American Journal of Emergency Medicine 33 (2015) 1844.e3–1844.e6

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Case Report

Facial nerve palsy in a 3-year-old child with severe hypertension

Abstract

We report an interesting case of a child with new-onset malignant hypertension (HTN) associated with facial paralysis. A review of the medical literature on this association and discussion of diagnostic and management aspects are included.

A 3-year-old Pakistani boy was transferred to the emergency department (ED) from his pediatrician's office due to concern for facial weakness. He had presented to the ED 2 days prior with a chief complaint relayed by his father of "swollen face; we see it more when he talks," which occurred several days after rhinorrhea and congestion. At that time, he was afebrile and well appearing (there was no blood pressure [BP] measurement); there was noted mild right facial swelling without erythema, tenderness, fluctuance, or rash. Cranial nerve function was intact. He was diagnosed with right facial swelling (due to probable allergic reaction) and discharged home with pediatrician follow-up.

Two days later, there was no improvement. He was evaluated by his pediatrician, who noticed unilateral facial muscle weakness and referred him to the ED. On second ED visit, parents denied patient had fever, rash, pain/headache, visual deficit, eye redness/ discharge, ear pain, or alteration in behavior/level of activity/mental status.

Perinatal history was unremarkable. Family history was positive for consanguinity (parents were cousins) but negative for HTN. Medical history was significant for thalassemia, eczema, and food allergies. Parents denied any recent travel, rashes, insect/tick bites, or known sick contacts affecting the patient. Immunizations were up to date for age. The patient was not taking any current medications.

On physical examination, he was well appearing, active, smiling/playful, in no apparent distress. He had mildly dysmorphic features with triangular facies, frontal prominence, low set ears, anteverted nostrils, and micrognathia. Developmentally, his body mass index was below the third percentile. He had right-sided upper and lower facial paralysis, specifically with weakness affecting the right corner of his mouth, right eyelid, and right forehead musculature. His vision was grossly normal, with intact extraocular movements and reactive pupils. With the exception of cranial nerve 7, cranial nerves 2 to 12 were intact. He had normal strength and sensation in all 4 extremities; deep tendon reflexes were 3+ and equal bilaterally. Head ears eyes nose throat examinations were within normal limits; specifically, ophthalmologic examination was without retinal abnormality or papilledema, and there was no evidence of acute otitis media. His neck was supple with no visualized or palpable masses. Cardiac examination was normal, lungs were clear to auscultation, and abdomen was soft and nontender with no masses or bruit.

Blood pressure measured at triage was 224/112; heart rate, 99 beats per minute; respiratory rate, 24 breaths per minute; peripheral oxygen saturation, 98% (in room air); rectal temperature, 97.6°F. His BP was measured 5 times over the next hour with readings of 194/110, 205/124, 195/124, 195/96, and 187/111. Blood pressure was measured in all 4 extremities, and there was no discrepancy in hypertensive readings.

The initial differential diagnosis included hypertensive emergency; central nervous system (CNS) pathology (stroke, central nervous system tumor, and encephalitis); malignant HTN due to cardiac, endocrine, or renal disease; and Bell's palsy.

The patient was placed on a cardiac monitor, and peripheral venous access was obtained. The initial priority was to therapeutically lower the BP by 10% to 20% while under ED supervision, before leaving the department to perform radiographic imaging. He initially received a dose of oral nifedipine 6 mg (0.5 mg/kg), with little change in BP. He next received intravenous (IV) labetalol 20 mg (1 mg/kg), with decrease in BP to 153/87. Upon BP reduction, he was transported to radiology for a cranial/facial computed tomography (CT). The CT scan was normal. Upon return to the ED, his BP was 177/121; he was given another dose of oral nifedipine 6 mg (0.5 mg/kg), after which his BP declined to 147/88. He was admitted to pediatric intensive care unit.

Emergency department workup was remarkable for urinalysis showing trace protein and moderate hemoglobin. Basic serum metabolic panel showed sodium 139 mmol/L, potassium 3.9 mmol/L, chloride 101 mmol/L, bicarbonate 24 mmol/L, blood urea nitrogen 9 mg/dL, creatinine 0.2 mg/dL, and glucose 132 mg/dL. Complete blood count showed white blood cell 10 000/mm^3, hemoglobin 13.9 gm/dL, hematocrit 43%, and platelet count 251 000/UL. Venous blood gas showed pH 7.42, $Paco_2$ 38 mm Hg, Pao_2 60 mm Hg, and lactate 2.2 mmol/dL. A chest x-ray showed clear lungs and normal heart size and contour.

Upon admission to pediatric intensive care unit, the patient had persistent marked elevation of BP despite receiving multiple IV doses of labetalol. An arterial line placed for continuous BP monitoring, and a nicardipine drip (1 μg/kg per minute) was initiated. Abdominal sonogram showed a relatively small left kidney (5.7 cm) compared to the right kidney (8.2 cm) with normal renal echogenicity; there was no hydronephrosis, mass, or stone. A CT angiogram of the abdomen with IV contrast was performed, which showed asymmetric left renal atrophy (Fig. 1) and decreased caliber of the left renal artery compared to the right (Fig. 2), although no focal stenotic segment was identified.

During hospitalization, he underwent cardiac, infectious, rheumatologic, nephrogenic, endocrinologic, and genetic workup. Echocardiogram showed moderate left ventricle hypertrophy but no evidence of coarctation of the aorta. Infectious workup showed no growth in blood, urine, and throat cultures; Lyme serology, liver function test, and coagulation studies were within normal limits. Serum, anti-streptolysin O titre, C3, and C4 were within normal limits as well as serum angiotensin-converting enzyme and aldosterone levels. Serum renin

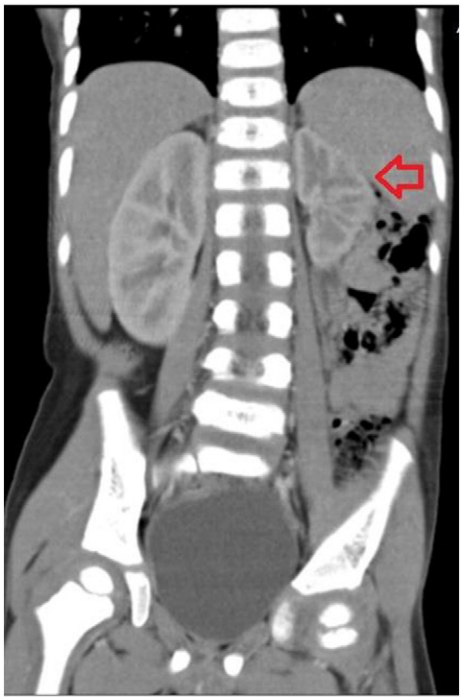

Fig. 1. Computed tomographic angiogram of abdomen, coronal view, showing smaller left kidney (arrow).

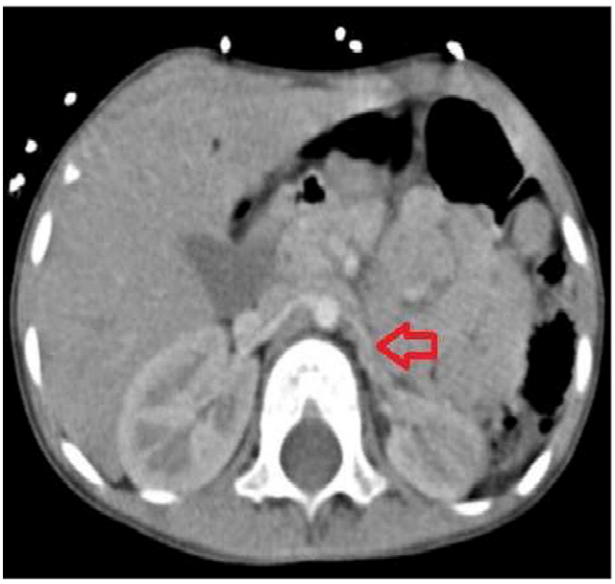

Fig. 2. Computed tomographic angiogram of abdomen, sagittal view, showing narrowed caliber of left renal artery (arrow) and smaller left kidney.

level was significantly increased 33 ng/mL per hour (0.25-5.82). Urine homovanillic acid concentration was within normal limits; there was an upward trend in serial vanillylmandelic acid urine concentrations with time (8.7 to 9.3 to 15.4 mg/g creatinine). Serum thyroid-stimulating hormone and T4 and lipid profile were within normal limits; serum cortisol level in the morning was 2.5 μg/dL. Genetic studies were unremarkable.

Over the next 7 days, his HTN persisted; despite initiating multiple oral antihypertensive drugs, including oral enalapril, labetalol, and amlodipine, he could not be weaned from IV antihypertensive medication. At that point, it was decided to transfer the patient to another facility that had the capability for vascular intervention to treat pediatric renal artery stenosis.

During hospitalization, the patient's facial paralysis gradually improved and was much less apparent at the time of transfer. At the transfer facility, a renal artery angiogram confirmed left renal artery narrowing without discrete focal stenosis. Angioplasty was performed but was unsuccessful in resolving his HTN. A renal artery stent was then placed; despite this, the patient required triple antihypertensive therapy of amlodipine, labetalol, and clonidine to control his HTN. Eventually, his facial palsy completely resolved approximately 1 month after presentation.

Acquired facial nerve palsy (FNP) is a relatively uncommon pediatric condition, affecting approximately 5 to 10 per 100 000 children per year [1]. Although sometimes associated with an underlying infectious, traumatic, malignant, or HTN etiology [2], the most common cause of acute unilateral facial weakness is idiopathic Bell's palsy (75% of cases) [3]. Although the diagnosis of Bell's palsy depends on exclusion of other etiologies, acute-onset FNP is often readily diagnosed and treated as idiopathic without an extensive workup when a child presents with isolated unilateral facial weakness.

There is a paucity of medical literature characterizing the interesting clinical association of FNP with HTN in children. Most are single case reports [4-20]. To our knowledge, only 1 comprehensive published series exists, which reviewed 26 published cases of FNP associated with malignant HTN reported over a 50-year span (23 cases involved children) [20]. In all, HTN was associated with underlying renal pathology in 10 pediatric cases (most commonly an associated congenital anomaly), a cardiovascular lesion in 4 cases, and endocrinopathy in 1 case. Interestingly, 3 cases occurred in conjunction with Guillain-Barré syndrome, presumably due to either autonomic instability or hyperreninemia [21]. Many of these cases did not have neuroimaging performed to assess for associated neurologic lesion. Of those with renal disease, only 2 cases involved children with renal artery stenosis (RAS). More than 90% of patients had complete or nearly complete resolution of the FNP within the range of a few days to up to 12 months.

Our case involves a young child with new-onset FNP due to underlying RAS-induced HTN. The RAS was likely due to fibromuscular dysplasia. Renal artery stenosis causes reduced renal arteriolar perfusion pressure, with activation of the renin-angiotensin-aldosterone system. The HTN was discovered serendipitously via routine vital sign measurement at ED triage. Because of young patient age, BP had not been routinely measured either a few days earlier at initial ED visit or at follow-up with the pediatrician 2 days later. Blood pressure

Table 1
Common etiologies of pediatric hypertension by age

Infants	Ages 1-6 y	Ages 7-12 y	Adolescents
Thrombosis of renal artery or vein	Renal artery stenosis	Renal parenchymal disease[a]	Essential HTN
Congenital renal anomaly	Renal parenchymal disease[a]	Renovascular abnormalities	Renal parenchymal disease[a]
Coarctation of the aorta	Wilms tumor	Endocrinopathy[b]	Endocrinopathy[b]
Bronchopulmonary dysplasia	Neuroblastoma	Essential HTN	
	Coarctation of the aorta		

[a] Considerations include glomerulonephritis, reflux nephropathy, and congenital anomalies.
[b] Considerations include hyperthyroidism, Cushing syndrome, pheochromocytoma, and hyperaldosteronism.