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SYMPTOMATIC HYPOCALCEMIA AND HYPOMAGNESEMIA DUE TO GENTAMICIN THERAPY IN AN 8-YEAR-OLD GIRL*

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In the literature seven cases of hypomagnesemia due to gentamicin therapy have been reported[1-3]. In three of them hypocalcemia was associated with hypomagnesemia, and all had hypokalemia. The documented cases were all adult patients. Here a child who was treated with gentamicin for 25 days and showed the signs of this syndrome is reported.

Case Report

An 8-year-old girl was admitted to the Hacettepe Children's Hospital with fever, eruptions, vomiting and headache of 10 days' duration. Her blood pressure was 80/50 mmHg, the pulse rate was 140/min, weight was 21 kg, and height was 120 cm. Upon physical examination, a pericardial friction rub was heard, and the liver was palpable 5 cm below the costal margin. There was a macular rash on her cheeks. Laboratory studies revealed: Hb 6.6 g/dl, WBC 5200/mm^3; erythrocyte morphology was normocytic and normochromic, and thrombocytes were abundant. The ECG revealed sinus tachycardia, and pericardial effusion was demonstrated in echocardiography. Pericardiocentesis produced 60 ml of exudate with 86% polymorphonuclear leukocytes and 14% lymphocytes. The protein concentration was 3600 mg/dl. Staphylococcus aureus was cultured from the effusion. CRP: (+ + +), latex: (−), anti DNA: (−), antinuclear antibody: (−).

Penicillin crystalline 500,000 U/kg and gentamicin 6 mg/kg were started. Penicillin was changed to methicillin after the culture revealed Staphylococcus aureus. On the tenth day of gentamicin therapy serum electrolyte values were as follows: Ca 8

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mg/dl, P 3.8 mg/dl, Na 136 mEq/l, K 2.9 mEq/l, Cl 96 mEq/l, CO_2 21 mEq/l. After three weeks of treatment, gentamicin was stopped. A week later Trousseau's phenomenon was elicited while withdrawing blood for a routine work-up. Control serum electrolytes were as follows: Ca 5 mg/dl, P 3.2 mg/dl, alkaline phosphatase 4 BU, Mg 0.5 mg/dl, K 2.5 mEq/l, Na 132 mEq/l, CO_2 34 mEq/l, BUN 15 mg/dl, blood pH 7.6, total protein 6 g/dl, albumin 4 g/dl, SGOT 24 U, SGPT 7 U, urine P 22-45 mEq/l, urine Na 105 mEq/l.

Oral calcium lactate 12 g/day and vitamin D_3 8000 u/kg/day were started. The following results were obtained after one week of treatment: serum Ca 5.7 mg/dl, P 3.5 mg/dl, CO_2 37 mEq/l, K 2.3 mEq/l, Na 134 mEq/l.

Because of the effects of a lack of vitamin D, parenteral magnesium was instituted and continued for seven consecutive days. On the third day of magnesium therapy, the serum calcium level rose to 9 mg/dl, CO_2 was 28 mEq/l, K 4.4 mEq/l.

Ten days after the magnesium therapy the calcium level was still 9.6 mg/dl, phosphorus was 3.5 mg/dl, and magnesium was 1.5 mg/dl. The child underwent pericardiectomy. The post-operative clinical course was uneventful, and she was discharged fully recovered.

Discussion

In this case symptomatic hypomagnesemia and metabolic alkalosis were probably caused by gentamicin therapy. On the tenth day of gentamicin therapy the serum calcium level was normal, and the only electrolyte imbalance was hypokalemia. After the third week of therapy metabolic alkalosis and symptomatic hypocalcemia developed. At that time, the serum magnesium level was also low. All these electrolyte abnormalities disappeared with the administration of magnesium. This result suggested to us that the magnesium deficiency could be the primary cause of this disorder.

In our patient, intake of magnesium was adequate and there was no history of malabsorption. In the absence of a deficient intake or an excessive intestinal loss, urine loss of magnesium and potassium seems to be a reasonable explanation for hypomagnesemia and hypokalemia.

Holmes et al[1] have speculated that gentamicin-induced secondary hypoaldosteronism may have been the cause of hypomagnesemia and hypokalemia. Their patients had elevated plasma renin activity and aldosterone level, but severe hypocalcemia was not found in these patients. Although the renin activity and aldosterone level were not determined in our case, the metabolic alkalosis and hypokalemia can be indirect evidence of hyperaldosteronism. Since we could not get any

response to administration of vitamin D and oral calcium salts, we attributed the hypocalcemia to hypomagnesemia.

The mechanism of hypocalcemia due to magnesium deficiency has been published in several reports[4-6]. The reported cases of gentamicin-induced hypomagnesemia are rare and all are adult cases. Therefore, the 8-year-old girl reported here is interesting and indicates the importance of the necessity of serum magnesium determination in patients on long-term gentamicin therapy and specifically in those who have clinical findings of hypocalcemia and hypomagnesemia.

Summary

An 8-year-old girl who developed hypomagnesemia, hypocalcemia and metabolic alkalosis during the course of gentamicin therapy is presented. Our findings are compared to previously reported cases, all of whom were adults.

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