Recommendations for Management of Diabetes During Ramadan

Response to Elhadd and Al-Amoudi and to Davidson

We thank Elhadd and Al-Amoudi (1) for their comments and interest in our article (2). Like them, we are also concerned by the very high rate of severe hypoglycemia and hyperglycemia in patients with diabetes who fast during Ramadan. We agree with them that patients with renal disease may have increased risk of hypoglycemia and that adolescent patients with poor glycemic control or recurrent hypoglycemia may also represent high-risk patients for developing hypoglycemia during fasting.

We thank Davidson (3) for his remarks. Our intent in recommending the addition of complex carbohydrates to a mixed meal at predawn was to keep a sustained increase in the appearance of glucose in the circulation to avoid hypoglycemia. We agree that initiation of hydrolysis of carbohydrates and the rate of appearance and the level of glucose soon after ingestion of simple or complex carbohydrates are fairly similar (4,5). However, these studies suggest that following the ingestion of complex carbohydrates, the day-long glucose concentrations (4) and the area under the curve for glucose (5) are larger for complex carbohydrates. Similar to these findings, Wolsdorf et al. (6) found that ingested uncooked starch behaves as a reservoir for continuous release of glucose compared with the absorption of ingested dextrose that occurs over a shorter period of time. Finally, and most importantly, ingestion of simple carbohydrates in the absence of additional protein or fat at Ifatr (the breaking of the fast) enables rapid absorption of glucose when blood glucose levels are apt to be at their nadir, levels that could explain the relatively higher rates of hypoglycemia in the pre-Iftar period (7).

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The Effect of Rosiglitazone on Overweight Subjects With Type 1 Diabetes

Response to Strowig and Raskin

The recent report by Strowig and Raskin (1) raises the intriguing issue as to whether some type 1 diabetic patients may benefit from a supplementary insulin sensitization approach to their management. As our and other studies have shown that an estimate of insulin sensitivity (eGDR) is strongly predictive of mortality (2), coronary artery disease events (3), coronary calcification (4), and overt nephropathy (5) in type 1 diabetes, we would strongly endorse further pursuit of this approach.

The eGDR measure is based on a regression equation (with terms for waist-to-hip ratio, hypertension status, and HbA_{1c}, i.e., eGDR = 24.39 − 12.97 [waist-to-hip ratio] − 3.39 [hypertension] − 0.60 [HbA_{1c}]) derived from 24 hyperinsulinemic-euglycemic clamp studies and has an r^2 of 0.63 for measured glucose disposal rate (6). As eGDR might therefore be a useful identifier of those who would benefit from thiazolidinedione therapy, it would be helpful to know if eGDR predicted response to rosiglitazone therapy in terms of HbA_{1c} in the Strowig and Raskin (1) study. In addition, was there any difference in change of waist circumference (or waist-to-hip ratio) by treatment group, consistent with the observation (7) that weight gain with rosiglitazone is mainly peripheral rather than central? Finally, it is notable that lipid concentrations were not generally affected by rosiglitazone therapy in contrast to blood pressure. This is similar to our eGDR studies wherein lipids did not help to predict glucose disposal rate, but hypertension status did (6). Do these dual observations thus suggest that in type 1 diabetes insulin resistance is more strongly linked to blood pressure than to lipids?

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