

Canada is unknown. We compared cohorts between 2 regional Canadian Cancer Centres for differences in patient factors, treatments, and outcomes. Methods Cohorts were constructed by a hybrid of retrospective chart review and prospective data collection consisting of all consecutive cases eligible for standard treatment. Demographics, pathology, treatment, and outcome data were obtained. Results The two cohorts (Winnipeg n=80 and Calgary n=103) were similar in terms of median age (57 and 56), percent male (62.5% and 63.1%), percent with good performance status (93.8% vs 85.4%) and extent of resection (gross total/sub-total/biopsy: 17.5%/66.3%/16.3% in Winnipeg and 7.8%/68.9%/23.3% in Calgary). Of patients with known MGMT promoter methylation status 28% were methylated in Winnipeg and 58% were methylated in Calgary. Greater than 6 cycles of chemotherapy was given to 42.5% of patients in Winnipeg and 28.1% in Calgary. The most common second line therapies differed: carboplatin and tamoxifen (31.3%) in Winnipeg; low dose temozolomide (26.2%) in Calgary. Significant poor prognostic factors for survival in the combined cohort included age (HR 1.02), extent of resection (sub-total HR 1.7; biopsy HR 8.9) and location (Calgary HR 1.17). Conclusion Comparison of cohorts from different parts of Canada can provide interesting descriptions of patterns of practice. These patterns may be useful in determining opportunities for quality improvement and clinical trial development.

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Glioblastoma multiforme in elderly and non-elderly patients in Newfoundland and Labrador: A province-wide six-year analysis.

E Morgan¹ and M Seal²

¹Department of Medical Oncology, University of Toronto, Toronto, Ontario; ²Discipline of Oncology, Faculty of Medicine, Memorial Hospital, St. John's, Newfoundland

Glioblastoma multiforme (GBM) is a devastating and generally incurable malignancy, with increasing incidence in elderly patients. Although advances in adjuvant chemoradiotherapy have shown promise in improving survival, the benefit of these therapies in elderly patients remains unclear. In this population-based retrospective study, we compared treatment patterns and outcomes in elderly (defined as age 65 or older) and non-elderly patients diagnosed with GBM in Newfoundland and Labrador. During 2006-2012, one-hundred-and-thirty-eight patients received a pathological diagnosis of GBM. Median age at diagnosis was 62.5 years (range 20-85) and 56(40.5%) were age 65 or older. Elderly patients were more likely to present with a performance status of ECOG 3 or greater ($p < 0.01$) and to undergo stereotactic biopsy rather than surgical resection ($p = 0.04$), and less likely to receive adjuvant temozolomide chemotherapy ($p < 0.001$). Presence of gross neurological defects and treatment with radiation therapy did not differ between elderly and non-elderly patients. Median survival was 245(CI[95%] 165-269) days for elderly patients versus 342(CI[95%] 274-440) days for non-elderly patients ($p < 0.01$). In Cox multivariate analysis, chemotherapy was associated with improved survival in elderly patients after adjusting for functional

status and extent of resection ($p < 0.001$) and was the strongest predictor of overall survival across patients in both age groups ($p < 0.001$). Despite receiving less aggressive surgery and chemotherapy, elderly patients showed evidence of improved survival with adjuvant temozolomide. These data support the growing body of evidence that adjuvant chemoradiotherapy may be beneficial in selected elderly patients with GBM.

SCIENTIFIC POSTER VIEWING

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SP1

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RXFP1 promotes temozolomide (TMZ) chemoresistance in brain cancer

T Thanasupawat^{1}, A Glogowska¹, H Bergen¹, J Stetefeld², J Krcek^{1,3}, S Hombach-Klonisch¹, T. Klonisch^{1,3}*

Depts. of ¹Human Anatomy and Cell Science, ²Chemistry, ³Surgery, University of Manitoba, Winnipeg, Manitoba

Current treatments for Glioblastoma (GB), the most aggressive form of primary brain cancer, include surgery, radiation and chemotherapy. TMZ is the most commonly used alkylating agent for GB treatment, but chemoresistance to TMZ is frequently an unsatisfactory treatment outcome. Relaxin Family Peptide Receptor 1 (RXFP1) mediates RLN2-induced cell migration and tissue invasion in many cancer entities including brain cancer. We have discovered RXFP1 expression in GB cells and tissues, but not in normal astrocytes. Down-regulation of RXFP1 in primary GB cells suppressed cell survival, cell invasiveness and induced cell death via a caspase3/7 mediated apoptosis pathway. Importantly, RXFP1- activation enhanced cell survival in primary GB cells treated with TMZ. To elucidate the mechanisms of RXFP1-mediated chemoresistance in GB cells, we identified the RXFP1-mediated up-regulation of anti-apoptotic proteins. In addition, several DNA repair proteins and Base Excision Repair (BER) members were regulated upon RXFP1 activation. Our results suggest that RXFP1 promotes TMZ chemoresistance by enhancing BER function and by suppressing apoptosis, thus, protecting primary GB cells from TMZ-induced DNA damage.

SP2

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Chloroquine inhibits the malignant phenotype of glioblastoma in vitro

L-O Roy¹, M-B Poirier², D Fortin²