

tensive colonoscopy screening in patients with diabetes (2). We recently managed two diabetic patients who developed acute renal failure following elective colonoscopy. The clinical presentation and biochemical parameters of these two patients are summarized in Table 1.

Both of the patients described had normal renal function at baseline, yet presented with acute renal failure within a few days following bowel preparation and colonoscopy, thus strongly implicating the bowel preparation in the development of the acute renal failure. Both patients received oral sodium phosphate (OSP) solution for bowel cleansing. OSP promotes colon evacuation by drawing large amounts of water into the colon and has been shown to be more effective and better tolerated than polyethylene glycol (PEG) solution. However, recent studies suggest that some patients given OSP are at risk of renal failure due to acute phosphate nephropathy. In a series of 31 cases of renal impairment with renal biopsies showing deposits of tubular calcium phosphate, the risk was highest among patients with preexisting renal impairment, elderly patients, and patients with hypertension or concurrent use of ACE inhibitor or angiotensin receptor blocker (ARB). In that series, 21 patients presented with acute renal failure, of which 4 had diabetes, with age ranging between 44 and 66 years. In a few patients, acute renal failure was discovered within 3 days of colonoscopy, at which time hyperphosphatemia was documented (3).

The U.S. Food and Drug Administration has recently issued an alert advising against the use of OSP products in patients with kidney disease, impaired renal function or perfusion, dehydration, or uncorrected electrolyte abnormalities. OSP should be used with caution in patients taking diuretics, ACE inhibitors, ARBs, and nonsteroidal anti-inflammatory drugs (NSAIDs) (4). In the recently published consensus document on bowel preparation before colonoscopy (5), there was no specific advice given for patients with diabetes aside from the statement that patients with diabetes have significantly poorer preparations with PEG solution than those without diabetes. Patients with diabetes often have reduced renal perfusion despite normal serum creatinine. Incipient diabetic nephropathy is marked by the presence of microalbuminuria, a powerful predictor of subsequent diabetic nephropathy. Our experience suggests that patients with diabetes and

normal renal function tests may be at increased risk of acute phosphate nephropathy after taking OSP. Clinicians should consider avoiding the use of OSP in patients with diabetes undergoing colonoscopy. Use of an osmotically balanced cleansing agent that does not cause significant shift of fluid and electrolytes, such as PEG, is likely to be a safer alternative (6). For patients receiving drugs that alter electrolyte balance, such as diuretics, ACE inhibitors, or ARBs, it may be prudent to withhold these drugs temporarily before OSP. Close monitoring of hydration status, glycemic control, and renal function is mandatory during the preparation and after colonoscopy in patients with diabetes.

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COMMENTS AND RESPONSES

Is Self-Monitoring of Blood Glucose Appropriate for All Type 2 Diabetic Patients? The Fremantle Diabetes Study

Response to Davis et al.

The analysis of self-monitoring of blood glucose (SMBG) in the community-based observational Fremantle Diabetes Study (1) becomes even more interesting when combined with three other studies of SMBG in type 2 diabetes: the Italian Qualità ed Esito in Diabetologia (QuED) Project (2), analyses of the Kaiser Permanente Northern California Medical Care Program (3), and the German ROSSO Study (4).

All four studies concur that patients using SMBG are younger at diagnosis by 3–4 years (1,2,4). Patients present with higher A1C (mean +0.9%) (4). Even during continuous use of SMBG, mean A1C levels are slightly higher (difference 0.2–0.3%) (2–4) or slightly lower (–0.3%) (1) than in patients not using SMBG (mean of all four studies +0.2–0.3%).

Where, then, is the assumed beneficial impact of SMBG on blood glucose control? Though one cannot see it in cross-sectional analyses, it is evident in longitudinal studies. In the ROSSO Study, mean A1C is different at diagnosis between later SMBG users and permanent nonusers by 0.9%. In

later years, the initial large difference almost disappeared; i.e., metabolic control improved significantly more in patients using SMBG than in nonusers (4). Similarly, in the Kaiser Permanente cohort, there was an improvement of A1C by ~0.6% after initiation of SMBG, whereas A1C deteriorated by 0.2% in nonusers. These opposing changes were also observed after adjustments for change of type of antidiabetic medication or other potential confounders (3).

This concordance of observational studies on three different continents is remarkable. In the real world, SMBG appears to be preferentially used by younger patients who exhibit worse than average metabolic control, and the initiation of SMBG is followed by improved metabolic control.

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Is Self-Monitoring of Blood Glucose Appropriate for All Type 2 Diabetic Patients? The Fremantle Diabetes Study

Response to Kolb et al.

Despite the negative results of our community-based study of the link between self-monitoring of blood glucose (SMBG) and glycemia (1), Kolb et al. (2) commented on the overall concordance of our study and several other observational studies. They also suggest that we might have missed a positive impact of SMBG since new users in our cohort were not differentiated from either prevalent users or never users.

In our prospectively followed 531 type 2 diabetic patients (1), 92 who self-monitored at study entry stopped without subsequent detriment to their A1C (median [interquartile range] 7.0% [6.3–8.0] vs. 7.3% [6.4–8.4] at annual visits before and after stopping, respectively; mean change +0.1%; $P = 0.47$). By contrast, 103 patients who started SMBG during follow-up improved their A1C (7.5% [6.3–9.1] vs. 7.2% [6.2–8.4] before and after, respectively; $P = 0.032$). The mean change (–0.3%) is similar to that in both the new-user cohort of Karter et al. (3) and the meta-analysis of the few randomized controlled trials of SMBG (4).

The remaining 336 patients either performed SMBG throughout follow-up (SMBG+; $n = 306$) or did not (SMBG–; $n = 30$). We calculated updated mean A1Cs over 5 years for patients in these two groups by baseline diabetes treatment. For diet-treated subjects, the medians were 6.6% (interquartile range 5.9–7.1) for SMBG+ ($n = 92$) and 6.6% (5.9–7.0) for SMBG– ($n = 17$). For those taking oral hypoglycemic agents (OHAs), the medians were 7.4% (6.8–8.2) ($n = 181$) and 7.0% (6.5–7.5) ($n = 13$), respectively ($P = 0.24$). All 33 insulin-treated patients who were not performing SMBG at baseline monitored at some time during follow-up.

Previously, we have shown in the same longitudinal cohort of 531 patients (5) that the median A1C was reduced by 0.3% when diet-treated patients started

OHA and by 1.5% when OHA-treated patients started insulin. The apparent benefit of initiating SMBG might, therefore, reflect intensification of treatment. However, when our patients started SMBG, 85% continued with the same treatment, 12% reduced treatment, and 3% intensified treatment.

The largest randomized controlled trial (6) has found that the glycemic improvement observed in non-insulin-treated patients allocated to SMBG occurred in the first 3 months, with a steady state thereafter. These and our longer-term data suggest that SMBG may have only a relatively transient beneficial effect on glycemia. There is a need for strategies that ensure its sustainability.

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