

Alcohol-Induced Psychotic Disorder with Suspected Pulmonary Embolism: A Case Report

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Case Introduction

The patient, a 51-year-old male, was admitted to the Department of Psychology of our hospital in 2020-10-05 because of “poor physique, mental abnormality and aggravation for 4 days”. Patients began to drink alcohol 20 years ago, drinking liquor 0.25Kg per day, there is morning drinking, physical condition has been OK. Before one month, poor appetite and food intake, weight loss, weight loss of more than 20 Kg, walking easy to fall, weak legs, has not been treated. Half a month ago, the patient stopped drinking, intermittent symptoms such as lethargy, hand scratching and nonsense, unstable walking, aggravated symptoms on October 1, and incontinence in the evening. On October 2, the family took the patient to the second Hospital of Mudanjiang for medical treatment. The wardrobe CT, DWI, biochemistry, blood routine and other related tests suggested multiple cavity infarction, due to the patient’s restlessness, head DWI was affected, not hospitalized and other special treatment. The patient has been in a bad state, restless, and does not recognize people. yesterday, the patient was accompanied by his wife to the brain hospital, wardrobe, lung CT, blood coagulation and other blood-related tests, suggesting multiple cerebral infarction, considering phlegm thrombus, it is recommended to come to our hospital, so the patient comes down to our hospital in a sedative state, and the outpatient is admitted to the hospital with “mental disorders to be examined”. The flat cart was pushed into the ward. The admission temperature of the patient was 36°C, pulse: 116bpm, respiration: 24bpm, blood pressure: 114/82mmHg. General poor state, unconscious, soft neck, no jugular vein, thoracic symmetry, abnormal breathing, auscultation and wet rales can be heard in both lungs. Heart rate 116 beats / min, regular rhythm, no pathological murmur in each valve auscultation area, no friction sound of pericardium, untouched liver and spleen, lower abdominal eminence, bladder filling, no edema of both lower limbs, nervous system: unclear consciousness, currently in a state of sedation, unable to cooperate with nervous system examination, no pathological signs were identified. Give first-class nursing; ECG, blood pressure, blood oxygen saturation monitoring, central oxygen inhalation, establishment of venous access, urgent consultation in respiratory department and psychiatric department. Suggestion 1. Blood gas analysis, lung CT, ECG, procalcitonin and D-dimer were examined. 2. Anti-infective, antiasth-

matic, expectorant treatment. 3. If necessary, transfer to ICU treatment. 1. The disease allows reexamination of head CT and head DWI two. It is suggested that the intensive care department should consult. Emergency blood gas analysis indicates that pH:7.080, PO₂:73.6mmHg, PCO₂:76.0mmHg, urgently asks ICU, for treatment: the patient is in critical condition and should be transferred to intensive care department for emergency treatment; strengthen respiratory tract management and keep respiratory tract unobstructed. Other examination returns: blood coagulation routine report: d-dimer test 33.24 μg / ml, high, fibrin (original) degradation products 107.80 μg / ml, high, myocardial zymogram: *aspartate aminotransferase 74U shock L, high, (dangerous) *creatinine kinase 16000.0Umax L, high, *lactate dehydrogenase 823Umax L, high, myoglobin 230.7ngmax ml, on the high side. The results showed that D-dimer was high, fibrin degradation products 107.80 μg / ml, myocardial zymogram: *aspartate aminotransferase 74U shock L, on the high side, *creatinine kinase 16000.0Umax, on the high side. Biochemical: *Urea 11.33mmol hammer L, high, *glucose 6.73mmol hammer L, high, *aspartate aminotransferase 77Umax L, high, glutamate grass / glutamic pyruvic 1.93, high, total bilirubin 22.5 μmol / L, high. Communicate the patient’s current examination results with respiratory and renal physicians. If you need to further clarify the diagnosis, you need to undergo CT, pulmonary angiography and other large-scale examination, but now the patient’s general condition is very poor, he is in a coma, breathing is abnormal and difficult to breathe, and his eyes are slow to light reflex under oxygen inhalation. Inform the patient’s family members of the seriousness of their condition and may be in danger of life at any time. The communication with the family members is transferred to ICU treatment, and the family members refuse to do so. It means bear the consequences, the patient’s general condition is poor, acidosis, 19:15 oxygen inhalation is adjusted to low flow oxygen inhalation, and then communicate with the family members and transferred to ICU for emergency treatment, but the patient’s family members refuse to further aggravate the patient’s condition at 21:43, abnormal breathing, blood oxygen 72%, blood pressure 81mmHg, heart rate 102bpm, adjust high flow oxygen inhalation, pay close attention to the patient’s vital signs. Blood gas analysis at 22:05 showed that the patient with pH:7.204, pCO₂:51.5mmHg, pO₂:48.2mmHg, was critically ill at 22:10. Shenmai injection was

given 50ml intravenous injection immediately. At 22:15, the patient's blood pressure continued to drop, and Shenmai injection was given 100ml intravenous injection. At 22:24, the patient was in a poor state of consciousness and had no spontaneous breathing, so he was given continuous cardiopulmonary resuscitation with 0.375g nikethamide, 3mg injection, epinephrine hydrochloride injection 1mg and atropine 0.5mg. 22:25, no improvement, no spontaneous breathing, adrenaline 1mg intravenous push, atropine 0.5mg intravenous push; 22:29, nikethamide 0.375g intravenous push, epinephrine hydrochloride injection 1mg intravenous push; 22:32, no improvement in symptoms, continue to give atropine 0.5mg intravenous push; epinephrine 1mg intravenous push. At 22:25, the patient's symptoms did not improve, continue to give atropine 0.5mg intravenous push; epinephrine 1mg intravenous push; 22:37 electrocardiogram for ventricular spontaneous rhythm, family members gave up rescue and stopped rescue. At 22:46, the carotid pulse disappeared, bilateral pupils dilated, direct and indirect light reflex disappeared, and there was no spontaneous heartbeat and breathing. The ECG showed a straight line. The clinical death was declared, and the family refused the autopsy.

Discussion

The clinical manifestations of mental disorders caused by alcohol are based on alcohol dependence, often drinking too much or relying on for too long, so that patients are often in mental and psychological cognition such as hallucinations, delusions, thinking disorders or irritability. Alcohol-induced mental disorders often lead to a series of dangerous behaviors, such as violence, aggressive behavior and other self-injury and self-harm behaviors, which pose a great threat to the family and society [1-2]. The patient drank alcohol for 20 years, drank 0.25Kg of liquor per day, stopped drinking half a month ago, and showed obvious withdrawal symptoms, such as intermittent drowsiness, hand scratching, nonsense, unstable walking, and so on. Family members failed to seek medical treatment in time for withdrawal reaction, resulting in life-threatening symptoms. In the relationship between alcohol and thrombus, data show that because alcohol has the ability to change liver function, it directly damages the synthesis of many coagulants and anticoagulants. Drinking alcohol can reduce plasma fibrinogen, thus protecting the body from venous thromboembolism. At the same time, the increase of plasminogen activator inhibitor and factor VIII seems to increase the tendency of blood coagulation [3]. And many studies have shown that the incidence of venous thromboembolism has a strong positive correlation with age [4]. These clinical data clearly show that the middle-aged and elderly patients with mental disorders caused by alcohol are likely to have coagulation dysfunction and cause thromboembolism. The patient had dyspnea on admission, the return of routine blood coagulation test, high D-dimer and high fibrinogen degradation products, which suggested that there may be abnormalities. The above examination indexes have high sensitivity to thrombosis, but poor specificity. It is of little significance to the diagnosis of thrombosis and cannot be diagnosed. A few hours later, the patient developed obvious hypoxemia and hypotension, and high flow oxygen inhalation had no effect. Combined with the above signs and preliminary examination results, combined with the above literature, it is highly suspected to be acute pulmonary embolism. Acute pulmonary embolism (APE) is the most serious clinical manifestation of venous thromboembolism (VTE). It is the third largest acute

cardiovascular syndrome in the world after myocardial infarction and stroke. Acute pulmonary embolism is the most serious clinical manifestation of venous thromboembolism. Most of acute pulmonary embolism may be asymptomatic or accidentally found, or even direct sudden death, so the accuracy of epidemiological data of acute pulmonary embolism is not high. However, acute pulmonary embolism is the main cause of death, medical treatment and hospitalization [5, 6]. Finally, it is concluded that this death has the following characteristics: 1. Middle-aged and elderly men, past history of hypertension, intermittent oral antihypertensive drugs, specific unknown, around August 2018 cerebral hemorrhage, in our department treatment, after poor physique, walking instability, has been unable to work two. The patient drank alcohol all the year round, had a poor physical condition, and stopped drinking for half a month. 3. Poor physique, mental abnormality for half a month, aggravated for 4 days. 4. If you don't have a clear consciousness, you shouldn't call it. In the differential diagnosis, first of all, from the aspect of organic mental disorders, the patient's physique is poor, although he has a history of drinking, but the patient's alcohol consumption is less than 0.25Kg of liquor / day, has stopped drinking for half a month, and the current state is unclear, should not be called, abnormal breathing, dyspnea, mostly considered to be caused by somatic diseases, but the patient's current physical condition is extremely poor, but it is not suitable for large-scale machine examination immediately, which cannot be ruled out for the time being. Secondly, from the perspective of mental disorders caused by psychoactive substances, patients have a history of drinking, and alcohol cannot be ruled out. The cause of death in this case was discussed, including pulmonary embolism, respiratory failure, ion disorder, acid-base imbalance, shock and decrease of effective circulatory blood volume. Death diagnosis: pulmonary embolism? Through this case, psychiatric clinical workers are required to pay special attention to middle-aged and elderly patients who are suspected of having mental symptoms that lead to confusion and complications caused by alcohol-related substance dependence, attach great importance to abnormal blood coagulation, and need to urgently eliminate life-threatening thromboembolism to provide valuable time for follow-up treatment of patients.

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