

Cocaine induced prolongation of the QT interval

D Taylor, D Parish, L Thompson, M Cavaliere

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Prolongation of the QT interval is a serious electrocardiogram finding because of its association with torsades de pointes and sudden cardiac death.¹ Both congenital and acquired factors can lead to abnormal lengthening of the QT interval. Six types of congenital long QT syndrome (LQT1–LQT6) have been described, each involving mutations in genes encoding potassium or sodium transmembrane channel proteins.²

Acquired causes of QT prolongation include hypokalaemia, hypomagnesaemia, hypocalcaemia, human immunodeficiency virus infection, and myocardial ischaemia.^{2–4} Numerous drugs have also been found to cause prolongation of the QT interval. A listing of these drugs can be found on a web site (<http://www.qtdrugs.org>). The main membrane channel these drugs affect is the human ether-a-go-go-related gene (HERG) encoded potassium channel; congenital mutations involving this gene lead to the LQT2 type of the inherited long QT syndromes.^{2,5}

Cocaine use has been associated with many cardiac complications including ventricular arrhythmias and sudden death,^{6,7} and cocaine induced torsades de pointes in patients with idiopathic long QT syndrome has been described in two case reports.^{8,9} A case report of cocaine induced QT prolongation (in the absence of congenital long QT syndrome) was published in 1997.¹⁰ In a study of 45 patients (with a history of chest pain, somnolence, or disorientation) admitted to the hospital after cocaine use, Gamouras *et al* showed that the QT interval was increased in patients with and without chest pain and that those with chest pain had greater QT prolongation.¹¹ Cocaine and its metabolites, like many other substances shown to prolong the QT interval, have been shown to block HERG encoded potassium channels.¹²

The patient, after smoking over 200 rocks of crack cocaine in 72 hours, attempted a several mile walk toward a destination at which he was to obtain more money for cocaine purchases. Nearing the end of his walk, the patient developed severe chest pain and shortness of breath and phoned for an ambulance. The patient's chest pain and dyspnea resolved before the ambulance arrived at the hospital. The patient had no medical history and no history of syncope or palpitation. He described himself as being "in good shape" and reported he exercised regularly. He had no family history of sudden death. He was not taking any prescription or over the counter medications and gave a history of occasional alcohol and marijuana use.

Physical examination revealed an anxious African-American man who had an athletic appearance. The rest of the examination was unremarkable. The patient's heart rate was 56 beat/min, and his blood pressure was 123/63 mm Hg. Serum electrolytes were within normal limits, and a urine drug screen was positive for cocaine and negative for other substances. Cardiac iso-enzymes were within normal limits and remained so on serial analysis. An initial electrocardiogram revealed a QT_c* of 621 ms (fig 1). A repeat electrocardiogram two hours later revealed a QT_c of 605 ms. The patient was admitted for observation and was placed on telemetry. The patient's third and fourth electrocardiograms, taken 7 and 15 hours after the initial electrocardiogram, revealed QT_cs equal to 530 ms and 543 ms, respectively. Calculation of QT_c from the patient's telemetry rhythm strips revealed that the QT_c had returned to normal at around 18 hours after the initial electrocardiogram. The patient remained asymptomatic throughout this period and was

CASE REPORT

We present the case of a 37 year old man with a history of chest pain occurring after a three day crack cocaine binge.

*QT_c is the QT interval corrected for the patient's heart rate. $QT_c = (QT) / (\sqrt{RR})$, where RR is the distance between consecutive R waves on the electrocardiogram.

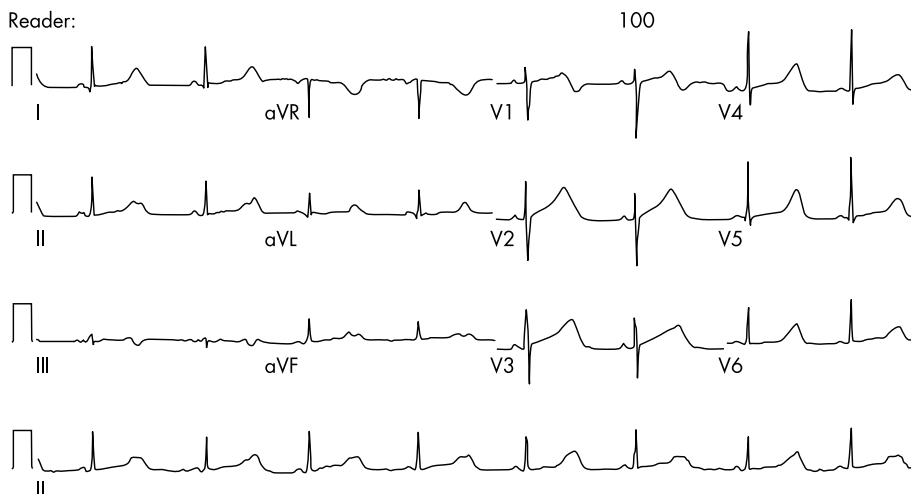


Figure 1 Initial electrocardiogram of the patient.

discharged from the hospital in good condition after appropriate substance misuse counselling. A follow up electrocardiogram, one month later, revealed a normal QT_c interval of 453 ms. The patient stated he had been free of cocaine since discharge.

DISCUSSION

We hope the case we present not only will reinforce the need for close cardiac monitoring in patients who have used cocaine but also will remind physicians that cocaine can exert dangerous electrophysiological effects on the heart that are not necessarily related to the drug's inducement of a hyperadrenergic or cardioischaemic state. It is important to note that our patient, though anxious, was not tachycardic or hypertensive, and there was no evidence of cardiac ischaemia. We also hope that the information presented will encourage physicians to exercise extreme caution in the prescription of QT prolonging drugs to patients who have recently used cocaine or have a history of cocaine misuse.

Authors' affiliations

D Taylor, D Parish, L Thompson, M Cavaliere, Mercer University School of Medicine, Department of Internal Medicine, Macon, USA

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Correspondence to: Dr D Taylor, 3901 Northside Drive, Apt 4 H, Macon, GA 31210, UK; derrickwtaylor@earthlink.net

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Herbal mind altering substances: an unknown quantity?

C Doughty, A Walker, J Brenchley

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Herbal drugs are increasingly marketed as a "safe" alternative to illicit drugs. The variety of constituents in these compounds and their potential pharmacological activity can present difficulties for the emergency physician in management of intoxicated patients. After a case at a recent music festival, we present a case report and review of herbal compounds.

CASE REPORT

A 17 year old woman was brought to the festival medical base by first aid ambulance. Friends said she had taken herbal drugs ("road runners") and alcohol. She was fully conscious, hyperventilating, tachycardic (pulse 155 bpm) with dilated pupils (6 mm). She became increasingly agitated and five minutes after arrival had a grand mal seizure, which was terminated with 5 mg intravenous diazemuls. She was transferred to a local emergency department and on arrival was fully alert, but still tachycardic. She was also noted to have nystagmus at this time. Routine blood tests (full blood count, urea, and electrolytes) were normal. She had no previous history of seizures. After a period of four hours of observation, she was discharged home with a responsible adult.

There is little awareness among healthcare professionals of these drugs and possible side effects of the constituents. Using a research grant from BAEM, a selection of these "herbal" drugs (including the type ingested by our patient) have been analysed and some of the results are presented

below. We also present a review of the ingredients listed on the product information.

CHEMICAL ANALYSIS

The predominant ingredients in the seven products tested are ephedrine and caffeine. Varying quantities of herbal substances may also be present, but cannot be identified on the spectrograph trace using current information. Figure 1 shows the gas chromatography mass spectrometry for the herbal product taken by the patient.

Peaks at 101 and 195 represent ephedrine and caffeine respectively. The peak at 285 is bupivacaine, which is used as a standard. The other peaks correspond to methyl norhexadecanoate (scan no 214), palmitic acid butyl ester (scan no 222), oleic acid butyl ester (scan no 244), and oleic acid butyl ester (scan no 252). On average each capsule contained 16 mg of ephedrine and 0.6 mg of caffeine.

No illicit drugs were found in any of the samples. The drugs tested for are amphetamine and derivatives, ecstasy and derivatives, ketamine, lidocaine, cocaine, methadone, codeine, dihydrocodeine, morphine, diamorphine, papaverine, propoxyphene, narcotine and derivatives, tetrahydrocannabinol and cannabidiol, diazepam, temazepam and flunitrazepam.

INGREDIENTS

Most of these products list an array of herbal constituents, which will be unfamiliar to most healthcare professionals. These include: