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Massive Ventral Hernias: Role of Tissue Expansion in Abdominal Wall Restoration Following Abdominal Compartment Syndrome

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Massive ventral hernias may result from a variety of clinical situations. One such clinical situation, a common problem in trauma patients, is abdominal compartment syndrome. Abdominal compartment syndrome frequently results in a massive abdominal defect when primary closure after surgical decompression is not possible. We offer a technique for repairing these massive ventral hernias by first expanding the lateral abdominal wall muscles, fasciae, and skin with tissue expanders and then closing the defect with elements of the "components separation" method. Additionally we present other clinical situations resulting in a massive ventral hernia that were repaired using this technique.

A BDOMINAL WALL RECONSTRUCTION of massive ventral hernias as a consequence of abdominal compartment syndrome (ACS) and other clinical situations that make primary closure difficult presents a unique surgical challenge. Massive abdominal hernias, which we define as greater than 11 cm in the largest dimension, are complicated by the extent of abdominal wall loss and subsequent tissue contraction. Many surgical techniques used today to close these large defects have limitations. Prosthetic mesh can cover large hernias but can cause adhesions, enteric fistula formation, and infection.[1, 2] Tensor fasciae latae and rectus femoris extra-abdominal flaps are limited by their size and arc of rotation and also have an inherent risk of fistula formation and infection. Donor site morbidity is not uncommon.[3-5] The rectus femoris free flap has been used with relative success for abdominal wall reconstruction, but along with donor site morbidity and infection this technique also requires microvascular expertise and may result in tissue necrosis.[6] Abdominal wall fascial release procedures without previous tissue expansion have limitations in the ability to advance the fascia and frequently interrupt the innervation and blood supply to the abdominal wall.[7, 8]

ACS, defined as the adverse physiologic consequences that occur as a result of an acute increase in intra-abdominal pressure,[9] is usually treated by de-

compressive laparotomy via a midline incision. Subsequent closure of the midline wound may not be possible due to ongoing bowel edema or intra-abdominal infection. To functionally restore the abdominal wall in this situation we describe a technique combining the method of tissue expansion of the lateral abdominal wall musculature, fasciae, and skin described by Jacobsen and colleagues[10] and elements of the components separation method for definitive closure introduced by Ramirez.[7] We present four patients (including one who had ACS) with massive ventral hernias that were repaired using these two techniques.

Anatomy

A description of the abdominal wall anatomy is present in numerous surgical texts[11, 12] and is beyond the scope of this article. The lateral abdominal wall fasciae and musculature derive their blood supply primarily from the intercostal, lumbar and the deep epigastric arteries. The innervation comes from the seventh to 12th intercostal and the first lumbar nerves. These intercostal and lumbar vessels and nerves travel from the posterior midline to the anterior midline in an oblique, anterior pathway between the internal oblique and transversalis muscles. The vasculature and innervation to the rectus abdominis muscle follow this same pathway.

Because of the aforementioned anatomy of the abdominal wall, vertical incisions in the abdominal wall musculature can disrupt both the vasculature and innervation to the external oblique, internal oblique, transversalis and rectus abdominis muscles. A trans-

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verse incision at the costovertebral margin through the external oblique fascia will avoid the major vessels and nerves to the abdominal wall and allow for blunt dissection between the external and internal oblique. The relative avascularity and absence of nerves between the external and internal oblique fasciae from the anterolateral abdominal wall to the lateral border of the rectus sheath provides an ideal plane for blunt dissection and subsequent expander placement in this space. This space is bordered superiorly by the costovertebral margin, medially by the lateral border of the rectus sheath, laterally at the mid-axillary line, and inferiorly by the inguinal ligament.

Technique

Once ACS is identified in a trauma patient the abdomen is surgically decompressed via a midline laparotomy incision. Subsequent primary closure after abdominal decompression is sometimes possible. However, if bleeding due to coagulopathy persists and abdominal repacking is necessary or if visceral edema makes recurrent ACS likely an alternative abdominal closure that can compensate for additional intra-abdominal volume is necessary. An empty gas-sterilized 3-liter saline IV bag is sutured to the fascia and functions as a temporary abdominal closure. The wound is then packed with moist sterile dressings. Definitive closure is attempted in 24 to 72 hours. If definitive closure is not possible the saline bag is removed and an absorbable polyglycolic acid mesh is sutured to the fascia to prevent evisceration. A split-thickness skin graft (STSG) is later applied over the granulated viscera. A massive ventral hernia is accepted and usually repaired approximately one year later. Others prefer mobilizing and suturing the skin flaps together as an alternative to using an STSG.[13] At this point each patient is left with a massive ventral hernia covered with an STSG (Fig. 1). The skin graft is allowed to mature for at least 12 months allowing enough time for graft separation from the viscera. A 5-cm superficial incision is made along the costal margin. Dissection is continued through the subcutaneous tissue until the external oblique fascia is visualized. The external oblique fascial layer is incised following the downward and medial course of its fibers. The medial extent of the incision is the conjoining of the external and internal oblique fascia. Blunt dissection is then possible within the areolar plane between the external oblique muscle and the superficial aspect of the internal oblique fascia.

Long rectangular or crescent-shaped tissue expanders with remote injection ports (PMT Corporation, Chan Hassen, MN) anywhere from 450 to 950 FFcm^3 depending on the size of the defect are placed verti-
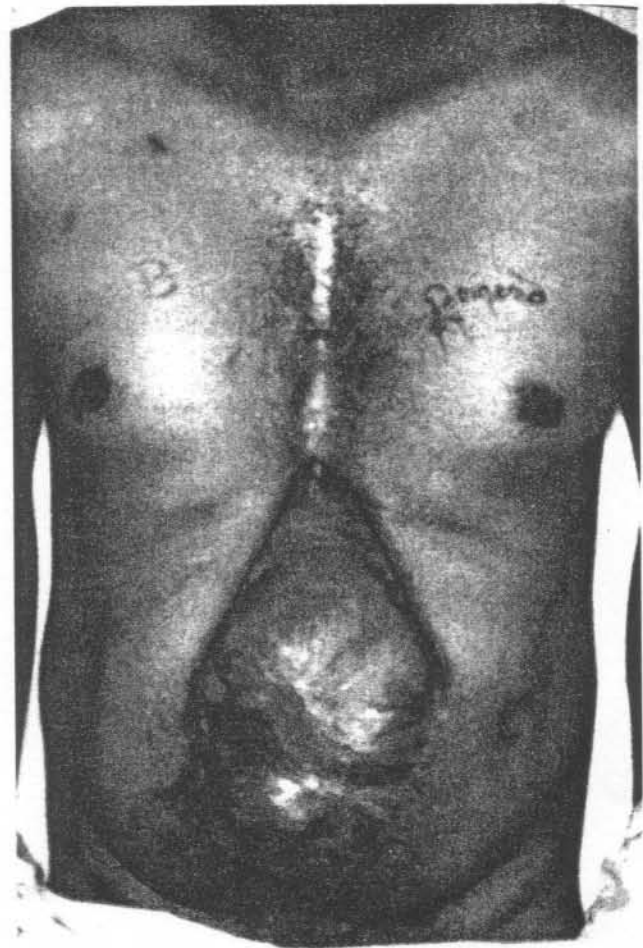

FIG. 1.   Ventral hernia 12 × 18 cm and covered with an STSG.

cally into the pocket just created (Figs. 2 and 3). The remote injection port is placed adjacent to the expander either medially or laterally. A mixture of dilute methylene blue and normal saline is injected into the expanders to allow for easier cannulation of the port during the saline injections.

The external oblique fascia and skin are then closed. The same technique is applied to the contralateral side. The wounds are then allowed to heal for at least 3 weeks before the initial expansion. Expansions are then performed weekly or biweekly with as much saline injected as tolerated by the patient. The patient is given an oral analgesic just before and during the saline injections to help ameliorate the pain of tissue expansion. As much as 875 cm^3 of saline into the 450-cm^3 expanders and 1130 cm^3 of saline into the 950-cm^3 expanders is injected over 4 to 9 months depending on the size of the defect to be covered (Fig. 4). If there is concern about the structural integrity of the expanders, CT of the abdomen may be performed. However, in our clinical experience we have not experienced failure of a tissue expander.

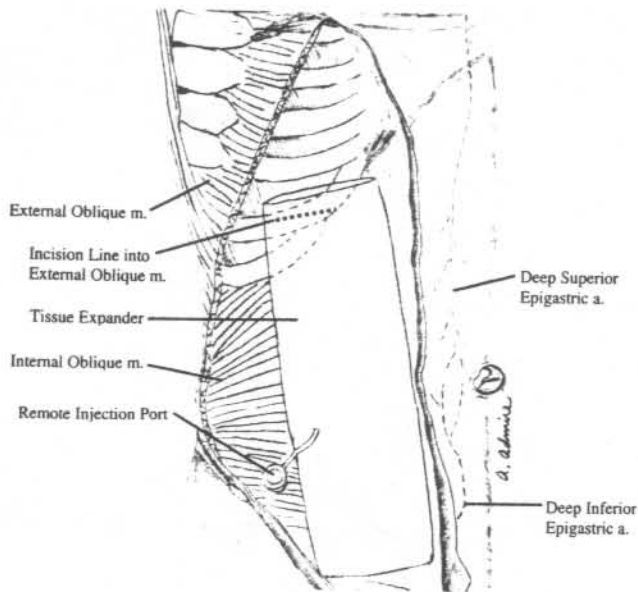To achieve adequate coverage we make sure the

FIG. 2.   Tissue expander placement site in relation to the anatomy of the anterolateral abdominal wall.

width of each expander subtracted from the distance of the arc of the expanded tissue is greater than one-half the defect. The value of placing expanders on each side of the defect should be obvious, as it quickens the expansion process and allows for a midline approximation of the skin and fascia.

In anticipation of definitive abdominal wall closure patients undergo routine mechanical bowel preparation the day before surgery. After general anesthesia a rapid intraoperative expansion is performed with as much as 400 cm^3 of saline injected into each expander. The STSG is sharply excised from the underlying viscera with care taken to avoid enterotomy.

The expanders are then drained. Next the skin borders are undermined to allow for tension-free closure. Dissection is then performed between the rectus abdominis muscles and the posterior rectus sheath as described by Ramirez et al[7] for maximum medial ad-
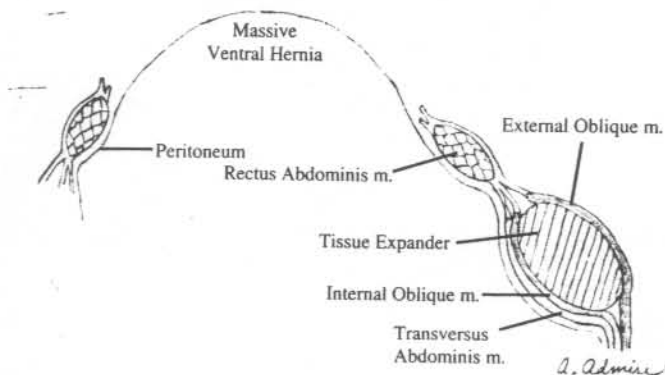

FIG. 3.   Cross-sectional schematic of a massive ventral hernia showing tissue expander placement site in relation to the abdominal wall musculature.
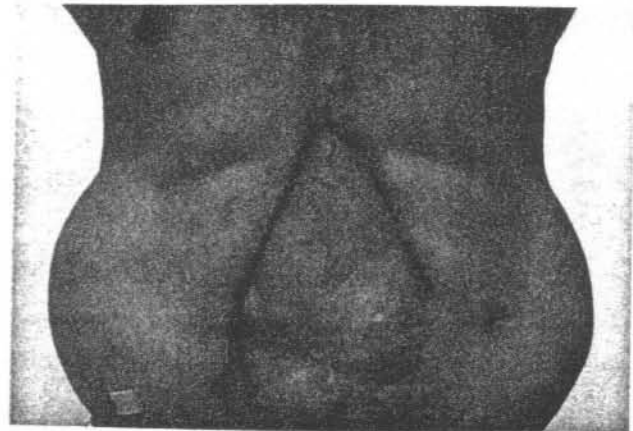

FIG. 4.   1110 cm^3 saline injected into the right expander and 1120 cm^3 into the left expander over 5 months.

vancement of these muscles. The posterior rectus sheath is then incised by making a vertical incision along the entire length of the posterior sheath. Ramirez et al[7] do not include scoring the posterior rectus sheath as part of the components separation method. However, it is our experience that this technique allows for further medial advancement of the abdominal wall muscles and fasciae.

Peak inspiratory pressure (PIP) is carefully monitored throughout the procedure. If the PIP rises by more than 5 to 10 cmH$_2$O over baseline at any time during closure we consider abandoning complete closure at this time following the recommendations of Jacobsen et al[10] If the PIP is determined to be adequate the abdominal wall muscle, fasciae, and skin are then closed in the midline.

Closed suction bulb drains are placed in the wound before skin closure if necessary. Patients are discharged after the return of bowel function. Patients are given specific instructions not to lift anything over 5 lb for a minimum of 4 to 6 weeks and are followed on an outpatient basis (Fig. 5).

Case Reports

Case One

A 23-year-old man presented with a 12 × 18-cm ventral hernia and an STSG covering the defect. This patient suffered a gunshot wound previously and had a complicated hospital course, which included ACS requiring surgical decompression and a small bowel fistula. Ultimately the fistula was resected and an STSG was placed, which left a large abdominal wall defect. Eleven months later one 950-mL expander was placed on each side of the defect. The expanders were incrementally filled to a total of 1110 cm^3 on the right side and 1120 cm^3 on the left side on an outpatient basis over 5 months. Then the skin graft was excised and the adhesions were lysed in preparation for primary closure. The expanders were emptied and removed and the
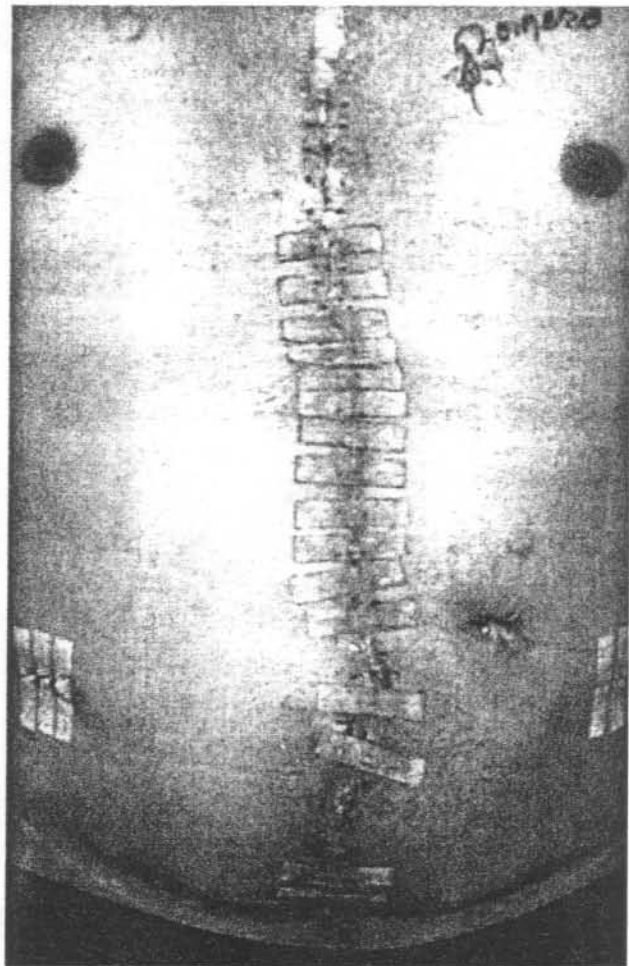
FIG. 5. Two weeks after bilateral tissue expander removal and primary abdominal wall closure.

hernia was closed primarily using the components separation method.

Case Two

A 55-year-old man presented with a 13 × 20-cm ventral hernia covered with an STSG. The patient had previously sustained a spontaneous rupture of the esophagus with an unsuccessful gastric pull-up. Ultimately a jejunostomy was placed on the left side of the abdominal wall. Seven months after skin graft placement a 950-mL expander was placed on the right side the abdominal wall defect. However, as a result of numerous adhesions from previous surgeries a plane between the external and internal oblique fasciae could not be dissected on the left side. A skin pocket was created and a 450-mL expander inserted. The right expander was incrementally filled to 1130 cm^3 and the left expander incrementally filled to 875 cm^3 over 5 months. After 12 hours of surgery gastrointestinal continuity could not be restored and because of marked intra-abdominal swelling only partial closure was achieved.

Case Three

A 78-year-old man presented with a 4 × 30-cm ventral hernia covered with an STSG. The patient previously had a gastrectomy with a Billroth II reconstruction for gastric cancer. The patient had a complicated postoperative course after the gastrectomy, which included an intra-abdominal abscess and wound dehiscence. The dehiscence led to the placement of polyglycolic acid mesh, and then later an STSG was applied. Nineteen months after skin graft placement, 950-mL expanders were placed on each side of the wound. The expanders were incrementally filled to a total of 845 and 850 cm^3 on the right and left side respectively over 4 months. Then the skin graft was excised as well as the adhesions. The expanders were emptied and removed, and the fascia was advanced bilaterally, using polypropylene mesh for reinforcement. Closed suction bulb drains were placed in the wound and the abdominal wall closed primarily.

Case Four

A 53-year-old man presented with a 7 × 11-cm ventral hernia covered with polypropylene mesh and an STSG. The patient previously had two abdominal operations for a liver transplant and a kidney cyst excision. The patient had a complicated hospital course following these surgeries. The patient developed a ventral hernia that was repaired with polypropylene mesh. The mesh subsequently became infected, and the patient was left with a chronic nonhealing midline wound. Two years after the liver transplant 750-cm^3 tissue expanders were placed in the lateral abdomen. One month later the right expander became infected and was removed, while expansions were continued on the left side. The left expander was incrementally filled to 920 cm^3 over 9 months. Three months later the remaining nonabsorbable mesh and STSG were then excised and the wound was closed primarily.

Discussion

Richardson and Trinkle[14] first recognized the phenomenon of ACS in the early 1970s, and although ACS can occur with any major abdominal operation it is most frequently seen in trauma patients. The increase in intra-abdominal pressure results in deleterious cardiovascular, pulmonary, and renal changes. Consequently a decrease in cardiac output, increased peripheral resistance, increased PIPs, decreased pulmonary compliance, hypoxia, hypercapnea, and oliguria leading to renal failure occur.[13, 14] The definitive treatment for ACS is surgical decompression. Subsequent primary closure is possible in some of these cases.

However, when definitive closure is not feasible after decompression for ACS a massive ventral hernia covered with an STSG is then accepted temporarily. We believe our technique of tissue expansion and abdominal wall closure has many benefits and provides the most functional restoration of the abdominal wall. The lateral abdominal wall is usually free of scars and defects after major abdominal surgery and provides a

well-vascularized soft-tissue donor site. The abdominal wall can be anatomically restored with minimal tension and without compromising the integrity of the abdominal muscles, vessels, and nerves. This technique can still be used even if trauma or multiple surgeries compromise the structural integrity of the rectus sheath and rectus abdominis muscles. There is minimal donor site morbidity as compared with the tensor fasciae latae and rectus femoris extra-abdominal rotational flaps and the rectus femoris free flap. This technique has the added benefit of being applicable to a wide variety of abdominal wall hernias.

The technique of inserting tissue expanders between the external and internal oblique fasciae creates minimal disruption of the nerves and vessels of the lateral abdominal wall and also avoids some of the disadvantages of other modalities. Hobar et al.[15] and Byrd and Hobar[16] have described placing tissue expanders through the rectus sheath in the plane between the internal oblique and transversalis fascial layers. However, the structural integrity of the rectus sheath is usually compromised in massive abdominal hernias due to multiple laparotomies and attempts at closure, which limits the ability of this technique. The nerves and vasculature can also be easily damaged. Carlson et al.[17] and Paletta et al.[18] describe placing tissue expanders into a subcutaneous pocket superficial to the abdominal wall musculature and fasciae. However, prosthetic mesh is then required for fascial closure, which can lead to the complications related to using mesh including infection, fistula formation, and adhesions as mentioned earlier.

The disadvantages of our technique in the repair of massive ventral hernias include the requirement of a two-stage closure: the first stage for tissue expander placement and the second stage for definitive abdominal wall closure. Good patient compliance is also required for weekly or biweekly expansions. Patient discomfort during the saline injections can prolong the time necessary to obtain adequate tissue expansion. Finally the expanders can become infected and require immediate surgical removal. However, wound infections, fistulas, tissue necrosis, or partial or total flap loss that can occur with the alternative methods may result in more surgical procedures than our two-stage repair technique. Also it has been our experience that patient compliance with serial saline injections is quite good when the patient is properly informed about the importance of weekly or biweekly expansions. The pain during the expansions is usually tolerable when the patients are given an oral analgesic just before and during the injection of saline. Lastly the infection risk of tissue expanders used in abdominal wall reconstruction appears minimal, as several authors[10, 15–18] have reported few expander infections in their cases.

Three of the four patients in this study (with Case Two being the exception) were able to resume their normal activities within a few months after their final abdominal wall reconstruction. Furthermore none of the patients have experienced hernia recurrence with our technique. However, some complications did occur in three of the four patients. In Case Two a clear plane between the internal and external oblique fasciae to the left of the abdominal wall defect could not be dissected because of numerous adhesions from previous surgeries. Therefore a skin pocket was created for the left abdominal expander. This patient also suffered from significant visceral edema necessitating partial closure. In Case Three reinforcement of the fasciae with polypropylene mesh was necessary for primary closure. This patient subsequently developed an abdominal wound infection 2 weeks after closure and was reopened with a delayed primary closure 3 months later. This patient has not suffered any major complications since. In Case Four the patient developed an infection of the right abdominal expander. However, this infection may be at least partially attributable to concurrent immunosuppressive therapy following his liver transplant.

We believe that this technique may be used commonly when primary closure after decompression for ACS cannot be achieved and a massive ventral hernia ensues. Our technique can also be used successfully for all clinical situations that result in a massive midline abdominal wall defect.

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