



# Lessons from research for doctors in training

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Recognition and early management of  
meningococcal disease in children and young people



This is an educational resource. It is not an evidence-based guideline on the management of meningococcal disease in children.

The handbook uses individual case histories as a basis for group discussion and learning. The clinical management points are based on the good practice guide "Early Management of Meningococcal Disease in Children" developed by St Mary's Hospital, London and produced by Meningitis Research Foundation.

## Lessons from research for doctors in training: recognition and early management of meningococcal disease in children and young people

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Meningococcal disease (MD) remains an important cause of mortality in children in the UK but aggressive treatment of severe cases can lead to an improvement in outcome<sup>1,2</sup>.

Recent research by the Royal College of Paediatrics and Child Health and Imperial College London, funded by Meningitis Research Foundation, looked at health care delivery for almost 500 children with MD. During this study it was seen that a few clinical errors repeatedly led to delayed or inadequate treatment of cases of MD. In some cases shock and raised intracranial pressure (RICP) associated with MD were not recognised early enough. In other cases the severity of the illness was recognised but the management was not aggressive enough and did not follow the protocol, Early Management of Meningococcal Disease in Children (see back page fold-out). Although shock and RICP are commonly seen in MD they also occur in other conditions and **it is extremely important for doctors in training to be aware of the early signs, as prompt action can save lives.**

### The aims of this handbook are to:

1. use clinical case examples to teach about the signs of septicaemia and meningitis;
2. clarify the important differences between meningococcal septicaemia and meningitis;
3. outline basic management of meningococcal septicaemia and meningitis in line with Early Management of Meningococcal Disease in Children protocol<sup>3,4</sup>;
4. describe the clinical pathophysiology of meningococcal disease.

This handbook begins with case histories based on clinical examples from the research study which illustrate how the early signs of severe MD can be missed. Examples are from a range of settings to accurately reflect where children presenting with this disease are looked after: not all cases were managed by paediatric teams. Non-clinically relevant details have been changed in order to preserve anonymity of children and doctors without obscuring the clinical teaching points these cases bring to light.

On the first page of each case study, the history is recounted in the left-hand column, accompanied by questions in the middle column to guide your learning and reflection. The third column gives references to relevant sections in the handbook to test your knowledge and understanding. On the page following each case history is the outcome for the patient and a series of discussion and learning points. We hope that these will guide individual learners and group discussions in a clinical context. The reader is also encouraged to consult the many review articles on the subject for a more in-depth understanding of pathophysiology and management of meningococcal disease.

## Case 1

### CASE HISTORY

*Child of 5 years attends casualty with sudden onset fever and painful right hand.*

**A&E Triage assessment:**

1)? Injury soft tissue 2) unwell, pyrexia.

Sudden onset pain in right hand. No history of trauma, she is reluctant to have it touched. She is also generally unwell. Spots erupting on arm and back. Last had Calpol 2.5 hours ago.

Observation taken: temp 39.9

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**2 hours later—A&E SHO assessment:**

Presenting complaint: right hand swollen and painful, hand painful for 4 hours, no history of trauma. Has been in contact with chickenpox.

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**On examination:** temp 40.1 (55 minutes after Calpol and Brufen). Small blanching spots on body. ENT / ABDO clear. No photophobia.

### QUESTIONS

What do you think of this assessment?

Is there anything else you would want to know?

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What do you think of this assessment?

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What is your differential diagnosis?

### LOOK IT UP

See page 35 – Symptoms of septicaemia.

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See page 35 – Making the diagnosis: Taking a history.

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See page 36 – Examining the patient: The rash.

See page 38 – Initial assessment of any febrile child.



### Case 1 Outcome

Diagnosis: probable early chickenpox. Child sent home with antipyretics. The child died 14 hours later of meningococcal septicaemia.

#### DISCUSSION

##### **A&E Triage assessment:**

A full set of vital signs should have been measured. The child may have had tachycardia and tachypnoea.

No description of the spots was made, which is inadequate.

The time delay between triage and SHO assessment was unacceptable.

##### **A&E SHO assessment:**

Full history not taken to seek explanation of painful hand.

Lack of response to antipyretics not taken seriously.

The girl had been in contact with chickenpox 5 days previously. Chickenpox incubation period is 10-14 days so this is an extremely unlikely diagnosis.

Although limb pain is well recognised in meningococcal septicaemia, the differential diagnosis includes osteomyelitis or septic arthritis.

It was too early in the disease process to specifically diagnose meningococcal disease while the child was in A&E. However there was sufficient cause for concern, namely an unremitting fever, a new rash, general malaise and a potential focus of infection. This child should undoubtedly have been referred to the paediatricians.

#### LEARNING POINTS

- Measure and record vital signs.
- All febrile children must be fully assessed however well they look.
  
- Don't forget the less common symptoms such as limb pain.
  
- Beware 'red herrings'.
  
- The early rash of meningococcal disease can be blanching in 30% of cases.
- Photophobia may be absent in a young child with meningitis and is not seen in pure septicaemia.

### Conclusion

This case history illustrates how an inadequate assessment of a child allowed a serious illness to be missed.



## Case 2

### CASE HISTORY

*Child 3 years old with short history of fever, shaking and generally unwell.*

**A&E Triage assessment:**

High temperature, he looks flushed, no rash, unwell child.

**Ten minutes later– A&E SHO:**

Febrile child, listless, irritable and drowsy.

Temp 39.7, HR 170, RR 55.

Pyrexial and drowsy: ? cause, refer to paediatric team.

**Two hours later:** admitted to paediatric ward.

**Nursing assessment:** Temp 38.4, HR 172, RR 45, BP 112/50.

Small pin prick rash on abdomen.

**Ward SHO reviewed child:**

Sleepy but rousable, no neck stiffness or photophobia, HR 171.

No rash but he has a few old chickenpox scars.

Chest clear.

### QUESTIONS

What are the normal ranges for these vital signs? Are there any other observations you would record?

What do you think of the timing of this admission?

What do these signs tell you?

What do you think of this assessment? Is there anything else you would want to know?

### LOOK IT UP

See page 38 Table – Normal values of vital signs.  
See page 38 – Initial assessment of any febrile child.

See page 39-40 – Clinical signs.

See page 41 – Initial laboratory assessment.  
See page 36 – Examining the patient: The rash.



### Case 2 Outcome

Diagnosis: viral URTI. Child sent home.

The child re-presented 11 hours later in uncompensated shock, with a widespread rash and died despite full resuscitation.

#### DISCUSSION

**Triage assessment:**

Appropriate in that this boy was given high priority to see the doctor.

**A&E SHO assessment:**

Abnormal vital signs were noted and need to refer to paediatricians identified. However, a full assessment would have included saturation monitoring, blood pressure and capillary refill time (CRT).

Long delay between A&E department and paediatric ward not explained in clinical notes. Such delays are totally unacceptable.

**Paediatric ward triage assessment:**

The vital signs on admission remained abnormal 2 hours after they were first recorded, indicating a problem. This is what early shock with cardio-vascular compensation looks like.

To confirm presence of shock, base excess (venous blood gas) should have been measured and urine output monitored.

A new pinprick rash was documented on the ward but was not taken seriously.

**Paediatric SHO assessment:**

Totally inadequate assessment. Still no assessment of peripheral perfusion. This doctor was looking for meningitis and missed the early signs of septicaemia. At this stage full laboratory investigations should have been done.

#### LEARNING POINTS

- Children with septicaemia often have rigors.
- Children in early stages of septicaemia may look reasonably well and remain relatively alert.
- If you assess a sick child and decide they need further assessment, it is your responsibility to ensure this happens speedily.
- Isolated pinprick spots may appear where the rash is mainly maculopapular so it is important to search the whole body for small petechiae especially in a febrile child with no focal cause. The early rash in meningococcal disease can be very diverse in appearance.
- The septicaemic rash does not necessarily develop at the same rate as the septicaemia. Always examine the child for the clinical signs of shock.
- If an experienced nurse is concerned about a child then you should be too. Take note.
- Children with signs of shock require assessment by a senior paediatrician.
- Neck stiffness and photophobia are uncommon in a young child even if they have meningitis and their absence should not be reassuring.

### Conclusion

In this case history, some clinical assessment was made. But the significance of the persistently abnormal vital signs was not understood and therefore not acted on.



## Case 3

### CASE HISTORY

*2.5 year old boy admitted with purpura and fever.*

#### **Paediatric assessment:**

Temp 39.3, Pulse 134, CRT 6 seconds, BP unrecordable, femoral pulses present but weak.

Cyanosed, Saturation 75% in air. Widespread creps.

GCS 9/15, Neck stiffness+

Purpuric rash on chest.

**Diagnosis:** Meningococcal meningitis.

#### **Treatment:**

Antibiotics intravenously.

Fluids 40 ml/kg colloid, 10 ml/kg crystalloid over 1 hour, then maintenance fluids.

Some improvement of CRT so left on the ward.

#### **Two hours after admission:**

P178, BP 112/60 RR 46.

Increasing rash, drowsy, some response to parents.

No urine output.

#### **Results:**

WCC 3.2, INR 2.2, Urea 8.3.

Chest X-ray shows pulmonary oedema.

Frusumide given and fluids slowed down. The child has had a total of 80ml/kg by now.

**SHO review:** very fast tachycardia, ? need blood gas, ? needs LP.

#### **Six hours after admission:**

HR 194, not recognising parents. Consider Mannitol infusion if CNS decreases further.

### QUESTIONS

How would you interpret these signs?

What is the normal value for oxygen saturation in air?

What does purpuric rash suggest?

Meningitis or septicaemia?

What do you think of the treatment given?

What is this child's prognosis?

Why has pulmonary oedema developed?  
How would you manage it?

What do you think of this treatment?

Is there any contraindication to a lumbar puncture in this child?

Why is this child confused?  
Why is Mannitol inappropriate in this situation?

### LOOK IT UP

See page 38 Table – Normal values of vital signs.  
See page 38 – Initial assessment of any febrile child.  
See pages 39-40 – Clinical signs.

See page 45–46 - Principles of management of meningitis and septicaemia and protocol Early Management of Meningococcal Disease in Children.

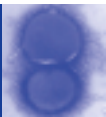
See page 34 – Clinical features of severe disease.

See Section 5 – Pathophysiology.

See protocol Early Management of Meningococcal Disease in Children.

See page 41 – Contraindications to Lumbar Puncture.

See pages 39-40. See protocol Early Management of Meningococcal Disease in Children.



### Case 3 Outcome

Seven hours after admission, child had a cardiac arrest and died.

#### DISCUSSION

##### Paediatric ward assessment:

Although there was mild meningism, the predominant clinical picture was one of advanced shock. Paediatric intensive care should have been called immediately.

##### Treatment:

The initial bolus of fluid and administration of intravenous antibiotics were appropriate, but the treatment was too slow. Improvement in CRT alone did not mean that shock had been reversed. The continuing presence of shock after 50ml/kg showed that the child urgently required intensive care for early elective intubation, ventilation and further aggressive resuscitation.

The pulmonary oedema was the result of advanced capillary leak. The treatment is to ventilate the child, not further deplete the intravascular volume with diuretics.

##### Results:

Blood gas should have been done on admission to see extent of the metabolic acidosis.

There was a significant coagulopathy which needed treatment with fresh frozen plasma.

The raised urea reflected inadequate renal perfusion secondary to intravascular hypovolaemia caused by capillary leak syndrome.

There were at least three indicators of severe disease on admission: hypotension, widespread purpura and low white cell count.

##### SHO review:

This SHO did not understand the illness. The very fast tachycardia indicated very advanced shock. A lumbar puncture should not even have been considered. The child's deteriorating neurological state was a pre-terminal sign of shock. A Mannitol infusion was considered as the doctor was confused as to the cause of the neurological depression.

#### LEARNING POINTS

- **Meningococcal septicaemia with shock is a medical emergency.**
- In meningococcal disease, extensive purpura is indicative of septicaemia with coagulopathy. It is very rare for this to be accompanied by raised intracranial pressure.
- When signs of established shock are present, it is essential that early aggressive management is instituted, and protocol followed with help from experts in PICU used to dealing with children in multi-organ failure.
- If features of severe disease are present (see page 34) then seek expert help urgently.
- Mannitol is used for raised intracranial pressure associated with meningitis. It is not used for septicaemia/shock.

### Conclusion

This case history clearly demonstrates the importance of understanding the difference between septicaemia and meningitis, and shows how children with advanced disease need expert care.



## Case 4

### CASE HISTORY

*15 year old boy non-specifically unwell for a day. Woke with a widespread purpuric rash and taken straight to hospital.*

#### **A&E assessment:**

Temp 39.0, HR 120, RR 20, BP 90/60.

Alert no meningism; purpuric rash spreading.

Diagnosis: meningococcal septicaemia.

Hb 11.5, WCC 4.3, Platelets 50.

Na 136, K 4, urea 6.2, creatinine 138.

PT (prothrombin time) >180, APTT (activated partial thromboplastin time) >240, INR 12.

Please see chart on following page for subsequent management and clinical course.

#### **7.5 hours:**

Formal referral to PICU: telephone advice given to start aggressive resuscitation as per protocol, Early Management of Meningococcal Disease in Children.

#### **8 hours:**

CRT is 7 seconds. A venous gas is done: pH 7.15, PCO<sub>2</sub> 5.5, PO<sub>2</sub> 15.0, HCO<sub>3</sub> 11.5, Base excess -16.5.

### QUESTIONS

How would you interpret these signs?  
What other clinical signs are important to record?

What is causing the renal impairment?  
How would you interpret these results?  
What test would help you establish the degree of shock?  
What is this boy's prognosis?

From the chart comment on the overall fluid management.

Is there any contraindication to the lumbar puncture done at hour 2?

From the chart explain the significance of the fall in blood pressure at hour 5.  
How would you manage this?

How would you interpret this gas?

### LOOK IT UP

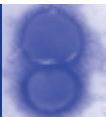
See page 38 – Normal values of vital signs.  
See page 38 – Initial assessment of any febrile child.

See Section 5 – Pathophysiology.  
See Section 5 – Pathophysiology.  
See page 39 – Clinical signs of septicaemic shock.  
See page 34 – Clinical features of severe disease.

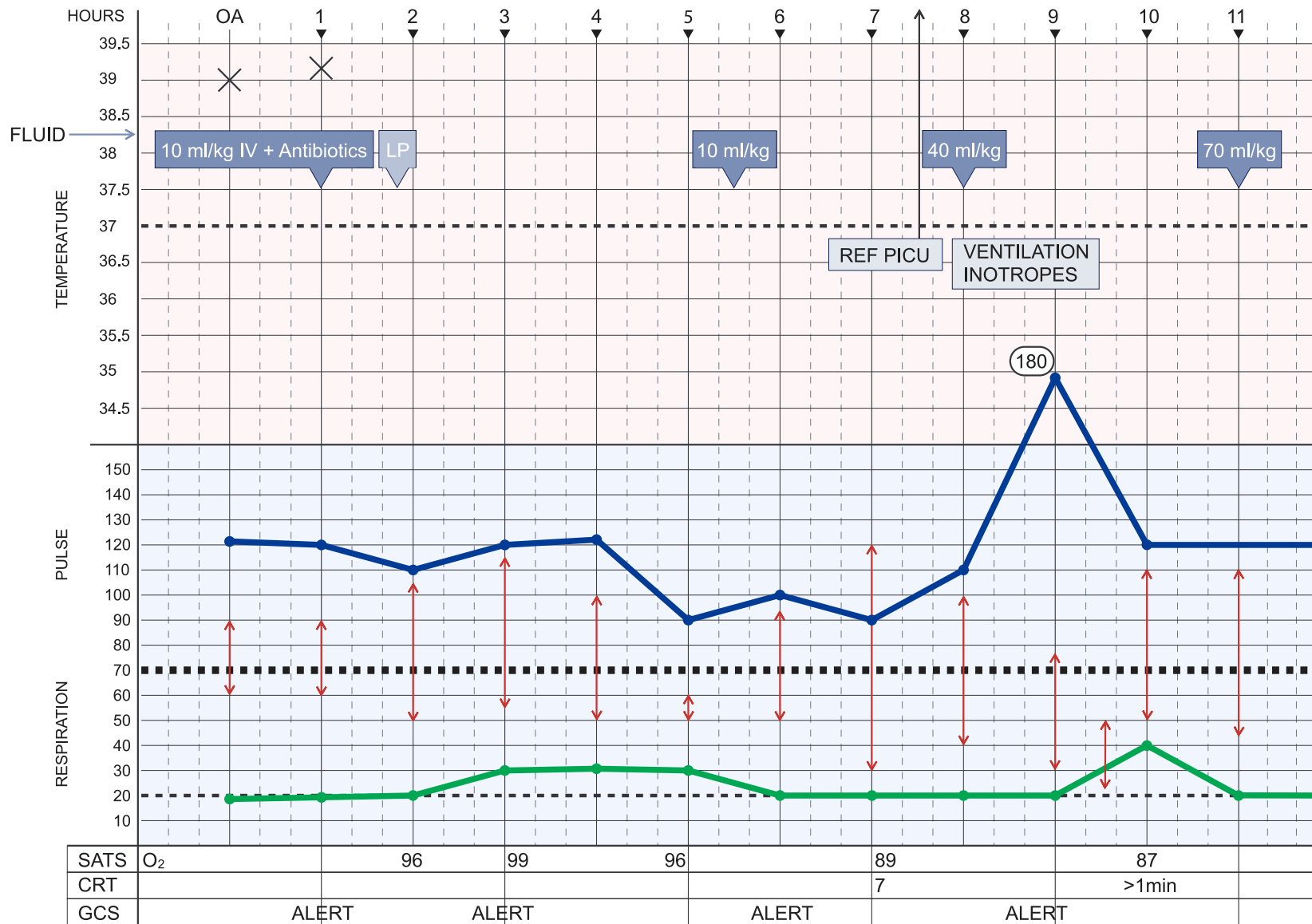
See protocol, Early management of meningococcal disease in children.

See page 41 – Contraindications to lumbar puncture.

See Section 5 – Pathophysiology.



**CASE 4 CHART**



PURPURA ++ EXTENDING  
NO MENINGISM



### Case 4 Outcome

At 7.5 hours after admission a paediatric intensive care unit was called for advice and elective intubation, ventilation and aggressive fluid management commenced. Unfortunately these measures were commenced too late and the patient had a cardiac arrest 3 hours later.

#### DISCUSSION

##### A&E assessment:

This boy presented with meningococcal septicaemia and shock. The initial medical assessment did not record the peripheral perfusion and oxygen saturation.

The results show that the patient had a low white cell count, which is a marker of severe disease. There was also laboratory evidence of disseminated intravascular coagulation which should have been treated immediately with fresh frozen plasma and then monitored closely. The raised urea and creatinine were the result of intravascular hypovolaemia secondary to capillary leak syndrome.

A venous blood gas would give a base excess which is a measure of the metabolic acidosis associated with shock.

The clinical and laboratory features indicated very severe disease.

##### A&E management:

The fluid management was totally inadequate. In the first 6 hours after admission only 20ml/kg had been given, no urine output had been measured. See management section on page 44-46 for further details of the principles of resuscitation.

A lumbar puncture is absolutely contraindicated in the face of widespread purpura, severe coagulopathy and cardiovascular shock

*Vital signs on chart.* Note that the tachycardia remained well above the normal range for age throughout the day. This was the result of intravascular hypovolaemia. The patient should have been catheterised to monitor the urine output on an hour-by-hour basis. By hour 3 the respiratory rate had risen to 30, most likely as a result of acidosis, pulmonary oedema and hypoxia. The boy remained alert which is often seen in septicaemia and may falsely reassure doctors as to the severity of the illness.

At hour 5 a very low blood pressure was recorded. This indicated that the patient needed much more aggressive resuscitation as per protocol, Early Management of Meningococcal Disease in Children.

The blood gas done eventually at 8 hours showed a severe metabolic acidosis.

#### LEARNING POINTS

- **Meningococcal septicaemia with shock is a medical emergency.**
- Children who present with meningococcal septicaemia in the morning may have very advanced disease as they have many hours during the night, unobserved by their parents, in which to become ill.
- If features of severe disease are present ( see page 34), then seek expert help immediately.
- Children with shock may be alert until late in the illness and this may make them look less sick than they actually are.
- Hypotension is a late sign of shock in children and does not need to be present to diagnose shock.
- Children with shock need assessment by a senior paediatrician.
- Refer early to a regional paediatric intensive care unit.

#### Conclusion

This case history shows that despite the correct diagnosis of meningococcal septicaemia being made, the resuscitation was slow and inadequate. The patient remained in Accident and Emergency for 8 hours instead of being transferred to a PICU immediately. A diagnosis of meningococcal septicaemia should bring about urgent medical treatment, and expert help should be sought if there are signs of shock.



## Case 5

### CASE HISTORY

*10 month old boy. Taken to GP with h/o sudden onset of fever, vomiting and lethargy for 4 hours. Mother very anxious about child. GP referred child to walk-in clinic at hospital.*

**History on admission:** Feverish and drowsy – sudden onset.  
2 episodes of vomiting, 1 soft stool, no rash.

**Assessment on admission:**

Drowsy and pale, dark rings around eyes.

Temp 37.7

CVS: P 181, BP 120/52. CRT 4 secs. Child peripherally shutdown.

RS: RR 32 breathing laboured and child cyanosed.

SaO<sub>2</sub>: 100% in oxygen.

NS: GCS10 then 9, no neck stiffness.

Fine blanching rash on abdo/chest. 1 petechial spot on abdo.

**Action taken :**

1. Immediately given antibiotics and 40 ml/kg albumin.
2. “Crash call” put out for PICU team.
3. Full set bloods taken.

**Results:**

WCC 2.4, Hb 10.5, pl 70.

Na 149, K 3.4, urea 10.9, Creat 121.

HCO<sub>3</sub> 15, BE -7.

PT 30, APPT 75, INR 2.5.

Taken to PICU. Still shocked after 40ml/kg. Electively intubated and ventilated, Commenced adrenaline, bicarbonate and K corrections.

Extensive purpuric rash developed.

PICU consultant called in to supervise care.

### QUESTIONS

What might sudden onset of illness in an otherwise well child suggest?

What do you think of this assessment?

What do these signs tell you?

How would you interpret the normal blood pressure in the context of other observations?

When conscious level is depressed and/or falling, is severity of disease likely to be worse when signs of meningitis are present, or when they are absent?

Is the very scanty rash a reassuring sign?

What do you think of this course of action?

What do you think of these results?

### LOOK IT UP

See page 35 – Making the diagnosis: Taking a history.

See page 38 – Initial assessment of any febrile child & pages 39-40 – Clinical signs. See Section 5 – Pathophysiology.

See page 34 – Clinical features of severe disease.

See page 36 – Examining the patient: The rash.

See protocol Early Management of Meningococcal Disease in Children.

See page 34 – Clinical features of severe disease.



### Case 5 Outcome

Subsequent PICU care (summary): Severe respiratory failure with pleural effusion - ventilated for total of 3 weeks. High dose inotropes required for several days. Severe coagulopathy - treated with fresh frozen plasma and cryoprecipitate. Renal replacement therapy needed. Peritoneal dialysis later that evening for fluid overload and renal failure - progressed to haemofiltration after several days. 3 weeks PICU, in hospital 2 months.

#### DISCUSSION

Sudden onset of illness in otherwise well child.

##### Assessment:

Very thorough and entirely appropriate. Evidence of shock. Tachycardia, cool peripheries. Note normal blood pressure which in association with signs of shock indicates cardiac compensation.

Child had evidence of respiratory decompensation secondary to acidosis, hypoxia and capillary leak syndrome.

Depressed or falling conscious level must always be taken seriously, but it may occur quite early in meningitis. Depressed or falling conscious level in a patient with septicaemia, in the absence of signs of meningitis, indicates very advanced shock.

The rash was not dramatic on admission. There was only one non-blanching spot. This shows how the typical haemorrhagic rash may only appear once the child is very ill. Do not be reassured if a child has only a scanty rash: you must try to determine how advanced is the underlying septicaemia.

The results show a low white cell count, falling platelets, coagulopathy and rising urea and creatinine. These are all features of severe disease.

##### Action:

The severity of the child's illness was appreciated immediately and aggressive resuscitation commenced. Senior help was called for and the child was admitted to an appropriate intensive care unit.

Once on PICU the aggressive management was continued following the early management protocol. Senior PICU help was sought to ensure this child had one to one medical attention whilst being stabilised. The typical rash of meningococcal septicaemia was by then apparent. Multi-organ failure was managed in PICU.

#### LEARNING POINTS

- Febrile illness of sudden onset = classic picture of meningococcal disease, mainly affecting well children. However, respiratory illnesses, particularly flu, may predispose to meningococcal disease. The less typical picture is of initially trivial symptoms suddenly becoming more serious with a high fever and other symptoms.
- Always take a parent's anxiety very seriously.
- **Meningococcal septicaemia is a medical emergency.**
- Falling conscious level in a shocked child is a poor prognostic sign.
  
- Isolated pinprick spots may appear where the rash is mainly blanching so it is important to search the whole body for small petechiae.
- Underlying disease may be very advanced by the time a rash appears. The rapidly evolving haemorrhagic 'text book' rash may be a very late sign. It may be too late to save the child's life by the time this rash is seen.
  
- Once shock is advanced, it can only be reversed by aggressive resuscitation and management of complications in intensive care.

#### Conclusion

Children with severe septicaemia and multi-organ failure have a high risk of mortality especially if they are under 1 year of age. In this case all the signs of severe illness were recognised immediately and acted on appropriately. It is likely that without such rapid medical attention this child would have died.



## Case 6

### CASE HISTORY

*14 year old girl admitted to hospital with 24 hour history of fever, increasing headache associated with 6 episodes of vomiting in evening. She has developed photophobia. Also feels generally unwell with myalgia.*

GP visited and gave IM penicillin as meningitis considered the most likely diagnosis.

#### **SHO assessment on arrival:**

Responsive, mild photophobia, no neck stiffness.

Temperature 39.7.

Pale macular rash over trunk, no purpura.

CVS: Pulse 85 regular, BP 115/75. Heart sounds normal.

Chest clear, Abdomen normal.

NS: Glasgow coma score 15/15.

Full power in arms and legs- all movements.

Cranial nerves intact, no papilloedema.

Differential diagnosis made of meningitis or viral illness. Given intravenous antibiotics immediately and blood tests sent.

#### **1.5 hours later (registrar review):**

Conscious level has deteriorated over the past hour. Now no communication, eyes open. Neck stiffness+++

BP 152/90, HR 88.

? to CT ? to do lumbar puncture.

Lumbar puncture is performed.

CSF is cloudy and under very high pressure. Patient deteriorates rapidly with falling conscious level, decrease in respiratory effort. Patient is intubated and ventilated and taken to intensive care.

### QUESTIONS

What diagnosis does this history of symptoms suggest?

What do you think of the action taken by the GP?

What do you think of this assessment? Are there any important clinical signs that have not been commented on.

What observation would you make about the rash?

Is the absence of papilloedema important?

Should antibiotics be delayed until a more definitive diagnosis is made?

What has occurred?

What treatment does the patient need now?

Are there any contraindications to LP?

### LOOK IT UP

See page 35 – Making the diagnosis: Taking a history.

See page 38 – Initial assessment of any febrile child.

See page 36 – Examining the patient: The rash.

See page 40.

See page 44 – Management of septicaemia and meningitis.

See page 40.

See protocol Early Management of Meningitis and Septicaemia in Children.

See page 41 – Contraindications to lumbar puncture.



### Case 6 Outcome

The patient did not recover and was found to be brain dead.

#### DISCUSSION

This history is typical of meningitis. The patient was generally unwell with fever and myalgia but had features of CNS infection with headache, vomiting and photophobia.

Appropriate treatment from the GP.

#### SHO assessment:

This is a good assessment. The conscious level was recorded and signs of raised intracranial pressure looked for, however papillary responses and size should also have been recorded.

The rash is non-specific.

Immediate administration of IV antibiotics was appropriate.

#### Registrar review:

LP should only be undertaken once it has been decided that the patient is stable enough to undergo this procedure.

There was a dramatic change in the patient's condition. The patient had developed features of raised intracranial pressure. The patient urgently needed treatment to reduce the intracranial pressure.

With the dramatic change in conscious level it was dangerous to take the patient to the scanner without securing the airway. LP was contraindicated.

The patient unfortunately coned whilst having the lumbar puncture. All efforts to resuscitate the patient after this were unsuccessful.

#### LEARNING POINTS

- Always look for signs of raised intracranial pressure (RICP) in all patients with evidence of meningitis.
- In cases of pure meningitis, the rash is more often scanty, absent or atypical than in septicaemia or meningococcal disease with mixed presentation.
- Papilloedema does not have to be present to diagnose RICP, it is a late sign.
- Antibiotics should be given immediately if the diagnosis of meningitis is included in the differential.
  
- **Raised intracranial pressure is a medical emergency.**
- Lumbar puncture is strictly contraindicated when there is RICP, e.g. if the conscious level is deteriorating and the blood pressure is rising.
- Call for senior help and Paediatric Intensive Care immediately if there are signs of RICP.

### Conclusion

All patients with meningitis must have clinical signs of raised intracranial pressure looked for and always rechecked prior to doing an LP. Beware the patient who deteriorates after admission. If in doubt delay lumbar puncture until senior advice can be sought.



## Case 7

### CASE HISTORY

15 year old boy, 30 hours of flu like illness. On day of presentation his mother found him febrile and confused in bed.

#### Assessment on admission 07:30:

Temp 38.2.

HR 103, BP 148/102.

Incoherent and behaving inappropriately.

Some neck stiffness, Kernig's sign negative.

Movements almost decerebrate.

Meningococcal rash noted.

#### Action taken:

Given intravenous antibiotics.

Admitted to ward.

#### On examination on ward:

Agitated, disorientated and confused with fluctuating conscious level. He had developed a convergent squint.

#### Investigations:

Hb 14, WCC 15.2, pl 190.

Urea 4.7, creatinine 54.

Na 140, K 4.2, Bicarbonate 24.

INR 1.0, PTTR 1.2.

CT scan showed no signs of raised intracranial pressure.

**10.30:** The patient's conscious level fell to 8/15 and then he suffered a respiratory arrest. The BP was 225/115. He had no respiratory effort and so was intubated and ventilated.

He was turned onto his side for a lumbar puncture, which was performed. He suffered a sudden onset of bradycardia and hypotension with desaturation.

### QUESTIONS

In teenagers, CNS symptoms and confusion are sometimes misinterpreted. What mistaken diagnosis might be reached?

What do these observations indicate?  
What do you think of this assessment?  
What further assessment should be made?

What do you think of this treatment?

What needs to be done now?

Are there any signs of co-existing shock or coagulopathy?

Is CT scanning sensitive to RICP? What other tests could be done?

Are there any contraindications to LP in this situation? Is LP necessary?

### LOOK IT UP

See page 39-40 – Clinical Signs.

See page 39-40 – Clinical Signs.

See page 44 – Management of septicaemia and meningitis.

See protocol Early Management of Meningitis and Septicaemia in Children.

See page 39 – Clinical signs of septicaemic shock, and page 43 – Specific organ dysfunction in shock.

See page 40 – Clinical signs of raised intracranial pressure.

See page 41 – Contraindications to lumbar puncture.



### Case 7 Outcome

The pupils were noted to be fixed and dilated when examined a few hours later. The following day brain death tests were performed and the patient was declared brain dead.

#### DISCUSSION

##### Assessment on admission:

There were signs of raised intracranial pressure (RICP) present on admission. There was systemic hypertension, depressed conscious level and abnormal movements.

There should have been an assessment of pupil size and reactivity and examination of the fundi.

##### Action taken:

Antibiotics are essential but the patient should also have been given Mannitol immediately and electively intubated and ventilated to try and reduce the RICP. This patient urgently needed expert treatment in intensive care.

##### Investigations:

Note that even in patients with severe meningitis the investigations remain relatively normal. There was no acidosis, coagulopathy, renal dysfunction.

##### 10.30:

There were clear clinical signs that the patient's condition was deteriorating and unstable and the RICP needed immediate action. CT scanning and waiting for test results resulted in 3 hours delay and was unacceptable.

The raised intracranial pressure was severe leading to respiratory arrest. There was grossly elevated systemic hypertension.

The patient coned. Clearly he had advanced disease on presentation but no efforts were made prior to his respiratory arrest to stabilise him and reduce the raised intracranial pressure. The lumbar puncture was unnecessary for initial diagnosis and totally contraindicated after his respiratory arrest.

#### LEARNING POINTS

- Acute confusion in a teenager may be mistaken for drug or alcohol intoxication. Meningitis and encephalitis must be included in the differential diagnosis of a teenager who is acutely confused or disruptive.
- Raised intracranial pressure is a medical emergency. Call for senior help and Paediatric Intensive Care immediately if there are signs of RICP.
- Ophthalmoplegia (new squint) is a further sign of raised intracranial pressure with herniation of supratentorial part of the brain through the tentorial opening. This must be acted on immediately.
- CT scanning is not a sensitive tool in detecting RICP. It is dangerous to put a child with fluctuating conscious level into the scanner without securing the airway first.
- It is crucial to look for the signs of RICP before attempting LP and defer if signs are present. Lumbar puncture is contraindicated when there are signs of RICP and neurological failure.

### Conclusion

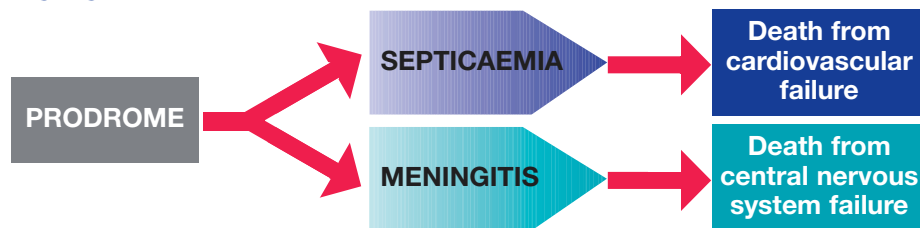
This boy presented with raised intracranial pressure which is a medical emergency. This was not appreciated. Inappropriate investigations were conducted and no emergency management of the condition was undertaken.



### CHARACTERISTICS OF MENINGOCOCCAL DISEASE

There are two major clinical forms of meningococcal disease: meningitis and septicaemia. Most patients will present with the meningitic form of the disease or have a mixed presentation. A minority will have pure septicaemia - it is these patients who carry the worst prognosis and maximum effort must be made to identify them early<sup>5</sup>. There are important differences in the pathophysiology of meningitis and septicaemia which underlie the clinical presentation and management of the two main forms of the condition (see section 5).

### DISEASE PATHWAY



### CLINICAL FEATURES OF SEVERE DISEASE

The diagram above illustrates the main causes of death from MD. In the majority of patients, one disease process predominates. Patients presenting with mixed disease will also tend, as the disease worsens, to become either profoundly septicaemic or profoundly meningitic. A few will have combined severe septicaemia with shock and severe meningitis with raised intracranial pressure and these need expert management. Patients presenting with septic shock without meningitis carry the worst prognosis<sup>5</sup>. Although a few patients with meningitis will die from raised intracranial pressure, most deaths from MD result from shock and multi-organ failure<sup>6</sup>.

Features which predict poor prognosis at the time of presentation include:

- Presence of shock
- Absence of meningism
- Rapidly progressive purpuric rash
- Low peripheral white blood cell count
- Thrombocytopenia
- Markedly deranged coagulation
- Depressed conscious level

### A. TAKING A HISTORY

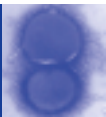
Meningococcal disease is extremely unpredictable. The presentation can be very varied and patients may be difficult to differentiate from those with viral illnesses during the early stages. Most children with MD present as an acutely febrile child and may not have a rash in the early stages.

It is important to take a detailed history and ask parents about the specific symptoms of septicaemia and meningitis. Beware of simply 'eyeballing' a child and assuming they have a trivial illness. This is how many mistakes are made. **Make sure you have understood what exactly is worrying the parent and why they are seeking help at this point.** Be careful if the child has had contact with a case of meningococcal disease even if they have had prophylactic antibiotics as they can still become ill. Ask about travel to sub-Saharan Africa or contact with Hajj pilgrims.

At the initial assessment look for signs and symptoms of septicaemia or meningitis. Some symptoms can be subtle and must be specifically asked about when taking a history.

### SYMPTOMS OF SEPTICAEMIA

- **FEVER:** Many children become suddenly ill with a fever: the classic picture is of a disease of rapid onset. However, some children develop septicaemia after a simple viral illness. In these cases the symptoms may be initially trivial and last for some time and then suddenly become more serious with a high fever and other symptoms of sepsis. A history of a fever in a child presenting afebrile is important.
- **RIGORS:** Children with septicaemia often have rigors. Occasionally the shaking, if very severe may be mistaken for fitting, but children having rigors will remain conscious.
- **ACHES:** They usually experience very bad muscle aches and joint aches making them restless and miserable.
- **LIMB PAIN:** Isolated severe limb pain in the absence of any other physical signs in that limb is a well recognised phenomenon in MD. The pain can be very severe and children have been mistakenly put into plaster to treat presumed fractures.
- **GASTROINTESTINAL SYMPTOMS:** Abdominal pain, vomiting and diarrhoea are very common in septicaemia. Children may become faecally incontinent.
- **WEAKNESS:** This can become profound.
- **RASH:** Ask about any new rashes or marks on the child's skin that the parents may have noticed. Note that parents may not realise that the purpura / bruises or petechiae on the child's skin is a 'rash' as they associate the word rash more with a pink 'measles-like' rash.



# Section 4 | Making the diagnosis

## SYMPTOMS OF MENINGITIS

The main symptoms of meningitis are all due to the dysfunction of the central nervous system. Be aware that symptoms can vary according to the age of the child. Symptoms include:

- Fever
- Headache
- Vomiting
- Drowsiness/confusion
- Fits
- Photophobia
- Neck stiffness

Young children may have fever and vomiting associated with irritability, drowsiness and confusion. **They may be very hard to assess and a parent's anxiety about their state of responsiveness and alertness must always be taken seriously.**

Older children are more likely to have fever, vomiting and complain of headache, stiff neck and photophobia.

Teenagers may present with symptoms related to a change in behaviour such as confusion or aggression. These may mimic the symptoms of alcohol or drug intoxication.

## B. EXAMINING THE PATIENT

### THE RASH

Most patients with meningococcal septicaemia develop a rash - it is one of the clearest and most important signs to recognise. In meningitis it can be scanty, atypical or even absent.

It is **crucial** to remember that the underlying meningitis or septicaemia may be very advanced by the time a rash appears. The rapidly evolving haemorrhagic 'text book' rash may be a very late sign, it may be too late to save the child's life by the time this rash is seen. It is very important to examine children for the signs of meningitis or septicaemia and investigate and treat if necessary based on those findings.



(early) Maculopapular rash with scanty petechiae

Courtesy Dr A Flordan

### Early stages

In the early stages the rash may be blanching and macular or maculopapular (sometimes confused with flea bites), but it nearly always develops into a non-blanching red, purple or brownish petechial rash or purpura. Isolated pin-prick spots may appear where the rash is mainly maculopapular, so it is important to search the whole body for small petechiae, especially in a febrile child with no focal cause. A rapidly evolving petechial or purpuric rash is a sign of very poor prognosis.

Although some of the causes of petechial rashes are self-limiting conditions, many others, including MD are fulminant or life-threatening, and a non-blanching rash should therefore be treated as an emergency.

The rash can be more difficult to see on dark skin, but may be visible in paler areas, especially the soles of the feet, palms of the hands, abdomen, or on the conjunctivae or palate.

Purpuric areas that look like bruises can be confused with injury or abuse.

Extensive purpuric areas often over the feet, legs and hands are usually called 'purpura fulminans'.



Purpuric rash of septicaemia. Purple blotches may be larger, resembling bruises or even blood blisters.

Courtesy Dr A Flordan



Purpuric rash on dark skin



Petechial rash on conjunctivae

Courtesy Prof DA Warrel

## Section 4 | Making the diagnosis

### INITIAL ASSESSMENT OF ANY FEBRILE CHILD

For all febrile children the following should be undertaken:

- Fully undress and examine systematically. Make a thorough search for a focus of infection: think about the 'hidden sites' such as meninges, urinary tract and bloodstream (septicaemia). Mildly pink tympanic membranes or throat do not constitute a focus.
- If a rash is found, it is important to decide if it is non-blanching. **All febrile children with haemorrhagic rashes must be taken very seriously.** Although many children with fever and petechiae will have viral illnesses, there is no room for complacency when assessing these children. They must all have their vital signs measured (see below), a decision made as to whether they have signs of meningitis or septicaemia (see below) and given intravenous antibiotics. A senior paediatrician should be informed immediately.
- Children without a rash or with a blanching rash can still have MD. The rash may appear later or not at all if the child has pure meningitis and occasionally with septicaemia. Thorough clinical assessment should ascertain whether there are physical signs of serious systemic illness.

The following clinical signs must be measured and recorded to complete a full assessment:

- Temperature
- Heart rate
- Respiratory rate
- Blood pressure
- Capillary refill time or toe-core temperature gap
- Oxygen saturation measurement (normal value is >95% in air)
- Assessment of conscious level
- Pupil size and reaction
- If rash present record if blanching, extent of rash, speed of development and if it is petechial or purpuric

### NORMAL VALUES OF VITAL SIGNS

Age	HR/min	RR/min	Systolic BP
<1	110-160	30-40	70-90
1-2	100-150	25-35	80-95
2-5	95-140	25-30	80-100
5-12	80-120	20-25	90-110
Over 12	60-100	15-20	100-120

From *Advanced Paediatric Life Support - the Practical Approach*. Mackway Jones K, Molyneux E, Phillips B, Wieteska S, editors. 3rd ed London: BMJ Books; 2001.

### ASSESSMENT OF A FEBRILE CHILD WITH SUSPECTED MENINGOCOCCAL DISEASE

If MD is suspected, the purpose of the initial assessment should be to identify whether shock or raised intracranial pressure is present and the severity of the illness.

### CLINICAL SIGNS OF SEPTICAEMIC SHOCK

Septicaemia will lead to shock and multi-organ failure. Shock is a clinical diagnosis. The signs are a result of circulatory failure. The underlying pathophysiology of septicaemia and the capillary leak syndrome leading to these signs are briefly summarised in section 5.

A child in early shock may still be alert and have a normal blood pressure.

The early signs of shock include:

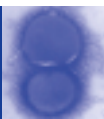
- Tachycardia
- Cool peripheries (CRT>4 seconds) or toe-core temperature gap of >3 degrees
- Pallor
- Decreased urine output (<1ml/kg/hr)
- Tachypnoea – secondary to acidosis and hypoxia

As shock progresses further signs develop:

- Metabolic acidosis with base excess worse than -5
- Hypoxia: PaO<sub>2</sub> <10kPa in air or saturation < 95% in air
- Increasing tachypnoea, tachycardia and gallop rhythm

Late signs of shock include:

- Drowsiness or agitation
- Hypotension: blood pressure can be normal until the child is very shocked



# Section 4 | Making the diagnosis

## CLINICAL SIGNS OF MENINGITIS

When examining a child for signs of meningitis it is crucial to remember that the younger a child **the less likely it will be to have neck stiffness or photophobia** (especially those <2 years of age). Be guided by the parents as to whether the child is drowsy or behaving inappropriately. Often parents are quick to recognise that the cry of a baby has changed or they are making poor eye contact.

- Babies with meningitis may have a full or bulging fontanelle reflecting the raised intracranial pressure. They may feel stiff or have jerky movements or they may be very floppy. Fits are common.
- Drowsiness or decreased conscious level (or fluctuating level) is a very important sign in children of all ages.
- Teenagers with meningitis often present in an aggressive and combative manner rather than becoming drowsy. Drug and alcohol intoxication may be suspected.
- Rash: can be present, but more likely to be scanty or petechial than in septicaemia (see page 36).

## CLINICAL SIGNS OF RAISED INTRACRANIAL PRESSURE

Children with meningitis are at risk of developing RICP (see section 5 for the pathophysiology).

Signs of RICP are:

- Falling or depressed conscious level
- Abnormal posturing: decorticate or decerebrate
- Dilated pupils or unequal pupils
- Focal neurology
- Bradycardia and hypertension
- Abnormal breathing pattern
- Cushing's triad: slow pulse, raised blood pressure and abnormal breathing pattern – late sign of RICP
- Papilloedema is a late sign, its absence does not mean there cannot be any RICP

The diagnosis of raised intracranial pressure is a clinical one:

- Routine CT scanning is not indicated in patients with meningitis as CT scans are not sensitive in picking up signs of RICP.
- **Lumbar puncture is contraindicated in patients with signs of raised intracranial pressure as 'coning' can be precipitated.**

## CONTRAINDICATIONS TO LUMBAR PUNCTURE:

- Prolonged or focal seizure
- Focal neurological signs (including ocular palsies)
- Widespread purpuric rash in ill child
- Glasgow coma score <13
- Papillary dilatation
- Impaired oculocephalic reflexes
- Abnormal posture
- RICP: inappropriately low pulse, elevated blood pressure and irregular respirations (indicating impending brain herniation)
- Coagulopathy
- Papilloedema
- Hypertension

## INITIAL LABORATORY ASSESSMENT

The tests below should be done on all suspected cases of MD and children who are suspected of having an invasive bacterial infection:

- Full blood count
- Electrolytes and urea
- Calcium and magnesium (metabolic derangements are common in septicaemia and may contribute to myocardial dysfunction)
- Clotting studies
- Venous blood gas to measure base excess
- Blood culture
- Meningococcal PCR to send to reference laboratory



# Section 5 | Pathophysiology and principles of management

## UNDERSTANDING THE PATHOPHYSIOLOGY OF MENINGOCOCCAL INFECTION AND THE PRINCIPLES OF MANAGEMENT

The management of meningitis and septicaemia are best understood by having a basic knowledge of the pathophysiology of meningococcal infection. The section below is a summary. For more detailed information see references at the end of this section.

### CLINICAL PATHOPHYSIOLOGY OF SEPTICAEMIA

#### CAPILLARY LEAK SYNDROME

When meningococci enter the bloodstream endotoxin is released from the bacteria. This triggers an intense inflammatory response, damaging the endothelium lining the blood vessels so that it becomes 'leaky'. Plasma then leaks out of the blood into interstitial tissues. This is called the 'capillary leak syndrome'. The patient becomes hypovolaemic as fluid is lost from the blood stream. Hypovolaemia results in diminished venous return to the heart, and reduced filling of the ventricles decreases cardiac output.

The patient increases the heart rate and the force of contraction of the heart, in order to maintain the blood pressure and cardiac output ('cardiac compensation'). Blood is also diverted away from non vital organs such as the limbs and kidneys. **Early in sepsis children have tachycardia, cool peripheries and normal blood pressure. They are also alert as blood flow to the brain is being maintained at the cost of the other organs.**

Continuing capillary leak results in tissues becoming underperfused and hypoxic. Hypoxia is exacerbated by capillary leak into the lung vascular beds causing pulmonary oedema. Cardiac compensation becomes increasingly difficult in the presence of acidosis, hypoxia and severe hypovolaemia and eventually the blood pressure falls. This is a very late sign in septic shock in children.

#### DISSEMINATED INTRAVASCULAR COAGULATION

Endotoxin causes activation of the coagulation cascade together with down-regulation of anticoagulant pathways. There is also up-regulation of antifibrinolytic proteins leading to a procoagulant state. This is demonstrated by prolongation of clotting times and thrombocytopenia. Micro-vascular thrombosis is a major contributing factor to multiple organ failure and purpura fulminans.

#### MYOCARDIAL DYSFUNCTION

Endotoxin and inflammatory cytokines together with poorly characterised myocardial depressant factors reduce myocardial contractility despite adequate fluid resuscitation. This may also cause a relative inotrope unresponsiveness.

### SPECIFIC ORGAN DYSFUNCTION IN SHOCK

#### Respiratory failure

(arterial  $PO_2 < 10\text{kPa}$  in air or  $PCO_2 > 6$ )

Common in shock. Capillary leak into lung parenchyma → acute pulmonary oedema.

**Clinically: tachypnoea, chest wall retraction, hypoxia.**

#### Metabolic derangement

Septicaemia causes profound acidosis and derangements in metabolism, which may affect myocardial function and need correcting.

Hypoglycaemia is common. Hypokalaemia, hypocalcaemia, hypomagnesaemia and hypophosphataemia all occur.

#### Coagulopathy (purpuric rash)

Coagulopathy occurs early in patients with septicaemia. The laboratory findings of disseminated intravascular coagulation (DIC) are common in such patients: increased PT (prothrombin time), APTT (activated partial thromboplastin time), TT (thrombin time) and decrease in plasma fibrinogen with elevation of fibrin degradation products and thrombocytopenia. The coagulopathy is generally associated with the presence of a purpuric rash, but significant coagulopathy may infrequently occur in the absence of purpura.

#### Neurological dysfunction

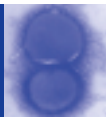
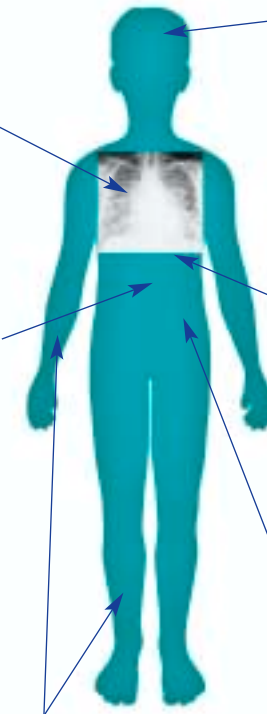
In septicaemia, patients may be alert until late in the illness. Falling conscious level results from impaired cerebral blood flow and disturbed brain metabolism due to hypotension, hypoxia and acidosis.

#### Myocardial failure

Depressed myocardial function is multifactorial, including endotoxin cytokines and multiple metabolic derangements, hypoxia, and hypovolaemia. **Clinically: tachycardia, gallop rhythm, cool peripheries and eventually hypotension.**

#### Renal failure

Little or no urine output ( $< 1\text{ml/kg/hour}$ ) is a very early sign in septic shock, initially due to hypovolaemia. If shock persists then renal failure may occur.



## CLINICAL PATHOPHYSIOLOGY OF MENINGITIS

Meningococcal meningitis usually carries a better prognosis than the septicaemic form of the illness. Deaths do occur, however, due to the severity of the inflammatory process within the brain.

Bacteria invade through the nasopharynx, multiply in the bloodstream and then penetrate the blood brain barrier. Bacterial products, including endotoxin, initiate inflammatory changes in the CSF and blood brain barrier. Changes in the integrity and permeability of the blood-brain barrier together with alteration of brain cell function causes cerebral oedema. As a consequence there is an increase in total brain water content leading to an increase in intracranial pressure. In addition, the inflammatory process causes micro-vascular thrombosis in blood vessels of the meninges and brain substance. Both the cerebral oedema and micro-vascular thrombosis lead to a reduction in cerebral blood flow.

## MANAGEMENT OF SEPTICAEMIA AND MENINGITIS

The aim of this section is to outline the principles of management of septicaemia and meningitis which are based on understanding the pathophysiology. A fuller explanation of the management of meningococcal disease can be found in Archives of Diseases in Childhood, July 2003, Volume 88, No 3, p 608-614 by SB Welch and S Nadel. The protocol from this article has been published as 'Early Management of Meningococcal Disease in Children' and is at the back of this handbook.

### ANTIBIOTICS

- All children with fever and a haemorrhagic rash
- Children with shock with or without a rash
- Children with clinical evidence of meningitis. If lumbar puncture is contraindicated (see page 41), treat immediately with antibiotics and lumbar puncture when safe.

### Cefotaxime or Ceftriaxone

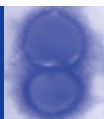
For prophylaxis of contacts speak to Public Health department. Close family contacts often are treated by the clinicians caring for the ill child with Rifampicin, Ciprofloxacin (not in children) or Ceftriaxone. See protocol, Early Management of Meningococcal Disease in Children for doses.

## PRINCIPLES OF MANAGEMENT OF SEPTICAEMIA WITH SHOCK

Children with evidence of shock need immediate resuscitation:

- A- assess airway for patency.
- B- give oxygen to all patients even if oxygen saturations are normal in order to optimise tissue oxygenation.
- C- secure good venous access. The goal of circulatory support in shock is the maintenance of tissue perfusion and oxygenation. Remember in shocked children the intra-osseous route may be the most effective way of giving large volume replacement.
  - Fluid boluses should be initiated. Boluses of 20ml/kg should be given whilst monitoring the clinical response (HR, RR, BP, CRT, urine output). Unless the clinical response is excellent, up to 40-60 ml/kg should be given over the first hour. If there is still evidence of shock at this stage the patient will need further management, preferably on a paediatric intensive care unit.
  - Advice should be sought early. Intensive care is required in order to monitor and treat the patient adequately, which will require central venous and arterial access.
  - Elective intubation and ventilation of shocked patients who have received 40-60 ml/kg is recommended to reduce the risk of pulmonary oedema and reduce the work of breathing and myocardial workload.
  - If shock persists once the circulating volume has been restored (as measured by central venous monitoring) then inotropic support of the heart is indicated.
  - Metabolic derangements of calcium, magnesium and potassium are common, and need frequent checking and correction.
  - Renal replacement therapy may be necessary in patients with incipient or established renal failure.

**Remember – Call for senior help early. Sick septic children need experienced doctors. This is not the time to 'have a go!'**



## PRINCIPLES OF MANAGEMENT OF MENINGITIS WITH RAISED INTRACRANIAL PRESSURE

See page 40 for signs of RICP.

The main objective in managing patients with RICP is to maintain oxygen and nutrient delivery to the brain. **Call for senior help and Paediatric Intensive Care immediately if there are signs of RICP.**

- A- Maintain the airway in patients with depressed conscious level (GCS < 8). Elective intubation if GCS falling or patient only responding to pain.
- B- Optimise breathing to prevent hypoxia or hypercapnia both of which exacerbate raised ICP. Early elective intubation and ventilation.
- C- Resuscitate cautiously with fluid. Gain early venous access. Patients may have a mixed picture of shock and RICP and resuscitation of the shock should be the primary concern.

PLEASE NOTE Patients with RICP may have prolonged capillary refill time and a mild metabolic acidosis. If this is present with a normal heart rate or bradycardia, and a normal or high blood pressure it is not due to shock. Overly aggressive fluid resuscitation in these circumstances will worsen cerebral oedema.

Nurse patient in head-up position, 20-30 degrees from horizontal. Avoid inserting central venous lines into the internal jugular vein as this impedes venous drainage of the head and the insertion of the line may exacerbate the raised intracranial pressure.

## REFERENCES FOR FURTHER READING

1. Booy R, Habibi P, Nadel S, de Munter C, Britto J, Morrison A, Levin M. Reduction in case fatality rate from meningococcal disease associated with improved healthcare delivery. *Archives of Disease in Childhood*. November 2001;85:386-390
2. Thorburn K, Baines P, Thomson A, Hart CA. Mortality in severe meningococcal disease. *Archives of Disease in Childhood*. November 2001;85:383-385
3. Welch, S B and Nadel S. Treatment of meningococcal infection. *Archives of Disease in Childhood*. July 2003; 88:608-614.
4. Pollard AJ, Britto J, Nadel S, DeMunter C, Habibi P, Levin M. Emergency management of meningococcal disease. *Archives of Disease in Childhood*. March 1999;80:290-296
5. Cartwright K A V, Early management of meningococcal disease. *Infectious Clinics of North America*. September 1999;13(3):661-683
6. Nadel S, Levin M, Habibi P. Treatment of meningococcal disease in childhood. In : Cartwright K, ed. *Meningococcal Disease*. Chichester: John Wiley and Sons, 1995:207-43.



Estimate of child's weight (1-10 years)

Weight (kg) = 2 x (age in years + 4)

Systolic blood pressure = 80 + (age in years x 2)

N.B. Low BP is a pre-terminal sign in children

### Normal Values

Conscious Level	Age	Respiratory Rate/min	Heart Rate/min
Alert		30-40	110-160
Responds to Voice	<1	30-40	110-160
Responds to Pain	1-2	25-35	100-150
Unresponsive	2-5	25-30	95-140
	5-12	20-25	80-120
	>12	15-20	60-100

Observe HR,RR,BP,Perfusion, Conscious Level  
Cardiac monitor and pulse oximetry. Take blood for Glucose, FBC, Clotting, U&E, Ca<sup>++</sup>, Mg<sup>++</sup>, PO<sub>4</sub>, Blood cultures, Blood Gas (bicarb, base deficit), Cross-match

Colloid bolus (20ml/kg)

4.5% Human Albumin Solution (or Fresh Frozen Plasma or Hemacel/Gelofusine) i.v. or intra-osseous

Inotropes

Dopamine or Dobutamine 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 line only at 0.1 mcg/kg/min. Make up 300 mcg/kg in 50 ml of saline at 1 ml/hour=0.1 mcg/kg/min.

Intubation (call anaesthetist)

Atropine 20 mcg/kg (max 600 mcg) AND Thiopentone 3-5 mg/kg AND Suxamethonium 2 mg/kg (caution, high potassium) ETT size = age/4 + 4, ETT length (oral) = age/2 + 12, Then: morphine (100 mcg/kg) and midazolam (100 mcg/kg) every 30 mins

Hypoglycaemia (Glucose < 3 mmol/l)

5ml/kg 10% dextrose bolus i.v. and then dextrose infusion at 80% of maintenance requirements over 24 hours

Correction of metabolic acidosis pH < 7.2

1 mmol/kg NaHCO<sub>3</sub> i.v. = 1 ml/kg 8.4% NaHCO<sub>3</sub> over 20 mins or 2 ml/kg 4.2% NaHCO<sub>3</sub> in neonates

If K<sup>+</sup> > 3.5 mmol/l

Give 0.25 mmol/kg over 30 mins i.v. with ECG monitoring  
Caution if anuric

If total Calcium < 2 mmol/l or ionized Ca<sup>++</sup> < 1.0

Give 0.1 ml/kg 10% CaCl<sub>2</sub> (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)

If Mg<sup>++</sup> < 0.75 mmol/l

Give 0.2 ml/kg of 50% MgSO<sub>4</sub> over 30 mins i.v. (max 10 ml)

Prophylaxis of household contacts

Inform Public Health Department, Give 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 as single 500 mg dose (adults only)

Diagnosis

Blood cultures, throat swab, whole blood (EDTA specimen) for PCR, rapid antigen test. Aspirations/scrapings from skin showing haemorrhagic rash

Serology

For suspected cases with no isolate or where PCR does not identify serogroup, clotted blood sample to reference laboratory\* (acute within 72 hrs and convalescent 10-28 days after presenting symptoms)

Samples from hospitals in England, Wales and Northern Ireland

\*HPA Meningococcal Reference Unit

Tel: 0161 276 6757 Fax: 0161 276 6786

Samples from hospitals in Scotland

\*Scottish Meningococcus and Pneumococcus Reference Laboratory

Tel: 0141 201 3836

For further copies of this resource call Meningitis Research Foundation 01454 281811

\* A.J. Pollard, S. Nadel, P. Habibi, S.N. Faust, I. Maconochie, N. Mehta, J. Britto, M. Levin (1998). Department of Paediatrics, Imperial College School of Medicine, St Mary's Hospital, London W2  
Rev 03/03 (\*Arch Dis Child, March 1999; 80: 290-296)

# Early Management of Meningococcal Disease in Children\*

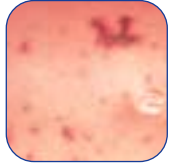
4th Edition



## RECOGNITION

May present with predominant SEPTICAEMIA (with shock), MENINGITIS (with raised ICP) or both. Purpuric/petechial non-blanching rash. Rash may be atypical or absent in some cases.

- Call consultant in A&E, Paediatrics, Anaesthesia or Intensive Care.
- DO NOT ATTEMPT LUMBAR PUNCTURE
- Initial assessment, looking for features of early shock/raised ICP
- i.v. Cefotaxime (80 mg/kg) or Ceftriaxone (80 mg/kg)



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### SIGNS OF EARLY COMPENSATED SHOCK ?

- Tachycardia
- Cool peripheries/pallor
- Increased capillary refill time (> 4 sec)
- Tachypnoea/pulse oximetry < 95%
- Hypoxia on arterial blood gas
- Base deficit (worse than -5 mmol/l)
- Confusion/drowsiness/decreased conscious level
- Poor urine output (< 1ml/kg/hr)
- Hypotension (late sign)

YES

- ABC and Oxygen (10 l/min), bedside glucose
- Insert 2 large i.v. cannulae (or intra-osseous)

### VOLUME RESUSCITATION

- Colloid bolus (20 ml/kg 4.5% HAS) and review
- Repeat colloid bolus if necessary
- Observe closely for response/deterioration
- Do not attempt lumbar puncture

After 40 ml/kg to 60 ml/kg fluid resuscitation  
STILL SIGNS OF SHOCK ?

NO  
Repeated Review

### WILL REQUIRE ELECTIVE INTUBATION AND VENTILATION

- Call anaesthetist and contact PICU
- Continue boluses of 10-20 ml/kg of colloid
- Start peripheral inotropes (Dopamine, Dobutamine)
- Nasogastric tube and urinary catheter
- Consider cuffed ET Tube and CXR
- Anticipate pulmonary oedema (consider PEEP)
- Central venous access
- Start Adrenaline infusion (central) if poor response to volume resuscitation and peripheral inotropes

Anticipate, monitor and correct:

- Hypoglycaemia
- Acidosis
- Hypokalaemia
- Hypomagnesaemia
- Hypocalcaemia
- Anaemia
- Coagulopathy (fresh frozen plasma 10 ml/kg)
- Raised intracranial pressure

### RAISED INTRACRANIAL PRESSURE ?

- Decreasing or fluctuating level of consciousness
- Hypertension and relative bradycardia
- Unequal, dilated or poorly reacting pupils
- Focal neurological signs
- Abnormal posturing or Seizures
- Papilloedema (late sign)

YES

- ABC and Oxygen (10 l/min), bedside glucose
- Give Mannitol (0.25 g/kg) bolus followed by Frusemide (1 mg/kg)
- Steroids (Dexamethasone 0.4 mg/kg bd x 2 days)
- Treat shock if present

Call anaesthetist and contact PICU

- Intubate and ventilate to control PaCO<sub>2</sub> (4-4.5 kPa)
- Urinary catheter and monitor output, NG tube
- Do not attempt lumbar puncture

### NEUROINTENSIVE CARE

- 30° head elevation, midline position
- Avoid internal jugular lines
- Repeat Mannitol and Frusemide if indicated
- Sedate (muscle relax for transport)
- Cautious fluid resuscitation (but correct coexisting shock)
- Minimal handling, monitor pupillary size and reaction

### 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 (18 mg/kg over 30 min i.v. with ECG monitoring)

If persistent seizures

- Thiopentone 4 mg/kg in intubated patients (beware of hypotension)
- Midazolam/Thiopentone infusion

NO

### CLINICAL FEATURES OF MENINGITIS ?

YES

Dexamethasone  
(0.4mg/kg bd x 2 days)

NO

Close monitoring for signs of raised ICP and repeated review

Repeated Review

Transfer to Intensive Care

www.meningitis.org

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## CONTRAINDICATIONS TO LUMBAR PUNCTURE:

- Prolonged or focal seizure
- Focal neurological signs (including ocular palsies)
- Widespread purpuric rash in ill child
- Glasgow coma score <13
- Papillary dilatation
- Impaired oculocephalic reflexes
- Abnormal posture
- RICP: inappropriately low pulse, elevated blood pressure and irregular respirations (indicating impending brain herniation)
- Coagulopathy
- Papilloedema
- Hypertension

## About Meningitis Research Foundation

Meningitis Research Foundation is a national registered charity whose vision is a world free from meningitis and septicaemia. The Foundation operates a **Freefone** 24 hour helpline **080 8800 3344**. At all hours of the day or night, trained staff and nurses speak to callers concerned about meningitis and septicaemia, provide information for people dealing with a case, and offer support and befriending to patients and their families, whether they are currently ill, recovering, coping with after effects, or bereaved.

On the basis of research and consultation with health professionals and their national associations, the Foundation produces guidance notes and protocols to promote best practice in the diagnosis and treatment of patients with these diseases. Along with the algorithm overleaf, these include:

- [Early Recognition of Meningitis and Septicaemia: Vital Signs for Frontline Nurses](#)
- [Early Management of Suspected Bacterial Meningitis and Meningococcal Septicaemia in Adults](#)
- [Meningococcal Meningitis and Septicaemia: Guidance Notes for Diagnosis and Treatment in General Practice](#)

The Foundation organises targeted scientific and medical conferences and other training events.

In addition to resources for health professionals, the charity produces a range of materials for patients, including the booklet [Meningitis and Septicaemia – What Happens Next?](#) explaining the possible sequelae of meningitis and septicaemia. All of these resources can be obtained free of charge by calling any of our offices, or through our website.

Funding for the charity's work is almost entirely from voluntary donations.

For further copies of this handbook, or for information on the services that Meningitis Research Foundation provides contact us at:

[www.meningitis.org](http://www.meningitis.org)

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