The Increasing Threat of Antibiotic Resistance in India a Comprehensive Review

Harshit Sharma[1], Mahin Khan[2]
[1]*Doctor of Pharmacy, Apex Institute of Pharmacy*
[2]*Pharmacist, Apex Welcare Trust Hospital*
doi.org/10.64643/IJIRTV12I8-191457-459

***Abstract*—Antibiotic resistance in India has reached crisis levels, with routine infections – including urinary tract infections (UTIs), pneumonia, and sepsis increasingly untreatable by standard drugs. Surveillance data show that commonly used antimicrobials (e.g. beta-lactams, fluoroquinolones, carbapenems) have lost efficacy against key Gram-negative pathogens (E. coli, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa) and even Gram-positive cocci (enterococci). Globally, one in six bacterial infections is now drug-resistant, foreshadowing millions of deaths by 2050. Here we review laboratory methods for resistance testing, summarize dominant pathogens and resistance patterns in Indian clinical settings, and examine the consequences and drivers of this pandemic. Tables summarize resistance levels by antibiotic class and pathogen. We conclude with strategic recommendations for surveillance, stewardship, and innovation to avert a "post-antibiotic" era.**

***Index Terms*—Antimicrobial resistance, Antibiotic stewardship, Multidrug-resistant bacteria, Gram-negative pathogens, India, Hospital infections.**

I. INTRODUCTION

Antimicrobial resistance (AMR) poses a critical health crisis in India. A recent national report (ICMR AMRSN 2024) documents that antibiotics widely used for UTIs, pneumonia, sepsis and diarrheal disease are failing at alarming rates. In hospital surveillance of ~100,000 infections, drug-resistant Gram-negative bacteria dominated: for example, Escherichia coli (leading UTI and bloodstream pathogen) showed steadily declining susceptibility, and Klebsiella pneumoniae (major cause of pneumonia/sepsis) was resistant to piperacillin–tazobactam in ~75% of cases and to carbapenems in most samples. In intensive care units (ICUs), Acinetobacter baumannii exhibited ~91% resistance to meropenem, and 72% of bloodstream infections were caused by multidrug-resistant Gram-negatives.

This dire situation in India reflects a global trend. The WHO's 2025 GLASS report finds that nearly one in six bacterial infections worldwide no longer responds to first-line antibiotics. AMR-related deaths are projected to rise from ~4.7 million (2021) to ~8.2 million by 2050. In India, the burden already exceeds global averages: for instance, over 41,000 hospital bloodstream infections involve organisms resistant even to last-line drugs. Formerly reliable medications like amoxicillin, ciprofloxacin, and third-generation cephalosporins are rapidly losing effectiveness. Experts warn that without urgent intervention, "even common infections may soon become untreatable," imperiling routine surgeries, childbirth and standard medical care.

The following sections outline standard laboratory methods for detecting resistance, review the major pathogens and infection types involved (UTI, sepsis, wound, pneumonia), and detail resistance trends in key antibiotic classes. We then examine the mechanisms and drivers of resistance in India, the clinical/public health impacts, and conclude with recommended actions.

II. LABORATORY METHODS FOR RESISTANCE TESTING

Conventional phenotypic antimicrobial susceptibility tests (AST) remain the cornerstone of resistance detection in clinical microbiology. The most common approach is the Kirby–Bauer disk diffusion assay: bacterial isolates are spread on agar, antibiotic-impregnated disks are applied, and zones of inhibition are measured. This standardized, low-cost method (CLSI/EUCAST guidelines) rapidly classifies isolates as susceptible, intermediate or resistant, making it ideal for routine monitoring. Its limitation is that it yields only qualitative results, not minimum inhibitory concentrations (MICs). For precise quantification, dilution methods (broth or agar dilution) are used to determine the MIC – the lowest antibiotic concentration preventing growth. Broth microdilution in 96-well plates is the accepted gold standard for AST, applicable to diverse pathogens including fastidious bacteria and fungi.

Gradient diffusion (E-test) strips combine the simplicity of agar-based testing with MIC estimation: a strip with a concentration gradient is placed on inoculated agar, and the MIC is read where inhibition meets the strip. Automated instruments (e.g. VITEK, BD Phoenix) perform broth microdilution and provide MICs quickly for large sample volumes. Molecular assays (PCR, qPCR) detect specific resistance genes (e.g. bla_NDM, mcr-1) and can greatly speed identification of known mechanisms. Whole-genome sequencing and MALDI-TOF MS are also emerging tools for resistance surveillance, though cost and complexity currently limit routine use. In practice, Indian hospitals rely on a combination of disk diffusion and automated MIC platforms for daily AST, as these methods are standardized, economical, and essential for guiding therapy.

2.1. Dominant Pathogens and Infection Syndromes

In India's hospitals, Gram-negative "superbugs" dominate critical infections. The leading pathogens in UTIs, sepsis and ventilator-associated pneumonia include E. coli, K. pneumoniae, P. aeruginosa, and A. baumannii. Specifically, E. coli remains the most common cause of community and hospital UTIs and abdominal infections; K. pneumoniae is a major agent of pneumonia and bloodstream infections; P. aeruginosa and A. baumannii are prominent in ICU-acquired respiratory and wound infections. Among Gram-positives, enterococci (including E. faecalis/E. faecium) are important causes of catheter-associated UTIs and wound infections, and Staphylococcus aureus causes skin/wound infections and sepsis.

These organisms are associated with specific syndromes: for example, community-acquired UTI and gastrointestinal infections frequently involve E. coli (often extended-spectrum β-lactamase producers) and Salmonella/Shigella spp.; hospital-acquired pneumonia and ventilator infections involve Klebsiella, Acinetobacter, and Pseudomonas (often carbapenem-resistant). Surgical-site and wound infections often involve S. aureus (including MRSA) and Gram-negatives. In summary, UTIs, bloodstream sepsis, wound infections and nosocomial pneumonia are the major clinical settings for resistant pathogens in India. Table 1 summarizes key pathogens and resistance impacts by syndrome.

2.2. Resistance in Key Drug Classes

The surveillance data paint a bleak picture across major antibiotic classes. Table 2–5 summarize trends for representative drugs, pathogens, and clinical consequences.

- Beta-lactams (Penicillins and Cephalosporins). Common penicillins (ampicillin/amoxicillin) have become virtually useless against Gram-negative pathogens. E. coli, Klebsiella and Salmonella spp. now produce ESBL β-lactamases (e.g. CTX-M, TEM) conferring high resistance. As a result, simple community infections (UTIs, sinusitis, enteric fever) often fail first-line therapy. Third-generation cephalosporins fare no better: ceftriaxone (a standard for typhoid fever, pneumonia, meningitis) is now inactivated by ESBLs, with >90% of Salmonella Typhi isolates resistant. Cefepime (4th-generation cephalosporin) was a last-resort agent, but reports indicate >90% resistance in Typhi and widespread resistance in other Gram-negatives. Piperacillin–tazobactam (an anti-pseudomonal penicillin/β-lactamase inhibitor) also shows very high failure rates (e.g. ~75% of Klebsiella spp. are now resistant). In short, broad-spectrum β-lactams now fail against most ESBL-producing Enterobacter ales, forcing reliance on more toxic agents (Tables 2 and 3).
- Carbapenems (Imipenem, Meropenem). Once considered "last-line" drugs, carbapenems have lost efficacy against the most problematic Gram-negatives. National data show >50% of ICU Gram-negative isolates (especially A. baumannii) are imipenem-resistant, and meropenem resistance is ubiquitous (A. baumannii ~91%). Critically, K. pneumoniae and E. coli with metallo-β-lactamases (NDM) and OXA-carbapenems are increasingly common. The result is that meropenem- or imipenem-resistant infections (e.g. septicemia, ventilator pneumonias) require "salvage" use of nephrotoxic drugs like colistin or tigecycline. ICU mortality has risen due to untreatable carbapenem-resistant sepsis.
- Fluoroquinolones (Ciprofloxacin, Ofloxacin). Quinolone resistance is near-universal among enteric pathogens. Historically the first-line therapy for typhoid and gastroenteritis, ciprofloxacin is now ineffective in >95% of Salmonella Typhi and similarly fails against Vibrio, Shigella and many E. coli strains. Treatment failures with ciprofloxacin (and the related ofloxacin) in UTIs and pneumonia are common. The main drivers are widespread OTC use, incomplete courses, and chromosomal mutations (gyrA, parC) plus plasmid-mediated qnr genes. In practice, empiric quinolone use is now avoided for systemic infections.
- Glycopeptides (Vancomycin) and Polymyxins (Colistin). Among Gram-positives, vancomycin resistance is emerging. About 5–10% of Enterococcus faecium isolates in India are vancomycin-resistant (VRE) and a small fraction (<5%) of S. aureus are vancomycin-intermediate or -resistant. This endangers treatment of MRSA/MRSE bacteremias and serious enterococcal infections. Colistin, a "last-resort" polymyxin for carbapenem-resistant Gram-negatives, is also under threat. Clinical isolates of A. baumannii, K. pneumoniae and P. aeruginosa with plasmid-mediated mcr genes and mgrB mutations are increasingly reported. Colistin-resistant NICU and ICU

outbreaks have been observed, raising the prospect of pan-drug-resistant strains.

[Table 2] [Table 3] [Table 4] [Table 5] below summarize these trends. (β-lactams: Table 2; Carbapenems: Table 3; Fluoroquinolones: Table 4; Vancomycin/Colistin: Table 5 and Table 6; Remaining Antibiotics.

Table 2. Beta-Lactam Antibiotics: Resistant Pathogens and Clinical Impact

Antibiotic (Class)	Resistant Pathogens and Mechanisms	Clinical Impact / Comments
Ampicillin, Amoxicillin (aminopenicillins)	E. coli, K. pneumoniae, Salmonella, Shigella (ESBL producers)	Routine UTIs, enteric fever, respiratory/GI infections often fail first-line therapy; hospitalization required for IV drugs.
Piperacillin–Tazobactam (extended-spectrum penicillin + BLI)	K. pneumoniae (~75% resistant), P. aeruginosa, E. coli (ESBL/carbapenems producers)	Empirical therapy for Gram-negative sepsis now largely ineffective for Klebsiella and other enteric pathogens; ICU cases rising.
Ceftriaxone, Cefotaxime (3rd-gen cephalosporins)	ESBL E. coli, K. pneumoniae; Salmonella enterica (Typhi/Paratyphi)	Former first-line for typhoid, meningitis, pneumonia; >90% of typhoid cases are now ceftriaxone-resistant, undermining standard treatment.
Cefepime (4th-gen cephalosporin)	ESBL/carbapenems E. coli, K. pneumoniae; Pseudomonas	"Last-resort" cephalosporin; resistance >90% in S. Typhi and high among other Gram-negatives, severely limiting empiric therapy options.

Table 3. Carbapenems: Resistant Pathogens and Clinical Impact

Antibiotic (Class)	Resistant Pathogens	Clinical Impact / Comments
Imipenem (Carbapenem)	A. baumannii, K. pneumoniae, E. coli (NDM/OXA producers)	Once "last-line" therapy for severe sepsis; now >50% of isolates (especially A. baumannii) are resistant. ICU mortality has risen due to untreatable infections.
Meropenem (Carbapenem)	A. baumannii (~91% resistant), K. pneumoniae, E. coli	Resistance patterns similar to imipenem; meropenem resistance is common in ICU pathogens, forcing use of toxic alternatives (e.g. colistin).

Table 4. Fluoroquinolones: Resistant Pathogens and Clinical Impact

Antibiotic (Class)	Resistant Pathogens	Clinical Impact / Comments
Ciprofloxacin	Salmonella spp. (>95% S. Typhi resistant), E. coli, Vibrio, Shigella	Historically first-line for typhoid and GI infections; now almost universally ineffective. High failure rates in UTIs and pneumonia. Empiric use has been largely abandoned.
Ofloxacin	Salmonella, E. coli, others (similar to ciprofloxacin)	Former common oral quinolone; likewise, now ineffective against most Gram-negative pathogens.

Table 5. Glycopeptides and Polymyxins: Resistant Pathogens and Clinical Impact

Antibiotic (Class)	Resistant Pathogens	Clinical Impact / Comments
Vancomycin (Glycopeptide)	Enterococcus faecium (VRE ~5–10%), Staphylococcus aureus (VRSA rare <5%)	Last-line for MRSA and enterococcal infections; emerging VRE and VRSA strains threaten management of endocarditis, bacteremia and wound infections.
Colistin (Polymyxin)	A. baumannii, K. pneumoniae, P. aeruginosa (carbapenem-resistant isolates)	"Last-resort" for carbapenem-resistant Gram-negatives; rising plasmid-mediated resistance (mcr genes) and NICU/ICU outbreaks jeopardize treatment of pan-resistant infections.

Table 5. Remaining antibiotics

Antibiotic (Class)	Resistant Organisms	Clinical Impact	Root Causes
Ampicillin (Aminopenicillin)	E. coli, K. pneumoniae, Salmonella spp., Shigella spp.	UTIs, enteric fever, pneumonia often untreatable; treatment failures in GI/resp infections	Overuse/OTC dispensing and incomplete courses; ESBL-mediated β-lactamase (CTX-M, TEM) confer resistance.
Amoxicillin (Aminopenicillin)	Similar spectrum as ampicillin (resistance equally high)	Ineffective for common infections (sinusitis, UTIs); leads to hospitalizations	Same as ampicillin (community misuse, livestock use).
Amoxicillin/Clavulanate (β-lactam/β-lactamase inhibitor)	ESBL-producing E. coli, Klebsiella spp., Acinetobacter (CTX-M, OXA-48 producers)	Broad-spectrum combination often fails against MDR Gram-negatives; limits outpatient options	Widespread use in community and animal husbandry; β-lactamase hyperproducers exceed inhibitor capacity.
Ampicillin/Sulbactam (β-lactam/β-lactamase inhibitor)	Acinetobacter baumannii, Pseudomonas, ESBL-Enterobacter ales	Empirical therapy for intraabdominal and respiratory infections often fails; ICU cases surged	Same drivers (OTC, hospitals, animal use); many pathogens co-harbor multiple β-lactamases.
Piperacillin–Tazobactam (Extended-spectrum penicillin + BLI)	K. pneumoniae (~75% resistant), P. aeruginosa, E. coli (rising)	Once-empirical anti-pseudomonal agent; now largely ineffective for Klebsiella and enteric sepsis	Overuse in hospitals and clinics; prevalent carbapenems and ESBLs.
Aztreonam (Monobactam)	P. aeruginosa, ESBL-Enterobacterales	Limited use for GN rods; many isolates (especially Pseudomonas) now resistant, reducing salvage options	Misuse in severe infections; β-lactamase enzymes (ESBL/MBL) hydrolyze it (CTX-M, NDM).
Ceftriaxone (3rd-gen cephalosporin)	ESBL-E. coli, ESBL-Klebsiella, Salmonella enterica (typhi/paratyphi)	Standard therapy for typhoid, pneumonia, meningitis – now fails in >90% Typhi cases and many GN sepsis	Long-term overuse, including in agriculture; ESBLs (CTX-M-15 etc) completely inactivate it.
Cefotaxime (3rd-gen ceph)	Same as ceftriaxone (ESBL-Enterobacter ales)	Similar to above (often interchangeable with ceftriaxone); increasingly useless for resistant GN infections	See ceftriaxone.
Ceftazidime (3rd-gen ceph)	Same (ESBL-producers, plus many Pseudomonades with AmpC β-lactamase)	Formerly used for pseudomonads; rising ceftazidime-resistant Pseudomonas and Klebsiella hamper ICU therapy	Intensive hospital use, selection of AmpC and ESBLs.
Cefixime (3rd-gen ceph, oral)	Salmonella spp., E. coli, Enterobacter (ESBLs)	Oral therapy for typhoid and UTIs failing; requires IV backup.	Same drivers; frequent community misuse (self-medication).
Cefepime (4th-gen ceph)	ESBL/Klebsiella/E. coli, Pseudomonas with carbapenems	Last-resort cephalosporin – resistance >90% in Typhi cases and high in other GN, undermining empiric choices	Broad-spectrum misuse in hospitals and dairies; overproduction of β-lactamases.
Imipenem (Carbapenem)	A. baumannii, Klebsiella spp., E. coli (NDM/OXA producers)	Once "last-line" therapy; now >50% of GN isolates (especially A. baumannii) are resistant. ICU mortality has risen due to untreatable sepsis.	Rampant use in ICUs and lack of stewardship; emergence of MBL (NDM) and OXA carbapenems eroding efficacy.
Meropenem (Carbapenem)	A. baumannii (~91% resistant), K. pneumoniae, E. coli	Similar impact as imipenem; meropenem resistance now common in	Same as imipenem (nosocomial overuse, animal

Antibiotic (Class)	Resistant Organisms	Clinical Impact	Root Causes
		ICU pathogens, forcing use of toxic or untested drugs.	feedlot use); prolific carbapenems.
Ciprofloxacin (Fluoroquinolone)	Salmonella spp. (>95% Typhi), E. coli, Vibrio, Shigella	Historically first-line for typhoid and gastroenteritis – now almost universally ineffective; UTI/pneumonia treatments fail at high rates.	Long-term OTC misuse, including incomplete courses; chromosomal mutations + plasmid Qnr genes widespread.
Levofloxacin (Fluoroquinolone)	Similar to ciprofloxacin (GN Enterobacter ales, Acinetobacter)	Also, largely ineffective for GN infections (UTIs, hospital-acquired infections).	Same misuse/selection as ciprofloxacin.
Ofloxacin (Fluoroquinolone)	Salmonella spp., E. coli, others (see ciprofloxacin)	Once common oral quinolone; now similarly resisted, forcing use of injectables.	Same causes.
Trimethoprim–Sulfamethoxazole (Folate synthesis inhibitor)	Salmonella spp., E. coli, Staphylococcus (MSSA/MRSA), Pneumococcus	Former "co-trimoxazole" for typhoid and UTIs. Widespread resistance means many UTIs and bloodstream infections no longer respond.	Over-the-counter availability (often used for diarrhea); efflux pumps and target mutations in bacteria.
Gentamicin (Aminoglycoside)	Salmonella spp., E. coli, Klebsiella, Pseudomonas	Used for severe infections and neonatal sepsis; rising gentamicin-resistant strains lead to treatment failures and necessitate amikacin/colistin use.	Hospital use in sepsis (ICUs) selects resistant strains; aminoglycoside-modifying enzymes common.
Amikacin (Aminoglycoside)	E. coli, K. pneumoniae, Acinetobacter (20–30% resistance)	Last-resort aminoglycoside; some improvement noted (ICMR reports slight rise in susceptibility), but resistant strains still cause treatment failures.	Often used as second-line for GN, fueling resistance; enzymatic modification (AAC, etc).
Tetracycline	Salmonella spp., Vibrio, Acinetobacter	Once broad-use antibiotic; now many pathogens carry Tet genes, so tetracycline therapy is unreliable. Used in some diarrheas – failure rates high.	Long-term widespread use in farming (growth promoter); efflux/tetM ribosomal protection genes prevalent.
Azithromycin (Macrolide)	Salmonella (Typhi/Paratyphi) emerging, Campylobacter, some Streptococcus	Often the only oral option left for typhoid/malaria prophylaxis. Isolated resistance is rising globally; in India, some Typhi isolates show decreased susceptibility.	Increasing use as alternative to quinolones; point mutations in 23S rRNA reported.
Erythromycin (Macrolide)	Staphylococcus aureus (MSSA/MRSA inducible MLS resistance), Streptococci	Rarely used for serious infections due to intrinsic Gram-neg resistance and erm-mediated staph resistance. Its ineffectiveness contributes to limited options for staph skin infections.	Long-standing use; methylase (erm) genes widespread.
Clindamycin (Lincosamide)	Staphylococcus (MLSB-inducible), Bacteroides	Used for some staph/anaerobic infections. Inducible resistance (erm genes) in MRSA/MSSA limits its use. Increasing clindamycin failures in skin/soft tissue infections noted.	Widespread prior use; clindamycin-resistance (erm methylases) is common.
Vancomycin (Glycopeptide)	Enterococci (VRE ~5–15%), Staphylococcus aureus (VRSA rare ~2%)	Last-line for MRSA/MRSE. Vancomycin-intermediate/resistant S. aureus (<5%) and ~5–10% of	Overuse in hospitals (IV catheters, ICUs) selects

Antibiotic (Class)	Resistant Organisms	Clinical Impact	Root Causes
		Enterococcus faecium (VRE) are now resistant, endangering treatment of endocarditis and bacteremia.	VRE/MRSA; van gene spread from animal agriculture.
Teicoplanin (Glycopeptide)	Similar spectrum as vancomycin (no VRSA reported, some VRE cross-resistance)	Alternative to vancomycin for Gram-positives. Though largely effective (in vitro ~100% against MRSA), reliance on it can select teicoplanin-resistant enterococci.	Often used interchangeably with vancomycin; similar selective pressures apply.
Linezolid (Oxazolidinone)	Enterococci, Staphylococcus (rare linezolid-resistant strains)	Last-resort for MRSA and VRE. Very rare resistance in India so far (case reports), but its high-cost limits use. Resistance would leave few treatments for vancomycin-resistant infections.	Minimal use in India to date; however, use of linezolid analogs in livestock could propagate cross-resistance.
Colistin (Polymyxin)	A. baumannii, K. pneumoniae, Pseudomonas (colistin-resistant isolates rising)	"Last-resort" for carbapenem-resistant GN. Increasing colistin resistance (plasmid mcr genes and mgrB mutations) has been reported, especially in NICUs and ICUs, threatening pan-drug-resistant infections.	Heavy use in agriculture (poultry, cattle) and hospitals; plasmid-mediated resistance (mcr-1) spreading globally.
Chloramphenicol (Amphenicol)	Salmonella (currently lower resistance), Haemophilus, Bacteroides	Historically first-line for typhoid; regained some efficacy (ICMR reports ~95% Typhi sensitivity) but rapid toxicity limits use. Many other bacteria have cat acetyltransferases; it is considered unsafe for systemic use in India.	Though less used now, past widespread use selected chloramphenicol-acetyltransferase genes; substandard formulations in market further propagate resistance.

III. ROOT CAUSES AND MECHANISMS OF RESISTANCE

Antibiotic resistance is multifactorial, but misuse and overuse of antimicrobials are universally cited as dominant drivers. In India, lax regulation has long allowed antibiotics to be sold over-the-counter (OTC) without prescription. Patients frequently self-medicate or discontinue treatment early when symptoms improve, exposing bacteria to sub-lethal drug concentrations. In agriculture, antibiotics are used indiscriminately for livestock growth promotion and prophylaxis, disseminating resistant strains into the food chain. Within hospitals, empirical "blind" prescribing is common – often without culture confirmation – due to limited laboratory access and acute patient load. These practices create intense selective pressure: susceptible bacteria are killed off while resistant mutants proliferate.

Molecular mechanisms compound this problem. Indian isolates frequently harbor extended-spectrum β-lactamase (ESBL) genes (e.g. CTX-M, TEM) that hydrolyze penicillin's and cephalosporins. Carbapenems enzymes (NDM-1, OXA-48 etc.) destroy carbapenems. Plasmid-borne genes (e.g. qnr, aac(6')-Ib-cr) confer quinolone resistance, while recent detection of the mcr-1 gene enables transferable colistin resistance. These mobile elements move between species via horizontal gene transfer, accelerating spread of multi-drug resistance. Efflux pump overexpression and target-site mutations also play roles (e.g. gyrA mutations in fluoroquinolone resistance, 23S rRNA mutations for macrolides). In summary, a history of excessive antibiotic exposure – in patients, hospitals and farms – has driven the evolution and dissemination of resistance mechanisms, eroding drug efficacy nationwide.

IV. CLINICAL AND PUBLIC HEALTH IMPACT

The clinical consequences of this resistance epidemic are profound. Treatment failures for once-routine infections are now common. Critically ill patients, especially in ICUs, face unacceptably high mortality from untreatable sepsis. The ICMR report notes that India's "most widely used antibiotics are losing power, and critically ill patients are already facing the consequences". Ventilator-associated pneumonias are increasingly caused by pan-resistant

Acinetobacter/Klebsiella, against which no standard therapy exists. Neonatal and pediatric units report multi-drug resistant Gram-negative sepsis with very limited therapeutic options.

At a public-health level, AMR threatens the very foundation of modern medicine. If current trends continue, many common procedures (surgeries, chemotherapy, childbirth) could become dangerous due to untreatable infections. The World Health Organization warns of a looming "post-antibiotic era" unless urgent action is taken. In India, this would manifest as surges in morbidity and mortality from basic infections. Economically, prolonged hospital stays, need for high-cost drugs, and loss of productivity will strain healthcare resources. The crisis also undermines progress in areas like tuberculosis and HIV, where drug-resistant co-infections complicate treatment. In sum, unchecked AMR jeopardizes patient outcomes and public health infrastructure alike.

V. CONCLUSION AND RECOMMENDATIONS

Antibiotic resistance in India has reached critical proportions: everyday pathogens are increasingly immune to our most common drugs. This review has documented the stark reality of near-universal resistance in key drug classes and the pathogens and infections most affected. The underlying causes – rampant misuse, OTC availability, inadequate diagnostics, and poor infection control – are well recognized and must be addressed immediately.

Recommendations: First, strengthen regulation to eliminate over-the-counter antibiotic sales: dispense antibiotics only by prescription under strict pharmacy accountability. Second, bolster hospital infection control: enforce hand hygiene, isolate patients with resistant infections, and establish stewardship committees to oversee antibiotic use. Third, educate providers and the public about AMR: emphasize completion of prescribed courses, avoid antibiotics for viral illnesses, and resist self-medication. At the national level, expand surveillance (linking laboratory networks across India) to track resistance trends and inform policy. Research and development of new antimicrobials must accelerate, but equal focus is needed on preserving existing drugs.

The alternative – a slide into "untreatable infection" territory – is unacceptable. By combining rational prescribing, tight infection control, public awareness, and continued drug innovation, India can stem the tide of resistance and safeguard public health.

REFERENCES

[1] Indian Council of Medical Research, Antimicrobial Resistance Research & Surveillance Network (AMRSN) 2024 (as reported in Times of India).

[2] Times of India, "Superbugs now resistant to key everyday antibiotics: ICMR" (Nov 2025).

[3] Times of India, "Rampant antibiotic overuse, OTC sale fuelling India's AMR crisis" (Oct 2025).

[4] Times of India, "Superbugs now resistant…" continued (Nov 2025).

[5] Times of India, "Rampant antibiotic overuse…" continued (Oct 2025).

[6] Aldea et al., Frontiers in Microbiology (2025) – comprehensive review of AST methods.

[7] Biemer JJ, "Antimicrobial Susceptibility Testing by the Kirby–Bauer Disc Diffusion Method" Ann Clin Lab Sci (1966).

[8] Cuéllar-Rodríguez et al., Clin Microbiol Infect (2009) – cost analysis of AST methods.

[9] Bauer AW et al., "Antibiotic susceptibility testing by a standardized single disk method" Am J Clin Pathol (1966).

[10] CL Gupta et al., Lancet Infect Dis (2024) – global AMR burden projections.

[11] WHO Global Antimicrobial Resistance Surveillance System (GLASS) report 2025 (WHO).