Olfactory Neuropathy in Severe Acute Respiratory Syndrome: Report of A Case

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Abstract- This case was a 27 years old female with severe acute respiratory syndrome (SARS). She suffered from typical symptoms of SARS. Although she got almost complete recovery from most symptoms after treatment, she noted acute onset complete anosmia 3 weeks after the onset of her first symptom. Her brain MRI examination did not show definite lesion except an incidental finding of left temporal epidermoid cyst. Her anosmia persisted for more than 2 years during following up. Peripheral neuropathy and myopathy have been reported as a concomitant problem during the convalescent stage of SARS, while the sequel of permanent anosmia in SARS was not reported before. Olfactory neuropathy, which rarely occurred in typical peripheral neuropathy, could be a special type of neuropathy induced by corona virus infection in SARS. Olfactory function test should be taken into routine check-up for patients with SARS. The pathophysiology and therapeutic strategy of this special type of permanent olfactory dysfunction deserve further investigation.

Key Words: SARS, Anosmia, Neuropathy

INTRODUCTION

The patient was a 27-year-old female, who was working as a nurse in the hospital that cared patients with severe acute respiratory syndrome (SARS) during the outbreak in Taipei City in the Spring of 2003. She suffered from the symptoms of SARS, including: fever, cough, headache, myalgia and diarrhea in April, 2003. Under the diagnosis of SARS she was admitted to an isolation room for further treatment. Her serum test for the antibody against corona virus showed positive conversion after admission, which further confirmed the systemic infection of SARS. During the hospitalization, she had intermittent fever, cough, headache, and severe diarrhea. Her symptoms of upper respiratory tract improved two weeks after the therapy with antiviral agent (Ribavirin) and steroid (prednisolone). However, this patient’s fever and diarrhea persisted for about three
weeks. Then, one day, she suddenly noted complete anosmia. She totally lost her smell function to all kinds of orders in both sides of her nasal cavity. An otolaryngological consultation did not show any abnormal finding in her nasal cavity. Under the impression of acute olfactory nerve inflammation, nasal spray steroid via nasal spray and oral vitamin B12 were prescribed. The patient recovered from most symptoms of SARS two months latter. However, her complete anosmia persisted during more than two years of follow-up.

Her laboratory data showed: mild leukopenia with white blood cell of 1710, and lymphocyte percentage of only 17% on admission. The patient got rapid recovering of leucopenia and lymphopenia to normal level two days latter. Biochemistry examination data were within normal limit. Serology tests for HIV virus, hepatitis B, hepatitis C virus and herpes virus were negative. In the meantime, one of her roommate colleague was even admitted to intensive care unit for ventilator supporting therapy, and also diagnosed to have SARS by positive serum antibody test.

The four limbs peripheral nerve conduction study of this patient did not show any abnormal finding, except bilateral carpal tunnel syndrome. Her brain MRI examination showed no definite lesion in the subfrontal area, nasal cavity and paranasal sinuses. There was an incidental finding of left temporal small epidermoid cyst in the brain MRI (Figure). However, this is apparently not the cause of her anosmia.

This patient was followed up at outpatient clinic for more then two years. The symptom of anosmia did not show any change. MRI followed up six months after the onset of the disease showed no any new lesion except for the previous finding of an epidermoid cyst in the left temporal area. There was no interval change.

**DISCUSSION**

SARS is a new disease that was announced by WHO in April, 2003. At that time, the first outbreaking of this disease affected more than 30 countries and regions on five continents. According to the WHO report, there were totally 8422 cases with 916 deaths during that outbreak around the world\(^1\)\(^-\)\(^4\). People of all ages were affected, predominant female adults. Health care workers were at high risk and accounted for one-fifth of all cases\(^4\). The etiology of SARS was a new variant of corona virus, which was mostly transmitted through upper respiratory tract by contaminated spreading mucus droplets. The typical symptoms include: high fever, dry cough, dyspnea, and even acute respiratory failure\(^5\)\(^-\)\(^6\). Meanwhile, the rare concomitant symptoms of skin rush,
headache, vomiting, diarrhea, myalgia have been reported (7-9).

Tsai et al (10) reported 4 cases of SARS with concomitant four limbs peripheral neuropathy and/or myopathy. Among these, two experienced motor neuropathy, one was myopathy, and another was neuro-myopathy. All these patient’s neuromuscular problems developed approximately three weeks after the onset of SARS. Their cerebrospinal fluid study disclosed normal protein content, absence of pleocytosis and SARS corona virus antibody.

The common etiologies of olfactory dysfunction (11) have been excluded in this patient. Otolaryngological consultation did not find any abnormality in the oral and nasopharyngeal mucosa. Brain MRI showed normal appearance in the nasal cavity, paranasal sinuses, and subfrontal area. This patient did not have any past history of significant head injury. All the drugs used for this patient usually did not cause neuropathy of olfactory dysfunction.

Though acute olfactory neuropathy has been found in some types of viral infection (11,12), the complication of acute olfactory neuropathy in patients with SARS was not reported before. This patient’s acute anosmia occurred about three weeks after the onset of first symptom related to SARS. The time course was compatible with the previous study of SARS-related peripheral neuropathy. This supported the notion that the acute anosmia could be a corona virus-related olfactory bulb or olfactory nerve damage. According to the delay onset of anosmia during the course of SARS, autoimmune system should have played a role in its pathogenesis, and this damage could be nonreversible.

The study of cerebral spinal fluid (CSF) was not arranged for this patient because of the acute suffering of this patient during the SARS, and because of the relatively trivial evidence of her CNS infection. Meanwhile CSF study has been reported to be normal in the previous reported SARS-induced peripheral neuropathy (16).

There is no definite and convenient electrophysiology test for the evaluation of olfactory nerves. Meanwhile, olfactory function was not routinely examined by clinicians. Though olfactory neuropathy is not common in typical polyneuropathy, it could be a special neuropathy induced by corona virus infection and could cause permanent damage. Olfactory function test should be taken into routine work-up for patients with SARS during the acute and convalescence stages. The pathophysiology and therapeutic strategy of this special type of severe olfactory dysfunction requires investigation.

REFERENCES