Severe hypotension in transforaminal lumbar interbody fusion surgery: Is it vasovagal or?

Sir,

Hemodynamic disturbances are commonly reported in cervical and upper thoracic spine surgeries. But significant cardiovascular changes during lumbar spine surgery are rarely reported. Here, we report such a case and its possible mechanism.

A 72-year-old female patient weighing 70 kg came to the hospital with lower back pain radiating to both lower limbs. Magnetic resonance imaging showed multiple level disc protrusion at the lower lumbar vertebral (L2/L3/and L4/L5) level. Patient was posted for transforaminal lumbar interbody fusion (TLIF) with vertebroplasty. Patient was a known case of hypertension and diabetes, and was on regular medications. All laboratory investigations were normal. As our routine protocol in a hypertensive patient, 2D echo was done and found to be normal. Patient was premedicated with intravenous glycopyrrolate 0.2 mg in the OT. Routines monitors were attached. General anesthesia was induced with fentanyl 2 mcg/kg, propofol 2 mg/kg, and tracheal intubation facilitated with rocuronium 1 mg/kg. Trachea was intubated using 7.5-mm cuffed flexometalic endotracheal tube. Patient was turned prone. Anesthesia was maintained with sevoflurane in oxygen air mixture (40:60) and intermittent boluses of fentanyl as and when required. All the pressure points were checked and neck was kept in neutral position. No muscle relaxation was given as neurophysiological monitoring was planned. Depth of anesthesia was maintained with minimum alveolar concentration between 1 and 1.5. Intravenous morphine 6.0 mg was also given. At the time of dilatation near L2 vertebral level, patient had severe bradycardia (PR < 38/min) followed by hypotension (60/43 mm Hg). Within 20 s, the heart rate returned to normal, but hypotension persisted for 1.5 min, for which 6 mg ephedrine was given. During this time, patient’s ETCO2 graph also showed signs of respiration. Surgeon was also noted during this time. ABG was sent for and found to be normal. After a few minutes, patient became hemodynamically stable and there was no sign of respiration too. Rest of the intraoperative course was uneventful. After completion of surgery, patient was turned back to supine position and trachea was extubated after ascertaining full neurological recovery. Patient was shifted to post-anesthesia care unit for observation.

In this case, we ruled out the other possible causes of hemodynamic disturbances such as prone positioning, anesthetic-induced blood loss, electrolyte and acid base disturbances, and diabetic autonomic neuropathy. In other case reports, the vasovagal reflex and Bezold–Jarisch reflex were postulated as the probable mechanisms of bradycardia. Its mechanism has been described as resulting from stimulation of afferent parasympathetic nerve endings due to traction on the dura, causing a reflex celiac (vasovagal) reaction.1,2 In our patient, the possibility of this dural traction cannot be ignored. In a recent case report, the author highlighted that autonomic dysreflexia and some central cord connections of the nerve fibers persist through which these kinds of hemodynamic perturbances occurred.3 The severe hemodynamic alteration in our case was associated with changes in respiration too and all events occurred only during the dilatation phase of TLIF. This means the surgical stimulus might be associated with some kind of central stimulation and warrants further research. It is noteworthy here that the heart rate became normal within a few seconds, but hypotension remained for a few minutes. In this regard, invasive arterial line could detect the sudden hypotensive changes which could be easily missed by noninvasive blood pressure monitoring (usually set as 3-5 min interval).

In conclusion, one should always be vigilant regarding hemodynamic disturbances even in any level of spine surgery and routine use of invasive monitoring can detect even the subtle changes, so should be used.

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Letter to Editor

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