

Silent Myocardial Infarction Presented with Homonymous Hemianopia: A Rare Case Study

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ARTICLEINFO	ABSTRACT
Article type: Case report	Silent myocardial infarction is a little-known phenomenon, the mechanisms of which have still remained unclear. Herein, we presented the case of a middle-aged man suffering from silent myocardial infarction who presented with homonymous hemianopia and no other major cardiovascular risk factors, except for stage 1 hypertension.
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Introduction

Silent myocardial infarction (MI) is defined as the appearance of pathological Q waves in the electrocardiogram (ECG) without any objective signs of MI or any minimal and atypical symptoms (1, 2). Currently, silent MI is most often diagnosed by the existence of Q wave in a 12-lead ECG and reduction of R wave or abnormalities of ST segment and/or T wave (1, 3). However, the ECG has a very low sensitivity.

The newer imaging techniques, such as myocardial perfusion single photon emission computed tomography and cardiac magnetic resonance, offer better diagnostic capability, particularly test sensitivity. Nonetheless, the early initiation of effective treatment and secondary prevention of silent MIs, such as the application of antiplatelet therapy, are frequently missed. The prevalence of silent MI is less known, compared with that of silent myocardial ischemia (1).

However, the prevalence of silent MI considerably increases with aging in the general population (up to>5% in the elderly subjects) (3). Although silent MI has been known for a long

time, its mechanisms have been not identified yet. The coincidence of this condition with stroke is also still a controversial issue. In the present case report, we presented a patient who was inflicted with silent MI and had brain infarction symptoms simultaneously.

Case Presentation

Our case was a 52-year-old man suffering from weakness, lethargy, and sweating initiated three days before admittision to Shahid Beheshti Hospital, Qom, Iran, in August, 2014. He was admitted when no improvements appeared, and the right lower limb paresthesia and visual field reduction were also developed. Right homonymous hemianopia was confirmed by the physical examination, confrontation visual field exam, and perimetry. Furthermore, heart auscultation showed S4 murmur.

The limb forces were normal, except for the proximal (4-/5) and distal (4+/5) paresis of the

right upper extremity. The relative afferent pupillary defect and central visual impairment

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Figure 1. Electrocardiogram showing Q wave in aVL and QS in V1-V5 leads

were negative. In addition, ophthalmoscopic findings as well as heel to knee and finger to nose tests were normal. The patient did not have aphasia or dysarthria. He had a two-year history of stage 1 hypertension with irregular treatment of using Losartan (25 mg). Furthermore, the blood pressure was 145/80 mmHg, and the regular heart rate was 78 per minute.

However, the patient did not have any other diseases or risk factors, such as diabetes, smoking, family history of cardiovascular diseases, hypercholesterolemia, or history of angina. The biochemical test results (i.e., lactate dehydrogenase=1669 [50-150 U/L], creatine kinase MB=26 [0-4 ng/mL], troponin=9.55 [0-0.01 ng/mL], creatine phosphokinase=173 [25-200 U/L], C-reactive protein=50 [<5 mg/L], and erythrocyte sedimentation rate=49 [26 mm/h]) and other important factors (i.e., complete blood



Figure 3A. Left anterior descending artery: cut-off at proximal with retrograde filling via right system; obtuse marginal artery: cut-off at proximal with poor run-off



Figure 2. Computed tomography scan showing left temporal and occipital lobes ischemia

count, full blood count, blood sugar, triglycerides, high-density lipoprotein, low-density lipoprotein, sodium, potassium, chromium, partial thromboplastin, and partial thromboplastin time) were at normal levels.

The ECG showed Q wave in aVL and QS in V1-V5 leads (Figure 1). Moreover, the transthoracic echocardiography revealed apical left ventricular thrombus, apical akinesia, and 25% left ventricular ejection fraction. The brain computed tomography showed ischemic damages in the occipital and temporal lobes (Figure 2). Additionally, the coronary angiography demonstrated left anterior descending artery and obtuse marginal artery proximal cut-offs as well as some ectasias and plaques in the right coronary artery (figures 3A and 3B). However, the color Doppler sonography was normal with no evidence of atherosclerosis. Finally, the patient was subjected to coronary artery bypass grafting surgery.



Figure 3B. Right coronary artery: some ectasias and plaques throughout vessel

Discussion

Homonymous hemianopia is a type of visual field loss indicating the presence of a lesion involving the visual pathway posterior to the chiasm. Occipital infarction in the territory of the posterior cerebral artery due to embolism (usually from cardiac sources) is the most common cause of this defect (4, 5). Temporal lobe lesions also usually involve upper quadrant visual field and are associated with auditory and memory problems as well as seizures (5).

Some studies have indicated that the risk of stroke is higher in the individuals with unrecognized MI, compared to those with recognized MI (6). Our patient presented with acute neurologic defects, pathologic Q waves, and significant troponin enzyme rising (recent non-ST-elevation MI or recent ST-elevation MI with rapid resolution of ST elevation), suggesting the coincidence of these events. According to the literature, the prevalence of silent MI is higher in the patients with diabetes mellitus than that in others (1, 7).

In a study conducted by Wackers et al., 1,123 patients with type II diabetes were assessed to determine the prevalence and severity of inducible myocardial ischemia. In the mentioned study, it was reported that silent myocardial ischemia occurred in 113 (22%) asymptomatic patients with type II diabetes (7). Diastolic blood pressure is another important risk factor suggested to be associated with higher prevalence of silent MI (3). However, few studies have focused on the prevalence and incidence of silent MI in the patients with hypertension.

In a case report study, the coincidence of silent MI and stroke was reported in a man with major cardiovascular risk factors (e.g., smoking, hypercholesterolemia, family history of cardiovascular diseases, and specially diabetes mellitus) (8). Nevertheless, our patient did not have those risk factors. The low incidence of stroke in the occipital lobe (9) and the rare occurrence of silent MI in the middle-aged men encouraged us to present this case (10).

Furthermore, considering the aforementioned studies (i.e., 1, 3, 7) and given the lack of major risk factors, the coincidence of silent MI and stroke in this patient was noteworthy.

Conclusion

In conclusion, this case report indicated that the existence of only one cardiovascular risk factor (i.e., systolic hypertension stage 1) even in the absence of others, including smoking, hypercholesterolemia, diabetes mellitus, and family history of cardiovascular diseases, would lead to the development of MI or stroke.

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Acknowledgments

None.

Conflict of Interest

The authors declare no conflict of interest.

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