Case Report

An autopsy case of Group A Streptococcus meningoencephalitis

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Received 7 February, 2015; Accepted 9 June, 2015

Group A *Streptococcus* (GAS), which frequently colonizes the oropharynx, causes significant morbidity worldwide due to invasive infections such as pneumonia, necrotizing fasciitis, bacteremia and streptococcal toxic shock syndrome. However, this organism does not usually invade the central nervous system. Here, we report the autopsy of an adult Japanese patient who died from a fulminant infection, and examination of her cerebrospinal fluid and blood cultures showed GAS.

Keywords: Group A streptococcus, Meningitis, Cerebrospinal fluid and blood cultures.

INTRODUCTION

Group A Streptococcus (GAS) frequently colonizes the oropharynx and is a major cause of global morbidity, causing invasive infections such as pneumonia, necrotizing fasciitis and streptococcal toxic shock syndrome (Poradosu and Kasper, 2007; Carapetis et al., 2005). However, GAS does not usually invade the central nervous system and causes infections such as meningoencephalitis (Brouwer et al., 2012; Chaudhuri et al., 2008) and the organism accounts for less than 2% of all systemic streptococcal infections (Lamagni et al., 2008). Similar to other countries (Schlech et al., 1985; Davies et al., 1996), only a few cases of adult GAS meningoencephalitis have been reported in Japan. Therefore, the clinical picture and epidemiological features of this disease are unclear. Here, we report the autopsy of an adult Japanese patient who died from fulminant infection and examination of her cerebrospinal fluid and blood cultures showed the presence of GAS.

CASE HISTORY

A 48 year old Japanese woman who had visited Guam

for 4 days experienced fever and vomiting during her return flight to Japan. On the day of arrival in Japan, she visited the emergency department of her local community hospital. Her temperature, blood pressure, and SpO₂ were 38.5°C, 105/33 mmHg, and 97% (room air), respectively. She was subsequently discharged with a painkiller and antiemetic drugs. The next morning, she was found lying dead in bed. Her past medical history unremarkable was except for an episode of pyelonephritis 4 months before her death. In addition, she had no previous history of immunodeficiency.

Computed tomography findings

Postmortem computed tomography (CT) examination was performed on the day of her death. No abnormalities were seen on head CT (Figure 1).

Autopsy findings

After an external examination had been conducted by the

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Figure 1. Postmortem head CT image revealing no remarkable changes.

police, a judicial autopsy was performed the day after her death. The skin of her face, neck, and anterior chest was reddish, showing putrefactive networks (Figure 2). The surface of the brain was markedly hyperemic but no cloudy abscess was seen. The severely softened brain barely kept the original shape (Figure 3). The other organs showed no remarkable macroscopic or microscopic changes including her kidneys, skins or meninges. Culture of the blood and cerebrospinal fluid (CSF) revealed GAS-positive colonies (Figure 4). The white blood cell count of the CSF was 80 cells/mm³ with 75% neutrophils. To ascertain the cause of death, we performed microscopic examination of the brain tissue. Gram staining of brain tissue sections revealed grampositive cocci in chains (Figure 5).

DISCUSSION

GAS meningoencephalitis is rare. According to van de

Beek et al. (2002), the incidence of GAS meningoencephalitis in adults was 0.03 cases per 100,000 persons, which remained constant during the period studied. Unlike meningoencephalitis caused by Haemophilus influenzae and Streptococcus pneumoniae, considered secondary to bacteremia, the clinical picture and epidemiological features of GAS meningoencephalitis have not been well studied. In addition, the specific contributing risk factors for GAS meningoencephalitis are unclear. GAS meningo-encephalitis is associated with various underlying diseases (85.3%) including upper respiratory tract infections such as otitis media or sinusitis, and upper respiratory tract infections are considered to be the major cause (44.1%) (van de Beek et al., 2002).

In the present case, the patient experienced pyelonephritis 4 months before her death, which might have caused GAS meningoencephalitis. The clinical symptoms of GAS meningoencephalitis in adults include fever (89%), headache (68%), neck stiffness (76%), focal



Figure 2. Redness with putrefactive networks on the neck and anterior chest.



Figure 3. Appearance of the surface and frontal section of the severely softened brain.

neurological deficits (36%), seizures (32%), and coma (11%) (van de Beek et al., 2002), which are similar to those of adult meningoencephalitis caused by other

organisms. In the present case, the patient only had fever and vomiting, so it would be difficult to diagnose meningoencephalitis instead of infectious enteritis in the



Figure 4. Culture of the cerebrospinal fluid revealing GAS-positive colonies.



Figure 5. Histological examination with Gram staining of the hippocampus revealing grampositive cocci in chains.

absence of other symptoms.

Conclusion

Clinicians should be aware that sporadic GAS infections with a fulminant course might occur. In addition, forensic and anatomical pathologists need to study more cases of GAS meningoencephalitis in order to clarify the clinical picture and epidemiological features of this rare infection.

Conflict of interest

Authors have none to declare.

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