A Case Study

Hypertension Presenting As Bell's Palsy in Children

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Abstract

Background Bell's palsy is a rare symptom of hypertension. We describe a case of childhood hypertension of underlying renovascular etiology presenting as recurrent Bell's palsy.

Case Presentation A 2-year-old male was admitted to hospital for severe hypertension. His solepresenting symptom was recurrent episodes of Bell's palsy The elevated blood pressure was noted incidentally during anaesthesia assessment for magnetic resonance imaging of the brain. He was otherwise healthy, and had not had blood pressure measurementedd during assessments prior to hospitalilization. On admission, blood pressure was 220/120 mm Hg, with evidence for moderate concentric left ventricular hypertrophy indicating both a chronic process and end organ damage from his hypertension. The patient's blood pressure was initially managed with labetalol and nitroprusside, and later he was converted to enalapril (0.5 mg/kg/day), amlodipine (0.5 mg/kg/day), minoxidil (0.625 mg/kg/day) and metoprolol (2.5 mg/kg/day). A duplex right kidney with stenosis of the cranial of the two renal arteries was diagnosed using 99mTc MAG3 scintigraphy with ACE inhibitor, Doppler renal ultrasound and MR angiography.) The patient is awaiting angiography for further management while being normotensive on the medications named above.

INTRODUCTION

Bell's palsy is an acute lower motor neuron cranial nerve (CN) VII paralysis. CN VII is responsible for control of the salivary and lacrimal glands, motor function of the facial muscles, as well as of the stapedius muscle in the middle ear. Bell's palsy often present with rapid onset of partial or complete paralysis of the facial muscles. On physical exam, patients are unable to raise the eyebrow or tightly close the eye on the affected side.

The nasolabial fold is absent, the forehead is flattened, and the mouth may be drawn to the unaffected side with smiling. The remainder of the neurological exam is normal [1, 2].

The incidence of CN VII palsy is 2.7/100,000 child population per year in children less than 10 years of age, and most are peripheral, involving the forehead. There is a long list of differential diagnosis for Bell's palsy in the pediatric population. The most common causes include infection, trauma, congenital abnormalities and neoplasms [2]. Table 1 lists the various etiologies [2, 3]. Most reviews on acquired CN VII palsy include hypertension as a rare cause with few studies actually focusing on Bell's palsy as the presenting symptom [1]. Between 1943-1950 in Boston, 2 of 37 cases (5.4%) of Bell's palsy were attributed to hypertension [4]. In 1966, Lloyd reviewed ten years of cases of lower motor neuron paralysis in the Hospital for Sick Children in London, England. 7 of 87 patients (6%) had underlying hypertension. Of 35 patients treated for hypertension during this time, only 3 presented with Bell's palsy.

The paralysis occurred when the blood pressure was elevated, and resolved with its management [5]. Most recently, in 1990, Siegler et al noted 3 cases of severe hypertension presenting with Bell's palsy. The elevated blood pressure coincided with the facial paralysis. Each patient then received a full hypertension workup, with two cases being attributable to renal anomalies [6]. Conflicting recommendations exist on the work up and management of Bell's palsy. Otoscopy is necessary,and if auditory acuity is unclear, an audiogram should be performed. Most authors recommend a complete blood count to screen for malignancy, and a lumbar pucture if meningitis or Guillain-Barre syndrome are suspected. If any neurological features are identified or if malignancy is probable, radiological

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imaging by either computed tomography (CT) or magnetic resonance imaging (MRI) should be done [3]. Furthermore, several authors recommend a blood pressure measurement, given the possibility, albeit rare, of hypertension-related etiology [2, 3, 6]. Management includes the use of steroids and/or antiviral agents, although it remains controversial as to whether this significantly improves outcome. Referral to an appropriate specialist is indicated for additional symptoms including hearing loss, associated neurological abnormalities, progression of paralysis beyond three weeks, recurrence of episodes, and hypertension [2].

Table 1. Diverse etiologies of acquired facial nerve paralysis

Infective or inflammatory
Otitis media
Mastoiditis
Herpes zoster
Temporal lobe abscess
Varicella
Mumps
Meningitis
Encephalitis
Mycoplasma
Cat scratch disease
Kawasaki disease
Guillain–Barre syndrome
HIV
Lyme disease
Trauma
Facial burn
Basal skull fracture
Blunt and penetrating trauma
Facial surgery
Neoplastic
Leukaemia
Cerebellar astrocytoma
Rhabdomyosarcoma
Haematological
Haemophilia
Histiocytosis
Congenital
Melkersson–Rosenthal syndrome
Osteopetrosis
Intracerebral arteriovenous malformation
Other
Hypertension

Case Presentation

We describe a 2-year-old male who first presented to the emergency department at one year of age with Bell's palsy. He had recurrent episodes of facial paralysis occurring seven times in fifteen months. Each episode lasted one to two weeks and resolved without treatment.

The patient remained asymptomatic between episodes. Most commonly, the left side of the face was involved, however, at least once deficits occurred on the right. Findings were never bilateral. Episodes presented as drooping of the eye, cheek and corner of the mouth. The patient had no weakness of the ipsilateral limbs, nor history of ataxia, vision changes or rashes. He did not have headaches, fever or neck stiffness. There was no history of tic bites and the family resides in an area without documented lyme disease. On one occasion, an associated sore throat was reported, but otherwise no preceding viral illnesses were recorded. There was no history of trauma or travel. The past medical history was non-contributory. He was born at term with no birth trauma, and had no prior hospitalizations or surgeries.

In investigation of the described episodes, the patient was booked for acranial MRI. On the day ofthe scheduled imaging, his blood pressure measured 220/120 mm Hg. His concurrent clinical exam was otherwise completely benign, without evidence of facial drooping. He was admitted to the critical care unit for the management of his hypertension. The admitting diagnosis was urgent hypertensive crisis. He had not had his blood pressure measured at any previous visits to the emergency room or by his pediatrician for comparison.

A workup for his hypertension was completed. An echocardiogram showed moderate concentric left ventricular hypertrophy, indicating that his hypertension was a chronic process, which had caused end-organ damage. Fundoscopy was normal. A renal ultrasound was completed and the patient was found to have two kidneys, with a duplex right kidney and no hydronephrosis.

Bloodwork included a normal complete blood count and normal urea and creatinine. Serum aldosterone was undetected and renin was normal. A workup for pheochromocytoma was completed, including urine metanephrines and catecholamines, which were normal. Neuroblastoma was ruled out with a normal urine homovanillic acid and vanillylmandelic acid. Further endocrinology workup included a normal TSH and cortisol level. A chest x-ray was normal, with no cardiomegaly or mediastinal mass.

Figure 1. MR Angiography showing two right renal arteries (enhanced image, unfortunately higher resolution not possible).

Further imaging including an MR angiogram showed 2 right-sided renal arteries (with separate ostia), but spatial resolution was insufficient to demonstrate stenosis in one of the two vessels (Figure 1).

A 99mTc MAG3 renogram was done while the patient was on an ACE inhibitor with the intent to rule out renal stenosis. However there was delayed excretion of tracer (significant retention of tracer) in the upper pole of the right kidney (Figure 2). Differential function favored the left kidney, with renal uptake of 59% compared to 41% to the right kidney.

This led to a repeat renal ultrasound with Doppler in deep sedation. There was a significant difference in the peak velocities between the right upper renal artery (up to 158 cm/s), compared to the lower right renal artery and left renal artery. On a later scan, a peak velocity of up to 472 cm/sec was seen from the right upper renal artery. The ultrasound findings, similar to the nuclear medicine scan, were also compatible with a renal artery stenosis of the right upper renal artery (Figure 3). Therefore, we concluded that renal artery stenosis of the upper pole of the right kidney was likely the

underlying cause for the hypertension.

Figure 2. Abnormal 99mTc Mag-3 renal scan showing diminished differential renal function to the right (41% vs. 59%) and delayed excretion of isotope, which in the absence of any hydronephrosis is typical for renal artery stenosis (positive captopril scan). Initial management for his hypertension included labetolol and nitroprusside infusions, after which he was transitioned to a four drug regimen of amlodipine 2.5 mg BID (0.31 mg/kg/day), enalapril 2.0 mg daily (0.125 mg/kg), metoprolol 8 mg BID (1 mg/kg/day) and minoxidil 4mg BID (0.5 mg/kg/day). Initially, his blood pressure remained at 145/76 mm Hg, greater than the 95th percentile for his age. His blood pressure medications were optimized with amlodipine increased to 4 mg BID (0.5 mg/kg/day), enalapril increased to 3.5 mg BID (0.44 mg/kg/day), metoprolol increased slowly to 40 mg BID (5 mg/kg/day) and minoxidil increased to 5mg BID (0.625 mg/kg/day). By discharge from hospital, his blood pressure was below the 90th percentile for age.

At his last follow-up, three months after presentation, the patient's blood pressure was 96/45 mm Hg, approximately at the 50th percentile for his height. Given the improvement of his blood pressure, his medications have since been adjusted to minimize side effects. The patient is awaiting an angiography with the question about feasibility of a right heminephrectomy to alleviate the hypertension. To date, the Bell's palsy has not recurred.

Figure 3 (a, b, c): Right lower renal artery and left renal artery showing normal peak velocity of 56.4 and
89.9 cm/s, whereas peak velocities of up to 472 cm/s were seen in the upper right renal artery.

DISCUSSION

Bell's palsy is a known, but rare, presentation of underlying hypertension [2]. In our case, the patient had recurrent episodes of Bell's palsy, without having his blood pressure measured at any time prior to his pre-MRI anesthesia assessment. At that time, he had already developed end-organ damage with left ventricular hypertrophy. Given the incidence of Bell's palsy and the low proportion of patients with hypertensive etiology, it may be deemed reasonable to assume a post-viral cause. However, in our case there was a clear underlying renal anomaly, and the resultant hypertension required urgent management.

Siegler, et al. [6] presented similar cases in 1990. In one, akin to our patient, a ten-year-old presented with four episodes of Bell's palsy over eleven months. Only on her fourth presentation and at her pre-operative assessment, was her blood pressure measured and found to be severely elevated. An underlying explanation for her hypertension was never found. The two other patients reported by Siegler did not have recurrent episodes, but, similar to our patient, were both found to have a renovascular cause for their hypertension. One underwent a left nephrectomy, which partially controlled the blood pressure, while the other was maintained primarily on medication, as both of her kidneys were small and contracted [6]. Unlike the aforementioned cases, our patient did not have symptoms of his Bell's palsy at the time that he was diagnosed with urgent hypertensive crisis.

Siegler's [6] comments however support this feature of our case, as he states that the paralysis can be intermittent and independent of blood pressure control. Interestingly, recurrence of facial nerve paralysis has yet to occur in our patientsince establishing normotension. Measuring blood pressure in the presentation of Bell's palsy is particularly important in its implications for management. Corticosteroids are one of the two widely used treatments for Bell's palsy. Various studies have examined the effectiveness of corticosteroids in time to recovery and ability to fully recover without residual weakness. Not all studies have demonstrated significant difference between steroids and placebo; therefore no conclusive recommendations could be given [1]. However, given corticosteroids are not uncommonly administered for patients with Bell's palsy, the potential morbidity of worsening hypertension during a hypertensive crisis cannot be overstated [2]. Fortunately, in this case, the patient did not receive any treatment for his recurrent episodes. Once the patient's hypertension was identified, he had a complete workup for the underlying etiology. The standard workup includes the following [7]: Cushing syndrome can be ruled out with normal cortisol levels, as well as hyperthyroidism with a normal TSH. Normal plasma aldosterone and renin rule out mineralocorticoid excess. Pheochromocytoma can be ruled out with normal plasma and urine catecholamine levels.

Mal disease can be ruled out with normal urea and creatinine. Other tests can be done if there is an indication of drug-induced or primary hypertension or sleep apnea. Initial standard imaging includes an echocardiogram to rule out coarctation of the aorta, and to assess for end organ damage with left ventricular hypertrophy. A renal ultrasound with Doppler must be done to ensure normal kidney size with no congenital abnormalities, and to examine vasculature and assess renal artery blood flow [8]. Our patient had the aforementioned workup, but it was only the imaging, rather than any laboratory testing, thatled to the diagnosis.

Infants and young children are more likely to have secondary hypertension rather than primary, with renovascular disease and renal parenchymal disease account for the majority of the cases. Renovascular disease is the less common of these two entities, only accounting for 5-10% of childhood hypertension overall. As was the case with our patient, when renovascular disease is unilateral, the urea and creatinine remain normal through compensation by the healthy kidney [9]. We essentially used a Captopril renal scintigraphy for the diagnosis of renovascular hypertension [10]. Administration of an ACE inhibitor leads to a decrease in glomerular filtration pressure and a prolonged transit time of tubular agents (99mTc-MAG3). In adults, this test is a predictor of blood pressure reduction after surgery or angioplasty [11].

The patient's management followed the accepted approach for urgent hypertension, later redefined as malignant with completion of the echocardiogram. He was started on an intravenous drip of labetolol and nitroprusside. Labetolol, an alpha and beta sympathetic blocker, reduces

peripheral vascular resistance, and is the drug of choice for malignant hypertension in infants and young children [12]. Sodium nitroprusside, an arterial and venous vasodilator, has been used for many years in the treatment as well. Other options include nicardipine, hydralazine, fenoldopam and clevidipine [13]. Once renovascular hypertension was identified, and the acute situation was managed, the patient was shifted to the appropriate combination of antihypertensives. In particular, he was managed with amlodipine, a calcium channel blocker, metoprolol, a beta-blocker, enalapril, an ACE-inhibitor and minoxidil, a vasodilator [9]. The use of an ACE inhibitor was acceptable in his case, because he had unilateral disease. With bilateral renal disease, ACE inhibitors are contraindicated as they cause dilation of efferent arterioles with a reduction in glomerular filtration rate and acute renal failure [11]. In fact, perhaps the most important medication was the ACE inhibitor as it counteracted the proposed pathomechanism of the renovascular hypertension, although clinically, the minoxidil's effect was the most significant. We previously reported the efficacy of minoxidil in renovascular hypertension, but this drug also leads to considerable hypertrichosis [14], as was the case in our patient. A surgical approach would be preferred, even though the ability to come off anti hypertensive drugs completely cannot be guaranteed. The parents do not perceive the hypertrichosis as a problem, nor do they struggle with giving four different medications at multiple times of the day, whereas they have concerns about complications of an angiography.

CONCLUSIONS

Bell's palsy has many underlying etiologies. Hypertension is one of the more rare causes, with only a few cases reported in the literature in relation to children. However, any child presenting with this condition requires a blood pressure measurement, as hypertension may be the underlying cause and lead to a very different workup and management plan.

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