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Surgery Quiz – Case 28

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ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2020, 37(6):849–850

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A 78-year-old male patient with a history of coronary artery bypass grafting (CABG) for three-vessel disease and associated ACC/AHA stage C congestive heart failure referred to our surgical department for evaluation of a giant left retroperitoneal mass. Abdominal computed tomography (CT) and magnetic resonance imaging (MRI) showed that the mass was fat-containing with thick irregular enhancing septa suggestive of a liposarcoma. The mass extended from the subsplenic space to minor pelvis and caused remarkable displacement without infiltration of the spleen, left kidney, left ureter, urinary bladder, root of mesentery, small intestine, left colon and pancreas (fig. 1). There was no evidence of metastatic disease. The patient submitted, under combined general and epidural anesthesia, to R0 resection of a 39×29×18 cm stage IIIB pT4pN0cM0 G2 well differentiated liposarcoma along with distal pancreatectomy and splenectomy. On immediate postoperative period the patient had persistent hypotension.

What was the cause of hypotension?

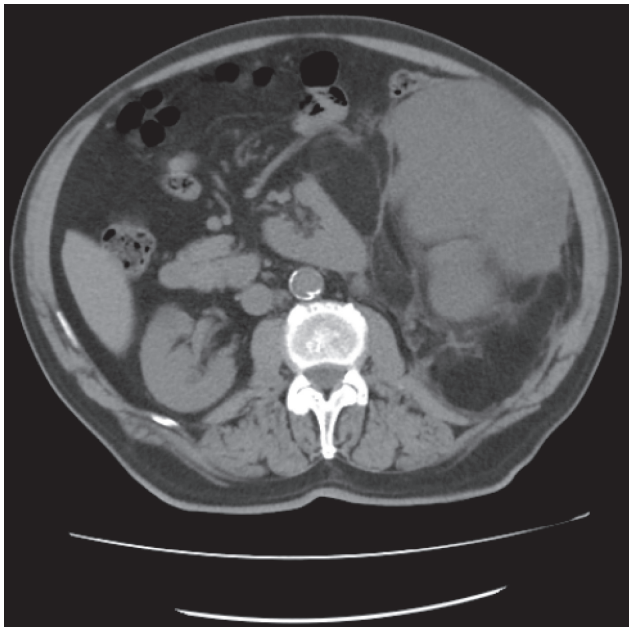

Figure 1

Comment

In our patient's case, hemorrhage and epidural anesthesia were the most prominent causes of hypotension on the immediate postoperative period. Hemorrhage was excluded as: (a) no blood loss observed from the two closed passive drainage tubes, (b) no alteration in hemoglobin observed on repeated measurements, and (c) central venous pressure (CVP) was at the highest normal values on repeated measurements. Epidural anesthesia was the apparent diagnosis. Hypotension is a common complication of epidural anesthesia which causes sympathetic blockage with subsequent arterial and venous vasodilatation causing functional hypovolemia and resulting in systolic and diastolic blood pressure, and heart rate decrease. Management includes discontinuation of epidural analgesia, vasopressors, restricted fluid loading with crystalloids or colloids and titration to determine optimal dosage for blood pressure and hourly diuresis normalization. In our patient's case, persistence of hypotension despite optimal management was exceptional as vasopressors required for hemodynamic stabilization until the third postoperative day.

A fact that was neglected on preoperative planning was the physiologic effects of the space-occupying retroperitoneal mass, as well as the effects after abdominal decompression. On the one hand, the mass caused chronic grade III intraabdominal hypertension which resulted in: (a) cardiac dysfunction with decreased cardiac output due to inferior vena cava (IVC) and portal vein compression, (b) pulmonary dysfunction with decreased thoracic volumes and elevated peak pressures due to compression of the diaphragm, (c) renal involvement with decreased glomerular filtration rate (GFR) and low urine output, and (d) reduced visceral blood flow. On the other hand, abdominal decompression after resection resulted in: (a) increase in diaphragmatic excursion which led to improved ventilation without evidence of pulmonary re-expansion syndrome, (b) decompression of the IVC which led to improved cardiac output, and (c) reversion of renal dysfunction. Although abdominal decompression should have led to decompression of IVC resulting in cardiac output and blood pressure improvement, patient's hemodynamic instability persisted.

Another fact that was neglected on preoperative planning was the ischemia/reperfusion injury after abdominal decompression. Ischemia/reperfusion injury may have systemic inflammatory mediated local and systemic consequences including (a) microvascular injury ranging from damage to the endothelial barrier resulting in leakage and tissue edema to the no-reflow phenomenon, (b) myocardial stunning, (c) renal dysfunction, (d) central nervous injury, (e) gastrointestinal injury, and (f) multiple organ dysfunction syndrome (MODS).

In conclusion, the cause of hypotension can be explained in microvascular level. On the one hand, epidural anesthesia caused arterial and venous vasodilatation causing functional hypovolemia. On the other hand, ischemia/reperfusion injury after abdominal decompression caused damage of the endothelial barrier resulting in third-space fluid shift and deterioration of hypovolemia. In the present patient, the crucial mistake on preoperative planning was the addition of epidural to general anesthesia. In analogous cases of resections of giant abdominal masses, epidural anesthesia should be avoided.

References

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