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Repair of Symptomatic Forearm Hernias Using Acellular Dermal Matrix– Two Case Reports

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Abstract

Symptomatic forearm hernias are troublesome conditions that are infrequently presented in the literature. We describe a novel approach using cadaveric acellular dermal matrix in an inlay technique to restore fascia integrity. In two patients who perform heavy labor, this technique resolved their hernia related complaints and allowed them to resume their work activities.

Keywords

Forearm hernia; acellular dermal matrix

Fascial defects in the extremities with subsequent symptomatic herniation of muscle are rare but can be troublesome problems in active patients. Only fifteen cases of symptomatic forearm hernias have been described in the modern literature^{1–7}. Based on our experience with two patients who developed forearm hernias, this condition can be painful and may lead to subjective loss of strength and hamper heavy manual labor. The most optimal treatment of this condition is uncertain. In patients with delayed presentations, primary repair may not be technically feasible. In these cases, some advocate completion of the fascial release, which does not address the fascial defect problem¹. Other repair techniques have the disadvantage of requiring the use of additional donor sites such as tensor fascia lata^{2–3, 5} or alloplastic materials such as synthetic meshes⁴ that cannot become fully bio-incorporated. We describe two cases of symptomatic forearm hernias that were successfully treated by repairing the hernia using an acellular dermal matrix with an inlay technique that has not been previously described for forearm hernia repair.

CASE REPORTS

Case 1

A healthy 21-year-old right-hand dominant male laborer presented to clinic eight months after sustaining a paper roller (weighing approximately 3000 lbs) crush injury to his right arm. He did not have any forearm lacerations or fractures, but complained of forearm pain without evidence of compartment syndrome. His initial catalog of injuries included rib and scapula fractures. After recovering from his injuries, he complained of a painful bulge along the ulnar border of his mid-forearm when lifting weights and carrying heavy materials at work. On examination, he had tenderness over the flexor muscle bellies which protruded through a fascial defect during wrist flexion (Figure 1). The fascial edges were palpable, and we planned to repair the hernia defect by restoring fascia integrity with an acellular dermal matrix used in a fashion that has been shown to be effective in reconstructing abdominal hernias⁵.

An incision made directly over the muscle bulge revealed a 3×7 centimeter fascial defect (Figure 2). After undermining below the fascial edges for two centimeters, a piece of acellular dermal matrix (Alloderm, LifeCell Corporation, Branchburg, New Jersey) was sewn under the edges of the fascia with horizontal mattress sutures in an in-lay technique (Figure 3) using 2-0 synthetic, braided, non-absorbable sutures (Ethibond, Ethicon, Somerville, New Jersey). The tension of the repair was set so the dermal product was taut without the sutures pulling through the fascial edges. The skin was then closed in a layered fashion with deep dermal and superficial sutures. The patient was placed in a volar based, intrinsic-plus splint for ten days after the surgical procedure. He was kept on a 5-lb lifting restriction for one month and then increased to 15-lbs for another month. He was allowed to resume unrestricted activity two months after surgery. At one year follow-up, his symptoms were completely resolved and he had resumed unrestricted weight lifting and his manual labor work. There was no recurrent hernia or bulge on examination (Figures 4).

Case 2

A healthy 20-year-old right-hand dominant male, who was a self-employed landscaper, presented to clinic complaining of generalized right forearm ache after work. He had a volar forearm laceration from a knife injury three years before when the skin laceration was repaired in the emergency room. On examination, he had a well healed scar across his volar forearm. With active wrist and finger flexion, there was a 4×6 centimeter fascial defect with palpable edges and flexor musculature that bulged through the defect. This area was mildly tender at rest and became more painful with wrist flexion. His forearm hernia was repaired in a similar fashion as the previous case, except a different acellular dermal matrix was used (Flex HD, Ethicon, Somerville, New Jersey). By six weeks after the operation, he had already resumed full, unrestricted activity, albeit against medical advice, with resolution of his symptoms. At one year follow-up, the patient was happy with his result, had complete resolution of his presenting symptoms, and was working full-time as a landscaper. There was no evidence of a hernia or bulge on examination.

DISCUSSION

Symptomatic forearm hernias are most commonly located over the mid to proximal muscle belly¹. This injury can develop secondary to either a sharp penetrating injury or a closed crush mechanism. Although the pathophysiology of the pain and weakness from these fascial defects has not been studied, we agree with Olch et al. who believe that inflammation of the muscle bellies occurs after rubbing against the fascial edges and it is this continuous cycle of irritation and inflammation that can lead to pain in some heavy laborers¹. Not all patients are symptomatic from their hernia and the necessary size of the fascial defect to become symptomatic is not known. It is suspected that symptomatic hernias only occur when the fascial defect is limited to a partial length of the forearm¹. We also agree with Olch et al. that full length defects in the fascia are less likely to be symptomatic because these large defects do not impinge on the forearm muscles during heavy activities. Similarly, distal fascia defects over the tendons are likely asymptomatic, given the lack of change in cross sectional area of the tendons with flexion¹. In contrast, we believe that defects located over the muscle bellies can potentially be symptomatic due to the increase in cross-section area of the forearm muscles with contraction that results in outward pressure against the fascial defect and subsequent irritation of the muscle against the fascial edge.

Previous surgical management of these injuries has included a variety of techniques. We believe that primary closure of the defect is ideal, if possible, because it would most closely mimic the normal anatomy. To strengthen a primary repair, Roberts et al. described reinforcing the primary repair with a palmaris longus tendon graft weave². Although primary repair may be possible in the acute setting, the injury may not be initially recognized and neither of our patients developed symptoms until after resuming their normal activities following the trauma. In both of our patients, their presentations were delayed and primary repair was not possible because of the size of the defect and shortening of the fascia.

Completion of the fasciotomy is another approach to this problem and has shown some success when used in the lower extremity. However, Olch et al. reported in their series of seven patients undergoing completion fasciotomy for symptomatic upper extremity hernias, six patients had some improvement of symptoms, but only three of the six had complete relief and one patient continued to be bothered¹.

Others have also described repair techniques that attempt to reconstitute the fascial layers. Roberts et al. described two patients repaired with fascia lata grafts used in an onlay fashion². Although both patients did well, one developed an asymptomatic hernia at the donor site. This same technique was refined to an inlay repair, and was used successfully in one patient as described by Golshani et al³. To avoid the morbidity of a secondary donor site, Giladrino et al. had a successful repair of an upper extremity hernia using prolene mesh instead of fascia lata⁴.

Although a variety of techniques have been shown to successfully address the fascial hernia, each has potential disadvantages including incomplete symptom relief, secondary donor site complications, or the risks of alloplastic materials. We have developed a technique that addresses the anatomic defect without the need for a secondary donor site or use of

alloplastic materials. Acellular dermal matrices have gained widespread use over the last fifteen years including repair of abdominal wall hernias, breast reconstruction, and soft tissue augmentation^{8–11}. Dermal grafts are harvested from human cadavers and then decellularized to remove all immunogenic and potentially infectious components. The remaining dermal scaffold has been shown to provide sufficient tensile strength for complex abdominal hernia repair⁸ and can be re-vascularized when placed in an appropriate tissue bed⁹, allowing for bio-integration and increased resistance to infection¹². We believe this ability for bio-integration is an advantage of acellular dermal matrices compared to alloplastic meshes. Acellular dermal matrices have been well demonstrated to be safe and reliable materials for human usage^{11, 12} including in the upper extremity where these products have been used for coverage of radial forearm flap donor sites¹³ and in thumb carpometacarpal arthroplasty¹⁴. Porcine and bovine versions are also available commercially; however, we used human derived products in both of our cases due to the selection available at the individual hospitals and not for any specific biomechanical reasons. Based on the best available current evidence, there is no consensus on which product is better and each company's products have reported different handling property, cost, and applications, although none of this has been appropriately studied to define the better product¹².

In these two cases, we have demonstrated the successful repair of symptomatic forearm hernias using an acellular dermal matrix, without the potential problems associated with donor site morbidity. This technique has allowed for both patients to return to heavy labor.

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Figure 1.
Pre-operative demonstration of the forearm during active flexion showing the muscle bulging through the fascial defect.

Figure 2.
The fascia edges were separated from the underlying muscle. A crossing vein was preserved and a 3×7 cm defect was defined. The arrow identifies the fascial edges.

Figure 3.

A piece of acellular dermal matrix was trimmed to fit under the fascial defect. Horizontal mattress sutures were used around the periphery to secure the acellular dermal matrix underneath the fascial edge.

Figure 4.
Post-operative demonstration of the lack of muscle bulge with active flexion.