

Does bradycardia and hypertension always equal to Cushing's response during supratentorial craniotomy?

Sir,

Bradycardia and hypertension are the common haemodynamic presentations in patients with raised intracranial pressure (ICP). Whenever patients present with hypertension and bradycardia, it needs to be correlated with radiological imaging. In the absence of radiological findings, the cause for hypertension and bradycardia needs to be evaluated.

A 45-year-old woman (weight 55 kg and height 163 cm) was diagnosed with high-grade glioma and was planned for supratentorial craniotomy. She was on anticonvulsant (sodium valproate) and dexamethasone preoperatively. The magnetic resonance imaging brain showed right temporal mass with peritumoral oedema with no midline shift, ventriculomegaly, cisternal obliteration or uncal herniation.

On the day of surgery, after the routine standard monitoring and line placement, we have noted that her baseline blood pressure (BP) was 140/80 mmHg with the heart rate (HR) of 63/min. Since the imaging was done 1 month earlier, the possibility of rapid growth of high-grade glioma causing bradycardia and hypertension was suspected. The patient was induced and intubated after fentanyl (150 µg), propofol (150 mg) and vecuronium (6 mg). Mayfield head clamp was fixed after the administration of additional fentanyl, propofol and 2% xylocaine local infiltration. There was no haemodynamic response to the pin insertion. Anaesthesia was maintained with air, oxygen and isoflurane.

Since there was no noxious stimulation between the pin insertion and surgical incision, systolic BP came down to 90 mmHg, which was treated with 5 mg of ephedrine. Soon after the ephedrine administration, BP rose to 150/70 mmHg and the HR dropped from 70 to 44/min. This was treated with 60 mg of propofol, which failed to decrease the BP. Instead, the BP further rose to

200/112 (150) mmHg with the HR drop of 39/min. When we mentioned the haemodynamic changes to the surgical colleagues, they informed us that they had just carried out scalp infiltration with 7 ml of 2% xylocaine with adrenaline (1 in 200,000). This hypertensive crisis was treated with additional propofol, fentanyl and labetalol, which brought the BP down to 160/80 mmHg. As there was sudden bradycardia along with hypertension, the possibility of seizures (high-grade temporal lesion) or pin site extradural haematoma causing Cushing's response was our differential diagnosis. Since, the ICP reducing effect is better and the cerebral perfusion pressure is higher with total intravenous anaesthesia (TIVA) compared to inhalational agent,^[1] we had changed the inhalational anaesthetics to TIVA.

During the episode of hypertensive crisis, the bispectral index value was 40–45 and bilateral pupils were equal (2.5 mm) and reacting to light which ruled out the seizure. After craniotomy, the brain was found to be lax and pulsatile, which ruled out the pin site extradural haematoma. Then, the BP was brought down further with two bolus doses of glycerol trinitrate (20 µg each). The procedure lasted for 3 h and thereafter, the episodes of hypotension were treated with 25–50 µg of phenylephrine. The patient was extubated awake and was transferred to Neuro Intensive Care Unit with no deficits. She was discharged in a good clinical state on the 7th post-operative day.

We re-enquired the patient's relatives for any other history which was not revealed to us during the pre-operative evaluation, as we were unable to explain the erratic BP changes that occurred intraoperatively. After repeated questioning, the patient's husband told us that she was taking drugs for anxiety disorder. The next day, we found out from the patient that she was taking propranolol (20 mg) and clonazepam (0.5 mg) (combination drug) for the same, and she continued to take this drug during the pre-operative period without informing the primary physician or anaesthesiologist. We were now able to offer an explanation for the cause of hypertensive crisis. As the patient was on propranolol, a non-selective beta blocker, administration of ephedrine and adrenaline prevented vasodilatation leaving unopposed alpha stimulation leading to hypertensive crisis.^[2] This patient was seen by two different anaesthesiologists both in pre-anaesthesia clinic (1 week prior) as well as in the ward (the day before), at both times she refused to tell about the anxiety disorder and the treatment history.

This case gave us an insight that all bradycardia and hypertensive response during craniotomy need not be a

Cushing's response. When the pre-operative bradycardia and hypertension do not correlate with radiological finding, it is better to evaluate the cause before surgery to prevent morbidity. Lateral thinking and proper communication at each stage of surgery are the key elements to prevent morbidity. Caution is needed when we use ephedrine or epinephrine in a patient who is on non-selective beta blocker to avoid life-threatening hypertensive crisis.

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