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PHENOMENOLOGY OF PTSD AND PSYCHOTIC SYMPTOMS

Harriet Pepper[1] & Mark Agius[2]

[1]Magdalene College University of Cambridge, England
[2]Senior Research Fellow Bedfordshire Centre for Mental Health Research in Association with The University of Cambridge, England

It is well established that PTSD may develop following a traumatic experience such as war combat. In addition it has been noted that such trauma may lead to symptoms of psychosis. Childhood psychological abuse and sexual abuse have also been linked to psychosis. It may be the case that trauma predisposes to the development of psychosis and this can be compared to psychosis of neurodevelopmental origin (schizophrenia).

Most widely explored is psychosis following trauma alongside PTSD. This has been well documented following trauma in Wartime situations. PTSD groups of combat veterans were found to manifest a significant increase in psychotic symptoms over control groups (Butler 1996, Kozaric-Kovacic 2005). Not only have positive psychotic symptoms been recorded in PTSD but also negative symptoms (Hamner 2000). PTSD and psychosis following childhood related and other trauma have also been reported (Neria 2003). A review of data shows 30-40% of combat veterans with PTSD report auditory or visual hallucinations and/or delusions (Lindley 2000). Much debate surrounds the nature of the relationship between PTSD and psychosis. Morrison et al. (2003) discusses three main alternatives. The first is that psychosis may cause PTSD for which there is evidence that 46% of those recovering from an acute psychotic episode developed PTSD after 4 months (McGorry 1991). Secondly it is suggested that trauma may cause psychosis and lastly that PTSD and psychosis are part of a spectrum of disorders that can occur in response to trauma.

Other traumatic life events including childhood psychological and sexual abuse have been linked to the onset of future psychosis (Read 1997). A study found 70% of hallucinations to develop following a traumatic event (Romme & Escher 1989). Additionally, chronic auditory hallucinations were commonly preceded by an episode of trauma or an event that activated a memory of past trauma (Honig 1998).

Trauma also appears to predispose to subsequent 'borderline personality disorder' and depression. Co-morbidity is common in sufferers of PTSD with psychotic symptoms (Hamner 1999, David 1999, Ivezic 2000).

Of particular interest is the pattern of development of psychosis following trauma in comparison to that of psychosis of neurodevelopmental origin (schizophrenia). Brain functional and anatomical changes have been implicated in the development of schizophrenia. Similar abnormalities have also been observed in PTSD. The structure of the brain leading up to and following the onset of neurodevelopmental psychosis (schizophrenia) has been extensively studied using MRI. There has been particular focus on the hippocampus, which has been shown to be of reduced volume in first onset psychosis and chronic schizophrenia (Velakoulis 1999). It is likely that the hippocampus shrinks at some stage in the lead up or shortly after the onset of psychosis. However, this is uncertain and further longitudinal studies of hippocampal volume are taking place (Wood 2001, Velakoulis 2006). Other changes in medial temporal lobe structure (including the amygdala and anterior cingulate cortex) have also been proposed (Velakoulis 2006, Yucel 2003). It is thought that these structural changes superimpose upon previous (pre and peri-natal) neurodevelopmental insults leading to the onset of psychosis (Pantelis 2005, Velakoulis 2000).

We would like to draw attention to a pilot study which we carried out (Agius 2007, 2008), assessing how the development of symptoms of patients with PTSD and psychotic symptoms acquired during war compared with the development of symptoms in patients with PTSD and

psychotic symptoms acquired as a result of sexual abuse in young adulthood (as opposed to child sex abuse, when patients are often diagnosed as suffering from 'borderline personality disorder').

We studied five such cases, two with war related PTSD, three with Sex-abuse related PTSD.

In each of the cases, in both groups, the symptoms seemed to develop incrementally. Patients would develop PTSD, but no Psychotic Symptoms, after the first incident of exposure to trauma. Psychotic symptoms developed in each of the cases after exposure, some time later, to a second episode of trauma. This phenomenology seems to us to suggest that a Stress Diathesis model does indeed occur in patients with PTSD and psychotic symptoms, and that the first episode of PTSD confers a vulnerability in these patients which leads to development of psychotic symptoms when exposed to further stress. Clearly it will be necessary to test this hypothesis further by detailed history taking to study the phenomenology of the development of a large number of patients with psychotic symptoms and PTSD.

We would like to invite comment on our findings. We ask whether others have noted the incremental development, based on repeated trauma of PTSD with Psychotic symptoms which we have described, particularly in the War Trauma variant of this condition.

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Correspondence:

Mark Agius MD, Early Intervention Service
Charter House Alma Street Luton LU12PJ
E-mail: ma393@cam.ac.uk