Borderline personality features and development of psychosis in an ‘Ultra High Risk’ (UHR) population: a case control study

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Abstract

Aims: There is clinical uncertainty as to whether borderline personality disorder (BPD) traits in those with an ‘at risk mental state’ have an effect on the risk of ‘transition’ to psychosis. We aimed to investigate the relationship between baseline BPD features, risk of transition and type of psychotic disorder experienced.

Method: This is a case-control study of ‘Ultra High Risk’ (UHR) for psychosis patients treated at the clinic, between 2004 and 2007. ‘Cases’ were UHR individuals who made the ‘transition’ to full threshold psychotic disorder within 24 months; ‘Control’ group was a matched UHR sample who had not developed a psychotic disorder at 24 months. Individuals were matched on time of entry to the clinic, age and gender. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) BPD features were assessed from clinical assessments using a structured instrument (Structured Clinical Interview for DSM-IV Axis II Disorder for BPD (SCID-II BPD)). Psychosis diagnosis following transition was rated from the clinical files using the operational criteria in studies of psychotic illness (OPCRIT) computer algorithm. The number of BPD traits and number with full threshold BPD were compared in those who developed psychosis and those who did not.

Results: We analysed data from 48 cases and 48 controls. There was no statistically significant difference in the rate of transition to psychosis for those with baseline full-threshold BPD, compared with those without BPD. The number of BPD traits or number with full threshold BPD did not differ by psychosis diagnosis grouping.

Conclusions: Co-occurring BPD or BPD features does not appear to strongly influence the risk of short-term transition to psychosis or the risk of developing a non-affective psychotic disorder in this population.

Key words: borderline personality disorder, case control study, prodrome, psychotic disorder, risk factor.

INTRODUCTION

Over the past 15 years, there has been considerable research focus on individuals deemed to be at increased ‘clinical’ risk for developing psychotic illnesses such as schizophrenia. This has led to the development of clinical criteria, assessed by instruments such as the Comprehensive Assessment of At-Risk Mental States (CAARMS) and the Structured Interview for Prodromal Syndromes (SIPS) for identifying such ‘at risk’ or putatively ‘prodromal’ individuals using a so-called ‘close in’ strategy. This consists of a combination of attenuated psychotic phenomena, reduced psychosocial functioning and/or family history of psychotic illness. These individuals have been described as being at ultra-high risk (UHR) for psychosis to distinguish them from other high-risk groups. From a combination

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of predictive studies in UHR individuals, the proportion who develop a psychotic disorder without antipsychotic medication within 12 months was initially reported at about 37% (range 10–45%); however, recent studies have reported considerably lower rates over this time period.6–8

Stern9 originally used the term ‘borderline personality disorder’ (BPD) to describe patients who manifested both neurotic and psychotic symptoms. The status of psychotic symptoms in the contemporary view of BPD remains controversial.10 The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR)11 states that individuals with BPD can develop ‘psychotic-like symptoms’, such as hallucinations, body-image distortions, ideas of reference and hypnagogic phenomena during times of stress. These symptoms are similar to the sub-threshold or ‘attenuated’ psychotic phenomena that are a major component of the ‘at risk’ criteria. There is evidence to suggest that the ‘psychotic’ symptoms in BPD patients are more likely to be ‘broad’ in nature (e.g. paranoid experiences, magical thinking and depersonalization/derealization) and less likely to be the ‘narrowly defined’ psychotic symptoms (e.g. thought disorder or delusions of control) more associated with schizophrenia spectrum psychoses.12–14 However, studies have described both transient and persistent paranoid and hallucinatory symptoms in BPD.15–19

Several studies have supported a link between psychotic symptoms in BPD and affective disorder.12,13,20 Recent research has found that patients with BPD have greater exacerbation of psychotic symptoms in response to stress than patients with psychotic disorders.21 This same research group has suggested a separate ‘affective pathway to psychosis’ involving affective responses to stress increasing liability to psychosis.22 The relationship between BPD and later transition to psychotic disorder in the UHR population might inform such theories if outcomes are distinctly different in UHR individuals with BPD, where affective regulation is often disordered,23 than in UHR individuals without BPD.

Rosen and colleagues24 reported the degree of axis II co-morbidity in an ‘at risk’ cohort. They found that 17% of help-seeking individuals meeting their ‘prodromal’ criteria had a diagnosis of BPD. This rate was no different in comparison with individuals from the sample that did not meet ‘prodromal’ criteria. The study was limited by its small sample and that it did not investigate the relationship between BPD and transition to a frank psychotic illness. However, other groups report lower levels of full threshold BPD in their ‘Clinical High Risk’ sample (5%).25 It is not clear whether this is a difference in ascertainment, or a true difference in samples. We aimed to investigate the influence of co-morbid BPD traits on outcome in a UHR group, primarily not only to determine whether the presence of BPD traits alters the risk of subsequent transition to psychosis but also to determine BPD prevalence rates.

This study investigates two groups of UHR patients, namely those with and those without BPD features. It seeks to compare transition rates to a psychotic disorder between the two groups and to determine whether, for those who develop a psychotic disorder, the type of psychotic disorder differs between these two groups. We hypothesized that:

1 UHR individuals with BPD will have a lower rate of transition to psychotic disorder than those without BPD.
2 Compared with those without BPD, UHR individuals with BPD who develop a psychotic disorder will be more likely to be diagnosed as having a non-schizophrenia spectrum psychosis.

METHODS

Study design

The study was a retrospective ‘case-control’ comparison study, which was carried out at the Personal Assessment and Clinical Evaluation (PACE) clinic, Melbourne. Given the recent reduced rates of ‘transition’ to a psychotic disorder in our clinic,6 we used a case control design in order to investigate the relationship between BPD on transition to psychotic disorder. Case control designs are often used when investigating outcomes with a lower prevalence26

Participants

Participants were all patients initially assessed and treated by the PACE clinic between January 2003 and January 2007. There were 471 individuals assessed and treated within this time period. The PACE clinic is an individual clinic of a wider youth health service (Orygen Youth Health), a public mental health service for 15 to 24-year-olds in western Melbourne, Australia. Individuals are referred to Orygen Youth Health from a variety of sources in the catchment area, such as general practitioners, private psychiatrists, school counsellors, support workers, family members, as well as the
individuals themselves, and then assessed by a dedicated assessment team. The full details of the Orygen Youth Health service are described in detail elsewhere.27 Patients are accepted to the PACE clinic if they have had a drop in functioning or persistent low functioning for at least 1 month within the previous 12 months and meet criteria for at least one of three UHR groups: (i) presence of attenuated (sub-threshold frequency/intensity for a diagnosis of a psychotic disorder) psychotic symptoms within the previous 12 months; (ii) history of brief self-limited psychotic symptoms which spontaneously resolve (within 7 days) in the previous 12 months; and (iii) genetic vulnerability to psychotic disorder (trait), with either schizotypal personality disorder or family history of psychotic disorder in a first-degree relative. The full criteria can be found elsewhere.28,29

UHR status was assessed using the CAARMS.1 The CAARMS has been demonstrated to have excellent reliability.1 All patients accepted into PACE are allocated a clinical psychologist who provides case management and a comprehensive psychosocial intervention for 6 to 12 months on an outpatient basis. For a comprehensive description of the PACE treatment model, see Nelson and Yung.30

Sample

We examined the clinical files of two pre-identified groups of individuals: ‘cases’ were those treated at the clinic between January 2003 and January 2007 who subsequently transitioned to full-threshold psychotic disorder within a 24-month period. ‘Controls’ were a matched group of individuals from the clinic who were treated in the same time period but who had not transitioned to psychosis within 24 months of follow-up. Control individuals were matched on entry date into the PACE clinic (nearest entry date to the case individual) to attempt to control for differences in length of treatment offered during the time period, age at entry to the service and gender. We hypothesized that these variables might affect both the outcome and the possibility of having BPD features at baseline and it was possible to easily match individuals on these variables.

The sample of ‘transitioned’ cases was ascertained via clinical notes from our service and our own internal database. This database records all patients who have attended the PACE clinic since its inception and whether a psychotic diagnosis has been documented. We supplemented this information to identify missing ‘transition’ cases by searching a clinical database detailing all psychiatric contact in the state of Victoria, Australia. This database contains details of all psychiatric service contacts (including diagnosis) within the state and has been operational since 2000. All individuals treated by the clinic and found to have transitioned during this time period were included in the sample; there were no exclusions. The 24-month follow-up period was chosen because recent data from the North American Prodrome Longitudinal Study group in North America suggest that the majority of UHR patients who transition to psychosis do so within the first 24 months.6,7 This has also been the experience from data at the PACE clinic.6,31,32

Outcomes of interest

Transition to psychotic disorder

Psychosis threshold was assessed by the CAARMS, which stipulates daily psychotic symptoms of sufficient intensity for longer than 1 week (see Yung et al.29,33).

Psychotic diagnosis

Psychosis diagnosis on discharge from the service (or last recorded diagnosis if currently a patient of the service) was extracted and rated from the clinical files using DSM-IV-TR criteria. These diagnoses had been made following consultation with a consultant psychiatrist. We also used information from the clinical files in order to obtain an objective diagnosis using the operational criteria in studies of psychotic illness (OPCRIT) diagnostic tool.34 The OPCRIT diagnostic system has been shown to have good concurrent validity compared with consensus best estimate diagnostic procedures.35

Psychosis diagnoses were classified according to Baldwin and colleagues’36 groupings for first-episode psychosis, which have also been applied to the UHR population. This defines three categorical groups: schizophrenia spectrum disorders (including schizophrenia, schizoaffective disorder and schizophreniform disorder); affective psychoses (including bipolar I disorder, or major depressive disorder with psychotic features); and the other psychoses group (including brief psychotic disorder, delusional disorder, psychotic disorder due to a medical condition, substance induced psychotic disorder or substance induced mood disorder with manic features and psychotic disorder not otherwise specified (NOS) ). As the numbers for the affective psychoses were low, we combined the affective psychoses and other psychoses groups to produce a larger ‘non schizophrenia spectrum psychoses’ group.

Psychiatric co-morbidity at initial assessment (baseline) was documented by experienced
Psychiatrists using *DSM-IV-TR* criteria as part of the routine clinical assessment. Demographic and functioning data were available from the clinical files.

**Exposure of interest/independent variables:**

**BPD features**

*DSM-IV-TR* borderline personality features were routinely recorded by clinicians at service entry using Structured Clinical Interview for DSM-IV Axis II Disorder for BPD (SCID-II BPD) diagnostic module as part of the initial triage assessment by the assessment team prior to admission to the PACE clinic. This was part of a comprehensive assessment procedure. The assessment team clinicians were multidisciplinary and consisted of a consultant psychiatrist, a psychiatric registrar, psychiatric nurses, psychologists and social workers. Clinicians were trained to a rigorous standard, using SCID-II BPD operational criteria. Diagnoses for each individual are derived using the SCID-II BPD interview and all available clinical data. A consensus process with a senior clinician is used to maintain reliability of diagnoses, although interrater reliability data are not routinely collected. The assessment documents the presence of the nine DSM-IV BPD criteria or features, with the presence of five or more features signifying a diagnosis of BPD. We used a dimensional method of scoring in which 0 signifies the trait was not present, 1 signified that the criteria for the trait was probably met and 2 signified that the criteria was definitely met for the trait. Using these assessments, we were able to calculate the number of definite borderline personality features (maximum of nine), a diagnosis of full threshold BPD (five features and over) and also a BPD dimensional score out of 18 (as each of the features were rated as either absent, probable or definite (0, 1 or 2)). The diagnostic module was not specifically scored for some patients. In these cases, two experienced PACE clinicians rated ‘missing’ data on borderline features using the same diagnostic BPD module and the initial patient assessment documentation. This patient assessment includes a detailed 10-page document with a specific section for recording and assessing axis II pathology that is separate to the diagnostic BPD module.

**Statistical analysis**

All analyses were conducted using Statistical Package for Social Sciences (SPSS) for windows version 16 (SPSS Inc., Chicago, IL, USA). The case and control groups were compared in terms of demographic characteristics at baseline using simple chi-squared and *t*-tests. The number of patients with BPD and BPD features in the two groups (cases and controls) was compared using Pearson’s chi-square for categorical data (BPD/no BPD) and independent sample *t*-tests for continuous data (BPD feature dimensional score means) and non-parametric tests (Mann–Whitney U) for ordinal data (number of BPD traits). Similarly, the association between borderline personality diagnosis/features and psychosis diagnosis group was assessed using Pearson’s chi-square and the dimensional scores using parametric and non-parametric (Kruskal–Wallis) analysis of variance. Fisher’s exact test statistic was used when expected frequencies were observed to be less than 5. For both these analyses, the outcome diagnosis was the dependent variable.

**Ethics approval**

Ethical approval was obtained from the local research and ethics committee prior to commencement of the study.

**RESULTS**

**Sample characteristics**

Fifty-one ‘cases’ were matched to 51 UHR ‘controls’. Full data on borderline personality features were only available for 48 cases, due to three of the clinical files being untraceable. Therefore, the sample was restricted to 96 (48 cases and 48 controls). Demographic data are presented in Table 1. There were no differences in the number of at-risk criteria met, functioning level at baseline, or assessment diagnosis between the case and control groups.

**BPD features/diagnoses by group**

SCID-II BPD interview assessments were available for 47 individuals. BPD ratings were made from the assessment documentation for 49 out of 96 (51.4%) of the total sample; there was no significant difference in the number of these ‘retrospective’ ratings undertaken for the control group (*n* = 28) and case (*n* = 21) group. Those who had BPD rating completed at assessment were more likely to be female than those who had them completed retrospectively. Otherwise, there were no differences in demographics or functioning between these patients. The interrater reliability of these
Overall, 14.6% of the sample met *DSM-IV-TR* criteria for BPD. Fewer cases had a diagnosis of BPD at entry to the service (5 out of 48, 10.4%) compared with controls (9 out of 48, 18.8%), although this difference was not statistically significant (Pearson’s chi² = 1.34, *P* = 0.25). Neither the number of BPD features nor the BPD dimensional scores were significantly different between those who made the transition to psychosis and those who did not (see Table 2). This was similar for median scores as well as mean scores. The BPD dimensional scores that were subject to retrospective ratings were lower than those whose BPD rating was made by clinicians at initial assessment. A secondary analysis was conducted investigating the difference between controls and cases excluding the retrospective ratings. Although the number of BPD features was higher in the control group (3.27 compared with 2.45), the difference was non-significant at the 0.05 level (Mann–Whitney U = 177.5, *P* = 0.28). The effect sizes for the difference in BPD ratings between controls and cases were small for all analyses (Table 2).

**TABLE 1.** Sample demographics, UHR intake group, co-morbidity and functioning scores split by cases and controls, with appropriate statistical comparison (total N = 96 unless stated)

<table>
<thead>
<tr>
<th></th>
<th>Cases (transitions)</th>
<th>Controls (non-transitions)</th>
<th>Total (n = 96)</th>
<th>Cases vs. controls</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender:</strong> Male (n, %)</td>
<td>22 (45.8)</td>
<td>22 (45.8)</td>
<td>44 (45.8)</td>
<td><em>P</em> = 1.0</td>
</tr>
<tr>
<td><strong>Age on referral:</strong> mean years (median, range, SD)</td>
<td>18.3 (18, 14.7–25.3, 2.7)</td>
<td>18.4 (18, 15.2–24.0, 2.6)</td>
<td>18.4 (18, 14.7–25.3, 2.7)</td>
<td><em>P</em> = 0.83</td>
</tr>
<tr>
<td><strong>UHR intake group:</strong> (n, %)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TG</td>
<td>5 (10.4)</td>
<td>5 (10.4)</td>
<td>10 (10.4)</td>
<td><em>P</em> = 0.61</td>
</tr>
<tr>
<td>APS</td>
<td>34 (70.8)</td>
<td>36 (75.0)</td>
<td>70 (72.9)</td>
<td></td>
</tr>
<tr>
<td>BLIPS</td>
<td>2 (4.2)</td>
<td>0 (0.0)</td>
<td>2 (2.1)</td>
<td></td>
</tr>
<tr>
<td>TG + APS</td>
<td>5 (10.4)</td>
<td>4 (8.3)</td>
<td>9 (9.4)</td>
<td></td>
</tr>
<tr>
<td>TG + BLIPS</td>
<td>0 (0.0)</td>
<td>1 (2.1)</td>
<td>1 (1.0)</td>
<td></td>
</tr>
<tr>
<td>BLIPS + APS</td>
<td>1 (2.1)</td>
<td>0 (0.0)</td>
<td>1 (1.0)</td>
<td></td>
</tr>
<tr>
<td>TG + APS + BLIPS</td>
<td>1 (2.1)</td>
<td>2 (4.2)</td>
<td>3 (3.1)</td>
<td></td>
</tr>
<tr>
<td><strong>Functioning level at intake:</strong> SOFAS (mean, SD)</td>
<td>49.3 (10.7)</td>
<td>51.5 (9.7)</td>
<td>50.5(10.1)</td>
<td><em>P</em> = 0.50</td>
</tr>
<tr>
<td><strong>Primary diagnosis at intake:</strong> (n, %)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>19 (39.6)</td>
<td>25 (52.1)</td>
<td>44 (45.8)</td>
<td><em>P</em> = 0.41</td>
</tr>
<tr>
<td>Anxiety disorder†</td>
<td>7(14.6)</td>
<td>6 (12.5)</td>
<td>13 (13.5)</td>
<td></td>
</tr>
<tr>
<td>Dysthymia/Cyclothymia</td>
<td>5 (10.4)</td>
<td>1 (2.1)</td>
<td>6 (6.3)</td>
<td></td>
</tr>
<tr>
<td>Adjustment disorder</td>
<td>2 (4.2)</td>
<td>2 (4.2)</td>
<td>4 (4.2)</td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>1 (2.1)</td>
<td>2 (4.2)</td>
<td>3 (3.1)</td>
<td></td>
</tr>
<tr>
<td>Substance abuse/dependence</td>
<td>2 (4.2)</td>
<td>2 (4.2)</td>
<td>4 (4.2)</td>
<td></td>
</tr>
<tr>
<td>Conduct disorder</td>
<td>0 (0.0)</td>
<td>2 (4.2)</td>
<td>2 (2.1)</td>
<td></td>
</tr>
<tr>
<td>None given</td>
<td>12 (25.0)</td>
<td>8 (16.7)</td>
<td>20 (20.8)</td>
<td></td>
</tr>
</tbody>
</table>

†Agoraphobia, social phobia, obsessive–compulsive disorder and panic disorder.

APS, attenuated positive symptoms; BLIPS, brief limited psychotic symptoms; PTSD, post-traumatic stress disorder; SD, standard deviation; SOFAS, Social and Occupational Functioning Scale; TG, Trait Group; UHR, ultra-high risk.

**TABLE 2.** BPD features (*DSM IV-TR*), BPD dimensional scores and number of patients with BPD diagnosis for cases and controls with appropriate statistical comparison and effect sizes

<table>
<thead>
<tr>
<th></th>
<th>Cases (transitions)</th>
<th>Controls (non-transitions)</th>
<th>Total (n = 96)</th>
<th>Cases vs. controls</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BPD features:</strong> mean, SD</td>
<td>1.94 (2.37)</td>
<td>1.75 (2.21)</td>
<td>1.84 (2.28)</td>
<td><em>P</em> = 0.62</td>
</tr>
<tr>
<td><strong>BPD features (excluding retrospective ratings): mean, SD #</strong></td>
<td>3.27 (2.96)</td>
<td>2.45 (2.58)</td>
<td>2.88 (0.77)</td>
<td><em>P</em> = 0.28</td>
</tr>
<tr>
<td><strong>BPD dimensional score:</strong> mean, SD</td>
<td>4.95 (4.78)</td>
<td>4.59 (4.43)</td>
<td>4.77 (4.59)</td>
<td><em>P</em> = 0.66</td>
</tr>
<tr>
<td><strong>BPD diagnosis:</strong> n, %</td>
<td>5 (10.4)</td>
<td>9 (18.8)</td>
<td>14 (14.6)</td>
<td><em>P</em> = 0.25</td>
</tr>
</tbody>
</table>

†Independent sample *t*-tests for continuous variables, chi-squared for categorical data and Mann–Whitney *U*-test for ordinal data.

BPD, borderline personality disorder; *DSM-IV-TR*, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision; SD, standard deviation.

retrospective assessments was very good (ICC 0.89).
Baseline BPD features/diagnosis and psychosis diagnosis at follow-up

Of those individuals with baseline BPD who developed a psychotic disorder, 2 of the 15 individuals (13.3%) developed a schizophrenia spectrum psychosis and 3 out of 30 (10.0%) developed ‘other’ psychoses. No individuals developed an affective psychosis.

There was no statistically significant difference in BPD features or dimensional scores for cases in the schizophrenia spectrum, affective and other psychoses groups (Table 3). As the numbers for the affective psychoses were low, we combined the affective psychoses and other psychoses groups as a secondary analysis to produce a larger ‘non schizophrenia spectrum psychoses’ group. Again, there was no statistically significant difference in BPD feature scores between these two diagnostic groups.

DISCUSSION

The main findings from this study were: (i) there was a substantial and clinically important rate of full-threshold BPD in a relatively young UHR population; (ii) individuals with full-threshold BPD at baseline or higher baseline borderline features were no more or less likely to make a transition to a frank psychotic disorder at follow-up than those without BPD or those with lower BPD features; and (iii) in those who made the transition to a psychotic disorder, the presence of baseline BPD or BPD features was unrelated to the type of final psychotic disorder.

Rates of BPD

The rate of full-threshold BPD in our UHR sample was similar, if slightly lower (14.6% compared to 17%), to that reported in Rosen and colleagues from the United States but higher than that of Lencz and colleagues. However, given the study design, we cannot be sure that this rate is truly representative of the clinical population as a whole. Samples were similar in terms of age and gender distribution. These are substantial and clinically important rates, especially in a cohort with a mean age of just over 18 years. Our reported rates are around five times that of community-derived samples of young people, where the prevalence is estimated at around 3% and similar to the rate of BPD in non-UHR samples of outpatient adolescents and youth.

TABLE 3. Number of borderline personality features (DSM-IV-TR), BPD feature dimensional scores and number of patients with threshold BPD diagnosis by psychosis diagnosis group, with appropriate statistical comparison

<table>
<thead>
<tr>
<th></th>
<th>Schizophrenia spectrum psychosis†</th>
<th>Affective psychosis‡</th>
<th>Other psychoses§</th>
<th>Total</th>
<th>Statistical comparison¶</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPD features (mean, SD)</td>
<td>2.27 (2.27)</td>
<td>2.00 (1.41)</td>
<td>1.83 (2.21)</td>
<td>1.97 (2.38)</td>
<td>P = 0.56</td>
</tr>
<tr>
<td>BPD features score (mean, SD)</td>
<td>5.45 (4.72)</td>
<td>5.50 (2.12)</td>
<td>4.74 (4.56)</td>
<td>5.00 (4.82)</td>
<td>P = 0.89</td>
</tr>
<tr>
<td>BPD diagnosis (n, %)</td>
<td>2 (13.3)</td>
<td>0 (0.0)</td>
<td>3 (10.0)</td>
<td>5 (10.4)</td>
<td>P = 0.87</td>
</tr>
</tbody>
</table>

†Schizophrenia, schizophreniform psychosis, schizoaffective disorder.
‡Bipolar I, major depressive disorder with psychotic features.
§Psychosis NOS, substance induced psychotic disorder, substance induced mood disorder with manic features, delusional disorder.
¶ANOVA for continuous variables, chi-squared for categorical data and Kruskal–Wallis test for ordinal data.

ANOVA, analysis of variance; BPD, borderline personality disorder; DSM-IV-TR, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision; NOS, not otherwise specified; SD, standard deviation.
Type of psychosis experienced in those with co-morbid BPD

The results indicate that BPD pathology at intake was not related to the onset of any particular type of psychotic disorder. This failed to support the hypothesis that UHR patients with BPD features would be more likely to develop non-schizophrenia spectrum diagnoses. Neither did they appear to be experiencing briefer psychotic episodes which would be reflected in diagnoses such as psychosis NOS and brief reactive psychosis. This finding is consistent with Miller and colleagues who found that psychotic episodes in patients with BPD typically last many weeks and were therefore diagnostically indistinguishable from schizophreniform illnesses.

The question still arises as to whether it might be possible to distinguish between individuals who have psychotic phenomena as part of their personality structure, especially manifest under stress, and those who are truly at risk of a schizophreniform psychosis. Both of these groups would be included in the UHR identification approach. Improving our ability to distinguish between these groups of patients is significant from the perspective of conceptual clarification of psychotic disorder and ‘borderline’ pathology. The overlap between the two constructs, and also from the perspective of treatment strategies. In accordance with the clinical staging model proposed by McGorry and colleagues, it is conceivable that there is an indistinct difference in clinical presentation between these two groups early in the course of symptoms and that they might only become more clearly distinguishable with the progression of illness. However, it is possible that there are subtle clinical indicators of differences between these two groups from fairly early in their presentation. One possibility is that the types of attenuated psychotic symptoms (i.e. their form and content) might be different in UHR individuals with BPD compared with UHR patients without BPD features; for example, patients with BPD might be less likely to experience thought disorder or less likely to experience delusions, or the content of these delusions might be different. Another possibility discussed in the literature is that there is a different type of self-disturbance underlying attenuated psychotic symptoms in individuals with borderline features (a disturbance of ‘narrative’ selfhood) compared with individuals with vulnerability to schizophrenia spectrum conditions (a disturbance of ‘basic’ or ‘minimal’ selfhood). It is also possible that the BPD feature of affective instability increases vulnerability to the effect of other unrelated risk factors (such as substance use, for example) for psychotic symptoms. Longitudinal follow-up of UHR cohorts will enable us to address these issues further.

Clinical implications

From our results, it appears that UHR patients with concurrent BPD pathology experience similar psychotic outcomes to those without BPD pathology, at least over the short term. The current study does not address whether UHR patients with borderline pathology might respond differently to current treatments compared with UHR patients without borderline pathology. There is evidence that psychosocial interventions, such as cognitive therapy, are effective in UHR individuals but it is not known whether this approach might have different effects upon particular UHR patient subgroups, such as those with previous trauma, or impulsive personality traits. Personality factors do appear important in the outcome of treatment for schizophrenia with ‘better’ personality functioning related to both improved symptom and functional outcomes. Treatment studies in the UHR population should endeavour to include assessments of personality and trauma in order to explore these issues further.

Limitations

There are a number of limitations to the current study. Firstly, it is possible that the relatively small sample size meant that the study lacked sufficient statistical power to reliably detect a difference between the two groups, increasing the possibility of a type II error. The majority of studies in UHR research have had the limitation of small sample sizes and therefore low numbers of individuals developing a frank psychotic disorder. Our case control methodology attempted to address this problem by ‘enriching’ the number of individuals who have developed a psychotic illness. We would argue that using this methodology, our sample is large enough to detect if any substantial difference existed between the groups.

Secondly, approximately half of the patients had missing borderline personality feature scores at initial assessment and required retrospective ratings using the assessment documentation. Although the interrater reliability of these ratings was very good, it is likely that such a method produced lower scores given that the assessment was made from clinical assessment notes alone. There were relatively more retrospective ratings made in the control group, which might have led to a
possible bias towards lower scores in this group. We completed a secondary analysis using only the data from those individuals who did not receive retrospective ratings and found no systematic difference. However, we cannot be sure that lowered scores overall did not reduce the likelihood of finding a difference between the groups. We were also unable to present reliability data on the ratings of BPD features made at initial assessment, given the assessments were made by highly trained clinicians and not researchers. We encourage the use of structured Axis II assessments in future UHR research, an area that has been thus far neglected, to help address this issue.

Thirdly, although we controlled for age, gender and treatment time received in the design, we were unable to control for other possible factors thought to be important in terms of transition to psychosis in young people.49 For this reason, we endeavoured to use BPD feature dimensional scores as well as a simple diagnosis of BPD.

Fourthly, the final psychosis diagnosis was made by the objective OPCRIT tool34 in order to derive an independent diagnosis from the clinical notes. The OPCRIT tool has been shown to have good concurrent validity35 but does rely on the quality of the record keeping. The results with regard to BPD and final psychosis diagnosis remained the same when we used diagnoses made by clinicians derived from the clinical files.

Finally, although controversial,49 there is now strong support for the validity of the BPD diagnosis in young people.39,50,51 However, we acknowledge the potential problems of rating BPD or personality traits in general in a youth population.

For this reason, we endeavoured to use BPD feature dimensional scores as well as a simple diagnosis of BPD.

CONCLUSION

Borderline personality pathology is relatively common in the UHR population. In this sample, the presence of borderline personality features was neither associated with a reduced risk of transition to psychosis, nor of developing a non-schizophrenia spectrum psychosis. In this sample, the presence of borderline personality features was neither associated with a reduced risk of transition to psychosis, nor of developing a schizophrenia spectrum psychosis. This remains a clinically important question for treatment approaches and improving predictive validity of UHR criteria. Furthermore, treatment trials in this population should examine the role of such personality factors in the prediction of outcome and response to treatment.

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