Diabetic ketoacidosis (DKA) is an acute, major, life-threatening complication of diabetes mellitus. DKA results from severe insulin deficiency and mainly occurs in type 1 diabetes. DKA may be the first symptom of previously undiagnosed diabetes, but it may also occur in people known to have diabetes as a result of a variety of conditions such as infections, myocardial infarction, stroke, pancreatitis, trauma or poor compliance with insulin therapy. Vomiting, nausea, deep gasping breathing (Kussmaul respiration), pronounced thirst, abdominal pain, malaise, and generalised weakness are typical symptoms (1). Patients may also present with altered consciousness, disorientation, confusion or occasionally coma when the condition is severe (1).

Diabetic ketoacidosis is diagnosed with combination of hyperglycaemia, acidosis and ketonuria. The American Diabetes Association categorises DKA in adults into three stages of severity (Table 1) (1). Treatment includes correction of dehydration, hyperglycaemia, and electrolyte imbalances (1).

Laboratory investigations revealed hyperglycaemia, acidosis and ketonuria. He was admitted to the intensive care unit with a diagnosis of diabetic ketoacidosis. He was not considered a typical case of diabetic ketoacidosis because of the tendency to hypokalaemia, persistent tachycardia and bronchoscopic findings. We learned from his friends that he had used cannabis for a week and used bonsai on the day that he was brought to the emergency service.

**Conclusion:** Diabetic ketoacidosis with prolonged acidosis and tendency to hypokalaemia are investigated for the consumption of synthetic cannabinoids.

**Keywords:** Cannabinoids, diabetic ketoacidosis, hypokalaemia
We report here a 17-year-old male patient with diabetic ketoacidosis after consumption of synthetic cannabinoids. Written informed consent was obtained from the patient’s family.

**CASE PRESENTATION**

A 17-year-old male was admitted to the emergency department with sudden onset of dyspnoea. The report given by his relatives stated that the patient, who tended to fall asleep, had experienced a cough, weight loss and polydipsia for several weeks. There was no history of chronic disease or drug use. Clinical examination revealed a dehydrated male with a heart rate of 140 beats/min, temperature of 37°C and a blood pressure of 160/90 mmHg. He was tachypnoeic and dyspnoeic with a respiratory rate of 28 breaths/minute. The patient’s skin was hot and dry, pupils were isochoric and had no focal neurological deficit. Left lung sounds were decreased with diffuse rhonchi and the abdomen was soft and non-tender; bowel sounds were present in 2 of the 4 quadrants. Electrocardiogram (ECG) showed sinus tachycardia at a rate of 118 beats per minute. Laboratory investigation revealed a metabolic acidosis with arterial blood pH of 6.99 and bicarbonate of 4.1 mmol/L. Arterial pO₂ and pCO₂ levels were 78 mm Hg and 16 mm Hg, respectively. Plasma glucose was 392 mg/dL and 3+ ketones were present in the urine. Potassium was 3.4 mmol/L, and liver and renal function tests were normal. Complete blood count showed an elevated white blood cell count of 31,300 mm³/μL.

The patient was admitted to the intensive care unit with a diagnosis of diabetic ketoacidosis. Intravenous fluids, insulin and potassium replacement and empirical antibiotic treatment was started. Chest computed tomography (CT) was performed due to markedly decreased left lung sounds (Figure 1). Consolidation areas were detected in the posterior segment of the left upper lobe and posterobasal segment of the right lower lobe. Also, the left main bronchus was found to be narrowed at chest CT and bronchoscopy was performed. More pronounced on the left, bilateral oedema and haemorrhage were observed.

The patient was relieved after opening the narrowed bronchus. Bronchoscopic bronchoalveolar lavage culture was negative. The patient’s fluid replacement, intravenous insulin, 20-40 mEq/h potassium and empirical antibiotic treatment was continued. The patient showed persistent hypokalaemia and deep acidosis. The patient was not considered a typical case of diabetic ketoacidosis because of the tendency to hypokalaemia, although an average of 20 mEq/h of potassium was given, with persistent tachycardia and bronchoscopic findings. His relatives were interviewed regarding the use of the toxic substance. It was learned from his friends that the patient had used cannabis for a week and used bonzai on the day that he was brought to the emergency service. Urine drug screens were negative and no blood alcohol was detected. Urine-specific laboratory analysis for cannabinoids metabolites could not performed in our laboratory and the consumption of synthetic cannabinoids could not be confirmed with laboratory analyses.

The patient was in respiratory distress again on the fourth day of admission and granulation tissue that nearly completely obstructed the lumen was found on the upper lobe of the left lung in bronchoscopy. The patient improved after the obstructed lumen was opened. Acidosis, hyperglycaemia and patient respiratory symptoms were improved and oral feeding was initiated on the sixth day of admission. Urine drug screens were negative and no blood alcohol was detected. Urine-specific laboratory analysis for cannabinoids metabolites could not performed in our laboratory and the consumption of synthetic cannabinoids could not be confirmed with laboratory analyses.

The patient was in respiratory distress again on the fourth day of admission and granulation tissue that nearly completely obstructed the lumen was found on the upper lobe of the left lung in bronchoscopy. The patient improved after the obstructed lumen was opened. Acidosis, hyperglycaemia and patient respiratory symptoms were improved and oral feeding was initiated on the sixth day of admission. Urine drug screens were negative and no blood alcohol was detected. Urine-specific laboratory analysis for cannabinoids metabolites could not performed in our laboratory and the consumption of synthetic cannabinoids could not be confirmed with laboratory analyses.

The patient was admitted to the emergency department with massive haemoptysis one day after the polyclinic visit and was not saved despite resuscitation.

**TABLE 1. Diagnostic criteria for DKA**

<table>
<thead>
<tr>
<th></th>
<th>Mild (plasma glucose level &gt;250 mg/dL)</th>
<th>Moderate (plasma glucose level &gt;250 mg/dL)</th>
<th>Severe plasma glucose level &gt;250 mg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial pH</td>
<td>7.25-7.30</td>
<td>7.00 to &gt;7.24</td>
<td>&lt;7.00</td>
</tr>
<tr>
<td>Serum bicarbonate (mEq/L)</td>
<td>15-18</td>
<td>10 to &lt;15</td>
<td>&lt;10</td>
</tr>
<tr>
<td>Urine ketone</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Serum ketone</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Effective serum osmolality</td>
<td>Variable</td>
<td>Variable</td>
<td>Variable</td>
</tr>
<tr>
<td>Anion gap</td>
<td>&gt;10</td>
<td>&gt;12</td>
<td>&gt;12</td>
</tr>
<tr>
<td>Mental status</td>
<td>Alert</td>
<td>Alert/drowsy</td>
<td>Stupor/coma</td>
</tr>
</tbody>
</table>

DK: diabetic ketoacidosis

**DISCUSSION**

Cannabinoids are divided into 3 groups: endocannabinoid, phytocannabinoid and synthetic cannabinoid. They act as
neuromodulators and have effects on motor learning, appetite, pain, emotions, synaptic transmission, memory, immune system, autonomic nervous system and microcirculation (7). Unlike endocannabinoids, synthetic cannabinoids are full agonists with increased potency (3, 6). They have a longer duration of action and their side effects are evident.

“Spice” and “K2” represent two of the more popular brand names that became widely available in the United States (3). Although these products are typically labelled as “incense” and “not for human consumption” or “for aromatherapy only”, they are ingested by smoking (3). In many countries, they are easily accessible and marketed as reliable herbal products.

Severe and life-threatening symptoms may occur due to the consumption of synthetic cannabinoids (Table 2) (3). In the U.S., 11,000 patients per year consult the emergency services because of the side effects of synthetic cannabinoids (6). Synthetic cannabinoids cannot easily be detected in urine and blood studies using conventional methods (6).

Clinical case reports regarding acute intoxication of synthetic cannabinoids describe a variety of somatic and neuropsychiatric effects like nausea and vomiting, tachycardia, hypertension, hypokalaemia, hyperglycaemia, convulsions, agitation, hallucination, and acute psychosis (8, 9). Similarly, our patient had tachycardia, hyperglycaemia, hypertension and hypokalaemia.

Loschner reported a patient with hypoxaemia and haemoptysis after the inhalation of a synthetic cannabinoid and diagnosed diffuse alveolar haemorrhage revealed with bronchoscopy (10). Also, our patient had hypoxaemia and bilateral oedema, with haemorrhage observed on bronchoscopy.

Hermanns-Clausen and his friends reported four cases of acutely intoxicated patients after the consumption of synthetic cannabinoids. They observed a wide spectrum of symptoms including mild hyperglycaemia, hypokalaemia and tachycardia (2). They detected the synthetic cannabinoid metabolites of JWH-018 and JWH-073 by urine laboratory analysis. We could not confirm exposure to synthetic cannabinoids by urine laboratory analysis for cannabinoid metabolites.

Synthetic cannabinoids cannot be detected in the urine and blood using conventional methods and sometimes induce serious side effects. It is recommended that patients with diabetic ketoacidosis with prolonged acidosis and a tendency to hypokalaemia are investigated for the consumption of synthetic cannabinoids.

**Ethics Committee Approval:** N/A.

**Informed Consent:** Written informed consent was obtained from the patient’s parents for the publication of this case report and any accompanying images.

**Peer-review:** Externally peer-reviewed.


**Conflict of Interest:** No conflict of interest was declared by the authors.

**Financial Disclosure:** The authors declared that this study has received no financial support.

### REFERENCES

6. Fattore L, Fratta W. Beyond THC: the new generation of cannabinoid designer drugs. *Front Behav Neurosci* 2011;5:50. [CrossRef]
8. Forrester MB, Kleinschmidt K, Schwarz E, Young A. Synthetic cannabinoid exposures reported to Texas poison centers. *Drug Test Anal* 2011;3:466-78. [CrossRef]