Index of Suspicion

THE CASE

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EDITORS NOTE

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CASE 1 PRESENTATION

A previously healthy 4-year-old boy presents for evaluation of a unilateral nose bleed refractory to compression lasting for 1.5 hours. He has had episodes of epistaxis in the past secondary to nasal manipulation but none this severe. Three weeks ago he had a runny nose and congestion, which are now resolved. His family denies vision changes, headaches, nausea, hematuria, dysuria, any known ingestions, or any skin flushing. There is no history of bleeding disorders in his family.

On physical examination he is afebrile, with a heart rate of 121 beats per minute, a respiratory rate of 24 breaths per minute, a right arm blood pressure of 200/130 mm Hg, and an oxygen saturation of 99% on room air. His weight is 17 kg (50th percentile) and height is 112 cm (95th percentile). He appears well and has normal physical examination findings, with the exception of an abdominal bruit and barely palpable femoral pulses.

Initial laboratory results are as follows: white blood cell count, $21,900/\mu\text{L}$ ($21.9 \times 10^9/\text{L}$); absolute neutrophil count, $18,400/\mu\text{L}$ ($18.4 \times 10^9/\text{L}$); absolute lymphocyte count, $2100/\mu\text{L}$ ($2.1 \times 10^9/\text{L}$); hemoglobin, 9.9 g/dL (99 g/L); platelet count, $324 \times 10^3/\mu\text{L}$ ($324 \times 10^9/\text{L}$); mean corpuscular volume, 85 fL; red blood cell distribution width, 13.5%; an unremarkable peripheral blood smear; urea nitrogen, 45 mg/dL (16.1 mmol/L); creatinine, 1.39 mg/dL (123 $\mu\text{mol/L}$); and normal serum electrolyte levels. The results of thyroid function studies, coagulation profile, and urinalysis are normal. Renal ultrasonography with Doppler and head computed tomography results are interpreted as normal. Electrocardiography suggests left ventricular hypertrophy, and echocardiography reveals left ventricular hypertrophy. An additional imaging study helps establish the suspected diagnosis.

CASE 1 DISCUSSION

Abdominal ultrasonography revealed a long segment of abdominal aortic narrowing, involving the mesenteric and renal arteries (initial renal ultrasonography result was interpreted as normal), consistent with a diagnosis of midaortic syndrome (MAS). Abdominal magnetic resonance angiography (MRA) confirms this diagnosis.

Pediatric hypertension affects roughly 5% of all children. Typical symptoms include epistaxis, headaches, blurred vision, and vomiting; however, hypertension

may be asymptomatic in chronic processes. For this reason, blood pressure monitoring should begin on all children 3 years and older at every physician visit. Most pediatric hypertension cases are secondary to underlying renal, cardiac, urologic, endocrine, and neoplastic conditions. Approximately 10% of cases are secondary to renovascular anomalies, with renal artery stenosis accounting for most of these cases.

Differential Diagnosis

Epistaxis is commonly encountered by the pediatrician and is typically anterior or posterior in origin. Anterior nosebleeds account for most nosebleeds because of the highly vascular Kiesselbach plexus in this region. Anterior nosebleeds are typically caused by direct trauma, foreign bodies, mucosal dryness, infection, or irritants. Posterior nosebleeds are less common and may be the result of significant nasal trauma or potentially a tumor. Systemic causes include bleeding disorders, vasculitis, or hypertension as was found in our patient. Worrisome findings concerning nosebleeds are recurrent episodes and episodes refractory to routine management.

The Condition

MAS is an uncommon cause of renovascular hypertension in children. It results from narrowing of the abdominal aorta, typically involving the renal arteries and other visceral branches. Most cases of MAS are primarily idiopathic. However, it may be associated with underlying conditions, such as neurofibromatosis type I, mucopolysaccharidosis, William syndrome, Alagille syndrome, giant cell arteritis, or congenital rubella. Hypertension is present in most MAS cases. Claudication, oliguric renal failure, and intestinal ischemia occur infrequently. The classic diagnostic triad consists of an abdominal bruit, diminished or absent femoral pulses, and a discrepancy between blood pressures of the upper and lower extremities. The physical examination findings of diminished femoral pulses and blood pressure discrepancy between the upper and lower extremities can be seen in coarctation of the aorta; however, echocardiography typically detects this condition and is isolated to the aorta.

Once the diagnosis of MAS is suspected, MRA can confirm the diagnosis, with angiography remaining the gold standard. Aortic narrowing may also be seen on computed tomography angiography, abdominal ultrasonography, or echocardiography during the evaluation of hypertension. If MAS is suspected to be secondary, then a genetic evaluation should be considered to evaluate the patient for the potential underlying conditions mentioned above. In addition, evaluating for an inflammatory state by obtaining an erythrocyte sedimentation rate and C-reactive protein may help suggest giant cell arteritis as a cause of MAS.

Management

Hypertension secondary to MAS is typically severe and often requires numerous antihypertensive medications and/or surgical correction. Although hypertension can be controlled with several antihypertensive medications, surgery is the definitive treatment. Surgery is often indicated for unresponsive hypertension to medical therapy, claudication, intestinal ischemia, or when the survival of the kidney is in jeopardy. The decision for timing of surgical intervention is often case dependent. Most cases are managed by a single-staged aorto-aortic bypass of the diseased segment of the aorta along with a surgical revascularization procedure of the renal and splanchnic arteries as required.

Patient Course

On arrival, antihypertensive therapy was initiated for our patient. He was initially administered a nicardipine drip and clonidine patch with the goal of maintaining a higher systemic blood pressure to ensure adequate perfusion of his kidneys in the setting of acute kidney injury. Unfortunately, the patient became oliguric and developed progressively worsening renal failure, ultimately requiring hemodialysis. He subsequently developed respiratory failure and was intubated. After identifying MAS as the underlying cause of his hypertension, he was transferred to a facility experienced in surgical correction of this condition and underwent bilateral renal revascularization. Two months after surgical repair, he is normotensive and not taking any antihypertensive medications.

Lessons for the Clinician

- Epistaxis may be a presenting sign of hypertension.
- Routine blood pressure screening for children should begin at 3 years of age, which may identify asymptomatic hypertension.
- Midaortic syndrome is an uncommon cause of hypertension and can present with the triad of abdominal bruit, diminished lower extremity pulses, and a discrepancy in blood pressures between upper and lower extremities.

(Emily Vander Schaaf, MD, University of North Carolina, Chapel Hill, NC, Nicholas M. Potisek, MD, Wake Forest School of Medicine, Winston-Salem, NC)*

*Dr Vander Schaaf's current affiliation is Wake Forest School of Medicine, Winston-Salem, NC.

CASE 2 PRESENTATION

A 5-year-old boy is referred for a significantly elevated white blood cell count of $40,000/\mu\text{L}$ ($40.0 \times 10^9/\text{L}$), with 76% eosinophils (absolute eosinophil count of $30,400/\mu\text{L}$ [$30.4 \times$

$10^9/L$), 16% neutrophils, and 7% lymphocytes, and normal hemoglobin and platelet levels. The environmental health clinic has been monitoring his persistently elevated blood lead levels for the previous 6 months, with levels ranging from $10.4 \mu\text{g/dL}$ to $12.8 \mu\text{g/dL}$ (0.50 – $0.62 \mu\text{mol/L}$). The environmental health clinic had ordered the complete blood cell count to evaluate for anemia.

There is no history of weight loss, loss of appetite, cough, loose stools, photophobia, rash, focal respiratory symptoms, or cardiovascular symptoms. The patient's mother reports that the child has occasional abdominal pain and intermittent low-grade fevers. He was born prematurely (28 weeks) and had a brief stay in the neonatal intensive care unit for feeding difficulties. The mother also reports that the child has behavioral issues, notably head-banging, hand-to-mouth behaviors, and pica. There is no significant family or travel history. His twin has an elevated blood lead level but a normal complete blood cell count. They have a pet dog and cat; he frequently plays in the dirt and in a sandbox at his daycare.

On physical examination, the patient has nonfocal examination findings, pertinently negative for any lymphadenopathy, rash, abnormal pigmentation, masses or lumps, or evidence of malnutrition. Laboratory findings include a normal serum electrolyte level and a uric acid level of 2.9 mg/dL ($170.6 \mu\text{mol/L}$), mildly elevated lactate dehydrogenase (1090 IU/L), normal IgE (97 IU/mL), and a stool specimen negative for ova and parasites. A T-cell receptor test result for V_β (variable segments of the β chain of T-cell receptor) expression is negative for monoclonality and, hence, for hematologic malignant neoplasms. His chest radiographic findings are normal. The results of computed tomography of the chest, abdomen, and pelvis are normal; echocardiography reveals no evidence of eosinophilic myocarditis. Additional evaluation reveals the cause of his eosinophilia.

CASE 2 DISCUSSION

An infectious diseases consultant recommended serologic testing for *Toxocara* and ophthalmologic examination by slitlamp to rule out ophthalmologic larva migrans (OLM). The *Toxocara* serologic test (IgG by enzyme-linked immunosorbent assay) result was positive, with titers of 0.304 optical density (OD) at presentation (reference range, 0 – 0.299 OD). Ophthalmologic examination did not reveal evidence of OLM.

Differential Diagnosis

The initial differential diagnoses in a child presenting with severe hypereosinophilia includes neoplasm (including acute

myeloid leukemia, acute lymphoblastic leukemia, and Hodgkin lymphoma); allergic disorders, such as atopic dermatitis, asthma, and various forms of rhinitis; Addison disease; collagen vascular diseases (including vasculitides and eosinophilia-myalgia syndrome); hypereosinophilic disorders (including mastocytosis); parasitic infections (including *Trichinella*, schistosomiasis in travelers, and, rarely, fungal infections, such as aspergillosis and coccidiomycosis); and medication effects. The mnemonic NAACP (Neoplasms, Allergic disorders, Addison disease, Collagen vascular diseases, Parasitic infestations) is often used as a memory aid.

In our patient, initial evaluation was not concerning for malignant neoplasms, increased cell lysis, myocarditis, or intestinal parasites. However, the patient had a history of pet exposure and pica, further raising red flags for parasite exposure.

The Condition

Eosinophilia is defined as eosinophil percentage greater than 3% to 5% in the peripheral blood, with severity corresponding to absolute eosinophil count: mild, eosinophil count of 600 to $1500/\mu\text{L}$ ($1.5 \times 10^9/L$); moderate, eosinophil count of 1500 to $5000/\mu\text{L}$ (1.5 – $5.0 \times 10^9/L$); and severe, eosinophil count greater than $5000/\mu\text{L}$ ($>5.0 \times 10^9/L$). Primary eosinophilia may be due to various myeloid and lymphoid neoplasms. However, secondary eosinophilia, due to infections and other systemic diseases, such as collagen vascular disorders, pulmonary eosinophilic diseases, allergic disorders, and metabolic diseases, must be excluded first.

Toxocariasis is a syndrome caused by the nematode *Toxocara canis* or *Toxocara cati*. The dog and the cat are the definitive hosts for *T canis* and *T cati*, respectively. Eggs are shed in stool by the animal, become infective after approximately 3 weeks (after embryonation), and remain infective for several years in the environment. After ingestion by dogs or cats, the eggs hatch and the larvae penetrate the gut wall; migrate through the lungs, the bronchial tree, and the esophagus; and develop into adults in the small intestine, from where they are shed in stool. Humans are the unintentional hosts. Transmission is fecal-oral, by ingestion of the eggs or larvae, through contaminated hands or other objects, or by ingestion of contaminated soil.

Toxocariasis is classically characterized by eosinophilia, hepatomegaly, fever, and hyperglobulinemia due to the systemic penetration of the worm larvae. Toxocariasis is now formally classified into the classic systemic form, compartmentalized (neurologic larva migrans), or OLM, covert and asymptomatic forms. Several authors have reported significant prevalence rates of asymptomatic infection, ranging from 5% in the white pediatric population to 25% in the

African American pediatric population. Further seropositivity for *Toxocara* in various developed countries varies from 0.7% in New Zealand to 7.5% in Australia and up to 14% in the United States. Seroprevalence is higher in tropical countries (30% in Nigeria, 36% in Brazil, and up to 81% in Nepal). The high rate of seroprevalence in the United States, compared with other industrialized nations in Europe, may be due to asymptomatic toxocariasis, requiring a heightened awareness of the potential for OLM and need for treatment.

Management and Prevention

Treatment of clinical features and to prevent complications, such as OLM and neurologic larva migrans, is recommended in all forms except the asymptomatic, in which only preventive treatment may be considered in the case of positive serologic test results and significant eosinophilia. Treatment is typically with anthelmintics: albendazole, mebendazole, or thiabendazole. Albendazole, 15 mg/kg daily, divided into 2 doses, for a 5-day course, is considered first-line therapy. Treatment may also include corticosteroids and anti-inflammatory agents to counter the inflammatory reaction produced when the organism is killed. OLM treatment includes anti-inflammatory agents, and the patient may also require procedural intervention to remove or dissolve the eosinophilic granulomatous lesion.

Good hygiene practices, such as hand washing (especially after contact with pets or high-risk areas, such as playgrounds and sandboxes), timely disposal of pet feces, and routine deworming of pets, are critical for prevention of toxocariasis.

Patient Course

Our patient's condition may be classified into either an asymptomatic or covert form based on his symptoms of abdominal pain. The significant eosinophilia and the high titers of anti-*Toxocara* antibodies prompted treatment to prevent complications. He was prescribed mebendazole therapy, 100 mg twice daily for 5 days, because albendazole was not available at his pharmacy. A follow-up white blood cell count 4 weeks later was significantly improved at 10,000/ μ L (10.0×10^9 /L), with 22% eosinophils and an absolute eosinophil count of 2230/ μ L (22.3×10^9 /L). Follow-up *Toxocara* antibody levels were elevated at 0.585 OD; however, his complete blood cell count continued to normalize. Most recently, his lead level was 9.3 μ g/dL (0.45 μ mol/L), and the complete blood cell count was significant for a white blood cell count of 9100/ μ L (9.1×10^9 /L), with 13% eosinophils (absolute erythrocyte count of 1180/ μ L [11.8×10^9 /L]), 28% lymphocytes, and 58% neutrophils.

We never positively identified the source of the infection but strongly suspected the sandbox at the daycare. The cats

getting into it were strays, and the family and the health department tried to convince the owner to cover the sandbox when not in use, but without effect. Ultimately, the children stopped going to that daycare. The pets were never tested or treated. Unfortunately, the health department jurisdiction where the case occurred has a limited budget for enforcement activities related to environmental health.

Lessons for the Clinician

- Toxocariasis is an important cause of asymptomatic eosinophilia in children, especially in a situation with risk factors such as pica, pet exposure, and poor hand hygiene.
- Preventive measures should be included in the anticipatory guidance and counseling of parents to prevent toxocariasis in such settings, such as hand washing and timely disposal of cat and dog feces.

(Kriti Puri, MD, Elizabeth P. Schlaudecker, MD, MPH, Nicholas C. Newman, DO, MS, Cincinnati Children's Hospital Medical Center, Cincinnati, OH)

CASE 3 PRESENTATION

A 3-month-old full-term girl with trisomy 21 presents to the gastroenterology clinic with progressive feeding intolerance. For the past 3 weeks, she has had large nonbloody spit-ups after every feed, either formula colored or light brown in appearance. She has a normal appetite and does not appear to be distressed after spit-ups. Her parents report that she previously tolerated 3 oz of formula every 3 hours. She has been stooling regularly without diarrhea and has no other signs of illness. Birth weight was 3.85 kg (80th percentile). She has a history of a small ventricular septal defect and congenital bilateral clubfoot.

Physical examination reveals a well-appearing girl with typical Down syndrome dysmorphic features. Her temperature is 37.2°C, pulse is 89 beats per minute, respiratory rate is 22 breaths per minute, blood pressure is 97/56 mm Hg, and oxygen saturation is 97% on room air. Her perfusion is normal. She currently weighs 3.93 kg (less than the third percentile), her length is 57.5 cm (25th percentile), and her head circumference is 39 cm (25th percentile). She has normal heart sounds with a 2/6 systolic murmur over the left sternal border. Her abdomen is soft, nondistended, and nontender with normal bowel sounds. There are no abdominal masses or hepatomegaly. She has normal female external genitalia without lesions or hernias.

The result of a basic electrolyte panel is normal. A pyloric ultrasonography result is normal. An upper gastrointestinal

(GI) tract series reveals distension of the stomach and duodenal bulb with a transition point at the level of the proximal duodenum and gastric stasis with reflux of residual gastric contents. Surgical exploration reveals the ultimate diagnosis.

CASE 3 DISCUSSION

Exploratory laparotomy revealed an annular pancreas, causing extrinsic duodenal compression. The patient underwent duodenal duodenostomy from which she recovered well and subsequently tolerated oral feeds.

Diagnosis of Feeding Intolerance

The most common cause of recurrent spit-ups after feeds is gastroesophageal reflux. However, this is typically seen in otherwise healthy infants as a result of intermittent lower esophageal sphincter relaxation, leading to retrograde flow of gastric fluid. The clinician must recognize red flags in the history and physical examination to determine whether further workup is necessary; these signs include poor weight gain, bilious emesis, repeated large volume emesis, respiratory distress, and abdominal distension.

In the face of persistent spit-ups after feeds associated with poor weight gain, the clinician must consider the possibility of proximal GI obstruction. This can occur at any point from the esophagus to the duodenum, keeping in mind that obstructions past the ampulla of Vater at the junction between the first and second portions of the duodenum typically result in bilious emesis. The most common cause of proximal GI obstruction in early infancy is pyloric stenosis, which can be excluded by sonography. The most serious cause of intestinal obstruction to consider in infancy is malrotation because an associated volvulus can lead to bowel ischemia due to compression of the superior mesenteric artery. Additional causes to consider are antral webs, duodenal stenosis, and annular pancreas. Flat and upright abdominal radiographs may be helpful if obstruction is suspected. The absence of a gastric bubble may suggest a left upper quadrant mass, displacing the stomach to the right. A distended stomach bubble would be more consistent with a gastric outlet or duodenal obstruction. In the absence of an acute surgical abdomen, an upper GI tract radiocontrast study is helpful to define the anatomical structures of the patient. An upper GI tract study should be performed when the infant's predominant symptom is bilious vomiting because midgut volvulus may be catastrophic and early surgical intervention is essential.

Non-GI tract causes of emesis in an infant should also be considered, depending on the clinical situation. These causes

include congenital heart disease with congestive heart failure, milk protein intolerance, metabolic disorders, renal disorders, and significant elevations in intracranial pressure.

The clinician must have a higher suspicion for anatomical anomalies in patients with underlying genetic syndromes, such as trisomy 21 as in this patient. The GI symptoms that may be common in infancy, such as gastroesophageal reflux and constipation, may in fact represent symptoms related to underlying anatomical defects, such as duodenal malformations or Hirschsprung disease. Approximately 15% of patients who have trisomy 21 have a congenital obstructive GI tract abnormality. Five percent of patients with trisomy 21 will have duodenal atresia or annular pancreas.

The Condition

Annular pancreas is a relatively rare congenital condition, with an incidence of 1 in 2000 live births. In 70% of cases, annular pancreas is associated with other anomalies, such as esophageal atresia, imperforate anus, congenital heart disease, and malrotation. Exact embryonal origins are unclear, but the leading theory is that it results from aberrant rotation of the ventral portion of the pancreas, preventing its fusion with the dorsal portion. This causes wrapping of the pancreas 270° around the duodenum, typically at the second portion as the duodenum descends in the abdomen.

Most patients with annular pancreas remain asymptomatic through their lifetime. However, of those children who are symptomatic, more than two-thirds present as infants with signs of obstruction, including bilious emesis, feeding intolerance, and abdominal distension. When presenting in later childhood and adulthood, patients experience symptoms of peptic ulcer disease, duodenal obstruction, or pancreatitis.

Imaging studies of symptomatic infants, such as abdominal radiography, may reveal the classic "double bubble" sign consistent with duodenal obstruction. An upper GI tract series can reveal associated findings, such as a transition point between the first and second parts of the duodenum. Endoscopic retrograde cholangiopancreatography, magnetic resonance cholangiopancreatography, and endoscopic ultrasonography are more recent additional modalities that can be used to diagnose annular pancreas. The gold standard of diagnosis is exploratory laparoscopy or laparotomy.

Management

The first step in treating a patient with an apparent upper GI tract obstruction is to provide gastric decompression through the placement of a nasogastric tube. Intravenous rehydration and correction of any electrolyte abnormalities should be performed. Surgical repair is the ultimate treatment for patients with annular pancreas to relieve the

external compression it imposes on the duodenum. This involves bypass procedures, such as duodenoduodenostomy, gastrojejunostomy, or duodenojejunostomy, which are favored over pancreatic resection due to its associated complications, including pancreatitis and pancreatic fistula formation.

Lessons for the Clinician

- Clinicians must have a high suspicion for intestinal obstruction in patients with persistent feeding intolerance when their emesis is nonbilious and associated with poor weight gain.

- In patients with existing genetic syndromes, such as trisomy 21, rare anatomical anomalies, such as annular pancreas, should be considered.

(Patricia M. Notario, MD, Rajeev Nagpal, MD, Advocate Children's Hospital, Oak Lawn, IL)

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