NETS
Part of the Sydney Children's Hospitals Network

# MENINGOCOCCAL DISEASE - NETS

## PRACTICE GUIDELINE ©

## DOCUMENT SUMMARY/KEY POINTS

- Recent changes in the epidemiology in invasive meningococcal disease (IMD) include increases in disease notifications, and the emergence of serogroup W and serogroup Y disease.
- IMD predominantly presents as sepsis and meningitis with atypical clinical presentations of pneumonia and gastroenteritis, also occurring
- Vaccines for serogroup A, B, C, W and Y are used in Australia and in 2018, the quadrivalent ACWY vaccine has replaced the monovalent MenC vaccine on the national immunisation programme
- Overall the incidence of IMD in Australia is low, but this disease can be rapidly fulminant and requires proactive care
- Anaesthetic support should be sought early
- Where possible a NETS consultant will attend to support these retrievals
- The NETS ultrasound should be taken as added equipment with a skilled user
- IMD is a notifiable disease in Australia
- Consideration of chemoprophylaxis for staff is necessary in this disease
- Lumbar puncture should not be part of NETS team's actions in the context of meningococcal shock.





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| **Approved by:** | SCHN Policy Procedure and Guideline Committee | NETS Executive |
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| CHANGE SUMMARY |
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| • Changes in epidemiology of IMD in Australia with increases in MenW and MenY disease with atypical presentations reported |

| READ ACKNOWLEDGEMENT |
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| • All NETS clinical staff are to read and acknowledge they understand the contents of this guideline |

## Meningococcal Disease

Invasive meningococcal disease (IMD) is caused by infection with the bacterium *Neisseria meningitidis,* a commensal of the human nasopharynx, and an opportunistic pathogen. IMD is a rare but significant cause of morbidity and mortality in children and young people. There are at least 13 serogroups of meningococci, however the majority of disease in Australia is caused by serogroups B, C, W and Y. Historically, serogroups B and C were responsible for the majority of IMD, in Australia, however the introduction of meningococcal C vaccine in 2003 has decreased MenC IMD by 96%, and IMD due to serogroup C is now uncommon. From 2003-2013 MenB was the predominant cause of IMD but by 2016 MenW was the predominant serogroup, with the global emergence of a hypervirulent MenW strain. Clinically this strain may present atypically, as septic arthritis, pneumonia, gastrointestinal as well as sepsis and meningitis, and has a higher case fatality rate.

The diagnosis of IMD may be confirmed by blood or CSF culture or by PCR of blood (EDTA tube), or CSF. Scrapings taken from purpuric lesions and sent for gram stain and culture are less sensitive and have been largely superseded by PCR. Serological testing based on the demonstration of IgM antibody by enzyme immunoassay to the N. meningitidis outer membrane protein can also provide laboratory confirmation of disease. PCR and serology results are not affected by antibiotics if taken early in the disease. In general terms, the PCR will be affected by antibiotic treatment over time. If treatment is given and successful, the dead bacterial DNA will be detected in the short term but will degrade over time. However the serology measures the antibody response so is not affected by antibiotics.

## Rationale/Background

- To ensure early recognition and prompt management of IMD
- To have a high index of suspicion for meningococcal disease in children without the classic signs of a purpuric rash or petechiae
- To eliminate meningococci from any carrier within the network of contacts close to each index case

## Acute Management

**SIMULTANEOUS ASSESSMENT AND MANAGEMENT IS ESSENTIAL**

In general, the ABCD approach should be used, but there will need to be consideration of A and B simultaneously with C during and after the initial evaluation.

***AIRWAY***

- Give oxygen via non-rebreather mask
- If airway not patent, intubate and ventilate utilising the skills of the most senior airway specialist available
- Semi-elective intubation should be considered once 40-60mL/kg fluid boluses have been given, as there is a high risk of ongoing capillary leak with pulmonary oedema
- Ketamine 2mg/kg is the drug of choice as an induction agent in the presence of shock. It will preserve BP in the short term through SVR but it is a myocardial depressant as well. A smaller dose of 0.5-1.0mg/kg is recommended if patient is drowsy with borderline BP. Low dose midazolam 0.05 mg/kg is another option for induction sedation
- Thiopentone and propofol should be avoided as induction agents; particularly if there is evidence of shock and hypovolaemia
- Ideally central access and inotropes (low dose adrenaline) are connected prior to induction for intubation. Typically if there are any concerns with borderline haemodynamics, dopamine/ adrenaline should be running; fluid boluses of saline and arrest doses of adrenaline should be readily available.

**The rationale for the above is there is a very real risk of hypotensive shock and arrest during induction or in the 20 minutes following**.

***BREATHING***

- If airway is patent assess adequacy and efficacy of respiratory function
- Observe for signs of pulmonary oedema
- If intubated, controlled ventilation with sedation and muscle relaxation may also improve cardiac output by as much as 30%, but be aware of the myocardial depressant effects of sedative/analgesic medications

***CIRCULATION***

- Assess circulation and severity of shock – pulse pressure and presence of peripheral pulsesshould be assessed and documented. Observe for metabolic acidosis, raised serum lactate and a venous saturation helps to assess the severity of shock. Do not solely rely on capillary refill time to define the need for fluid boluses but a global assessment of the patient
- Establish intravenous access – two large bore cannulae are preferred. Consider intraosseous route for rapid central vascular access. Establish ultrasound guided central venous line as soon as practical, blood should be taken as access is achieved for blood culture, blood gas/lactate, FBC, coagulation screen, electrolytes, blood glucose, calcium, magnesium, phosphate (depending on availability of local services) and meningococcal PCR
- If shocked give 20mL/kg bolus normal saline (or colloid) and reassess
- Repeat this bolus if needed and observe closely as further fluid bolus requirement is likely. Over 100mL/kg in fluid boluses is not uncommon in the early stages of fulminant sepsis. Inotropes are usually recommended if requiring > 40mL/kg. Dopamine can be given peripherally initially or adrenaline centrally when central access available. Adrenaline should be given peripherally in dire situations acknowledging its life saving benefits balanced with the risk of extravasation injury
- The safest central access route in a (potentially) coagulopathic child is the femoral venous route
- Intra-arterial access should be established when possible
- FFP is sometimes required for anticipated or established coagulopathy. If FFP is used for fluid boluses do not give more rapidly than over 1 hour as ionised calcium may fall rapidly (binding with citrate). Monitor and aim to keep ionised calcium > 1.0 mmol/L
- 4% albumin may be used as an alternative to saline – without any proven benefit. FFP, platelets and Packed Red Cell transfusions are recommended after the initial 40mL/kg of crystalloid
- There is no evidence that colloid is better than crystalloid in the treatment of shock, although higher volumes of crystalloid may be required to sustain circulating volume
- Intractable Shock: If shock or hypotension cannot be managed with fluids and inotropes, the receiving intensivist may advise the use of steroids and /or vasopressin and ECMO maybe a consideration. If possible, a clotted serum sample of blood taken before steroids are given to allow measurement of baseline random cortisol levels, but this should not delay treatment
- Vasopressin infusion is occasionally used in irreversible shock, but evidence for its use in children is limited

***MEDICATIONS***

- Antibiotic treatment: Give 50mg/kg (maximum 2g) of cefotaxime every 6 hours or ceftriaxone every 12 hours as soon as practicable. Do not delay if cultures are difficult to obtain. Ceftriaxone may be preferred as it is the only one which has been shown to eliminate nasal carriage
- Steroids are not recommended for meningococcal septicaemia except for those with adrenal dysfunction. Dexamethasone 0.4mg/kg BD x 2 days is recommended in patients treated for undifferentiated bacterial meningitis. While there is evidence of protection from unfavourable neurological sequelae in Strep. Pneumoniae meningitis and H. Influenzae meningitis but not in N. Meningitidis meningitis, steroids would be recommended when treating undifferentiated bacterial meningitis.
- Mannitol 0.25-0.5g/kg; or hypertonic saline 3% 2-6mL/kg for signs of raised ICP. Hypertonic saline is considered safer in the scenario of haemodynamic shock with renal impairment
- Avoid hypotonic intravenous solutions, aim for a sodium of 145mmol/L

***DISABILITY***

- Rapidly assess for signs of raised intracranial pressure (ICP) and treat appropriately. Aim for normocarbia. Discuss the use of mannitol or hypertonic saline with the intensivist
- Seizures should be managed as per the NETS seizure management guideline. If seizures persist, discuss midazolam infusion with receiving intensivist. Monitor for BP when using midazolam
- Limb perfusion may be severely compromised in severe septic shock. Treatment is primarily aimed at improving cardiac output, however, limb elevation, localised warming or the use of vasodilators may help. Assess peripheral pulses (consider Doppler if available) and discuss this with the intensivist

## Infection Control

- This is mandatory in situations of suspected IMD
- Universal precautions always apply and override the following if greater precautions are stipulated
- Gloves are to be worn when handling any body fluids or secretions
- Use of surgical mask is essential when tending the patient or within 1 m of the patient when antibiotic therapy is <24 hrs
- Protective eyewear is to be used in the first 24 hours of antibiotic treatment and splash is likely eg mouth care, suctioning, intubating, or patient is coughing
- Fluid-resistant gown to be worn when tending the patient when antibiotics started less than 24 hrs ago and soiling is likely

## Equipment or Specific Requirements

- Personal protective equipment (PPE)
- Point of care blood gas machine should be carried with the team
- Terason Tablet ultrasound with skilled operator to assist with central access

## Management in Transport (Leg 2)

- Continuous monitoring and assessment is essential using ABC approach
- Continuous cardiorespiratory monitoring, noting and attending to any deviation from normal parameters
- Continue to assess and document central pulses
- Preferably continuous Invasive BP or 15 minutely non-invasive BP monitoring, aiming for age appropriate MAP – discuss targets with intensivist
- If Central Venous Pressure (CVP) being measured, discuss and agree on targets with receiving intensivist or NETS consultant
- Indwelling catheter (IDC) to monitor, measure and document urinary output hourly, aiming for urinary output > 1.0mL/kg/hr
- Neurological observations, assessing for signs of raised ICP (Cushing's triad)
- If signs of raised ICP – continue to manage as discussed with the receiving intensivist. Nurse with head midline, elevate head of stretcher 30 degrees.
- Assessment of limb perfusion
- Measure BSL and treat hypoglycaemia with dextrose boluses and consider intravenous steroids if refractory
- Continue antibiotics

## Documentation

- Time of medications commenced
- Time of appearance/ extension of rash: purpuric rash or petechiae (if any). Diagram may be helpful
- Documentation of limb perfusion, if compromised

## Complications

- Almost any complication possible, because of the severity of the illness, or the treatment involved.
- Fluid overload & pulmonary oedema.
- Cardiorespiratory collapse and profound hypotension
- Electrolyte abnormalities (any), resulting in arrhythmias, worsening cardiac output, seizures, etc.
- Purpura fulminans, hypoperfusion of extremities/limb and tissue ischaemia/necrosis
- Raised ICP leading to coning

## Staff Chemoprophylaxis

- Staff should be reassured. A patient with IMD is not an efficient transmitter of the meningococcus that is causing their illness. Even in the pre-antibiotic era, hospital-acquired meningococcal disease in medical/nursing staff was rare.
- It is the carrier who transmits the organism to the patient in the first instance, who is much more likely to transmit the meningococcus again and cause further cases.
- Respiratory droplets shed from the upper respiratory tract transmit meningococci from one person to another. Humans are the only natural hosts for meningococci and the organism dies quickly outside the human host.
- Public health follow-up focuses on identifying the subset of ‘higher-risk’ contacts who require information and clearance antibiotics and vaccination in some instances.
- For NETS staff there are usually two distinct clinical scenarios of exposure – those where the diagnosis is fairly clear on clinical grounds with a diagnostic rash and shock and those where the diagnosis is made post retrieval and the service is contacted by the Public Health Unit or the staff find out when following up the patient.
- In all diagnostically obvious cases it is imperative that staff are encouraged to use PPE to protect themselves from respiratory droplet transmission of the IMD.
- For further information refer to section 5.2 of ‘Meningococcal Infection: Infection Control Management – CHW’

  http://www.schn.health.nsw.gov.au/_policies/pdf/2011-8054.pdf

## Educational Notes

- Meningococcus is solely a human pathogen. This organism is carried in the nasopharynx in up to 50% of the population. The trigger for meningococcal disease is unknown, but there is marked seasonal variation, with higher incidence in winter months associated with outbreaks of respiratory illnesses. The disease is transmitted by droplet spread or by respiratory secretions, with an increased incidence in close personal contacts of index cases. The peak incidence of invasive disease occurs in preschool children (less than 4 years) with a second peak in adolescents (15-19 years). The most common clinical manifestation of invasive disease is meningitis, but up to 20% of patients will develop meningococcal septicaemia which is associated with high mortality
- Survivors of the acute infection may have significant morbidity, including skin loss, limb loss, deafness and neurological impairment
- Early recognition and aggressive therapy in children offers the best chance of a good outcome
- The classic signs of a purpuric rash or petechiae are often absent early in the disease and in children under 16, leg pain, cold extremities and abnormal skin colour are frequent in the first 12 hours. Clinicians must maintain a high index of suspicion to maximise early diagnosis and the best outcome
- Rapid assessment and simultaneous treatment is essential. Remember the management of airway and ventilation will improve cardiac output just as management of shock will affect ventilation
- Pulmonary oedema is likely due to capillary leak and high fluid bolus volume. High PEEP may improve oxygenation if required $FiO_2$ is high. Normal blood gases will be unachievable in severe cases
- Do not even consider doing a lumbar puncture. There are many reasons why NETS teams should avoid this, including coagulopathy, shock and the risk of raised ICP. In addition, once antibiotics have been given the information to be gained from an LP will be just as useful if done 24-48hrs later. In milder cases of meningitis without septicaemia, an LP may be performed prior to antibiotics, but such cases are less likely to require NETS transfer
- Referring staff should organise family support/social workers/notification of Public Health and antibiotic prophylaxis of contacts
- Involve family and update progress; where possible

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