Hospital acquired pneumonia is most frequently associated with bacterial infections and accounts for 15% of all hospital-associated infections, it has 6-21 times higher risk in patients receiving continuous mechanical ventilation (Tabian et al., 2004). This continuous ventilation caused pneumonia is also known as Ventilator-Associated Pneumonia (VAP) and results in 60% of all hospital-associated infection deaths. It usually results from bacterial infections and Woske et al. (2001) found Staphylococcus aureus followed by Pseudomonas aeruginosa and Haemophilus influenzae as its major causal agents. Furthermore, they also proposed that its diagnosis is difficult but bronchoscopic tracheal secretion analysis may be helpful up to some extent. The correct diagnosis of VAP is necessary, because delayed application of antibiotics can lead to disease severity and high mortality rates (Iregui et al., 2002). But many techniques like bronchoalveolar lavage and examination of serum procalcitonin levels failed in its reliable diagnosis (Luyt et al., 2008). Bronchoalveolar lavage cultures give false information in almost 50% VAP patients, while estimation of procalcitonin levels has only 24% specificity to VAP diagnosis. Moreover, procalcitonin estimation if compared before and after VAP has only 41% specificity. Thus VAP diagnosis is difficult procedure, but it is not the only reason of VAP patient’s suffering, the incorrect application of initial antibiotics also play key role in increased disease complication and mortality (Dupont et al., 2001). According to Kollef (2000) inadequate exposures to antibiotics and use of broad-spectrum antibiotics lower the efficiency of treatment. This can be treated through the correct combination of antibiotic according to patient’s physiological characteristics and through accurate identification of predominant bacteria. But despite of correct diagnosis, restriction to patient’s recovery arises due to increased antibiotic resistance in bacteria (Mashouf et al., 2005). For example S. aureus has developed more than 80% resistance against ampicillin, amoxicillin and chloramphenicol; it also developed resistance against many other antibiotics. Likewise P. aeruginosa has developed 100% resistance against cephalaxin, ampicillin, amoxicillin and sulfamethoxazole-trimethoprim. Thus to treat VAP identification of responsible bacteria and effective antibacterial antibiotic is necessary, moreover its correct and rapid diagnosis is also necessary. For which, new technique other than bronchoalveolar analysis is essentially required.

Recently Gupta et al. (2011) performed a study on 107 ventilated patients to evaluate the frequency of bacterial infection, their antibiotic sensitivity, duration of mechanical ventilation and VAP risks. Moreover, they also studied the efficiencies of APACHE II ("Acute Physiology and Chronic Health Evaluation II") system in diagnosis of VAP. From these 107 studied patients 30 developed the VAP symptoms thus its incidence was 28.04%, which caused high mortality 53.4% rate. This VAP infection rate was not significantly depend on disease type (snake bites, nervous system problems, sepsis etc.), age and gender but on ventilation duration and hospital stay. However, the death rate due to VAP was dependent on the disease type and was significantly higher in patients who showed late onset of VAP. These late-onset VAP patients were mostly infected with P. aeruginosa and S. aureus, as all 9 P. aeruginosa strains and 7 out of 8 S. aureus strains were isolated from late-onset VAP patients. Thus, these two bacteria were majorly responsible for VAP in this study; in addition P. aeruginosa some strains were resistant to 11 studied antibiotics, while all 9 strains were sensitive to only two antibiotics; polymyxin B and colistin. The other leading antibacterial resistant bacterium was Klebsiella pneumoniae, as its some strains were resistant to 12 antibiotics and its all strains were sensitive to only polymyxin B and colistin. Both these drugs were also effective against all strains of another most common bacterium, Acinetobacter baumannii and its strains were resistant to 11 antibiotics. A. baumannii and K. pneumoniae strains were also found in high ratio in late-onset VAP group and were the major reason of VAP patient’s mortality, as 5 out of 6 A. baumannii and 5 out of 7 K. pneumoniae strains were present in non-surviving VAP patients. Thus, high incidence of VAP was probably due to increased antibiotic resistance in its causal agents. Moreover, it was also found that APACHE II could efficiently diagnosed VAP due to its significantly different values in VAP and non-VAP groups. As in VAP patients it showed high scores (22.47±8.382), while in non-VAP patients scores were significantly low viz. 14.74±7.491. In addition, its other components; acute physiology score
and Glasgow coma score were also significantly different in both groups. Thus APACHE II could accurately classify the patients as VAP and non-VAP groups along with precise estimates of its mortality rates. As in non-surviving patients its mortality score was nearly two times higher than surviving patients. Hence, its high scores indicated low survival rate and huge disease severity outcomes. Through above discussion, this can be said that VAP incidence and resultant mortality was determined by the bacterial strains, which got resistance against number of antibiotics. However, APACHE II scoring and use of effective antibiotics could reduce this incidence.

VAP is one of the major problems faced by hospitalized patients, whose immediate diagnosis and rapid therapy is extremely required. Gupta et al. (2011) through their research on APACHE II and VAP patients provided a reliable diagnostic system. As they found APACHE II scoring as competent differentiation between VAP and non-VAP patients, whose high value indicated the VAP severity. Besides this they also specified some biological and physical reasons of VAP in hospitalized patients, this would help in reducing VAP incidence and patients suffering. Thus, least use of mechanical ventilation, application of APACHE II scoring and strong antibiotics is the only way to reduced VAP risks.

REFERENCES


