

A case of neonatal peroneal neuropathy with intrauterine onset

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Peroneal neuropathy presenting at birth is a rare disorder. Although neonatal mononeuropathies may be related to obstetrical complications, prenatal mechanisms should be also considered. We describe an infant who was born at term by cesarean section due to breech presentation with a unilateral footdrop. Lack of compound muscle action potential in the peroneal nerve and denervation potentials confined to the tibialis anterior and the extensor hallucis longus muscles in the electrophysiological studies on the fourth day of life strongly suggest an isolated peroneal neuropathy of intrauterine onset. Early and sequential electrodiagnostic studies will be important to provide better temporal and pathophysiologic definitions, the better timing of onset and prognosis for mononeuropathies presenting in newborn infants. (*Korean J Pediatr* 2007;50:585-587)

Key Words : Peroneal neuropathy, Electromyography, Newborn

Introduction

Mononeuropathies in pediatric age occur infrequently. Peroneal neuropathy is one of the most frequently encountered mononeuropathy in children. However, peroneal neuropathy presenting at birth is an extremely rare disorder. A total of seven cases of neonatal peroneal mononeuropathies clarifying its etiology have been reported in the English literature¹⁻⁴⁾. Three cases out of seven neonatal peroneal neuropathies were associated with breech presentation^{1,4)}. We describe the clinical and electrophysiological findings of unilateral peroneal neuropathy of possibly intrauterine onset in a newborn complicated with breech presentation.

Case Report

A 3,760 g male infant was born at term to a 35-year-old gravida 2 and para 1 mother. He was delivered by cesarean section because of breech presentation. Otherwise fetal movement was normal, and there was neither obstet-

ric complication nor obvious birth trauma. APGAR scores were 7 and 9 at 1 and 5 minutes, respectively. At birth, he was noticed to have a left footdrop (Fig. 1). Neurological examination demonstrated definite limitation of dorsiflexion and eversion of the left foot and toe in response to painful stimuli. The general sensory examination seemed to be intact to pin prick stimulation. Deep tendon reflexes and neonatal primitive reflexes including Moro reflex were normal. No abnormal findings were noted in the simple X-rays of both feet, knees, and hips.

Nerve conduction studies (NCS) and needle electromyography (EMG) were performed on the fourth day of life. No compound muscle action potential was evoked in the extensor digitorum brevis after stimulation of the left peroneal nerve at the ankle and fibular head. A normal response was present when the right peroneal (20.6 m/s) and both tibial nerves (21.6 m/s, 21.9 m/s) were stimulated. Needle EMG showed fibrillation potentials and positive sharp waves in the left tibialis anterior and extensor hallucis longus with a decreased number of motor units potentials (Fig. 2). Evaluation of the left peroneus longus, gastrocnemius, hamstring, and vastus lateralis muscles revealed normal findings. A repeated NCS and needle EMG performed 10 days later did not show any remarkable changes. Compound muscle action potential of the left peroneal nerve was not evoked in the last follow-up NCS

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at 1 month after birth.

He was managed conservatively with a short leg splint followed by passive exercise. He had a partial improvement of left foot dorsiflexion at 1 month visit, and subsequently showed complete clinical recovery at 2 months of age.

Discussion

The sciatic nerve divides into its two component divisions immediately proximal to the popliteal fossa. The smaller common peroneal nerve leaves the popliteal fossa laterally and winds around the neck of the fibula before dividing into superficial and deep peroneal branches. The peroneal nerve is usually damaged at the fibular head. It is just underneath the skin as it winds around the head of the fibula. Its superficial and relatively fixed position at the fibular head renders the nerve susceptible to compression⁵. The nerve leaves the fibular head region to enter the fibular tunnel. The peroneal nerve innervates the muscles that are responsible for dorsiflexion and eversion of the foot.

Multiple etiologic mechanisms have been invoked for neonatal peroneal neuropathies, including complications of delivery, intrauterine pressure from amniotic bands, umbilical cord, sacral promontory, and symphysis pubis, prolonged labor with a rigid cervix and uterine contraction

ring, and clear postnatal cause, such as footboard compression, compartment syndromes due to intravenous fluid infiltration as well^{1,3,6,7}. These patients were usually born at term to primiparous mothers after a complicated labor and delivery or prolonged delivery leading to cesarean section. Previous three newborns with neonatal peroneal neuropathy associated with breech presentation were born by spontaneous or forceps assisted vaginal delivery. However, our newborn was the second baby and delivered by elective cesarean section. In addition, there were no remarkable uterine abnormalities during the section. Although there was neither a uterine contraction ring nor any skin lesion suggestive of an intrauterine onset, the early appearance of fibrillation potentials at 4th day of life suggests that the infant have sustained the peroneal lesion in utero before delivery⁸. Considering the electrophysiological findings, the lesion of our case could be localized to a site between the popliteal fossa and the innervation point of the tibialis anterior muscle. Although the electrophysiological findings do not define the pathogenesis of neuropathy, prolonged extension during the breech presentation in utero of the lower limbs could induce traction of the peroneal nerve. The traction exerts a selective effect on the peroneal nerve, which can be explained by the relative fixation of the nerve at the neck of fibula. The consequent local ischemia accounts for the paralysis distal to the neck of the fibula.

Since the distance between the site of nerve lesion and the muscles is shorter in neonate compared with the adults, the traditional 10 to 14 days which is necessary for positive sharp waves and fibrillation potentials to be present after nerve lesions would be less in newborn³. However, denervation potentials and lack of a compound muscle action potential of peroneal nerve at 4th day of life in our infant could suggest that axonal injury possibly occurred in utero. Our patient made a good recovery in spite of electrophysiological findings suggestive of total axonal loss. It is likely that the short distance between the nerve lesion site and the denervated muscles would make a quick reinnervation possible. Also, some other unknown attributes of



Fig. 1. Immediately after birth a severe degree of left footdrop associated with slight inversion of the foot was noted.

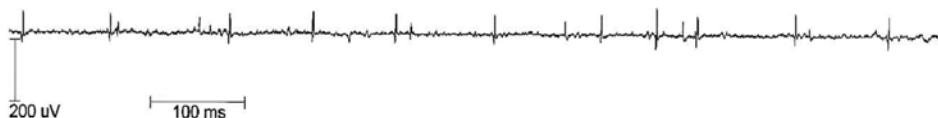


Fig. 2. Needle electromyography on the 4th day after birth shows numerous fibrillation potentials in the left extensor hallucis longus.

the newborn neuromuscular system could promote the rapid reinnervation process⁴⁾. As a matter of fact, many neonatal peroneal neuropathies are often self-limited. This fact may account for the reported rarity of this disorder.

Encountered the neonatal peroneal neuropathy, early electrodiagnostic approach may provide better temporal definition of the lesion and sequential electrophysiological studies may be helpful for appreciating the prognosis.

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출생시에 나타나는 신생아 종아리신경병증은 아직 국내에는 보고된 적이 없는 매우 드문 질환으로, 대부분이 자연 치유되는 양성 경과를 취한다. 신생아의 홀신경병증은 대개 산과적 합병증이지만, 출생 전 원인도 고려하여야 한다. 저자들은 불기태위로 인해 제왕절개술로 만기 태어난 신생아에서 발견된 발처짐을 보고한다. 생후 4일째 시행한 전기생리학적 검사에서 종아리신경의 복합근육활동전위 소실과 앞정간근과 긴엄지평근의 탈신경전위가 관찰된 것으로 미루어 자궁 내에서 발병한 종아리신경병증으로 추정한다. 조기에 전기생리학적검사를 시행하고 추적검사를

하면 발병시기와 병리적 기전 및 예후를 판단하는데 도움이 된다.

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