FA and tract changes in obsessive compulsive disorder

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Introduction

Obsessive-compulsive disorder (OCD) is an anxiety disorder that is characterized by recurrent, unwanted thoughts and/or repetitive behaviors. It has been hypothesized that some forms of OCD may be the result of abnormalities in cortico-striato-thalamocortical circuitry [1, 2]. In particular, evidence of abnormal conflict monitoring and response inhibition deficits may implicate dysfunction of the anterior cingulate in OCD [3]. The purpose of this study was to use DTI derived metrics to explore disease affected circuits in OCD patients compared to healthy volunteers.

Subjects and Methods

We studied: (1) nine pediatric OCD patients; and (2) ten age-matched controls. Diffusion tensor imaging (DTI) was performed using a GE 3T clinical scanner. A 3D volumetric image set, diffusion tensor images including 5 b0 and 31 diffusion weighted images were acquired. The b-value was 1000 s/mm^2. The DTI protocol included 51 slices of 2.5 mm thickness, FOV 240 mm, image matrix of 128x128 zero filled to 256x256 and 10 sec TR. After DTI data acquisition, fractional anisotropy (FA) maps were reconstructed from DTI images and registered to a standard MNI template using FSL software [4]. The two groups were compared voxel-wise over the entire brain volume using SPM software [5]. Group differences were considered significant at a voxel-level threshold of $p<0.001$ with a correction for multiple comparisons at $p<0.05$ and a cluster cutoff of 100 voxels. FA values for each significant cluster were compared across groups using Student's *t*-tests and were considered significant for $p<0.05$. We also performed group tractography [6] using these FA difference regions as seed volumes.

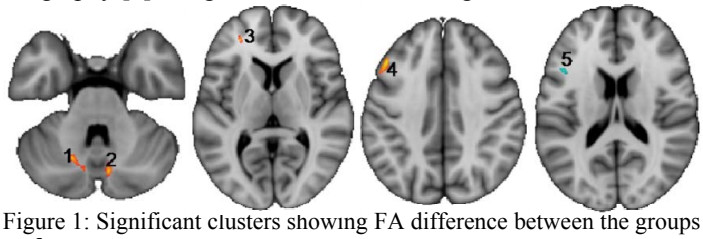

Figure 1: Significant clusters showing FA difference between the groups

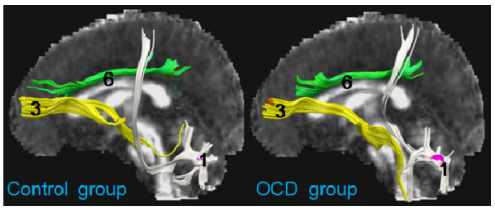

Figure 2: Tractography in control and OCD groups

Results

There were 5 clusters with significant differences in FA between patients and controls (Fig. 1). There were significant FA decreases in four clusters including the cerebellum (1, 2), BA9 (3), and BA10 (4) and a FA increase in one cluster (BA44; 5) in OCD patients. Fiber tracts passing through clusters 1, 3 and 6 in the two groups are presented in Fig. 2. The coordinates, cluster size, FA, post-hoc *p* value, tract count and tract volume of each significant cluster are provided in the table below. We also examined fiber tracts in the cingulum bundle (6) to test the hypothesis that this bundle was affected in OCD.

	Region	MNI coordinates			Cluster size	FA			Fiber tract count		Fiber tract volume (ml)	
		x	y	z	(voxels)	Control	OCD	p <	Control	OCD	Control	OCD
1	Cerebellum (left)	-14	-65	-30	305	0.375 ± 0.025	0.304 ± 0.035	0.0002	1180	942	3.075	2.634
2	Cerebellum (right)	9	-73	-34	118	0.351 ± 0.047	0.266 ± 0.024	0.0002	712	276	1.055	0.521
3	BA10 (gfm)	-35	54	12	197	0.379 ± 0.021	0.295 ± 0.030	0.0001	1775	1613	4.489	4.413
4	BA9 (gfm)	-45	21	40	638	0.216 ± 0.017	0.158 ± 0.013	0.0001	243	0	0.763	0
5	BA44 (gfi)	-45	17	16	112	0.226 ± 0.046	0.343 ± 0.061	0.0003	25	947	0.204	2.614
6	Cingulum (L+R)				2877	0.286 ± 0.055	0.280 ± 0.032	ns	1972	2419	3.979	5.209

Discussion/Conclusions

Our results demonstrate significant FA changes in the prefrontal cortex and cerebellum of OCD subjects compared to healthy volunteers. Among patients there were significant FA and fiber tracts decreases in the cerebellum, prefrontal cortex BA9, and BA10, which may relate to abnormalities in cortico-striato-thalamo-cortical circuitry (CSTC) in OCD. In addition, we found increased tract counts and volumes of the cingulum in OCD patients compared to healthy volunteers, which may relate to conflict monitoring deficts observed in this disorder.

Acknowledgments

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References

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