Psychotic PTSD? Sudden traumatic loss precipitating very late onset schizophrenia

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SUMMARY

Early theories of schizophrenia considered the illness as a fragmentation of mental content in response to psychological trauma. Here we present a case of very late onset schizophrenia in a previously high-functioning man in his mid-60s, precipitated by having lost his family in a terrorist attack, while he was living in Africa. He presented with symptoms consistent with post-traumatic stress disorder, however also exhibited visual and auditory hallucinations and marked deterioration in daily functioning. He showed mild impairment on cognitive testing, however brain imaging and screening for reversible causes of cognitive impairment were normal. The case highlights the need for a formulation-based approach to understanding and managing responses to severe trauma, from resolution through to psychotic disintegration.

BACKGROUND

It is known that post-traumatic stress disorder (PTSD) can manifest with secondary psychotic symptoms, for example, flashbacks and hypervigilance can be associated with persecutory delusions. However, in clinical practice hallucinations and delusions that are readily understood as reactions to stress are liable to be labelled ‘pseudo-psychotic’. To draw attention to the putative role of trauma in the aetiology of psychosis, we describe a case in which a more generalised psychotic illness, with features of PTSD, followed a severe traumatic loss event in later life.

CASE PRESENTATION

We present a case of a 68-year-old man, of East African ethnicity, who developed first episode psychosis after he was involved in a terrorist attack in his home country, which bercaved him of his wife and children. He was one of only a handful of survivors.

We have little information pertaining to the patient’s personal experience of the attack, since he was unable to give a coherent account of this. Several months after the attack his siblings, all of whom were in the UK, heard reports from others that he was neglecting his personal care, eating out of bins and living on the street, despite his having a home. Due to his relatives’ concern for his health they organised a flight for him to come to the UK. He sought asylum and he has since been living with his brother.

On arriving in the UK he was experiencing florid visual and auditory hallucinations of his wife and children. Despite explanations to the contrary, he continued to believe his family were alive and that they were present with him. He also showed marked deterioration in his ability to care for himself, requiring the assistance of his family for washing, dressing and toileting, and constant supervision due to chaotic, distracted and disinhibited behaviour.

His family reported that he showed symptoms consistent with increased startle and hypervigilance, for example, if he heard loud noises he would jump under the bed. His brother said that this is how the family would have had to respond while in hiding during the civil war several years previously. The patient also became distressed at the sight of potential weapons, such as knives.

The patient is the second eldest of nine children, bought up in a stable family home in Africa with no birth or developmental problems. He completed his education to college level and worked as a security officer and a salesman. He married and had nine children.

He had no previous mental health problems, and premorbidly was described by his brother as reliable, kind and generous. All his siblings moved to the UK some years ago, however he remained in his home country, reportedly because he enjoyed life there. His family are wealthy and own several properties, which he used to manage on behalf of his siblings, indicating a relatively high level of social functioning premorbidly.

Several years before the incident that precipitated his move to the UK, his country had experienced civil war, during which the patient’s family went into hiding. On occasion he was held captive by militants for several days. There was no history of his having endured torture.

He has no known history of substance or alcohol misuse, and no notable medical history besides treated hypertension. His maternal aunt developed a psychosis when in her 60s, which manifested as chaotic and disinhibited behaviour requiring residential care.

Mental state examination

Appearance and behaviour

On initial assessment he presented with unkempt hair and beard, though was dressed appropriately, and his personal hygiene was adequate. He was highly distracted, continually looking around the room, and his manner fluctuated from passive indifference to a degree of social disinhibition, for example, ‘high fiving’ the doctor. He showed some facial grimacing and made writhing movements of his hands.
Speech
Even with the assistance of an interpreter, the patient was initially electively mute, requiring prompting to vocalise his answers to questions, rather than miming with his hands. When vocalising, his speech was limited to short sentences, with little subjective elaboration.

Mood
He was objectively labile in affect, at times grinning. When asked for a subjective appraisal of his mood he gave a ‘thumbs up’. His sleep was disturbed, with increased autonomic arousal and nightmares. He had normal appetite. He reportedly derived enjoyment from having a bath and going to a local café with his brother.

Thought
The form of his thought could not be fully assessed due to his poverty of speech, however there was no de facto evidence of thought insertion, thought block or thought withdrawal nor of passivity phenomena. He reported a delusional belief that his wife and children were still be alive and with him. He expressed no thoughts of harming himself or others.

Perception
He was seen to be responding to visual hallucinations, gesturing to his wife to go away, smiling and laughing to himself. It was also reported that he could hear sounds of people fighting, and experienced tactile hallucinations of his children climbing on him at night.

Insight
He had no insight into his illness.

Cognition
Our patient was oriented in place and person, and remembered clinicians between visits. He was oriented to the time of day and season, though reported that the year was 2014, a date prior to the traumatic event. He struggled to name objects without prompting, tending to give approximate answers. On cognitive assessment using the Rowland Universal Dementia Assessment Scale, a cross-culturally valid tool, he scored 21/30, 23 is considered to be the lower limit of normal; his predominant impairments were in left-right judgements and visuoconstructional drawing.

INVESTIGATIONS
An MRI of the brain showed unremarkable intracranial appearances with no evidence of significant intracranial pathology. There was an incidental observation of prominent bilateral reactive cervical chain lymph nodes. Screening bloods including full blood count, urea and electrolytes, liver function tests, HIV and syphilis serology, calcium, copper and caeruloplasmin, erythrocyte sedimentation rate and vitamin B12 were all normal. On first presentation he had mild folate deficiency and vitamin D deficiency, these were corrected with supplements.

DIFFERENTIAL DIAGNOSIS
The case presented a diagnostic challenge. Some features of the presentation, namely cognitive impairment, visual hallucinations and abnormal athetoid movements, suggested an organic process, such as frontotemporal dementia, encephalitis, HIV dementia or neurosyphilis. Normal brain imaging and screening bloods were to an extent reassuring in excluding these. Furthermore, over the course of a year of follow-up the patient showed gradual improvement in his ability to manage his daily living skills, rather than a decline as might have been expected from a dementia. However we note that a frontotemporal dementia, which often presents with gradual decline and subtle imaging changes, remains a possible differential.

Our patient’s history and mental state were consistent with PTSD. First, he had experienced a traumatic stressor, defined according to the International Classification of Diseases, 10th Revision (ICD-10) criteria for PTSD as a ‘a stressful event or situation of exceptionally threatening or catastrophic nature, which would be likely to cause pervasive distress in almost anyone’. Second, his deterioration followed a few weeks to months after the trauma, in keeping with ICD-10 criteria. Finally, the patient showed symptoms of PTSD, namely flashbacks (episodes of reliving trauma), nightmares, increased startle reaction, insomnia and avoidance of reminders of the trauma. Notably some of these symptoms, for instance startle in response to sharp objects, related to earlier traumas experienced during a civil war, rather than the attack in which he lost his family. Our patient also appeared to lack an emotional numbness or anhedonia often seen in PTSD.

Psychotic depression is another important differential. Collateral history from his brother revealed that early on in his illness he was often tearful. Also, his delusions and hallucinations could be considered congruent with depression and he had significant self-neglect. Yet during our consultations he presented with neither low mood nor flat affect, as would be expected for psychotic depression. Rather, his affect was labile with social disinhibition and he would often laugh and smile, at times in response to hallucinations. He also subjectively described his mood with a ‘thumbs up’ gesture and did not exhibit biological symptoms of depression.

Finally persistent visual hallucinations of the deceased, with associated delusional beliefs that his family were still alive, were consistent with a pathological grief reaction. However, when taken together with his incongruent affect and significant decline in his daily functioning lasting for over a year, these symptoms would also be sufficient for a diagnosis of schizophrenia according to ICD-10 criteria.

TREATMENT
► Olanzapine and sertraline were started at low doses a year after the trauma and slowly increased to doses of 15 mg and 100 mg, respectively.
► An occupational therapy assessment was arranged, leading to modifications to the home environment, for example, grab rails in the bathroom, and a safe-box in the kitchen to keep knives.
► A carer’s assessment was arranged, and an application made for direct payments to the patient’s brother, who had significantly reduced his working hours due to the burden of care.
► The patient was followed up in the outpatient psychiatry clinic.
► A referral was made for a neuropsychiatric opinion, which concurred that a non-organic psychosis was the most likely diagnosis.

OUTCOME AND FOLLOW-UP
Treatment with antipsychotic medication was associated with a reduction in agitation, improved sleep and reduced lability of mood. He has now been able to go swimming with his family, and is able to manage spending time at a local café. At interview
he remained distracted, and spoke little spontaneously, though appeared more relaxed, and was well kempt and groomed. The concerted efforts of the patient’s family to promote his independence likely played a key role in his improvement.

### DISCUSSION

Historical conceptions of the pathogenesis of psychosis recognised the role of psychological trauma. In the later nineteenth and early twentieth century, splitting apart of consciousness or personality (dissociation) in response to trauma was considered central to the psychopathology of psychosis (for a review see 7). The term ‘schizophrenia’, literally ‘split mind’, as coined by Eugen Bleuler, reflects this fragmentation of mental content.8 The degree of instigating trauma required was thought to vary between individuals; in the most susceptible even everyday hassles could accumulate to trigger a psychotic breakdown. This aetiological model lost prominence in the second half of the twentieth century, with the introduction of diagnostic approaches to schizophrenia (from the Third Edition of the Diagnostic and Statistical Manual of Mental Disorders, DSM-III, onwards) organised around symptoms.9 Symptom-based definitions of psychiatric disorder shifted everyday practice towards viewing psychotic symptoms as primary and incomprensible, rather than as aberrant responses to a person’s life circumstances,3 a view associated with a concept of psychosis as rooted primarily in biology.10

Psychiatry has since acknowledged the link between psychosis and trauma once more, as evidence has emerged strongly suggesting trauma contributes to the expression of psychosis. In observational studies, childhood trauma confers vulnerability to schizophrenia.10,11 In adulthood there is also an excess of adverse life events prior to the onset of psychosis.12 Similarly, psychotic symptoms associated with PTSD are well documented.1 A recent review identified common developmental and symptomatological processes between PTSD and psychosis, for example, delusions and hallucinations have similarities to intrusions and flashbacks, and the negative symptoms of psychosis are akin to avoidance and emotional numbing seen in PTSD.1

A more plausible model therefore is that psychosis arises in the interaction between a person’s premorbid brain function and their life experience,13,14 with dissociation offering a route by which trauma can give rise to psychosis.15 Importantly, such a model allows psychotic symptoms to be understood psychologically. Although a framework for marrying dissociative processes with the neurobiology of schizophrenia remains the subject of ongoing research,16 having this model in mind guards against logical errors that can follow from an excessively narrow conception of its aetiology.17

Understanding the role of trauma in psychosis also has important implications for treatment. Treatment for PTSD focusses on a trauma-focussed psychological approach, with National Institute for Health and Care Excellence (NICE) recommending use of antipsychotics only where psychotic features are present.6 By contrast, medication remains the mainstay of treatment for schizophrenia; NICE recommends the use of cognitive behavioural therapy, however this is focussed on addressing symptoms.18 In a recent randomised controlled trial trauma-focussed therapy (TFT) has been shown to be safe and effective for patients with PTSD and comorbid psychosis,19 and significantly improved rates of remission from schizophrenia at 12-month follow-up,20 suggesting that TFT may also prove efficacious in psychosis more generally, including in cases of more insidious developmental trauma.21 A psychological approach appears particularly relevant for patients who have experienced severe trauma, where illness narratives are often highly complex, including political upheaval and the cultural dislocation associated with migration.

Previous authors have proposed that a psychotic PTSD subtype, also termed PTSD with secondary psychosis (PTSD-SP), should be considered a discrete diagnosis, where both psychosis and PTSD are precipitated by trauma in a person with no prior features of psychosis.1,12 Intact reality testing and a lack of thought disorder differentiate PTSD-SP from schizophrenia.1,2 Furthermore delusions in PTSD-SP are typically persecutory, whereas in schizophrenia they may also be bizarre and complex.2,22 In keeping with this, we postulate a spectrum of responses to trauma, organised according to the pervasiveness of dissociation, from peritraumatic dissociation leading to resolution (acute stress reaction), through failure to integrate traumatic memories with everyday experiences (PTSD and PTSD-SP), to a complete fragmentation of ego-consciousness (schizophrenia). We envisage that the less severe end of this spectrum is more commonly seen to follow abrupt trauma. As such, relatively few cases of schizophrenia result from an identifiable, sudden traumatic event.

In summary, the literature suggests psychosis following trauma is relatively common, however, not everyone who experiences trauma will develop a psychotic illness. Similarly, psychosis is multifactorial in origin and in most cases the contributing role of trauma will be difficult to disentangle. Cases such as this, with a precipitous and atypical presentation of schizophrenia following trauma in later life, are rare. The sudden and catastrophic nature of the trauma followed by the patients’ functional decline highlighted a probable causal relationship between the trauma and a psychotic disintegration, particularly when other causes, such as dementia, had been excluded. It is likely the family history of mental illness, and his previous exposure to traumatic stressors, predisposed the patient to a pathological response to new trauma. His illness was precipitated by the sudden and devastating loss of his family, an event he was unable to integrate into his understanding of the world, manifesting in hallucinations of his deceased family and disintegration of his personality. Loss of his support network following the trauma might further have contributed to his functional decline. Finally his recovery was impaired by his lack of insight and poor reality testing.

### Learning points

- Psychosis can follow trauma in adulthood; similarly post-traumatic stress disorder (PTSD) can present with psychotic features, such as visual hallucinations and associated affect-congruent delusions.
- Formulation of aetiological factors in mental illness is a helpful adjunct to diagnosis.
- A rule-of-thumb is to consider the predisposing, precipitating and perpetuating factors.
- NICE (National Institute for Health and Care Excellence) recommends treating psychotic symptoms in PTSD with antipsychotic medication.
- Consideration should also be given to the patient’s sources of social support, and possibilities for trauma-focussed therapy after initial stabilisation.

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Case report

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