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CASE REPORT

Delirium Due to Sepsis-associated Encephalopathy Mimicking Alcohol Withdrawal Delirium

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Delirium is caused by an underlying physiological disturbance, and treatment requires identification of the underlying cause. Making this diagnosis requires careful differential diagnosis. However, often the cause of delirium is hard to recognize. The aim of this study was to report a case of sepsis-associated encephalopathy presented as delirium in a patient during alcohol withdrawal. This case study is about a 58-year-old male patient who had a significant history of alcohol dependence and was admitted for abdominal abscess-related sepsis. Delirium was noted during hospitalization.

Key words: Alcohol withdrawal syndrome, delirium, sepsis-associated encephalopathy

INTRODUCTION

Infection can induce encephalopathy even pathogens do not directly enter the brain. Sepsis-associated encephalopathy (SAE) is defined as diffuse cerebral dysfunction caused by the systemic response to the infection without direct brain infection or other types of encephalopathy.[1] Delirium is one of the presentations of SAE, and the other features include agitation, confusion, disorientation, hypersomnolence, stupor, and coma. Patients may developmental dysfunction before sepsis can be detected.[2]

Alcohol withdrawal syndrome (AWS) is a major cause of morbidity and mortality among alcoholics, and alcohol withdrawal delirium (AWD) is the most severe form. The features included delirium and autonomic hyperactivity, perceptual distortions, global confusion.

We present a case with abdominal abscess-related sepsis and alcohol dependence, in which delirium was noted.

CASE REPORT

A 58-year-old man with a history of alcohol dependence was admitted to this hospital because of fever with back pain. He has been drinking for about 40 years with 30 units of alcohol (600 ml of 58% liquor) four times a week. There was no previous withdrawal seizure or delirium. He stopped drinking 3 months before this admission for progressive weakness.

On examination, abdominal fullness, jaundice, and cachexia were noted. The body temperature (BT) was 37.8°C and blood pressure was 105/78 mmHg, the pulse 88 beats/min, and the respiratory rate 16 breaths/min. Glasgow Coma Scale was 14 (four for spontaneous eye opening, four for incomprehensible sounds, and six for response to pain). The blood tests showed leukocytosis (white blood cell: 11600/μl), elevated C-recreative protein (CRP: 10.69 mg/dL), anemia (hemoglobin: 7.9 g/dl), hyperammonemia (289 μg/dL), and hypoalbuminemia (1.7 g/dL). The liver and renal function were normal, as were platelet count, results of coagulation, total bilirubin, creatine kinase, troponin I. Blood ethanol concentration was <5.00 mg/dl. Blood culture revealed no growth. Computed tomography (CT) scan of the brain showed senile cortical atrophy and old lacunar infarctions in the right basal ganglion. Colonoscopy with biopsy showed tubular adenoma at splenic flexure of colon and adenocarcinoma of

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sigmoid colon. He developed acute disturbance in attention, disorganized speech, agitation, and disorientation the 3rd day of admission and therefore psychiatric consultation was requested for evaluation of AWD.

Abdominal CT scan revealed left hepatic artery occlusion, liver cirrhosis, suspect hepatocellular carcinoma (HCC), and diverticulitis at the cecum causing pericolonic abscess at the right lower abdomen. Positron emission tomography for malignancy survey showed 18 F-fluorodeoxyglucose (FDG)-avidity at right lower quadrant (RLQ) to the pelvic cavity which might be a retroperitoneal abscess, and at RLQ which might be tumor lesion or inflammation. There was also liver FDG-avid lesion indicating metastasis or HCC.

Drainage of a retroperitoneal abscess was performed with culture: *Fusobacterium varium* and *Bacteroides ovatus*, and antibiotics were prescribed. Loop ileostomy for obstruction due to abscess was performed.

Finally, delirium due to hyperammonemia, sepsis or AWD was suspected. The ammonia was corrected 4 days after admission, but delirium still noted. Lorazepam was suggested for suspect AWD, but the actual date of prescription delayed to the 8th day of admission, initially 8 mg/day from the 8th day of admission and then tapered to 2 mg/day the 13th day of admission. Autonomic hyperactivity was noted after admission, and his heart rate was up to 114 beats/min the 2nd day of admission, and 104 beats/min the 3rd day of admission. Fever was noted (BT 39.0°C) on the 4th day of admission, and subsided within a day. Leukocytosis and elevated CRP improved 5 days after admission. His consciousness improved 12 days after admission.

DISCUSSION

Alcohol is central nervous system (CNS) depressant and acts by multiple mechanisms. It inhibits α2 receptors and induces excretion of epinephrine, norepinephrine, and their metabolites; it also increases in hydrocortisone and corticotrophin-releasing factor.[3] Alcohol inhibits the excitatory neurotransmitter glutamate by suppression of postsynaptic-methyl-d-aspartate glutamate receptor.[4] Alcohol reinforces the inhibitory neurotransmitter gamma-aminobutyric acid (GABA) at GABA-A receptors.[3] After prolonged intake of alcohol, reductions lead to CNS hyperexcitation such as tachycardia, hypertension, diaphoresis insomnia, anxiety, increased BT, as well as hand tremor. More severe withdrawal syndromes include hallucinations, tonic-clonic seizures. The most severe form of AWS is AWD, characterized by acute onset of mental status change, inattention, disorganized thought, and altered level of consciousness which fluctuates in severity during the day.

AWD usually begins within 3 days after abstinence and usually persists 2 or 3 days, but sometimes it can last from 1 to 8 days or more.[4] The risk factors for AWD include prior episodes of alcohol withdrawal seizures or delirium, advanced age, comorbid conditions, detectable blood alcohol level on admission, high daily intake of alcohol, abnormal liver function, misuse of other depressant agents, and male sex.[3] First line management of AWD is benzodiazepines (BZD) such as lorazepam, diazepam, and chlordiazepoxide.[4] Other medications include phenobarbital, clomethiazole, midazolam, carbamazepine, oxcarbazepine, clonidine, dexmedetomidine, and propofol in BZD resistant case.[3,4]

SAE can sometimes develop in patients with remote infection, even in walled-off abscess in abdomen.[1,5] Therefore, the absence of bacteremia does not exclude SAE. Biliary tract infections and intestinal infections are associated with higher rate of SAE. The main pathogens included *Staphylococcus aureus, Enterococcus faecium, Acinetobacter* spp, *Pseudomonas aeruginosa,* and *Stenotrophomonas maltophilia*.[6] SAE is a diagnosis of exclusion. Mimics include neuroleptic malignant syndrome, malignant hyperthermia, thyroid storm, drug fever, hypothalamic brain stem stroke, heat prostration, cancer, trauma, pancreatitis, seizures, CNS infection, and endocarditis.[7] In this case, the patient was febrile and not sedated; there were no focal signs, no meningeal signs, no trauma, whereas Confusion Assessment Method indicated delirium, therefore SAE was likely.

The management of SAE relies on the determination of the underlying cause and treatment of the infection. Sedatives are often used; dexmedetomidine may be efficacious, but lorazepam should be avoided.[8] This is opposite to the management of AWD. In our case, these two conditions are taken into account, so we prescribed low dose lorazepam and avoided over-sedation, and tapered once withdrawal delirium is excluded from this study.

There are many potential causes that lead to mental dysfunction; among them, infections and alcohol withdrawal are two of the common causes. When treating a patient with both alcohol dependence and sepsis, it is hard to distinguish which is the actual cause of acute conscious disturbance, and this makes the treatment challenging. Therefore, careful history taking and close monitoring are crucial.

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Conflicts of interest

There are no conflicts of interest.

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