Positive Culture from Normal CSF of Streptococcus pneumoniae Meningitis

We successfully treated a non-immunocompromised patient with meningitis due to Streptococcus pneumoniae whose initial lumbar puncture was normal. A 52-year-old male farmer was admitted because of general malaise and shaking chill (body temperature 39.3 °C) with headache and nausea. He had been in good health and the family history was negative. The bilateral tenderness in the costovertebral angle was noted. He was fully conscious. No signs of meningeal irritation nor focal neurological deficits were noted. Urinalysis revealed numerous white blood cells and bacteria (Pseudomonas aeruginosa). A lumbar puncture yielded a clear colorless cerebrospinal fluid (CSF) containing no red blood cells, six white cells/mm³ (polymorphonuclear:mononuclear = 98:2), 26 mg/dl of protein and 71 mg/dl of glucose. The circulating blood count showed leukocytosis (27,800/mm³) with a shift to the left. Intravenous cefmenoxime (2 g/day) was initiated for suspected pneumococemia. He became gradually disoriented, accompanied by dilated pupils and Kernig’s sign. The brain CT was normal. The second CSF obtained 12 h after the initial one contained 2,709 white cells/mm³ (polymorphonuclear:mononuclear = 98.2), 360 mg/dl of protein, 13 mg/dl of glucose and numerous gram-positive diplococci. The CSF reacted with the latex coated with the antipneumococcal polysaccharide antibody. In spite of 24 million units/day of intravenous penicillin-G, the patient’s consciousness declined further to deep coma. Streptococcus pneumoniae was cultured from the CSF obtained at the initial and the second lumbar puncture and from the initial blood cultures. Penicillin-G was switched to ampicillin (15 g/day plus rifampin 450 mg/day according to the sensitivity test). He regained consciousness and was discharged without deficit.

Bacterial meningitis which was already present in the lumbar region, not introduced by the initial lumbar puncture. On the other hand, the patient complained of headache beforehand, which suggests that a meningeal irritation was already present in the cranium. Seemingly, it takes some time for the inflammatory reaction to spread from the intracranial cavity to the lumbar subarachnoid space. Okabe et al. [10] reported a similar case and which was already present in the lumbar region, not introduced by the initial lumbar puncture. In our patient, the ‘normal’ CSF obtained at the initial lumbar puncture contained a clear colorless cerebrospinal fluid (CSF) containing no red blood cells, six white cells/mm³ (polymorphonuclear:mononuclear = 2:4), 26 mg/dl of protein and 71 mg/dl of glucose. The circulating blood count showed leukocytosis (27,800/mm³) with a shift to the left. Intravenous cefmenoxime (2 g/day) was initiated for suspected pneumococemia. He became gradually disoriented, accompanied by dilated pupils and Kernig’s sign. The brain CT was normal. The second CSF obtained 12 h after the initial one contained 2,709 white cells/mm³ (polymorphonuclear:mononuclear = 98.2), 360 mg/dl of protein, 13 mg/dl of glucose and numerous gram-positive diplococci. The CSF reacted with the latex coated with the antipneumococcal polysaccharide antibody. In spite of 24 million units/day of intravenous penicillin-G, the patient’s consciousness declined further to deep coma. Streptococcus pneumoniae was cultured from the CSF obtained at the initial and the second lumbar puncture and from the initial blood cultures. Penicillin-G was switched to ampicillin (15 g/day plus rifampin 450 mg/day according to the sensitivity test). He regained consciousness and was discharged without deficit.

References