Group A Meningogoccal Outbreak in Peshawar

Shahid Jamil*, M.B.B.S.*
and
Sheikh Mohammad Suhail*
M.B.B.S., M.R.C.P., F.C.P.S.
Lady Reading Hospital,
Peshawar, Pakistan.

Summary
During a group A meningococcal outbreak in Peshawar, 454 hospitalised patients were studied from 26th December 1987 to 1st May 1988. The study was conducted at Lady Reading Hospital, Postgraduate Medical Institute, Peshawar. The incidence of the disease increased in the last week of December, during a dry and cold weather. It rained at intervals throughout the period of the epidemic, suggesting no influence of rains on the incidence of meningococcal disease. The disease typically started with fever, accompanied by chills, leading to a wrong diagnosis of malaria by the local unqualified "practitioners". More males were affected than females. Acute fulminant meningococcemia was much more common in the males. The overall mortality was 8.4%, with a higher mortality in males.

Introduction
In December, 1987, an unprecedentedly high incidence of meningitis was noticed. Four hundred and fifty-four patients of proven as well as suspected meningococcal disease admitted to the Medical units of Lady Reading Hospital, Peshawar from December, 1987 to May, 1988 were selected for the study. The study includes the epidemiological as well as clinical pattern of meningococcal disease in and around Peshawar.

Meningococcal disease occurs mostly in overcrowded populations and, if not treated promptly, carries a high mortality. The victims are usually healthy young adults. There is an alarming haemorrhagic skin rash in many cases. The mass media often label it as some mysterious virus disease. Such factors inevitably spread scare and alarm in the society.

* Trainee Registrars, Medical A Unit, Lady Reading Hospital, Peshawar.
In Sweden meningococcal disease is being recorded from 1875 onwards and in the United States since 1920, with epidemics occurring at an average of ten years interval. Endemic cases are mostly caused by group B, while group A is responsible for most of the epidemics. Sulphonamide resistant group C organisms have become the most common cause of the disease in the U.S.A. since 1965. By now sulphonamide resistance is shared by most of group A and B organisms. Major epidemics caused by group A meningococcus occurred during World War I and II in Detroit in 1928-29 and in the sub-Saharan Africa in the Fifties and early Sixties. Group A meningococcus was the cause of meningococcal disease in the U.S.A. during and immediately after World War II. Deaths from meningococcal infection amongst soldiers were more than from any other infectious disease. Major group A epidemics occurred in Brazil in 1974 and in Finland in 1978. There were minor outbreaks of group A meningococcal disease in Alaska in 1976.

Methods and Material

A study was conducted on 454 patients of meningococcal disease admitted to the Lady Reading Hospital, Peshawar during an outbreak of this disease. The age of the patients was from 6 months to 65 years. In 136 patients (30%) the diagnosis was confirmed by positive blood or cerebrospinal fluid (C.S.F.) cultures. Two hundred and seventeen patients (48%) had Gram-negative, intracellular diplococci on stained smears of the C.S.F. Eighty-two patients (18%) had a pyogenic C.S.F. and a purpuric rash suggestive of meningococcal disease. Only 19 patients (4%) had just a pyogenic C.S.F., and belonged to localities where the incidence of meningococcal disease was already very high. Other sporadic cases of pyogenic C.S.F. of unknown etiology were not included in the study. The patients were treated with combination of Benzyl Penicillin and Chloramphenicol. Cultures were grown on chocolate and blood agar. Though blood inoculation was done on special liquid media, positive growths after inoculation were transferred to chocolate agar. Blood and C.S.F. inoculation onto culture media was done at bedside. Agar plates were incubated at 37°C in a candle jar, with 5-10% CO₂. Positive cultures were confirmed by oxidase test. Slide agglutination tests of all the positive cultures yielded group A organisms.

Results

Thirty percent of the patients had positive cultures. Four patients
with meningococcemia had a positive blood and a negative C.S.F, culture. Most of the patients with negative cultures had already received some kind of antibiotic. Out of the culture positive specimens 130 were sero-grouped and all were found to be group A organisms, uniformly resistant to sulphonamides but sensitive to penicillin, chloromphenicol, minocin and rifampacin. Two hundred and forty patients (53%) were examined by quacks prior to hospitalization, Three hundred and eighty-two (84%) of these 454 patients were diagnosed to be suffering from malaria because of the associated chills. All were given antimalarials by the unqualified local ‘practitioners’ Almost all patients were of low socioeconomic status and lived in overcrowded localities.

Initially, cases admitted to this hospital belonged to Chamkani, a suburban village of Peshawar (Table-I). Then cases from another nearby village were noted and all these patients had either travelled to Chamkani within the preceding week or had met somebody from Chamkani village. Meanwhile cases started coming from Peshawar city and all could be linked epidemiologically to Chamkani or the other affected village Badaber. In the second month of the outbreak, more cases from the initially affected areas and many other areas of Peshawar city began to arrive. The cases occurred in clusters in different localities. In the third month, cases were also admitted from other suburbs of Peshawar. The initial increase in the incidence occurred after a prolonged draught of about 4 months. According to Table-II, it rained with regular intervals throughout the duration of the epidemic, suggesting that the rain had no effect on the epidemic. Only 22 cases (5%) were Afghan refugees. Two hundred and ninety three cases were male (65%), while one hundred and sixty one cases (35%) were females (Table-III). As shown in FIG-I, males were predominantly affected in almost all age groups except age group 0-5 years. Majority of the patients were below the age of 20 years (FIG-II). Acute fulminant meningococcemia was much more common in the male patients.

Serious complications included hemiplegia, monoplegia, paraplegia, cranial nerve palsy, seizures, dysarthria, emotional liability and arthritis. 10% of the patients had either of these complications alone or in combination (FIG-III).

Discussion

Recently an increase in the incidence of meningococcal disease, in different parts of U.K., has been reported. The organism was mostly
An outbreak of group A meningococcal disease in Haj pilgrims of Mecca and the occurrence of such a disease in the contacts of these pilgrims has been reported. The reported group A organisms were uniformly resistant to sulphonamides. Since the relatives of the initially admitted patients in our study had been Haj pilgrims and that the organism isolated was group A uniformly resistant to sulphonamides, the possibility of a link cannot be excluded.

Features that suggested the epidemic nature of the disease were that the cases were linked epidemiologically. The incidence of the disease in the previous years was less than 5% of the present incidence. The organisms were of group A, uniformly resistant to sulphonamides. Two or three cases occurred in single households, on nine occasions, within one month of the primary cases. People affected were mainly adults.

Since rains did not decrease the incidence of the disease in this study, it favours the hypothesis that epidemic of meningococcal disease is not linked to a particular climate but is probably linked to overcrowding of people who stay indoor in a weather unsuitable for them to stay outside. There is a high incidence of the disease in Egypt in winter, which is cold and damp, but a high incidence of the disease occurs in hot and dry season in Northern Nigeria. Our finding confirmed to the general increase in the incidence of meningococcal disease in late winter and spring. It is interesting to note that some workers have not recorded any seasonal variation in the incidence of this disease in non-epidemic conditions. Our pattern was similar to that of sub-Saharan Africa where group A epidemics typically start in January, peak occurs in March and April and then decrease. The decrease in the incidence in our case may have been, partly, because of the extensive vaccination carried out by private agencies in and around Peshawar.

The high incidence of the disease in the males may be because males meet many people as compared to females. The high incidence of acute fulminating meningococcemia in males may be because of some X-linked immunological defect. Mortality was higher amongst males (Table IV). Overall mortality was 8%. Fig. III shows the proportion of major complications affecting the patients studied. The age distribution given in Fig. II does not depict the age distribution of the disease in the population but just shows the age of different patients included in the study, by virtue of their
getting admitted to the hospital where the study was conducted. Neonates were not included in the study but it has already been established that the incidence of the disease in this age group is rare. Though, recently rifampacin resistant meningococci have been reported, but our isolates were uniformly sensitive to the drug. There were no reports of the disease in the relatives of the patients who were given chemoprophylaxis with rifampacin.

Acknowledgement

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We are also grateful to Professor Alaf Khan, Prof. Bakht Biland and Prof. Imran, Postgraduate Medical Institute, Peshawar to allow us to include their patients in the study.
<table>
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<th>Badaber</th>
<th>Charsadda</th>
<th>Charkhani</th>
<th>Nowshera</th>
<th>Other</th>
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<td>11</td>
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<td>11</td>
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<td>6</td>
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<td>4</td>
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<td>54</td>
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<td></td>
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**TABLE 1**

Geographic and Month-Wise Distribution of 454 Cases
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<thead>
<tr>
<th>Month</th>
<th>Date When Rain Started</th>
<th>Duration of Rain</th>
<th>Rainy Days Per Month</th>
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<td>February</td>
<td>20.2.1988</td>
<td>3 Days</td>
<td>3 Days</td>
</tr>
<tr>
<td></td>
<td>06.3.1988</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>09.3.1988</td>
<td>3 Days</td>
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<td>March</td>
<td>15.3.1988</td>
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<td>6 Days</td>
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<td></td>
<td>25.3.1988</td>
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</tr>
<tr>
<td>April</td>
<td>16.4.1988</td>
<td>1 Day</td>
<td>1 Day</td>
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### TABLE-III
#### SEX DISTRIBUTION

<table>
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<th>Sex</th>
<th>No.</th>
<th>%</th>
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<td>293</td>
<td>65</td>
</tr>
<tr>
<td>Female</td>
<td>161</td>
<td>35</td>
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<td>Total</td>
<td>454</td>
<td>100</td>
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### TABLE-IV
#### MORTALITY

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<td>293</td>
<td>27</td>
<td>9.2</td>
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<tr>
<td>Female</td>
<td>161</td>
<td>11</td>
<td>6.8</td>
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<tr>
<td>Total</td>
<td>454</td>
<td>38</td>
<td>8.4</td>
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</table>
Fig-I.

AGE AND SEX DISTRIBUTION

Number

[Bar chart showing age and sex distribution with bars for male and female.]

AGE (YEARS)

MALE

FEMALE
Age distribution of 454 cases

Number of cases

32 67 96 130 55 46 32

Age in years

0 5 10 15 20 30 40 45 50 55 60 65 70 75 80 85 90 95 100 105 110 115 120 125 130
OUTCOME OF 454 CASES OF MENINGOCOCCAL DISEASE

- FULL RECOVERY: 70.7%
- DEATHS: 8.5%
- SERIOUS COMPLICATION: 9.9%
- MINOR COMPLICATION: 11.1%

- HEMI, MENO AND PARAPLEGIAS: 11.7%
- CRANIAL NERVE PALSY: 17.7%
- PERMANENT SEQUELAE: 28.5%
- NERVE DEAFNESS: 2.1%
- RECURRENT HEADACHES: 22.2%
- RECURRENT SEIZURES: 2.2%
- ARTHRITIS: 15.5%
- DYSARTHRIA: 2.2%
- EMOTIONAL LABILITY: 8.8%
References


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15. Munford, R.S; Augusto De, E; T; Souzarde Morais, J; Fraser, D.W. Feldman, R.A; Spread of meningococcal infection within a household. Lancet, 1982; 1: 980-83.


