A neonate with acute kidney injury

Farzana Ahmed, Sanjoy Kumar Dey, Md. Abdul Mannan and Mohammod Shahidullah

Case Presentation

Dr. Rezaul Hayat: An outborn, 2 hours old male newborn was admitted at the neonatal intensive care unit due to delayed cry after birth and respiratory distress soon after birth. The baby was born at 39 weeks of gestation at another hospital to a 30 year old primi mother by lower uterine cesarian section due to the obstructed labor. Although the mother was not in regular antenatal checkup. Her pregnancy period was uncomplicated.

The baby was limp and blue immediately after birth and was managed accordingly by the attending physician and was referred to the Neonatal Intensive Care Unit of Bangabandhu Sheikh Mujib Medical University for better management. APGAR score was not documented. On initial examination on arrival, the baby was lethargic, eu glycemic, vitally stable except tachypnea but maintaining saturation with oxygen inhalation at the rate of 2 liter per min. Initial septic screening was positive. So, the first line antibiotics- injection ampicillin and gentamicin were started. Gradually his respiratory distress increased. So, the baby was put on mechanical ventilator at 16 hours of age. Antibiotics were changed due to poor response to medications and injection meropenum and amikacin were started. Gradually his respiratory distress improved and was continued for total 14 days.

From second day onward, the baby developed edema and unusual weight gain. The baby was normotensive with adequate urine output. There was no sign of heart failure. Investigation report revealed high level of serum creatinine which further increased on the following day despite of renal dose adjustment of medication (Figure 1).

The serum calcium level was low which was corrected by the calcium supplementation (Table 1). The serum sodium level was also low despite of fluid restriction. So, further total fluid restricted to insensible loss (30 mL/kg) plus replacement of equal amount of the urine output. Gradually, his condition was improved and was weaned from the mechanical ventilator on day 4 of life, edema subsided, serum creatinine level decreased, hyponatremia corrected. So, the baby was discharged from hospital on 18th days of age.

Provisional Diagnosis

Term with appropriate gestational age with perinatal asphyxia with neonatal sepsis with acute kidney injury.

Differential Diagnosis

Dr. Farzana Ahmed: The newborn baby presented with acute kidney injury. The cause of acute kidney injury might be due to perinatal asphyxia or neonatal sepsis. The prerenal, postrenal causes and congenital anomaly in the urinary tract might also cause the acute kidney injury in this age group which also should be excluded.

Acute kidney injury due to perinatal asphyxia

Acute kidney injury can occur in 50-60% neonates with severe perinatal asphyxia. APGAR score was less than 4 and the base deficit >15 mEq/L at one hour after birth are consider as independent risk factors for development of acute kidney injury in newborn. In perinatal asphyxia, the redistribution of blood flow occurs to provide the optimal blood circulation to the brain, heart and adrenal glands, circulation decrease in skin and splanchnic vessels. This mechanism is called “Diving Reflex”. By this mechanism, acute tubular necrosis may occur which leads to acute kidney injury in newborn.

Acute kidney injury due to neonatal sepsis

Sepsis contributes up to 78% cases of the acute kidney injury in newborn. Preterm low birth weight newborns with sepsis are vulnerable to develop acute kidney injury than their term counterpart. Previously, it was assumed that hypotension associated with systemic inflammatory responses cause the renal failure. Now-a-days it had been observed that renal failure might occur despite of normal blood pressure suggesting direct kidney injury in sepsis by effects on renal microvasculature.
**Dr. Hayat’s Diagnosis**

Term with appropriate gestational age with perinatal asphyxia with neonatal sepsis with acute kidney injury.

**Discussion**

*Dr. Ahmed:* With proper antenatal and postnatal care, accelerated change in the child survival is observed over the last decade but neonatal mortality rate is still a burning issue. Global neonatal mortality rate fell from 37 deaths per 1,000 live births in 1990 to 19 per 1,000 in 2016. In Bangladesh, neonatal mortality rate is 20 per 1,000 live births. Acute kidney injury is one of the common preventable and treatable pathological conditions in newborn which occurs in 1-24% newborn admitted in the neonatal intensive care unit. The incidence of acute kidney injury in Bangladesh is 14.1% in newborn admitted at the neonatal intensive care unit, among them, mortality rate is 17.2%.

The causes of neonatal acute kidney injury are multiple and the three classic categories into which they are divided are: prerenal, renal (intrinsic or organic) and postrenal. Prerenal acute kidney injury (also known as vasomotor nephropathy) is the most common form in the neonatal period, with an incidence of 85% of cases of acute kidney injury. Acute kidney injury in newborn admitted at the neonatal intensive care unit, among them, mortality rate is 17.2%.

Dr. Iffat Rahman: Can prerenal causes of acute kidney injury cause intrinsic renal failure? If yes then how?

*Dr. Ahmed:* Although prerenal form of acute kidney injury, post-glomerular blood flow remains sufficient but following a serious and prolonged ischemia, a series of anatomic alterations might takes place which may cause acute tubular necrosis.

Dr. Rumman Idris: How prerenal causes can be distinguished from the intrinsic renal causes of acute kidney injury?

*Dr. Ahmed:* By the following ways, prerenal causes

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<td><strong>Investigation profile</strong></td>
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<tr>
<td>Investigation</td>
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<tr>
<td>Hemoglobin (g/dL)</td>
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<td>White cell count (x10⁹/L)</td>
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<td>C-Reactive protein (mg/dL)</td>
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<td><strong>Difference between prerenal and intrinsic renal failure</strong></td>
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<td>Urine osmolality (mOsmol/Kg)</td>
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<td>Urine sodium (mmol/L)</td>
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<td>Urine/plasma urea</td>
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<tr>
<td>Urine/plasma creatinine</td>
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<tr>
<td>Urine/plasma osmolality</td>
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<td>FENa (Fractioned excretion of sodium)</td>
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<td>Renal failure index</td>
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Figure 1: Serum creatinine level (mg/dL)
can be distinguished from postrenal causes (Table II).

Dr. Sumon Sorkar: Is there any rule of fluid challenge test in newborn?

Dr. Ahmed: Fluid challenge test can differentiate prerenal from the intrinsic renal causes of acute renal failures but the fluid should be given slowly in newborn specially in preterm babies. 24-26

Dr. Bipin Karki: Is there any rule of ionotopic agents test for acute kidney injury in newborn?

Dr. Ahmed: Although it is augmented that low dose dopamine (0.5-2.0 μg/kg/min) renal vasodilatation and increase salt water excretion but no study yet done on this topic in neonate. Furthermore, α receptors are relatively high in preterm newborn, so, even low dose of dopamine might put them in risk of systemic hypertension. 24, 31-33

Dr. Sorbori Saha : What is the outcome of newborn suffered from acute kidney injury?

Dr. Ahmed: Both immediate and long term outcome in babies with acute kidney injury are not good. In many long term prospective studies proved that even those neonate who had achieved full recovery initially, later developed chronic kidney disease. So, to combat this fatal disease, recent KDIGO practice guidelines recommend that all patients who experience acute kidney injury should be evaluated after 3 months for new onset or worsening of chronic kidney disease. 34-37

Final Diagnosis

Term with appropriate gestational age with perinatal asphyxia with neonatal sepsis with acute kidney injury.

References


