TREATMENT OF REFRACTORY SEIZURES IN ECLAMPSIA WITH PROPOFOL – A Case Report

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SUMMARY

We describe the management of a case of refractory status epilepticus evolving in post partum eclampsia. The seizures were refractory to therapy with benzodiazepines, MgSO4, barbiturates and phenytoin, but responded rapidly to propofol infusion.

Keywords: Refractory seizures, Eclampsia, Epilepticus, Propofol.

Introduction

Convulsive status epilepticus refractory to treatment with benzodiazepines, phenytoin sodium or barbiturates presents a challenge to critical care specialists. Such cases of refractory status epilepticus (RSE) are not uncommon. Prompt seizure control is probably essential to prevent mortality and morbidity.

Propofol, which is a unique non barbiturate anaesthetic agent, has been used in nearly 33 studies in RSE with good results. However the pro or anticonvulsant effects of propofol remain a matter of controversy, with many reports describing abnormal movements, posturing and seizure like activity with its use. On the other hand, systematic studies in both humans and animals strongly suggest that it has anti-epileptic properties.

However, no case report so far has described the use of propofol in eclampsia induced RSE. We describe the management of such a case.

Case report

A 23 year old primigravida with twin pregnancy at 38 weeks of gestation, was admitted at 16:55 hrs with complaints of ‘leaking membranes’ since afternoon. She was a registered case but on irregular antenatal checkup. A physical examination revealed the presence of pedal edema and a blood pressure of 160/100 mm Hg. She was fully conscious, oriented and asymptomatic otherwise. Laboratory investigations revealed mild proteinuria. Her haemoglobin was 10 g%, packed cell volume 33%.

Biochemical investigations were within normal limits. She was diagnosed as a case of pre-eclampsia with twin pregnancy and given prophylactic MgSO4 I.M (5g in each buttock). Induction of labor was started at 23:30 hrs. The patient developed a generalized seizure at 04:55 hrs and her blood pressure was 170/110 mm Hg, which was managed with inj. diazepam I.V. She had another seizure at 05:15 hrs. She was taken up for emergency caesarian section at 06:30 hrs for which general anaesthesia with rapid sequence induction was given. Intra operative course was unremarkable and she was responding to commands following extubation. Her blood pressure was 140/92 mmHg. She was shifted to the ward at 07:30 hrs. She developed another generalized seizure in the ward at 10:30 hrs, which was treated with inj. diazepam (BP of 160/110) and inj. MgSO4 4g I.V. She went into a state of “status” from 11:30 hrs with the convulsions not responding to diazepam with the patient remaining unconscious. She was intubated at this point of time and shifted to the CCU, put on ventilatory support (SIMV10, FiO2 0.4, TV 500 ml, Ppeak 23 cm of H2O) and started on inj. thiopentone infusion 2 mgkg⁻¹hr⁻¹ after a bolus of 250 mg to control the seizures. Her blood pressure was persistently greater than 140/100 mm of Hg and a nitroglycerine (NTG) infusion was started and gradually titrated to maintain the blood pressure around 140/90. One hour after starting the infusion, she again developed seizures, which was controlled with a bolus of inj. thiopentone 100 mg and inj. diazepam 10 mg. She however had a repeat seizure after 15 minutes, following which, she was given a loading dose of inj. phenytoin 1000 mg over a period of 30 minutes. She however continued to have focal seizures and myoclonus. Arterial blood gas analysis revealed mild respiratory alkalosis. An emergency CT scan revealed only the presence of mild cerebral edema.

2 hours after starting thiopentone infusion, she still had focal seizures with a blood pressure of 140/90 mmHg with NTG infusion of 2 µgkg⁻¹min⁻¹. Her pupils were constricted and not reacting to light, plantars were equivocal. Spontaneous respiratory efforts were present.

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At this point of time, thiopentone infusion was stopped and propofol infusion was started at a dose of 4 mgkg⁻¹hr⁻¹ after an initial bolus of 50 mg. Five minutes after starting infusion, the blood pressure came down to 110/70 mm of Hg and the NTG infusion was stopped. The blood pressure then gradually rose over a period of 15 minutes to settle at 130/90 mm of Hg. The patient had no recurrence of focal seizures or myoclonus after starting propofol infusion. Tab.amlodepin 5 mg was started OD through the ryles tube and we were able to taper the dose of propofol over the next 24 hours to 1.5 mgkg⁻¹hr⁻¹; the infusion rate of which was further reduced over a period of next 48 hrs. The patient was successfully extubated on the fifth day with a Glasgow coma scale (GCS) of 15.

Discussion

In the above mentioned case, the patient had persistent seizures in spite of receiving the full therapeutic dose of MgSO₄ (blood levels were 4.7 meql⁻¹). Her seizure activity was not controlled even with thiopentone, diazepam and phenytoin. Also, it was necessary to start NTG infusion to have her blood pressure under control. However, on starting propofol infusion, her seizure activity was rapidly brought under control and we also achieved a good control on her blood pressure. Thus, propofol infusion not only afforded us a control of her seizure activity but also of her blood pressure.

Propofol is a unique, non barbiturate anaesthetic agent with proven anticonvulsant properties, although the exact anticonvulsant mechanism is not known.² It probably acts by causing a uniform depression of the central nervous system, potentiation of GABA mediated pre and post synaptic inhibition and by decreasing the release of excitatory transmitters, glutamate and aspartate.³ Stecker et al compared propofol with high dose barbiturates in the treatment of refractory status epilepticus and they concluded that propofol resulted in a rapid control of RSE compared to high dose barbiturates. Also, recurrent seizures were common with sudden discontinuation of propofol but not with gradual tapering.⁴ In our case, we had a gradual decrease in the dose of propofol without having any recurrent seizure activity. Classen et al, in an excellent review, systematically reviewed the literature for treatment strategies of RSE using pentobarbital, propofol and midazolam.⁵ They concluded that treatment of RSE with any continuous I.V anti epileptic drug infusion so as to attain EEG background suppression may be more effective than other strategies for RSE treatment. Harrison et al have recommended adjusting the infusion rate of anti epileptic drug so as to attain EEG burst suppression.⁶ In our case, we adjusted the infusion rate so as to control further seizure activity. Our patient was on respiratory support while on propofol infusion. Propofol is a potent respiratory depressant and hence it is strongly advised to have facilities for airway control and ventilatory support during its use.

References


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