INTRODUCTION

The term cycloid psychosis[1] is not included explicitly in the international classification system and this has hampered both clinicians and researchers alike. In the present international diagnostic classification systems, in order to increase the diagnostic validity and reliability, such cases are categorized as acute polymorphic psychotic disorders, with or without symptoms of schizophrenia, F-23 in ICD-10,[2] and brief psychotic disorder, 298.8 in DSM-IV,[3] but these categories appears to have outlived its validity and reliability, utility though remains. Also the duration of remitting psychosis specified in these classification systems is less than 1 month. The researchers in this study [4] found a modal duration of 2-4 months, with 43% falling in this range and suggest that the forthcoming edition of ICD look into this.

In the literature the varied presentation of acute psychotic episode is known by the synonyms of psychogenic psychosis (coined by Faegerman) or reactive psychosis (coined by Eric Stromgren)—popular terms used in Scandinavia.[5,6] The other synonyms are hysterical psychosis, atypical psychosis, Bouffée De’lirante (French psychiatry Mangan first described this way back in 1880), and cycloid psychosis (German school). This possibly highlights the cultural and sociological contexts of psychosis, but these differing terms lack reliability and validity, but possibly have utility in clinical practise.

It was Karl Kleist in 1926[7] who introduced the term cycloid marginal psychosis to describe cases, which do not meet the typical presentation of schizophrenia or bipolar affective disorder. These conditions include many cases of acute psychotic illnesses of limited duration, with recovery between recurrences. The acute features are psychotic, as in schizophrenia, but the course is episodic, as in manic-depression.

But it was Karl Leonhard[1] in 1957 who described cycloid psychosis and the three forms of cycloid psychosis.

The first one is anxiety-blissfulness psychosis, which may resemble agitated depression, i.e. in which affective symptoms predominate (on one pole, there are periodic states of overwhelming anxiety and paranoid ideas of reference and sometimes hallucination. The blissful phase/pole presents with expansive behaviour and grandiose ideas).
The second type is called confusion psychosis, presents similar to manic episode, i.e. in which thought disorder is dominant and presents with anxiety, distractibility, and a degree of speech incoherence out of proportion to the severity of flight of ideas. The clinical picture varies between excitement and a state of under activity with poverty of speech.

The third is motility psychosis, in which the striking changes are psychomotor activity (akinetic—which presents similar to catatonic stupor and hyperkinetic—which resemble catatonic excitement).

Despite the fact that cycloid psychosis contains a high prevalence of mood symptoms, Carlos Perris[8,9] felt that cycloid psychosis did not equal schizoaffective disorder and considered it a separate entity. He presented evidence to support its clinical diagnosis and also its predictive validity.

Perris and Brockington published diagnostic guidelines for cycloid psychosis in 1981 [Table 1].

Peralta and Cuesta[10] reported in their clinical and nosological study that with regards to most clinical variables and morbidity risk of mood disorders, cycloid psychosis was closer to mood disorders. Cycloid psychosis had higher psychosocial stressors than schizophrenic and mood disorders. Affective and non-affective groups of cycloid psychosis differed in a number of variables indicating an overall better outcome for the non-affective group.

Brockington, Perris, Kendell et al. reported good outcomes in their study.[11]

I would now like to describe one such case, which got us thinking, a 40-year-old lady presenting the fourth time with brief and acute onset of confusion, hypo-motility, and psychotic features.

**CASE REPORT**

Ms. A, a 42-year-old Caucasian lady, divorced but currently in a stable relationship since the last 10 years, and working full time as a cleaner in a school were admitted the fourth time with acute onset of confusion, hypo-motility, altered behaviour, and psychotic features.

Her first admission had been in the summer of 2006 followed by three more episodes. She had good inter episode recovery between the episodes.

Prior to 2006, the only significant history was a high TPO Ab (Antithyroid Peroxidase Antibodies) which was diagnosed in December 2003 and she was started on thyroxine 50 μg. The dose was increased in June 2004 to 75 μg, which was further increased to 100 μg in July 2005. The current dose in July 2009 was 175 μg.

The first presentation was in August 2006, occurred after Ms. A returned from a holiday abroad with her partner. She was admitted with acute confusion, hyperpyrexia, agitation, and inability to walk and was also queried to have a seizure. She was admitted to the medical ward. Extensive investigations including haematological and biochemical studies, antibody studies, cerebrospinal fluid analysis, CT and MR imaging found no abnormality, except for raised free T4 (for which she was being treated with thyroxine). EEG was reported to be normal, and so also serum prolactin (the possibility of temporal lobe epilepsy was considered as a differential). No organic cause was identified.

She was then transferred to the psychiatric ward. Later on, in the ward she expressed thoughts that her body was dead and rotting, and that she was ‘Satan’. There were some olfactory and tactile disturbances as she could smell burning and feel ‘ooze’ coming from her, although she could not see it. She believed her family could hear her thoughts and was suspicious of them and also of the staff. Her memory of past events remained intact and her mood was euthymic both during the current presentation and also prior to the admission.

There was no past psychiatric history.

There was no family history of any mental illness.

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**Table 1: Diagnostic criteria for cycloid psychosis**

| 1. | An acute psychotic condition, not related to the administration or the misuse of any drug, or to brain injury, occurring for the first time in subjects aged 15-50 years. |
| 2. | The condition has a sudden onset with a rapid change from a state of health to a full-blown psychotic condition within a few hours or at the most a few days. |
| 3. | At least four of the following must be present: |
| a. | Confusion of some degree, mostly expressed as perplexity or puzzlement |
| b. | Mood-incongruent delusions of any kind, mostly with a persecutory content |
| c. | Hallucinatory experiences of any kind, often related to themes of death |
| d. | An overwhelming, frightening experience of anxiety, not bound to particular situations or circumstances (pan-anxiety) |
| e. | Deeper feelings of happiness or ecstasy, most often with a religious coloring |
| f. | Motility disturbances of an akinetic or hyperkinetic type, which are mostly expressional |
| g. | A particular concern with death |
| h. | Mood swings in the background, and so pronounced as to justify a diagnosis of affective disorder |
| 4. | There is no fixed combination of symptoms; in contrast, the symptoms may change frequently during an episode and show bipolar characteristics. |
There was no history of any illicit substance misuse, alcohol abuse, and no misuse of over the counter prescribed medication or other herbal medication.

The patient was started on olanzapine 10 mg OD (for psychosis) and sodium valproate 200 mg BD (for query seizure). She improved on the ward until all symptoms resolved within a couple of weeks. She was discharged and the episode was documented as an acute psychosis of unknown cause and was referred to the neurologist.

The olanzapine was stopped 6 months later as the patient continued to be well. In the outpatient follow up no evidence was found to suggest any psychotic or affective illness. The sodium valproate was stopped after a year, by the neurologist, just prior to the second episode.

The second episode, September 2007, occurred after Ms. A returned from a holiday. There was no recent life event. Within a few hours the patient experienced a sudden deterioration. She sat motionless, staring, and not engaging; only repeating “I think I might be dead.” There was no suicidal ideation or ideas of self-harm. She had paucity of speech and was unable to concentrate. The delusional beliefs that her body was rotting had also returned. She was treated by the crisis resolution and home treatment team, in the community, on this occasion and was recommenced on the olanzapine and sodium valproate, with a provisional diagnosis of intra-ictal psychosis/psychotic episode. The patient’s family and her partner did not report any history to suggest recent seizures. She recovered fully within a couple of weeks.

The neurologist did not find convincing evidence of intra-ictal or post-ictal psychosis (three EEGs were done in the tertiary hospital, as an out patient and no ictal events were recorded). The olanzapine was stopped subsequently in the outpatient follow up and she remained on the sodium valproate.

She presented in the same way, 1 year later, October 2008, after Ms. A returned from a holiday with sudden deterioration and responded well, within a few weeks to treatment in the community with olanzapine and support from the crisis resolution and home treatment team. Sodium valproate was continued. Again the family and the patient denied any drug misuse or alcohol misuse and urine drug screen was negative (15/10/08). There were no eye witness account of any seizures (On 13/10/08, the patient also had a video EEG during the episode of confusion and vagueness, which was again reported as normal).

In January 2009, because of weight gain, sodium valproate was tapered and stopped and she was commenced on levetiracetam by the neurologist, who continued with the follow up.

The most recent episode presented in July 2009, 3 days prior to going on a holiday. Again an acute presentation with confusion, answering in monosyllabic, looking perplexed, and believing that there were cameras in her flat and that she was part of a T.V. serial. She had symptoms of being unable to move her legs and was admitted to the hospital. She had delusions about her being dead and her body rotting were more elaborate and included in her thinking that she had killed everyone and was a mass murderer. This resulted in an incident of attempted hanging while in the ward, believing that she deserved to die as she had killed everyone. All the investigations were negative. EEG that was done 3 days after the admission was reported as normal. MRI head was requested and this showed several, tiny, non-specific hyper intense foci within the subcortical and deep white matter of both cerebral hemispheres. No new changes were noted as compared to the MRI done in August 2006. AntiNMDA receptor antibodies and antineutrophil antibodies, requested by the neurologist were negative. MMSE was done, 5 days after admission and it was reported to be normal. A second opinion was sought from an experienced psychiatrist. She opined that the clinical presentation matches that of cycloid psychosis. Also the family reported that the patient responded to olanzapine within 2 weeks, as evidenced on the previous three occasions. She was started on olanzapine again and made a full recovery within a few weeks and was discharged.

Subsequent to her discharge, Ms. A was seen in the OPC, both by the psychiatrist and the neurologist and there was no evidence, either of psychosis or affective illness. Her family raised no concerns either. She remains on 20 mgs of olanzapine and levetiracetam.

**DISCUSSION**

Given the fact that the first three episodes were following the holidays abroad, drug-induced psychosis was suspected but UDS (urine drug screen) has been negative on all four occasion and the partner and her family categorically deny alcohol or drug intake.

Clinical depression with psychotic features was also queried and discussed. The mood was euthymic prior to the first admission and the patient denied feeling low during the episodes and also after the episodes. The patient did have features of nihilistic delusions during the episodes. It can be argued that the patient might have had an underlying mood disorder which was aggravated during times of stress. Also both olanzapine and sodium valproate have been used in
of psychopathology, has been noted previously, but which has effects on the expression of a variety of forms of the four occasions.

Epilepsy was queried (post-ictal and inter-ictal psychosis), but EEG done during these episodes including video EEG was reported as normal. Serum prolactin done in the first and fourth admission was negative. Also the fact that there has been rather a consistent pattern, once every year, and that the epilepsy was well controlled makes epilepsy less accountable for the presentation. Forced normalization was queried, but the episodes were not related to changes in dosage of anti-convulsants. There was no definite eyewitness account of seizures. The consultant neurologist was not convinced and he reported that it was not linked to epilepsy.

The consultant neurologist queried Hashimoto’s encephalopathy on the fourth presentation, but in the absence of raised CSF protein, the absence of myoclonus and persistent cognitive decline, he dismissed it. Moreover, it is very rare and most cases of Hashimoto’s encephalopathy present at a later stage in life, and the patient responded to olanzapine on each of the four occasions.

A link with hormonal changes, especially estrogen, which has effects on the expression of a variety of forms of psychopathology, has been noted previously, but there was no link found in this patient. She had normal menstrual cycle and did not have any symptoms of PMT (premenstrual tension).

The relation between cycloid psychosis and thyroid disorder is interesting but as yet unconfirmed. Thyroid disorder cannot, however, be the direct cause in this case since the patient was being treated for hypothyroidism, well before (3 years prior) the first presentation in 2006.

Dissociative disorder was also queried, but there were no life events or precipitating events on each of the four episodes, nor was there any loss of personal identity or problems with self-perception.

Even though the patient was referred to the psychiatry team early on, it was only on the fourth admission that the team discussed cycloid psychosis and revisited Perris criteria, after the diagnosis was suggested by an experienced second opinion psychiatrist who had come to give a second opinion and who had come across such cases previously. The patient indeed met all the four points from the diagnostic criteria of cycloid psychosis as operationally defined by Perris and Brockington. The team now plans to continue with long-term antipsychotic treatment.

Rapid introduction of neuroleptics, followed by the addition of lithium, if required is advocated by Brockington, Perris, and Kendell et al. If there is severe anxiety, which does not respond to neuroleptics, clonazepam may be helpful. ECT has successfully been used in case reports.

CONCLUSION

Cycloid psychosis recognition has an important implication in the assessment, investigation, treatment, and effective management of recurrent confusional states with alteration of psychomotor activity and brief psychotic episodes. This will help both clinicians and researchers alike.

Learning points

- The accumulated evidence suggests that cycloid psychosis is a separate and an independent category. The current international classification system, i.e. the DSM IV and ICD 10, fails to categorize cycloid psychosis as a separate entity and this has hampered both clinicians and researchers alike. Will the forthcoming editions of DSM V and ICD 11 in 2011-12 categorize cycloid psychosis in a separate category?
- There is no fixed combination of symptoms, and in contrast, the symptoms may change frequently during an episode, resulting in potentially unnecessary invasive investigations. Educating medical doctors, psychiatrists and neurologist about cycloid psychosis is imperative given the pitfalls in the diagnosis.
- I have no conflict of interests.

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