Salmonella infection may be increased by alterations in the gastrointestinal tract, including decreased gastric acidity and chronic gastrointestinal disease. Leukocytosis was noted in 25% of patients. In fact, two thirds of the patients had a normal leukocyte count with immature leukocytes, which may be attributable to their relatively impaired cell-mediated immunity.

The predominant organism in this series was S. Choleraesuis, followed by S. Typhimurium. In Taiwan, the rate of resistance of S. Choleraesuis to ampicillin, chloramphenicol, or sulfamethoxazole-trimethoprim increased to approximately 90% for all 3 drugs and the rate of resistance to ciprofloxacin was from 7.7% to 59% (5–7). The resistance rate of S. Choleraesuis to ciprofloxacin in this study was similar to our previous report (7).

Nine of the 11 patients who completed follow-up information received appropriate antimicrobial drugs with drainage; however, 4 died. These 4 deaths (57%) were due to S. Choleraesuis-related empyema; 3 patients had underlying malignancy. Although appropriate antimicrobial drugs were used, our data suggest that more aggressive treatment with open drainage of the pleural effusion might have contributed to a better outcome than closed tube thoracostomy or simple thoracostomy alone. In contrast to S. Choleraesuis-related infection, all 4 patients with non–S. Choleraesuis-related thoracic empyema survived. One of these patients did not receive appropriate antimicrobial drug treatment, but did have adequate drainage with simple thoracostomy. This suggests adequate and aggressive drainage of pleural effusion may be as important as appropriate antimicrobial drugs. However, the overall death rate (36%) in this study was still higher than that of other reports (9). This might have been due to the high number of immunocompromised patients in this study.

In conclusion, thoracic empyema is a rare complication of nontyphoid Salmonella infection and is closely associated with an immunocompromised condition, even in patients <65 years of age. Higher rates of resistance and death were noted in patients with empyema thoracicus caused by S. Choleraesuis. Early diagnosis, appropriate antimicrobial drug therapy, and aggressive drainage are necessary to improve the outcome of patients with thoracic empyema due to S. Choleraesuis.

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References


Asymptomatic Yersinia pestis Infection, China

To the Editor: Plague is one of the oldest identifiable diseases. Modern public health measures and effective antimicrobial treatments have led to a decrease in plague cases worldwide. However, plague remains endemic in many natural foci. Since the early 1990s, the World Health Organization (WHO) has reported a steadily increasing trend in human plague cases, which has resulted in the recognition of plague as a reemerging disease (1). The emergence of antimicrobial drug-resistant strains of Yersinia pestis, along with an increasing number of plague cases, remind us that plague still poses a serious public health threat (2,3). In China, human cases of plague have been reported to WHO nearly every year from 1989 to 2003; these account for 9.5% of cases and 15.5% of deaths from this disease in Asia (1).

Human cases of plague in China are usually caused by contact with plague-infected rodents. Here, we report the results of a serologic survey by using 3 methods (passive hemagglutination assay, Western blot, and protein microarray analysis) in marmot hunters in Qinghai Province, China.

One hundred twenty serum samples were collected in 2 villages in
Huanyuan County, Qinghai Province, from marmot hunters (63 samples) and their family members (57 samples); none had a history of fever in the past 2 years. One hundred nineteen serum samples were collected from persons with no history of marmot hunting in 2 nearby counties in Qinghai Province in which plague was not endemic. Thirty serum samples were collected from persons in Beijing and used as negative controls.

All serum samples were initially screened with a passive hemagglutination assay to detect immunoglobulin (Ig) G antibody against F1 antigen of *Y. pestis*, by using a standard protocol (4). We then used an F1 antigen–based Western blot to analyze all serum samples. The protein microarray analysis was performed with 149 purified recombinant proteins of *Y. pestis* (5).

The results of the serologic survey are summarized in the Table. The passive hemagglutination assay showed 17 positive samples in the marmot hunter population. None of the control serum samples were positive for F1 antigen in this assay. Western blot identified 9 additional positive samples in the marmot hunter population, resulting in a seropositivity rate of 21.7% (26/120). We also found positive samples in 4 (3.4%) of 119 serum samples by using Western blot in persons from areas in which plague was not endemic. Identical results were also obtained by using protein microarray analysis, which validated the results of Western blot.

Previous studies have shown that plague antibodies were more prevalent in males in the exposed population. Thirty serum samples were collected from persons in Beijing and used as negative controls. The protein microarray analysis was performed with 149 purified recombinant proteins of *Y. pestis* (5).

The results of variations in exposure to the pathogen, not intrinsic factors (6,7). Our study showed that in the marmot hunter population, the plague seropositivity rate was significantly higher in males (36.8%, 25/68) than in females (2.0%, 1/52, p<0.01). Among the marmot hunter population, 63 (92.6%) of 68 males were hunters. Plague antibodies were also more prevalent in marmot hunters (39.7%, 25/63) than in their family members (1.8%, 1/57, p<0.01).

This is the first serologic survey of plague in the marmot hunter population. The plague seropositivity rate of 21.7% (26/120) in hunters and their families is much higher than the 3.4% (4/119) in the population from regions in which plague was not endemic (p<0.01). Seroprevalence in marmot hunters was even higher (39.7%), which suggests that marmot hunting is a risk factor for plague infection.

The marmot (*Marmota himalayana*) is the main host of *Y. pestis* in Qinghai Province. Plague-infected marmots are more easily captured by hunters. When persons hunt and butcher marmots without any effective protection, *Y. pestis* can be transmitted through tiny wounds in the skin, by bites of infected fleas, or by the respiratory route. Asymptomatic plague infection in marmot hunters might be explained by prophylactic use of antimicrobial drugs. Most hunters usually take sulfamethoxazole or tetracycline as a prophylactic measure. Even if the hunters were infected with *Y. pestis*, they would likely not develop symptomatic plague. However, if the antimicrobial drugs are not effective or hunters do not use prophylaxis, symptomatic infections will occur. Most reported human cases of plague in Qinghai Province were caused by hunting or butchering marmots, as shown by a recent outbreak of plague in October 2004 in Qinghai, in which 19 cases were reported and 8 persons died (M. Li et al., unpub. data).

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### References


### Table. Analysis of sera for plague antibody by 3 methods

<table>
<thead>
<tr>
<th>Method</th>
<th>Marmot hunter population, no. positive/no. tested (%)</th>
<th>Population from nonendemic areas, no. positive/no. tested (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>PHA</td>
<td>16/68 (23.5)</td>
<td>1/52 (1.9)</td>
</tr>
<tr>
<td>WB</td>
<td>25/68 (36.8)</td>
<td>1/52 (1.9)</td>
</tr>
<tr>
<td>PMA</td>
<td>25/60 (41.7)</td>
<td>1/52 (1.9)</td>
</tr>
</tbody>
</table>

*PHA, passive hemagglutination assay; WB, Western blot; PMA, protein microarray analysis.*

†These authors contributed equally to this study.
To the Editor: In May 2003, a previously healthy, 42-year-old rice farmer and miller, living on the Plain of Jars (Xieng Khuang Province) in northeast Lao People’s Democratic Republic (PDR) (Laos), dehusked and polished glutinous rice in her hand-operated rice mill. While milling, her hand slipped, removing the skin covering the interpharyngeal joint of her right index finger, on a dusty, wooden part of the machine. She did not recall the implantation of a wood splinter. During the following 4 weeks, multiple firm, erythematous lesions developed, which were not tender, fluctuant, or itchy, at the site of the injury and on the medial and anterior aspects of the lower and upper arm (Figure). The lesions spread proximally from the site of injury, but they remained confined to her right arm. She had no fever, and no lymphadenopathy developed. Her household had no domestic animals, including cats. No systemic disease developed, and she showed no evidence of immunosuppression, diabetes, or alcoholism. While waiting for a diagnosis, she persuaded a surgeon to excise all the lesions, but they soon recurred. She believed that the only solution would be to have her arm amputated. Initial biopsy specimens demonstrated no organisms and showed no growth on Sabouraud dextrose agar. Without facilities for further fungal diagnostic work in Lao PDR, but with a probable clinical diagnosis of sporotrichosis, we sent one of the excised lesions to Taiwan for molecular analysis by previously described methods (1,2). Polymerase chain reaction (PCR) was negative for mycobacteria but positive for Sporothrix schenckii, the cause of sporotrichosis, and the diagnosis was confirmed by sequencing the 18S rRNA gene, which showed 100% identity to that of S. schenckii (1,2). The lesions resolved with 6 months of oral itraconazole therapy (100 mg every 12 h).

S. schenckii is a dimorphic fungus found in soil, hay, decaying vegetation, and moss. Persons exposed to these environmental foci, such as farmers and gardeners, are especially at risk. Percutaneous inoculation is presumably the main method of infection, although inhalation and insect and mammal bites and scratches, especially from armadillos and cats, have been implicated (3,4). Our patient presumably contracted the fungus from the wood frame of the milling machine. In the 1940s, contamination from untreated wood was responsible for an epidemic that affected ≈3,000 gold miners in South Africa (from timbers in the mine). Lymphocutaneous sporotrichosis is the most frequent presentation, and the traditional treatments are oral saturated potassium iodide solution and local hyperthermia, but oral itraconazole for 3 to 6 months is now recommended (3,4).

Sporotrichosis has been described from North and South America, Europe, and Japan. In Asia and Australasia, it has been described from India (5), Taiwan (1), Australia (6), and Thailand (7), but apparently not from Laos, Cambodia, and Burma (Myanmar). Serologic evidence for human sporotrichosis infection is found in highland areas of southwest Vietnam (8). At least in part, the relative paucity of reports probably reflects the lack of sophisticated fungal diagnostic techniques in much of Southeast Asia. Some evidence

Figure. Lesions on the right arm of the patient.