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Highlights

- Invasive bacterial infections were frequent in hospitalised advanced-HIV patients, Kinshasa
- Non-typhoidal *Salmonella* was the leading cause of community-acquired invasive infections
- *K. pneumoniae* was the most common pathogen in hospital-acquired invasive infections
- Invasive bacterial infections contributed to an increased risk of death
- High ceftriaxone and emerging carbapenem resistance pose serious concern

Community and hospital-acquired invasive bacterial infections in hospitalised patients with advanced HIV disease: a prospective study in Kinshasa, DRC

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ABSTRACT

Background

Advanced HIV patients face high mortality, often from invasive bacterial infections (IBI), while rising antimicrobial resistance (AMR) threatens treatment. This study reports IBIs and AMR in hospitalised advanced HIV patients in Kinshasa, Democratic Republic of the Congo (DRC).

Methods

In this prospective study, all patients with blood (BC) or cerebrospinal fluid (CSF) culture on admission or during hospitalisation were eligible to participate. An IBI was defined as a positive blood or CSF culture and categorised as community-acquired IBI if occurring <48h since admission, or hospital-acquired IBI if occurring ≥48h after admission.

Results

We included 724 patients over one year. Community-acquired IBI was suspected in 648 hospitalisations and confirmed in 108 (16.7%). The incidence of hospital-acquired IBI was 2.4 per 1000 patient-days. Non-typhoidal *Salmonella* and *K. pneumoniae* were the leading cause of community- (46%, 53/116) and hospital-acquired IBI (42%, 10/24), respectively. Ceftriaxone resistance was observed in 80% of Enterobacterales from community-acquired IBI. In-hospital mortality was significantly higher in hospital-acquired IBI (55%) compared to community-acquired IBI (35%, p<0.001) and BC-negative patients (21%, p<0.001).

Conclusions

IBI are frequent in hospitalised advanced HIV patients in DRC, with high mortality and alarming resistance patterns, highlighting the need for carbapenem-sparing strategies.

Keywords: Invasive bacterial infection; advanced HIV disease; antimicrobial resistance; low-resource settings

3619/3500 words

INTRODUCTION

Sub-Saharan Africa (SSA) bears a significant dual burden of advanced HIV disease (AHD) and antimicrobial resistance (AMR), two critical public health challenges. AHD is characterised by a CD4 count below 200 cells/mm^3 or a WHO HIV clinical stage of 3 or 4, a condition that predisposes individuals to severe infections [1]. In SSA, it is estimated that around 10% of people living with HIV (PLHIV) can be included in the AHD definition because of their CD4 count [2].

PLHIV are exposed to an elevated risk of invasive bacterial infections (IBIs). A systematic review highlights that the proportion of community-acquired IBIs in hospitalised PLHIV ranges from 10% to 30%, compared to 9% in HIV-negative individuals [3]. In Africa, PLHIV are disproportionately affected by bacterial pathogens such as *Streptococcus pneumoniae* and non-typhoidal *Salmonella* (NTS) compared to PLVIH in other world regions and to HIV-negative populations [4]. Mortality rates associated with IBIs in PLHIV range from 7% to 46% [3].

Simultaneously, the AMR pandemic poses a growing threat in the region. In 2021, AMR contributed to an estimated 4.71 million deaths globally, with the highest associated death rate in SSA, reaching 81.5 deaths per 100000 (95%CI 64.6– 98.4) [5]. Despite limited epidemiological data, the available evidence underscores alarming AMR prevalence and associated negative outcomes in SSA [6].

Despite these challenges, only a few studies have described AMR in IBI among PLHIV in SSA. Studies report ceftriaxone-resistant NTS IBIs in 13% of cases in rural Uganda (1996–2007) [7] and up to 40% in Ethiopia (2017–2018) [8]. In South Africa, extended-spectrum beta-lactamase (ESBL)-producing *Escherichia coli* accounted for 31% of IBIs in HIV-positive children [9], while 96% of *Klebsiella pneumoniae* IBI

showed resistance to third-generation cephalosporins, with HIV-positive status being identified as a risk factor [10]. Reduced penicillin susceptibility in *S. pneumoniae* IBIs ranges from 32% to 87% across HIV cohorts in SSA [7,11]. Additionally, methicillin-resistant *Staphylococcus aureus* (MRSA) is emerging as a significant cause of both community- and hospital-acquired IBIs in PLHIV, with prevalence ranging from 23% to 47% across studies [11,12].

However, there is a lack of research focusing on hospitalised AHD patients that comprehensively analyses IBI aetiologies and antimicrobial susceptibility profiles while distinguishing between community- and hospital-acquired infections.

This study aims to address this gap by describing the prevalence, aetiology, and antimicrobial resistance profiles of community- and hospital-acquired IBIs among hospitalised AHD patients in a Médecins Sans Frontières (MSF)-supported HIV treatment centre in Kinshasa, Democratic Republic of the Congo (DRC). The findings are intended to inform evidence-based antibiotic stewardship strategies, with the dual objective of reducing hospital-related mortality and limiting the emergence of AMR, while improving standards of inpatient care.

METHODS

Study site

The study took place in the Centre Hospitalier de Kabinda (CHK), in Kinshasa, DRC, a referral hospital centre supported by MSF that provides care for PLHIV. The CHK consists of a 41-bed inpatient department and an outpatient department where a cohort of around 1,500 patients is followed up. The hospital has been admitting around 2,000 AHD patients a year since 2016. Patients can be admitted 24 hours a day, 7 days a week. The majority of admitted patients have a previously known HIV status, as they are either followed within the cohort or referred from MSF-supported

peripheral health facilities where the HIV diagnosis has already been established and confirmed by viral load testing. For patients referred from other health facilities or self-referred patients, HIV infection is confirmed at admission through viral load measurement and CD4 cell count.

Study design and study population

This was a prospective descriptive study of AHD patients admitted to the CHK from August 2021 to July 2022. The eligibility criteria for the study were based on the indications for prescribing a blood culture (BC) and/or a cerebrospinal fluid (CSF) culture on admission or during hospitalisation as part of routine care:

- Fever (axillary temperature >38°C) or hypothermia (<36°C)
- And/or signs of shock:
    - Patients aged 12 years or older: hypotension defined as systolic blood pressure <90mmHg;
    - Patients under 12 years of age: capillary refill time > 3 seconds and/or cold hands and/or temperature difference between the upper and lower limbs and/or tachycardia;
- And/or a patient in whom a doctor strongly suspects meningitis or septicaemia on clinical grounds and decides to prescribe a blood culture or a lumbar puncture even without fever or signs of shock.

There were no exclusion criteria.

Inclusion and data collection

A study nurse assessed the eligibility of all patients with HIV on admission, within the 48 hours following admission or in the event of clinical deterioration after at least 48 hours of hospitalisation. Patients admitted overnight or on Sunday were managed by

the medical team and assessed for eligibility the following morning. After obtaining written consent from the participant or their guardian (if the patient was under 18 or if their clinical condition did not allow consent to be sought), a nurse trained in the study procedures collected the patient's sociodemographic, clinical, biological and therapeutic characteristics after medical assessment by a physician. Data were collected using a standardised form, through individual interviews in French or Lingala with the patient or their carer and through clinical records up to the patient's discharge from hospital. Pseudonymised sociodemographic and clinical data were entered at the CHK using REDCap software (http://project-redcap.org/).

Sample collection and laboratory procedures

Blood samples were taken by trained nurses and transferred to blood culture bottle (BacT/ALERT®, bioMérieux, Marcy L'Étoile, France) as part of routine examinations prescribed by the medical doctors, preferably before initiating antibiotic therapy. One set of BC was collected per patient and per episode. For each BC set, two bottles were drawn for adults (10mL of blood per bottle) and one bottle for children (2.5-5mL of blood for children aged 2 to 15 years; 1-2mL for under 2years).

Blood cultures and CSF cultures were analysed in the HJ Hospital in Kinshasa during the first month of the study, August 2021 then later in CHK bacteriology laboratory. Upon receipt, blood culture bottles were incubated at +35°C in BacT/ALERT® 3D instrument. Blood cultures were declared negative if there was no evidence of bacterial growth after 5 days of incubation. CSF was analysed by cytology (leucocytes) and biochemistry (glycorrhachia and proteinorachia) and then inoculated onto appropriate culture media for pathogen isolation (irrespective of leucocytes count and biochemistry, except the first month of the study). Antibiotic susceptibility testing (AST) was performed using the Kirby-Bauer disk diffusion method and E-test

per the European Committee on Antimicrobial Susceptibility Testing recommendations, 2022. Pathogen identification was done using a metabolic method with API strips (bioMérieux).

Intermediate results (cyto-biochemistry and Gram staining on direct examination of the CSF and Gram staining on culture broth for BCs) and final results (identification of the pathogen and AST) were communicated to the medical doctors to allow adjustments to patient management if necessary. Additional routine biological tests were requested by the medical doctors as part of the patient's diagnosis and follow-up: malaria rapid test (SD Bioline Malaria Ag Pf, Alere), haemogram (QBC Autoread), CD4 count (PIMA), viral load (Abbott real-time), cryptococcosis (cryptococcal antigen [CrAg]). Tuberculosis (TB) screening was systematically performed in patients with unknown TB status using clinical assessment. When the clinical presentation was suggestive of TB, urine TB-LAM (Alere) and GeneXpert MTB/RIF (Cepheid) tests were used for diagnostic confirmation and to rule out rifampicin resistance. In patients without TB signs or symptoms, TB-LAM was used as a screening tool. Overall, TB diagnosis was primarily based on clinical presentation, supported by laboratory confirmatory results. Other opportunistic infections, such as Pneumocystis or Toxoplasmosis, were diagnosed clinically.

All bacteriological results were entered into the WHOnet data management software (http://www.whonet.org). They were then cross-referenced with clinical data from REDCap on the basis of the study inclusion number.

Clinical management

Patients included in the study were treated according to locally adapted MSF guidelines for empiric treatment. The 2021 version of those guidelines was guided by the overall microbiological results issued by the external validated

laboratory performing BCs before the CHK microbiological laboratory was established in September 2021.

Meropenem was the recommended first-line empiric treatment for septic shock or sepsis with one of the following signs or symptoms: hypotension with mean arterial pressure <65 mmHg, respiratory rate >30 cycles/minute and SpO2<90% in ambient air, or Glasgow coma scale <8/15. Vancomycin was added when the suspected source of infection was cutaneous.

Different treatment schedules may have been prescribed due to a shortage of meropenem or a different diagnosis upon admission.

Statistical analysis

Confirmed IBI was defined as the growth of clinically significant bacteria in BC. Non-pathogenic bacteria or skin flora, including coagulase-negative *Staphylococcus*, *Bacillus* sp, and *Micrococcus* sp, may have been considered contaminants depending on clinical considerations. Community-acquired IBI was defined as any IBI diagnosed within the first 48 hours of hospitalisation at the CHK. A hospital-acquired IBI (or nosocomial infection) was defined as any IBI that began 48 hours or more after the start of hospitalisation. The incidence of hospital-acquired IBI was calculated as the number of episodes per 1000 patient-days. Patient-days were defined as the duration (in days) of each patient's first hospitalisation during the study period, included only if the stay lasted two days or more.

Data were analysed using Stata®15 software (Stata Corporation, College Station, Texas, USA). Continuous variables were summarised by mean, median, standard deviation, minimum and maximum; categorical variables by percentages. Categorical data were analysed using Chi^2 or Fisher's exact test and continuous data using Student's t-test. The significance threshold was set at <0.05.

RESULTS

Characteristics on admission

Eligibility was assessed for 1725 admissions between August 2, 2021, and July 31, 2022. Among the 1123 admissions found to be eligible, consent could not be sought for 44, and 9 patients declined to participate. A total of 1070 admissions were included in the study, corresponding to 769 patients. Of the included admissions, 970 blood cultures and/or CSF cultures were prescribed, corresponding to 724 patients (Figure 1). We selected the first admission of each of those patients. The majority (573/724, 79%) had samples taken only within the first 48h of hospitalisation: 441 had a blood culture, 10 had a CSF culture, and 122 had both. A smaller proportion of patients (76/724, 11%) had samples taken only after 48 hours of hospitalisation. The median age was 40 years; 62% were women and 64% had <200 CD4/mm^3. On admission, 51% of patients had a viral load ≥1000 copies/mm^3, and of these, 55% had either never received antiretrovirals (ARVs) or had interrupted treatment. A total of 636 patients (88%) had tuberculosis, including 64% who were newly diagnosed and 24% who were undergoing treatment (Supplementary File). Approximately a quarter of patients had suspected cerebral toxoplasmosis or *Pneumocystis jirovecii* pneumonia (Table 1).

Community-acquired IBI

Community-acquired IBI was suspected within the first 48h of 648 hospitalisations, with a median length of stay of 5 days [IQR 3;8.5]. Of these, 108 (16.7%; 95% confidence interval [95%CI] 13.9–19.8) were confirmed, including one case of meningitis. The prevalence of community-acquired IBI

among all admitted patients was 8.8% (108/1233; 95%CI 7.2-10.5). Community-acquired IBI was significantly more frequent in patients with viral loads ≥1000 copies/mm^3 compared to those with viral loads <1000 copies/mm^3 (18.6% vs. 10.8%; p=0.008). Similarly, community-acquired IBI was more common in patients with very severe anaemia compared to those without (27.5% vs. 13.5%; p=0.001). We found no significant difference between the proportion of community-acquired IBI in patients with CD4 <200/mm^3 (16.5%) and ≥200/mm^3 (12.3%; p=0.13).

Among confirmed community-acquired IBI, 69% were caused by Enterobacterales. The most frequent were NTS (45.7%), *E. coli* (19.0%) and S. *pneumoniae* (17.3%), the latter being responsible for the meningitis case (Table 2).

Hospital-acquired IBI

Hospital-acquired IBI was suspected in 151 hospitalisations, with a median length of stay of 10 days [IQR 6;21]. Of these, 22 (14.6%; 95%CI 9.4–21.2) were confirmed by a positive BC, giving an incidence of 2.4 per 1000 patient-days (total patient-days=9005 for 1035 patients; 95%CI 1.5–3.7). No CSF had positive growth. The median time to onset of nosocomial infection was 12.5 days after the start of hospitalisation (IQR 8 to 18). Two patients had both community-acquired and hospital-acquired IBI. Half (11/22; 50%) of hospital-acquired IBI cases were aged between 41 and 60 years, and a majority had CD4<100/mm^3 and viral load ≥1000 copies/mm^3 (15/22; 68%; Table 1). Among hospital-acquired IBIs, the most frequent pathogens were *K. pneumoniae* (42%), followed by *S. aureus* (13%) and *A. baumannii* (13%) (Table 2).

Antimicrobial resistance

High proportion of NTS were resistant to fluoroquinolones (79%, 41/53), the first-line treatment. No resistance to azithromycin has been detected (Table 3). In community-acquired IBI, 80% (64/80) of NTS and other Enterobacterales were resistant to third-generation cephalosporins and 93% (14/16) in hospital-acquired IBI (mainly expressing extended-spectrum beta-lactamases, ESBL). Resistance to piperacillin-tazobactam in non-NTS Enterobacterales was 33% (9/27) in community-acquired IBI and 43% (6/16) in hospital-acquired IBI. Regarding resistance to carbapenems, in community-acquired IBI, 1 *E. coli* was resistant to meropenem while in hospital-acquired IBI, 4 meropenem-resistant Enterobacterales were isolated (1 *E. cloacae*, 1 *E. coli*, 2 *K. pneumoniae*). Among them, 1 was resistant to tigecycline (*E. cloacae*) and 2 to colistin (*E. cloacae* and *K. pneumoniae*; Table 3).

Among the 20 *S. pneumoniae* isolated in community-acquired IBI, 10 (50%) were non-susceptible to oxacillin (including the one isolated from the CSF), but all were susceptible to ceftriaxone; 17 (85%) were resistant to cotrimoxazole and 16 (80%) to tetracycline (Supplementary table 2).

Two of three *S. aureus* isolated in community-acquired IBI were MRSA and three of three *S. aureus* isolated in hospital-acquired IBI were MRSA.

Clinical outcomes

Among the 724 patients included in the analysis, 175 (24%) died during hospitalisation (Table 4). Of these, 50 deaths (29%) occurred within the first 48 hours of hospitalisation. The mortality was significantly higher among patients with community-acquired IBI (35%, 38/108) compared to those without IBI within the first 48 hours of hospitalisation (21%, 126/596, p=0.005). The mortality among hospital-

acquired IBI (55%, 12/22) was significantly higher than among patients without hospital-acquired IBI (55% vs. 23%; p=0.002). One of the two patients with both community- and hospital-acquired IBI died. No significant association was found between mortality and the bacterial aetiology of IBI. Among patients with community-acquired IBI, the proportion of in-hospital deaths was 31% (15/49) for those with NTS, 55% (18/33) for other Enterobacterales, and 35% (16/46) for other bacterial infections (p=0.082).

DISCUSSION

Community-acquired IBI were confirmed in 16.7% of suspected cases (8.8% of all admissions), with higher rates in patients with high viral load (18.6%) and very severe anaemia (27.5%). Non-typhoidal Salmonella (45.7%), *E. coli*, and *S. pneumoniae* were the main pathogens; 80% of Enterobacterales were resistant to third-generation cephalosporins, and 79% of NTS to fluoroquinolones, though all *S. pneumoniae* remained susceptible to ceftriaxone. Hospital-acquired IBI occurred at 2.4 per 1000 patient-days, mainly caused by *K. pneumoniae*, with over 90% of isolates resistant to third-generation cephalosporins. In-hospital mortality was 24% overall, rising to 35% and 55% in community- and hospital-acquired IBI, respectively, although no direct link with IBI aetiology was established in this highly comorbid population.

The prevalence of community-acquired IBI among hospitalised patients is similar to that reported in other studies in the PLHIV in Africa [3,13,14]. As expected, community-acquired IBIs were significantly more frequent in patients with high viral load, which may reflect poor ARV compliance or patient being naïve to ARV. In contrast to findings from other studies [7,15], no significant difference was observed according to CD4 counts.

The incidence of hospital-acquired IBI during the study period was estimated at 21.3 per 1000 patients (2.4 per 1000 patient-days), which is similar to the pooled incidence of hospital-acquired IBI (15.4 per 1000 patients; 95% CI 9.2–25.7) reported in a recent meta-analysis [16]. Notably, none of the studies included in that meta-analysis were conducted in SSA or on HIV patients. Our study is the first to report incidence of hospital-acquired IBI among such a large number of AHD patients in SSA. Monitoring the incidence of hospital-acquired IBI is important to assess and improve quality of care and IPC measures. However, hospital-acquired IBI are not always caused by in-hospital transmission. AHD patients are particularly susceptible to “colonisation to invasive infection events” due to translocation of gastrointestinal or upper airways microflora [17], reported to carry high proportion of multi-drug resistance organisms [18]. Therefore, IPC measures should play a central role in reducing the cross-transmission of drug-resistant bacteria between hospitalised patients, an established risk factor for subsequent invasive infections [19].

The most common pathogen involved in community-acquired IBI was NTS, followed by *E. coli* and *S. pneumoniae.* These results are consistent with other studies in DRC where several studies in referral hospitals showed that invasive NTS (iNTS) accounted for the main pathogen isolated from blood cultures among children and adults [20,21]. Those results are also in line with other studies in SSA [22], showing emergence of iNTS in this region, leading to a major public health concern, particularly in HIV or malaria-endemic area [23]. The main organisms causing hospital-acquired IBIs were *K. pneumoniae* followed by *S. aureus* and *A. baumannii*, all members of the ESKAPE group of

pathogens, which are particularly associated with multidrug-resistant hospital-acquired infections [24] and have been reported in other studies in SAA [25]. The antimicrobial resistance profiles of Enterobacterales resulting from this study are deeply concerning. Around 80% of NTS were resistant to ceftriaxone and ciprofloxacin, the first-line treatment recommended by international guidelines [26]. Encouragingly, 100% susceptibility to carbapenems and azithromycin was recorded, even if evidence of azithromycin-resistant NTS is rising in the DRC [27]. The 4-6 weeks of azithromycin exposure as oral step-down for AHD patients with invasive NTS infection, as recommended by international recommendations [26], likely contributes to rising resistance rates. Similarly, widespread use of cotrimoxazole as prophylaxis in people living with HIV may have driven high levels of resistance to this antibiotic. Further evaluation of azithromycin use and its impact on antimicrobial resistance is urgently needed to inform guidelines and ensure treatment efficacy. Around 70% of non-Salmonella Enterobacterales isolated in community-acquired IBI expressed ESBL, confirming circulation of multidrug-resistant organisms in the community [27] and increased resistance to third-generation cephalosporins. In addition, resistance to ciprofloxacin (70%), gentamicin (70%) and amikacin (50%) prevents the use of alternative treatments.

Resistance rates in hospital-acquired IBI are even higher. Ceftriaxone resistance in Enterobacterales reaches more than 93% of cases, and the usefulness of combination treatment with aminoglycosides is hindered by a resistance rate above 65% for both gentamycin and amikacin.

Although rare, carbapenem-resistant non-Salmonella Enterobacterales were isolated, both in community- (1 case) and hospital-acquired infections (4 cases). Such resistances on *K. pneumoniae* and *Enterobacter* sp. were already reported in the

DRC [28]. Confirmation and characterisation of carbapenemases by phenotypical or molecular methods are now crucial for better monitoring carbapenem resistance and informing the need for novel antibiotics' access. In their review of the literature, Lupande-Mwenebitu *et al*. noted that no studies have been conducted to screen for carbapenem resistance genes in the DRC [27], despite the emergence of isolates expressing carbapenem resistance in the country.

We also recorded a 50% non-susceptibility to oxacillin in *S. pneumoniae* and resistance rate over 80% for alternatives like cotrimoxazole and tetracycline. Additional testing is needed to estimate decreased susceptibility to benzylpenicillin, which may compromise penicillin-based treatments. Similar resistance rates have been reported in the DRC [27].

In our study, tuberculosis was present in 88% of patients, precluding meaningful assessment of TB as an independent risk factor for invasive bacterial infections or hospitalisation outcomes. The in-hospital mortality rate (24%) was higher than rates reported in TB-HIV co-infected patients in SSA [29], probably related to differences in immunosuppression levels and WHO stage. Mortality was significantly higher in both community- and hospital-acquired IBI compared to patients with negative blood cultures. The interplay between TB and invasive bacterial infections in AHD patients may reflect overlapping vulnerabilities: structural lung damage and immune dysregulation caused by TB can predispose to secondary bacterial invasion, while profound immunosuppression inherent to AHD heightens susceptibility to a broad range of pathogens. Biomarker studies in AHD populations have shown that immune dysfunction contributes to high mortality from infectious causes [30]. Other factors beyond IBI and TB likely contributed to mortality in AHD patients, including other

opportunistic infections, malnutrition, and cardiovascular disease. Longer hospital stays in suspected or confirmed hospital-acquired IBI likely increased both infection risk and mortality. Furthermore, the impact of care quality remains unclear, as antibiotic prescription practices were not assessed. No significant link was found between IBI aetiology, first-line antibiotic resistance, and mortality—possibly due to small subgroup sizes.

These findings highlight the need to reassess empirical antibiotic strategies for sepsis. While the high rate of ceftriaxone resistance in community-acquired IBI may justify carbapenem use as a first-line treatment, such an approach—particularly in non-septic shock cases—could raise costs, complicate treatment protocols, and heighten the risk of resistance selection, without clear evidence of better outcomes in AHD patients. In addition, antibiotic resistance rates described in this study only concern bloodstream infections and not other bacterial infections, where antibiotic resistance rates may be different (e.g. pneumonia). Moreover, 85% of blood cultures were negative despite signs and symptoms of sepsis, and no assumption can be made about the aetiology of the probable invasive infections. Antimicrobial stewardship (AMS) programs are needed to slow down the use of carbapenems and prevent a rise in carbapenem-resistant pathogens, whose treatment, in low-resource settings, is hindered by high costs, lack of clear regulation and national guidelines to monitor their use [31]. Since the MERINO study [32], several authors have endorsed the use of piperacillin-tazobactam as empirical and targeted treatment for ESBL-producing Enterobacterales, particularly for non-severe sepsis [33]. Clinical studies are urgent to clarify the best approach for sepsis management in terms of empirical and targeted antibiotic treatment with carbapenem-sparing strategy, according to

local epidemiology of antibiotic resistance, both in general population and vulnerable population like AHD patients.

Our study has several limitations. First, collecting accurate information on prior antibiotic use was challenging due to the absence of medical records from other health centres or hospitals, as well as the common practice of self-medication without prescriptions. Second, we abstained from analysing causes of death or conducting multivariate analyses as limited access to diagnostic tools like imaging or advanced laboratory tests might have made clinical assessments potentially unreliable. Additionally, antibiotic treatments administered were not assessed, preventing evaluation of empirical therapy appropriateness and its influence on outcomes. Collecting only a single set of blood cultures may have underestimated IBI prevalence. Two laboratories analysed the blood cultures; although the first processed only 9% of samples, positivity rates, isolated pathogens, and susceptibility profiles were comparable between laboratories. Finally, as a single-centre study, the generalizability of our findings to other settings is limited.

Nevertheless, the study has notable strengths. This is a prospective study involving a large population of AHD patients with access to high-quality microbiology, offering valuable insight into NTS susceptibility in a high-risk population, and providing important information to guide the design of future studies.

In conclusion, IBIs are common among hospitalised AHD patients and contribute to an increased risk of death. High resistance to first-line antibiotics is concerning, highlighting the need for AMR monitoring to inform therapeutic guidelines, rationalise antibiotic prescribing, and strengthen the control and prevention of hospital infections. Clinical studies are needed to define optimal sepsis management,

including empirical and targeted antibiotic treatment with carbapenem-sparing strategies, both in general and high-risk populations like AHD patients.

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Declaration of interests

The authors declare no competing interests.

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Ethical approval statement

The study was conducted in accordance with ethical principles of the Declaration of Helsinki along with good clinical practice guidelines. The study protocol was approved by the Ethics Committee of the School of Public Health of the University of Kinshasa (ESP/CE/239/2019) and the MSF Ethics Review Board (ID 1945). Written informed consent was obtained from all participants or representatives.

Authors' contributions

CL, FN, FGG, TK, FM, AK, RNB, RB and GML conceived and designed the study. CL, FN, MB, GM, FGG, AD, EB, TK, FM, and RB conducted the study. CL, and CB analysed the data. CL, CB and AC wrote the first draft of the manuscript. CL, CB, FN, AC, MB, FGG, EB, RB, GML, and PI contributed to the writing of the manuscript. All authors contributed to data interpretation. All the authors critically reviewed and approved the final version of the manuscript. All authors had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Data sharing

Epicentre and Médecins Sans Frontières (MSF) are committed to sharing and disseminating health data from its research in an open, timely, and transparent manner to promote health for populations while respecting ethical and legal obligations towards patients, research participants, and their communities. Upon publication and for as long as the ethical authorization permits it, the participant data

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REFERENCES

[1] World Health Organization. Consolidated guidelines on HIV prevention, testing, treatment, service delivery and monitoring: recommendations for a public health approach. 2021.

[2] Stelzle D, Rangaraj A, Jarvis JN, Razakasoa NH, Perrin G, Low-Beer D, et al. Prevalence of advanced HIV disease in sub-Saharan Africa: a multi-country analysis of nationally representative household surveys. Lancet Glob Heal 2025;13:e437–46. https://doi.org/10.1016/S2214-109X(24)00538-2.

[3] Huson MAM, Stolp SM, van der Poll T, Grobusch MP. Community-Acquired Bacterial Bloodstream Infections in HIV-Infected Patients: A Systematic Review. Clin Infect Dis 2014;58:79–92. https://doi.org/10.1093/CID/CIT596';

[4] Reddy EA, Shaw A V, Crump JA. Community-acquired bloodstream infections in Africa : a systematic review and meta-analysis. Lancet Infect Dis 2010;10:417–32. https://doi.org/10.1016/S1473-3099(10)70072-4.Community-acquired.

[5] Naghavi M, Vollset SE, Ikuta KS, Swetschinski LR, Gray AP, Wool EE, et al. Global burden of bacterial antimicrobial resistance 1990-2021: a systematic analysis with forecasts to 2050. Lancet (London, England) 2024;404:1199–226. https://doi.org/10.1016/S0140-6736(24)01867-1.

[6] Sartorius B, Gray AP, Davis Weaver N, Robles Aguilar G, Swetschinski LR, Ikuta KS, et al. The burden of bacterial antimicrobial resistance in the WHO African region in 2019: a cross-country systematic analysis. Lancet Glob Heal 2024;12:e201–16. https://doi.org/10.1016/S2214-109X(23)00539-9.

[7] Mayanja BN, Todd J, Hughes P, Van der Paal L, Mugisha JO, Atuhumuza E, et al. Septicaemia in a population-based HIV clinical cohort in rural Uganda,

1996-2007: incidence, aetiology, antimicrobial drug resistance and impact of antiretroviral therapy. Trop Med Int Health 2010;15:697–705. https://doi.org/10.1111/j.1365-3156.2010.02528.x.

[8] Mitiku H, Weldegebreal F, Marami D, Teklemariam Z. Nontyphoidal Salmonella bacteremia in antiretroviral therapy-naïve HIV-infected individuals at three public hospitals in Eastern Ethiopia: prevalence, antimicrobial susceptibility patterns, and associated factors. HIV/AIDS 2019;11:23. https://doi.org/10.2147/HIV.S193166.

[9] Malande OO, Nuttall J, Pillay V, Bamford C, Eley B. A ten-year review of ESBL and non-ESBL Escherichia coli bloodstream infections among children at a tertiary referral hospital in South Africa. PLoS One 2019;14:e0222675. https://doi.org/10.1371/JOURNAL.PONE.0222675.

[10] Buys H, Muloiwa R, Bamford C, Eley B. Klebsiella pneumoniae bloodstream infections at a South African children's hospital 2006-2011, a cross-sectional study. BMC Infect Dis 2016;16:1–10. https://doi.org/10.1186/S12879-016-1919-Y/TABLES/7.

[11] Jemal M, Deress T, Belachew T, Adem Y. Antimicrobial Resistance Patterns of Bacterial Isolates from Blood Culture among HIV/AIDS Patients at Felege Hiwot Referral Hospital, Northwest Ethiopia. Int J Microbiol 2020;2020. https://doi.org/10.1155/2020/8893266.

[12] Fortuin-de Smidt MC, Singh-Moodley A, Badat R, Quan V, Kularatne R, Nana T, et al. Staphylococcus aureus bacteraemia in Gauteng academic hospitals, South Africa. Int J Infect Dis 2015;30:e41–8. https://doi.org/10.1016/j.ijid.2014.10.011.

[13] Chaka W, Berger C, Huo S, Robertson V, Tachiona C, Magwenzi M, et al.

Presentation and outcome of suspected sepsis in a high-HIV burden, high antiretroviral coverage setting. Int J Infect Dis 2020;96:276. https://doi.org/10.1016/J.IJID.2020.04.004.

[14] Kenga DB, Gebretsadik T, Simbine S, Maússe FE, Charles P, Zaqueu E, et al. Community-acquired bacteremia among HIV-infected and HIV-exposed uninfected children hospitalized with fever in Mozambique. Int J Infect Dis 2021;109:99. https://doi.org/10.1016/J.IJID.2021.06.047.

[15] Musiime V, Cook A, Bakeera-Kitaka S, Vhembo T, Lutakome J, Keishanyu R, et al. Bacteremia, causative agents and antimicrobial susceptibility among HIV-1-infected children on antiretroviral therapy in Uganda and Zimbabwe. Pediatr Infect Dis J 2013;32:856–62. https://doi.org/10.1097/INF.0b013e31828c3991.

[16] Markwart R, Saito H, Harder T, Tomczyk S, Cassini A, Fleischmann-Struzek C, et al. Epidemiology and burden of sepsis acquired in hospitals and intensive care units: a systematic review and meta-analysis. Intensive Care Med 2020;46:1536–51. https://doi.org/10.1007/s00134-020-06106-2.

[17] Klatt NR, Funderburg NT, Brenchley JM. Microbial translocation, immune activation and HIV disease $watermark-text $watermark-text $watermark-text. Trends Microbiol 2013;21:6–13. https://doi.org/10.1016/j.tim.2012.09.001.

[18] Dimani BD, Founou RC, Zemtsa JR, Mbossi A, Koudoum PL, Founou LL, et al. Faecal carriage of multidrug-resistant and extended-spectrum β-lactamase-producing Enterobacterales in people living with HIV in Yaoundé, Cameroon. J Glob Antimicrob Resist 2023;35:26–34. https://doi.org/10.1016/J.JGAR.2023.07.021.

[19] Lewnard JA, Charani E, Gleason A, Hsu LY, Khan WA, Karkey A, et al. Burden of bacterial antimicrobial resistance in low-income and middle-income countries

avertible by existing interventions: an evidence review and modelling analysis. Lancet 2024;403:2439–54. https://doi.org/10.1016/S0140-6736(24)00862-6.

[20] Phoba MF, De Boeck H, Ifeka BB, Dawili J, Lunguya O, Vanhoof R, et al. Epidemic increase in Salmonella bloodstream infection in children, Bwamanda, the Democratic Republic of Congo. Eur J Clin Microbiol Infect Dis 2014;33:79–87. https://doi.org/10.1007/S10096-013-1931-8,.

[21] Falay D, Kuijpers LMF, Phoba MF, De Boeck H, Lunguya O, Vakaniaki E, et al. Microbiological, clinical and molecular findings of non-typhoidal Salmonella bloodstream infections associated with malaria, Oriental Province, Democratic Republic of the Congo. BMC Infect Dis 2016;16. https://doi.org/10.1186/S12879-016-1604-1,.

[22] Tack B, Vanaenrode J, Verbakel JY, Toelen J, Jacobs J. Invasive non-typhoidal Salmonella infections in sub-Saharan Africa: a systematic review on antimicrobial resistance and treatment. BMC Med 2020;18. https://doi.org/10.1186/S12916-020-01652-4.

[23] Gilchrist JJ, MacLennan CA. Invasive Nontyphoidal Salmonella Disease in Africa . EcoSal Plus 2019;8. https://doi.org/10.1128/ecosalplus.esp-0007-2018.

[24] Miller WR, Arias CA. ESKAPE pathogens: antimicrobial resistance, epidemiology, clinical impact and therapeutics. Nat Rev Microbiol 2024;22:598–616. https://doi.org/10.1038/S41579-024-01054-W,.

[25] Allegranzi B, Nejad SB, Combescure C, Graafmans W, Attar H, Donaldson L, et al. Burden of endemic health-care-associated infection in developing countries: systematic review and meta-analysis. Lancet 2011;377:228–41. https://doi.org/10.1016/S0140-6736(10)61458-4.

[26] National Institutes of Health. Guidelines for the Prevention and Treatment of

Opportunistic Infections in Adults and Adolescents With HIV | Bacterial Enteric Infections n.d. https://clinicalinfo.hiv.gov/en/guidelines/hiv-clinical-guidelines-adult-and-adolescent-opportunistic-infections/bacterial-enteric?view=full (accessed February 22, 2025).

[27] Lupande-Mwenebitu D, Baron SA, Nabti LZ, Lunguya-Metila O, Lavigne JP, Rolain JM, et al. Current status of resistance to antibiotics in the Democratic Republic of the Congo: A review. J Glob Antimicrob Resist 2020;22:818–25. https://doi.org/10.1016/J.JGAR.2020.07.008.

[28] Irenge LM, Kabego L, Kinunu FB, Itongwa M, Mitangala PN, Gala JL, et al. Antimicrobial resistance of bacteria isolated from patients with bloodstream infections at a tertiary care hospital in the Democratic Republic of the Congo. South African Med J 2015;105:752–5. https://doi.org/10.7196/SAMJnew.7937.

[29] Moges S, Lajore BA. Mortality and associated factors among patients with TB-HIV co-infection in Ethiopia: a systematic review and meta-analysis. BMC Infect Dis 2024;24. https://doi.org/10.1186/s12879-024-09683-5.

[30] Riitho V, Connon R, Gwela A, Namusanje J, Nhema R, Siika A, et al. Biomarkers of mortality in adults and adolescents with advanced HIV in sub-Saharan Africa. Nat Commun 2024 151 2024;15:5492-. https://doi.org/10.1038/s41467-024-49317-7.

[31] Carrara E, Savoldi A, Piddock LJV, Franceschi F, Ellis S, Sharland M, et al. Clinical management of severe infections caused by carbapenem-resistant gram-negative bacteria: a worldwide cross-sectional survey addressing the use of antibiotic combinations. Clin Microbiol Infect 2022;28:66–72. https://doi.org/10.1016/J.CMI.2021.05.002.

[32] Harris PNA, Tambyah PA, Lye DC, Mo Y, Lee TH, Yilmaz M, et al. Effect of

Piperacillin-Tazobactam vs Meropenem on 30-Day Mortality for Patients With E coli or Klebsiella pneumoniae Bloodstream Infection and Ceftriaxone Resistance: A Randomized Clinical Trial. JAMA 2018;320:984. https://doi.org/10.1001/JAMA.2018.12163.

[33] Gatti M, Cojutti PG, Pea F. Piperacillin-tazobactam vs. carbapenems for treating hospitalized patients with ESBL-producing Enterobacterales bloodstream infections: A systematic review and meta-analysis. J Glob Antimicrob Resist 2024;39:27–36. https://doi.org/10.1016/J.JGAR.2024.08.002.

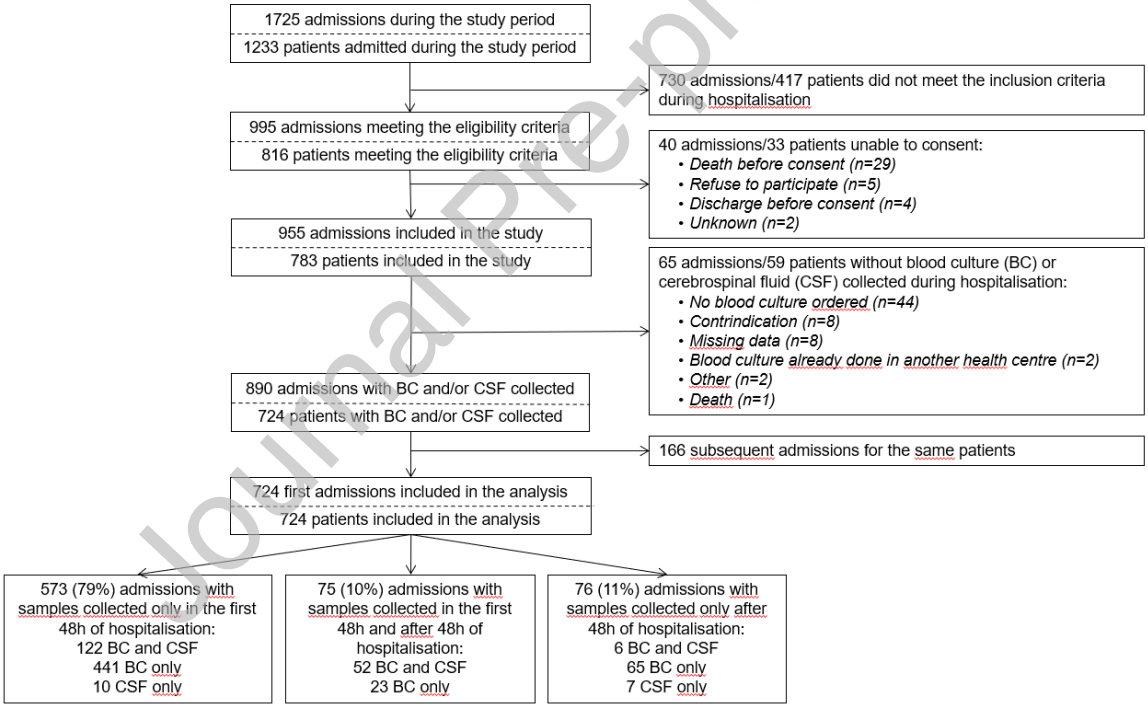

Figure 1. Study flowchart for inclusion and exclusion of admissions and corresponding patients in the study. Patients could be enrolled more than once if they were readmitted to the study site during the study period.

Table 1. Baseline characteristics of the study population, Kinshasa, 2021-2022[a, b]

Patient characteristics	All participants N=724	No bacteraemia N=596	Community-acquired IBI [c] N=108	Hospital-acquired IBI [c] N=22
Female	446 (61.6)	369 (62.0)	61 (56.5)	18 (81.9)
Age, years, median [IQR]	40 [31; 49]	39 [30; 48]	44 [32.5; 50]	41.5 [31; 52]
<15	47 (6.5)	39 (6.6)	7 (6.5)	1 (4.6)
[15 - 25]	83 (11.5)	69 (11.6)	12 (11.2)	3 (13.7)
[26 - 50]	448 (61.9)	375 (63)	63 (58.4)	11 (50.0)
>50	146 (20.2)	113 (19)	26 (24.1)	7 (31.9)
Symptoms on admission				
General symptoms[d]	677 (93.6)	560 (94)	99 (91.7)	20 (91.0)
Respiratory	473 (65.4)	385 (64.6)	76 (70.4)	12 (54.6)
Neurologic	412 (57.0)	322 (54.1)	72 (66.7)	19 (86.4)
Gastrointestinal	243 (33.6)	199 (33.4)	38 (35.2)	6 (27.3)
Hypotension/shock	211 (29.2)	166 (27.9)	42 (38.9)	3 (13.7)
Cutaneous	54 (7.5)	40 (6.7)	14 (13.0)	2 (9.1)
Other[e]	128 (17.7)	103 (17.3)	22 (20.4)	4 (18.2)
Intensive care admission	247 (34.2)	194 (32.6)	40 (37.1)	13 (59.1)
Viral load on admission, copies/mm^3				
Undetectable (<40)	144 (20.0)	128 (21.6)	15 (13.9)	2 (9.1)
≥40 and <1000	107 (14.8)	91 (15.3)	12 (11.2)	4 (18.2)
≥1000	372 (51.5)	289 (48.6)	69 (63.9)	15 (68.2)
Not analysed	100 (13.9)	87 (14.7)	12 (11.2)	1 (4.6)
CD4, cells/mm^3				
≤50	157 (22.1)	123 (21.1)	29 (27.4)	6 (27.3)
51-99	124 (17.5)	97 (16.6)	18 (17.0)	9 (40.9)
100-199	175 (24.7)	145 (24.9)	28 (26.4)	2 (9.1)
≥200	253 (35.7)	218 (36.9)	31 (29.2)	5 (22.8)
Nutritional care	350 (49.5)	293 (50.3)	46 (42.6)	13 (59.1)
Laboratory findings				
Severe anemia (Hb < 6g/dL)	80 (11.1)	54 (9.1)	22 (20.4)	5 (22.8)
Hypoglycemia (< 80 mg/dL)	131 (18.1)	101 (17)	27 (25)	3 (13.7)
Leukocytosis (>10000/mm^3)	196 (27.1)	155 (26.1)	35 (32.5)	6 (27.3)
Leukopenia (<4000/mm^3)	158 (21.8)	134 (22.5)	20 (18.5)	4 (18.2)
Opportunistic infections				
Tuberculosis	636 (88.1)	519 (87.3)	97 (89.9)	21 (95.5)
Cryptococcosis	64 (8.9)	56 (9.4)	5 (4.7)	3 (13.7)
Suspicion of Pneumocystis pneumonia	200 (27.7)	153 (25.7)	37 (34.3)	10 (45.5)
Suspicion of cerebral toxoplasmosis	167 (23.1)	129 (21.7)	29 (26.9)	9 (41)
Kaposi's sarcoma	8 (1.2)	6 (1.1)	1 (1)	1 (4.6)
Malaria	101 (14)	85 (14.3)	12 (11.2)	4 (18.2)

IBI: invasive bacterial infection

[a] Values are median [IQR] or n (%)

[b] Categories may not sum to total because of missing data.

[c] 2 patients had both community-acquired IBI and hospital-acquired IBI

[d] General symptoms include fever, sweats, loss of appetite, fatigue, and weight loss

[e] "Other" includes hematologic, psychiatric, cardiovascular, genital, nephrologic, urologic, hepatic, pancreatic, and endocrine symptoms.

1.1.1 Table 2. Organisms identified in community-acquired and hospital-acquired invasive bacterial infections (IBI), Kinshasa, 2021-2022

Organisms	Pathogens involved in community-acquired IBI[a], n=116	Pathogens involved in hospital-acquired IBI[b], n=24
Non-typhoidal *Salmonella*	53 (45.7)	2 (8.4)
Escherichia coli	22 (19)	1 (4.2)
Streptococcus pneumoniae	20[c] (17.3)	0 (0)
Pseudomonas aeruginosa	6 (5.2)	1 (4.2)
Klebsiella pneumoniae	4 (3.5)	10 (41.7)
Staphylococcus aureus	3 (2.6)	3 (12.5)
Gram negative bacilli	2 (1.8)	0 (0)
Haemophilus influenzae	2 (1.8)	0 (0)
Acinetobacter baumannii	1 (0.9)	3 (12.5)
Streptococcus group A	1 (0.9)	0 (0)
Klebsiella oxytoca	1 (0.9)	0 (0)
Burkholderia cepacia	1 (0.9)	0 (0)
Enterobacter cloacae	0 (0)	1 (4.2)
Enterococcus sp.	0 (0)	1 (4.2)
Proteus mirabilis	0 (0)	2 (8.4)

[a] *8 co-infections: 2 non typhoidal Salmonella + E. coli ; S. pneumoniae + non typhoidal Salmonella ; K. pneumoniae + non typhoidal Salmonella ; S. pneumoniae + E. coli; P. aeruginosa + H. influenzae; K .pneumoniae + A. baumannii; S. aureus + Streptococcus group A*

[b] 2 co-infections: *K. pneumoniae + A. baumannii; K. pneumoniae +* non-typhoidal *Salmonella*

[c] including 1 *S. pneumoniae* isolated in both blood and CSF culture of the same patient

1.1.1 Table 3. Antibiotic resistance of main pathogens isolated from community-acquired and hospital-acquired invasive bacterial infection (IBI), Kinshasa, 2021-2022

	Community-acquired IBI, n (%)		Hospital-acquired IBI, n (%)
	non-typhoidal *Salmonella* n=53	Other Enterobacterales[b] n=27	Enterobacterales[c] n=16
Ampicillin	48 (90.6)	27 (100)	14 (87.5)
Amoxicillin + clavulanate	33 (71.8)	26 (96.3)	12 (75)
Piperacillin + Tazobactam	5 (9.5)	9 (33.4)	6 (42.9)
Ceftriaxone	44 (83.1)	20 (74.1)	14 (93.4)
Meropenem	0 (0)	1 (3.8)	4 (28.6)
Gentamycin	34 (77.3)	18 (66.7)	11 (73.4)
Amikacin	33 (75)	12 (44.5)	10 (66.7)
Tobramycin	36 (81.9)	21 (80.8)	11 (73.4)
Ciprofloxacin/Pefloxacin[a]	41 (78.9)	19 (73.1)	8 (53.4)

Cotrimoxazole	46 (86.8)	25 (92.6)	14 (93.4)
Azithromycin	0 (0)	0 (0)	0 (0)
ESBL confirmed	44 (83.1)	18 (66.7)	10 (62.5)

ESBL: Extended spectrum beta-lactamases

[a] Pefloxacin tested for Salmonella sp.; Ciprofloxacin tested for other Enterobacterales as per EUCAST V12

[b] including *E. coli*, *Klebsiella* sp.

[c] including *E. coli*, *Klebsiella* sp., *Proteus mirabilis*, *Enterobacter cloacae*

Table 4. Hospitalisation outcomes and length of stay, *Kinshasa, 2021-2022* [a, b]

	All participants n=724	No bacteraemia n=596	Community-acquired IBI n=108[b]	Hospital-acquired IBI n=22[b]
Discharge status				
Discharged	483 (66.6)	411 (68.8)	63 (58.4)	10 (45.5)
Death	175 (24.2)	126 (21.1)	38 (35.2)	12 (54.6)
Transferred to another hospital	65 (9)	58 (9.7)	7 (6.5)	0 (0)
Left against medical decision	3 (0.5)	3 (0.6)	0 (0)	0 (0)
Length of stay, days	5 [3-9]	5 [3-8]	6.5 [3-12]	23.5 [17-38]

IBI: Invasive bacterial infections

[a] Values are median [IQR] or n (%)

[b] 2 patients had both community-acquired IBI and hospital-acquired IBI

Declaration of Interest Statement

☒ The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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