—Case Reports—

A Case of Meningeal Carcinomatosis Presenting with the Primary Symptoms of Facial Palsy and Sensorineural Deafness

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Abstract

We report the case of a 59-year-old man with meningeal carcinomatosis (MC) who presented with peripheral facial palsy and progressive sensorineural deafness. The patient had been operated on for gastric cancer 1 year previously, and no metastases had been detected in the retroperitoneum or thorax at follow-up examination 1 year later. However, he developed headache, deafness, and peripheral facial palsy and was referred to us for further evaluation, as magnetic resonance of the head had shown no abnormalities. Ramsay Hunt syndrome was suspected, but no increase in the cerebrospinal fluid cell count was detected. On the other hand, the balance test suggested a central disorder. In addition, the plasma level of carcinoembryonic antigen suddenly increased, suggesting MC. The cerebrospinal fluid was examined several times; in the end malignant cells and an increase in the cell count were detected, and the diagnosis of MC was established.
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Key words: meningeal carcinomatosis, sensorineural deafness, peripheral facial palsy

Introduction

Meningeal carcinomatosis (MC) is a rare metastatic solid tumor. Malignant cells infiltrate the subarachnoid space and cerebral pia mater causing meningeal irritation and cranial nerve symptoms. Gastric, breast, and lung cancers are often causes of MC. MC is an endocranial metastasis that develops during the treatment of a primary carcinoma. Thus, physicians usually treat the primary carcinoma and, during follow-up, evaluate the patient for possible endocranial metastasis.

The deafness and facial palsy caused by MC are often evaluated in the department of otolaryngology. Because otolaryngologists are usually not given sufficient information about the primary carcinoma, they may have considerable difficulty diagnosing MC. We report on a patient with MC who had been referred to evaluate the chief complaints of peripheral facial palsy and deafness.

Case Report

The patient, a 59-year-old man, had experienced both headaches and hearing problems since the end of July 2005 but had not sought treatment. He noticed right facial palsy on August 14, 2005, and

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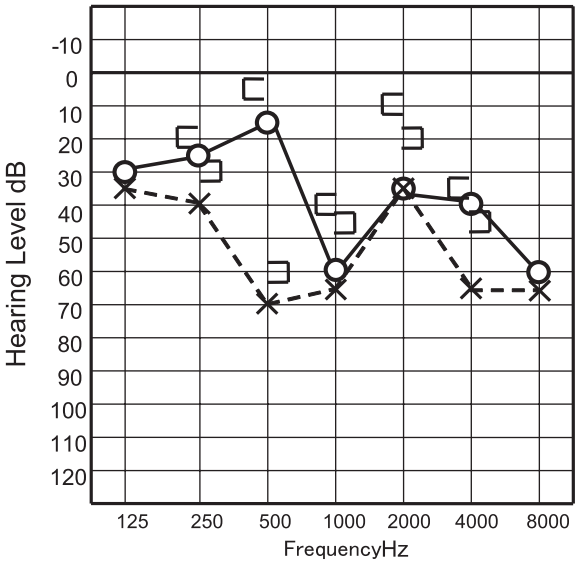

Fig. 1 Sensorineural deafness with laterality was accepted in audiogram in initial diagnosis of a case by both ears. The left ear audibility turned worse with course and became deaf. The right ear passed without a change.

was examined at the neurosurgery department of our hospital on August 17. He underwent computed tomography and magnetic resonance (MR) of the head but was referred to us on August 18 because the examinations had revealed no abnormalities. The patient was not aware of the vertigo associated with right peripheral facial palsy and lateral sensorineural deafness, but when he turned to the right, horizontal positional nystagmus was recognized (**Fig. 1**).

The patient had undergone pyloric gastrectomy for gastric cancer (stage IIIA, pT2N2M0) at our hospital in May 2004, and neither thoracic recurrence nor abdominal metastasis was detected on computed tomography in August 2005. In addition, carcinoembryonic antigen (CEA) was 17.2 ng/m*l* in June 2005.

On the basis of the patient's symptoms—peripheral facial palsy, sensorineural deafness, and dizziness—we first suspected Ramsay Hunt syndrome. We believed the headaches had been caused by localized cerebritis due to herpes zoster virus; therefore, we examined the cerebrospinal fluid (CSF) but found no elevation of cell counts. Electroneurography performed on the ninth day after the onset of symptoms showed a 62% degeneration of the facial nerve. As for examination of optokinetic nystagmus, development was poor by balance test, and eye tracking test findings showed center disorder in saccade together. The right-sided sensorineural hearing loss did not improve and instead progressed to complete deafness. The headaches could initially be controlled with nonsteroidal anti-inflammatory drugs, but the pain became so severe that morphine was required on August 29. In addition, on August 27 the plasma level of CEA suddenly rose to 1,460 ng/m*l*. Because we suspected MC, we re-examined the CSF but found only minor leukocytosis. However, on September 1 neoplastic cells (adenocarcinoma) were detected in the CSF, and MC was diagnosed (**Fig. 2**). An MR exam performed on September 2 revealed localized enhancement of both internal acoustic meatuses (**Fig. 3**). The patient became unconscious, and died on October 6, 2005.

Discussion

Because treatments for primary malignant tumors have become more effective, patients now survive longer. On the other hand, the possibility of MC developing in any systemic organ increases with the lengthened patient survival. The main metastatic sites for malignant tumors are the lymphatic tissue, lungs, and liver. However, metastatic brain tumors and MC have also become more common.

Saenger[1] in 1900, was the first to report MC presenting as sudden progressive bilateral hearing loss in a patient who had been operated on for gastric cancer. Maddox[2] described a case of metastatic temporal bone tumor in 1967, and thereafter temporal bone was recognized as a potential site for distant metastasis. Metastatic intracranial abscesses causing deafness, vertigo, and facial palsy have been reported in patients with temporal bone tumors and MC.

The clinical manifestations of MC are protean, and MC is often diagnosed after symptoms have progressed and the patient shows a disturbance of consciousness. Thus, patients have a poor prognosis and usually live no longer than 5 to 6 months under expectant management[3,4].

Headache is the most common symptom, but

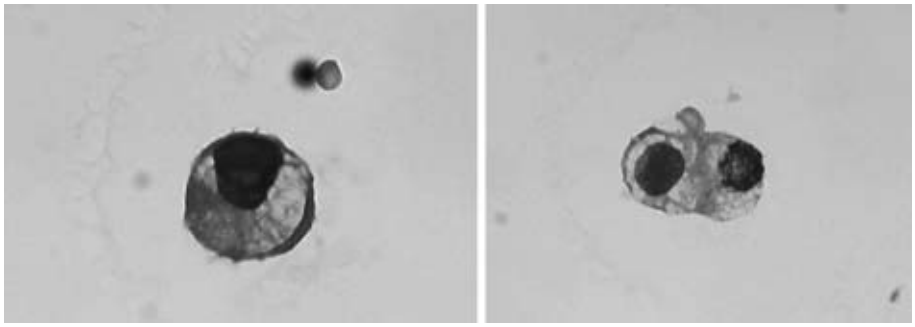

Fig. 2 Cytology of the cerebrospinal fluid showed atypical cell.

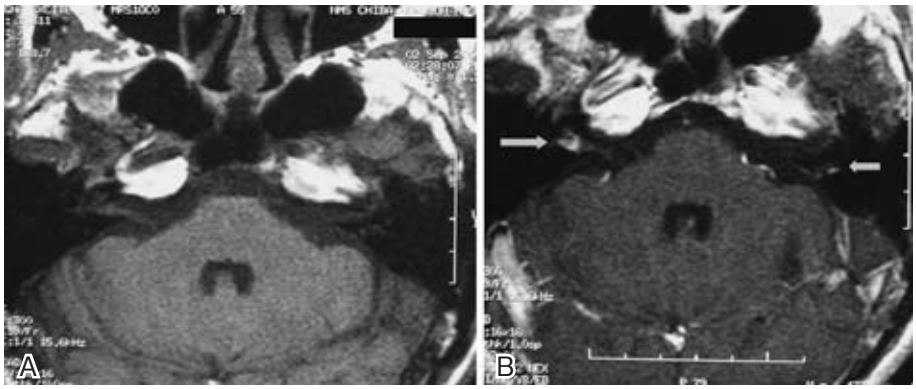

Fig. 3 **A**: In simple MRI, we did not accept findings. **B**: In Gadolinium contrasting MRI, high signal level was accepted by both internal acoustic meatus and thought with invasion of a tumor.

patients also present with various symptoms caused by cranial nerve involvement, such as nausea, emesis, vertigo, deafness, and peripheral facial palsy. Headaches are usually less severe with meningeal irritation than with purulent meningitis, and neck pain and head retraction tend not to appear at an early stage. Severe headache was a presenting symptom in the present case, but as there was no meningeal irritation, determining their cause was difficult[5,6]. Headaches and deafness have been recognized as primary symptom, followed by facial palsy for 2 weeks, and then VIII and VII cranial nerve palsies developed and agreed with a report of primary symptom of MC on our patient[7–9].

CSF examination is important for the diagnosis of MC, which causes increases in CSF pressure, CSF cellularity, and albumin levels and decreases in glucose levels. Moreover, the presence of malignant cells in CSF is necessary for diagnosis. However, malignant cells are detected on initial examination in less than 50% of cases. Thus, multiple convolutions must be examined. MC was not detected on the initial CSF examination in the present case, but acute increases in malignant cells and in the cell count were found on the third examination. There was a very short interval between the second and third CSF examinations. Perhaps malignant cells had multiplied rapidly in the subarachnoid space and entered the CSF. Thus, CSF examination is important for the diagnosis of MC.

However, the probability that neoplastic cells will be detected in the CSF is low, and other examinations are necessary. MR examination has been proposed as method of diagnosing MC. Morgan et al.[10] have reported two cases of MC in which gadolinium-enhanced MR revealed diffuse enhancement of both internal acoustic meatuses before CSF abnormalities were detected. The authors noted that gadolinium-enhanced MR was the most sensitive and least invasive neuroradiological imaging tool available to detect MC. No abnormalities were detected with simple MR in our

patient at an early stage, when cranial nerve palsy developed. Nevertheless, T2-weighed gadolinium-enhanced MR revealed the involvement of both internal acoustic meatuses. Neoplastic cells had likely infiltrated the internal acoustic meatuses, causing facial palsy, sensorineural deafness, and vertigo.

Thus, MR is an important diagnostic tool to identify lesions in the internal acoustic meatus, and gadolinium enhancement is necessary to detect malignant transformation of tissue. In contrast, simple MRI is of no diagnostic value in patients with suspected MC.

When patients have a history of malignant tumor and present symptoms of severe headaches and internal acoustic meatus neuropathy suggesting MC, CSF examination is imperative. In addition, gadolinium-enhanced MR should be performed to aid diagnosis. MC is a metastasis of a malignant tumor with an extremely poor prognosis. Therefore, when a patient with a history of a malignant tumor presents with symptoms of internal meatus neuropathy, such as sensorineural deafness, vertigo, and peripheral facial palsy, and severe headaches, the possibility of MC must be considered for the differential diagnosis and prompt measures should be taken to improve the patient's quality of life.

References

1. Saenger A: Ueber Hirnsymptome bei carcinomatose. Munch Med Wochenschr 1990; 47: 341–342.
2. Maddox HE 3rd: Metastatic tumor of the temporal bone. Ann Otol Rhinol Laryngol 1967; 76: 149–165.
3. Wasserstom WR, Glass JP, Posner JB: Diagnosis and treatment of leptomeningeal metastases frome solid tumors: Experience with 90 patients. Cancer 1982; 49: 759–772.
4. Heimann A, Merino MJ: Carcinomatous meningitis as the initial manifestation of a brest cancer. Acta Cytolog 1986; 30: 25–28.
5. Wagemakers M, Verhagen W, Borne B, Venderink D, Wauters C, Strobbe L: Bilateral profound hearing loss due to meningeal carcinomatosis. Journal of Clinical Neurosience 2005; 12: 315–318.
6. Kenmochi M, Sato S, Miyamoto Y, et al.: A case of sudden bilateral sensorineural hearing loss: a case report of the first manifestation of meningeal cartinomatosis. Otol Jpn 2003; 13: 657–661.
7. Nakagawa H, Murasawa A, Nakajima S, et al.: Study of cases with meningeal carcinomatosis: Diagnosis and treatment. Neurol Surg 1992; 20: 31–37.
8. Testoni S, Pirodda Trossello M, Minguzzi E, D'Alessandro R: Meningeal carcinomatosis causing isolated bilateral symmetric progressive hearing loss. Neurol Sci 2005; 25: 345–347.
9. Watanabe K, Hidaka K, Mizutani S, Yokoyama S, Ohshima T: A case of meningeal carcinomatosis initially presenting as rapid progression of bilateral sensorineural hearing loss. Otolaryngology Head and Neck Surgery 2003; 75: 890–893.
10. Morgan MK, Zammit-Maempel I, Hill J: Meningeal carcinomatosis: an unusual cause of deafness. J R Coll Surg Edinb 1998; 43: 119–112.

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