The prognostic value of preoperative systemic inflammation-based scoring in patients undergoing endovascular repair of abdominal aortic aneurysm

Nicholas A. Bradley, MBChB,[a] Amy Walter, MBChB,[b] Alasdair Wilson, MD,[c] Tamim Siddiqui, MD,[d] Campbell S. D. Roxburgh, PhD,[a] Donald C. McMillan, PhD,[a] *and* Graeme J. K. Guthrie, MD,[a,b] *Glasgow, Dundee and Aberdeen, United Kingdom*

ABSTRACT

Objective: Abdominal aortic aneurysm (AAA) is a common condition that is predominantly managed in the United Kingdom by endovascular aneurysm repair (EVAR). Activation of the systemic inflammatory response (SIR) appears to offer prognostic value in patients with vascular disease. The present study examines the relationship between the SIR and survival in patients undergoing standard and complex endovascular aneurysm repair (EVAR and fenestrated/branched [F/B]-EVAR).

Methods: Consecutive patients undergoing elective EVAR and F/B-EVAR were retrospectively identified from three tertiary vascular centers over a 5-year period. Neutrophil:lymphocyte ratio and modified Glasgow Prognostic Score were calculated from preoperative blood results and combined into the systemic inflammatory grade (SIG). The primary outcome was all-cause mortality during the follow-up period, which was compared between subgroups of SIGs.

Results: There were 506 patients included in the final study, with a median follow-up of 68.0 months (interquartile range, 27.3 months), and there were 163 deaths during the follow-up period. Mean survival in the SIG 0 vs SIG 1 vs SIG 2 vs SIG 3 vs SIG 4 subgroups was 80.7 months (95% confidence interval [CI], 76.5-85.0 months) vs 78.7 months (95% CI, 72.7-84.7 months) vs 61.0 months (95% CI, 51.1-70.8 months) vs 65.1 months (95% CI, 45.0-85.2 months) vs 54.9 months (95% CI, 34.4-75.3 months) ($P < .05$). In the entire cohort, age ($P < .001$), body mass index ($P < .05$), high creatinine ($P < .05$), and SIG ($P < .05$) were associated with survival on univariate analysis, with retained independent association for age (hazard ratio, 1.72; 95% CI, 1.29-2.31; $P < .001$) and SIG (hazard ratio, 1.20; 95% CI, 1.02-1.40; $P < .05$) on multivariate analysis. Increasing SIG (area under the curve, 0.68; 95% CI, 0.58-0.78; $P < .01$) predicted 1-year mortality.

Conclusions: Markers of the SIR such the SIG may be used to identify patients at higher risk of adverse outcome in patients undergoing EVAR and F/B-EVAR for abdominal aortic aneurysms. These findings warrant further investigation in large prospective cohort studies. (J Vasc Surg 2023;78:362-9.)

Keywords: AAA; EVAR; mGPS; NLR; SIG

Abdominal aortic aneurysm (AAA) is a pathological dilatation of the abdominal aorta to greater than 1.5× normal diameter. Increasing size is associated with increased risk of rupture, with the current threshold for elective intervention a maximal antero-posterior diameter of 5.5 cm.[1] In the United Kingdom (UK), endovascular aneurysm repair (EVAR) accounts for approximately 65% of interventions to treat AAA.[2] Infrarenal AAA may be managed by standard EVAR, with juxta/para-renal AAA requiring more complex fenestrated or branched devices (F/B-EVAR). Alternatively, in patients with favorable comorbid state and lack of abdominal copathology, open surgical repair may be preferred.[1]

Activation of the systemic inflammatory response (SIR) is a common response found in chronic illnesses.[3] Moreover, inflammation is thought to play a key role in not only the pathogenesis but also the prognosis of atherosclerotic disease such as ischemic heart disease.[4-8] The role of inflammation in the pathogenesis of AAA is incompletely described; however, there is evidence of inflammatory stimulus in both vascular endothelial cells and in thrombus contained within the aneurysm sac.[9,10]

From the Academic Unit of Surgery, University of Glasgow, Glasgow[a]; the Department of Vascular Surgery, NHS Tayside, Dundee[b]; the Department of Vascular Surgery, NHS Grampian, Aberdeen[c]; and the Department of Vascular Surgery, NHS Lanarkshire, Glasgow.[d]

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Correspondence: Nicholas A. Bradley, MBChB, Room 2.56, New Lister Building, Glasgow Royal Infirmary, Glasgow, G4 0SF, United Kingdom (e-mail: nicholasandrew.bradley@glasgow.ac.uk).

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The neutrophil:lymphocyte ratio (NLR) can be derived from routine blood tests performed during the preoperative period, and has been reported to predict survival in patients with cancer,[11,12] peripheral vascular disease,[13] and cerebrovascular and coronary artery disease.[14]

The modified Glasgow Prognostic Score (mGPS) is a scoring system based on C-reactive protein (CRP) and albumin results, which has been shown to predict outcomes in patients undergoing both radical and palliative treatment in a variety of cancers.[12,15-17] More recently, this has been applied to patients with cardiovascular disease, and has been shown to predict inferior outcomes.[18,19]

Recent studies have combined mGPS and NLR in recognition of the differing contributions to overall systemic inflammatory response from acute phase proteins (mGPS) and white cells (NLR). This has been reported in patients with colorectal[20] and esophagogastric[21] cancer, culminating in the systemic inflammatory grade (SIG) proposed by Golder et al, which offered superior prognostication compared with NLR or mGPS alone in patients with colorectal cancer managed by curative intent.[22]

There is a growing body of evidence describing an association between elevated NLR and inferior prognosis in patients undergoing intervention for AAA, described in both elective and emergency cases, as well as both EVAR and open surgical repair treatment modalities.[23-25] However, the existing literature is plagued with heterogeneity regarding timing of blood sampling, optimal cutoff of NLR to subgroup patients, and limited follow-up. Moreover, mGPS and SIG remain unreported in this patient cohort.

The aim of the present study was to examine the relationship between the SIG and long-term survival in patients undergoing elective endovascular intervention for AAA.

ARTICLE HIGHLIGHTS

- **Type of Research:** Multicenter retrospective cohort study
- **Key Findings:** In 506 patients undergoing elective endovascular aneurysm repair and fenestrated/branched endovascular aneurysm repair for abdominal aortic aneurysms, elevated preoperative markers of systemic inflammation were independently associated with inferior long-term survival (hazard ratio, 1.20; 95% confidence interval, 1.02-1.40; $P < .05$).
- **Take Home Message:** Markers of the systemic inflammatory response such the systemic inflammatory grade may be used to identify patients with abdominal aortic aneurysms with inferior long-term prognosis.

METHODS

Patient selection. Patients were retrospectively identified from theater records at three large tertiary referral centers in Scotland, UK, representing cases performed in three health boards (National Health Service [NHS] Grampian, NHS Lanarkshire, and NHS Tayside). Specific procedural technique and choice of stent graft were at the discretion of each institution, although practice was broadly similar between sites throughout the study period. Consecutive cases undergoing elective EVAR or F/B-EVAR to treat aortic aneurysmal disease between January 1, 2015, and October 1, 2021, were included. Patients with active infection, isolated iliac aneurysms, aortic dissection, penetrating aortic ulcer, or incomplete clinical or follow-up data were excluded. Clinical, demographic, and outcome data were recorded from electronic case records and patients' community health records. Comorbidity was assessed using American Society of Anesthesiologists score, which was recorded from operative records and subgrouped (≤2/>2) in keeping with previous literature.[26] Patients were subgrouped based on age (<65, 65-75, or >75). High creatinine was defined as >1.20 mg/dL (male patients) and >0.90 mg/dL (female patients), based on local laboratory values. In all patients, the date of follow-up was more than 2 years from the date of surgery. West of Scotland Research Ethics Committee approval was obtained for this study (Reference 21/WS/0146; approval granted November 23, 2021).

Primary outcome. The primary outcome was all-cause mortality during the follow-up period. Outcome data were obtained from the Community Health Index registry, a routinely available registry maintained at a national health board level and populated from both primary and secondary care data. Specific cause of death was not available from this registry.

Inflammatory profiling. NLR (from absolute neutrophil and lymphocyte counts) and mGPS (from CRP and albumin) were calculated based on preoperative blood investigations using previously described methodology.[11,17] Institutional policy during the study period was to admit patients to hospital on the evening prior to surgery, where preoperative blood work was routinely performed as part of existing patient care. NLR and mGPS were then combined into SIG as per Golder et al.[22] The calculation of these inflammation-based prognostic scores is summarized in Table I. Outcomes were compared between groups based on absolute values of SIG.

Statistical analyses. Differences between continuous variables were assessed using the Kruskal-Wallis and Mann-Whitney tests, and differences between categorical variables using the χ^2 test, with linear-by-linear P values reported. Time-to-event analyses were calculated

Table I. The calculation of inflammation-based prognostic scores using preoperative blood results

NLR	Absolute neutrophil count/Absolute lymphocyte count	
mGPS	mGPS 0	CRP ≤10 mg/L
	mGPS 1	CRP >10 mg/L and albumin >35 g/L
	mGPS 2	CRP >10 mg/L and albumin <35 g/L
SIG	SIG 0	mGPS 0 and NLR <3
	SIG 1	mGPS 0 and NLR 3-5
		or
		mGPS 1 and NLR <3
	SIG 2	mGPS 0 and NLR >5
		Or
		mGPS 2 and NLR <3
		or
		mGPS 1 and NLR 3-5
	SIG 3	mGPS 1 and NLR >5
		Or
		mGPS 2 and NLR 3-5
	SIG 4	mGPS 2 and NLR >5

CRP, C-reactive protein; *mGPS*, modified Glasgow Prognostic Score; *NLR*, neutrophil:lymphocyte ratio; *SIG*, systemic inflammatory grade.

using the Kaplan-Meier method, with differences between cohorts assessed using the log-rank *t* test. Where time to event survival data did not reach a median survival, the mean (95% confidence interval [CI]) values are reported. Receiver operating characteristic analysis was performed to determine the predictive value of SIG on 1-year mortality with area under the curve values evaluated. To examine the relative contributions of NLR and mGPS to SIG, 1-year survival rates and % standard error (SE) were compared between subgroups. The association between covariates and overall survival was assessed using a Cox proportional hazards model; covariates were initially interrogated in univariate analysis and those with univariate $P < .05$ were included in a multivariate model. The association between baseline variables and increasing SIG were assessed with multinomial logistic regression, with SIG 0 used as the reference category. All analyses were performed using IBM SPSS 28.0. *P* values $< .05$ were considered statistically significant.

RESULTS

A total of 674 patients were screened for inclusion into the study. Of these, 168 patients were excluded: 60 patients who underwent emergency or urgent repair for ruptured or symptomatic AAA, three patients due to hematological malignancy, one patient due to mycotic aneurysm, three due to no preoperative results available, and 101 patients due to missing preoperative CRP or albumin (Supplementary Fig, online only). This resulted in 506 elective patients with a preoperative SIG who were eligible for inclusion into the final study cohort. There were 108 F/B-EVAR cases (21%), 461 males (91%), and a median age of 74.0 years (interquartile range [IQR], 11.0 years). The median follow-up was 68.0 months (IQR, 27.3 months), and there were 163 deaths (32%) during the follow-up period.

Mean survival in the entire study population was 78.1 months (95% CI, 74.8-81.5 months). Mean survival in the SIG 0 vs SIG 1 vs SIG 2 vs SIG 3 vs SIG 4 subgroups was 80.7 months (95% CI, 76.5-85.0 months) vs 78.7 months (95% CI, 72.7-84.7 months) vs 61.0 months (95% CI, 51.1-70.8 months) vs 65.1 months (95% CI, 45.0-85.2 months) vs 54.9 months (95% CI, 34.4-75.3 months) ($P < .05$) (Fig 1).

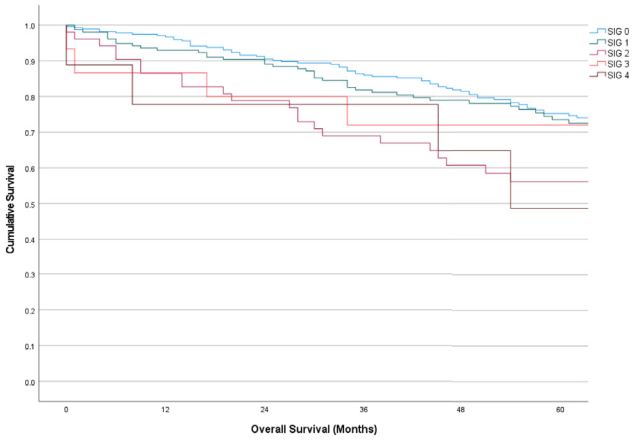
Table II shows the association of baseline covariates with all-cause mortality in the study cohort. On univariate analysis, increasing age ($P < .001$), high creatinine ($P < .05$), and increasing SIG ($P < .05$) were associated with increased mortality, whereas high body mass index ($P < .05$) was associated with decreased mortality. On multivariate analysis, increasing age (hazard ratio [HR], 1.72; 95% CI, 1.29-2.31; $P < .001$), and increasing SIG (HR, 1.20; 95% CI, 1.02-1.40; $P < .05$) were associated with increased mortality.

One-year survival and %SE stratified by NLR and mGPS subgroups is shown in Table III. In the NLR <3 group, there was a significant trend towards inferior survival with increasing mGPS ($P < .05$), whereas in other subgroups, these trends were non-significant.

Receiver operating characteristic analysis to determine the predictive value of SIG on 1-year mortality in the entire study cohort is shown in Fig 2; SIG significantly predicted 1-year mortality (area under the curve, 0.68; 95% CI, 0.58-0.78; $P < .01$).

To account for patients with missing preoperative SIG resulting in selection bias, patients with a missing SIG were compared with the final study cohort (Supplementary Table, online only). There was a lower rate of statin use among the patients with missing SIG (70% vs 80%; $P < .05$), but other characteristics were similar between groups. Survival analysis was performed on this subgroup and compared with the final study cohort; mean survival in the "missing SIG subgroup" was 72.9 months (standard deviation, 67.1-78.8 months), comparable to the final study cohort.

The association between each of preoperative diabetes, ischemic heart disease, hypertension, prior stroke, chronic obstructive pulmonary disease, tobacco smoking, statin use, and juxta-/para-renal aneurysm and increased SIG was examined using multinomial logistic regression. For each covariate of interest, there was no significant association with any magnitude of SIG ($P > .05$ for each).



	Number at Risk					
Months	0	12	24	36	48	60
SIG 0	274	266	250	226	186	135
SIG 1	156	145	141	120	96	76
SIG 2	52	45	41	34	29	21
SIG 3	15	13	12	9	8	6
SIG 4	9	7	6	5	4	3

Fig 1. Kaplan-Meier survival curves and life table for systemic inflammatory grade (*SIG*) (0/1/2/3/4) subgroups in patients undergoing elective endovascular aneurysm repair (EVAR) and fenestrated/branched (F/B)-EVAR for abdominal aortic aneurysms (AAAs). $P < .05$ (log-rank method). *CRP*, C-reactive protein.

DISCUSSION

The results of the present study suggest that markers of the SIR may offer prognostic value in patients undergoing elective EVAR and F/B-EVAR for AAA. These results indicate that activation of the SIR may provide useful routinely available clinical information on long-term outcomes in this patient group. In the present study, the systemic inflammation-based prognostic score of interest was SIG, which incorporates both NLR and mGPS. Prognostication is of paramount importance in the elective subgroup, where procedures are, by their very nature, prophylactic. Therefore, identifying a subgroup of patients in whom long-term cardiovascular outcomes are inferior may allow for direction of patients towards a conservative management strategy and effective allocation of resource. The need for an optimal prognostication tool is highlighted by the preliminary results of the UK-COMPASS trial[27]; where an overall mortality of 21.6% was observed during a median follow-up period of 3 years. This mortality rate is broadly in keeping with the 32% mortality rate over a median follow-up of 5.5 years observed by the present study.

It was of interest that, from visual inspection of the survival plots, SIG identifies subgroups who diverge in their survival within the first 3 years of follow-up. If this were to be the case, then SIG may be useful in identifying those patients who will benefit from intervention rather than conservative management. However, low absolute numbers in certain subgroups during later follow-up intervals may limit the generalizability of these conclusions. The present results, using routine clinical measure of systemic inflammation, may be readily validated in large external cohorts of patients.

In prior studies investigating NLR and AAA, some authors observed a significant association with survival[24,25]; however, this has not been universally reproduced.[23] The literature investigating this association is heterogenous, with inter-study variation in measurement timing, outcome measures, and definition of threshold for "abnormal" NLR. The present study used previously reported values to subgroup based on NLR; however, these were originally described in patients with cancer.[11] Furthermore, few studies reliably report which component of the NLR leads to the reported value (ie whether neutrophilia or lymphopenia predominate). The "normal" mean value of NLR recently reported in a cohort of over 400,000 disease-free controls in the UK Biobank was 2.35,[28] broadly in keeping with the lower threshold used in the present study. A benefit to the implementation of the mGPS to assess the SIR is the predefined subgroups removing inter-study heterogeneity. Although these are derived from continuous variables, the cutoff values for CRP and albumin (10 mg/L and 35 g/L, respectively) are typical "normal" values already used in routine clinical practice. Evaluating both NLR and mGPS allows for a comprehensive assessment of the multiple systems contributing to activation of the SIR, including both the differential white cell and acute phase responses.[29,30]

The mechanism by which chronic systemic inflammation confers increased long-term mortality in patients undergoing intervention for AAA is poorly described. Chronic inflammation is an established risk factor for long-term cardiovascular mortality.[4-7] Patients with AAA typically show other systemic features of cardiovascular morbidity and atherosclerotic disease[31]; indeed in the present study cohort, 35.0% and 12.6% of patients had prior diagnoses of ischemic heart disease and cerebrovascular accident, respectively, and the burden of cardiovascular disease in patients with AAA is well-recognized.[32] Chronic systemic inflammation has emerged as the leading etiological component of the development of atherosclerosis.[33] The underlying role of chronic inflammation in predisposing patients with AAA to increased risk of mortality may be associated with this heightened cardiovascular risk.

The effect of immunomodulation in patients with cardiovascular disease has been investigated; Ridker et al demonstrated that interleukin-1β blockade with canakinumab in patients with ischemic heart disease resulted in a decreased incidence of cardiovascular events

Table II. The effect of clinicopathological characteristics and systemic inflammatory grade (*SIG*) on all-cause mortality in patients undergoing elective endovascular aneurysm repair (*EVAR*) and fenestrated/branched (*F/B)-EVAR* for asymptomatic abdominal aortic aneurysms (AAAs) (n = 506)

		Univariate			Multivariate		
Covariate	No. (%)	HR	95% CI	*P*	HR	95% CI	*P*
Age, years		1.83	1.38-2.44	**<.001**	1.72	1.29-2.31	**<.001**
<65	26 (5)						
65-75	254 (50)						
>75	226 (45)						
Female sex	45 (9)	0.89	0.52-1.52	.67	–	–	–
F/B-EVAR	108 (21)	0.88	0.60-1.30	.54	–	–	–
BMI, kg/m^2		0.69	0.48-0.98	**<.05**	0.81	.56-1.17	.25
<25	100 (20)						
≥25	404 (80)						
ASA		1.27	0.93-1.73	0.14	–	–	–
≤2	282 (56)						
>2	222 (44)						
Statin use	404 (80)	0.71	0.50-1.01	0.06	–	–	–
High creatinine	123 (24)	1.43	1.02-2.00	**<0.05**	1.21	.86-1.71	.28
SIG		1.22	1.04-1.43	**<0.05**	1.20	1.02-1.40	**<.05**
0	274 (54)						
1	156 (31)						
2	52 (10)						
3	15 (3)						
4	9 (2)						

ASA, American Society of Anesthesiologists; *BMI*, body mass index; *CI*, confidence interval; *HR*, hazard ratio.
HR describes hazard of all-cause mortality during follow-up period generated through Cox proportional hazards analysis. For covariates with >2 subgroups, the first category was considered as the reference category.
Boldface *P* indicates statistical significance.

Table III. Percent of 1-year survival in patients undergoing endovascular aneurysm repair (EVAR) and fenestrated/branched (F/B)-EVAR for abdominal aortic aneurysm (AAA) (n = 566) stratified by neutrophil:lymphocyte ratio (*NLR*) (<3, 3-5, and >5) and modified Glasgow Prognostic Score (*mGPS*) (0/1/2) categories

	NLR <3		NLR 3-5		NLR >5		
	n	1-year OS, % (SE, %)	n	1-year OS, % (SE, %)	n	1-year OS, % (SE, %)	
mGPS 0	273	97 (1)	133	93 (2)	22	95 (4)	*P* = .15
mGPS 1	23	91 (6)	27	81 (7)	8	88 (12)	*P* = .56
mGPS 2	4	75 (22)	7	86 (13)	9	78 (14)	*P* = .97
		***P* < .05**		*P* = .08		*P* = .14	

OS, Overall survival; *SE*, standard error.
P generated through linear-by-linear χ^2 analyses.
Boldface P values indicate statistical significance.

(CANTOS).[8] Subsequent analyses have shown additional benefits; for example, a lower incidence of lung cancer.[34] Canakinumab has been investigated in patients with peripheral arterial disease[35]; however, long-term clinically relevant results are not yet reported. An additional promising target for immunomodulation, interleukin-6, is incompletely investigated and requires further prospective evaluation.[36] The cross-sectional results of the present study cannot differentiate between cause and effect. Specifically, it is not clear whether comorbid factors or aneurysm-specific factors drive the systemic inflammatory response, or whether the systemic inflammatory response drives the progression of aneurysmal disease. However, the results of CANTOS[8] would suggest that there may be an inflammatory basis to cardiovascular disease. If this would prove to be the case, then such studies in AAA cohorts may be warranted.

The present study did not identify an association between female sex and prognosis; this is an incompletely understood phenomenon, with some contemporary

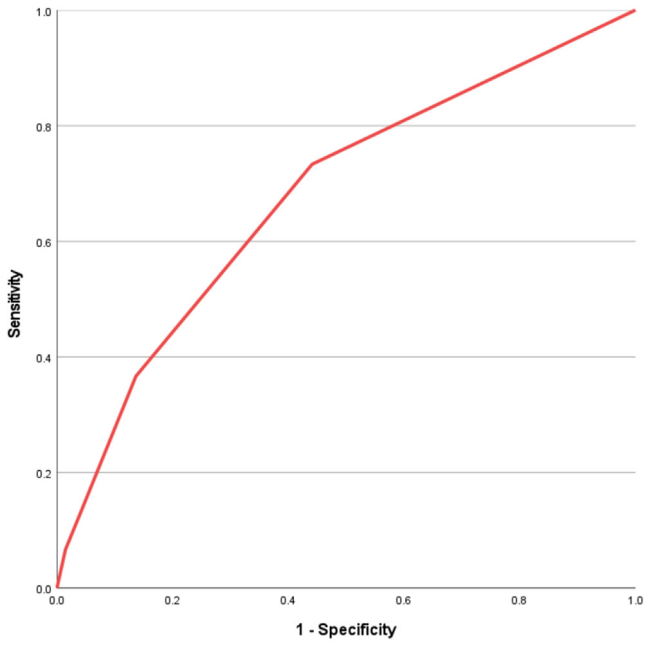


Fig 2. Receiver operating characteristic curve of the predictive value of systemic inflammatory grade (SIG) on 1-year mortality in patients undergoing elective endovascular aneurysm repair (EVAR) and fenestrated/branched (F/B)-EVAR for abdominal aortic aneurysms (AAAs) (n = 506). Area under the curve = 0.68; (95% confidence interval [CI], 0.58-0.78; $P < .001$).

literature reporting comparable mid- and long-term survival between males and females.[37,38] In our patient cohort, there was no significant difference in SIG between males and females ($P = .81$). Small absolute numbers preclude meaningful subgroup analyses of the female patients in the present study. Although we cannot fully assert whether systemic inflammation is responsible for a potential difference in prognosis, this warrants further investigation.

The role of inflammation in the pathogenesis of "conventional" (ie, non-inflammatory/mycotic) AAA has been described, though is incompletely understood.[39-43] Potential novel biomarkers and therapeutic targets may allow for modulation of AAA development and progression. An additional factor influencing postoperative systemic inflammation in patients undergoing EVAR and F/B-EVAR is the pro-inflammatory effect of stent graft deployment,[44] which may contribute to the effect of the inflammatory environment on outcomes.

The natural progression of chronic systemic inflammation in this patient cohort has not been completely defined. Despite this, there is a wealth of evidence observed in patients with cancer (another chronic disease) regarding the prognostic value of activation of the systemic inflammatory response, including multiple systematic reviews and meta-analyses. Given that these studies have been carried out in operable and inoperable cancer (for example, Dolan and colleagues[45,46]) it is likely that the SIR observed is chronic in nature. Therefore, it is of interest that, in cardiovascular disease, although the prognostic value is less well-established, there appears to be an association between atherosclerotic disease burden and magnitude of the SIR in patients with peripheral arterial disease.[47,48] Further study of the association between the magnitude of the SIR and atherosclerotic morbidity/mortality is warranted. Additionally, the magnitude of the postoperative inflammatory response, and its relationship with preoperative chronic systemic inflammation is of interest and requires further investigation.

Limitations. The present study was limited by its retrospective design and missing data for some patients who were otherwise eligible for inclusion; however, steps were taken to assess whether missing preoperative SIG introduced selection bias. There were heterogenous subgroup sizes based on SIG (with low absolute numbers in some subgroups); however, the patient group was representative of real-world practice with a clinically relevant follow-up interval. The specific cause of death was not available, and therefore, limits what can be concluded from the present study. Nevertheless, because the majority of patients would die due to cardiovascular morbidity, this is unlikely to be a major confounding factor in the present analysis in patients with AAA.[32] The present study did not include the use of immunosuppressive or anti-inflammatory medications as a covariate in analyses, which is a potential source of bias. However, it may be expected that patients taking such medications may have a reduction in SIR and may result in an underestimation of the prognostic value of the SIR in the present study.

CONCLUSIONS

SIG is readily calculable from routine preoperative blood investigations and appears to offer prognostic value in patients undergoing endovascular repair of AAA, although these observations require further validation. Further work including serial perioperative inflammatory profiling, and characterization of inflammatory pathways and therapeutic targets is required to accurately define this relationship and to aid perioperative risk prediction.

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AUTHOR CONTRIBUTIONS

Conception and design: NB, CR, DM, GG
Analysis and interpretation: NB, GG
Data collection: NB, AWa, AWi, TS
Writing the article: NB, AWa
Critical revision of the article: AWi, TS, CR, DM, GG

Final approval of the article: NB, AWa, AWi, TS, CR, DM, GG
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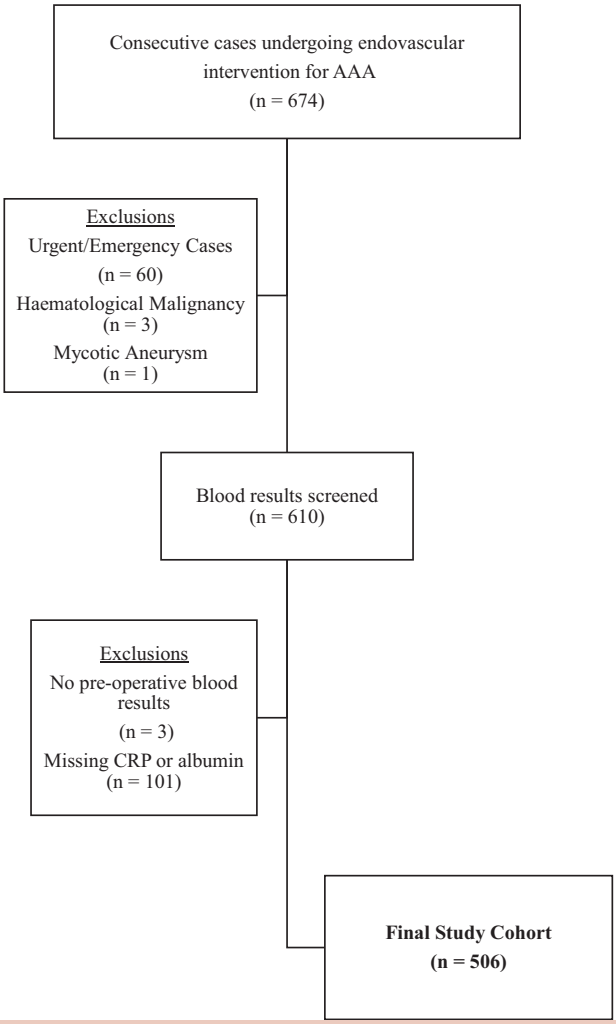
Additional material for this article may be found online at www.jvascsurg.org.

Supplementary Table (online only). Baseline clinical and demographic characteristics of the "missing systemic inflammatory grade (SIG)" patients and the final study cohort in patients undergoing elective endovascular aneurysm repair (*EVAR*) and fenestrated/branched (*F/B*)-*EVAR* for abdominal aortic aneurysm (AAA)

	Final study cohort (n = 506)	"Missing SIG" subgroup (n = 101)	*P*
Age, years			.22
<65	26 (5)	5 (5)	
65-75	254 (50)	57 (58)	
>75	226 (45)	36 (37)	
Sex			.06
Male	461 (91)	95 (96)	
Female	45 (9)	6 (4)	
Diabetes mellitus			.07
Yes	96 (19)	11 (11)	
No	410 (81)	87 (89)	
Ischemic heart disease			.87
Yes	181 (36)	36 (37)	
No	324 (64)	62 (63)	
Hypertension			.34
Yes	345 (68)	62 (63)	
No	161 (32)	36 (37)	
Prior stroke			.67
Yes	64 (13)	14 (14)	
No	440 (87)	84 (86)	
Chronic obstructive pulmonary disease			.69
Yes	123 (24)	22 (22)	
No	382 (76)	76 (78)	
Current tobacco smoking			.09
Yes	114 (23)	30 (31)	
No	389 (77)	68 (69)	
Current statin use			**<.05**
Yes	404 (80)	69 (70)	
No	102 (20)	29 (30)	
F/B-EVAR			.99
Yes	108 (21)	21 (21)	
No	398 (79)	77 (79)	
Body mass index, kg/m^2			.72
<25	100 (20)	21 (21)	
≥25	404 (80)	77 (79)	
ASA			.06
≤2	282 (56)	65 (66)	
>2	222 (44)	33 (34)	
One-year survival			.25
Yes	476 (94)	95 (97)	
No	30 (6)	3 (3)	

ASA, American Society of Anesthesiologists.
Data are presented as number (%).
P generated through linear-by-linear χ^2 analyses comparing proportion of each parameter within each subgroup.



Supplementary Fig (online only). Patient selection into the study. *AAA*, Abdominal aortic aneurysm; *CRP*, C-reactive protein.