

# Ischaemic stroke case study example

[Sociology](#), [Violence](#)



\n[[toc title="Table of Contents"](#)]\n

\n \t

1. [Part 1.](#) \n \t
2. [Part II.](#) \n \t
3. [References](#) \n

\n[/[toc](#)]\n \n

## **Part 1.**

The pathophysiological template of an ischemic stroke involving the dominant left hemisphere of the brain is usually caused by the arterial occlusion resulting from a thrombus that embolized from a more proximal anatomical structure in the body that is closer to the brain, such as the heart and lungs and arterial plaques. The thrombus may cause a significant occlusion to the major intracranial arteries like the middle cerebral artery which carries the blood flow to the frontal, parietal and temporal lobes. A left middle cerebral artery stroke will manifest a right hemiplegia and hemiparesis with the upper extremities more significantly affected than the lower extremities. (Stein, 2004). The manifestation of contralateral paralysis is explained by the anatomical cross section of the nervous system with the nerve fibers crossing the opposite side to allow one side of the brain hemisphere to control the opposite side of the body (Atchison and Durette, 2007). The left hemisphere is the dominant side of the brain that controls the motor and sensory function in the right side of the body and is responsible for language and speech. An ischaemic stroke is considered to be the most common type of stroke that accounts to 80% of all stroke incidence

(Moustafa and Baron, 2008). It occurs upon the occlusion of an artery that is located in the neck or in the brain, resulting to the deprivation of adequate circulation of the blood that supplies the brain with the proper nutrients, glucose and oxygen that is vital to its function (Silverman and Rymer, 2009). The brain is highly sensitive to ischemia which is a deprivation of blood supply in the brain that results to neuronal and cellular death called an ischemic brain injury. This involves a cascade of events that deplete the cellular energy necessary to keep the neuronal cells to function properly and continuous deprivation of blood flow in the brain will eventually result to neuronal death (Worp and Gijn, 2007). According to Maas and Safdeih (2009), there are different etiology why ischemic stroke occurs but its most common pathway involves an insufficient blood flow to perfuse the cerebral tissues. The extent of the interruption of the blood supply or ischemia to the left hemisphere defines the severity of the resulting disability and paralysis that will occur on the right side of the body. A left hemispheric stroke resulting from ischaemia in the middle cerebral artery thus will result in the paralysis and lack of awareness of the right side of the body, speech difficulty, cognitive dysfunction such as difficulty in understanding language and mood changes.

## **Part II.**

- Pathogenesis leading to the structural and functional changes resulting to Mr. Black's stroke.

An ischaemic stroke involves both structural and functional changes that affect the manifestations of symptoms seen in Mr. Black's condition. The occlusion involving the middle cerebral artery manifests the hallmark

symptoms of motor and sensory deficits that are more significant in the face and the arms (Marx, Hockberger, Walls and Adams, 2010). It can be pointed out that Mr. Black manifests a right facial drop and paralysis of the upper arm. The cellular response to ischemia plays an integral role in understanding the structural changes in the brain caused by an ischemic brain injury. Biochemical events occur when the arterial blood flow is occluded resulting to a severe focal hypoperfusion resulting to oxidative damage causing microvascular injuries that then initiates post ischemic inflammation (Lakhan, Kirchgessner and Hofer, 2009). The middle cerebral artery is a major artery in the body that supplies the frontal, parietal and temporal lobes. The frontal lobe controls the mental and cognitive process in the brain. Ischemia affecting the middle cerebral artery will affect the frontal lobe function which results in a slow mental process (Leskela, et al, 2003). Frontal lobe ischemia due to middle cerebral artery occlusion also results in hemiplegia and language impairment as seen in Mr. Black's condition. In ischemia involving the middle cerebral artery, the basal ganglia and the internal capsule is often spared or the least affected resulting to a milder pattern of motor and sensory deficits that are more severe in the upper extremities while sparing the lower extremities (Alway and Cole, 2009). This explains why Mr. Black manifests more severe motor deficit in the right arm than the right leg. The functional changes that manifest when the parietal lobe is involved include impaired spatial perception, hemineglect and sensory loss. When the temporal lobe is damaged owing to the infarction in the middle cerebral artery, Aminoff, Boller and Swaab (2009) point out that the clinical manifestation involves impaired visual perception, impaired

organization of verbal material, disturbance in the language function, selective attention to the auditory and visual input and a disturbance in the language comprehension. The pathophysiological changes occurring in the middle cerebral artery is likewise affected by a concurrent hypertensive condition of Mr. Black. Accordingly, he has a history of hypertension in the last 10 years which can contribute to the risks of ischemic stroke to occur involving the middle cerebral artery. Philipps (2013) points out that chronic hypertension aggravates the formation of plaques that caused the arteries to narrow down over time that occludes the circulation of the blood. This results in pathological changes along the arterial walls causing the arteries to narrow resulting in a deprivation of the adequate blood flow to the brain to support its structural function (Brust, 2004).

- The mode of action of Alteplase and Assasantin in ischaemic stroke.

The Alteplase and Assasantin are the pharmacologic therapy prescribed in managing the condition of Mr. Black. Alteplase is a medication that is prescribed for the prompt management of an acute ischemic stroke which has a pharmacokinetic action of reducing the disability that may result from an ischemic stroke. It is administered intravenously and works as a fibrin selective agent that binds to fibrin on the thrombus that occludes the blood flow that initiates an ischemic attack (Finkel, 2009). As it binds along the fibrin, it works to break the thrombus in order to prevent blood clot formation that impedes the circulation of the blood to the brain. It has the capability of lysing only to the fibrin in the thrombus without causing any degradation on the other healthy proteins. As it breaks down the clot or thrombus, it helps to reduce the incidence of more serious disability to occur. Alteplase reduces

further risks of cellular damage to the brain as it helps improve the blood flow along the major arteries to provide more adequate blood supply necessary for neuronal and cellular survival and to optimize clinical recovery from an ischemic stroke (Katz and Purcell, 2006). Assasantin on the other hand is a preventive drug that is administered in order to reduce the risks of having another ischaemic attack. It works to prevent the blood platelets from blocking the blood vessel in order to enhance the circulation of the blood to the brain (Kim, Caplan and Wong, 2008). It is prescribed for individuals who already have a stroke as a preventive pharmaceutical intervention that will reduce the risk of another ischaemic attack from happening.

## References

- Always, D. and Cole, J. (2009). *Stroke Essentials for Primary Care: A Practical Guide*. New York: Humana Press.
- Aminoff, M. J., Boller, F. and Swaab, D. E. (2009). *Stroke: Clinical manifestations and pathogenesis*. Netherlands: Elsevier.
- Atchison, B. and Dirette, D. K. (2007). *Conditions in Occupational Therapy: Effect on Occupational Performance*. Oxford: Lippincott Williams and Wilkins.
- Bougousslavsky, J. (2001). *Stroke Syndromes*. Cambridge: Press Syndicate of the University of Cambridge.
- Brust, J. C. (2004). *Neurological Aspects of Substance Abuse*. Philadelphia: Elsevier.
- Finkel, R. (2009). *Pharmacology*. Philadelphia: Lippincott Williams and Wilkins.
- Hale, J. B. and Fiorello, C. A. (2004). *School Neuropsychology: A Practitioner's Handbook*. New York: The Guilford Press.

- Katz, R. and Purcell, H. (2006). *Acute Coronary Syndromes*. London: Elsevier.
- Kim, J. S., Caplan, L. R. and Wong, K. S. (2008). *Intracranial Atherosclerosis*. Sussex: Blackwell Publishing.
- Lakhan, S. E., Kirchgessner, A. and Hofer, M. (2009). Inflammatory mechanisms in ischemic stroke: therapeutic approaches. *Journal of Translational Medicine*.
- Leskela, M. et al (2003). Executive functions and speed of mental processing in elderly patients with frontal or nonfrontal ischemic stroke. *European Journal of Neurology*, 6 (6): 653-661.
- Maas, M. B. and Safdeih, J. E. (2009). Ischemic Stroke: Pathophysiology and Principles of localization. *Hospital Physician. Neurology Board Review Manual*.
- Marx, J. A., Hockberger, R. S. Walls, R. M. and Adams, J. G. (2010). *Rosen's Emergency Medicine: Concepts and Clinical Practice*. New York: Elsevier Health Sciences.
- Moustafa, R. R. and Baron, J. C. (2008). Pathophysiology of ischaemic stroke: insights from imaging, and implications for therapy and drug discovery. *British Journal of Pharmacology*.
- Philipps, S. J. (2013). *Pathophysiology and Management of Hypertension in Acute Ischemic Stroke*. Texas: American Heart Association.
- Silverman, I. E. and Rymer, M. M. (2009). *An Atlas of Investigation and Treatment. Ischemic stroke*. Oxford: Atlas Medical Publishing.
- Stein, J. (2004). *Stroke and the Family: A New Guide*. USA: President and Fellows of Harvard College.

Worp, H. B. and Gijn, J. V. (2007). Acute Ischemic Stroke. The New England Journal of Medicine.