

# Abdominal Compartment Syndrome After rEVAR

How to identify and treat this serious complication of endovascular repair for ruptured abdominal aortic aneurysms before it's too late.

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The benefits, ease, and utility of endovascular aneurysm repair (EVAR) for ruptured abdominal aortic aneurysms (rAAAs) were first reported by Ohki and Veith in 2000.<sup>1</sup> Since then, it has become a preferred method of repair for treating rAAAs. Although recent data meta-analyses do not show an advantage of EVAR over elective open repair for AAAs, national and state published data have shown that EVAR for rAAAs (rEVAR) is the optimal treatment method.<sup>2-6</sup> Overall mortality rates with this approach are < 30%. As is also evident with open repair, achieving the best results with rEVAR relies upon coordinated systems of care, as well as the surgeon's expertise.

With the adoption of an endovascular approach to treating rAAAs, certain complications that are not associated with open repair become evident. These include abdominal compartment syndrome (ACS), early and late endoleaks, and late graft failure with sac expansion prompting reintervention and endograft explant. The most significant of these complications in the early postoperative period is the development of ACS. The combination of retroperitoneal hematoma left in place after REVAR and bowel edema can contribute to intra-abdominal hypertension and the development of this complication.

## DEFINITIONS AND INCIDENCE

Intra-abdominal pressure (IAP) normally runs in the range of 5 to 7 mm Hg in critically ill patients.<sup>7-9</sup> After rAAA repair, some degree of elevation of IAP is expected

(> 12 mm Hg). When the patient's IAP is > 12 mm Hg, some degree of renal impairment occurs. With a further increasing IAP, a greater degree of multiorgan system dysfunction occurs, and an IAP > 30 mm Hg is associated with multisystem organ failure.

IAP is most easily measured by means of bladder pressure through a urinary drainage catheter. There are several definitions of ACS: (1) IAP > 20 mm Hg in combination with organ system dysfunction or failure or (2) abdominal perfusion pressure (mean arterial pressure minus IAP) < 60 mm Hg along with organ dysfunction.<sup>7</sup> The second of these is important, as many patients may have some relative degree of hypotension after rAAA repair.

The development of ACS after surgical treatment for rAAA has also been reported after open repair.<sup>10,11</sup> The incidence of IAP > 20 mm Hg after open repair occurs in roughly 50% of patients. In contrast, the incidence after EVAR for rAAA patients, who are routinely monitored for this complication and treated expeditiously, was reported as 20% in the series by Mayer et al.<sup>12</sup> The implication of this is significant, as the mortality of patients with and without ACS was 30% versus 8%, respectively.

Aside from the aforementioned clinical markers, chemical markers have been studied in an attempt to diagnose the onset of ACS at an earlier time.<sup>13</sup> Horer et al placed microdialysis catheters into the peritoneal cavity and found that elevated lactate/pyruvate ratios and glycerol levels were early markers that were associated with the subsequent development of ACS.



**Figure 1.** On-table decompression laparotomy and colectomy for ACS after EVAR.

These markers most likely represent the inflammatory response that develops before overt clinical organ dysfunction and may provide a window of opportunity to reduce the mortality associated with ACS after rAAA repair.

### NONOPERATIVE TREATMENT

Decompressive laparotomy has been the routine treatment for managing patients with ACS. However, several authors have suggested nonoperative treatments, either as an adjunctive or a definitive treatment for ACS. If abdominal pain is the cause of a tense abdomen, Cheatham et al have suggested the use of epidural anesthesia to reduce IAP.<sup>9</sup> In the postoperatively ventilated patient, the use of neuromuscular blockade may also significantly reduce IAP. As reported by Papazarian et al, a short course of neuromuscular blockade may reduce IAP by 50%.<sup>14</sup> The problem in interpreting these data is that most of these studies were performed in patients with respiratory distress syndrome and not specifically those vascular patients who had undergone rEVAR. The importance of this nonoperative treatment is that it may be used if ACS is detected early or as a bridge to definitive decompressive laparotomy.

Another interesting modality is the use of positive end-expiratory pressure in ventilated patients with intravenous hypertonic albumin and furosemide treatment as reported by Cordemans et al.<sup>15</sup> This combination has been referred to as the PAL (PEEP albumin lasix) treatment and has been reported to result in



**Figure 2.** Three months after decompression laparotomy and VAC wound therapy.

negative fluid balance, reduce IAP, and reductions in mortality after open AAA repair.

An additional alternative to laparotomy is the use of tissue plasminogen activator for lysis of the retroperitoneal hematoma.<sup>16</sup> This technique has been described by Horer et al and involves CT guidance to place microcatheters into the retroperitoneum. The vast majority of patients in this series (out of 13 total) had IAPs > 20 mm Hg, intra-abdominal perfusion pressure < 60 mm Hg, and signs of multisystem organ failure. All but one patient responded favorably to this treatment. The authors concluded that this treatment may be used in selected cases but should not replace decompressive laparotomy.

### DECOMPRESSIVE LAPAROTOMY

In the clinical scenario where the IAP is elevated or the intra-abdominal perfusion pressure is reduced in association with multisystem organ failure, the performance of decompressive laparotomy can be a lifesaving procedure. This procedure is best performed earlier rather than later. If other aforementioned nonoperative adjunctive measures or procedures are used, close monitoring of the clinical sequelae and end-organ perfusion is absolutely necessary. Mortality rates after delayed recognition of ACS have been reported at 70%.<sup>17,18</sup>

In general, decompressive laparotomy is performed through a midline incision. Our present protocol involves initial midline decompression with laparotomy pads placed and coverage with plastic adhesive

drapes (Figure 1). The patient is kept on ventilator support and resuscitated appropriately. The patient is then re-examined after 48 hours. At that time, a decision is made about whether to plan serial returns to the operating suite for cephalad and caudad midline fascial closure.

Alternatively, a Wittmann patch (Starsurgical, Inc.) is placed, and closure is performed as previously described.<sup>19</sup> Some authors have also recommended the use of Vicryl (Ethicon, a Johnson & Johnson company) or prolene mesh with serial coverage.<sup>8</sup> One caveat to the use of any patch type of closure is the possible development of bowel erosions or fistula. Some patients have also been managed with VAC wound therapy devices (KCI, an Acclivity Company). Some patients managed in this way have had their residual skin/fascial defects covered with split-thickness skin grafts (Figure 2). Staged late hernia repair has been performed in these patients with good outcomes.

## INSTITUTIONAL EXPERIENCE AND RECOMMENDATIONS

Between 2002 and 2014, 184 patients underwent rEVAR, and the overall operative mortality rate was 21% (38/184). The incidence of ACS requiring decompressive laparotomy was 17% (32/184). The mortality rate without ACS was 12% (18/152) versus 66% (21/32) with ACS. What remains clear from these data is that ACS is still a significant issue after rEVAR.

In our experience with patients undergoing rEVAR, we found several factors that were associated with the development of ACS.<sup>20,21</sup> Patients with ACS had a higher incidence of (1) use of an aortic occlusion balloon for systolic blood pressure < 80 mm Hg, (2) coagulopathy as measured by markedly elevated partial thromboplastin times, (3) large-volume transfusion, and (4) intraoperative conversion from a bifurcated to an aorto-uni-iliac device due to an inability to cannulate the contralateral gate when compared to patients without ACS.

At present, our management of patients during and after rEVAR involves the following: (1) avoiding systemic anticoagulation with prompt correction of any coagulation defects, (2) checking IAP via bladder pressures hourly, and (3) routinely initiating treatment with decompressive laparotomy for IAPs > 20 mm Hg with end-organ dysfunction such as reduced urinary output or ventilator difficulties with peak airway pressures. We would also consider pre-emptive decompression in patients with either aortic occlusion balloon use for hypotension, massive transfusion (> 8 units PRBC), coagulopathy (partial thromboplastin time ratio > 3), or signs of end-organ dysfunction. ■

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