission. The patient had a history of hypertension, dyslipidemia, and diabetes. There was no previous history of CRAO in the affected eye. Examination showed visual acuity of 1/10 in the right eye (can count fingers) with loss of the upper visual field. Fundoscopic examination revealed retinal pallor in the right eye, and visual acuity in the left eye was 9/10 with no abnormalities noted. Laboratory tests showed elevated LDL-C levels and hyperglycemic, with other results within normal limits. MRI of the brain showed no lesions related to the visual areas.

Diagnosis: Incomplete central retinal artery occlusion (CRAO) in the right eye.

Standard treatments including ocular massage, anterior chamber paracentesis, and hyperbaric oxygen therapy were administered, but no improvement in visual acuity was observed after 10 hours. Therefore, intra-arterial infusion of nimodipine was initiated. Nimodipine at a concentration of 0.02 mg/mL was infused at the rate of 10 mg in 30 minutes via catheter into the right internal carotid artery. A total dose of 20 mg delivered in 60 minutes due to a decrease in blood pressure during the procedure.



A B

Figure 3: (A) Before infusion: image showed atherosclerosis at the origin and cavernous sinus segment of internal carotid artery, central retinal artery still be seen; (B) Post-treatment angiography demonstrated clearer visualization of the central retinal artery.

The patient tolerated the procedure without headaches.

Outcome: Visual acuity improved to 5/10 immediately post-treatment. Three days after treatment, visual acuity remained at 5/10. At 1 month post-treatment, visual acuity remained at 4/10.

III. DISCUSSION

In this study, we presented three cases of central retinal artery occlusion (CRAO) treated with intra-arterial nimodipine. The outcomes in these cases suggest that nimodipine may play a significant role in improving visual acuity and restoring retinal perfusion in patients with CRAO, even after the failure of standard treatments.

All three patients demonstrated immediate improvement in visual acuity following intra-arterial nimodipine treatment. Visual acuity improved from 1/10 to 4/10 or 5/10 post-treatment, with these improvements sustained at three days and one month follow-up. This is particularly noteworthy given that these improvements occurred after standard treatments (ocular massage, anterior

chamber paracentesis, and hyperbaric oxygen therapy) had failed to produce any improvement over several hours.

Time window: It's important to note that these improvements were observed in patients treated within different time windows after symptom onset - 6 hours, 12 hours, and 8 hours respectively. This suggests that intra-arterial nimodipine might be effective even when administered several hours after the onset of symptoms, potentially extending the treatment window for CRAO. This extended window could be crucial, as recent studies have shown that the traditional 6-hour window for CRAO treatment may be too restrictive [15].

The use of the adjunctive intra-arterial vasodilating agent in the present study was based on the hypothesis that nimodipine may have a direct vasodilating effect on ophthalmic and retina arteries, which might improve retinal perfusion and dislodge emboli to more-peripheral areas. However, there was no solid evidence of distal propagation of retinal emboli to support the hypothesis. The Digital Subtraction Angiography (DSA) images in all three cases showed notable vasodilation with marked improvement in blood flow to the retinal arteries and posterior ciliary artery. This supports the hypothesis that nimodipine may have a direct vasodilating effect on ophthalmic and retinal arteries, improving retinal perfusion. Particularly in Case 2, post-infusion images revealed the reappearance of the posterior ciliary artery, indicating enhanced perfusion to the ischemic retina. The vasodilatory effect of nimodipine on cerebral arteries has been well-documented [16], and our findings suggest a similar effect on retinal vascular.

The safety profile of intra-arterial nimodipine, as observed in this study, was generally favorable. Patients tolerated the procedure well, with only mild and transient adverse effects such as a slight drop in blood pressure or a mild headache during the infusion. No severe complications were reported. This aligns with the known safety profile of nimodipine in the treatment of cerebral vasospasm, where only minor side effects like headache, vertigo, flushing, nausea, diarrhea, and rash have been commonly reported [17].

Existing research on CRAO treatment has explored modalities such as thrombolysis, hyperbaric oxygen therapy, and vasodilators, but none have consistently shown significant improvements in visual outcomes [7]. For example, thrombolysis, which aims to dissolve the clot obstructing the retinal artery, has shown mixed results and is limited by a narrow therapeutic window [12]. Intra-arterial nimodipine presents a promising approach for CRAO by directly inducing vasodilation and improving microcirculation, with outcomes in this study suggesting potential efficacy even after the failure of standard treatments.

However, it's crucial to acknowledge the limitations of this study. The small sample size of three patients, lack of a control group, and single-center design limit the generalizability of the findings and may introduce bias. The short follow-up period of one month also restricts our ability to assess the long-term safety and efficacy of this treatment approach. These limitations are common in early-stage research on novel treatments for rare conditions like CRAO. In order to address these limitations, future research should focus on larger, controlled studies with longer follow-up periods. It would be beneficial to evaluate visual acuity and retinal perfusion over an extended period, perhaps up to 6 months or a year post-treatment. Additional imaging techniques such as fundus fluorescein angiography (FFA) and optical coherence tomography (OCT) could provide more detailed information about retinal perfusion and structure following treatment. Moreover, standardization of visual acuity measurement using Best Corrected Visual Acuity (BCVA) and logMAR charts instead of decimal charts would provide more accurate and comparable results across studies.

Finally, exploring the use of microcatheters to deliver nimodipine directly into the ophthalmic artery could offer even more precise targeting of the ischemic retina [18]. Additionally, investigating the potential synergistic effects of combining intra-arterial nimodipine with thrombolysis could provide valuable insights into optimizing treatment for CRAO.

IV. CONCLUSION

While these case reports provide promising initial evidence for the use of intra-arterial nimodipine in CRAO treatment, well-designed randomized controlled trials are necessary to confirm its efficacy and safety before it can be considered as a standard treatment option.

Competing Interests

The authors declare no competing interests related to this study.

REFERENCES

1. Chen C, Singh G, Madike R, Cugati S. Central retinal artery occlusion: a stroke of the eye. Eye. 2024: 1-8.
2. Leavitt JA, Larson TA, Hodge DO, Gullerud RE. The incidence of central retinal artery occlusion in Olmsted County, Minnesota. American journal of ophthalmology. 2011; 152(5): 820-823. e2.
3. Biousse V, Nahab F, Newman NJ. Management of acute retinal ischemia: follow the guidelines! Ophthalmology. 2018; 125(10): 1597-1607.
4. Hayreh SS, Zimmerman MB. Central retinal artery occlusion: visual outcome. American journal of ophthalmology. 2005; 140(3): 376. e1-376. e.
5. Park SJ, Choi N-K, Seo KH, Park KH, Woo SJ. Nationwide incidence of clinically diagnosed central retinal artery occlusion in Korea, 2008 to 2011. Ophthalmology. 2014; 121(10): 1933-1938.
6. Hayreh SS, Hayreh SS. Central retinal artery occlusion. Ocular Vascular Occlusive Disorders. 2015: 239-305.
7. Dumitrascu OM, Newman NJ, Biousse V. Thrombolysis for central retinal artery occlusion in 2020: time is vision! Journal of Neuro-Ophthalmology. 2020; 40(3): 333-345.
8. Masters TC, Westgard BC, Hendriksen SM, Decanini A, Abel AS, Logue CJ, et al. Case series of hyperbaric oxygen therapy for central retinal artery occlusion. Retinal Cases and Brief Reports. 2021; 15(6): 783-788.
9. Schrag M, Youn T, Schindler J, Kirshner H, Greer D. Intravenous fibrinolytic therapy in central retinal artery occlusion: a patient-level meta-analysis. JAMA neurology. 2015; 72(10): 1148-1154.
10. Olson EA, Lentz K. Central retinal artery occlusion: a literature review and the rationale for hyperbaric oxygen therapy. Missouri Medicine. 2016; 113(1): 53.
11. Sim S, Ting D. Diagnosis and management of central retinal artery occlusion. EyeNet Magazine. 2017: 33-34.
12. Schumacher M, Schmidt D, Jurklies B, Gall C, Wanke I, Schmoor C, et al. Central retinal artery occlusion: local intra-arterial fibrinolysis versus conservative treatment, a multicenter randomized trial. Ophthalmology. 2010; 117(7): 1367-1375. e1.
13. Hadanny A, Maliar A, Fishlev G, Bechor Y, Bergan J, Friedman M, et al. Reversibility of retinal ischemia due to central retinal artery occlusion by hyperbaric oxygen. Clinical Ophthalmology. 2016: 115-125.
14. Biondi A, Ricciardi GK, Puybasset L, Abdennour L, Longo M, Chiras J, et al. Intra-arterial nimodipine for the treatment of symptomatic cerebral vasospasm after aneurysmal subarachnoid hemorrhage: preliminary results. American Journal of Neuroradiology. 2004; 25(6): 1067-1076.
15. Chronopoulos A, Schutz JS. Central retinal artery occlusion - A new, provisional treatment approach. Survey of ophthalmology. 2019; 64(4): 443-451.
16. Carlson AP, Hänggi D, Macdonald RL, Shuttleworth CW. Nimodipine reappraised: an old drug with a future. Current neuropharmacology. 2020; 18(1): 65-82.
17. Diringer MN, Bleck TP, Claude Hemphill J, Menon D, Shutter L, Vespa P, et al. Critical care management of patients following aneurysmal subarachnoid hemorrhage: recommendations from the Neurocritical Care Society's Multidisciplinary Consensus Conference. Neurocritical care. 2011; 15: 211-240.
18. Schultheiss M, Härtig F, Spitzer MS, Feltgen N, Spitzer B, Hüsing J, et al. Intravenous thrombolysis in acute central retinal artery occlusion–a prospective interventional case series. PloS one. 2018; 13(5): e0198114.