Management of acute exacerbations of chronic obstructive pulmonary disease: the first 24 hours

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The common medical emergency of an acute exacerbation of chronic obstructive pulmonary disease (AECOPD) is part of the natural history of the disease. Although sometimes viewed with a degree of complacency, frequent exacerbations are often an indication that a patient is entering the final phase of the illness. Milder exacerbations are often unreported or managed in primary care, but AECOPDs sufficiently severe to require admission to hospital carry a poor prognosis with a third of patients readmitted and 15% mortality within three months. Those who exacerbate frequently have an increased rate of decline in FEV₁ and a worse quality of life than those who exacerbate infrequently. Most exacerbations are associated with infection, either a respiratory virus or the acquisition of a new strain of a colonising bacterium.

Some patients are so symptomatic even when stable that only minor deteriorations necessitate an assessment in secondary care. Others may have milder disease but their frequent presentation with equivocal worsening of chronic symptoms may be due to social factors such as limited access to primary care, anxiety or social isolation. In both cases rapid triage and treatment stabilise the patient's condition and allow return home within 24 hours with a package of care (nursing support and medication): the so-called 'hospital at home' or assisted discharge.

This article discusses the assessment and management of more severely affected patients attending the medical admissions unit with AECOPD.

Assessment

Breathlessness usually dominates the clinical presentation, but cough, change in

sputum volume or colour, wheeze, chest discomfort, peripheral oedema or somnolence may be the main complaint. A previous diagnosis of bronchitis, chest infection or asthma may have been made instead of COPD. Beware of making a diagnosis of COPD if there is a smoking history of less than 20 pack-years. It is useful to obtain a history from the patient or relatives early on about exercise tolerance and activities of daily living. This may be crucial information if the patient deteriorates and respiratory support is being considered.

The commonly observed signs are breathlessness at rest, pursed lipped breathing, quiet breath sounds and wheezing. Cyanosis, unless profound, may be difficult to detect; measurement of oxygen saturation is far more reliable. Warm hands and slight twitchiness may precede the more obvious flap of hypercapnia. The use of accessory muscles and a respiratory rate above 25 per min suggest severity.

There are undoubtedly valid physiological reasons to question the value of peak flow in the diagnosis of COPD but the measurement is useful as a comparison with the patient's normal value. It might help exclude worsening airflow obstruction as a cause of the current symptoms or even, in retrospect, identify a hitherto unappreciated degree of asthma.

Investigations to aid diagnosis and determine the patient's condition are listed in Table 1.

Drug treatment

Inhaled bronchodilators

High-dose inhaled bronchodilator is the cornerstone of management. In severe disease nebulised treatment is preferred. Beta-2 adrenergic agonists (salbutamol or terbutaline) and anticholinergic agents (ipratropium) are equally effective. Current

practice is to give both drugs 4–6 hourly, although evidence suggests no additive benefit from use of the combination. The pharmacology of the muscarinic receptor suggests the once-daily selective muscarinic blocker tiotropium should be stopped during ipratropium therapy. Frequent dosing (say 2–4 hourly) of short-acting beta-2 agonists is now common practice in asthma and is recommended for poorly responsive AECOPD. There is no role for parenteral beta-agonists. In the presence of type 2 respiratory failure, nebulisers should be air driven and controlled oxygen given by nasal cannulae simultaneously.

Aminophylline

In addition to acting as a bronchodilator, aminophylline might have additional benefits on diaphragm contractility and ventilatory drive. Side effects are common and there is a dearth of evidence to support its use as additional treatment to maximum inhaled bronchodilator therapy in non-acidotic exacerbation.¹ There may be a role in selected acidotic cases, perhaps in conjunction with respiratory support. If given, serum levels need close monitoring.

Table 1. Investigations useful in the management of an acute exacerbation of chronic obstructive pulmonary disease.

Investigation	Information obtained
Peak flow	See text
Chest X-ray	Excludes pneumonia, pneumothorax, pulmonary oedema, lung cancer
ECG	Excludes MI May show atrial arrhythmias or signs of right ventricular strain/hypertrophy
Pulse oximetry	If >94% on air, ABG not indicated If <94% on O ₂ , ABG needed to confirm acid-base status and ensure adequate ventilation

ABG = arterial blood gas; MI = myocardial infarction.

Antibiotics

Bacterial infection plays only a minor part in many exacerbations of COPD and some exacerbations are clearly non-infective. Fortunately sputum purulence remains a good predictor of likelihood of response to antibiotics. The most common organism isolated from sputum during AECOPD is *Haemophilus influenzae*. Treatment should take into account local patterns of resistance and any prior treatment in primary care. Other organisms that commonly cause exacerbations are *Moraxella catarrhalis*, *Streptococcus pneumoniae* and *Pseudomonas aeruginosa*. Unless there is obvious consolidation on the chest X-ray, there is no indication for treatment with the beta-lactam and macrolide combination used in severe community acquired pneumonia. Amoxicillin, doxycycline and clarithromycin are all suitable first-line choices. Failure of purulent sputum to resolve with treatment should prompt sputum culture to exclude resistant *H. influenzae* or infection with *P. aeruginosa*, both of which merit a change in antibiotic.

Steroids

Unlike acute asthma where eosinophilic airway inflammation predominates, the response to systemic steroids in AECOPD is less impressive. Nevertheless oral prednisolone 30–40 mg reduces the risk of treatment failure and improves lung function more quickly than bronchodilators alone. Parenteral hydrocortisone is required only if absorption of oral medication is likely to be impaired (eg vomiting, impaired consciousness). The combination of an inhaled steroid and a long-acting beta-agonist (fluticasone/salmeterol, budesonide/formoterol) reduces frequency of exacerbations and should be prescribed prior to discharge.

Oxygen therapy

Perhaps the most difficult aspect of managing AECOPD is the appropriate use of oxygen. Health professionals have to balance the necessity of

relieving dangerous hypoxaemia with the risks of worsening hypercapnia and respiratory acidosis. There is little doubt that over-oxygenation occurs, as judged by the observation that patients with AECOPD whose oxygen levels are above 10 kPa are far more likely to be acidotic and require intensive or high dependency care.²

Observational studies suggest that keeping saturations above 82% ($pO_2 > 6.7$ kPa) may be acceptable and safe. The recently published British Thoracic Society guideline for emergency oxygen use in adult patients emphasises that target oxygen saturations in COPD patients should be much lower than is traditionally thought necessary (ie 88–92%), and that initial treatment should be 28% oxygen at 4 l/min using a fixed flow device (Venturi mask).³ If the patient has previously needed non-invasive ventilation (NIV) and saturations are over 92%, a 24% mask should be used. If the patient has a normal pCO_2 , saturations above 94% can be a target. However, rechecking gases will be necessary, clearly documenting the concentration of inspired oxygen given at the time of the sample.

If pH is normal or the bicarbonate raised in the presence of hypercapnia, respiratory failure is probably chronic. The guideline emphasises the danger of rebound hypoxaemia (when a well meaning physician removes all oxygen treatment from an over-oxygenated patient). Useful algorithms on managing oxygen therapy in AECOPD are also

included and should be available in all acute admission units.

Respiratory support

Patients who fail to improve on the above treatments are at risk of worsening acidosis and death. Early use of NIV improves gas exchange, reverses acidosis, relieves dyspnoea and reduces both the need for intubation and the mortality rate. It should be considered in a patient with signs of worsening respiratory distress (eg respiratory rate > 30 /min) and respiratory acidosis ($pH < 7.35$, $pCO_2 > 6$ kPa). Treatment is now frequently initiated in the acute admission ward. The widespread availability of simple protocols and user-friendly ventilators means that this treatment is within the capacity of the acute physician.

Summary

Successful outcome is more likely with early treatment and lesser degrees of acidosis. While aiming for maximum treatment for the first 24 hours, some patients improve so rapidly that they can discontinue after a shorter time. Most patients need a full face mask and oxygen, and nebulised bronchodilators can be incorporated. If radiological consolidation, excessive secretions and/or confusion are present, the chance of failure is increased but is not an absolute contraindication. The presence of a pneumothorax necessitates intercostal drainage. A useful summary statement has recently been published.⁴

Key Points

Stratification allows rapid triage of severe exacerbations and early discharge of milder ones

High-dose inhaled bronchodilators, controlled oxygen and early institution of non-invasive ventilation are critical. Target saturations of 88–92% are appropriate for most patients

Parenteral bronchodilators are largely ineffective and should only be prescribed in exceptional circumstances

KEY WORDS: controlled oxygen, inhaled bronchodilators, non-invasive ventilation, stratification

Patients who are obtunded and peri-arrest require immediate intubation and mechanical ventilation. There is some evidence that intensivists are reluctant to accept COPD exacerbations to the intensive care unit because of the perceived low survival rates or concerns about weaning delays after intubation. In fact, the prognosis may be better than in many other patients with multi-organ failure. Patients can often be quickly weaned on to NIV and returned to the ward after an initial period of invasive support and secretion management.

Initial assessment and the past history should identify those markedly disabled

patients with recurrent admissions who are likely to be entering the terminal stages of their illness in whom intubation is inappropriate. Here, NIV may be the ceiling of treatment, providing useful symptom palliation while waiting for treatment to improve any reversible factors.

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