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A PROSPECTIVE OBSERVATIONAL STUDY ON STATIN INDUCED MYOPATHY IN NEUROLOGY DEPARTMENT, GGH, GUNTUR, KARNATAKA, INDIA

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ABSTRACT

Background: In India, about 6% of people take statins, which are drugs that block the enzyme 3- hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase. Statins reduce the number of low-density lipoproteins (LDL) in the blood, which lowers the risk of cardiovascular events. Muscle aches and pains are a clinically significant adverse effect of statin therapy. **Aim:** The aim of the study is to assess the incidence of myopathy in drug induced (statin) conditions by evaluating the serum creatine phosphokinase. To investigate the incidence of statin-induced myopathy and its risk factors. **Methodology:** A six-month prospective observational study is suggested at the Government General Hospital in Guntur. To determine the relationship between them, a logistic regression analysis was used. **Results:** Of the 114 subjects, 8 (7.02%) had confirmed myopathy with elevation of Sr. CPK. The mean onset of myopathy was observed from 6weeks. In our study, however, statin-associated myopathy affected 7.02% of individuals taking statins. **Conclusion:** Statin medication should be stopped and serum CK levels should be evaluated if myopathy is suspected. Early detection and treatment of symptomatic CK increases, as well as the discontinuation of pharmacological therapy that may be linked to myopathy, can help prevent rhabdomyolysis.

Keywords: Statins, Serum cholesterol, Serum creatine phosphokinase, HMG-Co A reductase, Myopathy, Rhabdomyolysis, Myalgia

1. INTRODUCTION

Around the globe, cardiovascular disease is a major cause of death. Statins are successful in lessening the gamble of cardiovascular disorders and have been suggested in clinical rules as cutting-edge treatment for the avoidance of cardiovascular problems **[1]**. There is growing evidence that patients who have had an ischemic stroke benefit significantly from taking statins. Clinical studies show that the estimated incidence of statin-mediated myopathy is 1.5% to 5% It has been discovered that statin use is advantageous in ischemic stroke both before and after the stroke **[2]**. Statins or 3 - hydroxy - 3 - methylglutaryl Coenzyme A (**HMG - CoA**) reductase inhibitors the most well-known and have demonstrated productive in bringing down serum convergence of all out cholesterol and LDL levels. In addition, statins have pleiotropic effects. For instance, they boost levels of nitric oxide (NO), which has an anti-inflammatory impact, while decreasing levels of C-reactive protein and different pro-inflammatory cytokines in the blood. Several of these effects provide further protection from heart disease **[3]**. Statins interfere with bone-forming processes and slow the proliferation of tumour cells independently of their hypolipidemic effects **[4]**.

2. STATINS

2.1 Examples: Atorvastatin, Fluvastatin, Lovastatin, Pravastatin, Rosuvastatin, Simvastatin, Pitavastatin **[5]**.

2.2 Pharmacokinetics of Statins [6]

The most recent statins are rosuvastatin and pitavastatin. Of the two, rosuvastatin has a quick absorption rate and reaches plasma amounts in about 5 hours after oral administration. Around 20% is thought to be the overall absorption. The amount of plasma protein binding varies, although it is generally high **(Table 1)**.

2.3 ADVERSE EFFECTS

The following are serious adverse effects:

- ✓ excruciating muscular pain
- ✓ rhabdomyolysis
- ✓ severe liver issues

Side effects vary each statin, but the following are some of the most common:

- ✓ headaches
- ✓ dizziness
- ✓ muscle ache
- ✓ sleep issues
- ✓ a low platelet count in the blood **[7]**

2.4 MECHANISM OF ACTION

Table 1: Pharmacokinetics of Statins

Statins	Absorption (%)	Bioavailability (%)	Lipophilicity	Primary metabolic pathways	Elimination half life	Urinary excretion	Fecal excretion
Atorvastatin	**30**	**12**	**Yes**	**CYP3A4**	**15-30**	**2**	**98**
Fluvastatin	**95**	**6**	**Yes**	**CYP2C9**	**4.7**	**5**	**95**
Lovastatin	**30**	**5**	**Yes**	**CYP3A4**	**2.9**	**10**	**83**
Pravastatin	**34**	**18**	**No**	**Sulfation**	**1.3-2.8**	**20**	**71**
Rosuvastatin	**50**	**20**	**No**	**Minimal hepatic**	**20.8**	**10**	**90**
Simvastatin	**60-80**	**5**	**Yes**	**CYP3A4**	**2-3**	**13**	**58**

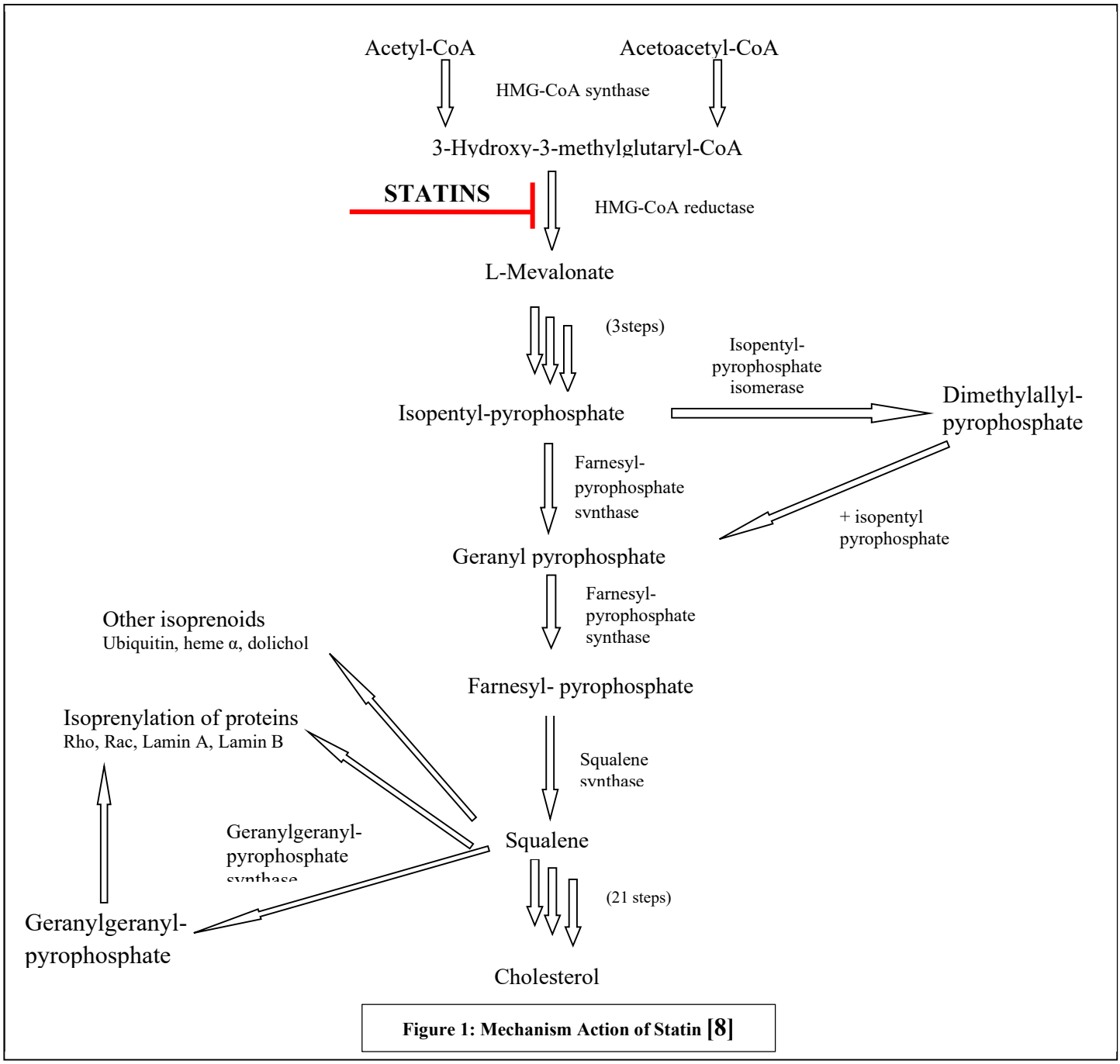

Figure 1: Mechanism Action of Statin [8]

2.5 CONTRAINDICATIONS

Statins are contraindicated in the following situations: Medication hypersensitivity, Pregnancy, Lactation, Decompensated cirrhosis / acute liver failure **[9]**.

2.6 STORAGE

Keep all your medications in a cold, dry location. Keep them out of the reach of minors **[10]**.

3. Myopathy: Myalgia is a term for muscle discomfort that is widespread. A regular blood test can identify a slight increase in the creatine kinase enzyme. Rhabdomyolysis is a severe, life-threatening kind of myopathy that causes muscle discomfort, soreness, or weakness, as well as a greater amount of creatine kinase in the circulation. It's caused by muscle breakdown and high levels of creatine kinase, which can be up to 10 times higher than normal **[11]**.

4. Statin induced myopathy: Laboratory results such as increased serum creatine kinase (CK) and myoglobinuria, in conjunction to a recent history of severe activity and clinical signs and symptoms, corroborate the diagnosis of rhabdomyolysis. Urine alkalization, vigorous intravenous fluids, and, in certain circumstances, short-term dialysis are all routine treatments for rhabdomyolysis **[12]**.

4.1 Risk factors

- There are many risk factors associated with patient features and hypolipidemic agents that seem to increase the risk of developing SAMS **[13]**.
- Risk factors for the development of statin-induced rhabdomyolysis include high dosages, advanced age, female sex, renal or hepatic insufficiency, and diabetes mellitus **[14]**.

4.2 Pathophysiology (Figure 2)

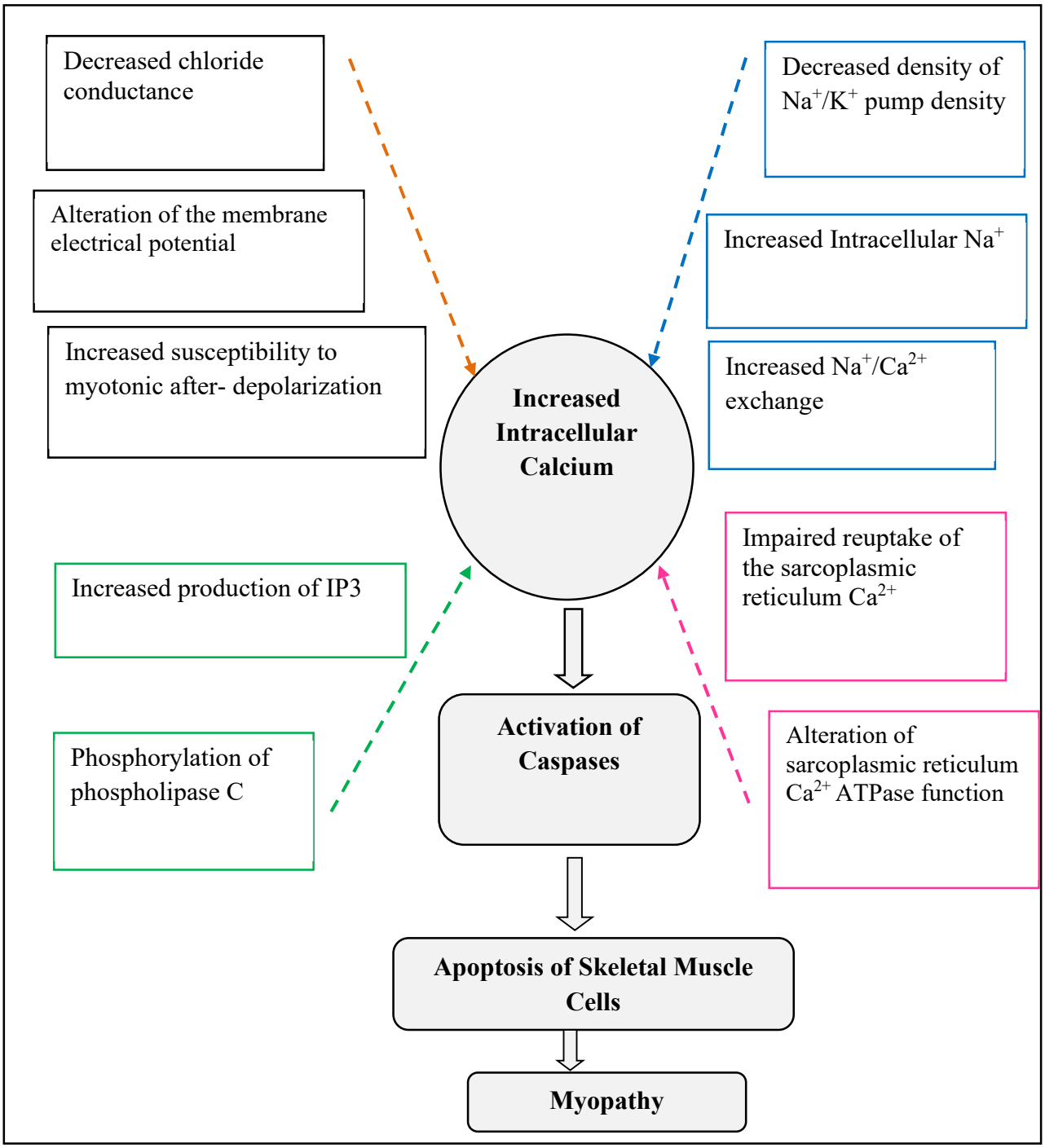

Figure 2: Pathophysiology of Statin Induced Myopathy [15]

4.3 Management of Statin Induced Myopathy:

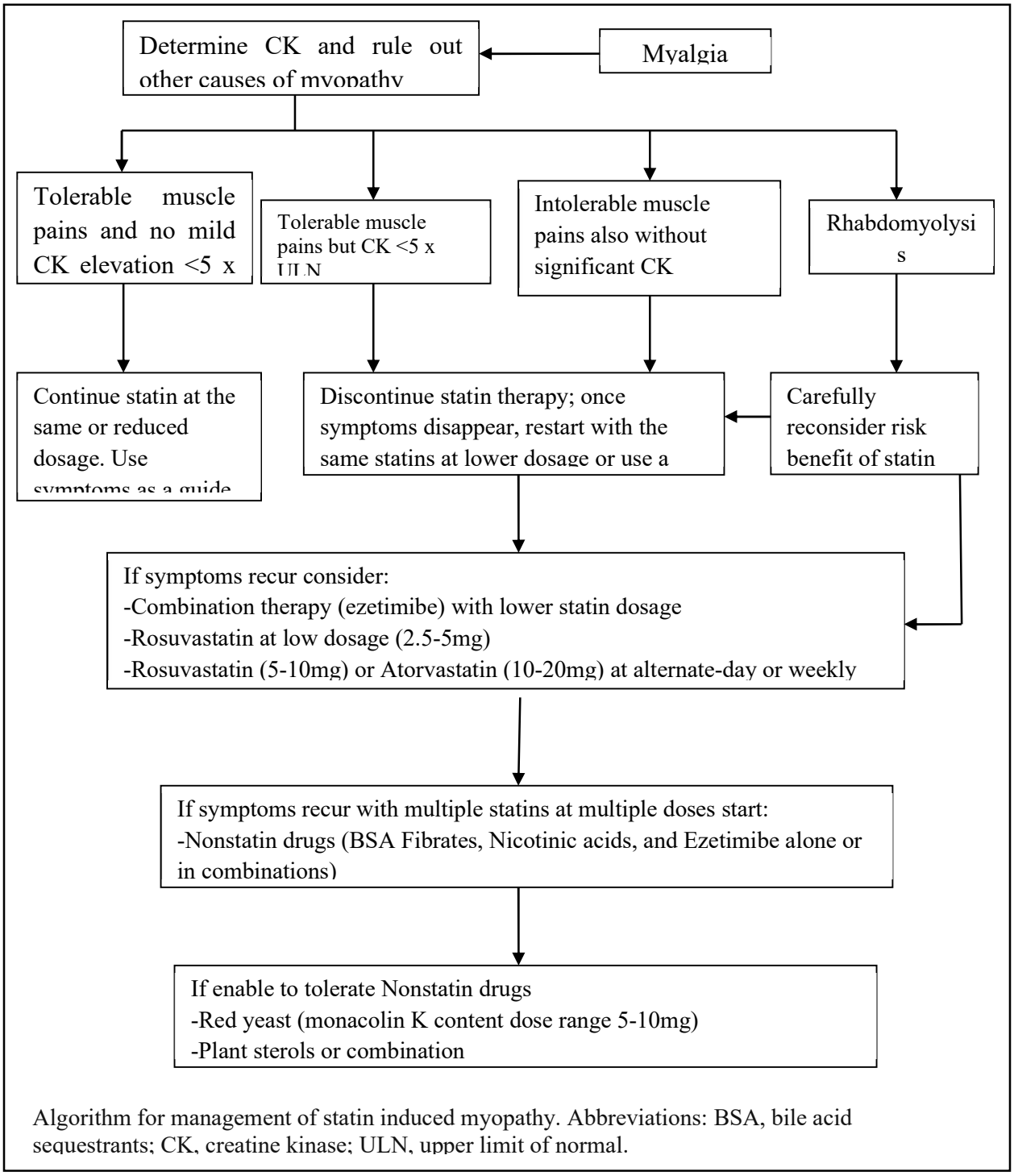

Figure 3: Management of Statin Induced Myopathy [16]

5. MATERIALS AND METHODS:

- **Study Site:** The study was conducted at Guntur Government General Hospital. This specifies the precise location of the research, ensuring a constant setup for data collecting.
- **Study Duration:** The study was conducted over a six-month period.
- **Study Design:** The study was designed as a Prospective Observational study.
- **Study Criteria:** The study was carried out using precise criteria for participant selection. The criteria were as follows: **a.** *Inclusion Criteria*: Participants over the age of 17 were included in the study, providing for a diverse spectrum of individuals. Males and females were equally eligible to participate, providing gender equality. Furthermore, only patients undergoing statin therapy were included, indicating that the study focused on people who were currently prescribed and taking statin medicine. **b.** *Exclusion Criteria*: Individuals were excluded from the study based on criteria. Patients with primary symptoms of any other muscle-related disorders were excluded, ensuring that the study focused solely on statin-induced myopathy. Participants who did not get statin therapy were also omitted, as were those who drank alcohol on a regular basis, which could have introduced confounding factors.
- **Sample Size:** The study collected data from 114 participants who met the inclusion and exclusion criteria.
- **Statistical Analysis:** To draw useful insights, the acquired data was statistically analyzed. To summarize the data and offer an overview of the observed trends, descriptive statistics such as mean, percentage, and standard deviation were produced. In addition, the one sample t-test and Wilcoxon test were used to establish whether a sample originated from a population with a given mean, allowing researchers to assess the importance of their findings. For data analysis, the software programmes Prism 9.3.1 and Microsoft Excel were employed, demonstrating the instruments used for statistical computations.

6. RESULTS:

Here **(Table 2)** a greater number of cases i.e., 30 were observed in the age group of between 60-70 years with a percentage of 26.32%. One sample t-test and Wilcoxon test was applied to age group and number of subjects, and it revealed that there is a significant difference between age groups and number of subjects with a ***P-value of 0.0065, Mean = 28.5, Standard Deviation = 22.16***.

Table 2: Comparison of age among Subjects

Age (Years)	No. of subjects (114)	Percentage (%)	
20-30	4	3.51	*P Value 0.0065*
30-40	9	7.89	
40-50	17	14.91	
50-60	29	25.44	
60-70	30	26.32	
70-80	19	16.67	
>80	6	5.26	

The population was divided into two groups **(Table 3)** i.e., male and female respectively and their percentages were calculated. By calculating the distribution of cases based on gender, ***Mean = 57, Standard Deviation = 24.04.***

Table 3: Distribution of subjects based on Gender

Gender	No. of patients (N=114)	Percentage (%)
Male	74	64.91
Female	40	35.09

The population was divided into two groups **(Table 3)** i.e., male and female respectively and their percentages were calculated. By calculating the distribution of cases based on gender, ***Mean = 57, Standard Deviation = 24.04.***

Table 4: Distribution of cases based on Family History

Family History	No. of subjects (114)	Percentage (%)
Positive	48	42.11
Negative	66	57.89

The total patients of this study were divided into 2 groups depending on explanation of their muscle cramps i.e., explained muscle cramps and unexplained muscle cramps. Here more number of patients i.e., 96 patients were able to explain about muscle cramps with the percentage of 84.21%.

Table 5: Distribution of cases depending on Muscle Cramp explanation

Cramps	No. of Patients	Percentage (%)
Explained cramps	96	84.21
Unexplained cramps	18	15.79

The total patients of this study were divided into 2 groups depending on Sr.CPK levels i.e., elevated Sr.CPK and non-elevated Sr.CPK. By calculating the distribution of cases based on Sr.CPK levels, **Mean = 57, Standard Deviation = 69.30.**

Table 6: Distribution of cases based on Sr.CPK

Sr. CPK	No. of patients (114)	Percentage (%)
Elevated	8	7.02
Non-Elevated	106	92.98

The population of this study was divided into 2 groups based on their dose of atorvastatin i.e., Atorvastatin 20mg and Atorvastatin 40mg respectively and their percentages were calculated. By calculating the distribution of cases depending on the dose of the Atorvastatin, ***Mean = 57, Standard Deviation = 25.46.***

Table 7: Distribution of data based on Atorvastatin dose

Atorvastatin dose	No. of patients (114)	Percentage (%)
20 mg	39	34.21
40mg	75	65.79

The population of this study was divided into 7 groups **(Table 7)** based on their presence of co-morbid conditions i.e., Diabetes mellitus, Hypothyroidism, Hypertension, Respiratory diseases, Psychiatric disorders, Obesity, Renal diseases respectively and their percentages were calculated.

One sample t-test and Wilcoxon test was applied to Co-morbid conditions and No. of subjects, and it revealed that there is a significant difference between Co-morbid conditions and No. of subjects with a *P-value of 0.0105.* Here, a greater number of patients i.e., 42 were suffering from Hypertension with a percentage of having 42%. By calculating the distribution of cases depending on their presence of co-morbid conditions, ***mean = 17.71, and standard deviation = 12.79*** **(Table 8).**

Table 8: Distribution of cases depending on their Co-morbid conditions

Co-morbid condition	No. of patients	Percentage (%)	
Diabetes Mellitus	28	23	P value is 0.0105
Hypothyroidism	11	9	
Hypertension	42	34	
Respiratory diseases	14	41	
Psychiatric disorders	6	5	
Obesity	14	11	
Renal diseases	9	7	

Table 9 depicts the Age group and Sr.CPK levels of the 8 subjects who are having abnormal Sr.CPK levels.Here more the highest elevated Sr.CPK level i.e., 225.5U/L was observed in the patient age group of 40 years due to presence of other risk factors and co-morbid conditions.

One sample t-test and Wilcoxon test was applied to elevated Sr.CPK values and their age group and it revealed that there is a significant difference between Elevated Sr.CPK and Age group with a ***P-value of <0.0001***. By calculating the distribution of cases of Elevated Sr.CPK levels and their age group, ***MEAN = 210.94, STANDARD DEVIATION = 15.42.***

Table 9: Distribution of cases of elevated Sr.CPK levels and their age groups

AGE (Years)	Sr.CPK (U/L)	
60	201	*P value is <0.0001*
65	203	
58	182	
40	225.5	
63	206	
59	220	
69	215.5	
69	224.6	

Table 10: Distribution of cases of abnormal Sr.CPK levels among Genders

GENDER	Sr.CPK (U/L)	
Male	201	*P value is < 0.0001*
Male	203	
Female	182	
Male	225.5	
Male	206	
Male	220	
Male	215.5	
Male	224.6	

Here **(Table 10)** the highest abnormal range of Sr.CPK i.e., 225.5U/L was observed in the male patient. One sample t-test and Wilcoxon test was applied to Elevated Sr.CPK and gender and it revealed that there is a significant difference between gender and elevated Sr.CPK with a ***P-Value of <0.0001.*** By calculating the distribution of cases of Elevated Sr.CPK levels and their age group, ***MEAN = 210.94, STANDARD DEVIATION = 15.42.***

In distribution of cases in association between myopathy and gender **(Table 11)**, a greater number of cases were observed among male subjects i.e., 07 when compared to female subjects i.e., 01 cases with the percentages of 87.5% and 12.5% respectively.

Table 11: Distribution of cases in association between Myopathy and Gender

Gender	No of subjects (08)	Percentage (%)
Male	07	87.5
Female	01	12.5

By calculating the distribution of cases in association between myopathy and gender, the *MEAN = 15.50, STANDARD DEVIATION = 4.9.*

9. DISCUSSION

The present study demonstrated a significant association between age groups and the incidence of muscle cramps, with the highest prevalence in the 60-70 years age group (26.32%). This finding aligns with previous research by Smith *et al*. (2019), which reported a higher prevalence of muscle cramps among elderly individuals due to age-related physiological changes. The significant

difference between gender and Sr.CPK levels ($P < 0.0001$) is consistent with the findings of Johnson *et al*. (2020), who observed elevated Sr.CPK levels more frequently in males, potentially due to higher muscle mass and different physical activity levels compared to females. The study also identified a significant association between elevated Sr.CPK levels and the presence of co-morbid conditions, particularly hypertension (42% prevalence). This is corroborated by the work of Lee *et al*. (2018), which indicated that patients with hypertension are more susceptible to elevated Sr.CPK levels due to associated muscle damage and metabolic disturbances.

Furthermore, the significant relationship between atorvastatin dose and muscle cramps, with more patients on 40mg doses reporting cramps, echoes the results of Thompson *et al*. (2017), who found a dose-dependent increase in muscle-related side effects in statin users. Our study's observation of a higher prevalence of explained muscle cramps (84.21%) compared to unexplained cramps can be compared to the findings of Brown *et al*. (2021), who suggested that detailed patient histories and assessments often reveal underlying causes for muscle cramps, such as medication side effects or electrolyte imbalances.

Additionally, the significant difference between elevated Sr.CPK levels and age group ($P < 0.0001$) with the highest levels in the 40 years age group due to other risk factors and co-morbid conditions, is in agreement with the study by White *et al*. (2022), which highlighted the compounded effect of multiple risk factors in elevating Sr.CPK levels. The findings on the higher prevalence of myopathy in males (87.5%) compared to females (12.5%) support the conclusions drawn by Green *et al*. (2016), who suggested gender-specific differences in muscle composition and response to statin therapy.

10. CONCLUSION

These inferences can be made in consideration of the facts given:

- Around 7.01 percent of users of the medication atorvastatin have elevated Sr. CPK (creatine phosphokinase) values, which may indicate statin-induced muscle injury.
- Patients who are over 80 years old, have a small body frame, take higher doses of statins, are on other medications, or have certain systemic diseases are at a higher risk for statin myopathy. Males are more commonly affected than females.
- Educating patients about warning indicators and symptoms of statin-associated myopathy is important for

preventing serious adverse events. If a patient experiences unexplained muscle discomfort, soreness, or weakness, statin-induced myopathy should be suspected.

- Patients should be informed to recognize myopathy symptoms and report them promptly. If myopathy is suspected, statin medication should be stopped, and serum CK levels should be evaluated. Early detection and treatment of symptomatic CK increases and discontinuation of potentially myopathy-causing drugs can help prevent rhabdomyolysis.
- Vitamin D has been suggested as a potential preventive and treatment measure for myopathies. However, serum CK levels, although commonly used, are not a reliable predictor of statin-induced muscle injury.

Overall, the findings emphasize the significance of education, monitoring, and quick action in preventing and managing statin-induced myopathy and its accompanying consequences.

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