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Cortical Dysplasia and Obstetrical Brachial Plexus Palsy

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We report 2 patients with obstetrical brachial plexus palsy, ipsilateral leg weakness, and contralateral motor cortical dysplasia. To our knowledge, this is the first description of such an association. In both cases, the diagnosis of obstetrical brachial plexus palsy was established clinically shortly after birth and later confirmed neurophysiologically. Motor cortex dysplasia was diagnosed by magnetic resonance imaging (MRI). The association of obstetrical brachial plexus palsy and contralateral motor cortex dysplasia, a condition known to produce congenital hemiparesis, raises the possibility that the cortical dysplasia was a predisposing factor for obstetrical brachial plexus palsy in these cases.

Keywords: congenital hemiparesis; cortical dysplasia; obstetrical brachial plexus palsy

Motor cortex dysplasia is a frequent cause of congenital hemiparesis.[1–7] We report 2 patients with obstetrical brachial plexus palsy and contralateral motor cortex dysplasia. The purpose of this report is to describe this new association and discuss its possible implication.

Case 1

An 18-month-old girl presented for evaluation of left obstetrical brachial plexus palsy. She was the product of an uncomplicated pregnancy. Delivery was complicated by shoulder dystocia. Vacuum extraction was used. Gestational age was 42 weeks. Apgar scores were 7/9 at 1 and 5 minutes. Birth weight was 4.3 kg. Head circumference was 34 cm. At birth, she was diagnosed with complete left obstetrical brachial plexus palsy. No evidence of asymmetrical leg movements were noted at birth. Occupational therapy was instituted shortly after discharge. Some improvements were noted during the following months.

At 3 months of age the patient's attempts to grasp an object resulted in left shoulder adduction, arm internal rotation, and forearm extension and pronation. Wrist and finger movements were normal. Right arm movements were normal. The left leg moved less and was shorter than the right. Physical therapy was started at 5 months of age.

At 6 months of age she had a febrile seizure. A plain computed tomography of the brain revealed a smaller right hemisphere, raising the possibility of right hemisphere dysplasia. An electroencephalogram revealed frequent vertex spikes. Magnetic resonance imaging (MRI) of the brain at 11 months of age revealed right motor cortex dysplasia.

At the age of 18 months, her general examination was normal except for size asymmetry of the legs (Figure 1). Neurological examination was normal except for (1) limited left upper extremity active movements manifested by minimal arm abduction and elbow flexion on attempts to reach for an object placed at the midline; (2) decreased left biceps and brachioradialis deep tendon reflexes; (3) increased left patellar deep tendon reflex; and (4) bilaterally increased Achilles tendon reflexes. MRI of the brachial plexus and spinal cord were normal. Nerve conduction studies revealed decreased amplitude of the compound muscle action potentials of the left supraspinatus, deltoid, and triceps when compared with the compound muscle action potentials of the corresponding muscles on the right side. Electromyographic findings revealed long polyphasic motor unit potentials from the left deltoid, biceps, extensor carpi radialis, flexor carpi ulnaris, and first dorsal interosseous. Fibrillations were present in the

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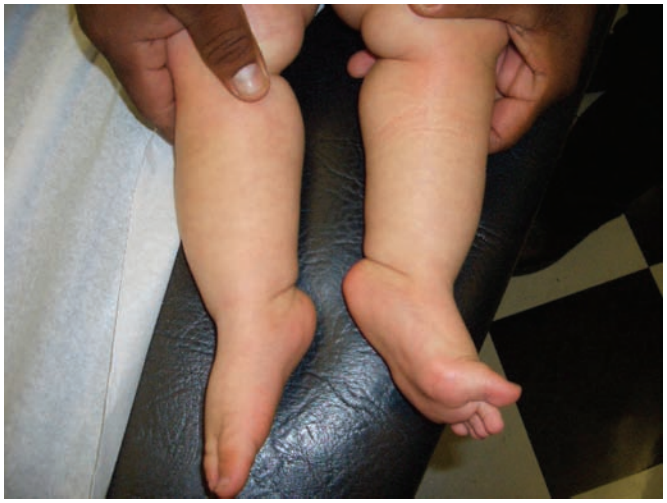

Figure 1. The distance between the knee and ankle cutaneous creases is shorter in the left leg than in the right. Left spontaneous Babinski.

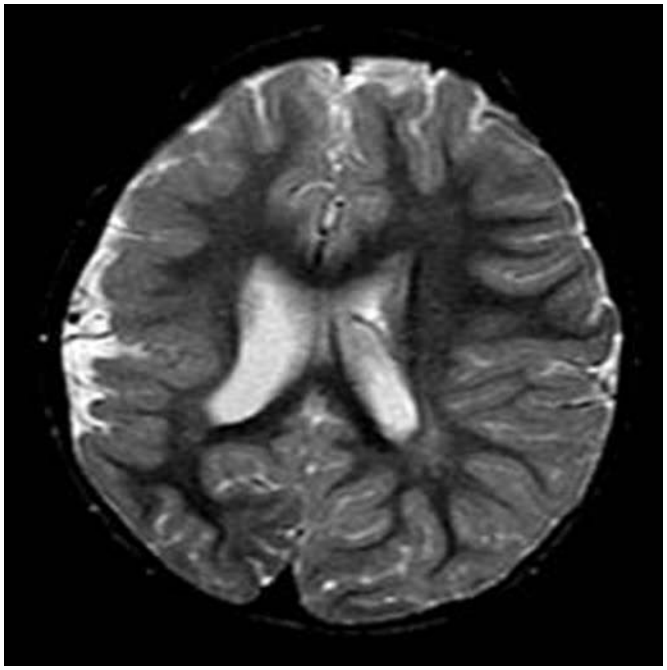

Figure 2. Magnetic resonance imaging (MRI) of the brain demonstrating diffuse cortical dysplasia of the right cerebral hemisphere manifested by thick frontoparietal cortex, lack of the normal gray–white matter interdigitations, volume loss of the underlying white matter, and increased size of the overlying subarachnoid space and lateral ventricles.

latter 2 muscles. Repeat MRI of the brain at 24 months of age confirmed the presence of right motor cortex dysplasia (Figure 2) and a smaller right cerebral peduncle.

Case 2

A 12-month-old boy with right brachial plexus palsy presented for evaluation of ipsilateral leg weakening. He was the product of an uncomplicated pregnancy. Delivery was by cesarean section due to meconium-stained amniotic fluid. Gestational age was 40 weeks. Apgar scores were 8/9 at 1 and 5 minutes. Birth weight was 4.2 kg. Head circumference was 35 cm. Decreased right arm movements were noted at birth. Right arm abduction and elbow flexion were limited and attempts to move the arm led to internal rotation of the arm, wrist flexion, and finger flexion. The right arm weakness improved during the following weeks without therapy but a left arm preference was still present at 3 months of age. Occupational therapy evaluation at approximately 4 months of age revealed right arm abnormalities manifested by shoulder girdle atrophy, limited arm abduction, limited flexion of the elbow, and a weak right hand with moderate flexion contraction of the fingers.

At 8 months of age, the patient was referred to a neurologist for the first time. Head circumference was 43.5 cm. Right shoulder atrophy was present. The neurological examination was normal except for the evaluation of the right arm. On attempts to reach objects with the right arm, the hand remained fisted, elbow flexion was limited to 100 degrees, and abduction was limited to 90 degrees. Right biceps tendon reflex was decreased. Radiographs revealed a hypoplastic right shoulder. MRI of the brachial plexus was normal. MRI of the brain revealed diffuse cortical dysplasia of the left hemisphere (Figure 3).

At 12 months of age, the mother noted right leg weakness when the infant attempted to walk. A second neurological opinion was obtained. General examination was normal. Neurological examination revealed arm weakness with characteristics similar to those previously described at 8 months. In addition, a tight right healcord and right leg weakness, manifested by decreased frequency of movement and decreased strength to confrontational testing, were present. Nerve conduction studies revealed decreased amplitude of the compound muscle action potentials in the right supraspinatus and deltoid. Electromyogram of the right supraspinatus, deltoid, biceps, extensor carpi radialis, and first dorsal interosseus were normal.

Discussion

The diagnosis of obstetrical brachial plexus palsy in these cases was based on clinical and electrophysiological findings. The clinical findings supporting the diagnosis of obstetrical brachial plexus palsy were the spatial distribution and temporal evolution of arm weakness, and the exclusion of nonobstetrical causes of brachial plexus palsy. In the first case, there was complete paralysis at birth, typical Erb's palsy posture at

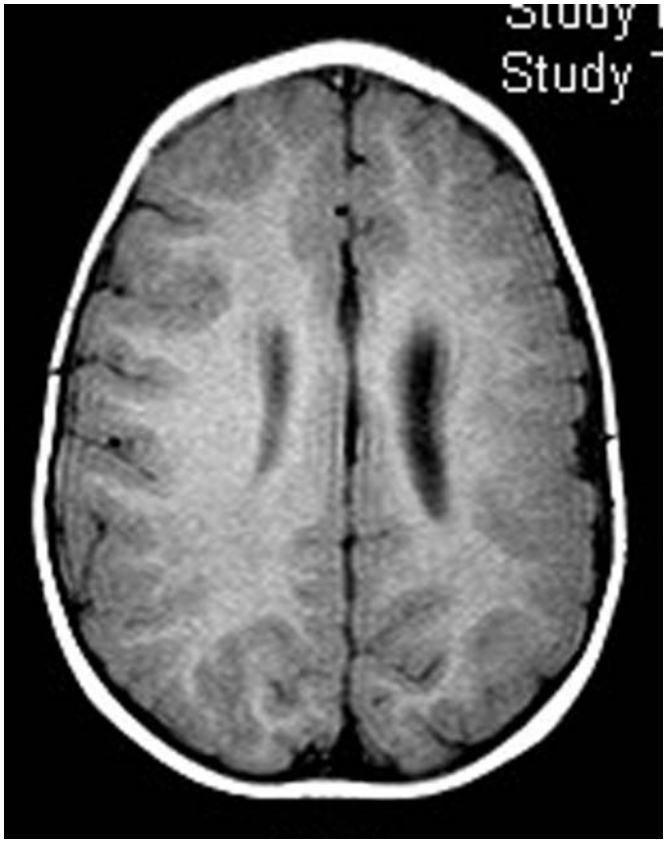

Figure 3. Magnetic resonance imaging (MRI) of the brain demonstrating diffuse cortical dysplasia of the left cerebral hemispheres.

3 months of age, weakness of the muscles innervated by the upper trunk of the brachial plexus, and decreased biceps and brachioradialis deep tendon reflexes at 18 months of age. A history of shoulder dystocia, a predisposing condition for obstetrical brachial plexus palsy, was also present in the first case. In the second case, there was proximal arm weakness at birth, typical Erb's palsy posture and right shoulder atrophy at 4 months of age, and decreased right deltoid and biceps strength at 12 months of age. The electrophysiologic finding supporting the diagnosis of brachial plexus palsy in both cases was the presence of chronic denervation in several of its terminal nerves.[8] In the first case, chronic denervation was manifested by decreased amplitude of the compound muscle action potentials, fibrillations, and long polyphasic motor unit potentials. In the second case, chronic denervation was manifested by decreased amplitude of the compound muscle action potentials.

The diagnosis of contralateral cortical dysplasia involving the motor area was suspected based on leg weakness ipsilateral to the affected arm in both cases and a smaller leg ipsilateral to the weak arm in the first case (Figure 1). The diagnosis of cortical dysplasia was confirmed by MRI in both cases (Figures 2 and 3).

To our knowledge, these are the first reported cases to describe the association of cerebral cortical dysplasia and contralateral obstetrical brachial plexus palsy. We believe that this association helps explain the pathophysiology of obstetrical brachial plexus palsy in these patients. Ultimately, obstetrical brachial plexus palsy occurs when the magnitude and acceleration of the force stretching the brachial plexus overwhelm the resistance of the plexus at its most vulnerable point. The resistance of the brachial plexus depends on (1) the intrinsic resistance of its nerve fibers, and (2) the resistance of the shoulder girdle bones, muscles, and cartilage.[9–12] Two mechanisms may have increased the vulnerability of the brachial plexus to stretch injury during delivery in these patients: shoulder girdle muscle weakness and an abnormal arm position.[13] The former leads to decreased resistance of the shoulder girdle muscles, and the latter leads to a change in the vector of the stretching force. Two mechanisms may have increased the vulnerability of the brachial plexus to stretch injury during delivery in these patients: weakness of the shoulder girdle muscles and an abnormal arm position.[sup]13 The former leads to decreased resistance of the shoulder girdle muscles, and the latter leads to a change in the vector of the stretching force. These factors can be expressed by the following formula:

p of OBPI: (m)(a)(cos of ∠) @bp / r(n+mu+j+b)

The probability (p) of an obstretical brachial plexus injury (OBPI) is directly proportional to the magnitude (m) and the acceleration (a) of the stretching force, and the parallelism (cos of) between the vector of the stretching force and the axis of the most vulnerable brachial plexus segment (@ bp), and inversely proportional to the resistance (r) of the brachial plexus nerve bundles (n) and shoulder girdle muscles (mu), joints (j) and bones (b).

We believe the association of motor cortex dysplasia and contralateral obstetrical brachial plexus palsy in these 2 cases suggests that fetal brain pathology can be a predisposing factor in some cases of obstetrical brachial plexus palsy.

References

1. Wu YW, Lindan CE, Henning LH, et al. Neuroimaging abnormalities in infants with congenital hemiparesis. *Pediatr Neurol*. 2006;35:191-196.
2. Chang BS, Apse KA, Caraballo R, et al. A familial syndrome of unilateral polymicrogyria affecting the right hemisphere. *Neurology*. 2006;10:133-135.

3. Caraballo RH, Cersosimo RO, Fejerman N. Unilateral closed-lip schizencephaly and epilepsy: a comparison with cases of unilateral polymicrogyria. *Brain Dev.* 2004;26:151-157.
4. Pascual-Castroviejo I, Pascual-Pascual SI, Viano J. Unilateral polymicrogyria: a common cause of hemiplegia of prenatal origin. *Brain Dev.* 2001;23:216-222.
5. Caraballo RH, Cersosimo RO, Mazza E, Fejerman N. Focal polymicrogyria in mother and son. *Brain Dev.* 2000;22:336-339.
6. Caraballo RH, Cersosimo RO, Fejerman N. A particular type of epilepsy in patients with congenital hemiparesis associated with polymicrogyria or unilateral pachygyria. *Rev Neurol.* 1997;25: 1058-1063.
7. Caraballo R, Cersosimo R, Fejerman N. A particular type of epilepsy in children with congenital hemiparesis associated with unilateral polymicrogyria. *Epilepsia.* 1999;40:865-871.
8. Pitt M, Vredeveld JW. The role of electromyography in the management of the brachial plexus palsy of the newborn. *Clin Neurophysiol.* 2005;116:1756-1761.
9. Symington J. The physics of nerve-stretching. *BMJ.* 1882;1: 770-771.
10. Sunderland S, Bradley KC. Stress-strain phenomena in human peripheral nerve trunks. *Brain.* 1961;84:102-119.
11. Sunderland S, Bradley KC. Stress-strain phenomena in human spinal nerve roots. *Brain.* 1961;84:120-124.
12. Sutherland S. Mechanism of cervical nerve root avulsion in injuries of the neck and shoulder. *J Neurosurg.* 1974;41: 705-714.
13. Alfonso I, Diaz-Arca G, Alfonso DT, et al. Fetal deformations: a risk factor for obstetrical brachial plexus palsy? *Pediatr Neurol.* 2006;35:246-249.