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Diabetologia

Letters to the Editor

Glycosylated Haemoglobin in Renal Failure

Dear Sir,

In a recent publication in your journal [1] De Boer and colleagues reported on elevated levels of glycosylated haemoglobin (HbA1) in patients with chronic renal failure without a correlation of HbA1 either with fasting blood glucose levels or with sum of glucose levels obtained during an oral glucose tolerance test. These findings are in conflict with the results reported by Dandona et al. [2] who found low levels of HbA1 in patients with chronic renal failure and also in renal transplant patients. Their results seemed obvious as young erythrocytes are known to have a low content of HbA1 [3] and the life-span of erythrocytes is shortened in renal failure [4].

Since there exist conflicting data we would like to report our findings of HbA1 levels in patients with various degrees of renal failure.

We have previously reported [5] that HbA1 values were elevated in patients with advanced renal failure on conservative therapy and that HbA1 values showed a significant correlation with the plasma creatinine. Patients on haemodialysis showed the highest levels of HbA1 independent of whether glucose-free or glucose-rich dialysate fluid was used. Now we have extended our studies by performing glucose tolerance tests in 28 patients with various degrees of renal insufficiency on conservative therapy.

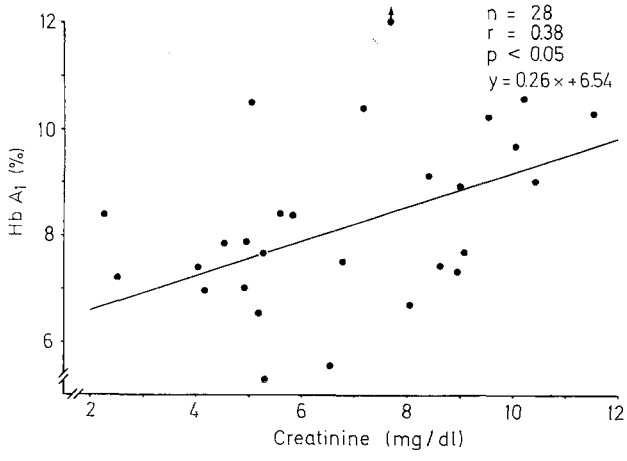

Fig. 1. Correlation between HbA1 and plasma creatinine

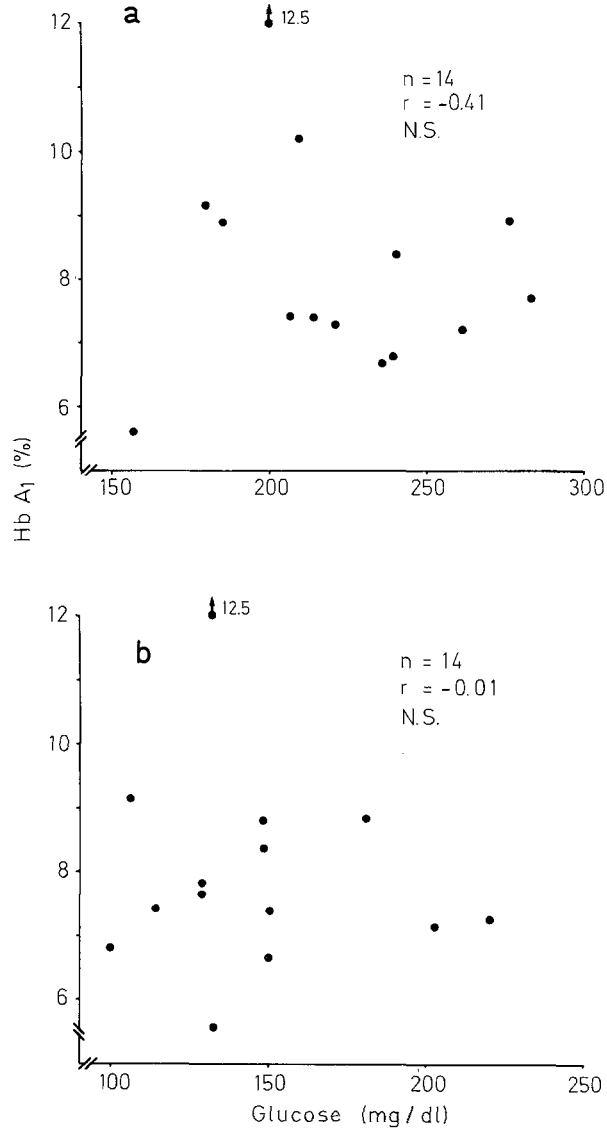

Fig. 2. Correlation between HbA1 and maximal glucose values **a** and 2 hours glucose values **b** after OGTT

HbA1 values were measured by a microcolumn technique [6], plasma creatinine by an autoanalyser method and blood glucose using a glucose-oxidase method.

There was a significant positive correlation between HbA1 and plasma creatinine values (Fig. 1). In 14 patients 100 g OGTT was performed. Confirming the results of De Boer there was no correlation between HbA1 and fasting blood glucose values, or maximal or 2 hours glucose values after OGTT (Fig. 2 a and 2 b). In another group of 14 patients an intravenous GTT was performed with 0.33 g glucose/kg bodyweight. No correlation between HbA1 and the k-values could be found.

Thus we can confirm the results of De Boer suggesting that renal failure itself causes an elevation of HbA1 values and that HbA1 is no parameter of carbohydrate intolerance in chronic renal failure.

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References

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6. Welch SG, Boucher BJ (1978) A micro-scale method for the measurements of haemoglobin $A_{1(a+b+c)}$. Diabetologia 14: 209–211

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Erratum

Diabetologia, Volume 18, No. 6 (June)/1980, Pages 493–500: Paper Østerby/Gundersen "Fast Accumulation of Basement Membrane Material and the Rate of Morphological Changes in Acute Experimental Diabetic Glomerular Hypertrophy".

The second formula in the right-hand column on page 494 should read:

$$L_v \text{ (capillaries/glomerus)} = \frac{2 \cdot \Sigma \text{ Q (lumina)}}{k^2 \cdot \Sigma \text{ P (polygon)}} \text{ mm/mm}^3.$$

The figure 2 in the numerator was omitted in the original publication of this paper.