

[⁶⁸Ga]-DOTA-conjugated somatostatin receptor-targeting peptide PET for the differentiation between meningioma and glioblastoma: A case report and review of the literature

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Abstract. [⁶⁸Ga]-tetraazacyclododecanetetraacetic acid (DOTA)-conjugated positron emission tomography (PET) is widely used to identify meningiomas due to their high expression of somatostatin receptor type 2 (SSTR2). However, recent evidence suggests that this tracer may also show uptake in high-grade gliomas, raising concerns about its diagnostic specificity. The current study presents a challenging case of a 56-year-old man who was initially diagnosed with a right temporal glioblastoma. Follow-up imaging revealed a local recurrence and a new extra-axial lesion suggestive of meningioma on magnetic resonance imaging and [⁶⁸Ga]-DOTA-octreotide (DOTATOC) PET. Unexpectedly, histopathological analysis following resection confirmed both lesions as glioblastomas, indicating that SSTR2 uptake is not exclusive to meningiomas. A systematic literature review further supports the fact that high-grade gliomas can exhibit [⁶⁸Ga]-DOTA tracer uptake, though generally at lower levels than meningiomas. These findings suggest that while [⁶⁸Ga]-DOTA PET provides useful diagnostic information,

interpreting results requires caution in cases where glioblastoma might mimic meningioma. Future research should focus on establishing clear thresholds to reliably distinguish between meningiomas and high-grade gliomas, enhancing diagnostic precision and treatment planning in neuro-oncology.

Introduction

Intracranial tumors represent a highly heterogeneous group of neoplastic diseases, exhibiting wide variability in prognosis and treatment approaches. The most common intracranial tumors are gliomas and meningiomas (1). Gliomas, particularly glioblastomas, represent the most prevalent malignant intrinsic brain tumors in adults, with a poor prognosis and a median 5-year survival rate of <7% (1). By contrast, meningiomas are mostly benign extra-axial brain tumors, with an overall 10-year survival rate of >95% (2). These types of tumors differ markedly in terms of prognosis, etiology and therapeutic approaches. Glioblastoma, considered to originate from astrocytic cells, is treated with surgery and radiochemotherapy (3), while meningioma arises from the tumorous proliferation of arachnoid mater cells and is primarily managed with surgery and, in some cases, radiotherapy (4).

Magnetic resonance imaging (MRI) is routinely performed to diagnose and distinguish cranial tumorous lesions. In cases of uncertainty, positron emission tomography (PET) is used to enhance diagnostic accuracy and determine the underlying tumor diagnosis. The use of [⁶⁸Ga]-labeled tetraazacyclododecanetetraacetic acid (DOTA) PET/MRI as a somatostatin-receptor binding tracer is well established for the diagnosis of neuroendocrine tumors (NETs) (5). However, similar to NETs, somatostatin receptors (SSTRs) are upregulated in numerous intracranial tumors, including

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meningioma (6). Currently, [⁶⁸Ga]-DOTA PET is primarily utilized to identify meningiomas due to their high expression of SSTR type 2 (SSTR2) (7). However, a recent report has highlighted that SSTR2 is not only expressed in meningioma but also in glioma (8). Consequently, the specificity of [⁶⁸Ga]-DOTA PET/MRI for diagnosing meningiomas should be discussed carefully.

The present study discusses the role of [⁶⁸Ga]-DOTA-conjugated SSTR-targeting peptide PET/MRI imaging in distinguishing meningioma and glioma. A challenging case is presented of a patient with two intracranial lesions, suggesting the comorbidity of glioblastoma and meningioma. Moreover, a systematic review of [⁶⁸Ga]-DOTA-conjugated SSTR-targeting peptide imaging is provided to evaluate its diagnostic value in identifying glioma.

Case report

Methods. The case is reported according to the Case Report (CARE) guidelines (9) and the patient provided written informed consent for this publication. The two tissue samples referenced in this report were obtained from the same patient in March 2024 during a single surgical procedure. One sample was collected from the lesion within the preexisting resection cavity, while the other was taken from a distant site at the planum sphenoidale. Following the surgery, the samples were fixed in formalin and subsequently transferred to the local biobank. The tissues were then made available for scientific research and subjected to detailed neuropathological analysis. This process included embedding the tissues in paraffin and preparing thin sections on microscope slides. Histological staining was conducted using hematoxylin and eosin (H&E) or hematoxylin alone, alongside immunohistochemical staining for SSTR2 and glial fibrillary acidic protein (GFAP), following standardized protocols.

All tissue samples were fixed in 4% buffered formalin at room temperature, embedded in paraffin, and sectioned to a thickness of 1 μ m. H&E and hematoxylin staining was performed using the VENTANA[®] HE 600 System (Roche Diagnostics GmbH). Immunohistochemical staining was carried out on the BenchMark ULTRA Slide Staining System (Roche Diagnostics GmbH) using an anti-GFAP polyclonal rabbit antibody (cat. no. Z0334; Dako; Agilent Technologies, Inc.) at a 1:4,000 dilution, with an incubation time of 16 min at 36°C. Staining for SSTR2 was performed using a polyclonal rabbit antibody (cat. no. RBK046-05; Zytomed Systems GmbH) at a 1:50 dilution, with antigen retrieval for 64 min at 90°C in CC1 buffer (Roche Diagnostics GmbH), followed by antibody incubation for 24 min at 36°C. All stained slides were examined under a light microscope (Eclipse 80i; Nikon Corporation).

Patient details. In September 2021, a 56-year-old male patient attended University Hospital Essen (Essen, Germany) and was diagnosed with an intracerebral, right temporal, ring-like contrast-enhancing lesion. The patient underwent microsurgical tumor removal, and a neuropathological assessment revealed the diagnosis of a glioblastoma of Central Nervous System World Health Organization (WHO) grade 4 (10). The prior medical history included a posterior fossa neurinoma,

which was previously treated with stereotactic irradiation. Postoperatively, concomitant radiochemotherapy according to Stupp *et al* (11) was initiated, followed by six cycles of adjuvant temozolomide, administered at 200 mg/m² daily for the first 5 days of a 4-week cycle, and tumor-treating fields.

In January 2024, at the age of 58 years, the patient experienced a new extra-axial lesion at the planum sphenoidale with homogeneous contrast-enhancement, radiographically consistent with meningioma but suspicious for a distant glioblastoma manifestation due to its *de novo* appearance. The case was discussed, and a [⁶⁸Ga]-DOTA-octreotide (DOTATOC) PET/MRI was recommended. The imaging was performed 2 weeks later (dosage, 85 MBq; uptake time, 30 min after injection). MRI confirmed the extra-axial lesion and additionally showed local tumor recurrence in the right temporal lobe. [⁶⁸Ga]-DOTATOC PET demonstrated pronounced tracer accumulation in the pituitary gland and doubled maximum standardized uptake (SUV_{max}) values in the extra-axial lesion compared to the glioblastoma recurrence [SUV_{max} extra-axial lesion of the planum sphenoidale (lesion_1), 2.91; SUV_{max} local glioblastoma recurrence (lesion_2), 1.35; SUV_{max} pituitary gland, 8.23]. The [⁶⁸Ga]-DOTATOC PET/MRI investigation is illustrated in Fig. 1A-C. Due to the uncertainty of the diagnosis of the extra-axial lesion, the patient underwent tumor removal, involving both the right temporal lesion and the extra-axial lesion. The neuropathological assessment revealed a glioblastoma diagnosis in both lesions. The right temporal lesion demonstrated a diffusely infiltrative glioma, composed of astrocytic, differentiated tumor cells with marked pleomorphism, while the extra-axial lesion revealed a highly cellular glioma with a small cell-like appearance. Representative neuropathological slices are presented in Fig. 2. Retrospectively, SSTR2 expression was assessed, and consistent with [⁶⁸Ga]-DOTATOC PET imaging, the right temporal lesion showed weak positivity for SSTR2, while the extra-axial lesion demonstrated increased SSTR2 expression. Representative staining is presented in Fig. 1D. Post-surgery, systemic treatment with lomustine, administered at 110 mg/m² on the first day of a 6-week cycle, combined with tumor-treating fields was initiated. Following the second recurrence, the treatment strategy was adjusted to systemic therapy with trofosamide and etoposide, administered at 100 and 25 mg/m², respectively, following a ‘1 week on - 1 week off’ schedule within a 4-week cycle, while continuing the use of tumor-treating fields. At the most recent follow-up in May 2024, the patient remains alive and under clinical observation, with a survival period of 2 years and 8 months since the initial diagnosis.

Literature review

To provide a comprehensive review of all reports on [⁶⁸Ga]-DOTA-conjugated SSTR-targeting peptide PET and glioma, a broad search strategy was implemented. To avoid database bias, research was conducted using PubMed/MEDLINE (<https://pubmed.ncbi.nlm.nih.gov/>), Scopus (<https://www.elsevier.com/en-gb/products/scopus>), Embase (<https://embase.com>) and Web of Science (<https://webofscience.com>), utilizing the search string: [‘⁶⁸Ga-DOTA*’ OR ‘⁶⁸Ga-DOTA*’ OR ‘Gallium-68 DOTA*’) AND (‘glioma*’ OR ‘glioblastoma*’ OR

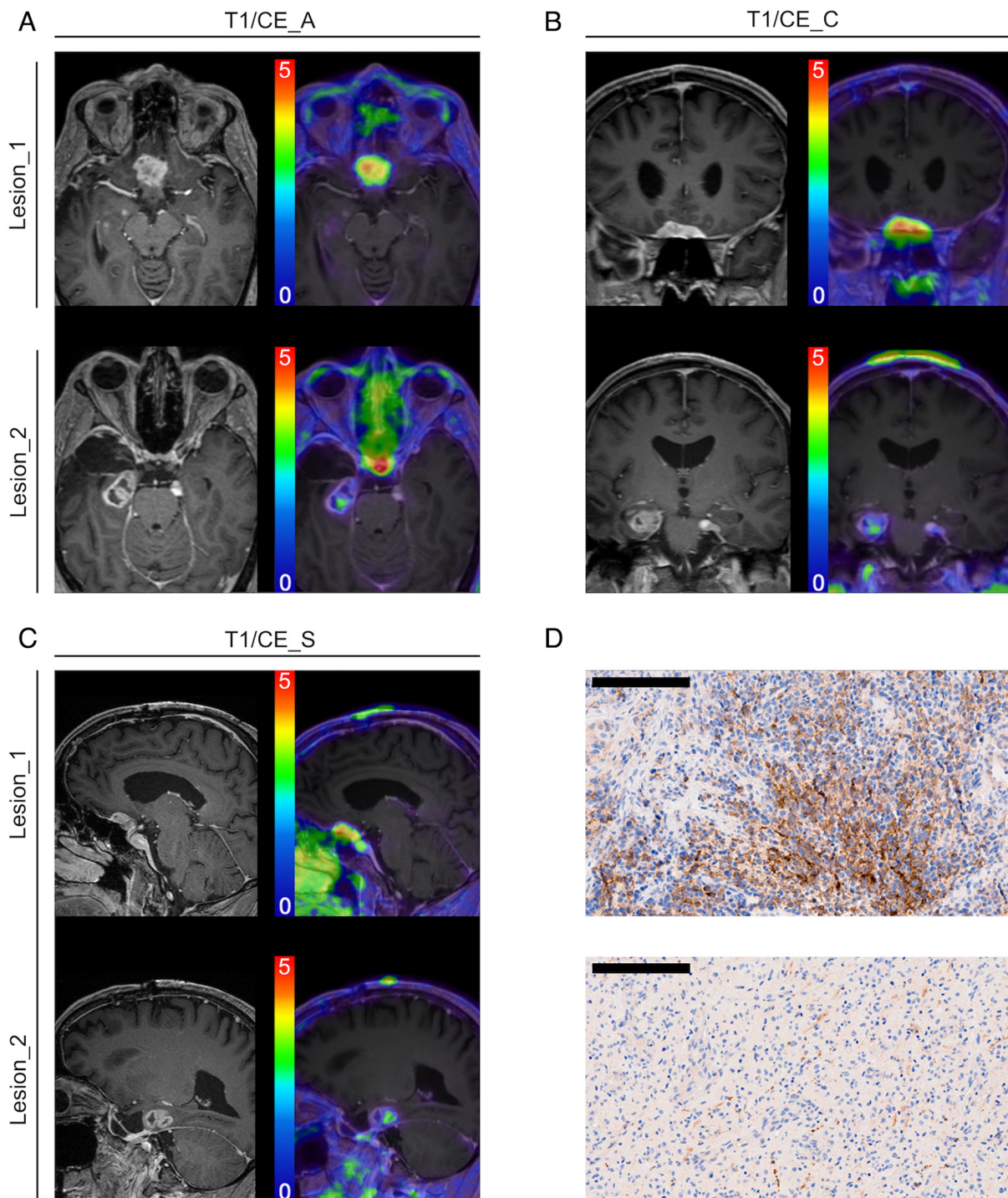


Figure 1. Preoperative imaging results of both lesions. (A-C) Preoperative MRI (left) and [⁶⁸Ga]-DOTA-octreotide-PET/MRI (right) images focusing on the extra-axial tumor manifestation at the planum sphenoidale (lesion_1; above) and the right temporal glioblastoma recurrence (lesion_2; below). MRI images demonstrate contrast-enhanced T1-sequences. All lesions are presented in (A) axial, (B) coronal and (C) sagittal images. The following SUVmax values were obtained: Lesion_1, 2.91; and lesion_2, 1.35. (D) Representative sections were stained for SSTR2, with high expression of SSTR2 found in lesion_1 (top) and weak positivity for SSTR2 found in lesion_2 (bottom) (scale bar, 200 μ m). CE, contrast-enhanced; SSTR2, somatostatin receptor 2; SUV, standardized uptake value; MRI, magnetic resonance imaging; T1/CE_A, axial, contrast-enhanced T1 MRI; T1/CE_C, coronal, contrast-enhanced T1 MRI; T1/CE_S, sagittal, contrast-enhanced T1 MRI.

'astrocytoma*' OR 'ependymoma*']. Following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (12), a computerized literature search was performed on May 25, 2024. All duplicates were excluded. The titles and abstracts of the remaining publications were reviewed, and suitable articles were extracted. All

articles were independently screened by two investigators for their suitability. The references of eligible publications were reviewed, and additional citations were extracted. Research studies were considered eligible for inclusion if they addressed the use of [⁶⁸Ga]-DOTA-conjugated SSTR-targeting peptide PET in glioma, including preclinical and clinical research.

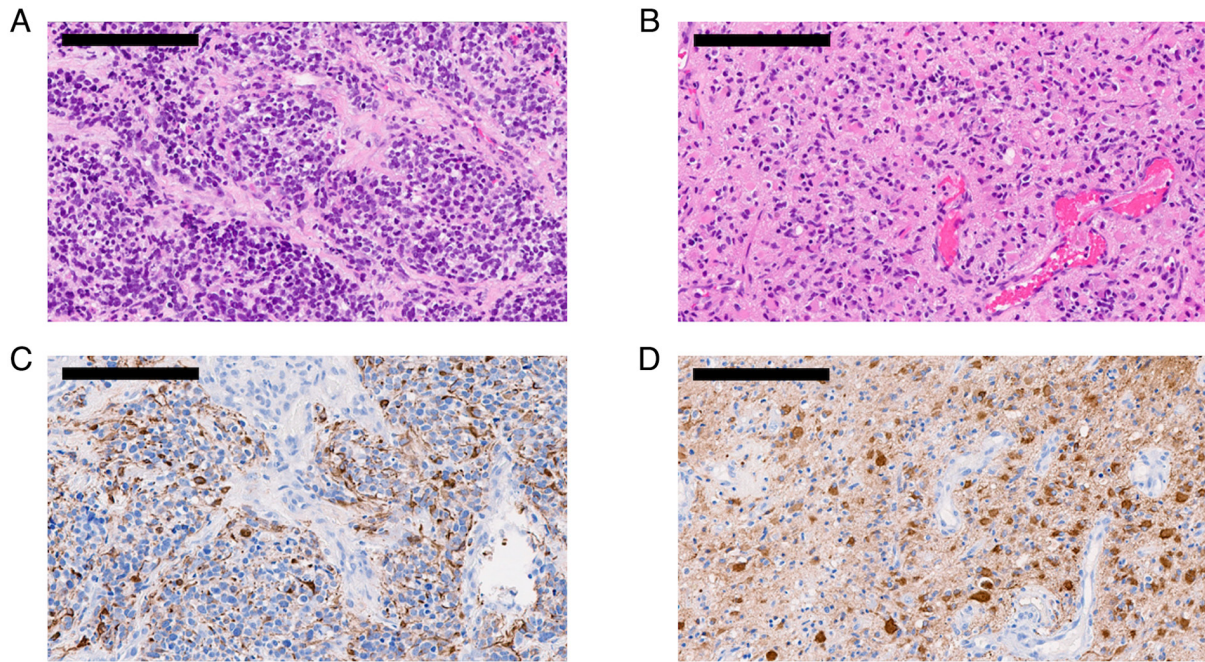


Figure 2. Microscopic examination results of both lesions. Representative images are shown of the extra-axial tumor manifestation at the (A) planum sphenoidale and the (B) right temporal glioblastoma recurrence. The sections were stained with hematoxylin and eosin. Representative images are shown of both lesions after staining for GFAP. GFAP is stained in brown, while a hematoxylin counterstain highlights cell nuclei in blue. (C) Images reveal a GFAP-positive tumor component with higher cellularity and a more small-cell-like appearance. (D) Images reveal a diffusely GFAP-positive infiltrating glioma, composed of astrocytic, differentiated tumor cells exhibiting marked pleomorphism. Scale bar, 200 μm . FAP, glial fibrillary acidic protein.

Review articles or conference abstracts were not included. The resulting publication list was used to prepare this review, and all included studies were subsequently analyzed in detail. The final publication list was reviewed by two investigators, and relevant data points were extracted.

The systematic literature search and subsequent analysis identified six publications investigating the use of [^{68}Ga]-DOTA-conjugated SSTR-targeting peptide PET in glioma patients or preclinical glioma models (8,13-17). Five publications were clinically oriented and examined patients with either primary or recurrent tumor entities. Results are presented in Table SI. One publication referred to the preclinical examination of PET imaging on chemically induced glioma in animal models. Details are provided in Table SII. In total, 44 patients with glioma underwent PET imaging. Extensive heterogeneity was observed among the studied entities, as the inclusion extended beyond high-grade tumors, with individual tumor grading occasionally unspecified. Additionally, the study protocols often differed regarding the administered tracer dose and uptake time, with some details occasionally missing. Absolute tumor-to-brain ratio or SUV values were rarely reported; however, a common finding was tracer uptake in malignant gliomas of WHO grades III and IV, corresponding to contrast enhancement in MRI according to the 2016 WHO classification (10).

Discussion

The use of [^{68}Ga]-DOTA PET imaging as a surrogate marker for SSTR-expressing tumors is well established in oncology, and is particularly valuable in neuro-oncology for assessing meningiomas. Compared to MRI, [^{68}Ga]-DOTA PET offers

superior sensitivity and specificity for diagnosing meningiomas based on imaging alone (18). Traditionally, intracranial tumors require histopathological confirmation before initiating specific treatment. However, in cases of meningioma, [^{68}Ga]-DOTA PET is gaining recognition as a sufficiently specific imaging modality, enabling radiotherapy to be initiated for non-surgical cases without the need for prior tissue biopsy.

Recently, case reports and series have raised concerns about the specificity of SSTR expression for meningiomas, noting uptake in intracerebral tumors such as high-grade gliomas, including glioblastoma (8,19,20). Both gliomas and meningiomas can demonstrate increased uptake of [^{68}Ga]-DOTA compounds, particularly in cases of elevated SSTR expression or high vascularization (7,16). This overlap in tracer uptake can make differentiation challenging, especially in cases with atypical presentations or unusual tumor locations. While gliomas and meningiomas are generally distinguishable, certain features may blur the distinction. Gliomas, particularly high-grade ones, often exhibit heterogeneous uptake due to necrosis and irregular blood-brain barrier permeability (8). They may also occasionally express SSTRs, though typically at lower levels than meningiomas (13). By contrast, meningiomas are predominantly extra-axial tumors with robust, homogeneous [^{68}Ga]-DOTA uptake, reflecting their high SSTR density. However, tumor location, such as the parasagittal region or skull base, can cause gliomas to mimic meningiomas on imaging (21). Additionally, atypical or recurrent meningiomas may display more aggressive features, further complicating the diagnostic process (22).

The current study presents a case of glioblastoma with two distinct SSTR-expressing intracranial lesions showing tracer

enhancement on [⁶⁸Ga]-DOTATOC PET. While MRI and PET data initially suggested recurrent glioblastoma and a distant meningioma, histological analysis confirmed both lesions as glioblastoma, further supporting concerns about the specificity of SSTR PET in meningioma diagnosis.

Currently, evidence-based data on [⁶⁸Ga]-DOTA PET imaging for gliomas is limited. The present systematic literature review confirmed minimal data on the role of [⁶⁸Ga]-DOTA PET in glioma imaging, although existing studies report tracer uptake in gliomas. The only preclinical study, which investigated [⁶⁸Ga]-DOTA uptake in chemically induced gliomas in rodents, showed minimal tracer enhancement (14).

Comparative studies on SSTR expression in meningiomas vs. gliomas are sparse but indicate that while [⁶⁸Ga]-DOTA uptake is detectable in gliomas, the uptake levels are lower than those observed in meningiomas (13). Studies by Kiviniemi *et al* (8), Li *et al* (16) and Lapa *et al* (15) emphasized the role of blood-brain barrier integrity in tracer uptake, with reduced uptake linked to an intact blood-brain barrier. Consequently, high-grade gliomas with disrupted barriers may be more appropriate for [⁶⁸Ga]-DOTA PET imaging than lower-grade gliomas, which often lack contrast enhancement on MRI.

In conclusion, the present complex case, coupled with the limited available literature, suggests that [⁶⁸Ga]-DOTA PET imaging is not exclusively specific to meningiomas and may demonstrate uptake in gliomas, particularly high-grade types. All reviewed studies report some degree of glioma tracer enhancement in [⁶⁸Ga]-DOTA PET, although meningiomas generally demonstrate higher uptake. Establishing standardized cut-off values to differentiate between tumor types remains a critical need, as no validated SSTR-PET uptake scale specific to meningiomas currently exists (23). Models that examine the diagnostic performance of a combined application of PET and MRI would be of interest, incorporating meningioma-specific PET parameters (e.g., high SSTR2 expression with a yet-to-be-defined tumor-to-brain ratio) and MRI parameters (e.g., homogeneous contrast enhancement and the dural tail sign in dura-attached tumors). Consequently, clinicians must currently integrate clinical presentation, MRI findings and PET imaging results, maintaining caution in presuming meningioma specificity for [⁶⁸Ga]-DOTA PET imaging. Accurate determination of the patient's prognosis is crucial, as the therapeutic approaches for meningiomas and gliomas differ significantly in treatment intensity. This becomes even more critical when some tumors are diagnosed as meningiomas based solely on PET imaging and are subsequently treated with radiation without prior histological confirmation. Further clinical studies are essential to improve PET imaging specificity for differentiating brain tumor types and to define specific cut-off values for clinical decision-making.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

LR and FG conceived the study, contributed to data acquisition, participated in the case analysis and interpretation, conducted the systematic literature review, and drafted the manuscript. CB conducted imaging analyses and interpretation, provided technical expertise on PET, and assisted with manuscript preparation. TB supported the acquisition and interpretation of tissue data, contributed to case management discussions, and reviewed the manuscript for clinical relevance. SK, CDo, and CDe provided input on study design, contributed to data interpretation, and offered expertise on imaging and tissue analyses. US and KH provided clinical oversight and the necessary infrastructure. PD supervised the study, contributed to conceptual design, critically revised the manuscript, and provided guidance on study direction and methodology. All authors have read and approved the final manuscript. LR and FG confirm the authenticity of all the raw data.

Ethical approval and consent to participate

Tissue sampling was performed in accordance with the guidelines of the Ethics Committee of the Medical Faculty at the University of Duisburg-Essen (Essen, Germany; ethical approval ID 19-8706-BO).

Patient consent for publication

The patient provided written informed consent for data use and publication.

Competing interests

The authors declare that they have no competing interests.

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