SYMPTOMLESS CARRIERS IN HOME CONTACTS IN SONNE DYSENTERY

BY

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The presence of symptomless carriers in outbreaks of Sonne dysentery has either been suspected or demonstrated by numerous workers (Charles and Warren, 1929; Laws, 1936; Bloch, 1938; Manson-Bahr, 1943), but I have been unable to trace any report on their incidence in large-scale outbreaks within the general population of a town. The present investigation was made during an epidemic of Sonne dysentery which originated in an infant school in Oxford in January-March, 1951, when an attempt was made to find out: (1) the number of symptomless carriers among the home contacts; (2) whether immediate exclusion of all siblings from school would limit the spread of the outbreak to other schools; and (3) whether the density of infection within any household was related to the standard of cleanliness of that household.

Details of the Outbreak

In January, 1951, shortly after the beginning of term, an outbreak of Sonne dysentery occurred in an infant school in Oxford. During the remainder of this school term the spread of the epidemic was investigated. The same health visitor was employed full-time to visit the school and homes of those affected, and she tried to persuade all home contacts to send their stools for bacteriological examination.

The siblings of a case were immediately excluded from school; this was the only direct preventive measure taken. Exclusion continued until all clinical evidence of infection had ceased in the household and the sibling himself had negative stools. In all cases the ruling of no symptoms plus one negative stool examination was adopted as "a cure." It was realized that this was a very low bacteriological standard on which to assess recovery, but it was accepted as the most practical one, and the results do not suggest that it led to any spread of infection.

There were 74 primary cases, all occurring in school-children, and these had 293 home contacts. At least one stool examination was obtained from 234 of these home contacts (130 adults and 104 children); the 59 home contacts not examined included 56 adults and 3 children.

Home Contacts.—The results of the stool examinations of home contacts (see Table) show that 38 (16.3%) of those examined later became clinical cases, 78 (33.3%) were symptomless carriers, and 118 (50.4%) had negative stools.

234 Home Contacts Whose Stools were Examined

Ages	Clinical Cases	Symptomless Carriers	No Symptoms. Stool's Negative	Total Contacts
Adults:				
Male	4 (10%)	12 (26.9%)	34 (63.1%)	50 (100%)
Female .. .	9	23	48	80
Children:				
8-15 years ..	7 (13.8%)	19 (37.2%)	25 (49.0%)	51 (100%)
0-7 " .. .	18 (33.9%)	24 (45.3%)	11 (20.8%)	53 (100%)
Total .. .	38 (16.3%)	78 (33.3%)	118 (50.4%)	234 (100%)

It is interesting that the number of symptomless carriers is double the number of clinical cases in the home contacts. The younger the age group the greater the proportion of home contacts who became either cases or symptomless carriers, and the difference noted between the adult and the 0-7 age group is statistically significant. Among the home contacts were 59 school-children, all of whom were

excluded from school and their stools examined. Of these children, 15 (25.4%) later developed a clinical attack of dysentery which was confirmed bacteriologically, 23 (39%) were found to be symptomless carriers, and 21 (35.6%) had no symptoms. These figures emphasize the high level of infection present among the siblings.

Spread within the Schools.—Of the primary cases, 47 (63.5%) came from the infant school in which the outbreak started; the remaining 27 attended 10 other schools, but all had either a history of personal contact with a case from the infant school or else lived near by. The infected siblings of the primary cases included children from seven additional schools, so that altogether 18 different schools contained cases or symptomless carriers. Marked spread within a school occurred only in the infant school originally attacked, and then only in two nursery classes, where spread of infection must have occurred between children within the classroom, as this was the only meeting-place for some of those who developed dysentery. In all other cases the home rather than the school appeared to be the place of infection.

Density of Infection and Cleanliness of Home

As regards density of infection, the following classification was used:

- (1) Families in which all members were found to have stools positive for *Shigella sonnei* 11 (15.1%)
- (2) Families in which more than half the members were found to have stools positive for *Shigella sonnei* 28 (38.3%)
- (3) Families in which less than half the members were found to have stools positive for *Shigella sonnei* 34 (46.6%)

In devising cleanliness categories all the homes were classified by the same health visitor, in the hope that the human error in this difficult classification would be reduced to a minimum. Four cleanliness categories were used, and results were compared both between the distribution of them within the sample as a whole and within the three density-of-infection groups mentioned above. The numbers were small, but analysis showed no connexion between the density of infection and cleanliness of the home: the disease was not more prevalent in dirty homes than in clean homes.

Discussion

The finding that in addition to the 16.3% of home contacts who developed a clinical attack of dysentery 33.3% became symptomless carriers shows that infection within the home was widespread, and suggests that, if symptomless carriers are a real danger, preventive measures might reasonably be mainly directed against spread of infection by them. An assessment of this danger can be made in adults, as no preventive measures were imposed on them (except for two persons working in the food trade) other than by stressing the importance of personal cleanliness. The subsequent absence of any general spread of Sonne dysentery in Oxford suggested that those adult symptomless carriers were of little or no danger to the community.

In the case of young children the problem is different, as with them there is a greater tendency for direct person-to-person infection to take place. The spread of infection in the epidemic described seemed to be mainly within the home, and it was only when a heavy density of infection was reached that the schoolroom appeared to play any prominent part. These findings suggest that immediate exclusion from school of all siblings of cases is a reasonable routine preventive measure, and the only one necessary.

Summary

An outbreak of Sonne dysentery which occurred in Oxford from January to March, 1951, is briefly described. Exclusion from school of siblings of cases was the only direct preventive measure adopted.

The results of examining bacteriologically the stools of 234 home contacts are given: 78 (33.3%) were found to be symptomless carriers.

Spread of infection within the schools is described. Apart from the original infant school affected, direct spread within the classroom did not appear to have been the usual mode. The home played a greater part in spreading infection.

Results did not suggest that the density of infection was greater in dirty than in clean homes.

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REFERENCES

- Bloch, E. (1938). *British Medical Journal*, 1, 836.
Charles, J. A., and Warren, S. H. (1929). *Lancet*, 2, 626.
Laws, J. J. (1936). *Ibid.*, 1, 192.
Manson-Bahr, P. (1943). *The Dysenteric Disorders*, 2nd ed. London.

AN OUTBREAK OF TRYPANOSOMIASIS

BY

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In December, 1948, a male African was admitted to the Native Hospital, Kisumu, in the Nyanza Province of Kenya, complaining of fever, headache, and general malaise. His temperature was 102° F. (38.9° C.), and in the posterior triangle of the neck several glands were enlarged to about the size of a hazel-nut. On examination of a blood slide I saw what appeared to be typical trypanosomes. The man was a railway employee from Kibigori, a village some 20 miles to the east of Kisumu. With the exception of three days in August, 1948, when he had returned to his home some 50 miles to the west of Kisumu, where sleeping sickness was known to exist, he had been continuously resident in Kibigori for nine years.

Although human trypanosomiasis was not known to exist at Kibigori, the tsetse fly *Glossina palpalis* was present along the whole course of the Nyando River, on which Kibigori stands, and its tributaries, from a point about 15 miles east of Kisumu. The Nyando River is about 75 miles long and enters Lake Victoria near Kisumu.

The Veterinary Department was at the time making a tsetse fly survey of the area in connexion with trypanosomiasis in cattle. Because of this case and the presence of *G. palpalis* at Kibigori, it was decided to investigate the area for further cases of human trypanosomiasis.

Present Investigation

The initial investigation was made on the station platform at Kibigori. Africans in the vicinity were persuaded to come to the station, where they were examined for the presence of enlarged axillary or cervical glands. When enlarged glands were found they were punctured and the gland fluid was examined under the microscope for trypanosomes. In this way some 15 cases were discovered in about a week. It became evident, therefore, that it would be necessary to extend the investigation over the whole area of the Nyando basin; this included two native reserves, European- and Indian-owned farms, and some newly established gold mines.

The investigating team consisted of two African laboratory attendants, an African dresser, and myself. The co-operation of the local chiefs in the native reserves was readily obtained, and with their assistance their people were persuaded to come to prearranged points for examination. The position as regards the examination of African farm labour was more difficult. It was necessary to address several farmers' meetings in order to explain the purpose of the investigation and to obtain their co-operation so that their native labour would come forward in sufficient numbers and at such times as to make the investigation practicable. A prearranged schedule was arranged with the farmers' representatives, and the team went from farm to farm examining the units of labour on each.

In all cases the method of examination and diagnosis was by palpation of cervical and axillary glands, gland puncture, and examination of the gland fluid directly under the $\frac{1}{4}$ -in. (4-mm.) objective of the microscope. The trypanosomes were recognized by their movement. This was found to be the simplest and quickest method, although it became tiring if a large number of enlarged glands were found. While the gland puncture was being done a blood slide was taken, numbered, and later examined—usually in the evenings. Staining a smear from the glands was one of the other methods tried, but this was found to take too long and to be too difficult to carry out in the dusty places where it was necessary to work. Lumbar puncture for C.S.F. cell counts and Pandy's test was also carried out, but the difficulties of doing this in the field, and in particular the adverse propaganda which it provided, soon ruled it out as a diagnostic measure.

At the beginning of the survey, if a patient was found with a positive blood, a sample of the blood was taken and injected intraperitoneally into a rat. In the laboratory the trypanosome was found to cause chronic infection in white rats, with short stumpy forms in the peripheral blood. These blood samples were discontinued after the trypanosome had been propagated in the laboratory. In each positive case the patient was questioned closely about his symptoms, which were often described with a characteristic vagueness. Some complained of no symptoms whatsoever, whilst others complained of many symptoms—lassitude, fever, headache, loss of appetite, vague skeletal pains, and general malaise. Most of them, however, had only transient attacks of fever and malaise. On general examination about one-third showed moderately severe enlargement of the spleen. This could not be considered unusual in an area where malaria was hyperendemic. About one-quarter of the cases showed some rise in temperature, and at least one-half had some form of rash, usually scabies.

In an area such as this, where many tropical diseases—malaria, bilharziasis, worm infestation, etc.—were endemic, no great reliance could be placed on ordinary signs and symptoms to form a single diagnosis. The character of the axillary glands, and especially the posterior cervical glands, was the most distinctive point. These glands had a characteristic rubbery consistency; they were single, were about the size of a hazel-nut, were mobile, and could be grasped between the fingers and thumb. After a little experience diagnosis could almost be made by the character of the glands alone, but confirmation was always made by gland puncture and demonstration of the trypanosome under the microscope. A few cases were found which gave a negative gland puncture but showed a positive blood film. These people were traced and told to report for treatment.

As a result of taking careful histories and obtaining details regarding movements with such accuracy as it was possible to obtain from Africans, it became evident that the main focus of infection was an area around Kibigori, with a small isolated subsidiary focus some 15 miles away.

Treatment

Treatment of cases was started at once, and throughout the survey weekly sessions of treatment were held every Tuesday at Kibigori, Tuesday being market day. As each