

CASE REPORT



Clinical Pearls—Andrew B. Mick, OD, FAAO

Clinicians routinely see patients who present with eye redness. While infectious conjunctivitis and episcleritis are common causes, a detailed history occasionally leads to a rare diagnosis. In this well-documented case report, a patient is suspected of having food-induced ocular vascular injection accentuated by a prior history of necrotizing scleritis with scleral thinning. The author proposes an underlying trigeminal autonomic etiology to explain the unique clinical features.

Food-Induced Red Eye

Susan G. Rodgin*

ABSTRACT

Purpose. This article addresses the etiology of red eyes and a novel case of food-induced red eye.

Case Report. An 82-year-old white male patient reported 50 years of episodes of severe unilateral bulbar injection in either eye following ingestion of certain foods. Thinned scleras in this particular patient made the injection in his eyes more observable. Photodocumentation of this reaction after he ingested these foods is shown.

Conclusions. It is hypothesized that this is a new explanation for an intermittent, unilateral, self-limited red eye—a food-induced red eye.

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Key Words: eyes, food trigger, gustatory flushing, migraine, necrotizing scleritis, ocular migraine, red eyes

Occasionally, patients present with vague complaints of intermittent redness of their eye(s), which is self-limited and, according to the patients, idiopathic. The eye doctor then tries to help them to discern the etiology and treatment, if necessary. A food-induced red eye is a novel clinical observation that is previously unreported.

CASE REPORT

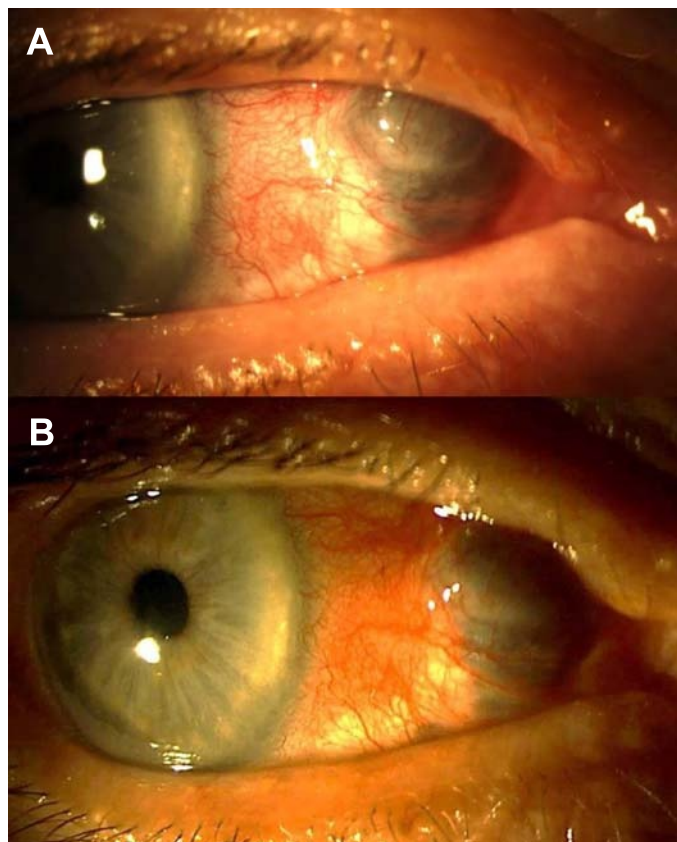
Informed consent according to the Veterans Administration guidelines was obtained from this patient after explanation of the nature and possible consequences of the publication of this case report.

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An 82-year-old retired man presented for his routine eye examination. During the examination, he mentioned incidentally that he had episodes over the previous 50+ years of red eyes (onset in his 20s) that occurred after ingesting peaches, celery, red grapes, red wine, cheeses, and radishes. The redness occurred 24 to 36 hours following ingestion of these foods. Each episode lasted 4 to 6 days and affected 1 eye or the other, but never both simultaneously. There were no associated headaches, itching, blurred vision, asthenopia, or family history of migraines. He reported mentioning these episodes to his eye doctors in the past, but reported that they “just dismissed” this complaint. The patient managed this problem simply by identifying provocative foods and eliminating them from his diet.

Medical history included pacemaker implantation in 1997 for recurrent arrhythmia with syncope, carotid stenosis (left carotid endarterectomy in 1999 and right carotid stent in 2006), hypertension,

**FIGURE 1.**

Nasal right eye: uninflamed (A) inflamed (B).

hyperlipidemia, and gout. He had no history of tobacco or alcohol use. He had no known drug or environmental allergies.

Medications included metoprolol XL 50 mg daily, lisinopril 20 mg daily, atorvastatin 10 mg daily, allopurinol 100 mg daily, a daily multivitamin and antioxidant supplement, lecithin 1200 mg, and aspirin 81 mg daily. Also, he used topical preservative-free ophthalmic lubricant nightly and carboxymethylcellulose 0.5% ophthalmic drops two or three times a day.

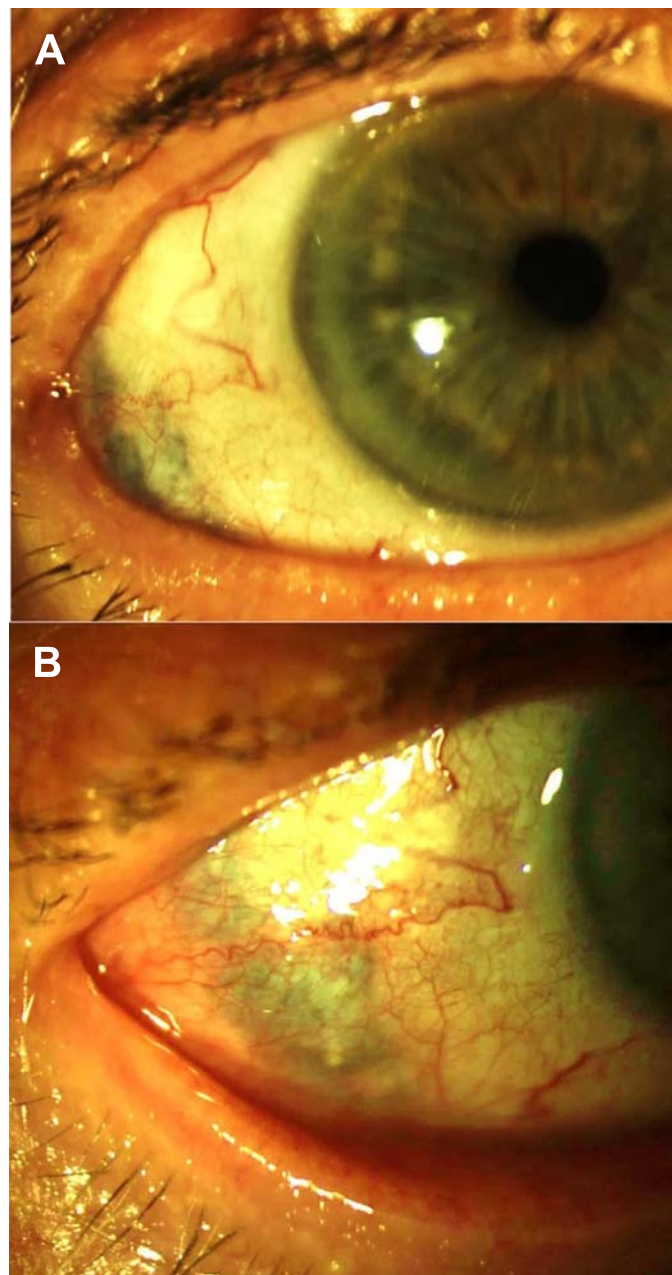
The patient's ocular history was remarkable for uveal ectasias in both eyes secondary to necrotizing scleritis episodes diagnosed 40 years earlier and followed by an ophthalmologic immunologist. The necrotizing scleritis began 10 years *after* his food-induced red eye episodes had occurred. His last necrotizing scleritis flare-up was in 2008. He had a cataract extraction from the left eye in 2006, with secondary chronic macular edema and 1+ epiretinal membrane.

Entering best-corrected acuities were 20/30 in the right eye (+1.50 −4.50 × 022) and 20/100 in the left eye (+2.00 −5.25 × 172). There was a history of bilateral meridional refractive amblyopia in both eyes and chronic cystoid macular edema in the left eye as a complication of cataract surgery.

Anterior segment examination showed moderate bilateral meibomian gland dysfunction, 2+ arcus, 1+ guttata, 1+ endothelial pigment, and mild inferior corneal thinning. The right lens had 3+ nuclear sclerosis, and the left intraocular lens had mild posterior cortical opacification behind it. There was a patent LPI at 1:00, right eye and posterior synechiae at 1:30 to 2:00 in the right eye, related to a previous anterior uveitis episode. Secondary to the cataract surgery, there were endothelial pigment clumps in the left eye. Large areas of

anterior uveal prolapse in both eyes with anterior scleral staphylomas were seen nasally, temporally, and superotemporally in the right eye and superiorly and superotemporally, inferotemporally, nasally, and temporally in the left eye. Intraocular pressures were 13 mmHg in each eye.

Posterior segment examination revealed healthy optic nerves with shallow .45 cupping, distinct borders, pink and healthy rim tissue, and moderate malinsertion. Maculae were remarkable for very mild drusen and retinal pigment mottling in the right eye and 2+ chronic cystoid macular edema and 1+ epiretinal membrane in the left eye following the cataract extraction. There was a posterior vitreous detachment in both eyes. Correlating to the location of the anterior ectasias, there were posterior staphylomas with chorioretinal disruption.

**FIGURE 2.**

Temporal right eye: uninflamed (A) inflamed (B).

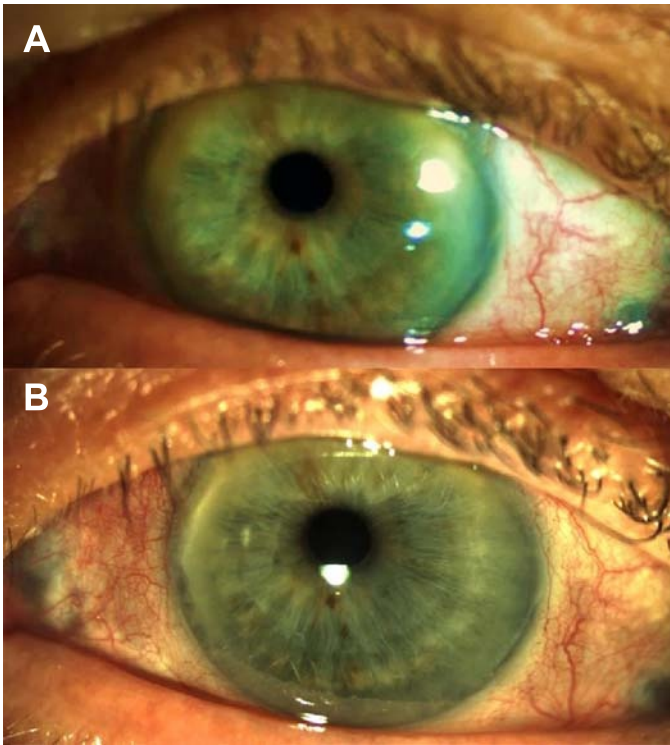


FIGURE 3.

Left eye: uninflamed (A) inflamed (bottom).

The patient consented to imbibe one of the culprit foods—either cheese, grapes, or peaches—on different visits within 24 to 36 hours before an examination so that we could evaluate and photodocument the ocular changes in whichever eye was affected, as shown in Figs. 1–5.

Necrotizing Scleritis

Necrotizing scleritis is a severe form of scleritis. Its immunopathology is based on vascular inflammation (fibrinoid necrosis and neutrophil deposition) in blood vessel walls and inflammatory cells in the sclera and the conjunctiva.^{1,2} Also, immunodeposits (immunoglobulins and complement components) have been identified in 93% of scleral and 79% of conjunctival tissue biopsied in patients with necrotizing scleritis.¹

There is a 50% to 80%² association of necrotizing scleritis with systemic autoimmune disease. Rheumatoid arthritis is the most commonly associated disease. Other associations include granulomatosis with polyangiitis, inflammatory bowel syndrome, relapsing polychondritis, and Reiter syndrome—although the patient in this case report had none of these diseases. Necrotizing scleritis also may occur as a complication of pterygium excision,³ following other ocular surgeries,^{4–6} or postinfection. Also, necrotizing scleritis has been reported as a complication of cosmetic eye-whitening procedures.^{7,8}

The diagnosis of necrotizing scleritis is based on several clinical and histopathologic forms. Idiopathic necrotizing scleritis, which this patient has, manifests with scleral wall infiltration of chronic inflammatory cells and a few small spots of necrosis and episcleral and uveal infiltration with mononuclear cells, but without vasculitis and without granulomatous inflammation, that is, not thought to be

a primary immunologic etiology, but more likely a delayed hypersensitivity reaction to a scleral-soluble antigen.⁹

When active, there are areas of white sclera because of capillary necrosis and closure. The adjacent cornea may become ulcerated, and there may be a uveitis present. In the postinflammatory phase, there is often characteristic barring of the blue choroid through the atrophic scleral area. Two-thirds of patients with necrotizing scleritis are treated with high-dose glucocorticoids and sometimes also with immunosuppressant agents.¹⁰ Necrotizing scleritis is more commonly found in women, at an older age onset (mean age, 66 years), and with a higher risk of complications and association with systemic disease.

Food-Induced Reactions

Known etiologic agents of food-induced reactions include migraines, gustatory flushing, allergic reactions, and red ear syndrome.

Migraines

Migraines affect up to 12% of people in the United States,¹¹ occur in up to 17% of women and 6% of men,^{11,12} run in families, and have associated auras in 25% of cases.

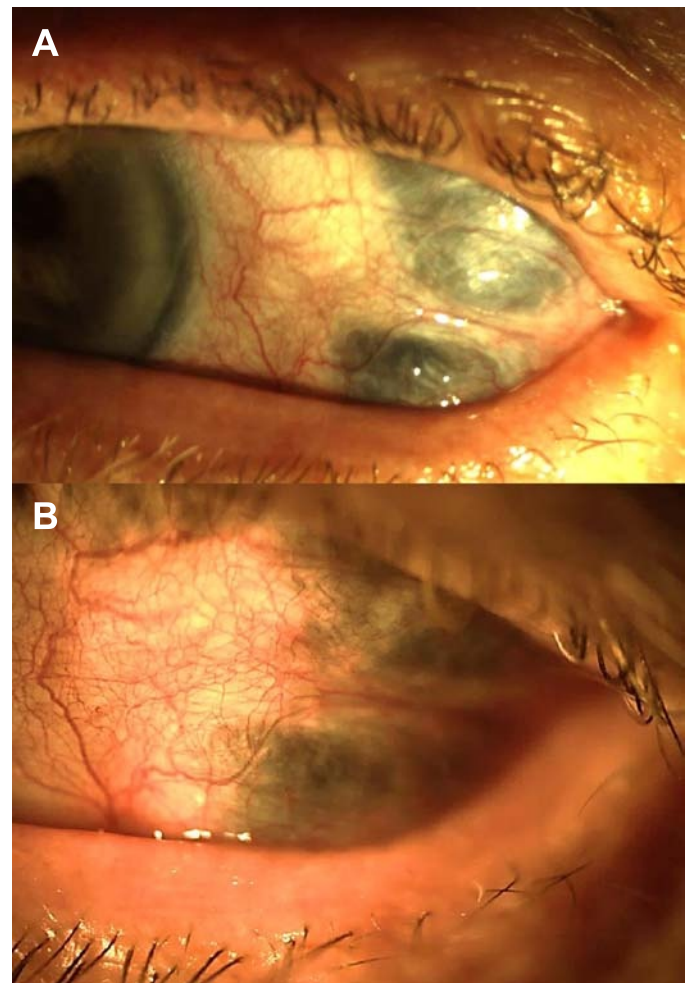


FIGURE 4.

Temporal left eye: uninflamed (A) inflamed (B).

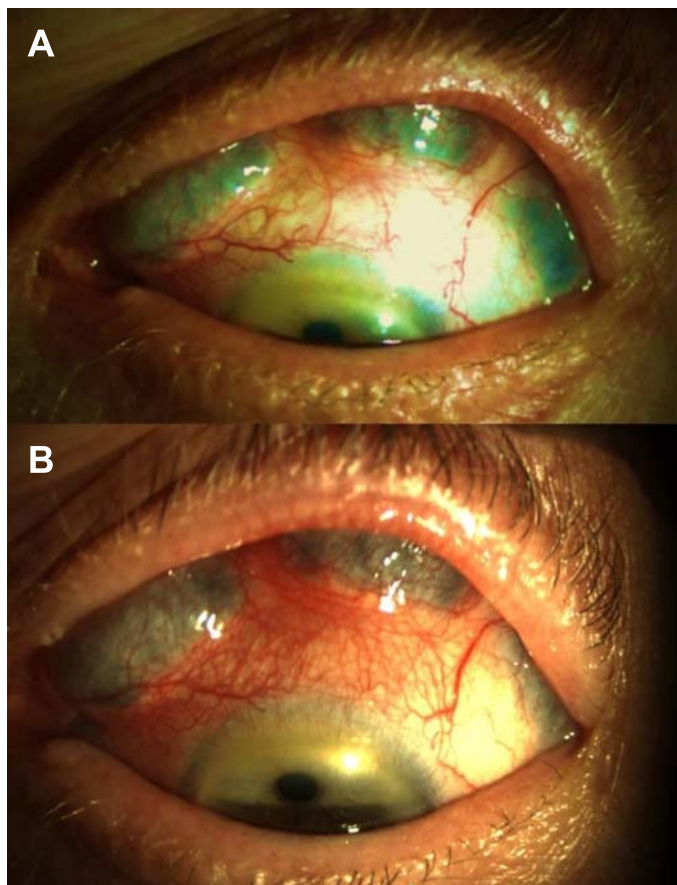


FIGURE 5.
Superior left eye: uninflamed (A) inflamed (B).

Ocular Migraines

The entity of ocular migraines (visual migraine, aura without headache, acephalic migraine, migraine equivalent) has an incidence of between 1 and 6%.^{13–16} They are painless visual obscurations that may be unilateral or bilateral. Shape, color, brightness, and location in the visual field may vary from one episode to the next.¹⁷ Usually, they remit within 30 minutes or less.

Current studies suggest that the etiology of migraines is a primary neuronal dysfunction. There is thought to be a spreading depression across the cerebral cortex.^{18,19} This neuronal change activates trigeminal afferents, which then cause secondary inflammatory changes²⁰ (vasodilatation and plasma protein extravasation), and alters blood-brain barrier permeability, causing the ocular migraine.

In a study of 1750 patients with migraines, a significant number of these factors were found to relate to food: not eating (57%), food odors (44%), alcohol (38%), and specific foods (27%).²¹

There are many studies identifying specific foods/ingredients that are often correlated with migraine headaches, including cheese, chocolate, citrus fruits, aspartame, monosodium glutamate, and red wine.²² Among specific chemicals thought to cause migraines are tyramine, nitrites, sulfites, and phenylethylamine—which readily crosses the blood-brain barrier. Food-induced migraines have an onset of 12 to 48 hours.

Retinal Migraines

In retinal migraines, unilateral visual phenomenon (scotoma, photopsia, aura) without headaches, it is thought that both the retinal and the ciliary circulation systems are involved.

Gustatory Flushing

Gustatory flushing, also known as auriculotemporal syndrome or Frey syndrome, is a food-induced blushing of the cheeks and preauricular and/or temporal areas. There is an accompanying sweating in the same location. It usually occurs unilaterally, except as related to bilateral forceps injury. Suspected pathophysiology includes aberrant regeneration of parasympathetic nerves that innervate sympathetic cutaneous vasculature and sweat glands²³ or an autonomic neural reflex of the trigeminal nerve, similar to ocular migraines.²⁴ Gustatory flushing typically occurs following local trauma (e.g., forceps delivery), injury, or surgery (e.g., parotid), or is mechanically induced from a local mass.²⁵ Onset of flushing is immediate with mastication and remits shortly thereafter.

Food-Induced Allergy

Food-induced allergies are immune-mediated (immunoglobulin E- or cell-mediated) reactions. Ocular manifestations of allergies are usually bilateral. All foods containing the offending chemical agent would provoke a reaction. Ocular allergic reactions usually include additional symptoms besides redness, such as itching, pain, irritation and watering, or nasal congestion. Symptoms of allergies affecting the eyes generally have an immediate onset and last until the offending agent is discontinued.

Red Ear Syndrome

Red ear syndrome is a rare condition of episodic erythema of the ear, accompanied by burning pain, tinnitus, hearing loss, and/or hyperacusis.²⁶ It is usually unilateral. Triggers include stress, touch, hot or cold temperatures, neck movement, sneezing, coughing, chewing, or hair brushing. Or, episodes can also just be spontaneous—especially in younger patients.²⁶ Some may be followed by a migraine headache. There is one reported case of food-induced red ear syndrome in the literature.²⁷ Each red ear syndrome episode was right sided, occurred with severe radiating pain, and lasted 1 hour. The suspected pathophysiology of red ear syndrome (excluding structural abnormalities) is either sympathetic dysregulation or trigeminovascular activation associated with migrainous etiology.^{27,28}

DISCUSSION

Chemicals that might be associated with food triggers in this patient include tyramine (cheese, red wine), nitrites (celery), sulfites (red wine), birch pollen (celery, peaches, cherries), mugwort pollen (celery), salicylates (peaches, cherries, radishes), endochitinase 4A and a lipid-transfer protein (red wine, red grape, peaches),²⁹ and 24-kd protein (red wine, red grapes, cherries).²⁹ However, there are other foods containing these chemicals that do

not induce red eyes in this patient. Also, there is not any one or two of these chemicals that cover all of the patient's sensitivities.

The thinned sclera in this patient with necrotizing scleritis facilitated the likely diagnosis of food-induced red eye. Thinned scleras reduced the underlying surface area of anterior vasculature and enhanced the relative exposure and observability of his conjunctival/episcleral/scleral vasculature.

Based on a review of food-induced reactions discussed in this report, it is the opinion of this author that food-induced red eye is most similar to ocular migraines.

In gustatory flushing, there is usually a predisposing local trauma/injury, there is sweating at the same location, and the episode lasts only as long as the offending food is being imbibed—none of which characterizes food-induced red eye.

It is unlikely that the etiology of this patient's food-induced conjunctival injection is allergic for several reasons. First, the reaction was unilateral, whereas allergies would likely affect both eyes. Second, there are no common chemicals common to the triggering foods. Third, the patient has no other allergic signs or symptoms. Finally, the period between the triggering event and the onset of signs is much longer than that of an allergic reaction.

This case is dissimilar to red ear syndrome because of the absence of associated pain and of other associated sensory symptoms such as hyperacusis or tinnitus.

This case most closely resembles a localized inflammatory reaction secondary to neurologically induced dysfunction as in ocular migraines.

It is well documented in the literature that foods can cause migraine headaches, which can be unilateral and are self-limited—all of which characterize this patient's episodes. Also, similar to food-induced migraine headaches, the time until onset of symptoms is 12 to 36 hours.

Also, it is known that unilateral migraine headaches, which can induce ipsilateral conjunctival injection during the episode (as well as migraine auras), are caused by trigeminal overaction stimulating the trigeminal-autoimmune reflex,^{30,31} which this author hypothesizes to be the pathophysiology of food-induced red eye.

As in retinal migraines, it is likely that the ciliary circulation is affected in this food-induced red eye event, as bulbar vasculature is the anterior extension of the ciliary circulation. The fluctuating laterality in this case may resemble retinal migraines, which provokes fluctuating images.

Food-induced red eye has not been previously described or studied because (1) when scleral tissue remains intact, there may be only minimal anterior extension of visible vascular abnormality during the episode; (2) a patient's eyes may be shut for much of the episode until it passes; (3) the episode duration is likely too short to attend to changes; and/or (4) transient episodes of redness are ignored by patients and clinicians because it always resolves.

CONCLUSIONS

This is a novel case documenting the association of food consumption with isolated episodic ocular conjunctival injection. This case is unusual because the patient's scleral tissue is compromised and thinned, facilitating and enhancing the appearance of the food-induced red eye. It is therefore suggested that when other patients complain about self-limiting intermittent red eyes they should be queried about potential dietary triggers for a food-induced red eye.

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