MEASLES OUTBREAK IN A TRIBAL POPULATION OF THANE DISTRICT, MAHARASHTRA

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ABSTRACT

In March 1992, an outbreak of measles, in the tribal population of Vavar village, Mokhada Taluk, Thane district, Maharashtra, was investigated. Two hamlets of Vavar village namely Sagpanipada (epidemic in October, November 199]) and Behedpada (epidemic in January, February 1992) were affected. In both hamlets, measles cases were confined to children below 10 yrs and 96% of the cases occurred in children below 6 yrs. Attack rates were 52.7% and 51.4% and case fatality rates were 31.2% and 15.6% at Sagpanipada and Behedpada, respectively.

All the convalescent patients' sera possessed IgM antibodies against measles. A clear drop in IgM and a rise in IgG antibodies against measles was observed in 35 paired samples from convalescent patients. Fifty four per cent of sera from controls, possessed IgM antibodies.

Migrating population appeared to have imported measles which flared up in an epidemic among the susceptible. Priority immunization of the children of remote isolated populations may prevent such epidemics.

Key words: *Measles outbreak, Tribals.*

Measles is endemic in urban parts of India. Epidemics of measles have occurred in rural areas and in isolated remote populations(l-4). In March 1992, an outbreak of measles occurred in the tribal population of Vavar village, Mokhada Taluk, Thane district, Maharashtra. We investigated this outbreak on being requested by the Department of Health, Government of Maharashtra. This report presents our observations on the epidemic, which has occurred during the present era, when world wide efforts are being made to overpower measles to the point of eradication(5).

Material and Methods

Vavar village, situated at the south western end of the north Sahyadri mountain ranges is in the Mokhada taluka of Thane district, Maharashtra *(Fig. 1)*. The village is comprised of 9 hamlets, locally called 'padas', separated from each other by a distance of 3-5 km. The hamlets are isolated, lack electricity and medical facilities. One had to walk 8 km to avail the nearest bus facility, travel 9 km to visit the nearest market at Ozar and transport a patient 20 km to the nearest Primary Health Centre (PHC) at Sakharshet to seek medical help.

The present outbreak of measles occurred in two hamlets of the Vavar village: Sagpanipada, situated on the top of the hill and Behedpada, situated 3 km away at the foot of the hill. Both these hamlets were

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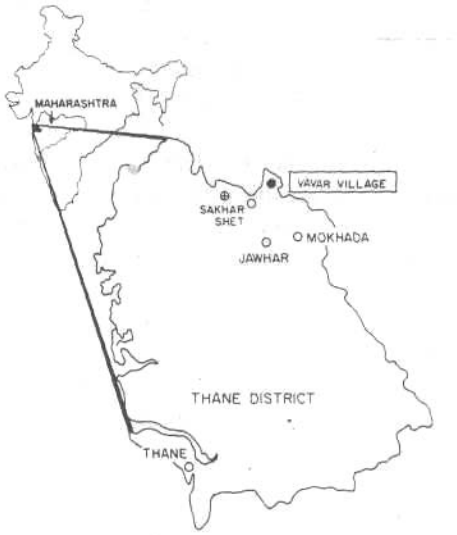

Fig. 1. Location of Vavar village, where measles outbreak occurred.

inhabited by intermingling tribal population.

Enquiries made at PHC Sakharshet revealed that one vaccinator from the PHC used to visit these hamlets for immunizing children with polio, triple and measles vaccines. The vaccinator had to carry the vaccine on foot to these hamlets as no transport facility was available. At the PHC no records of immunization at these hamlets were available.

Locally no qualified medical practitioner was available. However, there was one person locally, who used to treat some ailments with herbs and roots. The villagers preferred him to a Doctor as he did not take any remuneration in cash, instead accepted food/drink or even fowl which was easier for the villagers to pay. He did not treat patients too long and often referred them to PHC.

The team visited all the households in both the hamlets, enumerated the population, examined the patients and noted the details about their illness. In addition, the available children at Behedpada were weighed and their height and mid arm circumferences were noted to assess their nutritional status.

To serologically confirm the epidemic, 8 single convalescent sera from patients of Sagpanipada, 35 paired convalescent and 8 single convalescent samples from patients of Behedpada were collected. There was an interval of 2 months between the paired convalescent samples.

To serve as controls, 7 single serum samples from normal subjects at Sagpanipada and 6 paired sera and 20 single serum samples from normal subjects at Behedpada were collected. These normal subjects belonged to similar age groups and had not suffered an attack of measles during the epidemic.

All serum samples were tested for the presence of IgM and IgG antibodies to measles virus by indirect ELISA(6,7). Briefly the serum samples were diluted 1:100 (in phosphate buffered saline pH 7.2 supplemented with 1% bovine serum albumin). Then the samples were added to Nunc polystyrene plates, coated with purified measles antigen. After appropriate washing steps, goat antihuman IgM (Sigma Chemical Co., 1:1000 dil.) was added for detection of IgM antibodies and goat antihuman IgG (Sigma Chemical Co., 1:1000 dil.) was added for detection of IgG antibodies. This was followed by washing steps and addition of substrate (H_2O_2) and the Chromogen (orthophenylene diamine in phosphate citrate buffer pH 5). After the development of the color, the reaction was stopped with 4N

H_2SO_4 and the wells were read in a Dynatech ELISA reader at 492 nm. Appropriate strong positive, weak positive and negative controls for measles IgM and TgG antibodies were run in each test. Measles IgM and IgG ELISA carried out by us, were standardized by using a commercial kit (Pharmacia) and second international standard antimeasles serum obtained from Staten Serum Institute, Copenhagan, respectively.

A serum giving an optical density (OD) of 0.7 or more in the antimeasles IgM ELISA corresponding to a titre of 1:50 was considered positive for IgM antibodies to measles(8) and a serum giving an OD of 0.6 or more in the IgG ELISA corresponding to a titre of 1:100 was considered positive for IgG antibodies to measles(9).

Chi-square test was used for making comparisons between the males and females, and between the two hamlets Sagpanipada and Behedpada. Student 't' test was used for comparisons of anthropological measurements.

Results

Epidemiological Features

By mid March 1992, when we actually carried out investigations, the outbreak of measles in the two hamlets of Vavar village had waned. The records at PHC, Sakharshet, showed that the outbreak at Behedpada had commenced in the last week of January 1992, reached a peak in the third week of February and ended abruptly in the last week of February. No such information was available for the outbreak at Sagpanipada.

Sagpanipada had 44 households with a population of 270; 176 (65%) were adults and 94 (35%) were children. A total of 48 measles cases occurred and 15 died. In 103 household of Behedpada, 593 people resided, comprising 325 (55%) adults and 268 (45%) children. Among them 128 cases of measles occurred and 20 died.

The age specific measles morbidity at Sagpanipada and Behedpada is presented in *Table I.* In both the hamlets the measles cases were confined to children below 10 years and 96% of the cases occurred in children below 6 years. The overall attack rate of measles for children was 52.7% at Sagpanipada and 51.4% at Behedpada. Infants 9 months and younger had lower attack rate at both the hamlets.

The age specific measles mortality at both the hamlets is presented in *Table II.* The overall case fatality rate was 31.2% at Sagpanipada which differed significantly from 15.6% at Behedpada, ($p<0.05$, $\chi^2 = 4.414$, df=l). Three infants from each of the two hamlets, below nine months, contracted measles and one child expired at each place. At Sagpanipada the high case fatality rate (25-50%) was noticed in all age groups below 5 yrs (60 months), whereas at Behedpada the high case fatality rate of 30% and above was confined to children 2 yrs (24 months) and younger. However, in both the hamlets, majority of deaths occurred in children below 4 years.

The attack rates and case fatality rates for male children did not differ significantly from those for female children in both the hamlets ($p>0.05$).

The precise cause of death in fatal cases could not be ascertained; however, based on the history given by the parents of the survivors, 63% among them had suffered from diarrhea, and or dysentery and 60% from respiratory complications.

Anthropometric measurements of chil-

TABLE I—*Age Specific Measles Morbidity at Sagpanipada and Behedpada*

Age (mo)	Sagpanipada			Behedpada		
	Population	Cases (AR)*	Cumulative percentage	Population	Cases (AR)	Cumulative percentage
0-9	10	3 (30.0)	6.2	16	3 (18.7)	2.3
10-12	10	5[a] (50.0)	16.7	24	18[b] (75.0)	16.4
13-24	21	11 (52.3)	39.6	37	30 (81.1)	39.8
25-36	21	13 (61.9)	66.7	35	27 (77.1)	60.9
37-48	11	8 (72.7)	83.3	31	23 (74.2)	78.9
49-60	11	4 (36.4)	91.7	33	12 (36.4)	88.3
61-72	3	2 (66.7)	95.8	19	10 (52.6)	96.1
73-120	4	2 (50.0)	100.0	54	5 (09.3)	100.0
	91	48 (52.7)		249	128 (51.4)	

AR* = Attack rate.
a = 2 cases 10 months old and 3 cases 12 months old.
b = 2 cases 10 months old, 1 case 11 months old and 15 cases 12 months old.

TABLE II—*Age Specific Measles Mortality at Sagpanipada and Behedpada*

Age (mo)	Sagpanipada			Behedpada		
	Cases	Deaths (CFR)*	Cumulative percentage	Cases	Deaths (CFR)	Cumulative percentage
0-9	3	1 (33.3)	6.7	3	1 (33.3)	5.0
10-12	5	2[a] (40.0)	20.0	18	6[b] (33.3)	35.0
13-24	11	4 (36.4)	46.7	30	9 (30.0)	80.0
25-36	13	4 (30.8)	73.3	27	1 (03.7)	85.0
37-48	8	2 (25.0)	86.7	23	2 (08.7)	95.0
49-60	4	2 (50.0)	100.0	12	0	95.0
61-72	2	0		10	1 (10.0)	100.0
73-120	2	0		5	0	
	48	15 (31.2)		128	20 (15.6)	

CFR* = Case totality rate.
a = Both deaths in 12 months old.
b = 1 death in 11 months old and 5 deaths in 12 months old.

ren (1-5 yrs of age) of Behedpada were compared with the standards from the population of Bombay (ICMR Technical Report Series 26, Studies on pre-school children: 1986). The weights of the children in different age groups did not differ significantly than those for the children in the respective age groups of the standard population. However, arm circumference measurements of the 3-5 yrs old children in the study group were significantly lower than those for the children of the standard population.

Serological Findings

Serological results of the IgM tests and IgG on the single and paired sera from convalescing patients and from the control children are presented in *Table III.* All the single convalescent samples from both the hamlets were positive for IgM antibodies against measles. Among the 35 paired convalescent serum samples of the patients from Behedpada, first samples of all the pairs, obtained in the month of March 1992 possessed IgM antibodies at significant OD values. In the second samples collected after an interval of 2 months, IgM antibody levels showed a clear cut drop and in 16 patients OD values had become insignificant *(Fig. 2)* whereas in the first samples of all the 35 paired sera the IgG antibody titres were low and in 16 patients it was below the cut off values. All the second samples of 35 pairs showed an impressive rise in IgG antibody *(Fig. 3).*

As is seen from *Table III,* many of the samples from the control children also were positive for IgM antibody and most of them were positive for IgG antibody. Five out of six paired samples showed significant IgM levels in the first samples and a fall in the second samples. Both the samples of these pairs from the controls were positive for IgG antibody against measles.

Discussion.

In the outbreak of measles described here, all the cases were confined to children below 10 years of age and 96% of the cases occurred in children below 6 years. The overall attack rate was 52.7% at Sagpanipada and 51.4% at Behedpada. These features are common to the measles outbreaks described from the other parts of the country(l,2). However, in an outbreak in a remote isolated population, residing at the Himalayan foot hill, more than 50% of the cases were encountered in children between 5 and 14 years of age(3). The occurrence of this outbreak in the dry season also correlated with some of the rural epidemics described earlier from India and Bangladesh(l,5,10) although outbreaks have been described even in the rainy season by other(2,3,ll).

The overall case fatality rate (CFR) seen in the present outbreak was 31.2% at Sagpanipada, situated at the top of the hill and 15.6% at Behedpada, situated at the foot of the hill. The CFR at Behedpada was comparable with that described in a village of Tamilnadu where health care facility was not available(l).

The CFR seen at Sagpanipada appeared to be the highest for the epidemics reported so far from India. Thus, though the determinants of infection, as reflected by the attack rates appear to be similar, the determinants of mortality seem to differ in the two hamlets. The population pattern, socio-economic status, literacy, health seeking behavior were similar in both the hamlets. Both the hamlets suffered equally from lack of medical facility close by. In the light of this, it is difficult to comment as to why

TABLE III–*Results of ELISA to Detect IgM and IgG Antibodies to Measles*

	Sagpanipada		Behedpada	
	IgM	IgG	IgM	IgG
Patients				
Single convalescent samples	8.8*	4/8	8/8	5/8
Paired convalescent samples				
I sample	-	-	35/35	19/35
II sample (2 months later)	-	-	19/35	35/35
Controls				
Single samples	2/7	7/7	10/20	19/20
Paired samples				
I sample	-	-	5/6	6/6
II sample (2 months later)	-	-	4/6	6/6

* Num = Number positive.
Den = Total number of sera tested.

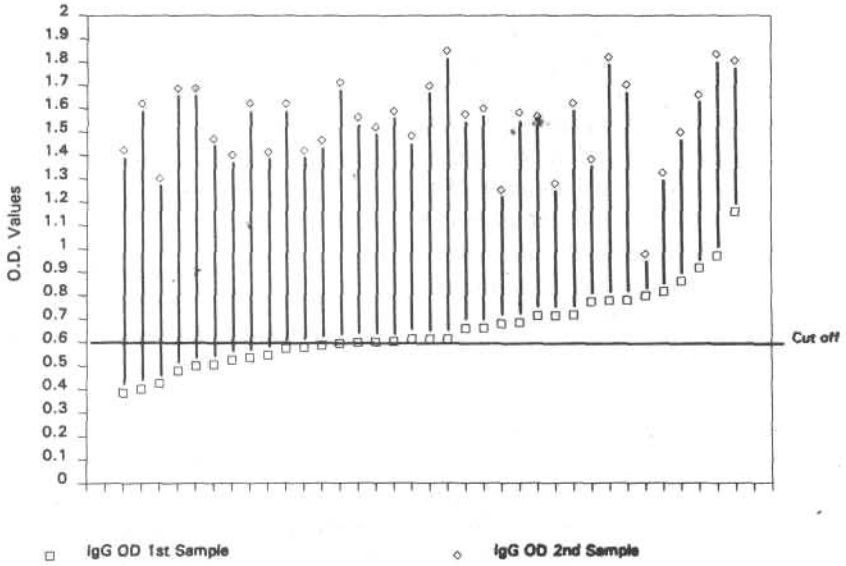

Fig. 2. ELISA OD values for IgM antibodies to measles virus in paired convalescent samples from patients at Behedpada.

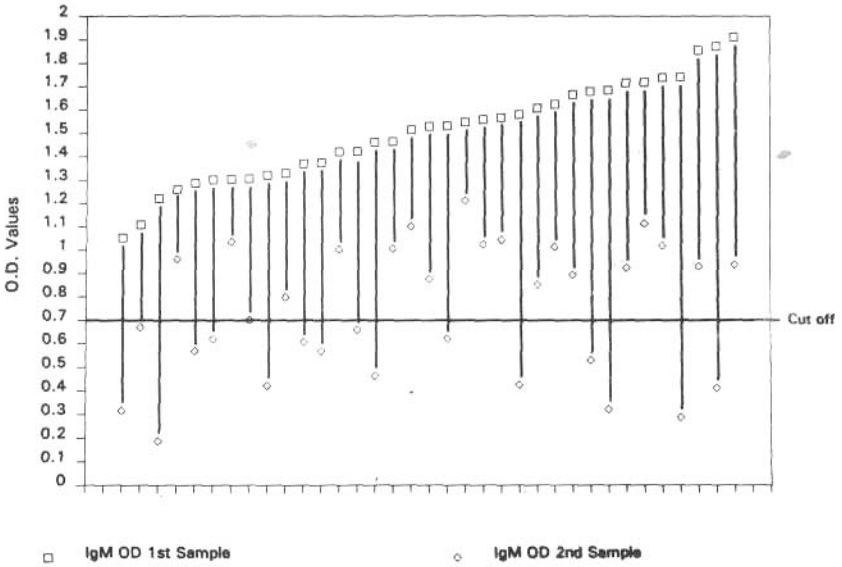

Fig. 3. ELISA OD values for IgG antibodies to measles virus in paired convalescent samples from patients at Behedpada

there was higher mortality at Sagpanipada. The outbreak at Sagpanipada occurred earlier (October/November 1991) whereas at Behedpada it occurred in February 1992 after a gap of three months. Tt is possible that during this period a lot of publicity was generated which resulted in active efforts of Health Services personnel to control the outbreak which might have resulted in better treatment of very sick children at Behedpada preventing the mortality to some extent.

It has been postulated that malnutrition, lack of easy access to medical facility, bad hygiene, poor management of measles patients at home and local beliefs contribute to high mortality due to measles(l,2). All these factors might have been responsible for the high mortality rates observed in the present epidemic.

Due to non cooperation, we could not assess the nutritional status of children at Sagpanipada. The post epidemic anthropological studies carried out by us on the survivors at Behedpada did not suggest severe malnutrition in children as most of the values did not differ significantly from those described for normal Indian children of similar age groups. However, no comment can be made on the nutritional status of the deceased children. It is quite possible that poor nutrition, dehydration and intercurrent respiratory and gastrointestinal infections might have played a role.

All the convalescent sera tested both from Sagpanipada and Behedpada showed the presence of IgM class of antibodies. The paired convalescent sera tested showed a drop in IgM titres. Both these features were suggestive of a recent measles infection.

The indirect ELISA test performed to

detect IgG class of antibodies to measles virus in 35 paired sera clearly showed a rise in antibody titres in all the patients. This observation confirms the earlier report by Lievens *et ah* on the suitability of indirect ELISA test for the diagnosis of measles infection using paired sera(12).

Fifty four per cent of the control children studied by us showed IgM class of antibodies in addition to good titres of IgG antibodies. These findings suggest a possibility of subclinical measles infections among the controls. Earlier studies using HI test have estimated the subclinical infections to the tune of 30%(l).

Discussions with the local population revealed that many families of Vavar village migrate to adjoining urban areas for work during winter and return home around the month of October for Diwali celebrations. This is the time when the outbreak occurred at Sagpanipada. It is possible that this migrating population imported measles from the urban area, which flared up into an epidemic among the susceptible young children in the village. Spread of measles , from urban to rural areas has been described earlier from India and Africa(4,13).

Measles epidemics do not occur in vaccinated villages(l,2). It is unfortunate that the epidemic such as the one described here should have occurred even after seven years of inclusion of measles vaccination in the EPI programme. It seems to reflect the lacunae in the strategy and implementation of our measles control programme.

We feel that for the success of measles control, the population living in isolated villages, far away from the medical facility should receive priority and all the children under five years of age of such population should receive vaccination.

REFERENCES

1. John TJ, Joseph A, George TI, Radhakrishnan Singh RPD, George, K. Epidemiology and Prevention of Measles in rural South India. Indian J Med Res 1980, 72: 153-158.
2. Pereira SM, Benjamin V. Measles in a South Indian community. Trop Geogr Med 1972, 24: 124-129.
3. Narain JP, Khare S, Rana SRS, Banerjee KB. Epidemic measles in an isolated unvaccinated population in India. Int J Epidemiol 1989, 18: 952-958.
4. Salunke SR, Natu M. Epidemiological investigations of measles: An outbreak in Ajiwali. Indian Pediatr, 1977,14: 519-521.
5. Mitchell CD, Balfour HH. Measles control: So near and yet so far. Prog Med Virol 1985, 31: 1-42.
6. Tuokko H, Salmi A. Detection of IgM antibodies to measles virus by Enzyme immunoassay. Medical Microbiol Immu nol 1983, 171: 187-198.
7. Christenson B, Bottiger M. Method for screening Jhe naturally acquired and vaccine intiuced immunity to the measles virus. Biologicals 1990, 18: 207-211.
8. Chen RT, Markowitz LE, Albrecht P, Stewart JA, Mofenson LM, Preblud SR, Orenstein WA. Measles antibody: Re-evaluation of protective titres. J Infect Dis 1990, 162: 1036-1042.
9. Salonen J. Vainionpaa R, Halonen P. Assay of measles virus IgM and IgG class of antibodies by use of peroxidase labelled viral antigens. Arch Virol 91: 93-106.
10. Fauveau V, Chakraborty J, Sarde AMN, Khan MA, Koenig MA. Measles among under-9-months-olds in rural Bangladesh: Its significance for age at immunization. Bull WHO 1991, 69: 67-72.
11. Sinha DP. Measles and malnutrition in a West Bengal village. Trop Geogr Med 1977, 29: 125-134.

12. Lievens AW, Brunnell PA. Specific immunoglobulin M enzyme linked immunosorbent assay for confirming the diagnosis of measles. J Clin Microbiol 1986, 24: 391-394.

13. Guyer B, McBean AM. The epidemiology and control of measles in Yaounde, Cameroun, 1968-1975. Int J Epidemiol 1981, 10: 263-269.