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CASE REPORT

Peritoneal dialysis in an infant with Type 1 diabetes and hyperosmolar coma

G. Multari*, B. Werner*, M. Cervoni*, R. Lubrano*, F. Costantino*, V. Demiraj, and P. Pozzilli**

*Departement of Pediatrics, University of Rome "La Sapienza", **Department of Internal Medicine, University of Rome "Tor Vergata" and University Campus Biomedico, Rome, Italy

ABSTRACT. Hyperosmolar coma which is characterized by severe hyperglycemia in absence of chetosis is very rare in pediatric age with only 11 cases reported in the literature. The outcome of the condition is usually poor with mental retardation being the most common event. Here a case of hyperosmolar coma is described in a female of three months of age who was treated with peritoneal dialysis 11 hours after admittance to hospital. This female patient has been receiving insulin from three months of age and today at the age of 10 years she leads a normal life despite being on insulin therapy. A very low level of C-peptide (<0.3 ng/ml) clearly confirms she is affected by Type 1 diabetes. To our knowledge this is the first case report of hyperosmolar coma in a neonate with Type 1 diabetes who survived this condition without late neurological consequences. (J. Endocrinol. Invest. *24:* 104-106, 2001)

INTRODUCTION

Hyperosmolar coma is characterized by severe hyperglycemia (>600 mg/dl), hypernatriemia, absence of chetosis, severe dehydratation, coma of various degrees, convulsion and hemiparesis (1). It is uncommon in pediatric age and until today only 11 cases have been described (2). The first case was reported in a child affected by Down syndrome, in another four cases patients died soon after diagnosis, and two patients were described as severely mentally retarded (3-5). In another infant, aged 6 weeks, the adoption of "exanguinotrasfusion" for the persistently high blood glucose level was unsuccessful (6).

A case is here presented of hyperosmolar coma occurring in a neonate of three months of age, resistant to insulin treatment with persistent hyperglycemia and anuria who underwent peritoneal dialysis. She survived and now at the age of ten, leads a normal life despite being on insulin.

Key-words: Hyperosmolar coma, Type 1 diabetes, peritoneal dialysis.

Correspondence: Prof. P. Pozzilli, Università Campus Biomedico, Via Emilio Longoni 83, 00155 Rome, Italy.

E-mail: p.pozzilli@caspur.it

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CASE REPORT

A 15-week-old female was brought to the hospital with a high temperature lasting for 48 hours, diarrhea lasting 12 hours and polypnea. Second-born by normal delivery at 40 weeks, she weighed 3050 g. Both Apgar score and neonatal period were normal. She was breast-fed until the second month of age and then mix-fed with soya milk with evidence for an increasing demand for milk over the previous 2 weeks (7 meals/*die*, 180 ml each or 220 ml/kg/*die*). There was also a noticeable decrease in body weight (from 5,900 to 5,500 g in a week). Maternal grandfather and both paternal grandmother and grandfather were affected by Type 2 diabetes.

At the time of admittance she was found to be dehydrated (weight decrease by 6%), with a temperature of 38.5 C, heart rate of 150/min and respiratory rate of 60/min.

Laboratory data at the time of admittance were: 44% Ht, 13.6% Hb, 23,300 mm^3 white cells (Neutrophils 66%), 650 mg/dl blood glucose, 6.2 mEq/l K, 158 mEq/l Na (not corrected for glucose levels), 118mEq/l Cl, pH 7.1, 20.2 mmHg pCO_2, 50.0 mmHg pO_2, 5.8 mEq/l bicarbonates, 376 mOsm/l serum osmolarity and presence of glycosuria.

Treatment began initially with 0.45% NaCl 8 ml/kg/h for 2 hours followed by 0.25 IU/h insulin in-

fusion. After 6 hours blood glucose levels raised to 1,388 mg/dl with pH 7.34, 170 mEq/l Na, 3.6 mEq/l K, 410 mOsm/l osmolarity, 3.3 mmol/l lactate, 23 mmHg pCO_2, 74 mmHg pO_2, and an arterial pressure of 85/48. However 11 hours later, blood glucose dropped to 650 mg/dl with 180 mEq/l Na, 4.3 mEq/l K, 135 mEq/l Cl, 9.9 mmol/l lactate, 70 mg/dl BUN and 1.8 mg/dl creatinine. The patient was oliguric (60 ml x 24 h).

Given that anuria with lactic acidosis developed and that hyperglycemia and hyperosmolarity persisted, the patient was placed on peritoneal dialysis while simultaneously administered insulin (0.25 IU/kg/h). Briefly, intermittent peritoneal dialysis was performed using a 14 diameter gauge positioned in the right pelvic fossa. For the in and out flow of dialysis fluid we used a Y system with a separate line for each flow. Isotonic bags (Traverol) with low glucose concentration (1.36%) were employed. The total quantity of fluids administered ranged between 100-150 ml, whereas fluids removed ranged between 66 and 165 ml. The total of peritoneal dialysis lasted for 45 hours. Two and a half hours after beginning dialysis, the patient experienced convulsions with each crisis lasting between 2 and 3 minutes at various intervals of 20-35 minutes. The dialysis was stopped during convulsion. A calcium value of 6.8 mg/dl was reported. She was then treated with benzodiazepine first and 10% calcium gluconate soon after, intubated and transferred to the Neonatal Intensive Care. Pulmonary ventilation with positive pressure followed by intermittent pulmonary ventilation (IPPV) were implemented. Eleven hours after beginning IPPV, her breathing became spontaneous. Subsequently, the oxymetric values gradually set off normality, improvement of *sensorium*, decrease of blood glucose up to near normal values, progressive normalization of sodium, lactate and base excess were observed. Lactate became normal in 20 hours and diuresis returned physiological 20 hours later.

The patient was transferred to the ward; her neurological examination at this stage was normal. Meals consisted of adapted milk every 3 hours with small insulin boluses. She was discharged from hospital 60 days later without any neurological defects, insulin being her sole therapy.

Today (2000) this girl is 10 years old, her metabolic control with subcutaneous insulin is good, weight-height development is above average (75° centile), neurological defects are not present and she regularly attends school succeeding in her studies. C-peptide levels in this patient are very low (<0.3 ng/ml) clearly indicating that she is affected by Type 1 diabetes. Measurement of islet cell antibodies was not performed at the time of admission to hospital 10 years ago.

DISCUSSION

To our knowledge this is the first report of hyperosmolar coma in a neonate undergoing peritoneal dialysis who survived such a condition without showing late neurological or other consequences than Type 1 diabetes. At the presentation therapeutic measures were taken as expected in a case like this. However, despite infusion therapy with saline hypotonic solution first, followed by insulin infusion after, blood glucose and sodium levels remained very high after 24 hours. The clinical situation got worse with the appearance of lactic acidosis and anuria. To compensate for the severe dehydration, further administration of fluids and bicarbonate were carried out to correct lactic acidosis. In a similar case reported in literature, exanguinotransfusion was unsuccessful (6). Persisting lactic acidosis and the appearance of anuria prompted us to implement peritoneal dialysis. This was carried out to maintain liquid in the abdomen for a short time to prevent high blood glucose from causing a massive re-absorption of fluids from the abdomen. A progressive reduction of blood glucose, sodium levels and lactate with a normalization of acid-base equilibrium was obtained 72 hours later. Neurological manifestation observed two hours after beginning peritoneal dialysis should have been caused by hyperosmolarity and the unlikely thrombosis of cerebral arteries for vascular disseminated coagulation, even though it was not detectable at computerized tomography.

Management of the hyperosmolar hyperglicemic coma is not easy especially when other complications arise. The key for a successful outcome is a prompt, rapid administration of crystalloid solution that has tonicity appropriate to the level of hyperosmolarity (7). In the presence of renal failure treatment becomes extremely difficult.

We have no evidence to state that implementation of peritoneal dialysis was the reason for the successful outcome of this baby girl.

However, we were forced to adopt such a procedure because of anuria and it might be possible that its use has accelerated the clinical improvement, therefore limiting the time of exposure to high blood glucose which could have caused brain damage.

In conclusion, implementation of peritoneal dialysis seemed to be the variable which might have changed the course of hyperosmolar coma in this neonate at least compared with other cases reported in the literature (2). Such an approach, therefore, should be considered in similar cases to re-

duce the risk of severe complications associated with hyperosmolar coma in neonates.

REFERENCES

1. Gupta S., Prabhu M.R., Gupta M.S., Niblett D.
Severe non-ketotic hyperosmolar coma-intensive care management.
Eur. J. Anaesthesiol. 1998, *15:* 603-606.
2. Eidiliz-Markus T., Nussinovic M., Varsano I., Kauschansky A.
Non ketotic hyperosmolar coma in children.
Isr. J. Med. Sci. 1994, *30:* 585-587.
3. Yamashiro Y., Yamamoto T., Mayama H.
Non ketotic hyperosmolar coma in two diabetic children.
Acta Paediatr. Scand. 1981, *70:* 337-340.
4. Rubin H.M., Kramer R., Drash A.
Hyperosmolarity complicating diabetes mellitus in childhood.
J. Pediatr. 1969, *74:* 177-186.
5. Hoffman W.H., Fernandos S.S.
Hyperglycemic hyperosmolar non-ketotic coma in a non-diabetic child.
Diabetologia 1983, *25:* 531.
6. Vemon D.D., Postellon D.C.
Non-ketotic hyperosmolar diabetic coma in a child. Management with low dose insulin, infusion and intracranial pressure monitoring.
Pediatrics 1986, *77:* 70-77.
7. Matz R.
Management of the hyperosmolar hyperglycemic syndrome.
Am. Fam. Physician 1999, *47:* 659-665.