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REVIEW

The Role of Anticoagulation in Acute Coronary Syndrome Patients with Coronary Artery Ectasia

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Background: The coexistence of coronary artery ectasia (CAE) and acute coronary syndrome (ACS) is uncommon and associated with poor clinical outcomes. However, evidence regarding the role of anticoagulant therapy and covered stents in this context remains limited.

Methods: To evaluate the potential impact of anticoagulant therapy in patients with CAE and ACS, we conducted a comprehensive systematic review of case reports and case series published up to August 2022.

Results: Our findings suggest that anticoagulant therapy may be beneficial in preventing ACS in patients with CAE. The adjunctive use of single or dual antiplatelet therapy should be individualized based on bleeding risk. In addition, covered stents may represent a viable treatment option, particularly for short lesions.

Conclusion: Patients with CAE often present with ACS and require tailored management strategies owing to altered vessel morphology and flow dynamics. Evidence from case series suggests that the combination of antiplatelet and anticoagulant therapy may reduce ischemic recurrence without a marked increase in bleeding events. Covered stents offer a viable intervention for short, isolated ecstatic segments.

Keywords: coronary artery ectasia, acute coronary syndrome

Introduction

Coronary artery ectasia (CAE) is defined as an abnormal dilatation – either diffuse or segmental – of a portion of a coronary artery to a diameter 1.5 times greater than that of an adjacent normal segment.[1] Although CAE is considered rare, with a reported prevalence ranging from 0.85% to 5.3%,[2] it has gained increasing clinical attention because of its association with adverse cardiovascular outcomes.

One of the most critical concerns arises when CAE coexists with acute coronary syndrome (ACS) – a group of conditions associated with sudden, reduced blood flow to the heart. CAE in the context of ACS may complicate the clinical course, as the abnormal vessel anatomy can reduce coronary blood flow and promote thrombus formation, even when standard ACS treatments are applied.[3,4]

Evidence suggests that patients with both CAE and ACS may experience worse outcomes than those with ACS alone. In such patients, impaired flow dynamics within ectatic arteries may increase the likelihood of thrombosis. While anticoagulants have been hypothesized to reduce adverse events in this setting, no randomized clinical trials have confirmed this approach.[4]

Currently, the treatment options for managing ACS in CAE patients vary, and include dual antiplatelet therapy (DAPT), DAPT combined with anticoagulants, and single antiplatelet therapy (SAPT) with anticoagulants. The emergence of non-vitamin K antagonist oral anticoagulants (NOACs) has further complicated decision-making, raising questions about whether to use NOACs or traditional warfarin in this context.

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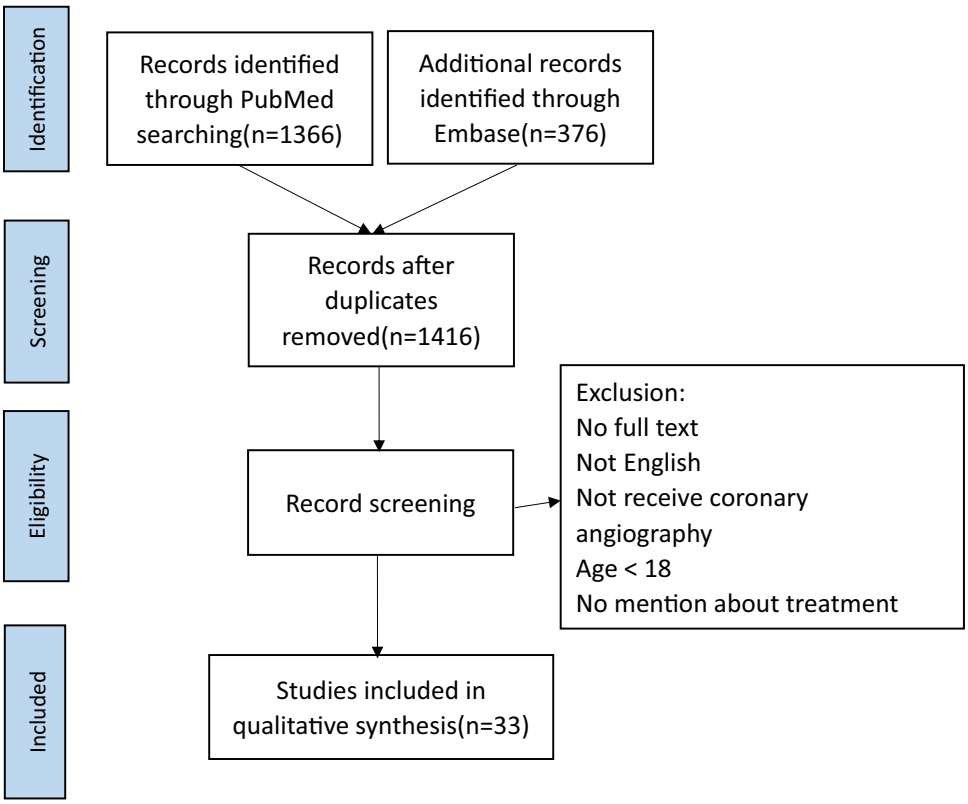

Figure 1 Study flow diagram.

This review aims to analyze the current treatment strategies for ACS in patients with CAE, highlight the gaps in evidence, and propose considerations for future research and clinical practice.

Methods

The papers to be included in this systematic review and meta-analysis were sought in MEDLINE and Embase up to August 31, 2022. The search strategy was conducted using the following terms: "coronary artery ectasia" and "acute coronary syndrome". Studies were screened by the title, abstract, and full text, by two independent investigators, based on the PRISMA 2020 checklist. The PRISMA flowchart is shown in Figure 1.

Selection Criteria

The inclusion criteria for this review were all studies that reported a patient with CAE who presented with ACS. The exclusion criteria included not being able to retrieve the full text, patients not receiving coronary angiography, age <18 years, and no mention of treatment.

Results

Patient Characteristics

In total, 31 patients were identified who presented with CAE and ACS. Four of them had another episode of ACS during follow-up. Thus, there were 35 cases compatible with ACS and CAE. Ten of them were female. The average age of the patients was 55 years old. In most cases, the infarct-related artery (IRA) was the same artery as the one with aneurysm formation.

The Patient Profiles are Separated According to Different Treatments in Tables 1–3

Most of the patients received standard DAPT (n=13), while some of them received DAPT with anticoagulant (n=9). Six of the patients receiving only DAPT had ACS during follow-up. No ACS was reported in those taking DAPT or SAPT

Table 1 Patients Receiving DAPT

Authors/Publication Year	Age (years)/Gender	ACS Type	Location of CAE	IRA	Antiplatelet	Anticoagulant	Stent	Duration of Follow-Up	Outcome
DAPT									
Tehrani et al 2021[5]	73/F	STEMI	RCA	RCA	A/C	–	Covered stent	12 mo	–
Biswas et al 2019[6]	32/M	STEMI	LAD	LAD	A/C		+	N/A	–
Sidhu and Kuar 2022[7]	56/M	NSTEMI	LAD/RCA	LAD	A/T		+	8 d	Recurrence
Moghadam et al 2021[8]	29/F	NSTEMI	LM/LCX	OM	A/C		+	12 mo	–
Lee et al 2020[9]	26/M	STEMI	p-LAD	LAD	A/T		–	N/A	Referral to CABG
Guo et al 2019[10]	30/M	NSTEMI	LAD	LAD	A/C		+	4 mo	Recurrence
Taskesen et al 2021[11]	64/M	STEMI	LCX	LCX	A/C		+	18 mo	–
Carrión-Barberà et al 2021[12]	43/F	STEMI	LAD	LAD	A/T		+	16 mo	Recurrence
Furugen and Takagawa 2012[13]	78/M	NSTEMI	LCX	LCX	A/C		N/A	3 mo	–
Mrdović et al 2004[14]	47/M	STEMI	LAD/RCA	RCA	A/C		N/A	2 mo	Recurrence
Latt et al 2017[15]	35/M	STEMI	LAD/RCA	RCA	A/C		+	1 mo	–
Tomioka et al 2016[16]	78/F	UAG	RCA	RCA	A/C		–	1 mo	Recurrence
Damay et al 2019[17]	61/M	UAG	RCA	RCA	+/C		+	1 mo	Recurrence

Abbreviations: DAPT, dual antiplatelet therapy; ACS, acute coronary syndrome; CAE, coronary artery ectasia; IRA, infarct-related artery; STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; UAG, unstable angina; RCA, right coronary artery; LAD, left anterior descending coronary artery; LM, left marginal coronary artery; LCX, left circumflex coronary artery; p-LAD, proximal left anterior descending coronary artery; A, aspirin; C, clopidogrel; T, ticagrelor; N/A, not mentioned; CABG, coronary artery bypass graft.

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Table 2 Patients Receiving DAPT and Anticoagulant Therapy

Authors/Publication Year	Age (years)/Gender	ACS Type	Location of CAE	IRA	Antiplatelet	Anticoagulant	Stent	Duration of Follow-Up	Outcome
DAPT + Anticoagulant									
Elhosseiny et al 2019[18]	66/M	STEMI	LM/LAD/LCX/RCA	LAD/RCA	A/C	R	–	N/A	–
Taskesen et al 2021[11]	68/M	STEMI	RCA	RCA	A/T	R	Covered stent	6 mo	–
Taskesen et al 2021[11]	64/M	NSTEMI	LCX	LCX	A/T	Api	–	12 mo	–
Sidhu and Kaur 2022[7]	56/M	STEMI	LAD	LAD	A/T	D	+	12 mo	–
Okada et al 2016[19]	40/F	STEMI	RCA	RCA	A/C	W	–	0.5 mo	–
Boles et al 2013[20]	64/F	STEMI	RCA	RCA	A/C	Unknown	N/A	1.5 mo	–
Damay et al 2019[17]	61/M	UAG	RCA	RCA	A/C	W	+	6 mo	–
Tehrani et al 2021[5]	79/M	STEMI	LCX	LCX	A/C	W	Covered stent	3 mo	–
Tehrani et al 2021[5]	84/F	STEMI	RCA	RCA	A/C	W	+	2 mo	–

Abbreviations: DAPT, dual antiplatelet therapy; ACS, acute coronary syndrome; CAE, coronary artery ectasia; IRA, infarct-related artery; STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; UAG, unstable angina; LM, left marginal coronary artery; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; RCA, right coronary artery; A, aspirin; C, clopidogrel; T, ticagrelor; W, warfarin; R, rivaroxaban; Api, apixaban; D, dabigatran; N/A, not mentioned.

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Table 3 Patients Receiving Single Anticoagulant Therapy or SAPT with Anticoagulant Therapy

Authors/Publication Year	Age (years)/Gender	ACS Type	Location of CAE	IRA	Antiplatelet	Anticoagulant	Stent	Duration of Follow-Up	Outcome
Anticoagulant									
Guo et al 2019[10]	30/M	STEMI	LAD	LAD	–	W	Covered stent	4 years	–
Biondi et al 2021[21]	79/M	NSTEMI	SVG-D1	LCX	–	W	–	7 mo	Recurrence
SAPT + anticoagulant									
Jiang et al 2021[22]	21/F	NSTEMI	RCA	PDA	A	W	–	3 mo	–
Tuncer et al 2008[23]	39/M	STEMI	LM	LCX	C	W	–	3 mo	–
Tuncer et al 2008[23]	70/M	NSTEMI		–	C	W	–	3 mo	–
Lima et al 2006[24]	43/M	NSTEMI	LAD/LCX/RCA	LAD	A	W	–	6 mo	–
Lima et al 2006[24]	60/M	UAG	RCA	–	C	W	–	12 mo	–
Perlman and Ridgeway 1989[25]	41/M	UAG	LAD/LCX/RCA	LAD	A	W	–	40 mo	–
Choi et al 2018[26]	58/M	STEMI	LAD/LCX/RCA	RCA	C	R	–	11 mo	–
Singab et al 2022[27]	39/F	STEMI	LAD/LCX/RCA	LAD	Unknown	Unknown	–	N/A	Referral to CABG

Abbreviations: SAPT, single antiplatelet therapy; ACS, acute coronary syndrome; CAE, coronary artery ectasia; IRA, infarct-related artery; STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; UAG, unstable angina; LAD, left anterior descending coronary artery; SVG-D1, saphenous vein graft to first diagonal; RCA, right coronary artery; LM, left marginal coronary artery; LCX, left circumflex coronary artery; PDA, posterior descending artery; A, aspirin; C, clopidogrel; W, warfarin; R, rivaroxaban; N/A, not mentioned; CABG, coronary artery bypass graft.

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with an anticoagulant. Those taking only warfarin more often had myocardial infarction (MI) during follow-up. Three patients received a covered stent, none of whom had recurrent ACS during follow-up. Those who received a covered stent showed immediate resolution of the coronary aneurysm under angiography, and none of them had another episode of ACS after that; however these three patients received different combinations of treatment (warfarin alone, DAPT, or DAPT plus warfarin).

Discussion

Coronary Artery Ectasia

CAE is a well-recognized angiographic finding, characterized by abnormal dilatation of the coronary arteries.[28] Although the specific mechanism leading to CAE is still unknown, the risk factors and pathophysiology behind CAD are also associated with CAE.[29] These risk factors include hypertension, hyperlipidemia, smoking cigarettes, and male gender. Atherosclerosis is the most well-known cause of CAE. Congenital and collagen vascular disorders, infections, iatrogenic disease, and cardiac lymphomas, among others, are also causes.[2] The hypothesis that CAE develops from CAD is supported by its frequent coexistence with CAD, and the observation of common histopathological findings, such as lipid deposition and hyalinization, destruction and reduction of the medial elastic fibers, and disruption of the internal and external elastic lamina.[30–32] Research has revealed that patients with CAE may have a higher incidence of ACS, as Takahito Doi et al reported that patients with CAE have an increased risk of major adverse cardiovascular events (MACE), cardiac death, or non-fatal MI.[33] However, Doi's findings were based on a small, retrospective cohort with limited ethnic diversity, which may affect generalizability.[33] The real prevalence of ACS in patients with CAE needs further investigation.

Coronary Artery Ectasia and Acute Coronary Syndrome

When ACS is encountered in this group, the standard treatment is still unknown owing to the clinical observation that a significant number of patients present with recurrent ACS after DAPT. An antiplatelet agent (or agents) combined with an anticoagulant seems more effective in this group. Doi et al reported that 43/51 patients receiving warfarin reaching a time in therapeutic range (TTR) >60% did not have MACE, cardiac death, or non-fatal MI.[33] Pranata et al reported that the addition of an anticoagulant seemed to be more effective than DAPT alone, especially in isolated CAE without other obvious stenotic lesions.[4] In our analysis, patients receiving a single anticoagulant had more recurrent ACS (1/2). Patients receiving DAPT also had a high rate of recurrence of MI (6/13). Those receiving DAPT with an anticoagulant, then transferring to a single antiplatelet agent after 1–3 months with an anticoagulant, had no recurrent MI (0/9). This is compatible with the previous observation. DAPT with an anticoagulant regimen is similar to the treatment for patients who had ACS with atrial fibrillation (AF). The IRA is most often the same artery as the one with aneurysm formation, which may explain why standard DAPT is associated with more complications, owing to the different mechanisms leading to ACS in the normal coronary artery and coronary ectasia. Abnormal vessel dilatation causing disturbed coronary flow is different from plaque rupture-induced ACS. The limited coronary artery flow, as in the case of the patient with AF, would increase the probability of thrombus formation in the coronary artery. This may explain the benefit of anticoagulants in this condition.

Covered Stents

Polytetrafluoroethylene (PTFE)-covered, balloon-expandable stents have been shown to be effective devices for percutaneous management and prevention of coronary aneurysms.[34] In our analysis, three patients received a covered stent, and no adverse events were reported, whether they received anticoagulants or not. This may provide an alternative treatment option for these patients.

Side Effects of Anticoagulation

In our analysis, some case reports did not mention the adverse effects of anticoagulants, such as anemia, gastrointestinal bleeding, or other bleeding events. Current guidelines for ACS suggest a duration of 1 week to 1 month for the combination of DAPT with NOAC in patients with high ischemic risk and low risk of bleeding events.[35,36] In our analysis, in patients receiving

anticoagulants with DAPT, the anticoagulant would be taken off after 1–3 months of therapy. Our study did not assess the outcome of bleeding events. According to the APPRAISE-2 and ATLAS ACS 2-TIMI 51 trials, the rate of intracranial bleeding events in patients receiving NOAC combined with DAPT ranged from 0.2% to 0.7%. It may be safe to use DAPT with an anticoagulant for 1 month in CAE with ACS.

Limitations

There are some limitations to our analysis. First, there has been no long-term follow-up in these patients. The longest follow-up was 4 years, in one patient who received a covered stent. Other studies had follow-up durations between 1 and 18 months. Thus, some adverse ischemic events may have been neglected. Second, there were no adverse events regarding bleeding episodes, which is another issue with the combination of antiplatelets and anticoagulants. Third, some risk factors, such as smoking, diabetes mellitus, or hyperlipidemia, could not be analyzed. This may be another confounder in this analysis.

Further randomized controlled trials (RCTs) are essential to evaluate the benefits and side effects of anticoagulants in this group.

Conclusion

Most of the patients with CAE were diagnosed as a result of the clinical presentation of ACS. One retrograde observational study revealed that the most frequent clinical indication for catheterization was ACS, followed by stable angina pectoris and a positive stress test. Observational data suggest that the majority of CAE patients are concurrently diagnosed with CAD. This means that concurrent CAE with CAD is an important issue to consider. No clinical trials have evaluated the treatment in this group. However, based on current case series and analysis, administering an anticoagulant with an antiplatelet agent seemed to result in fewer ischemic events and was not associated with more bleeding events. Otherwise, in suitable cases, such as short lesions or non-branch CAE, a covered stent may be another option to manage CAE with ACS. Given the complexity and rarity of CAE with ACS, individualized treatment remains essential. This review emphasizes the critical need for further RCTs to improve outcomes in this understudied population.

Data Sharing Statement

All data relevant to the study are included in the article or references.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no potential conflicts of interest in this work.

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