

Imaging the Nerve “Allograft to Muscle Target” Technique in Neuroma Management

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Summary: Neuroma management has gained significant attention in the peripheral nerve literature in the past decade. Alongside techniques such as targeted muscle reinnervation and regenerative peripheral nerve interface, another technique known as the “allograft to nowhere” has emerged. This approach involves the placement of an extended allograft at the end of a nerve, creating a regrowth zone in cases where muscle or nerve targets are not available. Although technique and outcomes research has been performed regarding the above techniques, there is a lack of imaging studies to examine postoperative outcomes. The authors present a case of recurrent neuroma management using a combined nerve allograft to nowhere + muscle target, supported by postoperative imaging. (*Plast Reconstr Surg Glob Open* 2024; 12:e6058; doi: 10.1097/GOX.0000000000006058; Published online 9 August 2024.)

Neuroma management has gained significant attention in the peripheral nerve literature in the past decade, largely driven by the introduction of targeted muscle reinnervation (TMR) and regenerative peripheral nerve interface (RPNI) techniques.¹⁻³ Alongside these well-established approaches, a novel technique for neuroma management has emerged, known as the “nerve allograft to nowhere.”⁴ This innovative approach involves the placement of an extended allograft at the end of a nerve, creating a regrowth zone in cases where muscle or nerve targets are not available. Although extensive research has focused on various aspects of these techniques before, during, and after surgery, there remains a lack of imaging studies that characterize the postoperative changes in patients undergoing neuroma management. The authors describe a case report of recurrent neuroma management after failed neurolysis and then failed RPNI, incorporating a combined nerve allograft to nowhere + muscle target, supported by postoperative imaging illustrating the absence of neuroma formation.

CASE REPORT

A 24-year-old right-hand dominant woman presented with a 3-year history of persistent right upper arm medial

antebrachial cutaneous (MABC) neuroma pain, unsuccessfully treated with medical management. The initial onset of pain occurred shortly after bilateral brachioplasty at an outside facility and remained unresponsive to therapy and medical treatment.

The patient underwent right upper arm MABC and lateral antebrachial cutaneous (LABC) neurolysis at another facility. Intraoperatively, scarring was observed on the medial antebrachial cutaneous nerve, while the lateral antebrachial cutaneous nerve appeared healthy. Axogen Avive amniotic nerve wrap was placed on the MABC nerve after neurolysis. Surgical intervention provided pain relief initially, until pain recurred in the proximal upper arm, axilla, elbow, and medial forearm at 10 months postoperatively. Neural blockades failed to alleviate the pain, and magnetic resonance imaging (MRI) revealed the presence of two MABC neuromas.

Subsequently, excision of both neuromas was performed, along with RPNI over the common MABC origin using a brachialis muscle graft to envelop the proximal stump at an outside facility. At 4 months postoperation, the patient presented to our institution with recurrence of right upper arm pain, leading to the initiation of nerve pain medication 8 months postoperation. MRI revealed multiple neuromas. [See figure, Supplemental Digital Content 1, which displays a medial antebrachial cutaneous nerve preoperative neuroma on sagittal T1 fat suppressed 3D post contrast VIBE sequence MRI demonstrating solidly enhancing soft tissue mass within the medial antebrachial cutaneous nerve (MABCN), reflecting neuroma (yellow

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Fig. 1. Medial right upper arm displaying MABC neuroma and associated scar.

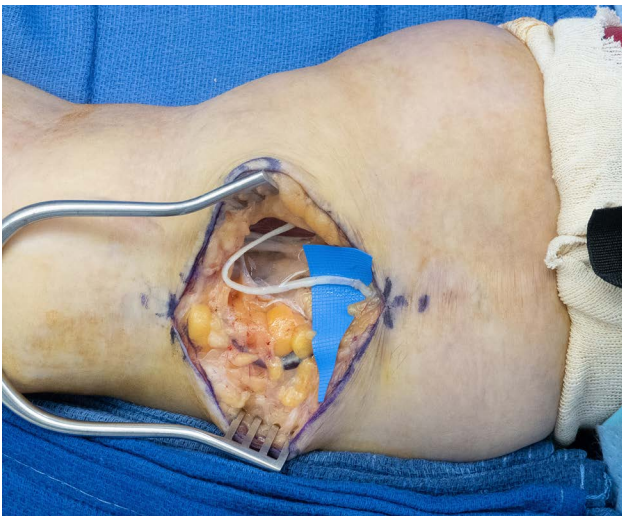


Fig. 2. Medial right upper arm with nerve allograft coapted to MABC proximally, with distal implantation to biceps muscle.

arrow). <http://links.lww.com/PRSGO/D422>.] [See figure, Supplemental Digital Content 2, which displays medial antebrachial cutaneous nerve preoperative neuroma on corresponding longitudinal grayscale ultrasound showing fusiform hypoechoic soft tissue mass involving the MABCN (red arrow). <http://links.lww.com/PRSGO/D423>.]

At this time, the patient was taking Lyrica 100 mg in the morning and 150 mg in the evening while still experiencing pain at the MABC neuroma site. Visual analog scale pain score was reported as 7. Subsequently, the patient underwent right MABC nerve neurolysis in the upper arm to address scar tissue followed by neuroma excision (Fig. 1). The proximal nerve stump was connected to an Axogen nerve allograft measuring 2–3 mm × 70 mm, which was also distally implanted into the biceps muscle (Fig. 2). One-month after the procedure, the patient reported pain improvement and decreased Lyrica use (down to 25 mg from 150 mg). At 2 months postoperatively, the patient discontinued Lyrica entirely, reported persistent pain improvement with visual analog scale score of 0, and

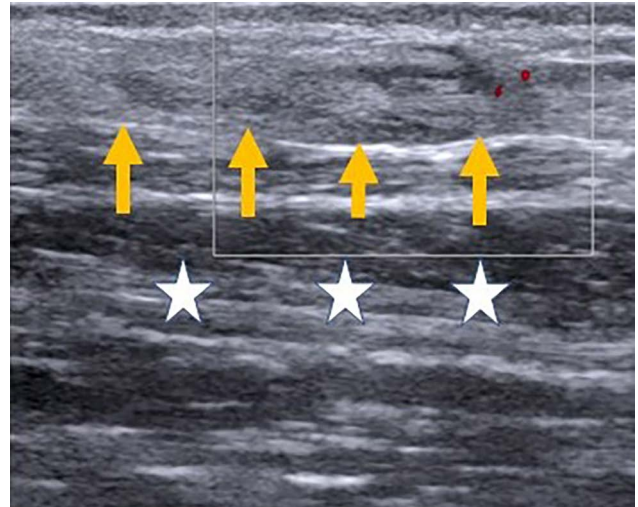


Fig. 3. Postoperative ultrasound. Longitudinal color Doppler ultrasound showing postsurgical changes of medial antebrachial cutaneous nerve neuroma excision with placement of allograft (yellow arrows). The anastomotic site appears intact, without appreciable neuroma formation, nerve discontinuity, or fluid collection. The allograft appears intact along its course (yellow arrows) and its coaptation implantation into the underlying biceps muscle (white stars).

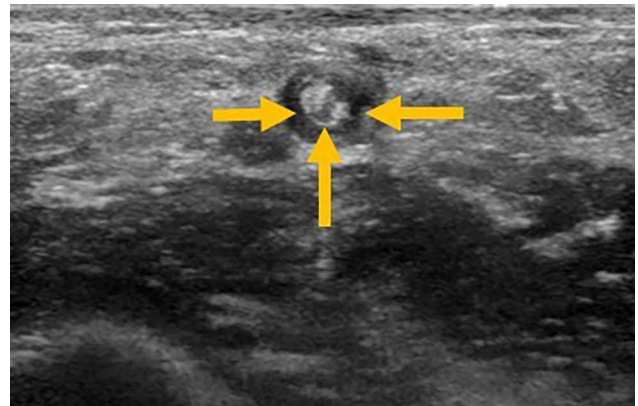


Fig. 4. Postoperative ultrasound. Axial gray scale ultrasound showing postsurgical changes of medial antebrachial cutaneous nerve neuroma excision with placement of allograft (yellow arrows). The anastomotic site appears intact, without appreciable neuroma formation, nerve discontinuity, or fluid collection. The allograft appears intact along its course (yellow arrows) and its coaptation implantation into the underlying biceps muscle (white stars).

expressed readiness to resume work. At 1 year postoperative, imaging confirmed the absence of postsurgical complications, with no neuroma formation at the proximal coaptation site or the distal coaptation site of the allograft within the biceps brachii muscle belly (Figs. 3 and 4).

DISCUSSION

Neuromas represent pathological proliferations of nerve tissue resulting from nerve injuries, trauma, or

surgical interventions. They frequently manifest at the sites of previous injuries, amputations, or surgical incisions, as observed in our presented case. Neuromas can lead to substantial postoperative pain, irritation, or phantom limb pain, with reported prevalence rates as high as 80%–90% in cases of amputation. Various surgical techniques have been used to mitigate these complications, including neurolysis, nerve capping, nerve grafting, and nerve ablation, each yielding varying degrees of success.¹ In recent years, TMR and RPNI have demonstrated efficacy in addressing postamputee pain and neuroma formation.^{2,3} TMR involves the transfer of a sensory or mixed nerve to a motor nerve target following amputation or limb loss, whereas RPNI involves the implantation of a nerve interface into a denervated muscle.

A lesser-known technique, the nerve allograft “bridge to nowhere,” has also been used in cases where traditional techniques have proven ineffective, similar to the presented case.⁴ This surgical procedure involves the placement of a nerve allograft to the end of the nerve of interest without a clear target for reinnervation. Applications of this technique are in situations where there is no muscle or available tissue to perform TMR or RPNI. In this procedure, the nerve allograft is coapted to the damaged end of the nerve and allowed to grow. Over time, the nerve fibers in the graft may begin to branch out and form new connections with nearby tissues, leading to improved function and sensation in the affected area. In our particular case, an adaptation in which a long allograft was directed toward a distal muscle target yielded a successful outcome in treating the neuroma. This adaptation involves extending the allograft such that it provides an avenue for nerves to regrow; however, the nerves are given such a long length to grow through that there is decreased growth through the allograft,⁵ and a muscle target for the fibers that do regenerate through the graft. This uses benefits of both nerve allograft bridge and TMR/RPNI techniques, decreasing the chance of neuroma formation. This technique may be suited for situations where TMR/RPNI are not anatomically feasible or have been tried unsuccessfully. It should be noted that this technique is not meant for optimization of future myoelectric prosthesis/nerve signal amplification, and only for treatment of neuroma.

Studies using imaging modalities such as MRI and ultrasound have provided valuable insights into the structural changes occurring after TMR. MRI has commonly been used to visualize the reinnervation of target muscles and the neural pathway establishment in patients who have undergone TMR, which correlate with improved motor function and reduced pain symptoms.^{3,6} Similarly, RPNI has been evaluated using imaging techniques to assess postoperative changes. Imaging modalities such as MRI and electromyography have been used to visualize the nerve-muscle interface and evaluate the degree of nerve regeneration and reinnervation. These imaging studies have shown promising results, confirming the formation of functional neural connections and improved sensory feedback in patients undergoing RPNI.^{1,6,7} In our patient, MRI was used to corroborate neuroma re-formation following unsuccessful excision and RPNI surgery.

In contrast to well-documented imaging of both TMR and RPNI techniques, there is limited literature exploring the specific imaging characteristics and outcomes following the technique used in our case of refractory neuroma post-RPNI, the adapted “allograft to muscle” technique. Although recent case reports have demonstrated the effectiveness of this technique using patient-reported outcomes like Patient-Reported Outcomes Measurement Information System and pain interference assessments, there remains a lack of imaging data confirming the absence of neuroma formation.^{8–10} This report, to the best of our knowledge, represents one of the first studies providing imaging confirmation that using a long nerve allograft with a muscle target is a viable technique for treating neuromas. As nerve imaging protocols become more popular, larger cohort studies may help guide surgical neuroma treatment techniques.

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DISCLOSURE

The authors have no financial interest to declare in relation to the content of this article.

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