Myocardial Ischemia Secondary to Synthetic Cannabinoid (K2) Use in Pediatric Patients

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K2 is a synthetic cannabinoid that has potential cardiovascular side effects, including myocardial ischemia, myocardial infarction, and arrhythmias. Cardiac testing of pediatric patients is often not performed owing to a lack of symptomatology. We report a series of pediatric patients with concern for myocardial ischemia temporally associated with K2 exposure. *(J Pediatr 2015;167:757-61).*

Although epidemiologic data are limited, the reported prevalence of synthetic cannabinoid (K2, spice) ranges between 6.5% and 12.6% in adolescents and adults in the US and United Kingdom.[1] Synthetic cannabinoids are more attractive than cannabis, owing to ease of purchase as well as increased odds of negative urine and blood testing. K2 has multiple known side effects, and serious events, including ischemic stroke, have been reported.[2] Although there are reports of adverse cardiovascular effects secondary to cannabis or synthetic cannabinoid use, including myocardial infarction (MI), arrhythmias, and sudden death in adults,[3-9] data on the cardiac effects of K2 in pediatrics is limited.[10-12] We report a series of pediatric patients who were seen at a tertiary care center over a 2-year period with evidence of varying degrees of myocardial injury secondary to the use of K2.

The Children's National Health System (CNHS) is a tertiary pediatric care center in Washington, DC that sees more than 100 000 visits to the emergency room (ER) annually. After Institutional Review Board approval was obtained from CNHS, the electronic medical record was queried for a combination of K2 use, ST segment changes, and elevated troponin levels. Individual charts were then reviewed to identify patients who used K2 and underwent evaluation for cardiac injury, which included electrocardiogram (ECG), echocardiogram, and laboratory testing. Each ECG represents an official reading from an attending pediatric cardiologist at CNHS.

Case 1

A 15-year-old previously healthy male was brought to the ER secondary to altered mental status. On later questioning, he admitted frequent K2 smoking, including smoking before presentation to the ER. He denied any chest pain, shortness of breath, or palpitations. ECG on admission showed ST elevation in the lateral leads, T wave inversions in the inferior leads, and left ventricular hypertrophy (LVH) based on voltage criteria (**Figure 1**, A). Laboratory testing was notable for an elevated troponin I level at 0.16 ng/mL (normal, <0.1 ng/mL) with normal creatine kinase (CK), CK-MB, and serum and urine drug screening. An echocardiogram revealed normal intracardiac anatomy, normal coronary artery origins, normal biventricular function, no wall motion abnormalities, and no evidence of pericardial effusion. The patient was admitted overnight for observation, and demonstrated a return of troponin I level to normal (0.03 ng/mL) and improved ST segments and T wave inversions (**Figure 1**, B). He was recently evaluated for mild hypertension in the setting of an elevated body mass index. An ECG obtained during that visit was unchanged from his discharge study. He was advised to lose weight.

Case 2

A 16-year-old healthy male was brought to the ER with altered mental status. He reported smoking K2 ("scooby"), but denied any other drug use. He denied chest pain, shortness of breath, palpitations, or dizziness. An ECG was obtained which showed ST elevation in an anterolateral injury pattern with T wave inversions in the inferior leads (**Figure 2**; available at www.jpeds.com). Laboratory testing was notable for a normal troponin I level (0.03 ng/mL) and positive urine drug screen for marijuana. An echocardiogram revealed normal intracardiac anatomy, normal coronary artery origins, normal biventricular function, no wall motion abnormalities, and no evidence of a pericardial effusion. He was monitored in the ER until his mental status returned to baseline and was discharged to home with a plan for outpatient cardiology follow-up. He has not followed up to date.

CK	Creatine kinase
CNHS	Children's National Health System
ECG	Electrocardiogram
ER	Emergency room
LVH	Left ventricular hypertrophy
MI	Myocardial infarction
THC	Delta-9-tetrahydrocannabinol

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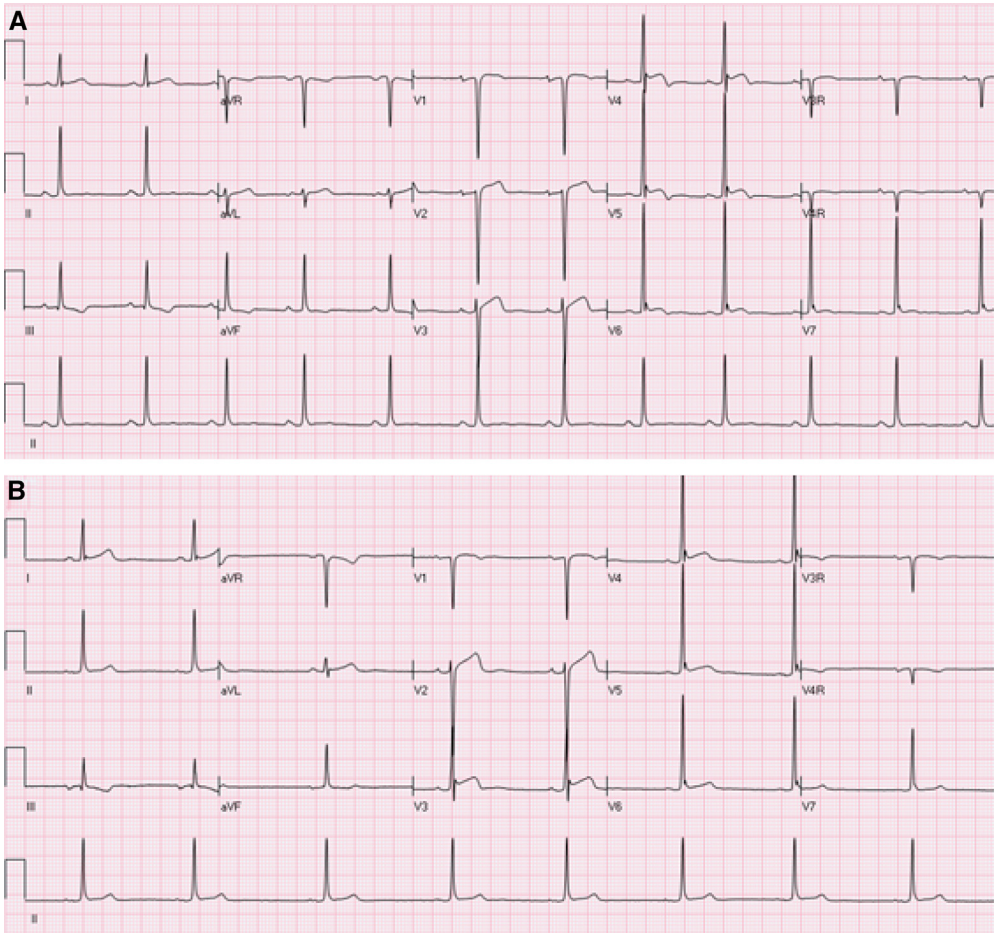

Figure 1. Case 1 ECG on **A,** presentation and **B,** discharge. **A,** Normal sinus rhythm, LVH, ST flattening in the lateral leads, T wave inversion in inferior leads. **B,** Marked sinus bradycardia, possible LVH, ST-T wave elevation in precordial leads, and T wave inversion in lead III.

Case 3

A 17-year-old male with a past medical history of impulse control disorder, depression, developmental delay, and attention deficit hyperactivity disorder presented to the ER with altered mental status and homicidal ideation. He reported smoking 4 cigarettes containing unknown substances, but there was a strong suspicion for K2 ingestion. He had reported a history of chest pain, which had resolved at the time of presentation. His ECG revealed sinus tachycardia, with ST elevation more pronounced in V2 and V3 (**Figure 3**, A; available at www.jpeds.com); there was evidence of RSR' (right ventricular conduction delay) in lead V1 initially concerning for possible Brugada syndrome, but no further workup was done in the absence of previous symptoms and family history. Troponin I level was elevated at 0.39 ng/mL, but normalized within 24 hours. A urine drug screen at admission was negative. An echocardiogram showed normal intracardiac anatomy, normal coronary artery origins, normal biventricular function, no wall motion abnormalities, and no evidence of pericardial effusion. The patient was discharged from the medical service at 36 hours after presentation with an ECG showing ST elevation, though decreased from that seen at presentation (**Figure 3**, B). One month later, the patient presented to the ER for an unrelated complaint, at which time a repeat ECG showed no evidence of ST changes.

Additional Cases

Five patients in addition to the 3 case report patients who presented for evaluation after using K2 had ECG changes concerning for ischemia with normal serum markers (**Table**). All of the patients were adolescent males exhibiting a variety of symptoms at presentation, including chest pain, shortness of breath, syncope, and/or palpitations. Evaluation included ECG, echocardiogram, and laboratory testing. The majority were observed overnight and discharged home the next day. In 1 patient, acute appendicitis was found incidentally on presentation,