

Case Study: Bipolar Disorder

(Name)

(Institution)

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Description of the possible biological causes of mental illness

Genetic Etiology

It increases the risk of acquiring mental illness, particularly bipolar disorders. It is greatly associated with becoming a first degree relative of an individual with bipolar disorders (Pavuluri, Nadimpalli, O'Connor, & Sweeney, 2006).

Medical Illnesses

Cardiac diseases and seizure disorders have a great correlation with increased susceptibility of having bipolar disorder among adults. Viral infections among immunosuppressed individuals also contribute to bipolar diathesis (Paluvuri, et al., 2006).

Brain Trauma - Consequent brain affectation (specifically to the prefrontal cortex, limbic system, and paralimbic structures) following a head injury that results to mood disorders among adults (Paluvuri, 2006).

Gamma-amino-butyric-acid (GABA)

Ergic system, glutamatergic system, polyamine system also have great implications on the occurrence of psychiatric disorders (Fiori & Turecki, 2010).

Neurotransmitters

An imbalance or non-regulation of serotonin, dopamine, and norepinephrine causes symptoms of depression (Johnson, Joormann, LeMoult, & Miller, 2008).

Identification and brief discussion of the function of the neurotransmitters in depression

- The major neurotransmitters affected during episodes of depression are serotonin and catecholamines, particularly norepinephrine and dopamine.
- Different paradigms suggest that a deficiency in serotonin leads to mood disorders and bipolar disorders. Johnson et al. (2008) stated that serotonin plays a major role in the

biological pathway of mood disorders. This neurotransmitter hinders the automatic response of the body to emotional stimuli. If serotonin becomes deficient, cognition in response to external stimuli will shift and will lead to a negative emotional control.

- Dopamine also plays a critical role in depression, especially those who have Parkinson's disease (PD). Knowing that the dopaminergic system is responsible for the motivation, motor and reward functions, an imbalance in this system would likely result to psychomotor disturbances and lack of pleasure (anhedonia) that is commonly observed among depressive patients (Dishman, Washburn, & Heath, 2004).
- Norepinephrine, on the other hand, is found to be responsible for the effectiveness of antidepressants. Together with serotonin, an imbalance in norepinephrine is said to be responsible for the symptoms of sleep disturbances, loss of appetite, and weight loss (Lovallo, 2005).

Brief outline of the cause of Margaret's mania and brief description of other possible biological causes of mania

The cause of Margaret's mania is due to her excessive intake of fluoxetine. Selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine can cause a variety of abnormal behavioral and mental conditions.

- Several phenomena can be observed from the client such as a change from agitation to mania, and obsession that is unusual for an individual (Breggins, 2004).
- Aside from side effects of drugs, other biological causes of mania would include genetics, "extra cells in the brain," and traumatic brain injury (Johnson, 2008).
- Bipolar disorder is one of the heritable psychiatric disorders. This claim is proven by evidences from animal models, and the genetic complexity of the disorder. Some of

the genes associated with this disorder are BDNF, DAOA, DISC1, GRIK4, SLC6A4 and TPH2 (Barnett & Smoller, 2009).

- Extra cells in the brain may also be a cause. The University of Michigan (2005) conducted a study about the abnormality in the brain biochemistry of people with bipolar disorder. It has been found out that in people with bipolar disorders, an extra 30 percent of signal-sending cells is identifiable on the two major brain areas. Consequently, this causes an overstimulation which is accountable to symptoms produced in bipolar disorders (University of Michigan, 2005).
- Mania has been associated with traumatic brain injury. Damage to the basal region of right orbitofrontal cortex and right temporal lobe among patients with history of bipolar disorder predisposes an individual to mania (Silver, Hales, & Kupfer, 1997). Mood disorders also became common among people who experienced traumatic brain injuries, with an estimation of 4 percent for the occurrence of mania (Taylor & Jung, 1998).

Brief description of the DSM IV diagnosis of bipolar affective disorder: Would

Margaret meet the DSM IV diagnosis for this disorder?

- DSM IV describes an individual as having bipolar disorder if he/she has one or more history of major depressive episodes accompanied with at least one history of hypomania. He or she has an elevated or irritable mood, and symptoms experienced by the individual interfere with his or her occupational and social functioning (Nettina, Lippincott Williams, & Wilkins, 2006).
- As with the case of Margaret, she satisfies the criteria of DSM IV for bipolar disorders. Episodes of depression resulted to suicidal attempts leading to frequent hospitalization in the past. Recently, she was admitted to the hospital because of

manic episode. Her symptoms became disturbing to her family, which consequently impaired her social functioning.

Three different medications which may be used to treat Margaret as described in the case study above

- Lithium, Quetiapine, Olanzapine

Side effects for the medications and biological reasons for each

Lithium

Side Effect 1: Polyuria. An initial loss of sodium and potassium occurs after the administration of lithium. It is then followed by the retention of sodium and water. This increase in sodium concentration (hyponatremia) allows an increase in blood volume, resulting to an increase in blood flow to the kidneys. The patient then experiences frequent urination in large amounts, a condition called polyuria (Goldberg, 2009).

Side Effect 2: Muscle weakness and cramps. On the other hand, hyponatremia lowers the potassium concentration of the body (hypokalemia), allowing the patient to manifest muscular weakness and cramps (Shives, 2007).

Quetiapine

Side Effect 1: Sedation or Drowsiness. Inhibition of histamine H1 receptors is said to be one of the most potent action of quetiapine. As these receptors are inhibited, the patient experiences sedation or drowsiness (Schulz, Olson, & Kotlyar, 2006).

Side Effect 2: Orthostatic Hypotension. It also acts as an antagonist to the alpha-1 adrenergic receptors, resulting to smooth muscle relaxation. Affectation of the smooth muscles of the blood vessels would likely result to orthostatic hypotension (Jeste, Sable, & Salzman, 2005).

Valproic Acid.

Side Effect 1: Headache. This interacts with the sodium channel blockers causing an inhibition to the rapid bursting of sodium (Camfield & Camfield, 2009). An increase in the dosage of valproic acid will cause a decrease in the serum sodium resulting to hyponatremia and one symptom of hyponatremia is headache (Shives, 2007).

Side Effect 2: Diarrhea. When there is a decrease in serum sodium, the potassium level increases resulting to hyperkalemia. One symptom of hyperkalemia is diarrhea (Shives, 2007).

Brief discussion of current research relating to the biological considerations associated with suicide and affective disorders

Suicide is one of the common causes of death among psychiatric patients. Several studies are made regarding suicide and its neurobiology; however, it is still unclear what is its precise pathophysiology and mechanism.

Dwivedi, et al. (2005) then proved the role of neurotrophins in suicide. Neurotrophins is associated with the cellular proliferation and migration, as well as the phenotypic differentiation and maintenance in the central nervous system. With the use of enzyme-linked immunosorbent assay and human-specific antibodies, Dwivedi and his colleagues compared the neurotrophin levels on the prefrontal cortex of normal subjects and suicidal subjects. It has been found out that neurotrophin levels on suicidal brains are significantly lower compared to the normal subjects. However, this variation on the neurotrophin levels is not dependent on gender, age, or pH of the brain. This observation is crucial when dealing with suicidal behavior since the prefrontal cortex contributes to the regulation of mood and has already been implicated in affective disorders and suicide.

This study was further supported by the study conducted by Karege and colleagues (2005), 30 victims of suicide were used in this study and tests revealed a decrease in the level

of neurotrophins on these subjects. However, the other control group used in this study is those medicated with psychotropic medications. This group was also treated with the same test and results showed no significant decrease in the levels of neurotrophins. This then suggested that neurotrophins mediate the action of psychotropic drugs (Karege, et al., 2005).

Dwivedi, et al. (2009) conducted another study to determine whether receptors of neurotrophins among suicidal subjects are altered. One of these receptors is called tropomyosin receptor kinases (TrKs). To start the study, researchers collected brain tissue samples from the Maryland Brain Collection at Maryland Psychiatric Research Centre. Several tests were done and results showed that there is compromised activation of the TrK receptors. In addition, its expression is also repressed resulting to a decrease in the availability of neurotrophins.

Together with the previous study, it can be concluded that the neurotrophins play an important role in suicide (Dwivedi, 2009).

With the studies presented, it can now be implicated that neurotrophin has a role in suicide. However, a study was conducted to determine whether genes have a role in the regulation of neurotrophin activity in suicidal individuals.

Sarchiapone, et al. (2008) studied 170 depressed patients. History evaluation and genotyping (for brain derived neurotrophic factor (BDNF) Val66Met polymorphism) were conducted and results showed that depressed patients carrying the BDNF were at high risk for suicide. It was also reported that severe depression is a significant factor to suicidal attempts, and is contributed by history of physical and sexual abuse. In conclusion, the BDNF influences suicidal behaviors.

Iga, et al. (2007) supported this conclusion by a study conducted among Japanese regarding the association of Val66Met polymorphism and psychotic behaviours. Their study

reflected that BDNF Val66Met polymorphism has no correlation with the development of major depressive disorder but on the clinical features of depression.

Reference List

- Barnett, J.H. & Smoller, J.W. (2009). The genetics of bipolar disorder. *Neuroscience* 164(1), 331-343.
- Breggins, P.R. (2004). Suicidality, violence and mania caused by selective serotonin reuptake inhibitors (SSRIs): A review and analysis. *The International Journal of Risk & Safety in Medicine*, (16)1, 31-49.
- Camfield, P. & Camfield, C. (2009). Valproic Acid. In J.W. Wheless, J. Willmore & R.A. Brumback (Eds.). *Advanced Therapy in Epilepsy*. China: PMPH.
- Dishman, R.K., Washburn, R.A. & Heath, G. (2004). *Physical Activity Epidemiology*. USA: Human Kinetics.
- Dwivedi, Y., Rizavi, H.S., Zhang, H., Mondal, A.C., Roberts, R.C., Conley, R.R. & Pandey, G.N. (2009). Neurotrophin Receptor Activation and Expression in Human Postmortem Brain: Effect on Suicide. *Biol Psychiatry*, 65(4), 319-328.
- Dwivedi, Y., Mondal, A.C., Rizavi, H.S. & Conley, R.R. (2005). Suicide brain is associated with decreased expression of neurotrophins. *Biol Psychiatry*, 58, 315-324.
- Fiori, L.M. & Turecki, G. (2010). Gene expression profiling of suicide completers. *European Psychiatry* 25, 287-290.
- Goldberg, R. (2009). *Drugs across the Spectrum* (6th ed.). USA: Wadsworth Cengage Learning.
- Iga, J.I., Ueno, S.I., Yamauchi, K., Numata, S., Tayoshi-Shibuya, S., Kinouchi, S., ... Ohmori, T. (2007). The Val66Met polymorphism of the brain-derived neurotrophic factor gene is associated with psychotic feature and suicidal behavior in Japanese major depressive patients. *American Journal of Medical Genetics: Neuropsychiatric Genetics* 114B (8), 1003-1006.

- Jeste, D.V., Sable, J.A. & Salzman, C. (2005). Treatment of Late-Life Disordered Behavior, Agitation, and Psychosis. In C. Salzman (Ed.). *Clinical Geriatric Psychopharmacology* (4th ed.). USA: Lippincott Williams & Wilkins.
- Johnson, S.L, Joormann, J., LeMoult, J. & Miller, C. (2008). Mood Disorders: Biological Bases. In P.H. Blaney & T. Millon (Eds.). *Oxford Textbook of Psychopathology* (2nd ed.). New York: Oxford University Press, Inc.
- Karege, F., Vaudan, G., Schwald, M., Perroud, N. & La Harpe, R. (2005). Neurotrophin levels in postmortem brains of suicide victims and the effects of antemortem diagnosis and psychotropic drugs. *Molecular Brain Research*, 136(2), 29-37.
- Lovallo, W.R. (2005). *Stress & Health: Biological and Psychological Interactions* (2nd ed.). United Kingdom : Sage Publications, Ltd.
- Nettina, S.M. & Lippincott Williams & Wilkins. (2006). *Lippincott Manual of Nursing Practice* (8th ed.). USA: Lippincott Williams & Wilkins.
- Pae, C.U., Kim, T.S., Kim, J.J., Lee, S.J., Lee, C.U., Lee, C. & Paik, I.H. (2005). Long term treatment of adjunctive quetiapine for bipolar mania. *Progress in Neuro-Psychopharmacology & Biological Psychiatry* 29, 763-766.
- Paluvuri, M.M., Henry, D.B., Nadimpalli, S.S., O'Connor, M.M. & Sweeney, J.A. (2006). Biological Risk Factors in Pediatric Disorder. *BIOL PSYCHIATRY* (60), 936-941.
- Sarchaipone, M., Carli, V., Roy, A., Iacoviello, L., Cuomo, C., Latella, M.C., di Giannantonio, M., ... Janal, M.N. (2008). Association of Polymorphism (Val66Met) of Brain-Derived Neurotrophic Factor with Suicide Attempts in Depressed Patients. *Neuropsychobiology* 57, 139-145.
- Schulz, S.C., Olson, S. & Kotlyar, M. (2006). Olanzapine. In A.F. Schatzberg & C.B. Nemeroff (Eds.). *Essentials of Clinical Pharmacology* (2nd ed.). USA: American Psychiatric Publishing, Inc.

Shives, L.R. (2007). *Basic Concepts of Psychiatric-Mental Health Nursing* (7th ed.). USA: Lippincott Williams & Wilkins.

Silver, J.M., Hales, R.E. & Kupfer, D.J. (1997). Neuropsychiatric aspects of traumatic brain injury. In S.C. Yudofsky & R.E. Hales (Eds.). *The American Psychiatric Press Textbook of Neuropsychiatry* (3rd ed.). USA: American Psychiatry Press.

Taylor, C.A. & Jung, H.Y. (1998). Disorders of mood after traumatic brain injury. *Semin Clin Neuropsychiatry* 3, 224-231.

The University of Michigan. (2005). *Evidence of Brain Chemistry Abnormalities in Bipolar Disorder*. Retrieved from http://bipolar.about.com/cs/menu_science/a/press_umich0210_2.htm