Letters to the Editor

No Rationale for Antiplatelet Drug Treatment in Diabetic Ketoacidosis

Dear Sir,

Paton [Diabetologia (1981) 21: 172–177][1] reported his findings on plasmatic coagulation factors during diabetic coma and recommended a trial with antiplatelet drugs in ketoacidosis. However, he did not show any data on platelet function during this medical emergency to support this idea. We would like to report our results on 14 ketoacidotic patients (age range 17–54 years) admitted to Schwabing City Hospital. The cause of ketoacidosis was usually discontinuation or inadequacy of insulin administration. Patients taking drugs, which may have had an influence on platelet function, were excluded. Platelet aggregability was expressed as the concentration of ADP necessary to induce 50% of maximal aggregation (threshold concentration). Plasma β -thromboglobulin was measured by radioimmunoassay [2]. Normal controls were healthy volunteers (age range 21–47 years).

The results are shown in Table 1. These data show that platelet aggregation in ketoacidosis is markedly depressed and β -thrombo-

globulin levels are not raised during the first 24 h of coma treatment. In view of the depressing effect of antiplatelet agents on prostaglandin I_2 synthesis and renal blood flow [3], drugs, such as aspirin, appear to be contra-indicated in ketoacidosis. Since thromboembolic complications during diabetic coma occur more commonly in elderly patients, in circulatory shock, and in hyperosmolar, non-ketotic coma [4, 5], heparin treatment may be indicated in such high risk patients. However, we would argue that there is no rationale for antiplatelet drugs in diabetic ketoacidosis.

Yours sincerely,
H. U. Janka and H. Mehnert

References

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H. U. Janka
Schwabing City Hospital
Koelner Platz 1
8000 Munich 40, FRG

Table 1. Platelet aggregation and plasma β -thromboglobulin levels during diabetic ketoacidosis

Time	Bicarbonate (mmol/l)	ADP threshold concentration (μ mol/l) (n = 14)	β -thrombo- globulin (ng/ml) (n = 8)
Start of treatment	3.9 $\pm$ 1.0	4.1 $\pm$ 1.6	63.6 $\pm$ 13.3
24 h	19.4 $\pm$ 1.7	2.8 $\pm$ 1.0	62.0 $\pm$ 16.3
120 h	20.9 $\pm$ 0.9	0.9 $\pm$ 0.1	65.1 $\pm$ 6.0
Control subjects (n = 61)		1.9 $\pm$ 0.2	61.5 $\pm$ 8.3

Results expressed as mean $\pm$ SEM

Are Islet Cell Antibodies Produced as a Result of Trauma?

Dear Sir,

It has been demonstrated in several organs that antibodies against those organs are produced following tissue damage. Thus, in the liver, Pinckard and Weir describe the transient appearance of mitochondrial antibodies following liver damage by carbon tetrachloride [1]; in the heart Van der Geld [2] demonstrated cardiac antibodies after myocardial infarction or cardiomy; and these have also been described in patients with idiopathic hypertrophic subaortic stenosis [3]. In the exocrine pancreas several early investigations have detected antibodies to pancreatic antigens in patients with chronic pancreatic disease and animals with experimentally induced pancreatitis [4–7].

In juvenile insulin-dependent diabetic patients islet cell antibodies can be demonstrated in the majority of patients at the time of diagnosis, but thereafter are seen to decline. This transient pattern of auto-antibody production is not typical of the situation usually seen in organ-specific auto-immune disease, where antibodies tend to be persistent. We therefore decided to investigate whether, by analogy with the con-

ditions described above, trauma to the pancreas per se could induce the appearance of islet cell antibodies.

We therefore investigated the presence of islet cell antibodies in 21 random subjects, not suspected of being diabetic, who were undergoing endoscopic retrograde cholangio pancreatography. Investigations were carried out for 6 weeks following the procedure. Islet cell antibodies were looked for by both the 1gG and C3 techniques. Thirty-four random blood donor specimens were examined over the same period.

All the sera from the 21 subjects were uniformly negative for islet cell antibodies pre- and post-endoscopic retrograde cholangio pancreatography. Anti-nuclear antibodies were present in two patients throughout the investigation. After endoscopic retrograde cholangio pancreatography, exocrine staining, similar to that described by Lendrum and Walker [7], was noted in some patients. All blood donor control subjects were negative for islet cell antibodies.

Endoscopic retrograde cholangio pancreatography may lead to a transient rise in serum amylase [8]; this could reduce fluorescence