Introduction

Cannabis is derived from Indian hemp plant ‘Cannabis Sativa’. This plant contains over 400 chemical substances, with about 60 responsible for its unique effect. The main psychoactive ingredient is Δ9-tetrahydrocannabinol (THC). The strength of cannabis depends on how much THC it contains. Concentration of THC varies with different preparations like bhang (1%), ganja (1–2%), sinsemilla (up to 6%), hashish (8–14%). Hashish oil is the most concentrated form of cannabis. Its concentration can range from about 15–50%. The patient in this case report had consumed tablets of bhang that was responsible for his vascular complications. Bhang, in our country, has got some sociocultural sanction and therefore, it is widely available and used. Though generally considered to be less harmful, it can have serious adverse effects as is documented in this case report.

Case Report

A 25-year-old male presented with alleged history of ingestion of 10 - 12 tablets of ‘Munakka’ (bhang = cannabis) with alcohol the previous night. Except for alcoholic factor, no other particular odour or colour was found on gastric lavage. On examination, the patient was drowsy, talking irrelevantly, pupils were normal sized and reacting to light, neck was soft, no motor deficit was noted upon verbal command and pain stimulus. BP was 104/70 mm Hg, pulse rate was 74/min irregularly irregular. Except for irregularity of the heart beat cardiac examination was normal. Abdominal and respiratory examination was also normal. ECG on day 1 documented atrial fibrillation with ventricle response rate of 80 beats per min with slight ST coving and T inversion in inferior and lateral leads (Figure 1). Atrial fibrillation resolved without any intervention. Blood investigations were within normal limits. The patient had no history of hypertension, diabetes or any cardiac illness. There was history of frequent use of recreational drugs and occasional use of alcohol. By evening on the same day his blood pressure plummeted to a very low level. Dopamine infusion was started. Hypotension persisted till next day when additional inotropic support with noradrenaline infusion plus hydrocortisone was given. Arterial blood gas analysis revealed metabolic acidosis for which sodium bicarbonate infusion was also started. Despite this support his blood pressure kept fluctuating from as low as 80/60 to as high as 180/100 mm Hg for next few days. The inotropic support was weaned off when the blood pressure got stabilized. Cardiac monitoring documented no new changes except for sinus tachycardia. On 5th day of admission, the patient complained of shortness of breath and chest discomfort. On auscultation the lungs were clear. ECG showed normal sinus rhythm with new onset ST coving with deep T inversion in V1 - V6 and II, III and aVF leads (Figure 2). Quantitative cardiac troponin I level was normal. Echocardiography was normal. The patient was put on antiplatelet drugs. On 7th day the patient developed a drop in the level of consciousness with motor deficit on left half of the body. Neurophysician advised for a non contrast CT of brain which documented an acute infarct in right frontoparietal region (right MCA territory infarct) (Figure 3). Two different vascular episodes necessitated a search for the typical risk factors. Our patient was overweight with BMI roughly around 28 kg/m². Lipid profile was normal. Thyroid function test was normal. Carotid doppler study revealed normal flow pattern. Echocardiography was repeated which documented normal findings. No intracardiac clot was visualized. Subsequent ECGs revealed no progression. The patient was then taken up for coronary CT angiogram which showed slightly decreased ejection fraction of 44% with normal coronary vessels (Figure 4). Later on, a repeat CT scan of brain with contrast showed the same finding of right MCA territory infarct but by then, the left sided weakness had improved considerably. The patient was finally discharged in a stable condition.
Discussion

Bhang is one of the least researched preparations of cannabis. Most of the literature from the west has focused on smoked forms of cannabis (ganja and charas). Discussion, therefore, drives from evidence collected from cases of cannabis smoking. Cannabis smoking is a possible risk factor for myocardial infarction. Cannabis may often be used in combination with other recreational drugs such as cocaine and amphetamines, which may have synergistic cardiovascular effects. The patient in this case report had consumed bhang and alcohol together. Bhang is generally believed to be a relatively less harmful form of cannabis. But again, the strength of cannabis depends on how much THC it contains. Since it is processed in a non-standardized and unauthorized setting, the exact amount of ingested cannabis was could not be determined. Together with alcohol, it must have had synergistic cardiovascular effects. The atrial fibrillation documented on admission followed by shock, later followed by hypotension was possibly related to the biphasic effect of cannabis on the autonomic nervous system. And it was possibly coronary vasospasm in the background of fluctuating blood pressure that produced these ischaemic changes on ECG.

The patient also developed right MCA territory infarct. Wolff V et al had proposed the following mechanisms of stroke relating to cannabis use: orthostatic hypotension with secondary impairment of cerebral blood flow, altered cerebral vasomotor function, labile hypertension cardioembolism with atrial fibrillation, or other arrhythmias, vasculopathy (toxic or with immune inflammatory), vasospasm, reversible cerebral vasoconstriction syndrome (RCVS) or reversible multifocal intracranial stenosis (MIS). They proposed that reversible cerebral angiopathy involving several arteries (MIS), associated with cannabis consumption in association with tobacco and alcohol use, was the most convincing mechanism of ischaemic stroke in young adults consuming cannabis. Zacchariah SB proposed that in patients with ischaemic stroke caused by marijuana smoking, elevation of blood pressure could be a reaction to cerebral vasospasm preceded by hypotension. Our patient had both of these - hypotension preceding hypertension as a cerebral vasospastic response. Potential triggering factors of stroke described in cannabis users are sexual activity, concomitant alcohol consumption or unusually high consumption of cannabis.

Because of the sequestration in fat, the tissue elimination half-life of the lipophilic THC is about 7 days, and complete elimination of a single dose may take up to 30 days. With repeated usage, high levels of cannabinoids can accumulate in the body and continue to reach the brain. This could be a possible explanation for slightly delayed development of the events - cardiac complication closely followed by cerebral complication.

Conclusion

Bhang, in our country, has got some sociocultural sanction and therefore, it is widely available and used. Though generally considered to be less harmful, it can have serious adverse effects as is documented in this case report.

References