

Research Article

Mucoid Variant of *Pseudomonas aeruginosa* in Previously Healthy Individuals With-out Cystic Fibrosis

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Pseudomonas aeruginosa is a well-known nosocomial pathogen that causes various infections in hospitalized patients, especially those with underlying immuno compromised state, but community acquired pneumonia (CAP) due to *Pseudomonas aeruginosa* in previously healthy adults is rare and very few cases have been documented in world literature. The review of literature of these rare cases depict a fatal outcome of these patients¹.

For the last six months, we have been constantly observing the growth of *Pseudomonas aeruginosa* from sputum samples of previously healthy individuals presenting to the OPD for the first time and suspected to have pneumonia or COPD. To analyze the clinical relevance of these findings, we conducted this prospective observational preliminary study during Jan2015-June 2015. A total of 1800 patients were enrolled in the study. Demographic details of the patients as well as history of any previous lung diseases were noted. Sputum samples of all these patients were received and the quality of sputum was assessed by

Bartlett grading. All the samples were processed as per standard guidelines. The identification and antimicrobial susceptibility of these isolates was done using automated B.D. Phoenix system.

Out of 1800 samples, pure growth of *Pseudomonas aeruginosa* was observed in 69 samples. The striking feature was that out of these 69 isolates, 10 were mucoid variants of *Pseudomonas aeruginosa*. Demographic profile of these 10 patients revealed mean age of 59 years with male to female ratio 1:4 in contrast to the non mucoid isolates where mean age was 55 years and male to female ratio was 1:1.3. About 60% of the patients, harboring mucoid strains had history of smoking and none of these patients had been diagnosed earlier for cystic fibrosis (CF) or any other underlying lung disease. Comparing the minimum inhibitory concentrations of mucoid vs non mucoid strains, the mucoid variants were found to be more sensitive to all routinely prescribed antibiotics. Table -1 depicts the comparative sensitivity pattern for mucoid vs non-mucoid variants.

Table 1: Comparative sensitivity pattern for mucoid vs non-mucoid variants of *Pseudomonas aeruginosa*

<i>Pseudomonas aeruginosa</i>	Ciproflox	Amikacin	Ceftazidime	Netilmicin	Meropenem	Imipenem	Aztreonam	Piperacillin-Tazobactam
Mucoid variant	90%	90%	100%	80%	100%	100%	100%	100%
Non-mucoid variant	87%	88%	88%	84%	97%	96%	64%	91%

Amongst the mucoid variants, there was no resistance to Imipenem, Meropenem, Piperacillin-Tazobactam, Aztreonam and Ceftazidime. Resistance to Amikacin, Ofloxacin and Netilmicin was 10%, 10% and 20% respectively. To the best of our knowledge, mucoid variants of *Pseudomonas* have not been reported earlier from previously healthy individuals. As per the previous reports

mucoid variants are commonly associated with CF disease, but CF is a less common in India as compared to COPD and bronchiectasis which are the other two conditions where mucoid variants are commonly observed. In our study none of the patients was found to be having CF, but about 60% of the patients had history of smoking which is a known predisposing factor for COPD and

bronchiectasis. Altered mucociliary clearance in COPD and bronchiectasis leadsto mucous stasis which facilitates the colonization of *P.aeruginosa* in lungs. Various research studies have shown that these colonizer can mutate to mucoid variants, which if not diagnosed and treated earlier, can lead to the formation of biofilm which is difficult to eradicate and may result in therapeutic failure².

The significance of this study is that the isolation of mucoid variants of *Pseudomonas aeruginosa* that seem to be mere colonization at the first visit may either be the indirect evidence of COPD or later on it can be responsible for the acute exacerbation of COPD or CAP. Studies have shown that *Pseudomonas aeruginosa* CAP occurring in previously healthy persons may cause bacteremia which can adversely affect the patients irrespective of use of appropriate anti-pseudomonal antibiotics³. In our study, the patients could not be followed further because they were OPD patients and no repeat samples were received for any of these patients, implying that none of them had disease severe enough to require hospitalization and further supporting our hypothesis that this could be mere colonization. The data presented in the article are preliminary and further studies are required to ascertain the role of mucoid

variants in disease causation as well as prognosis in cases of COPD and CAP.

We conclude that mucoid variants of *Pseudomonas aeruginosa* isolated from previously healthy persons should be dealt with a coordinated teamwork by clinicians and microbiologists so that proper protocols can be set up to manage *Pseudomonas aeruginosa* colonization in the very initial stage when they are still sensitive to common anti-pseudomonal drugs.

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