Delirium Tremens and Cardiovascular Collapse: A Case Report

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Abstract

Acute alcohol withdrawal syndrome leads to fluctuating changes in mental status. It also may cause cardiovascular collapse and death occasionally. We report a patient who dies due to cardiovascular complications of delirium tremens. Early clinical interventions such as hydration, sedation, preventing seizures by using benzodiazepines [1], infusion of dextrose and thiamine can be lifesaver. Hospitalization to Intensive Care Unit (ICU) must be provided immediately. Death is sometimes inevitable even if physicians perform an entire multidisciplinary clinical intervention. In this case, the situation of the patient, who suffers from alcohol withdrawal, worsens rapidly and he dies in two days during the follow-up in ICU.

Introduction

Delirium Tremens (DT), which contains severe mental and nervous system changes, is a clinical manifestation associated with withdrawal from alcohol. Occurrence of symptoms after stopping of alcohol is diagnostic criteria for DT. It occurs acutely. DT follows sudden alcohol withdrawal after prolonged intake of it. It is possible to be precipitated by external factors like infections, injury etc. Symptoms usually begin a few hours after cessation of alcohol intake and peak at 1 to 3 days, but they may last up to 1 week [2]. Major symptoms are tremors, hallucinations and seizures. Seizures related to alcohol withdrawal are more frequent in patients who had any episode of alcohol withdrawal syndrome before [3] Acoustic and verbal hallucinations, delusions and mood disturbances can also occur [4]. Other symptoms are malaise, weakness, nausea, vomiting, anxiety and fear. We aimed to emphasize serious cardiovascular complications of DT in addition to its severe mental status changes.

Case Report

A 43-year-old male patient was brought to our emergency service because of intractable seizures. The patient had no current medical history of epilepsy and had no any medication. He had a history of approximately twenty-year alcohol use and he had already stopped drinking alcohol. The patient was monitored and airway was ensured. Vital signs were noted as follows; blood pressure: 80/40 mmHg, body temperature: 37.5 °C, respiratory rate: 20/min, heart rate: 177/min, blood glucose: 144 mg /dl and oxygen saturation: 95%. Glasgow Coma Scale was counted as 10 (E3V2M5) and so the patient did not need to be intubated. We recognized agitation, autonomic hyperactivity and non-generalized convulsions. There was no focal abnormality or papilledema. Pupils were isochoric. Pathological Babinski was noted. Meningeal irritation signs were negative. The patient was hydrated for fluid loss and hypoglycemia. Diazepam was administered to reduce agitation and to promote sleep. 100 mg of thiamine was given as Intravenous (IV) in order to prevent Wernicke–Korsakoff Syndrome. 1000 mg of Phenytoin was started as IV infusion to stop ongoing seizures. Dextrose 5% was administered as IV infusion to prevent hypoglycemia. Vital signs were assessed every 30 minutes and the patient was observed for hypoglycemia, seizures and agitation. Abnormal laboratory findings were like that: APTT: 155 second, AST: 415 U/L, ALP: 388 U/L, GGT: 1015 U/L, LDH: 642 U/L, Ca: 7.6 mg/dL and WBC: 12.000 K/uL, PLT: 119 K/uL, MCV: 103 fL. Ethanol level was measured as normal (0.59 mg/dL). Brain Computerized Tomography (CT) was assessed as normal (Figure 1). Furthermore, hepatobiliary ultrasound was requested to clarify the abnormal levels of liver function tests. Ultrasound revealed grade 2 hepatosteatosis and excluded cholecystitis. The patient was firstly consulted to Neurology specialist in terms of intractable seizures. Diazepam was administered to reduce agitation and to promote sleep. 100 mg of thiamine was given as Intravenous (IV) in order to prevent Wernicke–Korsakoff Syndrome. 1000 mg of Phenytoin was started as IV infusion to stop ongoing seizures. Dextrose 5% was administered as IV infusion to prevent hypoglycemia. Vital signs were assessed every 30 minutes and the patient was observed for hypoglycemia, seizures and agitation. Abnormal laboratory findings were like that: APTT: 155 second, AST: 415 U/L, ALP: 388 U/L, GGT: 1015 U/L, LDH: 642 U/L, Ca: 7.6 mg/dL and WBC: 12.000 K/uL, PLT: 119 K/uL, MCV: 103 fL. Ethanol level was measured as normal (0.59 mg/dL). Brain Computerized Tomography (CT) was assessed as normal (Figure 1). Furthermore, hepatobiliary ultrasound was requested to clarify the abnormal levels of liver function tests. Ultrasound revealed grade 2 hepatosteatosis and excluded cholecystitis. The patient was firstly consulted to Neurology specialist in terms of intractable seizures. Then, consultations were requested from departments of Psychiatry, Gastroenterology and Anesthesiology, respectively. Psychiatry and Neurology specialists thought that convulsions were related to DT. The Gastroenterologist expressed that there was no need to any urgent intervention for abnormal liver function tests. Hospitalization was recommended by all consultant physicians. Before admission to ICU, Cardiopulmonary Resuscitation (CPR) was administered to the patient because of sudden cardiac arrest and he responded in 7 minutes after CPR. Then, he was admitted to ICU. The patient...
we tried to treat it according to recent guidelines. Our patient also had intractable seizures and was mentioned above.

Related to reduction of GABA-A after withdrawal from alcohol as it is often necessary, but the reasons for hospitalization are not directly related to alcohol abuse. Furthermore, detailed history for alcoholism is compulsory to identify mortality rate [6]. Ferguson, et al. performed a retrospective study by using patients, who were admitted to a hospital in a city of USA. 8% of those patients had died from complications of DT. It has been suggested that to have bad socio-economic conditions and to present severe symptoms of organic diseases related to alcoholism might facilitate death from DT [6]. In our study, the patient suffering from alcohol withdrawal had a bad socioeconomic status and that was a risk factor for mortality, because he had been brought to the emergency service just after intractable seizure not earlier. However, hospitalization wasn’t provided instantly due to bed shortage in ICU and so, we kept our patient in resuscitation room until admission to ICU.

Burin, et al. suggests that it has high possibility to see hypokalemia in patients diagnosed as having DT, because diarrhea and vomiting are often seen in these patients. They have seen also low plasma potassium levels in their own case and they have thought that even though vomiting and diarrhea were mild, low plasma potassium levels might be detected [7]. On the other hand, we didn’t determine hypokalemia in the first day and we found that our patient had mild hyperkalemia after kidney functions began to be impaired. We gave treatment for abnormal plasma potassium levels according to actual guidelines.

Mattoo, et al. performed a study as case report. They suggested that patients suffering from DT had to be managed in a general ward of a de-addiction unit instead of ICU as their case was managed successfully like that [8]. It was no possible for our own case to follow in a de-addiction unit because of serious cardiovascular instability. Therefore, our case was observed and monitored in resuscitation room and later he was admitted to ICU as quickly as possible.

DT is a medical emergency with considerably high mortality in case of cardiovascular complications. Approximately two million people suffer from alcohol withdrawal syndrome each year [9]. Nosocomial pneumonia, metabolic acidosis and other problems, which are born of sedative drugs, are some frequent reasons of death. Burin, et al. suggests that it has high possibility to see hypokalemia in patients diagnosed as having DT, because diarrhea and vomiting are often seen in these patients. They have seen also low plasma potassium levels in their own case and they have thought that even though vomiting and diarrhea were mild, low plasma potassium levels might be detected [7]. On the other hand, we didn’t determine hypokalemia in the first day and we found that our patient had mild hyperkalemia after kidney functions began to be impaired. We gave treatment for abnormal plasma potassium levels according to actual guidelines.

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DT is a toxic state that occurs withdrawal from alcohol after chronic intake. DT occurs due to disruption of alcohol after prolonged intake. DT is manifested by global confusion and sympathetic hyperactivity. Symptoms usually begin a few hours after disruption of alcohol intake and peak at 1 to 3 hours but may last up to 1 week [2]. Chronic intake of alcohol increases inhibitor neurotransmitters like GABA-A during withdrawal from alcohol GABA-A begins to decrease and this induces excessive nervous system excitability mental status change is the result of DT. Tremor, seizures, hallucinations, agitation and autonomic hyperactivity like tachycardia and diaphoresis are frequently seen symptoms and all these are caused by overstimulation of nervous system [5]. DT can easily progress to cardiovascular collapse and mortality rate is high in such a case. Early recognition and treatment is essential and this contributes low rate of mortality related to cardiovascular complications [5]. We observed that our patient was also tachycardic and hypotensive. We thought that it was related to reduction of GABA-A after withdrawal from alcohol as it was mentioned above.

In a study, it is suggested that the most important interventions are observation and continuous nursing because of increased risk for intractable seizure [6]. Our patient also had intractable seizures and we tried to treat it according to recent guidelines.

In another study, SR Mehta, et al. suggests that hospitalization experienced a large number of metabolic disorders during follow-up in ICU. Arterial blood gases values revealed metabolic acidosis and that was resistant to the treatment. Life-threatening arrhythmias like ventricular tachycardia were also determined in the first day. Kidney and liver function tests were found as elevated in the morning of the second day in ICU. Electrolyte imbalance was another problem. Despite positive inotropic agents, hypotension remained as it had already existed in the emergency unit. After all these problems, cardiovascular collapse occurred and the patient didn’t respond to CPR administered for 45 minutes. As a result, the patient died due to cardiovascular complications of DT although we tried to do our best.

Discussion

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Kim et al. suggested that low platelet count was a potential clinical predictor for occurrence of DT in patients with alcohol withdrawal seizures [11]. In our study, we detected mild thrombocytopenia with our case.

Hospitalization is usually needed because both requirement of detailed history for alcoholism and the high risk of cardiovascular instability. As the studies mentioned above also support, DT is not only about nervous system changes. Even though intractable seizure is premier manifestation, it must not be forgotten that mortality due to cardiovascular changes is no negligible. This is what we particularly try to emphasize in this case report.

Conclusion

In this case report, it seems obviously that DT has a high risk of mortality despite required medical interventions, because serious cardiovascular instability is no uncommon for DT. Just because of
this, early recognition and treatment is momentous. Hospitalization to ICU must be done quickly. Long waiting in emergency service may lead to delay for detailed investigation and treatment. As a result, we suggest that physicians must be aware of cardiovascular complication associated with DT.

References