

Case study: patient with shortness of breath



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Patient Identity

The patient is a 54 year old female, Mrs SK who is a housewife with a BMI of 25.7kg/m².

Presenting Complaints

She was brought in to the Accident and Emergency (A&E) department, complaining of shortness of breath (SOB) and a productive cough.

History of Presenting Complaints

The patient was experiencing SOB for the past 2-3 days, and was progressively worsening on the day on admission. It was not associated with chest tightness and she was able to sleep the night before. She was also having persistent productive cough with white sputum since she was last discharged 12 days ago.

Past Medical History

She was diagnosed with diabetes mellitus and hypertension 8 years ago and has history of gastritis for the past 5 years. She was newly diagnosed with bronchial asthma in her last admission two weeks ago.

Social History

The patient is a widow since 6 years ago and is a housewife with 3 children. She stays in a factory area and has a cat at home. She is a non-smoker and a non-alcoholic.

Family History

Her mother and father have no known medical illness, but she has a cousin who suffers from bronchial asthma and is frequently admitted to the wards.

Drug History

The patient was on Salbutamol and Budesonide inhalers, 200mcg when necessary and 200mcg once at night respectively for her bronchial asthma. For her hypertension, she was on 40mg Telmisartan tablets once at night. She was also taking Gliclazide tablets, 80mg twice daily and Metformin tablets, 500mg three times a day for her diabetes mellitus. For her hypercholesterolaemia, the patient was taking Lovastatin tablets 20mg once at night. Based on the Morisky Scale, she was compliant with her medication and she had no known drug allergy.

Examination Details

On examination, the patient was alert and conscious. She was pink and appeared to be fairly hydrated. She was also able to speak in full sentences, and was not tachypnoeic. A Chest X-ray showed that there was a pneumonic consolidation at the right lower lobe of her lungs. Her blood pressure (BP) was 152/82mmHg, pulse rate (PR) was 109 beats per minute (bpm) and was afebrile. Her oxygen saturation (SpO₂) was 96% under 3 litres of oxygen and her blood glucose was measured to be 4.7mmol/L.

Investigations

Upon admission, standard laboratory investigations were carried out and were obtained. From the renal function test, it was seen that the patient had a low potassium level of 2.8mmol/l and her calculated creatinine clearance

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was 60. 0ml/min which indicated that she had mild renal impairment. The liver function test showed that she had normal liver function. The following shows the results that were out of the reference values for her haematological tests.

C-Reactive Protein (CRP)

31.1 $\hat{+}$ '

Erythrocyte Sedimentation Rate (ESR) (0 - 15 mm/hr)

110 $\hat{+}$ '

Haemoglobin (Hb)

(13.5-18 g/dl)

10.3 $\hat{+}$ "

Haematocrit

(0.36-0.46 L/l)

0.303 $\hat{+}$ "

Red Blood Count (RBC)

(3.8-4.8 $\times 10^{12}$ /l)

3.45 $\hat{+}$ "

White Cell Count (WCC)

(4-11 x 10⁹ /l)

15.1 x 10⁹ /l

Neutrophil (Neutro)

(2 - 7.5 x 10⁹ /l)

10.57 x 10⁹ /l

Diagnosis/Impression

Patient was initially diagnosed with Acute Exacerbation of Bronchial Asthma (AEBA) secondary to an upper respiratory infection (URTI) to rule out pneumonia. However, later in the day when the chest X-ray came back, she was diagnosed with pneumonia with right parapneumonic effusion.

Management Plan

The patient's current medication was continued and was given 3 litres of oxygen via a nasal prong (NP). She was commenced on prednisolone tablets, 30mg once a day and was given nebulised Combivent (Ipratropium 20mcg/salbutamol 100mcg), every 4 hours. Her peak expiratory flow rate (PEFR) and SpO₂ was to be monitored. Antibiotics were kept in view to be started if necessary after the total white blood count results came back.

Clinical Progress

Upon admission, the patient was afebrile, was tolerating orally well, did not have any sorethroat but was having a non productive cough. An echocardiogram (ECG) was done and it showed that she had sinus rhythm

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with no ischaemic changes. As her chest x-ray showed a right lower zone consolidation, she was diagnosed with pneumonia. She was immediately commenced on 2g Ceftazidime intravenously, and then continued on 1g three times a day. She was also under nebulised combivent every 6 hours. Her metformin and gliclazide was stopped and she was started on subcutaneous 10 units of Humulin ® three times a day and 12 units of Humulin N once at night. On Day 2 of her stay, her blood results came back and as she has low potassium levels, she was given 15mls of Mist KCl three times daily and two Slow K tablets once daily. She was still complaining of cough without sputum and was given 15mls of Benadryl (diphenhydramine) syrup three times a day. The patient did not have any major complaints on the third day and was tolerating orally well. There was no SOB seen and she had good inhaler technique. She was then taken off the nebulizer combivent and the oxygen. By day 4, the patient was comfortable, and her cough and sputum had decreased. Examination on her lungs showed that she had prolonged expiratory phase. She was stopped on the Benadryl as well as Mist KCl and Slow K. After reinforcement on the inhaler technique by the pharmacist, the patient was discharged on day 5 as she was afebrile and had minimal cough. On discharge, she was then switched back to her oral hypoglycaemics and her intravenous antibiotic was switched to oral Cefuroxime 500mg twice daily for the next 10 days. She was also given Neulin SR 250mg once at night. Table 1 shows the vital signs chart for Mrs SK throughout her hospital stay.

Table 1: Vital Signs Chart

Day

Time

BP (mmHg)

PR (bpm)

SpO2

Blood Glucose (mmol/l)

1

13. 00

178/102

109

100%

6. 9

14. 00

152/82

109

98% â†“ NP

—

15. 40

—

—

—

4. 7

18. 40

133/73

114

97% $\hat{+}$ RA

—

21. 15

—

—

—

6. 2

23. 05

151/82

119

—

—

2

03. 15

143/81

106

—

—

06. 00

—

—

—

6. 3

08. 30

119/67

94

100%

—

10. 35

—

—

—

4. 8

11. 24

100/61

107

97%

—

15. 20

112/82

100

—

—

16. 30

128/70

100

—

6. 2

22. 00

—

—

—

3.6

23.50

118/59

66

98%â†“ 3L O2

—

3

04.10

124/64

104

100%â†“ 3L O2

—

06.15

—

—

—

8.2

09. 40

100/60

96

—

—

11. 50

—

—

—

8. 5

15. 30

108/67

94

—

—

17. 20

—

—

—

7. 4

20. 00

121/75

86

—

—

4

04. 00

110/56

62

—

—

06. 00

—

—

—

7. 9

08. 00

110/70

63

—

4. 2

16. 00

105/75

91

96%

—

17. 00

—

—

—

9. 7

22. 00

138/67

114

—

6. 1

5

05. 00

—

—

—

9. 6

07. 15

—

—

—

11. 1

Pharmaceutical Care Issues

The first care issue is to review the management of AEBA based on the British Guideline on the Management of Asthma. The dose of prednisolone should be increased to 50mg once a day for at least 5 days or until recovery. Since the patient is prescribed with theophylline on discharge, she should be counseled on the signs and symptoms of theophylline toxicity such as confusion, dizziness, diarrhoea, nausea, fatigue and headache.

The second issue is regarding the choice of antibiotics for the treatment of community acquired pneumonia in this patient. A sputum full examination microscopic examination (FEME) should be requested to identify the causative microorganisms of the lung infection. If empirical treatment is to be started the preferred drugs of choice would be amoxicillin 500mg three times a day plus either erythromycin 500mg four times a day or clarithromycin 500mg twice daily. Alternative choices would be levofloxacin
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500mg once daily or moxifloxacin 400mg once a day, should the patient be intolerant of the preferred regimen. Thirdly, there is no clear indication of the prescription of the diphenhydramine in the first place, as it would only suppress the patient's cough, which is inappropriate. Hence it should be stopped immediately.

Next, the patient's updated blood cholesterol levels should be taken and the appropriate use of statins should be reviewed. As she is on long-term statin use, her liver enzymes should be monitored regularly and if is raised by three-fold, she should stop taking the Lovastatin. She should also be counseled on the symptoms of rhabdomyolysis which is related to the long term use of statins, such as unexplained muscle pain, stiffness, weakness and the darkening of urine colour.

The following issue is regarding the patient's diabetes management. A HbA1c test should be done to determine her glycated haemoglobin level to see how well her self-management has been. She should also be advised on diet and lifestyle to keep her diabetes under control. Lastly, as she has low red blood count, haemoglobin and haematocrit levels, it is suspected that she has anaemia. Further tests should be done to confirm this, and if it is diagnosed, she should be given ferrous supplements such as ferrous sulphate tablets 200mg twice daily.

DISEASE OVERVIEW AND PHARMACOLOGICAL BASIS OF DRUG THERAPY

Acute Exacerbation of Bronchial Asthma: An Overview

Asthma is a chronic inflammatory disorder of the airways where many cells and cellular elements play a role. This leads to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the morning. These inflammatory symptoms are commonly associated with extensive but variable airflow obstruction within the lung as well as airway hyperresponsiveness and this is reversible either on its own or with treatment. 1

Asthma is a worldwide problem as it is estimated that about 4.5% of the world's population is affected, which amounts to 300million individuals approximately. The global prevalence of asthma varies from 1-18% of populations in countries all over the world.

Asthma has three distinguishing characteristics which are airflow limitation, airway hyperresponsiveness, and bronchial inflammation. Airflow limitation is usually resolved by itself with or without treatment but for individuals with chronic asthma, inflammation may result in irreversible airflow limitation. Stimuli such as irritants or allergens may pose as triggers in airway hyperresponsiveness and bronchial inflammation is associated with eosinophils, T-lymphocytes and mast cells which cause plasma exudation, smooth muscle hypertrophy, mucous plugging and epithelial changes. It is shown that inflammation of the airways play a major role in the pathology of asthma and this starts when allergens or irritant trigger the activation of cells such as epithelial cells, macrophages, lymphocytes and mast cells. This leads to cytokine or mediator release and smooth muscle contraction resulting in cellular infiltration of eosinophils and neutrophils causing airway inflammation including oedema, epithelial permeability or injury, mucous

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secretion and vascular permeability which eventually leads to airway obstruction and hyperresponsiveness.

The diagnosis of asthma is based on a collection of signs and symptoms without a reasonable explanation for them and spirometry is an early test which is easy to assess if there is any airflow obstruction present and its extent. For diagnosed patients with asthma, acute exacerbations may occur and because patients with severe asthma are at increased risk of death following exacerbations, assessments of exacerbations are crucial. Clinical features of acute asthma exacerbations include severe breathlessness, tachypnoea, tachycardia, silent chest, cyanosis, or syncope. Peak Expiratory Flow (PEF) or Forced Expiratory Volume in one second (FEV1) is also used to measure the lung capacity. Oxygen saturation (SpO₂) is measured using a pulse oximetry and this aids oxygen therapy as oxygen therapy is given in order to keep SpO₂ levels at 94-98%. Measurements of arterial blood gases (ABG) are usually not necessary unless patients present with features of life-threatening asthma or have SpO₂ of less than 92% as there may be a risk of hypercapnea if SpO₂ is lower than 92%. Chest X-rays are also not recommended unless patients are suspected of pneumonia or lung consolidations, suffering from life-threatening asthma, having unsatisfactory response to treatment or if they require ventilation.

Pharmacological Intervention in the management of AEBA2

Oxygen

Oxygen therapy is needed most of the time as patients who are having acute asthma usually present with hypoxia as well. Hence, all patients with hypoxia

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who are suffering from acute severe asthma should be given oxygen and their SpO₂ levels should be kept at 94-98%.

Î² agonist bronchodilators

As first line therapy, high dose inhaled Î² agonist bronchodilators are used as soon as possible as rapid relievers of bronchospasm. For patients who are unable to use inhaled therapy, intravenous Î²-agonists are used instead. Î² agonist bronchodilators work by stimulating the Î² adrenoceptors in the lungs, thus causing relaxation of the airways. Examples of short acting Î²-agonist are salbutamol and terbutaline, and a long acting Î²-agonist is salmeterol.

Glucocorticosteroids

Steroids should always be given in all cases of acute asthma. Examples of these are prednisone, prednisolone, dexamethasone, and hydrocortisone. They exert an anti-inflammatory effect by inhibiting transcription of the genes for the cytokines implicated in asthmatic inflammation and hence reduce airway hyper-responsiveness.

Anti-cholinergic agents

Ipratropium bromide is one of the anti-cholinergic agents that is used widely in treatment of acute exacerbations of asthma. Nebulised ipratropium bromide is used in combination with a Î²-agonist bronchodilator as treatment for patients with severe acute or life-threatening asthma. Anti-cholinergic agents work by inhibit muscarinic receptors M₁ and M₃ which then reduces cGMP formation and decreases smooth muscle contractility in

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the lungs. This eventually results in bronchodilation and reduces mucus secretion.

Other therapies

Other therapies include the use of magnesium sulphate. A single bolus dose of intravenous magnesium sulphate is administered to patients with acute severe asthma with previous unsatisfactory response to inhaled bronchodilator therapy or for patients who are suffering from life-threatening or near fatal asthma. It is believed that magnesium sulphate works by reducing calcium uptake by the bronchial smooth muscle cells, causing bronchodilation and also inhibits mast cells degranulation, thus reducing the release of inflammatory mediators such as histamines, and leukotrienes.

EVIDENCE FOR TREATMENT OF THE CONDITION

The management of asthma can be divided into two parts; acute treatment, and long term management.

Management of acute asthma

It has been shown that most patients suffering from acute severe asthma are hypoxaemic. Therefore it is essential that supplementary oxygen therapy be given to them. 3-6 This is administered via a face mask or nasal prong with the patient's SpO₂ kept between 94-98%. 7 Where nebulisers are needed in therapy, oxygen-driven nebulisers are favoured instead of those that are air-driven due to oxygen desaturation when driven by air alone. 8-10 However, the lack of provision of supplemental oxygen should not pose as a factor in omitting nebulised therapy from administration if deemed appropriate. 11

Referring to the case presented above, the patient was treated accordingly as she was immediately given supplemental oxygen and her SpO₂ was maintained well above 96% throughout hospital stay.

As acute asthma is associated with symptoms of bronchospasms such as wheezing and tachypnoea, the main aim of treatment is to quickly resolve these symptoms and most often, high doses of inhaled β_2 agonist bronchodilators are effective with minimum adverse effects. 12-14 Salbutamol is usually the drug of choice although there is no significant differences in terms of efficacy as compared to Terbutaline. It is shown that there are no significant clinical benefits by using a non-selective β_2 agonist such as epinephrine instead of selective β_2 agonists. 15 Based on a meta-analysis, it is seen that β_2 agonists administered via inhalation are more preferable and has similar efficacy with those administered intravenously in adult acute asthma. 16 In ventilated patients or those in life-threatening conditions, parenteral β_2 agonists may be added to inhaled β_2 agonist treatment although there is little evidence supporting this treatment. Although a single bolus nebulisation may relieve most acute asthma cases, it is shown that continuous nebulised treatment of β_2 agonists is more effective in relieving acute asthma for those with unsatisfactory response to initial therapy. 17, 18

Steroid therapy is always given in acute exacerbations of asthma and it is proven that it has better result if given earlier. It not only reduces mortality but it also reduces relapses and the number of hospital admissions as well. 19, 20 Oral steroids given are seen to be equally as effective as parenteral treatment hence there is no need for the use of parenteral administration of

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steroids unless the patient is unable to tolerate orally. 19 Prednisolone 40-50mg is given daily for at least five days or until recovery and this can be stopped abruptly after the patient has recovered. 2 As long as the patient is on inhaled steroids, there is no need for the dose to be tapered down slowly prior to discontinuation. 21

In the case presented, the patient was commenced on steroid therapy but was under-treated as she was only given prednisolone 30mg once daily for just one day. Hence, there is a need to increase the dose of prednisolone to 50mg and to continue it for at least another four days or until recovery before stopping this treatment.

In hospital therapy, anticholinergic treatments are given to severe exacerbations of asthma and nebulised ipratropium bromide is always the drug of choice used in clinical settings. A combination of nebulised ipratropium bromide with a β_2 agonist bronchodilator is often given as treatment as it is proven that a combination of these two agents has a significant increase in bronchodilatation as compared to the use of a β_2 agonist alone. Hence, there is faster recovery and will reduce the length of hospital stay. However, it is also seen that anticholinergic treatment is not particularly effective and favourable for cases of mild exacerbations of asthma as well as after the patient has been stabilized, thus is not necessary in these cases. 22-24

The patient in this case was seen to be having a mild exacerbation of acute asthma and hence nebulised ipratropium bromide treatment was not necessary. However, the use of nebulised Combivent, a combination of

ipratropium bromide and salbutamol was justified since this patient was re-attending with a relapse and she was also suffering from pneumonia as well. Hence, there was probably a need for a quicker rate of bronchodilation as well as faster recovery for her.

The use of magnesium sulphate in hospital treatment of AEBA is not widely seen, however there have been some evidence showing the bronchodilating effects of magnesium sulphate when used in adults. 25 There are also studies which report that nebulised magnesium sulphate combined with a β_2 agonist shows positive outcomes and good clinical effectiveness in hospital settings. 26, 27 The use of an intravenous bolus administration of magnesium sulphate is believed to promote lung function in patients who have severe asthma without harmful side effects. 28 Nevertheless, there have been no studies on the repeated administrations of magnesium sulphate, though it is presumed that repeated use may lead to hypermagnesaemia, causing muscle weakness and respiratory failure. As further extensive studies need to be done to determine the most suitable route and dosing of magnesium sulphate, this treatment is reserved only for patients with acute severe asthma without satisfactory response to inhaled bronchodilator therapy and patients with life-threatening or near fatal asthma.

Monitoring should be carried out constantly throughout hospital stay and in acute asthma cases, monitoring of PEF is crucial. PEF readings should be measured and recorded every 30 minutes after treatment has been started. PEF should also be monitored pre- and post- nebulisation therapies as long

as the patient is in hospital and until the asthma is well under control after discharge.

It is seen that after hospital discharge, a relative amount of patients either experience relapse or are readmitted into the hospital with at least 15% within two weeks following discharge. 29 Therefore it is essential that patient education such as proper inhaler technique, and well-documented PEF recordings with action plans depending on symptoms experienced should be instilled in order to reduce rate of relapses as well as minimize problems associated with exacerbations after discharge. 30

Monitoring of the patient's PEF was done consistently throughout her hospital stay and the patient was given sufficient counseling prior to discharge on her inhaler technique. However, there was no evidence that the patient was educated on self-documenting PEF recordings as well as action plans based on symptoms experienced following discharge and this should be done in this case to avoid another exacerbation of her condition.

Long Term Management of Asthma

The aim of management of asthma is to keep it well-controlled without the need of rescue medications, asymptomatic, no exacerbations, no hindrance to daily activities including exercise as well as normal lung function. A stepwise management approach is adopted for asthma patients and this is to acquire initial control and maintain it by stepping up treatment to improve control if necessary or stepping down treatment if there is good control over the condition to maintain the lowest step that will control the patient's condition.

As the patient is currently on regular preventer therapy with inhaled steroids, she is currently on step 2 of the management of asthma. There have been many studies being carried out to compare the different inhaled steroids that are being used for asthma and it is shown that beclomethasone dipropionate and budesonide are both similarly clinically effective although there may be different devices for delivery. It has also been seen that fluticasone and mometasone being administered at half the dosage of beclomethasone and budesonide shows equivalent clinical effectiveness, however there is somewhat inadequate evidence that fluticasone possesses fewer side effects and further studies need to be carried out on establishing the safety profile of mometasone. 31 A new inhaled steroid has been introduced which is ciclesonide and clinical trials have shown evidence that it has more local activity than systemic and less oropharyngeal side effects as compared to the regular inhaled steroids. 32-35 Although this seems promising, this clinical advantage is still controversial as its safety to efficacy ratio has yet to be established and compared with the conventional inhaled steroids. Inhaled steroids are recommended as preventer drug therapy for adults as they are most clinically effective in controlling asthma based on the treatment goals outlined. 36-39 The frequency of dosing of inhaled steroids are generally twice daily and it is shown that there is slight clinical benefit obtained when taken twice a day than once daily, however a once daily dosing may suffice for those with milder asthma. There is also limited evidence of advantage with increased frequency of greater than twice a day. 37 In addition to that, starting at higher than recommended doses have no significant effectiveness in management of mild to moderate asthma. 40 Hence the recommended dosage for inhaled steroids would be 200-800mcg

daily. This would be an add-on therapy to the step 1 management of using inhaled short acting β_2 agonist bronchodilator as required.

Based on the presented case, the patient was on budesonide 200mcg once at night prior to admission but this was immediately increased on admission and was in line with the recommended guidelines as she was continued on budesonide 400mcg twice a day together with salbutamol 200mcg as required following discharge.

Other preventer therapies may be included for the patient despite inhaled steroids being the first choice of drugs for preventer therapy. These alternatives are less effective although they have shown some clinical benefit in patients who are on short acting β_2 agonists only. Chromones which act as mast cell stabilizers such as sodium cromoglicate and nedocromil sodium have shown to be beneficial in adults. 41, 42 Apart from that, leukotriene receptor antagonists montelukast and zafirlukast too have clinical benefits. 37, 43, 44 Theophylline also have some evidence in showing benefits in adults. 36, 45

The patient in the case presented above was prescribed sustained-release theophylline on the last day of admission. Although it is another option that may be added to daily controller medications for step 2 management, there is very little evidence on the clinical efficacy of it as a long term controller. There is no reason to justify the use of theophylline in this case as the patient is responsive and can be controlled on inhaled steroids. Furthermore, theophylline has a narrow therapeutic index and close monitoring of plasma theophylline levels is necessary because at concentrations above

25µg/ml, there is high risk of tachycardia and seizures may occur if concentrations exceed 35µg/ml.

CONCLUSION

After reviewing the management of the patient's condition throughout hospital stay, it can be concluded that SK was treated adequately based on the current guidelines and evidences attainable. She was given all necessary treatment at point of admission and there was no lacking of medications in all four days of her hospital admission. Apart from that, monitoring of her condition was carried out consistently and all data was updated, leaving no room for questioning and doubt. However, there were a few issues that came to attention which were the prescribing of several drugs that were unnecessary such as diphenhydramine and theophylline. There were little and no clear evidence that these drugs prescribed would be of benefit to the patient, and may also increase the risk of harmful effects to her as well.

Alongside treatment of her acute condition, SK's controller medications were reviewed and subsequent changes were made as appropriate. Besides that, her other co-morbidities were also managed well as treatments for her hypertension and diabetes mellitus were given accordingly.

PATIENT MEDICATION PROFILE

PATIENT DETAILS

Name

S. K.

Consultant

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Dr YKS

General Practitioner

Address

Gender

Female

Weight

65kg

Height

1. 59m

Community Pharmacist

Date of Birth (Age)

54 years old

Known Sensitivities

NKDA

Social History

Widow of 6 years, Housewife, Non-smoker, Does not drink

PATIENT HOSPITAL STAY

Presenting complaint in primary care / reason for admission

Admission date

17/04/09

Shortness of breath for the past two days, progressively

Discharge Date Discharged to

21/04/09 Home

worsening today and productive cough.

RELEVANT MEDICAL HISTORY

RELEVANT DRUG HISTORY

Date

Problem Description

Date

Medication

Comments

2001

Diabetes Mellitus

T. Diamicron

80mg BD

2001

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Hypertension

T. Metformin

500mg TDS

2004

Gastritis

T. Telmisartan

40mg ON

2009

Bronchial Asthma

MDI Salbutamol

200mcg PRN

MDI Budesonide

200mcg ON

T. Lovastatin

20mg ON

RELEVANT NON DRUG TREATMENT

Prescribed Medication

Start

Stop

Clinical/Laboratory Tests

Date Result

1

T. Telmisartan 40mg OD

18/04

21/04

2

T. Gliclazide 80mg BD

17/04

17/04

3

T. Metformin 500mg TDS

17/04

—

4

T. Lovastatin 20mg ON

17/04

19/04

5

MDI Salbutamol 200mcg 2 puffs PRN

17/04

—

6

MDI Budesonide 200mcg 2 puffs BD

17/04

—

7

T. Prednisolone 30mg OD

17/04

17/04

8

Neb. Combivent 6-hourly

17/04

19/04

9

IV Ceftazidime 2g STAT, then 1g TDS

17/04

21/04

10

S/C Humulin R 10units TDS

17/04

21/04

11

S/C Humulin N 12units ON

17/04

21/04

12

Syrup Diphenhydramine 15mls TDS

18/04

20/04

13

T. Slow K 2tabs BD

18/04

20/04

14

Mist KCl 15mls TDS

18/04

20/04

15

T. Theophylline 250mg OD

20/04

—

CLINICAL MANAGEMENT

Diagn