CASE REPORT

Wernicke’s encephalopathy and anabolic steroid drug abuse. Is there any possible relation?

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Abstract

Wernicke’s encephalopathy is a reversible, neurologic disorder due to thiamine deficiency which is mainly related to chronic alcohol abuse. We report a case of a young male patient, who was bodybuilder and anabolic drug user, in whom encephalopathy was diagnosed after a short medical course in the ICU after a major upper gastrointestinal bleeding (Mallory-Weiss syndrome) and hypovolemic shock. His clinical condition was typical for Wernicke’s encephalopathy and although neuroimaging tests were not indicative, the patient received thiamine supplement therapy, which resulted in rapid clinical improvement. The diagnosis was based only on clinical signs and anabolic drug abuse was considered as a possible predisposing factor for the manifestation of the syndrome.

Key words: Wernicke’s encephalopathy; oculomotor disturbancies; thiamine deficiency; anabolic drugs; bodybuilding

Introduction

Wernicke’s encephalopathy, named after Karl Wernicke, who first described its clinical symptoms, in 1881, is attributed to thiamine deficiency and is characterized by the existence of a typical clinical triad: encephalopathy, oculomotor dysfunction and ataxia. Although malnutrition is the main cause especially in alcoholic patients, many other conditions are known to be related. Its prevalence is 0.1 – 2.8% and even greater in autopsy studies. Remarkably, during the last two decades it is recognized with increasing frequency among nonalcoholic patients in a variety of clinical settings, mainly iatrogenic ones. In this case the syndrome may give rise to the classic Wernicke-Korsakoff syndrome (WKS) which includes chronic mental disorders and memory deficits with mortality rate estimated up to 20%. While the diagnosis is mainly based on the characteristic clinical presentation, computed tomography (CT) and magnetic resonance imaging (MRI) of the brain may be very helpful and are considered mandatory in order to exclude other conditions. The only treatment option is thiamine replacement to avoid permanent damage.

Case Report

We report a case of a 33-year-old male bodybuilder patient who was admitted to the emergency department with acute gastrointestinal bleeding (hematemesis and hematochezia) after intense vomiting. He had been drinking alcoholic beverages the night before. Urgent endoscopy revealed a Mallory-Weiss syndrome (laceration of the gastroesophageal junction lining caused by the severe vomiting) which was treated with local ethanolamine application. The bleeding relapsed twelve hours later and he was admitted to the Intensive Care Unit (ICU) with hypovolemic shock. With rapidly deteriorating mental status, profound anemia and metabolic acidosis he was intubated and mechanically ventilated. Further management comprised of endoscopic ligation with clips and ablation of the bleeding lesion, while the hypovolemic shock was resolved after abundant whole blood, fresh frozen plasma, platelet and crystalloid/colloid transfusion. His situation was further complicated with rhabdomyolysis in need of continuous renal replacement therapy. During his stay in the ICU, he was under standard total parenteral nutrition (TPN), according to his caloric requirements (standard thiamine supplementation 3,5 mg/day).

Two weeks later he was stabilized, but during weaning from the ventilator, he showed impaired mental status [global confusional state with a Glasgow Coma Scale (GCS) 13/15], ophthalmoplegia (bilateral abducent nerve palsy), nystagmus, clonus and increased deep tendon reflexes of the lower extremities. A diagnosis of encephalopathy was suspected, with Wernicke’s being the most probable, so he underwent an MRI scanning; nevertheless, apart from some generalized atrophy, this was not indicative for the specific or any other encephalopathy. A thorough clinical history review revealed anabolic steroid drug abuse for the last 12 months, including testosterone analogues (Winstrol®, Anavar®, Trenbolone®), human growth hormone and insulin. A thiamine supplementation regimen was started immediately (Neurobion®, B-complex vitamin therapy containing 100 mg thiamine) intramuscular injection every 8 hours for five days, followed by oral intake for the next seven days.

Four days after treatment initiation he was successfully
The etiological factor in the presented case might be the patient’s history of anabolic steroid drug abuse for 12 months before his admission, and the acute overdose which triggered the manifestation of the syndrome. It is well established in the literature, that function of multiple systems in human body is impaired in case of abuse of these pharmacologic agents. Apart from the damage cased in organs like liver, heart, lungs and kidneys, anabolics are proved to be extremely toxic for the central nervous system (CNS). In many cases this toxicity against CNS cells is irreversible, especially if the abuse is chronic. Known anabolic steroids that can cause encephalopathy are methandrostenolone and nandrolone. Acute withdrawal of drugs with anabolic effects (such as gamma-hydroxybutyrate), can cause encephalopathy with symptoms that mimic Wernicke’s encephalopathy. A literature search failed to prove direct connection between the specific anabolics used by our patient and withdrawal syndrome, but both excessive use and sudden withdrawal from these agents could not be excluded as a possible predisposing factor.

In addition, a detailed and focused history as reported by the patient and his family, revealed the patient’s specific dietary habits for gain weight and muscle augmentation along with neglect of foods, sources of thiamine. He refused alcohol addiction. We did not find any other common cause for his clinical condition. Liver function was normal and no other diseases related with the syndrome such as HIV or malabsorption, were diagnosed.

Advanced neuroimaging techniques are indicated once clinical symptoms appear. While the specificity of structural MRI is high (up to 93%), its sensitivity remains relatively low (53%) for the detection of acute Wernicke’s syndrome, as it was in the presented case. Neuroimaging findings are more obvious and specific in alcoholics. New MRI techniques (MRI spectroscopy and functional MRI) have better sensitivity and are considered to be the most valuable methods to confirm the syndrome, since no specific routine laboratory test is available.

In conclusion, neurological disorders are not uncommon among ICU patients and may increase morbidity and even mortality when left undiagnosed. High grade of clinical suspicion, detailed medical history and careful neurologic examination or consultation from a specialist, may be extremely valuable for patient’s recovery or even survival. Once a diagnosis of Wernicke’s encephalopathy is suspected, administration of thiamine should be started immediately. If the disease has not fully progressed, this treatment not only prevents its progression but also reverses those lesions that are not yet permanently established.

Conflict of Interest
There is no conflict of interest.

Disclosure
This paper was presented as poster at the 13th Pan-Hellenic Congress of Intensive Care, Athens, 2010 under the title: Wernicke’s encephalopathy post hypovolemic shock in the grounds of Mallory Weiss syndrome, anabolic steroid drug and alcohol abuse, By Christopoulos P, Timplalexi G, Polyzoi A, Hasou E, Lathyris D, Katsanoulas C, and Antoniadou E.

References