INTRODUCTION

Myocardial bridging (MB) is a congenital anomaly characterized by an intramyocardial route of a coronary artery. The coronary arteries tunnel into the myocardium rather than resting on top of it [1]. MB can occur in almost all epicardial arteries; however, the incidence is highest in the left anterior descending artery (LAD) [1]. MB patients are often asymptomatic, and diagnosis is often made from incidental findings of diagnostic imaging, angiography, or autopsy. This congenital anomaly has a good prognosis, with a reported 5-year survival greater than 97% [2]. However, prognosis can vary from benign to fatal. In symptomatic patients, MB can present with a variety of clinical manifestations ranging from myocardial ischemia, myocardial infarction, cardiac arrhythmia, to sudden cardiac death [2].

CASE REPORT

A 56-year-old Indian woman with a history of diabetes mellitus presented with atypical chest pain. Her electrocardiogram (ECG), chest radiograph, cardiac enzymes and other blood parameters were unremarkable. Using the one-day, stress-rest gated Tc-99m tetrofosmin protocol, myocardial perfusion imaging (MPI) was performed for cardiac assessment. Pharmacological stress was applied using dipyridamole infused over 4 minutes at 140 µg/kg/min. There were no symptoms or ECG changes to suggest dipyridamole-induced myocardial ischemia. The myocardial perfusion single photon emission computed tomography (SPECT) images showed mild reversible reduced tracer uptake in the apical anterior segment of the left ventricle (Fig. 1). Other myocardial segments showed no demonstrable perfusion defect. The left ventricular cavity was not dilated.

Subsequently, the patient underwent cardiac CT, which showed a 2-cm-long superficial MB located at the mid segment of the LAD (Figs. 2 and 3). The lumen appeared patent without stenosis. The left circumflex and right coronary arteries were otherwise normal. The coronary calcium score (Agatston...
DISCUSSION

In patients with MB, the cardiac muscle applies external pressure across the bridged segment during systole, constraining the artery and causing ischemia [3]. The pathophysiology of MB involves a complex interplay between extrinsic and intrinsic factors, including epicardial coronary artery compression, sympathetic drive that prolongs MB contraction, phasic spasm of the coronary artery, an interval interactions among aortic pressure,
diastolic flow, heart rate, and transmural perfusion gradient [1,3]. Additionally, impaired diastolic flow can cause depressurization of the septal branches within the MB, resulting in an intrabridge ischemia, also known as the “branch steal” phenomenon [1,3].

Presently, there are no standardized diagnostic criteria or a gold standard for evaluation of MB [1]. The presence of MB is often noted on anatomical imaging, such as CT scan, which allows characterization of its location, length, and depth; arterial constriction and assessment of atherosclerosis [1,4]. However, the functional significance of MB is often determined by SPECT or positron emission tomography (PET) MPI. Based on the study by Gawor et al. [5] of 42 patients without coronary artery disease, the occurrence of myocardial perfusion defect in bridging patients is linked to the degree of coronary artery narrowing during systole, and patients usually experienced no symptoms if the coronary artery constriction was less than 50% and were symptomatic if constriction was greater than 70% [5].

Hybrid cardiac imaging with SPECT/CT [6] and PET/CT [4] allows concurrent evaluation of the coronary vascular anatomy and myocardial blood flow. In a study by Monroy-Gonzalez et al. [4] involving 131 patients who underwent both $^{13}$N-ammonia PET and cardiac CT, there was no significant correlation between quantitative perfusion analysis and the anatomical features of MB, such as its length and depth, displayed on CT [4]. Furthermore, myocardial perfusion reserve (MPR), a marker of microvascular dysfunction quantified by PET, was found to be low in patients with MB of the LAD [4]. MPR measured by PET can be used as a tool to identify MB patients who are at increased risk of cardiac event [4].

In practice, a combination of both functional and anatomical imaging can guide the clinician in treatment planning and monitoring of MB [6]. In a case report by Lim et al. [7], a 43-year-old man with established MB refractory to medical treatment underwent a combination of cardiac CT with MPI. The study demonstrated MB in the mid LAD and reversible perfusion defect in the anteroseptal wall [7]. The MB was surgically corrected with supraarterial myotomy, and subsequent imaging showed successful unroofing of the MB segment with a normal result on myocardial perfusion study [7].

Treatment is often unnecessary in the asymptomatic MB patient. For symptomatic patients, medical treatment with beta blockers causes reduction in compression by the muscular band, slows the heart rate, and prolongs the diastolic period [1]. Invasive therapy such as percutaneous coronary intervention and coronary artery bypass graft, are usually reserved for severely symptomatic refractory cases and cases that cannot be controlled with oral medications [1,2]. To date, there are no appropriate guidelines on suitable medical treatment or revascularization techniques for MB. Additionally, it is uncertain if intervention in bridging patients with mild or moderate ischemia achieves better outcomes. Thus, there is uncertainty in optimal patient management.

In conclusion, although uncommon, MB should be included in the differential diagnosis for chest pain. A combination of anatomical and functional imaging in patients with MB provides valuable hemodynamic information on the bridging segment, which guides the clinician’s determination of a treatment plan and aids in monitoring of therapy response.

Conflicts of Interest

The authors declare that they have no conflict of interest.

REFERENCES