AN UNUSUAL CASE OF REFRACTORY HYPOTENSION FOLLOWING SPINAL ANAESTHESIA

Lt Col KC KHANDURI

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Introduction

Serious complications following spinal anaesthesia has led to its varying popularity from place to place and time to time. But many vigilant anaesthesiologists still prefer major abdominal surgeries under spinal anaesthesia due to its inherent advantages of decreased blood loss and decreased metabolic and endocrine stress responses to surgery. With the recent advances in monitoring and refinement in techniques, morbidity can be further minimised and mortality can be almost eliminated. Successful management of resistant hypotension following subarachnoid block is presented. The possible causes and review of literature is discussed.

Case Report

A 37 year old female, ASA grade - I patient was posted for exploratory laparotomy for a suspected ovarian tumour. She gave a vague past history of allergic rhinitis, but was presently asymptomatic and on no medication. There was no history of IHD, hypertension, asthma or diabetes mellitus. She was of average built, weighing 56 kg. Her pulse and BP were 78/min and 124/78 mm Hg respectively. ECG was normal and her biochemical and haematological investigations were within normal limits.

Spinal anaesthesia was planned for the patient. She was premedicated with diazepam tab (10 mg). After preloading with 18 ml/kg of Ringer's lactate, lumbar puncture was done using 24 gauge spinal needle at L₂₋₃ space in left lateral recumbent position. Level of the table was maintained horizontal. After confirming free flow of the CSF, 3 ml of 0.5% heavy bupivacaine was injected in 4 secs. Patient was then turned into supine horizontal position. Pulse, oxygen saturation and ECG were monitored continuously till the end of operation. BP was recorded manually every min till 10 min, and then every 5 min till the end of the operation.

The upper level of sensory block after 15 min was at T₅ level, using pin prick method. After about 30 min of spinal anaesthesia, patient started showing progressive hypotension without significant change in pulse rate and ECG. In next 10 min BP fell to 60 mm Hg despite remedial measures like increase in rate of IV drip, oxygenation, repeated doses of inj Mephenteramine (6 mg x 3 doses, then 9 mg x 2 doses) and raising the legs. Inj Atropine 0.6 mg IV was repeated twice but it also had no significant affect on BP or pulse rate. Oxygen saturation also fell down to 85% and

there was not much improvement even after oxygenation by face mask. Patient also started complaining of dizziness and was apprehensive. There was no evidence of any significant bleeding or aortocaval compression by the surgical packs or retractor. There was no change in sensorium or respiration and patient was able to breathe deeply on command. Lungs were clear. Then inj Hydrocortisone hemisuccinate 200 mg was given IV prophylactically and to rule out adrenocortical suppression and inj Mephenteramine 15 mg IV repeated. There was no significant change in BP and SpO₂ inspite of all the above measures. Ultimately it was decided to continue the surgery under GA. So, after slow induction with 80 mg of IV ketamine hydrochloride, vecuronium bromide 4 mg IV was given, 7.5 mm oral cuffed endotracheal tube was passed and patient maintained on oxygen, N₂O (50:50), IPPV and inj Pentazocine 9 mg IV.

Soon after the endotracheal intubation, it was found that BP started rising to normotensive range and then remained stable throughout the surgery, ranging between 110 to 122 mm Hg. The oxygen saturation also rose to 100% and pulse rate to 86-94/minute. Rest of the procedure was uneventful. At the end of surgery the residual neuromuscular block was reversed with usual doses of neostigmine and atropine and patient was extubated. Patient remained haemodynamically stable post-operatively.

Discussion

Hypotension during spinal anaesthesia is mostly due to sympathetic block leading to vasodilation. Such cases show prompt response to vasopressors like ephedrine and mephenteramine. In higher blocks involving cardioaccelerator nerve (T₂₋₄), hypotension is accompanied with bradycardia which is further aggravated by unopposed vagal influence [1]. Bradycardia is an ominous sign of impending cardiac arrest. This may be the probable cause of unexplained deaths [2]. Myocardial contractility is also compromised by the extensive block, further leading to decrease in cardiac output. Furthermore, hypotension due to sympathetic block can trigger a vasovagal attack leading to severe bradycardia. This reflex is abolished under GA [3]. This may be the reason why in some centres light GA is given with spinal anaesthesia.

Intraoperative hypotension can also occur due to aortocaval compression, either due to tumour or surgi-

cal packs, but it was ruled out in this case. Acute haemorrhage was also ruled out.

Retrospectively speaking, probable cause of hypotension in this case might have been the combined effect of sympathetic blockade and anaphylactoid reaction to the local anaesthetic. Patient gave a history of allergic rhinitis and was a potential high risk case for anaphylactic or anaphylactoid reactions, though the incidence is far less as compared to the ester group of local anaesthetics. Vasantha Rao and FS Oscar have reported a case of progressive hypotension with bradycardia leading to asystole, following infiltration of bupivacaine [4]. In the present case, there was no history of exposure to local anaesthetic hence anaphylactoid reaction could be the cause as it may occur with the first exposure to an antigen. It is well established that plasma catecholamine levels are reduced after spinal and epidural anaesthesia and elevated after GA [5]. Intense sympathetic stimulation during laryngoscopy and intubation may have triggered the release of endogenous adrenaline and helped in overcoming the crisis. Timely anticipation of anaphylactoid reaction was missed in this case, consequently leading to resistant hypotension. Early administration of intravenous adrenaline is recommended in case of resistant hypotension and bradycardia [6].

Talking of anaphylaxis, in the recent years the exposure to latex products should also be kept in mind as a probable cause, eg. surgical gloves, urinary catheters, endotracheal tubes, face masks, airways, blood pressure cuffs, hoses delivering gases, etc. It is worth-

while to be warned of potential allergic reactions ranging from mild wheal to cardiovascular collapse arising out of latex allergy. A latex free operation room may be an answer to that. Severe anaphylactic reaction to haemacel infusion during spinal anaesthesia has also been reported [7]. It may be concluded that allergic phenomenon should be kept in mind when tackling a case of persistent hypotension or cardiovascular collapse. Future health of the patient depends on being able to identify the responsible antigen.

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