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CASE REPORT

Renal Immaturity Mimicking Chronic Renal Failure in an Infant Born at 22 Weeks Gestational Age

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Abstract

Glomerular function shows a progression directly correlated to gestational age (GA) and postnatal age in preterm infants. In preterms, glomerular filtration rate (GFR) is significantly lower than in term infants, and it matures more slowly in the postnatal period. In infants with very low GA, due to decreased GFR, normalization of serum creatinine values may be prolonged during recovery from acute renal failure (ARF). Herein, we report an extremely premature infant with high creatinine values from third week up to third month of life which normalized thereafter mimicking chronic renal failure.

Keywords: Prematurity, renal function, renal failure, renal immaturity, neonatal intensive care unit

INTRODUCTION

Premature kidneys have reduced functionality due to lower number of nephrons, lower renal blood flow, and glomerular and tubular immaturity.^{1–3} Glomerular function shows a progression directly correlated to gestational age (GA) and postnatal age in preterm infants.⁴ In infants with very low GA, due to decreased glomerular filtration rate (GFR), normalization of serum creatinine values may be prolonged during recovery from acute renal failure (ARF).

Herein, we report an extremely low-birth-weight infant born at 22 weeks GA with high creatinine values from third week up to third month of life mimicking chronic renal failure which normalized thereafter.

CASE REPORT

A 560 g female infant was born at 22 gestational weeks and admitted to neonatal intensive care unit due to extreme prematurity and respiratory distress. She was intubated, received surfactant, and was set on mechanical ventilation. Prophylactic antibiotics, as intravenous penicillin G and netilmicin, and total parenteral nutrition were started. On the first day of life, she had hypotension responsive to increased volume

of intravenous fluids (100 mL/kg/day) and dopamine (10 µg/kg/day). On the third day, the patient had lost 12% of her birth weight and total fluids were increased to 140 mL/kg/day. After the fifth day, total daily fluid was increased to 160 mL/kg/day. On the 16th day, the baby appeared dehydrated with weight loss of 7%. Although urine output was normal, renal function tests were in accordance with ARF (Table 1). Renal ultrasonography revealed mildly increased cortical echogenicity on both kidneys with no structural anomalies of the urinary tract. Renal Doppler ultrasonography showed normal flow in renal arteries and veins. Total parenteral nutrition was discontinued, total daily fluid volume was increased up to 230 mL/kg/day, and dopamine infusion (3 µg/kg/min) was started to improve renal blood flow. In contrast to decrease in blood urea nitrogen and sodium values, serum creatinine kept on increasing on the following days up to 2.7 mg/dL. Creatinine values sustained high around 2 mg/dL for the following 3 weeks. On the 43rd postnatal day, the baby was still on mechanical ventilator and chest X-ray was consistent with bronchopulmonary dysplasia, so that we decided to start hydrocortisone 1 mg/kg/day for 10 days and taper thereafter. She was extubated to nasal continuous positive airway pressure on 50th and weaned to supplemental oxygen on 60th postnatal day. Oxygen

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Table 1. The progress of body weight, urine output, daily fluid volumes, serum creatinine, blood urea nitrogen, and sodium levels.

| Postnatal age (day) | 3 | 16 | 21 | 28 | 43 ^a | 70 | 87 | 100 |
|-----------------------------|------|-----|-----|-----|-----------------|------|------|------|
| Weight (g) | 495 | 520 | 560 | 600 | 700 | 1030 | 1450 | 1730 |
| Urine output (mL/kg/h) | 2.5 | 1.2 | 3.5 | 3 | 2 | 2.1 | 2.4 | 2.1 |
| Total daily fluid (mL/kg) | 140 | 200 | 230 | 160 | 130 | 120 | 120 | 120 |
| Serum creatinine (mg/dL) | 0.63 | 2.2 | 2.7 | 2.2 | 1.8 | 0.7 | 0.74 | 0.56 |
| Blood urea nitrogen (mg/dL) | 40 | 90 | 66 | 37 | 18 | 24 | 18 | 20 |
| Serum sodium (mEq/L) | 141 | 163 | 137 | 132 | 138 | 140 | 145 | 140 |

Note: ^aHydrocortisone administration.

was discontinued on the 65th day. Decline in serum creatinine value was accelerated around that time as well (Table 1) and reached normal level on the 70th postnatal day. The baby was discharged on the 100th postnatal day. She is now 7 months old and had normal blood pressure and renal function test results during post-discharge follow-up.

DISCUSSION

ARF is a common problem in the neonatal intensive care unit. The reported incidence of neonatal ARF ranges from 8% to 24% of all admissions with these numbers being probably underestimated, because many cases of nonoliguric neonatal ARF are excluded.⁵ In newborns, kidneys have very low GFR which is normally sufficient for growth and development. Under pathologic conditions newborn kidneys are at risk of imbalance. In preterms, renal function is even more reduced rendering them more vulnerable to ARF. Moreover, several factors affecting renal function such as hypoxia, hypotension, hypovolemia, and administration of nephrotoxic drugs occur in combination in preterm infants. Fivefold higher incidence of ARF was reported in patients with birth weight <1000 g.⁶ The infant in our case had many risk factors, the most important of which is high-grade prematurity and extremely low birth weight. Resuscitation at birth, respiratory distress syndrome, hypotension on the first day of life, increased insensible water loss due to immaturity of the skin and mechanical ventilation requirement, use of nephrotoxic aminoglycosides and vancomycin, and finally sepsis acted in concert in development of ARF.

Nephron formation is dependent on GA and babies acquire their final number of nephrons only after 34–36 weeks of GA.¹ Due to incomplete nephronogenesis, GFR in preterms is significantly lower than in term infants, and it matures more slowly in the postnatal period. It was shown that glomerular function shows a progression directly correlated to GA and postnatal age in preterm infants born ≤32 weeks gestation.^{4,7} In preterm newborns, serum creatinine increases during the first 3–4 days of life and gradually decreases. In neonates with a lower GA, serum creatinine increases to higher values and physiological decrease occurs more

gradually.^{4,8} Normalization of serum creatinine values may take longer time in extremely premature babies after an insult causing pathological decrease in GFR. It was reported that ibuprofen-associated alterations in renal function in preterm babies with patent ductus arteriosus can be sustained up to 1 month of life.⁹ We believe immaturity of renal function and low GFR in this extremely premature baby led to high serum creatinine levels for weeks after the initial insult. Another interesting finding in our case was acceleration in the recovery of creatinine values after administration of hydrocortisone. Endogenous and exogenous glucocorticoids play a role in the maintenance of glomerular filtration by increasing renal blood flow and functional glomerular surface area. Antenatal and postnatal steroids are known to have maturative action on the premature kidney with effects at both glomerular and tubular level.¹⁰

In conclusion, in extremely premature infants recovery period after ARF can be prolonged and misdiagnosed as chronic renal failure.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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