Heart & Lung 48 (2019) 446–451

Contents lists available at ScienceDirect

Heart & Lung

journal homepage: www.heartandlung.com

A strong and reliable indicator for early postoperative major cardiac events after elective orthopedic surgery: Aortic arch calcification

Adem Adar, MD [a,*], Orhan Onalan, MD [a], Fahri Cakan, MD [a], Ertan Akbay, MD [a], Tugce Colluoglu, MD [a], Uygar Dasar, MD [b], Tansel Mutlu, MD [b]

[a] Karabuk University Faculty of Medicine, Department of Cardiology, Karabuk, Turkey
[b] Karabuk University Faculty of Medicine, Department of Orthopaedic Surgery, Karabuk, Turkey

ARTICLE INFO

Article history:
Received 2 September 2018
Received in revised form 28 November 2018
Accepted 3 December 2018
Available online 28 December 2018

Keywords:
Orthopedic surgery
Major adverse cardiac events
Aortic arch calcification

ABSTRACT

Background: Cardiovascular events after orthopedic surgery may result in mortality. Therefore, predictors of early cardiovascular events after elective orthopedic surgery are required.
Aim: The aim of this study is to investigate the relationship between aortic arch calcification and 30-day major adverse cardiac events following elective orthopedic surgery.
Methods: Patients who had undergone orthopedic surgery were screened. Preoperative detailed anamnesis was taken. Echocardiography and standard chest x-ray were performed.Patients were followed in terms of perioperative 30-days major cardiac events and were classified into two groups according to development of perioperative major adverse cardiac events.Aortic arch calcification was evaluated by two cardiologists, blinded to study findings and was graded as 0 to 3 on chest x-ray.
Results: A total of 1060 patients were approached for the study participation. Of these 714 were included in the study (mean age: 70.43, 65% female). Cardiovascular events occurred in 33 patients. As compared to the patients without cardiac events, the prevalence of aortic arch calcification, coronary artery disease, hypertension, and smoking were higher in patients with cardiac events. In addition, Lee index, left ventricular end-systolic, end-diastolic and left atrial diameter were significantly higher, GFR values were significantly lower in the group with cardiac events.Multivariate regression analysis showed that smoking (OR 5.031, 95% CI 1.602 to 15.794), presence of hypertension (OR 5.133, 95% CI 1.297 to 20.308) and aortic arch calcification (OR 6.920, 95% CI 3.890 to 12.310) are independent predictors of major cardiac events within 30-day of elective orthopedic surgery.
Conclusions: Presence of aortic arch calcification is associated with development of major cardiac events within 30-days after elective orthopedic surgery.

© 2018 Elsevier Inc. All rights reserved.

Introduction

A cardiac event rate of 4–6% has been reported in patients undergoing elective orthopedic surgery.[1,2] Various risk indices based on multivariate analyses of empirical data, indicating the association between preoperative clinical characteristics and perioperative mortality in patients undergoing non-cardiac surgery, have been developed.[3] The most known indices are Goldman, Detsky, and Lee indices.[3] Recently, a new risk calculation model is developed to estimate intraoperative and postoperative myocardial infarction or cardiac arrest risk by using the National Surgical Quality Improvement Program of American College of Surgeons.[4] Revised Cardiac Index (Lee index), which is recommended by guidelines and frequently used in daily practice, is helpful for the clinicians in the detection of patients with high perioperative cardiovascular risk.[5] The risk index has six variables: type of surgery, history of ischemic heart disease, heart failure, cerebrovascular disease, preoperative insulin treatment, and creatinine level >170 mmol/L (>2 mg/dL).[5] A simple risk indicator may help in the assessment of perioperative cardiac risk in patients undergoing elective orthopedic surgery.

Vascular calcification occurs as a result of calcium accumulation in intima and media layers of vascular structure[6] and it is an important predictor for subclinical atherosclerosis and future cardiovascular events.[7,8] Aortic arch calcification (AAC) indicates the calcification of aortic arch and a part of the descending aorta on standard chest X-ray and it is also closely associated with other vascular bed calcifications.[9] In addition, AAC has been shown to be associated with all component of the revised cardiac index including ischemic heart disease, heart failure, cerebrovascular disease, diabetes mellitus and

* Corresponding author.
E-mail address: aadar@karabuk.edu.tr (A. Adar).

https://doi.org/10.1016/j.hrtlng.2018.12.001
0147-9563/$ - see front matter © 2018 Elsevier Inc. All rights reserved.

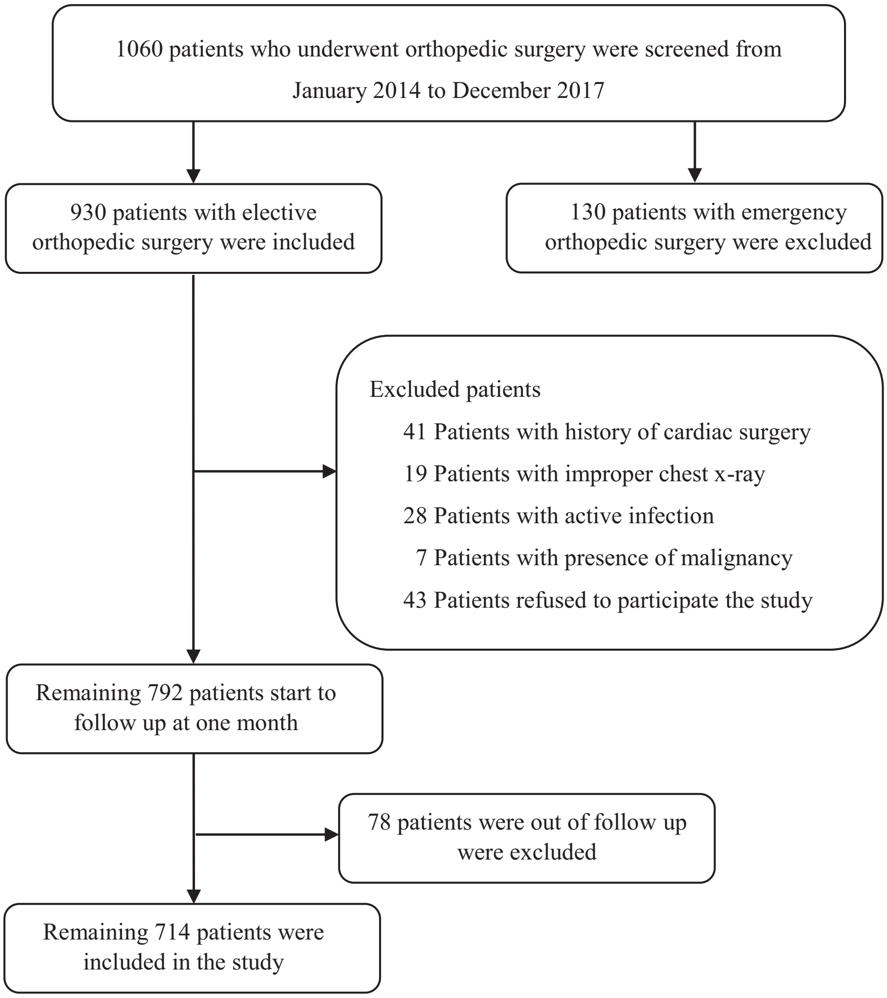


Fig. 1. Flow chart of patients from screening to inclusion.

chronic renal failure.[10–12] To date, no study evaluated the association of AAC with postoperative cardiac events in patients undergoing non-cardiac surgery. The aim of this study was to investigate the association of AAC with 30-day major adverse cardiac events (MACE) following elective orthopedic surgery.

Material methods

One thousand and sixty consecutive patients who underwent an orthopedic surgery were enrolled prospectively. The study was performed at Karabuk University Hospital from January 2014 to December 2017. Inclusion criteria were as follows: (1) age of ≥18 years, (2) willingness to participate in the study, (3) elective orthopedic surgery. Following patients were excluded from the study: (1) emergency orthopedic surgery (130 patients), (2) history of cardiac surgery (41 patients), (3) presence of an active infection (28 patients), (4) presence of malignancy (7 patients), and (5) improper chest X-ray (19 patients). 43 patients refused to participate in the study and 78 patients lost to follow-up. The remaining 714 patients were included in the study (Fig. 1). Adverse cardiac events were defined as an acute coronary syndrome (ST-segment elevation myocardial infarction (STEMI), non- STEMI, unstable angina pectoris (UAP)), decompensated heart failure, new onset atrial fibrillation, stroke and cardiac death within 30-day of surgery.

Patients were evaluated in two groups based on development of perioperative cardiovascular events (Group I: patients with at least one event, Group II: patients without event). Cardiovascular risk factors of all patients were examined. Patients who had previously received oral anti-diabetic and/or insulin treatment or who had fasting blood glucose ≥ 126 mg/dL twice were considered as diabetic. Patients who had previously received antihypertensive treatment or who had a blood pressure of ≥130/80 mm Hg at least twice were considered as hypertensive. Patients with a total cholesterol of >200 mg/dL or a low-density lipoprotein cholesterol (LDL) of >100 mg/dL, or who were using lipid-lowering drugs, were considered as hyperlipidemic.[13] The estimated glomerular filtration rate (GFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.[14] Body mass index (BMI) was calculated by dividing the weight (kg) by the square root of the height (m) squared.[15] Body surface area (BSA) was calculated by the square root of the product of the weight (kg) and height (cm) divided by 3600.[16] The institutional Ethics Committee approved the study protocol and each participant provided written informed consent.

Evaluation of aortic arch calcification

The posterior–anterior chest radiograph (40 cm × 40 cm; Curix HT 1.000G Plus, Agfa, Mortsel, Belgium) was acquired with the patient standing up (Thoramat, Siemens, Erlangen, Germany). The focus–patient distance was 150 cm. An automated exposure control with a fixed tube voltage of 117 kV was used. AAC was graded as follows: grade 0, no visible calcification; grade 1, small spots of

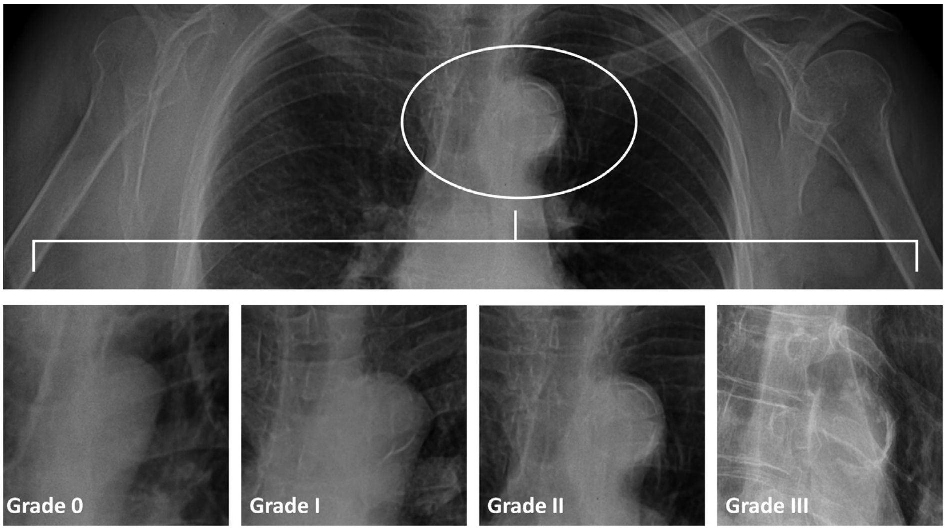


Fig. 2. Grading of aortic arch calcification.

calcification or thin calcification on the aortic arch; grade 2, one or more areas of thickened calcification, and grade 3, circular calcification on the aortic arch[17] (Fig. 2). 100 random selected chest radiography for evaluation of AAC was evaluated by two cardiologists who were blind to the study findings (Kappa value = 0.806 and $p < 0.001$).

Echocardiographic examination

All patients were examined using a commercially available system (Vivid 4; GE Medical Systems, Horten, Norway) with a phased-array 3.5 MHz transducer. The conventional M-mode, B-mode, and Doppler parameters were measured according to the American Society of Echocardiography guidelines. Left ventricular end-diastolic diameter (LVEDD), end-systolic diameter, posterior wall thicknesses (PWT) and interventricular septal wall thicknesses (IVST) were measured. LV mass (LVM) was calculated using the Devereux equation: $LVM = 0.8 \times [1.04(LVEDD + IVST + PWT)\ \ 3 - (LVEDD\ \ 3)] + 0.6$. LVM index (LVMI) was calculated by dividing the LVM by the body surface area. LV hypertrophy was defined as LVMI > 115 g/m^2 for men and >95 g/m^2 for women.[18]

Endpoints

The primary endpoints were a composite of MACE during the first 30-days after surgery including acute coronary syndrome (STEMI, non- STEMI, UAP), decompensated heart failure, new onset atrial fibrillation, stroke, and cardiac death. Perioperative myocardial infarction was not routinely screened, but if suspected, a troponin level > 3x normal 99th percentile level and a rise and/or fall in values with at least one value above the decision level was required for the diagnosis. Patients with ST-segment elevation at the J-point in 2 contiguous leads with the cutoff points ≥ 2 mm in men or ≥1.5 mm in women in leads V2–V3 and/or ≥1 mm in other leads in ≥2 contiguous leads were defined as STEMI. Patients without persistent ST-segment elevation were diagnosed as non-STEMI or UAP based on troponin test. Patients with normal troponin levels were classified as UAP. Patients with high troponin levels were accepted as non-STEMI.[19] Stroke as a permanent focal neurological deficit adjudicated by a neurologist and confirmed by computed tomography or magnetic resonance imaging. Diagnosis of decompensated heart failure was based on dyspnea and positive findings in chest X-ray and/or a diagnosis confirmed by a cardiologist. New onset atrial fibrillation was documented using electrocardiography if clinically indicated.

Statistical analyses

Descriptive analyses were used to define continuous variables. Normally distributed parameters were represented with a mean ± standard deviation values, and non-normally distributed parameters were represented with median (minimum–maximum) values. The relation between the presence of cardiovascular events and categorical parameters were analyzed using Fisher's exact test. For the comparison of continuous variables in patients with and without cardiovascular events; Student *t*-test was used for parameters with normal distribution and Mann–Whitney *U* test was used for parameters with non-normal distribution. In multivariate regression analysis (enter method), the effect size was adjusted for all potential variables with a univariate significance level of <0.10. Adjusted odds ratios (OR), along with their 95% CIs were presented. The cutoff points based on cardiovascular events for statistically significant parameters were calculated by using ROC analysis and the data with $p < 0.05$ were considered to be statistically significant and interpretable. MedCalc Statistical Software version 12.7.7 (MedCalc Software bvba, Ostend, Belgium; http://www.medcalc.org; 2013) was used for the analyses.

Table 1
Clinical and demographic characteristics of the sample.

		Cardiac outcome		*p*
		No 681 (95.4)	Yes 33(4.6)	
Gender, n(%)	Female	443 (65.1)	22 (66.7)	1.00
	Male	238 (34.9)	11 (33.3)	
Age (year)		71 (18–96)	73 (49–94)	0.098
Body mass index (kg/m^2)		28.23 (17–44)	28.7 (20–37)	0.578
Body surface area (m^2)		1.89 ± 0.19	1.93 ± 0.19	0.251
Heart failure, *n*(%)		41 (6.0)	3 (9.1)	0.449
Chronic kidney disease, *n*(%)		72 (10.6)	4 (12.1)	0.771
LEE index (score)		0(0–4)	1(0–4)	**0.014**
Coronary artery disase, *n*(%)		82 (12.0)	10 (30.3)	**0.006**
Diabetes mellitus, *n*(%)		208 (30.5)	13 (39.4)	0.335
Cerebrovascular accident, *n*(%)		40 (5.9)	2 (6.1)	1.00
Smoking, *n*(%)		57 (8.4)	7 (21.2)	**0.022**
Hyperlipidemia, *n*(%)		66 (9.7)	7 (21.2)	0.068
Hypertension, *n*(%)		473 (69.5)	30 (90.9)	**0.006**
Left ventricular hypertrophy,*n*(%)		353 (51.8)	20 (60.6)	0.375

A 2-tailed p value <0.05 was considered statistically significant.

Table 2
Laboratory and echocardiographic findings of the study population.

	Cardiac outcome		p
	No 681 (95.4)	Yes 33 (4.6)	
Left ventricular end-diastolic diameter (mm)	47 (40–68)	50 (39–60)	**0.001**
Left ventricular end-sistolic diameter (mm)	30 (19–56)	32 (22–46)	**0.032**
Left atrial diameter (mm)	36 (25–60)	37 (30–53)	**0.041**
Interventriculer septal thickness (mm)	11 (7–17)	11 (10–14)	0.603
Posterior wall thickness (mm)	11 (8–15)	11 (10–14)	0.896
E/A	0.82 (0.46–2.6)	0.8 (0.47–1.4)	0.222
Left ventricular ejection fraction (%)	61,3 (29–79)	62 (28 –72)	0.657
Left ventricular mass (gr)	192.5 (110–397)	206.8 (136–321)	0.098
Left ventricular mass index (gr/m^2)	101.2 (58–201)	104.5 (66–192)	0.252
Glomerular filtration rate (mL/min/1.73 m^2)	85.6 (20–188)	80.4 (44–101)	**0.010**
Glucose (mg/dL)	118 (56–531)	133 (92–405)	0.165
Urea (mg/dl)	37 (11–222)	42 (21–197)	0.144
Creatinine (mg/dL)	0.8 (0.32–6.53)	0.88 (0.5–3.6)	**0.004**
C-reactive protein, (mg/L)	10 (0.3–323)	22 (0.43–228)	0.205
Total cholesterol (mg/dL)	181 (87–383)	186.6 (90–297)	0.915
Low-density lipoprotein (mg/dL)	106 (37–233)	112 (34–191)	0.884
High-density lipoprotein (mg/dL)	44.3 (15–78,6)	43 (19–62)	0.242
Triglyceride (mg/dL)	132 (39–939)	136 (81–337)	0.294

A 2-tailed p value <0.05 was considered statistically significant.

Results

A total of 1060 patients were screened. Of these, 714 patients were included in the study (mean age: 70.43, 65% female). Prevalence of hypertension, diabetes, hyperlipidemia, coronary artery disease, chronic renal failure and cerebrovascular disease were 70.4%, 31%, 10.2%, 12.9%, 10.6% and 5.9%, respectively (Table 1).

512 patients (71.7%) had a non-fracture surgery including 312 (43.7%) total knee replacement, 157 (22%) total hip arthroplasty, 19 (2.7%) lower extremity amputation and 24 (3.4%) shoulder arthroplasty. The remaining 202 patients (28.3%) had a fracture surgery (lower limb fracture: 21, upper limb fracture: 31, femur neck fracture:150) (Table, supplemental data 1).

Thirty-three patients (4.6%) developed MACE including 9 (27%) NSTEMI, 3 (9%) STEMI, 3 (9%) UAP, 6 (18%) sudden cardiac death, 6 (18%) decompansated heaert failure, 4 (12%) new-onset atrial fibrillation and 2 (6%) cerebrovascular events (Figure, suplemental data 2). Cardiovascular mortality rate was 0.84%. As compared to the patients without a MACE, patients with MACE had significantly higher prevalence of hypertension (90.9% vs. 69.5%; $p = 0.006$), coronary artery disease (30.3% vs. 12.0%; $p = 0.006$), smoking (21.2% vs.57 8.4%; $p = 0.022$), and higher LEE index score (1 vs. 0; $p = 0.014$) (Table 1).

Table 3
Aortic arch calcification grades according to presence of adverse cardiovascular events in the study groups.

	Cardiac outcome		p value
	No 681(95.4)	Yes 33(4.6)	
Aortic arch calcification (n,%)			
Grade 0	242 (35.5)	0 (0)	<**0.001**
Grade 1	290 (42.6)	4 (12.1)	
Grade 2	111 (16.3)	13 (39.4)	
Grade 3	38 (5.6)	16 (48.5)	
Aortic arch calcification grade ≥1 (n, %)	439 (64.5)	33 (100.0)	<**0.001**
Aortic arch calcification grade ≥2 (n, %)	149 (21.9)	29 (87.9)	<**0.001**
Aortic arch calcification grade ≥3 (n, %)	38 (5.6)	16 (48.5)	<**0.001**

A 2-tailed p value <0.05 was considered statistically significant.

Table 4
Logistic regression analysis for development of adverse cardiac events.

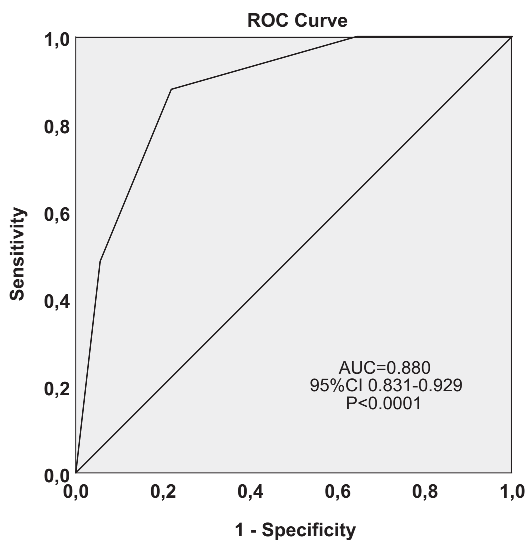
	p	OR	%95 CI lower	%95 CI upper
Age	0.070	0.958	0.914	1.004
Hyperlipidemia	0.394	1.721	0.494	5.994
Left ventricular mass	0.625	1.000	1.000	1.000
Smoking	**0.006**	5.031	1.602	15.794
Hypertension	**0.020**	5.133	1.297	20.308
Aortic arch calcification	<**0.001**	6.920	3.890	12.310
Coronary artery disase	0.080	3.222	0.868	11.953
Glomerular filtration rate	0.136	0.977	0.948	1.007
Left atrial diameter	0.454	1.050	0.925	1.191
Left ventricular end-diastolic diameter	0.078	1.202	0.980	1.051
Left ventricular end-sistolic diameter	0.177	0.896	0.764	1.051
C-reactive protein	0.220	1.005	0.997	1.013
LEE Index				
Score I	0.956	0.968	0.314	2.987
Score II	0.453	0.530	0.101	2.787
Score III	0.064	0.071	0.004	1.167
Score IV	**0.035**	0.020	0.001	0.754

CI: confidence interval, OR: Odds ratio.
A 2-tailed p value <0.05 was considered statistically significant.

Left ventricular end-systolic (median:32 mm and IQR:9 vs. median:30 mm and IQR:6; $p = 0.032$), end-diastolic (median:50 mm and IQR:9 vs. median:47 mm and IQR:5; $p = 0.001$) and left atrial diameter (median:37 mm and IQR:7 vs. median:36 mm and IQR:3; $p = 0.041$) were significantly higher in patienst with MACE. Estimated GFR values were significantly lower in patients with MACE (median: 80.4 mL/min and IQR: 15 vs. median: 85.6 mL/min and IQR: 17; $p = 0.010$, respectively) (Table 2).

In comparison to the patients without fracture, the prevalence of AAC was significantly higher in patients with fractures ($p = 0.009$). Moreover, patients operated for fractures were older ($p < 0.001$) and had a higher BMI ($p = 0.026$), serum creatinine ($p = 0.04$), triglyceride ($p = 0.03$) levels and higher prevalence of smoking ($p = 0.08$). There was no association between MACE and fracture-related orthopedic surgery ($p = 0.695$) (Table, Supplemental data 3).

Age, glucose, urea, creatinine, GFR, C-reactive protein, LVM, AAC, coronary artery disease, hypertension, hyperlipidemia, smoking, Lee index, left ventricular end-diastolic, end-systolic and left atrial



AUC: Area under the curve, AAC: Aortic arch calcification, CI: Confidence interval

Fig. 3. ROC curve of aortic arch calcification for development of advers cardiac events after elective ortopedic surgery.

Table 5
Predictive value of AAC and Lee index for adverse cardiac events.

	AUC	p value	Cut-off	Sensitivity	%95 Lower CI	%95 Upper CI	Specificity	%95 Lower CI	%95 Upper CI
AAC	0.880	<**0.001**	1	87.88	71.8	96.6	78.12	74.8	81.2
LEE index	0.608	**0.036**	0	54.55	36.4	71.9	64.61	60.9	68.2

AUC: Area under curve, CI: Confidance intervale, AAC: Aortic arch calcification.
A 2-tailed p value <0.05 was considered statistically significant.

diameter were associated with adverse cardiac events in univariate logistic regression analysis with a *p*-value of less than 0.1 (Table, supplemental data 4). In multivariate regression analysis; an independent and strong association was found between smoking (OR 5.031, CI 1.602–15.794), hypertension (OR 5.133, CI 1.297–20.308), aortic arch calcification (OR 6.920, CI 3.890–12.310) and development of MACE (Table 4).

Cross-tabulation and correlation analysis were performed to further investigate the potential association between AAC, smoking, and hypertension. Patients with AAC had a higher prevalence of hypertension ($p = 0.007$) but no difference was noted in smoking between the groups ($p = 0.365$). There was a weak correlation between AAC and hypertension ($p = 0.001$, $r = 0.127$) and no correlation was noted between AAC and smoking ($p = 0.398$, $r = 0.032$). These results suggest that hypertension and smoking may not be a significant confounder.

Receiver operator characteristics curve analysis yielded a strong predictive ability of AAC (grade ≥ 1) for the presence of MACE (AUC = 0.880, 95%CI 0.831–0.929, $p < 0.0001$) (Fig. 3). Presence of AAC on chest radiography had a sensitivity and specificity of 88% and 78%, respectively, for the presence of MACE (Table 5). A similar and strong discriminative performance of AAC was noted when ROC analysis was conducted for a grade 2 or higher AAC (AUC = 0.830, 95%CI 0.762–0.898, $p < 0.0001$). An AAC grade of ≥ 2 on chest radiography had a sensitivity, specificity, positive predictive value, negative predictive value and accuracy of 87.88%, 78.12%, 16.29%, 99.25%, and 78.57%, respectively, for the development of MACE.

Discussion

The presence of AAC has been shown to be an important indicator of atherosclerosis and future cardiac events.[20] Currently, no data is available regarding the association of AAC and perioperative MACE in patients undergoing non-cardiac surgery**.** In this study, we found a strong association between the presence of AAC on chest radiography and perioperative 30-day MACE in patients undergoing elective orthopedic surgery.

Advanced age and high prevalence of cardiovascular risk factors led to increased cardiovascular morbidity and mortality in patients undergoing elective orthopedic surgery. In addition, cardiovascular events are known to have a higher prevalence in patients with osteoporosis and high fracture risk.[21] At the same time, fracture risk is reported to be high in patients with cardiovascular diseases. In a study consisted of postmenopausal women, four times higher risk of major cardiovascular events such as myocardial infarction, stroke, and sudden death was reported for women with at least one serious vertebrae fracture in comparison to the women without such fracture.[22,23] Moreover, many studies showed an association between ischemic heart disease, heart failure and high hip fracture.[24,25] Possible reasons for a close association between fracture risk and cardiovascular diseases are considered to be advanced age, smoking, insulin resistance, vitamin D deficiency, decreased sex hormones, disrupted renal functions, inflammation and endothelial disruption**.**[26–29] In the present study, the high prevalence of AAC in patients with fracture have led us to think that vascular calcification may have a pivotal role in the pathogenesis of the association between fractures and cardiovascular diseases. In support of the present study, Szulc et al. reported in the MINOS study that abdominal aortic calcification was associated with high fracture risk in men.[30] In line with the literature, the present study also demonstrated that patients with fractures were older and smoked more.[31]

AAC is associated with arterial stiffness, left ventricular hypertrophy and diastolic dysfunction.[32] Abnormal arterial stiffness and diastolic functions may impair adaptation mechanisms for intravascular volume changes and blood pressure variations in the postoperative period. These changes may facilitate the development of heart failure events in postoperative patients. Moreover, AAC was found to be associated with atherosclerosis, acute coronary syndrome, stroke, MACE and atrial fibrillation.[12,33–36] Consistent with this data, aortic calcification was found to be a predictor of both cardiac and non-cardiac mortality.[37,38]

Predictors that estimate perioperative cardiovascular events for patients who will undergo orthopedic surgery are required during the preoperative assessment. For this reason, Anthony et al. have reviewed 1922 total shoulder arthroplasty patients retrospectively and found that heart failure had led to twelve times higher 30-day mortality.[39] In line with these results, Katsanos et al. Performed the preoperative assessment for 242 patients who will undergo orthopedic surgery. They found that preoperative BNP levels were important indicators for in-hospital mortality and one-year mortality like Goldman, Lee, Detsky and functional capacity index.[40] Guidelines recommend the use of the Lee index for perioperative risk assessment for non-cardiac surgery.[3] Possible reasons for AAC being a better predictors than the Lee index for perioperative adverse cardiac events in the present study: (I) Cardiovascular risk factors used in the Lee index also contributes to the development of AAC by causing vascular damage.[10–12] (II) Lee index is calculated without considering the duration of the risk factors, to what extent they could be controlled and how much end-organ damage they have caused. (III) Instead of solely knowing the presence of the risk factors, the extent of the vascular damage caused by these factors can also be understood after assessment of AAC. (IV) In addition, the present study demonstrated that hypertension and smoking contributed to the development of MACE after orthopedic surgery, however, these risk factors are not taken into consideration in Lee index. Whereas, assessment of AAC could be more valuable as these risk factors also contribute to the development of AAC.[10] (V) Finally, since risk factors like age, sex, inflammation, chronic renal disease, which are not the assessment in the Lee index but affecting development of MACE after orthopedic surgery are also effective in the development of AAC[10,40] and they may explain why AAC is better than Lee index in predicting MACE.

In the present study, we found an independent association between hypertension and postoperative cardiovascular events. In support of the current study, Paweł Łęgosz et al. demonstrated that presence of hypertension was associated with increased cardiovascular risk in patients underwent total knee and hip surgery.[41] In addition, Basim et al. reported that hypertension was associated with postoperative renal failure in patients who had undergone total knee replacement.[42] Moreover, we found that smoking was related with postoperative cardiac events. In line with the present study, Long et al. reported that smoking was associated with postoperative cardiac events in patients undergoing total knee replacement.[43]

AAC is a simple and widely available tool. We demonstrated that AAC is a strong predictor of perioperative MACE in patients undergoing elective orthopedic surgery. Moreover, a discriminative performance of AAC for perioperative MACE was significantly high in our study. Thus, it may have additional benefits in the identification of high-risk patients and preoperative evaluation of the patients undergoing elective orthopedic surgery. Similar studies conducted in other types of surgeries may provide more data on the predictive ability of AAC for MACE.

Limitations

Single-center design of the study, relatively few numbers of patients, the absence of post-operative routine troponin and electrocardiography follow-up and absence of bone densitometer measurements are among the limitations of the study.

Conflict of interest

The authors declare that they have no conflict of interest.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.hrtlng.2018.12.001.

References

1. Tan HB, MacDonald DA, Matthews SJ, Giannoudis PV. Incidence and causes of mortality following acute orthopaedic and trauma admissions. *Ann R Coll Surg Engl*. 2004;86:156–160.
2. Vetrugno L, Langiano N, Gisonni R, et al. Prediction of early postoperative major cardiac events after elective orthopedic surgery: the role of B-type natriuretic peptide, the revised cardiac risk index, and ASA class. *BMC Anesthesiol*. 2014;14:20.
3. Kristensen SD, Knuuti J, Saraste A, et al. 2014 ESC/ESA Guidelines on non-cardiac surgery: cardiovascular assessment and management: The Joint Task Force on non-cardiac surgery: cardiovascular assessment and management of the European Society of Cardiology (ESC) and the European Society of Anaesthesiology (ESA). *Eur Heart J*. 2014;35:2383–2431.
4. Gupta PK, Gupta H, Sundaram A, et al. Development and validation of a risk calculator for prediction of cardiac risk after surgery. *Circulation*. 2011;124:381–387.
5. Lee TH, Marcantonio ER, Mangione CM, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation*. 1999;100:1043–1049.
6. Chen NX, Moe SM. Vascular calcification: pathophysiology and risk factors. *Curr Hypertens Rep*. 2012;14:228–237.
7. Greenland P, LaBree L, Azen SP, Doherty TM, Detrano RC. Coronary artery calcium score combined with Framingham score for risk prediction in asymptomatic individuals. *JAMA*. 2004;291:210–215.
8. Mackey RH, Venkitachalam L, Sutton-Tyrrell K. Calcifications, arterial stiffness and atherosclerosis. *Adv Cardiol*. 2007;44:234–244.
9. Adar A, Erkan H, Gokdeniz T, Karadeniz A, Cavusoglu IG, Onalan O. Aortic arch calcification is strongly associated with coronary artery calcification. *Vasa*. 2015;44:106–114.
10. Iribarren C, Sidney S, Sternfeld B, Browner WS. Calcification of the aortic arch: risk factors and association with coronary heart disease, stroke, and peripheral vascular disease. *JAMA*. 2000;283:2810–2815.
11. Li LC, Lee YT, Lee YW, Chou CA, Lee CT. Aortic arch calcification predicts the renal function progression in patients with stage 3 to 5 chronic kidney disease. *Biomed Res Int*. 2015;2015:131263.
12. Yang TL, Huang CC, Huang SS, Chiu CC, Leu HB, Lin SJ. Aortic arch calcification associated with cardiovascular events and death among patients with acute coronary syndrome. *Acta Cardiol Sin*. 2017;33:241–249.
13. National Cholesterol Education Program Expert Panel on Detection E, Treatment of High Blood Cholesterol in A. Third report of the national cholesterol education program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143–3421.
14. Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. *Ann Intern Med*. 2009;150:604–612.
15. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser*. 2000;894:i–xii. 1–253.
16. Mosteller RD. Simplified calculation of body-surface area. *N Engl J Med*. 1987;317:1098.
17. Symeonidis G, Papanas N, Giannakis I, et al. Gravity of aortic arch calcification as evaluated in adult Greek patients. *Int Angiol*. 2002;21:233–236.
18. Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification: a report from the American society of echocardiography's guidelines and standards committee and the chamber quantification writing group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr*. 2005;18:1440–1463.
19. Smith JN, Negrelli JM, Manek MB, Hawes EM, Viera AJ. Diagnosis and management of acute coronary syndrome: an evidence-based update. *J Am Board Fam Med*. 2015;28:283–293.
20. Witteman JC, Kok FJ, van Saase JL, Valkenburg HA. Aortic calcification as a predictor of cardiovascular mortality. *Lancet*. 1986;2:1120–1122.
21. Farhat GN, Newman AB, Sutton-Tyrrell K, et al. The association of bone mineral density measures with incident cardiovascular disease in older adults. *Osteoporos Int*. 2007;18:999–1008.
22. Genant HK, Wu CY, van Kuijk C, Nevitt MC. Vertebral fracture assessment using a semiquantitative technique. *J Bone Miner Res*. 1993;8:1137–1148.
23. Tanko LB, Christiansen C, Cox DA, Geiger MJ, McNabb MA, Cummings SR. Relationship between osteoporosis and cardiovascular disease in postmenopausal women. *J Bone Miner Res*. 2005;20:1912–1920.
24. Sennerby U, Melhus H, Gedeborg R, et al. Cardiovascular diseases and risk of hip fracture. *JAMA*. 2009;302:1666–1673.
25. van Diepen S, Majumdar SR, Bakal JA, McAlister FA, Ezekowitz JA. Heart failure is a risk factor for orthopedic fracture: a population-based analysis of 16,294 patients. *Circulation*. 2008;118:1946–1952.
26. Carbone L, Buzkova P, Fink HA, et al. Hip fractures and heart failure: findings from the cardiovascular health study. *Eur Heart J*. 2010;31:77–84.
27. Sanada M, Taguchi A, Higashi Y, et al. Forearm endothelial function and bone mineral loss in postmenopausal women. *Atherosclerosis*. 2004;176:387–392.
28. Koh JM, Khang YH, Jung CH, et al. Higher circulating hsCRP levels are associated with lower bone mineral density in healthy pre- and postmenopausal women: evidence for a link between systemic inflammation and osteoporosis. *Osteoporos Int*. 2005;16:1263–1271.
29. Elsheikh N, Sherif N, Zeid SA, Eldamarawy M, Ali A, Sabry AI. The link between bone disease and cardiovascular complications in hemodialysis patients. *Electron Physician*. 2016;8:2483–2488.
30. Szulc P, Kiel DP, Delmas PD. Calcifications in the abdominal aorta predict fractures in men: MINOS study. *J Bone Miner Res*. 2008;23:95–102.
31. Weaver CM, Gordon CM, Janz KF, et al. The national osteoporosis foundation's position statement on peak bone mass development and lifestyle factors: a systematic review and implementation recommendations. *Osteoporos Int*. 2016;27:1281–1386.
32. Cho IJ, Chang HJ, Park HB, et al. Aortic calcification is associated with arterial stiffening, left ventricular hypertrophy, and diastolic dysfunction in elderly male patients with hypertension. *J Hypertens*. 2015;33:1633–1641.
33. Kalsch H, Lehmann N, Moebus S, et al. Aortic calcification onset and progression: association with the development of coronary atherosclerosis. *J Am Heart Assoc* 2017;6.
34. Elias-Smale SE, Odink AE, Wieberdink RG, et al. Carotid, aortic arch and coronary calcification are related to history of stroke: the rotterdam study. *Atherosclerosis*. 2010;212:656–660.
35. Fusaro M, Gallieni M, Rebora P, et al. Atrial fibrillation and low vitamin D levels are associated with severe vascular calcifications in hemodialysis patients. *J Nephrol*. 2016;29:419–426.
36. Okada H, Tada H, Hayashi K, et al. Aortic Root calcification score as an independent factor for predicting major adverse cardiac events in familial hypercholesterolemia. *J Atheroscler Thromb*. 2018;25:634–642.
37. Thomas IC, Thompson CA, Yang M, et al. Thoracic aorta calcification and noncardiovascular disease-related mortality. *Arterioscler Thromb Vasc Biol*. 2018;38:1926–1932.
38. Rodondi N, Taylor BC, Bauer DC, et al. Association between aortic calcification and total and cardiovascular mortality in older women. *J Intern Med*. 2007;261:238–244.
49. Anthony CA, Westermann RW, Gao Y, Pugely AJ, Wolf BR, Hettrich CM. What are risk factors for 30-day morbidity and transfusion in total shoulder arthroplasty? A review of 1922 cases. *Clin Orthop Relat Res*. 2015;473:2099–2105.
40. Katsanos S, Babalis D, Kafkas N, et al. B-type natriuretic peptide vs. cardiac risk scores for prediction of outcome following major orthopedic surgery. *J Cardiovasc Med (Hagerstown)*. 2015;16:465–471.
41. Legosz P, Kotkowski M, Platek AE, et al. Assessment of cardiovascular risk in patients undergoing total joint alloplasty: the CRASH-JOINT study. *Kardiol Pol*. 2017;75:213–220.
42. Hassan BK, Sahlstrom A, Dessau RB. Risk factors for renal dysfunction after total hip joint replacement; a retrospective cohort study. *J Orthop Surg Res*. 2015;10:158.
43. Long G, Suqin S, Li G, Weihong Y, Zhenhu W. Impact of atrial fibrillation on postoperative outcomes after total knee arthroplasty-a retrospective study. *J Orthop Sci*. 2016;21:652–657.