

18F-FLUORODEOXYGLUCOSE POSITRON EMISSION TOMOGRAPHY WITH COMPUTED TOMOGRAPHY IN THE DIAGNOSIS OF LOEFFLER'S ENDOCARDITIS: A CASE REPORT

Milica Kotur¹  Nikola Pantić¹  Lenka Grujić¹  Isidora Grozdić Milojević^{1,2} 
Dragana Šobić Šaranović^{1,2}  Vera Artiko^{1,2}  Strahinja Odalović^{1,2} 

¹Center for Nuclear Medicine with PET, University Clinical Center of Serbia, Belgrade, Serbia ²University of Belgrade Faculty of Medicine, Belgrade, Serbia

Loeffler's endocarditis (LE) is a rare form of inflammatory cardiomyopathy. The condition arises from diffuse eosinophilic infiltration of the endomyocardium, followed by eosinophil degranulation and progressive tissue fibrosis. The aim of this case report is to present a rare form of restrictive cardiomyopathy, LE, in a young female patient, and to highlight the importance of fluorodeoxyglucose positron emission tomography combined with computed tomography (FDG PET/CT) as a valuable diagnostic tool for accurate diagnosis, assessment of cardiac involvement, and determination of disease extent. We present the case of a female patient who underwent FDG PET/CT imaging due to suspected LE, following the detection of eosinophilia and a series of prior diagnostic procedures. FDG PET/CT contributed to the timely diagnosis and precise localization of inflammatory lesions in the cardiac walls. Based on the presented case, we conclude that FDG PET/CT imaging can serve as a helpful tool in the diagnosis and evaluation of LE due to its unique capability to visualize metabolic activity in tissues, a feature often beyond the reach of conventional diagnostic methods. This modality enables accurate identification of inflammatory lesions in the endocardium and myocardium, which are characteristic of LE.

Keywords: hypereosinophilic syndrome, eosinophilic infiltration, Loeffler's endocarditis, FDG PET/CT

Submitted: February 3, 2025 **Revised:** July 6, 2025

Accepted: August 2, 2025

Published online: March 15, 2026

Copyright: © 2026, Author(s). This is an open-access article published under the terms of the Creative Commons Attribution 4.0 International License. (<http://creativecommons.org/licenses/by/4.0/>).

Correspondence to:

Milica Kotur
University Clinical Center of Serbia, Belgrade, Serbia
Center for Nuclear Medicine with PET
Pasterova 2, Belgrade, Serbia
E-mail: koturmilica99@gmail.com

INTRODUCTION

Loeffler's endocarditis (LE) is a rare form of inflammatory cardiomyopathy, first described by Swiss physician Wilhelm Loeffler in 1936 (1, 2). The condition arises from diffuse eosinophilic infiltration of the endomyocardium, followed by eosinophil degranulation and progressive tissue fibrosis. This pathological process leads to impaired diastolic function and classifies LE as a subtype of restrictive cardiomyopathy (2). LE is estimated to occur in approximately 60% of patients with hypereosinophilic syndrome (HES), a disorder characterized by persistent overproduction of eosinophils (3, 4). In the differential diagnosis of eosinophilia-associated cardiac involvement, other potential etiologies should be considered, including eosinophilic granulomatosis with polyangiitis (formerly Churg–Strauss syndrome), early-stage giant cell myocarditis, drug-induced hypersensitivity reactions, and parasitic infections (5).

In addition to peripheral blood analysis, where hypereosinophilia represents a primary diagnostic criterion, other key diagnostic modalities include echocardiography, chest computed tomography (CT), cardiac magnetic resonance imaging (CMRI), bone marrow biopsy, and endomyocardial biopsy (6). Fluorodeoxyglucose positron emission tomography combined with computed tomography (FDG PET/CT) enables accurate detection of inflamed myocardial tissue during the earliest stages of LE, thereby facilitating timely therapeutic intervention and potentially preventing irreversible cardiac damage (7). This modality is particularly useful when CMRI is contraindicated, when standard diagnostic methods yield inconclusive results, or when evaluating the extent of systemic involvement in HES. However, it should be noted that while FDG PET/CT can reliably identify inflamed areas, it lacks the ability to differentiate the specific cellular components of the infiltrate (8).

The aim of this case report is to present a rare form of restrictive cardiomyopathy, LE, in a young female patient, and to highlight the importance of FDG PET/CT as a valuable diagnostic tool for accurate diagnosis, assessment of cardiac involvement, and determination of disease extent.

CASE REPORT

A 21-year-old female patient was referred to the Center for Nuclear Medicine with PET at the University Clinical Center

of Serbia for FDG PET/CT imaging, with suspected LE. Anamnestic data revealed that approximately nine months prior, she experienced an intermittent urticarial skin rash, treated with antihistamines and corticosteroids. Laboratory tests demonstrated marked eosinophilia ($7.96 \times 10^9/L$, 62%), elevated C-reactive protein (CRP) levels (90.9 mg/L), and significantly increased N-terminal pro B-type natriuretic peptide (NT-proBNP) concentrations, ranging from 2,911 pg/mL to 11,333 pg/mL over five days. Flow cytometry immunophenotyping revealed an increased eosinophil count (52% of total leukocytes) alongside a decreased absolute CD4+ T lymphocyte count (250 cells/ μL), without evidence of atypical T lymphocytes. Histopathological analysis of bone marrow biopsy suggested hypereosinophilia of reactive or neoplastic origin. Chest CT imaging showed no pathological abnormalities.

Two weeks prior to FDG PET/CT imaging, the patient was evaluated by a cardiologist due to severe chest pain, dyspnea, and elevated body temperature. Echocardiography revealed thickening of the left ventricular wall with heterogeneous echogenicity and minimal pericardial effusion. CMRI, performed ten days before FDG PET/CT, demonstrated reduced left ventricular systolic function (ejection fraction 49%) and an intracavitary thickened endocardial layer exhibiting edema and granulation along all walls except the septum, with thickness ranging from 14 to 21 mm. The presence of mural thrombosis and a thrombus at the apex of the left ventricle, along with the involvement of the posterior mitral leaflet and both papillary muscles by thrombotic changes, further indicated cardiac complications. The extent of myocardial fibrosis was estimated at approximately 21%. These findings were consistent with the diagnosis of LE.

FDG PET/CT imaging was performed using the Discovery PET/CT Elite scanner (GE Healthcare). Prior to the procedure, the patient provided informed consent for the study. The patient fasted for six hours before receiving an intravenous injection of FDG at a dose of 195 MBq. Scanning commenced 82 minutes post-injection. CT images were acquired from the skull vertex to the mid-thigh, followed by PET data acquisition over the same region. All PET/CT images were reconstructed and analyzed using Volume Viewer software on the AW 4.5 Workstation (GE Healthcare). FDG uptake was quantitatively evaluated by calculating the maximum standardized uptake value (SUVmax) of the radiotracer. FDG PET/CT imaging revealed cardiomegaly. The left

ventricular walls, including the interventricular septum, showed diffusely increased FDG uptake (SUVmax 15.5) (Figures 1 and 2), with focal wall thickening observed in the upper lateral segment corresponding to the papillary muscle region (Figure 3). Mild to moderate FDG uptake was also noted in the walls of the left atrium and right ventricle (SUVmax 9 in the right ventricle; SUVmax 7 in the left atrium) (Figure 4). No structural abnormalities were detected on the low-dose CT component of the PET/CT scan; thus, interpretation was based on subjective visual assessment and SUVmax values. Based on the observed pattern of FDG distribution and uptake in the cardiac walls, wall thickness was estimated to reach up to 20 mm in the left ventricle and up to 7 mm in the left atrium.

The conclusion of the FDG PET/CT scan is that the diffuse and intense radiotracer uptake in the walls of the left ventricle, and, to a lesser extent, in the right ventricle and left atrium, primarily reflects inflammatory changes associated with the underlying disease.

DISCUSSION

This study presents a rare case of HES-associated LE in a 21-year-old female patient. The patient had previously undergone CMRI, which revealed significant findings consistent with LE, corroborating the results of the FDG PET/CT examinations.

HES is a rare hematological disorder, and LE accounts for approximately 50% of all HES cases (4, 9). The presented case is noteworthy for several reasons. LE often occurs in younger populations, as demonstrated here, where it can cause permanent myocardial damage and increase morbidity and mortality. Furthermore, this case underscores the importance of utilizing both invasive and non-invasive diagnostic methods in the diagnosis of LE and assessment of disease extent.

Langwieser et al. (10) reported a case of a 73-year-old female patient with LE who underwent PET/MRI imaging following the detection of eosinophilia in the blood. Similar to our case, the patient presented with dyspnea, and laboratory tests revealed elevated cardiac enzyme levels three weeks prior to the PET/MRI examination. The MRI component of the hybrid PET/MRI system demonstrated late gadolinium enhancement (LGE) lesions in the endocardium of the apical regions of both left and right ventricles, along with masses, likely thrombi, also located in the apical regions of both ventricles, which did not exhibit LGE. Concurrently, the FDG PET/CT examination showed significant FDG uptake in the LGE areas and apical

masses, indicating the presence of active inflammatory tissue.

This case parallels ours, in which PET and MRI scans, although performed as separate modalities rather than a hybrid system, revealed characteristic findings that, in conjunction with clinical presentation, laboratory data, and other information, supported the diagnosis of LE.

Khalid et al. (11) reported a case of an 83-year-old female patient with asthma treated with corticosteroids who presented with worsening dyspnea. Laboratory findings demonstrated leukocytosis, eosinophilia, and elevated levels of troponin T, NT-proBNP, and CRP, findings that correlate with those in our study. Transthoracic echocardiography revealed an ejection fraction (EF) of 35%. CMRI identified LGE in the epicardium of the left ventricle. The FDG PET/CT scan, however, did not reveal any hypermetabolic lesions consistent with LE. Despite this, the diagnosis of LE was subsequently confirmed, and high-dose steroid therapy was initiated, resulting in significant clinical improvement.

Compared to our case, there is a similarity in the presenting symptoms and laboratory findings. However, the patient described by Khalid et al. exhibited a significantly reduced EF, which may be attributed to her advanced age, making it difficult to determine if her EF was preserved prior to the onset of HES. Furthermore, the FDG PET/CT results differ from ours, as hypermetabolic lesions indicative of LE were detected in our patient but not in theirs.

Chen P. et al. (12) reported a case of a 34-year-old patient who underwent FDG PET/CT imaging due to suspected LE. The patient initially presented with high fever and severe cough, and medical history revealed bronchial asthma diagnosed more than ten years earlier. Laboratory findings demonstrated marked peripheral eosinophilia, along with elevated troponin T, NT-proBNP, and C-reactive protein (CRP) levels. Echocardiography showed thickening of the mid to apical segments of the left ventricular wall, with the most pronounced changes in the apex, raising suspicion of eosinophilic endocarditis. FDG PET/CT imaging revealed diffusely increased FDG uptake in the left ventricular wall, further supporting the diagnosis of LE.

Unlike the patient in our case, this patient predominantly exhibited respiratory symptoms. Nonetheless, similar to our case, the integration of clinical presentation, laboratory results, echocardiography, and FDG PET/CT findings was essential in confirming the diagnosis of LE.

Chen et al. (12) also reported a case of a 46-year-old patient with liver cirrhosis who presented with abdominal

pain. The medical history revealed a previous episode of malaria. Laboratory tests demonstrated elevated NT-proBNP levels, eosinophilia, and increased total IgE, IgG, and IgG4 antibody concentrations. Transthoracic echocardiography showed dilated cardiac chambers, reduced right ventricular systolic function, and the presence of mural thrombi. Further serological analyses revealed elevated PR3-Ig (cANCA) antibody levels, suggestive of ANCA-associated vasculitis. CMRI demonstrated subendocardial late gadolinium enhancement (LGE) in both ventricles, consistent with findings characteristic of LE. Subsequently, FDG PET/CT identified multiple hypermetabolic lymph nodes in various anatomical regions, along with an enlarged cardiac silhouette. As a final diagnostic procedure, a biopsy of the right axillary lymph node was performed, with histopathology confirming lymphoid hyperplasia with abundant plasma cells and eosinophilic infiltration. This case was considerably more complex than ours, primarily due to the presence of multiple comorbidities. In both cases, CMRI and echocardiography revealed pathological cardiac changes indicative of LE. However, unlike our patient, FDG PET/CT did not show significant cardiac abnormalities, serving mainly to detect hypermetabolic lymphadenopathy. Nonetheless, when

combined with other diagnostic modalities, FDG PET/CT played a crucial role in establishing the diagnosis of ANCA-associated vasculitis and LE.

Based on this and other reported cases used for comparison, it is evident that FDG PET/CT holds significant diagnostic value in the evaluation of LE. However, it is important to emphasize that this imaging modality cannot differentiate whether myocardial infiltration is mediated by eosinophils or other inflammatory cells, which limits the sensitivity and specificity of FDG PET/CT in this context.

Based on the presented case, we conclude that FDG PET/CT imaging can serve as a helpful tool in the diagnosis and evaluation of LE, due to its unique capability to visualize metabolic activity in tissues, a feature often beyond the reach of conventional diagnostic methods. This modality enables accurate identification of inflammatory lesions in the endocardium and myocardium, which are characteristic of LE. When integrated with clinical presentation, laboratory findings, and other imaging techniques, FDG PET/CT substantially contributes to establishing a precise and timely diagnosis of LE.

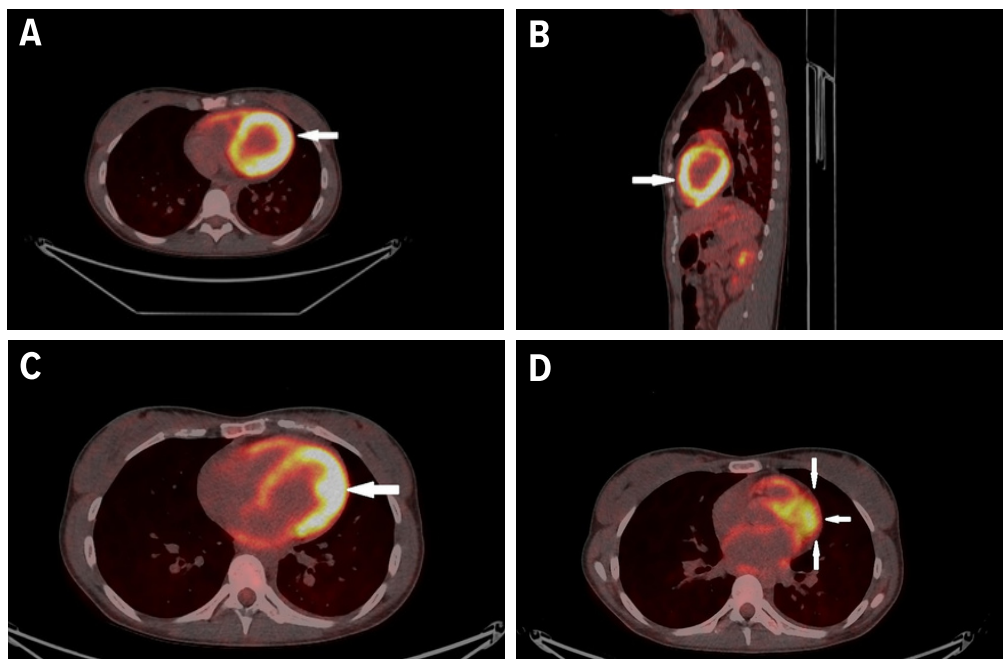


Figure 1. (A) The PET/CT image of the transversal section shows intense accumulation of FDG in the walls of the left ventricle and the septum (white arrow). (B) The PET/CT image of the sagittal section shows diffuse intense accumulation of FDG in the walls of the left ventricle (white arrow). (C) The PET/CT image of the transversal section shows focal left ventricular thickening, most likely corresponding to papillary muscle and less likely mural thrombus seen on CMRI septum (white arrow). (D) PET/CT image of the transversal section shows intense accumulation of FDG in the walls of the left atrium (white arrows).

Acknowledgements

The study was supported by Grant No 451-03-137/2025-03/200110, Ministry of Science, Technological Development and Innovation.

Authors' Contribution

Conceptualization & investigation: M.K., N.P., L.G., I.G.M., D.Š.Š., V.A., and S.O.; Writing – original draft, review, & editing: M.K., N.P., L.G., I.G.M., D.Š.Š., V.A., and S.O. All authors have read and approved the published version of the manuscript.

Statement of Ethics

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. All procedures performed were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration.

Statement of Competing Interest

The authors declare no relevant conflicts of interest.

Statement of Data Availability

All relevant data supporting the findings of this case report are included within the article. Additional information is available from the author upon reasonable request, in accordance with patient confidentiality requirements.

Statement of Generative AI Use

No generative AI was used.

Publisher's Note: The statements, opinions, and data contained in AFMN Biomedicine articles are solely those of the individual author(s) and contributor(s) and do not necessarily represent the views of the publisher or the editor(s). The publisher and editor(s) disclaim responsibility for any harm or damage caused by the use of information or products mentioned in the publication.

REFERENCES

- Zhang Q, Si D, Zhang Z, Zhang W. Loeffler endocarditis with intracardiac thrombus: case report and literature review. *BMC Cardiovasc Disord.* 2021;21(1):615. [\[CrossRef\]](#)
- Mubarik A, Iqbal AM. Loeffler Endocarditis. In: *StatPearls. Treasure Island (FL): StatPearls Publishing; 2024*
- Tefferi A, Patnaik MM, Pardanani A. Eosinophilia: secondary, clonal and idiopathic. *Br J Haematol.* 2006;133:468-92. [\[CrossRef\]](#)
- Parrillo JE. Heart disease and the eosinophil. *N Engl J Med.* 1990;323(22):1560-1. [\[CrossRef\]](#)
- Brucato A, Maestroni S, Masciocco G, Ammirati E, Bonacina E, Pedrotti P. Il coinvolgimento cardiaco nella sindrome di Churg-Strauss [Cardiac involvement in Churg-Strauss syndrome]. *G Ital Cardiol (Rome).* 2015;16(9):493-500. [\[CrossRef\]](#)
- Salih M, Ibrahim R, Tirunagiri D, Al-Ani H, Ananthasubramaniam K. Loeffler's endocarditis and hypereosinophilic syndrome. *Cardiol Rev.* 2021;29(3):150–15. [\[CrossRef\]](#)
- Kadkhodayan A, Chareonthaitawee P, Raman SV, Cooper LT. Imaging of Inflammation in Unexplained Cardiomyopathy. *JACC Cardiovasc Imaging.* 2016;9(5):603-17. [\[CrossRef\]](#)
- Polito MV, Hagendorff A, Citro R, Prota C, Silveiro A, De Angelis E, et al. Loeffler's Endocarditis: An Integrated Multimodality Approach. *J Am Soc Echocardiogr.* 2020;33(12):1427-41. [\[CrossRef\]](#)
- Curtis C, Ogbogu P. Hypereosinophilic Syndrome. *Clin Rev Allergy Immunol.* 2016;50(2):240-51. [\[CrossRef\]](#)
- Langwieser N, von Olshausen G, Rischpler C, Ibrahim T. Confirmation of diagnosis and graduation of inflammatory activity of Loeffler endocarditis by hybrid positron emission tomography/magnetic resonance imaging. *Eur Heart J.* 2014;35(36):2496. [\[CrossRef\]](#)
- Khalid F, Holguin F. Idiopathic Hypereosinophilic Syndrome in an Elderly Female: A Case Report. *Am J Case Rep.* 2019;20:381-4. [\[CrossRef\]](#)
- Chen P, Cheng H, Mou Y. Four challenging cases of eosinophilic endocarditis or myocarditis with literature review. *J Cardiothorac Surg.* 2025;20(1):241. [\[CrossRef\]](#)