CASE REPORT

Treatment resistant somatic delusions in bipolar disorder

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SUMMARY

Two patients each developed a single, fixed somatic delusion complicating their existing bipolar disorder. Both failed to respond to a range of antidepressant and antipsychotic medications. They each showed a partial response to clozapine and to electroconvulsive therapy, with resolution of mood symptoms and diminution of their ongoing somatic preoccupation. A review of case reports suggests a possible relationship between somatic delusions and affective disorders.

BACKGROUND

Two patients each developed symptoms creating diagnostic complexity. They had established episodic mood disorders but went on to develop single, sustained somatic delusions more typical of a delusional disorder.

There is a suggestion in previous literature that somatic delusions occurring in isolation represent a specific subtype of delusional disorder.¹ The literature also highlights a possible relationship between somatic delusions and affective disorders, with documented responses to antidepressants, antipsychotic medications or some combination of the two. However, there is very little information available regarding somatic delusions specifically in bipolar disorder.

These patients also increase our knowledge of effective treatment options. Both experienced high levels of distress and suffered sustained functional impairment as their symptoms failed to respond to a succession of treatments. However, both experienced clinically significant levels of improvement from electroconvulsive therapy (ECT) and clozapine. These treatments were sufficient to allow them relief from their distress and to resume community living.

CASE PRESENTATION**Case 1**

A 44-year-old woman with an established diagnosis of bipolar disorder developed a concern about discomfort in her mouth. This led her to consult her general practitioner, her dentist and, subsequently, a physician, seeking an explanation for this discomfort that she could not initially describe in clear terms. She had not previously had significant physical problems and had never described physical complaints at times of psychological distress. Over several months, this concern solidified into a fixed belief that food accumulated in her mouth after each meal, accompanied by worries that she would be unable to eat at her next meal. Her mood had

been stable for several years and there were no acute stresses in her life at the time, although she had been increasingly unmotivated and purposeless as her adolescent sons reduced contact. On this background, it was not immediately apparent that her mood was changing or that neurovegetative symptoms had emerged. After about 4 months, this clinical picture clarified to one of depressed mood with anhedonia, sleep disturbance and loss of 12–15 kg of weight. During and after the treatment of her depression she sought treatment from a number of specialists and later went on to have all of her teeth extracted.

The patient had previously experienced one episode of depression with perplexity and cognitive impairment but without delusions, which had responded to sertraline and olanzapine, and one episode of mania without psychosis, which had responded to sodium valproate. She had then been maintained solely on sodium valproate for several years. This mood stabilising drug should be prescribed with care in women of reproductive age, but the patient had obtained a tubal ligation after the birth of her second son (both of her children lived with her ex-husband).

She had no significant medical problems. There was no family history of psychiatric illness, although it was significant that her mother left the family when the patient was 6 years old and she was raised largely by her grandparents.

Case 2

A 45-year-old man with previous episodes of mania and depression developed a depressed episode approximately 6 months after his last manic episode. This occurred in the context of ongoing tensions with his wife, concerns for the younger of his two children, who had not been attending school, and probably non-adherence to the lithium and risperidone he had been prescribed. Over a period of a month, he lost interest in his usual activities and further withdrew from his family with reduced concentration, normal appetite and a fixed belief that he was sleeping no more than 1 or 2 h per night. He became preoccupied and increasingly distressed by this belief to the extent that his personal hygiene and self-care deteriorated. He presented to hospital demanding treatment and threatening suicide if he was not helped. He specifically believed that the duration of his sleep was inadequate rather than the experience of non-restorative sleep. While some patients may underestimate the amount of sleep they have, particularly when depressed, this patient's thoughts were

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significantly more fixed, persistent and preoccupying. This became more apparent as his depression resolved with treatment and the beliefs about his sleep persisted.

He saw this reduced sleep as a problem in itself—when pressed he would state that it tormented him and caused a pain in his mind, but he could not elaborate further and there were no identifiable feared consequences. It seems likely that this perception of suffering from the insomnia was a description of his depression, as this became less prominent as his depression lifted, although his preoccupation remained. Objective observations showed he was sleeping around 8 h a night; however, he maintained he was having no sleep whatsoever, and became agitated and irritable when this was discussed. He could not be convinced otherwise, despite measures such as maintaining a sleep diary and undergoing formal sleep studies. While observed by nurses to be sleeping, he would maintain that he had been awake and was aware of every check they had made. Similarly, he denied the results of the sleep study, stating that the machines were not accurate and claimed that although his eyes had been closed because he was trying to sleep, he had been awake for all but a couple of hours.

His previous episodes of mania and depression had resolved with usual treatment, and he had no residual symptoms, although his adherence to treatment had been inconsistent. He had no other medical problems other than previously diagnosed haemochromatosis, which had not required active treatment.

INVESTIGATIONS

Case 1

Nutrition: vitamin D 37 nmol/L (60–160), normal iron studies, elevated B12 and folate (presumably reflecting supplementation).

Endocrine: normal short synacthen test, normal thyroid function, low follicle-stimulating hormone and luteinising hormone, normal testosterone.

Immunology: negative for coeliac antibodies, normal ACE, normal α 1 antitrypsin and ceruloplasmin, negative antinuclear antibodies, negative cryoglobulins, negative anti-N-methyl-D-aspartate receptor antibodies.

HIV, hepatitis and syphilis serology all negative.

Cerebrospinal fluid analysis normal.

EEG and MRI normal, mandibular X-ray and orthopantomogram found no significant pathology.

SPECT scan showed minor bilateral prefrontal reduction in activity. Barium swallow normal.

Case 2

Nutrition: normal B12 and folate levels, elevated ferritin consistent with clinically mild haemochromatosis.

Normal thyroid function.

No significant abnormalities on CT of the head and EEG.

A sleep study showed stable oxygen saturation, periodic limb movements that did not disrupt sleep and several brief awakenings from rapid eye movement sleep (REM) sleep. There was a normal amount of REM sleep with medication-related delay to REM onset and it was concluded that 'overall sleep efficacy was preserved'.

DIFFERENTIAL DIAGNOSIS

As both patients had existing affective disorders with episodic courses when their somatic delusions developed, the most likely immediate diagnosis in each case would seem to be bipolar disorder and depressive episode with psychotic features.

However, where delusions are protracted and mood incongruent, the possibility arises of a primary delusional disorder with

comorbid affective symptoms. Affective disorders commonly occur comorbidly with delusional disorders but usually arise after the onset of the delusions.² In both of these patients, the somatic delusion coincided with the depressive symptoms and persisted when the affective symptoms had resolved. This is more consistent with the diagnosis of schizoaffective disorder as defined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), which requires that mood disorder symptoms have been present for most of the active and residual course of the illness, but that psychotic symptoms have continued for at least 2 weeks in the absence of significant mood disorder.³ Each of these patients have had a single, non-bizarre delusion for most of their illness course and have only had other psychotic symptoms during episodes of a mood disorder. Even then, the first patient described had never experienced psychotic symptoms sufficient to meet a diagnosis of schizophrenia and so could not meet the full criteria required in DSM-5 for schizoaffective disorder. So these patients' illnesses appear consistent with the core concepts of schizoaffective disorder, if not strictly with the current criteria.

This leads to some reflection on the relative merits of the diagnoses of bipolar disorder and schizoaffective disorder in our patients. Each of these diagnoses captures important elements of the patients' conditions, but the diagnosis of bipolar disorder with treatment-resistant psychotic features particularly describes the historic course of their illnesses and the later development of the treatment-resistant psychotic symptoms.

The diagnosis of schizophrenia is less relevant given the prominence of mood symptoms for both patients.

A preoccupation with a somatic problem also suggests the differential diagnosis of body dysmorphic disorder. This condition may be sufficiently severe that the preoccupation becomes delusional in intensity.⁴ However, this tends to be marked by 'flaws in physical appearance' rather than the disturbance of bodily function that occurred in these patients.⁵

The extent of the preoccupations experienced by these patients also raises the possibility of an obsessive-compulsive disorder, particularly as they contributed to subsequent behaviour. However, in both patients, their beliefs were accepted rather than resisted and were not recognised as being excessive or unrealistic. In Case 1, the patient would continually try to remove or push the perceived food in her mouth with her tongue. This movement was voluntary and could be ceased, and did not result in increased anxiety. In Case 2, the patient would become extremely frustrated if it were suggested to him that he had slept for longer than he believed was the case. He would continually seek treatment for his perceived insomnia in a variety of ways with no consistent or ritualised behaviour. It can still be difficult to delineate delusions from severe obsessive-compulsive disorder, but it is notable that in both patients their beliefs remained fixed over many years although their degree of distress and the severity of their other symptoms fluctuated.

The patients' beliefs could also be considered overvalued ideas. These can be difficult to distinguish from delusions, particularly as overvalued ideas have not been as clearly or consistently defined, but they tend to be strongly held beliefs or preoccupations that are less rigid than delusions. The beliefs held by our patients have some qualities of overvalued ideas, particularly as they were superficially more plausible and more preoccupying than delusions tend to be. In balance we felt that the rigidity with which the beliefs were held, and the lack of a relationship to other rational thoughts, made the beliefs more consistent with being delusions.

Case 1

The patient had sought extensive investigations and treatment for other possible oral pathologies from a dentist and a physician, with no cause being found. There was no impairment of swallowing, no swelling of the gums and no diseases of the teeth. The possibility of burning mouth syndrome was also considered, but the patient clearly described a belief that there was something in her mouth and distinguished this from any change to the sensation of her mouth (such as pain, discomfort or itching). Her only prescribed medication for the years prior to this presentation had been sodium valproate; this was raised as a potential cause by the physician and the mood stabiliser was changed to lithium, with no change in the severity of the patient's symptoms.

Her repetitive tongue movements also raised the possibility of a dyskinesia. This was considered less likely as the movements only developed subsequent to her belief that there was food in her mouth, she had not taken antipsychotics for years prior to the problem developing, and because she reported the movements to be in her voluntary control.

In the years since her initial presentation, she has not developed any other complaints, the sensation has not spread beyond her mouth and no medication initiation or cessation has brought any change to her sensation. This makes oral or neurological causes for her symptoms less likely, although they are difficult to exclude.

Case 2

As noted above, the patient underwent several sleep studies to objectively assess the quality of his sleep. Polysomnography did not reveal any marked impairment and was inconsistent with his self-reports. This suggested that he did not have a primary sleep disorder.

TREATMENT

Case 1

After trials of citalopram 20 mg and venlafaxine 150 mg, our patient had 15 treatments of bitemporal ECT. This brought a significant improvement in her mood. She continued to believe that food was stuck in her mouth but she was no longer as preoccupied by this and found it easier to eat. However, within weeks of discharge on escitalopram 20 mg and olanzapine 20 mg, her mood worsened. She was readmitted to hospital and had a course of 12 bitemporal ECT, again with improvement in her mood. Her mood gradually worsened over the subsequent months despite a trial of clomipramine to 150 mg.

After her delusion persisted on olanzapine 20 mg, quetiapine 600 mg and risperidone (dose unknown), each for extended periods, she was prescribed clozapine at 300 mg as the treatment most likely to be effective for treatment-resistant psychotic symptoms. It was recognised that clozapine is usually prescribed only for schizophrenia and that there is less evidence for its use in other psychotic illnesses. In this case, the use of clozapine was suggested by an experienced colleague who had been involved in the patient's community treatment. The patient was registered with the CPMS with a diagnosis of schizophrenia.

Combined with intensive rehabilitation in a community-based residential facility that provided daily contact and support with all activities, this brought a reduction in the intensity of the patient's preoccupation with her mouth and put her in a stable, euthymic mood. This was sustained for 3 years until she developed neutropaenia and the clozapine was stopped. On

clomipramine combined with olanzapine 30 mg and then amisulpride 400 mg, her mood worsened and her preoccupation with her mouth led her to stop eating.

She was admitted to hospital and started bifrontal ECT. The patient received a course of 23 thrice weekly ECT sessions followed by two at weekly intervals. Her mood, affect and oral intake improved although she remained preoccupied with somatic concerns. Her clomipramine was changed to phenelzine and her antipsychotic medication was changed to aripiprazole (up to 45 mg). The phenelzine was continued to allow the best possible trial although postural hypotension meant she was unable to tolerate a dose above 60 mg a day.

She is aware that previous assessments and investigations have failed to find a physical cause for her symptoms and will sometimes become distressed that 'nobody believes me' or that 'people think I'm imagining it'. However, she also denies being frustrated with health staff and continues to raise her delusions in almost all interactions. The responses of others make no difference to the fixity of her belief. Nevertheless, she has continued to accept psychiatric treatment willingly; her first depressed and manic historical admissions had been involuntary but her treatment throughout the period in question has been provided voluntarily.

The patient has been generally reluctant to engage in psychological approaches such as cognitive behavioural therapy. Behavioural treatments have included attempting to limit her seeking of medical treatment for her mouth and interrupting the pattern of continuously moving her tongue around to dislodge the imagined food, as both behaviours were felt to reinforce her preoccupation. Their success was limited by her poor insight, concrete thinking and limited willingness to alter her behaviour. She chose to limit her diet to soft foods but acknowledged that this did not alter her perception that the food remained in her mouth.

Case 2

After trials of mirtazapine to 60 mg and venlafaxine to 225 mg, and of olanzapine to 5 mg and risperidone to 4 mg, this patient received a course of 6 bitemporal ECT with rapid improvement. He was discharged on venlafaxine 225 mg and risperidone 4 mg a day although he remained concerned about his sleep to a lesser degree. Months later his preoccupation increased and he again presented in an agitated state. After trials of aripiprazole to 15 mg, quetiapine 200 mg and the introduction of lithium failed to produce a sustained improvement, he received a course of 12 bitemporal ECT.

His mental state stabilised on lithium, quetiapine 300 mg and venlafaxine 225 mg, to the extent that he did not require another admission for 4 years. During this time, he remained preoccupied with his perceived poor sleep.

He was then admitted to hospital involuntarily with elevated mood, grandiose beliefs and pressured speech. This period of mania was the only time in recent years that he was not concerned about a lack of sleep. His mood settled with lithium and olanzapine, whereupon he again became preoccupied with his perceived poor sleep. He received two further courses of bitemporal ECT, which improved his mood and decreased his preoccupation with his sleep. It was felt that his persisting preoccupation was of a degree that would significantly impair his functioning. When this failed to improve with olanzapine to 30 mg and then quetiapine to 800 mg, further opinions were sought from experienced colleagues and eventually a decision was made to trial clozapine as the medication most likely to resolve treatment-resistant psychotic symptoms. The patient was

prescribed clozapine up to 400 mg a day with levels in the therapeutic range.

Many of his hospital admissions have been involuntary, at least initially, during periods of greatest distress and agitation. However, other than during the manic admission described above, his treatment has generally continued on a voluntary basis after the first week of treatment.

To date, the patient has been unwilling to engage in psychological therapies and tends to quickly dismiss sleep hygiene, sleep diaries and other practical interventions as unsuccessful. He receives limited community support as he struggles to develop constructive relationships with workers around any goals other than treating his insomnia. However, he continues to make constant changes to his own routine such as altering the time and extent of exercise, and altering the timing of food and fluid intake, in attempts to improve his sleep.

OUTCOME AND FOLLOW-UP

Case 1

Given the patient's good response to ECT and relatively poor response to antidepressant and antipsychotic medications, it was decided to continue maintenance ECT after discharge. On discharge, the patient remained focused on her oral concerns but was no longer dysphoric. She had increased reactivity and there was increased prosody of speech. At this time she remains in the community with stable mental state and continues to receive weekly ECT.

In working with the patient, we have acknowledged that it is not possible to ever categorically exclude an organic cause for her somatic symptoms, but that in the absence of such a diagnosis or of a specific treatment, our goal is to minimise her distress and to maintain her functioning as far as possible. This approach has helped to maintain a therapeutic alliance.

Case 2

The patient continued clozapine for over a year. Although he remained concerned about his sleep there was a general improvement in his daily functioning and a reduction in his distress. This medication was eventually stopped due to concerns that he was unable to take it safely as he sometimes substantially increased the dose in an effort to improve his sleep. At this time he is able to care for himself and maintain restricted functioning at home, although his degree of preoccupation is greater than when taking clozapine and he continues to seek treatment to help his sleep. He has been prescribed a combination of lithium, clomipramine 300 mg daily, zuclopenthixol decanoate 300 mg once a fortnight and lorazepam 2.5 mg three times a day. This benzodiazepine was prescribed long-term as attempts to reduce it or change it to diazepam led to worsening agitation. As he had otherwise improved, it was decided to discharge him with this medication continued and his community psychiatrist has since been reluctant to risk the patient's brittle mental state by making further changes.

DISCUSSION

As originally described, delusional disorders have occurred in the absence of an affective disorder.⁶ However, over time, evidence has emerged that the relationship between these conditions may be more complex. These case reports, while they cannot demonstrate an association between these disorders, reflect the debate that has taken place.

There is evidence of delusional disorders occurring comorbidly with depressive disorder and that this may occur particularly frequently for the somatic subtype.⁷⁻⁸ Depressive disorders

with somatic delusions do not seem distinct from depression with other types of psychotic symptoms.⁹ However, 'perceptual' somatic delusions, such as feelings of fullness, tend to occur more in schizophrenia than in affective disorders, while disorders of functioning (such as insomnia) are more common in depression.¹⁰ The specific delusion of insomnia, such as occurred in Case 2, seems rare, but has been described by Chiu *et al*,¹¹ who reported a 56-year-old woman who 'complained bitterly' of insomnia that was not supported by observations and who took repeated overdoses of hypnotics in an attempt to sleep.

There is some evidence of a relationship between affective psychosis and bipolar disorder specifically; depression with delusional symptoms is associated with higher familial rates of bipolar disorder than non-psychotic depression, while delusions remain more common in schizoaffective disorder than in bipolar disorder.¹²⁻¹³ There are also case reports of comorbid bipolar disorder and delusional disorder. Vicens *et al*¹⁴ described four patients (two with paranoid type delusions, one with erotomanic delusions and one with delusions of infidelity), in three of whom the delusions persisted after the affective symptoms were successfully treated.

Delusional disorders are most often treated with antipsychotic medications, particularly pimozide.⁸⁻¹¹ It has also been observed that pimozide may be particularly effective for 'monosymptomatic hypochondriacal psychosis'.¹⁵ There are case reports where clozapine has been used and it has been noted that the delusion may persist although associated symptoms and general functioning improves.⁸ This is consistent with the cases discussed where clozapine brought some functional improvements until circumstances required it to be ceased. Clozapine is recommended in the NICE guidelines for schizophrenia in adults, the condition for which it is approved, where the patient has not shown an adequate response to two or more other antipsychotics.¹⁶ The guidelines do not make specific reference to clozapine in the related psychoses and it is notable that there is less guidance provided for such conditions where they prove treatment-resistant. While clozapine is not referred to in the NICE guidelines for bipolar disorder, the American Psychiatric Association's guideline for bipolar disorder recognised that while there is limited evidence for clozapine, it may have some advantages over 'treatment as usual'.¹⁷

There have also been suggestions that the delusional disorders have a relationship to the affective disorders. Depression commonly occurs comorbidly in delusional disorder, particularly when there are multiple delusions, and a factor analysis has suggested that depressive symptoms are a common feature of delusional disorder.¹⁸⁻²⁰ Munro reviewed a series of 49 patients with monosymptomatic delusions who were successfully treated with pimozide, eight of whom went on to develop depressive illnesses, and required a combination of pimozide and tricyclic antidepressants. He also reviewed a series of case reports where patients appeared to recover on antidepressant medications alone.²¹

In regard to somatic type delusions specifically, there have been a number of case reports where antidepressant medications have been effective.²²⁻²³ Clomipramine (at moderate doses of 120 mg or less) was used to treat a series of four patients with somatic delusions.²⁴ It has been noted by these various authors that this suggests some relationship between somatic delusions and the depressive/obsessive compulsive disorders.

These responses contrast with the observation that patients with psychotic depression are more likely to have a poor response to tricyclic antidepressants alone but a better response if combined with antipsychotics or if given ECT.²⁵ It has also

been suggested that the presence of mood-incongruent delusions in depression predict a good response to ECT.²⁶ In both of the cases described, ECT was the most effective treatment for their mood symptoms although it was less effective for delusions.

Learning points

- ▶ Somatic delusions should not be considered a treatment-resistant condition. There is evidence in the literature that they respond to a variety of treatments.
- ▶ There may be an association between somatic delusions and affective disorders. Some case reports suggest delusional disorder of a somatic type may respond to antidepressants, particularly clomipramine, although this was not the case in our patient.
- ▶ Where somatic delusions associated with depression do not respond to conventional medications, electroconvulsive therapy may be effective in reducing mood symptoms.
- ▶ Clozapine may also be effective for this patient group in maintaining symptom control and in improving functioning where delusions are treatment-resistant. However, this is not a generally accepted indication, should be carried out after other opinions have been solicited and may require specific approvals be sought.

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