

Retinal meteor

*Ramesh Venkatesh, Prachi Gurav,
Prachi Abhishek Dave, Sankhadeep Roy*

We describe a case of a 65-year old man diagnosed with retinal vasoproliferative tumour secondary to posterior uveitis. The fluorescein angiography shows an interesting meteor-like leak emanating from the tumour and rising towards the superior retina in the later frames of the angiogram. Pictorially, we call it the "Retinal Meteor" and also describe the possible mechanism for this pattern of leakage.

Key words: Choroiditis, fluorescein angiography, leakage, vasoproliferative tumor

Vasoproliferative tumors (VPTs) are characterized by the presence of one or more nodular lesions on the retina,

Access this article online	
Quick Response Code:	Website: www.ijo.in
	DOI: 10.4103/ijo.IJO_68_17

Department of Retina and Vitreous, Dr. Shroff's Charity Eye Hospital, New Delhi, India

Correspondence to: Dr. Ramesh Venkatesh, Dr. Shroff's Charity Eye Hospital, 5027, Kedarnath Road, Daryaganj, New Delhi - 110 002, India. E-mail: vramesh80@yahoo.com

Manuscript received: 26.02.17; Revision accepted: 20.07.17

commonly seen in the inferotemporal quadrant. They can be primary VPTs or secondary to other ocular pathology.^[1] Hard exudates, subretinal fluid, cystoid macular edema, subretinal or vitreous hemorrhage, and epiretinal or subretinal membrane are the common presenting features in cases of VPT. We describe the case of a VPT secondary to healed choroiditis and vasculitis showing a characteristic meteor-like leakage from the VPT on fluorescein angiography (FA) which we label as the "retinal meteor" and also explain the possible mechanism for this pattern of leakage.

Case Report

A 65-year-old male, a known case of old healed multifocal choroiditis and peripheral vasculitis in both eyes, presented to the uvea clinic for routine follow-up examination. His best-corrected visual acuity was 20/20 in both eyes. Anterior segment was quiet in both eyes. Dilated fundus examination showed patches of healed multifocal choroiditis in both eyes and sclerosed vessels in the left eye periphery. A bright red elevated nodular lesion protruding above the retinal surface was noted along the inferotemporal arcade. Fundus FA was done which showed a feeding artery filling up the entire lesion and a draining vein in the early phase of the angiogram with an interesting meteor-like leak emanating from the lesion and rising toward the superior retina in the late frames of the angiogram [Figs. 1 and 2]. Peripheral retina

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

Cite this article as: Venkatesh R, Gurav P, Dave PA, Roy S. Retinal meteor. Indian J Ophthalmol 2017;65:879-81.

showed collaterals with areas of capillary nonperfusion (CNP) anterior to it. No CNP areas were identified close to the lesion. Optical coherence tomogram (OCT) using the RTVue-100 was done through the macula and the lesion. A shallow pocket of subretinal fluid was noted at the macula. OCT scan through the pathology showed the lesion to be elevated from the retinal surface and protruding toward the vitreous cavity [Fig. 3a and b]. The clinical and retinal imaging findings were suggestive of an active VPT secondary to uveitis. Laser photocoagulation was done around the VPT. OCT imaging was repeated after 3 months [Fig. 3c]. Subretinal fluid at the macula has resolved. Our photo highlights this unusual FA finding in a patient with VPT secondary to multifocal choroiditis and vasculitis and pictorially, we call it the “retinal meteor.”

Discussion

Retinal VPTs are usually identified as one or more retinal nodules in the retinal periphery commonly seen in the inferotemporal quadrant. The inferior retina is affected in 60%–90% of cases and the temporal retina in 42%–75% of cases.^[1] These lesions can be preequatorial in location. VPTs can present with hard exudates originating in the tumor, subretinal fluid, subretinal or even vitreous hemorrhage, vitreous cells, cystoid macular edema, epiretinal membrane, subretinal membrane, and hypertrophy of retinal pigmented epithelium (RPE).^[1-4] Depending on the etiology, retinal VPTs can be classified as primary and secondary. Causes for the secondary VPTs include ocular diseases such as retinitis pigmentosa, sickle cell retinopathy, Coat’s disease, retinopathy of prematurity, toxoplasmosis, toxocariasis, tuberculosis, other uveitis, ocular trauma, retinochoroidal coloboma, and retinal detachment.^[1-5] VPTs exhibit feeder vessels, with less dilation and tortuosity compared to those seen in retinal capillary hemangioma – one of the closest

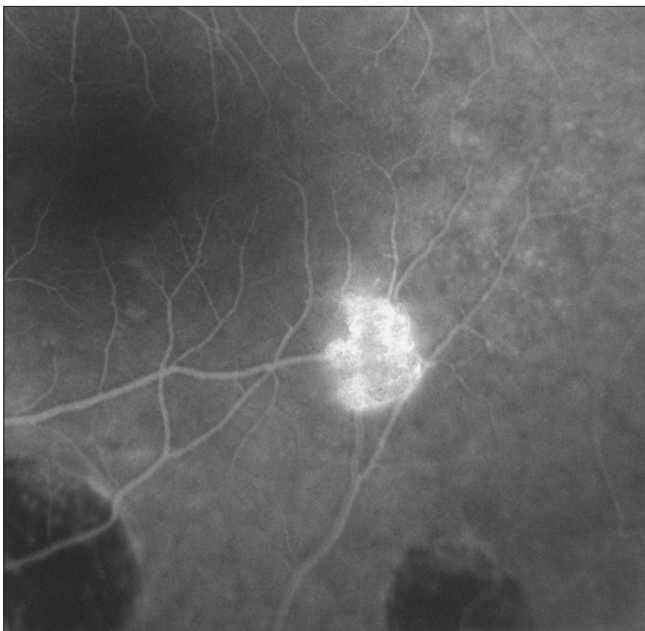


Figure 1: Magnified view of the vasoproliferative tumor with a feeding and draining vessel on fluorescein angiogram

differential diagnosis to VPT. Complementary tests include echography, FA, and OCT. FA is usually of limited value due to the preferred peripheral location of VPTs. When feasible, VPTs on FA reveal early filling in the arterial phase with increasing hyperfluorescence and late leakage.^[1,4,6] Another finding mimicking this pattern of leakage on FA is the presence of retinal neovascularization (NV). Retinal NV is seen as fine loops or networks of vessels lying on the surface of the retina and extending into the vitreous cavity. Unique features of NV include the formation of wheel-like networks, extension across both arterial and venous branches of the underlying retinal vascular network, and accompanying fibrous proliferations. On FA, NV demonstrates profuse leakiness which is usually located at the junction of perfused and nonperfused retina. None of these features were present in our case.

OCT examination through the lesion showed a nodular mass lying over the retinal surface and causing total backshadowing. On OCT, retinal NV is usually seen as flat, marginally elevated, hyperreflective loops of relatively uniform homogenous hyperreflectivity.^[7] OCT in VPT is frequently used to document and follow secondary retinal findings such as edema, fluid accumulation, and membrane formation.^[6] Our case highlights the unusual meteor-like leak from the VPT stretching along the superior retina, which has yet not been reported in literature. Our hypothesis for this pattern of leakage developing in a patient with VPT with uveitis is similar to the development of the smoke-stack pattern of leak in central serous chorioretinopathy.^[8] A combination of factors such as molecular weight of the fluorescein, protein content and oncotic pressure gradient, convection currents, and presence of unbound fluorescein may be responsible for developing this pattern of leakage. Our patient had healed choroiditis lesions with peripheral vasculitis. We presume the proteinaceous material to have leaked from the choroidal vessels in posterior uveitis due to the breakdown of the outer blood–retinal barrier similar to the mechanism of aqueous flare in anterior uveitis. This increased protein content creates an oncotic pressure gradient. Furthermore, the thermoregulatory mechanism of the choroid gets affected in choroiditis, causing a focal rise in temperature. This temperature difference between the cooler vitreous and warmer choroid could stimulate the development of convection currents. In FA, about 20% of the fluorescein is unbound, whereas in indocyanine green angiography, only 2% of the dye remains unbound. This explains the reason for the increased leakage in FA. Thus, the high protein content, increased unbound and low molecular weight of fluorescein, and the development of convection currents could have precipitated this meteor-type leakage pattern on FA.

Various treatment modalities are available including cryotherapy, laser photocoagulation, photodynamic therapy, brachytherapy, surgical resection, intravitreal injections, and immunomodulators. All of them can be used alone or in combination. In the largest study on VPT published by Shields *et al.*,^[1] the presence of subretinal fluid, macular edema, epiretinal membrane close to the macula, or exudates close to the macula was used as the criteria to treat or observe patients with VPTs.

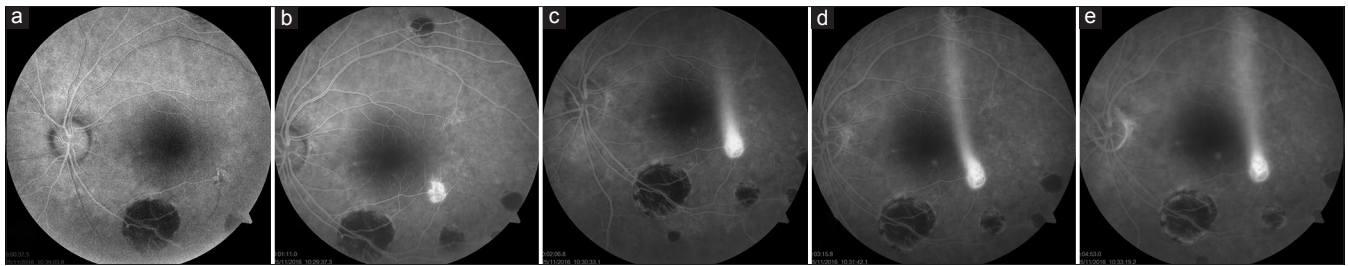


Figure 2: (a-e) Meteor-like leakage from the vasoproliferative tumor along the different phases of fluorescein angiogram

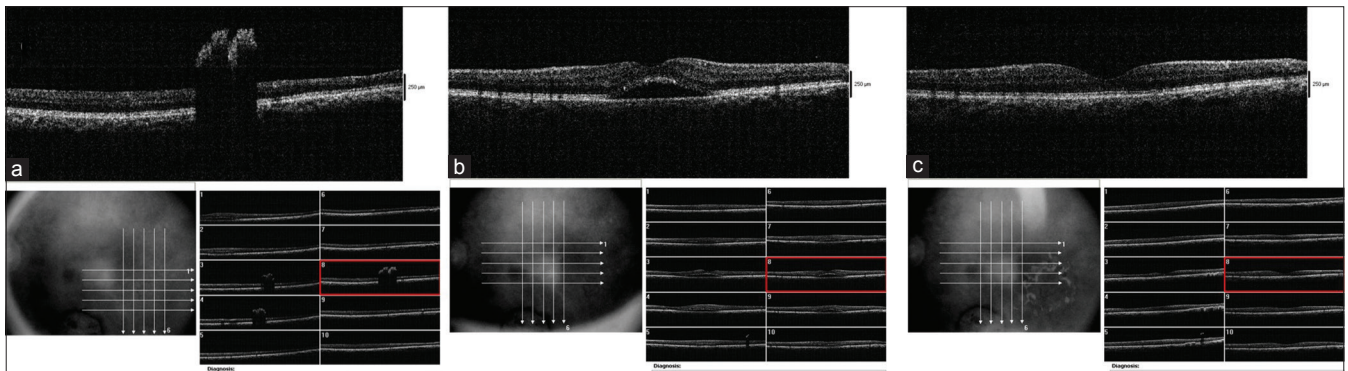


Figure 3: (a and b) Optical coherence tomogram through the lesion and macula at the time of presentation, (c) optical coherence tomogram through the macula at 3 months postpresentation

Conclusion

"Retinal meteor" sign on FFA may be a unique diagnostic sign of VPT.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

- Shields CL, Shields JA, Barrett J, De Potter P. Vasoproliferative tumors of the ocular fundus. Classification and clinical manifestations in 103 patients. *Arch Ophthalmol* 1995;113:615-23.
- Heimann H, Bornfeld N, Vij O, Coupland SE, Bechrakis NE, Kellner U, *et al.* Vasoproliferative tumours of the retina. *Br J Ophthalmol* 2000;84:1162-9.
- Makdoui K, Crafoord S. Vasoproliferative retinal tumours in a Swedish population. *Acta Ophthalmol* 2011;89:91-4.
- Rennie IG. Retinal vasoproliferative tumours. *Eye (Lond)* 2010;24:468-71.
- Shields JA, Shields CL, Honavar SG, Demirci H. Clinical variations and complications of coats disease in 150 cases: The 2000 Sanford Gifford memorial lecture. *Am J Ophthalmol* 2001;131:561-71.
- Maia Júnior OO, Morita C, Angotti Neto H, Bonanomi MT, Takahashi WY. Idiopathic vasoproliferative tumour of the retina associated with macular edema: Case report. *Arq Bras Oftalmol* 2005;68:845-9.
- Cho H, Alwassia AA, Regiatieri CV, Zhang JY, Bauml C, Waheed N, *et al.* Retinal neovascularization secondary to proliferative diabetic retinopathy characterized by spectral domain optical coherence tomography. *Retina* 2013;33:542-7.
- Bujarborua D, Nagpal PN, Deka M. Smokestack leak in central serous chorioretinopathy. *Graefes Arch Clin Exp Ophthalmol* 2010;248:339-51.