

Semitendinosus Muscle Transfer Flap for the Treatment of Canine Fecal Incontinence

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Abstract : A 4-year-old intact female Maltese was referred to the Animal Medical Center of Chonbuk National University with a history of consistent fecal incontinence over 4 months following sacculotomy surgery. We suspected that anal sacculotomy resulted in loss of the external anal sphincter. On physical examination, the external anal sphincter muscle on the left side was intact, while the external anal sphincter muscle on the right side could not be detected and exhibited severe laceration. To repair the defect, the left semitendinosus muscle was transposed around the anus. The left semitendinosus muscle was isolated and transected near the stifle, reflected dorsally and passed around the ventral rectum into the pararectal fossa. Care was taken to preserve the integrity of the vasculature and nerve supply. The muscle was secured dorsally with simple interrupted sutures to the levator ani and coccygeus muscles to simulate the external anal sphincter. Nine days after surgery, the dog was defecating normally with no evidence of incontinence. The use of semitendinosus muscle flaps is a good option for the treatment of fecal incontinence secondary to loss of sphincter muscle in dogs.

Key words : fecal incontinence, semitendinosus muscle flap, dog.

Introduction

Fecal incontinence is the inability to control defecation, resulting in involuntary passage of feces. Causes of fecal incontinence are neurologic and non-neurologic. Usually, non-neurologic disorders (i.e., reservoir or sphincter incontinence) can be identified based on history and physical examination. Such causes include perianal fistula, trauma to the external anal sphincter, and prior anal sac surgery. Neurologic fecal incontinence is caused by neuromuscular disease (i.e., cauda equina syndrome or lumbosacral stenosis). Neuromuscular disease is suspected if an abnormal anal reflex is found, usually in conjunction with other neurologic defects in the anal, perineal, hindlimb, or coccygeal regions (7).

No drugs that reverse fecal continence are available, but opiate intestinal motility-modifying drugs and anti-inflammatory agents may be tried. Opiate intestinal motility-modifying drugs such as diphenoxylate hydrochloride and loperamide hydrochloride increase segmental contraction of the bowel and slow the passage of fecal material, thus increasing the amount of water absorbed from the feces. Anti-inflammatory agents such as steroids and sulfasalazine may benefit dogs with suspected reservoir incontinence due to inflammatory bowel disease or inflammation of the colon.

Sphincter-enhancing procedures for treating fecal inconti-

nence have not been investigated adequately, but some methods of enhancing total continence are available. One study reported that fecal incontinence was successfully treated by the implantation of a silicone elastomer sling to form an artificial external anal sphincter (4). Other surgical options for the treatment of fecal incontinence include transection of the nerve innervating a segment of the biceps femoris, complete replacement of the external anal sphincter, facial sling and semitendinosus muscle flap. The transected nerve innervating a segment of the biceps femoris is anastomosed end-to-side to the pudendal nerve to improve the weakened anal sphincter (12). Complete replacement of the external anal sphincter is accomplished using the latissimus dorsi muscle to restore voluntary anal continence (14). Fascial slings use two strips of tensor fasciae latae from the lateral thigh to enhance anal sphincter function (8). The semitendinosus muscle flap technique uses the semitendinosus muscle to simulate the external anal sphincter (5).

In the present case, we describe the use of a semitendinosus muscle flap to treat fecal incontinence in a small dog.

Case

A 4-year-old intact female Maltese was referred to the Animal Medical Center of Chonbuk National University with a history of consistent fecal incontinence and fistula after sacculotomy 4 months prior. On physical examination, the external anal sphincter to the left of the anus was intact, but to the right of the anus the external anal sphincter was not

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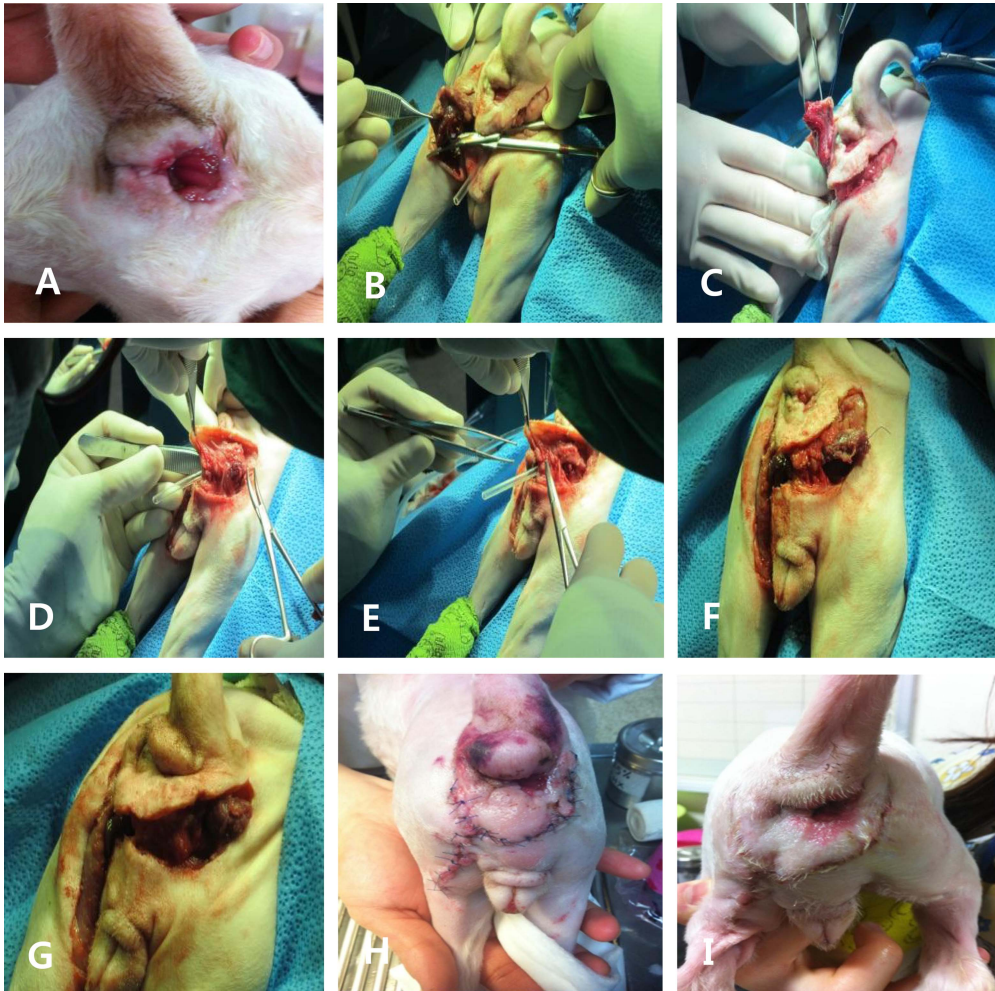


Fig 1. Semitendinosus muscle transfer flap. The external anal sphincter to the right of the anus was not detectable and severe laceration was observed (A). Subcutaneous tissue was dissected to expose the semitendinosus muscle (B). The left semitendinosus muscle was incised 2 cm proximal to its insertion into the tibia and the semitendinosus muscle was reflected dorsally (C). The semitendinosus muscle was passed around the ventral rectum and into the pararectal fossa (D). The muscle was secured dorsally with simple interrupted sutures (3-0 polydioxanone suture) to the levator ani and coccygeus muscles to simulate the external anal sphincter. Then, the semitendinosus muscle was secured to the surrounding tissues with a simple continuous pattern. The area was lavaged with sterile saline (E), (F), (G). The subcutaneous layer and skin were closed in a routine fashion (H). Posterior view 20 days after surgery (I).

detectable and severe laceration was observed. Some mucosa protruded from the anus, and the prolapsed mucosa became more evident with abdominal effort (Fig 1A). Perineal reflexes were normal on the left side of anus, but not on the right side. The patient was diagnosed with anal sphincter incompetence following deficiency of the right external anal sphincter. Four days after examination, the dog was fasted for 12 hours and anesthetized for surgical repair. One hour prior to surgery an enema was administered. Cefazolin (30 mg/kg, IV, Dongwha Pharm. Co. South Korea) and tramadol (3 mg/kg IV, Shinpoong Pharam. Co. LTD, South Korea) were given as premedication and propofol (6 mg/kg IV, Myung-moon Pharm. Co, South Korea) was administered as an induction agent, followed by isoflurane in oxygen administered through a cuffed endotracheal tube. The dog was positioned in sternal recumbency with the hind limbs over the padded edge of the surgery table, feces were removed from its rectum, and the tail was secured dorsocranially with an elastic band. A skin incision was made from the lateral aspect of the

right anus, curving ventromedially to the left side of the anus, and continuing distally along the caudal margin of the left hind limb to the level of the stifle joint. Subcutaneous tissue was dissected to expose the semitendinosus muscle (Fig 1B). The left semitendinosus muscle was incised 2 cm proximal to its insertion into the tibia. Most of semitendinosus muscle was transected near the stifle and cleared from the surrounding tissue, but its origin at the ischium was maintained. The muscle was reflected dorsally (Fig 1C), passed around the ventral rectum, and into the pararectal fossa (Fig 1D). Care was taken to preserve the integrity of the vasculature and nerve supply. The muscle was secured dorsally with simple interrupted sutures (3-0 polydioxanone suture) to the levator ani and coccygeus muscles to simulate the external anal sphincter. Then, the semitendinosus muscle was secured to the surrounding tissues with a simple continuous pattern (Fig 1E, F, G). The area was lavaged with sterile saline and the subcutaneous layer and skin were closed in a routine fashion.

An Elizabethan collar was used to prevent the dog from

licking the surgical site. Cefazolin (25 mg/kg, BID), tramadol (3 mg/kg, BID), meloxicam (0.1 mg/kg, SID), and famotidine (0.5 mg/kg, BID) were administered after surgery for 7 days. By 9 days after surgery, the dog was defecating normally and did not require daily medication for inflammation. Three months after surgery, the patient was consistently defecating normally with no evidence of incontinence. The owner was satisfied with the results and overall cosmesis, and the patient appeared to be pain-free.

This case was a good surgical candidate with fecal incontinence and we achieved good results using a semitendinosus muscle flap.

Discussion

Normal defecation and fecal continence are sustained by a complex interaction of nerve reflex and muscle activity (7, 13). Sphincter incompetence is caused by neurologic or muscular dysfunction (7). Continence is maintained by a combination of a passive high-pressure zone, created by the internal anal sphincter with intermittent external anal sphincter contractions creating an active high-pressure zone during periods of increased intrarectal pressure (6,7). Loss of the external anal sphincter may be congenital or the result of perianal fistula, trauma to the external anal sphincter, or prior anal sac surgery (7). In this case, the cause of fecal incontinence was anal sacculotomy 4 months prior to presentation. Many techniques are available to treat fecal incontinence. Attempts must be made to restore structural integrity, to increase the passive high pressure zone of the rectum, and to augment the action of the internal anal sphincter (5). A silicone elastomer sling is a good treatment for fecal incontinence in clinical cases, being well-maintained, and the material used is both relatively inexpensive and easily available. Use of silicone elastomer slings also requires a minimal amount of equipment and surgical expertise to place the material correctly. However, this procedure is often complicated by failure or recurrence of incontinence and infections (4). Complete replacement of the external anal sphincter using the latissimus dorsi muscle has been successful experimentally for restoring anal continence in dogs (14). Fascial slings easily enhance anal sphincter function, but may cause major complications including infection, dehiscence, and persistent tenesmus (8). Surgical procedures using vascular muscle may provide more stable and safer treatment of external anal sphincter incompetence. Muscles used for flap transfer without microanastomosis require adequate blood supply, easy accessibility, and the ability to reach the needed site without occlusion of the vascular pedicle. The muscle must also be large enough to cover the defects where necessary and be functionally redundant (8,11). Type-I muscles have one dominant vascular pedicle, and Type-II muscles have one dominant vascular pedicle and a minor vascular pedicle (3,9). Type-III muscles are supplied by two dominant vascular pedicles from the proximal and distal parts of the muscle. The semitendinosus muscle is a Type-III muscle in dogs (3,9). Type-I and Type-II muscles are ideal for use as transfer flaps (2,7,8,11). Transection of nerves innervating a segment of the biceps femoris is a ideal technique for the treatment of fecal incontinence, because

this muscle is a vascularized skeletal muscle that is innervated by the pudendal nerve. Such a procedure also preserves both the donor pudendal nerve and the original external anal sphincter. However, its application is characterized by many problems, including those associated with anatomic feasibility and functional outcomes (12).

The treatment of failed perineal hernia repair and atresia ani using the semitendinosus muscle flap has been described, but few cases in which semitendinosus muscle flaps were used for fecal incontinence have been reported (2,5). Although the semitendinosus is not an ideal muscle for use as a transfer flap, its use is made feasible by microanastomosis between the two major vessels supplying the semitendinosus muscle (1,10,11). Furthermore, this muscle can be easily dissected, and is long enough to reach the required site and cover the anatomical defect. Dissection of this muscle caused no lameness of the limb in our patient. Another advantage of semitendinosus muscle flap repair is its usefulness for reconstructions in which the ventral aspect of the perineum is severely affected, as in this case which presented with fistula after anal sacculotomy.

In this case, using the semitendinosus muscle flap led to a better prognosis than any other surgical alternatives for the treatment of fecal incontinence. There was no evidence of avascular necrosis of the muscle and no lameness of the left pelvic limb. The dog was able to defecate normally with no evidence of incontinence after surgery and the increase in support of the anus that resulted was evident upon rectal palpation.

Conclusion

The semitendinosus muscle flap reported here was used to obtain an excellent result in a canine patient with fecal incontinence and may be a viable alternative to euthanasia in similar cases. It is technically easy and especially useful when the ventral aspect of the perineum is severely affected. The use of the semitendinosus muscle flap is a promising technique for the treatment of fecal incontinence secondary to loss of sphincter muscle in a dog.

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