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Wernicke's Encephalopathy Postoperatively for a Gastric Tumor: A Pathology which could be Confused with Postoperative Peritonitis

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Abstract **Case Report**

***Background*:** Wernicke's encephalopathy is a neurological syndrome secondary to a thiamine deficiency which is a cofactor for several biochemical reactions in the brain. In the postoperative context, this condition is often underdiagnosed, especially in patients admitted to intensive care given the non-specificity of neurological symptoms which can often have multifactorial origins in this type of patient. We report the case of a patient with Wernicke syndrome postoperatively after a cancer gastrectomy and whose clinical symptoms were initially confused with postoperative peritonitis. ***Case presentation*:** An 78-year-old female patient, initially admitted to the gastrology for treatment of late post-prandial vomiting associated with weight loss. An esophago-gastro-duodenal fibroscopy was performed and revealed a stenosis and impassable mass at the antro-pyloric level. The patient was placed on parenteral undernutrition and rehydration. The pathological examination came back in favor of a moderately differentiated, ulcerated and invasive gastric adenocarcinoma. The indication for oncological gastrectomy was made after multidisciplinary consultation. On the 15th postoperative day, the patient was admitted to the emergency department of our hospital with a clinical presentation of evisceration. The patient was put on rehydration with potassium recharge as well as dual antibiotic therapy before her admission to the operating room for surgical treatment of her evisceration. The evolution was marked by installation on the fourth postoperative day of confusion associated with agitation. Postoperative peritonitis was strongly suspected, but given the atypia of the neurological disorder, a brain CT was performed, returning without abnormalities. An additional MRI was carried out revealing a T2 flair hypersignal and periaqueductal diffusion and mammillary bodies, compatible with Gayet Wernicke syndrome. ***Conclusion*:** Patients with advanced gastric cancer are at risk of malnutrition due to the presence of several risk factors. The association of medical complications can lead to a delay or even a failure of the diagnosis of WE which can contribute to an incomplete cure and/or an increase in mortality in this group of patients.

Keywords: Wernicke's Encephalopathy, Postoperative Complications, Gastric Cancer, Malnutrition, Delayed Diagnosis.

INTRODUCTION

Wernicke's encephalopathy is a neurological syndrome secondary to a thiamine deficiency which is a cofactor for several biochemical reactions in the brain [1].

The main cause is alcohol abuse. Other etiologies reported, are namely: chronic malnutrition, prolonged parenteral nutrition without thiamine substitution especially in cancer patients, prolonged pregnancy vomiting and post-operative neoplastic gastrectomies or in the context of bariatric surgery [2, 3] especially in the presence of other risk factors such as the presence of recurrent vomiting with malnutrition, or systemic involvement such as sepsis with exclusive parenteral nutrition [4].

In the postoperative context, this condition is often underdiagnosed, especially in patients admitted to intensive care given the non-specificity of neurological symptoms which can often have multifactorial origins in this type of patient.

We report the case of a patient with Wernicke syndrome postoperatively after a cancer gastrectomy and whose clinical symptoms were initially confused with postoperative peritonitis.

CASE PRESENTATION

This is a 78-year-old female patient, with no particular history, initially admitted to the gastrology department of our hospital for treatment of late post-prandial vomiting associated with weight loss estimated at 8 kilos in one month.

An esophago-gastro-duodenal fibroscopy was performed and revealed a stenosis and impassable mass at the antro-pyloric level. The patient was placed on parenteral undernutrition and rehydration while awaiting further investigations.

A thoraco-abdominopelvic scan revealed an antro-pyloric tumor wall thickening, at stenosis level, with a maximum thickness of 10 mm, classified T3N1M0.

The pathological examination came back in favor of a moderately differentiated, ulcerated and invasive gastric adenocarcinoma. The indication for oncological gastrectomy was made after multidisciplinary consultation.

The patient underwent total gastrectomy on the 15th day of her hospital admission. The operation took place without incident and the postoperative course was simple. The patient left the hospital on the 6th postoperative day.

On the 15th postoperative day, the patient was admitted to the emergency department of our hospital with a clinical presentation of evisceration. The biological assessment revealed an inflammatory syndrome with profound hypokalemia at 1.8 mEp/l. The patient was put on rehydration with potassium recharge on a central venous line as well as dual antibiotic therapy before her admission to the operating room for surgical treatment of her evisceration.

The evolution was marked by the persistence of hydroelectrolytic disorders despite the permanent correction as well as the progressive installation on the fourth postoperative day of confusion associated with agitation. Postoperative peritonitis was strongly suspected, but given the atypia of the neurological disorder, a brain CT was performed, returning without abnormalities. An additional MRI was carried out revealing a T2 flair hypersignal and periaqueductal diffusion and mammillary bodies, compatible with Gayet Wernicke syndrome (Figure 1 & 2).

The patient was put on injectable thiamine. The evolution was marked by a neurological worsening with repetitive convulsive attacks progressing towards death on the 6th day of appearance of neurological symptoms.

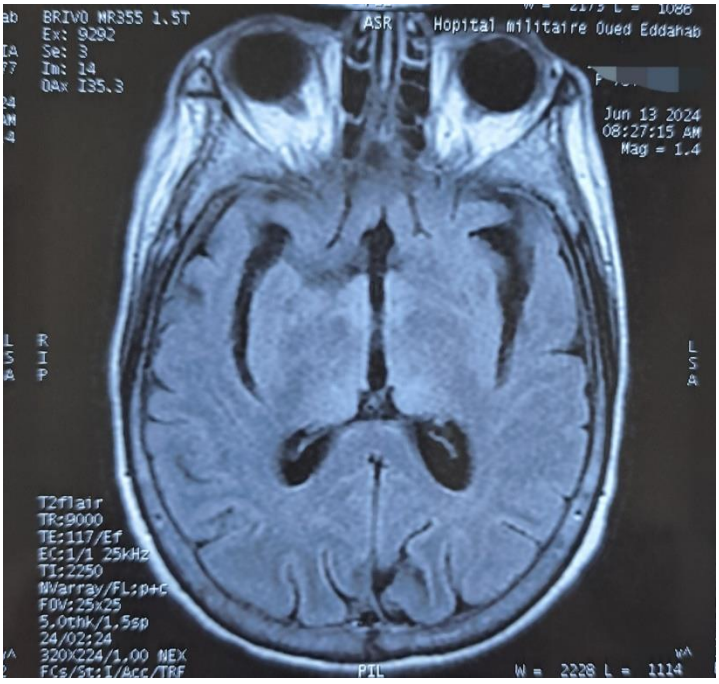

Figure 1: FLAIR axial shows hyperintensity of the periaqueductal grey

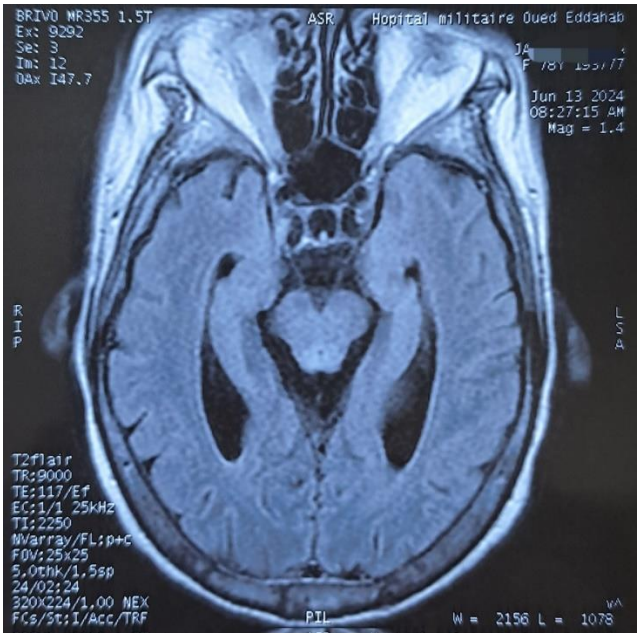

Figure 2: FLAIR axial shows hyperintensity of the periaqueductal grey

DISCUSSION

WE is caused by a deficiency of thiamine (vitamin B1), which is necessary for carbohydrate metabolism. Thiamine is activated into thiamine pyrophosphate which converts pyruvate to acetyl CoA, an essential molecule in the tricarboxylic acid cycle. In WE, thiamine deficiency causes a decrease in thiamine pyrophosphate, which alters carbohydrate metabolism in the brain, causing damage to DNA synthesis [5].

Early diagnosis and treatment of WE improves the prognosis and reduces cerebral aggression [3]. The diagnosis of WE is rather clinical, because it does not cause any abnormalities in the CSF, nor in EEG or during evoked potentials. For this, the best diagnostic method is clinical suspicion in any malnourished patient, or presenting a metabolic problem, or accusing intestinal absorption problems [6]. Diagnostic confirmation is carried out by measuring the blood thiamine concentration, however this measurement is limited because of its technical difficulty [7]. MRI represents another means that can help in the diagnosis. The alteration of the signal in the mammillary bodies and the periquaductal gray matter points towards WE [8].

Patients with advanced gastric cancer have a high risk of chronic malnutrition suggesting a high risk for developing WE [9]. However, the association of gastric cancer with WE is rarely reported in the literature, which may be due to a lack of knowledge of this combined condition.

Diagnosis of WE at an early stage in patients with gastric cancer is difficult. Neurological signs first suggest brain metastasis, metabolic disorder, but these signs might be secondary to hypoxia, hyperuremia or sepsis. It is always difficult to corelate neurological disorders to a relative nutritional deficit especially since the clinical symptoms of WE are non-specific [10].

In addition to the atypical clinical presentation, the diagnosis of WE in the postoperative period of digestive surgery in a patient with gastric cancer further complicates the clinical diagnosis since the neurological signs of WE can simulate postoperative peritonitis. This is especially true since the patient may be tachycardic in the postoperative period given the hypovolemia and pain, as well as the markers of inflammation often being high in the postoperative period. These same postoperative conditions can complicate WE, particularly in the presence of anemia, septic or hemorrhagic shock causing a decrease in cerebral flow [11], which leads us to think of WE in these conditions and in the face of an abdominal CT scan that does not suggest peritonitis.

Treatment of WE must be early because the prognosis depends on rapid treatment. These patients should receive high doses of thiamine three times a day for 2 to 3 days [7]. Patients with risk factors for WE disease should receive special attention with neurological monitoring [12] especially in the absence of consensus for preventive treatment by administration of thiamine in these subjects [9].

CONCLUSION

Patients with advanced gastric cancer are at risk of malnutrition due to the presence of several risk factors: digestive occlusion, anorexia, vomiting and deterioration of the general condition [9]. It is always crucial to consider a WE in these patients when neurological disorders are present.

The association of medical complications whether metabolic, septic or in the context of

postoperative complications related to the underlying gastric neoplastic pathology can lead to a delay or even a failure of the diagnosis of WE which can contribute to an incomplete cure and/or an increase in mortality in this group of patients [11].

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