INTRODUCTION

Intramyocardial dissecting hematoma (IDH) is a rare but life-threatening complication of acute myocardial infarction. The causative mechanism is postulated to be hemorrhagic dissection of spiral myocardial fibers after acute myocardial infarction. We present an unusual case of IDH after percutaneous coronary intervention (PCI) of an occluded left anterior descending artery (LAD) in a patient with old anterior myocardial infarction.

CASE PRESENTATION

A 59-year-old man who enjoyed good health in the past presented with chest discomfort, palpitation, and shortness of breath for 2 days. Electrocardiogram (ECG) showed regular wide complex tachycardia (140 bpm) with left bundle branch block (LBBB) morphology (Figure 1A). The ECG morphology was compatible with monomorphic ventricular tachycardia (VT). Radiography of chest showed mild congestion of lung. He was conscious, but his blood pressure (BP) was low (90/60 mm Hg). Synchronized cardioversion successfully reverted the VT into sinus rhythm (SR). However, his BP remained borderline. Repeated ECG showed SR with right bundle branch block morphology. Q wave was noted over V2 to V5, and 1 mm ST segment elevation was noted over V2 and V3. The corrected QT interval was prolonged (580 msec; Figure 1B). The first troponin I was 40.0 ng/L (normal, <19.8 ng/L). The next troponin I 4 hours later was 34.1 ng/L. The serial troponin I levels did not suggest acute ST elevation myocardial infarction. His BP worsened on post-PCI day 3 with systolic BP 109 mm Hg (normal, <120 mm Hg). Lignocaine was also not used because of liver disorder. Intravenous esmolol was used for VT control. Emergency coronary arteriogram was done to look for any persistent coronary ischemia. Coronary arteriogram showed total occlusion at the middle part of the LAD and moderate stenosis over the middle part of the right coronary artery and proximal part of the left circumflex artery (Figure 2A). We decided to open up all three vessels (Figure 2B). Stenting was performed for all three vessels (Figure 2B). In view of his borderline BP and recurrent VT, intra-aortic balloon pump (IABP) was inserted for hemodynamic support, and a temporary transvenous pacing wire was placed over the right ventricular apex for overdrive pacing to suppress VT. Anticoagulation with low molecular weight heparin was started for use of IABP.

Despite overdrive pacing of 100 bpm, the patient still had recurrent monomorphic VT, so oral mexiletine was added on top of intravenous esmolol. His BP worsened on post-PCI day 3 with systolic BP.
Figure 1  (A) ECG on admission showed regular wide complex tachycardia (140 bpm) with LBBB morphology. (B) ECG after synchronized cardioversion showed SR with right bundle branch block morphology, Q wave in V2-V5, 1 mm ST elevation in V2-V3, and prolonged QTc 580 msec.

Figure 2  (A) Coronary arteriogram (right anterior oblique cranial view) showed total occlusion over middle part of LAD. (B) Coronary angiogram (right anterior oblique cranial view) after stenting of the middle part of the LAD.
dropping from 100 mm Hg to 60-70 mm Hg. Echocardiogram on post-PCI day 4 showed similar thinning and akinesia of LV apex and middle-to-distal anteroseptal wall. In addition, a new flap-like mobile sheet extending from the LV middle anteroseptal wall to the apex was noted. There was a large echo-free space between the flap-like sheet and LV septum and apex (Figure 3A; Videos 1 and 2). The was no Doppler flow between the LV cavity and the echo-free space (Figure 3B; Video 3). The differential diagnoses of the mobile flap-like sheet in echocardiogram includes LV thrombus, pseudoaneurysm, prominent trabeculation, and IDH. No Doppler flow between the echo-free space and LV cavity speaks against pseudoaneurysm. The presence of a newly developed flap-like sheet in echocardiogram on post-PCI day 4 but not the echocardiogram on admission makes prominent trabeculation unlikely. Judging from the morphology of the mobile flap-like sheet on echocardiogram, the differential diagnosis of IDH should be higher on the list than LV thrombus. Further advanced imaging (e.g., magnetic resonance imaging or computed tomography) to confirm the diagnosis was not feasible because of the patient’s hemodynamic instability and the presence of IABP. The provisional diagnosis at that time was IDH causing reduction in volume of the LV cavity and reduction in LV filling and stroke volume. The cardiothoracic surgical team suggested conservative management at that time and monitoring for any progression of IDH. Anticoagulation was continued because of the presence of IABP. Ventricular tachycardia still recurred but at a lower frequency with the use of overdrive pacing and anti-arrhythmic drugs. The initial plan of VT ablation if medical therapy failed was not feasible in view of the presence of IDH.

The hemodynamics of the patient gradually improved over days. Repeated echocardiogram on post-PCI day 7 showed significant reduction in the echo-free space (Figure 4; Videos 4 and 5). The VT was also well controlled with anti-arrhythmic drugs and overdrive pacing. The overdrive pacing was stopped on post-PCI day 7. Intravenous esmolol was changed to oral metoprolol. There was no recurrent VT.

Figure 3  (A) Post-PCI day 4 echocardiogram showed a newly developed mobile flap-like sheet extending from the middle of the LV anteroseptum to the apex and an echo-free space.  (B) Color Doppler showed no flow between LV cavity and the echo-free space.
Figure 4 Post-PCI day 7 echocardiogram showed significant reduction in IDH.

Figure 5 (A) Post-PCI day 10 echocardiogram showed complete resolution of IDH. (B) Use of an ultrasound enhancing agent confirmed complete resolution of IDH.
on oral mexiteline and metoprolol. Repeated echocardiogram with echo contrast on post-PCI day 10 showed complete resolution of the flap-like sheet and echo-free space (Figure 5A and B; Videos 6-9). The IABP was removed on post-PCI day 11. Anticoagulation was stopped. He remained free of cardiac symptoms afterward. Implantable cardioverter-defibrillator was scheduled later for secondary prevention.

DISCUSSION

Intramyocardial dissecting hematoma is a rare but life-threatening complication of acute myocardial infarction. Intramyocardial dissecting hematoma after acute myocardial infarction is postulated to be related to hemorrhagic dissection of spiral myocardial fibers causing blood leaking within the myocardium while endocardium and epicardium remain intact. Intramyocardial dissecting hematoma can also occur after septal channel perforation during PCI of chronic total occlusion especially with retrograde approach. We present an unusual case of IDH after an uncomplicated antegrade PCI of an occluded LAD in a patient with old anterior myocardial infarction and VT.

Our patient presented with monomorphic VT with congestive heart failure. The initial ECG was a regular wide complex tachycardia of LBBB morphology with leftward axis suggesting possible inferior apical LV origin of VT. The post-cardioversion ECG suggested likely anterior myocardial infarction with Q waves in leads V2 to V5 and with 1 mm ST segment elevation in leads V2 and V3. The anterior myocardial infarction shown on the ECG was probably old because there was no typical rise and fall in cardiac enzymes in this admission. The echocardiogram also showed old anterior myocardial infarction as evidenced by the wall thinning of the LAD territory. The liver disorder (high alanine aminotransferase level) was likely due to ischemic hepatitis related to low cardiac output during VT. Whether to revascularize the likely infarcted LAD territory is controversial, but it seems appropriate to try opening the LAD up to correct any potential reversible causes of recurrent VT. Furthermore, it would be more appropriate to perform functional study (e.g., pressure wire) to document any significant ischemia over the left circumflex artery and right coronary artery territories before stenting. The development of IDH in our patient was unexpected because his anterior myocardial infarction was old and there was no visible vessel perforation during the PCI procedure. Nevertheless, we could not visualize extravasation of microvascularization on fluoroscopy. We could not define the exact time of myocardial infarction. However, judging from the successful crossing of LAD total occlusion with a non-chronic total occlusion guide wire, his myocardial infarction was unlikely to be very chronic. Previous proposed mechanisms of IDH include reduced tensile strength of infarcted myocardium, acute increase in capillary perfusion pressure, and rupture of intramyocardial vessels into the media.1 Our case might prove that reopening of the LAD in a chronically infarcted LAD territory could potentially cause an increase in perfusion pressure to infarcted myocardium and result in IDH even if the myocardial infarction is not acute. The Occluded Artery Trial2 showed that late PCI to open the occluded infarct-related artery did not improve composite endpoints of death, rehospitalization for heart failure, or myocardial infarction. Our case showed that late PCI to open the occluded infarct-related artery might not do no good but may also cause a rare complication of IDH.

The management of IDH is controversial and should be individualized.1 The decision to proceed to surgery or not depends on multiple factors including the patient’s hemodynamics and progression of IDH. Conservative treatment is an option for patients with small IDH and stable hemodynamics.4 Surgery is usually reserved for those with rapid progression. Our case was successfully managed with conservative treatment, although his IDH was not small and his initial hemodynamics were unstable. This case highlights the importance of individualized treatment at different time points. Frequent monitoring of the size of the IDH is essential. From the literature we can see that the resolution of IDH on echocardiogram varies from around 2 weeks to more than 3 months.4,5 The recovery after IDH in our case was surprisingly fast, at around 1 week. The use of IABP in our case might play a role in the hastened resolution of IDH. The deflation of the IABP balloon during systole leads to a reduction in LV afterload. Reduction in afterload and resting of the left ventricle speeds up the myocardial recovery and resolution of IDH. The in-hospital mortality of IDH was high (23%) in one case series.5 The risk factors for mortality in this case series including low ejection fraction (<35%) and anterior myocardial infarction were all present in our patient. Fortunately, our patient survived this life-threatening complication.

CONCLUSION

Our case shows that IDH can happen after revascularizing a chronically infarcted LAD territory in a patient with old anterior myocardial infarction. The management of IDH should be individualized depending on the patient’s hemodynamics and progression of IDH. The role of IABP in the recovery after IDH in our case is provocative and needs further study.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2021.07.015.

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