second, there was a large paraprotein band at 29 g/L in the beta region, which was determined to be an IgM-kappa by immunofixation electrophoresis.

To determine whether the presence of the paraprotein or an unusual property of this patient's albumin was responsible for the inability to quantify albumin by brom cresol green binding, I isolated the patient's albumin by affinity chromatography on Cibacron Blue-Sepharose and the IgM fraction by chromatography on DEAE-cellulose and G-150-Sephadex. The patient's isolated albumin showed normal ability to react with brom cresol green. When the isolated IgM paraprotein was added to the patient's albumin or to any other serum sample, the albumin concentration was underestimated in proportion to the amount of IgM added.

The kinetics of the reaction of brom cresol green and albumin were monitored in normal serum and in the patient's serum. Albumin normally reacts rapidly with brom cresol green, and color development is complete in <5 s. The presence of the IgM paraprotein in the patient's serum delayed the reaction such that color development required several minutes to reach its final value. Since the Astra Ideal reads absorbance at 10 s and computes albumin concentration as if the reaction were complete, albumin concentration was significantly underestimated.

To determine whether the IgM was an antialbumin, and hence was interfering competitively with the dye, I immobilized albumin and the IgM on agarose (Sepharose), and prepared affinity columns with these (I). The IgM column (12 mg of IgM per 5 mL of Sepharose) demonstrated no affinity for 2 mg of albumin, and the albumin column (120 mg of albumin per 20 mL of Sepharose) demonstrated no affinity for 14 mg of the IgM. Evidently the interaction between albumin and the IgM that interferes with the brom cresol green binding is not a specific antibody–antigen interaction.

To determine whether other IgM paraproteins might also slow the reaction of albumin with brom cresol green, I studied 15 additional serum samples containing IgM paraproteins in concentrations ranging from 0.4 to 10 g/L. None showed any evidence of a slowed reaction between albumin and brom cresol green, all reaching final color development within 5 s.

The suppression of reaction between albumin and brom cresol green that occurred in the presence of this IgM paraprotein does not appear to be a common phenomenon for IgM paraproteins but an isolated property of the specific IgM described.

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Reference

Absence of Statistical Association between Hyperglycemia and Metabolic Acidosis in both Type I and Type II Diabetic Patients on Chronic Dialysis, A. H. Tzamaloukas and P. S. Avasthi (Renal Section, VA Medical Center, Albuquerque, NM 87108)

Renal function contributes to the mechanisms of metabolic acidosis that is frequently seen in hyperglycemia. We studied the effects of absent renal function on metabolic acidosis by comparing concentrations of blood glucose and TCO2 in 41 (15 type I) diabetics on hemodialysis (HD; 2196 samples) and 14 (eight type I) diabetics on peritoneal dialysis (PD, 258 samples). We used the definitions: euglycemia, glucose 3.3–10 mmol/L; significant hyperglycemia, glucose >30 mmol/L; significantly low TCO2, below the 99% lower euglycemic confidence limit, or <15 mmol/L.

Frequencies of low TCO2 were as follows: in HD, euglycemia 9%, hyperglycemia 10% (not significant, NS, by χ2); in PD, euglycemia 14%, hyperglycemia 16% (NS). Frequencies of hyperglycemia were as follows: in HD, normal TCO2 11%, low TCO2 11% (NS); in PD, normal TCO2 14%, low TCO2 18% (NS). Low TCO2 was due to ketoacidosis in three HD and three PD samples with hyperglycemia. No other pathogenetic association between acidosis and hyperglycemia was detected. In 70% of the hyperglycemic samples with low TCO2, a clinical cause of metabolic acidosis unrelated to hyperglycemia was present. Overall comparison between TCO2 concentrations at different blood glucose concentrations (variance analysis) showed no statistical significance for either HD (Figure 1) or PD samples. Absence of low TCO2 means absence of uncomplicated metabolic acidosis.

For both type I and type II diabetics on dialysis, metabolic acidosis is not observed more frequently in hyperglycemia than in euglycemia. When (rarely) metabolic acidosis is caused by hyperglycemia, ketoacidosis is the only recognizable cause. The other causes (lactic acidosis, hypertonicity) of metabolic acidosis in hyperglycemia of patients with intact renal function are absent in hyperglycemic dialysis patients.


This method for assaying urinary oxalate is based on measurement of oxidation products produced by the catalytic activity of the enzyme oxalate oxidase (I–3). Reagents were from Sigma Diagnostics, St. Louis, MO. We used a Multistat III® microcentrifugal analyzer (Instrumentation Laboratory Inc., Lexington, MA).

After extraction of oxalate from control pools and patients' specimens, all calibrators, controls, patients' samples, and