[HOSPITAL / HEALTH AUTHORITY NAME]

ACUTE KIDNEY INJURY, OLIGURIA, AND DANGEROUS ELECTROLYTE DISORDERS PATHWAY

Protocol 28: Rapid Recognition, Cause Identification, Fluid and Medication Stewardship, Electrolyte Rescue, Renal-Replacement Escalation, Monitoring, and Safe Disposition

DRAFT FOR EMERGENCY MEDICINE, INTERNAL MEDICINE, NEPHROLOGY, UROLOGY, CRITICAL CARE, CARDIOLOGY, ENDOCRINOLOGY, PAEDIATRICS, OBSTETRICS, ONCOLOGY, TOXICOLOGY, PHARMACY, LABORATORY, NURSING, EMS, TRANSFER, AND CLINICAL-GOVERNANCE REVIEW

STATUS: This is a draft clinical-governance document. Exact fluid boluses and maintenance regimens, potassium and sodium treatment preparations, concentrated-electrolyte administration, calcium and magnesium products, medication-withholding rules, renal-dose adjustments, laboratory intervals, ultrasound access, nephrology and urology escalation, dialysis indications and modalities, paediatric and pregnancy pathways, transplant procedures, toxicology arrangements, discharge follow-up, and transfer logistics must be reconciled with current national guidance, local formulary, laboratory capability, specialist availability, and approved linked protocols before implementation.

IMMEDIATE SAFETY RULE: Acute kidney injury is a dynamic syndrome, not a creatinine number. Treat life-threatening hyperkalaemia, pulmonary oedema, shock, severe acid-base disturbance, symptomatic sodium or calcium disorder, and obstruction immediately while determining the cause. Do not delay emergency imaging or another life-saving intervention solely because kidney function is impaired. A patient may have severe AKI before creatinine has risen, especially when urine output is falling.

Document control	Details
Document owner	Emergency Department / Medical Services Directorate / Nursing Services / Clinical Governance
Clinical leads	Emergency Medicine; Internal Medicine; Nephrology; Critical Care
Supporting departments	Urology; Cardiology; Endocrinology; Paediatrics; Obstetrics; Oncology / Haematology; Toxicology; Pharmacy; Laboratory; Radiology; EMS; Transfer Coordination
Applies to	Adults, adolescents, children, and pregnant or postpartum patients with suspected acute kidney injury, reduced urine output, obstruction, acute fluid overload, severe acid-base disturbance, or dangerous electrolyte abnormality
Linked protocols	Shock; Sepsis; Altered Mental Status; Arrhythmias; Severe Hypertension; Vomiting / Dehydration / Electrolytes; Diabetic Emergencies; Abdominal / Flank Pain; Poisoning / Overdose; Major Haemorrhage; Pregnancy Emergencies; Paediatric Emergency Assessment; Renal Failure / Dialysis Emergencies; Transfer
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1. Purpose

To provide a standardized emergency-department pathway for early detection, stabilization, cause identification, and treatment of acute kidney injury (AKI), oliguria or anuria, acute urinary obstruction, fluid overload, metabolic acidosis, and dangerous potassium, sodium, calcium, magnesium, and phosphate disorders. The protocol emphasizes simultaneous resuscitation and diagnosis, measured fluid therapy, nephrotoxin and medication stewardship, urgent treatment of reversible threats, timely nephrology / urology / critical-care involvement, appropriate renal-replacement escalation, and safe follow-up after AKI.

2. Scope

This protocol applies to patients with new or worsening kidney dysfunction, reduced urine output, unexplained electrolyte or acid-base disturbance, rhabdomyolysis, tumour lysis, suspected nephritis or thrombotic microangiopathy, urinary obstruction, or complications that may require urgent kidney replacement therapy. It supports initial emergency management but does not replace dedicated protocols for established dialysis complications, toxic alcohol / lithium / salicylate poisoning, diabetic ketoacidosis, major burns, severe sepsis, tumour lysis prophylaxis, or paediatric and obstetric critical care.

3. Core policy statements

- Measure creatinine and compare it with a reliable baseline, but also measure and trend urine output. Creatinine may lag behind injury and may be misleading after fluid loading, muscle loss, pregnancy, or critical illness.
- Identify and document the likely cause or causes of AKI. Use a practical framework of reduced perfusion, intrinsic kidney injury, obstruction, and mixed disease; do not label all AKI as dehydration.
- Obtain urinalysis promptly in suspected AKI. Haematuria plus proteinuria without infection or catheter trauma should trigger consideration of glomerulonephritis, vasculitis, or another intrinsic renal process.
- Use point-of-care ultrasound, bladder scanning, formal renal ultrasound, or CT according to the clinical question. Suspected infected obstruction, bilateral obstruction, or obstruction of a solitary kidney requires immediate urological escalation.
- Treat shock with appropriately selected crystalloid boluses and repeated reassessment. Avoid both under-resuscitation and unmeasured fluid accumulation. Pulmonary oedema is not treated with routine fluid challenges.
- Do not routinely use low-dose dopamine, mannitol, or loop diuretics to reverse AKI. Diuretics may be used to manage fluid overload in a patient who can respond, but they do not substitute for dialysis when urgent indications exist.
- Review all prescribed, over-the-counter, herbal, and contrast exposures. Temporarily withhold or adjust medicines that worsen renal perfusion, potassium, volume status, or drug accumulation when clinically appropriate, and document a restart plan.
- A suspected falsely high potassium should be repeated urgently, but emergency treatment must not be delayed when potassium is severely elevated, ECG changes are present, or the patient is unstable.
- In hyperkalaemia, cardiac membrane stabilization, intracellular potassium shift, and potassium removal are separate tasks. Calcium does not lower potassium; insulin and beta-agonists do not remove it.
- After insulin-glucose treatment for hyperkalaemia, monitor glucose for delayed hypoglycaemia, especially in kidney failure, low body weight, non-diabetic patients, or low pretreatment glucose.
- Treat symptomatic hyponatraemia according to neurological severity rather than sodium concentration alone. Prevent overcorrection and have a rescue plan for an unexpectedly rapid sodium rise.
- Correct hypernatraemia with attention to duration, volume status, free-water deficit, ongoing losses, and urine output. Restore circulation first when shock is present, then correct free water in a controlled manner.
- Correct magnesium when treating refractory hypokalaemia or hypocalcaemia. Concentrated potassium, calcium, magnesium, phosphate, and hypertonic saline require approved preparations, infusion pumps, line labeling, and independent checking.
- Discuss potential kidney replacement therapy immediately when refractory hyperkalaemia, severe acidosis, uraemic complications, pulmonary oedema / fluid overload, certain poisonings, or another urgent indication is present. Do not wait for an arbitrary creatinine or urea threshold.
- Do not delay emergency contrast-enhanced imaging when delay is likely to cause greater harm. Use the minimum appropriate contrast dose and provide individualized hydration and follow-up when feasible.
- All patients with AKI require a documented recovery and follow-up plan, including repeat kidney function, medication reconciliation, blood pressure, urinalysis or proteinuria follow-up when indicated, and advice on dehydration and NSAID avoidance.

4. Definitions and staging

Term	Operational definition
AKI detection	Any of: serum creatinine rise at least 26 micromol/L within 48 hours; creatinine at least 1.5 times baseline known or presumed within 7 days; or urine output below 0.5 mL/kg/hour for more than 6 hours in adults.
Stage 1 AKI	Creatinine 1.5-1.9 times baseline or rise at least 26 micromol/L; or urine output below 0.5 mL/kg/hour for 6-12 hours.
Stage 2 AKI	Creatinine 2.0-2.9 times baseline; or urine output below 0.5 mL/kg/hour for at least 12 hours.
Stage 3 AKI	Creatinine at least 3 times baseline; or creatinine at least 354 micromol/L with an acute rise; or kidney replacement therapy started; or urine output below 0.3 mL/kg/hour for at least 24 hours; or anuria for at least 12 hours.
Oliguria	Urine output below 0.5 mL/kg/hour. Confirm measurement accuracy, duration, catheter patency, fluid status, and obstruction risk.
Anuria	No urine or near-complete absence of urine. Treat as a time-critical finding until obstruction, shock, vascular catastrophe, and severe intrinsic renal disease are addressed.
Hyperkalaemia	Mild 5.5-5.9 mmol/L; moderate 6.0-6.4 mmol/L; severe at least 6.5 mmol/L. ECG toxicity or instability overrides the numeric category.
Severe hypokalaemia	Potassium below 2.5 mmol/L or any level associated with arrhythmia, marked weakness, paralysis, rhabdomyolysis, or respiratory compromise.
Hyponatraemia	Sodium below 135 mmol/L. Severe or moderately severe neurological symptoms require immediate treatment regardless of the exact concentration.

Term	Operational definition
Hypernatraemia	Sodium above 145 mmol/L. Moderate or severe hypernatraemia, neurological symptoms, impaired thirst, high ongoing water loss, or rapid onset requires monitored correction.
Dangerous calcium / magnesium disorder	An abnormality causing seizure, tetany, arrhythmia, haemodynamic instability, altered consciousness, marked weakness, or another organ complication; ionized levels are preferred when albumin or pH is abnormal.

5. Roles and accountability

Role	Minimum responsibility
Triage / first-contact clinician	Recognize oliguria, anuria, shock, pulmonary oedema, weakness, arrhythmia, altered consciousness, seizure, toxic exposure, pregnancy, transplant status, dialysis history, and need for immediate ECG and blood testing.
Lead ED clinician	Direct ABCDE care; stage AKI; identify likely cause; prescribe fluid, electrolyte, and acid-base treatment; activate hyperkalaemia, obstruction, sepsis, toxicology, critical-care, nephrology, urology, and transfer pathways.
Nursing team	Provide monitoring; obtain accurate weight and urine output; administer high-alert infusions with required double checks; monitor glucose after insulin; track fluid balance, ECG, neurological state, and response; escalate deviations promptly.
Nephrology / critical care	Support severe or unexplained AKI, refractory complications, kidney replacement therapy, difficult volume management, glomerular / vascular emergencies, transplant recipients, and high-risk electrolyte correction.
Urology / radiology	Provide urgent decompression planning and imaging for obstruction, pyonephrosis, solitary-kidney obstruction, or urinary-tract injury.
Pharmacy / laboratory	Support renal-dose adjustment, nephrotoxin review, rapid and reliable electrolyte / blood-gas testing, haemolysis alerts, concentrated-electrolyte safety, and timely repeat testing.
Receiving / transfer team	Accept responsibility explicitly and ensure uninterrupted monitoring, infusions, airway / oxygen support, glucose checks, and escalation during transport.

6. Pathway activation and triage

Category	Operational criteria
RED / immediate resuscitation	Hyperkalaemic ECG change or potassium at least 6.5 mmol/L; malignant arrhythmia; shock; acute pulmonary oedema; severe symptomatic sodium or calcium disorder; seizure / coma; anuria with instability; severe acidosis; suspected renal vascular catastrophe; dialysis indication.
ORANGE / very urgent	Stage 2-3 AKI; rapidly rising creatinine; oliguria for at least 6 hours; transplant recipient with AKI; suspected infected obstruction; rhabdomyolysis or tumour lysis; haematuria plus proteinuria; severe hypertension with renal injury; moderate hyperkalaemia.
YELLOW / urgent	Stage 1 AKI without instability; mild-moderate electrolyte disturbance; dehydration with preserved perfusion; urinary retention without sepsis or upper-tract compromise; stable medication-related AKI requiring observation and repeat testing.
GREEN / lower acuity only after screening	Minor stable laboratory abnormality without AKI, symptoms, ECG change, impaired urine output, volume disturbance, high-risk comorbidity, toxic exposure, pregnancy, or follow-up barrier. Clinician review remains required.

7. First 10 minutes: parallel action

1. Begin ABCDE assessment, cardiac monitoring, pulse oximetry, temperature, frequent vital signs, and immediate point-of-care glucose. Obtain a 12-lead ECG when potassium, calcium, magnesium, acidosis, weakness, collapse, or arrhythmia is possible.

2. Establish IV access; obtain an accurate weight or best documented recent weight. Send venous blood gas, urea, creatinine, sodium, potassium, chloride, bicarbonate, glucose, calcium, magnesium, phosphate, CBC, and urinalysis. Add lactate, CK, urate, osmolality, cultures, pregnancy test, toxicology, haemolysis studies, or autoimmune tests according to presentation.
3. If severe hyperkalaemia or a toxic ECG is present, give IV calcium and begin potassium-shifting therapy immediately according to the approved local algorithm. Do not wait for a repeat sample when the result is clinically credible and the patient is at risk.
4. Assess perfusion and congestion. Give a measured crystalloid bolus only when hypovolaemia or shock is likely, then reassess. If pulmonary oedema is present, support oxygenation / ventilation, avoid routine fluid loading, and escalate for diuresis, vasodilator therapy, or dialysis as appropriate.
5. Measure and document urine output. Check catheter patency when present; use bladder scan when retention is possible. Insert a urinary catheter only when accurate output or decompression is clinically necessary and no contraindication exists.
6. Look immediately for obstruction, sepsis, haemorrhage, cardiogenic shock, rhabdomyolysis, tumour lysis, nephrotoxic exposure, glomerulonephritis / vasculitis, thrombotic microangiopathy, pregnancy-related disease, transplant complication, or a dialysable poisoning.
7. Review recent and current medicines, including NSAIDs, ACE inhibitor / ARB, diuretics, SGLT2 inhibitor, metformin, potassium supplements, trimethoprim, spironolactone, nephrotoxic antimicrobials, chemotherapy, lithium, herbal products, and recent contrast.
8. Contact senior ED, nephrology, critical care, urology, paediatrics, obstetrics, oncology / haematology, toxicology, or transfer coordination early when a time-critical cause or dialysis indication is possible.

8. Immediate stabilization: ABCDE

8.1 Airway and breathing

- Protect the airway in coma, recurrent seizure, severe uraemic encephalopathy, vomiting with impaired reflexes, or respiratory fatigue. Treat pulmonary oedema with upright positioning, oxygen for hypoxaemia, non-invasive ventilation when appropriate, and early critical-care support.
- Kussmaul breathing may be compensatory in severe acidosis. Intubation can precipitate rapid deterioration if minute ventilation falls; use senior airway planning and maintain adequate ventilation.
- Consider pulmonary-renal syndrome when hypoxaemia or haemoptysis accompanies haematuria, proteinuria, or rapidly progressive AKI.

8.2 Circulation

- Assess pulse, blood pressure, capillary refill, JVP, lung fields, oedema, skin temperature, perfusion, bedside ultrasound findings, and urine output. Distinguish intravascular depletion from venous congestion whenever possible.
- Use small, measured crystalloid aliquots with a defined reassessment endpoint in frailty, heart failure, liver disease, pregnancy, or advanced CKD. Stop fluid loading when perfusion no longer improves or congestion develops.
- Treat sepsis, haemorrhage, anaphylaxis, cardiac failure, hypertensive emergency, or another cause of shock in parallel. Use vasopressors after appropriate fluid assessment when distributive shock persists.

8.3 Disability and exposure

- Record GCS / AVPU, pupils, focal deficit, seizure, neuromuscular weakness, tetany, and serial neurological status. Check glucose immediately.
- Examine for dehydration, rash / purpura, oedema, bladder distension, flank tenderness, infection, muscle injury, compartment syndrome, haemolysis, jaundice, pregnancy, recent surgery, and signs of toxin exposure.

9. Confirm AKI, establish baseline, and assess severity

- Retrieve previous creatinine values, eGFR, urine protein, blood pressure, imaging, and kidney diagnoses. A low current creatinine does not exclude AKI in pregnancy, low muscle mass, or after large-volume fluids.
- Apply both creatinine and urine-output staging. Use the more severe criterion. Document timing, baseline source, urine-output measurement quality, and whether chronic kidney disease or acute-on-chronic kidney injury is likely.
- Repeat creatinine and electrolytes at an interval based on instability and treatment. In active electrolyte emergencies, repeat testing may be required within 30-120 minutes rather than daily.
- Trend weight, cumulative fluid balance, blood pressure, oxygen requirement, and urine output. Consider AKI present before creatinine rises when there is persistent oliguria, shock, obstruction, or a strong nephrotoxic / ischaemic insult.

10. Cause framework: perfusion, intrinsic injury, and obstruction

Category	Clues and immediate priorities
Reduced kidney perfusion / haemodynamic	Vomiting, diarrhoea, haemorrhage, sepsis, vasodilation, heart failure, cirrhosis, renal-artery compromise, excessive diuresis, or medicines affecting autoregulation. Restore effective circulation without causing overload; treat the underlying shock or congestion.

Category	Clues and immediate priorities
Intrinsic renal	Prolonged ischaemia, sepsis-associated tubular injury, nephrotoxins, glomerulonephritis, interstitial nephritis, vascular disease, thrombotic microangiopathy, rhabdomyolysis, haemolysis, tumour lysis, or cortical necrosis. Use urine findings, systemic clues, microscopy, and specialist testing.
Post-renal / obstruction	Retention, prostate disease, pelvic malignancy, stones, clots, neurogenic bladder, bilateral ureteric obstruction, solitary kidney, catheter blockage, or retroperitoneal disease. Bladder scan and image urgently when risk is present; decompress promptly.
Mixed disease	Common in older, critically ill, septic, postoperative, cardiorenal, or oncology patients. More than one mechanism may require simultaneous treatment.

11. Focused history and examination

- Time course of urine reduction; oral intake and losses; fever; urinary symptoms; colic; haematuria; rash; joint symptoms; haemoptysis; diarrhoea; bleeding; muscle pain; seizures; heat exposure; immobilization; weight change; dyspnoea; oedema; pregnancy symptoms.
- Kidney history: CKD stage, previous AKI, stones, prostate or pelvic disease, single kidney, transplant, nephritis, dialysis access, recent surgery or instrumentation, and baseline urine output.
- Medication and toxin timeline, including newly started drugs, dose changes, missed dialysis, contrast, illicit substances, supplements, and occupational or environmental exposures.
- Examine volume status, perfusion, cardiopulmonary congestion, abdomen / bladder, flank, skin, joints, muscle compartments, neurological system, fundi when indicated, and signs of systemic disease.

12. Investigations

Investigation	Use and interpretation
Core blood tests	Urea, creatinine, electrolytes, bicarbonate, glucose, calcium, magnesium, phosphate, CBC, venous blood gas. Add lactate and liver tests according to presentation.
Urine	Dipstick for blood, protein, leucocytes, nitrites, glucose and ketones; pregnancy test where relevant. Send microscopy, culture, albumin / protein ratio, or urine electrolytes when they will change management.
ECG	Immediate in suspected potassium, calcium, magnesium, or severe acid-base disturbance. A normal ECG does not exclude dangerous hyperkalaemia.
Imaging	Bladder scan for retention; renal ultrasound for unexplained AKI or obstruction risk; immediate imaging for pyonephrosis; CT when stone, vascular, abdominal, or pelvic pathology is suspected. Do not delay life-saving contrast imaging.
Intrinsic renal tests	When indicated: blood film, LDH, haptoglobin, bilirubin, reticulocytes, coagulation, CK, urate, complement, ANA, ANCA, anti-GBM, hepatitis / HIV serology, electrophoresis / free light chains, and renal biopsy planning with nephrology.
Infection / toxicology	Cultures, source imaging, drug levels, toxic alcohol / salicylate / lithium studies, and poison-centre advice according to history and acid-base pattern.

13. Fluid, haemodynamic, and congestion management

- Use a specific therapeutic goal: improve perfusion, replace a measured loss, treat shock, or relieve congestion. Avoid maintenance fluids by default in an oliguric patient without ongoing losses.
- For suspected hypovolaemia, give a measured isotonic crystalloid bolus and reassess blood pressure, capillary refill, mentation, lactate, lung fields, JVP / ultrasound, and urine output. Repeated blind boluses are unsafe.
- Use balanced crystalloid or 0.9% sodium chloride according to the clinical context and local policy. Avoid hydroxyethyl starch. Account for sodium and chloride load in severe acidosis, hypernatraemia, or oedema.
- In pulmonary oedema or venous congestion, restrict unnecessary fluid and sodium, support ventilation, treat the cardiac or renal cause, and consider loop diuretic only when response is plausible. Escalate early if oliguria, respiratory failure, or diuretic resistance persists.
- Record cumulative balance and daily / serial weight. A rising creatinine during effective decongestion may not always mean treatment should stop; reassess the whole patient with senior input.

14. Medication and nephrotoxin stewardship

Action	Examples and safeguards
Stop or withhold when appropriate	NSAIDs; ACE inhibitor / ARB during unstable hypovolaemia, sepsis, or severe AKI; potassium supplements; potassium-sparing diuretics; SGLT2 inhibitor during acute illness; metformin in severe AKI / hypoxia; nephrotoxic or renally cleared drugs when risk exceeds benefit.
Dose adjust	Antimicrobials, anticoagulants, opioids, gabapentinoids, insulin, colchicine, digoxin, sedatives, and other renally cleared medicines. Use current pharmacy / renal dosing support rather than eGFR alone in rapidly changing AKI.
Continue when essential	Do not reflexively stop time-critical antimicrobials, antiepileptics, steroids, transplant immunosuppression, or cardiovascular therapy without balancing immediate harm. Seek pharmacy / specialist advice.
Prevent recurrence	Document what was stopped, why, monitoring needed, and who will decide when or whether to restart. Provide written advice at discharge.

15. Urinary obstruction and retention

- Check bladder volume and catheter patency when retention is possible. Use appropriate analgesia and aseptic catheterization. Seek urology advice for difficult catheterization, urethral trauma, recent urological surgery, or contraindication.
- Suspected infected obstructed kidney, obstruction of a solitary kidney, bilateral upper-tract obstruction, anuria with obstruction risk, or AKI complications requires immediate urology and radiology involvement. Decompression is source control and should not be delayed by temporary biochemical improvement.
- After relief of chronic obstruction, anticipate post-obstructive diuresis, hypotension, sodium and potassium disturbance. Measure urine output frequently and replace losses in a controlled, individualized manner.

16. Intrinsic renal and systemic emergencies

Syndrome	Red flags and action
Glomerulonephritis / vasculitis	AKI with blood and protein on dipstick, red-cell casts, oedema, severe hypertension, rash, arthralgia, sinus disease, neuropathy, or pulmonary haemorrhage. Contact nephrology urgently; send targeted serology but do not delay stabilization.
Thrombotic microangiopathy	AKI with thrombocytopenia, haemolytic anaemia, schistocytes, high LDH, neurological change, fever, pregnancy / postpartum state, or severe hypertension. Activate haematology / nephrology and disease-specific emergency therapy.
Acute interstitial nephritis	New drug, fever, rash, eosinophilia, sterile pyuria, or systemic features. Stop the likely agent and discuss specialist assessment; the classic triad is often absent.
Rhabdomyolysis	Muscle injury, prolonged immobilization, exertion / heat, seizure, toxins, dark urine, dipstick blood with few red cells, high CK, hyperkalaemia, hypocalcaemia then hypercalcaemia. Give appropriate crystalloid while monitoring overload; treat electrolyte threats; routine bicarbonate or mannitol is not recommended.
Tumour lysis	Cancer therapy or high-burden malignancy with hyperkalaemia, hyperphosphataemia, hypocalcaemia, hyperuricaemia and AKI. Activate oncology / haematology and nephrology; provide protocol-based fluids, urate-lowering treatment, monitoring, and early dialysis assessment.
Renal vascular catastrophe	Sudden flank / abdominal pain, haematuria, severe hypertension, embolic risk, dissection, pregnancy complication, or abrupt anuria. Urgent vascular imaging and specialty management.

17. Hyperkalaemia: time-critical treatment

Step	Action
1. Confirm and monitor	Repeat a haemolysed or unexpected sample urgently, but do not delay treatment for credible severe hyperkalaemia or ECG toxicity. Stop potassium sources. Obtain continuous monitoring and a 12-lead ECG; involve senior ED and nephrology / critical care early.

Step	Action
2. Stabilize myocardium	For ECG changes, peri-arrest / arrest, or locally defined severe hyperkalaemia, give IV calcium using the approved product and dose. A commonly used adult regimen is calcium chloride 10 mL of 10% over 5 minutes in peri-arrest / arrest, or calcium gluconate 30 mL of 10% over 10 minutes otherwise. Repeat ECG and calcium if toxic changes persist according to protocol.
3. Shift potassium intracellularly	Give soluble insulin 10 units IV with 25 g glucose using the approved order set. Consider additional glucose support when pretreatment glucose is below 7 mmol/L. Add nebulized salbutamol 10-20 mg as an adjunct when not contraindicated; never use it as sole therapy.
4. Remove potassium	Treat the cause, stop potassium-retaining drugs, consider an approved rapid potassium binder, and use loop diuretic only if the patient is producing urine and volume status permits. Arrange urgent dialysis when severe or refractory, or when renal failure / overload limits medical removal.
5. Reassess	Repeat potassium and ECG at protocol-defined intervals, commonly at about 1, 2, 4 and 6 hours according to severity. Monitor glucose frequently for at least 6 hours after insulin, with longer surveillance in high-risk patients.
HYPERKALAEMIA SAFETY: IV calcium protects the heart but does not lower potassium. Insulin and salbutamol temporarily shift potassium but do not remove it. Every severe case requires a documented removal plan, repeat potassium, ECG reassessment, and contingency for dialysis.	

18. Hypokalaemia

- Assess severity, symptoms, ECG, magnesium, acid-base status, renal function, gastrointestinal losses, diuretics, insulin / beta-agonists, endocrine causes, and ongoing losses. Treat torsades or malignant arrhythmia under the resuscitation pathway.
- Use oral replacement when safe and sufficiently rapid. Use IV potassium for severe hypokalaemia, ECG change, paralysis, ileus, rhabdomyolysis, inability to take orally, or ongoing critical loss. Follow local maximum concentration and rate; use an infusion pump and continuous ECG at higher rates.
- Correct magnesium deficiency and stop ongoing losses. Recheck potassium after each defined replacement phase; avoid overcorrection in AKI or when renal function is changing.

19. Sodium emergencies

19.1 Symptomatic hyponatraemia

- Seizure, coma, cardiorespiratory arrest, marked reduced consciousness, persistent vomiting, confusion, or severe headache may indicate cerebral oedema. Exclude hypoglycaemia and other neurological emergencies in parallel.
- In severe or moderately severe symptoms, give 150 mL of 3% sodium chloride or equivalent over 20 minutes in a close-monitoring area, recheck sodium, and repeat up to two further boluses until symptoms improve or sodium has risen about 5 mmol/L, according to the approved pathway.
- After initial improvement, stop hypertonic saline and treat the cause. Limit sodium rise to no more than 10 mmol/L in the first 24 hours and 8 mmol/L in each subsequent 24 hours; use a lower ceiling in patients at high risk of osmotic demyelination.
- Monitor sodium closely. If correction is too rapid, stop active correction and obtain urgent specialist advice for desmopressin and electrolyte-free water according to protocol. Avoid routine vaptans in the emergency treatment of symptomatic hyponatraemia.

19.2 Hypernatraemia

- Assess duration, thirst / access to water, neurological state, volume status, urine output and osmolality, diabetes insipidus, osmotic diuresis, gastrointestinal / skin losses, and sodium administration.
- Restore circulation first with isotonic crystalloid when shocked. Then replace free water orally, enterally, or IV using an individualized deficit and ongoing-loss plan.
- For chronic or unknown-duration hypernatraemia, a commonly used maximum reduction is about 10 mmol/L in 24 hours; acute sodium loading may require faster specialist-directed correction. Check sodium every 2-4 hours during active correction and adjust for ongoing losses.
- Never omit prescribed desmopressin in a patient with known central diabetes insipidus without specialist review. Hypernatraemia with impaired consciousness or inability to self-regulate water requires high-dependency care.

20. Calcium, magnesium, and phosphate emergencies

Disorder	Immediate management principles
Symptomatic hypocalcaemia	Check ionized calcium, magnesium, phosphate, ECG and cause. Treat seizure, tetany, laryngospasm, prolonged QT or arrhythmia with IV calcium gluconate using the approved bolus and infusion pathway; correct magnesium and involve endocrinology / nephrology. Avoid extravasation.
Severe hypercalcaemia	Assess ECG, hydration, renal function and cause. Give cautious isotonic saline when volume depleted and not overloaded. Use calcitonin for rapid temporary effect and a renal-appropriate antiresorptive treatment for severe or malignancy-related disease after specialist review; consider dialysis if refractory or fluids / drugs are unsafe.
Severe hypomagnesaemia	Treat torsades, seizure, marked weakness, refractory hypokalaemia or hypocalcaemia with IV magnesium sulphate according to the resuscitation / electrolyte pathway. Reduce dose and monitor closely in renal failure.
Hypermagnesaemia	Stop magnesium sources. Support airway and circulation; give IV calcium for cardiac or neuromuscular toxicity, promote elimination only when renal function and volume status permit, and arrange dialysis for severe toxicity with renal failure.
Severe hypophosphataemia	Treat respiratory weakness, myocardial dysfunction, haemolysis, rhabdomyolysis, severe neurological symptoms, or very low phosphate with cautious protocol-based replacement. Monitor calcium, potassium, magnesium and renal function.
Hyperphosphataemia	Treat tumour lysis, rhabdomyolysis, renal failure, or phosphate load; avoid unnecessary calcium if the calcium-phosphate product is high; consider binders and dialysis with specialist input.

21. Metabolic acidosis and acid-base danger

- Obtain a blood gas, lactate, ketones, chloride, albumin, renal function, and toxin history. Calculate anion gap when useful and consider mixed disorders.
- Treat the cause: shock, sepsis, DKA, renal failure, diarrhoea, toxin, seizure, or respiratory failure. Improve perfusion and ventilation without causing fluid overload.
- Sodium bicarbonate is not routine for every low bicarbonate. Consider it in selected severe acidemia, specific poisonings, or bicarbonate-loss states under senior / specialist guidance, recognizing sodium load, volume expansion, potassium shift, and carbon dioxide generation.
- Refractory severe acidosis with AKI, especially when accompanied by hyperkalaemia, shock, or overload, is an indication for immediate nephrology / critical-care discussion and possible dialysis.

22. Kidney replacement therapy: urgent escalation

Potential indication	Action
Refractory hyperkalaemia	Immediate nephrology / critical-care activation; continue temporizing treatment and ECG monitoring while dialysis is arranged.
Refractory metabolic acidosis	Treat reversible causes and discuss urgent dialysis when severe acidemia persists or bicarbonate therapy is unsafe / ineffective.
Pulmonary oedema / fluid overload	Escalate when respiratory failure or congestion persists despite appropriate medical therapy, especially with oliguria or anuria.
Uraemic complication	Encephalopathy, pericarditis, bleeding attributed to uraemia, severe persistent nausea / vomiting, or another organ complication requires urgent specialist review.
Dialysable toxin or severe electrolyte burden	Contact toxicology and nephrology early for lithium, toxic alcohols, salicylate, valproate or other selected poisonings, and for refractory severe magnesium, calcium, sodium or tumour-lysis disturbance.
Decision principle	Base initiation on the whole clinical picture, trajectory, reversibility, complications, comorbidity and goals of care - not a single creatinine, urea, potassium, or pH value.

23. Special populations

Population	Additional requirements
Children / adolescents	Use age- and weight-specific AKI, fluid, urine-output, electrolyte and dialysis pathways. Early paediatric / nephrology involvement is required; avoid simply scaling adult concentrated-electrolyte regimens.
Pregnancy / postpartum	Consider hyperemesis, pre-eclampsia / HELLP, haemorrhage, sepsis, cortical necrosis, thrombotic microangiopathy, obstruction and medication effects. Involve obstetrics and nephrology early; interpret creatinine against the lower expected pregnancy baseline.
Older / frail adults	Baseline creatinine may underestimate CKD because of low muscle mass. Assess falls, cognition, access to water, medication burden, retention, heart failure, and goals of care; use smaller fluid challenges and closer monitoring.
Kidney transplant	Contact the transplant / nephrology team early. Assess rejection, obstruction, vascular compromise, infection, drug toxicity, adherence and interactions. Do not stop immunosuppression casually.
Advanced CKD / dialysis	Interpret baseline values and residual urine output correctly. Protect dialysis access, confirm last treatment and target weight, avoid unnecessary fluid, and coordinate urgent dialysis. Detailed dialysis complications are addressed in Protocol 47.
Oncology / haematology	Anticipate tumour lysis, cast nephropathy, sepsis, nephrotoxic therapy, obstruction, hypercalcaemia and thrombotic microangiopathy. Activate disease-specific pathways early.

24. Monitoring and treatment targets

Parameter	Minimum standard
Vital signs / clinical status	Frequency based on acuity; continuous ECG for severe potassium / calcium / magnesium disturbance or high-rate replacement; serial neurological and respiratory assessment.
Urine and fluid balance	Hourly urine output in severe AKI / critical illness; document catheter status, cumulative intake / output, oedema, oxygen need and serial weight.
Laboratory tests	Repeat potassium, sodium, calcium, magnesium, phosphate, glucose, creatinine and blood gas at intervals matched to treatment and instability. Do not use routine daily testing for an active emergency.
Hyperkalaemia glucose safety	Baseline and frequent glucose after insulin, extending for at least 6 hours and longer when risk is high. Provide rescue treatment and document hypoglycaemia events.
Response	Each fluid bolus, diuretic dose, electrolyte infusion, hypertonic-saline bolus, or temporizing hyperkalaemia treatment must have a documented reassessment time and contingency.

25. Disposition

Disposition	Typical criteria
Resuscitation / ICU / high dependency	Unstable airway or circulation; severe electrolyte or acid-base emergency; pulmonary oedema; ongoing hypertonic saline; high-rate electrolyte infusion; severe symptomatic uraemia; dialysis requirement; rapidly progressive intrinsic disease; need for continuous invasive monitoring.
Admission / monitored observation	Stage 2-3 AKI; persistent oliguria; stage 1 AKI with high-risk cause or comorbidity; obstruction; infection; transplant; rhabdomyolysis; tumour lysis; significant medication toxicity; electrolyte abnormality requiring serial treatment; unreliable follow-up.
Transfer	Required nephrology, urology, ICU, dialysis, interventional radiology, paediatric, obstetric, oncology, toxicology, or laboratory capability is unavailable locally.
Discharge only after senior review	Mild and improving AKI or corrected minor electrolyte disturbance; stable perfusion and volume status; no obstruction, infection, ECG risk, ongoing loss, or dangerous intrinsic cause; medications reconciled; oral intake and urine output adequate; repeat testing and clinician follow-up arranged.

26. Discharge and post-AKI recovery plan

- Provide the diagnosis, likely cause, AKI stage, latest and baseline creatinine, electrolyte trend, medicines stopped or adjusted, and pending results in writing.
- Arrange repeat creatinine and electrolytes according to severity and stability - often within 48-72 hours for a recent ED AKI, sooner when risk is higher. Name the clinician responsible for review and action.
- Explain hydration during illness, when to seek help for vomiting / diarrhoea or reduced urine, and avoidance of over-the-counter NSAIDs. Provide individualized sick-day medication advice rather than a generic permanent stop.
- Arrange nephrology follow-up for persistent kidney impairment, eGFR 30 mL/min/1.73 m2 or less after recovery, significant proteinuria / haematuria, hypertension, recurrent AKI, stage 3 AKI, transplant, unexplained cause, or suspected intrinsic disease.
- Use teach-back and address access to water, medicines, transport, laboratory testing, caregiving, cognitive impairment, and health literacy.

27. Transfer and handover

- Confirm receiving clinician, destination, dialysis / procedure plan, transport capability, and expected departure time. Escalate delay when a time-critical indication exists.
- Send baseline and serial creatinine, urine output, fluid balance, weight, ECGs, blood-gas and electrolyte trends, imaging, urinalysis, cultures, medicine exposures, and current infusions.
- Ensure adequate quantities of glucose, calcium, insulin, dextrose, electrolyte infusions, oxygen, vasopressors, and monitoring for the journey. State exactly when the next glucose, potassium, sodium, calcium, or blood gas is due.
- Use closed-loop handover and document who holds clinical responsibility during delay and transport.

28. Documentation and handover

- Record AKI stage and criteria, baseline source, urine-output trend, volume assessment, likely cause, key positives / negatives, obstruction assessment, medicine review, and treatment response.
- Document all concentrated-electrolyte products, doses, routes, line sites, pump rates, independent checks, start / stop times, repeat results, ECG changes, and adverse events.
- Record specialist discussions, dialysis decisions, goals-of-care decisions, transfer acceptance, pending tests, and named follow-up responsibility.

29. Quality indicators and audit

Indicator	Suggested measure
Early recognition	Percentage with AKI stage and baseline documented; percentage with urine output assessed.
Cause identification	Percentage with urinalysis and documented cause framework; time to obstruction imaging / decompression when indicated.
Hyperkalaemia safety	Time from result / ECG to calcium and insulin-glucose; repeat potassium and ECG; post-insulin glucose monitoring and hypoglycaemia rate.
Fluid stewardship	Percentage of fluid boluses with indication and reassessment; cumulative positive balance and pulmonary-oedema events.
Medication safety	Documented nephrotoxin / renal-dose review and discharge restart plan.
Renal escalation	Time to nephrology / critical-care discussion and dialysis initiation when indicated.
Recovery	Percentage discharged with repeat laboratory plan, patient information, and responsible clinician; 7- and 30-day reattendance.

30. Training and implementation

- Annual simulation should include hyperkalaemia with ECG toxicity, pulmonary oedema with oliguria, symptomatic hyponatraemia, obstructed infected kidney, and rapidly progressive intrinsic AKI.
- Nursing and medical competency should include urine-output measurement, ECG recognition, high-alert electrolyte administration, glucose surveillance after insulin, fluid reassessment, and transfer of ongoing infusions.
- The department should maintain ready access to hyperkalaemia and hypertonic-saline order sets, calcium products, infusion pumps, bladder scanning, ultrasound / imaging, nephrology / urology contacts, and dialysis transfer arrangements.

ANNEX A. One-page workflow

[ ] ABCDE + glucose + ECG + IV access + accurate weight + continuous monitoring when unstable.

[ ] Send creatinine / urea, electrolytes, bicarbonate, calcium, magnesium, phosphate, blood gas, CBC and urinalysis; add CK, lactate, osmolality, cultures, toxicology or haemolysis tests as indicated.

[ ] Treat immediate threats: hyperkalaemic ECG, arrhythmia, shock, pulmonary oedema, seizure / coma, severe symptomatic sodium or calcium disorder, severe acidosis.

[ ] Stage AKI using creatinine and urine output; retrieve baseline.

[ ] Assess perfusion versus congestion; give measured fluid only with a goal and reassessment.

[ ] Check obstruction: catheter, bladder scan, ultrasound / CT; urgent decompression when infected, bilateral, solitary kidney or complicated.

[ ] Classify cause: perfusion / haemodynamic, intrinsic, obstruction, mixed.

[ ] Review medicines and toxins; adjust doses and document restart plan.

[ ] Escalate early for dialysis indication, stage 3 / unexplained AKI, transplant, intrinsic red flags, or unavailable local capability.

[ ] Disposition + repeat tests + recovery plan + named responsibility.

ANNEX B. AKI staging card

Stage	Creatinine criterion	Urine-output criterion
1	1.5-1.9 x baseline or rise at least 26 micromol/L	<0.5 mL/kg/h for 6-12 h
2	2.0-2.9 x baseline	<0.5 mL/kg/h for at least 12 h
3	At least 3 x baseline; or at least 354 micromol/L with acute rise; or kidney replacement therapy	<0.3 mL/kg/h for at least 24 h or anuria for at least 12 h

Use the more severe creatinine or urine-output stage. Confirm duration and measurement reliability. Paediatric definitions and eGFR criteria require the approved paediatric pathway.

ANNEX C. First-hour checklist

[ ] Baseline creatinine and urine-output history obtained.

[ ] ECG reviewed and monitoring level assigned.

[ ] Potassium / sodium / calcium / magnesium / phosphate / bicarbonate reviewed.

[ ] Urinalysis completed and urine specimen saved if intrinsic disease possible.

[ ] Perfusion, congestion, weight and cumulative balance documented.

[ ] Bladder scan / obstruction risk addressed.

[ ] Nephrotoxins and renal-dose medicines reviewed.

[ ] Repeat laboratory times and clinical reassessment times prescribed.

[ ] Nephrology / urology / critical care / transfer contacted when indicated.

ANNEX D. Hyperkalaemia rescue card

[ ] Continuous ECG + 12-lead ECG; stop potassium intake / infusions and review medications.

[ ] Toxic ECG / severe hyperkalaemia: IV calcium per approved product, dose and setting; repeat ECG.

[ ] Shift: soluble insulin 10 units IV + glucose 25 g; consider additional glucose if pretreatment glucose <7 mmol/L.

[ ] Adjunct: nebulized salbutamol 10-20 mg unless contraindicated; not monotherapy.

[ ] Remove: cause treatment + approved binder / diuretic when appropriate + dialysis plan.

[ ] Repeat potassium and ECG; monitor glucose frequently for at least 6 h.

[ ] Document contingency if potassium rebounds or dialysis is delayed.

ANNEX E. Hypokalaemia replacement card

[ ] Symptoms / ECG / magnesium / acid-base / renal function / ongoing losses assessed.

[ ] Oral route used when safe and sufficiently rapid.

[ ] IV replacement only through approved concentration, pump, line and maximum-rate pathway.

[ ] Continuous ECG for severe disease or higher-rate infusion.

[ ] Magnesium corrected and cause treated.

[ ] Repeat potassium after each replacement phase; stop or slow if renal function worsens.

ANNEX F. Sodium-emergency card

[ ] Symptomatic hyponatraemia: 150 mL 3% saline or equivalent over 20 min; recheck and repeat up to 2 times to target about +5 mmol/L / symptom improvement.

[ ] After initial response: stop hypertonic saline, treat cause, limit +10 mmol/L first 24 h and +8 mmol/L each 24 h thereafter; lower limit when high risk.

[ ] Overcorrection: stop active therapy; urgent specialist plan for desmopressin / electrolyte-free water.

[ ] Hypernatraemia: restore perfusion first, then controlled free-water replacement; account for ongoing losses.

[ ] During active correction: frequent sodium and neurological checks; document target rate and stop points.

[ ] Known central diabetes insipidus: do not omit desmopressin without specialist review.

ANNEX G. Calcium / magnesium / phosphate card

[ ] Use ionized calcium when pH or albumin makes total calcium unreliable.

[ ] Hypocalcaemic seizure / tetany / ECG toxicity: IV calcium pathway + magnesium correction.

[ ] Severe hypercalcaemia: cautious saline if depleted, calcitonin + specialist antiresorptive therapy; dialysis if refractory / unsafe to hydrate.

[ ] Torsades / severe hypomagnesaemia: IV magnesium pathway; renal-dose caution.

[ ] Hypermagnesaemic toxicity: stop source + IV calcium + elimination / dialysis plan.

[ ] Severe phosphate disorder: treat cause and replace / remove with close calcium, potassium and renal monitoring.

ANNEX H. Fluid and congestion reassessment

Before intervention	After each bolus / treatment phase
Blood pressure, pulse, capillary refill, mentation, lactate	Did perfusion improve? Is vasopressor or source control needed?
JVP, lung fields, oedema, oxygen need, ultrasound	Any new crackles, B-lines, rising JVP, hypoxaemia or weight gain?
Urine output, bladder / catheter status	Output response? Obstruction excluded?
Weight, input / output, ongoing losses	Updated cumulative balance and next fluid goal
Sodium, chloride, potassium, bicarbonate	Any dilution, hyperchloraemia, sodium shift or worsening electrolyte risk?

ANNEX I. Obstruction checklist

[ ] Retention symptoms, prostate / pelvic disease, stone, cancer, clot, neurogenic bladder or recent instrumentation.

[ ] Catheter present: tubing, bag, position, blockage and flush policy checked.

[ ] Bladder scan documented.

[ ] Upper-tract imaging ordered when cause unclear or obstruction risk exists.

[ ] Infected obstruction / solitary kidney / bilateral obstruction / anuria: immediate urology + radiology.

[ ] After decompression: post-obstructive diuresis, blood pressure and electrolytes monitored.

ANNEX J. Intrinsic renal red flags

[ ] Blood + protein on dipstick without UTI or catheter trauma.

[ ] Pulmonary haemorrhage, rash / purpura, arthralgia, sinus disease or neuropathy.

[ ] Thrombocytopenia + haemolysis / schistocytes + AKI.

[ ] Severe hypertension, pregnancy / postpartum disease or abrupt neurological change.

[ ] High CK, muscle injury, heat exposure, seizure or prolonged immobilization.

[ ] Cancer with urate / potassium / phosphate rise and calcium fall.

[ ] Rapid progression, unexplained stage 2-3 AKI or failure to respond to initial treatment.

ANNEX K. Medication / nephrotoxin checklist

[ ] NSAIDs, ACE inhibitor / ARB, diuretics, spironolactone / eplerenone, potassium supplements.

[ ] SGLT2 inhibitor, metformin, insulin / sulfonylurea, trimethoprim, digoxin.

[ ] Aminoglycoside, vancomycin, amphotericin, antivirals, chemotherapy / immunotherapy.

[ ] Lithium, salicylate, toxic alcohol, contrast, herbal / traditional medicines, illicit drugs.

[ ] Renally cleared analgesics, sedatives, anticoagulants, antiepileptics and antimicrobials dose-checked.

[ ] Stop / continue decisions and restart responsibility documented.

ANNEX L. Dialysis-escalation card

[ ] Refractory hyperkalaemia.

[ ] Refractory severe metabolic acidosis.

[ ] Pulmonary oedema / fluid overload with respiratory compromise.

[ ] Uraemic encephalopathy, pericarditis, bleeding or severe symptoms.

[ ] Dialysable toxin or refractory severe magnesium / calcium / tumour-lysis disturbance.
[ ] Nephrology / critical care contacted; access, modality, destination and time documented.
[ ] Temporizing therapy and monitoring continue while waiting.

ANNEX M. Monitoring chart

Time	BP / HR / SpO2	Urine mL/h	Fluid balance	K	Na	Cr / HCO3	Ca / Mg / PO4	Glucose / ECG / actions
Arrival								
1 h								
2 h								
4 h								
6 h								
12 h								

ANNEX N. Discharge and recovery checklist

[ ] AKI stage, likely cause, baseline and latest creatinine documented.
[ ] Electrolytes stable without rebound and ECG risk resolved.
[ ] Volume status, oral intake and urine output adequate.
[ ] Obstruction, infection and dangerous intrinsic cause excluded or treated.
[ ] Medication reconciliation and restart plan completed.
[ ] Repeat laboratory date, location and responsible clinician documented.
[ ] Written hydration / illness / NSAID / return precautions provided with teach-back.
[ ] Nephrology / primary-care / specialty follow-up arranged when indicated.

ANNEX O. Transfer and handover minimum dataset

[ ] AKI stage, baseline creatinine, current creatinine and trajectory.
[ ] Urine output, catheter / obstruction status, weight and cumulative balance.
[ ] Potassium, sodium, bicarbonate / pH, calcium, magnesium, phosphate, glucose and ECG trends.
[ ] Likely cause, urinalysis, CK / haemolysis / tumour-lysis results, imaging and cultures.
[ ] Fluids, diuretics, calcium, insulin-glucose, hypertonic saline, electrolyte infusions and vasopressors given / running.
[ ] Next tests due, glucose-monitoring schedule, dialysis / decompression plan and contingency.
[ ] Receiving clinician, destination, transport capability and responsibility during delay.

ANNEX P. Audit tool

Audit item	Met / not met / N/A
AKI stage and baseline documented	
Urine output and volume status assessed	
Urinalysis completed and cause framework documented	
ECG obtained for relevant electrolyte emergency	
Hyperkalaemia treated within target time	
Repeat potassium and post-insulin glucose monitoring completed	
Fluid intervention had indication and reassessment	
Obstruction imaging / decompression timely	
Medication / renal-dose review completed	
Nephrology / critical-care escalation timely	
Discharge repeat testing and named responsibility documented	
Adverse event / dialysis delay / reattendance reviewed	

ANNEX Q. Local configuration checklist

[ ] Approved AKI staging, urine-output, fluid-bolus and overload pathways.

[ ] Hyperkalaemia order set: calcium products, insulin-glucose, glucose surveillance, salbutamol, binders, repeat testing and dialysis escalation.

[ ] Approved potassium, hypertonic saline, calcium, magnesium and phosphate concentrations, pumps, line rules and double checks.

[ ] Laboratory critical-value notification, haemolysis reporting, blood-gas / ionized-calcium capability and turnaround standards.

[ ] Bladder scanner, ultrasound / CT access, pyonephrosis pathway and urology decompression contacts.

[ ] Nephrology, critical care, dialysis, transplant, paediatric, obstetric, oncology and toxicology contacts and transfer agreements.

[ ] Renal-dose / nephrotoxin pharmacy support and discharge medication-restart process.

[ ] Post-AKI laboratory, primary-care and nephrology follow-up pathways and patient information.

ANNEX R. References and source tools

1. National Institute for Health and Care Excellence. Acute kidney injury: prevention, detection and management. NICE guideline NG148. Updated 16 October 2024.

2. Kidney Disease: Improving Global Outcomes (KDIGO). Clinical Practice Guideline for Acute Kidney Injury. Kidney International Supplements. 2012;2:1-138. The KDIGO 2026 AKI / AKD update was in public-review draft at the time of this protocol and was not treated as final guidance.

3. UK Kidney Association. Clinical Practice Guideline: Management of Hyperkalaemia in Adults. Final version, 19 December 2023; review date October 2026.

4. Ball S, Barth J, Levy M, et al. Emergency management of severe and moderately severely symptomatic hyponatraemia in adult patients. Society for Endocrinology. 2022.

5. Turner J, Gittoes N, Selby P. Society for Endocrinology Endocrine Emergency Guidance: Emergency management of acute hypocalcaemia in adult patients. Endocrine Connections. 2016; addendum 2019.

6. Walsh J, Gittoes N, Selby P. Society for Endocrinology Endocrine Emergency Guidance: Emergency management of acute hypercalcaemia in adult patients. Endocrine Connections. 2016;5:G9-G11.

7. Endocrine Society. Treatment of Hypercalcemia of Malignancy in Adults: Clinical Practice Guideline. Journal of Clinical Endocrinology & Metabolism. 2023.

8. Society for Endocrinology. Inpatient management of cranial diabetes insipidus / arginine vasopressin deficiency. Clinical guidance resource, current access June 2026.

9. World Health Organization and International Committee of the Red Cross. Basic Emergency Care: Approach to the Acutely Ill and Injured. WHO; 2018.

10. Local source tools to attach before approval: hyperkalaemia algorithm and order set; concentrated-electrolyte monographs; hypertonic-saline pathway; fluid and vasopressor guidance; renal-dose handbook; obstruction / pyonephrosis pathway; dialysis indications and transfer directory; paediatric and pregnancy pathways; post-AKI follow-up form.