Meningococcal disease in children

Meningococcal disease often begins suddenly and can prove fatal within hours. It is the most important infectious cause of death in childhood (outside the neonatal period) in the developed world. It often presents without a non-blanching rash contrary to popular belief among parents and healthcare practitioners. Early recognition and initiation of fluid and antibiotic therapy can improve the prognosis. Two cases of meningococcal disease in infants are described to give a snapshot view of how this disease may present in clinical practice.

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Keywords

meningococcal disease; fluid resuscitation; whole blood PCR; antibiotics; meningococcus serogroup B; corticosteroids; chemoprophylaxis; vaccination

Key points

Paul S.P. Meningococcal disease in children. *Infant* 2011; 7(4): 116-22.

1. Meningococcal disease is the leading infectious cause of death in children in the UK and often presents without a rash.
2. It can present as meningitis, septicaemia or a combination of both.
3. Meningococcal PCR test has improved the detection rate of meningococcal disease.
4. Aggressive fluid resuscitation and early administration of antibiotics are necessary.
5. Ciprofloxacin is currently the drug of choice for chemoprophylaxis for contacts with meningococcal disease.

Meningococcal disease is the leading infectious cause of death in early childhood in the UK and can present as a meningitis, septicaemia or a combination of the two[1]. A child with a non-blanching rash (**FIGURE 1**) is often encountered in paediatric practice and this may raise concern among the parents and health professionals alike. It should be noted that invasive meningococcal disease can present without a rash and not all non-blanching petechial rashes are caused by meningococcus[2,3]. Serogroup B meningococcus is currently the most common pathogen encountered in cases of bacterial meningitis (and septicaemia) in the UK[1,2]. Adequate fluid resuscitation and early administration of parenteral antibiotics may be associated with a favourable prognosis. This article describes two infants who presented with meningococcal disease, reviews the NICE guidelines (2010)[1] and highlights some of the current literature on meningococcal disease.

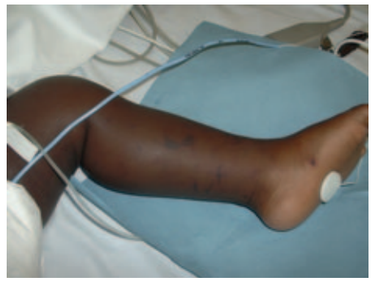

FIGURE 1 The petechial rash characteristic of meningococcal disease. Supplied and reproduced with permission from the Meningitis Research Foundation.

Case studies

Case 1 – a case of meningococcal sepsis

A healthy eight-month-old male infant presented with a high temperature up to 40°C, irritability and rash over an eight-hour period. He had tolerated about 8oz of milk earlier in the morning. He was reported to be fully immunised with no previous illnesses.

The initial assessment revealed an irritable infant with rapidly spreading non-blanching petechial rash over the body and limbs. He had a pulse rate of 194/min, temperature of 39°C, respiratory rate of 42/min, oxygen saturation of 100% in air, blood pressure of 94/44 mmHg and a central capillary refill time (CRT) of 3 seconds. A clinical diagnosis of meningococcal sepsis was made. He had a patent airway and was breathing comfortably. In view of his tachycardia and prolonged CRT, intravenous access was urgently obtained. Blood gases (**TABLE 1**) showed metabolic acidosis, bedside blood glucose was 7.7mmol/L and blood investigations including blood culture and meningococcal polymerase chain reaction (PCR) were sent.

Fluid resuscitation was started with a fluid bolus of 20mL/kg 0.9% sodium chloride. The infant remained tachycardic with a CRT of 4 seconds and was given a further fluid bolus along with a stat dose of IV cefotaxime. The infant remained in shock; an urgent anaesthetic review was requested and a discussion with the regional PICU team was initiated. Further rapid spread of his non-blanching petechial/purpuric rash was noted.

A third fluid bolus with 0.9% sodium chloride was given and a plan to intubate and ventilate the infant was made in view of refractory shock. The blood gases showed worsening of acidosis (**TABLE 1**). The infant was started on inotropic support with dopamine and was sedated with morphine and midazolam post intubation. Arterial access was obtained in the radial artery for intensive monitoring. A fourth bolus with 4.5% human albumin solution was given at 20mL/kg. The first three boluses were administered within the first hour of starting the resuscitation. A second inotrope, noradrenaline, was added later.

Time of gas	Sample type	pH	pCO_2	pO_2	Bicarbonate	BE	Comments
Prior to starting resus	Venous	7.322	4.35	3.48	16.4	-8.3	Compensated metabolic acidosis
After 2 fluid boluses	Capillary	7.324	4.54	5.34	17.2	-7.7	Compensated metabolic acidosis
90 min after starting resus, now intubated	Arterial	7.093	6.93	20.8	15.2	-13.8	Uncompensated metabolic acidosis
30 min post intubation	Arterial	7.102	6.80	23.4	14.8	-13.9	Uncompensated metabolic acidosis

TABLE 1 Blood gases from the start of resuscitation.

The blood results showed a white cell count (WCC) of 5.5x10/mm^3, C-reactive protein (CRP) of 23mg/L, prothrombin time of 14sec and corrected calcium 2.08 mmol/L. The infant was retrieved by the PICU team and remained ventilated for the next 36 hours. The blood culture was reported as negative after 48 hours; the blood PCR test was positive for Meningococcus serogroup B. Rifampicin prophylaxis was arranged for the family members.

The infant showed good improvement and after extubation was transferred to the local hospital twelve hours later. He was treated for a period of 10 days with third generation cephalosporins (cefotaxime for 72 hours, subsequently changed to IV ceftriaxone). He was well at discharge. At the clinic follow-up six weeks later he was reported to be back to his normal self. No concerns about his developmental milestones were reported and the hearing test was normal.

Case 2 – a case of meningococcal sepsis and meningitis

A healthy 10-month-old male infant was referred from primary care with an 18-hour history of mild fever, being non-specifically unwell, looking pale and floppy with cold extremeties and not feeding well. He was reported to be fully immunised with no previous illnesses.

The initial assessment revealed a floppy infant with some non-blanching petechial rash over the body and limbs. He had a pulse rate of 140/min, temperature of 37.9°C, respiratory rate of 40/min, oxygen saturation of 100% in air, blood pressure of 122/89 mmHg and a CRT of 4 seconds. A clinical diagnosis of meningococcal sepsis and circulatory shock was made. He had a patent airway and was given high flow oxygen by a face mask. In view of his tachycardia and prolonged CRT, intravenous access was urgently obtained. The blood gas (**TABLE 2**) showed metabolic acidosis, bedside blood glucose was 6.5mmol/L and blood investigations, including blood culture and meningococcal PCR were sent. Fluid resuscitation was started with a fluid bolus of 20mL/kg 0.9% sodium chloride. The infant remained tachycardic with a CRT of 3 seconds and was given a further fluid bolus along with a stat dose of IV cefotaxime.

The infant showed signs of improvement and a decision to manage him in the high dependency area was made after a discussion with the regional PICU team. No further spread of non-blanching petechial/purpuric rash was noted. He was started on IV maintenance fluids and continued on IV cefotaxime every eight hours. A urinary catheter was inserted to monitor urine output and enable strict monitoring of fluid balance. Further blood gases showed signs of recovery from the shock (**TABLE 2**). Lumbar puncture was deferred in view of his clinical condition.

Blood results showed a white cell count of 8.7x10/mm^3, and a CRP of 184 mg/L which increased to 331mg/L in the next 24 hours. In view of rising CRP values and initial clinical presentation, a lumbar puncture was performed 30 hours after the initial presentation as the clinical condition stabilised. The cerebrospinal fluid (CSF) sample was cloudy but the spinal tap was not traumatic. The blood glucose was 7.4mmol/L. The CSF analysis showed white cell count of 3510/mm^3, red cell count of 13/mm^3, glucose of 3.8mmol/L – indicative of a bacterial meningitis. The blood culture was reported as negative after 48 hours, however, the blood PCR test was positive for Meningococcus serogroup B. The CSF culture was reported as negative however the sample was obtained after starting antibiotics. Rifampicin prophylaxis was arranged for the family members.

The infant continued to improve and HDU monitoring, urinary catheter and IV fluids were discontinued after 72 hours. The infant was treated for a period of 10 days with third generation cephalosporins (cefotaxime for four days, followed by

Time of gas	Sample type	pH	pCO_2	pO_2	Bicarbonate	BE	Comments
Prior to starting resus	Capillary	7.296	5.9	6.5	20.9	-5.1	Metabolic acidosis
After 2 fluid boluses	Arterial stab	7.403	3.76	17.7	17.2	-5.7	Improvement in metabolic acidosis
7 hours after starting fluid resuscitation	Capillary	7.379	4.36	5.15	18.9	-4.8	Improving metabolic acidosis
10 hours after starting fluid resuscitation	Venous	7.36	4.75	5.02	19.8	-4.3	Normalising from metabolic acidosis

TABLE 2 Blood gases from the start of resuscitation.

ceftriaxone). He was well at discharge and at the clinic follow-up six weeks later was reported to be back normal. There were no concerns about the developmental milestones and the hearing test was reported as normal.

Discussion

Historical background

Meningococcal disease was first reported by Vieusseux in Europe (city of Geneva) in 1805 and referred it as 'fièvre cérébrale maligne non contagieuse'[4]. In 1887, Austrian pathologist Anton Weichselbaum, discovered the connection between *N. meningitides* and 'epidemic cerebrospinal meningitidis'[4]. In 1913, Flexner conducted the first trial of the meningococcal antiserum in humans with considerable success[4].

Epidemiology

In the western world, meningococcal disease is the leading cause of death from infection in children outside the neonatal period[5]. The availability of antibiotics in the last century has reduced the mortality rate from 20% to 5-6%, however the overall mortality has not fallen further since aggressive resuscitation and PICU care became the standard practice[6].

Many different serogroups of *Neisseria menigitidis* are known, however five main serogroups of meningococcal bacteria (A, B, C, W135 and Y) cause the majority of cases around the world[7]. Children <2 years of age have the highest incidence of meningococcal disease, with a second peak noted between 15 and 24 years[7]. It is to be noted that 1 in 10 people carry the meningococcal bacteria in their nasopharynx or other mucosal surfaces without actually suffering from the illness[1,2,8,9].

National mortality figures for England and Wales[9] show that in 2007 there were 38 deaths from meningococcal disease in the nought to 14 age group, and 41 deaths in the same age group in 2008. Most of these deaths were due to meningococcal septicaemia. Children under one year of age were the most vulnerable group to suffer from meningococcal disease accounting for 20 out of 75 deaths in 2007 (27%) and 19 out of 77 deaths recorded in 2008 (25%)[10].

Clinical presentation

Meningococcal disease can present in one

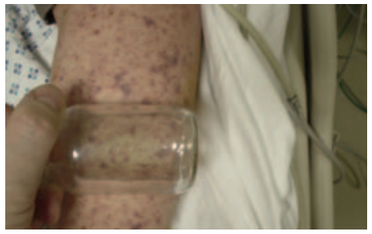

FIGURE 2 The tumbler test to identify a non blanching rash typical of meningitis. Supplied and reproduced with permission from the Meningitis Research Foundation.

of three forms: bacterial meningitis (15% of cases), meningococcal septicaemia (25% of cases), or a combination of the two (60% of cases)[1]. A non-blanching petechial rash is a not a specific marker as highlighted in an observational study in Nottingham with 197 children; only 11% of children presenting with such rashes had a confirmed diagnosis of a meningococcal disease[3]. A study in France with 123 parents highlighted that only 7% of parents were able to properly recognise a petechial rash and knew about the tumbler test[10] (**FIGURE 2**).

The common signs and symptoms that may be encountered in a child are[1,11-14]:

- Unwell looking child, toxic or moribund state
- Generalised petechial rash beyond the distribution of the superior vena cava, or spreading petechial or purpuric rash (**FIGURE 3**)
- Altered mental state or decreased consciousness
- Fever
- Headache
- Neck stiffness
- Vomiting or nausea
- Poor urine output
- Seizures
- Photophobia (as reported by older children or younger children may turn their face away from bright light)
- Teenagers may become unexpectedly combative, confused or aggressive
- Raised or tense anterior fontanelle in an infant
- Tachycardia, hypotension, central CRT >2 seconds
- Parental concern about something being not right about their child

Some non-specific symptoms worth remembering are: chills or shivering, gastroenteritis, abdominal pain or distension, sore throat, cough or other ENT symptoms, pain in extremities, hydrocephalus, etc.[2,14]

Management

A focused history and examination is necessary to establish a clinical diagnosis of meningococcal disease in the community and hospital. A strong suspicion of meningococcal disease in the community should be followed by a stat dose of parenteral benzylpenicillin and urgent transfer to the secondary care facilities[1].

Fluid resuscitation and administration of antibiotics can lead to a better outcome. The algorithm from the Meningitis Research Foundation (**FIGURE 4**) will guide practitioners in managing meningococcal disease and is depicted here as an aide memoir. A few aspects of the management which may present a dilemma for health-care practitioners are highlighted below.

Which fluid to use – colloids or crystalloids[1,6,12,14]

It is important to be aware that children may need large volumes of resuscitation fluids to restore their circulating volume; 0.9% NaCl is currently the first line used in most emergency departments. If an intravenous access is difficult, an intraosseous access should be urgently sited. Detection of signs of shock should initiate fluid resuscitation with 0.9% NaCl over 5 to 10 minutes. If shock persists a second bolus with 0.9% NaCl should be given. Persistent shock needs administration of 0.9% NaCl or 4.5% human albumin solution. If fluid boluses in excess of 40mL/kg are required, anaesthetist support is necessary for tracheal intubation and inotropic support.

Use of steroids[1,6,10,14,15]

Studies in children have found that absolute adrenal failure due to adrenal haemorrhage is rare in meningococcal disease, however, partial adrenal insufficiency has been described in children even in the absence of adrenal haemorrhage. Corticosteroids should not be used in children <3 months of age. Low dose corticosteroid viz. dexamethasone (0.15mg/kg, maximum dose 10mg) four times daily for four days may be used with the first dose of antibiotics or within the first four hours of antibiotic administration.

Which antibiotic to use[1,12,14]

It is suggested that children <3 months of age with suspected meningococcal disease should be urgently treated with IV

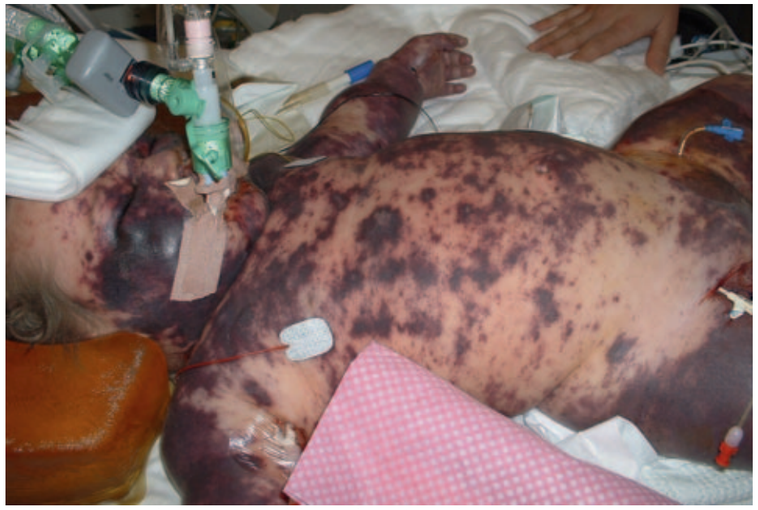

FIGURE 3 Child with meningococcal disease being treated in an intensive care unit. Supplied and reproduced with permission from the Meningitis Research Foundation.

cefotaxime. In children more than three months old IV ceftriaxone can be used, but care should be taken not to administer calcium-containing infusions at the same time (cefotaxime to be used instead). In confirmed meningococcal disease a 7 to 10 day course of IV cephalosporins is needed according to the hospital guidelines.

Maintenance fluids[1,14]

Fluid restriction is not necessary except in the presence of signs of raised intracranial pressure or syndrome of inappropriate antidiuretic hormone secretion. For correction of dehydration and/or IV maintenance fluids, isotonic fluids such as 0.9%NaCl + 5% glucose should be used. Regular monitoring of urea and electrolytes is necessary during IV fluid administration.

Blood culture or meningococcal PCR test[1,5,12,14]

In clinical practice, blood culture and whole blood meningococcal PCR are often performed. However, it should be noted that the blood culture is often reported to have no growth in paediatric practice and inflammatory markers such as raised WCC or CRP may give a more useful indication of serious pathology[16,17]. PCR testing has increased the detection rate of meningococcal disease in children.

Complications

Invasive meningococcal disease in children can have significant morbidity and families or carers of children who have survived meningococcal disease should be made aware of the potential of long-term complications. **TABLE 3** highlights what healthcare professionals should consider during the follow-up of such children[1,12,14].

A study of 115 survivors from meningococcal disease in Liverpool[18] did not find any gross neurological deficits in the survivors, when matched with similar age and sex controls, However, significant detrimental effects were noted in meningo-coccal disease survivors with regards to objective measures of motor function, cognitive ability, and behavioural issues.

A study in London[19] with 66 meningococcal disease survivors aged four to 17 years found psychiatric disorders were very common at a one year follow-up. The most commonly noted disorders were depression, oppositional defiant and anxiety disorders; these were more likely to happen in children who were severely ill such as in septic shock.

- Hearing loss (every child with meningitis should have formal audiological assessment)
- Orthopaedic complications (damage to bones and joints)
- Skin complications (including scarring from necrosis)
- Psychiatric, psychosocial and behavioural problems
- Neurological and developmental problems
- Renal failure

TABLE 3 Long-term complications associated with meningococcal disease.

Prevention

Immunisation is the key. The meningo-coccal C vaccination was introduced in the UK in 1999 and this resulted in a significant reduction in the number of cases due to meningococcus serogroup C[1,6,12]. Following the introduction of the vaccine in the UK, the incidence rate of invasive meningococcal disease per 100,000 population has decreased from 5.4 in 1999 to 2.6 in 2006[11].

The meningococcus serogroup B continues to the major pathogen for meningococcal disease in the UK accounting for nearly 89% of meningococcal infection across England and Wales in 2009[20]. Since the introduction of MeNZB (New Zealand meningococcal B vaccine) in 2004, meningococcal B epidemic has largely been abated through a combination of vaccine effect and natural waning of disease in that country[21]. A tetravalent vaccine with immunogenicity against serogroups A, C, Y and W135 was launched in the US in 2005 and is now recommended for everyone aged >11 years to 55 years.

A new tetravalent vaccine (ACWY) (Menveo, Novartis) is now available in the UK, however it is currently used as a travel vaccine for people travelling to high risk destinations such as parts of Africa and Saudi Arabia. It has been questioned whether this vaccine should be added to the immunisation schedule as a teenage booster.

It is important for practitioners to remember that about 10% of the population carries the meningococcus in their nasopharynx and other mucosal surfaces[1,2,8,22]. Although in hospital practice rifampicin is often prescribed, currently ciprofloxacin is the drug of choice for chemoprophylaxis of close contacts of a child with meningococcal disease[22].

Conclusion

Meningococcal disease can present a diagnostic dilemma, varying from an unwell child with non-specific symptoms to a child in shock. The two cases described here illustrate the importance of fluid resuscitation and supportive management, necessary in meningococcal disease, in addition to the antibiotic therapy. An early clinical diagnosis and initiation of early resuscitation is likely to be associated with a better outcome.

CASE REPORTS

Management of Meningococcal Disease in Children and Young People

Incorporates NICE Bacterial Meningitis and Meningococcal Septicaemia Guideline CG102. Distributed in partnership with NICE 7th Edition

Meningitis Research Foundation

MD1 Estimate of child's weight (1–10 years)
Weight (kg) = 2 x (age in years + 4)

MD2 Observe HR, RR, BP, perfusion, conscious level
Cardiac monitor & pulse oximetry.

Conscious Level
Alert
Responds to **V**oice
Responds to **P**ain
Unresponsive

Normal Values

Age	Heart Rate/min	Resp Rate/min
<1	110-160	30-40
1-2	100-150	25-35
2-5	95-140	25-30
5-12	80-120	20-25
Over 12	60-100	15-20

Normal systolic blood pressure = 80 + (age in years x 2)
N.B. Low BP is a pre-terminal sign in children

MD3 Take bloods for Glucose, FBC, CRP, Clotting, U&E, Ca++, Mg++, PO_4, Lactate, Blood cultures, Whole blood (EDTA) for PCR, Blood gas (bicarb, base deficit), X-match.

MD4 Intubation (call anaesthetist and consult PICU) see **BM5**
Consider using: Atropine 20 mcg/kg (max 600 mcg) AND Ketamine 1-2 mg/kg in shock or Thiopental (thiopentone) 3-5 mg/kg in RICP AND Suxamethonium 2 mg/kg (caution, high potassium). ETT size = age/4 + 4, ETT length (oral) = age/2 + 12 (use cuffed ET tube if possible). Then: Morphine (100 mcg/kg) and Midazolam (100 mcg/kg) every 30 min.
Do not use Ketamine in children with raised ICP.

MD5 Inotropes
Dopamine at 10-20 mcg/kg/min. Make up 3 x weight (kg) mg in 50 ml 5% dextrose and run at 10 ml/hr = 10 mcg/kg/min. (These dilute solutions can be used via a peripheral vein).
Start Adrenaline via a central or IO line only at 0.1 mcg/kg/min.
Start Noradrenaline via a central or IO line only at 0.1 mcg/kg/min. for 'warm shock'.
Adrenaline & Noradrenaline: Make up 300 mcg/kg in 50 ml of normal saline at 1 ml/hour = 0.1 mcg/kg/min.

MD6 Hypoglycaemia (glucose < 3 mmol/l) 5ml/kg 10% Dextrose bolus i.v.

MD7 Correction of metabolic acidosis pH < 7.2
Give half correction $NaHCO_3$ i.v.
Volume (ml) to give = (0.3 x weight in kg x base deficit ÷2) of 8.4%$NaHCO_3$ over 20 mins, or in neonates, volume (ml) to give = (0.3 x weight in kg x base deficit) of 4.2% $NaHCO_3$.

MD8 If K^+< 3.5 mmol/l
Give 0.25 mmol/kg over 30 mins i.v. with ECG monitoring.
Central line preferable. Caution if anuric.

MD9 If total Calcium < 2 mmol/l or ionized Ca^{++}< 1.0
Give 0.1 ml/kg 10% $CaCl_2$ (0.7 mmol/ml) over 30 mins i.v. (max 10 ml) or 0.3 ml/kg 10% Ca gluconate (0.22 mmol/ml) over 30 mins (max 20 ml).
Central line preferable.

MD10 If Mg^{++}< 0.75 mmol/l
Give 0.2 ml/kg of 50% $MgSO_4$ over 30 mins i.v. (max 10 ml).

MD11 Urgently **notify** public health of any suspected case of meningitis or meningococcal disease
Prophylaxis of household contacts of MD
http://www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1194947389261
- Rifampicin bd for 2 days: < 1yr 5 mg/kg; 1-12yrs 10 mg/kg ; > 12yrs 600 mg *or*
- Ceftriaxone single im dose: < 12yrs 125 mg; > 12yrs 250 mg or
- Ciprofloxacin single dose (not in children <2 or in pregnancy/breast-feeding) 2-4yrs 125mg; 5-12 yrs 250 mg ; > 12yrs 500 mg

For index case not treated with Ceftriaxone, prophylaxis when well enough.
Hib: prophylaxis may be indicated – consult public health

MD12 Antibiotics for confirmed and unconfirmed (but clinically suspected) meningococcal disease: i.v. Ceftriaxone for 7 days unless contraindicated **BM3** (see bacterial meningitis algorithm for antibiotics against other pathogens)

Based on Early Management algorithm, Dept Paediatrics, Imperial College at St Mary's Hospital as described in Arch Dis Child 1999;80:290 & 2007;92;283 & on NICE CG102
http://guidance.nice.org.uk/CG102/Guidance
http://guidance.nice.org.uk/CG102/QuickRefGuide/pdf/English
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Further copies from **www.meningitis.org** or 01454 281811.

RECOGNITION

May present with predominant **SEPTICAEMIA (with shock)**, **MENINGITIS (with raised ICP)** or **both**. **Purpuric/petechial non-blanching rash is typical**.
Some may have neither shock nor meningitis. Rash may be atypical or absent in some cases.

Bacterial meningitis algorithm www.meningitis.org

Call consultant in Emergency Medicine, Paediatrics, Anaesthesia or Intensive Care
Initial assessment looking for shock/raised ICP
Do not perform Lumbar Puncture

Give IV Ceftriaxone (80 mg/kg od) without delay
Do not use Ceftriaxone at the same time as calcium-containing solutions:
in this situation use Cefotaxime (50mg/kg qds)

SIGNS OF SHOCK? MD2

- Tachycardia
- Capillary refill time > 2 seconds
- Cold hands/feet; pale or blue skin
- Respiratory distress/ oxygen saturation <95% in air
- Altered mental state/decreased conscious level
- Decreased urine output (< 1ml/kg/hr)
- Hypotension (late sign)
- Hypoxia on arterial blood gas
- Base deficit (worse than -5 mmol/l)
- Increased lactate (>2 mmol/l)

YES → Do not perform Lumbar Puncture; Nil by mouth

- ABC & High flow Oxygen (minimum 10 l/min) by face mask
- Measure glucose.
- Insert 2 large i.v. cannulae (or intraosseous); Take bloods, see MD3

VOLUME RESUSCITATION

- Immediate bolus of 20ml/kg of 0.9% Sodium Chloride over 5-10 minutes and reassess immediately
- If shock persists immediately give second bolus of 20ml/kg of 0.9% Sodium Chloride or of 4.5% Human Albumin over 5-10 minutes and reassess immediately
- Observe closely for response/deterioration
- Consider Urinary catheter to monitor output

After **40ml/kg fluid** resuscitation
STILL SIGNS OF SHOCK?

NO → **Repeated review**

YES

WILL REQUIRE URGENT ELECTIVE INTUBATION AND VENTILATION MD4

Call anaesthetist and contact PICU

- Immediate bolus of 20ml/kg of 0.9% Sodium Chloride or 4.5% Human Albumin over 5-10 minutes and reassess immediately; Continue boluses if necessary with repeated clinical and laboratory assessments including blood gas measurements.
- Start peripheral inotropes (Dopamine); if IO access start Adrenaline MD5
- ET Tube (Cuffed if possible) and CXR
- Anticipate pulmonary oedema (ensure PEEP)
- Central venous access
- Urinary catheter to monitor output, Nasogastric tube
- Start Adrenaline infusion (central) if continuing need for Volume resuscitation & Inotropes
- For warm shock (warm peripheries, bounding pulses and low diastolic pressure) give Noradrenaline (central) or Dopamine (peripheral)
- If fluid and inotrope resistant shock discuss with paediatric intensivist.

Anticipate, monitor and correct:

- Hypoglycaemia MD6
- Acidosis MD7
- Hypokalaemia MD8
- Hypocalcaemia MD9
- Hypomagnesaemia MD10
- Anaemia
- Coagulopathy (Fresh Frozen Plasma 10ml/kg)

NO

RAISED INTRACRANIAL PRESSURE?

- Reduced (GCS ≤8) or fluctuating level of consciousness
- Relative Bradycardia and Hypertension
- Focal neurological signs
- Abnormal posture or posturing
- Seizures
- Unequal, dilated or poorly responsive pupils
- Papilloedema (late sign)
- Abnormal 'doll's eye' movements

YES → Do not perform Lumbar Puncture; Nil by mouth

- ABC and Oxygen (minimum 10 l/min) by face mask
- Measure glucose.
- Give Mannitol (0.25g/kg) bolus or 3% Saline (3ml/kg) over 5 minutes
- Treat shock if present

Call anaesthetist and contact PICU

- Intubate and ventilate to control $PaCO_2$ (4-4.5 kPa)
- Urinary catheter and monitor output, NG tube

NEUROINTENSIVE CARE

- 30° head elevation, midline position
- Avoid internal jugular lines
- Repeat Mannitol or 3% Saline if indicated
- Sedate (muscle relax for transport)
- Cautious fluid resuscitation (but correct coexisting shock)
- Monitor pupillary size and reaction
- Avoid hyperthermia
- Once patient is stabilised, consider CT scan to detect other intracranial pathology if GCS ≤8, fluctuating conscious level or focal neurological signs

Do not restrict fluids unless there is evidence of:
- raised intracranial pressure, or
- increased antidiuretic hormone secretion

STEPWISE TREATMENT OF SEIZURES

- i.v. Lorazepam (0.1 mg/kg) or Midazolam (0.1 mg/kg) bolus
- Consider Paraldehyde (0.4 ml/kg PR)
- Phenytoin (18mg/kg over 30 min i.v. with ECG monitoring)

If persistent seizures:
- Thiopental (thiopentone) 4mg/kg in intubated patients (beware of hypotension)
- Midazolam/Thiopental infusion

Perform delayed LP when stable and no contraindications

GO TO BACTERIAL MENINGITIS ALGORITHM

YES

CLINICAL FEATURES OF MENINGITIS?

NO

Close monitoring for signs of
- Raised ICP
- Shock

Perform LP if no contraindication
DO NOT DELAY ANTIBIOTICS

Repeated Review
Children with MD can deteriorate rapidly.
Deterioration detected?

Transfer to Intensive Care by Paediatric Intensive Care Retrieval Team

Notify public health, prophylaxis see MD11; Long-term management: see BM7 on Bacterial Meningitis Algorithm

FIGURE 4 Algorithm for management of meningococcal disease reproduced with permission from the Meningitis Research Foundation.

Acknowledgement

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