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Neonatal transitory hypomagnesaemia

By

V. F. Lukács

Apáthy Children's Hospital, Budapest

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The case of a 5-week-old baby is described who was suffering from neonatal transitory hypomagnesaemia which, accompanied by haemato-chemical, ECG and EEG anomalies, induced uncontrollable convulsions. The condition improved radically and definitively on treatment with magnesium. The five similar cases reported previously in the literature are reviewed and the aetiological aspects of the condition are discussed.

The two principal categories of hypomagnesaemia, a rare anomaly in newborn age, are

(1) congenital hypomagnesaemia with hypocalcaemic tetany;

(2) a) asymptomatic hypomagnesaemia and b) transitory hypomagnesaemia with tetany and hypocalcaemia.

In recent years we have observed 16 infants with slight hypomagnesaemia. In addition, a case of neonatal transitory hypomagnesaemia has been found and will be reported below.

Report of a case

G. F., a 5-week-old male infant weighing 4200 g was admitted in January, 1972, in a subfebrile condition. The family history was noncontributory; the baby was the second child of a 19-year-old healthy mother, her first child is normal. Gestation ran a smooth course, delivery was at term and smooth, the infant weighed 2700 g at birth. The baby was bottle-fed. No reliable information concerning vitamin-D supply was available. Before admission, the infant had been ill for a week, "moaning, and suffering from colics" with repeated vomitings. The stools had been normal, the temperature had not been taken.

At admission, the otherwise moderately developed infant's head appeared to be oversized (circumference, 37 cm); the anterior fontanelle was large. The baby had a mild upper respiratory catarrh, his temperature was normal. Calcium chloride 0.5 g t.i.d. was given for 5 days and then a single dose of 5 mg of vitamin D_3 intramuscularly. X-rays of the hand showed one centre of ossification; the epiphyses were broad, their calcium content was diminished. In serum, Ca was 8.8 mg, and P, 6.5 mg per 100 ml; alkaline phosphatase, 30 U; Mg, 0.9 mEq/litre; K, 4.6 mEq/l. Repeated afebrile convulsions extending over the entire body appeared on the sixth day. Intravenous calcium, diazepam, pyridoxine and lumbar puncture relieved them only for a short time. The CSF was negative. The blood sugar level was 90 mg per 100 ml; NPN, 19 mg per 100 ml. On the ECG the Q—T interval

was 0.36. The EEG showed a background activity approximately consistent with the patient's age, and a slight alteration on the left side. Several apparently independent spike-and-wave complexes were observed; the left precentral spike focus seemed to be the most active one, involving the homologous area from time to time. Besides, a less intensive independent parietal focus was found on the left, and a precentral one on the right side.

Treatment with magnesium sulphate (2×250 mg intramuscularly) instantly stopped the seizures which subsequently never reappeared despite a hyperpyretic episode. Four days later the serum values were: Ca, 9 mg per 100 ml; P, 6.4 mg per 100 ml; alkaline phosphatase 21 U; Mg, 1.9 mEq/litre. On the ECG the Q—T interval had become significantly shorter and then returned to normal, and the EEG no longer showed convulsive potentials. Electric activity was practically consistent with normal sleep at the baby's age.

Magnesium treatment was continued for 10 days. The baby repeatedly developed spastic bronchitis, bronchopneumonia and once contracted a grave Ps. pyocyanea septicaemia. Hence, it was only after more than three months that he could be discharged with a body weight of 4600 g and normal laboratory findings.

Urinary magnesium amounted to 0.08 mEq/l—0.16 mEq/day, strikingly low values. The corresponding values for a normal control baby of the same age and weight were 2.96 mEq/l—8.8 mEq/day. A single 25 mg/kg load of magnesium produced 0.33 mEqu/l—0.83 mEqu/day; these values still indicate an insufficient magnesium excretion, a phenomenon common under such conditions. In the healthy control baby, magnesium loading raised the urinary output to 3.60 mEqu/l—9.0 mEqu/day; these values were in harmony with those described as normal in the literature [11].

The infant has been healthy ever since and is developing normally.

Discussion

Transitory hypomagnesaemia develops in the first days of weeks of extrauterine life. In the present case, it had manifested itself on the 40th postnatal day. Such a quiescent postnatal period is frequent: the apparently healthy newborn suddenly develops convulsions in the form of classical hypocalcaemic tetany or eclampsia. The spasms may be restricted to one side of the body or to an extremity but may also appear in a generalized form. Intervals between the paroxysms may last hours, even several days. Increased neuromuscular and central irritability as a concomitant of hypomagnesaemia and hypocalcaemia is a well-known phenomenon.

Treatment with magnesium promptly relieves the convulsions. While large doses of intravenously administered calcium preparations failed to raise the low blood calcium level in the case under review, it became normal on treatment with magnesium, a principal feature of the condition at issue. In the present case, four days treatment sufficed to normalize the pathologic EEG which prior to treatment had shown a convulsive potential.

The blood Mg level forms the subject of a vast number of publications [7, 8]. A neonatal value of $1.61 \pm \pm 0.27$ mEq/litre would seem to be normal [8] but this value may lead to erroneous conclusions and for a reliable indication of hypomagnesaemia the value must be expressed in

terms of mg/kg of body weight comparred on the basis of the plasma volume. It would therefore seem that the cases of SALET and FOURNET [12], classified as belonging to category 2/a, should be revised. According to JUKARAINEN [7], convulsions will appear if the blood magnesium level drops below 1.0 mEqu/l.

diet; her food contained gluten. It is known that the blood magnesium level is low in coeliac disease: that of the mother amounted to 15—17 mg/l in the given case. In CLARKE and CARRÉ's case [1a] the mother had been treated with insulin every day for five years on account of severe diabetes. She succumbed to a hypo-

TABLE I

Reported cases of transitory neonatal hypomagnesaemia

	Authors	Year	Maternal disease
1	DAVIES et al. [2]	1965	Coeliac disease
2	CLARKE and CARRE [1a]	1967	Diabetes, hypoglycaemic coma
3	DOOLING and STERN [3]	1967	Hypophosphataemia, hyperthyroidism
4	FOURNET [5]	1968	—
5	ERTEL et al. [4]	1969	Hyperparathyroidism
6	Present case	1972	—

While some speculative interpretations point to a selective malabsorption [6, 14] or the permeability of the cell walls to magnesium [12] as the causative factor of hypomagnesaemia, the transitory neonatal form of the condition is aetiologically fairly clear. No dietetic factors are involved, nor do therapeutic doses of vitamin D influence the level of magnesium. The five cases published so far have been analysed by SALET and FOURNET [12] as well as by the present author; the case described in this paper is the sixth of its kind (Table I).

The mother [2] of the patient of DAVIS et al. [2] had coeliac disease and failed to observe the prescribed

glycaemic episode a few days after delivery. Magnesium deficiency is known to occur in cases of grave diabetes. The mother of the patient of ERTEL et al. [4] was suffering from hyperparathyroidism, a disease leading to a loss of magnesium which is excreted with the urine at an increased rate. Maternal hyperparathyroidism may induce hypoparathyroidism in the offspring. Likewise maternal hyperparathyroidism (and also hypophosphataemia) was present in the case of DOOLING and STERN [3]. FOURNET [5] would certainly have referred to some maternal disorder of this nature if it had existed.

In our case, the mother of the infant was apparently healthy but

refused to submit herself to detailed examinations.

Thus, owing to some maternal deficiency disease, all these infants seem to have been born with a low magnesium level, a condition which continued deteriorating by the increased urinary excretion. Also, the urinary calcium output kept increasing. A normally functioning kidney retains ions in cases of hypomagnesaemia and hypocalcaemia. The increased urinary excretion of magnesium is assumed to be due to a transitory hypoparathyroidism which improves on parathyroid treatment [2]. This theory makes it evident why a single or short treatment with magnesium ensures lasting improvement. In the present case the mother was healthy; the problem therefore remains why and on what grounds the condition had ensued.

Finally, according to a recent view, sudden unexpected death in infancy might be connected with hypomagnesaemia [1].

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Dr. F. V. LUKÁCS
Bethesda u. 3
1146 Budapest, Hungary