Peripheral facial paralysis as initial manifestation of hypertension in a child

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Hypertension is one of the rare causes of peripheral facial paralysis in children. The unawareness of this association at presentation may cause serious medical errors and result in delays in the diagnosis of hypertension, which may worsen with corticosteroid therapy given for Bell's palsy. We describe a severely hypertensive child who was first seen with peripheral facial paralysis and given corticosteroid therapy in another hospital. She presented to our clinic during the second facial paralysis attack with hypertensive pontine hemorrhage.

Key words: peripheral facial paralysis, hypertension, pontine hemorrhage.

Facial paralysis can be a rare initial feature of severe hypertension in children[1]. Even so, practitioners, pediatricians, neurologists and otorhinolaryngologists may be unaware of this association which may result in delay of the diagnosis of hypertension. Our case is an example of this problem which resulted in delayed treatment of hypertension and pontine hemorrhage.

Case Report

A nine-year-old female patient was first seen in another hospital with right peripheral facial paralysis. The diagnosis of idiopathic peripheral facial paralysis (Bell's palsy) was established and short-term methylprednisolone therapy was applied. Facial paralysis resolved completely in several weeks but headache did not. Four months later, she was admitted to our clinic with severe headache, vomiting, right peripheral facial paralysis and convulsion. Her headache became diffuse and continuous. Her blood pressure was 200/150 mmHg in the emergency department. On neurologic examination she was drowsy. Right peripheral facial palsy, pinpoint pupils, and gaze palsy together with internuclear ophthalmoplegia (one-and-a-half syndrome) were detected. Deep tendon reflexes were brisk on both sides and Babinski's sign was positive bilaterally. Fundoscopy showed bilateral severe hypertensive retinopathy and papilledema. Cranial magnetic resonance imaging (MRI) revealed basotegmental pontine hemorrhage (Fig. 1).

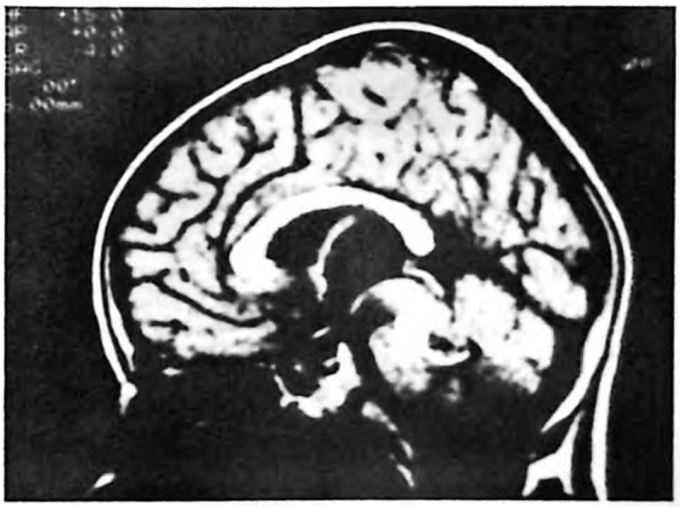

Fig. 1. T2-weighted magnetic resonance imaging (MRI) of the patient showed pontine hemorrhage.

Urine analysis showed mild proteinuria. On urine microscopy 4-5 leukocytes were seen and the urine culture grew E. coli. Creatinine clearance was within normal limits (80 ml/min). Other hematological and biochemical tests including urea nitrogen and creatinine, glucose, electrolytes, transaminases, creatine kinase, alkaline phosphatase, complete blood count, erythrocyte sedimentation rate, C3, C4, ANA,

and anti-DNA were normal. Blood renin, angiotensin and aldosterone levels were elevated. The renal ultrasonography revealed a mild degree dilatation of the left renal calices. Intravenous pyelography, renal angiography, renal scintigraphy with 99Tc-DTPA and 99Tc-DMSA, and voiding cystourethrography were performed. Bilaterally decreased renal vascularization, pyelonephritic scars, and cortical deformation (more prominent on the left than right), slow output function of the left kidney, and bilateral grade-I reflux were detected. Echocardiography showed left ventricular hypertrophy and mild aortic insufficiency. Cerebral angiogram revealed no pathology.

After admisison, she was treated with captopril, prazosin, atenolol and nitroprusside. Satisfactory and rapid control of her blood pressure was obtained. Her neurologic deficits resolved gradually. On the 18th day of admission, facial palsy recovered completely. She was discharged with 4 mg/kg captopril therapy after 33 days of hospitalization.

Discussion

Secondary hypertension is more common than essential hypertension in infants and children. Approximately 75-80% of children with secondary hypertension have a renal abnormality. Chronic pyelonephritis or reflux nephropathy with pyelonephritic scars, glomerulonephritis, renal obstructive disease and polycystic kidney disease are among the most common etiologies[2]. In our patient, pyelonephritic scars and activated renin-angiotensin system were present.

There is no specific symptom of hypertension. Headache is the most common complaint, and nausea and vomiting are initial complaints in many patients. Seizures occur more frequently in children[2,3]. Facial paralysis is a rare finding of hypertension. The most common causes of facial paralysis in children are otitis media and idiopathic Bell's palsy[4]. In addition, it can be secondary to trauma; skull diseases such as osteomyelitis and osteopetrosis; toxins; metabolic causes (hyperparathyroidism, hypothyroidism); neck lesions; infections (especially otitis media); intracranial space-occupying lesions; genetic, autoimmune, and muscular disorders and, rarely, hypertension[5,6]. The paralysis can be intermittent and independent of blood pressure control[1]. In 10 patients described in the literature, the facial paralysis was intermittent in six, as it was in our case[1,7,8]. Recurrent hemorrhage within the facial canal could account for the intermittent nature[1].

The cause of facial paralysis in the hypertensive child is unclear[6], but hemorrhage or edema in the facial canal may be important factors. As in our case, the prognosis in children is good[1,3,6,9]. The duration of palsy varies from days to weeks. Recovery begins when the pressure is reduced[6]. Our patient's paralysis resolved within 18 days following antihypertensive therapy.

The therapeutic effect of corticosteroids in acute idiopathic peripheral nerve paralysis is controversial. Some authors support early steroid treatment[10,11], others suggest that steroid therapy initiated at an early stage of childhood Bell's palsy does not significantly improve the outcome[12]. In contrast to cases of Bell's palsy, in hypertensive cases the use of glucocorticoids seems to be steroids[1]. It appears that unawareness of the fact that facial palsy can be the presenting sign of severe hypertension is aggravated by the use of steroids. In our case, failure to diagnose hypertension resulted in pontine hemorrhage.

In children with facial paralysis, blood pressure determination should be conducted repeatedly. This is particularly important in patients prescribed steroids. In patients who are normotensive on first evaluation, a careful history, and clinical and basic laboratory examination for hypertension should be performed. After the diagnosis of Bell's palsy was established, the follow-up of blood pressure should not have been ignored.

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