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# Early and Midterm Results after Endovascular Repair of Non-infected Saccular Lesions of the Infrarenal Aorta

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**WHAT THIS PAPER ADDS**

This paper evaluates early and midterm outcomes of endovascular aortic repair (EVAR) in a cohort of patients with non-infected saccular lesion of the abdominal aorta (sl-AA). The analysis reveals that, though an infrequent finding in a large cohort of aortic interventions, sl-AAs present with rupture in up to 8% of cases. In this type of lesion, EVAR was safe, with a low re-intervention rate and a high rate of freedom from aorta related death, while overall survival was significantly impaired by older (> 70 years) age and coronary artery disease.

**Objective:** The aim was to report short and midterm outcomes of a cohort of consecutive patients treated by endovascular aortic repair (EVAR) for saccular lesion of the abdominal aorta (sl-AA).
**Methods:** This was a multicentre, retrospective, financially unsupported physician initiated, observational cohort study that involved tertiary referral from Italian hospitals. For this study, between January 2010 and December 2020, only those patients treated by EVAR for non-infected sl-AA, namely blister/ulcer like projection and/or penetrating aortic ulcer, were analysed. Primary outcomes of interest were overall survival and freedom from aorta related mortality (ARM).
**Results:** The final cohort included 120 of 3 982 eligible aortic lesions. There were 103 (85.8%) males and 17 (14.2%) females. The median age was 76 years (interquartile range [IQR] 69, 80). Rupture on admission was observed in 10 (8.3%) cases. Early ($\leq$ 30 days) death occurred in two (1.7%) patients. There were five (4.2%) complications requiring surgical re-intervention (iliac limb occlusion $n = 4$; groin haematoma, $n = 1$). The median duration of follow up was 20 months (IQR 4, 59.5): the estimated overall survival was 85.5% (standard error [SE] 0.035; 95% confidence interval [CI] 77.3 – 91.1) at 12 months, 78.7% (SE 0.044; 95% CI 69.0 – 86.0) at 36 months, and 74% (SE 0.050; 95% CI 63.2 – 82.5) at 60 months. Only one (0.8%) patient required aortic re-intervention during follow up because of a late endograft infection. The estimated freedom from ARM was 96% (SE 0.050; 95% CI 90.3 – 98.2) at 36 and 60 months. Cox's regression analysis identified that death was associated with age > 70 years (hazard ratio [HR] 1.10; 95% CI 1.04 – 1.17, $p = .001$), and coronary artery disease (HR 1.14; 95% CI 1.04 – 1.26, $p = .006$).
**Conclusion:** EVAR for sl-AA proved to be safe and effective. The mortality rate was low for a group of patients known to be at high risk from open repair, and EVAR remained stable with no ARM during midterm follow up, and an acceptably low 0.8% endograft related re-intervention rate.

Keywords: Aortic blister, Focal aortic pathology, Saccular aneurysm, Penetrating aortic ulcer
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## INTRODUCTION

Management of saccular lesions of the infrarenal abdominal aorta (sl-AA) remains controversial.[1,2] A considerable overlap in definition has been reported across several series over the last two decades: saccular aneurysm, blister, ulcer like projection, pseudoaneurysm, penetrating aortic ulcer are all eponyms that have been used to define a

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focal, saccular lesion with diameter enlargement of the aorta.[3–8]

Unlike fusiform abdominal aortic aneurysms, discrepancy in definition and information regarding the natural history of sl-AAs has resulted in not having uniformly accepted practice guidelines for their management.[9,10] Notwithstanding these shortcomings, sl-AAs have been considered adequate anatomic targets for endovascular aortic repair (EVAR).[9,10] Large natural history and/or size progression of the aortic lesion studies have not reported consistent and homogeneous findings; also, large studies on outcomes of EVAR have not been published to date.[11–13] Data on results after EVAR are mainly characterised by small cohorts, and heterogeneity of sl-AA location and follow up data.[14–21]

The aim of this study was to describe a large cohort of patients with sl-AA managed by EVAR, and to report an analyses of major outcomes.

## MATERIALS AND METHODS

### *Study cohort*

This was a multicentre, retrospective, financially unsupported, physician initiated, observational cohort study that involved tertiary referral from Italian hospitals. A checklist of items followed the STROBE statement (Supplementary Appendix).[22] Clinical data collected from each centre were merged in a single database, recorded in a dedicated database, and analysed retrospectively. For this study, between 1 January 2010 and 31 December 2020, only those patients treated by EVAR for sl-AA were identified (Fig. 1). Information collected included demographics, comorbidities, morphological characteristics of the aortic lesion, type of intervention, and endograft (EG), as well as post-operative events (complications, death, re-intervention) during hospitalisation and follow up. Informed consent was obtained from all individual participants included in the study. Consent for publication was not required by the local Institutional Review Board, in accordance with the Italian National Policy for Privacy Act on retrospective analysis of anonymised data.

### *Operative details*

All interventions were performed according to the national guidelines of the Italian Society for Vascular and Endovascular Surgery, consistent with the most recent clinical practice guidelines on the management of abdominal aorto-iliac artery aneurysms of the European Society for Vascular Surgery (ESVS).[9,23] For patients included in this cohort, device selection and operative planning were left to the surgeon's judgement, and made following the manufacturer's instructions. The follow up protocol included at least computed tomography angiogram (CTA) at 30 days and at one year, for all patients at each centre. Thereafter, contrast enhanced ultrasonography or duplex ultrasound surveillance was performed annually; CTA use was individualised based on aortic growth, device failure, or endovascular re-operation if feasible.

### *Definitions and primary outcomes*

In the definition of sl-AA, all non-infected, focal lesions of the infrarenal aorta were included, namely blister/ulcer like projection and/or penetrating aortic ulcer. Intramural haematoma, ulcerated non-enlarged aortic plaque, and infected lesions were excluded. An infected lesion was defined by the combination of different clinical and morphological criteria such as clinical presentation (pain, fever $\geq$ 38°C, sepsis, and concomitant infection), laboratory tests (elevation of inflammatory markers like C reactive protein and white blood cells, and positive cultures), and computed

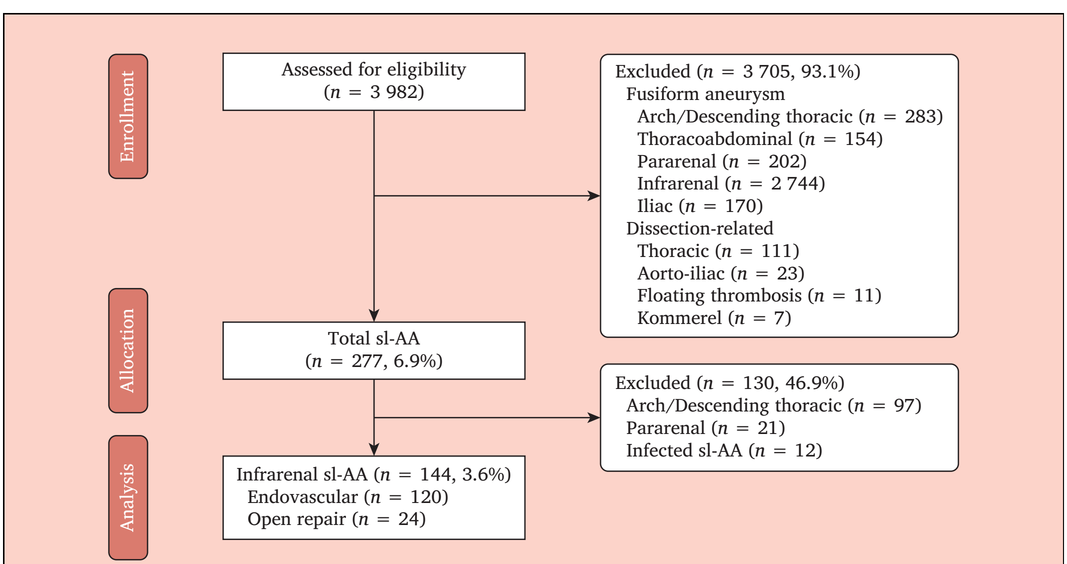



**Figure 1.** Consolidated Standards of Reporting Trials (CONSORT) study flow diagram of endovascular repair for non-infected saccular lesions of the abdominal aorta (sl-AA) during the study period of January 2010 to December 2020 in Italy.

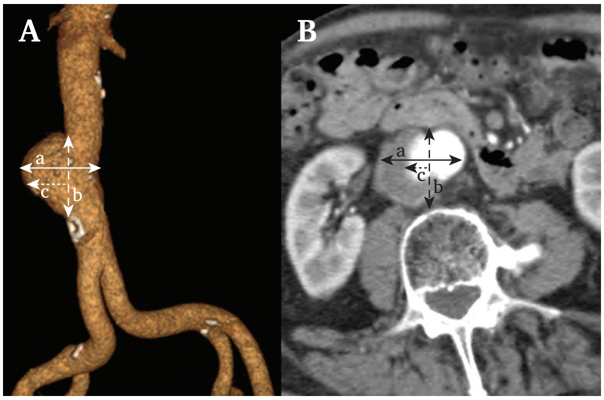



Figure 2. Measurement of aortic ulcer from (A) 3D reconstruction or (B) axial section of computed tomography angiography image, where A corresponds to maximum aortic diameter at ulcer site, B to length of intimal defect at ulcer site, and C to ulcer depth.

tomography findings on the abdominal part of the aorta (mainly multilobular eccentric lesions evolving from otherwise "healthy"/non-atherosclerotic aortas, peri-aortic gas, and peri-aortic soft tissue mass). Saccular lesion dimensions were measured according to maximum depth from the lumen into the aortic wall, maximum width at entry site from the lumen, and maximum length of the intramural component of the lesion itself (Fig. 2).[12] Medical comorbidity grading system and correlated operative risk profile, as well as operative outcomes, were defined according to the Society for Vascular Surgery (SVS) and ESVS practice guidelines on the care of patients with an AAA.[9,10,24] Primary technical success was defined according to the SVS reporting standards for endovascular aortic aneurysm repair on an intent to treat basis, including the successful introduction and deployment of the device in the absence of surgical conversion or death, type I or III endoleaks, or graft limb obstruction.[25] The follow up index (FUI) described follow up completeness at a given study end date as a ratio between the investigated and the potential follow up period.[26] Anatomic location of the sl-AA was classified in three levels: "type A", identifying a lesion that was located above the inferior mesenteric artery, in the proximal section of the infrarenal aorta, but presenting with $\geq$ 15 mm proximal aortic neck, to identify sl-AA generally not amenable of standard EVAR according to the instructions for use of the available devices; "type B" when the lesion was located at the level of the inferior mesenteric artery; "type C" when the lesion was located between the inferior mesenteric artery and the aorto-iliac bifurcation (Fig. 3). According to the Kidney Disease Improving Global Outcomes, acute kidney injury (AKI) was defined as an increase in serum creatinine (SCr) $\geq$ 0.3 mg/dL within 48 hours or an increase in SCr to $\geq$ 1.5 times baseline known or presumed to have occurred within seven days, or a urine volume of $<$ 0.5 mL/kg/h for six hours. AKI severity stage was classified according to the Acute Kidney Injury Network (AKIN) criteria.[27] Aneurysm sac shrinkage was defined a diameter reduction $\geq$ 1 cm from the pre-operative diameter. Through December 2020, information on aorta related re-intervention, vital status, and date of death of individual patients were validated by death certificates, electronic charts managed by the regional healthcare system, through General Practitioner interview, or certified data from Emergency Department admission. For this specific study, primary outcomes of interest were overall survival and freedom from aorta related mortality (ARM). Secondary outcomes were post-operative complications, and freedom from aortic re-intervention. Major outcomes were stratified according to the anatomic level of the sl-AA.

### *Statistical analysis*

Clinical data were collected in a prospective manner at each centre; thereafter, they were merged in a single database, recorded, and tabulated in Microsoft Excel (Microsoft Corp, Redmond, WA) and analysed retrospectively. To ensure correct data recording and limit missing records, an audit of the database was conducted, and each centre was asked to provide absent variables. Statistical analysis was performed with SPSS, release 26.0 for Windows (IBM SPSS Inc.; Chicago, IL). Categorical variables are presented as frequencies and percentages. Continuous variables were tested for normality using the Shapiro—Wilk test. Variables that were

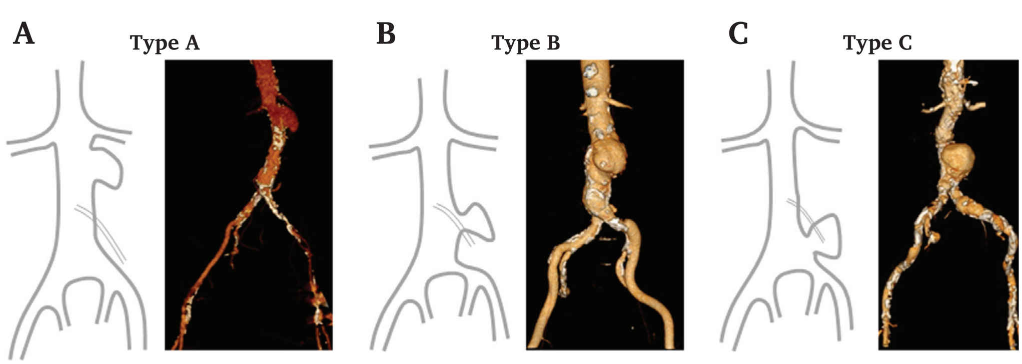



Figure 3. Location and extent of the saccular lesions of the abdominal aorta, where (A) type A lesion is located above the inferior mesenteric artery (IMA) but presenting with $\geq$ 15 mm proximal aortic neck; (B) type B lesion is located at the level of the IMA; and (C) type C lesion is located between the IMA and the aorto-iliac bifurcation.

normally distributed are presented as mean ± standard deviation and range; otherwise, they are presented as median and interquartile range (IQR). Categorical variables were analysed using a $\chi^2$ test or Fisher's exact test when appropriate. The unpaired Student's T test was used for normally distributed continuous variables; otherwise, the Mann—Whitney *U* test was adopted. The Kruskall—Wallis honest significance test was used for single step multiple comparisons to find significant differences among medians. Univariable analysis was used to identify potential predictors of post-operative EG related complication. Associations that yielded $p < .20$ on univariable analysis were then included in a binary logistic regression analysis using Wald's forward stepwise model. The strength of the association of variables with EG related complications was estimated by calculating the odds ratio (OR) and 95% confidence interval (95% CI: significance criteria 0.20 for entry, 0.05 for removal). Model discrimination was evaluated using the area under the receiver operating characteristic curve (AUROC), with $\geq .7$ being considered significantly accurate. Cox's regression analysis was used to assess the strength of the association of covariables with death during follow up. Initially, univariable analysis was used to identify potential predictors of death using the Kaplan—Meier estimator and log rank test for each categorical covariable. Associations that yielded $p < .20$ on univariable analysis were then included in a forward regression analysis, and the strength of association between covariables and death was estimated by calculating the hazard ratio (HR) and 95% CIs. Additionally, to assess which covariables were associated with ARM, a proportional hazards model, as proposed by Fine and Gray was implemented, to properly consider the presence of competitive risks.[28] All survival analyses were reported as percentage ± standard error (SE) with 95% CI. All reported *p* values were two sided; $p < .050$ was considered statistically significant.

## RESULTS

### *Clinical profile, anatomical characteristics, and surgical details*

The final cohort included 120 (3.0%) patients from all the aortic interventions. They were non-consecutive since 24 (16.7%) received open repair due to anatomical features not amenable to endovascular solutions according to the instructions for use of the available standard devices (e.g., juxtarenal/pararenal lesion), or due to the "non-viability" of both iliac axes. There were 103 (85.8%) males and 17 (14.2%) females. The median age was 76 years (IQR 69, 80). The median SVS score was 6 (IQR 3, 11). Rupture on admission was observed in 10 (8.3%) cases. Demographic data, comorbidities, and operative risk profile of the cohort are shown in Table 1. Classification of sl-AA subtype according to zone location was as follows: "type A" in 24 (20.0%) cases, "type B" in 72 (60.0%), and "type C" in 24 (20.0%) cases. The morphological characteristics of the aortic lesions are reported in Table 2: in summary, there were no significant differences except for "type C" lesions, which presented with the greatest depth ($p = .028$). Rupture at presentation was observed more frequently in sl-AAs > 45 mm (6/27, 22% *vs*. 3/79, 3.8%; OR 7.2, $p = .008$).

### *Operative details*

Primary technical success was obtained in all but two cases (98.3%). There were two deaths (1.7%) in patients who presented with rupture (both in "type B", $p = .51$). No

**Table 1. Demographics, comorbidities, and operative risk of the 120 patients treated for non-infected saccular abdominal aortic lesions at different anatomic locations: type A, above the inferior mesenteric artery (IMA), but presenting with ≥ 15 mm proximal aortic neck; type B, at the level of the IMA; type C, between the IMA and the aorto-iliac bifurcation**

| Covariate | Total cohort ($n = 120$) | Type A ($n = 24$) | Type B ($n = 72$) | Type C ($n = 24$) | *p* |
|---|---|---|---|---|---|
| *Demographics* | | | | | |
| Male | 103 (85.8) | 21 (87.5) | 62 (86.1) | 20 (83.3) | .91 |
| Age − y | 76 (69–80) | 75 (70–81) | 75.5 (67.5–79) | 76 (69.5–80) | .99 |
| *Comorbidity** | | | | | |
| Hypertension | 103 (85.8) | 20 (83.3) | 64 (88.9) | 19 (79.2) | .46 |
| Dyslipidaemia | 55 (45.8) | 12 (50.0) | 27 (37.5) | 16 (66.7) | .041 |
| Coronary artery disease | 54 (45.0) | 14 (58.3) | 28 (38.9) | 12 (50.0) | .22 |
| Chronic obstructive pulmonary disease | 35 (29.2) | 6 (25.0) | 24 (33.3) | 5 (20.8) | .45 |
| Chronic kidney disease | 21 (17.5) | 5 (20.8) | 12 (16.7) | 4 (16.7) | .89 |
| Atrial fibrillation | 18 (15.0) | 6 (25.0) | 8 (11.1) | 4 (16.7) | .25 |
| Obesity | 11 (9.2) | 2 (8.3) | 7 (9.7) | 2 (8.3) | .97 |
| Diabetes | 7 (5.8) | 3 (12.5) | 2 (2.8) | 3 (12.5) | .095 |
| *Risk factor* | | | | | |
| Previous cardiac surgery | 22 (18.3) | 9 (37.5) | 9 (12.5) | 4 (16.7) | .023 |
| Previous vascular surgery | 12 (10.0) | 5 (20.8) | 6 (8.3) | 1 (4.2) | .12 |
| Thoracic localisation | 5 (4.2) | 2 (8.3) | 1 (1.4) | 2 (8.3) | .18 |
| Rupture | 10 (8.3) | 2 (8.3) | 7 (9.7) | 1 (4.2) | .70 |
| SVS score | 6 (3–11) | 7.5 (3–11.5) | 5.5 (3–9.75) | 6.5 (3–12) | .42 |

Data are presented as *n* (%) or median (interquartile range). SVS = Society for Vascular Surgery.
* *J Vasc Surg* 2002;**35**:1061–6.

**Table 2.** Measurements and dimensions of 120 non-infected saccular abdominal aortic lesions at different anatomic locations: type A, above the inferior mesenteric artery (IMA), but presenting with ≥ 15 mm proximal aortic neck; type B, at the level of the IMA; type C, between the IMA and the aorto-iliac bifurcation

| Covariate | Overall ($n$ = 120) | Type A ($n$ = 24) | Type B ($n$ = 72) | Type C ($n$ = 24) | $p$ |
|---|---|---|---|---|---|
| Maximum diameter – mm | 38 (31–45) | 38 (35–50) | 38 (30–43) | 37 (31.5–47.5) | .98 |
| Length – mm | 31 (21–42) | 28 (20–39.5) | 31 (20–40) | 34 (24.5–50) | .84 |
| Depth – mm | 17 (12–23) | 18 (14–29.5) | 16 (11–22) | 21 (15–25) | .028 |
| Neck angle – degrees | 18 (13–30) | 23 (17.5–35.5) | 17 (13–30) | 22 (12.5–32) | .053 |

Data are presented as median (interquartile range).

immediate conversion to open surgery was required. A bifurcated EG was used in 61 (51.3%) cases, a straight tube graft in 52 (43.4%: aortic cuff $n$ = 49, balloon expandable aortic stent graft $n$ = 3), and an aorto-uni-iliac (AUI) configuration in six (5.3%), as reported in Table 3. The mean EG diameter was 24 ± 2 mm (range 22 – 28). In 19 (15.8%) cases, an additional intra-operative procedure was performed (Table 4). The mean intervention duration was 137 ± 75 minutes (range 29 – 285): 11 (9.2%) patients required blood transfusion (median 1 unit; IQR 1, 2.5).

### Early (< 30 days) results

The overall complication rate (4.2% *vs*. 4.2% *vs*. 16.7%, $p$ = .090) and severity grade ($p$ = 0.31) was not different among the types of sl-AA. There were nine complications in eight (6.7%) patients: five (62.5%) required surgical re-intervention for iliac limb occlusion ($n$ = 4) and groin haematoma ($n$ = 1). However, limb occlusion always occurred in "type C" lesions ($p$ = .019). Other complications were as follows: AKI in two (1.7%) cases requiring transient haemodialysis in one (AKIN stage 3: 0.8%), and urinary tract infection in the other two (1.7%). Binary logistic regression analysis confirmed only that early limb occlusion was associated with "type C" lesions (OR 12.2, 95% CI 1.25 – 117.6, $p$ = .031). The median hospitalisation was five days (IQR 3, 8), with no difference among the type of sl-AA (median, 7 *vs*. 5 *vs*. 6, $p$ = 0.46).

### Late results

No patient was lost at a median follow up of 20 months (IQR 4, 59.5); the median FUI was 0.6 (IQR 0.2, 1). During follow up, 31 (25.8%) patients died: mortality was not different among the sl-AA types (9.7% *vs*. 30.6% *vs*. 25.0%, $p$ = .22). The estimated overall survival was 85.5% (SE 0.035; 95% CI 77.3 – 91.1) at 12 months, 78.7% (SE 0.044; 95% CI 69.0 – 86.0) at 36 months, and 74% (SE 0.050; 95% CI 63.2 – 82.5) at 60 months. Estimated overall survival was not different among the types of sl-AA (log rank $\chi^2$ = 0.3, $p$ = .87). Excluding early iliac limb occlusions, no more EG occlusions were observed. Only one (0.8%) patient required

**Table 3.** Endograft configuration according to the location of non-infected saccular abdominal aortic lesions in 120 patients: type A, above the inferior mesenteric artery (IMA), but presenting with ≥ 15 mm proximal aortic neck; type B, at the level of the IMA; type C, between the IMA and the aorto-iliac bifurcation

| Location | Type of endograft | Total cohort ($n$ = 120) |
|---|---|---|
| Type A | | 24 (20.0) |
| | Tube | 11 |
| | Bifurcated | 13 |
| | AUI + FF | 0 |
| Type B | | 72 (60.0) |
| | Tube | 31 |
| | Bifurcated | 36 |
| | AUI + FF | 5 |
| Type C | | 24 (20.0) |
| | Tube | 9 |
| | Bifurcated | 14 |
| | AUI + FF | 1 |

Data are presented as $n$ (%). AUI + FF = aorto-uni-iliac repair + femorofemoral bypass.

**Table 4.** Intra-operative additional procedure during endovascular repair of non-infected saccular abdominal aortic lesions in different anatomic locations in 120 patients: type A, above the inferior mesenteric artery (IMA), but presenting with ≥ 15 mm proximal aortic neck; type B, at the level of the IMA; type C, between the IMA and the aorto-iliac bifurcation

| Location | Additional procedure | Total cohort ($n$ = 120) |
|---|---|---|
| Type A | | 4 (16.7) |
| | Renal artery stenting | 2 |
| | Kissing stent graft | 1 |
| | Iliac artery stenting | 1 |
| Type B | | 10 (13.9) |
| | Iliac artery stenting | 3 |
| | Kissing balloons | 2 |
| | Femoro-femoral bypass | 2 |
| | SMA stenting | 1 |
| | Renal artery stenting | 1 |
| | Sac embolisation | 1 |
| Type C | | 5 (20.8) |
| | Iliac artery stenting | 2 |
| | Femoro-femoral bypass | 1 |
| | HA embolisation | 1 |
| | IMA embolisation | 1 |

Data are presented as $n$ (%). SMA = superior mesenteric artery; HA = hypogastric artery; IMA = inferior mesenteric artery.

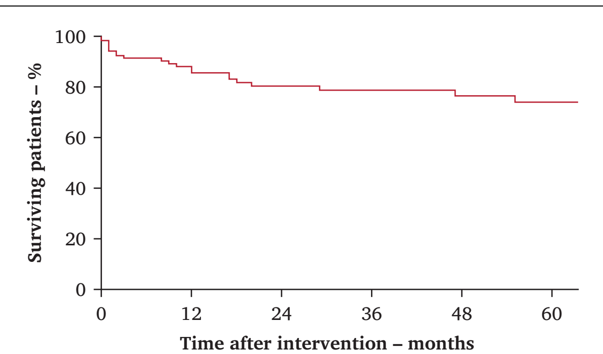
an aortic re-intervention during follow up because of a late EG infection. The overall survival is reported in Figure 4. Multivariable Cox regression analysis included age, hypertension, coronary artery disease, chronic kidney disease, and atrial fibrillation: it identified that mortality risk was associated with age > 70 years (HR 1.10; 95% CI 1.04 − 1.17, $p$ = .001), and coronary artery disease (HR 1.14; 95% CI 1.04 − 1.26, $p$ = .006). When adjusted by type of sl-AA, these covariables were still significantly associated with death. The AUC of this multivariable model was 0.68 (95% CI 56 − 79.5) and is reported in Figure 5. The estimated freedom from ARM was 96% (SE 0.050; 95% CI 90.3 − 98.2) at 60 months; ARM was associated only with rupture on admission (HR 8.1; 95% CI 1.36 − 48.90, $p$ = .022), while cause of death was never observed to be aorta related during follow up. At the last available follow up, no primary EG infections were observed. Endoleak was not detected during follow up. Shrinkage of the sl-AA was observed in 61 (51.7%) cases: no difference was observed between sl-AA types (69.2% *vs*. 52.9% *vs*. 68.2%, $p$ = .31). No predictor was identified to be associated with aneurysm shrinkage.

## DISCUSSION

There are two main findings of the analysis: first, EVAR for sl-AA is acceptably safe, feasible, and effective to protect from ARM at midterm follow up, and second, age and coronary artery disease were the two most impactful predictors of death during follow up.

No study has clarified the natural history or evolution of sl-AA but the presence of sl-AA is widely perceived to be a risk factor for rupture or adverse events, in both clinical series and computational models.[1,2,4,6,11] Using virtual models, Vorp *et al.*[29] demonstrated that aneurysms with the same diameter may not have the same propensity for rupture and that asymmetry of an aortic lesion, in addition to aneurysm diameter, was an important determinant of mechanical wall stress. Similarly, but based on clinical experience, Faggioli *et al.*[4] investigated the impact of aneurysm morphology on the risk of rupture and found that the presence of a focal, saccular lesion such as a blister was the only independent morphological predictor of impending rupture in a small aneurysm. In the largest cohort of saccular aneurysms published so far, Karthaus *et al.*[8] calculated a risk ratio of 15.3 for becoming acutely symptomatic compared with fusiform aneurysms for lesions < 45 mm, but the rupture rate was significantly lower in the saccular group.[6] The results of this experience agree with these findings. Despite the discrepant findings in the literature, it is believed that the unpredictable and alarmingly higher risk of impending rupture described in the literature irrespective of size, as well as their being an ideal morphological target for EVAR, remain two reasonable features to suggest operative intervention.[10,18] However, it is unquestionable that sl-AA behaviour is a pivotal aspect that requires further analysis to clarify the real impact of sizing on the evolution and prognosis of sl-AA.

Previous published studies have suggested that in such populations characterised by advanced age, major comorbidities, and risk factors due to an increased atherosclerotic burden, open aortic surgery may not be considered the best operative indication in all patients.[1,20] Furthermore, also considering the localised nature of sl-AA, EVAR has been considered a valuable treatment option. However, despite these features, EVAR has not been used in large cohorts.[6,8,11] In addition, limitations in the literature are due to the absence of consistent data on EVAR because of the heterogeneity of the analysed cohorts and to the lack of consistent follow




| No. at risk | | | | | |
|---|---|---|---|---|---|
| Patients | 71 | 56 | 44 | 34 | 30 |
| Survival – % | 85.5 | 80.3 | 78.7 | 76.5 | 74 |
| SE | 0.03 | 0.04 | 0.04 | 0.05 | 0.05 |
| 95% CI | 77.3–91.1 | 71.4–86.9 | 69.0–86.0 | 66.0–84.5 | 63.2–82.5 |

**Figure 4.** Cumulative Kaplan−Meier estimate of overall survival of 120 patients treated by endovascular aneurysm repair (EVAR) for non-infected saccular lesion of the abdominal aorta during the study period of January 2010 to December 2020 in Italy.

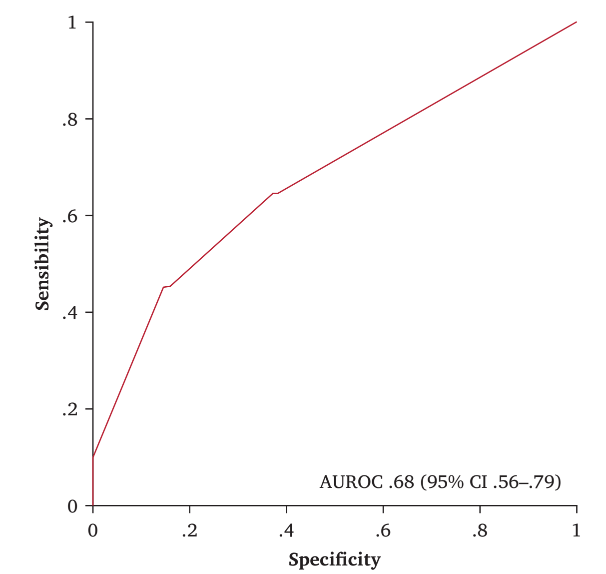



**Figure 5.** Area under the receiver operating curve (AUROC), defining the performance of the multivariable model for overall survival of 120 patients treated by endovascular aneurysm repair for non-infected saccular lesion of the abdominal aorta during the study period of January 2010 to December 2020 in Italy. The model includes age > 70 years and the presence of coronary artery disease.

up.[5,6,8,12,16–21] The 1.7% early mortality rate in this study is better than the 8.7% reported for open surgery.[1,18] Moreover, it compares well with the mean 6.8% of 63 patients from four previous similar EVAR experiences.[17,18,20,21] Although long term data are lacking, what the cohort is adding to the literature is the large number of patients with specific EVAR related results, coupled with reliable data at midterm follow up.

The long term safety and efficacy of EVAR remains an important aspect to be confirmed in this type of lesion, both technically and clinically.[6] Although they may represent an attractive, easy target for EVAR, their peculiar morphological features need careful attention for planning and sizing. Early iliac limb occlusions observed in the subgroup of lesions close to or involving the aortic bifurcation highlight their potentially tricky anatomy. More thorough morphological analyses will help operators to identify the most suitable sl-AA anatomies for EVAR as the definitive first line strategy. The results showed durable positive outcomes: the absence of EVAR failures as well as the absence of ARM beyond the early post-operative stage contribute to indicating the effective durability of EVAR for sl-AA (Table 5).[1,17,18,20,21] In addition, in this analysis, no endoleaks were observed during midterm follow up, but this is not surprising since this also occurred in half of the cases from two of the four series on EVAR so far.[20,21]

Finally, most of the series published so far have underlined the fact that patients with sl-AA are fragile and at risk because of their severe, systemic distribution of atherosclerotic disease.[1,2,6] Mortality was significantly associated with age (> 70 years) and the presence of coronary artery disease: these factors, further emphasise how these patients are severely diseased due to their diffuse atherosclerotic burden. These features need attention, particularly considering the fast progression shown on serial computed tomography scans, especially for abdominal lesions.[1,2,6,12] Currently there is no consensus regarding the threshold diameter for repair. In the light of rapid growth, the high risk already present by the age of 70 years, and the satisfactory durability of endovascular repair over time, the use of EVAR may be considered a reasonable alternative for early treatment of sl-AAs.

### Limitations

This study has several limitations. First, the analysis is retrospective. Large databases rely solely on accurate site reporting, but they have potential high value due to sample size. Thus, it is possible that the investigators might not have identified all aorto-iliac morphological variables. However, missing data were not defaulted to negative, and denominators reflect only reported cases: for the 120 patients, 7 080 overall data items were collected amongst 59 variables, with an overall missing data rate of 0.7%. Moreover, multiple review auditing was performed by the leading author at each centre to limit major inconsistencies. Second, there was sampling bias in those patients undergoing open repair who were not included for comparison. Lastly, while an attempt was made to correct for potential confounders using multivariable analyses, the small number of patients and events means results of multivariable models are not generalisable, and the absence of statistically significant differences could reflect a type II error. Notwithstanding all these limitations that may limit generalisability, the data compare well with the available literature owing to the consistency of follow up and data validation by official health documents.

This study is one of the largest real world experiences reporting both short and midterm outcomes of EVAR for non-infected sl-AAs. the EVAR mortality rate was satisfactorily low for a group of patients known to be at high risk of open surgical repair. Despite early iliac limb occlusions, endovascular reconstruction remained effective at midterm follow up with no ARM or very low need for re-intervention. Future refinement should pay attention to the correlation between aorto-iliac anatomy and EG configuration to identify the best type of EVAR reconstruction or to adapt new EG configurations specifically designed for this subgroup of aortic lesions.

**Table 5. Literature summary reporting on non-infected saccular lesions of abdominal aorta in all cohorts and in cohorts of > 15 cases, treated by endovascular aneurysm repair (EVAR)**

| Author | Year | Cases – *n* | Mean age – y | Males | Rupture | In hospital death | Overall survival | Mean follow up – mo | ARM | Re-intervention |
|---|---|---|---|---|---|---|---|---|---|---|
| *Mixed cohorts* | | | | | | | | | | |
| Lindblad *et al.*[5] | 1987–2007 | 11† | 72* | 84* | 0 | 0 | N.R. | N.R. | N.R. | N.R. |
| Nathan *et al.*[6] | 2003–2009 | 120 | 73* | 60* | 17.9* | N.R. | N.R. | N.R. | N.R. | N.R. |
| Karthaus *et al.*[8] | 2016–2018 | 313 | 74* | 82* | 8.7* | 3.2* | N.R. | N.R. | N.R. | N.R. |
| Shang *et al.*[11] | 2003–2011 | 22 | 73* | 86* | 8.8* | N.R. | N.R. | N.R. | N.R. | N.R. |
| Gifford *et al.*[12] | 1998–2012 | 8† | 76* | 62* | N.R. | 0* | N.R. | N.R. | N.R. | N.R. |
| *EVAR cohorts* | | | | | | | | | | |
| Hyhlik-Durr *et al.*[17] | 1997–2009 | 20 | 72 | 85 | 25 | 10 | 69 at 5 y | 22 | 0 | 10 |
| Engelberger *et al.*[20] | 2010–2019 | 28 | 75 | 96 | N.R. | N.R. | N.R. | 31 | 0 | 14.3 |
| Georgiadis *et al.*[18] | 2004–2012 | 19 | 70 | 95 | 37.5 | 10.5 | 68 at 3 y | 33 | 0 | 0 |
| Kruszyna *et al.*[21] | 2017–2019 | 24 | 67 | 54 | N.R. | 0 | N.R. | 20.5 | 0 | 0 |

Data are presented as %, unless stated otherwise. ARM = aorta related mortality; N.R.= not reported.
* Mean of the overall cohort.
† Cases performed only on abdominal lesions within a mixed cohort of aortic lesions.

## CONFLICT OF INTEREST STATEMENT AND FUNDING

None.

## AUTHOR CONTRIBUTIONS

Study design: G.P., D.B. Data collection: M.L., M.D., A.M., V.C., S.B., B.G., C.B. Data analysis: G.P., C.L., D.B., D.S., N.R. Writing: G.P., M.D., D.B., F.B., F.S., L.M. Critical revision and final approval: G.P., M.D., D.B., F.B., F.S., L.M., S.T., C.L., B.G., A.M., D.S., V.C., S.B., M.L., N.R., S.L., C.B., F.S., G.V. Overall responsibility: G.P.

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## APPENDIX B. SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ejvs.2022.03.004.

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COUP D'OEIL

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# A Large External Iliac Artery Pseudoaneurysm Encountered During Hip Surgery

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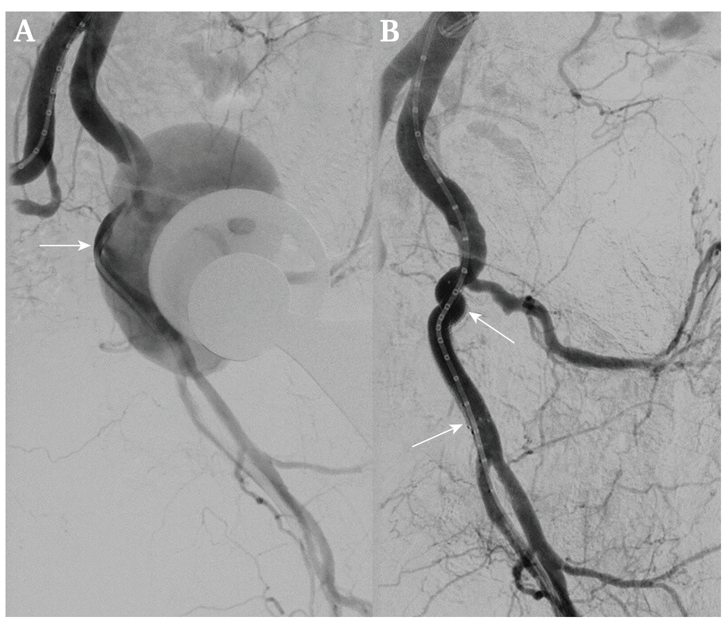


A 76 year old man presented with left peri-prosthetic hip infection with previous pelvic irradiation for rectal cancer. Hip dislocation during a Girdlestone procedure resulted in a significant bleed controlled by hip relocation and packing. Angiography revealed an external iliac artery pseudoaneurysm (A; arrow indicates origin of pseudoaneurysm from the external iliac artery). This was excluded by a covered stent (B; arrows [Viabahn 8 mm/10 cm; WL Gore & Associates, Flagstaff, AZ, USA]). After removal of the infected metal, the wound healed using vacuum assisted closure. The causative organism (*Esherichia coli*) was treated with eight weeks of meropenem in hospital.

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