ORIGINAL ARTICLE

Intussusception and the great smog of London, December 1952

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Aim: To discuss the possible significance of the increased incidence of intussusception in children in relation to the ''Great Smog'' of London in December 1952.

Methods: Cases of intussusception were recorded in two hospitals in East London for the years 1951, 1952, 1953, and 1954. For 1952 the actual dates of admission were recorded.

Results: During the year 1952 the total number of cases of intussusception greatly exceeded that in the previous and succeeding years. Immediately during and after the fog there was a clustering of cases, which only occurred during this period.

Conclusions: The increased incidence of cases during 1952 is thought to reflect the annual variation in incidence resulting from changes in the prevalence of viruses capable of causing intussusception. The clustering of cases in relation to the fog may reflect a facilitated entry of virus through the wall of the terminal ileum due to the effect of swallowed irritants such as sulphurous acid and smoke particles.

I asked him whether there was a great fire anywhere? For the streets were so full of dense brown smoke that scarcely anything was to be seen.

''Oh dear no, miss'' he said. ''This is a London particular''.

I had never heard of such a thing.

''A fog, miss'', said the young gentleman.

''O indeed!'' said I.

(*Bleak House*, Chapter 3; Charles Dickens, 1812–1870)

Then, in London, above all what I love is the fog ... It is the fog that gives it its magnificent breadth. Those massive regular blocks become grandiose within that mysterious cloak.

(Claude Monet, 1840–1926)

During the ''Great Smog'' of December 1952 I was responsible for admissions to The Hospital for Sick Children, Great Ormond Street, London. I noticed an unusually large number of cases of intussusception during this period. I did not investigate this at the time but have recently examined the pattern of admissions for intussusception to Great Ormond Street and to the Queen Elizabeth Hospital for Children, Hackney, London, during the years 1951, 1952, 1953, and 1954. The ''Great Smog'' was an extremely dense and polluted fog affecting the whole of the London area and lasted from the morning of 5 December until the evening of 9 December, a total of five days.[1] The areas worst affected were the eastern part of the Thames Basin, the London Administrative Area (the London County Council), and that part of Essex adjoining the London area. In parts of Central London visibility was reduced to 10–12 yards (9–11 metres) during the day of 7 December and was nil during that night. The cause of this exceptionally long lasting and toxic fog was an unusual combination of atmospheric conditions, with a lack of wind and a layer of cold air overlaid by warm air.

Wilkins[2] estimated that there was an excess of 4000 deaths during the fog and the following two weeks. A further 8000 deaths occurred during the next 10 weeks; he also noted that the level of pollution both before and after the fog was higher than in the corresponding period in 1951 which was free of fog. Deaths were mainly due to chronic bronchitis and emphysema exacerbated by chemical irritants (sulphur dioxide and possibly sulphur trioxide) and particulate matter (smoke).[1]

METHODS

Initially I examined the discharge diagnoses for intussusception at Great Ormond Street for the year 1952, extending this to 1951, 1953, and 1954 (there were no data available for 1950), as controls. I repeated this for the Queen Elizabeth Hospital for Children in East London. In each case I recorded the month of admission and the postal district of the home address. For 1952 the actual date of admission was recorded. The catchment area of the two hospitals coincided with the part of London worst affected by the fog.

RESULTS

In the 1950s Great Ormond Street had 270 medical and surgical beds, and the Queen Elizabeth Hospital had 160 beds, making a total of 430 beds. At that time the general hospitals in London had small paediatric units, with less than 20 beds. Data for the smaller children's hospitals in London are not available, and most of them are now closed; they were situated mainly in areas relatively little affected by the fog. It seems unlikely that the paediatric units in the general hospitals or the small children's hospital would have admitted a significant number of intussusceptions during the period under review.

During the years 1951–54 a total of 87 cases of intussusception were admitted to both hospitals. Eighty seven per cent (77) came from the London postal area and it is only these which are considered here. All 25 admissions to Hackney and 76% (55) of the cases admitted to Great Ormond Street came from the London postal area.

The annual admissions to both hospitals were: 1951, 10 cases; 1952, 36 cases; 1953, 20 cases; and 1954, 11 cases (fig 1), giving an annual incidence per 1000 live births[3] of 0.2 for 1951, 0.7 for 1952, 0.4 for 1953, and 0.2 for 1954. Admissions for 1952 were two and a half times the mean for the years 1951, 1953, and 1954. Both hospitals showed an excess of cases for 1952.
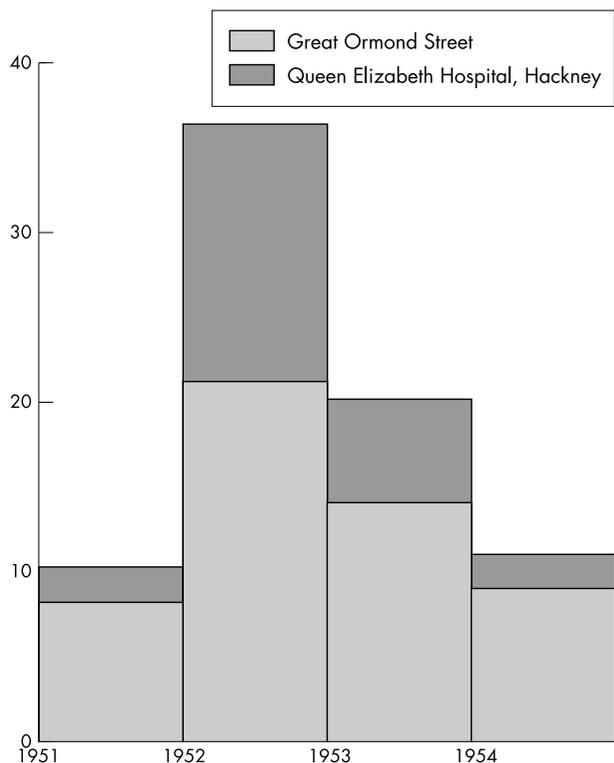
Figure 1 Annual admissions of cases of intussusception to Great Ormond Street and Queen Elizabeth Hospital.

Taking the monthly figures for admissions (fig 2) there was evidence of an increasing incidence at the end of 1951 continuing during 1952, and a tailing off during the beginning of 1953. There were three clusters of admissions, with five cases in August, November, and December 1952; there were no clusters in the other years. In August the admissions were evenly distributed through the month. In November there were three admissions over a period of three days. December was exceptional, with one admission on the first day of the fog, two cases on the last day, and two cases on the day after the fog had cleared, making five cases in five days. There were no deaths from intussusception in either hospital during the period 1951–54.

DISCUSSION

Three aspects of the findings described above require discussion: the clustering of cases during and immediately after the fog; the possible aetiology of intussusception during the period under review; and the high incidence of intussusception in both hospitals in 1952 compared to the preceding and following years.

Logan,[4] in his investigation of London fogs causing a high mortality, found that severe fogs had occurred in the winters of 1873, 1880, 1882, and 1892. After 1892 there was a gap of 50 years, probably due to a reduction in domestic coal consumption after the introduction of gas for heating and lighting, and later of electricity. In the winters of 1948, 1952, and 1956 severe fogs were again experienced in London, the most likely cause being exhaust fumes from an increase in motor vehicles in the years immediately after the end of the 1939–45 war. The fog of 1948 caused an excess of 300 deaths,[4] while that in 1956 was responsible for an excess of 1000 deaths.[5] In both episodes deaths were attributed to pneumonia and bronchitis in older patients with pre-existing conditions such as emphysema and chronic bronchitis; in 1948 and 1956 the peak of deaths occurred during the fog but as many deaths occurred during the week after the end of the fogs. Since 1956 there have been no severe fogs; this can be attributed to the Clean Air Act of July 1956.

The fog of December 1952 was different: the atmospheric pollution was much more severe, and the concentrations of sulphur dioxide and particulate matter were 10–15 times that for the corresponding period in 1951, which was free of fog.[1] Sulphur dioxide, when in contact with water droplets, becomes sulphurous acid; it is likely that smaller amounts of sulphuric acid were also formed from sulphur trioxide, but this was not measured. Both these acids are highly irritant to the respiratory and gastrointestinal tracts. Confirming the irritant effect on the respiratory tract, Lennox[1] found inflammation of the larger bronchi and shedding of the bronchial epithelium in eight adults who died during the fog.

A unique feature of the 1952 fog was the large number of sudden deaths at home during the actual fog, emphasising its extreme toxicity.[2] During the fog many adults experienced a sulphurous taste in the mouth and an increase in nasal secretion, and it is probable that the epithelium of the terminal ileum of infants with intussusception could have been damaged by swallowed saliva and nasopharyngeal secretions containing irritant substances. The delay of 1–2 hours in the terminal ileum before its contents pass through the ileo-caecal sphincter[6] would accentuate damage and facilitate the passage through the epithelium of infective material already present in the ileum, causing the swelling of the lymphoid follicles (Peyer's patches) in the wall of the ileum, which is the initiating cause of an intussusception. This may explain the clustering of five cases during and after the fog, within a period of five days.

The aetiology of intussusception is still under discussion. During the 1960s and 1970s a number of studies[7–9] found evidence that the adenoviruses were the responsible agents (the rotavirus was not identified until 1973). In Potter's
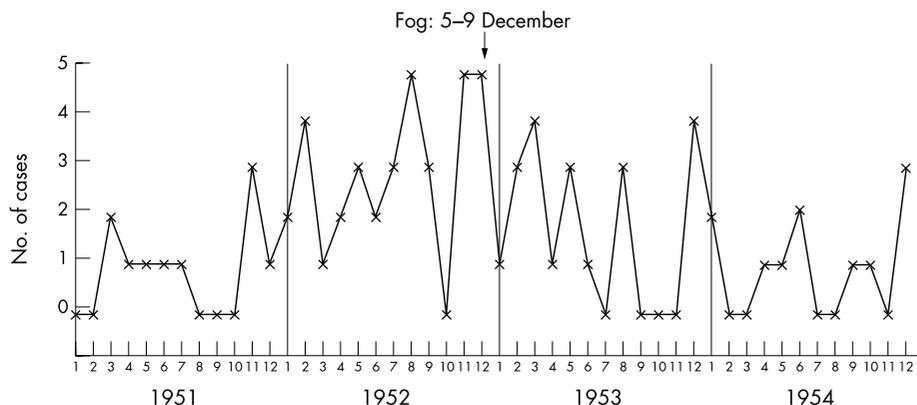

Figure 2 Monthly admissions for intussusception for the years 1951–54.

series[7] in Sheffield, the peak incidence for intussusception occurred in the summer, coinciding with the height of upper respiratory infections due to the adenovirus (it has since been shown that it is the respiratory adenoviruses, and not the gastrointestinal ones, which are associated with intussusception[10]).

However, in 1978, Konno and colleagues,[11] in Japan, described an association between rotavirus infection in 37% of 30 cases and adenoviruses in 27%. They found that the peak incidence of intussusception occurred at the height of rotavirus gastroenteritis, in the cooler months of the year. Their findings were extended and confirmed by Katsushima[12] in 1981. Konno et al suggested that both the rotaviruses and the adenoviruses could be responsible for intussusception. Two later studies[13 14] in France and Australia, found no evidence of an association between rotaviruses and intussusception; in the French series adenoviruses were the main aetiological agent, but without a seasonal peak.

In 1998 the emphasis shifted. The introduction in 1998 of a live oral rotavirus vaccine against infantile gastroenteritis resulted in a total of 102 confirmed or presumptive cases of intussusception; 57 of the conditions developed within seven days of vaccination.[15] As a result of these findings the vaccine was withdrawn in 1999. It was later shown however that the incidence of intussusception among recipients of the vaccine did not exceed that in New York State for the years 1991–97.[15] It was suggested[16] that the virus in the vaccine did not directly cause an intussusception, but rather that a large bolus of live virus (perhaps any large bolus of virus) in the ileum might facilitate the passage of another virus already present in the lumen, in the same way as has been suggested for chemical irritants (see discussion on the fog of 1952 above).

In the absence of viral studies in 1952 it is impossible to give a clear answer to the aetiology of intussusception in London during the early 1950s. However, in temperate climates infantile gastroenteritis is now a winter disease, due mainly to the rotaviruses.[17] In another study, coincidentally in the same two hospitals involved in the present survey, the incidence curves for rotavirus infections for the years 1986–87 and 1989–90[18] were similar to that for intussusception in 1952.

It seems likely therefore that the cases of intussusception during the autumn and winter of 1952, and probably of 1951, 1953, and 1954, were due to rotaviruses. Adenoviruses cannot be ruled out as a causative agent, however during spring and summer; in these years Strang[19] found peaks of incidence in winter and spring, which would confirm Konno's suggestion that both groups of viruses may cause intussusception: rotaviruses in the winter months and adenoviruses in the spring and summer.

The high incidence of intussusception during 1952 compared to the preceding year and the following two years probably reflects the large year to year changes in incidence commented on by Strang in Glasgow[19] and Court and Knox[20] in Newcastle, and suggests an increased prevalence of the causative viruses during 1952. The mean annual incidence of 0.3 per 1000 live births[3] in this study is much lower than that in Birmingham (1.49) and in Newcastle (4.3).[20] Both these investigations included the years 1951–52. However, the low incidence in London is compatible with the figures of <0.4–4.0.[21] The difference between London and those in Birmingham and Newcastle raises the interesting possibility that the incidence of intussusception is related to the general level of health of the infant population as indicated by the Infant Mortality Rate. During the years 1951–54 the Mean Infant Mortality Rate for London was 23, compared to 26 in Birmingham and 27 in Newcastle,[3] a significant difference, which supports this idea.

Conclusion

There is no doubt that the fog of December 1952 was an exceptional event in terms of its toxicity and the resulting morbidity and mortality. The clustering of five cases of intussusception during and immediately after the fog suggests that swallowed toxic chemicals could have facilitated the passage of viral pathogens through the ileal wall, causing swelling of the Peyer's patches, resulting in an intussusception. It is suggested that rotaviruses may have caused the cases during December 1952, and also during the winters of 1951, 1953, and 1954. During the spring and summer of these years adenoviruses may have been the responsible agents. The high incidence of cases during 1952 as a whole may reflect an increased prevalence of a virus or viruses responsible for intussusception.

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