

Abdominal compartment syndrome following open and endovascular repair of ruptured abdominal aortic aneurysm

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ABSTRACT

Objectives: This study aimed to reveal the incidence, treatment, and outcomes of abdominal compartment syndrome (ACS) following open or endovascular repair of ruptured abdominal aortic aneurysm (rAAA).

Patients and methods: The retrospective study included 36 patients (27 males, 9 females; mean age: 68.9±7.2 years; range, 61 to 81 years) who presented with rAAA between May 2016 and July 2023. In all patients, data regarding demographic characteristics, type of repair (open repair or endovascular aneurysm repair [EVAR]), ACS onset, morbidity, and mortality were recorded. The diagnosis of ACS was made by clinical signs and abdominal pressure measurements.

Results: The overall mortality was 41.7% (n=15). Abdominal compartment syndrome developed in five (13.9%) patients, including two (25%) of eight patients who underwent EVAR and three (10.7%) of 28 patients who underwent open repair. In the open repair group, three (60%) of five patients who developed ACS and 12 (38.7%) of 31 patients without ACS died while one (50%) of two patients who developed ACS died in the EVAR group. No death was noted among patients without ACS in the EVAR group.

Conclusion: This study shows that ACS can develop following both EVAR and open rAAA repair. Decompression laparotomy and open abdominal treatment should not be delayed when indicated. Although intra-abdominal pressure remains high, appropriate therapy may significantly affect outcomes.

Keywords: Abdominal compartment syndrome, EVAR, intra-abdominal hypertension, ruptured abdominal aortic aneurysms.

Ruptured abdominal aortic aneurysm (rAAA) remains to be a major surgical problem due to high surgical mortality and morbidity and need for rapid diagnosis and surgical repair despite contemporary advances. In aortic pathologies, endovascular repair techniques have replaced surgery with technological advances and improved experience.^[1,2] Although endovascular rAAA repair shows promising outcomes regarding survival in selected patients, it is controversial that the technique is associated with favorable outcomes in all patients.^[3,4]

As was the case in elective endovascular interventions, novel complications have been defined with unclear treatment paradigms by improving experience in emergent endovascular repair. Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are known causes of multiorgan failure (MOF) and leading causes of postoperative mortality in rAAA patients undergoing open repair.^[4-6] Abdominal compartment syndrome

development was observed following both open and endovascular rAAA repair.^[7-9] However, unlike open surgery, endovascular repair does not allow evacuation of retroperitoneal hematoma, which theoretically may lead to increased ACS incidence. Intra-abdominal hypertension and ACS resulting from elevated intra-abdominal pressure (IAP) have long been known in patients with acute aortic pathologies.^[10] However, the diagnosis of IAH and ACS is underestimated in many clinics. Although treated sufficiently, the IAH overlooked following open and endovascular rAAA repair can transform into ACS with remarkable

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mortality rate due to irreversible pathophysiological changes causing MOF.^[10]

The term IAP refers to pressure in the intra-abdominal cavity. Reference values are defined following intermittent measurements through the bladder. The measurement is performed during expiration at supine position with the abdominal muscles relaxed. It was reported to be 0-5 mmHg in healthy individuals.^[11] The mean IAP was reported as 5-7 mmHg in critically ill patients.^[12]

Intra-abdominal hypertension is a term used for IAP that continuously or recurrently exceeds 12 mmHg in pathological conditions.^[11,12] Four grades have been defined based on pressure level (Table 1).^[11] Abdominal perfusion pressure is a relative marker of abdominal blood flow, which is estimated using the following formula: abdominal perfusion pressure = mean arterial pressure-IAP.

Intra-abdominal hypertension is often associated with bleeding or splanchnic reperfusion-related massive fluid resuscitation, resulting in profound physiological derangement affecting each organ system in either a direct or indirect manner, and can also lead ACS.^[12-15] Vena cava inferior is stressed by IAP elevation, which results in decreased venous return, leading to systemic vascular resistance through decrease in end-diastolic ventricular volume, stroke volume, and cardiac output. In addition, IAP elevation also affects kidneys, leading to decreased renal flow and urine output together with unfavorable effects on cardiac function. Simultaneously, the elevated IAP compresses the diaphragm and leads to increased intrathoracic pressure by elevating the airway pressure, pulmonary artery pressure, and central venous pressure and decreasing pulmonary compliance. Individual organ perfusion dysfunction occurs at a different level of IAP. For instance, renal

blood flow and renal function will manifest as oliguria at an IAP level of 15 mm and anuria at 30 mmHg. If ACS is left untreated, MOF will develop. All pathophysiological changes are potentially reversible without severe organ dysfunction if IAH is diagnosed early and treated adequately.^[7-10] The management often requires decompression laparotomy and aims at reducing IAP. Failure to relieve pressure is almost always fatal and surgical decompression provides significant improvement in mortality.^[7]

In the literature, there is limited data regarding ACS development following open or endovascular rAAA repair and its effects on morbidity and mortality. Hence, this study aimed to reveal the incidence, treatment, and outcomes of ACS following open or endovascular repair of rAAA.

PATIENTS AND METHODS

This retrospective study examined 36 patients (27 males, 9 females; mean age: 68.9±7.2 years; range, 61 to 81 years) who underwent surgery with a diagnosis of rAAA in the Izmir Bakırçay University Faculty of Medicine between May 2016 and July 2023. Data regarding demographic characteristics, type of repair (open repair or endovascular aneurysm repair [EVAR]), and comorbid conditions were extracted from patient files. The clinical suspicion and close monitorization of IAP are keys for diagnosis of IAH/ACS. The diagnosis of ACS was made a series of clinical finding including increased airway pressure, impairment in respiratory parameters, oliguria, cerebral dysfunction, intestinal dysfunction, and abdominal wall tension, together with the measurement of IAP. A urine catheter was inserted into the bladder to monitor IAP. A sterile, disposable, noninvasive pressure monitorization kit (UnoMeter Abdo-Pressure; Convatec, London, UK) was attached to the catheter. Intra-abdominal pressure measurements were performed using a specific probe, and mean values were recorded. The outcome and discharge data were also recorded.

Statistical analysis

Statistical analyses were performed using IBM SPSS version 19.0 (IBM Corp., Armonk, NY, USA). Comparisons were made by the unpaired t-test. Categorical data was compared with Fisher exact test or the Mann-Whitney U test. A *p*-value <0.05 was considered statistically significant.

Table 1

Stages of intra-abdominal hypertension

Grade	IAP (mmHg)
1	12-15
2	16-20
3	21-25
4	≥25

IAP: Intra-abdominal pressure.

Table 2
Patient characteristics and patient outcomes

	EVAR (n=8)				Open (n=28)				<i>p</i>
	n	%	Mean	Median	n	%	Mean	Median	
Patient characteristics									
Age (year)			72.9				67.9		0.4
Sex									NS
Male	7	88			20	71			
Coronary artery disease	3	38			11	39			NS
COPD	3	38			9	32			NS
Diabetes mellitus	1	13			7	25			NS
Hypertension	4	50			16	57			NS
Chronic renal failure	0	0			2	7			NS
Outcome									
Abdominal compartment syndrome	2	25			3	11			NS
Myocardial infarction	0	0			2	7			NS
Pneumonia	1	13			7	25			NS
Renal failure	4	50			17	60			NS
Dialysis	1	13			5	18			0.09
Sepsis	1	13			4	14			NS
Ischemic colon	0	0			2	7			0.079
Multisystem organ failure	1	13			4	14			0.145
Death	2	25			13	47			0.146
Abdominal compartment syndrome/death	1	13			2	7			NS
Hospital length of stay (days)				8				15	0.215
Intensive care unit length of stay (days)				4				8	0.052

EVAR: Endovascular aneurysm repair; COPD: Chronic obstructive pulmonary disease; NS: Not significant.

RESULTS

The overall mortality was 41.7% (n=15). Abdominal compartment syndrome developed in five (13.9%) patients. Ruptured abdominal aortic aneurysm repair was performed using an endograft in eight (22.2%) patients, while open repair was performed in 28 (77.8%) patients. Patient characteristics were comparable in endovascular repair and open repair groups (Table 2). There was a tendency towards lower mortality, shorter length of hospital stay and ICU stay in endovascular repair group when compared; however, the difference did not reach statistical significance.

Abdominal compartment syndrome developed in five (13.9%) patients, including two (25%) of eight patients who underwent endovascular repair and three (10.7%) of 28 patients who underwent

open repair. No difference was observed in patient characteristics between patients with or without ACS in the endovascular repair and open repair groups. In the open repair group, three (60%) of five patients who developed ACS and 12 (38.7%) of 31 patients without ACS died, while one (50%) of two patients who developed ACS died in the endovascular repair group. No death was noted among six patients without ACS in the endovascular repair group (Table 2).

DISCUSSION

The overall mortality rate of 41.7% in our study on patients who underwent rAAA repair was consistent with those reported in the literature.^[4,7,10,16,17] Endovascular therapy in aortic aneurysms is preferred due to better early outcomes compared to open

surgery. In rAAA, mortality and morbidity were found as 24 and 44% after endovascular repair, respectively.^[18] In agreement with the literature, albeit not significant, there was a tendency towards lower mortality in patients treated with endovascular graft when compared to those who underwent open surgery in our study.^[16-20]

Abdominal compartment syndrome may develop both in patients treated with endograft repair and

open repair. In our study, ACS was observed in both groups. The incidence of ACS is unclear; however, it was reported to develop in 10% of patients.^[21] In a systematic review, it was reported that ACS developed in 5.5% of patients treated with endovascular repair. In a recent meta-analysis, it was estimated as 8%; however, authors proposed that actual incidence may be >20% with awareness and close monitoring.^[4] In our series, ACS rate was 25% in patients treated with

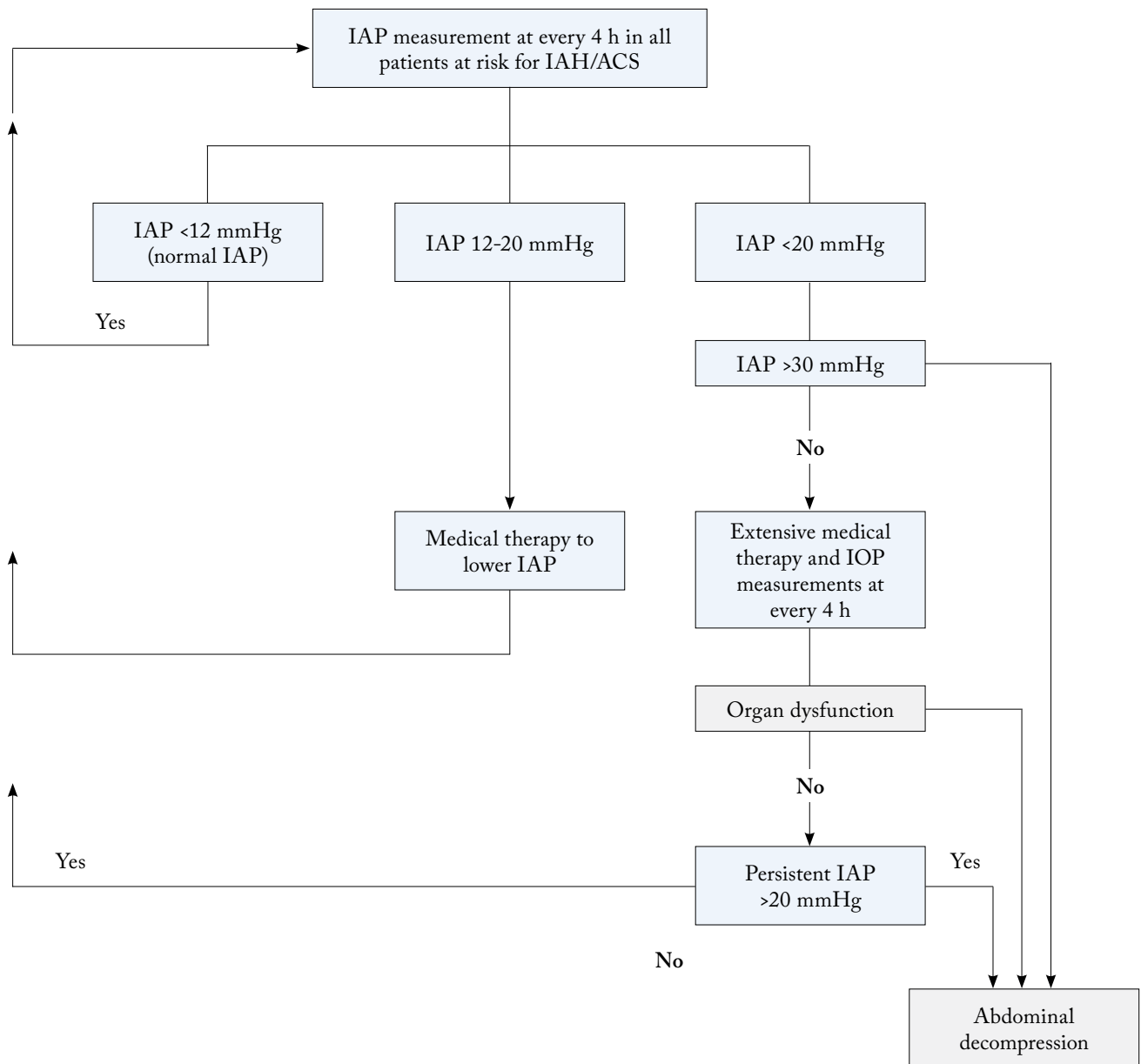


Figure 1. Algorithm for IAP monitorization and postoperative follow-up after repair of ruptured abdominal aortic aneurysm.^[28] IAP: Intraabdominal pressure; IAH: Intraabdominal hypertension; ACS: Abdominal compartment syndrome.

endovascular repair, whereas it was 10.7% in patients treated with open repair.

Following open repair, it has been suggested that the ACS-related mortality rate is generally over 50%, reaching up to 100%.^[22,23] In our study, ACS-related mortality was 60%. In previous studies, mortality rate up to 57% was reported in patients who developed ACS following endovascular repair.^[24] In agreement with the literature, mortality rate was found as 50% in patients who developed ACS following endovascular repair.

The mechanism underlying ACS development following endovascular or open repair of rAAA is not limited to a single mechanism and may be multifactorial in certain patients. There are several factors that may be involved in ACS development. For instance, patients who underwent endovascular repair may need substantial fluid after surgery due to effects of shock, while mass effect may be present due to retroperitoneal hematoma, or coagulopathy may exist. Similarly, patients who underwent open repair may require transfusion of blood and blood products due to coagulopathy or surgical bleeding in addition to retroperitoneal mass effect and intestinal edema caused by resuscitation. Blood transfusion need at an early phase after endovascular repair may be suggestive of ongoing bleeding related to surgical type 2 endoleak. This may warrant opening the aneurysmal sac. Early onset of ACS following open repair may result from ongoing hemorrhage due to coagulopathy or surgical bleeding. All efforts should be made to correct coagulopathy and achieve normothermia before decompression laparotomy.^[7]

Abdominal compartment syndrome can lead to progressive organ dysfunction and even death if not diagnosed early and treated appropriately.^[1,4,7,10,25] Thus, early diagnosis and treatment are of importance. It is widely accepted that treatment should include IAP reduction and abdominal decompression in patients with persistent IAP elevation above 20-25 mmHg.^[10,26-28] In our series, IAP measurements were performed periodically, and laparotomy was performed for abdominal decompression when IAP was >20 mmHg (Figure 1).

Intra-abdominal hypertension and ACS are severe complications with high incidence following rAAA repair. Abdominal decompression is deemed a life-saving intervention in patients who develop ACS. In the case of ACS, a successful outcome depends

on the early recognition of ACS, medical therapy directing to lower IAP, and decompression laparotomy at an early phase.^[28]

This study has some limitations. First, significant changes may have occurred in diagnostic and therapeutic procedures due to differences in healthcare providers and approaches over the lengthy duration of the study. Second, this is a retrospective, nonrandomized study with a relatively limited number of patients. In addition, there was no prespecified standard for IAP measurement or ACS indication.

In conclusion, intra-abdominal hypertension and ACS are commonly seen in patients treated for rAAA and are associated with a high risk for morbidity and mortality. However, IAH/ACS are overlooked by many clinicians; in addition, the diagnosis is generally delayed, and treatment often fails. All healthcare providers involved in the treatment of rAAA via open surgery or endovascular repair should understand the pathophysiology, risk factors, and presentation. This study shows that ACS can develop following both endovascular repair and open rAAA repair. Early decompression laparotomy should be performed in patients with ACS at an early phase after endovascular repair and signs suggestive of ongoing bleeding. Nonsurgical and surgical treatment, as well as a timely diagnosis, are of importance. Decompression laparotomy and open abdominal treatment should not be delayed when indicated. Although IAP remains high, appropriate therapy may significantly affect outcomes.

Ethics Committee Approval: The study protocol was approved by the Izmir Bakırçay University Non-Invasive Ethics Committee (date: 13.09.2023, no: 1192/1172). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Patient Consent for Publication: A written informed consent was obtained from each patient.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Idea, design, data collection, literature review, critical review, writing the article: İ.K.; Control, literature review, references and fundings, data collection, materials: A.D.

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