

## Administration of Anesthetics Like Propofol and its Possible Adverse Effect: Seizure Like Activity, an Exceedingly Rare Case Report

Juna Musa<sup>1\*</sup>, Loran Rakovica<sup>2</sup>, Blina Abdullahu<sup>3</sup>, Fiona Bushati<sup>3</sup>, Fareeha Nasir<sup>4</sup>, Ilda Zylalaj<sup>3</sup>, Mohammed Badi<sup>5</sup>, Amro Elrefaei<sup>6</sup> and Ali Guy<sup>7</sup>

<sup>1</sup>Postdoctoral Research Fellow, Department of Surgery, Physiology and Biomedical Engineering, Mayo Clinic, Rochester, Minnesota, USA

<sup>2</sup>University of Prishtina, School of Medicine, Kosovo,

<sup>3</sup>Medical Student, Mother Teresa Hospital, Albania

<sup>4</sup>Intern, Department of Surgery, Christian Hospital Quetta, Pakistan

<sup>5</sup>Research Fellow, Department of Neurology, Mayo Clinic, Florida, USA

<sup>6</sup>Research Fellow, Maternal and Fetal Medicine, Obstetrics and Gynecology Department, Mayo Clinic, Rochester, Minnesota, USA

<sup>7</sup>Department of Physical Medicine and Rehabilitation, Clinical Assistant Professor at New York University School of Medicine New York, USA

**\*Corresponding Author:** Juna Musa, Postdoctoral Research Fellow, Department of Surgery, Physiology and Biomedical Engineering, Mayo Clinic, Rochester, Minnesota, USA.

**Received:** May 02, 2020

**Published:** May 21, 2020

© All rights are reserved by **Juna Musa, et al.**

### Abstract

Propofol is a short-acting, lipophilic anesthetic. Intravenous propofol is the drug of choice for induction of anesthesia. Abnormal muscle movements, abnormal posturing and convulsions associated with the usage of propofol happen to be reported. The spinal cord plays an important role in modulating anesthetic-induced suppression of nociceptive transmission. Recent studies showed that propofol potentiates the inhibitory transmitter's glycine and 3~-aminobutyric acid (GABA) which enhance spinal inhibition during anesthesia.

In this case report a 40-year-old male had seizures shortly after receiving epidural steroid injection given under anesthesia induced by propofol and fentanyl. Serum prolactin levels were found to be elevated. CT brain, CT spine and MR imaging of the brain were negative for any suggestive findings of epilepsy.

**Keywords:** IV Propofol; Seizure; Epidural Injection

### Introduction

Cervical radiculopathy, also referred to as "pinched nerve" occurs when a nerve is compressed or irritated and branches away from the spinal cord [1]. Pain management is commonly achieved by a small non-invasive procedure consisting of interlaminar epidural steroid injections which aim to provide short term relief. Administration of steroids involves numbing the area with an anesthetic. Seizure like activity after anesthesia is rare, but it is reported as a potential complication [2]. Abnormal movements may be witnessed during the induction of anesthesia, but it does not necessarily confirm that a seizure is occurring [3]. Anesthetic medications act on the CNS in specific sites, like the hypothalamus, causing disintegration of neurons and a disturbance in cerebral blood flow [4]. This disturbance clinically manifests as tinnitus, disorientation, and finally, seizures. Under different circumstances, these neurological deficits can be transient or permanent [5]. Administration to a large blood supply, a combination of certain anesthetics or an excessive dosage could possibly cause this disturbance. Today we will dig deeper into this medical mystery as

to why administration of an anesthetic during pain management could cause seizure like activity.

In practice, a high serum prolactin level may be indicative of suspicious seizure like activity, if done within 20 minutes of onset of seizure. EEG may be falsely elevated after anesthesia induction, with otherwise normal findings.

### Case Report

This case represents one of the few medical mysteries as to why administration of anesthetics can cause seizure like activity in an otherwise rather healthy patient. Our patient, a 40-year-old male who presents for C7-T1 cervical interlaminar epidural injection for pain relief of cervical radiculopathy, in which he chose conscious sedation. The patient has no history of allergies or prior seizure activities. No history of epilepsy was noted in his past medical history.

Fentanyl and propofol were administered by the anesthesiologist; lidocaine was not given at the time. After transferring the patient to the recovery room from the OR, the patient was found to

be agitated, confused, with an abrupt onset of forceful tonic-clonic movements which lasted for approximately 20 - 30 seconds. Vital signs and oxygen saturation levels continued to remain stable throughout the whole event. A non-particulate solution was used at a C7-T1 cervical inter-laminar epidural injection. The contrast showed a particularly good flow with no obstruction. The entire medical staff was contemplating if what just recently occurred was a seizure. 40 minutes after the event, the decision was made to transfer the patient to the ER for a full seizure workup. In the ER, CT cervical spine showed no abnormality. Brain CT and CT of the arteries of the cervical spine were normal. The patient was then

admitted for neurology workup. Electroencephalogram showed a normal pattern. A repeat MRI of the brain and cervical spine showed solely disc pathology. The initial MRI showed C2-C3 disc prolapse and multilevel disc bulge. EMG revealed right C5-C6 cervical radiculopathy. After 20 minutes, prolactin levels appeared elevated, indicative of possible seizure activity. Motor, sensory functions and gait were intact.

**Images**

MRI: Neutral/Sitting; sagittal T1, Sagittal T2, Axial GE.

At C3/4, there is a bulging disc without stenosis.

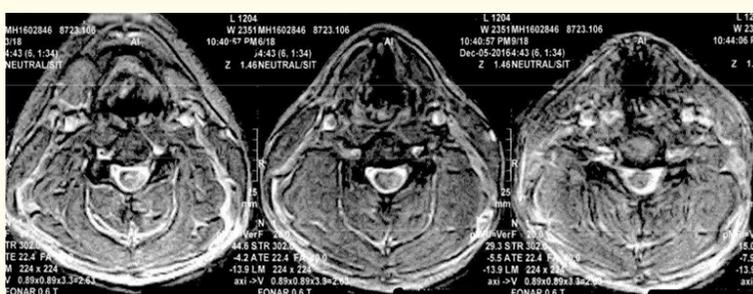


Figure 1

At C4/5, there is a bulging disc with thecal sac indentation.

At C5/6, there is a bulging disc without stenosis.

At C6/7 and C7/T1, the canal and foramina are patent.

Upper cervical lordotic accentuation with Grade I retrolistheses at C3/C4 and C4/C5 on extension position sequence.

EMG showed a right C5-C6 cervical radiculopathy.

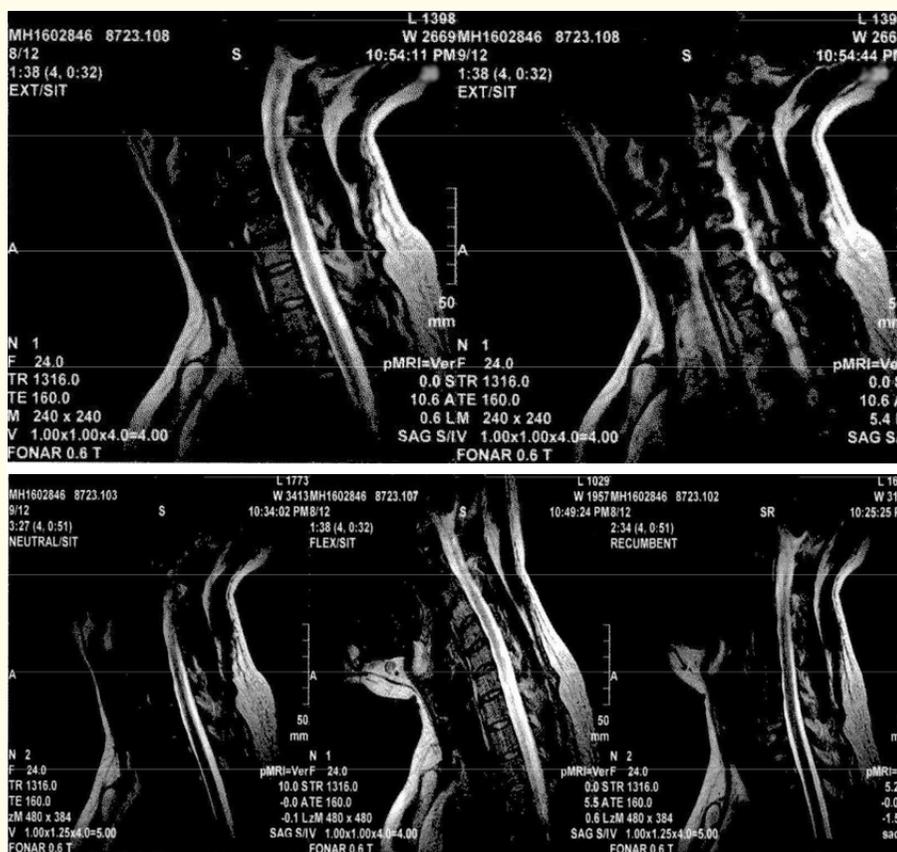


Figure 2

## Discussion

Propofol is one of the most commonly used medications in induction of anesthesia. It is considered generally safe. Propofol's main effect is in the GABA-related inhibitory receptors in the CNS. It decreases the rate of dissociation of the GABA from the receptor, thereby increasing the duration of the GABA-activated opening of the chloride channel and hyperpolarization of cell membranes [6].

This results in lowering of the excitability of the neurons in the cortex of the central nervous system. Therefore, theoretically propofol should have only anticonvulsant properties.

However, there are many cases where tonic-clonic seizures induced after the administration of propofol have been reported. The mechanism of action is not fully understood.

It is known that the main excitatory neurotransmitter in the CNS is glutamate. Recent studies and experiments have shown a possible link between low doses of propofol and the release of glutamate. This could explain its epileptogenic properties.

In 2009, Lu, *et al.* observed the effects of small doses of propofol on glutamate release in the cortical synapses of the rat brain and found that propofol can activate the 4-aminopyridine pathway and thus promote the release of glutamate. Additionally, activated 4-aminopyridine can also induce cell membrane potential depolarization.

The protein kinase C (PKC) pathway mechanism: the experiments of Lu, *et al.* revealed that when PKC is inhibited by propofol, glutamate release is suppressed. These authors suggested that propofol may promote cortical synaptic glutamate release through the activation of the PKC pathway, thereby increasing the likelihood of epileptic seizures [7].

Clinical reports suggest that propofol-induced seizures are also more often present during the combination of this anesthetic with other types of medications, especially opioids [8].

The pro-seizure effect of opioids happens because of the selective stimulation of  $\mu$  ( $\mu$ ) and  $\kappa$  ( $\kappa$ ) opiate receptors but not the activation of the  $\delta$  ( $\delta$ ) receptor system.

Stimulation of these receptor subtypes can lead to suppression of inhibitory GABAergic interneurons. This suppression may come by the decrease in GABA release in the presynaptic membrane. This effect of "inhibiting the inhibition" leads to an increased possibility of the precipitation of seizure activity [9].

## Conclusion

Propofol is considered a generally safe medication in the induction of anesthesia both in patients with and without past medical history of seizures. However, in some cases, propofol has been proven to induce tonic-clonic convulsions. This has been proven especially in cases where propofol is combined with medications, such as opioids. Even though this is a rare occurrence, interven-

tional pain management specialists as well as anesthesiologists and all medical professionals should be careful with all the procedures using combined anesthetics.

## Bibliography

1. Cervical Radiculopathy: Symptoms, Causes, and Treatment. WebMD (2019).
2. "Epidural Injections: Frequently Asked Questions". Hospital for Special Surgery (2020).
3. Voss LJ, *et al.* "The howling cortex: seizures and general anesthetic drugs". *Anesthesia and Analgesia* 107.5 (2008):1689-1703.
4. Alix Zuleta-Alarcon, *et al.* "Anesthesia-Related Perioperative Seizures: Pathophysiology, Predisposing Factors and Practical Recommendations". *Austin Journal of Anesthesia and Analgesia* 2.4 (2014): 1026.
5. "Neurology: The Information Resource for Clinical Neurology, Local anesthesia: neurologic complication". MedLink.
6. G Trapani, *et al.* "Propofol in anesthesia. Mechanism of action, structure-activity relationships, and drug delivery". *Current Medicinal Chemistry* 7.2 (2000): 249-271.
7. Lu CW, *et al.* "Facilitation of glutamate release from rat cerebral cortex nerve terminal by subanesthetic concentration propofol". *Synapse* 63.9 (2009): 773-781.
8. Bevan JC. "Propofol related convulsions". *Canadian Journal of Anesthesia* 40.9 (1993): 805-809.
9. Vaughan CW, *et al.* "Cellular actions of opioids on periaqueductal grey neurons from C57B16/J mice and mutant mice lacking MOR-1". *British Journal of Pharmacology* 139.2 (2003): 362-367.

### Assets from publication with us

- Prompt Acknowledgement after receiving the article
- Thorough Double blinded peer review
- Rapid Publication
- Issue of Publication Certificate
- High visibility of your Published work

Website: <https://www.actascientific.com/>

Submit Article: <https://www.actascientific.com/submission.php>

Email us: [editor@actascientific.com](mailto:editor@actascientific.com)

Contact us: +91 9182824667