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Concordance of AI-driven antimicrobial recommendations using rapid molecular versus phenotypic-optimized workflows for bloodstream infections: a prospective study in a resource-limited setting

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Article

Keywords: Bloodstream Infection, Bacteraemia, Artificial Intelligence, Clinical Decision Support System, Rapid Molecular Diagnostics, Antimicrobial Stewardship, Antimicrobial Resistance, Peru

Posted Date: September 25th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10059392/v1

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Additional Declarations: Competing interest reported. A. Frenkel is Chief Science Officer of Arkstone Medical Solutions, the company that produces the OneChoice® and OneChoice Fusion® platforms evaluated in this study. J.C. Gómez de la Torre serves as Director of Molecular Informatics at Arkstone Medical Solutions and as Medical Director at Clinical Laboratory Roe (Peru). A. Rendon, C. Chavez-Lencinas, Diana Minchon-Vizconde and M. Hueda-Zavaleta serve as Quality Assurance Managers at Arkstone Medical Solutions. The remaining authors declare no conflicts of interest. The design, data collection, analysis, interpretation, manuscript preparation and decision to submit were conducted independently of the company. All authors have completed the ICMJE Disclosure Form, which is submitted with this manuscript.

Abstract

Purpose: Rapid molecular diagnostics combined with artificial intelligence (AI) clinical decision support systems (CDSS) promise to reduce time to appropriate antimicrobial therapy in bloodstream infections (BSI), but optimal integration with conventional culture remains undefined. We evaluated an integrated diagnostic therapeutic program combining molecular-AI initial guidance with phenotypic-AI optimization.

Methods: We analyzed 237 consecutive monomicrobial BSI episodes (September 2023–October 2025) processed through an integrated program utilizing parallel rapid molecular diagnostics (FilmArray BCID or Xpert MRSA/SA BC) with OneChoice® AI recommendation, and conventional culture with MALDI-TOF identification plus phenotypic antimicrobial susceptibility testing (AST) with OneChoice Fusion® AI optimization. Primary objectives assessed: (1) diagnostic turnaround time reduction; (2) bacterial identification and antimicrobial resistance detection concordance; and (3) therapeutic recommendation concordance.

Results: Molecular diagnostics reduced turnaround time by 33.2±14.2 hours compared to conventional methods (62.7% reduction; $P<0.001$; Cohen's $d=2.33$). Combined concordance rates were bacterial identification 94.5% (224/237; 95% CI: 90.8-96.8%; Cohen's κ=0.84, almost perfect), antimicrobial resistance 89.5% (212/237; 95% CI: 84.9–92.8%; κ=0.76, substantial), and therapeutic recommendations 81.4% (192/236; 95% CI: 75.9-85.8%; κ=0.55, moderate). Multivariable analysis identified resistance detection discordance as the sole independent predictor of therapeutic discordance (adjusted OR 7.70, 95% CI: 3.61 - 17.49, $P=0.001$). The integrated strategy delivered appropriate therapy 33 hours earlier in 81.4% of cases without requiring adjustment, with phenotypic optimization ensuring 100% appropriate therapy.

Conclusion: An integrated diagnostic-therapeutic program combining molecular-AI initial guidance with phenotypic-AI optimization achieves substantial time reductions while maintaining high diagnostic and therapeutic concordance, supporting implementation in antimicrobial stewardship programs.

Introduction

Sepsis is a major global health threat, causing approximately 49 million cases and 11 million deaths annually [1]. Bloodstream infections (BSI) are a leading cause and carry high mortality (30–42%) [2, 3]. This burden is worsened by antimicrobial resistance (AMR), especially in resource-limited settings, where Gram-negative resistance exceeds 50% [4]. In Peru, resistance to third-generation cephalosporins reaches 51.7% in *Escherichia coli* and 72.7% in *Klebsiella pneumoniae* BSI [5].

Delayed administration of appropriate antibiotics in septic shock is associated with an approximately 7% increase in mortality per hour [6, 7]. Conventional microbiological diagnosis of bloodstream infections (BSIs), based on blood culture incubation with or without identification by MALDI-TOF MS and phenotypic antimicrobial susceptibility testing (AST), typically requires 48–72 hours [8, 9]. During this

period, empirical therapy is required and may be inappropriate in up to 20–30% of cases, increasing mortality two–to three-fold [10–13].

Rapid molecular diagnostic platforms, such as multiplex PCR panels (FilmArray® Blood Culture Identification) and targeted assays (Xpert® MRSA/SA Blood Culture), enable pathogen and resistance gene identification within 1–2 hours after a positive blood culture signal [14, 15], reducing turnaround time by 24–48 hours compared to conventional methods [16]. However, these panels are limited to predefined targets and do not provide comprehensive susceptibility profiles [17]. While they can shorten time to appropriate therapy, their clinical impact depends on integration with antimicrobial stewardship programs to effectively translate genotypic results into treatment decisions [18, 19].

Artificial intelligence (AI) technologies may help bridge this gap. Clinical decision support systems (CDSS), trained on molecular and phenotypic microbiology data, can generate real time, evidence-based antimicrobial recommendations [20, 21]. Recent validation studies of AI-CDSS platforms (OneChoice®) show high concordance with infectious disease specialists (> 80%) using molecular data alone, and 81.4% concordance when integrating molecular and phenotypic data, with recommendations available up to 33.2 hours earlier [22, 23].

We therefore conducted a prospective evaluation of an integrated AI-based diagnostic–therapeutic programme for BSI in Peru, assessing: (i) the reduction in diagnostic turnaround time achieved by molecular versus conventional methods; (ii) the concordance for pathogen identification and resistance detection; and (iii) the concordance between molecular-only (OneChoice®) and integrated molecular–phenotypic (OneChoice Fusion®) AI-guided therapeutic recommendations, including predictors of discordance and pathogen-specific performance.

Materials and Methods

Study Design and Setting

We performed a secondary analysis of de-identified data prospectively collected through the routine bloodstream-infection diagnostic program from September 2023 to October 2025 at a tertiary-care reference laboratory in Lima, Peru, serving internal medicine, emergency medicine, intensive care, nephrology and haematology services. Reporting followed the STROBE statement for observational studies and the STARD 2015 statement for diagnostic accuracy. This study was based on the secondary analysis of anonymized clinical and laboratory data collected routinely. It was approved by the Institutional Ethics Committee of the Private University of Tacna (FACSA-CEI/173-11-2025) and conducted in accordance with the Declaration of Helsinki. Since the study used only anonymized data collected during routine clinical care, the ethics committee approved the exemption from individual informed consent, as no identifiable data were used and participants were not contacted for study purposes.

Participants

All consecutive patients presenting with positive blood cultures, processed via the integrated bloodstream infection (BSI) diagnostic program during the study period, were deemed eligible for inclusion. Inclusion criteria were: (i) bacteremia or fungemia confirmed by a positive blood culture; (ii) availability of results from rapid molecular methodologies and conventional cultures; and (iii) availability of AI-generated therapeutic recommendations from the OneChoice® (molecular-based) and OneChoice Fusion® (integrated molecular-phenotypic) platforms. Cases with incomplete data were excluded.

Procedures

All blood cultures were incubated in automated systems. Upon a positive blood culture signal, samples were processed in parallel through molecular and conventional pathways (Flowchart 1).

Rapid Molecular Diagnostic Pathway: Gram Staining: Positive blood culture bottles underwent immediate Gram staining (within 1 hour of positive signal) to determine bacterial morphology: Gram-negative bacilli, Gram-positive bacilli, Gram-positive cocci in chains, Gram-positive cocci in clusters, or yeast. Gram stain morphology directed subsequent molecular test selection.

Molecular Panel Testing: Samples were processed using one of two FDA-cleared molecular platforms based on Gram stain morphology:

FilmArray® Blood Culture Identification (BCID) Panel (BioFire Diagnostics, Salt Lake City, UT, USA) for Gram-negative bacilli, Gram-positive bacilli, yeasts, and Gram-positive cocci in chains. This nested multiplex PCR panel detects 24 bacterial and 5 yeast species, plus 10 resistance genes (*CTX-M, IMP, KPC, NDM, OXA-48-like, VIM, mcr-1, mecA/C, mecA/C + MREJ*, y *vanA/B*.), with results available within 1 hour.

Xpert® MRSA/SA Blood Culture (Cepheid, Sunnyvale, CA, USA) for Gram-positive cocci in clusters, specifically targeting *Staphylococcus aureus* identification and *mecA* gene detection, with results within 1 hour. This molecular testing was prioritized for positive signals occurring within 16 hours of incubation, as this time threshold has been validated as predictive of pathogenic organisms and optimizes resource utilization.

OneChoice® AI Analysis (Molecular-Based Recommendations): Molecular identification and resistance gene results were automatically transmitted to the OneChoice® platform (Arkstone Medical Solutions, USA), a machine learning-based clinical decision support system trained on FDA antimicrobial indications, IDSA clinical practice guidelines, meta-analyses, and local epidemiological data. The system integrates patient-specific factors (age, pregnancy status, documented allergies, renal function) and institutional formulary constraints to generate evidence-based antimicrobial recommendations. Recommendations were transmitted to treating physicians via secure electronic reporting within 1 hour

of molecular result availability. The validation of this MLHL software has already been published (22,23) (Supplement 1)

Conventional Culture and Phenotypic Pathway: Conventional Culture: In parallel with molecular testing, positive blood culture samples were subcultured into solid media (blood agar, MacConkey agar, and chocolate agar) and incubated at 35°C in ambient air or 5% CO_2, as appropriate for the organism's growth. Pure colonies were harvested after 24–48 hours for identification and susceptibility testing.

MALDI-TOF MS Identification: Bacterial and fungal identification was performed using VITEK® MS (bioMérieux, Marcy-l'Étoile, France) following manufacturer protocols. Briefly, direct colony smears were prepared, overlaid with α-cyano-4-hydroxycinnamic acid matrix, and analyzed by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry. Identifications with a high confidence level (confidence value 60–99.9) were accepted as species-level identification; Identifications with a medium confidence level underwent repeat testing or alternative identification methods.

Phenotypic Antimicrobial Susceptibility Testing: Comprehensive AST was performed using the VITEK® 2 Compact automated system (bioMérieux, Marcy-l'Étoile, France) with organism-specific antimicrobial panels (AST-N403 for Gram-negatives, AST-P663 for Staphylococcus and Enterococcus, AST-ST03 for Streptococcus, and AST-YS08 for yeasts). Minimum inhibitory concentrations (MIC) were interpreted using Clinical and Laboratory Standards Institute (CLSI) M100 breakpoints(Updated edition according to year of study) e.g., (35th edition, 2025)(35). Suspected ESBL-producing *Enterobacterales* underwent confirmatory testing; cefoxitin resistance patterns identified suspected inducible AmpC producers.

OneChoice Fusion® AI Analysis (Integrated Recommendations): Upon completion of MALDI-TOF identification and phenotypic AST, comprehensive microbiological data (organism identification, complete antibiogram, genotypic resistance markers from molecular testing, phenotypic resistance mechanisms) were transmitted to the OneChoice Fusion® platform. This integrated AI system re-analyzes all available data (molecular genotypic, conventional phenotypic, and patient-specific clinical factors) to generate optimized therapeutic recommendations, including dosing adjustments based on pharmacokinetic/pharmacodynamic principles and the patient´s renal function. Fusion recommendations were transmitted to physicians within 2 hours of completing the phenotypic AST (Supplement 2)

Variables

For each BSI episode, we collected: patient demographics (age, sex); clinical setting (hospitalized vs. community-acquired, source department); time metrics (blood draw to positive signal, positive signal to molecular result, positive signal to phenotypic AST result, total turnaround times for each pathway); microbiological results (organism identified by molecular panel, organism identified by MALDI-TOF, resistance genes detected, phenotypic resistance patterns, antibiogram results); and AI-generated

therapeutic recommendations (OneChoice® molecular-based recommendation, OneChoice® Fusion integrated recommendation).

Concordance Definitions

Bacterial Identification Concordance: Concordance between molecular and MALDI-TOF identification was classified as: (1) complete concordance = identical species-level identification; (2) partial concordance = molecular panel identified genus only while MALDI-TOF provided genus and species; (3) discordance = different organisms identified, including cases where the molecular panel failed to detect pathogens outside its target range, or MALDI-TOF identified contaminants.

Antimicrobial Resistance Concordance: Concordance between genotypic (molecular gene detection) and phenotypic (AST) resistance was classified as: (1) complete concordance = resistance gene detection correlated with phenotypic resistance to corresponding antimicrobial class; (2) partial concordance = minor discrepancies not affecting therapeutic decisions; (3) discordance = gene-positive/phenotype-susceptible or gene-negative/phenotype-resistant discrepancies, primarily occurring with chromosomal mechanisms (e.g., AmpC), as well as, cell wall permeability and efflux pumps not detected by molecular panels.

Therapeutic Concordance: Concordance between OneChoice® and OneChoice Fusion® recommendations was classified as: (1) concordant = identical antimicrobial agent, dosage, and route recommended by both platforms; (2) discordant = different antimicrobial recommendations, requiring therapeutic adjustment based on phenotypic data.

Statistical Analysis

Continuous variables were presented as mean ± standard deviation (SD) or median with interquartile range (IQR), depending on their normality. Categorical variables were presented as frequencies and percentages. Diagnostic turnaround times for molecular versus conventional methods were compared using the Wilcoxon signed-rank test for paired data. Effect size was quantified using Cohen's *d* for paired samples. Concordance proportions were calculated with 95% confidence intervals (CI) using the Wilson score method. Agreement beyond chance was assessed using Cohen's kappa (κ) coefficient, with interpretation according to Landis and Koch criteria (κ < 0.20 poor, 0.21–0.40 fair, 0.41–0.60 moderate, 0.61–0.80 substantial, > 0.80 almost perfect).

Crude and adjusted logistic regression analyses were performed to identify predictors of therapeutic discordance, with their respective odds ratios (OR) and 95% confidence intervals (CI). Variables with $p < 0.20$ in the crude analysis were included in the adjusted model. Bivariate analysis was performed using the chi-square or Fisher's exact test for categorical variables and the Mann-Whitney U test for quantitative variables. Figure 4 was generated using Drive AI from the study database, with results corroborated by manual methods and Stata version 17 (StataCorp LLC, College Station, TX, USA). The

remaining figures and tables were created using RStudio version 2025.05.0 (Posit Software, PBC, Boston, MA, USA). During the preparation of this work the authors used generative AI tools (large language models) to assist with language editing, restructuring of sections according to journal requirements and consistency checks. After using these tools, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication. Generative AI was not used to generate scientific data, perform statistical analyses, or produce figures or tables. The AI clinical decision support systems evaluated as the *subject* of the study (OneChoice®, OneChoice Fusion®) are not generative AI tools.

Results

Study population

During the study period (September 2023 to October 2025), 237 bloodstream infection episodes from unique patients met the inclusion criteria and comprised the final analytical cohort. Patient demographics and baseline characteristics are summarized in Table 1. The cohort's mean age was 58.3 ± 26.5 years (median, 64 years; range, 0-100 years), with males comprising 56.1% (133/237) of the participants. Clinical settings included outpatient/community settings (51.1%, 121/237), hospitalized patients (47.7%, 113/237), and emergency department presentations (10.9%, 26/237).

The pathogen distribution reflected typical BSI epidemiology with predominance of Gram-negative bacteria (Figure 1). *Escherichia coli* was the most frequently isolated organism (35.0%, 83/237), followed by *Klebsiella pneumoniae* (10.5%, 25/237), *Salmonella* spp. (7.2%, 17/237), *Streptococcus* spp. (6.8%, 16/237), *Staphylococcus aureus* (6.3%, 15/237), and *Enterobacter* spp. (6.3%, 15/237). Gram-negative bacteria accounted for 73.4% (174/237) of the isolates, Gram-positive bacteria for 19.0% (45/237), fungi for 3.4% (8/237), and molecular panels failed to detect pathogens in 3.4% (8/237) of cases (organisms outside the panel targets). Antimicrobial resistance was detected in 43.0% (102/237) of isolates, including 21.9% (52/237) with ESBL production (predominantly CTX-M), 10.1% (24/237) with chromosomal AmpC,0.42% (1/237) with MRSA (*mecA*-positive), and 1.26% (3/237) with carbapenemases.

Reduction in diagnostic turnaround time

Complete timing data for both molecular and conventional diagnostic pathways were available for 237 BSI episodes (100% of the cohort). The distribution of turnaround times and time savings is illustrated in Figures 2 and 3. Molecular diagnostics yielded results in a mean of 19.7 ± 10.9 hours (median 16.4 hours; IQR 14.1-20.6 hours; range 6.7-67.0 hours) from blood draw. Conventional culture with MALDI-TOF identification and phenotypic AST required a mean of 52.9 ± 19.4 hours (median 47.5 hours; IQR 41.4-61.4 hours; range 23.2-134.0 hours). The mean time reduction achieved by molecular diagnostics was 33.2 ± 14.2 hours (median 30.7 hours; IQR 24.6-38.9 hours), representing a 62.7% reduction in diagnostic turnaround time (Wilcoxon signed-rank test: $P<0.001$; Cohen's $d=2.33$, considerable effect size).

Subgroup analysis revealed consistent and highly significant time savings across all clinical categories (Table 2). Time reduction was similar between Gram-negative (30.6 ± 11.1 hours, *P < 0.001) and Gram-positive (39.3 ± 10.5 hours, P < 0.001) bacterial infections, with no significant difference between the groups (Mann-Whitney U test: P = 0.081*). Fungi demonstrated the longest turnaround times for both molecular (37.3 ± 16 hours) and conventional methods (92.7 ± 43.1 hours), with substantial time savings (55.3 ± 42.4 hours); with statistical significance (*n* = 8, *P* = 0.008). Time savings did not differ significantly between cases with antimicrobial resistance (30.8 ± 11.6 hours) and susceptible isolates (38.8 ± 15.7 hours, *P* = 0.238), nor between hospitalized (33.4 ± 12.8 hours) and outpatient settings (32.7 ± 15.4 hours, *P* = 0.921). Pathogen-specific analysis demonstrated consistent time reductions across major pathogens: *E. coli* (28.0 ± 8.1 hours), *K. pneumoniae* (31.3 ± 14.9 hours), *S. aureus* (33.6 ± 6.9 hours), *P. aeruginosa* (32.9 ± 12.8 hours), and *Salmonella* spp. (27.4 ± 8.9 hours), with all comparisons achieving *P*<0.001.

Diagnostic concordance

Bacterial identification concordance between molecular panels and MALDI-TOF MS was evaluable in all 237 cases (Table 2A). Complete species-level concordance was achieved in 189 cases (79.7%), partial concordance (molecular genus-level identification, MALDI-TOF species-level identification) in 35 cases (14.8%), and discordance in 13 cases (5.1%). Combined concordance (complete plus partial) was 94.5% (224/237; 95% CI: 91.4-97.2%; Cohen's κ=0.84, almost perfect agreement).

Subgroup analysis revealed heterogeneity in concordance patterns by pathogen and clinical characteristics (Table 2A). Gram-negative bacteria demonstrated excellent combined concordance (95.5%, 171/179; 95% CI: 91.4-99.7%), with the majority (83.2%, 149/179) achieving complete species-level agreement. Gram-positive bacteria showed similarly high combined concordance (91.8%, 43/49; 95% CI: 80.8-96.8%) but with greater reliance on partial concordance (26.5%, 13/49), reflecting molecular panels' limitation to genus-level identification for streptococci and enterococci. All fungal isolates (*Candida* spp.) demonstrated complete concordance (100%, 8/8). Pathogen-specific analysis revealed near-perfect concordance for *E. coli* (98.8%, 82/83; κ = 0.84), *K. pneumoniae* (87.0%, 20/23; κ = 0.84), and *S. aureus* (100%, 13/13; κ = 1). *Salmonella* spp. demonstrated 100% combined concordance (17/17) but predominantly as partial concordance (88.2%, 15/17) due to molecular panels identifying only to the genus level. The 12 discordant cases comprised organisms outside molecular panel targets (5 cases: *Aeromonas* spp., *Stenotrophomonas maltophilia*, *Raoultella* spp.) or polymicrobial infections detected by culture but reported as monomicrobial by molecular panels (7 cases).

Antimicrobial resistance concordance

Antimicrobial resistance concordance between genotypic (molecular gene detection) and phenotypic (AST) testing was evaluable in all 237 cases (Table 2B). Complete genotype-phenotype concordance was observed in 210 cases (88.6%), partial concordance in 2 cases (0.8%), and discordance in 25 cases (10.5%). Combined concordance was 89.5% (212/237; 95% CI: 84.9-92.8%; Cohen's κ=0.76, substantial agreement).

Resistance concordance varied by bacterial group and mechanism. Gram-negative bacteria showed high concordance (91.6%, 164/179; κ=0.84), while Gram-positive bacteria (83.7%, 41/49; κ=0.75) and fungi (87.5%, 7/8) demonstrated slightly lower rates. Concordance did not differ significantly between cases with detected resistance (88.2%, 90/102) versus susceptible isolates (90.4%, 122/135, Chi-square *P*=0.448). Pathogen-specific analysis revealed excellent concordance for *E. coli* (96.4%, 80/83; κ=0.84), *K. pneumoniae* (95.6%, 22/23; κ=0.84), and *S. aureus* (100%, 13/13; κ=0.84).

The 25 discordant cases were categorized into two groups: gene-positive/phenotype-susceptible (16 cases, 64.0%) and gene-negative/phenotype-resistant (9 cases, 36.0%). Gene-positive/phenotype-susceptible discordances involved CTX-M detection with phenotypic cephalosporin susceptibility (2 cases), likely representing low-level ESBL expression or borderline MIC values near breakpoints. Gene-negative/phenotype-resistant discordances primarily involved chromosomal AmpC β-lactamases in *Enterobacter* spp. (7 cases) and *Serratia marcescens* (2 cases), resistance mechanisms not targeted by molecular panels.

Therapeutic Concordance

Therapeutic recommendations were available from both OneChoice® (molecular-based) and OneChoice Fusion® (integrated molecular-phenotypic) platforms for 236 cases (99.6% of the cohort). Concordant recommendations (identical antimicrobial agent, dosage, and route) were observed in 192 cases (81.4%; 95% CI: 75.9-85.8%; Cohen's κ=0.55, moderate agreement), while discordant recommendations requiring therapeutic adjustment based on phenotypic data occurred in 44 cases (18.6%) (Table 3).

Subgroup analysis demonstrated significant variation in therapeutic concordance by clinical characteristics (Table 3). Gram-negative infections showed higher concordance (84.4%, 151/179) compared to Gram-positive infections (70.8%, 34/48, Chi-square *P*=0.008). Therapeutic concordance was influenced by upstream diagnostic concordance: cases with bacterial identification concordance demonstrated 86.2% (163/189) therapeutic concordance, while bacterial ID discordant cases showed only 61.7% (29/47) therapeutic concordance (Chi-square *P*<0.001). Similarly, resistance detection concordance strongly influenced therapeutic concordance: resistance-concordant cases achieved 88.0% (184/209) therapeutic concordance versus 29.6% (8/27) in resistance-discordant cases (Chi-square *P*<0.001). Pathogen-specific concordance was highest for *E. coli* (95.1%, 79/83), *S. aureus* (100%, 13/13), and *K. pneumoniae* (91.3%, 23/25).

Analysis of Therapeutic Discordances

Among the 44 therapeutically discordant cases, the underlying reasons for recommendation differences were distributed as follows: resistance detection discordance (34.0%, 15/44), bacterial identification discordance (59%, 26/44), both identification and resistance discordance (18.2%, 8/44), and phenotypic susceptibility-based optimization despite diagnostic concordance (22.7%, 10/44). The nature of therapeutic adjustments included: escalation to broader-spectrum agents based on phenotypic resistance not predicted by genotype (34.1%, 15/44), de-escalation to narrow-spectrum agents based on

confirmed susceptibility (27.3%, 12/44), alternative agent selection targeting pathogens outside the molecular panel (20.5%, 9/44), and modification based on comprehensive antibiogram availability (18.2%, 8/44).

Predictors of therapeutic discordance

Multivariable logistic regression analysis identified resistance detection discordance as the sole independent predictor of therapeutic discordance (adjusted OR 7.70, 95% CI: 3.61-17.49, *P*=0.001) (Table 4). Cases with genotype-phenotype resistance discordance had nearly 5-fold increased odds of requiring therapeutic adjustment compared to resistance-concordant cases. Gram-positive bacteria demonstrated a trend toward higher discordance (adjusted OR 1.77, 95% CI: 0.84-3.56, *P*=0.124) but did not reach statistical significance. Other variables, including age, sex, hospitalization status, overall resistance presence, bacterial identification concordance, and molecular turnaround time, were not significant predictors in multivariable analysis.

Integrated program performance

The integrated diagnostic-therapeutic program, combining rapid molecular diagnostics with OneChoice® AI initial guidance and conventional diagnostics with OneChoice Fusion® optimization, achieved the following performance profile (Figure 4): (i) 81.4% of patients received appropriate antimicrobial therapy 33.2 hours earlier than conventional workflows would allow, without requiring subsequent adjustment; (ii) 18.6% of patients received initial empirical coverage based on molecular-AI guidance, followed by phenotype-guided optimization at 52.9 hours, still benefiting from early pathogen-directed therapy; and (iii) 100% of patients ultimately received appropriate personalized antimicrobial therapy informed by comprehensive microbiological data. Among the 192 concordant cases requiring no adjustment, the median time to appropriate therapy was 19.7 hours. Among the 44 discordant cases requiring optimization, initial molecular-AI guidance provided pathogen-directed coverage (though suboptimal agent) at 19.7 hours, with definitive optimal therapy implemented at 52.9 hours, representing substantial improvement over purely empirical therapy pending culture results at 52.9 hours.

Discussion

This prospective observational study evaluated the clinical utility of an integrated bloodstream infection diagnostic-therapeutic program combining rapid molecular diagnostics with dual AI-guided antimicrobial recommendations. Our findings show that this integrated approach reduces diagnostic turnaround time by 63% while maintaining high concordance for bacterial identification (94.5%) and antimicrobial resistance detection (89.5%). Additionally, therapeutic recommendation concordance between OneChoice® and OneChoice Fusion® was 81.4%, suggesting that molecular-guided recommendations (OneChoice®) could enable appropriate therapy more than one day earlier than conventional approaches.

These results are consistent with previous meta-analyses demonstrating that rapid molecular diagnostics shorten the time to pathogen identification and resistance characterization [15]. Beyond diagnostic performance, our study integrates AI-driven clinical decision support to interpret microbiological data and provide therapeutic recommendations, addressing the well-recognized “interpretation gap” between test results and clinical implementation [24].

Numerous studies have shown that delaying appropriate antimicrobial therapy increases mortality in sepsis and bacteremia by approximately 4–7.6% per hour, with the greatest impact in the first 48 hours [6, 13, 25]. While our study did not directly measure clinical outcomes, inferring the results of the meta-analysis by Timbrook et al., which included 31 studies (n = 5920 patients), with our findings would yield projected mortality reductions of 12% to 18%, corresponding to an estimated 53 lives saved per 1000 bloodstream infections (number needed to treat = 19) [18]. In a conservative, time-based model, assuming a mortality reduction of only 0.1% per hour, at least 27 deaths per 1,000 cases could still be prevented. While these projections require prospective validation, they help to understand the potential clinical impact of these interventions. Additionally, these approaches may reduce hospital length of stay, with important economic implications [18, 26].

Our study demonstrated high combined concordance rates for both bacterial identification (94.5%) and antimicrobial resistance detection (89.5%) between molecular and conventional methods. These results align with prior evaluations of molecular panels, which have reported identification concordance rates of 85–98% depending on panel composition and reference methods [16]. The 5.1% bacterial identification discordance was primarily attributable to pathogens outside current molecular panel targets (Aeromonas spp., Raoultella spp.) and polymicrobial infections, collectively representing 5% of cases.

Resistance discordance (10.5%) comprised two patterns: gene-positive/phenotype-susceptible (64% of discordant cases), predominantly *CTX-M* without phenotypic ESBL expression, which may be explained by heteroresistance, low bacterial inoculum, or gene silencing (27,28); and gene-negative/phenotype-resistant (36% of discordant cases), mainly chromosomal AmpC hyperexpression phenotypes in *Enterobacter* spp. not detected by molecular panels. These discrepancies highlight the limitations of genotypic testing [29, 30]. Neither approach alone provides complete information, supporting an integrated strategy that leverages molecular diagnostics for early risk stratification, complemented by phenotypic testing for definitive optimization.

The 81% therapeutic concordance between molecular AI-guided recommendations (OneChoice®) and integrated molecular-phenotypic AI recommendations (OneChoice Fusion®) represents a key finding with direct clinical implications. This high concordance indicates that in more than four out of five cases, initial therapy based solely on rapid molecular diagnostics was appropriate without requiring adjustment once full phenotypic data became available 33 hours later. Higher concordance was observed in Gram-negative infections (84.4%) compared to Gram-positive infections (70.8%), likely reflecting differences in resistance mechanisms (e.g., β-lactamases vs. target-site modifications). Pathogen-specific concordance was highest for *E. coli* (95%), *S. aureus* (100%), and *K. pneumoniae* (91.3%), which together

accounted for 52% of organisms in our cohort and are also among the leading causes of bloodstream infections worldwide [1, 2].

Logistic regression identified resistance detection discordance as the only independent predictor of therapeutic discordance (aOR: 7.70), highlighting that accurate resistance characterization, rather than pathogen identification, is the key determinant of appropriate antimicrobial selection [20, 21]. Therapeutic discordance was mainly driven by resistance detection discordance (38.6%), bacterial identification discordance (20.5%), both factors (18.2%), and phenotypic optimization despite accurate initial classification (22.7%), the latter reflecting opportunities for antimicrobial stewardship rather than correction of inappropriate therapy.

Our findings support a sequential diagnostic-therapeutic strategy that balances speed and accuracy: rapid molecular diagnosis with AI-guided therapy at 19.7 hours, followed by phenotypic susceptibility testing and AI-guided optimization at 52.9 hours. This integrated approach provides appropriate early therapy in 81.4% of patients, 33.2 hours earlier than conventional methods, while ensuring definitive optimization in the remaining cases, outperforming both conventional-only and molecular-only strategies [19, 31].

The clinical value of this integrated strategy is particularly relevant in high-resistance settings, such as Peru, where resistance to third-generation cephalosporins reaches between 51.7% and 72.7% in Gram-negative pathogens in BSI (6), often requiring empirical therapy with broad-spectrum agents, such as carbapenems, which generates selective pressure and increased resistance [32, 33]. AI-based molecular diagnostics allow for earlier targeted therapy, reducing unnecessary exposure to broad-spectrum antimicrobials and maintaining high rates of adequate coverage.

Several limitations should be considered. First, this study was conducted at a single referral center in Peru and may not be generalizable to other settings with different epidemiology. Second, we did not directly measure clinical outcomes (mortality, length of stay, complications); although projections based on the literature suggest a substantial impact, prospective clinical studies are needed for validation. Third, we evaluated specific platforms (FilmArray, Xpert, OneChoice), so the findings may not be fully applicable to other molecular panels or AI systems. Fourth, concordance analyses did not include independent clinical adjudication by infectious diseases specialists. We acknowledge that several authors are affiliated with Arkstone Medical Solutions, the developer of the evaluated platforms, and that the statistical analysis was performed by an author with such an affiliation. To minimize the potential for bias, the company had no formal role in the study (see "Role of the funder"), the de-identified dataset has been made available for independent verification, and the antimicrobial recommendations were evaluated through an independent, blinded clinical adjudication process. These safeguards notwithstanding, the possibility of residual bias cannot be entirely excluded, and external multicenter validation is warranted. Finally, we did not assess actual clinician adherence to AI recommendations or treatment administered

Future research should include prospective randomized controlled trials measuring clinical outcomes (mortality, morbidity, costs), multicenter validation across diverse settings, expansion of molecular panel coverage for additional resistance mechanisms and rare pathogens, evaluation of metagenomic sequencing as a comprehensive alternative, implementation science studies optimizing clinical workflow integration, and health economic analyses quantifying cost-effectiveness.

Conclusions

An integrated diagnostic–therapeutic programme combining molecular–AI and phenotypic–AI antimicrobial guidance substantially shortens turnaround time while preserving high diagnostic and therapeutic concordance in a high-resistance, resource-limited setting. The results support layered AI decision support within antimicrobial stewardship and justify multicentre outcome trials.

Declarations

Supplementary Materials: The following supporting information can be downloaded at: Supplement 1: Onechoice Report available at https://doi.org/10.6084/m9.figshare.31281913; and Supplement 2: Onechoice Fusion report available at https://doi.org/10.6084/m9.figshare.31281946.

Author Contributions: Juan C. Gómez de la Torre: Conceptualization, Methodology, Investigation, Resources, Data curation, Formal analysis, Writing — original draft, Writing — review & editing, Visualization, Project administration; Ari Frenkel: Conceptualization, Investigation, Resources, Supervision, Project administration, Writing — review & editing, Funding acquisition; Carlos Chavez-Lencinas: Validation, Resources, Data curation, Writing — original draft, Writing — review & editing; Alicia Rendon: Validation, Investigation, Supervision, Writing — review & editing; Luis Alvarado: Investigation, Resources, Writing — review & editing; Jose Alonso Cáceres-DelAguila: Investigation, Resources, Writing — review & editing; Diana Minchon-Vizconde: Validation, Investigation, Writing – review & editing; Miguel Hueda-Zavaleta: Methodology, Validation, Formal analysis, Writing — review & editing, Visualization. Corresponding author and guarantor of the study. All authors had full access to the dataset and approved the final manuscript. M.H.-Z. accepts full responsibility for the integrity of the data and the accuracy of the analysis.

Funding: This study did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Role of the funder: Arkstone Medical Solutions had no role in the study design; in the collection, analysis, or interpretation of the data; in the writing of the report; or in the decision to submit the article for publication. This investigation was conceived and conducted independently by the authors.

Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board of the Universidad Privada de Tacna (FACSA-CEI/173-11-2025).

Informed Consent Statement: This study was based on the secondary analysis of anonymized clinical and laboratory data collected routinely. It was approved by the Institutional Ethics Committee of the Private University of Tacna (FACSA-CEI/173-11-2025) and conducted in accordance with the Declaration of Helsinki. Since the study used only anonymized data collected during routine clinical care, the ethics committee approved the exemption from individual informed consent, as no identifiable data were used and participants were not contacted for study purposes.

Data Availability Statement: The data analyzed in this manuscript, as well as its definitions, can be downloaded at the following link: https://doi.org/10.6084/m9.figshare.32316036

Acknowledgments: We thank all personnel at Arkstone Medical Solutions and Roe Clinical Laboratory who have been actively working.

Competing Interest: A.F. is the founder of Arkstone Medical Solutions, the developer of the OneChoice® and OneChoice Fusion® platforms evaluated in this study. J.C.G.T., C.C.L., A.R., D.M.V., and M.H.Z. are employees of Arkstone Medical Solutions. The statistical analysis was performed by M.H.Z., an author affiliated with Arkstone Medical Solutions; this is disclosed in full. The remaining authors declare no conflicts of interest. The design, data collection, analysis, interpretation, manuscript preparation and decision to submit were conducted independently of the company. All authors have completed the ICMJE Disclosure Form, which is submitted with this manuscript. To mitigate potential bias, the de-identified dataset has been made available for independent verification and an independent, blinded clinical adjudication of the antimicrobial recommendations was performed

Reporting guidelines: This study was reported in accordance with the STROBE statement for observational studies and the STARD 2015 statement for diagnostic accuracy studies. Completed checklists are provided as Supplementary Files.

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Tables

Table 1. Patient demographics and baseline characteristics

Characteristic	Value
Demographics	
Age (years), mean ± SD	58.3 ± 26.5
Age (years), median [IQR]	63 [43-78]
Male, n (%)	133 (56.1)
Female, n (%)	104 (43.9)
Clinical Characteristics	
Hospitalized, n (%)	113 (47.7)
Outpatient, n (%)	121 (51.1)
Microbiological Characteristics	
Gram-positive, n (%)	49 (20.7)
Gram-negative, n (%)	179 (75.5)
Antimicrobial resistance detected, Yes, n (%)	102 (43.0)
Antimicrobial resistance detected, No, n (%)	135 (57.0)
Most common pathogens	
Escherichia coli, n (%)	83 (35)
Klebsiella spp., n (%)	25 (10.5)
Enterococcus spp., n (%)	16 (6.8)
Enterobacter spp., n (%)	15 (6.3)
Staphylococcus aureus, n (%)	15 (6.3)
Pseudomonas aeruginosa, n (%)	14 (5.9)
Acinetobacter spp., n (%)	2 (0.8)
Diagnostic Turnaround Times	
Molecular method (hours), mean ± SD	19.7 ± 10.9
Molecular method (hours), median [IQR]	16.4 [14.1-20.6]
Conventional method (hours), mean ± SD	52.9 ± 19.4
Conventional method (hours), median [IQR]	47.5 [41.8-61.4]
Time difference (hours), mean ± SD	33.2 ± 14.2
Time difference (hours), median [IQR]	30.7 [24.6-38.9]

Table 2: Diagnostic Turnaround Time by Clinical Characteristics

Characteristic	N	Molecular (h) Mean ± SD	Phenotypic (h) Mean ± SD	Difference (h) Mean ± SD	P-value
GLOBAL	237	19.9 ± 11.1	53.0 ± 19.3	33.1 ± 14.2	<0.001†
By Gram classification					
Gram-negative	179	19.4 ± 11.4	50.0 ± 16.8	30.6 ± 11.1	<0.001†
Gram-positive	49	18.8 ± 5.9	58.1 ± 13.1	39.3 ± 10.6	<0.001†
Fungi	8	37.3 ± 16.0	92.7 ± 43.1	55.3 ± 42.4	0.008†
By resistance presence					
With resistance	102	17.9 ± 10.2	48.7 ± 16.0	30.8 ± 11.6	<0.001†
Without resistance	135	21.4 ± 11.6	56.2 ± 20.9	34.8 ± 15.7	<0.001†
By clinical setting					
Hospitalized	113	19.4 ± 11.1	52.8 ± 18.1	33.4 ± 12.8	<0.001†
Ambulatory	121	20.5 ± 11.3	53.1 ± 20.6	32.7 ± 15.4	<0.001†
By specific pathogen					
Escherichia coli	82	18.3 ± 11.3	46.3 ± 14.3	28.0 ± 8.1	<0.001†
Klebsiella pneumoniae	23	16.1 ± 4.7	47.3 ± 15.7	31.3 ± 14.9	<0.001†
Staphylococcus aureus	13	16.4 ± 2.3	50.0 ± 8.0	33.6 ± 6.9	<0.001†
Pseudomonas aeruginosa	14	22.7 ± 13.7	55.6 ± 15.0	32.9 ± 12.8	<0.001†
Salmonella spp.	17	19.6 ± 5.1	47.0 ± 8.5	27.4 ± 8.9	<0.001†

Notes:

† Wilcoxon paired test comparing molecular vs. phenotypic times within each subgroup

SD=Standard Deviation

h = hours

Table 2A: Bacterial Identification Concordance By Clinical Subgroups

Clinical_Characteristic	N	Complete n (%)	Partial n (%)	Discordant n (%)	Combined %	95% CI
OVERALL	237	189 (79.7%)	35 (14.8%)	13 (5.5%)	94.5%	(90.8-96.8%)
By Gram Classification						
Gram-negative bacteria	179	149 (83.2%)	22 (12.3%)	8 (4.5%)	95.5%	(91.4-97.7%)
Gram-positive bacteria	49	32 (65.3%)	13 (26.5%)	4 (8.2%)	91.8%	(80.8-96.8%)
Fungi	8	8 (100.0%)	0 (0.0%)	0 (0.0%)	100.0%	(67.6-100.0%)
By Resistance Status						
With antimicrobial resistance	102	92 (90.2%)	7 (6.9%)	3 (2.9%)	97.1%	(91.7-99.0%)
Without resistance	135	97 (71.9%)	28 (20.7%)	10 (7.4%)	92.6%	(86.9-95.9%)
By Clinical Setting						
Hospitalized patients	113	100 (88.5%)	9 (8.0%)	4 (3.5%)	96.5%	(91.3-98.6%)
Outpatient/Community	121	87 (71.9%)	25 (20.7%)	9 (7.4%)	92.6%	(86.5-96.0%)
By Specific Pathogen						
Escherichia coli	83	82 (98.8%)	0 (0.0%)	1 (1.2%)	98.8%	(93.4-99.8%)
Klebsiella pneumoniae	23	20 (87.0%)	2 (8.7%)	1 (4.3%)	95.7%	(79.0-99.2%)
Staphylococcus aureus	13	13 (100.0%)	0 (0.0%)	0 (0.0%)	100.0%	(77.2-100.0%)
Salmonella spp.	17	2 (11.8%)	15 (88.2%)	0 (0.0%)	100.0%	(81.6-100.0%)
Streptococcus spp.	16	5 (31.2%)	11 (68.8%)	0 (0.0%)	100.0%	(80.6-100.0%)

Complete: Molecular and MALDI-TOF identify same species

Partial: Molecular identifies genus, MALDI-TOF identifies genus + species (clinically valuable)

Discord: Methods disagree (mainly pathogens outside molecular panel)

Combined: Complete + Partial concordance

Table 2B: Antimicrobial Resistance Concordance By Clinical Subgroups

Clinical_Characteristic	N	Complete n (%)	Partial n (%)	Discordant n (%)	Combined %	95% CI
OVERALL	237	210 (88.6%)	2 (0.8%)	25 (10.5%)	89.5%	(84.9-92.8%)
By Gram Classification						
Gram-negative bacteria	179	162 (90.5%)	2 (1.1%)	15 (8.4%)	91.6%	(86.6-94.9%)
Gram-positive bacteria	49	41 (83.7%)	0 (0.0%)	8 (16.3%)	83.7%	(71.0-91.5%)
Fungi	8	7 (87.5%)	0 (0.0%)	1 (12.5%)	87.5%	(52.9-97.8%)
By Resistance Status						
With antimicrobial resistance	102	88 (86.3%)	2 (2.0%)	12 (11.8%)	88.2%	(80.6-93.1%)
Without resistance	135	122 (90.4%)	0 (0.0%)	13 (9.6%)	90.4%	(84.2-94.3%)
By Clinical Setting						
Hospitalized patients	113	99 (87.6%)	2 (1.8%)	12 (10.6%)	89.4%	(82.4-93.8%)
Outpatient/Community	121	108 (89.3%)	0 (0.0%)	13 (10.7%)	89.3%	(82.5-93.6%)
By Specific Pathogen						
Escherichia coli	82	78 (95.1%)	2 (2.4%)	2 (2.4%)	97.6%	(91.5-99.3%)
Klebsiella pneumoniae	23	22 (95.7%)	0 (0.0%)	1 (4.3%)	95.7%	(79.0-99.2%)
Staphylococcus aureus	13	13 (100.0%)	0 (0.0%)	0 (0.0%)	100.0%	(77.2-100.0%)
Salmonella spp.	17	16 (94.1%)	0 (0.0%)	1 (5.9%)	94.1%	(73.0-99.0%)
Streptococcus spp.	16	11 (68.8%)	0 (0.0%)	5 (31.2%)	68.8%	(44.4-85.8%)

Complete: Genotype and phenotype agree on resistance mechanism

Partial: Minor discrepancy in resistance detection

Discord: Gene+/Phenotype- or Gene-/Phenotype+ (mainly chromosomal AmpC)

Combined: Complete + Partial concordance

Table 3: Therapeutic Concordance By Clinical Subgroups

Clinical Characteristic	N	Concordant n(%)	Discordant n(%)	Concordance %	95% CI
OVERALL	236	192 (81.4%)	44 (18.6%)	81.4%	(75.9-85.8%)
By Gram Classification					
Gram-negative bacteria	179	151 (84.4%)	28 (15.6%)	84.4%	(78.3-89.0%)
Gram-positive bacteria	48	34 (70.8%)	14 (29.2%)	70.8%	(56.8-81.8%)
By Resistance Status					
With antimicrobial resistance	102	87 (85.3%)	15 (14.7%)	85.3%	(77.1-90.9%)
Without resistance	134	105 (78.4%)	29 (21.6%)	78.4%	(70.6-84.5%)
By Bacterial ID Concordance					
Bacterial ID concordant	189	163 (86.2%)	26 (13.8%)	86.2%	(80.6-90.4%)
Bacterial ID discordant	47	29 (61.7%)	18 (38.3%)	61.7%	(47.4-74.2%)
By Resistance Detection Concordance					
Resistance detection concordant	209	184 (88.0%)	25 (12.0%)	88.0%	(82.9-91.8%)
Resistance detection discordant	27	8 (29.6%)	19 (70.4%)	29.6%	(15.9-48.5%)
By Clinical Setting					
Hospitalized patients	112	90 (80.4%)	22 (19.6%)	80.4%	(72.0-86.7%)
Outpatient/Community	121	99 (81.8%)	22 (18.2%)	81.8%	(74.0-87.7%)
By Specific Pathogen					
Escherichia coli	82	78 (95.1%)	4 (4.9%)	95.1%	(88.1-98.1%)
Klebsiella pneumoniae	23	21 (91.3%)	2 (8.7%)	91.3%	(73.2-97.6%)
Staphylococcus aureus	13	13 (100.0%)	0 (0.0%)	100.0%	(77.2-100.0%)

Concordant: OneChoice (molecular-based) and OneChoice Fusion (molecular+phenotypic) recommend same antibiotic

Discordant: Recommendations differ due to additional phenotypic susceptibility data

Table 4: Predictors Of Therapeutic Discordance

Predictor	Univariate OR	Univariate P-value	Adjusted OR	95% CI	Adjusted P-value
Resistance Detection Discordant	9.16	<0.001***	7.70	3.61 - 17.49	<0.001***
Gram-Positive (vs Gram-Negative)	2.10	0.031*	1.77	0.84 - 3.56	0.124
Hospitalized (vs Outpatient)	1.13	0.701	1.24	0.65 - 2.30	0.516
Resistance Present (vs None)	0.64	0.156	0.69	0.33 - 1.45	0.316
Bacterial ID Discordant	6.31	<0.001***	2.76	1.20 - 6.32	0.019*
Male (vs Female)	0.76	0.369	0.76	0.42 - 1.51	0.395
Age (per year)	1.00	0.858	1.00	0.99 - 1.02	0.501
Molecular Turnaround Time	1.02	0.117	1.02	0.99 - 1.06	0.197

OR: Odds Ratio; CI: Confidence Interval. Multivariable logistic regression analysis was performed to identify independent predictors of therapeutic discordance, defined as disagreement between OneChoice and OneChoice Fusion treatment recommendations. All variables shown were included simultaneously in the multivariable model (N = 224 complete cases, 42 discordant cases [18.8%]). Model fit: Pseudo R^2 (McFadden) = 0.202; Likelihood Ratio Test: $\chi^2 = 43.59$, df = 8, $P < 0.001$. * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

Figures

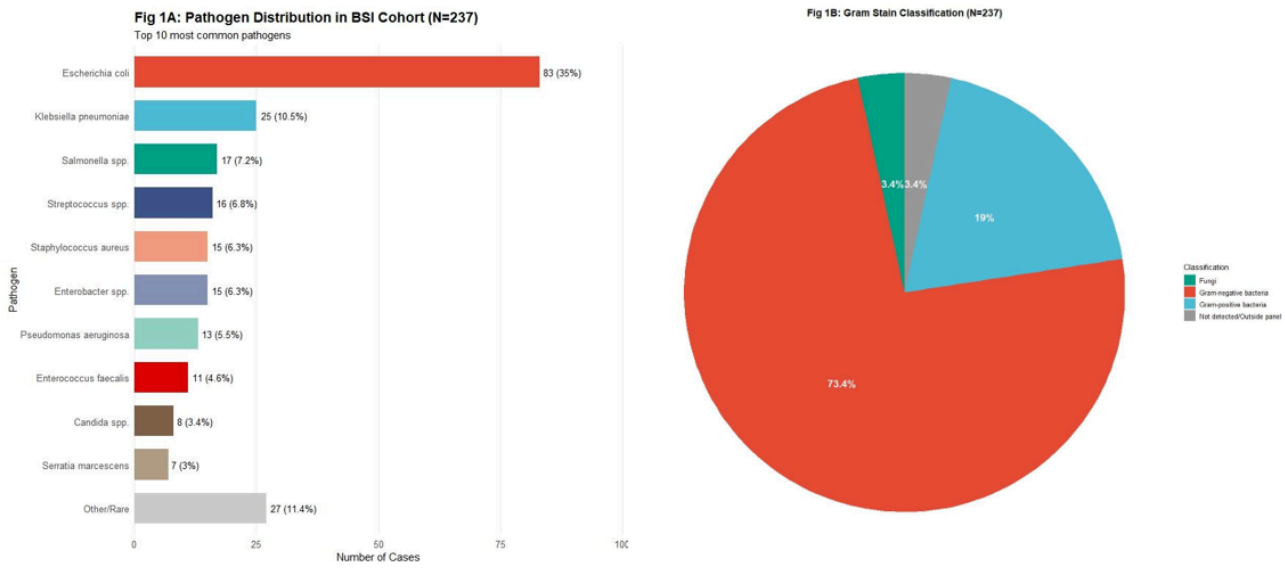


Figure 1

Pathogen Distribution in Bloodstream Infection Cohort (N=237)

(**A**) Horizontal bar chart showing distribution of the 10 most common pathogens: *Escherichia coli* 83 (35.0%), *Klebsiella pneumoniae* 25 (10.5%), *Salmonella*spp. 17 (7.2%), *Streptococcus* spp. 16 (6.8%), *Staphylococcus aureus*15 (6.3%), *Enterobacter* spp. 15 (6.3%), *Pseudomonas aeruginosa* 13 (5.5%), *Enterococcus faecalis* 11 (4.6%), *Candida* spp. 8 (3.4%), *Serratia marcescens* 7 (3.0%), Other/Rare 27 (11.4%). Bars color-coded by genus/family. (**B**) Pie chart showing Gram stain classification: Gram-negative 174 (73.4%, red), Gram-positive 45 (19.0%, blue), Fungi 8 (3.4%, green), Not detected/Outside panel 8 (3.4%, gray). Predominance of Gram-negative bacteria and *E. coli* reflects typical BSI epidemiology with additional burden of enteric pathogens (*Salmonella*) in Peru.

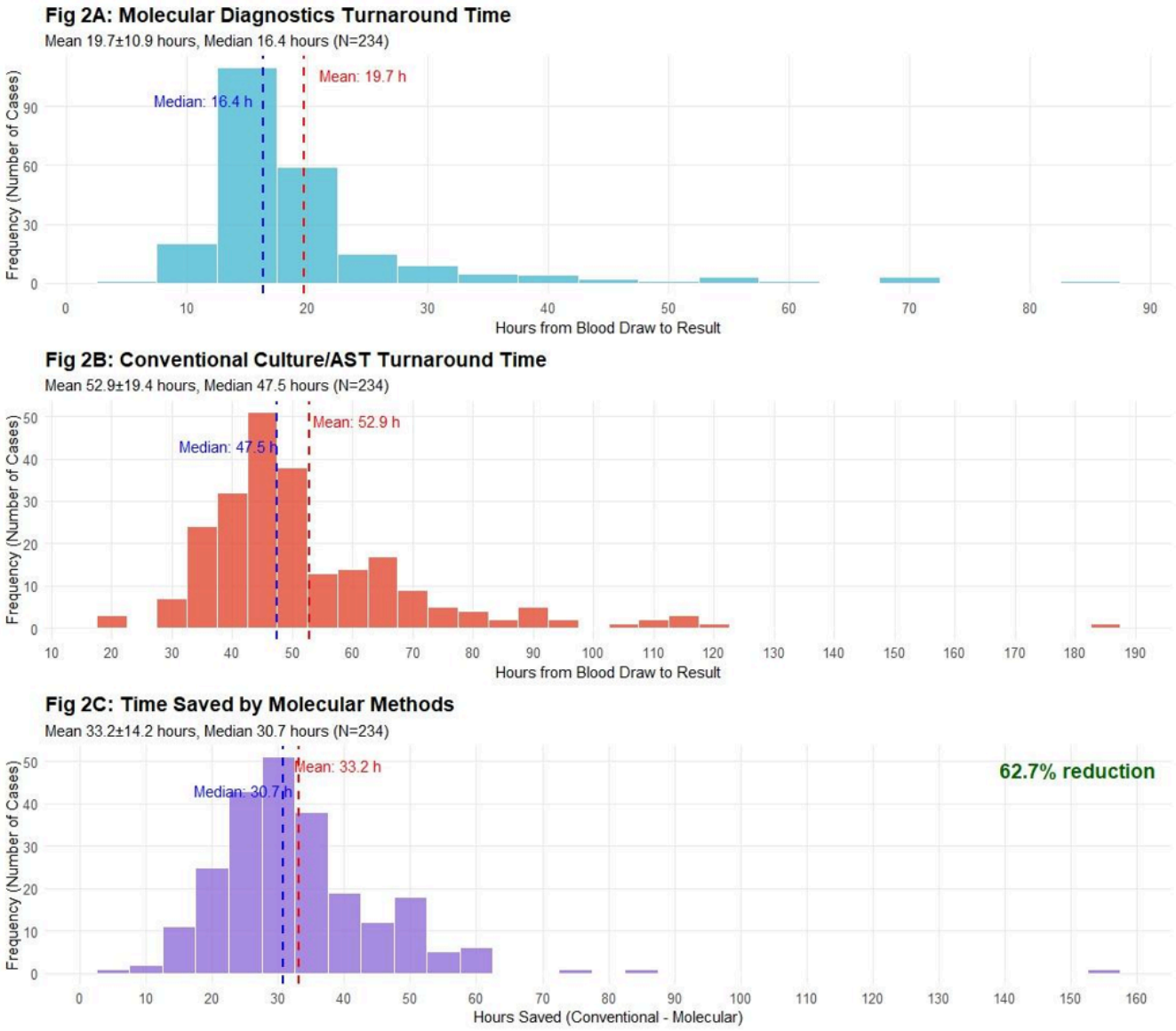


Figure 2

Distribution of Diagnostic Turnaround Times: Molecular vs Conventional Methods (N=237)

Histogram panels showing frequency distribution of turnaround times (hours from blood draw to result availability) for (**A**) molecular diagnostics (FilmArray BCID or Xpert MRSA/SA BC), mean 19.7±10.9 hours, median 16.4 hours (blue); (**B**) conventional culture with MALDI-TOF identification and phenotypic antimicrobial susceptibility testing, mean 52.9±19.4 hours, median 47.5 hours (red); and (**C**) time saved by molecular methods (conventional minus molecular turnaround time), mean 33.2±14.2 hours, median 30.7 hours, representing 62.7% time reduction (purple). Dashed red line = mean, dashed blue line = median. Wilcoxon signed-rank test $P<0.001$, Cohen's $d=2.33$ (very large effect size).

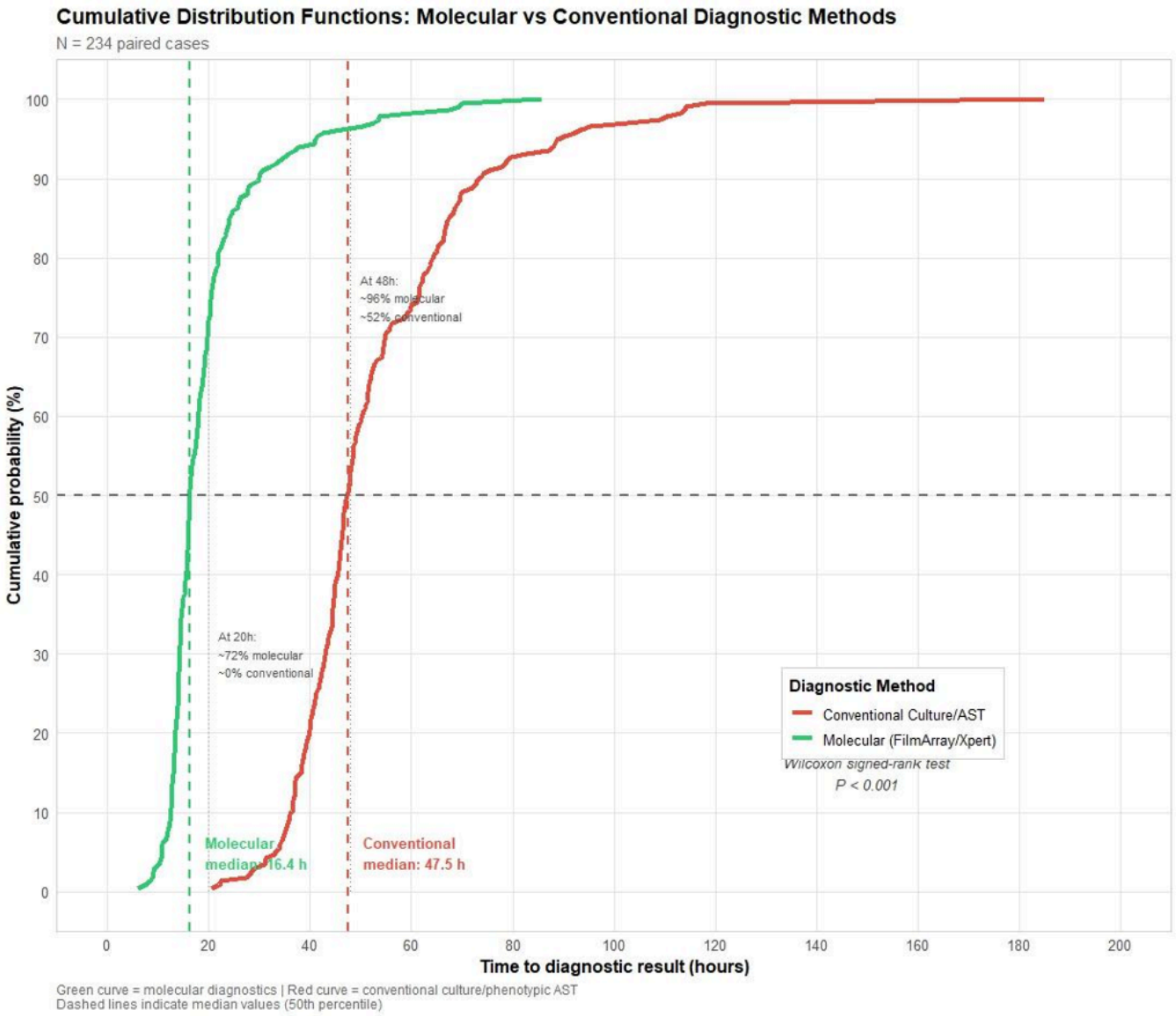


Figure 3

Cumulative Distribution Functions: Molecular vs Conventional Diagnostic Methods (N=237)

Cumulative probability plots showing proportion of cases achieving diagnostic results as a function of time. Green curve = molecular diagnostics (FilmArray/Xpert), median 16.4 hours. Red curve = conventional culture/phenotypic AST, median 47.5 hours. Horizontal dashed line indicates 50th percentile (median). Vertical dashed lines mark respective medians. The rightward shift of the conventional curve relative to molecular curve illustrates substantial time delay. At 20 hours, ~60% of molecular diagnostics are complete vs ~5% of conventional methods. At 48 hours, 95% molecular complete vs ~55% conventional. Wilcoxon signed-rank test *P*<0.001.

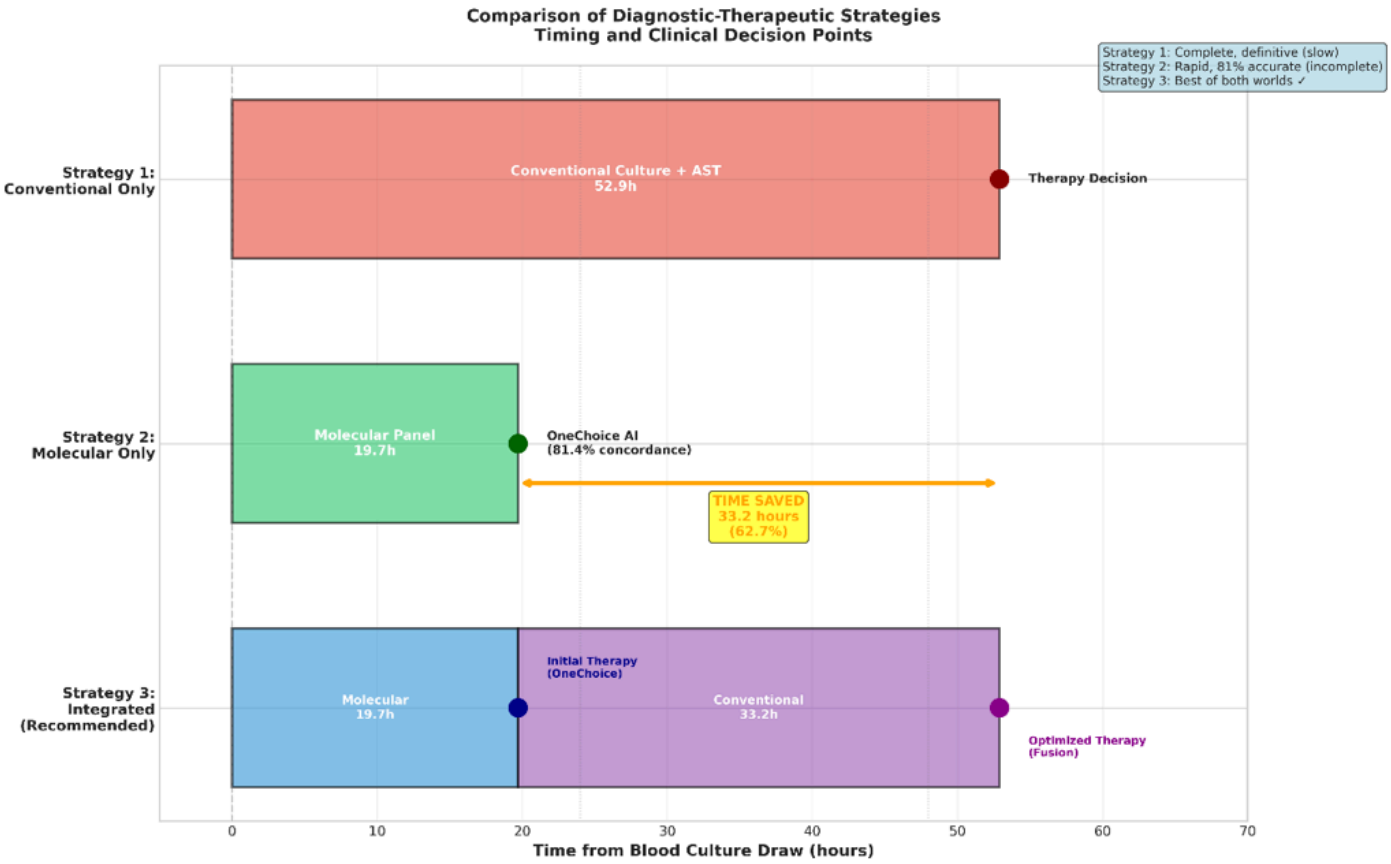


Figure 4

Timeline Comparison of Diagnostic-Therapeutic Strategies

Horizontal timeline comparing three diagnostic-therapeutic strategies from blood culture draw (0 hours) to therapy decision point. (**Strategy 1**: Conventional Only) Single red bar extending to 52.9 hours for conventional culture/AST completion before therapy decision. (**Strategy 2**: Molecular Only) Single green bar extending to 19.7 hours for molecular diagnostics/OneChoice AI, with annotation showing 81.4% concordance rate and orange arrow indicating 33.2-hour time advantage vs conventional. (**Strategy 3**: Integrated/Recommended) Blue bar to 19.7 hours (molecular/OneChoice initial therapy) followed by purple bar extending to 52.9 hours (conventional/Fusion optimization), with dual decision points marked. Clinical outcomes annotation box: 81.4% receive appropriate therapy without adjustment (green), 18.6% receive optimization (purple), 100% receive appropriate therapy ultimately. Time saved annotation emphasizes 33.2-hour therapeutic window representing 62.7% reduction.

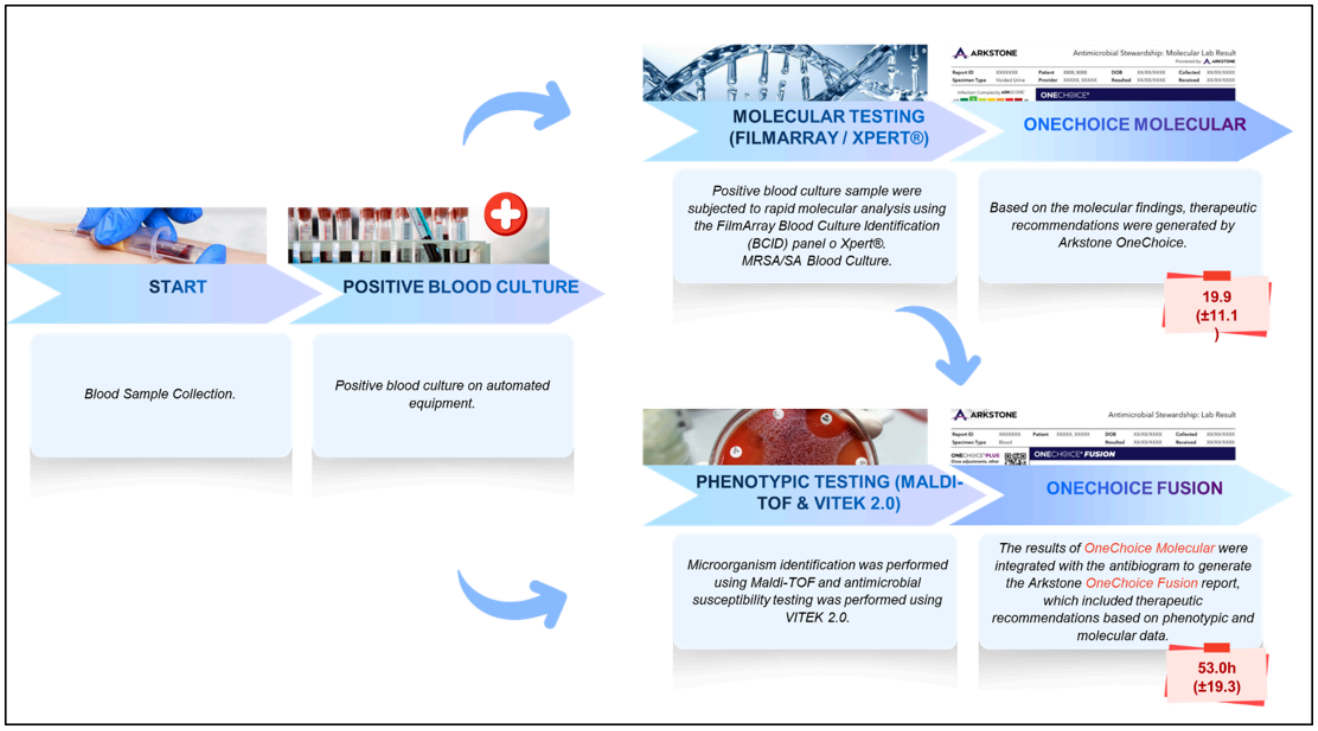


Figure 5

Flowchart 1.- Bacteriemia Workflow program

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- suppl2.pdf
- suppl1.pdf