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Risk Factors for Abdominal Compartment Syndrome After Endovascular Repair for Ruptured Abdominal Aortic Aneurysm: A Case Control Study

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WHAT THIS PAPER ADDS

Data on the risk of abdominal compartment syndrome (ACS) after ruptured endovascular aneurysm repair (rEVAR) are limited. Neither the extent to which haemodynamic derangement influences ACS nor the effect of morphological factors has been reported. Here, ACS after rEVAR was mainly associated with physiological factors, while morphological factors, such as treatment outside the instructions for use, or the number of patent branches from the stent graft treated aorta, were not. ACS was rare without a pre-operative blood pressure < 70 mmHg, aortic balloon occlusion, or more than five unit intra-operative blood transfusions. These results may help to better identify high risk patients for ACS who need more intensive monitoring.

Objective: Ruptured abdominal aortic aneurysms (rAAA) are treated by endovascular aneurysm repair (rEVAR) increasingly often. Despite rEVAR being a minimally invasive method, abdominal compartment syndrome (ACS) remains a significant post-operative threat. The aim of this study was to investigate risk factors for ACS after rEVAR, including aortic morphological features.

Methods: The Swedish vascular registry (Swedvasc) was assessed for ACS after rEVAR in the period 2008 — 2015. All patients identified were compared with controls (i.e., patients who did not develop ACS after rEVAR), matched by centre and repair date. Case records were reviewed, and radiology images analysed in a core laboratory. Comparisons were performed with respect to physiological and radiological risk factors.

Results: The study population consisted of 40 patients with ACS and 68 controls. Pre-operatively, patients with ACS had a lower blood pressure (BP) than controls (median 70 mmHg *vs.* 97 mmHg; $p < .001$). Intra-operatively, they had aortic balloon occlusion more often (55.0% *vs.* 10.3%; $p < .001$) and received more transfusions than controls (median nine units of packed red blood cells [pRBC] *vs.* two units; $p < .001$). Ninety-seven per cent of those who developed ACS had a pre-operative BP < 70 mmHg, aortic balloon occlusion, or received more than five pRBC unit transfusions. Treatment outside the instructions for use did not differ between patients and controls (57.5% *vs.* 54.4%; $p = .84$), and neither did the pre-operative patency of the inferior mesenteric artery (57.1% *vs.* 63.9%; $p = .52$) nor the number of visible lumbar arteries on pre-operative imaging (2 *vs.* 4; $p = .014$). In multivariable logistic regression, the number of intra-operative transfusions were predictive of ACS ($p < .001$), while pre-operative hypotension ($p = .32$) and aortic balloon occlusion ($p = .018$) were not.

Conclusion: ACS after rEVAR is mainly associated with physiological factors and is unlikely to develop without the presence of a pre-operative BP < 70 mmHg, the need for an aortic occlusion balloon, or more than five intra-operative pRBC unit transfusions. Treatment outside the IFU or any other morphological factor were not associated with a risk of ACS.

Keywords: Abdominal aortic aneurysm, Abdominal compartment syndrome, Endovascular aneurysm repair, Intra-abdominal pressure
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INTRODUCTION

Ruptured abdominal aortic aneurysms (rAAA) are treated increasingly often by endovascular aneurysm repair (rEVAR),[1,2] which is supported by evidence and clinical practice guidelines.[3,4] Despite the minimally invasive nature of rEVAR, 5% — 21% of those treated develop abdominal compartment syndrome (ACS)[5–10] of which 30% — 83% die.[5,8,10] Haemodynamic instability with massive transfusion, copious fluid delivery, aortic balloon occlusion, and conversion from bifurcated to aorto-uni-iliac stent grafts have been associated with ACS development after rEVAR. However, these observations originate from only a few small studies, each with < 10 patients with ACS included.[5,11] Thus, there is a need for additional data as to what degree different factors affect the risk of ACS and whether the risk of ACS can be predicted.

Possible additional risk factors for ACS after rEVAR include anatomical/morphological features such as treatment outside the instructions for use (IFU), risk of type I endoleak, and the presence of type II endoleak (T2EL). They may predispose the patient to continued leakage of blood from the ruptured aneurysm sac into the intraperitoneal/retroperitoneal space. While aortic morphology affects survival after rAAA repair, with short aortic necks and treatment outside the IFU being associated with increased mortality,[12,13] its significance in ACS is unclear. T2EL through patent inferior mesenteric and lumbar arteries are known risk factors for aneurysm sac expansion and rupture over time,[14] where the risk also seems proportionate to the specific patency of the inferior mesenteric artery and the number of patent lumbar arteries.[15] T2EL has been suggested to contribute to the development of ACS;[5] however, this has not previously been investigated and remains unclear.

The aim of this study was to investigate risk factors for ACS after rEVAR and whether these include morphological features of the aorta.

MATERIALS AND METHODS

Study design

This was a nationwide retrospective case control study of ACS after EVAR for rAAA. The study was approved by the regional ethical review board in Uppsala, Sweden (Dnr 2014-151 and 2014-151/1). The standard STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines for case control studies were observed, and the relevant checklist is available in the Supplementary Material. The Swedish Vascular Registry (Swedvasc) prospectively registers vascular procedures in Sweden, with nationwide coverage since 1994 and registration of ACS since May 2008. Recent international independent validation of the registry showed > 98.8% coverage (external validity) for aortic procedures.[16]

Patient selection

All AAA repairs registered in Swedvasc between May 2008 and September 2015 were assessed for indication for treatment (ruptured repair), treatment method (rEVAR), and development of ACS (Fig. 1). Each identified rEVAR patient with ACS was assigned the preceding and succeeding rEVAR patients treated at the same centre, but not recorded for ACS, as controls. If sequential patients were recorded for ACS, the two patients nearest in time to each patient with ACS were chosen as controls. In a classical case control study, cases are sick and controls are healthy. However, in this study cases had developed the complication of ACS while controls had not, and they were both

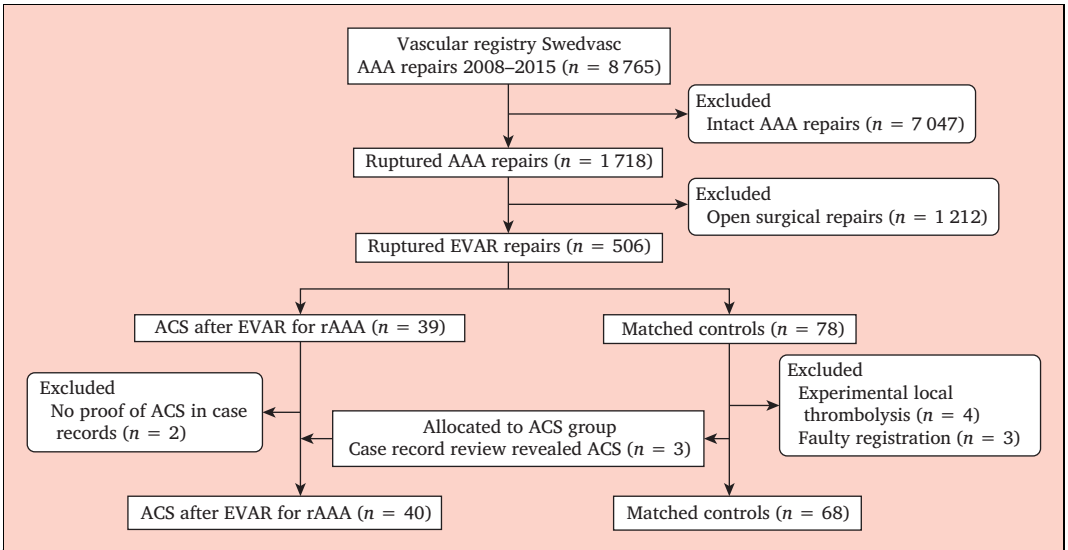

Figure 1. Selection of study population of 40 patients with abdominal compartment syndrome (ACS) patients and 68 controls treated by endovascular aneurysm repair (EVAR) for ruptured abdominal aortic aneurysm (rAAA) treated in Sweden in from 2008 to 2015.

nested in the population based cohort of patients treated for rAAA by rEVAR. The case records were reviewed retrospectively and all patients whose case records confirmed either rEVAR and ACS (cases) or rEVAR without ACS (controls) were included. All data presented in the study derive from the individual case records.

Intra-abdominal pressure (IAP) is not recorded in Swedvasc, while ACS is recorded as a "yes/no" tick box. The diagnoses of ACS were made by individual clinicians at each centre. Swedish vascular centres use the Foley Manometer technique for IAP measurement and adhere to the definition of ACS detailed in the clinical practice guidelines of the Abdominal Compartment Society:[17] a sustained IAP of $>$ 20 mmHg with new onset organ dysfunction or failure. However, IAP values could not always be found in the case records, resulting in a lot of missing values and less reliable IAP statistics. This was especially common in patients with ACS decompressed immediately on the table following the completion of rEVAR and in controls never admitted to the intensive care unit.

Treatment of the included patients was carried out according to the protocols and standards of each centre. The indication for aortic balloon occlusion during rEVAR was profound circulatory instability in all patients, but there were no standardised physiological thresholds. The case records did not detail the duration of aortic balloon occlusion, and thus data were compiled as "yes" or "no" answer.

Patients receiving experimental local thrombolysis treatment for intra-abdominal hypertension were excluded, and controls who, upon review of the case records, were found to have developed ACS, were crossed over to the ACS group (Fig. 1).

Patients referred from other hospitals were excluded from the time to hospital and time to surgery analysis, owing to uncertain time information in those cases. Patients who did not survive 24 or 48 hours post-operatively were excluded from post-operative transfusion analysis in those time periods, respectively.

Patients with ACS and controls were compared with respect to pre-, intra-, and post-operative physiological risk factors; morphological risk factors; and outcome. Pre-operative blood pressure (BP) refers to the lowest measured pre-operative BP. Receiver operator characteristic (ROC) curves were plotted for physiological risk factors of interest.

Radiology assessment

Radiology imaging analysis was performed with dedicated software for radiology imaging reconstruction (Vital Images [Minnetonka, MN, USA] and 3mensio Medical Imaging [Maastricht, The Netherlands]). The examiners were blinded to whether the patient had developed ACS or not. Measurements of diameter and angulation were made by reference to the centre lumen line, where diameter was the calculated average of anteroposterior and lateral projections using outer to outer vessel wall measurement. All pre-operative computed tomography (CT) images were analysed by an experienced vascular surgeon (S.E.). Borderline measurements were then separately analysed by another experienced vascular surgeon (H.B.). The following conditions mandated borderline designation: firstly, if the proximal neck had a 5% − 15% diameter increase (inverted funnel shape) along the required proximal neck length (the recommended maximum diameter increase is 10%); secondly, if the iliac artery had a 5% − 15% inverted diameter increase (funnel shape) along the distal fixation site; and thirdly, if the alpha and beta angulations of the proximal neck were within 15° range of the recommended maximum angulation. Conflicting measurements between examiners were handled by joint re-measurement to reach consensus. Pre-operative internal iliac artery occlusion, aneurysm rupture site, visible active extravasation, and the patency of the inferior mesenteric and lumbar arteries were not subject to borderline designation and were assessed by one examiner (S.E.).

Measurements were dichotomised, creating one group compliant with device specific IFU (inside IFU), as defined for intact AAA repair, and another group non-compliant with the IFU (outside IFU).

Statistical analysis

Continuous variables were checked for normality using visual assessment of histograms supplemented by the Shapiro—Wilk test. As non-normal distribution proved to be the norm, continuous variables were reported as median (interquartile range [IQR]). In the event of missing data, the case was excluded from respective analysis. Among physiological risk factors, dichotomised values for pre-operative BP and intra-operative packed red blood cell (pRBC) transfusions were tested alongside aortic balloon occlusion in models of two or all three factors. Comparison of continuous variables was done with the Mann—Whitney *U* test, while comparison of proportions was done with Fisher's exact test. Spearman's rank test was used to test for correlations. Logistic regression was performed by forced entry, evaluating risk factors. Statistical analysis was done in SPSS Statistics version 25 (IBM, Armonk, NY, USA) with the statistical significance threshold set to $p < .01$, adjusting for multiple testing, and all tests being two sided.

RESULTS

The final study population included 108 patients from seven different vascular centres; 40 patients with ACS and 68 matched controls (Fig. 1). Baseline characteristics were similar between groups, while all outcomes were worse among patients with ACS (Table 1).

Peak IAP was 23 mmHg (IQR 21 − 28 mmHg) in patients with ACS *vs.* 13 mmHg (IQR 11 − 18 mmHg) in controls ($p <$ .001). The number of IAP measurements per 24 hours was eight (IQR 5.3 − 12.0) in patients with ACS *vs.* 5.1 (IQR 3.6 − 8.0) in controls ($p = .035$).

Pre-operatively, patients with ACS had lower BPs (70 mmHg *vs.* 97 mmHg; $p < .001$) and higher rate of unconsciousness (62.5% *vs.* 32.3%; $p = .004$) than controls. Intra-

Table 1. Clinical characteristics and outcome of study population of 40 patients with abdominal compartment syndrome (ACS) and 68 controls treated by endovascular aneurysm repair for ruptured abdominal aortic aneurysm

	Valid − *n**	ACS (*n* = 40)	Controls (*n* = 68)	*p* value
Age − y	108	78 (74.5–81)	79 (72–82.5)	.70
Female sex	108	9 (23)	12 (18)	.62
Maximum aortic diameter − mm	108	78 (70–85)	74 (64–87)	.33
Cardiac disease	92	16 (40)	20 (38.5)	1.0
Pulmonary disease	95	9 (23.1)	15 (26.8)	.81
Diabetes	99	6 (15)	9 (15.3)	1.0
Previous cerebrovascular event	92	3 (7.7)	11 (20.8)	.14
Referrals	108	18 (45)	39 (57)	.24
30 d mortality	108	20 (50)	12 (18)	.001
90 d mortality	108	25 (63)	12 (18)	<.001
1 y mortality	108	27 (68)	20 (29)	<.001
Renal replacement therapy	108	18 (46)	7 (10)	<.001
Mechanical ventilation − d	108	2.5 (5.0–14.0)	0 (0–1.0)	<.001

Data are presented as *n* (%) or median (interquartile range).
*The number of cases with valid data out of 108.

operatively, patients with ACS had a higher rate of aortic balloon occlusion (55.0% *vs.* 10.3%; $p < .001$), greater external blood loss (1.7 L *vs.* 0.4 L; $p = .002$), received more transfusions (nine units of pRBC *vs.* two units of pRBC; $p < .001$), as well as intravenous fluids (3.2 L *vs.* 2.0 L; $p < .001$), and had longer operation time (183 minutes *vs.* 130 minutes; $p = .020$) than controls. Post-operatively, patients with ACS received more transfusions at 0 − 24 hours (five units of pRBC *vs.* none; $p < .001$) and at 24 − 48 hours (one unit of pRBC *vs.* none; $p = .002$) after rEVAR than controls (Table 2).

ROC curves indicated that pre-operative BP, intra-operative bleeding, and intra-operative and 0 − 24 hours post-operative pRBC transfusions were useful for predicting ACS (Fig. 2), where intra-operative pRBC transfusions showed the greatest area under curve (.857).

Among patients with ACS patients, 97.2% had a pre-operative BP $\leq$ 90 mmHg and 46.4% had a BP < 70 mmHg *vs.* 44.4% and 16.1% of controls, respectively ($p < .001$ and $p = .004$, respectively). Eighty per cent of patients with ACS received more than five units of pRBCs *vs.* 21.2% of controls ($p < .001$). In a model combining three risk

Table 2. Peri-operative characteristics of 40 patients with abdominal compartment syndrome (ACS) and 68 controls with endovascular aneurysm repair for ruptured abdominal aortic aneurysm

	Valid *n**	ACS (*n* = 40)	Controls (*n* = 68)	*p* value
Pre-operative				
Interval of symptoms to hospital − h	75	5.3 [1.5–13.6]	10.3 [2.1–29.4]	.24
Interval of hospital to surgery − h	88	2.1 [1.3–3.3]	3.4 [1.2–5.8]	.20
Lowest measured BP − mmHg	92	70 [58–80]	97 [70–110]	<.001
Unconsciousness	105	62.5 (47.5–77.5)	32.3 (20.9–43.7)	.004
Asystole	106	2.5 (0.0–13.2)	3.0 (0.0–10.5)	1.0
Intra-operative				
Aortic occlusion balloon	108	55.0 (38.5–70.7)	10.3 (4.2–20.1)	<.001
Operating time − min	105	183 [124–265]	130 [90–210]	.020
Operative bleeding − mL	49	1750 [900–3500]	400 [200–1000]	.002
Total amount of fluids − mL	85	3200 [2950–4000]	2050 [1600–3250]	<.001
pRBC − units	101	9 [6–16]	2 [0–5]	<.001
FFP − units	101	5 [3–12]	0 [0–3]	<.001
Platelets − units	101	4 [0–12]	0 [0–0]	<.001
Post-operative				
pRBC day 1 − units	93/96	5 [2–12]	0 [0–3]	<.001
FFP day 1 − units	93/96	5 [1–10]	0 [0–0]	<.001
Platelets day 1 − units	93/96	4 [0–8]	0 [0–0]	<.001
pRBC day 2 − units	89/93	1 [0–3]	0 [0–1]	.002
FFP day 2 − units	89/93	0 [0–3]	0 [0–0]	<.001
Platelets day 2 − units	89/93	0 [0–4]	0 [0–0]	<.001

Data are presented as median [interquartile range] or % (95% confidence interval). BP = blood pressure; pRBC = packed red blood cells; FFP = fresh frozen plasma.
*The number of cases with valid data out of 108 unless stated otherwise.

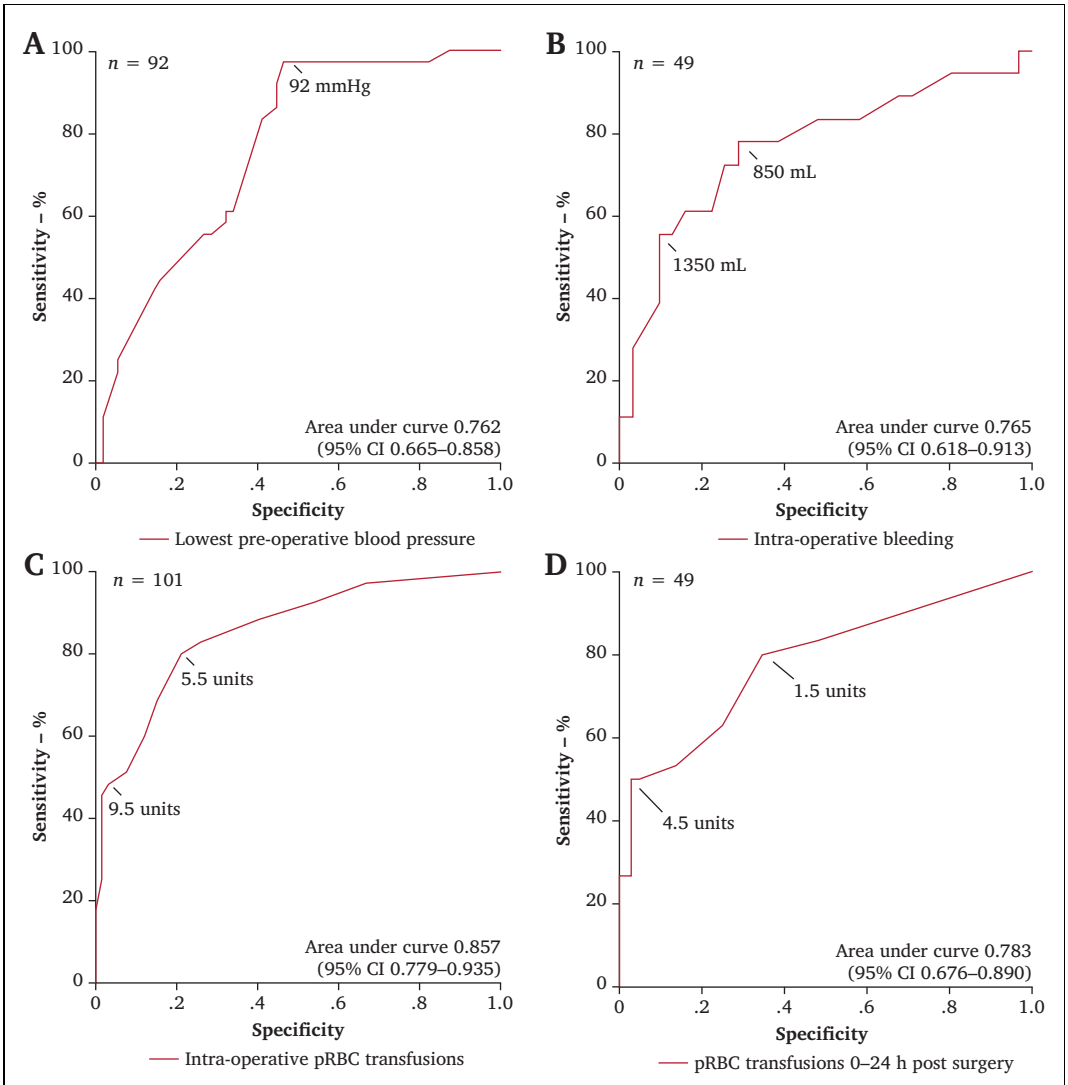

Figure 2. Receiver operator characteristic (ROC) curves of the accuracy of peri-operative risk factors for (A) lowest pre-operative blood pressure, (B) intra-operative bleeding, (C) intra-operative packed red blood cell (pRBC) transfusions, and (D) pRBC transfusions within 24 hours of surgery, to discriminate between 40 patients with abdominal compartment syndrome (ACS) and 68 controls after endovascular repair of ruptured abdominal aortic aneurysm. CI = confidence interval.

factors, 97.2% of patients with ACS had at least one of the following factors: a pre-operative BP $<$ 70 mmHg, aortic balloon occlusion, or more than five intra-operative pRBC unit transfusions *vs*. 38.3% of controls ($p < .001$). The presence of all three risk factors was found in up to 50.0% of patients with ACS and in, at most, 4.6% of controls, depending on which cutoff values were used (Table 4). In a logistic regression model testing these three variables, intra-operative transfusions was independently associated with ACS (Table 5).

The frequency of treatment outside the IFU, inferior mesenteric artery patency, the number of visible lumbar arteries, and visible extravasation on pre-operative CT did not differ between patients with ACS and controls (Table 3). The number of visible lumbar arteries was correlated with pre-operative BP ($r = -.372$, $p < .001$).

DISCUSSION

In this study of ACS predictors after rEVAR, the development of ACS was mainly associated with classical physiological risk factors, while morphological risk factors were not associated with ACS risk.

An association between haemodynamic instability, such as pre-operative hypotension, the need for an aortic occlusion balloon, and massive intra-operative transfusions, and the development of ACS after rEVAR has been reported previously in two small studies.[5,11] However, the extent of the association between haemodynamic derangement and ACS has not been described. This study showed that haemodynamic derangement was not only a risk factor among others, but also that it was present in nearly all of the cases and can almost be considered a prerequisite for ACS development after rEVAR. Development of ACS was rare

Table 3. Radiological characteristics of 40 patients with abdominal compartment syndrome (ACS) and 68 controls with endovascular aneurysm repair for ruptured abdominal aortic aneurysm

	Valid *n**	ACS (*n* = 40)	Controls (*n* = 68)	*p* value
CT with contrast	108	90.0 (80.7–99.3)	88.2 (80.6–95.9)	1.0
Maximum aortic diameter – mm	108	78 [70–85]	74 [64–87]	.33
Pre-operative IIA occlusion	96	2.9 (0.1–14.9)	7.1 (4.5–14.9)	.65
Neck outside IFU	108	45.0 (29.6–60.4)	33.8 (22.6–45.1)	.31
Neck angulation outside IFU	108	22.5 (9.6–35.4)	20.6 (11.0–30.2)	.81
Landing outside IFU	108	10.0 (0.7–19.3)	14.7 (6.3–23.1)	.57
Any outside IFU	108	57.5 (42.2–72.8)	54.4 (42.6–66.2)	.84
Patency of IMA	96	57.1 (40.7–73.5)	63.9 (51.9–76.0)	.52
Patent lumbar arteries	96	2 [1–4]	4 [2–5]	.014
Diameter > 2 mm	96	1 [1–2]	2 [1–3]	.041
Diameter > 1 mm	96	1 [1–2]	2 [1–3]	.14
Active extravasation	96	55.6 (39.3–71.8)	45.0 (32.4–57.6)	.40
Anterior rupture site	55	47.8 (27.4–58.2)	28.1 (12.5–43.7)	.16
Intra-operative adjuncts	105	42.1 (26.4–57.8)	34.3 (23.0–45.7)	.53

Data are presented as median [interquartile range] or percentage (95% confidence interval). CT = computed tomography; IIA = internal iliac artery; IFU = instructions for use; IMA = inferior mesenteric artery.
*The number of cases with valid data out of 108 unless stated otherwise.

Table 4. Comparison of separate and composite peri-operative variables between 40 patients with abdominal compartment syndrome (ACS) and 68 controls with endovascular aneurysm repair for ruptured abdominal aortic aneurysm

	Valid *n**	ACS (*n* = 40)	Controls (*n* = 68)	*p* value
Individual factor analysis				
BP ≤ 90mmHg	92	97.2 (85.5–99.9)	46.4 (33.0–60.3)	<.001
BP < 70mmHg	92	44.4 (27.9–61.9)	16.1 (7.6–28.3)	.004
AOB	108	55.0 (38.5–70.7)	10.3 (4.2–20.1)	<.001
> 2 units pRBC	101	88.6 (73.3–96.8)	40.9 (29.0–53.7)	<.001
> 5 units pRBC	101	80.0 (63.1–91.6)	21.2 (12.1–33.0)	<.001
> 9 units pRBC	101	48.6 (31.4–66.0)	3.0 (0.4–10.5)	<.001
Two factors combined analysis				
BP ≤ 90 mmHg + AOB	104	52.6 (35.8–69.0)	6.1 (1.7–14.8)	<.001
BP < 70 mmHg + AOB	104	28.9 (15.4–45.9)	3.0 (0.4–10.5)	<.001
BP ≤ 90 mmHg + >2 units pRBC	93	87.9 (71.8–96.6)	20.3 (11.0–32.8)	<.001
BP ≤ 90 mmHg + >5 units pRBC	96	72.7 (54.5–86.7)	12.5 (5.6–23.2)	<.001
BP < 70 mmHg + >2 units pRBC	92	33.3 (18.0–51.8)	8.5 (2.8–18.7)	.004
BP < 70 mmHg + >5 units pRBC	99	20.6 (8.7–37.9)	6.2 (1.7–15.0)	.043
AOB + > 2 units pRBC	101	57.1 (39.4–73.7)	6.1 (1.7–14.8)	<.001
AOB + > 5 units pRBC	105	47.4 (31.0–64.2)	4.5 (0.9–12.5)	<.001
Three factors combined analysis				
BP ≤ 90 mmHg + AOB + > 2 pRBC	101	50.0 (32.9–67.1)	4.6 (1.0–12.9)	<.001
BP ≤ 90 mmHg + AOB + > 5 pRBC	102	44.4 (27.9–61.9)	3.0 (0.4–10.5)	<.001
BP < 70 mmHg + AOB + > 2 pRBC	102	25.0 (12.1–42.2)	3.0 (0.4–10.5)	.001
BP < 70 mmHg + AOB + > 5 pRBC	103	22.2 (10.1–39.2)	3.0 (0.4–10.4)	.003
Any of the factors combined analysis				
BP ≤ 90 mmHg or AOB or > 2 pRBC	100	97.4 (86.2–99.9)	67.7 (54.7–79.1)	<.001
BP ≤ 90 mmHg or AOB or > 5 pRBC	97	97.4 (86.2–99.9)	57.6 (44.1–70.4)	<.001
BP < 70 mmHg or AOB or > 2 pRBC	99	97.3 (85.8–99.9)	54.8 (41.7–67.5)	<.001
BP < 70 mmHg or AOB or > 5 pRBC	97	97.3 (85.8–99.9)	38.3 (26.1–51.8)	<.001

Data are presented as percentage (95% confidence interval). BP = blood pressure; AOB = aortic occlusion balloon; pRBC = packed red blood cells.
*The number of cases with valid data out of 108 unless stated otherwise.

without pre-operative hypotension, and an overwhelming majority also received massive (more than five units of pRBC) intra-operative transfusions. In addition, more than half of all patients with ACS were also subjected to aortic balloon occlusion. Among these risk factors, which were tested for and had no multicollinearity, intra-operative transfusions were more influential in the development of ACS than the degree of pre-operative hypotension and the use of aortic balloon occlusion, according to the logistic regression model. The degree of pre-operative hypotension

Table 5. Univariable and multivariable logistic regression of abdominal compartment syndrome development in patients treated with endovascular aneurysm repair for ruptured abdominal aortic aneurysm

	Univariable logistic regression		Multivariable logistic regression	
	OR (95% CI)	*p* value	OR (95% CI)	*p* value
Pre-operative BP − mmHg	0.96 (0.94−0.98)	<.001	0.99 (0.96−1.01)	.32
Aortic balloon occlusion	10.65 (3.92−28.95	<.001	6.08 (1.36−27.21)	.018
Intra-operative pRBC transfusions	1.37 (1.2−1.56)	<.001	1.32 (1.13−1.54)	<.001

OR = odds ratio; CI = confidence interval; BP = blood pressure; pRBC = packed red blood cells.

indicates the extent of the bleeding from the rupture but does not necessarily indicate ongoing bleeding. However, aortic balloon occlusion and intra-operative transfusions indicate ongoing bleeding and haemodynamic instability, where an increasing number of transfusions will increase the space occupying lesion effect.

By combining the physiological risk factors into models containing either any or all risk factors, with suitable cutoffs derived from the ROC curves, optimised predictive values for ACS were obtained. In such a model the overall risk of ACS was low without the presence of a pre-operative BP < 70 mmHg, aortic balloon occlusion, or more than five intra-operative unit transfusions. In contrast, the risk of ACS was high in the presence of all three factors, which was found in up to 50% of those with ACS depending on which cutoff values were used but was rare among controls. In summary, these simple physiological risk factors can guide the clinician on which patients are at higher risk of developing ACS and thus need more intensive monitoring of IAP. The authors of this study suggest hourly IAP measurements during the first 12 post-operative hours in those with significant pre-operative hypotension, multiple intra-operative transfusions, or those subjected to aortic balloon occlusion. We also suggest intensified monitoring in all patients receiving multiple post-operative transfusions.

While short aortic necks and treatment outside the IFU have been associated with mortality after rAAA,[12,13] their role in the development of ACS is unknown. It was not possible to demonstrate that those factors, in turn, were linked to an increased ACS risk in this study. In the report by Baderkhan *et al.*,[13] challenging anatomy was found to be associated with long term mortality but not with 30 day outcome. Also, in the cohort study based on the Improve trial, short necks did not impact survival in the rEVAR subgroup.[12] However, given the appealing assumption that a challenging anatomy could contribute to the development of ACS, and given the limited size of this study with susceptibility to type II statistical errors, it cannot be ruled out that such a relationship does exist.

T2EL has been reported to be associated with and proportionate to the patency of the inferior mesenteric artery and the number of lumbar arteries on pre-operative imaging.[15] However, in this study neither patency of the inferior mesenteric artery nor the number of visible lumbar arteries were associated with ACS. There was a trend of fewer visible lumbar arteries on pre-operative imaging among patients with ACS, which may be a manifestation of more pronounced haemodynamic instability and vasoconstriction in patients with ACS. This is supported by the observed correlation between fewer visible lumbar arteries and lower pre-operative BP. Nevertheless, the present data cannot confirm the hypothesis that T2EL, measured indirectly as patent inferior mesenteric and lumbar arteries, contributes significantly to ACS development.

There are several shortcomings to this study, where the retrospective design and limited size of the study population constitute obvious limitations. Performing large studies on ACS is difficult, even in the setting of nationwide inclusion, as in this study. A larger study population would have reduced the risk of type II statistical errors. The initial setup of the study was to include cases up to 2015. The data collection involved multiple regulatory aspects, and was both complex and time consuming, leading to the fact that data were not available beyond 2015. Another limitation of this study is that among the minority of patients with ACS decompressed immediately after aneurysm exclusion (during the same procedure), their operating times included the decompressive laparotomy, and intra-operative transfusions included any units of pRBC given during the decompression, unlike the other patients in the study. The operative records were not specified to permit distinction between the two phases of the procedure.

In conclusion, ACS after rEVAR appears to be highly determined by physiological factors and was found to be amenable to prediction based on these factors. The likelihood of ACS was considerably lower in the absence of the following three risk factors: pre-operative blood pressure < 70 mmHg, more than five intra-operative pRBC transfusions, or the use of aortic occlusion balloon. Morphological factors, such as patent inferior mesenteric and lumbar arteries (as surrogate markers for T2EL), or treatment outside of the IFU were not associated with the development of ACS.

CONFLICTS OF INTEREST

None.

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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.2021.05.019.

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