What’s the Cause of This Mysterious Eyelid Twitch?

Eyelid spasm can signify brainstem disease — or it could be as benign as psychosomatic reaction to stress. Here’s how to tell the difference.

Case
A 25-year-old woman presents with a several-week history of twitching of the right lower eyelid lasting up to a few minutes and occurring 5-6 times per day. During the last several weeks, she has been under increased stress due to law school finals and has had less sleep than usual. She drinks a few cups of coffee a day. Past medical history is negative. Neurological examination was normal except for a single episode of fine contractions of the right lower eyelid lasting about 15 seconds.

What is your diagnosis?

Expert Opinion
This patient’s complaint of new-onset eyelid twitching occurring intermittently but on a daily basis is known as eyelid myokymia. Myokymia is a condition in which involuntary, fine, undulating contractions occur in select striated muscle groups, in this case confined to the eyelid. Electrophysiologically, the contractions are characterized by rhythmic or semi-rhythmic bursts of a single motor unit discharging several times per second. These myokymic discharges are non-synchronous in different motor units within the same muscle, thereby producing the fine, undulating appearance.

Eyelid myokymia tends to be unilateral and localized. If additional lower facial muscle groups are involved, consider facial myokymia or hemifacial spasm. If bilateral eyelid spasms are present, the likely diagnosis is benign essential blepharospasm (BEB). If BEB is present but there are also spasms of the perioral area, tongue or neck muscles, then consider Meige syndrome.

• What is the etiology? Eyelid myokymia is a subset of facial myokymia. Facial myokymia tends to be associated with underlying neurologic disease affecting the facial nerve nucleus in the pons or the peripheral nerve. Intrinsic brainstem disease such as pontine glioma or demyelination from multiple sclerosis may result in facial myokymia, as may extrinsic disease such as obstructive hydrocephalus or an extra-axial neoplasm compressing the brainstem.

The common final pathway in facial myokymia is deafferentation of nerve fibers—be it supranuclear, perinuclear or infranuclear—which in turn leads to disinhibition and hyperexcitability of facial nerve fibers. Ephaptic transmission with “cross-talk” of fibers or ectopic excitation may occur in neighboring branches of the facial nerve, which then manifests as myokymia.

However unlike facial myokymia, when the myokymic symptoms are limited to the eyelid, no underlying neurologic condition tends to be associated. A recent study of 15 patients with chronic isolated eyelid myokymia showed that in long-term follow-up (12-240 months, with a mean of 91 months), 12 patients (80 percent) had no evidence of any type of neurologic disorder. Of the remaining three patients, in only one patient who developed ipsilateral hemifacial spasm did the spasms herald progression of disease, though neuroimaging was normal. A second patient developed MS, and a third patient was diagnosed with Alzheimer’s disease, but in neither of these cases was the eyelid myokymia directly linked. Furthermore in the study, neuroimaging was negative in 13 of 15 patients (87 percent).

Thus, eyelid myokymia tends to be a benign condition, different from facial myokymia. The etiology of the former condition remains unclear. It may be hypothesized that because the process is localized to the eyelid and spares the remaining facial muscle groups, the lesion is quite peripheral; nerve fibers which innervate the orbitociliary oculi may be selectively affected and hyperexcitable. However, no electrophysiologic studies have yet been performed on eyelid myokymia patients in order to better understand the nature of this disorder.

Both ocular surface disorders as well as psychosocial factors should be investigated in cases of new onset, isolated eyelid myokymia. Local eyelid irritation from such entities as dry eye, ocular allergy, or a foreign body may precipitate symptoms. Lifestyle factors such as fatigue, lack of sleep, physical exertion, stress, smoking, alcohol, or excess caffeine intake, may also play a role in the onset of eyelid myokymia.

• Can the upper eyelid ever be involved? Eyelid myokymia is usually unilateral, affecting the lower lid. However, on occasion the upper eyelid may be involved. If so, this should not raise suspicion of any underlying neurologic lesion.

• When is diagnostic testing indicated? When the myokymia remains isolated to the eyelid, even if chronic, no further
work-up such as neuroimaging is warranted. However, if there is spread to neighboring ipsilateral facial muscle groups, an underlying cause for the symptoms should be investigated. As noted previously, facial myokymia tends to be associated with intrinsic or extrinsic disease of the brainstem or peripheral facial nerve; by contrast, eyelid myokymia is not.

- What is the prognosis? Eyelid myokymia, whether transient or chronic, is a benign entity with a good prognosis for spontaneous resolution. When local eyelid irritation or psychosocial factors are at play (as in the case in the patient described above), the myokymia will likely be transient. Symptoms commonly regress within several weeks to months, particularly upon removal of the aggravating factor(s). However, even when the myokymia is persistent, lasting beyond three months and even up to several decades, the symptoms may occasionally spontaneously remit at any time.8

- What treatment would you recommend? In the case of new-onset isolated eyelid myokymia, any exacerbating factors such as eyelid irritation or lifestyle factors must first be identified and addressed. For example, in the case above, the patient should be first advised to try to reduce her stress level, improve on her sleep, as well as decrease caffeine intake.

If these lifestyle modifications are not possible or not effective in eliminating the eyelid myokymia, and symptoms have persisted continuously for greater than three months, then treatment may be initiated. Botulinum toxin in low doses to the eyelid is quite effective, and, in some instances, may even be curative.9,10 Another alternative is surgical myectomy of the affected orbicularis oculi muscle, although even with resection symptoms may recur.11


Rudrani Banik, MD is Assistant Professor in the Departments of Ophthalmology and Neurological Surgery and Director of Neuro-Ophthalmology Service at Albert Einstein College of Medicine/Montefiore Medical Center in Bronx, New York.