Commentary

Inflammatory bowel diseases (IBDs) form a group of chronic remittent inflammatory affections of the gastrointestinal tract, among which ulcerative colitis is one of the most common. The overall IBD prevalence approximates 500-900 cases per 100,000 individuals, and has shown a marked increase during the last decades, mainly in westernized nations.[1] Ulcerative colitis has traditionally been considered a disorder limited to the colonic mucosa. However, as it has been shown that ulcerative colitis is frequently accompanied by various extraintestinal disorders, there is increasing evidence that ulcerative colitis may also manifest in the nervous system, both in the peripheral and in the central nervous system. The nervous system is affected through three major pathogenic entities, which can be differentiated as: (i) Cerebrovascular disease as a consequence of thrombosis and thromboembolism; patients with ulcerative colitis are at an increased risk for stroke. Postmortem examinations have suggested that venous thrombosis of all sites may complicate ulcerative colitis in 39% of the cases, but only 1% of the patients are clinically affected;[2,3] (ii) systemic and cerebral vasculitis;[2] (iii) probably immunemediated neuropathy and cerebral demyelination, which may lead to optic neuritis or multiple sclerosis. The prevalence of general autoimmune disorders in people with ulcerative colitis is three times greater than that in the general population. Acute and chronic inflammatory neuropathies occur more commonly than expected, and it has been postulated that *Campylobacter jejuni* may exacerbate preexisting IBD.[2,4]

It has recently been recognized that seizures may develop in patients due to antibodies against cell-surface antigens and synaptic proteins in the brain. Although rare, this diagnosis should be considered in patients who present with seizures and status epilepticus who fail to respond to conventional therapy. Patients with seizures due to autoimmune response would become seizure free or show significant improvement in their neurological status after treatment with steroids, intravenous immunoglobulins or other immunotherapy.[5] Moreover, the recently proved frequently co-occurrence between epilepsy and 12 autoimmune diseases: Type 1 thyroiditis, Crohn's disease, ulcerative colitis, systemic lupus erythematosus, antiphospholipid syndrome, Sjögren syndrome, myasthenia gravis and celiac disease (odds ratio, 3.8; 95% CI, 3.6-4.0; $P < 0.001$), supports that patients with either epilepsy or autoimmune disease should undergo surveillance for the other condition.[6]

Since Mccabe 1979, the autoimmune origin of sudden sensorineural hearing loss is well documented. Such linkage has been confirmed by the good response of the hearing loss to immunosuppressive therapy and detection of several antibodies in the serum of the patients, especially the

anti-68 KDa antibodies.[7] The association of sensorineural hearing loss and ulcerative colitis is well documented, and it is speculated that this is autoimmune in origin.[8] Kumar *et al*.[8] reported that pure tone audiometry shows significant sensorineural hearing loss over all frequencies in patients with ulcerative colitis compared with controls, although none of the patients in their study group complained of development of hearing loss. Their preliminary study indicated that subclinical sensorineural hearing loss is associated with ulcerative colitis.

Yazici *et al*.[9] endorse the autoimmune origin of sudden sensorineural hearing loss and epilepsy that developed in a patient with ulcerative colitis.

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