AN OUTBREAK OF SMALLPOX IN AN URBAN AREA

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Walmer is a complex municipality with a total population of 25,606 persons of whom 5,644 are Africans. Of this number 3,200 reside in 365 houses in a location covering 0.22 sq. miles, on the southern municipal boundary.

To this densely populated and congested area came a family from Nyasaland, and with them an outbreak of smallpox, which within a period of 21 days resulted in 46 proven cases. Forty-four were admitted to the emergency hospital, one (Dorah S.) was isolated and treated in Uitenhage, and one (Harry N.) isolated at home, since he showed no symptoms (described in detail later in this article). Eight proved fatal. Fig. 1 indicates the position of



Fig. 1. See text.

the 8 houses to which the outbreak was confined in Walmer, as also the emergency hospital and the 'Victory Hall' where the staff was accommodated.

Ben E., an African from Nyasaland, was returned from Walmer to his homeland towards the end of 1962, together with his wife Angelina and their 6 children. Ben deserted his family and they remained in Nyasaland until they were repatriated to the Republic of South Africa on 2 February 1964. It is reported that Assam, aged 15 years, received treatment for a fever and rash of unknown origin just before leaving Nyasaland. The family arrived in Walmer on 7 February 1964, and went to live with a family at 118 Camando Street. There they resided for a short while before moving to 60 January Street (also in Walmer location).

EPIDEMIOLOGY

On 11 February, Lydia aged 13 yrs. and her brother, Suluke, aged 11 yrs. attended the daily session at the Walmer Health Centre. Their clinical picture was typical of chicken-pox. Temperatures were normal. There were very scattered lesions on the face and arms, a few on the trunk and legs, and 1 or 2 lesions on the palms of the hands. These lesions were in various stages of eruptionpapular, vesicular and pustular. A number were beginning to dry out and form scabs. The presentation in these 2 patients was consistent with chicken-pox. Since they had recently returned from a smallpox endemic area, the findings were reported to the Walmer Medical Officer of Health, who decided to consult with a dermatologist. Assam E. was found to have scars on his face, but without pitting. The mother, Angelina, Mercia (7 years) and the 41-year-old twins, Joseph and Josephine, were quite clear of any lesions or scars, and were in good health. Angelina was the only member of the family who had vaccination scars. Except for the patients, all the members of the family, and known contacts, were then vaccinated. The dermatologist took scrapings from the base of the pustular and vesicular lesions of Lydia and Suluke, and submitted these to the SAIMR for an opinion. It was decided to place the family under house quarantine, pending the outcome of the laboratory findings, since the individual lesions appeared slightly larger and rounder than those normally associated with chicken-pox and since doubt regarding their origin arose and they were coupled with palm lesions. On the next day (12 February) it was reported that Tzanck cells were seen in the scrapings, indicating varicella.

Owing to the inconsistent cropping of the rash, the lack of fever, the laboratory findings, coupled with an epidemic of chicken-pox in the community, smallpox was considered, but eventually discounted. The consultant dermatologist confirmed the diagnosis of chicken-pox.

On 16 February the mother observed that Lydia's rash had become more profuse. Lydia and Suluke were then taken to Livingstone Hospital. Smallpox was suspected by the doctor in attendance, and the MOH was contacted. It was decided to return these 2 suspects to their home for treatment. On 28 February a private practitioner was called to 119 Camando Street to see Joseph T., an African male adult, who was suffering from bronchospasm, coupled with a pyrexia of 104°F. No rash was present. The patient was treated with a broad-spectrum antibiotic since the origin of the pyrexia was unknown. Two days later (1 March) the private practitioner again attended this patient, and found that he had developed a rash on the previous day. With the appearance of the rash, the temperature had abated. The rash at that stage was regarded as an allergic reaction, and this was attributed to the antibiotic used. On the same day the general practitioner attended Mzolise M. at 37 Bath Street. Not only were the lesions very similar but so was the prodromal picture. He had been ill for 3 days with hyperpyrexia, which abated on appearance of the rash. On 2 March the practitioner decided to refer both Joseph and Mzolise to Livingstone Hospital to consult with a dermatologist. The dermatologist was not in attendance, and the 2 patients remained in the outpatient department until the evening when Joseph decided to walk the 8 miles from the hospital to his home through the densely populated parts of Port Elizabeth.

On Tuesday 3 March, the same private practitioner was called to 118 Camando Street to attend Henry N. He was confronted by the same rash and prodromal history. He reserved his diagnosis until the following day, when he notified the Walmer MOH of the 3 cases which he now suspected as being smallpox. A dermatologist was immediately called in, and both he and the MOH confirmed the diagnosis. The Nyasaland family was revisited at 60 January Street, where they now lived with the M. family; and although none of them appeared to be ill, the following picture was suspicious:

Assam E. — very scattered scabs, face, legs, arms and palms.

Suluke — scattered scabs on face, limbs, and scars on palms.

Lydia — very scattered scabs on face, limbs and palms.

Joseph — very scattered scabs on face and limbs. No palm lesions.

Josephine — very scattered scabs on face and limbs. No palm lesions.

Mercia — very scattered scabs on face, limbs, and very few on trunk. No palm lesions.

Smallpox was diagnosed in retrospect, and the E. family became cases 1-6: Joseph, case 7; Henry N., case 8, and Mzolise M., case 9. On 5 March the MOH was called in to attend Goodwin, residing at 47 Witbooi Street, also in Walmer location. He had a temperature of $104^{\circ}F$ and a profuse rash on the face, trunk, limbs, soles of his feet, and palms of his hands. The lesions were in papular and vesicular stages on the face and arms, and papular on the rest of the body. He was diagnosed as smallpox case 10.

Emergency Measures

With a minimum of delay, an emergency hospital was established in the Walmer location, and cases 1-10 were moved in on 6 March. Very strict house quarantine arrangements of all direct contacts were enforced under guard. The fact that all 10 patients lived in the densely populated location area, 5 of them attending school and 2 of them having attended a crowded outpatient session of a major hospital, presented a very grave threat.

An intensive vaccination campaign was set in motion. A daily medical examination was made of all contacts residing in the then 6 quarantined houses. Any pyrexia within these quarantined houses was treated as smallpox; the patient being removed to 'suspect' wards, and kept under observation. In all, 12 suspects were removed, and all proved positive for smallpox within a period of 3 days.

On 7 March a third school contact of Lydia and Suluke E. was admitted as case 11. She resided at D40 Topia Street and was very ill on admission having a 4th day confluent rash covering the whole body. The 7th patient from 60 January Street, Nontotozele M. (case 12) was admitted to the isolation hospital on the same day.

On 8 March the first case outside Walmer was notified by the MOH for Uitenhage, which is 22 miles from Walmer. This patient, Dora, was isolated at Uitenhage, and was reported to be a contact of the Nyasaland family. At this stage 6 houses were under strict quarantine and daily medical surveillance. The secondary outbreak was expected from more or less 10 March. Then it was learned that a contact from 118 Camando Street was in gaol. The Senior District Surgeon traced this contact, one Harry N. and had him returned home on 7 March. Apart from badly infected scabies on his trunk, he appeared to be well, with no history of illness or lesions on his face, feet or hands. The senior district surgeon immediately had the 1,500 inmates of the gaol vaccinated, and observed any report of illness in this confined space.

The secondary outbreak started on 11 March with a further 3 members of the M. family being admitted from 37 Bath Street, becoming cases 13, 14 and 15. They were all contacts of case 9, as were cases 17 and 18, who were admitted from the same address on 12 March. The eighth case from 60 January Street, Elizabeth M. (case 16) was also admitted on 12 March. She was followed by cases 19 and 21 from the same address, admitted on 13 and 14 March respectively. Cases 20 and 22 were also admitted on 14 March from 119 Camando Street, and were contacts of case 7. On 16 March case 23 was admitted from the quarantined house, 118 Camando Street.

Outpatient and Gaol Contacts

The first hospital outpatient contact was admitted on 17 March from a farm in the Humansdorp district, approximately 50 miles from Walmer. This case, James (case 24) was admitted as a typical variola major, with a profuse vesicular and pustular rash over the whole body. He was discharged from the Livingstone Hospital on 2 March and awaited transport in the casualty department while cases 7 and 9 were there. The contact picture became more clouded when it was learned that he had travelled to his home by public transport after spending a week in hospital. The second contact from Livingstone Hospital, patient Jeremiah (case 25) was admitted the following day (18 March). Although he was employed at the hospital, it was learned that he too had attended the outpatient session with cases 7 and 9, as did case 35 who was admitted on 22 March.

On 18 March case 26 was admitted to hospital as the first of the gaol contacts. Harry N., the contact who had been removed from gaol and who had been kept under constant medical observation, was still well. His scabies rash had responded well to normal treatment, and he had developed no lesions which could be identified as smallpox. Blood submitted for compliment fixation returned a positive result, and the first of the smallpox cases without illness was recorded. Although this person is believed to have been in gaol for only 2 days, he was responsible for the infection of 13 patients (cases 26 - 31 and 36 - 42) being admitted to hospital, through their contact with him. A number of these cases were admitted from farms radiating as far afield as 70 miles from Walmer.

The potential of the outbreak at this stage looked very grave, and great.

The vaccination campaigns held throughout the day, and part of the night, by State Health and the various local authorities had been very successful, and it was thought that the benefit would become apparent from 20 March onwards. It says much for the team work of the various health authorities that, other than the Livingstone Hospital contact cases and the gaol contact cases, all 44 patients admitted in this outbreak were removed from the 8 quarantined houses plus one other. This other house was 126 Wesleyan Street, Walmer location, and the patient was Gladys (case 40). She was a close contact of Harry N., who was also responsible for the gaol outbreak although he himself never became ill.

The last case to be admitted in this outbreak was from the Uitenhage quarantined house on the 20th day of quarantine. She was Lizzie (case 44), a contact of Dora, and was a typical haemorrhagic type 2 case. Cases encountered during the outbreak were classified into 3 main categories, i.e. classical, modified or haemorrhagic. An example of each group follows, commencing with the classical or typical case.

Case 15

CLASSICAL SMALLPOX

Cynthia M.'s illness started with a temperature of 103°F and a very red, sore throat, which was maintained until the 3rd day, when a fine papular rash appeared on the face and forearms. Her temperature was then 104°F, and there were no palm lesions. On the 4th day of illness, the second-day rash had become more profuse on the face and forearms, and had spread over the body, with the back more involved than the abdomen, and the axillae and lower abdomen relatively free of lesions. The lesions were of different sizes, and now there were definite palm lesions. During the 3rd day of the rash, which had become more profuse, the temperature dropped to 100°F. The palm lesions were now well marked. It is an interesting observation that the rash was in different stages of development, varying from papules to vesicles, with 1 or 2 scabs on the face. The patient's general condition was improved. On the fourth day of rash, the number of vesicles increased, but the rash remained in different stages of development, with lesions on the trunk and legs mostly in the papular stage. The temperature was 101°F. The 5th day of the rash saw the number of vesicles on the trunk and legs increase, while the facial lesions became cloudy. The temperature rose to 104°F. On the 6th day the patient was very ill, with marked facial oedema. There were lesions in the mouth and throat, and the patient had difficulty in swallowing. The facial lesions were now all pustular, while those on the legs were in papular, vesicular and pustular stages. The patient's temperature remained constant at 104°F. The 7th day of rash saw most of the lesions become pustular. Facial oedema was still marked, while the patient experienced less difficulty in swallowing. The temperature dropped to 101°F. There was less facial oedema on the 8th day of the rash, and the patient was able to take fluids and soft solids without difficulty. Her temperature was 100°F and her general condition con-

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tinued to improve. The 12th day after onset, and the 9th day of the 1ash, can be described as the turning point in the illness. The temperature returned to normal, the general condition was much better, and while the facial oedema was much improved the lesions started to dry out. The lesions on the arms, trunks and legs were still in the pustular stage. Those on the legs were umbilicated. From now on the temperature remained normal, and the drying out and scabbing progressed in the order in which the rash had appeared. The pustules on the palms of the hands and the soles of the feet were the slowest to dry out. By the 18th day of illness, and the 15th day of rash, the scabbing of lesions was complete. Deep-seated scabs persisted for a long time on the palms and soles, and were

MODIFIED SMALLPOX

only completely separated approximately 6 weeks from

Case 32

onset.

Agnes, who was vaccinated as a child, was revaccinated at the onset of the outbreak but it did not 'take'. The onset of illness was sudden, commencing with a temperature of 103°F, backache, headache, a sore throat and rigors. This condition lasted for 2 days. On the 3rd day of illness, scattered papules appeared on the face and forearms. With the appearance of lesions, the temperature subsided to normal, and the patient's general condition was vastly improved. The temperature remained normal, and by the 6th day the lesions had all become vesicular. There was no further development of the lesions, and drying out commenced.

In all, there were 20 patients with modified smallpox, all of whom developed only a few scattered lesions, which in most cases did not go through a deep pustular stage, with many drying out to form scabs after the vesicular stage. There appeared to be a definite relationship between vaccination and this modified version of smallpox. With the appearance of the rash, the temperature returned to normal, and there was no secondary elevation of temperature, as with the classical cases. Two of the modified cases had no prodromal symptoms, commencing immediately with a rash. They were both vaccinated children, and the prodromal symptoms might easily have been missed.

HAEMORRHAGIC SMALLPOX

There were 2 types of haemorrhagic smallpox cases, 3 cases of type 1 and 6 cases of type 2. All the deaths in this outbreak occurred among this group. The 3 type 1 cases, and 5 of the 6 type 2 cases, died.

Type 1 (Case 10)

Portia was ill for 2 days with a high temperature. On the 3rd day she developed a fine rash which resembled measles. With the appearance of this rash, the temperature abated. On the 5th day the rash changed in appearance and was now a confluent fine papular rash all over the body. The patient was very lethargic, with a temperature of 98°F. Vesicles did not develop, and there was no further maturation of the rash. On the 9th day this patient developed large haemorrhagic blisters on her feet and hands. Her temperature remained subnormal, and she lapsed into a coma, and died on the 10th day of illness. There were 3 of these type 1 cases which were haemorrhagic from the start. All proved fatal. Two cases had haematuria and bleeding from pressure points in the skin. These symptoms were apparent early in the illness.

Type 2

Up to the pustular stage, the course of the illness was similar to that of the classical cases. The pustules, however, became confluent and filled up with blood. Three of these 6 patients developed haematuria, and all 6 were haemorrhagic to the point of free bleeding on being touched, and at pressure points. As in the case of the type 1 haemorrhagic cases, there was no improvement in the general condition of the patients. They were all very ill from the beginning and except for the 1 who recovered, remained so until they lapsed into a coma and died. Temperatures in these cases appear to have no significance, being slightly above or below normal without any consistency.

In the patient who recovered, the haemorrhagic lesions, on drying, separated from the body in large strips, leaving raw areas especially on his feet.

NOT CLASSIFIED

There were 4 cases which cannot be classified since they were not typical of any of the groups described. Two of these, cases 13 and 39, began with a high temperature and had typical prodromal symptoms. The temperature abated on the 3rd day but no rash appeared. Their condition continued to improve, and they were apparently in normal health on the 5th day. Blood taken from these 2 patients and submitted for compliment fixation returned a positive result in one (case 13). The other 2 cases were even less typical of smallpox. The one, Achronette T. (case 20), is the wife of case 7 and the mother of case 22. She complained of headache on the day of admission but had no temperature or any of the other prodromal symptoms, nor did any develop. Her blood, submitted for complement fixation, returned a positive result. The 4th and most alarming of the unclassified cases, Harry N., has been mentioned in the body of this report as being responsible for 14 proven cases while developing no symptoms himself. As in the case of the others the complement fixation test proved positive.

OBSERVATIONS

The most important observation of this outbreak is, without a doubt, the value of vaccination, not only as a prophylactic but as an abortive in the course of the illness itself. This was amply demonstrated in the majority of the modified cases and the following are examples:

Value of Vaccination

Margaret, aged 5 years (case 33), and Christopher, aged 2 years (case 34), are brother and sister and were vaccinated for the 1st time at the start of the outbreak. Margaret had 'no take' and Christopher had 3 good 'takes'. They both developed symptoms on the same day. Two days later both developed a rash. Margaret developed a profuse rash and was a severe classical case of smallpox. Christopher, however, was a mild modified case, with few scattered lesions confined to the face. Rosemary M. (case 14) and Lena M. (case 13) were vaccinated on 4 March. On 11 March they both became ill. Lena had a 3-day high temperature, headache, back-ache, sore throat and rigors. On 14 March, 10 days after vaccination, when her antibody level was presumed to be at its highest, the course of the illness was aborted. No rash developed and she improved to normal health. Rosemary had a 1-day prodromal illness, and then developed a fine papular rash. On 14 March, with antibody level at its peak, the papular rash disappeared and she was left with only 3 vesicular lesions on the face. The remaining members of this M. family, Cynthia (case 15), Gwen (case 17) and Muriel (case 18) had no previous vaccination scars or 'takes' when vaccinated on 4 March and all developed severe classical smallpox.

As will be apparent from this report it is extremely difficult, if not impossible, initially to distinguish between severe chicken-pox and a mild attack of smallpox. With the development of the illness, a differential diagnosis can be made but 4 or 5 days will have elapsed. This presents a major problem as regards isolation. It became obvious in this outbreak that the prodromal period must be highly infectious, and that the spread during this period is, in all probability, airborne. Approximately 80% of all cases had very red, sore throats.

The period of rash can be described as contagious rather than infectious. It is interesting to note that a number of patients with chicken-pox were (before observation facilities became available) admitted to the smallpox wards as 'suspects'. Although in close contact with proved cases in various stages of lesions, not one of these patients developed smallpox. In the event of smallpox being infectious during this period of rash, the possibility should be considered that these persons suffering from chicken-pox, a virus infection, had a virus immunity during this period. In several of the cases treated during this outbreak, the incubation period exceeded 14 days, and in 1 case, that of Lizzie (case 44), it was as long as 20 days. Therefore, quarantine measures should be taken for at least 21 days.

In the Enos children, none were previously vaccinated except the mother. Three were vaccinated in February with none 'taking'. All developed modified rash except the mother who remained healthy.

Scabs from these children showed negative complement fixation. Revaccination after their discharge from hospital showed immune reactions. Mild disease in the Enos children resulted in variola major in subsequent unvaccinated contacts, and a modified smallpox in vaccinated contacts.

Another interesting observation arising from this outbreak is the lack of reliable laboratory tests to prove smallpox. Cases 8-12 returned negative complement fixation results and yet the variola virus was isolated from vesicle fluid in each case. If the complement fixation negative result cannot be relied upon then there appears to be no way of detecting smallpox in the unclassified cases which develop no lesions. Until a satisfactory laboratory method of confirmation is discovered the clinical diagnosis must continue to play the major role.