Cureus
Part of SPRINGER NATURE

Open Access Case Report

Bilateral Choroidal Effusions With Acute Myopic Shift After Daratumumab

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Review began 07/23/2026
Review ended 11/10/2026
Published 11/18/2026

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DOI: 10.7759/cureus.119912

Abstract

Daratumumab is a CD38-directed monoclonal antibody increasingly used in haematological malignancies. Infusion-related reactions are well recognised, but ocular adverse events are uncommon and may be under-recognised. We report a man in his 30s with relapsed T-cell lymphoblastic lymphoma/leukaemia following allogeneic stem cell transplantation who developed acute bilateral blurred vision within 24 hours of his first daratumumab infusion. The infusion was complicated by a hypersensitivity reaction requiring treatment with antihistamines and corticosteroids. Ophthalmic assessment demonstrated unaided visual acuity of 6/9 in the right eye and 6/15 in the left eye, bilateral myopic refractive changes, normal intraocular pressures, and deep, quiet anterior chambers. Optical coherence tomography was unremarkable, whilst B-scan ultrasonography demonstrated small bilateral superior choroidal effusions. Following discussion with the treating haematology team, the patient was managed conservatively with ophthalmic monitoring whilst daratumumab was continued. At two weeks, his visual symptoms had resolved, unaided visual acuity had improved to approximately 6/4.5 bilaterally, and repeat ultrasonography confirmed complete resolution of the effusions. At approximately nine weeks, visual recovery was sustained, with near-plano autorefraction and no recurrent effusions. Daratumumab had subsequently been discontinued for unrelated haematological reasons. This case highlights ciliochoroidal effusion as a potential cause of acute myopic refractive change following daratumumab administration. Although the temporal association raises suspicion of drug-related ocular toxicity, causality cannot be established from a single case. Resolution during continued treatment illustrates a comparatively mild presentation but should not be interpreted as evidence that daratumumab can routinely be continued following ocular adverse events.

Categories: Pharmacology, Hematology, Ophthalmology
Keywords: acute myopic shift, angle closure glaucoma, choroidal effusion, ciliochoroidal effusion, daratumumab, monoclonal antibody, ocular adverse event

Introduction

Daratumumab is a CD38-directed monoclonal antibody used predominantly in the treatment of plasma cell malignancies, particularly multiple myeloma [1]. CD38 expression has also been demonstrated on malignant T-cell lymphoblasts, providing a rationale for its off-label use in selected patients with relapsed or refractory T-cell acute lymphoblastic leukaemia/lymphoma (T-ALL/T-LBL), although supporting clinical evidence remains limited [2]. Infusion-related reactions are well recognised, but ocular adverse events, including acute myopic shift, choroidal effusion, and secondary angle closure, are less commonly reported [3,4]. Acute visual symptoms may initially be attributed to concomitant medications, such as antihistamines, potentially delaying recognition of ocular toxicity.

Ciliochoroidal effusion can produce acute refractive changes through anterior rotation of the ciliary body and forward displacement of the lens-iris diaphragm. This may result in a transient myopic shift, or sudden increase in nearsightedness, and in more severe cases, narrowing of the iridocorneal drainage angle with secondary angle closure [3,5]. Recognition of this mechanism is particularly important in immunocompromised patients, in whom acute bilateral blurred vision may otherwise prompt extensive investigation for inflammatory, infectious, macular, or neuro-ophthalmic pathology. We report a case of small bilateral choroidal effusions and acute myopic refractive change occurring within 24 hours of daratumumab initiation for relapsed CD38-positive T-ALL/T-LBL. The effusions resolved without specific ocular treatment despite continued daratumumab administration, highlighting a comparatively mild presentation within the reported spectrum of ocular complications.

Case Presentation

A man in his 30s with relapsed T-ALL/T-LBL following allogeneic stem cell transplantation was referred for urgent ophthalmic assessment after developing acute bilateral blurred vision within 24 hours of his first daratumumab infusion. He had initially achieved remission following Hyper-CVAD chemotherapy and underwent allogeneic stem cell transplantation approximately eight months before presentation. Bone marrow assessment six months after transplantation demonstrated disease relapse, with 5%-10% lymphoblasts on morphological examination and approximately 8% abnormal T-cell precursors on flow cytometry, which demonstrated CD38 expression. His disease subsequently progressed despite treatment

with pegylated interferon and imatinib and remained refractory to FLAG salvage chemotherapy. Given the limited remaining treatment options, daratumumab was commenced as off-label CD38-directed salvage therapy.

The first intravenous daratumumab infusion was prescribed at 16 mg/kg (1,100 mg). Premedication consisted of oral loratadine, paracetamol, and intravenous dexamethasone 20 mg. The infusion commenced at 50 mL/hour and was increased to 100 mL/hour after one hour. Shortly after the rate increase, the patient developed chest and throat tightness, urticaria, and breathlessness, consistent with an infusion-related hypersensitivity reaction. The infusion was interrupted after approximately 67.7 mL had been administered. Intravenous hydrocortisone 100 mg, intravenous promethazine 12.5 mg, and nebulised salbutamol were administered, with subsequent resolution of the acute symptoms. Daratumumab was restarted at 50 mL/hour following clinical improvement. The precise total infusion duration was not available from the retained records.

The patient reported bilateral blurred vision beginning on the day of daratumumab administration and persisting the following morning. The visual disturbance had initially been attributed to promethazine. He described marked blurring of distance vision and a mild sensation of ocular swelling, without significant pain, photophobia, or diplopia. He denied any previous ocular history. His medical background included gastrointestinal graft-versus-host disease, previous severe enterocolitis, a treated possible invasive fungal infection, prior bloodstream infection, and ongoing poor graft function requiring blood transfusions.

At the initial ophthalmic assessment, unaided Snellen visual acuity was 6/9 in the right eye and 6/15 in the left eye. Pinhole acuity was 6/9 in the right eye and 6/7.5 + 1 in the left eye. Trial-frame correction improved left-eye acuity to 6/6 but did not improve right-eye acuity beyond 6/9. Autorefraction demonstrated −1.75/−0.25 × 164 in the right eye and −1.00/−0.25 × 80 in the left eye. Pretreatment visual acuity and refractive measurements were unavailable, and cycloplegic refraction was not documented. The patient reported no previous spectacle use. The acute onset of blurred vision and subsequent improvement in refraction and unaided visual acuity supported a transient myopic change, although its magnitude relative to the patient's baseline could not be established. Contemporaneous blood tests demonstrated mild hypoalbuminaemia (30 g/L; reference range 35-50 g/L) and reduced total protein (53 g/L; reference range 60-80 g/L), with preserved renal function and normal serum sodium. Serum albumin had been 36 g/L six days before daratumumab administration, decreased to 31 g/L on the day of infusion, and returned to 36 g/L three days after the initial ophthalmic assessment.

Intraocular pressure measured using iCare tonometry was 10 mmHg bilaterally. Pupils were equal and reactive, without a relative afferent pupillary defect. Ocular movements were full and painless. Ishihara colour vision testing was 17/17 in the right eye and 16/17 in the left eye, whilst Humphrey 30-2 visual fields showed no significant defects. Slit-lamp examination demonstrated clear corneas and deep, quiet anterior chambers bilaterally, with a small temporal subconjunctival haemorrhage in the right eye. Dilated fundus examination revealed no vitritis, retinal infiltrates, haemorrhage, or optic disc swelling.

Optical coherence tomography (OCT) of the macula and retinal nerve fibre layer was unremarkable bilaterally, without macular oedema, subretinal fluid, or optic nerve swelling. B-scan ultrasonography demonstrated small superior choroidal effusions in both eyes, which were not clinically apparent on dilated fundus examination (Figure *1*). The findings raised suspicion of daratumumab-associated ciliochoroidal effusion with acute myopic refractive change.

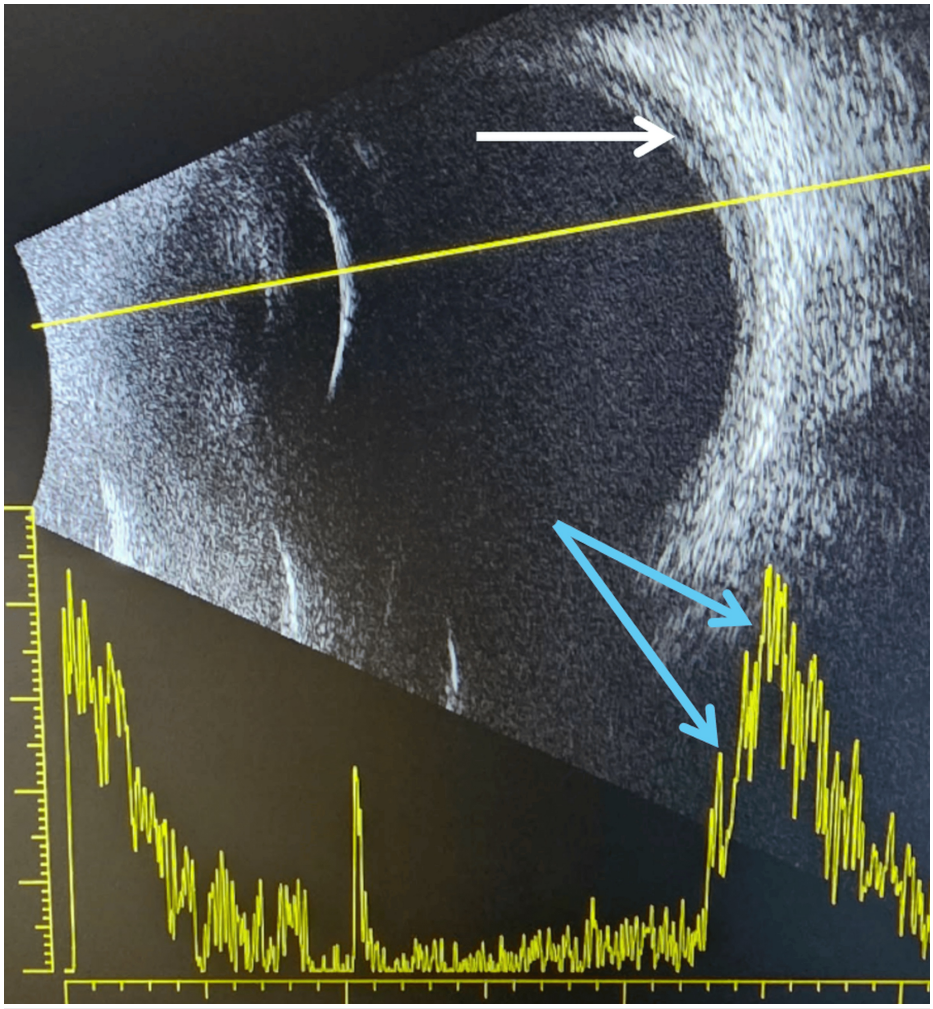


FIGURE 1: Representative initial B-scan ultrasonography of the right eye showing a superior choroidal effusion

B-scan ultrasonography of the right eye at presentation with A-scan overlay, demonstrating a small superior choroidal effusion. The white arrow identifies the region of choroidal separation on B-scan. The blue arrows highlight two high-reflectivity peaks on the corresponding A-scan trace, consistent with echoes from the choroidal and scleral interfaces separated by suprachoroidal fluid. B-scan ultrasonography of the left eye also demonstrated a small superior choroidal effusion.

Alternative explanations included medication-related visual disturbance from promethazine, inflammatory or infectious choroidal disease, posterior scleritis, uveal effusion syndrome, hypotony, and systemic causes of choroidal effusion. Promethazine could have contributed to blurred vision but would not readily explain the bilateral effusions demonstrated on ultrasonography. Posterior scleritis and inflammatory or infectious ocular disease were considered less likely given the absence of significant pain, anterior chamber inflammation, vitritis, or associated retinal abnormalities. Hypotony-related effusion was less likely given intraocular pressures of 10 mmHg bilaterally. The anterior chambers were clinically deep, with no elevated intraocular pressure or clinical features of acute angle closure. However, gonioscopy and quantitative anterior-segment imaging were not documented, and the available examinations did not definitively exclude all alternative causes.

Given the small effusions, normal intraocular pressures, and absence of significant ocular inflammation, conservative management with observation was selected following discussion with the supervising ophthalmology consultant. No specific ocular pharmacological treatment was commenced. The patient was advised to seek urgent reassessment if his visual symptoms worsened, and ophthalmic follow-up was arranged for two weeks. Following discussion between the ophthalmology and haematology teams, daratumumab was continued in view of the patient's limited systemic treatment options.

At the two-week ophthalmic review, the patient reported that his vision had returned to normal. Unaided visual acuity had improved to 6/4.5 + 1 in the right eye and 6/4.5 − 2 in the left eye, with intraocular pressures of 11 and 12 mmHg, respectively. Both anterior chambers remained deep and quiet, visual fields

and OCT were unremarkable, and repeat B-scan ultrasonography confirmed complete resolution of the bilateral choroidal effusions (Figure 2). Daratumumab treatment was ongoing at this assessment. Repeat refraction was not documented.

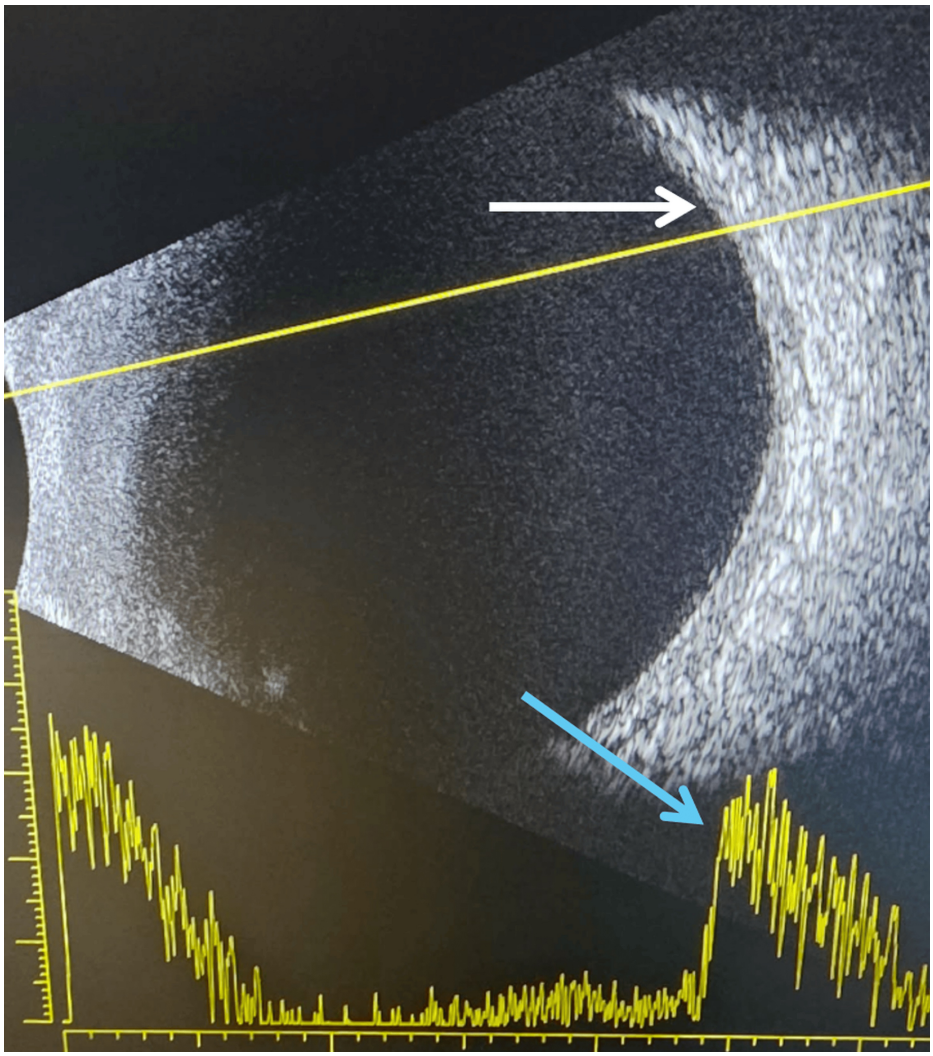


FIGURE 2: Resolution of the right-eye choroidal effusion on B-scan ultrasonography at two-week follow-up

Repeat B-scan ultrasonography of the right eye with A-scan overlay, demonstrating resolution of the previously identified superior choroidal effusion. The white arrow indicates the posterior choroid-sclera complex, with no residual separation evident. The blue arrow identifies the corresponding posterior A-scan echo complex.

At the final ophthalmic review approximately nine weeks after presentation, the patient reported no recurrent visual symptoms. Unaided visual acuity was 6/4.5 in the right eye and 6/6 + 1 in the left eye, with intraocular pressures of 12 and 10 mmHg, respectively. Autorefraction demonstrated plano/−0.25 D in the right eye and +0.50/−0.50 D in the left eye. Anterior segment examination remained unremarkable, OCT demonstrated no macular pathology, and repeat B-scan ultrasonography showed no recurrence of the choroidal effusions. Daratumumab had been discontinued approximately three weeks earlier for haematological reasons unrelated to the ocular findings. The patient was discharged from routine ophthalmic follow-up with advice to return if new ocular symptoms developed. The clinical course is summarised in Table 1. Written informed consent for publication of this case report and accompanying clinical images was obtained from the patient.

Time	Treatment or ophthalmic findings
Day 0	First IV daratumumab infusion, 16 mg/kg. Hypersensitivity reaction requiring temporary interruption and rescue medication. Reported onset of blurred vision.
Day 1	Initial ophthalmology review. 6/9 OD, 6/15 OS (unaided); 6/9 OD, 6/6 OS (trial frame). Myopic autorefraction, IOP 10 mmHg OU, and small bilateral superior choroidal effusions on B-scan.
Week 2	Vision recovered to approximately 6/4.5 OU (unaided). B-scan confirmed resolution of bilateral effusions during ongoing daratumumab therapy.
Week 9	No recurrent symptoms or effusions. VA 6/4.5 OD and 6/6+1 OS. Near-emmetropic autorefraction. Daratumumab had been discontinued for haematological reasons.

TABLE 1: Clinical course following daratumumab administration

OD: right eye; OS: left eye; OU: both eyes; IOP: intraocular pressure; IV: intravenous; VA: visual acuity

Discussion

Daratumumab is a CD38-directed monoclonal antibody with a generally favourable safety profile. Although infusion-related reactions are well recognised, ocular adverse events remain uncommon. Published case reports and postmarketing surveillance have identified acute myopic shift, ciliochoroidal effusion, and secondary angle-closure glaucoma occurring in temporal association with daratumumab administration [3,4]. These presentations vary considerably in severity, ranging from transient visual disturbance to marked intraocular pressure elevation and potentially sight-threatening angle closure.

The precise mechanism of daratumumab-associated ciliochoroidal effusion remains uncertain. CD38 is expressed in the non-pigmented epithelium of the ciliary body, providing a possible basis for a local ocular effect of CD38-directed treatment [4]. Altered vascular permeability or an inflammatory response associated with drug administration may also contribute, although these mechanisms remain hypothetical. Accumulation of fluid within the suprachoroidal space may cause anterior rotation of the ciliary body and forward displacement of the lens-iris diaphragm, producing a transient myopic shift. In more severe cases, this anatomical displacement can narrow the iridocorneal angle and cause secondary non-pupillary block angle closure [5].

Previous reports illustrate the spectrum of daratumumab-associated ocular complications. Singh et al. described a patient with multiple myeloma who developed choroidal effusion, with subsequent resolution following discontinuation of daratumumab [3]. Saengsirinavin et al. reported bilateral ciliochoroidal effusions with acute angle closure and intraocular pressures reaching 50 mmHg following daratumumab infusion. Their patient required cycloplegic and intraocular pressure-lowering treatment, although subsequent daratumumab infusions were administered with prophylactic cycloplegia without recurrence [6]. More recently, Yuksel Elgin et al. described a patient with relapsed T-ALL following bone marrow transplantation who developed bilateral acute angle closure with markedly elevated intraocular pressures after daratumumab administration. The patient was treated with topical medication, and further infusions were administered with prophylactic atropine [7]. These reports demonstrate that continued daratumumab treatment has occasionally been possible following ocular complications, but only under individualised management strategies.

In contrast, our patient presented with small bilateral superior choroidal effusions, clinically deep anterior chambers, and normal intraocular pressures. There was no clinical evidence of acute angle closure, and no specific ocular pharmacological treatment was required. At the two-week ophthalmic review, visual acuity had improved to approximately 6/4.5 bilaterally, and B-scan ultrasonography demonstrated complete resolution of the effusions whilst daratumumab treatment was ongoing. Repeat ophthalmic assessment approximately nine weeks after presentation demonstrated sustained visual recovery without recurrent effusions. Daratumumab had been discontinued approximately three weeks before the final review for haematological reasons unrelated to the ocular findings. This course illustrates a comparatively mild presentation within the recognised spectrum of daratumumab-associated ocular complications.

The close temporal relationship between the first infusion and symptom onset, together with previously reported similar adverse events and objective ultrasonographic findings, raises suspicion of daratumumab-associated ciliochoroidal effusion. However, temporal association alone does not establish causality. Promethazine exposure was a potential confounding factor, although an anticholinergic effect would not readily account for the bilateral choroidal effusions demonstrated on B-scan ultrasonography. Posterior scleritis and other inflammatory or infectious ocular conditions were considered less likely given the absence of significant ocular pain, anterior chamber inflammation, vitritis, or associated posterior segment

abnormalities. Normal intraocular pressure argued against hypotony-related effusion. Nevertheless, other causes, including primary uveal effusion syndrome and graft-versus-host disease, could not be definitively excluded. Transient mild hypoalbuminaemia and hypoproteinaemia were also present around the time of symptom onset and may have contributed to altered choroidal fluid dynamics, although their contribution to the effusions remains uncertain.

A retrospective assessment using the World Health Organisation-Uppsala Monitoring Centre (WHO-UMC) criteria classified the association between daratumumab and the ocular findings as possible [8]. This reflects the reasonable temporal relationship and objective clinical findings, whilst recognising the patient's complex medical history, potential confounding factors, and absence of a positive response to drug withdrawal. Furthermore, resolution during continued daratumumab exposure, without documented recurrence following subsequent administration, limits the certainty of a direct causal relationship.

The management of suspected daratumumab-associated ocular toxicity requires particular caution. Current prescribing guidance recommends interruption of daratumumab when ocular symptoms occur and immediate ophthalmic evaluation before treatment is restarted [9]. Although the effusions in this patient resolved despite continued treatment, this observation should not be interpreted as evidence that daratumumab can routinely be continued without risk. Decisions regarding further administration should involve individualised multidisciplinary assessment, taking into account the severity of the ocular findings, intraocular pressure, anterior chamber anatomy, and the patient's systemic treatment requirements. Patients should also receive clear advice regarding symptoms requiring urgent reassessment, particularly ocular pain, worsening vision, headache, or halos.

The principal limitations of this report include the absence of documented pretreatment refraction, cycloplegic refraction, gonioscopy, quantitative anterior chamber measurements, and comprehensive investigation of all potential systemic contributors. The visual disturbance was not fully correctable with refraction in the right eye at presentation, and no definitive explanation for this finding was established. Although the subsequent recovery supports a transient process, the findings cannot establish the precise mechanism or prove drug causality. Nevertheless, the documented bilateral choroidal effusions, objective resolution during ongoing treatment, and sustained visual recovery contribute to the limited literature describing the milder end of this clinical spectrum.

Conclusions

This case describes acute bilateral choroidal effusions and transient myopic refractive change occurring shortly after daratumumab administration, with complete anatomical resolution and visual recovery during continued treatment. Although the temporal relationship raises suspicion of a drug-associated adverse event, a definitive causal relationship cannot be established from a single case.

Prompt ophthalmic assessment is essential when patients receiving daratumumab develop acute visual symptoms, particularly to identify potentially sight-threatening complications such as secondary angle closure. Although this patient recovered without specific ocular treatment, the favourable outcome should not be interpreted as evidence that continued daratumumab administration is routinely safe. Any decision to continue or recommence treatment following suspected ocular toxicity requires individualised multidisciplinary risk-benefit assessment, careful ophthalmic monitoring, and appropriate patient counselling. Further evidence is needed to establish the mechanisms and optimal management of these uncommon ocular complications.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Angus van der Nagel, Sheetal Shirke

Acquisition, analysis, or interpretation of data: Angus van der Nagel, Sheetal Shirke

Drafting of the manuscript: Angus van der Nagel

Critical review of the manuscript for important intellectual content: Sheetal Shirke

Supervision: Sheetal Shirke

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all

authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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