Tumour cell dormancy as a contributor to the reduced survival of GBM patients who received standard therapy.


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Glioblastoma multiforme (GBM) is a fatal cancer with varying life expectancy, even for patients undergoing the same standard therapy. Identification of differentially expressed genes in GBM patients with different survival rates may benefit the development of effective therapeutic strategies. In the present study, key pathways and genes correlated with survival in GBM patients were screened with bioinformatic analysis. Included in the study were 136 eligible patients who had undertaken surgical resection of GBM followed by temozolomide (TMZ) chemoradiation and long-term therapy with TMZ. A total of 383 differentially expressed genes (DEGs) related to GBM survival were identified. Gene Ontology and pathway enrichment analysis as well as hub gene screening and module analysis were performed. As expected, angiogenesis and migration of GBM cells were closely correlated with a poor prognosis. Importantly, the results also indicated that cell dormancy was an essential contributor to the reduced survival of GBM patients. Given the lack of specific targeted genes and pathways known to be involved in tumour cell dormancy, we proposed enriched candidate genes related to the negative regulation of cell proliferation, signalling pathways regulating pluripotency of stem cells and neuroactive ligand-receptor interaction, and 3 hub genes (FTH1, GRM1 and DDIT3). Maintaining persistent cell dormancy or preventing tumour cells from entering dormancy during chemoradiation should be a promising therapeutic strategy.

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