A 28-year-old male with no previous medical history and worsening shortness of breath for a year suffered a cardiac arrest while playing football. Spontaneous circulation returned after 15 minutes of cardiopulmonary resuscitation. The electrocardiogram showed sinus rhythm, a right bundle branch block (RBBB), and large R waves in leads V1-V2. A pulmonary embolism and intracranial pathology were excluded with computed tomography. A bedside transthoracic echocardiogram revealed dilatation of the right chambers, impaired right ventricular (RV) systolic function, and preserved left ventricular systolic function. The pulmonary artery systolic pressure was calculated at 30–35 mm Hg. An invasive coronary angiogram was not deemed necessary at this point, as channelopathy or arrhythmogenic right ventricular cardiomyopathy was considered a possible diagnosis.

The patient was transferred to the intensive care unit (ICU) due to multiorgan failure following cardiac arrest. A transesophageal echocardiogram confirmed dilatation of the right chambers and impaired RV systolic function (Fig. A). It also showed 2 separate jets of interatrial communication (Fig. B, Video 1 *), comprising a moderate-sized atrial septal defect (ASD) (Fig. C), both of which had bidirectional flow, and a significantly stretched patent foramen ovale (PFO) (Fig. D, E). Three-dimensional modality helped to create an en face view of the interatrial septum. The entire interatrial septum and the complex anatomy with 2 defects could be seen from both the left and right atrial side. (Fig. F, H and Videos 2, 3 * - view from the left atrial perspective; Fig. G and Video 4 * - from the right atrial perspective). In view of these findings, we presumed that chronic volume and pressure overload of the right heart secondary to atrial septal defect had resulted in significant stretching of the PFO. An arrhythmic event due to RV dysfunction was considered the most probable mechanism of the cardiac arrest. An embolic event (paradoxical embolism) in a coronary artery may be another possible mechanism; however, no regional wall motion abnormalities were noted on echocardiography. The electrocardiogram did not reveal any abnormalities apart from RBBB, which was compatible with the underlying pathology.

The RBBB pattern was not suggestive of Brugada syndrome: there were no signs of pre-excitation and the QTc was normal. The co-existence of a primary arrhythmic event (i.e., idiopathic ventricular tachycardia [VT]/fibrillation, catecholaminergic VT, etc.), was considered extremely unlikely in the presence of structural heart disease. After a long stay in the ICU, the patient was transferred to a neurorehabilitation clinic with irreversible hypoxic brain injury.

Figures—(A) Transesophageal echocardiogram, 4-chamber view, showing the dilatation of right chambers (RV basal diameter: 46 mm, RV mid-diameter: 41 mm) and impaired RV systolic function (fractional area change: 28%). LA: Left atrium; LV: Left ventricle; RV: Right ventricle. Transesophageal echocardiogram. (B) Modified mid-esophageal 4-chamber view (20°) with zoom on interatrial septum (IAS) showing the atrial septal defect (ASD) (white arrow) and the patent foramen ovale (PFO) (red arrow). (C) Mid-esophageal 4-chamber view with zoom on IAS showing the ASD. (D) Mid-esophageal bicaval view illustrating left-to-right flow through the PFO. (E) Mid-esophageal bicaval view showing right-to-left flow through the PFO. (F) 3D zoom of the IAS (view from left atrial perspective) showing the ASD and the PFO. (G) 3D zoom of the IAS (view from right atrial perspective) showing the ASD and the PFO. (H) Transesophageal echocardiogram 3D zoom of the interatrial septum (view from left atrial perspective) showing the atrial septal defect (ASD) and the patent foramen ovale (PFO).* Supplementary video files associated with this presentation can be found in the online version of the journal.