

THE ABERDEEN EPIDEMIC OF MILK-
BORNE BACILLARY DYSENTERY,
MARCH TO MAY, 1919.

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BACILLARY dysentery occurs in endemic and epidemic form. It appears in temperate regions as well as in the tropics, and while in the past it has not been regarded as a common disease in Britain, it has almost always been the cause of dysenteric outbreaks in ships, in camps and in institutions in this country, where the presence of carriers permitted recurring outbreaks of the disease in epidemic times. It has developed every now and then in British seaports, where it has been introduced by travellers from the East, and the serious milk-borne epidemic of bacillary dysentery in Aberdeen early in 1919, as about to be described, has provided abundant justification for the action of the Scottish Board of Health in making bacillary dysentery compulsorily notifiable, as a precautionary measure in anticipation of the probable spread of bacillary dysentery among the civilian population owing to increased exposure to infection resulting from the demobilisation of an unmeasured number of dysentery carriers.

The great importance of the Aberdeen epidemic of milk-borne dysentery, however, depends on the fact that its elucidation has provided for the first time bacteriological proof of the nature of a definite group of diarrhoeal epidemics due to milk infection. Epidemics of milk-borne diarrhoea have been experienced in the past in this and other countries, and a typical epidemic of this kind occurred in Aberdeen in 1908. Bacteriological investigation of such former outbreaks had entirely failed to determine the nature of the infecting organism, and in the absence of such information, and in view of the fact that the infection had not been proved to be of human origin, attention had been directed to the possibility of such infection having a bovine source. Reference to the epidemiological and clinical features of such former epidemics, however, makes it practically certain that they were essentially of the same description as the Aberdeen epidemic of 1919. That the 1919 Aberdeen epidemic has been proved to be due to infection of milk with dysentery bacilli of the Flexner type, warrants the results of the investigation being recorded with considerable detail.

EPIDEMIOLOGY.

On March 9th, 1919, it became evident that a widespread epidemic of acute enteritis had developed suddenly in Aberdeen, the first of the cases having had their onset two days previously. A circular letter was at once addressed

to the medical practitioners in the city, and with their cooperation a total of 978 cases of fever with pronounced diarrhoeal symptoms was eventually brought to the knowledge of the Health Department as having occurred in the epidemic period—March to May, 1919.

In the absence of compulsory notification, information concerning the total number of cases in the epidemic was necessarily incomplete, but, on inquiry, the number of deaths from the disease, however certified, was obtained with considerable accuracy in consequence of the prominence of dysenteric symptoms. In the epidemic period, altogether 978 cases were recorded, and of registered deaths, altogether 72 were determined to be deaths from bacillary dysentery contracted during the epidemic period. With a total of 978 cases and 72 deaths, the case mortality is about 7 per cent., but since a considerable number of cases was not put on record, the proportion of deaths to cases is unduly heightened, and gives no certain indication of the true case mortality. Of 650 of the cases in which the sex was determined, 249 were males and 401 females, giving a proportion of 38 per cent. of males to 62 per cent. of females. Of 657 cases in which the age distribution was determined it was found that 2·9 per cent. were aged 0 to 2 years, 5·2 per cent. were aged 2 to 5 years, 7·9 per cent. 5 to 15 years, 12·2 per cent. 15 to 25 years, 32·1 per cent. 25 to 45 years, 27·5 per cent. 45 to 65 years, and 12·2 per cent. 65 years and upwards. This age and sex distribution has many of the peculiarly selective characteristics of a milk-borne epidemic. Of the 657 cases 249 occurred in the Royal Mental Hospital, 31 in the Morningfield Hospital for Incurables, and 21 in the Royal Hospital for Sick Children. In the Royal Mental Hospital and the Morningfield Hospital the disease was found to be excessively lethal to the aged. In the Royal Mental Hospital there were 28 deaths; in the Morningfield Hospital, seven deaths; and in the Sick Children's Hospital, no deaths. Adopting an arbitrary period of seven days as the limit of the period of incubation of bacillary dysentery from the time of the milk infection, it was found that 133 out of 784 cases of dysentery could be attributed to contact infection. These figures are only submitted for what they may suggest, reliable notification data not being available.

Milk Infection. On first inquiry, on March 9th, it was ascertained that all the cases were receiving milk from one dairy. Further inquiries at certain large institutions supplied with milk from the dairy showed that a considerable number of diarrhoeal cases had occurred within the preceding two days. Accordingly, arrangements were at once made for the pasteurisation on the following morning of all the milk being distributed from the dairy, and this precaution was continued until the source of infection was accurately determined and eliminated. The dairy proved to be one of the largest in the city and one of the best conducted, and inquiry and examination there made it practically certain that the infection had not arisen in the handling of the milk within the dairy itself, but had been introduced through the pollution of one or other of the 36 milk supplies obtained from various farms in the surrounding

districts, including four farms in southern Scottish counties. All the farmers were at once communicated with, and a reply was received from one of the farms in the vicinity of Aberdeen indicating that certain cases with diarrhoeal symptoms had occurred in the household of the farmer a few days before the outbreak in the city. Further investigation of the cases at the farm made it clear that they were of the same type as those in the city, and that they had been the source of the infection. The farmer's wife and two children were suffering from diarrhoea at the time the epidemic originated, and one of the children ultimately died of the illness. Not only so, but one of the milkmaids was at the time recovering from a similar acute diarrhoea which had begun on March 1st, and had endured for a week, and which, although very urgent, had not prevented the maid carrying on her daily work as a milker.

It was of interest to trace the course of transmission of the infected milk from the farm to the dairy, and its subsequent distribution in the city. The milk from the infected farm arrived at the dairy in the forenoon and was bulked with other milk consignments in a vat containing about 500 gallons of milk for distribution on the following morning. As the milk from this vat was run off to the first distributing milk carts early in the morning, consignments of milk from other farms arriving sufficiently early for distribution on the same morning were added to the vat. In all, ten milk carts were sent out each morning, each taking from 80 to 90 gallons of milk. The first six were loaded about 6 a.m., and the remaining four about 6.30 a.m. Of the first six carts, two supplied milk to Ferryhill district, two to Rosemount district, and one to Kittybrewster, and these were the districts of the city in which the incidence of the disease was by far the greatest. The sixth cart supplied 70 gallons to the Royal Mental Hospital, where some 250 cases of diarrhoea occurred. The remaining four carts supplied other districts of the city, and in these districts the incidence of diarrhoea was slight.

As regards the possible source of the disease at the infecting farm, it was found that a sister of the farmer's wife was living at the farm at the time of the outbreak and had been resident there during the preceding two months subsequent to her return from nursing dysentery in Salonica. She was unaware, however, of ever having had any symptoms of dysentery herself.

Incubation Period. The incubation period of the Aberdeen infection was accurately determined in three individuals living in a suburb of the town beyond the distribution of the infected milk. These individuals had each partaken of milk on one occasion only in tea-rooms in receipt of infected milk, and with them the incubation period was exactly 48 hours. In most cases, however, the period appeared to be variable, extending from one to seven days.

Symptoms. The clinical appearances of bacillary dysentery in Aberdeen were similar in the essential features to the appearances of the disease as observed elsewhere. The dysentery was characterised by the presence of blood in the stools, and the choleraic type of the disease was not in evidence. The onset was sudden, with fever, diarrhoea and colic, and within two or three

days the stools contained mucus and frequently blood. In cases ending in early death prostration and general toxæmia were profound. In the majority of fatal cases, where death ensued in from three to four weeks, the early urgent diarrhoea and hæmorrhage were followed by the more irregular and excessively offensive diarrhoea and general misery that accompany the extensive pathological changes in the great intestine.

Treatment. The epidemic had run its course for about four weeks and had caused 72 deaths before serological proof of bacillary dysentery was available. From that time onwards, contact cases, treated at once with intravenous injections of polyvalent anti-dysenteric serum in hypertonic saline and with the oral administration of magnesium or sodium sulphate, rapidly subsided, the patients returning to normal in from a week to ten days' time.

Bacteriology. In beginning the bacteriological investigation of an acute diarrhoeal disease of this type, it was obvious that while similar diarrhoeal infections due to milk had occurred previously, there was no evidence that bacteriological proof had been obtained of the causative organism in any epidemic. Sufficient work, however, had been done in the investigation of diarrhoeal diseases in general to point to the probability that the infecting organisms in most cases belong to the great group of aerobic, gram-negative, non-lactose fermenters. Specimens of faeces sometimes duplicated and triplicated were obtained from 89 patients suffering from the diarrhoeal infection, and it soon became evident on routine examination that non-lactose fermenting organisms warranting further investigation were appearing on many of the plates. Several of these seemingly non-lactose fermenters proved later to be slow fermenters of lactose, and produced acid and gas in glucose and mannite, and finally non-lactose fermenting organisms non-motile and gram-negative were isolated from the stools of 21 of the 89 patients.

These isolated organisms did not liquefy gelatin; they produced acid only on glucose, laevulose, mannite, maltose, dextrin, galactose and raffinose; they caused no change in dulcitate, cane sugar, lactose, inulin, salicin and sorbite; they formed no indol in peptone water; they produced acidity on the first day in litmus milk and thereafter the milk was alkaline. From two out of the 21 patients having organisms with these cultural reactions, the organism was obtained a second time in later samples of faeces.

It was evident that all these organisms were of the same type, and that they had the main characteristics of *B. dysenteriae* Flexner.

When, however, the serological reactions of these organisms were tested against the monovalent dysentery Y serum of the Lister Institute which covered the greatest range of mannite fermenters, no agglutination was obtained even in dilutions of 1 in 40. All available monovalent dysentery sera were next obtained from the Standards Laboratory, Oxford, by the courtesy of Dr A. D. Gardner, and it was found that the Standards Dysenteriae Flexner Monovalent Serum containing 100 agglutinin units per cubic centimetre, gave complete agglutination of all the isolated organisms in dilutions 1 in 160 to

1 in 320, and partial agglutination in much higher dilutions. It is only right to say that it is improbable that the full serological identification of the Aberdeen Flexner organism about to be described would have been attained in the absence of the agglutinating sera supplied in the first case by Dr A. D. Gardner on behalf of the Medical Research Council. The polyvalent dysentery serum of the R.A.M. College containing five strains of dysentery bacilli and having a titre of 1 in 4000, was next obtained, and constantly gave complete agglutination of the Aberdeen Flexner bacilli in dilutions of 1 in 1500. Further monovalent sera were put at our disposal by the courtesy of Dr C. J. Martin of the Lister Institute, and it was found that the monovalent Lister Institute *B. dysenteriae* (Y strain) Cable serum, titre 1 in 6000, gave complete agglutination of the Aberdeen Flexner bacilli in dilution 1 in 5120, and that the monovalent Lister Institute *B. dysenteriae* Flexner serum, titre 1 in 3000, gave complete agglutination of the Aberdeen Flexner bacilli in dilution 1 in 2560. The Aberdeen Flexner organism was not agglutinated by Dysentery Shiga serum, nor by normal blood serum.

Cultures of the Aberdeen organism from two patients were submitted to Miss Mabel Rhodes of the Lister Institute and Dr A. D. Gardner of the Standards Laboratory, and they have confirmed the results of our biochemical tests. Miss Rhodes found agglutination of the Aberdeen Flexner bacillus to the full titre of three monovalent sera, namely, *B. dysenteriae* (Flexner strain) Elstree serum titre 1 in 4000, *B. dysenteriae* (Y type Z strain) Whittington serum titre 1 in 4000, and *B. dysenteriae* (Flexner strain) Lentry serum titre 1 in 8000. Miss Rhodes also found agglutination to half the titre with *B. dysenteriae* (Y strain) Cable serum titre 1 in 8000, to quarter the titre with *B. dysenteriae* (Y strain) His and Russell serum titre 1 in 2000, and to one-eighth the titre with *B. dysenteriae* (Y type X strain) Hughes serum titre 1 in 4000. Dr Gardner found agglutination of the Aberdeen Flexner organism to half the titre of B. Flexner V (original Flexner bacillus) serum, to a fifth the titre of B. Flexner Y serum, and to a tenth the titre of *B. dysenteriae* Flexner W, X, Z and Shiga sera.

Evidence of the specificity of the isolated organisms as the cause of the Aberdeen epidemic was obtained when it was found that the sera of three of the patients out of a batch of eight sera examined agglutinated the isolated organisms in the following dilutions and days of illness.

No. of case	Day of illness	Agglutination of patient's organism	Agglutination of a known <i>B. dysenteriae</i> Flexner
1	21st	Complete, 1 in 400 Partial, 1 in 800	Complete, 1 in 200
2	23rd	Complete, 1 in 100 Partial, 1 in 200	Complete, 1 in 200
9	24th	Complete, 1 in 200 Partial, 1 in 400	Complete, 1 in 200

These three sera also agglutinated the organisms with Flexner characteristics isolated from the other infected patients.

It was of interest that the agglutinins of these patients' sera should be further tested against the standard cultures of the various types of Flexner

group bacilli supplied by the Standards Laboratory. The sera of the three patients showing agglutinins had contained no agglutinins during the first 20 days of illness, and after 30 days of illness these patients' sera failed to agglutinate not only the standard cultures but the Aberdeen Flexner bacillus itself. These results were corroborated by Dr A. D. Gardner, and it would appear that the sera of patients suffering from the disease developed agglutinins but feebly, and that in a month's time they had sunk below a measurable level.

Animal inoculation experiments were only undertaken after the Aberdeen Flexner bacilli had been on subculture for at least one month, and it was then found that large doses (the whole of a 24 hours' culture on agar) injected intraperitoneally were required to kill a rabbit.

The bacteriological results obtained from the investigation of cases of dysentery occurring at the farm where the original infection of the milk took place are of interest. Case No. 6 of the 21 cases was that of the farmer's child which subsequently died of the disease, and a typical Aberdeen Flexner bacillus fulfilling all the cultural and serological tests was isolated from the faeces of this child. As regards the nurse who was staying at the farm on her return from Salonica and who was suspected of being the originator of the infection, only two separate specimens of faeces were available for examination, and they gave a negative result, a few seemingly non-lactose fermenting organisms proving later to be fermenters of lactose, glucose and mannite. This negative result has but little significance, however, since, as is pointed out in the *Medical Research Committee Special Report*, No. 29, the infection is known frequently to be markedly intermittent in carriers of the Flexner bacillus, the organisms appearing in the stools for one or two days in succession and then being absent for four or five weeks before reappearing. This nurse's blood serum did not agglutinate any of the Aberdeen Flexner organisms, but it completely agglutinated a known *B. dysenteriae* Flexner culture in dilution of 1 in 160. She had not had a dysentery vaccine.

Pathology. Autopsies were obtained in seven of the 72 fatal cases of the disease.

One of these cases, that of a child aged two years and three months, showed an early stage of the disease process, death having occurred within 15 hours of the earliest manifestation of illness. In this case there was no apparent thickening of the bowel wall, but the whole mucous membrane of the great intestine was hyperaemic and practically every solitary follicle showed a pin-point necrosis similar to the earliest stage of follicular ulceration. There was none of this necrosis in the small intestine, but the Peyer's patches and solitary glands commonly prominent in children were greatly swollen in the lower half of the small intestine giving an admirable demonstration of the distribution of lymphoid tissue. In this case at least there could be no doubt that the starting-point of the disease was in the lymphoid tissue.

In the other six autopsies the duration of illness was at least three weeks. In all these cases the large intestine was extensively involved in the disease

process. The entire mucous membrane in each case was greatly swollen, and throughout the whole great intestine in two of the cases and in large areas in the others, the mucous membrane was gangrenous and showed extensive serpiginous ulceration where the sloughs had separated. In all six cases the entire intestinal wall was greatly thickened, and much of the ulceration had penetrated into the submucous coat, but the disease process had clearly originated throughout the mucous membrane with its lymphoid tissue. In this respect the radical difference between amoebic and bacillary dysentery was defined. In these six cases there was diffuse inflammation and a few ulcers in the small intestine, the ulcers being confined to the lower end of the ileum, and this localisation is in conformity with the unpublished findings of Captain R. Richards in 31 fatal cases of bacillary dysentery in which the ulceration in the small intestine in no case extended above the terminal five feet of the ileum.

In all seven cadavers there was definite swelling of the mesenteric lymphatic glands, most marked in the child dying early in the disease.

An Aberdeen Flexner bacillus fulfilling all the cultural and serological tests was isolated from the intestine of the child dying within 15 hours from the first onset of the disease. Cultures from the ulcerated intestine, the bile, the spleen and the heart's blood of the other six cadavers failed to show dysentery bacilli, with the exception of a non-lactose fermenter from one spleen. This organism gave all the cultural reactions of Flexner's bacillus, but failed to agglutinate with any of the agglutinating sera at our disposal.

CONCLUSIONS.

1. An epidemic of diarrhoea or gastro-enteritis occurring in Aberdeen in 1919, and causing over 1000 cases and 72 deaths, has been proved to be due to infection of milk with dysentery bacilli of the Flexner type.

2. Similar epidemics of milk-borne diarrhoea have occurred formerly, but bacteriological investigation had failed to determine the nature of the infecting organism. Reference to the epidemiological and clinical features of such former epidemics, however, makes it practically certain that they were essentially of the same description as the Aberdeen epidemic of 1919, and accordingly indicates that they were likewise due to infection of milk with dysentery group bacilli.

3. The Aberdeen epidemic of milk-borne bacillary dysentery had run its course and had caused 72 deaths before serological proof of bacillary dysentery was available. Since the time of the Aberdeen investigation, notable advances have been made in methods for the bacteriological diagnosis of the dysenteries as a result of the fundamental work of F. W. Andrewes, A. D. Gardner, C. J. Martin and others. Nevertheless, modern methods of bacteriological diagnosis are not infallible, and the Aberdeen experience clearly indicates that deaths will be prevented if polyvalent anti-dysenteric serum and saline treatment are used at once in a disease having the clinical symptoms of bacillary dysentery, or in an acute enteritis from which has been isolated organisms with the cultural reactions of the dysentery group bacilli.