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Pulsatile unsteady flow of blood through porous medium in a stenotic artery under the influence of transverse magnetic field

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Abstract

The periodic nature of the cardiac cycle induces a pulsatile, unsteady flow within the circulatory system. The pulsatile model of blood flow provides data to analyse the physiological situation in close proximity. The distribution of fatty cholesterol and artery-clogging blood clots in the lumen of the coronary artery is assumed as a porous medium. A mathematical model for pulsatile flow through an stenosed artery filled with porous medium in the presence of transverse static magnetic field has been formulated under the consideration of hematocrit dependent viscosity of blood that governed by Einstein equation. The velocity profile, volume flux, pressure gradient and wall shear stress are obtained and the effects of magnetic number, Darcy number, Womersely number are computed and represented through graphs.

Keywords: pulsatile flow, stenosis, MHD, wall shear stress

1. Introduction

Blood flow under normal physiological conditions is an important field of study, as is blood flow under disease condition. The majority of deaths in developed countries result from cardiovascular diseases, most of which are associated with some form of abnormal blood flow in arteries. Globally, physicians are of the same opinion that there are three major features of the vascular diseases that potentially need treatment (i) vasospasm (spasm of blood vessels), (ii) aproliferative vasculopathy (thickening of blood vessels), and (iii) thrombosis (blood clots) or structural occlusion of the vessel lumen (blockage of blood vessels).

Blood clotting is the body's natural defense against bleeding. A clot, or "thrombus, develops whenever there is damage to a blood carrying vessel. The platelets and proteins in the blood work together to regulate the clotting process. If the process does not work correctly, a clot can be formed in the blood vessels. Several authors reported that thrombus superimposed on ruptured atherosclerotic plaque is commonly found in autopsy studies of heart disease (Davies and Thomas, 1984; 1985; Davies, 1990). Constantinides (1990) discussed the cause of thrombosis in human atherosclerotic artery and reported that thrombosis is also associated with carotid artery plaque rupture in stroke and transient ischemic attack. Dash *et al.* (1996) showed that in some pathological situations, the distribution of fatty cholesterol and artery-clogging blood clots in the lumen of the coronary artery can be considered as equivalent to a fictitious porous medium. David *et al.* (2001) has developed a species transport model of platelet accumulation which included mechanisms of convection, shear-enhanced diffusion, near-wall platelet concentration, and a kinetic model of platelet activation and aggregation for an initial quantitative estimate of the likelihood of occlusive thrombus in individual patients due to plaque erosion, artery spasm, incomplete angioplasty, or plaque rupture. Xu *et al.* (2010) considered the blood clot as a porous medium to account for the transport property of blood flow in the extension of multiscale model by including a detailed submodel of surface-mediated control of blood coagulation (Xu *et al.*, 2008; 2009). EL-Shahed (2003) studied the pulsatile flow of blood through stenosed porous medium in the presence of periodic body acceleration. EL Shehawey and EL Sebaei (2000) have studied peristaltic transport in a cylindrical tube through a porous medium.

Kolin (1936) has coined the idea of electromagnetic field in the medical research for the first time in the year 1936 and has established that the biological systems in general are greatly affected by the application of external magnetic field. As per the investigations reported by Barnothy (1964-1969), the heart rate decreases by exposing biological systems to an external magnetic field. Korchevskii and Marochnik (1965) have discussed the possibility of regulating the blood movement in human system by applying magnetic field. In the decade of eighties, engineers attracted towards applications of magnetic field in biomedical flow primarily with a view to utilizing MHD (magnetohydrodynamics) in controlling blood flow velocities in surgical procedures and also establishing the effects

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of magnetic fields on blood flows in astronauts, citizens living in the vicinity of EM (electromagnetic) towers *etc*. Keltner *et al.* (1990) reported an analysis of the pressure changes in vessels of the human vasculature under the action of strong magnetic fields. Their study indicated that 15% Sodium Chloride solutions are retarded by transverse magnetic fields of 2.3 and 4.7 Tesla for fluxes below 0.5 l/min. Several researchers have worked out significant studies on hydromagnetic blood flow in artery (Halder and Ghosh, 1994; McKay *et al.*, 2007; Mekheimer and EI Kot, 2008; Rathod and Tanveer, 2009; Tzirtzilakis, 2005). Layek and Mukhopadhyay (2008) and Kumar *et al.*(2011) worked on a mathematical model to study flow through a variable shape stenosed artery under the influence of magnetic field and demonstrated the effect of stenosis shape and magnetic field on the resistance to the flow .

Several researchers Fung (1984), Mazumdar (1992), McDonald (1960) and Zamir (2005) have given mathematical treatment to the blood flow in arteries subject to various physiological conditions. Young (1968) presented analysis of flow through an occluded tube under a pulsatile pressure gradient. Mazumdar *et al*. (1996) studied the time variation of various characteristic of Newtonian flow of blood through a stenosed artery and observed that the pressure gradient attains the maximum at the point of maximum constriction and decreases with increase of hematocrit parameter. Sanyal and Maiti (1998) have studied a mathematical model on arterial blood flow in the presence of mild stenosis and obtained the pressure gradient and wall shear stress using series solution. They found that the pressure gradient increases with the increase in hematocrit value which indicates that there is higher value in systolic and lower value in diastolic pressure. Experimental and numerical study is carried out by Deplano and Siouffi (1999) for pulsatile flow of blood through stenosis; wall shear analysis and explained the adversity of the stenosis on a healthy artery. Lee and Xu (2002) worked out the results on velocity profiles, wall shear stress intramural strain and stress for the rigid and compliant cases for the mild stenosed tube.

These researches motivated for the present study to deal with the blood flow through an artery filled with porous medium.

2. Formulation of Problem

The present mathematical model is modeled with the assumption that blood flow in the tube is a suspension of RBC in plasma. Hematocrit concentration dependent viscosity of the blood is considered that is governed by the Einstein equation

$$\mu = \mu_0[1 + \beta\ h(r)] \tag{1}$$

here, μ_0 is the coefficient of viscosity of plasma, β a constant, and h(r) the hematocrit concentration which vary along the radial direction described by the equation

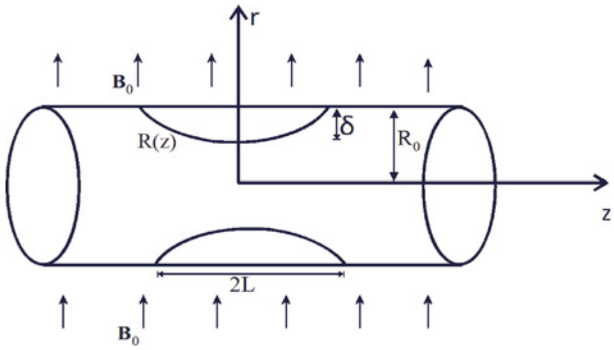

Fig. 1. Physical model of the stenosed artery.

$$h(r) = Hm\left[1-\left(\frac{r}{R_0}\right)^n\right] \tag{2}$$

with Hm the maximum hematocrit concentration at the axis of the vessel.

The blood vessel geometry is determined by the radius R_0 of the inlet and outlet unconstricted segment, whereas the radius of the smooth axisymmetric constricted segment is given by

$$R(z) = \begin{cases} R_0 - \frac{\delta}{2}\left(1+\cos\frac{\pi z}{L}\right) & -(L \le z \le L) \\ R_0 & \text{otherwise} \end{cases} \tag{3}$$

here 2L is the length of stenosis and δ is maximum thickness of the stenosis. In the cylindrical coordinate system (r, θ, z), the axis of the vessel coincides with the z-axis and the origin $z = 0$ corresponds to the neck of the stenosis. We are concerned with the static boundary wall, therefore, on the rigid no-slip wall the velocity is vanished. At the inflow section, the radial velocity vanishes and the main flow is assumed to be fully developed. The diameter of the artery is not less than 1mm so that Fahreus-Lindquist effect is not significant. The tube is filled with porous material of constant permeability K.

When an electrically conducting fluid like blood flow in a magnetic field, an electromagnetic force will be produced due to the interaction of current with magnetic field. The electromotive force is proportional to the speed of motion and the magnetic flux intensity B (Tashtoush and Magableh, (2008)). The Maxwell's equations are

$$\text{div}\mathbf{B} = 0 \tag{4}$$

$$\text{curl}\mathbf{B} = \mu_m J \tag{5}$$

$$\text{curl}\mathbf{E} = -\frac{\partial \mathbf{B}}{\partial t} \tag{6}$$

Where **E** is the electric field intensity, **B** is the magnetic flux intensity μ_m is the electric permeability and **J** is the current density. If σ is the electrical conductivity, Then generalized Ohm's law is

$$\mathbf{J} = \sigma(\mathbf{E}+\mathbf{V}\times\mathbf{B}). \tag{7}$$

The induced electromagnetic force $F^{(em)}$ is defined as

$$F^{(em)} = \mathbf{J}\times\mathbf{B} = \sigma(\mathbf{E}+\mathbf{V}\times\mathbf{B})\times\mathbf{B}. \tag{8}$$

Following Cowling (1957) and Gupta (1960) that there is no applied or polarization voltage so that $\mathbf{E}=0$. We assumed a magnetic field $\mathbf{B}=(B_0,0,0)$ with a constant transverse magnetic flux density B_0 of moderate strength so that induced magnetic field is negligible. The resultant force, the magnetohydrodynamic force is

$$F^{(em)} = \mathbf{J}\times\mathbf{B} = -\sigma B_0^2 w\hat{k}. \tag{9}$$

Invoking these assumptions the governing equations of the motion of blood as Newtonian incompressible fluid through a porous medium with axisymmetric condition is given by

$$\rho\frac{\partial w}{\partial T} = -\frac{\partial p}{\partial z}+\frac{1}{r}\frac{\partial}{\partial r}\left(\mu\, r\frac{\partial w}{\partial r}\right)-\sigma B_0^2 w-\frac{\mu}{K}w. \tag{10}$$

The boundary conditions are

$$w=0 \quad for \quad r=R(z), \tag{11}$$

$$\frac{dw}{dr}=0 \quad for \quad r=0. \tag{12}$$

Taking unconstricted radius R_0 and t_0 as length and time scaling parameter respectively, the governing equation reduced to

$$\frac{\rho R_0^2}{t_0\mu_0}\frac{\partial w}{\partial t} = \frac{-R_0^2}{\mu_0}\frac{\partial p}{\partial z}+\frac{1}{y}\frac{\partial}{\partial y}\left[(a-ky^n)y\frac{\partial w}{\partial y}\right] -\frac{R_0^2\sigma B_0^2 w}{\mu_0}-\frac{(a-ky^n)}{Da^2}w \tag{13}$$

where $y = \frac{r}{R_0}$, $t=\frac{T}{t_0}$, $\beta Hm = k$, and $a = 1 + k$.

The driving force for the motion of blood in the cardiovascular system is a local pressure gradient along the longitudinal direction of the vessel, which in turn is determined by the propagation of the heart pressure pulse. The blood pumped by the heart is of periodic nature, that is pulsating, therefore pressure is periodic, can be expressed in Fourier series (Burton, 1996; Chakravarty and Sen, 2005; Javadzadegan *et al.*, 2009; McDonald, 1960). Therefore, for the sake of simplicity, it is assumed that the pressure gradient is known as a function of time. Taking

$$-\frac{R_0^2}{\mu_0}\frac{\partial p}{\partial z} = ce^{i\omega t} \tag{14}$$

where $\omega=2\pi f$, f is the heart pulse frequency and c is the amplitude of the pulsatile flow.

Also, taking $w(y, t) = W(y)\, e^{i\omega t}$, then (15)

we have from (13)

$$\frac{1}{y}\frac{d}{dy}\left[(a-ky^n)y\frac{dW}{dy}\right]-\left(\alpha^2 i+H^2+\frac{a}{Da^2}\right)W+\frac{ky^n}{Da^2}W=-c \tag{16}$$

where $\frac{R_0^2\sigma B_0^2}{\mu_0}=H^2$; $\frac{K}{R_0^2}=Da^2$; $\alpha^2=\frac{\rho R_0^2}{t_0\mu_0}\omega$ are dimensionless parameters, H the Hartmann number, Da the Darcy number and α the Womersley number.

The corresponding boundary conditions (11) and (12) are transformed to

$$W=0 \quad \text{at} \quad y=\frac{R(z)}{R_0}, \tag{17}$$

$$\frac{dW}{dy}=0 \quad \text{at} \quad y=0. \tag{18}$$

3. Method of Solution

For the solution of the differential equation (16) we have used Frobenius method. For implementing Frobenius method, it is required that W is bounded at y = 0. The only admissible solution satisfying the boundary condition (17) is

$$W=D\sum_{m=0}^{\infty}A_m y^m-\frac{c}{4a}\sum_{m=0}^{\infty}\lambda_m y^{m+2}. \tag{19}$$

Here, the second term of the right hand side is the solution corresponding to non-homogenous part of the equation (16). A_m and λ_m are the series constant, D is an arbitrary constant to be determined by the boundary condition (17). Firstly, we find the solution of homogenous part of the equation (16) with

$$W=D\sum_{m=0}^{\infty}A_m y^m, \tag{20}$$

$$\frac{dW}{dy}=D\sum_{m=1}^{\infty}A_m m y^{m-1}, \tag{21}$$

$$\frac{d^2W}{dy^2}=D\sum_{m=2}^{\infty}A_m m(m+1)y^{m-2}. \tag{22}$$

Clubbing these with the homogenous part of the equation (16) we get

$$[ay-ky^{n+1}]\sum_{m=2}^{\infty}A_m m(m+1)y^{m-2}+[(a+-ky^n-kny^n)]\sum_{m=1}^{\infty}A_m m y^{m-1}$$

$$-\left[\left(\alpha^2 i+H^2+\frac{a}{Da^2}\right)y+\frac{ky^{n+1}}{Da^2}\right]\sum_{m=0}^{\infty}A_m y^m=0. \tag{23}$$

Comparing the coefficient of y^m, we have

$$A_{m+1} = \frac{\left(\alpha^2 i + H^2 + \frac{a}{Da^2}\right)A_{m-1} + k(m+1)(m-n+1)A_{m-n+1} - \frac{k}{Da^2}A_{m-n-1}}{a(m+1)^2} \tag{24}$$

For the solution of non-homogenous part, let

$$W = -\frac{c}{4a}\sum_{m=0}^{\infty}\lambda_m y^{m+2}, \tag{25}$$

$$\frac{dW}{dy} = -\frac{c}{4a}\sum_{m=1}^{\infty}\lambda_m(m+2)y^{m+1}, \tag{26}$$

$$\frac{d^2W}{dy^2} = -\frac{c}{4a}\sum_{m=2}^{\infty}\lambda_m(m+2)(m+1)y^m. \tag{27}$$

The equation (16) gives,

$$[(a-ky^n)y]\sum_{m=2}^{\infty}\lambda_m(m+2)(m+1)y^m$$

$$+[a-ky^n-kny^n]\sum_{m=1}^{\infty}\lambda_m(m+2)my^{m+1}$$

$$-\left(\alpha^2 i + H^2 + \frac{a}{Da^2}\right)y\sum_{m=0}^{\infty}\lambda_m y^{m+2} + \frac{ky^{n+1}}{Da^2}\sum_{m=2}^{\infty}\lambda_m y^{m+2} = 4ay. \tag{28}$$

Comparing the coefficient of y^{m+2} we have

$$\lambda_{m+1} = \frac{\left(\alpha^2 i + H^2 + \frac{a}{Da^2}\right)\lambda_{m-1} + k(m+3)(m-n+3)\lambda_{m-n+1} - \frac{k}{Da^2}\lambda_{m-n-1}}{a(m+3)^2} \tag{29}$$

with $A_0 = \lambda_0 = 1$ and $A_{-m} = \lambda_{-m} = 0$.

The constant D involved in the solution (19) is obtained with the help of boundary condition (17) and given by

$$D = \frac{c}{4a}\frac{\sum_{m=0}^{\infty}\lambda_m\left(\frac{R(z)}{R_0}\right)^{m+2}}{\sum_{m=0}^{\infty}A_m\left(\frac{R(z)}{R_0}\right)^m}. \tag{30}$$

Then

$$W(y) = \frac{c}{4a}\frac{\sum_{m=0}^{\infty}\lambda_m\left(\frac{R(z)}{R_0}\right)^{m+2}\sum_{m=0}^{\infty}A_m y^m - \sum_{m=0}^{\infty}\lambda_m\left(\frac{R(z)}{R_0}\right)^m\sum_{m=0}^{\infty}\lambda_m y^{m+2}}{\sum_{m=0}^{\infty}A_m\left(\frac{R(z)}{R_0}\right)^m} \tag{31}$$

and

$$w(y,t) = W(y)e^{i\omega t}. \tag{32}$$

In particular, in the absence of the hematocrit, the average velocity w_0 is given by

$$w_0 = \frac{c_0 e^{i\omega t}}{\left(\alpha^2 i + \frac{1}{Da^2}\right)}\left[1 - \frac{I_0\left(\sqrt{\alpha^2 i + \frac{1}{Da^2}}\,y\right)}{I_0\left(\sqrt{\alpha^2 i + \frac{1}{Da^2}}\right)}\right]. \tag{33}$$

The dimensionless form of w(y,t) with respect to w_0 is now obtained from the equations (32) and (33) and given by

$$\bar{w} = \frac{w}{w_0}. \tag{34}$$

Volumetric Flow Rate : The volumetric flow rate Q of the fluid in the stenotic region is given by

$$Q = 2\pi R_0\int_0^{R/R_0} wy\,dy. \tag{35}$$

Let Q_0 denotes the flow rate of plasma fluid in unconstricted tube which is given by

$$Q_0 = -\frac{\pi R_0^3}{8\mu_0}\left(\frac{\partial p}{\partial z}\right)_0 \tag{36}$$

where $\left(\frac{\partial p}{\partial z}\right)_0$ being the pressure gradient of the fluid in unconstricted uniform tube.

Thus non-dimensional flow rate $\bar{Q} = \frac{Q}{Q_0}$ is given by

$$\bar{Q} = \frac{4}{a}\frac{\frac{\partial p}{\partial z}}{\left(\frac{\partial p}{\partial z}\right)_0}\times\frac{\left[\sum_{m=0}^{\infty}\lambda_m\left(\frac{R}{R_0}\right)^{m+2}\sum_{m=0}^{\infty}\frac{A_m}{m+2}\left(\frac{R}{R_0}\right)^{m+2} - \sum_{m=0}^{\infty}A_m\left(\frac{R}{R_0}\right)^m\sum_{m=0}^{\infty}\frac{\lambda_m}{m+4}\left(\frac{R}{R_0}\right)^{m+4}\right]}{\sum_{m=0}^{\infty}A_m\left(\frac{R}{R_0}\right)^m} \tag{37}$$

The expression for the relative pressure gradient can be obtained by

$$\frac{\left(\frac{\partial p}{\partial z}\right)}{\left(\frac{\partial p}{\partial z}\right)_0} = \frac{\frac{a\bar{Q}}{4Q_0}\sum_{m=0}^{\infty}A_m\left(\frac{R}{R_0}\right)^m}{\sum_{m=0}^{\infty}\lambda_m\left(\frac{R}{R_0}\right)^{m+2}\sum_{m=0}^{\infty}\frac{A_m}{m+2}\left(\frac{R}{R_0}\right)^m - \sum_{m=0}^{\infty}A_m\left(\frac{R}{R_0}\right)^m\sum_{m=0}^{\infty}\frac{\lambda_m}{m+4}\left(\frac{R}{R_0}\right)^{m+4}}. \tag{38}$$

Wall Shear Stress : The shear stress at the surface of stenosis is described by

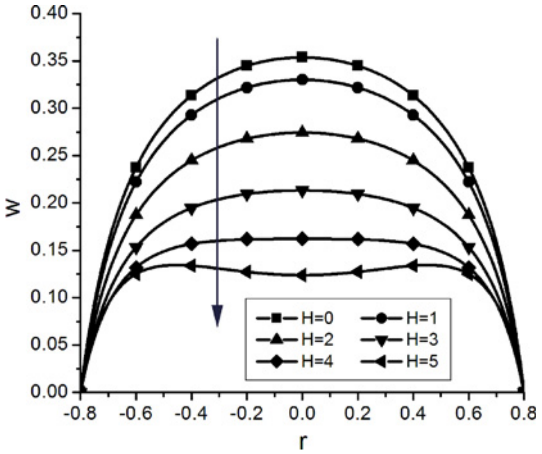

Fig. 2. Variation of axial velocity radially with magnetic number (H) at n=2, Hm=0.45, Da=10, α=1.83, β=2.5.

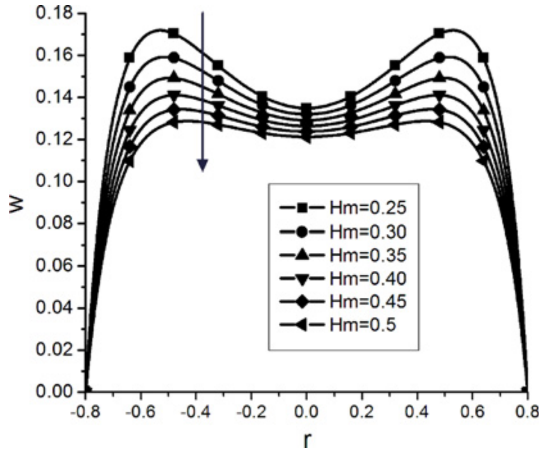

Fig. 3. Variation of axial velocity radially with Hematocrit(Hm) at n=2, Da=10, H=5, α=1.83, β=2.5, z=0.5.

$$\tau_s = -\left[\mu(r)\frac{dw}{dr}\right]_{r=R}. \tag{39}$$

With the help of (32) and value of $\mu(r) = \mu_0[1 + \beta h(r)]$, it can be written as

$$t_s = -\mu_0\left[a-k\left(\frac{R}{R_0}\right)^n\right]\frac{ce^{i\omega t}}{4aR_0}\frac{\left[\begin{array}{l}\sum\lambda_m\left(\frac{R}{R_0}\right)^{m+2}\sum(m+1)A_{m+1}\left(\frac{R}{R_0}\right)^m\\ -\sum A_m\left(\frac{R}{R_0}\right)^m\sum(m+3)\lambda_{m+1}\left(\frac{R}{R_0}\right)^{m+2}\end{array}\right]}{\sum A_m\left(\frac{R}{R_0}\right)^m}. \tag{40}$$

Also, if τ_N is the shear stress at the wall in the absence of stenosis, then

$$\tau_N = \frac{\mu_0 c_0 e^{i\omega t}}{R_0}\frac{I_1\left(\sqrt{\alpha^2 i+\frac{1}{Da^2}}\,y\right)}{I_0\left(\sqrt{\alpha^2 i+\frac{1}{Da^2}}\right)}. \tag{41}$$

The non-dimensional form of shear stress is now obtained as

$$\bar{\tau} = \frac{\tau_s}{\tau_N}. \tag{42}$$

4. Results and Discussion

The expression of velocity, wall shear stress, volumetric flow rate and pressure gradient are obtained and computed data are plotted for different values of Hartmann number H, Darcy number Da, Wormersley number α and Hematocrit Hm. The profiles of axial velocity versus radial coordinate for various physical parameters are shown in Figs. 2-5. Fig. 2 depicts that with the increase of Hartmann number (H) the flowing fluid is slowed down in axial direction. Besides, deceleration in the flow with increase of Hartmann number the axial velocity profile ceases to remain parabolic and becomes flatten at the centerline region. For H=4 the velocity profile is almost flat at the centerline region while on further increasing the value of Hartmann number the profile at centerline region bends in reverse direction. Fig. 3 shows the velocity profiles at a various level of Hematocrit concentration at the fixed value of Hartmann number H=5 (at which in the centerline region profile bend in reverse direction of flow). Besides, slowed down in the axial flow velocity with the increase of Hematocrit concentration, it is plausible that the velocity profile in centerline region for less Hematocrit concentrated blood corresponds more curvature as compared to higher Hematocrit concentrated blood. Fig. 4 demonstrates that the axial flow velocity increases on increasing Womersley number, *i.e.* flow velocity will be augmented by raising the oscillation in the flow. Fig. 5 depicts that if porosity of the medium in the vessel increases there is amplification in the

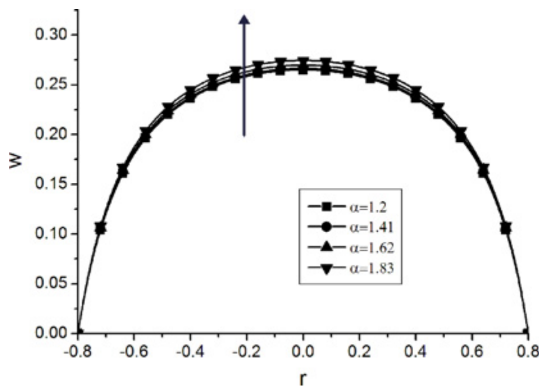

Fig. 4. Variation of axial velocity radially with womersley number(α) at n=2, Da=10, H=2, Hm=0.45, α=0.5, β=2.5.

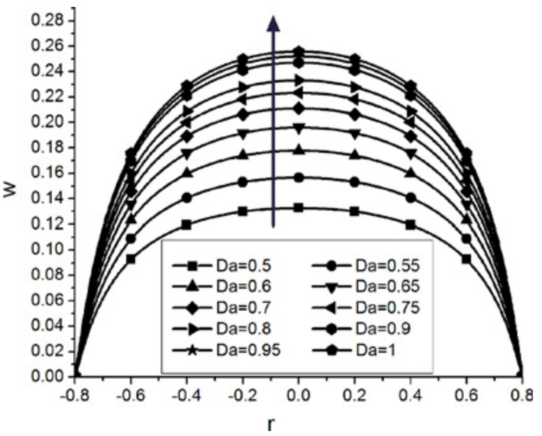

Fig. 5. Variation of axial velocity radially with Darcy number(Da) at n=2, Hm=0.45, H=2, α=1.83, β=2.5.

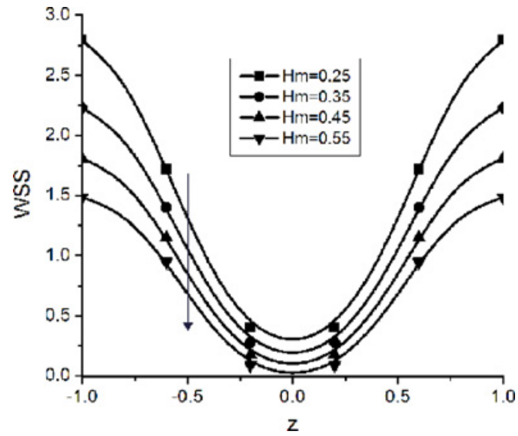

Fig. 8. Variation of Wall Shear Stress along the stenosis at n=2, H=5, Da=10, α=1.83, β=2.5.

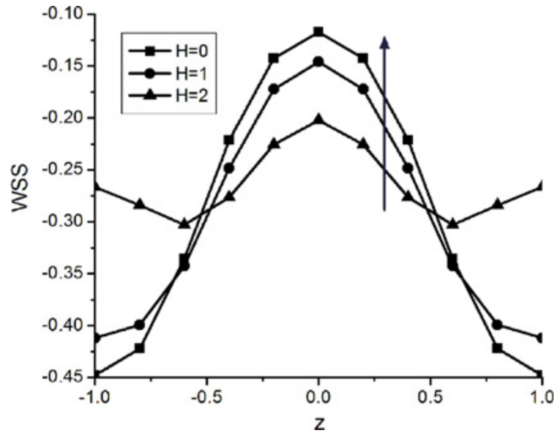

Fig. 6. Variation of Wall Shear Stress along the stenosis at n=2, Da=10, Hm=0.45, α=1.83, β=2.5.

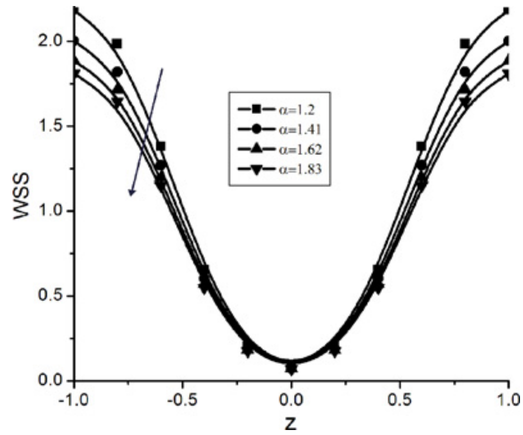

Fig. 9. Variation of Wall Shear Stress along the stenosis with Womersley number(α) at H=5, Hm=0.45, Da=10, β=2.5.

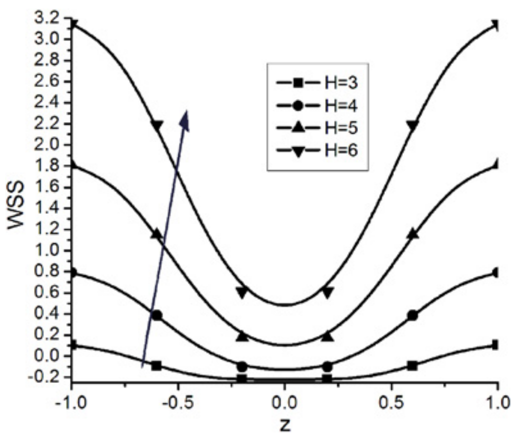

Fig. 7. Variation of Wall Shear Stess along the stenosis at n=2, Da=10, Hm=0.45, α=1.83, β=2.5.

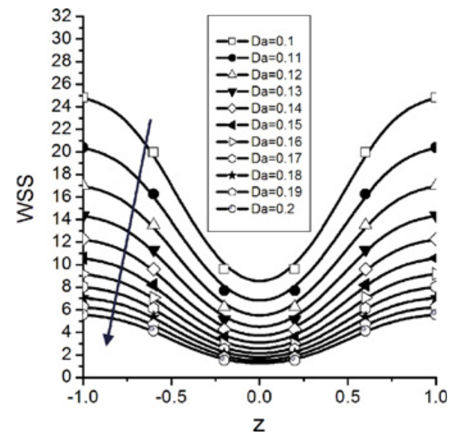

Fig. 10. Variation of Wall Shear Stress along the stenosis with Darcy number(Da) at n=2, H=2, Hm=0.45, α=1.83, β=2.5.

axial velocity. Also, for less porous medium the profiles in centerline region are flatted and converge to parabolic form on increasing porosity of the medium. Figs. 6 and 7 demonstrate the effect of magnetic field on wall shear stress (WSS) in the stenosed region of the vessel. WSS increases with the increase of magnetic field which is in good agree-

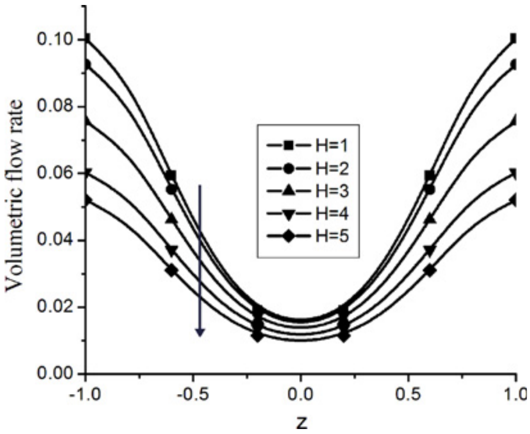

Fig. 11. Variation of volumer flow rate though stenosed region with Magnetic number(H) at Hm=0.45, Da=10, α=1.83, β=2.5.

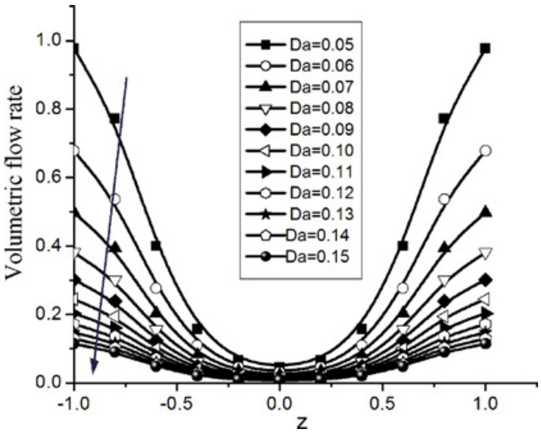

Fig. 14. Variation of volumetric flow rate through stenosed region with Darcy number(Da) at n=2, Hm=0.45, H=2, α=1.83, β=2.5.

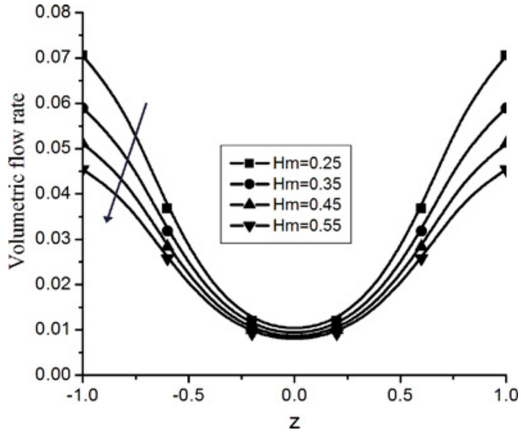

Fig. 12. Variation of Volumetric flow rate through stenosed region with Hematocrit(Hm) at H=5, Da=10, p=0.5, α=1.83, β=2.5.

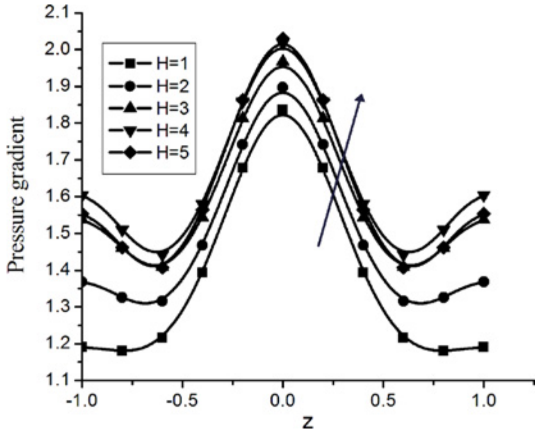

Fig. 15. Variation in pressure gradient through stenosed region with magnetic number(H) at n=2, Da=10, Hm=0.45, α=1.83, β=2.5, Q=0.5.

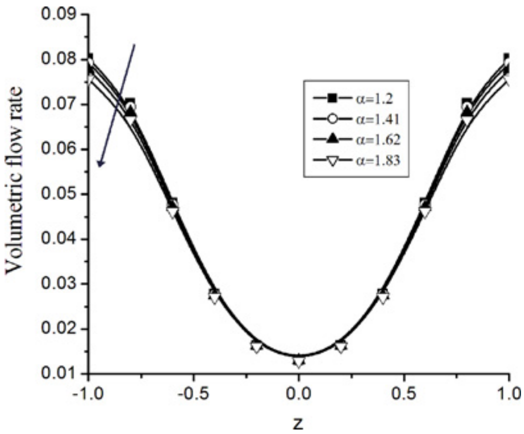

Fig. 13. Variation of voluemetric flow rate in stenosed region with Womersely number(α) at H=2, Hm=0.45, p=0.5, Da=10, β=2.5

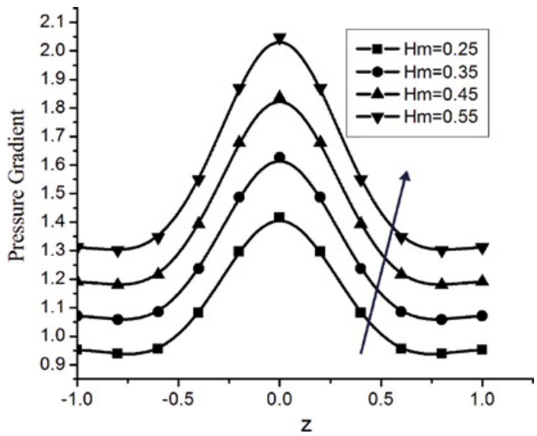

Fig. 16. Variation of pressure gradient through stenosed region with Hematocrit(Hm) at n=2, H=1, Da=10, α=1.83, β=2.5, Q=0.5.

ment with studies carried out by Sud and Sekhon (2003), Halder and Ghosh (1994), Mekheimer and El Kot (2008), Prakash *et al.* (2004). In the present study we found that WSS changes its sign twice in the region of stenosis near by entry and exit of the stenosis as seen in Fig.7. The occurrence of these variation suggests that there will be two region of circulation for the value of Hartmann number H>2. The magnetic field strength also affecting the location of the circulation region, for weak magnetic field the circulation regions occurs closures to the extremes of the stenosis. While, on increasing strength of the magnetic field the circulation region moves towards the neck of the stenosis that may affect more adversely on the hemodynamic conditions of the flow. Figs. 8-10, reveal that the WSS decreases with the increase of Hematocrit concentration, Womersley number and porosity of the medium. But there is no significant occurrence of the circulation region. Figs. 11-14 show that volume flow rate of the blood decreases with the increase of Hartmann number, Hematocrit concentration, Womersley number and Darcy number. The effect of magnetic field, porosity of the medium and Hematocrit concentration is quite significant in compare to Womersley number. Fig. 15 and Fig. 16 reveals that pressure gradient increases with the increase in Hartmann number and hematocrit concentration respectively.

5. Conclusions

1. In the magnetic field zones- in magnetic laboratory, nearby EM transmission tower *etc.*, the patient suffering with CV disease will have more risk on hemodynamic point of view as the flow in centerline region perturbed from parabolic to zero velocity gradient and to reverse flow development.

2. Both magnetic field and presence of porous medium (the resultant of thrombus in the blood vessel) reduces the amount of blood to be transported to the organs.

3. The adverse effect of magnetic field is inversely proportional to the Hematocrit concentration.

List of Symbols

A_m	series constant used in Frobenius solution in Eqn. (19)
B	magnetic flux intensity
B_0	transverse magnetic flux density
c	amplitude of the pulsatile flow
D	arbitrary constant used in Frobenius solution in Eqn. (19)
Da	Darcy number
E	electric field intensity
f	heart pulse frequency
$F^{(em)}$	induced electromagnetic force
h(r)	hematocrit concentration
H	Hartmann number,
Hm	maximum hematocrit concentration at the axis of the vessel
I_n	modified Bessel's function of second kind of order n
J	current density
$\hat{k}$	unit vector in axial direction
K	permeability of porous medium
2L	length of the stenosis
p	pressure
Q	volumetric flow rate
Q_0	flow rate of plasma fluid in unconstricted tube
$\overline{Q}$	dimensionless flow rate
r	radial coordinate
R_0	radius of normal artery
R(z)	radius of stenosed artery
t_0	time scaling parameter
T	time in dimensional form
w_0	average velocity in the absence of the hematocrit
w(y, t)	axial velocity
W(y)	steady axial flow velocity
$\overline{w}$	dimensionless axial velocity
y	dimensionless radial coordinate
z	axial coordinate

Greek Letters

α	Womersley number
β	constant equals to 2.5, that used in Einstein equation (1)
δ	maximum thickness of the stenosis
λ_m	series constant used in Frobenius solution in Eqn. (19)
μ	viscosity of the blood
μ_0	viscosity of the plasma
μ_m	electric permeability
ρ	density of the blood
σ	electrical conductivity
τ_N	shear stress at the wall in the absence of stenosis
t_s	shear stress at the surface of stenosis
$\overline{\tau}$	dimensionless shear stress

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