Received: 21 March 2024 | Revised: 25 April 2024 | Accepted: 6 May 2024

DOI: 10.1002/ags3.12816

AGSurg Annals of Gastroenterological Surgery Open Access WILEY

REVIEW ARTICLE

Abdominal aortic calcification among gastroenterological and transplant surgery

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Funding information
Japan Society for the Promotion of Science, Grant/Award Number: JP22K16535 and JP23H02981; Japan Agency for Medical Research and Development, Grant/Award Number: 24fk0210108

Abstract

This review discusses the increasing global trend towards an aging population, which has resulted in a growing number of surgeries being performed on elderly patients, particularly those living with cancer. The focus was on the implications of abdominal aortic calcification (AAC), an indicator of systemic atherosclerosis, in these patients. This comprehensive review provided evidence detailing the complex processes of atherosclerosis and vascular calcification and various approaches to assess this condition. The prevalence of AAC is related to multiple factors, including cardiovascular disease, inflammation, frailty in various types of gastroenterological surgery. Additionally, notable links were found between AAC, postoperative complications, and patient survival following gastroenterological surgery. This study highlights how AAC could negatively impact the health status of elderly patients and undermine treatment efficacy, stressing the need for more research in this domain to improve patient outcomes.

KEYWORDS
abdominal aortic calcification, gastroenterological surgery, vascular calcification

1 | INTRODUCTION

There has been a global increase in both life and healthy life expectancies.[1] This is particularly evident in developed countries, which are experiencing a growing trend towards an aging population structure, as reported in the World Population Prospects 2022 (https://population.un.org/wpp/Download/Standard/Population/). As a result, the number of surgeries performed in elderly patients is expected to increase. In Japan, the proportion of gastroenterological surgeries performed on patients aged ≥80 years has exceeded 20%, and the proportion of surgical cases performed on patients aged ≥70 years has exceeded half, resulting in an increase in the number of surgeries performed on elderly patients over the past few years.[2] It is worth noting that elderly patients diagnosed with cancer are at increased risk of multiple health problems that add to their vulnerability. These include multimorbidity and geriatric syndromes, such as malnutrition, sarcopenia, and frailty, all of which are known to increase adverse outcomes, such as all-cause mortality and cardiovascular mortality.[3]

Atherosclerosis is a systemic, chronic, and progressive disease that can affect the entire vascular tree.[4] The presence of calcium in arterial walls is considered a direct marker of atherosclerosis and

can be readily assessed using computed tomography (CT).[5] In the context of thoracic surgery, recent investigations have focused on thoracic aortic calcium (TAC), an important subclinical marker of atherosclerosis.[6]

At the same time, an increase in studies investigating abdominal aortic calcification (AAC) has been observed in gastroenterological and transplant surgical practices.

Given that comorbidities related to atherosclerosis can increase the risk of mortality in the elderly, any short- and long-term postoperative complications could potentially worsen their health status and undermine the treatment efficacy. In this context, the intersection of atherosclerosis, postoperative complications, and cancer in patients undergoing surgery is an important research focus. Therefore, this study aimed to consolidate the current understanding of the adverse effects of AAC in these patient groups and to highlight the remaining research gaps.

2 | ATHEROSCLEROSIS AND AORTIC CALCIFICATION

2.1 | Process of calcification

Atherosclerosis is a common pathological condition (as summarized in Figure1). The persistence of risk factors for vascular events can induce vascular endothelial dysfunction and increase permeability, allowing apolipoproteins to infiltrate the intima and undergo oxidation. The formation of oxidized apolipoprotein and endothelial expression of cellular adhesion molecules, monocyte chemotactic protein 1, and other chemokines provoke the migration of monocytes into the intima and their differentiation into macrophages. These macrophages release additional inflammatory cytokines and extracellular matrix molecules, and take up oxidized lipoproteins to transform into lipid-laden foam cells.[7] When the number of oxidized lipoproteins taken up by macrophages exceeds their clearance capacity, foam cells undergo apoptosis and shed lipoproteins and other cellular components. This leads to formation of an extracellular lipid core and facilitates plaque accumulation within the arterial wall. Ultimately, atheromatous plaques undergo several processes including hypoxia, neovascularization, and microcalcification. The resulting microcalcification crystals initiate a positive feedback loop that further stimulates the proinflammatory response of macrophages and spreads the pro-calcific stimulus within the arterial wall.[8]

Vascular calcification (VC) is a complex, organized, regulated, and active process similar to bone formation. The understanding that chronic inflammation contributes significantly to the initiation and progression of atherosclerosis has stimulated the investigation of the inflammatory factors that induce calcification in atherosclerotic plaque lesions. Evidence suggests that macrophage-derived inflammatory cytokines such as interleukin-1β (IL-1β), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and oncostatin M (OSM) play a critical role in the transformation of vascular smooth muscle cells into osteoblast-like cells and subsequent calcification of the extracellular matrix.[9–11]

VC is a two-step process, consisting of an initial stage of microcalcification followed by a subsequent stage leading to macroscopic calcium formation (macrocalcification).[12] Depending on its location, VC can be divided into two distinct forms: intimal calcification (within the intima) and medial calcification (within the medial layer of the vessel). The pathogenesis and clinical implications of calcification within the intimal and medial layers of arteries are thought to be different.[13] The intimal layer, which is composed of endothelial cells, undergoes various processes that lead to the formation of atheromatous plaques, potentially causing plaque rupture and subsequent thromboembolic events,[14] whereas the

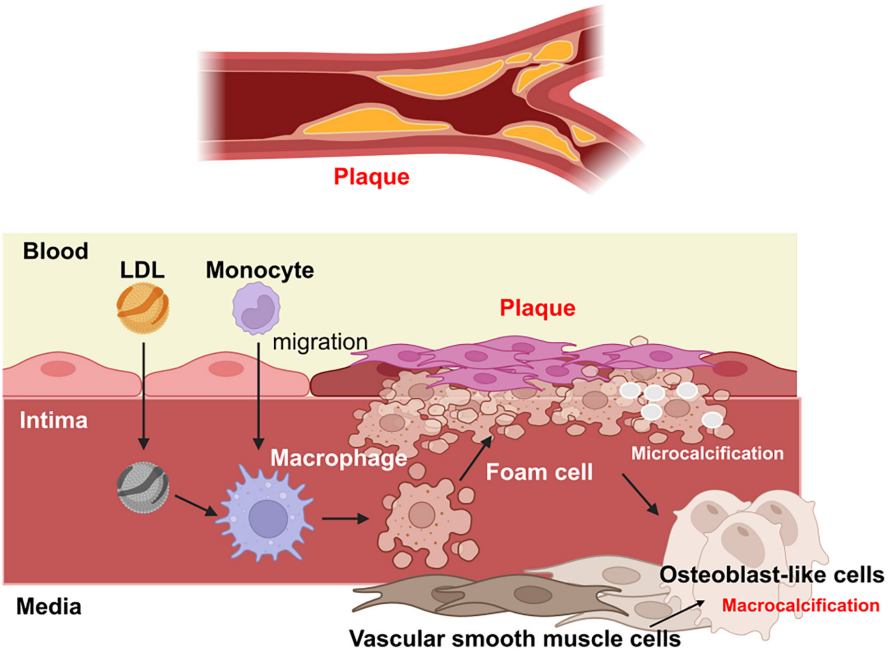

FIGURE 1 The process of vascular calcification. Edited by biorender.

medial layer is composed of smooth muscle cells and elastic fibers that are involved in the regulation of blood flow and arterial pressure. Medial calcification is thought to cause arterial stiffening, reduced compliance, and limited distensibility. Currently, ex vivo histological analysis is the gold standard for differentiating intimal and medial calcifications.[13]

2.2 | Assessment of aortic calcification

Typical indicators of atherosclerosis include carotid intima-media thickness (IMT), subclavian stenosis (SS), abdominal aortic calcification (AAC), thoracic artery calcification (TAC), cardiac-ankle vascular index (CAVI), and ankle-brachial index (ABI).[15,16] AAC can be conveniently assessed using lateral radiographs of the lumbar spine and lateral spine scans obtained using dual-energy X-ray absorptiometry (DXA).[17,18] The AAC-24, a 24-point semiquantitative score, is used to assess AAC and reflects the extent of calcification at the posterior and anterior aortic walls adjacent to the first four lumbar vertebrae.[17] In 2006, Schousboe et al. proposed a simplified 8-point semiquantitative score (AAC-8) based on the original AAC-24 score.[18] Of note, AAC-8 is less influenced by microcalcifications distributed across segments and is faster to apply. Quantitative computed tomography (QCT) is another approach to assess AAC.[19,20] There was a significant correlation between AAC severity assessed using DXA scans and AAC quantified using QCT.[21] However, the results of these two methods are not interchangeable. QCT provides more accurate quantification of AAC than semiquantitative scores on radiographs or DXA scans. The Agatston score, which is calculated as the product of the calcified lesion area and calcium score (reflecting the average density expressed in Hounsfield units [HU]), determines the calcification burden[22] (Figure 2). The AAC evaluations are summarized in Table 1.

In the field of gastroenterological surgery, plain and contrast-enhanced CT scans are often performed for preoperative evaluation, allowing simultaneous AAC assessment. Additionally, CT is routinely performed after cancer surgery for follow-up and recurrence monitoring. The frequency of CT scans in these procedures provides ample opportunities for the visual evaluation of the severity of AAC, which can be measured precisely to facilitate efficient screening processes. This provides an ideal opportunity for both prospective and retrospective AAC assessments, making it suitable for research on gastroenterological surgery cases. Patients with advanced age or severe atherosclerosis, both risk factors for increased AAC levels, may also have additional risk factors such as obesity, smoking, alcohol consumption, diabetes, dyslipidemia, and a history of cardiovascular disease. These additional risk factors classify these patients as high-risk groups, making them more susceptible to short-term complications and poor long-term prognoses. Furthermore, if AAC is present at a young age, the patient may require a drastic lifestyle change and may be part of a hidden high-risk group. Automatic quantification of AAC can be achieved by either X-ray or CT facilitated by artificial intelligence, which could potentially become routine practice.[23]

2.3 | Cardiovascular disease and other complications

AAC tends to increase with age and is correlated with traditional cardiovascular risk parameters.[24,25] Furthermore, there were independent associations between AAC progression and factors such as age, baseline AAC, diabetes mellitus, body mass index (BMI), systolic blood pressure, and pulse pressure.[26–28] Notably, risk factors appear to manifest differently in men and women. A Korean study showed that AAC was associated with smoking in men and diabetes and hypertension in women.[29] Calcification contributes to arterial stiffness, thereby affecting the standard arterial physiology, including Windkessel function.[30] AAC correlates with vascular function tests such as pulse wave velocity (PWV)[30,31] and low ankle brachial index (ABI).[32] In addition, the length of the AAC, as seen on radiographs, is associated with the

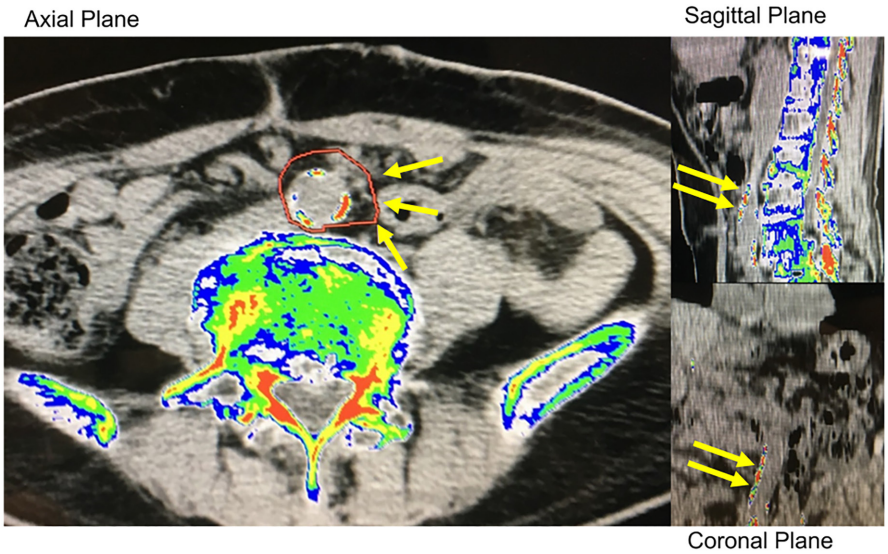

FIGURE 2 Abdominal aortic calcification evaluation using computed tomography scans.

TABLE 1 Abdominal aortic calcification (AAC) assessment methods.

Methods	Device	Characteristics
AAC-24[17]	Radiographs or DXA scans	Evaluate the AAC and determine the level of calcification present at the posterior and anterior aortic walls close to the initial four lumbar vertebrae
AAC-8[18]	Radiographs or DXA scans	Less influenced by microcalcifications that are distributed across various segments and simple to apply
AAC volume[19,20]	Plane CT scan	More accurate quantification of AAC
Agatston Score[22]	Plane CT scan	Calculated as the product of the calcified lesion area and the calcium score, reflecting the calcification burden
Other[68,82]	Plane CT scan	Scored for the presence of branch calcification in addition to AAC

height of the PWV.[33] Therefore, AAC measurements may serve as useful markers for the assessment of systemic atherosclerosis.[34] The Multiethnic Study of Atherosclerosis highlighted the relationship between AAC quantified using CT, coronary artery calcium (CAC), and new cardiovascular events after a mean follow-up of 5.5 years in 1974 participants without cardiovascular disease (CVD) at baseline.[16] When AAC and CAC were adjusted for each other in the multivariate analysis, the association between AAC and CVD and total mortality was stronger than that of CAC.

Despite these associations, only a limited number of studies have examined the relationship between non-alcoholic fatty liver disease (NAFLD) and VC. NAFLD has been reported to be significantly associated with AAC,[35–37] whereas other reports suggest that TAC more directly reflects NAFLD.[38] The AAC score is associated with coronary heart disease and cerebral infarction morbidity in patients with type 2 diabetes mellitus. Low BMI and Fib-4 index >2.67, which is known as a noninvasive fibrosis score, are potential indicators of AAC in this population.[39] Patients with a history of coronary artery disease (CAD) before liver transplantation have an increased risk of death from any cause, particularly cardiovascular death, and this risk is particularly increased by the coexistence of NAFLD.[40] In addition, in a cohort of 98 patients with inflammatory bowel disease (IBD), more than one-third had moderate-to-severe AAC.[41]

The abdominal aorta plays a critical role in maintaining constant peripheral blood flow through the Windkessel effect.[42] AAC can decrease vascular elasticity, leading to fluctuations in blood pressure and unstable blood flow to organs. Thus, high AAC levels can affect peripheral tissue perfusion and prolong wound healing at anastomotic sites.[43] Tissue ischemia leading to anastomotic leakage (AL) is thought to result from a combination of generalized vascular disease and inadequate local perfusion.[44] For example, after biliary reconstruction, biliary blood flow is dependent on the hepatic artery and biliary complications can increase due to atherosclerosis.[45] Furthermore, even in adults without manifest CVD, AAC has been associated with calcification in other arterial beds, such as the superior mesenteric, celiac, coronary, and iliac arteries, which are critical for gastrointestinal function, even in adults without manifest CVD.[46]

Thus, the impact of AAC on cardiovascular events, lifestyle-related diseases including NAFLD, and blood flow failure is significant, suggesting that AAC may have a direct or indirect negative impact on the postoperative outcomes of gastroenterological surgery.

2.4 | Inflammatory and frailty

A strong association between AAC and systemic inflammation has been reported. In a cross-sectional study involving 3036 representative participants, Xie et al.[47] demonstrated positive correlations between the Systemic Immune-Inflammation Index (SII) and AAC. They noted an age-dependent significance in this association, suggesting that elevated levels of systemic inflammation may increase the risk of AAC in the elderly population. This suggests that the SII may have clinical relevance in diagnosing AAC risk and determining disease severity. Results from a cohort study of 97 patients with chronic kidney disease showed a significant association between elevated serum high-sensitivity C-reactive protein levels and AAC progression. This association was particularly pronounced in obese participants.[48]

In addition, calcified atherosclerotic lesions can release local and systemic osteochondrogenic factors that can influence both regional and systemic bone homeostasis.[49] This highlights the potential association between AAC and the overall burden of atherosclerotic disease compared to the CAC score, which is primarily a marker of coronary atherosclerosis.[49] There was a positive linear relationship between VC severity and frailty in older adults. These findings suggest that early diagnosis and treatment of VC could contribute to risk reduction in frail patients.[50] According to Lee et al.,[51] among 9223

asymptomatic adults (mean age 57 ± 7 years [SD], 5152 women, and 4071 men), muscle weakness and aortic calcification had the highest diagnostic power for predicting death. The best predictors of mortality risk were muscle weakness in men and aortic calcification in women, using the Agatston score.

Nakano et al.[52] using atherosclerosis model mice (ApoE−/−) and C57BL/6J wild-type mice (WT) and showed that T cells and natural killer cells in the liver of ApoE−/− mice exhibited more inflammatory phenotypes than WT mice, especially after reperfusion.

Although further studies are needed on the association between AAC and anti-tumor immunity, Imaoka et al.[53] showed that a high low-density lipoprotein/high-density lipoprotein (LDL/HDL) cholesterol ratio and AAC, both associated with atherosclerosis, are strongly inversely correlated with the expression of tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) expressed on natural killer cells in the liver. In addition, they noted the presence of soluble death receptors, such as osteoprotegerin (OPG), implicated in VC, that can bind to extracellular TRAIL, thereby preventing TRAIL from stimulating death receptors.[54,55]

The fragility of individual patients, including sarcopenia[56–58] and chronic inflammation,[59,60] has been extensively studied in gastroenterological, hepatobiliary, and pancreatic cancers, and is known to be strongly associated with the risk of cancer recurrence and postoperative complications. Aspects of atherosclerosis severity, including AAC, should be included in future analyses to assess combined risks.

3 | THE ASSOCIATION OF AAC WITH CLINICAL OUTCOME

Table 2 summarizes the clinical impact of AAC on the postoperative outcomes of gastroenterological and transplant surgeries.

3.1 | Upper gastrointestinal surgery

In the field of esophageal cancer surgery, a comprehensive study by Koyanagi et al.[61] investigated the influence of indocyanine green (ICG) blood flow velocity within the gastric tube wall and AAC on AL prediction after esophagectomy in a cohort of 119 patients. The results highlighted both a blood flow velocity below 0.7 cm/s ($p < 0.01$) and superior mesenteric artery (SMA) calcification ($p = 0.03$) as significant independent predictors of leakage. Interestingly, SMA calcification also showed a significant correlation with ICG fluorescence blood flow velocity within the gastric tube ($p = 0.03$).

In a separate retrospective study, Van Rossum et al.[44] reviewed the preoperative CT scans of 246 patients undergoing McKeown esophagectomy and manually calculated the calcification scores at various arterial sites. Multivariate regression analysis of the individual scores revealed an association between AL and both minor (OR: 2.00, 95% CI: 1.02–3.94) and major (OR: 2.87, 95% CI: 1.22–6.72) aortic calcifications.

This finding was confirmed in a study of 167 patients who underwent Ivor Lewis esophagectomy by Goense et al.[62] using a similar scoring method. Borggreve et al.[63] elucidated the association between AL and calcification in the coronary, supra-aortic, and thoracic arteries.

Zhao et al.[64] studied 673 post-McKeown esophagectomy patients in a Chinese population. Rather than estimating the degree of arterial calcification, they used a simple binary scoring system based on the presence or absence of calcification and reported a significantly higher rate of AL in patients with calcification of the aorta, celiac trunk, and right/left postceliac arteries.

In contrast, Jefferies et al.[65] found no statistically significant association between arterial calcification and AL in a retrospective analysis of preoperative CT scans from 411 patients undergoing esophagogastric anastomosis. Tzortzakakis et al.[66] emphasized the importance of radiological assessment, including the evaluation of aortic calcification and stenosis of its branches, to identify potential risk factors associated with AL in esophageal cancer surgery.

In a separate study on gastric cancer, a retrospective data review was performed on 30 patients with esophagojejunal anastomosis (EJA) complications due to total resection compared to a matched group without complications. (10) The study concluded that there was no significant association between AAC and EJA complications ($p = 0.44$). However, the study showed a positive correlation between SMA calcification and EJA complications in the complicated group ($p = 0.02$). Furthermore, larger SMA calcifications were predominantly associated with anastomotic stenosis rather than leakage, a phenomenon detected in 13% of 23 leakage cases and 28.6% of seven stenosis cases ($p = 0.03$). Finally, a multivariate analysis of 856 gastrectomies presented by Tao et al.[67] reported that aortic calcification was an independent predictor of AL ($p = 0.03$, OR: 2.43, 95% CI = 1.10–5.49).

Taken together, these studies contribute to a better understanding of the intricate role of arterial calcification and blood flow velocity in relation to surgical complications in gastric and esophageal surgery. Further exploration of these associations can pave the way for strategies that can significantly reduce postoperative complications and improve patient outcomes.

3.2 | Hepatopancreaticobiliary surgery

Kakizawa et al.[68] performed a thorough analysis of postoperative outcomes after pancreaticoduodenectomy (PD) in patients aged ≥70 years. Their results highlighted two distinct risk factors for clinically relevant postoperative pancreatic fistula (CR-POPF): a body mass index (BMI) greater than 25 (OR 29.4, 95% CI 5.77–150) and the presence of a high AAC (OR 10.8, 95% CI 2.08–56.6). In contrast, Imaoka et al.[69] performed a retrospective analysis of 214 patients who underwent major hepatopancreatic biliary surgery (HPB)

TABLE 2 Summary.

Report	Year	Procedure	Case number	Factor	Findings
Upper GI					
Van Rossum et al.[44]	2015	McKeown esophagectomy	246	Major and minor aortic calcification	AL (an aortic calcification score of 1 or 2 (OR, 2.00 and OR, 2.87))
Goense et al.[62]	2016	Ivor Lewis esophagectomy	167	Major and minor aortic calcification	AL (an aortic calcification score of 1 or 2 (OR, 5.35 and OR, 7.01))
Zhao et al.[64]	2016	McKeown esophagectomy	673	Calcifications of the supplying arteries of the gastric tube	AL (calcification of aorta (OR, 2.30) and calcification of celiac axis (OR, 1.82))
Borggreve et al.[63]	2018	Esophagectomy	406	Calcification of the supra-aortic arteries	AL (minor calcification of the coronary arteries (OR, 2.29) and calcification of the supra-aortic arteries (OR, 2.48 for minor calcification and OR, 2.72 for major calcification))
Jefferies et al.[65]	2019	Esophagectomy	414	Arterial calcification	AL and/or conduit necrosis (N.S.)
Koyanagi et al.[61]	2021	Esophagectomy	109	SMA calcification	AL (ICG fluorescence blood flow speed of the gastric conduit wall of 2.07 cm/s or less (OR, 44.5) and SMA calcification (OR, 9.09; $p=0.02$))
Tao et al.[67]	2021	Gastrectomy	856	Aorta calcification	AL (Aorta calcification (OR, 2.43))
HPB					
Kakizawa et al.[68]	2018	Pancreaticoduodenectomy	271 (70 yo <)	AAC and branch calcification	Postoperative pancreatic fistula (BMI ≥25 (OR, 29.4) and a high ACC score (OR, 10.8))
Imaoka et al.[69]	2022	Major HPB surgery	214	AAC	Comprehensive complication index (CCI ≥40) (higher AAC (OR, 10.21))
Imaoka et al.[70]	2022	Hepatectomy for HCC	202	AAC	OS (high AAC (HR,2.51)), RFS (high AAC (HR, 1.69))
Watanabe et al.[43]	2023	Surgery for biliary tract cancer	97	AAC	Severe complication (CD grade ≥ III) (high AAC (OR,1.29))
Imaoka et al.[71]	2023	Hepatectomy for colorectal cancer	99	AAC	OS (high AAC (HR, 2.22), mGPS (≥1) (HR, 2.05) and high CA19-9 (HR, 2.89) RR (high AAC (HR, 2.14), TBS (≥3.6) (HR, 1.96), and CA19-9 (HR, 2.86))
Lower GI					
Eveno et al.[78]	2016	Colorectal surgery	60	AAC	AL rate (grade 0: 0%, grade 1: 18%, grade 2: 44%)
Shen et al.[75]	2019	Anterior resection of rectal cancer	423	AAC	AL (high AAC (HR, 2.39))
Deguelte et al.[79]	2021	Colorectal surgery	141	AAC	AL (high AAC (OR, 1.80))
Namba et al.[80]	2021	Colorectal surgery	147	AAC	AL (high AAC (OR, 6.09)
Knight et al.[83]	2021	Colorectal cancer resection	231	AAC	Clinical complete response to neoadjuvant chemoradiotherapy (N.S.)
Zhang et al.[74]	2022	Colorectal cancer surgery	292	SMA calcification	AL (SMA calcium volumes score (OR, 6.8))
Morita et al.[76]	2022	Laparoscopic surgery for colorectal cancer	98	AAC	AL (high AAC (HR, 1.09))

TABLE 2 (Continued)

Report	Year	Procedure	Case number	Factor	Findings
Gunji et al.[77]	2022	Colorectal surgery	124 (65 yo <)	AAC	Severe postoperative complications (CD grade ≥ III) (CAVI (OR, 1.52) high AAC (OR, 1.08) and operative time (OR,1.01))
Liu et al.[81]	2023	Colorectal surgery	1955 (meta-analysis)	AAC	AL (high AAC (OR, 1.80))
Lee et al.[82]	2020	Rectal surgery	583	AAC and branch calcification	AL (aortic calcification score ≥3 (OR, 2.67))
Imaoka et al.[84]	2023	Surgery for stage II–III colorectal cancer	362	AAC	OS (higher AAC (HR, 2.38)) CSS (higher AAC (HR, 5.22)) RFS (higher AAC (HR, 1.83))
Transplant					
Imaoka et al.[85]	2019	Liver transplantation	156	Recipient AAC	OS (high AAC level (HR, 2.2) and old donor age (HR, 2.2))
Imaoka et al.[45]	2020	LDLT	133	Donor AAC	Biliary complications (donor AAC (HR, 4.15), right lobe graft (HR, 2.81), and splenectomy (HR, 0.39))
Ide et al.[86]	2023	LDLT	110	Recipient AAC	Postoperative CKD (age ≥50 years (HR, 5.85), high AAC (HR, 2.05), and preoperative eGFR <75 mL/min per 1.73 m^2 (HR, 1.99))
Bekki et al.[87]	2024	LDLT	164	Recipient AAC	Metabolic syndrome (AAC (OR for diabetes mellitus, 3.49, OR for hypertension, 2.91, OR for dyslipidemia, 3.55))

Abbreviations: AAC, abdominal aortic calcification; AL, anastomotic leakage; CCS, cancer-specific survival; CKD, chronic kidney disease; GI, gastrointestinal; HCC, hepatocellular carcinoma; HPB, hepatopancreatic-biliary; LDLT, living donor liver transplantation; mGPS, modified Glasgow prognostic score; NS, no significant; OS, overall survival; RFS, recurrence-free survival; RR, recurrence rate; SMA, superior mesenteric artery.

surgery. Their results showed a significant increase in the incidence of site infections. In addition, a higher comprehensive complication index (CCI) was observed in patients with high AAC ($N = 71$) than in other patients, even after adjusting for confounders using propensity score matching. A separate retrospective study by Watanabe et al.[43] of 97 patients who underwent surgery for biliary tract cancer showed significant correlations between AAC and postoperative complications ($p < 0.01$) as well as a Clavien–Dindo grade greater than III ($p < 0.01$).

In their analysis of 203 patients who underwent hepatectomy for HCC, Imaoka et al.[70] found that the overall survival (OS) was significantly lower in the high AAC group than in the low AAC group. Interestingly, high AAC was related to a higher recurrence rate (RR) and this is the first report of the relationship between AAC and antitumor immunity.

In a separate study of 99 patients who underwent liver resection for colorectal liver metastases (CRLM), Imaoka et al.[71] found that OS and RR in the remnant liver were significantly inferior in the high AAC group.

The high complication rate of HPB surgery is a challenge, and risk management using the evaluation of preoperative AAC can be an important countermeasure.

3.3 | Lower gastrointestinal surgery

In the long-term results, patients with colorectal cancer (CRC) had a significantly higher risk of death from atherosclerosis (standardized mortality ratio 1.47; 95% CI, 1.11–1.9).[72] In a study by Wang et al.,[73] of 486 patients previously diagnosed with CRC and patients with right-sided colon cancer (RCC) had a higher likelihood of clinical CAD and radiographic evidence of calcific atherosclerosis compared with patients with left-sided colon cancer (LCC). In addition, the rate of coexisting atherosclerosis was also high among patients with CRC.

In the field of lower gastrointestinal surgery, the association between AAC and AL has been reported in many cases. In an analysis of the postoperative outcomes after CRC surgery, Zhang et al.[74] performed a retrospective review of 292 patients. Multivariate analysis identified several independent risk factors for postoperative AL, including tumor location, preoperative albumin level, preoperative lymphocyte count, preoperative neutrophil/lymphocyte ratio, and SMA calcium volume score, but not the AAC score.

However, Shen et al.[75] performed an analysis of 423 patients with rectal cancer who underwent anterior resection (AR). They found an increased incidence of AL in patients with a high AAC rate. Risk-adjusted multivariate regression analysis identified a high ACC as an independent risk factor for AL. Similarly, Morita et al.[76] found that the calcified volume fraction was one of the most robust risk factors for AL. Gunji et al.[77] found a strong correlation between AAC and mortality in their analysis of 60 patients who underwent colectomy. They also found that the incidence of AL positively correlated with increased ACC severity. This finding has been confirmed in several other studies, including those by Eveno et al.,[78] Deguelte et al.,[79] and Namba et al.,[80] whose retrospective analysis confirmed a strong correlation between a higher incidence of AL and higher AAC. Liu et al.[81] performed a pooling-up analysis of eight studies including 1955 patients and reported that AAC is a potential risk factor for AL after colorectal surgery. Lee et al.[82] created an original scoring to evaluate the predictive role of aortic calcification on AL after CRC surgery.

In an analytical study on 231 CRC resections by Knight et al.,[83] AAC was not associated with clinical complete response (CR) to neoadjuvant chemoradiotherapy, and AAC was not significantly correlated with AL.

Finally, in a study by Imaoka et al.[84] higher AAC was associated with worse overall survival (OS), cancer-specific survival (CSS), and recurrence-free survival (RFS) in stage II-III CRC patients.

In the field of lower gastrointestinal surgery, measures to place a cover stoma to prevent AL can be considered in high-risk cases, such as those with high AAC. There have also been reports of higher CRC recurrence in patients with higher AAC levels. Considering that high AAC is strongly associated with fragility and lower renal function, the success and induction rates of adjuvant chemotherapy should be evaluated in the future.

3.4 | Transplant surgery

In a study by Imaoka et al.[85] of 156 liver transplant (LT) recipients, the results indicated longer survival in the low AAC group than in the high AAC group, even after propensity matching ($p < 0.01$). Subsequent multivariate analysis identified high AC and older donor age as prognostic factors for overall survival, each with a hazard ratio (HR) of 2.2.

Imaoka et al.[45] also performed a specific investigation of donor AAC in living donor liver transplantation (LDLT). They reported a significantly higher incidence of postoperative biliary complications in the AAC group ($N = 17$) than in the non-AAC group ($N = 116$; HR, 2.77; 95% CI, 1.32–5.83; $p < 0.01$). Cox proportional hazard regression modeling identified donor AAC and right lobe transplantation as factors that significantly increased the risk of biliary complications.

Ide et al.[86] performed an analysis of 110 of 184 LTs with normal preoperative renal function, as determined by an estimated glomerular filtration rate (eGFR) of 60 mL/min/1.73 m^2. The study results highlighted a significantly higher cumulative incidence of postoperative chronic kidney disease (CKD) in the high AAC group than in the low AAC group. Multivariate analysis using the Cox proportional hazards model showed significant differences in preoperative AAC ≥100 mm^3, recipient age ≥50 years, and preoperative eGFR <75 mL/min/1.73 m^2 as contributing factors to postoperative CKD.

Finally, a review of 164 liver transplant patients by Bekki et al.[87] found that preoperative AAC was a risk factor for the development of all components of metabolic syndrome after liver transplantation, although the degree of risk development varied with calcification (OR for diabetes = 3.49, $p < 0.01$; OR for hypertension = 2.91, $p = 0.047$; OR for dyslipidemia = 3.55, $p < 0.01$).

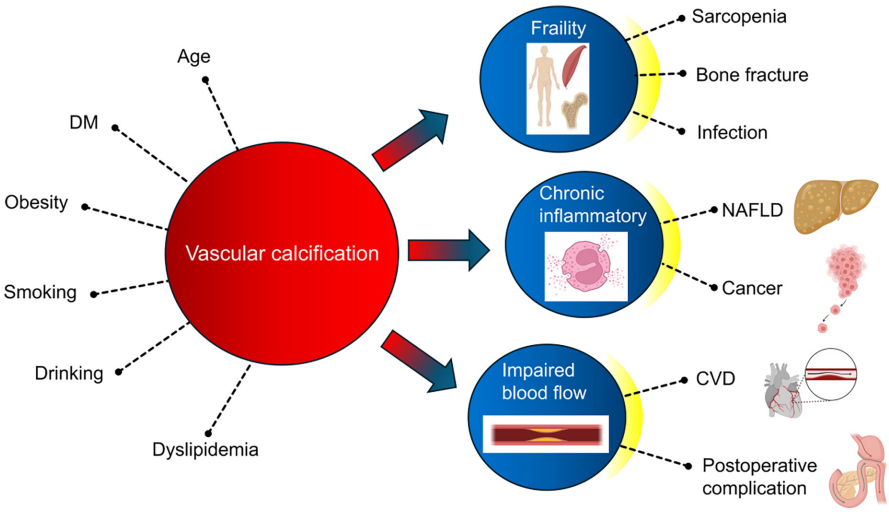

FIGURE 3 The impact of abdominal aortic calcification. Edited by biorender.

Taken together, these findings provide valuable insights into the complexities of AAC and postoperative outcomes, and highlight the need for further multicenter research in this area.

4 | CONCLUSION

The number of gastroenterological and transplant surgeries performed in the elderly is increasing. More detailed risk management indicators for the elderly are required to ensure safety. Patients with high AAC and severe atherosclerosis are more likely to have concomitant risk factors, such as advanced age, obesity, smoking, drinking, diabetes, dyslipidemia, and pre-existing CVD. The combination of fragility, chronic inflammation, and impaired blood flow is expected to have a negative impact on the postoperative outcomes of gastroenterological surgery, as shown in Figure 3. To mitigate the potential risks of short-term complications associated with advanced AAC, proactive measures should be implemented. These measures may include the utilization of minimally invasive surgical techniques and stringent perioperative blood pressure management, as well as the placement of a cover stoma when suture integrity is a concern. It is also essential to coordinate care across multiple medical disciplines to effectively manage comorbidities and ensure a positive long-term prognosis. Regular screening for cardiac and renal diseases is recommended, in addition to the adoption and maintenance of healthy lifestyle habits, to slow the progression of AAC. Furthermore, systemic vascular damage from chemotherapy such as cisplatin has been reported to promote atherosclerosis and cause hypertension and renal damage.[88,89] In addition, patients with fragility often do not tolerate postoperative chemotherapy well, and the induction and completion of postoperative chemotherapy cannot be completely achieved. As a specific effort, cardiologists need to carefully perform an initial evaluation before initiating cardiotoxic chemotherapy, continuously monitor cardiac safety to implement preventive measures, and perform regular long-term checkups to ensure early detection of CVD.[90] Dietary management and dyslipidemia control should be continued to prevent further progression of atherosclerosis during postoperative follow-up. In addition, to improve prognosis after gastroenterological surgery, gastroenterological surgeons, cardiologists, and oncologists need to work together from multiple angles as an "Oncology Team." Clinicians would undoubtedly benefit from a better understanding of the relationship between the increased risk of postoperative complications and the type of procedure, as well as the degree of AAC. Nonetheless, it is important to recognize that AAC should not be the sole factor considered in surgical decision-making. The absence of a standardized method for assessing AAC across institutions, as well as the fact that it is analyzed either as a continuous variable or as a cutoff value, creates significant challenges for conducting a more accurate meta-analysis and clarifying the correlation between postoperative complications and AAC. We hope that this review will lead to more rigorous evaluations of AAC in the future, resulting in higher-quality meta-analyses, multi-institution studies, and the development of new recommendations. This study provides a valuable guide for surgeons and clinicians to identify the potential risks associated with surgical procedures in their patients.

AUTHOR CONTRIBUTIONS

YI and MO drafted the manuscript. YI, MO, MA, KS, and HO revised the manuscript critically.

FUNDING INFORMATION

This work was supported in part by JSPS KAKENHI grant numbers 22K16535 and 23H02981 and AMED grant number 24fk0210108.

CONFLICT OF INTEREST STATEMENT

Hideki Ohdan is an editorial board member of *AGS*.

ETHICS STATEMENTS

Approval of the research protocol: N/A.
Informed Consent: N/A.
Registry and the Registration No. of the study/trial: N/A.
Animal Studies: N/A.

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How to cite this article: Imaoka Y, Ohira M, Akabane M, Sasaki K, Ohdan H. Abdominal aortic calcification among gastroenterological and transplant surgery. Ann Gastroenterol Surg. 2024;8:987–998. https://doi.org/10.1002/ags3.12816