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Inside . . .

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Leptospirosis as a Cause of Febrile Illness Requiring Hospitalization in Dhaka, 2001

In 2000, the ICDDR,B established surveillance at two major Dhaka hospitals to identify patients with dengue. Evaluation of serum from enrolled patients during 2001 with negative laboratory tests for dengue revealed that leptospirosis is an important cause of serious febrile illness in the urban setting. Compared with patients with dengue, patients with leptospirosis were of poorer socioeconomic status and had higher fever on presentation. The mortality rate among patients with leptospirosis was 5%.

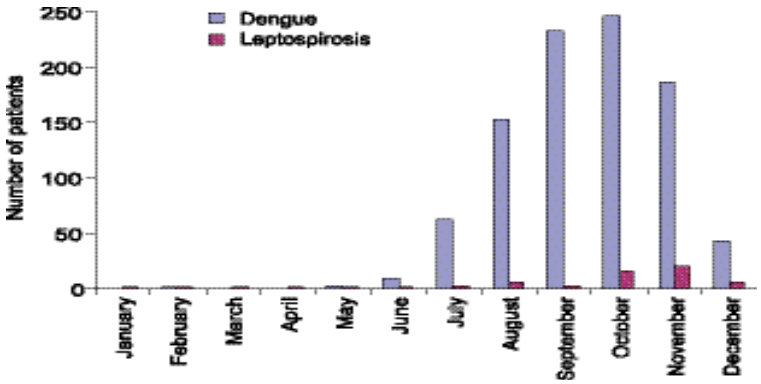
Hospital-based surveillance for dengue fever and dengue haemorrhagic fever at Dhaka Medical College Hospital (DMCH) and Holy Family Red Crescent Hospital (HFRCH) in Dhaka (1) was used to assess the role of leptospirosis as a cause of febrile illness requiring hospitalization. Clinical and epidemiologic data and serum specimens were collected systematically from patients with dengue-like illness. Serum specimens were assessed for the presence of dengue virus antibodies by IgG and IgM capture ELISA (2). In addition, sera collected during the first five days of illness were evaluated for the presence of dengue virus ribonucleic acid (RNA) by reverse-transcriptase polymerase chain reaction (PCR) (3). Sera already obtained for diagnostic purposes from dengue-negative patients were evaluated by PCR to detect leptospira genetic material at the Centers for Disease Control and Prevention (CDC) in Atlanta, Georgia.

Specimens from 1297 patients who were hospitalized between January 1 and December 31, 2001 were evaluated; 935 (72%) patients were diagnosed with dengue fever by paired serology or RT-PCR. Serum specimens from the 362 patients without laboratory evidence of dengue were evaluated for evidence of

leptospirosis; 63 (17%) had ***Leptospira*** species detected by PCR.

The peak incidence of leptospirosis occurred from October through December. Similarly, the period with highest dengue activity was July through December with the peak activity in October (Figure 1).

Figure 1: Dengue and leptospirosis hospitalizations at DMCH and HFRCH, by month of presentation in 2001



Patients with leptospirosis did not differ in age or gender from patients with dengue (Table 1). Patients with leptospirosis came from households with lower income and less education compared with patients with dengue.

Table 1: Demographic characteristics of dengue positive patients compared with leptospirosis positive patients

Demographic Characteristics	Dengue patients (n=935)	Leptospirosis patients (n=63)	p value*
Age (years)	27.5 ±11.1	27.9 ± 13.3	NS**
Male Gender	691 (74%)	46 (73%)	NS**
Monthly Household Income (Tk)			
< 3,000	174 (19%)	20 (32%)	0.02
3,000-5,999	203 (22%)	17 (27%)	
6,000-10,000	127 (14%)	7 (11%)	
>10,000	431 (46%)	18 (29%)†	
Household Size	5.4 ± 2.7	4.8 ± 2.1	NS**
Level of Education			
Illiterate	83 (10%)	16 (27%)	<0.001
Primary	144 (17%)	13 (22%)	
Secondary	423 (49%)	22 (37%)	
University	212 (25%)	8 (14%)§	

* Data is mean ± S.D. P-value calculated using Pearson chi-square or ANOVA tests.
**NS = not significant
† data unavailable for one patient
§ data unavailable for 4 patients

The characteristics of fever in leptospirosis differed from that of dengue (Table 2). Patients with leptospirosis complained of a longer duration of fever than patients with dengue, and the fever was more commonly intermittent rather than continuous.

The presenting complaints of patients with leptospirosis and dengue were similar (Table 2). Aside from fever, the most common complaints in both diseases were headache, myalgias, nausea and vomiting. Although present in both leptospirosis and dengue, complaints of rash were slightly more common with dengue.

The median temperature and heart rate on presenting physical exam were significantly higher in leptospirosis patients than in dengue patients. Evidence of bleeding, including petechial rash, positive tourniquet test and gum bleeding, were more common in patients with dengue, although they were also present in some patients with leptospirosis. Subconjunctival haemorrhage was more commonly reported in patients with leptospirosis, although it is possible that conjunctival inflammation was confused with this sign.

On laboratory examination, total white blood cell counts were similar in patients with dengue and patients with leptospirosis; however lymphocytic predominance was more common with dengue.

Three hundred seventy-two patients in this study underwent ultrasound; a higher proportion of patients with dengue than those with leptospirosis had evidence of ascites (54% vs. 23%, $p = 0.002$) and pleural effusion (58% vs. 27%, $p = 0.002$). These findings may be due to capillary leakage in patients most severely ill with dengue. Haemoconcentration was also more commonly seen in dengue.

Death occurred in three (5%) leptospirosis patients whose outcome was known, compared with eleven (1.2%) patients with dengue. Data on income and level of education was available for two of the three leptospirosis patients who died; both had no education and had incomes <3000 taka/month.

Reported by: Dhaka Medical College Hospital; Holy Family Red Crescent Hospital, Dhaka; Division of Bacterial and Mycotic Diseases, and Division of Vector-borne Infectious Diseases, National Center for Infectious Diseases, Centers for Disease Control and Prevention (CDC), Atlanta, USA; Massachusetts General Hospital, Boston, USA. Laboratory Sciences Division and Health Systems and Infectious Diseases Division, ICDDR,B.

Supported by: Canadian International Development Agency (CIDA); Centers for Disease Control and Prevention, USA.

*Table 2: Distinguishing clinical characteristics of dengue positive patients compared with leptospirosis positive patients**

Clinical Characteristics	Dengue patients (n=935)	Leptospirosis patients (n=63)	p value
Presenting complaints			
Median duration of fever on presentation (days)	5 (1-40)	6 (2-12)	0.04
Characteristic of fever			
Continuous	891 (95%)	53 (85%)	0.001
Intermittent	44 (5%)	9 (15%)	
Rash	561 (60%)	24 (39%)	0.001
Headache	843 (90%)	51 (82%)	0.05
Myalgias	833 (89%)	53 (85%)	NS+
Abdominal pain	443 (47%)	24 (39%)	NS+
Pruritus	202 (22%)	6 (11%)	0.05
Rhinitis	9 (1%)	3 (5%)	0.03
Nausea	896 (96%)	60 (97%)	NS+
Vomiting	777 (83%)	52 (84%)	NS+
Diarrhoea	334 (38%)	23 (37%)	NS+
Melaena	477 (52%)	32 (53%)	NS+
Haematochezia	9 (1%)	0 (0%)	NS+
Physical findings			
Median temperature on presentation (ºF)	98.6 (94-106)	101.4 (98-108)	<0.001
Median heart rate	82 (48-160)	90 (60-180)	0.001
Hepatomegaly	78 (8%)	7 (11%)	NS+
Jaundice	17 (2%)	3 (5%)	NS+
Petechial rash	347 (38%)	12 (20%)	<0.01
Positive tourniquet test (>20/sq inch)	798 (76%)	20 (33%)	<0.001
Gum bleeding	327 (36%)	12 (20%)	<0.01
Subconjunctival haemorrhage	296 (32%)	31 (51%)	<0.01
Laboratory findings			
Mean WBC count	6.9±5.3	7.0±5.7	NS+
% Neutrophils	44±13	59±18	<0.001
% Lymphocytes	47±14	31±15	<0.001
Mean platelet count ($x10^3$)	85±74	128±83	<0.001
Mean haematocrit	41±5.8	37±8	<0.001
Outcome of hospitalization**			
Recovered or left against medical advice	915 (98.8%)	57 (95%)	0.05
Death	11 (1.2%)	3 (5%)	

* Data are presented as N (%), mean ± standard deviation, or median (range). Pvalues where calculaed using the Pearson chi-square test for categorical data and Mann-Whitney U or ANOVA test for continuous data

\+ NS = not significant

** Outcome information not available for all patients

Comments

The findings of this report suggest that leptospirosis is an important cause of serious febrile illness necessitating hospitalization in Dhaka. Leptospirosis is a zoonotic infection with worldwide distribution, caused by spirochetes of the genus ***Leptospira***. The disease is maintained in nature by chronic infection of the renal tubules of animal hosts, including rodents, domestic animals and farm animals. Infection in humans usually results when skin abrasions come in contact with water or soil contaminated with the urine of an infected animal (4). The incidence of leptospirosis is highest in countries with warm, humid climates where the organism is able to survive longer in the environment, and in areas where people are readily exposed to contaminated water sources.

Leptospirosis patients identified in Dhaka were impoverished and poorly educated. This may reflect more frequent exposure to environments contaminated with urine from rodents or other animals. In contrast, dengue patients tended to come from households with higher income and education; if societal differences are confirmed in future studies, they will need to be considered as strategies evolve to prevent these two diseases.

Studies conducted in dengue-endemic areas have shown that leptospirosis can be confused with dengue (5). Most of the presenting clinical symptoms of leptospirosis patients in Dhaka were non-specific and not distinguishable from symptoms associated with dengue, as well as with other viral illnesses. While fever in leptospirosis patients was notably higher and of longer duration than that observed in dengue patients, there is sufficient overlap of clinical findings to suggest that clinicians should maintain a high index of suspicion for both diseases, especially during peak incidence periods.

Leptospirosis can be a biphasic illness with late complications such as aseptic meningitis occurring due to the host immune response. Severe disease can also be characterized by jaundice, renal dysfunction and haemorrhage. This retrospective serologic study did not allow for the assessment of these outcomes among leptospirosis patients in Dhaka.

Techniques available for the diagnosis of leptospirosis are available in specialized laboratories and include culture, serology and PCR testing. Treatment with doxycycline (6) or penicillin (7) has been shown to shorten the course of illness and duration of shedding of the organism in urine, although controversy exists regarding the clinical benefits of specific treatment given the variable natural history of the disease.

The case-fatality rate among leptospirosis patients in Dhaka was 5%. This finding underscores the need for increased awareness of leptospirosis, further data on its incidence in the community, and optimal management regimens that can be applied in Bangladesh. Although the patient populations at DMCH and HFRCH may differ from those in other regions of Bangladesh, leptospirosis should be considered in patients presenting with clinical illnesses consistent with dengue or dengue haemorrhagic fever.

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Nipah Encephalitis Outbreak Over Wide Area of Western Bangladesh, 2004

During January and February 2004, an outbreak of Nipah encephalitis occurred. Twenty-nine laboratory confirmed or probable cases were identified; 22 patients died. While most cases occurred within Goalanda in Rajbari District, Nipah associated illness was also identified in Joypurhat, Naogaon, Natore, Faridpur, Gopalganj, Manikganj, and Dhaka Districts. There was no clear evidence of person-to-person transmission during this outbreak. Fruit bats (***Pteropus giganteus***) appear to be the principal reservoir for the virus; ongoing studies have the objective of defining the modes of transmission.

During 12-17 January 2004, twelve residents of two contiguous villages in Goalanda, Rajbari District developed febrile illnesses progressing to coma; ten of these illnesses resulted in death. All but two of the patients were between the ages of seven and 15 years and eight were male. The patients lived in close proximity to each other, including three brothers who lived in one house and two brothers in another house, as well as a mother and toddler. Following notification about the cluster on 21 January, ICDDR,B and the Institute of Epidemiology and Disease Control Research (IEDCR) began a joint investigation on January 22 at the request of the MoHFW. Staff from the Centers for Disease Control and Prevention, Atlanta and from the World Health Organization, Geneva, arrived on 4 February to assist in the investigation.

Testing clinical specimens from these patients (serum specimens, cerebrospinal fluids (CSF)) for IgG and IgM antibodies and throat swabs and CSF for culturable virus and presence of Nipah virus RNA by reverse transcriptase-polymerase chain reaction (RT-PCR) have demonstrated that Nipah virus is the cause of this outbreak.

The investigation team is focusing on defining the magnitude and geographic scope of infection with Nipah viruses during this outbreak, and on identifying reservoirs of the virus and sources of transmission. Civil surgeons in a variety of surrounding districts were contacted and asked to report similar cases, defined as fever associated with mental status change (including disorientation, delirium or coma). When possible, specimens from these cases were collected for laboratory testing to document infection with Nipah virus—many patients died before specimens could be obtained. A laboratory confirmed case was defined as a patient with evidence of acute infection demonstrated by the presence of IgM antibodies to Nipah viruses in serum or CSF. A probable case was defined as a patient with fever and mental status changes living near a confirmed case. The investigation also included collection and testing for Nipah virus infection from a variety of animals in areas with reported cases. Both human and animal specimens were sent to CDC in Atlanta for testing for evidence of Nipah infection.

Through 5 April 2004, 29 cases were identified through active case detection, including 14 laboratory confirmed cases; 22 (76%) died. While many were from Goalanda in Rajbari District, confirmed cases were also identified from as far as 150 kilometers away in Joypurhat, Naogaon, Natore, Faridpur, Gopalganj, Manikganj, and Dhaka Districts (Figure 1). Disease onset of most identified cases in these other areas was simultaneous or within a few weeks of that of the cases in Rajbari District (Figure 2). No information is available to link the two cases occurring in March in Natore and Dhaka with the other cases described in this report. No illnesses were identified among health care workers in facilities where cases were managed.

Figure 1: Location of residences for several clusters of cases

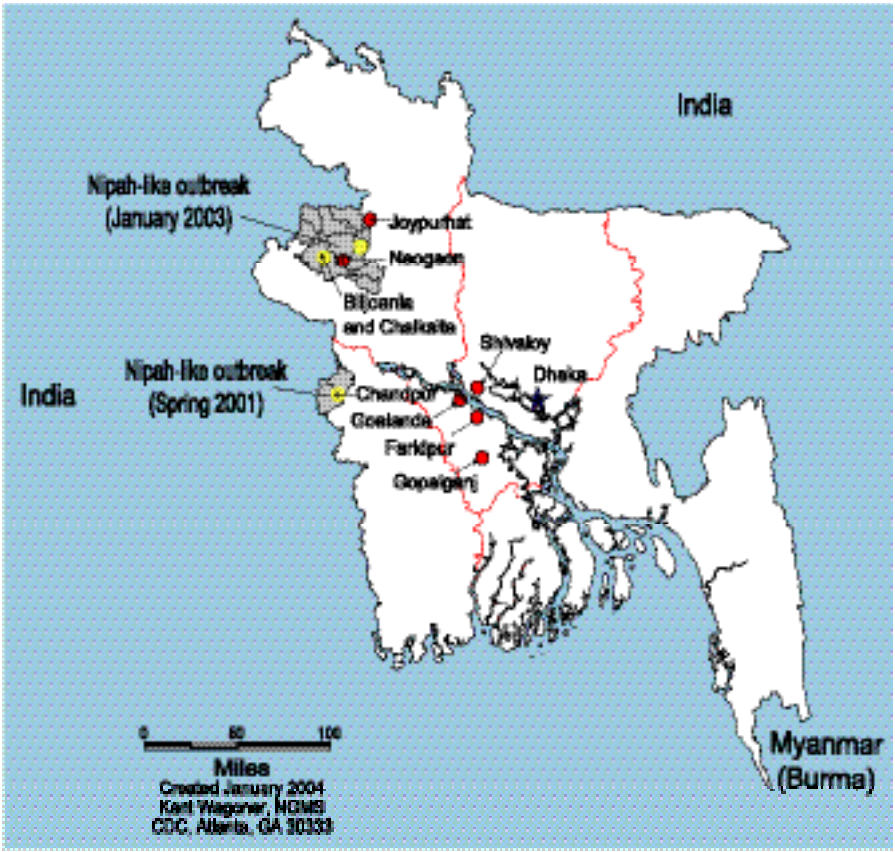
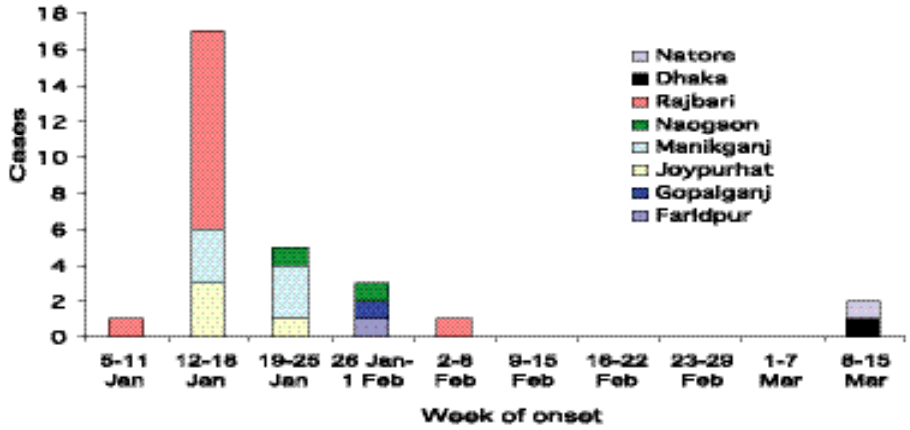


Figure 2: Epicurve of Bangladesh Nipah outbreak 2004 by week of onset and district (n=29)



The investigation is ongoing and includes a case-control study to evaluate risk factors for infection, which may be helpful in characterizing how infection occurred, and a Nipah virus antibody prevalence study among 266 residents of Goalanda to determine the scope of current and previous virus transmission and the spectrum of clinical involvement following infection with Nipah virus (to evaluate whether milder illness or asymptomatic infection occurred). Over 450 animals, including bats, fowl, pigs, horses, goats, cows, rodents, shrews, cats and dogs were sampled and their specimens will be tested for evidence of Nipah infection over the coming weeks. Preliminary studies confirm that fruit bats (***Pteropus giganteus***) of the genus ***Pteropus*** have serological evidence of past Nipah virus infection.

Reported by: Institute of Epidemiology and Disease Control Research, Dhaka Medical College Hospital, Rajshahi Medical College Hospital, Faridpur Medical College Hospital, Directorate General of Health Services, Ministry of Health & Family Welfare (MoHFW), Bangladesh; World Health Organization, Dhaka and Geneva; Division of Viral and Rickettsial Diseases, National Center for Infectious Diseases, CDC-Atlanta, USA; Clinical Sciences Division, Laboratory Sciences Division and Health Systems and Infectious Diseases Division, ICDDR,B, Dhaka, Bangladesh

Supported by: Centers for Disease Control and Prevention, USA; World Health Organization

Comment

This is the third recognized outbreak of encephalitis in Bangladesh due to Nipah or Nipah-like viruses (1). Clinical materials from this outbreak investigation have allowed the isolation and genetic characterization of the

causative agent, a strain of Nipah virus closely related but distinguishable from the virus that had caused a large outbreak of encephalitis in Malaysia in 1998-1999 (2). Diagnoses during the previous outbreaks were based on serological tests which made it impossible to define the precise identity of the etiologic agent; however, the isolation and characterization of Nipah viruses during the current outbreak increases the likelihood that the Nipah virus identified from patients is responsible for all three outbreaks. This suggests that there is a zone of periodic transmission of Nipah viruses from their reservoir hosts to man in western Bangladesh, and that this may result in clusters of human encephalitis.

Bats (fruit bats, genus ***Pteropus***) continue to be suspected to be the main reservoir for Nipah viruses (3-5). The ongoing investigation will attempt to define whether transmission occurred directly from bats to humans (perhaps via bat secretions or excrement or through ingesting fruits which had been contaminated by an infected bat) or whether other animals are involved in amplifying virus and transmitting to humans. The predominance of young boys within the Goalanda cluster suggests that a specific type of activity may have resulted in exposure to the virus; further epidemiologic studies combined with animal surveys may provide some suggestions as to the mode(s) of transmission. As in the previous Nipah virus outbreaks in Bangladesh, pigs did not appear to play a central role in maintenance and transmission of Nipah virus to man as occurred with the first outbreak of Nipah encephalitis in Malaysia and Singapore (6). Limited person-to-person transmission cannot be ruled out in this outbreak since several of the cases occurred within the same households but also shared common environmental exposures.

The occurrence of three outbreaks of Nipah encephalitis since 2001 raises questions about potential prevention and control strategies. The outbreaks have had devastating effects upon the villages where they have occurred. Current therapy for Nipah infection is supportive—with careful fluid management and respiratory care. Ribavirin, a very expensive antiviral therapeutic, has been said to improve outcomes (7). However, ***in vitro*** and laboratory animal studies have found the drug to have no effect on replication of the virus or disease outcome (Rollin, CDC, unpublished data). Prevention strategies await more information on mode of transmission, and may involve interventions that reduce the likelihood of direct exposure to ***Pteropus*** secretions, urine or faeces.

Added Note:

A new outbreak of Nipah virus encephalitis in Faridpur District is currently under investigation. As of April 25, 2004, 33 cases have been identified with 24 deaths. In contrast with previous outbreaks, there is evidence that person-to-

person transmission may be responsible for many of the cases, and several patients have acute respiratory distress syndrome. The focus of the current efforts involving the MoHFW, WHO, ICDDR,B and CDC is on restricting transmission of the virus in the community and in hospitals, and on further characterizing how disease is being transmitted, defining the magnitude of the problem, identifying persistent animal reservoirs of the virus, and determining the genetic diversity of Nipah viruses in Bangladesh. Updated information will be available in future issues of the HSB, and may be posted on the ICDDR,B website at www.icddrb.org.

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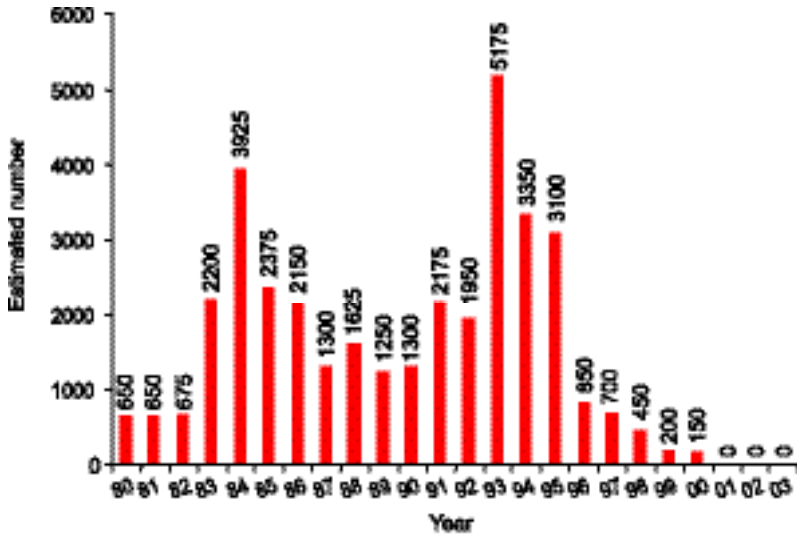
Increasing Antibiotic Resistance of *Shigella* species

Surveillance for diarrhoeal disease pathogens at ICDDR,B hospital in Dhaka has shown that since 1980, ***Shigella*** species have been becoming increasingly resistant to commonly used antimicrobial drugs. ***Shigella dysenteriae*** type 1 (***Sd***1), responsible for major epidemics during the 1980s and 1990s has acquired resistance to trimethoprim/sulphamethoxazole, tetracyclines, chloramphenicol, and most recently, fluoroquinolones, raising the potential for a new outbreak of ***Sd***1-associated diarroheal illness.

Diarrhoeal disease surveillance data from ICDDR,B hospital in Dhaka, has made it possible to identify changing patterns of antimicrobial resistance of ***Shigella*** species during the period 1980 to 2003. All four species of ***Shigella*** were isolated during this period. ***Shigella flexneri*** was the most common species isolated during each year of surveillance; overall, ***S. flexneri*** represented 57% of isolates and was followed in frequency by ***Shigella dysenteriae*** which accounted for 21%. ***S. dysenteriae*** serotype 1 (known as Shiga bacillus or ***Sd***1) was isolated during two peak periods in 1983-5 and 1993-4 (Figure 1). While ***Sd***1 has not been isolated from surveillance specimens during recent years, there have been scattered outbreaks due to ***Sd***1 in India, Bangladesh and Nepal (1,2).

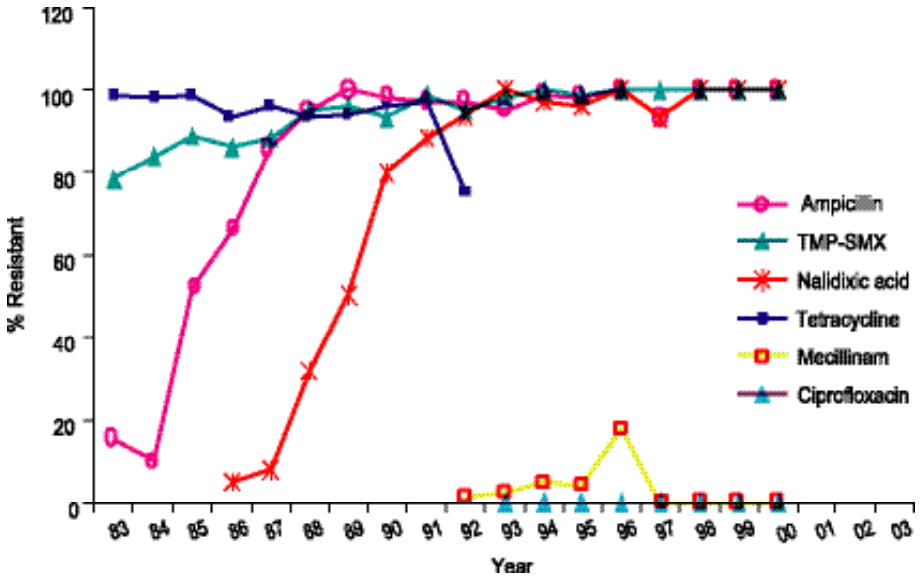
Figure 1: Estimated number of S. dysenteriae ***type 1 isolates, Hospital Surveillance, Dhaka, 1980-2003***



All ***Shigella*** species have shown increasing resistance to multiple antimicrobial drugs formerly useful for treatment. The pattern has been for these bacteria to become resistant to an antibiotic, or group of antibiotics, and then to retain this antibiotic resistance pattern with little suggestion of ever losing antibiotic resistance. This is in contrast to ***V. cholerae*** in which resistant strains have appeared, but then the dominant strain often becomes susceptible once again (3).

Historically, shigellae were susceptible to tetracycline and choramphenicol and other commonly used drugs, but by 1984, 98% of ***Sd***1 isolates were resistant to tetracycline, 84% to trimethoprim-sulphamethoxazole (TMP-SMX), 84% to chloramphenicol, and 10% to ampicillin. Ampicillin resistance increased to 52% in 1985 (Figure 2). By 1993, all (100%) ***Sd***1 isolates were resistant to nalidixic acid, 98% to TMP-SMX, and 95% to ampicillin. Among ***Sd***1 strains, resistance to nalidixic acid increased from 5% in 1986 to 80% in 1990, and 100% in 1993. The proportion of resistant ***S. flexneri*** also increased; for instance, resistance to nalidixic acid increased from 4% in 1986 to 66% in 2003.

Figure 2: Antimicrobial resistance pattern of S. dysenteriae *type 1 isolates, Hospital Surveillance, Dhaka, 1983-2003*



Nearly all ***S. flexneri*** strains have remained susceptible to mecillinam and ciprofloxacin; however, a few strains of ***Sd***1 (not included in this surveillance system) have demonstrated resistance to ciprofloxacin.

Reported by: Laboratory Sciences Division and Clinical Sciences Division, ICDDR,B

Supported by: United States Agency for International Development

Comment

It is notable that the previous epidemics of ***Sd***1 were due to strains that were resistant to the antimicrobial drug which had been commonly used. For example, the outbreak strain during 1984 became resistant to TMP-SMX and the 1993 epidemic had acquired resistance to nalidixic acid. Thus, the emergence of ciprofloxacin resistant strains, as occurred in 2003 was not surprising (1,2). Whether ciprofloxacin resistant strains of ***Sd***1 will be associated with an epidemic similar to that seen in 1984 and 1993 cannot be assured, but this seems likely. If such an ***Sd***1 epidemic were to occur, the most suitable antibiotic for use in Bangladesh is mecillinam. Shigellae strains are susceptible ***in vitro*** to other antimicrobial drugs, such as gentamicin and certain cephalosporins, but these drugs have not been useful clinically. Fortunately, most cases of shigellosis continue to be caused by strains other than ***Sd***1, and they remain susceptible to ciprofloxacin.

Systematic monitoring of the species and serotypes of Shigellae and their antimicrobial susceptibility can help to guide therapy and reveal periodic epidemics due to ***Sd***1, which may have acquired resistance to antibiotics that have previously been effective.

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Surveillance Update

With each issue of the HSB, updates of surveillance data described in earlier issues will be provided. These updated tables and figures will represent the most recent observation period available at the time of publication. We hope these updates will be helpful to health professionals who are interested in current patterns of disease and drug resistance.

Proportion of diarrhoeal pathogens susceptible to antimicrobial drugs: March 2003 - February 2004

Antimicrobial agent	*Shigella* (n=318)	*V. cholerae* O1 (n=516)	*V. cholerae* O139 (n=24)
Nalidixic acid	45.1	NT	NT
Mecillinam	99.0	NT	NT
Ampicillin	47.5	NT	NT
TMP-SMX	34.0	0.2	100.0
Ciprofloxacin	99.0	100.0	100.0
Tetracycline	NT	100.0	100.0
Erythromycin	NT	99.8	100.0
Furazolidine	NT	0.2	100.0

NT=Not Tested

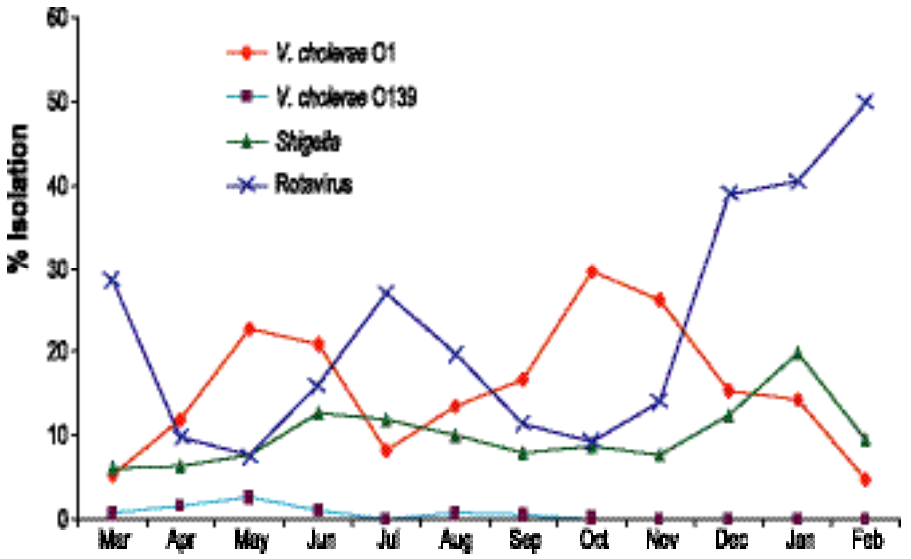
Antimicrobial resistance patterns of 172 M. tuberculosis ***isolates***: ***January - November 2003***

Drugs	Resistance type		Total (n=172)
	Primary (n=139)	Acquired* (n= 33)	
Streptomycin	79 (56.8)	21 (63.6)	100 (58.1)
Isoniazid (INH)	19 (13.7)	9 (27.3)	28 (16.3)
Ethambutal	3 (2.2)	6 (18.2)	9 (5.2)
Rifampicin	4 (2.9)	4 (12.1)	8 (4.7)
MDR (INH+Rifampicin)	4 (2.9)	4 (12.1)	8 (4.7)
Any drug	82 (59.0)	21 (63.6)	103 (59.9)

( ) column percentages

* Antituberculous drugs received for 1 month or more

Monthly isolations of V. cholerae ***O1,*** V. cholerae ***O139,*** Shigella ***and Rotavirus: March 2003 - February 2004***



Antimicrobial susceptibility of N. gonorrhoeae ***isolated during October - December 2003 (n=40)***

Antimicrobial agent	Susceptible (%)	Reduced susceptibility (%)	Resistant (%)
Azithromycin	100.0	0.0	0.0
Ceftriaxone	100.0	0.0	0.0
Ciprofloxacin	5.0	0.0	95.0
Penicillin	17.5	30.0	52.5
Spectinomycin	97.5	2.5	0.0
Tetracycline	2.5	2.5	95.0
Cefixime (n=23)	100.0	0.0	0.0

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Photo of ***Pteropus*** bats, courtesy of Dr. Ivan Kuzmin

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