exacerbates neurological symptoms when used to treat uveitis. Tumour necrosis factor antagonists such as infliximab and etanercept are reserved for very aggressive forms of the disease, relapses after immunosuppressant treatment, or poor response to steroids. Venous sinus thrombosis is treated with antithrombotic drugs. Choice of drug and length of treatment are controversial topics.

Regarding prognosis, patients tend to respond well to steroids, and only a third of them suffer relapses or progressive disease. Up to 20% of these patients have died at 7 years after diagnosis, which testifies to the severity of the disease. In conclusion, BD should be included in the differential diagnosis of BME cases. BD is even more likely if BME is associated with mucocutaneous lesions (especially urogenital ulcers) or if the red nucleus is unaffected.

References


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Wernicke’s encephalopathy secondary to hyperthyroidism and ingestion of thiaminase-rich products

Encefalopatía de Wernicke secundaria a hipertiroidismo e ingesta de productos ricos en tiaminas

Wernicke’s encephalopathy (WE) is a neurological disease caused by thiamine deficit. In some autopsy series, its incidence rates range from 0.4% to 2.8%, and prevalence is especially high among susceptible populations, including patients with alcoholism. It is estimated that 70% of all cases are not diagnosed until severe complications have appeared; the mortality rate approaches 17%.

Although alcoholism is its most frequent cause, WE has also been described in patients with malnutrition, hyperemesis, a history of bariatric surgery, cancer, and parenteral nutrition. It may not be detected in these situations, which can lead to serious complications. This letter presents an exceptional case of WE secondary to hyperthyroidism and consumption of foods rich in thiaminase.

We present the clinical case of a 54-year-old woman referred to the Emergency Department with a 3-week history of asthenia and vomiting. During that time she had been ingesting little more than water and tea. The patient

status presents in 82% of all patients, with manifestations that include disorientation, apathy, lethargy, confusion and agitation, memory impairment, and even stupor and coma. In contrast, ophthalmoparesis presents in 29% of cases and includes horizontal nystagmus, paralysis of the lateral rectus muscles with diplopia and internal strabismus, conjugate gaze palsy, ptosis in rare cases, and miosis in long-standing processes. Ataxia appears in 23% of cases with unstable tandem gait, short strides, and increased base of support. Other signs may include polyneuropathy as a manifestation of nutritional deficiency, and tachycardia with postural hypotension due to autonomous nervous system disorders or cardiac beriberi. Diagnosis is basically clinical, and the disease is reversible with administration of thiamine. MR imaging studies provide additional information with a low sensitivity of about 53% and a higher specificity of 93%. Studies show increased T2-weighted signal intensity in the periventricular area, mamillary bodies, and periaqueductal grey matter, as well as demyelinating lesions in the cortex and corpus callosum.

Treatment consists of administering intravenous thiamine. Ophthalmoparesis was the first manifestation to improve (resolving over a few hours except for nystagmus), followed by ataxia within a few days (40% recovery) and state of consciousness (improving in 2–3 weeks). Our review of the literature yielded few cases of Wernicke’s encephalopathy associated with or secondary to hyperthyroidism. Most cases were associated with hypercatabolism and increased nutritional needs. Thiaminases are known to be present in raw fish, shellfish, tea, and coffee. These enzymes act as natural thiamine antagonists and may have contributed to the development of WE in our patient, who had been consuming little more than tea in the preceding weeks.

In conclusion, the case was diagnosed as WE secondary to hyperthyroidism that was probably exacerbated by the thiaminases present in tea. The case we present is interesting as it demonstrates that symptoms compatible with WE may develop without the classic antecedents of malnutrition or alcoholism.

References

LETTERS TO THE EDITOR


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