

Intraoperative Management of a Patient for Deep Brain Stimulation with Severe Dyskinesia and Tremors: Ketamine to the Rescue!

Abstract

The loss of dopaminergic neurons from the substantia nigra pars compacta characterizes the classical pathology of Parkinson's disease (PD). Deep brain stimulation (DBS) has become an increasingly common treatment for PD. Sometimes excessive tremors due to exacerbated PD hinder the surgery and may almost make it impossible. This is a case report highlights use of IV ketamine for intraoperative sedation of a patient with PD, with severe dyskinesia & tremors, posted for DBS. IV ketamine resulted in prompt abolition of tremors and dyskinesia, which were unresponsive to previous traditional sedative drugs.

Keywords: Deep brain stimulation, dyskinesia, ketamine, Parkinson's disease, sedation

Introduction

The loss of dopaminergic neurons from the substantia nigra pars compacta characterizes the classical pathology of Parkinson's disease (PD).^[1,2] However, persistent activation of glutaminergic N-methyl-D-aspartate receptors has been implicated as a major component in the pathogenesis as well.^[3] Symptoms of PD include slowness of movement, tremor, rigidity, and loss of postural balance.^[4] Deep brain stimulation (DBS) has become an increasingly common treatment for PD. Currently, none of the anesthetic technique is superior to others for neurostimulator implantation, although awake and sedative techniques are preferred as they facilitate simultaneous neurological testing. Sometimes, excessive tremors due to exacerbated PD hinder the surgery and may almost make it impossible. This is a case report regarding the use of intravenous (IV) ketamine for intraoperative sedation of a patient with PD, with severe dyskinesia and tremors, posted for DBS. IV ketamine resulted in prompt abolition of tremors and dyskinesia, which were unresponsive to previous traditional sedative drugs.

Case Report

A 63-year-old 60 kg female patient was posted for deep brain stimulation (DBS).

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She had severe Parkinson's disease (PD) with bilateral tremors, dyskinesia, and dystonia secondary to disease progression and treatment complications. She had a prior history of DBS in 2003, followed by improvement in signs and symptoms. Currently, she presented with the complaints of resting tremor (right > left), rigidity, bradykinesia, and increased tone in all the four limbs. She had no other systemic illness. Her medications at the time of admission included: tablet syndopa 110 mg ½ five times a day, tablet pacitane 2 mg TDS, and tablet rasagiline 1 mg OD.

The patient had undergone magnetic resonance imaging on the morning of surgery and a stereotactic head frame was fitted prior to surgery, following which she was shifted to operating room (OR), and the procedure was begun under local anesthesia under monitored anesthesia care. O₂ supplementation was done through nasal prongs at 2 Lt/min.

The procedure included burr hole drilling and bilateral electrode placement in the subthalamic nuclei along with intraoperative neurological testing. Soon after the procedure began, it became increasingly difficult for the surgeons to place electrodes because of excessive patient movement resulting from tremors and dyskinesia. Hence, it was decided to complete the procedure under conscious sedation. The

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**Suman Sokhal,
Keshav Goyal,
Navdeep Sokhal,
Niraj Kumar,
Shweta Kedia¹**

Department of Neuroanaesthesiology and Critical Care, Neurosciences Centre, All India Institute of Medical Sciences, ¹Department of Neurosurgery, All India Institute of Medical Sciences, New Delhi, India

Address for correspondence:

Dr. Keshav Goyal,
Department of Neuroanaesthesiology and Critical Care, 710, Neurosciences Centre, All India Institute of Medical Sciences, New Delhi - 110 029, India.
E-mail: keshavgoyalster@gmail.com

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patient received a total of 150 mcg of injection. The patient received a total of 150mcg of IV fentanyl in repeated boluses, IV Midazolam 4mg followed by dexmedetomidine infusion starting @ 0.5 mcg/kg/hr which was gradually increased to 1mcg/kg/hr. In spite of this patient had no satisfactory sedation and control of excessive patient movement. Finally, we tried sedating the patient with low-dose intravenous (IV) ketamine.

The patient was administered injection ketamine 25 mg along with injection midazolam 1 mg slowly IV. We noticed prompt resolution of tremors and dyskinesia. Although the patient was unresponsive to verbal commands, respiration was regular. The effect of single 30 mg IV ketamine bolus weaned off after approximately 36 min with no significant cognitive dysfunction. She received additional boluses of IV ketamine 10 mg followed 10 min later by another bolus of 15 mg along with 0.5 mg midazolam, by which time, the electrodes were placed successfully. After electrode implantation, battery implantation in the right infraclavicular fossa was done in the same sitting under general anesthesia (GA), and the patient was extubated uneventfully. The head frame was removed before giving GA. The procedure is usually well tolerated without sedation/GA, except for uncooperative patients or those with severe dystonia.

Discussion

DBS has become an increasingly common treatment for PD and other movement disorders. Current medical treatments, while effective for symptomatic relief, may eventually produce long-term motor complications with chronic use. These long-term complications are the most common indication for neurosurgical intervention in the disease.^[1] The abundance, structure, and function of striatal receptors are altered by the dopamine depletion and further modified by the pharmacological treatment used in PD.^[1]

Benzodiazepines and other gamma-aminobutyric acid agonists can interfere with patient's cooperation and tremor interpretation and thus may be better avoided.

In a study by Sherman *et al.*,^[5] they discovered that a low-dose subanesthetic IV ketamine infusion treatment, which is already known to reduce treatment-resistant depression and chronic pain, is also beneficial in effectively reducing PD. In animal models, N-methyl-D-aspartate (NMDA) receptor antagonists are effective anti-Parkinsonian agents and can reduce the complications of chronic dopaminergic therapy (wearing off and dyskinesias).^[1] At subanesthetic doses (<0.3 mg/kg), IV ketamine possesses centrally-mediated analgesic properties with minimal effects on consciousness and cognition/arousal.^[6]

Different studies displayed the advantages of ketamine over isoflurane, pentobarbital, and droperidol when motor- and sensory-evoked potentials are monitored.^[7]

In our case, ketamine used for sedation resulted in prompt resolution of dyskinesia in a patient with severe PD. Any such case regarding the intraoperative use of ketamine for sedation in such patients has not yet been reported. Our case report definitely outlines the role of low-dose IV ketamine as NMDA antagonist that provided optimal patient sedation and control of symptoms intraoperatively in patients with severe PD. Furthermore, because of increasing population of octogenarians, we are more likely to come across such patients. Thus, this case report warrants further research in this area and may also have applicability in the perioperative management of such patients.

Thus, we conclude that low-dose IV ketamine, titrated to effect, could be used for providing optimal intraoperative sedation in patients with severe PD with troublesome dyskinesia and tremors that preclude optimal surgical conditions.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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