Anesthetic Management of an Infant Undergoing Radical Nephrectomy for Wilms Tumor: A Case Report

Markus Goodrich, CRNA, MSN
Virginia Keatley, RN, DNSc
Barbara Norwood, RN, EdD

Wilms tumor (WT), also called nephroblastoma, is a solid, malignant renal mass that can sometimes grow so large it spreads outside the kidney and invades other structures. Most experts recommend complete tumor resection as a primary intervention. The clinical manifestations caused by the WT, most significantly hypertension, can appear as a barrage of pathophysiological events to the nurse anesthetist. The case presented involves an 8-week-old infant who underwent a radical nephrectomy because of a WT. The occurrence, symptoms, pathophysiology, and intraoperative anesthetic management of WT are discussed.

Keywords: Nephroblastoma, pediatric anesthesia, pediatric hypertension, Wilms tumor.

Wilms tumor, a childhood cancer that arises from immature kidney cells, accounts for approximately 6% of all cancers in children. Approximately 500 new cases are diagnosed in the United States each year.[1] One-third of these tumors occur in children younger than 1 year old. The tumor usually is discovered as an asymptomatic abdominal mass by family or during a routine well-baby examination. Wilms tumor arises anywhere in the kidney as an embryologic precursor to renal cells. It grows rapidly, doubling its size in 11 to 13 days.[2] After repeated doubling, the tumor grows outside its pseudomembranous capsule into renal sinuses, intrarenal lymphatics, and blood vessels.[3] The tumor can grow so large that it spreads outside the kidney and invades other structures. Symptoms begin to occur when the tumor disrupts renal physiology or compresses adjacent organs or vessels. The symptoms are a firm lump or swelling in the child's abdomen or side, abdominal pain, hematuria, and hypertension. Without proper treatment, the tumor will continue to grow until the body cannot adapt to its ever-increasing size.

Before the 1930s, the prognosis for children diagnosed with Wilms tumor was poor.[4] Over the next 30 years, advances in chemotherapy, radiation therapy, and surgical technique significantly improved the outcome. In 1969, the leading children's cancer research groups in the country developed the National Wilms Tumor Study Group. Its goals were to improve survival rates in children with Wilms tumor and to study the long-term outcomes of successful and unsuccessful treatments.[5] One of the first objectives of this group was to develop a classification system to describe the spread of the tumor. Physical assessment and laboratory and radiological testing provide a basis for a diagnosis; however, diagnosis is best verified by surgical excision with biopsy. Most experts recommend early and, if possible, complete tumor resection, followed by tumor categorization and chemotherapy.[6]

For patients with Wilms tumor, anesthetic management can be especially challenging and usually begins preoperatively because greater than 25% of these patients have hypertension.[6] Presumed mechanisms for the hypertension include elevated renin levels secondary to renal ischemia produced by mechanical compression of the renal artery, secretion of renin by the tumor itself, and a possible pressor substance produced by the tumor[7] (Figure). The hypertension can be so severe that it can cause encephalopathy and cardiovascular compromise. Secondary hyperaldosteronism and hypokalemia can manifest the chronic hypertension. Anemia and other blood dyscrasias may also be present with late-stage tumors or a history of previous chemotherapy.

Wilms tumor excision can be a benign procedure; however, because of the pathophysiological manifestations of the condition, the intraoperative care can become quite complicated. Considerable swings in blood pressure can manifest during induction of anesthesia, tumor manipulation, blood loss, and clamping of the renal vein. Even though preoperative management consists mainly of decreasing blood pressure, the danger of significant intraoperative hypotension due to hypovolemia from acute hemorrhage also exists. Persistent bleeding most often occurs when the tumor has extended into the inferior vena cava and/or adhered to adjacent organs.[8]

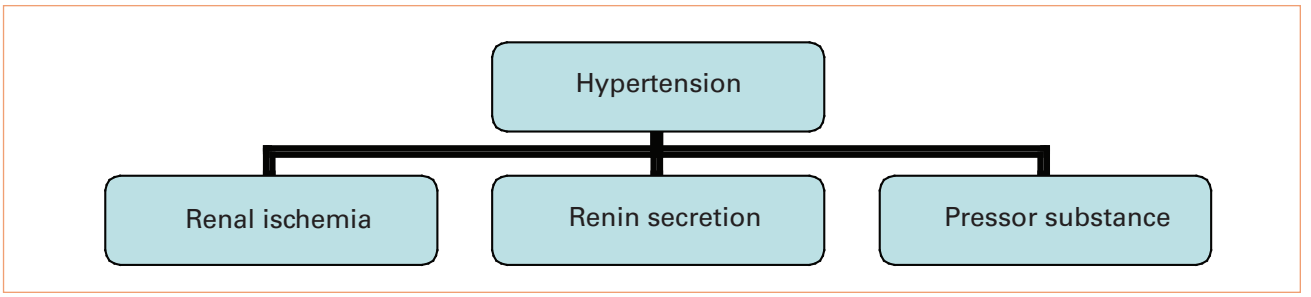

Figure. Presumed Mechanisms for Hypertension

Case Summary

An 8-week-old, 5.3 kg male infant was admitted to the emergency department with a sudden onset of gross hematuria. He was playful and in no acute distress. Further examination revealed a large, palpable right flank mass and a blood pressure reading of 180/110 mm Hg. An intravenous (IV) line was established. A complete blood count, urinalysis, and chemistry panel were ordered. An ultrasound and a computerized axial tomography of the abdomen also were scheduled.

Blood count and chemistry panel were normal, but the urinalysis showed blood at 4+ parts per microliter. The ultrasound revealed a large right intrarenal tumor measuring 9.6 cm × 7.5 cm with Doppler blood flow. The computertized axial tomography results confirmed that the mass was displacing the right kidney, bowel, pancreas, and liver. The inferior vena cava was intact. A preliminary diagnosis of Wilms tumor was reported. The pediatric surgery department was consulted about the mass, and the internal medicine department was consulted to manage the hypertension. On admission, the patient was given 0.1 mg/kg of captopril orally, which decreased his blood pressure to 130/90 mm Hg. Because the patient responded to antihypertensive therapy, he was scheduled for surgery the next day. Before the surgery, the patient was given 6 doses of 0.1 mg/kg of captopril by mouth overnight to control his hypertension.

Before arrival at the preoperative holding area, the patient was sedated with midazolam, thus calming him to conserve energy needed for surgery. In the preoperative area he was awake and smiling with his parents. After a review of the history and physical, a plan of care was developed by the anesthesiology team and the patient was taken to the operating room.

On arrival in the operating room, the patient was given 100% fractional concentration of oxygen in inspired gas while standard monitors were applied. For induction, the patient, who weighed 5.3 kg, was given 10 µg of fentanyl, IV; 25 mg of thiopentothal, IV; and 0.5 mg of vecuronium, IV. Once intubating conditions were achieved the patient was successfully intubated with a metal #1 Miller blade and a size 4.0 styletted endotracheal tube on the first attempt. A left radial arterial line was then placed. After induction, systolic blood pressure was in the low 60s. The patient was immediately treated with 2 10-mL/kg fluid boluses that increased systolic blood pressure to the 90s. Maintenance anesthesia consisted of 1.0% isoflurane and 50% fractional concentration of oxygen in inspired gas. The surgeon then placed a right subclavian central line for intraoperative fluid maintenance and possible postoperative chemotherapy. A nasogastric tube was placed to decompress the stomach.

Throughout the 4-hour surgery the patient was kept paralyzed with subsequent doses of 0.05 mg/kg of vecuronium. Maintenance IV fluid was administered at 15 mL/hr. In the event of severe hypertension that was not controlled by opioids or increasing isoflurane concentration, sodium nitroprusside was prepared and available to start at 0.5 µg/kg per minute, IV. The patient remained hemodynamically stable after induction except for 3 episodes of sudden hypotension with a systolic blood pressure in the low 70s. The surgeon was immediately made aware of this change. The surgeon repositioned the kidney/tumor off the vena cava and systolic blood pressure quickly started increasing back to the 90s.

At the end of the case estimated blood loss was 6.0 mL. The arterial line and peripheral IV were removed. The patient was recovered from anesthesia with 0.25 mg of neostigmine, IV, and 0.06 mg of glycopyrrolate, IV. The patient was then evaluated for extubation. Following satisfactory assessment of ventilatory and neuromuscular parameters, the trachea was extubated. The patient was placed in left lateral position with oxygen by face mask at 8 L/min and transferred to the postanesthesia care unit, where there were no complications and the patient's vital signs were stable.

Discussion

The preoperative assessment of any patient provides the basis for the anesthetic plan. This is particularly important in pediatric patients with WT because of the likelihood of blood dyscrasias, electrolyte imbalances, acid-base disturbances and, in rare instances, coexisting congenital abnormalities, such as cryptorchidism, hemihypertrophy, hypospadias, sporadic aniridia, Denys-Drash syndrome, or Beckwith-Wiedemann syndrome.[9] Blood dyscrasias, most often anemia and thrombocytopenia, usually occur from the tumor bleeding into the ab-

dominal cavity. Electrolyte imbalances and acid-base disturbances are derived from chronic hypertension (HTN) secondary to WT formation. Whether WT is diagnosed early or late, HTN can occur in up to 80% of patients.[9]

Both recognition of the HTN and appropriate preoperative and perioperative treatment is mandatory for the safe surgical treatment of children with this condition.[10] As previously discussed, HTN from WT is related to renin secretion from the tumor or renin release from the kidney due to renal ischemia. Therefore, preoperative selection of pharmacologic agents that counter the specific pathophysiological mechanisms responsible for the increased arterial pressure is logical. Sympatholytics, both centrally acting, such as methyldopa and clonidine, and some beta antagonists such as propanolol, suppress the stimuli for renin release.[11] However, since most long-acting beta antagonists do not affect the underlying vasoconstriction, they can cause severe hypotension. This can occur because renin-induced HTN is typically labile and, in the event of a decrease in renin production, beta blockers could prevent a compensatory increase in cardiac output by stopping the reflex increase in heart rate during periods of hypotension. Thus, nonselective beta antagonists are frequently avoided.

Since activation of the renin angiotensin system is the cause of the HTN, the most common preoperative classification of medication given to control blood pressure is an angiotensin-converting enzyme inhibitor, usually captopril. Captopril interrupts the renin angiotensin system cascade by inhibiting the conversion of angiotensin I to angiotensin II, thereby reducing the vasoconstrictive properties of angiotensin II and stopping the release of aldosterone. It has a plasma clearance half-life of 2 hours and a clinical half-life of 4 hours. Therefore, the last dose of captopril should not be given within 4 hours of tumor removal to prevent rebound hypotension.[12]

In severe cases of HTN, the patient may have central nervous system complications or cardiovascular decompensation.[13] Control of the blood pressure in these situations is prudent to prevent further deterioration. Intravenous sodium nitroprusside is usually used in these instances and carried over to the perioperative setting. Sodium nitroprusside does have negative effects. It also may cause reflex tachycardia that usually can be controlled with the short-acting beta antagonist, esmolol.

Intraoperatively, manipulation of large tumors can cause wide variations in arterial pressure. Tumor manipulation can cause blood pressure elevations due to excessive renin release from compression. These instances usually require interventions from the anesthetist such as increasing the depth of anesthesia, giving opioids, or using sodium nitroprusside and esmolol. Large decreases in blood pressure can also occur because of blood loss or compression on the vena cava by the tumor, which decreases venous return. Intravascular volume replacement and vasopressors, such as phenylephrine are used for hypotension. Hypertension usually remains postoperatively for 1 to 3 weeks even though renin concentrations rapidly return to normal levels. This is most likely a result of residual hypertrophy of the left ventricular wall and arterial wall media that occurred preoperatively in response to chronic increased afterload.[11]

Wilms tumor is a rare condition that is most often found incidentally and cannot be diagnosed officially without a biopsy. Hypertension is the most detrimental manifestation. Increased renin levels are usually the initial cause of the HTN, and preoperative treatment is based on decreasing the conversion of angiotensin I to angiotensin II. The goal of intraoperative interventions mainly consists of controlling the exaggerated swings in blood pressure throughout the surgery to support structural integrity.

REFERENCES

1. American Cancer Society. Detailed Guide: Wilms Tumor. What are the key statistics for Wilms Tumor? http://www.cancer.org/docroot/CRI/content/CRI_2_4_1X_What_are_the_key_statistics_for_Wilms_tumor_46.asp?sitearea=. Accessed April 17, 2008.
2. Craft AW. Growth rate of Wilms' tumor. *Lancet.* 1999;354(9184):1127.
3. Caty M, Shamberger R. Abdominal tumors in infancy and childhood. *Pediatr Clin Surg North Am.* 1993;40(6):1253-1271.
4. Zderic S. Renal and adrenal tumors in children. *Urol Clin North Am.* 2004;31(3):607-617.
5. National Wilms tumor Study. The NWTS Mission Statement. http://www.nwtsg.org/about/about_mission.html. Accessed March 11, 2005.
6. Ruddy R. Emergency presentations of cancer in children. *Clin Ped Emerg Med.* 2005;6(3):184-191.
7. Cobb ML, Vaughan RW. Severe hypertension in a child with Wilms' tumor: a case report. *Anesth Analg.* 1976;55(4):519-521.
8. Ritchey ML, Shamberger RC, Haase G, Horwitz J, Bergemann T, Breslow NE. Surgical complications after primary nephrectomy for Wilms' tumor: report from the National Wilms' Tumor Study Group. *J Am Coll Surg.* 2001;192(1):63-68.
9. Glick R, Hicks J, Nuchtern J, et al. Renal tumors in infants less than 6 months of age. *J Pediatr Surg.* 2004;39(4):522-525.
10. Steinbrecher H, Malone P. Wilms' tumour and hypertension: incidence and outcome. *Brit J Urol.* 1995;76(2):241-243.
11. Cody R. Haemodynamic responses to specific renin-angiotensin inhibitors in hypertension and congestive heart failure. a review. *Drugs.* 1984;28(2):144-169.
12. Khan AB, Carach R, Leckie BJ, Lindop GB. Hypertension associated with increased renin concentrations in nephroblastoma. *Arch Dis Child.* 1991;66(4):525-526.
13. Stine KC, Goertz KK, Poisner AM, Lowman JT. Congestive heart failure, hypertension, and hyperreninemia in bilateral Wilms' tumor: successful medical management. *Med Pediatr Oncol.* 1986;14(1):63-66.

AUTHORS

Markus Goodrich, CRNA, MSN, is currently practicing at Memorial Hospital, Chattanooga, Tennessee. He is a member of Anesthesiology Associates.

Virginia Keatley, RN, DNSc, is an associate professor at the University of Tennessee at Chattanooga School of Nursing, Chattanooga, Tennesee.

Barbara Norwood, RN, EdD, is an associate professor at the University of Tennessee at Chattanooga School of Nursing.