FORM 37

Rule 60(1)

FINDING INTO DEATH WITH INQUEST

Section 67 of the ***Coroners Act 2008***

Court reference: 2614/06

Inquest into the Death of ADAM MICHAEL FRANCIS FABRE

Delivered On:	24 March, 2010
Delivered At:	Coroners Court of Victoria, Melbourne
Hearing Dates:	21st October, 2009
Findings of:	JUDGE JENNIFER ANN COATE
Representation:	Mr John SNOWDON, Southern Health

Place of death/Suspected death: Dandenong & District Hospital.

SCAU	Leading Senior Constable G. McFarlane

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Section 67 of the ***Coroners Act 2008***

Court reference: 2614/06

In the Coroners Court of Victoria at Melbourne,

I, JUDGE JENNIFER ANN COATE, State Coroner

Having investigated the death of :

Details of deceased:

Surname: FABRE
First name: ADAM
Address: 18 Kensington Place, Narre Warren, Victoria, 3805

AND having held an Inquest in relation to this death on 21 October, 2009 at Melbourne find that the identity of the deceased was ADAM MICHAEL FRANCIS FABRE and death occurred on the 15th of July, 2006 at Dandenong & District Hospital, David Street, Dandenong, Victoria from

1. ACUTE BACTERIAL MENINGITIS WITH SECONDARY CEREBRITIS (MENINGOCOCCUS)[1]

In the following circumstances:

Background summary:

1. 19 year old Adam presented in the early hours of the morning of July 14, 2006 to Casey Hospital with symptoms and signs of fever, headache, tachycardia, severe muscle pain, neck stiffness, vomiting and blanching rash. He had been unwell for about three days. There were differing views amongst the medical staff about whether or not he was experiencing photophobia and differing views about the nature of his neck stiffness. Adam was diagnosed with and treated for an upper respiratory tract infection. Meningitis was considered and rejected as a cause of his illness. Adam was treated with painkillers, IV saline and anti-inflammatory medication. His mother, who was present with him was advised by about 8am that Adam was to be discharged home. She raised a protest at such a course. About 6 hours after Adam's presentation, he suffered a tonic-clonic seizure. He was noted to have a Glascow Coma Score of 4 after which he was intubated, given broad spectrum antibiotics and had a CT scan showing widespread oedema. He was transferred to Dandenong Hospital Intensive Care Unit. Adam died at 1.35pm on July 15,

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[1]. Statement of Dr Noel Woodford Forensic Pathologist, Victorian Institute of Forensic Medicine

2006. His cause of death was found to be acute bacterial meningitis with secondary cerebritis (meningococcus).[2]

Circumstances:

2. Adam Fabre was born on November 30, 1986. He was the much loved son of Christiane Fabre and Patrice Fabre. He lived at home with his mother at the time of his death at 18 Kensington Place, Narre Warren.

3. At about 3.45am on Friday July 14th, 2006, Adam[3] was taken by his mother to the Emergency Department of Casey Hospital in Berwick. Mrs Fabre, in her statement, described Adam's condition at this time as having "*a high temperature, vomiting, stiff neck and cluster rashes on his hands and feet.*"[4]

4. Adam was triaged by Nurse Matthew Swales, a senior emergency nurse and clinician who recorded in his nursing assessment as follows:

> *"2/7 days of temps headache and vomiting, non-petechial rash. Reports pain at back of neck. Reluctant to touch chin to chest, otherwise good neck movement. Nil photophobia. Skin warm and dry. Feels muscles are saw(sic).''*

5. Nurse Swales[5] had measured observations of Adam's temperature at 38.5 and a pulse rate of 120. At triage, Adam was given a category 5 triage rating. This category was described as "*non urgent: to see within two hours*". As it happened, it was not a busy time at the hospital and Adam was seen within six minutes of his presentation.[6]

6. Nurse Swales stated he was aware of the possibility of meningococcal infection but did not consider that Adam presented with symptoms of anything more serious than a modest viral illness.[7]

7. Adam was taken into an emergency department cubicle and then seen by RN Vianney Allison, a nurse of 18 years experience who had been working at Casey Hospital Emergency Department since February 2005. Nurse Allison assessed Adam as photophobic. She found him to be in severe pain which Adam rated at 10 out of a maximum of 10. RN Allison accepted Adam's assessment of his pain level on the basis of his whole body language. RN Allison stated

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2. Statement of Dr Noel Woodford Forensic Pathologist, Victorian Institute of Forensic Medicine
3. The family's permission was sought to refer to Adam by his first name.
4. Exhibit 1: Letter of Mrs Fabre dated July 24th, 2006.
5. Nurse Swales described himself as an experienced Emergency Triage Nurse in his statement prepared for the Coroner.
6. Hospital records
7. Statement of Nurse Swales in Inquest Brief 20.8.08

that Adam complained of neck stiffness on forward extension of his chin to his chest. She noted a blanching rash which turned white under pressure. She noted when Adam was stripped to his underpants that the rash was full body but not typical of meningitis.[8]

8. Dr Pin Pin Diaz was the junior resident working in the Emergency Department at Casey Hospital when Adam came in on that morning. Dr Elsa Pin Pin Diaz was present when RN Allison examined Adam.

9. Dr Pin Pin Diaz ("Dr Diaz")qualified as a medical practitioner in the Philipines in 1990. However, until Dr Diaz entered the medical graduate program in Australia, she had not practised as a treating doctor for a number of years. In 2005 Dr Diaz entered the International Medical Graduate Program conducted by Southern Health and participated in that program from September 2005 to March 2006. Dr Diaz had commenced work in the Emergency Department at Casey Hospital on July 3, 2006. That is, when Adam presented at the hospital, Dr Diaz had been in the Emergency Department for 2 weeks.

10. RN Allison stated that at 4:35 a.m. Adam was administered oral Panadeine Forte which he vomited a short time later. RN Allison stated she had a discussion with Dr Diaz after which time an intravenous line was set up and blood samples were taken and a venous blood gas test was carried out on Adam, which was normal.

11. At around 5:15 am RN Allison states that Adam was administered Panadol per rectum. She further stated that Maxolon 20 mg was administered to Adam at around 5:25 am and normal saline was continued intravenously at 5:45 am.

12. At around 6:30 am according to RN Allison, Adam complained to her and Dr Diaz of neck pain. At that time Adam was assessed as febrile. RN Allison stated that Dr Diaz went to consult with the senior medical officer on duty, Dr Darren Le Brocque.

13. According to RN Allison when Dr Diaz returned to the cubicle, she ordered 400 mg of Neurofen which was given to Adam at 6:45 am.

14. RN Allison stated that she spoke to Dr Le Brocque at about 6:30am to 6:45am and told him she was concerned that Adam was in pain and that they were not "*on top of that pain*".

15. RN Allison also told Dr Le Brocque that she was concerned about the apparent inexperience of Dr Diaz. RN Allison stated that on two previous occasions earlier in that morning she had asked Dr Le Brocque to come and review Adam.

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8. Statement of Nurse Allison: Inquest Brief 19.7.08

16. RN Allison spoke to the senior nurse on duty that morning before handover and told her that she was concerned about Adam's condition. She further stated that at the day shift handover at around 7:15 am she further explained her concern about Adam's ongoing pain and increased temperature as well as her view that the medical team were not "*on top of his condition.*"

17: It was Dr Diaz's evidence that on the night of the 13th of July 2006 she started work at approximately 11 p.m. Dr Diaz stated that she took a history from Adam's mother upon his presentation that he had been unwell for approximately 3 days, experiencing fever off and on with muscle aches. Dr Diaz formed the initial view that Adam was suffering from a viral illness. In oral evidence, Dr Diaz confirmed that her initial diagnosis was that Adam had an upper respiratory tract infection.[9] Dr Diaz, consistent with the evidence of RN Allison, stated that she ordered Panadeine Forte for Adam which Adam vomited back up.

18. Dr Diaz then consulted Dr Le Brocque.

19. After that consultation, Dr Diaz inserted an intravenous line and commenced saline solution. In her statement, Dr Diaz noted consistent with RN Allison, that Panadol was then given per rectum and Maxolon was given intravenously. Dr Diaz's assessment was that Adam's condition seemed to settle after the administration of Maxalon but she remained concerned that his temperature was still elevated. Dr Diaz prescribed Brufen orally at about 6:45 a.m. Adam's temperature rose from 38.6 at 6:30am to 39.2 at 7:15 am.

20. Dr Diaz stated she had a further discussion with Dr Le Brocque and advised him that the patient's temperature was not coming down, despite administration of Brufen. Dr Diaz stated she discussed with Dr Le Brocque the question of blood tests at that time.

21. It was the evidence of Dr Diaz that she checked Adam's condition frequently and although he looked unwell, he stated that he felt okay except for his ongoing headache. Dr Diaz stated that Adam reported that his headache was not as severe as it had been previously.

22. Dr Diaz stated that she had previously been involved in the care of patients with meningitis and was familiar with the presenting symptoms. She stated that she conducted a neck examination which revealed to her that Adam could rotate his head and look left to right without pain although he did complain of pain on touching his chin to his chest.

23. Dr Diaz noted that although Adam told her that he preferred the examination cubicle lights to be dimmed, Adam "*did not exhibit any obvious evidence of photophobia.*"

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9. Transcript p 53

24. She also noted that although Adam had a rash on the anterior side of his legs, it appeared to be "*a blanching rash*" and consequently Dr Diaz did not consider it suggestive of meningitis.

25. Dr Darren Le Brocque was the senior medical officer on duty that night. As at July 2006 he had been working in the Emergency Department at Casey Hospital for approximately 2 years. He accepted that he was responsible for the supervision of Dr Diaz during the night that Adam presented to the Emergency Department.

26. Dr Le Brocque gave a very short written statement which formed part of the Inquest Brief. In that statement Dr Le Brocque stated that he had not at any stage attended to examine Adam. Dr Le Brocque noted that when the handover to the day shift took place at about 8am, Adam reported that his headache was worse and his temperature was elevated and it was at that time that his blood sample was sent for testing and a chest x-ray was taken.

27. In oral evidence during the Inquest Dr Le Brocque stated that, given the symptoms Adam presented with and given that he had been unwell for 3 days, he did not think Adam was at "*high risk of having meningitis*".[10]

Family Concerns

28. Before turning to the medical issues, during the course of the investigation, Adam's mother and father both wrote letters to the Coroner. In the family letters, a few discrete issues were raised about Adam's treatment and communications with the family. I note that Mr Snowden on behalf of Casey Hospital raised no objection to those letters going into evidence without requiring any cross examination.

(i) Sending Adam home:

29. Adam's mother raised her concern that the Hospital was trying to send Adam home on this morning and that it was only at her insistence that Adam remained at the Hospital. This was put to Dr Diaz in oral evidence. Initially she denied that she had tried to send Adam home. However, when the detail of Mrs Fabre's letter was put to her about what she had said to Mrs Fabre, that is, that Adam had a virus and that he was going to be sent home, Dr Diaz implicitly agreed that this was said by responding as follows:

> *"Because that's what I got from my senior. He....he doesn't think that this is really something serious because any viral illness can be presenting with a high temperature".*[11]

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10. Transcript p 68
11. Transcript p 55

30. Later in her evidence Dr Diaz confirmed that Dr Le Brocque had told her she could send the patient home with a management plan for a viral illness for the family to care for Adam that included giving him Panadol and lots of fluids.[12] When Dr Le Brocque was asked about this, he stated that he did not recall advising Dr Diaz that Adam could go home, but agreed the general plan with a viral illness would be to send someone home, although after some improvement is seen.

Conclusion:

31. Given the evidence of Mrs Fabre in her statement, effectively confirmed by Dr Diaz, and given Dr Le Brocque's evidence that he was of the view that it was a viral illness and the normal plan would be to discharge, I am satisfied that Mrs Fabre was told by Dr Diaz that Adam was to go home.

(ii) Delay in response to Adam's condition:

32. The issue about the lack of appropriate diagnosis and its impact on Adam's chances of survival lie at the heart of this investigation and thus goes to the overall conclusions in this investigation. However, understandably, the family have raised concerns that go to aspects of what was said and done during the hours of Adam's stay at Casey Hospital. Much of this information is consistent with the overall picture that the treating doctors did not assess Adam's condition as serious until he went into seizure. For example, in Mrs Fabre's letter she stated that just after the changeover shift as new nurses were arriving, she drew attention to Adam's condition. She told nurses that Adam was "*acting strange*" and he was moaning. He had his arms up in the air and was twisting his fingers. Mrs Fabre stated that she tried to get the attention of a nurse who told her she would get the doctor. It was Mrs Fabre's evidence that several minutes past before the nurse walked past again and asked had the doctor been yet. When the nurse came closer to Adam, she then pushed the emergency button and doctors came rushing in.

33. I note that Dr Summers summarises the nursing and doctor's notes, and describes this scene somewhat differently. Dr Summers was not present and has relied on the notes to summarise what happened. According to the notes, Adam developed a tonic-clonic seizure somewhere around 8.45 to 9am whilst being examined by Dr Wei Lum and then a code blue was called. Adam was thereafter treated with a range of medications, including antibiotics and anti-seizure medication and intubated and placed on a ventilator.

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12. Transcript p 63-4

Conclusion:

34. Given that Mrs Fabre was not only present but would have been focussed only on Adam and his welfare, I am satisfied that Mrs Fabre's version of events is more likely to be the accurate one. I add to this conclusion that there were inadequacies in some of the hospital notes from which Dr Summers was working, and thus his second hand reliance on someone else's inadequate notes is not likely to be as reliable as the observations of Mrs Fabre.

(iii) Communication:

35. Both Mr and Mrs Fabre reported being told by a senior nurse (and apparently confirmed by a male doctor) at Casey Hospital that Adam had an 80% chance of recovery as he was young and strong. Sadly, by the time Adam reached the Dandenong Intensive Care Unit later that afternoon, his chances of survival were rated by the treating medical staff as nil according to the evidence of both Mrs Fabre and Mr Fabre. Mr Fabre indicated in court that even though it was extremely distressing information to receive at Dandenong Hospital, he appreciated being told exactly what the family were facing.

The Medical Evidence/Issues

Triaging

36. Upon arrival at Casey, Adam was triaged at Category 5. Although nothing turns on it as Adam was seen within 6 minutes of arrival, it was the opinion of Dr Summers, Dr Chan and Dr Eddey that the triage category was too low. Dr Summers stated that whilst some aspects of clinical risk analysis are hard to define because they are based on clinical impression, the triaging in this case demonstrated a lack of realisation of the potential severity of Adam's presentation.[13]

Clinical examination of Adam

37. A detailed and considered statement was provided by Dr Ian Summers, the Deputy Director of the Emergency Department at Casey Hospital. This statement was adopted by Dr Chan, the Director of the Emergency Department at Casey Hospital who attended the inquest to give evidence.

38. Dr Chan gave evidence that clinical staff should have been alerted by the symptoms with which Adam presented. He gave unequivocal evidence that Dr Le Brocque should have personally examined Adam. He added that Dr Le Brocque's failure on this occasion was out of

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13. Statement of Dr Summers

character for Dr Le Brocque.[14]

39. Dr David Eddey, the Director of Emergency Medicine at Geelong Hospital, was requested to provide an independent expert opinion about the clinical management of Adam for the coronial investigation. Dr Eddey prepared a comprehensive, written opinion that was distributed to the family and Southern Health.[15] Dr Eddey completed that report dated May 18, 2009 after being provided with a range of documents and material which he has noted in his report.

40. Counsel on behalf of the Casey Hospital acknowledged Dr Eddey's expertise and did not take issue with Dr Eddey's opinion or his description of the various forms of meningococcal disease, symptoms and treatment contained therein. During the Directions Hearing for this inquest, Mr Snowden helpfully advised the Court that his client took no issue with the contents of Dr Eddey's report and did not require him for cross-examination. Each of the witnesses from Southern Health confirmed, when asked, that they accepted Dr Eddey's report and his conclusions and recommendations.

41. On the issue of the need for a personal clinical examination of Adam by the supervising senior doctor, Dr Eddey was clear that this should have happened. Dr Eddey stated that whilst it would be reasonable practice to expect that a junior doctor would discuss cases with the senior doctor, "*it is often very difficult to get a clear clinical impression from a verbal report from a junior doctor, particularly in the absence of pathology tests that might assist with decision making in these circumstances.*"

42. It was clear from Dr Diaz's evidence that she felt unable to assert herself against the senior doctor, Dr Le Brocque, despite being very concerned about Adam's condition.

43. When asked about the communication between herself and Dr Le Brocque and whether or not she felt she had conveyed how serious it was, Dr Diaz said in evidence:

> "*.....I did my best to come and see him and - and to express my concern but probably I am just so scared of insisting what I wanted because I'm - I'm new and I don't want to be like embarrassed or laughed at if I just tell that this is this and that is that's why I just decided to - to follow what he wanted and follow what he ordered to me*".

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14. Transcript p 92
15. That report is contained in Appendix 1 to this finding

Conclusion:

Clinical examination

44. The evidence is that both Dr Diaz and RN Allison were expressing concern to the senior supervising doctor about Adam's condition. Dr Le Brocque's evidence is that he did not examine Adam and he could not provide a reason as to why this was so. Both Dr Chan and Dr Eddey were clear that Dr Le Brocque should have attended upon Adam to personally examine him, given the differing symptoms being reported to him and the inexperience of the junior doctor reporting to him.

Diagnosis

45. Dr Summers, in his statement posed the question, should clinical staff have recognized that Adam was at serious risk of bacterial meningitis or bacterial sepsis? Dr Summers noted that RN Allison had recorded that Adam had a raised pulse rate but otherwise normal blood pressure. She noted a blanching rash. She also noted that Adam had body ache/pain which he rated as 10/10 severity. There were notations of "*chills*", neck stiffness and photophobia and that Adam had been unwell for three days.

46. Dr Diaz recorded that Adam had a three day history of cough, cold, frontal headache and fever and slight tenderness to the frontal sinus area with nasal discharge. She saw a rash on his *? Upper extremity* which she described as *macular, blanching and non-itchy*. She made a provisional diagnosis of an upper respiratory tract infection and treated him with Panadeine Forte and Brufen and Maxolon to reduce the vomiting and 2L of Saline intravenous solution.

47. A venous blood gas sample was taken and all values were normal. It was Dr Summers' opinion that this would have given a treating doctor some reassurance that he/she was not dealing with an overwhelming sepsis. It was Dr Summers' opinion that it was not obvious from the notes whether the treating clinicians should have recognized that the patient's illness might be bacterial. He did note however that the high fever and raised pulse rate and neck soreness were elements of concern.[16]

48. Dr Summers made the point that both viral illnesses and meningococcal sepsis can give a rash and in a small proportion of cases meningococcus can present with an atypical rash, such as in Adam's case. Dr Summers stated that meningococcal disease is a very difficult disease to pick from the larger group of patients with viral illnesses with similar symptoms, who present daily to the Emergency Department. He noted that even experienced senior doctors "*repeatedly missed*" the diagnosis. He assessed the major failing of the hospital on this occasion as a lack of

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16. Statement of Dr Summers

recognition of the potential seriousness of Adam's presentation and the lack of review of the patient by the senior doctor. He noted that the situation was compounded by a misdiagnosis at triage, an inexperienced junior doctor who lacked the confidence to push for a review by the senior doctor on duty and the delays in pathology services.

49. Dr Eddey was firmer in his view about the missed diagnosis. He stated that ,

> *"the constellation of symptoms and signs and the description of the patient as "looking unwell" suggests that the patient has a significant illness. The diagnosis of an URTI fails to recognise the significance of many of these signs and symptoms."*[17]

Neck stiffness:

50. Dr Eddey gave a very helpful analysis of Adam's presentation in the context of what is known about meningococcal disease.[18] For example, with respect to examining a patient with suspected meningitis or meningeal irritation, Dr Eddey stated:

> *"Most commonly one would examine the patient for neck stiffness or nuchal rigidity. In meningeal irritation when the neck is bent forward (or flexed forward) there is reflex (involuntary) spasm of the neck muscles, resisting the flexion. This gives the feeling of rigidity in the neck to the examining clinician. This occurs whether the patient is conscious or unconscious."*

Dr Eddey noted that patients with viral illness often complain of neck stiffness, but when examined have no meningism.

51. Dr Eddey was also concerned in his report to dispel some misconceptions about the rash commonly associated with meningococcal disease. He noted that both Dr Diaz and Nurse Swales wrongly concluded that the presence of a *"blanching or non-petechial rash was not suggestive of meningitis."* It was Dr Eddey's opinion that this demonstrated *"an ignorance of the pathophysiology and significance of a rash in meningococcal disease."*

Blood Tests

52. It was agreed between the doctors that blood tests were a *"useful adjunct"* to doctor's clinical assessments, in particular in borderline cases. Blood tests were taken for Adam at about 5am on July 14, but not sent off to the pathology laboratory until about 8.30am. The explanation for this was that the Hospital did not have an after hours pathology service on site after 10 or

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17. Report of Dr Eddey
18. Rather than quote extensively from Dr Eddey's report, I have appended a copy of the report to this Finding to assist those in the health sector who receive a copy of this Finding.

11pm. The service was not available until 8am each morning. The evidence was that pathology samples would normally be batched hourly and couriered to Monash Medical Centre in Clayton when testing was needed after hours.[19] Dr Chan conceded in evidence that there was less use made of pathology when the facility was not on site.

53. Blood test results were not available before Adam had his seizure. According to Dr Eddey, the test results demonstrated a raised white cell count and a grossly elevated CRP at 239. Dr Eddey stated that had these results been available to the treating medical team, it may have alerted them to the possibility or probability of a more serious illness. Dr Chan gave evidence that even if a blood sample had been sent off for Adam at 6am, the results would not have been available before about the time Adam went into seizure and serious deterioration.

General Principles of Diagnosis

54. Dr Eddey stated that clinical diagnosis required a doctor to look at the whole pattern of illness rather than for the presence or absence of any particular symptom or sign. He then went through an analysis of the missed opportunities for re-assessing the working diagnosis for Adam. For example, he noted that whilst the URTI (Upper Respiratory Tract Infection) may have been a reasonable working diagnosis at the first instance, at the point at which Adam's rapid pulse rate did not resolve with the IV therapy intended to treat the dehydration that was thought to be causing it, it would have been reasonable practice to review and reassess the working diagnosis. This did not happen in Adam's case and thus the treatment continued consistent with the URTI diagnosis.

55. Dr Eddey stated "*A conventional standard of care would expect that once treatment had failed to produce the expected improvement, that the patient would be reassessed and alternative diagnosis considered and other investigations expedited.*" Dr Eddey noted that a working diagnosis was made, tested with a treatment plan which did not produce sufficient apparent improvement but did not cause a review by the senior doctor or a revision of the diagnosis.

56. On a slightly different note, Dr Eddey commented that the 2 litres of saline solution that were administered for the working diagnosis of URTI, may have contributed to the deterioration of Adam, although unintentionally of course, as it may have accelerated the development of cerebral oedema.

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19. Transcript p 85 Evidence of Dr Chan

Conclusion:

Diagnosis

57. As stated above, Dr Le Brocque, when questioned about his diagnosis, stated "*he missed it*". Dr Eddey has analysed the symptoms and signs which were either missed or misinterpreted in the diagnosis of Adam. He has also made some considerable observations about the need to observe the whole pattern of illness when making a diagnosis. The weight of the evidence was that once the treatment plan for the working diagnosis was not producing sufficient apparent improvement in Adam, that should have caused a reassessment of the working diagnosis. It did not.

The administration of antibiotics

58. The diagnosis of Adam's condition and the timing of the administration of antibiotics to him were significant in this investigation. Dr Summers raised concerns about being too hasty to administer antibiotics. However, it was the evidence of Dr Chan that if a doctor is starting to think of meningitis, then the doctor should err on the side of caution and give antibiotics rather than not. He stated that in Adam's case, that at the point at which the nurses were observing and reporting vomiting, signs of photophobia and headache that was the time to administer antibiotics to him. Dr Chan agreed that the administration of antibiotics at that time would have given Adam a better chance of survival.

59. Dr Chan gave evidence that he agreed with Dr Summers that it was not possible to say whether the administration of antibiotics to Adam shortly after his presentation would have changed the outcome. Dr Chan said that Adam should have had antibiotics earlier noting that it could have made a difference but he did not know to what extent.[20]

60. Dr Eddey expressed a firm view that it was common practice not to wait for the return of blood results if it was thought that a patient probably had meningitis, antibiotics should be started.

61. Dr Summers, Dr Chan and Dr Eddey all agreed that it was not possible to say, even if Adam had been treated with antibiotics a short time after arrival at the hospital, if that would have made any substantial difference to the outcome.

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20. Transcript p 98

Conclusion:

Administration of antibiotics

62. Mr Snowden conceded in closing submissions that the weight of the evidence was that the failure to make a timely diagnosis and administer antibiotics to Adam reduced his chances of recovery. No doctor was able to say that the administration of antibiotics, even at the time of his arrival at the hospital, would have saved Adam.

Communication between medical and nursing staff and supervision or review

63. During this investigation, evidence emerged about a lack of responsiveness by a senior doctor to the expressed concerns of a junior doctor and an experienced nurse. It was the opinion of both Dr Chan and Dr Summers that the senior doctor who was supervising Dr Diaz on that evening should have personally reviewed Adam. As stated above, no explanation could be provided by Dr Le Brocque as to why this did not happen.

64. In the course of giving his opinion about why the review system did not work properly on this occasion, Dr Summers noted that there were a large number of residents on brief rotations at that time and this made it very difficult to work out who may need a higher degree of orientation or supervision.

65. Dr Summers further noted that at handover at 8 am there was a further opportunity for a review by a senior doctor. This opportunity was assessed by Dr Summers as a missed opportunity on the basis that the senior doctor at handover was under the impression that Adam had been personally reviewed by the senior doctor before him and a management plan was in place. This miscommunication gave the handover senior doctor a false sense of reassurance. I do note however that RN Allison told the senior nurse on duty that morning that in her view Adam's condition remained of concern. It is not clear what happened in response to that information.

Conclusion:

Communication between medical and nursing staff and supervision or review.

66. The evidence is that there was communication between Dr Diaz and her supervisor Dr Le Brocque and RN Allison and Dr Le Brocque, drawing his attention to Adam. Dr Le Brocque did not respond. This had the compounding effect of causing a miscommunication at handover as the morning senior doctor inferred that Adam had been properly assessed and there was no reason to reassess his diagnosis.

67. Finally, I note that no issue was taken by the family with the treatment Adam received after he commenced his seizures. There was no issue taken with his transfer to or treatment at Dandenong Hospital. Further, there was no issue of concern raised in the evidence before me as to Adam's treatment post seizure or his transfer to and treatment at Dandenong.

Comments:

68. The fact-finding role of the Coronial investigation has often been stated as one focused on establishing the truth of what happened as best the evidence allows, to achieve a range of purposes. A recognized significant purpose of the Coronial investigation is to identify any opportunities for improvement in systems of public health and safety which may contribute to the reduction in preventable deaths.

69. It is not an exercise in apportioning blame against individuals, even though it is acknowledged that the process of the investigation and inquest may well be perceived by those individuals involved in the death as such an exercise. However, to achieve improvements to our systems of public health and public safety, it may be necessary to put under scrutiny the actions of individuals trained in working inside those systems to highlight the need for improvement.

70. In this investigation, a number of issues about the Hospital's system came under scrutiny. The report of Dr Eddey and the statement of Dr Summers touched upon a number of areas that were either shortfalls in systems, poor diagnostic techniques or inadequate responses. The evidence is that Casey Hospital has done a series of things in the wake of Adam's death.

71. In evidence during the inquest, Dr Diaz was clearly very distressed. She was tearful throughout much of her evidence. She gave a powerful and heartfelt apology to Adam's mother and father and family present in the courtroom. She stated that at all times in her care of Adam "*she had done her best.*"

72. Dr Le Brocque made it clear in evidence that he now understood much more about the range of possible presentations of meningitis and that he "*totally missed it*" with Adam's presentation. He also apologised to the family.

73. He made a very frank statement in evidence that, despite the lack of experience of Dr Diaz and the concerning information that he was provided with by both Dr Diaz and RN Allison about the range of symptoms that Adam had, including some conflicting information about photophobia, neck stiffness and a rash, he did not attend to examine Adam. He agreed that despite at some stage being in the tearoom only 10 metres away from the cubicle in which Adam was lying, he did not visit him. Dr Le Brocque stated in evidence that "*he did not really have a*

reason" why he did not visit Adam and accepted that he should have done that.[21]

Changes Since Adam's Death at Casey Hospital

74. The evidence is that Casey Hospital has made a series of changes in the wake of Adam's death.

On-going Professional Developement at Casey Hospital

75. Dr Chan gave evidence that in the wake of Adam's death, everyone at his hospital has become a *"lot more vigilant about serious illness"*[22] and renewed its efforts in on-going professional development forums. In the statement provided by Dr Summers, he advised that, whilst both doctors and nurses take part in an education program that has included the topics of meningococcus sepsis/meningitis, these sessions were **repeated** as a result of Adam's death.

Protocols for disagreement over diagnosis/treatment

76. Dr Chan, gave evidence that, in the wake of Adam's death, the Hospital now has a written policy setting out what a junior doctor or nurse should do in the event that they feel they are not getting a response from the senior doctor. He detailed the protocol which sets out the steps a junior doctor or nurse may take in the event that a senior supervising doctor is not being responsive to their concerns. Dr Chan stated that the staff now have clear permission to go to the nurse in charge who has permission to call the on-call consultant.

New on-site pathology service

77. The Casey Hospital also now has a 24 hour on-site pathology service available to it which was not available at the time of Adam's death.

78. Given the appropriate range of responses and developments by Casey Hospital as set out above, it has not been necessary for me to make recommendations directed to Casey Hospital.

79. To ensure the appropriate level of awareness of the potential complexities of diagnosing meningococcal disease are brought to the attention of the practising medical community, I have directed a copy of this Finding (together with Appendix 1) be published on the Court's web-site and that a copy of this Finding (and Appendix) be distributed to the list of persons and agencies below.

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21. Transcript p 73 and 80
22. Transcript p 95 and 110

80. Finally, I note that the family generously wished to acknowledge their gratitude to the doctors for their openness and honesty during this inquest and did so through the Coroner's Assistant in open court and in the doctor's presence.

81. I wish to record my deepest sympathy to Adam's family for their loss.

Signature:

JUDGE JENNIFER COATE
State Coroner

Date: 24.3.2010

I direct that this Finding and the Appendix be published on the court's web-site and be distributed to the following:

Distribution List:

Mr Fabre
Mrs Fabre
Medico-legal Officer, Casey Hospital, Southern Health
The Hon. Minister for Health
Chief Health Officer, Department of Health
The Hon. Attorney-General
Medico-legal Officer, Monash Medical Centre
Medico-legal Officer, Dandenong Hospital
Dr David Eddey

Coroners Court of Victoria

Coroners Court of Victoria

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APPENDIX 1

2614/06
ADAM FABRE

Dr David Eddey,
MB,BS, DipRACOG, DTM&H, FACEM
Emergency Physician.

8-10 Gully Rd.,
Ceres,
Victoria, 3221.

18 May 2009,

Mr John Hildebrand,
Coroner's Registrar,
State Coroner's Office,
57-83 Kavanagh St,
Southbank,
Victoria 3006.

Re: Adam FABRE, dob 30/11/1986 Coroner's Case No. 2614/06

Thank-you for asking me to provide an opinion on this matter.

Please find my report including the following:

1. Summary of qualifications and experience
2. Material viewed
3. Hindsight Bias and Outcome Bias
4. Specific issues and opinion
5. Summary
6. References

Summary of Qualifications and Experience

In summary my qualifications, experience and current positions are:

- Bachelor of Medicine and Bachelor of Surgery (Monash University) 1983.
- Diploma of the Royal Australian College of Obstetricians and Gynaecologists 1987.
- Fellowship of the Australasian College for Emergency Medicine 1991.
- The Diploma of Tropical Medicine and Hygiene (Liverpool) 2003.

Currently I am employed in the full-time practice of Emergency Medicine and hold the positions of:

- Director of Emergency Medicine at The Geelong Hospital (1995-present).
- Staff Specialist in Emergency Medicine at The Geelong Hospital (1992-present).
- Member of Geelong Hospital Mortality Review Committee.

- Honorary Emergency Physician, Alfred Hospital, Melbourne.
- Honorary Forensic Physician with the Victorian Institute of Forensic Medicine.
- Area Medical Officer, Barwon Region, Ambulance Victoria.
- Member of Medical Standards Committee, Ambulance Victoria.
- Member of South West Region Consultative Committee on Critical Care and Emergency Services (Department of Human Services, Victoria).
- Member of Court of Examiners, Australasian College for Emergency Medicine.

In the past I have held the following positions:

1. Intern and Resident Medical Officer, Alfred Hospital Melbourne 1984-5.
2. Hospital Medical Officer – (Obstetrics and Gynaecology and Emergency Medicine) North West General Hospital, Burnie, Tasmania 1986.
3. Paediatric Registrar and Neonatal Emergency Transport Service (NETS) Registrar, Royal Women's Hospital, Melbourne 1987. Part-time NETS Registrar 1988-9
4. Emergency Medicine Registrar, Anaesthetics Registrar, Medical Registrar, Box Hill Hospital 1988-9.
5. Emergency Medicine Registrar, Prince Henry's Hospital, Monash Medical Centre and Moorabbin Hospital 1990.
6. Emergency Physician, Monash Medical Centre and Director of Emergency Department, Moorabbin Hospital 1991.
7. Locum Consultant in Emergency Medicine, Cork University Hospital and Mercy University Hospital, Cork, Ireland Feb-Sept 2003.
8. Clinical attachment in Paediatric Intensive Care, Royal Liverpool Children's Hospital, Liverpool, United Kingdom, Dec 2003-Jan 2004.

Material Viewed

1. Inquest brief Case No 2614/2006 including relevant statements from
    a. Christine FABRE
    b. Matthew SWALES
    c. Vianney ALLISON
    d. Dr Else PIN PIN DIAZ
    e. Dr Darren LE BROCQUE
    f. Dr Ian SUMMERS
    g. Dr Thomas CHAN
    h. Dr Noel WOODFORD
2. Medical Records from Casey Hospital

Hindsight and Outcome Bias

Every attempt has been made to minimize hindsight and outcome bias in the preparation of this report. Such bias can never be completely eliminated when outcomes are known or suspected from the outset. This fact has been well demonstrated in experimental psychology literature[1,2]. Hindsight bias refers to the tendency of those with outcome knowledge to exaggerate the extent to which they would have predicted the event beforehand. Outcome bias refers to the influence of outcome knowledge upon retrospective evaluations of decision quality. This opinion will clearly be subject to both hindsight and outcome bias.

It should also be emphasized that this opinion is generally based on documented medical records. I was therefore not placed in an identical position to the involved clinicians who were no doubt exposed to additional data and distractions, which may have made assessment and diagnosis more difficult.

Case Overview and Course of Events

Casey Hospital

14 July 2006

Adam FABRE arrives at hospital and triaged at 0344 hours

Presenting problem recorded as "febrile, feels unwell"

Triaged by Registered Nurse Matthew SWALES

Attended by Dr Else PIN PIN DIAZ and Nurse Vianney ALISON

Triage Nurse (Matthew SWALES) Assessment:

- 2 days headache vomiting, non-*petechial*[a] rash. Reports pain at back of neck. Reluctant to touch chin to chest, otherwise good neck movement, nil photophobia. Skin warm and dry. Feels muscles are saw (sic).
- Observations: Temp = 38.5^{oC}, Respiratory Rate = 16, Pulse Rate = 120

Doctor's (Dr Elsa DIAZ) Notes (Dated 14/07/06. No time or signature.)

- Had coryza (runny and or blocked nose) and cold and headache (frontal/sinus area) for the past 3 days.
- Had fever and chills, vomited several times
- Complaining of muscle aches everywhere
- Took fiorinal last night, didn't help, took panadeine forte and vomited again.
- Headache became worse
- Not exposed to anyone with same or similar
- Rashes noted on lower extremities yesterday pm
- (?) pain in the neck

- Allergies – NKA (nil known allergies)
- Past Medical Hx (history) – asthma-childhood,
- Not on any medications/over the counter drugs
- Social – patient works as a technician

Examination

- Conscious, coherent, looks unwell
- Tenderness frontal sinus area
- Can move head side to side
- (+) difficulty/pain doing chin-chest touch
- Kernigs sign[b] negative
- Pearly TM's (tympanic membranes or eardrums)
- (+) nasal discharge
- (-) sore throat
- chest clear
- heart – tachycardic (120 bpm)
- glabellar tap non-tender
- macular[c] rash, blanching

Impression

- URTI (upper respiratory tract infection)

Plan

- Panadeine forte – vomited
- IV fluids 2 litres normal saline stat.
- Panadol PR
- Maxolon 20 mg IV(metoclopramide) – antiemetic medication
- Brufen (ibuprofen) 400 mg

No further medical notes appear until 0845 hours

Nursing (Vianney ALLISON) Notes

- Headache since Wednesday
- Chills, Muscle soreness, rash
- Vomited x5
- Neck stiffness
- Pain score 10/10

0350

- A (airway) patent,

- B (breathing) normal, chest clear, nil wheeze, RR 16
- C (circulation) Tachycardic, normotensive (normal blood pressure)
- GCS (Glasgow Coma Score) 15 (out of a maximum 15)
- PEARL (pupils equal and reactive to light)
- Blanching rash – full body
- Pain 10/10 bodyache, chills, neck stiffness, photophobia (discomfort and reluctance to look at light)
- Overweight 19yo unwell since Wednesday on panadol and fiorinal
- Vomiting, nil diarrhoea

0445

- Panadeine forte, vomiting post

0513

- IV inserted by Dr Elsa
- VBG (venous blood gas) done – WNL (within normal limits)
- Normal saline 2 litres commenced
- Panadol PR

0545

- N Saline 1000 commenced

0630

- Patient complaining of neck pain, febrile, saline completed
- Seen by Dr Elsa and discussed with Senior MO. Nurofen 400mg given
- T = 38.4, Pulse = 116

0715 handover

- Patient reporting pain not changed with nurofen, although patient asleep on first examination. T = 39.4. MO aware.

0820

- Patient seen by Dr Wei Lum. Blood sent for pathology. Patient for CXR (chest x-ray)

0900

- Patient acting strange according to mother. Patient became rigid and unresponsive, GCS 8. Monitor attached, BSL (blood sugar level) 7.9 mmol. 15 litres Oxygen given by HM (Hudson Mask).

Patient is subsequently intubated, placed on a ventilator and commenced on appropriate antibiotic and antiviral therapy to cover both bacterial and viral causes of central nervous system infection.

He is then transferred to the Intensive Care Unit at Dandenong Hospital where he dies on 15 July 2006.

I will not comment upon the remaining care undertaken after transfer as it is not concerned with the issues being examined from the care at Casey Hospital.

Notes on Meningococcal disease

Meningococcal disease is caused by a bacteria Neisseria menigitidis (commonly referred to a meningococcus). This is a common bacteria which lives in the throat and in this community is carried by up to 25% of the population. The vast majority of people who carry the bacteria remain asymptomatic. In a very small proportion of people who carry or come into contact with meningococcus, the bacteria becomes invasive and causes disease. It is not clearly known why this occurs, but it is likely to be a combination of patient factors such as genetic background, immune status, environmental factors and the properties of the particular strain of the organism.

Once the bacteria has invaded the blood stream the clinical expression of infection may range from a transient self limited illness to an overwhelming septicaemia or to meningitis without septicaemia.

The most common presentations of meningococcal disease in this community are of septicaemia or meningitis or a combination of the two. Septicaemia may occur without meningitis and meningitis may occur without septicaemia. They may occur together.

Septicaemia occurs when the bacteria enters the blood stream and multiplies. In the early stages the presence and multiplication of the meningococcus (or any other bacteria) in the blood stream causes fevers and 'rigors'. Rigors are episodes of violent shaking associated with fever, sweats and feelings of extreme unwellness. They are very significant symptoms if they occur. Meningococcal septicaemia has a very high mortality rate, in the order of 40% or more.

The bacteria cause damage to blood vessels, resulting in the characteristic rash that is characteristically seen with meningococcal septicaemia. General effects on the body are caused by 'endotoxins' produced by the bacteria and by substances produced by the body in response to the bacteria and its endotoxins. These effects include depression of cardiovascular function (low blood pressure and cardiac failure), widespread damage to blood vessels leading to organ damage and failure (eg brain, kidneys, heart) and failure of the clotting system (a condition called DIC - disseminated intravascular coagulation). Damage to blood vessels and tissues in the limbs can be so severe as to cause limb loss.

This process, once it becomes established, may become rapidly progressive leading to collapse and rapid death of the patient. This is often referred to as a fulminating infection. The further along the path of this process one goes, the greater the damage to body organs and their function and the more likely one is to have a bad outcome such as permanent organ damage, brain damage, limb loss or death. Once established these processes are difficult to reverse, hence the importance of early recognition and aggressive treatment.

Meningococcal infection may become localised to the tissues covering the brain (the meninges) resulting in meningitis. This may occur with or without co-existing infection in the blood. In the presence of meningitis only, one

would not expect to see the characteristic rash and, in the early stages at least, the progression of the disease may not be as rapid as with septicaemia. Meningococcal meningitis without septicaemia is not clinically distinguishable from other forms of bacterial meningitis

Treatment of meningococcal infection is with antibiotics and supportive care as needed. This might include drugs to help maintain cardiovascular function as well as other intensive care therapies.

In 2003, Yung and McDonald published an article in the Medical Journal of Australia outlining an approach to the early diagnosis of meningococcal disease. They list the common signs and symptoms of meningococcal disease in children and adults, as found on the Victorian Department of Human Sevices Infectious Diseases website[4]:

- Fever, pallor, rigors, sweats
- Headache, neck stiffness, photophobia, backache, cranial nerve palsy
- Vomiting or nausea, and sometimes diarrhoea
- Lethargy, drowsiness, irritability, confusion, agitation, seizures or altered conscious state
- Moaning, unintelligible speech
- Painful or swollen joints, myalgia, difficulty in walking.
- While the absence of a rash does not exclude meningococcal disease, any haemorrhagic rash should be particularly noted.

The article is appended to this report, but several other salient comments from this journal article include:

- **Only 40% of patients with invasive meningococcal disease present with a haemorrhagic rash**
- Meningitis is the most common clinical syndrome (80%–85% of cases). The diagnosis is relatively straightforward when the patient presents with the typical clinical picture (fever, headache, vomiting and change in conscious state), and treatment is not likely to be delayed. In most cases of meningococcal meningitis (as well as other bacterial meningitis), there is a non-specific illness one to three days before signs of meningitis appear. Mortality of patients presenting with meningococcal meningitis is low (1%–5%), considerably lower than that of patients with invasive meningococcal disease without meningitis (up to 40%).
- **Blanching macular or maculopapular rash**

  It is not commonly known that the early meningococcal rash may be diffuse macular or maculopapular (or rarely urticarial), a rash which blanches with pressure. It mimics a non-specific viral rash and may completely disappear or dramatically evolve into the typical petechiae.
- **Severe pain in extremities, neck, back or elsewhere.**

  Severe muscle pain, even in the absence of overt fever, may be an

early symptom of meningococcal, staphylococcal or streptococcal bacteraemia. Muscle pain is more prominent in patients with meningococcal meningitis than in those with other forms of bacterial meningitis. It occurs in both adults and children.

We pay a great deal of attention to any febrile patient with severe pain at any site.

- **Vomiting, especially in association with headache or abdominal pain.**

  Vomiting is not a common symptom in previously healthy individuals. If it occurs without diarrhoea, it should not simply be dismissed as gastrointestinal infection, as it is a common symptom of central nervous system infection and occult sepsis.

- The message is to look at the whole pattern of the illness rather than exclusively at the presence or absence of any particular symptom or sign. It is not a matter of deciding whether the headache or shoulder pain is significant, or whether meningitis has been excluded by a normal cerebrospinal fluid examination, but why this patient is seeking help at this time.

Specific Issues

1. Optimal Practice

Adam FABRE has presented to hospital with the following symptoms described in the medical record:

- Headache
- Coryza
- Vomiting
- Neck pain
- Severe muscle pain (described as 10 out of 10 on a pain scale)
- Fever and chills

On examination by medical and nursing staff he was found to have the following signs:

- Looks 'unwell'
- Temperature 38.5^{oC}
- Tachycardia 120
- Photophobia
- Difficulty and pain when flexing neck (chin to chest)
- Widespread non-petechial rash

The constellation of symptoms and signs and the description of the patient as "looking unwell" suggests that the patient has a significant illness. The diagnosis of an 'URTI' fails to recognise the significance of many of these signs and symptoms.

Some of these symptoms might be considered to be 'non-specific' (ie not diagnostic of a particular illness), but when taken as a whole and in particular if one considers their reported severity, it is evident that this is not a minor illness.

Other illnesses that might present non-specifically with these symptoms include some forms of pneumonia, 'true' influenza, other more exotic viral infections (for example Dengue fever from north Queensland) and other causes of septicaemia. These have not been considered nor has a history of travel etc been elucidated.

An URTI would commonly be considered to be a very common, relatively minor illness with symptoms of nasal blockage and discharge, minor sore throat, cough and possibly other constitutional symptoms such as mild fever and a feeling of mild unwellness.

Severe headache, vomiting, high fever, neck pain, severe generalised muscle pain and a generalised rash would not commonly be considered to be features of a simple, common and benign 'URTI' that might be expected to respond to simple symptomatic treatment with fluids and paracetamol.

There are no guidelines or protocols for the assessment of patients presenting with what are common symptoms.

Reasonable practice in a patient presenting with these signs and symptoms would be.

- Triage to a more urgent triage category than category 5. This has made no difference in this instance as the patient was attended within a short period of time.
- Timely medical assessment with a view to formulating a differential diagnosis and management plan as well as providing effective analgesia. A directed history, particulalry of travel, animal exposure etc would also be usual.
- Medical examination of an unwell patient with these symptoms and signs would be directed at looking fo a cause of the fever and symptoms. Specifically this would include looking for evidence of infection in the upper and lower respiratory tracts, abdomen, genitourinary system and central nervous system.
- It may be that at the first examination there is no clear diagnosis based upon physical signs. In this case the issue becomes not "does the patient have meningitis?", but one of looking at the whole pattern of the illness rather than exclusively for the presence or absence of any particular symptom or sign and "is this patient unwell and could they develop a serious illness?"
- Initiation of appropriate investigations to look for a cause for this febrile illness. Investigations could include:
    - full blood examination,
    - blood cultures,
    - inflammatory markers such as CRP (C-reactive protein)
    - chest x-ray

  - urine analysis
  - other more specific tests as directed by history and other results

- If the history and clinical examination findings suggested a specific diagnosis one would likely perform additional investigations to confirm or refute this diagnosis.
- In the event that meningitis was suspected as the result of the clinical examination further investigation in the form of a lumbar puncture would be undertaken (unless there was a specific contraindication to doing this). Following this appropriate antibiotic therapy would be commenced. If the patient was very unwell with clear cut meningitis or if there was going to be a significant delay in confirming the diagnosis with a lumbar puncture then antibiotic therapy would be commenced as soon as possible.
- In this case an initial medical assessment has come up with a working diagnosis and management plan.
- This may have been reasonable in the first instance, but it is notable in this case that the diagnosis has not been reviewed or revised at the completion of the initial therapy despite the patient not apparently improving (and possibly worsening) with simple treatment that would be expected to provide symptomatic relief to a patient suffering from a minor illness. The patient's rapid pulse rate (tachycardia) has not resolved with the intravenous therapy intended to treat the dehydration that was thought to be causing the tachycardia.
- At this point it would be reasonable practice for the patient to be reassessed and the working diagnosis and management reviewed. This has not happened and further treatment has been effectively more of the same.
- It would also be reasonable practice to expect that a junior or inexperienced doctor discusses the case with a more experienced clinician at this point. Ideally a 'fresh set of eyes' should see the patient. It is often very difficult to get a clear clinical impression from a verbal report from a junior doctor, particulalry in the absence of pathology tests that might assist with decision making in these circumstances.

There has been no clinical 'hands on' review by a more experienced doctor, although there has been a verbal consultation between the junior doctor and the doctor supervising the ED that night. The cubicle nurse comments in her statement that she raised concerns with the senior doctor.

It is not known what clinical information or impression was conveyed by Dr DIAZ to Dr Le BROCQUE or what questions Dr Le BROCQUE asked Dr DIAZ, but it would be reasonable practice for a senior doctor to personally review a patient who looked and felt unwell with fever, headache, neck pain, vomiting, severe muscle pain and who had failed to improve with simple treatment. If nursing staff had expressed concerns about both the patient's condition and the experience of the doctor caring for them, then a personal review of the patient would be even more prudent.

It is also notable that the assessments of the Triage nurse and the 'cubicle' nurse differ in the presence and or severity of some symptoms and signs. This might be accounted for if there was a significant time between triage and assessment in the cubicle, but this has not been the case

2. Other Assessments

The patient may have benefited from the following:

a. Examination looking for the clinical sign of nuchal rigidity (neck stiffness).

In the statements by Dr DIAZ and Triage Nurse Matthew SWALES, both comment upon neck examination and the pain experienced in flexing the neck forward:

- **Dr Else PIN PIN DIAZ**

  Point 11

  "...On this occasion I conducted an neck examination. Adam could rotate his head and look from left to right without pain. He did however complain of pain on touching his chin to his chest"

- **Matthew SWALES – Triage Nurse**

  Point 5

  " Adam reported to me that he was not experiencing pain on rotation of the head, but that he was having pain on extreme of extension of his chin to his chest. He said that his muscles felt sore"

Signs of Meningitis or Meningeal Irritation (Meningism)

When examining a patient with suspected meningitis or meningeal irritation there are a number of physical signs that would be looked for.

Most commonly one would examine the patient for neck stiffness or nuchal rigidity.

In meningeal irritation when the neck is bent forward (or flexed forward) there is reflex (involuntary) spasm of the neck muscles, resisting the flexion. This gives the feeling of rigidity in the neck to the examining clinician. This occurs whether the patient is conscious or unconscious.

Neck stiffness in this context is a physical sign found on examination, rather than something complained of (ie a 'symptom') by the patient. Patients with minor viral illnesses often complain of feeling stiff, including in the neck, but when examined have no evidence of meningism.

However in the presence of meningeal irritation patients may avoid flexing or bending their neck because of the discomfort or pain it causes.

In this case the patient has been described by 2 clinicians as experiencing the symptom of pain when flexing his neck to touch his chin to his chest.

No clinician has documented that they have examined this patient for the physical sign of neck stiffness or nuchal rigidity.

I am not aware of the rotation of the head from left to right being a valid sign or symptom of meningeal irritation.

One would also look for Kernig's sign. The hip and knee are bent to 90 degrees and the knee extended (straightened) by the examining clinician. This sign is positive (for evidence of meningeal irritation) if there is reflex hamstring resistance to this. Dr DIAZ has perfromed this test according to her notes.

b. Timely Blood Test Results

Blood tests were taken at 0510 on 14 July, but not sent to a pathology laboratory until approximately 0831, by which time the situation was about to become critical and the opportunity lost.

Test results had not been returned by this time, but the Full Blood Count demonstrated a raised White Cell Count and the CRP was grossly elevated at 239.

CRP (c-reactive protein) is a protein found in the blood that is referred to an an 'inflammatory marker'. It is not specific for any particular ilnness, rather it is a marker of infection and or inflammation of any cause. Normal levels are <10, and so a level of 239 is extremely high, suggesting some significant inflammatory process. One would not see a level this high in a patient with a viral URTI.

It is possible that if this test had been performed in a more timely fashion the extremely abnormal result may have alerted medical staff to the possibility or probability of a serious illness.

Whilst it appears that there was no pathology laboratory services on site at Casey hospital at this time, it is assumed that there was still a system of transporting specimens to an off-site laboratory afterhours, but that this was not utilised.

A venous blood gas performed in the ED at 0507 hours demonstrated that the patients electrolytes, renal function, glucose and lactate levels were normal. This does not exlude the presence of serious infection, but indicates that the patient was not significantly metabolically deranged or shocked at the time. The equipment used to perform this test would not be able to perform a blood count or a CRP.

In common practice it would be usual not to wait for blood test results to be returned if it was thought that the patient probably had meningitis on clinical grounds at the time of first assessment, and treatment with antibiotics would be commenced.

c. Clinical assessment from a more experienced doctor

The patient has not been reviewed or sighted by a more experienced clinician. It is possible that earlier clinical review by an astute clinician may have considered alternative diagnoses.

d. Lumbar puncture

A lumbar puncture is not a 'routine' or screening test as it is not without risk, is not always easy to perform and requires a degree of skill and experience. In this setting would be done to confirm or exclude a diagnosis of central nervous system infection in cases of suspected meningitis.

Unfortunately the possibility of meningitis has to be considered in order to find it necessary to perfrom the test, and this did not occur in this case.

If it is clear that the patient has menigitis on clinical grounds, a lumbar puncture is often not performed in the interest of expediting treatment.

3. Standard of Care.

Care has been provided in a conventional and timely fashion, however it has underestimated the severity of the patients illness based upon assessment by a junior doctor.

A management plan has been recorded and implemented. This consisted of analgesia, intravenous fluids and anti-emetic medication. This has been implemented and completed.

By 0630 the patient has received 2 litres of intravenous normal saline, and 2 separate doses of analgesic medication. Further analgesia is given shortly after this. Despite these therapies the patients symptoms of pain and signs of fever and tachycardia have not changed. The persistence of the fever has prompted Dr DIAZ to seek advice from a senior colleague. It is not clear if the whole clinical picture and failure to respond to treatment has been conveyed to Dr Le BROCQUE, but the only additional treatment commened was to administer another dose of analgeisa/anti-inflammatory/anti-pyretic medication.

A working diagnosis has been made and tested with a simple treatment plan. From the clinical notes it is apparent that the treatment has failed to produce any improvement in the patients condition.

The nurse caring for the patient (Vianney ALLISON) comments in her statement that she was concerned that "we were not on top of that pain". This seems to be the only comment from any staff that, despite treatment, the patients condition was not improved.

Beyond this there has been no patient review or reassessment, no revision of the diagnosis or consideration that this might be something

other than a minor illness. It is concerning that the patients relatives comment that Dr DIAZ was planning to send the patient home.

A conventional standard of care would expect that once treatment had failed to produce the expected improvement, that the patient would be reassessed and alternative diagnoses considered and other investigations expedited.

It would also be convention for a junior or inexperienced doctor, in a setting such as this, to discuss the case with a more experienced clinician at this point.

In this case the patient has been discussed with a more senior clinician, but this has not resulted in a 'hands on' review by the senior doctor, nor has it resulted in any reconsideration of the diagnosis and management plan.

4. Antibiotics

Antibiotic and antiviral therapy has been commenced after the patient has become unconscious with fitting and brain swelling. At this time it is probably too late for antibiotics to alter the outcome.

If antibiotics had been commenced earlier it is possible that the outcome of this illness may have been different. It is not possible to say exactly as to what extent as this would depend upon the time of administration and the repsonse of the infection to the drugs.

It is possible that the earlier administration of antibiotics may have prevented the progress of the infection and resulted in complete recovery or attenuated the infection so that the patient survived with or without complications or disability.

However meningococcal meningitis may be rapidly progressive or fulminant and it is possible the administration of antibiotics within a short time of arrival at the hospital may have made no substantial difference to the outcome.

Antibiotics do not halt the meningeal inflammatory process immediately. Some studies show even a transitory worsening of the inflammation after antibiotic administration, possibly due to the enhancement of endotoxin release.

5. Fluid administration.

This warrants particular comment. 2000 mls of saline were administered over 75 mins. This is a relatively rapid rate of infusion in a patient who is not shocked and thought to be suffering from a viral URTI. Although clearly not intentional, it is possible that this rapid rate of saline administration has contributed to the deterioration of the patient by accelerating the development of cerebral oedema.

When saline is administered intravenously, a significant percentage of the volume ends up in the tissues, as opposed to remaining in the intravascular space and expanding the volume of the circulation.

In a condition where there is pre-exisiting inflammation (as in the brain and meninges covering the brain), the blood vessels in these tissues may be 'leaky' and fluid leaks more easily out of them into the tissues, causing oedema, in this case cerebral oedema.

6. Significance of Rash

In witness statements by Matthew SWALES (point 3) and Dr Else PIN PIN DIAZ (point 13), both witnesses comment upon the rash that is described as "non-petechial" and "blanching" and not suggestive of meningitis.

- **Matthew SWALES – Triage Nurse**

  Point 3

  "It was my assessment that Adam presented with a non-petechial rash, rather than a rash which gave rise to the prospect of a diagnosis of meningitis"

- **Dr Else PIN PIN DIAZ**

  Point 13

  " Adam did have a rash on the anterior side of his legs. It appeared to be a blanching rash. Consequently I did not consider it to be suggestive of meningitis"

In their statements both Matthew SWALES and Dr DIAZ consider that the presence of a blanching or non-petchial rash was not suggestive of meningitis.

Unfortunately this demonstrates an ignorance of the pathophysiology and significance of a rash in meningococcal disease.

It also demonstrates an ignorance or lack of consideration of other forms of bacterial meningitis or encephalitis that do not present with a characterisitic rash and an ignorance of other symptoms of a potentially severe illness.

A petechial, hemorrhagic or non-blanching rash is characteristic of infections with meningococcus, specifically meningococcal septicaemia. If meningococci are not present in the blood then the characteristic rash does not develop, even though there may be meningococcal infection around the brain. Septicaemia may occur without meningitis and meningitis may occur without septicaemia. They may occur together.

Meningococcal meningitis without septicaemia is not clinically distinguishable from other forms of bacterial meningitis. In this community and in the young adult age group the major bacterial pathogens are Neiserria meningitidis (meningococcus) and Streptococcus pneumoniae (pneumococcus).

In the case of Adam FABRE no bacteria have been grown in blood cultures and meningococcal DNA has not been detected in the blood. This indicates that meningococcal septicaemia was not present.

Meningococcal DNA was detected in the CSF (cerebrospinal fluid) indicating the presence of meningococci that were not able to be grown in culture due to antibiotic therapy.

This demonstrates that meningitis existed in the absence of septicaemia and that reliance by staff on the abscence of a non-blanching rash to suggest the diagnosis of meningitis has delayed or missed the diagnosis.

7. Suggestions re improvements

In the case of the specific Emergency Department concerned the following might useful

- education of both medical and nursing staff on the modes of presentation of patients with potentially serious infective illness
- clearly defined reporting responsibilities for junior or inexperienced staff to senior medical staff
- staffing levels or staffing of sufficient experience that enable supervising senior medical staff to have a more supervisory and consulting role
- clearly defined responsibilities for supervising or senior medical staff, particularly in regard to review of patients at nursing staff request.
- establishment of system to enable concerned medical or nursing staff to circumvent the supervising doctor if required to gain other senior medical input.
- establishment of on-site pathology services to enable timely return of blood test and other pathology results.

From the public health and education perspective the following might improve the pick up and early diagnosis of meningitis and serious bacterial illness in general:

- Education regarding signs and symptoms serious bacterial disease of any cause (as in Yung and McDonald's article) might be useful to help staff identify patients at risk.
- Education regarding significance of rash in meningococcal disease and the possibility of rashes of different appearance
- Education regarding other causes of meningitis or meningo-encephalitis
- Education of the medical community regarding the presenting symptoms and signs of serious bacterial infection.

8. Other

The major issue central to this case is not that meningococcal menigitis was 'missed', but rather that a patient with signs and symptoms of serious illness has been dismissed as an 'URTI'

The message is to look at the whole pattern of the illness rather than exclusively at the presence or absence of any particular symptom or sign. It is not a matter of deciding whether the headache or shoulder pain is significant, or whether meningitis has been excluded by a normal cerebrospinal fluid examination, but why this patient is seeking help at this time.

Summary

1. Adam FABRE presented to hospital with symptoms and signs of fever, headache, severe muscle pain, vomiting, neck pain, tachycardia and a blanching rash.
2. Both the triage nurse and treating doctor were reassured about the presence of a blanching rash as not being suggestive of meningitis. This demonstates ignorance of other causes of meningo-encephalitis, the pathology of meningococcal infection and that fact that many patients with meningococcal infection do not have a rash at presentation.
3. He was diagnosed by a junior doctor as suffering from a viral URTI, despite many of the patients symptoms not being consistent with a viral URTI.
4. There has been no documentation of an examination of the patient for the clinical sign of neck stiffness or 'nuchal rigidity', even though there is documentation of the patient experiencing pain when flexing the neck forward. Correct interpretation of this symptom and the correct examination of the patient for nuchal rigidity may have raised the level of suspicion of meningitis.
5. A nurse considered that he had photophobia, neck stiffness, 10/10 whole body pain. This does not seem to have raised concern amongst medical staff.
6. He was treated with intravenous fluids and analgesia. This did not improve the patients observations or symptoms.
7. Despite the failure of treatment, the treatment plan and diagnosis was not revised, nor did the patient receive an assessment from a senior doctor.
8. Blood was taken in the early hours of the morning, but was not sent to pathology for several hours. Some of these results were grossly abnormal and may have given rise to a much greater level of suspicion of a serious cause for the patients illness.
9. The patient has been given 2000 mls of fluid intravenously over a relatively short period of time (75 mins). This may have contributed to the development of cerebral oedema.

10. It is possible that the earlier administration of appropriate antibiotics may have resulted in an improved outcome, but it is also possible that this may not have affected the outcome in the case of fulminant and rapidly progressing disease.

11. It is possible that there is a commonly held misconception that the rash is associated with meningitis as opposed to septicaemia and the absence of a rash excludes the diagnosis of meningococcal infection, either septicaemia or meningitis. Specifically it ignores other causes of meningo-encephalitis that do not present with a characterisitc rash – eg viral, pneumococcal.

12. Some of this may be due to the publicity around meningococcal disease and the emphasis placed upon the rash, so that both clinical staff and the public may become fixated upon a rash, rather than the constellation of other symptoms and signs that may indicate a serious infective illness of any cause.

13. Education of the medical community regarding the presenting symptoms and signs of serious bacterial (or other) infection (as in Yung and McDonald's article) might be useful to help staff identify patients at risk.

I trust that this has been of assistance in this matter.

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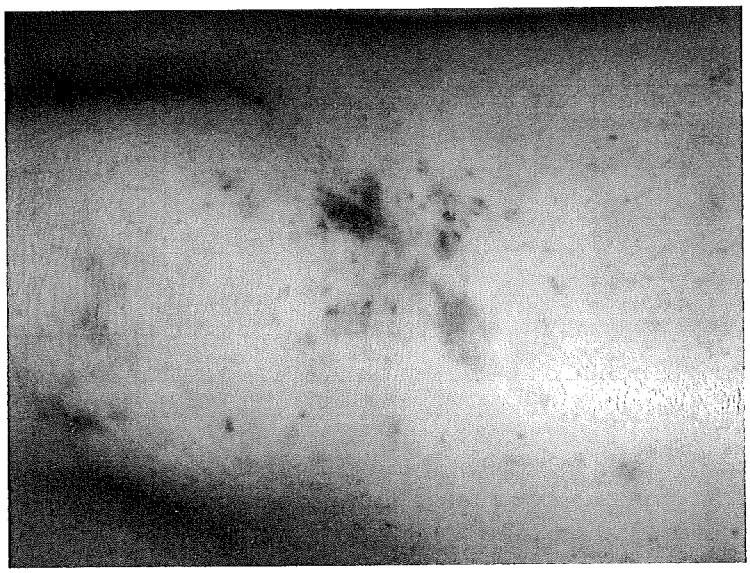
Glossary

a) *Petechial* rash refers to the appearance of a rash as small hemorrhages or bruises within the skin. Because petechiae are small hemorrhages in th skin, they do not disappear when pressure is applied to the skin. A blanching rash implies that there is localised redness the disappears (blanches or turns white) when pressure is appiled. The redness would retun when pressure is released. A petechial rash is characteristically associated with meningococcal septicemia (the presence of meningococcus in the blood).



b) *Kernigs sign* is tested for by flexing the hip to 90 degress and then straightening the knee. If meningeal irritation is present attempting to straighten the knee results in reflex contraction of and resistance in the hamstring muscles and pain in the lower back. If positive Kernig's sign is taken as a sign of meningeal iritration due to either meningitis or blood.

c) *Macular* refers to a small flat 'spot' of different colour on the skin.

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Early clinical clues to meningococcaemia

Allen P Yung and Malcolm I McDonald

ABSTRACT

- Meningococcal septicaemia has high mortality, especially when the diagnosis is delayed or missed.
- Early recognition is not always straightforward, as classic clinical features may be absent or overlooked at initial presentation.
- Septicaemia without focal infection accounts for 15%–20% of cases of meningococcal disease and is the most worrisome manifestation in terms of diagnosis and outcome; in contrast, meningococcal meningitis is usually straightforward to diagnose, with a relatively good prognosis.
- Useful early clinical clues to meningococcaemia include:
  - a haemorrhagic (petechial or purpuric) rash;
  - blanching macular or maculopapular rash that appears in first 24 hours of illness;
  - true rigors;
  - severe pain in extremities, neck or back; vomiting, especially in association with headache or abdominal pain; rapid evolution of the illness;
  - concern of parents, relatives or friends;
  - patient age (highest incidence at age 3–12 months, followed by 1–4 and then 15–19 years); and
  - contact with a patient with meningococcal disease.
- In addition to specific clues, clinicians should look at the whole pattern of the illness.
- Timely clinical review is essential if there is doubt about the diagnosis.
- In any acutely febrile patient, it is prudent to ask "Why is this patient seeking help now?", then "Could this patient have meningococcaemia?".

MJA 2003; 178: 134–137

DEATHS FROM MENINGOCOCCAEMIA in previously healthy children and young adults continue to occur in Australia. Newspapers and radio talkback shows regularly feature disturbing stories, and general practitioners and emergency departments are often blamed for delay in diagnosis and appropriate treatment. Is it true that we are poor in our recognition of meningococcal disease? If so, what can be done about it?

Guidelines on early management of meningococcal disease

Several recent Australian publications have highlighted this issue. An editorial by Hall in the Journal in June last year reminded us that "deaths from meningococcal septicaemia may be prevented by early antibiotic treatment".[1] All general practitioners are advised to be ready to administer benzylpenicillin immediately to a patient with an acute systemic febrile illness and either a petechial or purpuric rash. Likewise, the Department of Health Services, Victoria, has issued *Advice on meningococcal disease for medical practitioners* (modified January 2002), which stresses that "prompt diagnosis of meningococcal septicaemia and meningitis and preadmission treatment of presumptive cases can be life saving".[2] Common signs and symptoms of meningococcal disease are shown in Box 1.

In September 2001, the Communicable Diseases Network Australia published *Guidelines for the early clinical and public health management of meningococcal disease*.[3] They also urged immediate administration of benzylpenicillin in suspected cases of meningococcal septicaemia, and emphasised the importance of haemorrhagic rash as the most characteristic clinical feature. The authors pointed out that "less commonly, the rash has a maculopapular appearance, the discrete pink macules or papules blanching under pressure".

All three documents advocate early antibiotic treatment for suspected meningococcal septicaemia and stress the importance of a haemorrhagic rash. Unfortunately, the two government documents may not be commonly accessed by general practitioners. Even more importantly, only 40% of patients with invasive meningococcal disease present with a haemorrhagic rash; this usually does not appear until six to 12 hours after the first symptoms. About half the patients who die of meningococcal disease do so within 24 hours of the first symptoms.[4] Thus, to significantly reduce the risk of death, we need to suspect the presence of meningococcaemia in the first 12 hours of the illness. Can we do so, even in the absence of a haemorrhagic rash?

We would like to share our approach to early diagnosis of meningococcal disease, based on several decades' experience of teaching medical students and junior doctors about infectious diseases. We acknowledge that our advice is largely descriptive and is based on clinical observation and case reports and series.

Syndromes of meningococcal disease

First, we need to understand the varied nature of meningococcal disease. The rate of illness progression varies widely between individuals; it may be fulminant and cause death

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1: Common signs and symptoms of meningococcal disease*

In children and adults:

- Fever, pallor, rigors, sweats
- Headache, neck stiffness, photophobia, backache, cranial nerve palsy
- Vomiting or nausea, and sometimes diarrhoea
- Lethargy, drowsiness, irritability, confusion, agitation, seizures or altered conscious state
- Moaning, unintelligible speech
- Painful or swollen joints, myalgia, difficulty in walking
- While the absence of a rash does not exclude meningococcal disease, any haemorrhagic rash should be particularly noted.

In infants and young children the following may also occur:

- Irritability; dislike of being handled; unwillingness to interact or make eye contact
- Loss of interest in the surroundings
- Tiredness, floppiness, drowsiness, altered mental state
- Twitching or convulsions
- Grunting or moaning
- Turning from light
- Pallor despite a high temperature.

Note in particular:

- Rapid deterioration in clinical condition
- Repeat presentation to surgery or hospital
- Normally calm friends and relatives whose worry seems more extreme than the symptoms appear to justify.

*Modified from *Advice on meningococcal disease for medical practitioners* (Department of Human Services, Victoria, 2002 — modified January 2002).[2]

within 12 hours, or it may assume a chronic form that goes on for weeks. The determining factor is the clinical syndrome[5] (Box 2). The time elapsed between first symptoms and admission also correlates with the clinical syndrome.[4]

Meningitis is the most common clinical syndrome (80%–85% of cases). The diagnosis is relatively straightforward when the patient presents with the typical clinical picture (fever, headache, vomiting and change in conscious state), and treatment is not likely to be delayed. In most cases of meningococcal meningitis (as well as other bacterial meningitis), there is a non-specific illness one to three days before signs of meningitis appear. Rarely, the clinical picture is dominated by coma, which is both sudden and deep; this syndrome is sometimes referred to as fulminant meningococcal encephalitis.[6] Mortality of patients presenting with meningococcal meningitis is low (1%–5%), considerably lower than that of patients with invasive meningococcal disease without meningitis (up to 40%). Furthermore, prognosis of patients with bacterial meningitis (from all causes) was no worse in those whose disease was not recognised for as long as two to four days before admission than in those admitted when first seen.[7]

A few patients have less common syndromes, such as conjunctivitis, pneumonia, septic arthritis and pericarditis. Localising symptoms and signs ensure that diagnosis and treatment are unlikely to be delayed.

Meningococcaemia is another matter: between 15% and 20% of patients present with septicaemia unaccompanied by meningitis or other focal features. The illness in patients with pure septicaemia is generally more severe, progresses more rapidly, and has a high case fatality rate. Unfortunately, there is often confusion in the minds of the public, the media and even health professionals between meningitis and meningococcaemia; this may occasionally be a factor in delayed diagnosis.[8]

In most patients, the beginning of meningococcal septicaemia is marked by acute onset of fever, chills, and generalised muscle aches or pains in the back or thighs. There may be a transient clinical improvement after four to six hours; this is often the stage when patients are sent home from emergency departments. Six to 12 hours after onset, a rash typically appears, which may initially resemble a viral rash. The characteristic haemorrhagic rash appears soon after. How many patients suffer from meningococcal septicaemia without meningitis or a rash is unknown, as the diagnosis may well be missed in these cases. Similarly, every patient with fulminant meningococcaemia who lives long enough develops a haemorrhagic rash.

It is in this group with septicaemia but no obvious features of meningitis that the illness progresses most rapidly, has the most non-specific clinical features, and the highest fatality rate. Can we suspect meningococcaemia early? Several clues may help. Most of these also apply to sepsis caused by other bacterial pathogens, but meningococcal sepsis is the most fulminant.

Clinical clues to early diagnosis of meningococcaemia

Haemorrhagic rash: In Australia, the acutely ill patient with fever and haemorrhagic rash (petechial or purpuric) usually has bacteraemia, and the most common cause is meningococcaemia.[9,10] Typically, the rash begins within 24 hours of onset of illness — a useful clinical pointer. In the early stages, sparse petechiae can be easily missed unless specifically sought in body folds, groin and axillae, along flexor surfaces, on the ankles, or on the conjunctiva, sclera or oral mucosa. The rash evolves over time, and may become apparent on repeat examination.

2: Clinical syndromes associated with meningococcal disease*

- Meningococcal meningitis
- Meningococcal bacteraemia
- Meningococcaemia (purpura fulminans and the Waterhouse–Friderichsen syndrome)
- Respiratory tract infection
  - Pneumonia
  - Epiglottitis
  - Otitis media
- Focal infection
  - Conjunctivitis
  - Septic arthritis
  - Urethritis
  - Purulent pericarditis
- Chronic meningococcaemia

*Modified from Rosensteln et al.[5] Individual patients may have more than one syndrome.

3: Suggested strategies for family doctors and emergency departments

Treat immediately with benzylpenicillin or ceftriaxone and then admit to hospital all patients:

- With suspected meningococcal septicaemia or meningitis;
- With fever and a haemorrhagic rash; or
- With fever or pain at any site and a history of contact with meningococcal disease.

Refer and admit to hospital:

- Any patient, young or old, presenting with a true rigor;
- Any patient with fever and severe generalised muscle pain or bilateral anterior thigh pain; or
- A febrile patient seen for a second time within a 24–48-hour period.

Suspect meningococcaemia and observe closely:

- Any unwell patient with acute onset of fever and a non-specific "viral" rash appearing within the first 12 hours of illness; or
- A previously healthy child or young adult with an acute febrile illness whose family or friends are concerned about his or her condition.

Blanching macular or maculopapular rash: It is not commonly known that the early meningococcal rash may be diffuse macular or maculopapular (or rarely urticarial), a rash which blanches with pressure.[8] It mimics a non-specific viral rash and may completely disappear or dramatically evolve into the typical petechiae. Indeed, it may resemble the first day of a measles rash. However, measles rash is preceded by a prodrome of several days with prominent respiratory symptoms. Similarly the length of the prodrome and associated symptoms and signs would generally help identify other viral causes. Meningococcaemia should be considered if the rash is present in the first 24 hours of illness, and the patient appears unwell.

True rigors: A rigor is a shaking chill that cannot be stopped voluntarily. Onset is sudden, and duration may be 10–20 minutes. It should be distinguished from a sensation of chill or shivers that lasts only for seconds. Although rigors occur in some viral infections, they should generally be regarded as indicators of significant sepsis, in conditions such as bacteraemia, pneumonia, abscesses, endocarditis, cholangitis, and pyelonephritis.[11,12] We preach the "rigor rule" to our students: any patient, young or old, presenting with a rigor should be admitted to hospital for observation and investigation. This rule has not been popular with some colleagues in emergency departments.

Severe pain in extremities, neck, back or elsewhere: Severe muscle pain, even in the absence of overt fever, may be an early symptom of meningococcal, staphylococcal or streptococcal bacteraemia. It is also a feature of myositis and necrotising fasciitis. Anterior thigh pain and tenderness has been found to be a useful indicator of bacteraemia.[13] Children may refuse to walk because of pain in the extremities.[14] Muscle pain is more prominent in patients with meningococcal meningitis than in those with other forms of bacterial meningitis. It occurs in both adults and children.

Abdominal, chest and joint pains occasionally occur in patients with sepsis. When pain is prominent, it can dominate the patient's illness and become a false localising symptom. Although we do not know how common this is in meningococcal sepsis, we have anecdotal evidence of local pain leading clinicians and their patients to disaster. We pay a great deal of attention to any febrile patient with severe pain at any site.

Vomiting, especially in association with headache or abdominal pain: Vomiting is not a common symptom in previously healthy individuals. If it occurs without diarrhoea, it should not simply be dismissed as gastrointestinal infection, as it is a common symptom of central nervous system infection and occult sepsis. On the other hand, diarrhoea may also be a non-specific feature of bacteraemia.

Rapid evolution of illness: Rapid evolution of an illness is usually an indication of its severity. Previously healthy individuals tend not to seek medical attention unless something is seriously amiss. An abrupt change in health is a warning sign, and we take special notice of patients who present within 24 hours of onset or whose illness has progressed rapidly over 24–48 hours, especially from being up and about to being bedridden. Likewise, we view seriously anyone who presents to the doctor or emergency department more than once over a 24–48-hour period.

Concern of parents, relatives or friends: Parents are usually the best judges of the health of their children. We also take notice when relatives or friends are more worried than the patient's symptoms appear to warrant.[15]

Age: By itself, this is of little value in the diagnosis of meningococcal disease, which may occur at any age. The attack rate is highest among children aged three to 12 months (incidence in Victoria in 2001 was 32.5/100 000), followed by those aged one to four years (11.0/100 000).[16] The third-highest attack rate occurred in the age group 15–19 years (8.4/100 000). Fewer than 10% of cases of meningococcal disease occur in patients aged over 45 years.

In recent years, the incidence of group C meningococcal disease has increased in Australia. It is more severe than group B disease, and is most common in adolescents and young adults. This is the age group that goes to discos, smokes and lives communally and in military camps. Closeness, persistence of contact and cigarette smoking (active and passive) are known risk factors for infection and disease. As adolescents and young adults tend to shun doctors, it is wise to take seriously patients in this age group who present with a short history of fever and complaints of pains anywhere.

Contact with a patient with meningococcal disease: This history is seldom present when the infection occurs sporadically, as secondary cases constitute no more than 5% of the total. However, if a contact history is present, it must not be ignored.

Discussion

A major concern for doctors is missing the diagnosis of meningococcaemia when the patient does not appear very ill on initial presentation. Whether to give antibiotics is a perennial question. There are no reliable algorithms capable of directing management. The apparent improvement in some patients a few hours after the onset of illness makes the diagnosis even more difficult. This may be part of the disease process or the result of analgesic treatment or fluid replacement.

We have outlined nine clinical clues that are helpful in assessing febrile patients with no obvious focal signs. Of course, there are other relevant factors to consider, such as previous state of health, comorbid conditions, occupation, and travel history.[17]

- The first clue in our list gives rise to the golden rule: fever plus a petechial rash is meningococcaemia until proven otherwise, and immediate action is mandated.
- The related clue (a blanchable rash) is less categorical, but also urges us to consider meningococcaemia.[8]
- Individually, none of the other clues are indications for immediate antibiotic treatment and admission to hospital, and there are no data on their specificity or sensitivity. However, when one or more of these clues is present, meningococcaemia should be considered before the consultation is concluded. The message is to look at the whole pattern of the illness rather than exclusively at the presence or absence of any particular symptom or sign. It is not a matter of deciding whether the headache or shoulder pain is significant, or whether meningitis has been excluded by a normal cerebrospinal fluid examination, but why this patient is seeking help at this time.

Regrettably, there will always be a few patients whose meningococcaemia goes unrecognised, because they do not appear "toxic" on presentation, and their symptoms are non-specific. It is essential that all patients be advised to seek review if there is any clinical deterioration. As a final step, when assessing any febrile patient, it is prudent to ask ourselves these two questions: "Could this patient have meningococcaemia?" and "Why is he or she here now?".

Competing interests

None identified.

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(Received 24 Jul 2002, accepted 25 Sep 2002)