INTERMITTENT ANTERIOR DIVISIONAL BLOCK AND FAR ADVANCED RIGHT BUNDLE BRANCH BLOCK INDUCED BY VASOSPASM DURING EXERCISE TESTING

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A patient is reported in whom exercise induced reversible ischemic left anterior fascicular block and far advanced right bundle branch block. Master's two step exercise test for pre-operative check-up revealed significant ST elevation in leads V1—5, negative U waves in leads V3—5 and fascicular blocks with retrosternal anginal chest pain. Long acting nitrate and nicorandil relieved the fascicular blocks. 

(Jpn Circ J 1992; 56: 1022—1024)

DIVISIONAL blocks (hemiblocks or fascicular blocks) of the left bundle branch induced by exercise are fairly rare phenomena. Only a few cases of isolated, transient anterior and posterior divisional blocks have been reported. We describe here a patient with reversible divisional block caused by transmural ischemia during an exercise test.

CASE REPORT

A 67-year-old man was referred to our department for a preoperative checkup for a sigmoid colon tumor (Borrmann type II, well differentiated adenocarcinoma). A physical examination of the heart and lungs was normal. The resting electrocardiogram (ECG, Fig. 1) showed sinus rhythm with a heart rate of 55 beats/min, and a complete right bundle branch block with a QRS of 0.12 sec and a mean frontal QRS axis (AQRS) of −20°. A minor degree of anterior divisional block may also have been present. There were no significant ST-T changes, and the U waves were positive in leads V3—5. The patient complained of typical retrosternal chest pain during Master’s single two step exercise test. Immediately after the exercise test his heart rate increased from 55 to 80 beats/min and his ECG (Fig. 2) revealed deeper S waves in leads II, III, and aVF, suggesting a marked left axis deviation with an AQRS of −85°, i.e. a marked anterior divisional block. In addition, the QRS amplitude in leads V1—4 increased significantly and decreased in leads V5, 6. The QRS pattern changed from rsR’ before exercise to a wide R after exercise in lead V1 and from RS to qR, respectively, in leads V2, 3.

After exercise, the QRS width was prolonged to 0.16 sec and ST segment elevated in leads aVL and V1—5 with an upward convex configuration. The ST segment was depressed in leads II, III, and aVF. U waves

Key words:
Ischemia induced bundle branch block
Vasospastic angina pectoris
Prominent anterior force

(Received September 26, 1991; accepted March 2, 1992)
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were inverted in leads V3—5. Three minutes after exercise the heart rate decreased from 80 to 63 beats/min but the abnormalities persisted and ST segments showed further elevation with T waves becoming more peaked in V4, 5 (Fig. 3). Five minutes after exercise, ECG findings returned almost completely to those before exercise (Fig. 4). Chest x-rays and two-dimensional echocardiograms were within normal limits. Complete blood counts, blood chemistry, and urinalysis were negative. Because an exercise-induced vasospasm of the left anterior descending coronary artery was suspected, 30 mg of diltiazem, 20 mg of a long acting nitrate and 5 mg of nicorandil tid were prescribed.

Ten days after antianginal therapy a treadmill exercise test was performed using the standard Bruce protocol. The test was terminated after 6 min (Stage II) because of leg fatigue, without inducing chest pain. The heart rate increased from 50 beats/min at rest to 110 beats/min at peak exercise and systolic blood pressure increased from 100 mmHg to 155 mmHg. However, ECG no longer showed ST-T changes, negative U waves, or marked left axis deviation.

**DISCUSSION**

Two major pathogenetic mechanisms for anterior divisional blocks have been suggested; one is atherosclerotic coronary artery disease and the other is aging of the cardiac fibroskeleton. According to Wayne et al. bundle branch blocks induced by exercise have been found in 0.39 per cent of tests performed over a 5 year period. However, they did not notice any divisional blocks. So far, to our knowledge, only six cases of anterior divisional block induced by exercise have been reported. Two mechanisms of exercise-induced bundle branch block have been postulated, i.e. tachycardia-dependent and ischemia-induced. The former is often considered to have a benign course. However, Wayne et al. reported that exercise induced bundle branch block is almost always associated with cardiovascular dis-
ease, most commonly coronary artery disease. Patients with anterior divisional block induced by exercise showed a proximal left anterior descending lesion. Chandrashekar et al. noted that glycercyl trinitrate relieved the block which did not appear after successful percutaneous transluminal coronary angioplasty. Tachycardia during exercise may not only cause or aggravate ischemia but may allow recovery time in a damaged conduction system? The present case showed anterior divisional block, a longer QRS duration with significantly larger R waves in leads V1—4, ST segment elevation, and negative U waves in leads V2—5 after exercise.

Transient U wave inversion in left precordial leads during the exercise test suggested significant coronary artery disease of the proximal left anterior descending coronary artery or multivessel disease. U waves may also be transiently inverted during an attack of the variant form of angina. However, the increased duration of QRS width and amplitude of QRS in the right and mid-precordial leads after Master's single two step exercise test cannot be ascribed solely to an anterior divisional block. AQRS width prolongation of 0.04 sec clearly exceeds the average increment in divisional blocks. Prominent anterior forces have been found in patients with anterior divisional block. Nakaya et al. postulated that if conduction through the septum division of the left bundle is slowed or blocked, the impulse must reach the regions innervated by the septal fascicle through the anterior and posterior divisions and change the course of propagation in the horizontal plane. Therefore, we assumed that in our patient the anterior QRS force in the horizontal plane and the marked left axis deviation in the frontal plane were caused by exercise-induced ischemia of three divisions of the bundle branch system (right bundle, anterior and septal divisions) and that the ischemia was probably due to a vasospasm in the proximal portion of the left anterior descending coronary artery. Coronary angiography including an ergonovine test, was not performed because of the malignant underlying disease.

REFERENCES


Japanese Circulation Journal Vol.56, October 1992