

Management and treatment and psychosocial aspects of pneumonia biology essay



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This essay will explore the pathophysiology, management and treatment, and psychosocial aspects of pneumonia in an adult patient. Information has been obtained by the means of history taking, examination, and analysing the patient's medical records to form a case study in which the sections mentioned will be considered in relation to the case study.

Section A – Case History

VD is a 68 year old female who was admitted into the acute medical unit (AMU) on 29/1/11 following complaints of generally feeling unwell and chest pain. She had a history of feeling unwell since 25/1/11 coupled with chest pain. This pain was a sharp pain under her right breast which was intermittent and radiated around her chest to her back. The pain was worse on inspiration or when coughing, and was relieved by over-the-counter analgesia. She also felt sweaty, pyrexia (39.7 °C), had rigors and aches over her body, however she was not short of breath. She also had symptoms of a non-productive dry cough, poor appetite and vomiting once in AMU (watery and colourless). She previously had no episodes of nausea and vomiting, no palpitations or headaches, no urinary symptoms and normal bowel movements. She has not had any recent contact with anyone who had similar symptoms.

In her past medical history she was diagnosed with Sjögren's syndrome and systemic sclerosis last year; both systemic autoimmune diseases. She was on two courses of antibiotics last year for associated pleuritic chest pain.

Her family history consisted of her father having ischaemic heart disease (IHD) and her mother dying from lung cancer, although she was a heavy

smoker. She currently lives with her husband at home and her occupation is as a shop assistant; this indicates that the infection she has is most likely to be community acquired. She has been a lifelong non-smoker and she does not drink alcohol. She was on no regular medication prior to being admitted, but is now on 1000mg of paracetamol four times a day (QDS) and 500mg of amoxicillin three times a day (TDS). She has no allergies.

On examination in a respiratory ward, VD was afebrile with a blood pressure reading of 95/65, a heart rate of 95 beats per minute and a respiratory rate of 18 breaths per minute. Oxygen saturations (SATS) were 96% in air and she was speaking in full sentences, whilst looking generally comfortable at rest. Her hands seemed dry and scaly on inspection but there were no abnormalities on her face. On palpation of her chest, there was equal chest expansion and no tracheal deviation. There were also no enlargement of cervical or supraclavicular nodes. On percussion, there were dull sounds that could be heard on both right and left lung bases. On auscultation, coarse crackles could also be heard in the right and left lung bases. There were no abnormal heart sounds heard and her capillary refill time (CRT) was less than 3 seconds. Her abdomen was soft and non tender, and normal bowel sounds were heard. There was normal tone, power and reflexes in all 4 limbs and her Glasgow Coma Scale (GCS) score was 15/15.

Her arterial blood gas values were as follows: pH 7.43, pCO₂ 5.49, pO₂ 10.1, HCO₃⁻ 26.8, basal excess of 2.8 and glucose of 5.6; these values indicated that she was not in respiratory failure. She was also found to have a raised C reactive protein (CRP) of 210, with a high neutrophil count of 10.1.

Her chest x-ray film revealed consolidation in her right lung base and no

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pneumothorax. The impression from the x-ray was that it was right lower lobar pneumonia (Figure 1 is an example of what VD's x-ray would have looked like).¹ No blood cultures were recorded in her notes as it was assumed that due to the neutrophilia the likely source was bacterial.

After being initially treated with intravenous (IV) antibiotics in hospital, her symptoms were relieved, no crackles could be heard and her chest was clearing up on 1/2/11. She was then discharged in the afternoon on 2/2/11 given the instructions to continue with her course of oral amoxicillin.

Section B – Pathophysiology

Introduction

Pneumonia can be described as an inflammation to the lungs' distal airways, particularly the alveoli, usually with a bacterial infection being the origin.^{2 3} It clinically presents as an acute illness which can include fever, cough and purulent sputum, although the latter was not present in VD. Pneumonias can be classified by the site of the consolidation (anatomically), or by the aetiology of the disease (see Table 1).^{2 3} VD was suspected to have lobar pneumonia after looking at her chest x-ray. The majority of lobar pneumonias are due to *Streptococcus pneumoniae* and can affect a large part, or a whole lobe of the lung.³

Lobar Pneumonia

There are four stages to the pathology of lobar pneumonia, which is a classic example of acute inflammation; these are: congestion, red hepatisation, grey hepatisation and resolution.³ Congestion is the first stage and lasts for approximately 24 hours. This is represented by protein-rich exudates leaking

into the alveolar spaces and also causing venous congestion consequently causing the lung to become oedematous, heavier and redder in colour. 3 The next stage is red hepatisation which has a duration of a few days. Large numbers of polymorphs (neutrophils and basophils) accumulate in the alveolar spaces along with some lymphocytes and macrophages. 3 Many erythrocytes are extravasated from the distended capillaries into the lung tissue, along with the overlying pleura being covered with fibrinous exudates. 3 The lung is now solid and airless, resembling a fresh liver. Figure 1 supports the latter statement by showing a solid consolidation in the right lower lobe. When the lung becomes grey and solid, this is grey hepatisation. This also lasts a few days and represents further accumulation of fibrin coupled with the destruction of leukocytes and erythrocytes. 3 The final stage is resolution, whereby the lung reverts to its normal condition. 4 This happens at approximately 8-10 days in cases which are untreated and is when the cells and fibrin in the alveoli undergo fatty degeneration. 3 4 This causes the exudates to be converted into an emulsion, producing a yellow pus-like appearance. 4 The exudates are now in a condition where they can be reabsorbed, whilst preserving the underlying alveolar wall structure. 3 4 The lungs would be softer but remain solid, and this would be confirmed on an x-ray by consolidation of the lungs.

Co-morbidities

VD's history also mentioned having a background history of Sjögren's syndrome and systemic sclerosis; both systemic autoimmune diseases.

Sjögren's syndrome is an inflammatory disease that predominantly affects the exocrine glands with an association to HLA-B8/DR3, which usually causes

dryness in the eyes and mouth. 2 5 However it can also cause extra glandular problems such as: Raynaud's phenomenon, arthritis and lung inflammation, causing degradation of the lining of the bronchioles and alveoli consequently causing lung infections. 2 5 6

Systemic sclerosis, also known as systemic scleroderma, is a multi-system autoimmune disease in which the cause is unknown. 2 It mainly causes tightness and hardening of the skin (such as VD's hands) but other systems can also be affected, such as the lungs. 2 There is some destruction to the lungs in patients with scleroderma which can lead to right heart failure due to pulmonary hypertension. 7 Other complications that involve the lungs include pulmonary haemorrhage, pneumothorax and pneumonia. 7

Summary

VD had come in with an acute infection and was diagnosed with pneumonia. Her right lower lobe was consolidated meaning that she has had it for a few days as protein exudates have leaked into the alveolar spaces and becoming fibrinous, showing up as solid on the chest x-ray, with her CRP (a marker of inflammation) also being raised. VD's medical history last year stated that she had suffered from two previous chest infections that required antibiotics for her to recover. This could possibly be due to the autoimmune diseases aforementioned that she had recently been diagnosed with, causing her to be more predisposed to contracting infections, especially in her respiratory tract. She is currently not on immunosuppressant drugs, but if she were to be for her autoimmune conditions it would then be detrimental to her immune system. This would leave her still being prone to acquiring infections, leaving her in quite a predicament.

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Section C – Treatment and Management

VD was on 1000mg of oral paracetamol QDS and 500mg of oral amoxicillin three TDS by the time she was moved to the respiratory ward. The main actions of these drugs were to improve her feverish symptoms and pain whilst also attempting to clear up her infection.

Paracetamol

Paracetamol (also known as acetaminophen in the USA) is one of the most widely used non-narcotic, analgesic and antipyretic over-the-counter drugs in the world. 8-11 It has properties resembling those of nonsteroidal anti-inflammatory drugs (NSAIDs) such as its analgesic and antipyretic actions, which can be traced back to the inhibition of the central nervous system's prostaglandin (PG) synthesis. 8 9 It also shares some anti-inflammatory properties, however it does not produce the platelet or gastric side effects that the other NSAIDs do, thus causing argument as to whether it should even be classified as an NSAID at all. 8 It is commonly given orally and is metabolised in the liver, with a half life of approximately 2-4 hours, hence why VD was given it QDS to avoid toxic doses.

Mechanism of Action

It is considered that the main mechanism of paracetamol is the inhibition of the enzyme cyclooxygenase (COX), COX-2 in particular as studies have shown that it is highly selective towards it. Due to its high selectivity towards COX-2, its inhibition towards pro-clotting thromboxanes is limited. 9 It is said that paracetamol works centrally and is a weak inhibitor of PG synthesis in intact cells, if the concentrations of arachidonic acid available are low

enough, through the inhibition of COX-1 and COX-2. 12 This concept was based on research that discovered that PG production in the brain was more sensitive to inhibition from paracetamol by tenfold compared to the spleen. 9 The COX enzymes are responsible for the metabolism of prostaglandin H₂ from arachidonic acid. 9 This is an unstable molecule that can form many pro-inflammatory compounds; COX is highly active when oxidised. 9 12 The oxidised form of the COX enzyme is reduced by paracetamol, stopping it from creating pro-inflammatory compounds. This lowers the amount of PGE₂ in the central nervous system, therefore decreasing the set-point of the thermoregulatory centre in the hypothalamus. 9

Exactly how the mechanism of the inhibition of the COX enzymes is still in discussion. Due to the differences of activity between NSAIDs and paracetamol, it is thought that there may be another variant of the COX enzyme that paracetamol interacts with, COX-3 - a splice variant of the COX-1 enzyme; however this is just a hypothesis and has yet to be proven. 8 9 12

Side Effects

When paracetamol is given at therapeutic doses adverse effects are uncommonly seen, although allergic skin reactions are sometimes observed. 8 Fatal hepatotoxicity can be potentially caused by toxic doses of paracetamol (10-15 grams). 8 Initial symptoms are nausea and vomiting, followed by liver damage after 24-48 hours. 8 13 This happens when the enzymes in the liver, cytochrome P450, catalysing the normal conjugation reactions become saturated, and consequently causes the drug to be metabolised instead by mixed function oxidases. 8 A toxic metabolite, N-acetyl-p-benzoquinoneimine, is formed and inactivated by conjugation with <https://assignbuster.com/management-and-treatment-and-psychosocial-aspects-of-pneumonia-biology-essay/>

glutathione. 8 13 However when glutathione levels are depleted, the toxic metabolite accumulates and reacts with nucleophilic constituents in the cell, causing necrosis in the liver and kidney tubules. 8

Contraindications

Paracetamol is generally well tolerated by the liver when polypharmacy is involved. However, evidence has shown that chronic alcoholics are more susceptible to paracetamol hepatotoxicity, even at therapeutic levels. 13 Chronic alcohol ingestion induces hepatic microsomal enzymes (CYP2E1) by twofold and can increase paracetamol hepatotoxicity, due to increased amounts of the toxic metabolite. 13

Amoxicillin

Amoxicillin is a moderate to broad spectrum, β -lactam antibiotic that is commonly used to treat infections that are caused by susceptible bacteria, pneumonia being one of them. 8 14 A derivative of penicillin, this semi synthetic β -lactam antibiotic is created by adding different side chains to the penicillin nucleus, causing it to become broad-spectrum. 8 It is sometimes combined with clavulanic acid in treatment to form co-amoxiclav, which is more effective in treatment nowadays due to the increase in antibiotic resistance; microorganisms are now developing a resistance to penicillins by secreting β -lactamases and the addition of clavulanic acid inhibits this enzyme. 8

Pharmokinetic Aspects

The routes of administration are quite vast: when given orally, amoxicillin is absorbed to a different degree compared to other penicillins as it depends on

their stability in acid and their adsorption to foodstuffs in the gut. 8 It can also be administered through intramuscular or intravenous injections; however intrathecal administration is generally avoided as it can cause convulsions. 8 Elimination of amoxicillin is rapid and mainly due to the kidneys, with 90% being through tubular secretion. This however may be advantageous as the inhibition to cell wall synthesis is intermittent rather than continuous, and exposure to the drug is reduced. 8

Mechanism of Action

β -lactam antibiotics inhibit the synthesis of the bacterial cell wall peptidoglycan leading to lysis of the bacterium; this peptidoglycan is crucial for the structural integrity of the cell wall in bacteria, particularly organisms that are Gram-positive. 8 In the synthesis of a peptidoglycan, the final stage is transpeptidation which involves transpeptidases known as penicillin binding proteins (PDPs). β -lactam antibiotics attach to these PDPs on bacteria and inhibit the transpeptidases that cross-link the peptide chains attached to the backbone of the peptidoglycan. 8 15 16 The β -lactam antibiotics are closely related to D-alanyl, the terminal amino acid of the peptidoglycan layer. 15 16 The similarity between these two structures allows for the antibiotic and the amino acid to promote their binding to the PDP. 15 The binding of the β -lactam nucleus to the residue of the PDP is irreversible, and it is this irreversible binding of the PDP that disrupts the final transpeptidation of the peptidoglycan layer and consequently inhibits bacterial cell wall synthesis. 8 15 16 The inhibition of transpeptidation due to the β -lactams causes an accumulation of peptidoglycan precursors, which initiates autolytic enzymes to lyse the excess peptidoglycan. 8 Under normal

circumstances, the peptidoglycan precursors inhibit the autolytic enzymes; however the β -lactams inactivate this and halt the process. 8

Unwanted Effects

Penicillins are mainly free from toxic effects. The main side effects are hypersensitivity reactions caused by by-products of the breakdown of penicillin, which combine with the host protein and become antigenic. 8 Skin rashes and fever are common but much more serious is acute anaphylactic shock which can be fatal in some cases. 8 When administered orally, penicillins, particularly broad-spectrum types such as amoxicillin, can disturb the bacterial flora in the gut; this can be associated with gastrointestinal (GI) disturbances. 8

Summary

VD had no complications and responded well to the treatment that she was given. It was suspected that she had community acquired pneumonia and that the treatment would be a broad-spectrum antibiotic to fight the infection. Paracetamol was also prescribed to alleviate her symptoms. As paracetamol and amoxicillin work on different receptors, there were no contraindications to her treatment. She was given medication intravenously, but once she moves onto oral amoxicillin, she must be aware of GI side effects that may occur.

Section D – Psychosocial Aspects and Public Health

Psychosocial Aspects

Although initially there may not be many psychosocial aspects to pneumonia, VD could be suffering indirectly from it. A sufferer of Sjögren's

syndrome, VD is susceptible to fatigue which can be physically and mentally exhausting. This can lead to depression, emotional stress and general lethargy. As VD is susceptible to getting infections such as pneumonia due to Sjögren's syndrome, it can also further impact on her psychologically whilst dealing with those infections as her quality of life may be significantly reduced. 17 It is important that a patient with Sjögren's syndrome can address these issues to a health professional if they are ever in distress as psychosocial factors may lead to non-compliance in their treatment.

Epidemiology

There have been many population studies that have been investigating the annual incidence rate of community acquired pneumonia (CAP). In adults, this can vary from, "... 2. 6-13. 4 per 1000 inhabitants, with somewhat higher figures in males and at the extreme ages of life." 18 19 Rates of hospitalization range between 22-51%, with annual mortality rates between 0. 1-0. 7 per 1000 patients. 20 In approximately 50% of patients with CAP, a pathogen of cause was determined. Streptococcus pneumoniae is found in 20-75% of the cases followed by Mycoplasma pneumoniae at 1-18%, Chlamydia pneumoniae at 4-19% and other viruses from 2-15%. 20 C. pneumoniae, however, has arisen as a noteworthy pulmonary pathogen in adult pneumonia patients requiring hospitalization. 20

Cost-effectiveness of Patient Care

On average there are roughly 4. 5 million visits annually to outpatient clinics, emergency departments and physician's offices due to CAP. 21 However, there has been very little in terms of studies gathering national data on the costs of CAP treatment. One study showed that there was, "...a total cost of <https://assignbuster.com/management-and-treatment-and-psychosocial-aspects-of-pneumonia-biology-essay/>

\$4.8 billion for treating patients aged ≥65 years and \$3.6 billion for treating patients aged <65 years" with an average hospital stay for a patient over 65 being \$7166 and \$6042 for patients younger than that. 21 The board and room represented the highest percentage of average hospital bills with patients who had CAP. 21 It was also noted that, " Inpatient physician service costs were \$305 million and \$192 million for the ≥65 and <65 groups, respectively. Based on 1.1 million outpatient office visits for those aged ≥65 years and 3.3 million visits for those aged <65, total outpatient costs were \$119 million and \$266 million, respectively". 21

Conclusion

VD was an elderly woman who was admitted into hospital complaining of acute chest pain and fever for the last few days. After taking a detailed history and examination from her, and with confirmation from a chest X-ray she was diagnosed with right lower lobe community acquired pneumonia. Due to the high neutrophil count in her alveolar spaces, the causative pathogen was most likely to be bacterial and so VD was promptly treated with intravenous amoxicillin, a broad spectrum β -lactam antibiotic that works by inhibiting peptidoglycan synthesis in bacterial cell walls. Paracetamol was also prescribed to alleviate her feverish and chest pain symptoms by inhibiting COX enzymes and PG synthesis in the CNS. Having been diagnosed with chronic autoimmune diseases that can lead to increased susceptibility to chest infections, this can lead to psychological issues such as depression. Recurrent admissions will also be costly to the NHS; if alternative treatments that can allow patients to be treated in an outpatient setting are possible,

then there could be significant reductions in cost, particularly for patients over 65.