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CASE REPORT

Acute left heart failure with pulmonary edema during resection of pediatric neuroblastoma: case report

Shan Gao, Youjing Dong ⓘ *

Shengjing Hospital of China Medical University, Department of Anesthesiology, ShenYang, China

Abstract   Resection of an unknown neck mass in a 6-year-old child triggered acute left-sided heart failure and pulmonary edema. The lesion was confirmed as neuroblastoma by post-operative tissue examination. Such tumors regularly synthesize and secrete catecholamines, warranting caution in advance of surgical manipulation.
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Introduction

Neuroblastoma is one of the most common extracranial solid tumors in children. It always occurs on adrenal region, abdominal cavity, chest, neck, or pelvis. Early diagnosis is difficult and relies heavily on tissue biopsy. However, it is important to note that a key physiologic characteristic of neuroblastoma, as a neuroendocrine tumor, is the synthesis and secretion of catecholamine. Patients with neuroblastoma may have particular clinical manifestations, such as excitement, palpitation, facial flushing, headache, hypertension, and tachycardia, easily confused with pheochromocytoma. Furthermore, if the depth of intraoperative anesthesia is inadequate, or the tumor is tugged or compressed during resection, there is a danger of cat-echolamine surge. Severe hemodynamic fluctuations may then ensue, causing irreversible damage. In this report, we describe a child afflicted with acute heart failure during treatment of neuroblastoma. It is imperative that anesthesiologists are cognizant of this dire possibility and prepare adequately in advance of surgery to avoid a similar course of events.

Case report

This 6-year-old child was admitted to the hospital with the chief complaint of a right-sided neck mass. There was no history of other diseases, and computed tomography (CT) confirmed a mass of the intermuscular space in the right neck. To discern its nature, the surgeon in charge chose resection under general anesthesia.

Once in the operating room, the child was monitored regularly, and venous access was established. Sufentanil, propofol, and cisatracurium were used to induce general

* Corresponding author.
  E-mail: dyj1cn@163.com (Y. Dong).

anesthesia. The child's blood pressure, heart rate, and oxygen saturation of blood were stable during the first 20 minutes of operative time. However, by exposing and tugging on the mass, the BP increased to 220/110 mmHg, and the HR to 140 beats/min. The anesthetist promptly intervened and suspended the operation, concerned that the depth of anesthesia was inadequate. More propofol and a higher concentration of inhalation sevoflurane were subsequently administered. After 30 seconds, the BP responded (140/72 mmHg), but HR was little affected (135 beats/min). Upon resuming resection, the surgeon triggered another BP spike (210/105 mmHg), with no change in HR (138 beats/min).

Given the uncertain nature of this mass, pheochromocytoma was immediately considered by the anesthesiologist in charge. In accord with consensus anesthesia management of such tumors, phentolamine (1 mg), and esmolol (10 mg) were given. Radial artery and left subclavian vein catheterizations were also performed to invasively monitor BP and gauge central venous pressure (CVP). A standard anesthesia protocol for pheochromocytoma, including continuous phentolamine infusion and attention to intravascular volume expansion, was then implemented. At this point, BP was under control (130/75 mmHg), and HR had retreated somewhat (120 beats/min), so the operation continued.

Shortly thereafter (10 min), SpO_2 (79%) and arterial BP (77/43 mmHg) had fallen. We first suspended the intravenous infusion of phentolamine, giving dexamethasone (10 mg) and norepinephrine (10 μg). Airway pressure and CVP readings continued to rise at this juncture, and moist rales were detectable in both lungs by auscultation. X-ray fluoroscopy also showed characteristic Kerley B lines at costophrenic angle. A state of acute left heart failure was evident, complicated by pulmonary edema, necessitating immediate cardiotonic diuresis. Furosemide (10 mg), cedilanid (0.25 mg), and morphine (2 mg) were injected intravenously, and continuous intravenous dopamine infusion served to maintain BP. The patient was switched to dorsal elevated position, and positive end-expiratory pressure was boosted to 5 cmH2O.

Within 30 minutes, the child's vital signs had stabilized (BP, 95/62 mmHg; SpO_2, 95%; HR, 120 beats/min), allowing surgery to proceed. Total operative time was 230 minutes. After a fair volume of Ringer's solution (1250 mL), urinary output was only 130 mL, but another 120 mL of fluid (pale pink) was suctioned via endotracheal tube. Postoperatively, the child was taken to the pediatric surgical intensive care unit for further observation. Once there, all vital signs were stable, and no heart failure was evident. Extubation took place the next day. One week later, echocardiography, cardiac function, and brain natriuretic peptide level had normalized, so the patient was returned to the ward.

Throughout the surgical procedure, observed clinical manifestations resembled those of ectopic pheochromocytoma. Repeat abdominal CT indicated that the adrenal glands were disease-free. Postoperative pathologic examination of neck mass disclosed a type of neuroblastoma.

Discussion

Similar to sympathetic postganglionic fibers and adrenal medulla, neuroblastoma cells are capable of catecholamine release and uptake. However, most of the catecholamine is inactivated internally, and less is released.[1] This is why children regularly escape cardiovascular symptoms until cardiac enlargement, heart failure, or even cardiogenic shock develops. Without adequate preoperative provisions, the risk of anesthesia increases substantially.

In this case, we believe such lack of preparedness led to abrupt hemodynamic instability. As we stressed: (1) any mass of unknown nature requires caution, calling for a careful history, needle biopsy, and preoperative catecholamine testing of blood and urine to exclude neuroblastoma or occult pheochromocytoma; (2) in the absence of diagnostic clarity, stark hemodynamic fluctuations in children during surgery raise a question of neuroblastoma or pheochromocytoma and demand a protocol devised for pheochromocytoma; and (3) active intraoperative monitoring is helpful in this setting to guide appropriate usage of various drugs and infusions that are key to ensuring circulatory stability.

Traditional BP and CVP hemodynamics correlate poorly with volume loads, failing to accurately reflect patient status, and are unduly influenced by intrathoracic pressure shifts during mechanical ventilation. The pulse index contour continuous cardiac output systems now available are minimally invasive and better suited for this purpose, generating whole-body hemodynamic metrics via arterial access and central venous catheter. Cardiac output is measured as isolated values by thermal dilution and charted continuously through area under arterial pressure wave curve analysis.[2] Pulse index contour continuous cardiac output monitoring is based on cardiac index, intrathoracic blood volume index, and extravascular lung water index as sensitive gauges of cardiac function, cardiac preload state, and pulmonary edema, respectively. These parameters are almost never affected by catecholamines, circulatory capacity, mechanical ventilation, or other factors. They help determine the need for speed/volume of rehydration and whether cardiotonic diuretic or vasoactive drugs are indicated to augment cardiac function and improve perfusion/oxygenation of tissues. Reducing the adverse effects of pulmonary edema and hypoxemia bolsters the prognosis, limiting durations of mechanical ventilation and ICU hospitalization, and lowering mortality.[3]

In this particular patient, pre- and postsurgical management in certain aspects of anesthesia were not optimally addressed. When the sudden hemodynamic fluctuation occurred, pheochromocytoma was first suspected. Thus, we used low dose phentolamine immediately to control the BP.[4] In addition, when BP firstly decreased to 77/43 mmHg, aside from initial adjustment, maintenance anesthesia had not changed, so anesthetic overdose was doubtful. Likewise, the child's prior history and marginal decline in end-tidal CO2 level were not supportive of acute pulmonary embolism. Still, anaphylactic shock could not be excluded, even without cutaneous signs. We first suspended the intravenous infusion of phentolamine, giving dexamethasone (10 mg)

and norepinephrine (10 μg). However, according to the CVP and imaging examination results, the child was diagnosed with acute left heart failure and pulmonary edema. Our first choice on an emergency basis was dopamine. In retrospect, however, dobutamine is preferential, given its positive effects on cardiac contractility and lesser impact on HR.[5] Nonetheless, it was gratifying that the patient quickly rebounded and recovered uneventful, with no lingering complications.

Conclusion

The pathophysiologic mechanism of neuroblastoma during resection may be similar to that observed in pheochromocytoma. It is necessary to make a timely diagnosis and take active measures. The purpose of perioperative period is maintenance of hemodynamic stability. In addition, perfect preoperative examination can help us make correct clinical judgments.

Conflicts of interest

The authors declare no conflicts of interest.

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