Pseudohypoglycaemia in Acute Renal Failure with Wernicke-Korsakoff Syndrome

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Sir,

Lacour [1] has recently suggested that the glucose oxi-dase method is unreliable for determining blood glucose in uraemic patients receiving ascorbic acid, since oxidizable substances (such as ascorbic acid) interfere with the enzymatic reaction. As a result, glucose levels are deceptively lower than those obtained with for example the hexokinase and glucose dehydrogenase methods, where this interference does not occur.

We also have observed marked pseudohypoglycaemia with the glucose oxidase method [2] in uraemia, as shown by the observation of normal values when the orthotoluidine method [3] is used. This enzymatic system embraces glucose, mannose, galactose, gulose, and talose, in other words, the entire aldohexose group. Our 4 cases (table I) had the following in common: abdominal surgery, acute renal failure, and the neurological symptoms of the Wernicke-Korsakoff (W-K) syndrome (confusion, loss of memory, psychomotor agitation, nystagmus, ataxia).

All patients were parenterally fed with hypertonic glucose infusions with insulin; blood insulin was normal, and the picture was unchanged by acute glucose loading without insulin. There was no evidence of hypoglycaemic coma. Parenteral administration of 600–800 mg/day of thiamin led to regression of the cerebral picture, followed by normalisation of blood sugar in our first patient only, who is now undergoing regular dialytic treatment and has been free from glycidic abnormalities and neurological symptoms for 3 years.

The other 3 patients died within 1 week after the pseudo-hypoglycaemic pattern without neurological or haemato-logical normalisation. Thiamin (400 mg/day) was administered 2 days before death in cases 2 and 3.

The orthotoluidine method is unaffected by oxidizable interfering substances, and an explanation close to that proposed by Lacour [1] can be suggested. However, we

think that ascorbic acid (even if overdosed) is unlikely to be responsible: first, because only 2 of our patients (cases 1 and 3) received 2×0.5 g/day of vitamin C, secondly, because the glucose levels in our series are much lower than those of Lacour's patients, and lastly, because vitamin C management is extremely frequent in seriously ill patients fed parenterally, while this abnormal blood glycidic pattern is very uncommon.

For this last reason, we consider it unlikely that a simple uraemic toxin is the interfering substance, because this is a very uncommon finding in acute renal failure. Moreover, it should be noted that in case 1 blood glucose returned to the normal range after correction of the neurological symptoms, whereas renal failure persisted. On the other hand, the presence of interfering substances is not the only explanation for the discrepancy between the glucose oxidase and orthotoluidine methods. We cannot rule out the possibility that a glycidic metabolism enzymatic alteration may give rise to a glucose transformation in other aldohexoses normally absent. In this context, it is interesting to observe the association with the W-K syndrome, itself dependent on a glycidic-related pathology.

This syndrome is recognised by necropsy in 2% of all cases. It is often misdiagnosed [4], especially in seriously ill patients, when glucose hypertonic infusions increase the thiamin requirement [5]. In Italy omission of thiamin in the multivitaminic preparations for intravenous use often leads to the onset of an unrecognised beri-beri. A favourable genetic [6] or acquired background may lead to the W-K syndrome, and nephrologists must remember that renal failure is an excellent acquired background, either for an enzymatic deficiency, such as defective transketolase activity [7,8], or for a water-soluble vitamin loss [9].

Can the complex changes in sugar metabolism caused by the thiamin deficiency responsible for the neurological pattern also explain a glucose shunt to other normally insignificant aldohexoses? The reaction in which thiamin

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Table I. Patients with acute renal failure, W-K syndrome and pseudohypoglycaemia

Sex

Age, years

Dialytic treatment

Surgical picture

cholecystic empyema and pancreatic abscess haemodialysis

acute appendicitis peritoneal dialysis severe

intestinal ischaemic infarction haemodialysis

intestinal neoplastic occlusion haemodialysis

is involved in the penthose-phosphate pathway does not appear to offer the theoretical premisses for such a shunt. Further observations and research are necessary before satisfactory results can be obtained.

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