Pathophysiology And Pharmacology Of Asthma

Patient Status

A boy named Benji Smith that is 11 years old admitted to the hospital suffering from moderate exacerbation of asthma. Asthma is an overreaction but is not an auto immune disease. Benji was diagnosed with asthma at the age of 7. He was experiencing an upper respiratory tract bacterial infection from last week with unresolved cough and breathlessness. The level of aggravation does not identify with asthma seriousness but rather identifies with airway hyper responsiveness (AHR). At that time, he was able to talk in phrases and had an audible wheeze. He acknowledged that he faces the difficulty in playing games specially soccer and he started coughing and wheezing while laughing. When admitted to the hospital, he appears to have increased distress, tracheal tug and moderate accessory muscle use and was speaking in single words with great difficulty. His usual asthma treatment includes salbutamol 2 puffs when required and he has been taking cough syrup and 2 puffs of salbutamol in every 4 hours because of the cold and cough he had from past few days.

Benji Smith feels breathlessness while playing soccer as the amount of oxygen required in the body is less due to smooth muscle constriction and moreover, this also increases the pulse rate as the heart beats faster to generate the amount of energy required while playing and it also pumps faster to compensate the lack of oxygen in the body. As asthma triggers the sympathetic nervous system (SNS) hence the asthmatic patient remains confused at many times. Benji avoided to run or play around his friends as he was having infection in his upper respiratory tract (trachea) and it can be transmitted to other children around him as it was a bacterial infection that can be spread when he will shake hand or come into contact with them.

When admitted to the ED his respiratory rate was 24 breaths per minute while after 30 minutes it reached at 30 breaths per minute. His pulse rate was 110 beats per minute but after 30 minutes it was 130 beats per minute. His temperature was 38.6 degrees C and it turned to 38.4 degrees C and his SpO2 was 94% on room air at the time of admission and it decreased to 90% on air after half hour. He was speaking in phrases at the time of admission but after half hour he was not able to speak in phrases and due to this he was just talking in few words.

Nursing Diagnosis

There are some diagnosis which are needed to be done immediately while considering an asthmatic patient such as observing the respiratory rate, pulse rate, temperature and SpO2 level which will help the health professionals to treat the person immediately and critical thinking is required to understand the reason behind every vital sign that is observed and among them the signs and symptoms which were observed in Benji Smith includes:

- Smooth Muscle Constriction
- Tachycardia
- Excess Mucus Production
- SpO2 Level

Additionally, Benji Smith is suffering from upper respiratory tract infection caused by bacteria. At the time the bacteria enter the body the immune system gets activated to protect the body from the bacteria and in response to this the temperature of the body may rise or fall. (Pokoroski, 2015) It also causes inflammation in the upper respiratory tract that is clearly observed by 5 symptoms that are: heat, redness, pain, swelling and loss of function.

Heat and redness in the body is due to vasodilation as the endothelial cells of the blood vessels get loosen up and allows white blood cells (WBC), Platelets, red blood cells (RBC) or even plasma to move out of the cell that produces heat and redness is due to the amount of RBC's that gets out of the cell.

During inflammation the cells got apart that is increase in the vascular permeability that give rise to swelling of skin and due to swelling the pressure in the nerves increases that causes pain. As a result of all these activities the upper respiratory system does not function well.

Smooth Muscle Constriction: The lumen that is located inside the bronchioles is surrounded by a layer of smooth muscles. When an allergic person comes into the contact with any allergen that may include pollens or dust particles it will lead to an immune response by the immune system of the body as when the allergens enters the body it binds with the immunoglobulin E (IgE) antibodies that were released by the immune system in response to that allergen and it binds at the IgE receptor present on the mast cell. (Judy Craft, 2018) The mast cell gets degranulated and causes chemotaxis that will result in the constriction of smooth muscles also called Broncho Constriction or inflammation in the trachea or bronchioles that will result in swelling and pain and it will further block the air passage.

It does not allow the air to pass through it in a normal manner hence the exchange of gases cannot be done appropriately by the alveoli that are connected to the blood capillary. Due to this the person will breathe at a faster rate as compared to the normal respiration rate of that person.

Tachycardia: It is a condition that makes the heart beat more than 100 times per minute. At the time of asthma attack, the air passage gets blocked and hence the oxygen in the blood decreases and the carbon dioxide level in the blood increases and it causes difficulty in breathing Hence the heart starts beating at a faster rate to compensate for the lack of oxygen that results in increased pulse rate and person become fatigue due to the energy spend in breathing rapidly that also creates stress on the acromion muscles and the person feels dizzy or sometimes get faint.

Excess Mucus Production: Due to inflammation in the bronchioles at the time of asthma the mucus is produced and secreted greatly in increased quantity and an increase in mucus in the airway lumen is noted. (Gordan, 2018) The excess of mucus in the lumen, bronchioles or trachea causes respiratory tract infection, wheeze, pain and swelling and this brings about the increase or decrease in temperature of the person.

SpO2 Level: The SpO2 is the peripheral capillary oxygen saturation level in the human body that is the amount of oxygen in the blood. During asthma the amount of oxygen decreases as the person is not able to breath in the amount of air which is required and the oxygen is not properly supplied to the brain thus the person remains confused or agitated and all this is due to smooth muscle constriction and the drop in the level of SpO2 will further increase the level of

carbon dioxide in the body.

Care Plan

To cure the disease proper medication is needed that will reduce the risk of aggravating the symptoms. The medications given to Benji Smith includes:

- Salbutamol 12 puffs of 100µg /puff after 20 minutes for 3 times
- Ipratropium 8 puffs of 20µg /puff after each salbutamol via spacer
- Oral Prednisolone 1mg/kg (maximum 60kg) for 1-2 days
- 2 Liters of Oxygen (if the SpO2 level is under 93%)

These medications given to the asthmatic patients are absorbed and metabolized in different organs within the body and also have different pharmacodynamics (provides clear understanding of action of drug in the body) and pharmacokinetics (includes absorption, distribution, metabolism and excretion of drug in body) that needs to be focused while administering the dose to a patient.

1. Pharmacodynamics & Pharmacokinetics of Salbutamol

1.1 Pharmacodynamics of Salbutamol

The dosage of salbutamol would relax and ease the air passageways as the patient has asthma. (J Barnes May 9, 2007) Salbutamol is a beta-2 agonist receptor I.e. it binds to the receptor and activates it to produce the biological response and causes suitable action. It activates the G protein that activates the adenylate cyclase and leads to the synthesis of cyclic AMP (ATP to cAMP). It further phosphorylates and will stop the activity of myosin light chain kinase that converts the myosin phosphate into myosin with the help of phosphatase enzyme that causes bronchiole smooth muscle relaxation. Spacer should be used to consume the medicine as it decreases the opportunity of the “racing heart” reaction by decreasing the amount of medicine that stores in the mouth. (Johnston NW, 2005)

1.2 Pharmacokinetics of Salbutamol

The mode of action is through aerosol. The salbutamol is absorbed directly through the lungs and acts within 3 to 5 minutes. 10 to 70% of salbutamol binds with the plasma protein and gets metabolized in the liver. The half-life of this medication is 2 to 4 hours after intake and it is further excreted through urine.

2. Pharmacodynamics & Pharmacokinetics of Ipratropium

2.1 Pharmacodynamics of Ipratropium

Ipratropium is a cholinergic muscarinic receptor antagonist that causes bronchodilation as it inhibits the activity of in through receptor that are bind with G protein on bronchiole smooth muscle cells and inactivates the G protein that will stop the action of phospholipase C enzyme on hydrolyzing the membrane.

It further reduces the production of IP3 and DAG that will lead to the closure of calcium channel

and will decrease the concentration of calcium in the smooth muscle cells. The high calcium concentration inactivates the activity if myosin light chain kinase.

Simultaneously, it activates the myosin phosphatase that converts myosin phosphate into myosin and results in the smooth muscle relaxation and adding to this it also exhibits the production of mucus in the respiratory tract that will help to get relief from cough and excess sputum production.

2.2 Pharmacokinetics of Ipratropium

The mode of action is through aerosol. 10 to 30% of the dose directly goes to the lungs within 2 to 3 minutes and the rest of the 10 to 70% goes to the stomach through inhalation technique. 20% of Ipratropium binds to the plasma protein and 77% of the dose gets metabolized. The half-life of the drug is 2 to 4 hours and is excreted through the renal system. (Pokorski, 2015)

3. Pharmacodynamics & Pharmacokinetics of Prednisolone

3.1 Pharmacodynamics of Prednisolone

Prednisolone is an anti-inflammatory corticosteroid that are most prominent and effective. These are transported by the Glucocorticoid binding protein and it binds to the glucocorticoid receptor complex that is coupled to the 2H protein I.e. HSP70 and HSP90 located in the cytoplasm of cell.

It further activates the receptor and dissociate the 2H protein from the complex that allows the activated Glucocorticoid receptor complex to translocate in the nucleus, activates the complex and binds to the glucocorticoid complex element that alters the genes transcription that will upregulate the gene for lipocortin and will inhibit the phospholipase A2 enzyme and prevents the liberation of Arachidonic acid and reduces the production of prostaglandins that will perform the anti-inflammatory action.

It also reduces the cytokines to be made specially Interleukin1 to Interleukin8 and do not allow the neutrophil to leave the blood stream and goes to the tissue that is damaged. It also down regulates the fibroblast function. Lastly, it decreases the synthesis of histamine and prostaglandins that are proinflammatory chemicals and increases the transcription that will allow to perform the anti-inflammatory action.

3.2 Pharmacokinetics of Prednisolone

Prednisolone is an anti-inflammatory drug or immuno-suppressive agent that is absorbed through the gastrointestinal track and gets metabolized in the liver. It has a half-life of 2 to 3 hours and is excreted through the urine.

In case of Benji Smith immediate SpO2 was given as he was having lack of oxygen in the body that was just 90% on air after 30 minutes of admitting to the hospital. Lack of oxygen can cause serious problems in the body as it creates a stress on the heart that may even lead to heart failure in some cases. Lack of oxygen in the blood will increase the amount of carbon dioxide and due to that person is not able to breath properly that causes breathlessness. In severe case this condition may cause death of the person due to poor functioning of

respiratory system. (Hammerman SI,2002) Being a nurse proper diagnosis and treatment are required to help the diseased patient. Frequent checkups must be done timely to check for the vital signs and symptoms and the treatment or medication for that. Complete documentation of the patient is crucial to access the medical history of the patient like any allergies or diseases, current medications and treatment to plan the care for the patient.

Referencing

1. Craft, J., Gordon, C., & Tiziani, A. (2011). Understanding pathophysiology. Chatswood, N.S.W.: Mosby
2. Bryant, B. J., Knights, K. M., Darroch, S., & Rowland, A. (2019). Pharmacology for health professionals. Chatswood, NSW: Elsevier Australia
3. Symptoms of asthma. (2005). Asthma Magazine, 10(2), 31–32. doi: 10.1016/j.asthmamag.2005.03.001
4. Lehne, R., 2007. Pharmacology for Nursing Bullock, S. and Hales, M., n.d. Principles of Pathophysiology
5. Currie, G. and Baker, J., 2012. Asthma. Oxford: Oxford University Press
6. Hilty, M., Burke, C., Pedro, H., Cardenas, P., Bush, A., Bossley, C., Davies, J., Ervine, A., Poulter, L., Pachter, L., Moffatt, M. and Cookson, W., 2010. Disordered Microbial Communities in Asthmatic Airways. PLoS ONE, 5(1), p.e8578
7. MIMS Australia. 1st issue. Australia: KHL Printing Co Pvt Ltd Holgate, S. and Douglass, J., 2013. Fast Facts. 4th ed. Health Press
8. Holgate, S. and Douglass, J., 2013. Fast Facts. 4th ed. Health Press
9. CAROTENUTO, M., PERFETTI, L., CALCAGNO, M.G. and MERIGGI, A., 2018. Comparison Of Acute Bronchodilator Effects Of Inhaled Ipratropium Bromide And Salbutamol In Adults With Bronchial Asthma. Journal of Allergy and Clinical Immunology, 141(2)
10. Dryden, Donna M, and United States Agency for Healthcare Research Quality Content Provider. Exercise-induced Bronchoconstriction and Asthma. 2010. AHRQ Publication Exercise-induced Bronchoconstriction and Asthma. Web
11. Ozier, A., Allard, B., Bara, I., Girodet, P. O., Trian, T., Marthan, R., & Berger, P. (2011). The pivotal role of airway smooth muscle in asthma pathophysiology. Journal of allergy, 2011
12. Van Schoor, J., Joos, G. F., & Pauwels, R. A. (2000). Indirect bronchial hyperresponsiveness in asthma: mechanisms, pharmacology and implications for clinical research. European Respiratory Journal, 16(3), 514-533