A case of an alcohol user complicated with both Wernicke’s encephalopathy and Boerhaave syndrome

Ikuto Takeuchi MD., Hiromichi Ohsaka M.D., PhD., Kei Jitsuiki MD., Toshihiko Yoshizawa MD., Kouhei Ishikawa MD., Kazuhiro Omori MD,PhD., Youichi Yanagawa MD., PhD.
Department of Acute Critical Care Medicine, Shizuoka Hospital, Juntendo University, Shizuoka, Japan

*Corresponding author
Youichi Yanagawa
Email: yyanaga@juntendo.ac.jp

Abstract: A 45-year-old male was found lying unconscious in his room by his uncle. The room was littered with alcohol bottles. Upon arrival, his Glasgow Coma Scale score was 9. He had a blood pressure of 98/72 mmHg and a body temperature of 27.0 °C. The main abnormal results of a biochemical analysis of the blood suggested that he had hepatitis, renal failure, rhabdomyolysis, hyperglycemia, and cardiomyopathy. Based on the findings of aCTand MRI, he was diagnosed with Wernicke’s encephalopathy, Boerhaave syndrome induced by alcohol. After medical treatment and rehabilitation, he was discharged on Day 60 of hospitalization. As alcoholic patients tends to have a variety of underlying diseases, physicians must conduct a whole-body evaluation for alcoholic patients using radiological, physiological, and biochemical analyses if the patient cannot describe their symptoms well due to unconsciousness or dementia.

Keywords: accidental hypothermia, dehydration, hepatitis.

INTRODUCTION
Alcohol abuse is a major problem among hospitalized patients, and alcoholics are predisposed to developing critical illness while also facing increased rates of complications and mortality compared to non-alcoholics.[1] Alcohol abuse causes diverse neurologic and gastro-enteric conditions, including Wernicke’s encephalopathy and Boerhaave syndrome. We herein report the first case of combination of these diseases in English literature.

CASE REPORT
A 45-year-old male was found lying unconscious in his room by his uncle. The room was littered with alcohol bottles. The uncle grossly confirmed that he had been conscious at least one week prior to this incident. When emergency medical technicians checked him, he was comatose and was transported to our hospital. His medical and family history were unremarkable. Regarding his employment, he ran a bar. Upon arrival, his Glasgow Coma Scale score was 9, and he had isocoric reactive pupils. He had a blood pressure of 98/72 mmHg, a heart rate of 68 beats per minute (BPM), a respiratory rate of 40 BPM, an SpO2 of 100% in room air, and a body temperature of 27.0 °C. As the physiological findings, he had bedsores on his right chest and right extremities and a weak motor response to pain stimulation. His chest roentgen was negative, and electrocardiogram showed depression of the ST segment at II, III, aVF, V, and V6 with premature ventricular contractions. The main abnormal results of a biochemical analysis of the blood were as follows: pH level of 7.28, lactate level of 9.4 mmol/L, white blood cell count of 15,000/μl, hemoglobin level of 16.6 g/dl, platelet count of 8.9 × 109/μl, total bilirubin level of 3.6 mg/dl, aspartate aminotransferase level of 91 IU/L, alanine aminotransferase level of 61 IU/L, glucose level of 288 mg/dl, blood urea nitrogen level of 84.6 mg/dl, creatinine level of 2.21 mg/dl, creatinine phosphokinase level of 4211 IU/l and activated partial thromboplastin time of 58.5 (27.5) seconds. A pan scan using computed tomography (CT) for evaluation of unconsciousness and shock state was negative for the brain but showed pneumomediastinum around the esophagus without abscess (Figure 1)[2,3] Urgent head magnetic resonance imaging (MRI) using fluid-attenuated inversion recovery revealed high intensity at the medial thalamus and around the third ventricle and periaqueductal area (Figure 2). Given these findings, he was diagnosed with Wernicke’s encephalopathy, Boerhaave syndrome, accidental hypothermia, dehydration, hepatitis, renal failure, rhabdomyolysis, hyperglycemia, and cardiomyopathy induced by alcohol. His Acute Physiology and Chronic Health Evaluation II score was 30 points, and the approximate mortality rate was 73%. As the CT lacked signs of mediastinitis around the esophagus, he received only thiamine, antibiotics, and a proton pump inhibitor without surgery. He temporarily showed delirium and convulsion, which were treated by medication. After these treatments, he regained consciousness but experienced right peroneal paralysis due to compression from a prolonged period of unconsciousness at his hospitalization.
Ikuto Takeuchi et al.; Sch J Med Case Rep, Aug 2016; 4(8):620-622

house. His thiamine level on arrival was found to be 11.1 (normal range: 24-66) ng/mL, which was compatible with a diagnosis of Wernicke’s encephalopathy, and endoscopy on Day 10 of hospitalization revealed healing of an esophageal lesion just above the cardia. After medical treatment and rehabilitation, he was discharged on Day 60 of hospitalization.

Fig-1. Truncal CT on arrival. CT shows pneumomediastinum around the esophagus without abscess.

Fig-2: Magnetic resonance imaging (MRI) using fluid-attenuated inversion recovery on arrival. The MRI shows high intensity at the medial thalamus and around the third ventricle and periaqueductal area.

DISCUSSION
To our knowledge, this is the first case of an alcohol user being complicated with both Wernicke’s encephalopathy and Boerhaave syndrome. Given the patient’s chronic alcoholism, his unconscious state on arrival, the MRI findings, and the low level of blood thiamine led to a diagnosis of Wernicke’s encephalopathy.[4,5] As CT demonstrated no signs of mediastinitis, suggesting a short duration from esophageal perforation to arrival at the hospital, we were able to exclude a vitamin B1 deficiency induced...
by persistent vomiting for a long time as the cause of his Wernicke’s encephalopathy.[6]

Vomiting induced by alcohol has been reported to be a risk factor for Mallory-Weiss tears in the mucus membrane of the lower part of the esophagus.[7] While there are few reports of Boerhaave syndrome induced by alcohol, if such a tear were to deepen, perforation of the esophagus could occur, resulting in Boerhaave syndrome.[8,9] Delayed diagnosis of Boerhaave syndrome results in a poor prognosis.[10] Fortunately, an aggressive whole-body evaluation using CT and biochemical analysis led to early recognition of this disease in the present case, allowing our team to select the appropriate treatments to ensure a favorable outcome.

The Department of Alcohol-related Disease Impact at the Centers for Disease Control and Prevention has defined over 50 alcohol-related diseases[11]. As alcoholic patients tends to have a variety of underlying diseases, physicians must conduct a whole-body evaluation for alcoholic patients using radiological, physiological, and biochemical analyses if the patient cannot describe their symptoms well due to unconsciousness or dementia.

CONCLUSION

As alcoholic patients tends to have a variety of underlying diseases, physicians must conduct a whole-body evaluation for alcoholic patients using radiological, physiological, and biochemical analyses if the patient cannot describe their symptoms well due to unconsciousness or dementia.

Conflict of interest: We do not have conflict of interest to declare.

Acknowledgements

This manuscript obtains financial support form Ministry of Education, Culture, Sports, Science and Technology (MEXT)-Supported Program for the Strategic Research Foundation at Private Universities, 2013-2019 concerning [The constitution of total researching system for comprehensive disaster, medical management, corresponding to wide-scale disaster].

REFERENCE


Available Online: http://saspjournals.com/sjmcr