Prevention of Strokes Caused By Sickle Cell Anemia, Ischemia or Hemorrhage

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Abstract:

Purposes: To propose models of prevention of strokes caused by sickle cell anemia, ischemia or hemorrhage as well as models of blood (cells) velocities in arteries (or capillaries) with different cross sections.

Methods: Published hematologic data and hemodynamics theories are applied or referenced.

Modeling Results: When there are sickle cells, emboli (or thrombi) in or external obesity tissues around an artery (or capillary), the action and reaction forces between the blood vessel wall and the external fats increase. The forces reduce the compliance (or elasticity) of the vessel. The sickle cells, fats, emboli or thrombi narrow the artery (or capillary); increase the pressure and resistance within the vessel; block the blood (cells) flowing; cause anemia or ischemia or hemorrhage strokes; or raise the flowing velocities. The raised velocities increase risks of hemorrhage strokes. A hemorrhage stroke is modeled as an artery with hematoma or hypertension and too little external fatty tissues. The models explain why or how fats increase and decrease probabilities of anemic, ischemic and hemorrhage strokes respectively.

Conclusions: As long as we completely and moderately ingest nutrients daily as well as maintain our mental and physical health, we can prevent most strokes caused by sickle cell anemia, ischemia or hemorrhage. The smaller the cross section of a blood vessel, the faster the velocity, for the blood (cells) flowing.

Keywords: strokes, sickle cell, anemia, ischemia, hemorrhage, emboli, thrombi, obesity, hematoma

Abbreviations: CVR - Cerebrovascular accident, RBC - Red blood cells, SCA - Sickle cell anemia, TCD - Transcranial Doppler, TMA - Thrombotic microangiopathy, ICH - Intracerebral hemorrhage, BMI - Body mass index, VTE - Venous thromboembolism, DVT - Deep vein thrombosis, PE - Pulmonary embolism

1. INTRODUCTION

Neurons are very sensitive to oxygen deprivation and will start to deteriorate within 1 or 2 minutes, and permanent damage (cell death) could result within a few hours. The loss of blood flow to part of the brain is known as a stroke, or a cerebrovascular accident (CVA) [1-2]. Sickle cell anemia is a genetic disorder. A characteristic change of erythrocytes (red blood cells, RBC) is a sickle shape. It is caused by production of an abnormal type of hemoglobin, called hemoglobin S, which delivers less oxygen to tissues and causes Figure 2. Model for hemoglobin thickness. erythrocytes to assume a sickle (or crescent) shape, especially at low oxygen concentrations. These abnormally shaped cells can then become lodged in narrow capillaries because they are unable to fold in on themselves to squeeze through, blocking blood flow to tissues and lead a stroke: a sickle cell anemic stroke. [1-2]. Cerebrovascular disease is an important complication in children with this condition. In sickle cell anemia, the peak incidence of overt stroke is 1.02 per 100 patient years between the ages of two and five years [3-4]. Most cases of stroke in children with sickle cell anemia are associated with narrowing of large intracranial arteries. Transcranial Doppler (TCD) scanning demonstrated that high blood velocities in the middle cerebral artery (MCA) were strongly associated with increased risk of stroke [4-5]. Additionally, thrombotic microangiopathy (TMA) syndromes are characterized by endothelial dysfunction, microangiopathic hemolytic anemia, and microvascular ischemia, with diverse etiologies that include drugs [6]. Anischemic strokes the loss of blood flow to an area because vessels are blocked or narrowed. This is often caused by an embolus, which may be a blood clot, fat deposit [1-2, 7-10], a thrombus [4-5], thrombocytopenia [11] or an atherosclerotic plaque [12]. A hemorrhagic stroke is bleeding into the brain because of a damaged blood
vessel. Accumulated blood fills a region of the cranial vault and presses against the tissue in the brain. Physical pressure on the brain can cause the loss of function, as well as the squeezing of local arteries resulting in compromised blood flow beyond the site of the hemorrhage [1-2]. A pooled analysis of 97 prospective cohorts with 1·8 million participants elucidate: obesity increases risks of strokes [11]. This analysis report is consistent with other research results [1-2, 7-10, 14-15]; and obesity has been found to be an independent risk factor for stroke [8].

Two critical reviews reported: within an individual, obesity may co-occur with anemia, due to shared underlying determinants or physiologic links. The co-existence of under-and over nutrition, a phenomenon known as the “dual burden”, poses a novel public health challenge and a threat to children’s health in low- and middle-income countries [16-17]. Hemorrhagic stroke is a severe stroke subtype with high rates of morbidity and mortality [18]. The majority of non-traumatic intracerebral hemorrhages (ICHs, primary ICH) originate from the spontaneous rupture of small vessels damaged by chronic hypertension or amylod angiopathy. ICH is well-known for its high overall stroke mortality and disability. It carries a 1-year fatality of >50% [19]. Obesity was associated with an increased risk of ischemic stroke and a decreased risk of hemorrhagic stroke (heterogeneity p < 0.0001) [20]. Based on above published data, I consider a comprehensive study and propose my models of prevention of strokes caused by sickle cell anemia, ischemia or hemorrhages as well as models of blood (cells) velocities in arteries (or capillaries) with different cross sections, in this article.

2. METHODS

Published hematologic data and hemadynamics theories [21-22] are applied or referenced. Obesity is defined by the body mass index (BMI), which is a measure of an individual’s weight-to-height ratio. The normal, or healthy, BMI range is between 18 and 24.9 kg/m². Overweight is defined as a BMI of 25 to 29.9 kg/m², and obesity is considered to be a BMI greater than 30 kg/m² [1].

3. MODELING RESULTS

Compliance (or elasticity) allows an artery (or capillary) to expand when blood is going through it, and then to recoil after the surge has passed. This helps promote blood flow [1]. When there are sickle cells, emboli (or thrombi) in or external obesity tissues around an artery (or capillary), the action and reaction forces between the blood vessel wall and the external fats increase. The forces reduce the compliance (or elasticity) of the vessel. The sickle cells, fats, emboli or thrombi narrow the artery (or capillary); increase the pressure and resistance within the vessel; block the blood (cells) flowing; cause anemia or ischemia strokes (Figure 1 and 2); or raise the flowing velocities (Figure 3).

When there are sickle cells, emboli (or thrombi) in or external obesity tissues around an artery (or capillary), the action and reaction forces between the blood vessel wall and the external fats increase. The forces reduce the compliance (or elasticity) of the vessel. The sickle cells, fats, emboli or thrombi narrow the artery (or capillary); increase the pressure and resistance within the vessel; block the blood (cells) flowing; cause anemia or ischemia strokes (Figure 1 and 2); or raise the flowing velocities (Figure 3).

Figure 1. Strokes, normal RBC, sickle cells, external fats and arteries (or capillaries): (a) normal RBC are going through an artery with external normal fatty tissues; (b) sickle cells are blocked in an artery with emboli; (c) sickle cells are blocked in an artery with external obesity tissues. See the text. The draw is not to the scale.

Figure 2. Strokes, normal RBC, sickle cells, emboli (or thrombi) and arteries (or capillaries) with external obesity tissues: (a) normal RBC are blocked in an artery; (b) normal RBC are blocked in an artery with emboli; (c) sickle cells are blocked in an artery with emboli. See the text. The draw is not to the scale.
Figure 1 illustrates my models of strokes, normal RBC, sickle cells, and arteries (or capillaries) with external fats: (a) There is not any block when normal RBC are going through the artery with external normal fatty tissues; (b) when sickle cells are blocked in the artery with external normal fatty tissues, a stroke caused by an anemia or ischemia probably occurs; (c) when sickle cells are blocked in the artery with external obesity tissues, a stroke caused by an anemia or ischemia occurs more probably.

Figure 2 illustrates my models of strokes, normal RBC, sickle cells, fats, emboli (or thrombi) and arteries (or capillaries) with external obesity tissues: (a) when normal RBC are blocked in the artery, a stroke caused by an ischemia probably occurs; (b) when normal RBC are blocked in the artery with emboli, a stroke caused by an ischemia occurs more probably; (c) when sickle cells are blocked in the artery with emboli, a stroke caused by an anemia or ischemia occurs most probably. Here, the external fatty tissues play a role to increase the probability of anemia or ischemia strokes.

Figure 3 illustrates my models of blood (cells) velocities, normal RBC, sickle cells, fats, emboli (or thrombi) and arteries (or capillaries) with external obesity tissues: (a) when normal RBC are going through the artery, the flowing velocities increase if the blood vessel is narrowed; (b) when normal RBC are going through the artery with emboli, the flowing velocities increase more if the blood vessel is narrowed more; (c) when sickle cells are going through the artery with emboli, the flowing velocities increase most, if the blood vessel is narrowed most. Therefore, the smaller the cross section of a blood vessel, the faster the velocity, for the blood (cells) flowing. The raised velocities increase risks of hemorrhage strokes, especially in curved blood vessels, based on hemodynamics theories [21-22].

Figure 4 illustrates my models of strokes, normal RBC, arteries (or capillaries) with external fatty tissues and hematoma or hypertension: when normal RBC are going through an artery [18] and too little external fatty tissues (a), a stroke caused by a hemorrhage probably occurs more often compared to that with external normal fatty (b) and obesity (c) tissues [20]. Here, the external fatty tissues protect blood vessels from rupture or burst. Therefore, the fats play a role to decrease the probability of a hemorrhage stroke.

Published data showed, the emboli (or thrombi) and obesity strongly correlate the strokes [7-14]; and emboli and obesity are mostly caused by over intake of high energy nutrients [1-2]. Therefore, as long as we completely and moderately ingest nutrients daily as well as maintain our mental and physical health (not too slim and nor too fat) [23-24], we can prevent most strokes caused by sickle cell anemia, ischemia or hemorrhage.
4. DISCUSSIONS
For the co-occurring disease of obesity and anemia, we must remain committed to reducing under nutrition while simultaneously preventing over nutrition, through integrated health programs that incorporate prevention of infection, diet quality, and physical activity. [16-17].

Although my models in this paper focus on preventing a stroke or a cerebrovascular accident (CVA), I believe the principles in this study are also helpful to prevent other diseases, such as myocardial infarction, or an atherosclerotic plaque (atherosclerosis); venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE) [25-26].

5. CONCLUSIONS
As long as we completely and moderately ingest nutrients daily as well as maintain our mental and physical health, we can prevent most strokes caused by sickle cell anemia, ischemia or hemorrhage. The smaller the cross section of a blood vessel, the faster the velocity, for the blood (cells) flowing.

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