

Refining the public health response to primary meningococcal conjunctivitis

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Abstract

Primary meningococcal conjunctivitis (PMC) is accepted as an uncommon condition. This report describes two recent cases of PMC in newborn infants in a hospital nursery. In both cases the organisms identified were non-groupable strains of *N. meningitidis*, considered to be of low pathogenic potential. Both infants received systemic therapy and recovered without sequelae. The *Guidelines for the early clinical and public health management of meningococcal disease in Australia* recommend the notification of PMC to public health authorities and chemoprophylaxis of contacts. However, our 2 cases suggest that the guidelines should allow for an assessment of risk in determining the public health response. This assessment should include the severity of the conjunctivitis and the serogroup of the *N. meningitidis* isolate. *Commun Dis Intell* 2002;26:592–595.

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Introduction

Primary meningococcal conjunctivitis (PMC) is accepted as an uncommon condition, although the true incidence is unknown as most patients presenting with acute conjunctivitis receive antibiotic treatment empirically and recover without the collection of conjunctival exudate for culture. A review of 1,030 children with acute bacterial conjunctivitis, presenting to a hospital emergency department in Spain, found pure and abundant growths of *N. meningitidis* in conjunctival exudate in only 21 children (2% of cases).¹ A similar incidence of 2 per cent has been reported in a British paediatric accident

and emergency department.² Another series, however, suggests a lower figure, identifying *N. meningitidis* in only one case from 126 children presenting to an outpatient department, with acute conjunctivitis.³ Amongst 63 reported cases of PMC where serogrouping was performed (adult and paediatric cases) Barquet *et al*⁴ report that 34.9 per cent belonged to serogroup A, 44.4 per cent to serogroup B, 14.3 per cent to serogroup C, and 6.4 per cent were not groupable. In Australia however, serogroup A disease is rare, so the ability to generalise the results of this international review to our population must be questioned.

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Primary meningococcal conjunctivitis has been known to precede invasive disease. In a review of reported cases of PMC, Barquet *et al*¹ found that 17.8 per cent of cases developed systemic meningococcal disease. The risk of invasive disease in those treated initially with topical therapy alone was estimated to be 19 times greater than for those receiving systemic antibiotic treatment.¹ Consequently, systemic antibiotic therapy has been recommended for all patients with PMC.^{1,4,5}

In response to reports of PMC associated with invasive disease⁵ and a case report of invasive meningococcal disease in a contact of a child with PMC,⁴ Australian guidelines now require a public health response following the diagnosis of primary meningococcal conjunctivitis.⁶ Two recent cases of PMC in a hospital nursery where the identified organisms were non-groupable strains of *N. meningitidis*, are reported. These cases raise questions about the public health response, and highlight inconsistencies in the response to PMC compared to that required when meningococci are cultured from other non-sterile sites.

The Human Research and Ethics Committee of the South Eastern Sydney Area Health Service (Eastern Section) has approved publication of this paper.

Case 1

Case 1 was a male infant born by emergency caesarean section at 28 weeks gestation, and transferred to the Newborn Care Unit (NBCU) for management of respiratory distress. At 8 weeks of age, whilst still in the NBCU, he developed right eyelid swelling and erythema, with purulent discharge. An eye swab and blood for culture were collected, and the infant was commenced on intravenous cefotaxime (50 mg/kg q8h), flucloxacillin (50 mg/kg q12h), and topical chloramphenicol (q6h), whilst awaiting culture results. He was isolated and nursed in a single room. After 24 hours, his eye was much improved, with no discharge noted. Intravenous antibiotics were ceased after 48 hours, and oral cephalixin was commenced. Four days after the eye swab had been taken, the hospital laboratory advised that a light growth of a non-groupable *N. meningitidis* (sensitive to penicillin) had been isolated. Oral cephalixin was ceased, and intravenous cefotaxime was recommenced and administered for a total of 5 days. The public

health unit, assisted by the Newborn Care Unit's clinical staff and the hospital infection control staff, organised an information session for health care workers who had been involved in the care of the infant in the week prior to onset of his symptoms. The health care workers were treated as 'household-like' contacts, as the staff had been the main carers. The infant's parents and 8 staff members received rifampicin (600 mg twice daily for 2 days); one pregnant staff member received ceftriaxone (250 mg by intramuscular injection). The infant did not develop any sequelae or systemic meningococcal disease. He was discharged home on day 94 of life after resolution of his prematurity related problems.

Case 2

The second case was a full term male infant, born after normal vaginal delivery to a methadone dependent mother. The infant was admitted to the Newborn Care Unit for a brief period at birth for management of neonatal abstinence syndrome. He was transferred to the postnatal ward on day 2 of life. On day 4 he was noted to have a sticky left eye, which was managed with normal saline eye toilet. A swab from the left eye, taken on day 7 for Gram staining and culture, returned on day 10, a light growth of a non-groupable *N. meningitidis*, sensitive to penicillin. This isolate was phenotypically distinct from the isolate from Case 1. The conjunctivitis was considered mild, with redness of palpebral conjunctiva and a small amount of discharge. The infant was afebrile with no other constitutional symptoms or signs. He was commenced on intravenous cefotaxime (50 mg/kg q8h). The public health unit and the hospital infection control staff were notified, and the infant was nursed in isolation. Prophylaxis (rifampicin 600 mg twice daily for 2 days) was given to the infant's parents. As with Case 1, staff from the public health unit, assisted by clinicians and infection control staff, attended the unit to provide information to the health care workers who had been involved in the infant's care since his birth. Staff were advised that chemoprophylaxis was not warranted unless their contact with the infant had been close and prolonged. No nursing staff received prophylaxis. After 48 hours therapy the child's eyes were free of redness and discharge. Intravenous antibiotic therapy was ceased after 8 doses. There were no sequelae and the infant was discharged home on day 41 of life.

Discussion

Onset of PMC during the first week of life has been reported previously^{7,8,9} and it has been speculated that the source of the meningococcus in these cases has been the maternal genital tract.⁷ A recent case report of PMC adds weight to this theory, with strains identified from the conjunctival exudate of the newborn infant, the mother's endocervix and the mother's partner being of the same antigenic composition.¹⁰ Unfortunately, no swabs were able to be collected from the mother of the neonate (Case 2) to enable investigation of this possibility.

In slightly older infants, infection is likely to have been acquired after birth.⁷ Direct inoculation of *N. meningitidis* into the conjunctival sac from manual contact (e.g. rubbing the eyes) or through infectious airborne respiratory secretions have been suspected modes of infection.¹ Secondary seeding of the conjunctiva from the nasopharynx has been suggested by findings of identical strains (same serogroup, serotype and subtype) of meningococci isolated from the conjunctiva in PMC as from the nasopharynx.^{11 cited in 4} Indeed, from a microbiological perspective, the throat and conjunctiva of a newborn may be considered as essentially continuous mucosal surfaces, and the conjunctiva is often colonised by nasopharyngeal organisms when naso-lacrimal duct blockage occurs.

Systemic antibiotic therapy of PMC is recommended because topical therapy does not eliminate pharyngeal carriage or the risk of developing systemic meningococcal disease.¹ Out of 15 cases of invasive disease described by Barquet *et al*,¹ the serogroup of 13 strains was reported. Most were from serogroups A, B or C (A, n=3; B, n=6; C, n=2). Two cases were reported as non-groupable. However, one of the non-groupable cases was reported in 1936, raising the possibility of misidentification. The authors did not find any statistically significant difference between patients who developed systemic disease and those who did not develop systemic disease, in terms of serogroup or local (ocular) complications, but this may have been due to the small numbers in the study.

Strains of meningococci identified from deep isolates, such as blood or cerebrospinal fluid, are almost always encapsulated (with serogroups B and C accounting for most disease in Australia). In contrast, meningococci grown from the nasopharynx are more variably capsulated, and those with little or no capsular material most often represent colonisation or a

carrier state. Due to the lower pathogenicity of non-groupable meningococci and the absence of systemic signs or serious local disease (e.g. orbital cellulitis), Case 2 received a short course of parenteral treatment, with clinical resolution being reached by the conclusion of treatment.

The major rationale for chemoprophylaxis of contacts is that eradication of *N. meningitidis* from the nasopharynx of presumed carriers will prevent transmission to other (susceptible) people. Additionally, it may eliminate colonisation, and thus the risk of subsequent invasion in others also exposed to the carrier, or in those very close contacts exposed to the case after the onset of the illness, but prior to the commencement of antibiotic treatment.⁶ The virulence of the particular meningococci in cases of invasive meningococcal disease has been demonstrated through their ability to invade.

In the case of PMC, however, the Australian guidelines⁶ recommend that a public health response be mounted in the absence of invasive disease, and they do not allow for a differential response in the case of those meningococci whose virulence is well-recognised compared to the less pathogenic non-groupable organisms.

Unnecessary chemoprophylaxis of contacts increases the risk of bacterial resistance developing, and can eliminate non-virulent meningococci and other non-pathogenic bacteria (e.g. *Neisseria lactamica*) which have been shown in infants and young children to have an important role in induction of natural immunity to invasive meningococcal disease.¹² Further, as the public health response itself can create anxiety within the network of contacts and amongst those 'exposed' to the case, unnecessary alarm should be avoided wherever possible. A great deal of concern was evident amongst parents of infants placed in the same neonatal nursery as Case 1, and the event was reported in the media. Experience with Case 1 allowed a more rational approach from the hospital and public health unit when the second case was identified some weeks later. In this hospital nursery setting, the circle of close contacts was relatively small. However, situations exist where the routine public health response might include a greater number of contacts, such as the staff and children of a child-care facility, and the potential for inadvertent harm would be greater.⁶

The intent of the guidelines with respect to chemoprophylaxis following a case of PMC is to

prevent the rare, but nonetheless documented, secondary cases of invasive meningococcal disease.^{4,5} However, Australian guidelines for the management of meningococcal disease do not recommend a public health response when *N. meningitidis* is isolated coincidentally from other superficial sites (e.g. from oropharyngeal, genital or anal swabs).⁶ Further, in the absence of invasive disease, a public health response is not required when meningococci are isolated in the sputum (for example, in the case of pneumonia), despite the fact that transmission of *N. meningitidis* from cases of pneumonia has been reported.^{13,14} Clearly, there is a lack of consistency within the Australian guidelines on the public health response to the identification of *N. meningitidis*.

The existing guidelines recommending that PMC be notifiable to public health authorities should be supported, and the recommendation for chemoprophylaxis of contacts of cases of PMC should stand. However, the guidelines do not allow for an assessment of risk — the same response is required for cases of low pathogenic potential, as for cases of higher risk. We argue that rather than the routine implementation of chemoprophylaxis for contacts of all cases of PMC, the guidelines should allow for an assessment of risk. This assessment should include the severity of the conjunctivitis and the serogroup of the *N. meningitidis* isolate.

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