

developed CAS secondary to a transdermal hyoscine patch after an anaesthetic.¹⁰

No specific diagnostic studies exist for anticholinergic syndrome and serum drug concentrations are not helpful and rarely available to aid in the initial management. The antidote for anticholinergic toxicity is physostigmine salicylate. However, this drug has potentially serious cardiac side effects and is no longer recommended for use.

Prolonged hyperthermia may lead to devastating multi-organ failure and rapid cooling is essential to reduce the damage caused by excessively high temperatures. CAS has been previously well recognised in the elderly, but has rarely reported in children. Hyoscine patches are commonly used to control symptoms in children with cerebral palsy, often to good effect. The possibility of this syndrome, however, should be considered in any child on anticholinergic medications with a fever or the other symptoms described, particularly in the absence of sweating.

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Cardiovascular complications induced by cannabis smoking: a case report and review of the literature

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Cannabis is generally considered a drug of low toxicity. Although attention has focused on its neuropsychiatric effects, little has been given to cardiovascular side effects. Here we report a case of atrial tachyarrhythmias following cannabis use, and review the literature on its cardiovascular effects and complications.

Cannabis is generally considered a drug of low toxicity. Despite this a variety of cardiovascular complications have been documented. Here we report a case of atrial tachyarrhythmias and review the literature.

A 35 year old Afro-Caribbean female presented with a 1 month history of headaches and was found to be hypertensive with a sitting blood pressure of 179/119 mmHg on average. Her past medical history included polycystic ovary disease. She smoked 20 cigarettes a day and had a history of infrequent cannabis use although none in the year preceding presentation. She denied using any other recreational drugs and was on no regular medication. There was a positive family history of essential hypertension. Clinical examination was unremarkable. She was admitted to hospital for further investigations particularly to rule out secondary hypertension.

Laboratory investigations showed normal urine analysis, biochemistry, thyroid function tests, plasma aldosterone/renin ratio, and 24 hour urinary catecholamines. Abdominal CT scan was normal. A 12-lead electrocardiogram (ECG) suggested left ventricular hypertrophy using the Sokolow and Lyon voltage criteria (R in V5 + S in V1 \geq 35 mm), however, the patient was slim built. An echocardiogram showed

normal ventricular wall thickness and cavity size, good biventricular function, and normal transmitral inflow Doppler profile and atrial dimensions.

The patient was started on amlodipine tablets 10 mg once daily and her blood pressure improved to 159/107 mmHg. Whilst in hospital, she smoked cannabis, and approximately 20–30 minutes later she developed palpitations, chest pain, and shortness of breath. Her blood pressure was found to be elevated at 233/120 mmHg with a pulse rate of 150 beats per minute. An ECG showed a narrow complex tachycardia, which was confirmed as typical atrial flutter with 2:1 atrioventricular block, following the administration of intravenous adenosine. The cardiac rhythm shortly degenerated into atrial fibrillation at a rate of 146 beats/minute. She was treated with intravenous flecanide to relieve significant discomfort and sinus rhythm was promptly restored. Cardiac troponin at 12 hours was normal. Urine toxicology was positive for cannabis but no other recreational drugs were detected. Two weeks post discharge her blood pressure was 117/85 mmHg on amlodipine 10 mg once daily and atenolol 25 mg once daily. A 24 hour ECG Holter monitoring demonstrated normal sinus rhythm. She remains well with excellent blood pressure control and has not smoked cannabis since.

DISCUSSION

The arrhythmogenic potential of cannabis smoking has rarely been reported. It is likely that the real incidence of arrhythmias is substantially underreported given the prohibition of cannabis use. Epidemiological data indicate that cannabis users are significantly more likely to experience palpitations with the majority being dose related sinus tachycardia.^{1,2} Other reported arrhythmias include sinus

bradycardia, second-degree atrioventricular block and atrial fibrillation.³⁻⁵ The onset of arrhythmias can begin within a few minutes of smoking cannabis, reaching a peak within 30 minutes, but can persist for longer than 90 minutes.¹ The cardiovascular effects of cannabis are largely related to its biphasic effect on the autonomic nervous system.⁶ At low or moderate doses, the drug leads to an increase in sympathetic activity and a reduction in parasympathetic activity, producing a tachycardia and increase in cardiac output. Blood pressure therefore increases. At high doses, sympathetic activity is inhibited and parasympathetic activity increased, leading to bradycardia and hypotension. Animal data suggests that the bioactive constituent of cannabis may cause sympathetic inhibition via CB1 receptors on the presynaptic nerve terminals of postganglionic sympathetic fibres.⁷ The influence of the autonomic nervous system in relation to the mechanism of atrial fibrillation has been previously examined.⁸ Both sympathetic and parasympathetic mechanisms have been implicated.

Vagal stimulation reduces action potential duration, shortens the atrial refractory period and produces cellular hyperpolarisation.⁸ The net result is a reduction in the wavelength of atrial activation, predisposing to the re-entrant mechanism of atrial fibrillation.⁸ Vagal induced atrial fibrillation is usually seen in hearts with no obvious structural disease. Adrenergic stimulation reduces action potential duration and alters the electrophysiological characteristics of the atria, favoring automaticity, triggered activity and micro-re-entry. Adrenergic mediated atrial fibrillation is usually seen in patients with structural heart disease often with a triggering event such as drugs, sepsis, or post operatively. Our patient, who used cannabis infrequently, may have had adrenergic induced atrial flutter. The heightened sympathetic response following cannabis use may have destabilized the arrhythmogenic substrate, in this case a hypertensive atrium, initiating atrial flutter. Adenosine, which shortens atrial refractory period, may have been the trigger that induced atrial fibrillation.

Evidence-based guidance for the management of cannabis induced atrial tachyarrhythmias is limited to case reports,^{3,4} but some general principles can be used to guide treatment. Haemodynamically unstable patients require prompt DC cardioversion. In haemodynamically stable patients a period of observation is recommended as the majority of these arrhythmias usually spontaneously revert to sinus rhythm. If the arrhythmia persists then the usual management protocols, as recommended by the American College of Cardiology/American Heart Association and the European Society of Cardiology,⁹ should be followed. Class 1a antiarrhythmic agents (flecainide and propanolol) can be used to terminate these arrhythmias in structurally normal hearts.

Cannabis use has also been associated with premature ventricular contractions,¹ and other reversible ECG changes affecting the P and T waves, and the ST segments.⁶ Although it is not clear if these changes are related to drug ingestion independent of effects on heart rate.

In patients with ischaemic heart disease, cannabis increases the frequency of anginal symptoms at low levels of exercise,¹⁰ owing to an increase in heart rate and myocardial contractility, and a reduction in the oxygen carrying capacity of blood due to the formation of carboxyhaemoglobin.¹¹ These adverse haemodynamic changes may trigger plaque rupture in vulnerable individuals culminating in myocardial infarction,¹¹ and sudden cardiovascular death.¹² Myocardial infarction has also been reported in the presence of normal coronary arteries, suggesting coronary vasospasm.¹² Other reported cardiovascular effects associated with cannabis consumption include transient ischaemic attacks and strokes.¹²

These recent reports confirm that cannabis use alone can result in cardiovascular complications. Furthermore, cannabis may often be used in combination with other recreational drugs such as cocaine and amphetamines, which may have synergistic cardiovascular effects.⁶ A careful recreational drug history should always be elicited, particularly when associated with unusual cardiovascular presentations and altered mental states.

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CORRECTION

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In the case report titled, Left flank pain as the sole manifestation of acute pancreatitis: a report of a case with an initial misdiagnosis (*Emerg Med J* 2005;**22**:452-3) the affiliation for Dr Jiann-hwa Chen was incorrect. The correct affiliation is Department of Emergency Medicine, General Cathay Hospital-Taipei, Taiwan, ROC.