

Wernicke-Korsakoff syndrome in a patient with tuberculous peritonitis

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Abstract. – Tuberculous peritonitis is an uncommon type of extrapulmonary tuberculosis and can be present in different and rare conditions. Wernicke-Korsakoff syndrome is a neuropsychiatric disorder due to thiamine deficiency which is caused by poor nutrition for any reason. The relationship between tuberculous peritonitis and the Wernicke-Korsakoff has not been declared yet. Therefore, we aim to report a case of tuberculous peritonitis which presented with Wernicke-Korsakoff syndrome.

Key Words:

Tuberculous, Peritonitis, Wernicke-Korsakoff.

Introduction

Tuberculous peritonitis is an uncommon type of extrapulmonary tuberculosis. It continues to be a significant health problem especially in developing countries. Symptoms and signs are nonspecific and there is no specific diagnostic test. This leads to delays in diagnosis of the disease. Therefore tuberculous peritonitis may present in different and rare conditions^{1,2}. Wernicke-Korsakoff Syndrome (WKS) is a neuropsychiatric disorder due to thiamine deficiency. The main cause of thiamine deficiency is poor nutrition for any reason. WKS usually presents with ocular disturbances, gait abnormalities and mental status changes^{3,4}. Until today, in literature, the relationship between tuberculous peritonitis and the WKS has not been declared yet. Therefore, we aim to report a case of tuberculous peritonitis which presented with Wernicke-Korsakoff syndrome and review of the literature.

Case

A 26-year old male patient was admitted to the Emergency Department with complaints of low-

grade fever, vomiting, and unconsciousness. So his medical history was taken from his friend. He was of Nigerian origin and was living in Northern Cyprus for 2 years as a student. He had no history of alcohol and smoking use. He had loss of appetite, nausea, weight loss for two months and he had been vomiting for two months and these complaints worsened day after day. Approximately 15 days before admission memory impairment, hearing loss was remarkable and was unable to walk without assistance. But two days before admission constant drowsiness started and they admitted to Emergency Department. On physical examination he was somnolent, uncooperative and disoriented. We also found nystagmus in horizontal axis, hyporeflexia, trunkal ataxia and mild paraparesis. He could not cooperate with mini-mental test. On abdominal examination there was mild tenderness in all quadrants. His blood pressure 110/70 mm/hg, pulse 116 beats per minute, and temperature 37.8°C. Electrocardiogram revealed sinus tachycardia. In his blood test: white blood cell: 8640/uL (3700-10500), hemoglobin: 9.1 gr/dl (10.8-14.2), platelets: 473000/uL (150000-450000), C-reactive protein: 11 mg/dl (0.0-0.5), albumin: 3 g/dl (3.5-5.29), erythrocyte sedimentation rate: 114 mm/h (0-15). Hepatitis B and C, Human immunodeficiency virus, malaria and brucella were all ruled out using the serologic tests. Abdominal ultrasound revealed small amount of ascites and computed tomography revealed peritoneal thickening and irregularity. Brain magnetic resonance imaging (MRI) demonstrated hyperintense signal alterations in dorsomedial thalami, mammillary bodies, around periaqueductal region, around third ventricle and cerebellar vermis (Figure 1). MRI findings were compatible with WKS when evaluated with the patient's clinical findings. Thiamine replacement treatment started urgently and by the twelve hour

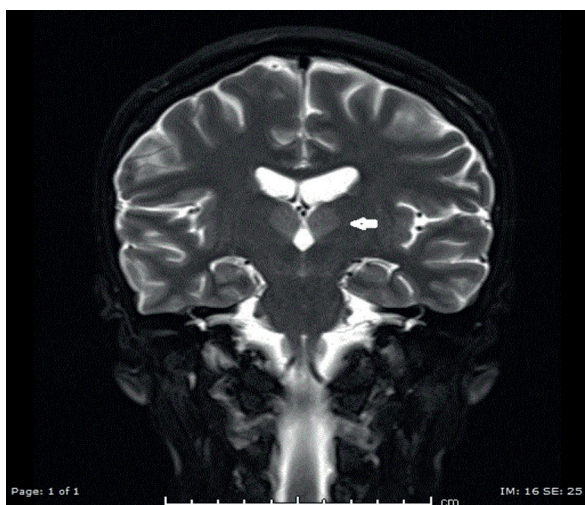


Figure 1. Magnetic resonance coronal T2 section. Bilateral symmetric hyperintense areas are noted around the 3rd ventricle.

of supplementation somnolent state resolved, the patient started playing his smart-phone, and on the 3rd day patient started to walk around.

Ultrasound guided diagnostic abdominal paracentesis was performed and serum-ascites albumin gradient analysis found 0.3 g/dl which was less than 1.1 g/dl. Acid fast stained smear and polymerase chain reaction (PCR) for tuberculosis were found to be negative. Ascited adenosine deaminase (ADA) level was 53 U/L. Ascites white blood cell was 6740/mm³. Sputulum acid fast stained smear was negative and chest computed tomography revealed no signs of tuberculosis. Ultrasound guided tru-cut peritoneum biopsy was performed and the pathology report eventually confirmed the peritonitis with granulomas. Finally, clinical and laboratory findings with pathology report confirmed Tuberculous peritonitis. An anti-tuberculous regimen was commenced along with peroral methylprednisolone 40 mg/day and the patient was discharged one week later with good response. One month later only hearing loss and amnesia did not completely improve, while all the other neurological findings improved. The sedimentation rate was in normal ranges and he gained 5 kg weight.

Discussion

To the best of our knowledge, this is the first case report of tuberculous peritonitis presenting

with WKS. The patient responded well to the treatment and nearly all of the symptoms resolved after the tuberculosis treatment.

WKS is a result of thiamine deficiency, and poor nutrition is the main risk factor. Chronic alcohol abuse, malnutrition, starvation, gastrointestinal tract malignancies, chronic vomiting are the other causes. Clinical features characterized by a triad of mental impairment, ophthalmoplegia and gait ataxia. Nystagmus is also an important ocular sign. Clinical suspicion is the most important point of WKS diagnosis. Patients' history, clinical findings and MRI findings should be considered altogether. Plasma thiamine level is available but not reliable for diagnosis. Typical findings on MRI and high clinical possibility are diagnostic. Even starting the treatment, in a systematic review of WKS, in cancer patients only 36% of WKS patients recovered completely⁵. Our patient was suffering from chronic vomiting and malnutrition which can cause thiamine deficiency. He had classical clinical findings of WKS such as mental impairment, gait ataxia, nystagmus, and MRI also revealed all typical lesions for WKS. He responded to thiamine treatment in a short time and his somnolence improved within the first hours; at the third day of the treatment he was walking around without assistance and memory impairment, nystagmus, hearing loss improved substantially. But in the third and sixth month's follow-up controls partially hearing and memory problems were still observed.

Tuberculous peritonitis is a subacute disease and has an insidious character. It usually presents with nonspecific symptoms. The most common symptoms are abdominal pain, anorexia, weight loss, and fever⁶. Ascites and abdominal tenderness are the most prominent findings. For these reasons diagnosis is often delayed. In laboratory, high sedimentation rate, anemia, high crp, hypoalbuminemia are common but nonspecific findings as we detected in our patient. But ascites analysis provides much more useful findings for diagnosis. High ascitic ADA level and low serum ascitic albumin gradient (< 1.1) have %97 sensitivity and %100 specificity^{7,8}. In our patient ascites ADA level was high (53 U/L) and serum-ascites albumin gradient level was lower than 1.1 (0.3 g/dl). Acid fast stained smear on ascites has low sensitivity and also PCR has low sensitivity in smear negative patients⁹. We also found them negative. In a study Dülger et al¹⁰ average time from onset of complaints to diagnosis was 2 weeks to 8 months and mean time was

2.6 months. In our patient this time was about 2 months. High index of suspicion is necessary for earlier diagnosis. Abdominal computed tomography also provides valuable findings like presence of ascites, peritoneal thickening, omental thickening and intraabdominal lymphadenopathy¹¹. We detected the same tomography findings in our patient.

CT findings, high ascites ADA level and low SAAG level very likely indicated tuberculous peritonitis. But to rule out peritonitis carcinomatosa and to confirm the diagnosis we planned peritoneum biopsy. Biopsy under direct visualization with laparoscopy is the recommended procedure. It is safe with low complication rate and high diagnostic sensitivity and specificity. Ultrasound-guided biopsy of the peritoneum can be considered as safe alternative with low incidence of complications but this technique is less appropriate in the presence of large amount of ascites. Our patient had ascites which could not be detected with physical examination and we performed ultrasound-guided tru-cut peritoneum biopsy with no complication. Pathology report confirmed our diagnosis as tuberculous peritonitis.

The main treatment option for tuberculous peritonitis is administration of antituberculous regimen for six months; 2 months INH, RIF, PZA, and EMB followed by 4 months INH and RIF. But adding steroids to treatment is an enigma. A meta-analysis of Soni et al¹² showed that adjunctive steroids with antituberculous therapy are effective compared to antituberculous therapy alone. This approach in tuberculous peritonitis patients prevents symptomatic stricture and intestinal obstruction. Our discretion was to start methylprednisolone 40 mg/day with anti tuberculous therapy.

Conclusions

Tuberculous peritonitis has an insidious nature with nonspecific symptoms. The most important step in diagnosis is suspecting tuberculosis. Patients presented with ascites, weight loss, fever, abdominal pain and distention should be considered for tuberculous peritonitis. Delays in diagnosis lead to the progression of the disease and rarely may cause neuropsychiatric disorders like WKS with irreversible complications.

Conflict of Interest

The Authors declare that they have no conflict of interests.

Consent to Participate

The patient gave the permission to use his clinical image and data.

References

- 1) Atzori S, Vidili G, Delitala G. Usefulness of ultrasound in the diagnosis of peritoneal tuberculosis. *J Infect Dev Ctries* 2012; 6: 886-890.
- 2) Dogru T, Sonmez A, Tasci I, Yagci G, Mas MR. Perforated tuberculous appendicitis and peritoneal tuberculosis. *East Mediterr Health J* 2008; 14: 742-744.
- 3) Arts NJ, Walvoort SJ, Kessels RP. Korsakoff's syndrome: a critical review. *Neuropsychiatr Dis Treat* 2017; 13: 2875-2890.
- 4) Manzo G, De Gennaro A, Cozzolino A, Serino A, Fenza G, Manto A. MR imaging findings in alcoholic and nonalcoholic acute Wernicke's encephalopathy: a review. *Biomed Res Int* 2014; 2014: 503596.
- 5) Isenberg-Grzeda E, Rahane S, DeRosa AP, Ellis J, Nicolson SE. Wernicke-Korsakoff syndrome in patients with cancer: a systematic review. *Lancet Oncol* 2016; 17: 142-148.
- 6) Suntur BM, Kuşçu F. Pooled analysis of 163 published tuberculous peritonitis cases from Turkey. *Turk J Med Sci* 2018; 48: 311-317.
- 7) Cho JK, Choi YM, Lee SS, Park HK, Cha RR, Kim WS, Kim JJ, Lee JM, Kim HJ, Ha CY, Kim HJ, Kim TH, Jung WT, Lee OJ. Clinical features and outcomes of abdominal tuberculosis in southeastern Korea: 12 years of experience. *BMC Infect Dis* 2018; 18: 699.
- 8) Dwivedi M, Misra SP, Misra V, Kumar R. Value of adenosine deaminase estimation in the diagnosis of tuberculous ascites. *Am J Gastroenterol* 1990; 85: 1123-1125.
- 9) Sanai FM, Bzeizi KI. Systematic review: tuberculous peritonitis presenting features, diagnostic strategies and treatment. *Aliment Pharmacol Ther* 2005; 22: 685-700.
- 10) Dülger AC, Karadaş S, Mete R, Türkdoğan MK, Demirkıran D, Gültepe B. Analysis of cases with tuberculous peritonitis: a single-center experience. *Turk J Gastroenterol*. 2014; 25: 72-78
- 11) Gulati MS, Sarma D, Paul SB. CT appearances in abdominal tuberculosis. A pictorial essay. *Clin Imaging* 1999; 23: 51-59.
- 12) Soni H, Bellam BL, Rao RK, Kumar PM, Mandavdhare HS, Singh H, Dutta U, Sharma. Use of steroids for abdominal tuberculosis: a systematic review and meta-analysis. *Infection* 2019; 47: 387-394.