Medical Emergencies

Medical emergencies are the bread and butter of emergency medicine. Whether we are discussing the patient presenting with chest pain or shortness of breath or fever, or the patient presenting with acute headache or confusion or syncope or palpitations, medical conditions head the list of acute causes that need to be identified and treated. In many cases, a failure to recognise and treat these conditions will lead to serious morbidity or mortality.

During the past decade, the ageing population together with the improved management of chronic illness has resulted in increasing patient complexity and a growing awareness of the impact of social factors on health and well-being. This not infrequently makes the assessment of these patients challenging and time intensive and is a driver for the increasing utilisation of investigations in the workup of these patients.

While economic drivers promote early discharge and more rapid decision making in relation to emergency medical practice, a considerable degree of care is required in managing medical patients. This is especially the case in patients with significant comorbidities, on multiple medications or experiencing mobility, cognitive decline, social isolation and other issues often related to aging. It is essential to maintain a holistic approach to clinical care that considers not only the patient's presenting problem and medical history but takes into account the impact of social, physical, cognitive and emotional factors. We need to remind ourselves that as clinicians we treat patients not a symptom or a disease.

Although the breadth of knowledge required to assess and manage these patients is considerable the fundamental approach to diagnosis has not changed and begins with the history and examination followed by focused use of investigations. The following chapters together with the clinical cases studies and eTutorials on LearnEM provide a strong foundation to the assessment, diagnosis and management of patients presenting to the emergency department with medical disease.

On-line Resources @ www.learnem.com.au

Clinical case studies, e-tutorials, procedural videos and clinical resources relevant to each of the 10 sections in the ABCDs of Emergency Medicine may be found on the LearnEM website and listed under headings of "Crit Care", "Emerg Med 1", "Emerg Med 2" and "Prim Care" in the top nav bar.

The CPD accredited Courses relevant to the topic of Medical Emergencies include :

1. ABCDs of Resuscitation
2. Medical Emergencies in Primary Care
3. ECG Interpretation (1) and (2). ECG in Primary Care
4. Cardiac Arrhythmias (1) and (2)
5. Bedside Blood Gases

Chapter 53
Sepsis

Key points

1. **Sepsis is defined as life threatening organ dysfunction caused by a dysregulated host response**
2. **Clinical findings of organ dysfunction include confusion, tachypnoea, hypotension, oliguria, jaundice and thrombocytopenia.**
3. **Septic shock is a subset of sepsis in which there is persisting hypotension requiring vasopressors to maintain MAP ≥ 65 or a serum lactate level > 2 mmol/L despite adequate volume resuscitation.**
4. **Management involves primary survey, interventions to support airway and ventilation, treatment of hypotension with IV fluids and vasopressors and the early administration of antibiotics.**

Sepsis is a life-threatening condition that arises when the body's response to infection injures its own tissues and organs. It is the most common lethal diagnosis presenting to the emergency department and is associated with an overall mortality of 30% !

Recognising Sepsis

Sepsis is defined as life threatening organ dysfunction caused by a dysregulated host response[1]. The definition emphasises that sepsis results from an abnormal host response to infection and is most evident clinically by the development of organ dysfunction.

Clues to organ dysfunction include :

- Impaired renal function (elevated creatinine, decreased urine output)
- Impaired liver function (elevated bilirubin)
- CNS dysfunction (altered GCS)
- Coagulation impairment (thrombocytopaenia)
- Respiratory dysfunction (increased oxygen requirement)
- Cardiovascular dysfunction (hypotension).

Although a formal assessment of organ dysfunction may be undertaken using the SOFA[2] score a more useful screening tool, validated for identifying sepsis, is the Quick SOFA Criteria. This is shown in the box below.

qSOFA criteria : A new screening tool for Sepsis

The presence of any two of the following is strongly predictive of Sepsis

- Respiratory rate > 22 /min
- Altered mentation
- Systolic BP < 100 mmHg

In practice, any patient with suspected infection should automatically trigger the search for organ dysfunction using the qSOFA criteria initially followed by a more thorough clinical assessment looking for biochemical evidence of renal or liver impairment and/or thrombocytopaenia.

[1] In 2016 revised definitions for sepsis and septic shock were published. The aim was to address the "limitations of previous definitions including the excessive focus on inflammation and the misleading model that sepsis follows a continuum through severe sepsis to shock and the inadequate specificity and sensitivity of the systemic response syndrome (SIRS) criteria". Singer et al, "The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis - 3)" JAMA. 2016; 315(8):801 - 810. These new definitions and the associated revised understanding of Sepsis and Septic Shock have been incorporated into this chapter.

[2] Sequential (Sepsis-related) Organ Failure Assessment Score

As the presence of sepsis substantially increases the risk for ICU care or death, early recognition and treatment are critical and it follows that :

- Any infection with evidence of organ dysfunction should lead to a presumptive diagnosis of sepsis.
- In a patient with evidence of organ dysfunction consideration should be given to the possibility of occult sepsis and trigger a careful search for infection.

Presenting symptoms in early sepsis, especially in the elderly, are often non-specific and include confusion, lethargy, nausea, fever and rigors. Elderly patients are at high risk for sepsis and delayed diagnosis is common. A clinical approach that assumes sepsis and has a low threshold for initiating treatment and investigation is advised in the elderly patient with nonspecific symptoms or possible infection.

Septic Shock

Septic shock is a subset of sepsis in which the circulatory and cellular metabolic abnormalities associated with sepsis are severe and as a consequence greatly increase patient mortality.

Patients with septic shock present with the clinical features of sepsis and have <u>one</u> of the following

- Persisting hypotension requiring vasopressors to maintain MAP $\geq$ 65
- Serum lactate level > 2 mmol/l despite adequate volume resuscitation.

Patients with these clinical findings have a hospital mortality exceeding 40%.

Investigations

In the patient with suspected sepsis investigations will generally include :

- Blood Cultures
- Complete Blood Picture (FBE)
- Electrolytes / Liver Functions tests
- Coagulation testing
- Serum Lactate

Imaging and microbiology testing will be guided by the clinical presentation and may include CXR, CT scan, Ultrasound and Microscopy / Culture of urine, faeces, CSF, joint aspirate, pleural fluid, vaginal discharge or skin abscess / wounds.

Serum Lactate

An elevated lactate identifies patients with sepsis who are at significantly increased risk of death and in whom aggressive fluid resuscitation +/- inotropes are required urgently to prevent deterioration into septic shock.

Normal lactate levels are less than 1.0 mmol/l in both arterial and venous blood. Elevated lactate is a predictor for increased morbidity and mortality. A level above 4 mmol/l is associated with a mortality rate of close to 30% compared with 7% for patients with a lactate between 2.5 - 4.0 mmol/l.

Lactate is produced by anaerobic cell metabolism. It is produced in large amounts in the setting of inadequate tissue oxygen delivery (eg hypovolaemic shock), conditions associated with increased oxygen requirement (eg hyperthermia, seizures) and by some drugs (metformin, phenformin, salbutamol, HIV medications).

It has recently been shown that in the setting of sepsis, elevated lactate is not caused by anaerobic cell metabolism and does not reflect tissue hypoperfusion as was previously believed. The elevated lactate results instead, from high levels of (endogenous) adrenaline that stimulate the beta-2 receptors on cells triggering biochemical changes in the cell that produce large amounts of pyruvate that is then converted to lactate.

Lactate therefore, is a marker of excessive endogenous catecholamines produced by the body working hard to maintain blood pressure in response to sepsis. Septic patients with an elevated lactate may have normal vital signs but be precariously balanced on the precipice of septic shock (organ dysfunction and death). The normal vital signs provide a false sense of security and serve to mask the severe catecholamine-dependent shock state associated with sepsis, a state that is referred to as occult or cryptogenic shock[3].

A serum lactate should be performed in patients with suspected sepsis. A venous or arterial sample may be used with bedside testing or sent to the lab in a grey (fluoride oxalate) tube. Where the level is > 4 mmol/l in adults (or > 2 mmol/l in children) immediate fluid +/- inotropic resuscitation should be commenced.

[3] Although most often associated with sepsis, occult or cryptogenic shock with an elevated lactate may be seen with any cause of shock.

Management

Management of the septic patient begins with *Primary Survey* and *Resuscitation*. Treatment aims to ensure a clear airway and provide adequate ventilation and oxygenation.

In the critically ill patient early intubation and mechanical ventilation are often required. The circulation should be assessed and fluid boluses of normal saline administered to restore intravascular volume. IV access should be obtained and blood taken for haematology, biochemistry, lactate and blood culture. Empirical antibiotics should be administered. Close monitoring is critical and will include cardiac monitoring, oximetry and monitoring of the mental state and urinary output. In advanced sepsis, central venous access and arterial line placement will be required.

Managing Circulatory Shock

The patient with severe sepsis or septic shock will be severely fluid depleted due to loss of fluid to the tissues from "leaky" vessels. Early fluid replacement is essential to restore organ perfusion. Normal saline is used for fluid resuscitation and administered in boluses of 10 – 20 ml/kg monitoring the response to each bolus. Patients will often require 30 ml/kg 0.9% normal saline during the first few hours of resuscitation. The aim of fluid resuscitation is to maintain a systolic blood pressure > 90 mm Hg or if an arterial line and CVC have been placed to maintain a mean arterial pressure (MAP) > 65 mm Hg and a central venous pressure (CVP) of 8 – 12 cm H_2O. Serum lactate is increasingly used as a marker of tissue hypoperfusion and trends in the lactate level may be used to determine adequacy of resuscitation.

Vasopressors are often needed and are commenced once the intravascular fluid volume has been restored. Noradrenaline is the recommended inotropic agent in sepsis beginning at a dose of 3 µg/min and titrating the infusion to achieve a mean arterial pressure MAP of 65 mm Hg. There is no well-established maximal dose. Adverse effects include cardiac arrhythmias, severe hypertension and tissue necrosis due to extravasation.

Noradrenaline Infusion

• Preparation	6 mg in 100 ml 5% dextrose or normal saline
• Concentration	The above mixture gives a concentration of 60 µg/ml
• Infusion rate	Begin at 3 µg / min (equivalent to infusion rate of 3 ml/hr)
• Dose Titration	No defined upper dose. Titrate to MAP > 65 mmHg
• Administration	May be given via peripheral IV for short period. Continuing use requires CVC line

Inotropes may be safely commenced through a peripheral IV line (placed in a large vein in the cubital fossa) for a period of at least 4 hours[4]. Check to ensure the cannula is functioning before commencing the infusion and place the IV cannula in as large a vein as possible. Monitor the infusion site closely for extravasation. Continuous cardiac and BP monitoring will be required. Ideally an arterial line should be placed, however where this is not feasible frequent NIBP monitoring is a short-term alternative approach.

Antibiotics and control of possible source

After completing the primary survey and initiating fluid resuscitation, consideration should be given to the administration of antibiotics. It is worthwhile undertaking a brief examination looking for localising signs that will assist the selection of antibiotics however where the source is not obvious empirical antibiotics should be commenced after drawing blood cultures. If an IDC has been inserted a urine specimen should be taken.

Empirical therapy[5] for sepsis in the immunocompetent adult with no source of infection :

- Flucloxacillin (2 gram IV 4 hourly)

  and

- Gentamicin 4 - 7 mg/kg IV for first dose[6] (Further doses are based on renal function)

This therapy is intended for 24 – 48 hours only and should be modified as soon as laboratory results indicate the pathogen and sensitivities. For patients with penicillin hypersensitivity (excluding immediate hypersensitivity) replace Di/Flucloxacillin with Cephazolin (2 gram IV 6 hourly). In patients with immediate hypersensitivity use Vancomycin (25 - 30 mg/kg IV as a loading dose) in place of Di/Flucloxacillin. In addition to antibiotics a search should be made for a focus of infection such as an abscess that can be drained and foreign bodies such as IDC or PIC lines that can be removed.

[4] Loubani OM. 2015, Cardenas-Garcia J. 2015
[5] Antibiotic Guidelines, Version 15, 2014, Therapeutic Guidelines Ltd. West Melbourne Victoria. http://www.tg.org.au/
[6] Use a lower dose of Gentamicin of 4 - 5 mg/kg in the patient with renal impairment (CrCL < 60 ml/min)

Selection of Antibiotics for Sepsis in Adults based on the likely Source of the Infection[2]

Suspected Meningitis : Give Dexamethasone 10 mg IV 6 hourly + IV Ceftriaxone 2 gm 12 hourly

- In the seriously ill patient, add IV Vancomycin 25 - 30 mg/kg loading dose
- If Anaphylactic Penicillin allergy - Use either Moxifloxacin 400 mg IV daily (as a single agent)

  or Ciprofloxacin 400 mg IV 8 hourly + Vancomycin 25 - 30 gm IV (loading dose)

Suspected Pneumonia : Give IV Ceftriaxone 1 gm 12 hourly + IV Azithromycin 500 mg daily

- If Anaphylactic Penicillin allergy – Use IV Moxifloxacin 400 mg daily + IV Azithromycin 500 mg daily
- In Cystic fibrosis, Immunosuppressed, colonised - appropriate antibiotic based on sensitivities

Suspected Pyelonephritis : Give IV Amoxycillin 2 gm 6 hourly + IV Gentamicin 4 - 7 mg/kg[3]

- Obtain urine specimen if possible before commencing treatment
- If Penicillin allergy - Use IV Gentamicin 4 - 7 mg/kg[2] alone

Suspected Cellulitis : Give IV Flucloxacillin 2 gm 6 hourly

- If Penicillin allergy – Use IV Cephazolin 2 gm 8 hourly
- If Anaphylactic Penicillin allergy - Use IV Vancomycin 25 - 30 mg/kg **or** IV Clindamycin 600 mg 8 hourly
- If MRSA patient, use 2 antibiotics to which previous isolate was sensitive

Suspected Abdominal Sepsis (eg pancreatitis, SBO, cholecystitis, appendicitis, diverticulitis)

- Give IV Amoxycillin 2 gm 6 hourly + IV Gentamicin 4 - 7 mg/kg[3] + IV Metronidazole 500 mg 12 hourly
- If Penicillin allergy - Use Ceftriaxone 1 gm IV daily + IV Metronidazole 500 mg 12 hourly
- If Anaphylactic Penicillin allergy - Gentamicin 4 - 7 mg/kg[3] + Clindamycin 600 mg IV 8 hourly

Febrile Neutropenia

Urgent administration of broad spectrum antibiotics is required in febrile patients who are or are suspected to be neutropenic with neutrophils $< 0.5 \times 10^9$/L and fever $\geq 38^{o}$ C. Neutropenic patients with sepsis or even severe sepsis may not have a fever, particularly elderly patients or those taking steroids.

Draw blood cultures before administering antibiotics. The standard of care is for the administration of antibiotics within 60 minutes of ED presentation / ward review or within 30 minutes is there are signs of severe sepsis / septic shock.

Suggested regimes for empirical therapy for suspected Febrile Neutropenia[7]

- Piperacillin + Tazobactam 4 + 0.5 gram IV 6 hourly

  or
- Ceftazidime 2 gm IV 8 hourly

  or
- Cefepime 2 gm IV 8 hourly

For patients with immediate hypersensitivity to penicillin seek speciality advice

For critically ill patients with sepsis or septic shock, consider giving the 6 hourly dose of Piperacillin + Tazobactam as an extended infusion over 3 - 4 hours, because this increases the percentage time above minimum inhibitory concentration (MIC) and may achieve better outcomes. If resistance to above antibiotics is a risk or suspected based on local microbiological data or the patient is critically ill with severe sepsis or septic shock, Gentamicin (4 - 7 mg/kg loading dose[8]) should be added to the above regime.

[7] Antibiotic Guidelines, Version 15, 2014, Therapeutic Guidelines Ltd. West Melbourne Victoria. http://www.tg.org.au/

[8] Use a lower dose of Gentamicin of 4 - 5 mg/kg in the patient with renal impairment (CrCL < 60 ml/min) or in patients who are **not** critically ill with severe sepsis or septic shock.

Chapter 54
Common Infectious Disease

Key points

1. **Cellulitis is most often caused by Group A Streptococci and Staph aureus. Management involves local treatment, rest and elevation and antibiotics.**
2. **Urinary tract infections are more common in women. The majority of uncomplicated UTI's are caused by E. Coli and Staph. saprophyticus. Most UTI's can be managed with oral antibiotics. The commonest complication is pyelonephritis characterised by loin/flank pain, high fever and raised WCC.**
3. **Diarrhoea due gastroenteritis is most often due to viruses, noninvasive bacteria or protozoa. An invasive bacterial cause should be suspected in the patient with severe illness, high fever or bloody stools. In most cases, the illness is short and self-limited. Treatment is focused on fluid replacement and symptomatic treatment.**

For a discussion on Pneumonia, Meningitis and ENT infections refer to the chapters entitled Community Acquired Pneumonia, Bacterial Meningitis and ENT Emergencies

Cellulitis

Cellulitis is an infection involving the dermis and subcutaneous tissues of the skin. The typical clinical finding is a diffuse area of erythema that is warm and tender to touch and has developed over a period of several days. The commonest sites are the face and the lower extremities.

Cellulitis is common in the elderly and may progress rapidly to severe sepsis. In the elderly patient cellulitis often presents with nonspecific symptoms such as confusion, dizziness or weakness and the diagnosis may be overlooked unless the patient is carefully examined. Areas where cellulitis may be easily missed include the legs, lower back and perineum.

Presentation

The patient presents with a diffuse spreading area of red skin that is warm and commonly painful. In severe cases the patient may have systemic symptoms of illness including malaise and fever.

On examination there is a well-demarcated area of erythema that is tender to touch and regional lymphadenopathy may be present. There may be an obvious break, laceration, burn, puncture or pre-existing wound where bacteria have entered. Breaches in the skin barrier can be microscopic and the skin appear intact to the naked eye.

Complications occur secondary to spread through subcutaneous tissue, the lymphatics or the circulation and include abscess formation, necrotising fasciitis and sepsis. The risk of complications is greatest for patients unable to mount an effective immune response including the elderly, the immunocompromised and patients with diabetes or peripheral vascular disease.

Differential diagnosis

Differential diagnosis includes erysipelas, necrotising fasciitis, acute contact dermatitis, septic bursitis and gout. Erysipelas is a superficial infection of the skin due to Strep pyogenes and is characterised by a well demarcated (red) lesion that is raised above the level of the surrounding skin. It commonly involves the face.

Necrotising fasciitis is a life threatening infection of the deep tissues resulting in severe constant pain, bullae, skin necrosis or bruising and a "woody" hard feel to the subcutaneous tissue. There is the rapid development of severe toxicity with high fever, delirium, renal failure and death.

Causes

Cellulitis is most commonly caused by Group A Streptococci. Less commonly it is due to Staphylococcus aureus. These bacteria are part of the normal flora of the skin. In immunocompromised patients, organisms such as Pneumococcus, H. influenzae B and Mycobacteria may cause serious infection. There is an increasing prevalence of MRSA as a causative organism for cellulitis even in non-immunocompromised patients.

A particularly important cause for cellulitis is associated with lacerations or puncture wounds that occur in water such as in rivers or in the sea. In these settings aquatic organisms may cause the cellulitis and include Aeromonas hydrophila, Pseudomonas and Plesiomonas species, Vibrio species, Erysipelothrix rhusiopathiae and Mycobacterium marinum. Management of these infections requires antibiotics that cover common aquatic gram positive and gram-negative organisms.

Management[9]

Management of cellulitis consists of local treatment, rest and elevation and antibiotics. In patients without systemic symptoms or fever, oral antibiotics are generally adequate.

IV antibiotics and admission, or a period of close observation, will often be required in patients at high risk for sepsis and in patients with significant fever or systemic symptoms.

Mild Cellulitis

Recommended management for mild early cellulitis and erysipelas is oral Flucloxacillin (500 mg 6 hourly for 5 – 10 days). Oral Cephalexin (500 mg 6 hourly for 5 – 10 days) is suitable for patients with penicillin hypersensitivity (excluding immediate hypersensitivity) and is an alternative to Flucloxacillin. Oral Clindamycin (450 mg 8 hourly for 5 - 10 days) should be used in patients with immediate penicillin sensitivity.

For patients discharged from the emergency department on oral antibiotics, follow up should be advised with their local general practitioner within the next 24 – 48 hours. A line may be drawn using a surgical marker around the periphery of the area of cellulitis to assist the GP to identify whether the cellulitis is resolving.

IV antibiotics are indicated in the patient who has been taking (appropriate) oral antibiotics for a period of 48 hours but in whom the area of redness is not resolving (or is continuing to spread) or in whom systemic symptoms develop or fail to resolve.

Severe Cellulitis

For patients requiring IV antibiotics recommended management is Flucloxacillin (2 g IV 6 hourly). Cephazolin (2 g IV 8 hourly) may be used for patients with penicillin hypersensitivity (excluding immediate hypersensitivity). Clindamycin (450 mg IV 8 hourly) or Vancomycin (25 - 30 mg IV loading dose) is used in the patient with immediate hypersensitivity. For home-based IV therapy (Hospital at Home) Cephazolin (2 g IV 12 hourly) is recommended.

Urinary tract infections[6]

Urinary tract infections (UTI) are common presentations to the emergency department. Women are more prone to urinary tract infections (even more so in pregnancy) than men because of their shorter urethras and proximity of the urethral orifice to the anus. Amongst the elderly however the frequency of UTI is roughly equal in women and men.

Most infections are limited to the bladder (cystitis) and do not involve the kidneys (pyelonephritis). Underlying medical, immune, structural or neurological disease can cause the body's natural defense mechanisms to fail resulting in ascending infection and pyelonephritis.

Recurrent UTI's are most commonly caused by incomplete emptying of the bladder. Urine pools within the bladder and bacteria multiply, overwhelming local defense mechanisms. UTI's are uncommon in men under 50 and often indicate a structural problem within the genitourinary system. In older men, UTI's are generally caused by prostatitis or an enlarged prostate or IDC obstructing bladder emptying.

Asymptomatic bacteriuria occurs frequently in the elderly and those with urinary catheters. Screening and treatment of asymptomatic bacteriuria is not recommended as antibiotic treatment is ineffective and encourages the emergence of resistant organisms.

[9] Antibiotic Guidelines, Version 15, 2014, Therapeutic Guidelines Ltd. West Melbourne Victoria. http://www.tg.org.au/

Presentation

Uncomplicated UTI typically presents with frequency and dysuria. Urgency, nocturia and suprapubic pain are also common. Occasionally macroscopic haematuria may occur in the setting of urinary tract infection and is referred to as haemorrhagic cystitis.

The presence of flank or loin pain, fever or rigors suggest pyelonephritis. In severe cases of pyelonephritis, the patient may present with signs of dehydration and appears toxic.

Investigation

In non-pregnant women with suspected UTI, empiric treatment should be commenced and urine cultures are not required. A urine specimen for culture should be collected before commencing antibiotics in pregnant women, men and in patients with recent antibiotic use, treatment failure, recurrent infection or suspected pyelonephritis.

Urine is collected using a mid-stream specimen or a specimen obtained from catheterisation. A mid-stream specimen is obtained by cleaning the peri-urethral area with soap or antiseptic, initiating urination into the toilet and collecting a sample mid-stream. The urine specimen is tested using dipstix for nitrites and leucocytes. Nitrites (from urease splitting organisms) and leucocytes are highly suggestive of a UTI. Proteinuria and microscopic haematuria are common but nonspecific for UTI.

Laboratory testing (blood picture, electrolytes and renal function tests, urine cultures) will be required in patients with suspected pyelonephritis. Patients with recurrent UTI's and pyelonephritis will often require further investigation with ultrasound and CT angiography.

Causes

UTI is more common in patients with diabetes, immunosuppressive disease or neurologic problems such as neurogenic or irritable bladder. Poor toilet habits, pregnancy and the presence of indwelling or suprapubic catheters or prostatic stents are common predisposing factors for UTI.

Most uncomplicated UTIs are caused by Escherichia coli (70% - 95%) and Staphylococcus saprophyticus (5% - 10%).

Complicated UTIs are associated with anatomical or functional abnormalities such as diabetes, neurogenic bladder, kidney stone and are caused by E coli (20% - 50%) and other gram-negative bacteria (eg Klebsiella, Proteus), enterococci and Streptococcus agalactiae (group B strep).

Management[10]

Empiric antibiotic management can be expected to cure between 80% - 90% of uncomplicated UTI's. Antibiotic treatment is assisted by encouraging high fluid intake and complete bladder emptying with double voiding especially at night. Urinary alkalinisation (e.g. Ural) may relieve symptoms especially if dysuria is severe.

Uncomplicated UTI

Recommended antibiotics for uncomplicated UTI in women are oral Trimethoprim (300 mg daily for 3 days) or Cephalexin (500 mg 12 hourly for 5 days) or Augmentin (500/125mg 12 hourly for 5 days) or Nitrofurantoin (100 mg 12 hourly for 5 days). In resistant cases, Norfloxacin 400 mg 12 hourly for 3 days may be effective.

Pyelonephritis

Mild cases of pyelonephritis may be managed with a 10 - 14 day course of oral Cephalexin (500 mg 6 hourly), Augmentin (875+125 mg 12 hourly) or Trimethoprim (300 mg daily). Norfloxacin 400 mg orally 12 hourly for 7 days may be used in the case of resistance to the above agents.

In severe pyelonephritis, IV antibiotics are recommended using Amoxicillin (2 gram IV 6 hourly) plus Gentamicin (4 – 7 mg/kg[11] IV with dosing adjusted for renal function). For those with penicillin allergies Gentamicin may be used on its own. If Gentamicin is undesirable (elderly or renally impaired patient) Ceftriaxone (1 gram IV daily) may be substituted.

[10] Antibiotic Guidelines, Version 15, 2014, Therapeutic Guidelines Ltd. West Melbourne Victoria. http://www.tg.org.au/
[11] Use a lower dose of Gentamicin of 4 - 5 mg/kg in the patient with renal impairment (CrCL < 60 ml/min)

UTI in Men

UTI in men < 50 years is uncommon and when present indicates a high likelihood of abnormality of the posterior urethra, prostate or epididymis. Urethritis resulting from STD may present in similar manner to UTI and is an important differential diagnosis. The frequency of UTI increases in men over 50 due to prostatic enlargement.

All men with a confirmed first UTI should be referred for further investigation. Empiric treatment includes Trimethoprim or Cephalexin, or Augmentin or Nitrofurantoin administered for 10 days.

Asymptomatic bacteriuria

Asymptomatic UTI should not be treated. Patients with IDC and suprapubic catheters only require antibiotic therapy if they symptomatic or immunosuppressed.

Gastroenteritis

Gastroenteritis is a leading cause of morbidity and mortality worldwide. The majority of acute diarrhoea in adults in Australia results from viral infections, noninvasive bacteria and protozoa.

For patients without serious underlying illness, investigations and antibiotics are not required unless there is suspicion of an invasive bacterial cause with a history of persistent high fever, bloody diarrhoea or rigors. It is essential to exclude serious causes such as bowel obstruction, appendicitis, sepsis and diabetic ketoacidosis especially in the patient with vomiting and minimal or no diarrhoea.

Presentation

Patients present with diarrhoea alone or with any combination of the associated symptoms of nausea and vomiting, abdominal cramps, loss of appetite, fever, increased flatulence or headaches possibly indicating dehydration. Bloody stools associated with gastroenteritis are uncommon and are associated with a bacterial rather than a viral cause.

Gastroenteritis is commonly complicated by dehydration. Clinical findings include dry mucous membranes, tachycardia, reduced skin turgor and sunken eyes. In severe cases life threatening circulatory shock may develop with signs of poor peripheral perfusion (cold, pale peripheries with delayed capillary refill), altered mental state (confusion, drowsy), anuria, postural hypotension, supine hypotension and finally death.

Differential diagnosis for diarrhoea is extensive and includes inflammatory bowel disease, lactose intolerance, adverse drug reactions, thyrotoxicosis, irritable bowel disease and malabsorption syndromes. Diarrhoea may also be associated with surgical disease including appendicitis, bowel obstruction, diverticulitis, mesenteric ischaemia and bowel malignancy. A careful history and examination is required to rule out these causes. Particular care is required in patients at the extremes of age (children < 1 year and elderly) as serious surgical disease will often present atypically and delayed diagnosis is common.

Investigation

Investigations are determined by the patient's clinical state. Mild cases without evidence of dehydration and no suspicion of other contributing pathology may be managed symptomatically and require no investigation.

In patients with clinical features of moderate or severe dehydration, laboratory testing should be performed and include blood biochemistry (electrolytes, renal and liver function tests), haematology (blood count) and inflammatory markers (C-reactive protein, ESR).

Other testing will be determined by specific clinical findings. In immunocompetent patients, a stool culture is rarely necessary unless a bacterial cause is suspected. Stool cultures are indicated in patients with persistently high fever or bloody stools.

Abdominal X-rays involve high amounts of radiation and are not helpful in decision making unless there is a clinical suspicion of bowel obstruction or perforation.

Causes

Infective diarrhoea may result from several mechanisms including increased secretion or reduced absorption of fluids by mucosal cells and exudation due to inflammation of the intestinal mucosa. The mechanism depends on the causative agent. For example, enterotoxigenic E. coli produces toxins that interfere with secretory mechanisms in the bowel wall whilst Shigella causes mucosal destruction and inflammation. As most absorption of fluid occurs in the small intestine, interference of absorption in the small intestine will quickly result in diarrhoea.

Viruses are responsible for between 50 to 70% of cases of gastroenteritis with norovirus the commonest cause in adults and rotavirus in children. Other common viruses are adenovirus, parvovirus and astrovirus.

Norovirus is a small single stranded RNA virus that is highly infectious via the oro-faecal route. It is resistant to detergents, heat and cold. Incubation period for disease is 12 to 48 hours. Early symptoms are nausea, sudden onset of vomiting, moderate diarrhoea, headache, myalgia and fever. Early and frequent vomiting is characteristic of norovirus infection. Most symptoms resolve within 36 hours of onset. The only active management required is rehydration and antiemetic administration.

Bacteria account for between 15% to 20% of cases and include Salmonella, Shigella, Staphylococcus, Campylobacter jejuni, Clostridium (pseudo membranous colitis), E. Coli and Yersinia. Risk factors are consumption of improperly prepared food, reheated meat dishes, seafood and dairy products.

Parasitic causes account for between 10% to 15% of cases and include Giardia, Amoebiasis, Cryptosporidium and Cyclospora. Other causes include toxins (Staph. Aureus, V. Cholera, Enterotoxigenic E Coli), shellfish poisoning, drug related (antibiotics and laxatives) and inflammatory bowel disease.

Management

Management of the patient with gastroenteritis involves rehydration and symptomatic relief with analgesics, antispasmodics and antiemetics. Antibiotics and antidiarrhoeal agents are not required and may often exacerbate or prolong the illness.

Whenever possible, rehydration should be administered orally. In the patient with mild to moderate dehydration the proprietary oral replacement solutions such as Gastrolyte, Hydralyte or Pedialyte are recommended as they contain balanced quantities of sodium, potassium, glucose and water. The use of balanced oral replacement solutions is particularly important in the treatment of at risk patients such as infants, children, the elderly and patients with renal disease[12].

Soft drinks and fruit juices are not appropriate rehydrating fluids due their high carbohydrate content.

Intravenous rehydration is indicated in patients with clinical features of severe dehydration, serious underlying co-morbidity, suspicion of a surgical cause (eg appendicitis, bowel obstruction) or in whom oral rehydration has been unsuccessful.

If resuscitation is required, this should be done by administering 20 ml/kg boluses of 0.9% Normal Saline through large bore cannulas and assessing response. For continuing IV fluid replacement isotonic solutions such as Hartmanns or Ringer's Lactate is usually appropriate. Glucose can be administered using a 0.45%NS + 5% Dextrose solution. Additional KCl may be required using pre-prepared solutions containing potassium.

It is common for patients presenting to the emergency department with a history of severe symptoms to be managed symptomatically with antiemetics and to receive 1 – 2 litres of IV normal saline over 2 - 4 hours. Many of these patients respond well to this IV rehydration regime and are able to be discharged on oral rehydration.

Antibiotics

Most cases of bacterial diarrhoea in adults are self-limiting and do not require antibiotics. Antibiotics are indicated in severely ill patients with clinical features of septicaemia, persistent fever or bloody stools. Risk factors for severe disease include infants < 3 months old, patients who are immunosuppressed and the elderly.

Recommended agents in patients with severe illness in whom a bacterial cause is suspected include one of the following antimicrobials : Ciprofloxacin, Azithromycin or Ceftriaxone.

For a discussion on Pneumonia, Meningitis and ENT infections refer to the chapters entitled Community Acquired Pneumonia, Bacterial Meningitis and ENT Emergencies

[12] See chapter 47 on Paediatric "Gastroenteritis" for a detalied discussion of rehydration in children.

Chapter 55
Anaphylaxis

Key Points

1. **Anaphylaxis is characterised by the life threatening complications of upper airway oedema, severe bronchospasm and haemodynamic compromise.**
2. **Anaphylaxis requires a high index of suspicion and a rapid clinical assessment and decision-making process. Delay can result in the development of airway obstruction, hypoxia, shock and cardiac arrest.**
3. **Immediate management involves ensuring a patent airway, commencing high flow oxygen and administration of IM Adrenaline.**
4. **Symptomatic treatment includes IV fluids for hypotension and nebulised Adrenaline for upper airway oedema.**
5. **IM Adrenaline should be repeated after 5 minutes if there is continued upper airway symptoms, bronchospasm or hypotension.**
6. **If there continuing airway compromise, severe dyspnoea, resistant shock intravenous adrenaline will be required.**

Anaphylaxis is one of the situations in which the clinician is required to act decisively and often with a minimal amount of information. There may be little opportunity to gather background history to the event, past medical history, medications etc. A high index of suspicion and immediate intervention is essential to prevent deterioration and death.

Anaphylaxis is a clinical diagnosis that should be considered when any of the following life threatening clinical findings develop rapidly in a patient :

- Laryngeal oedema
- Bronchospasm
- Hypotension

Diseases that may mimic anaphylaxis include pulmonary embolism, foreign body airway obstruction, tension pneumothorax, acute asthma, hereditary angioedema and vasovagal syncope.

Pathophysiology

Anaphylaxis results from the degranulation of mast cells and basophils releasing mediators including Histamine, Bradykinin, Platelet Activating factor, Heparin, Protease and Leukotrienes.

These mediators produce the clinical signs and symptoms of anaphylaxis due increased vascular permeability, vasodilatation, bronchial constriction, smooth muscle contraction, increased mucous gland secretion and attraction of inflammatory cells.

Anaphylaxis is an IgE mediated phenomena and is the basis of the classic 'Type 1 Immediate Hypersensitivity reaction'. It is exemplified by allergy to beestings, penicillin and food (Shellfish, Nuts).

Anaphylactoid Reactions are non-immunologically mediated systemic reactions caused by the release of histamine and other biologic mediators. They present a clinically indistinguishable picture to anaphylaxis. Examples include reaction to radiocontrast media, aspirin and NSAIDs.

Clinical Presentation

Anaphylaxis is a severe hypersensitivity reaction characterised by cardiovascular collapse and respiratory compromise. Symptoms develop rapidly often within several minutes, (but may occur within seconds or take up to an hour to develop), after contact with the allergen. Some patients describe feeling an aura of impending disaster early in the syndrome. Patients exhibit one or more of the three life threatening clinical findings in anaphylaxis : laryngeal oedema, bronchospasm and hypotension.

Clinical features of life threatening anaphylaxis

1. Laryngeal oedema

- This often begins with a vague tightening in the throat or chest and progresses to hoarseness, changes to the voice, stridor, respiratory distress, cyanosis and complete airway obstruction

2. Bronchospasm

- Early signs include coughing and wheezing, progressing to severe respiratory distress, cyanosis and respiratory arrest

3. Hypotension

- In the early stage this is manifest by light-headedness and syncope progressing to confusion, tachycardia, cardiac arrhythmias and chest pain

Most patients (90%) will have an associated rash such as pruritus, generalised erythema, urticaria or angioedema. Gastrointestinal symptoms are common and include colicky abdominal pain, nausea, vomiting and diarrhoea. Other systems may rarely cause symptoms including genitourinary (bleeding, uterine cramps, incontinence) and the CNS (confusion, seizures, dizziness).

Clinical Course

The leading causes of death in anaphylaxis are airway obstruction and intractable hypotension. Severity of the reaction is increased by the quantity of antigen, route and rapidity of administration (IV and IM exposure > oral) and past medical history of asthma, cardiac disease and use of β blockers.

Most patients develop a *uniphasic response* ie they develop signs and symptoms of anaphylaxis within 30 to 60 minutes of exposure to the allergen and recover with appropriate therapy and remain asymptomatic. A small number of patients (3 - 20%) develop a *biphasic response* characterised by a second wave of signs and symptoms that occurs 4 to 8 hours after the initial episode. A smaller percentage will rebound at 24 to 48 hours and in a few patients anaphylaxis will be continuous and protracted lasting as long as 24 to 48 hours.

There are no tests or clinical features that help to distinguish patients at risk for a biphasic or protracted response. Regardless of response to therapy, all patients with systemic features of anaphylaxis should be observed for 4 - 8 hours for biphasic reaction.

Management of Acute Anaphylaxis

The management of anaphylaxis begins with a high index of suspicion and requires a rapid assessment and decision-making process. Delay can result in respiratory obstruction, hypoxia, shock and cardiac arrest.

Immediate management in the patient with Anaphylaxis

- Assess Airway and apply high flow oxygen
- Administer Adrenaline : 0.5 mg IM (0.01 ml/kg 1: 1000)
- Consider Adrenaline infusion in severe cases : 6 mg in 100 ml NS, give 5 to 10 ml over 10 mins in adult then commence an infusion at 5 – 10 ml/hour[13]
- Consider symptomatic treatment : IV Normal Saline 10 - 20 ml/kg for severe hypotension, Nebulised Adrenaline 5 mg for stridor or bronchospasm
- Once stabilised consider steroids (eg Prednisolone 50 mg)

[13] Where an infusion pump or syringe driver is not available and/or advanced monitoring not possible, an Adrenaline infusion may be safely commenced by placing 1 mg (1 ampoule of 1:1000) Adrenaline in 1000 mls NS and commencing the infusion at 5 ml/kg/hour (approximately 300 - 400 mls /hour in an adult) and then titrating the infusion to clinical effect.

Immediate Management

Begin by assessing the airway and applying high flow oxygen. Place the patient on continuous pulse oximetry and cardiac monitoring. Administer IM (intramuscular) Adrenaline 0.5 mg (0.5 ml 1:1000). IM Adrenaline may be repeated 5 – 10 minutely if required.

In the presence of severe symptoms unresponsive to IM Adrenaline, an intravenous adrenaline infusion will be required. Except in cardiac arrest never administer IV Adrenaline as a bolus – always use an infusion.

Adrenaline Infusion

- Preparation: 6 mg (6 ampoules of 1:1000 Adrenaline) in 100 ml Normal Saline
- Concentration: The above mixture gives a concentration of 60 μg / ml
- Infusion rate: 2 – 20 μg / min (equivalent to an infusion rate of 2 – 20 ml/hr)

Symptomatic Treatment

In the presence of bronchospasm or upper airway oedema give Nebulised Adrenaline (5 mg = 5 ml of 1: 1000) via nebuliser. Although providing (transient) relief of symptoms nebulised Adrenaline does not replace the need for IM Adrenaline to treat the systemic anaphylactic process

In the presence of hypoperfusion give a fluid bolus using 10 – 20 mls/kg Normal Saline. Repeat as required.

Steroids

Once the patient is stabilised administer Prednisolone 50 mg PO (1 mg/kg in children) or Hydrocortisone 200 mg IV (4 mg/kg in children).

In patients not responding to Adrenaline or with persistent symptoms the following therapies may be considered :

- IV / IM Glucagon (1 - 2 mg) should be considered if patient is on ß-Blockers
- Nebulised Salbutamol (5 mg) may be used for patients with persistent wheeze
- Selective vasoconstrictors : Metaraminol (2 - 10 mg) or Vasopressin (10 - 40 units) may be used in adults with persistent hypotension. (In children, metaraminol 10 micrograms/kg/dose can be used).

Observation and Discharge

Observe all patients with significant anaphylaxis for at least 4 - 8 hours. Follow up advice is extremely important. Provide instructions on how to avoid triggers, prescribe and demonstrate the use of an EPI-PEN (self-administration of adrenaline) and arrange referral for allergy testing.

Indications for Admission

- Severe or protracted anaphylaxis (e.g. required repeated doses of adrenaline or IV fluid resuscitation)
- Past history of asthma or severe/protracted anaphylaxis
- Concomitant illness (e.g. asthma, history or arrhythmia)
- Lives alone or in an area remote from medical care
- Presents for medical care late in the evening

Clinical Guideline : Allergic reactions

Low Risk
Mild skin reaction only
Eye symptoms only

→

Management
Loratadine or Cetirizine 10 mg PO.
Observe for one hour
- Deteriorating → Treat as High risk
- Not Improving → Treat as Moderate risk
- Improving → Discharge. Antihistamine 3/7 PO, Advice Re Allergen, GP letter

Moderate Risk
Severe rash
Facial swelling
Diarrhoea
Abdominal cramps

→

Management
Adrenaline 0.5 mg (0.5 ml 1:1000) IM
(Repeat in 5 minutes if no improvement)
Prednisolone 50 mg PO
Observe 6 – 8 hours in ED
- Deteriorating → Treat as High risk
- Not Improving → Admit to ward
- Improving → Discharge on 5/7 Prednisolone 50 mg PO, GP letter advising immunology referral and Medi-Alert ID

High Risk
Airway problem
Breathing problem
Circulatory Problem

→

Management
Adrenaline 0.5 mg (0.5 ml 1:1000) IM
(Repeat in 5 minutes if no improvement)
Nebulised Adrenaline (5 mg)
Airway support / Call Anaesthetic backup
IV fluids 10 - 20 ml/kg (repeat if required)
Commence Adrenaline infusion if not responding to IM Adrenaline

↓

Continuing Management
Prednisolone 50mg PO
or IV Hydrocortisone 200 mg (4 mg/kg)
Admit to ICU / HDU

←

If not improving
If on ß blockers give Glucagon 1 mg IV every 5 minutes
Consider Nebulised Salbutamol

Paediatric Doses

IM Adrenaline 1:1000 Dose : 0.01 ml/kg IM

Adrenaline Infusion : 6 mg in 100 mls NS. Commence at 0.1 ml/kg/hr and titrate to effect

Adrenaline Nebulised : 0.5 mg/kg to a maximum of 5 mg

Cetirizine : Child 1 - 2 years 2.5 mg once daily, Child 2 – 12 years < 30 kg 5 mg daily

Prednisolone : 1 mg / kg

Glucagon : < 25 kg 0.5 mg > 25 kg 1 mg

Chapter 56
Acute Asthma

Key Points

1. **Asthma is a common cause for wheezing and dyspnoea. Other causes include COPD, anaphylaxis, acute pulmonary oedema and foreign body.**
2. **RED Flags indicating a high-risk for severe asthma include a history of sudden severe exacerbations, previous ICU admissions, lack of response and/or increasing use of β agonists and the recent use of oral steroids.**
3. **Unless continuous oxygen is required for hypoxia, β agonists are best given via a spacer device using a metered dose inhaler (MDI).**
4. **Management of severe asthma includes inhaled β agonists and Ipratropium, administration of steroids and in patients not responding to inhaled therapy the use of intravenous Magnesium and/or Salbutamol.**

The patient with a history of wheezing and dyspnoea is a common presentation to general practitioners and emergency departments and most often due to asthma and chronic obstructive pulmonary disease (COPD). Other causes that need to be considered in a patient who presents with respiratory distress and wheezing include upper airway obstruction, pulmonary oedema (cardiac asthma), inhaled foreign body and anaphylaxis.

Asthma

Asthma is a clinical syndrome characterised by increased responsiveness of the tracheobronchial tree to a variety of stimuli and presents most frequently with the symptom triad of cough, wheeze and dyspnoea.

Although asthma is a commonly encountered presentation, failure to recognise the severity of the attack and under-treatment of severe illness are major contributing factors to the significant mortality resulting from asthma. It is critical for clinicians to evaluate the patient carefully and identify severe or life threatening asthma requiring urgent bronchodilator therapy and supportive care.

History

An important component in the assessment of the patient with asthma is to look for "RED flags" in the history that identify the patient at high risk for life threatening asthma.

One of the most significant RED flags that should warn of severe or life threatening asthma is a history of increasingly frequent use of bronchodilators. This occurs as a consequence of reduced symptom relief and/or rapid wearing off of the bronchodilator effects, prompting the need for more frequent doses.

Red Flags indicating a high risk for deterioration or poor response to treatment include :

- Increasing use of inhaled bronchodilators
- Lack of responsiveness to inhaled bronchodilators
- Current use of steroids or recent withdrawal from steroids (Steroids are expected to be protective from asthma)
- Past history of sudden severe exacerbations
- Prior intubation or intensive care admission
- Two or more hospitalisations for asthma in the past year
- Serious co-morbidities eg cardiovascular disease, chronic lung disease, illicit drug use

Examination

Assess degree of respiratory distress by examining the following four clinical features :

1. **Conscious state :** Impaired conscious state implies critical cerebral hypoxia and severe hypercapnoea
2. **Vital signs :** Severe tachypnoea and tachycardia are found in severe respiratory distress. Decreasing oxygen saturation provides a marker for developing hypoxia.
3. **Conversation :** In severe respiratory distress a patient is able only to speak in single words between breaths or is too breathless to speak any words.
4. **Chest examination :** Increased work of breathing is indicated by recession (sucking in) of the soft tissues in the supraclavicular, suprasternal notch, intercostal and subcostal areas and prominent contraction of the sternocleidomastoid muscles in the neck with inspiration.

Classification of Severity

The National Asthma Handbook provides a useful guide for rapidly identifying severity of asthma[14]

- **Mild / Moderate**
  Can walk and speak whole sentences in one breath.
- **Severe** - This is indicated by any of the following :
  Unable to speak in sentences
  Visibly breathless
  Increased work of breathing (soft tissue recession, use of accessory muscles)
  Oxygen saturation 90 - 94%
- **Life Threatening** - This is indicated by any of the following :
  Drowsy, collapsed or exhausted,
  Cyanotic
  Poor respiratory effort
  Oxygen saturation < 90%

Management

Mild to moderate asthma

Management of the patient with mild to moderate asthma begins with administration of Salbutamol via metered dose inhaler (MDI) using a spacer device. In adults administer 4 - 12 puffs. Each puff is given separately : ie the spacer device is loaded with one puff at a time, the patient takes 3 - 4 (normal) breaths and then breaths a couple of (normal) breaths while the spacer device is reloaded with another (single) dose from the MDI.

Reassess the patient after completing administration of Salbutamol. If not improved manage as for severe asthma

- Repeat Salbutamol (every 20 - 30 mins for the first hour)
- Add Ipratropium (8 puffs by MDI using the spacer in adults)

Commence steroids : Give Prednisolone 50 mg daily for 3 - 5 days.

Discharge

Observe the patient for at least one hour after breathing difficulty resolves. Ensure patients are able to monitor and manage asthma at home. Provide Salbutamol MDI + Spacer device and Prednisolone for 3 - 5 days. Consider commencing inhaled steroids to prevent further episodes. Check inhaler technique and provide an interim asthma management plan. Arrange follow up.

[14] National Asthma Handbook, National Asthma Council Australia : https://www.asthmahandbook.org.au/figure/show/67

Severe / Life Threatening Asthma

Severe asthma is managed with supplemental oxygen, repeated inhaled β agonists / ipratropium and oral steroids. Respiratory support and intravenous therapy may be required in life threatening asthma.

Clinical features of a patient with severe asthma requiring urgent treatment

- Sitting upright, shoulders braced using accessory muscles, anxious, marked sweating
- Markedly abnormal vital signs : Tachycardia and Tachypnoea
- Too dyspnoeic to speak
- Altered consciousness (CNS hypoxia / Severe Hypercapnoea)
- Silent chest – absence of audible air movement on auscultation of the chest

Oxygen

Administer supplemental oxygen in patients with an Oxygen saturation < 95%. Titrate the oxygen therapy aiming for a target saturation of at least 95%. In life threatening asthma prepare equipment and call for anaesthetic support for possible airway management.

Salbutamol

Begin treatment with the administration of Salbutamol via MDI using the spacer. The administration of Salbutamol by intermittent inhalation using a MDI plus spacer is now recommended in the management of mild, moderate and moderately severe acute asthma.

Administer 12 puffs using the MDI/spacer. Each puff is given separately : ie the spacer should only be loaded with one puff at a time. Repeat salbutamol every 20 minutes (or sooner if required) for the first hour and then reassess to titrate further doses.

Delivery of Salbutamol via MDI and spacer is equally effective to nebulisation in most patients with severe acute asthma. Nebulised therapy is indicated in the patient who cannot inhale adequately or is requiring supplemental oxygen. The nebulised dose of Salbutamol in an adult is 5 mg. Administer nebulised salbutamol every 20 minutes for three doses and review to determine continuing therapy.

In life-threatening asthma continuous salbutamol nebs may be administered and the salbutamol dose doubled to 10 mg (2 x 5 mg nebules).

Ipratropium

In severe asthma Ipratropium bromide should be given in addition to Salbutamol. Administer 8 puffs by MDI via spacer every 20 minutes for three doses and then reduce the frequency of administration to every 4 - 6 hours. If using nebulised therapy, administer ipratropium bromide in a dose of 0.5 mg. Give three doses every 20 minutes for the first hour then every 4 - 6 hours.

Oral Steroids

Steroids are critical to the treatment of severe asthma and should be administered orally where possible. Give an initial dose of Prednisolone 50 mg and continue for 3 - 5 days. For short courses (< 7 days) there is no need to taper the dose. For patients unable to tolerate fluids, give IV hydrocortisone 100 mg 6 hourly.

Ventilation

Non-invasive ventilation may be considered in the patient who is becoming tired / exhausted or developing signs of respiratory failure. While NIV has not been shown to reduce risk of death or the need for intubation it may reduce hospital admissions, length of hospital and ICU stay. Intubation and mechanical ventilation is indicated in patients with respiratory arrest, acute respiratory failure that does not respond to treatment, severe exhaustion with impending respiratory arrest or failure of non-invasive ventilation.

Hospital Admission

Indications for Admission include

- Severe / Life threatening Asthma
- Moderate asthma in whom breathing difficulty persists despite ≥ 2 doses of β-agonists +/- Ipratropium
- Patients unable to maintain improvement after treatment with Salbutamol for at least 2 hours

Asthma Not Responding to Bronchodilators

For patients with severe or life threatening asthma that is not responding to inhaled bronchodilator therapy, the next step in management is intravenous treatment.

There are two currently recommended options for IV therapy in the adult : Magnesium and Salbutamol.

IV Magnesium sulfate

- Administer 10 mmol diluted in 50 - 100 ml normal saline or 5% dextrose over 20 minutes.
- Do not use in children < 2 years

IV Magnesium is commonly used in the treatment of life threatening asthma. Magnesium relaxes smooth muscle and it is theorised that this may result in a bronchodilatory effect. Adverse effects include facial warmth, flushing, tingling, nausea and mild hypotension. It should not be used in children < 2 years.

The recommended dose is 10 mmol diluted in normal saline or 5% dextrose and administered over 20 minutes. It may be continued as an infusion.

IV Salbutamol (Use with caution)

- Use only in critical care environments
- Add 5 mg to 50 ml normal saline (makes up a 100 ug/ml solution)
- Administer an initial 200 ug (2 ml) bolus over 1 minute and continue as an infusion beginning at 5 ug/minute (3 ml/hr) titrating up to 10 - 20 ug/min over 15 - 30 minutes if required.
- Reduce dose in the elderly and patients with impaired renal function. Monitor electrolytes, heart rate and blood lactate (see below)

There is limited evidence of benefit for the use of IV Salbutamol in severe asthma. A major issue with the use of IV Salbutamol is related to the risk for causing cardiac toxicity including tachycardia and arrhythmias.

Other significant adverse effects include hypokalaemia, hyperglycaemia and lactic acidosis. Lactic acidosis is seen in more than 70% of patients within 2 - 4 hours and aggravates underlying respiratory acidosis. The lactic acidosis however resolves within 4 - 6 hours of the infusion being reduced or ceased.

Hypotension is a common problem and results from vasodilation of muscle vessels from ß receptor stimulation. Continuous cardiac monitoring, monitoring of the blood pressure and access to resuscitation equipment is essential.

Salbutamol is administered as an infusion in a loading dose of 200 ug over one minute followed by an infusion of 5 ug/min titrated up to 10 - 20 ug/min. Beware of the risk for salbutamol toxicity - tachycardia, tachypnoea and metabolic acidosis. Consider stopping / reducing Salbutamol as a trial if these adverse effects are encountered.

Other Agents ?

Does Adrenaline have a role in Asthma ?

- Systemic adrenaline administered via the intramuscular route or intravenous infusion is indicated for patients with anaphylaxis and angioedema, however current evidence does not support its routine use in the management of acute asthma in the absence of anaphylaxis[2].
- Nebulised adrenaline does not have a significant benefit over salbutamol or terbutaline in the management of moderate-to-severe acute asthma in adults and children[15].

Is there a role for Ketamine in Asthma ?

- Evidence is lacking to support the therapeutic benefit of Ketamine in the management of acute asthma[2].
- Ketamine is often the preferred option for sedation of patients requiring intubation for respiratory failure caused by acute asthma because it stimulates the release of catecholamines and may contribute to bronchodilation through direct relaxation effect on bronchial smooth muscle[2].

[15] National Asthma Council Australia : https://www.asthmahandbook.org.au/acute-asthma/clinical/add-on-treatment

Clinical Guideline : Management of Asthma

Mild - Moderate

Normal Mental State
Normal activity
Talks normally
SaO2 > 95% (Air)

→

Management

Administer Salbutamol via MDI spacer
Review after 20 - 60 minutes

- Poor response → treat as severe
- Good response → discharge after observation. Prescribe Salbutamol +/- Steroids (3 - 5 days)

Severe

Unable to speak sentences
Visibly breathless
Increased work of breathing
SaO2 90 - 94% on (Air)

→

Management

Commence oxygen (aim for Sat > 95%)
Give 3 doses Salbutamol MDIspacer q20min
Give 3 doses Ipratropium MDIspacer q20min
Give Prednisolone
Review at one hour.

- Breathing improves. Review frequency of salbutamol. Refer for admission for close observation and titration of Salbutamol
- If no improvement or deteriorating :
  Treat as for life threatening (table below)
  Seek urgent specialist advice
  Arrange retrieval / transfer to ICU

Life Threatening

Drowsy, collapsed, exhausted
Cyanotic
Poor respiratory effort
SaO2 < 90% on (Air)

→

Management

Commence High flow Oxygen
Give continuous nebulised Salbutamol
Give 3 doses Ipratropium via neb q20min
Give Prednisolone (or Hydrocortisone)
If not improving commence IV therapy
  IV Magnesium
  IV Salbutamol
Consider Non-invasive ventilation
Impending collapse: Prepare for Intubation
Retrieval to ICU

Discharge

Discharge may be considered in the patient who has demonstrated a significant and sustained response to bronchodilators and is unlikely to show sudden deterioration.

Careful planning is required prior to discharge :

- Review the patient's current therapy and adequacy of symptom control
- Discuss an asthma action plan with the patient
- Advise the patient to use salbutamol MDIs for symptom control
- Commence a corticosteroid inhaler (eg Fluticasone) or review the dose if already on inhaled steroids
- Consider commencing long acting β agonist inhaler (eg Salmeterol) if not on this therapy (or using a combined inhaler eg Salmeterol/Fluticasone)
- Arrange follow up with the patient's usual GP and provide a discharge letter

Chapter 57
Chronic Obstructive Pulmonary Disease

Key Points

1. **COPD results in irreversible expiratory airflow limitation as a consequence of the loss of elastic recoil due to lung tissue destruction and increased resistance of the conducting airways.**
2. **Patients present with gradually increasing dyspnoea and reduced exercise intolerance, chronic cough, recurrent pulmonary infections and progressive respiratory failure.**
3. **The disease is characterised by intermittent exacerbations of severe dyspnoea and marked respiratory failure on a background of gradually deteriorating lung function.**
4. **Assessment focuses on determining the degree of respiratory distress and identifying conditions that may contribute to the patient's respiratory distress such as infection, pneumothorax, cardiac failure and pulmonary embolism.**
5. **Management of an acute exacerbation of chronic obstructive pulmonary disease (COPD) includes oxygen, bronchodilators, non-invasive ventilation and steroids.**

Chronic obstructive pulmonary disease (COPD) is a term used to describe 2 disease processes, chronic bronchitis and emphysema.

Chronic bronchitis is characterised by chronic inflammation involving the small airways in the lung with excessive production of mucous and fibrous thickening of the walls of the airways resulting in airway obstruction. It is most commonly associated with a history of smoking and the process may continue even after cessation of smoking. Progression of the disease is signalled by increasing hypoxemia and hypercapnia (type 2 respiratory failure), polycythaemia, pulmonary artery vasoconstriction and cor pulmonale.

Emphysema is characterised by gradual destruction of the alveoli walls and pulmonary capillary bed resulting in decreased ability to oxygenate blood and reduced cardiac output. Progression of the disease is recognised by the development of type 2 respiratory failure and peripheral tissue hypoxia resulting in pulmonary cachexia (muscle wasting and weight loss).

Assessment

COPD results in irreversible expiratory airflow limitation as a consequence of the loss of elastic recoil due to lung tissue destruction and increased resistance of the conducting airways. Patients present with gradually increasing dyspnoea and reduced exercise intolerance, chronic cough, recurrent pulmonary infections and progressive respiratory failure. The disease is characterised by intermittent exacerbations of severe dyspnoea and marked respiratory failure on a background of gradually deteriorating lung function.

Examination focuses on the assessment of respiratory distress by assessing the patient's conscious state, vital signs, ability to speak and examination of the chest for soft tissue recession and use of accessory muscles. Altered mental state occurs as a consequence of severe hypercapnia and/or hypoxia and indicates a life threatening clinical state. Other useful clinical indicators for severe distress include diaphoresis, marked tachycardia and tachypnoea, poor oxygen saturation (relative to their "normal" saturation), prominent soft tissue recession and use of accessory muscles and silent chest on auscultation.

It is important during assessment to consider the possibility of concurrent conditions that may be contributing to the deterioration in the patient's respiratory function. These include infection, pneumothorax, cardiac failure and pulmonary embolism.

Investigations

Chest X-ray is one of the most important early investigations in a patient with a history of COPD who presents with severe respiratory distress and should always be considered where the patient fails to respond to standard therapy for COPD (and other conditions need to be considered).

Blood gases are also useful in the patient with severe COPD but should be performed only after commencing oxygen and bronchodilators. They are particularly useful in severely ill patients where intubation and ventilation are being considered.

Acute Management

Management of patients presenting with an acute exacerbation of chronic obstructive pulmonary disease (COPD) includes oxygen, bronchodilators, non-invasive ventilation and steroids.

Identify and treat underlying concurrent conditions such as infection (antibiotics), cardiac failure (frusemide) and pneumothorax (tube thoracostomy). Consider the possibility of pulmonary embolism and in the rapidly deteriorating patient consider tension pneumothorax.

Oxygen

Oxygen should be commenced to relieve hypoxia. Although it has been widely believed that oxygen may trigger respiratory depression in COPD, research does not support this view.

Oxygen should be commenced in all patients with severe respiratory distress and then titrated using non-rebreather masks or nasal specs aiming for an O2 saturation of between 90% - 94% (or the patient's normal saturation if this is known).

Bronchodilators

Although only about 10 - 15% of all patients with COPD have clinically reversible disease, predicting response is impossible on presentation and all patients should be treated with aggressive bronchodilator therapy.

- Commence Nebulised Salbutamol in a dose of 5 - 10 mg administering it either continuously in severe cases or intermittently (eg every 20 minutes).
- Administer Nebulised Ipratropium bromide in a dose of 0.5 mg in three doses every 20 minutes for the first hour then every 4 - 6 hours.

In mild - moderate exacerbation Salbutamol and Ipratropium may be administered via MDI using the spacer. However in most patients nebulised therapy is required due to the need to administer oxygen and decreased ability to adequately inhale and use the spacer.

Non-invasive Ventilation

The use of non-invasive ventilation should be considered early in the patient with severe COPD well before the patient becomes tired / exhausted or develops severe hypercapnia (causing decreased conscious state).

The use of non-invasive ventilation in COPD results in more rapid clinical improvement with decreased hospital stay and is associated with lower mortality, decreased complications and lower rate of intubation. Both CPAP or BiPAP may be used in COPD. BiPAP is preferred in COPD as the additional inspiratory pressure improves ventilation and aids in removing the pCO2, correcting hypercapnia.

Oral Steroids

Steroids should be administered orally where possible. Give an initial dose of Prednisolone 50 mg. For patients unable to tolerate fluids, give IV hydrocortisone 100 mg 6 hourly.

Antibiotics

Many patients with COPD are colonised with bacteria most commonly Haemophilus influenzae, Moxarella catarrhalis and Streptococcus pneumoniae. Sputum cultures are often positive and do not indicate acute infection in the patient with an exacerbation.

It is currently recommended that antibiotic therapy should not be used unless the patient has clinical signs of infection with increased purulent sputum / volume of sputum in association with increased dyspnoea. Oral Amoxycillin 500 mg 8 hourly for 5 days or Doxycycline 200 mg for the first dose and then 100 mg daily for 5 days. In patients with fever, sepsis or CXR findings of pneumonia, management should follow the guidelines for community acquired pneumonia (discussed in the following chapter).

Mechanical Ventilation

Mechanical ventilation may be used to improve oxygenation and ventilation in the patient with acute respiratory failure due to COPD. Indications include respiratory arrest, acute respiratory failure that does not respond to treatment, severe exhaustion with impending respiratory arrest or failure of non-invasive ventilation. Endotracheal intubation will be required.

Careful consideration should be given before deciding on mechanical ventilation in the patient with end stage disease and in this context the decision should be discussed (where time allows) with the patient/family and consultants with the intensive care / retrieval service.

Disposition

Discharge may be considered in the patient with a mild exacerbation and who reports they are "feeling back to normal". The patient's home environment and social supports should factor in the decision to discharge and follow-up arrangements made with their GP.

Hospital Admission

Indications for admission in patients with COPD include

- Poor response to therapy
- Continuing respiratory distress
- Requiring oxygen (or higher levels of oxygen in patients on home oxygen)
- Poor home environment / lacking social supports
- Presence of an exacerbating condition such as pneumonia, pneumothorax or cardiac failure
- Significant comorbidities

Chapter 58
Community Acquired Pneumonia

Key Points

1. **Common presenting symptoms for community-acquired pneumonia (CAP) are productive cough, dyspnoea and fever.**
2. **In the elderly CAP commonly presents with the nonspecific systemic features of illness such as lethargy, anorexia, confusion or a fall.**
3. **Pneumococcus, H. Influenzae and Mycoplasma pneumoniae are the most common causes.**
4. **Immediate assessment is focused on assessing respiratory function and includes careful assessment of the vital signs and oxygen saturation and the administration of oxygen +/- respiratory support to correct hypoxia**
5. **Admission is indicated in patients with : tachypnoea > 30 / min, hypoxia, hypotension, acute confusion or multilobar involvement on chest X-ray**

Community acquired pneumonia is defined as pneumonia occurring in patients who have not been recently hospitalised and are not significantly immunocompromised. It is usually contracted by inhalation or aspiration of respiratory organisms. Although usually due to bacteria, other causes include atypical bacteria, viruses, fungi and parasites. In most cases, CAP is caused by a single pathogen. The elderly and patients with underlying COAD are particularly susceptible and will often require hospital admission. Most other cases can be managed as an outpatient.

Presentation

Common presenting symptoms include shortness of breath, cough productive of green or yellow sputum, pleuritic chest pain and fever accompanied by sweating and chills. Less commonly patients may present with systemic features of illness such as headache, fatigue, arthralgia, myalgia, nausea, vomiting and diarrhoea.

CAP is a common cause of infection in the elderly patient. It is easily overlooked and elderly patients more commonly present with the nonspecific systemic features of illness such as lethargy, anorexia, confusion or a fall. In a small number of cases patients may present with hypothermia (rather than fever).

Examination of the chest may reveal clinical evidence of consolidation including reduced air entry, dullness to percussion, bronchial breath sounds and vocal fremitus. Occasionally a pleural effusion may be present.

Causes

The common causative agents for community-acquired pneumonia are Streptococcus pneumoniae, Haemophilus influenzae and Mycoplasma pneumoniae. Viruses account for approximately 15 - 20% of cases and include Influenza, Parainfluenza, RSV, Metapneumovirus and Adenovirus.

Less common causes include Chlamydophila (Chlamydia) pneumoniae, Moraxella catarrhalis and Legionella pneumoniae. Gram negative enteric organisms from the bowel such as Escherichia coli and Klebsiella pneumoniae are associated with CAP in the elderly, nursing home residents and those with significant co-morbidities. Pseudomonas aeuroginosa, although an uncommon cause of CAP, is difficult to treat and should be thought of in patients with bronchiectasis or those on long term steroids.

Investigations

Assessment of the patient with suspected pneumonia should begin with a careful assessment of vital signs and monitoring of the oxygen saturation. Baseline blood tests including complete blood count, electrolytes and liver function tests. A high neutrophil count with left shift may indicate a bacterial cause, a high lymphocyte count may indicate a viral cause and a high eosinophil count may indicate an unusual cause such as a parasitic infection.

Blood gas measurement is useful in patients with severe disease where the result assists management decisions such as need for transfer or retrieval, admission to intensive care or requirement for ventilation. Blood gases are particularly useful for monitoring COAD patients for CO_2 retention and the patient in whom noninvasive ventilation (CPAP or BiPAP) or mechanical ventilation are being considered.

Chest X-ray is useful for diagnosis. In patients presenting with early CAP however, there may be no abnormalities on initial X-ray and a repeat X-ray will be required in 24 hours. Chest X-ray findings may provide clues to the aetiology of the pneumonia. For example, in viral and atypical pneumonias there may be minimal consolidation or where consolidation is present the pattern is usually more often perihilar, bilateral, symmetrical and interstitial. Bacterial causes demonstrate a predominantly focal segmental or lobar pattern.

Management

Initial management of a patient with suspected pneumonia includes administration of high flow oxygen, monitoring (particularly SaO2), analgesics (for pleuritic pain), antipyretics and IV rehydration. In the patient with chronic lung disease such as COAD, bronchodilators (salbutamol and ipratropium) may be useful.

In the patient with severe disease respiratory assistance may be required with noninvasive ventilation (CPAP or BiPAP) or mechanical ventilation.

A clinical decision is required whether to admit the patient to hospital or to manage them at home. This decision will often influence the regime of antibiotics prescribed. In determining admission and the use of IV antibiotics, factors such the patient's age, co-morbidities, social circumstances, ability to tolerate oral therapy and potential requirement for oxygen should be considered.

Mild - Moderate disease

Outpatient management of CAP using oral antibiotics may be considered in patients with mild illness and in whom none of the risk factors listed below are present.

- Give oral Amoxycillin (1 gram 8 hrly) or oral Doxycycline 100 mg 12 hrly for 5 - 7 days
- Doxycycline or Clarithromycin (500 mg 12 hourly for 5 – 7 days) should be added if atypical pneumonia due to Mycoplasma, Legionella or Chlamydia is suspected

For moderate disease begin with Benzylpenicillin 1.2 g IV 6 hourly until significant improvement and then oral Amoxycillin (1 gram 8 hourly) + Doxycycline (100 mg 12 hourly) for a total (IV + oral) of 7 days[16].

Severe Disease

Red flags for the patient at risk and indicating the need for admission include :

Clinical

- Respiratory rate > 30 / minute
- Systolic blood pressure < 90 mm Hg
- Oxygen saturation < 92 %
- Acute onset confusion

Investigations

- Arterial (or venous) pH < 7.35
- Partial pressure of oxygen (PaO2) < 60 mm Hg
- Multilobar involvement on chest X-ray

The presence of any of the above indicates a high likelihood of the patient having severe disease and indicates the need for hospital admission. Urgent administration of antibiotics is required in these patients. Administer

- IV Ceftriaxone (1 gram daily) plus IV Azithromycin (500 mg daily) switching to oral antibiotics once the patient's clinical condition improves and they become afebrile and are no longer tachycardic.
- Generally antibiotic therapy for a total period of 7 days (IV and oral) is required although therapy for 14 days is recommended in pneumonia due to Legionella, Staph Aureus and Gram negative bacilli[17].

[16] Antibiotic Guidelines, Version 15, 2014, Therapeutic Guidelines Ltd. West Melbourne Victoria. http://www.tg.org.au/
[17] Antibiotic Guidelines, Version 15, 2014, Therapeutic Guidelines Ltd. West Melbourne Victoria. http://www.tg.org.au/

Chapter 59
Pneumothorax

Key Points

1. **Pneumothorax should be considered in any patient who presents with the acute onset of pleuritic chest pain and / or dyspnoea.**
2. **Tension pneumothorax is the most serious early complication and untreated leads to life threatening hypoxia and circulatory compromise. Management involves immediate decompression.**
3. **Spontaneous pneumothorax may be managed using a conservative approach or by aspiration of air.**
4. **Traumatic pneumothorax requires insertion of a large bore chest tube due to the significant risk of tension pneumothorax or haemothorax.**

Pneumothorax refers to the presence of free air in the pleural space and the diagnosis should be considered in any patient who presents with the acute onset of pleuritic chest pain and/or dyspnoea. Tension pneumothorax is the most serious early complication and untreated leads to life threatening hypoxia and circulatory compromise.

Pneumothorax may occur spontaneously or secondary to iatrogenic causes or as a consequence of trauma. Spontaneous pneumothorax may be successfully managed in most cases, by the aspiration of air from the chest. Traumatic pneumothorax requires insertion of a large bore chest tube due to the high risk of complications including tension pneumothorax or haemothorax.

Types of Pneumothorax

- *Spontaneous* : Primary or Secondary (as a complication of associated lung disease)
- *Iatrogenic* : Insertion of CVC line, Pleural tap, Mechanical ventilation
- *Traumatic* : Simple Pneumothorax, Open Pneumothorax, Haemopneumothorax

Pathophysiology

In the normal lung the negative intrapleural pressure balances the elastic recoil of the lungs. Disruption of visceral pleura / alveolar wall results in air entering the pleural space. Loss of the negative pressure results in collapse of the lung as a result of elastic recoil.

Generation of positive intrapleural pressure due to the ball-valve effect of an alveolar/pleural defect can result in the accumulation of increasing amounts of air in the pleural space resulting in tension pneumothorax. This results in hypoxia and decreased cardiac output due to compression of the ipsilateral lung and deviation of mediastinum and heart compressing the contralateral lung and great veins.

Estimating Pneumothorax size

The size of the pneumothorax can be estimated using a method described by Rhea J[18]. On the CXR divide the lung into two halves by drawing a horizontal line halfway down the thorax – there is an upper half and a lower half. Next calculate the following three measurements in "cm" – the maximum apical interpleural distance (apex of the lung to pleural apex), interpleural distance (laterally) at the midpoint of the upper half of the lung and the interpleural distance (laterally) at the midpoint of the lower half of the lung.

Add these three measurements (in cms) multiply by 10 and then divide the result by three. This gives the approximate % value of the pneumothorax : eg 4 (cm) + 2 (cm) + 1 (cm) x 10 / 3 = 70/3 = 23%.

[18] Rhea J, Deluca S, Greene R. *Determining the size of pneumothorax in the upright patient*. Radiology 144:733-736, Sept 1982.

Spontaneous Pneumothorax

Spontaneous pneumothorax may be classified as either ***Primary*** indicating no underlying lung disease or ***Secondary*** associated with underlying lung disease (eg COPD, asthma, bronchial carcinoma).

Primary

Primary spontaneous pneumothorax occurs most commonly in young, healthy, tall, lean males and is due to rupture of a (congenital) cystic lesion (bleb) on the apex of the upper lobe of the lung. It accounts for approximately two thirds of spontaneous pneumothoraces.

Risk factors include smoking, acute air pressure changes such as scuba diving and flying and a previous episode of spontaneous pneumothorax. In patients with their first pneumothorax there is a 30% chance of them having another pneumothorax. After two episodes there is 50% risk of a further pneumothorax.

The patient most often presents with the sudden onset of pleuritic chest pain and dyspnoea. Clinical findings include decreased breath sounds and hyperresonant percussion note.

Secondary

Secondary spontaneous pneumothorax is most common in patients > 50 years old and accounts for one third of spontaneous pneumothoraces. It is associated with significant mortality (16%) and is most commonly associated with obstructive airway disease (COPD, Asthma). Other less common causes include pneumonia, lung abscess, TB, lung cancer, interstitial lung disease, pulmonary infarction, and drug abuse.

It presents often as an exacerbation of the underlying disease and for this reason may be easily missed clinically. It is essential that the diagnosis is considered in all patients presenting with an exacerbation of COPD or asthma, particularly where there is minimal or no response to aggressive bronchodilator therapy or in whom cardiorespiratory arrest occurs.

Unlike primary pneumothorax, secondary pneumothorax may be associated with minimal or no pleuritic pain and the typical signs of pneumothorax are frequently absent or difficult to detect in the patient with severe obstructive airways disease. It is usually associated with less collapse on CXR and the degree of collapse may be very small and is easily missed.

Tension pneumothorax in patients with chronic lung disease may occur with only 30% - 40% collapse on X-ray. Dyspnoea however is often severe due to the pre-existing decreased respiratory reserve.

Investigation

Diagnosis is generally confirmed on CXR. X-ray findings include a sharply defined lung edge and a radiolucent band devoid of lung markings. Expiratory erect / decubitus (CXR) views may demonstrate small pneumothoraces not visible on inspiratory films.

Very small pneumothoraces may only be identified on a CT scan (generally ordered for investigation of an alternative diagnosis such as Pulmonary Embolism).

Ultrasound is more accurate than CXR for the diagnosis of pneumothorax with a sensitivity and specificity approaching 100%. The patient is placed in a supine position and a high frequency probe is placed on the most superior aspect of the chest in the mid clavicular line. Findings indicative of a pneumothorax include absence of lung sliding (confirmed by a "Barcode sign"), absence of comet tails and identification of a lung point[19].

Spontaneous Pneumothorax : Management

There are three options for managing spontaneous pneumothorax :

1. Non-invasive (conservative) approach

This approach is generally preferred in patients with small pneumothoraces < 20%.

2. Aspiration

This approach is most suited to larger pneumothoraces > 20% provided there are no contraindications.

3. Small Bore thoracostomy tube attached to a Heimlich valve

This approach is most often used in patients with severe underlying lung disease or failed aspiration.

[19] See the LearnEM online courses on Emergency Ultrasound for a detailed discussion of this topic aided by video and images.

Non-invasive Approach

The non-invasive or conservative approach involves regular review of the patient and is used most often in small spontaneous pneumothoraces < 20%. It has however been used successfully in pneumothoraces up to 100% with reported success rates of up to 90%.

The technique avoids the risks associated with invasive procedures but is limited to the patient with primary spontaneous pneumothorax without tension pneumothorax, marked dyspnoea or reduced respiratory reserve.

Procedure

The non-invasive method is suitable only in the patient who is clinically stable and demonstrates a good understanding of the possible complications. The patient must have ready access to transport and live within a reasonable distance of the hospital.

Observe the patient for a period of time (4 – 6 hours). Provided they remain stable they may be discharged and reviewed the next day with a CXR. The patient should then be reviewed every 3 - 5 days until resolved. Patients may return to their job after the first follow up CXR provided it does not involve strenuous activity.

Major disadvantages include the possibility of unrecognised tension pneumothorax developing, delays to instituting treatment in cases that fail to resolve and a reported increased risk of pleural space infection. Recurrence risk is equivalent to other treatment options.

Aspiration

The technique of aspiration may be considered for spontaneous pneumothoraces > 20%.

Contraindications include :

- Haemopneumothorax
- Tension pneumothorax or severe respiratory distress
- Bilateral pneumothorax
- Severe underlying lung disease
- Recurrent pneumothoraces

The success rate of aspiration is approximately 60% (after 1 aspiration) and 80% (after 2 or 3 aspirations). Success is not related to duration of pneumothorax, presence of underlying lung disease or smoking history. Size is only a minor determinant with pneumothorax > 50% having a slightly lower success rate.

Procedure

The procedure uses a pneumothorax aspiration kit such as Cook or Arrow. A central line catheter kit using the seldinger technique is an alternative. An anterior approach is preferred. A 50 ml syringe is used to aspirate air using a 3-way tap. A continuing air leak is suggested if either more than 3 litres of air is aspirated or if after aspiration is completed more than 300 ml can then be aspirated following a Valsalva manoeuvre or cough.

Complications are rare but may include subcutaneous emphysema, catheter blockage / kinking, lung laceration or empyema.

Small Lumen Catheters

This technique uses a small lumen (eg 12F) catheter attached to a Heimlich valve. The catheter used in the pneumothorax aspiration kit may be used replacing the three-way tap with a Heimlich valve. Use of the Heimlich valve permits the option of outpatient management of pneumothorax.

Complications are rare and treatment failures are uncommon (< 5%). Most treatment failures also fail to respond to large bore catheters. The advantages of the technique include the ease of insertion, reduced patient discomfort, possibility of administering a pleurodesing agent via the catheter, possibility of outpatient management and low rate of tube blockage.

The use of small lumen catheters has replaced the need for large bore chest tubes in the management of spontaneous pneumothorax. Large bore tubes are associated with a high rate of complications including haemorrhage, empyema, focal lung infarction, abdominal insertion, re-expansion hypotension and pulmonary oedema. In addition, they are poorly accepted by patients, mandate admission and the success rates are no greater than small lumen catheters. *The use of large bore catheters is therefore best reserved for management of haemothorax, large effusions and failed small lumen management.*

Traumatic Pneumothorax

The management of a patient with traumatic pneumothorax differs significantly from spontaneous pneumothorax. Traumatic pneumothorax is associated with a high risk of complications including tension pneumothorax, haemothorax and underlying lung injury.

All patients with traumatic pneumothorax should be admitted. Most will require insertion of a large bore tube attached to an underwater seal drain.

Tension Pneumothorax

Tension pneumothorax is the most serious immediate complication of pneumothorax and may occur as a consequence of any type of pneumothorax. It should be considered in any patient presenting with severe, life threatening respiratory distress and particularly in the patient with progressive symptoms and increasing hypoxia. It is easily missed if the clinician's attention is focused on an alternative (concomitant) lung disease such as asthma or COPD and fails to consider the possibility of tension pneumothorax.

It must be identified early and treated urgently based on clinical signs. Waiting for a CXR may prove fatal. It is important to maintain a high index of suspicion in any patient with respiratory distress, underlying lung disease, or following chest trauma.

Clinical signs of Tension Pneumothorax

- Severe respiratory distress +/- cyanosis
- Absent unilateral breath sounds + hyperresonant percussion note
- JVP elevated, Tachycardia and Hypotension (related to impaired venous return)
- Tracheal shift and displaced apex beat

Management

Management involves the immediate decompression of the pneumothorax.

In the patient with suspected tension pneumothorax immediate needle or finger thoracostomy is required. Traditionally the advice has been to insert a large bore IV cannula or 5 cm angiocatheter into the second intercostal space in the mid clavicular line. In about 50% of patients however the chest wall thickness exceeds the length of the standard IV cannula and angiocatheter resulting in a high failure rate.

Currently suggested approaches to the management of tension pneumothorax include :

1. *Placement of the needle or open thoracostomy laterally* in the 5th intercostal space, midaxillary line. Laterally the chestwall is thinner with a decreased risk of injury to the great vessels or the heart.
2. *Insertion of small bore catheters designed for aspiration of spontaneous pneumothorax* such as available in the Cook or Arrow pneumothorax kits. These are easily placed and use the seldinger technique or catheter over the needle approach to insertion, are wider in diameter and less likely to kink or obstruct.

Where this equipment is not available, a surgical incision and blunt dissection using forceps or a finger may be used to create a surgical opening in the lateral chest wall.

After the tension pneumothorax has been decompressed a thoracostomy tube should be placed.