Burdenko's Journal of Neurosurgery
2026, No. 4, pp. 114–119
https://doi.org/10.17116/neiro202690031114

Evolution of methodological standards for systematic reviews and meta-analysis protocol on the influence of molecular genetic profile of meningioma on tumor recurrence

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ABSTRACT

Background. Traditional histological classification of meningiomas has low prognostic accuracy and necessitates integration of molecular biomarkers. However, current data are fragmented and require rigorous synthesis.

Objective. To develop a meta-analysis protocol for quantitative assessment of the influence of molecular profile of meningiomas on long-term outcomes (overall survival and recurrence-free period) after surgical or combined treatment.

Material and methods. The article provides historical analysis of transformation of evidence-based medicine standards from narrative reviews to algorithmic guidelines (QUOROM, MOOSE, PRISMA). The choice of the PRISMA-P 2020 protocol and Cochrane methodology as the standard for this study is justified.

Results. A meta-analysis design was developed to assess the impact of genetic profile (*TERT, CDKN2A/B, NF2* status, DNA methylation) on the risk of recurrence. Inclusion criteria (PICOTS) cover all types of meningiomas. The protocol regulates searching strategy, independent screening (Rayyan) and risk of bias assessment using the QUIPS tool. Statistical synthesis of data is planned to identify the hierarchy of risk factors.

Conclusion. Implementation of this protocol will ensure creation of a valid evidence base for stratification of risk of meningioma recurrence based on molecular genetic profile.

Keywords: *meningioma, tumor recurrence, molecular genetic profile, progression risk stratification, DNA methylation-based classification, CDKN2A/B deletion and TERT, systematic review and meta-analysis.*

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TO CITE THIS ARTICLE:
Sergeev GS, Nesterchuk MV, Klimov VS, Gaytan AS, Kuftov VV, Danilov GV, Konovalov NA, Usachev DYu, Krivoshapkin AL. Evolution of methodological standards for systematic reviews and a meta-analysis protocol on the influence of the molecular genetic profile of meningiomas on tumor recurrence. *Burdenko's Journal of Neurosurgery.* 2026;90(4):114–119. (In Russ.). https://doi.org/10.17116/neiro202690041114

Introduction

Meningiomas are the most common primary intracranial neoplasms (41.7% of all tumors and 56.8% of all benign tumors) [1]. Their prevalence associated with aging population and advanced neuroimaging techniques increased clinical significance of this problem [2].

For decades, a three-stage WHO grading system based on histological features underlay classification of meningiomas. Despite its prognostic value, clinical outcomes often

contradict histological grade [3—5]. Grade I meningiomas can recur in 7—25% of cases, while up to 50% of Grade II tumors demonstrate indolent course. This discrepancy, compounded by subjective histological assessment, creates uncertainty in choice of treatment strategies (especially adjuvant therapy) [6—8]. The current model does not allow for accurate risk stratification and necessitates more significant molecular biomarkers to avoid treatment errors.

Simpson grade of resection is one of the most significant predictors of recurrence [3]. However, long-term recurrence rate remains at 20—30% throughout 10—15 years even after macroscopically total resection (Simpson grade I) [9]. In some cases, total resection is impossible due to involvement of critical neurovascular structures [10].

These limitations of classical systems inspired large-scale molecular studies. The last ones laid foundation for a new classification and treatment paradigm for meningiomas.

Some genetic mutations are included in the current 2021 WHO classification of meningiomas (WHO CNS5): *SMARCE1* — clear cell subtype, *BAP1* — rhabdoid and papillary subtype, *KLF4/TRAF7* — secretory subtype, *TERT* promoter mutation and/or homozygous deletion of *CDKN2A/B* — Grade III meningiomas [11]. In previous review, we demonstrated that malignant progression of meningiomas follows the theory of clonal evolution with accumulation of genetic aberrations [12]. Grade I is characterized by mutations in chromosome 22q and genes *NF2*, *AKT1*, *KLF4*, PIK3CA, *SMO*, *TRAF7*, *POLR2A*, as well as angiogenesis factors. Grade II is characterized by deletions of chromosome arms 1p, 6q, 9p, 10q, 14q, and 22q, as well as *SMARCE1* mutation. Grade III is associated with loss of 9p, gain of 17q23, and mutations in *TERT* and *CDKN2A/B*.

Discrepancy between formal classification criteria and actual clinical outcomes when assessing markers of Grade III meningiomas is particularly noteworthy. Although the current WHO classification equates prognostic significance of *TERT* mutations with *CDKN2A/B* deletions, available clinical data indicate a strict prognostic hierarchy between the two. Aggressiveness of *TERT*-mutant tumors is often due to concomitant loss of *CDKN2A/B*. *CDKN2A/B* deletion is a more powerful independent predictor. Median disease-free survival with *CDKN2A/B* deletion is 26.1 months vs. 180 months for wild-type tumors. This demonstrates fundamental role of *CDKN2A/B* that is not yet fully reflected in the WHO classification [13, 14].

Spinal and certain intracranial meningiomas have unique mutational profiles that influence the likelihood of recurrence and sensitivity to adjuvant chemo- and radiotherapy [15, 16].

The integrated models combining WHO grade, methylation class and gene copy number changes are a modern approach to risk prediction. However, implementation of integrated models faces challenges with standardization, cost and availability of tests. This poses a significant obstacle to their widespread clinical application. Literature data are scattered, and authors most often focus on specific mutations and their impact on carcinogenesis.

Modern biomedical science including neuro-oncology is characterized by a shift from accumulation of disparate empirical data to rigorous algorithmic synthesis. Historical tradition of writing narrative reviews [17] is being replaced by systematic reviews and meta-analyses. The last ones are free of methodological shortcomings such as lack of clear inclusion and exclusion criteria, data searching strategy protocols and predominance of qualitative assessments. In context of meningiomas, such methodology generated conflicting clinical recommendations and, as a consequence, led to variability in treatment outcomes.

The first large-scale initiative aimed at standardizing the reporting of meta-analyses was the QUOROM (QUality Of Reporting Of Meta-analyses) guidelines published in 1999 [18]. These guidelines were strictly limited to randomized controlled trials. This limitation was critical for neuro-oncology, and especially for research of prognostic factors. Indeed, analysis of genetic markers cannot be conducted in design of randomized trials for ethical and practical reasons. The MOOSE (Meta-analysis Of Observational Studies in Epidemiology) guidelines were developed in 2000. This standard was specifically focused on observational studies [19]. In 2005, the REMARK (REporting recommendations for tumor MARKer prognostic studies) guidelines were published as a standard for assessing the quality of articles included in systematic reviews. All these standards were fragmented and did not allow for a comprehensive approach to meta-analysis. A response to fragmentation of standards was the PRISMA guidelines (2009) (Preferred Reporting Items for Systematic Reviews and Meta-Analyses). These recommendations integrated best practices for analyzing and synthesizing literature data. Their updated version (2020) has finally cemented status of these guidelines as a universal tool. Unlike QUOROM guidelines, PRISMA allows for protocol changes throughout the study, provided they are strictly documented and justified. This is especially important for genetic reviews, where new data may require *post-hoc* adjustments to searching strategy. Furthermore, PRISMA shifted the focus from formal Quality Assessment to Risk of Bias Assessment. This is a more accurate tool for refining evidence level [20]. While PRISMA prescribes how to write a report, Cochrane methodology dictates how to conduct a study to increase analytical rigor. This methodology utilizes the MECIR (Methodological Expectations of Cochrane Intervention Reviews) extensive literature search strategy covering not only bibliographic databases but also clinical trials and dissertations.

We present a protocol for systematic review and meta-analysis of data on genetic profile of meningiomas using the PRISMA guidelines. This prospective systematic review aims to examine the impact of three key factors on tumor recurrence: resection quality, tumor type (Grade I–III) and genetic profile. The last factor can significantly adjust treatment approaches and determine mutational composition of genetic diagnostic panel.

Table 1. **Inclusion/exclusion criteria based on the PICOTS system.**

Component	Description	Justification
Population	Adults (>18 years) Any localization Grade I–III	Inclusion of all malignancy grades is necessary to assess the ability of genetic markers to reclassify risks
Index Factor	Specific somatic mutations or aberrations determined by IHC, FISH, NGS, Sanger methods	Coverage of a wide range of markers allows for comparative analysis of their predictive power
Comparator	Patients with wild type genes	Control group is mandatory for analysis of relative risks
Outcome	Primary: freedom from recurrence Secondary: overall survival	Freedom from recurrence is the clearest indicator of biological aggressiveness of tumor
Timing	Prognosis assessment time: postoperative. Prognosis period: long-term (>3 years)	Short-term (<3 years) studies can underestimate risks
Setting	Neurosurgical and cancer hospitals	Data from specialized centers ensures higher quality of surgery and morphology

Note: FISH — fluorescence in situ hybridization; Sanger — Sanger sequencing; IHC — immunohistochemistry; NGS — next-generation sequencing.

The purpose of the study was to rationalize a study design devoted to quantitative assessment of impact of molecular genetic profile of meningiomas on long-term outcomes (overall survival and freedom from recurrence) after surgical or combined treatment.

Material and methods

Study design and data collection

The methodology was developed in compliance with the PRISMA 2020 guidelines. The protocol was developed according to the Cochrane methodology.

Inclusion criteria

Study design and selection criteria were defined using the PICOTS (Population, Intervention, Comparator, Outcome, Timing, Setting) system **(Table 1)**. The review included publications devoted to all types of primary and recurrent meningiomas, as molecular profiling is critical for risk reclassification (identifying aggressive Grade I and indolent Grade II forms).

We will exclude reviews, meta-analyses, case reports, in vitro studies and trials where molecular data are not accompanied by assessment of clinical outcomes.

Databases and searching strategy

Searching will be performed in major electronic databases: PubMed, Web of Science, Embase, Google Scholar, eLibrary and Scopus. Searching strategy is developed using dictionary terms (MeSH for PubMed and Emtree for Embase) and free-text searching. An expanded set of keywords, including general terms («genetic profile», «mutation») and specific genes (*CDKN2A/B*, *TERT*p, *NF2*, *AKT1*), is essential to achieve maximum sensitivity and sample completeness. Examples of keywords: meningioma AND («genetic profile» OR «gene expression» OR «mutation» OR «genomic analysis») AND («tumor recurrence» OR «recurrence risk» OR «recurrence») AND (*CDKN2A/B* or *TERT* or *NF2*).

In addition to traditional databases, we will use Publish or Perish software (https://harzing.com/resources/publish-or-perish). It enables a structured searching in Google Scholar and other open sources with mandatory filters by publication date (after 2010) and keywords.

Study selection

Studies will be selected according to the PRISMA guidelines. Deduplication is performed in Zotero (https://www.zotero.org/), independent two-stage screening — on Rayyan platform (https://www.rayyan.ai/). Reviewers sequentially assess titles/abstracts and full texts for compliance with PICOTS criteria. Any disagreements are resolved through consensus or by a third reviewer.

Data extraction

Two independent reviewers will extract data using a standardized form. Hierarchy of molecular alterations is of particular attention. Not only mutation per se, but also its status (e.g., homozygous *CDKN2A/B* deletion and/or *TERT* mutation) and DNA methylation class are considered. Indeed, this integrated classification more accurately stratifies risks. Extracted variables include study design, demographic data, clinical parameters (WHO Grade, Simpson Grade, therapy), molecular profile, outcomes (freedom from recurrence, overall survival), and statistical measures of effect (hazard ratio (HR) with 95% confidence interval) **(Table 2)**.

Table 2. **Parameters extracted from full-text articles.**

Domain	Parameter	Data	Note
Identification and design	Author, year, country	Text	Necessary to assess publication bias
	Study design	Category	Randomized controlled trial, prospective/retrospective cohort
	Total number of patients (*n*)	Number	Total *n* and *n* in each subgroup (genetic status)
	Follow-up (median)	Months/ years (±SD/IQR)	
Patient characteristics	Age	Number (median / mean)	
	Gender	% (men : women)	
	Functional status	Number	Karnofsky scale or WHO performance status scale
Tumor characteristics	2021 WHO Grade	I, II, III	Mandatory co-founding for HR adjustment
	Resection grade	Simpson Grade I–V	N of patients with total resection
	Tumor localization	Category	Convexital, skull base, spinal
Molecular profile	NF2	Mutation/wild type	
	TERT	Mutation/wild type	
	CDKN2A/B	Category	Homozygous, heterozygous deletion or wild type
	Other key mutations	Category	*AKT1, KLF4, TRAF7, BAP1* and others
	Classification of methylation	Category	Low-Risk, Intermediate-Risk, High-Risk (if applicable)
	Method of analysis	Text	NGS, Array methylation (for example, Infinium), IHC
Primary outcomes	Tumor recurrence	%	Number of events /*n* for each group
	PFS	Months/HR	Median PFS (95% CI); number of events PFS
Secondary outcomes	OS	Months /HR	Median OS (95% CI); number of events OS
	HR	Number (HR, 95% CI)	Preferably adjusted HR taking into account Grade and Simpson Grade
	Side effects	%	New neurological deficits, complications

Note: SD — standard deviation; IQR — interquartile range; PFS — progression-free survival; CI — confidence interval.

Risk of bias assessment

The Quality in Prognosis Studies (QUIPS) tool with update and traffic light diagram for randomized controlled trials will be used to assess methodological quality of studies [21]. Unlike tools for assessing randomized controlled trials or interventional cohort studies, this tool most accurately assesses potential threats to validity of prognostic cohort studies.

Two reviewers will independently assess each study against seven QUIPS domains: domain 1 — confounding (influence of confounding factors); domain 2 — patient selection and inclusion; domain 3 — intervention (treatment/surgery or not); domain 4 — protocol discrepancy; domain 5 — missing data; domain 6 — endpoint assessment; domain 7 — presentation of results. Domain 1 («Confounding factors») will be of particular attention, as it is the most common source of high-risk bias in prognostic literature devoted to meningiomas.

One of the central issues in this area is under-recognition of traditional prognostic factors, such as resection quality (Simpson Grade) and WHO grade. These factors are powerful predictors of recurrence regardless of genetic status. Therefore, studies should use multivariate models to derive adjusted HRs for clinically meaningful results. The protocol prioritizes synthesis of adjusted HR. If a study does not provide adjustment data, its prognostic value is low.

Statistical analysis and meta-analytic synthesis

A meta-analysis will be advisable for sufficient number of studies with clinically and methodologically homogeneous data. If quantitative analysis will be impossible, we will present a detailed narrative description of results.

To analyze progression-free and overall survival rates, we will pool the logarithms of hazard ratios and their standard errors in a random-effect model using inverse variance. For dichotomous outcomes, we will summarize hazard ratios or odds ratios using the DerSimonian and Laird method and random-effect model [22, 23]. If HRs and their variances will be unavailable in articles, we will attempt to estimate these parameters using available Kaplan–Meier curves and data on the number of patients and events [24].

Statistical heterogeneity will be assessed using the I2 statistic and the Pearson χ^2 test. Significant

heterogeneity (I2 >50% or $p<0.10$) will require further analysis of causes through subgroup analysis and meta-regression. Analysis will be performed using SPSS statistical software (IBM SPSS Statistics; IBM Corp., USA).

Heterogeneity assessment and subgroup analysis

Considering high biological and clinical variability of meningiomas, a thorough subgroup analysis is an integral part of our protocol. Subgroup analysis will be based on the following key parameters:

1. Molecular status: comparison of prognostic value of the *TERT* promoter mutation versus wild-type (*TERT*p-WT), assessment of isolated or combined effect of homozygous *CDKN2A/B* deletion, comparison of the major molecular subtypes (*NF2*-mutant, *KLF4/TRAF7*-mutant, *BAP1*-mutant, etc.). If sufficient data will be available, DNA methylation classes will be assessed.
2. Clinical factors: resection quality (Simpson Grade I versus II-V) and WHO grade.
3. Tumor localization: subdivision into convexital, parasagittal, basal/skull base and spinal meningiomas.

If combination of HRs for different molecular subtypes will be possible, we plan to use ranking methods similar to those used in network meta-analysis (NMA). Mean Ranks and Surface Under the Cumulative Ranking Curve Area (SUCRA) will be calculated. This approach will allow us not only to establish prognostic significance of certain markers, but also to quantitatively rank molecular profiles by their impact on progression-free and overall survival.

Meta-regression (if the number of studies exceeds 10) will be used to examine the influence of covariates.

Assessment of bias and evidence base

Publication bias will be assessed using funnel plots and statistical tests such as Egger's test if meta-analysis will include more than 10 studies for each outcome.

Overall evidence base for each primary outcome and prognostic factor will be assessed using the GRADE (Grading of Recommendations, Assessment, Development, and Evaluation) system. Evidence level will be downgraded in case of high risk of bias (especially in domain of incorrect accounting for confounding according to QUIPS), imprecision (wide confidence intervals), indirect evidence, or significant inconsistency between studies).

Conclusion

Implementation of this protocol will ensure significant evidence base for stratifying the risk of meningioma recurrence based on molecular profiling.

Author contribution:
Concept and design of the study — Sergeev G.S., Nesterchuk M.V., Klimov V.S., Konovalov N.A., Usachev D.Yu., Krivoshapkin A.L.
Collection and analysis data, statistical analysis — Sergeev G.S.
Writing the — Sergeev G.S.
Editing — Klimov V.S., Gaitan A.S., Kuftov V.V., Danilov G.V., Krivoshapkin A.L.

No conflict of interests to declare.

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Received 16.01.2026
Revised 09.03.2026
Accepted 07.05.2026