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CASE REPORT **OPEN ACCESS**

A CASE REPORT: PREVOST'S AS EARLY SIGN OF SUBCORTICAL SYMPTOM TO BE CONSIDERED FOR SCREENING IN ACUTE ISCHEMIC STROKE

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ABSTRACT

Background: Stroke is the leading cause of death and disability. Stroke symptoms depend on the location of the lesion, whether cortical or subcortical (white matter, basal ganglia, or thalamus). Prevost's sign or horizontal conjugate eye deviation (CED) must be considered an early motoric sign of ischemic stroke.
Case: Mrs. F, 61 years old, came to the ER complaining of headache and dizziness for a day without any complaints about her eyes, and had a history of diabetes mellitus. Neurological examination revealed conjugate eye deviation to the right side without other neurological deficits. A head CT scan showed a minimal infarction of the left putamen and an old minimal infarction of the right corona radiata.
Discussion: Pathophysiologic mechanism of Prevost's sign due to cerebral-mesencephalic-phonto-cerebellar pathway disorder and related to cranial nerves III and VI. The putamen is a nuclear structure part of the basal ganglia involved in that pathway. A provost's sign that appears in less than 90 days can be used for stroke screening for ischemic patients. Previous studies describe CED >140 as being identified as ischemic stroke. Patients with strabismus have an average of CED 14.20. Another retrospective study in 543 acute ischemic strokes showed that CED had a higher specificity value (0.78; 95% CI) than hemiparesis (0.35; 95% CI). The sensitivity values were 0.76 and 0.85 (95% CI).
Conclusion: Prevost's sign is one of the signs of motor deficit in acute cerebral infarction. It needs to be considered as a subcortical lesion in stroke patients.

Keywords: conjugate eye deviation, ischemic stroke, Prevost's sign, stroke

Introduction

According to the WHO, stroke is one of the clinical signs of vascular problems in the brain, which are focal or global deficits of function and occur suddenly. In Indonesia, stroke has been the leading cause of death and disability since 2013, with the incidence increasing every year. A clot or blockage causes ischemic stroke in an artery leading to the brain that has previously undergone an atherosclerotic process.[1-3]

Stroke has many risk factors. Generally, there are two risk factors: modifiable and non-modifiable. Modifiable factors include hypertension, smoking, Diabetes Mellitus (DM), cardiac abnormalities, dyslipidemia, physical exercise, obesity, alcohol, drug abuse, oral contraceptives, sleep disorders, lipoproteins, and homocysteine.[4]

In ischemic stroke, brain cell death can occur in about 1.9 million patients if medication is delayed for a few hours.[5-6] Stroke screening tool with BE FAST symptoms (Balance, Eye, Face, Arm, Speech, Time to Hospital) has been introduced to clinicians and the general public since 2017.[7-9] However, the knowledge of the screening has not been considered, especially dizziness symptoms (Balance) and visual disturbances such as blurred vision or double vision (Eye) which can be suspected of stroke.[10] Meanwhile, the means of

FAST is facial muscle paralysis (Face), paralysis of the extremities (Arm), speech disorders, or slurred speech (Speech), and is immediately taken to a health facility (Time to Hospital).[11]

Clinical symptoms in stroke vary depending on the location of the lesion, which can occur in the cortical or subcortical (white matter, basal ganglia, or thalamus).[12] Infarct lesions may present a variety of clinical symptoms depending on the vascular occlusion.[13-15] The most common infarction is in the middle cerebral artery (MCA), and the least common is in the anterior cerebral artery (ACA), which is <2% of all strokes worldwide.[16]

Superficial infarction of the MCA has speech/language impairment symptoms and facial asymmetry.[17] In contrast, infarction of the large MCA has more varied clinical symptoms such as eye deviation, contralateral hemiplegia, global aphasia, hemianopia, and loss of consciousness.[18] Infarctions from the MCA's anterior and superior branches show dominant symptoms such as hemiparesis, paresthesia, and eye deviation.[19-22] Approximately 90% of patients with conjugate eye deviation are caused by the internal carotid artery and MCA.[23]

A Study from Chengdu stroke from January 2010 until November 2020 described that the most common location of acute subcortical microinfarction was in the basal ganglia (47,8%), followed by the thalamus (21,7%), centrum semiovale/CSO (17,4%), and brainstem (13%). It also described that the most common neurological deficit was hemiparalysis, followed by central facial/lingual palsy, hemidysesthesia, dysarthria, dizzy/vertigo, aphasia, ataxia, dysphagia, conscious disturbance, and eye movement disorder, respectively.[24] Reinforced by a study by Yuya Kobayashi et al. in 2022, the infarction extended to the corona radiata aggravates the worst outcomes.[25]

However, several studies have shown that horizontal conjugate eye deviation, performed for neurological manifestations or measurement of deviation on head CT-Scan >140, predicts patients at high risk for ischemic infarcts in the cerebral cortex or subcortical structure. Participants with an eye deviation have right cerebral hemisphere infarction (63,8%) and left cerebral hemisphere infarction (43,8%).[26–28] Prevost's signs are symptoms of conjugate eye deviation that occur at early onset, starting an hour before, and can be used as a diagnostic symptom of ischemic stroke.[29]

Another case study in Saudi Arabia explains that a man, 62 years old, presented with a sudden onset of left-sided body weakness and showed conjugate horizontal left gaze palsy. A head CT scan showed an area of infarction over the right subcortical corona radiata region.[30] Therefore, in this study, the author aims to describe an isolated Prevost's sign as a subcortical symptom of ischemic stroke. It is part of the early sign-on screening for patients with suspected ischemic stroke.

Case Report

Mrs. F, 61 years old, came to the emergency room of Queen Latifa General Hospital (RSU QL) with complaints of severe headache and dizziness since the day before. It was getting worse, and I had fallen at home. The patient's family took her to the clinic, where her blood pressure was checked at 200 mmHg. The doctor advised her to go to the emergency room of a hospital.

While in the emergency room, the family realized that the patient's gaze tended towards the right corner. The patient had no complaints of weakness in his four limbs. The patient denied having black dot-like vision, curtain-like vision, speech impairment, difficulty swallowing, spraying vomit, loss of consciousness, fever, and seizure.

The patient claimed a previous history of type 2 diabetes mellitus/DM and had been taking metformin 1x500 mg for a year. There was no family history of the disease. The patient admitted that he likes to eat oily food and does not exercise regularly, and a history of alcohol consumption was denied.

Physical examination found composites of consciousness. Vital signs examination found blood pressure 205/105 mmHg, temperature 36.0 °C, pulse 82 x/min and regular, respiration 20 x/min, and SpO2 99% on room air.

On examination of neurological status, conjugate eye deviation was found towards the right. Then, during the examination of eye movements, the patient's eyeball tends not to move to the left when instructed to look at the left side. So, the patient's NIHSS (National Institutes of Health Stroke Scale) score is 1. A visual examination of the right eye and left eye was obtained on 6/18 and 6/18. There was no narrowing of the visual field and diplopia in both eyes. Then, a follow-up examination with an ophthalmoscope was obtained. The Cupping Disk (CD) ratio in the right and left eyes was 0.3: 0.3. In the examination, any abnormalities of meningeal stimulation, sensory function, motor system, and physiological and pathological reflexes were noted.

Hematological examination showed hemoglobin 13.7 g/dL, leukocytes 10,560 /µL, erythrocytes 4.45 million / mm3, hematocrit 39%, platelets 286,000/µL,

segments 80.7%, lymphocytes 17%, monocytes 2.2%. Blood chemistries showed an increase in glucose of 288.09 mg/dL, total cholesterol of 207.75 mg/dL, and LDL (low-density lipoprotein) of 139.6 mg/dL.

On a non-contrast CT scan of the head, which refers to a diagnostic imaging procedure known as Computed Tomography (CT) that utilizes X-rays to create detailed cross-sectional images of the brain, there were observations indicating the presence of a minimal infarction in the left putamen, a part of the brain involved in movement control. Additionally, there was evidence of a minimal, previously occurred infarction within the right corona radiata, a region of white matter in the brain responsible for transmitting signals between the cortex and other parts of the central nervous system. These findings were in Figure 1.

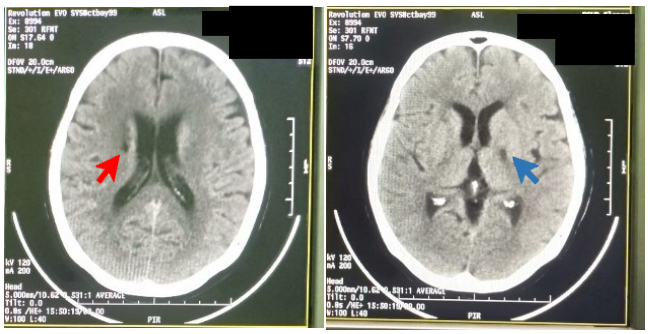

Figure 1. Non-contrast axial head CT scan, with a well-defined hypodense lesion in the corona radiata dextran (red arrow). Minimal hypodense lesion in the putamen sinistra, indistinct border (blue arrow)

The patient received management per established procedural standards and was subsequently admitted to QL Hospital for further care. Throughout the hospitalization, the patient continued exhibiting conjugate eye deviation symptoms. However, she did not report experiencing any headaches or dizziness during her stay. Additionally, her comorbid conditions, including hypertension and hyperglycemia, were effectively managed and remained under control throughout her treatment.

Discussion

Stroke screening with FAST symptoms has been introduced to the public and clinicians in the emergency department, showing 14% of undetected cases. In 2017, symptom screening was added to BE FAST for balance disorders and visual impairment, and undetected cases decreased to 4.4%.[31-33]

This case report provides a detailed account of a 61-year-old female patient, Mrs. F, who presented with a clinical manifestation characterized by a horizontal conjugate deviation of the eyes, specifically directed toward the right side. A head computed tomography (CT) scan was performed, revealing an infarction localized in the subcortical structures of the brain, with particular involvement of the left putamen and the right corona radiata. This case study, based on several previous studies, mentions that conjugate eye deviation or Prevost's sign can be a sign of early symptoms of ischemic stroke (Table 1).

In line with research conducted by Ilana Spokoyny in 2016, Conjugate Eye Deviation/CED is considered a sign of stroke patients and proven on CT-Scan to indicate ischemic stroke. CED also correlates with the NIHSS gaze score.[17] NIHSS is a tool to measure stroke severity based on symptoms found quantitatively.[34]

A study from Kaditis Dimitrios G. et al. in 2016 stated that Prevost's sign or conjugate eye deviation might be a symptom of ischemic stroke occurring in less than 1 or 3 hours. Some other studies have also shown that Clinical symptoms of conjugate eye deviation with suspected brainstem TIA should be followed up after 48–72 hours. A study by Ilana Spokoyny et al. in 2016 also shows that patients with Prevost's sign without any complaint of their eyes for 90 days before, and also present an angle of CED > 140 by CT scan, can be predicted to have ischemic stroke.[6,7,17,35]

This particular patient presents with a conjugate deviation of the eyes towards the right side, which indicates an abnormality in the movement of both eyes in the same direction. Following this, a head CT scan was performed without using contrast material, which revealed a minimal, somewhat indistinct hypodense lesion in the left putamen. A clearly defined hypodense lesion was also observed in the right corona radiata. The imaging findings suggest a recent, small infarction affecting the left putamen and an older, minor infarction in the right corona radiata.

Conjugate eye movements are divided into horizontal (eyes glance to the right and left) and vertical (eyes glance up and down). People with abnormalities in eye movement complain of blurred vision and dizziness. The four categories of conjugate eye movements are saccadic, pursuit, vestibular-ocular reflex (VOR), and optokinetic nystagmus (OKN).

The most common pathway disorders in conjugate eye movements are saccadic and pursuit. The saccadic functions are for rapid or spontaneous movement.[36] The process of horizontal conjugate gaze eye (described in Figure 2-3) begins with impulses triggered from the FEF (Frontal Eye Field) in both brain hemispheres, especially Brodmann area 8.[37–42]

Table 1. Some studies were shown Prevost's sign or conjugate eye deviation as a symptom of ischemic stroke[6,7,26–28]

No	Characteristic of population	Location of brain infarction	Neuroimaging	Result	Reference
1	One hundred nineteen patients with neurological manifestations underwent two imaging studies (brain NECT followed by CT with contrast or MRI). Thirty-six subjects (mean age 63.2 ± 17.2 years; 55.6% female) were excluded. Thus, data from 73 subjects were analyzed (64.2 ± 20.8 years; 49.3% female). The median interval (interquartile range) between the two examinations was 56 (22-109.3)	• 32 patients with ischemic brain injury, 19 (59.4%) had a stroke of the cortex and adjacent white matter, 6 (18.8%) had subcortical lesions (basal ganglia, thalamus, Reil' s isle, brainstem, and cerebellum). The cerebral cortex and subcortical structures were affected in 7 (21.9%) cases • 15 subjects (46.9%) had a right cerebral stroke, and 17 (53.1%) patients had a left cerebral stroke	Brain Non-enhanced Computed Tomography (NECT), CT-Scan with intravenous contrast and Magnetic Resonance Imaging-MRI	Measurement of horizontal ocular gaze deviation (OGD) > 14^0 on an initial brain NECT performed for neurological manifestations is an early predictor of ischemic brain injury (OR = 10,5; 95% CI 3,33 – 33,9)	Kaditis, D et al. (2016). Conjugate Eye Deviation As Predictor Of Acute Cortical and Subcortical Ischemic Brain Lesions
2	1.275 patients with eye deviation from the Albumin in Acute Stroke (ALIAS) trial. The admission CT examination was performed 1.57±1.28 hours after the symptom onset. On admission, in neuroimaging, 331 (27,1%) patients had leftward, and 352 (28.8%) had rightward eye deviation	Participants with an eye deviation have right cerebral hemisphere infarction (63,8%) and left cerebral hemisphere infarction (43,8%)	Head CT-Scan	Patients with eye deviation on CT had higher admission NIHSS scores and more significant middle cerebral artery (MCA) territory infarct volume (p-value <0.001)	Payabvash, S et al. (2016). Clinical Implications of Eye Deviation on Admission CT Examination of Acute Ischaemic Stroke Patients
3	76 years old Japanese woman with a history of hypertension and dyslipidemia developed a sudden onset of vertigo, left posterior neck pain, and hemiparesis in the right upper and lower extremities	An ischemic lesion in the left upper medial medullary	Brain MRI	This study represents a rare case showing contralateral conjugate eye deviation (CED) and right-sided hemiparesis. CED gradually improved and disappeared within 2 weeks after admission	Ogawa, T et al. (2018). Conjugate Eye Deviation Caused by Upper Medial Medullary Infarction: A Case Report

No	Characteristic of population	Location of brain infarction	Neuroimaging	Result	Reference
4	157 patients with horizontal conjugate eye deviation were performed in 1) 50 patients [age 58±15 years (mean±SD), 74% male, NIHSS 2±1] 2) 54 patients with transient brainstem symptoms [transient ischemic attack of brainstem (TIA) group; age 69±16 years, 59% male] 3) 53 patients (age 59±20 years, 49% male) with diagnoses other than stroke (control group)	Lateral medullary infarction • An initial CT scan was obtained for 43 patients (35 within 1 day after symptom onset, five on days 1–5, and 3 on days 5–10) • An initial MRI was performed on the other seven patients (5 within- 1 day and two within 5 days	CT/MRI	Conjugate eye deviation in patients with suspected acute lateral medullary infarction is a helpful and sensitive sign that supports the diagnosis. The extent of eye deviation was significantly greater in the infarction group ($p<0.05$), mainly if the deviation is > 20^0	Teufel, J et al. (2019). Conjugate Eye Deviation in Unilateral Lateral Medullary Infarction
5	A 62-year-old right-handed male with a longstanding history of diabetes mellitus and hypertension presented with sudden-onset left-sided body weakness as well as a loss of feeling over the left side of his body. These symptoms started approximately three days before	An area of infarction over the right subcortical corona radiata region.	Head CT-Scan	This study reports a man – 62 years old – who presented sudden onset left-side body weakness and showed conjugate horizontal left gaze palsy. A head CT scan showed an area of infarction over the right subcortical corona radiata region.	Alshahrani, Abdulrahman M. (2021). Horizontal Gaze Defect As A Result of Subcortical Stroke: Case Report and Review Of The Literature

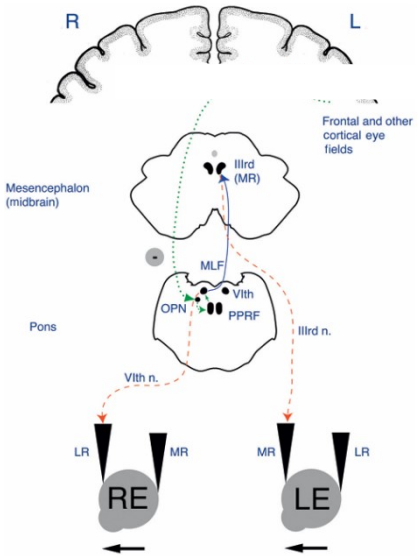

Figure 2. Saccadic pathway in eye movement
Suppose there is an impulse of eye movement to the right. In that case, impulses are formed in the contralateral FEF (left Broadman 8 area) and forwarded to the ipsilateral pons (right) precisely at the paramedian pontine reticular formation (PPRF), then continued to the abducens cranial nerve / Nn. VI right to move the right eyeball towards the right through contraction of the rectus lateralis muscle. In addition, the impulse is also continued to the midbrain, precisely the medial longitudinal fasciculus (MLF), for the coordination of the left eyeball movement so that it is rhythmic. Impulses from the MLF in the mesencephalon/midbrain are transmitted to the left oculomotor nerve / Nn. III left to move the medial rectus muscle of the left eyeball so that it can see to the right[38]

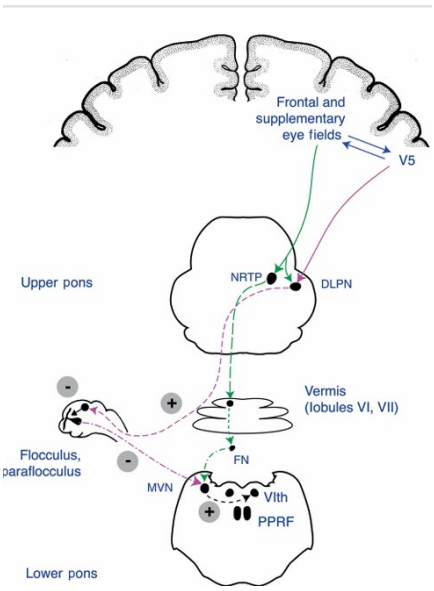

Figure 3. Pursuit pathway in eye movement
For the pursuit pathway, it is used for commands to follow the movement of an object. However, the difference in the initial impulse is triggered by the POT or Parietal Occipital Temporal[38]

As for some studies, horizontal eye movement comes from activities of the cortical pathway in the frontal, occipital, parietal, and temporal lobes, which are directly related to the subcortical structures of the thalamus, hippocampus, midbrain, brainstem, and cerebellum called the cerebral-mesencephalic-ponto-cerebellar pathway.[39] The putamen is a nuclear structure and part of the basal ganglia. The basal ganglia are considered part of the motor system. The structures are located in the subcortical substantia alba of the telencephalon.[40-42]

The pons and corona radiata structures are located between the cortex and brainstem and are associated with the subcortical motor pathways.[43-45] The basal ganglia, internal capsule, striatum, corona radiata, and thalamus are vascularized by the small perforator artery, which includes the lenticulostriate artery. Blockages at these locations are commonly caused by the LACI (Lacunar Circulation Infarction) type.[46-49] Acute subcortical cerebral infarction is a nonspecific and frequent clinical symptom of small vessel disease named LACI type stroke. Typical symptoms include hemiparesis, central weakness of the facial nerve, or lateral weakness of the tongue muscles. The most common locations for subcortical infarcts are the basal ganglia, thalamus, and centrum semiovale.[50]

The vascularization of the human brain is divided into two sources of large arterial branches. The internal carotid artery vascularizes the anterior brain, and the vertebrobasilar artery vascularizes the posterior brain. The internal carotid artery branches into the anterior cerebral artery (ACA), the middle cerebral artery (MCA, the anterior choroidal artery, the anterior communicating artery, and the posterior cerebral artery (PCA). The horizontal MCA (M1) branches into the lenticulostriate artery, which supplies nutrients to the inner brain structures, especially the basal ganglia.[51-52]

In another study, Dimitrios G. et al. in 2016 examined 119 patients with neurological manifestations that indicated stroke and underwent non-contrast head CT scans and MRI (Magnetic Resonance Imaging) of the head, which showed CED with a shift angle of > 140 in association with brain ischemia up to 10 times. Almost all showed the symptoms ipsilateral to the location of ischemic lesions.[53] The cerebral hemisphere lesions, either infarction or hemorrhage, show right-sided CED symptoms associated with the internal capsule or ipsilateral frontoparietal subcortical region.[53-54]

Lena-Alexandra Baume, in 2018, conducted a retrospective study on 543 patients with acute stroke, in which 153 patients showed symptoms of hemiparesis and 137 patients showed symptoms of CED. It means people with cortical symptoms (aphasia

or conjugate eye deviation) can be used as an indicator of ischemic stroke requiring thrombectomy therapy with a sensitivity of 0.91 and specificity of 0.70. Compared to motor deficits, extremities showed a sensitivity of 0.85 and specificity of 0.53.[55] Another study showed that patients with strabismus symptoms showed an average eye deviation of 14.2^0 compared to those without.[56]

In this case, the patient had a risk of stroke and a history of diabetes mellitus in the past year. The patient's blood sugar was found to be 288.09 mg/dL. In addition, blood pressure showed hypertension, 205/105 mmHg. The lipid profile showed an increase in total cholesterol of 207.75 mg/dL and an increase in LDL of 139.6 mg/dL.

A history of DM can increase the risk of stroke up to 2-6 times. It is related to complex problems, from molecular disorders of insulin function to the tendency to make dietary changes and adopt a sedentary lifestyle. Chronic conditions of DM and AGEs (advanced glycation end products) can increase the formation of atherosclerosis due to endothelial cell damage, which then causes infarction, stroke, angina pectoris, and myocardial infarction. Hyperglycemia causes vascular damage, as described in Figure 4. It triggers increased oxidative stress, activation of protein kinase C, and increased receptors for advanced glycation end products (RAGE).[57]

The activation of protein kinase C disrupts the function of nitric oxide (NO), which is a vasodilator. Hyperglycemia also inhibits the enzyme eNOS (endothelial nitric oxide synthase), which reduces NO. The decrease in NO impact of increased endothelin-1 (ET-1) and activation of the renin-angiotensin-aldosterone system is called angiotensin II (AT II). Then, it causes vasoconstriction, which increases hypertension and vascular smooth muscle cell (VSMC) growth.[58]

In addition, oxidative stress conditions lead to increased expression of the nuclear factor kappa-light-chain-enhancer of activated B cells (NF-kB) gene, which increases other inflammatory mediators, such as the cytokine interleukin-1 (IL-1). It can promote atherogenesis combined with vasoconstriction on a blood vessel. Hyperglycemia also causes an increase in the concentration of plasminogen activator inhibitor type 1 (PAI-1), which causes impaired fibrinolysis and hypercoagulation. It also increases the process of atherosclerosis.[59] Hyperglycemia in stroke patients increases the possibility of expanding the infarction area. It can risk bleeding, increased white matter damage, impaired blood-brain barrier (BBB) function, impaired function or tissue reperfusion process, and increased inflammatory response in the brain. It also causes an increase in stroke morbidity and mortality. White matter damage can occur because it is more sensitive to ischemia than gray matter. Ischemia causes white matter damage due to shortening of axons and damage to the myelin sheath, as well as decreased proliferation of progenitor cells from oligodendrocytes and inhibited myelin sheath repair.[60]

This case study shows that Prevost's sign can be considered an early sign of ischemic stroke, which is consistent with several studies. However, this case study has a limitation in that it can't illustrate the angle of deviation of the Prevost's sign by head CT scan. So, there is a need for further studies.

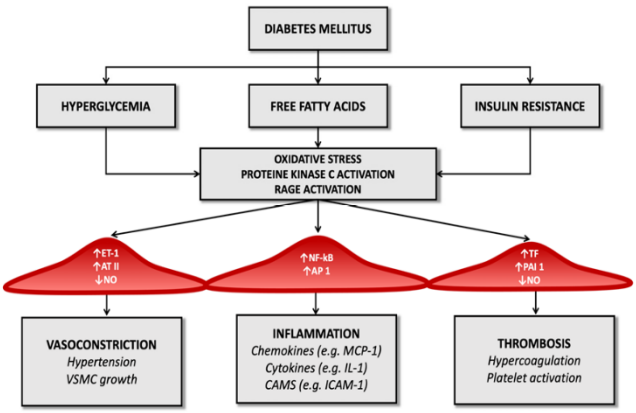

Figure 4. Pathophysiological process of hyperglycemia that causes vascular damage[60]

Conclusion

Prevost's sign is one of the signs of motor deficit in subcortical lesions, showing conjugate eye deviation. Therefore, Prevost's sign can be used as an early sign of an ischemic stroke.

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