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Chapter

Coronary Embolic Phenomena: High-Impact, Low-Frequency Events

Qasim Malik, Ambreen Alam, Stanislaw P. Stawicki and Peter Puleo

Abstract

Coronary embolic phenomena (CEP) are difficult to diagnose yet carry potentially devastating clinical consequences. The goal of this chapter is to outline key processes and pathophysiologic mechanisms underlying CEP, primarily in the context of acute coronary syndrome (ACS). Not surprisingly, most reported cases of CEP occur in the left coronary circulation, but some right-sided events have been reported. Overall, causes include thrombotic, septic/infectious, neoplastic, valve-related, and iatrogenic mechanisms such as air embolization. Coronary angiography remains the definitive diagnostic and therapeutic approach, with computed tomography being increasingly utilized. Transthoracic echocardiography (TTE) should be part of a routine work up for patients with suspected CEP. Holter/event monitoring for atrial fibrillation may also be indicated in patients with embolic phenomena. Clinical management includes procedural restoration of coronary blood flow, followed by appropriate anticoagulation or antiplatelet therapy, in conjunction with appropriate treatment of any arrhythmias or other associated cardiac manifestations or conditions. Timely diagnosis, based on a high index of suspicion (especially in high-risk population) may be important in improving morbidity and mortality in affected patients. Since CEPs are often underdiagnosed and may be due to a number of heterogeneous causes, the need arises for increasing provider awareness of these important phenomena, as well as for the implementation of appropriate clinical management guidelines.

Keywords: coronary artery embolism, coronary embolic phenomena, diagnosis, management, risk factors

1. Introduction

Coronary embolic phenomena (CEP) constitute an under-reported and underdiagnosed set of clinical phenomena, with potentially devastating consequences if not recognized and treated promptly [1–3]. From coronary air embolism to paradoxical venous thromboembolism, CEPs represent an etiologically heterogeneous group of events [4–7]. It has been postulated that CEPs are the underlying cause of up to 3% of acute coronary syndromes (ACS) [6]. Given their rarity, CEPs require a high index of suspicion by the treating clinician [8–10]. In this chapter, we will aim to cover the various processes and pathophysiology underlying this cause of acute coronary

syndrome. Our focus will be on the more commonly seen forms of coronary embolism, with an abbreviated overview provided of the less common etiologies.

2. Methods

A thorough literature search was conducted using PubMed, Google™ Scholar, and Bioline International. The following search terms were utilized, in various combinations/derivations/iterations, listed alphabetically: "cardiac," "coronary," "emboli," "embolism," "embolus," "heart," "infarction," "myocardial," "myocardium," "paradoxical," "phenomenon," "vascular," "vasculature," and "vessel". Secondary identification of additional literature sources was performed using articles referenced by our primary sources.

3. Classification

Coronary emboli may be classified based on etiology (i.e., thrombotic, septic, neoplastic, valvular heart disease-related, iatrogenic), although other classifications (i.e., direct, paradoxical and/or iatrogenic) have been proposed and/or described [6, 11–13]. A list of all previously reported types/causes of coronary emboli is provided in **Table 1**.

Thrombotic	Paradoxical thrombus/embolus
	Left atrial appendage thrombus
	Left atrial thrombus
	Left ventricular mural thrombus
Autoimmune	Inherited coagulation factor deficiencies (prothrombin deficiency, protein C/S deficiency)
	System lupus erythematosus
	Antiphospholipid syndrome
Infectious	Infective endocarditis
	Rheumatic heart disease
Valve-related	Fibroelastoma
	Mitral valve calcifications
	Blood cysts
Neoplastic	Malignancy
Iatrogenic	Post-cardiac procedure
Miscellaneous causes	Pregnancy
	Air embolism

Table 1.
Causes of coronary embolism.

4. Mechanisms and pathophysiology

Coronary emboli may originate in the left or right side of the heart [14, 15]. Of course, for emboli originating in the right heart to lodge in the coronary arteries, they would need to be somehow "shunted" to the left-sided system, possibly

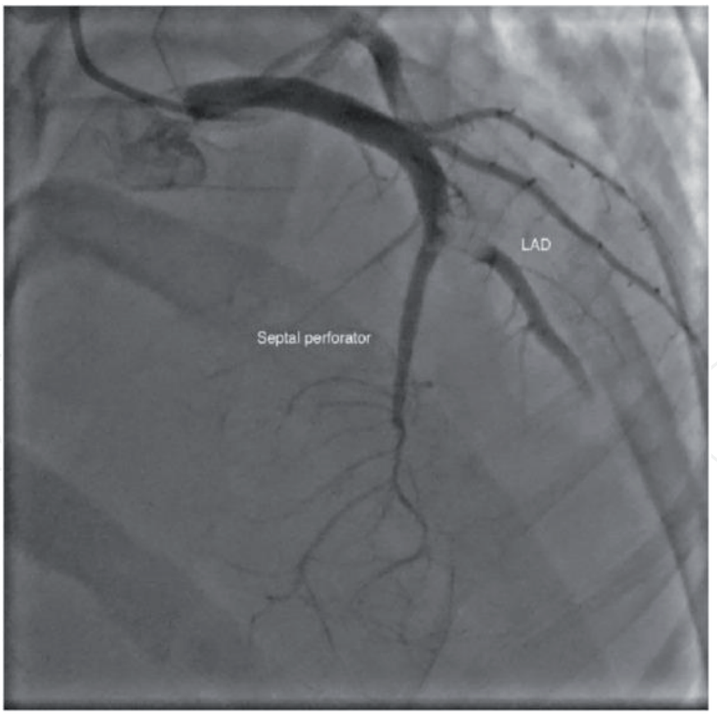


Figure 1.
Angiographic example of a large coronary artery embolus located in the mid-left anterior descending artery. Source: Zhang et al. [19]. Image used under the terms of the Creative Commons Attribution 4.0 License (http://www.creativecommons.org/licenses/by/4.0/) which permits any use, reproduction and distribution of the work without further permission provided the original work is attributed as specified on the SAGE and Open Access pages (https://us.sagepub.com/en-us/nam/open-access-at-sage).

through a patent foramen ovale [16–18]. An angiographic example of paradoxical coronary artery embolism is show in **Figure 1** [19].

It must be mentioned here that systemic emboli finding their way to the left heart are still more likely to embolize to the carotid or intracranial vasculature, primarily due to two particular considerations. Firstly, the coronary anatomy and coronary artery takeoff is typically such that emboli are less likely to specifically dislodge and enter into their ostia [13, 20, 21]. Secondly, it is hypothesized that coronary vessels may be protected to some degree, mainly due to them receiving flow primarily during diastole [22–26]. For similar reasons, one might extrapolate that most reported cases of coronary embolism occur in the left coronary circulation due to the anatomy of the right coronary artery takeoff making it potentially less conducive to emboli [22–28].

Coronary emboli may become lodged in major epicardial arteries supplying a sizable area of myocardium, and smaller emboli may even embolize distally so as to affect small arterioles which do not supply a large area [29–31]. These events may or may not be clinically symptomatic or readily diagnosable, but evidence in this generally poorly understood area of cardiac pathophysiology continues to be lacking. It is important to note, however, that coronary emboli may occur in the setting of concomitant atherosclerosis, where even a small embolus could lodge at the site of atherosclerotic lesion and result in significant epicardial coronary occlusion, thus exposing potentially significant area of myocardium at risk for a subsequent secondary ischemic event [31–35]. An association with infectious etiology may be present as well in this context [32].

5. Coronary embolic events: a heterogeneous pathologic grouping

Due to various mechanisms being responsible for coronary embolic phenomena leading to acute coronary syndromes, we will address them one by one in the

subsequent discussion. The authors' goal is not to provide an exhaustive description of each mechanism, but rather to point the reader to other definitive sources for further details.

5.1 Thrombotic causes

Coronary emboli may be formed due to thromboembolic causes involving different etiologies and pathways (**Table 1**). As with all thromboembolic phenomena, predisposing conditions of the Virchow's triad (hypercoagulability, stasis, endothelial injury) will need to be present for thrombi to form [36, 37].

For venous thromboemboli to "transform" into coronary emboli, the presence of a patent foramen ovale is required [6, 38]. This enables the embolus to cross from "right to left" side of the heart and thus develop the potential to lodge in the coronary circulation [18, 38]. A thrombus may originate in the left atrial appendage, as seen among patients with atrial fibrillation [39, 40], or it may originate in the left atrium/ventricle itself, as in patients with severely reduced ejection fraction or those who have had an anterior/apical myocardial infarction in the past [6, 41]. The former is of particular clinical importance, as patients diagnosed with coronary embolus may benefit from ambulatory monitoring to look for atrial fibrillation as a possible underlying cause.

Arterial emboli are more likely to be reported in the setting of hypercoagulable states including autoimmune diseases, inherited coagulation factor deficiencies, hyperviscosity syndromes, and acquired hypercoagulabe states (e.g., pregnancy, malignancy, previous heparin exposure, **Table 1**) [42–44]. As coronary emboli are a rarely reported phenomenon, no randomized trials or guidelines exist regarding diagnostic workup for these, although it would not be unreasonable to initiate workup for thrombophilia whenever appropriate diagnosis or suspicion exists [37, 42–45].

5.2 Septic/infectious causes

Infective endocarditis is one of the most dreaded infectious etiologies associated with significant morbidity and mortality [46, 47]. Coronary septic arterial emboli (CSAE) secondary to infectious endocarditis have been reported and according to one source such events may carry a mortality of up to 50% [48]. CSAE appear to be more likely to occur in patients having vegetations of the mitral valve or fungal infections, as fungal vegetations are known to reach larger overall dimensions, thus increasing the cumulative possibility of embolization [49, 50]. Rheumatic heart disease, though more common in low-income countries, is another possible etiology that can be associated with CSAE and must be kept in mind when evaluating patients from high-incidence geographic areas [51, 52].

5.3 Neoplastic causes

Tumors originating in the heart such as atrial myxomas, or on valves such as papillary fibroelastomas, are well known to cause cryptogenic brain infarctions [14, 53]. There are also reports of embolization to the coronary circulation [54–56]. Given that end-organ damage, including cerebrovascular accidents may constitute the initial clinical presentation of such neoplasms, it would not be unreasonable to propose that an embolic myocardial infarction may occur in this setting [56, 57]. It is also likely that such occurrences are under-recognized and probably more common than generally thought, thus requiring high index of clinical suspicion and prompt diagnosis [56, 57]. The overall urgency is highlighted by the possibility that subsequent presentations in cases of "missed diagnosis" may manifest as unexplained/

sudden death [57, 58]. Appropriate high-quality imaging may include but is not limited to transthoracic and/or transesophageal echocardiography [56, 59–61].

5.4 Valve-related causes

Stenotic heart valves resulting from progressive calcification process also pose the possibility of calcific embolization to distal locations, including the coronary circulation [48, 62, 63]. Rheumatic valvular heart disease could be another possible risk factor for coronary embolization [48]. Long-term valvular heart disease leads to structural changes in the myocardium, eventually increasing the risk of atrial fibrillation, which in itself may be a contributor to both systemic and coronary embolization [39, 64]. Of note, coronary embolism has been reported following aortic and mitral valve replacement, with successful management reported to involve abciximab and urokinase [65]. Another report describes acute myocardial infarction due to coronary embolism in a patient with mitral valve prosthesis. That particular case was successfully managed using angioplasty [66]. An example of a left coronary embolism associated with subtherapeutic oral anticoagulation in a patient with mitral and aortic mechanical valve prostheses is shown in **Figure 2** [67].

5.5 Iatrogenic causes

Ruptured atherosclerotic plaques in the coronary arteries may lead to acute thrombotic occlusions and are the frequent pathophysiologic factor behind acute ST-elevation myocardial infarctions [68, 69]. Vessels affected by such processes may be characterized by a high thrombotic burden. For example, saphenous venous grafts in post-coronary artery bypass graft patients seem particularly vulnerable [70, 71], with various pathophysiologic mechanisms proposed including immune-mediated process [71, 72].

Cardiac catheterization procedures may also cause distal embolization of intravascular particles [73]. Depending upon where, and how far, any dislodged thrombi or microthrombi travel, periprocedural myocardial infarction can become a very real risk [74]. Various procedural techniques including specialized "wire filter" protection devices [74, 75] and thrombus extraction catheters [76] can be utilized during coronary interventions to prevent or reduce distal embolization. Finally,

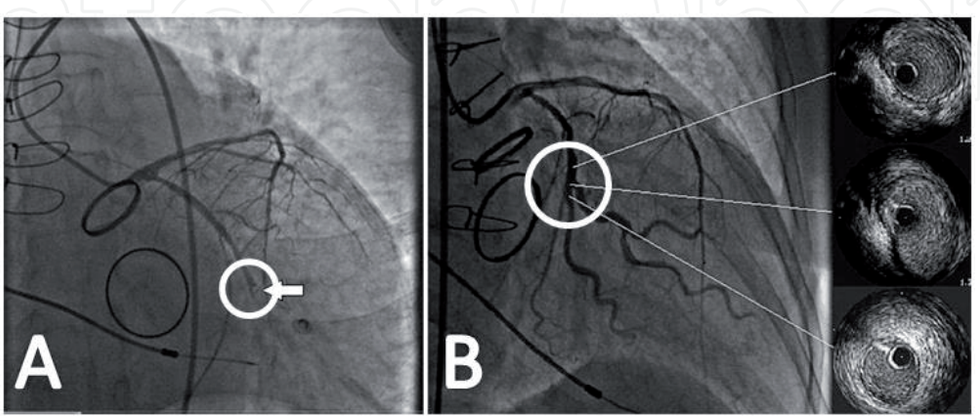


Figure 2.
An example of a left coronary artery coronary embolism associated with subtherapeutic anticoagulation in the setting of mitral and aortic mechanical valve prostheses. [A, left] Note the filling defect present upon initial diagnosis. [B, right] Following thrombectomy, the left coronary artery is seen to be patent. Source: Protasiewicz et al. [67]. Images used under the terms of the Creative Commons Attribution-Noncommercial 3.0 Unported License, which permits all noncommercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

distal coronary embolization involving cholesterol particles is also a possibility in patients undergoing diagnostic coronary angiography or thrombolysis [77, 78].

6. Diagnosis of coronary embolism

A careful history and physical examination is necessary, with specific focus on finding any systemic signs of emboli in septic patients, as well as the possibility of an autoimmune disease in the subset of non-septic patients [79–81]. As with suspected coronary artery disease, patients suffering from coronary embolism may present with typical or atypical chest pain or with "angina equivalents" such as dyspnea [79, 81, 82]. As with all acute coronary syndromes, electrocardiography will be very important in determining the diagnosis and may dictate the urgency for cardiac catheterization (e.g., the presence of ST-elevation myocardial infarction). The presence of Q-waves in contiguous leads may be indicative of a "silent" myocardial infarction. Troponin and other cardiac enzyme testing certainly plays an important role in determining the extent and the progression of myocardial ischemia [83, 84]. Subsequent workup should include transthoracic and transesophageal echocardiography, advanced high-resolution imaging (e.g., CT or MRI), and coronary angiography [18, 85–87]. In addition, miscellaneous adjunctive diagnostic tools, such as Holter/event monitoring, can also be helpful in cases where etiology of the event(s) in question may be uncertain [88, 89].

6.1 Coronary angiography

Coronary angiography remains the mainstay of CEP diagnostics [87]. As outlined previously, patients affected by this condition may have "silent" myocardial infarction or may present with an acute ST segment elevation myocardial infarction. When performing angiography, associated thrombi have a distinct hazy angiographic appearance [87, 90, 91]. Moreover, angiography can help document the evolution and resolution of coronary embolism [92]. Finally, diagnostic angiography can be converted into a therapeutic procedure if indicated [87, 93].

The angiographer should keep in mind that the presence of multiple acute thromboembolic lesions in various vessels increases the suspicion for embolic coronary phenomena [94]. As mentioned above, these emboli may also acutely occlude parts of vessels with pre-existing atherosclerosis, further complicating the diagnosis. Intravascular ultrasound following aspiration atherectomy may be useful when assessing for underlying atherosclerosis versus purely acute thromboembolic phenomena. Optical coherence tomography of these vessels may also be useful but has not yet been studied sufficiently in this particular setting [95, 96]. A patient with angiographic evidence of coronary embolism but with no traditional risk factors for coronary artery disease should raise the suspicion for some of the less common causes (e.g., autoimmune, infectious, inflammatory, or neoplastic) [6, 94, 97].

After diagnostic confirmation, coronary thrombi are often removed using aspiration catheters, as outlined in previous paragraphs. Biopsy of these specimens would aid in differentiating between thrombotic, septic, and neoplastic causes of embolism, particularly due to the fact that these may be the presenting events in some neoplasms. Autoimmune disease may also need to be ruled out [6, 94, 97].

6.2 Transthoracic echocardiography

Transthoracic echocardiography should be a part of the routine workup for patients with suspected CEP. Diagnostically, it will be critically important to

demonstrate or rule out the presence of patent foramen ovale [98–100] and identify any thrombi in left-sided cardiac chambers, particularly with the help of ultrasonic contrast [33, 59]. Any suspicion should be further supplemented with transesophageal echocardiography to ascertain any transthoracic echocardiography findings, especially those of uncertain significance or insufficiently granular detail(s) [59, 101]. In addition, this would also be helpful to visualize the left atrial appendage when looking for evidence of either stasis or thrombus formation there [102, 103]. Such findings can be present in the setting of atrial fibrillation [102].

As outlined earlier in this manuscript, Holter/event monitoring to look for atrial fibrillation would also be reasonable in patients being seen for embolic phenomena [88, 89]. As for all thromboembolic diseases, thrombophilia workup would also be useful in ascertaining the etiology of coronary embolism in appropriately selected at-risk patients [104].

7. Clinical management

Coronary embolic syndromes are quite heterogeneous, and lack randomized controlled trial data or specific guidelines on their management. The initial approach including timing of cardiac catheterization for coronary embolism should be the same as for routine acute coronary syndrome (with classification of available evidence quality provided in parentheses) [106].

Oxygen (Class 1), nitrates (Class 1), and beta blockers (Class 1) are the mainstay of the initial medical management [106] in addition to parenteral anticoagulation (Enoxaparin/unfractionated heparin [UHF]/Bivalirudin) [109].

Decision regarding the use of percutaneous coronary intervention versus balloon angioplasty would be up to the clinician's judgment given plaque morphology as assessed by intravascular ultrasound as well as on optical coherence tomography.

Following the initial management, dual antiplatelet inhibition would be recommended for these patients [107] for a duration of 6–12 months as per the 2017 American College of Cardiology (ACC)/American Heart Association (AHA) Guidelines [108].

As no randomized controlled data are available on lipid management for the particular subset of patients suffering from coronary embolism, we would recommend following current society guidelines for lipid management in these patients.

For patients with reduced ejection fraction on echocardiography, angiotensin converting enzyme (ACE) inhibitors or angiotensin receptor blockers in addition to aldosterone antagonists are recommended (Class 1) [106, 109].

Long-term anticoagulation in patients diagnosed with embolic coronary disease remains a question to be answered. As with other embolic phenomena, 3–6 months of anticoagulation with warfarin or with direct anticoagulants would be reasonable, with further therapy to be decided upon ascertaining the underlying etiology.

Workup to determine the etiology is essential, and treatment of the cause of embolism is of course necessary. As mentioned above, remote cardiac monitoring to look for atrial fibrillation is essential as it may necessitate lifelong anticoagulation particularly in patients with high CHADS2VASC scores.

Lastly, in patients possibly requiring triple antithrombotic therapy, data are limited, with current management approaches based on consensus recommendations with only a brief mention in the 2016 ACC Guidelines [108]. The decision regarding the duration or discontinuation of triple therapy versus P2Y12 inhibitor plus vitamin K antagonists (VKA)/direct oral anticoagulants (DOAC) would be based on the individualized bleeding risk versus the potential risk of discontinuing these medicines [108].

8. Miscellaneous causes

It has been reported that air embolism can complicate a variety of invasive procedures involving the vasculature, from central venous access placement to coronary artery bypass grafting [105, 106]. In the context of CEPs, the presence of patent foramen ovale (PFO) plays an important contributory role [107]. Though rarely reported, air embolism due to decompression illnesses or due to iatrogenic causes may also cause coronary embolism. Finally, iatrogenic CEPs are fortunately uncommon, yet they are dreaded events that may occur in the cardiac catheterization lab or during coronary artery bypass graft (CABG) surgery [103, 104].

Amniotic fluid embolism (AFE) in pregnant women can also lead to coronary embolization [105, 108]. Of note, for amniotic fluid to embolize to the coronary arteries, the patient must also have a PFO which helps facilitate the right-to-left transit of causative particles, which then lodge in the systemic arterial system and, potentially, the coronary arteries. It has been noted that the appearance of amniotic fluid emboli in the coronary circulation may be associated with elevated mortality when compared with cases not involving the coronary vessels [108]. Marked constriction of coronary arteries has also been described in the setting of AFE, although it is not known if that is a direct or an indirect effect [109, 110].

9. Conclusion

Coronary embolic phenomena are a heterogeneous group of clinicopathologic entities attributable to a variety of etiologic factors. Due to their rarity and the tendency to clinically mimic other coronary syndromes, CEPs are often underdiagnosed. Timely diagnosis using an elevated index of suspicion in high-risk patients is important to improving the associated morbidity and mortality. Scarcity of high quality data regarding CEPs necessitates further studies and dedicated consensus guidelines. Progress in diagnosis and treatment of CEPs will require concerted efforts by clinicians, educators, and researchers.

Author details

Qasim Malik[1,2], Ambreen Alam[1,2], Stanislaw P. Stawicki[1,2*] and Peter Puleo[1,2]

1 Department of Research and Innovation, St. Luke's University Health Network, Bethlehem, Pennsylvania, USA

2 Heart and Vascular Center, St. Luke's University Health Network, Bethlehem, Pennsylvania, USA

*Address all correspondence to: stawicki.ace@gmail.com

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