

Management of Lethal Complications Following a Ruptured Abdominal Aortic Aneurysm: A Case Report and Literature Review

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Abstract

Objective: This case report aimed to introduce key therapies and improve survival for patients with lethal complications following ruptured abdominal aortic aneurysm (rAAA).

Case report: We present a case of a patient with rAAA who developed hypovolemic shock, abdominal compartment syndrome (ACS), acidosis and intestinal perforation. Due to the successful management of damage control operation (DCO), conservative fluid strategies and renal replacement therapy, he recovered and was discharged in the end.

Conclusion: Early detection, suitable operation method and negative fluid resuscitation are key treatments of rAAA.

Keywords: Abdominal compartment syndrome; Complicated intra-abdominal infection; Intestinal perforation; Ruptured abdominal aortic aneurysm.

Abbreviations: ACS: Abdominal Compartment Syndrome; APACHE-II: Acute Physiology and Chronic Health Evaluation II; CT: Computed Tomography; CVVH: Continuous Renal Replacement Therapy; DCO: Damage Control Operation; EVAR: Endovascular Aneurysm Repair; IAH: Intra-abdominal Hypertension; IAP: Intra-abdominal Pressure; ICU: Intensive Care Unit; MODS: Multiple Organ Dysfunction Syndrome; rAAA: Ruptured Abdominal Aortic Aneurysm; TAC: Temporary Abdominal Closure

Introduction

Abdominal compartment syndrome (ACS) is defined as sustained intra-abdominal pressure (IAP) of >20 mmHg with the presence of an attributable organ failure [1]. Intra-abdominal hypertension (IAH) may contribute to splanchnic hypoperfusion, intestinal perforation and multiple organ failure [2]. A major cause of morbidity and mortality after ruptured abdominal aortic aneurysm (rAAA) is ACS, which is estimated to develop in 20% of the patients [3]. In the largest series of patients with rAAA treated with endovascular aneurysm repair (EVAR), mortality of ACS patients was 30% [4].

Case Report

A 69-year-old man presented our emergency department with a 12-hour history of acute severe abdominal pain, back pain and nausea. On arrival, his blood pressure was 132/100 mmHg, pulse rate was 72 beats per minute, and body temperature was 36.5°C. Physical examination disclosed tenderness over the left mid lower abdomen. Computed tomography (CT) scan showed abdominal aortic aneurysm (11.3*7.7 cm) with hematoma and vessel thrombosis (Figure 1). The patient had a medical history of hypertension and chronic cholecystitis. Diuretics were taken irregularly for the hypertension, but he withheld taking his anti-hypertensive drugs before the onset of pain. Although he took annual physical examination (ultrasound or other non-invasive assessments), he wasn't aware of the aneurysm before the acute abdominal pain. His symptoms worsened 6 hours later: his pulse

rate was 140 beats per minute and blood pressure was 79/55 mmHg. In consideration of the possibility of aortic aneurysm rupture, we performed emergency surgery on him immediately. In view of the appearance of CT (proper aneurysm's neck and favorable iliac arteries), significantly less operating time and blood loss and superior perioperative results of EVAR, the patient was considered fit for EVAR

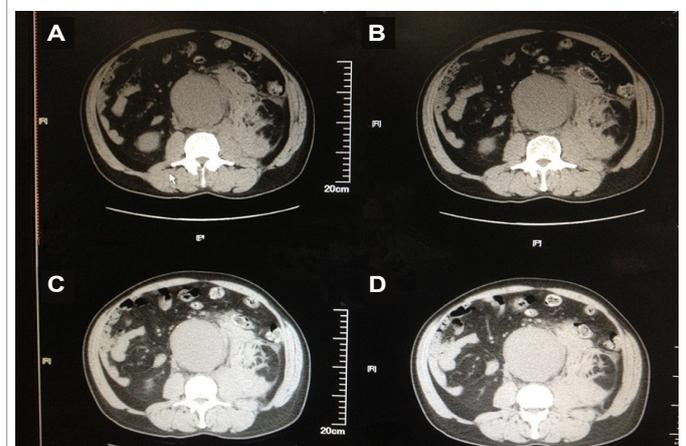


Figure 1: Computed tomography scan showed abdominal aortic aneurysm (11.3*7.7 cm) with hematoma and vessel thrombosis.

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and local anaesthesia. Three endovascular stent grafts (ENDURANT) were implanted into artery (Figure 2). Postoperatively, he presented with hemorrhagic shock: his pulse rate was 60/min and blood pressure was undetectable. Laboratory data showed a decreased hemoglobin level of 41 g/L. Fluid resuscitation and blood transfusion were all used to restore the intravascular volume. To be worse, he developed ACS: abdominal expansion, abdominal wall tension, oliguria, and high IAP (bladder pressure > 40 mmHg). Exploratory laparotomy, intestinal adhesions lysis and abdominal decompression were performed 13 hours later. 600 mL of blood was aspirated and another 1000 mL of blood from the retroperitoneum was removed. The IAP fell to 19.5 mmHg, followed by diuresis a few hours later. Bogota bag was used for temporary abdominal closure (TAC). However, his condition did not improve after the surgeon, function of multiple organs continued deteriorating (Figure 3). The multiple organ dysfunction syndrome (MODS) score (24 h) was 16 and the acute physiology and chronic health evaluation II (APACHE-II) score (24 h) was 36. Due to the edematous retroperitoneal space during last surgical intervention and deterioration of renal function, the decision was to introduce dehydration of edematous tissue, mainly by increasing of fluid removal of continuous renal replacement therapy (CVVH). Negative fluid balance (-4500 ml) was reached 40 h after initiating fluid removal strategy. IAP decreased from 19.5 to 12 mmHg. 16 days after the operation, a widely colonic perforation was found. Pancolectomy and ileostomy had to be performed and the IAP fell rapidly after the surgeon. Other effective measures were also taken as soon as possible: mechanical ventilation, liver and gastrointestinal tract protection and coagulopathy treatment. Meanwhile, abdominal drainage and sensitive antibiotics continued until his condition improved: temperature fell, laboratory data turned normal. He recovered and was discharged from ICU 10 weeks later.

Discussion

ACS should be a deadly attack to critically ill patients. Aggressive fluid resuscitation after EVAR and a large retroperitoneal hematoma expanding into the abdominal domain may account for the development of ACS. Against the initial idea of aggressive fluid resuscitation in the management of hemorrhagic shock, there is considerable evidence that vigorous fluid replacement may exacerbate bleeding by causing

dilutional and hypothermic coagulopathy associated with infusing large volumes of cold fluids; and secondary clot disruption from increased blood flow, increased perfusion pressure and decreased blood viscosity [5,6]. Massive fluid resuscitation of rAAA also leads to increased intraperitoneal and retroperitoneal volume, visceral edema, abdominal wall edema, and may lead to ACS [7]. In retrospect, if we had performed the limited fluid resuscitation, ACS may have been avoided. Hypotensive resuscitation might have a beneficial effect on the survival in case of rAAA [8]. Experience has shown that systolic arterial pressures of 50–70 mm Hg are well tolerated for short periods and limit internal bleeding and its associated loss of platelets and clotting factors [8].

When this patient had developed the lethal cycle of acidosis, coagulopathy and hypothermia (Figure 3), damage control operation (DCO) was performed to interrupt this cycle, allowing rapid controlled resuscitation in the intensive care unit (ICU). A laparotomy was performed to clinically inspect the abdominal organs, to evacuate the hematoma and decompress the abdominal compartment, and to relieve the pressure from the swollen intestines. Cheatham et al. [7] have demonstrated early abdominal decompression in patients at risk significantly improves survival from IAH/ACS. However, in this case, when the intravesical pressure had been 30 mmHg, damage control laparotomy wasn't performed as soon as possible because of the man's severe shock after EVAR. The hypovolemic shock forced us to dilate blood volume. As a result, intra-abdominal pressure continued rising over 40 mmHg and organ dysfunctions deteriorated 10 hours later. According to international recommendations, when IAP is >20 mmHg and/or ACS develop, and medical treatment is not effective, decompression of the abdomen is necessary and often life-saving. If IAP is >30 mmHg, decompression should not be delayed because there is a risk of acute circulatory collapse [9]. In retrospect, the patient should have benefited more from the early decompression surgery if we had followed the ACS guideline strictly.

After ACS occurred, the fluid strategy aimed at reaching negative fluid balance and avoiding crystalloid over-resuscitation played an important role in treating IAH. Approaches that aimed for neutral and slightly negative fluid balance or 'dry' patients after initial fluid resuscitation were favored. This was achieved by conservative fluid strategies, diuretics and renal replacement therapy in this case. The IAP decreased sharply. Negative fluid balance turned to be effective in decreasing IAP and improving of hemodynamics and ventilation [10]. In the RENAL study, a negative mean daily fluid balance was associated with increased renal replacement therapy (RRT)-free days. The RENAL study also demonstrated that a negative fluid balance in patients requiring RRT was associated with increased survival and shorter ICU and hospital stay [11]. Due to the timely admission of damage control resuscitation protocols, we prevented hemodilution and decreased IAP in this patient successfully.

Intestinal Perforation

In this case, the repeated bowel perforation indicated the serious bowel ischemia after EVAR. Several theories, including interruption of inferior mesenteric arteries or hypogastric arteries, poor remaining collateral networks, ischemia and reperfusion injury, ACS, and macroembolic and microembolic phenomena, have been proposed to explain bowel ischemia after endovascular aortic surgery [12]. The precise etiology remains undefined, yet the most compelling

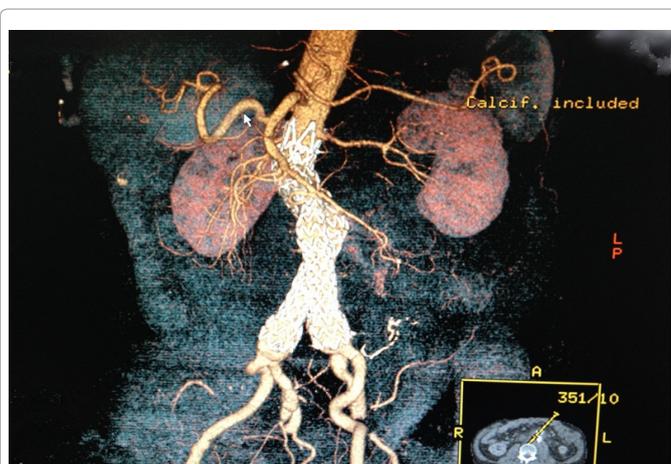


Figure 2: Computed tomography angiography at 2 years post endovascular aneurysm repair showed the successful treatment of ruptured abdominal aortic aneurysm.

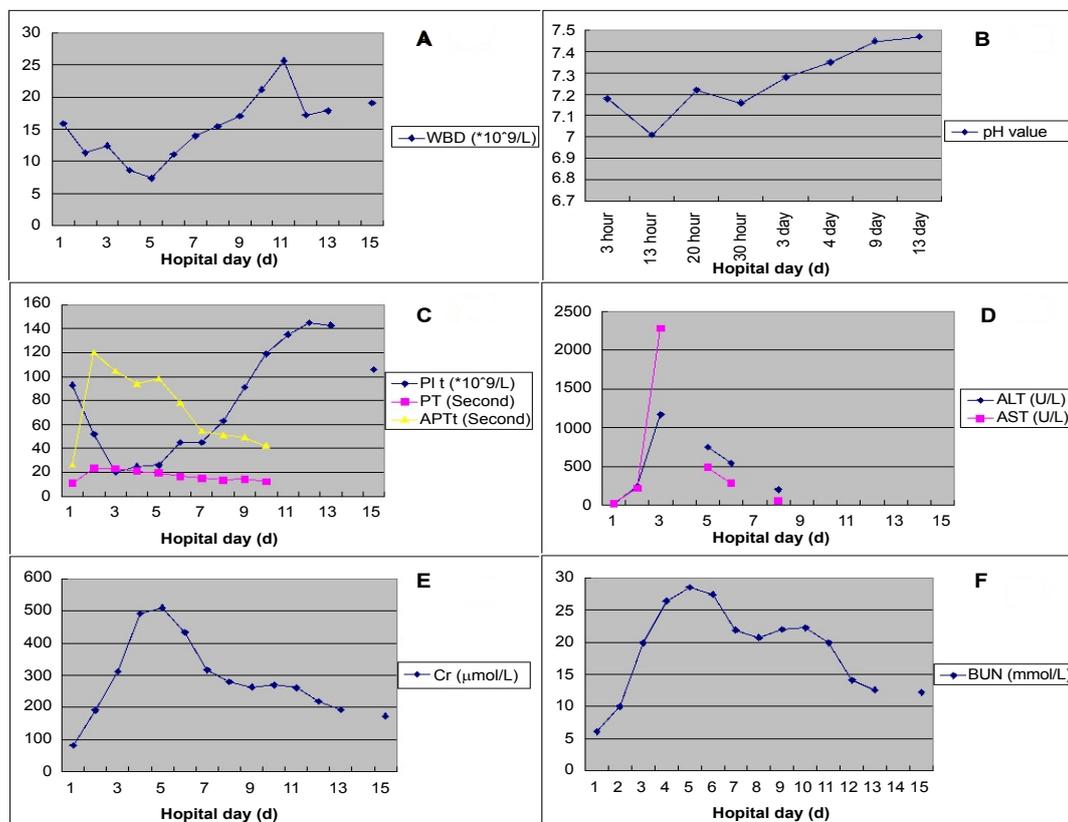


Figure 3: Laboratory reports during the patient's hospitalization. (a) Trends of leukocytes during the hospitalization. (b) Trends of pH value during the hospitalization. (c) Trends of coagulation function during the hospitalization. (d) Trends of liver function during the hospitalization. (e) (f) Trends of renal function during the hospitalization.

evidence exists for the idea of microembolization. The microemboli may be flushed into inferior mesenteric artery, hypogastric artery, and distal vessels, leading to acute colon ischemia [12]. Dadian and colleagues [13] pathologically assessed all resected colon specimens in patients with ischemic colitis after rAAA and found evidence of embolization. Aortic cross-clamping during AAA repair may also cause ischemia-reperfusion injury of the intestine and subsequently result in the translocations of bacteria and fungi across intestinal mucosal barrier, leading to the systemic releases of reactive oxygen species and inflammatory cytokines, which may damage gut again [14,15]. Meanwhile, laparotomy for ACS leaves the underlying alimentary tract exposed and at risk for the development of intestinal perforation as a result of bowel wall edema, serosal injury, and frequent manipulation during temporary abdominal closure (TAC) dressing changes [4]. Bowel mucosa seems to be quite sensitive to elevations of IAP and is associated with: (a) reduction of mesenteric blood flow; (b) compression of mesenteric veins, with subsequent intestinal ischemia and edema; (c) decrease of electrical and mechanical motor activity of the small intestine [16]; (d) inhibition of contractile responses which may lead to structural alterations through an ischemia-reperfusion model [17,18]. Appropriate management of intestinal perforation includes adequate delivery of nutrition, electrolyte/fluid deficit correction, effective control of sepsis and early surgical intervention when possible [4]. In this case, we diagnosed the intestinal perforation and took surgical interventions appropriately and timely. Although pancolectomy had to be performed due to colon perforation, IAP truly decreased and patient's condition turned better after the surgeon. On

the other hand, it also strengthens the necessity of early identifying and controlling IAH.

Conclusion

ACS and intestinal perforation are identified as predictive factors for increased ICU mortality and morbidity, for which reason future vigilance and appropriate management of them remains critically important in rAAA. The lesson to be learned from this case is that DCO and conservative fluid strategy should be taken timely to avoid the subsequently ACS. DCO is conducted to break the reinforcing cycle of hypothermia, coagulopathy, and acidosis after catastrophic intra-abdominal vascular events. Efforts should be made to find a balance between giving sufficient fluid therapy to maintain hemodynamic stability and organ perfusion while avoiding overzealous volume administration. This may be achieved by a neutral or slightly negative fluid balance. It is anticipated that these important concepts need to be even wider application in the future.

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