Journal of Affective Disorders 183 (2015) 57–67

Contents lists available at ScienceDirect

Journal of Affective Disorders

journal homepage: www.elsevier.com/locate/jad

Review

Comparison of precipitating factors for mania and partial seizures: Indicative of shared pathophysiology?

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ARTICLE INFO

Article history:
Received 12 November 2014
Received in revised form
23 March 2015
Accepted 30 April 2015
Available online 11 May 2015

Keywords:
Bipolar disorder
Mania
Temporal lobe
Epilepsy
Partial seizures
Precipitating factor

ABSTRACT

Objectives: Mania in bipolar disorder (BD) and partial (focal) seizures (PS) arising from the temporal lobes, have a number of similarities. Typically, a chronic course of the disorders is punctuated by acute illness episodes. Common features of episodes may include sensory, perceptual, cognitive and affective changes. Both respond to anticonvulsant treatment. Common mechanisms imputed include neurotransmitters and kindling processes. Further investigation may improve understanding of the occurrence of both mania and PS, casting light on the relevance of temporal lobe mediated processes and pathology. One avenue of investigation is to compare aetiological factors and determine the extent of overlap which may indicate shared brain localization or pathophysiology. Aetiology includes predisposing, precipitating or perpetuating factors. This paper examines the literature on precipitating factors of mania, first or subsequent episode, and of PS in diagnosed epilepsy, which is the second or subsequent seizure, to identify the extent and nature of their overlap.
Method: Narrative review based on a literature search of PubMed and Google Scholar.
Results: Precipitating factors for both mania and PS were stress, sleep deprivation, antidepressant medication and, tentatively, emotion. For mania alone, goal-attainment events, spring and summer season, postpartum, and drugs include steroids and stimulants. For PS alone, winter season, menstruation and specific triggers in complex reflex epilepsies. Those not substantiated include lunar phase and menopause. A wide range of chemicals may provoke isolated seizures but by definition epilepsy requires at least two seizures.
Conclusions: The overlap of precipitating factors in mania and PS imply that common brain processes may contribute to both, consistent with findings from neuroscience research.

Contents

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http://dx.doi.org/10.1016/j.jad.2015.04.057
0165-0327/

1. Introduction

The precise pathogenesis of bipolar disorder (BD) is unknown, though it is generally agreed that its development is influenced by genetic and environmental factors (Craddock and Jones. 1999). Currently there are no biological markers available for a definitive diagnosis of BD, rather diagnosis is based on signs and symptoms. The cardinal feature of BD type I (BDI) is mania, characterized by "a distinct period of abnormally and persistently elevated, expansive, or irritable mood, lasting at least 1 week (or any duration if hospitalization is necessary)" (American Psychiatric Association, 2013). Episodes of mania may lead to marked impairments in social and occupational functioning and impose a significant financial burden on both patient and health care system. In order to alleviate this burden a more detailed understanding of the aetiology and pathogenesis of mania is important to direct development of improved treatments.

Aetiology is commonly classified in terms of predisposing, precipitating and perpetuating factors, all of which are highly relevant to understanding the onset and course of a disorder. This review considers one of these dimensions, precipitating factors, which consist of recurrences of episodes in established cases of illnesses distinct from the cause of an initial episode. Two reviews have focused on the precipitating factors of mania/ hypomania in young adults (Proudfoot et al., 2012) and adults (Proudfoot et al., 2011). The examination of precipitating factors that are temporally linked to illness episodes may inform research into underlying neurobiological mechanisms involved in the occurrence of episodes.

Further insights may be gained by comparing and contrasting precipitating factors between two disorders which share common features. Several studies have taken this approach in the fields of psychiatry and neurology. In the Iowa 500 study, the precipitating factors of schizophrenia and primary affective disorders (Clancy et al., 1973) were compared to assist in delineating the two syndromes. Precipitating factors have also been reviewed as a component of a comparison between migraine and epilepsy (Haut et al., 2006). Following this approach, a potential reference condition for comparison with mania is partial seizures arising from the temporal lobes. The term partial seizures (PS) includes both simple partial and complex partial seizures and is synonymous with focal seizures in the International League Against Epilepsy (ILAE) classification (Berg et al., 2010).

Both BD and epilepsy constitute striking manifestations of brain disorders. Speculation has included the extent to which these two superficially very different disorders share common aetiology, pathogenesis and treatment responses. The two disorders have long been of interest to the medical profession. In particular, the unusual behavioural disturbances rather than convulsions that may occur in epilepsy have been of interest since ancient times, reaching a peak in 19th century medical literature (Schmitz and Trimble, 1992). Renewed interest came with the advent of the electroencephalogram (EEG) and the discovery of forced normalization, a process which involves, in patients with epilepsy, the normalization of EEG recordings during psychotic states (Krishnamoorthy and Trimble, 1999). The then presumed beneficial relationship between epilepsy and psychosis resulted in the introduction of convulsive therapy as a treatment for psychosis (Sachdev, 1998).

Further, it was observed that the nature of the psychotic disturbance bore some relationship to the laterality of the epileptic focus. Flor-Henry (1969) proposed that manic-depressive and schizophrenia-like disturbances originated from the right and left temporal lobes respectively. With advances in neuroimaging, some degree of laterality of BD and schizophrenia has since been observed. Meta-analysis has shown that in patients with schizophrenia and bipolar disorder, compared to neurotypical control participants, gray matter volume is lower in the prefrontal cortex, thalamus, left caudate and medial temporal lobe and right insula. Schizophrenia has been associated with gray matter deficits in the left insula and amygdala (Yu et al., 2010). In epilepsy, PS typically originate from one medial temporal lobe, although they may be propagated from other structures which project to limbic areas (Travers, 1991). PS may be lesional or non-lesional and can be idiopathic or secondary, referring to the predisposing aetiology; all of these are included in this review which focuses on the precipitating factors for PS in established epilepsy.

Other similarities between BD and epilepsy include their episodic and often chronic clinical course, proposed involvement of kindling mechanisms and the observed efficacy of antiepileptic medications (Mula et al., 2010). In both disorders, kindling mechanisms may be involved in perpetuating illness episodes. In order to understand human limbic epilepsies, experimental kindling has been performed in rodents. This involves the observation of progressive changes that result from repeated electrical stimulation (Goddard et al., 1969). The premise that *seizures beget seizures* has long been postulated and in BD it has been hypothesised that stressors and relapses leave traces and cause vulnerabilities for future recurrences (Post, 1992). Mazza et al. (2007) provide a comprehensive explanation of the kindling paradigm with respect to BD and epilepsy.

The similarities between the two extend to their episodic manifestations. Mania and PS share psychopathological symptoms including sensory, perceptual, cognitive and affective changes (Silberman et al., 1985). Not only do the two disorders share common features, they can also co-occur. The prevalence ratio for diagnosed BD in epilepsy is 2.11% (95% CI 1.82–2.45) (Ottman et al., 2011). In a recent study in a sub-Saharan African population it was demonstrated that epilepsy occurs at a higher rate among first-degree relatives of patients with BD 15.2% than controls 2% and the rate of BD among those with epilepsy 14.5% compared with controls 2.1% (Jidda et al., 2014). In a population-based registry study, the proportion of BD among people with epilepsy was twice as high relative to individuals without epilepsy (Bakken et al., 2014). Mania is more common in patients with TLE than in the general population (Lyketsos et al., 1993) and symptoms of BD are often found in patients with epilepsy (Ettinger et al., 2005).