Myocardial bridge (MB) is a segmental tunnel in the left anterior descending artery (LAD) beneath the myocardium which can cause several significant cardiac disorders such as stable angina, acute coronary syndrome, and even sudden cardiac death. However, it seldom induces conduction block. Herein, we report a case of severe MB who presented as acute non-ST elevation acute coronary syndrome (NSTE-ACS) along with paroxysmal Mobitz type I atrioventricular block.

**Case Presentation**

A 51-year-old male patient without previous systemic disease except for hepatitis B, presented to the emergency department with crescendo dizziness, suffocating chest pain, and cold sweating for 3 hours after alcohol drinking. The chest pain was partially lessened after rest. The patient recalled that he had had similar experiences while strenuously exercising or drinking alcohol. He had a 10-year smoking history but has quitted for more than 20 years. He denied taking any regular medication. Family history of cardiovascular disease was unremarkable.

**ABSTRACT**

Chest pain complicated with electrocardiographic changes is not an uncommon scenario in emergency departments, which should be examined cautiously. We describe a 51-years-old man with a myocardial bridge of coronary artery presenting with simultaneous Mobitz type I atrioventricular block on electrocardiography. Echocardiography excluded valvular abnormality and systolic/diastolic dysfunction. Coronary angiography confirmed the diagnosis of a myocardial bridge at the middle segment of the left anterior descending artery, involving the most dominant septal perforator branch with marked systolic compression. The patient underwent coronary artery bypass grafting surgery and was followed up uneventfully at the outpatient department with medical treatment of diltiazem and clopidogrel. The present case is being reported to highlight that clinicians should be alert to such a congenital abnormality as a potential cause of repeated myocardial infarction and conduction abnormality.

**KEY WORDS**: Atrioventricular block, electrocardiography, myocardial bridge, myocardial infarction
Upon physical examination, his blood pressure was 126/76 mmHg with a heart rate of 46 beats per minute. Irregular heart rhythm was noted on auscultation. Electrocardiography (ECG) demonstrated a first-degree AV conduction block with paroxysmal Mobitz type I AV block and ST-segment depression in precordial leads [Figure 1]. Chest radiography was noncontributory. Laboratory tests disclosed a serum potassium level of 3.0 mmol/L (normal 3.5–5.1), serum lactate level of 3.5 mmol/L (normal 0.5–2.2). Cardiac biomarker revealed an elevated troponin I level of 1.4 ng/mL (normal below 0.04). Others were unremarkable.

Echocardiography revealed normal LV systolic and diastolic functions without structural abnormality. Coronary angiography was implemented under the impression of NSTE-ACS, which confirmed an MB at the middle segment of the LAD [Figure 2, arrow], involving the dominant septal perforator branch (arrowhead); (b) Coronary angiography in the diastolic phase of the left ventricle disclosing both the patent middle left anterior descending artery (arrow) and dominant septal perforator branch (arrowhead)

Figure 1: (a) Baseline electrocardiography on admission demonstrating the progressive prolongation of the PR interval (arrows) on consecutive beats followed by a blocked P wave, compatible with a Mobitz type I (Wenckebach) atrioventricular block; (b) Repeated electrocardiography after coronary artery bypass grafting surgery revealing the recovery of every PR interval (arrowheads)

Figure 2: (a) Coronary angiography in the systolic phase of the left ventricle showing the myocardial bridging of the middle segment of the left anterior descending artery (arrow) with severe systolic compression involving the dominant septal perforator branch (arrowhead); (b) Coronary angiography in the diastolic phase of the left ventricle disclosing both the patent middle left anterior descending artery (arrow) and dominant septal perforator branch (arrowhead)

Ischemia-related AV block was mostly reported in scenarios of acute inferior wall ischemia mainly owing to RCA occlusion with impaired blood supply of the AV node artery. The AV node artery is the first and longest inferior septal perforating branch of the right (90%) or left (10%) coronary artery. This origin is dependent on coronary arterial dominance, either by the right coronary artery or left circumflex artery. There is no definite intracoronary atherosclerotic lesion in the RCA or the LCX in our patient, which excluded the assumption of inferior wall ischemia. Blood supply to the subnodal conduction system is mainly through the penetrating branches of the LAD. While Mobitz type I (Wenckebach) AV block occurring at the nodal site is decremental, Mobitz type II AV block takes place in the His-Purkinje system which demonstrates a fixed PR interval. The right ventricular lary of LAD typically supplies the subnodal conduction system, and hence, ischemia here should theoretically induce Mobitz type II AV block. Interestingly, Kenneth et al. proposed that both type I and II blocks may occur with either functional or structural abnormality of the conduction system, which may reflect either nodal or subnodal conduction disturbance according to the electrophysiologic study. Moreover, in patients with symptomatic second-degree AV block investigated by the electrophysiologic study, 25% of patients exhibited the intra-His Wenckebach phenomenon. Such evidence partly explained the Wenckebach phenomenon induced by severe MB with the hindrance of blood supply to the subnodal system in our patient.

Discussion

We have presented a case with severe MB complicating with Mobitz type I AV block and NSTE-ACS, which was successfully reversed by CABG. The incidence of MB has been reported to vary between 15% and 85% and in angiographic series it ranges from 0.5% to 2.5%. The LAD is by far the most affected vessel. The diagnosis could be established on the change in the vessel diameter between the systole and diastole phases within the bridged segment by coronary angiography. Most authors consider MB as a congenital anatomic variant but it could induce several clinical events, such as acute coronary syndrome, coronary spasm, myocardial stunning, syncope, arrhythmia, or even sudden cardiac death.
For symptomatic patients with MB, treatment modalities consist of pharmacological therapy, percutaneous coronary intervention with stenting, and surgical intervention. Antiplatelet agents, beta blockers, and calcium channel blockers, instead of nitrates could be considered for medical treatment.\(^1\) Beta blocker or calcium channel blocker was not used in our patient owing to the existing AV block. Multiple studies have demonstrated high rates of target lesion revascularization and complication of stent fracture and coronary perforation by percutaneous coronary intervention and stenting.\(^9,10\) Surgical options for myocardial bridging include surgical myotomy and CABG. CABG is preferred to myotomy in cases of extensive (>25 mm) or deep (>5 mm) bridged segments or when the involved segment fails to decompress completely in the diastole phase.\(^1\)

Treatment of patients with MB, acute coronary syndrome, and symptomatic bradycardia remains controversial. Previous reports suggested pacemaker implantation in patients with MB and solely AV block may be feasible.\(^1,11,12\) However, for those with MB with virtually myocardial ischemia, restoration of blood supply may be essential.\(^3\) Our patient had symptoms of both AV block and acute coronary syndrome. CABG was chosen based on the considerations of the repeated symptoms, the degree of systolic compression, and the range and size of the involved coronary vessel. By CABG, both angina and the AV block were relieved, which appears a successful intervention in this context.

**Conclusion**

The present case should remind clinicians that MB should also be taken into differential diagnosis in patients with paroxysmal atrioventricular block and acute coronary syndrome, especially among those with low cardiovascular risk.

**Declaration of patient consent**

The authors certify that appropriate patient consent was obtained.

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**Conflicts of interest**

There are no conflicts of interest.

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