Original Article

Bacterial profile of community acquired lower respiratory tract infections in adults at a tertiary care center

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Abstract

Introduction: Lower respiratory infections contribute significantly to the morbidity and mortality of the patients. It also puts significant burden on the cost of care. It includes entities like acute bronchitis (AB), community acquired pneumonia (CAP), and acute exacerbation of COPD (AECOPD). This study was aimed to determine the pathogens responsible for causation of LRTIs. **Materials and Methods:** Hundred hospitalized patients at M.G.M Medical College, Aurangabad between November 2012 and September 2014. Culture of clinical samples were done to seek major pathogens of lower respiratory tract infections. Identification and antimicrobial susceptibility patterns were determined using standard bacteriological techniques. **Results:** In our study of 100 cases of LRTI, acute exacerbation of COPD was the most common (55%), followed by pneumonia (39%) and acute bronchitis (6%).In cases of CAP, *Streptococcus pneumoniae* was the most frequent organism (15.38%) followed by *Klebsiella pneumoniaesubsp. pneumoniae* (7.69%) and *Staphylococcus aureus* (2.56%). In severe CAP, Streptococcus pneumoniae (20%) was the only pathogen detected. In acute exacerbation of COPD, *Streptococcus pneumoniae* was the most frequent organism (14.54%) followed by *Pseudomonas aeruginosa* (12.72%), *Klebsiella pneumoniaesubsp. pneumoniae* (5.5%), and *Escherichia coli* (3.6%). *Haemophilus influenzae* and *Moraxella catarrhalis* were not detected in our study. **Conclusion:** The current data provide relevant information about distribution of major pathogens of lower respiratory tract in infections in India.

Keywords: Acute bronchitis, acute exacerbation of chronic obstructive pulmonary disease (AECOPD), bacterial profile, community acquired pneumonia (CAP), lower respiratory tract infections (LRTI).

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INTRODUCTION

Lower respiratory tract infections are those which are present for twenty one days of less, having cough as the main symptom with at least one other lower respiratory tract symptom (sputum production, dyspnoea, wheeze or chest discomfort/pain) and no other explanation. It is an umbrella term which includes syndromes like acute bronchitis (AB), community acquired pneumonia (CAP), and acute exacerbation of COPD (AECOPD).[1] In most of the cases, pathogen is not identified. However, in a few cases, especially in the young, the aetiology is polymicrobial. In cases of CAP, amongst extracellular bacteria, *S. pneumoniae* are in the first place, followed by *Hemophilusinfluenzae*, *Staphylococcus aureus* and *Moraxella catarrhalis*. Methicillin Resistant *Staphylococcus aureus* (MRSA), which was predominantly a nosocomial pathogen, has spread into the community, and has become an etiological agent of CAP. As far as intracellular bacteria are concerned, *Mycoplasma pneumoniae* are the most common followed by *Legionella* and *Chlamydia* species.Viruses cause upto 60% cases of community acquired LRTI and up to 30% cases of CAP. Recently occurring sporadic viral pneumonias are due to new viruses, avian influenza virus,

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hantavirus and coronavirus. In cases of AECOPD, more or less the same organisms are responsible. Additionally, long term colonization of *P. aeruginosa* occurs in COPD patients. The most common viral causes of AECOPD are rhinovirus, influenza virus, parainfluenza virus and Respiratory syncytial virus (RSV).[1] Studies determining the aetiology of different forms of lower respiratory tract infections:

Acute bronchitis (AB)

Macfarlane *et al.* (2001) investigated 316 patients. Pathogens were found in 173 (55%) cases: extracellular bacteria in 82 (*Streptococcus pneumoniae*54, *Haemophilus influenzae*31, *Moraxella catarrhalis*7), atypical organisms in 5 (*Chlamydia pneumoniae*55, *Mycoplasma pneumoniae* 23), and viruses in 61 (influenza 23).[2]

Community-acquired pneumonia (CAP)

Blanquer *et al.*(1991) conducted a study of 510 patients with pneumonia. A cause was identified in only 281cases-208 of bacterial, 60 of viral, and 13 of mixed infection. The most commonmicroorganisms were *Streptococcus pneumoniae* (14.5%), *Legionella* sp (14%), Influenza virus (8%), and *Mycoplasma pneumoniae* (4%).[3] Karalus *et al.* (1991) conducted a prospective study of 92 patients. A microbiological diagnosis was established in72%: *Streptococcus pneumoniae* (33%), *Mycoplasma pneumoniae* (18%), andinfluenza A virus (8%) were the most common microorganisms. Othercausative organisms were *Legionella pneumophila* (4 cases), *Staphylococcus aureus* (3), *Klebsiella pneumoniae* (2), *Haemophilus influenzae* (2), *Nocardiabrasiliensis* (1), and *Acinetobacter calcoaceticus* (1). *Chlamydia* species, influenza B virus and adenovirus were each found in one case; all were cultured on nasopharyngeal aspirates.[4] Garbino *et al.* (2002) studied three hundred and eighteen adult patients with CAP requiring hospitalization in seven large medical centres in Switzerland during two winter periods. The most frequently isolated organisms were *Streptococcus pneumoniae* (12.6%), *Haemophilus influenzae* (6%), *Staphylococcus aureus* (1.6%), and *Moraxella catarrhalis* (1.6%). Atypical pathogens were also found with the following distribution: *Mycoplasma pneumoniae*, 7.5%; *Chlamydia pneumoniae*, 5.3%; and *Legionella pneumophila*, 4.4%. The microbiological diagnosis in CAP could be established in only about 50% of cases using a combination of several diagnostic tools.[5] Mcnabb *et al.* (1984) studied 80 consecutive adults admitted to St Stephen's Hospital with community-acquired pneumonia.*S. pneumoniae* was diagnosed as the predominant cause of pneumonia in 40 (50%). Evidence of *Mycoplasma pneumoniae* infection was not found in any of the patients, and *Legionella pneumophila* serology was positive only in one case. In 29 patients (36%) no organism was demonstrated. The diagnosis of pneumococcal infection was concluded in 15 cases by isolating *S. pneumoniae* from the sputum, in another 13 cases by demonstrating pneumococcal capsular antigen in sputum, and in 12 cases by detecting pneumococcal antigen in serum only. Only 2 cases with pneumococcal pneumonia had bacteraemia.[6]

Holmberg *et al.* (1987) conducted a prospective study of 147 adult patients with community-acquired, radiologically verified, hospital treated pneumonia at the Department of Infectious Diseases, Orebro Medical Center Hospital, Orebro, Sweden. Special efforts to diagnose pneumococcus were accomplished by antigen detection of the pneumococcal C-polysaccharide (PnC) in sputum and saliva samples and by serological methods for determination of antibody titres against PnC. A pneumococcal aetiology was found in 46.9% of the patients, including 8.1% with mixed (double) infections.[7] Leesik *et al.* (2006) conducted a prospective study of the aetiology of community-acquired pneumonia in 209 inpatients at the Lung Hospital of Tartu University, Estonia. Streptococcus pneumoniae, beta-hemolytic streptococci, *Klebsiella pneumoniae*, and *Moraxella catarrhalis* were the most frequently found (22.0, 12.2, 11.4, and 10.2%, respectively). Mixed aetiology was detected in 17.2%. Frequency of Gram-negative pathogens was higher than gram-positives, and were significantly more frequent in patients aged > or =60 years as in those with underlying diseases. An age of > or =60 years and previous antibacterial therapy were significant risk factors for *Klebsiella pneumoniae* pneumonia.[8] Song *et al*. (2008) performed a prospective observational study of 955 cases of adult CAP in 14 hospitals in eight Asian countries. *Streptococcus pneumoniae* (29.2%) was the most common pathogen, followed by *Klebsiella pneumonia* (15.4%) and *Haemophilus influenzae* (15.1%). Serology was positive for *Mycoplasma pneumoniae* (11.0%) and *Chlamydia pneumoniae* (13.4%). Only 1.1% had *Legionella pneumophila* urinary antigen test positive.[9] Ortquist *et al.* (1990) prospectively studied 277 hospitalized adult patients with communityacquired pneumonia. The aetiology was established in 68%, with *S. pneumoniae* as the main cause.[10] Logroscino *et al*. (1999) conducted a prospective multicentre observational trial between October 1994 and February 1996. Out of 409 cases studied, the aetiology was defined by serological and quantitative microbiological tests in 184 (44.9%) patients. A total of 194 strains of pathogen were identified. The most frequently detected pathogen was *Streptococcus pneumoniae* (18.5% of pathogen strains). High percentages of intracellular pathogens (32.5%), mostly due to *Chlamydia pneumoniae* (13.4%), and of

Enterobacteriaceae and *Pseudomonas aeruginosa* (12.5%) were also found.[11] Rello *et al*. (2003) conducted a study to determine the impact of microbiological investigations on therapeutic decisions and outcome in patients with severe community-acquired pneumonia (SCAP). It was a retrospective analysis of prospectively collected data. The study included 204 consecutive patients admitted to intensive care with SCAP. The microbiologic diagnosis was found in 57.3% of patients. The most common pathogens were *Streptococcus pneumoniae*, *Legionella pneumophila*, and *Haemophilus influenzae*.[12]

Acute exacerbation of chronic obstructive pulmonary disease (AECOPD)

Ko*et al*. (2007) aimed to study the infectious aetiology in AECOPD. Patients admitted to an acute care hospital in Hong Kong with an AECOPD were recruited prospectively. Amongst sputum samples from the 530 episodes of AECOPD hospital admissions, 13.0%, 6.0%, and 5.5%, respectively, had *Haemophilus influenzae*, *Pseudomonas aeruginosa*, and *Streptococcus pneumoniae*. Among the 505 hospital admissions with patients who had NPA samples saved, 5.7%, 2.3%, 0.8%, and 0.8%, respectively, had influenza A, respiratory syncytial virus (RSV), influenza B, and parainfluenza 3 isolated from viral cultures. Paired serology test results revealed a fourfold rise in viral titres in 5.2%, 2.2%, and 1.4% of cases, respectively, of influenza A, RSV, and influenza B.[13] Eller *et al.* (1998) analysed clinical data and sputum culture results from 211 unselected COPD patients admitted to their hospital with an acute infective exacerbation of COPD. The protocol criteria of reliable microbiologic results and reproducible lung function tests were fulfilled in 112 patients. Bacteria were classified into three groups: group 1 contained *S. pneumoniae* and other Gram-positive cocci; group 2, *H.influenzae* and *Moraxella catarrhalis*; and group 3, *Enterobacteriaceae*and *Pseudomonas* species. For all patients together, the most frequently isolated bacteria were group 3 organisms (*Enterobacteriaceae*and *Pseudomonas* species, 48.2%). This was followed by group 1 organisms (*S. pneumoniae* and other Gram-positive cocci, 30.4%), and group 2 organisms (*H.influenzae* and *M.catarrhalis*, 21.4%).[14] Roche *et al*. (2007) conducted a study to evaluate the yield of sputum microbiological examination in hospitalized AECOPD patients with purulent sputum. Two hundred consecutive exacerbations in 118 patients were studied. Patients underwent sputum microbiological examination on admission and baseline lung function tests and CT scans were noted. Factors causing positive culture were analysed. Sputum culture was positive (more than or equal to 10^7 CFU/ml) in 59% of samples. *Haemophilus influenzae*and *Streptococcus pneumoniae* werethe most frequent pathogens. *Pseudomonas* spp. were found in 8.5% of all patients, who were older and had a FEV_1< 50% of predicted. Only 25% of sputum samples satisfied all quality criteria for lower respiratory tract specimen. Sputum culture was positive in a high proportion of these samples (80.5%), but also in 50% of samples with > 25 leukocytes but > 10 epithelial cells per field. Finally, a predominant aspect in Gram stain was found in all positive samples.[15] Alamoudi *et al*. (2007) undertook a study to determine the predominant bacterial pathogens cultured from sputum in community-based patients with AECOPD. Forty-five stable COPD patients were prospectively followed in the outpatients' clinic of King Abdulaziz University Hospital. *Moraxella catarrhalis* (25.2%), *Pseudomonas aeruginosa* (12.2%) and *Haemophilus influenza* (11.5%) were the most common isolated pathogens.[16]

MATERIALS AND METHODS

Study type: Observational

Study design: Observational Model: Cohort Time perspective: Prospective

Data source: Hundred hospitalized patients at M.G.M Medical College, Aurangabad between November 2012 and September 2014. The approval of the Ethics Committee was taken before commencement of study. Written informed consent was taken from all the patients enrolled in the study

Inclusion Criteria

These included patients with an acute illness (present for 21 days or less) usually with cough as the main symptom with at least one other lower respiratory tract symptom (sputum production, dyspnoea, wheeze, or chest discomfort/pain) and no other alternative explanation (e.g. asthma).[1] In addition to above, if fever > 4 days or dyspnoea/tachypnoea was present and chest radiograph shows a lung shadow that is likely to be new, Community Acquired Pneumonia (CAP) was diagnosed, if no other obvious cause was present. In the elderly, lung shadowing in the presence of any acute illness (unspecified) was diagnosed as CAP, if no other apparent cause is present.[1] In the absence of lung shadowing, if the patient is not a known case of any chronic lung disease, acute bronchitis was diagnosed.[1] If however the patient is a known case of COPD, and in the absence of shadowing, there was a worsening of symptoms (cough, dyspnoea and/or sputum production) warranting change in management, then exacerbation of COPD was diagnosed.[1] If lung shadowing was present during an exacerbation of Chronic Obstructive Pulmonary Disease (COPD), then CAP was diagnosed.[1] If a patient of CAP had an episode of witnessed aspiration or the pneumonia occurred in the

presence of risk factors for aspiration such as reduced consciousness level or dysphagia due to mechanical or neurological upper digestive tract dysfunction, then aspiration pneumonia was diagnosed.[1] Only adults equal to or more than 18 years of age were included.
Adults with age equal to or more than 60 years were considered as elderly. Community acquired pneumonia requiring admission to ICU because of mechanical ventilation or unstable condition requiring intensive medical or nursing care were termed as severe community acquired pneumonia.[17]

Exclusion Criteria

1. Patients with known respiratory disorder other than CAP, COPD or acute bronchitis like, but not limited to lung cancer, pulmonary tuberculosis, lung fibrosis, cystic fibrosis and sarcoidosis.[18]
2. Primary immune deficiency or secondary immune deficiency related to HIV infection, or drug or systemic disease-induced immunosuppression. Patients receiving oral corticosteroid therapy were not included as this is a not uncommon situation for patients admitted on medical take.[19]
3. Patients with hospital associated pneumonia (HAP), ventilator associated pneumonia (VAP) or health care associated pneumonia (HCAP). HAP is defined as pneumonia that occurs 48 hours or more after admission, which was not incubating at the time of admission. VAP refers to pneumonia that arises more than 48–72 hours after endotrachealintubation. HCAP includes any patient who was hospitalized for two or more days within 90 days of infection; resided in a nursing home or a long term care facility, received intravenous antibiotic therapy, chemotherapy, or wound care within the past 30 days of current infection; or attended a hospital or haemodialysis clinic.[20]
4. Children with CAP (<18 years of age).

Specimen collection and transport:

1. Sputum: Wherever possible, early morning sputum samples were obtained after the patients brushed their teeth and gargled with water. This procedure reduces the contaminating oropharyngeal flora. The sputum was collected in a wide mouth container and was promptly sent to the laboratory for processing.[21]
2. Broncho-alveolar lavage (BAL): Semi quantitative cultures were performed[21].

Diagnostic thresholds for BAL have ranged from 10^3 to 10^5cfu/ml. Adopting a lower diagnostic threshold breakpoint increases the sensitivity and lowers the specificity of the test. The diagnostic threshold in our study was 10^3cfu/ml.[21]

Table 1: Specimens submitted for the study

	Specimens (n=100)
Sputum	98 (98%)
Broncho-alveolar lavage (BAL)	2 (2%)

Microscopy:[22]

Sputum: A Gram stained was performed on the purulent portion of the sputum and was examined. The slides were evaluated for quality under low power (x10). Salivary contamination was detected by noticing the presence of squamous epithelial cell, and purulence was detected by noticing the presence of polymorph nuclear cells. Sputum were considered of good quality if they had < 10 epithelial cells and > 25 polymorph nuclear cells. Otherwise the sputum sample was considered contaminated with saliva and rejected.

Culture:

Specimens were inoculated on blood agar, chocolate agar and MacConkey agar. Blood and chocolate agar plates were incubated in a candle jar with a moistened gauze kept at the bottom of the jar. MacConkey agar was incubated in ambient air. Incubation was done at 35 to 37^{o}C for 48 hours. Identification and antimicrobial susceptibility testing of isolates was done as per standard protocols

OBSERVATION AND RESULTS

In the present study of 100 cases of LRTI, acute exacerbation of COPD was the most common (55%), followed by pneumonia (39%) and acute bronchitis (6%). In our study, no organism was detected in patients with acute bronchitis.

Table 2: Organisms detected in community acquired pneumonia

	Community acquired pneumonia (n=39)
Streptococcus pneumoniae	6 (15.38%)
Klebsiella pneumoniae subsp. Pneumoniae	3 (7.69%)
Staphylococcus aureus	1 (2.56%)
Haemophilus influenza	0 (0%)
Moraxella catarrhalis	0 (0%)
Total	**10 (25.64%)**

In our study, aetiology was detected in 25.64% of cases of community acquired pneumonia. *Streptococcus pneumoniae* was the most frequent organism (15.38%) followed by *Klebsiella pneumoniae subsp. pneumoniae* (7.69%) and Staphylococcus aureus (2.56%). *Haemophilus influenzae* and *Moraxella catarrhalis* were not detected in our study. Out of six cases of *Streptococcus pneumoniae*, one was isolated in severe community acquired pneumonia. Out of cases of *Klebsiella pneumoniae* pneumonia, 66% were detected in elderly patients. In the present study, out of all cases of community acquired pneumonia, 12.82% of patients had severe community acquired pneumonia requiring admission to intensive care unit. In the present study, in cases of severe community acquired pneumonia, *Streptococcus pneumoniae* (20%) was the only pathogen detected.

Table 3: Organisms detected in acute exacerbation of COPD

	Acute exacerbation of COPD (n=55)
Streptococcus pneumoniae	8 (14.54%)
Pseudomonas aeruginosa	7 (12.72%)
Klebsiella pneumoniae subsp. Pneumoniae	3 (5.5%)
Escherichia coli	2 (3.6%)
Haemophilus influenza	0 (0%)
Moraxella catarrhalis	0 (0%)
Total	**20 (36.36%)**

In this study, aetiology was detected in 36.36% of cases of acute exacerbation of COPD. *Streptococcus pneumoniae* was the most frequent organism (14.54%) followed by *Pseudomonas aeruginosa* (12.72%), *Klebsiella pneumoniae subsp pneumoniae* (5.5%), and Escherichia coli (3.6%). *Haemophilus influenzae* and *Moraxella catarrhalis* were not detected in our study.

DISCUSSION

In our study, no organism was detected in acute bronchitis, whereas Macfarlane *et al*. had detection of 55%. They had a larger sample size (n=316). In their study, in addition to standard bacteriological techniques, pneumococcal infection required detection of one or more of sputum pneumococcal capsular antigen, serum pneumococcal immune complexes (ICs) including pneumolysin-specific IC (in a titre of more than or equal to100), pneumococcal surface antigen IC (titre more than or equal to100), C-polysaccharide-specific IC (titre more than or equal to150), or a twofold or greater rise in pneumolysin and C-polysaccharide-specific antibodies. *S. pneumoniae* was the most common bacterial pathogen isolated (17%). Other bacterial pathogens detected were *H. influenzae* (9.81%) and *M. catarrhalis* (2.21%). This could be attributed also to the additional serological testing done for *H. influenzae* and *M. catarrhalis*. In addition to the above, serological and molecular tests were done for atypical and viral pathogens. In our study, aetiology of community acquired pneumoniae was detected in 25.64% of cases. However several other authors have detected higher rates of detection of microorganisms. Blanquer *et al*. had a detection of 55%. The reason for this could be attributed to the following factors. Firstly, their sample size was much larger (n=510) as compared to ours. In their study, in addition to bacterial pathogens, atypical pathogens and viruses were also sought. Three blood cultures were performed before treatment. *Legionella* was sought by culture on BCYE-alpha medium and direct immunofluorescence tests. Capsular antigens of *Streptococcus pneumoniae* and *Haemophilus influenzae* type b were sought in blood and sputum by latex agglutination. Serology was done for atypical pathogens and viruses. Antibodies to *L. pneumophila* were detected by indirect immunofluorescence. Karalus *et al*. had a detection of 72%. Their sample size was very similar to ours (n=92). In their study, in addition to bacterial pathogens, atypical and viral pathogens were also sought. Additional test were done other than sputum cultures. Pneumococcal antigen was detected by latex agglutination and counter immune electrophoresis. Direct fluorescent antibody tests for *L. pneumophila*. Nasopharyngeal aspirates were submitted for viral culture, chlamydial culture, *L. pneumophila* and direct fluorescent tests. Two set of blood cultures were taken. Pneumococcal antigen was detected in urine. Paired serum samples were taken for *Legionella* serology. *Chlamydial* species were also sought by culture and serology. Empirical antibiotic treatment was given only to 47 (51.08%) patients. However in our study 89.74% received empirical antibiotic therapy. All these factors contributed to low detection rate in our study. Garbino *et al*. (26) also had a higher detection of 50%. Their sample size (n=318) was larger than ours (n=100). In their study, blood cultures were also taken. Serology testing for atypical pathogens was performed. *Legionella* antigen was detected in urine. In addition to the above, molecular techniques i.e. PCR was employed for *Mycoplasma pneumoniae* and *Chlamydia pneumoniae*. In our study, *S.pneumoniae* was the most common organism isolated in community acquired pneumonia. Many other authors had a similar finding. In our study, *S. pneumoniae* was isolated in 15.38% cases of community acquired pneumonia. However, some authors had a higher rate of detection of *S. pneumoniae*. McNabb *et al*. had a higher detection of *S. pneumoniae* (50%). But in their study, in addition to sputum culture, pneumococcal antigen detection was done in sputum and

serum. Holmberg *et al*. also had a higher detection of *S. pneumoniae* (46.9%). This is probable, because in their study, pneumococcal antigen detection was done in sputum and pneumococcal antibodies were sought in serum. However our findings are similar to some authors when compared to detection of *S. pneumoniae* in sputum cultures only. McNabb *et al*. had a detection of *S. pneumoniae* in 18.75% of sputum cultures only. Blanquer*et al.* and Kalarus*et al*. had detection of *S. pneumoniae* in 7% and 13% of sputum cultures, respectively. In our study, *K. pneumoniae subsp. pneumoniae* was isolated in 7.69% of cases. This finding is similar to Leesik*et al*., who detected *K. pneumoniae* in 11.4% cases of community acquired pneumonia. Song *et al.* also had a similar detection of *Klebsiella pneumoniae subsp. pneumoniae* in15.4% cases of community acquired pneumonia. However, *K. pneumoniae subsp. pneumoniae* was infrequently detected in sputum cultures of community acquired pneumonia in some studies. Karalus*et al*. and Garbino*et al*. had a detection of 2.6% and 0.6% in sputum cultures, respectively. Other *Enterobacteriaceae*were also infrequently detected in community acquired pneumonia. Karalus*et al.* had a detection of *E. coli* (1.33%) and *Serratiamarcescens* (1.33%). Ortquist *et al*. and Logroscino *et al.* detected *Enterobacteriaceae* in 1% and 11.4% cases of community acquired pneumonia. However we did not isolate any organism other than *K. pneumoniae subsp. pneumoniae* from the *Enterobacteriaceae* family. Out of cases of *Klebsiella pneumoniae* pneumonia, 66.66% were detected in elderly patients. The higher detection of *Enterobacteriaceae* was also seen in other studies. In the study by Ortquist*et al*., infection with *Enterobacteriaceae* was seen only in the elderly (equal to or more than 65 years) whereas in study by Leesik *et al*., *Enterobacteriaceae* were seen more in elderly (equal to or more than 60 years of age). In our study, *Staphylococcus aureus* was detected as a cause of community acquired pneumonia in 2.56% cases from sputum cultures. Other authors had a similar finding. Karalus *et al*. (2.66%), Garbino*et al*. (1.6%), McNabb *et al*. (1.25%), and Ortquist *et al.* (1%) also isolated *Staphylococcus aureus* from sputum cultures.

In our study, *Streptococcus pneumoniae* (20%) was the only organism isolated as cause of severe community acquired pneumonia. Rello *et al*. 2003 also had *Streptococcus pneumoniae* (20.1%) as the most common organism. But other organisms were also isolated. *P. aeruginosa* (3.9%), *Staphylococcus aureus* (2.4%), *Escherichia coli* (0.9%), *Klebsiella pneumonia* (0.5%), and *Enterobacter aerogenes* (0.5%). Aetiology was detected in 57.3% patients. This was probable, because they had a larger sample size (n=204). Additionally, blood cultures, pleural fluid cultures, bronchoalveloar lavage, protected specimen brush sampling, and serology were also done. In our study, organisms were detected in 36.36% cases of acute exacerbation of COPD. Ko *et al*.(2007) had a similar isolation of 32.3%. However Eller *et al*. had a higher isolation (53.08%) in patients with acute exacerbation of COPD. The predominant isolated organism in our study was *S. pneumoniae* (14.54%) in acute exacerbation of COPD. Eller *et al*. (16.96%) and Roche *et al*. (7.5%) also had *S. pneumoniae* as the predominant organism. Ko *et al.* had *H. influenzae* (13%) as the predominant organism whereas Alamoudi *et al*. had *P. aeruginosa* (12.2%) as the predominant organism. The variability of isolation of predominant organism could be attributed to the geographical distribution of organisms. In our study, *P. aeruginosa* was isolated in 12.72% cases of acute exacerbation of COPD. Eller *et al.* had a similar isolation (12.5%) of *P. aeruginosa*. However Roche *et al*. (8.5%) and Ko *et al*. (6%) had a lower isolation of *P. aeruginosa*. In our study, *Klebsiella pneumoniae subsp. pneumoniae* was detected in 5.5% cases of acute exacerbation of COPD. Eller *et al*. (6.25%) and Alamoudi *et al*. (4.3%) had similar isolation. Ko *et al.* (1.9%) had a lower isolation of *K. pneumoniae subsp. pneumoniae*. In our study, *E.coli* was detected in 3.6% cases of acute exacerbation of COPD. *E.coli* was also detected by Eller *et al.* (4.46%), Roche *et al.* (1.5%), Alamoudi *et al*. (0.7%), and Ko *et al*. (0.4%). Other organisms inconsistently and infrequently detected in acute exacerbations of COPD include Methicillin Resistant *Staphylococcus aureus* (MRSA), *Enterobacteriaceae* (*Enterobacter* spp., *Citrobacter*spp, *Serratia* spp., *Proteus* spp.,), *Acinetobacter* spp., and *Stenotrophomonasmaltophila*. Our study was not devoid of limitations. We were unable to get samples like pleural fluid, blood, protected specimen brush (PSB) samples, and lung biopsy for culture, which could have established the diagnosis more specifically. Antigen testing was not done in clinical samples for *S. pneumoniae*, *Haemophilus influenzae* and *Legionella pneumophila*. Atypical pathogens were not sought, either by culture, immunofluorescence or by serology. Finally, methods were not used as very few of them have been validated for diagnosis of respiratory pathogens.

CONCLUSION

Lower respiratory infections contribute significantly to the morbidity and mortality of the patients. It also puts significant burden on the cost of care. In a country like ours, sputum culture may be useful to detect the aetiology and susceptibility patterns. However it may be unable to differentiate between colonizers and pathogens. The more invasive approaches like pleural fluid culture, blood

cultures, lung biopsy are more specific but may not be affordable to many and carry risk of complications. Many pathogens causing LRTI are not cultivable and require serological and molecular techniques. Hence to determine the aetiology of LRTIs may be a laborious and expensive task. The molecular techniques for pathogens causing LRTI are in infancy and future studies are required to ascertain the specific gene targets in pathogens. Moreover it requires expertise which may not be available everywhere. Many patients receive empirical antibiotic therapy, which may not be warranted. The antibiotic pressure gives rise to emergence of antibiotic resistance which further aggravates the issue. Hence there is a need to develop guidelines pertaining to workup and treatment of patients with LRTI. These guidelines should be specifically tailored to our country, keeping in mind the socio-economic status of the population.

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