

**Figure 2.** Microscopic examination of the honey bee stinger. A, A chitinous stylet with a proximal bulb and sawlike tip. B, Two separate lancets are situated within the stylet. This could be seen only after the stylet had been crushed. C, The foreign body from the patient's upper tarsus resembled a single lancet.

eign body, which resembled a bee stinger (Figure 1D). The patient was treated with combined topical antibiotics and steroids. At 2 weeks' follow-up, he was asymptomatic and the eyelid swelling and corneal epithelial defect had resolved. The stinger of a honeybee normally comprises 2 lancets wrapped together by a single stylet (Figure 2A). Slight pressure was experimentally applied to a fresh stinger by rubbing the stinger between 2 fingers. This caused breakage of the stylet, releasing the 2 lancets (Figure 2B). In comparison with the normal honeybee stinger, the foreign body from our patient resembled a single lancet (Figure 2C).

**Comment.** Complications from bee venom and toxins have been widely discussed in the literature, ranging from anterior segment inflammation to severe vision loss from toxic optic neuritis.<sup>1-5</sup> Mechanical complications from the bee stinger itself, however, have been less mentioned. Little is known about the effects of the chitinous stinger. Some authors suggest that it is inert and can be left in the eye.<sup>6</sup>

The honeybee stinger possesses a sawlike architecture; therefore, once buried in the tissue, an attempt to grasp it and pull it out in the reverse direction usually results in retention of part of the stinger. Moreover, external pressure may cause the stylet to crush, resulting in release of the 2 lancets into the tissue. The structure assumed to be the entire stinger that had been previously removed in our patient was truly only a single lancet.

Physicians should be aware of the possibility of incomplete removal of a bee stinger even with a history of assumed successful removal, particularly when inflammation persists chronically. Careful examination of the site of injury and adjacent tissue for retained parts of the stinger is mandatory prior to concluding that the inflammation is due to bee venom and toxins. Gentle removal of the stinger is suggested to avoid chronic inflammation and mechanical injury to ocular tissue from possible late migration.

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### Percentage of Fellow Eyes That Develop Full-Thickness Macular Hole in Patients With Unilateral Macular Hole

**P**atients with a unilateral macular hole (MH) have an increased risk of developing an MH in the fellow eye.<sup>1-4</sup> However, to our knowledge, the incidence of developing an MH in the fellow eye has not been analyzed in a large cohort of eyes after macular hole surgery. The purpose of this study was to determine the probability of developing a full-thickness MH in the fellow eyes of patients with a unilateral MH.

**Methods.** A retrospective longitudinal study of 1082 patients with a unilateral, idiopathic, full-thickness MH who underwent vitrectomy by one of us (N.O.) between October 1990 and December 2010 was conducted. All of the patients were confirmed to have a unilateral full-thickness MH at the initial visit by dilated indirect slit-lamp biomicroscopy. Patients with any other fundus diseases or history of ocular trauma or surgery in either eye were excluded.

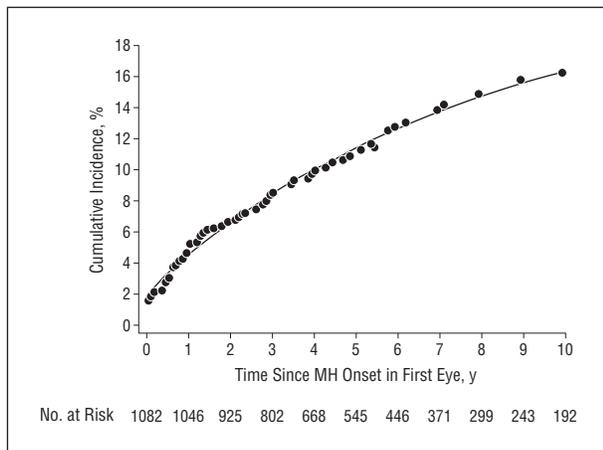
Kaplan-Meier life-table analysis was used to estimate the risk of developing an MH in the fellow eye. In addition, the cumulative incidence of bilateral MHs was fit to a hyperbolic function:  $G = G_{max} \times T / (T_m + T)$ , where the visual gain ( $G$ ) was defined as the preoperative best-corrected visual acuity minus postoperative best-corrected visual acuity in logMAR units; the maximum visual gain ( $G_{max}$ ) was defined as the preoperative best-corrected visual acuity minus final best-corrected visual acuity in logMAR units; the average visual gain was plotted as a function of the postoperative time ( $T$ ) in months; and  $T_m$  was defined as the postoperative time required to reach one-half  $G_{max}$ . This equation was found earlier to describe the recovery of visual acuity after treatment of different macular diseases.<sup>5</sup>

**Results.** There were 394 men and 688 women in the study. The mean (SD) age at the initial surgery was 64.2 (8.3) years (range, 21-95 years). The mean (SD) follow-up period was 71.8 (49.6) months (range, 6-246 months).

**Table. Baseline Characteristics of Study Patients**

Characteristic	Unilateral Group (n=960)	Bilateral Group (n=122)	P Value
Sex, No. (%)			.63
Male	352 (36.7)	42 (34.3)	
Female	608 (63.3)	80 (65.7)	
Age at MH onset in first eye, mean (SD), y	64.4 (8.6)	64.5 (6.1)	.98
Axial length in first eye, mean (SD), mm	23.5 (1.4)	23.3 (1.4)	.42

Abbreviation: MH, macular hole.



**Figure.** The cumulative incidence of macular hole (MH) bilaterality can be described by a hyperbolic function,  $y = 2.6 + 29.8x / (130.1 + x)$ , with  $R^2 = 0.99$ . Curve-fit analysis showed that the estimated risk of MH in the fellow eye was 12.0% at 5 years and 16.9% at 10 years.

Nine hundred sixty patients (88.7%) remained with a unilateral MH (unilateral group) and 122 patients (11.3%) developed an MH in the fellow eye (bilateral group). The sex distribution, age at onset in the first eye, and axial length in the first eye were not significantly different between the unilateral and bilateral groups (**Table**).

We defined the interval between the onset of the first MH and that in the second eye as the bilateral interval. If the second eye developed an MH within 1 month of onset in the first eye, the bilateral interval was set to 0. The mean (SD) bilateral interval among all patients was 26.1 (28.0) months (range, 0-122 months). The difference in the mean bilateral interval between men and women was not significant ( $P = .38$ ). The age at onset of an MH in the first eye and its axial length were not significantly correlated with bilateral interval.

The risk of the fellow eye developing an MH estimated by the Kaplan-Meier method was 11.6% at 5 years and 16.7% at 10 years. The cumulative incidence of bilaterality can be described by the following hyperbolic function:  $y = 2.6 + 29.8x / (130.1 + x)$ , with  $R^2 = 0.99$  (**Figure**). Curve-fit analysis showed that the estimated risk of the fellow eye developing an MH was 12.0% at 5 years and 16.9% at 10 years.

**Comment.** Earlier retrospective studies reported that the incidence of developing an MH in the fellow eye with or without a posterior vitreous detachment was 22% for a

mean follow-up of 57 months (37 patients)<sup>1</sup> and 13% within 48 months (340 patients).<sup>2</sup> Ezra et al<sup>3</sup> reported that the incidence of developing an MH in the fellow eye without a posterior vitreous detachment (114 patients) was 15.6% at 5 years by Kaplan-Meier analysis. Although the long-term incidence of developing an MH in the fellow eye may depend on the patient demographic characteristics and vitreoretinal interface features, our large-scale study showed that the cumulative incidence of bilaterality was well fit by a hyperbolic function. The findings of the curve-fit analysis suggested that the estimated risk was 21.9% at 20 years and 24.5% at 30 years, although these estimates will have to be confirmed by longer longitudinal studies. Because the appearance of the vitreoretinal interface in spectral-domain optical coherence tomographic images is associated with the risk of developing an MH in the fellow eye,<sup>6</sup> further studies are required to determine the long-term risk in the fellow eye based on spectral-domain optical coherence tomographic features.

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## Reversal of Poliosis and Vitiligo Following Vogt-Koyanagi-Harada Disease

**V**ogt-Koyanagi-Harada (VKH) disease is a chronic multisystem disorder characterized by an acute onset.<sup>1-4</sup> The disease may be associated with signs of meningeal irritation and may later develop integumentary signs of poliosis and vitiligo that are valuable in the diagnosis of the disease. Poliosis and vitiligo occur as late clinical manifestations of VKH disease and help