Primary Care Respiratory Academy 2021

Clinical Webinars 3 & 4 Q&A

DIAGNOSIS

Ren Lawlor
Nurse

Steve Holmes
GP

Dominika Froehlich-Jeziorek
Pharmacist

Does previous history matter in the diagnosis of COPD?

The short answer is yes. Taking a thorough patient history supports correct diagnosis and helps to narrow down the list of differentials.

Could you explain a bit more about the PEF <75% on the 2-week peak expiratory flow rate? Would variability/reversibility peak expiratory flow rate be helpful in differentiating between COPD and asthma?

A predicted PEFR of <75% suggests a degree of airflow obstruction and a serial measurement over 2 weeks that does not vary but also remains low (despite the use of salbutamol for symptom relief) would suggest fixed airflow obstruction. This, combined with a supporting clinical history, would point towards COPD. Confirmatory spirometry should be carried out later when safe and readily available.

What do you mean by empirical trial of dual bronchodilator?

Patients who do not have variation in serial peak flow (carried out over 2 weeks), despite the use of salbutamol for symptom relief can have an empirical trial of dual bronchodilator therapy. In short, this involves the patient trying the dual therapy for a set period of time, and, if their symptoms improve during this time, the treatment has been successful.

Do we need to give the patient a bronchodilator before doing the spirometry test for diagnosis?

Yes, when spirometry is undertaken for diagnostic purposes, post-bronchodilator values are required to demonstrate irreversible obstruction.

Further information can be accessed through the BTS website: https://www.brit-thoracic.org.uk/quality-improvement/clinical-resources/copd-spirometry/spirometry/ and on the ARTP site https://www.artp.org.uk/en/spirometry

Is there a minimum age I should consider alpha-1 antitrypsin deficiency as a diagnosis?

It would be sensible according the NICE guidelines to consider alpha-1 antitrypsin deficiency if early onset (<40 years old), minimal smoking history or family history. Having said that, GOLD guidelines and the World Health Organisation both suggest checking in all patients.

Can someone get COPD from chronic passive exposure to smoking even if they themselves have never smoked?

The majority of people with COPD have a smoking history (estimated at around 90%). Chronic exposure to biomass fuels, a family history of alpha-1 antitrypsin deficiency and some occupational exposures can leave people at risk. Though it is thought that exposure to passive smoke potentially will cause problems, it is worth reflecting that there must be other factors at play. Many people who smoke throughout their lives do not develop COPD – around 20% of those with prolonged tobacco smoking develop COPD.

Primary Care Respiratory Academy 2021

Clinical Webinars 3 & 4 Q&A

TREATMENT & MANAGEMENT

Ren Lawlor
Nurse

Steve Holmes
GP

Dominika Froehlich-Jeziorek
Pharmacist

With patients that have exacerbations needing frequent steroids, after 3-4 courses they may need bone protection. How long do we keep patients on bone protection treatment?

It would be first sensible to assess the extent of osteoporosis then provide an appropriate level of osteoporosis treatment and determine when further monitoring is required according to the national guidelines.

More information can be found in a consensus guideline produced in 2020: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7311204/ and in the Clinical Knowledge Summaries and Scottish Intercollegiate Guideline Network Osteoporosis guidelines: https://www.sign.ac.uk/media/1741/sign142.pdf

Is osteoporosis likely to be related to steroid usage?

Steroid use is strongly linked to osteoporosis as is smoking exposure and reduced activity (all of which can be common in patients with COPD). It is certainly worth considering osteoporosis in all people with COPD (depending on their smoking history and activity) but those with high dose inhaled corticosteroids or several courses of oral corticosteroids.

Would you suggest a DEXA in all patients diagnosed with COPD?

It would be sensible to evaluate the risk in patients using a FRAX score and careful clinical assessment rather than testing all at diagnosis. It would also be appropriate to review every patient annually relating to their risk (but again, not an annual DEXA scan) as courses of steroids, continued smoking, and reduced activity can increase risk. Similarly, people with COPD should be assessed regularly for their cardiovascular risk.

In the absence or delay of spirometry, could we treat as suspected COPD with combined INH for airway protection?

If it is not feasible to undertake spirometry for a period of time (as happened at times during the pandemic) it is firstly important to have a system to ensure that the patient can be called back to make a robust diagnosis. Many clinicians have made pragmatic decisions to use inhalers (considering whether the diagnosis is likely to be COPD and whether there may be an asthma element or raised eosinophils) which would guide treatment in keeping with NICE guidelines.

What is the difference or role of patients in using MDI and PDI? I noted sometimes these two are mixed – what advice can we give patients when choosing INH type?

A lovely complex question that could take all day to answer. It is essential to ensure that the patient can use any inhaler prescribed and that the device and drug is appropriate to their needs. You may want to consider the green impact of inhalers in addition to patient factors – and the cost implications of prescribing (to our patient and to our health care system). Ideally we would want patients to have the same inhaler device for their needs – but this is often not possible. Essential though is that the patient knows *why* they are prescribed the drug and clearly how to use it and that they buy into this. Equally, when they are reviewed, the appropriateness of the drug and the inhaler device should be monitored and we check they can use the device.

Primary Care Respiratory Academy 2021

Clinical Webinars 3 & 4 Q&A

TREATMENT & MANAGEMENT (continued)

Ren Lawlor
Nurse

Steve Holmes
GP

Dominika Froehlich-Jeziorek
Pharmacist

Can you explain the mechanism of beta blockers that makes it not suitable for people with asthma?

It would be sensible to refer to basic pharmacology books – an agonist like a short-acting beta agonist (SABA) (like salbutamol) triggers the action of a beta receptor whereas a beta blocker (like bisoprolol) blocks the receptor – this means they act against each other. If a person who needs a beta agonist gets the opposite (beta blocker) when they are poorly will have worse outcomes and can indeed die. Use of beta blockers in asthma is associated with rare but important asthma deaths.

However in people with fixed obstructive airway disease (COPD), the problem is not reversible and use of beta blockers is much safer.

In simple terms, a person with asthma given a beta blocker may die; in someone with COPD with established heart disease, it can improve outcomes.

Is optional treatment available avoiding overuse of steroids?

It is possible to avoid overuse of steroids in people with COPD by:

a) Not allowing the patient to get these easily as a repeat prescription unless indicated

b) Ensuring that if a steroid is prescribed it is used for a genuine exacerbation

c) Reviewing after every exacerbation to check inhaler technique, best medication, that the patient is not smoking (unfortunately, 35% of the UK population with COPD who are admitted still smoke) and that the person used the medication appropriately

What oxygen saturation would you send a patient to the hospital?

With asthma, if a patient who is normally well has a saturation below 92% that would cause concern and should make us consider admission. This would be true of all people but should always be considered with wider use of pulse oximetry. Do other parameters fit in? Symptoms, pulse, respiratory rate, blood pressure, general well being?

In people with COPD, again if they are not clearly established as having a level of oxygen that is low (and I hope they are already evaluated and on long term oxygen if appropriate), then I would have a low threshold to admit these people.

Should oxygen therapy be reviewed in patients in whom we find the inhalers were not optimised in the first place? Or is it unlikely in such cases with better use of inhalers there may not be a need for oxygen?

Yes – it is surprising how often people can't use their inhalers and get better outcomes when using them properly. I have to be honest but the majority of people on oxygen, even if managed really well, can have their oxygen discontinued – however they can function much better and have better outcomes if they use their inhaler properly, are active and stop smoking!

Any practical tips on how to tackle overuse of rescue packs?

Every rescue pack that is used should be reviewed (ideally face-to-face but otherwise by telephone) after recovery to check for appropriate use and that all other risk factors have been identified. Is this only due to COPD? Have I managed other problems? Did my patient use the treatment properly at the right time?

Primary Care Respiratory Academy 2021

Clinical Webinars 3 & 4 Q&A

REFERENCES & RESOURCES

NICE guidance COPD in over 16s: https://www.nice.org.uk/guidance/ng115

NICE decision aid inhalers for asthma: https://www.nice.org.uk/guidance/ng80/resources/inhalers-for-asthma-patient-decision-aid-pdf-6727144573

BTS website: https://www.brit-thoracic.org.uk/quality-improvement/clinical-resources/copd-spirometry/spirometry/

ARTP website: https://www.artp.org.uk/en/spirometry

Scottish Intercollegiate Guideline Network Osteoporosis guidelines: https://www.sign.ac.uk/media/1741/sign142.pdf

NRAD 2014: https://www.asthma.org.uk/globalassets/campaigns/nrad-full-report.pdf

Ren Lawlor
Nurse

Steve Holmes
GP

Dominika Froehlich-Jeziorek
Pharmacist

Bhatt SP, Wells JM, Kinney GL et al. β-Blockers are associated with a reduction in COPD exacerbations. *Thorax.* 2016;71(1):8-14

Gupta A, Jayes LR, Holmes S et al. Management of Fracture Risk in Patients with Chronic Obstructive Pulmonary Disease (COPD): Building a UK Consensus Through Healthcare Professional and Patient Engagement. *Int J Chron Obstruct Pulmon Dis.* 2020;15:1377-1390.

Hurst JR, Quint JK, Stone RA, Silove Y, Youde J, Roberts CM. National clinical audit for hospitalised exacerbations of COPD. *ERJ Open Res.* 2020;6:00208-2020

Ling Y, Saleem W, Shee CD. Concomitant use of beta-blockers and beta2-agonists. *Eur Respir J.* 2008;31(4):905-906

Lipworth B, Wedzicha J, Devereux G, Vestbo J, Dransfield MT. Beta-blockers in COPD: time for reappraisal. *Eur Respir J.* 2016;48(3):880-888.

Rutten FH, Groenwold RHH. β blockers for adults with chronic obstructive pulmonary disease. *BMJ* 2013;347:f7050.

Rutten F, Zuithoff N, Hak E, Grobbee D, Hoes A. Beta-blockers may reduce mortality and risk of exacerbations in patients with chronic obstructive pulmonary disease. *Arch Intern Med.* 2010;170(10):880 - 887.

Short P, Lipworth S, Elder D, Schembri S, Lipworth B. Effect of beta blockers in treatment of chronic obstructive pulmonary disease: a retrospective cohort study. *BMJ* 2011;342:d2549.

Singh D, Agusti A, Anzueto A et al. Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Lung Disease: the GOLD science committee report 2019. *Eur Respir J.* 2019 May 18;53(5):1900164.

Smith GB, Redfern OC, Pimentel MA et al. The national early warning score 2 (NEWS2). *Clin Med (Lond).* 2019;19(3):260.