

# Emergence of Othello Syndrome (Morbid Jealousy) in the Context of Executive Dysfunction and Stroke

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## Case Study

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## ABSTRACT

Morbid jealousy is a pathological state in which an individual becomes convinced that their partner has become unfaithful. Morbid jealousy can emerge as either part of a primary psychiatric disorder or secondary to an underlying medical condition. We report a 46-year-old male who presented with Othello syndrome and depression 24 years after a left frontotemporal haemorrhagic stroke complicated by epilepsy. Neuropsychological assessment identified evidence of executive dysfunction. Antipsychotic and antidepressant medication, alongside Cognitive Behaviour Therapy (CBT) resulted in full remission of his delusions of jealousy and associated psychiatric symptoms. Neuropathological and neuropsychological factors in the case are discussed alongside the existing literature. Whilst the underlying mechanisms underpinning morbid jealousy remain elusive, the case illustrates the potential role of executive dysfunction in the aetiology of morbid jealousy.

**Keyword:** Case report; Psychosis; Morbid jealousy; Delusion; Othello syndrome; Delusional jealousy; Pathological jealousy; Conjugal paranoia

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## INTRODUCTION

Morbid jealousy can be defined as a pathological state in which an individual becomes convinced that their partner is unfaithful. It is estimated to occur in up to 8% of patients under psychiatric services in the community [1]. Morbid jealousy can be delusional, whereby it is characterized by fixed false beliefs, or obsessional, whereby ego-dystonic ruminations are characteristic [2]. Morbid jealousy was first described over a century ago and the description of Shakespeare's eponymous Othello gave rise to the alternative name 'Othello syndrome' [3]. Morbid jealousy may arise in the context of a primary psychiatric disorder or secondary to an "organic" condition. Among psychiatric disorders, it is associated with schizophrenia and related psychotic disorders. Whilst among secondary causes, the strongest associations are with drug-induced, cerebrovascular, and neurodegenerative disorders [4]. There is limited evidence around the optimal treatment of morbid jealousy; however both pharmacological and psychological approaches have been described. In general, the prognosis of morbid jealousy is thought to be poor and is associated with high levels of suicide and homicide [2-6]. Despite several diseases, neuroanatomical, and physiological correlates of morbid jealousy, the precise underlying mechanisms remains unclear. Frontal lobe dysfunction is commonly implicated alongside right-sided lesions, and left-sided over activity has also been postulated [4-12]. Researcher have formulated that biological factors interact with psychodynamic factors to produce morbid jealousy [10,11]. We present a case of morbid jealousy arising in the context of an acquired brain injury that was successfully treated with a combination of pharmacological and psychological therapy. We discuss the potentially synergistic role of biological and psychological factors. The case supports the idea that the onset of morbid jealousy arises in the context of susceptibility toward fearing that one's partner is unfaithful combined with one or more precipitating events. In addition, the case illustrates the potentially important a etiological role of executive dysfunction.

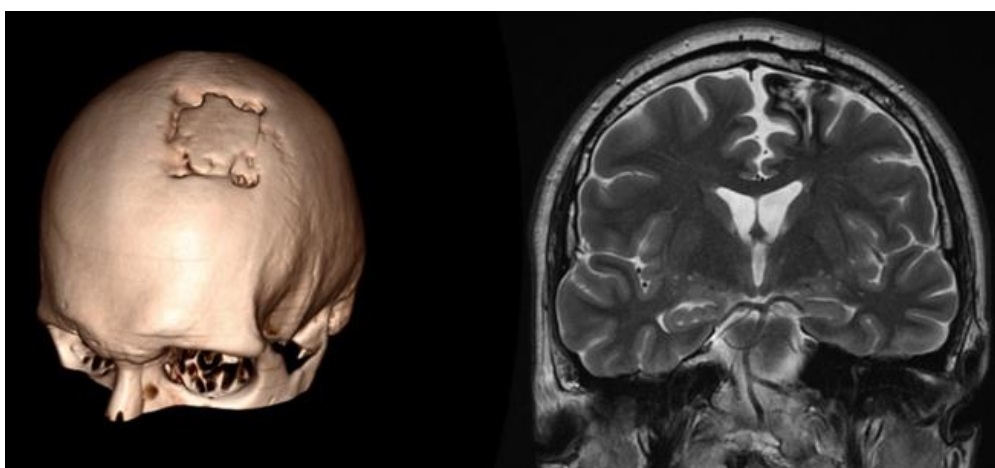
## CASE PRESENTATION

A 46-year-old right-handed male from Bosnia and Herzegovina who had lived in the UK for 28 years was referred by his neurologist to a regional neuropsychiatric outpatient service due to depression and attention-related difficulties. His wife reported long-standing difficulties with empathy, self-reflection, and preoccupation around her faithfulness. Over the course of three years, he subsequently developed a fixed belief that his wife was having an affair with associated ruminations and catastrophic thoughts. There was evidence of frequent reassurance-seeking, and occasional stalking and controlling behaviours. In an attempt to compel his wife to confess, the patient disclosed that he previously been involved in an extramarital relationship (this was later verified).

In terms of his medical history, 24 years prior he had developed seizures and was diagnosed with a left frontal lobe hemorrhage due to a ruptured arteriovenous malformation and underwent an emergency craniotomy. He was diagnosed with focal epilepsy with secondary generalization and had two episodes of status epilepticus requiring emergency intubation. He was on long-term treatment with phenytoin and had remained seizure-free for a 5-year period. In terms of previous psychiatric disorders, he had a diagnosis of post-traumatic stress disorder. Five years prior he had an episode of depression with irritability associated with commencing levetiracetam. There was no family history of psychiatric illness and no history of substance misuse. The patient's personal and developmental history was recorded where we got to know that he was delivered by a caesarean section but had an unremarkable perinatal period. The patient lived through adolescence during the Bosnian war, encountering significant psychological trauma as a civilian. At the age of 17, he relocated to UK, eventually graduated from University and embarking on a full time career. At this period itself, he got to know that his both father and mother were in multiple extramarital affairs. Despite a close relationship with both parents where he was unable to withstand knowing about their parents issue, subsequently, lead to lack of confidence in establishing intimate connections and had only been in one romantic relationship before getting married. At the time of referral, he was employed full-time in sales, was married and had 2 adolescent children. Mental state examination revealed psychomotor poverty and speech lacked free flow. His mood state was depressed with reduced affect and suicidal ideation. He experienced early morning waking, excessive guilt, reduced initiative, and anxiety. He had delusions of spousal infidelity with obsessive ruminations, which interfered with his thinking and behaviour. He exhibited occasional impulsive behaviors. His thought form was characterized by rigidity and perseverance. There were no hallucinatory experiences. Cognitively, he was fully oriented, however, he was vulnerable to distraction and his concentration faltered. He recognized that he was experiencing a psychiatric illness but lacked full recognition of his primary symptoms.

In terms of investigations, an MRI showed long-standing damage in the left superior frontal gyrus with focal loss of cortex and subcortical white matter and peripheral signal loss consistent with blood degradation products (Figure 1). These changes are compatible with sequelae of hemorrhage and resection of an arteriovenous malformation. A resting state EEG following the resolution of status epilepticus revealed background beta wave activity with superimposed focal slow waves present over the left frontal and temporal regions is shown in Figure 1.

**Figure 1.** MRI showing focal damage to the left superior frontal gyrus with loss of cortex and subcortical white matter and peripheral signal loss on T2 imaging consistent with blood degradation products (right) underneath the left mini-craniotomy (left).



Neuropsychological assessment found perceptual reasoning and processing speed were within the expected range (Wechsler Adult Intelligence Scale-IV (WAIS-IV). Memory (Doors and People test; Autobiographical Memory Interview) was also normal. He demonstrated mixed performance on executive function tasks (scoring at the expected level on the Brixton and Design Fluency tasks, but under-performing on Trial Making, and on the Working Memory index of the WAI-IV) showing some evidence of executive dysfunction. In addition, both the patient and their partner reported that he had executive difficulties on the Dysexecutive Questionnaire-Revised, although the only discrepancy in reporting was on items measuring 'executive cognition' items where the patient's partner reported a markedly higher level of difficulties, likely reflecting reduced insight into some of these difficulties.

A diagnosis of morbid jealousy (delusional type), in addition to organic personality disorder (dysexecutive syndrome) and a depressive episode (moderate severity), was made. Factors suggesting a poor prognosis at the time of diagnosis included an acquired brain injury with dysexecutive features, persistent depression, and delusional jealousy.

He was treated initially on sertraline, which was switched to mirtazapine (45 mg) due to lack of clinical efficacy. This was later augmented with venlafaxine (225 mg). His mood demonstrably improved within a short period, however, his psychotic symptoms persisted. He was also commenced on quetiapine (250 mg), which was later switched to risperidone (3 mg). Once stabilized on antipsychotics (risperidone) and dual antidepressant medication (mirtazapine and venlafaxine), he commenced a course of Cognitive Behavioral Therapy (CBT). He received an initial course of twelve weekly sessions, which was extended to eighteen due to persistent symptoms. Sessions focused on reducing the intensity and frequency of intrusive thoughts and his depressive symptoms. Behaviors targeted included reassurance seeking from his wife, reducing his ruminations, and improving his sleep. CBT strategies utilized included behavioral activation, core beliefs identification, behavioral experimentation, automatic thought detection, cognitive re-conceptualization, Exposure and Response Prevention (ERP), and relapse prevention planning. Risperidone was associated with hyperprolactinaemia with sexual side effects (reduced libido, erectile dysfunction). Interventions were otherwise not associated with adverse reactions and adherence was maintained throughout.

By the end of a course of CBT, the patient's conviction that his wife was committing adultery had reduced from 100% to 0%. He had also disengaged from all safety behaviours and his anxiety (GAD-7=7) and depression (PHQ-9=8) symptoms were in the low range. At 8 months follow-up, morbid jealousy and depression remained in remission whilst being maintained on mirtazapine (45 mg), venlafaxine (225 mg) and risperidone (3 mg). He had maintained a high level of functioning (GAF=78), with ongoing psychiatric symptoms largely transient. He described a high level of general life satisfaction and had a high-level occupational functioning. Main difficulties related to marital discord, for which he was referred to a psychosexual service.

## RESULTS AND DISCUSSION

The case describes the onset of morbid jealousy with concomitant depression and executive dysfunction following a background of previous cerebral hemorrhagic and long-standing treatment for epilepsy (with no recent seizures or anticonvulsant medication change). Treatment encompassed pharmacotherapy and psychological therapy and at a 8-month follow-up, he exhibited full remission of his morbid jealousy and depressive symptoms with excellent functional recovery. Several aspects of this case warrant interest. Firstly, how do we explain the emergence of morbid jealousy? The patient experienced two organic processes that may have contributed to the onset of this delusion, namely a left frontal hemorrhagic stroke and secondary focal epilepsy. Stroke has been associated with

morbid jealousy, particularly in the frontal lobe and right side, however, there are some reports of left-sided frontal lesions [7]. Epilepsy is also a recognised cause of brain injury, especially when leading to status epilepticus. Structural and neurophysiological changes in the left temporal and frontal regions were confirmed and it is likely that the focal pathology contributed to organic personality disorder dysexecutive syndrome.

In terms of alternative explanations, epilepsy-induced psychotic phenomena (ictal or post-ictal) are unlikely in view of the chronic nature of the delusion and a seizure free period of over 5 years. An aetiological role for medication phenytoin was similarly not considered likely. Psychotic symptoms, with abrupt onset, have been reported in the context of phenytoin-induced toxicity, however, they are typically associated with other neurological features. Here, there was no biochemical or clinical evidence of phenytoin toxicity and the patient had been treated with phenytoin for several years. An idiopathic (i.e. primary psychiatric) disorder was considered unlikely in view of the late onset and absence of risk factors typically associated a primary psychotic disorder (e.g. family history, an at risk mental state). However, this should not negate the potentially important role of contributory psychosocial factors in the development of the emergence of morbid jealous including his early life experiences of parental infidelity, his own insecurity, guilt lead to own an extramarital affair and the impact of this made him lose his self-identity which is discovered in pre-existing neurological diagnoses. Of particular interest in this case was the neuropsychological assessment, through which we were able to identify evidence of executive dysfunction with relative preservation of other cognitive domains. Alongside this was evidence of reduced self-awareness of his difficulties. These cognitive impairments provide a mechanistic link between the acquired brain injury and the subsequent emergence of morbid jealousy. Executive dysfunction is likely to have underpinned elements of behavioural impulsivity, impairments in working memory and cognitive flexibility that may have provided susceptibility towards morbid jealousy.

Previous studies broadly support a strong biological basis underpinning morbid jealousy. In a retrospective study of 105 patients with Othello syndrome', 70% had a secondary cause, most commonly a neurodegenerative disorder, particularly dementia with Lewy bodies, Parkinson's disease and Alzheimer's' disease [4]. Interestingly, of reported cases of morbid jealousy occurring in the presence of stroke, lesions have been more commonly reported in the right cerebral cortex, rather than the left. Neurological disorders were present in most cases, with neurodegenerative diseases accounting for over three-quarters of cases. In terms of neuroanatomical correlates, reduced grey matter volume in the dorsolateral frontal lobes have been identified relative to age and disease matched controls [4]. In those for whom structural MRI was available, atrophy was most marked in the dorsolateral frontal cortex, an area of particular importance in executive function.

Also, of note in this case was that the delusion focused on an area of personal salience, that is, the faithfulness of his wife. Several factors may have played a role in predisposing him to develop a delusion of infidelity. In this context, his own intense experience of an extramarital affair may have also played a role acting both as a trigger to his presentation and resulting in him projecting guilt onto his partner and his delusion of her infidelity [13]. Early life experiences, especially the revelation of his parents' infidelity and his early struggles with feelings of inadequacy in establishing romantic relationships, could have played a role in fostering susceptibility to delusions of infidelity, which may have been further reinforced by a loss of identity (and social relationships) after acquiring a major brain injury and change in dynamic with his wife, who adopted a career role. Interestingly, among dementia patients, risk factors for morbid jealousy include a history of spousal infidelity, and health disparities between patient and spouse [14]. Morbid jealousy has also been associated with attachment style, low self-directedness, trait anxiety, and poor social adjustment which may also be relevant to the present case [15].

The patient made a full recovery with psychological and pharmacological treatment demonstrating that even in severe cases of morbid jealousy, remission can be achieved. Treatment with risperidone, venlafaxine and mirtazapine was both effective and generally well-tolerated. This is consistent with the evidence base, that often the combination of an antipsychotic and antidepressants may be indicated in cases of morbid jealousy in the context of a primary or secondary cause [16]. There is also evidence in a small number of cases of patients with delusional jealousy, following an ischemic brain injury, that this combination is effective [17,18]. There is also evidence in favour of the use of psychotherapy, in particular cognitive-based therapy; however, this has tended to focus on patients with non-psychotic symptoms [5,19].

In terms of limitation, it is important to acknowledge that as a case report, the case has limited generalizability. Furthermore, due to the close temporal correlation between the commencement of antipsychotic treatment and CBT, it is difficult to ascertain the relative contribution of these treatments to the patient's remission. Morbid jealousy is an important and under-investigated area of psychiatry with important implications for patients and carers. Whilst several aetiological factors have been identified, the pathophysiological mechanisms are still not well understood. There is a need for the systematic study of morbid jealousy, including putative aetiological factors, to widen our understanding of this fascinating phenomenon and identify potential new treatment targets.

### **PATIENT PERSPECTIVE**

I have experienced transformation with CBT therapy but still feel there is so much more on my road to recovery. I am adamant that Psychotherapy will help me with my health goals.

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Written informed consent was gained from the patient

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### **AVAILABILITY OF DATA AND MATERIALS**

Not applicable

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