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Diagnosis of Loeffler Endocarditis in a Patient with a Cerebrovascular Event

Serebrovasküler Olay Yaşayan bir Olguda Löffler Endokarditi Tanısı

ABSTRACT

Cardiac embolic origin is a significant etiological factor in patients presenting with stroke. Thrombi located in the left ventricle may result from various underlying causes. This case report examines a thrombus located in the left ventricular apex of a 76-year-old female patient and presents a comprehensive analysis of the clinical findings, laboratory results, and multimodality imaging techniques used. Furthermore, this report provides a detailed summary of the diagnostic approach to Loeffler endocarditis, a rare but important differential diagnosis.

Keywords: Echocardiography, endocarditis, stroke

ÖZET

İnme hastalarında kardiyak embolik köken önemli bir etyolojik nedendir. Sol ventrikül yerleşimli trombüsler farklı nedenlere bağlı oluşabilmektedir. Bu olguda 76 yaşında bir kadın hastanın sol ventrikül apeksinde yerleşimli bir trombüse klinik, laboratuvar ve özellikle farklı görüntüleme yöntemleri ışığında yaklaşılmış ve hastamız üzerinden nadir görülen Löffler Endokardit tanısına yaklaşım özetlenmiştir.

Anahtar Kelimeler: Ekokardiyografi, endokardit, inme

CASE REPORT
OLGU SUNUMU

Loeffler endocarditis (LE) is a clinical condition caused by rare hypereosinophilic syndromes characterized by persistent eosinophilic infiltration into various tissues. It was first described by W. Loeffler in 1936.[1] Patients may present with nonspecific symptoms such as fatigue, fever, weight loss, and cough, or may develop severe complications such as stroke, heart failure, or malignant arrhythmias. In this case report, we present a patient diagnosed with LE during an evaluation for stroke.

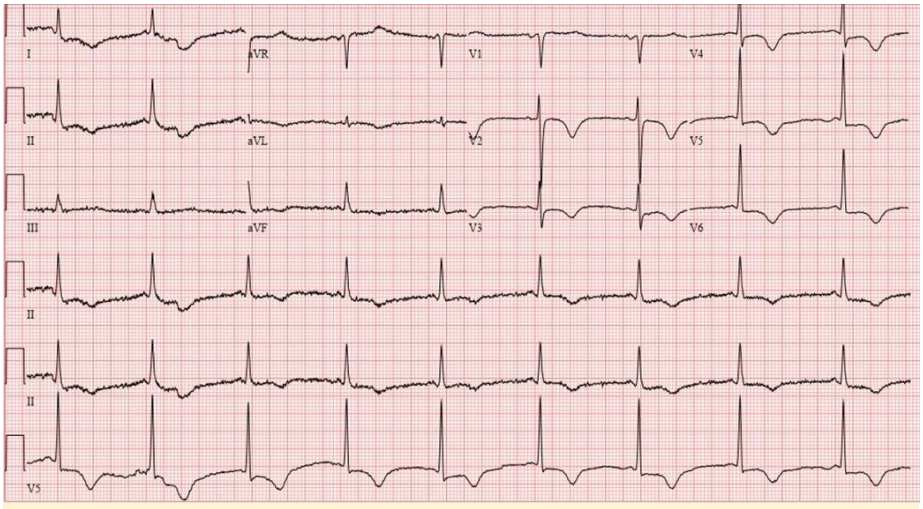

Figure 1. Twelve-lead surface electrocardiography showing diffuse T-wave inversion at admission.

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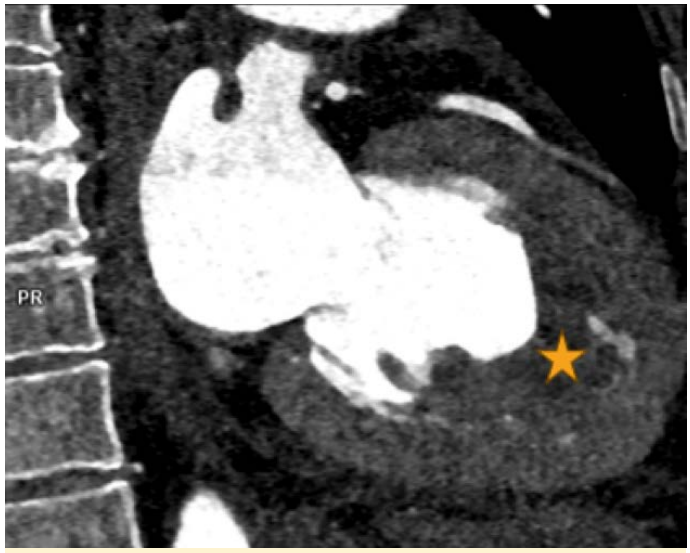

Figure 2. Coronal section image from cardiac computed tomography showing a filling defect (indicated by a star) at the left ventricular apex.

ABBREVIATIONS

AHA	American Heart Association
CCT	Cardiac computed tomography
CMR	Cardiac magnetic resonance imaging
CT	Computed tomography
HCM	Hypertrophic cardiomyopathy
LE	Loeffler endocarditis
LV	Left ventricle
TTE	Transthoracic echocardiography
VKA	Vitamin K antagonist

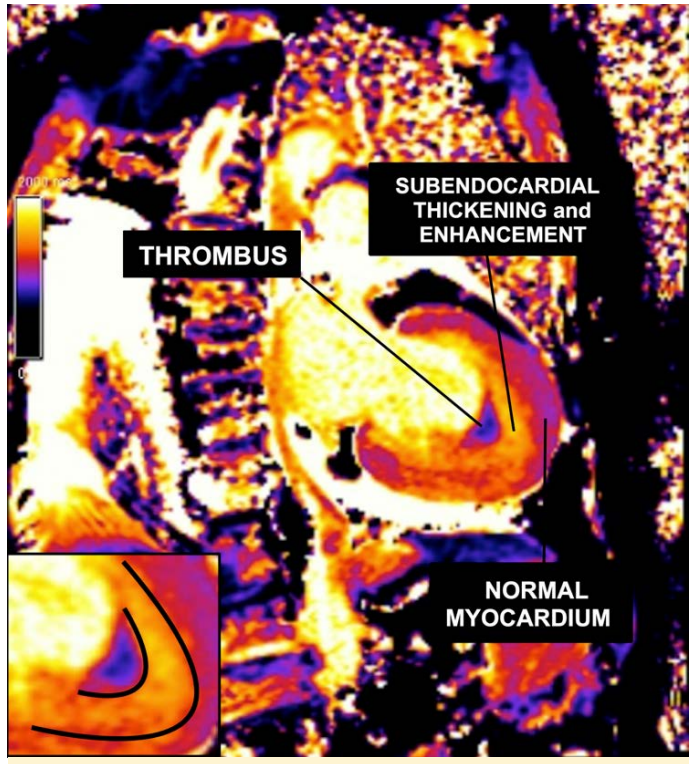

Figure 3. Cardiac magnetic resonance imaging demonstrating the "double V" sign (magnified area at the bottom left) at the ventricular apex. This sign is characterized by a three-layered appearance from the outer to the inner wall, consisting of normal myocardium, a thickened and contrast-enhanced subendocardium, and an overlying thrombus.

Case Report

A 76-year-old female patient with a history of diabetes mellitus, hypertension, and allergic asthma presented with cardiac chest pain. Due to elevated cardiac biomarkers and diffuse T-wave inversions on a 12-lead surface electrocardiogram (Figure 1), coronary angiography was performed. However, no critical stenosis was observed in the coronary arteries.

Following the coronary angiography, the patient complained of weakness in the left lower and upper extremities, along with disorientation. Computed tomography (CT) angiography showed no evidence of an acute intracranial event or major vascular occlusion. However, cranial magnetic resonance imaging revealed acute-to-subacute infarct foci of predominantly embolic origin in the bilateral internal carotid artery border zones of the cerebral and cerebellar cortical-subcortical regions. Intravenous unfractionated heparin therapy was initiated. No rhythm abnormalities were detected during 24-hour Holter monitoring. Transthoracic echocardiography (TTE) revealed an ejection fraction of 45%, with hypokinesia in the apical, apical lateral, and apical septal walls. Additionally, a well-circumscribed iso- to hypoechogenic mass was observed in the apex, appearing as a filling defect (Videos 1-2). Based on echocardiographic findings, apical hypertrophic cardiomyopathy and Takotsubo syndrome were considered in the differential diagnosis. Further TTE findings included a septal e′ of 7 cm/sec, lateral e′ of 10 cm/sec, E/e′ ratio of 21, a left atrial volume index of 41 mL/m^2, and a peak tricuspid regurgitation velocity of 2.7 m/sec, indicative of severe diastolic dysfunction. Subsequently, the patient underwent cardiac computed tomography (CCT), which revealed a lobulated filling defect in the left ventricular apex, along with dyskinesia of the surrounding apical segments (Figure 2). Cardiac magnetic resonance imaging (CMRI) was then performed and demonstrated a characteristic 'double V' sign at the ventricular apex. The sign consisted of a three-layered structure comprising normal myocardium, thickened and contrast-enhanced subendocardium, and an overlying thrombus at the apex (Video 3, Figure 3). The findings were considered suggestive of LE. Given the presence of nasal polyposis, late-onset asthma, and eosinophilia (1881/μL), the patient was referred to the Rheumatology department. To confirm the diagnosis, endomyocardial biopsy and bone marrow aspiration were recommended; however, the patient declined due to the associated risks. Following further evaluation, eosinophilic granulomatosis with polyangiitis was considered, and steroid therapy was initiated. The patient's eosinophil levels normalized during follow-up. The patient was discharged with a Glasgow Coma Scale score of 15, following improvement in left-sided weakness during neurological follow-up. At discharge, treatment included a vitamin K antagonist (VKA), metoprolol, and an angiotensin-converting enzyme inhibitor.

Discussion

The mechanisms underlying stroke in cases of LE are not yet fully understood. However, several contributing factors have been proposed, including embolization from intracardiac thrombi, primary eosinophilic involvement of the central nervous system, and microthromboembolisms resulting from inflammation of the vascular tree within the central nervous system.1 From a cardiological perspective, LE can affect all three layers of the heart: endocardium, myocardium, and pericardium. Consequently, it can lead to valvular heart disease, restrictive cardiomyopathy, and pericarditis.[2] Pathologically, myocardial fibrosis and collagen deposition occur due to fibroblast proliferation, resulting in a stiffer myocardium and ventricular diastolic dysfunction. Additionally, cytokines released from eosinophil granules activate platelets and promote thrombus formation.[3] In this case, we presented an instance of LE causing thrombus formation within the left ventricle (LV). TTE findings initially suggested apical hypertrophic cardiomyopathy (HCM) and Takotsubo syndrome. However, cardiac magnetic resonance imaging (CMR) is the diagnostic tool of choice in cases of uncertainty, as it can demonstrate endocardial involvement and thrombus formation, even in the early stages of the disease.[4] Furthermore, studies have shown that CMR is effective in the differential diagnosis of LV apical HCM and apical masses.[5] LE-related thrombus differs from LV apical thrombus occurring after myocardial infarction, due to the absence of myocardial thinning and transmural contrast enhancement, as well as the the characteristic 'shell-like' structure surrounding the thrombus.[6] Diastolic dysfunction in our patient was another finding supporting the diagnosis of LE.[7] In a previously reported case, a patient presented with dyspnea, orthopnea, and fatigue; TTE revealed diastolic dysfunction and a thrombus at the LV apex. CMR confirmed the thrombus at the LV apex, and bone marrow aspiration showed 25% eosinophils, leading to a diagnosis of LE.[8]

In the present case, multidisciplinary imaging findings supported that the cerebral embolic event originated from the left ventricular apical thrombus. Although cardiac imaging is an essential diagnostic step in clarifying the etiology, endomyocardial biopsy is the gold standard for diagnosing Loeffler endocarditis.[9] Endocardial biopsy and bone marrow aspiration were recommended to the patient; however, she declined due to the associated risks. The high sensitivity and specificity of cardiac magnetic resonance imaging (MRI) make it a valuable tool in distinguishing LE from other types of cardiomyopathies.[10]

A review of the literature indicates that most patients with LE and thrombus are treated with steroids and VKAs as anticoagulant therapy. Approximately 43% of patients demonstrated complete resolution of intracardiac thrombus during follow-up, with no additional thromboembolic events.[3] In the same study, mortality was found to be higher among patients who did not receive anticoagulation therapy and those with concomitant heart failure. Clinical guidelines, including those from the American Heart Association (AHA) and American Stroke Association (ASA) Stroke guidelines, recommend anticoagulant therapy for at least three months in the presence of an intracardiac thrombus.[11-15] Our patient had experienced a stroke, and due to the presence of an intracardiac thrombus, was discharged with VKA therapy. Additionally, the patient responded well to steroid treatment and showed neurological improvement. Although LE is a rare condition, it can be diagnosed through clinical, laboratory, and imaging findings, with CMRI playing a particularly important role in the differential diagnosis. The most critical aspect of managing these patients is addressing the underlying cause of hypereosinophilia, optimizing heart failure treatment according to ejection fraction, and initiating anticoagulant therapy in cases where thrombus is present.[16]

Conclusion

Although rare, Loeffler endocarditis should be considered and suspected by cardiologists, particularly in patients undergoing evaluation for stroke. An elevated eosinophil count may serve as an important laboratory clue. In this case, we presented a diagnostically challenging example of LE and aimed to emphasize the critical role of cardiac imaging in identifying cardiac etiologies in patients presenting with stroke.

Ethics Committee Approval: This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

Informed Consent: Written informed consent was obtained from the patient for the publication of this case report.

Conflict of Interest: The authors have no conflicts of interest to declare.

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Peer-review: Externally peer-reviewed.

Video 1. Transthoracic echocardiography (apical long-axis view) showing a well-circumscribed, iso- to hypoechogenic mass in the apical region of the left ventricle, appearing as a filling defect, along with mild pericardial effusion.

Video 2. Transthoracic echocardiography (apical four-chamber view) showing a well-circumscribed, iso- to hypoechogenic mass in the apical region of the left ventricle, appearing as a filling defect.

Video 3. Coronal cine image from cardiac magnetic resonance imaging showing left ventricular apical akinesia and an apical thrombus.

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