REFERENCE

1. <http://guidance.nice.org.uk/CG101/Guidance/pdf/English>.

P250 INCIDENCE, CLINICAL PROFILE AND OUTCOMES OF VENTILATOR-ASSOCIATED PNEUMONIA IN A TERTIARY HOSPITAL

doi:10.1136/thx.2010.151076.1

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Background Pneumonia is still the most important nosocomial infection among critically ill patients receiving mechanical ventilation despite advancement in the diagnostic technology available, introduction of new antimicrobials and employment of preventive strategies. Our aim was to prospectively identify the occurrence of ventilator-associated pneumonia (VAP) in a tertiary hospital as well as to determine its clinical profile and outcomes.

Methods A prospective, observational study was performed including 52 patients with VAP in a tertiary hospital. All adult patients admitted who required mechanical ventilation regardless of site of care was included and followed up until successful weaning, discharge or death.

Results During the 12-month study period (1 May 2009–30 April 2010), a total of 52 patients (7.6%) developed VAP among the 686 patients who received mechanical ventilation. Most common comorbid conditions documented were diabetes mellitus and hypertension. Higher APACHE II scores ($p \leq 0.001$), inappropriate antimicrobial use ($p = 0.027$) and the presence of underlying malignancy ($p = 0.03$) are correlated with mortality. The most common pathogen isolated was *Pseudomonas aeruginosa* (23%) and carbapenems as the most commonly used antimicrobial. Mortality rate was 38.5%. Non-survivors had numerically longer ICU stay and length of mechanical ventilation (Abstract P250 Table 1).

Abstract P250 Table 1

	Non-Survivors (n=20)	Survivors (n=32)	Total (n=52)	p-value
Age	68.5 ± 14.68	63.59 ± 17.26	65.48 ± 16.34	0.297 (NS)
Gender				
Male	9 (45%)	21 (65.63%)	30 (57.69%)	0.162 (NS)
Female	11 (55%)	11 (34.38%)	22 (42.31%)	
Ward Type				
Clinical division	5 (25%)	11 (34.38%)	16 (30.77%)	0.549 (NS)
Pay division	15 (75%)	21 (65.63%)	36 (69.23%)	
Co-morbidities				
Hypertension	6 (30%)	16 (50%)	22 (42%)	0.25 (NS)
Diabetes Mellitus	11 (55%)	14 (44%)	25 (48%)	0.57 (NS)
COPD*	2 (10%)	2 (6%)	4 (8%)	0.63 (NS)
Underlying Malignancy	7 (35%)	3 (9%)	10 (19%)	0.03
Renal Failure	1 (5%)	6 (19%)	7 (13%)	0.23 (NS)
CAD**	4 (20%)	2 (6%)	6 (12%)	0.19 (NS)
Reason for Intubation				
Medical	17 (85%)	21 (65.63%)	38 (73.08%)	0.200 (NS)
Surgical	3 (15%)	11 (34.38%)	14 (26.92%)	
VAP Onset				
Early	11 (55%)	22 (68.75%)	33 (63.46%)	0.382 (NS)
Late	9 (45%)	10 (31.25%)	19 (36.54%)	
Definite Etiology				
Without	5 (25%)	9 (28.13%)	14 (26.92%)	1.000 (NS)
With	15 (75%)	23 (71.88%)	38 (73.08%)	
With Monomicrobial	9 (60%)	13 (56.52%)	22 (57.89%)	
With Polymicrobial	6 (40%)	10 (43.48%)	16 (42.11%)	
Appropriateness of Empiric Antibiotics				
Yes	4 (20%)	18 (56.25%)	22 (42.31%)	0.027
No	9 (45%)	6 (18.75%)	15 (28.85%)	
Unknown	7 (35%)	8 (25%)	15 (28.85%)	
Number of Drugs				
Monotherapy	10 (50%)	18 (56.25%)	28 (53.85%)	0.777 (NS)
Combination	10 (50%)	14 (43.75%)	24 (46.15%)	
Concomitant Infection				
With	9 (45%)	20 (62.5%)	29 (55.77%)	0.260 (NS)
Without	11 (55%)	12 (37.5%)	23 (44.23%)	
Mean APACHE II score	27.25 ± 6.26	20.88 ± 5.59	23.33 ± 6.59	0.000

*tested combining High and Very High levels.
Continuous Variables are compared using Independent t-test
Categorical Variables are compared using Chi-Square Test/Fisher's Exact Test
*Chronic Obstructive Pulmonary Disease
**Coronary Artery Disease

Conclusion In our setting, current VAP incidence rate is 7.6% with mortality of 38.5%. This highlights the current magnitude of VAP which should prompt hospital administrators and health care workers to complement rational antibiotic use with non-pharmacologic strategies to at least decrease VAP incidence and alleviate its burden especially in a developing country.

P251 FLOW CYTOMETRIC DETECTION OF INTRACELLULAR ACTIVATION MARKERS OF PULMONARY CELLS DURING ACUTE LUNG INJURY

doi:10.1136/thx.2010.151076.2

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Background Activation of intracellular signalling cascades such as mitogen-activated protein (MAP) kinase pathways has been implicated in various animal models of acute lung injury. However, such data rely almost exclusively on measurements within whole lung homogenate samples, so little information is available regarding cell type specific processes. In order to address this, we have developed a flow cytometric technique to identify discrete pulmonary cell populations and examine their early activation in terms of phosphorylation of intracellular MAPkinase pathway intermediates (ERK, p38 and its downstream target MK2).

Abstract P251 Figure 1 Level of intracellular phosphorylated MK2. A. Alveolar macrophages. B. Epithelial cells.