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Review Article

FDG PET/CT in the post-treatment evaluation of breast cancer: an emphasis on triple-negative breast cancer

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Abstract: Triple-negative breast cancer (TNBC) is an aggressive subtype accounting for 10-17% of all breast cancers, characterized by the absence of estrogen receptor, progesterone receptor, and human epidermal growth factor receptor 2 expression. TNBC is associated with high recurrence rates and poor prognosis, particularly after metastatic progression. Current surveillance guidelines rely on clinical evaluation and conventional imaging, which have limited sensitivity for detecting early recurrence. [^{18}F]fluorodeoxyglucose positron emission tomography/computed tomography (FDG PET/CT) offers the potential to detect metabolic changes indicative of disease activity before anatomical changes become apparent. This review examines the emerging role of FDG PET/CT in the post-treatment evaluation of TNBC in two distinct clinical contexts: (1) surveillance for recurrence after remission, with specific attention to hepatic, pulmonary, osseous, and cerebral metastases, and (2) treatment response assessment in advanced disease following chemotherapy and immunotherapy. Across both contexts, FDG PET/CT offers incremental diagnostic and prognostic value over conventional imaging. However, much of the available evidence is derived from general breast cancer populations and not from TNBC-specific cohorts. Dedicated recurrence-surveillance data in TNBC remain particularly scarce. FDG PET/CT demonstrated 98% sensitivity and 100% specificity for hepatic metastases in a mixed-malignancy cohort, PET Response Criteria in Solid Tumors (PERCIST) criteria showed higher predictive accuracy than Response Evaluation Criteria in Solid Tumors (RECIST) for both progression-free and disease-specific survival, and early interim PET/CT after two cycles of chemotherapy identified metabolic non-responders (<42% SUV_{max} decrease) who had a 100% rate of residual tumor at surgery in a small TNBC cohort. However, FDG PET/CT remains limited for sub-centimeter pulmonary nodules (due to partial volume effects and respiratory motion) and brain metastases (due to high physiological brain glucose uptake). Additionally, limitations of the current evidence include small study populations, short follow-up periods, cost considerations, and risks of false-positive findings due to post-treatment inflammation and immunotherapy-related pseudoprogression. Beyond FDG, emerging PET tracers, such as radiolabeled fluorothymidine (FLT), fibroblast activation protein inhibitor (FAPI), and prostate-specific membrane antigen (PSMA)-targeted agents, may offer complementary information for TNBC surveillance. Larger, TNBC-specific prospective studies with longer follow-up are needed to establish evidence-based guidelines for incorporating FDG PET/CT into routine TNBC surveillance and response-assessment protocols, alongside formal cost-effectiveness analyses.

Keywords: FDG PET/CT, humans, inflammation, liver neoplasms, triple-negative breast cancer

Introduction

Triple-negative breast cancer (TNBC) is an aggressive subtype of breast cancer that is characterized by the absence of the estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor 2 (HER2). TNBCs account for 10-17% of all breast cancers and are prone to recurrence [1]. Moreover, the annual incidence of TNBC has been steadily increasing, with some reports estimating it comprises up to 24% of new breast cancer diagnoses [2, 3]. Demographically, TNBC disproportionately affects African American women and younger patients. Compared to other breast cancer subtypes, it is more strongly associated with a BReast CAncer gene (BRCA)1 mutation, as more than 75% of breast cancers that develop in BRCA1 mutation carriers are triple-negative [4, 5]. The 5-year survival rate is much lower than that of any other breast cancer subtype, making TNBC one of the most aggressive subtypes and the most prone to metastasis [6].

The treatment approach for TNBC varies by stage. Early-stage disease is managed with surgery (breast-conserving surgery or mastectomy) followed by adjuvant chemotherapy for tumors larger than 10 mm [7, 8]. For locally advanced disease (stages II-III), neoadjuvant chemotherapy combined with pembrolizumab has become the standard of care, with pathologic complete response rates reaching approximately 64.8% with the combination versus 51.2% with chemotherapy alone [9-12]. Despite these advances, approximately 40% of patients with stage I-III TNBC experience recurrence after initial treatment cumulatively, with annualized locoregional and distant recurrence rates of approximately 3.8% and 3.1%, respectively, underscoring the critical need for effective post-treatment surveillance [13, 14].

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The current standard of care for post-treatment surveillance follows general breast cancer guidelines and includes regular physical examinations every 3-6 months for the first 3 years, every 6-12 months for years 4 and 5 and annually thereafter, along with annual mammography to monitor for new cancer in the remaining breast tissue [15, 16]. Routine imaging with computed tomography (CT) scans is not recommended unless patients develop symptoms suggestive of metastatic disease. Additionally, although tumor markers such as carcinoembryonic antigen (CEA) and Cancer Antigen (CA) 27.29 may be elevated in the setting of active or recurrent disease, they lack the sensitivity and specificity required for early detection of recurrence in TNBC and are not recommended for routine surveillance by current guidelines [15]. Once metastatic TNBC is detected through standard surveillance (clinical examination, mammography, and symptom-directed imaging), the prognosis is poor, with median survival after diagnosis of metastasis dropping to 12-18 months [17].

Therefore, given the limitations of conventional surveillance methods (i.e., clinical examination, mammography, and symptom-directed imaging), there is a growing interest in the potential role of [^{18}F]fluorodeoxyglucose positron emission tomography/computed tomography (FDG PET/CT) for post-treatment monitoring in TNBC patients. The biological characteristics of TNBC make it well-suited for metabolic imaging with this tool. TNBC cells exhibit higher glucose uptake compared to other breast cancer subtypes due to their rapid growth rate and aggressive nature, with previous studies showing that TNBC demonstrates higher FDG uptake than hormone receptor-positive tumors [18-21]. One of the advantages of metabolic imaging is that changes in cellular metabolism occur weeks to months before anatomical changes become visible on conventional imaging methods like CT or magnetic resonance imaging (MRI), potentially allowing for earlier detection of recurrence. Furthermore, whole-body imaging with PET/CT offers the advantage of detecting metastatic disease in unexpected sites which is beneficial because TNBC tends to metastasize to visceral organs and the brain. According to the recently updated joint EANM-SNMMI guideline on FDG PET/CT in breast cancer, FDG PET/CT is recommended for staging of locally advanced disease and may play a role in post-treatment surveillance, although further research and standardized imaging protocols are needed to define its optimal use in TNBC specifically [22].

In this review, we aim to evaluate the current evidence related to the use of FDG PET/CT in the post-treatment evaluation of TNBC. More specifically, this review examines the evidence for FDG PET/CT in two distinct post-treatment contexts in TNBC: (1) surveillance for recurrence after remission, focusing on the most common metastatic sites including hepatic, pulmonary, osseous, and cerebral disease, and (2) treatment response assessment in patients with advanced or metastatic TNBC undergoing chemotherapy or immunotherapy. These two questions are distinct. The first asks whether routine metabolic imaging can detect recurrence earlier than conventional surveillance in asymptomatic patients, and the second asks whether PET/CT can more accurately gauge whether an ongoing treatment is working. While most of the studies we reviewed included patients with TNBC as well as hormone receptor-positive breast cancer, we have attempted to emphasize the findings specific to TNBC in this review article. Baseline staging and the initial diagnostic work-up of newly diagnosed disease are considered only insofar as they inform post-treatment interpretation and are otherwise outside the scope of this review.

Post-treatment surveillance after remission

Hepatic metastases

The liver is one of the most common sites of metastatic disease in TNBC, with hepatic involvement occurring in approximately 13.7% of patients [23]. FDG PET/CT performs well in detecting hepatic metastases in TNBC patients because of the metabolic characteristics of both the tumor and the normal liver. Normal liver parenchyma has relatively low baseline glucose uptake which in turn results in metabolically active metastases appearing as areas of increased FDG uptake. In a study evaluating 82 patients with various malignancies including 27 (not exclusively TNBC) breast cancer patients, FDG PET/CT showed 98% sensitivity and 100% specificity for detecting hepatic metastases. These figures, however, derive from a mixed-malignancy cohort in which breast cancer patients were a minority and no separate TNBC analysis was reported, and should therefore be regarded as extrapolated and not as TNBC-specific. It outperformed CT alone which showed 95% sensitivity and 81% specificity [24]. More specifically, for TNBC, FDG PET/CT was able to upstage to stage IV in 30 of 232 newly diagnosed TNBC patients and was able to detect unsuspected metastases, with 8 of those metastases being in the liver and these metastases were confirmed by pathology and/or follow-up imaging [25].

Pulmonary metastases

The lungs present a more complex challenge for FDG PET/CT surveillance. Pulmonary metastases develop in 36.9% of TNBC patients who experience recurrence [26]. Small lung nodules, particularly those under 8-10 mm, frequently do not demonstrate sufficient glucose uptake to be reliably detected by PET imaging and therefore PET has decreased sensitivity because of the partial volume effect and respiratory movements. The lungs are also more prone to inflammatory processes and infections that can cause false-positive findings on PET scans. These inflammatory cells are metabolically active and therefore can consume glucose, making it difficult to distinguish between a benign inflammatory nodule and actu-

al metastatic disease based on metabolic activity alone. In a head-to-head comparison of [^{18}F]PSMA-1007 and [^{18}F]FDG PET/CT in 10 TNBC patients, two patients had uncountable lung metastases, where larger metastatic lesions above 5 mm demonstrated detectable FDG uptake. Absolute SUV values vary with scanner type, blood glucose level, and reconstruction parameters, so lesion detectability, and not specific SUV thresholds, is of primary clinical relevance. In one of these patients, FDG demonstrated a maximum standardized uptake value (SUV_{max}) of 3.8 with a target to background (TBR) ratio of 9.5 for lung metastasis. Another patient presented with a larger (>20 mm) pulmonary metastatic nodule that showed high FDG uptake with an SUV_{max} of 12.0 and TBR of 20.0. A solitary pleural metastatic lesion with diameter of 10 mm had an SUV_{max} of 6.1 with TBR of 7.6 [27]. These studies demonstrate that FDG uptake varies considerably based on lesion size, even among confirmed metastases. However, the role of FDG PET/CT in detecting pulmonary metastases specifically in TNBC requires further investigation, as studies addressing this topic remain scarce. More evidence is needed before definitive conclusions can be drawn about its utility as a surveillance tool for lung metastases in TNBC.

Osseous metastases

Bone is among the most common sites of distant metastasis in breast cancer, though TNBC tends to metastasize to visceral organs more frequently than to bone compared with hormone receptor-positive subtypes [28-30]. Nevertheless, osseous metastases do occur in TNBC and present unique challenges for FDG PET/CT imaging. Osteolytic lesions, which are more common in TNBC, typically demonstrate high FDG avidity and are readily detected on PET/CT [31-34]. However, sclerotic or mixed lesions, which may develop after treatment, can be more difficult to characterize, as the metabolic activity may not correlate directly with disease burden [32, 35]. FDG PET/CT has demonstrated advantages over conventional bone scintigraphy in detecting lytic bone metastases, as bone scans rely on osteoblastic activity and may underestimate the extent of purely lytic disease [36, 37]. Additionally, [^{18}F]sodium fluoride (NaF) PET/CT has demonstrated superior sensitivity compared to conventional bone scintigraphy for detecting sclerotic bone lesions due to its higher spatial resolution and uptake at sites of active bone remodeling [38, 39]. In the context of post-treatment surveillance, FDG PET/CT can help distinguish active osseous disease from post-treatment sclerotic healing, providing information about treatment response in bone. However, the flare phenomenon, which is a transient increase in FDG uptake in healing bone lesions shortly after initiating therapy, must be considered to avoid false-positive interpretations [36, 40].

Brain metastases

Brain metastases are a major clinical challenge in TNBC, with an incidence of approximately 25-46% over the course of the disease, considerably higher than in other breast cancer subtypes [29, 41, 42]. Despite this clinical importance, FDG PET/CT has well-recognized limitations in evaluating brain metastases due to the high physiological glucose metabolism of normal brain parenchyma, which creates a high background signal that can obscure small metastatic deposits [43, 44]. Consequently, contrast-enhanced MRI remains the gold standard for the detection and monitoring of brain metastases in TNBC patients [43, 44]. FDG PET/CT may occasionally detect large or highly metabolically active brain lesions incidentally during whole-body staging, but it should not be relied upon for systematic brain surveillance. Emerging PET tracers with lower background brain uptake, and dedicated brain PET protocols, may improve the detection of cerebral metastases in the future, but currently, MRI should complement FDG PET/CT for comprehensive post-treatment surveillance in TNBC patients at high risk for brain metastases.

Treatment response assessment in advanced/metastatic disease

Response assessment: PERCIST versus RECIST

Accurate evaluation of therapeutic response in metastatic TNBC relies on the selection of an appropriate response assessment method, as this directly influences interpretation of treatment efficacy. The standard approach has been Response Evaluation Criteria in Solid Tumors (RECIST) 1.1, which measures tumor size changes on CT or MRI imaging. The RECIST approach relies exclusively on anatomic imaging to quantify structural tumor changes, determining response based on whether lesions increase, decrease, or remain stable in size [45]. However, RECIST has limitations when it comes to TNBC, especially after treatment.

Residual lesions following therapy may consist of necrotic or inflammatory tissue and not active malignancy; however, RECIST cannot differentiate viable tumor from non-viable tissue. Studies have shown that PET Response Criteria in Solid Tumors (PERCIST) offers advantages over traditional RECIST criteria [46]. Instead of solely focusing on tumor size, PERCIST measures glucose consumption by cancer cells using FDG PET/CT, which provides both anatomical and functional metabolic data. Given the highly proliferative and aggressive nature of TNBC, early identification of metabolic alterations provides essential insight into therapeutic response [47]. In a study conducted by Riedl et al. involving 65 patients with metastatic breast cancer, 40% of patients who were classified as non-responders by RECIST 1.1 criteria were metabolic responders when evaluated using PERCIST. PERCIST also showed higher predictive accuracy for both progression-free survival (0.70 vs 0.60) and disease-specific survival (0.65 vs 0.55) compared to RECIST 1.1 [48]. For TNBC specifically, this is worth exploring because the metabolic data are especially important given the aggressive and

metabolically active nature of TNBC, and it will complement the anatomical information from RECIST 1.1 to give a more complete picture of treatment response. The cohort studied by Riedl et al. comprised metastatic breast cancer of mixed receptor subtypes and did not report outcomes separately for the TNBC subgroup; the superiority of PERCIST over RECIST in this setting is therefore inferred for TNBC and has not been directly demonstrated.

In addition to PERCIST, complete metabolic response is one of the most important indicators of how effectively treatment is working in all types of metastatic breast cancer including TNBC. When FDG PET/CT shows a complete metabolic response, it means the tumor's glucose uptake has dropped down to background levels (there is no evidence of metabolically active disease left). However, the aggressive nature of TNBC makes it less likely that patients will achieve this level of response. In a study of 570 metastatic breast cancer patients treated between 2003 and 2005, only seven out of 90 patients (8%) who achieved no evidence of disease had TNBC, which is a small subgroup that limits the generalizability of these findings to TNBC specifically. Nevertheless, among these 90 patients, in a Fine-Gray competing-risks analysis of time to no evidence of disease (NED), TNBC was significantly associated with a lower likelihood of achieving NED, with a reported subdistribution hazard ratio (HR) of 0.32 (P = 0.005), indicating that TNBC patients had a significantly decreased likelihood of achieving NED status [49]. A study of interim PET/CT during neoadjuvant chemotherapy confirmed this pattern, showing that TNBC patients demonstrate lower rates of complete metabolic responses compared to hormone receptor-positive or HER2-positive tumors. Despite this, patients who achieved early complete metabolic response had 5-year overall survival rates of 80%, which is lower than the 92% seen in ER-positive/HER2-negative tumors with similar responses. Nevertheless, achieving early complete metabolic response was associated with improved overall survival even in TNBC, underscoring the value of FDG PET/CT as a tool for treatment response assessment in this subtype [50]. A related and increasingly important scenario is oligometastatic disease, in which a limited number of metastases are treated with local ablative approaches such as stereotactic radiotherapy or surgical resection [51, 52]. Whether a PET-defined complete metabolic response after such therapy predicts durable long-term outcomes in TNBC has not been established, and dedicated studies correlating metabolic response with progression-free and overall survival in oligometastatic TNBC are needed.

Predicting pathologic complete response after neoadjuvant chemotherapy

FDG PET/CT is useful for evaluating treatment response in TNBC patients after chemotherapy. FDG PET/CT yields important prognostic information that can guide future clinical decisions after treatment. In a study of 186 patients with locally advanced breast cancer, including 23% with TNBC, post-chemotherapy FDG PET/CT performed 2 weeks after completion of neoadjuvant chemotherapy (NAC) showed strong correlation with pathologic complete response [53]. TNBC had the highest pathologic complete response (pCR) rate of 32.9% among all subtypes of breast cancer. Goktas Aydin et al. found that an SUV_{max} cutoff of 1.1 after NAC predicted pCR with an area under the curve of 0.784, and PET/CT showed sensitivity of 100%, specificity of 72%, accuracy of 85%, and negative predictive value of 100%. Patients who achieved this complete metabolic and anatomic response had better 3-year disease-free survival rates of 90% compared to 70% in those with only partial response. TNBC was also an independent predictor of pCR with an odds ratio of 4.38 [53]. Early interim FDG PET/CT imaging during treatment has also shown value in identifying non-responders who may benefit from switching therapies. In a study performed by Groheux et al. involving 20 TNBC patients, performing FDG PET/CT after 2 cycles of chemotherapy showed FDG PET/CT to be of predictive value. Participants with less than a 42% decrease in SUV_{max} after 2 cycles had a 100% risk of having residual tumor at time of surgery compared to only 45% in patients who showed better metabolic response. The risk of early relapse within 2 years was 44% in these non-responders versus 0% in responders. The mean change in SUV_{max} was -73% in patients who achieved pathologic complete response versus only -33% in those who didn't [54]. **Figure 1** highlights a 53-year-old patient with TNBC who had an absence of residual tumor cells in the breast following chemotherapy. This early assessment offers a window to change treatment strategies in patients whose tumors are not responding to the planned chemotherapy regimen, which is important given the aggressive nature of TNBC. Two caveats temper these conclusions. First, the study by Groheux et al. was small (n = 20). Second, it was conducted before platinum-based and pembrolizumab-containing regimens became standard for locally advanced TNBC, so its specific SUV_{max} thresholds require revalidation in the current treatment era. More recent TNBC-specific evidence has begun to address this gap: in a bicentric study of 191 patients with early-stage TNBC treated with neoadjuvant chemotherapy with or without pembrolizumab, Seban et al. found that a high baseline tumor SUV_{max} and a low total metabolic tumor volume independently predicted pathologic complete response in both the chemotherapy-alone and chemo-immunotherapy cohorts, albeit at different thresholds, with pathologic complete response rates of 53% and 70%, respectively [55]. This underscores that optimal metabolic cut-offs are regimen-dependent and must be defined separately for immunotherapy-containing protocols.

Timing considerations after chemotherapy

Timing of imaging is an important consideration when using FDG PET/CT after chemotherapy. Chemotherapy

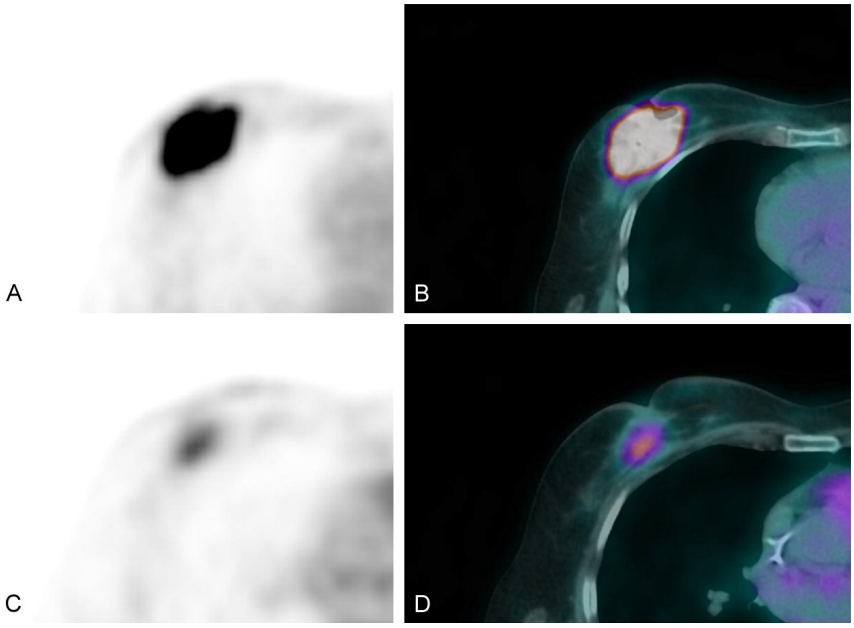

Figure 1. Early interim FDG PET/CT response in a 53-year-old woman with triple-negative breast cancer (80-mm grade 3 invasive ductal carcinoma of the right breast; T4N1M0, stage IIIB). A, B. Transaxial PET and fused PET/CT at baseline, showing an intensely FDG-avid primary tumor (SUV_{max} = 23.3). C, D. Corresponding images after two cycles of neoadjuvant chemotherapy, with markedly reduced residual uptake (SUV_{max} = 3.7; ΔSUV = -84%). The patient completed chemotherapy (two additional cycles of epirubicin [75 mg/m^2] plus cyclophosphamide [750 mg/m^2], followed by four cycles of docetaxel [100 mg/m^2]) and achieved a pathologic complete response at mastectomy, with no residual invasive tumor in the breast and nine tumor-negative axillary lymph nodes. Reproduced with permission from Groheux D, et al. J Nucl Med 2012; 53(2): 249-254. © SNMMI [54].

has been shown to cause inflammation that results in increased FDG uptake on PET scans, and this can persist for months after the end of treatment. Specifically, elevations in inflammatory markers remain statistically significant at 6, 12, and 18 months post-treatment in patients who received both chemotherapy and radiation [56]. Quantitative SUV values can remain elevated in inflammatory tissue, making it difficult to distinguish between true residual cancer and the inflammatory response related to medical treatment. The inflammatory response to treatment involves activation of macrophages and other immune cells which results in increased glucose uptake similar to cancer cells, with preclinical studies demonstrating that activated macrophages can exhibit up to a sixfold increase in glucose uptake compared to resting macrophages [57].

Because of these potential false positives, most experts and current data recommend waiting 8-12 weeks after completion of chemotherapy prior to performing FDG PET/CT scan to evaluate for post-treatment response [22, 58-60]. This waiting period gives the treatment-related inflammation time to normalize to obtain a more accurate determination of treatment response. Alternatively, dual-time-point (delayed) FDG PET imaging may help differentiate residual tumor from post-treatment inflammation, as malignant lesions tend to show increasing FDG uptake over time while inflammatory uptake typically decreases on delayed images [32]. Furthermore, the use of alternative PET tracers with lower susceptibility to inflammatory confounding, such as FAPI or FLT, may also help overcome this limitation. Similar caution applies after locoregional radiotherapy, which can induce FDG-avid inflammatory changes in the chest wall, breast, and regional nodes that persist for months and may be misinterpreted as residual or recurrent disease [61]. When dual-time-point imaging is employed, acquiring a delayed scan (typically around three hours after injection in addition to the standard one-hour acquisition) and calculating the percentage change in SUV_{max} between the two time points (the retention index) can aid differentiation, because malignant lesions tend to show a rising retention index whereas inflammatory foci more often plateau or decline; the prospective dual-time-point data reported by Hildebrandt et al. provide a practical reference for applying this approach in suspected recurrent breast cancer [32].

Post-immunotherapy response assessment

FDG PET/CT is also used to monitor TNBC patients receiving immunotherapy with agents such as checkpoint inhibitors. Beyond immune checkpoint inhibitors, antibody-drug conjugates (ADCs) such as sacituzumab govitecan and datopotamab deruxtecan have become increasingly important treatment options in metastatic TNBC, and response assessment with these agents presents unique considerations for FDG PET/CT interpretation, including treatment-induced inflammation at responding lesions. According to joint EANM/SNMMI/ANZSNM practice guidelines, FDG PET/CT is recommended at interim time points, commonly 8-12 weeks (3-4 cycles) after the treatment starts. The FDG PET/CT scan can also be performed earlier or later during treatment in cases of suspicious progression on a contrast-enhanced CT. For patients receiving maintenance therapy or undergoing long-term treatment with immune checkpoint inhibitors, FDG PET/CT can also be used to assess metabolic response, particularly in partial responders or those with stable disease on CT imaging. FDG PET/CT restaging is also recommended before restarting treatment to reestablish a new baseline for subsequent response assessment.

However, current guidelines acknowledge that FDG PET should be included in prospective randomized clinical

trials to determine whether FDG PET responses can predict the effectiveness of immunotherapy more accurately than response assessment by RECIST criteria [62]. Despite its utility, there are important considerations when using FDG PET/CT during and after immunotherapy. As with the chemotherapy-related inflammation described above, checkpoint inhibitors activate immune cells whose heightened glucose uptake can mimic malignancy on FDG PET. Studies using radiolabeled pembrolizumab in humanized mice have shown that the antibody distributes primarily to lymphoid organs where PD-1 expressing immune cells are present, including in the spleen, lymph nodes, bone marrow, and thymus. These activated immune cells cause increased FDG uptake in many normal tissues and not only in localized tumor regions, which makes interpretation difficult [63]. One of the most important considerations is pseudoprogression, a phenomenon where the disease appears to become initially worse but is responding to treatment. This occurs because of initial recruitment of immune cells at tumor sites, and tumors can appear larger, or more active, on FDG PET scans in the first months of treatment. Scanning too early can lead to incorrect conclusions about treatment failure potentially leading providers to prematurely discontinue effective therapy. The general recommendation from current guidelines is that if there is uncertainty regarding progression or pseudoprogression, especially after first post-treatment evaluation, performing a confirmatory follow-up FDG PET/CT study 4 to 8 weeks later is recommended to assess immunotherapy response in TNBC patients [62]. However, no single response framework has been validated specifically for TNBC treated with checkpoint inhibitors. Immune-adapted metabolic criteria such as iPERCIST and PERCIMT were developed largely in melanoma and lung cancer to reduce the misclassification of pseudoprogression, typically by requiring confirmation of apparent progression on a subsequent scan; whether they outperform standard PERCIST in TNBC is unknown, and the four-to-eight-week confirmatory interval is likewise extrapolated from other tumor types and has not been derived from TNBC-specific data [64, 65].

Emerging PET tracers beyond FDG

While FDG remains the dominant PET radiotracer in oncology, its inability to distinguish tumor from inflammation and its high background brain uptake have motivated investigation of alternative tracers relevant to TNBC. [^{18}F] fluorothymidine (FLT), a marker of cellular proliferation and not of glucose metabolism, is less susceptible to inflammatory false positives and can detect proliferative changes within days of initiating chemotherapy, though it has lower overall sensitivity for metastasis detection than FDG [66, 67]. Prostate-specific membrane antigen (PSMA)-targeted tracers, which bind neovasculature in various solid tumors, have been directly compared to FDG in TNBC by Andryszak et al., who found FDG more sensitive overall but PSMA PET complementary for characterizing certain bone lesions [27]. Fibroblast activation protein inhibitor (FAPI)-targeted PET tracers target cancer-associated fibroblasts (CAFs) in the tumor microenvironment. FAPI PET demonstrates minimal background uptake in normal tissue, including the brain, which directly addresses one of the most important limitations of FDG in TNBC, and it is less confounded by post-treatment inflammation [68-70].

These tracers remain investigational, and clinical experience in TNBC specifically is limited. However, multi-tracer protocols combining FDG with one or more complementary agents may ultimately provide more comprehensive post-treatment metabolic characterization than any single tracer alone, and their evaluation in TNBC-specific prospective trials should be a research priority. A more direct FDG-versus-FAPI comparison is instructive. Because fibroblast activation protein is expressed by cancer-associated fibroblasts and not by tumor cells, FAPI uptake is largely independent of glucose metabolism and of blood glucose, exhibits very low physiological background in the liver and brain, and appears less confounded by post-treatment inflammation, all of which are advantages precisely in the settings where FDG performs least well; conversely, FDG retains higher lesion-level intensity in many avid TNBC deposits and is supported by a far larger evidence base. Other receptor-targeted tracers are relevant chiefly through the lens of tumor heterogeneity. 16-alpha-[^{18}F]fluoro-17-beta-estradiol (FES), which images estrogen-receptor expression, has limited direct application in receptor-negative TNBC but may help characterize discordant or heterogeneous lesions and mixed tumors [71]; similarly, HER2-targeted PET agents are not applicable to HER2-negative disease as classically defined, yet the emergence of the HER2-low category and the activity of HER2-directed antibody-drug conjugates in this setting have renewed interest in non-invasive receptor mapping [72, 73]. These agents are therefore best viewed as complementary problem-solving tools and not as replacements for FDG in TNBC.

PET-based global disease quantification

Beyond lesion-level assessment, PET-based global disease quantification is an emerging paradigm for evaluating total disease burden and therapeutic response in metastatic breast cancer, including TNBC. Traditional PET interpretation focuses on individual lesion SUV values; however, whole-body metabolic parameters such as total metabolic tumor volume (MTV) and total lesion glycolysis (TLG) capture the aggregate metabolic activity across all disease sites, providing a more comprehensive measure of disease burden [74-76]. Software platforms such as ROVER (ABX GmbH, Radeberg, Germany) now enable semi-automated, reproducible global quantification of PET data, making such analyses feasible for clinical research and, increasingly, for routine practice [77]. The advent of total-body PET scanners, which offer extended

axial field of view covering the entire body in a single acquisition, has further enhanced the potential of global PET quantification by enabling simultaneous imaging of all metastatic sites with improved sensitivity and signal-to-noise ratio. Total-body PET facilitates more accurate measurement of whole-body metabolic parameters and may detect low-grade metastatic deposits that would be missed by conventional PET/CT with limited axial coverage [78]. For TNBC, where metastatic disease is often multifocal and involves diverse organ sites, PET-based global quantification may be useful for monitoring treatment efficacy and guiding therapeutic decisions.

Limitations

While this review suggests that FDG PET/CT has potential to monitor TNBC after treatment, there are several limitations that should be acknowledged. One of the most important limitations is the need for larger studies focusing specifically on TNBC; it is important to have larger TNBC-specific prospective studies validate today's preliminary findings. Although FDG PET/CT is already incorporated into existing guidelines for staging and response assessment in breast cancer, its cost-effectiveness as a routine surveillance tool in TNBC remains to be established. The relative cost of FDG PET/CT compared to other imaging modalities such as MRI varies across institutions and healthcare systems, and formal cost-effectiveness analyses specific to TNBC surveillance are lacking. Without further studies focusing on cost-effectiveness and improved outcomes to justify the extra expense that comes with FDG PET/CT imaging, widespread use of PET-based surveillance may not be realistic in routine clinical use. Furthermore, most of the studies included in this review have short follow-up periods, usually 2 to 3 years at the most. Since TNBC can recur beyond that timeframe, longer-term studies are needed to clarify FDG PET/CT's role in detection of late recurrences and its impact on long-term survival. Finally, FDG PET/CT does not operate in isolation; emerging modalities such as circulating tumor DNA (ctDNA) analysis are being developed for minimal residual disease detection and may serve as complementary tools to metabolic imaging in future TNBC surveillance protocols. Knowing this, future research addressing these gaps is imperative to establish evidence-based guidelines on how to best use FDG PET/CT in TNBC management. Direct, TNBC-specific evidence also remains sparse for several of the applications discussed here. A recent systematic review of PET imaging in TNBC identified no published study evaluating FDG PET/CT for the detection of recurrence in TNBC specifically, and only limited data in the metastatic setting, meaning that much of the surveillance rationale presented here is necessarily extrapolated from mixed breast cancer cohorts [79]. Correspondingly, although the treatment-context literature cited here is current, the imaging-evidence base for TNBC response assessment in the immunotherapy era is still maturing and warrants dedicated prospective study.

Conclusion

FDG PET/CT offers clear but uneven value across the post-treatment evaluation of TNBC, and recognizing where it helps, and where it does not, is central to its appropriate use. In the surveillance setting, the strongest case is for detection of hepatic metastases, where low baseline uptake in normal liver parenchyma yields a favorable tumor-to-background ratio and high reported sensitivity and specificity. In the response-assessment setting, the strongest case is for early metabolic evaluation using PERCIST criteria, which can identify non-responders before anatomical changes emerge on conventional imaging and appears to outperform RECIST 1.1 for predicting progression-free and disease-specific survival.

These advantages are not uniform across metastatic sites or clinical scenarios. FDG PET/CT performs poorly for brain metastasis surveillance because of high physiological cortical glucose uptake, and its sensitivity for sub-centimeter pulmonary nodules is limited by partial volume effects and respiratory motion. Post-treatment inflammation and immunotherapy-related pseudoprogression further complicate interpretation, making scan timing (typically 8 to 12 weeks after chemotherapy completion) and confirmatory follow-up imaging essential when findings are equivocal.

Translating these observations into evidence-based guidelines will require TNBC-specific prospective studies with standardized PERCIST-based response assessment, follow-up beyond three years to capture late recurrences, and formal cost-effectiveness analyses benchmarked against conventional surveillance. Complementary approaches also require dedicated evaluation in this subtype and should not be extrapolated from mixed breast cancer cohorts. These approaches include non-FDG tracers such as FLT, FAPI, and PSMA-targeted agents for sites where FDG underperforms, and PET-based whole-body metabolic quantification for multifocal disease. Addressing these gaps will determine whether FDG PET/CT becomes a routine component of TNBC management or remains a selectively applied tool.

Disclosure of conflict of interest

None.

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