

Management of peroneal nerve paralysis in a three month old puppy using a tendon transplantation technique

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ABSTRACT: A severe right hind limb paralysis due to injury to the peroneal (fibular) nerve was characterized by knuckling of the paw, over extension of the hock, dragging of the limb and excoriations on the dorsum of the digits. Management of the case therapeutically and conservatively over a period of three weeks was not rewarding. The case was eventually and successfully managed by the relocation of the superficial digital flexor tendon (SDFT) to the long digital extensor tendon (LDET). The surgical procedure was relatively easy. However, the post-operative management and retraining of the puppy to enable it use the flexor tendon as an extensor was difficult and protracted but was successful. The puppy regained normal limb placement and near normal gait about two months postoperatively. It was eventually discharged after three and half months of management.

Key words: Neuropathy, management, peripheral, peroneal, prognosis, tendon, transplantation.

INTRODUCTION

Natural recovery from peripheral neuropathies is usually unlikely even after protracted conservative and/or therapeutic interventions (Hart and Tremblay, 1982; Bennett and Vaughan, 1976a, Leighton, 1982; Kilic et al., 2009). In most of such patients, the affected limb is either amputated or the patient eventually is euthanized except if, the condition is managed surgically in which case, the outcome may be favourable. (Bennett and Vaughan, 1976a; Leighton, 1982).

The sciatic nerve and/or its branches (the tibial and peroneal nerves) may be injured giving rise to varying degrees of peripheral neuropathies (neuropaxia, axonotemesis or neurotemesis) affecting different parts of the hind limb distal to the hock joint in animals and man. Such injuries often result from trauma, surgical procedures around the pelvis, hip joint, proximal and distal femur, and proximal fibular regions, during perineal herniorrhaphy or tumours in the vicinity of the nerve (Borrows and Harvey, 1973; Spruell, 1976; Omamegbe, 1976; Weaver and Omamegbe, 1981; Forterre, et al., 2007). Other common causes are dog fights, inappropriate hind limb dressings,

intramuscular injections deep in the vicinity of the nerve in the distal thigh muscles and compressions on the nerve at pressure points especially in unconscious, recumbent and emaciated animals (Bennett, 1976; Kilic et al., 2009).

The classical clinical signs of peroneal nerve paralysis include an inability to extend the stifle or flex the hock joints, loss of muscle mass in the tibial region and folding under of the paw (knuckling) which (paw) soon develops excoriations on its dorsum. The affected limb shows loss of cutaneous sensation on the cranial and lateral aspects of the leg below the hock joint and on the dorsum and dorso-lateral aspects of the digits, loss of the extensor reflex and occasional positive cross extension thrust reflex among others (Bennett and Vaughan, 1976a, b). The diagnosis of peroneal nerve paralysis is usually based on the case history and presenting clinical signs (Fortre et al., 2007). Nerve conduction studies are optional only if the rare possibility of re-innervation of the paralysed muscle/tendon is envisaged (Bennett, 1976).

Generally, peripheral neuropathies have rather very poor prognosis in humans and animals if treated

therapeutically and/or conservatively only because the response to such management is usually very poor. Most affected animals so managed usually have the affected limb(s) amputated or are eventually euthanized out of frustration of both the client and the attending veterinarian (Hart and Tremblay, 1982; Bennett and Vaughan, 1976a). In valuable patients like guide, guard, hunting, sport or indeed intimate pets, these options are often unacceptable to most clients. Therefore, some alternative treatment options with better prognoses are usually required.

It has been shown in humans (Wapner et al., 1993; Bradon and Harris, 2015) and animals (Bennett and Vaughan, 1976a, b; Hart and Tremblay, 1982; Leighton, 1982) that certain peripheral neuropathies associated with locomotor dysfunction can be relieved by the relocation of the tendon of some functional and innervated muscle/tendon to the muscle/tendon which has been denervated as a result of nerve damage. These techniques have been used mainly in the management of radial paralysis in the fore-limbs of animals (Sterner and Mollier, 1960; Hussain and Petit, 1967; Bacher and Potkay, 1976).

For this treatment approach to achieve the desired end, the function(s) of the muscle/tendon to be relocated must be duplicated by another muscle/tendon in its group so that the over-all function(s) of the muscle/tendon group is not adversely affected when one member of the group is relocated. Also, the muscle(s)/tendon(s) paralysed, the specific nerve traumatised and the appropriate tendon to be relocated must be clearly determined. This is because these techniques are best suited for the management of mono-neuropathies (Bennett and Vaughan, 1976a). For optimal post-operative results, these procedures must be executed carefully, aseptically and with minimal trauma to and desiccation of tissues at the surgical site (Bennett and Vaughan, 1976a; Leighton, 1982).

A surgical procedure for the relocation of the superficial digital, long digital or caudalis tibialis flexor tendons (SDFT, LDFT or CTT) to the LDET for the management of peroneal nerve paralysis in the hind limb of the dog was described by Bennett and Vaughan (1976a,b), Hart and Tremblay (1982) and others.

Although muscle/tendon relocation procedures have been credited with very good postoperative outcomes in the treatment of such cases, the retraining or rehabilitation of patients to enable them use the flexor tendon as an extensor is usually rather protracted (Malnati 1981; Wapner et al., 1993). Such retraining or rehabilitation programme must be carried out skilfully with attention to details if they are to serve as useful adjuncts to the primary surgical therapy (Bennett and Vaughan 1976a, b; Hart and Tremblay, 1982).

CASE REPORT

On the 8th of May, 2015, a three-month old, male

Caucasian puppy weighing 5.0 kilogram was referred to the Veterinary Teaching Hospital (VTH) of the University of Abuja (UA) with the primary history of having been mauled by a much bigger dog three weeks earlier. The attending veterinarian referred it to the VTH because its general health condition had gotten progressively worse while on antibiotic, multivitamin, analgesic and suturing and dressing of cutaneous wounds over a period of three weeks during which period, the puppy had become dehydrated, progressively weak, anorexic, constipated and unkempt. Neither the puppy nor the other dog had been vaccinated against rabies but there was no report of any change of attitude or dog fight among the dogs in the household-kennel after the reported incident.

At presentation, the puppy was weak, recumbent and anorexic. It groaned continuously apparently in pain and could not stand or walk even when encouraged to do so. Further history revealed that the puppy and three others of the same litter were kept in a compound with five adults of large breed of dogs and that they were all feed commercial canned dog food (bonju) and left-overs from the household kitchen. Generally, vaccination regimens for the dogs were haphazard and most times were not carried out. Each adult dog was housed individually in a kennel while puppies were left with their bitches until weaned. It was during one of the feeding times that the reported fight occurred.

On clinical examination, all the vital parameters measured were elevated—(rectal temperature-39.9°C, heart rate-140 beats per minute, respiratory rate-80 cycles per minute). The respiration was laboured with respiratory heaves audible at both inspiration and expiration and the heart sounds appeared more prominent on the right thoracic wall than the left but no murmurs or other abnormal cardiac or respiratory sounds were detected. There were several bite-puncture wounds on the head, thoracic and abdominal walls, and the caudo-lateral aspect of the right hind limb around the region of the stifle joint. Several of these showed signs of healing or carried old suture lines. Of particular note was the deep and penetrating bite wound on the lateral aspect of the lower thigh region of the right hind-limb. The puppy had a very heavy tick (*Repicephalussanguinius*) infestation. It was mildly to moderately dehydrated (about 5 to 7% body weight loss), with pale ocular and oral mucous membranes and a prolonged capillary refill time (>3min). Apart from piercing wounds on the nasal bones, no other osseous injury or locomotor dysfunction was observed at this stage.

A tentative diagnosis of trauma due to a dog-fight, deep piercing wounds, a diaphragmatic hernia, ecto-prasite associated with haemo-parasitisms, dehydration and severe anaemia was made. While admitted at the kennel of the VTH for further observation and treatment, it was noticed that the puppy was constipated and oliguric.

A survey radiographic examination of the thoracic and abdominal cavities/regions and bony structures revealed no obvious abnormality. No evidence of a diaphragmatic

hernia was seen. A venous (cephalic vein) blood sample was positive for Babesiacanis infection (++) and yielded haemogram values as follows: red blood cell (RBC) count- 4×10^6 cells/ml, white blood cell (WBC) count - 12×10^3 cells/ml, Packed cell volume (PCV) - 22.0% and haemoglobin concentration (Hb)- 7grams/ml. The differential white blood cell count showed a neutrophilia (82%) and a relative lymphopenia (14%) -a shift to the left; eosinophilia (3%) and a monocyte count of 1%.

The case was comprehensively managed therapeutically and conservatively and in particular against haemoparasites with two doses of imidocarp di-propionate @ 6.6 mg/kg body weight at two weeks interval, low doses of prednisolone injections @ 1.0 mg/kg body weight for five days, fluid replacement therapy with Ringers solution given intravenously @ 50.0 ml /kg body weight once daily for two consecutive days and a tick bath with dilute solutions of cypermethrin (twice at 10 days interval), vitamin supplementations, dolo-neurobion tablets, vitamin E injections intramuscularly for one week and anal suppository administration for three days. The puppy's condition improved markedly over the next nine days. Most of the bite wounds were healing actively and the puppy was able to stand and walk slightly. At this stage, it was noticed that the puppy was mildly ataxic, circled and knuckled slightly on the right hind paw. Its appetite, bladder and bowel movements had returned to normal. It was discharged after three weeks of hospitalisation but to be re-presented for re-assessment especially with respect to the locomotor dysfunction a week later.

On re-presentation for review, the puppy had gained some weight (8.9 kg) but knuckled on the right hind foot almost consistently (about 85 to 90% of limb placement). The stifle joint was over-flexed and the hock joint was hyper-extended and the puppy dragged the right hind limb trailing behind as it walked. Weepy excoriations were present on the dorsum of the right foot and the puppy was again heavily infested with ticks. There were no cutaneous sensations to mild – severe painful stimuli on the cranial aspect of the right foot specifically below the hock joint and the lateral digits. Sensations were present on the volar and dorso-medial aspects of the paw. The muscles of the affected limb below the stifle joint were severely atrophic (Figure 1).

On the basis of these findings, a diagnosis of peroneal nerve paralysis due to injury to the peroneal nerve below the point of separation from the tibial nerve at the distal thigh region was made. A radiographic examination of the limb showed a normal tarso-metatarsal joint.

The puppy was further de-ticked with a bath of dilute cypermethrin solution and administered two intramuscular injections of Diaminazene acetate (Berenil®) at 3.5 mg per kilogram body weight two weeks apart in the presumptive diagnosis of babesiosis as a result of the tick infestation and going by the previous history of the case. Penicillin and streptomycin powders were applied topically to the weepy excoriations on the dorsum of the paw and



Figure 1. Notice hyper-extension of the hock and stifle joints, flexion and knuckling of the right paw, trailing of the right leg behind and atrophy of the muscles particularly around the tibial region.

the limb was then placed on an Elmer sling (Figure 8 bandage- Egger and Wittick, 1990) in an attempt to encourage flexion of the hock and prevent knuckling of the paw. The dressings and Elmer sling were changed at four to five days intervals. After two weeks of the above management, the excoriations on the dorsum of the foot had healed substantially but the locomotor dysfunction was worse than before the management was initiated.

At this point, it was certain that therapeutic and conservative treatment would yield no positive results in the management of the case. It was then decided to manage the case using a tendon re-location technique.

Pre-Surgical Evaluation

A pre-surgical assessment/evaluation of the puppy a day before surgery indicated generally a good bodily condition with adequate hydration, a body weight of 13.5.0 kilogram, heart and pulse rates of 128 beats per minute, a respiratory rate of 24 cycles per minute, a rectal temperature of 37.9°C, and pink mucous membranes with a capillary refill time less than 2 seconds. A venous blood sample yielded haemogram values that were essentially normal viz: total RBC count - 7.8×10^6 cells/ml, PCV- 37%, Hb -13 grams/dl, total WBC count - 9×10^3 per ml and differential count of neutrophils - 68%, lymphocyte - 27% eosinophil -2% basophils 2% and monocytes 1%. On a risk

assessment basis, the puppy was considered a low risk patient.

Aseptic preparation and Anaesthesia

The entire right hind limb was prepared aseptically for a major surgery in a routine manner and the puppy was pre-medicated with a combination of atropine sulphate at 0.02 milligram per kilogram body weight and xylazine at 2.0 mg/kg body weight intramuscularly. General anaesthesia was induced with ketamine hydrochloride at 15.0 mg/kg body weight intramuscularly and maintained intra-operatively with the intermittent intravenous administration of a 1.25% thiopentone sodium solution at 10.0 mg/kg body weight via an over the needle cannula pre-placed in the cephalic vein.

Surgical Procedure

The puppy was placed in right lateral recumbency and a 5 to 6 cm linear skin incision was made on the medial aspect of the leg from the proximal 1/3 of the tibial region to just distal to the hock joint. The sub-cutaneous fascia was bluntly dissected and revealed the tendons of the gastrocnemius, superficial digital flexor, hallucis longus, the long digital flexor and the caudalis tibialis muscles starting from the caudal aspect of the incision towards the tibia bone. The SDFT was separated from the gastrocnemius tendon (GT) from the mid tibial region to just distal to the hock joint and was transected at this point. The proximal end of the transected SDFT was tagged with two stay sutures of 2/0 nylon. The tendon pulled with the aid of the stay sutures was then passed under the proximal transverse ligament and sutured side-to-side to the long digital extensor tendon LDET with five simple interrupted sutures of 2/0 monofilament nylon on an eye-less needle proximal to the point at which the LDET divides into the individual ligaments of the meta-tarsal bones and digits. The surgical site was constantly moistened with physiological saline through-out the procedure to avoid desiccation of the tissues at the surgical site. The muscles were not sutured, while the subcutaneous tissues were approximated with sub-cutaneous sutures of 2/0 chromic catgut. The skin incision was closed with horizontal sutures of size 0 nylon suture material.

Post-Operative Management

The recovery from anaesthesia was uneventful. The post-operative management included topical application of penicillin and streptomycin powders to the surgical site, intramuscular administration of penicillin and streptomycin at 10,000 IU and 20 mg/kg body weight respectively for four days, dressing of the surgical site, bandaging and placing the limb in an Elmer sling. Low doses of Oxymorphone (20 µg/kg/12hrs) were also administered to



Figure 2. Notice the hyper flexed hock (dropped hock) with the metatarsal bones being in contact with the floor. The region of the gastrocnemius tendon shows tautness.

the puppy intramuscularly for three days postoperatively. On removal of the Elmer sling and dressing six days later, the puppy was able to place the digits/paw normally without knuckling but with a dropped (hyper-flexed) hock when it stood (Figure 2).

The GT appeared intact on palpation and there was no depression (dimple) above the tuber calcis when the hock joint was manually over extended. Neurological examination revealed very little or no changes from the pre-surgical findings. The dropped hock complication was managed by placing the limb in a splint that was bent at the hock joint so that the joint assumed a near-normal angulation over a period of four weeks after which the puppy was able to stand and walk with the limb in appropriate positions both at the hock joint and the digits (Figures 3 and 4).

The puppy was eventually discharged to the owners. A follow-up of the case three weeks later showed appreciable clinical improvement as the puppy placed the limb properly and the hock joint was held in normal flexion though slightly abducted.

DISCUSSION

Tendon relocation/transplantation/transfer techniques have been employed to successfully manage a large spectrum of cases of peripheral neuropathies associated with limb dysfunctions in animals (Lesser, 1978; Lesser and Solimanss, 1980; Leighton, 1982). The primary cause of the peroneal nerve paralysis in the case reported appears to be trauma due to a dog fight. The nerve injury would probably have occurred through the bite wound noticed on the lateral distal aspect of the right thigh region



Figure 3. The limb with a dropped hock joint placed in a splint pre-formed to conform to the normal flexion of the stifle and extension of the hock joints.



Figure 4. Notice extension of the digits, flexion of the hock and extension of the stifle joints. The limb bore some weight and was close to normal positioning. The musculature over the tibial region had regained some mass.

when the puppy was first presented at the VTH of UA. It appears that an initial neuropraxia or axonotmesis caused by the dog bite may have been exacerbated by pressure on the nerve as a result of prolonged right lateral recumbency assumed by the patient, faulty application of dressings to the limb or spreading/intensification of inflammatory reaction around the nerve and has progressed to a neurotmesis. The very severe atrophy of

the muscles below the stifle of the affected limb seen at the later stage of the case seems to support this view. These have been cited as common causes of peroneal nerve paralysis by others (Bennett and Vaughan, 1976a, b; Hart and Tremblay, 1982). The peroneal nerve appears more prone to injury than the tibial nerve because it is closer to pressure points, is composed of larger folliculi and has less connective tissue between the folliculi than the tibial nerve. These factors render the nerve more likely to be traumatised than the tibial nerve (Bennett, 1976; Bennett and Vaughan, 1976a).

It would appear that cases of peripheral neuropathy in animals may be under reported given the wide range of its causation like intramuscular administration of medications in both pet and farm animals, surgical procedures around the hip and stifle joints, perineal herniorrhaphy, fights among animals and pelvic fractures and their repair. Some of these are part of routine case management in Veterinary Practices. In large animals particularly where highly irritant medications like long acting oxytetracyclines are traditionally administered in regions close to the sciatic, tibial and peroneal nerves, peripheral neuropathies are theoretically expected to be more common than is currently reported. This may be due to poor reporting, inadequate professional attention to large/farm animals generally or poor awareness of the occurrence of this condition in animals. This may account for the very few reports of the condition in large animals (Kilic et al., 2009; Kilic et al., 2014).

The clinical signs of peroneal nerve paralysis reported in this case are similar to those in most recent reports (Bennett and Vaughan, 1976a, b; Leighton, 1982). They became evident as soon as the patient was ambulant. The continuous maintenance of right lateral recumbency in preference to any other position during the initial presentation at the VTH was a fair clinical manifestation of an attempt to protect and hide an injured and painful part of the body from external interference (Hansen, 2003). This may have caused compression to the nerve or exacerbated the inflammatory response attendant to the initial deep bite wounds.

The poor result from therapeutic and conservative managements in the initial stage of this case agrees with the reports of Bennett and Vaughan (1976a, b), Lesser (1978), Leighton (1982), Forttre et al. (2007), Kilic et al. (2009). This poor outcome may be because in cases so managed recovery is dependent on the resolution of the neural damage which may take a much longer time than most clients may wish to wait. Traumatic injury to the tendon itself such as lacerations or transactions requires healing of the tendon and re-establishment of continuity before any recovery can occur. This process is reputedly slow due to the poor blood supply to tendons and the retraction of the tendon ends following such injuries. Such cases and those due to neural damage have a better chance of recovery following physical re-establishment of continuity of the tendon or relocation to it of a functional

tendon (Spinella et al., 2010; Knecht, 1985).

Most long-standing cases of peripheral peroneal neuropathy which had been managed unsuccessfully by conservative or/and therapeutic interventions have eventually been treated successfully by the relocation of one flexor tendon or the other to the tendon of the long LDET (Bennett and Vaughan, 1976a, b; Hart and Tremblay, 1982). In these and in the case reported, this last method of treatment appears superior to therapeutic or/and conservative management because of its simplicity and more for its better overall outcome.

The basic surgical principle involved in this technique is that most functions of muscles or tendons of the appendicular skeleton are performed by two or more muscles or tendons usually of the same group and innervations. Therefore, one of these muscles/tendons can be spared and used for relocation procedures without affecting the overall function(s) of the group of muscles/tendons. In the case reported, the main flexor of the hock and extensor of the digits- the LDET was denervated and paralyzed due to some damage to the peroneal nerve. On the other hand, the extensors of the hock and flexors of the digits- the GT, the SDFT, the HLT, the LDFT and the CTT all innervated by the tibial nerve were still innervated and functional. Of these, the SDFT, the LDFT and the CTT have been relocated to the LDET for the management of peroneal nerve paralysis in dogs while the HLT has been relocated for the treatment of chronically ruptured Achilles tendon in dogs all with acceptable results (Hart and Tremblay, 1972; Bennett and Vaughan, 1976a, b; Wapner et al., 1993). There is no previous report of a dropped hock joint as a post-operative complication sequel to this procedure irrespective of which flexor tendon was relocated. It may have occurred in this case because the splint applied postoperatively was removed too early or because the function of the GT was substantially compromised by the excision from it of the SDFT as the latter is the second most important component of the GT. The result of the management of the dropped hock joint in this case shows that it may be avoided if the external splint is maintained a little bit longer than six days post-surgically or the LDFT, CTT or the HLT which could be spared is used for the relocation rather than the SDFT. There does not appear to be any report of the re-location of the HLT for the management of peroneal nerve paralysis in dogs.

However, it has been used extensively in the repair of GT injury in humans (Mahalan and Dalal, 2009) and may well be used in dogs and other animals for the management of peroneal nerve peripheral neuropathy.

Although, this procedure is credited with a good prognosis, it must be carried out under optimum aseptic conditions, with minimal trauma to the tendons and with the surgical site kept constantly moistened with some physiological fluid to prevent tissue desiccation. Most non-absorbable suture materials seem suitable for the anastomosis of the tendons. In all, only moderate tension

should be applied to the simple interrupted sutures irrespective of the non-absorbable suture material used. It would appear that a side to side should be preferred to an end to end anastomosis of the tendons as it is easier to carry out, is less time consuming and preserves the integrity of the entire long digital extensor muscle and tendon so that it could return to normal function if subsequently the de-enervated muscle/tendon becomes re-innervated (Bennett and Vaughan, 1976a).

The long period of retraining or rehabilitation appears important for proper recovery as shown in this case and emphasised by others (Malnati, 198; Wapner et al., 1993). Such retraining could be just passive physical therapy like massaging, walking the dog on a leash, guided exercise or swimming (Hulse, 1990). In most cases, although limb placement in terms of extension of the digits returns to near normal a few days after the operation, appropriate hock flexion requires a period of external casting which must be done with care and the hock joint held at the appropriate angulation (Bennett and Vaughan, 1976a; Hart and Tremblay, 1982). Although a side to side anastomosis of SDFT to the entire (un-section) LDET was employed in this case, the anastomosis needed sufficient time to heal and organize properly to be able to withstand the strain and tension required to adequately hold the hock joint, in particular, in an appropriate weight bearing position.

Conclusion

Tendon relocation/transplantation procedures appear to be superior to therapeutic or/and conservative treatments in the management of peripheral mono-neuropathies. However, once an appropriate diagnosis of the condition is made, no time should be wasted on conservative and/or therapeutic management because the latter are unrewarding in the management of such cases.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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