ASRS American Society of Retina Specialists

Journal of VitreoRetinal Diseases
2025, Vol. 9(2) 257–261

DOI: 10.1177/24741264241302870
journals.sagepub.com/home/jvrd
Sage

Tractional Retinal Detachment in a Patient With a History of Methamphetamine Use

Angela J. Kim, MD[1,2], Omar Sabbagh[3], Osama Sabbagh, MD[1,2,4], Michelle Abou-Jaoude, MD[1,2], and John W. Kitchens, MD[2]

Abstract

Purpose: To describe the development of tractional retinal detachments (TRD) in a patient with diabetic retinopathy (DR) and a history of methamphetamine use. **Methods:** A single case was evaluated. **Results:** A 38-year-old man with type 2 diabetes mellitus presented to the clinic with worsening blurry vision in both eyes. A fundus examination, fundus photography, and optical coherence tomography were performed. The patient's bilateral TRDs were attributed to poorly controlled DR; thus, he was administered 1 intravitreal aflibercept injection in each eye and scheduled for surgery. His glycosylated hemoglobin A_{1c} was only 6.3% and was controlled with metformin. In the interim, the patient was admitted for a stroke workup and diagnosed with Moyamoya disease and methamphetamine abuse. **Conclusions:** In patients with underlying DR and presumed Moyamoya disease, TRDs can be exacerbated by methamphetamine use.

Keywords

tractional retinal detachment, Moyamoya disease, methamphetamine, ischemic retinopathy, diabetic retinopathy

Introduction

One of the most common etiologies of tractional retinal detachment (TRD) is proliferative diabetic retinopathy (PDR). Chronic ischemia results in an increase in vascular endothelial growth factor (VEGF), which stimulates neovascularization (NV) and subsequent fibrovascular complexes, placing tractional and shearing forces on the neurosensory retina.[1]

We present a patient with underlying diabetic retinal disease who developed bilateral TRDs. Multiple comorbidities, especially unilateral Moyamoya disease and chronic methamphetamine abuse, could contribute to chronic vasoconstriction and exacerbate the condition.

Case Report

A 38-year-old Black man without an ocular history presented to the clinic with progressive painless decreased vision in both eyes for 3 weeks. His medical history was significant for type 2 diabetes mellitus, hypertension, obesity, and alcohol and tobacco dependence. Medications included metformin, escitalopram, and varenicline. A review of the patient's systems was unremarkable. The visual acuity (VA) was 3/200 OD and 20/150 OS, and the intraocular pressure was 17 mm Hg and 19 mm Hg, respectively. The pupils were symmetric and equally round and reactive without an afferent defect. The external and anterior segment examinations were significant for a mild cataract in both eyes. However, a fundus examination and optical coherence tomography of both maculas (Figures 1 and 2) showed bilateral and symmetric macula-off TRDs, extensive NV overlying the nerves, an old vitreous hemorrhage, and macular edema. The periphery was flat without tears or breaks in either eye.

The patient was given a working diagnosis of PDR and administered intravitreal (IVT) aflibercept injections in both eyes. Plans were made for surgical intervention, beginning with the left eye. The surgery was without complications. One week postoperatively, the patient's VA in the left eye remained 20/150 with resolution of the TRD (Figure 3).

On postoperative day 10, the patient was admitted to the hospital for new left upper extremity hemiplegia and right facial droop and was found to have multiple acute cerebrovascular events involving the right middle cerebral artery. He was outside the window of time for receiving treatment with tissue plasminogen activator. An extensive stroke workup was unremarkable and included thyroid-stimulating hormone, factor V Leiden, prothrombin mutation, anticardiolipin, lupus anticoagulant, protein

[1] Department of Ophthalmology, University of Kentucky, Lexington, KY, USA
[2] Retina Associates of Kentucky, Lexington, KY, USA
[3] West Virginia University, Morgantown, WV, USA
[4] Retina Group of Washington, Greenbelt, MD, USA

Corresponding Author:
Angela J. Kim, MD, University of Kentucky Ophthalmology, 110 Conn Terr, Shriner's Bldg, Lexington, KY 40508, USA.
Email: Angela.kim@retinatexas.com

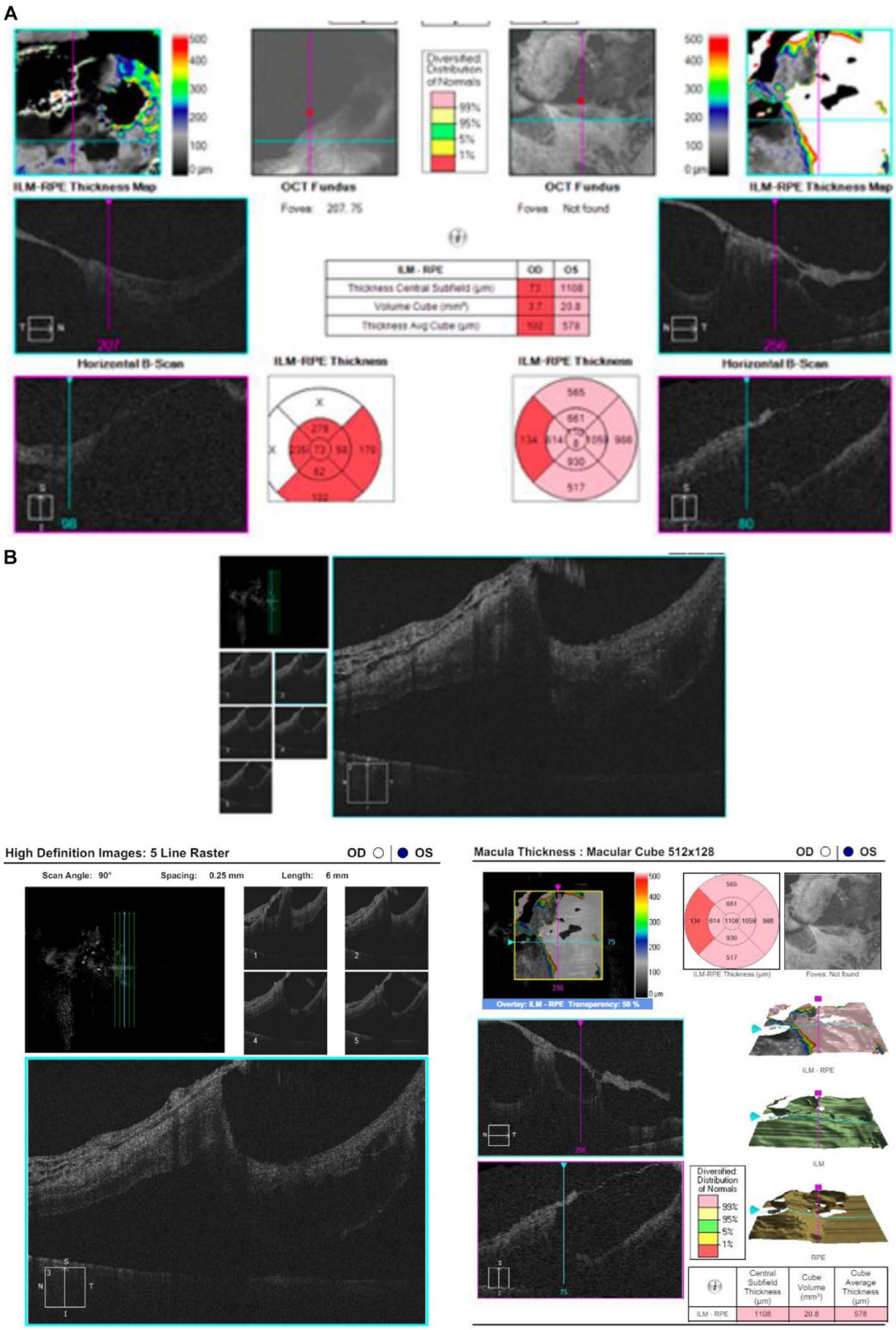

Figure 1. (A) OCT of both maculas shows detachment with subfoveal traction. (B) OCT of the left macula.
Abbreviations: ILM, internal limiting membrane; OCT, optical coherence tomography; RPE, retinal pigment epithelium.

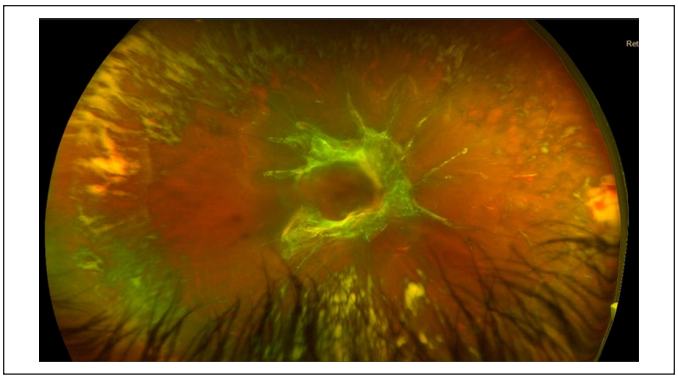

Figure 2. Preoperative fundus photograph of the right eye.

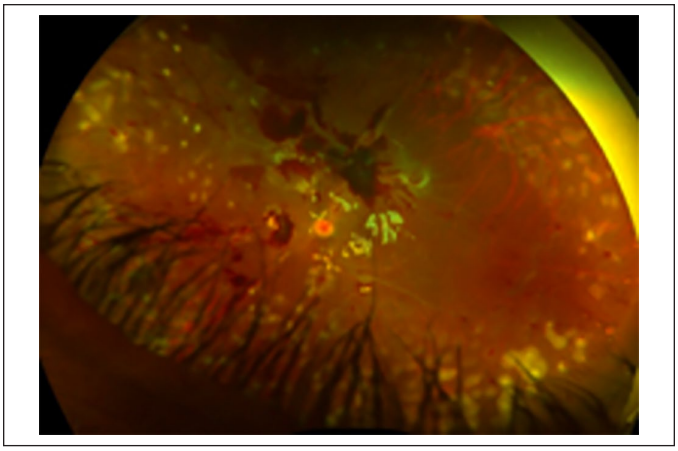

Figure 3. Left eye 1 week postoperatively. Unfortunately, no preoperative fundus photograph was available.

C and S activity, antithrombin III, factor VIII, rheumatoid factor, antinuclear antibody, C-reactive protein, erythrocyte sedimentation rate, double-stranded DNA, smooth muscle antibody, antineutrophilic cytoplasmic antibody, lipid panel, and an echocardiogram with bubble study.

Computed tomography angiography of the head and neck did not show significant stenosis of the cervical carotid arteries or cervical vertebral arteries but did show occlusion of the right middle cerebral artery and narrowing of the right anterior cerebral artery. No high-grade stenosis, occlusion, or aneurysm was noted in the remaining major intracranial arteries. Magnetic resonance imaging of the brain (Figure 4) showed acute infarcts in the right cerebral hemisphere along the middle cerebral artery territory. Cerebral angiography (Figure 5) showed complete, chronic-appearing occlusion of the right internal carotid artery, given the small wispy collaterals emerging from the distal right internal carotid artery and robust collaterals from the posterior circulation. The left distal internal carotid artery and proximal branches were unremarkable. These findings were consistent with the diagnosis of Moyamoya syndrome.

Further workup showed a glycosylated hemoglobin A_{1c} (HbA_{1c}) of 6.3%, and urine drug testing was positive for methamphetamines. Over the past 3 years, the patient's HbA_{1c} had ranged from 4.9% to 7.6%. The patient was discharged on aspirin 81 mg, clopidogrel 75 mg, and atorvastatin. Several months later, he had uneventful right-sided encephaloduroarteriosynangiosis with neurosurgery as an outpatient. Subsequently, the patient was lost to follow-up. To our knowledge, this is the first case report of bilateral TRDs related to methamphetamine use in addition to concomitant Moyamoya disease.

Conclusions

Moyamoya disease is an idiopathic vasoconstrictive and occlusive syndrome with a peak prevalence in the second decade of life in men or the third decade in women.[2] The syndrome, which results from the progressive stenosis of the branches of the Circle of Willis, was named by Japanese researchers, who noted unique "puff of smoke" collaterals on cerebral angiography, which is the primary mode of diagnosis.[3] Patients present with stroke-like symptoms resulting from recurrent cerebral ischemia and hemorrhages. Ocular manifestations noted in case reports include amaurosis fugax, central retinal artery occlusion, ocular ischemic syndrome, and morning glory disc anomaly.[3]

Although initially presumed to be a typical case of PDR, our patient's vascular abnormalities were less straightforward. He had fibrovascular membranes and retinal hemorrhages, yet his HbA_{1c} of 6.3% could not explain his severe ischemic DR. The diagnosis of Moyamoya disease and the likelihood of ocular ischemic syndrome only partly explain the patient's bilateral retinopathy because imaging showed cerebral vessel involvement on the right side only. One possible explanation for the bilateral ischemic retinopathy is his concomitant use of methamphetamine, a synthetic amine that can be ingested, inhaled, or injected, inducing both acute and chronic effects on the central and peripheral nervous systems by raising the levels of monoamine neurotransmitters. Methamphetamine has been reported to cause cerebral vasculitis, leading to ischemic or hemorrhagic cerebral infarctions in young patients through repeated vasospasms, arterial narrowing, and intracranial arterial beading.[4]

One other case of methamphetamine use in a patient diagnosed with Moyamoya disease has been reported. A 31-year-old woman developed similar stroke-like symptoms, including weakness in the right arm, speech disturbance, and seizures; however, no vision symptoms were reported.[5] The stroke and Moyamoya disease were confirmed on imaging. Moyamoya disease was diagnosed in the patient in that case. The disease is likely multifactorial, with chronic cerebrovascular changes lowering the threshold for acute stroke with the use of sympathetic substances, such as cocaine.[6] In our patient, however, the symptoms were brought on by the use of methamphetamine.

Ocular manifestations associated with methamphetamine use include episcleritis, scleritis, corneal ulceration by way of neurotoxicity, and retinal vasculitis resembling that seen in polyarteritis nodosa.[7] Few individual cases have been published that report bilateral ischemic retinopathy in the presence of methamphetamine use, with a potential mechanism being the production of oxidative stress that in turn causes vasoconstriction

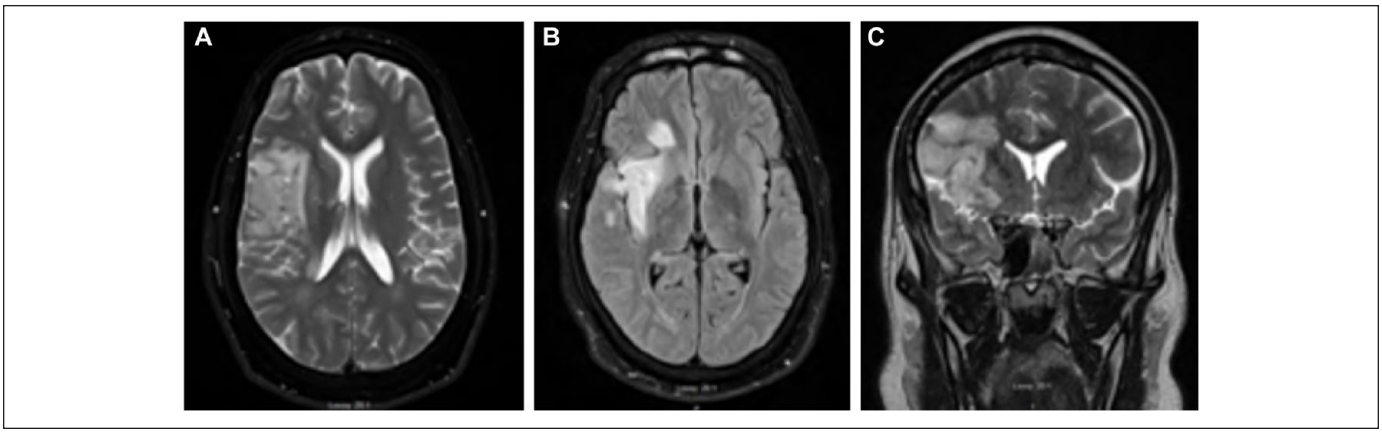

Figure 4. (A and B) Magnetic resonance imaging of the brain. Axial section shows an acute right middle cerebral artery infarct. (C) Coronal section shows corresponding middle cerebral distribution.

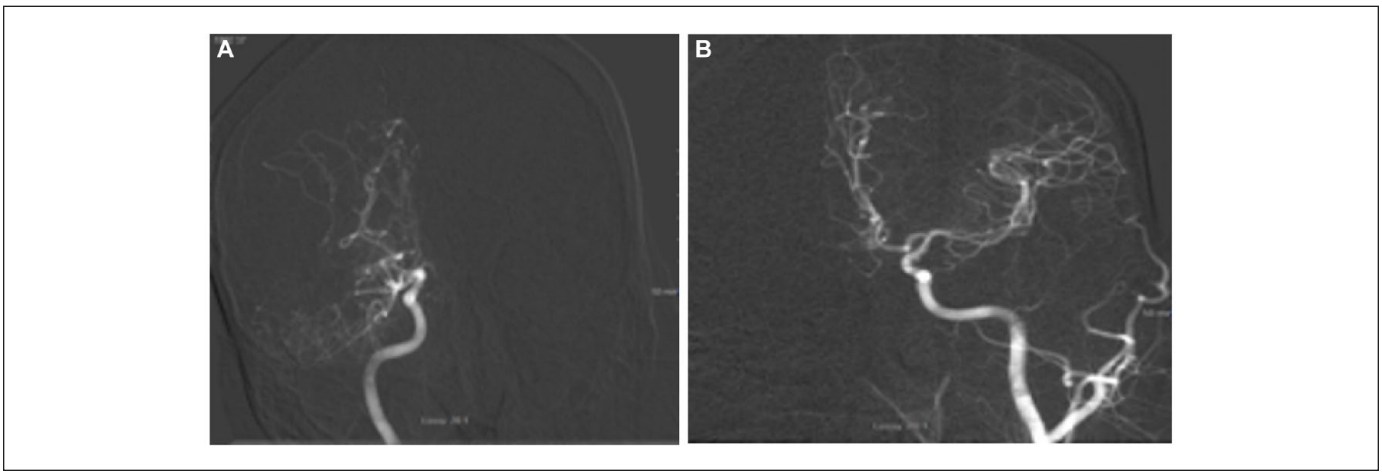

Figure 5. (A) Cerebral angiography of the right intracranial vasculature. The diagnosis of Moyamoya disease was based on complete right internal carotid artery terminus occlusion with collaterals suggestive of chronicity. (B) The left internal carotid and branches appear patent.

and vasospasms, leading to ischemia and NV. Although the pathophysiology remains unclear, Wallace et al[8] reported a patient who used intranasal methamphetamine for 7 years. OCT angiography showed dropout of both superficial and deep capillary plexuses within the neurosensory retina. An in vivo study measuring inflammatory markers and retinal proteins in mice after administration of methamphetamine showed vascular loss of platelet endothelial cell adhesion molecule-1 and glycocalyx in the central retinal artery and an increase in several matrix metalloproteinases in vessel walls. They postulated that methamphetamine is involved in retinal degeneration by way of vascular endothelial wall dysfunction.[9] Other reported retinal manifestations include central retinal artery or vein occlusions, intraretinal hemorrhages, and retinal vasculitis, all of which are attributed to vascular spasms.[10]

Treatment options specific to proliferative retinopathy resulting from methamphetamine use have not been reported. Based on the pathophysiology of NV and TRD, however, treatment could include the use of IVT anti-VEGF injections to control NV, followed by release of traction via vitrectomy and peeling fibrous membranes and then reopposing the retina with a tamponade agent such as silicone oil, similar to how one would treat diabetic proliferative changes. This approach seemed to have worked well for our patient (with limited follow-up). In addition to ophthalmology, multidisciplinary coordination of care involving addiction medicine to help with substance abuse and a primary care team to optimize vascular comorbidities is recommended.

There are limitations to this report. The patient was inconsistent regarding the duration of methamphetamine inhalation, but documentation by neurology noted use of only 4 or 5 months before his first admission. If this were accurate, it is unclear whether there was sufficient time for methamphetamine-induced bilateral TRDs to develop. In addition, one cannot rule out that the presence of venous occlusive events, which could have occurred

concomitantly with methamphetamine use, led to eventual TRDs. Obtaining a complete history is necessary to further understand and properly treat patients dealing with these same conditions. Future studies should provide education about the dangers of abusing methamphetamine, and the development of TRDs can be included as a serious possible side effect of the drug.

In conclusion, our patient's underlying diabetic retinal disease was likely exacerbated by his presumed Moyamoya disease, concomitant stimulant use, and other poorly controlled vascular comorbidities, all of which contribute to chronic retinal ischemia.

Ethical Approval

This case report was conducted in accordance with the tenets of the Declaration of Helsinki. The collection and evaluation of all protected patient health information were performed in a US Health Insurance Portability and Accountably Act–compliant manner.

Statement of Informed Consent

Informed consent was obtained before the procedure was performed. Informed consent was not obtained for publication because the patient was lost to follow-up.

Declaration of Conflicting Interest

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

ORCID iD

Omar Sabbagh https://orcid.org/0009-0008-3443-329X

References

1. Ghazi NG, Green WR. Pathology and pathogenesis of retinal detachment. *Eye (Lond)*. 2002;16(4):411-421.
2. Kuriyama S, Kusaka Y, Fujimura M, et al. Prevalence and clinico-epidemiological features of Moyamoya disease in Japan: findings from a nationwide epidemiological survey. *Stroke*. 2008;39(1):42-47. doi:10.1161/STROKEAHA.107.490714
3. Güçlü H, Gurlu VP, Ozal SA, Esgin H. A Moyamoya patient with bilateral consecutive branch retinal vein occlusion. *Neuroophthalmology*. 2016;40(2):93-96. doi:10.3109/01658107.2016.1148174
4. Storen EC, Wijdicks EF, Crum BA, Schultz G. Moyamoya-like vasculopathy from cocaine dependency. *AJNR Am J Neuroradiol*. 2000;21(6):1008-1010.
5. Lin T, Yam C, Lai S-L, Cloud G. Moyamoya vasculopathy in a young Caucasian woman with significant methamphetamine use. *BMJ Neurol Open*. 2020;2(1):e000066. doi:10.1136/bmjno-2020-000066
6. Unger MD, Georges J, Shaikh HA, Kavi T. Moyamoya tipping point: fatal bilateral MCA territory infarction following cocaine abuse. *BMJ Case Rep*. 2018;2018:bcr-2017-222883. doi:10.1136/bcr-2017-222883
7. Hazin R, Cadet JL, Kahook MY, Saed D. Ocular manifestations of crystal methamphetamine use. *Neurotox Res*. 2009;15(2):187-191. doi:10.1007/s12640-009-9019-z
8. Wallace RT, Brown GC, Benson W, Sivalingham A. Sudden retinal manifestations of intranasal cocaine and methamphetamine abuse. *Am J Ophthalmol*. 1992;114(2):158-160. doi:10.1016/S0002-9394(14)73979-0
9. Lee M, Leskova W, Eshaq RS, Harris NR. Acute changes in the retina and central retinal artery with methamphetamine. *Exp Eye Res*. 2020;193:107964. doi:10.1016/j.exer.2020.107964
10. Guo J, Tang W, Liu W, Zhang Y, Wang L, Wang W. Bilateral methamphetamine-induced ischemic retinopathy. *Am J Ophthalmol Case Rep*. 2019;15:100473. doi:10.1016/j.ajoc.2019.100473