

over, these antibodies recognized  $\beta 2$ -glycoprotein I or  $\beta 2$ -glycoprotein I-cardiolipin complex (8). Patients with antiphospholipid antibodies related to autoimmune diseases show reactivity against this protein, whereas patients with transient antiphospholipid antibodies do not (1).

In conclusion, we have described the association of type I diabetes and APS in a patient who presented a retinal artery occlusion. We consider that APS should be taken into account in type I diabetic patients with thrombotic events because preventive therapy is readily available.

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## Acarbose: In Search for its Real Indications in Current Medical Practice

Controversy exists regarding what drug to use as the next therapeutic step for patients with NIDDM when diet alone does not achieve adequate glycemic control or what drug to add for patients not controlled by a single drug or insulin treatment. When two drugs are similarly effective, the choice between them is determined by adverse effects and costs.

Recently, several articles have appeared regarding the benefits of acarbose in clinical practice. Chiasson et al. (1) compared acarbose against placebo in a large group of patients with well-controlled diabetes (mean glycosylated HbA<sub>1c</sub> <8%) by diet alone, oral hypoglycemic agents, or insulin. The addition of acarbose only modestly improved the blood glucose control during 1 year of therapy. This was associated with gastrointestinal side effects in a large number of patients. The authors conclude that acarbose improved glycemic control in patients with NIDDM regardless of concurrent hypoglycemic medications. Coniff et al. (2) studied the safety and efficacy of three doses of acarbose compared with placebo in NIDDM patients on dietary therapy alone. Acarbose significantly reduced HbA<sub>1c</sub> levels and postprandial hyperglycemia. However, patients had an average HbA<sub>1c</sub> <9%, and after 16 weeks

of treatment, HbA<sub>1c</sub> levels were similar in both groups. In the July issue of *Diabetes Care*, Coniff et al. (3) presented the results of using acarbose in conjunction with diet and insulin therapy in insulin-requiring type II diabetic patients. Treatment with acarbose was associated with significant reductions in HbA<sub>1c</sub> and in total daily insulin dose. It must be considered that the main HbA<sub>1c</sub> at entry to the study was <7%. Gomez Pérez et al. (4) reported in 1992 significant reductions in fasting and postprandial glycemia but not significant differences in HbA<sub>1c</sub> in poorly controlled NIDDM patients who had acarbose added to sulfonylureas compared with placebo.

From these studies, we may conclude that treatment with acarbose is a safe and effective adjunct to dietary therapy for the treatment of NIDDM and has a low risk of serious side effects (particularly hypoglycemia, weight gain, and/or hyperinsulinism) when compared with insulin or sulfonylureas. Novel therapeutic approaches that may improve metabolic control and prognosis in diabetic patients are always welcome. Acarbose, an inhibitor of intestinal  $\alpha$ -glucosidase, impairs carbohydrate digestion and delays and partially prevents absorption of carbohydrates. The main advantages of acarbose are related to a diminished postprandial increment in blood glycemia with diminished insulin requirements and overall better glycemic control.

Many questions remain regarding the clinical use of acarbose. Can it help obese NIDDM patients in poor metabolic control as a first-line drug? Is it worthwhile to add another drug to reduce postprandial glycemia and hyperinsulinism when patients are in good metabolic control? Are the gastrointestinal effects severe enough to produce low adherences during long-term therapy? Can adding acarbose be useful in patients with secondary failure to oral agents before starting them on insulin? Hopefully, this last question will be answered with a study that is currently being carried out in our department.

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## Reply to Selby et al.

In the April issue of *Diabetes Care*, the article, "Risk factors for lower extremity amputation in persons with diabetes," by Selby et al., discusses risk factors that are evaluated in the database, including smoking status, serum cholesterol and creatinine, blood pressure, height, weight, obesity, and other complications such as neuropathy, stroke, retinopathy, proteinuria, and myocardial infarction. Peripheral vascular disease and its relation to smoking is mentioned in the discussion. However, no data were presented for evidence of peripheral vascular disease. I found no reference to symptoms (claudication), signs (decreased pulses), and/or noninvasive testing (Doppler pressures and wave forms, and Tc Po<sub>2</sub> values) in this article.

Numerous articles, old and new, have discussed the importance of the assessment of the peripheral vascular status to foot salvage. The references listed below are only a small sample from a vast amount of literature on this important subject. There are diabetic patients in whom detection of surgically treatable peripheral vascular disease might lead to foot salvage or permit more distal surgical amputations.

Vascular evaluation is an essential component of assessment of diabetic lower-extremity disease. Clinicians should be aware of the relevance of peripheral vascular disease to lower-extremity amputations.

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