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Research report

White matter microstructural characteristics in Bipolar I and Bipolar II Disorder: A diffusion tensor imaging study

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ABSTRACT

Background: Diffusion tensor imaging (DTI) studies of bipolar disorder (BD) report contrasting results and are mainly focused on bipolar I (BD-I) samples. We aimed at investigating how and where DTI parameters differ between BD-I and bipolar II (BD-II) and between BD and healthy control subjects (HC).
Methods: We conducted a tract-based spatial statistics analysis of DTI derived parameters, namely fractional anisotropy (FA), axial diffusivity (AD) and radial diffusivity (RD) in a matched sample of 50 BD (25 BD-I and 25 BD-II) during the chronic course of the illness and 50 HC.
Results: Compared to BD-I and HC, BD-II showed lower FA but no significant AD or RD differences in the right inferior longitudinal fasciculus (ILF). Both patient groups showed lower AD and RD in the left internal capsule and lower AD across the left ILF, the cortico-spinal tract within the right hemisphere and bilaterally in the cerebellum with respect to HC.
Limitations: Patients were medicated at the time of scanning; the BD-II group had higher Hamilton Rating Scale for Depression scores than the BD-I group.
Conclusions: BD-II patients differ from BD-I in the ILF. Both BD subtypes showed widespread white matter (WM) changes in the internal capsule, cortico-spinal tract and cerebellum. The loss of WM integrity in BD-II might be due to demyelination whereas WM changes common to both subgroups could be attributable to axonal damage.

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1. Introduction

Structural brain imaging studies in individuals with bipolar disorder (BD) have described white matter (WM) changes in early (Vita et al., 2009) and late phases (Vederine et al., 2011) of the illness. However, there is no definitive evidence that patients with BD type I (BD-I) and II (BD-II) are differently characterized by specific structural abnormalities (Mahon et al., 2009).

Diffusion Tensor Imaging (DTI), a magnetic resonance technique sensitive to the movement of water molecules, has been used to investigate WM microstructure in BD; however, DTI studies have reported conflicting results. Indeed, fractional anisotropy (FA), the most replicated WM integrity index reflecting axonal coherence, was found reduced (Bauer et al., 2015; Haller et al., 2011; Sussmann et al., 2009; Vederine et al., 2011), increased (Wessa et al., 2009) or unchanged (Beyer et al., 2005) in patients with BD compared to healthy controls (HC). Fewer studies have investigated other DTI parameters, such as axial diffusivity (AD) and radial diffusivity (RD). These parameters give an in vivo measure of water diffusion parallel and perpendicular to WM fibers and reflect axonal and myelin integrity respectively (Beaulieu, 2002). RD values have been found increased at the onset of BD (Lu et al., 2012) and when comparing BD patients to those with unipolar depression (Benedetti et al., 2011) or unchanged in elderly BD (Haller et al., 2011). However, most of the above mentioned studies focused on patients suffering from BD-I; only a few included mixed groups of BD-I and BD-II (Bruno et al., 2008; Mahon et al., 2009). Thus, these findings may reflect the specific neurobiological substrate of BD-I.

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From a clinical point of view, BD-II is characterized by more frequent episodes of depression (Vieta et al., 1997), shorter euthymic periods (Judd et al., 2003) and cognitive deficits comparable with those found in BD-I (Bora et al., 2011). Indeed, episodes of depression and depressive symptoms have been found to lead to psychosocial disability as well as manic or hypomanic events (Judd et al., 2005). Whether or not these phenotypic characteristics of BD-II have different brain structural correlates than those found in BD-I has still to be clarified. The under-recruitment of BD-II patients in neuroimaging studies may be due to greater misdiagnosis of BD-II compared to BD-I (Fiedorowicz et al., 2011). Recently, functional and structural studies comparing the two subgroups suggested that BD-II patients have increased activity in the ventral striatum as well as higher volume in the left putamen (Caseras et al., 2013), while BD-I show reduced cortical volume in the orbitofrontal region and reduced thickness of the temporal cortex (Maller et al., 2014). Moreover, DTI studies on BD-II patients found widespread FA reductions in all major WM tracts, including cortico-cortical associative and interhemispheric fibers (Ambrosi et al., 2013; Yip et al., 2013).

To date, few studies have compared BD-I, BD-II and HC for WM abnormalities. Compared to HC, both BD subgroups were shown to have FA reductions in the WM of the corpus callosum, cingulum and right frontal regions (Ha et al., 2011; Liu et al., 2010). A direct comparison between BD-I and BD-II indicated that BD-I have lower FA values in the right temporal WM (Ha et al., 2011) while BD-II have lower FA values in the right precuneus, frontal and prefrontal regions (Liu et al., 2010) and widespread prefrontal and temporal RD increase (Maller et al., 2014).

Hence, we performed a cross-sectional study on DTI parameters of BD-I, BD-II and HC. We aimed at investigating FA, AD and RD differences among the three groups and at identifying potential axonal or myelination damage. We expected to find WM impairments in both BD-I and BD-II compared to HC. Due to the lack of data on BD-II, we could not make an a priori hypothesis about specific brain regions in which the two subgroups may have microstructural differences. We performed a whole brain analysis to address this issue.

2. Methods

2.1. Subjects

Sixty patients with BD-I ($n=32$) and BD-II ($n=28$) were initially recruited at the IRCCS Santa Lucia Foundation of Rome. The diagnosis of BD was made according to the Diagnostic and Statistical Manual of Mental Disorders IV-Edition, text revised (DSM-IV-TR) (American Psychiatric Association, 2000). The clinician who had been treating the patients and knew their clinical history, but who was blind to the aims of the study, made the preliminary diagnosis. Then a senior research psychiatrist confirmed all preliminary diagnoses using the Structured Clinical Interview for DSM-IV-TR-Patient Edition (SCID-I) (First et al., 2002a). From this original group, 7 patients with BD-I and 3 patients with BD-II were excluded for the presence of moderate or severe brain vascular lesions (see exclusion criteria). The final sample for this study consisted of 50 patients (BD-I=25; BD-II= 25). Inclusion criteria were: (a) age >16 years and <75 years; (b) no additional axis I diagnosis; (c) at least five years of education. Exclusion criteria were: (a) traumatic brain injury with loss of consciousness; (b) lifetime history of major medical (e.g. chronic kidney failure, chronic heart failure, decompensated diabetes, etc.) or neurological disorders; (c) history of substance abuse or dependence; (d) dementia or cognitive deterioration according to DSM-IV-TR criteria and a Mini-Mental State Examination (MMSE) (Folstein et al., 1975) score lower than 25, according to the normative data of the Italian population (Measso et al., 1993); (e) contraindication for MRI; (f) any potential brain abnormality or microvascular lesion as apparent on conventional FLAIR-scans through white matter hyperintensities (WMH); in particular, the presence, severity and location of WMH were computed using the semi-automated method recently published by our group (Iorio et al., 2013). Patients with BD-II were recruited only if they had a stable diagnosis for at least six years, to avoid as much as possible diagnostic type changes. Mood symptoms were rated using the Young Mania Rating Scale (YMRS) (Young et al., 1978) and the 17-item Hamilton Rating Scale for Depression (HAM-D) (Hamilton, 1960). All but two patients were receiving drug treatment during the evaluation.

We also recruited 50 HC subjects in the same geographical area,

Table 1
Sociodemographic and clinical characteristics of BD-I and BD-II patients and HC subjects.

Characteristics	BD-I ($n=25$)	BD-II ($n=25$)	HC ($n=50$)	t, F or χ^2	df	p
Age (years), mean (SD)	48.6 (11.4)	48.4 (12.7)	48.3 (12.0)	0.006	2	0.99
Males, n(%)	13 (52)	13 (52)	26 (52)	0.00	2	>0.999
Educational level (years), mean (SD)	14.2 (4.2)	14.3 (3.6)	14.2 (3.2)	0.986	2	0.98
Duration of illness (years), mean (SD)	20.3 (10.7)	23.9 (11.7)	–	−1.13	45	0.26
Number of past manic/hypomanic episodes, mean (SD)	5.09 (5.4)	7.05 (7.41)	–	−0.97	38	0.34
Number of past depressive, mean (SD) episodes	7.5 (9.8)	9.7 (17.9)	–	−0.50	39	0.61
HAM-D score, mean (SD)	9.2 (8.3)	14.5 (7.4)	–	−2.39	48	0.02
YMRS score, mean (SD)	3.2 (2.2)	3.5 (2.8)	–	−0.52	45	0.60
Current medication , n(%)	23 (92)	22 (88)	–			
Antidepressants, n(%)	10 (40)	6 (24)	–	2.1	2	0.34
Antipsychotics, n(%)	16 (64)	12 (48)	–	2.22	2	0.32
Antiepileptics, n(%)	11 (44)	14 (56)	–	0.87	2	0.64
Lithium, n(%)	16 (64)	10 (40)	–	4.01	2	0.13
Benzodiazepines, n(%)	6 (24)	13 (52)	–	5.24	2	0.07
Other treatments	–	–	–	–	–	–

BD-I=patients with type I bipolar disorder, BD-II=patients with type II bipolar disorder, HC=healthy controls; SD=standard deviation; df=degrees of freedom.
Note that for some patients we were unable to collect data on duration of illness ($n=3$), number of past manic/hypomanic ($n=10$) and depressive episodes ($n=9$), and YMRS score ($n=3$).

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