



Figure 1. Darkening of a 6-month-old facial skin graft in a 68-year-old woman together with subtle periocular hyperpigmentation and eyelid-margin hyperemia. She had been using topical latanoprost in both eyes for the past 3 years.



Figure 2. Significant lightening of the skin graft together with resolution of the periocular hyperpigmentation and eyelid-margin hyperemia 1 month after stopping topical latanoprost treatment.

ening of the skin graft would suggest it was more susceptible to the effects of prostaglandins than the surrounding facial skin. We propose 2 hypothetical mechanisms for this. First, postinflammatory changes within the skin graft tissue could predispose it to hyperpigmentation as previously reported.⁴ Second, the graft tissue that came from the neck could be more susceptible than the surrounding facial skin to up-regulation of the PGF_{2α} receptor by UV light. We conclude that at-risk patients should be warned of the possibility of severe darkening of a facial skin graft from topical latanoprost and that the use of alternative topical ocular antihypertensive medications in these patients would be sensible.

Daniel Calladine, BMBS, BMedSci
Rosalind J. Harrison, FRCOphth

Correspondence: Dr Calladine, Department of Ophthalmology, Prince Charles Eye Unit, King Edward VII Hospital, Windsor SL4 3DP, England (drdancalladine@doctors.org.uk).

Financial Disclosure: None reported.

1. Wand M, Rich R, Isbey EK, Zimmerman TJ. Latanoprost and periocular skin changes. *Arch Ophthalmol.* 2001;119(4):614-615.
2. Scott G, Jacobs S, Leopardi S, et al. Effects of PGF_{2α} on human melanocytes and regulation of the FP receptor by ultraviolet light. *Exp Cell Res.* 2005;304(2):407-416.
3. Scott G, Leopardi S, Printup S, Malhi N, Seiberg M, Lapointe R. Proteinase-activated receptor-2 stimulates prostaglandin production in keratinocytes: analysis of prostaglandin receptors on

human melanocytes and effects of PGE₂ and PGF_{2α} on melanocyte dendricity. *J Invest Dermatol.* 2004;122(5):1214-1224.

4. Tomita Y, Maeda K, Tagami H. Melanocyte-stimulation properties of aracidonic acid metabolites: possible role in post inflammatory pigmentation. *Pigment Cell Res.* 1992;5(5, pt 2): 357-361.

Pseudoduplication of Fovea in a Human Eye

Duplication of the optic disc and fovea is known to occur in lower vertebrates.^{1,2} Although additional optic discs usually serve no purpose, bifoveate birds (eg, swallows) use the additional fovea for limited conjugate binocular movements.² Doubling of the optic disc, true diastasis as well as coloboma, has been described in the human eye.¹ We report the unusual clinical finding of 2 foveae in 1 eye of a patient.

Report of a Case. A 25-year-old man had experienced diminished vision in the right eye for several months. There was no history of ocular injury or inflammation. His perinatal and family histories were unremarkable. Best-corrected visual acuity was 20/80, N12 OD and 20/20, N6 OS, with low myopic correction. The anterior segment was unremarkable in each eye. Fundus examination of the right eye revealed macular pucker and peripheral telangiectasia with exudation, suggestive of Coats disease. Fundus examination of the left eye revealed 2 parafoveal halos with 2 foveal reflexes; the temporal reflex was

slightly distorted (**Figure, A**). No other ocular structure exhibited duplication. Optical coherence tomography using a Stratus OCT 3 (Carl Zeiss Meditec, Dublin, California) supported the clinical appearance of foveal duplication: the temporal fovea demonstrated a similar slope of clivus and the recession of inner retinal layers as the central fovea. However, the recession was not complete; a thin layer of inner retinal neurons remained above the photoreceptor layer, the probable cause of blunted foveal reflex. Despite additional layers, the central fovea (thickness, 165 μm) and the accessory fovea (thickness, 160 μm) were similar in thickness, probably owing to less-elongated photoreceptors in the latter (**Figure, B**). Fundus fluorescein angiography demonstrated only 1 foveal avascular zone, corresponding to the central fovea. A nonspecific mottled hypofluorescence was observed in the area of the temporal fovea (**Figure, C**). Fundus fluorescein angiography of the right eye revealed leaking telangiectasia and avascular areas in the inferotemporal periphery. Orthoptic evaluation revealed no fixation abnormalities in the left eye; the patient fixed consistently with the central fovea. Automated perimetry using the central 10-2 threshold test protocol (model 720i, Humphrey Field Analyzer II; Carl Zeiss Meditec) showed a normal foveal threshold (31 dB) at the fixation point, with a typical gradient of reducing sensitivity toward the

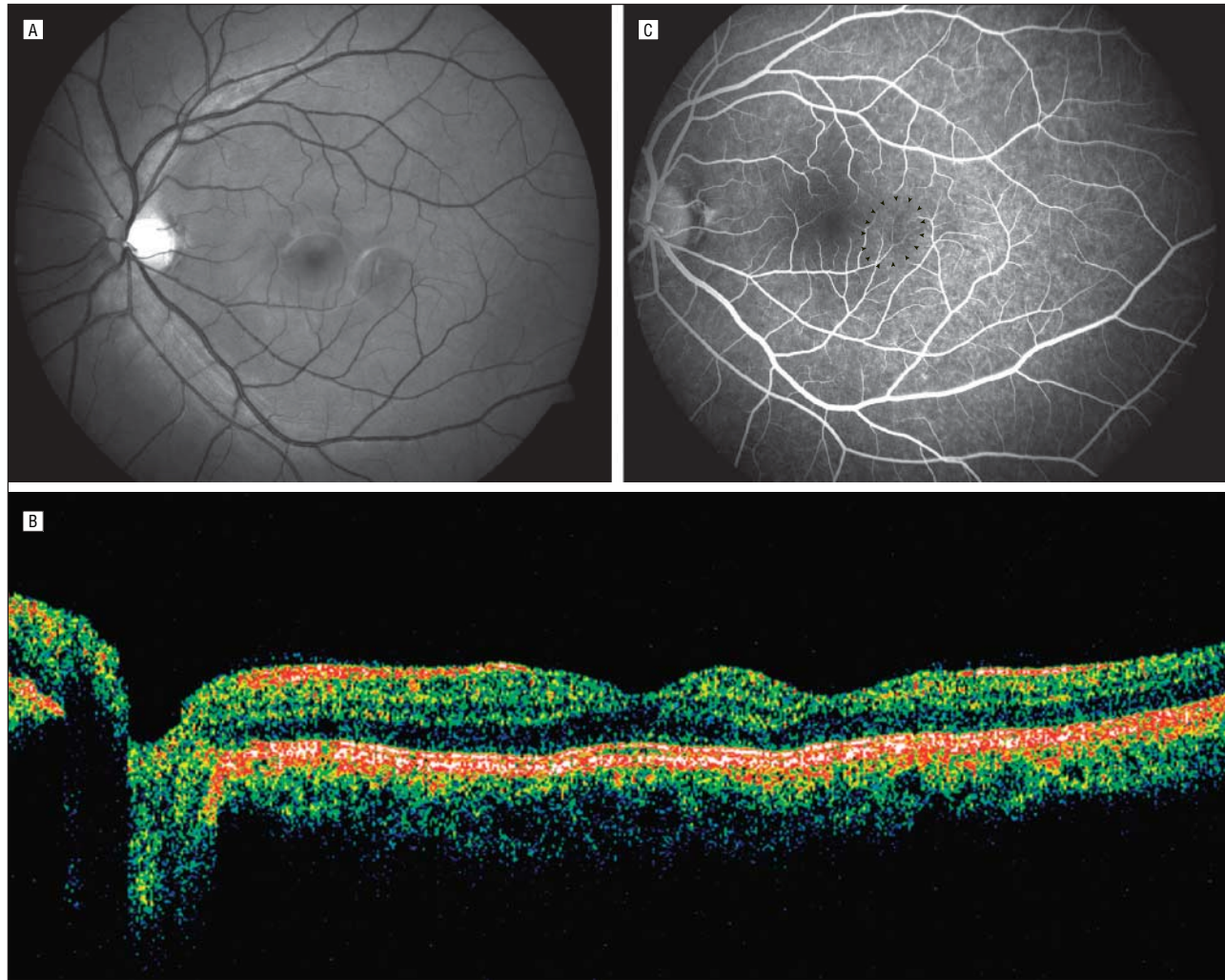


Figure. Clinical, tomographic, and angiographic representation of pseudo-duplication of the fovea in a human eye. A, Fundus view of the left eye shows 2 parafoveal annular reflexes with 2 foveal reflexes; the temporal foveal reflex is distorted into a vertical streak. The satellite fovea is situated approximately 1 optic disc diameter temporal and slightly inferior to the horizontal plane of the central fovea. B, Horizontal (345°) optical coherence tomogram traversing through both the foveal depressions shows the similar topography of the central and satellite foveae. Immaturity of the temporal fovea is evidenced by a residual layer of inner retinal neurons and less-elongated photoreceptors. C, Midphase fundus fluorescein angiogram of the left eye shows a normal foveal avascular zone at the central fovea and nonspecific hypofluorescence in the area corresponding to the temporal fovea (arrowheads).

periphery; no anomalous increase in sensitivity was registered temporal to the fixation point (central fovea). The patient was advised, but declined, to undergo pars plana vitrectomy and endophotocoagulation in the right eye.

Comment. It has recently been suggested that slow development of central retinal vasculature creates hypoxic stress in the inner retinal neurons, which adapt by thinning out into incipient foveal depressions at approximately the seventh month of gestation.³ Active centrifugal migration of neuronal cells further deepens the depression, and stretching of Muller cells condenses and elongates the foveal cones.⁴ The foveal avascular zone

is created 3 to 4 weeks postnatally by the inhibition of centripetal migration of astrocytes, the templates for endothelial proliferation. Foveal maturation is complete by age 3 to 4 years.³ Although we cannot explain this foveal duplication, it is possible that a transient metabolic injury in the perinatal period resulted in an additional zone of hypoxic stress and that consequent focal thinning began to create an immature foveal architecture, aborted before vascular differentiation. Although the extra fovea in this patient was incidentally discovered and nonfunctional, we are unaware of any study documenting its presence in a human eye. The presence of Coats disease in the fellow eye was most likely a chance

occurrence, unrelated to the foveal duplication.

Umesh C. Behera, MS
Dhananjay Shukla, MS
Ramasamy Kim, DNB

Correspondence: Mr Shukla, Aravind Eye Hospital and Postgraduate Institute of Ophthalmology, 1 Anna Nagar, Madurai 625 020, Tamil Nadu, India (daksh66@gmail.com).

Author Contributions: Mr Shukla had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Financial Disclosure: None reported.

1. Brown GC, Tasman WS. Other anomalies of the optic disc. In: *Congenital Anomalies of the Optic*

- Disc. New York, NY: Grune & Stratton; 1983: 266-269.
2. Tychsen L. Binocular vision. In: Hart WM Jr, ed. *Adler's Physiology of the Eye*. 9th ed. St Louis, MO: Mosby-Year Book; 1992:841-842.
 3. Provis JM, Penfold PL, Cornish EE, Sandercoe TM, Madigan MC. Anatomy and development of the macula: specialisation and the vulnerability to macular degeneration. *Clin Exp Optom*. 2005;88(5):269-281.
 4. Hendrickson AE. Primate foveal development: a microcosm of current questions in neurobiology. *Invest Ophthalmol Vis Sci*. 1994;35(8):3129-3133.

Macular Infarction Following Viperine Snake Bite

Macular infarction has been reported following toxic influences, for example, aminoglycoside toxicity.¹ Venomous snake bites may result in neurologic or hemostatic dysfunction. Viperine (hemotoxic) snake bites may produce coagulopathy, which may result in several systemic complications. Ocular involvement is rare. Common ocular problems encountered after a snake bite are generally neurologic (ptosis, ophthalmoplegia, accommodation paralysis, and optic neuritis). Visual loss may result from direct inoculation of venom into the eye (globe necrosis, keratomalacia, and uveitis), from optic neuritis, or secondary to hemostatic abnormality (vitreous hemorrhage, cortical infarction, and central retinal artery occlusion).²⁻⁵

Report of a Case. A 17-year-old girl was bitten by a viperine snake. She was admitted to a local hospital in an unconscious state and administered first aid, anti-snake venom serum,

and supportive care. She regained consciousness 14 hours after the snake bite and 6 hours later reported loss of vision in her left eye. She came to us 5 days later. Visual acuity was recorded as 20/20 OD and no light perception OS. Ophthalmological examination disclosed unremarkable anterior segment and normal intraocular pressures in both eyes. Relative afferent pupillary defect was observed in the left eye. Fundus examination revealed optic disc hyperemia, splinter-shaped hemorrhages at the posterior pole, and a cherry-red spot at the center of the macula (**Figure 1**). Fluorescein angiography demonstrated normal arteretina (10-second) and arteriovenous transit (11-second) times. Blocked choroidal fluorescence was observed in relation to the nerve fiber layer hemorrhages. The most

striking feature on fluorescein angiography was pruning of the perifoveal capillaries. The silhouette of occluded macular capillaries was observed against the choroidal flush. Late-phase angiograms showed optic disc staining (**Figure 2**).

Systemic examination revealed no deficit. Laboratory investigations showed mild anemia (hemoglobin level, 10.4 g/dL [to convert to grams per liter, multiply by 10.0]), leukocytosis (13 800/ μ L [to convert to $\times 10^9$ per liter, multiply by 0.001]), and neutrophilia (74%). Results of renal function tests, abdominal ultrasonography, electrocardiography, echocardiography, and magnetic resonance imaging of the brain were normal. Dual antiplatelet therapy (aspirin, 75 mg/d, and clopidogrel, 75 mg/d), systemic antibiotics, and oral prednisone (40 mg/d tapered by 10

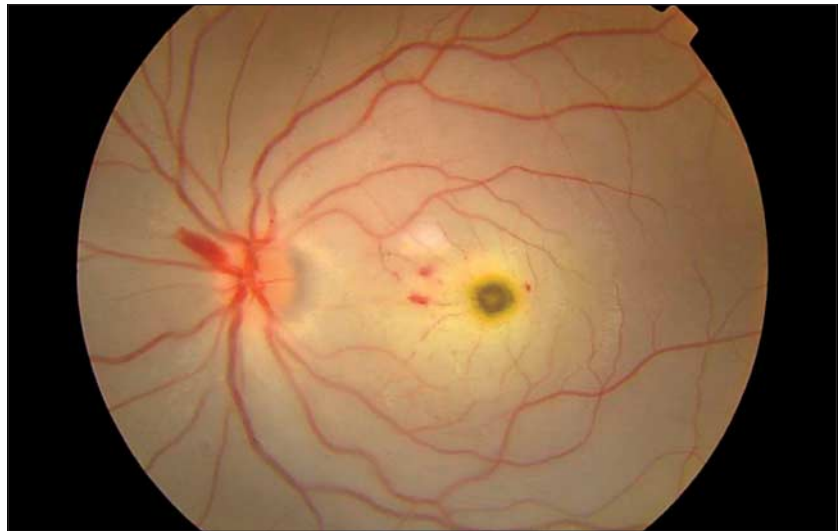


Figure 1. Fundus photograph showing a cherry-red spot at the macula and superficial retinal hemorrhages.

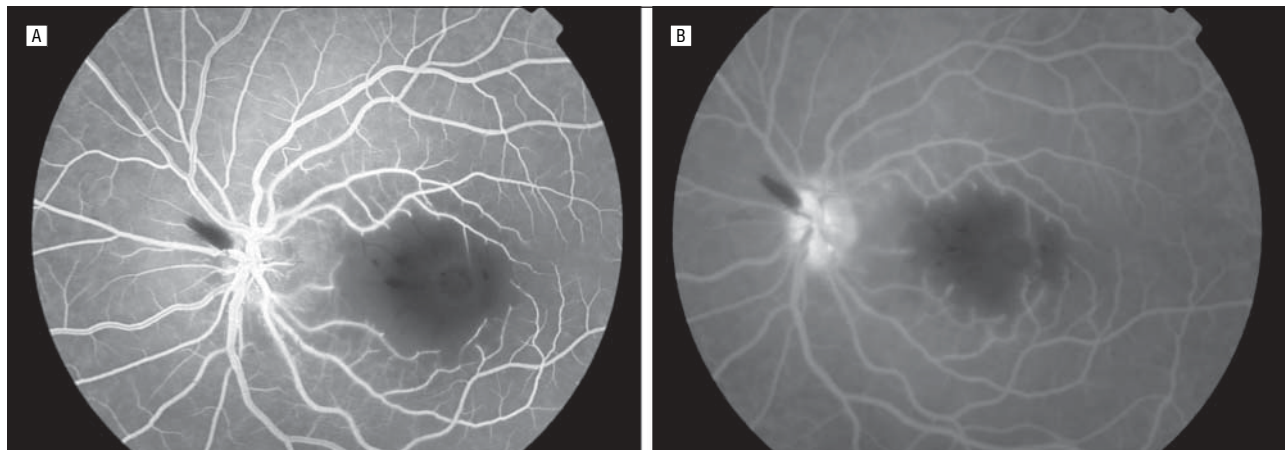


Figure 2. Early (A) and late phase (B) fluorescein angiograms demonstrate pruning of the perifoveal capillaries and optic disc staining, respectively.