Original Research Article

Clinical profile of patients with established coronary artery disease and who were on atorvastatin therapy

V Rajendran[1], Aruna Ramani[2*]

[1]Associate Professor, Department of Medicine, Mount Zion Medical College, Chayalode, Ezhamkulam, Adoor, Kerala, INDIA.
[2]Consultant Neurologist, Trichirappalli, Tamil Nadu, INDIA.
Email: parithirajendra@yahoo.com

Abstract

Atherosclerosis affects varying circulatory compartments of the body but the manifestations are different. It affects the cerebral circulation and presents as transient ischemic events and stroke. It affects the coronary circulation and manifests as stable angina, unstable angina and myocardial infarction. It affects the renal arteries causing renal artery stenosis and it affects the peripheral vascular system causing claudication pain and even life threatening gangrene. The study subjects include Patients on established coronary artery disease that were on atorvastatin therapy 10 mg for more than 1 year, who developed an ischemic stroke evidenced by CT scan or MRI within 5 years of occurrence of the first coronary event. In our study the mean age of the population was 66.4 years. The majority of patients (25) in our study were in the age group 60 to 69 years. 66.6 % of women and 75.8% of men were having a higher than normal waist circumference. The results showed that with increasing obesity the prevalence of multiple ischemic events was increasing.

Keywords: Clinical Profile, coronary artery disease, atorvastatin therapy.

***Address for Correspondence:**
Dr. Aruna Ramani, Consultant Neurologist, Trichirappalli, Tamil Nadu, INDIA.
Email: parithirajendra@yahoo.com

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INTRODUCTION

The basic pathophysiology in understanding a myocardial ischemia is demand supply mismatch. That is the demand between oxygen supply and coronary blood flow. Whenever there is any demand for oxygen it is the work of myocardium to ensure that there is availability of adequate oxygen rich blood to the tissues in demand. When this does not happen that part of myocardium will suffer from ischemia.[1] The coronary circulation is regulated by the hearts oxygen needs on a minute by minute basis. This is achieved by the ability of coronary bed to greatly alter its resistance so that the myocardium always has a very high & almost stable fraction of oxygen. Ordinarily, intra myocardial resistance vessels have a greater capacity for dilation. During exercise and emotion the heart needs to pump at a faster rate resulting in great changes in its oxygen need. This is accomplished by the change in vascular resistance to a great extent. This is termed metabolic regulation of coronary vascular bed. The change in resistance of coronary vascular bed to changes in blood pressure that happens several times in a day is called auto regulation. Since the lumen of arteries is narrowed in atherosclerosis when there is an increase in demand the required oxygen supply cannot be met. This results in ischemia. When there is severe obstruction even the blood supply at rest is reduced.[2] This results in rest angina. When spasms of vessels occur as in prinzmetals angina the blood supply is reduced also resulting in ischemia. Aortitis results in narrowing of the ostia and reduction in blood supply. Congenital anomalies like anomalous origin of left anterior descending artery from pulmonary artery can lead to myocardial ischemia in infancy which is a rare cause.[3]

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Epicardial coronary arteries are the most common site for the development of atherosclerosis. Plaque formation and later rupture at segments is the cause of epicardial coronary vessel narrowing. When the rupture of plaque occurs platelets are recruited and activated and then the coagulation cascade is initiated and activated.

All this leads to accumulation of numerous fibrin strands at the site of injury. The thrombi thus formed also attract red blood cells and grows in size and can cause total occlusion of the vessel.

Atherosclerosis affects varying circulatory compartments of the body but the manifestations are different. It affects the cerebral circulation and presents as transient ischemic events and stroke. It affects the coronary circulation and manifests as stable angina, unstable angina and myocardial infarction. It affects the renal arteries causing renal artery stenosis and it affects the peripheral vascular system causing claudication pain and even life threatening gangrene. All the atherosclerotic events are accelerated in a patient with diabetes mellitus.[4]

Lipoproteins have high affinity for matrix extracellular proteins. When there is excess cholesterol they get deposited in the walls of arteries as fatty streak. They are the first macroscopic evidence of atherosclerosis. They generally adhere to tunica intima, the innermost layer of the arteries. They interact with glycosaminoglycan which causes difficulty in removal of these fatty streaks. They undergo various metabolic changes including oxidation which causes change in morphology and secures their place in the blood vessel.[3]

Once the fatty streaks are formed, there is ongoing inflammatory process and the release of various inflammatory mediators and the recruitment of scavenger macrophages[18] and monocytes into the lesion thus begetting further inflammation.[4]

The monocytes thus recruited engulf lipoproteins by receptor mediated endocytosis and accumulate lipid and become the giant lipid laden macrophages otherwise called foam cells.

There are various complicating factors behind the development of a complex atheroma. Many fatty streaks remain unmodified without causing any complication throughout life.

Hypercholesterolemia causes LDL-C to accumulate in intima. Matrix interactions cause oxidative reactions of lipoproteins and inflammatory reactions are initiated. Altered lipoprotein particles actively promote inflammation. Chemo attraction by various chemokines including macrophage attractant protein cause increased entry of lipoproteins into the zone.

As the lesion matures smooth muscle cells migrate superficially from the layer of media into the layer of intima where they play an active role in attracting further inflammatory particles and increasing the size of the lesion[5]

The leading cause of mortality in diabetes is complications due to atherosclerosis. With changing lifestyle and adaptation of unhealthy food practices these are considered as an epidemic now.

Diabetic dyslipidemia, the term used to describe abnormal lipid profile in type 2 diabetes mellitus is the key factor behind the atherosclerosis which occurs prematurely in diabetes. Though the LDL levels in diabetes are not significantly altered but the size of the particles and their density is condensed in diabetes which increases atherosclerosis risk Also these patients have reduced LDL and increased triglyceride levels. Hypertension is also commonly prevalent in diabetes and obese individuals with dyslipidemia.[6]

METHODOLOGY

Inclusion criteria

Patients on established coronary artery disease who were on atorvastatin therapy 10 mg for more than 1 year, who developed an ischemic stroke evidenced by CT scan or MRI within 5 years of occurrence of the first coronary event.

Exclusion criteria

- Age less than 40 years.
- Patients on irregular statin therapy.
- Patients with chronic kidney disease.
- Patients with chronic liver disease
- Patients who had poor left ventricle function

Study Subjects

Number of study groups : two

Group 1 CASES: 50 patients with a history of coronary artery disease with ECG or ECHO confirmation and who were on regular atorvastatin therapy 10 mg daily for more than one year who developed a cerebrovascular event in the form of ischemic stroke with CT or MRI Brain evidence within 5 years of occurrence of the first coronary event were included in the study.

Group 2 CONTROLS: A suitable control of 50 patients matching age, sex, smoking, alcohol and diabetes who had coronary artery disease and were also on atorvastatin therapy 10 mg for more than 5 years were included. These patients should have normal CT brain and no prior history suggestive of transient ischemic attacks.

RESULTS

50 patients with a history suggestive of coronary artery disease with ECG or ECHO confirmation and who were on regular atorvastatin therapy 10 mg for more than one year who developed a cerebrovascular event in the form of ischemic stroke with CT or MRI Brain within 5 years of

the occurrence of the first coronary event were included in the study.

Table 1: Age distribution

Age range	Number of persons
40-49	1
50-59	9
60-69	25
70-79	15
Total	**50**

In our study the mean age of the population was 66.4 years. The majority of patients (25) in our study were in the age group 60 to 69 years.

Table 2: Gender distribution

Sex	No. of cases
Males	29
Females	21
Total	**50**

Out of the 50 cases 29 were males and 21 were females.

Table 3: Age sex ratio

Age	Males	Females	Ratio
40-49	1	O	0% female
50-59	6	3	50% FEMALE
60-69	16	9	56.25% FEMALE
70-79	6	9	150 %FEMALES
Total	**29**	**21**	

The age sex ratio indicates that females had later onset of vascular events compared to males. There were 29 males & 21 females. Their average age was 67.72 years; females were somewhat elder to their male counterparts. Most of them belonged to the 60-69 years age group (50%). Youngest male was a 45 years old electrician, who had hyper homo cystinemia. He also had evidence of peripheral arterial disease. He had strong family history of Coronary and cerebro vascular disease.

Table 4: Body mass index

	Cases	Controls
18-22.9	6	10
22.9-24.9	7	17
25-30	33	21
>30	4	2
Total	**50**	**50**

The Body mass index was calculated by the formula. Weight in kg divided by height in cm squares. 88 % of cases and 80 % of controls were having a higher than normal BMI. The results showed that with increasing obesity the prevalence of multiple ischemic events was increasing. An increase in BMI with increasing age was observed. The average BMI among cases was 27.03. SD was 3.42. Average BMI among controls was 25.4. SD was 2.97. The p value was 0.0125 which is considered statistically significant. There was high prevalence of sedentary life style and faulty eating habits. Since both the cases and controls were on atorvastatin therapy they had the wide spread belief that they can adopt these faulty habits. Proper counselling was given to all the 100 patients included in the study.

Table 5: Waist hip ratio in women

In women	Cases	Controls
<o.8	5	7
>0.8	16	14
Total	**21**	**21**

Table 6: Waist hip ratio in men

In men	Cases	Controls
<o.9	7	10
>o.9	22	19
Total	**29**	**29**

66.6 % of women and 75.8% of men were having a higher than normal waist circumference.

DISCUSSION

In our study the mean age of the population was 66.4 years. The mean age of first incidence of a coronary event in our population is 53.2 years and that of CVA is 62.4 years. (meenaksi *et al*)[7]. In India we have early onset of ischemic events due to genetic factors. In our study males were more affected than females (29:21). This has been reported in various studies. Leading examples include studies by Elizabeth *et al*[8]. In the majority of studies the ratio is around 2:1. In our study the ratio is 29:21. The difference in results could be ignored due to small sample size. This could be because of the greater prevalence of metabolic syndrome in females and also because of a slightly elderly population as a study group. Majority of women had attained menopause which removes cardio protective activity of oestrogen. Modifiable and non-modifiable risk factors for ischemic stroke have been identified and include age; gender; race/ethnicity; heredity; hypertension; cardiac disease, particularly atrial fibrillation; diabetes mellitus; hypercholesterolemia; cigarette smoking; and alcohol abuse, hypercoagulable states and patent foramen ovale.[9] Brain or neuronal dysfunction occurs at cerebral blood flow levels of below 50 mg/dL, and irreversible neuronal injury is initiated at levels below 30 mg/dL. Both the degree and duration of reductions in cerebral blood flow are related to the likelihood of sustained neuronal injury. When blood supply is completely interrupted for 30 seconds, brain metabolism is altered. After 1 minute, neuronal function may cease. After 5 minutes of interruption, anoxia initiates a chain of events that may result in cerebral infarction; however, if oxygenated blood flow is restored quickly enough, the damage may be reversible, as with a TIA. The following steps occur in the evolution of an infarct: (1) local vasodilatation and (2) stasis of the blood column, with segmentation of the red

cells, followed by (3) oedema and (4) necrosis of brain tissue. The earliest ischemic changes are visualized by increased water content in diffusion-weighted MRI while with time, an infarct is well delineated by fluid-attenuated inversion recovery (FLAIR) and T2-weighted changes on MRI.[10]

Atherosclerotic plaque that occurs at the site of a bifurcation in any of the larger vessels will cause stenosis progressively, as the final large artery occlusion is because of thrombosis in the lumen that is narrowed. Arteriosclerotic plaques can originate at any site along the carotid artery & the vertebrobasilar system. Yet the most common sites are the bifurcation of the common carotid artery into the external and internal carotid arteries, the origins of the middle and anterior cerebral arteries, and the origins of the vertebral from the subclavian arteries.

An atherosclerotic stenosis or occlusion may also lead to a cerebral infarction through an embolic mechanism. In this case, emboli arising from the proximally situated atheromatous lesions occlude otherwise healthy branches located more distally in the arterial tree.

This particular subtype has various named entities, because of ischemia confined to the territory of a single vessel. They reflect arterial disease of penetrating vessels which supply the internal capsule, basal ganglia, thalamus, corona radiata, and paramedian regions of the brainstem. Disagreements exist about the pathogenesis of lacunar infarcts; some studies consider the use of the term lacunae to describe size and location, without indicating a specific pathology. The pathologies of only a handful of such infarcts have been studied by serial section, and only a few of those studies have documented a tiny focus of microatheroma or lipohyalinosis stenosing one of the deep penetrating arteries. The arterial damage is generally due to long-standing hypertension or diabetes mellitus.

CONCLUSION

In our study males were more affected than females. This has been reported in various studies.

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