Background

Synthetic cannabinoids have become widely used due to their commercial availability, a perceived more intense “high” compared with cannabis, and the fact that they are not detected on routine urine drug immunoassays.

There are several synthetic cannabinoids that are commercially available including those designated as JWH-018, JWH-073, JWH-398, JWH-250, HU-210, and CP-47,497, and its homologues; and oleamide (Figure 1). Synthetic cannabinoids are marketed under a variety of names including Spice, K2, Genie, Sence, Moon Rocks, and Black Mamba among others (Figure 2).

Case Report

A 16 year old male with past medical history of asthma and attention deficit hyperactivity disorder (ADHD) presented to the emergency department (ED) complaining of 24 hours of non-radiating pressure-like substernal chest pain associated with dyspnea, nausea, and vomiting.

Initial vital signs included a blood pressure of 127/57, heart rate of 82, respiratory rate of 22, oral temperature of 98.6° F, and pulse oximetry of 100% on room air. His initial electrocardiogram (EKG) revealed ST segment elevations in leads II, III, AVF, and V4, V5, V6 (Figure 3). The initial troponin was 1.47 ng/ml (normal: 0-0.03 ng/ml) and the initial CKMB was 17.5 ng/mL (normal: 0-7 ng/ml).

The patient eventually admitted to smoking “K2” 60-90 minutes prior to the onset of symptoms.

The patient manifested persistent ST elevations with a peak troponin of 8.29 ng/ml and a peak CKMB of 33.9 ng/mL. The patient also had a urine drug immunoassay that was positive for benzodiazepines and opiates; of note, his urine was tested after receiving lorazepam and morphine during his hospital stay. On hospital day 4, cardiac catheterization revealed a subendocardial myocardial infarction due to coronary spasm with normal coronary arteries.

Discussion

Δ9-tetrahydrocannabinol (THC) has been shown to cause a dose-dependent increase in both heart rate and blood pressure thought to be due to sympathetic stimulation and reduced parasympathetic activity. The proposed mechanisms for cardiovascular events due to marijuana use include an increase in catecholamines, carboxyhemoglobin levels, postural hypotension, increased cardiac workload, and an increase in oxygen demands with a decrease in myocardial oxygen supply. These same mechanisms may increase the risk of myocardial infarction in the setting of synthetic cannabinoid use.

Myocardial infarction has been previously reported in the setting of marijuana and synthetic cannabinoid use. To our knowledge, this is the first report of ST-elevation myocardial infarction in the setting of synthetic cannabinoid use without concomitant marijuana use.

Conclusions

While chest pain is not an uncommon presentation in adolescents, chest pain due to myocardial ischemia is rare in this population. When evaluating patients with chest pain, it is important to elicit a comprehensive drug history, including the use of designer drugs such as synthetic cannabinoids. It is important to remember that commercially available urine drug immunoassays are unreliable in this setting and may not detect synthetic cannabinoids.

References

