A Case of Normal Tension Glaucoma Associated with Buerger’s Disease

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Open angle glaucoma, a slowly progressive optic atrophy, is clinically characterized by visual field defects corresponding to excavation of the optic disc, called glaucomatous cupping. Open angle glaucoma is further divided into primary open angle glaucoma caused by elevated intraocular pressure (higher than the normal limit of 21 mmHg), and normal tension glaucoma, in which intraocular pressure is in the normal range. Here we report a case of normal tension glaucoma associated with Buerger’s disease, also known as thromboangiitis obliterans, which causes systemic blood flow disturbance. A 66-year-old man suffering from Buerger’s disease for 10 years was diagnosed as having branch retinal artery occlusion in his left eye. He was referred to our clinic due to progressive visual field disturbance in that eye. Ophthalmologic examinations revealed occlusion in the inferotemporal retinal artery in the left eye, and glaucomatous cupping, normal intraocular pressure, retinal vessel tortuosity and retinal arteriosclerosis in both eyes. Visual field examination revealed decreased retinal sensitivities in the areas within the visual field arches above and below fixation from the blind spot to the median raphe, corresponding to the arcuate retinal nerve fibers comprising the Bjerrum areas and the area corresponding to the retinal artery occlusion. Buerger’s disease is characterized by the development of segmental thrombotic occlusions and vasospasm of the medium and small arteries. Our case suggests that the blood flow disturbance due to arteriosclerosis, thrombotic occlusions and vasospasm associated with Buerger’s disease might affect the ophthalmic circulation system, thereby contributing to the etiology of normal tension glaucoma.

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revealed that more than 90% of OAG is NTG in Japan (Iwase et al. 2004).

Although the precise pathological mechanisms causing NTG are still unknown, many investigators have reported that several kinds of factors may be involved in the etiology of NTG, including an autoimmune mechanism (Cartwright et al. 1992; Maruyama et al. 2000), vasospasm (Cartwright et al. 1992), endothelin-1-related microvascular dysfunction (Gass et al. 1997), platelet hyperaggregation (Matsumoto et al. 2001). Clinically, the optic disc hemorrhages are recognized more frequently in patients with NTG than those with primary OAG (Hendrickx et al. 1994). This therefore suggests that changes in local blood circulation within the optic nerve head are most likely involved in the causative mechanisms of NTG. Nevertheless, no one has been paid attention to the relationship between NTG and systemic vascular disease so far. In the present case report, we demonstrate a rare case of NTG associated with Buerger’s disease, which causes systemic blood flow disturbances. This provides us with an etiological consideration for the level of IOP accompanying NTG.

CASE REPORT

A 66-year-old man was referred to our glaucoma clinic due to progressive visual field disturbance in his left eye in August 2003. He had a lumbar sympathectomy for treatment of Buerger’s disease in 1993, an acute myocardial infarction in 1995, and thereafter had been taking nitroglycerin 25 mg and nifedipine 10 mg a day.

In our first examination, visual acuity was 1.5 in the right eye and 1.2 in the left, and IOP was 13 mmHg in both eyes. Slit-lamp examination showed slight nuclear sclerosis in both eyes. Ophthalmoscopic examination revealed plaques and occlusion in the infero-temporal retinal artery and retinal edema in the infero-temporal area of the macula in his left eye, and retinal vessel tortuosity, arteriosclerosis and glaucomatous cupping in both eyes (Fig. 1). With Goldmann 3-mirror contact lens, the cup-to-disc ratio was 0.8 in the right eye and 0.6 in the left, and the temporal superior rim showing undermining of the cup margin in the right eye was thinner than the temporal inferior rim. In comparison, the temporal superior rim only showed slight undermining of the cup margin in the left eye and was same as the temporal inferior rim. Humphrey Field Analyzer, program central 30-2 (Humphrey Instruments, San Leandro, CA, USA) revealed decreased retinal sensitivities in the Bjerrum area (the areas within the visual field arches above and below fixation from the blind spot to the median raphe, corresponding to the arcuate retinal nerve fibers) of both eyes and retinal artery occlusion area of the left eye (Fig. 2). Magnetic resonance image...
showed no remarkable changes. Fluorescein angiography revealed retardation of arm-to-retina circulation time and filling defect of the infero-temporal retinal artery in the left eye. From these findings, we diagnosed him as having NTG with branch retinal artery occlusion. In addition, to evaluate disturbances of the systemic vascular circulation, ankle brachial pressure index (ABI) and pulse wave velocity, which are non-invasive methods for evaluation of arteriosclerosis, were simultaneously measured using ABI-form (Japan Colin, Komaki). These measurements of bilateral arm and ankle (brachial and posterior tibial arteries, respectively) blood pressure by oscillometric method, according to the protocol described by manufacturer, were 0.69 (normal 0.9-1.3) and 1,839 cm/s, respectively.

**Discussion and Conclusion**

Buerger’s disease (Flammer et al. 2001), which is pathologically known as thromboangiitis obliterans and is mainly found in young males and cigarette smokers, is a clinical syndrome characterized by the development of segmental thrombotic occlusions and vasospasm of the medium and small arteries mostly in the lower extremities. In terms of the etiology of this syndrome, recent immunohistochemical studies have suggested that Buerger’s disease has relation to the autoimmune mechanism. In fact, Buerger’s disease is known to be associated with increased anti-cardiolipin antibodies and hyperhomocysteinemia (Flammer et al. 2001). As ocular changes in this disease, early sclerotic changes and arteriosclerosis of retinal arteries were reported (Bernardczykowa and Zawilski 1991), suggesting that changes in blood flow by vasospasm, arteriosclerosis and thrombotic occlusions occur within the ocular arteries as well as systemic arteries in Buerger’s disease. In the present report, we described a rare case of Buerger’s disease associated with NTG. Many factors have been suggest-
ed in the literature as a possible major etiology of NTG, such as the autoimmune mechanism (Cartwright et al. 1992; Maruyama et al. 2000), vasospasm (Cartwright et al. 1992), endothelin-1-related microvascular dysfunction (Gass et al. 1997), and platelet hyperaggregation (Matsumoto et al. 2001). In contrast, local ocular blood flow disturbance within the optic nerve head, associated with Buerger’s disease, may have caused the glaucomatous optic neuropathy in our present case. In fact, there has been very little attention in the literature concerning the relationship between systemic vascular circulation disturbances and OAG so far. Our present case indicates the potential etiologic significance of systemic vascular circulation disturbance, such as Buerger’s disease, in relation to the diagnosis and management of OAG, especially NTG.

References


