Capnothorax induced subclavian artery compression

The Editor,

The advent of video-assisted thoracoscopic surgeries (VATS) has greatly decreased the number of thoracotomies that are being done. Though this procedure decreases morbidity, it has its own drawbacks such as capnothorax induced respiratory acidosis, mechanical compression of the major airway leading to ventilation difficulty, and vascular injuries. We report a case of capnothorax induced subclavian artery compression causing unilateral limb ischemia and its successful management.

A 25-year-old American Society of Anesthesiologists 1 man (weight – 55 kg, height – 158 cm) was diagnosed to have a large (13 cm × 8 cm × 5 cm) proximal and mid esophageal tumor abutting the trachea, bronchi, and the adjacent major blood vessels. He was planned for a McKeown’s esophagectomy using VATS. As per our institutional protocol, induction was carried out with the standard monitoring, such as an electrocardiogram, noninvasive blood pressure (NIBP), pulse oximeter (right hand), and a radial arterial line (left hand). A 37F right sided double lumen tube (DLT) was placed and confirmed with the fiberoptic bronchoscope. The patient was positioned in left lateral position; there was a good correlation between the NIBP and the invasive blood pressure (BP) recordings. After instituting one-lung ventilation (OLV), capnothorax was induced, and the intrapleural pressure was maintained between 9 and 10 mmHg. Ten minutes later, there was a wide discrepancy between NIBP and the invasive BP reading. While the NIBP was 60/30/40 mmHg, the arterial line BP was 110/60/75 mmHg (systolic/diastolic/mean). On palpation of the right radial pulse, it was found to be feeble, and the pulse oximeter trace looked damped while the left radial pulse was normal. On shifting the pulse oximeter to the left hand, the pattern of the trace was normal. The position of the right upper limb was rechecked to rule out axillary artery compression. The surgeons were informed about this discrepancy and the possibility of subclavian artery compression was thought due to the combined pressure effects of capnothorax and the tumor. The intrapleural pressure was reduced from 10 mm of Hg to 6–7 mm of Hg. Soon after, the pulse oximetry trace normalized, pulse volume on the right side improved, and the NIBP readings correlated well with the invasive BP readings. The thoracoscopic resection continued uneventfully for the next 2 h with a total surgical time of 4 h.

Thoracoscopic surgery is most often done with the institution of OLV using a DLT. Capnothorax facilitates surgery by providing adequate space thereby, better visualization of structures and better dissecting conditions. Studies have shown that a pleural pressure of up to 10 mmHg can be safely used without compromising hemodynamics. Pinto and Galketiya demonstrated that, insufflation pressures of 6–8 mm of Hg is associated with no adverse respiratory or cardiovascular effects and minimal hemodynamic changes.

It has been found that the mean BP measured invasively and noninvasively correspond, while there can be differences in the systolic BP especially in the setting of hypotension (systolic blood pressure <95 mmHg). In our case, the discrepancy in both mean and systolic BP with other supportive clinical findings led us to realize that the vessel was probably being compressed. The disappearance of the discrepancy and the normalization of pulse oximetry waveform soon after releasing the capnothorax confirmed our clinical diagnosis.
The importance of monitoring the BP in both upper limbs to prevent limb ischemia is highlighted here. In the presence of a large esophageal tumor, maintaining lower intrapleural pressure may be advisable. As always eternal vigilance and lateral thinking are the key factors to prevent morbidity.

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Conflicts of interest
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