Pure Word Deafness with Possible Transfer of Language Dominance

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Abstract: A 55-year-old, right-handed male patient with a past history of a stroke followed by a difficulty of speech and hearing fell and manifested a left hemiplegia. He could neither comprehend spoken language and melody nor repeat them, though he spoke with paraphasia and understood written language and nonverbal sound. An electroencephalogram, pneumoencephalogram and cerebral angiogram suggested the existence of old infarcts in the left temporal lobe and a probable new one in the right cerebrum. A diagnosis of this case was made as pure word deafness which might be caused by a reimpairment of the language function possibly transferred to the nondominant, right hemisphere following the early stroke.

Key Words: pure word deafness, stroke, hemiplegia, electroencephalogram, pneumoencephalogram, cerebral angiogram, transfer of language dominance

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INTRODUCTION

Pure word deafness or subcortical sensory aphasia is a neuropsychological disorder the main symptom of which is an inability to comprehend and discriminate spoken language.^{8 15 22} Auditory perception *per se* is intact, so that the ability in recognition of nonverbal sounds is mostly preserved. Repetition and dictation are impossible. However, spontaneous speech, reading and writing are not very problematic. Such a condition is categorized not only in sensory aphasia but in auditory agnosia.^{8 15}

Our case report of pure word deafness is unusual in that a dextral with a past history of aphasia at a younger age manifested left hemiplegia, sensory aphasia and bilateral cerebral lesions confirmed by an electroencephalogram (EEG), pneumoencephalogram (PEG) and cerebral angiogram (CAG). It would be worth while discussing the neuropsychological focus which is engaged in a part of the linguistic function possibly transferred from the left to right nondominant hemisphere, though the case is old and lacking in postmortem examination.

CASE REPORT

Clinical Course

The patient was a 55-year-old, righthanded farmer with a character of stubbornness, punctiliousness and talkativeness but patience. He never had a personal or family history of left-handedness. His mother had

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died suddenly of an unknown disease. At the age of 28, he fell backward when sitting on a chair and suffered difficulties of speech as well as hearing followed by stuttering with paraphasia spanning several months.

Around 7 a.m. on January 5th, 1972, the patient abruptly fell again on his back while changing his clothes. He could not stand up and only moved his mouth without any voice at first. Then he could say his wife's name and began a confusional and garrulous speech. He was admitted to a local hospital, where talkativeness and a left hemiplegia were obvious. Upon physical examinations muscle rigidity of the limbs and neck was found, but the pupils were normal and he complained of neither nausea nor vomiting. His blood pressure was 180/90 mmHg. Cerebral thrombosis was suspected and citicoline was infused as treatment. A few days later both the confusional state and the hemiplegia subsided, and the blood pressure returned to his usual level (150/80). He gradually became taciturn and ill-humored. Penicillin, 600,000 units a day, was administered for 20 days, because the Wassermann reaction of serum was 2 positive in the Ogata agglutination method. He was discharged from the hospital on February 17th when he managed to walk by himself. He, however, talked selfishly irrelevant to questions, so he was taken to the psychiatry clinic of Mie University Hospital on April 21st. He was hospitalized for a further examination from April 10th till June 17th, 1972.

Recent information that was received stated that the patient's condition remained unchanged until his death on March 21st, 1979.

Mental Condition on Admission

The patient was a well-conscious, welloriented and honest man without apparent abnormalities except for linguistic disturbances and stubbornness. He had an insight into his problem and a desire for early recovery. However, he concomitantly felt disgraced, anxious and isolated, and was mildly depressed. A catastrophic reaction like an emotional incontinence was not observed. A formal, rigid and sometimes perplexed behavior was seen overlapped on his original personality. Remote as well as recent memory was intact.

Physical Condition on Admission

Temperature: 36.4°C. Blood pressure: 132/84 mmHg. Pulse: 66/min., regular, high tension. Eyes: good movement, no nystagmus, good reaction to light and good convergence of the pupils. Tongue: mildly trembling, slightly deviated to the left. Heart sounds: sinus rhythm without murmur. Cranial nerves except for the acoustic nerve: intact. Deep tendon reflex: slightly increased in all the limbs. Superficial abdominal reflex: disappeared on the left side. Babinski's sign: positive in the left foot. Oppenheim's sign: negative. Chaddock's sign: negative. Foot and knee clonus: negative. Romberg's sign: negative. Standing on one foot: possible. Grasping power: 27 kg with right hand, 24 kg with left hand. Two-point discrimination, finger naming, right-left orientation, skin graphaesthesia, stereognosis and constructional ability were normal.

Consultations

Oto-rhinolaryngologic examinations showed no abnormal finding except for difficulty in audiometry. Ophthalmologic examinations revealed myopic astigmatism, arteriosclerosis (Scheie, grade I) but no abnormal visual field.

Language Examinations

[Verbal Language]

The patient's spontaneous speech was basically fluent, though he was somewhat taciturn. The speech included mild paraphasia, word amnesia and circumlocutions, but developed neither into anarthria nor into jargon. Vocabulary and serial words were well preserved in his speech and there was no difficulty in finding the names of materials. Verbal comprehension was impossible for any spoken words, phrases or sentences. Semantic comprehension was fairly good, but phonemic comprehension was very difficult.

Repetition of remarks was also difficult for him even in single sounds or single words. Accidentally it sounded correct; however, this coincidence occurred by reading the movement of examiner's mouth. The difficulty of repetition was probably due to the disturbance of verbal comprehension.

[Literal Language]

Reading "Kana" (Japanese syllabary) and "Kanji" (Chinese characters) was possible, but sometimes paralexia was noted in parts of Kana and Kanji.

Taking written dictation was impossible due to the disturbance of verbal comprehension.

The meaning of words, as far as it was practical, was understandable up to double negative sentences. However, if a question was abstract, the answer tended to be off the point.

Spontaneous writing was possible, but it occasionally took a long time. Sometimes paragraphia and paragramatism were also seen in the sentences.

Construction of a sentence using given words was difficult owing to either the paragramatism or constructional apraxia.

Mapping the roads surrounding his house was well done.

Copying letters was also well done with comprehension of the meaning of words.

Calculation

Calculation with figures was correctly done, but it took a little long. Mental arithmetic was impossible.

Auditory, Visual and Tactile Recognition

Various sounds and noises were audible and distinguishable. Amusia was present, since recognition of rhythm and melody as well as spontaneous singing was impossible. Visual and tactile agnosias were ruled out.

Laboratory Examinations

Blood tests showed an increased icteric index (13) and a low level of total cholesterol (114 mg/dl).

The Wassermann reaction in serum was 4 positive in Ogata's method, while negative in the microscopic slide precipitation test, agglutination test and TPHA test, and all negative in the cerebrospinal fluid (CSF). Number of cells in the CSF: 2/3, lymphocyte: 1, neutrocyte: 1. Nonne-Apelt reaction: negative. Pandy's reaction: borderline. Total protein: 25 mg/dl.

The electrocardiogram (ECG) had a normal pattern with sinus bradycardia.

EEG recorded on February 28th (Fig. 1, left) showed irregular delta waves in the right temporal area concomitant with irregular theta waves in the left temporal area. EEG on April 22nd (Fig. 1, right) showed much improvement in the incidence and amplitude of abnormal waves in the right temporal area but unchanged theta waves in the left cerebrum.

A simple skull X-ray exhibited nothing abnormal.

PEGs (Fig. 2) presented distinct shadows made by large air spaces which suggested lacunae in the left temporal lobe, *inter alia*, in the part corresponding to the posterior Sylvian fissure. A faint shadow made by a small air space was also seen in the right temporal area near the Sylvian fissure. The posterior horn of the left lateral ventricle and the inferior horn of the right lateral ventricle were dilated.

A right carotid angiogram (Fig. 3, left) showed defects of the precentral artery, central artery and anterior and posterior parietal arteries which are branches of the middle cerebral artery. A left carotid angiogram (Fig. 3, right) showed no evidence that main branches of the middle cerebral artery were obstructed except for a part of the angular artery. However, these branches which looked narrow and a little displaced in the temporo-parietal lobes suggested their

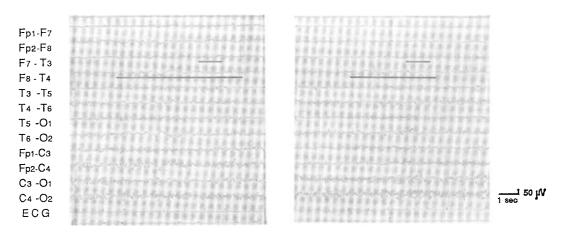


Fig. 1: Left: EEG recorded on February 8th showed irregular delta waves in the right temporal area (long underline) and irregular theta waves in the left temporal area (short underline). Right: EEG on April 22nd showed lower amplitude and less incidence of delta waves in the right temporal area (long underline) but unchanged theta waves in the left cerebrum (short underline).

revascularization occurred after the necrosis. These photographs taken for CAG a week after the PEG examination also showed a shadow made by the air indicating a lacuna in the anterior part of the temporal lobe.

DISCUSSION

Roughly described, this case had symptoms limited to the disturbance of verbal comprehension, which secondarily led to the inability in repetition and dictation. Little disturbance of internal speech might explain the relatively good performances in spontaneous speech and writing, literal comprehension and reading aloud. These clinical pictures meet the criteria of pure word deafness according to the classical nosology of aphasia.15 This entity, however, could be shifted from Wernicke's aphasia, because the patient was in a confusional state with talkativeness shortly after the second stroke and still retained traces of paraphasia, paralexia, paragraphia and paragramatism on admission to the university hospital. Cortical or mental deafness should be excluded, since nonverbal sounds were distinguished. Expressive as well as receptive amusia was present as seen in the other reported cases of pure word deafness.^{10 22} This aphasia mostly unimpared in internal speech could be regarded as a kind of auditory agnosia in which the function of linguistic recognition is selectively impared.¹⁵

The etiologies of the double stroke are unknown, a cerebral embolism, a cerebral thrombosis or a cerebral hemorrhage being possible. The first candidate may be an embolism in view of the relatively young age when he had the first stroke and the later finding of a positive Wassermann reaction, but no abnormal sign in the heart. As the second one, however, a thrombosis or a hemorrhage is not ruled out on account of the high blood pressure at the second stroke.

The large lacunae proved by PEG in the posterior part of the left temporal lobe and the probable connected lacuna accidentally casted on CAG in the anterior part of the lobe seem to be quite old, because the branch arteries in these regions could be revascularized in evidence of their narrowness and displacement. Other supporting evidences of the old lacunae are the unchanged EEG on the left side for the few months and the

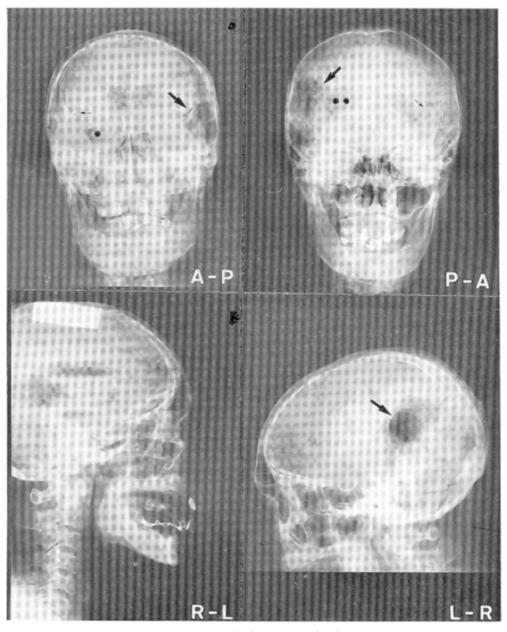


Fig. 2: PEGs presented distinct shadows made by large air spaces in the left temporal lobe near the posterior Sylvian fissure (\rightarrow) , and a faint shadow made by a small air space in the right temporal area (\rightarrow) . The posterior horn of the left lateral ventricle (**) and the inferior horn of the right ventricle (*) was dilated. A-P: anterior to posterior projection. P-A: posterior to anterior projection. R-L: right to left projection. L-R: left to right projection.

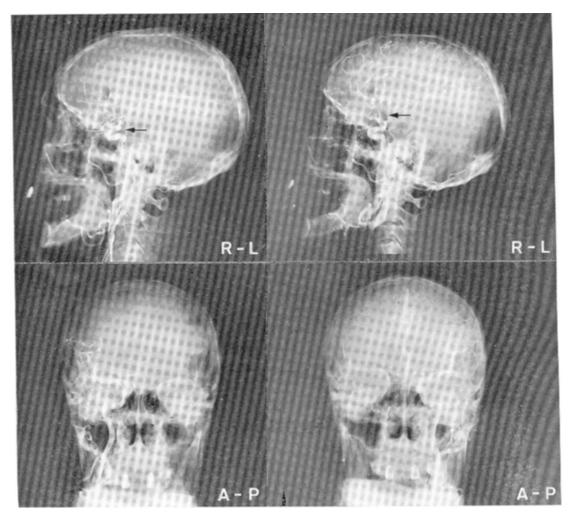


Fig. 3: Left: (R-L & A-P): A right carotid angiogram showed defects of the precentral artery, central artery as well as anterior and posterior parietal arteries which are branches of the middle cerebral artery. Right: (R-L & A-P): A left carotid angiogram showed an obstruction in a part of angular artery. The branches of the left middle cerebral artery looked narrow and displaced in the temporo-parietal lobes. These photographs taken for CAG also showed a shadow made by residual air infused a week before the PEG examination in the anterior part of the temporal lobe (\rightarrow) .

lesions in the left hemisphere corresponding to the clinical symptoms which included motor aphasia of dextral after the first stroke. A probable lesion in the right temporal area seen on PEG may be newly made considering the EEG in the right cerebrum improved for a short period, the widespread vanishment of branches of the right middle cerebral artery and the second aphasia concomitant with the left hemiplegia.

A matter of primary concern in the present case is the mechanism of how language function recovered after the early episode of aphasia and how it was impaired again at the later stroke.⁷ Luria¹³ proposed three ways of functional recovery from the brain injury, i.e. 1) recovery of injured tissues per se, 2) shift of function to the contralateral hemisphere and 3) reorganization compensated by different neuronal structures. All these explanations could apply to the restoration process in our case. However, in view of the old-looking infarcts in the left temporal lobe and the relatively well-maintained vascularity in the left cerebrum, it would be unlikely that by the second stroke with left hemiplegia, the lesions made by the first stroke or their surroundings in the left hemisphere were reinjured after functional recovery. Paying attention to the word deafness with left hemiplegia, the small infarct-like shadow on PEG in the right temporal area, the EEG abnormality predominant in the right temporal area and the arterial defects in the right cerebrum, it would be more possible that the new language center transferred to the nondominant right hemisphere was injured by the second stroke. The capacity of the right hemisphere for developing linguistic function seems to involve not only prosody,^{17 23} but also major functions like vocabulary and grammar usually managed by the left hemisphere.9 12 13 Vignolo24 and Prins et al.¹⁶ further indicated that the right hemisphere has a greater capacity for language comprehension than for expression. If their insistence is true, it could be more likely that the comprehensive function transferred to the right hemisphere was disordered by the later stroke. In fact several cases suggesting the transfer of language dominance following the left cerebral infarction,2 9 18 commissurotomy³ or left hemispherectomy⁴ ¹⁹ and similar cases in which a new lesion destroyed the language function shifted to the right hemisphere⁵¹¹¹⁴ were reported in literature.

From the viewpoint of age in language development, our case at the early stroke was already far beyond the critical period of speech acquisition. It is generally believed that the damaged language function in the dominant hemisphere is not very easy to recover after the critical period.⁶ However, one case which developed supranormal language ability after a left hemispherectomy was reported by Smith.²⁰ Several other cases of ages over 15^{35914} and extreme cases of over 50^{211} were presented with a partial restoration of the language system following a lesion of the left hemisphere. Therefore, in our case the age of language development in the nondominant hemisphere is not thought to be too old.

Another possible explanation for the functional restoration is that the language capacities originally existed in either bilateral or crossed nondominant hemispheres.^{1 21} In either case no symptom of aphasia would be manifested following the injury to the left hemisphere. However, it was actually observed in our case. Thus, we finally conclude that the language function transferred from the left to right hemisphere after the first stroke was reimpaired by the second one.

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