

Repair of Peroneal Paralysis by Muscle Transposition in Sheep ^[1]

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Summary

This study was performed for the treatment of peroneal paralysis, occurred as a result of iatrogenic injection and/or medicine, with tendon transposition in sheep. Thirty six sheep from a flock with peroneal paralysis occurred following intramuscular injection of a commercial medicine used as metabolic activator were hospitalized for 15 days and medical treatment was applied. After failure of the medical treatment it was decided to perform tendon transposition to 20 sheep with peroneal paralysis. Remaining 16 sheep were received medical therapy for 3 more weeks. Six of these 16 sheep were also subjected to surgical intervention because of unsatisfactory medical treatment results. Others were slaughtered because of clinical deterioration. Following spinal anaesthesia, m. vastus lateralis and m. ext. dig. longus were dissected from their insertio and origos, respectively. Freed ends were adjoined on the dorsal part of m. tensor fascia lata by bringing them side by side and stitched with Locking loop style. Related leg was bandaged for three weeks. Three weeks after the surgical intervention a mild limping with recovered walking style was observed. Two months after the surgery all the animals were walking uneventfully with the exception of two sheep developed an infection as a result of decubitis. Tissue samples taken from sheep sent to slaughterhouse were investigated histopathologically. Histopathological examination revealed residues of the medicine even six weeks after the commercial medicine application. Peroneal nerves and perineural tissues showed degenerative changes and inflammatory cell infiltration. In conclusion this study showed that tendon transposition technique which was previously used in dogs could also be satisfactorily used in sheep with peroneal paralysis.

Keywords: *Iatrogenic injection, Peroneal paralysis, Muscle transposition, Sheep*

Koyunlarda Peroneal Paralizinin Kas Transpozisyonu ile Sağaltımı

Özet

Bu çalışma, koyunlarda hatalı enjeksiyon ve/veya ilaç uygulamasına bağlı olarak gelişen peroneal paralizinin kas transpozisyonu ile sağaltımı amacıyla yapıldı. Metabolizma aktivatörü amacıyla kullanılan ticari bir preparatın kas içi yolla enjeksiyonu sonrasında peroneal paralizi şekillenen bir sürüdeki koyunlardan 36'sı hospitalize edilerek 15 gün süreyle medikal sağaltım uygulandı. Olumlu bir gelişme sağlanamaması üzerine koyunlardan 20'sinde kas transpozisyonu yapıldı. Diğerlerinde medikal sağaltıma 3 hafta daha devam edildi. Medikal sağaltımdan sonuç alınamayan 16 koyundan 6'sında da aynı işlem gerçekleştirildi. Diğerleri, klinik tablonun olumsuzlaşması üzerine kesime sevk edildi. Kas transpozisyonu için, spinal anesteziyi izleyerek m. vastus lateralis insertio, m. ext. dig. longus ise origo noktalarından diseke edildi. Serbestleştirilen uçlar m. tensor fascia lata'nın dorsalinde yan yana getirilerek. Locking loop dikiş tekniği ile birleştirildi. İlgili bacağa 3 hafta süreyle bandaj uygulandı. Postoperatif 3. haftanın sonunda koyunlarda hafif topallıkla birlikte düzgün basış saptanırken, 2 ay sonraki kontrollerde ise dekubitise bağlı enfeksiyon gelişen 2 olgu haricinde tüm olguların problemsiz yürüyebildikleri görüldü. Kesime sevk edilen koyunlardan alınan doku örnekleri histopatolojik açıdan değerlendirildi. Histopatolojik incelemede enjeksiyonun üzerinden 6 hafta geçmesine rağmen bölgede ilaç kalıntısına rastlanırken sinir dokusu ve çevre perinöyral oluşumlarda dejeneratif değişikliklere ve yangısal hücre infiltrasyonuna rastlandı. Sonuçta peroneal paralizinin sağaltımında daha önce köpeklerde denenilen kas transpozisyonu tekniğinin koyunlarda da başarılı sonuçlar verdiği ortaya konmuştur.

Anahtar sözcükler: *Hatalı enjeksiyon, Peroneal paralizi, Kas transpozisyonu, Koyun*



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INTRODUCTION

Peripheral nerve injuries and related paralysis can often occur in animals¹⁻⁴. In domestic animals, similar to human, bone fractures, firearm wounds, deep burns and iatrogenic intramuscular injections could cause paralysis by directly affecting peripheral nerves³⁻¹². Furthermore inflammation, oedema, hematoma, and tumoral formations occurring nearby the nerves could also affect nerves and called entrapment neuropathy^{3,7,9,11,13-16}. In addition it was reported that iatrogenic paralysis can also occur as a consequence of some toxic medicine injected close to nerves¹⁷⁻²¹.

Prognosis of peroneal paralysis, although it depends on various factors including the cause, severity of lesion and direct relation to nerve, is generally accepted as unfavourable^{1-4,7-9,11-16}. Medical treatment could be tried in cases where nerve is not directly injured^{3,7,12,14}. Arthrodesis is generally applied where medical treatment is not satisfactory. However it does not always give effective results. Amputation is treatment of choice where serious complications develop¹⁻³.

Treatment of peripheral nerve paralysis^{2,3,7,20} and some orthopaedic disorders such as ligament rupture²² and luxation^{23,24} by muscle or ligament transposition is recently the most widely used technique. Peroneal paralysis is satisfactorily treated by tendon transpositions in cases clinically occurred or induced experimentally in dogs^{2,3,7,20}.

To the best of our knowledge there is no such study performed in sheep with peroneal paralysis. In this study, results and treatment of peroneal paralysis occurred in sheep as a consequence of iatrogenic application and/or toxic effect of commercial medicine, by tendon transposition is presented.

MATERIAL and METHODS

Animals

One day after IM injection of a commercial medicine used as a metabolism activator paralysis occurred in 145 animals in a flock of 300 sheep. Because of the owner's refusal treatment could not be applied to the all animals. Nevertheless, the owner persuaded to treat 36 of diseased animals and they were hospitalised in Veterinary Surgery Clinics of Faculty of Veterinary Medicine, University of Kafkas, Kars, Turkey.

Clinical Examination

Anamnesis revealed that all the animals were subjected to the injection with same medicine (Cu gluconate, Na selenite, Mn gluconate, Zn gluconate) and in similar way (IM). Palpation revealed local changes including swelling, an increase on local temperature, touch sensitivity etc. It was diagnosed as peroneal paralysis following clinical observation and examination results including leg posture and walking style, responses of the animals to passive forcing (extensor pushing and flexor pulling reflexes), needle pricks or haemostat, and electrical stimulation tests (*Figure 1*).

Treatment

The treatment included a combination of B complex vitamins, corticosteroids, antibiotics and application of protective bandage to preventing of decubitis for 15 days. After having failure of this medical treatment protocol it was decided to do muscle transposition to 20 animals. Other were continued to receive medical treatment and protective bandage application three more weeks.

For the muscle transposition, the animals were anesthetized with bupivacaine (5 ml), (Marcaine 0.5%, Astra Zeneca, İstanbul, Türkiye) intratechal and lateral parapatellar skin was incised in a length of 10-12 cm. M. vastus lateralis and m. ext. dig. longus were dissected from their insertio and origos, respectively (*Figure 2*). Freed ends were adjoined on the dorsal part of m. tensor fascia lata by bringing them side by side and stitched with Locking loop style (*Figure 3*). Related legs were bandaged for three weeks.

Similar surgical intervention was also applied to 6 of 16 sheep with no successful medical treatment outcome. Tissue samples taken from animals sent to abattoir were investigated histopathologically.

Histopathology

For histopathological examinations, tissue specimens (nerve and surrounding tissues) were collected from affected area, fixed in 10% neutral buffered formalin and processed routinely, sectioned at 5 µm and stained with haematoxylin-eosin (HE).

RESULTS

Clinical Results

Anamnesis revealed that problems were started

following first day after application of previously mentioned medicine in (145/300) a sheep flock.

Clinical examination showed a hard mass in injection area on palpation. Some animals were walking on front faces of phalanxes (*Figure 1*). Most of animals did not willing to walk and exhibited tendency to lying down. Needle pictures done to related extremities and electrical stimulation tests were negative in a certain area starting from front side of tarsal joints to distal parts of phalanxes. Similarly, despite passive forcing the animals were not able to keep heel area in extension.

Intraoperatively, muscle segments freed for the transposition were easily brought face to face and following stitching required tension was readily obtained.

Three weeks after the surgical intervention and debandaging animals were allowed to free walking showed a mild limping with recovered walking style. Two months after the surgery all the animals were walking uneventfully with the exception of two cases developed an infection as a result of decubitis (*Figure 4*).

Histopathology

Histopathological examination of the samples revealed yellow coloured and crystallised remnants of the medicine free or in the adipose tissue (*Figure 5*). Nerve bundles and epineurium were separated because of oedema and peripheral tissue was infiltrated by mononuclear cells with presence of oedema. Lymphocytes and hemosiderin laden macrophages were evident around the nerve fascicles (*Figure 6*). In some cases pyogranulomatous inflammation were seen.

Muscle and fat necrosis were seen in some areas surrounded by lymphocytes, plasma cells, hemosiderin laden macrophages and some giant cells (*Figure 5*). In some cases nerve bundles revealed myelin and axonal degeneration characterised by lymphocyte infiltration, vacuolation, and necrosis accompanied by peripheral lymphomonocytic infiltration (*Figure 6*).

DISCUSSION

Muscle paralysis could occur as a result of direct effects of peripheral nerve laceration and contusion or affected indirectly because of tumoral formations in the vicinity of nerves, abscess, haemangioma and other inflammatory type masses which compress nerves and cause trap neuropathies^{10-15,17,18}. Peroneal

paralysis can occur after trauma of an area where n. peronealis passes from lateral part of articulation genu or it could also occur following either erroneous injections or application of toxic medicines to vicinity of nerves^{1-3,7,8,12-14,17,21}. In the present study, anamnesis revealed peroneal paralysis of 145 animals from a flock of 300 sheep following application of a medicine used as metabolism activator. In most cases palpation showed a hard and disseminated mass on the back of articulation genu.

The most significant clinical sign of peroneal paralysis is animal steps on the ground with the dorsal face of heel joint as a result of tarsal joint extension and disappearance of phalanx extension. In chronic cases excoriation is common finding^{2,3,8,9}. In human, particularly in children stepping on front face of foot also called fallen ankle syndrome is generally caused by peroneal paralysis which generally occurs in consequence of erroneous injection^{13,16,17}. Most cases that we examined in early periods exhibited clinically similar symptoms. In some cases animals showed typical stepping on posture, starting from phalanxes, with only dorsal face of foot.

Medical treatment is first choice in peripheral nerve paralysis. However, the success of treatment is generally rely on the degree of nerve damage^{3,7,12-15}. In order to select the treatment way clinical examination and EMG, EEG is required to determine neuro-muscular nerve sign transmission loss level. Ultrasonographical examination may also require for preoperative evaluation of nerve lesions. These examinations may not only help to determine nerve lesions but existence of scar tissue surrounding the lesion and neuromas^{3,5,7,10,13,15,17}. In our cases, the diagnosis was built on anamnesis, clinical examination, picture applications, and pulling and pushing reflexes. Histopathological examination showed a wide segment of soft tissue damage including nerve, muscle and adipose tissue with presence of perineural medical residues. These findings indicate the main cause of peroneal paralysis as both erroneous injection and toxic effect of medicine. Furthermore, histopathological findings showed why the medical treatment was unsuccessful. When nerve tissue is totally damaged axon and Schwann cells are degenerated. Fibrosis developed in damaged area is generally main cause for the failure of regeneration. Regeneration is rather slow even after appropriate surgical intervention³.

In recent years muscle or ligament transposition methods which gives satisfactory results in a short

period of time is rather common technique in both human and veterinary medicine^{2,3,7,20,22-24}. The success of muscle transposition is largely depends on existence of strong muscle groups which is anatomically located close to transposition area with good nerve innervations and strong enough to meet the bio-mechanical needs of paralysed muscle groups^{2,3,6,16}. In recent years clinical and experimental muscle transposition studies done on cats and dogs with fibular paralysis gave successful results^{1-5,7}. To the best of our knowledge there is no such study conducted on sheep.

It is widely known that some agents and the area selected for injection may cause nerve degeneration and paralysis^{2-4,7-10,18-21}. In the present report the problem was originated from nerve degeneration which is caused by toxic effect of active ingredient or carrier of the medicine which is probably erroneously injected very close to the nerves. Medical treatment attempt was not successful. This is probably originated from medicine which is affecting nerves by several ways including direct pressure effect of deposited mass that is not absorbed after a long period of application and intra and perineural degeneration caused by toxic effect of medicine.

In conclusion, present study showed that muscle transposition could be successfully used for the treatment of peroneal paralysis in sheep.

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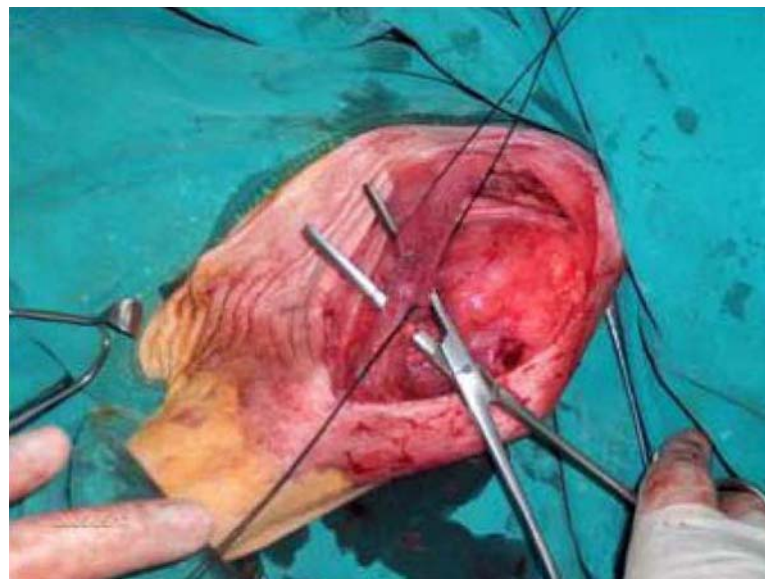


Şekil 1. Peroneal paralizli 3 nolu olgunun operasyon öncesi görünümü. Falanks ekleminin aşırı fleksiyonu

Fig 1. General appearance of sheep previous to surgery. Hiperflexion of phalanx joint (Case no 3)

Şekil 2. M. vastus lateralis kasının patellar inzersiyon bölgesinden serbestleştirilmesi

Fig 2. Dissection of M. vastus lateralis from patellar insertion area



Şekil 3. Transpozisyon amacıyla m. vastus lateralis ile m. ext. dig. longus arasında Locking loop dikiş tekniği ile sağlanan anastomoz

Fig 3. Anastomosis with use of Locking loop suture technique for the transposition in between m. vastus lateralis and m. ext. dig. longus

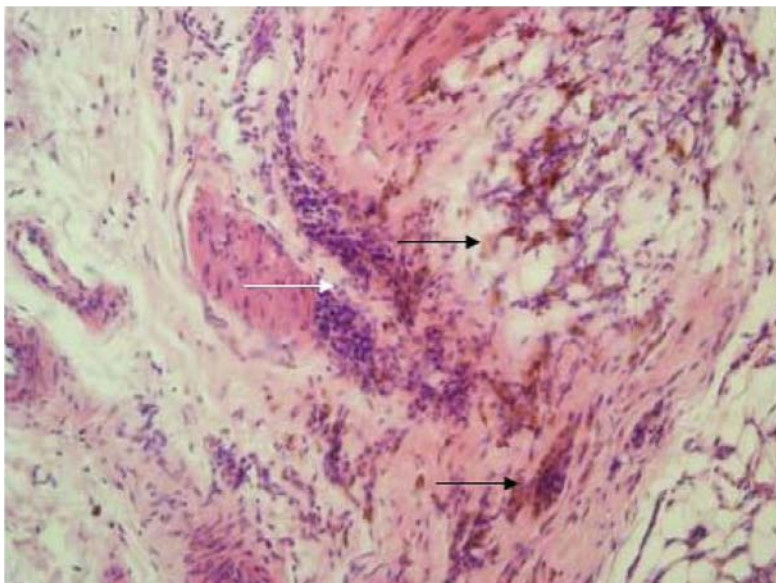
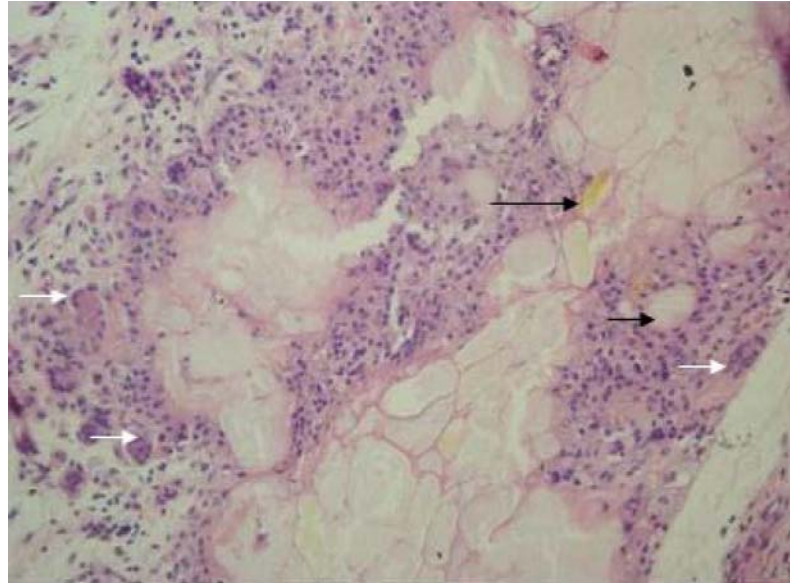


Şekil 4. Peroneal paralizli 3 nolu olgunun operasyonu izleyen 2. aydaki görünümü

Fig 4. General appearance of the sheep with peroneal paralysis two months after the surgery (Case no 3)

Şekil 5. Dev hücreleri ve makrofajlarla çevrelenen yağ nekrozu. Bazı yağ hücrelerinde sarı renkli, kristal halinde ilaç kalıntısı, (oklar), H&Ex20.

Fig 5. Fat necrosis surrounded by giant cells and macrophages. Some fat cells exhibit yellow colored medical residues, (arrows), H&Ex20.



Şekil 6. Sinir lifi fasikülleri çevresinde lenfosit ve bazıları hemosiderin pigmenti ile yüklü makrofaj infiltrasyonu ile karakterize kronik yangı, oklar. H&Ex20

Fig 6. Chronic inflammation characterised by infiltration of lymphocytes and hemosiderin laden macrophages surrounding nerve fascicles, arrows, H&Ex20