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EDITORIAL

Incidence of Periprocedural Myocardial Infarction and use of Statin

Statins are the main pharmaceutical agents of cholesterol-lowering therapy for years. The lipid reduction results from the inhibition of 3-hydroxy-3-methylglutaryl coenzyme A reductase. During these many years statins delivered much more than the expectations. Still statins are continuing the miracle. These additional beneficial properties of statins are known as pleiotropic effects.[1] These additional properties, appear to be independent of the lipid-lowering effect. The pleiotropic effects of statins include decreased inflammation and increased plaque stability, along with an improvement in endothelial function attributed to increased nitric oxide at the vasculature.[1]

Percutaneous Coronary Interventions (PCIs) are invasive procedures that often result in adverse cardiovascular (CV) events, including increased inflammation, platelet adhesion and plaque instability during the procedure, which may result in elevations in cardiac enzymes or periprocedural MI. Periprocedural MI has been associated with adverse CV outcomes.[2]

Several recent studies have documented the beneficial effects of statins in the acute setting. One study found that patients pretreated with statin for at least 1 week before PCI had a reduced incidence of periprocedural MI, defined as CK-MB greater than three times the upper limit of normal. Another study found a decrease in all-cause mortality, after 30 and 180 days, in patients on statin therapy during the time of PCI. Atorvastatin for Reduction of Myocardial Damage during Angioplasty (ARMYDA) was aimed to determine whether atorvastatin 40 mg daily in statin-naive patients with stable angina, beginning 7 days before elective PCI, could reduce periprocedural MI (defined as CK-MB greater than two times the upper limit of normal). ARMYDA found a lower composite endpoint of death, MI and revascularization in the statin group (5% vs. 18%; P=.025).[3]

Another study, Novel Approaches for Preventing or Limiting Events II (NAPLES II) trial found a reduction in periprocedural MI after a single dose of atorvastatin 80 mg, up to 24 hours before PCI. NAPLES II enrolled patients with stable angina undergoing elective stenting procedures. Patients were randomly assigned to receive atorvastatin 80 mg or no statin treatment on the day before PCI. The incidence of periprocedural MI (CK-MB elevation greater than three times the upper limit of normal) in the atorvastatin group was 9.5% vs. 15.8% in the control group (OR=0.56; 95% CI, 0.35-0.89).[4]

The ARMYDA-ACS trial aimed to evaluate the effects of a loading dose of atorvastatin in statin-naive patients with non-ST segment elevation acute coronary syndrome. The randomized, placebo-controlled trial investigated whether an 80-mg loading dose of atorvastatin 12 hours before angiography, along with an additional 40-mg dose 2 hours before PCI, would reduce 30-day incidence of major adverse CV events. ARMYDA-ACS found that the 30-day incidence of major adverse CV events occurred in 5% of the statin group vs. 16.5% in the control group (P=.01).[5]

All these demonstrated reduction in periprocedural MI in statin-naive patients, but most patients who require PCI are already taking statins. Previous studies showing that statins lose their protective anti-inflammatory effects after as little as 1 or 2 weeks of use in animal models.[6] However, the loss of protection due to long-term statin therapy may be overcome by an acute loading dose.

The ARMYDA investigators studied again whether an 80 mg loading dose of atorvastatin 12 hours before angiography along with an additional 40 mg dose 2 hours before PCI would lower 30 day incidence of major adverse CV events, in patients with stable angina and acute coronary syndrome (ACS) who were receiving chronic statin therapy for at least 30 days before enrollment in the study. ARMYDA-RECAPTURE, found a significant decrease in 30-day major adverse CV events (3.7% vs. 9.4%; P=.037). This result was due to the reduction in periprocedural MI.[7]

These studies provide a growing body of evidence for statin use beyond the traditional cholesterol-lowering indication. These findings may support the indication of administration of high-dose statins in patients with acute coronary syndromes and stable angina who are treated with an invasive strategy. However judicious use of statin

is always mandatory, though the drug delivered beyond its expectations but it is not wise to use it like mixing it in flour and serve the population to prevent ischemic heart disease like iodized salt preventing iodine deficiency.

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