A Patient with Japanese Spotted Fever Complicated by Meningoencephalitis

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Introduction

Many unclear points remain in the pathology and differences in Japanese spotted fever from other rickettsial infections. We report a case complicated by meningoencephalitis.

Case report

A 77-year-old man was admitted August 14, 2000, for a 2-day history of spiking fever. Cefmetazole was administered, because of suspected cholangitis, but the fever did not subside, and disturbance of consciousness and convulsions appeared. Encephalitis was suspected, and he was transferred to our department August 17. On physical examination, he was in a semicoma (Japan coma scale III-100). The thoracoabdominal region appeared normal and superficial lymphonodes were not palpable. Generalized maculopapular erythema and an eschar on the left shoulder skin were noted.

Laboratory results were as follows: WBC count 11,180/µl (neutrophil 81% monocyte 3.9% lymphocyte 14.0%), RBC 372 × 10^6/µl, Hb 11.2 g/dl, Ht 33.5%, Platelet 13.7 × 10^6/µl, BUN 21.4mg/dl, Creatinine 0.95mg/dl, GOT 122 IU/l, GPT 56 IU/l, LDH 720 IU/l, AIP 339 IU/l, γ-GTP 306 IU/l. Total bilirubin 0.8 mg/dl, Cholinesterase 0.35 AolH, CRP 7.1mg/dl, Fibrin degradation product (FDP) 9 µg/ml, NH₃ 20 µg/dl, Arterial blood gas pH 7.398, Cerebrospinal fluids (CSF) : protein 52 mg/dl, glucose 68 mg/dl (blood glucose 132 mg/dl), Cell 103/3 (Neutrophil 64%, Lymphocyte 27%), Adenosine deaminase (ADA) 2.5 IU/l. EEG showed 3-4 Hz slow waves, suggesting encephalopathy (Figure).

Starting with an antiepileptic drug for treating convulsions, intravenous administration of minocycline 200mg/day was begun, considering rickettsiosis based on the fever, exanthema, and eschar. His conscious gradually improved and fever decreased after 2 days. Slight fever due to encephalitis disappeared in early September and he was discharged on September 5. Specific antibodies were detected by immunoperoxidase reaction using *Rickettsia japonica* Aoki strain.

Serum IgG titer was × 2,560 and IgM titer was × 2,560 on hospital day 17 during recovery, showing
Fig. EEG indicates 3–4 Hz slow waves on admission

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an increase. The specific antibody in the CSF was IgG < 10 on hospital day 7, but was weakly positive converted to × 10 on hospital day 20.

Many serious cases of tsutsugamushi disease and Rocky mountain spotted fever are complicated by meningoencephalitis, but few reports have described Japanese spotted fever complicated by encephalitis. CSF during meningitis in rickettsiosis showed a mononuclear cell-dominating increase in cell count, but dominance of multinucleated cells was also observed in some cases. CSF glucose was reported to exceed than 40mg/dl, in many cases showing a normal or slightly lower level, and differential diagnosis from tuberculosis and viral meningitis is important. In the CSF in this patient, the glucose level was slightly lower than the normal range, excluding viral meningitis, and cytosis dominated by neutrophils was observed. The cell count was dominated by lymphocytes in recovery (cell 33/3, neutrophil 12%, lymphocyte 61%). ADA in the CSF did not increase, excluding tuberculosis. FDP was normal, indicating no hypercoagulability, and acidosis was not observed. NH₃ was also normal, excluding the possibility of encephalopathy accompanied by DIC or metabolic disorder. Since symptoms subsided during administration of minocycline, we diagnosed meningoencephalitis due to Japanese spotted fever. It should be noted that encephalitis complications make the pathology of the patient more complex, which may delay diagnosis. In pandemic areas, rickettsiosis should be considered in differential diagnosis of nonbacterial meningitis.

References

髄膜脳炎を合併した日本紅斑熱症例

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