were generally treated with globe-preserving therapies with no histopathological confirmation of the malignancy.

The 2 lesions described in this report exhibited many of the clinical features predictive of growth and documented growth, but only one of the lesions was malignant. With the exception of one feature, posterior margin adjacent to the disc, the benign choroidal nevus demonstrated the same array of predictive factors for growth as the malignant melanoma did. Moreover, the nevus showed the greater growth in height (2.0 mm vs 1.7 mm), but over a longer period (5 years vs 2 years). On a clinical basis, it was not possible to distinguish this enlarging nevus from a growing melanoma.

Augsburger and colleagues have advocated the use of transvitreal biopsy of small melanocytic choroidal tumors to achieve an accurate diagnosis. With their technique, they were able to obtain a sufficient aspirate for cytdiagnosis in 65% of cases, but in 18% of those cases, the biopsy specimen showed intermediate cells consistent with either atypical nevus or low-grade melanoma.

Growth or enlargement of choroidal nevi has been previously documented. Our report demonstrates that documented growth is not an unequivocal indicator of melanoma for small melanocytic tumors.

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Bilateral Conjunctival Nodules: An Unusual Manifestation of Vogt-Koyanagi-Harada Syndrome

Vogt-Koyanagi-Harada (VKH) syndrome is an autoimmune disorder against melanocytes causing inflammation of melanocyte-containing tissues, such as uvea, skin, ear, and meninges. Patients

Figure 5. Cross section of the peripapillary choroidal tumor of case 1 shows an epithelioid cell-type malignant melanoma (hematoxylin-eosin, original magnification ×100).

Figure 6. Cross section of the central aspect of the macular tumor of case 2 shows a benign spindle nevus of choroid with scattered melanophages within the lesion (hematoxylin-eosin, original magnification ×100).
with VKH syndrome usually have bilateral granulomatous panuveitis associated with poliosis, vitiligo, alopecia, dysacusia, and signs of meningeal irritation.

The exact pathogenesis of VKH syndrome is not certain. Sugiuira reported that autoantibodies of VKH play a major role in melanocyte destruction via antibody-dependent, cell-mediated cytotoxicity. Melanoocyte-specific antigens, such as tyrosinase proteins, can induce a VKH-like autoimmune condition in rats and have been considered to be VKH specific. Previous viral infection might trigger immune reactions because of the homology in protein sequence between such microorganisms and ocular antigens, a process known as molecular mimicry. The strong associations of HLA-DR53, HLA-DR4, and HLA-DQ4 antigens with VKH syndrome further underscore an immunogenetic mechanism of the disease.

Because of the variations in the clinical manifestation of VKH syndrome, early detection and accurate diagnosis have been challenging. Herein, we report a case of VKH syndrome with bilateral conjunctival nodules, the rare initial sign of VKH syndrome. Likely differential diagnoses are also discussed.

**Report of a Case.** A 27-year-old Ethiopian woman arrived for examination in February 2001 complaining of bilateral eye pain and redness for 1 month. The patient had chronic headache for several months, which did not respond to oral, nonsteroidal, anti-inflammatory drugs. No history of surgery or ocular trauma was reported. The patient had no history of tuberculosis or syphilis.

The best-corrected visual acuity was 20/25 OD and 20/20 OS. The intraocular pressures were normal. Slitlamp examination revealed moderate anterior chamber inflammation with 2+ to 3+ cells and flare, without keratic precipitates in either eye. The conjunctiva was injected, and multiple small, elevated, and pigmented nodules were noted in the superior bulbar conjunctiva of both eyes (Figure 1). The initial fundus examination showed a normal fundus without vitritis.

Uveitic laboratory workups were performed. Results from the fluorescent treponemal antibody absorption test result and the purified protein derivative skin test were negative. Results from other laboratory tests, including complete blood count, serum chemistry, and urinalysis, were within normal limits. Test results for serum angiotensin-converting enzyme, lysozyme, and antinuclear antibody showed normal values. HLA-B27 antigen was not detected. A chest x-ray film showed a normal image without pulmonary opacifications or nodules.

The patient was treated with a topical corticosteroid in both eyes for 1 week. However, the anterior uveitis was more severe with an increased reaction of 3+ cells and flare in the anterior chamber. Her vision was markedly decreased to 20/50 OD and 20/400 OS. The patient also suffered from persistent headache, severe back pain, neck stiffness, and dysacusia. Fundus examinations demonstrated serous retinal detachment and retinal pigment epithelial detachment at the macular area of both eyes (Figure 2). A fluorescein angiogram demonstrated multiple punctate areas of hyperfluorescence in the macular region in the transient phase. In the late phase of the angiogram, we found rather dramatic dye leakage into the area of the detached pigment epithelium as well as the detached neurosensory retina in both eyes. The areas of hyperfluorescence corresponded nearly identically to the clinically observed areas of detachment.

Based on the presence of bilateral iridocyclitis, bilateral posterior uveitis with exudative retinal detachment, and neurological signs (headache, dysacusia, and neck stiffness), fulfilling the diagnostic criteria of VKH syndrome established by the American Uveitis Society in 1978, a diagnosis of VKH syndrome was made. The patient was initially treated with topical corticosteroids combined with 80 mg of oral prednisolone daily. One month later, gradual resolution of the conjunctival nodules, anterior uveitis, and serous retinal detachment was noted. The dose of oral prednisolone was then slowly tapered during the next 2 months. Three months later, the conjunctival nodules completely resolved in both eyes. Her final visual acuity improved to 20/20 OU after 5 months of treatment. Remission was maintained for at least 1 year after oral prednisolone treat-

**Figure 1.** Slitlamp photographs of the conjunctival nodules. Multiple small pigmented nodules appear at the superior bulbar conjunctiva of both eyes. A, Right eye. B, Left eye.
ment was discontinued. During follow-up, no cutaneous involvement, such as alopecia, poliosis, or vitiligo, was noted.

**Comment.** Inflammatory nodules are an important characteristic of granulomatous uveitis. For example, Koeppe and Busacca nodules of the iris in sarcoidosis or Dalen-Fuchs nodules of the retina in VKH syndrome can often be observed. However, a Dalen-Fuchs nodule is not pathognomonic for VKH syndrome; it can also be found in other diseases with granulomatous inflammation, eg, sympathetic ophthalmia, sarcoidosis, and tuberculosis. Typically, a Dalen-Fuchs nodule is a dome-shaped lesion consisting of epithelioid cells and lymphocytes with overlying retinal pigment epithelium.

Conjunctival nodules are a common feature of various forms of granulomatous uveitis, such as sarcoidosis. To the best of our knowledge, such nodules in VKH syndrome have never been reported. Since melanocytes of the conjunctiva and the uvea are known to have a common embryological origin in the neural crest, this may explain why antimelanocytic immunity can develop not only in the uvea but also in the conjunctiva in VKH syndrome. Although there is no direct pathological proof, a similar mechanism to that of Dalen-Fuchs nodules might be responsible for the development of conjunctival nodules.

Autoimmunity against melanocytes within conjunctival epithelium causes the accumulation of inflammatory cells and the migration of melanocytes to form these pigmented nodules. The conjunctival nodules in our patient appeared primarily in the superior bulbar conjunctiva, while Dalen-Fuchs nodules are known to occur mostly in the inferior midperipheral retina. Such a geographic predilection is of interest, and further evidence from conjunctival biopsy is necessary to confirm the pathogenesis.

Vogt-Koyanagi-Harada syndrome can be categorized clinically into 4 phases: prodromal, acute uveitic, chronic convalescent, and recurrent. Posterior segment involvement with serous retinal detachment in the acute uveitic phase is the most common initial ocular finding. Compared with Dalen-Fuchs nodules in the convalescent phase and iris nodules in the recurrent phase, the clinical course of conjunctival nodules in our patient is in the early uveitic phase, even prior to the development of serous retinal detachment. Several reports suggest that complications in VKH syndrome are related to a longer duration of disease and more episodes of recurrence. Accurate diagnosis of VKH syndrome and prompt therapy with systemic steroids in the early stage might help decrease the visual sequelae following posterior uveitis, such as subretinal neovascularization or macular edema. The conjunctival nodules in VKH syndrome, similar to other ocular manifestations, would remit with systemic steroid treatment. Its complete regression in our case suggests that inflammation may play an important role in the formation of nodules.

There is no specific diagnostic test for VKH syndrome; therefore, the diagnosis is based on a combination of clinical suspicion and exclusion of other forms of uveitis. Sarcoidosis is a multisystem granulomatous disease of unknown etiology that commonly affects the lung, lymph nodes, skin, and eyes. In about 28% of patients with sarcoidosis, the eyes are involved. Among all ophthalmic sarcoid manifestations, conjunctival granulomas and uveitis are found most frequently at initial examination; they account for 47% and 28%, respectively, of ocular manifestations. However, the appearance of conjunctival nodules in this patient was small and pigmented on the bulbar conjunctiva, which is not a typical finding for sarcoidosis, with tan-yellow, millet seed–like nodules on the palpebral or bulbar conjunctiva. Results from tests for sarcoidosis, including a chest radiograph and tests to measure levels of serum lysozyme, and angiotensin-converting enzyme, were all normal. Other characteristic posterior segment findings of sarcoidosis, such as chorioretinitis and periphlebitis with a candlewax-drippings appearance, were not seen in our patient.
Another differential diagnosis is tuberculosis, which can be manifested as both anterior and posterior uveitis or as conjunctival nodules and phlyctenules. However, the majority of ocular tuberculosis is unilateral, and cases of ocular involvement is only rarely noted in systemic tuberculosis. One study reported only 28 cases of iritis among 10,535 patients with tuberculosis.10 The chest x-ray film and a purified protein derivative skin test in this patient showed no evidence of tuberculosis.

Gout, known to deposit urate crystals in ocular tissues, can cause conjunctival nodules, band keratopathy, and rarely anterior uveitis. However, deposits in the conjunctiva, as well as in the cornea, have been described within interpalpebral areas. The location of urate deposition differs from our patient with conjunctival nodules only in the superior bulbar conjunctiva.11

This article describes a patient with VKH syndrome with an initial manifestation of bilateral conjunctival nodules and anterior uveitis. The diagnosis was supported by the fulfillment of the diagnostic criteria of VKH syndrome and the exclusion of other possible diseases. To the best of our knowledge, this article represents the first case of conjunctival nodules in VKH syndrome. We thereby stress the importance of clinical awareness of conjunctival nodules as the initial sign of VKH syndrome.

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Multifocal Choroiditis and Acute Posterior Multifocal Placoid Pigment Epitheliopathy Occurring in the Same Patient

The white spot syndromes are a group of idiopathic inflammatory diseases of the retina characterized by visual loss in association with areas of retinal whitening. This category includes diseases such as multifocal choroiditis (MFC), punctate inner choroidopathy, multiple evanescent white dot syndrome (MEWDS), serpiginous choroiditis, and acute posterior multifocal placoid pigment epitheliopathy (APMPE). To our knowledge, no definite infectious or immune etiology has been proved for these various entities.1 There have been reports of 2 of these entities occurring in the same patient (acute macular neuroretinopathy and MEWDS2 and MFC and MEWDS3). Patients have been described as having overlapping features of these various conditions, eg, MEWDS and MFC. We describe a patient who at age 18 years showed findings consistent with APMPE, with visual loss in both eyes. This resolved with a return of vision to 20/20 OU. Sixteen years later, he developed new symptoms and exhibited lesions of MFC. The old APMPE lesions remained unchanged.

Report of a Case. An 18-year-old man sought care because of headaches and bilateral central scotomas of 1 week’s duration. He reported...