Q1- the early phase response of bronchial asthma characterized by:

1. Increase in the number of mast cell.
2. Usually start after 10 hours.
3. Include the reactivation of already existed cell.
4. Recruitment of basophil and T helper cell.

Q2- one of the following drugs is consider as quick relieve medication:

1. Salmetrol .
2. Budesonide.
3. Monteleukast.
4. Salbutamol.

Q3- which one is true in management of bronchial asthma with corticosteroid?

1. The oral role is preferred.
2. Corticosteroid bind to G protein in cell surface membrane.
3. It is considered as quick relieve medication.
4. Corticosteroids inhibit inflammatory cytokine release and reduced activity of inflammatory immune cells.

Q4- you are treating patient with asthma at ER with frequent administration of salbutamol nebulizer, all of the following side effect can be expected EXCEPT:

1. Tachycardia
2. Bradycardia
3. Hand tremor
4. Hypokalaemia

Short assay:

Q1- Theophylline is relatively inexpensive drug however it is not used as a first line in management of bronchial asthma, explain why?

* **Theophylline is a relatively non-selective inhibitor of phosphodiesterase and it is this lack of specificity that may contribute to some of the adverse effects that are observed with theophylline use.**
* **Theophylline has a wide range of potential serious side effect.**
* **It is a drug with narrow therapeutic index**

**Q2-** what are the feature of acute severe asthma?

1. PEF 33-50% of the predicted
2. Heart rate more than 110 beat/min.
3. Respiratory rate more than 25 cpm.
4. Inability to complete sentences in one breath.

Q3- what are the mechanism of action of corticosteroid in management of bronchial ashma?

* Corticosteroids exert multiple anti-inflammatory actions including inhibition of inflammatory cytokine release and reduced activity of inflammatory immune cells.
* Corticosteroids also interact with specific receptors in tissues to regulate expression of corticosteroid-responsive genes. Several inhibitor proteins such as annexins and lipocortins are generated in response to corticosteroid receptor binding, which appear to inhibit the release of the arachidonic acid substrate from membrane lipids.