Multimodality imaging of Cerebral Schistosomiasis

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Introduction

Schistosomiasis is an endemic parasitic disease in dozens of countries with more than 200 million patients worldwide, although neuroschistosomiasis is uncommon. It is associated most with S. Mansoni and S. Japonicum. Symptomatic cerebral complications are reported in 2 - 4%. However, asymptomatic neuroschistosomiasis was seen in 25% of patients with hepatosplenic S. Mansoni infection at autopsy. In Europe and the USA schistosomiasis is a rare condition. [1]. Here we report the findings of multimodality imaging of a case of cerebral schistosomiasis and correlate these with the pathological findings.

Patient and methods

A 66 year old European female presented with a progressive right drop foot and spasm which started one year ago and spontaneously resolved with conservative therapy. After six months a progressive drop left foot with spasm developed. At this time the cerebral MRI showed a nodular parasagittal lesion, enhancing after gadolinium at the right primary motor cortex. She was planned for a neuronavigation biopsy. The neuronavigation MRI, five months after the first MRI, showed a progressive lesion at the left parasagittal primary motor cortex with a dissolving lesion at the right side. T2 weighted imaging showed peritumoral edema, especially left, expanding to the corpus callosum **(figure 1)**.

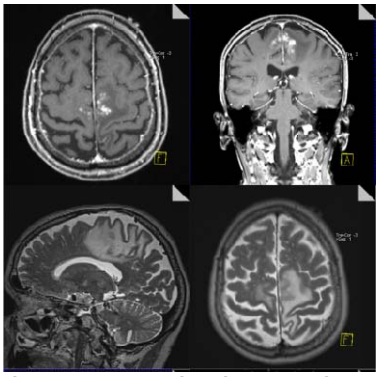

Figure 1. Conventional MR Imaging with contrast enhancement at T1 and T2 imaging.

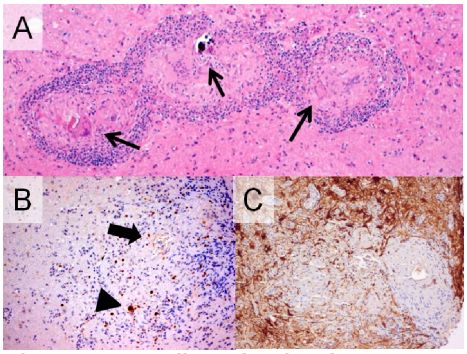

Figure 2. A. HE slices showing three granulomas with (partially degraded) eggs (small arrows). B. MIB-1 labeling (brown) showing increased index with mitosis (arrowhead) and egg (arrow). C. GFAP staining showing glioneuronal loss.

Next to the conventional MRI a perfusion weighted imaging (DSC, EPI, 6mm slices) and multivoxel 3D MR Spectroscopy (MRS) (semi-LASER [2], TE 30 ms, TR 1500 ms) were performed. After analyzing with LCModel the spectra with FWHM < 0.1 and SN > 10 were selected. Only metabolites levels with CRLB's $\leq$20% are used. At the same day a 3'-deoxy-3'-18F-fluorothymidine (18F-FLT) PET scan (emission after 60 minutes, 260 MBq, hybrid PET/CT, Siemens Biograph Duo Scanner) was performed.

Results and Discussion

The neuronavigation biopsy showed a granulomatous inflammation with worm eggs. Examination of the faeces revealed Schistosomia Mansoni. MIB-1 staining of the inflammatory cells was positive in up to 10%. The granulomas replaced the glioneuronal tissue and some granulomas showed necrosis **(figure 2)**.

The perfusion MR did not show evident increased cerebral blood volume or flow. The FLT-PET revealed an increased activity at the left side of the brain with a maximal standard uptake value (SUVmax) of 1.42 and lesion to background ratio of 4.7 (comparable to glioblastoma and lymphoma [3]). This activity is most probably attributed to the active proliferation of inflammatory cells, as confirmed by the MIB-1 labeling. There was a good correlation between the FLT activity and the gadolinium enhancement **(figure 3)**. Therefore, FLT-PET does not seem to be able to discriminate between proliferating tumor cells and active, proliferating inflammatory cells in the brain, which was earlier shown for lymph nodes in the neck [4]. The MR spectroscopy showed an increased Choline (Cho)/N-Acetyl-Aspartate (NAA) ratio and Cho/Creatine (Cr) ratio but with only a slightly increased absolute choline level, especially at the right side. There was a strong decreased NAA, especially at the left side and a slightly decreased Cr, corresponding to the glioneuronal loss. The combination with the increased lactate and lipids at 0.9 and 1.3 ppm confirms the more acute phase of inflammation at the left side and more chronic phase at the right side [5]. Gluthatione (GSH) and aspartate were increased at both sides, GSH more at the right side **(figure 4)**. GSH is an anti-oxidant which detoxificates reactive oxygen species and is especially found in astrocytes [6]. Asparte has not been associated previously with a parasitic infection. Although lower NAA is commonly associated with neuronal loss/displacement in these situations it also may have been degraded to aspartate and acetate explaining the increased aspartate. The increased GSH could be correlated with the more chronic inflammation at the right side.

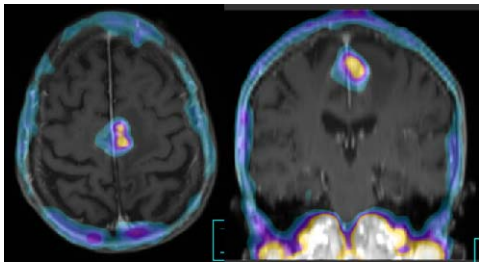

Figure 3. FLT-PET activity at the gadolinium enhanced lesions.

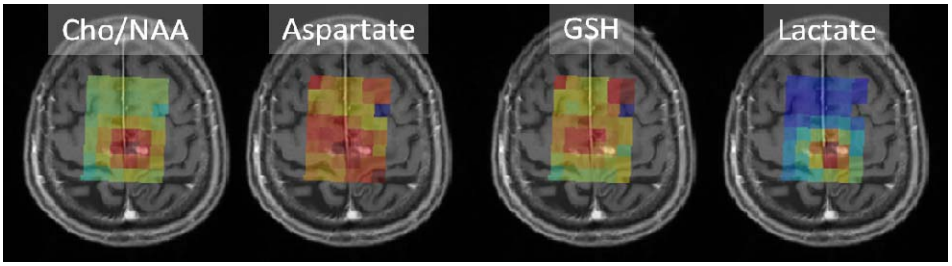

Figure 4. Metabolite maps of Cho/NAA, Aspartate, Glutathione and Lactate.

Conclusion

We describe the results of multimodality imaging of cerebral schistosomiasis. Conventional MRI shows nodular enhancement that is fairly typical for this disease [7]. The active infection resulted in an increased FLT activity which can not be distinguished from activity in (high malignant) neoplasm's. MR spectroscopy corresponds to an inflammation process. A high grade glioma may be inferred from the high lactate and low NAA, but perfusion and Cho levels are not strongly increased (increased respect to gray matter, however comparable to normal values of white matter). The Increase of aspartate found in this granulomatous infection has not been described earlier. Follow up imaging and evaluation of more patients are needed for a more detailed evaluation of this phenomenon.

References:*[1] Carod-Artal, T Roy Soc Trop Med H 2008, 102;p107 [2] Scheenen, MRM 2008, 59;p1 [3]Saga, Clin Nucl Med 2006, 31;p774 [4] Troost, J Nucl Med 2007, 48: p726 [5] Mader, Eur J Radiol 2008, 67; p250 [6] Dringer, Prog Neurobiol 2000, 62; p649 [7] Liu, AJR 2008, 191; p582.*