ASIA PACIFIC JOURNAL of MEDICAL TOXICOLOGY
APJMT 11;3 http://apjmt.mums.ac.ir September 2022

CASE REPORT

A Rare Cause of St-Segment Elevation: Ethanol Intoxication

ESMA BUSRA GUZES[1], SUMEYYE ALKAN[1], EMEL ALTINTAS[1,*]

[1]Ankara Training and Research Hospital, Department of Emergency Medicine Altındag, Ankara

Abstract

Introduction: The cardiovascular effects of alcohol consumption are variable. In addition to its protective effects, it may also result in mortality. In this study, we presented a case, who developed cardiac arrest after excessive ethanol intake.
Case Report: A 68-year-old patient, who was brought to the emergency room due to out-of-hospital cardiac arrest, showed ST elevation in the electrocardiogram after returning to spontaneous circulation, and no vascular occlusion was detected in the coronary angiography. The patient's blood ethanol level was found to be 605 mg/dl and he had cardiac arrest again in the follow-up. He did not respond to cardiopulmonary resuscitation (CPR) for 30 minutes and died.
Discussion: Acute alcohol intoxication causes various metabolic changes, cardiovascular side effects, gastrointestinal side effects, and respiratory depression. It has been stated that coronary vasospasm can occur even when ethanol levels reach a basal level after 9 hours of excessive alcohol intake. We believe that ST-segment elevation developed as a result of vasospasm and respiratory depression emerged after ethanol intake and that the patient had a cardiac arrest.
Conclusion: We contend that ST-segment elevation after spontaneous return to circulation in out-of-hospital cardiac arrest patients may be caused by excessive ethanol intake, besides acute coronary syndromes.

Keywords: ST elevation, Ethanol intoxication, Death

How to cite this article: Guzes EB, Alkan S, Altintas E. A Rare Cause of St-Segment Elevation: Ethanol Intoxication. ***Asia Pac J Med Toxicol 2022; 11(3):121-123.***

INTRODUCTION

Alcohol consumption is responsible for 3.8% of mortality and 4.6% of morbidity worldwide [1]. Although it is known that moderate alcohol use has a protective effect on cardiovascular diseases, there is an increased risk of myocardial infarction in the 24 hours following a heavy drinking period, especially in elderly patients [2]. In this research, we aimed to present a rare case that developed cardiac arrest after high-dose ethanol intake in which we detected ST-segment elevation after spontaneous return to circulation and resulted in death.

CASE REPORT

A 68-year-old male patient developed an out-of-hospital witnessed cardiac arrest, and cardiopulmonary resuscitation protocol was started by emergency medical technicians. The patient's blood sugar was 189 mg/dl. He was resuscitated by the guideline protocols and was brought in approximately 20 minutes after the development of cardiac arrest. Throughout the cardiopulmonary resuscitation process, asystole rhythm was observed in the patient, and a pulse was detected 6 minutes after his arrival. The patient, who returned to spontaneous circulation, had no known additional disease and had a history of chronic alcohol use. Arterial blood pressure was 110/60 mm/Hg, heart rate was 90/min, oxygen saturation was 95%, and body temperature was 36°C. Glasgow Coma Score was E1M1V1 (intubated). Electrocardiogram revealed 1 mm ST-segment elevation in leads D2, D3, and AvF, and ST-segment depression in lead AvL. (Figure 1)

In the laboratory, WBC: 19.000/mcgL (4-10.5) Hgb: 14.7 g/dl (13.5-18) platelet 202 10^3 u/L (150-450), glucose: 201 mg/dl (75-99) urea: 35 mg /dl (18-55) creatinine: 1.04 mg/dl (<1.20) alanine aminotransferase 24 U/L (0-41 U/L) , aspartate aminotransferase 33 U/L (0-37 U/L), Sodium: 148 mmol/L(136-145), Potassium: 5.1 mmol/L (3.5-5.5) hs-Troponin T: 13.7 ng/L (<14) CKMB: 6.42 mcg/L (<14) ethanol: 605 mg/dl In blood gas: pH:7.00 pCO2: 62.3 mmHg (35-48) PO2:80 mmHg HCO3: 14.6 mmol/L (24-28) lactate: 10.6 mmol/L (0.9-1.7). Cranial and thoraco-abdominal CT angiography images of the patient were evaluated as normal. In echocardiography, EF was measured to be normal and there was no wall motion defect. Coronary anjiography showed coronary arteries with plaques with no significant stenosis.

Due to the presence of metabolic acidosis, the patient was accepted as cardiovascular unstable and 1 mEq/kg/h NaHCO3 infusion was started. The patient's blood pressure was 60/40 mmHg and noradrenaline infusion was started. Cardiac arrest developed in the follow-up of the patient, who did not respond to cardiopulmonary resuscitation for approximately thirty minutes, was accepted as exitus approximately 3 hours after his admission.

*Correspondence to: Emel Altıntas, MD, Emergency Medicine Specialist, Ankara Training and Research Hospital, Department of Emergency Medicine, Altındag, 06230 Ankara, Turkey.
E-mail: emelaltintas61@gmail.com, Tel: 00-90-554-5982714

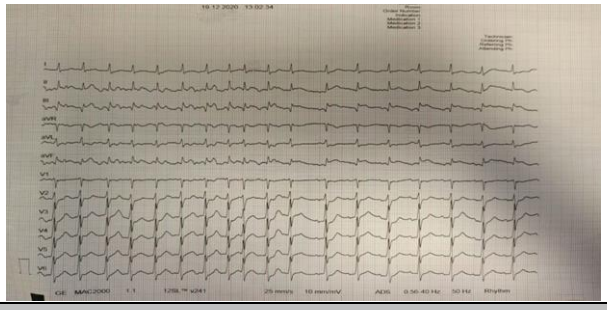

Figure 1. ST segment elevation in leads D2, D3, and AVF, ST segment depression in lead AVL.

DISCUSSION

The annual incidence of Out-of-Hospital Cardiac Arrest (OHCA) in Europe is 67-170 /100.000. The average rate of hospital discharge is 8% (0-18) [3]. Acute coronary syndromes are responsible for 60-80% of OHCA causes. Approximately 5-10% of them are electrolyte disorders, metabolic disorders, drug intoxications, and non-cardiac causes [4]. Emergency coronary angiography is recommended if the patient's electrocardiogram after OHCA has ST-segment elevation and cardiac origin was suspected or in high-probability acute cardiac occlusions without ST-segment elevation. [5] In a study, it was reported that ST-segment elevation after the return of spontaneous circulation (ROSC) in OHCAs was 95% specific for a significant coronary lesion [6]. A low perfusion index was found in patients with false positivity with STEMI after ROSC [7]. It has been stated that patients with age>85, non-VF initial rhythm, unwitnessed arrest, ROSC after 30 minutes, end-stage renal failure, cancer disease, pH<7.2, lactate>7 in patients with ST-segment elevation, and Non-ST myocardial infarction after ROSC will benefit less from coronary angiography [8].

Acute alcohol intoxication causes various metabolic changes, cardiovascular side effects (causing tachycardia, peripheral vasodilation, and volume reduction, which induces hypothermia and hypotension), gastrointestinal side effects (nausea, vomiting, alcoholic hepatitis), and respiratory depression [9]. After ethanol intake, impairment in situations requiring skills and increase in talkativeness are observed in blood alcohol concentration below 50 mg/dl, while above 100 mg/dl relaxation, ataxia, hyperreflexia, lack of coordination, personality and behavioral changes, nystagmus, speech disorder are seen. Above 200 mg/dl, amnesia, diplopia, dysarthria, hypothermia, nausea, and vomiting are observed, while over 400 mg/dl results in respiratory failure, coma, and death. [9]

It is known that many intoxications such as carbon monoxide, cocaine, and cannabis cause acute myocardial infarction [10-12]. Cases of myocardial infarction have been reported as a result of ethanol intoxication. After excessive intake of ethanol, the increase in plasmogen activator inhibitor and the decrease in tissue plasmogen activator causes a decrease in fibrinolysis [13]. Repeated doses of excessive ethanol intake accelerate the progression of atherosclerosis and pose a risk for myocardial infarction. It has also been reported that excessive alcohol intake accelerates blood flow with tachycardia and causes thrombocyte activation [14-15].

Hypovolemia, hypoxia, acidosis, hypo/hyperkalemia, hypothermia, tension pneumothorax, cardiac tamponade, pulmonary and cardiac thrombosis, and toxins should be considered in return to spontaneous circulation (known as 5H-5T) [16]. Diagnoses of hypovolemia, tension pneumothorax, cardiac tamponade, pulmonary, and cardiac thrombosis were excluded by echocardiography, thoracoabdominal CT angiography, and coronary angiography in our case. Body temperature was normal in his vitals. There was no hypo/hyperkalemia in his laboratory. We believe that ST-segment elevation developed as a result of vasospasm and respiratory depression emerged after ethanol intake and that the patient had a cardiac arrest. It has been stated that coronary vasospasm can occur even when ethanol levels reach a basal level after 9 hours of excessive alcohol intake [17].

As a result, it has been published that excessive alcohol intake accelerates atherosclerosis, decreases fibrinolysis, increases platelet activation, and increases vascular tone. Thus, it poses a risk for acute myocardial infarction, while moderate and mild alcohol intake reduces the myocardial incidents as it slows atherosclerosis, accelerates fibrinolysis, decreases platelet activation, and decreases vascular tone [15-18-19]. Treatment of acute alcohol intoxication includes ensuring airway safety, providing mechanical ventilation support and sedation if necessary, controlling hypoglycemia and electrolyte disorders, and the administration of metadoxin to increase ethanol elimination [9].

CONCLUSION

Based on the results of this study, it can be concluded that ST-segment elevation after spontaneous return to circulation in out-of-hospital cardiac arrest patients may be caused by excessive ethanol intake, besides acute coronary syndromes.

Funding and Support: None to be declared
Conflict of interest: None

REFERENCES

1. Marx J, Hockberger R., Walls R, editors. Rosen's Emergency Medicine-Concepts and Clinical Practice. 9 th edition. Elsevier Health Sciences; 2013.p. 1838-1851.
2. Biyik I, Ergene OJJoimr. Alcohol and acute myocardial infarction. J Int Med Res. 2007 Jan-Feb;35(1):46-51.
3. Perkins GD, Graesner JT, Semeraro F, Olasveengen T, Soar J, Lott C, et al. European Resuscitation Council Guidelines 2021: Executive summary. Resuscitation. 2021 Apr;161:1-60.
4. Kelly EM, Pinto DS. Invasive Management of Out of Hospital Cardiac Arrest. Circ Cardiovasc Interv. 2019 Sep;12(9):e006071.
5. Nolan JP, Sandroni C, Böttiger BW, Cariou A, Cronberg T, Friberg H, et al. European Resuscitation Council and European Society of Intensive Care Medicine guidelines 2021: post-resuscitation care. Intensive Care Med. 2021 Apr;47(4):369-421.
6. Dumas F, Cariou A, Manzo-Silberman S, Grimaldi D, Vivien B, Rosencher J, et al. Immediate percutaneous coronary intervention is associated with better survival after out-of-

hospital cardiac arrest: insights from the PROCAT (Parisian Region Out of hospital Cardiac ArresT) registry. Circ Cardiovasc Interv. 2010 Jun 1;3(3):200-7.
7. Compagnoni S, Gentile FR, Baldi E, Contri E, Palo A, Primi R, et al. Peripheral perfusion index and diagnostic accuracy of the post-ROSC electrocardiogram in patients with medical out-of-hospital cardiac arrest. Resuscitation. 2021 Sep 8;168:19-26.
8. Rab T, Kern KB, Tamis-Holland JE, Henry TD, McDaniel M, Dickert NW, et al. Cardiac Arrest: A Treatment Algorithm for Emergent Invasive Cardiac Procedures in the Resuscitated Comatose Patient. J Am Coll Cardiol. 2015 Jul 7;66(1):62-73.
9. Vonghia L, Leggio L, Ferrulli A, Bertini M, Gasbarrini G, Addolorato G. Alcoholism Treatment Study Group. Acute alcohol intoxication. Eur J Intern Med. 2008 Dec;19(8):561-7.
10. Gao Y, Yang J, Ma L, Zhang Y, Li Z, Wu L, et al. Non-ST elevation myocardial infarction induced by carbon monoxide poisoning: A case report. Medicine (Baltimore). 2019 Apr;98(15):e15151.
11. Muñoz Pousa I, Hernández U, Jiménez Díaz VA. A case report of cocaine abuse, acute coronary syndrome, and eroded plaque: stent or not stent? Eur Heart J Case Rep. 2021 May 31;5(6):ytab128.
12. Matta A, Elenizi K, Elbaz M, Roncalli J. Left main coronary artery thrombus after cannabis consumption: a case report. Eur Heart J Case Rep. 2021 Jun 14;5(6):ytab179.
13. Van de Wiel A, Van Golde PM, Kraaijenhagen RJ, Von Dem Borne PA, Bouma BN, Hart HC. Acute inhibitory effect of alcohol on fibrinolysis. Eur J Clin Invest 2001; 31: 164 – 170.
14. Zhu Y, Carmeliet P, Fay WP. Plasminogen activator inhibitor-1 is a major determinant of arterial thrombolysis resistance. Circulation. 1999 Jun 15;99(23):3050-5.
15. Serebruany VL, Lowry DR, Fuzailov SY, Levine DJ, O'Connor CM, Gurbel PA. Moderate alcohol consumption is associated with decreased platelet activity in patients presenting with acute myocardial infarction. J Thromb Thrombolysis 2000; 9: 229 – 234.