Olmesartan may play a role in reducing coronary plaque

Author: Amy Proal

5 Dec 2008

Not long ago I reported on the results of multiple studies, the results of which indicate that the class of medications known as Angiotensin Receptor Blockers or ARBs have the potential to ameliorate a variety of cardiovascular conditions.

Today another study joins the list - this one conducted by Atsushi Hirohata, M.D, Ph.D and his team at the the Sakakibara Heart Institute of Okayama in Japan. Hirohata, who presented his findings at last week's 20th annual Transcatheter Cardiovascular Therapeutics (TCT) scientific symposium, presented study data, which strongly suggests that Olmesartan may play a role in reducing coronary plaque.

The trial, "Impact of olmesartan on progression of coronary atherosclerosis; evaluation by IVUS [OLIVUS]," was performed on 247 angina patients with native coronary artery lesions. Angina is chest pain or discomfort that occurs when the heart muscle does not get enough blood. Patients were randomly assigned to receive 20-40mg/day of Olmesartan or a placebo. Then, depending on the guidance of their individual physicians, they were treated with a combination of beta blockers, calcium channel blockers, diuretics, nitrates, glycemic control agents and/or statins.

Subjects underwent examination by a technique know as Serial Intravenous Ultrasound (IVUS) which allowed the research team to assess the amount of coronary plaque before and 14 months after the start of Olmesartan administration. At the trial's onset, patient characteristics and all IVUS measurements were identical between the two groups. However, after 14 months of treatment, IVUS showed significant decreases in measurements of plaque volume in the Olmesartan group, despite the fact that subjects displayed similar blood pressure readings. In addition, a multivariate analysis ruled out the other forms as treatment (the beta blockers, statins, etc.) as the source of the decrease in plaque, confirming that Olmesartan administration was indeed one of the factors responsible for the decrease in plaque volume.

"Management of plaque is a key front in the war on sudden heart attack," states Hirohata. "These results suggest a positive role in potential plaque regression through the administration of Olmesartan, an angiotension-II receptor blocking agent, for patients with stable angina pectoris."

Hirohata's results are welcome news for patients on the Marshall Protocol (MP) - a novel treatment that uses Olmesartan in concert with carefully chosen pulsed, low-dose antibiotics to eliminate chronic pathogens that are increasingly implicated in inflammatory disease. Cardiovascular diseases are included in this category. "Many years ago, atherosclerosis was thought to be related to lipids and to the excessive deposit of cholesterol in the arteries," states Luigi Fontan, MD, PhD, assistant professor of medicine at Washington University in St. Louis and an investigator at the Instituto Superiore di Sanita, Rome, Italy. "Nowadays, it's clear that atherosclerosis is an inflammatory disease.

But why the reduction in plaque formation? According to biomedical researcher Trevor Marshall, macrophages begin to clump together at injured areas along the blood vessel wall in people with heart disease. Since macrophages are meant to be extremely active immune cells that engulf and kill invading pathogens, Marshall views aggregation of these listless white blood cells in individuals with cardiovascular disease as a sign that the immune system is not functioning

optimally. In his opinion, the reason that the white blood cells are weakened in the first place is because they are infected by chronic bacteria. If these bacteria kill their host cells, dead macrophages may eventually accumulate and form part of the plaque that perpetuates cardiac disease - a perspective supported by the fact that Streptococcus was recently identified in arterial plaque. If the immune system were able to function correctly, it might be able to clear away developing plaque, but “When the immune system is compromised, it simply can’t clear away the obstruction,” states Marshall.

Enter Olmesartan. MP patients take 40 mg of Olmesartan four times a daily. Molecular modeling data shows that Olmesartan is a potent Vitamin D Receptor (VDR) agonist, meaning that it binds and activates the receptor. Because the VDR controls expression of the bulk of the body’s antimicrobial peptides - proteins that serve as natural antibiotics- VDR activation greatly stimulates the innate immune response. This means that the innate immune systems of people taking Olmesartan may regain the ability to recruit active monocytes and other white blood cells to areas where they can clear away dead or dying macrophages, keeping them from becoming part of arterial plaque.

Regardless of whether Marshall’s hypothesis is right or wrong, Hirohata’s study adds yet another benefit to what’s become a laundry list of positive outcomes associated with Olmesartan administration. Let’s hope that the medical community takes notice!