Epithelial-to-mesenchymal transition is involved in BCNU resistance in human glioma cells.

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Abstract
Chemotherapy has been considered as an effective treatment for malignant glioma; however, it becomes increasingly ineffective with tumor progression. Epithelial-to-mesenchymal transition (EMT) is a process whereby cells acquire morphologic and molecular alterations that facilitate tumor metastasis and progression. Emerging evidence associates chemoresistance with the acquisition of EMT in cancer. However, it is not clear whether this phenomenon is involved in glioma. We used the previously established human glioma cell lines SWOZ1, SWOZ2 and SWOZ2-BCNU to assess cellular morphology, molecular changes, migration and invasion. We found that BCNU-resistant cells showed multiple drug resistance and phenotypic changes consistent with EMT, including spindle-shaped morphology and enhanced pseudopodia formation. Decreased expression of the epithelial adhesion molecule E-cadherin and increased expression of the mesenchymal marker vimentin were observed in BCNU-resistant SWOZ1 and SWOZ2-BCNU cells compared to SWOZ2 cells. Migratory and metastatic potentials were markedly enhanced in SWOZ1 and SWOZ2-BCNU cells compared to SWOZ2 cells. These data suggest that there is a possible link between drug resistance and EMT induction in glioma cells. Gaining further insight into the mechanisms underlying chemoresistance and EMT may enable the restoration of chemosensitivity or suppression of metastasis.


KEYWORDS: BCNU, EMT, chemotherapy, drug resistance, glioma

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