In an effort to elucidate the heterogeneity of schizophrenia much research has been aimed at exploration of the relationships between its various symptoms and co-morbid disorders. It is hoped that the neurobiological implications of diagnostic overlap may bring new insight to the pathophysiology underlying these disorders, and conversely offer improved treatment options for these patients. In particular co-morbid OCD and schizophrenia may significantly influence the functional outcome of patients.1 2

Previous studies have reported the prevalence of OCD in patients with schizophrenia to be between 7.8% and 31.7%. 3 7 Defining obsessions as ‘persistent unwanted ideas not related to delusions’, Eisen et al.3 found that 7.8% of patients with schizophrenia or schizoaffective disorder who were directly interviewed (N = 77) (SCID - DSM-III-R) met the criteria for OCD.3 Using DSM-IV8 criteria, Bermanzohn et al.5 found that 29.7% of consecutively admitted chronic schizophrenia patients could be diagnosed with co-morbid OCD. Community surveys such as the Epidemiological Catchment Area (ECA) study9 (N = 20 861) have yielded an OCD co-occurrence rate of 23.7%, but the measurement instrument did not include diagnostic hierarchy rules. In a study6 that did include diagnostic hierarchy rules (SCID - DSM-IV), 14% of 50 first-episode schizophrenia, schizophreniform or schizoaffective patients met criteria for OCD.

The use of different definitions of OCD (symptoms versus disorder) and methodologies (chart review versus direct interview, patient versus community samples, schizophrenia versus schizophrenia spectrum subjects, lay versus clinician assessments, cross-sectional versus longitudinal design) has complicated comparisons between studies and has therefore possibly contributed to the considerable variation in prevalence estimates. Nevertheless, calculated co-morbidity rates support the conclusion of many studies that co-morbidity of OCD with schizophrenia is more than an incidental finding.10 This work raises the question of whether shared susceptibility factors, such as dopamine dysregulation, may in fact characterise patients with co-morbid OCD and schizophrenia.

To date, studies have mostly focused on Caucasian patients. Both schizophrenia11 and OCD12 are disorders with significant commonality across different cultures and ethnic groupings. Nevertheless, there is some evidence of variation in the phenomenology of schizophrenia across ethnic groups,13 and it has also been suggested that OCD may be less common in certain communities.14 Up to the present, however, with the exception of Koen et al.15 who reported a low rate (1.1%) of co-morbid OCD in first- and multi-episode male South African patients with schizophrenia, there exists a paucity of published data on the co-morbidity of OCD in non-Caucasian schizophrenia patients.

This study investigated the prevalence of OCD in a Xhosa-speaking schizophrenia group.
Subjects and methods

Subjects

Five hundred and nine participants with schizophrenia between the ages of 13 and 84 years completed all study-related assessments. Participants were recruited from inpatient and outpatient hospital services and community clinics throughout the Western and Eastern Cape provinces of South Africa.

Local mental health care workers familiar with the inclusion and exclusion criteria referred potentially suitable subjects for screening. Participants represented the full range of clinical syndromes seen in schizophrenia and demonstrated a range of behaviour with regard to adherence to medication regimens. Fully informed, written consent was obtained from participants and where necessary from a caregiver.

The study was conducted with the written approval of the ethics committee of Stellenbosch University and complied with the ethical guidelines for research involving human participants as set out in the Declaration of Helsinki (Edinburgh, 2000).

Patient inclusion criteria

Our sample included male and female participants: (i) known to have schizophrenia; (ii) who had maternal and paternal grandparents of Xhosa origin; and (iii) who had at least one living parent and one living sibling or alternatively two living parents, to complete the triad.

Patient assessment

After giving informed consent, each proband was interviewed by a psychiatrist (DN) or an experienced, trained research sister (IM) who administered the standardised Diagnostic Interview for Genetic Studies (DIGS) version 2 in Xhosa, the native tongue of all participants. The DIGS is a validated clinical assessment tool designed for diagnosing psychotic spectrum disorders and includes assessment for the lifetime presence of OCD. In all cases the clinical interview using the DIGS provided the primary diagnostic information. Additional information relating to family history and treatment record was sought from hospital chart records and information gathered from family members. Relevant demographic data, medical history, treatment history and pedigree information were collected from the proband and his or her family.

Patients were recruited over a 3-year period. All subjects were assessed simultaneously by both raters during the first year of the study, and thereafter regular calibration meetings were held.

Data analysis

The demographic parameters and prevalence of OCD in this sample were calculated using SPSS 10.0 for Windows.

Results

Demographics

Our sample comprised a non-sibship group (N = 301) and 100 sibships (N = 208) divided into 95 pairs, 2 trios and 3 fours. The mean age of the sample (predominantly male (77%)) was 36.4 (± 10.07) years at interview, with duration of illness being 13.25 (± 8.8) years. The mean age of onset of schizophrenia was 22.86 (± 6) years, and 4.4% of the participants were in the first year of their illness. Twenty-two per cent of the sibships were recruited from the greater Cape Town area, 14.8% from Port Elizabeth and East London, while the majority of patients were from rural Western, Southern and Eastern Cape areas.

The participants presented with a range of symptomatology as indicated by the global scores on the Schedule for the Assessment of Positive Symptoms (SAPS) and the Schedule for the Assessment of Negative Symptoms (SANS). Twenty-eight per cent had a positive score (mild to severe) on the hallucinations items, while 40% suffered delusions, 34% thought disorder, 27% behavioural changes and 70% affective changes. The total score (global items) of the SANS ranged from 0 to 24, with a mean of 10.21 (standard deviation (SD) 4.88), and the SAPS total score (global items) ranged from 0 to 20 with a mean of 4.25 (SD 4.82). Only 3 patients (none part of a sibship) in this group fulfilled criteria for lifetime OCD on the relevant section of the DIGS, therefore representing a total lifetime prevalence of only 0.5% in this sample.

Discussion

The prevalence of co-morbid OCD in this Xhosa-speaking schizophrenia group was surprisingly low at 0.5%. In addition, no concordance was noted in the sibships. Previously reported prevalence rates have tended to vary depending on patient and disease characteristics. A recent study of hospitalised patients with chronic schizophrenia found a 23.5% prevalence of OCD, while a 3.8% prevalence of OCD was documented in patients with first-admission psychosis. On the other hand, Craig et al. reported only a lifetime prevalence rate of 7.8% OCD. Importantly, however, our finding contrasts starkly with most OCD co-morbidity data in schizophrenia populations of Caucasian ethnicity, and therefore seemingly raises interesting questions about this phenomenon in non-Caucasian groups and black Africans in particular.

In addition to the low rate of co-morbid OCD in schizophrenia patients reported by Koen et al., Carey et al. (personal communication) found a similarly low prevalence rate for OCD (0.5%) in a randomised general community clinic setting (sample of Xhosa origin). The Carey study utilised the
translated and previously used Xhosa version of the MINI neuropsychiatric interview, translated and previously used Xhosa version of the MINI neuropsychiatric interview,13 administrated by experienced Xhosa native language speakers. Importantly, the prevalence rates for other diagnostic categories in this study, including anxiety disorders and psychosis, were in line with similar samples in the developing world.2 Furthermore, a large ongoing South African study on the genetics of OCD has only included 1 black patient (Christine Lochner — personal communication).

With regard to our study population, it is of particular note that no participants were using any of the newer atypical antipsychotic medications, therefore lessening the possibility of treatment-induced obsessive-compulsive (OC) behaviour.23 Furthermore, instruments with established reliability and validity were used and in an effort to make our sample more representative of the schizophrenia population at large, patients from both hospital and community settings were included.

However, our results are subject to a number of limitations. Firstly, the use of the DIGS as our structured clinical interview meant that results could only reflect lifetime full syndromal OCD. Therefore assessment of OC symptom presence and severity in the study group was not possible. Secondly, the diagnosis of OCD was based on a single cross-sectional interview which may have resulted in important historical information being lost. It is believed that symptom dimensions of OCD and schizophrenia have different patterns of exacerbation and remission during the course of the illness.1 Craig et al recently demonstrated that only 33% of patients with schizophrenia/schizoaffective disorder diagnosed with OC symptoms still had symptoms 24 months later. Thirdly, assessment instruments were orally translated into Xhosa. While every attempt was made to ensure equivalence, cross-cultural adaptation of instruments may have been preferable.

While the focus of this study was the determination of prevalence in order to subtype the sample for future genetic studies, further work is needed to delineate OCD symptom profiles in patients with schizophrenia across different ethnic populations. Certainly, cross-national comparative studies suggest that cultural factors may affect the symptom expression of OCD, although the reasons for the wide variation in prevalence are not known. If these data are replicated, it may support the hypothesis that cultural or genetic factors play a role in protecting against co-morbid OCD in patients with schizophrenia of Xhosa descent or mixed race. However, further work is needed to develop more culturally sensitive instruments to screen and diagnose OCD in patients with schizophrenia and other psychotic disorders. While it is recognised that specific biological mechanisms, including genetic and auto-immune mechanisms, may play a role in the pathogenesis of OCD, ethnic variations in these underlying factors are likely protective factors in certain groups. Further comparative studies to delineate these putative factors are warranted.

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References


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