DIFFERENTIATING GLIAL TUMOURS FROM DEMYELINATING DISEASE WITH SV MRS AT 1.5T. A MULTICENTRE STUDY.

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Introduction: Idiopathic inflammatory demyelinating lesions may rarely present as single or multiple focal lesions that may be clinically and radiographically indistinguishable from a brain tumor. This situation represents a diagnostic challenge, which reasonably calls for a biopsy despite the clinical suspicion of demyelination.

Purpose: To differentiate pseudotumoral inflammatory-demyelinating lesions (PIDL) from low-grade (LGG) and high-grade glial (HGG) tumors with long TE SV MR spectroscopy at 1.5T.

Methods: ***Patient selection***: patients were included in the study if they fulfilled the following criteria: (i) presence of a gadolinium-enhanced pseudotumoral lesion on brain MRI, (ii) MRS performed within 2 weeks of the onset of symptoms and (iii) a diagnosis of PIDL was established based on biopsy or clinico-radiological follow-up.

Data acquisition: Long TE (136 ms) PRESS SV MRS data acquired from patients bearing a PIDL were obtained at 1.5T with three different scanners: Siemens Magnetom Vision plus, Siemens Magnetom Symphony (Siemens Medical Solutions, Erlangen, Germany) and Philips Gyroscan (Philips Medical Sytems, Best, The Netherlands) at two different medical centres. LGG and HGG tumor spectra were acquired with the following 1.5 T scanners: GE Signa, Signa Advantage, LX CV/i 1.5 T, Philips NT and ACS NT 1.5 T, Siemens Vision 1.5 under acquisition conditions described in [**1**] and downloaded from a web-accessible MRS database [**2**]. ***Data processing***: LGG (n=34) and HGG (n=88) tumor spectra were processed with a series of modules (data manipulation software, DMS) that automatically process MRS data from Philips and GE [**1**]. PIDL (n=13) spectra were first processed with jMRUI 2.0 [**3**] and then converted into DMS format with a dedicated conversion software module (jDMS). ***Data post processing***: 196 spectral variables in the DMS format spectra (normalized to unit length, resolution 0.02ppm/point) were analyzed in the region 0.0-3.74 ppm. A first variable selection process was performed using Pearson correlation coefficient as described in [**1**], then feature reduction was performed taking the 10 variables showing the highest bivariate correlation values between groups as input for principal components (PC) calculation. The first 6 PC were then selected as they explained 80% of the total variance and were used as input features for a Fisher's discriminant classifier. Validation was performed with the leave-one-out method (LOO). Data post processing was performed with SPSS 14.0. Classification Sensitivity (SE) and Specificity (SP) were calculated as in [**4**].

Results: *Table 1* and *Figure 1* show classification results. Total accuracies were: 84.4% and 82.2% for the LOO test. SE and SP and 95% confidence interval (CI) values obtained were: For PIDL, SE=81% (CI 57-94%), SP=97% (CI 93-98%); for HGG, SE=93%(CI 87-96%), SP=79% (CI 68-87%); and for LGG, SE=68% (CI 55-79%), SP=93% (CI 88-97). The 5 wrongly classified PIDL spectra had in common the presence of lactate and in two the choline/creatine ratio was higher than 1, similarly to prototypical LGG spectra. The source of misclassification errors in the HGG spectra was absence of a necrotic pattern (astrocytomas/oligodendrogliomas of grade III were included in the HGG as well as glioblastomas).

Conclusion: It is possible to differentiate between spectra of PIDL, LGG and HGG with SV MRS at 1.5T. Misclassification in the PIDL group seems to be related to the temporal heterogeneity of the spectra of this pathology [**5**]. Future studies with more PIDL data and incorporating stratification by time after onset should improve the classifiers that aim to differentiate among these diseases.

			PREDICTED GROUP			Total cases
			LGG	HGG	PIDL	
ORIGINAL GROUP (CLASSIFICATION EXPERIMENT)	Number of cases	LGG	28	6	0	34
		HGG	9	77	2	88
		PIDL	4	0	9	13
	%	LGG	82.4	17.6	0	100.0
		HGG	10.2	87.5	2.3	100.0
		PIDL	30.8	0	69.2	100.0
ORIGINAL GROUP (LEAVE-ONE-OUT CROSS VALIDATION)	Number of cases	LGG	26	8	0	34
		HGG	9	77	2	88
		PIDL	5	0	8	13
	%	LGG	76.5	23.5	0	100.0
		HGG	10.2	87.5	2.3	100.0
		PIDL	38.5	0	61.5	100.0

Table 1: Classification results

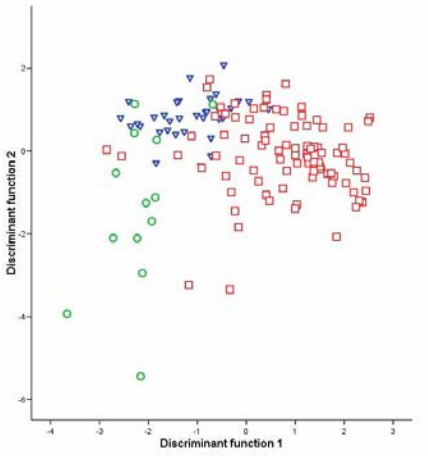

Figure 1: Classification plot. PIDL: green circles, HGG: red squares, LGG: blue triangles

References:

[**1**] Tate AR. et al. NMR Biomed. 2006;19: 411-34
[**2**] Julia-Sape M. et al. MAGMA. 2006; 19: 22-33
[**3**] http://www.mrui.uab.es/mrui/
[**4**] Julia-Sape M. et al. J Neurosurg. 2006;105: 6-14
[**5**] Cucurella MG. et al. NMR Biomed. 2002; 15: 284-92