



Ceftazidime-Avibactam as Salvage Therapy for Carbapenem-Resistant *Klebsiella pneumoniae* Bacteraemia: A Narrative Review of the Stewardship Decision

Georgios Vlachos^{1*}, Ivana Horvat² and Bram Wouters³

¹Department of Infectious Diseases, Ionian University Hospital, Ioannina, Greece

²Department of Clinical Microbiology, Dalmatian Medical Centre, Split, Croatia

³Department of Clinical Pharmacy, Meuse Institute of Clinical Research, Maastricht, Netherlands

*Correspondence to: Georgios Vlachos, Department of Infectious Diseases, Ionian University Hospital, Ioannina, Greece

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Abstract

Stewardship usually means finding a reason not to use the newest agent. Carbapenem-resistant *Klebsiella* bacteraemia is the situation where that instinct is most likely to be wrong, and where acting on it costs lives. The difficulty is that resistance to the new agent emerges during treatment, so the drug is being spent as it is used.

Objectives: To review the published evidence on ceftazidime-avibactam for carbapenem-resistant Enterobacterales infection: how it compares with the alternatives, how often treatment fails, how readily resistance emerges, and what that means for the stewardship decision at the bedside.

Material and Methods: Narrative review of prospective observational cohorts, retrospective multicentre studies, randomised trials of comparator agents, resistance surveillance and society guidance indexed in PubMed and the major infectious diseases and microbiology journals. No new patient data were generated; every estimate quoted is one published by the original investigators.

Results: In a prospective multicentre cohort, 38 patients treated with ceftazidime-avibactam were compared with 99 treated with colistin. Adjusted all-cause hospital mortality 30 days after starting treatment was 9 per cent against 32 per cent (difference 23 per cent, 95% bootstrap CI 9 to 35, $p = 0.001$), with an adjusted probability of a better outcome of 64 per cent (95% CI 57 to 71). In a separate series of 37 treated patients, clinical success was 59 per cent and 30-day survival 76 per cent; microbiologic failure occurred in 27 per cent, and ceftazidime-avibactam resistance was detected in 3 of those 10 failures.

Conclusion: The comparative evidence is observational and the authors of the principal study said explicitly that it requires confirmation in a randomised trial, which has not been done. Even so, a 23 percentage point adjusted mortality difference against colistin is large enough that withholding the agent to preserve it is difficult to defend for an individual patient with bacteraemia.

Abbreviations: CAZ-AVI – Ceftazidime-Avibactam, CRE – Carbapenem-Resistant Enterobacterales, DOOR – Desirability of Outcome Ranking, IPTW – Inverse Probability of Treatment Weighting, KPC – *Klebsiella pneumoniae* Carbapenemase, MBL – Metallo-Beta-Lactamase.

Keywords: Carbapenem-Resistant Enterobacterales; Ceftazidime-Avibactam; Antimicrobial Stewardship; Bacteraemia; Emergent Resistance

Introduction

Carbapenem-resistant Enterobacterales are among the pathogens for which the World Health Organization has assigned the highest priority for new agents, and *Klebsiella pneumoniae* carbapenemase producers dominate the picture across southern and eastern Europe [1,2,3]. Bacteraemia caused by these organisms carries mortality in the region of a third to a half in most series [4,5,6].

For a decade the fallback was colistin, an agent withdrawn from routine use in the 1970s for nephrotoxicity and reintroduced because nothing else worked. Its pharmacokinetics are poor, dosing is difficult, nephrotoxicity is common, and the evidence supporting it was never strong [7,8,9].

Ceftazidime-avibactam pairs a cephalosporin with a non-beta-lactam beta-lactamase inhibitor active against Ambler class A and D serine carbapenemases, including KPC and OXA-48-like enzymes, but not against metallo-beta-lactamases [10,11,33]. In vitro surveillance confirms activity across contemporary resistant isolates [34]. This review examines how well it performs, how quickly resistance appears, and how those two facts should be weighed at the bedside.

Material and Methods

Search strategy: PubMed and the archives of the major infectious diseases, microbiology and antimicrobial chemotherapy journals were searched for studies of treatment for carbapenem-resistant Enterobacterales. Search terms combined carbapenem-resistant, *Klebsiella pneumoniae* and bacteraemia with ceftazidime-avibactam, colistin, meropenem-vaborbactam, cefiderocol, stewardship and emergent resistance. Reference lists of society guidance [12,13] were screened by hand for primary sources.

Eligibility: Studies were included if they reported clinical outcomes of treatment for carbapenem-resistant Enterobacterales infection, documented emergence of resistance during therapy, or addressed the stewardship of newer beta-lactam combinations. In vitro studies are cited where they explain a mechanism and are identified as such.

Data extraction and handling: Design, sample size, infection site, comparator, carbapenemase type where reported and the outcome estimate were extracted as published. Whether a comparison was randomised or observational and adjusted is reported alongside every figure, because the principal comparative evidence for this agent is observational and the confounding by indication runs in both directions. No pooling or re-analysis was performed. Figures 1 to 4 and Tables 1 and 2 reproduce published values only. This is a narrative synthesis and was not registered as a systematic review.

Results

The principal comparison: Patients treated first with ceftazidime-avibactam ($n = 38$) or colistin ($n = 99$) for carbapenem-resistant Enterobacterales infection were drawn from a prospective multicentre observational consortium. Bloodstream infections accounted for 46 per cent of cases and respiratory infections 22 per cent. Unadjusted 30-day all-cause in-hospital mortality was 3 of 38 (8 per cent) against 33 of 99 (33 per cent); after inverse probability of treatment weighting the adjusted figures were 9 and 32 per cent, a difference of 23 per cent (95% bootstrap CI 9 to 35, $p = 0.001$) (Figure 1) [14].

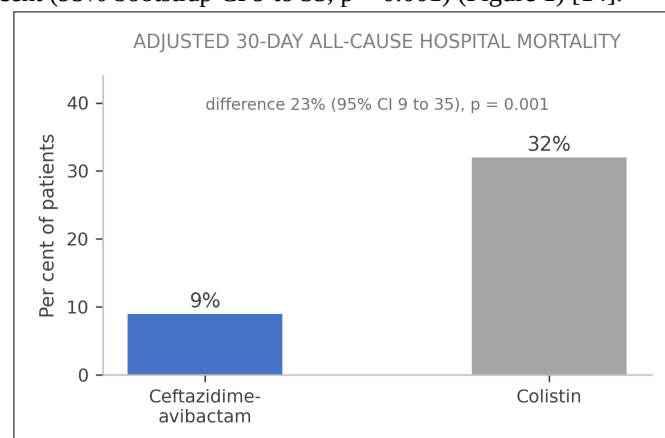


Figure 1: Adjusted 30-day all-cause hospital mortality, ceftazidime-avibactam against colistin [14]

How the analysis was framed: The investigators used a desirability of outcome ranking approach, scoring disposition at day 30 as home, alive but not home, or death in hospital. The adjusted probability that a patient treated with ceftazidime-avibactam would have a better outcome than one treated with colistin was 64 per cent (95% CI 57 to 71), and partial credit analyses indicated uniform superiority [14]. The authors nonetheless concluded that the agent may be a reasonable alternative to colistin and that the finding required confirmation in a randomised trial. That trial has not been performed.

Failure is common even when it works: A single-centre series of 37 patients treated with ceftazidime-avibactam reported clinical success in 59 per cent (22 of 37) and 30-day survival in 76 per cent (28 of 37). Microbiologic failure occurred in 27 per cent (10 of 37), and among those with clinical success, 23 per cent (5 of 22) had recurrence of infection within 90 days (Figure 2) [15]. These are considerably less impressive numbers than the comparative mortality figure, and they describe the same drug. Both are true: it is better than colistin and it fails often.

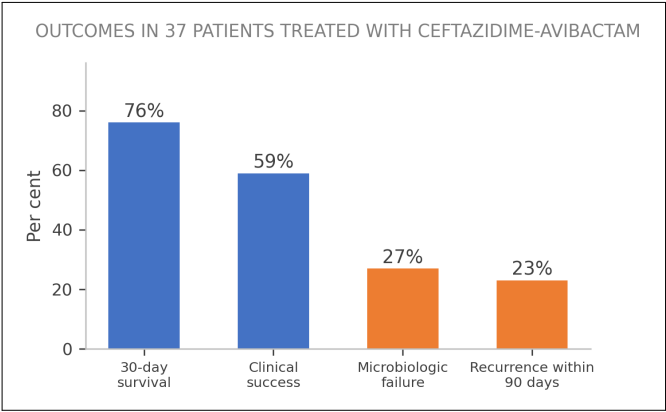


Figure 2: Outcomes among 37 patients treated with ceftazidime-avibactam for carbapenem-resistant infection [15]

Resistance emerges during treatment: Ceftazidime-avibactam resistance was detected in 3 of the 10 microbiologic failures in that series, that is 30 per cent of failures and 8 per cent of all treated patients (Figure 3) [15]. The mechanism is typically mutation within the KPC gene rather than acquisition of a new element, which means the resistant organism arises from the population being treated [16,17]. Combination with colistin does not suppress it: in vitro work found no potentiation of killing and no suppression of emergent resistance [18].

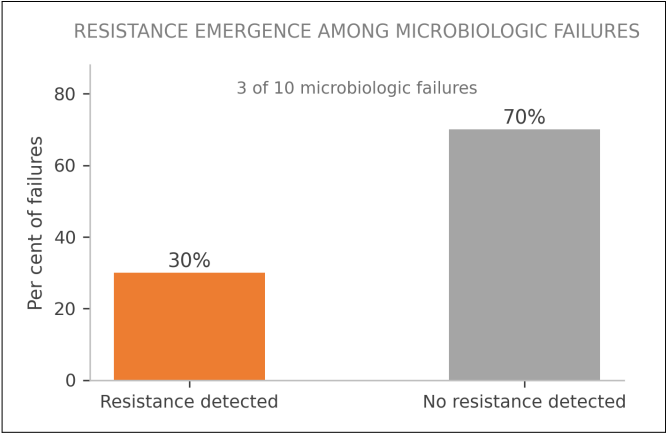


Figure 3: Detection of ceftazidime-avibactam resistance among microbiologic failures [15]

Other comparative data: A retrospective two-centre study of 61 patients with carbapenem-resistant bacteraemia found clinical success in 46.8 per cent of those receiving ceftazidime-avibactam against 20.4 per cent receiving colistin ($p = 0.047$), with an adjusted 14-day mortality hazard ratio of 0.32 (95% CI 0.10 to 0.99, $p = 0.049$) while the crude comparison was not significant (Figure 4) [19]. Larger observational series and meta-analyses point the same way [20,21,22], and randomised evidence exists for the comparator agent meropenem-vaborbactam against best available therapy [23]. Cefiderocol occupies a different position, with a randomised trial in which mortality was numerically higher in the cefiderocol arm [24].

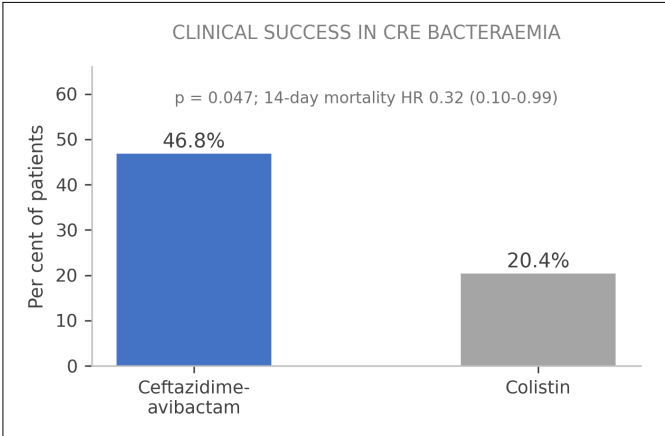


Figure 4: Clinical success in carbapenem-resistant bacteraemia, ceftazidime-avibactam against colistin [19]

Study	Design and cohort	Principal reported finding
van Duin et al. 2018 [14]	Prospective cohort, 38 CAZ-AVI vs 99 colistin	Adjusted 30-day mortality 9% vs 32%; DOOR probability 64%
Shields et al. 2018 [15]	Series of 37 treated patients	Clinical success 59%; resistance in 3 of 10 microbiologic failures
Alraddadi et al. 2021 [19]	Retrospective, 61 patients with bacteraemia	Clinical success 46.8% vs 20.4%; 14-day mortality HR 0.32
Tumbarello et al. 2019 [20]	Multicentre observational, KPC bacteraemia	Lower mortality with CAZ-AVI-containing regimens
Wunderink et al. 2018 [23]	TANGO II randomised trial	Meropenem-vaborbactam against best available therapy
Bassetti et al. 2021 [24]	CREDIBLE-CR randomised trial	Higher numerical mortality in the cefiderocol arm
Shields et al. 2017 [16]	Genomic analysis of failures	KPC mutations arising under treatment pressure

Table 1: Principal studies reviewed, with the finding as reported by the original investigators

The stewardship question: Antimicrobial stewardship exists to restrain the use of agents whose value is collective and depletable. Applied to this situation it produces an uncomfortable structure: the individual patient in front of the clinician has a bloodstream infection with mortality around a third, the newer agent is associated with substantially lower mortality than the alternative, and every course given carries some probability of selecting resistance that will disadvantage future patients [25,26]. Guidance from both European and North American societies now recommends newer beta-lactam-beta-lactamase inhibitor combinations in preference to colistin-based regimens for susceptible carbapenem-resistant isolates [12,13].

What stewardship can still do: The lever is not withholding but precision. Rapid molecular identification of the carbapenemase type determines whether the agent can work at all, since metallo-beta-lactamase producers are not covered and require a different approach [10,27,28]. De-escalation once susceptibilities return, source control, appropriate duration and avoidance of empirical use in patients without documented resistant organisms, a balance set out in reviews of when each

newer agent should be reached for [35], all reduce exposure without denying treatment to those who need it [25,29,30]. Susceptibility testing for this agent has its own technical difficulties that laboratories need to anticipate [31,32].

Consideration	What the evidence shows	Implication
Versus colistin	Adjusted mortality 9% vs 32%	Observational; awaiting randomised confirmation [14]
Absolute performance	Clinical success 59%; recurrence 23%	Better than the alternative, not reliable [15]
Emergent resistance	3 of 10 microbiologic failures	The drug is consumed as it is used [15,16]
Combination with colistin	No potentiation in vitro	Does not suppress emergent resistance [18]
Carbapenemase type	No activity against metallo-enzymes	Molecular identification is decisive [10,27]
Stewardship lever	Precision rather than restriction	De-escalate, shorten, avoid empirical use [25,29]

Table 2: Factors bearing on the decision to use ceftazidime-avibactam and their implications

Discussion

The comparative result is large. A 23 percentage point adjusted difference in 30-day mortality is not a marginal gain, and it was produced using analytic methods designed to handle confounding by indication rather than to ignore it [14]. Set against colistin, an agent with poor pharmacokinetics and substantial nephrotoxicity, the direction is unsurprising even if the magnitude is.

It is nonetheless observational, and the investigators said so themselves. Patients given a newly available agent early in its availability differ from those given colistin, in ways that weighting can adjust for only if they were measured. Severity at the moment of prescribing, the judgement that a patient was salvageable, and access to the drug through a research-active unit are all plausible confounders and all imperfectly captured. The call for a randomised trial was reasonable and it has not been answered, which is itself worth noting: once a drug is believed to work this much better, randomising against colistin becomes difficult to justify.

The second finding is the one that complicates the stewardship argument rather than settling it. In the series reporting detailed microbiology, clinical success was 59 per cent, a quarter of successes recurred within 90 days, and resistance emerged in nearly a third of microbiologic failures [15,16]. Emergent resistance through KPC mutation means each course carries a probability of producing an organism against which the agent no longer works. That is a genuine collective cost, not a theoretical one, and it accrues to the same institution that used the drug.

Where does that leave the decision. The conventional stewardship instinct, to preserve the newer agent and use the older one first, does not survive contact with a 23 percentage point mortality difference in a patient with bacteraemia.

Preserving a drug by allowing a patient to die of a susceptible organism is not stewardship. But the corollary matters equally: the justification for using it rests on the patient having a documented carbapenem-resistant organism of a susceptible carbapenemase type and a serious infection. Empirical use in patients who do not meet that description spends the collective resource without the individual benefit that justified spending it, and that is the pattern rapid diagnostics and de-escalation exist to prevent [25,27,29].

Study limitations

The limitations belong to the underlying literature. The principal comparative evidence is observational, from 38 treated patients, with adjustment able to address only measured confounders [14]. Most patients in that cohort received additional anti-CRE agents, so the comparison is between regimens rather than between single drugs. Resistance and outcome data come largely from single centres with local carbapenemase epidemiology that does not generalise, particularly to settings dominated by metallo-beta-lactamases [15,16,27]. Colistin dosing was not standardised across studies and was often below currently recommended regimens, which flatters the comparator arm less than it should [7,9]. Definitions of clinical success differ substantially between series [15,19,20]. Susceptibility testing methods for this agent are technically demanding and were not uniform [31,32]. And there remains no randomised comparison against colistin, which was the stated requirement of the investigators who produced the principal result [14].

Conclusion

For a patient with bacteraemia caused by a carbapenem-resistant, ceftazidime-avibactam-susceptible *Klebsiella*, the evidence favours using the agent rather than reserving it. The adjusted mortality difference against colistin is 23 percentage points, and no stewardship argument outweighs that for an individual with a bloodstream infection. What the same evidence shows is that the drug fails in around four patients in ten, that a fifth of successes recur, and that resistance emerges under treatment in a substantial minority of failures. The correct reading is not that stewardship is irrelevant here but that its target has moved: away from withholding the agent from the patient who needs it, and towards ensuring that everyone who receives it has a documented organism it can actually treat.

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Conflict of Interest

The authors declare no competing interests.

Ethics Statement

This article reviews previously published studies only. No new patient data were collected and no participants were recruited, so ethical approval was not required.

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