CASE REPORT / OLGU SUNUMU

Patients Are Not Always Honest! Case Report

Hastalar Yalan Söyler: Olgu Sunumu

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ABSTRACT

Aim: Methanol intoxication is well known for its ocular, neurologic, metabolic and gastrointestinal findings but cardiac effects are rarely found in literature. Patients often admit to hospital with a witnessed alcohol intake history.

Case Report: This case had admitted to the hospital with back pain. He had ST segment elevated myocardial infarction (STEMI). According to the clinical findings, methanol intoxication was also detected. Case A 34 years old male patient was admitted to the hospital with severe back pain via ambulance. He was oriented and cooperated. His GCS score was 15. His vital findings revealed TA 90/50 mmHg, blood oxygen saturation 92%, pulse 110/bpm. On electrocardiography (ECG) inferolateral ST elevation was noted. During monitorization, lack of eye contact continued, he stated that he lost sight for 3 hours. He stated that he had consumed a bottle of cologne (the size of the bottle is unclear) the night before. The patient's blood gas test revealed pH 6,8, HCO^3 6, base excess -34mmol/l. On biochemical examination, blood urea nitrogen (BUN) was 42 mg/dl, creatinine was 1,1 mg/dl. His complete blood count revealed hemoglobin 14,5 g/dl and white blood cell (WBC) 16,500/mm^3. The blood methanol level at admission was 156 mg/ml.

Conclusion: The ECG findings of methanol intoxication are limited. Previous reports have documented axis variations, non-specific T wave changes, QRS complex alterations and prolonged QTc intervals. Taking into consideration the limited number of these cases in literature, the systematic ECG analysis of this population has not been reported yet. In coronary angiography, it is shown that all the coronary vessels were normal.

Physicians should always be alert for alternative diagnosis and should take detailed anamnesis. Our sustained clinical suspicion of a patient with an obvious different diagnosis has taken us to another and accurate result.

Keywords: Methanol, intoxication, acute coronary syndrome

ÖZ

Amaç: Metanol zehirlenmesi oküler, nörolojik, metabolik ve gastrointestinal bulguları ile iyi bilinir, fakat kalp etkileri literatürde nadir görülmektedir. Hastalar sıklıkla tanıklı alkol alım öyküsü ile hastaneye başvurmaktadır.

Olgu Sunumu: Bu vaka sırt ağrısı ile hastaneye başvurdu. ST segment yükselmeli miyokard infarktüsü (STEMI) tespit edildi. Klinik bulgulara göre, metanol zehirlenmesi de saptandı. Vaka 34 yaşında erkek hasta ambulansla ağır bel ağrısı şikayeti ile hastaneye yatırıldı. Oryante ve koopereydi. GCS skoru 15 idi. Hayati bulguları TA 90/50 mmHg, kan oksijen satürasyonu % 92, nabız 110/bpm olduğu görüldü. Elektrokardiyografide (EKG) inferolateral ST yükselmesi kaydedildi. Monitörizasyon sırasında göz teması eksikliği devam etti, görüşünü son 3 saattir kaybettiğini belirtti. Bir gece önce bir şişe kolonya içtiğini (şişenin büyüklüğü belli değil) ifade etti. Hastanın kan gazı testi pH 6,8, HCO^3 6, baz fazlalığı -34mmol/l olarak gösterdi. Biyokimyasal incelemede kan üre azotu (BUN) 42 mg/dl, kreatinin 1,1 mg/dl idi. Tam kan sayımı hemoglobin 14,5 g/dl ve beyaz kan hücresi (WBC) 16,500/mm^3 idi. Giriş sırasındaki kan metanol seviyesi 156 mg/ml idi.

Sonuç: Metanol zehirlenmesinin EKG bulguları sınırlıdır. Önceki raporlarda, eksen değişiklikleri, spesifik olmayan T dalgası değişiklikleri, QRS karmaşık değişiklikleri ve uzun QTc aralıkları kaydedilmiştir. Literatürdeki bu vakaların sınırlı sayıda olduğu göz önüne alındığında, bu popülasyonun sistematik EKG analizi henüz bildirilmemiştir. Koroner anjiyografide tüm koroner damarların normal olduğu gösterilmiştir.

Doktorlar alternatif tanı için her zaman uyanık olmalı ve detaylı anamnez almalıdır. Belirgin, farklı bir tanısı olan bir hastanın sürekli klinik şüphesi bizi başka ve doğru bir sonuca götürdü.

Anahtar kelimeler: Metanol, intoksikasyon, akut koroner sendrom

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Introduction

Methyl alcohol (methanol) is a toxic, colorless and odorless chemical substance which tastes like ethyl alcohol. This substance is used in anti-freeze solutions, solvents and many other materials like cologne [1]. Methanol intoxication occurs due to formation of formic acid and formaldehit which results in metabolic acidosis [2]. Formic acid accumulation promotes systemic symptoms whereas formaldehit leads to tissue hypoxia via inhibiting mitochondrial respiration and so increasing lactate production [3]. Symptoms generally occur following 12-24 hours of oral intake due to the slow degradation of methanol to its toxic substances; formaldehit and formic acid [4]. Subsequent to latent period, broad spectrum of clinical features from headache to death is seen associated mostly with neurologic and ocular findings [5].

Methanol intoxication is well known for its ocular, neurologic, metabolic and gastrointestinal findings but cardiac effects are rarely found in literature [6]. Patients often admit to hospital with a witnessed alcohol intake history. Altered conscious or atypical symptoms and findings may also be present. This case had admitted to the hospital with back pain. He had ST segment elevated myocardial infarction [STEMI]. Although the patient did not give any information about alcohol intake, with clinical findings, methanol intoxication was also detected. We suggest to emphasize the possibility and importance of concomitant life-threatening diseases especially when the patient has a very precise diagnosis.

Case Report

A 34 years old male patient was admitted to the hospital with severe back pain, perspiration and general condition disorder via ambulance. He was agitated and he had hyperhidrosis. He was oriented and cooperated. His GCS score was 15. His vital findings revealed TA 90/50 mmHg, blood oxygen saturation 92%, pulse 110/bpm. On oscultation, bilateral pulmonary rales were detected. On electrocardiography [ECG] inferolateral ST elevation was noted and the patient was consulted to Cardiology department. Coronary angiography [CAG] was planned [Fig.1]. On physical examination, many incisive scars were seen on chest anterior wall and on arms. While taking history due to the patient's acidotic respiration, disrupted eye contact and agitation, drug or alcohol abuse was suspected. The patient insistently denied. During monitorization, lack of eye contact continued, the patient is questioned whether he could see, and he stated that he lost sight for 3 hours and everywhere seemed all white. Methanol intoxication suspicion rouse and the patient was questioned for cologne intake. He stated that he had consumed a bottle of cologne [the size of the bottle is unclear] the night before. It was learned that the patient was a chronic alcohol consumer when his relatives arrived to the emergency department.

The patient's blood gas test revealed pH 6.8, HCO3 6, base excess -34mmol/l. On biochemical examination, blood urea nitrogen [BUN] was 42 mg/dl, creatinine was 1,1 mg/dl. His complete blood count revealed hemoglobin 14.5 g/dl and white blood cell [WBC] 16.500/mm3. Other laboratory tests were normal. The patient was intubated after deepening of acidotic respiration and tachypnea of 20/min. The patient is diagnosed as methanol intoxication and medical treatment was started. CAG was done and the patient was transferred to intensive care unit. CAG revealed global hypokinetic and dilated cardiomyopathy without any coronary artery pathologies. During intensive care unit follow up, increased intravenous hydration, NaHCO3, ethanol, Fomepizol and venovenous hemodiafiltration was done. Methanol blood level cannot be measured in our hospital. The patient is accepted as forensic case so blood sample was sent for testing to Forensic Medicine laboratory. The result was obtained the next day.

The blood methanol level at admission was 156 mg/ml. The fourth day measure was 10mg/dl. Unfortunately, the patient died at the end of the fourth day of hospitalization. Written informed consent was obtained from the relatives of patient.

Discussion

Acidosis is well established in methanol intoxication but the real cause- whether it is due to formic or lactic acid formation- is still unclear [7]. Independent from the etiology, it is well known that metabolic acidosis has negative impacts on cardiovascular system. Cardiac contraction and flow decrease and arterial vasodilation that contributes hypotension occurs [8]. In animal models of metabolic acidosis, ventricular arrhythmia tendency is often seen [8].

The ECG findings of methanol intoxication are limited. Previous reports have documented axis variations, non-specific T wave changes, QRS complex alterations and prolonged QTc intervals [9-10]. Taking into consideration the limited number of these cases in literature, the systematic ECG analysis of this population has not been reported yet. There is not an association between metabolic acidosis and ECG changes.

In animal models, response to methanol exposure, cardiac dilatation and myocyte degeneration have been reported [11]. In a study of dogs, intravenous methanol injection resulted in decreased cardiac flow, stroke volume and systemic arterial pressure and increased total peripheral resistance [9]. With these findings, we can speculate that methanol intoxication has a depressive effect on myocardium in humans. In another study, it is stated that methanol intoxication may cause minor changes [10].

The ECG of our case revealed inferolateral ST elevation; however, in CAG, it is shown that all the coronary vessels were normal and the left ventricle was globally hypokinetic.

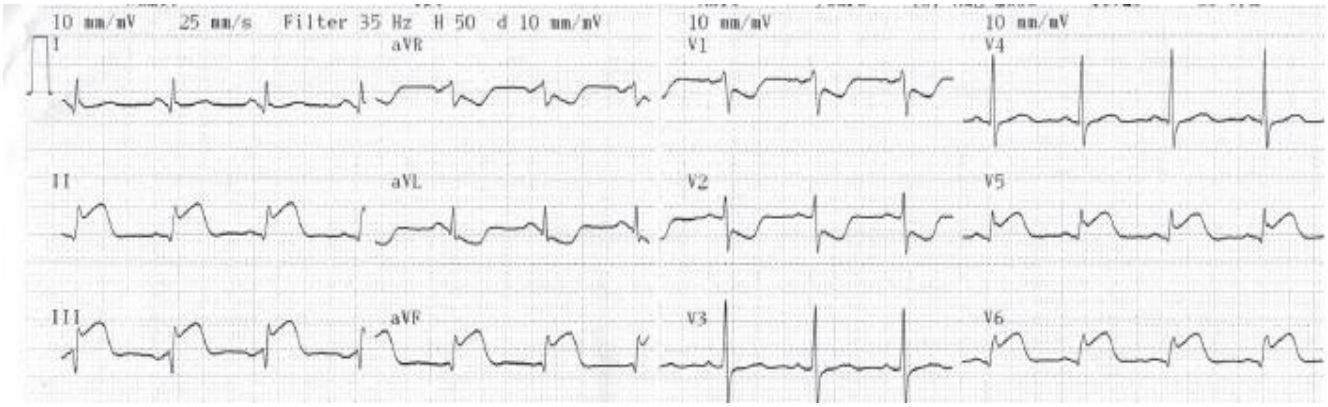

Figure 1. T segment elevation at DII, DIII, aVF, V5 and V6 derivations

The concomitance of enhancement of these ECG and clinical findings of the patient, it is speculated that the findings were associated with methanol intoxication. Methanol and formic acid may both depress cardiac pace and myocardium contraction [9]. Since formic acid may inhibit cytochrome oxidase activity in mitochondria, it has the potential to promote histotoxic hypoxia if this inhibition takes place in the terminal stage of respiratory chain [12]. Sympatic system activation results in pulmonary hypertension and may trigger right atrial overload and right ventricle pause [9].This presented case is the only patient with ST elevation detected in ECG with methanol intoxication reported in literature.

As a conclusion, methanol intoxication still surprises with novel clinical findings. This case is one of these. One should always be alert for alternative diagnosis and should take detailed anamnesis. Our sustained clinical suspicion of a patient with an obvious different diagnosis has taken us to another and accurate result.

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Authors' Contribution: ÇK conceived the case report. ÇK, AŞ contributed reagents, materials, analysis tools or data. ÇK, AŞ, TK drafted the manuscript and all authors contributed substantially to its revision.

Informed Consent Statement: Written informed consent was obtained from the relatives of patient for publication of this case report and any accompanying images. A copy of the written consent is available for review in this journal.

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