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Bacterial Etiology: Significant In Allergic Respiratory Diseases

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ABSTRACT

Amongst allergic respiratory diseases allergic rhinitis & asthma are the most common. Asthma is a chronic inflammatory disease of the airways which in susceptible individuals causes recurrent episodes of wheezing, breathlessness, chest tightness & cough. Inflammation can cause increased airway hyperresponsiveness which can lead to infections by bacteria including atypical bacteria, fungi & viruses. Microbial infections associated with allergic respiratory infections increase severity & duration of the disease as well as they themselves act as an allergen. Therefore their treatment with appropriate antimicrobials is essential. Total 300 patients suffering from Asthma which is most common allergic respiratory disease were investigated for detection of bacterial pathogens in their sputum samples. Bacterial pathogens were isolated in 20% cases, which were *M. catarrhalis*, *S. aureus*, *S. pyogenes*, *K. pneumoniae*, & *P. aeruginosa*. *M. catarrhalis* was the most common isolate found in 19% cases followed by *S. aureus* in 28.33 % cases, *K.pneumoniae* & *P. aeruginosa* in 16.66 % cases each & *S. pyogenes* in 6.6 % cases. Antibiotic sensitivity testing was performed to determine their sensitivity pattern & it was observed that *M. catarrhalis* & *S. pyogenes* were sensitive to all the antibiotics tested. Other isolates showed variable susceptibility pattern. These isolates showed susceptibility to Aminoglycosides & cephalosporin group. Various bacteria play a major role in severity of exacerbations, symptoms & overall progress of allergic respiratory disease such as asthma. These bacterial infections can be diagnosed & treated with appropriate & specific antibiotics. This antibiotic treatment will help in fast recovery of patients with reduced duration of corticosteroid consumption of patients.

Keywords

Asthma,
Allergen,
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Introduction

The respiratory system is the most common site for infection by pathogenic microorganisms. Though these infections are common and usually mild, they are frequently taken for granted; they represent an immense disease

burden on our society. The respiratory tract becomes infected frequently as it is in direct contact with the environment and is continuously exposed to microorganisms suspended in the air we breathe. Some of these microorganisms are highly virulent and only a

few of them are needed to infect even healthy person. However, most of these microorganisms do not cause infection unless other factors interfere with the defenses. The warm, moist environment of the respiratory tract is an ideal place for growth of microorganisms' (Schaechter *et al.*, 1989).

Amongst allergic respiratory diseases allergic rhinitis (hay fever) and asthma are two of the most common allergic diseases. Both diseases appear to have increased significantly in past two centuries (Seema *et al.*, 1997).

Asthma is a chronic inflammatory disease of the airways which in susceptible individuals causes recurrent episodes of wheezing, breathlessness, chest tightness and cough. Inflammation can increase airway hyperresponsiveness which can lead to infections by bacteria including atypical bacteria, fungi and viruses (Brinke *et al.*, 2005).

Recurrent exacerbations are a major cause of morbidity and medical expenditure in patients with asthma. Factors significantly associated with frequent exacerbations include severe nasal sinus disease, gastrointestinal reflux, recurrent respiratory infections, physiological dysfunctioning and obstructive sleep apnoea (David Issacs *et al.*, 2002).

In asthma condition allergic, toxic, fungal, viral and other initiators of inflammation play a major role. There are varieties of triggering factors identified for asthma. Amongst these respiratory tract infections mainly by fungi, viruses and bacteria including atypical bacteria like *M. pneumoniae* and *C. pneumoniae* are important.

Bacterial lipopolysaccharide plays a major role in asthma. Inhaled lipopolysaccharide can exacerbate airway inflammation and airway obstruction in allergic asthmatics. As

bacterial infections play a major role in etiopathogenesis of allergic respiratory infections their consequent treatment is required by means of wide spectrum antibiotics as well as prescription of bacterial immunotherapy (Nagayama *et al.*, 1999).

Role of Bacteria in Allergic Respiratory Infections

In several publications it has been insisted that bacterial infections are important in the etiopathogenesis of bronchial asthma and other allergic respiratory infections. Bacterial antigens potentiate the action of inhalant allergens. It has been already proven that the action of these bacterial antigens increases the number of intraepithelial dendritic cells in the bronchial mucosa after inhalation of bacterial lipopolysaccharide. The structural elements of bacteria and toxins produced by them intensify the release of mediators (Leucotrienes, histamine, IL1, IL 4, IL 6, IL 8 and TNF – alpha) of the inflammatory reactions.

Bacteria commonly found to be associated with respiratory allergies include *Moraxella catarrhalis*, *Streptococcus pneumoniae*, *H. influenzae* etc. (Alic Verghese, 2001). Amongst atypical bacteria *M. pneumoniae* and *C. pneumoniae* are associated with bronchial asthma as well as COPD. It is found that bacterial flora of the upper respiratory tract in patients with bronchial asthma is more diverse in comparison with microflora of airways in healthy subjects.

Bacterial Components that Contribute to Allergic reaction

Of all microbial products, endotoxin has been studied extensively. Along with endotoxins a variety of other microbial

agents are known to have immune stimulatory properties which includes beta (1, 3) – glucans, bacterial DNA and other bacterial components. Beta (1, 3) glucans are glucose polymers present in the cell wall of most fungi and yeasts, some bacteria and vegetable materials.

Beta (1, 3) glucans have been measured as a marker of mould exposure in field studies. Moulds often grow together with different bacteria and hence levels of beta (1, 3) glucan in house dust have been found to be highly correlated with endotoxin levels. Thus endotoxin could be a marker for broader range of microbial exposure.

Bacterial Lipopolysaccharide

Inhaled LPS can exacerbate airway inflammation and airflow obstruction in allergic asthmatics. Allergic subjects are more sensitive than nonallergic subjects to the bronchoconstrictive properties of inhaled LPS. In addition prior allergen exposure significantly augments the inflammatory response to inhaled LPS.

In healthy nonasthmatic subjects, approximately 10%-15% of subjects are highly sensitive to developing airflow obstruction after inhaling small amounts of LPS, whereas a similar percentage of normal nonasthmatic subjects are highly resistant to developing airflow obstruction after inhalation of high doses of LPS.

Bacterial DNA and Immunostimulatory DNA CpG sequences

Bacterial DNA is highly enriched in immunostimulatory DNA containing CpG motifs that bias the immune response to antigens to a nonallergic Th1 phenotype. This property of bacterial DNA provides a potential mechanism for gastrointestinal bacterial DNA to modulate the developing

immune response in infancy. CpG motifs are a 6-base pair sequence of noncoding DNA that is highly enriched in bacterial DNA.

In mouse models of asthma immunostimulatory DNA sequences inhibit eosinophilic inflammation, Th2 cytokines including IL-5 and airway hyperreactivity to methcholine. The immunostimulatory DNA sequences are effective when administered systemically or mucosally (Henry, 1992).

Mycobacterial Infection and Allergy

In a study of Japanese schoolchildren, there was a strong inverse association between delayed hypersensitivity to *Mycobacterium tuberculosis* and atopy.

Positive tuberculin test responses in these children predicted a lower incidence of asthma, lower serum IgE levels, and cytokine profiles biased towards a Th1 response. Based on this study, it was postulated that exposure to *M. tuberculosis* may, by modification of immune profiles, inhibit atopic disorder. In mouse models BCG and *M. vaccae* inhibit allergic responses. The potential therapeutic effects of *M. vaccine* in human allergic inflammation are currently being investigated.

Long term use of steroids can give a lot of side effects including suppression of immune system making individual more prone to fungal infections similarly prolonged and irrational use of antibiotic may result in emergence of drug resistance organisms. Unnecessary and random use of medicines without prior diagnosis and also increases total cost of therapy.

Benefit of Antibiotics in Asthma Treatment

Some macrolides are known to exert immunomodulatory properties that are

independent of their antibacterial activity. These agents modulate the functions of inflammatory cells, including polymorphonuclear leucocytes, lymphocytes and macrophages. Macrolides influence several pathways involved in the inflammatory process, including the migration of neutrophils, the oxidative burst in phagocytes and the production of proinflammatory mediators and cytokines and several of these agents have shown anti-inflammatory effects. Macrolides inhibit the synthesis and /or secretion of proinflammatory cytokines (e.g. TNF- α , IL-8, IL-6, IL-1 β), whereas their effects on anti-inflammatory cytokines (IL-10, IL-4) are more variable.

Objective of the present study was to detect presence of bacterial pathogens in respiratory tract of patients with allergic respiratory conditions such as Asthma & to check antibiotics effective against these pathogens.

Materials and Methods

A total of 300 patients were included in the study.

These patients were enrolled for treatment of allergic respiratory disease such as Asthma & were visiting Medicine Department of T. N. Medical College & B. Y. L. Nair charitable Hospital, Mumbai Central, Mumbai, Maharashtra, India.

These patients were located in different areas in Mumbai as well as outside Mumbai. The senior clinician of Medicine department, T. N. Medical College did the selection of subjects on the basis of their clinical and radiological findings.

Patients selected were either hospitalized (Indoor basis) or outdoor patients at B. Y. L. Nair charitable Hospital.

Inclusion Criteria

1. Patients able to give productive sputum & clinically suspicious of infective etiology.
2. Patients who are able to produce brief clinical history.
3. Patients in whom antibiotics have not been administered within last 48-72 hours.

Following Exclusion Criteria were used while Selecting Patients for the Study

1. Patients who are not able to produce adequate sputum or give any other relevant respiratory samples.
2. Patients on prolonged antibiotic treatment.
3. Patients requiring ICU care.
4. Pregnant females.

Collection of Respiratory Specimens

Sputum samples were collected & processed. Minimum 3 consecutive samples were studied for confirmation of results. All universal safety precautions were taken while collection, transportation, handling and processing of specimens. All specimens were processed within one hour of collection. Fresh morning sputum specimens were collected with aseptic in a clean, sterile, leak-proof container. Each patient was advised to collect an early morning sputum sample after washing the mouth and gargling with tap water.

All sputum samples were graded by Murray & Washington grading system & unsatisfactory samples were discarded. Fresh samples were procured.

Processing of Respiratory Specimens

All specimens were studied microscopically as well as macroscopically. In macroscopic

study characters like color, appearance, presence or absence of blood in specimens were noted.

Culture – A loopful of each specimen (Representative portion) was inoculated or streaked on various culture media for isolation of pathogens. All aseptic precautions were taken while processing of samples.

Following media were used

1. Blood agar
2. Chocolate agar
3. MacConkeys agar

Study of following organisms was excluded

1. Anaerobic organisms
2. Mycobacterium spp.
3. Protozoa

Blood agar and MacConkey agar were incubated aerobically at 37+/- 0.5°C and growth was observed after 24 hours. If after 24 hours no growth was observed then the plates were incubated further for 24 hours before concluding as No growth. In case growth is observed colony morphology which includes size, shape, consistency, opacity and elevation was recorded.

Blood agar was checked for presence or absence of hemolysis and if present it's type was noted. MacConkeys agar was used to differentiate between lactose fermenters and lactose non-fermenters. Chocolate agar was incubated at 37C +/- 0.5°C in 5-10% CO₂ for 48-72 hours. After incubation colony characteristics were noted.

Identification of the isolates obtained **Identification of Gram negative bacilli –** **(Lactose fermenting colonies)**

Identification of *Klebsiella pneumoniae*

Lactose fermenting (pink) mucoid colonies

were obtained on MacConkeys agar which were further identified by performing following biochemical tests

Indole test, Methyl Red test, VP test, Citrate test (IMViC), Motility, Lysine decarboxylase, production, Fermentation of Lactose, Sucrose, Sorbitol, Arabinose, Urease production

Identification of Gram negative bacilli – **(Lactose non-fermenting colonies)**

Identification of *Pseudomonas aeruginosa*

Lactose nonfermenting (yellow)colonies were obtained on MacConkeys agar which were further identified by performing following biochemical tests

Oxidase test, Motility, Pigment production, Arginine decarboxylase production, citrate test, Nitrate reduction, Fermentation of Glucose.

Production of bright bluish green diffusible pigment was determined using nutrient agar. *P. aeruginosa* was distinguished from others by it's ability to grow at 4°C.

Identification of Gram positive cocci

Identification of *Streptococcus pyogenes*

Small pinpoint beta hemolytic, catalase negative colonies were obtained on Blood agar plate. They were further identified by performing following biochemical tests Bacitracin sensitivity test, Hydrolysis of arginine, PYR test, Growth inhibition on Bile esculin agar, Trimethoprim-sulfamethoxazole resistance.

Identification of *Staphylococcus aureus*

Smooth, butyrous, entire edge and about 1-2 mm in diameter, catalase positive, beta

hemolytic colonies were obtained on Blood agar plate. They were further identified by performing following biochemical tests

Coagulase test, Voges-Proskauer test, Nitrate reduction test, Urease test, Anaerobic fermentation of glucose & mannitol.

Identification of *Moraxella catarrhalis*

Non-motile, nonfermentative and oxidase positive. Colonies on chocolate agar gray to white, smooth, opaque. They were further identified by performing following biochemical tests: Catalase, Gelatin hydrolysis, Nitrate reduction test, Penicillin sensitivity test, Ability to grow on chocolate agar at 22⁰C, Ability to grow on nutrient agar at 35⁰C.

Antibiotic Susceptibility testing

Antibiotic susceptibility testing of the organisms of study and control group obtained from clinical specimens was carried out by Kirby-Bauer disk diffusion method according to CLSI guidelines.

A few colonies of the isolate to be tested were inoculated in 5 ml of suitable broth and incubated for 3-4 hours at 37°C. The turbidity of the broth was matched visually with 0.5 McFarland's standard using sterile saline. A sterile non-toxic cotton swab on a wooden applicator was dipped in to the standardized inoculum. This soaked swab was rotated firmly against the upper inside wall of tube to remove excess fluid. The surface of Mueller Hinton agar plate was uniformly streaked with swab three times by turning the plate at 60° angle between each streaking.

The plates were allowed to dry at room temperature for 5 mins. Each antibiotic disc was placed aseptically using a pair of sterile

forceps on the surface of the culture at an optimum distance on the inoculated plates. The plates were incubated at 37°C aerobically.

Plates were incubated at 37°C in presence of 5-10% CO₂ for *Streptococcus* species and *Moraxella catarrhalis*. Blood agar plates were used to carry out Antibiotic sensitivity pattern for these isolates. For *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Staphylococcus aureus* Mueller Hinton agar plates were used.

The Antibiotic Discs Employed in the Method are as follows

For *Klebsiella pneumoniae*

Amikacin (30 mcg), Amox+clav. (30 mcg), Cefotaxime (30 mcg), Ciprofloxacin (5 mcg), Gentamicin (10 mcg), Piperacillin (100 mcg), Imipenem (30 mcg)

For *Pseudomonas aeruginosa*

Ceftazidime (30 mcg), Cefoperazone (30 mcg), Gentamicin (10 mcg), Amikacin (30 mcg), Ciprofloxacin (5 mcg), Piperacillin (100 mcg), Aztreonam (30mcg), Imipenem (30 mcg)

For *Moraxella catarrhalis*

Ampicillin (10 mcg), Cefotaxime (30 mcg), Cefixime (30mcg), Azithromycin (15 mcg), Imipenem (30mcg), Aztreonam (30 mcg), Tetracycline (30mcg), Cefotaxime (30mcg)

For *Streptococcus pyogenes*

Erythromycin (15 mcg), Vancomycin (30mcg), Ampicillin (10mcg), Clindamycin (30 mcg), Ofloxacin (5mcg) cefotaxime (30 mcg) ceftazidime (30 mcg)

For *Staphylococcus aureus*

Oxacillin(30 mcg), Vancomycin (30 mcg), Clindamycin (30 mcg), Erythromycin (15 mcg), Gentamicin (10 mcg), Ciprofloxacin (5mcg), Tetracycline (30 mcg), Amox+clav. (30 mcg),

After overnight incubation at 37°C, diameter of each zone were measured and recorded in mm. The results were interpreted according to CLSI guidelines, by comparing with the results stated with standard ATCC strains. The pattern obtained was documented as Sensitive, Resistant or Intermediate.

All culture media as well as biochemicals used were procured from HiMedia Laboratories, Mumbai, Maharashtra, India

All media, reagents & Antibiotic discs were procured from HiMedia Laboratories, Mumbai, Maharashtra, India

Quality control of culture media, Biochemicals & antibiotic discs was checked by standard methods prescribed.

To confirm their allergic status total serum IgE estimation was also done.

Serological Tests

3-5 cc of whole blood was collected by venipuncture using a disposable 5.0 ml syringe & 21 gauge hypodermic needle in a sterile plain test tube taking all aseptic precautions. The sterile plain tube was incubated at 37°C in a slanting position for 1-1.5 hours & later held at 4°C for 1 hour. This method facilitates clotting of blood. (This was a routine practice followed at T. N. Medical College & B. Y. L. Nair charitable Hospital, Mumbai Central, Mumbai, Maharashtra, India where the current study was carried out).

The supernatant serum layer was separated & centrifuged at 2000 rpm to remove cell debris.(centrifuge REMI, Temperature 28+/- 2° C) The clear serum sample was preserved in absence of preservatives in plastic storage vials after labeling properly with patient's registration number & date at -20°C until utilization. (Maximum capacity of storage vials was 5.0ml & were obtained from Himedia Laboratories, Pvt. Ltd., Ghatkopar, Mumbai, Maharashtra, India)

The stored serum was used for detecting Total IgE by solid phase ELISA (Details are mentioned below)

*Total IgE estimation was done by solid phase Enzyme Linked Immunoassay by IBL ELISA. (Quantitative)

This is IBL manufactured kit. Kit contained 6 standard samples which had IgE concentrations from 0 IU/ml to 1000 IU/ml.

On a semi-logarithmic graph paper the concentrations of the standards (abscissa logarithmic) were plotted against their corresponding optical density (ordinate linear) The concentration of the samples can be read directly from this standard curve by using their average optical density. Any sample reading greater than the highest standard should be diluted appropriately. The result has to be multiplied with corresponding dilution factor.

As all the above ELISA used were quantitative titers were determined & then interpretation was done.

Results and Discussion

In recent decades it is seen that bacterial infection factor has been overlooked in the causal treatment of bronchial asthma and allergies. But literature evidence suggests that bacterial infections play a major role in

the etiopathogenesis of allergic diseases. Therefore along with treatment for allergic manifestations it is necessary to give treatment for these bacterial infections with wide spectrum antibiotics. This will result in cure in asthmatic patients without maintaining them on inhalers and unnecessary corticosteroid therapy.

In our study 20% allergic cases showed presence of bacterial etiology in their respiratory tract, as determined by culture of their sputum sample. *M. catarrhalis* was found to be most prominent isolate obtained in 31.66% cases, followed by *S. aureus* obtained in 28.33% cases. Other isolates obtained included *K. pneumoniae* & *P. aeruginosa* both in 16.66% cases each. *S. pyogenes* was obtained in 6.66% cases. (Table 1) Similar findings are reported by Nagayama and Tsubaki T *et al.*

Nagayama and Tsubaki *et al* cultured sputum specimens quantitatively from asthmatic children aged 0-14 years in order to determine the relationship between asthmatic status and bacterial species present in the respiratory tract.

In children with acute asthma attack, *H. influenzae*, *Streptococcus pneumoniae*, *M. catarrhalis* were distributed evenly in the samples. In patients with prolonged asthma attack pathogenic bacteria were present in 34.7% of patients. In these patients *H. influenzae* was predominant. In patients with pneumonia without asthma attack 40.9% of pathogenic bacteria were obtained. This study showed that there is a significant relationship between the presence of bacteria in sputum and clinical symptoms such as fever and pneumonia episodes during acute asthma attacks.

Antibiotic sensitivity testing was performed by standard Kirby –Bauer disc diffusion method as prescribed by CLSI. All *S.*

pyogenes isolates (total 4) & All *M. catarrhalis* isolates (total 19) were sensitive to all the antibiotics tested

All *S. aureus* isolates (Total 17) were sensitive to oxacillin, augmentin, Clindamycin, gentamicin, ciprofloxacin, Tetracycline & vancomycin. 70% *S. aureus* isolates were sensitive to Erythromycin. Erythromycin resistance was observed in 30% isolates. As reported by Shrestha and Singh *et al* an in vitro study has documented cloxacillin as the most effective antibiotic for *S. aureus* & ciprofloxacin & erythromycin as the least effective antibiotics.

Levels of erythromycin resistance have reached 20-40% in some parts of the world. Steady increase of MIC from 11.5 mcg/ml in 2.7% isolates in 1980 to >100 mcg/ml in 5.1% isolates in 1985 have been reported in India.

At present around 80-90% of strains of *M. catarrhalis* are shown to acquire resistance to ampicillin in recent years. 24% strains from Southern India were found to have reduced susceptibility to co-trimoxazole.

In present study all *M. catarrhalis* isolates (Total 19) obtained were sensitive to all antibiotics tested.

All isolates of *K. pneumoniae* obtained (Total 10) in the present study were sensitive to cefotaxime, ceftazidime, Amikacin, Ciprofloxacin & Imipenem. Augmentin resistance was observed in 50% isolates, Gentamicin & Piperacillin resistance was observed in 20% isolates for each antibiotic.

In case of *P. aeruginosa* isolates obtained (Total 10) were sensitive to cefotaxime, ceftazidime, Amikacin, Aztreonam

Imipenem & piperacillin. Gentamicin & ciprofloxacin resistance was observed in 20% isolates for each antibiotic.

Shrestha & Singh *et al* reported amikacin as the most effective drug for Gram negative bacteria & cephalixin as the least effective drug.(Shrestha *et al.*, 2005)

Patients gave history of single or multiple criteria like previous history of allergic disease, known allergy, history of pulmonary disease, history of addiction, sensitivity to dust, fumes and cold products.

Patients enrolled in the study often complained about chest pain, fever, breathlessness and exacerbations and sputum purulence. One or more of the above symptoms might be due to bacterial infections. Total immunoglobulin levels were also found to be elevated above normal levels in few cases. There is also a possibility that these bacteria may themselves act as allergens and aggravate the already existing allergic conditions. Based on the diagnosis of allergic manifestation corticosteroid, inhaler therapy is subscribed taking into consideration the allergic condition. But at the same time presence of bacterial agents in respiratory tract of allergic patients should be also considered and antibiotics specific for that particular pathogen. The effective combination of steroid therapy and focused antibiotic therapy will result in complete cure of allergic condition.

In the present study total serum IgE levels were estimated by ELISA as total IgE levels provide the evidence in support of atopy. All patients showed IgE levels above 1000IU/ml, which correlated with

respiratory symptoms, history & atopy.

Chowdhary and Vinaykumar *et al* reported elevated IgE levels in 90% allergic rhinitis cases. In their study of allergic rhinitis associated with bronchial asthma cases, IgE values were more than 1000 IU/ml. They also proved that 90% patients with allergic rhinitis with peripheral eosinophil counts in normal ranges. When rhinitis was associated with bronchial asthma, the eosinophil values showed an increase above the normal.

In conclusion, respiratory infections are important causes of diseases and disability worldwide. They are the most common cause of death in underdeveloped countries, especially in children. Allergic conditions affect 20% of the world's population, amongst which respiratory allergies are important ones.

Study of respiratory infections in allergic conditions generally includes study of the risk factors, associated symptoms and clinical history. Microbial etiology is often overlooked.

Previous studies has proved that microorganisms appear to be involved in some of the cases the etiology of some of the cases of allergic manifestations. Hence, there is a need to study the role of microorganisms in allergic conditions, which will help in identifying the exact cause of the exacerbations and symptoms of allergic conditions. Focused treatment considering these infectious agents is important in minimizing the severity of allergic conditions. Infectious etiology can itself act as an allergen aggravating the allergic as well as COPD conditions.

Table.1 Showing number of bacterial isolates in sputum specimens collected from Asthma patients

Total number of samples processed	Total number of samples showing positive bacterial culture findings	Bacterial isolates				
		<i>M. catarrhalis</i>	<i>S. pyogenes</i>	<i>S. aureus</i>	<i>K. pneumoniae</i>	<i>P. aeruginosa</i>
300	60 (20%)	19 (31.66%)	04 (6.66%)	17 (28.33%)	10 (16.66%)	10 (16.66%)

Along with steroid therapy antibiotic targeting the infectious agents can be given which will result in minimizing the cost of treatment and side effects associated with irrational and random use of antibiotics and steroids.

Hence there is an urgent need to focus on the microbial etiology associated with infections in allergic conditions.

The main objective of this prospective longitudinal study was to determine the etiology and clinical history of patients suffering from infections in allergic conditions

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References

Alic Verghese. 2001. Immune Profile in Respiratory Viral Infections in Asthmatics. *Indian J. Allergy Immunol.*, 15(2): 87-92 .
 Bauer, A.W., Kirby, W.M.M., Sheries, J.C., Turuck, M. 1966. Antibiotic susceptibility testing by standard single disk method. *American J.*

Pathol., 5: 433-496.
 CLSI. 2014. guidelines Performance Standards for Antimicrobial Susceptibility Testing 24th informational supplement M00 s24.
 David Greenwood, Richard, C. B. 1992. Black Medical Microbiology 14th edition, ELBS Churchill Livingstone.
 David Issacs, Preeti Joshi. 2002. Respiratory infections and Asthma *MJA*, 177(6 suppl): S50-S51.
 David, H. Broide. The Role of Bacterial infections in Allergy : A clinical Paradox Review from 57th Annual Meeting of the American Academy of Allergy, Asthma and Immunology, March 16-21, 2001, New Orleans, Louisiana.
 Ekiel, A., Friedek, D., Szulakowski, Romanik, M. 2005. Incidence of selected bacterial pathogens of the respiratory tract in patients with bronchial asthma *Wiad Lek*, 55(1-2): 11-8.
 Elmer, W., Konemann Stephen, D., Allen, William, M., Janda. 1992. In Colour Atlas and Textbook of diagnostic Microbiology, 14th edition, J. B. Lippincott Company
 Frankel, S., S. Reitman, S, Sonnenwirth, A.C. 1970. Gradwohl's Clinical Laboratory methods and diagnosis: 7th edition: vol. I and vol. II: C. V. Mosby Company.
 Gehring, U., J. Heinrich, G. Hoek, M. Giovannangelo. 2007. Bacteria and mould components in house dust and children's allergic sensitization.

- Eur. Respir. J.*, 29: 1144-1153.
- Gupta, P.R., D.K. Mangal. 2006. Prevalence of risk factors for bronchial asthma in adults in Jaipur district of Rajasthan. *Lung India*, 23: 53-58.
- Henry, D. 1992. Isenberg Clinical Microbiology Procedures Handbook, Vol I, 1992, *American Soc. Microbiol.*
- Holt, S.J.G., N.R. Krieg, P.H.A. Sneath *et al.* 1994. Bergeys Manual of Determinative Bacteriology. 9th edition; William and Wilkins, Baltimore.
- Jindal, S.K. 2006. Respiratory Disease Epidemiology In India. *Lung India*, 23: 93-94.
- Nagayama Y., Tsubaki, T. 1999. Role of bacterial infection in the exacerbation of acute or prolonged asthma attack in children *Allergol. Int.*, vol. 48, no.02, pp 137-144(8).
- Oehling, A.K. 1999. Bacterial infection as an important triggering factor in bronchial asthma. *J. Investg. Allergol Clin. Immunol.*, 9(1): 6 -13.
- Reba Kanungo, A. Kumar, S. Bhattacharya. 2000. Antimicrobial Resistance among common bacterial respiratory pathogens *Indian J. Med. Microbiol.*, 18(2): 55-61.
- Reeves, S., D.S., Philips, I., Williams, J. D., Wise, R. 1978. Laboratory methods in antimicrobial chemotherapy, Churchill Livingstone.
- Revised National Tuberculosis Control Programme (RNTCP). 1999. Manual for Laboratory Technicians Central TB division, Directorate General of Health services, Ministry of health and Family Welfare, Nirmal Bhavan, New Delhi.
- Schaechter, M., G. Medeff, D. Schlessinger. 1989. Mechanisms of Microbial Disease International edition, Williams and Wilkins.
- Sebastian, L., Johnston, Richard, J. 2005. Martin *Chlamydomphila pneumoniae and Mycoplasma pneumoniae : A Role in Asthma Pathogenesis ? Am. J. Respir. Crit. Care Med.*, Vol. 172, pp 1078 -1089.
- Seema, M.T., P.R. Bindhu. 1997. Serum Immunoglobulins in Asthma *Indian J. Med. Microbiol.*, 15(2): 77-78.
- Shrestha, U., Singh, A., B.M. Pokhrel. Cross-sectional study of respiratory pathogens and their antibiotic susceptibility pattern in Tribhuvan University Teaching Hospital. *J. Institute of Med.*, 28: 2: 5-9.
- Tarfe, K.S., Deshpande, S.D. 2015. A typical bacteria & Candida: Threats for allergic respiratory diseases. *Life Sci. Int. Res. J.*, vol 2 spl Issue, ISSN 2347-8691.
- Ten Brinke, A., P.J. Sterk, A.A.M. 2005. Risk factors of frequent exacerbations in difficult to treat asthma. *Eur. Resp. J.*, 26: 812 – 818

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