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.

ACUTE HEART FAILURE DUE TO TRANSIENT LEFT VENTRICULAR DYSSYNCHRONY: CASE STUDY

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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)

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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

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

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. Experimental use of 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. 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.

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...
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. Interestingly, fluid retention including exudative pleural effusions related to the presence of an LBBB demonstrated heart disease. The risk of biopsy outweighed its benefit. 

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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 nitrates relieved signs of congestive heart failure. The beta-blocker was used specifically to improve systolic function of the left ventricle through reversal of dysynchrony. 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. 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.

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