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Editor's Choice – Abdominal Compartment Syndrome after Surgery for Abdominal Aortic Aneurysm: Subgroups, Risk Factors, and Outcome

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WHAT THIS PAPER ADDS

This is the largest study to date on abdominal compartment syndrome (ACS) after abdominal aortic aneurysm (AAA) repair, including intact and ruptured AAAs. ACS subgroup outcomes and the impact of duration of intra-abdominal hypertension (IAH) were investigated. Outcome was poor regardless of whether ACS was associated with post-operative bleeding, bowel ischaemia, or oedema, and regardless of timing of decompression. The duration of IAH before treatment predicted the need for renal replacement therapy. This emphasises the need to focus on prevention, through careful monitoring of intra-abdominal pressure, strategies for pre-emptive treatment of IAH, and swift treatment when ACS develops.

Objectives: Abdominal compartment syndrome (ACS) is a serious complication after abdominal aortic aneurysm (AAA) repair. The aim was to investigate outcome among subgroups and factors associated with outcome, with emphasis on the duration of intra-abdominal hypertension before treatment.

Methods: Since 2008, ACS and decompressive laparotomy (DL) after AAA repair are registered prospectively in the Swedish vascular registry (Swedvasc). Registry data and case records were reviewed. Subgroups were defined by main pathophysiological finding at DL, timing of DL after AAA repair, and treatment modality.

Results: During 2008–2015, 120 of 8765 patients undergoing surgery for infrarenal AAA developed post-operative ACS (1.4%). Eighty-three followed ruptured AAA (rAAA); 45 open surgical repairs (OSR) and 38 endovascular (EVAR), and thirty-seven after intact AAA (iAAA); 30 OSR and seven EVAR. The main pathophysiological findings at DL were bowel ischaemia in 27 (23.3%), post-operative bleeding in 34 (29.3%), and general oedema in 55 (47.4%). DL was performed <24 hours after AAA repair in 56 (48.7%), 24–48 hours in 30 (26.1%), and >48 hours in 29 patients (25.2%). The overall 90 day mortality was 50.0%, neither different depending on main pathophysiological finding, nor on the timing of DL. In multivariable regression analysis, age was a predictor of mortality ($p = .017$), while duration of intra-abdominal hypertension (IAH) prior to DL predicted the need for renal replacement therapy (RRT) ($p = .033$). DL was performed earlier after EVAR compared with OSR in rAAA ($p < .001$).

Conclusions: Mortality in ACS was high, irrespective of the main pathophysiological finding and timing of DL. The duration of IAH prior to DL predicted the need for RRT. DL was performed earlier after EVAR than after OSR for rAAA, underlining the importance of monitoring IAP after EVAR for rAAA.

Keywords: Abdominal compartment syndrome, Aortic aneurysm-abdominal, Bowel ischaemia, Intra-abdominal pressure, Mortality, Renal replacement therapy

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INTRODUCTION

Abdominal compartment syndrome (ACS) is a serious and often lethal complication after surgery for abdominal aortic aneurysm (AAA).[1,2] Many studies have demonstrated a poor outcome for these patients despite treatment.[3–8] ACS is not defined as a mere pressure, but as the combination of an intra-abdominal pressure (IAP) ≥ 20 mmHg and new onset of organ dysfunction or failure.[2] Decompressive laparotomy (DL) remains the standard therapy for fully developed ACS; however, in patients with less pronounced intra-abdominal hypertension (IAH), medical (i.e. non-surgical) management is first choice,[2,9] with the aim of preventing ACS and the need for open abdomen treatment (OAT). Thus the contemporary focus is on the prevention of ACS, reducing

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IAP, and targeted organ support. Massive transfusion protocols, balanced resuscitation, and optimisation of coagulation help limiting fluid overload.[10]

The incidence of ACS after ruptured abdominal aortic aneurysm (rAAA) repair is reported at between 4% and 34%.[3,5–7,11,12] Among those affected, 30 day mortality is between 30% and 70%.[3,5–7,13] After intact abdominal aortic aneurysm (iAAA) repair, less than 1%[3,14] develop ACS, with mortality at 30 days approximately 12%, and nearly twice as high at 90 days.[3]

Although the incidence and mortality rate in patients with ACS after aortic surgery has been reported in a previous study by the same authors,[3] as well as by others,[5–8,11–13] there are no previous data on potential differences between subgroups of patients. ACS may develop as a result of, or in association with, bowel ischaemia, post-operative bleeding, and/or oedema. ACS may also develop at different time intervals after surgery: early, within 24 hours, between 24 and 48 hours, or later. The present study hypothesises that these subgroups may have different outcomes.

The World Society of the Abdominal Compartment Syndrome (WSACS) guidelines recommend early decompression for fully developed ACS.[2] It is possible that the duration of intra-abdominal pressures (IAP) exceeding 20 mmHg before DL, the cut off for ACS diagnosis, or even 15 mmHg, a more moderate level of IAH, will affect outcome.

The aims of this population based study were to study the outcome and prognostic factors for ACS and OAT after AAA repair, with special focus on the importance of the underlying main pathophysiological finding, the timing of DL, and the duration of IAH before decompression.

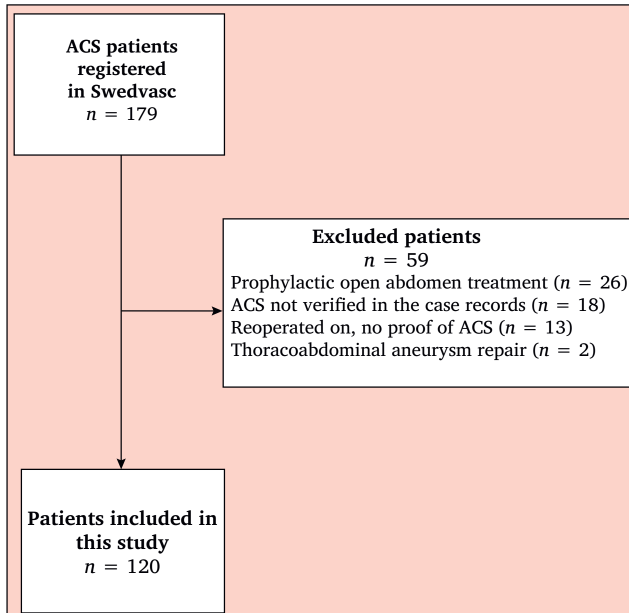

Figure 1. Study population for aortic compartment syndrome (ACS) after surgery for abdominal aortic aneurysm.

MATERIALS AND METHODS

The Swedish national vascular registry, Swedvasc, has prospectively registered ACS since May 2008. All AAA repairs between May 2008 and September 2015 were examined. Patients registered for ACS were identified and their case records were reviewed, with subsequent inclusion if the following exclusion criteria were not met; the case records could not verify the diagnosis of ACS, aortic repairs were other than infra- and juxtarenal AAA, or prophylactic open abdomen treatment was performed (Fig. 1).

Compared with the previous publication,[3] which used solely registry data, and a validation of those data through a short one page questionnaire, this investigation scrutinised the case records in all registered patients. The study period was also prolonged for a further two years, to get more contemporary data and to include more patients who developed ACS after endovascular aneurysm repair (EVAR).

In Sweden, all centres performing AAA surgery report to the Swedvasc registry, ensuring national coverage. Several studies have validated Swedvasc,[15,16] with a consistent internal validity of >90%. The latest international independent validation regarding AAA surgery showed an external validity of 99.5%.[17] Thus, very few patients are not registered after AAA repair. However, the internal validity of registering ACS and OA treatment was not as good (see more in Results and the Discussion). This, together with the fact that complete case records add a wealth of more detailed data, explains the importance of performing this second investigation.[3]

The definition of ACS was made according to the consensus definitions and clinical practice guidelines of the Abdominal Compartment Society;[2] a sustained intra-abdominal pressure ≥20 mmHg, in combination with new onset organ dysfunction or failure.

The subgroups were defined and are listed as follows: the main pathophysiological finding at DL (bowel ischaemia, post-operative bleeding, or oedema), timing of DL (early: within 24 hours, intermediate: between 24 and 48 hours, and late: after 48 hours), and treatment modality (open surgical repair [OSR] or EVAR). Although all patients operated on for AAA exhibit some degree of visceral and abdominal wall oedema, those who had bowel ischaemia or post-operative bleeding as the main pathophysiological finding at DL were grouped as such, with the remainder grouped as oedema. Bowel ischaemia can both be a cause and an effect of IAH, each potentiating the other.[18] Bowel ischaemia is aggravated by an increasing IAP, and a gangrenous bowel results in inflammation, visceral oedema, and an increased IAP.

Ninety day survivors and non-survivors were compared in terms of risk factors influencing outcome. The subgroups were analysed for outcome, with analyses relating to treatment modality performed separately for intact and ruptured AAA repairs. The subgroups were also analysed for their respective DL timing. Mortality was analysed at 30 days, 90 days, and one year. Morbidity was analysed with respect to rate of renal replacement therapy (RRT), duration of RRT, duration of mechanical ventilation, and duration of OAT.

Table 1. **Clinical characteristics of study population**

	All patients *n* = 120	90 day survivors *n* = 60	90 day non-survivors *n* = 60	*p* value[a]	Ruptured AAA *n* = 83	Intact AAA *n* = 37	*p* value[b]
Age – y	74 (67–79)	71 (67–75)	78 (71–80)	<.001	75 (68–80)	69 (66–76)	.008
Female sex	23 (19.2)	8 (13.3)	15 (25.0)	.16	17 (20.5)	6 (16.2)	.80
Aortic diameter – mm	70 (60–80)	65 (56–80)	70 (61–80)	.045	76 (70–85)	57 (55–65)	<.001
Comorbidities							
Cardiac disease	45 (37.5)	23 (38.3)	22 (36.7)	1.0	29 (34.9)	16 (43.2)	.42
Pulmonary disease	30 (25.2)	14 (23.7)	16 (26.7)	.83	20 (24.4)	10 (27.0)	.82
Diabetes	14 (11.7)	4 (6.7)	10 (16.7)	.15	9 (10.8)	5 (13.5)	.76
Previous CVE	12 (10.1)	6 (10.2)	6 (10.0)	1.0	6 (7.3)	6 (16.2)	.19
Ruptured AAA	83 (69.2)	34 (56.7)	49 (81.7)	.005	–	–	–
EVAR	45 (37.5)	18 (30.0)	27 (45.0)	.13	38 (45.8)	7 (18.9)	.007
Open abdomen	107 (89.1)	58 (96.7)	49 (81.7)	.016	73 (88.0)	34 (91.9)	.75
Main pathophysiological finding							
Bowel ischaemia	27 (23.3)	13 (22.0)	14 (24.6)	.83	17 (21.5)	10 (27.0)	.64
Bleeding	34 (28.3)	15 (25.4)	19 (33.3)	.42	23 (29.1)	11 (29.7)	1.0
Oedema	55 (46.4)	31 (52.5)	24 (42.1)	.27	39 (49.4)	16 (43.2)	.56
Duration of IAP							
Duration IAP ≥ 20 mmHg – hours	2.0 (0–8.0)	1.0 (0–7.0)	3.0 (0–9.0)	.39	2.0 (0–9.0)	1.0 (0–6.0)	.27
Duration IAP ≥ 15 mmHg – hours	8.5 (2.0–24.0)	7.0 (2.0–23.0)	13.0 (1.0–26.5)	.50	8.0 (1.0–25.0)	9.0 (3.0–23.0)	.68
Timing of DL from AAA surgery – hours	27.9 (7.3–48.3)	29.1 (7.5–56.0)	23.6 (4.7–45.6)	.39	14.1 (2.8–39.8)	45.0 (27.1–75.0)	<.001

Data are given as *n* (%) or median (interquartile range). AAA = abdominal aortic aneurysm; CVE = cerebrovascular event; DL = decompressive laparotomy; EVAR = endovascular aneurysm repair; IAP = intra-abdominal pressure.
[a] *p* values refer to comparison between 90 day survivors and non-survivors.
[b] *p* values refer to comparison between ruptured AAA and intact AAA.

Swedvasc is linked to the national population registry through cross matching with the help of personal identity numbers used in both registries. Thus, mortality data are 100% correct.

One patient who underwent conversion from EVAR to OSR was grouped within the EVAR group, on the basis of intention to treat. Five patients were found to have both post-operative bleeding and bowel ischaemia at DL. Based on the data in the case records, however, the main pathophysiological finding responsible was considered to be bleeding in all those cases.

Patients referred from other hospitals were not included in the analysis of the time from onset of symptoms to arrival at hospital, and from arrival at hospital to start of surgery, because of uncertain or missing information regarding the elapsed time in those cases.

Post-operative transfusions were analysed from the end of surgery until 24 hours (post-operative day 1), and from 24 to 48 hours (day 2). Patients who did not survive the entire time period of 24 or 48 hours were excluded from these analyses. The ratios of packed red blood cells (pRBC) to fresh frozen plasma (FFP), pRBC:FFP, and pRBC to platelets, pRBC:Platelets, excluded patients who did not receive both components, as those resulted in division by zero. Platelets are described multiplied by four, as one unit in Sweden is derived from four donors.

Estimated hourly IAP, between two existing IAP measurements, was calculated using linear interpolation. The last IAP value recorded before decompressive laparotomy served as the value for the remaining hours until decompression.

To adjust for competing risk of death, analyses related to RRT excluded those who died within 48 hours after decompressive laparotomy. Duration of RRT, duration of OAT, and duration of mechanical ventilation were analysed among survivors at 90 days. Patients who needed continuous RRT, and who were later transferred to intermittent RRT, were defined as having received RRT for 90 days.

The duration of IAH was analysed using two levels of IAP; IAP ≥15 mmHg and IAP ≥20 mmHg.

Simplified Acute Physiology Score (SAPS) III data, an intensive care unit (ICU) scoring system predicting mortality,[19] was obtained from the Swedish intensive care registry (SIR).[20] Swedish ICUs prospectively enter SAPS III data in the registry and the registered values are entered within 24 hours from arrival to the ICU.

Statistical analysis

Continuous variables are expressed as median (interquartile range [IQR]). For comparison of proportions, X^2 or Fisher's exact test were used where appropriate. Comparison of continuous variables were undertaken using the Mann–Whitney *U* test for groups of two, and Kruskal–Wallis test for groups of three. Multivariable logistic regression by forced entry and Kaplan–Meier curves were used for

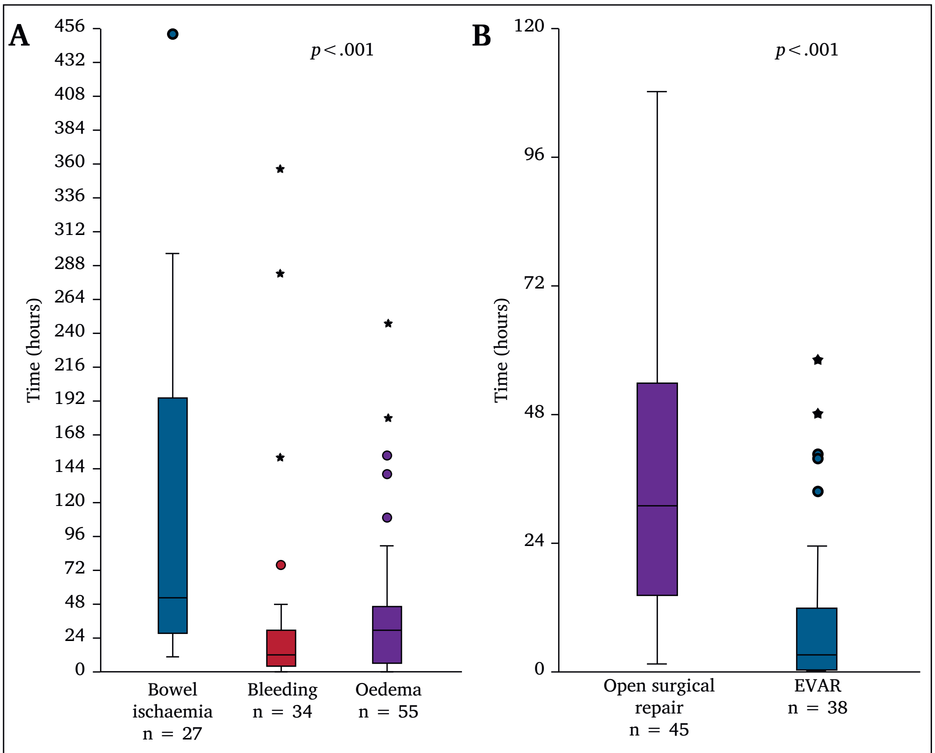

Figure 2. Interval from abdominal aortic aneurysm (AAA) repair to decompressive laparotomy depending on the (A) main pathophysiological finding and (B) treatment modality in ruptured AAA. Dots represent suspected outliers (≥1.5 interquartile range [IQR]) and stars represent outliers (≥3.0 IQR). EVAR = endovascular aneurysm repair.

outcome and survival analysis. Those who developed ACS as a result of post-operative bleeding, where the bleeding could not be controlled, were excluded from the multivariable regression analyses. The threshold for significance was set to $p < .05$, with all tests being two sided. SPSS Statistics version 22 (IBM, Armonk, NY, USA) was used for statistical analyses.

Ethics

The study was approved by the ethical committee of the Uppsala region. No informed consent was required from patients or relatives, for this retrospective review.

RESULTS

Among all 8765 AAA repairs entered in the Swedvasc registry between May 2008 and September 2015, 179 patients registered for having ACS were identified and their case records were reviewed. The study population originated from 24 different vascular centres and is shown, with exclusions, in Fig. 1. Prophylactic OAT was the most common exclusion (26/59, 44%). Seven patients with wound dehiscence received OAT. Although IAH may have contributed to wound dehiscence, the case records stated poor fascial edges as the reason for initiation of OAT.

All survival data refer to 90 day mortality unless otherwise stated.

After exclusions, 120 patients with verified ACS were included in the study. Among 1718 repairs for rAAA and 7009 repairs for iAAA, the risks of ACS were 45/1212 (3.7%) after OSR for rAAA, 38/506 (7.5%) after EVAR for rAAA, 30/2859 (1.0%) after OSR for iAAA, and 7/4150 (0.2%) after EVAR for iAAA. The clinical characteristics of the study population are shown in Table 1.

Pre-operative characteristics

Patients having rAAA repair were analysed with regard to time interval from onset of symptoms to arrival in hospital, time interval from arrival in hospital to start of surgery, the lowest recorded pre-operative blood pressure, presence of pre-operative unconsciousness, and presence of pre-operative asystole. There were no differences between survivors and non-survivors (Table S1).

Intra-operative characteristics

Non-survivors had a higher rate of suprarenal aortic control (clamping or balloon occlusion) than survivors and received more intra-operative transfusions of pRBC, FFP, and platelets. Post-operatively, no transfusion differences were found between survivors and non-survivors, and the ratios of pRBC to FFP and pRBC to platelets were below 2:1 both intra- and post-operatively (Table S1).

ACS characteristics

Survivors and non-survivors did not differ regarding duration of IAP ≥15 or ≥20 mmHg before DL, maximum IAP, IAP after DL, or timing of DL. The interval from the end of primary surgery to maximum IAP was shorter among non-survivors compared with survivors (10 [IQR: 3—29] vs. 21 [9—42] hours; $p = .047$).

Timing of ACS and subgroups

DL was performed ≤24 hours in 56/115 (48.7%), 24—48 hours in 30 (26.1%), and >48 hours in 29 (25.2%).

The three main pathophysiological findings at DL were bowel ischaemia in 27/116 (23.3%), post-operative bleeding in 34 (29.3%), and oedema in 55 (47.4%). The proportions were similar in iAAA and rAAA repair. The timing of DL differed depending on the underlying main pathophysiological finding: post-operative bleeding median 11.4 hours (IQR: 2.8—29.3), oedema 29.2 hours (5.0—46.2), and bowel ischaemia 51.9 hours (27.5—194.2) ($p < .001$, Fig. 2A).

DL was performed earlier after rAAA repair compared with iAAA repair ($p < .001$). Among those with rAAA, DL was performed earlier after EVAR compared with OSR (median 2.8 hours [IQR: 0—12.0] vs. 30.8 hours [14.1—58.2]; $p < .001$, Fig. 2B).

ACS after iAAA repair

In EVAR, operative time was 233 (IQR: 180—345) min, operative bleeding 0.8 (0.4—3.0) L, one patient (14.3%) received suprarenal control with an aortic balloon and no patients were re-operated on prior to developing ACS. The main finding at DL was bowel ischaemia in five patients, three of whom experienced bleeding complications during the EVAR, and one who had the only patent internal iliac artery embolised. Bleeding was found in two patients, which in both was persistent bleeding from missed arterial perforation during EVAR.

In OSR, operating time was 390 (IQR: 300—510) min, operative bleeding 4.5 (2.7—9.0) L, 14 (46.7%) had suprarenal control during surgery, and nine (30%) were re-operated on prior to developing ACS. The main pathophysiological finding at DL was bowel ischaemia in five (16.7%), bleeding in nine (30.0%), and oedema in 16 (53.3%).

Outcome

Mortality did not differ depending on main pathophysiological finding or timing of DL (Figs. 3 and 4). Mortality was higher with EVAR compared with OSR after iAAA repair (Table 2).

The rate of RRT, the durations of RRT, OAT, or mechanical ventilation did not differ between any of the subgroups. Ninety day survivors spent a median of nine (IQR: 0—23) days on RRT, 13 (5—24) on mechanical ventilation, and 14.5 (8—27) with OAT.

SAPS3 estimated mortality rate score was higher in non-survivors compared with survivors (0.39 [IQR: 0.13—0.55] *vs.* 0.18 [0.05—0.36]; $p = .008$), and in those treated with EVAR compared with OSR in iAAA (0.20 [IQR: 0.12—0.24] *vs.* 0.05 [0.02—0.08]; $p = .002$).

Multivariable analysis

Age, ruptured/intact AAA, treatment by OSR or EVAR, intra-operative pRBC transfusions, suprarenal aortic control, main pathophysiological finding (post-operative bleeding, bowel ischaemia or oedema), and duration of IAP ≥20 mmHg prior to DL, were entered into two multivariable logistic regression models with mortality and need for RRT as dependent variables (Table 3). Only age remained an independent predictor for mortality. Duration of IAP ≥20 mmHg was an independent predictor for RRT, whereas rAAA repair and ACS from post-operative bleeding were independent negative predictors. In an otherwise identical regression model, but in which duration of IAP ≥20 mmHg was exchanged for ≥15 mmHg, the latter was also an independent predictor for RRT (OR 1.03, [95% CI 1.00—1.07]; $p = .043$).

Treatment of ACS

Neuromuscular blockade was applied as first line treatment in five (4.2%) patients and local thrombolytic therapy of the retroperitoneal haematoma in two (1.7%) patients.[21] They all received decompressive laparotomy at a later stage, however.

Of 120 patients in whom DL was planned, three deteriorated and were palliated, and another seven had findings during DL that were not compatible with life. Of the remaining 110, 107 (97.3%) received OAT, another three (2.7%) had enough intra-abdominal space after measures during decompressive laparotomy to permit closure.

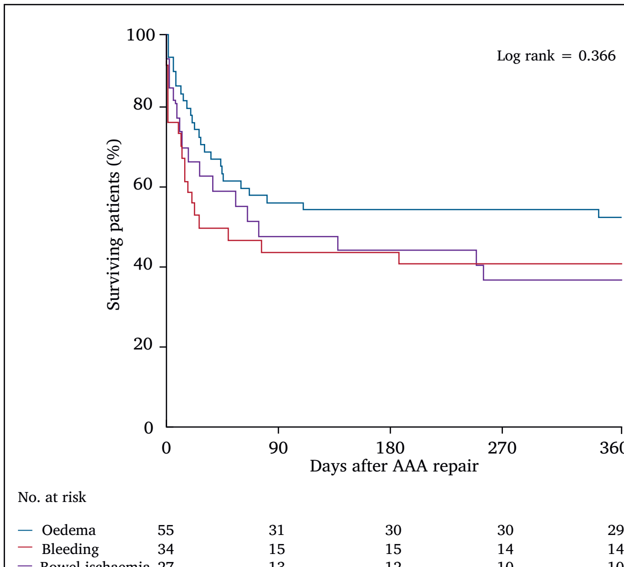

Figure 3. Survival with abdominal compartment syndrome after surgery for abdominal aortic aneurysm (AAA) repair depending on the main pathophysiological finding.

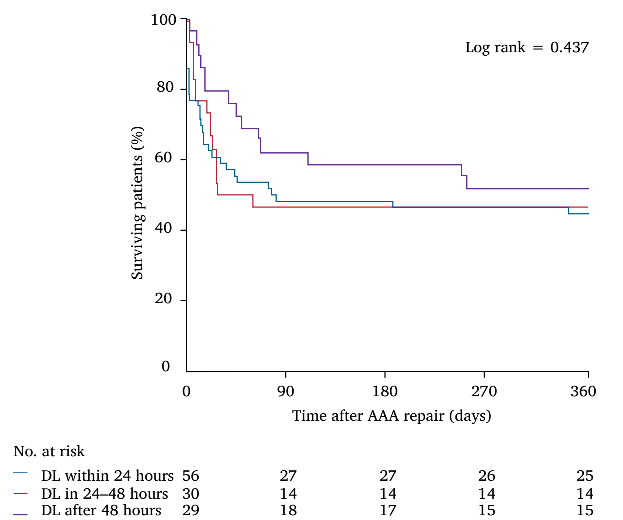

Figure 4. Survival with abdominal compartment syndrome depending on the timing of decompressive laparotomy (DL). AAA = abdominal aortic aneurysm.

OAT and fascial closure

Ninety-nine of 107 (92.5%) had negative pressure wound therapy (NPWT), most combined with mesh mediated traction.[22–24] Six received a "Bogotá bag"[25] and two a dual layer mesh. Among 85 patients with NPWT who survived until abdominal closure, 75 (88.2%) had primary delayed fascial closure, five (5.9%) fascial closure with an onlay mesh, and one (1.2%) with abdominal wall component separation. In total, 81 (95.3%) had fascial closure.

Five (4.7%) had complications of OAT: two entero-atmospheric fistulae (bowel to open abdomen wound), one recurrent bleeding from the abdominal wound, and two sub fascial abscesses after closure. Both patients with fistulae and one patient with sub fascial abscess died within one year.

DISCUSSION

This study found poor outcomes in all subgroups, with no differences in mortality or morbidity rates depending on the main pathophysiological finding at DL, the timing of DL, or whether primary treatment in rAAA was by OSR or EVAR.

Non-survivors had a higher rate of temporary suprarenal aortic control and received more intra-operative pRBC and FFP than survivors, indicating more unstable haemodynamics. However none of these variables remained independent predictors of mortality in multivariable analyses, where the only independent predictor was age. Age was also associated with mortality in two papers reporting on DL for ACS[4] and patients treated with OA.[24]

Table 2. Outcome with abdominal compartment syndrome depending on ruptured or intact abdominal aortic aneurysm (AAA) and open surgical (OSR) or endovascular (EVAR) repair.

Outcome	Ruptured AAA			Intact AAA		
	OSR $n = 45$	EVAR $n = 38$	p value[a]	OSR $n = 30$	EVAR $n = 7$	p value[b]
30 day mortality	20 (51.3)	19 (48.7)	.66	6 (20.0)	1 (14.3)	1.0
90 day mortality	25 (55.6)	24 (64.9)	.51	8 (26.7)	3 (42.9)	.40
1 y mortality	25 (55.6)	26 (70.3)	.26	9 (30.0)	6 (85.7)	.011
RRT	23 (63.9)	15 (57.7)	.79	19 (65.5)	4 (66.7)	1.0

Data are given as n (%). AAA = abdominal aortic aneurysm; EVAR = endovascular aneurysm repair; OSR = open surgical repair; RRT = renal replacement therapy.

a p values refer to comparison between OSR and EVAR among ruptured AAA.

b p values refer to comparison between OSR and EVAR among intact AAA.

Table 3. Multivariable logistic regression for mortality and need of renal replacement therapy (RRT) after open surgical or endovascular (EVAR) repair of ruptured or intact abdominal aortic aneurysm.

Predictor	1 y mortality		Need for RRT	
	OR (95% CI)	p value[a]	OR (95% CI)	p value[b]
Age – y	1.09 (1.02–1.17)	.017	1.04 (0.97–1.13)	.27
Rupture	1.48 (0.49–4.52)	.49	0.20 (0.05–0.71)	.014
EVAR vs. open surgical repair	2.48 (0.81–7.59)	.11	1.62 (0.47–5.58)	.44
Suprarenal aortic control	1.83 (0.68–4.92)	.23	2.09 (0.69–6.35)	.19
Intraop transfusion pRBC – units	1.04 (0.98–1.10)	.17	1.03 (0.97–1.09)	.34
Duration of IAP $\geq$ 20 mmHg – hours	1.03 (0.99–1.08)	.15	1.10 (1.01–1.21)	.033
Bowel ischaemia *vs.* oedema	1.81 (0.55–5.93)	.33	0.33 (0.09–1.31)	.12
Post-operative bleeding *vs.* oedema	2.73 (0.83–9.01)	.10	0.23 (0.06–0.83)	.025

EVAR = endovascular aneurysm repair; IAP = intra-abdominal pressure; Intraop = intra-operative; pRBC = packed red blood cells; RRT = renal replacement therapy; OR = odds ratio; CI = confidence interval.

a p values refer to logistic regression of 1 y mortality.

b p values refer to logistic regression of need for RRT.

Although the durations of IAH before treatment, tested as hours with IAP $\geq$15 mmHg and $\geq$20 mmHg, did not predict mortality, they were independent predictors of the need for RRT. Twenty years ago, in a study on temporary abdominal closure, Sugrue et al. hypothesised that earlier use of temporary closure in situations of elevated IAP could reduce the rate of RRT.[26] The findings in this study support that hypothesis in patients with ACS, which is an important message for clinical decision making.[4] The need for RRT after surgery is considered a proxy for adverse outcome, and it is possible that the lack of correlation with mortality may be a result of too few patients, leading to a type II statistical error.

Data on outcome among different subgroups with ACS after AAA repair are scarce, with reports most often comparing those who develop ACS to those who do not.[6,8,13] Acosta et al. reported bowel ischaemia as an independent predictor of mortality in a report on patients treated with OAT after aortic repair.[23] It was hypothesised that bowel ischaemia would add to the burden of ACS, because it in itself is associated with poor survival after AAA repair.[27] This assumption could not be verified, however, nor was bowel ischaemia a predictor of death in multivariable analysis. The two studies are not completely comparable because of differences in casemix, however. Acosta et al. included all patients with OAT, also those with intestinal ischaemia in whom the abdomen was left open for reasons other than ACS, and those with OAT at the primary operation, two groups not included in this investigation.[24]

Although outcome of ACS after AAA repair depending on timing of DL has not been reported previously, studies on ACS after trauma report higher mortality in those with later decompression after admission.[28,29] This study found no difference in mortality based on timing of DL, in relation to the primary operation. Perhaps an even more important aspect of DL timing is the relation to diagnosis of ACS, and the possible effect thereof on outcome. Experimental animal data indicate that a well timed DL can reduce mortality.[30] This possible association could not be analysed in the present study. The case records rarely specified at which exact time organ dysfunction or failure developed, and ACS is defined as a combination of elevated IAP and newly developed organ dysfunction/failure.[2] Some reports suggest that patients might benefit from prophylactic OAT after rAAA repair,[7,31] based on retrospective cohort data. The WSACS clinical practice guidelines do not recommend prophylactic open abdomen treatment.[2] Patients identified with prophylactic OAT in this study were excluded.

There was a subgroup with difference in outcome, iAAA repair, where mortality was higher after EVAR compared with OSR in patients who developed ACS. The risk of developing ACS after EVAR for iAAA was very low however; there were only seven of >4100 undergoing elective EVAR procedures (0.2%), five of whom suffered serious perioperative complications. It is possible that the very low frequency is in itself a part of the explanation, as such an uncommon event may be diagnosed with delay. Furthermore, most centres do not routinely monitor IAP after EVAR for iAAA, resulting in delay to diagnosis of ACS.

DL was performed earlier after EVAR compared with OSR in rAAA. This has clinical implications as rAAA is treated increasingly by EVAR, and knowledge of ACS is necessary to optimise post-operative care. The IMPROVE trial[32] recently showed a survival benefit with EVAR *vs.* OSR for rAAA at three years, results likely to increase the use of EVAR even further. There are several explanations for the finding in the present study. Even though OSR is more invasive, haematoma removal is possible during OSR and those most physiologically deranged are also more likely to receive prophylactic OAT. The rate of fascial closure was high, around 95%, on a par with the Nordic collaboration study,[24] showing that the method also works well in routine clinical practice.

This is the largest study to date on ACS after AAA repair providing detailed information from individual case records for both iAAA/rAAA and OSR/EVAR, in a population based setting. Even this series might exhibit type II statistical error however, with regards to mortality of subgroups. The Kaplan—Meier survival analysis shows a separation of the curves depending on main pathophysiological finding at DL, yet this did not reach statistical significance.

Although a previous investigation showed external validity for AAA repair of 98.8%, and internal validity (when individual data fields were compared with data in the patients' case records) of 96.2%,[17] the specific validity of registering ACS had not been studied previously. This investigation verified that registrations of ACS were not of equally high validity. However, registrations did improve during the study period, as two thirds of the erroneous registrations originated from the first half of the study period. The shift towards early treatment of IAH preventing ACS with, for instance neuromuscular blockade,[1,2] makes the definition of IAH/ACS less clear cut, and creates a risk of misclassification. A number of patients treated with prophylactic OAT were identified, and it was not clear to all the participating centres how these patients should be registered.

In conclusion, outcome among patients developing ACS after AAA repair was poor, irrespective of the main pathophysiological finding at DL, and the timing of DL. The durations of IAP exceeding 15 and 20 mmHg were independent predictors of the need for RRT, emphasizing the need for close monitoring of ACS and early treatment, recommendations also highlighted by the recently published European Society for Vascular Surgery (ESVS) 2019 Clinical Practice Guidelines on the Management of Abdominal Aorto-iliac Artery Aneurysms.[9] ACS developed early after EVAR for rAAA, and post-operative protocols should reflect this fact.

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APPENDIX A. SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ejvs.2019.04.007.

CONFLICT OF INTEREST

None.

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COUP D'OEIL

Placement of a Central Venous Catheter Into the Right Vertebral Artery Despite Ultrasound Guidance

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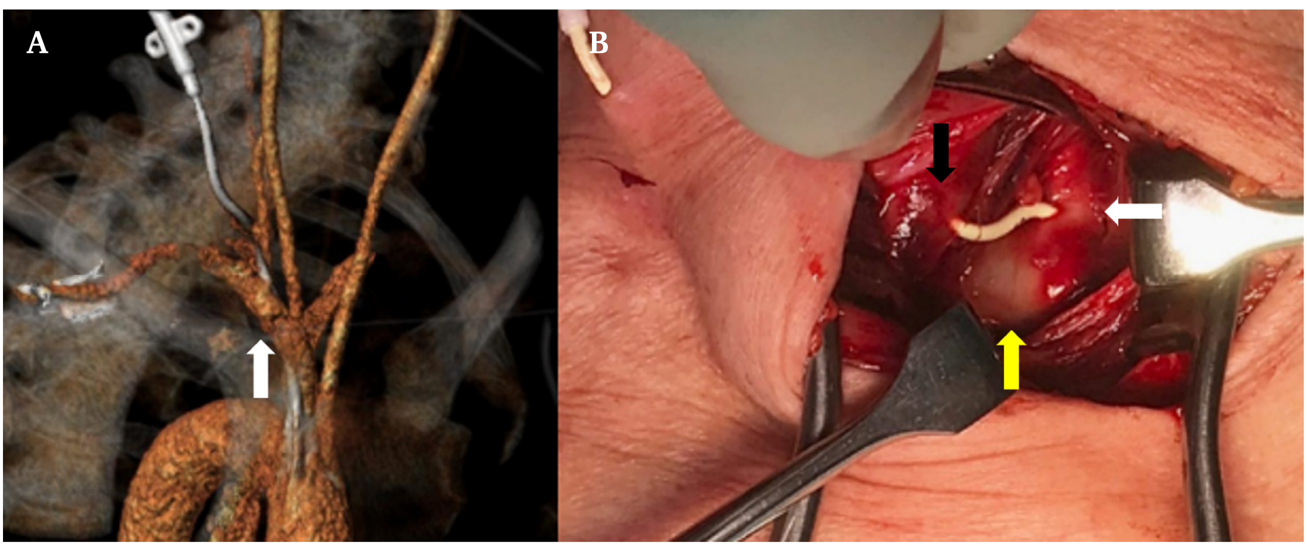

A 57 year old patient, admitted to the intensive care unit with sepsis and severe dehydration caused by a *Clostridium difficile* infection, received a central venous catheter (CVC) on the right side of her neck. After several days, doubts arose about the CVC position. Computed tomography angiography (panel A) and subsequent surgery (panel B), showed that the CVC had been placed through the right internal jugular vein (black arrow) into the right vertebral artery just after its origin (white arrows) from the subclavian artery (yellow arrow) despite being placed under ultrasound guidance. The vertebral artery was approached medial to the sternocleidomastoid. Post-operative recovery was uneventful.

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