PRESCRIPTION PATTERN OF STATINS IN CORONARY ARTERY DISEASE: A REVIEW

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ABSTRACT

Statins helps to reduce cardiovascular events in patients with cardiovascular disease. This review was undertaken to examine the current evidence for the effect of statins in patients with coronary artery disease. Statins are effective in both primary and secondary prevention of coronary heart disease (CHD), and other conditions. The present study is mainly focused on prescription pattern of statins in coronary artery disease. The different statins prescribed are Atorvastatin, Rosuvastatin, Simvastatin and Lovastatin. Statins are first-line agents in most cardiovascular disease situations.

KEY WORDS

Cardiovascular disease, Cholesterol, Secondary Prevention, Statins.

INTRODUCTION

A safe and effective drug therapy mostly is possible only when patients are well informed about the medications and their use. Every member of the healthcare team should practice rational drug therapy. Rational drug use means patients receive medications appropriate to their medication, in doses that meet their own individual requirements, for an adequate period of time and at the lowest cost to them and their community. Lack of knowledge about drugs, unethical drug promotions and irrational prescribing habits of clinician's leads to irrational prescribing of drugs. (1). Accurate diagnosis, proper prescribing, correct dispensing, suitable packing and patient adherence are the five important criteria for rational prescribing of drugs (2). Elevated cholesterol levels are a proven risk factor for cardiovascular diseases (CVDs) including coronary artery disease. Observational studies give an idea about the relationships between increased cholesterol and mortality, CVD, and decreased quality of life. The 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors (Statins) reduce atherogenesis (3). Prescribing patterns and drug utilization studies help the prescribers to create awareness about the problems and to provide feedback about rational use of drugs (4). Therefore, the study on prescribing pattern definitely improve the quality of prescription writing, so study of drug prescribing pattern is relevant in the present scenario (5).

CLASSIFICATION OF STATINS

- Statins are classified according to the below criteria
- How they are obtained
- Liver metabolism
- Physiochemical properties
- Specific activity (6).

EFFECTS OF STATINS ON PATIENTS WITH CORONARY DISEASE

Statins are responsible for reducing serum low density lipoprotein by inhibiting 3-hydroxy –methyl glutaryl coenzyme A (HMG CoA reductase). Statin therapy before percutaneous coronary intervention (PCI) has been shown to reduce the incidence of periprocedural myocardial necrosis and to improve outcomes. Intensive lipid-lowering therapy with statins not only improves survival rates and clinical outcomes but also

reduces the progression of atherosclerosis a meta-analysis showed that statin pretreatment in patients with stable angina resulted in a relative reduction in

procedural myocardial necrosis and overall major adverse cardiac events (7).

Table 2: Statin Equivalent Dosages

% LDL Reduction (approx.)	Atorva-statin	Fluva-statin	Lovastatin	Prava-statin	Rosuvastatin	Simva-statin
10–20%	—	20 mg	10 mg	10 mg	—	5 mg
20–30%	—	40 mg	20 mg	20 mg	—	10 mg
30–40%	10 mg	80 mg	40 mg	40 mg	5 mg	20 mg
40–45%	20 mg	—	80 mg	80 mg	5–10 mg	40 mg
46–50%	40 mg	—	—	—	10–20 mg	80 mg*
50–55%	80 mg	—	—	—	20 mg	—
56–60%	—	—	—	—	40 mg	—
* 80 mg dose no longer recommended due to increased risk of rhabdomyolysis						
Starting Dose						
Starting dose	10–20 mg	20 mg	10–20 mg	40 mg	10 mg; 5 mg if hypothyroid, >65 yr, Asian	20 mg
If higher LDL reduction goal	40 mg if >45%	40 mg if >25%	20 mg if >20%	—	20 mg if LDL >190 mg/dL (4.87 mmol/L)	40 mg if >45%
Optimal timing	Anytime	Evening	With evening meals	Anytime	Anytime	Evening

Source: Wikipedia, 2012b.

STATINS: MECHANISM OF ACTION

HMG CoA reductase inhibitors competitively inhibit the activity of HMG CoA reductase, the rate-limiting enzyme in cholesterol synthesis which results in to active site and produces the action. (8)

INHIBITING CHOLESTEROL SYNTHESIS

Statins inhibit HMG CoA reductase there by inhibit the pathway for synthesizing cholesterol in liver.Statins with short half

lives are taken at night to maximize their effect. (9)

OTHER EFFECTS

As noted above, statins exhibit action beyond lipid-lowering activity in the prevention of atherosclerosis.

1. Improve endothelial function
2. Modulate inflammatory responses
3. Maintain plaque stability
4. Prevent thrombus formation. (10)

CONCLUSION

CAD is markedly more common in men than in women. Hypertension and diabetes were commonly associated with coronary artery disease. The prescribing pattern can be improved by reducing the

number of drugs per prescription. The study of prescription pattern provides an important framework for necessary modifications required to achieve decreased cellular concentration. Statins have similar structure of HMG CoA, which fits in patient oriented, cost effective and rational prescriptions.

REFERENCE

1. Pharmacy communication In Hassan WE editor Hospital Pharmacy Lea and febiger, Phildelpia, 5th edition 1985; 154-1594.
2. The rational use of drugs: report of the conference of experts. Nairobi, 25-29 November 1985. Geneva, world health organization, 1987.
3. Edward J. Mills, Beth Rachlis, Ping Wu, Philip J. Devereaux, Paul Arora, Dan Perri. *Journal of the American College of Cardiology.* 2008; 52(22):1769-81.
4. Pradhan SC, Shewade DG, Shashindren CH, Bapna IS. Drug utilization studies. National Med J India. 1988; 1: 185-189.
5. Shah B, Mathur P., Surveillance of cardiovascular disease risk factors in India: the need and scope, Indian Journal of Medical Research, 2010; 132: 634 - 42.
6. Statins: mechanism of action and effects, NicolaeSimionescu" Institute of Cellular Biology and

Pathology, Bucharest, Romania. J.Cell.Mol.Med. Vol 5, No 4, 2001 pp. 378-387.

7. World Health Organization, Cardiovascular diseases, Available at: http://www.who.int/mediacentre/factsheets/fs317/en/index. html, Accessed February 2009.

8. Cilla DD, Gibson DM, Whitfield LR, Sedman AJ (July 1996). "Pharmacodynamic effects and pharmacokinetics of atorvastatin after administration to normocholesterolemic subjects in the morning and evening". *Journal of Clinical Pharmacology.*

9. *Miettinen TA (March 1982).* "Diurnal variation of cholesterol precursors squalene and methyl sterols in human plasma lipoproteins". *Journal of Lipid Research. 23 (3): 466–73.*

10. Ridker PM, Danielson E, Fonseca FA, Genest J, Gotto AM, Kastelein JJ, Koenig W, Libby P, Lorenzatti AJ, MacFadyen JG, Nordestgaard BG, Shepherd J, Willerson JT, Glynn RJ (2008).

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