Case report / *Olgu sunumu*

Temporal lobe epilepsy manifests as dementia: a case report

**Min-Hsien HSU,[1] Hsiu-Ching HUNG,[1] Tsui-Hung HUNG,[1]
Shangfen LIN,[2] Chung-Hsin YEH[2,3]**

ABSTRACT

Temporal lobe epilepsy is the most common type of localization-related epilepsy and may manifest as behavior change or psychosis. Sometimes it is hard to diagnose because we are used to thinking about dementia probably. Here we reported an unusual case that a 73 years-old male patient with dementia-like symptoms previously, but finally he was diagnosed as temporal lobe epilepsy by the electrophysiological and radiological finding. ***(Anatolian Journal of Psychiatry 2019; 20(2):217-220)***

Keywords: *temporal lobe epilepsy, dementia-like symptoms, antiepileptic treatment*

Bunama gibi görünen temporal lop epilepsisi: Bir olgu sunumu

ÖZ

Temporal lop epilepsisi yerleşimi belli olan epilepsinin en yaygın tipidir ve davranış değişikliği veya psikoz olarak görülebilir. Bunama olasılığını düşündüğümüz için kimi zaman tanı koymak güçtür. Bu yazıda, başlangıçta bunama benzeri belirtileri olan, fakat elektrofizyolojik ve radyolojik tetkiklerle temporal lop epilepsisi tanısı konan 73 yaşındaki bir erkek hastayı sunduk. ***(Anadolu Psikiyatri Derg 2019; 20(2):217-220)***

Anahtar sözcükler: *Temporal lop epilepsisi, bunama benzeri belirtiler, antiepileptic tedavi*

INTRODUCTION

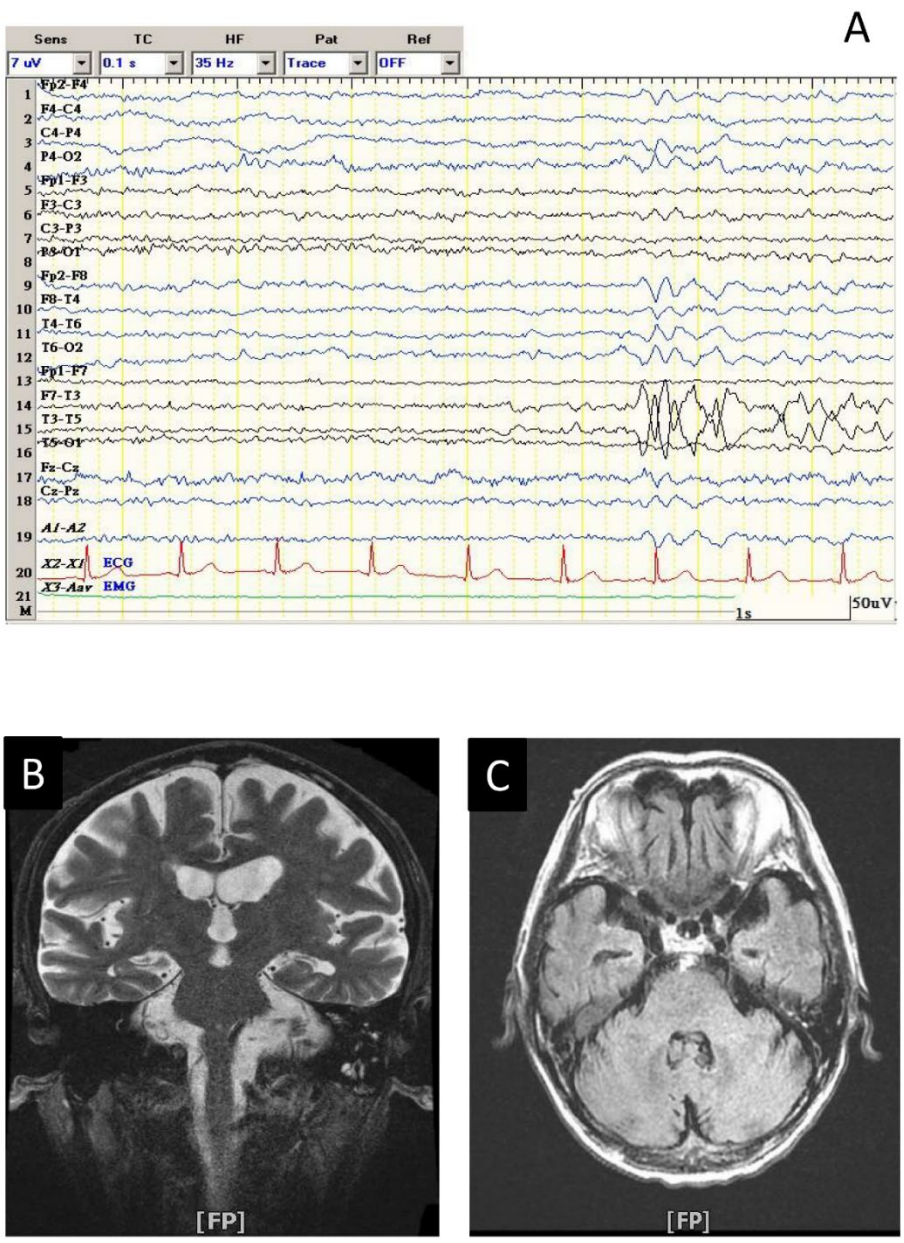
Temporal lobe epilepsy (TLE), a chronic neurological condition characterized by recurrent, unprovoked ictal discharges from the temporal lobe of the brain, could be difficult to diagnose because of vague symptoms. Including strange sense, hallucination, psychosis, cognitive impairment, or automatism, they are all responsible for possible symptoms of temporal lobe epilepsy.[1,2] Simultaneously, a worldwide of 107 epilepsy surgery centers confirmed that TLE is the most common type of localization-related epilepsy. Of 8.234 operations performed between 1985 and 1990, 66% involved the temporal lobe.[3] While the patients are getting older, they may manifest as dementia symptoms, such as memory decline, agitation, or speech difficulty. Sometimes it is hard to differentiate between these cognitive changes and TLE. Here we reported a case of TLE found in a 73 years-old man who displayed as preliminarily behavior change, irritable mood, and did not present any limbs convulsion or upward gaze.

[1] Department of Neurology, Show-Chwan Memorial Hospital, Changhua, Taiwan
[2] Department of Neurology, Yuan Sheng Hospital, Changhua, Taiwan
[3] Department of Sport and Health Management, College of Nursing and Health, Da-Yeh University, Changhua, Taiwan
Yazışma adresi / ***Correspondence address:***
Chung-Hsin YEH, M.D., Ph.D., Department of Neurology, Yuan Sheng Hospital, Changhua, Taiwan
E-mail: shium8852@gmail.com
Received: January, 16th 2019, Accepted: February, 09th 2019, doi: 10.5455/apd.26413

CASE PRESENTATION

A 73 years-old man was admitted to our hospital because he was suffered from behavior change recently. The patient became hypertalkative, but his speech content was usually irrelevant. Besides, irritable mood, insomnia, memory decline, and visual hallucination were also accompanied. Furthermore, he often lost his way, he even snarled at the policemen because he could not find his motorcycle. These symptoms were worse and worse for several months prior to his admission. The patient was ever brought to Psychiatry Clinic for help, but his condition.

Figure 1. EEG and MRI: **A**, EEG shows several spikes over right temporal area (solid arrow). **B** (T2WI sagittal view). **C** (FLAIR transverse view), brain MRI indicate medial temporal lobe atrophy.



showed no improving after treatment

Physical examination revealed no limbs weakness, nor any cranial nerve signs. The laboratory data revealed no bacteremia, without thyroid dysfunction, nor other electrolyte imbalance. However, several bursts of high voltage spikes dominantly over right temporal area were found by the electroencephalography (Figure 1A). Magnetic resonance imaging indicated global cortical atrophy and medial temporal lobe atrophy (Figure 1B and 1C). Hence the anti-epileptic drug (valproic acid, 1000 mg/day) was prescribed; those behavioral symptoms got improved.

DISCUSSION

According the recent studies, TLE can be classified into two main types: mesial temporal lobe epilepsy (involved as internal structures of the temporal lobe) and neocortical temporal lobe epilepsy (involved as outer portion of the temporal lobe).[4] Clinical semiology of temporal lobe epilepsy consists of prodrome (irritability or anxiety), aura (viscerosensory symptoms or hallucination), altered consciousness (absence episodes or disorientation), amnesia, and automatism.[5] When a young patient presented as epigastric rising, hallucination, then followed by disoriented consciousness, we can easily make a diagnosis of epileptic syndrome. But if old patients with previously mentioned symptoms, sometimes clinical physicians may think of dementia firstly. There were several literatures to indicate the association or difference between epilepsy and dementia in the elderly. Older patients with dementia were associated with an increased risk of developing epilepsy as compared with groups with non-dementia. Cases of a younger age reported to be particularly to have epilepsy compared with older cases.[6] Maybe these conditions lead us to easily make a wrong diagnosis. Electroencephalography and magnetic resonance imaging are important tests in the diagnosis of this condition.

This reported case is unusual because he could complete those whole bizarre conduct, he was brought to the psychiatric clinic first. Therefore, his clinical manifestation indicated behavioral or psychological symptoms mimic dementia. The patient had no past history of epilepsy and intracranial insults. In fact, his family sent the patient to the psychiatric clinic at that time due to suspicion of schizophrenia or dementia with Lewy bodies (DLB). Sometimes it is not easy to make a distinction between truely schizophrenic psychosis and ictal or postictal psychosis. According to Seyed M. Mirsattari's literature, patients with typical schizophrenia may have high premorbid function, preserved affect, and preoccupation with religious, moral, or ethical matters.[7] Besides psychosis, our case also presented as visual hallucination in the beginning clincal course. Visual hallucination is one of the supportive features in the diagnostic criteria for DLB, but there are less literatures reporting that epilepsy could present as dementia with Lewy bodies. Relatively more literatures show that TLE may be simulating Alzheimer's disease (AD),[8] and our patient's performance is very rare. Not like AD, DLB is less or even not reporting associated with an increased risk of developing epilepsy.[9] The symptom of transient loss of consciousness has been mentioned in DLB cases, but that may not be related with epilepsy, rather than more likely to be related to the autonomic dysfunction. In addition, we need to notice that if any possibility of not just TLE or maybe frontal lobe epilepsy (FLE) semiology according to our patient's initial symptoms. FLE can occur usually during sleep, typically short, in clusters, and can have brief postictal confusion with hypermotor activity.[10] Our patient's initial symptoms are not like frontal lobe epilepsy.

It should be noted that this study has examined only EEG and MRI data of the patient's record. If we can collect his Mini-Mental State Examination (MMSE) or Clinical Dementia Rating (CDR) of this case, we will have more information to establish the correlation between the patient's epileptic syndrome and possibly dementic symptoms, or otherwise we just make the unfortunate misdiagnosis. Furthermore, the result can't be taken as evidence of the difference between this patient's memory functions after and before antiepileptic treatment. We expect that behavioral symptoms can be improved after antiepileptic treatment, but the real impact of memory functions after antiepileptic treatment may need more consideration. The previous literatures display the results may be still controversial. Memory functions can be affected by antiepileptic treatment or interictal discharge. Three patients' case-study show that memory decline could be improved with antiepileptic medicine, and the possible mechanism might be related with sufficiently treated temporal lobe spike activity.[11] However, memory functions also could be improved after withdrawal of antiepileptic medication. A randomized double-blind Norway study reveal that comparing to placebo groups, withdrawal of carbamazepine or valproate in well-

controlled epilepsy patients significantly improved memory functions.[12] It will be helpful for clinicians by collecting the patient's scores of memory tests to distinguish his outcome after from before antiepileptic treatment.

Despite its preliminary character, this study can clearly indicate that TLE is difficult to be diagnosed early in aged patients because we are used to consider about senile dementia probably. In treating them, it is still necessary to make a clear-cut history taking.

Authors' contributions: *M.H.H.: conceptualization, investigation, data curation, writing-original draft preparation, project administration; H.C.H.: investigation; T.H.H.: data curation; C.H.Y.: conceptualization, validation, writing-review & editing, supervision; All authors: reviewed the final version of the manuscript.*

Acknowledgments: *We would like to thank Professor Chung-Hsin Yeh and Nurse Practitioners Shang-Fen Lin, Hsiu-Ching Hung and Tsui-Hung Hung in the Show-Chwan Memorial Hospital for their idea and discussion about this patient care and condition analysis.*

REFERENCES

1. *Wassenaar M, Leijten FSS, de Haan GJ, Uijl SG, Sander JW. Electro-clinical criteria and surgical outcome: Is there a difference between mesial and lesional temporal lobe epilepsy? Acta Neurol Scand 2017;136:708-714.*
2. *Didato G, Chiesa V, Villani F, Pelliccia V, Deleo F, Gozzo F, et al. Bitemporal epilepsy: A specific anatomo-electro-clinical phenotype in the temporal lobe epilepsy spectrum. Seizure 2015; 31:112-119.*
3. *Wiebe S. Epidemiology of temporal lobe epilepsy. Can J Neurol Sci 2000; 27(Suppl.1):S6-10; discussion S11-20.*
4. *Benbadis SR, Thomas P, Pontone G. A prospective comparison between two seizure classifications. Seizure 2001; 10:247-249.*
5. *Blair RD. Temporal lobe epilepsy semiology. Epilepsy Res Treat 2012; 2012:751510. doi: 10.1155/2012/751510.*
6. *Sherzai D, Losey T, Vega S, Sherzai A. Seizures and dementia in the elderly: Nationwide inpatient sample 1999-2008. Epilepsy Behav 2014;36:53-56. doi: 10.1016/j.yebeh.2014.04.015.*
7. *Mirsattari SM, Gofton TE, Chong DJ. Misdiagnosis of epileptic seizures as manifestations of psychiatric illnesses. Can J Neurol Sci 2011; 38:487-493.*
8. *Sinforiani E, Manni R, Bernasconi L, Banchieri LM, Zucchella C. Memory disturbances and temporal lobe epilepsy simulating alzheimer's disease: A case report. Functional Neurology 2003; 18:39-41.*
9. *Jahodova A, Krsek P, Komarek V, Kudr M, Kyncl M, Zamecnik J, et al. Frontal lobe epilepsy with atypical seizure semiology resembling shuddering attacks or wet dog shake seizures. Epileptic Disorders: International Epilepsy Journal with Videotape 2012;14:69-75.*
10. *McGonigal A, Chauvel P. Frontal lobe epilepsy: Seizure semiology and presurgical evaluation. Practical Neurology 2004;4:260-273.*
11. *Hogh P, Smith SJ, Scahill RI, Chan D, Harvey RJ, Fox NC, Rossor MN. Epilepsy presenting as ad: Neuroimaging, electroclinical features, and response to treatment. Neurology 2002;58:298-301.*
12. *Hessen E, Lossius MI, Gjerstad L. Improvement in verbal memory after withdrawal of carbamazepine and valproate in patients with well-controlled epilepsy: A randomized, double-blind study. Acta Neurol Scand 2011; 123(6):385-389.*