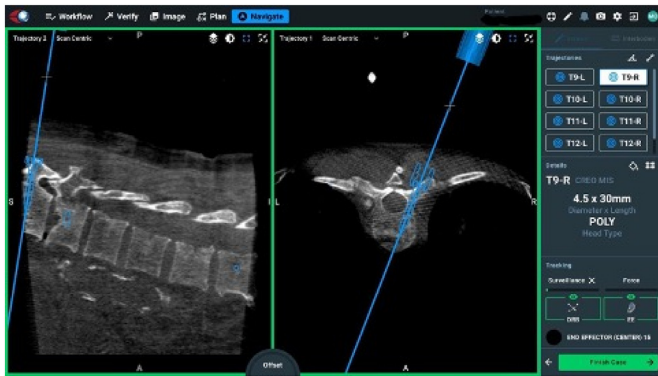


and optimal short/medium-term pain control.

Optional Image



1408

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A DISTINCT ENDOTHELIAL CELL POPULATION RESCUES THE METASTATIC PHENOTYPE OF B16 MELANOMA CELLS AFTER EPHRINB2-EPHB4 INTERACTION DEPLETION

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Oral e-Poster Presentations - Booth 3: Spine 2 (Tumors), September 26, 2023, 4:10 PM - 4:50 PM

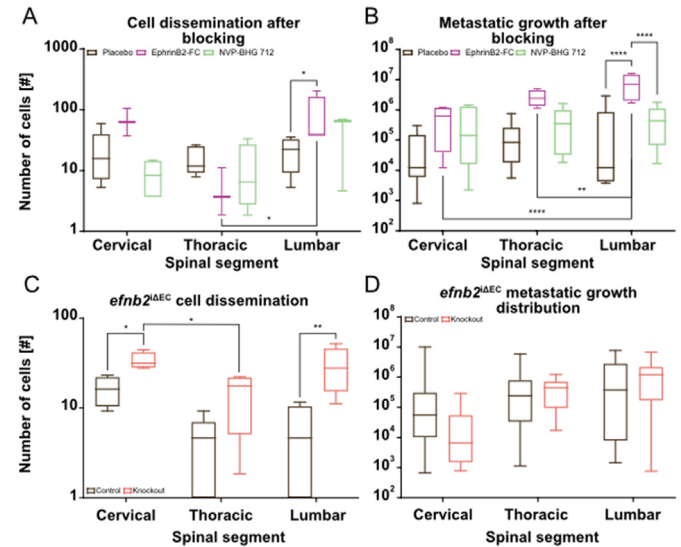
Background: Spinal metastasis remains a persistent and oftentimes urgent challenge in the neurosurgical operating room. We aim to understand metastatic spread to the spinal bone on a molecular level in endothelial cells and tumor cells to facilitate improved therapeutic approaches and diagnostics.

Methods: We established a murine syngeneic spinal bone metastasis model. In vivo dissemination was first evaluated using fluorescent beads, followed by murine cancer cell lines (B16, LLC1). We investigated short-term seeding and long-term growth to identify correlations between seeding and tumor formation. EphrinB2-Eph4 interaction has been described as a crucial mediator of spinal bone metastasis. Transient (pharmacological) and permanent (genetical) ephrinB2-Eph4 interventions were performed.

Results: Dissemination of microbeads to distinct spinal segments depended on segment and particle size. Disseminated tumor cells on the contrary showed less frequent arrest in the bone and equal distribution among segments. EphrinB2 intervention changed the dissemination behavior towards the lumbar segment. Interestingly, only transient intervention retained this distribution, permanent ephrinB2 depletion on endothelial cells (efnb2iΔEC) resulted in equal dispersion of metastases. Histological staining revealed a reduction of Endomucin (Emcn) positive structures in combination with a reduction of Type H (Emcn high/CD31 high) endothelial cells in naïve efnb2iΔEC animals. In tumor tissue, these Type H endothelial cells were unaffected. However, an increase in CD31-expressing endothelial cells was observed under endothelial ephrinB2 depletion. These CD31-expressing endothelial cells have been recently described as Type E (Emcn low/CD31 high) and implicated in angiogenesis and osteogenesis.

Conclusions: We here describe a subpopulation of endothelial cells in efnb2iΔEC mice that seems to resemble pro-angiogenic and possibly pro-adhesive type E endothelial cells. Based on these findings we propose a compensatory pro-angiogenic mechanism in efnb2iΔEC mice that is highjacking pre-existing developmental pathways, which is critical for late-stage spinal metastatic growth independent of the initial seeding and extravasation of metastatic cells.

Optional Image



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THE ROLE OF EPILEPSY IN ELDERLY PATIENTS WITH GLIOBLASTOMA: AN AUSTRIAN MULTICENTER ANALYSIS

Matthias Demetz¹, Constantin Hecker², Aleksandrs Krigeris¹, Lukas Machegger², Johannes Kerschbaumer¹, Johannes Pöppe², Philipp Geiger², Antonio Spinello¹, Christoph Griessenauer², Claudius Thomé¹, Christoph Schwartz², Christian Freyschlag¹. ¹Medical University Innsbruck, Austria; ²Paracelsus Medical University Salzburg, Austria

Oral e-Poster Presentations - Booth 3: Neuro-Oncology 1, September 25, 2023, 10:00 AM - 10:40 AM

Background: Higher age is a significant predictor of poor outcome in glioblastoma multiforme (GBM) patients. The impact of epilepsy in GBM patients on outcome parameters is poorly defined. Furthermore, persisting epilepsy significantly influences the patients' quality of life (QoL). This study aims at specifically evaluating the impact of epilepsy in elderly GBM patients.

Methods: Two Austrian academic neurosurgical centers retrospectively analyzed all elderly (≥ 65 years) GBM patients with de-novo tumors, who underwent surgery between 09/2006 and 07/2021. Epidemiological, histopathological and survival data were gained from patients' electronic charts. Tumor volume was assessed using standardized software.

Results: 391 patients (55%males, 45%females) with a median age at surgery of 73 years (Interquartile Range (IqR) 68.5-77.5) were analyzed. The mean predicted OS was 12.4 months (CI95% 10.9-14.0). Median preoperative Modified Rankin Scale (mRS) was 2 (IqR 1-3). Mean follow-up was 10.4 months (CI95% 9.1-11.6). Median tumor volume amounted to 26.47cm³ (IqR 12.65-43.49).

95/391 patients (24%) suffered from preoperative epilepsy. 17 (4,3%) patients still suffered from epilepsy after tumor resection. Eight patients developed new postoperative seizures, and four patients (1.0%) showed a worsening of already preoperatively diagnosed seizures. Major surgery-associated neurological complications included new motor deficits in 29 (7%) and new aphasia in 16 (4%) patients.

Patients with lower tumor volumes experienced significantly more often seizures compared to patients with larger tumors, $p < 0.001$.

Logistic regression showed patients with seizures had significantly lower mRS ($p = 0.032$) and less frequently occipital tumor location ($p = 0.018$). Moreover, they showed a significantly increased risk for postoperative aphasia ($p = 0.002$). Postoperative epilepsy resulted in significantly prolonged hospitalization after the surgery ($p = 0.009$).