Sudden cardiac arrest in the setting of coronary artery ectasia: Mechanistic and clinical perspectives

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Coronary artery ectasia (CAE) has been defined as diffuse dilatation of a particular coronary segment presenting with a diameter >1.5 times the diameter of the reference coronary segment [1–4]. In this context, focal coronary dilatation is called a "coronary aneurysm" that involves less than one-third the length of the coronary artery [2–4]. Pathogenetically, these entities might be attributable to a variety of factors including atherosclerosis, vasculitis (such as Kawasaki disease, etc.), and iatrogenic causes, etc. [1–4]. As expected, prognosis largely depends on the underlying etiology and anatomical features [1–4]. The recent report by Zalewska-Adamiec et al. [1] has described a case of CAE involving the whole left coronary system (widely termed as type-1 CAE [2, 4]) complicated by aborted sudden cardiac arrest (SCA) during coronary angiography [1]. Accordingly, we would like to highlight some mechanistic and clinical implications of SCA in the setting of CAE (and coronary aneurysms):

In the out-of-hospital setting, SCA in patients with CAE or coronary aneurysms might have particular aspects:

- First, SCA in these patients might be caused by exercise-induced myocardial ischemia possibly as a consequence of substantial flow stagnation at the macrovascular level [1, 2, 4]. This seems consistent with the positive exercise testing in the patient despite her non-obstructive coronary anatomy [1].
- Second, severe coronary microvascular dysfunction (associated with diffuse endothelial dysfunction [2–4]) might primarily account for or contribute to exercise-induced myocardial ischemia and might also be associated with coronary flow stagnation in these patients [4]. Did the authors plan further tests for potential microvascular dysfunction including positron emission tomography, etc.?
- Third, these patients might also be prone to coronary vasospasm [1, 2, 4], and might unexpectedly incur SCA at rest. Therefore, well-known strategies for the management of vasospastic angina (VSA) (including avoidance of potential VSA triggers (high dose acetylsalicylic acid, non-selective beta-blockers, etc.) along with initiation or up-titration of calcium channel blockers) should also be implemented [4]. We wonder about the doses of prescribed agents and the typical history of VSA in the patient [1].
- Fourth, evolution of acute coronary syndromes (ACSs) due to distal coronary embolism (manifesting as myocardial infarction with non-obstructive coronary arteries) is also quite likely and accounts for SCA in some cases as well [2–4]. Therefore, long-term antiplatelet therapy and/or anticoagulation have been used in these patients largely based on the size of the ectatic or aneurysmatic segments that might change drastically in time [3, 4]. Moderate or giant aneurysms (with diameters of >2 and >4 times the diameter of the reference segment, respectively) usually require long-term anticoagulation (particularly for the secondary prevention of coronary thromboembolism) [3]. Did the patient have an overt history of ACS?
- Fifth, CAEs and coronary aneurysms (particularly the giant ones) may also be complicated by mechanical complications including rupture and fistula formation that might present with a non-arrhythmic SCA and require urgent surgery [3, 4].

- Finally, persistent giant dilatations, rapid expansion, intractable anginal symptoms, recurrent ACSs (despite optimal therapy), and co-existing stenotic lesions may warrant elective percutaneous or surgical intervention [3, 4] to prevent possible SCA. Regardless of the management strategies, aborted SCA in the out-of-hospital setting should warrant implantable-cardioverter defibrillator implantation in these patients. We also wonder about the schedule of surveillance (Holter monitoring, frequency of coronary imaging on follow-up, etc.).

Importantly, these patients may also have a significant proclivity for arrhythmic SCA in the setting of coronary angiography and coronary interventions due to a variety of specific triggers [1]. One such trigger might be catheter-induced coronary spasm (a phenomenon well known to be more frequent in patients with vasospastic angina). Therefore, excessive manipulation of engaged coronary catheters and also other tools (guidewires, etc.) should be avoided in these patients. Notably, intermittent intracoronary nitrate injection might be a potential strategy for the prevention of coronary vasospasm during coronary interventions (though long-term use of nitrates is discouraged in these patients [1, 4]). Another SCA trigger in this context might be excessive and forceful injection of contrast agents (for better visualization) that might potentially lead to contrast-induced arrhythmogenesis [1] and also possible fragmentation and embolism of a pre-existing coronary thrombus. Consequently, prolonged (but not forceful) injection of contrast agents during cineangiography seems more prudent. Therefore, we wonder about the pattern and magnitude of cardiac troponin elevation (if any) that might have suggested coronary embolism [1].

In conclusion, SCA in patients with CAE might be considered a multi-faceted phenomenon mostly associated with a variety of ischemic triggers [1–4]. Therefore, strategies aiming to mitigate such ischemic triggers (both in interventional and out-of-hospital settings) are necessary for SCA prevention in these patients.

Article information

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