ORIGINAL RESEARCH PAPER

Anaesthesiology

CHALLENGES IN ANAESTHETISING PATIENTS WITH SUBSTANCE ABUSE

KEY WORDS: Substance Abuse , Anesthesia .

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ABSTRACT

Substance abuse is a rising concern world over and administering anesthesia to substance abusers has a variety of concerns and anesthetic implications. It is our humble attempt to describe anesthetic management for upper GI endoscopy after tactfully elicited history of substance abuse.

INTRODUCTION

Cannabis addiction is of major concern in the young population, world over. Such patients coming for surgery or procedures requiring anesthesia pose a challenge to the anesthesiologists. History of drug abuse has to be thoroughly and tactfully elicited in order to plan and execute anesthesia for such patients as cannabis and its different forms have a significant impact on the autonomic stability of the patient and on dosages and interaction with anesthetic agents used for general anesthesia or sedation.

The anesthetic challenges that were faced during the endoscopic procedure on a 29 year old male would be discussed.

Our case was challenging as a thorough pre anesthetic evaluation was done ,history was elicited and case was induced, during the procedure after induction, post failure to attain adequate depth ,history was elicited again and then attenders gave history of cannabis addiction which was not given prior due to taboo associated with it and then case was managed successfully due to adequate know how on management and risk anticipation

Case Study

A 29-year-old male patient, an autorickshaw driver by profession, presented to the emergency with history of accidental ingestion of a foreign body- a cigarette lighter. He was posted for emergency retrieval of foreign body under endoscopic guidance by the medical gastro-enterologist.

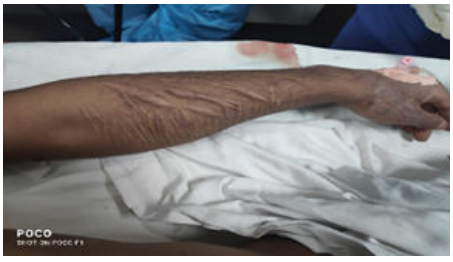
Pre-anaesthetic evaluation: The patient had no known comorbidities. He had a history of accidental burns at the age of 13years, leading to burns of the neck and upper chest, developing contractures of the same thereafter.

[Picture 1]

History of occasional smoking and consumption of alcohol was elicited, which was assumed to be common among this age group.

Examination of the patient revealed a difficult airway due to the neck contracture. Mallampati Grade 2. Minimal neck extension was possible., with reduced thyromental distance. Dentition, neck circumference, mouth opening were normal.

General physical examination and Systemic examination were unremarkable. The patient was conscious, oriented and co-operative. He had multiple hesitation marks on his left forearm, indicating previous suicidal attempts.



[Picture 2]

Investigations were normal. NPO status was confirmed, and the patient was prepared for the procedure as ASA 2E

Baseline vitals recorded were within normal limits with sinus bradycardia 48bpm, BP: 120/80mmHg, Spo2 on room air 99-100%.

The preparation for the case was done mainly with **difficult airway** in mind, with ENT consultant scrubbed in and ready for emergency tracheostomy SOS. Difficult airway cart was prepared with 2 working laryngoscopes, Mc Coy blade, video laryngoscope, suction, endotracheal tubes of adequate size and airways.

Inj. Dexmedetomidine 1mcg per kgBW was infused over 20 min prior to the procedure. The patient was premedicated with Inj Midazolam 1mg, Inj Glycopyrrolate 0.2 mg, Inj Ondansetron 4mg IV and 2mcg per kgBW of Inj Fentanyl intravenously. After preoxygenating the patient with 100% O2 for 3 minutes, an induction dose of 2mg per kgBW of Inj Propofol was given. The drugs did not have any effect on the patient. Half the dose of Inj Propofol was repeated again. Aliquots of 30-50mg Inj Propofol and Inj Ketamine were given (Total of Inj Propofol 320 mg, Inj Ketamine 150 mg, Inj Fentanyl 150mcg). Even with all the above medication, almost 3 times the dose required for induction in patients of similar age group, the patient was still awake and un co-operative for the procedure.

A thorough probing with explanation of the problem we were facing, revealed a significant history of chronic drug abuse, since the age of 13 years, with different forms of Cannabis- Hashis, Ganja, Charas and HCV positive status . This history corroborated the HCV positive status . Inj Propofol 50mg, Inj Fentanyl 50mcg was repeated and Inj Succinlycholine 100 mg was given, and at the 3rd attempt, the patient could be intubated successfully, with a Macintosh size 3 blade, with bougie. The patient was maintained on Inj Dexmedetomidine 0.5mcg/kg/hr, and Inj Propofol 1 mg/kg/hr, and Inj Atracurium with air and oxygen. The procedure was completed, with unsuccessful retrieval of the lighter, due to oedema at the crico pharynx. Decision to electively ventilate the patient was taken, keeping in mind the difficult airway, and probability of the procedure being repeated the next morning.

[Picture 3]

DISCUSSION

Cannabis is the most widely used drug, branded illegal in India (1). Cannabis sativa, a plant that grows mostly in temperate and tropical regions, is commonly ingested by smoking (2). Cannabis/Marijuana consists of more than 421 components and 60 pharmacologically active cannabinoids. The two best-described cannabinoids are THC- Tetra Hydro Cannabinol and cannabidiol (CBD) (7). Marijuana is highly lipophilic which leads to its storage in adipose tissue, liver, muscle and spleen and redistributed into the users blood stream long after ingestion. Because of the persistence in the body, marijuana can cause highly potent mental, physical and toxic effects in the users that are hard to control or predict (8).

Marijuana's main sites of action are in the brain and the spinal cord. It binds to two types of G-protein-coupled receptors, CB1 and CB2. CB1 receptors are predominantly expressed in the brain and located in the basal ganglia, cerebellum, hippocampus, association cortices, spinal cord and peripheral nerves (9)

Both cannabinoid receptors, CB1 and CB2, are G-protein coupled and become activated through inhibition of the adenylate-cyclase. The activation of these receptors cause an inhibition of the release of the neurotransmitters acetylcholine and glutamate while indirectly affecting y-aminobutyric acid, N-methyl-D-aspartate, opioid and serotonin receptors. The cannabinoid receptors are predominantly located presynaptic rather than postsynaptic which means that cannabinoids modulate the neurotransmitter releases (10). Most of our anesthetic agents act through the very same neurotransmitters.

Acute Exposure to Cannabis(Intoxication):

Anesthesia during acute exposure with excitatory drugs is dangerous and should be avoided until the acute effects have disappeared. It may enhance the sedative-hypnotic effects of drugs that depress the CNS. Studies have shown cross tolerance of marijuana/ cannabis with barbiturates, opioids, benzodiazepines, and phenothiazines (4) . Additive effects of marijuana and potent inhaled anaesthetic can result in pronounced myocardial depression in general anaesthesia (5) . Drugs that increase heart rate such as atropine, ketamine, pancuronium and ephedrine should be avoided. Barbiturate and ketamine sleep times are prolonged in THC-treated animals, and opioid induced depression of ventilation may be potentiated (6).

Chronic Exposure To Cannabis (Addiction):

Due to a strong parasympathetic response and baroreceptor deregulation, significant bradycardia, postural hypotension and rarely coronary vasospasm leading to sudden cardiac deaths is seen in chronic users. Unlike in acute intoxication, where the CNS is more affected than the other systems, in chronic cases, the respiratory system is affected more. Increased bronchial tone, Upper airway oedema, Pulmonary embolism and pulmonary embolism is seen.

In effect, after acute intoxication, the anaesthetic dose is decreased and after chronic usage of cannabis, the anaesthetic dose is to be increased.

Table A. Main physiologic effects of cannabinoids(11)

System	CB-	Physiologic effects	
Cardiovascular	CB1-R	Newly users, naïve, SCB	a) Dose-dependent; initial β-adrenergic effect + parasympathetic inhibition: ↑ HR, ↑ LV contractility, ↑ CO, ↑ SBP, VPCs, AFib, malignant arrhythmias (VTach, VFib, Brugada pattern) b) After 30 min of exposure: Norepinephrine levels peak and stay elevated up to 120 min after cessation
		Chronic and/or heavy-users (THC ≥ 10 mg)	a) Strong parasympathetic response + baro reflexes deregulation: ↓ HR, postural hypotension (not compensated by sympathetic stimulation); cardiac arrest b) Coronary spasm in patients with coronary disease → MI c) MI in young individuals due to an increase in MVO_2, high levels of CO Hb, and coronary thrombosis
	VR	Heavy users (THC ≥ 10 mg)	Mesenteric vasodilation through release of CGRP
Cerebrovascular	CB1-R	Naïve users or Low-dose THC	Vasodilation and ↑CBF
		Chronic use, heavy "spice" or K2 consumption	Cerebral vasospasm → ischemic stroke (posterior cerebral circulation affected in half of the cases)
Respiratory	CB1-R	Chronic or heavy user	a) ↑ bronchial tone → bronchial hyper reactivity b) Pharyngeal and uvular oedema → upper airway obstruction c) Diffuse alveolar haemorrhage and necrotizing bronchiolitis → pulmonary oedema d) Pulmonary embolism (more common with SCB)
Temperature	CB1-R	Chronic use, heavy smoking, SCB	Altered central thermoregulation → intraoperative hypothermia → severe postoperative shivering
Coagulation	CB1-R?	Chronic or heavy user	a) Increased clotting time b) Decreased platelet count c) Increased risk of bleeding in patients taking warfarin

CB-R: cannabinoid receptor; CB1-R: cannabinoid receptor type 1; HR: heart rate; LV: left ventricle; CO: cardiac output; SBP: systolic blood pressure; VPCs: ventricular premature

contractions; AFib: atrial fibrillation; VTach: ventricular tachycardia; VFib: ventricular fibrillation; THC: tetrahydrocannabinol; MI: myocardial infarction; MVO_2: myocardial oxygen consumption; COHb: carboxyhemoglobin; CGRP: calcitonin gene-related peptide; VR: vanilloid receptor; CBF: cerebral blood flow; SCB: synthetic cannabinoids.

In Chronic users, cross tolerance with barbiturates, opioids and benzodiazepines is seen, which explains the requirement of increased dose of anaesthetics. Synergism with volatile anaesthetics also is seen ion all other chronic of cannabinoids. There are a limited number of small studies and case reports suggesting a greater risk of complication related to anaesthesia or higher tolerance to procedural-related sedation amongst cannabis users (12, 13). Plausible hypotheses regarding a propofol-cannabis interaction include down-regulation of the cannabinoid (CB)-1 receptor in chronic cannabis users versus partial agonism/antagonism at the CB-1 receptor by other phyto cannabinoids in marijuana products that may compete with propofol, increasing the required dose (14). A study done in 2009 by Flisberg et al shows that cannabis use increases the propofol dose required for satisfactory clinical induction when inserting a laryngeal mask (15). A study to evaluate cannabinoid-glutamate interactions in humans by researchers in Yale university, is to be completed by Dec 2021, would throw more light on the safety and tolerability of the combination of NMDA antagonist, ketamine, and the cannabinoid, delta-9-tetrahydrocannabinol (THC)(16).

CONCLUSION

The importance of accurate history taking cannot be over-emphasized. The symptoms, signs and investigations put together aid in diagnosis. The eyes can see only what the mind knows. Missing out on obvious pointers may lead to disastrous consequences

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