Diabetologia 18, 513–514 (1980)

Diabetologia

Short Communications

Abnormalities of Glucose Tolerance as a Late Consequence of Hypertrophic Pyloric Stenosis Surgically Treated in the Neonate

H. Chimènes[1], D. Gautier[2,3], R. Bon[2], and M. Fromantin[2]

[1] Hôpital Beaujon, Clichy, and American Hospital of Paris, Neuilly sur Seine, and [2] Hôpital d'Instruction des Armées Begin, Saint Mandé, France

Summary. 75,375 males called to military service were screened to discover the incidence of hypertrophic pyloric stenosis. 49 men gave a history of previous surgery for this condition and were given a 45 g/m2 oral glucose tolerance test. Fourteen had impaired glucose tolerance. In no case was there a family history of diabetes mellitus.

Key words: Diabetes mellitus, hypertrophic pyloric stenosis in the neonate, glucose intolerance.

During the last five years we have observed four cases of diabetes mellitus in patients with a previous history of hypertrophic pyloric stenosis (HPS) corrected surgically in infancy. In view of this we investigated glucose tolerance in men with a previous history of HPS derived from a population of young males called to military service.

Material and Methods

This study was performed in collaboration with the "Centre de Sélection N° 1 de Vincennes" during a 15 month period (January 1977–March 1978). 75,375 young men, aged 20 years, were questioned about a history of HPS and examined; those with a positive history underwent an OGTT with 45 g/m2 glucose. Blood sugar was measured in venous whole blood with a Technicon autoanalyzer (neocuproin method); plasma insulin levels were measured simultaneously by Lepetit kit. Thirty eight matched control subjects were also given an OGTT.

The criteria for interpretation of the OGTT were those recently described in a editorial of Diabetologia [12].

[3] Address reprint requests to: D. Gautier, Hôpital d'Instruction des Armées Begin, 69, Avenue de Paris, F-94160 Saint-Mandé, France

Results

Of the 75,375 subjects studied, 49 (0.6 per 1000) had a past history of HPS. None of our subjects were overweight; mean weight 63 kg, mean height 1.72 m. The mean height of the control group was 1.72 m, mean weight 63.1 kg. The age of all the subjects studied, as of those of the control group, was 20 years. None of the HPS subjects had a family history of diabetes mellitus nor of obesity. The results of the 49 OGTT are illustrated in Figure 1; 35 were normal

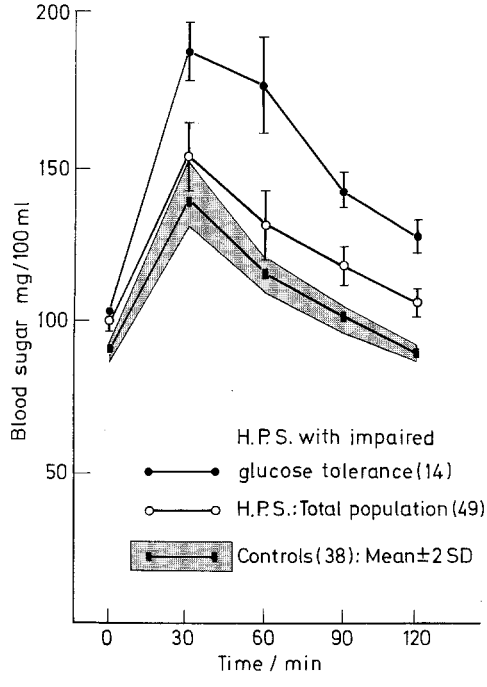

Fig. 1. Glucose concentration in peripheral venous blood during an oral glucose tolerance test in HPS patients (mean ± SEM) and in control subjects (mean ± 2 SD in hatched area). Control subjects are selected among young men aged 20 years, non obese and without family history of diabetes mellitus

0012-186X/80/0018/0513/$01.00

and 14 (28%) abnormal. There was no significant difference between the levels of insulin in the subjects with HPS and the normal population:

normal HPS subjects (35):
basal 11.2 μU/ml (± 0.8) (± SEM)
1 h 50 μU/ml (± 12.4)

glucose intolerant HPS subjects (14):
basal 12.8 μU/ml (± 0.7)
1 h 70 μU/ml (± 15.3)

control group subjects (38):
basal 16.3 μU/ml (± 1.3)
1 h 74 μU/ml (± 17.8)

Discussion

The survey by Berglund [4] of young men entering military service in Sweden found 203 cases with a past history of HPS. He reported an association with shortness of stature, not confirmed in the present study, but there was no reference to glucose metabolism.

In our study, impaired glucose tolerance (IGT) was observed in nearly 1 case in 3 (28.5%). This figure is nearly 15 times greater than the 2% incidence of diabetes mellitus in the general population [5].

This difference in incidence is so great that a chance association is excluded between HPS and IGT; it suggests a causal relationship between either HPS and IGT or between surgical correction of HPS and IGT.

The mechanism of this relationship is unknown, but there are several possible explanations. The glucose and insulin curves in the HPS patients differ from those observed in gastrectomized patients or from those submitted to selective vagotomy [6, 7], so that direct surgical interference can be excluded. A common genetic mechanism for diabetes mellitus and HPS could be postulated. The total absence of a family history of diabetes in the 49 subjects does not, however, support this hypothesis. Nonetheless, a study of HLA groups in these subjects may prove to be of interest.

The anatomical lesions of HPS exclusively involve muscle and the duodenal mucosa is normal. The surgical procedure itself is extramucosal. However, a possible role of the intestinal hormones and in particular that of GIP could be postulated. There could be an acceleration of gastric emptying and an excessive secretion of GIP [8]. Ross et al. [9], who confirmed the hypersecretion of GIP among diabetics, suggested that this "potentially glucagonotropic" hormone could contribute to the production of diabetes in patients whose beta-cells are relatively resistant to its insulinotropic action. The possible role of GIP in the glucose intolerance of the HPS patients remains to be assessed.

There is no explanation for the fact that only one in 3 of those operated for HPS should later develop IGT. It is possible that the explanation lies in a difference in surgical techniques.

From the results of our study, we suggest that children who have undergone surgical correction for HPS should be followed regularly to detect and treat the earliest abnormality of glucose tolerance.

Acknowledgement. The authors are indebted to Dr. S. R. Nightingdale for his help in the English translation of the French manuscript.

References

1. Daudet M, Chappuis JP, Deffrenne P (eds) (1973) Encyclopédie Médico-Chirurgicale, no 10: Pédiatrie: Sténose hypertrophique du pylore chez le nourrisson. Encyclopédie Médico-Chirurgicale, Paris, p 4018
2. WHO (1970) Rôle des facteurs génétiques dans les malformations congénitales. WHO Tech Rep Ser 438
3. Podesta AM, Tosoni Dalai J (1972) Sull'eredita della stenosi congenita del piloro. Minerva Med 63: 4687–4691
4. Berglund G, Rabo E (1973) A long term follow-up investigation of patients with hypertrophic pyloric stenosis with special reference to the physical and mental development. Acta Paediatr Scand 62: 125–129
5. Fromantin M, Gautier D (1977) Journées annuelles de Diabétologie de l'Hôtel-Dieu. Diabète et Armées. Ed. Flammarion, Paris, p 244–252
6. Chimenes H, Vallin J, Felber JP, Delorme ML, Grassi L, Chevalier RJ (1967) Etude clinique et métabolique des troubles glycorégulateurs des gastrectomisés: le diabète des gastrectomisés. Ann Endocrinol (Paris) 28: 611–640
7. Bittner R, Beger HG, Kraas E, Gogler H (1976) Effects of selective gastric vagotony and pyloroplasty on oral and intravenous glucose tolerance and insulin secretion. Arch Surg 111: 850–853
8. Lauritzen KB, Moody AJ (1978) The reponse of GIP and Insulin to glucose in duodenal ulcer patients. Diabetologia 14: 149–155
9. Ross SA, Brown JC, Dupre J (1977) Hypersecretion of GIP following oral glucose in diabetes mellitus. Diabetes 26: 525–529
10. Freni G, Tiotto R, Di Francesco G (1977) Stenosi ipertrofica del piloro. Minerva Pediatr 29: 1575–1577
11. Schäfer, KH, Bingel G (1975) Konservative und chirurgische Therapie der spastischen, hypertrophischen Pylorusstenose (SHP). Eine Katamnestische Studie. Monatsschr Kinderheilkd 123: 503–508
12. Keen H, Jarrett RJ, Alberti KGMM (1979) Diabetes mellitus: A new look at diagnostic criteria (editorial). Diabetologia 16: 283–285

Received: June 26, 1979,
and in revised form: December 12, 1979

Dr. H. Chimènes
74 rue Saint Didier
F-75116 Paris
France