

Research Article

Effect of Blood Viscosity Variation on the Flow of Blood in an Artery Having Time Dependent Stenosis

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Abstract: The aim of this paper is to examine the impact of blood viscosity variation on the flow of blood in a diseased artery having time-dependent stenosis. The viscosity of blood is axial co-ordinate dependent so that the viscosity of the blood increases up to the highest point of stenosis in the whole artery after which it decreases. Analytical methods have been used to explore the problem. The equations of volumetric rate of flow, resistance to flow, wall shear stress, and axial velocity have been obtained. It is noticed that as stenosis height increases, the resistance to flow and the wall shear stress increases. Also, investigation has been done to investigate how the wall shear stress and flow resistance vary with different time-related parameters and varying viscosity index values.

Keywords: volumetric flow rate, blood flow, wall shear stress, time dependent stenosis

MSC: 92B05, 92C10

1. Introduction

Stenosis is a chronic illness in which calcium deposits in the lumen cause the artery walls to thicken, stiffen, and lose their pliability, impairing blood flow. A blood vessel or other tubular organ or structure, such as foramina and canals, may experience this aberrant constriction. Aging, hypertension, diabetes, hyperlipidemia, and other vascular diseases all contribute to its development. The factors that cause stenosis are high cholesterol, smoking, high blood pressure, and some ailments, like obesity, etc. The vessel walls get harder and their lumen becomes smaller due to stenosis, which reduces blood flow to different areas of the body. A stroke may result from stenosis in the blood arteries that supply the brain or heart. The first thickening of the stenotic artery was thought to be the first stage of atherosclerosis, an illness of the arteries characterized by the deposition of adipose tissue on their interior walls.

Here, we have reviewed some of the important work done regarding the stenosis on the blood flow. A considerable number of experiments and several research works have been conducted to understand the flow of dynamics and stresses in an elastic collapsible tube. Young [1] presented the study of an axially symmetric, time-dependent growth into the lumen of a constant cross section tube through which a Newtonian fluid was flowing slowly. Possible biological ramifications were examined along with the effect of growth on pressure distribution and wall shearing stress. Kobayashi [2] did a numerical simulation to examine the hemodynamic in moderate stenosis with consideration of pulsatile wall motion. A mathematical model for the flow of blood in confined arteries with modest stenosis was investigated by Jain

et al. [3]. The wall shear stress of blood flow in stenosis arteries was studied by Saleh and Khan [4], and it was found that shear stress rises as the length and height of the stenosis grows. Considering that the flow of blood is represented by Newtonian fluid, Srivastava et al. [5] investigated the impact of overlapping stenosis on blood flow parameters. Axial flow and the size of the constriction are successful in holding the shear stress in a stenosed arterial segment, according to research by Nanda and Bose [6] who worked on a mathematical model for the circulation of blood through a small artery with numerous arteries. The impact of hematocrit on wall shear stress for the flow of blood in tapered arteries was investigated by Singh and Singh [7]. Jain and Singh [8] present a mathematical model that considers the viscosity of blood in the peripheral layer as constant and radial variation of viscosity in the core region to analyze how these factors, along with the size of stenosis, influence blood flow and shear forces in an artery. The findings suggest that decreasing peripheral layer viscosity can reduce resistance to flow, while larger stenosis increases peripheral resistance and wall shear. Wall shear stress has been found to directly correlate with the height of the stenosis. The literature on arterial catheterization with and without stenosis has been briefly reviewed by Srivastava et al. [9], who have also looked at the mathematical model of the flow of blood through a composite stenosis in an intubated artery with permeable wall. The impact of bell-shaped stenosis on the pressure drops and shear stress in an artery was demonstrated by Jain and Singh [10]. The analysis shows that as height and length of stenosis increases, not only pressure drop increases but shear stress also increases. The results are compared with the usual Newtonian fluid model. A model to study the effects of accumulation of red cells caused by obstruction due to stenosis in an artery on resistance to flow and shear stress was proposed and analyzed by Jain et. al. [11]. The analysis shows that as viscosity increases, not only resistance to flow increases but shear stress also increases. Owasit and Sriyab [12] took vertical symmetric stenosis and asymmetric stenosis compared the flow of quantities and investigated that the flow quantities in the single shape (bell shape or cosine shape) have the same behavior as the flow quantities in the combined shape in the first half part, but have slightly different behavior in the last half part. Carvalho et al. [13] have made attempts in computational techniques to simulate realistic conditions of blood flow in both diseased and healthy arteries, and their main aim was to give an overview of the most recent numerical studies focused on coronary arteries, by conveying the blood viscosity models, and adding physiological flow conditions. This review focused on coronary arteries by dealing with blood viscosity. Yi et al. [14] have investigated the effect of rough surface on the flow in arterial stenosis and it was found that the flow was oscillatory downstream of the stenosis. Blood flowing via an inclination pipe with stricture and expansion after stricture (widening) underneath the influence of a constant incompressible Casson liquid flowing with the magnetic field was investigated by Dhange et al. [15].

None of the above studies considered the effect of variation of viscosity in the case of time dependent stenosis. Here, we have considered the same in order to help biomedical sciences for the treatment of a diseased artery.

2. Formulation of the model

2.1 Development of the model

Here, a cosine-shaped stenosis is taken, and the radius of that stenosis changes depending on the time parameter T . The arterial geometry is shown in Figure 1. This problem is first approximated by assuming that the flow is stable and laminar. Furthermore, an asymmetrical development in the stenosis is assumed.

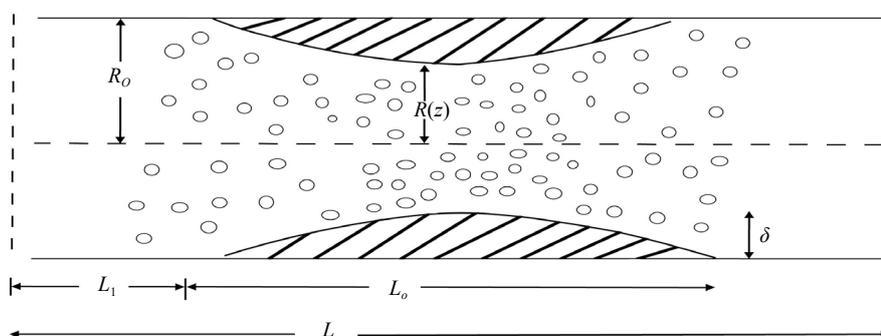


Figure 1. Geometry of stenosis in an artery

Mathematically, the radius of an artery with this sort of stenosis is expressed by Young [1]:

$$R(z, t) = \begin{cases} R_o - \delta(1 - e^{-z/T}) \left[1 + \cos \frac{2\pi(z - L_1 - \frac{L_o}{2})}{L_o} \right], & L_1 < z < L_1 + L_o \\ R_o, & \text{otherwise} \end{cases} \quad (1)$$

where L_1 is the position of stenosis, R_o is the radius of the artery that is outside of the stenosis, $R(z, t)$ is the radius of the stenosed fraction of the arterial segment under consideration, and is expected to be a function of time t , and the longitudinal distance z from the left end of the segment, parameter T is the time constant, δ is the maximum height of the stenosis and L_o is the length of the stenotic region with L as the length of arterial portion. Here, we assumed that a one-dimensional, laminar, axisymmetric, and fully developed Newtonian fluid would flow through the artery in a steady state. The equation of motion is given as follows by Jain et al. [11]:

$$-\frac{dp}{dz} + \frac{\mu(z)}{r} \frac{d}{dr} \left\{ r \frac{dw}{dr} \right\} = 0 \quad (2)$$

where w stands for axial velocity, p stands for fluid pressure, stands for pressure gradient, and $\mu(z)$ is for the viscosity of blood and is a function of z . The viscosity variation in the artery given by Equation (3) in the stenotic region is very natural because blood cells gathered just before the minimum gap where the radius is also minimal, so the viscosity is maximal [11].

$$\begin{aligned} \mu(z) &= \mu_1 \left(\frac{R(z, t)}{R_o} \right)^{-\alpha} & L_1 \leq z \leq L_1 + L_o \\ &= \mu_1 & \text{otherwise} \end{aligned} \quad (3)$$

where the index of viscosity change in the stenotic region is given as α . Its value can be taken as any constant parameter; μ_1 stands for the constant viscosity of the blood. Given below are the boundary conditions pertaining to Equation (2):

$$\begin{aligned} w &= 0 & \text{at } r = R(z, t) \\ \frac{dw}{dr} &= 0 & \text{at } r = 0 \end{aligned} \quad (4)$$

2.2 Method of solution

Working on Equation (2) with the implementation of Equation (4), we get,

$$u = \frac{1}{4\mu} \frac{dp}{dz} \left\{ \frac{(r^2 - R^2(z, t))}{\mu(z)} \right\} \quad (5)$$

the volumetric rate of flow/flux Q is given by:

$$Q = \int_0^R 2\pi w u \, dr \quad (6)$$

or

$$Q = -\frac{\pi R^4(z, t)}{8\mu(z)} \frac{dp}{dz} \quad (7)$$

where Q is constant.

From Equation (7), we can obtain a pressure gradient:

$$\frac{dp}{dz} = -\frac{8\mu(z)Q}{\pi R^4(z,t)} \quad (8)$$

Now, integrating the Equation (8), and taking $p = p_0$ at $z = 0$ and $p = p_L$ at $z = L_1$, we get:

$$\Delta p = p_0 - p_L = \frac{8Q}{\pi} \int_0^L \frac{dz}{\frac{R^4(z,t)}{\mu(z)}} \quad (9)$$

Thus, the resistance to flow λ is defined by:

$$\lambda = \frac{\Delta p}{Q} = \frac{8}{\pi} \int_0^L \frac{dz}{\frac{R^4(z,t)}{\mu(z)}} \quad (10)$$

This equation, i.e., Equation (10) shows the effect of viscosity change on the flow of resistance at the arterial wall, and the concept of this is used here for viscosity variation as it is given in Equation (3), Now we can rewrite Equation (10) as:

$$\lambda = \frac{8\mu}{\pi R_o^4} \left[L - L_o + \int_{L_1}^{L_1+L_o} \frac{dz}{\left(\frac{R(z,t)}{R_o}\right)^{4+\alpha}} \right] \quad (11)$$

The peripheral resistance in this equation is affected by the viscosity change α . In the case of no viscosity variation $\alpha = 0$, i.e., for constant viscosity, the result is the same as given in the case of Young [1]. The following three cases ($\alpha = 0, \alpha = 1, \alpha = 2$) are considered to observe the effect of α on λ :

For $\alpha = 0$,

$$\bar{\lambda} = \frac{\lambda \pi R_o^4}{8\mu L} = 1 - \frac{L_o}{L} \sum_{i=1}^n X_i Y_i + \frac{L_o}{L} \left\{ \left(1 - \frac{\delta(1-e^{-\gamma/r})}{R_o} \right) \left(\left(1 - \frac{2\delta(1-e^{-\gamma/r})}{R_o} \right)^{-\gamma/2} \right) \right. \\ \left. \left(1 - \frac{2\delta(1-e^{-\gamma/r})}{R_o} + \frac{5}{2} \left(\frac{\delta(1-e^{-\gamma/r})}{R_o} \right)^2 \right) \right\} \quad (12)$$

For $\alpha = 1$,

$$\bar{\lambda} = \frac{\lambda \pi R_o^4}{8\mu L} = 1 - \frac{L_o}{L} + \frac{L_o}{4L} \left(1 - \frac{2\delta(1-e^{-\gamma/r})}{R_o} \right)^{-3/2} \left\{ 4 \left(1 - \frac{\delta(1-e^{-\gamma/r})}{R_o} \right)^4 + 12 \frac{\delta^2(1-e^{-\gamma/r})^2}{R_o^2} \left(1 - \frac{\delta(1-e^{-\gamma/r})}{R_o} \right)^2 \right. \\ \left. + \frac{3}{2} \left(\frac{\delta(1-e^{-\gamma/r})}{R_o} \right)^4 \right\} \quad (13)$$

For $\alpha = 2$,

$$\bar{\lambda} = \frac{\lambda \pi R_o^4}{8\mu L} = 1 - \frac{L_o}{L} + \frac{L_o}{4L} \left(1 - \frac{\delta(1-e^{-\gamma/r})}{R_o} \right) \left(1 - \frac{2\delta(1-e^{-\gamma/r})}{R_o} \right)^{-1/2} \left\{ 4 \left(1 - \frac{\delta(1-e^{-\gamma/r})}{R_o} \right)^4 \right. \\ \left. + 20 \frac{\delta^2(1-e^{-\gamma/r})^2}{R_o^2} \left(1 - \frac{\delta(1-e^{-\gamma/r})}{R_o} \right)^2 + \frac{25}{2} \left(\frac{\delta(1-e^{-\gamma/r})}{R_o} \right)^4 \right\} \quad (14)$$

where $\bar{\lambda} = \frac{\lambda}{\lambda_k}$ and $\lambda_k = \frac{8\mu L}{\pi R_o^4}$.

The wall shear stress is now obtained by using the formula given below:

$$\tau_w = -\mu(z) \left. \frac{dw}{dz} \right|_{r=R(z,t)} \quad (15)$$

$$\tau_w = -\frac{4\mu(z)Q}{\pi R(z,t)^3} \quad (16)$$

This gives shear stress on the wall at maximum stenosis height as:

$$\tau_w = \frac{4\mu_1}{\pi R_o^3} \left(\frac{R(z)}{R_o} \right)^{-3-\alpha} \quad (17)$$

After solving this equation, we will get

$$\bar{\tau}_w = \left(1 - \frac{2\delta(1 - e^{-t/T})}{R_o} \right)^{-3-\alpha} \quad (18)$$

If $\alpha = 0$, the result is similar to the result given by Young [1]. The following three cases are examined to observe the effect of α on wall shear stress:

$$\text{For } \alpha = 0, \bar{\tau}_w = \left(1 - \frac{2\delta(1 - e^{-t/T})}{R_o} \right)^{-3} \quad (19)$$

$$\text{For } \alpha = 1, \bar{\tau}_w = \left(1 - \frac{2\delta(1 - e^{-t/T})}{R_o} \right)^{-4} \quad (20)$$

$$\text{For } \alpha = 2, \bar{\tau}_w = \left(1 - \frac{2\delta(1 - e^{-t/T})}{R_o} \right)^{-5} \quad (21)$$

3. Results and discussion

The changes in flow resistance in the presence of stenosis given by Equations (12) to (14) for different parameters of viscosity and time are depicted in Figures 2 to 4. The change in flow resistance with the height of stenosis for various values of dimensionless time t/T , when the viscosity is constant, i.e., α is zero is shown in Figure 2. Figures 3 and 4 show the change in flow resistance due to stenosis height for various values of stenosis length for $\alpha = 1$ and 2. It has been shown in these figures that flow resistance increases with increasing stenosis height δ/R_o , and dimensionless time t/T . The changes in wall shear stress in the presence of stenosis are revealed in Figures 5 and 7. The result shows that wall shear stress increases as δ/R_o i.e., stenosis height, and t/T (dimensionless time) increases. The changes in resistance to flow and wall shear stress with different values of index of viscosity variation are seen in Figures 8 and 9. It has been shown that the resistance to flow and wall shear stress increases as a consequence of the increase in the value of alpha, i.e., the rate of the viscosity variation.

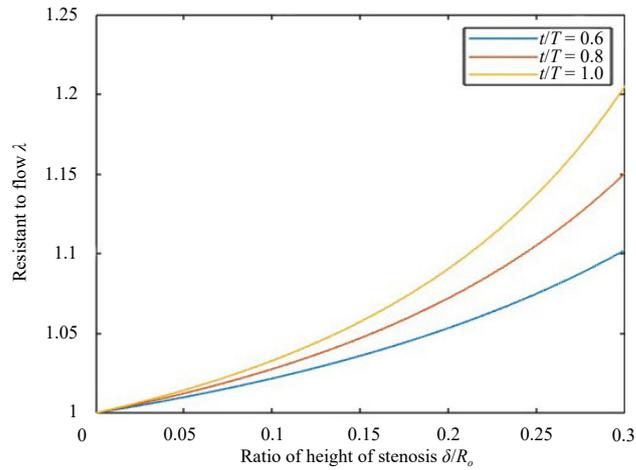


Figure 2. Resistance to flow when $\alpha = 0$ with stenosis height for different parameters of time

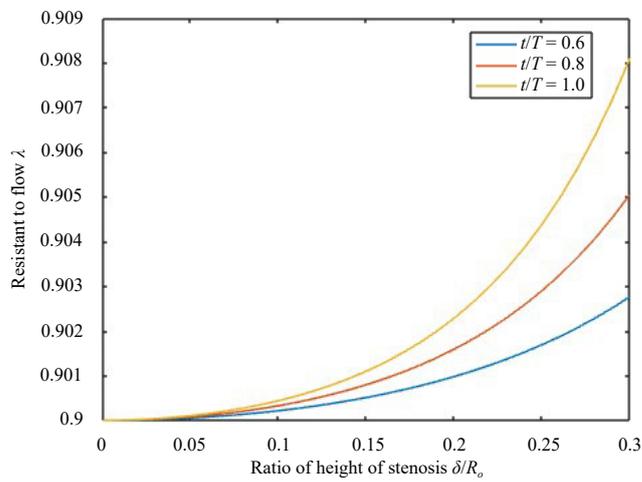


Figure 3. Resistance to flow when $\alpha = 1$ with stenosis height for different parameters of time

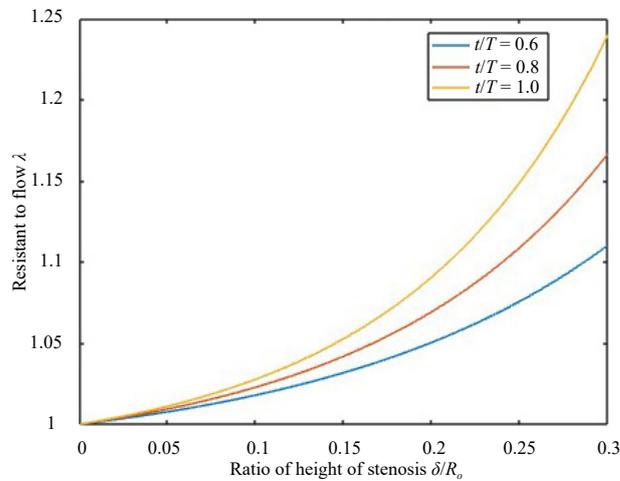


Figure 4. Resistance to flow when $\alpha = 2$ with stenosis height for different parameters of time

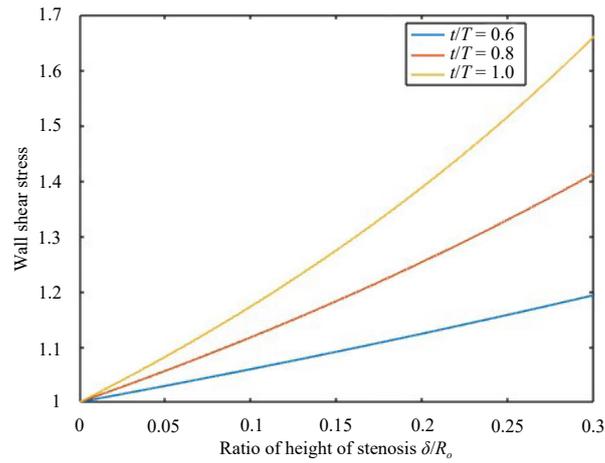


Figure 5. Wall shear stress when $\alpha = 0$ with stenosis height for different parameters of time

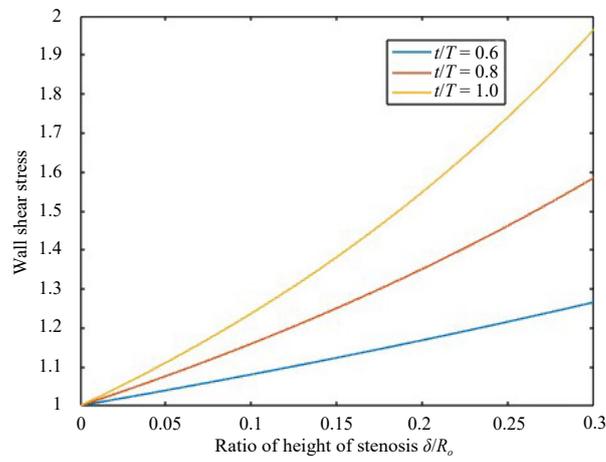


Figure 6. Wall shear stress when $\alpha = 1$ with stenosis height for different parameters of time

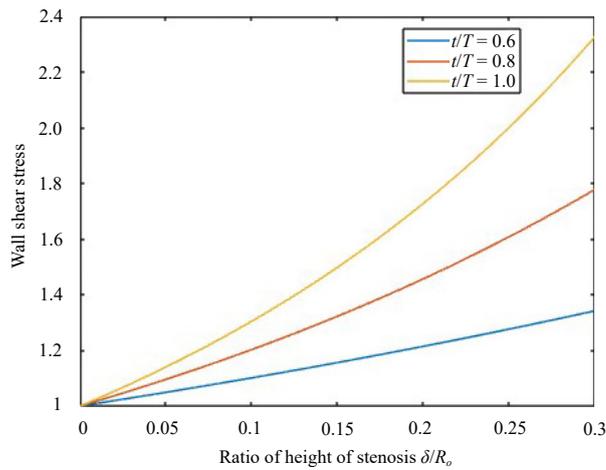


Figure 7. Wall shear stress when $\alpha = 2$ with stenosis height for different parameters of time

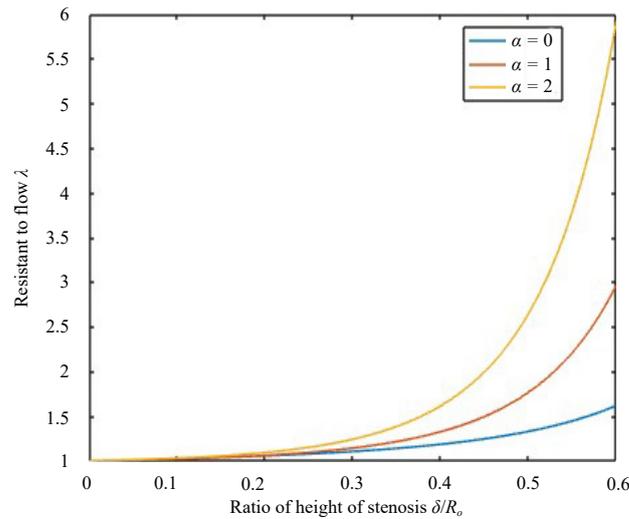


Figure 8. Variation of resistance to flow with height of stenosis for different values of α

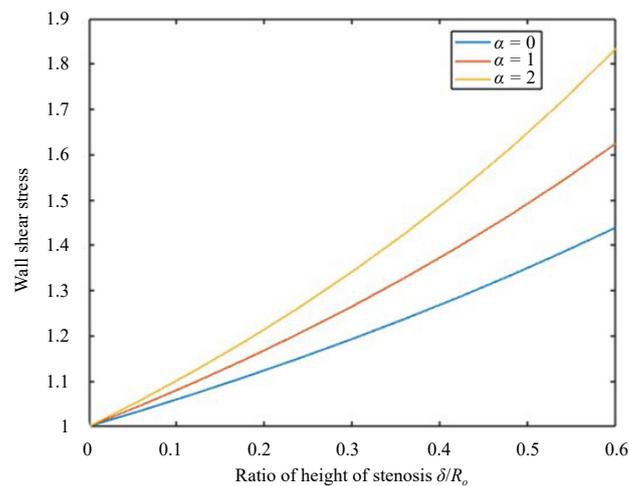


Figure 9. Variation of wall shear stress with height of stenosis for different values of α

4. Conclusion

A model has been proposed and analyzed to study the effects of aggregation of red cells on the blood flow, causing the axial variation of viscosity in the stenotic region of an artery with time-dependent stenosis. It has been shown that resistance to flow and wall shear stress increases because of an increase in the value of alpha, i.e., the rate of viscosity variation. Moreover, it has been shown that for a fixed value of stenosis length and rate of viscosity variation, the peripheral resistance and wall shear rise as the height of stenosis grows. Hence, it is concluded that as the height of stenosis increases, the wall shear stress and resistance to flow also increase. Furthermore, if the time parameter shoots up, the resistance to flow and wall shear stress also becomes greater. The study also shows that blood viscosity variation has a dramatic effect on blood flow and pressure. Viscous blood has greater resistance to flow, and increased total peripheral resistance and hence reduces blood flow. Therefore, this also raises blood pressure. This work is helpful for biomedical sciences in treating stenosis arteries as the presence of stenosis in the vessels partially blocks the supply of blood to the brain and heart which can lead to a stroke. The results can be used by medical scientists to maintain the viscosity of blood

in case of a heart or brain stroke. It has contributed to a better understanding of the development or deterioration of this disease, its diagnosis, and treatment.

Conflict of interest

The author declare no conflict of interest for this study.

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