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النشرة الوبائية السعودية
تصدرها وزارة الصحة
الوكالة المساعدة للطب الوقائي وبرنامج الوبائيات الحقلي
المجلد الثاني – العدد الثالث - يوليو، أغسطس، سبتمبر ١٩٩٥م

ACUTE TOXIC HEPATITIS AND ACUTE RENAL FAILURE IN DIABETIC PATIENTS USING RAW SHEEP BILE AS AN UNCONVENTIONAL REMEDY FOR DIABETES

Recent reports of acute hepatic and renal toxicity in Asians who drank raw bile from fish (grass carp) in the United States suggested a potential danger to diabetic patients in southwestern Saudi Arabia who had been advised by a traditional healer to drink raw sheep bile as a cure for diabetes. To assess the possibility that raw sheep bile was also toxic we investigated this practice in Al-Wadein village (population 5640) in the Asir.

From the 73 diabetic patients using two village Primary Health Care Centers, 30 who had used traditional medicine for diabetes were selected. We interviewed these diabetics about underlying illnesses, drinking bile and illnesses that immediately followed. From medical records we obtained serum chemistries taken over the year before the bile exposure (baseline), immediately after, and two months after bile exposure.

Fourteen of the 30 diabetics (age 53 to 78) who had used traditional medicine (including five on dialysis for chronic renal failure) reported that they had tried drinking sheep's bile to cure diabetes once during a four year period. This was done during the Eid holidays when slaughter of sheep was common and bile readily available. Two patients drank a minimum of 15 ml of bile while those who could tolerate it drank as much as 210 ml of bile over one to seven consecutive days. All 14 drank from 15 to 30 ml of bile after awakening in the morning before breakfast.

All 14 patients develqped nausea and anorexia immediately after drinking the bile and the 12 who drank more than 15 ml also developed vomiting with diarrhea (six with blood) within 36 hours. All remained afebrile. All 14 sought medical treatment and 12 were hospitalized for gastrointestinal symptomatology during the week after drinking bile. One became oliguric and another comatose but none died.

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Plasmodium malariae in Ahsa region, Saudi Arabia, 1994-1995

From January 1994 through May 1995, 45 cases of *Plasmodium malariae* were reported from Ahsa region. Ahsa was malarious before 1970 and since the vector, *Anopheles stephensi* is still present in the area, an epidemiologic investigation was begun. Initially, 17 smears were reviewed by a reference laboratory and the parasitologic diagnosis was reconfirmed on all smears. Nine cases were excluded from the investigation because eight were asymptomatic and were detected through residence permit screening immediately after arrival in Saudi Arabia, and one was from Gizan and could not be found for an interview.

The 36 symptomatic *P. malariae* cases occurred without seasonal pattern. They were scattered throughout the population centers of the Ahsa region. No cases occurred among permanent Saudi, Ahsa residents, or in nationals of non-malarious countries. All but two were nonprofessional workers.

Twenty-two cases (61%) had onset of fever in the first 70 days after arrival in Saudi Arabia, with 13 cases occurring before the first 20 days (minimum incubation period for *P. malariae*). Of these 22 cases, 19 (86 %) were Indian. For each case, five controls were selected from the residence permit list of the malaria center and were matched with case-persons by nationality, sex and age.

According to their passports, all Indian-cases had passed through Bombay in comparison to 73% of Indian control-workers selected at random from the resident permit list ($p=<0.05$,OR=16). Case-patients reported median staying from one to 90 days (median seven days) in Bombay compared to one to 14 days (median two days) for the controls who passed through Bombay ($p<0.001$, Kruskal Wallis test). These case-persons were not associated with any particular home state in India. The remaining 14 cases had onset after 70 days of arrival. We were able to contact and interview seven cases and 70 matched controls. *P. malariae* was associated with sleeping in an open field (odds ratio [OR]=16, confidence interval [CI]=1.2- 222), and a preference for injection for medical treatment (OR=undefined, CI=3.7-infinity).

-- Reported by Dr. Musaad Al-Sulaiman, Field Epidemiology Training Program

Editorial note: The course of *P. malariae* is not unduly severe but its long incubation period and persistence in a human host is notorious. Because recrudescence may occur as long as 52 years after exposure, it is difficult to determine if individual cases are acquired locally. Several findings in this investigation suggest that *P. malariae* with onset more than 70 days after arrival was recrudescent and not locally acquired in Ahsa. The cases did not cluster in time or location. There were no cases among permanent Ahsa residents or in nationals of non malarious countries. The time of exposure did not coincide with the expected season of transmission of malaria in Ahsa. Although two indirect indicators of vector borne or accidental malaria local transmission (sleeping outdoors and preference for injections) were suggested by the case control study, interviews were done up to 16 months after the illness onset, and results were subject to recall bias.

The association of imported cases (onset under 70 days after arrival) with Bombay, suggest foci of transmission in Bombay, India. The continuing importation and possible local transmission of *P. malariae* will require improved surveillance with prompt epidemiologic and case investigations to identify and control introduced malaria transmission in Ahsa.

Automatic conversion of Hejira dates to Gregorian dates in Epi Info version 6.02 software

In the past, a problem with using Epi Info for epidemiological analysis in Saudi Arabia has been the need to convert Hejira dates to Gregorian dates. Recently, this problem became critical when we decided to design a program for the Pediatric Nutrition Surveillance System (PedNSS) in the Primary Health Care Centers (PHCCs). The anthropometric program in Epi Info requires a Gregorian date to accurately calculate a child's age for calculating growth indices.

A formula written for Database was modified for use in the Check or Analysis program of Epi Info version 6.02.[1] This conversion gives an accuracy of +/- one day. This error is acceptable for most epidemiologic applications and is no worse than other conversion programs. To make the conversion, a Hejira date, month, and year should be separated as numerical variables e.g. ***Hejira Day ## Month ## Year ####*** in the questionnaire file. A date variable is also added to the questionnaire file as a place to put the result of the date conversion, e.g. ***{Greg}orian <dd/mm/yy>.*** After using ***ENTER*** to make a data entry file, the following formula should be added in a CHECK file:

```
Year
   LET Greg = "01/01/00"
```

(This sets 01/01/1900 as the starting reference point.)

```
   LET Greg = Greg +((year-1)*354.3848121+(month-1)*29.53206786+day-1)-466607.5
```

(This computes the number of days between the reference point and the Hejira date that is entered and automatically converts these days to the Gregorian calendar.)

```
   UPDATE
END
```

Since Hejira dates may be needed for administrative purposes, a new variable (e.g. S.Hejira <A >) (note: the Hejira variable has eight spaces) can be added to the questionnaire. The following command in a CHECK file will make a single string variable from the three numeric Hejira variables:

```
S.Hejira
   LET S.Hejira=(day+"/"+month+"/"+year)
   UPDATE
END
```

to display the Hejira date as a single string variable in the day, month, year format.

-- Reported by Dr. Adel M. Turkistani and Dr. Robert E. Fontaine, Field Epidemiology Training Program

Reference:

1. T.A. Mohammed. Date program conversion. Alam Al-Computer 1994;7(84): 28-30.

Bile induced toxic hepatitis

(Continued from page 1)

Stool cultures on 13 yielded no pathogens. After (<1 week) drinking bile mean serum ALT was 295 U/l compared to a baseline of 27 U/I (P<0.001, paired t-test). Serum bilirubin, AST and alkaline phosphatase showed similar elevations. Attending physicians evaluated all patients for hepatitis infection. However, anti-HAV IgM, HBsAg and anti HCV were all negative. Patients also showed evidence of renal toxicity. Following exposure, the mean BUN was 144 mg/100ml compared to a baseline of 77 mg/100 ml (P<0.001, paired t-test) and the mean creatinine was 6 mg/100ml compared to a baseline of 0.9 mg/100ml (P<0.001, paired t-test). Since several had underlying diabetic renal disease attending physicians had not further evaluated renal function. There was a dose response effect--both serum ALT (r=0.88, 95%, CI 0.76 to 0.96), and creatinine (r=0.63, 95% CI 0.34 to 0.81) increased in a direct linear relationship in proportion to an increasing dose of bile. (Figire 1.) All biochemical indicators of hepatic and renal toxicity returned to baseline levels two months after the acute illness. Mean baseline ALT, AST, alkaline phosphatase, serum bilirubin BUN, serum creatinine and blood glucose of the 14 who drank bile and 16 who did not drink bile were the same. All patients had stopped insulin or oral antidiabetic medications during the bile treatment. Following exposure, the mean blood glucose (random blood sugar) was 253 mg/100 ml compared to a baseline of 196 (p<0.01, paired t-test). However, serum glucose did not increase, nor was it otherwise related to the amount of bile drunk.

-- Reported by Dr. Mohammed S. Al-Qahtani, Field Epidemiology Training Program

Editorial note: The similarity of the reaction of these diabetic patients to sheep bile and the toxicity described from carp bile suggests a common toxic component of bile.[1,2] However, smaller quantities of carp bile than sheep bile were involved. The hepatotoxic and nephrotoxic components of carp bile have been shown to be in fractions of bile containing principally bile salts and bile acids.[3] In humans, bile salts will cause diarrhea in disease states that allow bile salts to reach the colon.[4] Hydrophobic bile salts are also known as cytotoxins.[5] Bile salts are conserved through an enterohepatic circulation with reabsorbtion in the ileum. Since they are recycled six times in 24 hours the total bile salt pool in the human is relatively small. Thus, the amounts of sheep bile drunk by these diabetic patients could potentially contain sufficient cytotoxic bile salts to overload this enterohepatic circulation and produce the observed toxicity.

As illustrated by this investigation, unconventional treatment can present a danger to the patient by direct toxicity. In this investigation the patients also neglected their prescribed antidiabetic medication, and demonstrated poor control of blood sugar. Even if unconventional medicines are effective the preparations are not standardized, so incorrect dosing is a common problem. The scope of this investigation was limited to only one village. However, the healer who promoted this remedy had disseminated his message widely. Thus, the sheep bile remedy and other potentially dangerous practices may be widespread. Physicians in Saudi Arabia should routinely question patients about unconventional therapy and report any suspect toxicity or other adverse events from these therapies. In order to define the extent of the bile remedy, physicians are asked to identify and report to the Ministry of Health any incident involving the drinking of bile for the treatment of any condition.

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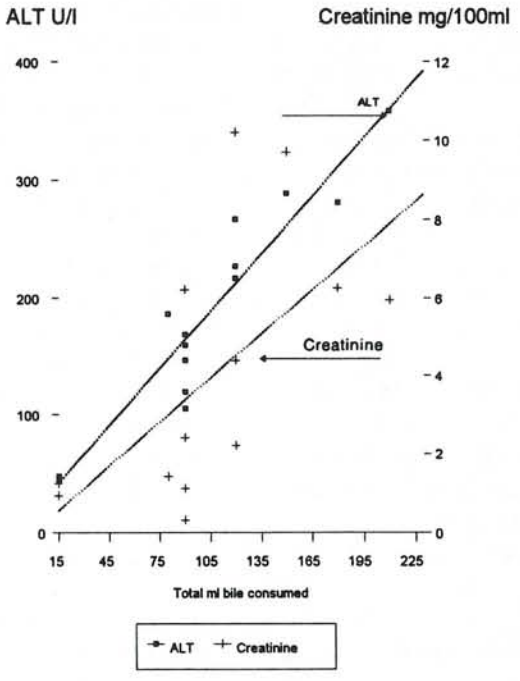
Figure 1. Increase in serum alanine aminotransferse (ALT) and creatinine relative to baseline by bile consumed by diabetic patients in Al-Wadein, Asir Region, Saudi Arabia.



Potential risk factors for acquiring cutaneous leishmaniasis in a hijra in Al-Majmaah

During 1994, the director of Majmaah General Hospital noticed an increase in the number of reported cases of cutaneous leishmaniasis (CL) and requested an epidemiologic investigation. Al-Majmaah district is located 200 km to the north west of Riyadh, with a population of more than 41000. It has one general hospital and 17 primary health care centers (PHCCs).

There were 377 CL cases in Al-Majmaah district in 1994, with an overall attack rate (AR) of 9.15 per 1000 population. Omrajowm had 44 cases with an AR of 110 per 1000 population, which was the highest among Al-Majmaah sub-districts. Omrajowm is a hijra or bedouin village of 400 persons living in 58 single floor houses. It was founded about 27 years ago and located 120 km east of Al-Majmaah city at the western border of the Al-Dahana desert. It has no farms. The hijra has minimal population movement. We visited all of Omrajowm's households and determined demographic information, clinical presentation of CL lesions and possible risk factors for CL through interviews. The case definition for CL was any person who had cutaneous lesion(s) that had been clinically diagnosed as a CL at any time in his/her life.

The first case of CL acquired in Omrajowm appeared in July 1992 in a 40 year old Saudi woman followed by 54 cases through July 1994 (Figure 1).

Mean age for the CL cases ranged from 1 to 70 years (mean 19.7), and 54% of cases were in women. Forty cases (74%) were Saudis, 3 (5.5%) were Palestinians, another three were Egyptians and eight (14.8%) were of other nationalities. CL is not statistically associated with occupation. The commonest site of lesions was the lower extremities (40.6%) followed by the upper extremities (39 .1%), the face (17.2%), and the trunk (3.1%). More than one lesion occurred in 28 cases (51.9%). Permanent scars were present in 98.4% of cases. Forty-nine patients (90.7%) had received chemotherapy, local healers treated two with cauterization and three persons denied receiving any type of treatment.

Of the 46 houses with cracked walls, 52% had one or more CL cases compared to 8% of 12 houses with no cracked walls (p-<0.05, Yates corrected Chi-square test). In the 12 houses adjacent to abandoned houses, one case or more of CL were found in nine (75%), whereas of 46 the houses not standing adjacent to abandoned houses, only 16 houses (34.8%) had one case or more of CL (p-<0.05, Yates corrected Chi-square test). Screened windows, the presence of animal enclosures attached to or nearby the house, or the presence of rodents indoors or outdoors were not statistically associated with the transmission of the disease. Questioning about knowledge of leishmaniasis revealed that 94.8% of the adult residents did not know how the disease occurs, or about the role of the sandfly and rodents in transmission.

Reported by Dr. Ali Al-Zahrani, Dr. Nasser Al-Hamdan, Field Epidemiology Training Program and Dr. Yasser Al-Ghamdi, Majmaah Hospital

Editorial note: Leishmaniasis is a group of infections of the viscera, skin, and mucous membranes caused by intracellular protozoan parasites of the genus transmitted by sandflies of the genus *Phlebotomous* in the Old World Zoonotic (ZCL) caused by *Leishmania major* is an increasing public health problem in the Old World, from Senegal in West Africa to the western parts of India.[1,2] Anthroponotic CL (ACL) is caused by *L. tropica* and occurs in urban centers in southwest Asia.

Both ZCL and ACL occur in Saudi Arabia, particularly in the Al-Hassa region in the east, in the Asir region in the south-west and in the Riyadh region in the central part of the country.[2,3] *P. papatasi* is the vector for ZCL and P. *sergenti* for ACL.[2,3]

In the Riyadh region, *L. major*, Montpellier zymodeme 26 (MON 26), has been isolated from several human CL cases, *P.papatasi* and from the rodent *Meriones libycus*.[3]

Reported cases of CL in the Riyadh region have gradually decreased from 1987 to 1991, when they began to increase through 1994. The same resurgent pattern has been found in the Al-Majmaah district which is a part of the region (Leishmaniasis department, MOH).

Because it is a zoonotic disease, ZCL spreads easily and is extremely difficult to control. Studies done elsewhere in Saudi Arabia have shown that CL occurs more frequently in males than in females; it is associated with working in farming areas with peak incidence in children below the age of 10 years.[4] In contrast in Omrajown, there were no associations of CL with age, sex, nationality or occupation. In addition the associations between the CL and cracks in the walls of houses and abandoned houses adjacent to the cases-houses, suggest that CL in Omrajown was recently introduced and that the transmission is domestic or peridomestic.

The only control measure that had been applied was fogging with insecticides. However, new CL continued to appear. The spraying may be ineffective because a) it is frequently done at midday, a time when the sandflies are in protected resting sites; b) respraying is not done frequently giving the sandflies a chance to recover, and c) the abandoned houses are not sprayed.

Another consideration is the lack of knowledge by the community about the disease and its way of transmission. The appropriate method to control the transmission of the disease in Omrajown will be to educate the people about the disease; to spray insecticides at short intervals and at the appropriate time; do something about the abandoned houses, and to close any cracks in the walls of houses.

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(Continued on page 5)

TUBERCULOSIS IN A PRISON, JEDDAH, SAUDI ARABIA, JULY 1993-MARCH 1995

After recognizing four extra pulmonary tuberculosis cases among prisoners hospitalized in a Jeddah hospital, an additional 40 prisoners with pulmonary TB were identified from TB surveillance between July 1993 to December 1994. An epidemiologic investigation was begun to determine the reasons for the large number of TB cases among prisoners in a prison in Jeddah.

The prison has two sections: a correctional institute and a general prison according to the crime. One clinic serves both sections of the prisons. prisoners with suspect TB are referred to the Jeddah TB Center for chest x ray, tuberculin skin testing, and sputum examination. Those with acid fast bacilli on sputum examination or *Mycobacterium tuberculosis* on culture are referred to the Chest Hospitals in Taif for treatment. New prisoners had not been screened for TB.

To find cases we reviewed patients' records of the two hospitals used by the prison and the Jeddah TB Center. A pulmonary TB case was defined as a prisoner who developed a cough illness with a sputum smear positive for acid fast bacilli and extra pulmonary TB cases as an illness with TB granuloma demonstrated by histology from July 1993 to March 1995. Prisoners with TB were interviewed in the prison or Chest Hospital.

From July 1993 to February 1995, 53 cases of pulmonary (49 cases) and extra pulmonary (4 cases) TB were diagnosed among prisoners (incidence rate= 456 per 100,000 per year). TB cases had been detected in the correctional institute since the beginning (July 1993) of the study period and the first seven cases had onset of illness more than one month after im prisonment (prison-acquired). In contrast, no TB cases had been discovered in the general prison during the first five months of the case review and one of the first three cases had onset less than one month after imprisonment (community-acquired). For all TB cases time between imprisonment and onset of symptoms ranged from 0 to 1162 days (median 216) and 87% were prison-acquired.

Referral from the prison clinic for TB diagnosis was delayed from 31 to 65 days (mean 54) after onset of cough. The risk of TB increased with crowding in the ward (Chi square for trend = 5.1, $p< 0.05$) and time spent in prison ($p< 0.01$, ANOVA). When compared to control prisoners selected at random from all prisoners, prisoners with diabetes mellitus (Odds ratio [OR]=16, 95% Confidence Interval [CI]7-37) and smokers (OR=2.9, 95% CI 1.1-7.9) had a greater risk of TB.

We screened 297 prisoners using chest film sputum smears and tuberculin skin testing. Prisoners were selected because they were in a ward with known TB cases or because they had a risk factor for TB (HIV positive, diabetes mellitus, chronic renal failure, hematological disorder). Twenty-six previously undetected TB cases were identified (8754 per 100,000). The mean PPD reaction was 7 mm with a range from 0-20 mm among all screened prisoners. The rate of tuberculin positively increased with increasing months of imprisonment (R= 0.27, 95% confidence limits 0.08-0.44). Screening of guards and social workers (18) detected one guard and one social worker from the Correctional Institute with pulmonary TB.

-- Reported by Dr. Nadir Hassan Al Shareef, Dr. Robert Fontaine, Field Epidemiology Training Program and Ilham Qattan, Diseases Control Department, Jeddah

Editorial note: Prisons throughout the world represent a situation where risk

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LEISHMANIASIS

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(Rodentia:Gerbillidae), a possible reservoir host of zoonotic cutaneous leishmaniasis in Riyadh province, Saudi Arabia. Trans R Soc Trop Med and Hyg 1994; 88:39.

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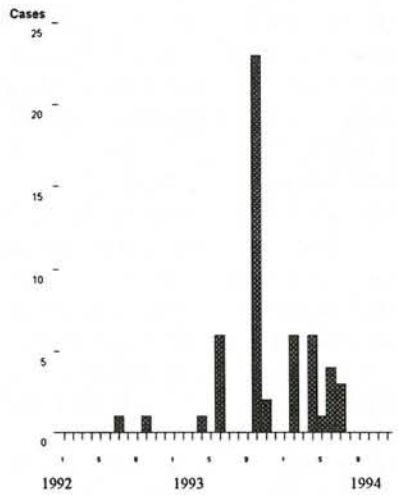
Distribution of CL cases by month of onset
Omrajown town, Al Majmaah, Riyadh region, KSA
1992-1994



AN OUTBREAK OF SALMONELLOSIS IN AN EXTENDED FAMILY IN RIYADH

On January 21, 1995, the preventive department in Al Malaz selected primary health care center in Riyadh city was notified by Al Salame primary health care center that 14 members of four related families (median age 23 years) with acute febrile gastroenteritis had been treated at the center. All had diarrhea, fever (median 38.8C), abdominal cramps, and 60% had vomiting. No one was hospitalized. Rectal swabs from the 10 patients yielded *Salmonella enteritidis*.

The four families had gathered at their grandfather's house on Friday, January 20, 1995. At 9 p.m. they brought four plates of chicken shwarma (equivalent to 40 sandwiches), mayonnaise, taheena and French fries for dinner from a restaurant. During the three days preceding the onset of illness they did not share any other meal. Of the 21 persons in the four families, 17 ate the food. Of the 17 who ate, 14 (82%) became ill from 6 to 21 hours (median 13) after eating. The four persons who did not share the meal remained well. The attack rate (AR) among persons exposed (eating shwarma) was 82%, and the AR among those who did eat shwarma was 0% (RR= INF; $p<0.005$, Fisher's exact test). There was no association of illness with eating any other food items at that Friday dinner. In addition, *S. enteriditis* was isolated from left over shwarma in the patients' house.

None of the restaurant staff had been ill but a rectal swab from one yielded *S. enteritidis* and nasal swabs from three *Staphylococcus aureus*. The restaurant inspection revealed that only one table was used for all food preparation such as cutting the raw chicken, preparing shwarmas, preparing hamburgers from raw meat, and mixing raw eggs for mayonnaise. The same utensils were used for all food preparations. Each day, 80 frozen chickens (each weighing 1300g) were thawed in two containers of water large enough to hold 40 chickens per container. The chickens were kept immersed in the water for 10 hours at room temperature to thaw. After thawing, the staff cut the chicken into small pieces, removed the skin and bones, and added salt and spices. The chicken was then stored in the refrigerator until the next day when the staff started putting the chicken on the spit. It took a total of 14 hours to thaw and prepare the chicken for cooking.

Reported by Mrs. Shadia K. Al-Sudany, Field Epidemiology Training Program

Editorial note: Chicken shwarma was the most likely vehicle of transmission of salmonellosis in this outbreak because all 14 persons who became ill had eaten chicken shwarma and there was no association of illness with any other foods served for the Friday dinner. Moreover, the ill people shared no other common meal during the four days (maximum incubation period for salmonellosis) before the outbreak. The isolation of *Salmonella enteriditis* from the leftover chicken shwarma also supports this conclusion. The relatively short incubation period (13 hours) and the high attack rate indicate that these people had been exposed to a relatively heavy dose (over 10^6 organisms per person) of *Salmonella* in the chicken shwarma.[1]

Salmonella may have originated in the frozen chicken or by cross-contamination or from surfaces contaminated by raw eggs, raw meat or other raw foods of animal origin. Since fresh and frozen raw chicken is often contaminated with *Salmonella* and since *S. enteriditis* frequently infects chickens and eggs, the chicken is the most likely source.[2] Proper preparation and cooking of chicken is therefore always necessary to assure safety of any food prepared from chicken. The infected food handler is a less likely source because he did not have diarrhea and was cultured after the outbreak. His asymptomatic infection may have resulted from eating the same chicken as the affected family.

Temperatures during the thawing, preparation and cooking were not measured but were probably within the range conducive for the multiplication of *Salmonella* (7 to 55 C).[2] Since Salmonella bacteria divide every 20 minutes, a single bacteria may multiply to a population exceeding 10^6 after six hours. Cooking at temperatures above 65C for 12 minutes will kill *Salmonella*[3,4] However, shwarmas are cooked only on the surface and the interior left under cooked. This family bought the equivalent of 40 sandwiches and we suspect that under cooked chicken below the cooked surface of the chicken on the shwarma spit was included in the shwarma bought by this family.

To prevent foodborne disease, emphasis should be placed on educating and training food handlers in proper food handling practices, appropriate thawing and cooking temperatures and times, and proper storage temperatures. Raw chicken should be either thawed at warm temperatures in less than one hour, or in the refrigerator at temperatures under 4C for longer periods. Preparation surfaces for raw and cooked food should be separate to avoid contact. Utensils should be washed after every use and not used for multiple purposes. Reporting outbreaks of salmonellosis and other foodborne infections helps identify and correct the type of incorrect foodhandling practices seen in this outbreak.

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TB

(Continued from page 5)

factors for acquiring tuberculosis (TB) are common and require special attention for TB control and prevention.[1,2] The prisoners themselves come from groups of people in the community that are at relatively high risk of TB. In the prisons relatively crowded conditions and close contact between prisoners increase the risk of TB transmission.[3]

Many of the factors previously noted in other prisons were demonstrated as contributing to TB in this prison. These include crowding and length of time spent in the prison, smoking, and underlying chronic disease.[4] Prisoners may have been selected from a high risk social groups in the community. However, this could not be shown because prisoners were not screened when first imprisoned. Transmission had probably been ongoing in the correctional institute before July 1993, whereas the community-acquired case among the initial cases in the general prison suggests that transmission there was more recent. The lack of screening of new prisoners and the delay of diagnosis of TB through the prison clinic were important contributors to this outbreak. In addition the screening indicated that there was a significant reservoir of undiagnosed pulmonary TB among the prisoners.

The problem of TB in prisons must be dealt with by increasing awareness of prison medical staff and authorities. To eliminate introduction of community-acquired TB, all new prisoners and prison employees should be screened with TB skin tests and chest x ray.[2] In prison , a more comprehensive screening of existing prisoners and staff is indicated. To prevent spread to the community, similar screening should also apply to released prisoners, especially those in contact with TB cases. Surveillance should include full access of prisoners to clinic services and heightened awareness of TB among clinic staff to promptly identify any new case of TB developing among prisoners. As soon as TB is diagnosed treatment should be started and contacts evaluated.

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Mark your calendar . . .

In the Kingdom

April 2-3, 1996: "Symposium on Recent Advances in Medical Microbiology and Infectious Diseases." Sponsored by the College of Medicine, King Saud University. Contact: Dr. Saleh S.A. Al-Hedaithy, Chairman, Organizing Committee, Symposium on Recent Advances in Medical Microbiology and Infectious Diseases, Postgraduate Center. College of Medicine, King Saud University, P.O. Box 2925, Riyadh 11461, Saudi Arabia. (01)467-1551/1554/1556/1564 (phone) or (01)481-1853 (fax).

May 25-30, 1996: "3rd Safety & Occupational Health Conference & Exhibition." Sponsored by the Chamber of Commerce and Industry in cooperation with King Faisal University, ASSE and AIHA. Contact: Saeed H. Al-Ghamdi, Chairman, Technical Committee, 3rd Safety and Occupational Health Conference & Exhibition, Chamber of Commerce & Industry, Eastern Province, P.O. Box 719, Dammam 31421, Saudi Arabia. (966)3 857-1111 ext. 3333 (phone) or (966)3 857-0607 (fax).

Outside the Kingdom

April 15-19, 1996: Field Epidemiology Training Programs' (FETP) Scientific Meeting; April 22-26, 1996: 1996 Centers for Disease Control (CDC) EIS Conference; April 23, 1996: International Night of the 1996 EIS Conference All three CDC sponsored events are to be held in Atlanta. Contact: Dr. Douglas Hamilton, International Branch, DFE, EPO, Mailstop C-08, Centers for Disease Control, Atlanta, Georgia 30333, USA. Electronic mail via Internet: DHH0@EPO.EM.CDC.GOV.

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).

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Selected notifiable diseases by region, April-June 1995

	Riyadh	Jeddah	Makkah	Madinah	Taif	Asir	Gizan	Najran	Al Baha	Eastern	Al Ahsa	Tabuk	Al Jouf	Goriat	Arar	Hail	Qassim	Hafr al-Batin	Bisha
Measles	53	123	36	203	41	101	2	1	14	31	15	66	112	0	21	45	131	15	0
Mumps	70	36	17	26	5	42	3	5	7	56	9	19	6	11	7	1	29	15	7
Rubella	6	10	2	13	9	35	0	2	0	12	2	1	0	0	1	1	7	1	1
Varicella	2035	557	231	527	316	1173	124	155	54	4495	1155	730	92	57	120	232	245	229	87
Brucellosis	193	21	21	75	82	307	26	137	58	37	21	12	47	14	14	232	287	130	148
Meningitis, mening.	3	4	0	4	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0
Meningitis, other	20	9	2	3	5	4	7	1	0	10	10	0	0	0	0	0	1	6	1
Hepatitis A	38	38	28	37	20	135	38	139	20	57	27	50	25	1	13	2	30	31	21
Hepatitis B	55	147	17	22	9	76	7	3	63	164	17	12	1	0	4	1	8	81	1
Hepatitis, unspecified	92	124	62	11	0	100	57	46	46	6	10	12	0	0	7	13	0	1	0
Typhoid & paratyphoid	11	7	4	4	0	12	2	1	0	20	1	0	0	0	0	2	3	17	5
Shigellosis	58	11	0	13	0	0	0	25	0	106	3	21	0	11	0	1	2	8	0
Salmonellosis	170	87	0	14	25	7	0	16	4	370	15	24	0	0	0	0	7	1	14
Amoebic dysentery	23	48	0	4	76	414	44	2	0	47	13	21	123	4	0	0	1	0	2
Syphilis	9	33	3	0	0	6	1	0	1	25	5	0	0	0	0	0	0	1	3
VD, other	7	20	0	0	0	11	23	1	3	22	39	0	0	7	0	0	0	4	3

Comparisons of selected diseases, 1994-1995

	Apr-Jun 1995	Apr-Jun 1994	Jan-Jun 1995	Jan-Dec 1994
Diphtheria	0	0	0	1
Pertussis	13	4	20	14
Tetanus, neonatal	5	6	16	33
Tetanus, other	4	3	10	16
Poliomyelitis	0	0	1	6
Measles	1010	343	1442	1253
Mumps	371	648	688	2276
Rubella	103	212	170	610
Varicella	12614	11134	22362	31708
Brucellosis	1862	1490	3328	4929
Meningitis, mening.	13	10	33	30

	Apr-Jun 1995	Apr-Jun 1994	Jan-Jun 1995	Jan-Dec 1994
Meningitis, other	79	118	150	377
Hepatitis A	750	608	1331	2485
Hepatitis B	688	964	1343	3826
Hepatitis, unspecified	587	651	1100	2582
Typhoid & paratyphoid	89	152	172	564
Shigellosis	259	145	487	844
Salmonellosis	754	367	1289	1723
Amoebic dysentery	822	1070	1654	4353
Syphilis	67	132	179	511
VD, other	140	287	308	1129

Diseases of low frequency, April-June 1995

Pertussis: 13 (Easterr 4, Asir 3, Gizan 2, Jeddah 2, Bisha 1, Gassim 1)

Tetanus, neonatal: 5 (Jeddah 3, Gizan 2)

Tetanus other: 4 (Jeddah 2, Gizan 2)

Yellow fever, plague, rabies, diptheria : No cases

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