

key points

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About 20% of all adult emergency admissions are affected by acute kidney injury (AKI) and the mortality rate is almost 25%. One estimate is that AKI, excluding AKI in the community, causes more than 10,000 preventable deaths a year in England.

AKI represents a wide spectrum of injury to the kidneys, not just kidney failure, the vast majority of AKI cases start with an illness in the community. The diagnosis of AKI is currently based on a rise in creatinine, and/or reduced urine output, eGFR is not used in the diagnosis of AKI. AKI may only be seen as a small rise in creatinine. The rise needs to be on a short, acute timescale to distinguish it from the slower steady rise of progressive chronic kidney disease (CKD). AKI episodes are common in patients with CKD. Such episodes are an important factor in the progression of CKD.

In AKI, loss of kidney function contributes to morbidity and mortality. Patients die from AKI rather than with AKI as a complication of an underlying illness. The metabolic work of the kidney tubule imposes major energy demands and the tubule normally operates under conditions of borderline oxygen supply. A modest fall in blood pressure of about 15-20 mmHg can lead to a range of tubular injury. The definition of AKI now includes any adult with $\geq 26 \mu\text{mol/L}$ rise in creatinine from baseline over 48 hours or less, or $\geq 50\%$ rise in creatinine from baseline known or presumed to have occurred over seven days or less.

The causes of AKI have traditionally been divided into pre-renal, intrinsic renal, and post-renal or obstructive. Any cause of hypovolaemia or reduced effective circulating volume can inflict ischaemic tubular injury. Intrinsic renal causes of AKI can be thought of as conditions manifested as direct injury to the glomerulus, the tubule, the interstitium, or the vasculature. Post-renal AKI refers to any obstruction within the tubule itself, ureters, bladder or the bladder outflow/urethra. A key step in the initial assessment is to identify the likely cause of AKI. Following an episode of AKI, focus of care should switch to monitoring and prevention of further episodes.

In patients with known or suspected AKI all nephrotoxic drugs should be suspended. The use of NSAIDs should be avoided, ACE inhibitors and angiotensin receptor blockers should be suspended during any episode of vomiting and/or diarrhoea.