Case Reports

Bacterial Endocarditis Caused by *Lactobacillus acidophilus*

Leading to Rupture of Sinus of Valsalva Aneurysm

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Lactobacillus acidophilus *rarely causes bacterial endocarditis, because it usually resides in the mucosa of the vagina, gastrointestinal tract, and oropharynx. Moreover, sinus of Valsalva aneurysms are rare cardiac anomalies, either acquired or congenital. We present the case of a middle-aged man whose bacterial endocarditis, caused by* Lactobacillus acidophilus*, led to an aneurysmal rupture of the sinus of Valsalva into the right ventricular outflow tract. The patient underwent successful surgical repair, despite numerous complications and sequelae.* ***(Tex Heart Inst J 2016;43(2):161-4)***

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Lactobacilli are gram-positive, rod-shaped bacteria that are rarely infectious, and their presence as commensal organisms in the gastrointestinal tract is associated with protection against pathogens, stimulation of the immune system, and positive effects on colonic health and the host's nutrition. Nevertheless, lactobacilli have also been identified in some clinical reports as causal agents of dental caries, infectious endocarditis, urinary tract infections, and intra-abdominal, liver, and spleen abscesses.[1] Endocarditis due to *Lactobacillus* is associated with impaired immunity, structural heart disease, recent surgery, prolonged antibiotic therapy, and severe comorbid conditions.[2] Some clinical reports show a 30% mortality rate associated with endocarditis caused by lactobacilli.[3] Probiotic agents that serve as entry vehicles for *Lactobacillus* have been identified as a relatively new risk factor in the genesis of these infections.[4]

Case Report

A 48-year-old man with a history of paroxysmal supraventricular tachycardia (ablated in the past), mild aortic regurgitation, and the implantation (4 years earlier) of upper and lower dental bridges, presented at our emergency department. He reported exertional shortness of breath, increased lower-extremity edema, 3 months of intermittent fever ranging from 100.4 to 102.9 °F, and recent left-upper-quadrant abdominal pain.

The patient was lethargic. His vital signs included a blood pressure of 112/43 mmHg, a heart rate of 113 beats/min, a respiratory rate of 20 breaths/min, and an oral temperature of 99 °F. His examination was notable for jugular venous distention (12 cm), a grade III/IV holosytolic murmur heard over the left lower sternal border, crackles in the lung bases bilaterally, splenomegaly, 3+ pitting edema bilaterally up to the knees, and scattered red macules anterior to the shins. His laboratory results indicated pancytopenia, acute renal failure, and hypoalbuminemia. His white blood cell count was 3.9×10^3/μL, hemoglobin 7.1 g/dL, platelet count 114×10^3/μL, serum creatinine 4.51 mg/dL, and albumin 2.3 g/dL. The urinalysis showed many bacteria, hyaline and granular casts, microscopic hematuria, and 50 white blood cells per high-power field. Blood cultures from 2 different venipuncture sites grew *Lactobacillus acidophilus*.

A transthoracic echocardiogram revealed a left ventricular ejection fraction of 0.60 and a membranous ventricular septal defect with a mobile, echodense, vegetative mass 1.1 cm in diameter, moving between the left ventricular outflow tract and the right ventricle. Mild dilation of the aortic root was found, together with mild aortic regurgitation.

Initial antibiotic treatment with empiric vancomycin and piperacillin/tazobactam was switched to penicillin on treatment day 4 for a 6-week course after species

identification of *L. acidophilus*. The microorganism was resistant to vancomycin but susceptible to penicillin, daptomycin, linezolid, gentamicin, erythromycin, and clindamycin. Gentamicin was withheld as an initial therapy because of the patient's kidney injury. The patient denied probiotic intake. He tested negative for human immunodeficiency virus infection by Western blot. However, his dental caries and lack of dental care for the past 4 years might have contributed. Our oral surgeon did extract tooth #15 because of a carious lesion but found no acute infection or abscess formation.

The patient remained in heart failure, with a brain natriuretic peptide value of 16,400 pg/mL. A chest radiograph, however, showed no signs of pulmonary vascular congestion—only a left pleural effusion, probably related to a low albumin level, a superimposed acute kidney injury, and proteinuria.

The patient was discharged from the hospital on treatment day 17 after stabilization, but he was readmitted 8 days later (treatment day 25) with a diffuse rash covering his entire body and worsening dyspnea. The rash was maculopapular in nature and tended to bleed easily upon touch. Given his elevation in alanine transaminase level to 698 from 23 U/L, his new medications, and his mild eosinophilia at 2.1%, there was a high suspicion of drug reaction—that the eosinophilia and systemic symptoms had been caused by penicillin. Penicillin was replaced with daptomycin on treatment day 25. Blood cultures during hospitalization were negative.

During this 2nd hospitalization, transesophageal echocardiography was performed because of his worsening dyspnea, peripheral pitting edema, cardiomegaly on chest radiography, and history of endocarditis. That echocardiogram showed a ruptured sinus of Valsalva aneurysm (SVA) with fistula formation from the right coronary sinus to the right ventricular outflow tract (RVOT), a healed vegetation prolapsing into the SVA with each beat, and a 6-mm piece of linear tissue in the RVOT attached to the interventricular septum. Right-sided heart catheterization showed elevated systolic pulmonary artery pressure (58 mmHg) and elevated capillary wedge pressure (26 mmHg), along with a Qp/Qs ratio of 2.08 that suggested a large cardiac shunt at the level of the right ventricle. Because of his critical condition, he was kept on intravenous daptomycin and underwent diuresis and optimization of his clinical condition. Given the tendency of his skin to bleed, he was judged to be at high risk for surgical bleeding and was therefore discharged from the hospital on treatment day 33 by the primary medical team, on a planned total 6-week course of antibiotic agents to end on treatment day 42. Follow-up with our infectious disease and cardiothoracic surgery departments was scheduled.

He was readmitted on day 55 after the initial hospitalization (he did not attend clinic appointments) for decompensated heart failure. He had finished his required 6-week course of antibiotics. During medical optimization for surgery, the patient had 6 teeth removed and was started on a 7-day course of clindamycin for dental abscess, to be completed before surgery. A preoperative computed tomographic scan was ordered to evaluate the cardiac and coronary anatomy in relation to the cardiac fistula (Fig. 1). After the intravenous administration of iodinated contrast medium, the cardiac computed tomogram was electrocardiographically gated with prospective triggering for image acquisition and reconstruction, which was performed on a fast dual-source computed tomographic scanner; 3-dimensional post-processing was performed on a dedicated workstation (Fig. 2).

Findings at surgery included a large 1.5-cm right SVA fistula into the RVOT, with destruction of the aortic valve anulus and part of the corresponding aortic valve cusp. No ventricular septal defect was found intraoperatively, although one had been noted on prior echocardiograms. The operation consisted of repair of the sinus of Valsalva fistula with a bovine pericardial patch, along with an aortic valve replacement with a 27-mm porcine valve. The patient progressed well after surgery and was started on daptomycin because of his history of endocarditis and tooth abscess but had an allergic reaction to this antibiotic. He was switched to clindamycin and was maintained on that antibiotic treatment for 6 weeks after hospital discharge, because of gram-positive bacteria found on gram stain of the excised aortic valve (no species identification could be obtained because of decalcified tissue block). A follow-up echocardiogram

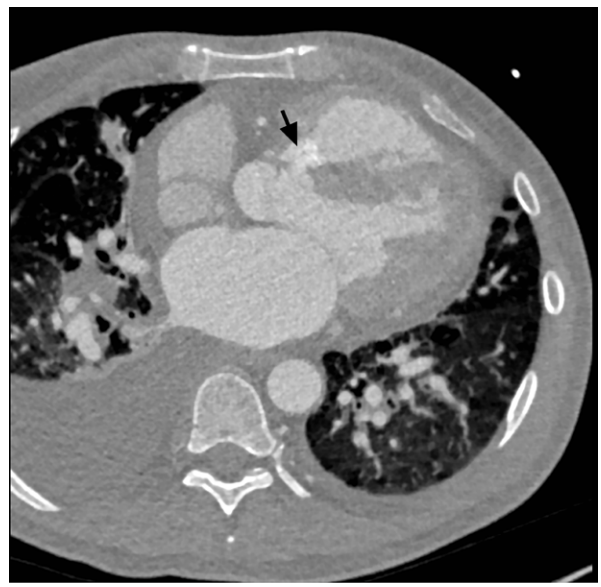

Fig. 1 *Electrocardiographically gated computed tomogram with iodinated contrast medium shows an intracardiac fistula (arrow) from the right sinus of Valsalva to the right ventricular outflow tract.*

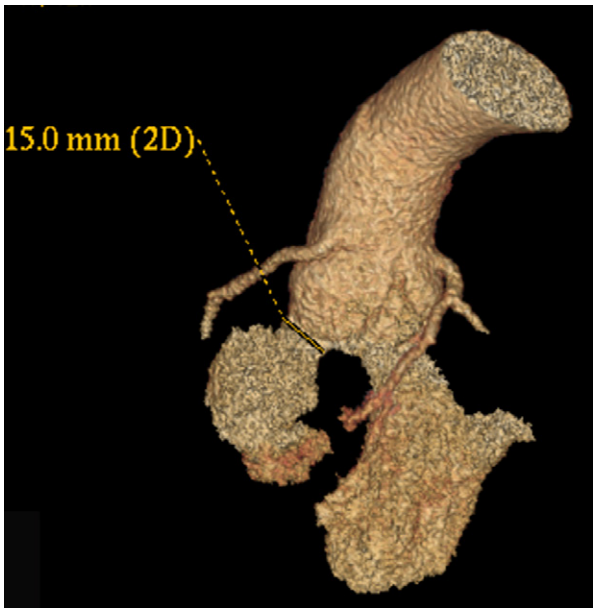

Fig. 2 *Computed tomogram (3-dimensional reconstruction) shows a fistula 15 mm in diameter (dotted line) between the right sinus of Valsalva aneurysm and the right ventricular outflow tract. The failure of left ventricular filling indicates severe aortic regurgitation.*

showed normal heart function and solid placement of the porcine aortic valve, with no regurgitation or paravalvular leak. The patient's renal function returned to normal. As of March 2016, he had completed his antibiotic therapy, had had no further hospitalizations, and reported his return to normal activity.

Discussion

Lactobacillus causes 0.05% to 0.4% of all endocarditis cases.[4] Classically, *Lactobacillus* endocarditis has been linked to structural heart disease, invasive procedures, prosthetic valves, heart transplantation, and dental infections or procedures. *Lactobacillus* bacteremia and endocarditis are effectively treated via the synergistic interaction of penicillin and gentamicin.

The incidence of sinus of Valsalva aneurysms ranges from 0.1% to 3.5% of all congenital heart defects. Such aneurysms account for only 0.14% of all open-heart surgical procedures.[5] Sinus of Valsalva aneurysms mostly present in the right coronary (75%–90%), followed by the noncoronary (10%–25%), with the remainder occurring in the left coronary sinus.[6] These aneurysms are typically congenital and are associated with ventricular septal defects (40% of patients) and aortic regurgitation (30% of patients).[6-8] Acquired SVAs are also associated with bacterial endocarditis and syphilis.[5] The major complication of a SVA is rupture into the right heart chambers, or rarely into the left chambers. Unruptured lesions can remain silent for several years, usually rupturing during the 3rd or 4th decade of life. A ruptured aneurysm typically leads to aortocardiac shunting, aortic regurgitation, and progressively worsening heart failure.[8]

Symptoms associated with rupture are shortness of breath, chest pain, and fatigue.[7] Ruptures can be small at first, with progressive enlargement. The patient might be unaware of his or her condition, or note only progressive dyspnea. Symptoms can be precipitated by physical stress. In most patients, a continuous murmur, generally heard best along the left sternal border from the 2nd to the 4th left intercostal space, are clearly audible.[5]

The consequences of rupture depend on the SVA's size and rapidity of the process leading to rupture. In one third of patients, left-to-right shunting immediately after the rupture of an aneurysm—through a fistulous tract into the right side of the heart—produced acute dyspnea and chest pain. However, half of the patients noted dyspnea, fatigue, chest pain, and peripheral edema that gradually worsened over several months or even years after rupture.[8]

Clinical diagnosis of a ruptured SVA can be difficult, because the SVA can cause continuous murmurs when it ruptures into the right side of the heart. Diastolic accentuation of this murmur is an important sign that differentiates a ruptured SVA from a patent ductus arteriosus. Low diastolic blood pressure is another clinical finding. Clinical diagnosis can easily be confirmed by means of echocardiography. Two-dimensional echocardiography with color-flow Doppler provides an accurate, noninvasive means of revealing both the aneurysm itself and the left-to-right shunting after rupture.[8]

Surgical treatment consists of closure of the fistulous tract with autologous or bovine pericardium. With or without aortic valve replacement or repair, it remains the mainstay of therapy. Our patient continues to do well after the repair of his aorto-RVOT fistula.

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