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Association of tumor metabolic parameters on amino acid PET/CT with EGFR expression

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ABSTRACT

Background. According to the 2021 WHO classification, molecular-genetic profiling of CNS tumors is the standard for diagnosis. Beyond *IDH* mutation status, EGFR receptor alterations are significant factors associated with unfavorable prognosis and treatment resistance. Identifying correlations between genetic abnormalities and amino acid PET/CT parameters is important for discovering non-invasive surrogate markers of aggressive phenotype.

Objective. To analyze the relationship between *EGFR* / *EGFRvIII* gene expression and metabolic PET/CT parameters in patients with glial tumors.

Material and methods. The study included 71 adults with gliomas: glioblastoma (n=52), Grade 3 astrocytoma (n=11), and Grade 3 oligodendroglioma (n=8). All patients underwent PET/CT with ^{11}C-MET (n=41) or ^{18}F-FET (n=30). The tumor-to-background ratio (TBR) and metabolic tumor volume (MTV) were calculated using a TBR segmentation threshold of 1.6. *EGFR* / *EGFRvIII* expression was determined via RT-PCR.

Results. *EGFR* expression was significantly higher in glioblastomas compared to astrocytomas and oligodendrogliomas. A significant positive correlation was found between *EGFR* and MTV (Rs=0.44, p=0.02). In patients with astrocytomas, there was a moderate negative correlation between TBR and *EGFR* expression (Rs=–0.64, p=0.04). No significant correlations were observed in glioblastomas and oligodendrogliomas that may be attributed to high variability of *EGFR*. *EGFRvIII* was expressed in 25% of gliomas (exclusively glioblastomas). However, we found no association with metabolic parameters (possibly due to small sample size).

Conclusion. Differences in *EGFR* expression patterns across various glioma types were demonstrated. Correlation between *EGFR* gene expression and tumor metabolic volume suggests potential role for PET/CT parameters as prognostic markers.

Keywords: *PET/CT, gliomas, EGFR expression, EGFRvIII.*

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Abbreviations

EGFR — epidermal growth factor receptor
EGFRvIII — mutant form of the epidermal growth factor receptor
PET/CT — positron emission tomography combined with computed tomography
MET — ^{11}C-methionine
FET — ^{18}F-fluoroethyltyrosine
LAT — L-type amino acid transporters
SUV — standardized uptake value
TBR — tumor-to-brain ratio
MTV — metabolic tumor volume

Molecular-genetic profiling of CNS tumors in accordance with the 2021 WHO classification is a key objective for non-invasive prediction of aggressive biological behavior of cerebral gliomas [1]. Research of molecular alter-

ations of transmembrane epidermal growth factor receptor (EGFR) is important along with IDH gene mutation status in evaluating prognostic markers for aggressive glioma behavior [2, 3].

To date, EGFR is one of the most extensively studied proteins. The gene encoding this protein is located on chromosome region 7p12. EGFR amplification and subsequent molecular alterations (including overexpression and deletion resulting in EGFRvIII isoform) detected in approximately 50—60% of primary glioblastomas drive activation of oncogenic signaling pathways (RAS/MAPK, PI3K/Akt). This determines them as independent factors associated with poor prognosis and therapeutic resistance [3—6].

Current research focuses on correlations between these molecular abnormalities and tumor metabolic parameters assessed via PET/CT with various radiopharmaceuticals [7—11]. Such correlations allow for consideration of integrated molecular neuroimaging data as potential non-invasive surrogate markers of aggressive behavior of glioma.

Metabolic activity of glioma, as assessed by amino acid PET/CT, is also among the most actively investigated non-invasively derived prognostic factors of tumor behavior [8, 12, 13]. Correlating diagnostic data with morphological characteristics of tumor currently represents a major area of neuroimaging research aimed at identifying surrogate markers of poor prognosis prior to treatment.

The purpose of this study was to investigate the relationship between EGFR / EGFRvIII gene expression and PET/CT metabolic parameters in patients with glial tumors.

Material and methods

The study included patients with glial brain tumors (n=71). There were grade 3 astrocytomas (n=11), grade 3 oligodendrogliomas (n=8) and glioblastomas (n=52). Prior to surgery, all patients underwent PET/CT (Siemens Biograph TruePoint scanner) with standard static scanning protocols for amino acid-based radiopharmaceuticals: ^{11}C-methionine (MET) and ^{18}F-fluoroethyltyrosine (FET). These tracers reflect amino acid transport via LAT ensuring their biological comparability.

Standard assessment of metabolic characteristics of tumor was performed using PET/CT data:

— tumor-to-brain ratio (ratio of standardized uptake value (SUV) of the radiopharmaceutical in the most active part of tumor (SUV_{tumor}) to mean SUV in intact contralateral brain tissue (SUV_{normal}) including gray and white matter);

— metabolic tumor volume (volume of metabolically active tumor tissue semi-automatically delineated using recommended threshold values ($1.6\times SUV_{normal}$) and designated as MTV_1.6).

We evaluated EGFR and EGFRvIII alterations considering the following data. The EGFRvIII gene mRNA contains deletion spanning exons 2 through 8. Primers targeting its cDNA were designed to span the junction of other exons in a manner that prevented annealing to the wild-type template. One of the primers for the wild-type EGFR gene was designed within EGFRvIII gene deletion region. Thus, we excluded amplification from mutant gene template. Primer specificity was verified using genes cloned in plasmids.

We harvested total RNA from tissue samples using the RNAzol reagent (MRC, USA) according to the manufacturer's protocol. Concentration was determined using a Nano-500 spectrophotometer (Allsheng, China). We verified the quality of RNA via agarose gel electrophoresis and test PCR targeting a reference gene.

First-strand cDNA synthesis was performed using the MMLV RT kit (Evrogen, Russia) following the kit's protocol.

We performed quantitative real-time PCR using a CFX96™ Real-Time System thermal cycler (Bio-Rad, USA) and a reagent kit for real-time PCR with SYBR Green I (Cat. No. R-402, Syntol, Russia). The amplification protocol was as follows: 95°C for 5 min followed by 40 cycles of 95°C for 10 s and 60°C for 30 s. A melting curve analysis cycle was included at the end.

We prepared a summary table for data analysis. Descriptive and comparative statistical analysis was performed using Jamovi software (version 2.3.18). Medians and interquartile ranges were calculated for all parameters, as the majority of parameters had non-parametric distribution (Shapiro-Wilk test). The non-parametric Mann-Whitney test was used to assess differences between two independent groups, while the non-parametric one-way ANOVA (Kruskal-Wallis test) with corrections for multiple comparisons was used to compare three groups. The Spearman's rank correlation coefficient was used to quantify the relationships between quantitative values. Differences were significant at $p < 0.05$.

Results

When comparing EGFR gene expression across various glioma groups and control group without neoplasm (intraoperative biopsy), we found significantly higher EGFR expression in glioblastomas compared to the control group (p<0.01). There was less active expression in oligodendroglioma and astrocytoma **(Fig. 1)**. We found no significant correlations between metabolic activity (TBR) and EGFR in glioblastomas and oligodendrogliomas, possibly due to high variability of EGFR values. However, moderate negative correlation was observed in the astrocytoma group. Notably, this group consisted of small sample that complicated assessment of statistical significance (N=11, Rs=-0.64, p=0.04).

Expression of deleted EGFRvIII variant was analyzed in 71 glioma samples. Of these, 13 samples demonstrated positive expression, and all 13 were glioblastomas. All control samples were negative for this isoform. Thus, EGFR-

vIII-positive samples among glioblastomas comprised 25% (13 out of 52) that is consistent with available data. Quantitative EGFRvIII expression for six samples is presented in **Fig. 2**.

EGFR gene expression in glioma samples and tumor metabolic characteristics (TBR and MTV_1.6) were primarily compared with morphological characteristics of tumor samples **(Table 1)**.

Spearman rank correlation analysis was used to investigate the relationship between EGFR gene expression in glioma samples and PET characteristics. We found significant positive ($p<0.05$) correlation between EGFRwt gene expression and metabolic tumor volume MTV_1.6 cm³ ($R=0.44$, $p=0.02$). There was no relationship between EGFRvIII gene expression and metabolic tumor characteristics in this small sample.

Table 1. **EGFR gene expression and tumor metabolic characteristics (TBR and MTV_1.6) depending on histological type of glioma.**

Parameter	Type	N	EGFR gene expression, Me (IQR)
TBR	Astrocytoma	11	1.68 (1.21—2.67)
	Oligodendroglioma	8	2.51 (2.09—3.17)
	Glioblastoma	52	3.66 (3.25—4.24)
MTV_1.6	Astrocytoma	7	21.00 (8.55—32.40)
	Oligodendroglioma	8	12.52 (4.12—19.35)
	Glioblastoma	52	43.18 (26.52—63.15)
EGFR	Astrocytoma	11	4.08 (1.50—4.93)
	Oligodendroglioma	8	1.36 (1.30—1.56)
	Glioblastoma	52	0.57 (0.21—6.27)

Discussion

Amplification of the EGFR locus is extreme in glioblastomas and frequently extrachromosomal in nature. This is a key mechanism driving intratumoral heterogeneity and drug resistance due to unequal segregation of extrachromosomal DNA in mitosis [14, 15].

Subsequent accumulation of additional genetic alterations such as EGFRvIII deletion, point mutations and gene fusions has a clear subclonal pattern. This is confirmed by deep and single-cell sequencing methods that reveal multiple therapeutically significant EGFR variants. Although EGFR protein expression correlates with gene amplification, it is a continuous spectrum rather than bimodal distribution diagnosed by FISH analysis. This complicates determination of diagnostic threshold [16]. Thus, complex architecture of EGFR alterations including high-level amplification, extrachromosomal nature and dynamic subclonal evolution is a fundamental factor in aggressive course of gliomas and warrants in-depth study. This issue prompted us to investigate the relationships between quantitative expression of EGFR and EGFRvIII genes and metabolic characteristics of tumors as determined by PET/CT [15].

Our results demonstrate complex and significant associations between metabolic parameters assessed via amino acid PET and molecular biology of gliomas, specifically EGFR gene expression. Significant positive correlation between EGFR expression and metabolic tumor volume (MTV) aligns with current understanding the biology of malignant gliomas [17, 18]. Oncogenic signaling via EGFR is a key driver disrupting cellular metabolism and promoting proliferation [19]. At the whole-tumor level, this can manifest as expansion of metabolically active cell pool and consequent MTV increase. Unlike TBR, this volumetric parameter characterizes not merely metabolic intensity of a "hot" focus but total mass of biologically aggressive tumor. This makes it a potentially more sensitive marker of tumor burden associated with oncogenic pathway activity [18, 20].

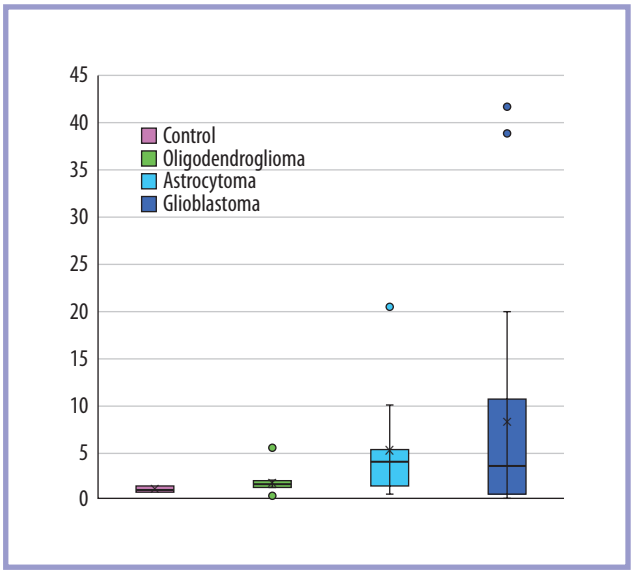

Fig. 1. **EGFR gene expression in human glioma tissues of various malignancy grades.**

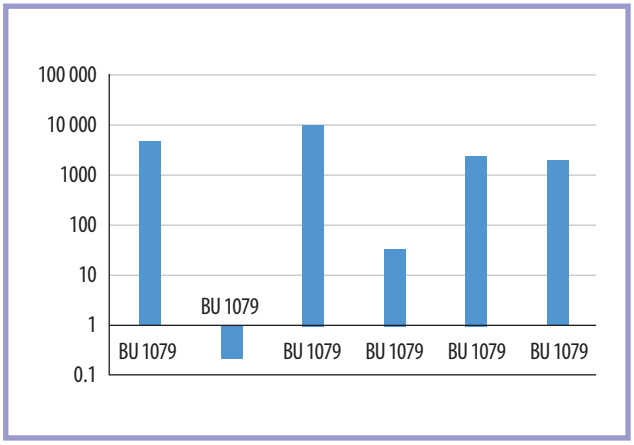

Fig. 2. **EGFRvIII gene expression in glioblastoma samples from six patients.**

No significant correlation between TBR and EGFR expression in the glioblastoma and oligodendroglioma groups, alongside a weak negative correlation in the astrocytoma group, likely reflects a fundamental difference in biological information conveyed by these parameters. Researches of amino acid metabolism highlight that intensity of radiopharmaceutical uptake (TBR) primarily reflects activity of amino acid transporters in cell membrane. This activity can be regulated independently of EGFR-driven proliferative activity [13, 18]. Moreover, high EGFR variability in our study aligns well with the concept of intratumoral heterogeneity. Recent studies attribute this heterogeneity to non-uniform distribution of molecular subclones throughout tumor volume [11, 22].

Regarding the EGFRvIII mutant variant, its detection exclusively within the glioblastoma subgroup (25% incidence) is entirely consistent with available data confirming its role as a specific marker for the most aggressive phenotype [16, 21]. However, no significant association between EGFRvIII and metabolic parameters is logically explained by its subclonal distribution pattern. As indicated by researches devoted to pathogenesis of glioblastoma, EGFRvIII is often expressed not in all tumor cells but only in specific regions. This causes signal to become "diluted" when averaged across the entire metabolic volume, particularly in small sample [23]. Thus, our findings contribute to the paradigm of radiogenomics suggesting that PET/CT metabolic parameters (MTV) may be perspective non-invasive surrogate markers of biological aggressiveness mediated by EGFR signaling pathways [9, 24]. Nevertheless, small size of subgroups and retrospective design necessitate validation in multiple-center studies employing standardized protocols. Further large-scale prospective studies, including analysis of survival and response to therapy, are required to confirm clinical value of these associations and their prognostic significance.

EGFR-blocking therapy has become a prime example of personalized targeted treatment in oncology and has served as a model for similar strategies in neuro-oncology [14]. Despite multiple researches in this field, many aspects of EGFR's role in regulating brain processes are still insufficiently understood. This study demonstrates that quantitative parameters of EGFR expression and metabolic profiles are not sufficiently correlated. Further research is needed to identify imaging patterns either by analyzing larger datasets or by focusing on the relationship between specific EGFR receptor alterations and other diagnostic parameters, including those derived from radiomics [9, 25]. Furthermore, expanding list of drugs targeting the EGFR receptor and the need to evaluate their efficacy require integration of diagnostic and morphological data to identify optimal theranostic pairings [19]. Combination of molecular-genetic profiling with metabolic imaging across large cohorts of various gliomas could establish a new paradigm for patient stratification, facilitating personalized approaches and targeted treatment selection.

Conclusion

This study reveals significant differences in EGFR expression patterns across various gliomas. The most active expression was found in glioblastomas, whereas astrocytomas and oligodendrogliomas were characterized by lower expression. We found significant correlation between EGFR expression and volumetric metabolic parameters derived from amino acid-based PET/CT imaging. This suggests their potential utility as non-invasive prognostic markers and indicators of tumor molecular status.

Author contribution:
Concept and design of the study — Vikhrova N.B., Pavlova G.V., Pronin I.N., Usachev D.Yu.
Collection and analysis of data — Vikhrova N.B., Kalaeva D.B., Drozd S.F., Kalennik O.V., Kosyrkova A.V., Danilov G.V.
Statistical analysis — Kalaeva D.B., Danilov G.V.
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