Case Report

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A Rare Complication due to Methanol Poisoning: MINOCA

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Abstract

Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a relatively novel clinical puzzle characterized by limited research. In this case, we aimed to present the co-occurrence of methanol poisoning and MINOCA syndrome. This case demonstrated the importance of monitoring cardiac function with electrocardiography and cardiac biomarkers in patients within a high anion gap metabolic acidosis environment. This case report was showing co-occurrences of MINOCA syndrome with methanol poisoning. This was the first case of myocardial infarction in non-obstructive coronary arteries associated with methanol poisoning accompanied by high anion gap metabolic acidosis in the literature.

Keywords: Emergency, metabolic acidosis, MINOCA, poisoning

Introduction

Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a relatively novel clinical puzzle, accounting for approximately 10% of all acute myocardial infarction cases, characterized by limited research (1,2). In patients presenting with chest pain and acute myocardial injury detected by high-sensitivity troponin (hs-Tn) testing, the diagnosis is established when there is no stenosis of 50% or more in the coronary arteries on angiography (1). MINOCA constitutes a heterogeneous group of conditions that can develop due to various etiologies, including epicardial vascular causes such as coronary plaque rupture, spasm, and spontaneous dissection, as well as microvascular causes such as coronary thromboembolism and microvascular dysfunction, or non-ischemic events such as myocarditis and myopathies (3). Due to the differing pathophysiology, the management of patients with MINOCA differs significantly from that of other acute myocardial infarction (AMI) patients (4). In this case, we aimed to present the co-occurrence of methanol poisoning and MINOCA syndrome. We did not encounter any reported cases of myocardial infarction in non-obstructive coronary arteries associated with methanol poisoning accompanied by high anion gap metabolic acidosis in the literature we reviewed.

Case Report

A 41-year-old male patient was brought to our hospital's emergency department due to the sudden onset of altered consciousness. According to information obtained from the patient's relatives, a history of hypertension, hyperlipidemia and chronic alcohol consumption was reported. It was learned that on the day of admission, the patient consumed approximately one liter of homemade raki. Upon initial evaluation in the emergency department, the patient's overall condition was poor, with a Glasgow Coma Scale score of 6. His body temperature was 36.4°C, heart rate was 119 beats per minute, oxygen saturation (SpO_2) in room air was 85%, blood pressure was 70/40 mm-Hg, and blood glucose level was 100 mg/dL (Table-1). Electrocardiography (ECG) showed sinus tachycardia with T wave inversions in chest leads (Figure 1.A). Bedside echocardiography revealed an ejection fraction of 60%, no segmental wall motion abnormalities, and no evidence of pericardial effusion. The patient was intubated and connected to a mechanical ventilator.

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Methanol intoxication was considered due to the patient's history, clinical findings, and severe metabolic acidosis. Methanol level cannot be measured in our hospital. Paracetamol and salicylate levels were measured as toxicological parameters and the result was negative. Treatment for methanol poisoning was initiated. Fluid boluses and the dual inotropes were initiated due to hypotension. Intravenous sodium bicarbonate therapy was administered to correct acidosis. 50 mg of folic acid was started and the patient was urgently admitted to hemodialysis. The hourly monitoring of blood gas parameters for the patient who underwent continuous hemodialysis for 9 hours, along with admission and follow-up biochemical parameters, is presented in Table 1. After hemodialysis, the blood gas returned to normal. Throughout this period, the patient underwent ECG monitoring. The ECG findings associated with hyperkalemia resolved by the 3rd hour of hemodialysis in the patient whose hyperkalemia was corrected with hemodialysis (Figure 1.B). During the patient's latest follow-up, inferior ST-segment elevation myocardial infarction was detected on the ECG (Figure 1.C). The patient underwent emergency coronary angiography, which revealed normal coronary arteries.(Figure 2) Subsequently, the patient was admitted to the intensive care unit. During his hospitalization, he needed hemodialysis twice and plasmapheresis three times. The patient, who clinically improved during follow-up, was discharged after 47 days. The patient did not require hemolysis in subsequent follow-up.

Discussion

The MINOCA syndrome was first described 80 years ago. The initial studies conducted by De Wood et al. included demonstration of occluded coronary arteries in 90% of ST-elevation myocardial infarction (STEMI) patients. While this groundbreaking study emphasized the importance of coronary obstruction in the pathogenesis of AMI, it also confirmed the absence of obstruction in approximately 10% of patients (5). With the publication of a position paper on MINOCA by the ESC in 2016, its significance has been better understood and has paved the way for many subsequent studies. Particularly due to its occurrence in younger patients and the differences in etiology, prognosis, and treatment protocols, clinicians should pay special attention to it (3). In its etiopathogenesis, coronary artery spasm and coronary plaque rupture are the two most commonly known causes (3). Approximately 40% of MINOCA cases arise from plaque rupture. Plaque rupture leads to less than 50% stenosis of the coronary artery lumen. Coronary artery spasm is another major factor implicated in its etiology. This condition is thought to occur as a result of abnormal responses of vascular smooth muscles to endogenous or exogenous stimuli (3). When we look at other causes, myocarditis is also one of the most common non-coronary reasons for MINOCA. It is estimated to account for approximately one-third of cases. It primarily stems from Coxsackie virus, adenovirus, influenza

Table 1: Monitoring of the patient's blood gas, complete blood count, and biochemical tests

Arterial Blood Gas Parameters		pH	HCO_3	pCO_2 (mmHg)	Base excess	Potassium(mmol/L)	Lactate (mg/dL)
0th hour		6.81	7.2	46.6	-26.9	6.3	21
1st hour		6.91	8.9	49.8	-22.7	5.9	20
2nd hour		7.05	10.3	34.5	-20.9	5.9	16
3rd hour		7.14	14.4	45.0	-13.5	3.9	20
4th hour		7.25	17.2	41.7	-8.5	3.2	27
5th hour		7.29	21.7	51	-1.8	3.2	12
6th hour		7.34	21.3	40.8	-3.7	2.8	10
7th hour		7.33	24.2	51.8	+1.4	2.7	7
8th hour		7.37	23.9	43.7	+0.5	3.1	10
9th hour		7.41	23.3	35.8	-1.6	3.7	12
Biochemical Parameters	**0th hour**	**9th hour**	**1st day**	**2nd day**	**3rd day**	**7th day**	**15th day**
Hs Troponin-I (ng/L)	25	73	-	233500	56500	-	315
Urea (mg/dL)	125	19	32	124	188	116	142
Creatinine (mg/dL)	4.75	0.79	1.1	4.16	5.74	1.62	4.33
Aspartate aminotransferase (U/L)	122	167	433	393	105	47	178
Alanine aminotransferase (U/L)	84	69	85	98	62	32	490
White Blood Cell (10^3/µl)	17.6	4.6	3.8	16.2	24.3	35.2	19.2
Hemoglobin (g/dL)	12.8	12.9	11.4	12.8	10.5	10.2	6.7
hematocrit (%)	46.5	37.1	32.4	37.8	30.5	31.7	21.4
Platelet counts (10^3/µl)	89	50	38	55	80	247	177
INR	1.19	0.94	1.58	1.0	1.08	1.18	1.35
APTT (s)	53.3	30.7		33.2	27.3	29.6	37.5
Ethanol (Promil)	0.074						

HCO_3: Bicarbonate, pCO_2 : partial pressure of carbon dioxide, mmHg: millimeters of mercury,dl: deciliter, g: gram, L: liter, mEq: milliequivalent, mg: milligram, mL: milliliter, mm: millimeter, mmol: millimoles, s: second, U:unit, ng:nanogram

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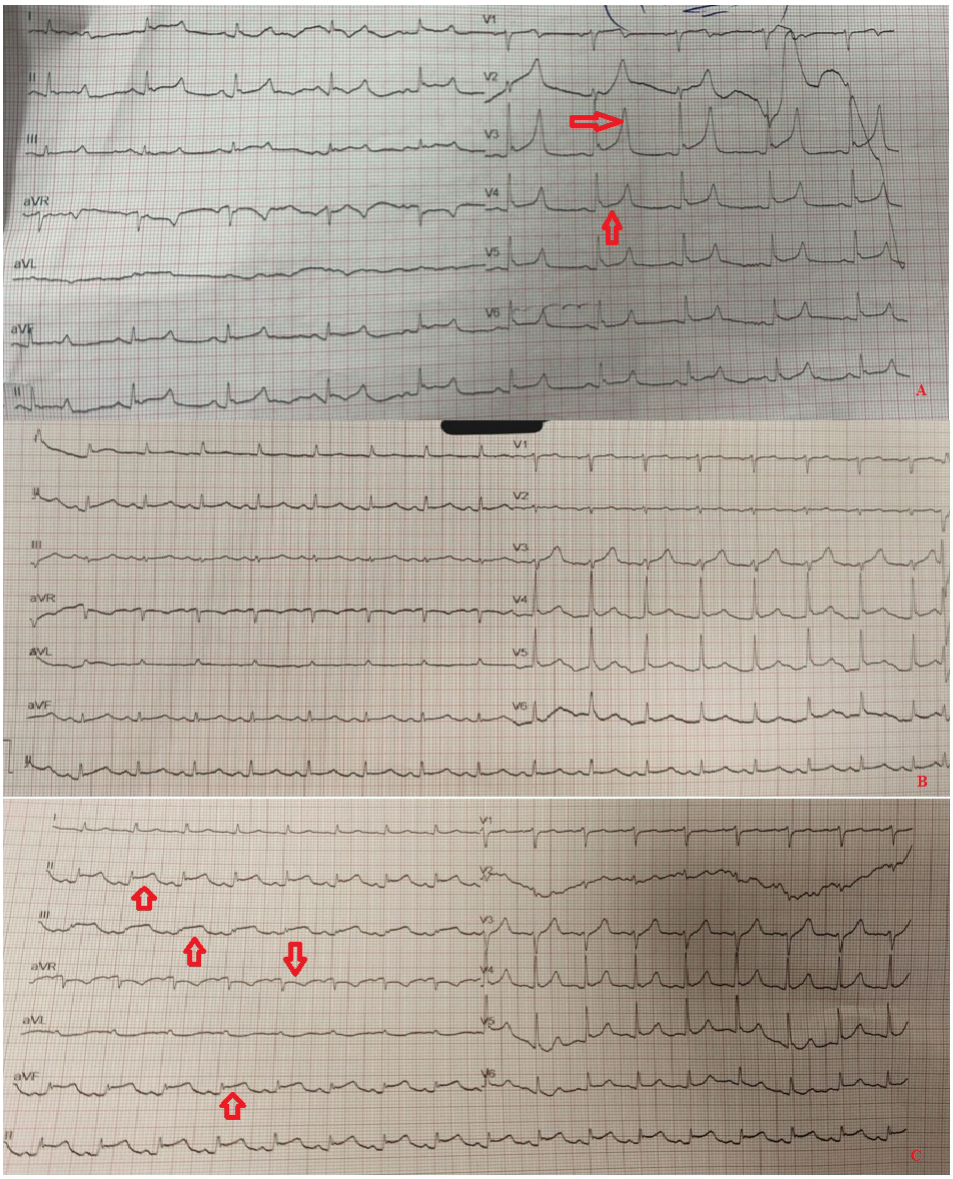

Figure 1. The initial electrocardiogram of the patient highlights T-wave inversions in the chest leads, as indicated by the red arrow **(A)**. This was accompanied by hyperkalemia. It is observed that the T-wave inversions on the electrocardiogram returned to normal after the potassium level returned to normal **(B)**. In the subsequent electrocardiogram, ST segment elevation observed in the inferior leads, indicated by red arrows, is visible **(C)**.

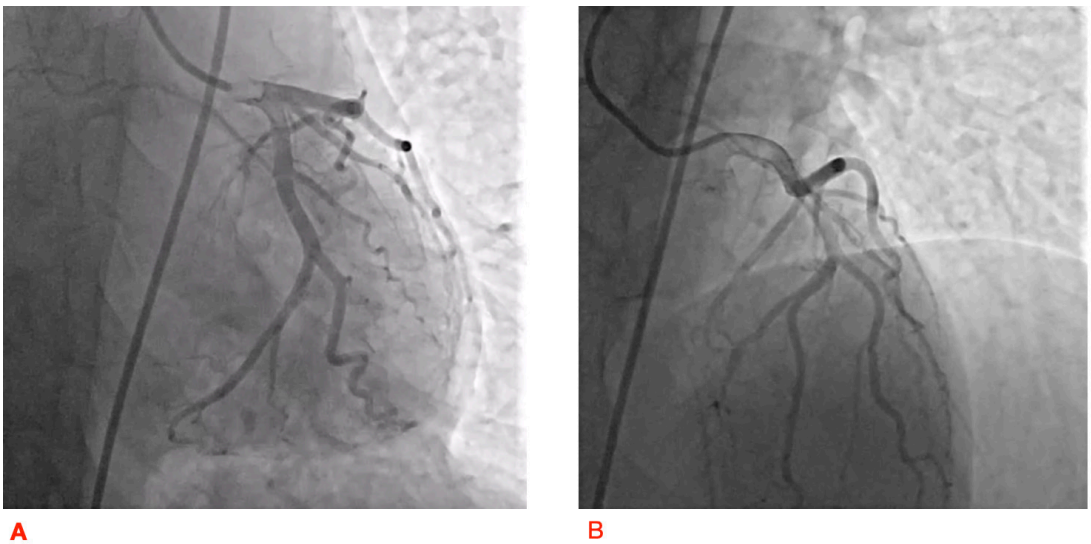

Figure 1. The patient's coronary angiography findings. It is observed the normal right coronary artery and its branches (A). It is observed the normal left coronary artery and its branches (B).

virus, and Epstein-Barr virus (3). When the literature is reviewed, it is observed that MINOCA syndrome accompanied by methanol poisoning has not been reported.

Although methanol level was required for the diagnosis of methanol poisoning, this test was not available in our hospital, as in most centers. Diagnosis is made with clinical suspicion, history of illicit alcohol use, characteristic examination findings (neurological, ophthalmological, etc.) and the presence of metabolic acidosis with a wide anion gap. (6,7) There are very limited datas in the literature regarding the effects of methanol poisoning on the cardiovascular system.(6)

Although the direct cardiovascular effects of methanol or its metabolites are unknown, The hemodynamic instability and high anion gap metabolic acidosis disrupted in methanol poisoning can lead to both impaired coronary artery perfusion and suppression of myocardial tissue function. Some studies have found ECG changes in cases of methanol poisoning.(6-8) Although the observed ST segment elevation in our case provided metabolic and hemodynamic stability, it indicates that coronary and myocardial involvement persists. This case demonstrates the importance of monitoring not only cardiac function but also ECG and cardiac biomarkers in patients within a high anion gap metabolic acidosis environment.

Conclusion

Cardiac involvement, which is less common in patients with methanol poisoning than gastrointestinal and neuro-ophthalmological findings, should definitely be kept in mind. Therefore, routine cardiac monitoring is important in suspected patients.

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