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Management of acute renal failure in the newborn

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KEYWORDS
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Summary Acute renal failure is common in the neonatal period. It is usually manifest by abnormal biochemistry and decreased urine output (<1 ml/kg/h), but non-oliguric renal failure is also common. A detailed understanding of the common pathophysiological mechanisms is rarely needed but an understanding of the common aetiologies (pre-renal, renal and post-renal) will enable the clinician to approach the problem in a logical manner. A standard approach to fluid and electrolyte management is described, along with a practical approach to the investigation and management of renal failure. A working understanding of the principles of peritoneal dialysis is important and a brief overview of the role of haemodialysis in neonatal renal failure is provided.

Introduction

Impairment in renal function, diagnosed by abnormal biochemistry or decreasing urine output, is common in the neonatal period. When it develops, an appreciation of the likely aetiologies and a working knowledge of normal renal physiology should enable the clinician to approach the problem in a logical manner. This paper reviews the definition, epidemiology, and aetiology of acute renal failure (ARF), describes a standard approach to fluid and electrolyte management in the newborn, emphasises the importance of recognising early signs of ARF, and describes management options and prognosis.

Definition

The kidney has many functions, including controlling the extracellular fluid (ECF) space through the processes of urinary excretion and tubular resorption. A functional definition of impairment or failure includes: (1) an increase in constituents that normally 'leak' from the intracellular space (potassium, phosphate, etc.); (2) a build-up in the normal end-products of nitrogen metabolism either from the diet (parenteral and enteral) or normal tissue catabolism, reflected in a rising creatinine; and (3) inadequate tubular resorption of sodium, bicarbonate loss, and inadequate excretion of water, resulting in oedema. Depending on the stage and clinical situation, not all of these features will be present. In babies who are also sick, leakage of intracellular electrolytes and endogenous catabolism exacerbate the biochemical effects. Defining ARF in terms of urine output excludes those who have non-oliguric renal failure but in those that are oliguric, urine output typically decreases to <0.5 mL/kg/h.

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Epidemiology

Mild ARF resulting from insufficient intravascular filling is easy to reverse. Similarly, non-oliguric renal failure is typically relatively easily managed conservatively. The epidemiology of this level of renal failure is not comprehensively described. Studies suggest an ARF prevalence rate of 3–8% of neonatal unit admissions.[1,2] Of these, about one-third are preterm.[1] With appropriate management, many cases are reversible but longer-term problems are still frequently observed,[3] and 25–50% of babies with ARF will die.[1,4] At the severe end of the spectrum, where referral for dialysis has been recorded, 20% of all cases were newborns, the majority following cardiothoracic surgery, which also accounted for the majority of the neonatal deaths. The neonatal and infant age group had the highest incidence of acute renal failure referred for dialysis when corrected for age-specific population (Fig. 1).

A level II unit will have a very different experience from a large level III unit, and even this will vary if, for example, it is part of a neonatal surgical service or a tertiary fetal assessment unit, supported by a tertiary paediatric nephrology unit.

Aetiology and assessment

The traditional classification identifies prerenal, intrinsic renal, and postrenal 'obstructive' aetiologies. Fig. 2 shows that the predominant pathophysiology affecting the newborn is prerenal compared to the other paediatric age groups.

The presence of oliguria or anuria usually signifies a pre- or postrenal aetiology, but a thorough history and examination, including biochemical markers in the serum and urine along with urinalysis, will identify the most likely aetiologies. Common aetiologies are shown in Box 1. A good history and examination (Box 2) is the starting point and might identify the presence of common syndromic abnormalities, such as the oligohydramnios deformation sequence (Potter's syndrome) or other congenital abnormalities. Careful examination of the genital and perianal regions must be performed to check for any abnormal surface anatomy. Severe dehydration or excess gastrointestinal losses should be apparent in the history.

Haemorrhage will usually be obvious when it is secondary to acute placental abruption or bleeding from the umbilical cord for example, but significant blood loss might not always be apparent either before or after birth. Feto-maternal haemorrhage can be massive and unrecognised, and significant blood can be lost in subgaleal haemorrhage before it becomes clinically apparent. Most of these infants will present with signs of shock and many might go on to develop ARF.[5] Third-space losses as a result of sepsis are very common in sick infants and many will need additional fluid boluses (20 mL/kg).

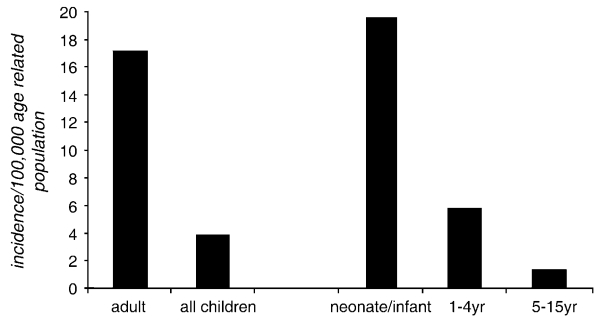

Figure 1 Incidence of acute renal failure, corrected for age-related populations.[4]

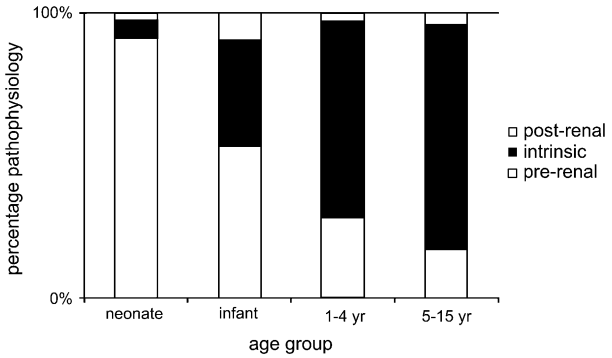

Figure 2 Pathophysiology of acute renal failure by age.[4]

ARF can occur in situations where intravascular fluid volume is normal but there is inadequate renal perfusion from hypotension. Cardiac 'pump' failure might occur with congenital heart disease (and is particularly common during and following surgical correction) but is usually secondary to some other condition, such as perinatal ischaemia/hypoxia, sepsis (especially Gram-negative and group B streptococcal), or a large patent ductus arteriosus (PDA) in a preterm infant. Inotropic agents are frequently needed. Occasionally, both cardiac function and intravascular volume are normal, but marked peripheral vasodilatation results in hypotension that compromises adequate renal perfusion. Careful vasoconstriction using dopamine, dobutamine, noradrenaline or even vasopressin may be helpful. Excessive vasoconstriction may worsen renal perfusion. Cardiac tamponade (pericardial effusion or air leaks) usually presents with other signs of decreased cardiac output long before a diagnosis of ARF is made.

Assess by examining the urine. Acute tubular necrosis (ATN) is common following perinatal ischaemia/hypoxia. One report suggests that over half of all severe cases develop non-oliguric ARF.[6] ATN can be drug-induced, although aminoglycoside-induced renal toxicity is now rare, and most non-steroidal anti-inflammatory drug (NSAID)-associated renal failure (which is common[7]) usually results from impairment of intrarenal prostaglandin homeostasis rather than true ATN[8] (see below). Interstitial nephritis is very rare. There are occasional case reports of maternal NSAID usage causing neonatal ARF.[9,10] The urinary β2-microglobulin/creatinine ratio and fractional excretion appear to be more sensitive and specific for the early detection of proximal tubular renal dysfunction than standard tests,[11] but commercial 'dipstick' tests, urine biochemistry, and microscopy are usually sufficient in clinical practice.

Several conditions cause ARF through multiple mechanisms. Sepsis can cause hypotension, third-space loss,