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A RARE EVENT OF CONVULSIONS WITH PROPOFOL: CASE REPORT

Anaesthesiology

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KEYWORDS

INTRODUCTION

Various anaesthetic agents are known to produce seizures . Propofol is an inducing anaesthetic agent which is used for anaesthesia induction/sedation and for Total intravenous anesthesia. This case highlights a rare (incidence<1%) but potentially harmful complication of this intravenous anesthetic agent.

CASE REPORT

A 50 Year old male patient was posted for wound debridement for necrotising soft tissue infection. On pre-anesthetic evaluation, patient was American society of Anesthesiologist physical status class- II weighing 54 kg. History was not suggestive of any other comorbid illness including hypertension, diabetes , seizure disorder and tuberculosis. The patient was smoker, alcoholic and non vegetarian.His pulse rate was 113/min and regular. Blood pressure was 90/60 mm of hg in supine position , and respiratory rate was 17/min. Investigations included complete hemogram, liver and renal function test, serum electrolytes ,coagulation profile, electrocardiography, Doppler ultrasonography of the affected gangrenous foot and chest X ray.

In the operating room monitoring included ECG, pulse oximetry, non invasive blood pressure monitoring. After securing intravenous access with 18 G cannula , the patient was preoxygenated with 100% oxygen for 3 minutes. 150 mg of fentanyl was slowly administered intravenously. Anesthesia was induced with intravenous propofol 100mg but after a few seconds patient had generalized tonic clonic seizures which were more prominent over upper trunk and upper extremity but lesser in lower extremity. It was difficult to maintain airway because of sudden unexpected jerky movement and stiffness. Immediately Midazolam 2 mg was given after which Seizures subsided. The airway was secured with cuffed tracheal tube of 7.5 mm internal diameter and fixed at 22 cm after confirming bilateral air entry equal. Thereafter the patient remained hemodynamically stable and surgery was uneventful. At the end residual muscular blockade was reversed and trachea was extubated when patient was fully conscious and achieved regular spontaneous breathing. He was shifted to surgical ward after careful postoperative evaluation for 30 min. post operatively there was no evidence of postictal phase. Patient had an uneventful recovery.

DISCUSSION:-

Propofol has gained worldwide acceptance as an induction agent since its launch in 1986.[1] The administration of propofol is associated with generalized tonic-clonic seizures, focal motor seizures, increased tone with twitching and rhythmic movements, opisthotonus and involuntary movements, collectively termed as seizure-like phenomenon (SLP).[2] Despite numerous case reports, there is no clear consensus regarding the prevention and management of such adverse events. SLP has been reported with propofol administration in healthy as well as in epileptic patients.[1,3,4,5] Many instances of mild dystonic reactions after administration of propofol may go unreported, some have even questioned about their occurrence as adverse. Such movements were observed in 75% of children receiving propofol as compared to 20% receiving thiopental. [5]These movements after propofol induction seem to be rare in adults. At present, the influence of dosage and speed of injection on their incidence is not clear.

Despite the claims that propofol has proconvulsant activity, there is significant evidence to the contrary. Propofol reduces the effective duration of convulsion during modified electroconvulsive therapy, thus favoring its anticonvulsant property. [7]Hewitt et al ., concluded from their study, using electrocorticography in patients with intractable epilepsy, that propofol has anticonvulsant activity without having any proconvulsant activity. [1]Propofol infusions have been shown to be effective in controlling status epilepticus (as assessed neurophysiologically). [3] The use of propofol, barbiturates or midazolam in the management of refractory status epilepticus has been reported. [8]Baraka et al., reported a patient with no history of epilepsy, undergoing spermatic veins ligation under epidural anesthesia with propofol sedation. At the end, after 5 min of propofol discontinuation, patient developed tonic-clonic convulsions. He concluded that these convulsions might be due to lidocaine- induced systemic toxicity, which was masked by the anticonvulsant action of propofol. [6]

The pathophysiological mechanisms of the neuroexcitatory symptoms with propofol are unknown. The typical electroencephalogram (EEG) pattern following propofol sedation in healthy volunteers is biphasic, consisting of an initial increase in frequency from alpha to beta waves, which is then followed by a slowing to delta waves. Meyer et al ., demonstrated in their study, distinct EEG patterns following propofol-induced sedation in children with epilepsy and children with learning difficulties with no occurrence of SLP of epileptic origin. [9] Drugs or a physiological state such as sleep which produces partial functional differentiation of the cortex create conditions which promote synchronization of neuronal activity. The balance between inhibition and suppression of excitation, on the one hand, and production of synchrony on the other, determines if the overall effect is to activate or suppress epileptiform EEG discharges. [1] Spontaneous movements induced by propofol are not related to cortical activity and subcortical origin has been suggested.[5] In high doses propofol depresses both the cortex and subcortex thus acting as anticonvulsant and in low doses it may act as proconvulsant by inhibiting the inhibitory subcortex, resulting in the 'release' of normal hyperexcitability in the cortex. [3]

A criticism of these reports is that on many occasions combinations of drugs were administered, making the implication of propofol impossible, as many of the anesthetic agents may cause clinically evident seizures. There are reports of grand mal seizure-like motor behavior in patients after fentanyl. [10] The abnormal movements could be due to nonepileptic myoclonus produced by the interaction of these narcotics with opiate receptors leading to blockade of cortical inhibitory pathways. Patients treated with higher doses of fentanyl and its analogues may fail to exhibit seizure-like movements as high plasma opioid levels depress central neural centers. [10,11]

Lidocaine is capable of producing seizures in toxic dose ranges by cortical hyperstimulation related to selective depression of inhibitory cortical synapses and neurons. [12] For attenuating the pain of propofol injection, 20 mg of preservative-free lidocaine was used in our patient, which is much less than its toxic dose.

Propofol preservatives could be the inciting factors which need to be investigated. Seizures subsequent to anaphylaxis with the drug or its adjuvants may be a possibility. But in the present case, this possibility is unlikely because no bronchospasm, erythema or hypotension was observed.

Seizures during anesthesia induction can result in dislodgement of intravenous cannula/lines, monitoring devices may injure the patient and dislodge laryngeal mask airway. [4] In our patient, the course of postoperative awakening was normal. Normal CT scan rules out any

major anatomic visual causes. In this case, seizures developed after propofol administration, so we were in a dilemma whether to give more propofol for seizure suppression or to use some other drug. Controversy persists whether to use more propofol or some other anticonvulsant for seizure suppression as it is liable to precipitate seizures. Therefore, midazolam were used in our patient as they have been used effectively in the past.[11]

CONCLUSION

Since daycare surgery has gained great popularity and in the future probably will be even more common, we would like to stress the importance of seizure activity with propofol and a high vigilance is warranted perioperatively.

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