Hypertension in a 3-Year-Old Boy

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A 3-year-old boy was brought into the office for vague abdominal pain of 5 days’ duration. His mother stated that he had had several episodes of nonspecific pain in the abdomen that lasted a few minutes and resolved spontaneously.

The patient was born at 34 weeks' gestation with no complications and no catheter placement; he had neonatal hyperbilirubinemia that was treated with phototherapy. At his 3-year well-child visit, his blood pressure was recorded as 116/72 mm Hg, but at 3 subsequent rechecks, his blood pressure was within the normal range for his age. His grandfather and uncle both had essential hypertension. There were no known kidney problems or hearing difficulties in any family members.

During this visit, the child was active and playful and in no acute distress. However, when asked, he admitted to having abdominal pain. His weight was 14.2 kg (25th percentile) and his height was 94.2 cm (10th percentile). Heart rate was 120 beats per minute, and blood pressure was 150/118 mm Hg (in repeated measurements and verified with similar values in multiple extremities). He was afebrile and in no respiratory distress. The remainder of the physical examination findings were normal; there were no skin lesions, ear anomalies, genitourinary anomalies, abdominal masses, or bruits.
Results of a neurological examination were also normal. Laboratory evaluation revealed a normal complete blood cell count, a sodium level of 132 mEq/L, potassium level of 3.4 mEq/L, chloride level of 93 mEq/L, and bicarbonate level of 22 mEq/L. Blood urea nitrogen level was 10 mg/dL, and creatinine level was 0.3 mg/dL. Results of a urinalysis were negative, as were results of urine catecholamine measurements and thyroid function tests. The patient was admitted to the hospital and initially given a 2.5-mg dose of nifedipine, which lowered his blood pressure to 147/76 mm Hg. Oral captopril was also started, at a dosage of 6 mg (= 0.4 mg/kg/dose) every 6 hours. Over the next few days, the dosage was titrated up to 10 mg (= 0.7 mg/kg/dose) every 6 hours. The patient continued to have normal blood pressures on this dosage. During his hospitalization, a hypertension evaluation was initiated with a Doppler renal ultrasonogram, which revealed no definite abnormality. CT angiography showed apparently normal right and left main renal arteries. An accessory left renal artery was identified as probably a normal anatomic variant, as was a horseshoe kidney. Subsequently, an aortogram and bilateral renal angiograms utilizing digital-subtraction angiography were obtained; these revealed moderately severe stenosis of the left main renal artery just proximal to the bifurcation, with associated post-stenotic dilatation (Figure). The impression given by this last study was of renal artery stenosis, likely the result of fibromuscular disease. Subsequently, left renal artery angioplasty with a balloon catheter was performed. The procedure was successful, and no residual stenosis was evident. There were no apparent complications. The patient was discharged home the following day with a prescription for captopril at the dosage that he had been receiving (10 mg every 6 hours); the dosage was tapered over the next 2 to 3 weeks. The child's blood pressure remained within normal limits for his age without medication during subsequent follow-up visits. His abdominal pain had resolved before hospital discharge and was attributed to his hypertension in the absence of an alternative explanation.

SECONDARY HYPERTENSION IN CHILDREN

Given the increasing incidence of childhood obesity, primary or essential hypertension is now routinely identified in children and adolescents. Thus, in addition to a thorough history and physical examination, weight measurement and body mass index calculation have become critical parts of the evaluation of a hypertensive child. These values can help determine when clinicians need to evaluate for secondary causes of hypertension. In this patient, several factors increased the suspicion for secondary hypertension—namely, his very young age, the fact that he was not overweight, and the severity of his high blood pressure. The latest report of the National High Blood Pressure Education Program Working Group (NHBPEP) defines stage 2 hypertension in children and adolescents as a systolic or diastolic blood pressure greater than the 99th percentile plus 5 mm Hg.1 Repeated blood pressure measurements confirmed that this child had stage 2 hypertension as defined by the NHBPEP guidelines. Those guidelines suggest prompt referral for evaluation and therapy in asymptomatic children with stage 2 hypertension. "Immediate referral and treatment" are warranted in children with stage 2 hypertension who are symptomatic. The severity of this patient's hypertension, together with his complaints of vague abdominal pain, compelled us to initiate inpatient evaluation and management. The differential diagnosis of secondary hypertension in children is broad; findings from the patient's history and physical examination help guide the workup. A history of gross hematuria, edema, or previous urinary tract infections may suggest renal disease. Numerous prescription and over-the-counter medications can cause hypertension. The presence of a murmur or variable extremity blood pressures may point to coarctation. Café au lait spots may suggest neurofibromatosis, while a palpable abdominal mass accompanied by diaphoresis, flushing, and persistent tachycardia is highly suggestive of pheochromocytoma. Still, despite the myriad possibilities, renal disease and renovascular disease remain the most common causes of secondary hypertension in children.2 Thus, in the absence of findings that might suggest other causes, it is reasonable to begin an evaluation of secondary hypertension by focusing on renal or renovascular causes.

DIAGNOSING RENAL ARTERY STENOSIS IN CHILDREN

Renal artery stenosis results in reduced arteriolar perfusion pressure and activation of the reninangiotensin-aldosterone system.3 The enzyme renin is released by the affected kidney, with subsequent activation of angiotensin II by angiotensin-converting enzyme (ACE). Angiotensin II causes hypertension by mediating arteriolar vasoconstriction and facilitating sodium reabsorption. In turn, the secretion of aldosterone, as a consequence of salt and water retention, further contributes to systemic hypertension. In adults, renal artery stenosis is often caused by atherosclerosis. In children and adolescents,
fibromuscular dysplasia is typically the underlying cause. An evaluation for renovascular disease should include an evaluation of any predisposing risk factors such as stigmata of neurofibromatosis or a history of admission to a neonatal ICU, which could be significant for complications of umbilical artery catheterization.

Doppler ultrasonography can be a useful noninvasive screening study; however, which diagnostic testing modality is preferred remains somewhat controversial. Standard angiography and digital subtraction angiography (DSA) are the gold standards for evaluation of renal artery stenosis. Despite relatively little data and experience in the pediatric population to date, CT angiography and magnetic resonance angiography are available as newer, less invasive alternatives for diagnosing renovascular disease. In patients who have renal artery stenosis of greater than 50%, CT angiography has been found to be as reliable as DSA in making the diagnosis. In this patient, the choice of CT angiography was based on a desire to simultaneously evaluate the renal arteries and image the abdomen and pelvis because of his complaint of vague abdominal pain.

CT angiography failed to demonstrate the stenosis. However, because of the high index of suspicion for renovascular disease in this patient, based on the high incidence of renovascular disease in children with secondary hypertension and on his response to an ACE inhibitor, we subsequently proceeded to perform DSA. DSA finally confirmed the presence of renovascular disease. While a thorough differential diagnosis and stepwise approach should guide any evaluation of a hypertensive child, even the most reliable diagnostic testing cannot supersede high clinical suspicion based on a complete history and physical examination.

It is important to note that although a response to an ACE inhibitor may provide a diagnostic clue, care should be taken in using ACE inhibitors in patients in whom renal artery stenosis is suspected, particularly in those with bilateral disease. Impairment of renal function, including instances of acute renal failure, has been reported in patients with renal artery stenosis who have undergone long-term ACE inhibitor therapy.

TREATMENT OF RENAL ARTERY STENOSIS IN CHILDREN

Alternatives to medical therapy for the management of renal artery stenosis include percutaneous transluminal angioplasty, with or without stent placement, and surgical bypass. Unlike in older patients who have atherosclerotic disease, patients with fibromuscular dysplasia rarely experience progression to complete stenosis. Thus, the potential benefit of revascularization in these patients is generally improved hypertensive control rather than prevention of renal failure. Percutaneous transluminal angioplasty has been shown to be an effective treatment in children with single artery involvement. Compared with surgery, angioplasty is less invasive and does not preclude subsequent surgical intervention in the event of angioplasty failure.

There are currently no prospective studies on the long-term prognosis of children who undergo revascularization for renal artery stenosis, nor have any follow-up guidelines been established. Although this is an area in need of further investigation, case reports have suggested that most patients do well and do not require further surgical intervention or medication management beyond the immediate postoperative period.

References:

REFERENCES:


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