Loeffler's endocarditis and possible coronary spasm secondary to eosinophilic coronary periarteritis with ECG change mimicking acute non-ST-segment elevation myocardial infarction: A case report

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Case report

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Abstract

Background

Loeffler's endocarditis is an inflammatory cardiac condition of hypereosinophilic syndrome which rarely involves coronary artery. When coronary artery is involved, known as eosinophilic coronary periarteritis, the clinical presentation, electrocardiographic changes and troponin level are extremely nonspecific and may mimic acute coronary syndrome. It is very important to make differential diagnosis for ECPA in order to avoid the unnecessary further invasive coronary angiography.

Case presentation

We report a case with chest pain, ST-segment depression in electrocardiogram and increased troponin-I mimicking acute non-ST-segment elevation myocardial infarction. However, quick echocardiography showed endomyocardial thickening with normal regional wall motion, which corresponded to the characteristics of Loeffler's endocarditis. Emergent blood analysis showed marked increase in eosinophils and computed tomography angiography found no significant stenosis of coronary artery. Manifestations of magnetic resonance imaging consisted with findings of echocardiography. Finally, the patient was diagnosed as Loeffler's endocarditis and possible coronary spasm secondary to eosinophilic coronary periarteritis.

Conclusion

This case exhibits the crucial use of quick transthoracic echocardiography and the emergent hematological examination for differential diagnosis in such scenarios as often if electrocardiogram change mimicking myocardial infarction.

Background

Hypereosinophilic syndrome (HES) is a rare heterogeneous group of disorders characterized by unexplained hypereosinophilia and organs involvement in varying degrees. Cardiac involvement, presented in 40%-50% of HES, is classically divided into three phases: eosinophilic infiltration, thrombosis and fibrosis, which can be well detected by transthoracic echocardiography (TTE)[1–3]. Loeffler's endocarditis, one of the main cardiac manifestations complicated with HES, is characterized by endocardial tissue damage from eosinophil toxic effect and restrictive cardiomyopathy. Coronary lesions known as eosinophilic coronary periarteritis (ECPA) have been seldom described[1]. When coronary artery is involved, the clinical presentation, electrocardiographic changes and troponin level are extremely nonspecific and may mimic acute coronary syndrome (ACS)[2]. We report a case with changes of electrocardiogram (ECG) and troponin level mimicking acute non-ST-segment elevation myocardial infarction (NSTMI). Combined with TTE characteristics of Loeffler's endocarditis and marked increase in eosinophil (EO) count, we inferred eosinophilic coronary periarteritis may occur. The preliminary

diagnosis of acute NSTMI was rejected and unnecessary further invasive coronary angiography was avoided.

Case Presentation

A 46-year-old male came to the emergency department with severe ongoing chest pain radiating to back for 2 days. He had a 2-year history of diabetes mellitus. Emergent ECG suggested acute NSTMI with ST-segment depression and biphasic T-wave in leads II, III, aVF and V_3-V_6 (Fig. 1), accompanied with elevated troponin I (TnI, 0.46ug/L). Vital signs and physical exams were normal. The preliminary diagnosis was acute NSTMI, TTE and coronary angiography was proposed.

Before coronary angiography, a quick TTE revealed endomyocardial thickening in left ventricular (LV) apex and mild enlargement of left ventricle and atrium dimensions. Regional wall motion was normal and left ventricular ejection fraction (LVEF) was within normal range (LVEF, 61%) (Fig. 2A, Additional file 1). Furthermore, two-dimentional speckle-tracking echocardiography (STE) showed that LV global longitudinal strain (LS) reduced to -13%, indicating impaired LV function which was covered up by normal LVEF. And the peak LS prominently decreased in apical segments of all wall and mid segment of anterior wall, which corresponded to the involved endocardial region showed by TTE and STE but did not consist with the perfusion area of any coronary artery (Fig. 2B). Thus, Loeffler's endocarditis was diagnosed by TTE, and damaged apical myocardial function with depressed global myocardial contractility was detected by STE. In addition, the emergent blood analysis showed a marked increase in EO count (5.87×10^9/L) and percent (47.5%) as well as total white blood cell count(12.36×10^9/L).

Considering the possible eosinophilic infiltration in coronary artery, we performed computed tomography angiography (CTA) instead of coronary angiography. Coronary CTA showed no significant stenosis of coronary artery. However, cardiac magnetic resonance imaging (cMRI) revealed high signal intensity of thickened LV apical subendocardium on T2 weighed images and late enhancement of the LV apical subendocardium on late enhancement images, which was consistent with the region of the endocardial lesion TTE showed (Fig. 3). The findings of cMRI indicated the change of endomyocardial inflammatory exudate combined with fibrosis. Meanwhile, endomyocardial biopsy and bone marrow puncture were advised but the patient refused. Fluorescence in situ hybridization for the FIP1L1-PDGFRA gene was taken in peripheral blood instead and the negative result suggested a lymphocytic variant of HES, for which corticosteroids remain one of the cornerstones of therapeutic agents.

Combining the above findings, a diagnosis of Loeffler's endocarditis and possible ECPA leading to coronary artery spasm was established. Therefore, the patient was started on prednisone (30 mg/day) and caltrate (600 mg/day) in addition to the previous administration of ramipril, metoprolol, isosorbide dinitrate, glucobay and gliclazide. However, he didn't respond to the medicine treatment and repeated ECGs kept deteriorating in the following days (Fig. 4). The patient refused to take prednisone and requested discharge from the hospital a week later. Unfortunately, the patient died soon after his hospital discharge.

Discussion And Conclusion

HES is a rare group of disorders characterized by persistent and marked peripheral eosinophilia (> 1.5 × 10^9/L for > 6 months) of unknown origin with evidence of organs involvement[2]. Cardiac involvement, one of the most frequently involved organs, is a major cause of morbidity and mortality[2].

Loeffler's endocarditis is one of main cardiac manifestations for which TTE is a valuable diagnostic tool. TTE could reveal typical increased endocardial thickness because of interstitial myocardial edema in early stage. The intermediate thrombotic stage is characterized by thromboembolic events in 4–29% of adult patients with HES [2]. Specific obliterative LV apical mural thrombus can be discovered by TTE and the thrombus may extend to the ventricular outflow tracts, the subvalvular regions or occasionally the atrium[2]. Finally, restrictive diastolic dysfunction can also be detected in late fibrotic stage[2]. Furthermore, LS could reflect true global and regional myocardial contractility while LVEF reflects only global LV volumetric changes. Thus, crucial information for diagnosing Loeffler's endocarditis and true myocardial dysfunction can be well seen in TTE and STE.

Severe eosinophil inflammatory infiltration into coronary artery, known as ECPA, is seldom described and classically diagnosed in post-mortem examination in HES [1, 2]. The cytokines released by eosinophils into the coronary inflammatory lesion might contribute to the coronary artery spasm, which may lead to secondary angina pectoris, acute myocardial infarction (AMI) or even sudden death in ECPA[4].

Patients with ECPA often have similar clinical and ECG presentations as ACS. The change of diffuse ST-segment depression and biphasic T-wave in ECG mimicking acute NSTMI like our patient was inconsistent with non-obstructive stenosis of coronary artery. And the region of apical endocardial lesion showed by STE was not compliant with perfusion area of any coronary artery, which refuted the preliminary diagnosis of acute NSTMI. It is a pity that the patient refused endomyocardial biopsy which remains the gold standard for the diagnosis of cardiac involvement in HES. But considering the obvious increase of EO count, we could make a diagnosis of Loeffler's endocarditis by cMRI on account of the significant correlation between late gadolinium enhancement and cardiac inflammatory infiltration presented by biopsy in HES[5]. And it could reasonably be inferred that the clinical presentation mimicking acute NSTMI in our patient occurred secondary to coronary spasm resulting from ECPA.

Thus, the significant increase of EO count and the detection of Loeffler's endocarditis by quick TTE and regional myocardial dysfunction by STE could provide implicit information for coronary involvement in HES and thus avoided the unnecessary invasive coronary arteriography timely in patients with similar clinical and ECG presentations as ACS.

Corticosteroids remain the first-line therapy for a lymphocytic variant of HES to reduce eosinophil count and counteract inflammation, while a myeloproliferative variant with the FIP1L1-PDGFRA mutation should be given a tyrosine kinase inhibitor of possible effectiveness[1]. Although overall prognosis has improved for HES, cardiac involvement remains a major cause of morbidity and mortality[1]. Unfortunately, our patient gave up the treatment and died soon after discharge. We speculated the

sudden death of our patient may derive from another severe coronary spasm due to ECPA, high atrioventricular block or ventricular arrhythmias.

Abbreviations

HES: hypereosinophilic syndrome; TTE: transthoracic echocardiography; ECPA: eosinophilic coronary periarteritis; ACS: acute coronary syndrome; ECG: electrocardiogram; NSTMI: non-ST-segment elevation myocardial infarction; EO: eosinophil; LV: left ventricular; LVEF: left ventricular ejection fraction; LS: longitudinal strain; CTA: computed tomography angiography; cMRI: cardiac magnetic resonance imaging

Additional File

Additional file 1: Video clip of endomyocardial thickening in LV apex at the apical 4 chamber view on TTE. (AVI 77441 kb)

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Consent was obtained from the relatives of the patient for publication of this case report and any accompanying images.

Availability of data and materials

The datasets generated and analyzed in this study are available from the

corresponding author on request

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

KFX, LM, MCY, MPP, WYH and LGY collected, analyzed and interpreted the data, KFX and MCY performed the operation and drafted the article. MCY, LM and YJ revised written English of the article. All authors have seen and approved the final version of the manuscript being submitted. The article is the authors' original work, hasn't received prior publication and isn't under consideration for publication elsewhere.

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Figures

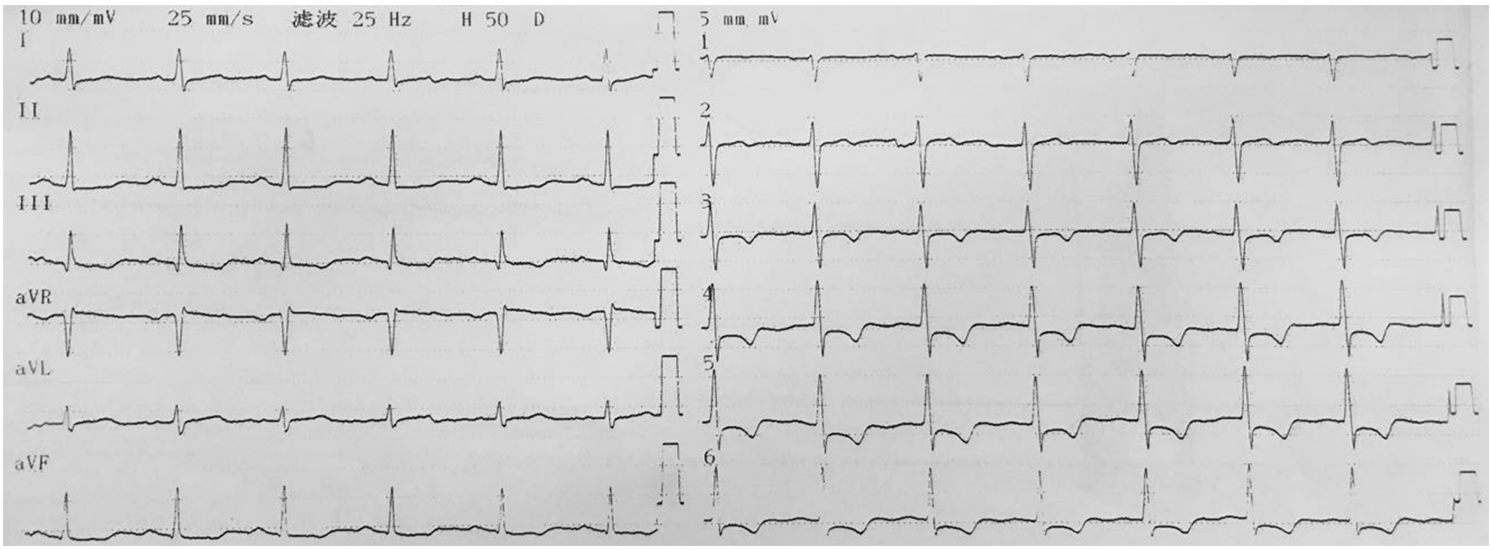

Figure 1

ECG on admission showing ST-segment depression and biphasic T-wave in leads II, III, aVF and V3-V6.

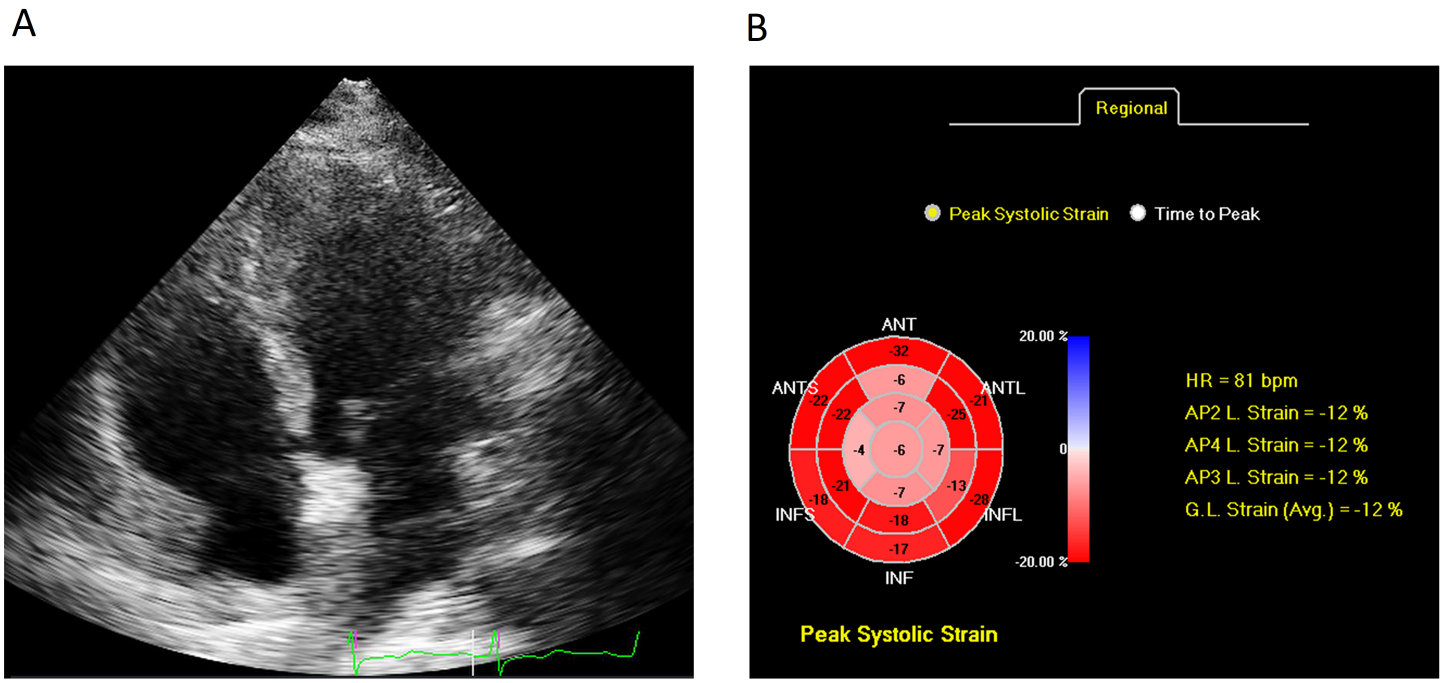

Figure 2

TTE showing endomyocardial thickening in LV apex at the apical 4 chamber view (A). STE showing reduction of LV global LS and the peak LV LS in apical segment of all wall and mid segment of anterior wall (B).

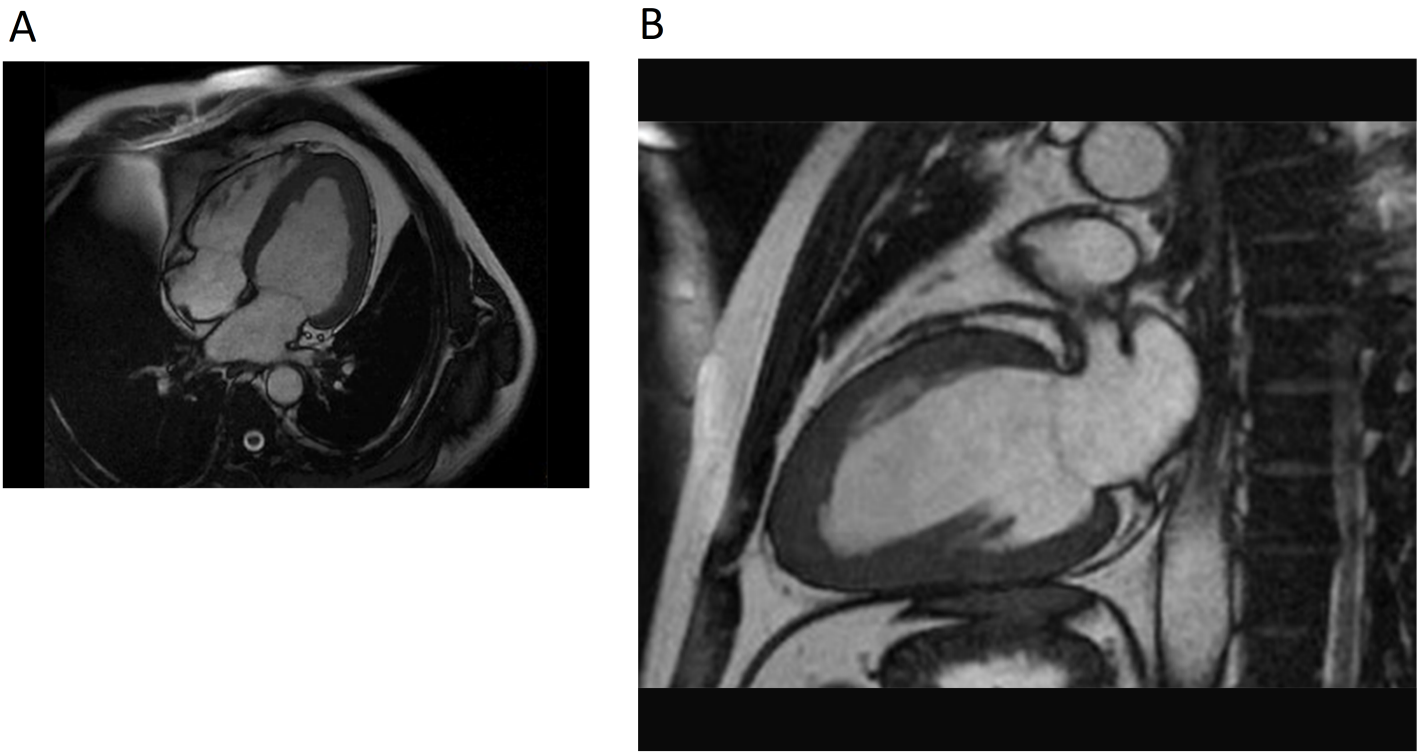

Figure 3

CMRI showing high signal intensity of thickened apical subendocardium on T2 weighed images (A) and late enhancement in the subendocardium of the LV apex on late enhancement images (B).

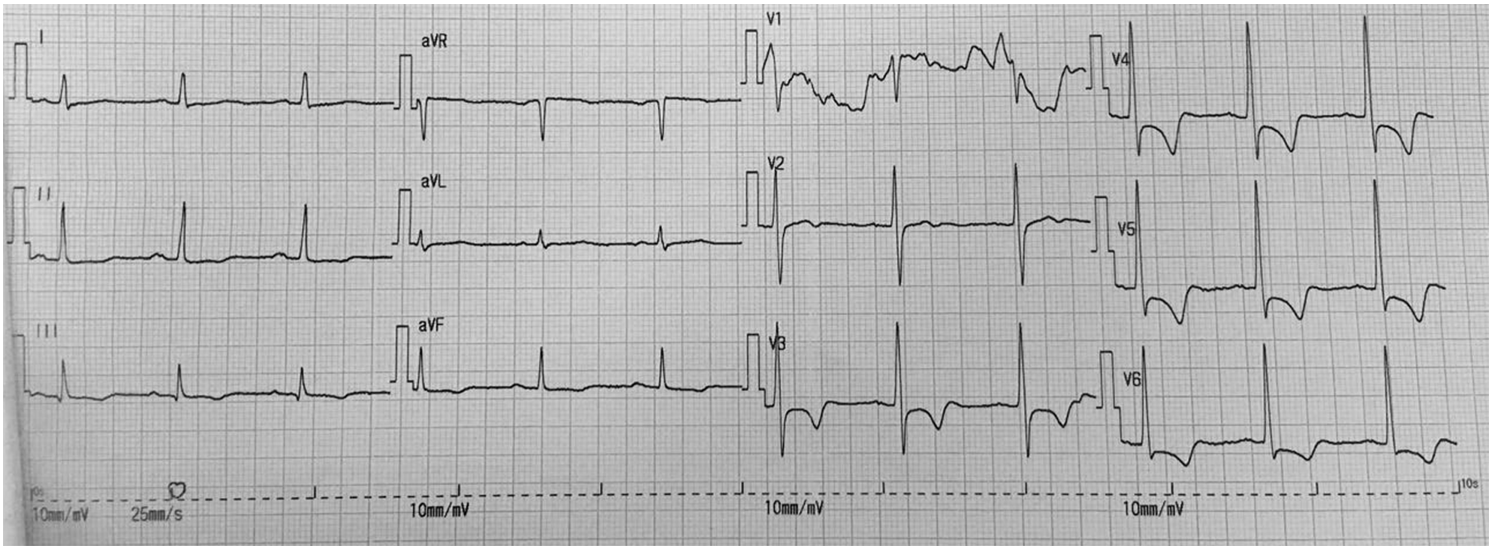

Figure 4

ECG showing deepening amplitude of ST-segment depression and biphasic T-wave in leads V3-V6 after prednisone was initiated.