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Original Article

Microbiological Yield, Factors Associated with Culture Positivity, and Coverage of Empirical Antibiotic Therapy in Intracranial Suppurative Infections: A Ten-Year Ivorian Cohort

Rendement Microbiologique, Facteurs Associés à la Positivité des Cultures et Couverture de l'Antibiothérapie Empirique dans les Infections Suppurées Intracrâniennes : Une Cohorte Ivoirienne sur Dix Ans

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ABSTRACT

Introduction. Microbiological yield in intracranial suppurative infections (ISI) is low in resource-limited settings, where empirical antibiotics often precede sampling. We quantified this yield, sought factors associated with culture positivity, and assessed the adequacy of empirical therapy against isolated organisms**. Methods.** Secondary analysis of a single-centre cohort of 150 episodes (148 patients; 2016–2026, Bouaké Teaching Hospital, Côte d'Ivoire). Factors associated with positivity were examined among sampled patients (n = 116; 50 events) using Firth penalized logistic regression with internal bootstrap validation. Adequacy was assessed both by recorded in-vitro activity against the antibiogram and by a prespecified expert classification of expected spectrum-based coverage. **Results.** Pus was sampled in 77.3 % of episodes; cultures were positive in 50/116 sampled patients (43.1 %; 95 % CI 34.5–52.2) and in 33.3 % of the cohort. Gram-positive cocci predominated (82 %): streptococci 42 %, Staphylococcus aureus 34 %. No prespecified factor was associated with positivity; only continuous age was (aOR 1.23 per decade; 95 % CI 1.03–1.48), without usable discrimination (corrected AUC 0.58–0.61). Initial therapy included at least one in-vitro-active agent in 34/44 episodes with an antibiogram (77.3 %); expected spectrum-based coverage was 74–76 % but showed no individual concordance with the antibiogram (kappa −0.04). The main gap concerned S. aureus (53 % covered). Two-thirds of the cohort (100/150) were treated without an exploitable isolate; 93 % of regimens contained a WHO "Watch" antibiotic. **Conclusion.** Routine variables did not identify culture-positive episodes, arguing for deep sampling whenever feasible rather than clinical selection. Empirical therapy was broadly adequate, antistaphylococcal coverage being the one easily correctable weakness. Strengthening microbiological capacity and structured stewardship is the priority.

RÉSUMÉ

Introduction. Le rendement microbiologique des suppurations intracrâniennes (SIC) est faible dans les contextes de ressources limitées, où l'antibiothérapie probabiliste précède souvent le prélèvement. Nous avons quantifié ce rendement, recherché les facteurs associés à une culture positive et évalué l'adéquation de l'antibiothérapie probabiliste vis-à-vis des germes isolés**. Méthodes.** Analyse secondaire d'une cohorte monocentrique de 150 observations (148 patients ; 2016–2026, CHU de Bouaké). Les facteurs associés à la positivité ont été recherchés parmi les patients prélevés (n = 116 ; 50 événements) par régression logistique pénalisée de Firth, avec validation interne par bootstrap. L'adéquation a été évaluée par l'adéquation enregistrée vis-à-vis de l'antibiogramme et par une classification experte préspécifiée de la couverture attendue selon le spectre. **Résultats.** Un prélèvement était réalisé dans 77,3 % des cas ; les cultures étaient positives chez 50/116 patients prélevés (43,1 % ; IC95 % 34,5–52,2). Les cocci à Gram positif prédominaient (82 %) : streptocoques 42 %, Staphylococcus aureus 34 %. Aucun facteur préspécifié n'était associé à la positivité ; seul l'âge en continu l'était (ORa 1,23 par décennie ; IC95 % 1,03–1,48), sans discrimination exploitable (AUC corrigée 0,58–0,61). L'adéquation vis-à-vis de l'antibiogramme était de 77,3 % (34/44) ; la couverture attendue selon le spectre était de 74–76 % mais sans concordance individuelle (kappa −0,04). La principale faille concernait S. aureus (couvert dans 53 %). Deux tiers de la cohorte (100/150) ont été traités sans isolat exploitable ; 93 % des schémas comportaient un antibiotique « Watch » de l'OMS. **Conclusion.** Les variables de routine ne permettaient pas d'identifier les épisodes à culture positive, ce qui plaide pour un prélèvement profond chaque fois qu'il est techniquement possible plutôt que pour une sélection clinique. L'antibiothérapie probabiliste était globalement adéquate, la couverture antistaphylococcique constituant le seul point faible aisément corrigeable. Le renforcement des capacités microbiologiques et des programmes structurés de bon usage des antibiotiques apparaît prioritaire.

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HIGHLIGHTS FOR READERS IN A HURRY

What is known about the subject. In resource-limited settings, where empirical antibiotics frequently precede sampling, the microbiological yield in intracranial suppurative infections (ISI) remains low.

The question addressed in this study. We quantified this yield, identified factors associated with culture positivity, and evaluated the adequacy of empirical antibiotic therapy against isolated organisms through a secondary analysis of a single-centre cohort comprising 150 episodes (148 patients; 2016–2026, Bouaké Teaching Hospital, Côte d'Ivoire).

What this study adds to our knowledge. Despite frequent pus sampling, the microbiological yield in intracranial suppurative infections remains low (dominated by Gram-positive cocci), highlighting widespread empirical use of WHO "Watch" antibiotics in the absence of an exploitable isolate.

Implications for practice. Routine clinical variables fail to predict culture positivity, making systematic deep sampling, optimized empirical anti-staphylococcal coverage, and enhanced microbiological capacity essential priorities.

INTRODUCTION

Intracranial suppurative infections (ISI) — brain abscess, subdural and extradural empyema, and ventriculitis — are neurosurgical emergencies managed by surgical drainage combined with prolonged antibiotic therapy [1–3]. Identifying the causative organism and its susceptibility optimises treatment: it allows targeting, spectrum narrowing, duration adjustment and reduced selection pressure, and their management has been extensively reviewed [4–12]. In resource-limited settings, microbiological yield nonetheless remains low, often below that reported in high-income countries.

This sits within the global challenge of antimicrobial resistance (AMR), now a leading public-health threat [13]. The World Health Organization (WHO) Global Action Plan and the GLASS surveillance system place antibiotic stewardship at the centre of the response, and the AWaRe (Access, Watch, Reserve) classification offers a practical framework to rationalise prescribing [14–18]. In sub-Saharan African hospitals, scarce microbiological documentation favours broad, prolonged use of “Watch” antibiotics, sustaining selection pressure [19,20]. Neurosurgical infections, where treatment is necessarily early and prolonged, are a particularly sensitive setting for stewardship.

Several mechanisms drive the low culture yield. Empirical therapy is frequently started before any sampling. Pre-analytical constraints — transport delays and conditions, inconsistent availability of anaerobic media — degrade sensitivity; anaerobes and fastidious organisms are under-detected, and molecular amplification is rarely accessible [21]. Antibiotic therapy therefore remains largely empirical, and its appropriateness can be verified in only a minority of patients.

This raises two practical questions, rarely addressed together in African series. Is culture positivity predictable — could clinical or lesional factors guide the sampling strategy? And did empirical therapy actually cover the isolated organisms — which determines the room for stewardship improvement? Using a single-centre Ivorian cohort of 150 episodes, this analytical study aimed to (i) describe microbiological yield and the spectrum of isolates; (ii) identify factors associated with culture positivity by logistic regression; and (iii) assess the adequacy of empirical therapy against isolated organisms, both by recorded in-vitro activity and by a prespecified expert classification of expected spectrum-based coverage. To our knowledge, no West African study has simultaneously evaluated these three dimensions in intracranial suppuration.

METHODOLOGY

Design, setting and unit of analysis

This is a secondary, microbiological and pharmacotherapeutic analysis of a single-centre observational cohort of intracranial suppurative infections managed at Bouaké Teaching Hospital (Côte d’Ivoire) between 2016 and 2026. Data collection was retrospective through 2024, then prospective in 2025–2026. Reporting follows the STROBE recommendations; the completed checklist is provided as supplementary material (Supplement 1) [22]. The unit of analysis was the episode of intracranial suppuration. The 150 observations corresponded to 148 patients; two duplicate pairs being retained independently after verification. The near-complete independence of observations (≈99 %) was judged compatible with the analyses; a sensitivity analysis restricted to the first episode per patient did not change the conclusions.

Inclusion and exclusion. All clinically and radiologically documented ISI episodes were included: brain abscess (ring-enhancing parenchymal collection), subdural or extradural empyema, ventriculitis (ependymal enhancement or intraventricular sediment) and extracranial scalp suppuration with intracranial extension or risk thereof. Records with insufficient clinical or imaging data, and unusable duplicates, were excluded. Postoperative infections and documented recurrences were included. The flow diagram is shown in Figure 1.

Microbiological data and definitions

When surgery was performed, pus was sampled intra-operatively; diagnostic punctures or wound samples were less frequent. Samples were sent for direct examination, Gram staining, aerobic and — when available — anaerobic cultures, and antibiogram of isolates. Molecular amplification was unavailable during the study period. Pre-analytical conditions (pus volume, medium and transport delay, availability of anaerobic culture, pre- vs post-antibiotic sampling), the identification method and the antibiogram interpretation standard followed laboratory procedures and were not prospectively standardised; this heterogeneity is acknowledged as a limitation [21]. For each observation the microbiological result was classified into three mutually exclusive categories — “not sought”, “sterile” and “positive” — to use the appropriate denominator. Yield was reported against two denominators: sampled patients (conditional

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positivity) and the whole cohort (overall positivity). Coagulase-negative staphylococci, potential contaminants, were interpreted cautiously.

Assessment of empirical coverage

Two complementary approaches were used. The first, recorded adequacy against the antibiogram, relied on the charted variable for patients with an antibiogram: was therapy active against the isolate per laboratory breakpoints? The second, expected spectrum-based coverage, applied to each isolate a prespecified expert classification based on intrinsic spectrum, independent of the antibiogram. Coverage rules were fixed a priori from usual spectra and current recommendations (full rules in Supplement 3). Briefly, Streptococcus spp. was considered covered by a Gram-positive-active third-generation cephalosporin, amoxicillin–clavulanate, vancomycin or meropenem (ceftazidime excluded); S. aureus by a reliable antistaphylococcal agent (cloxacillin for methicillin-susceptible strains, vancomycin, meropenem or amoxicillin–clavulanate), a third-generation cephalosporin alone being insufficient; coagulase-negative staphylococci by vancomycin only; Pseudomonas aeruginosa by an antipseudomonal agent; and Enterobacterales and anaerobes by their usual agents [23,24]. These rules are a theoretical approximation that does not prejudge acquired resistance, does not replace the antibiogram, and ignores central-nervous-system penetration, dosing and intralesional diffusion [25]. Agents were also classified by WHO AWaRe category [15]. Two sensitivity analyses assumed all S. aureus methicillin-susceptible (MSSA) then methicillin-resistant (MRSA).

Statistical analysis

Categorical variables are described by counts and percentages, quantitative variables by median and interquartile range (IQR); 95 % CIs for proportions used the Wilson method [26]. Missing-data proportions are reported per variable (Supplementary Table S2); model predictors were complete among sampled patients, and a complete-case analysis was used without imputation. Factors associated with culture positivity were examined among sampled patients ($n = 116$; 50 events): bivariate comparisons by Fisher exact or χ^2 and Mann–Whitney tests, then a prespecified multivariable model with five clinically motivated covariates (community-acquired origin, intra-operative sampling, immunosuppression, age ≥ 50 years, referral status as a proxy for prior antibiotics), chosen before result analysis to respect ten events per variable [27]. Firth penalized logistic regression was the primary analysis [28,29]; absence of collinearity was checked. Discrimination was assessed by AUC and internal validity by bootstrap (2000 resamples) with optimism estimation and an optimism-corrected AUC (Harrell method) [30,31]; Tjur R^2 is reported. Sensitivity analyses (intra-operative samples only; excluding wound samples) were performed. As the model was explanatory and not intended for clinical prediction, calibration indices and decision-curve analysis were not deemed essential [32,33]. Significance was set at $p < 0.05$. Analyses used Python 3.11 (pandas, SciPy, statsmodels) and a study-specific Firth implementation; the full model equation is in Supplement 4. No a priori sample-size calculation was performed; size was set by available recruitment, the 116 sampled patients providing 50 events (≈10 per candidate covariate). Several potentially relevant variables — pre-hospital antibiotics and self-medication, precise sampling-to-culture delay, transport conditions, prolonged corticosteroids, prior surgery — were unavailable or too incomplete for the multivariable analysis; this is discussed as a major limitation.

Ethics

The study was conducted after anonymisation, in accordance with the Declaration of Helsinki and respecting data confidentiality, and was approved by the institutional ethics committee of Bouaké Teaching Hospital (approval no. CEI/CHU-BKE/104/2026).

RESULTS

Overall microbiological yield

Pus was sampled in 116/150 patients (77.3 %). Among sampled patients, 50 cultures were positive (43.1 %; 95 % CI 34.5–52.2) and 66 negative (56.9 %); cohort-wide positive yield was 33.3 % (95 % CI 26.3–41.2). Sampling was most often intra-operative (105/116), less often a diagnostic puncture (9) or a wound sample (2). An antibiogram was obtained for 44 of 50 isolates. The microbiological pathway is summarised in Figure 1. Surgery — the sampling occasion — occurred a median of 2 days (IQR 1–3) after admission, hence after empirical therapy had started in nearly all cases. The precise sampling-to-culture delay, transport conditions and pre-/post-antibiotic status of samples were not usably documented. Blood cultures were obtained in only 39 patients (26 %), positive in 4, and cerebrospinal fluid analysis in only 10.

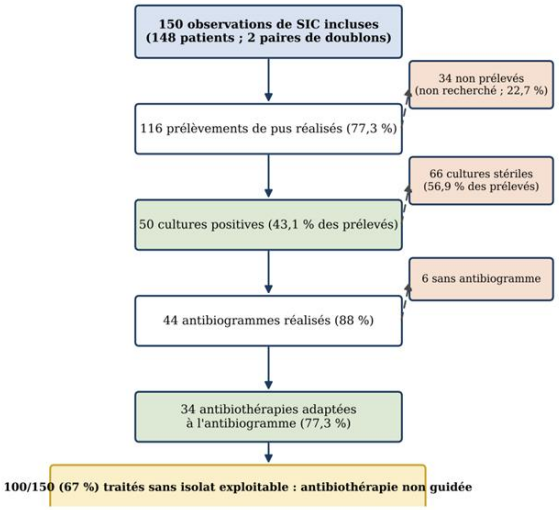

Figure 1: Flow diagram of the microbiological pathway (yield and documentation). ISI, intracranial suppurative infection

Microbiological spectrum

Gram-positive cocci strongly predominated (41/50; 82 %), Gram-negative bacilli representing 7/50 (14 %) and polymicrobial samples 2/50 (4 %). Streptococci and

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S. aureus alone made up more than three-quarters of isolates (Figure 2): Streptococcus spp. 42 %, S. aureus 34 %, Gram-negative bacilli (Enterobacterales) 8 %, coagulase-negative staphylococci 6 %, Pseudomonas aeruginosa 6 % and polymicrobial flora 4 %.

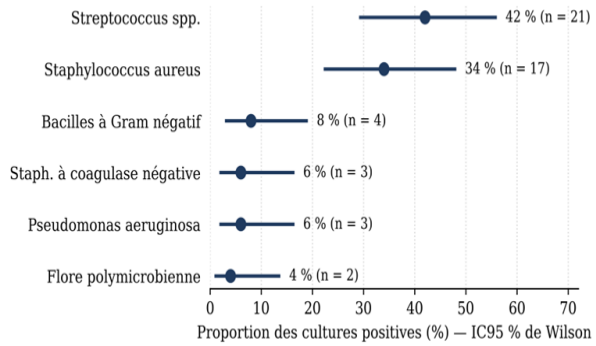

Figure 2: Distribution of isolated micro-organisms (n = 50) with 95 % Wilson confidence intervals

Factors associated with culture positivity

No clinical, lesional or epidemiological factor was significantly associated with positivity in bivariate analysis. Community-acquired origin tended toward lower yield (37 % vs 53 % for post-traumatic/postoperative forms; OR 0.51; p = 0.120) and older age toward higher yield (median 37 vs 23 years; p = 0.059), without reaching. Sample type, suppuration type, immunosuppression, admission delay, lesion size and referral status were not associated with positivity. In the Firth multivariable model, no factor reached significance: community-acquired origin (aOR 0.51; 95 % CI 0.23–1.12; p = 0.093) and age ≥ 50 years (aOR 2.21; 95 % CI 0.90–5.47; p = 0.085) only suggested a trend (Figure 3). The model discriminated poorly (apparent AUC 0.64; bootstrap-corrected 0.58; Tjur R^2 0.06; Brier 0.230). Although not intended for prediction, calibration is reported for transparency: apparent slope 1.02 (intercept 0.00), falling to 0.63 after optimism correction — expected overfitting for a model without discriminative signal. Sensitivity analyses (intra-operative only, n = 105; excluding wound samples, n = 114) gave comparable estimates; an exploratory signal for age ≥ 50 years appeared in the intra-operative subgroup (aOR 2.66; 95 % CI 1.04–6.77), to be read cautiously given no multiple-testing correction. In short, culture positivity was not predictable from routine clinical variables.

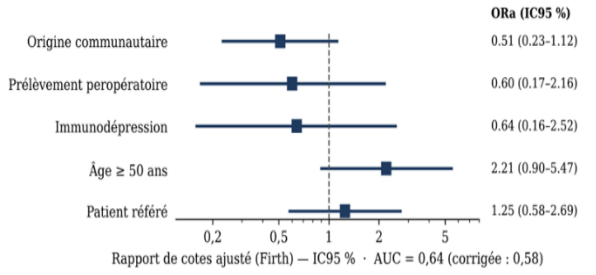

Figure 3: Adjusted odds ratios (Firth regression) for factors associated with a positive culture, with 95 % CIs

Empirical therapy and coverage of isolates

All patients received empirical therapy — triple in 56 %, dual in 44 % — for a median of 7 weeks (IQR 6–7). Regimens most often combined a third-generation cephalosporin and metronidazole, with variable vancomycin, cloxacillin, gentamicin or a carbapenem (Table I). Anaerobic coverage was near-universal (metronidazole or a carbapenem in 100 %). A dedicated antistaphylococcal agent (cloxacillin or vancomycin) was present in only 77/150 regimens (51 %) and an antipseudomonal agent in 56/150 (37 %). Under WHO AWaRe, 139/150 regimens (93 %) contained at least one "Watch" antibiotic; no "Reserve" agent was used.

Table I: Empirical antibiotic regimens and WHO AWaRe category (N = 150)

Regimen	N	%	AWaRe
Ceftazidime + vancomycin + metronidazole	32	21	Watch
Ceftriaxone + metronidazole	27	18	Watch
Ceftriaxone + vancomycin + metronidazole	18	12	Watch
Cloxacillin + ceftriaxone + metronidazole	16	11	Watch
Ceftriaxone + metronidazole + gentamicin	15	10	Watch
Amoxicillin–clavulanate + metronidazole	11	7	Access
Meropenem + vancomycin	11	7	Watch
Ceftazidime + metronidazole	10	7	Watch
Cefotaxime + metronidazole	7	5	Watch
Ceftriaxone + ciprofloxacin + metronidazole	3	2	Watch

Triple therapy 84 (56 %); dual therapy 66 (44 %). Median duration 7 weeks (IQR 6–7). AWaRe: highest category present in the regimen. Antistaphylococcal coverage 51 %; antipseudomonal 37 %; anaerobic ≈100 %. — Schémas d'antibiothérapie probabiliste (WHO AWaRe).

Among the 44 episodes with an antibiogram, initial therapy included at least one in-vitro-active agent in 34 (77.3 %; 95 % CI 63.0–87.2) and none in 10 (22.7 %). Expected spectrum-based coverage gave a similar magnitude: 38/50 isolates covered (76 %; 95 % CI 63–86) in the MSSA scenario and 37/50 (74 %) in the MRSA scenario (Table II). This closeness of overall percentages, however, reflected no individual concordance: episode-by-episode agreement was 63.6 % (28/44) but Cohen kappa was −0.04 (bootstrap 95 % CI −0.28 to 0.27) — null beyond chance, with eight discordances in each direction (Table III). The two measures thus evaluate distinct objects — documented in-vitro activity versus theoretical expected spectrum — and are not interchangeable. Expected coverage varied markedly by organism (Table II): high for streptococci (86 %), Enterobacterales and coagulase-negative staphylococci, but clearly insufficient for S. aureus (9/17; 53 % [47 % under MRSA]).

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Table II: Expected coverage of empirical therapy by organism (prespecified expert classification)

Isolate	N	Covered (MSSA)	% MSSA	% MRSA
Streptococcus spp.	21	18	86	86
Staphylococcus aureus	17	9	53	47
Gram-negative bacilli	4	4	100	100
Coagulase-negative staphylococci	3	3	100	100
Pseudomonas aeruginosa	3	2	67	67
Polymicrobial flora	2	2	100	100
Overall	**50**	**38**	**76**	**74**

MSSA/MRSA: scenarios assuming all S. aureus methicillin-susceptible or -resistant. Coverage defined by presence of ≥1 spectrum-appropriate agent active on the isolate (Supplement 3). Theoretical approximation, distinct from adequacy against the antibiogram (34/44; 77.3 %). — Couverture attendue selon le spectre.

Table III: Concordance between expected spectrum-based coverage and documented in-vitro activity (n = 44 episodes with an antibiogram)

	Active in vitro	Not active in vitro	Total
Covered by spectrum	26	8	34
Not covered by spectrum	8	2	10
Total	**34**	**10**	**44**

Crude agreement 63.6 %; Cohen kappa −0.04 (bootstrap 95 % CI −0.28 to 0.27), i.e. null beyond chance. In-vitro activity and theoretical coverage must be reported separately; neither validates the other. — Concordance grille théorique / antibiogramme.

This gap arose from the absence of an antistaphylococcal agent in eight regimens given to S. aureus carriers (usually ceftriaxone + metronidazole). One Pseudomonas case likewise received a regimen without an antipseudomonal agent. Finally, adequacy could be assessed only in a minority: the 66 negative cultures and 34 unsampled patients — 100/150 (67 %) of the cohort — received therapy that, lacking an isolate, could never be microbiologically guided.

Supplementary analyses: selection, period and microbiological sensitivity

Sampled vs unsampled. The 34 unsampled episodes differed from the 116 sampled ones on management, not severity: more often brain abscesses (85 % vs 54 %; p = 0.001), less often empyemas (12 % vs 34 %; p = 0.010) and far less often surgical (59 % vs 91 %; p < 0.001), with no difference in age, sex, GCS, admission delay or referral (Supplementary Table S7). Sampling was therefore driven by the operative indication rather than clinical selection: positivity among sampled episodes cannot be extrapolated unreservedly to medically treated ones. Temporal analysis. Practices showed no significant change across periods (Supplementary Table S8): sampling rate 80 %→72 % (p = 0.619) and positivity 38 %→48 % (p = 0.491), supporting broadly stable practice without formally excluding a period effect. Microbiological sensitivity. Excluding the three coagulase-negative staphylococci (possible contaminants) lowered positivity from 43.1 % to 40.5 % (47/116) and expected coverage from 76 % to 74 %: conclusions unchanged. The two polymicrobial cultures were not documented species-by-species; their classification as "covered" rests on a strong assumption and remains uncertain.

DISCUSSION

Principal findings

This study yields four main findings. First, pus was sampled in over three-quarters of episodes, yet cultures were positive in only 43.1 % of sampled episodes — an overall documentation of one-third of the cohort. Second, routine clinical variables did not usefully identify culture-positive episodes: only age, modelled continuously, was associated with positivity, without usable discrimination. Third, initial therapy included an in-vitro-active agent in about three-quarters of evaluable episodes, S. aureus coverage being far less consistent; the theoretical spectrum grid, though close on average, was discordant with the antibiogram individually. Fourth, two-thirds of episodes had no exploitable isolate, while nearly all regimens contained a "Watch" antibiotic. Together, these results shift the problem: the main bottleneck is not the choice of empirical therapy but microbiological documentation. The greatest room for improvement therefore lies in systematising deep sampling, improving the pre-analytical circuit and re-evaluating treatment early — that is, in diagnostic stewardship — rather than in broadening the initial spectrum.

Microbiological yield: comparison and mechanisms

The observed yield matches many ISI series, where culture positivity rarely exceeds 40–60 % [5,6,34,35]. This apparent proximity to high-income cohorts must be read cautiously, as the mechanisms of negative cultures probably differ: in high-income settings they stem chiefly from prior antibiotics and fastidious organisms, whereas in resource-limited settings transport delays, irregular anaerobic media and the absence of molecular methods add to these. In our series pus was usually obtained in theatre, after empirical therapy had begun, mechanically lowering the yield of conventional cultures. African series are heterogeneous and often do not report positivity — from 4 % (2/49) in one Nigerian series to higher rates elsewhere — while high-income cohorts documenting pus culture reach up to 60 % [5,36] (Supplementary Table S9). Other mechanisms likely contribute to negative cultures: reduced inoculum under antibiotics, hard-to-culture anaerobes, fastidious organisms and possible low-metabolic biofilms that escape conventional culture and favour recurrence. The predominance of streptococci and S. aureus is consistent with the literature [1,23] and with the frequency of ENT and dental portals of entry in our cohort. A fraction of negative cultures may reflect agents not sought by usual bacterial cultures — mycobacteria (relevant in a high-tuberculosis-burden setting), Nocardia, filamentous fungi or yeasts, even parasites in immunocompromised patients; mycobacterial and fungal cultures were not routine, a concrete avenue for improving the aetiological work-up.

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No clinically discriminating factor: implications

The most operational result is the absence of a clinically discriminating factor: neither suppuration type, sampling mode, immunosuppression, delay nor lesion size anticipated a positive culture. Continuous age was associated with positivity but with a modest effect size and no useful discrimination (corrected AUC 0.61): a statistical association is not a selection criterion. Absence of predictors does not mean positivity is random — it suggests the main determinants are pre-analytical (time to culture, prior antibiotic exposure, sample quality and transport, anaerobic-culture availability) rather than clinical. These were not directly measured — a limitation — but are the most plausible targets. The practical consequence is clear: there is no clinical subgroup in which to forgo sampling, nor one where it would be especially high-yield. The logical strategy is not to select samples but to systematise them, improve their quality and invest in more sensitive methods. Because the model discriminated poorly, no predictive score was derived — a deliberate choice and, in our view, a result in itself. Molecular approaches — 16S rRNA amplification and sequencing, syndromic PCR, MALDI-TOF, metagenomic sequencing including real-time Nanopore — raise the diagnostic yield of brain abscess, especially for anaerobes and culture-negative forms, and are most useful in previously treated patients [37–46]. Their gradual integration into African reference centres, balanced against cost and local constraints, is a concrete prospect.

Empirical coverage and the antistaphylococcal gap

Empirical therapy — usually a third-generation cephalosporin, metronidazole and, as needed, an antistaphylococcal agent — matched recommended regimens for community abscess, with an appropriate median duration (7 weeks) [47–51]. Anaerobic coverage was near-universal, an important asset given these organisms are frequent but under-detected [23,24]. Both adequacy measures lay around 74–77 %, reassuring for empirical prescribing and robust to the methicillin-resistance scenario. We refrain, however, from presenting this proximity as cross-validation: the concordance analysis shows null agreement beyond chance (kappa −0.04). A theoretical spectrum grid cannot replace the antibiogram, and the two indicators must be reported separately — a point seldom made explicit in the literature using such classifications. The clearest gap concerned S. aureus, the second most frequent organism, covered in only 53 % (47 % under MRSA). This species is a leading cause of brain abscess, notably after surgery, trauma or haematogenous spread; nearly one infected patient in two received no dedicated antistaphylococcal agent initially, potentially delaying microbiological control. The gap is easily correctable: systematically adding an antistaphylococcal agent — cloxacillin where methicillin-resistance risk is low, vancomycin where it is deemed significant — to the empirical regimen for parenchymal, post-traumatic or postoperative ISI would raise expected S. aureus coverage toward 100 % without needless broadening. Conversely, antipseudomonal coverage, less often lacking, should stay reserved for at-risk situations to avoid superfluous selection pressure. Initial therapy needs rebalancing, not broadening. Our data cannot characterise resistance profiles: the antibiogram was available for only 44/50 isolates, the MRSA proportion was not exploitable, and no longitudinal resistance surveillance was in place — a priority for prospective collection.

Stewardship and practical implications in resource-limited settings

Beyond drug choice, the striking result is that two-thirds of the cohort received therapy that could be neither confirmed nor adjusted for lack of an isolate, while 93 % of regimens contained a WHO “Watch” antibiotic. Amid rising resistance — methicillin-resistant staphylococci, extended-spectrum β-lactamase- and increasingly carbapenemase-producing Enterobacterales, of particular concern in sub-Saharan Africa — this sustains a cycle where microbiological uncertainty drives broad, prolonged regimens that themselves generate selection pressure [13,18,52–55]. Stewardship finds here a priority setting [56–63]. Several low-cost levers follow from our observations, summarised as a management algorithm (Figure 4). Before antibiotics: obtain, whenever the emergency allows, a blood culture and a local sample before the first dose; standardise intra-operative sampling and secure rapid, including anaerobic, transport. At prescription: apply a protocolised empirical regimen guaranteeing the three useful pillars — activity against streptococci, S. aureus and anaerobes — with antipseudomonal broadening reserved for at-risk situations. After results: narrow the spectrum per antibiogram and adapt duration to clinico-radiological course. De-escalation, a stewardship pillar, requires microbiological documentation: positive cultures allow it, whereas negative cultures often impose empirical continuation, with direct effects on cost, adverse events and resistance selection. Tracking simple indicators — pre-antibiotic sampling rate, positive-culture rate, de-escalation proportion, carbapenem use — would offer a pragmatic continuous-improvement tool (Supplement 6).

Strengths and limitations

Although single-centre, this study covers a referral teaching hospital serving several regions, so the profile likely reflects routine neurosurgical practice in many resource-limited West African centres rather than a highly selected institutional population; the lessons appear transferable to comparable settings in Central and East Africa and to low- and middle-income countries elsewhere [64–66]. The main strengths are the joint treatment, within one West African cohort, of positivity prediction and empirical-therapy adequacy, with enough events for reasoned modelling, internal bootstrap validation, and two distinct, non-redundant adequacy approaches. Several limitations must be stressed. The mainly retrospective, single-centre design exposes to information and selection biases, and a ten-year span may introduce a period effect. Sampling depended on the operative indication rather than clinical severity, so the reported positivity describes yield in operated patients and cannot be transposed to non-operated forms, in which diagnostic puncture remains under-used. The expert

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coverage grid does not prejudge acquired resistance, does not replace the antibiogram (available in a minority), and ignores pharmacokinetics, CNS penetration and intralesional diffusion; being discordant with the antibiogram individually, it should be read only as a description of the prescribed spectrum. Prior antibiotic exposure was approximated only by referral status. The absence of molecular microbiology, local resistance data, strain genotyping, longitudinal microbiological follow-up and ecological surveillance, together with small subgroups and no external validation, limits characterisation and power.

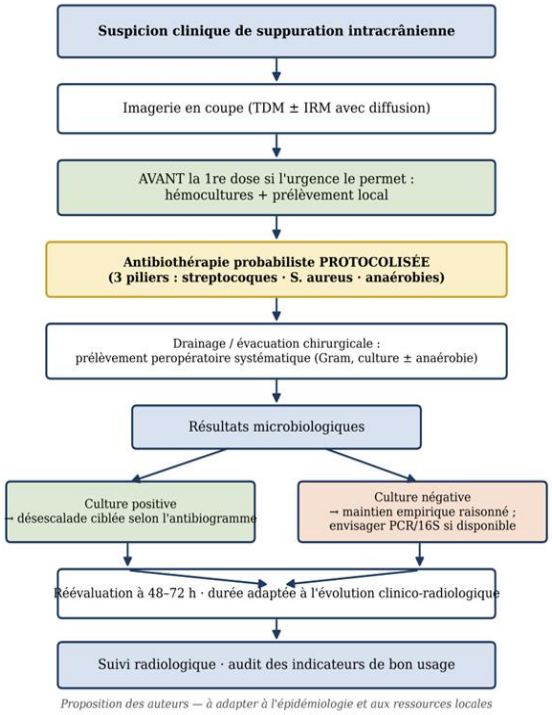

Figure 4: Proposed microbiological and antibiotic management algorithm (authors' proposal, to be adapted to local resources)

These limitations justify a prospective multicentre study capturing prior antibiotic exposure, molecular techniques, resistance surveillance and a health-economic evaluation.

CONCLUSION

In this Ivorian cohort, microbiological yield was low and unpredictable from routine clinical variables, so no patient-selection strategy for sampling is justifiable. Empirical therapy was largely appropriate, antistaphylococcal coverage being the one notable, easily correctable gap through protocolisation; yet two-thirds of patients were treated without an exploitable isolate. Our findings argue for deep sampling whenever technically feasible and without delaying treatment, a protocolised empirical regimen with optimised antistaphylococcal coverage, and early microbiology-guided re-evaluation. Improving microbiological diagnosis, rather than broadening empirical therapy, is probably the main opportunity to optimise the management of intracranial suppurative infections in resource-limited settings.

DECLARATIONS

Ethics approval

The study received internal approval from the Department of Neurosurgery at Bouaké University Hospital, under the supervision of the Head of Department, as the hospital did not have an ethics committee at the time.

Consent for publication

Not applicable; no identifying individual data are published.

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Competing interests

The authors declare no competing interests.

Data availability

The anonymised analytic dataset is available from the corresponding author on reasonable request, subject to institutional ethics-committee approval.

Code availability

The analysis script (Python 3.11), including the Firth implementation and bootstrap procedures, is available from the corresponding author on reasonable request.

Authors' contributions (CRediT)

Fionko Y.B. and Teti F.S.L.: conceptualisation, methodology, investigation, data curation, formal analysis, visualisation, writing (original draft) and critical revision. Dongo K.Y.S., Keke K.J.B., Yao K.S., Kouassi K.J.J., Traoré D. and Kouadio A.: investigation, data curation, validation and critical revision. Haidara A.: supervision, conceptualisation, methodology, validation and critical revision. All authors approved the final version and accept responsibility for the integrity of the work.

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REFERENCES

1. Brouwer MC, Tunkel AR, McKhann GM 2nd, van de Beek D. Brain abscess. N Engl J Med. 2014;371(5):447-456. doi:10.1056/NEJMra1301635
2. Brouwer MC, Coutinho JM, van de Beek D. Clinical characteristics and outcome of brain abscess: systematic review and meta-analysis. Neurology. 2014;82(9):806-813. doi:10.1212/WNL.0000000000000172
3. Brouwer MC, van de Beek D. Epidemiology, diagnosis, and treatment of brain abscess. Curr Opin Infect Dis. 2017;30(1):129-134. doi:10.1097/QCO.0000000000000334
4. Bodilsen J, Dalager-Pedersen M, Schønheyder HC, Nielsen H. Incidence and mortality of brain abscess in Denmark: a nationwide population-based study. Clin Microbiol Infect. 2020;26(1):95-100. doi: 10.1016/j.cmi.2019.05.016
5. Bodilsen J, Duerlund LS, Mariager T, Brandt CT, Petersen PT, Larsen L, et al. Clinical features and prognostic factors in adults with brain abscess. Brain. 2023;146(4):1637-1647. doi:10.1093/brain/awac312
6. Nathoo N, Nadvi SS, Narotam PK, van Dellen JR. Brain abscess: management and outcome analysis of a computed

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tomography era experience with 973 patients. World Neurosurg. 2011;75(5-6):716-726. doi: 10.1016/j.wneu.2010.11.043
7. Helweg-Larsen J, Astradsson A, Richhall H, Erdal J, Laursen A, Brennum J. Pyogenic brain abscess, a 15 year survey. BMC Infect Dis. 2012;12:332. doi:10.1186/1471-2334-12-332
8. Mathisen GE, Johnson JP. Brain abscess. Clin Infect Dis. 1997;25(4):763-779. doi:10.1086/515541
9. Sonneville R, Ruimy R, Benzonana N, Riahi H, Wolff M. An update on bacterial brain abscess in immunocompetent patients. Clin Microbiol Infect. 2017;23(9):614-620. doi: 10.1016/j.cmi.2017.05.004
10. Moorthy RK, Rajshekhar V. Management of brain abscess: an overview. Neurosurg Focus. 2008;24(6):E3. doi:10.3171/FOC/2008/24/6/E3
11. Muzumdar D, Jhawar S, Goel A. Brain abscess: an overview. Int J Surg. 2011;9(2):136-144. doi: 10.1016/j.ijsu.2010.11.005
12. Patel K, Clifford DB. Bacterial brain abscess. Neurohospitalist. 2014;4(4):196-204. doi:10.1177/1941874414540684
13. Antimicrobial Resistance Collaborators. Global burden of bacterial antimicrobial resistance in 2019: a systematic analysis. Lancet. 2022;399(10325):629-655. doi:10.1016/S0140-6736(21)02724-0
14. World Health Organization. Global action plan on antimicrobial resistance. Geneva: World Health Organization; 2015.
15. World Health Organization. The WHO AWaRe (Access, Watch, Reserve) antibiotic book. Geneva: World Health Organization; 2022.
16. World Health Organization. Antimicrobial stewardship programmes in health-care facilities in low- and middle-income countries: a WHO practical toolkit. Geneva: World Health Organization; 2019.
17. World Health Organization. Global antimicrobial resistance and use surveillance system (GLASS) report 2022. Geneva: World Health Organization; 2022.
18. World Health Organization. WHO bacterial priority pathogens list, 2024. Geneva: World Health Organization; 2024.
19. Cox JA, Vlieghe E, Mendelson M, Wertheim H, Ndegwa L, Villegas MV, et al. Antibiotic stewardship in low- and middle-income countries: the same but different? Clin Microbiol Infect. 2017;23(11):812-818. doi: 10.1016/j.cmi.2017.07.010
20. Udoh A, Ernawati DK, Akpan M, Galbraith K, Hussein R, Ashiru-Oredope D. Implementation of antimicrobial stewardship programmes in African countries: a systematic literature review. J Glob Antimicrob Resist. 2020; 22:317-324. doi: 10.1016/j.jgar.2020.03.009
21. Miller JM, Binnicker MJ, Campbell S, Carroll KC, Chapin KC, Gonzalez MD, et al. A guide to utilization of the microbiology laboratory for diagnosis of infectious diseases. Clin Infect Dis. 2018;67(6):e1-e94. doi:10.1093/cid/ciy381
22. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement. Lancet. 2007;370(9596):1453-1457. doi:10.1016/S0140-6736(07)61602-X
23. Brook I. Microbiology and treatment of brain abscess. J Clin Neurosci. 2017;38:8-12. doi: 10.1016/j.jocn.2016.12.035
24. Brook I. Antimicrobial treatment of anaerobic infections. Expert Opin Pharmacother. 2011;12(11):1691-1707. doi:10.1517/14656566.2011.576672
25. Nau R, Sörgel F, Eiffert H. Penetration of drugs through the blood-cerebrospinal fluid/blood-brain barrier for treatment of central nervous system infections. Clin Microbiol Rev. 2010;23(4):858-883. doi:10.1128/CMR.00007-10
26. Newcombe RG. Two-sided confidence intervals for the single proportion: comparison of seven methods. Stat Med. 1998;17(8):857-872. doi:10.1002/(SICI)1097-0258(19980430)17:8<857::AID-SIM777>3.0.CO;2-E
27. Riley RD, Snell KIE, Ensor J, Burke DL, Harrell FE Jr, Moons KGM, et al. Minimum sample size for developing a multivariable prediction model. Stat Med. 2019;38(7):1276-1296. doi:10.1002/sim.7992
28. Firth D. Bias reduction of maximum likelihood estimates. Biometrika. 1993;80(1):27-38. doi:10.1093/biomet/80.1.27
29. Heinze G, Schemper M. A solution to the problem of separation in logistic regression. Stat Med. 2002;21(16):2409-2419. doi:10.1002/sim.1047
30. Harrell FE Jr. Regression modeling strategies. 2nd ed. Cham: Springer; 2015. doi:10.1007/978-3-319-19425-7
31. Steyerberg EW. Clinical prediction models. 2nd ed. Cham: Springer; 2019. doi:10.1007/978-3-030-16399-0
32. Van Calster B, McLernon DJ, van Smeden M, Wynants L, Steyerberg EW. Calibration: the Achilles heel of predictive analytics. BMC Med. 2019;17(1):230. doi:10.1186/s12916-019-1466-7
33. Collins GS, Reitsma JB, Altman DG, Moons KGM. Transparent reporting of a multivariable prediction model for individual prognosis or diagnosis (TRIPOD): the TRIPOD statement. Ann Intern Med. 2015;162(1):55-63. doi:10.7326/M14-0697
34. Boukassa L, Ngackosso OB, Kinata SB, Ekouele Mbaki HB. Intracranial suppurations in Africa: systematic review. Open J Mod Neurosurg. 2020;10(2):244-253. doi:10.4236/ojmn.2020.102023
35. Ooi SZY, Sichimba D, Dalle DU, Higginbotham G, Takoutsing BD, Bankole NDA, et al. Management and outcomes of paediatric intracranial suppurations in low- and middle-income countries: a scoping review. Front Surg. 2021;8:690895. doi:10.3389/fsurg.2021.690895
36. Kabré A, Zabsonré DS, Diallo O, Cissé R. Prise en charge médico-chirurgicale des abcès du cerveau à l'ère du scanner en Afrique subsaharienne. Neurochirurgie. 2014;60(5):249-253.
37. Al Masalma M, Armougom F, Scheld WM, Dufour H, Roche PH, Drancourt M, et al. The expansion of the microbiological spectrum of brain abscesses with use of multiple 16S ribosomal DNA sequencing. Clin Infect Dis. 2009;48(9):1169-1178. doi:10.1086/597578
38. Al Masalma M, Lonjon M, Richet H, Dufour H, Roche PH, Drancourt M, et al. Metagenomic analysis of brain abscesses identifies specific bacterial associations. Clin Infect Dis. 2012;54(2):202-210. doi:10.1093/cid/cir797
39. Kommedal Ø, Wilhelmsen MT, Skrede S, Meisal R, Jakovljev A, Gaustad P, et al. Massive parallel sequencing provides new perspectives on bacterial brain abscesses. J Clin Microbiol. 2014;52(6):1990-1997. doi:10.1128/JCM.00346-14
40. Stebner A, Ensser A, Geißdörfer W, Bozhkov Y, Lang R. Molecular diagnosis of polymicrobial brain abscesses with 16S-rRNA-based next-generation sequencing. Clin Microbiol Infect. 2021;27(1):76-82. doi: 10.1016/j.cmi.2020.03.028
41. Keller PM, Rampini SK, Bloemberg GV. Detection of a mixed infection in a culture-negative brain abscess by broad-spectrum bacterial 16S rRNA gene PCR. J Clin

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Microbiol. 2010;48(6):2250-2252. doi:10.1128/JCM.00317-10
42. Wilson MR, Sample HA, Zorn KC, Arevalo S, Yu G, Neuhaus J, et al. Clinical metagenomic sequencing for diagnosis of meningitis and encephalitis. N Engl J Med. 2019;380(24):2327-2340. doi:10.1056/NEJMoa1803396
43. Gu W, Miller S, Chiu CY. Clinical metagenomic next-generation sequencing for pathogen detection. Annu Rev Pathol. 2019; 14:319-338. doi:10.1146/annurev-pathmechdis-012418-012751
44. Chiu CY, Miller SA. Clinical metagenomics. Nat Rev Genet. 2019;20(6):341-355. doi:10.1038/s41576-019-0113-7
45. Patel R. MALDI-TOF MS for the diagnosis of infectious diseases. Clin Chem. 2015;61(1):100-111. doi:10.1373/clinchem.2014.221770
46. Petti CA. Detection and identification of microorganisms by gene amplification and sequencing. Clin Infect Dis. 2007;44(8):1108-1114. doi:10.1086/512818
47. Bodilsen J, D'Alessandris QG, Humphreys H, Iro MA, Klein M, Last K, et al.; ESCMID Study Group for Infections of the Brain. European Society of Clinical Microbiology and Infectious Diseases guidelines on diagnosis and treatment of brain abscess in children and adults. Clin Microbiol Infect. 2024;30(1):66-89. doi: 10.1016/j.cmi.2023.08.016
48. Tunkel AR, Hasbun R, Bhimraj A, Byers K, Kaplan SL, Scheld WM, et al. 2017 Infectious Diseases Society of America's clinical practice guidelines for healthcare-associated ventriculitis and meningitis. Clin Infect Dis. 2017;64(6):e34-e65. doi:10.1093/cid/ciw861
49. van de Beek D, Cabellos C, Dzupova O, Esposito S, Klein M, Kloek AT, et al. ESCMID guideline: diagnosis and treatment of acute bacterial meningitis. Clin Microbiol Infect. 2016;22 Suppl 3:S37-S62. doi: 10.1016/j.cmi.2016.01.007
50. Arlotti M, Grossi P, Pea F, Tomei G, Vullo V, De Rosa FG, et al. Consensus document on controversial issues for the treatment of infections of the central nervous system: bacterial brain abscesses. Int J Infect Dis. 2010;14 Suppl 4:S79-S92. doi: 10.1016/j.ijid.2010.05.010
51. Bodilsen J, Brouwer MC, Nielsen H, van de Beek D. Anti-infective treatment of brain abscess. Expert Rev Anti Infect Ther. 2018;16(7):565-578. doi:10.1080/14787210.2018.1489722
52. Holmes AH, Moore LSP, Sundsfjord A, Steinbakk M, Regmi S, Karkey A, et al. Understanding the mechanisms and drivers of antimicrobial resistance. Lancet. 2016;387(10014):176-187. doi:10.1016/S0140-6736(15)00473-0
53. Laxminarayan R, Duse A, Wattal C, Zaidi AKM, Wertheim HFL, Sumpradit N, et al. Antibiotic resistance — the need for global solutions. Lancet Infect Dis. 2013;13(12):1057-1098. doi:10.1016/S1473-3099(13)70318-9
54. O'Neill J. Tackling drug-resistant infections globally: final report and recommendations. London: Review on Antimicrobial Resistance; 2016.
55. Mendelson M, Matsoso MP. The World Health Organization global action plan for antimicrobial resistance. S Afr Med J. 2015;105(5):325. doi:10.7196/SAMJ.9644
56. Barlam TF, Cosgrove SE, Abbo LM, MacDougall C, Schuetz AN, Septimus EJ, et al. Implementing an antibiotic stewardship program: guidelines by the Infectious Diseases Society of America and the Society for Healthcare Epidemiology of America. Clin Infect Dis. 2016;62(10):e51-e77. doi:10.1093/cid/ciw118
57. Dellit TH, Owens RC, McGowan JE Jr, Gerding DN, Weinstein RA, Burke JP, et al. IDSA and SHEA guidelines for developing an institutional program to enhance antimicrobial stewardship. Clin Infect Dis. 2007;44(2):159-177. doi:10.1086/510393
58. Schuts EC, Hulscher MEJL, Mouton JW, Verduin CM, Stuart JWTC, Overdiek HWPM, et al. Current evidence on hospital antimicrobial stewardship objectives: a systematic review and meta-analysis. Lancet Infect Dis. 2016;16(7):847-856. doi:10.1016/S1473-3099(16)00065-7
59. Dyar OJ, Huttner B, Schouten J, Pulcini C. What is antimicrobial stewardship? Clin Microbiol Infect. 2017;23(11):793-798. doi: 10.1016/j.cmi.2017.08.026
60. Pulcini C, Binda F, Lamkang AS, Trett A, Charani E, Goff DA, et al. Developing core elements and checklist items for global hospital antimicrobial stewardship programmes. Clin Microbiol Infect. 2019;25(1):20-25. doi: 10.1016/j.cmi.2018.03.033
61. Morency-Potvin P, Schwartz DN, Weinstein RA. Antimicrobial stewardship: how the microbiology laboratory can right the ship. Clin Microbiol Rev. 2017;30(1):381-407. doi:10.1128/CMR.00042-16
62. Godman B, Egwuenu A, Haque M, Malande OO, Schellack N, Kumar S, et al. Strategies to improve antimicrobial utilization with a special focus on developing countries. Life (Basel). 2021;11(6):528. doi:10.3390/life11060528
63. Mudenda S, Daka V, Matafwali SK, Skosana P, Chabalenge B, Mukosha M, et al. A systematic review of pharmacist-led antimicrobial stewardship programs in sub-Saharan Africa. Int J Environ Res Public Health. 2022;19(19):12469. doi:10.3390/ijerph191912469
64. Dewan MC, Rattani A, Fieggen G, Arraez MA, Servadei F, Boop FA, et al. Global neurosurgery: the current capacity and deficit in the provision of essential neurosurgical care. J Neurosurg. 2018;130(4):1055-1064. doi:10.3171/2017.11.JNS171500
65. Meara JG, Leather AJM, Hagander L, Alkire BC, Alonso N, Ameh EA, et al. Global Surgery 2030: evidence and solutions for achieving health, welfare, and economic development. Lancet. 2015;386(9993):569-624. doi:10.1016/S0140-6736(15)60160-X
66. Park KB, Johnson WD, Dempsey RJ. Global neurosurgery: the unmet need. World Neurosurg. 2016;88:32-35. doi:10.1016/j.wneu.2015.12.048

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