Editor's Choice — Structured Computed Tomography Analysis can Identify the Majority of Patients at Risk of Post-Endovascular Aortic Repair Rupture

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WHAT THIS PAPER ADDS

This manuscript reports that the use of a structured protocol for analysis for computed tomography review after endovascular aneurysm repair (EVAR) increases detection of the precursors of post-EVAR rupture. Mechanisms and precursors of post-EVAR rupture were identified. Failure at the distal sealing zone and graft interconnections were the mechanism in 61% of ruptures and the proximal sealing zone accounted for 39%. The incidence rate was 4.5/1 000 person years and the median time to post-EVAR rupture was 4.1 years, with no decline in incidence over time passed after EVAR. The results suggest that increased detection of precursors could enable pre-emptive treatment and reduce the incidence of post-EVAR rupture.

Objective: The main objective was to report mechanisms and precursors for post-endovascular aneurysm repair (EVAR) rupture. The second was to apply a structured protocol to explore whether these factors were identifiable on follow up computed tomography (CT) prior to rupture. The third objective was to study the incidence, treatment, and outcome of post-EVAR rupture.

Methods: This was a multicentre, retrospective study of patients treated with standard EVAR at five Swedish hospitals from 2008 to 2018. Patients were identified from the Swedvasc registry. Medical records were reviewed up to 2020. Index EVAR and follow up data were recorded. The primary endpoint was post-EVAR rupture. CT at follow up and at post-EVAR rupture were studied, using a structured protocol, to determine rupture mechanisms and identifiable precursors.

Results: In 1 805 patients treated by EVAR, 45 post-EVAR ruptures occurred in 43 patients. The cumulative incidence was 2.5% over a mean follow up of 5.2 years. The incidence rate was 4.5/1 000 person years. Median time to post-EVAR rupture was 4.1 years. A further six cases of post-EVAR rupture in five patients found outside the main cohort were included in the analysis of rupture mechanisms only. The rupture mechanism was type IA in 20 of 51 cases (39%), IB in 20 of 51 (39%) and IIIA/B in 11 of 51 (22%). One of these had type IA + IB combined. One patient had an aortoduodenal fistula without another mechanism being identified. Precursors had been noted on CT follow up prior to post-EVAR rupture in 16 of 51 (31%). Retrospectively, using the structured protocol, precursors could be identified in 43 of 51 (84%). In 17 of 27 (63%) cases missed on follow up but retrospectively identifiable, the mechanisms were type IB/III. Overall, the 30 day mortality rate after post-EVAR rupture was 47% ($n = 24/51$) and the post-operative mortality rate was 21% ($n = 7/33$).

Conclusions: Most precursors of post-EVAR rupture are underdiagnosed but identifiable before rupture using a structured follow up CT protocol. Precursors of type IB and III failures caused the majority of post-EVAR ruptures.

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INTRODUCTION

Endovascular aneurysm repair (EVAR) is associated with a lower 30 day mortality rate than open surgical repair (OSR), and also with a higher rate of aneurysm related complications requiring re-intervention,[1] affecting approximately 20% of patients treated by EVAR for intact abdominal aortic aneurysm (AAA) and 35% of patients treated for ruptured AAA (rAAA).[2,3]

Post-treatment rupture is a serious complication, reported to have an incidence of 0.6 — 0.9/1 000 person years for OSR[4,5] and 2.0 — 6.0/1 000 person years for EVAR.[4,6] The time from the index EVAR to post-EVAR rupture has been reported to be a median of 2.6 — 3.5 years[7,8] and 3.1 — 5.1 years in studies presenting mean follow up times.[9—12] Post-EVAR ruptures have been shown to occur at a constant rate over the years following EVAR,[5,6] with a 30 day peri-operative mortality of 25% — 32%.[6,9] However, as a significant proportion of patients are not treated, total 30 day mortality of post-EVAR rupture is higher (42% — 67%).[6,12]

The design of EVAR follow up programmes is currently being discussed, and algorithms with the reduced use of computed tomography (CT) scans have been proposed.[13,14] However, there is limited knowledge of the mechanisms and warning signs of post-EVAR rupture. Increased understanding of factors associated with post-EVAR rupture is essential if the long term durability of EVAR is to be improved, and is an important contribution to the discussion of stratified follow up after EVAR.

The primary objective of this study was to identify the mechanisms and precursors of post-EVAR rupture. The second objective was to explore whether these precursors were identifiable with the use of a structured protocol for review of follow up CT. The third objective was to study the incidence, treatment, and outcome of post-EVAR rupture.
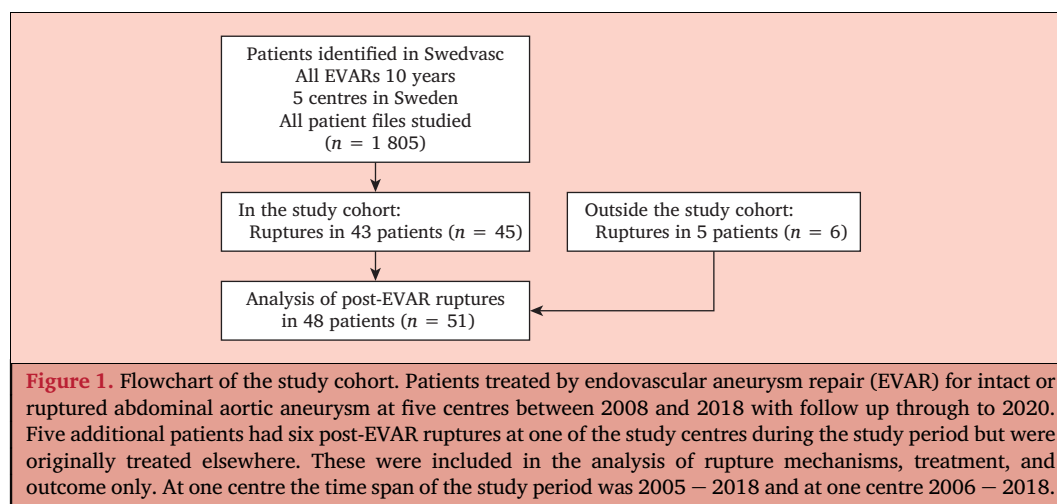
MATERIALS AND METHODS

Design

This was a retrospective observational multicentre study of all patients treated with standard bi- or uni-iliac infrarenal EVAR at five Swedish hospitals between 2008 and 2018. The primary outcome was post-EVAR rupture, defined as rAAA following discharge after the primary EVAR. Patients with a primary repair were identified in the Swedish National Quality Registry for Vascular Surgery (Swedvasc) and baseline data were extracted from the registry. Swedvasc has an agreement with hospital statistics of 96.2%, and almost 100% national coverage regarding primary AAA registrations.[15] All 1 805 patients' medical records were reviewed up to 2020 at the five participating centres. Data regarding the index EVAR procedure, complications, re-interventions, aneurysm development, and post-EVAR rupture were recorded and added to the Swedvasc baseline data.

Study population

Patients who had a primary standard bi- or uni-iliac infrarenal EVAR at any of the participating centres during the study period were included in the main study cohort. The participating centres and study periods were Sahlgrenska University Hospital, Gothenburg (2005 — 2018); Sunderbyn Hospital, Sunderbyn (2006 — 2018); Karolinska University Hospital, Stockholm; Södersjukhuset University Hospital, Stockholm; and Ryhov Hospital, Jönköping (2008 — 2018). Fenestrated and chimney EVAR, and Nellix stent grafts were excluded. A cohort of 1 805 consecutive patients with standard EVAR was identified and reviewed.

Follow up began on the date of the primary EVAR and ended on the date of death, conversion to open repair, or on the date of patient file review.

Only patients from the main study cohort were included in the analysis of incidence and risk factors.

Figure 1. Flowchart of the study cohort. Patients treated by endovascular aneurysm repair (EVAR) for intact or ruptured abdominal aortic aneurysm at five centres between 2008 and 2018 with follow up through to 2020. Five additional patients had six post-EVAR ruptures at one of the study centres during the study period but were originally treated elsewhere. These were included in the analysis of rupture mechanisms, treatment, and outcome only. At one centre the time span of the study period was 2005 — 2018 and at one centre 2006 — 2018.

Patient ID...........................
Date of primary repair ....

CT Date ....
CT with contrast □ CT no contrast □ Slice thickness ....

Lowest renal artery
Right (R) □ Left (L) □

Distance to stent graft marker.....
Proximal attachment length ......

Endoleak Type IA Yes □ No □

Proximal aortic diameter:
⌀ Renal artery ...... x ......
⌀ Stent-graft edge ...... x ......
⌀ 10 mm ...... x ......
⌀ 20 mm ...... x ......
⌀ Distal ...... x ......
Graft nominal diameter ....

Distance flow-split marker to
most proximal marker
contralateral limb ......
Placement within IFU
Yes □ No □
Endoleak type III Yes □ No □

Overlap between main body and
distal limb R ......

Overlap between main body and
distal limb L ......

Overlap between distal limb 1
and 2 R ......

Overlap between distal limb 1
and 2 L ......

Iliac landing zone R
Common □ External □

Iliac landing zone L
Common □ External □

Iliac diameter
⌀ Proximal ...... x ......
⌀ 20 mm ...... x ......
⌀ 10 mm ...... x ......
⌀ Stent graft edge ...... x ......
⌀ At internal iliac ...... x ......
Graft nominal diam ....
Attachment length ......
Length aortic bifurcation to
distal stent graft edge ......
Int. iliac to stent graft ......

MPR at maximal AAA diameter

Max diam ......
AP ......
90 ° to max ......
Transv ......

Endoleak type II Yes □ No □

Iliac diameter
⌀ Proximal ...... x ......
⌀ 20 mm ...... x ......
⌀ 10 mm ...... x ......
⌀ Stent-graft edge ...... x ......
⌀ At internal iliac ...... x ......
Graft nominal diam ....
Attachment length ......
Length aortic bifurcation to
distal stent graft edge ......
Int. iliac to stent graft ......

Endoleak type IB R Yes □ No □

Endoleak type IB L Yes □ No □

IF landning zone external iliac
(replaces iliac diameter)
Internal iliac to stent graft edge ......
⌀ Proximalt ...... x ......
⌀ 20 mm ...... x ......
⌀ 10 mm ...... x ......
⌀ Stent graft edge ...... x ......
Attachment length ......
Graft nominal diam ......
Length aortic bifurc. to distal graft edge ......

IF landning zone external iliac
(replaces iliac diameter)
Internal iliac to stent graft edge ......
⌀ Proximalt ...... x ......
⌀ 20 mm ...... x ......
⌀ 10 mm ...... x ......
⌀ Stent graft edge ...... x ......
Attachment length ......
Graft nominal diam ......
Length aortic bifurc. to distal graft edge ......

Figure 2. Protocol for structured computed tomography (CT) or CT angiography (CTA) review of the abdominal aortic aneurysm and the stent graft dimensions. Measurements were performed after creating a centre lumen line from the thinnest available CT/CTA slices, using Aquarius, version 4.4.11 (TeraRecon, Durham, NC, USA). IFU = instructions for use; MPR = multiplanar reconstruction; AAA = abdominal aortic aneurysm.

At one centre the records of all EVAR procedures were reviewed in addition to the Swedvasc data files, which identified another six post-EVAR ruptures in five patients. These patients had their index EVAR outside the study period or at other hospitals but were treated for their post-EVAR rupture at the study centre. These were included in the analysis for rupture mechanism and outcome of treatment at post-EVAR rupture only, giving a total of 51 ruptures in 48 patients (Fig. 1). Follow up after EVAR at the participating centres generally consisted of a contrast

enhanced CT scan at 30 days and yearly thereafter with variations due to changes in local routines during the study period and patient related factors.

Analysis of rupture mechanisms

In patients with post-EVAR rupture, the CT or CT angiography (CTA) at post-EVAR rupture and during follow up were reviewed using a structured protocol. Firstly, CT scans at post-EVAR rupture were reviewed to establish the rupture mechanism defined as the type and site of failure of seal leading to rupture. In cases where a CT at post-EVAR rupture was missing, operative findings and autopsy protocols were investigated.

Next, the pre-operative CT, CT at 30 days, and the last CT before rupture were studied, to determine whether the EVAR had been performed within instructions for use (IFU), and to identify precursors of the subsequent post-EVAR rupture mechanism. When needed to differentiate between migration or vessel dilatation as the primary precursor, additional CT scans were reviewed. A review of patient files, CT and ultrasound reports, and conference notes prior to rupture was performed for each case to determine whether any precursor of the subsequent rupture mechanism had been noted during the follow up. If so, the reason not to treat it was determined. All sac diameters during follow up were recorded. The reviewers were not blinded for the clinically established rupture mechanism. Images were reviewed at a core laboratory by two vascular imaging specialists (C.S. and H.R.) in consensus.

Protocol for computed tomography review

CT or CTA were performed at several different hospitals and scanners in the catchment area of the participating hospitals. The thinnest available CT/CTA slices were transferred to a commercially available post-processing workstation (Aquarius, version 4.4.11; TeraRecon, Durham, NC, USA).

A centre lumen line was created using the semi-automatic or manual multiclick function and thereafter adjusted manually to the anatomy.

Proximal and distal attachment lengths were defined as lengths along centre lumen line with stent graft apposition to the vessel wall. Measurements of attachment length and vessel diameter of the attachment area were made. Diameters at the landing zones were correlated with the nominal graft diameter. Diameters were measured at each graft edge, 10 and 20 mm from the edge and at the level of renal and internal iliac arteries (Fig. 2). Sealing zone expansion was defined as an increase of the vessel diameter above nominal graft diameter and loss of graft apposition to the vessel wall. Stent graft overlap was reviewed with the centre lumen protocol and post-processed three dimensional (3D) imaging to facilitate the identification of each graft component's edges and markers (Supplementary Fig. S1).

Settings for windowing of post-processed 3D imaging was adjusted for optimal visualisation of the stent graft components (width range between 0 and 200 and level between 650 and 1 200).

The findings were correlated with each manufacturer's IFU. A shorter attachment length than IFU at first post-operative imaging was defined as inadequate seal at primary repair. Dilatation of the attachment zones was defined as a dilatation of the vessel exceeding the nominal stent graft diameter, resulting in loss of seal. Migration was defined as movement of the graft > 5 mm. Migration at the infrarenal (proximal) landing zone was defined as an increase in distance from the lowest renal artery to the stent graft edge of > 5 mm. Migration at the iliac (distal) landing zones was defined as an increase of the distance from the internal iliac ostium to the distal graft edge and a corresponding decrease in the distance from the graft edge to the aortic bifurcation, in order to discriminate from vessel elongation. Migration in the interconnection zones was defined as a shortened overlap measured between component markers. Expansion or AAA shrinkage was defined as a change in the maximum diameter of the aneurysm sac of > 5 mm and was measured on the CT at 30 days, one year, and at the last follow up CT.

Statistical analysis

Continuous variables are presented as mean $\pm$ standard deviation, and categorical variables as frequencies and percentages. Differences between group means were analysed with Student's t test. Associations between categorical variables and differences between proportions were analysed with chi square tests. Any p value $< .050$ was considered statistically significant. In the calculation of interval between the index EVAR and post-EVAR rupture, only time to the first post-EVAR rupture of each study person was included. Two patients had repeated events; consequently, the time to the second event was disregarded. The crude time to rupture from the index EVAR procedure was presented using Kaplan–Meier analysis. The number of post-EVAR ruptures of each stent graft type was presented as number, proportion, and incidence rate with confidence intervals. All calculations were performed with SPSS 26 (IBM, Armonk, NY, USA) and OpenEpi (www.OpenEpi.com).

Ethical permission and reporting guidelines

This study was approved by the Research Ethics Committee in Gothenburg (number 508-14). The study was conducted according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for the reporting of observational studies.[16]

RESULTS

Of the 1 805 patients treated by EVAR, 260 (14.4%) were female and the indication was rAAA in 253 (14.0%; Table 1). In the main study cohort, 45 post-EVAR ruptures in 43 patients were identified. Of these, five (12%) were female and in six (14%) the indication was rAAA. The cumulative incidence was 2.5% over a mean follow up of 5.2 years, and with a total of 9 469 person years of follow up, the incidence rate was 4.5/1 000 person years. The median time to post-EVAR rupture was

Table 1. Baseline characteristics of 1 805 patients treated by endovascular aneurysm repair (EVAR) with 45 ruptures in 43 patients after EVAR

Characteristics	All (n=1 805)	No post-EVAR rupture (n=1 762; 97.6%)	Post-EVAR rupture (n=43; 2.4%)	p value
Male sex	1 545 (85.6)	1 507 (85.5)	38 (88)	.60
Age − y	75.2 ± 7.5	75.2 ± 7.5	77.6 ± 6.6	.036
Indication rAAA	253 (14.0)	247 (14.0)	6 (14)	.99
AAA diameter − mm	63.6 ± 12.8	63.4 ± 12.7	70.4 ± 16.4	.009
Diameter main body − mm	28.7 ± 3.8	28.7 ± 3.8	29.7 ± 4.0	.089
Diameter right iliac component − mm	17.4 ± 4.5	17.4 ± 4.5	18.5 ± 4.2	.11
Diameter left iliac component − mm	17.4 ± 5.1	17.3 ± 5.1	18.6 ± 4.3	.13
EVAR re-intervention	410 (22.7)	392 (22.2)	18 (41.9)	.002
Ever smoker	1 203/1 480 (81.3)	1 181/1 443 (81.8)	22/37 (59)	.001
Diabetes	293/1 787 (16.4)	289/1 745 (16.6)	4/42 (9)	.22
Heart disease	736/1 770 (41.6)	716/1 731 (41.4)	20/39 (51)	.21
Cerebrovascular disease	238/1 764 (13.5)	236/1 724 (13.7)	2/40 (5)	.11
Hypertension	1 327/1 774 (74.8)	1 296/1 733 (74.8)	31/41 (76)	.90
Pulmonary disease	434/1 765 (24.6)	426/1 724 (24.7)	8/41 (19)	.45

Data are presented as n (%) or as mean ± standard deviation. AAA = abdominal aortic aneurysm; rAAA = ruptured AAA.

4.1 years (Fig. 3). Patients with subsequent post-EVAR rupture were older (77.6 ± 6.6 years *vs.* 75.2 ± 7.5 years; $p = .036$) and had larger aneurysms at the index EVAR (70.4 ± 16.4 mm *vs.* 63.4 ± 12.7 mm; $p = .009$). Previous EVAR re-interventions were twice as common among patients with post-EVAR rupture (42% *vs.* 22%; $p = .002$). History of smoking was less frequent among post-EVAR rupture patients (60% *vs.* 82%; $p = .001$; Table 1). There was no significant difference in the post-EVAR rupture incidence rate between stent graft types (Table 2).

Analysis of mechanisms

All 51 post-EVAR ruptures in 48 patients were included in the analysis of mechanisms. The mechanism of rupture could be established on review of the CT at post-EVAR rupture in 49 of the 51 cases. In two patients where a CT at the post-EVAR rupture had not been performed, autopsy established the diagnosis. In one of these patients, treatment of a type IA endoleak was planned but had not been performed before rupture, and in one a type IB endoleak was known, but the patient had declined treatment.

Precursors of the subsequent mechanism of post-EVAR rupture had been noted on CT follow up prior to post-EVAR rupture in 16 of 51 (31%) cases. Retrospectively, using the structured protocol, precursors could be identified on follow up CT in 43 of 51 (84%) of the cases.

Type IA endoleak was the mechanism in 20 cases (39%); this was due to sealing zone expansion in 16, migration in three, and graft infection with pseudo-aneurysm formation and rupture in one. Of the patients with type IA endoleak three had a proximal aortic neck < 10 mm on pre-operative CT. Type IB endoleak was the mechanism in 20 cases (39%); this was due to sealing zone expansion in eight, migration in eight, and inadequate seal distally at primary repair, despite long landing zones at the pre-operative CT, in four. In one of the patients with inadequate seal at primary repair this was due to a pre-operative vessel diameter larger than the

selected stent graft. One patient had a type IA and IB failure, and was reported in both groups. Type IIIA/B failure was the mechanism in 11 (22%) cases, nine of these due to component separation, and fabric tear in two cases. In one of 51 (2%) a graft infection led to rupture into the duodenum, with no other apparent mechanism (Table 3).
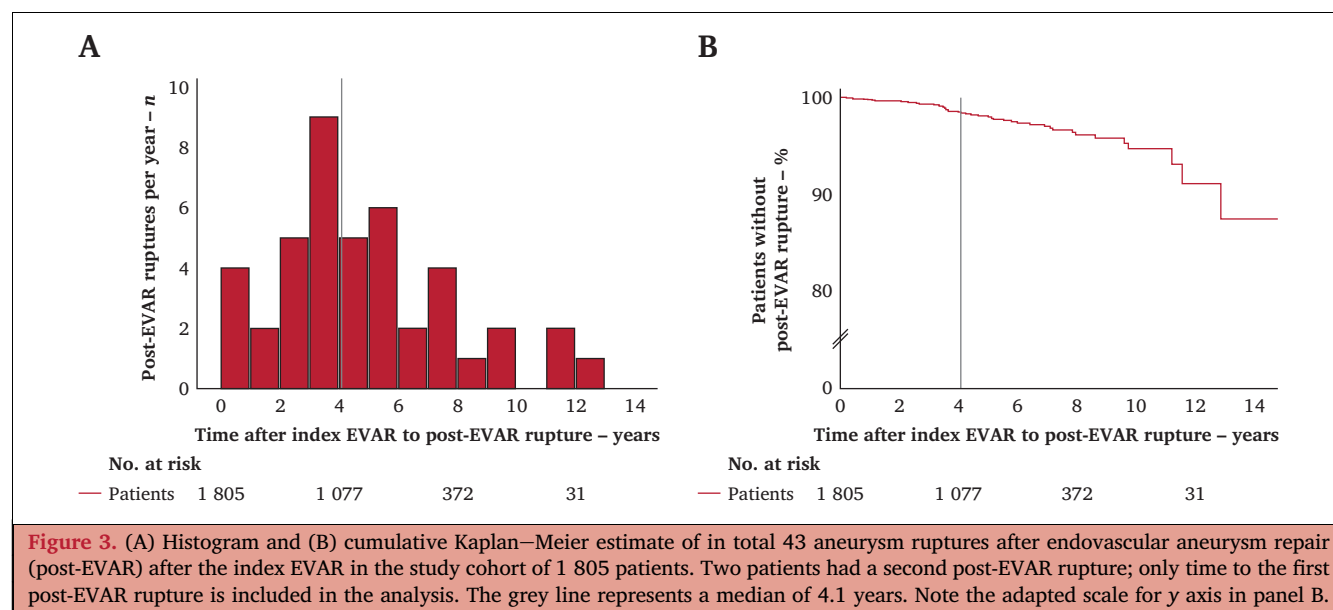
Of the 27 cases not diagnosed on follow up, but with retrospectively identifiable precursors on structured CT review, the mechanism was type IA in 10 (37%), type IB in 11 (41%), and type III in six (22%). No clear pattern of specific stent graft design related risks of post-EVAR rupture was observed (Supplementary Table S1).

Of the 16 cases where precursors of the subsequent mechanism of post-EVAR rupture had been noted but not treated, the reasons were an active decision by the doctor or patient not to treat in eight cases (50%) and rupture occurring before planned treatment in six (37%), while the reason remained unclear in two cases (12%).

Re-interventions, aneurysm sac expansion, and endoleaks

Expanding aneurysm sac between the CT at 30 days and the last follow up CT preceded rupture in 22 of 48 patients (46%). The diameter of the aneurysm sac was unchanged in 10 of 48 (21%) and shrinking in 13 of 48 (27%). Information for three (6%) of the patients was missing: in one case because the patient had been followed up outside the study centres and in two cases because only a follow up CT at 30 days had been performed. Among the 13 patients with shrinking aneurysm between the first and last follow up, five had a late expansion > 5 mm prior to rupture (Supplementary Table S2).

In total, 22 of 48 (46%) of all cases of post-EVAR rupture had an endoleak detectable on either CT ($n = 18$) or ultrasound ($n = 4$) during follow up before the post-EVAR rupture. Of these, there were six type IA, four type IB, 10 type II, one type III, and one of unclear cause. Among the 10 type II endoleaks, the subsequent rupture mechanism was type IA in four, type IB in four, and type

Figure 3. (A) Histogram and (B) cumulative Kaplan−Meier estimate of in total 43 aneurysm ruptures after endovascular aneurysm repair (post-EVAR) after the index EVAR in the study cohort of 1 805 patients. Two patients had a second post-EVAR rupture; only time to the first post-EVAR rupture is included in the analysis. The grey line represents a median of 4.1 years. Note the adapted scale for *y* axis in panel B.

Table 2. Implanted stent graft types, number of post-endovascular aneurysm repair (EVAR) ruptures and incidence rates per 1 000 person years in 1 805 patients treated with EVAR

Stent graft type	EVAR − n	Post-EVAR ruptures	Incidence rate of post-EVAR rupture (95% CI)*
Medtronic Endurant	978	27 (2.8)	4.8 (3.5−7.4)
Cook Zenith Alpha	222	3 (1.4)	4.3 (1.1−11.7)
Cook Zenith Flex	205	9 (4.4)	6.5 (3.2−11.9)
Gore C3	129	1 (0.8)	1.6 (0.8−8.1)
Gore Excluder	123	0	−
Cook Zenith LP	54	0	−
Medtronic Talent	29	2 (6.9)	8.6 (1.4−28.4)
Ovation	21	0	−
Cordis Incraft	12	1 (8.3)	23.3 (0.1−11.5)
Treovance	8	0	−
AFX	3	0	−
Cook custom made 40 mm	2	0	−
Missing information about stent graft type	19	0	−

Data are presented as n (%) unless stated otherwise. CI = confidence interval.
* Incidence rate calculated as the ratio of the total number of post-EVAR ruptures for each stent graft type, divided by the sum of years of follow up for each stent graft type, multiplied by 1 000. Only the first rupture/patient was included.

III in two cases. The proportion with either an expanding aneurysm sac or endoleak before post-EVAR rupture was 29 of 48 (60%) among patients followed either with CT or ultrasound.

Between the CTs at 30 days and one year, six of 48 patients (12%) had a shrinking aneurysm, three of 48 patients (6%) had an expansion, and 34 of 48 patients (71%) were unchanged. Two patients ruptured before the follow up CT and three patients had no CT at one year. Of all 48 patients with post-EVAR rupture, 19 of 48 (40%) had a previous graft related re-intervention (Supplementary Table S2).

Treatment of post-EVAR rupture

Of 51 cases of post-EVAR rupture, 33 (65%) were treated, 29 (88%) by endovascular methods. The most common intervention was distal extension (*n* = 15), followed by

relining (*n* = 8) and proximal extension (*n* = 6). Two of these patients were treated with a combination of proximal and distal extension. In four patients treated by open repair three had a proximal (type IA) failure and one had a graft infection with a verified aortoduodenal fistula (Table 3).

Most untreated post-EVAR rupture patients (*n* = 16/18) were diagnosed in hospital; two were diagnosed post mortem.

Mortality rate of post-EVAR rupture

Overall, 30 day mortality following post-EVAR rupture was 24 of 51 (47%) and among patients undergoing re-intervention seven of 33 (21%). Patients treated for rupture with distal extension or relining due to type IB and III endoleak had a 30 day mortality rate of 9% (*n* = 2/23), and those treated for type IA endoleak had a 30 day mortality rate of 56% (*n* = 5/9; Table 3).

Table 3. Mechanisms and precursors, treatment, and death among 51 cases of rupture after endovascular aneurysm repair (post-EVAR) in 48 patients

Mechanism	Post-EVAR ruptures	Precursor (*n*)	Treatment (*n*)	30-day mortality
All	51 (100)	All (51)	All (51)	Overall: 24/51 (47)
			All treated (33)	All treated: 7/33 (21)
Type IA*	20/51 (39)	Sealing zone expansion (16*)	No treatment (10*)	Overall: 15/20 (75)
			Open repair (2)	All treated: 5/9 (56)
			Proximal extension (4)	Untreated: 10/11 (91)
				Open repair: 2/3 (66)
				Proximal extension: 3/6 (50)
		Migration (3)	No treatment (1)	
			Open repair (1)	
			Proximal extension (1)	
		Graft infection (1)	Proximal extension (1)	
Type IB*	20/51 (39)	Sealing zone expansion (8*)	No treatment (2*)	Overall: 5/20 (25)
			Distal extension (6)	All treated: 0/15 (0)
				Untreated: 5/5 (100)
				Distal extension: 0/15 (0)
		Migration (8)	No treatment (2)	
			Distal extension (6)	
		Inadequate seal at primary EVAR (4)	No treatment (1)	
			Distal extension (3)	
Type III	11/51 (22)	Component separation (9)	No treatment (3)	Overall: 5/11 (45)
			Relining (6)	All treated: 2/8 (25)
				Untreated: 3/3 (100)
				Relining: 2/8 (25)
		Fabric tear (2)	Relining (2)	
Graft infection	1/51 (2)	No precursor identified[†] (1)	Open repair (1)	All treated 0/1 (0)
				Open repair 0/1 (0)

Data are presented as *n* (%) unless stated otherwise.
* One patient had mechanism IA and IB due to sealing zone expansion proximally and distally, and was untreated. This patient is presented in both groups.
[†] One patient had a graft infection leading to an aorto-duodenal fistula, with no other apparent precursor identified.

DISCUSSION

The most common mechanisms of post-EVAR rupture were distal and interconnection failures. Using a structured protocol for review of follow up CTs precursors of the subsequent rupture mechanism could be identified in the majority of patients. The incidence of post-EVAR rupture has remained unchanged over time post-EVAR and the total mortality is high.

The use of ultrasound as a follow up modality is increasing, and less intense follow up programmes are being implemented in many institutions.[14] These changes have continued, despite an increasing body of evidence suggesting that the incidence of post-EVAR rupture remains unchanged, even in the era of the latest generation stent grafts.[12] Regardless of the modality used in post-EVAR follow up, it is worrying that a large proportion of patients with post-EVAR rupture had precursors that were not identified. The results of this study indicate that, with the use of a structured protocol for CT review, important information regarding landing zones, migration, and graft integrity can be identified. This increases the detection of precursors of post-EVAR rupture, a requisite for preemptive treatment. Most importantly, if a CT is performed during follow up, all available information should be extracted in order to optimise EVAR durability.

An important finding of the present study is that aneurysm sac expansion or visible endoleak is not always present

before post-EVAR rupture. It is worth noting that endoleaks may not be detectable, even in cases with complete loss of seal (Supplementary Fig. S2). Thus, their absence cannot exclude the risk of post-EVAR rupture.

In most cases, the rupture mechanism was clearly visible on the rupture CT with massive endoleak and total loss of attachment, in some cases confirmed by operative and autopsy findings. Most post-EVAR ruptures were caused by distal and interconnection failures due to expanding common iliac vessels, stent graft migration, and component separation. Notably, of the 20 cases of post-EVAR rupture caused by migration, 17 concerned the distal and interconnection points of EVAR grafts. Biomechanical studies have previously shown that angulated stent grafts and grafts with a large iliac diameter are subjected to higher displacement forces, which might explain the relatively large proportion of migration at graft interconnections and distal landing zones.[17] Sealing zone expansion and migration leading to distal and interconnection failures are precursors of post-EVAR rupture that are treatable prophylactically, with low morbidity if detected,[18] and are also the ones most successfully treated at post-EVAR rupture. The follow up modality should have the capacity to detect these precursors, making CT the most suitable option.

In this study, type II endoleak was not an independent rupture mechanism in any patient. However, it remains unclear whether the type II endoleaks present in 10

patients prior to rupture contributed to the development of the subsequent mechanism of rupture.

Inadequate seal at the primary EVAR is one possible cause for late failure, as mentioned in the ESVS guidelines.[14] In the present study this was noted as the cause of the subsequent rupture mechanism in only four of 48 patients with post-EVAR rupture. In addition, review of the preoperative CT showed that another three of 48 patients did not have the anatomical prerequisites for treatment within the IFU.

The mean follow up time, as well as median time to rupture, in the present study was in the upper range in comparison to other studies. Importantly, the risk of post-EVAR rupture does not seem to decline over time after the primary EVAR. In this study, the vast majority of post-EVAR ruptures could not have been predicted from the findings on the first post-EVAR CT only. These observations motivate long term monitoring in order to detect precursors and offer prophylactic re-intervention.

The retrospective design has the inherent limitation of selection bias and non-registered confounders. Identification of patients with a primary EVAR was made through the Swedvasc registry, in which the registration of primary AAA procedures is high, but some cases may still have been missed. Data collection, including the detection of post-EVAR ruptures, was done manually through review of patient hospital records, strengthening the detection of post-EVAR ruptures. This might explain the higher incidence rate found in the present study *vs.* a recently published population based study on the same topic.[12] However, the true incidence of post-EVAR rupture will most likely be higher due to an unknown number of patients dying of post-EVAR rupture without being diagnosed. The autopsy rate was less than 10% in Sweden during the study period.[19]

The participation of several centres representing different levels of specialisation and the long follow up present other advantages of this study in terms of its generalisability.

Review of CT scans was standardised and all measurements recorded in a structured protocol allowing for visualisation of continuous migration, expansion, and other complications. However, the review was retrospective, with knowledge of the resulting rupture mechanism, clearly deviating from the situation of prospective EVAR follow up. No comparative measurements were made in the group of patients with no post-EVAR rupture. Further studies are needed to evaluate the effect of the protocol in an unselected cohort.

There were considerable variations in intervals, length, and modalities of follow up, which limits the possibility to account for follow up compliance and of reporting the occurrence of precursors over time.

Conclusion

Most precursors of post-EVAR rupture are under diagnosed in current clinical practice. The use of a structured protocol for CT review may increase the detection rate of precursors of post-EVAR rupture, allowing for prophylactic treatment. The most important precursors and mechanisms of rupture are distal seal failures and component separations, which can be treated with low mortality.

CONFLICTS OF INTEREST

None.

APPENDIX A. SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ejvs.2022.04.042.

REFERENCES

1 Paravastu SC, Jayarajasingam R, Cottam R, Palfreyman SJ, Michaels JA, Thomas SM. Endovascular repair of abdominal aortic aneurysm. *Cochrane Database Syst Rev* 2014;**1**:CD004178.
2 Stather PW, Sidloff D, Dattani N, Choke E, Bown MJ, Sayers RD. Systematic review and meta-analysis of the early and late outcomes of open and endovascular repair of abdominal aortic aneurysm. *Br J Surg* 2013;**100**:863−72.
3 Columbo JA, Ramkumar N, Martinez-Camblor P, Kang R, Suckow BD, O'Malley AJ, et al. Five-year reintervention after endovascular abdominal aortic aneurysm repair in the Vascular Quality Initiative. *J Vasc Surg* 2020;**71**:799−805.
4 Li B, Khan S, Salata K, Hussain MA, de Mestral C, Greco E, et al. A systematic review and meta-analysis of the long-term outcomes of endovascular versus open repair of abdominal aortic aneurysm. *J Vasc Surg* 2019;**70**:954−69.
5 Giles KA, Landon BE, Cotterill P, O'Malley AJ, Pomposelli FB, Schermerhorn ML. Thirty-day mortality and late survival with reinterventions and readmissions after open and endovascular aortic aneurysm repair in Medicare beneficiaries. *J Vasc Surg* 2011;**53**:6−12.
6 Wyss TR, Brown LC, Powell JT, Greenhalgh RM. Rate and predictability of graft rupture after endovascular and open abdominal aortic aneurysm repair: data from the EVAR Trials. *Ann Surg* 2010;**252**:805−12.
7 Powell JT, Sweeting MJ, Ulug P, Blankensteijn JD, Lederle FA, Becquemin JP, et al. Meta-analysis of individual-patient data from EVAR-1, DREAM, OVER and ACE trials comparing outcomes of endovascular or open repair for abdominal aortic aneurysm over 5 years. *Br J Surg* 2017;**104**:166−78.
8 Candell L, Tucker LY, Goodney P, Walker J, Okuhn S, Hill B, et al. Early and delayed rupture after endovascular abdominal aortic aneurysm repair in a 10-year multicenter registry. *J Vasc Surg* 2014;**60**:1146−53.
9 Antoniou GA, Georgiadis GS, Antoniou SA, Neequaye S, Brennan JA, Torella F, et al. Late rupture of abdominal aortic aneurysm after previous endovascular repair: a systematic review and meta-analysis. *J Endovasc Ther* 2015;**22**:734−44.
10 Rajendran S, May J. Late rupture of abdominal aortic aneurysm after endovascular repair. *J Vasc Surg* 2017;**65**:52−7.
11 Huang Y, Gloviczki P, Duncan AA, Kalra M, Oderich GS, Fleming MD, et al. Maximal aortic diameter affects outcome after endovascular repair of abdominal aortic aneurysms. *J Vasc Surg* 2017;**65**:1313−22.

12 Andersson M, Talvitie M, Benson L, Roy J, Roos H, Hultgren R. A population-based study of post-endovascular aortic repair rupture during 15 years. *J Vasc Surg* 2021;**74**:701—10.

13 Chaikof EL, Dalman RL, Eskandari MK, Jackson BM, Lee WA, Mansour MA, et al. The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm. *J Vasc Surg* 2018;**67**:2—77.

14 Wanhainen A, Verzini F, Van Herzeele I, Allaire E, Bown M, Cohnert T, et al. Editor's Choice — European Society for Vascular Surgery (ESVS) 2019 Clinical Practice Guidelines on the Management of Abdominal Aorto-iliac Artery Aneurysms. *Eur J Vasc Endovasc Surg* 2019;**57**:8—93.

15 Bergqvist D, Mani K, Troëng T, Wanhainen A. Treatment of aortic aneurysms registered in Swedvasc: development reflected in a national vascular registry with an almost 100% coverage. *Gefasschirurgie* 2018;**23**:340—5.

16 von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *Lancet* 2007;**370**: 1453—7.

17 Roos H, Tokarev M, Chernoray V, Ghaffari M, Falkenberg M, Jeppsson A, et al. Displacement forces in stent grafts: influence of diameter variation and curvature asymmetry. *Eur J Vasc Endovasc Surg* 2016;**52**:150—6.

18 Naughton PA, Garcia-Toca M, Rodriguez HE, Keeling AN, Resnick SA, Morasch MD, et al. Endovascular treatment of delayed type 1 and 3 endoleaks. *Cardiovasc Intervent Radiol* 2011;**34**:751—7.

19 Socialstyrelsen. Tabeller — Dödsorsaker 2017. Available at: https://www.socialstyrelsen.se/en/statistics-and-data/statistics/ [Accessed 20 May 2022]
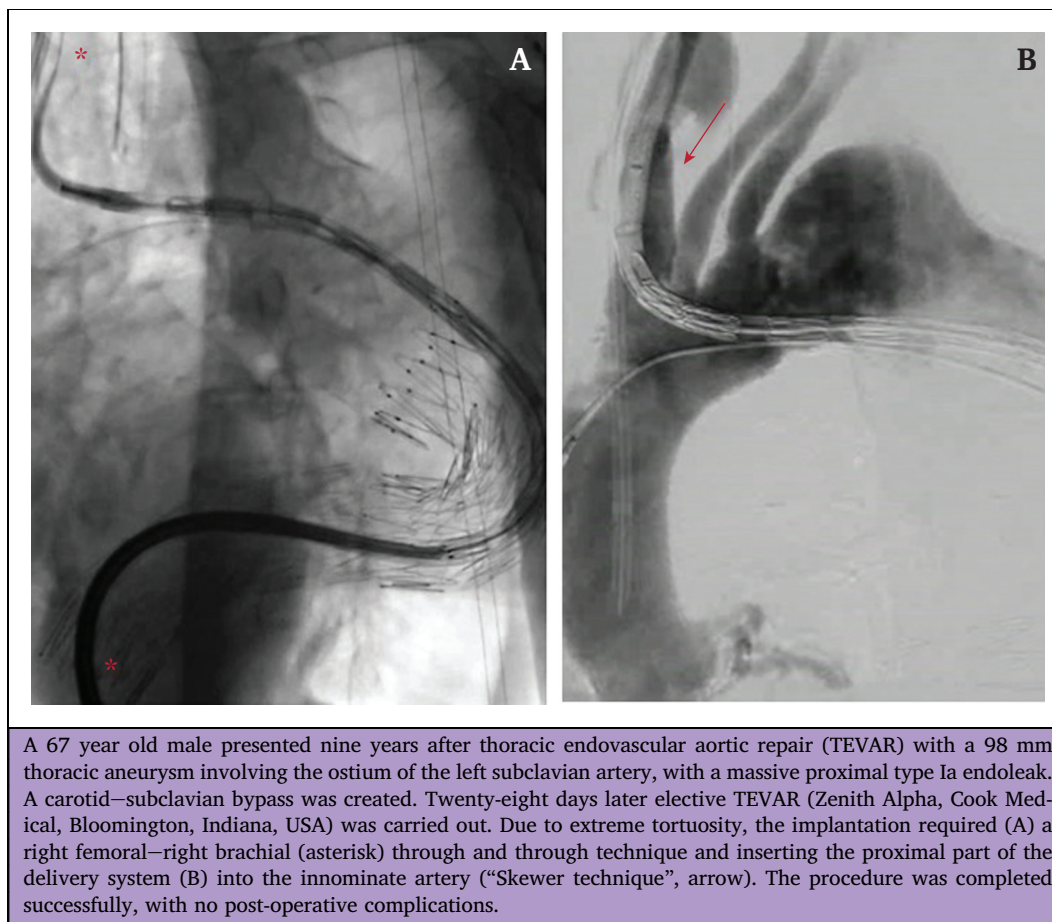
COUP D'OEIL

Through and Through Wire Is the Unique Way to Force Tortuous Anatomy

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A 67 year old male presented nine years after thoracic endovascular aortic repair (TEVAR) with a 98 mm thoracic aneurysm involving the ostium of the left subclavian artery, with a massive proximal type Ia endoleak. A carotid—subclavian bypass was created. Twenty-eight days later elective TEVAR (Zenith Alpha, Cook Medical, Bloomington, Indiana, USA) was carried out. Due to extreme tortuosity, the implantation required (A) a right femoral—right brachial (asterisk) through and through technique and inserting the proximal part of the delivery system (B) into the innominate artery ("Skewer technique", arrow). The procedure was completed successfully, with no post-operative complications.

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