Letters to the Editor

Glucagon and Hyperkalaemia in Decompensated Diabetes

Dear Sir

Massara et al. recently reported in this journal [1] the role played by glucagon in the regulation of plasma potassium. In their study of six normal volunteers, the authors described an increase in serum potassium resulting from exogenously induced hyperglucagonaemia and insulinopenia. They also presented evidence that the phenomenon of hyperkalaemia was not mediated by hyperglycaemia or by a reduction in aldosterone secretion. We wish to contest the relevance of Massara's findings to the mechanism of hyperkalaemia in cases of severe decompensated diabetes.

We have recently reported the initial potassium values of 22 consecutive hospital admissions for diabetic ketoacidosis: mean pH, 7.4 ± 0.17 ; mean initial blood glucose, 37.9 ± 17.7 mmol/l; mean serum potassium value, 5.58 ± 1.14 mmol/l [2]. From the laboratory profile on admission, we calculated statistical correlations

between different parameters. A highly significant correlation was found between initial serum potassium levels and initial blood glucose ($r = 0.80$; $p < 0.001$, Fig. 1). Although no data on aldosterone and glucagon were available in this retrospective study, our results suggest that the combination of hypertonicity due to hyperglycaemia, together with insulinopenia, may be the most important mechanism for the hyperkalaemia. Although hyperglucagonaemia is a classical finding in diabetic ketoacidosis [3, 4], Muller et al. could find no correlation between hyperglucagonaemia and hyperkalaemia in 26 patients with diabetic acidosis [3]. Recalculation of their data confirms a correlation coefficient without statistical significance ($r = 0.15$, $p > 0.1$). It is probable that other factors or hormones, such as renal function, degree of acidosis, aldosterone, somatostatin or glucagon may be relevant, but we conclude that hyperglycaemia is the single most important cause of hyperkalaemia in diabetic ketoacidosis. Further investigation is urgently needed to elucidate the origin of hyperkalaemia in decompensated diabetes, but we would question whether glucagon has a major role in potassium regulation when this conclusion is derived from normal volunteer studies.

Yours sincerely,

L. Van Gaal and I. de Leeuw

Fig. 1. Correlation between initial blood glucose and initial serum potassium in 22 consecutive hospital admissions for diabetic ketoacidosis. Linear regression analysis showed a highly significant correlation ($r = 0.80$; $p < 0.001$)

References

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Dr. L. Van Gaal

Department of Endocrinology and Metabolism
University of Antwerp
Universiteitsplein 1
B-2610 Wilrijk, Belgium