



Predictors of Treatment Response of Cystic Brain Metastasis to Gamma Knife Radiosurgery

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Introduction

Gamma knife is an established treatment modality for treatment of solid brain metastases, but few studies have solely focused on the efficacy of treating cystic metastases with SRS. Two studies, by Franzin et al, 2008 and Higuchi et al, 2012, respectively, have shown favourable response to GKS for aspirated cystic tumours. A recent study by Ebinu et al. (2013) supports use of GKS for treating non-aspirated cystic metastases, showing comparable local control rates as non-cystic brain metastases.

Objective

To quantify the percentage of the cystic and solid components of cystic brain metastases pre/post GK to

I. Calculate the specific growth rate (SGR) as a measure of response of cystic component *And*

II. Identify factors predictive of response of cystic component

Table 1: Patient characteristics

Characteristic	Value
Gender (34 patients)	
Female	24 (70.6)
Age (range)	57.4 (33-76)
Median Follow up (Months)	12
Primary pathology (34 patients)	
Lung	20 (58.8%)
Breast	9 (26.5%)
Colorectal	5 (14.7%)
Location (52 lesions)	
Deep	16 (30.8%)
Superficial	36 (69.2%)
Prior WBRT (52 lesions)	
Distribution of lesions (n=52)	
Frontal	16 (30.8%)
Parietal	11 (21.2%)
Occipital	7 (13.5%)
Temporal	6 (11.5%)
Cerebellum	11 (21.2%)
Brainstem	1 (1.9%)

Methods

Database of 73 consecutive patients treated with GKS for brain metastases with cystic components, from 2006 – 2010

Inclusion criteria:

- Primary lung, breast, or colorectal cancer
- No prior GKS or surgical resection on lesions studied

- Complete dosimetric, clinical, and imaging (MRI + contrast) follow up available

Radiosurgical protocol: software; multidisciplinary team (neurosurgeon, radiation oncologist, and medical physicist)

Volumetric analysis: Volume of cystic & solid components measured on MRIs using ITK-SNAP

Supratentorial lesions: Superficial if > 60% of the tumour in the cortex (otherwise deep)
 Infratentorial lesions: Superficial if present in the posterior lobe of the cerebellum as seen on sagittal view.

$$\text{Specific Growth Rate} = \ln(V2/V1)/(t2-t1)$$

Results

Fig. 1. Median volume of the cystic components of brain metastases based on primary location. Metastatic lesions from the lung had significantly larger cystic components than metastasis of colorectal origin (p=0.039)

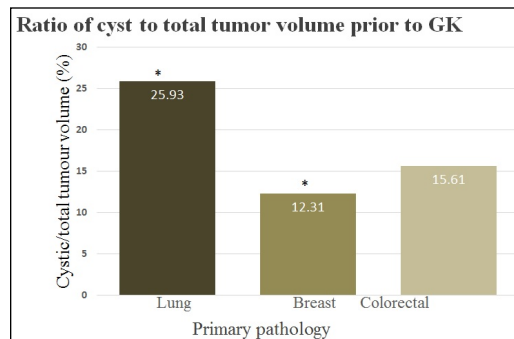
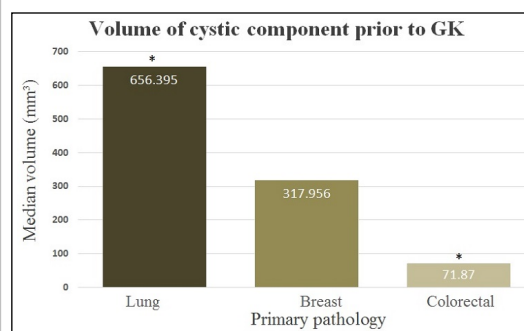


Fig. 2. The cystic component expressed as percentage of the total tumour volume, represented by sample median, compared by primary tumour pathology. Metastatic lesions from the lung had significantly larger cystic/total ratios than metastases from the breast (p=0.023).

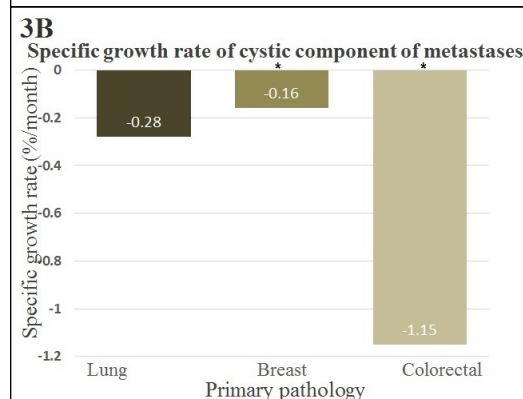
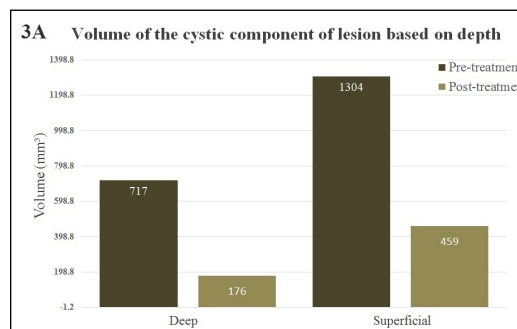
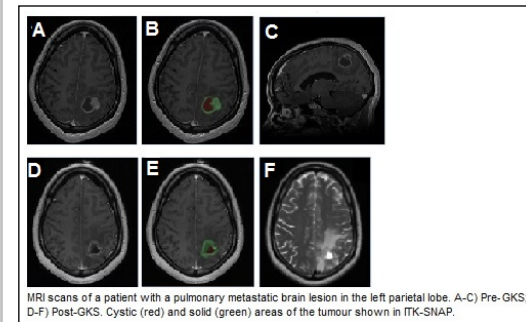


Fig. 3A. Deep tumours had lower cystic volumes pre-GKS than superficial tumours and significantly lower post-GKS cystic volumes (p=0.041). 3B) Specific growth rates (SGR) for cystic component of tumour; colorectal brain metastases demonstrated the best treatment response of the cystic component, significantly higher than those from the breast (p = 0.007)



Conclusions

Previously, we had shown that GK is effective for cystic brain metastases, with control rates comparable to a matched cohort of non-cystic metastases. However, the response of the cystic component was not specifically analyzed. This study shows:

- 1) Pre-treatment ratio of cyst to total tumor volume varied based on primary pathology Lung > Colorectal > Breast
- 2) Primary pathology influenced response of cystic component. Colorectal > Lung > Breast
- 3) Depth of tumor influenced response of cystic component.

GKS is a valid treatment option for cranial metastases with cystic components without prior aspiration and primary tumor type (colorectal) and depth of tumor location (deep) are predictors of better response for cystic brain metastases.

Limitations: A retrospective study, with only top three most common pathologies analyzed, leading to a small sample size. Other volumes (e.g. surrounding brain region, edema) were not measured.

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