Dear Editor:

EDNOS (Eating disorder not otherwise specified) accounts for three quarters of all community cases with eating disorders (Bulik et al., 2007). Among EDNOS, the most common (Hudson et al., 2007) is Binge Eating Disorder (BED). BED is a good diagnostic construct and a stable condition, associated with elevated psychiatric comorbidity and impairment in psychosocial functioning (Pope et al., 2006).

The few available epidemiological studies in Italy suggest a lifetime prevalence of 0.64% for EDNOS (Favarelli et al., 2006), whereas in other samples lifetime estimated prevalences of sub-threshold BED and any binge eating are 0.72%, and 2.15% respectively (Preti et al., 2009). The comorbidity of BED and other psychiatric diagnoses is high: overall, 73.8% of patients with BED have one additional lifetime psychiatric disorder and 43.1% have at least one current psychiatric disorder (Javaras et al., 2008). Though often associated with obesity, BED should be considered a separate condition (Hudson et al., 2007).

This disorder often goes undetected and untreated both in primary care and general psychiatric services (Striegel-Moore et al., 2010). Mond et al. (2007) have shown that only a small percentage of subjects with BED (22.8%) receive a specific treatment for eating problems in primary care setting.

In MHS, time constraints usually prevent clinicians from administering clinical interviews intended for EDs diagnosis. The utility of questionnaires as a screening for BED seem clear (Freitas et al., 2006). Among available tools, one of the most used and validated is the Binge Eating Scale questionnaire (Gormally et al., 1982).

The main assumptions of this study were

1) the EDNOS diagnosis is often neglected by primary care physicians referring their patients to a MHS;
2) a significant percentage of subjects who seek treatment for anxiety and depression in MHS present a BED or a sub-threshold BED symptomatology (s-BED or EDNOS binge type);
3) subjects treated at MHSs for anxiety or depressive disorders with BED (or s-BED) present higher levels of clinical severity compared to subjects without BED (or s-BED) comorbidity;
4) the use of self-administered questionnaires could be helpful in detecting BED in subjects referred to MHSs for anxiety/depressive disorders.

MATERIALS AND METHODS

Subjects

All outpatients referred by a primary care physician to the MHS of Chivasso after April 1st, 2008, with an indication for anxious or depressive symptoms were triaged by a trained nurse and later referred to a psychiatrist or a psychologist according to the “as usual” practice of the MHS. During triage, some patients were excluded according to these criteria:

1) subjects aged 65 or more,
2) subjects with an acute full-syndrome Axis I disorder (DSM-IV-TR, American Psychiatric Association, 2000) requiring inpatient care,
3) presence of an acute addiction disorder,
4) mental retardation,
5) a clear mistake of primary care physicians,
6) refusal to give informed consent to participate in the study.

As a whole, 71 patients were excluded for the following reasons:

1) age out of the range (N=46);
2) acute Axis I disorder (N=7) requiring inpatient treatment;
3) acute substance abuse disorder (N=4);
4) Mental Retardation (N=2);
5) psychotic disorder (N=2), bipolar disorder (N=4) or Anorexia Nervosa (N=3) and (6) refuse to participate in the study or to sign informed consent (N=4).
The subjects included were asked to complete Barratt Impulsiveness Scale (BIS), Binge Eating Scale (BES) and Temperament and Character Inventory (TCI). This screening stage was called “DGN0 phase”. Subsequently, subjects were assigned to a Psychiatrist or to the MHS Psychologist, who were blind as to the results of the questionnaires. During the first session the therapists rated the level of severity through the CGI-Severity Index (CGI-SI) and registered their diagnosis in Axis I and II of DSM-IV-TR (American Psychiatric Association, 2000) according to the “as usual” practice (“DGN1 phase”).

After that, the researchers (DGN2 phase) identified respondents who had scored BES 17 or more; these subjects were submitted to a face-to-face diagnostic interview in accordance with the Eating Disorder Examination (Fairburn & Cooper, 1993) and SCID-I (First et al., 1995), in order to assess possible EDs (DSM-IV-TR, American Psychiatric Association, 2000).

The researchers determined that the recruiting phase could be ended after identification of 100 subjects meeting the inclusion criteria. Such methodological choice was aimed at maintaining a naturalistic setting.

Assessment instruments

Clinical Global Impression (CGI). This is a well-known assessment tool, administered by clinicians in order to evaluate the severity of an illness (item 1; CGI-SI).

Binge Eating Scale (BES). The continuous scale (0-46) BES questionnaire (Gormally et al., 1982) is a self-administered tool with an overall good test-retest reliability (r 0.87, p<.001). Scores of 27 or more have served as a cut-off value for identifying severe binge eating disorders, while scores of 17 have been chosen as a cut-off value for mild or no binge eating. Therefore patients scoring 17 and less were considered non-bingers (no-BED); those scoring between 18 and 26, moderate or sub-threshold bingers (s-BED, including EDNOS binge type), while patients scoring 27 or above (p-BED) were considered severe binge eaters (Greeno et al., 1995).

Barratt Impulsiveness Scale (BIS-11). The BIS conceptualizes impulsivity as containing three main components (Patton et al., 1995): nonplanning impulsivity (NPI), motor impulsivity (MI) and attentive impulsivity (AI).

Temperament and Character Inventory (TCI). The TCI (Cloninger et al., 1994) assess temperaments under four dimensions (Novelty Seeking [NS], Harm Avoidance [HA], Reward Dependence [RD], Persistence [P]), whereas the remaining three dimensions (Self-Directedness [SD], Cooperativeness [C], Self-Transcendence [ST]) are intended to evaluate character. Low SD and C scores appear to be the most important predictors of a DSM-IV Axis II disorder (Cloninger et al., 1994).

Data analysis

All data analyses were performed using the SPSS. Chi-square was calculated to evaluate the association among categorical variables (Axis I and Axis II comorbidity), as well as DGN0 (screening), DGN1 (diagnosis of anxious or depressive disorders) and DGN2 (diagnosis of EDs through interview) diagnostic groups. An evaluation through GLM and ANOVA has been made (Table II) to compare the three groups identified by the BES questionnaire (DGN0): no-BED (BES score <17), s-BED (BES between 17 and 26) and p-BED (BES of 27 or more).

Finally, a T-test for independent samples was made, in order to compare BED+ and BED- groups in continuous variables; a logistic regression (stepwise forward) was calculated to detect the independent predictors of the BED+ and BED- groups. Variables included were both continuous (age, CGI, BMI, NS, HA) and categorical (gender, presence vs. absence of a personality disorder, DGN0 classification, BIS CUTOFF). The variable BIS CUTOFF has a value of “0” with BIS scores of 59 or less, and “1” with BIS scores above 60. This cut-off was calculated in accordance with relevant literature and data distribution in our sample.

RESULTS

Table I shows the personal and clinical features of patients included in the sample.

DGN0 phase

Table II compares a number of data concerning p-BED, s-BED and no-BED subgroups through GLM and ANOVA, controlled for Gender (0=female; 1=male), presence of Personality Disorder (no=0, yes=1), and age.

As regards Axis I comorbidity (anxious or depressive disorders or both) at DGN1, no significant differences emerged (Chi Square=2.342, p<.673, df=4) among these three DGN0 groups (data will be available upon request for interested readers).

Also in the case of Axis II comorbidity at DGN1, no significant differences were identified among the three groups in Personality Disorder (19/64 in the no-BED...
group, 6/18 in the s-BED group, and 8/18 in the p-BED group; Chi Square=1.385, p<.510, df=2), but the presence of Cluster B personality disorders resulted higher in the p-BED group (7/18 in p-BED, 3/18 in s-BED, 4/64 in no-BED; Chi Square=12.560, p<.002, df=2).

DGN2 phase

T-test analysis showed differences between BED+ and BED- groups in CGI-SI, Novelty Seeking, BIS Total Score, BIS Attentive and motor subscales (data will be available upon request for interested readers). Table 3 shows the independent predictors of BED diagnosis (DGN2) as detected through logistic regression.

DISCUSSION

A large number of patients referred to MHSs with indications of an anxious or depressive disorders present a full-criteria BED or a sub-threshold binge symptomatology, but this diagnosis is widely neglected.

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Only in a limited number of cases, equivalent to nearly 20% of subjects who were subsequently diagnosed through a specific interview, the therapist had already formulated a DGN1 diagnosis of EDs. This could be due to several factors, such as:

1) the absence in our Mental Health Department of an Eating disorder service increasing awareness of such disorders among clinicians,
2) scarce sensitivity to EDs among primary care physicians (who were responsible for only 3% of EDs diagnoses),
3) the fact that BED diagnoses are included in DSM-IV-TR as “criteria for further study” and have been granted attention as a stable syndrome only in the last five years (Pope et al., 2006).

Table II – Comparison among the three DGN0 groups (p-BED, s-BED, no-BED).

<table>
<thead>
<tr>
<th>Group</th>
<th>p-BED (18)</th>
<th>s-BED (18)</th>
<th>no-BED (64)</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>37.8±9.3</td>
<td>35.6±9.9</td>
<td>37.1±10.7</td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
</tr>
<tr>
<td>Schooling</td>
<td>10.6±2.9</td>
<td>11.0±2.7</td>
<td>10.9±2.8</td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
</tr>
<tr>
<td>BMI</td>
<td>24.7±2.8</td>
<td>25.6±3.4</td>
<td>24.2±2.8</td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
</tr>
<tr>
<td>CGI</td>
<td>4.9±0.7</td>
<td>4.4±0.6</td>
<td>4.0±0.7</td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

Table III – Predictors of BED diagnosis (BED+/BED-) identified through clinical interview (DGN2).

<table>
<thead>
<tr>
<th>Models</th>
<th>Variable predictive</th>
<th>B</th>
<th>WALD</th>
<th>p</th>
<th>OR</th>
<th>IC (95%)</th>
<th>% correct classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>no-BED</td>
<td>6.733</td>
<td>.035</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>s-BED</td>
<td>-23.28</td>
<td>.000</td>
<td>.996</td>
<td>.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>p-BED</td>
<td>-2.30</td>
<td>6.733</td>
<td>.009</td>
<td>.100</td>
<td>.180-569</td>
<td>90%</td>
</tr>
<tr>
<td></td>
<td>Constant</td>
<td>2.08</td>
<td>7.687</td>
<td>.006</td>
<td>8.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>°BIS CUTOFF</td>
<td>-2.69</td>
<td>6.28</td>
<td>.012</td>
<td>.068</td>
<td>.008-556</td>
<td></td>
</tr>
<tr>
<td></td>
<td>no-BED</td>
<td>7.105</td>
<td>.029</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>s-BED</td>
<td>-23.35</td>
<td>.000</td>
<td>.996</td>
<td>.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>p-BED</td>
<td>-2.84</td>
<td>7.105</td>
<td>.008</td>
<td>.058</td>
<td>.007-471</td>
<td>94%</td>
</tr>
<tr>
<td></td>
<td>Constant</td>
<td>4.046</td>
<td>10.471</td>
<td>.001</td>
<td>57.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>°BIS CUTOFF</td>
<td>-2.97</td>
<td>4.90</td>
<td>.027</td>
<td>.051</td>
<td>.004-711</td>
<td></td>
</tr>
<tr>
<td></td>
<td>°NS</td>
<td>.326</td>
<td>3.89</td>
<td>.048</td>
<td>1.38</td>
<td>1.002-1.913</td>
<td></td>
</tr>
<tr>
<td></td>
<td>no-BED</td>
<td>6.862</td>
<td>.032</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>s-BED</td>
<td>-25.05</td>
<td>.000</td>
<td>.995</td>
<td>.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>p-BED</td>
<td>-4.13</td>
<td>6.862</td>
<td>.009</td>
<td>.016</td>
<td>.001-313</td>
<td>95%</td>
</tr>
<tr>
<td></td>
<td>Constant</td>
<td>-2.15</td>
<td>.494</td>
<td>.482</td>
<td>.116</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. °BIS CUTOFF: BIS values of 60 or more = 1, values below 60 = 0. ° = inserted as continuous variable. ° Other categorical (personality disorders and gender) and continuous (CGI severity index /CGI-SI, AGE, Harm Avoidance, Body Mass Index) variables were inserted in the analysis, but were not significant in the final model.
The rate of BED diagnoses (12%, +10% of s-BED) in our sample seems quite high (Favarelli et al., 2006), despite unavailability of comparison data collected in similar settings. This high prevalence rate confirms the significant frequency of BED comorbidity with anxious/depressive disorders (Gruzca et al., 2007).

The third aim of this study was to understand whether patients belonging to this specific subgroup could be considered significantly different from patients without BED comorbidity. A t-test analysis showed that subjects belonging to the BED+ group present a higher level of overall clinical severity (CGI-SI), and also higher levels of Novelty Seeking (TCI) and impulsivity (BIS) if compared to subjects with anxious or depressive disorders with no BED. The higher level of CGI seems to indicate that although therapists at MHSs often neglect EDs diagnoses, there is a clear perception of clinical severity. Also, higher levels of CGI could possibly be related to a higher prevalence of cluster B Personality Disorders (DSM-IV-TR) in the BED+ group.

Impulsivity is a complex dimension that influences the pathogenesis and the course of several mental disorders. Researchers have demonstrated that impulsivity is a trans-nosographic dimension which presents features of the endophenotypes, that it is of extreme relevance for assessing the severity of mental disorders, and that stronger impulsivity may decrease response and adherence to treatments. A high level of impulsivity in subjects with BED (or s-BED) should not come unexpected, because a pathological increase of impulsivity is at the core of most eating disorders of the bulimic spectrum (BED, s-BED or BN). The drawing from this study is consistent with those of previous ones (Freitas et al., 2006). Particularly, the BES could be considered an easier screening instrument in order to ascertain the possible presence of an eating disorder within the bulimic spectrum (BED, s-BED or BN). The drawbacks of self-administered questionnaires in the assessment of eating disorders are obvious; on the other hand, self-administered instruments tend to increase the likelihood that respondents will faithfully disclose socially undesirable behaviours (Gruzca et al., 2007). This could be particularly convenient in a MHS whenever the therapist is not confident with the assessment of binging behaviours and with the consequent psychosocial distress.

Submitting three different questionnaires to the same patient could be rather problematic in a MHS setting; nevertheless, the results of this study show that the use of BES, combined with BIS and TCI could lead to early identification of subjects with anxious or depressive disorders and with a high likelihood for BED comorbidity. Subjects with higher NS scores, with high BIS (more than 60) and BES (more than 27) have a high probability for BED comorbidity.

Essentially, the BED+ group was characterized by a higher level of impulsivity, a temperament prone to impulsivity and disinhibition, a higher prevalence of Cluster B Personality Disorders and a greater clinical severity. These data seem to ascribe further relevance to the detection of EDNOS diagnoses (BED and s-BED) in community MHSs.

Our results confirm the need for the recognition of comorbid EDNOS among anxious or depressed patients in community settings: BED could affect in different ways both clinical characteristics and course of the illness. A delayed detection of eating-related and non-eating-related impulsivity could produce incomplete response to treatment programs or early dropout in the group of subjects with BED (or s-BED) comorbidity.

As concerns the validity of self-administered instruments in the screening of BED (or s-BED) in MHSs, findings from this study are consistent with those of previous ones (Freitas et al., 2006). Particularly, the BES could be considered an easier screening instrument in order to ascertain the possible presence of an eating disorder within the bulimic spectrum (BED, s-BED or BN). The drawbacks of self-administered questionnaires in the assessment of eating disorders are obvious; on the other hand, self-administered instruments tend to increase the likelihood that respondents will faithfully disclose socially undesirable behaviours (Gruzca et al., 2007). This could be particularly convenient in a MHS whenever the therapist is not confident with the assessment of binging behaviours and with the consequent psychosocial distress.

According to the TCI, subjects with BED are characterized by high Novelty Seeking and low Self Directedness and Cooperativeness (Fassino et al., 2002). Recent data also report high Harm Avoidance (HA) in BED subjects (Gruzca et al., 2007). In our study, only Novelty Seeking resulted to predict the BED+ diagnosis, whereas SD, C and HA did not. This is due to the fact that all subjects included in our sample were affected by an anxious or depressive disorder, implying altered levels of these three dimensions (Cloninger et al., 1994).
This study has two major limitations:

1) the inadequate number of subjects included;
2) the self-reporting quality of the Body Mass Index (BMI) assessment questionnaire, since subjects probably minimized their real weight and the BMI did not result a predictor of BED diagnosis.

In conclusion, our results seem to confirm that in MHSs the vast majority of individuals with anxiety and depression do not receive treatment for their comorbid eating disorders (BED and s-BED). The detection of BED comorbidity in MHS patients could promote:

1) the adoption of more appropriate psychopharmacological and psychological treatments (Leombruni et al., 2008), e.g. avoiding drugs which increase weight or appetite, and integrating the therapeutic program with a cognitive-behavioral psychotherapy aimed at reducing both anxious/depressive symptomatology and binge eating behaviours,
2) the planning of an integrated treatment program involving nutrition specialists and dieticians,
3) the diffusion of a “culture” of eating disorder treatment in MHSs.

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