Renal diseases that occur in association with infectious diseases have been decreasing in number especially in the temperate zones. Infectious diseases such as dengue hemorrhagic fever, typhoid fever, leptospirosis, lepromatous leprosy, malaria, and schistosomiasis, are unique to the tropics (1).

Typhoid fever caused by Salmonella typhi was once a major public health problem in several developing countries. However, the number of typhoid patients has been dramatically declined in the past two decades because of the improvement of community sanitation and hygiene (2). Typhoid glomerulonephritis is a rare complication affecting 2 to 4% of typhoid patients in endemic areas and in subjects traveling from endemic regions (2). To date, several cases have been reported in tropical regions such as Thailand, India, Turkey, Israel and South Africa (2–10). In addition to the clinical symptoms of typhoid fever (pyrexia >39°C, anorexia, nausea, vomiting, abdominal pain with constipation or diarrhea, splenomegaly and initial leucopenia), patients with renal complications usually show edema for prolonged periods, macro- or microhematuria, and proteinuria. In some patients signs of volume overload associated with hypertension are present (11). Renal biopsy examination shows mesangial or endocapillary proliferation. Immunofluorescent microscopy reveals granular deposits of IgM, traces of IgG, and C3 in the mesangium, sometimes along the glomerular capillary wall. Salmonella Vi antigen is detectable in some glomeruli (10). Clinically, the observations are compatible with a transient mild glomerular disease with rapid recovery; the hematuria and proteinuria usually disappear within 2 to 3 weeks, but in others the symptoms may persist for at least 4 weeks (11).

Hayashi et al reported a 45-year-old woman who presented macroscopic hematuria, proteinuria and transient renal insufficiency as well as high fever and malaise (12). Salmonella typhi was isolated from her blood and stool culture and then she was diagnosed as typhoid fever. She was treated with levofloxacin resulting in gradual improvement of her symptoms, fever, renal function and proteinuria. She had never been abroad and had no contact with people who had a history of traveling to an endemic area. Therefore this case was a domestically acquired typhoid fever with acute nephritic syndrome.

In Japan, the cases of acute glomerulonephritis remarkably decreased in number in the 1980’s and afterward it’s frequency has been stable, namely, 1 to 2% of all kidney diseases (13). On the other hand, according to the Infectious Agents Surveillance Report in Japan, fourteen to nineteen cases of typhoid fever have been reported since 2001 (14); interestingly, 47–58% of those cases were domestically acquired.

As far as I investigated, in Japan, kidney disease associated with typhoid fever has been reported in only one paper by Hoshino et al (15). They retrospectively investigated 130 patients with typhoid fever presenting to the Tokyo Metropolitan Komagome Hospital during the period 1975–1998. Out of 130 patients, 3 patients had complicated acute renal failure. However, no glomerulonephritis was described as a complication. Therefore, the patient reported by Hayashi et al is the first case of acute glomerulonephritis associated with typhoid fever in Japan. Typically when we see the patient who presents high fever and acute nephritic syndrome, we first consider an ANCA (antineutrophil cytoplasmic autoantibody)-associated necrotizing glomerulonephritis and vasculitis as a differential diagnosis. Without realizing that typhoid fever could occur domestically in Japan and also it is possible to complicate glomerulonephritis in a typhoid patient, reaching a correct diagnosis would be very hard. Hayashi et al (12) described in their report that the diagnosis for typhoid fever was relatively difficult because of its low incidence of classical symptoms and signs such as relative bradycardia, rose spots and splenomegaly and that it took about two weeks until their patient was diagnosed as having typhoid fever. Hoshino et al reported that relative bradycardia occurred in 48% of such patients, and rose spot and splenomegaly are found in only 26% and 24%, respectively (15). Therefore, they emphasized that medical professionals should consider typhoid fever for all febrile patients regardless of other symptoms. The diagnosis was delayed for an average of 14 days after the disease onset in their study, interestingly the same as the case reported by Hayashi et al (12). Hoshino et al discussed that the delay in diagnosis was composed of two factors: one was the late presentation to the hospital consultation by the patient, and the other was the delay of diagnosis after the physician had seen the patient (15). Physicians should be keen to reduce the latter period by suspecting the disease and appropriately culturing specimens for the bacteria.
The case reported by Hayashi et al (12) is the first case with typhoid glomerulonephritis in Japan. It emphasizes the basic issue that infectious disease can be a cause of acute nephritic syndrome. We should remember a typhoid fever when we see a febrile patient with acute nephritic syndrome even in patients who have no history of traveling to endemic regions.

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References