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same team who conducted a prospective study on 26 POAG patients and 27 PEG patients undergoing glaucoma surgery and 31 patients undergoing phacoemulsification cataract surgery and concluded there was an increased concentration of IgG HP antibodies in the aqueous humor of surgical eye specimens from glaucoma patients. These studies by Kountouras et al were used in correspondence addressed to Weinreb et al, in which Kountouras criticized the lack of mentioning of the role of HP in glaucoma by Weinreb.

Deshpande et al also performed a similar prospective, nonrandomized comparative study based on the serological and aqueous humor examination of 50 glaucoma patients and a control group of patients undergoing phacoemulsification cataract surgery. They found a statistically significant difference between POAG patients and PEG and control patients, as far as the concentration of serum HP IgG antibodies was concerned. However, they did not find any significant correlations between the patient groups in regard to HP IgG antibody concentration in the aqueous humor. Galloway et al performed a prospective study based on a sample of 97 patients with POAG, PEG, and ocular hypertension and an analogous control group; they did not find statistically significant differences in the concentration of serum HP-IgG antibodies between groups, thus leaving an open field for further research and scientific discussion. Similarly, Kountouras and associates investigated seropositivity to HP and to its cytotoxin-associated gene A (CagA) product in 51 patients with various types of glaucoma and compared the findings to those of a control group of 36 cataract patients. They found no significant association between HP infection and CagA seropositivity with glaucoma. Handa et al noted the connection of CagA to various pathological pathways, leading to irregular gastric cell activity, apoptosis, and carcinogenesis.

There has also been much debate regarding the mechanisms that might be involved in the possible association of HP infection with glaucoma. Is there a causal relationship between them or do they share common predisposing or precipitating factors and pathophysiological backgrounds? Various authors believe that HP infection acts through the release of proinflammatory and vasoactive substances, through the release of endothelin-1, nitric oxide, and oxygen free radicals, and through the induction of oxidative stress, the destruction of mitochondrial DNA and the induction of apoptotic cell death, which may be the main reason for glaucomatous neuropathy. This apoptotic process may be linked with the FASL protein, a type-II transmembrane protein belonging to the tumor necrosis factor (TNF) family. The binding of this protein with its receptors differentiates the response of the immune system, is believed to play a role in oncogenetic processes, and is also connected with apoptotic cell death.

**Discussion**

Over the last decade, various researchers have examined the existence of a possible link between HP infection and glaucoma. Some studies have supported such an association, with suggested underlying mechanisms including the induction of inflammatory responses as well as apoptotic processes that lead to glaucomatous neuropathy. Although some support for this seems to exist, contradictory results from other related studies leave room for further investigation. This controversy is best reflected in the scientific debate that took place between Weinreb et al and Kountouras et al, with Kontouras et al claiming there was a positive correlation between HP and glaucoma. The establishment of such a causal relationship will probably have important practical applications as the eradication of HP might lead to developments in the treatment of glaucoma.

**Disclosure**

The authors report no conflicts of interest in this work.

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