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Status of coronary disease and results from early endovascular aneurysm repair after preventive percutaneous coronary revascularization

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Abstract

Background: The incidence of coronary artery disease (CAD) is high in patients with an aortic aneurysm but preoperative routine coronary angiography and preventive coronary revascularization are not recommended to reduce cardiac events in patients with severe CAD.

Aim: This study evaluated the safeness and efficacy of preventive percutaneous coronary intervention (PCI) in patients with severe CAD scheduled for endovascular aneurysm repair (EVAR).

Methods: All patients with descending thoracic aneurysm (DTA) or abdominal aortic aneurysm (AAA) scheduled for EVAR underwent preliminary coronary angiography. Based on coronary angiography results, 917 patients (40.7%) had significant CAD and were treated by percutaneous coronary intervention (PCI; CAD group) and 1337 patients (59.3%) were without or with mild/moderate CAD and were considered as controls (no-CAD group). To evaluate the safeness and efficacy of preventive PCI in patients with severe CAD undergoing EVAR, groups were compared for hospital and 12-month cardiac adverse events.

Results: CAD was present in 1210 patients (53.6%): significant in 917 patients (38%) and mild to moderate in 293 patients (5.3%). Hospital and 12-month cardiac events occurred in 15 (1.6%) and 13 (1.4%) CAD group patients and in 9 (0.7%) and 8 (0.4%) no-CAD group patients ($p = .05$ and $p = .08$), respectively. Hospital and 12-month cardiac deaths occurred in 3 (0.3%) and 2 (0.2%) CAD group patients and in 3 (0.2%) and 2 (0.2%) no-CAD group patients ($p = .9$ and $p = .9$), respectively.

Conclusion: The strategy to treat severe CAD preoperatively by PCI and early subsequent EVAR brings a similar outcome to that in patients without or with mild/moderate CAD.

KEYWORDS

aortic aneurysm, coronary artery disease, endovascular procedures, percutaneous coronary intervention, risk assessment

Abbreviations: AAA, abdominal aortic aneurysm; BMS, bare-metal stents; CABG, coronary artery bypass graft; CAD, coronary artery disease; CHF, congestive heart failure; CTCA, computed tomography coronary angiography; DAPT, dual antiplatelet therapy; DES, drug-eluting stents; DTA, descending thoracic aneurysm; EVAR, endovascular aneurysm repair; MI, myocardial infarction; RCRI, revised cardiac risk index; TAAA, thoracoabdominal aneurysm.

1 | INTRODUCTION

The prevalence of aortic aneurysms in the health population older than 65 years was reported to range between 4% and 7%.[1] Coronary artery disease (CAD) may be present in 45%–65% of patients with aortic aneurysms who are often asymptomatic and have not a history of myocardial ischemia. The presence of CAD could account for 60%–70% of perioperative mortality and could be responsible for 40%–70% of late deaths in patients who underwent surgery.[2–6] Consequently, a cardiological workup aimed to identify the presence of CAD and an adequate preoperative strategy are mandatory to improve the surgical outcome.

Some studies support the choice of routine preoperative coronary angiography before major vascular surgery.[7,8] However, current guidelines on cardiological evaluation of patients undergoing noncardiac surgery are lacking of specific indications regarding the patients with aortic aneurysms undergoing elective repair, and the routine coronary angiography in the preoperative workup of these patients is suggested only in symptomatic patients.[9,10] Preventive coronary revascularization is not suggested even in patients with significant CAD.[9]

Based on our experiences in major vascular surgery, we included computed tomography coronary angiography (CTCA) or conventional coronary angiography as a part of routine cardiological workup in all candidates for aneurysm repair in the last decades.[8] All patients with severe CAD underwent preoperative myocardial revascularization preferentially by PCI with the implant of coronary stents. All patients treated by PCI underwent aneurysm repair within a few days. Endovascular aneurysm repair (EVAR) was the treatment of choice, wherever suitable, in all patients with descending thoracic aneurysm (DTA) or abdominal aortic aneurysm (AAA).

The aim of this study was to evaluate whether this aggressive strategy reduces the risk of hospital and midterm cardiac events. Results were compared to that of a homogeneous population of patients with DTA or AAA needing repair but free from significant CAD.

2 | METHODS

2.1 | Patients and preoperative cardiac evaluation

From January 2005 to December 2018, 2748 consecutive patients referred for DTA (632—23%) or AAA (2116—77%) repair. Preliminary exclusion criteria were as follow: thoracoabdominal aneurysm (TAAA) needing open repair, autoimmune diseases (systemic lupus erythematosus, rheumatoid arthritis, scleroderma, Sjögren's syndrome, or psoriatic arthritis), connective tissue disorders (Marfan syndrome, Ehlers–Danlos syndrome, and Loeys–Dietz syndrome), ascending aorta and/or aortic and/or mitral valve diseases needing surgical treatment. All patient had preoperative coronary angiography included in their cardiological workup independently of they reported history or symptoms of CAD or their revised cardiac risk index (RCRI).[11] CTCA was the first choice for screening all patients. When CTCA was not possible, not exhaustive, qualitatively inadequate, or positive for CAD, patients were referred for conventional angiography. To avoid the bias due to different surgical strategies with deeply different clinical implications and outcomes, 251 patients (9.1%) who had indications for open repair of aneurysm were also excluded. Similarly, 85 patients (3.1%) who had indications for coronary artery bypass graft (CABG) were excluded.

Based on the results of coronary angiography, 2412 patients were divided into two groups: 917 patients who had evidence of severe CAD (CAD group) and 1495 patients without evidence of CAD or with mild to moderate CAD (no-CAD group). To obtain the most significant and homogeneous possible no-CAD group for the aim of the study and avoid the possible bias due to a possible "gray zone" between groups, 158 patients with a history of PCI but free from significant residual stenosis at angiography were excluded from the no-CAD group. At least the study cohort included 2254 patients: 917 in the CAD group (40.7%) and 1337 in the no-CAD group (59.3%) underwent EVAR early after angiography (Figure 1). The most pertinent clinical data and cardiac risk factors of study groups are summarized in Table 1. Notably, the CAD-group included 123 patients who were treated previously by PCI (from 1.2 to 7.5 years before; mean = 2.9 ± 1.1 years) and revealed significant CAD at coronary angiography.

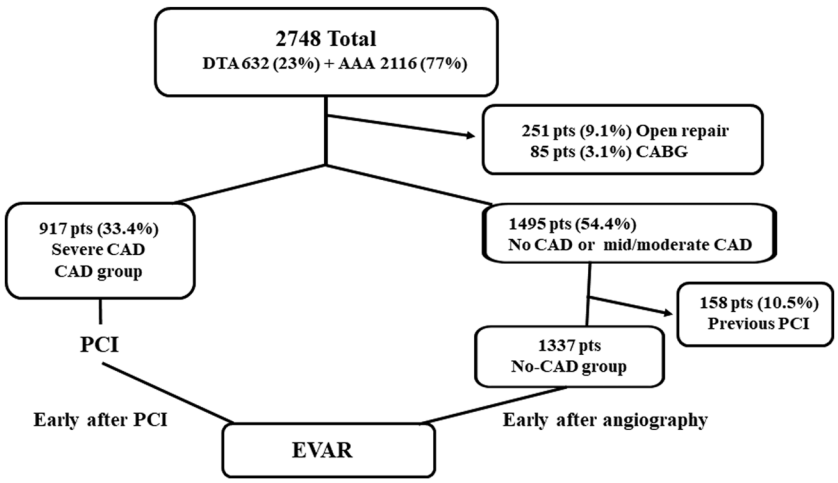

FIGURE 1 Flow chart summarizing enrollment criteria. AAA, abdominal aortic aneurysm; DTA, descending thoracic aneurysm; CABG, coronary artery bypass graft; CAD, coronary artery disease; EVAR, endovascular aneurysm repair; PCI, percutaneous coronary intervention

TABLE 1 Demographic and clinical characteristics

	CAD group 917 pts	No-CAD group 1337 pts	*p*
Age, mean (years)	72.8 ± 5.6	73.1 ± 5.7	.3
IQR	65–79	64–79	
Female sex	292 (31.8%)	431 (32.2%)	.9
BMI mean	27.8 ± 5.4	26.6 ± 4.4	.03
IQR	22–28	23–30	
Hypertension	782 (85.3%)	1083 (81%)	.005
Hypercholesterolemia	549 (59.8%)	722 (54%)	.004
Diabetes mellitus	288 (32.2%)	444 (27.9%)	.3
Insulin requiring	108 (11.7%)	127 (9.5%)	.08
Smoking status			
Previous smoker	223 (24.3%)	413 (30.9%)	<.001
Current smoker	95 (10.3%)	139 (10.4%)	1
Aneurysm diameter (mm)	64 ± 7	65 ± 8	.1
IQR	58–71	60–74	
Thoracic aneurysms	211 (23%)	331 (24.7%)	.3
Abdominal aneurysms	706 (77%)	1006 (75.3%)	.3
Familiarity for CAD	312 (34%)	427 (31.9%)	.3
RCRI score	2.18 ± 0.5	2.07 ± 0.7	.03
Previous MI	101 (11%)		
Previous PCI	123 (13.4%)		
Single	75 (8.2%)		
Double	48 (5.2%)		
Mild-moderate CAD		293 (22%)	

Note: Values are means ± *SD* or numbers.

Abbreviations: BMI, body mass index; CAD, coronary artery disease; IQR, interquartile range; MI, myocardial infarction; PCI, percutaneous coronary intervention; RCRI, revised cardiac risk index.

The study complies with the 2013 version of the Declaration of Helsinki. We gained Ethical Committee approval from the Institutional Research Ethics Committee. Given the retrospective nature of the study, the need for individual patient consent was waived, but all patients had preliminarily granted permission for use of their medical records for research purposes and provided written informed consent to be treated according to our protocol.

2.2 | Study end-points and definitions

End-points of the study were (a) the incidence of CAD in candidates for DTA or AAA repair and (b) the results of preventive myocardial revascularization by PCI in patients with preoperative significant CAD quantified as hospital and 12-month cardiac events. Bleeding and endoleak were additional events evaluated as related to the surgical procedure and antiplatelet therapy.

Cardiac events were a composite including cardiac deaths, angina, nonfatal myocardial infarction (MI), and congestive heart failure (CHF) defined according to the Consensus Report, 2017 Cardiovascular and Stroke Endpoint Definitions for Clinical Trials.[12] Hospital were events occurred within 30 days or at any time after the operation if the patient did not leave the hospital. The time point for midterm evaluation was fixed at 12 postoperative months in all patients to avoid the bias due to different duration of follow-up. We assumed that, for the present study, this time point better reflected the impact on the outcome of interest. Bleeding was graded according to the Bleeding Academic Research Consortium.[13] For the present study, we chose to compute only Type 2 and 3 bleeding events. Severe CAD were lesions exceeded 70% stenosis in a major coronary vessel, or 30%–70% stenosis with fractional flow reserve ≤0.8.

2.3 | Coronary intervention and antiplatelet therapy

Coronary angiography was performed via radial access in all patients wherever not contraindicated. This approach was preferred over femoral access mainly for three reasons: (a) possibility of mechanical damage of the aneurysm, (b) mobilization of intraluminal thrombotic material, and (c) the frequent groin hematoma after femoral puncture that can support catastrophic infection of the devices used for EVAR. Patients with severe CAD (CAD-group) were treated by preventive PCI and had at least one stent implanted. All patients who underwent PCI received a loading dose of clopidogrel consisting of 600 mg and subsequent dual antiplatelet therapy (DAPT) with clopidogrel 75 mg/day + aspirin 100 mg/day. The DAPT was not interrupted for the EVAR procedure.

2.4 | Surgical procedures

EVAR, via femoral access, was the approach of choice in all our patients with DTA or AAA whenever not contraindicated by unfavorable extension of the aneurysm, difficult vascular accesses, or unreliable landing zone. Local anesthesia was usually preferred. Heparin (100 IU/kg) was administered with subsequent boluses given to maintain an activated clotting time >250 s. Patients needing thoracic aorta repair had a Medtronic Talent Stent Graft always implanted (Medtronic). Patients needing abdominal aorta repair had Endurant or Talent Stent Graft (Medtronic), Anaconda (Vascutek), or E-vita (Jotec) implanted. Patients stayed in the intensive care unit for 12–24 h after the procedure and were monitored for myocardial ischemia with serial electrocardiography, serum cardiac enzyme analysis, and echocardiography.

2.5 | Statistical analysis

Based on the assumption that the cumulative risk of hospital and midterm postoperative cardiac events for patients with CAD who

underwent EVAR is about 8%,[14–16] the number of patients required in this retrospective study to obtain a statistical power of at least 90% and one-sided α-level of .05 (estimated difference 0%, margin of non-inferiority of +4%) was of at least 1800 taking into account a 99% complete follow-up at 12 months (calculated by Sample Size Calculator: www.raosoft.com/samplesize.html).

Continuous variables were summarized by mean, standard deviation, and interquartile range (25th–75th percentile), categorical data as proportions. The normal distribution of continuous values was tested by means of the Anderson–Darling test. $p > .05$ was considered indicative of normal distribution. The comparison between continuous variables was made by means of the Student's *t*-test for normally distributed values. The Mann–Whitney *U* test was used for variables not normally distributed. Categorical variables were analyzed with the χ^2 test with Yates correction when appropriate. Differences resulting in $p < .05$ were considered significant. Pearson's correlation coefficient was used to assess the linear association between RCRI score and significant CAD. Data were analyzed by SPSS version 15 for Windows (SPSS, Inc.).

3 | RESULTS

3.1 | Incidence of CAD and preventive PCI

Out of 2254 patients included in the study, 1210 (53.6%) patients had CAD at coronary angiography: significant in 917 patients (38%) and mild/moderate in 293 (5.3%). The mean RCRI score in the whole population of patients enrolled was 2.1 ± 0.6 without any difference between groups. By Pearson's correlation, only a weak correlation between preoperative RCRI score and presence of significant CAD was found ($r = .21$).

Within CAD-group, 1326 coronary arteries were treated: left anterior descending 703 (53%), left coronary artery 375 (28.3%), and right coronary artery 248 (18.7%). Single-vessel PCI was performed in 61.3% (562/917) of patients, double-vessel in 32.8% (301/917), and triple-vessel in 5.9% (54/917). Overall, 1485 stents were implanted (mean = 1.6 stent/patient): bare-metal stents (BMS) in 7% (64/917) of patients, early generation drug-eluting stents (DES) in 10.4% (96/917), and new generation DES in 82.6% (757/917). The distribution of target vessels for PCI and stent type is detailed in Table 2.

No access complications occurred in both groups related to diagnostic angiography. No aneurysm ruptures occurred in the interval between PCI and EVAR. No death occurred related to PCI in the CAD group. Periprocedural MI occurred in 0.7% of patients (6/917). The mean interval from PCI to EVAR was 2.0 (range = 1.0–4.0) days.

3.2 | End-point hospital outcome

Conversion from local to general anesthesia was necessary in 37 (4%) of CAD patients and in 50 (3.7%) no-CAD patients ($p = .7$). Hospital cardiac events occurred in 1.6% CAD group patients (15/917) versus 0.7% no-CAD patients (9/1337; $p = .05$). Cardiac deaths occurred in 0.4% CAD patients (3/917%) versus 0.2% no-CAD patients (3/1337; $p = .8$; Table 3). Causes of cardiac deaths were MI and CHF. Further deaths occurred due to renal failure 3, sepsis 2, intestinal infarction 2, and pneumonia 1 equally distributed between groups.

Within the CAD group, out of six patients who experienced hospital MI, four had been treated by double or triple PCI for complex lesions. One patients of these had BMS, two an early generation DES and one a new generation DES implanted, two had a chronic total left anterior descending occlusion at the time of PCI.

In the no-CAD group, MI occurred mainly in patients who had moderate CAD at preoperative angiography.

Comparative analysis of asymptomatic versus symptomatic patients within the CAD-group evidenced a tendentially higher incidence of cardiac events in symptomatic patients (2/65—3.1% vs. 13/852—1.5%), which, however, did not raise statistical significance (odds ratio [OR] = 0.5, 95% confidence interval [CI] = 0.11–2.18; $p = .3$).

TABLE 2 Angina status, angiographic, and procedural characteristics at PCI

	CAD group 917 pts	No-CAD group 1337 pts	*p*
Asymptomatic	852 (92.9%)	1310 (97.9%)	<.001
Stable angina	65 (7.1%)	27 (2.1%)	<.001
LVEF (mean)	43 ± 5	47 ± 6	<.001
Median (IQR)	45 (36–61)	45 (42–60)	.005
Type of coronary lesions			
Chronic complete occlusion	146 (15.9%)		
Complex lesions	293 (31.9%)		
PCI procedures	1326		
Single vessel	562 (61.3%)		
Double vessels	301 (32.8%)		
Triple vessels	54 (5.9%)		
Target vessels for PCI			
Left anterior descending	703 (53%)		
Left coronary artery	375 (28.3%)		
Right coronary artery	248 (18.7%)		
Number of stents implanted	1485		
Number of stents/pts	1.6		
Stent diameter mm (mean)	3.1 ± 0.2		
Type of stent			
BMS	64 (7%)		
Early generation DES sirolimus	96 (10.5%)		
New generation DES everolimus	270 (29.4%)		
Zotarolimus	372 (40.6%)		
Sirolimus	115 (12.5%)		

Note: Values are means ± *SD* or numbers.

Abbreviations: BMS, bare-metal stent; DES, drug-eluting stent; IQR, interquartile range; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention.

TABLE 3 Clinical results. Hospital and 12-month cardiac deaths and cardiac events

	CAD group	No-CAD group	*p*
Hospital	917 pts	1337 pts	
Cardiac events	15 (1.6%)	9 (0.7%)	.05
Cardiac deaths	3 (0.4%)	3 (0.2%)	.8
Acute MI	6 (0.6%)	3 (0.2%)	.2
CHF	6 (0.6%)	3 (0.2%)	.2
Bleedings	8 (0.9%)	3 (0.2%)	.06
Endoleaks			
Type I	23 (2.5%)	32 (2.4%)	.8
Type II	23 (2.5%)	19 (1.4%)	.06
12 months	908 pts	1326 pts	
Cardiac events	13 (1.4%)	8 (0.6%)	.08
Cardiac deaths	2 (0.2%)	2 (0.2%)	.9
Acute MI	4 (0.4%)	2 (0.2%)	.9
Angina	5 (0.5%)	3 (0.2%)	.8
CHF	2 (0.2%)	1 (0.07%)	.7
Bleedings	1 (0.1%)	2 (0.2%)	.5

Note: Values are means ± *SD* or numbers.

Abbreviations: CAD, coronary artery disease; CHF, congestive heart failure; MI, myocardial infarction.

Eight CAD group patients and three no-CAD group patients ($p = .06$) experienced bleeding requiring revision of the surgical site. Seven infections of the surgical site occurred in the CAD group and 10 in the no-CAD group ($p = .8$). No paraplegias occurred in both groups. Three (0.3%) transient and reversible contrast-induced nephropathy occurred in the CAD group and in 5 (0.4%) in the no-CAD group ($p = .8$). The incidence increased to 13% in patients with preexisting renal impairment (estimated glomerular filtration rate (eGFR) < 60 ml/min) and/or with diabetes mellitus and/or patients who received high contrast volume.

Type I and II endoleaks occurred respectively in 23 (2.5%) and 23 (2.5%) CAD group patients and in 32 (2.4%) and 19 (1.4%) no-CAD patients ($p = .8$ and $p = .06$). Additional endovascular procedures to treat Type I endoleak were necessary in 19 CAD patients and in 30 no-CAD patients. No additional procedures were necessary in patients with Type II endoleak who showed only a delayed complete thrombus formation in the perigraft space.

3.3 | End-point 12-month outcome

The evaluation of 12-month outcome was 99.4% complete. The incidence of 12-month cardiac events is depicted in Table 3.

In adherence to our current policy, all patients who experienced angina or MI underwent coronary angiography. In-stent restenosis was evidenced in seven (0.7%) patients. All patients with in-stent restenosis were previously treated by double or triple PCI and two of these had BMS implanted. Five patients with in-stent restenosis were treated by repeated stenting, one patient underwent successfully CABG surgery.

Comparative analysis of asymptomatic versus symptomatic patients within CAD-group confirmed also for 12-month outcome the tendentially higher incidence of cardiac events in the subgroup of symptomatic patients (2/63—3.2% vs. 11/839—1.3%), which, however, did not raise statistical significance (OR = 0.4, 95% CI = 0.08–1.83; $p = .2$).

Significant bleeding occurred in three patients: two intestinal bleeding, one in each group, and one brain hemorrhage in the no-CAD group.

4 | DISCUSSION

The present study focused on two queries regarding the approach to patients who need surgery for DTA or AAA: the most appropriate cardiological workup related to the high incidence of CAD and the most effective strategy for the treatment of patients with significant CAD.

Our results indicate that: (1) the incidence of significant CAD is high in patients with DTA or AAA needing repair; (2) routine CTCA or conventional coronary angiography offer the opportunity to identify preoperatively all these patients; and (3) preventive treatment of patients with significant CAD by PCI ensures similar hospital and 12-month rates of cardiac events than patients free from CAD or with mild/moderate CAD.

The most recent ACC/AHA guidelines on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery indicate a stepwise strategy based on the evaluation of clinical and surgical risk factors.[9,10] Guidelines suggest the RCRI as the tool of choice to evaluate the cardiovascular risk: patients with RCRI ≤ 2 are at low or medium risk and should be referred only for appropriate therapy (beta-blockers and statins) without further cardiological evaluations; patients with RCRI > 2 are considered as high-risk patients and should undergo further noninvasive tests. Guidelines suggest coronary angiography only after positive noninvasive testing whereas coronary revascularization is not recommended to reduce cardiac events in patients with significant CAD (Class III, Level of Evidence B).[17]

The limitations of these statements are evident in patients needing aneurysm repair. Guidelines include generically all types of noncardiac surgery neglecting the real and specific characteristics of patients with aortic aneurysms. These patients are often asymptomatic and usually unaware of the presence of severe CAD that increases the risk of dynamic or pharmacological tests and sometimes shows serious mobility limitations that hinder dynamic evaluations. Nonetheless, the incidence of CAD in patients undergoing elective aneurysm surgery is reported to range from 40% to 55%, which is considered responsible for 60%–70% of perioperative mortality and 40%–70% of late deaths in patients who underwent aneurysm repair.[2–6,15,16,18]

In the absence of clear and specific indications by guidelines, the challenge related to the optimal treatment of CAD in patients with AAA or TAA has been addressed and resolved differently over the years. At first, an open approach for both CAD and aortic aneurysm (delayed or performed contemporarily) was the only possible option.[19] More recently, PCI and subsequent open aneurysm repair has been reported to be effective to improve early and late outcome.

Only in the recent, technical advances of devices suggested the possibility to treat both CAD and aortic aneurysm by endovascular approach. This consisted initially of a two-staged strategy with stenting PCI and subsequent EVAR performed at least 4–8 weeks later, due to the necessary waiting time for stents endothelization.[20,21] The natural evolution of this approach was one stage strategy with early EVAR after PCI. Before this our study, the encouraging results of this strategy have been published only by Pecoraro et al.,[22] who analyzed a small series of 20 patients treated by EVAR for AAA performed early (mean 2.5 days) after PCI.

In agreement with the literature, we found coronary lesion/s in 53.6% (1210/2254) at coronary angiography of our patients undergoing DTA or AAA repair. Nonetheless, it was striking to note that as high as 90% of patients with significant CAD (852/917) were asymptomatic and only 16.1% (148/917) had a documented history of CAD. Therefore, according to the statements of guidelines, only a few patients would fulfill the indications for coronary angiography and revascularization. The problem is anything but negligible as these results, taken together, raise several perplexities on the ability of noninvasive tests to provide sufficient assistance for the assessment of cardiac risk in asymptomatic patients with RCRI < 2.

In this study, we hypothesized that the preventive treatment by PCI, whenever possible, of all patients with severe CAD, could reduce the hospital and midterm rate of deaths and cardiac events of subsequent EVAR. Our results evidence that patients with severe CAD treated by preventive PCI displayed similar results compared with patients without CAD or with mid/moderate coronary lesions. Compared with results from the largest reviews on this topic, our strategy of preventive treatment of severe CAD and less invasive approach for aneurysm repair, allowed a threefold hospital and 12-month reduction of cumulative cardiac events.[4–6,17,18]

Several concerns could be related to the DAPT necessary after PCI that could expose the patients to additional bleeding risk at surgery. Nonetheless, we never suspended DAPT before EVAR in our patients and this did not increase the risk of perioperative bleeding likely due to the minimal surgical approach. Furthermore, the very short interval between coronary and aortic procedures could increase the risk of stent thrombosis in the early postoperative period of EVAR due to the incomplete endothelization of the stents.[23] However, the low rate of this complication in our CAD group could be related to the persistent protective effect of clopidogrel load before PCI and the low level of hypercoagulability related to the low invasive surgical approach necessary for EVAR, contrarily to that reported in conventional open aneurysm repair.[24] In this regard, it was not surprising that more than 80% of ischemic events occurred in patients who had BMS or first-generation DES implanted. However, this data must be interpreted cautiously related to the low events rate that hinders every significant analysis of risk factors for stents thrombosis in this study. The persistent effect of clopidogrel load and of continuative DAPT could be responsible for the tendentially higher incidence of Type II endoleaks registered in the CAD group. However, none of these events required any additional treatment and the complete thrombus formation in the perigraft space was only delayed without any clinical consequence.

The low rate of bleeding and Type II endoleak registered perioperatively in our series in addition to the low rate of stents thrombosis support the hypothesis that EVAR can be safely performed under DAPT. This experience justifies reconsidering the general policy of postponing EVAR for a long time when PCI with stents has been performed. Postponing the aneurysm repair to wait for a complete stents endothelization exposes to an increased rupture risk. Literature reports that patients with DTA or AAA diameter > 60 mm had an estimated annual rupture/dissection rate ranging from 10% to 20%.[25] Hence, in our cohort of patients who had a mean diameter > 60 mm, we should expect a significant number of such catastrophic events.

Notably, the comparative analysis of asymptomatic versus symptomatic patients within the CAD-group did not provide any statistically significant result. Although the events rate was tendentially higher in symptomatic patients, the differences did not raise statistical significance. Likely, the considerable disproportion between groups and the low incidence of events hindered a conclusive analysis.

This study has several limitations. First, the retrospective nature. However, it could be very difficult to collect a similar number of patients in a prospective study. Second, the study was not designed to evaluate the long-term outcome, which may not be related to the end-point of interest. Finally, although a large number of patients enrolled, events of interest occurred at a very low rate to provide an absolute determination of inherent risk factors.

5 | CONCLUSION

The incidence of CAD is significant in patients undergoing elective aneurysm repair. Patients with severe CAD, identified by routine preoperative coronary angiography and treated by preventive PCI, had similar hospital and 12-month cardiac events and bleeding rates than patients without significant CAD. Hence, routine coronary angiography, preventive PCI in patients with severe CAD, and early EVAR may be a safe and effective approach in patients with DTA or AAA needing elective repair.

This study although it includes more than 2000 patients is far to be definitive. However, the combination of recent evidence in the literature and new data from this study may support some criticism of ACC/AHA guidelines on the preoperative assessment and treatment of candidates for aneurysm surgery.

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CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

AUTHOR CONTRIBUTIONS

Conception and design of the study: Vito A. Mannacio and Luigi Mannacio. *Acquisition of data*: Luigi Mannacio, Emilio Mileo, Michele Mottola, and Anita Antignano. *Analysis and interpretation of data*:

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