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Direct Application of Amikacin in Pressure Sores - A Prospective Cohort Study

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Abstract

Amikacin *is an antibiotic medication used for a number of bacterial infections.[8] This includes joint infections, intra-abdominal infections, meningitis, pneumonia, sepsis, and urinary tract infections.[8] It is also used for the treatment of multidrug-resistant tuberculosis.[9] It is used by injection into a vein using an IV or into a muscle.[8]*

Amikacin, like other aminoglycoside antibiotics, can cause hearing loss, balance problems, and kidney problems.[8]Other side effects include paralysis, resulting in the inability to breathe.[8] If used during pregnancy it may cause permanent deafness in the baby.[8][1]Amikacin works by blocking the function of the bacteria's 30S ribosomal subunit, making it unable to produce proteins.[8]

INTRODUCTION

Amikacin was patented in 1971, and came into commercial use in 1976.[10][11] It is on the World Health Organization's List of Essential Medicines.[12] It is derived from kanamycin.[8]

Amikacin is most often used for treating severe infections with multidrug-resistant, aerobic Gram-negative bacteria, especially *Pseudomonas*, *Acinetobacter*, *Enterobacter*, *E. coli*, *Proteus*, *Klebsiella*, and *Serratia*.[13] The only Gram-positivebacteria that amikacin strongly affects are *Staphylococcus*[13] and *Nocardia*.[14]Amikacin can also be used to treat non-tubercular mycobacterial infections and tuberculosis (if caused by sensitive strains) when first-line drugs fail to control the infection.[8] It is rarely used alone.[15]

It is often used in the following situations:[8]

- Bronchiectasis[16]
- Bone and joint infections
- Granulocytopenia, when combined with ticarcillin, in people with cancer[17]
- Intra-abdominal infections (such as peritonitis) as an adjunct to other medicines, like clindamycin, metronidazole, piperacillin/tazobactam, or ampicillin/sulbactam
- Meningitis:
  - for meningitis by *E. coli*, as an adjunct to imipenem
  - for meningitis caused by *Pseudomonas*, as an adjunct to meropenem
  - for meningitis caused by *Acinetobacter*, as an adjunct to imipenem or colistin
  - for neonatal meningitis caused by *Streptococcus agalactiae* or *Listeria monocytogenes*, as an adjunct to ampicillin
  - for neonatal meningitis caused by Gram negative bacteria such as *E. coli*, as adjunct to a 3rd-generation cephalosporin
- Mycobacterial infections, including as a second-line agent for active tuberculosis.[18] It is also used for infections by *Mycobacterium avium*, *M. abcessus*, *M. chelonae*, and *M. fortuitum*.
- *Rhodococcus equi*, which causes an infection resembling tuberculosis

- Respiratory tract infections, including as an adjunct to beta-lactams or carbapenemfor hospital-acquired pneumonia
- Sepsis, including that in neonates,[13] as an adjunct to beta-lactams or carbapenem
- Skin and suture-site infections[13]
- Urinary tract infections that are caused by bacteria resistant to less toxic drugs (often by *Enterobacteriaceae* or *P. aeruginosa)*

Amikacin may be combined with a beta-lactam antibiotic for empiric therapy for people with neutropenia and fever.[8]

Amikacin irreversibly binds to 16S rRNAand the RNA-binding S12 protein of the 30S subunit of prokaryotic ribosome and inhibits protein synthesis by changing the ribosome's shape so that it cannot read the mRNA codons correctly.[13][25] It also interferes with the region that interacts with the wobble base of the tRNAanticodon.[26] It works in a concentration-dependent manner, and has better action in an alkaline environment.[7]

At normal doses, amikacin-sensitive bacteria respond within 24–48 hours.[13]

Resistance

Amikacin evades attacks by all antibiotic-inactivating enzymes that are responsible for antibiotic resistance in bacteria, except for aminoacetyltransferase and nucleotidyltransferase.[27] This is accomplished by the L-hydroxyaminobuteroyl amide (L-HABA) moiety attached to N-1 (compare to kanamycin, which simply has a hydrogen), which blocks the access and decreases the affinity of aminoglycoside-inactivating enzymes.[27][28][29] Amikacin ends up with only one site where these enzymes can attack, while gentamicin and tobramycin have six.[15]

Bacteria that are resistant to streptomycinand capreomycin are still susceptible to amikacin; bacteria that are resistant to kanamycin have varying susceptibility to amikacin. Resistance to amikacin also confers resistance to kanamycin and capreomycin.[30]

Resistance to amikacin and kanamycin in *Mycobacterium tuberculosis*, the causative agent of tuberculosis, is due to a mutation in the *rrs* gene, which codes for the 16S rRNA. Mutations such as these reduce the binding affinity of amikacin to the bacteria's ribosome.[31] Variations of aminoglycoside acetyltransferase (AAC) and aminoglycoside adenylyltransferase(AAD) also confer resistance: resistance in *Pseudomonas aeruginosa* is caused by AAC(6')-IV, which also confers resistance to kanamycin, gentamicin, and tobramycin, and resistance in *Staphylococcus aureus*and *S. epidermidis* is caused by AAD(4',4*)*, which also confers resistance to kanamycin, tobramycin, and apramycin.some strains of *S. aureus* can also inactivate amikacin by phosphorylating it.[17]

Amikacin is not absorbed orally and thus must be administered parenterally. It reaches peak serum concentrations in 0.5–2 hours when administered intramuscularly. Less than 11% of the amikacin actually binds to plasma proteins. It is distributed into the heart, gallbladder, lungs, and bones, as well as in bile, sputum, interstitial fluid, pleural fluid, and synovial fluids. It is usually found at low concentrations in the cerebrospinal fluid, except when administered intraventricularly.[8] In infants, amikacin is normally found at 10–20% of plasma levels in the spinal fluid, but the amount reaches 50% in cases of meningitis.[13] It does not easily cross the blood–brain barrier or enter ocular tissue.[7]

While the half-life of amikacin is normally two hours, it is 50 hours in those with end-stage renal disease.[15]

The majority (95%) of amikacin from an intramuscular or intravenous dose is secreted unchanged via glomerular filtration and into the urine within 24 hours.[8][15] Factors that cause amikacin to be excreted via urine include its relatively low molecular weight, high water solubility, and unmetabolized state.

MATERIALS AND METHODS

30 patients were taken who came in PMR OPD of New building PMSSY Building of GSVM Medical college and LLR Hospital Kanpur U.P. In all patients Dressing using normal 0.9 % saline was done followed by local application of amikacin directly into the wound ulcer.

The study was approved by the Institutional Review Board and was conducted according to the Declaration of Helsinki and the GCP; all patients signed informed consent.

Male or female patients aged 20–50 years

Exclusion criteria include patient having kidney problems , ear problems and those having allergies to amikacin.

The primary study objective was to confirm the superior efficacy of amikacin as compared to other similar antibiotics and to it direct application of it helpful or not.

DISCUSSION

Amikacin is an antimicrobial with activity against more resistant gram-negative bacilli such as Acinetobacter baumanii and Pseudomonas aeruginosa. It also has excellent activity against most aerobic gram-negative bacilli from the Enterobacteriaceae family, including Nocardia sp. and some Mycobacterium spp. (M. avium-intracellulare, M. chelonae, and M. fortuitum). Unlike gentamicin, amikacin does not provide synergistic activity against Enterococcus faecium when combined with beta-lactam antibiotics as in our study we find gram negative bacteria basically in almost all cases since many cases were neglected ones and there was lack of care of affected wounds (1,2,3,4) .

Amikacin binds to the 30 S bacterial ribosome subunit, resulting in interference with a reading of the genetic code and inhibition of protein synthesis, e.g., elicits premature protein termination and incorporation of incorrect amino acid. Amikacin, as well as the rest of the aminoglycosides, are generally bacteriocidal and probably have an additional mechanism of action, which as yet remains undetermined. Aminoglycosides demonstrate bacterial killing that is concentration-dependent and also have a post-antibiotic effect(6,7,8,9).

In our study we finded out E coli , Proteus and all types of gram negative bacteria in the wound ulcer.

Result and Conclusion

It is observed that the local therapy as an adjuvant is cost effective and without any significant local or systemic adverse effects in the prevention of dirty and contaminated pressure ulcers. But it was also observed that it did not have sustained effect for prolonged period beyond its time of administration.It may be suggested that a further combination of suction drainage of the subcutaneous DT along with local antibiotic treatment may have added advantage in further preventing contaminated wounds.Wounds produce a moist, warm, and nutritious environment that is perfect for the colonization and proliferation of bacteria. Infections from the outside environment significantly slow the healing of wounds. As a result, dressings for wounds should

prevent harmful bacteria from the environment from infiltrating the wound's surface.This amikacin in turn reduces this moist state. In all the 30 cases nearly all patients were healed and also there was less recurrences.

Out of 30 cases no one reported any adverse effects this indicates that the drug can now be used on daily basis without any complications as observed in this study.

LIMITATIONS

since there were only 30 patients in the study. We advocate that future studies should atleast include 100 or more patients so there shall be less follow up problem as in our study few 4 patients didnt followed or showed up

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Institutional Review Board Statement

Not applicable.

Informed Consent Statement

consent were taken from all patients who participated in the study.

Data Availability Statement

The data presented in this study are available within the text.

Conflicts of Interest

The authors declare no conflicts of interest.

REFERENCES

1. Wenzel, R.; Perl, T. The significance of nasal carriage of Staphylococcus aureus and the incidence of postoperative wound infection. *J. Hosp. Infect.* **1995**, *31*, 13–24. [Google Scholar] [CrossRef] [PubMed]

2. Endo A, Nemoto A, Hanawa K, Maebayashi Y, Hasebe Y, Kobayashi M, Naito A, Kobayashi Y, Yamamoto S, Isobe K. Relationship between amikacin blood concentration and ototoxicity in low birth weight infants. J Infect Chemother. 2019 Jan;25(1):17-21.[PubMed]

3. Nolt VD, Pijut KD, Autry EB, Williams WC, Burgess DS, Burgess DR, Arora V, Kuhn RJ. Amikacin target achievement in adult cystic fibrosis patients utilizing Monte Carlo simulation. Pediatr Pulmonol. 2019 Jan;54(1):33-39. [PubMed]

4. Kulengowski B, Clark JA, Burgess DS. Killing activity of meropenem in combination with amikacin against VIM- or KPC-producing Enterobacteriaceae that are susceptible, intermediate, or resistant to amikacin. Diagn Microbiol Infect Dis. 2019 Apr;93(4):372-375.[PubMed]

5. Block M, Blanchard DL. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jul 17, 2023. Aminoglycosides. [PubMed]

6. Sturkenboom MGG, Simbar N, Akkerman OW, Ghimire S, Bolhuis MS, Alffenaar JC. Amikacin Dosing for MDR Tuberculosis: A Systematic Review to Establish or Revise the Current Recommended Dose for Tuberculosis Treatment. Clin Infect Dis. 2018 Nov 28;67(suppl_3):S303-S307. [PubMed]

7 Illamola SM, Huynh HQ, Liu X, Bhakta ZN, Sherwin CM, Liou TG, Carveth H, Young DC. Population Pharmacokinetics of Amikacin in Adult Patients with Cystic Fibrosis. Antimicrob Agents Chemother. 2018 Oct;62(10) [PMC free article] [PubMed]

8. Liu X, Smits A, Wang Y, Renard M, Wead S, Kagan RJ, Healy DP, De Cock P, Allegaert K, Sherwin CMT. Impact of Disease on Amikacin Pharmacokinetics and Dosing in Children. Ther Drug Monit. 2019 Feb;41(1):44-52. [PubMed]

9. Arnold A, Cooke GS, Kon OM, Dedicoat M, Lipman M, Loyse A, Chis Ster I, Harrison TS. Adverse Effects and Choice between the Injectable Agents Amikacin and Capreomycin in Multidrug-Resistant Tuberculosis. Antimicrob Agents Chemother. 2017 Sep;61(9) [PMC free article] [PubMed]

10. Wrohan I, Redwood L, Ho J, Velen K, Fox GJ. Ototoxicity among multidrug-resistant TB patients: a systematic review and meta-analysis. Int J Tuberc Lung Dis. 2021 Jan 01;25(1):23-30. [PubMed]

11. Anniko, M.,D.Berggren and S.Holm.1998. Tissue binding kinetics of tobramycin:an experimental study in thd mouse ear in vitro. Acta otolaryngol. 105 : 120-25.

12. Bagley, D.H.,J.Maclowry,R.M.Beazely, C.Gorschboth, and A.S. Ketcham.1978. Antibiotic concentration in human wound fluid after intravenous administration. Ann.Surg.188:202-208.

13. Bergen,T.,and A.Engeset. Penetration of antibiotics to tissues and infected foci. Scand J.Gastroenterol. Suppl. 19:29-34.

14. Berger,S. A.,M.Barza,J.Haher,D.bell,S.waintraub,M.L.Burtyk, and A.kane.1981.Penetration of antibiotics into decubitus ulcers.J.Antimicrob.Chemother.7:193-195.

15. Cull, J.G.,and O.H. Smith.1973. A preliminary note on demographic and personality correlates of decubitus ulcer incidence. J.Psychol.85:225-227.

16. Galpin,J.E.,A.W.Clow , A.S.Bayer, and L.,B.Guze. 1976. Sepsis associated with decubitus ulcers. Am.J.Med.61:346-350.