Outcomes of Symptomatic and Ruptured Abdominal Aortic Aneurysm Repair: A Comparative Analysis Between Open and Endovascular Treatment

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ABSTRACT **Background:** The management of symptomatic abdominal aortic aneurysms (AAA) remains a surgical challenge.

Objectives: To compare the outcomes of endovascular aneurysm repair (EVAR) versus open surgical repair (OSR) in patients with symptomatic AAA.

Methods: Patients treated for symptomatic AAA between April 2020 and April 2025 were retrospectively analyzed, comparing perioperative mortality and major adverse events between EVAR and OSR.

Results: A total of 494 AAA patients were identified, 49 (9.9%) were symptomatic (40 [81.6%] EVAR group, 9 [18.4%] OSR group). Patients undergoing OSR had a higher rate of juxtarenal involvement (OSR 6/9 [66.7%] vs. EVAR 3/40 [7.5%]; $P < 0.001$). Any signs of rupture were more prevalent in the EVAR group (27/40 [67.5%] vs. OSR 2/9 [22.2%]; $P = 0.013$). Technical success was achieved in 83.7% (n=41/49). In-hospital mortality was 22.4% (n=11/49), with no difference between groups (EVAR 9/40 [22.5%] vs. OSR 2/9 [22.2%]; $P = 0.986$). At logistic regression analysis, open repair was associated with a significantly higher risk of major complications (odds ratio [OR] 16.9, 95% confidence interval [95%CI] 1.79–158.3, $P = 0.013$), and a shock index > 0.9 remained an independent predictor of intra-hospital mortality (OR 372.5, 95%CI 1.58-87889.4, $P = 0.034$). During a mean follow-up of 28.8 ± 18.6 months, late mortality was 18.4% (n=7/38). Estimated survival analysis over 60 months did not demonstrate a significant difference between groups (log-rank test, $P = 0.317$).

Conclusions: Both EVAR and OSR yield satisfactory technical outcomes. Hemodynamic instability at presentation remains a critical predictor of mortality.

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KEY WORDS: abdominal aortic aneurysm (AAA), endovascular aneurysm repair (EVAR), open surgical repair (OSR), shock index

An abdominal aortic aneurysm (AAA) is one of the most common yet challenging aortic conditions managed by vascular surgeons, as its rupture risks are closely linked to aging and cigarette smoking [1,2]. While the overall incidence of AAA is declining, largely due to smoking cessation and improved cardiovascular risk management, earlier studies reported prevalence rates of up to 5% among older male smokers, whereas contemporary screening studies suggest a marked decline, with prevalence approaching 1% in some populations [1-5]. Most AAAs are asymptomatic and treated by endovascular aneurysm repair (EVAR), which lowers perioperative morbidity and mortality, compared to open surgical repair (OSR) [6,7].

The management of symptomatic abdominal aortic aneurysm (sAAA) differs substantially from elective cases. In the setting of rupture (rAAA), mortality may reach up to 80%, and the optimal approach, EVAR versus OSR, is still debated [1,2,7]. A comprehensive meta-analysis of over 267,000 patients with ruptured AAA demonstrated lower perioperative mortality with EVAR compared with OSR (odds ratio [OR] 0.54, 95% confidence interval [95%CI] 0.51–0.57), although heterogeneity and selection bias were present across included studies [8]. The authors emphasized the need for further studies with more detailed data, focusing on clinical and anatomical factors [8,9].

In this context, we reviewed 5 years of our institutional experience with symptomatic and ruptured AAAs, comparing perioperative and during follow-up outcomes, morbidity, and mortality between patients with EVAR or OSR.

PATIENTS AND METHODS

STUDY DESIGN AND PATIENT ANALYSIS

All consecutive patients treated for symptomatic AAA at our institution between April 2020 and April 2025 were retrospectively analyzed from a prospective database. Those with prior aortic interventions or pararenal AAA were excluded. Patients were stratified by treatment modality into an endovascular symptomatic abdominal aortic aneurysm repair group (EsAAA) and an open surgical symptomatic abdominal aortic aneurysm repair group (OsAAA) groups for comparison. This study was approved by the Sheba Medical Center institutional review board (SMC 2209-25).

DATA COLLECTION

Patient demographic data, intraoperative, postoperative, and survival data were collected from medical records and follow-up visits. In case of death, the date was verified through the national civil registry. Patients who underwent EVAR were classified according to the Reporting Standards for Endovascular Aortic Aneurysm Repair of the Society for Vascular Surgery (SVS) [10]. Physiological parameters and laboratory results at admission were assessed, including the calculated shock index, defined as the ratio between heart rate and systolic blood pressure [11]. All patients underwent computed tomography angiography (CTA) at admission.

DEFINITIONS AND ENDPOINTS

Symptomatic AAA was defined according to the European Guidelines on the Management of Abdominal Aorto-Iliac Artery Aneurysms as the presence of abdominal and/or back pain or a tender AAA on palpation, without evidence of aortic wall rupture [2]. Ruptured AAA was defined as aortic wall disruption with hemorrhage into the retroperitoneal or intraperitoneal space. CTA findings classified ruptures as free (active contrast extravasation), contained (periaortic hematoma sealed by adjacent structures), or impending (wall discontinuity, fat stranding, or crescent sign suggesting imminent rupture) [2]. The variable any sign of rupture was defined as the presence of free rupture or radiological findings consistent with contained or impending rupture on admission CTA.

The primary endpoint was in-hospital mortality. Secondary endpoints included length of stay, reintervention rates, and major adverse events (myocardial infarction, prolonged intubation > 48 hours, renal dysfunction, bowel ischemia). Perioperative mortality was defined as death within 30 days or during hospitalization. Clinical and technical success were defined by survival without procedure-related mortality and correct aneurysm exclusion without conversion or unplanned adjuncts. Major complications comprised cardiac, cerebrovascular, pulmonary, and abdominal events, bleeding requiring reintervention, and renal failure per AKIN criteria [12].

MANAGEMENT OF SYMPTOMATIC AAA

Patients presenting with suspected rAAA triggered immediate vascular team activation. CTA was performed without delay, and permissive hypotension was maintained when rupture was evident. EVAR feasibility was assessed, and unsuitable cases underwent open repair by experienced vascular surgeons.

STATISTICAL ANALYSIS

Continuous variables were expressed as medians with IQRs and compared using the *t*-test or Mann–Whitney U test. Categorical data were analyzed with chi-square or Fisher's exact test. Variables with $P < 0.05$ entered logistic regression; multivariable results are shown as ORs with 95% CIs. Survival was assessed by Kaplan–Meier and compared by log-rank test. Analyses were performed using Wizard Statistics (Version 1.9.36, EvanMiller.org) and RStudio (Version 1.1.463, RStudio, Inc., Boston, MA, USA).

RESULTS

STUDY POPULATION

During the study period, 494 patients were treated for AAA at our institution. Of them, 445 (90.1%) were asymptomatic and 49 (9.9%) presented with sAAA. Among the symptomatic cohort, 40 patients (81.6%) underwent EsAAA and 9 (18.4%) OsAAA [Figure 1].

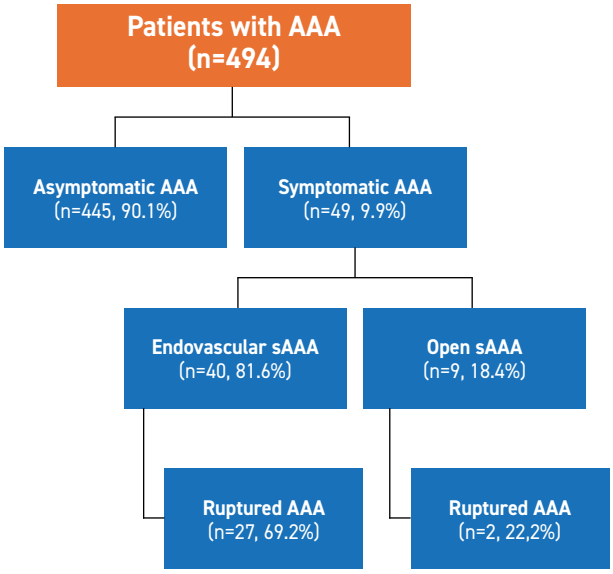
PREOPERATIVE CHARACTERISTICS

Baseline characteristics are summarized in Table 1. The groups were similar in age (75.3 ± 9.3 years for EsAAA vs. 68.6 ± 11.2 years for OsAAA; $P = 0.060$). and co-morbidities, except for dyslipidemia (100% vs. 55%; $P = 0.011$) and current smoking (88.9% vs. 40%; $P = 0.008$), both more frequent among open-repair patients. Chronic obstructive pulmonary disease (COPD) was also more common in the open group (44.4% vs.

12.5%; $P = 0.025$). Median hemoglobin and hematocrit levels were higher in OsAAA (13.5 g/dl [IQR 13.1–13.8] vs. 11.3 g/dl [10.4–12.7]; $P = 0.035$). Eleven patients (22.4%) presented with a shock index > 0.9, but their prevalence did not differ between groups ($P = 0.367$).

Figure 1. Flow diagram of patient distribution and treatment strategy for patients with abdominal aortic aneurysm between April 2020 and April 2025

AAA = abdominal aortic aneurysm, sAAA = symptomatic abdominal aortic aneurysm



ANEURYSM MORPHOLOGY AND CLINICAL PRESENTATION

Anatomic features are detailed in Table 1. Juxtarenal (JRAAA) involvement was more frequent in the OsAAA cohort (66.7% vs. 7.5%; $P < 0.001$), whereas infrarenal aneurysms predominated in the EsAAA group (92.5% vs. 33.3%; $P < 0.001$). Abdominal or back pain was the leading symptom (40.8%) and more frequent among OsAAA patients (77.8% vs. 32.5%; $P = 0.013$). Signs of contained or impending rupture were observed in 29 patients (59.2%) and more common in the EsAAA group (67.5% vs. 22.2%; $P = 0.013$). Associated iliac aneurysms were present in 30.6% of patients overall, with similar rates between groups (32.5% vs. 22.2%, $P = 0.542$).

Proximal neck anatomical characteristics were analyzed in the endovascular cohort (n = 40). The median proximal neck diameter was 21 mm (range 13–32 mm), with a median neck length of 29 mm (range 5–69 mm) and a median neck angulation of 15° (range 0–70°). A cylindrical neck configuration was present in 24 patients (60.0%), whereas 16 patients (40.0%) exhibited a conical morphology. The proximal main body diameter of the implanted endografts ranged from 16 to 36 mm, reflecting individualized device selection based on patient-specific anatomy. An aorto-uni-iliac configuration was used in four patients (10.0%), and femoro–femoral crossover bypass was performed in three patients (7.5%). One patient with chronic iliac artery occlusion did not require femoro–femoral crossover bypass. Tube graft configuration was used in six patients (15.0%).

Regarding bifurcated device selection, the Medtronic Endurant Stent Graft System (Medtronic Vascular, Minneapolis, MN, USA) was used in 25 patients (62.5%), the GORE® EXCLUDER® AAA Endoprosthesis (Gore Medical, Flagstaff, AZ, USA) in 4 patients (10.0%), and the Zenith Alpha Thoracic Endovascular Graft (Cook Medical LLC, Bloomington, IN, USA) in 1 patient (2.5%).

PERIOPERATIVE DETAILS AND EARLY OUTCOMES

Operative data are reported in Table 1. The median procedure time was longer for OsAAA (144 min [123–171]) compared with EsAAA (104 min [81–132]; $P = 0.050$). Intraoperative blood transfusion requirements were significantly higher among OsAAA patients (Red Blood Cells [RBC] 100% vs. 30%, $P < 0.001$; plasma 55.6% vs. 10%, $P = 0.006$). Technical or clinical success was achieved in 41 patients (83.7%) with no significant difference between repair types (85% vs. 77.8%; $P = 0.596$). Overall, in-hospital mortality was 22.4% (n=11), similar in both groups (EsAAA 22.5% vs. OsAAA 22.2%; $P = 0.986$). The median length of hospital stay was significantly shorter after EVAR (4 days [1–6] vs. 8 days [4-13]; $P = 0.003$).

Major adverse events occurred in 20 patients (40.8%) and were significantly more frequent in the OsAAA group (77.8% vs. 32.5%; $P = 0.013$). Complication patterns included cardiac (16.3%), pulmonary (20.4%), renal (44.9%), bowel ischemia (10.2%), and postoperative bleeding (6.1%). Postoperative acute kidney injury occurred in 22 patients, with most cases classified as AKIN 1 (28.5%) [Table 1].

Among the patients included in this endovascular group, three patients were identified with JRAAAs. One was treated with chimney endovascular aneurysm repair (ChEVAR) and two underwent standard EVAR. In the two standard EVAR cases, admission CTA demonstrated free rupture, and despite suboptimal proximal neck anatomy for an ideal sealing zone, standard EVAR was selected to allow rapid hemorrhage control. The patient treated with ChEVAR

Table 1. Preoperative demographic data at admission, aortic pathology characteristics, intraoperative and postoperative outcomes

Variable	Total (n=49, 100%)	EsAAA (n=40, 81.6%)	OsAAA (n=9, 18.4%)	*P*-value
Sex, male	47 (95.9%)	38 (95%)	9 (100%)	0.493
Age, years	74.0 ± 9.9	75.3 ± 9.3	68.6 ± 11.2	0.060
BMI, kg/m^2	25.9 ± 4.7	26.2 ± 4.6	24.9 ± 4.9	0.442
Hypertension	39 (79.6%)	32 (80%)	7 (77.8%)	0.881
Smoking history	24 (51%)	16 (40%)	8 (88.9%)	**0.008**
Diabetes	14 (28.6%)	10 (25%)	4 (44.4%)	0.243
Dyslipidemia	31 (63.3%)	22 (55%)	9 (100%)	**0.011**
CKD, eGFR < 60 ml/min	15 (30.6%)	12 (30%)	3 (33.3%)	0.845
COPD	9 (18.4%)	5 (12.5%)	4 (44.4%)	**0.025**
Ischemic heart failure	23 (46.9%)	20 (50%)	3 (33.3%)	0.365
Shock index > 0.9	11 (22.4%)	10 (25%)	1 (11.1%)	0.367
Hematocrit	35.8 [30.8–39.1]	34.3 [30.4–38.4]	41.4 [39.0–41.7]	**0.020**
Hemoglobin, g/dl	11.7 [10.4–13.3]	11.3 [10.4–12.7]	13.5 [13.1–13.8]	**0.035**
Hemoglobin < 11 g/dl	20 (40.8%)	19 (47.5%)	1 (11.1%)	**0.045**
WBC, × 10^3/μL	11.0 [7.9–15.0]	11.0 [8.8–15.2]	9.3 [7.6–11.8]	0.293
Fibrinogen	396 [275–450]	408 [297–470]	311 [226–405]	0.071
Platelet × 10^3/μL	215 [159–287]	236 [153–287]	207 [189–216]	0.639
ASA 5	16 (32.6%)	12 (30%)	3 (33.3%)	0.962
Juxtarenal AAA	9 (18.4%)	3 (7.5%)	6 (66.7%)	**< 0.001**
Aneurysm diameter, mm	67.3 ± 24.1	65.2 ± 25.2	76 ± 16.8	0.210
Saccular	10 (20.4%)	10 (25%)	0 (0%)	0.093
Pain	20 (40.8%)	13 (32.5%)	7 (77.8%)	**0.013**
Syncope	9 (18.4%)	8 (20%)	1 (11.1%)	0.534
Free rupture	13 (26.5%)	12 (30%)	1 (11.1%)	0.246
Any signs of rupture*	29 (59.2%)	27 (67.5%)	2 (22.2%)	**0.013**
Procedure time, min	106 [84–135]	104 [81–132]	144 [123–171]	**0.05**
Patients that received RBC	21 (42.9%)	12 (30%)	9 (100%)	**< 0.001**
Patients that received plasma	9 (18.4%)	4 (10%)	5 (55.6%)	**0.006**
Major complications	20 (40.8%)	13 (32.5%)	7 (77.8%)	**0.013**
Cardiac	8 (16.3%)	5 (12.5%)	3 (33.3%)	0.127
Cerebrovascular	2 (4.1%)	1 (2.5%)	1 (11.1%)	0.238
Pulmonary	10 (20.4%)	7 (17.5%)	3 (33.3%)	0.287
Bowel ischemia	5 (10.2%)	3 (7.5%)	2 (22.2%)	0.187
ACS	2 (4.2%)	1 (2.5%)	1 (11.1%)	0.247
Postoperative bleeding	3 (6.1%)	1 (2.5%)	2 (22.2%)	**0.048**
Renal	22 (44.9%)	17 (42.5%)	5 (55.6%)	0.516
Intra-hospital mortality	11 (22.4%)	9 (22.5%)	2 (22.2%)	0.986
LOS, days	4 [2–8]	4 [1–6]	8 [4–13]	**0.003**

AAA = abdominal aortic aneurysm, ACS = abdominal compartment syndrome, ASA = American Society of Anesthesiologists, BMI = body mass index, CKD = chronic kidney disease, COPD = chronic obstructive pulmonary disease, eGFR = estimated glomerular filtration rate, EsAAA = endovascular symptomatic abdominal aortic aneurysm repair group, OsAAA = open surgical symptomatic abdominal aortic aneurysm repair group, LOS = length of stay, RBC = red blood cells, WBC = white blood cells

*Defined as the presence of free rupture or radiological findings consistent with contained or impending rupture on admission computed tomography angiography (CTA)

Data are presented as number (%), mean ± standard deviation, or median [interquartile range]

Bold signifies statistical significance

presented with a contained rupture, enabling the use of a proximal chimney configuration to optimize proximal sealing. Technical success was achieved in all cases, with no evidence of type I or III endoleaks and no mortality within 24 hours postoperatively. During follow-up, non-aortic-related mortality occurred in the standard EVAR group at 5.4 and 21.9 months, respectively. The patient treated with ChEVAR remains clinically stable at 41.4 months of follow-up, with no imaging evidence of endoleak.

IN-HOSPITAL PREDICTORS OF COMPLICATIONS AND MORTALITY

At logistic regression [Table 2], open repair was independently associated with a higher risk of major complications compared to endovascular repair (OR 16.87, 95%CI 1.79–158.31; $P = 0.013$). In multivariate analysis [Table 3], a shock index > 0.9 on admission emerged as the only independent predictor of in-hospital mortality (OR 372.47, 95%CI 1.58–87889.4; $P = 0.034$). Age > 80 years and eGFR did not retain statistical significance after adjustment. Sex was not included in the multivariable model because of the marked male predominance and lack of between-group differences, limiting its discriminatory value and increasing the risk of model instability.

FOLLOW-UP AND LONG-TERM SURVIVAL

During a mean follow-up of 28.8 ± 18.6 months, late mortality occurred in 7 of 38 patients (18.4%), including two aortic-related deaths (5.3%). Kaplan–Meier survival analysis up to 60 months [Figure 2] demonstrated no significant difference between endovascular and open repair (log-rank = 0.317). Although no significant difference in survival was observed, the wide and overlapping confidence intervals indicate limited statistical power, and results should be interpreted cautiously.

Follow-up imaging analysis was performed in patients treated with endovascular repair after hospital discharge (n=31). Among these patients, the median maximum aneurysm diameter at the last available CTA was 48 mm (range 18–120 mm). Complete aneurysm sac regression was observed in 10 patients (32.3%), whereas the remaining patients demonstrated sac stability or residual aneurysm diameter without complete regression. During the follow-up period, no type I or type III endoleaks were identified, and no secondary conversion to open repair or reinterventions were described. Type II endoleak occurred in 6 patients (19.4%) during surveillance. Overall technical success during follow-up was achieved in 29 patients (93.5%).

Table 2. In-hospital outcome after symptomatic abdominal aortic aneurysm repair

Variable	EsAAA	OsAAA	OR	95%CI	*P*-value
Mortality	9 (22.5)	2 (22.2)	65.04	0.52–8041.33	0.089
Major complications	13 (32.5)	7 (77.8)	16.87	1.79–158.31	**0.013**

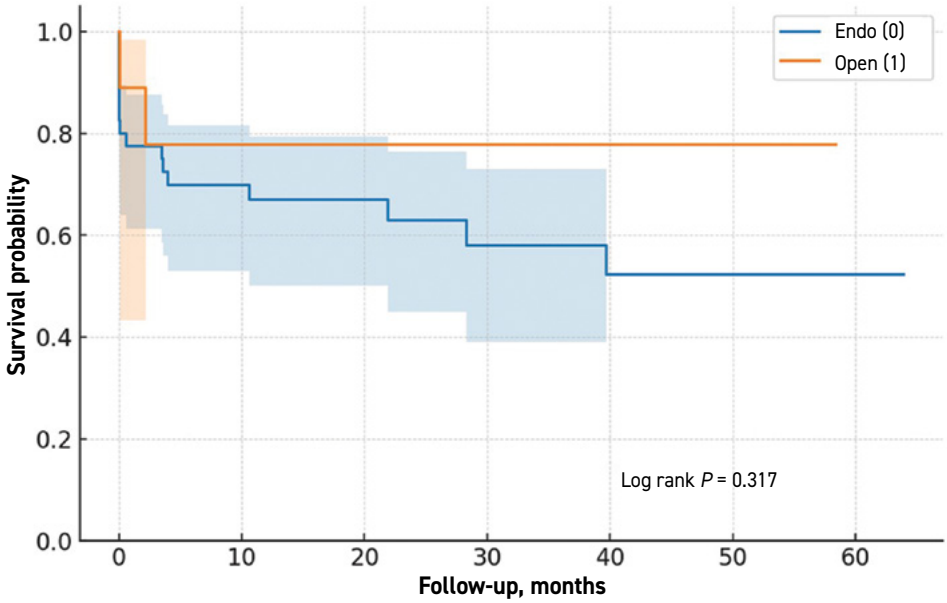
95%CI = 95% confidence interval, OR = odds ratio, AAA = abdominal aortic aneurysm, EsAAA = endovascular symptomatic abdominal aortic aneurysm repair group, OsAAA = open surgical symptomatic abdominal aortic aneurysm repair group
*Odds ratio is based on open abdominal aortic aneurysm repair compared to endovascular aortic aneurysm repair
Bold signifies statistical significance

Table 3. Univariate and multivariate analyses of factors associated with in-hospital mortality

Variable	Univariate analysis			Multivariate analysis		
	OR	95%CI	*P*-value	OR	95%CI	*P*-value
Age > 80 years	6.4	1.468–27.91	0.013	7.2	0.657–79.19	0.106
Shock index > 0.9	81	10.02–655.96	**< 0.001**	372.47	1.58–87889.4	**0.034**
eGFR, +1	0.96	0.938–0.997	0.030	0.99	0.952–1.039	0.816
Any sign of rupture*	10	1.163–85.998	0.036	1.35	0.082–22.34	0.833
Syncope	14	2.628–74.59	0.002	1.51	0.096–23.65	0.771

95%CI = 95% confidence interval, OR = odds ratio, eGFR = estimated glomerular filtration rate
*Defined as the presence of free rupture or radiological findings consistent with contained or impending rupture on admission computed tomography angiography
Bold signifies statistical significance

Figure 2. Kaplan-Meier survival curves comparing the open and endovascular groups (95% confidence interval)



DISCUSSION

In this study, we evaluated contemporary outcomes of patients treated for symptomatic and ruptured AAA, comparing EVAR and open surgical repair OSR. In this cohort, in-hospital mortality was similar between treatment modalities, but major complications were significantly more frequent after open repair. Shock index > 0.9 independently predicted in-hospital death, whereas EVAR was associated with a shorter hospitalization and lower morbidity. These results reinforce that, when anatomically feasible, EVAR represents a safe and effective approach for managing sAAA, with outcomes approximating those reported for elective AAA repair.

Although current ESVS guidelines assign a Class I, Level A recommendation to an EVAR-first strategy for ruptured AAA in anatomically suitable patients, this recommendation is largely informed by a combination of randomized trials and large observational datasets [2]. The IMPROVE trial provides the longest randomized follow-up to date, with 7-year outcome data, but its pragmatic design, treatment crossover, and inclusion of patients not anatomically eligible for EVAR limit its ability to reflect how contemporary EVAR-first pathways function in daily practice [2,13]. In contrast, real-world registries and institutional series capture the integrated effects of rapid triage, anatomical selection, modern device technology, and experienced multidisciplinary aortic teams, all of which strongly influence outcomes in emergency AAA care. The consistently lower perioperative mortality observed with EVAR in large observational datasets therefore likely reflects the effectiveness of EVAR-based systems of care rather than simply the technical performance of the device itself [2]. In this context, carefully characterized real-world cohorts, such as the present study, provide important complementary evidence to randomized trials by reflecting how ruptured AAA is actually managed in modern vascular centers.

Our findings align with those of Soden and colleagues [13] who analyzed more than 5500 AAA repairs in the ACS-NSQIP database and found that symptomatic AAAs carried an approximately twofold higher perioperative mortality compared with asymptomatic cases, yet significantly lower mortality than ruptured aneurysms. In their series, EVAR achieved lower 30-day mortality than OSR (3.8% vs. 7.7%), confirming the benefit of a minimally invasive approach [13]. More recently, Mulatti and co-authors [14] reported a 10-year experience implementing a structured endovascular-first protocol for ruptured AAAs, achieving an overall 30-day mortality of 44%, with EVAR associated with a significantly lower crude mortality (34.7%)

compared with open repair ($P = 0.001$). Even after propensity score matching, outcomes between repair types remained comparable, supporting the safety and reproducibility of EVAR under a dedicated system of care [14]. Together, these findings and our study highlight that expanding endovascular treatment to the symptomatic population can provide early mortality rates approaching those seen in elective repairs, when managed within optimized clinical pathways.

In our series, 83.7% of procedures were technically successful, consistent with prior reports showing 80–90% success for urgent EVAR in symptomatic settings [13-15]. The longer operative time and higher transfusion requirements observed in the OSR group are consistent with patterns reported for both elective and emergency aneurysm repairs [13,16]. Similar to von Meijenfeldt et al. [7,17], who demonstrated a threefold higher risk of major complications after OSR for ruptured AAA compared with EVAR, we observed that OSR independently predicted postoperative morbidity. Overall, these results illustrate the higher perioperative impact of open repair and the clear advantages of endovascular strategies in selected cases.

OSR was required predominantly in patients with complex juxtarenal anatomy, two-thirds of OsAAA cases, reflecting the anatomical constraints that still limit the broader use of EVAR in symptomatic aneurysms. Previous series have shown that complex neck anatomy, short sealing zones, and visceral involvement significantly influence both feasibility and outcomes [2,15,18]. The independent association between shock index > 0.9 and mortality in our cohort is consistent with evidence from ruptured AAA populations, where hemodynamic instability remains the strongest predictor of death regardless of repair type [7,16,19,20], These physiologic parameters should therefore guide triage and treatment prioritization in patients presenting with symptomatic aneurysms.

During a mean follow-up of nearly 30 months, late mortality was low and comparable between the groups, with Kaplan–Meier analysis confirming no survival difference up to 60 months. Once patients recover from the index hospitalization, long-term survival appears equivalent between OSR and EVAR, paralleling observations from large registry studies and prior meta-analyses [13,15,16]. The 18% late mortality observed in this study is notably lower than that reported for rAAA, reinforcing the intermediate-risk profile of the symptomatic population.

Our study adds to the growing body of evidence demonstrating that endovascular treatment offers at least equivalent early survival and reduced morbidity compared with open repair in patients with sAAA. Together with contemporary series on rAAA [14,15], these findings emphasize the value of institutional preparedness, standardized workflows, and dedicated vascular teams. Future multicenter research should aim to integrate sAAA within structured aortic emergency programs, refining patient selection and further optimizing the balance between anatomical feasibility, physiological stability, and procedural safety.

LIMITATIONS

This study has several limitations that should be acknowledged. First, the retrospective and single-center nature of this study inherently limits the ability to control for selection bias and potential confounding factors. The relatively small sample size, particularly in the OSR group, limits statistical power and constrains the ability to draw definitive comparative conclusions between treatment strategies. Consequently, the findings should be interpreted as hypothesis-generating rather than confirmation. Furthermore, the choice between endovascular and open repair was guided by anatomical feasibility and surgeon judgment rather than random allocation, which may have introduced treatment-selection bias. It is important to note that open repair was predominantly performed in patients with more complex anatomical features, particularly JRAAA, which likely accounts for the higher incidence of major adverse events observed in this group and precludes causal inferences regarding treatment superiority. Accordingly, the discussion and conclusions should avoid making conclusive comparative claims. Although follow-up was available for most patients, its duration was variable, and long-term data on reinterventions and aneurysm-related mortality remain limited.

CONCLUSIONS

In this single-center study, EVAR and open repair showed similar survival in symptomatic AAA; however, open surgery had more complications and longer hospitalization. EVAR offered faster recovery and lower morbidity. However, given the limited sample size and nonrandomized design, these comparisons should be interpreted as hypothesis-generating rather than confirmatory. A shock index > 0.9 independently predicted early mortality, emphasizing the prognostic value of hemodynamic instability and the need for prompt, specialized aortic care.

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Capsule

Local B cell immunity and durable memory after live-attenuated influenza intranasal vaccination of humans

Despite being a respiratory pathogen, vaccines for influenza virus are typically administered intramuscularly. The exception, FluMist, is a live-attenuated vaccine administered intranasally. **Stacey** and colleagues compared immune responses elicited by FluMist versus an intramuscular inactivated influenza vaccine. The authors found that intramuscular vaccination elicited, expectedly, systemic immune responses. In contrast, FluMist vaccination promoted vaccine-specific B cell responses, as determined by nasopharyngeal swabs. Thus, despite eliciting relatively poorer systemic immunity, FluMist generates a robust immune response against influenza where it matters, the portal of virus entry. These data support continued development of intranasal vaccines for influenza and other respiratory pathogens.

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