SUMMARY

A number of experiments indicate that the use of bupivacaine as a local anaesthetic may be associated with increased risk of systemic toxicity. This risk being further increased by pregnancy, acidosis, hypoxia, and the use of drugs like cimetidine which impair the metabolism of the amide type of local anaesthetics.

A 21 year old female underwent emergency caesarean section under subarachnoid block with bupivacaine. Baby delivered with Apgar score 9/10. She subsequently developed hypertension, tachycardia, convulsions followed by respiratory depression, and bradycardia. The mother could be saved due to timely and proper management.

Keywords: Subarachnoid block, Hypertension, Hypotension, Bradycardia, Tachycardia, Convulsion.

Introduction

Subarachnoid block is an effective means of providing analgesia for caesarean section because of its time tested safety and efficacy. Its safety lies in the fact that mother remains conscious throughout the procedure at the same time hazards of general anaesthesia are avoided. However, life threatening emergency though rare with this technique may occur. One such rare complication of subarachnoid block is presented.

Case report

A 21-year old female with full term pregnancy in labor was admitted to emergency obstetric ward. Ultrasonography showed reduced foetal movements and oligohydramnios. She was brought to operation theatre for emergency caesarean section and subarachnoid block was planned. Her Pulse rate was 86/min, Blood Pressure – 176/90 mmHg, haemoglobin – 9.7 gm%, urine examination was normal, Blood group – B+ve and ECG within normal limits. After intravenous access and employing monitoring ECG, pulse-oximetry and non invasive BP, she received inj. ranitidine 50 mg, metoclopramide 10 mg intravenously. Lumbar puncture was performed in L4-L5 space with 25G spinal needle in sitting position and after getting clear and free flow of CSF, 2.5 ml of 0.5% bupivacaine heavy was injected and the patient was put in supine position. 100% oxygen was administered via ventimask; level of analgesia was tested and surgery started. Within 5 min of commencement of surgery patient became restless and there was tightness in the jaw. However the obstetrician continued with the surgery as BP was 170/100 mmHg, pulse rate 90/min and SpO₂ 100%. Healthy baby delivered with Apgar score of 9/10. During surgery she received 800 ml of 5% dextrose, 300 ml Ringer’s lactate. Urine output was 500 ml. Rest of the course of surgery was uneventful and duration was 40 minutes.

She was immediately shifted to ICU; there she complained of headache and heaviness in the chest. She was given inj. diclofenac sodium 2 ml IM and O₂ inhalation was continued. ECG showed ST depression in lead II, III, AVF and chest leads (V2-V6) Blood Pressure was 170/100 mmHg. Suddenly she developed generalised convulsions which lasted for about 2 mins. There was fall in respiratory rate and pulse rate, blood pressure was 170/110 mmHg. Immediately inj. diazepam 10 mg was given IV and infusion was started with 40 mg of diazepam in 500 ml normal saline at the rate of 10 micro drops/min⁻¹. Respiration was shallow and bradycardia persisted. She was intubated and put on ventilator. She also received intravenous atropine 0.6 mg, furosemide 20 mg. and sublingual nifedepine 5 mg. After 15 mins, she became conscious, responding to verbal commands, pupils reacting to light and respiratory efforts were good. She was extubated after keeping on T-piece for 10 mins. Heart rate and blood pressure stabilized within half an hour. ECG recordings became normal. Urine output was 500 ml. Patient remained stable thereafter. Diazepam drip was tapered off slowly in 24 hrs. CT scan of the brain and fundus examination of eye was done after 48 hrs, and the reports were normal. Rest of the hospital stay was uneventful. She was discharged after 10 days.
Discussion

Examination of data from the American Society of Anaesthesiologist (ASA), a closed claim project, identifies several cases of cardiac arrest during spinal anaesthesia. Since most of these cases predate the routine use of pulse oximetry, many physicians believed oversedation and unrecognized hypoxia as the cause. However, now large prospective studies continue to report a relatively high incidence of cardiac arrest in patients under spinal anaesthesia perhaps as high as 1:1500. A recent examination of this problem has identified vagal response to decreased preload to be the key factor. Local anaesthetic toxicity may occur in various ways. Animal experiments indicate that bupivacaine may be especially toxic in pregnancy. Acidosis and hypoxia further increase the toxicity of bupivacaine. Pregnancy may also increase circulatory toxicity of bupivacaine. The use of cimetidine but probably not ranitidine impairs metabolism of amide type of local anaesthetics and may increase their potential toxicity. Solution of 0.5% bupivacaine can be recommended and a volume of 1.8 – 2.2 ml will suffice for spinal anaesthesia in caesarean section. The patient was pregnant, she had long fasting hours, without preloading with intravenous fluid before subarachnoid block. She was of small built and had received 2.5 ml of bupivacaine. All these factors when put together may have resulted in systemic toxicity. Fateh Singh Bhati et al — reported loss of consciousness following spinal anaesthesia for caesarean section using lignocaine heavy.

Cardiac arrest has been reported in healthy patients during administration of spinal anaesthetics. Local anaesthetics vary in their toxicity, chloroprocaine being the least and bupivacaine as the most toxic. Systemic absorption of local anaesthetics can exert direct effect on cardiac muscle and vascular smooth muscle, resulting in broad range of effects. CNS toxicity is characterized by "light headedness" or convulsions. There was no history of diabetes, hypertension, loss of consciousness or epilepsy in our patient. Her blood sugar and serum electrolytes were normal. Fundus of the eye and CT scan of the brain were normal. She could be resuscitated in a short time by timely intervention, and absence of associated feature ruled out the possibility of amniotic fluid or air embolism. A healthy female given subarachnoid local anaesthetic should not loose consciousness. This can happen only if patient develops total spinal block characterized by hypotension and respiratory depression. Our patient exhibited sequence of cardiovascular and central nervous system features in the form of brief initial excitation followed by depression. Despite our effort cause of this problem could not be detected however, bupivacaine toxicity could have been the possible cause.

References

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