

CARDIOLOGY UPDATES NEWSLETTER

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Dear Doctor,

Greetings from Micro Labs Limited. We are happy to bring you “Cardiology updates” which contains latest and interesting news in the field of Cardiology

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ASSOCIATION BETWEEN TRIGLYCERIDE-GLUCOSE-WAIST CIRCUMFERENCE INDEX AND THE OCCURRENCE OF CARDIOVASCULAR DISEASE

INTRODUCTION- Cardiovascular disease (CVD) has become more prevalent worldwide as a result of changing socioeconomic structures and an aging population. Considering the correlation between CVD and a higher risk of premature death and all-cause mortality, public health initiatives ought to concentrate on lowering the burden of CVD. Thus, in order to promote disease prevention and early medical intervention, it is imperative to develop screening indicators to predict the development of CVD.¹ Insulin resistance (IR) is a well-established indicator of mortality and CVD. For determining insulin resistance, the gold standard is the hyperinsulinemic-euglycemic clamp test. The use of indirect clinical approaches, such as the triglyceride glucose (TyG) index and the homeostatic assessment of insulin resistance (HOMA-IR), has become necessary due to its complexity and invasiveness. Recent data indicated that the TyG index alone was not as strongly associated with IR as TyG index-related markers, such as TyG-waist circumference (WC) and TyG-body mass indices (BMI). Nevertheless, there was little data to support a link between the TyG-WC index and the prevalence of cardiovascular disease (CVD). In order to confirm a relationship between the TyG-WC index and the incidence of CVD, all-cause mortality was taken into account as a competing risk in this investigation.²



CARDIOVASCULAR DISEASE

METHODS- A total of 7482 participants were split up into four groups according to the TyG-WC index quartiles. The groups were divided into four quartiles: lowest (Q1, n=1872), second (Q2, n=1869), third (Q3, n=1870), and highest (Q4, n=1871). During the follow-up period, Kaplan-Meier curves showed the cumulative incidence rates of CVD and all-cause mortality. Group differences were ascertained using log-rank testing. The dose-dependent connection between the incidence CVD and the TyG-WC index was illustrated by the Cox proportional hazard spline curve. When estimating hazard ratios (HRs) with 95% confidence intervals for incident CVD, modified Cox regression (Fine and Gray) used death as a competing risk.

RESULTS—The current study results showed that a total of 691 (9.24%) new-onset CVD cases and 562 (7.51%) all-cause death cases were confirmed over the course of the follow-up period, resulting in a median 15.94 years. According to Cox proportional hazard spline curves, there was a positive, dose-dependent connection between the TyG-WC index and incident CVD (Figure 1). According to Kaplan–Meier curves, the groups with higher TyG-WC index quartiles had a significantly greater cumulative incidence rate of CVD. When comparing Q2–Q4 to Q1, the adjusted HR (95% CI) for incident CVD was 1.47 (1.12–1.93), 1.91 (1.44–2.54), and 2.24 (1.63–3.07), respectively. The TyG-WC index did not significantly correlate with all-cause mortality. Specifically, compared to myocardial infarction and peripheral artery disease, angina and stroke were significantly correlated with the TyG-WC index.

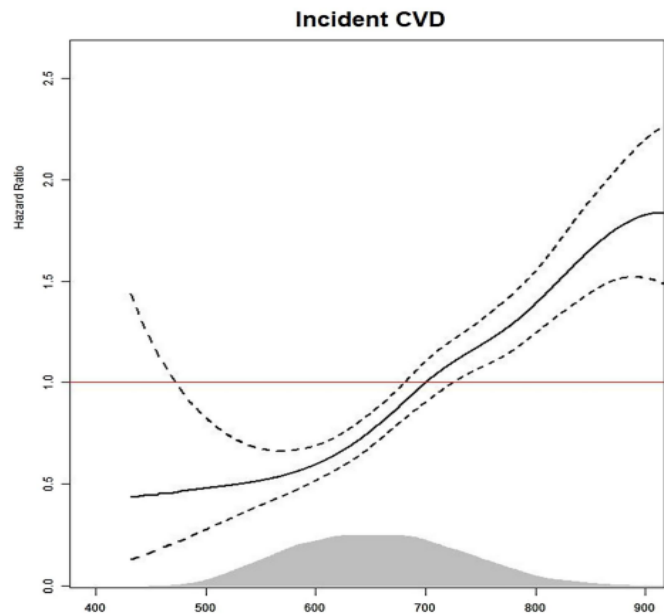


Figure 1. Incidence CVD Cox proportional hazard spline curve and density plot based on TyG-WC index. A hazard ratio of one was indicated by the red line. The TyG-WC index hazard ratio for incident CVD was shown by the black line. The dashed black area showed the hazard ratio's 95% confidence interval. Value density was shown by the gray area. CVD stands for cardiovascular disease.

DISCUSSION—The current study showed that there was a correlation between the TyG-WC index and the incidence of CVD, even when all-cause mortality was taken into account as a competing risk. These results suggested that keeping an eye on the TyG-WC index may be a useful tool for assessing CVD risk. Moreover, the study findings indicated that when the TyG-WC index increased, the risk of CVD increased in a dose-dependent manner.² Similar to this, Cho YK et al. and Hu J et al. showed that the TyG-WC index could be a useful indicator for explaining the prevalence of CVD.^{3,4}

CONCLUSION—The current study concluded that even after accounting for all-cause mortality as a competing risk, the TyG-WC index showed a favorable correlation with incident CVD. Consequently, the TyG-WC index might be a useful indicator for forecasting the development of CVD.

The TyG-WC index was a helpful tool for estimating the risk of cardiovascular disease.

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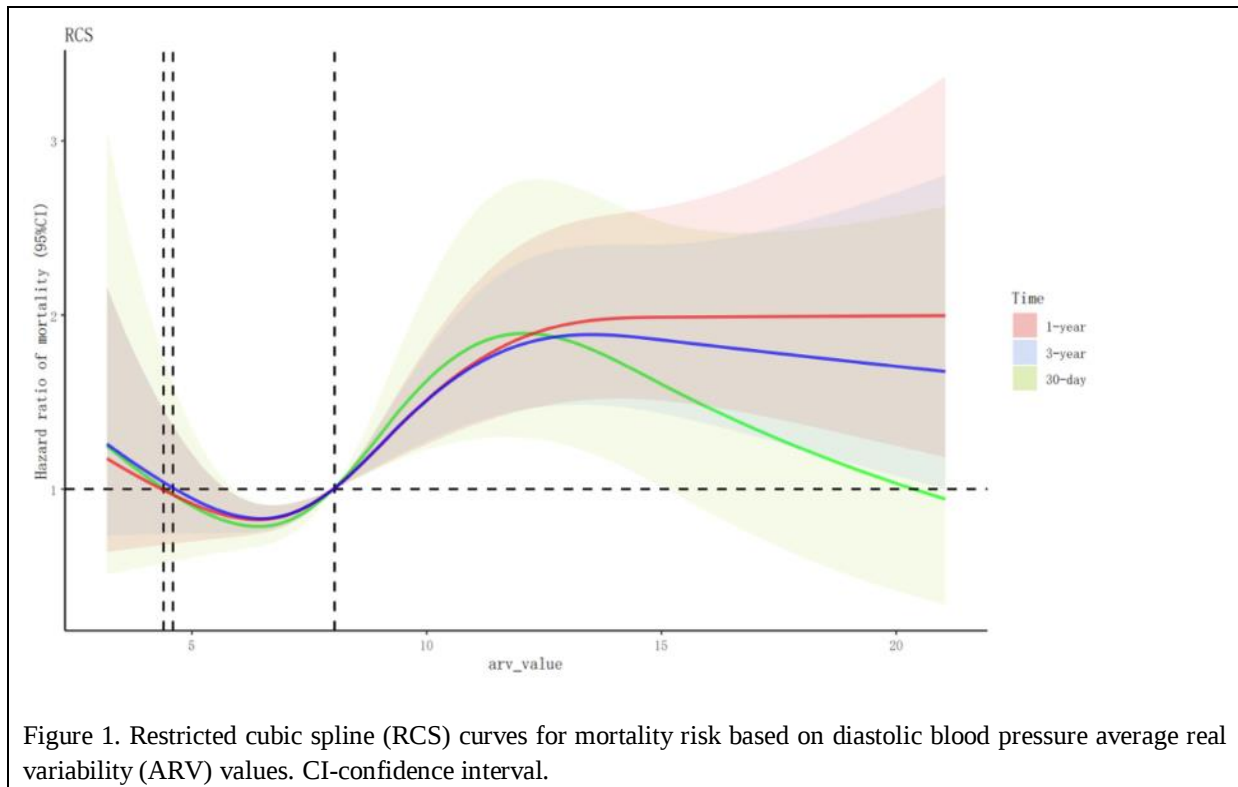
RELATIONSHIP BETWEEN MORTALITY IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION AND 24-HOUR BLOOD PRESSURE VARIABILITY

INTRODUCTION-Acute myocardial infarction (AMI) is a severe cardiovascular condition that causes significant morbidity and death. In AMI, ischemia and necrosis impact certain cardiomyocytes, resulting in a reduction in myocardial contractility, which was followed by an immediate proinflammatory response and elevated sympathetic tone. Meanwhile, excessive blood pressure variability (BPV) increases left ventricular workload, heart rate, myocardial oxygen demand, and promotes inflammation and endothelial dysfunction. As a result, a high BPV and its accompanying pathological consequences were likely to exacerbate the physiological function of the heart and influence the formation of acute cardiac problems in AMI patients.¹ The mortality rate among AMI patients has not decreased considerably in recent years. The correlation between blood pressure variability (BPV) and acute myocardial infarction (AMI) remains unclear. The aim of the study was to investigate the correlation between 24-hour BPV and death in AMI patients.²

METHODS-This was a retrospective cohort study and enrolled 1291 patients with AMI, including 475 females. The MIMIC-IV 2.0 database provided the patient's data. The following three factors were required for inclusion: (1) an AMI diagnosis; (2) age more than 18; and (3) a first ICU hospitalisation. Age, sex, race, length of stay in the intensive care unit, comorbidities, clinical conditions (hypertension, diabetes, acute heart failure, cardiogenic shock, cardiac arrest), first 24-hour blood pressure recordings after admission, and survival outcomes (all-cause mortality at 30 days, 1 year, and 3 years) were among the patient data extracted for this study from the MIMIC-IV 2.0 database. All disease diagnoses were categorised using the International Classification of Diseases, Ninth and Tenth Editions (ICD-9 and ICD-10). In this investigation, average real variability (ARV) was used to evaluate BPV. The key outcome measures were 30-day, 1-year, and 3-year all-cause mortality rates.

RESULTS-Four groups of patients were created based on the diastolic blood pressure (DBP)-ARV characteristics. The 30-day, 1-year, and 3-year death rates across the four groups showed significant differences ($p = 0.02$, $p < 0.001$, and $p < 0.001$, respectively). Systolic blood pressure (SBP)-ARV could not predict AMI patient mortality after confounding factor adjustment ($p > 0.05$). However, the highest DBP-ARV was significantly linked to higher 30-day mortality (HR: 2.291, 95% CI 1.260-4.168), 1-year mortality (HR: 1.933, 95% CI 1.316-2.840), and 3-year mortality (HR: 1.743, 95% CI 1.235-2.461). The Kaplan-Meier

graphs showed that patients in the highest ARV group had considerably poorer long-term survival probability than patients in other groups, irrespective of SBP or DBP. When DBP-ARV <8.04 , RCS curves demonstrated that the mortality risk of patients with AMI initially declined and subsequently rose with an increase in ARV. When DBP-ARV >8.04 , the 30-day mortality risk elevated and then reduced, while the 1- and 3-year death rates climbed and then stabilized with ARV rise (Figure 1).



DISCUSSION-The current study showed that DBP-ARV was a significant risk factor for increased mortality in patients with AMI.² Similar to this study, Hassan AKM et al. showed that systolic and diastolic BPV duration of the daytime and night-time interval (SD_{dn}) were the only independent predictors of in-hospital major adverse cardiovascular events (MACEs) in patients with ACS, including those with or without ST-segment elevation myocardial infarction and hypertension. A receiver operating characteristic curve analysis revealed that SBP- SD_{dn} has the highest specificity (98.4%) for predicting MACEs in hospitals, with a sensitivity of 47.3% at a threshold of 12.6 mm Hg.³

CONCLUSION-The current study concluded that this investigation shed light on the connection between BPV and mortality risks in AMI patients. To further understand the underlying processes of BPV and to assess the predictive usefulness of short- and long-term BPV for cardiovascular events and mortality risk, more clinical research was required. Additionally, the association between long-term BPV and prognosis in patients with AMI has to be explored.

If a patient's DBP or ARV was higher or lower within the first 24 hours in the intensive care unit, they might be at a higher risk of both short- and long-term mortality.

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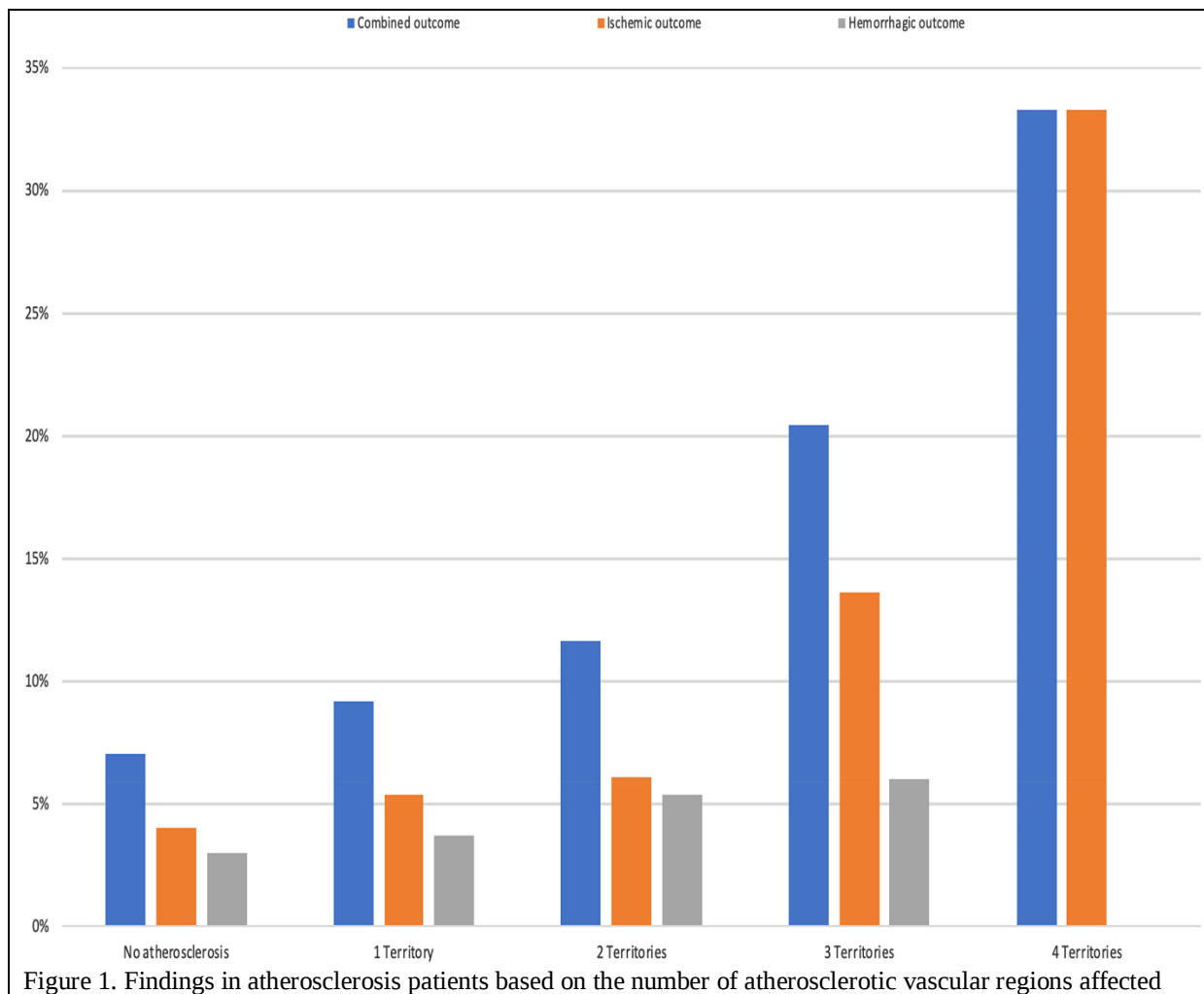
EFFECT OF ATHEROSCLEROTIC VASCULAR DISEASE BURDEN ON THE RISK OF VASCULAR EVENTS IN PATIENTS WITH RECENT ISCHEMIC STROKE AND ATRIAL FIBRILLATION

INTRODUCTION-Atrial fibrillation (AF) is the most prevalent cardiac arrhythmia, with a 5-fold higher risk of thromboembolic stroke and a 2-fold risk of all-cause death. AF is commonly associated with atherosclerotic vascular disease (ASVD), and the two disorders share a number of vascular risk factors such as hypertension, diabetes, obesity, and heart failure. Patients with AF were more likely to suffer coronary artery disease (CAD) than healthy individuals with normal sinus rhythm. Individuals with AF were more likely to develop peripheral artery disease (PAD), which increases their risk of stroke, cardiovascular events, and death.¹ Recurrent vascular events were more common in patients with atrial fibrillation (AF) and ischemic stroke (IS). In individuals who have had an ischemic stroke and AF during the last 90 days, this study assessed the effect of the burden of atherosclerotic vascular disease in various vascular areas on the risk of vascular events.²

METHODS-This was a prospective observational study and included 2148 patients with IS and AF from the International RAF network in a prospective 90-day follow-up. Atherosclerotic vascular disease was identified by at least one of the following: Symptomatic ischemic heart disease, symptomatic peripheral artery disease, internal carotid stenosis $\geq 50\%$, or the presence of plaques in the aorta. The primary outcome was a composite of stroke, transient ischemic attack, systemic embolism, cerebral bleeding, and major extra cranial bleeding within 90 days post-acute stroke. Patients were categorized into 5 groups based on the number of affected atherosclerotic vascular territories, with those with no atherosclerotic vascular disease as the reference.

RESULTS-The current study results showed that atherosclerosis affected 744 people (34.6%). A higher baseline NIHSS-score at admission, diabetes mellitus, hypertension, prior stroke/TIA, congestive heart failure, current smoking, and leukoaraiosis were all more common in older patients with AVD. Following the index stroke, patients without atherosclerosis were more likely to be prescribed oral anticoagulant medication. Patients with AVD were treated as follows: 441 (20.5%) patients received acute revascularization (rt-PA or mechanical

thrombectomy, or both) less frequently than patients who received oral anticoagulation therapy (76.6%, $P < 0.001$), and statins were more frequently given as pretreatment at admission (35.2%, $P < 0.001$) and aspirin after the index event (16.1%, $P < 0.001$). Following an acute stroke, NOACs were used to treat 344 patients with AVD (46.2%): 126 (36.6%) received rivaroxaban, 119 (34.5%) received Apixaban, and 99 (28.7%) received Dabigatran. The findings of the univariate analysis for the AVD-related variables. The following factors were substantially linked to atherosclerosis: history of stroke/TIA 237, hypertension 652, hyperlipidemia 342, and male sex 407. Variables that were independently correlated with AVD on multivariate analysis. The combined result was noted in 97 (6.9%) no-AVD patients and 77 (10.3%) AVD patients throughout a 90-day follow-up period (HR 1.22, 95% CI: 0.87-1.92). 33 (11.3%), 26 (14.7%), 26 (15.8%) and 35 (9.7%) patients with AVD had a history of ischemic heart disease with symptoms, PAD, aortic plaques, and carotid stenosis. For all groups, there was overlap in the Kaplan Meier Curve for the combined outcomes in patients with and without AVD. Compared to patients without AVD, those with AVD affecting three or four vascular areas showed a significantly higher risk of composite vascular outcomes in the multivariable analysis. In particular, the hazard ratio (HR) was 2.80 (95% CI: 1.20–6.53) for three territories involved and 6.81 (95% CI: 1.02–36.24) for four territories (Figure 1). Furthermore, when three territories were involved (HR 2.88, 95% CI: 1.04–7.95), and much more so when four territories were involved (HR 7.76, 95% CI: 1.22–49.23), the risk for ischemic outcomes increased dramatically.



DISCUSSION-The current study showed that there was a considerable elevated risk of combined vascular and ischemic events in patients with AVD covering three or four territory. On the other hand, there does not seem to be a relationship between the level of atherosclerotic involvement and bleeding consequences. This suggested that ischemic rather than hemorrhagic events were predominantly responsible for the higher risk in the combined patient group.² The current study results were in coincides with Park et al. that found a dose-dependent increase in the incidence of major adverse vascular events (MACE) and all-cause death in stroke patients with concurrent AVD. The study found that extracranial stenosis was highly prevalent.¹

CONCLUSION-The current study concluded that in patients who have had a recent stroke and AF, AVD was a prognostic indication, especially if three or four territory were involved. An increased risk of combined and ischemic events was strongly correlated with the degree of vascular territory involvement.

An increased risk of combined and ischemic events was strongly correlated with the degree of vascular territory involvement.

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CASE REPORT ON ACUTE MYOCARDIAL INFARCTION DUE TO GIANT CORONARY ARTERY ANEURYSM AND ARTERIOVENOUS FISTULA

INTRODUCTION

An area of the coronary artery that is focally larger than the adjacent normal segment by 1.5 times its diameter is known as a coronary artery aneurysm (CAA). Kawasaki illness in children and atherosclerosis in adults are the primary causes. CAA is a silent, progressive illness that is unintentionally discovered during coronary angiography. However, it can have deadly side effects, including rupture, distal embolization, thrombus formation, and compression of nearby cardiac structures. Uncertainties exist regarding the pathophysiological mechanisms. The establishment of aneurysmal defects was associated with atherosclerosis, proteolytic imbalance, and inflammatory response.¹ A diameter of greater than 20 mm indicates a "giant" CAA, which was frequently accompanied with CAFs. The improper communication between a coronary artery and a cardiac chamber or a portion of the coronary venous system was known as a CAF, which was a major abnormality of the coronary arteries. The aim of this case report was to present a case on acute myocardial infarction due to giant coronary artery aneurysm and arteriovenous fistula.²

CASE PRESENTATION

A 46-year-old female reported to the emergency department with intermittent chest pain radiating to her back and left subclavian area that began 8 hours prior. She had 20 pack-years of smoking history and a favorable cardiac family history. The remainder of the personal history was negligible. The physical exam was unremarkable. The initial 12-lead electrocardiogram indicated no aberrant findings; nevertheless, laboratory results showed an elevated troponin level, indicating myocardial injury. Thus, the patient was hospitalized to the coronary care unit with suspected NSTEMI, and a coronary angiography was scheduled. The pharmacological treatment approach comprised dual antiplatelet therapy (aspirin and

clopidogrel), atorvastatin, and omeprazole. Interestingly, bedside echocardiogram revealed a structurally and functionally normal heart, with the exception of a 25-mm-diameter cystic development with a low flow gradient in the infero-lateral cardiac wall. Coronary angiography the next day revealed a coronary aneurysm with arteriovenous connection and thrombus development without stenosis in the LCx's periphery in the absence of coronary artery disease. Cardiac computed tomography (CCT) revealed a 4.3 cm diameter aneurysm at the right-posterior cardiac boundary, with thrombus development (~13 mm diameter) within the aneurysmal sac and an arteriovenous contact with the distal LCx. The patient complained pain relapse the next day, along with a fresh increase in troponin levels and electrocardiographic abnormalities in the inferolateral wall (negative T waves in leads II-III, aVF, and V5-V6). Repeat echocardiogram revealed a marginally reduced LVEF of 50-55%, as well as posterior cardiac wall and right ventricle hypokinesia. In addition, a hyperechoic region was found in the aneurysmal space, indicating thrombus growth. Following consultation with the hospital's cardiac doctors, surgical removal of the aneurysm was recommended. After elevating the heart apex, the aneurysm became evident macroscopically and measured around 6 cm. The aneurysm was removed, exposing the thrombus. After removing the thrombus, the proximal and distal orifices were exposed and sutured. The aneurysm was ligated, and the patient was safely weaned off cardiopulmonary bypass (CPB). Histopathological examination of the aneurysmatic tissue revealed an intima with modest myxomatoid alterations, whereas the media was disorganized with smooth muscle and elastic fibers. These findings appear to have led to structural weakening in the vascular wall, which contributed to the creation of the sac [10]. There was clear separation of the media wall, with thrombus material detected within that was identical in composition to the discrete parts of the big thrombus, with numerous erythrocytes, fibrin deposits, and inflammatory cells. There was no evidence of atherosclerotic lesions (Figure 1). The histological examination revealed no particular abnormalities indicating vasculitis or connective tissue disorder. As a result, congenital malformations were thought to be the most common cause of CAA. The patient was discharged with a suggestion for 12 months of dual antiplatelet therapy based on acute coronary syndrome (ACS) recommendations. During follow-up, the patient remained asymptomatic, and both the LVEF and the electrocardiogram returned to normal.

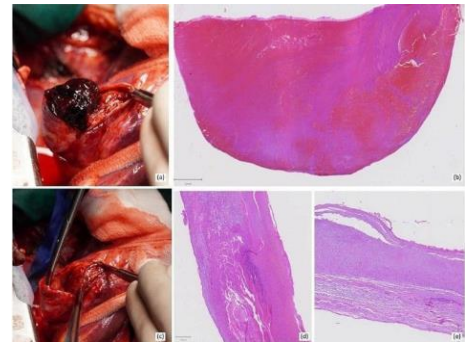


Figure 1. Macroscopic (a, c) and microscopic (b, d, e) view of the coronary aneurysm with arteriovenous communication and thrombus formation

DISCUSSION

This case report showed that CAAs and CAFs present major challenges for both interventional cardiologists and cardiac surgeons. The diverse clinical symptoms and structural intricacy of abnormalities require customized treatment.² Similar to this, Pham V et al. showed that heart failure, thrombosis, distal embolization of the vessel, rupture, vasospasm, cardiac compression, and cardiac tamponade were among the uncommon but potentially deadly complications of CAA.³

CONCLUSION

It was concluded that management of unusual entities, such as "giant" CAAs and CAFs, can be difficult. Cases like this can be used as precedents to help build treatment plans and

consistent recommendations, underlining the significance of individualized tactics for prospective patients.

Large CAAs and CAFs are examples of uncommon entities that can be difficult to manage. Examples like this one can assist in the development of treatment plans and uniform guidelines, highlighting the significance of customized approaches for subsequent patients.

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