e - ISSN - 2349 - 8005

INTERNATIONAL JOURNAL OF ADVANCES IN CASE REPORTS

Journal homepage: www.mcmed.us/journal/ijacr

KETAMINE-INDUCED PRIAPISM IN A YOUNG NIGERIAN PATIENT

Iyalomhe GBS*

Associate Professor and Head, Department of Pharmacology and Therapeutics, College of Medicine, Ambrose Alli University, Ekpoma, Nigeria/Medical Director, Osigbemhe Hospital, Auchi, Nigeria.

Corresponding Author:- **GBS Iyalomhe**
E-mail: goddyiyalo@yahoo.com

Article Info
Received 15/11/2014
Revised 27/11/2014
Accepted 02/12/2014

Key words:
Ketamine, Intravenous anesthesia, Priapism.

ABSTRACT
Ketamine is widely used as a sole general anesthetic agent in Nigeria, although the data on its use and the associated side effects are scanty. This paper presents a case of priapism developed following intravenous ketamine anesthesia. Literature on complications of ketamine use is reviewed; the treatment of drug induced priapism is discussed and the need to be alert and cautious in the use of this drug as a single anesthetic agent is emphasized.

CASE REPORT

MA was a 26-year old student of Auchi Polytechnic Auchi in Edo State of Nigeria who presented at Osigbemhe Hospital, Auchi, Nigeria, on 25th January, 2013 with the complaint of a painless lump in the right arm of three years' duration. He neither was on any routine drug nor was involved in the use of illicit drugs of abuse. He denied using any aphrodisiac or penile injections. He had no fever. There was no history of trauma to the perineum or spinal cord. He was not a sickler.

On general physical examination, he looked healthy. His vital signs were Temperature 36.8^{o}C (armpit); Radial Pulse 68 beats/min; Respiration 22/min; Blood pressure (BP) 110/60 mmHg and Weight 65kg. Systemic examination was unremarkable except for the presence of a lipoma (10cm x 7.5cm) at the anterior lower third of the right arm. He was admitted for a lumpectomy. Urinalysis and hematocrit were normal. Since the patient refused local anesthesia, the operation was done using intravenous ketamine hydrochloride (manufactured by RotexmedicaTrittan Germany on April 2010, expiry date April 2014; NAFDAC No 04-7218); at a dose of 1.5mg/kg body weight given as a bolus dose for induction of anesthesia after premedication with intravenous atropine sulphate 0.5mg and diazepam 10mg. At surgery, a yellowish lobulated fluctuant mass was excised. Histological diagnosis of the specimen was consistent with the diagnosis of a lipoma.

The patient developed a rock-hard penis with a soft glans (priapism) 3 minutes after the administration of ketamine and this continued for the next 5 hours. Ice packs were applied to the penis and perineum, and detumescence occurred within an hour. Intravenous fluids, antibiotics (gentamicin and ampiclox) and analgesics were administered. Three days post-operatively the patient was discharged to continue out-patient treatment. He was followed up for 6 months with no complaint of reoccurrence of priapism.

DISCUSSION

Ketamine (Ketalar^{R} or Cl-581) is an aryl-cyclohexamine, a derivative of phencyclidine with two optical isomers. The commercial preparation is a racemic

mixture which is water soluble and available in sodium chloride solution plus the preservative benzathonium chloride. It is hepatically metabolised to norketamine, dehydronorketamine and other metabolites which are excreted in urine and bile. Peak plasma levels occur within one minute of intravenous injection and plasma half-life is 79 minutes. Since it has a large volume of distribution and rapid clearance, it is suitable for continuous infusion or intermittent parenteral administration but notorious for having a high potential for drug interactions and cumulative effects [1-3]. Although the actual mechanism of action of ketamine is unknown, earlier studies by Domino [4] suggested that it causes a dissociation of thalamic function and limbic system activity from neocortical function, thus producing dissociative anesthesia with very minimal depression of the reticular activating and the limbic systems [5].

A number of complications or side effects have been reported following ketamine use, chief among which are cardiovascular, gastrointestinal and cerebrovascular effects. Specifically, ketamine use has been associated with excessive vasopressor activity leading to hypertension and tachycardia, pronounced salivation and exaggerated reflex activity. It also exhibits a cumulative action after multiple supplemental doses; it causes increased intracranial pressure, nystagmus with pupillary dilation, purposeless involuntary movements with increased overall muscle tone (often mistaken for anesthetic excitement), emergence delirium, confusion and prolonged recovery. Metabolic and endocrine changes reported include moderate to profound increase in blood sugar, elevated cortisol levels and reduced triiodothyronine (T3) levels [6]. Changes in renal or hepatic function have not been reported. Of these side effects, post-anesthetic psychotomimetic effects have been the main cause of reluctance amongst anesthesiologists to accept the use of ketamine [7, 8]. Nevertheless, this complication can be mitigated by the concomitant use of diazepam, chlorpromazine or haloperidol.

In the case reported, ketamine was associated with priapism- the occurrence of a persistent, usually painful, erection of the penis for more than 4 hours unrelated to sexual stimulation or desire [9]. Although it is hard to establish a cause effect relationship, it is important to realise that ketamine-induced cataleptic state is accompanied by spontaneous limb movement with increased overall muscle tone and vasodilating activity, both of which could predispose to priapism especially where there is an imbalance between vasodilation and ketamine-induced indirect sympathomimetic action. Priapism may well be due to unusual response because the patient in question had no other ailment eg sickle cell disease that could be implicated. He never used any of the drugs that are reported to induce priapism: papaverine, antipsychotics including thorazine and chlorpromazine; antidepressants like trazodone; prostaglandin E1, androstenedione, citalopram, cocaine, marijuana and ethanol. So, to knowledge, this is the first report of priapism complicating ketamine in humans.

Amongst Africans nay Nigerians, a high premium is placed on sex and procreation. The observation of priapism, therefore, in a young Nigerian following ketamine use sounds a note of alarm, for apart from being a psychological and emotional insult; the problem is a true urologic emergency which, if not well managed, could lead to permanent erectile dysfunction, necrosis or total loss of the penis. The goal of treatment of priapism is to make the erection subside and preserve erectile function. The faster detumescence occurs the better the outcome. It is therefore, crucial to develop a high index of suspicion, to be alert and know when priapism occurs in patients undergoing surgery after ketamine anesthesia.

The modalities of treatment include the application of ice packs to the penis and perineum; intracavenous penile injection of alpha adrenoceptor agonists including epinephrine or oral ingestion of pseudoephedrine and terbutaline which may act to decrease blood flow to the penis; needle aspiration to drain blood from the penis and reduce pressure and swelling; and the use of a surgical shunt. Since infection is common, antibiotics are indicated. Analgesia and intravenous fluids must be given liberally.

This report raises vital issues about the use of ketamine as a sole anesthetic agent. It is important to establish conditions in which ketamine anesthesia is safe. There is need for studies to document the existing scope of use, dosing and administration with prospective data on complications. Such information will be crucial for the provision of safe anesthetic services in developing countries where anesthetic materials and manpower are perennially inadequate, thus contributing significantly to high patient mortality [10-12].

REFERENCES

1. Zsigmond EK, Domino EF. (1980). Ketamine clinical pharmacology, pharmacokinetics and current clinical use. *Anesth Rev*, 7,13-33.
2. Patel PM, Patel HH, Roth DM. (2011). General anesthetics. In, Brunton LL, ed. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 12th Edition, McGraw Hill Medical, New York, 538-539.
3. White PF. (1982). Ketamine- its pharmacology and therapeutic uses. *Anesthesiology*, 59, 294-300.
4. Domino EF. (1965). Pharmacologic effects of Cl-581, a new dissociative anesthetic in man. *Clin Pharmacol Ther*, 6,276-291.
5. Longo DL, Kasper DL, Jameson JL, Hauser SL, Fauci AS, Loscalzo J, eds. (2012). Harrison's Principles of Internal Medicine, 18th Edition. McGraw Hill Medical, New York, 2211.

6. Greenstein B, Greenstein A. (2007). Concise Clinical Pharmacology, Pharmaceutical Press, London, 150.
7. Otoide VO, Omuemu C, Ojobo S. (2001). Elevated serum glucose levels following ketamine intravenous anesthesia. *Int J Obst Anesth*, 10, 206-208.
8. Wilson RO, Richey JO, Forster JE, Hendricksen MH. et al. (1979). Cardiovascular effects of ketamine drip. *Anesthesiology*, 51,835-837.
9. Bertram RA, Webster GD, Carson CC. (1985). Priapism, etiology, treatment and results in series of 35 presentations. *Urology*, 26, 229-232.
10. Adesunkanmi ARK. (1997). Where there is no anesthetist, a study of 282 consecutive patients using intravenous, spinal and local infiltration anesthetic techniques. *Trop Doc*, 27,82-88.
11. Leppaniemi AK. (1991). Where there is no anesthetist. *Br J Surg*, 78, 245-246.
12. Alufohai EF. (2000). Coping with Rural Surgery, Sam Bookman Publishers, Ibadan, 14.