CASE REPORTS

Left Ventricular Outflow Tract to Left Atrial Communication Secondary to Rupture of Mitral-Aortic Intervalvular Fibrosa in Infective Endocarditis: Diagnosis by Transesophageal Echocardiography and Color Flow Imaging

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Infection of the mitral-aortic intervalvular fibrosa occurs most commonly in association with infective endocarditis of the aortic valve. Infection of the aortic valve results in a regurgitant jet that presumably strikes this subaortic interannular zone of fibrous tissue and produces a secondary site of infection. Infection of this interannular zone then leads to the formation of subaortic abscess or pseudoaneurysm of the left ventricular outflow tract. This infected zone of mitral-aortic intervalvular fibrosa or subaortic aneurysm can subsequently rupture into the left atrium with systolic ejection of blood from the left ventricular outflow tract to the left atrium.

This report describes the echocardiographic findings in three patients with pathologically proved left ventricular outflow tract to left atrial communication. Precise preoperative diagnosis is important, and this lesion should be differentiated from ruptured aneurysm of the sinus of Valsalva and perforation of the anterior mitral leaflet. Transthoracic echocardiography using color flow imaging and conventional Doppler techniques may show an eccentric mitral regurgitation type of signal in the left atrium originating from the region of the left ventricular outflow tract. However, transesophageal echocardiography provides an accurate preoperative diagnosis and should be used intraoperatively during repair of such lesions.

Various cardiovascular complications of infective endocarditis affecting native or prosthetic aortic valves include (1-13) the following: 1) perforation and destruction of the cusps with aortic regurgitation; 2) perivalvular aortic regurgitation in a prosthetic valve; 3) ring or annular abscess and heart block; 4) sinus of Valsalva aneurysm with or without rupture; 5) mycotic aneurysm; 6) secondary infection of the ventricular surface of the anterior mitral leaflet with abscesses and aneurysm formation, which may eventually perforate and cause severe mitral regurgitation; 7) subaortic, left ventricular outflow tract aneurysm arising from the mitral-aortic intervalvular fibrosa; 8) pericarditis; and 9) systemic embolization.

Annular abscesses have been reported in up to 30% of native valve endocarditis (3,4). The incidence of ring abscesses is much higher in prosthetic valve endocarditis, and the frequency varies from 68% to 100% (5,6). Occasionally, there can be secondary infection of the mitral-aortic intervalvular fibrosa, which is a junctional tissue between the anterior mitral leaflet and the aortic valve. Rupture of this zone may form a pseudoaneurysm of the left ventricular outflow tract (12,13). The pseudoaneurysm may perforate and communicate with the left atrium, causing shunting of blood from the left ventricle through the aneurysm into the left atrium (14,15). Rarely, secondary infection of the mitral-aortic intervalvular fibrosa tissue forms a subaortic abscess that subsequently ruptures and creates a direct communication of the left ventricular outflow tract with the left atrium without an aneurysm formation (16). We report three cases in which transesophageal echocardiography was used to diagnose the infective endocarditis-related rupture of the mitral-aortic intervalvular fibrosa with communication of the left ventricular outflow tract with the left atrium.

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Over a 12 month period after May 1988, three patients were seen who had rupture of the mitral-aortic intervalvular fibrosa with communication of the left ventricular outflow tract to the left atrium. All patients had preoperative transthoracic and transesophageal echocardiography, and the findings were subsequently confirmed at surgery. Echocardiography was performed using a Hewlett-Packard 77020AC color flow ultrasound imaging system. Transthoracic echocardiography was performed from the standard windows with a 2.25 MHz transducer (model 21225A). Transesophageal echocardiography was performed using a Hewlett-Packard, model 21362A transesophageal probe (5 MHz phased array transducer, 64 elements and 90° sector angle). After explanation of the procedure and the potential risks and benefits, informed consent was obtained from each patient before the examination. Each patient fasted for at least 4 h and received local pharyngeal anesthesia (0.5 to 1 ml of 10% Xylocaine spray) and light sedation (2 mg of intravenous midazolam). The technique of transesophageal echocardiography is described elsewhere (17). The duration of each outpatient study was 15 to 20 min and there were no complications.

Case 1

Clinical features. A 45 year old man presented with the complaints of slurred speech and left-sided weakness. His history was significant for chronic intravenous drug abuse. Twelve years earlier, he developed endocarditis of his native aortic valve and underwent surgical replacement of the valve with a Björk-Shiley prosthesis. He admitted to continued intravenous drug abuse, noncompliance with anticoagulant therapy and a 3 month history of fever, night sweats and weight loss.

On examination, the patient was noted to have left-sided hemiparesis with a temperature of 97.5°F, pulse rate 88 beats/min and blood pressure 130/70 mm Hg. Lungs were clear. Cardiac examination showed normal opening and closing clicks of the mechanical aortic valve prosthesis, a grade 2/6 ejection systolic murmur and a grade 2/6 early diastolic decrescendo murmur along the left sternal border. A grade 3/6 apical holosystolic murmur was also noted. There were no peripheral stigmata of infective endocarditis. The white blood cell count was 8,200/mm³ with 70% polymorphonuclear cells, 4% bands and 26% lymphocytes. Blood cultures were positive for penicillin-resistant Streptococcus viridans. He was treated with intravenous vancomycin and gentamicin.

Echocardiography. A real-time two-dimensional transthoracic echocardiographic examination demonstrated a mildly dilated, well contractile left ventricle. Color flow imaging and conventional Doppler studies showed moderate perivalvular aortic regurgitation and an eccentric, high velocity systolic signal in the left atrium originating from the left ventricular outflow tract (Fig. 1 and 2). Transesophageal echocardiography showed moderate perivalvular aortic regurgitation and rupture of the mitral-aortic intervalvular fibrosa and direct shunting of blood from the left ventricular outflow tract to the left atrium during systole (Fig. 2).

Surgery. Two weeks after treatment with intravenous antibiotics, the patient underwent open heart surgery. He was monitored with intraoperative transesophageal echocardiography. After the patient was placed on cardiopulmonary bypass, the aorta was opened and the Björk-Shiley valve was removed from the aortic anulus. Direct inspection revealed a small periannular abscess with dehiscence of the sewing ring. Rupture of the mitral-aortic intervalvular fibrosa and direct shunting of blood from the left ventricular outflow tract to the left atrium during systole was confirmed. Two pledgeted sutures were used to close the fistulous communication. The aortic valve was then replaced with a size 21
Figure 2. Case 1. Transthoracic apical four-chamber echocardiographic view (A) and its schematic (C) show an eccentric, high velocity systolic jet (arrow) from the left ventricular outflow tract (LVOT) to the left atrium (LA). This view is oriented with the apex up and the left ventricle (LV) on the left side. Transesophageal four chamber with outflow view (B) and schematic (D) show clearly that the mitral valve (MV) apparatus is intact and the systolic signal (arrow) originates in the left ventricular outflow tract (LVOT) just below the aortic valve prosthesis (AVP). I = inferior; L = left; R = right; RA = right atrium; RV = right ventricle; S = superior. Calibration marks are 1 cm apart.

Figure 3. Case 2. Transesophageal echocardiographic four chamber view (A) and its schematic (B) showing a thin anterior mitral leaflet and a small systolic, mosaic color signal originating between the mitral and aortic anuli. A small aneurysm noted in real time in the subaortic region is not seen in this figure. This subaortic aneurysm communicated with the left atrium. This represents an aneurysm of the left ventricular outflow tract from the mitral-aortic intervalvular fibrosa with subsequent rupture and communication with the left atrium. AML = anterior mitral leaflet; PML = posterior mitral leaflet; other abbreviations as in Figure 2.

Figure 4. Case 3. Transesophageal four chamber with aorta view (A) and its schematic (C) show an abscess (A) around the ring of the Starr-Edwards aortic valve prosthesis (AVP) and vegetations (V) in the left ventricular outflow tract (LVOT). Four chamber view (B) and schematic (D) show two systolic mosaic color high velocity jets in the left atrium. The jet to the right represents mitral regurgitation (MR), and the other jet originates between the anuli of the mitral and aortic valves and occurs as a result of a ruptured mitral-aortic intervalvular fibrosa. Other abbreviations as in Figure 2 and 3.
St. Jude prosthesis. Transesophageal echocardiography was performed after the heart was rewarmed and the aortic cross clamp removed. It showed persistent shunting of blood from the left ventricular outflow tract into the left atrium through the fistulous communication. The patient was placed back on cardiopulmonary bypass and the left atrium was opened. The mitral valve leaflets appeared in good condition. Multiple sutures with pledgets were placed in an attempt to close the fistula. Transesophageal echocardiography was repeated after closure of the left atrium, rewarming of the heart and removal of the aortic cross clamp. It showed persistent shunting of blood through the fistulous communication, and also moderate mitral regurgitation, probably as a result of excessive traction on the anterior mitral leaflet causing incomplete coaptation of the mitral valve. The patient was finally placed back on cardiopulmonary bypass. The mitral valve was excised and replaced with a size 29 St. Jude prosthesis. Another area of fistula was identified and closed. Repeat transesophageal echocardiography showed good function of both valve prostheses and no evidence of shunting through the fistulous communication. The patient recovered uneventfully and was discharged in stable condition.

Case 2

Clinical features. A 31 year old man was transferred from a local community hospital in critical condition from infectious and hemodynamic sequelae of Streptococcus sanguis endocarditis involving a congenitally bicuspid aortic valve. On examination, he appeared cachectic; temperature was 98°F, pulse 100 beats/min, respiratory rate 24/min and blood pressure 110/70 mm Hg. There was mild jugular venous distension. Carotid pulses showed good volume and rapid upstroke. There were bibasilar pulmonary rales.

Cardiac examination showed a soft first heart sound, normally split second heart sound, a third heart sound, a grade 3/6 systolic ejection murmur and a grade 3/6 early diastolic decrescendo murmur at the base. The systolic murmur radiated widely to the suprasternal notch and axilla. There was mild hepatomegaly. There were no peripheral stigmata of infective endocarditis. Chest radiography revealed moderate cardiomegaly and early pulmonary edema. The white blood cell count was 32,200/mm³ with 74% segmented polymorphonuclear cells, 12% bands, 11% lymphocytes and 3% monocytes. He was treated with penicillin, gentamicin, digitalis, furosemide and captopril.

Preoperative echocardiography. The real-time two-dimensional transthoracic echocardiographic examination showed a markedly dilated left ventricle with normal ejection fraction, a bicuspid aortic valve with vegetation on both leaflets and perforation of the posterior aortic cusp with severe aortic regurgitation. The mitral leaflets appeared normal, but an abscess was noted involving the mitral-aortic intervalvular fibrosa in the subaortic region.

Surgery. Surgical inspection revealed a bicuspid aortic valve with vegetations and perforation; a subaortic abscess was confirmed. The aortic valve was replaced with a size 23 St. Jude prosthesis. The patient was discharged home 2 weeks postoperatively.

Postoperative echocardiography. The patient underwent both transthoracic and transesophageal echocardiography 9 days postoperatively. Transthoracic echocardiography showed a dilated left ventricle with global hypokinesia and an estimated ejection fraction of 35%. Severe perivalvular regurgitation of the aortic prosthesis was noted, as well as eccentric systolic jet in the left atrium originating from the subaortic region. Transesophageal echocardiography showed a small subaortic aneurysm arising from the region of the mitral-aortic intervalvular fibrosa with rupture into the left atrium (Fig. 3). This complication apparently developed postoperatively.

Case 3

Clinical features. A 68 year old man was transferred for management of Staphylococcus aureus infective endocarditis involving his prosthetic aortic valve. Fifteen years earlier, he had undergone replacement of the aortic valve with a Starr-Edwards prosthesis. At the time of transfer, he had congestive heart failure and was being treated with a combination of vancomycin, rifampin and furosemide.

On examination, he had a temperature of 98.3°F, an irregular pulse of 100 beats/min and blood pressure 100/60 mm Hg. There was mild jugular venous distension and there were basilar rales up to the mid-lung fields. Cardiac auscultation revealed a decreased first heart sound, a grade 3/6 ejection systolic and grade 2/6 early diastolic decrescendo murmur at the base and a grade 3/6 pansystolic murmur at the apex. There were no peripheral stigmata of infective endocarditis. The electrocardiogram revealed atrial fibrillation and left bundle branch block. Chest radiography revealed cardiomegaly and moderate pulmonary vascular congestion. The white blood cell count was 11,000/mm³ with 93% segmented polymorphonuclear cells, 2% bands and 5% lymphocytes.

Echocardiography. Transthoracic echocardiography revealed a dilated and hypertrophied left ventricle with global hypokinesia and estimated ejection fraction of 35%. Doppler echocardiography showed moderate mitral regurgitation and aortic regurgitation. The prosthetic aortic valve was poorly visualized. Transesophageal echocardiography showed small vegetations at the base of the Starr-Edwards aortic prosthesis with a subaortic ring abscess and moderate aortic regurgitation. It also showed an aneurysm of the noncoronary aortic sinus that had ruptured into the right atrium. An abscess and a small aneurysm were noted in the subaortic region arising from the mitral-aortic intervalvular fibrosa, with rupture into the left atrium and systolic shunting of
blood from the left ventricular outflow tract into the left atrium (Fig. 4).

Surgery. Direct inspection at the time of surgery confirmed the transesophageal echocardiographic findings. The aortic valve prosthesis was replaced with a size 23 St. Jude prosthesis. The subaortic left ventricle to left atrial communication was closed with pledgeted sutures. A repeat transesophageal echocardiographic examination after completion of the surgery revealed no significant shunt from the left ventricular outflow tract to the left atrium.

Discussion

Anatomy and pathophysiology. The mitral-aortic intervalvular fibrosa is the junctional tissue between the elements of the aortic and mitral valves (12). Infection of the aortic valve (native or prothetic) causes regurgitation, and the regurgitant jet presumably strikes the subaortic structures including the mitral-aortic intervalvular fibrosa, anterior mitral leaflet and the mitral apparatus, and creates secondary sites of infection and complications related to these structures (Fig. 5 and 6). Infection of the mitral-aortic intervalvular fibrosa forms a subaortic abscess that may lead to one of four complications: 1) rupture with slow seepage of blood and formation of a pseudoaneurysm originating from the left ventricular outflow tract (12,13); 2) rupture of the pseudoaneurysm with cardiac tamponade (12,14); 3) rupture of the pseudoaneurysm into the left atrium (14,15); and 4) direct rupture of the subaortic abscess with communication of the left ventricular outflow tract to the left atrium (Fig. 5). The latter two complications can result in shunting of blood from the left ventricular outflow tract into the left atrium, simulating mitral regurgitation by clinical and routine transthoracic Doppler echocardiographic criteria. However, if the secondary site of infection is the anterior mitral leaflet (Fig. 6), it can form an abscess with eventual perforation and mitral regurgitation or aneurysm of the anterior mitral leaflet (9,10).

Diagnosis. Fisher et al. (16) utilized transthoracic echocardiography and color flow imaging and identified a subaortic abscess probably involving the region of the mitral-aortic intervalvular fibrosa; the abscess subsequently ruptured into the left atrium, forming a direct communication between the left ventricular outflow tract and left atrium. Taliercio et al. (15) reported a case of ruptured left ventricular outflow tract aneurysm into the left atrium. Review of the transthoracic echocardiographic and operative findings from these two case reports suggests that the lesions described were probably related to rupture of the mitral-aortic intervalvular fibrosa.

The transthoracic echocardiographic study usually shows an eccentric systolic flow signal in the left atrium, best seen from apical four chamber or parasternal long-axis views. The systolic flow signal arises below the aortic anulus in the left ventricular outflow tract and goes across the left atrium toward the lateral wall. It is frequently not possible to be certain whether it is due to perforation in the anterior mitral leaflet or to perforation in the mitral-aortic intervalvular fibrosa. If the transthoracic echocardiographic examination is technically difficult, as in our Case 3, then this complication may be completely missed. Introduction of transesophageal echocardiography has provided a new acoustic window to the heart (17). By using a high frequency transducer
in close proximity to the heart, it provides a high resolution image of the mitral apparatus and subaortic structures and is invaluable in making a correct and precise diagnosis of this lesion. The successful closure of the communication can and should be confirmed by intraoperative transesophageal echocardiography. This technique has previously been reported to be superior to transthoracic echocardiography for detecting small vegetations in patients with infective endocarditis (18).

Clinical implications. Anatomically, the perforation or defect in the mitral-aortic intervalvular fibrosa is located above the plane of the mitral anulus and below that of the aortic anulus. This complication should be precisely diagnosed preoperatively and differentiated from mitral regurgitation caused by a perforation of the anterior mitral leaflet (Fig. 5 and 6). In severe mitral regurgitation, the surgical treatment is repair or replacement of the mitral valve. However, the lesion described in this report is located above the plane of the mitral anulus; it cannot be corrected by mitral valve surgery alone and will require direct surgical attention. This report underscores the importance of transesophageal echocardiography for the diagnosis and assessment of surgical repair of such unusual lesions related to rupture of the mitral-aortic intervalvular fibrosa.

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References