Cases of Note features peer-reviewed case reports and case series that document clinically relevant findings from critical and high acuity care environments. Cases that illuminate a clinical diagnosis or a management issue in the treatment of critically and acutely ill patients and include discussion of the patient's experience with the illness or intervention are encouraged. Proposals for future Cases of Note articles may be e-mailed to ajcc@aacn.org.

Acute Heart Failure Due to Transient Left Ventricular Dyssynchrony: Case Study

By Matthieu Jourdain, MD, Jean Jacques Bauchart, MD, Jean Luc Auffray, MD, Thierry H. LeJemtel, MD, Philippe Asseman, MD, and Pierre Vladimir Ennezat, MD

Abstract This case study describes an unusual cause of acute heart failure that resolved with early beta-blockade therapy. A 52-year-old woman who had acute heart failure with severe left ventricular systolic dysfunction and left bundle branch block was admitted to a university medical center. Contrast-enhanced magnetic resonance images of the heart did not show any evidence of myocardial infarction or myocarditis. Rate-related left bundle branch block and subsequent left ventricular dyssynchrony resulted in acute systolic dysfunction that resolved with beta-blockade therapy that allowed heart rate control and narrowing of the QRS complex. Of note, the use of inotropic agents would have dramatically worsened the cardiac condition. (*American Journal of Critical Care.* 2010;19:e12-e14)

Acute heart failure remains a challenging diagnostic issue when it is not triggered by myocardial ischemia, critical valvular disease, or excessive loading conditions. This case report describes a patient in whom rate-related left bundle branch block (LBBB) and subsequent left ventricular dyssynchrony induced acute heart failure that completely resolved with the early use of beta-blockade.

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Case Report

A 52-year-old woman who had been receiving immunosuppressive therapy with dasatinib for myeloblastic leukemia type 7 for the past 12 months was hospitalized for acute onset of shortness of breath. She had no past medical history of cardiovascular diseases and no chest pain, palpitations, or syncope. The patient's blood pressure was 100/80 mm Hg and heart rate was 120/min in the emergency department. Body temperature was 38°C and oxygen saturation was 90% on room air. Physical examination revealed orthopnea, distension of the jugular vein, bilateral pulmonary crackles, and a third heart sound in the left ventricle. Sinus tachycardia and complete

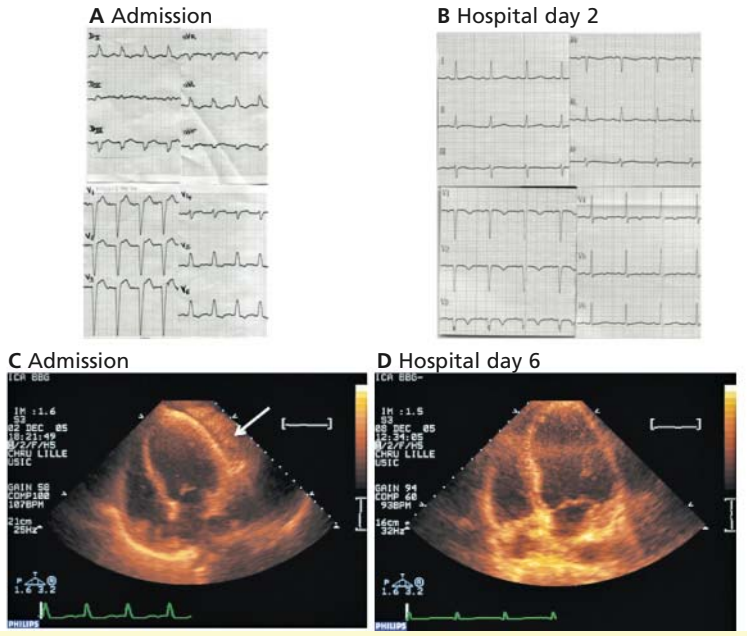

Figure Electrocardiography tracings: left bundle branch block is present (A) at a heart rate of 120/min. QRS width normalized with decrease in heart rate to 85/min (B). C and D, Echocardiograms: Apical 4-chamber view shows a large pleural effusion (arrow). Left atrial and ventricular cavities are nondilated, indicating an acute process for congestive heart failure (C). Apical 4-chamber view at day 6 shows normalization of left ventricular function and the absence of pleural effusion (D).

LBBB were present on the electrocardiogram (see Figure A). Diffuse pulmonary infiltrates and bilateral pleural effusions were noted on the chest radiograph. The bedside transthoracic echocardiogram showed reduced systolic function in the left ventricle with septal oscillatory contractions but without left ventricular enlargement, wall thinning, or segmental akinesis (see Figure C). The asynchrony between the ventricles and within the left ventricle were both measured at 50 ms. The left ventricular ejection fraction was 35%. Mild mitral regurgitation was present. The right ventricular systolic pressure was estimated at 45 mm Hg. The level of troponin I was 1.14 μg/L (normal <0.1 μg/L) without any increase in the level of creatine kinase. The level of brain natriuretic protein was 180 pg/mL (normal <100 pg/mL). Pleural effusion was transudative and thereby suggestive of congestive heart failure. Coronary angiography revealed the absence of coronary artery disease. Venous ultrasonography showed normal findings. Contrast-enhanced magnetic resonance imaging of the heart did not show any evidence for myocardial infarction or myocarditis.

Acute myocardial infarction was excluded as the cause of acute heart failure in this patient.

The patient improved rapidly while receiving a broad-spectrum antibiotic, oxygen, loop diuretic, and vasodilator therapy with resolution of the pulmonary infiltrates on chest radiographs. Body temperature was normal 24 hours after admission, and blood cultures remained negative. On the second day, intravenous administration of atenolol resolved the LBBB. As expected, within 3 hours, beta-blockade produced a progressive decrease in heart rate to 85 beats per minute, which was associated with significant QRS narrowing on the electrocardiogram (see Figure B). The patient could walk in the corridor on the fourth day and was then transferred to the cardiology department while being treated with antibiotics and oral atenolol (25 mg daily). The diuretic and nitrate were discontinued. A repeat echocardiogram obtained 6 days after the onset of symptoms showed normalization of the left ventricular ejection fraction and pattern of contraction (see Figure D). Beta-blockade therapy was stopped 1 month later. Four years later, the patient had not experienced further episodes of heart failure.

Discussion

The presence of LBBB results in early activation of the right ventricular myocardium, slow activation from right to left of the septum, and delayed activation of the lateral and basal walls of the left ventricle. The development of LBBB finally reduces global systolic function of the left ventricle.[1] Experimentally, LBBB induced by right ventricular pacing reduces myocardial blood flow in the septum when compared with other walls of the left ventricle. Myocardial perfusion defects are present in patients with LBBB and normal coronary arteries.[2,3] In addition, the development of LBBB delays contraction in the lateral and basal walls of the left ventricle until early diastole and thereby reduces the left ventricular filling period.[4]

The transient LBBB in this patient was clearly associated with an abnormal systolic motion of the septum and a reduction in global systolic function of the left ventricle that was responsible for the syndrome of acute heart failure. Although LBBB appears in patients with underlying heart disease (see Table), as many as 12% of patients with LBBB have no

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demonstrable heart disease.[5] The presence of an LBBB can obscure the diagnosis of acute ST-T elevation myocardial infarction.[6] However, the modest elevation in the level of troponin I, the lack of akinesis of wall segments, the normal findings on coronary angiography, and the absence of delayed contrast enhancement by magnetic resonance imaging allowed the exclusion of an acute myocardial infarction as the cause of acute heart failure in this patient.

Fulminant and acute forms of viral myocarditis are well recognized causes of acute heart failure that are definitively diagnosed by means of right ventricular endomyocardial biopsy. However, considering the absence of suggestive findings on contrast-enhanced magnetic resonance images, the rapid symptomatic improvement of this patient, and the poor sensitivity of myocardial biopsy, it was believed that the risk of biopsy outweighed its benefit.

Dasatinib, an oral inhibitor of multiple tyrosine kinases, might be involved in the pathogenesis of acute systolic dysfunction of the left ventricle. No other reports, however, have been published that suggest that dasatinib is a likely cause of this problem. In addition, the molecules were tested in isolated rat heart mitochondria. Of the 4 kinase inhibitors (imatinib, dasatinib, sunitinib, and sorafenib), only sorafenib directly impairs mitochondrial function at clinically relevant concentrations.[7] Interestingly, fluid retention including exudative pleural effusions related to an immune-mediated mechanism is a significant side effect of dasatinib.[8]

The hospital course of our patient showed that rate-related left ventricular dyssynchrony resulted in acute heart failure that resolved completely when the heart rate was slowed down after beta-blockade. Importantly, beta-blockade was introduced after administration of a diuretic and nitrate relieved signs of congestive heart failure. The beta-blocker was used specifically to improve systolic function of the left ventricle through reversal of dyssynchrony. The mechanisms that underlie intraventricular conduction delays in this patient remain uncertain. An infectious process (of unknown origin in this case) triggering excessive tachycardia and degenerative lesions in the conduction system both may be suggested.

The use of inotropic agents in this patient would have dramatically worsened the cardiac condition by increasing the heart rate and thus exacerbating the rate-related LBBB. In addition, this case supports the early use of bedside echocardiography in the management of acute heart failure.[9,10] Prompt understanding of the mechanisms that led to the acute condition such as severe cardiac valve dysfunction, right ventricular dysfunction, left ventricular obstruction, or uncoordinated contraction is mandatory to initiate therapy tailored for this acute condition.

Table
Causes of left bundle branch block

Coronary artery disease
Valve disease
Dilated cardiomyopathy
Infiltrative cardiomyopathy
Hypertensive cardiomyopathy
Congenital heart disease
Degenerative conduction system disease (Lenegre and Lev)
Myocarditis, infective endocarditis
Heart trauma/surgery
Hyperkalemia
Myxedema
Systemic sclerosis

FINANCIAL DISCLOSURES
None reported.

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