Manifestation of Bipolar Affective Disorder: Post Brain Injury

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Among the many debilitating psychiatric disorders, bipolar affective disorder, previously called manic-depressive illness, can be one of the most difficult to understand, and even more difficult to diagnose. To begin with, there are two ways in which the disorder can be broken down. According to the American Psychiatric Association (APA), the first type, known as bipolar I disorder, features "one or more Manic Episodes or Mixed Episodes," as well as incidences of at least one "Major Depressive Episode" (1994). They also refer, in detail, to each of these mood episodes. They define a *manic episode* as a "distinct period during which there is an abnormally and persistently elevated, expansive, or irritable mood" (APA, 1994). A *mixed episode* is described as a minimum of a week-long phase containing characteristics of both manic and major depressive episodes, almost daily. A *major depressive episode* lasts at least two weeks and consists of a "depressed mood or the loss of interest or pleasure in nearly all activities" (APA, 1994).

The second type, simply called bipolar II disorder, is also characterized by a major depressive episode, but is set apart by the fact that it includes a hypomanic episode, rather than a manic or mixed episode. A hypomanic episode has all of the mood characteristics of a manic episode, but lasts at least four days. The other major difference lies in the symptoms of the episode, as there are no delusions or hallucinations in a hypomanic episode. In the DSM-IV, it was specifically noted that the hypomanic episode is "not [to] be confused with the several days of euthymia that may follow remission of a major depressive episode" (APA, 1994).

There are multiple reasons why bipolar disorder, in its entirety, is such an important topic to be studied, not only by psychologists and psychiatrists, but by other medical professionals. First, those who are diagnosed and their families need to be able to understand, identify, and treat this illness properly. Bipolar individuals can suffer from psychomotor retardation or agitation, insomnia, grandiose delusions, uncontrollable impulsive and high-risk behaviors, and even suicidal thoughts (APA, 2000). Second, the completed suicide rate among persons with bipolar I and bipolar II disorder is between 10%-15%. Additionally, child abuse, spousal abuse, or other violent behavior can occur, and it is not uncommon for one to have problems with school truancy

and failure, occupancy failure, divorce, or episodic antisocial behavior (APA, 1994). Third, without extensive research, the disorder is completely free to continue taking over the lives of countless Americans, not to mention the world population. Edward Craighead reports that around 1.4% of the population is affected by this disorder (Bipolar Affective Disorder, 2004). By this estimate, over 4.25 million Americans are affected by bipolar disorder (U.S. Census Bureau, 2009).

There have been countless studies trying to prove, or disprove, whether bipolar disorder has any genetic and/or biological predispositions. One study, conducted by Gershon in 1990, which was described in the Corsini Encyclopedia (2004), reported that "20% of the first-degree relatives of Bipolar patients have major affective disorders" (Bipolar Affective Disorder, 2004). Also, the disorder in identical twins averages 57%, and only 14% between fraternal twins (Alda, 1997). Biologically, there are a few important findings that point to predisposition. For example, a theory currently gaining some acceptance states that a G-protein's activity affects the signal of neurotransmitters. In addition, when bipolar patients were compared to normal subjects, researchers found a larger amount of the G-protein in bipolar patients, even in a remitted state (euthymia) (Mitchell, Manji, Chen, Jolkovsky, Smith-Jackson, Denicoff, Schmidt, & Potter, 1997).

Another study, conducted by van der Schot and others in 2010, had extremely specific results, showing certain areas of the brains of bipolar patients having "subtle abnormalities," including less grey matter density than a normal brain, particularly in the frontal and limbic regions (van der Schot, Vonk, Brouwer, van Baal, Brans, van Haren, Schnack, Boomsma, Nolen, Pol, & Kahn 2010). Widespread grey matter abnormalities related directly to the illness itself. The results also showed less white matter density in the frontal parts of the superior longitudinal fasciculi, which are thought to be connected to the genetic risk of developing bipolar disorder. This causal relationship and the supporting data behind it are new to bipolar disorder pathology in the frontal lobe and 45% can be explained by common genetic factors (van der Schot et al., 2010). Furthermore, multiple reports stated that many of the (cognitive) symptoms experienced by patients with bipolar disorder seem to be associated with dysfunction of the frontal lobe (Drevets, Price, Simpson, Todd, Reich, Vannier, & Raichle, 1997; Philips, Ladouceur, & Drevets, 2008).

Bipolar I disorder manifests itself in many different ways, depending on the person's

genetic and biological make-up, but it seems to follow a general pattern. Typically, the illness is recurrent, meaning that 90% of diagnosed patients will have more than one manic episode in their lifetime. These manic episodes usually precede or immediately follow a major depressive episode in a continuous cycle (60%-70% of the time). If the patient experiences four or more episodes in less than a year, they are considered to be rapid-cycling in their diagnosis (APA, 1994). These are the basic guidelines for diagnosis and course of bipolar I disorder.

Bipolar II disorder follows a similar course by presenting itself through repeated episodes. Approximately 60%-70% of hypomanic episodes "occur immediately before or after a Major Depressive Episode" in bipolar II patients and do so in a particular cycle, influenced by many factors specific to each individual (APA, 1994).

Although genetic factors play an important role in the manifestation of bipolar disorder, the importance of environmental variables should not be overlooked (Savitz & Drevets, 2009). For example, unnatural changes in the brain's biology, like those associated with traumatic brain injury (TBI), can cause the illness to manifest itself. A TBI is formally defined by Silver, Hales & Yudofsky (2004), as "the result of mechanical forces on the skull and transmitted to the brain leading to focal and/or diffuse brain damage, as well as secondary effects." If an individual were to endure a head injury, and therefore suffer some brain damage, it is my hypothesis that since the physical make-up of the brain directly affects the probability or actualization of the illness, the injury to the brain could cause a direct manifestation of bipolar affective disorder, regardless of any genetic predisposition. In order to support this, I will describe three case studies in which it is argued that the direct manifestation of bipolar disorder took place after traumatic brain injury, which ranged from mild to severe.

The first study, recorded by Zwil, McAllister, Cohen & Halpern (1993), described the case of a 32-year-old man who, after suffering a mild closed-head injury resulting in diffuse brain injury, was diagnosed with rapid cycling bipolar disorder. The man, who remains anonymous, had "no previous history of affective disorder or central nervous system disease [and] was in his usual state of health" until the accident, which consisted of a 4 foot fall onto a metal scaffold support "striking the back of his head" (Zwil, et al., 1993). He reported no loss of consciousness, only dizziness, along with irritability and an ever-changing mood. Once at the hospital where he was treated, he experienced increasingly worsening symptoms such as insomnia, crying spells, agitation, increased irritability, suspicion, belligerence and even rage,

which were noted as being markedly different from his mood before the accident. One week after being discharged from said hospital, the man was again hospitalized at a psychiatric facility for an acute manic episode. A CT scan revealed nothing remarkable. Upon treatment, the patient's symptoms receded and he was therefore released a second time. Nearly three months after the injury, the man was admitted for severe depression and suicidal thoughts, provoking an expert evaluation. A single-photon emission computed tomography (SPECT) scan of his head showed decreased function in both frontal lobes as well as temporal abnormalities, which were noticeably worse on the left side. An electroencephalogram (EEG) showed unusual forms in the left hemisphere, including "sharp and slow waves" in the left temporal lobe (Zwil et al., 1993). The experts performed multiple neuropsychological tests and concluded that the man displayed signs of dysfunctionality in "anterior brain regions" (Zwil et al., 1993), which can be translated to the frontal lobes.

There are four major reasons why bipolar disorder could be considered secondary to the head injury. The first is that the manifestation of the first hypomanic episode took place only a week after the injury. Second, the man never indicated any signs of manic or depressed moods before the injury, and had no family history of the illness, or others like it. Third, an EEG and a SPECT scan clearly showed that the injury caused damage in the frontal and temporal regions. Lastly, the very conclusive and extensive neuropsychological testing demonstrated that the effects from the accident had diminished over roughly two years, but the mood cycles continued persistently, leading experts to conclude that the disorder was a direct result of the original head injury (Zwil et al., 1993). This case showed that even minor head trauma could be capable of causing bipolar disorder, specifically such a critical and ultra-rapid cycling form.

Another case, documented by Murai and Fujimoto (2003), was of a rapid cycling bipolar patient who was diagnosed after an isolated (focal) left-temporal-polar lesion due to a closed head injury, caused by a traffic accident. Subject KS was a 48-year-old woman with no prior history of neurological or psychiatric illness whatsoever. A computed tomography (CT) scan revealed a bruise, or cerebral contusion, on the left temporal polar base of the brain, which had resulted in a loss of consciousness for two days. Upon awakening, she was hypersomniac and was released in a calm state of recovery. However, only six weeks later, her family reported a restless, irritable and easily angered insomniac, who exhibited all of the easily identifiable symptoms of a manic episode. Three weeks later, she became hypoactive, staying in bed all day

and showing no interest in daily activities, as well as a loss of appetite – all symptoms of a major depressive episode. After almost a year of cycling approximately every 30 days, KS was examined by a psychiatrist and properly diagnosed as having a bipolar I disorder with rapid cycling. Upon further examination it was discovered via magnetic resonance imaging (MRI) that there was abnormal high signal intensity in the left temporal pole, indicating restricted blood flow to the area. It was noted that there were no other apparent brain injuries outside of the specified area.

Previous research has shown that temporal polar lesions play a major role in manic episodes (Jorge, Robinson, Starkstein, Arndt, Forrester, & Geisler, 1993), but can now be directly associated with rapid cycling bipolar disorder (Murai et al., 2003). The temporal pole is currently thought to control two important things and if damaged facilitates symptoms like the ones found in a bipolar patient. It has been revealed that this region is pivotal in processing emotional stimuli (Daugherty, Shin, Alpert, Pitman, Orr, Lasko, Macklin, Fischman, & Rauch, 1999; Blair, Morris, Frith, Perrett, & Dolan, 1999), as well as judging the pleasantness or unpleasantness of sensory stimuli (Royet, Zald, Versace, Costes, Lavenne, Koenig, & Gervais, 2000). Although it is highly unlikely that a temporal pole lesion is the single mechanism that causes bipolar disorder, it can be speculated that the temporal pole may play a key role in preventing exaggerated mood swings, like those found in bipolar disorder. This case is consistent with the notion that a TBI, which causes damage to a particular part of the brain, can lead to the manifestation of bipolar disorder. However, it was unclear whether or not KS carried a biological predisposition (Murai et al., 2003).

The third report, by Brissos & Dias (2005), tells of a 47-year-old man (JPF) with no personal or familial psychiatric history who developed bipolar disorder after a severe traumatic brain injury, sustained in a traffic accident. JPF's injury was characterized by "acute focal brain damage" and widespread "diffuse axonal injury." After waking from a 4-day coma, a CT scan revealed temporal and parietal damage and an EEG revealed abnormalities in the left fronto-temporal area of the brain. A year and a half later, he was referred to a psychiatric treatment center for exhibiting a series of behavioral symptoms including pressure of speech, aggressiveness, uninhibited behavior, and irritability (but without any signs of psychosis). Treatment and release followed, but euthymia was short-lived as another episode presented itself in the form of depression, sadness, apathy and suicidal thoughts only a month later. After

another week of medication, JPF was released. A month later he became dysphoric (depressed), with pressured speech, excessive spending, and psychotic symptoms (delusions). Only four weeks later he was again experiencing depression, and within 1 week was disinhibited and slightly euphoric (these symptoms are characteristics of a manic episode). The constant cycling of manic and depressive episodes eventually led to the diagnosis of "post-TBI bipolar disorder" (Brissos, et al., 2005).

It can be argued that because the symptoms of bipolar disorder in JPF were so delayed, the onset of the illness was not caused by the brain injury. However, because he was at an age where late onset bipolar disorder is quite unlikely, especially with no history of mental illness, it was carefully hypothesized that there was in fact a causal relationship (Brissos, et al., 2005). Also, the two researchers made a startling statement about the cause of such disorders:

> Mood disorders are more frequent in patients with TBIs than in patients with similar background characteristics who underwent similar levels of stress but without brain injury, which would suggest that neuropathological processes associated with TBI constitute an important contributing factor to the development of mood disorders [and] rates for post-TBI bipolar disorder have ranged from 1.7 to 17%.(Brissos, et al., 2005)

This case indicates that traumatic brain injury can cause vulnerability to psychiatric disorders.

As described above, the genetic predispositions of bipolar disorder are now thought to be paired with biological changes in the brain. Van der Schot, et al.'s study (2010) showed that in 49 twin pairs diagnosed with bipolar disorder, there were grey and white matter density anomalies in the frontal lobes of all the bipolar patients, which suggested that the changes were linked to the disorder. These differences were found in major parts of the brain, but mostly in the frontal lobes. Also, in all three case studies summarized above, the patients who suffered some form of brain injury were shown, through electroencephalograms and computed tomography scans, to have suffered frontal lobe injuries. Both genetically predisposed patients and TBI patients had similar brain abnormalities and were diagnosed with bipolar disorder, although the TBI patients had no indicated genetic predisposition. These three case studies, as well as pertinent information from the recent study regarding biological abnormalities in bipolar brains, suggest that men and women who have suffered some form of traumatic brain injury may be at risk of developing bipolar disorder, independently from any genetic predisposition and regardless of the injury's severity.

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