allosaccharophila, Aeromonas bestiarum, Aeromonas media, Aeromonas salmonicida, and A. trota), in addition to the invalid species mentioned above. If the detection of these species independently of their pathological significance in clinical samples is the basis for inclusion, it would be appropriate to include these last 5 species, but then Aeromonas encheleia and Aeromonas eucrenophila, isolated from an ankle fracture and a wound, respectively, would be missing [7]. Aeromonas salmonicida was classified by Janda and Abbott [7] under the group of environmental species. However, similar to the experience of other investigators, we encountered this species with a low incidence in feces of patients with diarrhea [9]. We agree, therefore, with the authors of the list published in Clinical Infectious Diseases [1] that these species should be included. In view of these comments, it is important that the list as published be amended in order to be properly updated.

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Klebsiella pneumoniae Necrotizing Fasciitis Associated with Diabetes and Liver Cirrhosis

SIR—We read with interest the two articles in Clinical Infectious Diseases on the association of necrotizing fasciitis and Klebsiella pneumoniae liver abscess by Hu et al. [1] and Dylewski and Dylewski [2]. Although K. pneumoniae is a common co-pathogen in patients with polymicrobial necrotizing fasciitis, the bacterium is rarely the sole cause of this serious soft tissue infection [3]. As was pointed out by Hu et al. [1], all 4 patients who had liver abscesses and fascitis also had diabetes. Therefore, an awareness of the spectrum of underlying diseases in association with monomicrobial klebsiella fasciitis is important. We describe a case of K. pneumoniae necrotizing fasciitis and spontaneous bacterial peritonitis in a patient with diabetes and cirrhosis.

A 52-year-old man with diabetes and hepatitis B–related liver cirrhosis (Pudge-Child grade C) was admitted for evaluation of fever, and abdominal pain and distension. Physical examination revealed stigmata of chronic liver disease, ascites, and mild diffuse abdominal tenderness. There was no evidence of infection elsewhere. Abdominal centesis on the day of admission was unsuccessful. Ultrasonography of the hepatobiliary system revealed no evidence of liver abscess or biliary infection. Blood culture was obtained and he was given empirical treatment with cefoperazone/sulbactam for presumed spontaneous bacterial peritonitis. Two days later, blood culture yielded K. pneumoniae that was susceptible to cefoperazone/sulbactam, amoxicillin/clavulanate, and ofloxacin. Screening for production of extended-spectrum β-lactamase was negative. Culture of ascitic fluid obtained on day 6 was negative, but there was an increased polymorphonuclear leukocyte (PML) count of 129 × 10⁹/L, a very low protein level of 5 g/L, and a low pH of 7. The patient’s condition improved over the next few days and the antibiotic was continued for a total of 10 days.

Four days later, the patient was readmitted for evaluation of a 2-day history of left leg pain, erythema, and swelling. His temperature was 35.6°C; pulse rate, 140; and blood pressure, 67/33 mm Hg. There were multiple patches of purpuric lesions over the right groin, left calf, and mid-thigh. Skin necrosis was present in one of the mid-thigh lesions. Emergency surgery revealed necrotizing fasciitis, and extensive debridement was performed. Parenteral antibiotics including amoxicillin/clavulanate and ofloxacin were administered. Cultures of blood and tissue specimens from the calf both yielded a pure growth of K. pneumoniae. The antibiogram and biotype of this isolate were identical to that of the isolate 10 days earlier. The patient’s condition deteriorated further despite intensive therapy, and he died 5 days later.

Both diabetes and liver cirrhosis predisposed our patient to serious bacterial infection. Patients with diabetes have elevated rates and severity of K. pneumoniae infections, including pneumonia, bacteremia, hepatic abscess, and meningitis [4–6]. In—
Infections are also frequent in cirrhotic patients because of their defective defense mechanisms. The most important is spontaneous ascitic fluid infection, which has a prevalence of 10%–27% among cirrhotic patients who are admitted to hospitals with ascites. This type of infection is usually caused by an enteric bacterium, such as Escherichia coli or Klebsiella species. In one-third to one-half of the cirrhotic patients with spontaneous ascitic fluid peritonitis, bacteremia was also present [7]. Diagnosis of spontaneous ascitic fluid peritonitis is made on the basis of clinical features, an increased ascitic fluid PML count (> 250 × 10⁶ cells/L), and/or positive ascitic fluid culture.

Spontaneous ascitic fluid peritonitis cannot be definitely confirmed in our patient because a sample of the ascitic fluid was not available before administration of antibiotics. However, spontaneous ascitic fluid peritonitis is likely because of the abdominal signs, absence of an evident intra-abdominal surgically treatable source of infection, abnormal parameters in the ascitic fluid (day 6), and absence of another source for the K. pneumoniae bacteremia. Our case is unique in that a rare complication occurred shortly after an apparently adequate course (10 days) of treatment. During the second hospital admission, there was no evidence of recurrent spontaneous ascitic fluid infection or liver abscess. The bilateral necrotizing fasciitis in the lower limbs, therefore, could be spontaneous or more likely a result of metastatic seeding during the earlier ascitic fluid infection–related bacteremia.

Monomicrobial K. pneumoniae necrotizing soft tissue infection is rare. In addition to the 4 reported cases in diabetics with liver abscess, 2 cases have been described in association with bacteremia and advanced cirrhosis [1,2,8]. Data from the small number of reported cases thus suggest that monomicrobial K. pneumoniae is associated with diabetes and cirrhosis. When K. pneumoniae bacteremia occurs in these patients, clinicians should be vigilant about metastatic soft tissue infections.

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Handwashing—The Semmelweis Lesson Misunderstood?

SIR—Larson’s excellent review about hand hygiene [1] highlights the persisting reluctance in the United States to use alcohol-based hand disinfection for preventing the spread of hospital-acquired microorganisms. This letter is intended to provide some further clarifications about Semmelweis’ discovery to introduce hand disinfection and not hardwashing into clinical practice.

More than 150 years ago, Ignaz Semmelweis (1818–1865) demonstrated that puerperal fever was a contagious disease caused by infectious organisms, which were spread from patient to patient via the hands of health care workers (HCWs). This discovery led to the introduction of hand dips with chlorinated lime at Vienna General Hospital. Since then, many scientists have cited Semmelweis’ observations, but, amazingly, grossly misleading impressions still arise about Semmelweis and his original idea of antiseptic hand disinfection, often wrongly cited as “handwashing” in the English-language literature [2–4]. In fact, Semmelweis never promoted handwashing with soap and water; he was opposed to it, since he wrote: “The cadaveric particles clinging to the hands are not entirely removed by the ordinary method of washing the hands with soap…. For that reason, the hands of the examiner must be cleansed with chlorine, not only after handling cadavers, but likewise after examining patients” [5].

Nowadays, sink-based handwashing with water and antiseptic soaps could reduce the risk of cross-transmission. However, many studies have shown that compliance of HCWs with recommended handwashing practice remains unacceptably low; in the range of 10% to 50% [6]. Noncompliance is even higher in intensive care units (ICUs), during procedures that carry a high risk for contamination, and during periods of increased workload [7]. As recently suggested by Pittet et al., full compliance with handwashing guidelines seems unrealistic. The average hand wash duration in routine patient care ranges between 8 and 20 seconds, which is too short for most handwashing agents to be fully effective [7, 8]. Moreover, HCWs need 40–80 seconds for fully effective handwashing [7, 8]. It is not possible to promote handwashing at the sink and returning to their workplace.