Govaresh/ Vol. 20, No.1, Spring 2015; 70-2.

Case Report

A 44 Year Old Woman With Acute Pancreatitis And Confusion: Case Report Study

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ABSTRACT

Wernike encephalopathy(WE)caused by thiamine deficiency was a critical but reversible disorder. It can be occurred after acute pancreatitis due to prolonged fasting. WE after acute pancreatitis was rarely suspected and diagnosed at early stages. We reported a 44 year-old woman with severe acute pancreatitis who developed altered mental status and bilateral nystagmus and ophthalmoplegia soon after 14 days of fasting. The brain MRI confirmed the diagnosis. After empiric treatment with thiamine all of the neurological symptoms reversed except only some antegrade and retrograde amnesia . In this case report considering thiamine deficiency in any patients with fasting and neurological symptoms, was addressed since WE was a reversible disorder if it was diagnosed in early stage.

Keywords: Wernike encephalopathy; Acute pancreatitis; Nystagmus; Neurological symptoms

please cite this paper as:
Mousavi-Fatemi KH, Ebrahimi Daryani N, Teimouri A, Alborzi F. A 44 Year Old Woman With Acute Pancreatitis And Confusion: Case Report Study. *Govaresh* 2015;20:70-2.

INTRODUCTION

WE, characterized by ataxia, global confusion, nystagmus, ophthalmoplegia, was a life threatening but preventable disorder(1-4). It was suggested that in any patients with more than 10 days fasting, thiamine replacement should bconsidered(3,5).WE was rarely suspected especially in nonalcoholic conditions such as prolonged IV fasting or malnutrition. It can occur rarely in severe acute pancreatitis. The diagnosis of this disorder in early stage is critical in which it could be reversible (1-5).

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Received: 20 Jan. 2015
Edited: 15 Mar. 2015
Accepted: 16 Mar. 2015

Herein, we present a 44 year-old woman with severe acute pancreatitis and WE after only 14 days of fasting. The purpose of presenting this case was to emphasized on early suspicion to WE in any patients with fasting and neurologic symptoms in clinical practice.

This was the first case of WE followed by acute pancreatitis diagnosed and reported in Iran.

CASE REPORT

A 44 year old woman was admitted to the hospital because of epigasteric and right upper quadrant pain accompanied by fever, chills and icterus .The lab tests revealed elevated aminotransferase and alkaline phosphatase in a cholestatic pattern. Ultrasonography showed dilated common bile duct with an echogen lesion in it resembling of a stone. An endoscopic retrograde cholangiography was performed and the stone was extracted. One day after performing ERCP, she developed severe abdominal pain radiating to back with elevated serum amylase up to 680 mg/dl. She was diagnosed as post ERCP pancreatitis and received

aggressive fluid and remained fast for 7 days because of severe nausea and vomiting which made her intolerant to oral intake. She was discharged from hospital one day after beginning oral feeding, but two days later she came back to the hospital with nausea, vomiting and abdominal pain. The computed scan of abdominal pelvic revealed fluid collection about 126×70 mm in left pararenal area (Figure1). Radiologic drainage was performed. During this admission she could not tolerate oral feeding again and a nasojeujonal tube placed, but it was not efficious.7days later she developed dizziness without obvious neurologic deficit, two days later she suddenly became confused without orientation to time, person, and place. She also had lower extremities paresthesia. On neurologic examination she had bilateral 6 nerve palsy, vertical and horizontal nystagmus. A contrast brain MRI showed hypersignal mammillary body, tegmentum and periaqueductal (Figure 2). According to these findings Wernicke encephalopathy was diagnosed and Intravenous thiamine administered. Soon after that ophthalmoplasia improved and after 48 hours nystagmus disappeared. The confusion and disorientation took 10 days to get better but did not resolve completely. She was discharged with oral thiamine and on follow ups one month later she was completely well except having some antegrde and retrograde amnesia which could be a long sequel of mammillary body involvement.

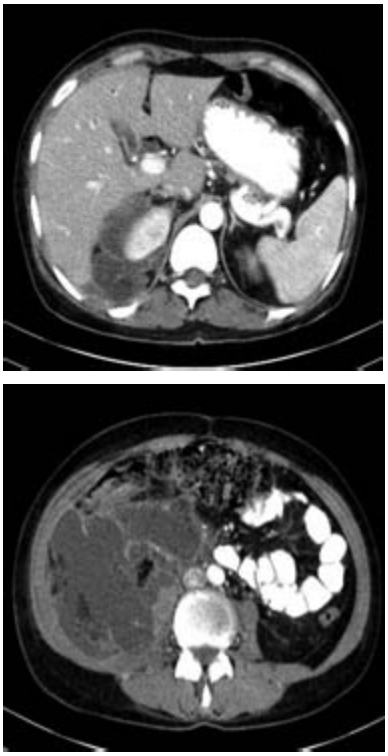

Fig.1: Fluid collection about 126×70 mm in left pararenal area

DISCUSSION

WE is a life threatening condition and an acute syndrome requiring emergent treatment , therefore high degree of suspicious should be held to diagnose it in at risk patients to prevent mortality and morbidity(1,2). Although the most common cause of Wernicke's encephalopathy is chronic alcoholism but other conditions which are associated with this disorder, are prolonged intravenous feeding, hyperemesis of pregnancy, anorexia nervosa, refeeding after starvation, thyrotoxicosis , regional enteritis, malabsorption syndromes, renal dialysis, peritoneal dialysis, uremia, HIV, cancer Transplantation, rarely gastric surgery and other causes of malnutrition(1-4). Chen et al. suggested that in patients with more than 10 days of fasting we should consider thiamine deficiency(3,5). One of the associated conditions with WE is pancreatitis et al followed 12 patient with sever pancreatitis from 1999 to 2006 and reported 9 of them to develop WE(2,4,6) in a survey of 93 patients with severe pancreatitis, Sun GH et al. reported 4(0.7%) patients with WE and 2 of them died because they were misdiagnosed and 2 of them survived with treatment(3)similar to these studies our presented case showed evidence of WE after severe pancreatitis. Hong-wei et al. reported a 47 year old woman who developed WE after 50 days (2).Carlos Rdrigo et al. also reported a17 year old woman with WE 14 days after severe pancreatitis(4). WE Symptoms revealed about 14 days after pancreatitis diagnosed.

In a patient with ophthalmoplegia , ataxia, and global confusion, Wernicke encephalopathy should be considered(1-5). But this classic triad is seen in only one third of the patients(1,3). In 1997 a diagnostic criteria(Caine criteria) was published that requires 2 of the following signs: dietary deficiencies, occulomotor abnormalities, cerebellar dysfunction, and either an altered mental state or mild memory impairment(1-6). Our patient had disorientation to person, place and time with bilateral 6 nerve palsy, vertical nystagmus and dietary deficiency(long fasting with nausea as risk factors). In Fei et al. study that reported 12 patients with WE ,dizziness was the initial symptom in 5 patients(6). The symptoms in our case started with dizziness, like Fei study. The most preferred imaging study is brain MRI with 93% and 53% specificity and sensitivity respectively(1-8). Increased T2 and decreased T1 signal surrounding the aqueduct and third ventricle and within

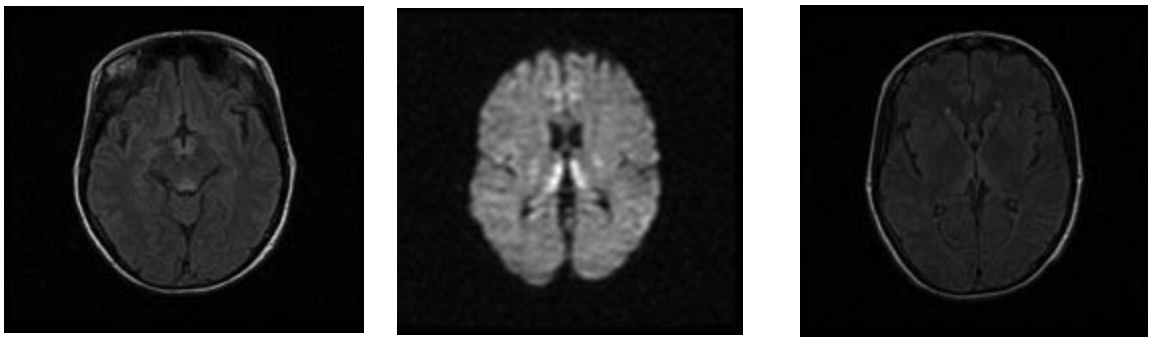

Fig.2: A contrast brain MRI showed hypersignal mammillary body, tegmentum and periaquaduct

the medial thalamus and mammillary bodies is typical finding(2-8). Like other studies, CT-imaging of our patient did not show any abnormality and her MRI helped us to recognize and confirm the disorder. In her MRI abnormal signals in dorsomedial aspects of both thalamus and periaqueductal gray matter and floor of 3rd ventricle and mammillary bodies were seen that with her clinical symptoms(severe pancreatitis with neurologic exams mentioned above) WE was diagnosed. Patients with suspected WE require immediate intravenous thiamine therapy, in combination with other B vitamins (2,3,9). 100 mg of thiamine should be continued after the completion of parenteral treatment and after discharge from the hospital until patients are no longer considered at risk (10,11).

In conclusion, WE is a serious but preventable disorder although it is usually underestimated especially in nonalcoholic patients. The internist should always consider WE as one of the differential diagnosis of pancreatic encephalopathy and it is better to add thiamine in patients with pancreatitis who remain fasting more than 10 days.

ACKNOWLEDGMENT

We would like to thank Dr.Tfakhori A, for helping us in early diagnosis of this disorder.

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