Case report

Adenosine Stress Induced Left Bundle Branch Block During Technetium-99m Tetrofosmin Myocardial Perfusion Imaging

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Abstract

The occurrence of left bundle branch block (LBBB) in electrocardiogram during exercise testing is a relatively rare finding. The incidence of LBBB during exercise testing ranges from 0.5% to 1.1%. The mechanism of exercise-induced LBBB (EI-LBBB) is poorly understood, but ischemia is a proposed etiology. Stress myocardial perfusion imaging (MPI) can be useful in patients with EI-LBBB to rule out coronary artery disease. Adenosine vasodilator stress is the preferred mode of stress in patients with LBBB for performing stress-MPI. Here we present an interesting case of adenosine-induced LBBB during stress-MPI in a 67-year-old female patient with normal coronary angiography.

Keywords: Adenosine-induced left bundle branch block, left bundle branch block, myocardial perfusion imaging, tetrofosmin

Introduction

Exercise-induced left bundle branch block (EI-LBBB) has been reported to occur in approximately 0.5–1.1% of all patients undergoing exercise testing. [1] By definition, EI-LBBB cannot be diagnosed on a resting electrocardiogram (ECG) and may not be apparent at low work levels on a stress test. The precise causative mechanism for EI-LBBB remains unclear, but it may be a reflection of underlying myocardial dysfunction, structural heart disease or compromised coronary circulation. Several authors have attributed EI-LBBB to functional alterations of the conduction system mediated by autonomic influences. As of our knowledge, there are very few reported cases on the scintigraphic pattern of myocardial perfusion in the setting of EI-LBBB. This

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report concerns a 67-year-old female with EI-LBBB, who developed LBBB during adenosine infusion for stress myocardial perfusion imaging (MPI) study.

Case Report

A 67-year-old female patient presented with complaints of intermittent atypical chest pain for 1 year. She underwent treadmill testing (TMT) using Bruce protocol. During TMT, her baseline heart rate (HR) was 78/min and blood pressure (BP) of 130/70 mmHg. She developed LBBB with QRS complex width of 160 ms in Stage I (2nd min) of exercise at the HR of 98/min. She was asymptomatic during exercise and recovery phases. LBBB reverted to normal ECG at 3:40 min into recovery phase at HR of 84/min. She was referred for adenosine stress-MPI as her TMT was not adequate. She was administered adenosine intravenously at the rate of 140 μg/kg/min. Her baseline HR and BP was 75/min and 130/70 mmHg

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How to cite this article: Jayanthi MR, Sasikumar A, Gorla AR, Sood A, Bhattacharya A, Mittal BR. Adenosine stress induced left bundle branch block during technetium-99m tetrofosmin myocardial perfusion imaging. World J Nucl Med 2017;16:78-80.



Figure 1: (a) Baseline electrocardiogram before adenosine infusion showing normal sinus rhythm with heart rate of 78/min. (b) Electrocardiogram at the end of 2nd min of adenosine infusion showing heart rate of 98/min and wide QRS interval of 200 ms. (c) Electrocardiogram reverts to normal at 2:50 min of the recovery phase with heart rate of 90/min

respectively, and baseline ECG was normal [Figure 1a]. She developed LBBB with a QRS width of 200 ms at the end of 2nd min of adenosine infusion at the HR of 93/min [Figure 1b]. 7 mCi of technetium-99m (Tc-99m) tetrofosmin was injected intravenously at a 3rd min of infusion and adenosine infusion was continued for 3 min after radiotracer injection. Patient's ECG reverted back to normal at 2:50 min into recovery phase when the HR was 90/min [Figure 1c]. Poststress gated single-photon emission computed tomography (SPECT) images were acquired 45 min later under gamma camera. Four hours later, resting gated SPECT images were acquired after the second injection of 21 mCi of Tc-99m tetrofosmin. Analysis of stress and rest images revealed a nonreversible perfusion defect of mild severity in the apical anteroseptum with normal thickening and contractility in gated images. Considering adenosine-induced LBBB, the findings of the stress-MPI were interpreted as likely due to LBBB-induced artefact [Figure 2]. Invasive coronary angiography performed 5 days after scintigraphy revealed normal coronaries.

Discussion

It is well-known that interpretation of myocardial perfusion SPECT images in patients with persistent LBBB is inaccurate in the presence of fixed or reversible perfusion defects in the septal or anteroseptal regions, even with normal blood flow through the left anterior descending (LAD) artery.^[2-4] In previous studies, septal, or anteroseptal perfusion defects were estimated to be observed in approximately 75% of patients with LBBB, although significant LAD stenosis was detected only in 39%.^[5-7] But since the LBBB, in this case, was induced by adenosine stress and was spontaneously terminated at

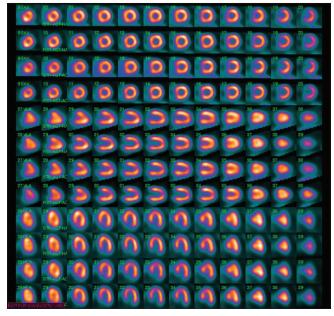


Figure 2: Stress and rest images reveal a nonreversible perfusion defect of mild severity in the apical anteroseptum

rest, scintigraphic findings of nonreversible perfusion defect in anteroseptal region with no associated abnormalities in gated SPECT is most likely the artefact due to LBBB. The prognostic significance of EI-LBBB is poorly understood with the general consensus in the literature being the prognosis of EI-LBBB is good if there is no underlying structural heart disease. [8,9] It has been shown that the onset of EI-LBBB at an HR of 120–125/min or lower correlated strongly with the presence of occlusive coronary artery disease (CAD), compared to patients who develop EI-LBBB at an HR of 120–125/min or higher who show normal coronary arteriograms and have a better prognosis. [8]

Normally adenosine vasodilator stress is the preferred mode of stress in patients with LBBB, but paradoxically our patient developed LBBB during adenosine infusion at an HR of 90/ min. The stress-MPI was useful in ruling out CAD in this patient which was later confirmed on coronary angiography.

Conclusion

This case highlights the facts that even adenosine infusion can induce LBBB although rarely, and the stress-MPI is a useful investigation to rule out the significant CAD as the incriminating cause behind EI-LBBB.

Financial support and sponsorship

Conflicts of interest

There are no conflicts of interest.

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