Treatment of Acute Exacerbation of COPD

AG Ghoshal¹, Raja Dhar², Susmita Kundu³

COPD exacerbation is a nightmare not only to all clinicians but also for the patients themselves. Despite adequate COPD control, patients experience episodes of exacerbation at times with a huge impact on the overall health condition, both physically and mentally. Moreover, COPD exacerbation incurs a huge cost and can cause significant dent in pulmonary function and the quality of life. It is therefore extremely important for practicing physicians to:

- Recognize the crisis early
- Perform optimal management under appropriate settings
- Escalate and de-escalate the therapy based on patient response
- Bring back the patient to the initial baseline status
- Help the patient and family to cope with the situation with adequate support
- Discharge the patient in a stable clinical condition
- Give necessary instruction about the proper follow up.

Definition¹

An exacerbation of COPD is an event in the natural course of COPD characterised by an acute change in the patient’s baseline dyspnoea or breathing difficulty, cough and/or sputum production beyond day-to-day variability sufficient to warrant a change in management.

Its cause can be infective or non-infective in nature.

Classification of exacerbations¹

In the absence of a universal agreed criterion, the following operational classification can help rank the clinical severity of the episode and its outcome.

- Level I: treated at home.
- Level II: requires hospitalization.
- Level III: leads to respiratory failure.

Causes

About 50% of COPD exacerbations are caused by lower respiratory tract infections, while the remaining are caused by exposure to indoor or outdoor air pollutants, changes in weather, and several host factors including prior compliance to therapy.² Bacterial etiology in AECOPD varies, one study from India reporting Gram negative bacteria as the most common organism, which may not reflect the entire scenario.³ Another study from India stressed evidence of past pulmonary tuberculosis in 28.4% of patients and active tuberculosis in 5 patients with Type II diabetes mellitus (Table 1).⁴

Symptoms

Symptoms of COPD exacerbation include

i. Increase in cough
ii. Increase in breathlessness
iii. Increase in sputum volume and change in its colour (white to green, yellow or blood streaked)
iv. Fever
v. Chest pain
vi. Increased tiredness
vii. Increase in oxygen requirement (for those on long-term oxygen therapy)

Usually patients present with varying combination of these symptoms. During clinical assessment, further objective evaluation is necessary. Several clinical elements must be considered when evaluating patients with exacerbations. These include the assessment of severity of underlying COPD, the presence of comorbidities and history of previous exacerbation. The physical examination should evaluate the effect of the episode on hemodynamic and respiratory systems. The diagnostic procedures to perform such as chest radiology, sputum and blood examination and spirometry would depend on the clinical setting of evaluation.

Where to treat⁵,⁶

The decision to treat a patient at home or hospital is based both on subjective evaluation as well as objective criteria. The choice of the patient/patients’ family should also be taken into account.

Table 2 below offers a ready-reckoner

However it must be noted that there may be a sudden deterioration of condition and the decision to treat at home or at hospital can change rapidly.

Table 3 shows the elements of the clinical evaluation and diagnostic procedures that are usually informative in patients with exacerbations according to the severity of the episode.⁷,⁸

Investigations

 Diagnosis of an exacerbation is primarily clinical and not so much dependant on investigation results though they are useful in assessing the severity and planning the optimum treatment. The outline of the general investigation is provided in table 3.

Primary care

In patients with an exacerbation managed in primary care:

- Sending sputum samples for culture is not recommended in routine practice.⁸

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Table 1: Precipitating factors for AECOPD

<table>
<thead>
<tr>
<th>Infectious process</th>
<th>Viral (Rhinovirus spp., influenza); Bacteria (Streptococcus pneumonia, Haemophilus influenzae, Moraxella catarrhalis, Enterobacteriaceae spp., Pseudomonas spp.).</th>
</tr>
</thead>
<tbody>
<tr>
<td>Environmental conditions</td>
<td>Sudden change in temperature and humidity, air pollution, exposure to tobacco smoke, noxious gases or irritating chemicals</td>
</tr>
<tr>
<td>Host factors</td>
<td>Patients with poor general heath condition, poor nutritional status, immunocompromised status, lack of compliance with prescribed medical therapy.</td>
</tr>
</tbody>
</table>

Table 2: Where to treat

<table>
<thead>
<tr>
<th>Level</th>
<th>Where to treat</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Treated at home</td>
</tr>
<tr>
<td>II</td>
<td>Requires hospitalization</td>
</tr>
<tr>
<td>III</td>
<td>Leads to respiratory failure</td>
</tr>
</tbody>
</table>

Table 3: Elements of clinical evaluation and diagnostic procedures

- Chest radiology
- Sputum and blood examination
- Spirometry
• Pulse oximetry is useful if there are clinical features of a severe exacerbation.

Patients referred to hospital
In all patients with an exacerbation referred to hospital:
• A chest radiograph should be obtained
• Arterial blood gas tensions should be measured and the inspired oxygen concentration recorded
• An ECG should be recorded to exclude cardiac co-morbidities

Table 2: Factors to consider where to treat AECOPD

<table>
<thead>
<tr>
<th>Factor</th>
<th>Indication to treat at home</th>
<th>Indication to treat at hospital</th>
</tr>
</thead>
<tbody>
<tr>
<td>Able to cope at home</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Severity of breathlessness</td>
<td>Mild</td>
<td>Severe</td>
</tr>
<tr>
<td>General health condition</td>
<td>Good/Satisfactory</td>
<td>Poor/deteriorating</td>
</tr>
<tr>
<td>Level of activity</td>
<td>Good</td>
<td>Poor/confined to bed</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Worsening peripheral oedema</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Level of consciousness</td>
<td>Normal</td>
<td>Impaired</td>
</tr>
<tr>
<td>Already receiving LTOT</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Social circumstances</td>
<td>Good/adequate support</td>
<td>Living alone/not coping</td>
</tr>
<tr>
<td>Acute confusion/ altered</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>mental status</td>
<td>Rapid rate of onset</td>
<td>No</td>
</tr>
<tr>
<td>Significant co-morbidity</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>(particularly heart disease</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>and insulin-dependent</td>
<td>SaO2 &lt; 90%</td>
<td>Yes</td>
</tr>
<tr>
<td>diabetes)</td>
<td>Changes on the chest</td>
<td>Present</td>
</tr>
<tr>
<td>radiograph</td>
<td>Arterial pH level</td>
<td>≥7.35</td>
</tr>
<tr>
<td>Arterial PaO2</td>
<td>≥ 7kPa</td>
<td>&lt;7kPa</td>
</tr>
</tbody>
</table>

Patients referred to hospital
In all patients with an exacerbation referred to hospital:
• A chest radiograph should be obtained
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• An ECG should be recorded to exclude cardiac co-morbidities

Table 3: Stratification of AECOPD

<table>
<thead>
<tr>
<th>Level I: Outpatient treatment</th>
</tr>
</thead>
</table>

• A full blood count should be performed and urea and electrolyte concentrations should be measured
• If sputum is purulent, a sample should be sent for microscopy and culture
• Blood cultures should be taken if the patient has fever.

What should be the outpatient management for COPD Exacerbation?

The treatment of exacerbation has to be based on the clinical presentation of the patient, as shown in table 3.

Level I: Outpatient treatment

Bronchodilators^8,10
• Short-acting β₂-agonist and/or ipratropium by metered dose inhaler with spacer or nebuliser as needed
• Consider adding long-acting bronchodilator e.g., formoterol (by MDI plus spacer or nebulised route) if patient is not using it

Delivery systems for inhaled therapy during exacerbations
• Both nebulisers and hand-held inhalers can be used to administer inhaled therapy during exacerbations of COPD.
• The choice of delivery system should reflect the dose of drug required, the ability of the patient to use the device and the resources available to supervise the administration of the therapy.

Corticosteroids^11,12

In the absence of significant contraindications, oral corticosteroids should be considered in patients managed in the community who have an exacerbation with a significant increase in breathlessness which interferes with daily activities.
• Oral prednisone 30–40 mg per day for 10 days
• Consider using an inhaled corticosteroid ^11

Antibiotics^13,14

• May be initiated in patients with altered sputum characteristics.

Table 3: Stratification of AECOPD

<table>
<thead>
<tr>
<th>Clinical history</th>
<th>Level I (Treated at home)</th>
<th>Level II (Requires hospitalization)</th>
<th>Level III (Leads to respiratory failure)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Co-morbid conditions</td>
<td></td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Frequent exacerbations</td>
<td></td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Severity of COPD</td>
<td>Mild/Moderate</td>
<td>Moderate/Severe</td>
<td>Severe</td>
</tr>
<tr>
<td>Physical findings</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Haemodynamics</td>
<td>Stable</td>
<td>Stable</td>
<td>Stable/unstable</td>
</tr>
<tr>
<td>Use of accessory muscles</td>
<td>Not present</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Persistent symptoms after</td>
<td>No</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>therapy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnostic procedures</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxygen saturation</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Arterial blood gases</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Chest radiograph</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Blood tests</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Serum drug concentration^*</td>
<td>If applicable</td>
<td>If applicable</td>
<td>If applicable</td>
</tr>
<tr>
<td>Sputum gram stain and culture</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Electrocardiogram</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Keys: ^ unlikely to be present; ++ likely to be present; +++ very likely to be resent; # the more common co-morbid conditions associated with poor prognosis in exacerbations are congestive heart failure, coronary artery disease, diabetes mellitus, renal and liver failure; ^ Blood test include complete blood count, serum electrolytes, renal and lever function; * consider if patients are using theophylline, warfarin, carbamazepine, digoxin; $ consider if patient has been recently on antibiotics.
Outcome risk factors for poor exacerbations with Group B patients? What is the ideal pharmacologic management for these patients? Room discharge.5,6 Relapse rate, administration of aminophylline, and the use of nebulised bronchodilators, use of home oxygen, previous emergency room visit within 7 days, the number of doses of resistant organisms (β-lactamase producing, penicillin resistant S. pneumoniae), Enterobacteriaceae (K. pneumoniae, E. coli, Proteus, Enterobacter, etc). Group A plus presence of Group B plus Group C Severe exacerbations with risk factors for P. aeruginosa infection Group A mild exacerbations: no risk factor for poor outcome Group B moderate exacerbations with risk factors for poor outcome Group C severe exacerbations with risk factors for P. aeruginosa infection * Choice should be based on local bacterial resistance patterns, if available. * As the severity of COPD exacerbation is an important determinant of the type of microorganism, empirical antibiotics need to be guided accordingly. Options are: * If the patient has failed prior antibiotic therapy, antibiotics for group B may be initiated. However, there is a growing concern about undiagnosed tuberculosis and overuse of fluoroquinolones in India (Table 4). Patient education * Check inhalation technique * Consider use of spacer devices What should be the Inpatient management of COPD Exacerbation? Systemic Corticosteroids * Inhaled corticosteroids by MDI or hand-held nebuliser should be considered. * In the absence of significant contraindications, oral corticosteroids should be used in conjunction with other therapies in all patients admitted to hospital with an exacerbation of COPD. * Prednisolone 30 mg orally should be prescribed for 7 to 14 days. * If patient cannot tolerate oral intake, consider equivalent dose i.v. for up to 14 days (20 mg hydrocortisone is equivalent to 5 mg prednisolone, 4 mg methyl-prednisolone and 0.75 mg dexamethasone). * It is recommended that a course of corticosteroid treatment should not be longer than 14 days as there is no advantage in prolonged therapy. * Osteoporosis prophylaxis should be considered in patients requiring frequent courses of oral corticosteroids. Antibiotics (as discussed above) * Antibiotics should be started for patients with history of producing purulent sputum. * Any change in sputum characteristics like colour, consistency and volume should also prompt the initiation. * Antibiotics need not be started for patients having exacerbations without increase in sputum purulence, unless there are signs of consolidation on chest radiograph or clinical symptoms of pneumonia. * Antibiotic choice should be based on local bacteria resistance patterns, if available.

### Table 4: Stratification of AECOPD with likely bacteriological profile and treatment

<table>
<thead>
<tr>
<th>Group</th>
<th>Microorganisms</th>
<th>Oral Treatment</th>
<th>Alternative Oral Treatment</th>
<th>Parenteral Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group A</strong></td>
<td>Mild exacerbations: no risk factor for poor outcome</td>
<td>Patient with only one cardinal symptom need not receive antibiotics</td>
<td>β-lactum/β-lactamase inhibitor (co-amoxiclav).</td>
<td></td>
</tr>
<tr>
<td></td>
<td>H.influenzae, S.pneumoniae, M.catarrhalis, Chlamydia pneumoniae, Viruses</td>
<td>If indication then : - β-lactum (penicillin, ampicillin/amoxicillin) - Doxycycline - Trimethoprim/sulfamethoxazole.</td>
<td>Macrolides (azithromycin, clarithromycin, roxithromycin).</td>
<td>Cephalosporins (2nd and 3rd generations)</td>
</tr>
<tr>
<td><strong>Group B</strong></td>
<td>Moderate exacerbations with risk factors for poor outcome</td>
<td>β-lactum/β-lactamase inhibitor (co-amoxiclav).</td>
<td>Fluoroquinolones (gemifloxacin, levofloxacin, moxifloxacin)</td>
<td>β-lactum/β-lactamase inhibitor (co-amoxiclav, ampicillin/salbactum).</td>
</tr>
<tr>
<td></td>
<td>Group A plus presence of resistant organisms (β-lactamase producing, penicillin resistant S. pneumoniae), Enterobacteriaceae (K. pneumoniae, E. coli, Proteus, Enterobacter, etc)</td>
<td></td>
<td>Fluoroquinolones (ciprofloxacin, levofloxacin, moxifloxacin)</td>
<td>Fluoroquinolones (ciprofloxacin, levofloxacin-high dose) or β-lactum with P. aeruginosa activity</td>
</tr>
<tr>
<td><strong>Group C</strong></td>
<td>Severe exacerbations with risk factors for P. aeruginosa infection</td>
<td>In patients at risk for pseudomonas infections : - Fluoroquinolones (ciprofloxacin, levofloxacin-high dose)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Group B plus P. aeruginosa</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* If a patient is hypercapnic or acidotic, the nebuliser should be driven by compressed air, not oxygen (to avoid worsening hypercapnia). If oxygen therapy is needed it should be administered simultaneously by nasal cannulae. * The driving gas for nebulised therapy should always be specified in the prescription.

**Level II and III: Inpatient treatment**

As mentioned earlier, the decision to admit a patient is derived from subjective interpretation of clinical features, such as the severity of dyspnoea, determination of respiratory failure, short-term response to emergency room therapy, degree of cor pulmonale and the presence of complicating features, such as severe bronchitis, pneumonia or other co-morbid conditions. Other factors that identify "high-risk" patients include a previous short-term response to emergency room therapy, degree of cor pulmonale and the presence of complicating features, such as severe dyspnoea, determination of respiratory failure, short-term response to emergency room therapy, degree of cor pulmonale and the presence of complicating features, such as severe bronchitis, pneumonia or other co-morbid conditions. Other factors that identify “high-risk” patients include a previous exacerbation of COPD. * If patient cannot tolerate oral intake, consider equivalent dose i.v. for up to 14 days (20 mg hydrocortisone is equivalent to 5 mg prednisolone, 4 mg methyl-prednisolone and 0.75 mg dexamethasone). * It is recommended that a course of corticosteroid treatment should not be longer than 14 days as there is no advantage in prolonged therapy. * Osteoporosis prophylaxis should be considered in patients requiring frequent courses of oral corticosteroids. Antibiotics (as discussed above) * Antibiotics should be started for patients with history of producing purulent sputum. * Any change in sputum characteristics like colour, consistency and volume should also prompt the initiation. * Antibiotics need not be started for patients having exacerbations without increase in sputum purulence, unless there are signs of consolidation on chest radiograph or clinical symptoms of pneumonia. * Antibiotic choice should be based on local bacteria resistance patterns, if available.

**Bronchodilators**

- Short acting inhaled β₂-agonist (salbutamol 100-200 mcg inhaler/levosalbutamol) and/or
- Ipratropium MDI (20, 40mcg) with spacer or nebuliser as needed.
When sputum has been sent for culture, the sensitivity to antibiotics should be checked to ensure appropriate therapy. If Pseudomonas spp. and/or other Enterobactereaceae spp. are suspected, consider combination therapy such as Piperacillin/tazobactam (3.375 g q4h IV) or imipenem (500 mg q6h IV) or meropenem (1 g q8h IV) plus Amikacin (7.5 mg/kg q12h or 15 mg/kg q24h IV) or Levofloxacin (500mg daily) for 10-14 days.

Intravenous theophylline should only be used as an adjunct to the management of exacerbations of COPD if there is an inadequate response to nebulised bronchodilators. Care should be taken when using intravenous theophylline because of interactions with other drugs (e.g., ciprofloxacin, clarithromycin, allopurinol, phenytoin etc.) and potential toxicity if the patient has been on oral theophylline.

Respiratory stimulants

It is recommended that doxapram is used only when non-invasive ventilation is either unavailable or considered inappropriate.

Oxygen Therapy

The goal is to prevent tissue hypoxia by maintaining arterial oxygen saturation (SaO2) at >90%.

All patients with COPD exacerbation should have their arterial oxygen tension (PaO2), arterial carbon dioxide tension (PaCO2) and pH measured upon admission, during the course of treatment and whenever there is any change in patient’s clinical condition.

In case of unavailability of arterial blood gas measurement facility, oxygen saturation should be measured by Pulse Oximeters.

Even if oximeter is not available for any reason, oxygen should be given to all patients with an exacerbation of COPD who are breathless. But careful monitoring of the patient condition is essential to prevent excessive CO2 washout and cause respiratory depression since CO2 acts as the respiratory drive for many COPD patients.

It is important to note that prevention of tissue hypoxia supersedes CO2 retention issue.

The principal delivery device for oxygen comprises of nasal canula and venturi mask. Alternative delivery devices include nonrebreather mask, reservoir cannula, or transtracheal catheter.

The aim of oxygen therapy during exacerbation of COPD is to maintain adequate level of oxygen (SaO2 >90%) without worsening or precipitating respiratory acidosis or worsening hypercapnia. If pH falls below 7.35 (acidemia), consider mechanical ventilation.

Management of AECOPD in critical care

The decision to shift/admit a patient to the Critical Care Unit largely depends on subjective assessment and clinician's judgment, although various diagnostic parameters help in decision making.

Presence of the following features merits consideration for shifting to IRCU-

- Persistent respiratory distress despite standard management
- Inability to maintain oxygen level above 90% despite oxygen therapy by high-flow mask
- Persistent hypercapnia
- Abnormal blood gas parameters (PaO2< 60 mm of Hg and/or PaCO2>60 mm of Hg)
- Alteration of mental status, acute confusion, drowsiness
- Increased signs of infection (pyrexia, increased sputum purulence/volume etc)
- Significant abnormal changes in chest radiograph
- Clinical deterioration

**Assisted Ventilation**

Despite optimal pharmacologic management, severe cases of exacerbation necessitates ventilatory support, when the patient is unable to maintain the breathing function and/or unable to maintain sufficient gaseous exchange for normal physiologic function (Figure 1).

Ventilatory support can be of two types –

i. Non-invasive ventilation given through
   - nasal or face masks
   - negative pressure ventilation (e.g. iron lung)- not practiced now

ii. Invasive ventilation

Mechanical ventilation, either “invasive” or “noninvasive”, is not a therapy but is a form of life support until the cause of underlying acute respiratory failure is reversed with medical therapy.

**Non invasive Positive Pressure Ventilation (NIPPV)**

NIPPV is an option for patients who have respiratory failure and can no longer breathe on their own. It provides ventilatory support to a patient through the upper airways. It enhances...
The breathing process by giving the patient a mixture of air and oxygen from a flow generator through a tightly fitted facial or nasal mask. Also known as just Noninvasive Ventilation (NIV), it assists the patient in taking a full breath and helps to maintain an adequate oxygen supply to the body.

NIPPV is by far the most popular mode of providing noninvasive ventilation with a combination of continuous positive airway pressure (CPAP 4–8 cmH2O) plus pressure support ventilation (PSV 10–15 cmH2O) (Figure 2).

It improves the gas exchange by improving the alveolar ventilation, without causing significant modifications in the alveolar ventilation/perfusion mismatching and gas exchange in the lungs. The application of the combination of CPAP and PSV offers a better outcome than either alone because CPAP counterbalances the intrinsic positive end expiratory pressure.

NIV should be offered to patients with exacerbations when, after optimal medical therapy and oxygenation, hypercapnia (PaCO2 >50 mmHg), respiratory acidosis (pH <7.35) and/or excessive breathlessness persist. All patients considered for mechanical ventilation should have arterial blood gases measured.

- If pH <7.30, NIPPV should be delivered under controlled environments such as intermediate intensive care units (ICUs) and/or high-dependency units. If pH <7.25, NIPPV should be administered in the ICU and intubation should be readily available.
- Indian recommendations insist use of NIPPV even in absence of ABG and/or ICU facilities.
- NIV may also be considered for patients who are slow in weaning from ventilator.
- When put on NIV, there should be a well-defined plan for every patient on course of action on deterioration as well clear indicators of the limits of such deterioration, when the next action is to be taken.

**What are the parameters for judging the success of NPPV?**

NPPV can be called successful if
- Arterial blood gases and pH improve
- Dyspnoea is relieved
- The acute episode resolves without the need of endotracheal intubation
- Mechanical ventilation can be discontinued
- The patient improves steadily and can be discharged from the hospital.

It is important to note that the one-year mortality was reported to be lower in patients receiving NIPPV for exacerbations of COPD, compared to both optimal medical therapy alone and conventional mechanical ventilation.

**Mechanical Ventilation (MV)**

Mechanical ventilation is a mode of assisted or controlled ventilation through intubation using mechanical devices that cycle automatically to generate airway pressure.

**Indications of mechanical ventilation**

Intubation should be considered in patients with the following:
- NPPV failure: worsening of arterial blood gases and or pH in 1–2 h; lack of improvement in arterial blood gases and or pH after 4 h.
- Severe acidosis (pH <7.25) and hypercapnia (Pa, CO2 >8 kPa (60 mmHg)).
- Life-threatening hypoxaemia.
- Tachypnoea >35 breaths/min.
- Impaired mental status.

**Indication for discharge**

Discharge should be planned when:
- There is significant improvement in the clinical status of the patient.
- Significant improvement of symptoms like breathlessness, cough, remission or steady improvement in infection symptoms (reduction in sputum volume, non-pyrexial status, non-purulence of sputum as confirmed by culture)
- Ability to maintain blood gas at acceptable range.
- Ability and inclination to perform day to day activities, even with some degree of assistance.

**Planning of Discharge:**

- Spirometry measurement is a must for all patients before discharge (though daily monitoring of PEF or FEV1 does not help during the exacerbation).
- Patients should be re-established on their optimal maintenance bronchodilators.
- Patients and/or home carers must be given the necessary information so that they fully understand the correct usage of medication including oxygen (if applicable).
- It is useful to hand over a written plan on daily self care, medication management, emergency action plan during exacerbation to all patients/carers before discharge, with adequate explanation.
- Vaccination: Influenza (annual) and Pneumococcal (once in a lifetime).
References


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