Prosthetic Mitral Valve Endocarditis With Left Atrial Dissection

Tamami Fujiishi, MD; Toshimi Koitabashi, PhD; Takayuki Inomata, PhD; Katsuhiko Ohori, MD; Ko Shibata, PhD; Hirotsugu Okamoto, PhD; Kagami Miyaji, PhD; Tohru Izumi, PhD

Figure 1. (A) Transesophageal echocardiography (TEE) showing an 11×4-mm mobile vegetative mass on the lateral mitral annulus (arrow) on admission. (B) A cavity was formed in the left atrial (LA) wall by an abscess (arrow; TEE angle, 15°). (C) The cavity perforated and opened into the LA chamber. Paravalvular leakage (arrow) occurred because of detachment of part of the prosthetic valve from the lateral mitral annulus near the P1 segment of the mitral valve. The left ventriculoatrial shunt was organized with perforation and mitral paravalvular leakage (TEE angle, 15°). (D) TEE showed an abnormal cavity in the LA wall, and blood flow into this cavity on admission (TEE angle, 15°). (E) TEE showed blood flow into the cavity 1 month after hospitalization. Blood flow into the cavity and paravalvular leakage had increased (TEE angle, 75°). (F) Posterior location of this abnormal cavity (*) and an opening into the LA chamber (arrow) were evident on intraoperative real-time 3-dimensional TEE.
The valve is common, reported, however, paravalvular abscess affecting the aortic localized lesion may have been a paravalvular abscess-induced new cavity in the LA wall (Figure 1B). A paravalvular leakage resulted from the detachment of part of the prosthetic valve from the lateral mitral annulus. Moreover, an abnormal cavity that had been formed in the LA wall by an abscess (Figure 1B) had perforated, opening into the LA chamber. The left ventriculattrial shunt was organized with perforation and mitral paravalvular leakage (Figure 1C). Prosthetic valve malfunction and valve seat agitation were not evident. Inflammation diminished after 1 month of optimal antimicrobial pharmacotherapy; subsequently, TEE showed that the vegetation and LA abscess had disappeared. Blood flow into the paravalvular leakage and LA wall cavity, however, had increased (Figures 1D,E). We performed surgical reconstruction of the mitral annulus and redo MVR. During intraoperative real-time 3-dimensional (D) TEE, posterior location of this abnormal cavity and an opening into the LA chamber were evident (Figure 1F). Furthermore, the new cavity was epithelialized inside, with a 1-cm hole penetrating into the true LA chamber (Figure 2A). The mitral annulus was destroyed at the P1 segment of the mitral valve (Figure 2A). Paravalvular leakage resulted from the detachment of part of the prosthetic valve from the lateral mitral annulus. Moreover, an abnormal cavity that had been formed in the LA wall by an abscess (Figure 1B) had perforated, opening into the LA chamber. The left ventriculattrial shunt was organized with perforation and mitral paravalvular leakage (Figure 1C). Prosthetic valve malfunction and valve seat agitation were not evident. Inflammation diminished after 1 month of optimal antimicrobial pharmacotherapy; subsequently, TEE showed that the vegetation and LA abscess had disappeared. Blood flow into the paravalvular leakage and LA wall cavity, however, had increased (Figures 1D,E). We performed surgical reconstruction of the mitral annulus and redo MVR. During intraoperative real-time 3-dimensional (D) TEE, posterior location of this abnormal cavity and an opening into the LA chamber were evident (Figure 1F). Furthermore, the new cavity was epithelialized inside, with a 1-cm hole penetrating into the true LA chamber (Figure 2A). The mitral annulus was destroyed at the P1 segment of the mitral valve, and 1/5 rounds detached from the valve seat, forming a new cavity in the LA wall (Figure 2B).

Regarding the pathomechanism of this rare abnormality, the localized lesion may have been a paravalvular abscess-induced intramyocardial cavity formation in the LA wall. As previously reported, however, paravalvular abscess affecting the aortic valve is common, whereas that affecting the mitral valve is rare. In contrast, one study reported that the site most susceptible to LA dissection following MVR is the posterior LA wall. In the present case, the intramyocardial cavity in the LA wall occurred in the lateral mitral annulus near P1 of the posterior mitral leaflet. Therefore, LA dissection might have existed ab initio in injured annular tissue, which may have been vulnerable due to heavy calcification, and may have been developing since the original MVR. Finally, LA dissection may have been compounded by the vegetation adherent to the annulus and within the abnormal cavity.

In many cases of LA dissection, urgent MVR reoperation is required immediately after its discovery on TEE because of the serious complications of the low cardiac output state and heart failure. In cases of endocarditis with LA dissection, even if the general status, including antimicrobial control, is favorable, serial and close observation with TEE and 3-D TEE is recommended for evaluation of morphological alterations, facilitating selection of suitable and optimally timed surgical correction.

References