Small Choroidal Melanoma Associated with Choroidal Neovascularization

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The prevalence of choroidal nevi associated with choroidal neovascular membrane (CNV) is estimated to range between 0.58% and 8.6% [1]. The pathogenesis of CNV is not completely understood. Researchers have suggested that damage caused to the choroid capillaries above the nevi affects the overlying retinal pigment epithelium and triggers production of angiogenic factors that, in turn, cause the development of CNV [2,3]. Hypoxia and inflammation may be involved in the process. Data have been inconsistent with both theories [4].

PATIENT DESCRIPTION

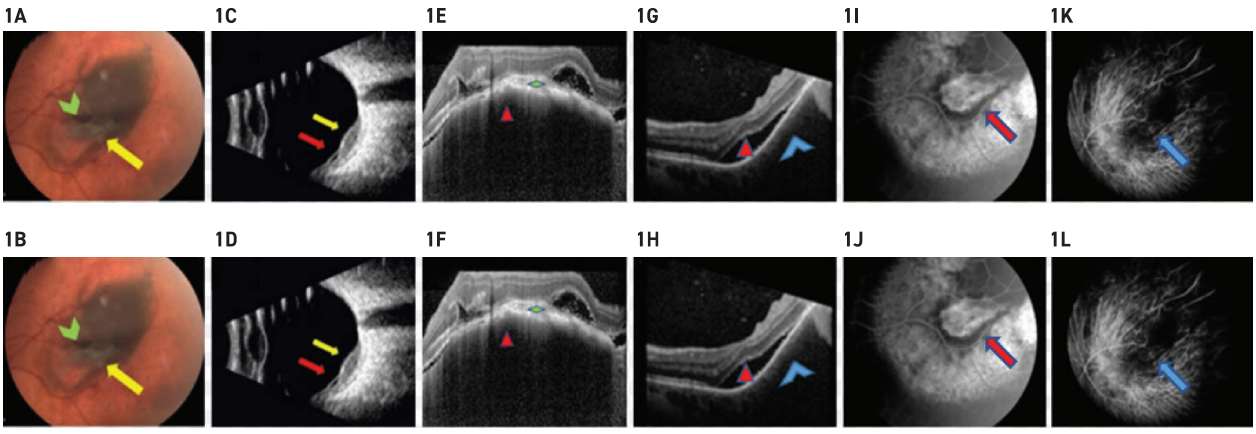
A 49-year-old male with complaints of visual disturbance in his left eye for 2 weeks was examined at our ocular oncology clinic. His past medical history was positive for myectomy due to hypertrophic obstructive cardiomyopathy (2015), infective endocarditis (2010), and non-metastatic grade 2 cutaneous malignant melanoma (2005).

At presentation, his best-corrected visual acuity (BCVA) was 0.1 LogMAR in the left eye and 0 in his right eye. Intraocular pressure and anterior segments were within normal limits in both eyes. Right fundus examination was unremarkable.

Figure 1. Choroidal melanoma with CNV before and after treatment

CNV = choroidal neovascular membrane, FA = fluorescein angiography, ICG = indocyanine green angiography, OCT = optical coherence tomography

[A] On presentation shows an elevated hyperpigmented lesion with a minor pre-retinal hemorrhage (green arrowhead) and exudative detachment surrounding the lesion with a white sub-retinal neovascular membrane (yellow arrow). **[B]** At 5-month follow-up visit, tumor regression, absorption of the pre-retinal hemorrhage (green arrowhead) and atrophic membrane (yellow arrow). **[C]** B mode ultrasound scan on presentation revealed dome shaped small choroidal melanoma measuring 14.17 mm in base, 2.58 mm in thickness (yellow arrow), and serous retinal detachment (red arrow) **[D]** Ultrasound at 5-month follow-up, flat choroidal melanoma (yellow arrow), tumor thickness decreased to 1.46 mm, and maximal basal width to 12.9 mm with resolution of the and serous retinal detachment. **[E]** OCT on presentation imaging cross section of the melanoma (red triangle) with CNV on the apex (green rhombus) and pockets of subretinal fluid, before the treatment. **[F]** OCT at 5-month follow-up visit depicting resolution of the CNV full absorption of the sub-retinal fluid from the tumor apex and flattening or the melanoma (red triangle) after therapy. **[G]** OCT of the macula, melanoma (blue arrowhead) with sub-foveal sub-retinal fluid (red triangle). **[H]** OCT at 5-month follow-up visit, depicting regression of the melanoma (blue arrowhead) and absorption of the sub foveal fluid after therapy (red triangle). **[I]** FA on presentation, early phase (14 seconds) and **[J]** late phase (56 seconds) demonstrates CNM (red arrow) over the melanoma with central and surrounding hyperfluoresence extending to the temporal boarder of the macula **[K]** ICG on presentation, CNV demonstrated at 17 seconds and **[L]** at 80 seconds (blue arrow)



Left fundus examination revealed a superior-temporal elevated pigmented choroidal lesion with a small preretinal hemorrhage, a white subretinal neovascular membrane at the apex of the lesion, and exudative retinal detachment surrounding the lesion [Figure 1A-L].

B mode ultrasound revealed serous retinal detachment, dome shaped solid choroidal hyperechogenic mass measuring 14.17 mm in base, and 2.58 mm in thickness [Figure 1C]. A mode ultrasound showed medium to low internal reflectivity. A white subretinal neovascular membrane was observed at the apex of the lesion as seen in fundus photography [Figure 1A]. Optical coherence tomography (OCT) depicted a CNV over the choroidal mass with subretinal fluid at the apex of the lesion and subfoveal subretinal fluid [Figures 1E, 1G]. On fluorescein angiography (FA) an early phase central and surrounding hyperfluorescence extending to the temporal boarder of the macula were demonstrated [Figure 1I]. In addition, CNV was demonstrated on indocyanine green angiography [Figure 1K]. These findings were suggestive of a small choroidal melanoma with choroidal neovascularization. A systemic workup was negative for extraocular or secondary disease.

The patient was treated with brachytherapy with 125I plaque followed by extra foveal TTT at the time of plaque removal without complications. Due to the presence of CNV and the macular location of the lesions, the patient received three consecutive monthly intravitreal injections of bevacizumab in addition.

After 5 months of follow-up, BCVA remained 0.1 LogMAR in the left eye. The bleeding resolved [Figure 1B], the tumor thickness decreased to 1.46 mm, and the maximal basal width reduced to 12.9 mm [Figure 1D]. OCT depicted resolution of the CNV full absorption of the subretinal fluid [Figures 1F, 1H] with flattening of the melanoma [Figure 1D]. His vision remained stable, and no tumor recurrence or metastasis was noted during 12 months of follow-up.

COMMENTS

To the best of our knowledge, our case is one of the few reports in the literature where CNV was diagnosed along with choroidal melanoma at primary presentation, and it is the first reported case treated with three consecutive intravitreal injections of bevacizumab, which is frequently used in other types of CNV.

From our experience, bevacizumab injections may have contributed to preservation of the 12-month BCVA (0.1 logMAR) in this macular melanoma. This therapeutic approach may be an appropriate treatment for choroidal melanoma and CNV.

Furthermore, we speculate, based on the report by Guerin et al. and colleagues [5], that choroidal melanoma associated CNV may not be as rare as previously believed. Prompt treatment with intravitreal injections of bevacizumab may prevent rapid visual acuity deterioration in these cases.

CONCLUSIONS

Plaque brachytherapy, TTT, and bevacizumab injections can be an effective therapeutic treatment combination for choroidal melanoma associated with CNV.

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Capsule

The painful consequences of long COVID

After the resolution of infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which causes COVID-19, some individuals experience neurological issues such as generalized pain. **Serafini** and co-authors investigated the basis for pain associated with long COVID and other neuropathies. Mechanical hypersensitivity in SARS-CoV-2-infected hamsters was associated with a durable gene expression signature in sensory neurons that partly resembled those of mouse models of neuropathic pain. Some of these genes, including the one encoding the RNA-binding protein ILF3, were validated as potential therapeutic targets in mice.

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