Acute myocardial infarction associated with disulfiram-alcohol interaction in a young man with normal coronary arteries

Koroner arterleri normal olan genç bir erkekte disülfiram-alkol etkileşimi ile ilişkili akut miyokard infarktüsü

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Acute myocardial infarction due to acetaldehyde syndrome has been rarely reported. A 22-year-old, chronic alcoholic man was admitted to our hospital with typical angina pectoris that developed after oral intake of disulfiram and alcohol together. The electrocardiogram showed hyperacute inferior myocardial infarction and he was successfully treated by thrombolytic therapy. Coronary angiogram revealed normal coronary arteries; thus, the event was probably secondary to coronary artery thrombosis and/or coronary vasospasm. Disulfiram is not a safe drug in patients unable to adhere to the strict restriction of alcohol intake, requiring a close supervision of individuals on disulfiram therapy.

Key words: Alcohol deterrents/adverse effects; coronary vasospasm; disulfiram/adverse effects; myocardial infarction/chemically induced; thrombosis.

Asetaldehit sendromu sonucunda gelişen akut miyokard infarktüsü nadirdir. Kronik alkol bağımlısı olan 22 yaşında bir erkek hasta hastanemize, disülfiram ile birlikte alkol aldıktan sonra başlayan tipik angina pektoris ile başvurdu. Elektrokardiyogramında hiperakut inferior miyokard infarktüsü saptanan hasta trombolitik tedavi ile başarıyla iyileşti. Koroner anjiyografisi normal olan hastada, olayın büyük olasılıkla koroner arter spazmı veya trombozuna bağlı olarak geliştiği düşünüldü. Disülfiram, beraberinde alkol alma kısıtlamasına tam olarak uyamayan hastalar için güvenli bir ilaç değildir ve bu kişilere disülfiram verilirken yakından takip gerekir.

Anahtar sözcükler: Alkolden caydırıcı/yan etki; koroner vazospazm; disulfiram/yan etki; miyokard infarktüsü/kimyasala bağlı; tromboz.

The underlying pathology in acute myocardial infarction (AMI) is generally acute coronary artery obstruction due to atherosclerotic plaque erosion or rupture.[1] However, AMI may also occur when the coronary arteries are normal or nearly normal.[2,3] The underlying mechanisms proposed for myocardial infarction with normal coronary arteries (MINCA) include hypercoagulable states, coronary embolism, nonatherosclerotic coronary artery disease, coronary vasospasm, coronary thrombosis, and coronary trauma. We report a rare case of a chronic alcoholic young man who suffered an inferior myocardial infarction despite normal coronary anatomy while on disulfiram therapy.

CASE REPORT

A 22-year-old chronic alcoholic man was admitted to our emergency department with severe chest pain of sudden onset, two hours after ingesting 500 mg disulfiram and high-dose alcohol. He was a smoker (20 cigarettes/day) for five years and there was no family history of premature coronary artery disease. On physical examination, he was agitated, his blood pressure was 80/50 mmHg, heart rate was regular and 98 beats/min, and respiration rate was 32/min. The electrocardiogram showed 1-2-mm ST-segment elevation in DII, DIII, and AVF derivations accompanied by 1-mm ST-segment horizontal depression in V1-V3 leads (Fig. 1). The diagnosis was acute inferior myocardial infarction. Because his chest pain started half an hour before admitting to hospital, vasodilatator therapy with intravenous nitroglycerine infusion was given for eradication of probable coronary vasospasm. However, both the chest pain and ischemic changes on the electrocardiogram persisted despite 30 minutes of

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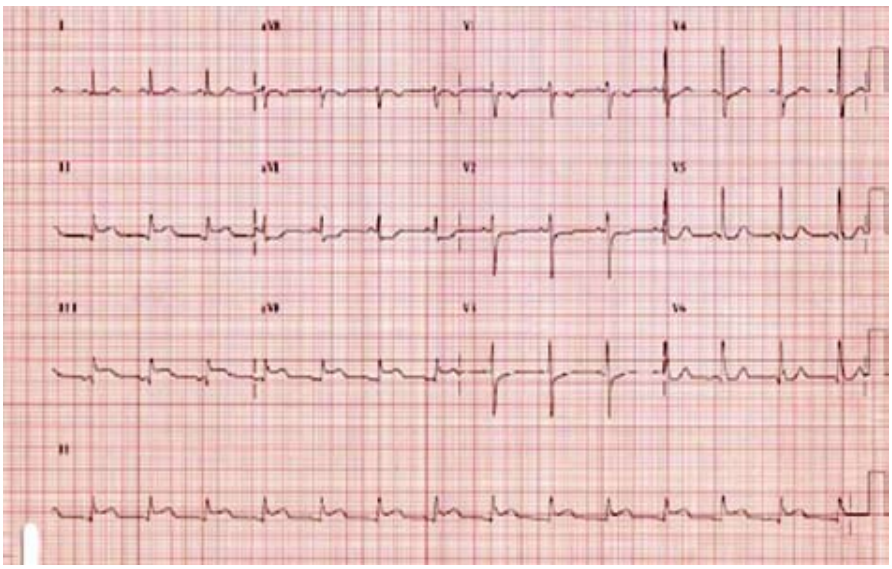

Figure 1. The electrocardiogram showed 1-2-mm ST-segment elevation in DII, DIII, and AVF derivations accompanied by 1-mm ST-segment horizontal depression in V1-V3 leads.

parenteral vasodilatator therapy. Therefore, intravenous thrombolytic therapy with tissue-type plasminogen activator was instituted. Two hours after the initiation of thrombolytic therapy, his chest pain disappeared accompanied by complete ST-segment resolution on the electrocardiogram. The peak troponin T was 1.38 ng/dl and creatine kinase MB was 128 ng/dl). Echocardiography showed mild hypokinesia in the basal and mid-segment of the inferior wall. Three days after admission, the patient was transferred to a tertiary centre for further assessment. Coronary angiography showed normal coronary arteries and normal left ventricular function and he was discharged with medical therapy (Fig. 2). There was no other cause associated with coronary vasospasm or thrombosis and the development of AMI was attributed to disulfiram-alcohol interaction. The disulfiram therapy was stopped and the patient was advised to give up alcohol intake and smoking.

DISCUSSION

Myocardial infarction generally arises from coronary artery disease. However, in selective coronary angiography series, the incidence of AMI patients with normal or near-normal coronary arteries ranges from 1% to 12%.[4] The mechanism leading to AMI in these patients remains unknown. It has been hypothesized that the probable mechanism may be tempo-

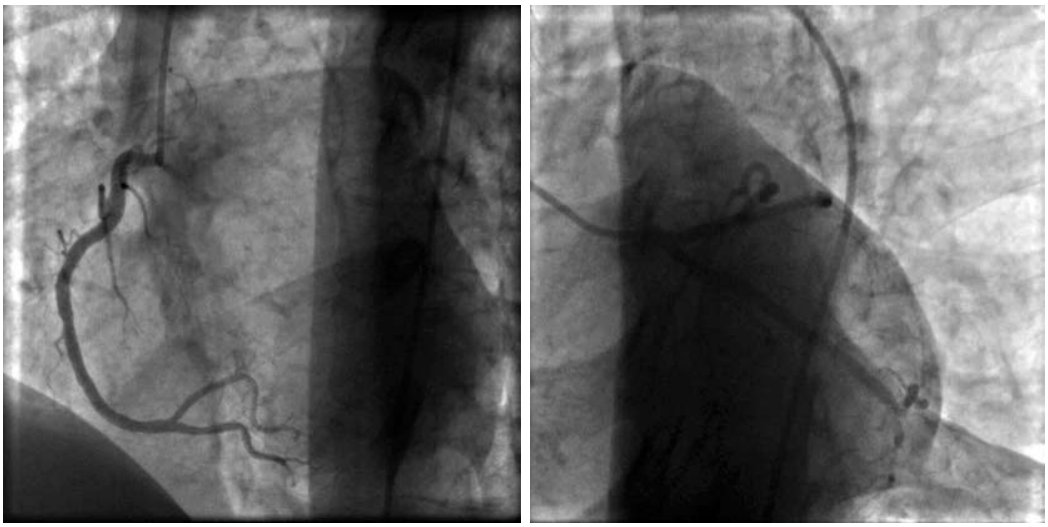

Figure 2. Coronary angiograms showing normal coronary arteries.

rary occlusion of the infarct-related vessel by spasm or thrombus or a combination of both.[5]

Myocardial infarction with normal coronary arteries mainly affects young people and it is distinctly rare in patients older than 50 years.[6] The main underlying mechanisms are hypercoagulable states, coronary embolism, nonatherosclerotic coronary diseases, coronary vasospasm, coronary thrombosis, and coronary trauma. Smoking, high-dose alcohol intake and disulfiram intake and cocaine addiction have also been implicated as causes of MINCA.[7]

Disulfiram, which has been used since 1940 for treatment of alcohol addiction, is not a purely safe drug since, other than known acetaldehyde syndrome, there are some reported cases of intracranial hemorrhage and paroxysmal hypertension with convulsions and acute pulmonary edema secondary to ingestion of disulfiram and alcohol together.[8,9] Clinical signs of acetaldehyde toxicity due to disulfiram-alcohol interaction include flushing, nausea, tachycardia, and hypotension. Development of AMI has also been reported in an alcoholic subject receiving disulfiram therapy.[10]

The initial management of patients presenting with disulfiram-induced AMI is the standard treatment for myocardial infarction, including pain relief, aspirin, thrombolysis if indicated, and beta-blockade. The prognosis of MINCA is more favorable than that of AMI with angiographic disease. In our case, acetaldehyde syndrome probably caused an acute inferior myocardial infarction through vasospasm and acute coronary thrombosis. Thrombolytic therapy was successful and coronary angiography showed normal coronary anatomy as expected.

In conclusion, it is important to remember that disulfiram therapy is not a safe treatment in patients unable to adhere to the strict restriction of alcohol intake throughout the duration of disulfiram action, requiring a close supervision of these individuals on disulfiram therapy.

REFERENCES

1. Germing A, Lindstaedt M, Ulrich S, Grewe P, Bojara W, Lawo T, et al. Normal angiogram in acute coronary syndrome-preangiographic risk stratification, angiographic findings and follow-up. Int J Cardiol 2005; 99:19-23.
2. McKenna WJ, Chew CY, Oakley CM. Myocardial infarction with normal coronary angiogram. Possible mechanism of smoking risk in coronary artery disease. Br Heart J 1980;43:493-8.
3. Salem BI, Haikal M, Zambrano A, Bollis A, Gowda S. Acute myocardial infarction with "normal" coronary arteries: clinical and angiographic profiles, with ergonovine testing. Tex Heart Inst J 1985;12:1-7.
4. Alpert JS. Myocardial infarction with angiographically normal coronary arteries. Arch Intern Med 1994; 154:265-9.
5. Lindsay J Jr, Pichard AD. Acute myocardial infarction with normal coronary arteries. Am J Cardiol 1984;54:902-4.
6. Rigatelli G, Rigatelli G, Rossi P, Docali G. Normal angiogram in acute coronary syndromes: the underestimated role of alternative substrates of myocardial ischemia. Int J Cardiovasc Imaging 2004;20:471-5.
7. Heath MJ, Pachar JV, Perez Martinez AL, Toseland PA. An exceptional case of lethal disulfiram-alcohol reaction. Forensic Sci Int 1992;56:45-50.
8. Guarnaschelli JJ, Zapanta E, Pitts FW. Intracranial hemorrhage associated with the disulfiram-alcohol reaction. Bull Los Angeles Neurol Soc 1972;37:19-23.
9. Fourrier A, Mouton YJ, Raviart B, Roger J. A paradoxical disulfiram-alcohol reaction: paroxysmal hypertension with convulsions and acute pulmonary edema. Lille Med 1973;18:146-9. [Abstract]
10. Nucifora G, Cassin M, Brun F, Nicolosi GL. Anterior myocardial infarction in a chronic alcoholic man on disulfiram therapy: a case report. Ital Heart J Suppl 2004; 5:900-4. [Abstract]