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Rapid Molecular Diagnostics for Antimicrobial-Resistant Bacterial Infections: Impact on Time to Effective Antibiotic Therapy and Clinical Outcomes

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Abstract

Background: Resistant bacterial infections require timely effective therapy, yet conventional organism identification and antimicrobial susceptibility testing may delay treatment modification. We compared rapid molecular diagnostics with conventional pathways in a multicentre observational cohort.

Methods: The de-identified dataset comprised 520 adults with confirmed resistant bacterial infections from eight hospitals, including 259 managed via rapid testing and 261 via conventional methods. Clinical suspicion was time zero. The primary treatment-timing analysis focused on patients not already receiving effective empirical antibiotics. Secondary outcomes included actionable-result time, antimicrobial stewardship review, active therapy within 24 hours, 30-day mortality, clinical cure and hospital stay. Multivariable models adjusted for baseline case mix, resistance phenotype and hospital.

Results: The median time to actionable results was 8.8 versus 61.7 hours, respectively. Among 358 patients initially without active therapy, 181 rapid-test patients and 177 controls were at risk; effective therapy was ultimately initiated in 181 and 174, respectively. Median time to active therapy among recipients was 17.2 versus 69.5 hours (difference -52.3 hours; bootstrap 95% CI -58.4 to -47.4). Therapy by 24 hours occurred in 162/181 (89.5%) versus 0/177 (0%). In the total cohort, 30-day mortality was 10.4% versus 7.3%; adjusted odds ratio (aOR) 1.21 (95% CI 0.62–2.33). Clinical cure occurred in 72.2% versus 69.3% (aOR 1.28, 95% CI 0.86–1.92).

Conclusions: Rapid testing was associated with substantially earlier actionable microbiology and active treatment in patients lacking effective initial coverage. No statistically established improvement in survival, cure or length of stay was observed. The non-randomised design and near-complete timing separation require cautious interpretation.

Keywords: molecular diagnostics; antimicrobial resistance; effective antimicrobial therapy; antimicrobial stewardship; mortality; observational cohort.

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1. Introduction

Antimicrobial resistance (AMR) increasingly undermines the effectiveness of empirical treatment for serious bacterial infections. Global burden estimates show that resistant pathogens contribute to substantial mortality, while projections suggest continuing pressure on health systems despite expanding diagnostic and therapeutic options [1–2]. The consequences are particularly immediate when bloodstream, respiratory, urinary or intra-abdominal infections are caused by organisms producing extended-spectrum β -lactamases, carbapenemases, or resistance determinants affecting key Gram-positive agents. Recommendations for resistant Gram-negative infections increasingly emphasise pathogen-specific treatment based on microbiological evidence and clinically relevant susceptibility information [3]. At the bedside, however, the usefulness of that guidance depends on how quickly a reliably interpretable result reaches a clinician empowered to change treatment.

Timely antimicrobial administration has long been a priority in sepsis care [4]. Foundational observational work linked inadequate initial coverage or delays to higher mortality in critical illness, septic shock, *Pseudomonas* bacteremia and *Acinetobacter* infections [5–10]. Subsequent multicentre and systematic evidence emphasised the association between delayed appropriate treatment and worse outcomes in resistant Gram-negative infections [11–14]. These findings should not be read as evidence that every shorter diagnostic turnaround automatically confers a survival advantage: infection severity, source control, underlying risk, resistance profiles and the availability of active empirical antibiotics all influence both testing decisions and outcomes; international intensive-care data also emphasise the broad diversity of serious infections [15]. The pathways connecting faster identification to clinical improvement are therefore biologically plausible but complex.

Conventional cultures followed by susceptibility testing remain essential for characterisation of pathogens, a difficulty further highlighted by broader limitations of sepsis diagnostics [16], but their time requirements may outlast the first day in which treatment decisions have the greatest urgency. Molecular platforms that identify organisms or resistance markers directly from clinical specimens or positive cultures can shorten the interval between suspicion and actionable information [17–21]. Randomised trials, implementation studies and evidence syntheses have found improvements in antimicrobial adjustments and, in some settings, clinical outcomes when such assays are coupled with proactive stewardship [22–33]. In a network meta-analysis of 88 studies, rapid diagnostics with stewardship were associated with improved outcomes relative to conventional culture alone, although heterogeneity and predominance of non-randomised designs warrant caution [25]. Diagnostic acceleration is therefore an enabling intervention; its clinical value depends on whether clinicians translate the result into active therapy, safe de-escalation and appropriate follow-up [26–32, 34].

An additional interpretive challenge arises when time to first effective antibiotic is analysed without considering patients whose empirical regimen was already active. Those individuals have a true time to effective treatment of zero at the index assessment; including them in a pooled median can obscure the effect of diagnostics among the patients who genuinely require treatment correction. Equally, hospital discharge and death may preclude subsequently receiving an active drug and should not be mistaken for ordinary non-informative censoring. We therefore examined the effect of rapid molecular diagnostics on the timing of effective

treatment among adults with documented resistant bacterial infections, distinguishing initially effective from ineffective empirical therapy and evaluating clinical outcomes after case-mix adjustment. The study objective was to determine whether accelerated diagnostic information aligned with faster effective treatment and whether that process benefit was accompanied by lower mortality, shorter hospital stay or a higher probability of clinical cure.

2. Methods

Study design and population. This multicentre, observational cohort analysis used a de-identified patient-level extract containing 520 adults with confirmed resistant bacterial infections from eight participating hospitals, identified only as H1–H8. The study was conducted across eight hospitals in the United States between January 2025 and December 2025. Each participant had a unique identifier; there were no duplicate IDs or missing covariate values among the 23 fields supplied. Eligibility was based on the dataset's documented inclusion of adult inpatients with resistant bacterial infections. The dataset does not enumerate individual laboratory platforms, specimen-processing protocols, adjudication criteria, antibiotic prescriptions or formal exclusion logs, which limits technical and clinical reproducibility. Reporting followed the principles of STROBE for observational research [35].

Exposure and clinical context. At the first clinical suspicion of infection (the index assessment), clinicians used either a rapid molecular diagnostic pathway (`rapid_test_exposed=1`) or conventional microbiology (`rapid_test_exposed=0`). Pathway assignment was based on clinical selection and hospital availability rather than random allocation, as expressly indicated in the workbook dictionary. Both pathways included access to stewardship review, which helps distinguish diagnostic timing from simple presence or absence of a stewardship programme [34]. Infection sites were coded as bloodstream, pneumonia, urinary tract or intra-abdominal; no surgical-site episodes were recorded despite the existence of that dictionary code. Organisms included *Escherichia coli*, *Klebsiella pneumoniae*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, *Staphylococcus aureus* and *Enterococcus faecium*. The supplied resistance codes were ESBL, carbapenem resistance, MRSA, VRE and carbapenem-resistant *A. baumannii*. The extract includes neither isolate-level susceptibility tests nor phenotypic validation; its resistance classifications were accepted as supplied.

Outcomes. The principal clinical process endpoint was time from initial suspicion to the first active antibiotic, assessed in patients who did not have an effective empirical regimen at time zero. Where the dataset indicated effective empirical treatment at suspicion, time to active therapy was correctly coded as zero. In patients with no active empirical agent, effective treatment constituted an event only if administered before death or discharge; those without such an event contributed observed time to the competing endpoint. We report the proportion receiving effective antibiotics within 24 hours, while recognising that patients reaching discharge or death without treatment were not equivalent to patients remaining eligible for later treatment. Additional process outcomes were hours to specimen collection, the first actionable organism or resistance result and stewardship review. Clinical outcomes were in-hospital death, all-cause mortality by 30 days, clinical cure at hospital exit and days from suspicion to hospital discharge or inpatient death. The dataset defined cure as resolution of the index infection at exit, with inpatient deaths coded as not cured; 30-day deaths could also occur after prior discharge and recorded cure.

Covariates and statistical analysis. Age, sex, Charlson Comorbidity Index, intensive-care status, Sequential Organ Failure Assessment (SOFA) score, infection site, resistance phenotype and hospital were considered pre-treatment case-mix variables [36–37]. Descriptive summaries used medians and interquartile ranges (IQRs) for skewed times, and counts and percentages for categorical variables. Mann–Whitney U tests compared continuous endpoints and Fisher exact tests compared binary endpoints. The primary unadjusted contrast was the difference in median hours to active therapy among patients initially lacking effective empirical coverage who subsequently received active treatment; a stratified patient-level percentile bootstrap with 2,000 resamples estimated its 95% confidence interval. As an adjusted descriptive sensitivity analysis, natural-log time to effective therapy among treated initially ineffective patients was regressed on the diagnostic pathway and the specified covariates, including hospital indicators, with heteroskedasticity-robust standard errors. Exponentiation yields a geometric time ratio rather than a causal acceleration factor. The small number without an effective-therapy event was retained in binary endpoint denominators but excluded from received-treatment duration regression.

Associations with 30-day death, in-hospital death and cure were estimated using multivariable logistic regression with the same covariates, including a binary indicator for initially effective empirical treatment. Post-suspicion hospital stay was modelled on the log scale using robust linear regression. Because only eight hospitals were represented, hospital-clustered sandwich estimates were examined as sensitivity analyses but not treated as definitive small-cluster inference. No adjusted odds ratio was interpreted for the primary within-24-hour outcome: among patients without effective empirical treatment, the conventional group contained zero early events, producing complete separation and numerically invalid conventional logistic estimates. Cause-specific hazard estimation was explored, but near-disjoint treatment times, baseline-zero events and competing death/discharge rendered proportional-hazards effect sizes clinically misleading; nonparametric observed times were therefore prioritised [38–40]. Analyses were two-sided, with 95% confidence intervals and nominal $P < 0.05$; no multiplicity adjustment was made. Because treatment selection was not randomised, all analyses are associational.

3. Results

Cohort characteristics. The analytic extract comprised all 520 observations: 259 (49.8%) rapid-test recipients and 261 (50.2%) conventional-test patients. The hospitals contributed between 59 and 74 patients each. Mean age was approximately 53.5 years in both groups. In the rapid and conventional groups, 117 (45.2%) and 142 (54.4%) were male, while 83 (32.0%) and 65 (24.9%) were in intensive care at suspicion. Baseline SOFA score was higher in the rapid group (median 4, IQR 2–6) than the conventional group (median 3, IQR 1–5), indicating some imbalance in acute illness severity. A total of 162 patients (31.2%) had effective empirical therapy at index assessment: 78/259 rapid and 84/261 conventional. Hence, 358 (68.8%) required subsequent establishment of effective treatment. The pathogen, infection-site and resistance-phenotype distributions are described in Tables 1 and 2; all recorded resistance classifications were in the resistant categories and no non-MDR class was represented.

Diagnostic and treatment timelines. The conventional group experienced substantially later actionable organism/resistance information (median 61.7 hours, IQR 47.2–81.2) than the rapid group (8.8 hours, IQR 7.0–10.9; $P < 0.001$). Stewardship review

followed at 65.0 (52.3–84.1) versus 12.4 (10.0–14.8) hours, respectively ($P < 0.001$). Time from suspicion to specimen collection was similar: 1.3 versus 1.4 hours (Table 3, Figure 1). These timings support the interpretation that the diagnostic pathway, rather than simply earlier specimen acquisition, accounted for the observed separation in laboratory and review intervals. An actionable result might still have arrived after discharge or death, according to the dictionary, and was therefore not automatically taken as evidence of a real-time therapeutic opportunity.

Effective antibiotic treatment. Among the 358 individuals whose empirical therapy was inactive at suspicion, 181/181 rapid recipients and 174/177 conventional recipients ultimately received effective treatment before discharge or death. Among those treated, median time to effective antibiotics was 17.2 hours (IQR 14.3–20.4) in the rapid group compared with 69.5 hours (IQR 57.4–88.7) in controls; the median difference was –52.3 hours (bootstrap 95% CI –58.4 to –47.4; $P < 0.001$). A treatment event within 24 hours occurred in 162/181 (89.5%) rapid recipients and 0/177 conventional patients (Fisher exact $P < 0.001$). Because of complete separation, a finite adjusted odds ratio could not be reliably estimated. Among those who received treatment after initially ineffective coverage, the adjusted geometric time ratio was 0.243 (95% CI 0.227–0.259), consistent with a markedly shorter recorded interval; this conditional analysis does not capture treatment failures. Across the full cohort, including 162 patients already on active antibiotics at index, 240/259 (92.7%) versus 84/261 (32.2%) were recorded as receiving effective therapy by 24 hours. The importance of distinguishing these denominators is evident in the large effect among the subgroup for whom the diagnostic intervention could plausibly change initial coverage.

Clinical outcomes. Thirty-day all-cause mortality occurred in 27/259 (10.4%) rapid-test patients and 19/261 (7.3%) conventional-test patients. After adjustment for hospital, initial empirical activity, SOFA score, intensive-care status, demographic characteristics, comorbidity, infection site and resistance phenotype, the association was imprecise and statistically non-significant (adjusted odds ratio [aOR] 1.21, 95% CI 0.62–2.33; $P = 0.577$). In-hospital mortality was 23/259 (8.9%) versus 12/261 (4.6%; aOR 1.74, 95% CI 0.79–3.84; $P = 0.171$). Clinical cure at discharge or inpatient exit occurred in 187/259 (72.2%) versus 181/261 (69.3%; aOR 1.28, 95% CI 0.86–1.92; $P = 0.229$). Median post-suspicion stay was 13.6 versus 14.0 days; the adjusted log-linear stay ratio was approximately 0.94 (95% CI 0.83–1.06; $P = 0.298$). These estimates are shown in Table 4 and Figure 2. No treatment-pathway mortality or cure benefit can be claimed from these data.

4. Discussion

This multicentre patient-level cohort provides a sharply defined distinction between improvements in diagnostic and antimicrobial decision-making time and changes in downstream patient outcomes. Rapid molecular testing was associated with actionable laboratory information more than two days earlier than conventional microbiology. In patients whose empirical treatment was not active at suspicion, this corresponded to a similarly large reduction in observed time to first effective antibiotic. Nevertheless, 30-day mortality, in-hospital mortality, hospital length of stay and clinical cure did not show convincing improvements after adjustment. The results support the clinical process value of rapid diagnostics while challenging the assumption that diagnostic speed alone can be equated with survival benefit.

Our timing estimates are larger than some previously reported advantages, although direct comparison is difficult because earlier studies differ in assay type, resistance prevalence, inclusion criteria

and time origin. In a 2015 randomised trial, multiplex PCR together with stewardship facilitated more rapid antimicrobial modification than conventional methods, but the effect varied according to the drugs and organisms involved [22]. The multicentre RAPIDS-GN trial likewise demonstrated faster antibiotic changes and earlier escalation in resistant Gram-negative bloodstream infections without detecting a significant improvement in patient-centred outcomes [23]. MacVane and Nolte observed shorter time to effective therapy after adding a blood-culture identification panel to stewardship, also without clear differences in mortality or postculture length of stay [27]. Studies combining rapid identification with stewardship across varying settings similarly emphasise that the interval between test result and clinician response is a critical determinant of success [28–33].

Evidence syntheses provide a broader perspective. Timbrook and colleagues reported both a reduction in time to effective therapy and an association with lower mortality, especially when rapid molecular testing was accompanied by stewardship activities [24]. The 2024 network meta-analysis by Peri and colleagues likewise concluded that the combination of rapid diagnostics and antimicrobial stewardship may improve survival compared with conventional blood culture, while documenting substantial heterogeneity and reliance on quasi-experimental cohorts [25]. Neither finding mandates a survival signal in every individual hospital dataset. Differences in pre-test probability, spectrum of pathogens, resistance prevalence, access to active empiric drugs, staffing, severity of illness and clinical follow-through can all alter the absolute potential for benefit. Economic models additionally favour coupling microbiological innovations with stewardship rather than deploying testing in isolation [26].

An important feature of this cohort is that almost one-third of patients already had active therapy at the index assessment. Their time to effective treatment was necessarily zero regardless of downstream laboratory performance. In contrast, the initially ineffective subgroup isolated the clinical problem that rapid testing is intended to solve: discovering an actionable pathogen or resistance determinant quickly enough to redirect therapy. The strikingly non-overlapping treatment times also require scepticism. No initially ineffective conventional patient was recorded as receiving an active agent within the first 24 hours, whereas almost 90% of comparable rapid-pathway patients did. Such near-deterministic separation may reflect a genuine diagnostic workflow, but may also partly reflect synthetic or protocol-driven timing definitions, site-selection patterns, incomplete recording of clinician-initiated empirical changes, or other upstream factors. It should therefore be validated against source timestamps and medication-administration records before strong effectiveness claims are made.

The clinical-outcome results reinforce the need for risk adjustment and careful causal interpretation. Rapid-pathway recipients had somewhat greater acute severity, with a higher mean SOFA score and more frequent intensive-care exposure at the index assessment. The crude difference in 30-day mortality pointed towards higher mortality in the rapid group rather than lower mortality; adjustment reduced precision but did not convert that result into evidence of benefit. These are not contradictory findings. Even substantial reductions in treatment delays can coexist with similar mortality if outcomes are driven principally by baseline physiology, host reserve, source control, pathogen virulence or effective supportive management. Moreover, the cohort contains only 46 deaths within 30 days, leaving wide confidence intervals and insufficient precision to exclude clinically important harm or benefit. Prior reports about timing and mortality

[5–14] should therefore be regarded as motivation for better-designed testing evaluations rather than justification for declaring a causal survival advantage in the present data.

Rapid test read-outs also require clinical translation. A resistance-marker signal may be incomplete, discordant with phenotypic susceptibility or ambiguous when multiple organisms are present. Conventional culture, identification and susceptibility testing remain indispensable for confirmation and for determining definitive therapy; molecular results should be interpreted in context, alongside local epidemiology and current resistance-treatment guidance [3, 17–21]. Stewardship teams can assist with drug selection, optimisation, avoidance of unnecessary broad-spectrum exposure and safe de-escalation. In parallel, reducing antibiotic exposure can diminish undesirable collateral effects, including *Clostridioides difficile* infection, although these outcomes were not measured in this cohort [41–43]. The data consequently justify improvement initiatives targeting both result turnaround and the timeliness and correctness of subsequent prescribing decisions.

A practical implication is that hospitals should define performance metrics extending beyond the analytic sensitivity or intrinsic laboratory turnaround time of a molecular panel. Relevant measures include specimen-collection-to-result intervals, the proportion of results communicated while patients remain eligible for treatment, time between result release and clinician acknowledgement, the fraction of prescribing recommendations implemented, and the accuracy of treatment selection after those recommendations. Surveillance should separately assess patients already adequately covered at suspicion and those for whom available regimens offer inadequate coverage. These two populations differ fundamentally in what faster testing can accomplish. A balanced implementation assessment should also consider whether results prompt unnecessarily broad escalation, whether negative resistance-marker findings are overinterpreted, and whether access to infectious-diseases pharmacists and clinicians is maintained outside normal working hours. Data on downstream toxicity, subsequent resistance, antimicrobial consumption and economic performance would complement the outcomes available here. Such metrics would clarify whether a health system is merely producing faster tests or actually delivering safer and more effective care.

Several limitations are substantive. First, pathway selection was non-randomised and influenced by clinical choice and test availability, and adjustment cannot rule out confounding by indication or unmeasured hospital-level practice. Second, the dataset does not report test brand, specimen type for each episode, susceptibility interpretation, exact administered drugs, prior antibiotic exposure, source control, treatment escalation, treatment changes after laboratory results or antibiotic-related adverse events. Third, the extract does not include detailed exposure-assignment records or hospital-level workflow documentation, limiting assessment of selection mechanisms. Fourth, the cure outcome is defined at hospital exit and therefore includes a built-in disadvantage for inpatient decedents, while mortality covers 30 days including post-discharge deaths. Fifth, among initially ineffective patients, three did not receive an active drug before a competing exit event. Analysing only recipients can introduce conditioning bias, and ordinary Kaplan–Meier censoring would not necessarily be appropriate for death or discharge [39].

A further limitation is statistical identifiability: when no conventional-pathway patient reaches the 24-hour active-treatment endpoint, conventional logistic regression is separated, and an extremely large fitted coefficient would falsely suggest

precision. We therefore reported observed risks, Fisher testing and more interpretable continuous-time contrasts rather than a spurious adjusted odds ratio. Hospital-level uncertainty is also difficult to quantify robustly with only eight clusters, while the conditional log-time model cannot itself establish causality or a competing-risk-adjusted treatment-incidence effect. In an ideal follow-up analysis, investigators would prespecify the treatment-decision window; audit exact medication and result timestamps; report cumulative incidence under death/discharge as competing events; and use prespecified balancing or trial-emulation methods to address nonrandom test selection [38–40]. A larger prospectively assembled multicentre study would also support resistance-specific effects and more precise survival estimates.

5. Conclusion

In adults with documented resistant bacterial infections, rapid molecular diagnostic pathways were associated with major reductions in time to actionable microbiological information, stewardship review and the initiation of effective antibiotics when initial empirical treatment was inactive. The magnitude of the timing advantage was clear in this dataset but requires source-record confirmation because of unusual between-pathway separation. No significant improvement in mortality, clinical cure or hospital stay was demonstrated. Rapid diagnostics should be understood as an instrument for earlier evidence-informed decisions, not a substitute for high-quality stewardship, clinical assessment and definitive susceptibility testing.

6. Declarations

Ethics approval and consent: The study was approved by the hospital ethics committee (approval reference: HR/12-13/FE/2-1-0). Participant consent was obtained. The analysis used de-identified patient records.

Site confidentiality: Eight participating hospitals are shown only as H1–H8.

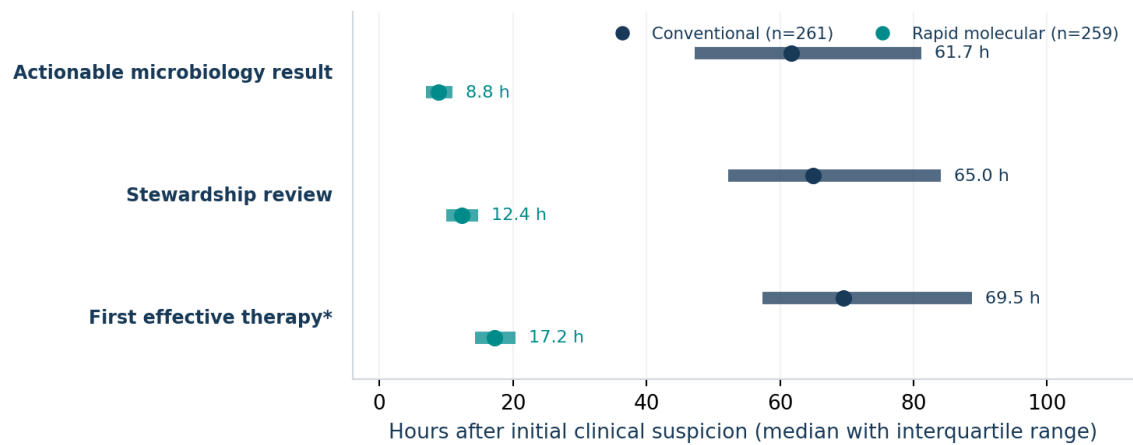
Data availability: The study data are not available.

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7. References

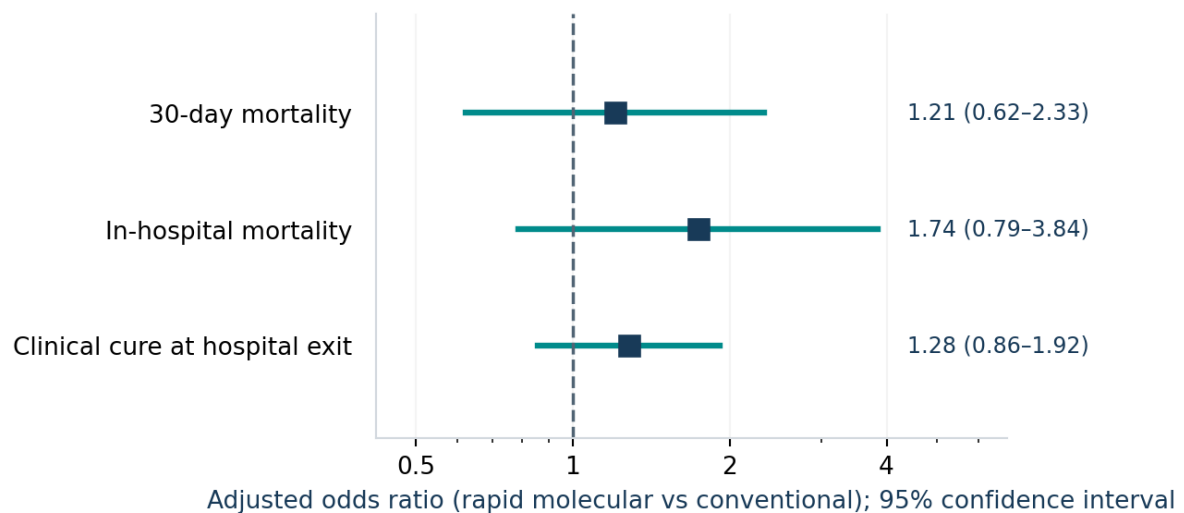
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Figure 1. Diagnostic and treatment timing by diagnostic pathway

*Effective-therapy timing among patients without active empirical therapy who subsequently received active treatment (n=355).

Dots show medians; thick lines indicate interquartile ranges. The first two comparisons include the whole cohort; the last includes initially ineffective empirical therapy followed by a documented effective-treatment event.

Figure 2. Adjusted association of rapid testing with clinical outcomes

Adjusted for age, sex, Charlson index, ICU/SOFA, initial effective empirical therapy, infection site, resistance phenotype, and hospital.

Squares represent multivariable adjusted odds ratios and whiskers their 95% confidence intervals. All confidence intervals include the null (OR=1); the figure does not support causal interpretation.

Table 1. Baseline clinical characteristics and infection sites

Characteristic	Conventional (n=261)	Rapid molecular (n=259)
Age, years, mean (SD)	53.6 (20.8)	53.5 (21.0)
Male sex, n (%)	142 (54.4)	117 (45.2)
Charlson comorbidity index, median (IQR)	1 (0–2)	1 (0–2)
ICU at index, n (%)	65 (24.9)	83 (32.0)
SOFA, median (IQR)	3 (1–5)	4 (2–6)
Effective empirical antibiotics at index, n (%)	84 (32.2)	78 (30.1)
Bloodstream infection, n (%)	49 (18.8)	58 (22.4)
Pneumonia, n (%)	70 (26.8)	63 (24.3)
Urinary tract infection, n (%)	74 (28.4)	62 (23.9)
Intra-abdominal infection, n (%)	68 (26.1)	76 (29.3)

ICU, intensive care unit; SOFA, Sequential Organ Failure Assessment; IQR, interquartile range. Percentages use full exposure-group denominators.

Table 2. Organisms and coded resistance phenotypes

Organism / resistance phenotype	Conventional n (%)	Rapid molecular n (%)
Escherichia coli	43 (16.5)	33 (12.7)
Klebsiella pneumoniae	40 (15.3)	48 (18.5)
Acinetobacter baumannii	34 (13.0)	39 (15.1)
Pseudomonas aeruginosa	51 (19.5)	46 (17.8)
Staphylococcus aureus	47 (18.0)	31 (12.0)
Enterococcus faecium	46 (17.6)	62 (23.9)
RESISTANCE PHENOTYPE		
ESBL-producing	39 (14.9)	46 (17.8)
Carbapenem-resistant	95 (36.4)	81 (31.3)
MRSA	47 (18.0)	31 (12.0)
VRE	46 (17.6)	62 (23.9)
Carbapenem-resistant A. baumannii	34 (13.0)	39 (15.1)

Resistance categories were supplied in the dataset, not independently verified through susceptibility reports; organism and phenotype classifications represent distinct distributions.

Table 3. Diagnostic workflow and effective antimicrobial treatment

Process outcome	Conventional (n=261)	Rapid molecular (n=259)
Index to specimen, h; median (IQR)	1.3 (0.9–2.3)	1.4 (1.0–2.4)
Index to actionable result, h; median (IQR)	61.7 (47.2–81.2)	8.8 (7.0–10.9)
Index to stewardship review, h; median (IQR)	65.0 (52.3–84.1)	12.4 (10.0–14.8)
Effective empirical therapy present, n (%)	84 (32.2)	78 (30.1)
Active antibiotics by 24 h, all patients, n (%)	84 (32.2)	240 (92.7)
INITIALLY INEFFECTIVE EMPIRICAL THERAPY		
No initial active empirical agent, n	177	181
Ever received active antibiotic before exit, n (%)	174 (98.3)	181 (100)
Time to first active treatment among recipients, h; median (IQR)	69.5 (57.4–88.7)	17.2 (14.3–20.4)
First active antibiotic within 24 h, n (%)	0 (0.0)	162 (89.5)

The originally ineffective empirical-therapy subgroup comprised 358 patients; the time-to-treatment medians include only recipients (174 conventional; 181 rapid). Differences in actionable-result and stewardship-review times were $P < .001$; no adjusted odds ratio was estimable for treatment within 24 hours in the subgroup due to complete separation.

Table 4. Patient-centred clinical outcomes and multivariable associations

Clinical endpoint	Conventional	Rapid molecular	Adjusted effect (95% CI)
30-day all-cause death, n (%)	19/261 (7.3)	27/259 (10.4)	aOR 1.21 (0.62–2.33); $P = .577$
In-hospital death, n (%)	12/261 (4.6)	23/259 (8.9)	aOR 1.74 (0.79–3.84); $P = .171$
Clinical cure at hospital exit, n (%)	181/261 (69.3)	187/259 (72.2)	aOR 1.28 (0.86–1.92); $P = .229$
Stay after suspicion, median days (IQR)	14.0 (8.9–20.7)	13.6 (7.7–21.7)	aTR 0.94 (0.83–1.06); $P = .298$

aOR, adjusted odds ratio; aTR, adjusted geometric mean time ratio for log-transformed stay. All estimates compare rapid testing with conventional testing, adjusted for age, sex, comorbidity, ICU/SOFA, initially active therapy, infection site, resistance phenotype and hospital.