Are obsessive–compulsive symptoms expression of vulnerability to bipolar disorder?

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More than half of patients with bipolar disorder (BD) have an additional diagnosis, one of the most difficult to manage being obsessive–compulsive disorder (OCD). French psychiatrist Bénédic Augustin Morel first described patients with BD-OCD arising questions around the nosological and clinical meaning of this condition. In a standard 1969 psychiatry textbook, Mayer-Gross and colleagues, mostly considering course of illness, included patients with BD-OCD in the manic-depressive disorders (1). Although recent studies have investigated the co-occurrence of anxiety and bipolar disorders, the topic is insufficiently studied and the relationship between BD and OCD remains unclear. However, given the available scientific evidence, some observations can be made.

i) Apparent BD-OCD comorbidity is a common condition in psychiatry. In our recent meta-analysis, the pooled prevalence of OCD in BD was 17.0% (95% CI 12.7–22.4%), which was comparable to the results reported by the pooled prevalence of BD in OCD (18.35%, 95% CI 13.2–24.8%) (2). Although limited by retrospective study design, small sample size, different thresholds for BD diagnosis (i.e. categorical vs. dimensional approach) and a different accuracy in diagnosing OCD (i.e. discrimination between true ego-dystonic obsessions and depressive ruminations), these results confirm the relevance of comorbid BD-OCD.

ii) As reported by recent studies, OC symptoms in childhood and adolescence increase the risk of a later BD diagnosis (3). These results would be suggestive of partially shared aetiopathogenetic mechanisms between these severe mental disorders.

iii) In our previous systematic review, considering course of illness as a key diagnostic validator, especially among patients with a primary diagnosis of BD, the majority of comorbid OCD cases appeared to be related to mood episodes (1). OC symptoms in comorbid patients appeared more often – and sometimes exclusively – during depressive episodes, and comorbid BD and OCD cycled together, with OC symptoms often remitting during manic/hypomanic episodes.

iv) Results from our meta-analysis showed higher comorbidity rates in youths (24.2%, 95% CI = 10.36–41.60, n = 345, z = −9.5) compared to adults (13.56%, 95% CI = 10.4–16.25, n = 4,539) (2). In other words, OC symptoms would initially coexist with BD symptoms and they would gradually tend to decrease in the adulthood.

v) From a neurobiological perspective, BD mostly showed hypoactivity in orbitofrontal cortex (OFC) (i.e. decision making, impulse control) and in dorsolateral prefrontal cortex (DLPFC) (i.e. planning, attentional set shifting) with grey matter volume reduction associated to manic episodes, while OCD mainly presented hyperactivity of OFC with deficit in emotional processing (4). The overlap of similar cortical–subcortical circuits may partially explain clinical features of comorbid patients with BD-OCD during the course of illness.

vi) The clinical features of comorbid patients with BD-OCD would explain why OCD and BD symptoms respond to adequate mood stabilizer treatment (5). Only in a minority of comorbid patients with persistent OCD, despite improvement in mood episodes, addition of low doses of antidepressants could be considered while strictly monitoring emerging symptoms of mania or mixed states.

To conclude, according to the available literature, we speculate that OC symptoms in childhood and adolescence may be expression of vulnerability to BD increasing the risk of a later BD diagnosis. OC symptoms would initially coexist with BD symptoms even cycling together, and they would gradually tend to decrease in the adulthood.

Considering the important nosological, clinical and therapeutic implications, further original studies are needed to clarify BD-OCD comorbidity. In particular, studies addressing neurobiological substrates are essential to illuminate pathogenetic mechanisms that underlie comorbid BD-OCD.

Declaration of interests

Dr. Tonna, Dr. Amerio, Dr. Odone, Dr. Ossola and Dr. Marchesi report no conflict of interests. Dr. Ghaemi has provided research consulting to Sunovion and Pfizer and has obtained a research grant from Takeda Pharmaceuticals. Neither he nor his family hold equity positions in pharmaceutical corporations.

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Letters to the editor

References

1. Amerio A, Odone A, Liapis CC, Ghaemi SN. Diagnostic validity of comorbid bipolar disorder and obsessive-compulsive disorder: a systematic review. Acta Psychiatr Scand 2014;129:343–358.

2. Amerio A, Stubbs B, Odone A, Tonna M, Marchesi C, Ghaemi SN. The prevalence and predictors of comorbid bipolar disorder and obsessive-compulsive disorder: a systematic review and meta-analysis. J Affect Disord 2015;186:99–109.

3. Cederlöf M, Lichtenstein P, Larsson H et al. Obsessive-compulsive disorder, psychosis, and bipolarity: a longitudinal cohort and multigenerational family study. Schizophr Bull 2014;41:1076–1083.

4. Ekman CJ, Lind J, Rydén E, Ingvar M, Landén M. Manic episodes are associated with grey matter volume reduction—a voxel-based morphometry brain analysis. Acta Psychiatr Scand 2010;122:507–515.

5. Amerio A, Odone A, Marchesi C, Ghaemi SN. Treatment of comorbid bipolar disorder and obsessive-compulsive disorder: a systematic review. J Affect Disord 2014;166:258–263.