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W3_L4_Obstructive Airway Diseases – Approach

Hello friends, my name is Dr. Durga Krishnan from Chettinad Hospital and Research Institute, Chennai. Today, I am here to discuss you about the obstructive airway diseases. I hope before coming to this lecture, you would have the basic idea about the anatomy and physiology of the respiratory system. So, we will move on to this topic. In this obstructive airway disease, today I am going to cover these four topics of asthma, chronic obstructive pulmonary disease, bronchiectasis and bronchiolitis.

So, first is asthma that is bronchial asthma. So, what is bronchial asthma? It is a disease which is characterized by episodic airway obstruction and airway hyper-responsiveness, which is accompanied by airway inflammation. And usually, the patient presents in this with a history of shortness of breath or chest tightness or dry cough. We will discuss it later.

So, when we are talking about the bronchial asthma, in the development of bronchial asthma, there are certain exposures or risk factors may be in prenatal or childhood or adult period, which predisposes the patient for development of this disease. So, this is mostly the allergen exposure in those who is already atopic or that is predisposed atopic patient in which repeated allergen exposure can lead to development of this disease or it could be occupational exposure in which the other it is immune or irritant things or it is tobacco or air pollution, certain infections like viral or mycoplasma infections, obesity itself, diet for which you are following, you could be allergic to that or then fungal infections or high intensity exercises in allied people. So, these are the risk factors or exposures in our life, which can predispose us for the development of bronchial asthma. In this slide, the same thing like the person who is atopic, atopic is those who have the allergy and then if they have the genetic susceptibility or risk genes and if they are being exposed to the high risk what I have described in the previous in their prenatal or childhood or adult groups, this leads to development of symptoms due to bronchoconstriction which is further because of the airway hyper-responsiveness along with inflammation and structural change. This sometimes gets treated on its own or we treat and then it subsides, but then this triggers when it is there repeatedly, then it can lead again it can lead to increase in the airway hyper-responsiveness, inflammation and structural changes and recurrent exacerbation.

So, this is interrelated with each other. So, here you can see this in this graph, this is this and these are being interrelated with each other. So, in bronchial asthma, the most important thing which we have to understand is airway hyper-responsiveness. So, what is this airway hyper-responsiveness? This airway hyper-responsiveness is the hallmark of asthma. It is defined as the narrowing of the airways on exposure to allergens which would not have caused the narrowing in a non-affected person or an excess response to an inhaled contents which would not have produced the same response in the non-affected persons.

This usually occurs by the stimulation of the smooth muscles. Because the smooth muscles affection can be direct or indirect. When it is direct, it is direct release of the histamine and methacholine which causes bronchoconstriction or indirectly, indirectly certain triggers stimulate the inflammatory cells and then there could be release of these allergens and then that leads to bronchoconstriction, mucus secretion and leads to the development of this asthma. So, this airway smooth muscles can contribute to the asthma in three ways. First, it is just a hyper-responsiveness to the stimuli.

In the second, because of this hyper-responsiveness, as a consequence of this hyper-responsiveness, there is hypertrophy and hyperplasia which can lead to air wall thickening. And lastly, this airway smooth muscles can produce chemokines and cytokines which can cause increased mucus production and this bronchial wall thickening and leads to the disease. The factors which are responsible, which are related, first is the, in the pathogenesis is the thickening of the sub epithelium basement membrane. And this thickening is because of the repair type collagen deposition. So, there is a collagen and matrix deposition which leads to stiffening of the airways.

This leads to stiffening of the airways and it is difficult in the relaxations and then mucus production contributes to the chronic airway inflammation. Second is the airway epithelium itself. Whenever there is a separation of the airway epithelium columnar cells from the basal cells, there is a formation of trophic unit and this trophic unit secretes lot of growth factors which in turn cause bronchospasm and contribute to the, and mucus secretion and contribute to the chronic airway inflammation. Vascular proliferation, so that is what is called angiogenesis. These angiogenesis postcapillary venue they secrete lot of immune mediators which can lead to bronchospasm and contribute to the airway obstruction.

Then airway edema, when this airway edema occurs, this airway edema occurs mainly when the patient is having an acute episode. So, whenever the patient is having an acute episode, this produces airway edema which also contribute to the chronic airway inflammation. Likewise, the globular cells, metaplasia and mucus hypersecretion, what they do is this forms a mucus plus in the medium sized bronchioles and airways. Then it goes down to the smaller airways and cause the obstruction of the smaller airways along

with secretions and contribute to the chronic inflammation. Neuronal proliferation, so this neurotrophins can lead to neuronal proliferation.

This neuronal proliferation supplies to these smooth muscles which can lead to bronchospasm and mucus secretions. Ultimately, all these things are doing nothing but increasing the chronic airway inflammation which is the main thing in the development of bronchial asthma. In this inflammation, there are two types, that is type 2 and non-type 2 inflammation. So, what is this type 2 inflammation and this non-type 2 inflammation? See this type 2 inflammation is innate and adaptive, that is it is inside with the immune system, causes changes and causes the bronchoconstriction. Why the name is given as type 2? Because it belongs to the type 2, because it is associated with the type 2 subset of the CD4 helper T cell which in turn produces interleukin 4, 5 and 13.

And what is this non-type 2? Non-type 2 is more commonly associated with the severe type of asthma that does not respond to the common anti-inflammatory therapy such as corticosteroids. It may occur in separation or it may occur along with type 2 also. Sometimes in some cases it may be associated with chronic infections occasionally with atypical pathogens like neoplasm. So, while going towards the diagnosis, so history and clinical features are one of the most important things which we have to give importance while diagnosing a case of bronchial asthma. The first most important thing is in the history you have to find out whether the patient is having any allergic history.

Allergic history means whether there is a seasonal variation or there are changes in the environment like humidifying or exposure to air conditions. All these things it is triggering the symptoms. So, what are the symptoms which is being triggered? Usually, it presents as a V's or dry curve or chest tightness along with shortness of breath. So, these are the symptoms. If these things are triggering these, also you have to take the history of the household history like exposure to pets or moths or occupational exposures or certain hobbies like swimming, all these things if these are increasing the episodes.

If in the history you have the patient gives you the history of the use of inhaled corticosteroids or nebulization and along with these aggravating factors, these are the important history which you have to take. See all these bronchial asthma patients are usually a topic and half of them gives you history of allergic rhinitis. So how will you find out whether the patient had allergic rhinitis or not in the past though? So the features like pale nasal mucosa’s or if there is a presence of nasal polyps during examination, this says the patient is having allergic rhinitis. Usually they have these patients, the most important features on examination, on auscultation the wheeze. It could be inspiratory and expiratory, but it is mainly expiratory.

And whenever the patient has an acute episode of asthma, they usually have this acute, mainly inspiratory, sorry, expiratory V's. And then when it goes on increasing, sometimes

the patients, this expiratory V's goes on increasing, the attack goes on increasing, then usually the patient becomes silent. There are no breath sounds which is being audible. It is a severe form of bronchoconstriction that says the patient is having a silent chest. And that is a severe form of asthmatic attack.

Pulmonary function test, this pulmonary function test is one of the most important parameters in diagnosing the bronchial asthma. So, what happens is this in this asthma is actually there is reduction in the airway lumen, which produces increased resistance to airflow, which can be detected by expiratory airflow during forced expiratory manures. So, one of the things which we are going to see in pulmonary function test is peak expiratory flow rate, forced expiratory volume and the ratio of forced expiratory volume with forced vital capacity, which is all lower than its normal limit. You can also go with the reversibility testing. You can also go with the reversibility testing.

So, what is this reversibility testing? You will go give some bronchodilators and see whether the patient is having reversal of these volumes or not. So it is defined as reduction of greater than equal to 12% increase in FEV1 and an absolute increase of greater than 200 ml at least 15 minutes after the administration of beta 2 agonist or after several weeks of corticosteroids therapy. If it is there, that is the reversibility testing and it is the diagnostic for bronchial asthma. Now in cases where the pulmonary function test is not able to diagnose, then you have to go with the other confirmatory testing that is methacoline. Methacoline, a cholinergic agonist inhaled in increasing concentration is most commonly used.

A provocative dose producing 20% drop in FEV1 is calculated with a value less than equal to 400 microgram that is indicative of the airway reactivity. Now the other test we do, the routine test of chest x-ray, ECG to rule out whether there is any cardiac involvement and other blood investigations and sputum analysis. Other than that other important adjuvant test is these three that is absolute eosinophil count, immunoglobulin E levels and fraction of exhaled nitric oxide. See this nitric oxide, fraction of exhaled nitric oxide can be done to assess the eosinophil inflammation related because of the eosinophils in the airways. If you have done the previous eosinophil counts and if you go with the inhaled corticosteroid therapy, then there can be reduction in this and you can see that the patient is adherent to the treatment or not with this fraction of exhaled nitric oxide test.

While considering treatment for bronchial asthma, we use two tracks that is track 1 and track 2. First of course we have to assess, we have to confirm the diagnosis and symptoms, how it is occurring and whether there is any modified respect around that, whether there are any comorbidities or not, whether patient is using the correct technique of inhalers and adherent to the treatment or not. So, because of this we have divided it into track 1 and track 2. See track 1 we use for those patients who are poorly adherent to the treatment.

We use, low dose formoterol we use as a controller and preferred reliever. So, we have divided the treatment into step 1, step 2, step 3, step 4 and step 5 of course. Here we consider for step 1 and step 2 we use this controller and preferred reliever as and when needed, as and when needed. In step 2 when the symptoms are more then you have to use low dose maintenance therapy with this reliever. And in step 4 when the symptoms are much more along with low lung function then you have to use medium dose maintenance inhaler therapy with this inhaler that is formoterol.

And still, you are getting episodes then you have to use this along with this you have to use llama and then you have to assess the patient s phenotype and consider for the maintenance therapy. Along with this you can also use immunomodulators. In the next I will show you what the immunomodulators which we are or biologics which we are using in the bronchial asthma. Then in track 2 is used for patients who are adherent to the treatment. In this the controller and alternative reliever is the SABHA that is short acting beta agonist.

So, in this we have divide in this also we have divided into step 1, step 2, step 3 and step 4, step 5. In step 1 the patient is less than that is twice a month you use as and when needed you have to use inhaler corticosteroids along with that SABHA or you are using SABHA along with it you are also using at that time you are also using the inhaled corticosteroids. In step 2 when it is more than 4 or 5 days a week then you have to use low dose maintenance inhaled corticosteroids and as and when in it you can also use this SABHA. Then in step 3 and step 4 you have to use along with this low dose maintenance inhaled corticosteroids you have to use long-acting beta agonist. In the step 4 you have using medium dosed inhaled corticosteroid therapy along with long acting and as in the previous first step in track 1 the same we are going to repeat it here also we are going to use the muscarinic antagonist long-acting muscarinic antagonist for if the symptoms are not being controlled.

So, these are the biologics in asthma that is immunomodulators which acts to binds to different immunoglobulin interleukins for its action that is omazimab or mepolizumab or raslizumab, benralizumab or dupilizumab. Root of administration is subcutaneous or IV. So, this is this was about the bronchial asthma now comes to the other is the bronchiectasis. So, what is bronchiectasis? bronchiectasis can be defined as a chronic irreversible destruction and distortion of the airways mostly because of the inflammation to the mucosal and elastic component of the airways, airways wall or bronchial wall. So, bronchiectasis refers to irreversible airway dilatation that involves the lung in either focal or a diffuse manner.

This is focal or diffuse manner and that classically categorized in cylindrical tubular and most commonly tubular or cylindrical, tubular is the most common type and then it could be varicose or cystic in nature. Okay. So, cylindrical is like this varicose, varicose is a torsity and then there can be formation of small cyst. Okay. So, when you say that it is

focal or diffuse, this bronchiectasis can occur because of infectious or non-infectious process.

When it is focal, it is localized to some part of the lung and it is usually because of the obstruction. This obstruction can be intrinsic in the airway or extrinsic. When it is extrinsic, it could be because of the lymph node enlargement or some parenchymal tumor which is compressing the airways or inside the airways also there can be some tumor or there can be some mucus plugs or foreign bodies which is obstructing this airway and leading to this localized bronchiectasis. Diffused bronchiectasis involving most of the part of the lungs and it is usually because of the systemic or infectious process. So, when we are dividing it, we can say that the bronchiectasis can occur in the upper lobe or middle or central lobe or in the lower lobe.

When it is occurring in the upper lobe, it could be because of cystic fibrosis or tuberculosis in the upper lobe, it is cystic fibrosis or tuberculosis. In the central, it could be because of cystic fibrosis or because of the aspergillosis or because of congenital trachea-bronchomegaly. In the lower part, it is because of childhood infection, repeated aspirations or could be because of immunodeficiency. The most common clinical presentation in bronchiectasis is productive cough with copious amount of sputum production which is foul smelling, thick and tenacious. Physical findings frequently include crackles and wheeze on lung auscultation and some patient bronchiectasis exhibit digital clubbing.

All the chest radiographs lack sensitivity, but sometimes we can get a tram track appearance. The tram track appearance is because of the dilated airways which is indicating dilated airway and which is consistent with the bronchiectasis. Chest CT is more specific and confirmatory as far as this bronchiectasis is concerned. So, the CT findings include airway dilatation which gives a tram track appearance or signet ring cell, signet ring sign or there can be a lack or loss of bronchial tapering also. Along with this bronchial wall thickening in dilated airway, there can be inspecific secretions that is giving appearance of tree invert or cyst emanating from this bronchial wall, especially pronounced in patient who have cystic bronchiectasis.

So, here you can see the form in the cystic bronchiectasis, these are formation of the cyst. Here in varicose you can see the tortuosity and here in the cylindrical you can see the cylindrical pattern of bronchiectasis. Signet ring appearance, so this arrow is showing you the signet ring appearance, this is nothing but the dilated airways adjacent to the pulmonary artery which is giving the appearance of a signet ring. This tree invert appearance it can occurs in all the infectious diseases of the airways which is because of this mucus plugging and thickening which gives a tree invert appearance. So, when we are moving to the management of bronchiectasis the most important, usually you get exacerbation in bronchiectasis is because of the infections related.

So, you have to give appropriate antibiotics in the patients and then the antibiotics has to be continued for 7 to 10 days, sometimes you may might need to give it for 2 weeks that is 14 days. Other therapies are bronchodilator therapies along with mucolytic therapy to which helps to bring out the sputum and also it should be assisted with the chest physiotherapy. Sometimes this repeated infection can cause erosion of the superficial mucosal vessels which can lead to severe hemoptysis and when the patient is going for severe hemoptysis definitely you have to admit the patient in the emergency go have to ventilate the patient, try to stop the bleeding and also save the other part of the lung. When you are not able to manage with this then you of course have to go with the bronchial artery embolization. That was the bronchiectasis then we are moving out over the next topic that is chronic obstructive pulmonary disease.

So, what is this chronic obstructive pulmonary disease? It is defined as a state characterized by persistent respiratory symptoms and airflow obstruction. It is comprised of 3 things that is emphysema, chronic bronchitis and small airway involvement. So, this what is this emphysema? Emphysema is nothing there is destruction of the alveoli along with the dilatation of the airways. What are bronchi chronic bronchitis? Chronic bronchitis is chronic cough along with the phlegm production, copious amount of phlegm production. And this is small airways usually there is reduction in the number of small airways along with the narrowing of these small airways.

These are the 3 things which comprises chronic obstructive pulmonary disease in this. So, I have just used the term previously only we used to call it blue blotters and pink puffers. These blue blotters nothing but the chronic bronchitis. Why they are being called as blue blotters is usually there is a hypoxemia and because of this hypoxemia there is cyanosis and blue discoloration that is why they will call as blue blotters. The most important symptoms with which the chronic and the patient with chronic bronchitis presents is chronic productive cough which is mostly purulent in nature.

There can be associated hemoptysis in case when they have infection. Dyspnea is usually mild the patient has cyanosis due to hypoxemia giving it a bruise discoloration. Peripheral edema when the patient has developed signs of right-handed heart failure because of pulmonary artery hypertension. And in the findings, you get scraggles, wheeze, prolonged expiration and usually the patients with chronic bronchitis are obese. And as a complication of this chronic bronchitis, you can have secondary polycythemia where are due to hypoxemia and pulmonary hypertension due to reactive vasoconstriction because of hypoxemia.

Then emphysema, emphysema are also known as pink puffers previously. Why they are known as pink puffers because they have a gaps in between breathing. So, they go for the repeated frequent short breaths and because of this doing before these frequent short breaths they develop pinkish discoloration of the cheeks and skin of the face which gives

a pink color then that is why they are known they were known called as pink puffers. Usually, they present with dyspnea with only minimal cough. Their minute volume is increased and then there is a pink skin with parsley breathing, cachexia they have they usually use accessory muscles for their respiration or breathing and then there is hyperinflation because of the increase in air space there is hyperinflation in barrel chest, there is decreased breath sounds and patient usually are tachyapneic that is increase in the respiratory rate.

The COPD is related very much in the history. One of the most important things is that you have to rule out tobacco history in these patients and smoking history in this patient. And then usually these people go for the type 2 kind of respiratory failure which helps us in diagnosing these diseases. And as a complication you can develop pneumothorax due to bulla formation or rupture of this bulla can lead to pneumothorax as a complication, weight loss because of the increased workload of breathing and then carpalmeneal secondary to chronic pulmonary hypertension. For diagnosing we use pulmonary function tests and gold criteria for severity of air pull obstruction COPD. These are the criteria with which using pulmonary function tests we diagnose the COPD.

It has been divided into stage 1, stage 2, stage 3 and stage 4 that is mild, moderate, severe and very severe. So, the volumes which we use is forced expiratory volume that is V1 and forced and the ratio of forced expiratory volume in 1 second with forced vital capacity. So, this FVV1 by FVZ ratio remains constant that is less than 7. Only this forced expiratory volume you have to predict you get the predicted value. If it is more than 80 percent then it is, so that the patient is having mild that is stage 1.

If it is less than 80 but more than 50 then it is the patient is having moderate that is gold stage 2. When it is greater than equal to 30 percent but less than 50 percent it is severe and it says the patient is having stage 3 and when it is less than 30 percent it says the patient is having a very severe stage 4 COPD. Chest X-ray also assists in diagnosis of COPD mainly emphysema. When you look at the X-ray you can see the most important is the lung hyperinflation increasing in the space and then there is a flattening of the hemidiaphragm.

This diaphragm should be seen like this. So, there is a flattening in this dome hemidiaphragm. These are the X-ray findings along with that when you take lateral X-ray you can see a barrel shape because there is widening of the anterior posterior diameter. And also, you get a saber-sheath trachea sign which refers to when you take a coronal section you can see there will be marked narrowing of the intrathoracic trachea and when in the same when you take a lateral X-ray there is a widening of the trachea which says the patient is having a saber-sheath sign which is also one of the diagnostic sign to say patient is having COPD emphysema. Then to diagnose and categorize into severity we use MMRC that is MMRC is nothing but the modified medical research counseling classification of dyspnea in patient with COPD which ranges from 0 to 4 and CAT that is COPD assessment

test which ranges from 0 to 40. So, if MMRC is greater than equal to 2 it says the patient is having severe disease or if the patient is having CAT of more than equal to 10 that also say the patient is having a severe COPD.

With that likewise symptom that is low symptoms high risk or high symptoms high risk we have divided into A, B, C and D and accordingly we add the bronchodilators for each of the severe severity of each group we add the bronchodilator therapy. So, SABA that is nothing but short acting beta2 beta agonist that is salbutamol. LABA that is nothing long-acting beta2 beta agonist that is formeteral. LAMA long-acting muscarinic antagonist that is styrotropium. Inhaled corticosteroid that is proticosone or budisonide and oral corticosteroid that is most commonly we use is methylprednisone.

And we have come to the last that is bronchiolitis. What is bronchiolitis? It is the inflammation of the smaller airways that is lower respiratory bronchiole that is smaller airways. Usually, it is secondary to the infection with the respiratory syncytial virus also with the rhinovirus, paramyxovirus or human metabolize or influenza virus. Clinically it is usually commonly occur in kids less than 2 years of age often preceded by 1 or 2 day of URI prodrome. Severe fever, cough and respiratory distress like techiepnea, retraction, wheezing all can be present. As a complication there can be dehydration, apnea in premature or less than 2 months old and then aspiration pneumonitis or respiratory failures.

Non severe bronchiolitis you have to anticipate to guide the patient and then do nasal suctioning and hydration. Severe bronchiolitis that you can give trial of inhaled bronchodilators, heated humidifier, high flow nasal oxygen therapy then continuous positive airway pressures or endotracheal intubation if needed. So, this completes our 4 topics. In the next 3 slides I am just going to give certain scenarios to find out what could be the cause. So, here it is a 26-year-old female who is a IT worker, works in an air conditioned office, comes to the OPD with the complaints of cough and rhinitis for 3 days and shortness of breath since today morning.

There is no history of fever. The patient is having tachypnea, tachycardia with saturation of slightly lower that is 94% at room air. And in the examination, you are getting wheeze all over the lung field. So, what do you think? So, in this as you see the allergic that this patient is having rhinitis and this is being exposure history to the air-conditioned office that is one thing and then the patient is having an increased respiratory rate and also there is a predominant wheeze in all parts. So, this says that the patient is probably having bronchial asthma exacerbation or it could be allergic asthma exacerbation. What will be the route of administration of drugs? See, once you are getting a patient you can use nebulization or you can use bronchodilators and both inhaled corticosteroids and if the symptoms are not paid responded with this you can go for the parenteral therapy.

In this scenario a 70-year-old laborer that is who is diabetic but having a known smoker smokes around 20 cigarettes per day has presented the emergency department with a history of or complain of cough for 3 days followed by breathlessness since morning. And on examination the patient is having a tachycardia with normal BP and saturation is on the very lower side that is 88 percent room air and respiratory rate of 32 which has to be the normal seen has to be in this 15 to 16 which is 32 here. And in the respiratory system examination bilateral infrascapular, interscapular inspiratory wheeze is present along with crepitations. And ABG that is RTL blood gas analysis showing you that the patient is having type 2 respiratory failure. The most important catchy point is that the patient is a chronic smoker and smoking round about 20 cigarettes per day.

So, what I told you in COPD usually you have a chronic smoking history with a patient might have a respiratory failure usually type 2. So, here the most probable diagnosis is COPD and emphysema. And this is the third and last scenario in which the 60-year-old female which is a known patient had tuberculosis 35 years back that is past history is there the patient has taken treatment for that and being stopped treatment only following the sputum was negative. Now, the patient presented to you with a history of COPD for 1 week with foul smelling sputum and fever since 5 days and shortness of breath since 3 days. On auscultation you are getting a harsh bilateral infraaxillary inspiratory crepitations on the infraaxillary, infrascapular and intrascapular area.

So, what is the most probable diagnosis? The patient is having a past history of TB with history of foul-smelling sputum which provokes diagnosis towards bronchiectasis. So, with this I am completing my topic that is obstructive airway disease. These are my references and thank you so much.