8th BigBrain Workshop - Challenges of Multimodal Data Integration



Contribution ID: 56

Type: Poster

Glioma-Induced Alterations in Structural-Functional Connectivity Integration

Tuesday 10 September 2024 18:15 (45 minutes)

Gliomas alter white matter (WM) integrity[1] and grey matter (GM) functions[2] through intra-tumoral changes, invasion and expansion[3], affecting structural (SC) and functional (FC) connectivity. Nevertheless, many researchers investigated functional/structural/microstructural abnormalities within specific WM[4,5]-GM[6] regions, lacking an integrated overview[7,8].

This study aims to examine SC-FC link at the individual level, evaluating if alterations in this relationship may influence tumours effects on key brain networks, especially in association with tumours topological characteristics.

41 glioma patients were enrolled in the study. Images were acquired with a 3T Siemens Biograph mMR PET/MR scanner: dMRI images (b-values:0/710/2855s/mm2, 100-directions, MRtrix3[9]-pre-processing/tractography[10,11]-10Mstreamlines-iFOD2[12]), rs-fMRI (15min-TR/TE-1260/30ms-3x3x3mm3, state-of-the-art pre-processing[13,14]). An expert neuroradiologist provided lesion masks.

Connectivity matrices were derived with the Yan-homotopic-functional atlas (100-parcels/17-networks)[15]. Number-of-streamlines matrix (SCnos) was computed. Eight dMRI microstructure maps were quantified with NODDI[16]-DTI[17]-DKI[18] models, to be fused[19] into a single-microstructure matrix (SNFmicro). FC matrix represented Pearson correlations[15].

Integration Matrix (IM-100x300), to define an individual SC/FC measure, was derived concatenating the three single modalities (FC, SCnos and SNFmicro).

To evaluate the interplay between an integrated approach and single modalities, connectivity abnormalities were derived by single and integrated connectivity matrices.

For each connectivity type, pseudo-reference matrix was computed as the median across all the patients. Tracts and parcels overlapping with the lesion were removed by the computation. Then, Spearman correlation was computed to measure the parcel's connectivity similarity between each patient and its pseudo-reference group (row-by-row). Further, given the similarity distribution of all the subjects, a parcel was labelled as altered if the distance from the pseudo-healthy profile fell in the lower tail of the distribution (below 5%). Finally, the percentage of altered parcels within the same network represented the connectivity impairment of each network[20].

To investigate abnormalities-topology link, patients Network-Lesion distances were classified as Near/Far, according to the mean Euclidean distance between parcel-lesion centroids (threshold=76.17mm).

Panels A-B of Figure 1 illustrate, for networks Near/Far to the lesion, IM-alterations percentage across the subjects. Right-SalVentAttnB, Right-DefaultA and Right-LimbicB were the near networks more often altered among the patients (range 24%-29%). Left-DefaultA, Right-DefaultA and Right-SalVentAttnA were the networks far from the lesions more frequently altered across the patients (range 17%-27%). Just mentioned networks overlayed with higher frequency lesions position.

Panels A-B of Figure 1 also distinguish IM-alone (pink bar) and IM-in-overlap (grey bar) abnormalities. It is important to note that many networks highlighted both IM-alone and IM-in-overlap alterations. Networks near to the lesion were more frequently characterized by IM-alone rather than IM-in-overlap with single-modalities alterations (53%vs.32%).

These results suggest that, when performing studies on glioma, IM provides, especially for networks near to the lesion, a complementary view of connectivity changes. Hence, integration of brain functions with mi-

crostructure and structural integrity, could provide major insights about key networks alterations and highlights specifics topological-relevant tumours characteristics.

References:

- 1. https://doi.org/10.1093/brain/awac360
- 2. https://doi.org/10.1093/neuonc/noaa189
- 3. https://doi.org/10.1093/braincomms/fcaa216
- 4. https://doi.org/10.3390/cancers15143631
- 5. https://doi.org/10.3389/fonc.2022.998069
- 6. https://doi.org/10.3389/fonc.2020.00794
- 7. https://doi.org/10.1093/brain/aww194
- 8. https://doi.org/10.1155/2017/3530723
- 9. https://doi.org/10.1016/j.neuroimage.2019.116137
- 10. https://doi.org/10.1016/j.neuroimage.2012.06.005
- 11. https://doi.org/10.1016/j.neuroimage.2012.11.049
- 12. https://archive.ismrm.org/2010/1670.html
- 13. https://doi.org/10.1016/j.neuroimage.2004.07.051
- 14. https://doi.org/10.1016/J.NEUROIMAGE.2010.09.025
- 15. https://doi.org/10.1016/j.neuroimage.2023.120010
- 16. https://doi.org/10.1016/J.NEUROIMAGE.2012.03.072
- 17. https://doi.org/10.1016/S0006-3495(94)80775-1
- 18. https://doi.org/10.2214/AJR.13.11365
- 19. https://doi.org/10.1371/journal.pbio.3002314
- 20. https://doi.org/10.1152/jn.00338.2011

Primary author: COLPO, Maria (Padova Neuroscience Center, University of Padova, Padova, Italy; Department of Information Engineering, University of Padova, Padova, Italy)

Co-authors: BERTOLDO, Alessandra (Department of Information Engineering, University of Padova, Padova, Italy; Padova Neuroscience Center, University of Padova, Padova, Italy); CECCHIN, Diego (Department of Medicine, Unit of Nuclear Medicine, University of Padova, Padova, Italy; Padova Neuroscience Center, University of Padova, Padova, Italy; SILVESTRI, Erica (Department of Information Engineering, University of Padova, Padova, Italy); COR-BETTA, Maurizio (Department of Neuroscience, University of Padova, Padova, Italy; Padova Neuroscience Center, University of Padova, Italy)

Presenter: COLPO, Maria (Padova Neuroscience Center, University of Padova, Padova, Italy; Department of Information Engineering, University of Padova, Padova, Italy)

Session Classification: Poster Session