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RENAL DOSE ADJUSTMENT OF COMMON DRUGS-A COMPREHENSIVE CLINICAL REFERENCE FOR HEALTHCARE PROFESSIONALS-REVIEW ARTICLE

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Abstract:

Background: *Chronic kidney disease (CKD) affects over 700 million people worldwide and significantly alters drug pharmacokinetics through reduced renal clearance, impaired hepatic metabolism, altered protein binding, and changed drug distribution. These changes lead to drug and metabolite accumulation when standard doses are used without adjustment, contributing to preventable adverse drug events in a population that is already highly medicated.*

Objective*: To provide a concise, evidence-based reference guide for renal dose adjustment of commonly prescribed drugs across five therapeutic classes — antibiotics, cardiovascular agents, antidiabetics, analgesics, and miscellaneous drugs — stratified by eGFR and dialysis status.*

Methods*: Dosing recommendations were compiled from authoritative sources including KDIGO 2022 CKD Guidelines, BNF 85, UpToDate, Micromedex, and Drug Prescribing in Renal Failure (Aronoff et al.). Drugs were selected based on clinical prevalence and the degree of renal adjustment required, stratified across eGFR bands of 30–59, 10–29, and <10 mL/min/1.73m² including haemodialysis.*

Results*: Over 60 drugs are presented with eGFR-stratified recommendations. Key findings: NSAIDs are contraindicated in significant CKD; nitrofurantoin is ineffective and toxic below eGFR 45; metformin is contraindicated below eGFR 30; apixaban is the preferred DOAC in advanced CKD; fentanyl is the opioid of choice in ESRD; and gabapentin require strict dose reduction to prevent neurotoxicity. TDM is mandatory for vancomycin, aminoglycosides, digoxin, and lithium.*

Conclusion*: Systematic eGFR-guided dose adjustment is essential for safe prescribing in CKD. This guide provides a practical reference framework to support clinicians across all settings in making appropriate medication decisions throughout the full spectrum of renal impairment.*

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INTRODUCTION:

Chronic kidney disease (CKD) represents one of the most challenging and consequential clinical syndromes encountered in modern medicine. Defined by the kidney disease: Improving Global Outcomes (KDIGO) organisation as the presence of kidney damage or a glomerular filtration rate below 60 mL/min/1.73m² for a period exceeding three months, CKD affects an estimated 10–12% of the global adult population. Its prevalence is highest among older adults and individuals with diabetes mellitus and hypertension — two of the most common chronic conditions worldwide — making CKD an inevitably common accompaniment to the complex, multimorbid patients encountered daily in general practice, internal medicine, and specialist clinics. Beyond its direct impact on renal function, CKD significantly amplifies cardiovascular risk, accelerates anaemia and bone mineral disease, promotes metabolic acidosis, and contributes to frailty, cognitive impairment, and reduced quality of life.

The pharmacological management of CKD and its associated comorbidities is inherently complex. Patients with CKD are, paradoxically, both the most heavily medicated and the most pharmacologically vulnerable group in clinical practice. Polypharmacy — typically defined as the concurrent use of five or more medications — is nearly universal in CKD, with studies reporting an average of 8–12 prescribed medications per patient in moderate-to-severe stages. This creates a compounding risk: each additional drug represents an opportunity for dose-related toxicity, drug-drug interaction, and further nephrotoxic injury in an already vulnerable kidney. The clinical imperative to prescribe effectively for comorbidities such as heart failure, diabetes, hypertension, and infection must therefore be balanced against the heightened risk of pharmacological harm.

Renal impairment fundamentally alters all four key phases of drug pharmacokinetics. With respect to absorption, uraemia reduces gastrointestinal motility, alters intestinal pH, and induces mucosal oedema, collectively impairing oral drug absorption. Distribution is affected by the hypoalbuminemia and fluid overload common in CKD: reduced protein binding increases the free (pharmacologically active) fraction of highly protein-bound drugs such as phenytoin and warfarin, potentially leading to enhanced effects at standard doses. Metabolism is disrupted because uraemic toxins suppress cytochrome P450 enzyme activity in the liver, reducing the presystemic and systemic metabolism of many substrates. Finally, and most critically, renal excretion — comprising glomerular filtration, active tubular secretion, and passive tubular reabsorption — is directly impaired in proportion to the loss of functional nephrons, leading to reduced clearance and prolonged half-lives for renally eliminated drugs and their metabolites.

The clinical consequences of unrecognised or under-corrected pharmacokinetic disturbances in CKD are well-documented and serious. Drug toxicity in CKD may manifest as neurotoxicity (from accumulation of opioid metabolites or gabapentin), bleeding (from excessive anticoagulant exposure), cardiovascular events (from digoxin toxicity or antihypertensive over-dosing), hypoglycaemia (from insulin or sulfonylurea accumulation), and acute-on-chronic kidney injury (from NSAIDs, aminoglycosides, or nephrotoxic contrast agents). Conversely, under-dosing — driven by excessive caution — risks therapeutic failure: antibiotic sub-therapeutic levels promote resistance, anti-hypertensive under-treatment accelerates renal and cardiovascular disease progression, and under-anticoagulation increases thromboembolic risk. Achieving the therapeutic window in CKD therefore demands precision, not simply caution.

Estimated glomerular filtration rate (eGFR), derived from serum creatinine using validated equations such as the CKD-EPI 2021 creatinine equation or the MDRD formula, provides the primary metric for stratifying renal function and guiding dose adjustment. However, clinicians must appreciate the limitations of creatinine-based eGFR estimation: in the elderly, sarcopenic, or severely malnourished patient, reduced muscle mass results in low serum creatinine that may substantially overestimate true GFR. In such cases, the Cockcroft-Gault formula using actual body weight — which explicitly incorporates age and sex — may better reflect functional renal capacity for the purposes of drug dosing and is specifically recommended by many drug prescribing references. Cystatin C-based eGFR equations offer further refinement in populations where creatinine is unreliable, though these are less universally available.

This clinical reference guide was developed in response to the demonstrable and persistent gap between best-practice prescribing guidance and real-world clinical practice in CKD. The guide synthesises dosing recommendations for more than 60 of the most commonly prescribed medications across five major therapeutic classes —antibiotics and antifungals, cardiovascular and anticoagulant agents, antidiabetic drugs, analgesics and pain management agents, and a broad miscellaneous category — stratified by four levels of renal function aligned with the KDIGO CKD staging framework. Special sections address the principles of renal dose adjustment, drugs requiring avoidance in CKD, special patient populations including haemodialysis and peritoneal dialysis patients, and the management of acute kidney injury superimposed on CKD. All recommendations are

derived from and cross-referenced against current authoritative prescribing references and guidelines, and are intended to complement, not replace, clinical pharmacist input and individualised clinical assessment.

CKD Staging & eGFR Classification

Estimated glomerular filtration rate (eGFR), calculated from serum creatinine using equations such as CKD-EPI or MDRD, serves as the primary basis for renal dose adjustment. The table below summarizes the KDIGO CKD staging framework:

Table .1 KDIGO CKD STAGING FRAMEWORK

CKD Stage	eGFR (mL/min/1.73m²)	Description	Dosing Consideration
1	≥ 90	Normal or high	Usually, no change
2	60–89	Mildly decreased	Monitor; minor adjustments
3a	45–59	Mildly-moderately decreased	Dose reduction may be needed
3b	30–44	Moderately decreased	Dose reduction required for many drugs
4	15–29	Severely decreased	Significant adjustments; avoid nephrotoxins
5 / ESRD	< 15	Kidney failure	Dialysis dose adjustments needed

⚠ In elderly patients, muscle mass is reduced and creatinine may underestimate renal impairment. Use clinical judgment alongside laboratory values.

✓ For patients on haemodialysis (HD), time the dose after dialysis sessions for renally-eliminated drugs whenever possible.

Key Principles of Renal Dose Adjustment

Understanding a few core pharmacokinetic principles will help clinicians apply these recommendations appropriately:

Creatinine clearance (CrCl) vs. eGFR: Many older drug monographs reference CrCl (Cockcroft-Gault formula) rather than eGFR. CrCl is preferred for drug dosing in extremes of body weight or muscle mass.

Dose reduction vs. interval extension: Both strategies can achieve safe drug levels. Interval extension is preferred when peak concentration drives efficacy (e.g., aminoglycosides). Dose reduction is preferred when steady-state concentration matters (e.g., most antibiotics).

Active metabolites: Some drugs have active or toxic renally-cleared metabolites (e.g., morphine-6-glucuronide from morphine, normeperidine from meperidine). These require particular attention in CKD.

Therapeutic drug monitoring (TDM): For narrow therapeutic index drugs (e.g., vancomycin, digoxin, lithium, aminoglycosides), TDM is mandatory in CKD patients.

Dialysis clearance: Some drugs are significantly cleared by haemodialysis (HD) or peritoneal dialysis (PD), requiring supplemental post-dialysis doses. Others are not dialyzable and require separate adjustments.

Nephrotoxic drugs: NSAIDs, aminoglycosides, contrast agents, and calcineurin inhibitors should be avoided or used with extreme caution in CKD, as they may precipitate acute kidney injury (AKI) superimposed on CKD.

Antibiotics & Antifungals

Antimicrobial dosing in CKD is among the most clinically critical adjustments. Inadequate dosing risks treatment failure; excessive dosing risks toxicity. For renally-cleared antibiotics, both the dose and interval may need adjustment. Note that for TDM-guided drugs (vancomycin, aminoglycosides), serum levels must guide individualization beyond these general recommendations.

TABLE.2 : ANTIBIOTIC DOSING IN CKD

Drug	Normal Dose	eGFR 30–59	eGFR 10–29	eGFR < 10 / HD	Notes
Amoxicillin	500 mg q8h	500 mg q8–12h	500 mg q12–24h	250–500 mg q24h	
Amoxicillin-Clavulanate	875/125 mg q12h	875/125 mg q12h	500/125 mg q12h	500/125 mg q24h	Avoid if eGFR <30 for 875mg tab
Ciprofloxacin	500 mg q12h	500 mg q12h	250–500 mg q12–24h	250 mg q24h; dose after HD	-
Levofloxacin	500 mg q24h	250–500 mg q24h	250 mg q24–48h	250 mg q48h; dose after HD	-
Trimethoprim-Sulfamethoxazole	1 DS tab q12h	1 DS tab q12–24h	1 DS tab q24h	Avoid or use with caution	Risk of hyperkalaemia
Nitrofurantoin	100 mg q12h	Avoid (eGFR <45)	Contraindicated	Contraindicated	Ineffective + toxic in CKD
Vancomycin	15–20 mg/kg q8–12h	15–20 mg/kg q24–48h	Individualize per levels	Supplement post-HD	TDM required; AUC-guided preferred
Gentamicin	5–7 mg/kg q24h	Reduce dose/extend interval	Extend to q48h+	Post-HD supplement	TDM mandatory; avoid prolonged use

⚠ Aminoglycosides and vancomycin are nephrotoxic themselves — use the shortest effective course and monitor renal function frequently.

⚠ Nitrofurantoin is both ineffective (concentrations too low in urine) and potentially toxic in significant CKD — avoid when eGFR < 45 mL/min.

✓ When prescribing extended-spectrum beta-lactams for critically ill CKD patients, consider extended infusion strategies (e.g., 3-hour meropenem infusion) to optimize pharmacodynamic target attainment.

Cardiovascular & Anticoagulant Drugs

Cardiovascular disease is the leading cause of mortality in CKD. Many first-line cardioprotective drugs — including ACE inhibitors, beta-blockers, and anticoagulants — require careful renal dose adjustment. ACE inhibitors and ARBs slow CKD progression but carry risks of hyperkalaemia and acute rises in creatinine, which should be monitored closely upon initiation or dose change.

TABLE .3 CKD DOSE ADJUSTMENT

Drug	Normal Dose	eGFR 30–59	eGFR 10–29	eGFR < 10 / HD	Notes
Lisinopril	10–40 mg q24h	No change; monitor K+	Start low; monitor closely	Start 2.5–5 mg; caution	Risk of hyperkalaemia & AKI
Ramipril	5–10 mg q24h	2.5–5 mg q24h	1.25–2.5 mg q24h	Avoid or use with caution	Monitor K+, creatinine
Metformin	500–1000 mg q12h	Reduce dose; reassess	Contraindicated (eGFR <30)	Contraindicated	Risk of lactic acidosis
Atenolol	50–100 mg q24h	25–50 mg q24h	25 mg q24–48h	25 mg q48h; dose after HD	Renally excreted
Digoxin	0.125–0.25 mg q24h	0.125 mg q24h	0.125 mg q48h	0.0625 mg q48h	TDM required; narrow TI
Spironolactone	25–50 mg q24h	Use with caution	Avoid (eGFR <30)	Contraindicated	Severe hyperkalaemia risk
Furosemide	20–80 mg q24h	Higher doses may be needed	Higher doses may be needed	IV often required	Effect diminished in CKD
Rivaroxaban	15–20 mg q24h	15 mg q24h (AF)	Avoid if eGFR <30 (AF)	Contraindicated	Use with caution
Apixaban	5 mg q12h	Assess dose reduction criteria	2.5 mg q12h if 2/3 criteria met	Use with great caution	Criteria: age≥80, wt≤60kg, Cr≥1.5
Dabigatran	150 mg q12h	110–150 mg q12h	Avoid if eGFR <30	Contraindicated	80% renal excretion
Warfarin	Variable (INR-guided)	No change; INR monitoring	No change; close INR monitoring	No change; consider alternatives	Sensitivity may increase in CKD
Allopurinol	300 mg q24h	100–200 mg q24h	100 mg q24h or less	100 mg after HD	Severe toxicity if not reduced

⚠ Direct oral anticoagulants (DOACs) have varying renal elimination: dabigatran (80%) > rivaroxaban (35%) > apixaban (27%). Apixaban has the lowest renal dependence and is generally preferred in advanced CKD.

⚠ Allopurinol accumulation in CKD causes severe toxicity including Stevens-Johnson syndrome. Dose must be reduced proportionally to eGFR.

✓ When initiating ACE inhibitors or ARBs in CKD, a creatinine rise of up to 30% is acceptable and not a reason to stop — it often reflects beneficial hemodynamic effects.

Antidiabetic Drugs

Diabetes is the leading cause of CKD worldwide. Managing hyperglycaemia in CKD requires careful drug selection — some agents are contraindicated, while others paradoxically have extended indications for cardiorenal protection. The risk of hypoglycaemia increases in CKD due to impaired gluconeogenesis and reduced insulin clearance.

TABLE 4. ANTIDIABETIC DRUGS DOSE ADJUSTMENT

Drug	Normal Dose	eGFR 30–59	eGFR 10–29	eGFR < 10 / HD	Notes
Metformin	500–1000 mg BID	Max 1000 mg/day; reassess	Contraindicated	Contraindicated	Lactic acidosis risk
Glipizide	5–10 mg q24h	No change	2.5–5 mg q24h	2.5 mg q24h; hypoglycaemia risk	Preferred sulfonylurea in CKD
Glipalamide (Glyburide)	5 mg q24h	Avoid	Avoid	Contraindicated	Active metabolite accumulates
Sitagliptin	100 mg q24h	50 mg q24h	25 mg q24h	25 mg q24h (post-HD)	Dose-adjusted; generally safe
Empagliflozin	10–25 mg q24h	10 mg q24h; avoid if eGFR <30	Avoid (eGFR <30)	Contraindicated	CV/renal benefit; glycemia efficacy reduced
Dapagliflozin	10 mg q24h	Glycaemic use: avoid <45; Heart failure/CKD: use if ≥25	CKD/HF use only (eGFR≥25)	Contraindicated for glycemia	Cardiorenal indication extends use
Semaglutide (oral)	3–14 mg q24h	No dose change	Use with caution	Limited data; caution	Monitor for GI side effects
Insulin	Individualized	Reduce by ~25%	Reduce by ~25–50%	Reduce by ~50%; monitor closely	Insulin clearance reduced in CKD

⚠ Metformin is contraindicated when eGFR < 30 due to lactic acidosis risk. It should be withheld peri-procedurally when iodinated contrast is used.

✓ SGLT2 inhibitors (empagliflozin, dapagliflozin) have proven cardiorenal protective benefits beyond glycaemic control and can now be used at lower eGFR thresholds specifically for these indications.

✓ Insulin remains effective at all levels of renal function but doses typically need reduction in advanced CKD due to prolonged insulin half-life and reduced renal insulin degradation.

Analgesics & Pain Management

Pain management in CKD presents unique challenges. NSAIDs — the most commonly used analgesics — are contraindicated in moderate-to-severe CKD. Opioids require careful selection, as renally-cleared active metabolites can accumulate and cause respiratory depression, prolonged sedation, or neurotoxicity. Paracetamol remains the preferred first-line analgesic.

TABLE.5 ANALGESIC DOSE ADJUSTMENT IN CKD

Drug	Normal Dose	eGFR 30–59	eGFR 10–29	eGFR < 10 / HD	Notes
Paracetamol (Acetaminophen)	500–1000 mg q4–6h	No change	q6h dosing preferred	Max 2 g/day; use cautiously	Safest analgesic in CKD
Ibuprofen	200–400 mg q6–8h	Avoid (eGFR <60)	Contraindicated	Contraindicated	NSAIDs worsen renal function
Diclofenac	50 mg q8h	Avoid if possible	Contraindicated	Contraindicated	All NSAIDs nephrotoxic in CKD
Tramadol	50–100 mg q4–6h	Max 100 mg q6h	Max 50–100 mg q12h	Avoid	Seizure risk from metabolite accumulation
Codeine	30–60 mg q4–6h	Use with caution	Avoid (metabolite accumulates)	Contraindicated	Active metabolite morphine-6-glucuronide
Morphine	5–10 mg q4h	Reduce dose; monitor	Avoid if possible	Avoid	M6G accumulation causes toxicity
Oxycodone	5–10 mg q4–6h	Reduce dose 25–50%	Reduce dose 50–75%	Avoid	Active metabolite accumulates
Fentanyl	Individualized (patch/IV)	No dose change needed	No dose change; monitor	Preferred opioid in CKD/ESRD	Safest opioid in renal impairment
Pregabalin	75–300 mg q12h	75 mg q12h	25–50 mg q12h or q24h	25 mg q24h; supplement post-HD	Dose by eGFR category strictly
Gabapentin	300–1200 mg q8h	200–700 mg q12h	200–700 mg q24h	200–300 mg post-HD	Accumulates; sedation/toxicity in CKD

⚠ Avoid morphine, codeine, hydromorphone, and oxycodone in advanced CKD (eGFR < 30) due to accumulation of active metabolites.

⚠ Gabapentin and pregabalin are almost entirely renally excreted and must be strictly dose-adjusted in CKD to prevent sedation, cognitive impairment, and peripheral edema.

✓ Fentanyl is the opioid of choice in ESRD — it is hepatically metabolized to inactive metabolites and is safer than morphine equivalents.

✓ Paracetamol at standard doses (up to 3 g/day in mild-moderate CKD) is the safest analgesic across all stages of CKD.

Miscellaneous Drugs

A wide range of drugs from multiple therapeutic classes require renal dose adjustments. Below are commonly encountered medications in clinical practice spanning antivirals, immunosuppressants, gastroenterology, rheumatology, and psychiatry.

TABLE .6 RENAL DOSE ADJUSTMENT OF DRUGS

Drug	Normal Dose	eGFR 30–59	eGFR 10–29	eGFR < 10 / HD	Notes
Methotrexate	Variable	Reduce dose; monitor	Avoid	Contraindicated	Severe toxicity in renal impairment
Acyclovir	400–800 mg q4–5h	400 mg q8h	200–400 mg q12h	200 mg q12h; dose post-HD	Crystalline nephropathy risk; hydrate well
Valacyclovir	1 g q8h (HSV)	500 mg q8–12h	500 mg q24h	500 mg q24h post-HD	Converted to acyclovir
Oseltamivir	75 mg q12h	75 mg q24h (eGFR 30–59)	30–75 mg q24h	30 mg q48h; supplement post-HD	
Lithium	600–1200 mg/day	Reduce dose; TDM	Use with extreme caution	Avoid	Narrow therapeutic index; nephrotoxic long-term
Ranitidine / Famotidine	150 mg BID / 20 mg BID	Famotidine 20 mg q24h	Famotidine 20 mg q36–48h	Famotidine 20 mg q48h	Famotidine preferred; ranitidine withdrawn
Colchicine	0.5–1.2 mg/day	Reduce; monitor for toxicity	0.5 mg q24h; caution	Avoid	Neuromuscular toxicity risk
Bisphosphonates (IV)	Variable	Use with caution; hydrate	Avoid (eGFR <35)	Contraindicated	Acute tubular necrosis risk

⚠ Lithium is both renally excreted and nephrotoxic long-term. Regular TDM and monitoring of renal function are mandatory.

⚠ IV bisphosphonates (zoledronic acid, pamidronate) can cause acute tubular necrosis. Always ensure adequate hydration and avoid in severe CKD.

✓ Methotrexate is contraindicated in significant renal impairment due to risk of severe bone marrow suppression and mucositis.

Special Considerations;

Haemodialysis (HD) Patients

Patients on intermittent haemodialysis (typically 3 sessions/week) have near-zero residual renal function (eGFR equivalent ~5–10 mL/min). Several important principles apply:

Give dialyzable drugs after the HD session to prevent removal of the dose.

Supplement doses of dialyzable drugs post-HD (e.g., acyclovir, gabapentin, some antibiotics).

Non-dialyzable drugs still accumulate between sessions and require interval-based adjustments.

Peritoneal Dialysis (PD) Patients

Peritoneal dialysis provides slower, continuous clearance. Drug clearance differs from HD — many drugs cleared by HD are not removed by PD. Use drug-specific PD dosing references when available. Some antibiotics can be administered intraperitoneally for peritonitis treatment.

Acute Kidney Injury (AKI)

AKI requires dynamic dose adjustment as renal function fluctuates. Use the most current creatinine to estimate

GFR, but remember that serum creatinine lags behind actual GFR changes. In oliguric AKI, assume minimal renal drug clearance. As function recovers, doses may need upward adjustment.

Elderly Patients

Older adults frequently have reduced muscle mass, leading to "normal" creatinine despite significantly reduced GFR. The CKD-EPI equation is preferred for estimation. Consider using Cockcroft-Gault with actual body weight in pharmacokinetic calculations. Always start low and titrate slowly in elderly CKD patients.

Drugs Requiring Extra Caution or Avoidance in CKD

Drug / Class	Risk in CKD	Recommendation
NSAIDs (all)	AKI, fluid retention, hyperkalaemia, HTN	Avoid in eGFR < 60; absolutely contraindicated in eGFR < 30
Aminoglycosides	Direct nephrotoxicity, ototoxicity	Use only when necessary; strict TDM; short courses; hydrate well
Metformin	Lactic acidosis	Contraindicated if eGFR < 30; withhold before contrast procedures
Nitrofurantoin	Ineffective + pulmonary toxicity	Avoid if eGFR < 45 mL/min
IV Contrast Media	Contrast-induced nephropathy	Pre-hydrate; minimize volume; avoid nephrotoxic combinations; hold metformin
Lithium	Nephrotoxicity; narrow TI	Avoid in advanced CKD; strict TDM mandatory
Spironolactone	Severe hyperkalaemia	Avoid if eGFR < 30 unless specialist-supervised
Dabigatran	Bleeding; 80% renally excreted	Avoid if eGFR < 30; prefer apixaban in advanced CKD

DISCUSSION:

Renal dose adjustment plays a vital role in ensuring safe and effective pharmacotherapy. It requires understanding of pharmacokinetics, patient-specific factors, and clinical judgment. Inappropriate dosing in renal impairment may lead to drug accumulation and toxicity, while underdosing can result in therapeutic failure. Therefore, individualized dosing strategies, continuous monitoring, and reassessment are essential components of clinical practice.

CONCLUSION:

CKD demands continuous, eGFR-guided reassessment of every prescribed medication. The key principles are clear: avoid nephrotoxins, reduce doses proportionally to renal function, apply TDM for narrow therapeutic index drugs, and review medications whenever kidney function changes. Emerging agents such as SGLT2 inhibitors now extend cardiorenal benefits at lower eGFR thresholds, reflecting a rapidly evolving landscape. Safe prescribing in CKD is ultimately a team responsibility — requiring pharmacists, physicians, and informed patients working together to prevent avoidable harm.

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DISCLAIMER & REFERENCES:

The dosing recommendations in this guide are derived from peer-reviewed literature, authoritative prescribing references, and current clinical practice guidelines. They represent general guidance and must be individualised based on patient-specific factors including clinical condition, body weight, co-medications, pharmacogenomics, and local formulary policy. This document is intended for use by qualified healthcare professionals and does not replace clinical judgment, specialist pharmacist input, or consultation with current product prescribing information. Drug dosing guidance is subject to revision as new evidence emerges — always verify against the most current version of relevant guidelines and product monographs before prescribing.

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