ABSTRACT

Unconsciousness in seventy-five year old male after peribulbar block is reported. The patient was hypertensive, non diabetic, not well oriented, non sedated became unconscious, nonresponsive to painful stimuli without excitatory effect like convulsion.

Key words: Peribulbar block, Unconsciousness, Local anaesthetics.

INTRODUCTION:

Local anaesthetic rapidly crosses the blood brain barrier causing central nervous system stimulation followed by depression at a higher doses. In retrobulbar block local anaesthetic mixtures of equal volumes of 2% lignocaine and 0.5% (plain) bupivacaine are commonly used for quicker onset and longer duration of local anaesthetic. Accidental injection of drug into optic nerve sheath results with spread of drug into CSF and CNS is exposed to higher concentrations of drug leading to apprehension and unconsciousness. The patient of old age, malnourished and debilitated are usually less expressive and may undergo unconscious without previous prodromal signs like perioral tongue numbness, restlessness etc.

CASE NOTE:

A geriatric male of 75 years, weight 42 kg was admitted into ophthalmology ward with bilateral mature cataract. During admission his heart rate was 68/min. Blood pressure 180/100 mmHg; investigations showed low haemoglobin 7.9 gm/dl with normal ECG, blood sugar and serum creatinine. Chest skiagram coincides with COPD. The patient was planned for intraocular lens implant in both eyes. No pre-medication, sedation and anti-hypertensive were given. He was less communicative before operation. Facial nerve was blocked with 3 ml 2% lignocaine and 2 ml 0.5% (plain) bupivacaine by surgeon. The same procedure was done on the opposite side by same amount of local anaesthetic. Peribulbar block was done by equal volume and concentration of both local anaesthetics. As a result total 12 ml 2% lignocaine (240 mg) and 8 ml 0.5% (plain) bupivacaine (40 mg) were infiltrated to the patient.

Five minutes after, the patient became unconscious and was unresponsive to painful stimuli, anaesthesiologist was called for help. On arrival of anaesthesiologist it was found that patient’s HR was 110/m, BP 220/110 mmHg, respiration normal, haemoglobin- saturation was 97% detected by oximetry. Patient was observed closely for 40 minutes and gradually responded to painful stimuli and vocal command. No convulsion was observed.

DISCUSSION:

Local anaesthetic agents using lignocaine, combined with bupivacaine produce excellent sensory and motor block with rapid onset of action and reasonable duration of about 3 hours. Central nervous system is especially vulnerable to local anaesthetic toxicity and may be taken as guideline for signs of overdose in awake patient. Early symptoms are perioral numbness, tongue paraesthesia, dizziness, tinnitus, drowsiness, unconsciousness convulsion and finally respiratory arrest and cardiovascular collapse often occurs.

Toxicity depends on dose of the drug, systemic absorption, and accidental intravascular injection. Recommended maximum ‘safe’ doses are rough estimations only, since other factors are involved. Maximal safe doses for lignocaine are 3 mg.-Kg without vasoconstrictor and for bupivacaine is 2 mg.-kg.

In the reported case the dose of lignocaine was 240 mg and bupivacaine 40 mg. The recommended ‘safe’
dose of lignocaine in 42 kg is 146 mg. Administered dose of lignocaine 240 mg, which was about double of the safe dose, was absorbed into brain through CSF and blood stream. As mentioned earlier higher dose of local anaesthetic causes depressions of CNS without excitatory effect like convulsion.

CONCLUSION:
In regional anaesthesia LA drugs above the recommended safe dose in aged and less communicated patient can undergo unconsciousness without prodormal manifestations and they should be monitored during intraoperative period preferably by an anaesthesiologist.

REFERENCES: