OBSERVATIONS

Predictive Value of Postoperative Glycosuria After Partial Elective Pancreatectomy in Focal Congenital Hyperinsulinism

Congenital hyperinsulinism (CHI) is the most frequent cause of persistent hypoglycemia in infants (1). Early diagnosis and management are mandatory to prevent permanent brain damage. Two main forms are distinguished based on anatomical and genetic characteristics: the focal and the diffuse forms (2). In focal forms, the complete removal of the lesion allows the cure of the disease, but residual affected tissue may be left behind. This situation, observed in about 15% of cases, often necessitates additional surgery (3). We evaluated the value of detecting postoperative glycosuria as a predictor of successful surgery for focal CHI.

The study included 51 children operated on for focal CHI from 1989 to 2006. All patients had a constitutional paternal mutation in one of the genes encoding the ATP-sensitive K^+ channel subunits in chromosome 11p15. The focal form was confirmed at the surgical procedure (4). Postoperative blood glucose levels and glycosuria were measured at the following postoperative time points: 30 min, every 2 h for 12 h, and then every 3 h during the following 24 h. Postoperative recurrence of hypoglycemia was used as the first criterion of incomplete resection of the lesion and persistent CHI.

In 34 children (67%), glycosuria was noted after the end of surgery with a median time of detection of 120 min (range 0–720). At the time of glycosuria, median glucose intake was 0 mg · kg^{-1} · h^{-1} (0–0.23) and median plasma glucose concentration was 13.9 mol/l (8.1–26.5). Glycosuria spontaneously disappeared at a median of 6.5 h (1–18). Among these 34 patients, 2 (6%) had postoperative recurrence of hypoglycemia.

No glycosuria was observed in the remaining 17 children (33%). At 120 min after the end of surgery, median glucose intake was 0.2 mg · kg^{-1} · min^{-1} (range 0–0.25) and median plasma glucose concentration was 9.9 mmol/l (5.7–17.5). Among these 17 children, 10 (59%) were definitively cured while 7 (41%) had recurrences of hypoglycemia. Six of these last patients underwent reoperation, and one was controlled by medical treatment.

In this study, the recurrence of postoperative hypoglycemia was significantly lower in the presence of postoperative glycosuria (6 vs. 41%; $P = 0.004$), whereas the postoperative glucose requirement was significantly lower ($P =$ 0,001). We did not find any significant difference in the postoperative plasma glucose concentration ($P = 0.19$).

In focal CHI, the active endocrine cells switch off the uninvolved β-cells (5), explaining how a complete resection of the lesion could lead to temporary episodes of insulin deficiency and glycosuria. Therefore, immediate postoperative glycosuria could be used as a simple criterion to predict the complete resection of focal forms of CHI. The absence of glycosuria can be explained by active β-cells at the end of surgery, whether they are from incomplete resection of the lesion or residual activity of the normal tissue not switched off by the lesion. Complementary postoperative insulin measurement and continuous plasma glucose concentration monitoring should help to understand the underlying physiological processes.

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