Case reports

Acute non-Q Myocardial Infarction Associated with Cocaine Abuse in a Young Man with Normal Coronary Arteries

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As cocaine abuse has become widespread, the number of cocaine- related cardiovascular complications has increased significantly. This report describes a young man with angiographically normal coronary arteries in whom cocaine abuse lead to coronary thrombosis and infarction.

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Cocaine is an alkaloid extracted from the leaves of the plant Erythroxylon Coca and it presents properties similar to a potent sympathomimetic agent in man[1,2].

Cocaine has a powerful effect on the cardiovascular system causing heart rate increase, hypertension and increased myocardial contractility.

In the CNS, its effects include transient cheerfulness, euphoria, hallucinations, increase of working performance and reduction of the feeling of fatigue. Due to the pleasant feelings it causes, to its easy use and to the erroneous notion that it is not dangerous, its abuse in USA in the 80s reached epidemic proportions and in 1999 cocaine abusers numbered 1.5 million people[3]. In this large population of users, significant increase of cardiovascular events has been observed, such as angina, myocardial infarction, cardiomyopathies and sudden deaths due to cocaine abuse[4].

In this paper, we describe the case of a young man who suffered an acute myocardial infarction due to cocaine abuse. We also review Greek and international literature with regard to the effects of cocaine on the heart, the diagnosis and treatment of cardiovascular events in cocaine users.

Case description

A 32 year old male, working as a cashier in a night club, came for cardiological examination, on an outpatient basis, approximately two days after an episode of prolonged retrosternal pain, accompanied by intense diaphoresis following cocaine use.

Aside from hyperthyroidism regulated with anti-thyroid drugs (thiamazole 20 mg/day), no history of other diseases was mentioned. The patient said that he was a cocaine user, usually in the form of powder, through nose inhalation, but never intravenously. He was also a mild smoker, while he did not consume excessive alcohol.

During the examination, the patient was anxious and hyperactive, without any further symptoms.

The objective examination did not reveal any special pathological findings. The patient was slim and presented vitiligo with a distribution of the white spots on the face, hands and trunk. The peripheral veins of the upper and lower limbs were

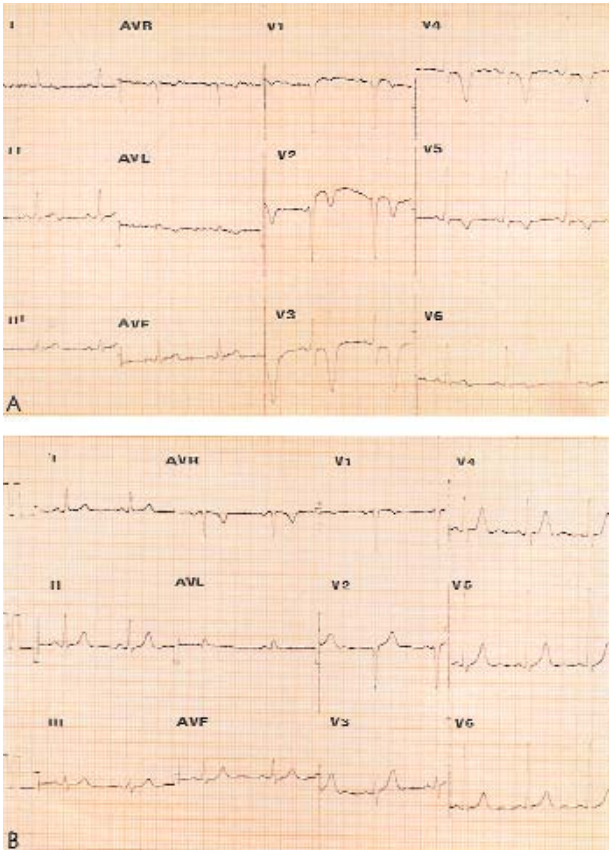

Figure 1. (A). Initial ECG: it shows T wave inversion in leads I, aVL, V1-V6. (B). Normal ECG eight months later.

normal. Arterial pressure was 110/70 mmHg, his heart rate was 87 bpm and his temperature was 36.8˚C. The ECG showed sinus rhythm, 83 bpm, negative T waves in leads I, aVL, V1-V6 and rS V1,V2 (Figure 1A). The chest X-ray was normal. The echocardiograph showed a left ventricle with normal dimensions (EDD 55mm, ESD 36mm), normal wall thickness (8mm) and normal systolic function (EF 64%, SF 35%). The apex of the left ventricle was akinetic with a large thrombus (Figure 2). The heart valves, the left atrium and the right cardiac cavities were normal. Also, there was no pericardial effusion. Hematological and biochemical blood tests including general blood culture, red cell sedimentation rate, prothrombin time, urea, glucose, uric acid, lipidemic profile (cholesterol, HDL, LDL, triglycerides) were normal while a slight increase of SGOT (72 IU) was observed that gradually returned to normal in the next few days. T3, T4 and TSH values were within normal limits. VDRL, HBV and ELISA tests were negative.

The patient underwent cardiac catheterization that showed a slight transparent area at the end of the first third of the anterior descending, after the origin of a big diagonal branch, probably due to a recent thrombosis of the artery. The middle and peripheral part of the vessel did not present any atheromatous lesions. The stem and its bifurcation, the circumflex artery a large, centrally originating marginal branch, and the right coronary artery did not present any stenoses. The left ventricle had normal dimensions and ejection fraction (EF 55%): although in the apical area, localized hypokinesia – akinesia was observed with radiographic deficit due to the presence of thrombi (Figure 3).

Upon completion of the clinical and laboratory evaluation, the patient left the hospital with the diagnosis of myocardial infarction due to thrombosis of the anterior descending branch of the left coronary artery.

The following drugs were prescribed to the patient: anticoagulant treatment with 4 mg acenocoumarole per day, acetylsalicylic acid in daily doses of 325 mg for platelet aggregation inhibition and nifedipine at a dose of 10 mg three times a day for the prevention of coronary artery spasm. The administration of anticoagulants was suggested due to the presence of a large thrombus in the akinetic apex of the left ventricle. The patient was also informed of his condition in detail and of the effects of cocaine on the heart, he was encouraged to stop using cocaine and to seek help from a psychologist experienced in treating drug addicts.

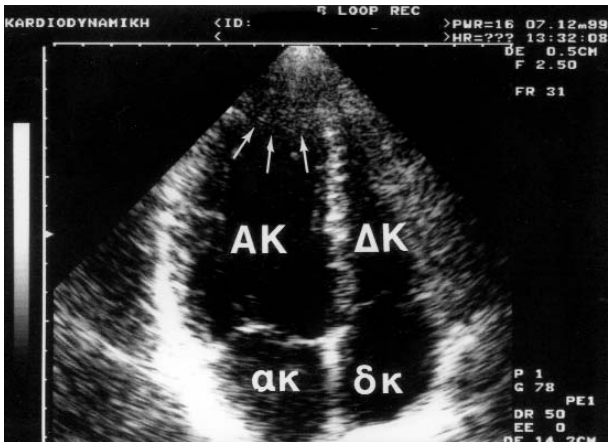

Figure 2. Two-dimensional Echocardiogram, 4-chamber view demostrating a left ventricular (AK) apical thrombus (arrows). ΔK:right ventricle, δκ:right atrium, ακ:left atrium.

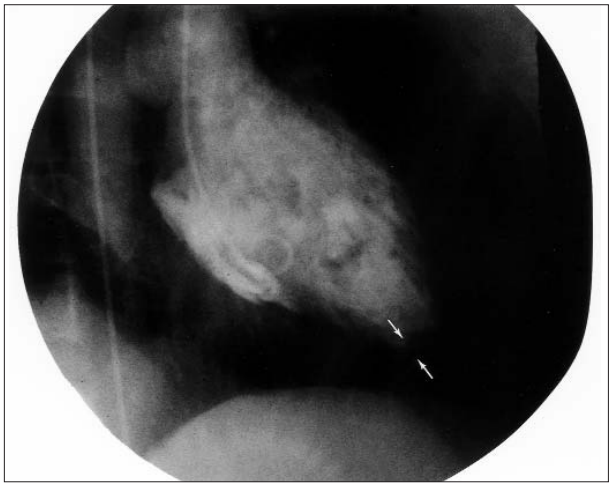

Figure 3. Left ventriculogram in the right oblique view: a large mural thrombus attached to the endocardium of the apex (arrows).

The patient systematically followed the treatment and gradually regained his activities with a complete absence of symptoms. With regard to cocaine abuse he said that he had discontinued the habit.

The last examination was performed eight months after the first one. The ECG revealed normal T waves, while the rS morphology remained in the V1 and V2 leads (Figure 1B). The echocardiograph showed improved apical motility with a permanent wall thrombus in this area. The dimensions, wall thickness and the ejection fraction of the left ventricle were normal. Arterial pressure was normal. T3, T4 and TSH values were normal. We advised him to stay on 325 mg aspirin daily. A few months after his last examination, we were informed that he had died of pulmonary edema and that he had continued cocaine abuse.

Discussion

Cocaine abuse has acquired impressive dimensions: in the USA, in 1999, 25 million people had made use of cocaine at least once in their lives, while 1.5 million were frequent users[3].

In Greece, the precise number of cocaine users is not known. In epidemiological surveys conducted by the School of Medicine of the University of Athens and the Mental Health Research University Institute (EPIPSI), it was calculated that the percentage of drug and other illegal substances users in 1998 reached 12.2%[5]. The same survey reports that cannabis was the most popular hallucinogenic substance followed by cocaine, with a much lower percentage. The percentage of those who responded that they had used cocaine once in their life time reached 2.7% for age groups 25-35 years old and 1.1% for the total of the sample population.

A remarkable phenomenon observed recently in the population of cocaine users in the States, is the dramatic increase of cardiovascular events and sudden deaths[4].

Angina pectoris in cocaine users is so frequent and the population of users so large, that it is deemed appropriate for individuals admitted to the emergency department with chest pain of non traumatic etiology to be examined for cocaine use[3].

The mechanism through which cocaine causes ischemia and myocardial infarction seems to be complex and multifactorial[6]. The main effect of cocaine is the inhibition of noradrenaline and dopamine re-uptake, at the presynaptic level, causing their excessive accumulation in the metasynaptic receptors, acting, as a potent sympathomimetic agent[1,2]. In the cardiovascular system it causes increase of the heart rate, rise of the arterial pressure and increased myocardial contractility. These three major hemodynamic changes increase myocardium oxygen demand. At the same time, by stimulating a-adrenergic receptors it causes an intense spasm of the coronary arteries, particularly of the epicardiac arteries. Other effects of cocaine that further aggravate arterial spasm include increased endothelin production by the endothelium, which has been known to be a potent vasoconstrictive agent and the reduction of nitric oxide production that is a vasodilating agent[3,6].

The increase in myocardial oxygen demand on the one hand and its reduced offer on the other hand are responsible for myocardial ischemia[3,6]. Another mechanism participating in the mechanism of infarction is the activation of platelets and the creation of intracoronary thrombi[6].

The treatment of choice for the vasospasm is the administration of nitroglycerin, verapamil or a-adrenergic receptors antagonists (fentolamine), while the use of b-adrenergic blockers is contra-indicated, since they usually aggravate the vasospasm. The administration of aspirin is also suggested for the inhibition of platelets agglutination. Benzodiazepines are useful because they reduces the heart rate and the arterial pressure (Table 1)[7].

Table 1. Suggested therapy by the American Society of Cardiology for the treatment of ischemia or myocardial infarction due to cocaine.

First line drugs
Oxygen
Aspirin
Nitroglycerin
Benzodiazepine
Second line drugs
Verapamil
Fentolamine
Thrombolytics or emergency angioplasty (following coronary angiography showing artery occlusion)
Contraindicated
Propranolol

There is only limited experience of thrombolysis in patients with myocardial infarction caused by cocaine.The use of standard ECG criteria (ST elevation) alone for the diagnosis of an acute myocardial infarction in cocaine users is an insufficient factor for determining administration of thrombolytics and their erroneous use has had devastating results due to known complications (major bleeding). Furthermore, the frequent presence of significantly increased arterial pressure, due to cocaine, is another factor that can increase thrombolysis complications[7]. In individuals who were admitted to the emergency department with chest pain following cocaine use, 56 - 84% presented with an "abnormal ECG", while 43% of cocaine users without myocardial infarction fulfilled the ECG criteria (ST elevation) for the administration of thrombolytics[8,9]. The reduced sensitivity of the ECG is partially due to the frequent presence of "early repolarization" because of the young age of the majority of users[8].

Consequently, in accordance with the American Cardiology Society guidelines, thrombolysis is indicated if the first line pharmaceutical treatment fails or if an occluded artery is proven through angiogram[7]. Thrombolysis is contra-indicated in the presence of non-controlled arterial hypertension[7].

Other effects of chronic cocaine use in the heart include accelerated atherosclerosis[6], left ventricle hypertrophy[10], a large variety of arrhythmias and conduction disorders, dilated cardiomyopathy and endocarditis[3,11,12].

Finally, cases of acute aortic dissection have been recorded in cocaine users. The high increase of the arterial pressure[13] has been proposed as the causative mechanism.

The present case is representative of myocardial infarction with normal coronary arteries, caused by cocaine use. Coronary arteries angiography did not reveal any stenosis, while in the anterior descending there was an intra-luminal transparency indicative of vessel thrombosis. A thrombus was observed in the apex of the left ventricle, that had developed in the necrotized area of the myocardium.

The post-infarction course of the patient was uncomplicated. Left ventricular function remained normal and the patient did not present any arrhythmias. Unfortunately, despite our perseverance and efforts to warn the patient of the risks he ran, after a period of probable abstinence, he continued cocaine abuse and died of acute pulmonary edema.

The frequency of recurrences in cocaine addicts is very high even in cases that were optimally treated with drugs, psychotherapy and social rehabilitation programs[14]. It is obvious that the prognosis of patients with cardiovascular manifestations due to cocaine use depends on whether they stop taking cocaine or not.

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