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PREVALENCE OF CO-MORBID BIPOLAR DISORDER AND MIGRAINE IN A REGIONAL HOSPITAL PSYCHIATRIC OUTPATIENT DEPARTMENT

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SUMMARY

Current literature suggests patients with bipolar disorder are at increased risk of developing migraine compared with the general population and patients with other affective disorders. This study examined whether this finding was evident in the outpatient department of a regional psychiatric hospital. Using a patient database (n=1083), records were screened for bipolar disorder (n=169) or self-report of migraine (n=46). 8 cases of co-morbid migraine and bipolar disorder were revealed (4.7% prevalence). This and the general prevalence of migraine (4.2%) are substantially lower than previously reported. Reasons for and implications of this finding are discussed.

Key words: *migraine - bipolar disorder – prevalence – headache - depressive disorders*

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INTRODUCTION

There is a long reported association between prevalence of bipolar affective disorder and that of migraine. However the estimates of such co-morbidity show considerable variation. It has been suggested that because of such apparently high co-morbidity, this may permit elucidation of the pathogenesis of the conditions using techniques such as genome wide analysis (Oedegaard 2010). Therefore it is important to evaluate whether this association is true or co-incidental – many psychiatric conditions appear to predispose to physical conditions (and vice-versa) but often with little biological basis. The present study aims to expand upon current knowledge by examining the co-morbidity of the conditions in a UK regional psychiatric hospital outpatients department.

METHODS

A database of main patient diagnoses and other reported co-morbidities, covering the previous four years and including 1083 individuals was searched manually for diagnosis of 'bipolar disorder' and 'migraine'.

RESULTS

169 bipolar patients were identified, representing 15.6% of the sample – this included those diagnosed as bipolar types I and II and 'probable' bipolar disorder where the patient did not meet the exact clinical diagnostic criteria based on the DSM-IV-TR classification. It did not include single episodes of mania.

46 patients with supplementary diagnosis of migraine were found, 1 recorded as 'basilar migraine'. For some cases this represented spontaneous voluntary reporting of migraine by the patient, others an answer upon direct inquiry and the remainder from definite medical diagnosis by a neurologist as found in patient records. Based on the patient sample of 1083, this would give an estimated prevalence of 4.2% in this population.

Further analysis showed that of the 169 cases of bipolar disorder, 8 had co-morbid migraine, giving a prevalence of 4.7%.

Routinely asking patients whether they had suffered from migraine had only been done in the past year, thus patients not seen within this time period were excluded to leave 619 individuals. This included 123 cases of bipolar, 30 of migraine and co-morbidity of 7, altering prevalence to 19.9%, 4.8% and 5.7% respectively.

DISCUSSION

Previously reported population-wide estimates of the prevalence of migraine have varied but range around 10% (McIntyre 2006), whilst that of bipolar is nearer 1% (Oedegaard 2010); co-morbidity estimates are much more variable, for example from 24.8% (McIntyre 2006) to 39.8% (Low 2003).

Thus the prevalence estimates in the present study are considerable lower regarding migraine and co-morbidity, and significantly higher for bipolar disorder (as would be expected from an outpatient psychiatric patient sample). There are several possible reasons for this.

The first explanation is that prevalence of migraine is genuinely lower in the UK – the figures quoted above drew from population questionnaire data (McIntyre 2006) and outpatient screening (Low 2003) in Canada. A number of neurological conditions show significant geographical variation in prevalence (such as multiple sclerosis) and this may extend to migraine.

Alternatively, the previously reported data may be incorrect – its wide variability indicating that there is no biological association. Questionnaire analysis for example is subject to recall bias, whilst certain clinical studies drew upon 'extreme' patient groups – in whom co-morbidity is more likely due to the severity of their condition. For example, one study considered patients with recurrent inpatient admissions for affective disorders and identified that 77% of those with type II bipolar illness were affected by co-morbid migraine (Fasmer 2001). Publication bias is a further possibility for the apparent lack of low reported co-morbidity.

A final reason for the observed difference is the nature of the collection of data in the database. Patients self-reporting 'migraine' may not truly be affected or received accurate physician diagnosis; similarly the criteria for bipolar disorder may have been variable. Patients recorded as having unipolar depression may have been misdiagnosed – observation suggests that the former may evolve into bipolar depression with time (Fiedorowicz 2011). Equally, treatment for both condition can include anti-epileptic drugs; these may prevent or alter the course of development the disease – for example by affecting neurotrophic factor expression (Rosenberg 2007). Thus much as early intervention for psychosis may improve outlook with regard to future development of schizophreniform illness (Marshall 2005), antidepressant treatment for bipolar disorder could limit progression of migraine. It is also possible that another contributing factor is simply inconsistent questioning and reporting by patients of migraine.

From a relatively small patient sample it is difficult to determine which of these factors is responsible in this case. The prevalence estimates are considerably lower than those previously reported to the extent that the results found here are likely to be an underestimate. Nonetheless, there is still a small (0.5%, or 0.9% with altered inclusion criteria) difference in prevalence estimates between the 'background' rate and that of co-morbidity with bipolar illness, possibly representing the 'tip of an iceberg' of genuinely increased prevalence. A combination of reduced patient report/diagnosis of migraine and confounding effects of bipolar treatment may thus have produced this pattern.

Previously it has been suggested that migraine is particularly prevalent amongst those patients with type II bipolar disorder (Fasmer 2001); however, of the 74 patients recorded as bipolar II in this sample, only 3 had co-morbid migraine – although not all patients were listed specifically as type I or type II in the database.

Interestingly, of the 8 cases of co-morbidity, 6 were female and 2 were male. Increased co-morbidity amongst females has often been reported (McIntyre 2006), (Low 2003), (Fasmer 2001). This may reflect migraine being more prevalent amongst women than men in the general population, but some studies have suggested that bipolar males are similarly likely to be affected as bipolar females with migraine (Mahmood 1999).

This work raises the need for further examination of the prevalence of migraine as a co-morbid condition with bipolar disorder. In particular, further analysis of UK regional psychiatric hospitals, examining prevalence with type of bipolar disorder and between sexes may assist elucidating the relationship.

Genome linkage studies alone may go some way towards identifying particular aspects of the conditions (Oedegaard 2010), but it is clear that there multiple pharmacological (Fiedorowicz 2011, Mahmood 1999) and neuronal (Kato 2008) processes as yet to be understood.

If, as suggested here, treatment for bipolar illness can reduce the incidence of migraine, this may permit novel approaches for the determination of the neurobiology of bipolar disorder and migraine.

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