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تصدرها وزارة الصحة

الوكالة المساعدة للطب الوقائي وبرنامج الوبائيات الحقلي

المجلد الثالث – العدد الأول – يناير ، فبراير ، مارس ١٩٩٦م

A novel flavivirus: Makkah Region 1994-1996

Since the identification of Congo-Crimean hemorrhagic fever (CCHF) in the Western region in 1991, surveillance of viral hemorrhagic fevers (VHF) has continued to identify patients for whom neither CCHF or dengue was confirmed. In 1995 a previously unidentified flavivirus was isolated from a specimen from a patient who died from suspected CCHF in 1994. The Division of Vectorborne Infectious Diseases, Fort Collins Laboratory, Centers for Disease Control and Prevention examined the virus serologically and by partial nucleotide sequence analysis of the NS5 gene and confirmed that it was a member of the tick borne encephalitis (TBE) group.

Some of the previously negative serum and blood specimens from 1994 and 1995 from CCHF (4), VHF (17), and dengue (19) surveillance were retested for this virus. A case was defined as any febrile patient from whom the flavivirus was isolated or had seroconversion for the new flavivirus antigen without seroreactivity to dengue or CCHF. In addition to the index case, eight other patients had either virus isolation (5) or seroconversion (3).

Medical records were complete on eight patients and lacked laboratory information for one patient who died shortly after admission. All patients had fever (mean 38.7°C, range 37.1°-40 C°) with a mean duration of 5.6 days. Fever was accompanied with chills in four patients and sweating in two patients. Five patients had hemorrhagic phenomena including epistaxis (2), echymosis at needle puncture sites (3), extensive subcutaneous bleeding (1), bloody diarrhea or rectal bleeding (2), and hematemesis (1). Three patients had a morbilliform rash. Two patients with hemorrhagic disease also had encephalitis manifested by convulsions semicoma and coma. Both had normal computerized tomography scans of their brains. Six others had either unusual irritability or drowsiness. Two patients died. During the first week after onset of fever all eight patients with

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Flavivirus, Makkah region, 1994-1996

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laboratory testing had leucopenia, thrombocytopenia and elevated serum transaminases and two had elevated serum urea and creatinine (Table 1). Maximum abnormalities were reached at the end of first week from onset of fever.

Additional exposure and demographic information was obtained by reviewing patients' medical files, interviewing the patients (6), and by interviewing the relatives or coworkers of patients (1) who had either died or left the country and could not be interviewed. All patients were adults with a mean age of 34 years (range 24-55) and eight were males. Four were Egyptian, three came from the Indian subcontinent, and two were Saudis. All non-Saudi patients had been in Saudi Arabia for more than three months before their illness. Onset of illness occurred either in the spring or fall months of 1994 or 1995 (Figure). Three patients lived in Jeddah, five in Makkah and one was a butcher from Bisha. The Bisha butcher developed fever while attending Hajj on 21/5/1994 and died without providing the date that he traveled from Bisha to Makkah. Of the seven non-Saudi patients, six were butchers, and one was a zoo worker. The Saudi patients were a soldier and a housewife.

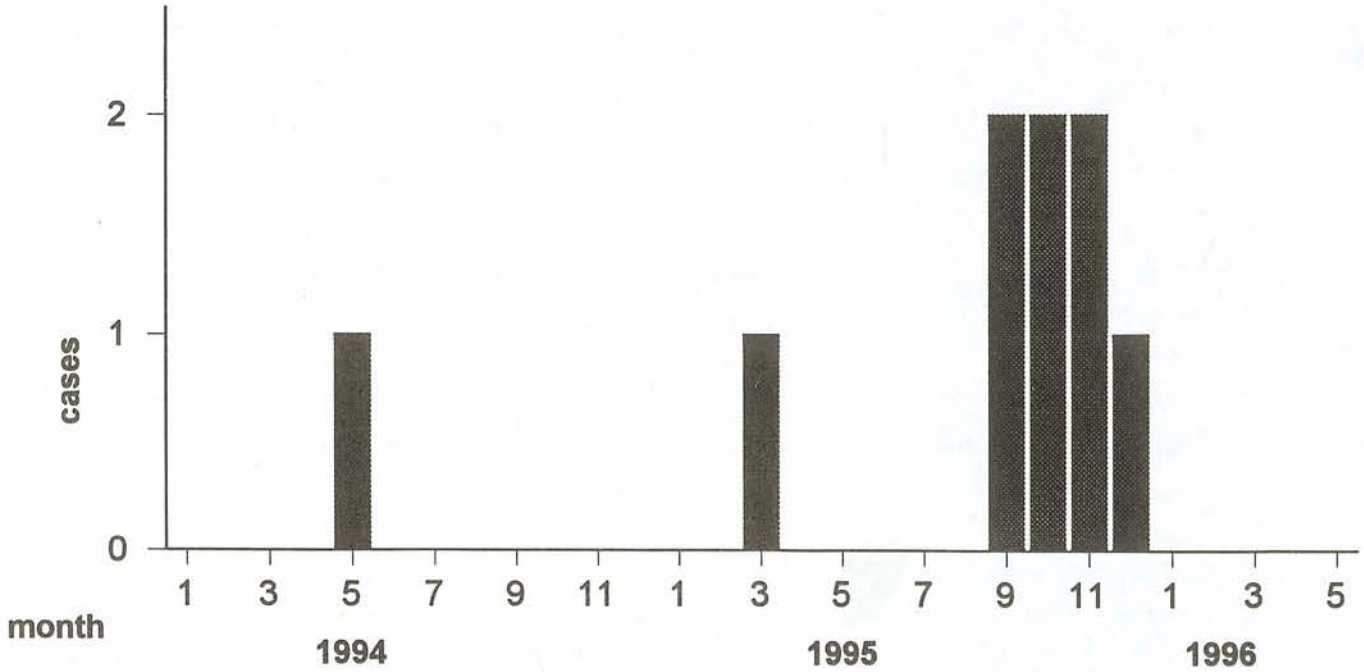
The six butchers handled raw meat either through slaughtering (2), or butchering in butcher shops (2) or kitchens (2). All had daily exposure to sheep meat. In addition to sheep meat, two handled beef and camel meat, and histories for three were unavailable. Three were interviewed directly and reported that they cut their hand frequently while working. One recalled a deep cut in his foot with a bloody knife one week before onset of illness. The zoo worker only carried raw meat (beef or camel) in his bare hands to feed the zoo animals. The housewife's servants butchered freshly slaughtered sheep carcasses at her home every week. Once around the time of her onset of illness she assisted the servants in butchering a sheep carcass. However, she could not remember whether she had done this before or after her illness.The Saudi Soldier reported visiting a camel pinfold one week before his illness where he drank a large amount of raw camel milk. He denied slaughtering or butchering meat or having tick bites during the month before onset of illness.

Among the six patients that were interviewed, ticks were reported at the work sites of three patients, but not from their residences. Mosquitoes were reported from both work sites and residences of two patients. Rat infestations at home or work were not reported by any of the patients. No patient reported any history of insect bite, blood transfusion, hospitalization, or contact with patients with a similar disease two weeks prior to their illness.

The Department of Agriculture and the Municipality in the region were informed and participated in control measures. Livestock pinfolds in residential areas were closed. In other legal pinfolds, flooring was scraped, sterilized, sprayed with Diazenon and covered with new treated soil. Pre-slaughtering veterinary check up was emphasized, and butchers and slaughterhouse workers were instructed to apply precautions such as gloving and booting to reduce skin contact and pericutaneous exposure to blood. Hospitals and medical departments were informed to report any suspect VHF or encephalitis.

—Reported by: Disease Control Department - King Saud Hospital, Jeddah Region Health Affairs (Dr. Ilham Qattan, Dr. Naeema Akbar, Dr. Huda Afif,

Figure. Cases of a novel flavivrus infection by month of onset. Makkah Region, Saudi Arabia, 1994-1996.



(continued from page 2)

Table 1. Serum chemistry and blood cell counts for eight patients with a novel flavivirus. Makkah Region, 1994 to 1995.

	N*	Mean	Range	Abnormal
Serum glutamic oxaloacetic transaminase (U/l)	8	676	60-1950	100%
Serum glutamic pyruvic transaminase (U/l)	8	329	45-689	100%
Lactic dehydrogenase (U/l)	7	2757	284-4641	86%
Alkaline phosphatatase (U/l)	7	127	51-197	71%
Creatine phosphokinase (U/l)	7	4435	58-8280	86%
Urea (mg/100ml)	8	63	9-186	25%
Creatinine (mg/100ml)	8	2.1	1-6	25%
Thrombocytes (cellsx10^3 µ/l)	8	61	11-110	100%
Leucocytes (cellsx10^3 µ/l)	8	2.0	1.1-2.9	100%

*N = number of patients

Dr. Somayah Abu Azmah and Dr.Taha Al-Khateeb); Primary Health Care Department in Makkah; Dr. Ali Zaki, Soliman Faqeeh Hospital Jeddah; Division of Vector Borne Infectious Disease, National Center for Infectious Disease, Centers for Disease Control and Prevention, USA; Dr. Nasser Al-Hamdan and Dr. R.E. Fontaine, Saudi Arabian Field Epidemiology Training Program, Ministry of Health.

Editorial note: Flaviviruses in the TBE group are known to range geographically from Negishi in Japan to Omsk hemorrhagic fever (OHF) and Russian spring summer Encephalitis (RSSE) in west and central Asia to Central European Encephalitis (CEE) in Europe to louping ill in the British Isles and southward as Kyanasur Forest disease (KFD) in India. Individual viruses can produce encephalitis alone (RSSE, CEE, and louping ill), VHF or a combination of VHF, encephalitis, and renal dysfunction (KFD, OHF). In their natural cycles these viruses are transmitted by ticks. The natural vertebrate hosts are small mammals and birds. Livestock become infected by tick bite. Transmission to man has been reported from tick bites, trapping and skinning muskrats (OHF), consuming raw milk or cheese (CEE, RSSE), butchering infected sheep (louping ill), and accidental laboratory infections (CEE, KFD, louping ill). Treatment of disease resulting from TBE flaviviruses is supportive.

The exposure information from the nine patients cases presented here indicates that the principal mode of transmission to humans for this novel flavivirus is pericutaneous from slaughtering livestock or butchering fresh meat and from drinking raw camel milk.

Control measures should include the continuous cleaning and spraying of animal markets, trucks, pinfolds , and slaughter houses to eliminate ticks. Veterinary services should be available at these places to identify sick animals. Drinking raw milk or products made from raw milk (cheese, laban, yogurt) should be discouraged.

Physicians and veterinarians should report cases of suspected VHF in humans or animals within 24 hours to the DCD (02-647-9775) in the Makkah region and to the Ministry of Health (01 405 7494) for other areas of Saudi Arabia. VHF is defined as onset of fever followed within one week by hemorrhagic manifestations. These include subcutaneous hemorrhage, a rash with a positive tourniquet test, excessive bleeding at needle puncture sites, any gastrointestinal bleeding, and epistaxis. Encephalitis is also reportable. Since nosocomial transmission via blood from patients to medical staff is documented for CCHF and is possible for TBE viruses, any suspect VHF patient should be placed under strict isolation until fever and hemorrhage have resolved. For diagnosis please submit the following specimen to DCD in Jeddah: 10 ml of clotted venous blood. Postmortem serum, liver, lung, spleen and kidney samples should be submitted on fatal VHF cases. All specimens should be submitted in sealed plastic containers enclosed inside a second sealed plastic container. Specimens should be sent immediately on wet ice or frozen (-70 C) and sent on dry ice.

References:

1. Monath TP: Flaviviruses in Fields BN, Knipe DM (2nd ed): Fields Virology.Raven Press,

New Versions of Epidemiology Software Available

Epi Info Version 6.04 and Epi Map 2.0 are now available from the Field Epidemiology Training Program. If you would like these programs please send three diskettes for Epi Info and /or four diskettes for Epi Map to:
Abdulbaki Saleh, Secretary
Field Epidemiology Training Program
Ministry of Health
PO Box 6344
Riyadh 11442
Kingdom of Saudi Arabia

Malaria Control in the Kingdom of Saudi Arabia

Currently, about 1.6 million people live in areas of Kingdom of Saudi Arabia where malaria is transmitted. In the southwestern region (Tihama), *Plasmodium falciparum* causes over 90% of cases and in the northwestern region 35% of malaria cases. *P. vivax* is a predominant species in the northwest and accounts for over 50% of the cases, whereas *P. malariae* is scarce (1-2% of the cases in KSA). The peak of malaria transmission occurs between October and April and coincides with the rainy season (70-550 mm/year). There is a noticeable decline in the incidence of malaria during summer months. Although KSA is a dry country, permanent springs in the central and eastern regions, and streams that traverse the coastal mountainous range along the Red Sea provide suitable breeding places for Anopheline mosquitoes.

Geographically, malaria endemicity in KSA can be divided into four categories (Figure): Non-malarious areas in Central KSA (associated with low density of *Anopheles sergentii*) where only occasional imported cases are reported every year; areas where transmission of malaria has been halted by institution of effective control measures in Eastern and Northern KSA (*An. superpictus* and *An. stephensi*); areas with low malaria incidence (1 to 3 per 1,000 per year [foci in remote areas in western KSA] *An. superpictus* and *An. sergentii*), and some parts of the southern KSA (*An. arabiensis*); and areas with medium or high malaria incidence (>3 per 1,000/ year [foothills and lowlands of Tihama and the coastal plain along the Red Sea in the southern and southwestern KSA down to the border with Yemen] *An. arabiensis* and *An sergentii*).

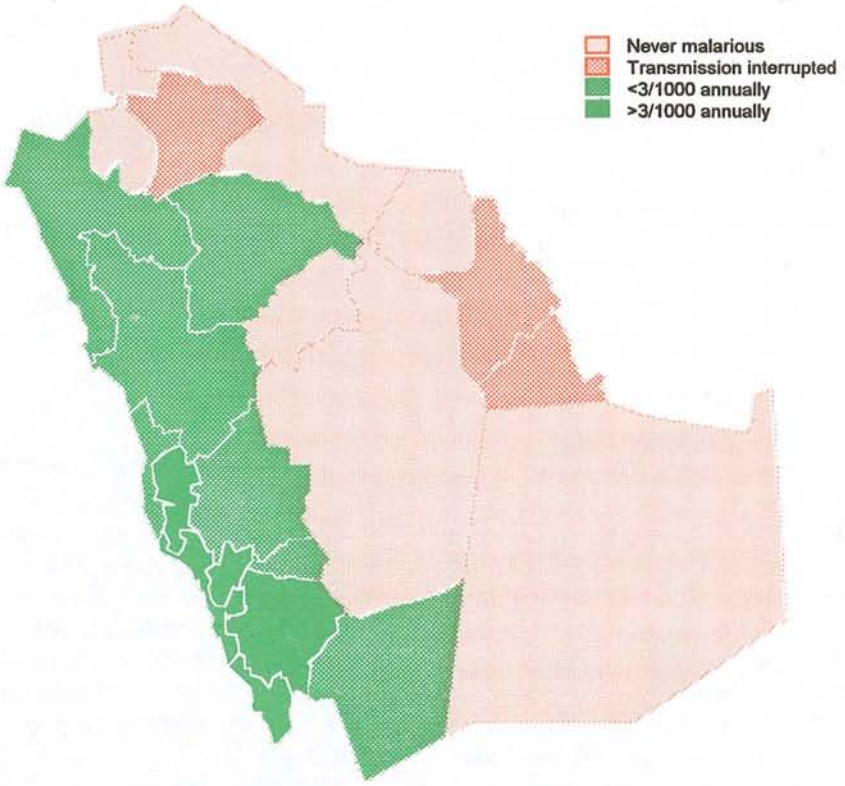
Malaria control activities were started in 1948 in the Eastern Province around the oil fields by ARAMCO oil company. A joint Saudi-World Health Organization pilot malaria control project was launched in March 1952. In 1956, national malaria control services were established. Instituted malaria control activities included spraying residential houses with D.D.T. in hyperendemic areas (annual parasite rate >10 per 1000 inhabitants). Mass drug distribution stopped in 1992. *An. arabiensis* developed resistance to D.D.T. in Gizan, and a switch to Fenitrothion was made in 1987. Other control measures include weekly application of larvicides (Temephos 500 EC), ultra low volume space spraying, and treatment of malaria cases.

Ongoing malaria control activities focus on evaluation of newly applied insecticides and evaluation of new biological control measures (susceptibility of the anophelines to adulticides, larvicides, chemical and bio-larvicides (e.g., indigenous fish species and *Bacillus thuringiensis*). Use of impregnated bed nets is being assessed. The susceptibility of *P. falciparum* to 4-aminoquinolines is periodically monitored. Training and refresher courses are organized in the Malaria Research and Training Center in Gizan to upgrade the quality of field operations. Trainees include medical officers, health inspectors, laboratory technicians and students in health institutes. Public health education is done to help encourage community participation in the malaria control program.

The progress made in malaria control in KSA has resulted in the interruption of malaria in the Eastern and Northern provinces since 1972 and in the greater parts of the Western province since 1970. Over 2.5 million people living in areas previously known to be malarious have been protected. In other areas epidemiological indicators have shown a substantial decline in the incidence of the malaria. The malaria surveillance system is credited with prompt control and investigation of emerging outbreaks of malaria.

—Reported by: Suleiman M. Al-Seghayer, MPH; Assistant Director General for Parasitic Diseases, Ministry of Health.

Figure. Malaria endemicity in Saudi Arabia, 1995.



Outbreak of Salmonella Gastroenteritis among Guests in 3 Wedding Halls, Riyadh, 1995

On November 12, 1995, a Riyadh hospital reported 18 patients with gastroenteritis. All had attended wedding receptions in three unrelated wedding halls on November 8. Other hospitals were alerted and by November 15, 83 gastroenteritis cases had been reported all among children below 12 years of age and females persons attending three wedding halls. *Salmonella enterica* (Serogroup D) was recovered from the stool of 25 of the 83 reported. Although the three wedding halls were unrelated, all served a buffet for females that had been prepared by one caterer.

A retrospective cohort study was conducted to determine the foods responsible for the outbreak. Gastroenteritis was defined as the acute onset of diarrhea (more than three loose stools per day) after attending one of the three weddings. From 265 families that attended these weddings, 49 were contacted. We determined gastroenteritis symptoms and food and drink consumed at the weddings for 284 family members in these families. Of the 284 persons, 92 (Attack rate=32%) developed gastroenteritis within 6 to 57 hours (median 18 hours) after eating at the wedding banquets. All developed diarrhea; 73% reported vomiting; and 90% had fever (mean 38.8°C); 65% had chills. The median duration for illness was four days (range 1-9 days). Symptoms developed in the 90% of the patients within 6 to 24 hours, and no longer than 2.5 days after the weddings. For all three weddings, gastroenteritis affected only those who attended the female and children's section and who ate from a supplemental buffet prepared by the caterer (AR = 45%). Attack rate among women and children was 35% in hall A, 59% in hall B, and 42% in hall C.

Comparison of food specific attack rates for foods served showed that eating cooked foods prepared by the caterer was associated with gastroenteritis (AR= 51%, Risk Ratio [RR]=14, 95% confidence interval [CI]=3.6-55≤0.0000). Food prepared by the wedding halls and salads and desserts from the buffet were not associated with gastroenteritis. The caterer served 24 cooked dishes. All nine food items made of ground mutton were associated with development of gastroenteritis with RR range from 15 (95%CI= 6.5-33) for edam mosaqaa to 8.1 (95%CI= 3.2-20) for the kobah (Table 1). Also ground chicken kofta and hot dog slices were associated independently with development of gastroenteritis illness with RR of 2.5 (95% CI = 2.0-3.3) and RR of 2.0 (95%CI=1.5-2.7) respectively. Guests who ate only from the 13 other cooked foods did not develop gastroenteritis.

The incubation period was increased by 4.9 hours for each addition food item consumed containing ground mutton (r = -0.63, 95% CI = -0.74, -0.48). The attack rate for developing gastroenteritis also increased as the number of food items containing ground mutton consumed increased from 8% for guests who ate cooked foods which did not contain ground mutton to 43% for one ground muttom item and reached 100% for four or more ground mutton items (P <0.001, Chi Square for trend).

Thawed ground chicken used to make the chicken kofta was first reground in meat grinder. The thawed (<1 hour) ground mutton was then run through the same meat grinder. All ground mutton was first cooked in single container and then used in preparing the nine different food items. After cooking, the ground mutton food items and other cooked foods were held at ambient temperatures for

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Table 1: Food specific attack rates among 233 guests (92 gastroenteritis cases) for cooked foods prepared by one caterer for three wedding halls on Wednesday, 8 November 1995. Riyadh, Saudi Arabia.

Food items	Ill (92)	Well (141)	AR	RR	95% CI
Food containing ground mutton (G.M)	**86**	**50**	**63%**	**10.2**	**4.7-22.4**
Edam Mosaqaa	9	1	90%	14.6	6.5-32.5
Kofta	25	5	83%	13.5	6.1-29.7
Kobaneah	13	4	76%	12.4	5.5-28.0
Mahshee Tomato	11	4	73%	11.9	5.2-27.3
Sambosa	40	16	71%	11.6	5.2-25.5
Mahshee Pepper	12	5	71%	11.4	4.9-26.3
Mahshee Kossa	42	21	67%	10.8	4.9-23.9
Fatera	11	6	65%	10.5	4.5-24.5
Kobah	8	8	50%	8.1	3.2-20.2
No food with G.M (Reference)	**6**	**91**	**6%**		
Food containing whole mutton @	**6**	**15**	**29%**	**0.7**	**0.4 - 1.4**
Food containing ground chicken @	**22**	**10**	**69%**	**2.0**	**1.5-2.7**
Kofta @	15	2	88%	2.5	2.0-3.3
Fatera @	8	8	50%	1.4	0.9-2.4
Boned Chicken @	**25**	**26**	**49%**	**1.3**	**0.9-1.9**
Whole Chicken @	**20**	**20**	**50%**	**1.3**	**0.9-1.9**
Fish @	**14**	**19**	**42%**	**1.1**	**0.7-1.7**
Other					
Hot Dog Slice @	21	9	70%	2.0	1.5-2.7
Macaroni @	18	32	36%	0.9	0.6-1.3
Pizza @	16	34	32%	0.8	0.5-1.2
Spanish Fatera @	1	9	10%	0.3	0.1-1.6
Cheese Fatera @	12	19	39%	1.0	0.6-1.6
Cauliflower @	11	34	24%	0.6	0.3-1.0

@ = Reference used are those who did not eat from that foods and its main ingredients.
NS= Statistically not significant.

Hepatitis A in Southwest Riyadh and Immune Globulin Administration to Household Contacts

In 1995 the epidemiologist at a southwest Riyadh hospital noticed an increase in the number of newly diagnosed cases of unspecified hepatitis. Although these cases were suspected of being hepatitis A (HA), serologic confirmation had not been done and immune globulin (IG) had not been administered to household contacts. An epidemiologic investigation was begun to identify the causative agent, estimate the size of the outbreak, and to identify the risk factors that were maintaining transmission in the area.

A case of hepatitis was defined jaundice and one or more of the following signs and symptoms: dark urine, nausea, vomiting, abdominal pain, or increased serum transaminase levels between October 1994 and November 1995 in a person living in southwest Riyadh. Confirmed HA was a hepatitis case with anti HA virus (HAV) IgM detected. Hepatitis cases were found by reviewing case reports in the selected primary health care centers (PHCC), logbooks in school health clinics, and private clinics. Additional unreported cases were found among family members of reported cases during household interviews. Exposure histories of confirmed HA cases were compared to control-persons selected by systematic random sampling of families registered at the PHCC in same catchment areas as confirmed cases.

From October 1994 to November 1995, 203 cases of hepatitis were identified and 154 (75%) had been reported through the Riyadh health surveillance system. Of 84 hepatitis cases with sera submitted for anti HAV IgM, 71 (85 %) were serologically confirmed as HA. All except two were Saudi children under 15 years of age (median 7 years). Interviews were completed for 114 hepatitis cases in 40 extended families with one or more confirmed HA cases. IG had not been administered in any of these families, and the secondary attack rate ranged from 10% to 75%. Up to 12 secondary cases occurred in one extended family. The median interval between the onset of the first hepatitis case in the family and the onset of symptoms in the next case in the same household was 21 days (range 1 to 42). The median duration between date of onset of symptoms and drawing the blood sample for HAV testing was 8 days (range 0 to 109). The delay between drawing the blood sample and reporting the result of the HAV test back to the selected PHCC was 36 days (range 8 to 89). This resulted in a median delay between onset and reporting of laboratory results of 51 days (range 18 to 374). Before July 1995, IG had not been given to family contacts because secondary cases had already occurred by the time the laboratory confirmation of HA was made. Thereafter, IG was given to family contacts of any hepatitis case when the patient first presented to PHCC with acute symptoms. Numbers of reported hepatitis cases decreased after this change in control (Figure).

Confirmed HA case persons were more likely to share glasses for drinking water with a known case of HA (OR 14, 95% CI 1.6-337). There were no differences between cases and controls by socioeconomic status, family size, education of parents, management of household sewage, household drinking water, or indicators of contact between children in the house.

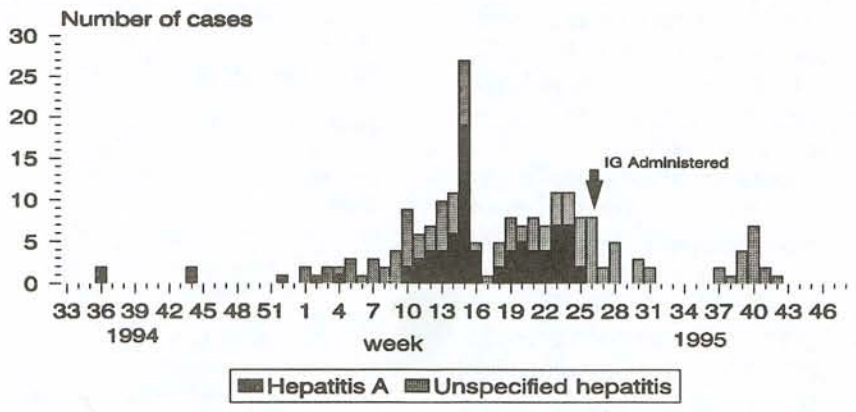
—Reported by: Faida M. Abu Al Jadayel and Hassan E. El Bushra

Saudi Arabian Field Epidemiology Training Program, Ministry of Health

Editorial note: Viral hepatitis is a reportable disease in the Kingdom of Saudi Arabia. Patients with clinical hepatitis are usually referred from PHCC to hospitals for laboratory investigations. Once HA is confirmed, the PHCC nearest to the residence of the patient is informed for institution of public health control measures. The propagated pattern, the high secondary attack rate and the lack of evidence for water or food borne transmission indicates that person to person spread was maintaining this out break. Since indirect or direct personal contact is difficult to manage within a household, effective control is accomplished through active or passive immunization. The MOH provides IG for passive immunization of household contacts as the principal control measure for HA. An effective vaccine is also available commercially for active immunization against HA.

In order to be effective IG must be given to contacts within 14 days of the onset of the first hepatitis case in the household. In this outbreak, the long delays between onset of illness and laboratory confirmation hindered effective use of IG to control secondary spread in families. Giving IG to family contacts before confirmation resulted in a decrease in hepatitis cases.

Figure. Hepatitis cases by week. Southwest Riyadh, 1994-1995.



Gastroenteritis from 3 Weddings

(Continued from page 5)

two hours at the caterer's establishment and in his delivery van. At the wedding halls they were kept warm on serving trays for another three to four hours before eating.

—Reported by Dr. Adel M. Turkistani and Dr. R.E. Fontaine, Field Epidemiology Training Program.

Editorial note: Salmonella is found in a wide variety of raw animal products including meat, eggs, and milk. Prevention of foodborne salmonellosis requires complete cooking to kill salmonella, clean utensils and surfaces to avoid cross-contamination of raw and cooked food, and storage of cooked food at temperatures above 60° C or below 4° C. In this outbreak all three of these preventive measures were violated.

Mark your calendar . . .

In the Kingdom

November 26-28: The Third Scientific Conference -Saudi Society of Family and Community Medicine. Primary Care in Saudi Arabia and the Obstacles for the Optimal Implementation. For more information write to PO Box 40161 Khobar 31952 Saudi Arabia. (03)899-0251.

Outside the Kingdom

August 27-30, 1996: The 14th International Scientific Meeting of the International Epidemiological Association -Changing Environment and Global Health Issues. Contact Secretariat of the 14th International Scientific Meeting of IEA, c/o Department of Preventive Medicine, Nagoya University School of Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya 466, Japan. (81)52-733-6729 (fax).

February 18-24, 1997: The International Clinical Epidemiology Network, the Field Epidemiology Training Programs, the Centers for Disease Control and Prevention, and the International Epidemiolotgical Association. Global Scientific Meeting, Hotel Equatorial, Penang, Malaysia. For more information write to: INCLEN Executive Office, 3600 Market Street, Suite 380, Phildelphia, PA 19104-2644, USA. Phone: (215)222-7700 Fax: (215)222-7741.

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Selected notifiable diseases by region, Jan-Mar 1996

	Riyadh	Jeddah	Makkah	Madinah	Taif	Asir	Gizan	Najran	Al Baha	Eastern	Al Ahsa	Tabuk	AlJour	Goriat	Arar	Hail	Qassim	Hafr al Batin	Bisha	Gonfuda	Total
Measles	43	235	12	182	6	31	59	2	1	19	4	4	1	1	1	24	20	5	1	0	651
Mumps	39	154	47	50	9	34	9	8	8	48	11	8	5	3	1	6	27	19	1	1	488
Rubella	14	33	1	2	1	11	2	1	0	9	8	0	0	0	1	0	12	4	0	1	100
Varicella	1755	1945	329	295	516	1219	125	451	105	1820	922	275	134	43	65	200	303	222	189	15	10926
Brucellosis	116	35	20	46	25	367	36	83	80	15	12	4	12	6	6	164	184	57	84	9	1361
Meningitis, mening.	1	3	0	1	1	1	0	0	0	0	0	0	2	0	0	0	0	0	1	0	10
Meningitis, other	29	12	2	6	7	4	6	1	0	8	7	2	0	0	0	4	4	13	0	0	105
Hepatitis A	49	74	111	127	0	135	48	48	9	24	17	11	2	7	2	19	60	16	8	0	767
Hepatitis B	66	178	64	30	9	69	6	7	69	106	11	7	0	1	1	12	33	12	5	8	694
Hepatitis, unspecified	22	96	26	40	0	41	70	11	0	2	5	11	0	0	0	21	0	0	0	0	345
Typhoid & paratyphoid	9	5	1	1	1	20	2	1	2	3	2	1	0	0	0	2	2	3	9	1	65
Shigellosis	31	27	3	6	0	28	15	23	0	62	4	57	0	0	0	0	5	23	0	0	284
Salmonellosis	82	50	1	5	0	22	5	10	12	166	2	29	0	0	0	0	17	14	0	0	415
Amoebic dysentery	12	1379	0	13	118	416	25	4	0	35	17	30	0	2	0	26	2	0	25	0	2104
Syphilis	8	33	1	0	0	4	2	3	0	22	7	0	0	0	0	1	0	0	0	0	81
VD, other	13	223	0	0	0	5	17	0	0	23	20	0	0	7	0	0	0	5	3	0	316

Comparisons of selected diseases, 1995-1996

	Jan-Mar 1995	Jan-Mar 1996	Change %	Jan-Mar 1995	Jan-Dec 1996
Diphtheria	0	0	0	0	1
Pertussis	1	7	-85	1	33
Tetanus, neonatal	6	11	-45	6	25
Tetanus, other	3	6	-50	3	14
Poliomyelitis	0	1	-100	0	3
Measles	651	432	51	651	2574
Mumps	488	317	54	488	1601
Rubella	100	67	49	100	385
Varicella	10926	9748	12	10926	35244
Brucellosis	1361	1467	-7.2	1361	5997
Meningitis, mening.	10	13	-23	10	58

	Jan-Mar 1995	Jan-Mar 1996	Change %	Jan-Mar 1995	Jan-Dec 1996
Meningitis, other	105	71	48	105	261
Hepatitis A	767	591	30	767	2697
Hepatitis B	694	655	6	694	3031
Hepatitis, unspecified	345	492	-30	345	1487
Typhoid & paratyphoid	65	83	-21	65	335
Shigellosis	284	228	25	284	1223
Salmonellosis	415	535	-22	415	2973
Amoebic dysentery	2104	832	152	2104	5949
Syphilis	81	92	-12	81	386
VD, other	316	168	88	316	961

Diseases of low frequency, Jan-Mar 1996

Pertussis: 1 (Najran 1)
Tetanus, neonatal: 6 (Makkah 3, Jeddah 2, Gizan 1)
Tetanus, other: 3 (Jeddah 3)
Rabies 4 (Eastern 4)
Plague, diphtheria, poliomyuelitis, viral enephalitis: No cases