

fasting plasma glucose level between 100.8 mg/dL and 126 mg/dL.

Sixty-six were randomized to treatment with aliskiren and 75 were assigned to treatment with irbesartan. The average blood pressure at baseline was 156/94 mm Hg in the aliskiren group and 154/92 in the irbesartan group.

During the first 2 weeks of treatment, patients received either 150 mg of aliskiren once daily or 150 mg irbesartan once daily. After 2 weeks, the daily dosage in both arms was doubled to 300 mg once daily, and that dosage was maintained for an additional 10 weeks.

After a total of 12 weeks of treatment, blood pressure was cut by an average of 13.8/7.1 mm Hg in the 66 aliskiren-treated patients who completed the study and an average of 5.8/2.8 mm Hg in the 72 irbesartan-treated patients who finished the study. The differences in average reduction in both systolic and diastolic blood pressure were statistically significant. The percent of patients reaching the goal blood pressure of less than 135/85 mm Hg was 29% in the aliskiren group and 17% in the irbesartan group, a statistically significant difference.

Both treatments were generally well

tolerated, with no serious adverse events in either arm. Neither drug was associated with a significant change in blood glucose or lipid profile and neither led to hyperkalemia or an increase in serum creatinine or blood urea nitrogen.

The study tracked changes in the levels of several biomarkers of inflammation, thrombosis, fibrosis, and oxidative state. No differences were seen between the two drugs for any of these, except that aliskiren therapy led to greater reductions of renin-system biomarkers, and irbesartan raised the level of eotaxin, an inflammatory cytokine. ■

Aspirin No Aid For Metabolic Syndrome

BY MITCHEL L. ZOLER

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NEW ORLEANS — Apparently healthy people with a family history of coronary artery disease who also had metabolic syndrome showed elevated platelet aggregation and reduced platelet responsiveness to aspirin in a study of more than 2,000 people.

These findings suggest that “low-dose aspirin therapy alone may not be sufficient to provide optimal antiplatelet protection” in people with metabolic syndrome and an increased risk for coronary artery disease, Dhananjay Vaidya, Ph.D., and his associates reported in a poster at the annual scientific sessions of the American Heart Association. The link between metabolic syndrome and aspirin resistance in platelets was examined because metabolic syndrome is known to be proinflammatory and prothrombotic, they said.

The study involved 2,088 apparently healthy siblings, sibling offspring, and co-parents of the sibling offspring of more than 500 patients who were younger than 60 years and hospitalized for coronary artery disease. The average age of the relatives was about 44 years, and about 58% were women. The group included 591 people (28%) who met the criteria for metabolic syndrome of the Adult Treatment Panel III guidelines of the U.S. National Cholesterol Education Program; the remaining 1,497 people (72%) did not have metabolic syndrome.

After a baseline assessment and blood collection, the subjects were treated with 81 mg/day of aspirin for 2 weeks and then were reassessed and had a second blood specimen drawn. The aggregability of each person's platelets was tested before and after aspirin treatment with two different in vitro assays. In one assay, the platelets were treated with arachidonic acid; in the second, they were treated with urinary thromboxane B₂.

Before starting aspirin, the platelets of the people with metabolic syndrome showed significantly more aggregation in both in vitro assays than the platelets from people without metabolic syndrome in an analysis that adjusted for baseline differences in age, gender, race, serum levels of LDL cholesterol and high sensitivity C-reactive protein, and smoking status, said Dr. Vaidya, a vascular researcher in the department of medicine at Johns Hopkins University, Baltimore, and his associates.

Immediately after 2 weeks of daily aspirin treatment, the platelets of the people with metabolic syndrome continued to show a significantly higher level of aggregation, compared with platelets from those without metabolic syndrome, in both assays, again in an analysis that adjusted for the same baseline differences.

The finding has clinical implications because aspirin prophylaxis for coronary artery disease is recommended for metabolic syndrome, said the researchers. ■

Are you just treating the symptoms of gout?

- The underlying cause of gout is hyperuricemia—a chronic, metabolic disease
- Dietary restrictions alone are not usually adequate to manage hyperuricemia¹
- Pain management with antiinflammatory drugs may block the inflammatory response, but does not address the underlying disorder^{2,3}
- Over time, serum uric acid levels maintained at less than 6 mg/dL with continuous urate-lowering therapy can reduce the risk of gout attacks and disease progression^{1,3}

To learn more about managing hyperuricemia and gout, visit

www.Gout.com