Original Article

Monitoring Myocardial Metabolic Changes in Lymphoma Patients Undergoing Chemotherapy Using FDG PET/CT

Diego Rafael Freitas Berenguer,[1,2] Gustavo Freitas Alves de Arruda,[1] Monica de Moraes Chaves Becker,[1] Mayara Lais Coelho Dourado,[2] Roberto de Oliveira Buril,[1] Felipe Alves Mourato,[2] Paulo José de Almeida Filho,[3] Brivaldo Markman-Filho,[1] Simone Cristina Soares Brandão[1,4]

Universidade Federal de Pernambuco,[1] Recife, PE – Brazil
Hospital das Clínicas da Universidade Federal de Pernambuco,[2] Recife, PE – Brazil
Real Hospital Português de Beneficência em Pernambuco,[3] Recife, PE – Brazil
Brigham and Women's Hospital,[4] Boston, Massachusetts – USA

Abstract

Background: Cardiotoxicity is a serious adverse effect of chemotherapy, often identified only after irreversible myocardial damage has occurred. Positron emission tomography/computed tomography (PET/CT) using fluorine-18 fluorodeoxyglucose (^{18}F-FDG) allows for the evaluation of myocardial glucose metabolism and may help detect early metabolic changes related to chemotherapy.

Objective: To assess changes in ^{18}F-FDG standardized uptake values (SUVs) across different cardiac regions before and after chemotherapy in patients with lymphoma, and to identify which region exhibits the greatest increase.

Methods: This retrospective cohort study included 62 lymphoma patients who underwent ^{18}F-FDG PET/CT before and after chemotherapy. SUV measurements were obtained in the left ventricular (LV) free wall, interventricular septum (IVS), right ventricular (RV) free wall, and global myocardium. Control regions included the liver and aorta. Pre- and post-treatment SUV values were compared to evaluate metabolic changes related to chemotherapy.

Results: Myocardial ^{18}F-FDG uptake increased significantly after chemotherapy across all cardiac regions, with the most pronounced rise observed in the LV free wall (maximum SUV increase of 73%, $p<0.001$). The RV free wall showed a non-significant increase in SUV, and no significant changes were observed in the liver or aorta.

Conclusions: Chemotherapy was associated with a global increase in myocardial ^{18}F-FDG uptake, with the most pronounced elevation observed in the LV free wall. This regional predominance highlights the LV free wall as the most sensitive site for detecting early metabolic changes potentially related to cardiotoxicity.

Keywords: Cardiotoxicity; Chemotherapy; ^{18}F-FDG PET/CT; Myocardial metabolism; Lymphoma.

Introduction

Cardiotoxicity (CTX) is one of the most severe long-term adverse effects of cancer treatment, potentially leading to heart failure with reduced ejection fraction (HFrEF) and other cardiovascular complications.[1] Although initially defined as a reduction in left ventricular ejection fraction (LVEF), CTX has been expanded in recent guidelines to include a broader range of cardiovascular conditions.[2,3] Among chemotherapy drugs, anthracyclines remain one of the leading causes of CTX due to their well-documented association with HFrEF, which is often irreversible and carries high morbidity and mortality.[4-8]

Mailing Address: Diego Rafael Freitas Berenguer •
Hospital das Clínicas da Universidade Federal de Pernambuco. Av. Professor Moraes do Rego, Cidade Universitária. Postal Code: 50670-901, Recife, PE – Brazil
E-mail: diegoberenguer@gmail.com
Manuscript received September 19, 2025; revised September 30, 2025; accepted September 30, 2025
Editor responsible for the review: Marcelo Dantas

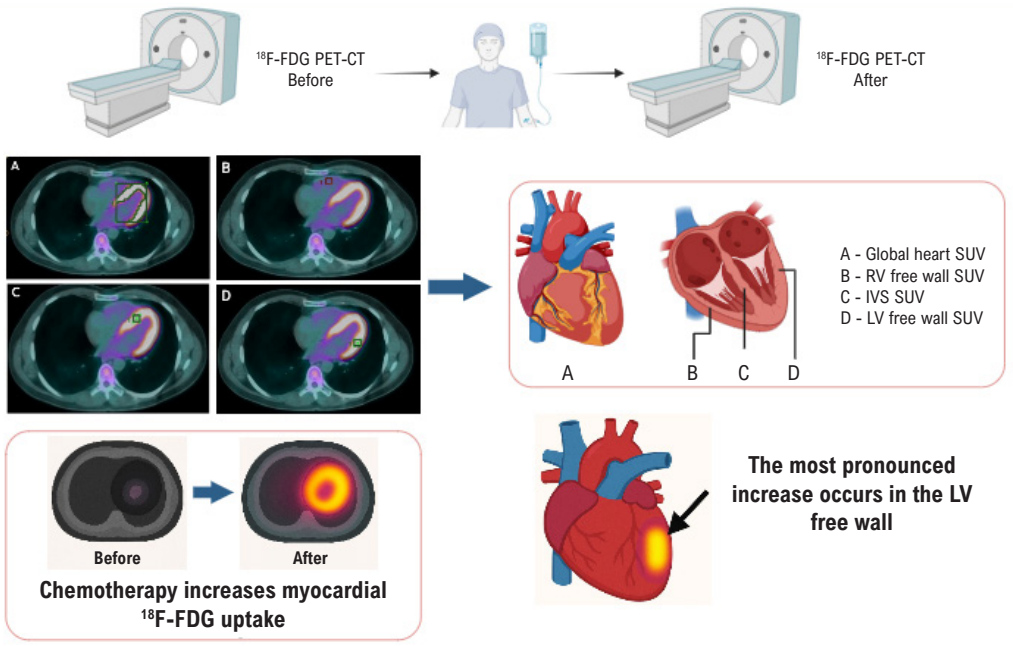
DOI: https://doi.org/10.36660/abcimg.20250067i

The clinical manifestations of CTX often appear months or years after initial chemotherapy, limiting the utility of traditional methods like LVEF assessment, which detects systolic dysfunction only after substantial myocardial injury has occurred.[3,9,10] Positron emission tomography/computed tomography (PET/CT) with fluorine-18 fluorodeoxyglucose (^{18}F-FDG) offers a promising alternative by providing insights into myocardial metabolism and detecting subtle metabolic changes preceding overt contractile dysfunction.[11] Studies suggest that ^{18}F-FDG uptake alterations in cardiomyocytes during or after chemotherapy may reflect early mitochondrial and metabolic stress, potentially serving as an early marker of CTX.[9-15]

Interestingly, ^{18}F-FDG uptake varies physiologically between different cardiac regions. The left ventricle, due to its higher myocyte density and energy demand, exhibits greater glucose consumption than the right ventricle.[16,17] This disparity may be further influenced by oxidative stress induced by chemotherapy, which could preferentially affect certain myocardial regions. Understanding these regional differences in ^{18}F-FDG uptake may provide deeper insights into the mechanisms of CTX and its early detection.

This study aimed to evaluate changes in ^{18}F-FDG standardized uptake values (SUV) in different myocardial regions before and

Central Illustration: Monitoring Myocardial Metabolic Changes in Lymphoma Patients Undergoing Chemotherapy Using FDG PET/CT



Arq Bras Cardiol: Imagem cardiovasc. 2025;38(4):e20250067

Monitoring myocardial metabolic changes after chemotherapy. ^{18}F-FDG PET-CT: Positron emission tomography/computed tomography with fluorine-18 fluorodeoxyglucose; IVS: Interventricular septum. LV: left ventricle. RV: right ventricle. SUV: standardized uptake values. Created with BioRender.com.

after chemotherapy, offering a novel perspective on metabolic markers of CTX during cancer treatment monitoring.

Materials and Methods

This is a retrospective cohort study that reexamined the medical records and imaging exams of 70 patients who participated in a previous study conducted by the Nuclear Medicine Department of a private hospital between January 1, 2012, and August 28, 2017.

The inclusion criteria were age 18 years or older; diagnosis of lymphoma; and having undergone at least two ^{18}F-FDG PET/CT scans – one prior to chemotherapy and another during or after treatment, as illustrated in the Central Illustration.

Patients were excluded due to insufficient data in their medical records, unavailability or inability to evaluate the ^{18}F-FDG PET/CT images, prior mediastinal chemotherapy or radiotherapy (before the baseline PET/CT of this study), or insulin therapy administered on the day of any ^{18}F-FDG PET/CT exam. After reviewing the exams, eight patients were excluded due to the inability to assess the images.

Clinical data and personal history were retrieved from the medical records attached to the initial assessment form, which was completed by the patient prior to each PET scan. For PET/CT analysis, we collected the following parameters: ^{18}F-FDG injected dose, activation time, and both maximum and mean SUV of the descending aorta, liver, and heart. We measured ^{18}F-FDG SUV in various cardiac regions: the whole heart, the mid-region of the septal wall, the left ventricular (LV) free wall, and the right ventricular (RV) free wall (see Central Illustration; Figure 1).

The evolution of SUVs was analyzed based on the numerical measurements in the different cardiac segments evaluated, as well as in the control organs (aorta and liver). A comparison between the final and baseline maximum SUV values was also performed.

^{18}F-FDG PET/CT Protocol

Patients were instructed to fast for six hours prior to the exam, continue taking their usual medications, and avoid physical activity for 24 hours beforehand. To proceed with the exam, blood glucose levels had to be below 180 mg/dL. The radiopharmaceutical ^{18}F-FDG was administered at a dose of 3.7 to 4.8 MBq/kg, and patients were instructed to rest for 60 minutes (corresponding to the uptake period). After this interval, they were taken to the imaging room for acquisition.

All exams were performed using the same PET/CT scanner (Biograph 16, Siemens Healthcare, USA). Images were acquired from the skull base to the proximal middle third of the lower limbs (femur) in three-dimensional mode, with a scan time of

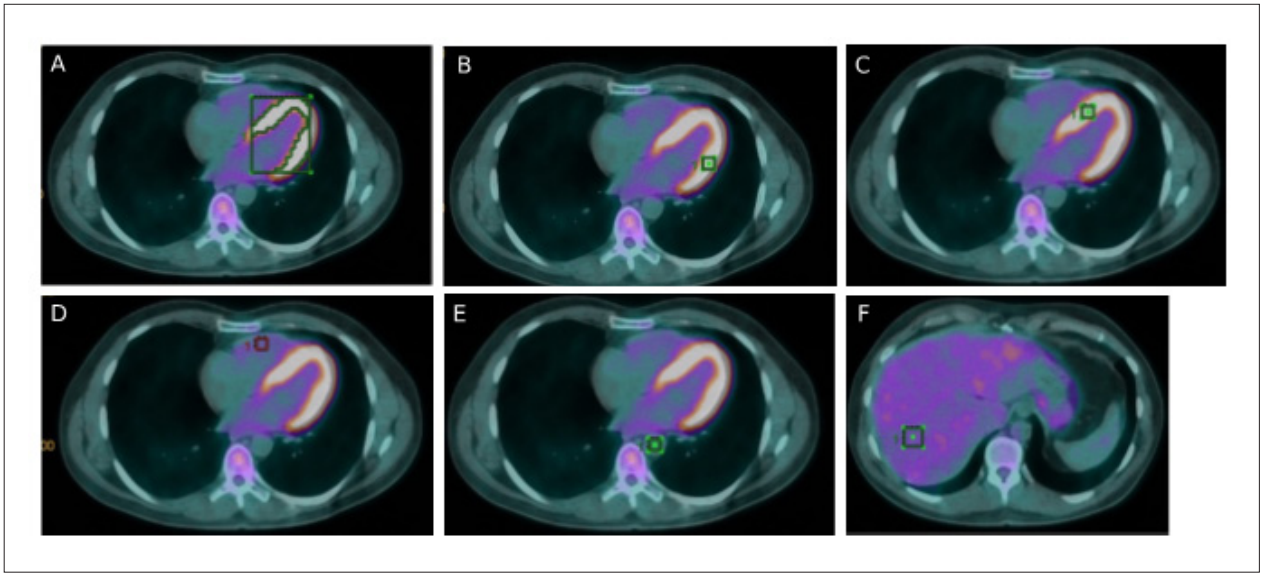


Figure 1 – *Regions of interest (ROIs) for SUV acquisition in various cardiac sites and in control regions (aorta and liver) (A), global heart SUV; (B), LV free wall SUV; (C), IVS SUV; (D), RV free wall; (E), Aorta SUV; (F), Liver SUV. SUV: standardized uptake value, LV: left ventricle, IVS: interventricular septum, RV: right ventricle*

three minutes per bed position (each body segment). The images were processed using iterative reconstruction (2 iterations with 8 subsets and a Gaussian filter).

Computed tomography (CT) acquisition parameters included a 5 mm slice thickness, 120 kV voltage, and no intravenous contrast administration. Additionally, the exam was complemented by a chest helical acquisition performed during maximum inspiration.

Statistical Analysis

Data were analyzed using R software version 4.0.0 (R Foundation for Statistical Computing, Vienna, Austria). Numerical data are presented as median and interquartile range, as they do not follow a normal distribution, while categorical data are expressed as n (%). The Bayesian Repeated Measures Profile Modeling (BRPM) method was used to compare SUV values between baseline and final PET groups. SUV analysis was performed across all cardiac regions as well as in the liver and aorta. A significance level of 5% was adopted to reject the null hypothesis.

Results

General Descriptive Analysis

A total of 62 patients were included in this study. The sample characteristics are further detailed according to socio-demographic and clinical criteria (Table 1). The mean age was 49 ± 15 years, and the average weight was 73 ± 9 kg. Clinical characteristics and patient data at the time of the exams are also presented (Table 2).

Evolution of the SUV During Cancer Therapy

Both maximum and mean SUV values increased significantly during cancer treatment across all assessed cardiac regions (see Central Illustration). The most prominent elevations were observed in the global cardiac SUV and the LV free wall SUV. In contrast, the aortic and hepatic SUVs (both mean and maximum) showed no statistically significant differences between the baseline and final PET scans (Table 2). Figure 2 illustrates the progression of maximum and mean SUVs across all cardiac segments and control regions (aorta and liver).

Discussion

This study demonstrated a statistically significant increase in cardiac ^{18}F-FDG uptake across all left ventricular regions during chemotherapy. The most pronounced uptake was observed in the lateral wall of the LV. Although an increase in SUV uptake was also noted in the lateral wall of the RV, this change did not reach statistical significance.

Baucknet et al.[18] reported that cardiac ^{18}F-FDG uptake is directly proportional to the oxidative stress induced by anthracycline use. Therefore, the increased myocardial ^{18}F-FDG uptake in these segments may suggest a greater rise in oxidative stress within the LV regions compared to other sites.

The more noticeable changes in the left heart segments may be due to the higher density of cardiac muscle cells in the left ventricle. Since the left ventricle has a larger muscle mass, it contains more cells that have been affected by oxidative damage caused by chemotherapy.[16] Another possible explanation is that the fibers on the left side of the heart have a greater energy demand. Under oxidative stress, these cells can no longer produce enough energy to function properly, leading to cell death (apoptosis). Because the fibers in the LV require more energy, this process of cell damage occurs more frequently in these fibers, resulting in greater ^{18}F-FDG uptake in the myocardium.[17]

Bulten et al.[19] observed that only some cardiomyocytes are susceptible to anthracycline-induced injury and that this damage

Table 1 – Sociodemographic and clinical characteristics of patients undergoing ^{18}F-FDG PET/CT in the assessment of myocardial uptake during chemotherapy treatment.

Variable (N=62)	N (%)
Female	31 (50%)
Hypertension	13 (20.9%)
Dyslipidemia	9 (14.5%)
Diabetes mellitus	8 (12.9%)
Coronary artery disease	3 (4.8%)
Smoke	
Smoker	2 (3.2%)
Not smoker	44 (70.9%)
Ex-smoker	16 (25.8%)
Type of Lymphoma	
Hodgkin	23 (37%)
Não Hodgkin	39 (62.9%)
Chemoterapy used[a]	
ABVD	12 (37.5%)
ABVD + alternative scheme	1 (3.1%)
R-CHOP	14 (43.9%)
R-CHOP + alternative scheme	2 (6.2%)
BEACOPP	1 (3.1%)
DA-EPOCH-R	1 (3.1%)
Imunotherapy	1 (3.1%)
Mediastinal radiotherapy	9 (14%)
Use of medication	
No	4 (6.4%)
Yes (Cardioprotective)[b]	15 (24.1%)
Sim (Non-cardioprotective)[b]	43 (69.3%)

[a]Available for 32 patients. [b]Cardioprotective medication: angiotensin II receptor blocker, beta-blocker, angiotensin-converting enzyme inhibitor. ABVD: Adriamycin or Doxorubicin + Bleomycin + Vinblastine + Dacarbazine, BEACOPP: Bleomycin + Etoposide + Doxorubicin + Cyclophosphamide + Vincristine + Procarbazine + Prednisone, DA-EPOCH-R: Etoposide + Prednisone + Vincristine + Cyclophosphamide + Doxorubicin + Rituximab Dose adjusted, R-

occurs even with low doses of doxorubicin. A greater ^{18}F-FDG uptake in the different cardiac segments may be a consequence of the greater presence of these susceptible cells in those segments.

It is possible that PET/CT provides a diagnosis of this process in which the oxidative stress increases until the cell starts its apoptosis. It is known that the apoptosis process can be reversed until the moment immediately prior to the cell membrane lysis.[20] If there is a possibility of suppression of this oxidative stress, the cardiomyocytes death could be avoided. Hence the importance of diagnosing this cellular damage as early as possible.

When analyzing the SUV changes, a notable rise in all cardiac segments examined was observed. This finding aligns with the results from the study by Dourado et al.,[21] where they reported an average increase of 66.5% in the maximum global cardiac SUV when comparing baseline PET/CT to PET/CT after chemotherapy.

Baucknehet al.[22] performed ^{18}F-FDG PET/CT scans on 69 patients with Hodgkin's lymphoma undergoing doxorubicin therapy. Four scans were conducted: one at baseline, one during treatment, another four to six weeks after chemotherapy, and a final scan six months post-treatment. The study revealed a significant increase in LV SUV following chemotherapy. Moreover, the LV SUV was notably low prior to treatment and was identified as a potential predictive marker for the development of CTX.

Kim et al.[13] evaluated not only the increase in myocardial ^{18}F-FDG uptake but also the uptake pattern. In their study, among 121 patients monitored, 15 developed CTX, diagnosed via echocardiography one week after treatment with anthracyclines (doxorubicin and epirubicin) or trastuzumab. A diffuse uptake pattern and increased LV ^{18}F-FDG uptake were more frequently observed in patients who developed CTX.[12]

Another key finding was that an increase of 0.4 in the RV maximum SUV, as well as an RV maximum SUV greater than 1.8, were statistically significant indicators of CTX.[12] In the present study, although less pronounced, both the maximum and mean RV SUV values increased during cancer therapy, partially corroborating the findings of Kim et al.[13] These results highlight the need for more targeted studies to better understand the role of the RV and its association with the development of CTX.

While this study offers valuable insights, several limitations should be acknowledged. First, its retrospective design limits control over confounding factors, such as pre-existing cardiac conditions or concurrent medications, which may have influenced the results. Future prospective studies with stricter controls could help mitigate these biases. The small sample size also restricts the generalizability of the findings; expanding the cohort in future research would enhance statistical power and enable subgroup analyses.

Additionally, the lack of functional data – such as echocardiography or cardiac magnetic resonance imaging (MRI) – makes it challenging to correlate metabolic changes with functional outcomes. Including these assessments in future studies would provide a more comprehensive understanding of the relationship between metabolic alterations and CTX. Finally, although the follow-up period was short and the study was conducted at a single center without standardized dietary preparation, larger multi-center studies with extended follow-up and consistent protocols would help overcome these limitations.

Still, the results help to further clarify the behavior of ^{18}F-FDG myocardial uptake before and after chemotherapy. Moreover, this investigation offers an innovative perspective, given the limited number of studies in this field and the fact that CTX diagnosis remains delayed.

Conclusion

This study demonstrated an increased ^{18}F-FDG uptake in various cardiac regions in patients with lymphoma undergoing chemotherapy. Among the evaluated cardiac sites, the maximum

Table 2 – Evolution of standardized uptake value, body weight, and test characteristics in patients undergoing ^{18}F-FDG PET/CT for myocardial uptake assessment during chemotherapy

Variable	Basal[1] (N = 62)	Final[1] (N = 62)	P[2]	Final vs Baseline Dif. (95%CI)[3]
Weight (kg)	73 (66 - 82)	74 (65 - 83)		
Activation time (min)	67 (60 - 76)	65 (60 - 76)		
Dose injected (MBq)	344 (303 - 394)	333 (296 - 392)		
Mean heart SUV	1.80 (1.40 – 2.73)	2.55 (1.61 – 3.77)	< 0.001	0.75 (0.35 – 1.37)
Maximum heart SUV	2.96 (2.29 – 5.47)	5.12 (3.16 – 8.77)	< 0.001	2.23 (1.04 – 3.43)
Mean interventricular septum SUV	1.87 (1.42 – 2.74)	2.62 (1.64 – 4.39)	0.024	0.75 (0.15 – 2.05)
Maximum interventricular septum SUV	2.16 (1.67 – 2.49)	3.00 (1.96 – 5.61)	0.020	0.83 (0.10 – 2.62)
Mean right ventricular SUV	1.33 (1.16 – 1.54)	1.49 (1.31 – 1.72)	0.059	0.16 (-0.01 – 0.31)
Maximum right ventricular SUV	1.69 (1.43 – 1.96)	1.83 (1.60 – 2.10)	0.068	0.14 (-0.01 – 0.36)
Mean left ventricular SUV	2.03 (1.54 – 3.66)	3.62 (1.86 – 5.64)	< 0.001	1.60 (0.68 – 2.66)
Maximum left ventricular SUV	2.43 (1.87 – 4.58)	4.51 (2.28 – 7.18)	< 0.001	2.08 (0.87 – 3.07)
Mean aorta SUV	1.48 (1.28 – 1.67)	1.44 (1.22 – 1.68)	0.766	-0.03 (-0.16 – 0.12)
Maximum aorta SUV	1.80 (1.51 – 2.11)	1.67 (1.45 – 2.02)	0.405	-0.12 (-0.28 – 0.105)
Mean liver SUV	2.20 (1.86 – 2.50)	2.29 (2.04 – 2.63)	0.125	0.09 (-0.05 – 0.24)
Maximum liver SUV	2.57 (2.18 – 2.98)	2.63 (2.38 – 2.99)	0.331	0.06 (-0.10 – 0.22)

[1]Median (interquartile range). Kg: kilograms. min: minutes. MBq: megabecquerel. [2]BRPM Method [3]Difference between medians and their respective 95% confidence interval.

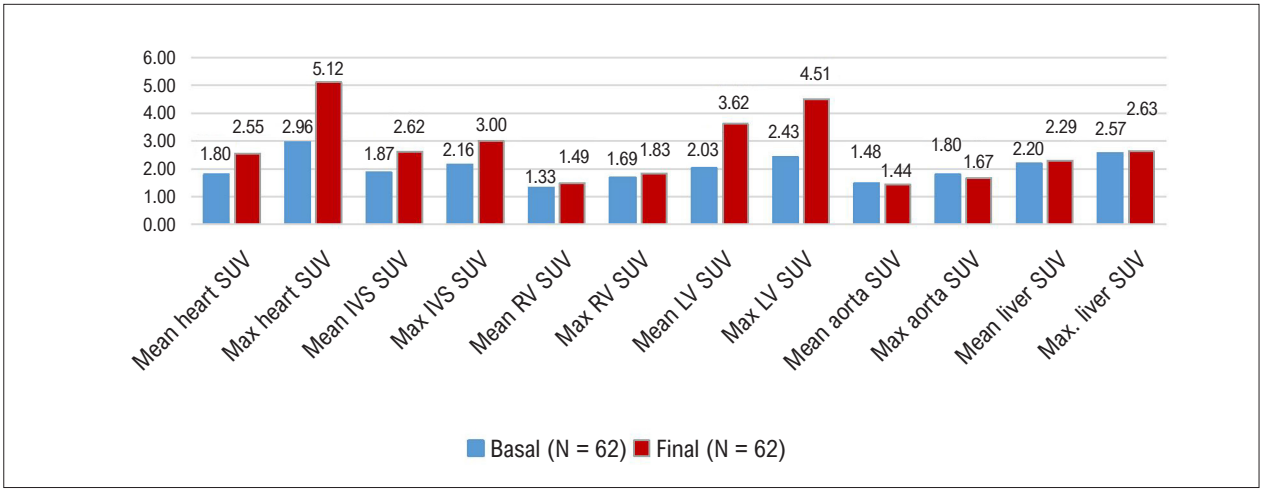


Figure 2 – *Evolution of maximum and mean SUVs (median values) across different cardiac regions and control sites (aorta and liver) at baseline and at the end of chemotherapy treatment. [1]median (interquartile range). IVS: Interventricular septum. LV: left ventricle. RV: right ventricle. AO: aorta. Created with Microsoft Excel.*

SUV in the LV free wall showed the most significant increase during treatment.

Author Contributions

Conception and design of the research: Becker MMC, Markman-Filho B, Brandão SCS. Acquisition of data: Berenguer DRF, Arruda GFA, Dourado MLC, Almeida-Filho PJ, Mourato FA. Analysis and interpretation of the data: Berenguer DRF, Arruda GFA, Becker MMC, Dourado MLC, Buril RO, Almeida-Filho PJ, Mourato FA, Markman-Filho B, Brandão SCS. Statistical analysis: Berenguer DRF, Brandão SCS. Writing of the manuscript: Berenguer DRF, Becker MMC, Markman-Filho B, Brandão SCS. Critical revision of the manuscript for intellectual content:

Berenguer DRF, Becker MMC, Buril RO, Markman-Filho B, Brandão SCS.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

This study was partially funded by FACEPE (Fundação de Apoio a Ciência e Tecnologia de Pernambuco).

Study Association

This article is part of the thesis of master submitted by Diego Rafael Freitas Berenguer, from Graduate Program in Translational Health at the Universidade Federal de Pernambuco (UFPE).

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Hospital das Clínicas of UFPE under the protocol number 4.052.412. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Availability of Research Data

The data cannot be made publicly available because it comes from the database of the Real Hospital Português de Beneficência in Pernambuco, containing clinical information on patients and subject to the institution's confidentiality and secrecy rules. Interested researchers may contact the management committee of the Real Hospital Português de Beneficência in Pernambuco for any formal access requests, which will be analyzed on a case-by-case basis.

Reference

1. Kalil R Filho, Hajjar LA, Bacal F, Hoff PM, Diz MP, Galas FR, et al. I Brazilian Guideline for Cardio-Oncology from Sociedade Brasileira de Cardiologia. Arq Bras Cardiol. 2011;96(2 Suppl 1):1-52.
2. Hajjar LA, Costa IBSDSD, Lopes MACQ, Hoff PMG, Diz MDPE, Fonseca SMR, et al. Brazilian Cardio-Oncology Guideline - 2020. Arq Bras Cardiol. 2020;115(5):1006-43. doi: 10.36660/abc.20201006.
3. Zamorano JL, Lancellotti P, Muñoz DR, Aboyans V, Asteggiano R, Galderisi M, et al. 2016 ESC Position Paper on Cancer Treatments and Cardiovascular Toxicity Developed Under the Auspices of the ESC Committee for Practice Guidelines: The Task Force for Cancer Treatments and Cardiovascular Toxicity of the European Society of Cardiology (ESC). Eur Heart J. 2016;37(36):2768-801. doi: 10.1093/eurheartj/ehw211.
4. Giordano SH, Lin YL, Kuo YF, Hortobagyi GN, Goodwin JS. Decline in the Use of Anthracyclines for Breast Cancer. J Clin Oncol. 2012;30(18):2232-9. doi: 10.1200/JCO.2011.40.1273.
5. Nabhan C, Byrtek M, Rai A, Dawson K, Zhou X, Link BK, et al. Disease Characteristics, Treatment Patterns, Prognosis, Outcomes and Lymphoma-Related Mortality in Elderly Follicular Lymphoma in the United States. Br J Haematol. 2015;170(1):85-95. doi: 10.1111/bjh.13399.
6. Chihara D, Westin JR, Oki Y, Ahmed MA, Do B, Fayad LE, et al. Management Strategies and Outcomes for Very Elderly Patients with Diffuse Large B-Cell Lymphoma. Cancer. 2016;122(20):3145-51. doi: 10.1002/cncr.30173.
7. Ainger LE, Bushore J, Johnson WW, Ito J. Daunomycin: A Cardiotoxic Agent. J Natl Med Assoc. 1971;63(4):261-7.
8. Felker GM, Thompson RE, Hare JM, Hruban RH, Clemetson DE, Howard DL, et al. Underlying Causes and Long-Term Survival in Patients with Initially Unexplained Cardiomyopathy. N Engl J Med. 2000;342(15):1077-84. doi: 10.1056/NEJM200004133421502.
9. Curigliano G, Cardinale D, Suter T, Plataniotis G, Azambuja E, Sandri MT, et al. Cardiovascular Toxicity Induced by Chemotherapy, Targeted Agents and Radiotherapy: ESMO Clinical Practice Guidelines. Ann Oncol. 2012;23(Suppl 7):vii155-66. doi: 10.1093/annonc/mds293.
10. Negishi K, Negishi T, Haluska BA, Hare JL, Plana JC, Marwick TH. Use of Speckle Strain to Assess Left Ventricular Responses to Cardiotoxic Chemotherapy and Cardioprotection. Eur Heart J Cardiovasc Imaging. 2014;15(3):324-31. doi: 10.1093/ehjci/jet159.
11. Berenguer DRF, Becker MMC, Buril RO, Bertão PA, Markman B Filho, Brandão SCS. Progression of Myocardial 18F-FDG Uptake in a Patient with Cardiotoxicity. Arq Bras Cardiol. 2024;121(2):e20230276. doi: 10.36660/abc.20230276.
12. Sarocchi M, Bauckneht M, Arboscello E, Capitanio S, Marini C, Morbelli S, et al. An Increase in Myocardial 18-Fluorodeoxyglucose Uptake is Associated with Left Ventricular Ejection Fraction Decline in Hodgkin Lymphoma Patients Treated with Anthracycline. J Transl Med. 2018;16(1):295. doi: 10.1186/s12967-018-1670-9.
13. Kim J, Cho SG, Kang SR, Yoo SW, Kwon SY, Min JJ, et al. Association between FDG Uptake in the Right Ventricular Myocardium and Cancer Therapy-Induced Cardiotoxicity. J Nucl Cardiol. 2020;27(6):2154-63. doi: 10.1007/s12350-019-01617-y.
14. Becker MMC, Arruda GFA, Berenguer DRF, Buril RO, Cardinale D, Brandão SCS. Anthracycline Cardiotoxicity: Current Methods of Diagnosis and Possible Role of 18F-FDG PET/CT as a New Biomarker. Cardiooncology. 2023;9(1):17. doi: 10.1186/s40959-023-00161-6.
15. Hirata K, Kamagata K, Ueda D, Yanagawa M, Kawamura M, Nakaura T, et al. From FDG and Beyond: The Evolving Potential of Nuclear Medicine. Ann Nucl Med. 2023;37(11):583-95. doi: 10.1007/s12149-023-01865-6.
16. Bengel FM, Higuchi T, Javadi MS, Lautamäki R. Cardiac Positron Emission Tomography. J Am Coll Cardiol. 2009;54(1):1-15. doi: 10.1016/j.jacc.2009.02.065.
17. Pinker K, Riedl C, Weber WA. Evaluating Tumor Response with FDG PET: Updates on PERCIST, Comparison with EORTC Criteria and Clues to Future Developments. Eur J Nucl Med Mol Imaging. 2017;44(Suppl 1):55-66. doi: 10.1007/s00259-017-3687-3.
18. Bauckneht M, Pastorino F, Castellani P, Cossu V, Orengo AM, Piccioli P, et al. Increased Myocardial 18F-FDG Uptake as a Marker of Doxorubicin-Induced Oxidative Stress. J Nucl Cardiol. 2020;27(6):2183-94. doi: 10.1007/s12350-019-01618-x.

19. Bulten BF, Sollini M, Boni R, Massri K, de Geus-Oei LF, van Laarhoven HWM, et al. Cardiac Molecular Pathways Influenced by Doxorubicin Treatment in Mice. Sci Rep. 2019;9(1):2514. doi: 10.1038/s41598-019-38986-w.

20. Okada DR, Liu Z, Johnson G 3rd, Beju D, Khaw BA, Okada RD. 99mTc-Glucarate Kinetics Differentiate Normal, Stunned, Hibernating, and Nonviable Myocardium in a Perfused Rat Heart Model. Eur J Nucl Med Mol Imaging. 2010;37(10):1909-17. doi: 10.1007/s00259-010-1495-0.

21. Dourado MLC, Dompieri LT, Leitão GM, Mourato FA, Santos RGG, Almeida PJ Filho, et al. Chemotherapy-Induced Cardiac18F-FDG Uptake in Patients with Lymphoma: An Early Metabolic Index of Cardiotoxicity? Arq Bras Cardiol. 2022;118(6):1049-58. doi: 10.36660/abc.20210463.

22. Bauckneht M, Ferrarazzo G, Fiz F, Morbelli S, Sarocchi M, Pastorino F, et al. Doxorubicin Effect on Myocardial Metabolism as a Prerequisite for Subsequent Development of Cardiac Toxicity: A Translational 18F-FDG PET/CT Observation. J Nucl Med. 2017;58(10):1638-45. doi: 10.2967/jnumed.117.191122.