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Wernicke Encephalopathy After Gastrointestinal Surgery for Cancer: Causes of Diagnostic Failure or Delay

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ABSTRACT

Wernicke encephalopathy (WE) is a neurological emergency due to thiamine deficiency. We aimed to identify clinical course and causes of diagnostic delay or failure of WE in a group of patients who underwent surgery for gastrointestinal tumors. A retrospective review of clinical, laboratory, neuroimaging, and therapeutic features of 10 patients with WE following abdominal surgery for cancer was carried out. Four patients died; in these subjects, diagnosis was delayed and supplementation of vitamin was absent or likely inadequate. Diagnostic delay or failure was also related to the coexistence of several medical complications at presentation masking typical symptoms of WE. In the surviving patients, outcome was influenced by promptness and type of therapy. Postoperative abdominal bleeding and number of subsequent operations may also had an effect. Postsurgical patients with gastrointestinal tumors may develop a subtle WE. The number of subsequent operations and the severity of postoperative complications may increase the risk of unrecognized WE. The disease should be suspected in postsurgical patients who have unexpected mental status changes, even under prophylactic treatment with vitamins. We suggest that prophylaxis with high doses of thiamine should be undertaken in patients with gastrointestinal tumors before surgery.

KEYWORDS: Gastrointestinal tumors, MRI, neuro-ophthalmology, outcome, therapy, Wernicke encephalopathy

INTRODUCTION

Wernicke encephalopathy (WE) is a well-defined thiamine (vitamin B1) deficiency syndrome leading to an acute neurological disorder associated with high morbidity and mortality (Sechi & Serra, 2007). The classical clinical triad has been well established since Wernicke's first description (Thomson et al., 2008) and includes ocular signs, altered consciousness, and ataxia. The diagnosis, however, is still difficult in clinical practice, with only 20% of diagnosed cases during life (as compared with postmortem autopsied cases; (Donnino, Vega, Miller, & Walsh, 2007; Harper, Giles, & Finlay-Jones, 1986). Therefore, new criteria for diagnosis have been proposed, including the presence of at least two of the following four conditions: dietary deficiency, oculomotor abnormalities, cerebellar dysfunction, and mental status changes or memory impairment (Caine, Halliday, Kril, & Harper, 1997). WE, however, remains a challenging clinical diagnosis, as magnetic resonance imaging (MRI) of the brain may be considered as an adjunctive evidence of disease, potentially affected by low sensitivity (Antunez et al., 1998; Chung, Kim, Yoo, Lim, & Lee, 2003; Fei, Zhong, Jin, Lim, & Lee, 2008; Liu et al., 2006; White, Zhang, Andrew, & Hadley, 2005; Zuccoli, Gallucci, Capellades, Regnicolo, Tumiati, Giadás, Bottari, Mandrioli & Bertolini, 2007; Zuccoli & Pipitone, 2009). Early diagnosis is mandatory because prompt replacement therapy can reverse the neurological condition and brain MRI changes (Thomson, Cook, Touquet, & Henry, 2002). Otherwise, disease progression leads to coma and death (Cook, 2000).

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