

The PRECISE framework: a structured approach to antibiogram interpretation for antimicrobial optimization at the bedside

El marco PRECISE: un enfoque estructurado para la interpretación del antibiograma y la optimización del tratamiento antimicrobiano a pie de cama

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Abstract

Antimicrobial resistance claimed an estimated 4.95 million lives in 2019. When clinicians prescribe empiric therapy that follows guidelines, mortality drops by roughly a third; when they de-escalate on the basis of culture results, it drops by more than half. Yet the document that should drive both decisions, the antibiogram, is often read superficially or misinterpreted, particularly by physicians without infectious-disease training. Existing stewardship frameworks address prescribing principles, timing, or ecological impact, but none walks the clinician step by step through an individual susceptibility report and out the other side with a treatment plan. PRECISE is a seven-step mnemonic designed for that purpose. Each letter corresponds to a clinical action: Pathogen assessment, Resistance phenotype interpretation, Evaluate MIC, Curtail spectrum, Individualize PK/PD, Switch route, and Endpoint optimization. Three clinical vignettes (including one in which the steps deliberately collide) illustrate how the framework operates in practice.

Keywords: Anti-bacterial agents. Antimicrobial stewardship. Microbial sensitivity tests. Drug resistance. Bacterial. Sepsis. Clinical decision-making.

Resumen

La resistencia a los antimicrobianos se asoció a unos 4,95 millones de muertes en 2019. Cuando el tratamiento empírico sigue las guías, la mortalidad se reduce en torno a un tercio; cuando se desescala según los resultados de los cultivos, se reduce en más de la mitad. Sin embargo, el documento que debería orientar ambas decisiones, el antibiograma, se lee con frecuencia de forma superficial o se malinterpreta, sobre todo por médicos sin formación en enfermedades infecciosas. Los marcos actuales de optimización de antimicrobianos abordan los principios de la prescripción, su cronología o el impacto ecológico, pero ninguno acompaña al clínico

paso a paso a través de un informe de sensibilidad individual hasta un plan de tratamiento. PRECISE es una regla nemotécnica de siete pasos diseñada con ese fin. Cada letra corresponde a una acción clínica: evaluación del Patógeno (Pathogen), interpretación del fenotipo de Resistencia, Evaluación de la CMI, reducción del espectro (Curtail), individualización de la PK/PD, cambio de vía (Switch) y optimización del punto final (Endpoint). Tres viñetas clínicas (una de ellas diseñada para que los pasos entren deliberadamente en conflicto) ilustran cómo funciona el marco en la práctica.

Palabras clave: Agentes antibacterianos. Programas de optimización del uso de antimicrobianos. Pruebas de sensibilidad microbiana. Resistencia bacteriana a los fármacos. Sepsis. Toma de decisiones clínicas.

The antibiogram interpretation gap

Antimicrobial resistance is now among the leading causes of death worldwide, associated with an estimated 4.95 million deaths in 2019 [1]. Ask an internal-medicine resident how to read an antibiogram, and you will likely get a hesitant answer. In a US survey, 22% of residents did not know how to access their own institution's cumulative antibiogram, and fewer than half had completed an infectious-disease rotation [2]. This is not a trivial knowledge gap. The Schuts meta-analysis showed that guideline-concordant empiric therapy reduces mortality by 35% (RR 0.65, 95% CI 0.54–0.80) and that culture-guided de-escalation reduces it by 56% (RR 0.44, 95% CI 0.30–0.66) [3]. In other words, how a clinician reads the susceptibility report has a measurable effect on whether the patient survives.

The stewardship community has built an impressive set of tools over the past two decades. The “5 D’s” (Diagnosis, Drug, Dose, Duration, De-escalation) provide a simple checklist [4]. Start Smart Then Focus structures the prescribing arc into initiation and a 48–72-hour review [5]. The AHRQ Four Moments framework offers four decision nodes [6]. Australia's MIND ME captures prescribing principles in six letters [7]. The antibiotic time-out concept systematizes reassessment at 48–72 hours [8]. The WHO AWaRe classification sorts drugs by ecological risk [9]. These are useful, but they all share a limitation: none of them tell the clinician what to do with the specific susceptibility report sitting in front of them.

On the laboratory side, the European tradition of interpretive reading, systematized by Courvalin in the 1990s and codified in the EUCAST expert rules [10,11], offers a rigorous method for deducing resistance mechanisms from phenotypic patterns. But it was built by microbiologists for microbiologists. It stops at mechanism identification. It does not extend to drug selection, dosing, route, or duration.

The result is a gap between two established traditions: stewardship frameworks that address the “when” and “what” of prescribing and interpretive

reading that addresses the “what mechanism.” Neither provides a systematic “how” for the frontline clinician at the moment of antibiogram review. Three recent developments have made this gap clinically urgent. First, the EUCAST redefinition of “I” as “Susceptible, Increased Exposure” (v9.0, 2019) demands that clinicians understand PK/PD optimization to use “I” results correctly [12,13]. Second, mechanism-specific drugs for carbapenem-resistant Enterobacteriales now require knowing the carbapenemase type (KPC, OXA-48, or NDM) to select the right agent [14]. Third, a wave of landmark trials (BLING-III, BALANCE, POET, OVIVA, and SABATO) has redefined how we dose, for how long, and by what route, but none of this evidence reaches the patient unless it is integrated into bedside reasoning.

This viewpoint proposes PRECISE, a seven-step mnemonic that guides non-ID clinicians from antibiogram receipt to treatment optimization.

The PRECISE framework

Each letter in PRECISE represents a clinical action. The steps are intentionally sequential: the output of one feeds the input of the next, creating a structured pathway from organism identification through treatment endpoint. **Figure 1** summarizes the seven steps and their sequence.

P – Pathogen: identify and validate

Nothing else matters if the organism on the report is a contaminant. Before touching the susceptibility panel, the clinician should ask: “is this a true pathogen?”

For blood cultures, the answer is usually straightforward. *S. aureus*, *E. coli*, *P. aeruginosa*, and *Candida* species represent true bacteremia in over 90% of episodes. *Coagulase-negative staphylococci* are contaminants in the majority of cases; the decision hinges on the number of concordant sets (≥ 2 with matching species and antibiogram), species identity (*S. lugdunensis* is always a pathogen), and the clinical picture [15]. The CDC benchmark for contamination is $<3\%$ [16].

PRECISE

Antibiogram interpretation and antimicrobial optimization at the bedside

A seven-step clinical pathway that turns an individual susceptibility report into the right drug, exposure, route, duration and reassessment plan.

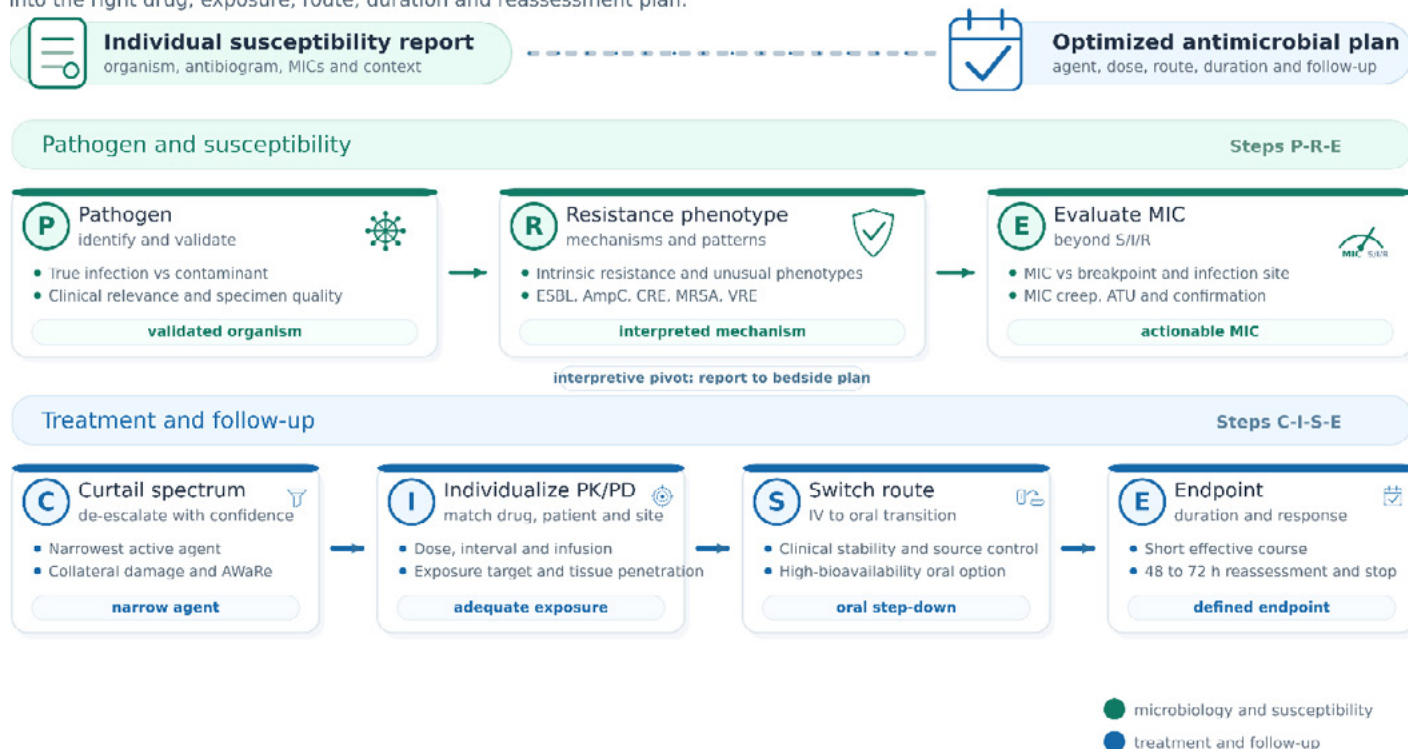


Figure 1. The PRECISE framework for antibiogram interpretation and antimicrobial optimization. The first three steps address pathogen and susceptibility; the last four address treatment and follow-up

P: Pathogen; R: Resistance phenotype; E: Evaluate MIC; C: Curtail spectrum; I: Individualize PK/PD; S: Switch route; E: Endpoint.

Intrinsic resistance serves as a built-in quality check. If an *Enterococcus* appears susceptible to cephalosporins or *P. aeruginosa* susceptible to ertapenem, something is wrong: either the identification or the susceptibility test. The EUCAST intrinsic resistance tables (v3.3, 2021) provide the reference [17]. Other critical intrinsic resistances: *S. maltophilia* versus carbapenems; *Proteus mirabilis* versus nitrofurantoin; and *Serratia marcescens* versus ampicillin, first/second-generation cephalosporins, and colistin [17].

Local epidemiology completes the picture. The institutional cumulative antibiogram (CLSI M39) [18] or, where available, a weighted-incidence syndromic combination antibiogram (WISCA) [19] contextualizes individual results and flags unusual phenotypes that warrant confirmation.

R – Resistance: interpret the phenotype

Once the organism is validated, the next question is: what resistance mechanism is driving this phenotype?

This was once an academic exercise; it is now therapeutically essential. With mechanism-specific drugs available for CRE, identifying the carbapenemase type directly determines which drug to use [14].

- ESBLs: The classic pattern, third-generation cephalosporin resistance with clavulanate synergy and cefoxitin susceptibility, remains the key phenotypic marker. MERINO settled the treatment debate: piperacillin-tazobactam is inferior to meropenem for ESBL-producing *E. coli*/*K. pneumoniae* bacteremia [20]. The 2024 IDSA guidance is clear: novel BL/BLI combinations are reserved for CRE, not for ESBL producers [14].
- AmpC: The ESCPM group (*Enterobacter cloacae* complex, *Serratia*, *Citrobacter freundii*, *Providencia*, and *Morganella*) and *K. aerogenes* harbor inducible chromosomal AmpC that can derepress during cephalosporin therapy. Current IDSA guidance recommends against using ceftriaxone for serious

non-urinary infections even when the isolate tests susceptible, favoring extended-infusion cefepime or carbapenems instead [14,21]. Phenotypically, AmpC differs from ESBL in showing cefoxitin resistance and no clavulanate synergy [21].

- Carbapenemases: Ambler class now maps directly to treatment. Class A (KPC): ceftazidime-avibactam or meropenem-vaborbactam. Class B (NDM, VIM, IMP): aztreonam-avibactam, cefiderocol, or the ceftazidime-avibactam plus aztreonam combination. Class D (OXA-48-like): ceftazidime-avibactam [14]. Phenotypic detection includes mCIM/eCIM (CLSI), lateral flow immunoassays, and CarbaNP [22]. Geographic distribution varies: KPC in the Americas and southern Europe, OXA-48 in the Middle East and North Africa, and NDM in South Asia [23].
- MRSA: Cefoxitin or oxacillin resistance means resistance to all β -lactams except ceftaroline and ceftobiprole (EUCAST expert rules, Grade A) [11]. Ceftobiprole, authorized in the European Union since 2014, and obtained a specific indication for *S. aureus* bacteremia on the basis of the ERADICATE trial, with FDA approval in April 2024; alongside ceftaroline, it remains a β -lactam option in this setting [24].
- VRE: VanA means high-level resistance to both vancomycin and teicoplanin; VanB means variable vancomycin resistance with preserved teicoplanin susceptibility. Treatment options: linezolid, daptomycin, and tigecycline [11].

Other indicators, such as the D-test for inducible clindamycin resistance, are mandatory on erythromycin-resistant, clindamycin-susceptible staphylococci and streptococci [25]. Stepwise fluoroquinolone resistance suggests accumulating *gyrA*/*parC* mutations or plasmid-mediated mechanisms (PMQR). Selective aminoglycoside resistance patterns point to specific modifying enzymes that determine which agent retains activity [10].

E – Evaluate MIC: calibrate beyond S/I/R

The S/I/R category is a summary, not the whole story. The actual MIC value matters in several clinical scenarios [26]:

- Site-specific breakpoints: EUCAST sets distinct thresholds by infection site. Penicillin for *S. pneumoniae*: $S \leq 0.06$ mg/L in meningitis versus $S \leq 2$ mg/L in pneumonia. Missing this distinction can mean treating meningitis with a drug that the laboratory technically calls “susceptible”.

- MIC creep: For MRSA bacteremia, vancomycin MICs ≥ 1.5 mg/L by Etest correlate with higher treatment failure and mortality even within the susceptible range [27]. When the MIC approaches the breakpoint, alternative agents (daptomycin, ceftobiprole) deserve consideration.
- The EUCAST “I” redefinition: Since Breakpoint Table v9.0, “I” means “Susceptible, Increased Exposure”: not intermediate, not a grey zone, but an explicit instruction to use higher doses, prolonged infusions, or both [12,13]. For *P. aeruginosa*, where wild-type organisms fall into “I” for multiple antipseudomonals, this means optimized dosing is not optional: it is the default.
- Area of Technical Uncertainty (ATU): EUCAST flags MIC/zone ranges near breakpoints where misclassification risk is high. In one study, retesting ATU results reclassified 35.9% of *P. aeruginosa* piperacillin-tazobactam results from S to R [28]. Results within the ATU warrant caution and, ideally, confirmatory testing.

C – Curtail spectrum: de-escalate with confidence

The instinct to keep broad coverage “just in case” is among the hardest habits to break in antimicrobial prescribing. The evidence, however, is unambiguous: de-escalation is safe.

A systematic review found reduced ICU mortality with de-escalation (RR 0.68, 95% CI 0.52–0.88) [29]. A target trial emulation in 36,924 patients with community-onset sepsis across 67 hospitals found that de-escalation from anti-MRSA or antipseudomonal coverage was associated with similar 90-day mortality, fewer antibiotic days (RR 0.91), and shorter hospital stays (RR 0.88) [30]. Despite this, the DIANA study found that only 16% of ICU patients actually undergo de-escalation, a striking gap between evidence and practice [31].

Not all antimicrobials are equal in their collateral damage. A large case-control study (159,404 CDI cases) quantified the hierarchy: clindamycin topped the list (OR 25.39), followed by later-generation oral cephalosporins (OR 9–12) and amoxicillin-clavulanate (OR 8.53), while doxycycline was neutral and minocycline was actually protective (OR 0.79) [32]. Fluoroquinolone restriction programs have driven substantial reductions in CDI, largely through the elimination of fluoroquinolone-resistant lineages [33]. The WHO AWaRe classification provides an additional lens for assessing ecological impact [9]. The practical rule is simple: once you know the pathogen and its susceptibility profile, pick the narrowest agent that can

achieve adequate exposure at the site of infection. Which leads directly to the next step.

I – Individualize PK/PD: match drug to patient and site

Choosing the right drug is not enough. Delivering it at the right dose, through the right infusion strategy, to the right anatomical compartment is what separates adequate from optimal therapy.

- PK/PD targets: β -lactams are time-dependent (target: 100% fT>MIC in critical illness); aminoglycosides are concentration-dependent ($C_{max}/MIC \geq 8-10$); vancomycin and fluoroquinolones are AUC/MIC-driven (vancomycin AUC/MIC 400–600 per the 2020 ASHP/IDSA/PIDS/SIDP consensus) [34,35].
- Prolonged β -lactam infusion: The evidence for prolonged infusion is now strong, though not unequivocal. In BLING-III (7,031 patients, 104 ICUs), continuous infusion did not significantly reduce 90-day mortality in the primary analysis (24.9% vs 26.8%; OR 0.91, 95% CI 0.81–1.01; $p=0.08$). The companion Bayesian meta-analysis (17 RCTs, 9,014 patients) found a risk ratio of 0.86 (95% credible interval 0.72–0.98), a 99.1% posterior probability of benefit, and a number needed to treat of 26. Seven professional societies endorse prolonged infusion as preferred practice [36–38]. Intermittent bolus dosing of β -lactams in critically ill patients should now be considered suboptimal.
- Site-specific penetration: Fluoroquinolones and macrolides reach ELF/plasma ratios >1 (good for pneumonia); vancomycin penetrates ELF poorly ($\sim 0.1-0.2$ corrected); meropenem and linezolid achieve adequate CSF concentrations for meningitis; rifampin has unique biofilm and bone penetration. Augmented renal clearance ($CrCl \geq 130$ mL/min), present in 30–65% of critically ill patients, leads to subtherapeutic levels of renally cleared drugs unless doses are increased [39,40].

S – Switch route: transition to oral when appropriate

Three randomized trials have fundamentally changed how we think about IV-to-oral transition:

POET (NEJM 2019) randomized 400 patients with stable left-sided endocarditis to oral step-down versus continued IV after ≥ 10 days. Oral was noninferior (composite endpoint 9.0% vs. 12.1%); hospital stays dropped from 19 to 3 days. At a 5.4-year follow-up, mortality was actually lower with an oral switch: 23.4% versus 35.2% (HR 0.61, 95% CI 0.42–0.88) [41,42].

OVIVA (NEJM 2019) enrolled 1,054 patients with bone and joint infections. Oral was noninferior to IV (failure

14.6% vs. 13.2%), and IV catheter complications dropped from 9.4% to 1.0% [43].

SABATO (Lancet Infect Dis 2024) showed noninferiority of early oral switch in low-risk *S. aureus* bacteremia, though it was terminated early. The larger SNAP trial is ongoing [44].

A 2025 systematic review (38 studies) confirmed noninferiority of oral switch in bacteremia, with hospital stay reduced by 5.19 days [45]. The key to success: high-bioavailability agents. Levofloxacin ($\sim 99\%$), linezolid ($\sim 100\%$), TMP-SMX ($\sim 90-100\%$), metronidazole ($\sim 100\%$), fluconazole ($\sim 90\%$), and clindamycin ($\sim 90\%$) are the workhorses of step-down therapy.

E – Endpoint: optimize duration and assess response

The final step asks two questions: “How long should we treat?”, and “is the treatment working?”

- Duration: BALANCE (NEJM 2025; 3,608 patients, 74 hospitals, 7 countries) established that 7 days is noninferior to 14 for non-*S. Aureus* bacteremia (90-day mortality 14.5% vs. 16.1%) [46]. STOP-IT showed 4 $\pm$ 1 days suffice for complicated intra-abdominal infections with source control [47]. ATS/IDSA 2019 endorses ≥ 5 days for CAP [48]. The pattern is consistent: shorter courses, when supported by clinical stability, perform as well as longer ones.
- Biomarkers: Procalcitonin-guided discontinuation shortens antibiotic duration by roughly 1.5–2 days in ICU patients [49,50]. In critically ill adults, a meta-analysis of 16 RCTs (5,158 patients) found it associated with lower mortality (RR 0.89, 95% CI 0.83–0.97) [51].
- Structured reassessment: The CDC Antibiotic Time-Out prompts review at 48–72 hours. In a single-centre before-and-after study in a Japanese academic ICU, structured antibiotic time-outs during multidisciplinary rounds improved survival to discharge (SHR 1.13, 95% CI 1.02–1.25) [8,52].

Applying PRECISE: clinical vignettes

Three vignettes illustrate the framework in practice. The third is deliberately designed to show what happens when steps conflict. All three cases are hypothetical and were constructed for illustration; they do not correspond to identifiable patients.

Community-acquired pneumonia: from broad to narrow

Case A: A 72-year-old man, CURB-65 score 2, admitted with CAP. Sputum culture grows *Streptococcus*

pneumoniae. Empiric therapy: ceftriaxone plus azithromycin (**Table 1**).

Teaching point: PRECISE enabled a triple de-escalation: combination → monotherapy, IV → oral, and long → short course. Each step is evidence-based and explicitly justified.

MRSA bloodstream infection: navigating route transition

Case B: A 54-year-old man with catheter-related MRSA bacteremia. Two of two blood culture sets were positive (**Table 2**).

Teaching point: The most cited duration evidence does not always apply. BALANCE established 7 days for bloodstream infection, but excluded *S. aureus*, and applying it here would shorten therapy in the one bacteremia where duration matters most. Step E forces the question of whether the trial population includes the organism at hand, which is where the framework prevents an error that guidelines alone would not.

KPC-producing *K. pneumoniae* VAP: when steps collide

Case C: A 62-year-old man, postoperative day 2, ventilated. New infiltrates, purulent secretions. Tracheal aspirate: *K. pneumoniae*. CrCl 35 mL/min (**Table 3**).

Teaching point: PRECISE does not always yield a clean linear answer. When C and I conflict, the MIC from step E and the patient context arbitrate. The framework’s value is in making that tension visible and

forcing explicit resolution, not in eliminating clinical judgment.

Positioning PRECISE within the stewardship landscape

The Cochrane review by Davey et al. (221 studies) demonstrated a 15% increase in prescribing compliance and a 1.95-day reduction in treatment duration with stewardship interventions [53]. Mnemonic-based educational tools improve pharmacotherapy decision-making in a randomized trial [54]. Yet the field suffers from a behavioral science deficit: only 4 of 2,014 records in a scoping review described theory-informed acute care stewardship interventions [55].

PRECISE targets the capability domain of the COM-B behavioral model [56] by providing a cognitive scaffold for a task that most non-ID clinicians find difficult. Structured antibiogram training increased resident comprehension from 57% to 89% ($p < 0.001$) in a quality improvement study [57]. Integration with clinical decision support systems (CDSS) could amplify the effect: a meta-analysis of 57 AMS-CDSS studies found an OR of 2.28 for appropriate therapy and an 18% relative mortality reduction [58]. In Spain, national PROA consensus documents and educational, non-restrictive stewardship programmes have shown that structured interventions can improve prescribing and reduce antimicrobial consumption without compromising outcomes [59,60]; PRECISE is designed to operationalize that philosophy at the level

Table 1. Case A.

Step	Clinical reasoning
P	<i>S. pneumoniae</i> in quality sputum with concordant clinical and radiological findings. No intrinsic resistance concerns.
R	Penicillin susceptible (MIC 0.03 mg/L). Erythromycin susceptible. No unusual resistance phenotype.
E	MIC well below pneumonia breakpoint (EUCAST: $S \leq 0.5$ IV; $S \leq 2$ non-meningitis). No ATU concern. Supports definitive narrow-spectrum therapy.
C	De-escalate from ceftriaxone + azithromycin to ampicillin monotherapy. Cephalosporin CDI risk (OR 9–12) drops dramatically with aminopenicillins (OR ~3).
I	Ampicillin 2 g IV q6h. Time-dependent killing; dosing interval ensures $fT > MIC$. Adequate ELF penetration at this MIC. No renal adjustment needed.
S	Switch to amoxicillin 1 g PO q8h once clinically stable (≥ 48 h afebrile, hemodynamically stable, functioning GI, and able to swallow). Bioavailability is ~80%, adequate at this MIC.
E	A total of 5 days per ATS/IDSA 2019 guidelines, provided stability criteria are met. No follow-up cultures are needed.

of the individual susceptibility report. **Table 4** maps PRECISE against the main stewardship frameworks across these clinical domains.

Limitations and future directions

PRECISE is a proposed framework, not yet validated. Its main limitations are the absence of prospective outcome data, dependence on MIC availability (not universal in resource-limited settings), the assumption

of adequate laboratory infrastructure, and the well-documented difficulty of translating any structured tool into sustained behavioral change [55,56]. Priority next steps include before-and-after implementation studies, cluster-randomized educational trials comparing PRECISE against standard stewardship training, CDSS integration studies, and adaptation research in low- and middle-income settings where the AMR burden is highest but microbiological resources are most constrained.

Table 2. Case B.

Step	Clinical reasoning
P	<i>S. aureus</i> in ≥ 2 sets = true pathogen (>99%). Remove the catheter. Local nosocomial MRSA rate: 35%.
R	MRSA confirmed (cefoxitin R). D-test negative. All β -lactams were excluded except ceftaroline/ceftobiprole.
E	MICs (mg/L): vancomycin 1 (S); daptomycin 0.5 (S); linezolid 1 (S); TMP-SMX ≤ 0.5 (S). Vancomycin MIC <1.5 mg/L: no creep concern.
C	Spectrum is inherently restricted for MRSA BSI. Options: vancomycin (AUC/MIC-guided), daptomycin (concentration-dependent bactericidal), and ceftobiprole (emerging β -lactam option).
I	Daptomycin preferred: rapid bactericidal activity, C _{max} /MIC-driven. Alternative: vancomycin AUC/MIC 400–600. Ceftobiprole 500 mg q8h is a new option (ERADICATE trial) [24].
S	Plan a switch to linezolid PO (~100% bioavailability) after 7–10 days IV + negative follow-up cultures. SABATO supports early oral switch in low-risk SAB [44].
E	≥ 14 days from first negative culture (SAB-specific; BALANCE 7-day data does not apply to <i>S. aureus</i>). Echocardiography is mandatory. Weekly CPK if daptomycin is used.

Table 3. Case C.

Step	Clinical reasoning
P	<i>K. pneumoniae</i> in tracheal aspirate with concordant clinical syndrome. Assess specimen quality (PMN predominance). Clinical correlation supports true infection.
R	Ertapenem and meropenem resistant. mCIM+, eCIM–: serine carbapenemase (KPC or OXA-48). Lateral flow immunoassay confirms KPC. Ambler Class A: both CAZ-AVI and MEV are expected to be active.
E	MICs (mg/L): meropenem 16 (R); CAZ-AVI 2 (S); MEV 0.5 (S); colistin 1 (S); tigecycline 0.5 (S). CAZ-AVI MIC 2 mg/L is susceptible with margin (EUCAST breakpoint S ≤ 8 mg/L); MEV MIC 0.5 mg/L is even lower.
C→I	Here the tension emerges. Step C favors CAZ-AVI as the narrower agent. But step I complicates things: CAZ-AVI ELF penetration data for VAP are limited, whereas MEV achieves better pulmonary concentrations. CrCl 35 mL/min requires dose reduction for both. At the renal-adjusted CAZ-AVI dose (1.25 g/0.31 g q8h for CrCl 31–50), the MIC of 2 mg/L sits comfortably within the susceptible range; MEV's MIC of 0.5 mg/L provides a 4-fold buffer. Resolution: CAZ-AVI 1.25 g/0.31 g q8h (renal dose for CrCl 31–50) via 3-hour extended infusion to maximize fT>MIC. The MIC of 2 mg/L remains achievable. Colistin excluded (nephrotoxicity with CrCl 35). Tigecycline was excluded for VAP (excess mortality signal in prior studies). Monitor response at 48–72h; if inadequate, reconsider MEV.
S	Not applicable. No oral CAZ-AVI formulation. A full IV course is required.
E	Duration: 7 days for VAP with adequate clinical response. Serial PCT for discontinuation guidance. Close renal monitoring.

Table 4. Comparative mapping of antimicrobial stewardship frameworks across clinical domains of antibiogram interpretation and therapeutic optimization

Framework	Pathogen validation	Resistance mechanism	MIC interpretation	Spectrum restriction	PK/PD optimization	IV→oral switch	Duration endpoint	Mnemonic format	Validation data
5 D's: Diagnosis, Drug, Dose, Duration, De-escalation	–	–	–	✓	Partial	Implicit	✓	✓	–
SSTF Start Smart Then Focus (UK)	–	–	–	✓	–	✓	Implicit	–	✓
Four Moments AHRQ (US)	Partial	–	–	✓	–	Implicit	✓	–	✓
MIND ME Antimicrobial Creed (AU)	Implicit	–	–	✓	Partial	–	✓	✓	–
Antibiotic Time-Out CDC (US)	–	–	–	✓	–	Implicit	✓	–	✓
WHO AWaRe Access / Watch / Reserve	–	–	–	✓	–	–	–	✓	✓
EUCAST Expert Rules Interpretive reading	✓	✓	✓	–	–	–	–	–	✓
PRECISE This proposal	✓	✓	✓	✓	✓	✓	✓	✓	Pending

✓: explicitly addressed; Partial/Implicit: touched on but not structured; –: not addressed; SSTF: Start Smart Then Focus; AHRQ: Agency for Healthcare Research and Quality; AWaRe: Access/Watch/Reserve.

Conclusion

The antibiogram remains one of the most powerful and underused tools in clinical medicine. PRECISE offers a structured pathway from susceptibility report to treatment plan, integrating interpretive microbiology with therapeutic optimization evidence. Each step rests on robust data: BLING-III for prolonged infusion [36], BALANCE for duration [46], and POET/OVIVA/SABATO for route switching [41-44]. Together with its Spanish-language companion MICROBE, PRECISE provides a globally applicable, evidence-grounded framework for what may be the most consequential daily decision in hospital medicine: how to turn a susceptibility report into the right treatment for the patient in front of you.

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Author contributions

CPO: Conceptualization, Investigation, Formal analysis, Writing – original draft, Validation, Writing – review and editing.

AG: Investigation, Formal analysis, Writing – original draft, Validation, Writing – review and editing.

MB: Validation, Writing – review and editing, Supervision.

All authors have read and approved the final version of the manuscript.

Conflicts of interest

The authors declare that there is no commercial or financial conflict of interest for this research.

AI statement

A large language model (Claude, Anthropic) was used to verify the bibliographic references against PubMed and CrossRef, to translate the abstract into Spanish, and to assist with editorial revision and reference formatting. It was not used to conceive the framework, to design or interpret the clinical vignettes, or to generate any clinical recommendation. All output was reviewed, corrected, and approved by the authors, who take full responsibility for the content of the manuscript.

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