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Editorial

Glaucomatocyclitic crisis and glaucomatous optic neuropathy

Glaucomatocyclitic crisis is a rare disease first described by Posner and Schlossman[1] in 1948. The disease is characterized by unilateral recurrent attacks of increased intraocular pressure (IIOP) associated with mild anterior uveitis and an open angle.[1,2] It is classified as an inflammatory glaucoma. In contrast to the common presentations of eye pain and nausea in acute angle-closure glaucoma, there is always minimal discomfort despite marked IIOP (up to 40–50 mmHg) when glaucomatocyclitic crisis attacks. The common presenting symptoms are mild unilateral eye pain and blurred vision or halo vision. The affected eye may show little or no hyperemia. Typically, the anterior uveitis is mild, and keratic precipitates are few in number and fine, round, and discrete in appearance.[2] There is no peripheral anterior synechiae in this disease. It tends to affect patients between 20 and 50 years of age.[3] The IIOP episode often spontaneously resolves in days to weeks. Medical treatment, including IOP-lowering and anti-inflammatory agents, is indicated to reduce IOP, inflammation, and prevent pressure-related optic nerve damage.[2] The etiology of this disease remains unknown, however, the role of cytomegalovirus in the pathogenesis of glaucomatocyclitic crisis has frequently been postulated.[4,5]

In this issue of the *Taiwan Journal of Ophthalmology*, a case report entitled "Detection of the progression of retinal nerve fiber layer loss by optical coherence tomography in a patient with glaucomatocyclitic crisis" by Dr Jen-Chia Tsai is included.[6] The author reports that a progressive decrease in the thickness of the retinal nerve fiber layer was demonstrated by Stratus optical coherence tomography following acute IOP elevation in a patient with glaucomatocyclitic crisis. The author suggests that prompt treatment and longitudinal monitoring are necessary to prevent and detect glaucomatous damage in such a disease. I would like to highlight two points regarding glaucomatocyclitic crisis and glaucomatous optic neuropathy.

1. Glaucomatous optic neuropathy can induce cumulative effect due to recurrent bouts of markedly IIOP in glaucomatocyclitic crisis. Just as the author states, glaucomatocyclitic crisis is a relatively benign and often self-limiting disease. However, some patients have been known to develop glaucoma-related visual field defects as a result of repeated episodes.[7,8] Therefore, the IOP spike should be treated adequately and promptly for each glaucomatocyclitic crisis attack.
2. Glaucomatocyclitic crisis may have underlying primary open-angle glaucoma (POAG). There are well-documented reports on patients with glaucomatocyclitic crisis having underlying POAG.[9–11] When a patient with POAG or normal tension glaucoma has an episode of monocular acute IIOP, we should consider the possibility of a secondary cause including a glaucomatocyclitic crisis episode.

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