23.1 Introduction

The neonatal period, defined as the first 4 weeks of life, is probably the most vulnerable period of life. The sudden change from intrauterine to postpartal life may lead to acute presentation of pre-existing renal diseases compensated by the mother prenatally. Birth may be complicated and lead to organ failure, or an acquired disease may start so early in life. The management of a baby with neonatal renal failure is a therapeutic challenge, and a well-equipped neonatal intensive care unit (NICU) is needed. This chapter describes the diagnostic and possibly therapeutic methods that can be offered by pediatric radiologists to these severely compromised neonates in an intensive care situation with sometimes limited potential for investigation.

23.2 Prenatal Situation

Significant fetal urine production starts at approximately 12 weeks of gestation and reaches values of 30–60 ml/h at the end of pregnancy. Fetal urine is a major constituent of amniotic fluid. Oligohydramnios may indicate fetal renal insufficiency such as in bilateral renal agenesis, which leads to Potter syndrome and includes pulmonary hypoplasia. Nephrogenesis has a centrifugal pattern and is completed at 36 weeks of gestation. Destroyed nephrons cannot be replaced by new filtering units. Thus, prematurely born infants are the only humans able to harvest new nephrons after birth. Fetal renal blood flow (RBF) and glomerular filtration rate (GFR) are low. Plasma renin activity is high, and production of renal prostaglandins is increased. Fetal homeostasis is maintained by the placenta and the maternal renal function.
Changes after Birth

There is a sharp rise in RBF and GFR after birth. Plasma renin activity and production of renal prostaglandins decrease, but remain elevated compared to older children. These changes with an increase in renal blood-flow velocity and a decrease in renal vascular resistance can be demonstrated by Doppler ultrasound in term and preterm neonates (Visser et al. 1992; Van de Bor 1995). Maturation of renal function is mostly due to enlargement of the glomerular capillary surface area, a rise in ultrafiltration pressure, and further development of tubular function. On the first day of life, serum creatinine roughly equals maternal values even in cases with fetal renal failure. Prematurely born infants have higher levels of creatinine, and their postnatal increase in creatinine clearance is delayed compared to term neonates. This is valid especially in very low birthweight infants (Bueva and Guignard 1994; Drukker and Guignard 2002). This may reflect the lower GFR and the mean blood pressure of premature neonates being too low to eliminate the excess creatinine transferred by the mother. In addition, tubular reabsorption of creatinine through the leaky immature tubules occurs (Guignard and Drukker 1999a). Despite the various processes of adaptation and maturation after birth, the newborn is in a physiologic state of renal insufficiency with hyperactive vasoactive systems and a GFR as low as 20 ml/min for 1.73 m² in term infants (Guignard et al. 1991; Toth-Heyn et al. 2000). This situation is appropriate for normal life after birth, but renal functional reserve is limited in disease states. Therefore, potentially nephrotoxic agents such as contrast media should be avoided in neonates. The dosage of drugs must carefully be adjusted to the actual renal function.

Urine Production after Birth

First voiding often takes place in the delivery room. Healthy newborns pass urine during the first 24 h of life in 92%–97% of cases, and nearly all void within 48 h (Clark 1977; Wang and Huang 1994). Normal urine volume is 1–3 ml/kg and h. Thus, polyuria is defined as urine output of more than 4 ml/kg and h and oliguria as less than 0.5–1.0 ml/kg and h. Acute renal failure (ARF) is rare in apparently healthy neonates with a normal fetal ultrasound. The incidence of ARF in a NICU ranges from 1.5% to 23%. Oligoanuria is the leading symptom in about 40% of cases, and 60% suffer from non-oliguric ARF (Andreoli 2004; Hentschel et al. 1996; Karlowicz and Adelman 1995; Kupferman 1994; Stapleton et al. 1987).

Diagnostic Workup

If oligoanuria is recognized, many urgent questions arise, and the potentially underlying conditions should be recognized (Table 23.1). Before starting extensive investigations, pitfalls should be excluded. Previous voiding may have been missed; urine collection may be inappropriate with loss around the collection bag. Urine can be mixed with stool, and a previously inserted bladder catheter may be displaced or blocked. If true oligoanuria is combined with an increased serum creatinine, renal failure is proven. It is traditionally classified as prerenal, intrinsic, or postrenal failure. The term “prerenal” or “functional” implies a systemic disease with normalization of urine flow and of renal function after appropriate therapy. Intrinsic renal failure occurs in congenital or acquired renal diseases or by transition from prolonged prerenal failure. Chronic renal failure may be the long-term consequence. Postrenal failure is mostly found in obstructive uropathy.

Clinical and Laboratory Investigations

History

Evaluation or re-evaluation of family history and of pregnancy may reveal previous cases of fetal or neonatal death suggestive of inherited disorders or syndromes. Administration of angiotensin-converting enzyme inhibitors (ACE-inhibitors) for hyperten-