

Are there any correlations between embolic stroke and previous post-traumatic epilepsy- Study from a case report

by Viskasari Pintoko Kalanjati

Submission date: 22-Jan-2020 04:32PM (UTC+0800)

Submission ID: 1244850172

File name: d_previous_post-traumatic_epilepsy-_Study_from_a_case_report.pdf (166.22K)

Word count: 2777

Character count: 15291

PAPER • OPEN ACCESS

Are there any correlations between embolic stroke and previous post-traumatic epilepsy? – Study from a case report

5
To cite this article: W Santosa *et al* 2018 *IOP Conf. Ser.: Mater. Sci. Eng.* **434** 012326

View the [article online](#) for updates and enhancements.

Are there any correlations between embolic stroke and previous post-traumatic epilepsy? – Study from a case report

W Santosa^{1,4*}, V P Kalanjati² and A Machin³

¹ Master Study Program of Basic Medical Science, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia.

² Department of Anatomy and Histology, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia.

³ Department of Neurology, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia.

⁴ Department of Anatomy and Histology, Faculty of Medicine, Universitas Surabaya, Surabaya, Indonesia.

*winniesantosa@gmail.com

Abstract. Stroke is one of the world's highest causes of morbidity and mortality. In USA, 50-100 of 100.000 people/ year were suffered from the stroke. Post-traumatic epilepsy (PTE) is a form of epilepsy occurs after a traumatic brain injury. This injury arguably causing an embolic stroke due to the blood clot emboli of the brain artery after trauma; this might be worsened by underlying diseases including myocardial infarct. A-56 years old man experienced seizures and then weakness in the right side of the upper and lower limb and also right-side of the face, with decreased consciousness after having a seizure and fall to the floor because of it. The patient had right hemiparesis with a negative Babinski reflex; the patient also had a right central facial palsy, right central type lingual and motor aphasia. This patient has an old myocardial infarct (inferior and antero-septal) that yet been treated properly. Difficulties in talking and weakness of the right-side of the limbs and face might occur as a result of the emboli in the left side of the cerebral artery branches; with decreased consciousness was likely due to the increased intracranial pressure. The emboli may be resulted from the blood clot produced after the fall when the patient was having a seizure; and/ or from the emboli of the untreated old myocardial infarct. In this case, radiology imaging may confirm the diagnosis thus the treatment; in which disrupted cerebral blood flow is likely underpinning the neuropathology observed.

1. Introduction

Epilepsy is a manifestation of impaired brain function with a variety of etiologies, but with a singular characteristic, periodic attack caused by excessive and paroxysmal electrical charge of brain neurons. Electrical discharge disorders can be caused by various conditions that affect the metabolism of brain neurons. The clinical features of focal attack depend on the involved cortical region, so that various types of attack can be found, e.g. motor, sensory and partial complex [1].

Approximately 70% of unknown cases of epilepsy are classified as idiopathic epilepsy or genetic and 30% are known to be classified as symptomatic epilepsy, for example head trauma. [2] Patients



Content from this work may be used under the terms of the [Creative Commons Attribution 3.0 licence](https://creativecommons.org/licenses/by/3.0/). Any further distribution of this work must maintain attribution to the author(s) and the title of the work, journal citation and DOI.

with post-traumatic epilepsy (PTE) may suffer recurrent post-traumatic seizures (PTS) after initial injury. PTE is a life-long complication of traumatic brain injury (TBI) [3,4].

Brain ischemia is most common due to embolism. To reach the intracranial arteries, a variety of embolic particles arise from the heart, aorta, and cervicocranial arteries, and other substances such as air, fat, tumor cells, and foreign objects are also occasionally introduced into the vascular system and reach the brain and other organs. [5] Emboli that reach the intracranial artery has a strong likelihood of causing stroke and cerebral infarction [6].

In ASEAN countries, stroke is a major health problem that causes death. The stroke mortality rate from South East Asian Medical Information Center (SEAMIC) data is found to be the largest in Indonesia followed by the Philippines, Singapore, Brunei, Malaysia, and Thailand respectively. Of all stroke patients in Indonesia, the most common type of stroke is ischemic stroke of 52.9%, followed sequentially by intracerebral hemorrhage, embolism and subarachnoid hemorrhage with an incidence rate of 38.5%, 7.2%, and 1.4% [7].

Stroke is an acute development of focal neurologic deficit as a result of sudden diffuse or local cerebral hypoperfusion which can cause morbidity or mortality. [8] An embolic stroke occurs when a blood clot formed elsewhere in the body is released and moves to the brain through the bloodstream. [9,10] Ischemic stroke can be caused by several types of diseases. The most common problem is the narrowing of arteries in the neck or head. An ischemic stroke can occur if the artery to the brain becomes blocked. The brain relies on an artery nearby to carry blood from the heart and lungs. This blood flow allows oxygen and nutrients to reach the brain. If one of these arteries is blocked, the brain can not produce the energy it needs to function. These brain cells will begin to die if the blockage lasts more than a few minutes. This is most often caused by atherosclerosis, or gradual cholesterol deposition. If the arteries become too narrow, the blood cells can collect and form blood clots. This blood clot can block the arteries where it will form (thrombosis), or it can be deflected and trapped in the arteries closer to the brain (embolism). Another cause of stroke is blood clotting in the heart, which can occur as a result of irregular heartbeat (e.g., atrial fibrillation), heart attack, or abnormalities in the heart valve [5,11].

Stroke attacks may result in some sudden (partial or partial paralysis) suddenness, loss of speech, sight, or walking sensation, leading to death. [12,13] Treatment of stroke patients, especially new patients should be done quickly and precisely. Certainty determination of early stroke type pathology is essential for proper administration of drugs to prevent more fatal effects [14].

2. Case Report

A 56-year-old male came to the Emergency Department of RSUD Dr. Soetomo, Surabaya with PTE and difficulties in talking and had a right side hemiparesis of the face and upper and lower limbs since 15 hours before the admission. After these, the patient was unconscious thus unable to feed. There was previous TBI due to traffic accident several months before the PTE. Vital signs: the score of Glasgow Coma Scale (GCS): 4x4 (eye 4, verbal motor aphasia-like, motoric 4), blood pressure 125/80 mmHg, pulse 91 times/ minute, respiratory rate 20 times/ minute, temperature 36°C. On physical examination, patient observed to develop upper motor neuron type lesion of the right-side of the face and the tongue. Thorax and abdomen within normal range. Both pupils were normal, no pathology reflex were found. Patient was also diagnosed with inferior and anteroseptal old myocardial infarct (OMI). Focal brain atrophy at the right cerebral hemisphere with occipital lobe gliosis and right cerebellar hemisphere encephalomalacia cyst of the previous CT scan were reported.

3. Discussion

In this case the initial symptoms are difficulties in talking and weakness of the right-side body. Difficulties in talking can be caused by the embolus to the central left- side of the cerebral artery branches of the brain arising suddenly resulting in aphasia and right hemiplegia and loss of hemisensory. The middle cerebral artery is the largest of the three cerebral arteries and its cortical territory is the most extensive. It passes laterally from its origin to enter the lateral fissure within

which it subdivides, its branches supplying virtually the whole of the lateral surface of the frontal, parietal and temporal lobes. This territory includes the primary motor and sensory cortices for the whole of the body, excluding the lower limb. It also serves the auditory cortex and the insula within the depths of the lateral fissure. [15] As it passes through the embolus and lenticulostrual artery supplying the internal capsule and the area of the disseminated basal ganglia, the hemiparesis may improve. Increased cortical blood flow may lead to better language function. If there is further deterioration after initial repair in patients with embolism, the deterioration usually occurs in one step and almost always occurs during the first 48 hours [16].

20 Seizures that occur in patients can be caused by blood clots. Emboli can block any artery depending on the size and nature of the embolic material. Blood clots that cause embolic stroke can form anywhere, usually coming from the heart or arteries in the upper chest and neck. Once released, blood clots flow through the bloodstream and might end up in the brain. When it enters the blood vessels that are too small in the diameter, the lump becomes stuck. Insufficient blood flow in the arteries in the brain can be compensated by the collateral system, especially between the carotid artery and the vertebrae with the anastomose in the circle of Willis and between the large arteries of cerebral hemispheres [17,18].

On cardiac examination found Old Infarction Myocardium (OMI) inferior and anteroseptal which means heart disease caused by coronary artery blockage. Obstruction occurs because of the atherosclerotic on the coronary artery wall, thus blocking blood flow to the heart muscle tissue. Atherosclerosis is a disease of large and medium arteries where a fat lesion called atheromatous plaque arises on the inner surface of the artery wall. It narrows and even blocks the supply of blood flow to the distal artery. The cause of OMI is due to atherosclerosis or total or partial blockage by embolism and/ or thrombus [19,20].

The patient has a history of the disease first epilepsy post trauma. Head injury may cause variety of disorders in the brain both in its structure and function including PTE [5]. Seizures can occur quickly (<24 hours of incident) or later (in the first weeks). A seizure that occurs in the immediate aftermath of a head injury is a risk factor for post traumatic epilepsy. Epilepsy from head trauma is symptomatic epilepsy, where the attack can be partial / focal or general attack or mixed form. Epilepsy is a condition that can make a person recurrent seizures. In the human brain there are neurons or nerve cells that are part of the nervous system. Each nerve cell communicates with each other using electrical impulses. In the case of epilepsy, seizures occur when the electrical impulses are over-produced resulting in uncontrolled behavior or body movements. The delay in the onset of the first seizure carries a higher risk of epilepsy. In patients with ischemic stroke, approximately 35% of epilepsy patients appear in rapid onset seizures and in 90% of patients with slow onset seizures. The risk of epilepsy is comparable to that of hemorrhagic stroke patients, about 29% of patients with epilepsy appear on fast-onset seizures while 93% with slow onset seizures [21].

Embolus blocks important distal branches, leading to further ischemia and worsening of symptoms. The embolic stroke most often starts suddenly. Clinical signs develop for several seconds or minutes. Symptoms may begin during physical activity but are more common at rest or daily activities [5,22].

The blood circulation in the brain can be divided into two major parts: the anterior circulation of the brain that supplies most of the subcortical white cortex and subcortex, the basal ganglia, and the internal capsule, comprising the internal carotid artery and its branches, the anterior choroidal artery, the cerebral artery anterior, cerebral artery media. The cerebral artery of the media provides a branch of the lenticulostrual artery. Stroke caused by anterior circulation disturbances will provide symptoms and signs of apraxia, apraxia, agnosia, hemiparesis, hemisensori and visual defects. The second posterior circulation of the brain supplies the brainstem, cerebellum, thalamus and also part of the occipital and temporal lobes. This circulation consists of: a pair of vertebral arteries, basilar artery and branches of the inferior posterior cerebellary artery, the anterior inferior cerebellary artery, the superior cerebellar artery, and the posterior cerebral artery. Stroke caused by a disturbance of the posterior circulation will provide symptoms and signs of brainstem dysfunction, including coma, vertigo, nausea and vomiting, cranial nerve palsy, ataxia and sensorimotor deficits that affect the face

on one side of the body and limb on the other. Hemiparesis, hemisensory and vision field deficits also occur, but are not specific to stroke caused by impaired posterior circulation [23-25].

4. Conclusion

In this case, patient was suspected with embolic stroke due to the PTE correlated with the previous TBI and OMI. [5] Further examinations are needed to make a definitive diagnosis including head magnetic resonance imaging (MRI), electroencephalogram (EEG) and angiogram with contrast. In this case, there is highly likely positive correlation between the embolic stroke and previous PTE.

References

- [1] Fisher RS, van Emde BW, Blume W, et al 2005 Epileptic seizures and epilepsy: definitions proposed by the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE) *Epilepsia*, **46**:470-472
- [2] Hauser WA, Annegers JF, Kurland LT 1991 Prevalence of epilepsy in Rochester, Minnesota: 1940-1980. *Epilepsia*, **32**:429-445
- [3] Boovalingam P, Witherall R, HO CL, Nagarajan R, Ardron M 2012 Post-stroke epilepsy UK: *GM Journal*
- [4] Frey LC 2003 Epidemiology of post traumatic epilepsy: a critical review. *Epilepsia*. **44**(Suppl 10):11-17
- [5] Caplan LR 2006 *Embolic particles*. In Caplan LR, Manning W (eds); *Brain Embolism*. New York: Informa Healthcare, pp 259-275
- [6] Molina C, Alexandrov A 2006 *Transcranial Doppler Ultrasound*. In Caplan LR, Manning W (eds); *Brain Embolism*. New York: Informa Healthcare, pp 113-128
- [7] Permatasari, Dwita, 2011. *Kejadian Hiperkolesterolemia Disertai Hipertensi dan Diabetes Mellitus pada Penderita Stroke Trombotik Akut*. Bulletin Penelitian RSUD Dr Soetomo, 13(3), 112-120.
- [8] Drake RL, Vogl AW, Mitchell AWM 2014 *Gray's Anatomy for Student*. Singapore: Elsevier ltd
- [9] Guyton, Arthur C, John EH, 2007. *Textbook of Medical Physiology* edisi 11. Terjemahan; Dian Ramadhani; Fara Indriyani; Frans Dany; Imam Nuryanto; Srie Sisca Prima Rianti; Titiek Resmisari; Joko Suryono. 2008. Buku Ajar Fisiologi Kedokteran edisi 11. Jakarta: EGC
- [10] Hananta, I Putu Yuda, Harry FLM, 2011. *Deteksi Dini dan Pencegahan Hipertensi dan Stroke*. Yogyakarta: Media Pressindo.
- [11] Babikian VL, Caplan LR 2000 Brain embolism is a dynamic process with variable characteristics. *Neurology* **54**:797-801
- [12] Caplan LR 1993 *Brain embolism, revisited*. *Neurology* **43**:1281-1287
- [13] Caplan LR 1999 *Brain embolism*. In LR Caplan, JW Hurst, M Chimowitz (eds), *Clinical Neurocardiology*. New York: Marcel Dekker, pp 35-185
- [14] Bahrudin M 2009 Diagnosa Stroke. *Jurnal Sainika Medika* Vol.5 No.11 Universitas Muhammadiyah, Malang
- [15] Crossman A R and Neary D 2015 *Neuroanatomy: an illustrated colour text* 5th ed (New York: Churchill Livingstone Elsevier)
- [16] Mohr JP, Gautier JC, Hier DB, Stein RW 1986 Middle cerebral artery, In Barnett HJM, Mohr JP, Stein BM, Yatsu FM (eds): *Stroke, Pathophysiology, Diagnosis, and Management*, vol 1. New York: *Churchill Livingstone*, pp 377-450
- [17] Japardi I, 2002. *Patogenesis Stroke Iskemik Tromboemboli*. Universitas Sumatera Utara.
- [18] Bladin CF, Alexandrov AV, Bellavance A, Bornstein N, Chambers B, Coté R 2000 Seizures after stroke: A prospective multicenter study. *Arch Neurol*; **57**:1617-22
- [19] Caplan LR 1996 *Posterior Circulation Disease, Clinical Findings, Diagnosis, and Management*. Boston: Blackwell.

- [20] Sliwka U, Job F-P, Wissuwa D, et al 1995 Occurrence of transcranial Doppler high-intensity transient signals in patients with potential cardiac sources of embolism, a prospective study. *Stroke* **26**:2067–2070
- [21] Stroke Association 2012 Epilepsy after stroke. London: Stroke Association.
- [22] Markus HS 1993 Transcranial Doppler detection of circulating cerebral emboli, a review. *Stroke* **24**:1246–1250
- [23] Daffertshofer M, Ries S, Schminke U, Hennerici M 1996 High-intensity transient signals in patients with cerebral ischemia. *Stroke* **27**:1844–1849
- [24] Sliwka U, Lingnau A, Stohmann W-D, et al 1997 Prevalence and time course of microembolic signals in patients with acute strokes, a prospective study. *Stroke* **28**:358–363
- [25] Chen JW, Ruff RL, Eavey R, Wasterlain CG 2009 Post traumatic epilepsy and treatment. *J Rehabil Res Dev*. **46**(6):685-696

Are there any correlations between embolic stroke and previous post-traumatic epilepsy- Study from a case report

ORIGINALITY REPORT

23%

SIMILARITY INDEX

16%

INTERNET SOURCES

16%

PUBLICATIONS

0%

STUDENT PAPERS

PRIMARY SOURCES

1

kyutech.repo.nii.ac.jp

Internet Source

3%

2

diseasedetail.com

Internet Source

2%

3

cco.cup.cam.ac.uk

Internet Source

2%

4

scitepress.org

Internet Source

2%

5

V Yudha, H S B Rochardjo, J Jamasri, R Widyorini, F Yudhanto, S Darmanto. "Isolation of cellulose from salacca midrib fibers by chemical treatments", IOP Conference Series: Materials Science and Engineering, 2018

Publication

2%

6

almagia.com

Internet Source

2%

7

Isaac E. Silverman, Lucas Restrepo, Gregory C. Mathews. "Poststroke Seizures", Archives of

2%

Neurology, 2002

Publication

-
- | | | |
|----|--|-----|
| 8 | Caplan, . "Brain Embolism", Clinical Neurocardiology Fundamentals and Clinical Cardiology, 1999.
Publication | 1% |
| 9 | Rohit Khurana. "Carotid Artery Stenosis Prevalence and Medical Therapy", Carotid Artery Stenting The Basics, 2009
Publication | 1% |
| 10 | "Carotid Artery Stenting: The Basics", Springer Science and Business Media LLC, 2009
Publication | 1% |
| 11 | pt.scribd.com
Internet Source | 1% |
| 12 | Thrombolytic Therapy in Acute Ischemic Stroke II, 1993.
Publication | 1% |
| 13 | "Cerebral Cortex", Springer Science and Business Media LLC, 1999
Publication | 1% |
| 14 | www.ncbi.nlm.nih.gov
Internet Source | 1% |
| 15 | perpusnwu.web.id
Internet Source | <1% |
-

16

docslide.us

Internet Source

<1%

17

Neil MacLennan, Arthur M. Lam. "Intraoperative neurological monitoring with transcranial doppler ultrasonography", Seminars in Anesthesia, Perioperative Medicine and Pain, 1997

Publication

<1%

18

"Pan Vascular Medicine", Springer Nature, 2002

Publication

<1%

19

Llinas, R.. "Neurologic complications of cardiac surgery", Progress in Cardiovascular Diseases, 200010

Publication

<1%

20

L CAPLAN. "Brain Embolism", Neurological Disorders, 2003

Publication

<1%

21

Handbook of the Cerebellum and Cerebellar Disorders, 2013.

Publication

<1%

22

"Atherosclerosis Disease Management", Springer Nature, 2011

Publication

<1%

Exclude bibliography On

Are there any correlations between embolic stroke and previous post-traumatic epilepsy- Study from a case report

GRADEMARK REPORT

FINAL GRADE

/100

GENERAL COMMENTS

Instructor

PAGE 1

PAGE 2

PAGE 3

PAGE 4

PAGE 5

PAGE 6
