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Acinetobacter: Its role in Respiratory colonization, Ventilator Associated Pneumonias and the role of Hospital Infection Control measures in prevention of the same

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Abstract
The most common clinical condition associated with Acinetobacter sp. is hospital-acquired pneumonia (HAP), particularly for patients receiving mechanical ventilator assistance. Multidrug-resistant Acinetobacter baumannii (MDRAB) is an important and emerging hospital-acquired pathogen worldwide. The basic purpose of this study was to evaluate (on the basis of evidence), whether we were justified in treating respiratory tract Acinetobacter sp. isolates with high grade antibiotics, or were we overprescribing and inadvertently contributing to antibiotic resistance. This was evaluated by calculating the relative risks of development of (Probable) Ventilator Associated Pneumonias in mechanically ventilated patients, whose Respiratory tract is colonized with Acinetobacter. Also, the Antibiotic Resistance patterns of hospital Acinetobacter isolates were studied. In addition, the role of basic infection control measures in controlling the rates of Acinetobacter colonisations and infections in ICU patients was studied.
Results: *(1) 20% of mechanically ventilated patients was colonised with Acinetobacter sp., of which, only 5.8% developed P-VAPs (2) Relative Risk for development of P-VAP in mechanically ventilated patients colonised with Acinetobacter sp. was 4.23 times higher than those not harbouring Acinetobacter sp. in their respiratory tract. (3) Sensitivity to antibiotics of Hospital Respiratory Acinetobacter strains was as follows: Colistin (100%)> Levofloxacin (64.4%)> Meropenem (60%)> Minocycline (24.4%)> PipTaz (13.3%). All hospital strains were MDRs (Multi Drug Resistant). (4) Relative risk of developing an AP-VAP (Acinetobacter P-VAP) can be reduced by 2.35 times by implementing Infection Control measures.*
Conclusion: *Using antibiotic coverage for all respiratory Acinetobacter sp. isolates, is probably counterproductive, since, it increases the selective antibiotic pressure, which enables, emergence of multi-drug resistance in Acinetobacter sp. Also, it was found that strict implementation of simple hospital infection control measures could reduce the colonization rates by half, and P-VAP rates by more than 2 times.*
Keywords- *Acinetobacter sp.,* VAP *(Ventilator Associated Pneumonia), HAP (Hospital Acquired Pneumonia), CRA (Carbapenem Resistant Acinetobacter)*

Introduction

Nosocomial pneumonia or Hospital Acquired Pneumonia (HAP) refers to any pneumonia contracted by a patient in a hospital at least 48–72 hours after being admitted (i.e., the patient neither presented with pneumonia, nor was in the incubation period of the same, at the time of admission). HAP is the second most common nosocomial infection (after urinary tract infections) and accounts for 15–20% of the total. It

is the most common cause of death among nosocomial infections and is the primary cause of death in intensive care units. HAP typically lengthens a hospital stay by 1–2 weeks. [1].

Ventilator-associated pneumonia (VAP) is a type of nosocomial pneumonia, defined as pneumonia that occurs 48-72 hours or thereafter following endotracheal intubation, characterized by the presence of a new or progressive infiltrate, signs of systemic infection (fever, altered white blood cell count), changes in sputum characteristics, and detection of a causative agent. VAP contributes to approximately half of all cases of hospital-acquired pneumonia[2]. VAP is the most common nosocomial infection in mechanically ventilated patients, estimated to occur in 9-27 % of all mechanically ventilated patients, with the highest risk being early in the course of hospitalization [2].

Recently, the Centers for Disease Control and Prevention (CDC) rolled out new surveillance criteria for all Ventilator Associated Events (VAE), including possible or probable VAP, for patients >18 years of age[2]. The goals were to capture other common complications of ventilator care, to improve objectivity of surveillance and to allow comparability across centers for public reporting.

In humans, *Acinetobacter* has been isolated from all culturable sites. It can form part of the bacterial flora of the skin, particularly in moist regions such as the axillae, groin, and toe webs, and up to 43% of healthy adults can have colonization of skin and mucous membranes, with higher rates among hospital personnel and patients[3]. The respiratory tract is an important site of colonization and is the most frequent site of infection.

Acinetobacter colonization has been reported from the nares, nasopharynx, and tracheostomy sites. Rates of colonization increase during ICU stays. The frequency of nasal colonization among healthy persons has been variable[11].

Acinetobacter is an opportunistic pathogen that typically causes serious infection in immune-compromised hosts. The organism is encapsulated and has a cell wall containing lipopolysaccharides, but the effect of the lipopolysaccharides in humans is not well-understood[11]. The prevalence of health care associated infections caused by *Acinetobacter* is increasing among patients in intensive care units (ICUs) and immunocompromised hosts. The most common clinical condition associated with these microorganisms is hospital-acquired pneumonia (HAP), particularly for patients receiving mechanical ventilator assistance[3]. Nosocomial transmission is responsible for the vast majority of *A.baumannii* infections, and is related both to the ability of the organism to survive in the environment and the organism's resistance to the major groups of antibiotics, resulting in a selective advantage in settings such as ICUs, where broad-spectrum antibiotic use is commonplace[10]. Multidrug-resistant *Acinetobacter baumannii* (MDRAB) is an important and emerging hospital-acquired pathogen worldwide [4].

When *A.baumannii* is isolated from patient-derived biological samples distinction is often made between colonisation and active infection (associated features of sepsis and/or a positive blood culture). In contrast to colonisation, infection was defined as isolation of *A.baumannii* from any biological site in conjunction with a compatible clinical picture warranting treatment with antibiotics effective against *A.baumannii*. Patients at risk are often critically ill with multiple co-morbidities, concurrent infections, and on prolonged courses of antibiotics, often making it difficult to distinguish between colonisation and infection. Moreover, colonisation is a risk factor for subsequent infection. The absence of multidrug-resistant strains in the community compared with 36.6% prevalence among hospital isolates suggested that the reservoir for epidemic strains resides in the hospital environment itself[8]. In the hospital environment, it has been isolated from food (including hospital food), ventilator equipment, suctioning equipment, infusion pumps, sinks, stainless steel trolleys, pillows, mattresses, tap water, bed rails, humidifiers, soap dispensers, and other sources[11].

The Standard VAP- Bundle Care Measures, as recommended by CDC-NHSN, include: Head

elevation at 30-40°, Daily assessment of suitability of extubation (i.e., extubation as soon as possible), Maintainance of oral hygine using 0.2% Chlorhexidine Gluconate washes before intubation and every 4-6 hrly thereafter, frequent subglottic suctioning of endotracheal secretions, Prophylactic antacids for reduction of gastric pH, Interruption of sedation, Promoting trials of Non Invasive Positive Pressure Ventilation, to reduce duration of intubation, DVT prophylaxis, and early ambulation and deep breathing post extubation. Outbreaks of *Acinetobacter* have been controlled by education on hand washing, patient and staff cohorting, implementing contact precautions, minimizing use of broad-spectrum antibiotics, closing hospital units, discharging colonized patients, and decontaminating the environment[11].

The following prospective study was carried out in a tertiary care hospital in central India (Jabalpur, Madhya Pradesh) with the aims of establishing the baseline rates of *Acinetobacter* colonization of the respiratory tract of mechanically ventilated patients, in this hospital, its effect on the rates of APVAP (*Acinetobacter*- Probable VAP), and the effect of basic infection control measures (in addition to the standard VAP- bundle care measures), in reducing these rates. The basic purpose of this study was to evaluate (on the basis of evidence), whether we were justified in treating respiratory tract *Acinetobacter sp*. isolates with high grade antibiotics, or were we overprescribing and inadvertently contributing to antibiotic resistance.

Objectives

1. To find the rate of colonization of the respiratory tract by *Acinetobacter* in mechanically ventilated adult patients admitted to the ICU in this hospital.
2. To determine the Relative risk of development of (Probable) Ventilator Associated Pneumonias in mechanically ventilated patients, whose Respiratory tract is colonized with *Acinetobacter.*
3. To evaluate the relative risk of development of P-VAP (P-VAP) in patients having bacterial respiratory co infections superimposed over *Acinetobacter* colonization.
4. To determine and compare the incidences of Nosocomial Pneumonias due to *Acinetobacter sp.* in mechanically ventilated as well as non-ventilated patients.
5. To evaluate the Antibiotic Resistance patterns of hospital *Acinetobacter* isolates.
6. To evaluate whether all isolations of *Acinetobacter sp.* from the respiratory tracts of mechanically ventilated patients warrant antimicrobial therapy.
7. To determine the role of basic infection control measures in controlling the rates of *Acinetobacter* colonisations and infections in ICU patients.

Methodology

Period of Study: 10 months (January 2017 to October 2017)

Type of Study: Prospective, Cohort, Interventional study

Active Screening was done by collection of Endotracheal Tube (ET) and/or Tracheostomy Tube (TT) secretions from all mechanically ventilated patients, on the day of intubation, and all these specimens were processed for gram's staining and cultured on Blood Agar and MacConkey's Agar. Sabouraud's Dextrose Chloramphenicol Agar was used if Gram's stain showed Gram positive yeast cells. Growth was further identified by routine biochemical testing and antibiotic susceptibility testing was done by modified Kirby Bauer's Disk Diffussion Method and interpreted as per CLSI 2017 guidelines.

Sputum and Bronchoalveolar Lavage samples from adult patients admitted to the ICU, who were not mechanically ventilated, but had acquired a nosocomial pneumonia (defined as fever >38°C or <36° with a TLC >12,000/cumm or <4000/cumm with new or changing chest infiltrates on CXR, appearing >48 hrs after hospital admission), were also collected, so as to determine the role of *Acinetobacter sp.* in nosocomial pneumonias in non- mechanically ventilated patients.

Acinetobacter infection was defined as isolation of *A.baumannii-calcoaceticus complex* from any biological site in conjunction with a compatible clinical picture warranting treatment with antibiotics effective against *Acinetobacter sp.* Patients at risk are often critically ill with multiple co-morbidities, concurrent infections, and on prolonged courses of antibiotics, often making it difficult to distinguish between colonisation and infection. Moreover, colonisation is a risk factor for subsequent infection. Active surveillance for Ventilator Associated Events (VAE), which includes VAC, I-VAC and P-VAP was carried out over the entire period of the study as per the new CDC-NHSN guidelines, wherein,

- **Ventilator Associated Condition (VAC)** was defined as a ventilated patient having period of at least 2 days of stable or decreasing ventilator settings (daily minimum positive end-expiratory pressure [PEEP] or fraction of inspired oxygen [FiO_2]) followed by consistently higher settings for at least 2 additional calendar days.
- **Infection/ Inflammation Related VAC (I-VAC)** was defined as a patient having VAC, along with Signs of infection/inflammation (temperature > 38°C or <36°C or white cell count > 12000/cumm or <4000/cumm) on atleast 1 occasion within the window period of 5 days (i.e., within 2 days before or after the date of worsening of oxygenation) AND administration of one or more new antibiotics for at least 4 days
- **Possible VAP (Possible Ventilator Associated Pneumonia)** was defined as Patients with an IVAC having either purulent secretions alone (as per Gram's staining criteria of >25 PMNs/ LPF and <10 epithelial cells/LPF) or pathogenic cultures alone.
- **Probable Ventilator Associated Pneumonia (P-VAP)** was defined as Patients with an IVAC having both, purulent secretions alone (as per Gram's staining criteria of >25 PMNs/ LPF and <10 epithelial cells/LPF) AND pathogenic cultures. If positive cultures could not be obtained (due to the presence of atypical organisms or viruses), then P-VAP can also be defined on the basis of suggestive histopathological features, positive pleural-fluid or lung tissue cultures, or non-culture based diagnostic tests for legionella and selected viruses.
- As per the new CDC-NHSN criteria, Chest radiograph findings were excluded from the definitions, because of their subjectivity without increased accuracy.

The standard CDC-NHSN recommended VAP-Prevention Bundle measures, which include, Head elevation at 30-40°, Daily assessment of suitability of extubation (i.e., extubation as soon as possible), Maintainance of oral hygine using 0.2% Chlorhexidine Gluconate washes before intubation and every 4-6 hrly thereafter, frequent subglottic suctioning of endotracheal secretions, Prophylactic antacids for reduction of gastric pH, Interruption of sedation, Promoting trials of Non Invasive Positive Pressure Ventilation, to reduce duration of intubation, DVT prophylaxis, and early ambulation and deep breathing post extubation, were strictly adhered to, throughout the study period. In addition to these, a few basic HIC measures were implemented, for patients harbouring *Acinetobacter sp.*, which included:

- Marking of files of these patients with pink labels
- Patient and staff cohorting
- Hanging of Hand Hygiene and Std. Universal Precautions signages on the beds of these patients
- Strict adherence to contact and droplet precautions and hand hygiene while handling these patients
- Drawing of Curtains during oral suctioning and all aerosol generating procedures.
- Twice daily cleaning of patient surroundings with 1% Hypochlorite followed by Peroxide based cleaning.

These measures were implemented in the last 2 months (September and October 2017) of the study, and their effects on the rates of

Acinetobacter colonization and infection were analysed.

Results and Discussion

A total of 189 patients received mechanical ventilation in the ICU of this hospital from Jan-2017 to Oct- 2017, out of which *Acinetobacter sp.* was isolated in the respiratory secretions as follows:

Table 1: *Acinetobacter* Colonisation Rate in mechanically Ventilated Patients

No.	Mon	Sam	Acinetobacter +ve			Age(yr)		Rate (%)
			T	M	F	< 60	>60	
1	JAN	12	0	0	0	0	0	0
2	FEB	16	7	2	3	4	1	43.75
3	MAR	19	4	2	2	0	4	21.05
4	APR	23	2	1	2	1	2	8.70
5	MAY	18	8	6	1	2	5	44.44
6	JUN	22	4	3	1	3	1	18.18
7	JUL	27	5	1	3	3	1	18.52
8	AUG	25	5	3	2	3	2	20.00
9	SEP	11	2	1	0	0	1	18.18
10	OCT	16	1	0	1	1	0	6.25
	T	**189**	**39**	**19**	**15**	**17**	**17**	**20.11**
	Bf	**162**	**35**					**21.61**
	After	**27**	**03**					**11.11**

[Mon= Month, Sam= Sample, T= Total, Rate= % of ventilated patients, who are colonized with Acinetobacter, Bf= Before Intervention (Jan to Aug), After= After Intervention (Oct- Nov)]

1. Overall Rate of Respiratory tract colonization with *Acinetobacter sp.* in mechanically ventilated patients over a period of 10 months was found to be about 20%. [Table 1].
2. In the period prior to the introduction of basic HIC measures (i.e., Jan to Aug) Rate of colonisation was nearly 22%, which was brought down to 11% post strict implementation of strict HIC measures (sept-oct). [Table 1].
3. *Acinetobacter* respiratory colonization was nearly equally distributed among males and females, as well as amongst the elderly (>60 yrs) and the young, indicating that age or sex were not found to be independent risk factors for *Acinetobacter* colonization in patients. [Table 1].

Table 2 Cases of Probable VAP (P-VAP)

No	Month	Total Venti pts	Acinet colonizn	P-VAP	A +other coinfectn	Organisms isolated in PVAP				
						A	*K*	*P*	*C*	*As*
1	JAN	12	0	0	0	0	0	0	0	0
2	FEB	16	7	2	0	0	2	0	0	0
3	MAR	19	4	3	0	3	0	0	0	0
4	APR	23	2	2	1 (K+P)	1	2	1	0	0
5	MAY	18	8	3	3 (P,P,As)	3	0	2	0	1
6	JUNE	22	4	4	0	2	1	0	1	0
7	JULY	27	5	3	1 (P)	1	2	1	0	0
8	AUG	25	5	0	0	0	0	0	0	0
9	SEP	11	2	0	0	0	0	0	0	0
10	OCT	16	1	4	1	1	4	1	0	0
	TOTAL	**189**	**39**	**21**	**6**	**11**	**11**	**5**	**1**	**1**

(A= Acinetobacter sp., K= Klebsiella sp., P= Pseudomonas sp., C= Candida sp., As= Aspergillus fumigatus)

4. Thus, *Acinetobacter* sp. was isolated in 5.8% of cases of P-VAPs.
5. Relative Risk for development of P-VAP in mechanically ventilated patients colonized with *Acinetobacter sp.* is 4.23 times than those not harbouring *Acinetobacter sp.* in their respiratory tract [Table 2(A)].
6. Relative Risk for development of P-VAP in mechanically ventilated patients colonized with *Acinetobacter sp*. and co infected with some other bacteria or fungi is 2.7 times higher than those harbouring *Acinetobacter sp.* alone in their respiratory tract.[Table 2(B)]

Table 2 (A)

	P-VAP	No P-VAP	
***Acinetobacter* +ve**	11	28	39
***Acinetobacter* -ve**	10	140	150
	21	168	189

RR= 4.23

Table 2 (B)

	P-VAP	No P-VAP	
***Acinetobacter* + other coinfection**	6	6	12
***Acinetobacter* +ve only**	5	22	27
	11	28	39

RR= 2.7

7. Mechanical ventilation increases the relative risk of acquiring an *Acinetobacter* nosocomial pneumonia by more than 24 times.[Table 2(C)]

Table 2(C)

	ANP*	No ANP*	
Ventilated	11	10	21
Non ventilated	6	273	279
	17	283	300

RR= 24.36

(*ANP= *Acinetobacter* Nosocomial Pneumonia)

8. A total of 279 sputum and BAL fluid samples were collected from patients with clinical features of LRTI/ Pneumonia with or without radiological evidence, out of which only 8 patients tested culture positive for *Acinetobacter sp.* Out of these 8 isolates, 2 were community acquired strains while 6 were nosocomially acquired strains. Thus, *Acinetobacter sp.* contributed to only 2.15% cases of nosocomial pneumonias in non-mechanically ventilated patients.
9. Both the community acquired *Acinetobacter* isolates were sensitive to all classes of antibiotics. However, this was not the case with the hospital acquired strains. All the Hospital strains were multi drug resistant (i.e., resistant to atleast >1 classes of antibiotics).
10. The overall pattern of antibiotic resistance in the hospital strains of *Acinetobacter sp.* isolated from the respiratory specimens, was as below:

Table 3 Antibiotic Resistance Pattern in Hospital Strains of *Acinetobacter sp.*

Antibiotics	Total "S" (n=45)	% Sensitive Isolates
LEVOFLOXACIN	29	64.4
MOXIFLOXACIN	01	2.2
PIPERACILLIN-TAZOBACTAM	06	13.3
MEROPENEM	27	60.0
IMIPENEM	02	4.4
AMIKACIN	02	4.4
GENTAMICIN	02	4.4
MINOCYCLINE	11	24.4
COLISTIN	45	100

Thus, Sensitivity to antibiotics of Hospital Respiratory *Acinetobacter* strains was as follows: Colistin (100%)> Levofloxacin (64.4%)> Meropenem (60%)> Minocycline (24.4%)> PipTaz (13.3%). [Table 3]. However, for non mechanically ventilated patients, both, Levofloxacin and Meropenem showed 100% sensitivity, while Minocycline was sensitive in 50% of the cases.

11. 10 isolates of Carbapenem resistant *Acinetobacter* (CRA) isolates were detected, all in mechanically ventilated patients. Thus, CRA was found in 5.3% of the mechanically ventilated patients, and in none of the non mechanically ventilated patients.
12. Relative risk of developing an AP-VAP (*Acinetobacter* P-VAP) can be reduced by 2.35 times by implementing a few Infection Control measures (as specified in the methodology section).

Table 4 Risk Reduction post Infection Control Measures

	APVAP	Other PVAP	
Before HIC measures	10	07	17
After HIC measures	01	03	04
	11	10	21

RR= 2.35

(PVAP= Probable Ventilator Associated Pneumonia, APVAP= Acinetobacter P-VAP, HIC= Hospital Infection Control)

Conclusions

1) The respiratory tract of 20% of mechanically ventilated patients was colonised with *Acinetobacter sp*
2) Age or sex were not found to be independent risk factors for *Acinetobacter* colonization in patients.
3) *Acinetobacter* sp. was isolated in 5.8% of cases of P-VAPs.
4) Relative Risk for development of P-VAP in mechanically ventilated patients colonised with *Acinetobacter sp*. was 4.23 times than those not harbouring *Acinetobacter sp.* in their respiratory tract.
5) Relative Risk for development of P-VAP in mechanically ventilated patients colonized with *Acinetobacter sp*. and co infected with some other bacteria or fungi was 2.7 times

higher than those harbouring *Acinetobacter sp.* alone in their respiratory tract.

Thus while, Acinetobacter sp. alone increases the risk of getting a P-VAP, significantly (>4 times), co-infection with another pathogen further increases the risk by nearly 3 times.

6) *Acinetobacter sp.* contributed to only 2.15% cases of nosocomial pneumonias in non-mechanically ventilated patients, *indicating that Acinetobacter respiratory isolates in non-ventilated patients need not be treated with antibiotics, unless symptomatic.*
7) Mechanical ventilation increases the relative risk of acquiring an *Acinetobacter* nosocomial pneumonia by more than 24 times.

   This is probably due to the tendency of Acinetobacter sp. to form biofilms over prosthetic devices. Thus, further research needs to be done to develop materials that can resist formation of such films, which can be suitably used for making Endotracheal and Tracheostomy tubes.
8) Community acquired *Acinetobacter* isolates were sensitive to all classes of antibiotics. All the Hospital strains were multi drug resistant.

   This indicates that Acinetobacter sp. has a very high tendency of acquiring resistance to multiple classes of antibiotics very easily, when exposed to selective antibiotic pressure, as in hospital settings. Hence, the need to be extremely judicious before starting antibiotic therapy for Acinetobacter sp.
9) Sensitivity to antibiotics of Hospital Respiratory *Acinetobacter* strains was as follows: Colistin (100%)> Levofloxacin (64.4%)> Meropenem (60%)> Minocycline (24.4%)> PipTaz (13.3%). However, for non mechanically ventilated patients, both, Levofloxacin and Meropenem showed 100% sensitivity, while Minocycline was sensitive in 50% 0f the cases.

   Thus, Levofloxacin was found to be an effective alternative to Carbapenems, especially in those who have had previous carbapenem exposure.
10) Carbapenem Resistant *Acinetobacter* was found in 5.3% of the mechanically ventilated patients, and in none of the non mechanically ventilated patients. *This was probably due to the relatively higher exposures of carbapenems in critical patients.*
11) In the period prior to the introduction of basic HIC measures (i.e., Jan to Aug) Rate of *Acinetobacter* respiratory colonisation was nearly 22%, which was brought down to 11% post strict implementation of strict HIC measures (sept-oct).
12) Relative risk of developing an AP-VAP (*Acinetobacter* P-VAP) can be reduced by 2.35 times by implementing a few Infection Control measures, which include, marking of files of these patients with pink labels, patient and staff cohorting, hanging of Hand Hygiene and Std. Universal Precautions signages on the beds of these patients, strict adherence to contact and droplet precautions and hand hygiene while handling these patients, drawing of Curtains during oral suctioning and all aerosol generating procedures and twice daily cleaning of patient surroundings with 1% Hypochlorite followed by Peroxide based cleaning.

In this study, it was found that while nearly 20% of mechanically ventilated ICU patients were found to be colonized with *Acinetobacter sp.*, less than one-third of them (<6%) developed a P-VAP, while in non ventilated patients, only about 1 in 10 colonized patients developed a nosocomial pneumonia. It was also found that all hospital isolates of *Acinetobacter sp.* were multi drug resistant (some of those found in intubated patients were resistant even to carbapenems), while all the community acquired strains were sensitive to all classes of antibiotics. This indicates that, *using antibiotic coverage for all respiratory Acinetobacter sp. isolates, is probably*

counterproductive, since, it increases the selective antibiotic pressure, which enables, emergence of multi-drug resistance in Acinetobacter sp. Also, it was found that strict implementation of simple hospital infection control measures could reduce the colonization rates by half, and P-VAP rates by more than 2 times.

Thus, this study highlights that *judicious use of antibiotics, along with strict hospital infection control measures, is the need of the hour, in prevention of emergence of multi drug resistance in Acinetobacter sp.*

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