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Early DCE-MRI changes and concurrent radiologic response after stereotactic radiosurgery for brain metastases: a prospective exploratory study

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Abstract

Background Conventional MRI may show limited or equivocal changes in the early period after stereotactic radiosurgery (SRS) for brain metastases. This study evaluated whether early dynamic contrast-enhanced MRI (DCE-MRI) changes at 4–8 weeks after SRS are associated with concurrent early radiologic response in brain metastases.

Methods This prospective, exploratory, lesion-level study included 17 brain metastases from 12 consecutive patients treated with single-session Gamma Knife SRS. DCE-MRI was performed before treatment and at 4–8 weeks after SRS using a 1.5-T MRI system. Quantitative parameters (Ktrans, Ve, Kep, and initial area under the concentration–time curve [iAUC]) were derived using the Tofts pharmacokinetic model, and semiquantitative parameters (upslope and initial gradient) were obtained from signal-intensity curves. Response was classified according to RANO-BM criteria. Pre-to-post changes, between-group differences in absolute change, and exploratory associations with response category were analysed using Wilcoxon signed-rank, Mann–Whitney U, and Spearman correlation tests, respectively. Benjamini–Hochberg false discovery rate correction was applied within each test family.

Results Six lesions (35.3%) were classified as partial response and 11 (64.7%) as stable disease. Ktrans, Kep, and iAUC decreased significantly after treatment and remained significant after false discovery rate correction. Initial gradient decreased in the unadjusted analysis ($p=0.044$) but did not survive correction. Ktrans showed a greater absolute reduction in partial response than in stable disease in the unadjusted analysis ($p=0.020$), but no between-group comparison survived correction. Percentage changes in Ktrans (rho = −0.553, $p=0.021$) and upslope (rho = −0.503, $p=0.040$) were associated with concurrent early radiologic response in unadjusted analyses; no correlation finding survived correction. In a one-lesion-per-patient sensitivity analysis, the direction of the Ktrans difference was consistent with the lesion-level analysis.

Conclusions Early DCE-MRI changes were detectable within 4–8 weeks after SRS. Ktrans showed the most consistent unadjusted association with concurrent early radiologic response. These hypothesis-generating findings require validation in larger, adequately powered, and methodologically standardised cohorts before clinical application.

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Keywords Dynamic contrast-enhanced MRI · Stereotactic radiosurgery · Brain metastases · Ktrans · Treatment response assessment

Introduction

Brain metastases (BM) are the most common intracranial tumours in adults and remain a major cause of morbidity and mortality in patients with systemic cancer [1–3]. Improved systemic therapies have extended survival, increasing the likelihood of detecting BM earlier in the disease course. This has heightened the need for effective local treatment and reliable methods for response assessment.

Stereotactic radiosurgery (SRS) delivers a high dose of radiation to a precisely defined target while minimising exposure to surrounding normal brain tissue [4, 5]. In selected patients, SRS provides local tumour control and may offer advantages over whole-brain radiotherapy [6]. However, conventional MRI in the early period after SRS may show limited or equivocal morphological changes because of treatment-related effects, making size-based interpretation difficult [7]. Quantitative imaging approaches may therefore help characterise early post-treatment changes in patients with brain metastases treated with whole-brain radiotherapy, systemic therapy, or SRS [8, 9].

Dynamic contrast-enhanced MRI (DCE-MRI) is increasingly used for treatment response assessment [10]. It reflects tumour microvascular structure, vascular permeability, and blood–brain barrier integrity [11]. DCE-MRI also enables pharmacokinetic modelling of contrast agent leakage, allowing derivation of quantitative parameters such as the volume transfer constant (Ktrans), extravascular extracellular volume fraction (Ve), rate constant (Kep), and curve-based indices [12]. Semiquantitative parameters may also provide practical measures of early vascular change. In neuro-oncology, DCE-MRI parameters have been associated with treatment response and survival outcomes [13].

Early assessment of treatment response after SRS is important for guiding subsequent management [14]. DCE-MRI has been investigated for this purpose in several post-SRS studies [15–17]. Volumetric and perfusion changes after SRS have also been characterised across sequential follow-up intervals [18]. However, it remains unclear whether early perfusion changes are consistent across heterogeneous BM populations or whether semiquantitative parameters, such as upslope and initial gradient, provide additional value beyond standard pharmacokinetic measurements [19, 20].

Therefore, this study evaluated whether early changes in DCE-MRI parameters at 4–8 weeks after SRS were associated with concurrent early radiologic response in brain metastases. It also explored whether semiquantitative parameters, including upslope and initial gradient, provided additional response-related information beyond standard pharmacokinetic parameters.

Materials and methods

In this prospective, exploratory, lesion-level study, consecutive patients with brain metastases scheduled for stereotactic radiosurgery were enrolled. The study was approved by the local clinical research ethics committee, and written informed consent was obtained from all participants before study enrolment. A total of 17 lesions from 12 patients were included. Eligible lesions had typical MRI findings of brain metastasis and a long-axis diameter greater than 1 cm; all patients had a known primary malignancy. Patients were excluded if they had an uncertain differential diagnosis or a history of prior surgery or radiosurgery for brain metastasis. Lesions smaller than 1 cm were excluded.

MRI examinations were performed before treatment and repeated 4–8 weeks after stereotactic radiosurgery. No additional neurosurgical or lesion-directed oncologic treatment was initiated during the 4–8-week follow-up interval. Corticosteroid exposure and concomitant systemic treatment during this period were not systematically

analysed as study variables and therefore could not be evaluated as potential contributors to DCE-MRI changes. Patients were treated with single-session Gamma Knife SRS using a Leksell Gamma Knife Perfexion device with a marginal dose of 18–20 Gy. All MRI examinations were performed on a 1.5-T system (Magnetom Aera, Siemens Healthineers, Erlangen, Germany) using a 15-channel head coil. In addition to conventional sequences, DCE-MRI was performed in all patients. T2-weighted images were acquired for anatomical evaluation and treatment planning, and the protocol included whole-brain three-dimensional T1-weighted imaging followed by DCE-MRI acquisition.

The DCE-MRI protocol included precontrast T1 mapping followed by dynamic image acquisition. T1 mapping was performed using three-dimensional T1-weighted volumetric interpolated breath-hold examination (VIBE) sequences with flip angles of 2° and 15°. Dynamic imaging was performed using a three-dimensional T1-weighted gradient-echo VIBE sequence with a 12° flip angle. Thirty-six consecutive dynamic series were acquired in the axial plane with a temporal resolution of 7.1 s per series. In all cases, 0.1 mmol/kg gadobutrol (Gadovist®, Bayer Healthcare, Berlin, Germany) was administered intravenously using an automatic injector at 3 mL/s after acquisition of two baseline dynamic series, followed by a 15 mL saline flush. Detailed MRI acquisition parameters are provided in Supplementary Table 1.

The acquired image series were transferred to a workstation for review by two radiologists. Image post-processing was performed using the syngo MR software (version E11C; Siemens Healthineers, Erlangen, Germany). DCE-MRI data were analysed using the Tissue 4D application within this software environment, based on the Tofts pharmacokinetic model. Semiquantitative analysis was performed using the Mean Curve application within the same software environment. Tumour size, volume, and morphology were assessed on conventional MRI images. Tumour dimensions were recorded on postcontrast 3D T1-weighted images in the anterior-posterior (AP), mediolateral (ML), and craniocaudal (CC) planes. For volume calculation, tumour boundaries were manually delineated using the software tool, and tumour volume was automatically calculated from the manually delineated areas and slice thickness. The arterial input function was automatically defined by the software without manual arterial selection, which may affect the absolute accuracy of pharmacokinetic parameter estimates, particularly Ktrans. After automatic motion correction, precontrast images were reviewed together with the dynamic series. Signal intensity was converted into contrast concentration, and contrast–time curves were generated. After pharmacokinetic parameter maps were generated, regions of interest (ROIs) were manually drawn freehand to encompass the contrast-enhancing solid portions of the tumour. Cystic tumour components and normal vascular structures were excluded from the ROI. A standardised circular ROI was also placed in the contralateral normal-appearing brain parenchyma as a reference measurement. ROI placement was performed by consensus between two experienced radiologists; formal interobserver variability analysis was not performed. Quantitative parameters, including Ktrans, Ve, Kep, and initial area under the concentration–time curve (iAUC), were automatically derived from contrast curve modelling, and parametric maps were saved for all patients.

Lesion signal intensity–time curves were generated from the tumour ROIs. Upslope was calculated as the relative increase from baseline to maximum signal intensity divided by the elapsed time between these two points: $[(SImax - SIbaseline)/SIbaseline]/\Delta t$, where Δt was expressed in seconds. Initial gradient was calculated from the steepest ascending portion of the curve as $[(SImax - SImin)/SImin]/\Delta t$, where Δt represented the elapsed time in seconds between the two signal-intensity points defining that segment. Both parameters were therefore expressed in s^{-1}. All lesions were classified according to the Response Assessment in Neuro-Oncology Brain Metastases (RANO-BM) criteria [21]. At the 4–8-week follow-up, the observed response categories were partial response (PR) and stable disease (SD). Because no complete response (CR) or progressive disease (PD) was observed, between-group analyses are reported directly as PR versus SD.

Statistical analyses

Statistical analyses were performed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). All analyses were conducted at the lesion level. Of the 17 lesions, 9 were contributed by 4 patients with multiple metastases;

therefore, within-patient correlation was acknowledged but not formally modelled. To assess whether the direction of the main findings was affected by lesion clustering, a sensitivity analysis was performed using one lesion per patient. For patients with multiple lesions, the first-listed lesion in the dataset was retained to avoid outcome-driven selection, yielding a sensitivity sample of 12 lesions (4 PR and 8 SD). As this sensitivity analysis was intended to assess directional consistency rather than formal statistical significance, no false discovery rate correction was applied. Continuous variables are reported as median [IQR], and categorical variables as number and percentage. Normality was assessed using the Shapiro–Wilk test. Absolute parameter changes were defined as pre-treatment minus post-treatment values, such that positive values indicated post-treatment reduction. Pre-to-post comparisons were performed using the Wilcoxon signed-rank test, and between-group comparisons of absolute change were performed using the Mann–Whitney U test. Exploratory associations between percentage changes and the early radiologic response category were assessed using Spearman rank correlation. For this analysis, the response category was coded as an ordered binary variable. Because both DCE-MRI perfusion parameters and response classification were derived from the same 4–8-week post-treatment MRI examination, associations were interpreted as concurrent imaging-response relationships rather than outcome prediction. To address multiplicity, Benjamini–Hochberg false discovery rate adjustment was applied separately within each test family: pre-to-post comparisons, between-group comparisons, and correlation analyses, each including six tests. Unadjusted $p < 0.05$ was used as a descriptive threshold, and findings not surviving false discovery rate correction were considered hypothesis-generating.

Results

A total of 12 patients with 17 brain metastases were included. The mean patient age was 58.0 ± 12.1 years; six patients were male and six were female. Fourteen lesions (82.4%) were supratentorial and three (17.6%) were infratentorial. Eight patients had a single lesion, three had two lesions, and one had three lesions. At the 4–8-week follow-up, six lesions (35.3%) were classified as PR and 11 (64.7%) as SD; no CR or PD was observed (Table 1).

In the overall lesion-level analysis, Ktrans, Kep, and iAUC decreased significantly after treatment and remained significant after Benjamini–Hochberg correction. Initial gradient decreased in the unadjusted analysis (median 0.047 to 0.033; $p = 0.044$) but did not survive false discovery rate correction (BH-adjusted $p = 0.066$). Ve and upslope did not change significantly (Table 2).

Baseline quantitative and semiquantitative DCE-MRI parameters did not differ between PR and SD lesions. In the unadjusted between-group analysis of absolute changes, Ktrans showed a greater reduction in PR than in SD lesions (PR: median Δ 0.238 [IQR 0.214–0.279]; SD: median Δ 0.055 [IQR 0.043–0.091]; $p = 0.020$). The between-group difference in upslope change did not reach the unadjusted descriptive threshold ($p = 0.063$; BH-adjusted $p = 0.189$). No significant between-group differences in absolute change were found for Ve, Kep, iAUC, or initial gradient. No between-group comparison survived false discovery rate correction (Table 3).

In exploratory correlation analyses, percentage changes in Ktrans (rho = −0.553, $p = 0.021$) and upslope (rho = −0.503, $p = 0.040$) were associated with the early radiologic response category in unadjusted analyses. No other parameter reached the descriptive threshold (Ve: rho = −0.050, $p = 0.848$; Kep: rho = −0.201, $p = 0.439$; iAUC: rho = −0.226, $p = 0.383$; initial gradient: rho = −0.075, $p = 0.774$). None of these associations survived false discovery rate correction (Table 4).

In the sensitivity analysis using one lesion per patient ($n = 12$), Ktrans change was greater in partial response than in stable disease (PR: median Δ 0.271 [IQR 0.247–0.329]; SD: median Δ 0.064 [IQR 0.047–0.113]; $p = 0.004$). Spearman correlation between Ktrans percentage change and response category was rho = −0.819 ($p = 0.001$) in the sensitivity sample (Table 5).

Table 1 Baseline patient and lesion characteristics

Variable	Value
Patient characteristics (n = 12)	
Age, years — mean ± SD†	58.0 ± 12.1
Sex, n (%)	
Male	6 (50.0)
Female	6 (50.0)
Primary tumour type, n (%)	
Lung cancer	7 (58.3)
Breast cancer	5 (41.7)
Lesion characteristics (n = 17)	
Location, n (%)	
Supratentorial	14 (82.4)
Infratentorial	3 (17.6)
Number of lesions per patient, n (%)	
1 lesion — 8 patients	8 (66.7)
2 lesions — 3 patients	3 (25.0)
3 lesions — 1 patient	1 (8.3)
Response category (RANO-BM), n (%)	
CR	0 (0.0)
PR	6 (35.3)
SD	11 (64.7)
PD	0 (0.0)

CR = complete response; PR = partial response; SD = stable disease; PD = progressive disease; RANO-BM = Response Assessment in Neuro-Oncology Brain Metastases. Data presented as mean ± SD or n (%). † Age is reported at the patient level

Table 2 Pre- and post-treatment DCE-MRI parameters

Parameter	Pre-treatment Median [IQR]	Post-treatment Median [IQR]	p	BH-adj p
Ktrans (min^{-1})	0.244 [0.159–0.350]	0.121 [0.100–0.148]	0.005*	0.009*
Ve	0.526 [0.316–0.644]	0.388 [0.278–0.454]	0.174	0.209
Kep (min^{-1})	0.530 [0.403–0.713]	0.329 [0.289–0.386]	0.003*	0.008*
iAUC	20.24 [11.07–32.81]	9.45 [7.83–13.98]	<0.001*	<0.001*
Upslope (s^{-1})	0.022 [0.016–0.032]	0.015 [0.012–0.024]	0.227	0.227
Initial Gradient (s^{-1})	0.047 [0.036–0.058]	0.033 [0.026–0.045]	0.044*	0.066

iAUC values are expressed in arbitrary units as derived by the Tissue 4D software. Median [IQR]. p values: Wilcoxon signed-rank test. BH-adj p: Benjamini–Hochberg false discovery rate adjustment within the pre-to-post comparison family. Ktrans, Kep, and iAUC remained significant after correction. Initial Gradient decreased (median 0.047 to 0.033) but did not survive correction. * $p < 0.05$ (unadjusted)

Table 3 Absolute change (pre-treatment − post-treatment) in DCE-MRI parameters in PR and SD Lesions

Parameter	PR (n = 6) Median [IQR]	SD (n = 11) Median [IQR]	p	BH-adj p
Ktrans (min^{-1})	0.238 [0.214–0.279]	0.055 [0.043–0.091]	0.020*	0.121
Ve	0.119 [−0.081–0.345]	0.079 [−0.052–0.275]	0.808	0.961
Kep (min^{-1})	0.243 [0.127–0.842]	0.109 [0.045–0.239]	0.256	0.512
iAUC	11.12 [2.69–23.59]	6.33 [1.89–15.03]	0.591	0.886
Upslope (s^{-1})	0.011 [0.004–0.014]	−0.003 [−0.006–0.006]	0.063	0.189
Initial Gradient (s^{-1})	0.007 [0.003–0.029]	0.012 [0.001–0.020]	0.961	0.961

Positive change indicates post-treatment reduction. iAUC values are expressed in arbitrary units as derived by the Tissue 4D software. p values: Mann–Whitney U test. BH-adj p: Benjamini–Hochberg false discovery rate adjustment within the between-group comparison family. No between-group comparison survived correction. * $p < 0.05$ (unadjusted)

Table 4 Spearman correlations between percentage changes in DCE-MRI parameters and PR/SD category

Parameter	Spearman's ρ_s	p (unadjusted)	BH-adj p
Ktrans (min^{-1})	−0.553	0.021*	0.119
Ve	−0.050	0.848	0.848
Kep (min^{-1})	−0.201	0.439	0.659
iAUC	−0.226	0.383	0.659
Upslope (s^{-1})	−0.503	0.040*	0.119
Initial Gradient (s^{-1})	−0.075	0.774	0.848

Percentage change was calculated as (pre-treatment value − post-treatment value) / pre-treatment value using raw data. Negative ρ_s indicates that greater percentage reduction was associated with the partial response category. BH-adj p: Benjamini–Hochberg false discovery rate adjustment within the correlation family. No association survived correction. * $p < 0.05$ (unadjusted)

Table 5 Sensitivity analysis using one lesion per patient

Parameter	PR ($n=4$) Median [IQR]	SD ($n=8$) Median [IQR]	Mann–Whitney p	Spearman's ρ_s	Spearman p
Ktrans (min^{-1})	0.271 [0.247–0.329]	0.064 [0.047–0.113]	0.004	−0.819	0.001
Ve	0.309 [0.150–0.434]	0.085 [−0.037–0.265]	0.368	−0.358	0.253
Kep (min^{-1})	0.243 [0.194–0.454]	0.092 [−0.005–0.354]	0.368	−0.205	0.523
iAUC	21.57 [14.33–27.54]	9.88 [4.63–18.35]	0.283	−0.512	0.089
Upslope (s^{-1})	0.013 [0.011–0.016]	−0.001 [−0.006–0.008]	0.073	−0.615	0.034
Initial Gradient (s^{-1})	0.023 [0.009–0.040]	0.013 [0.001–0.024]	0.570	−0.256	0.422

For patients with multiple lesions, the first-listed lesion in the dataset was retained. Positive change indicates post-treatment reduction. iAUC values are expressed in arbitrary units as derived by the Tissue 4D software. No false discovery rate correction was applied because this analysis was used to assess directional consistency. With four and eight lesions per group, $p = 0.004$ is the smallest attainable two-sided Mann–Whitney value in this design and reflects complete separation rather than a stronger effect

Discussion

Early changes in DCE-MRI-derived perfusion parameters were detectable within 4–8 weeks after stereotactic radiosurgery. Among the evaluated parameters, Ktrans was the only between-group comparison to reach the unadjusted descriptive threshold, while percentage changes in Ktrans and upslope showed unadjusted exploratory associations with the concurrent early radiologic response category. No between-group comparison or correlation finding survived Benjamini–Hochberg correction; these findings should therefore be considered hypothesis-generating.

Early radiologic response was assessed according to the RANO-BM criteria [21]. At the 4–8-week follow-up, six lesions showed partial response and 11 showed stable disease; no lesion met criteria for complete response or progressive disease, which may be related to the short follow-up interval. PR lesions showed numerically greater absolute reductions in Ktrans, Kep, and iAUC than SD lesions; however, only the Ktrans between-group difference reached the unadjusted descriptive threshold, and no comparison survived false discovery rate correction. Initial gradient change did not differ between response groups. Exploratory correlation analyses showed unadjusted associations between percentage changes in Ktrans and upslope and the early radiologic response category, although neither association survived false discovery rate correction. Kep decreased after treatment, yet it did not distinguish PR from SD lesions in this cohort. Because Kep is derived from the relationship between Ktrans and Ve, parallel or variable changes in these parameters may limit its ability to separate early response groups.

The observed Ktrans reduction is consistent with the findings of Almeida-Freitas et al., who reported that increased Ktrans was associated with progressive disease, indicating that permeability-related metrics may be relevant to response assessment [15]. Previous studies suggest that vascular permeability may transiently increase during the acute post-radiotherapy period owing to endothelial injury and blood–brain barrier disruption, whereas subsequent reductions may reflect evolving vascular occlusion and treatment-related microstructural changes [15, 22, 23]. The 4–8-week interval was chosen to reduce the potential confounding influence of acute treatment

effects. Knitter et al. reported that post-treatment reductions in Ktrans and rCBV could differentiate pseudoprogression from progressive disease [24]. In the present cohort, Ktrans showed the most consistent unadjusted association with concurrent early radiologic response, although this response-related finding did not survive false discovery rate correction.

Jakubovic et al. reported that Ktrans values at week 1 were associated with treatment response, whereas this association was no longer significant at month 1 [16]. In the present exploratory analysis, an unadjusted between-group difference in Ktrans change was observed at 4–8 weeks. Volumetric reduction after SRS has been reported to be greatest within the first one to three months, whereas transient enlargement consistent with pseudoprogression occurred predominantly between three and eight months [18]. The available data therefore point to timing as an important source of variation in post-SRS DCE-MRI, and the optimal follow-up window remains uncertain.

Shah et al. reported that pretreatment Ktrans and Ve values were associated with early radiologic response, with higher values observed in the non-responder group [20]. The present analysis did not identify a similar pretreatment pattern, although the small sample size limits direct comparison. Differences in tumour composition, imaging timing, and response definitions may also contribute to the discrepancy.

Previous studies have suggested that heterogeneity-based Ktrans metrics, including Ktrans standard deviation, may be related to treatment progression [19]. Although such metrics may provide additional information, the present analysis focused on lesion-level summary values rather than histogram-based parameters. Future studies may clarify whether variance- or histogram-based approaches provide additional response-related information. Kapadia et al. also reported that Ktrans values decreased during the first month after treatment and were associated with post-treatment response [25]. Whether heterogeneity-based Ktrans metrics add value beyond mean estimates warrants evaluation in larger cohorts.

Senturk et al. evaluated early post-SRS DCE-MRI and dynamic susceptibility contrast-enhanced MRI parameters in lung cancer brain metastases [17]. They reported that early post-SRS absolute Ktrans was the strongest parameter associated with midterm (≥ 6 months) response. The present study differs by evaluating a heterogeneous brain metastasis cohort, incorporating semiquantitative DCE-MRI metrics, and focusing on concurrent early radiologic response at 4–8 weeks. The applicability of this finding to histologically heterogeneous cohorts and longer follow-up intervals still requires confirmation. A range of quantitative MRI approaches, including perfusion, diffusion, and delayed-contrast techniques, continues to be evaluated for post-SRS response assessment [26]. Recent exploratory studies in this setting remain limited by small, heterogeneous cohorts [27].

Primary tumour histology may also contribute to variability in DCE-MRI measurements. Ktrans has been reported to differ among histological subtypes of lung cancer brain metastases [28] and to vary with HER2 status in breast cancer brain metastases [29]. The present cohort included both lung and breast primaries, but the sample size did not permit histology-stratified analysis. Therefore, the potential contribution of tumour-specific vascular characteristics to the observed between-lesion variability cannot be excluded.

This study has several important limitations. The sample was small (17 lesions, 12 patients), and analyses were conducted at the lesion level; 9 of 17 lesions were contributed by 4 patients with multiple metastases, and within-patient clustering was not formally modelled. Because lesions from the same patient are not statistically independent, the lesion-level analyses may have overstated the amount of independent information and produced more optimistic inferential results than analyses that explicitly accounted for within-patient correlation. The one-lesion-per-patient sensitivity analysis showed a consistent direction of the Ktrans difference, but it was intended only as a sensitivity check and does not eliminate the limitations arising from the small number of patients and unmodelled within-patient correlation. The cohort included only lung and breast primary tumours, which limits generalisability to other histologies; in addition, the small sample precluded assessment of whether DCE-MRI changes differed according to primary tumour type. Early radiologic response was assessed using size-based radiological criteria without histopathological confirmation, as in routine post-SRS follow-up. Pharmacokinetic quantification used an automatically determined arterial input function without individualised arterial sampling [30], which may affect the absolute accuracy of Ktrans estimates. The study was conducted at a single centre using a single 1.5 T MRI platform and manufacturer-specific post-processing software, which may limit reproducibility

across institutions, scanners, and analysis platforms. Although 1.5 T platforms are widely used in clinical practice, their lower signal-to-noise ratio compared with 3 T systems may limit quantification precision. ROIs were placed by consensus between two radiologists; however, formal interobserver reproducibility was not assessed, and the potential influence of ROI placement on quantitative DCE-MRI measurements could therefore not be evaluated [31]. Together, automatic AIF selection, manual ROI placement, and vendor-specific post-processing may limit the reproducibility of quantitative DCE-MRI measurements across readers and technical platforms. The response endpoint was limited to the 4–8-week examination; therefore, the findings do not establish associations with long-term local control, progression, radionecrosis, or survival. The 4–8-week follow-up window introduced temporal variability, which may have contributed to differences in post-SRS DCE-MRI changes between lesions. Future studies should include larger multicentre cohorts, tumour-type stratification, longer follow-up, and standardised DCE-MRI acquisition and post-processing protocols to assess external reproducibility.

Conclusion

Early changes in DCE-MRI perfusion parameters were detectable within 4–8 weeks after stereotactic radiosurgery. Among the evaluated metrics, Ktrans showed the most consistent unadjusted association with the concurrent early radiologic response category, while percentage changes in Ktrans and upslope showed unadjusted exploratory associations; however, none of the between-group or correlation findings survived false discovery rate correction. These hypothesis-generating findings support further evaluation of early Ktrans change as a concurrent imaging correlate of early post-SRS response; its predictive value for subsequent clinical outcomes remains to be established in larger, adequately powered, and methodologically standardised cohorts.

Abbreviations

AP	Anterior-posterior
BH	Benjamini–Hochberg
BM	Brain metastases
CC	Craniocaudal
CR	Complete response
DCE-MRI	Dynamic contrast-enhanced magnetic resonance imaging
FDR	False discovery rate
iAUC	Initial area under the concentration–time curve
IQR	Interquartile range
Kep	Rate constant
Ktrans	Volume transfer constant
ML	Mediolateral
MRI	Magnetic resonance imaging
PD	Progressive disease
PR	Partial response
RANO-BM	Response Assessment in Neuro-Oncology Brain Metastases
ROI	Region of interest
SD	Stable disease
SRS	Stereotactic radiosurgery
Ve	Extravascular extracellular volume fraction
VIBE	Volumetric interpolated breath-hold examination

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Author contributions O.T. conceived and designed the study, collected the data, performed DCE-MRI analysis, interpreted the results, and drafted the manuscript. A.Y.Ö. supervised the study, contributed to study conception and design, and critically revised the manuscript for important intellectual content. E.C. contributed to the statistical methodology, performed the statistical analyses, and critically revised the manuscript. P.E. contributed to patient management, radiosurgery treatment planning, and critical revision of the manuscript. H.E. contributed to patient management, neurosurgical evaluation, and critical revision of the manuscript. All authors read and approved the final manuscript.

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Data availability The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate This study was approved by the Keçiören Training and Research Hospital Clinical Research Ethics Committee (approval no: 1910). The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants before study enrolment.

Consent for publication Not applicable.

Competing interests The authors declare no competing interests.

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