Cardiac Arrest Triggered by Subepicardial Aneurysm Without Cardiac Rupture

Eriko Hasumi, MD, PhD; Katsuhito Fujiu, MD, PhD; Teruhiko Imamura, MD, PhD; Hiroshi Iwata, MD, PhD; Daigo Sawaki, MD, PhD; Yumiko Hosoya, MD, PhD; Jiro Ando, MD; Toshiya Kojima, MD, PhD; Yu Shimizu, MD; Gaku Oguri, MD, PhD; Takumi Matsubara, MD; Masaru Hatano, MD; Hiroshi Akazawa, MD, PhD; Masafumi Watanabe, MD, PhD; Minoru Ono, MD, PhD; Issei Komuro, MD, PhD

Figure 1. (A) Electrocardiogram during cardiopulmonary resuscitation. Pulseless electrical activity (idioventricular rhythm) was recorded immediately after onset of subepicardial aneurysm (SEA). Arrows, idioventricular rhythm. (B–D) Echocardiogram of SEA. (B) Transthoracic echocardiogram showing an abnormal cavity communicating with the left ventricle (LV) through a small hole (neck) at the apex, which expanded during systole and collapsed during diastole (Movie S1). (C) Blood is seen to flow through the hole from the LV in diastole on Doppler color flow imaging (Movie S1). The walls of the aneurysm were thin, and the myocardium showed sudden discontinuity at the neck of the aneurysm. (D) Thrombus was observed on the dividing wall between SEA and the LV. (E) Enhanced computed tomography showing SEA adjoining the LV. Ao, aorta; LA, left atrium.
Subepicardial aneurysm (SEA) is an infrequent complication after myocardial infarction (MI). SEA is defined as abrupt interruption of the endocardium/myocardium without interruption of the epicardium. The chief morphological feature of SEA consists of a narrow neck between the left ventricular (LV) chamber and the aneurysmal space.

Thirty percent of SEA patients progress to frank rupture and die if they do not receive surgical repair. SEA without cardiac tamponade, however, has been found in autopsy cases of MI, indicating that SEA can lead to death by causes other than cardiac free wall rupture and cardiac tamponade. We describe here the first case of cardiac severe arrhythmia caused by SEA.

Figure 2. Intraoperative findings and surgical repair. (A) The aneurysm (*) was seen at the apex of the left ventricle (LV). (B) The aneurysm wall was opened, and a thrombus in the cavity and the hole linking it to the LV cavity on the dividing wall (**) were observed. The diameter of the orifice connecting the aneurysm and the LV was 2.5 cm. (C) On pathology of the resected specimen, the aneurysm wall was composed of epicardium and scar fibers without a layer of myocardium. There was no tear in the surface of the aneurysm wall. (D) The dividing wall was composed of a layer of myocardium and coronary artery. The thrombus contained coagulative necrosis of the myocardium. (E) The aneurysm wall and dividing wall were excised, and the entrance path was closed with a Hemashield patch. (F) In addition, felt was used for patch reinforcement and prevention of hemorrhage.
without cardiac tamponade. A 70-year-old man with a history of hypertension and smoking had pulseless electrical activity (PEA) 6 days after percutaneous catheter intervention (PCI) due to MI (Figure 1A), and standard cardiopulmonary resuscitation restored sinus rhythm and hemodynamic state. SEA was identified on echocardiography (Figures 1B–D, Movie S1) and computed tomography (Figure 1E, Movie S2). Before PEA, no risk factors for cardiac arrest or fatal arrhythmia were observed, such as reduced LV dysfunction, ventricular arrhythmia, or thinning or significant aneurysmal change of the infarcted area. Because of the favorable clinical course after PCI, the patient was transferred to another hospital for cardiac rehabilitation without a wearable or implantable cardioverter-defibrillator.1 During transfer by ambulance, PEA suddenly occurred.

The SEA was resected and the defect was closed by patch repair (Figure 2). The diagnosis of SEA was confirmed on pathology of the resected specimen during surgical repair (Figures 2C,D).

The present patient developed SEA and subsequent cardiac arrest without cardiac rupture. This indicates that unruptured SEA is a potential cause of cardiac arrhythmia or cardiac arrest. Whereas cardiopulmonary resuscitation was initially required for stabilization of the hemodynamic condition, sinus rhythm and hemodynamic state were gradually restored and inotropic agents were not needed after sinus rhythm recovery. This suggests that SEA directly provoked PEA via disruption of the myocardium rather than through development of irreversible severe pump failure due to cardiac rupture or cardiac tamponade. In contrast, LV aneurysm after MI is also a major cause of ventricular arrhythmia even if it is not ruptured. LV aneurysm without other complications after MI, however, is not suitable for surgical repair. Only if LV aneurysm is combined with severe heart failure, severe arrhythmia, thromboembolism and/or post-MI angina, can surgical repair be considered.

Elective cardiac surgical repair can completely ameliorate the pathological condition, SEA with impending rupture could be a novel arrhythmogenic anatomical substrate of life-threatening ventricular arrhythmia. Cardiac surgical repair should be considered as soon as possible when SEA is found, to prevent not only irreversible severe pump failure but lethal arrhythmia.

Disclosures

None.

References


Supplementary Files

Supplementary File 1

Movie S1. Echocardiogram (Doppler color image) of subepicardial aneurysm (SEA; Figure 1C). An abnormal cavity communicating with the left ventricle through a small hole at the apex is observed. Blood flows through the hole from the left ventricle in diastole.

Supplementary File 2

Movie S2. Enhanced computed tomography series (whole body images of Figure 1E). Subepicardial aneurysm adjoining the left ventricle is seen.

Please find supplementary file(s); http://dx.doi.org/10.1253/circj.CJ-15-0830