

2-^[18F]FLUORO-2-DEOXY-D- GLUCOSE PET IN BRAIN TUMORS

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SUMMARY

[¹⁸F]FDG PET remains an important and widely available molecular imaging tool in neuro- oncology despite the availability of alternative PET tracers. Its widespread availability and ability to evaluate both primary brain involvement and systemic disease make it particularly valuable in the assessment of PCNSL, as well as in selected cases of gliomas, brain metastases, and pediatric brain tumors. In contrast, its role in meningiomas remains limited. Future developments, including artificial intelligence-based imaging analyses, may further enhance the role of molecular imaging in brain tumors, including the role of [¹⁸F]FDG PET.⁵²

KEY POINTS

- In gliomas, [¹⁸F]FDG PET can help assess tumor aggressiveness, guide biopsy, and evaluate residual or recurrent disease, although its value is limited by high physiologic brain uptake and lower accuracy than other PET tracers.
- In brain metastases, [¹⁸F]FDG PET may assist in differentiating tumor recurrence from treatment- related changes, although other PET tracers show higher diagnostic accuracy.
- In lymphomas, [¹⁸F]FDG PET plays a well-established role in initial diagnosis and may also provide significant value in treatment monitoring and management.
- In meningiomas, [¹⁸F]FDG PET has limited clinical value. Instead, PET imaging using somatostatin receptor ligands is preferred for tumor characterization.
- [¹⁸F]FDG PET can provide useful diagnostic information in pediatric central nervous system malignancies, although interpretation may be limited by high physiologic uptake.

ABBREVIATIONS

- [^{18}F]FDG 2- [^{18}F]fluoro-2-deoxy-D-glucose
- BBB blood-brain barrier
- CNS central nervous system
- FDA Food and Drug Administration
- GLUTs glucose transporters
- HD-MTX high-dose methotrexate-based chemotherapy
- IDH isocitrate dehydrogenase
- PCNSL primary central nervous system lymphomas
- SSTR somatostatin receptors
- TBRs tumor-to-contralateral-normal-brain
- SUV ratios
- WHO World Health Organization
- WHO fifth edition of the World Health
- CNS5 Organization Classification of
- Tumors of the Central Nervous System

INTRODUCTION

Brain tumors represent a major public health concern, as they constitute a significant cause of morbidity and mortality.¹ They encompass a broad spectrum of primary and secondary neoplasms involving the central nervous system (CNS).

Among primary brain tumors in adults, glioblastoma is the most common malignant tumor, whereas meningioma represents the most frequently diagnosed nonmalignant intracranial tumor.² Brain metastases occur approximately 10 times more frequently than primary brain tumors and account for 9% to 10% of all cancer diagnoses.³

MR imaging is the main imaging modality for the diagnosis and follow-up of brain tumors,⁴ due to its high sensitivity and specificity, excellent soft-tissue contrast, and relatively wide availability. However, PET provides complementary information by enabling the in vivo assessment of tumor biology beyond the structural information obtained with MR imaging. PET then can be used for differential diagnosis, tumor grading, delineation of tumor extent, surgical and radiotherapy planning, as well as posttreatment monitoring and prognostic assessment. A key advantage of PET compared with conventional MR imaging is that tracer uptake reflects specific pathophysiological processes and, in most cases, is largely independent of disruption of the blood-brain barrier (BBB),

whereas contrast-enhanced MR imaging sequences rely on BBB breakdown.⁵ Among PET radiotracers, 2- ^{18}F fluoro-2-deoxy-D-glucose (^{18}F FDG) is the most widely used, but many other tracers are clinically available such as amino acid analogs (AA PET) or radiolabeled somatostatin receptor (SSTR) ligands.⁵⁻⁷ However, it is also important to note that in the United States, no AA radiopharmaceuticals are Food and Drug Administration (FDA)-approved for clinical use. Thus, ^{18}F FDG is still the primary radiopharmaceutical for brain imaging, including for tumors.

Notably, a recent survey conducted by the European Organisation for Research and Treatment of Cancer–Brain Tumor Group reported that only 35% of participating centers used ^{18}F FDG PET in neuro-oncology, mainly for the evaluation of lymphoma and extracranial disease in patients with brain metastases. Nevertheless, in certain settings, ^{18}F FDG PET continues to be used because of the limited availability of AA PET, particularly in the United States.^{8,9}

Management of brain tumors is multimodal, with treatment strategies guided by tumor histology and preoperative clinical considerations.⁴ Therapeutic options include surgery, which remains the cornerstone of management, as well as radiation therapy, including stereotactic radiosurgery and conventional fractionated approaches. Additional treatments may involve corticosteroids, intravenous chemotherapy, or intrathecal chemotherapeutic agents, while novel therapies, including chemotherapy and immunotherapy agents, are continuously developed. Importantly, these therapeutic interventions can induce posttreatment changes within the tumor and surrounding brain parenchyma that must be carefully considered during neuroimaging assessment.¹⁰

Among brain tumors, the clinical role of ^{18}F FDG PET is not uniform. Although its value is well established in primary central nervous system lymphoma (PCNSL), its application in other brain tumors remains more selective and is generally complementary to MR imaging and other PET radiotracers. This review, therefore, provides an overview of the current role of ^{18}F FDG PET in the evaluation of brain tumors.

BIOLOGICAL BASIS OF ^{18}F FDG PET IN BRAIN TUMORS

^{18}F FDG PET imaging is based on the increased glycolytic activity of tumor cells, a phenomenon known as the Warburg effect. Malignant cells preferentially use glycolysis rather than oxidative phosphorylation for energy production, even in the presence of oxygen.¹¹ ^{18}F FDG is a glucose analog transported across the BBB by glucose transporters (GLUTs), mainly GLUT1 and GLUT3, independent of BBB disruption. Once inside the cell, ^{18}F FDG is phosphorylated by hexokinase and then trapped intracellularly, allowing visualization with PET imaging.¹²

The main limitation of ^{18}F FDG PET in brain tumors assessment is the high physiologic uptake in normal gray matter, which can reduce lesion contrast and may make difficult to detect tumors with moderate metabolic activity, particularly low-grade gliomas.⁵ In addition, ^{18}F FDG uptake is not tumor-specific: inflammatory or infectious processes may also show increased uptake, while necrotic regions in high-grade tumors may exhibit reduced uptake.¹⁰ As ^{18}F FDG PET reflects glucose

metabolism, it requires a fasting period of 4 to 6 hours, and factors such as hyperglycemia or insufficient fasting period can decrease the sensitivity of the examination.^{13,14}

[¹⁸F]FDG PET IN GLIOMAS

In 2021, the fifth edition of the World Health Organization Classification of Tumors of the Central Nervous System (WHO CNS5) was published. This updated classification introduced major changes, emphasizing the growing role of molecular diagnostic markers alongside histology and immunohistochemistry in CNS tumor classification.¹⁵

Adult-type diffuse gliomas are currently classified into 3 main entities: astrocytoma isocitrate dehydrogenase (IDH) mutant; oligodendroglioma IDH- mutant and 1p/19q-codeleted; and glioblastoma IDH-wildtype. Accurate molecular characterization of these tumors is essential, as prognosis and therapeutic strategies are strongly influenced by their genetic profile.¹⁵

Whenever available, amino acid PET tracers are generally preferred over [¹⁸F]FDG PET because of their superior tumor-to-background contrast and higher diagnostic accuracy, particularly for tumor delineation and treatment response assessment.¹⁶ Nevertheless, [¹⁸F]FDG PET remains clinically useful because of its wide availability and its ability to distinguish high-grade (WHO grade III/IV) gliomas from other malignant brain tumors. However, its specificity remains limited due to overlapping SUV_{max} values across tumor types and its reduced ability to differentiate gliomas from nonmalignant conditions, such as inflammatory or infectious diseases.¹⁷ In the United States, [¹⁸F]FDG PET is currently the only FDA- approved radiotracer for the diagnosis of brain tumors.

High-grade gliomas generally demonstrate greater [¹⁸F]FDG uptake than low-grade gliomas (WHO grade I/II),¹⁷ as illustrated in **Fig. 1**. Moreover, increased [¹⁸F]FDG uptake has been identified as an independent prognostic factor in WHO grade IV gliomas, both at diagnosis and after treatment initiation.¹⁸

Tumor-to-contralateral-normal-brain SUV ratios (TBRs) may further improve tumor grading. However, no clear threshold has been consistently established in the literature. A commonly proposed cutoff is a TBR_{max} of 0.6, although higher thresholds, such as 0.9, have also been suggested.^{19–21} Nevertheless, some low-grade gliomas may exhibit elevated TBR values, limiting the accuracy of [¹⁸F] FDG PET for tumor grading at diagnosis.

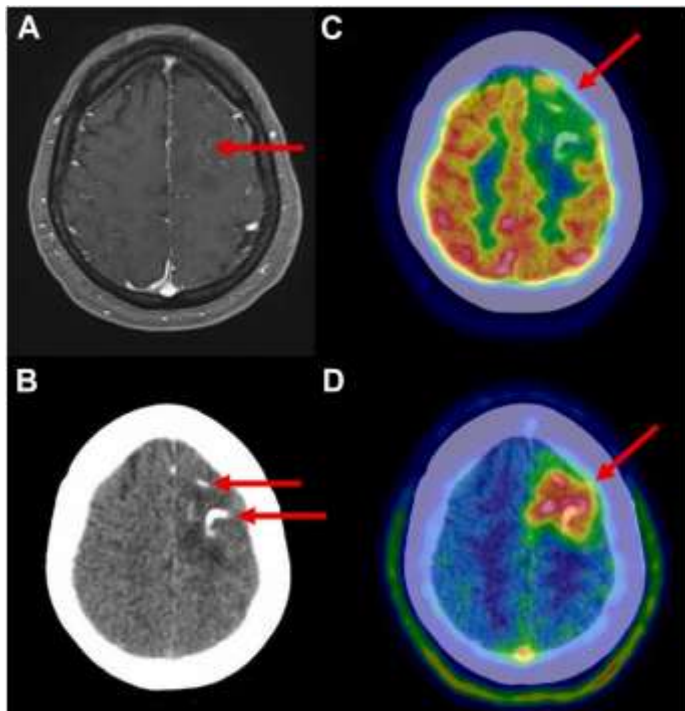
Delayed imaging, performed more than 3 hours after tracer injection, has been suggested to improve the specificity of [¹⁸F]FDG PET, as tracer uptake tends to decrease slightly over time in normal brain tissue while remaining stable or increasing in malignant lesions.^{5,22} However, the implementation of such delayed protocols remains challenging in the routine clinical setting.

[¹⁸F]FDG PET can be a valuable tool for guiding stereotactic brain biopsies by identifying regions with the highest uptake, which often correspond to higher grade tumor areas. AA PET tracers typically provide superior contrast between tumor and normal brain tissue; however, a small proportion of

gliomas may show little to no amino acid uptake.^{5,23} In such cases, [¹⁸F]FDG PET can be used to identify suitable biopsy targets.

Pseudoprogression and radionecrosis are recognized as treatment-related changes occurring within the first 3 months after treatment and beyond 3 months, respectively, with pseudoprogression potentially extending up to 6 months in patients receiving immunotherapy.²⁴ Differentiating tumor recurrence or progression from treatment-related changes remains challenging. The reported sensitivity and specificity of [¹⁸F] FDG PET for distinguishing high-grade glioma progression from treatment-related changes vary widely in the literature.¹³ Specificity may be limited by inflammatory processes, particularly in the early period following surgery and/or radiation therapy.¹⁹ Whenever available, previous PET examinations should be used for comparison during interpretation. In the absence of treatment, tumor uptake is expected to increase between 2 PET scans, whereas lesion uptake is expected to decrease after treatment.¹³

Figure 1.



(A–D) Brain images of a 39 year old patient with a left frontal lesion. (A) Postgadolinium T1-weighted MR imaging shows mild enhancement after gadolinium injection (*red arrow*). (B) CT scan of the PET/CT scan shows calcifications (*red arrows*), characteristic of oligodendroglioma. (C) [¹⁸F]FDG PET/CT scan shows a hypometabolic lesion, with a SUV_{max} 5.6 slightly higher than white matter and lower than gray matter. TBR_{max} was estimated at 0.5 suggesting a low-grade glioma. (D) [¹⁸F]FET PET/CT scan allowed to better estimate the extent of the lesion. TBR_{max} was estimated at 4.5, suggesting a high-grade glioma. The pathologic diagnosis was an oligodendroglioma WHO grade 2, IDH-mutant (IDH1 p.R132 mutation), and 1p/19q-codeleted. The [¹⁸F]FDG PET TBR_{max} correctly predicted the low-grade glioma whereas [¹⁸F]FET TBR_{max} overestimated it. (Nadia Withofs et al., Facts and Fictions About [18F]FDG versus Other Tracers in Managing Patients with Brain Tumors: It Is Time to Rectify the Ongoing Misconceptions, *PET Clinics*, 17 (3), 2022, 327-342, <https://doi.org/10.1016/j.cpet.2022.03.004>.)

[¹⁸F]FDG PET IN BRAIN METASTASES

The differential diagnosis between brain metastases and other malignant brain tumors remains challenging with PET imaging, as [¹⁸F]FDG PET typically demonstrates high uptake in both brain metastases and gliomas.⁵ Moreover, brain metastases arising from primary tumors with typically low [¹⁸F]FDG uptake are not optimally evaluated with this tracer. In such cases, the presence of a focal “cold spot,” reflecting central necrosis and/or extensive surrounding edema, particularly in larger lesions, may nonetheless suggest a metastatic lesion.

Consequently, [¹⁸F]FDG PET is not recommended for the initial diagnosis of brain metastases, for which MR imaging remains the reference technique.²⁵ Similarly, systematic brain acquisition for the detection of unsuspected brain metastases in patients with cancer undergoing whole-body [¹⁸F]FDG PET provides only limited additional value.²⁶

Assessment of treatment response in brain metastases must consider the status of the primary extra-CNS disease. Management decisions should, therefore, consider systemic tumor burden before any modification of treatment strategies is implemented.⁵

Radionecrosis following irradiation of brain metastases is relatively common, occurring in 25% of cases.⁵ In this context, contrast-enhanced MR imaging does not always reliably differentiate radiation-induced changes from recurrent brain metastases. The diagnostic performance of [¹⁸F]FDG PET for differentiating recurrent brain metastases from treatment-related changes varies substantially across studies. Overall, [¹⁸F]FDG PET demonstrates lower diagnostic accuracy than AA PET in this setting but also than MR imaging- based perfusion imaging using arterial spin labeling, MR spectroscopy, and diffusion-weighted imaging.²⁵

Nevertheless, [¹⁸F]FDG PET may still be considered when amino acid radiotracers are unavailable. This could be particularly useful when recurrence is suspected within a previously irradiated field, where reduced uptake in surrounding brain tissue can increase the tumor-to- background ratio, or in tumors known to exhibit high [¹⁸F]FDG avidity.²⁶

As reported for gliomas, delayed [¹⁸F]FDG PET acquisitions may improve diagnostic performance.^{5,27}

[⁶⁸Ga]Ga-FAPI (fibroblast activation protein inhibitor) PET is an emerging imaging approach currently under investigation. Because BBB disruption is common in brain metastases and fibroblast activation protein expression is frequently increased in these lesions, preliminary studies suggest that [⁶⁸Ga]Ga-FAPI PET may provide higher lesion-to-background contrast than [¹⁸F]FDG PET, as illustrated in **Fig. 2**. However, its role remains investigational and requires further validation.^{28–30}

[¹⁸F]FDG PET IN PRIMARY CENTRAL NERVOUS SYSTEM LYMPHOMA

PCNSL is an uncommon extranodal form of non-Hodgkin's lymphoma that involves the brain, spinal cord, leptomeninges, and/or eyes, in absence of systemic disease. The vast majority of PCNSL cases (approximately 90%–95%) corresponds to diffuse large B-cell lymphoma. Overall, PCNSL remains a rare malignancy, accounting for approximately 5% of all primary brain tumors, but its incidence has increased over the past 2 decades, most notably in the elderly.^{31–33}

MR imaging remains the modality of choice for the evaluation of PCNSL.³³ Nevertheless, [¹⁸F] FDG PET plays an important role in the diagnostic evaluation of PCNSL, as these lesions typically demonstrate intense and homogenous [¹⁸F]FDG uptake due to very high cellular density with an accelerated glycolytic metabolism, as illustrated in **Fig. 3**. [¹⁸F]FDG PET can assist in the differential diagnosis with gliomas and with nonmalignant conditions such as infectious diseases, particularly in immunocompromised patients (eg, toxoplasmosis). Compared with these conditions, PCNSL generally shows higher and more homogenous uptake. The diagnostic accuracy of pretreatment [¹⁸F]FDG PET is high, with pooled sensitivity and specificity reported to be exceed 84%.^{5,13,14,33,34}

Although extracerebral involvement is uncommon (approximately 4%–7%), whole-body [¹⁸F] FDG PET is recommended in patient with PCNSL for initial staging and to exclude systemic involvement, which is a key determinant in therapeutic management.^{33–36}

Notably, amino-acid PET has not demonstrated an additional diagnostic value compared with [¹⁸F] FDG PET in the evaluation of PCNSL.¹⁴

PCNSL is associated with a generally poor prognosis. Age and functional status at diagnosis are well-established prognostic factors. Additional prognostic indicators have been proposed, including biological markers and imaging features derived from MR imaging. On [¹⁸F]FDG PET, high tracer uptake and a large metabolic tumor volume have been identified as predictors of poor prognosis.^{33,36}

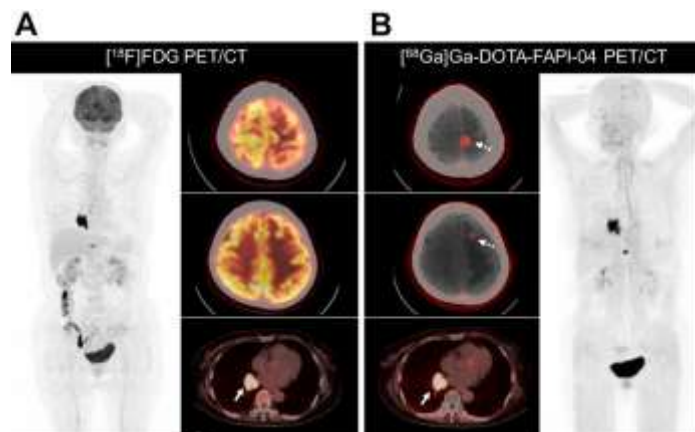
The treatment of PCNSL is primarily based on high-dose methotrexate-based chemotherapy (HD-MTX). In elderly patients, HD-MTX-based regimens are generally used alone, whereas in younger and fit patients, they may be combined with consolidation using autologous stem cell transplantation.³⁷

The International Primary CNS Lymphoma Collaborative Group recommend response assessment based mainly on MR imaging criteria. However, several studies have reported early relapse in patients considered to be in complete response on end-of-treatment MR imaging, raising concerns about the ability of MR imaging to reliably detect residual disease.³²

Interim [¹⁸F]FDG PET performed after 2 cycles of methotrexate-based chemotherapy has emerged as a robust early predictor of treatment response, particularly in patients with partial or unconfirmed responses on MR imaging.³⁸ The prospective LOCALYSE study, including 65 patients, demonstrated that persistent residual uptake on interim [¹⁸F]FDG PET, defined as tracer uptake higher than that of the contralateral normal brain, was associated with a suboptimal response at

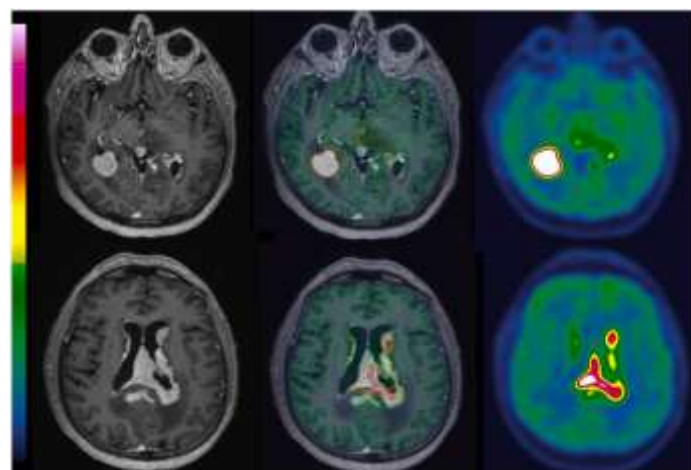
the end of therapy and with poorer prognosis. These findings highlight the potential value of interim [^{18}F]FDG PET for early therapeutic stratification in patient with PCNSL.³²

Figure 2.



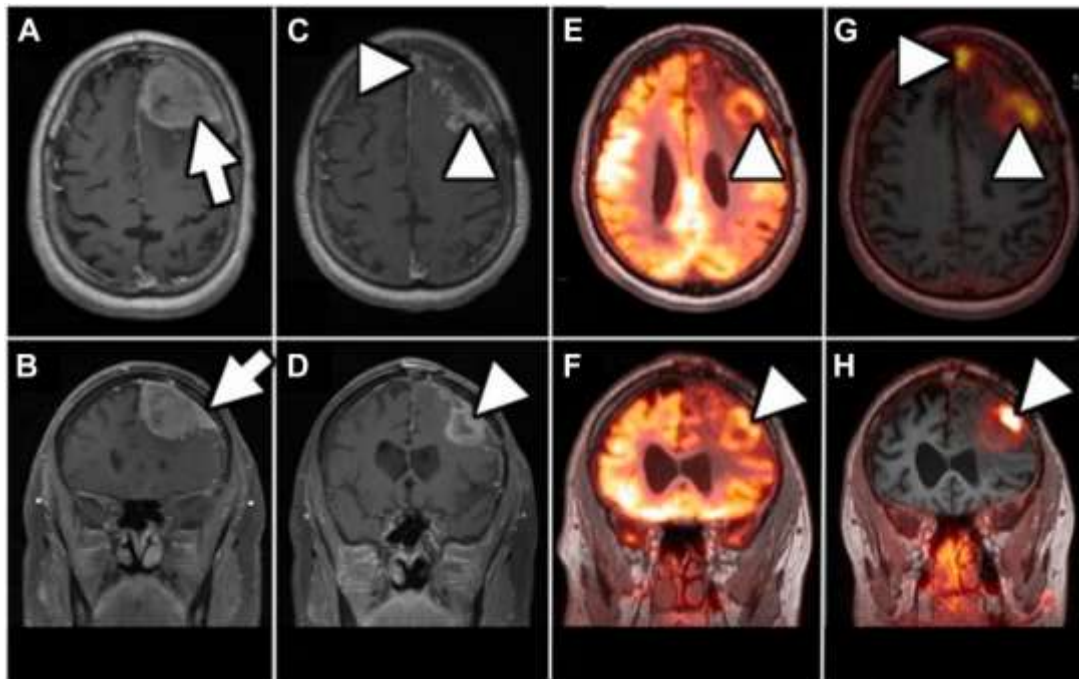
(A, B) PET/CT scans of a 76 year old female patient diagnosed with non-small cell lung cancer (adenocarcinoma). (A) [^{18}F]FDG PET/CT shows a ring-like accumulation with a perifocal hypometabolic area relative to the gray matter in the left parietal lobe (upper row) and frontal lobe (middle row), respectively. (B) [^{68}Ga]Ga-DOTA-FAPI-04 PET/CT shows clearer borders of the 2 lesions. One of the lesions is only 8 to 9 mm in diameter (dotted arrow), which was further confirmed by a cerebral MR imaging. The primary tumor in the right upper lobe revealed intense uptake of both [^{18}F]FDG and [^{68}Ga]Ga-DOTA-FAPI-04 (solid arrow, lower row). (Chen, H., Pang, Y., Wu, J. et al. Comparison of [^{68}Ga]Ga-DOTA-FAPI-04 and [^{18}F]FDG PET/CT for the diagnosis of primary and metastatic lesions in patients with various types of cancer. Eur J Nucl Med Mol Imaging 47, 1820–1832 (2020). <https://doi.org/10.1007/s00259-020-04769-z>.)

Figure 3.



Images of a 64 year old woman with PCNS lymphoma. Axial T1- weighted MR imaging shows enhanced periventricular lesions (left), with a corresponding intense hypermetabolism on [^{18}F]FDG PET (right) and fused MR imaging/PET (middle), and a relative extinction of cortical metabolism. (Reproduced from Horowitz T, Tabouret E, Graillon T, et al. Contribution of nuclear medicine to the diagnosis and management of primary brain tumours. Rev Neurol (Paris). 2023;179(5):394-404.13. Copyright © 2023 published by Elsevier Masson SAS. All rights reserved.)

Figure 4.



Recurrent anaplastic meningioma on [^{18}F]FDG and [^{68}Ga]Ga-DOTATATE PET. Axial (A) and coronal (B) postcontrast T1-weighted MR imaging shows a large enhancing left frontal WHO grade 3 anaplastic meningioma (white arrows). After stereotactic radiosurgery, axial (C) and coronal (D) postcontrast T1-weighted MR imaging show persistent enhancement in the treatment bed (white arrowheads). There is corresponding focal increased uptake (white arrowheads) on fused axial (E) and coronal (F) [^{18}F]FDG PET/MR imaging, indicating viable high-grade tumor. Subsequently performed axial (G) and coronal (H) fused- ^{68}Ga]Ga-DOTATATE PET/MR images show intense uptake (white arrowheads) in the left frontal region, corresponding to the previously documented regions of elevated [^{18}F]FDG uptake and MR imaging enhancement. This indicates SSTRs within the residual anaplastic meningioma, therefore potential benefit in SSTRs radionuclide therapies. (Jessica Zhang, Katie Suzanne Traylor, James M. Mountz, PET and SPECT Imaging of Brain Tumors, Seminars in Ultrasound, CT and MRI, 41 (6), 2020, 530-540, <https://doi.org/10.1053/j.sult.2020.08.007>.)

[^{18}F]FDG PET IN MENINGIOMAS

Meningiomas are the most common intracranial tumors in adults. Although most are WHO grade I, approximately 15% are grade II and around 5% are grade III.³⁹ PET imaging can be valuable in several clinical scenarios, including lesions located in complex anatomic regions, or when evaluating suspected recurrence of previously treated meningioma. Standard management relies on surgery and/or radiotherapy. When these treatments are not feasible or when the disease progresses despite therapy, meningiomas are considered refractory, and target radionuclide therapy may be considered.³⁴

Because of the increased glycolytic activity of tumor cells, uptake of [^{18}F]FDG is generally higher in tumoral than in nontumoral tissue.¹¹ However, [^{18}F]FDG is not considered as the optimal tracer for the evaluation of meningiomas.

Meningiomas are typically slow-growing tumors, and their glucose metabolism may be only moderately increased. In addition, the high physiologic uptake of [^{18}F]FDG in the cerebral cortex results in a low tumor-to-background ratio, thereby limiting the sensitivity for both detection and accurate delineation of meningiomas. Furthermore, [^{18}F]FDG uptake may also be increased in inflammatory tissue, which can further reduce specificity.⁴⁰

Meningiomas characteristically express somatostatin receptors subtype 2,⁴¹ making SSTR ligands the preferred radiotracers for imaging these tumors, as illustrated in **Fig. 4**.^{5,40,42}

AA PET may also be used in meningiomas. Compared to [^{18}F]FDG PET, it provides a higher tumor-to-background contrast. However, the availability of SSTR ligands with even greater contrast has limited its use in meningioma imaging. Notably, O-(2-[^{18}F]-fluoroethyl)-L-tyrosine ([^{18}F] FET) does not accumulate in the pituitary gland, unlike L-methyl-[^{11}C]methionine ([^{11}C]MET) and SSTR ligands, which may facilitate the detection of intrasellar invasion.^{40,43}

[^{18}F]FDG IN PEDIATRIC BRAIN TUMORS

Brain tumors are the second most common malignancy after leukemia and represent the most frequent solid tumor in the pediatric population, accounting for 20% to 25% of all childhood cancers. They remain the leading cause of cancer-related mortality in children.⁴⁴

Pediatric-type gliomas were recognized as a distinct entity in the 2021 WHO CNS5, reflecting their unique molecular features that distinguish them from adult-type gliomas.⁴⁵

The main therapeutic approach in pediatric gliomas is surgery in association with chemotherapy and/or radiotherapy.⁴⁶

Conventional MR imaging remains the gold standard for assessing tumor location and characteristics, although it has recognized limitations, including difficulty in differentiating tumors from tumor mimics and in accurately defining tumor type and grade. Pseudoprogression may also occur in pediatric-type gliomas, whereas pseudoresponse is less commonly observed compared with adult-type gliomas.⁴⁶

[^{18}F]FDG PET can be used in pediatric brain tumors, exploiting the increased glucose metabolism of malignant gliomas. However, as in adults, the high physiologic glucose uptake in normal brain parenchyma results in a low tumor-to-background contrast, making it challenging to distinguish intracerebral malignancies from normal tissue or nonneoplastic lesions. This limitation is particularly pronounced in low-grade tumors, where [^{18}F]FDG uptake often approximates that of normal white matter.⁴⁶

Consequently, the main clinical application of [^{18}F]FDG PET in pediatric brain tumors is the assessment of tumor biological behavior. Gliomas exhibiting markedly increased [^{18}F]FDG uptake are often associated with more aggressive and malignant phenotype, as illustrated in **Fig. 5**. [^{18}F]FDG PET also provides valuable information in low-grade gliomas, where higher uptake is generally correlated with an increased risk of disease progression. However, no universally accepted

threshold has been established to reliably identify patients at higher risk. A hotspot/brain index, calculated as $2 + 2 \times (\text{ROI}_{\text{tumor}} - \text{ROI}_{\text{white matter}} / [\text{ROI}_{\text{gray matter}} - \text{ROI}_{\text{white matter}}])$, of 1.83 has been proposed as the optimal cutoff to distinguish low-grade from high-grade tumors.^{46,47}

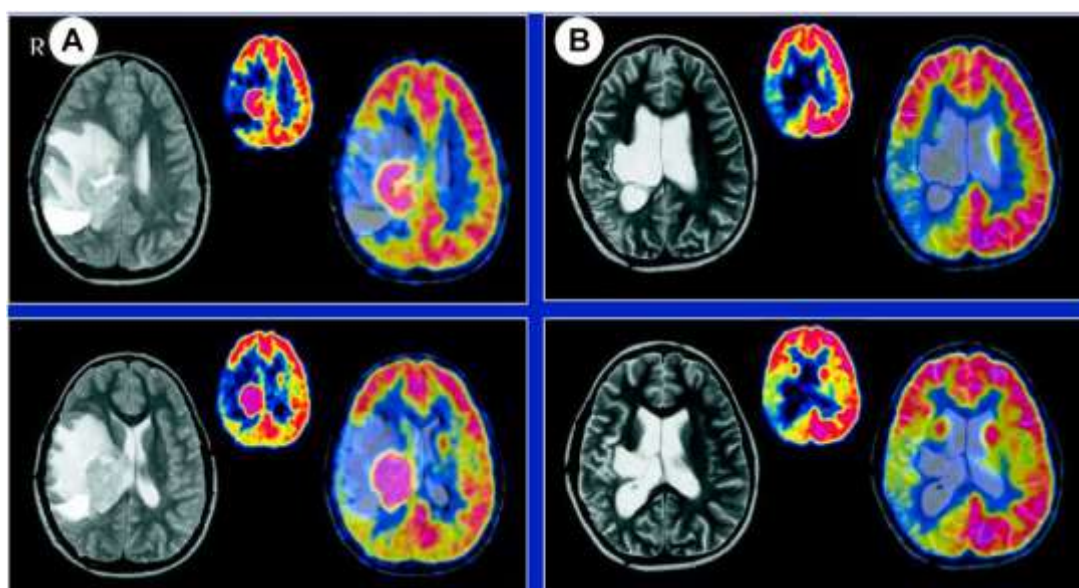
[¹⁸F]FDG PET can also be used to guide biopsy when coregistered with MR imaging, enabling precise identification of anaplastic regions, which often correspond to areas of highest tracer uptake. This strategy can reduce the frequency of nondiagnostic or noncontributory biopsies.^{46–48}

[¹⁸F]FDG PET may also be used to detect residual or recurrent disease and to differentiate it from posttherapeutic changes, such as necrosis.^{46,47}

When available, PET/MR imaging systems are preferred in the pediatric population, as they substantially reduce scanning time, minimize the need for sedation or anesthesia, and also lower patient radiation exposure.^{46,49,50}

In recent years, a growing body of evidence has demonstrated the amino acid PET radiotracers offer several diagnostic advantages over [¹⁸F] FDG PET.^{23,46,51}

Figure 5.



(A, B) 11 year old boy with an anaplastic ganglioglioma in the right frontal region close to the lateral ventricle. (A) [¹⁸F]FDG-PET scan with MR imaging coregistration (T2) at diagnosis (after biopsy) and (B) [¹⁸F] FDG-PET with MR imaging coregistration (T2) at follow-up when a recurrence was suspected. The T2-weighted MR imaging scan at follow-up (B) shows contrast enhancement in the lateral posterior periphery of the surgical cavity, but the [¹⁸F]FDG-PET scan with MR imaging coregistration (B) shows no sign of local recurrence. The boy was disease free at 2 year follow-up. (PET and MRI images courtesy of Dr. Lise Borgwardt, Rigshospitalet, Copenhagen, Denmark.)

DISCLOSURE

The authors have nothing to disclose.

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