

Wernicke's encephalopathy in a patient with masticator and parapharyngeal space abscess: a case report

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Abstract (J Korean Assoc Oral Maxillofac Surg 2016;42:120-122)

Wernicke's encephalopathy is a fatal neurological disease caused by thiamine deficiency. Many reports indicate that Wernicke's encephalopathy is caused by malnutrition. We report the case of a 79-year-old female patient who had a left masticator space and parapharyngeal space abscess who was diagnosed with Wernicke's encephalopathy. She reported problems while eating due to the presence of the abscess, but the true quantities of food she was ingesting were never assessed. Clinicians have a responsibility to provide adequate nutritional support by ensuring that patients receive adequate nutrition. Clinicians should also keep in mind that Wernicke's encephalopathy may occur in patients who experienced prolonged periods of malnutrition.

Key words: Wernicke encephalopathy, Malnutrition, Masticator space and parapharyngeal space abscess, Parenteral nutrition

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I. Introduction

Wernicke's encephalopathy is an acute neuropsychiatric disorder due to the lack of thiamine (vitamin B1), generally occurred in chronic alcoholics¹. In addition to alcoholism, other causes include continuous long term administration of glucose-only intravenous (IV), high calorie administration by parenteral nutrition, hyperemesis gravidarum, starvation of long duration, hemodialysis, gastrointestinal surgery, and cancer².

The characteristics are ophthalmoplegia, ataxia and mental status changes³. If a patient with Wernicke's encephalopathy remains untreated, severe amnesic deficits, Korsakoff psychosis, and death could result from the acute disease⁴.

Recently there are many studies showing that Wernicke's

encephalopathy develops in other diseases, for example malnutrition, malabsorption, AIDS, and malignant tumors, which result in thiamine deficiency⁵.

This case shows that poor oral intake and malnutrition due to masticator space and a parapharyngeal space abscess resulted in thiamine deficiency which led to Wernicke's encephalopathy. We report the case with a literature review and discuss Wernicke's encephalopathy and the management of patients with eating difficulty.

II. Case Report

A 79-year-old female patient with hypertension was referred to the oral and maxillofacial surgery department of Sanggye Paik Hospital because of severe pain and swelling of the left cheek and difficulty of opening the mouth that had been present since about 10 days prior to presentation. She had received treatment at a local clinic, but the patient's symptoms did not improve.

We confirmed that the patient had pain, swelling, and induration of the left cheek, including the left temporalis area. She could open her mouth only about 20 mm. Her vital signs were unstable (systolic blood pressure, 70 mmHg; diastolic blood pressure, 30 mmHg; pulse rate, 110 beats/min; respiratory rate, 28 breaths/min; body temperature, 37°C). The pa-

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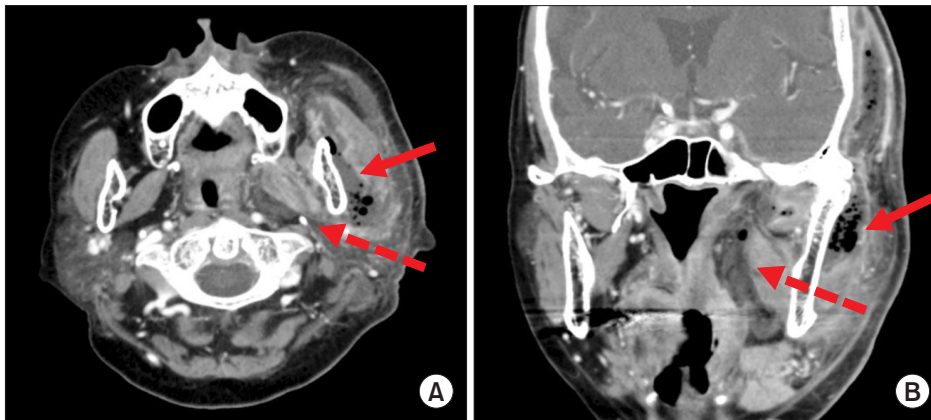


Fig. 1. Computed tomography with contrast enhancement shows an abscess in the the left masticator space (arrows) and parapharyngeal space (dotted arrows). A. Axial view. B. Coronal view.

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tient did not complain of dysphagia or dyspnea. On computed tomography with contrast enhancement, an abscess was detected in the left masticator space and parapharyngeal space. (Fig. 1)

The patient's condition improved after incision, drainage, and antibiotic therapy. After 2 weeks, the patient complained of nausea and vomiting even after simply drinking water. Her condition was improved after an additional incision and drainage of the palatal area. However, the patient was unable to eat meals normally. The patient's state of malnutrition continued and her electrolyte levels remained unstable. In particular, her potassium levels varied from 2.6 to 3.3.

After 2 more weeks, we consulted with the department of neurology because the patient displayed sudden confusion and seizures. She was tentatively diagnosed with Wernicke's encephalopathy by magnetic resonance imaging.(Fig. 2) Her condition improved after receiving IV thiamine. She was ultimately diagnosed with Wernicke's encephalopathy. We suspect that prolonged malnutrition due to abscess formation resulted in thiamine deficiency, in turn inducing Wernicke's encephalopathy.

III. Discussion

Thiamine plays an important role in glucose metabolism and the production of energy⁵. If it is depleted, cerebral hypoperfusion and functional disorders in the blood brain barrier may occur within 2 weeks⁵. The diagnosis of Wernicke's encephalopathy is based on clinical features and fast reversal of symptoms after the administration of thiamine⁶. However, in clinical situations, acute Wernicke's encephalopathy is often difficult to detect⁶.

Magnetic resonance imaging can be useful for diagnosis⁷, as it shows T2 signal hyper-intensity in the pulvinar of the thalamus, hippocampus, and mammillary body, and in the



Fig. 2. Magnetic resonance imaging shows characteristic Wernicke's encephalopathy with T2 signal hyper-intensity in the periaqueductal area (arrow).

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periaqueductal area^{3,6,8}. Bilateral paramedian thalamic infarction, cytomegalovirus encephalitis, primary cerebral lymphoma, multiple sclerosis, Behcet's disease, central pontine myelinolysis or extrapontine myelinolysis, Lyme disease, mitochondrial disorders such as Leigh's disease, and variant Creutzfeldt-Jakob disease should be considered in the differential diagnosis⁷. If the patient's symptoms improve dramatically after supplementation with thiamine, his or her condition may be confirmed as Wernicke's encephalopathy⁶.

Because Wernicke's encephalopathy can be avoided by the administration of thiamine supplements, Lindberg and Oyler⁹ recommended that all comatose patients with unknown cause of coma should receive parenteral thiamine supplementation before definite identification of thiamine deficiency.

The administration of thiamine supplements, either orally or parenterally, is recommended in patients with alcoholism for the prophylaxis of Wernicke's encephalopathy. Oral thiamine supplements are recommended for patients who are at low risk of Wernicke's encephalopathy¹⁰.

One of the responsibilities of clinicians is to provide adequate nutritional support¹¹. It is standard practice to assess electrolyte levels, but clinicians are likely to overlook the quantities of meals that patients actually ingest. The patient's state of nutrition should be confirmed not only by lab findings, but also through interviews with both the patient and (if relevant) guardians. If patients have difficulty swallowing, additional treatment should be offered such as the placement of a Levin tube or provision of total parenteral nutrition.

Our case illustrates that Wernicke's encephalopathy may result from malnutrition due to the presence of abscesses in the masticator space and parapharyngeal space. Clinicians should keep in mind that patients who have difficulty eating due to conditions affecting the oral cavity are at risk of Wernicke's encephalopathy due to malnutrition.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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