Correction of Severe Metabolic Acidosis by Peritoneal Dialysis in Cyanotic Babies

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In order to correct severe metabolic acidosis, peritoneal dialysis was attempted in 2 cases of cyanotic babies. The condition was markedly improved in both of them by peritoneal dialysis with buffer solution, though they died finally of cardiac failure.

It could be concluded from our experiences that peritoneal dialysis was effective in the correction of severe metabolic acidosis in newborn babies with marked cyanosis.

Because of tissue hypoperfusion and tissue hypoxia, cyanotic babies are apt to fall into severe metabolic acidosis. The metabolic acidosis is sometimes so severe that it is life-threatening, and rapid correction of the condition is necessary. Correction of metabolic acidosis can be achieved by intravenous administrations of tris (hydroxymethyl) amino methane (THAM) or sodium bicarbonate. It is, however, hazardous to give these substances to babies by rapid intravenous drip infusion, because of side effects of the solutions and/or overhydration. Recently, peritoneal dialysis has been used in order to correct severe metabolic acidosis in cyanotic babies with apparently good results.

CASE REPORT

Case 1. J.T. A male baby was born at 9 a.m., June 19, 1967. His birth weight was 3,620 g. Marked cyanosis and tachypnea were noted at birth. Harsh systolic murmur was heard over the entire precordium. Cardiac catheterization and cineangiography were carried out in the next morning and disclosed preductal coarctation of the aorta with large patent ductus.

On June 21, 48 hours after birth, the division of ductus arteriosus, resection of coarctation and end-to-end anastomosis of the aorta were carried out under hypothermia. The lowest body temperature during the procedure was 27°C. The time of aortic cross-clamping for anastomosis was 50 minutes.

Immediately after the operation, the patient's condition was seriously deteriorated with persisting cyanosis and tachypnea. Arterial blood gas analysis revealed severe acidosis as follows: pH 7.12; P_{O_2} , 74 mmHg; and base excess, -18.0 mEq/l. Serum electrolyte concentration showed no remarkable change. During the following 10 hours, 7% sodium bicarbonate in a total dose of 60 ml was given intravenously without any definite effects. His condition remained still serious and the peritoneal dialysis with 100 ml of Perisolita,* 20 ml of 7% sodium bicarbonate and 1 ml of Aspara-K† was started. Thirty minutes after the dialysis was started, blood gas analysis revealed marked improvement of base deficit, but respiratory acidosis still persisted, as indicated by the following data: pH, 7.20; P_{O_2} , 40 mmHg; P_{CO_2} , 62 mmHg; and base excess, -2.0 mEq/l.

Thereafter, his condition deteriorated progressively and the patient died from lung atelectasis at 5 a.m., June 22, 20 hours after the operation.

Case 2. H.Y. A 75-day-old female baby was admitted on July 13, 1967, with marked cyanosis and tachypnea. Her body weight was only 3,700 g. The transposition of the great vessels was suspected on the basis of plain chest x-ray films.

On the following day, right cardiac catheterization and cineangiography were carried out under general anesthesia and the diagnosis of complete transposition of the great vessels was confirmed. A No. 60 polyvinyl tube was inserted into the right femoral artery for continuous sampling of the arterial blood. At that time, blood gas analysis disclosed severe metabolic acidosis: pH, 7.10; P_{O_2} , 35.0 mmHg; P_{CO_2} , 35.0 mmHg; and base excess, -18.0 mEq/l. Peritoneal dialysis with 50 ml of THAM, 50 ml of Perisolita and 0.5 ml of Aspara-K was started immediately, and then the acid base balance was improved to: pH, 7.25; P_{O_2} , 38.0 mmHg; P_{CO_2} , 36.0 mmHg; and base excess, -9.0 mEq/l. No remarkable change was noted in her serum potassium concentration during the dialysis. Thereafter, the dilatation of the *Foramen ovale* with Rashkind's catheter was attempted under fluoroscopic monitoring, and the procedure seemed successful and her arterial P_{O_2} rose to 78.0 mmHg.

In the morning of the third hospital day, massive bleeding from the site of cannulation of the right femoral artery occurred. Immediately, the wound was reopened and the bleeding was adequately controlled. At that time, about 50 ml of blood were lost and she became seriously ill. Fifty ml of blood were quickly transfused, but her condition remained still serious. Blood gas analysis revealed that she became again acidotic and the data were: pH, 7.15; P_{O_2} , 42.0 mmHg; P_{CO_2} , 42.0 mmHg, and base excess, -16.0 mEq/l. The peritoneal dialysis was

* Solution manufactured by SHIMIZU SEIYAKU Co. Ltd., which contains 0.0152 per cent of magnesium chloride, 0.0331 per cent of calcium chloride, 0.5553 per cent of sodium chloride, 0.5043 per cent of sodium lactate and 1.3 per cent of glucose (w/v).

† Solution manufactured by TANABE SEIYAKU Co. Ltd., which contains 1 mEq/ml of potassium in the form of potassium aspartate.

repeated with 50 ml of THAM, 50 ml of Perisolita and 0.5 ml of Aspara-K. The effect of dialysis was rapid and dramatic and the result of blood gas analysis was improved to pH, 7.42; P_{O_2} , 42.0 mmHg; P_{CO_2} , 30.0 mmHg; and base excess, -3.5 mEq/l. She recovered quickly and appeared well on the following days.

On July 17, the fifth hospital day, marked bradycardia appeared. Pulse rate dropped to 72 per minute. Electrocardiogram showed an A-V block. Intravenous drip infusion of Isopreterenol was started, but did not bring about any effects. She died from heart block in the next morning.

COMMENT

A series of different pathologic processes may produce metabolic acidosis. Marked decrease in peripheral perfusion rate is considered to be one of the commonest factors producing metabolic acidosis. As mentioned above, cyanotic babies are apt to fall into severe metabolic acidosis and often need a rapid correction of derangement in acid-base balance.

According to Astrup *et al.*,¹ correction of metabolic acidosis can be achieved by administration of buffer solution in an appropriate dose given by the formula of $0.3 \times \text{bodyweight in kg} \times \text{base deficit}$. Several workers² accepted it on the basis of their own experiences. In small babies, however, a formula of $0.45 \times \text{bodyweight in kg} \times \text{base deficit}$ ³ seems to be more appropriate than the Astrup's formula. The calculated dose of buffer solution is often so large that its rapid intravenous infusion is harmful to small babies. In such a situation, peritoneal dialysis is the sole safe way, and a dose of buffer materials sufficient to correct severe metabolic acidosis can be given by this method.

Esperanca and Collins⁴ stated that the peritoneal surface area of the newborn infant was about twice as large as that of the adult in proportion to the bodyweight and peritoneal dialysis was twice as efficient in newborn infants as in adults. Seki⁵ reported cyanotic babies with successful recovery from severe metabolic acidosis by peritoneal dialysis. Severe metabolic acidosis in our infants with cyanotic heart diseases was markedly improved by peritoneal dialysis, although they finally died from cardiac insufficiency. In our first case, arterial blood pH was not adequately elevated by peritoneal dialysis because of severe respiratory insufficiency, but the base deficit was markedly improved by the dialysis. Progressive deterioration of the patient's condition after cessation of the dialysis seems to be due to underlying circulatory and respiratory insufficiency. In the second case of ours, improvement of arterial blood pH was slight in degree by the first dialysis, but the base deficit rose from -18.0 to -9.0 mEq/l. The second dialysis, which was carried out after improvement of hemodynamics, markedly corrected the acid-base status: arterial blood pH rose from 7.15 to 7.42, and base deficit was minimized from -16.0 to -3.5 mEq/l.

Our cases reported here proved that rapid correction of severe metabolic acidosis in neonates, especially that with base deficit, could be easily achieved by peritoneal dialysis with buffer solutions without any hazardous complications. It

should however, be kept in mind that the effects were only of short duration, unless the underlying diseases causing metabolic disturbances were adequately controlled.

References

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