OLGU SUNUMU CASE REPORT

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Drop Foot Resulting from Hereditary Multiple Exostoses

Herediter Multipl Ekzositoza Bağlı Gelişen Düşük Ayak

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ABSTRACT In this case report, the drop foot due to compression of osteochondroma in a 9-year-old male patient with hereditary multiple exostoses is described. On physical examination, bony enlargements were palpated around the left knee. There was a decrease in the left knee and ankle joint range of motion, ankle and finger dorsiflexion muscle strength. In electrodiagnostic examination, chronic denervation in the muscles innervated by the peroneal nerve was detected. In the radiological evaluation, osteochondromas were present in the fibular head and distal femur. The patient had previously undergone surgical decompression and resection around the head of the fibula twice. After the rehabilitation program sessions, there was an increase in walking speed, stride length, and an improvement in activities of daily living in the patient. Osteochondromas may rarely cause neuropathies as a result of chronic compression. Early decompression and early rehabilitation are required in a lesion such as an osteochondroma in neuropathies developing after compression.

Keywords: Hereditary multiple exostoses; drop foot; peroneal nerve palsy

ÖZET Bu olgu sunumunda, herediter multipl ekzostoz (HME) tanılı 9 yaşında erkek hastanın osteokondrom basısına bağlı düşük ayak gelişimi anlatılmaktadır. Fizik muayenede sol diz etrafında kemik genişlemeleri palpe edildi. Sol diz ve ayak bileği eklem hareket açıklığında, ayak bileği ve parmak dorsifleksiyonu kas gücünde azalma saptandı. Elektrodiagnostik incelemede, peroneal sinir innerve kaslarda kronik denervasyon bulundu. Radyolojik değerlendirmede fibula başı ve femur distalinde osteokondromalar mevcuttu. Hastaya daha önce 2 kez fibula başı etrafına cerrahi dekompresyon ve rezeksiyon uygulanmıştı. Rehabilitasyon programı sonrasında hastada yürüme hızı, adım uzunluğunda artış ve günlük yaşam aktivitelerinde iyileşme saptandı. Osteokondromalar kronik bası sonucunda nadirde olsa nöropatilere neden olabilmektedir. Bası sonrası gelişen nöropatilerde osteokondrom gibi bir lezyon varlığında erken dekompresyon yapılması ve erken rehabilitasyon gereklidir.

Anahtar Kelimeler: Herediter multipl ekzostoz; düşük ayak; peroneal sinir hasarı

Hereditary multiple exostose (HME) is a benign syndrome with autosomal dominant inheritance, consisting of cartilage-capped osteochondromas around the joints.[1] This case report aims to describe the presence of a drop foot due to compression of osteochondromas in a patient with HME by considering the rehabilitation approach.

CASE REPORT

A nine-year-old male patient was admitted to our clinic with the complaint of weakness in his left foot. In his history, he had been diagnosed with HME, his complaint had been for two years and he had undergone surgical decompression and resection of the head of the fibula twice for drop foot. But after the surgery, the muscle weakness of the left foot persisted. The patient's father and sister had also been diagnosed with HME. Physical examination disclosed an incision scar around the left knee and bone enlargements. The left knee joint range of motion (ROM) measured with goniometer; flexion was 80°, extension -5°, left ankle dorsiflexion 5°, plantar flex-

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ion 10°. The proximal lower extremity muscles and ankle plantar flexors muscle strength was Grade 5, but ankle dorsiflexion muscle strength was Grade 2, and dorsiflexion of fingers was Grade 1. He had non-dermatomal hypoesthesia in the ankle area. Deep tendon reflex was normoactive and pathological reflexes were negative. In the other extremities, ROM was complete and there was no neurological deficit. In gait analysis, a steppage walk was found. The leg length on the left was 2 cm shorter than the right side. Routine laboratory tests were optimal. Radiographs revealed osteochondromas in the distal of both femurs, around the tibia, fibula, and calcaneus (Figure 1). In the magnetic resonance imaging (MRI), two osteochondromatosis of 46 mm in the meta-physio diaphyseal region of the left femur distal and fibular head were observed (Figure 2).

In electromyography (EMG), nerve conduction studies could not be obtained from the peroneal nerve innervated muscles and the sural nerve on the left (Table 1). In needle EMG, abnormal spontaneous potentials and chronic denervation findings were found in the tibialis anterior, peroneus longus, extensor hallucis longus, extensor digitorum brevis muscles, while EMG was normal in the gastrocnemius medial head and biceps femoris short head (Table 2). As the result of the EMG studies, axonal neuropathy of peroneal nerve was detected. The patient was included in the rehabilitation program to increase ROM and muscle strength. ROM, stretching, muscle strengthening exercises, gait training, and electrical stimulation to the deficit muscles were applied for 21 sessions. The patient was recommended a high-density plastic gait mold and 1 cm of shortness reinforcement. After rehabilitation, ROM of the left knee flexion was 90°, and left ankle dorsiflexion was 10°. There was no increase in muscle strength, but an increase in endurance was detected. An increase in walking speed and stride length were observed in observational gait analysis and an improvement in activities of daily living were detected with the Barthel index. Written consent was obtained from the patient to publish the data.

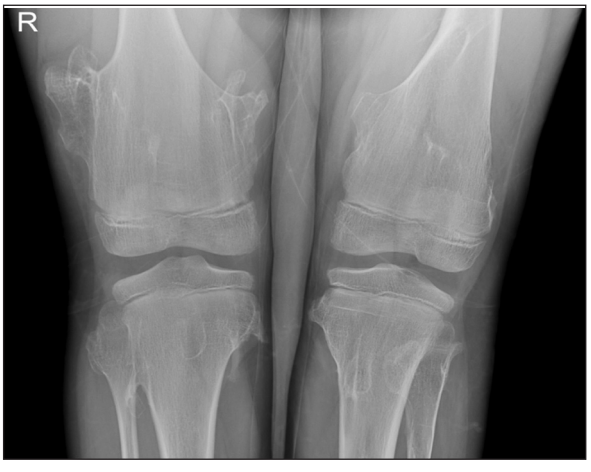

FIGURE 1: X-ray imaging of the exostoses.

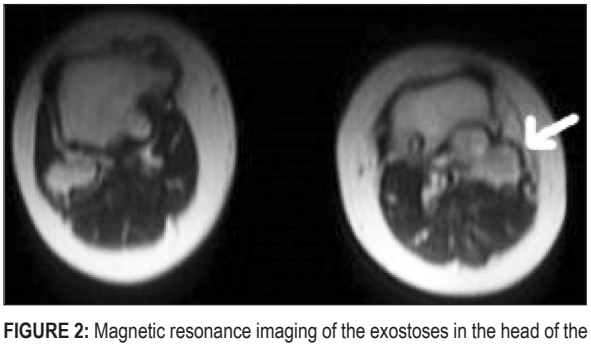

FIGURE 2: Magnetic resonance imaging of the exostoses in the head of the fibula is shown with white arrow.

DISCUSSION

HME is an autosomal dominant skeletal dysplasia consisting of multiple osteochondromas affecting the epiphyses of long bones.[2] It can be sporadic in about 40%.[3] Osteochondromas are benign tumors composed of cartilage-capped bone tissue. Malignant transformation may be at a rate of 1-25%.[4] Although the knee joint is involved in approximately 94% of HMEs, nerve compression due to osteochondromas has been detected very rarely.[3,4]

Peroneal neuropathies are the most common mononeuropathies in the lower extremity.[5] The peroneal nerve is one of the two main branches of the sciatic nerve and consists of the posterior divisions of the L4-S2 roots. After seperating the sciatic nerve, it proceeds from the posterior thigh to the popliteal fossa and then divides into two superficial and deep branches behind the head of the fibula. It also receives anterolateral leg sensation by giving branches to the sural nerve before it divides.[6] The head of the fibula is vulnerable to damage because it is located

TABLE 1: Nerve conduction study results.

Site	Latency	Amplitude	Area	Segment	Distance (mm)	Interval (ms)	Nerve conduction velocity (m/s)
Peroneal, right							
Ankle	2.14 ms	12.17 mV	18.27 mVms				
Head of fibula	6.84 ms	8.60 mV	11.43 mVms	Ankle-head of fibula	225	4.7	47.9
Knee	8.06 ms	8 mV	14.20 mVms	Head of fibula-knee	100	1.22	82
Peroneal, left							
Ankle	-	-	-				
Knee	-	-	-	Ankle-head of fibula	225	-	0
Head of fibula	-	-	-	Head of fibula-knee	100	-	0
Tibial, left							
Medial malleolus	3.92 ms	12.24 mV	12.41 mVms				
Poplitea	9.10 ms	19.29 mV	20 mVms	Medial malleolus-poplitea	300	5.18	57.9
Sural, right							
Lateral mallolus-cruris	2.44 ms	9.10 uV	5.591 uVms	Lateral mallolus-cruris	110	2.48	45.1
Sural, left							
Lateral mallolus-cruris	-	-	-	Lateral mallolus-cruris	-	-	0

TABLE 2: Needle electromyography results.

Muscle/Side	Insertional activity	Fibrillation/ Positive wave	Fasiculations	Motor unit potential	Polyphasy	Recruitman	Amplitude
Tibialis anteror, left	Normal	+1/+1	0	+2	+1	Full	High +2
Peroneus longus, left	Normal	+1/+1	0	0	-	No act	-
Extansor hallucis longus, left	Increased	+2/+2	0	0	-	No act	-
Extansor digitorum brevis, left	Increased	+2/+2	0	0	-	No act	-
Gastrocnemius medial head, left	Normal	0/0	0	+4	-	Full	Normal
Biceps femoris short head, left	Normal	0/0	0	+4	-	Full	Normal

behind the subcutaneous tissues and just under the skin.[7] Peroneal neuropathies are frequently observed after knee trauma, fibular head fractures, tight splint or bandage application, prolonged squatting, prolonged compression while asleep or in a coma, pneumatic compression, and iatrogenic trauma.[5] Osteochondromas may also cause peroneal neuropathies as a result of chronic compression.[4,8] As in our case, total peroneal nerve damage can be seen, and isolated deep peroneal nerve damage has also been reported in the literature.[8] Peroneal neuropathies should be considered in the diagnosis in the presence of drop foot, loss of sensation in the lower extremities, or pain.[9] With EMG examinations, peroneal neuropathy can be distinguished from conditions that cause muscle weakness in the other lower extremities.[5] The first-line treatment in peroneal neuropathy is decompression and relieving tension on the nerve.[10]

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