We report a case of left main coronary artery (LMCA) aneurysm rupture while the patient was hospitalized and immediately after imaging with 2 noninvasive cardiac modalities.

An 85-year-old man with history of atrial fibrillation, prostate cancer, hypertension, and hyperlipidemia presented with sudden lightheadedness, chest pressure, and junctional bradycardia (heart rate, 40 bpm). He was admitted to a telemetry bed; warfarin was held and unfractionated heparin was administered. The following day, heparin was held because of supratherapeutic-activated partial thromboplastin times. Transthoracic echocardiogram was performed demonstrating normal ejection fraction, normal wall motion, mild aortic root dilatation, and no pericardial effusion; nothing unusual about the coronary anatomy was noted. Computed tomography coronary angiography to evaluate for coronary artery disease and computed tomography of the chest with contrast were performed shortly afterward. Preliminary reports for these studies indicated no pulmonary embolus, mildly aneurysmal thoracic aorta, no thrombus in the left atrial appendage, and <50% stenosis of the left anterior descending ostium. A subsequent final interpretation reported an aneurysmal LMCA (Figure 1). Shortly after returning to his room, the patient developed sudden, severe hypotension and left chest discomfort. Intravenous fluids and vasopressors were administered with minimal effect. Urgent repeat transthoracic ECG showed a new, large pericardial effusion with tamponade (Figure 2). Because of refractory hypotension, emergency pericardiocentesis was arranged.

The patient arrived at the cardiac catheterization laboratory awake but with a blood pressure of 70/30 mm Hg. An arterial catheter was placed, a right heart catheter was advanced to the pulmonary artery, and subxiphoid pericardiocentesis was performed with echocardiographic monitoring; agitated saline injections confirmed correct placement of the drainage catheter. Bright red blood was obtained with pericardiocentesis. After removal of ≈120 cc, the patient became hypertensive to a systolic blood pressure >200 mm Hg; vasopressors were stopped. Within 60 seconds the patient complained of chest pain and became asystolic; resuscitation ensued. Echocardiography revealed no new structural changes but minimal myocardial contractility. Coronary angiography revealed disruption of the LMCA, and contrast pooling within the ascending aorta did not opacify the right coronary artery (Figure 3; Video I in the online-only Data Supplement). The aortic root appeared narrow but not disrupted. Thrombectomy and angioplasty were unsuccessful in restoring normal flow or cardiac function and the patient was pronounced dead.

Postmortem examination revealed the following: (1) a ruptured LMCA aneurysm with hematoma extending into the mediastinum and atrioventricular sulcus compressing the orifice of an otherwise normal right

Figure 1. Large left main coronary artery (LMCA) aneurysm on coronary computed tomography angiogram. A, Aneurysm originates at orifice and involves whole of LMCA (arrow). B, Maximal dimension of aneurysm is 3 × 1.5 cm (arrow). AO indicates aorta.
coronary artery, (2) significant atherosclerosis in the mid-left anterior descending, (3) no coronary thrombus, and (4) an intact aorta with no evidence of aortic dissection (Figure 4). Histopathology demonstrated thinning but not significant atherosclerosis or inflammation in the aneurysmal tissue (Figure 5). Subsequent 3-dimensional reconstruction of the coronary computed tomography provided premortem confirmation of the original aneurysm location and features (Figure 6).

Coronary artery aneurysms (defined as >1.5-fold dilation of a coronary segment) are uncommon, reported between 0.15% and 4.9% of patients undergoing coronary angiography; LMCA aneurysms are rare, with none seen in a survey of 1200 coronary aneurysms by Swaye et al. Topaz et al found 22 LMCA aneurysms in a series of 22,000 catheterizations, all associated with atherosclerotic coronary artery disease. Aneurysms are associated with atherosclerosis (about 50% cases), Kawasaki disease, Takayasu disease, syphilis, Marfan syndrome, Ehlers-Danlos syndrome, polyarteritis nodosa, or trauma. Complications of aneurysms include thrombosis, embolization, and ischemia, whereas rupture appears rarely; there are few reports describing ruptured coronary aneurysms, except those caused by Kawasaki disease in children.

Best treatment for coronary artery aneurysm is uncertain. Medical management includes use of anticoagulants.

Figure 2. A, Initial echocardiogram shows no significant pericardial fluid. B, Within 2 hours, large pericardial fluid collection with tamponade is seen. LA indicates left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; and PE, pericardial effusion.

Figure 3. Angiography during resuscitation efforts with a guiding catheter in the ostium of the left main and a PA catheter crossing the left main. The angiogram demonstrates a disrupted left main coronary artery with minimal blood flow into left anterior descending and LCX (large arrow) and no opacification of the right coronary artery from contrast pooling in aorta (small arrow).

Figure 4. Postmortem examination findings. A, Large, fresh mediastinal and pericardial hematoma (arrow). B, Probe passed through incision in anterior aorta (short arrow) into left main coronary artery orifice passes through rupture and into mediastinal space (long arrow). AO indicates aorta; LA, left atrium; and LV, left ventricle.
Left Main Coronary Artery Rupture

or antiplatelet drugs to prevent thromboembolism. Surgical ligation is not generally indicated but should be considered for large aneurysms with evidence of recurrent thrombosis and embolization. The role of percutaneous coronary intervention is unclear.

Rupture of an LMCA aneurysm has not been described previously in the literature. The timing and setting of the described event allows detailed analysis of the LMCA aneurysm as it was proceeding to rupture (Video II in the online-only Data Supplement). Presenting symptoms may have been triggered by aneurysm expansion and blood extravasation. Anticoagulation therapy may have exacerbated this, although frank rupture with tamponade occurred several hours after heparin was discontinued. The aneurysm was not atherosclerotic or inflamed. Although quite rare, the potential sensitivity of such aneurysms to anticoagulation and blood pressure changes should be noted, as the event may be lethal even when occurring in an intensive care setting.

The course of this case reinforces earlier reports of hemodynamic collapse after pericardiocentesis for tamponade complicating aortic dissection; these reports noted that a sudden rise in blood pressure associated with relief of tamponade can lead to acute decompensation. Sudden death in our patient followed a rapid rise in blood pressure and appeared to be caused by catastrophic disruption of the LMCA with bleeding around the atrioventricular sulcus, compressing the ascending aortic root and the right coronary orifice. Despite the need for pericardiocentesis to relieve severe tamponade, the potential for an acute rise in blood pressure to increase bleeding should be recognized. If the diagnosis of ruptured aneurysm is entertained, only sufficient pericardial fluid to alleviate critical hypotension should be removed and vasopressors should be discontinued, while preparations are made for prompt surgery or urgent catheter intervention to contain bleeding.

Disclosures

Dr Garratt is on the medical advisory board and serves as a consultant for the Medicines Company.

References


Key Words: cardiac catheterization • cardiac tamponade • heparin
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