EPIDEMIC OF CEREBROSPINAL FEVER IN A CLOSED COMMUNITY

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During the latter half of the winter and the spring of 1953, an epidemic of cerebrospinal fever occurred amongst the African employees of the St. Helena Gold Mine. A total of 22 cases were diagnosed and treated at the mine hospital. Two deaths occurred.

No cases of meningococcal meningitis had previously occurred at St. Helena since June 1952, about one year previously. Since the inception of the mine in 1946 only about 2 sporadic cases a year were observed.

FACTORS PROMOTING THE OUTBREAK

1. *Predisposing Factors*

(*a*) *Climatic Conditions.* It is well known that in Africa epidemics of cerebrospinal fever take place in the cold dry season of the year.[1] The enormous epidemics in Nigeria in 1948-49 and in the Sudan in 1940 are examples of this phenomenon.

In the north-western region of the Orange Free State where St. Helena is situated, the winter is very dry. From the middle of July to the middle of September cold dusty weather prevails. The winds disseminate dust throughout this flat exposed region. The nights are bitterly cold and the relative humidity is low.

(*b*) *Increased Incidence of Respiratory-Tract Infections.* These weather conditions and the great difference in the temperatures between underground and surface conditions were responsible for the high rate of respiratory infection. An increased incidence of respiratory diseases tends to increase the proportion of carriers of the meningococcus and susceptibility to the overt disease.[1] The resultant coughing, expectoration and sneezing further increases the incidence of illnesses that spread by droplet infection. Tropical Africans are particularly susceptible to respiratory infections.

In July, August, September, October and November 31, 35, 51, 22 and 15 cases of respiratory diseases respectively were treated, including sore throat, bronchitis, influenza and pneumonia. The incidence declined with warmer weather in October and November.

(*c*) *The Accommodation* that is provided may be compared with a military cantonment. Most of the dormitories (see Fig. 1), which also act as living rooms, are situated on the circumference of the compound, which measures about 20 acres and encloses the kitchens and other amenities and some other dormitories. Thus a community existed, the members of which slept in common dormitories. The population at the time was approximately 4,000 persons (all African males), who were living much closer than is usually the case.

Each dormitory is occupied only by members of one tribe. Recruits are allocated to their living quarters according to their tribal origin. Each room receives a few recruits at a time, where they associate with veterans of the same tribe. A maximum of 20 miners are allowed to occupy each dormitory. They sleep on two-tiered bunks, on similar lines to those that prevailed in military camps in World War II. The distance between bunk centres is 6 feet. The cubic space allocated to each person is 290 cubic feet, when the room contains its full complement. This capacity is slightly in excess of the minimum, viz. 200 cubic feet, that is required by the Native Labour Regulations.

In March a near-by secondary compound was closed and about 400 Natives were transferred to the main compound. This transfer assisted in the creation of a greater population than was present in former years.

Under wintry conditions the inhabitants are inclined to huddle around a central stove in their living quarters, and they spend longer periods indoors; this renders their association still closer.

2. *Precipitating Factor*

Movement of Population. From the beginning of July until the end of August, large numbers of new recruits had been entering the service of the mine. Mine workers who had completed their contracts were also leaving the mine in fairly substantial numbers. Approximately 1,600 mine workers were engaged and 900 men were discharged, the population being increased by 700, i.e. from 3,500 to 4,200. The vast majority of immigrants were new recruits who had not previously worked in the mining industry.

In all mine compounds there is a continuous inflow and exodus of people. Recruits enter and workers who have completed their contracts depart for their homes. At St. Helena about 250 new workers ordinarily arrive each month and an equal number of persons depart. However, during the months of April, May and June immigration was greatly reduced while departures continued at the usual rate.

During July and August the number of persons involved in emigration and immigration was 2,400—more than 50% of the total population. The turnover of population was therefore very great. By the end of August 37% of the total labour complement consisted of new recruits. These were the susceptibles.

It was observed that the majority of the new workers, viz. 90%, were residents of the tropical countries of Nyasaland and Tanganyika. By the end of August, the tropical population was 1,478, and of these 876 had arrived within the preceding 2 months, i.e. 59% were susceptibles. They emigrated from rural areas where they were not accustomed to the close association in large groups that occurs in mine compounds.

The average period of service of African labourers is 9 months.

THE EPIDEMIC

Preliminary Phase

On 24 July 1953 a case of meningitis, which ended fatally, was admitted to hospital. He was a recruit who had arrived only 3 days previously. A 2nd case was diagnosed on 27 July. This patient was also a recruit, who had only been engaged 6 days before his admission to hospital. Both had arrived on the same day, viz. 21 July, as members of a large batch of new recruits. On 3 August a 3rd case occurred.

No further persons contracted the illness during the following 16 days.

On 19 August another case was diagnosed. Soon afterwards (on 23 August) case 5 reported to hospital. On 25 August 2 more cases were admitted and next day 2 more.

We noticed that 8 of the 9 cases were immigrants from Nyasaland and Tanganyika.

It was decided to apply certain preventive measures immediately. In the meantime the 10th case was admitted on 28 August.

Preventive Measures

Unfortunately, as there was no reserve accommodation in the compound, we could not adopt the method of increasing the cubic space per person, which had proved to be so successful in British Army barracks [2] in combating epidemics. The possibility was explored of accommodating all new recruits in special dormitories for a period of 1 month before distribution amongst the general population. This objective proved to be impossible.

Active immunization by the injection of meningococcal vaccines not having proved successful (Bevan's mass administration of vaccines in the Cyprus epidemic in 1938 showed inconclusive results [6]), other measures had to be considered. We resorted to the following prophylactic measures:

(*a*) *Reduction of Immigration.* We strongly recommended that recruits should be temporarily prevented from joining the service of the mine. The mine authorities found they could not do this for about 10 days, because they were under contract to the recruiting companies; but by 5 September the influx of immigrants was considerably reduced, particularly that of tropical Africans.

(*b*) *Prophylactic Sulphonamide.* As the cases were nearly all in members of dormitories that were inhabited by Nyasa or Tanganyika tribesmen, it was presumed that the major source of the infection was meningococcal carriers who belonged to these tribes, which, as already stated, supplied the great majority of recent immigrants.

Since 1940 it has been proved on several occasions that the administration of sulphonamides reduces the carrier state by destroying the meningococci that are harboured in the nasopharynx. Meehan and Merrilees [8] showed that sulphapyridine caused a total disappearance of the carrier state in a certain section of a foundling home in Melbourne where several epidemics had occurred. Painton [9] proved that the administration of 5 g. of sulphadiazine simultaneously to 18,000 troops in an American Army camp immediately terminated an epidemic. Three divided doses were issued within a period of 24 hours. Phair,[10] in a survey in the United States Army discovered that in 2 groups of 100 men the carrier state was 40%; the administration to one group of 1 g. of sulphadiazine twice daily for 3 days

caused a decline of the carrier rate to 1% by the 2nd day after administration while the control group still showed a rate of 48%. A low carrier rate in the 1st group was maintained for 3 weeks, but had increased to 10% in 2 weeks.

We therefore decided to disinfect local carriers by the issue of sulphonamide to each member of tropical tribes (1 g. sulphapyridine twice daily for 3 days). On Friday, Saturday and Sunday, 28, 29 and 30 August respectively, 2 tablets were issued twice daily to each man living in the the 'tropical' rooms. This procedure was carried out under personal supervision. Each miner was required to open his mouth to show whether he had swallowed the tablets and also to open his hands. Each room was visited and the inmates were paraded. Tablets were swallowed under direct observation and the fact noted in a special register; 1,400 tropical Africans received sulphapyridine. The precautions were necessary because a few of the men tried to retain the tablets in their cheeks or hands. After the tablets were placed in the mouth the person was instructed to swallow a cup of water.

This was arduous and monotonous work, especially the night parades in bitterly cold and windy weather. It was undertaken by 4 persons, including ourselves, and was thoroughly performed. Each person supervised the issue to a quarter of the tropical complement.

Only 2 persons complained of unpleasant symptoms that might be ascribed to the sulphapyridine, one of nausea and vomiting and the other of burning on micturition.

This same dosage of sulphonamide was administered to the distributors of tablets. It was presumed that this would confer temporary immunity which would give protection while they were in close contact with tropical Natives, who were being paraded twice daily in their dormitories.

Sulphapyridine tablets, 1½ g. twice daily for 2 days, were also issued to all recruits who entered the mine during the following week.

(*c*) *Household Dust.* All sweeping of rooms was cancelled for 5 days. The objective was, as a temporary measure, to prevent dust dissemination from the floor that may have been contaminated by the sputum and respiratory droplets of carriers. Subsequently only wet sweeping methods were allowed. All dormitories occupied by tropical Africans were sprayed with 10% formalin by means of a stirrup pump.

The men were also advised to remain outside of their rooms as much as possible, but this was difficult after 4 p.m. because of inclement weather. They were also advised about the danger of spitting on the floors of their rooms. During the day, while the majority were at work, the windows were kept open to increase ventilation.

(*d*) *Isolation of Cases.* All cases of cerebro-spinal fever were isolated in the hospital for 2 weeks.

Further History of Epidemic

On 31 August (the day after the issue of sulphapyridine) a case of meningitis was diagnosed. However, his cerebrospinal fluid was clear and not under pressure, there was a slight increase of cells, and he was considered to be an abortive case. He was a non-tropical. No cases of meningitis were observed during the following week, and the epidemic seemed to have ceased, but on 8 September a patient (the 12th) was admitted. Three days later another tropical African was diagnosed as a case.

The epidemic was now resurrected, because further cases occurred on 11, 15, 16, 18 and 19 September respectively.

An epidemiological map of the compound, on which each case was plotted as it was confirmed, showed that all the recent patients had been admitted from the northern side of the compound, and that no cases had occurred in dormitories on the western part of the perimeter. However, more recruits had been housed in this area than in the other dormitories. A south-westerly wind was continuously blowing dust across the courtyard of the cantonment, and mainly into the rooms on the northern part of the perimeter. We presumed that the increase of dust, together with the greater entrance of recruits into these dormitories, was the cause of the increased incidence of meningitis in this quarter. The dust, probably contaminated with meningococci by sputum and respiratory droplets from carriers, was disseminated by the movements of the inmates and inhaled.

We decided to continuously water the non-grassed areas of the compound, more particularly the belt in front of the rooms. This was continued for 6 weeks, and millions of gallons of water were used. It reduced the amount of air-borne dust considerably.

The north-western perimeter rooms that were mainly affected at this stage were once again the Nyasa and Tanganyika dormitories.

On Sunday, 20 September, 350 tropical Natives from this area were injected with penicillin in oil (300,000 u.) in a further attempt to disinfect carriers. Two days later another 250 tropicals were injected at random when paraded for weighing.

Final Phase

Another 5 cases (making up the total of 22) occurred at gradually increasing intervals of 4, 5, 6, 7 and 12 days respectively until 23 October, when the epidemic ceased. The 21st case was fatal. The weather became much warmer from about 20 September and the rains commenced towards the end of October.

Fig. 1 shows the distribution of cases in the dormitories, and Fig. 2 the weekly incidence of cases.

Bacteriological Investigations

All cases were diagnosed by examination of cerebrospinal fluid. The organisms were observed on direct stained smears or, in the majority of cases, after culturing the cerebrospinal fluid on blood-agar in the hospital laboratory. It was found that, when the meningococci were scanty, it was preferable to incubate the cerebrospinal fluid in blood-broth for 12 hours and then plate on blood-agar and incubate at 37°C for 12 hours, when colonies were easily discerned.

There were no local facilities for carrying out serological or fermentation tests, and transport to the South African Institute for Medical Research, Johannesburg, was only occasionally available. However, on 3 occasions nasopharyngeal swabs from contacts were plated on

Fig. 1. Compound of St. Helena gold mine: distribution of meningitis cases. Approx. scale 1 inch = 350 feet.

Rooms blacked in are dormitories of tropical Africans; the great majority of recruits were accommodated in these rooms.

Each black dot indicates a case of meningitis in the adjoining room. The numerals denote the sequence of cases according to admission to hospital.

The arrows represent the direction of prevailing S.W. wind.

The diagram shows the greater incidence of meningitis in the N.E. sector of the compound.

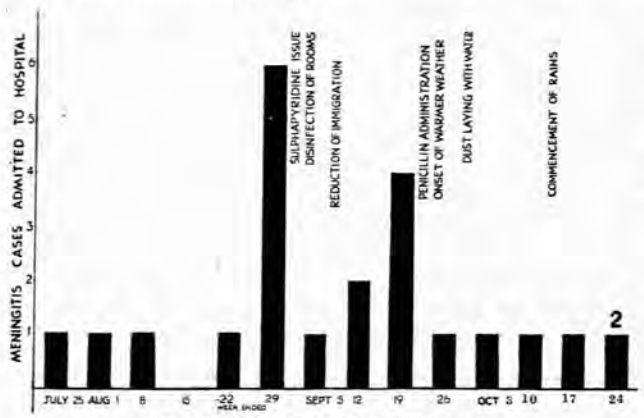

Fig. 2. Weekly incidence of cases.

blood-agar and the plates after incubation were sent to the Institute by motor vehicle (during transport they were not incubated). In this way cultures of swabs taken from 50 room-contacts were examined by the Institute. In all of these the Institute reported that no meningococci were isolated. The 3 occasions were as follows:

(i) In September nasopharyngeal swabs were taken from 17 contacts of case 8. This was done 1 week after the patient had been removed to hospital, and after sulphapyridine tablets had been distributed. The swabs were plated on blood-agar and after incubation for 12 hours the plates were sent to the Institute. No meningococci were isolated.

(ii) It had been observed that 4 cases of meningitis had been admitted from a certain dormitory. All inmates (16) of the room were swabbed and cultures made. On direct observation of the colonies, 2 persons out of the 16 were found to harbour organisms which according to morphology and staining characteristics were meningococci. However the Institute reported that meningococci were not isolated.

(iii) After the fatal case in October all inmates (17) of the patient's room were swabbed, and culturing was performed and the plates sent to the Institute.

Clinical Features

Of the 2 deaths (the 1st case and the 21st) the first was in a case which developed haemoglobinuria and fatal uraemia. The cause of this was probably the toxic action of sulphapyridine, by which haemolysis of the red cells occurred. The second was a case of fulminating meningitis of the encephalitic type. He died within 3 hours of admission.

Patients were treated with sulphapyridine, 2 g. stat. and 1 g. 4-hourly, by intramuscular injections for 36 hours and then orally for 3½ days more. Penicillin, 100,000 u. in 3 c.c. of distilled water, was injected intrathecally when the diagnostic lumbar puncture was performed. Pyrexia was relieved after an average interval of 72 hours. One case developed septic arthritis of the right knee. No cases of meningococcal septicaemia were observed. The recovery of the remainder was uneventful and after isolation for 14 days they were discharged.

General Observations

Racial Distribution. Eighteen of the 22 patients were tropical Natives (14 Nyasa and 4 Tanganyika). The others were 2 Xhosa and 2 Basuto.

Geographical Distribution. Eighteen dormitory rooms were affected out of a total of 230 rooms. Fourteen of these rooms were occupied by tropical Natives. The total number of 'tropical' dormitories is 57. Thus 25% of their rooms were affected.

Most of the persons who suffered from the illness were not room-contacts of actual cases of meningitis. However, in one dormitory there were 4 cases amongst the inmates and in another room 2. In 8 instances adjoining sleeping quarters were affected.

Three of the 4 non-tropical sufferers were housed in rooms adjoining those occupied by tropical Africans where meningitis had occurred.

In the earlier part of the epidemic, cases were irregularly distributed in various parts of the compound. The latter half of the epidemic displayed a much greater concentration in the north-eastern sector.

Meningitis is essentially a disease that spreads by close contact in dormitories. This factor increases the opportunities for droplet infection. Although employees are housed according to their tribal origin, working gangs both on the surface and underground consist of members of several tribes. Therefore the fact that the infection was confined mainly to tropical Natives, suggests that the infection was contracted in their sleeping quarters

and not at their working places, where in fact contact was not so close.

This observation does not however prove where infection was contracted, for the health records of the gold-mining industry over several years show a higher incidence of meningitis in tropical Africans.

Period of Residence. The majority of the meningitis sufferers were recent immigrants. The average duration of residence before the illness was contracted was 70 days. The average time that each mine worker spends on the mine is 270 days. Eleven of the patients had been employed for less than 30 days. Ten cases were recruits who had arrived during the epidemic.

Temporary Protection. Three Natives developed meningitis in spite of the fact that sulphapyridine was administered to them at the end of August, and 2 although they had received both sulphapyridine at the end of August and penicillin 3 weeks later. Only 3 persons who had commenced work after sulphapyridine was administered *en masse*, developed cerebrospinal fever.

These internal antiseptics had only a temporary effect in protecting persons or in removing meningococci from the nasopharynx of carriers.

Contemporary Epidemics

Epidemics of cerebrospinal fever were also present on several of the neighbouring mines. They were receiving recruits from the same recruiting depots as the St. Helena gold mine. It is quite possible that a fair proportion of the recruits were already carriers before arrival on the property. They had travelled from territories that are 1,500-2,000 miles north of St. Helena. During their journey southwards they had been housed temporarily in several depots and had the opportunity to develop into carriers. For this reason our recruits differed from those who entered the British Army, because the latter proceeded straight from their homes to the army barracks.

COMMENTS

The epidemiology of meningitis was not understood until Glover [3] performed his epic work in the British Army during the First World War. He proved that infection by the meningococcus was primarily one of the nasopharynx. Some of the infected persons showed signs of catarrh and sore throat, while others were quite asymptomatic. These were the carriers. It is only in a small minority of infected hosts that the meningococci break through the barriers of the nasopharynx, invade the blood stream, and attack the central nervous system.[7]

The epidemiology of meningitis in this respect resembles that of poliomyelitis, yellow fever and bacillary dysentery and is in marked contrast to that of smallpox and measles.

The factors that influence the breakdown of the nasopharyngeal barrier and the invasion of the blood by meningococci are largely associated with individual susceptibility.[7] Increased susceptibility results from upper respiratory infections and is promoted by close association of humans who are not accustomed to such conditions.

The large influx of recruits reduced the general herd immunity at St. Helena and the epidemic increased while the influx continued. This is in accordance with Greenwood's epidemiological experiments carried out on mice.[4]

It will be seen from the map of compound rooms (Fig. 1) that where recruits went in meningitis broke out. Tropical Africans were mainly afflicted because the great majority of immigrants came from tropical regions, and also because these people are more susceptible to cerebrospinal fever.

As stated above, only 50 room-contacts of cases of cerebrospinal fever were submitted to nasopharyngeal swabbing and subsequent culturing. The blood-agar plates were examined at the South African Institute for Medical Research and no meningococci were isolated. Possibly meningococcal colonies died out *en route*, because the plates were not incubated during the journey of 6 hours. No conclusion about the carrier rate can be drawn from this small sample of the population.

After the mass administration of sulphapyridine to the affected section of the labour complement there was a marked fall in the incidence of the disease for a period of 2 weeks. During this time weather conditions were still cold and immigration continued at a high rate. It is therefore assumed that the remaining factor (sulphapyridine administration) caused a decline in the epidemic.

When the epidemic became resurrected in the 3rd week of September penicillin was administered, on the presumption that it would also have an effect in eradicating the carrier state. It will be seen from Fig. 2 that a decline in case incidence followed this action. However, weather conditions had become warmer and immigration had been considerably reduced at this stage. It is thus difficult to assess the effect of the penicillin administration. We are, however, of the opinion that it was in part the cause of the sudden reduction of incidence at this stage.

Lack of reserve accommodation prevented us from reducing the number of persons in each dormitory and thereby increasing the cubic space for each inhabitant. We were thus deprived of a most important preventive weapon.

The curtailment of immigration had no immediate effect; for the incidence of the disease increased again 10 days after the adoption of this procedure. There is nevertheless little doubt that the restriction had a long-term effect in causing the ultimate decline of the epidemic.

It is difficult to assess the results of the general hygienic operations of wet sweeping, disinfection of rooms, and dust-laying. The onset of warmer weather and summer rains would, in the natural course of events, have eventually terminated the epidemic by virtue of the greater amount of outdoor life led by the inhabitants and the dust-laying effect of rain. Meningitis, in contrast to the ancient Roman legions, carries out its campaigns in winter and then goes into summer quarters.[5]

The case mortality rate in cerebrospinal fever, under the best conditions that modern treatment can offer, varies between 5% and 10%. Preventive measures are therefore imperative.

SUMMARY

An outbreak of cerebrospinal fever (22 cases) in a semi-closed community is described.

Large-scale immigration of recruits was the main precipitating factor in causing the outbreak.

The majority of sufferers were new recruits.

Mass administration of sulphapyridine to affected elements of the population resulted in a marked but temporary decrease of the dimensions of the outbreak.

Mass administration of penicillin to inhabitants of one area of the compound, where the epidemic had become localized, was followed by a further decrease in the incidence of the disease.

Other preventive measures consisted of restriction of immigration, disinfection and wet sweeping of rooms, dust-laying by means of water, and isolation of cases.

Owing to lack of reserve accommodation, the cubic space per head could not be increased.

The epidemic occurred during the dry cold season. No further cases were observed after 23 October soon after the onset of summer rains.

We wish to record our thanks to Dr. K. Sartorius, Chief Medical Officer of Union Corporation, for his advice on preventive measures and for his permission to publish this report, and to the members of the Bacteriology Department of the South African Institute for Medical Research, Johannesburg, for the investigations they performed.

We also wish to express our appreciation for the help and co-operation that we received from Mr. D. Venables (compound manager) and Mr. J. Kotze (of the compound staff), who organized the mass administration of sulphapyridine, and from Mr. D. Harebottle (hospital superintendent), Mr. P. Malherbe (ambulance officer) and Mr. H. van Rensburg, who helped in the distribution of sulphapyridine and penicillin.

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