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Suicide and other causes of mortality in bipolar disorder: a longitudinal study

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ABSTRACT

Background. The high risk of suicide in bipolar disorder is well recognized, but may have been overestimated. There is conflicting evidence about deaths from other causes and little known about risk factors for suicide. We aimed to estimate suicide and mortality rates in a cohort of bipolar patients and to identify risk factors for suicide.

Method. All patients who presented for the first time with a DSM-IV diagnosis of bipolar I disorder in a defined area of southeast London over a 35-year period (1965–1999) were identified. Mortality rates were compared with those of the 1991 England and Wales population, indirectly standardized for age and gender. Univariate and multivariate analyses were used to test potential risk factors for suicide.

Results. Of the 239 patients in the cohort, 235 (98·3%) were traced. Forty-two died during the 4422 person-years of follow-up, eight from suicide. The standardized mortality ratio (SMR) for suicide was 9·77 [95% confidence interval (CI) 4·22–19·24], which, although significantly elevated compared to the general population, represented a lower case fatality than expected from previous literature. Deaths from all other causes were not excessive for the age groups studied in this cohort. Alcohol abuse [hazard ratio (HR) 6·81, 95% CI 1·69–27·36, $p=0·007$] and deterioration from premorbid level of functioning up to a year after onset (HR 5·20, 95% CI 1·24–21·89, $p=0·024$) were associated with increased risk of suicide.

Conclusions. Suicide is significantly increased in unselected bipolar patients but actual case fatality is not as high as previously claimed. A history of alcohol abuse and deterioration in function predict suicide in bipolar disorder.

INTRODUCTION

The lifetime risk of suicide in affective disorders has been estimated at approximately 15% (Guze & Robins, 1970). Although widely quoted in textbooks and review articles, a decade ago Blair-West *et al.* (1997) had refuted such a high rate as mathematically impossible and this was subsequently supported by Inskip *et al.* (1998) using a different methodology. Another potential explanation for previously higher estimates of suicide is that follow-up studies that have examined suicide and other causes of mortality have suffered from methodological limitations that may have led to a bias towards poor outcome. For example, studies have followed up all in-patients rather than first-episode cases (Angst *et al.* 2002), or have focused on first hospitalizations rather than out-patient or community samples (Osby *et al.* 2001; Hoyer *et al.* 2004). Furthermore, follow-up time has been relatively short, which inflates suicide estimates due to

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high rates of suicide soon after discharge from hospital (Fawcett *et al.* 1987). Risk factors for suicide have not been well studied to date (Hawton *et al.* 2005). Studies of selected cohorts of manic patients from the 1980s reported an increased risk for natural causes of death, particularly those due to circulatory system diseases (Tsuang *et al.* 1980). This has been challenged by more recent data based on consecutive hospital admissions (Schneider *et al.* 2001), which suggest that deaths from natural causes are comparable to those expected from reference populations.

The current longitudinal study (mean 19 years) of first-onset bipolar I disorder patients from within an epidemiological catchment area allowed us to estimate long-term risk of suicide and natural death and to examine potential risk factors for suicide. This sample included all first in-patient and community psychiatric contacts within a defined area and was therefore representative of bipolar patients without selection bias. We hypothesized that the risk of suicide and natural deaths would be lower than in previous studies and that factors known to be relevant to suicide in major mental illness (Hawton & van Heeringen, 2000), including male gender, significant deterioration in pre-morbid function and alcohol abuse (Isometsa *et al.* 1994), would be predictive of a higher risk of suicide.

METHOD

Identification of cases

Clinical and demographic data were collected on all patients from the geographically defined area of Camberwell in South London (population approximately 120 000), who presented with psychosis and/or mania between January 1965 and December 1999. Cases were initially identified using the Camberwell Case Register (Wing & Hailey, 1972) and then using hospital computer records (from 1984) to generate a list of all patients admitted with any possible psychotic illness (ICD-9 codes 295, 295.6, 297, 296.0, 296.2, 296.4, 298, 292.1 and ICD-10 codes F20, F25, F22, F30, F31.3, F31.2, F31.6, F28, F29, F12.5, F16.6, F19.5, F16.75, F19.75) in the Camberwell catchment area. In addition, all case records of patients from the area were examined to identify those who made contact with services but were not admitted. Patients who were admitted to hospitals outside the area would usually be transferred back to local hospitals or referred to local services for continuing care. These records were also identified in the comprehensive search for case-notes. The identified cases were checked to ensure they included not only all those whose first presentation to services was with mania but also those who presented with a first manic episode after a previously assessed or treated depressive episode. Patients who were not resident in the catchment area, had presented previously with a psychotic or manic episode, had a clear organic cause for their symptoms, or had onset before age 16 years were excluded from this study. The methodology is described more fully elsewhere (Castle *et al.* 1998; Boydell *et al.* 2003; Kennedy *et al.* 2005*a*, *b*). The study was approved by the Joint South London and Maudsley and the Institute of Psychiatry Research Ethics Committee.

Diagnostic procedure

Case records of the subjects, including medical, nursing, social work, and occupational therapy notes, together with all correspondence relating to the case, were examined and then rated using the Operational Checklist for Psychotic Disorders (OPCRIT), Version 3.4. This is a well-validated symptom checklist based on the Present State Examination (PSE; Wing *et al.* 1974). It has a glossary of explicit descriptions for each constituent item of psychopathology and instructions for coding the items and was designed with case-note review in mind. This enabled computer diagnoses to be made using the OPCRIT program (McGuffin *et al.* 1991) for the year after each patient's first presentation. All mania cases were rated by J.B. and N.K. Inter-rater reliability was monitored and found to be good ($\kappa = 0{\cdot}75$–$0{\cdot}94$) (Boydell *et al.* 2003; Kennedy *et al.* 2004). The DSM-IV diagnosis of bipolar I disorder was chosen for the analyses in this study as it is based on the latest generally accepted operational criteria.

Case-tracing procedure

The Office for National Statistics traced the cases and provided information that patients were alive or dead; for the latter, death details from death certificates were provided. Those people who had emigrated were treated as

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having left the study alive at the date of emigration and those whose whereabouts became unknown (because they were removed from health authority listings) were treated as having left the study alive at a mid-point between the date of removal and the date they were last definitely alive from case records. Official death details were rechecked against case-notes and any deaths designated as 'undetermined' by the death certificates were categorized as suicide for this analysis if case records showed some evidence that death was self-inflicted but had not been proven 'beyond reasonable doubt'.

Deaths from all causes except suicide were classified into four broad categories of circulatory system diseases (including cardiovascular and cerebrovascular), cancer-related, infectious and respiratory, and 'other', reflecting the major causes of death in England and Wales.

Statistical analyses

We conducted survival analyses starting with a Lexis expansion to take into account the differing lengths of follow-up, and to generate rates of suicide and all-cause mortality in person-years. Indirect standardization by age and sex was carried out using the 1991 England and Wales population as the standard. This involved using the 1991 England and Wales death rate and suicide rate for each 10-year gender and age group (Office of Population Censuses and Surveys, 2003) to estimate the expected number of deaths (all-cause and suicide) in the study population. Observed numbers of deaths were then divided by the expected numbers of deaths to calculate standardized mortality ratios (SMRs).

Likely risk factors for suicide from the clinical and sociodemographic information (from the OPCRIT checklist rated on the first year of case-notes) were tested individually using χ^2 tests. These comprised OPCRIT items gender, age of onset before 40 years, significant deterioration in pre-morbid function, lifetime alcohol abuse, any cannabis use, suicidal ideation, reckless activity (with high potential for painful consequences that patients do not recognize) and persecutory delusions and hallucinations. Deterioration from pre-morbid level of functioning was a global OPCRIT indicator that a patient did not regain pre-morbid social, occupational or emotional functioning after the first episode of illness. Only a limited number of variables were chosen to minimize type 1 error, given the relatively small sample size.

Multivariate Poisson regression modelling was then carried out to determine the effect of each risk factor identified as statistically significant at the 5% level from the univariate analysis, after adjusting for the other risk factors. Each risk factor was added in a stepwise fashion. The likelihood ratio test at the 5% significance level was used to decide whether the addition of each factor improved the model.

Interactions between the remaining risk factors were then tested, again using the likelihood ratio test at the 5% significance level.

Table 1. *Number of cases, person-years, age at first presentation, follow-up time, deaths and deaths by suicide for patients with bipolar disorder in the Camberwell cohort (1965–1999)*

	Males	Females
No. of cases	102	133
No. of person-years	1895	2527
Age at first presentation, mean (S.D.)	30·1 (13·5)	35·4 (16·5)
Length of follow-up in years, mean (S.D.)	18·6 (9·6)	19·0 (9·5)
No. of deaths	18	24
No. of deaths by suicide	7	1

S.D., Standard deviation.

RESULTS

Of the 1443 cases screened for the 35-year period, 244 met the criteria for DSM-IV bipolar I disorder, first manic episode, of whom 52 (21·3%) had been treated for a previous episode of DSM-IV depression and 28 (11·5%) had experienced untreated depression. Sixty-two patients (25·4%) first presented as out-patients. Five patients were excluded as they were non-UK residents who had been staying in Camberwell. Of the remaining 239 patients, four could not be traced beyond their initial presentation. Six patients had emigrated and the whereabouts of 13 patients were unknown: these were dealt with as described above. Table 1 shows the characteristics of the cohort. The mean age at first presentation was earlier for men (30·1 years) than for women (35·4 years) ($t = -2{\cdot}61$, df = 233, $p = 0{\cdot}01$).

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Table 2. *Time to suicide from first presentation for patients with bipolar disorder in the Camberwell cohort (1965–1999)*

Suicide no.	Time from presentation to suicide (years)
1	2·2
2	5·4
3	6·2
4	6·6
5	9·7
6	10·5
7	11·6
8	12·6

The total number of person-years of follow-up (sum of individual years of follow-up) was 4422 years. Approximately 7% of the males and <1% of the females committed suicide. For those patients who could be traced after 10 years or more (202; 86·0%), 8·9% had died, 2·5% by suicide, at the end of the first decade. The mean time to suicide from first presentation was 8·1 years and the range was 2·2–12·6 years, as shown in Table 2.

Figure 1 shows Kaplan–Meier survival curves of deaths by suicide and deaths from all other causes.

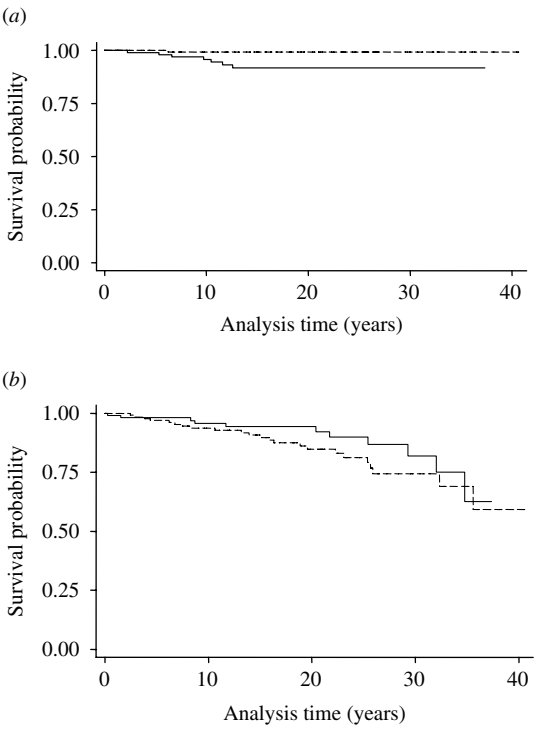

FIG. 1. Kaplan–Meier survival curves showing (*a*) deaths from suicide by gender and (*b*) deaths from all causes except suicide. —, Male; - - -, female.

Standardized mortality ratios

Table 3 shows that the SMR for all-cause mortality (SMR = 1·22) was not significantly higher than for the general population. The suicide mortality for males was more than 12-fold higher (SMR = 12·76) and for females was more than 4-fold higher (SMR = 4·27) than the 1991 England and Wales suicide mortality for the respective genders. Overall, the SMR for all causes of death other than suicide did not differ from that in the general population (SMR = 1·03), with the only statistically significant increase found in female patients for infectious or respiratory causes (SMR = 3·14). In particular, the risk of cardiovascular and cerebrovascular mortality was not elevated for either gender.

Risk factors for suicide

Taking into account the variable length of follow-up, the univariate analysis of possible sociodemographic and clinical risk factors (Table 4) showed that male gender, alcohol abuse, cannabis use and deterioration in pre-morbid function, up to a year after presentation, were significantly associated with completed suicide. There was no association with marital status or the potential clinical risk factors of suicidal ideation, reckless activity, persecutory delusions or persecutory hallucinations. Poisson regression modelling did not show a significant effect of age at first presentation [hazard ratio (HR) 0·97, 95% confidence interval (CI) 0·9–1·0, $p = 0·36$], on completed suicide.

Table 5 shows the results of the multivariate Poisson regression modelling, in which all statistically significant factors that were individually associated with suicide in the univariate analysis were included in a stepwise fashion. Only alcohol abuse and deterioration in pre-morbid function remained as statistically significant predictors and these were independent effects with no interaction between them. Cannabis use and male gender were no longer associated with suicide after adjusting for alcohol abuse and deterioration from pre-morbid function.

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Table 3. *Standardized mortality ratios (SMRs) by gender for patients with bipolar disorder in Camberwell, 1965–1999*

	Males		Females		Total	
Cause of death	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
All causes	18	1·60 (0·95–2·53)	24	1·14 (0·73–1·70)	42	1·22 (0·88–1·64)
Suicide	7	12·76 (5·13–26·29)	1	4·27 (0·11–23·78)	8	9·77 (4·22–19·24)
All causes except suicide	11[a]	1·07 (0·53–1·92)	23	1·13 (0·71–1·69)	34	1·03 (0·71–1·44)
Cardiovascular/cerebrovascular	5	1·04 (0·34–2·44)	8	0·87 (0·38–1·72)	13	0·86 (0·46–1·46)
Cancer	3	0·95 (0·20–2·76)	8	1·36 (0·59–2·68)	11	1·14 (0·57–2·03)
Infectious/respiratory	2	1·91 (0·23–6·88)	7	3·14 (1·26–6·46)	9	2·49 (1·14–4·72)

Obs, Observed; CI, confidence interval.

[a] One male patient was found dead at home and his cause of death remained unknown. He was therefore not included either as a suicide or in the subcategories of all causes except suicide.

Table 4. *Univariate analysis: risk factors for suicide adjusted for length of follow-up*

Characteristic		Suicide (% cases)	χ^2	*p* value
Gender	Male	6·9	6·56	0·01*
	Female	0·8		
Marital status	Single	4·7	1·26	0·26
	Other	1·9		
Age at first presentation	<40 years	4·4	2·41	0·12
	>40 years	0		
Suicidal ideation	Ideation	0	0·11	0·74
	No ideation	3·5		
Reckless activity	Reckless	3·1	0·28	0·60
	Not reckless	4·8		
Alcohol abuse	Abused alcohol	12·5	9·26	0·002*
	No abuse of alcohol	2·0		
Cannabis use	Used cannabis	10·2	8·64	0·003*
	Not used cannabis	1·6		
Persecutory delusions	Experienced	4·0	0·29	0·59
	Not experienced	2·7		
Persecutory hallucinations	Experienced	0	1·21	0·27
	Not experienced	3·9		
Deterioration in pre-morbid function	Deterioration	9·8	8·11	0·004*
	No deterioration	1·6		

Significance: * $p < 0·05$.

DISCUSSION

This article reports the findings from what we believe is the first long-term longitudinal mortality study of a well-defined sample of first-onset bipolar patients from a specified catchment area. Although mortality by suicide among bipolar patients in this cohort was more than nine times that of the general population, the actual case fatality was low over a long follow-up period compared to extrapolations from short-term studies of 15%. The risk of death from natural causes was not raised for the middle-aged groups we were able to study in the mean 19-year follow-up. Furthermore, alcohol abuse and deterioration from pre-morbid function were associated with suicide.

Suicide mortality

The results of our study broadly concur with those of the Danish and Swedish national in-patient studies, in which patients were followed from their first admission for bipolar disorder. Although our SMRs were lower, especially for females [male/female: 12·8/4·3 *v.* 16·2/20·3 (Denmark; Hoyer *et al.* 2000) *v.* 15·0/22·4 (Sweden; Osby *et al.* 2001)] our CIs were wider (owing to the relatively small cohort) and encompassed the Danish and Swedish SMRs. However, the low overall case fatality in a cohort that had more than 98% follow-up may reflect the fact that our study was not limited to those most severely ill bipolar patients who needed hospital admission and also that the risk

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Table 5. *Multivariate analysis: risk factors for suicide from Poisson regression modelling – best-fit model*

Risk factor	HR	95% CI	*p* value
Alcohol abuse	6·81	1·69–27·36	0·007*
Deterioration in pre-morbid function	5·20	1·24–21·89	0·024*

HR, Hazard ratio; CI, confidence interval.
Interaction between alcohol abuse and deterioration in pre-morbid function: HR 2·10, $p=0·63$, likelihood ratio test 0·25, $p=0·62$.
Significance: * $p<0·05$.

of suicide is not homogeneous over the course of the illness; our mean follow-up period was more than 7 years longer than the mean for either of these studies.

It is also possible that, over time, improvements in treating bipolar disorder (for example with lithium; Cipriani *et al.* 2005) and reduction in stigma associated with this disorder may be contributing to a reduced suicide rate. Further research would be needed to clarify these suggestions.

Mortality from all causes

In contrast to earlier studies, we found no major differences in the risk of death from all other causes for bipolar patients when indirectly standardized to the general population. The only elevated SMR was for infectious/respiratory causes (SMR = 2·49, 95% CI 1·14–4·72). Some recent research is in agreement with our overall findings that mortality from natural causes is not significantly increased (Schneider *et al.* 2001). However, a longer prospective study (34–38 years) reported elevated SMRs, particularly for all vascular diseases (SMR = 1·69; Angst *et al.* 2002), although this was not limited to first presentations of bipolar disorder.

One possible explanation is that excessive natural deaths occur when coexisting physical disorders are present and bipolar patients may be hospitalized more frequently when they have complicating medical problems (Black *et al.* 1987). Therefore, mortality rates derived from follow-up studies of hospitalized patients, usually in centres with a known research interest in mood disorders, may be higher than for bipolar patients generally. Of note, a recent 16-year follow-up study of patients from a lithium clinic also showed that the risk of death from cardiovascular disease did not differ from that of the general population (Brodersen *et al.* 2000). Apart from any effect of lithium, it may be that well-motivated, concordant patients who are managed in out-patient settings have a better prognosis with their physical health than other patients with bipolar disorder. The nature of our epidemiological sample means that such patients would all have been included in the cohort.

However, our cohort has clearly not been followed for sufficient time for the greatest period of cardiovascular or indeed neoplastic risk to be studied, so we are unable to draw conclusions about deaths in later life.

The lack of elevated mortality from natural causes in younger age groups might, however, be seen to strengthen the premise that suicides were not under-reported in this study, by misclassification into other causes of death.

Risk factors for suicide

We identified alcohol abuse and deterioration in function measured up to 1 year post-onset as predictive of suicide in bipolar disorder and showed that age at first presentation, suicidal ideation, reckless activity, persecutory delusions or persecutory hallucinations were not associated with suicide. Although male gender and cannabis use were associated with suicide in the univariate analysis, these factors were not significant after adjusting for alcohol abuse and deterioration in function using multivariate analysis. Individual studies to date have not examined the diverse range of risk factors we were able to analyse. In a recent meta-analysis of risk factors, Hawton *et al.* (2005) noted the paucity of data on many variables, remarking that this in part reflected the low base rate for suicide. The low number of suicides is indeed a factor limiting the analyses possible in our study. Nevertheless, our results concurred with this meta-analysis and individual studies (Osby *et al.* 2001; Angst *et al.* 2002), with respect to the limited influence of male gender and marital status for bipolar disorder, in contrast to suicides in the general population, and the lack of association of suicide with clinical psychopathology such as suicidal ideation early in the illness (Fawcett *et al.* 1987).

Co-morbid substance use is associated with a higher rate of suicide attempts in patients with

bipolar disorder (Dalton *et al*. 2003), and further work is needed to determine if it is also associated with completed suicide. Interactions may exist between gender, severity of illness, impulsivity, alcohol use and mania. Certainly among male bipolar I suicide victims in Finland, rates of co-morbid alcoholism were shown to be higher than for women (Isometsa *et al*. 1994). A rapid decline from pre-morbid level of function (Goodwin & Jamison, 1990), rather than an insidious deterioration, may mean that some patients with bipolar disorder experience intense hopelessness (Hawton & van Heeringen, 2000) soon after first presentation, leading to the higher suicide rate in the first years of follow-up.

Methodology

The unique strength of our study design is that we were able to follow-up an unselected cohort of bipolar patients from the point of first presentation. This made them truly representative of the population of bipolar I patients from a particular geographical area. Furthermore, the diagnostic homogeneity attained by using operationalized diagnostic research criteria was far less subjective than relying on clinical diagnoses.

Caution should be exercised in direct comparison of our cohort with others, not only because they are not first-episode cohorts but also because of different inclusion criteria. For example, the cohort of 220 patients studied over 34–38 years by Angst *et al*. (2002) included those with clinically diagnosed 'bipolar schizoaffective' and bipolar II disorder, and the Jorvi Bipolar Study (JoBS; Mantere *et al*. 2004) studied a total of 191 Structured Clinical Interview for DSM-IV disorders-diagnosed bipolar I and II cases. Considering only the 90 bipolar I patients from the JoBS, the proportion presenting first with depression (44·9%) is still higher than for our cohort (32·8%), but this is likely to represent the lack of help-seeking for depression in this deprived inner-London area.

An inevitable limitation of a cohort of this nature is that the size of the sample may be too small to detect modest differences in mortality and the rarity of suicide means we also risk spurious findings regarding risk factors. In this respect, large register-based studies have some advantages in allowing extensive study of specific causes of death and different methods of suicide, as well as stratification by age at diagnosis. However, information on potential risk factors is limited to hospital administrative data, whereas in our study we had access to detailed clinical case-record information including data on alcohol and drug abuse, as well as psychopathology.

To study the period of greatest risk of non-suicide mortality, particularly from cardiovascular/cerebrovascular causes, we would need to prolong the follow-up period for more complete ascertainment of mortality, ideally over a further 20 years.

Other limitations include the possibility of systematic bias. It is unlikely that the rating of risk factors was biased because this was done to create a case register and not to test any particular hypothesis. However, the risk factors were only assessed using OPCRIT for the period until the first year after presentation. Although this is a limitation for some of the negative findings, several of the factors cannot change (e.g. gender) and, if confirmed by larger studies, the positive findings from the first year offer hypothetical opportunities for preventative work. We do not believe that there was selection bias towards mania-dominated illness as we checked for those who may have presented with depression or a mixed affective episode initially.

Unfortunately, data on suicide attempts during the follow-up period are not available to allow analysis of this as a consistently tested risk factor in previous studies. The relatively small number of suicides limited the power and the analyses that could be performed, for example with regard to whether patients had been hospitalized or not. If the recording of suicide on death certificates was more likely if the deceased was known to have had recent contact with psychiatric services, then this would have been a source of bias towards higher mortality ratios for suicide. The cohort was based in an area of London that has a higher than average level of deprivation and this may limit the generalizability of the study, although we may have expected higher rates of suicide in a more socially fragmented area.

Clinical implications

In conclusion, this study concurs with population-based first-admission studies that suicide

rates are higher in people with bipolar disorder than the general population, but the actual case fatality may not be as excessive as hitherto supposed. In the light of our results, we advise caution concerning the use of the concept of a 'lifetime risk' for suicide (Bostwick & Pankratz, 2000), as rates clearly fluctuate over time and vary according to whether patients are in a depressive episode compared to remission (Hoyer *et al*. 2004). Rather than using proportionate mortality (the percentage of those who died who committed suicide: 15% as commonly cited), case fatality (the percentage of the original cohort who died by suicide: 2·5% after 10 years in our study) may be a more appropriate estimate of suicide risk as Bostwick and others point out. Certainly it is in accordance with other up-to-date calculations, such as a lifetime suicide risk of 2·4% for any affective disorder, modelled using data on completed suicides from one English Health District and community-based prevalence rates of affective disorders (Boardman & Healy, 2001). Patients with bipolar disorder in the young to middle age group (approximately 30–50 years) did not have a higher risk of dying from other causes, although a longer follow-up period would be needed to evaluate this risk for older patients. The important findings of clinical relevance are that alcohol abuse and deterioration in function during the first year after onset were associated with suicide.

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DECLARATION OF INTEREST

None.

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