

Anaesthetic Consideration in a Cannabis Addict

Karima Karam, Shemila Abbasi and Fauzia Anis Khan

ABSTRACT

This case report describes the anaesthetic management of a patient addicted to *Bhang* (cannabis) for 20 years. Cannabis use has implication in the anaesthetic management of a patient because of its effects on the cardiovascular system, respiratory system, and central nervous system and its interaction with anaesthetic drugs.

Key Words: *Anaesthesia. Illicit substance abuse. Cannabis.*

INTRODUCTION

Bhang or cannabis users pose a challenge for the anaesthetist and it is important that its action on different body system and possible interaction with the anaesthetic agents are understood. Cannabis affects almost every system of the body, particularly the central nervous system, cardiovascular, respiratory and immune systems. According to the technical summary report 2013 of Ministry of Narcotics Control Pakistan Bureau of Statistics, cannabis is the most commonly used drug in Pakistan and 3.6% of adult population is addicted to cannabis.¹

In this case report, we describe the anaesthetic management of a patient who was a cannabis addict along with a review of the pertinent literature.

CASE REPORT

A 35 years old, 60 kg ASA II, male was scheduled for an emergency fasciotomy following right leg fracture. He had a history of road traffic accident 2 days before his admission to the hospital. His past history was unremarkable except that he had been smoking 5 - 6 cigarettes per day for the last 20 years. He was also a chronic user of cannabis and had been smoking it 3 to 4 times per week, for the last 20 years. He had not used cannabis since the accident. He did not admit to any other addiction besides this.

On general physical examination, he had tachycardia of 130 beats/minute. Other baseline hemodynamic parameters were blood pressure, 126/84 mmHg, temperature 37°C and oxygen saturation 99% on room air. Rest of the physical examination was unremarkable. Airway assessment revealed poor oral hygiene and

Mallampati class I airway. Baseline laboratory values showed a haemoglobin value of 12.8 g/dl, hematocrit of 36.8%, creatinine of 1.2 mg/dl, sodium of 136 mmol/l, potassium of 4.5 mmol/l, chloride of 99 mmol/l and bicarbonate of 26.0 mmol/l. Liver function tests were normal.

Before induction of general anaesthesia, standard monitoring (three lead ECG, oxygen saturation probe, noninvasive blood pressure monitor) were applied. Anaesthesia induction was performed with propofol 2 mg/kg, atracurium 0.6 mg/kg and morphine 0.1 mg/kg (initial dose of 6 mg at induction). Trachea was intubated with size 8.0 polyvinylchloride tracheal tube and patient's lungs were ventilated on controlled mode with tidal volume of 6 ml/kg, respiratory rate of 12 breaths/minute and FiO₂ of 0.4. Anaesthesia was maintained with a mixture of oxygen/nitrous oxide in a ratio of 40:60. Isoflurane concentration was kept at 1 MAC and morphine boluses of 1 mg as required. Infusion of paracetamol 1 gram IV was given as co-analgesic. Additional analgesic requirement was assessed, based on more than 20% rise in blood pressure and heart rate compared to baseline as well as presence of any lacrimation and sweating. His requirement for morphine was seen to be more than average. He received a total of 10 mg of morphine over 2 hours of surgery. In addition to morphine, he received ketorolac 30 mg as a bolus dose. He underwent above knee amputation because of non-viability of the limb. Intraoperative blood loss was approximately 800 ml, which was replaced with a combination of crystalloids and colloids. Rest of anaesthesia was uneventful.

In the recovery room, patient was comfortable, and vital signs were stable. He was transfused one unit of packed cell in the recovery room. He was started on morphine infusion 2 - 3 mg/hour for postoperative pain management with infusion of paracetamol 1 gram 6 hourly as co-analgesic. He received a total of 72 mg morphine in first 24 hours postoperatively and 48 mg on second postoperative day, which showed an increased requirement for opioids.

Department of Anaesthesiology, The Aga Khan University Hospital, Karachi.

Correspondence: Prof. Fauzia Anis Khan, Department of Anaesthesiology, The Aga Khan University, P. O. Box 3500, Stadium Road, Karachi-74800.

E-mail: fauzia.khan@aku.edu

Received: January 09, 2014; Accepted: August 21, 2014.

DISCUSSION

Bhang (also known as *Cannabis indica*) is a mild preparation of marijuana made from young leaves and stems of the cannabis plant. In Pakistan, *Bhang* grows wild all over Potohar plateau, Khyber Pukhtunkhwa province and northern areas. According to an estimate, in Pakistan, almost 5.8% of the adult population and approximately 6.4 million person or one in every 27 person is using drugs while nearly 25% of youth are involved in some form of drug abuse.¹ Cannabis is easily available and its addiction is common especially in some rural areas. It is, therefore, important that history of drug abuse is sought from patients at time of pre-operative assessment and if there is history of cannabis use then the anaesthetist should ask about the frequency of use and the time since last use.² There is a possibility that because of social implications patient may be reluctant in disclosing this fact. This patient did reveal his history of smoking *Bhang* for the last 20 years.

There are several anaesthesia related concerns in these patients mainly due to the effects of Tetra Hydrocannabinol (THC) and its metabolites, and two cannabinoids; cannabidiol and cannabitol, on the different body systems. The tissue elimination half-life of THC is approximately 7 days and the total elimination may take up to 30 days.³ The main concern are its effects on the cardiovascular system. Low to moderate doses increase sympathetic activity and decreases parasympathetic activity and cardiac output.⁴ In high doses it depresses the sympathetic system and can cause hypotension. Chronic use of cannabis can lead to bradycardia as THC causes depression of calcium dependent ATPase activity in cardiac muscle.⁵

ECG of drug abuser may show premature ventricular contraction, reversible ST and T wave changes or decreased voltage of P-waves.^{2,6} THC augments the effects of drugs that cause cardiac depression and addition of potent inhalational agents may lead to further myocardial depression. This patient did not have a pre-operative ECG done but had tachycardia (130 beats/minute) on pre-operative examination. His surgical pathology could be contributory to this tachycardia but it did not entirely explain it as the patient was afebrile.

Addiction to cannabis smoking can also lead to chronic cough, airway irritability, wheeze and bronchitis.^{4,5} There have been reports of upper airway oedema particularly uvular oedema in these addicts to the extent that airway obstruction may occur.⁷ Some authors recommend prophylactic use of dexamethasone.⁸ Early morning sputum production is 144% greater in cannabis smokers than non-smokers.⁹ This patient did not give a history of any respiratory problems.

Cannabis use has implications for use of anaesthetic drugs particularly barbiturates, opioids, prostaglandin

inhibitors, benzodiazepines and phenothiazine.^{4,10} Cannabitol is a potent prostaglandin inhibitor.² The effect of above mentioned drugs can be potentiated in an addicted patient. We did not use barbiturates in this patient and he received propofol. There was no increase in the duration of action of this drug in this patient.

An observation in this patient was the increase in narcotic requirements both intra and postoperatively. He received a total of 10 mg of morphine (approximately 0.15 mg/kg) over a period of 2 hours, in addition to one gram of paracetamol and 0.5 mg/kg ketorolac intraoperatively. His postoperative narcotic requirement was 72 mg in the first 24 hours (approximately 3 mgs/hour) despite receiving paracetamol 1 gram six hourly regularly. This was nearly double the requirements of an average patient of same body weight and height. The trend for higher requirement for morphine continued into the second postoperative day.

As far as use of muscle relaxants is concerned, THC depletes acetyl choline stores and has an anticholinergic effect. It will, therefore, cause potentiation of non-depolarizing muscle relaxants. Atracurium was used in this patient which did not cause any problem related to reversal of neuromuscular block.

Serum levels of cannabis could not be determined in this patient due to cost implications. It is acknowledged as a limitation of this report.

REFERENCES

1. Yaqub F. Pakistan's drug problem: World report. *The Lancet* 2013; **381**:2153-4.
2. Dickerson SJ. Cannabis and its effect on anesthesia. *AANA J* 1980; **48**:526-8.
3. Cho CM, Hirsch R, Johnstone S. General and oral health implications of cannabis use. *Aust Dent J* 2005; **50**:70-4.
4. Hernandez M, Bimbach DJ, Van Zundert AA. Anesthetic management of the illicit-substance-using patient. *Curr Opin Anaesthesiol* 2005; **18**:315-24.
5. Kumar RN, Chambers WA, Pertwee RG. Pharmacological actions and therapeutic uses of cannabis and cannabinoids. *Anaesthesia* 2001; **56**:1059-68.
6. Kuczkoroski KM. Marijuana in pregnancy. *Arm Acad Med Singapore* 2004; **33**:336-9.
7. Mallat AM, Robertson J, Broch-Utne JG. Perioperative marijuana inhalation: an airway concern. *Can J Anaesth* 1996; **43**:691-3.
8. Symons IE. Cannabis smoking and anaesthesia. *Anaesthesia* 2002; **57**:1142-3.
9. Taylor DR, Poulton R, Moffitt TE, Ramankutty P, Sears MR. The respiratory effects of cannabis dependence in young adults. *Addiction* 2000; **95**:1669-77.
10. Ashton CH. Adverse effects of cannabis and cannabinoids. *Br J Anaesth* 1999; **83**:637-49.

