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Conference Abstract

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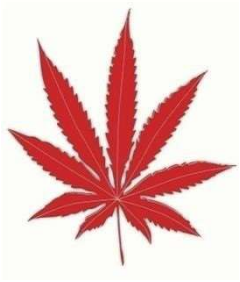

Conference Abstract

CHARACTERIZATION OF THE MINIMAL RESIDUAL DISEASE IN HUMAN GLIOBLASTOMA

MALEEHA QAZI

Ethics approval and consent to participate: No ethical approval needed for this research work.

Consent for publication: Author is agreed to submit this abstract for publication in this research journal.

Availability of data and materials: The information and data collected and/ or incorporated in this study is included in this manuscript.

Abstract

Despite aggressive standard-of-care (SoC) chemoradiotherapy, glioblastoma (GBM) remains incurable and inevitably relapses. Recent data has identified differential genomic and expression patterns between primary and recurrent GBM, suggesting that recurrence is a distinct biological entity. We hypothesized that profiling and characterizing the minimal residual disease post-SoC, a stage that remains elusive to our current diagnostic technologies, may offer an early window into identifying clonal composition and therapeutic targets in GBM recurrence.
Methods: Patient-derived GBM cells were barcoded using in-house cellular DNA barcode library and intracranially engrafted in immunocompromised mice. Using MRI, tumour volumes were monitored over the course of mouse-adapted SoC chemoradiotherapy and allowed for the identification of MRD timepoint. Clonal composition of GBM was assessed at multiple timepoints during the disease and treatment course by multi-plex barcode sequencing. Transcriptomic profiling of MRD cellular population was assessed using single-cell RNA-sequencing and validated using bulk RNA-sequencing and whole-cell proteomics.
Results: We developed a therapy-adapted xenograft model of GBM recurrence that can be used to study disease course at multiple timepoints throughout treatment course and disease progression. We successfully barcoded patient-derived GBM cells at single cells resolution to interrogate the temporal fate of distinct barcoded GBM subpopulations through SoC. We identified variable patterns of pre-existing and therapy-driven GBM subpopulations seeding different GBM tumour relapse, that correlated with patient survival. Through single-cell RNA-sequencing, we profiled the transcriptomic composition of GBM cells post-SoC at MRD and identified the impact of cell-state transitions in post-SoC in disease outcomes. We

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Competing Interest
The authors declare no competing interests.

Additional information is available at the end of the article.

also identified transcription and antigen-presentation signalling pathways as key mediators of survival outcomes in GBM patients.
Conclusion: Profiling the dynamic nature of heterogeneous GBM subpopulations through disease progression and SoC treatment may lead to the identification of novel targets in clonally evolving subpopulations of GBM for personalized treatment strategies

Key words: Glioblastoma, Minimal. Residual