International Journal of Scientific Advances
ISSN: 2708-7972
Volume: 2 | Issue: 5 | Sep - Oct 2021 Available Online: www.ijscia.com
DOI: 10.51542/ijscia.v2i5.31

Temporal Lobe Epilepsy Misdiagnosed as Schizophrenia: A Case Report

Gibson O. Anugwom, MD, MSc[1]; Alexsandra Urhi, MD[2*]; David O. Otuada, MD, MPH[3]; Funso Oladunjoye, MD, MPH[4]; Arthur Dilibe, MD[5]; Dolly Ogwu, MD[6]; Temidayo Odugbesan, MD[5]; Egbebalakhamen Osa, MD, MPH[7]; Toluwalope Ejiyooye, MBBS[8]; Hafiz Olatunde MD, MPH[9]

[1]Department of Psychiatry, Houston Behavioral Healthcare Hospital, Houston, TX, USA.
[2]Department of Psychiatry, Federal Medical Center, Asada, Delta, Nigeria.
[3]University of Alabama at Birmingham, Birmingham, AL, USA.
[4]Department of Psychiatry, Baylor College of Medicine, Houston, TX, USA.
[5]Windsor university school of Medicine, St Kitts and Nevis.
[6]University of Benin, school of Medicine, Benin, Nigeria.
[7]SUNY downstate Health Sciences University, NY, USA.
[8]New England College, Henniker, NH, USA.
[9]Jydes Family Clinics, Georgia, USA.

*Corresponding author details: Alexsandra Urhi, MD; sandyurhi255@gmail.com

ABSTRACT

Temporal lobe epilepsy is a neurological disorder of an unprovoked type of focal (partial) epilepsy that begins in the temporal lobe of the brain. Patients with this condition are often misdiagnosed due to similarities in presentation to other conditions. In this case report, we presented a 34-year-old male, who had symptoms of hallucination, anxiety, and depression which can be seen in patients with temporal lobe epilepsy. Due overlap in symptoms, he was misdiagnosed to have schizophrenia. Following subsequent review of his medical history and findings seen in his laboratory work and imaging studies, it was determined that his symptoms were caused by seizures originating from an atrophic lesion in his hippocampus found on magnetic resonance imaging of his temporal lobe.

Keywords: schizophrenia; epilepsy; seizure; electroencephalogram

INTRODUCTION

Temporal lobe seizures are an unprovoked type of focal (partial) epilepsy that begins in the temporal lobe of the brain, which processes emotions and is important for short-term memory. Most patients who experience this condition present with a variety of symptoms such as feelings of Deja-vu, amnesia, anxiety or panic, depression, a rising sensation coming from the stomach to the chest or throat, nausea, auditory or olfactory hallucinations (which can occur as aura), motionless stare, dilated pupils, and automatisms, and all these are due to neural connections within the temporal lobe [1,2]. Most times, patients with this condition are misdiagnosed due to some features that overlap with other conditions.

To better understand these similarities in symptoms, it is necessary to know the anatomy of the temporal lobe and its associated functions. The temporal lobe forms the cerebral cortex in conjunction with the occipital lobe, parietal lobe, and frontal lobe [3]. The temporal lobe contains the auditory cortex, olfactory cortex, part of the lateral ventricle, the tail of the caudate nucleus, the stria terminalis, the hippocampal formation, and the amygdala [3].

Different parts of the temporal lobe are involved in hearing (auditory cortex and association area), smell (olfactory cortex), object identification (posterior association area), memory (limbic association area) [3]. Seizures (epilepsy) in the temporal lobe can be subdivided into two categories based on the anatomical origin of epileptic focus:

(1) Mesial Temporal Lobe Epilepsy (MTLE): Involving the innermost structure of the temporal lobe, including the hippocampus, Para hippocampal gyrus, and amygdala; this is the most common form of temporal lobe seizures and is usually secondary to a pathological process known as hippocampal sclerosis (HS) [4].

(2) Lateral Temporal Lobe Epilepsy (LTLE): Also referred to as neocortical temporal lobe seizures. These are very rare and most commonly secondary to genetic or acquired structural/ anatomical lesions [4].

In this case report, we present a patient with temporal lobe epilepsy, who was misdiagnosed and treated for schizophrenia.

CASE PRESENTATION

A 34-year-old Caucasian male presented for psychiatric evaluation prior to his scheduled weight loss surgery. He is employed, unmarried, has no children and lives alone. He has a psychiatric history of schizophrenia with depressive symptoms diagnosed in his 20s. His condition was characterized by periods of depression, anxiety, auditory and olfactory hallucinations. His medical history was significant hypertension, foot ulcer, and obesity. He has a family history of depression in his mother and younger brother. His father and other two siblings are alive and healthy. On physical examination, his blood pressure was measured to be 164/108 mmHg, pulse was 89/min, respiratory rate was 22/min, weight was 309 Ibs, height was 5ft 8in, body mass index 47.0 kg/m2, and temperature 99.1F.

MENTAL STATUS EXAMINATION

On appearance, he is a well-groomed man, looks older than his stated age, has no dysmorphic features, and no abnormal movement. He avoids eye contact, has a sad expression, cooperative, and engage with the examination. His speech is fluent and normotonic. He has a logical thought process, good insight, and judgment. Intact short- and long-term memory, orientated to place, time and person. Normal attention span. On evaluation for suicidal risk, he denied current thoughts of suicide but states "sometimes, I feel there is no point living". His Patient Health Questionnaire (PHQ-9) score was 22, suggestive of severe depression. He denied the use of illicit drugs but used alcohol in the past to self-medicate.

On review of his psychiatric history, he was diagnosed with schizophrenia with depressive symptoms when he was in his 20s. At the time of diagnosis, he presented with a sudden onset of olfactory or auditory hallucination which lasts for few seconds to minutes, followed by bouts of depression and anxiety lasting for days to weeks. He described the olfactory hallucination as smelling strange things that are not there and auditory hallucination occurs as hearing people whispering behind him or hearing voice from the wall, telling him that he is worthless. This episode is followed by depression and anxiety that usually last for days to weeks. He was initially prescribed haloperidol at the time of initial diagnosis, which was titrated to the highest allowed dose, but his symptoms persisted. Clozapine was later added to the haloperidol to augment his treatment. However, after one year on this regimen, he was switched to Aripiprazole due to lack of significant improvement. He reported some improvement in his mood while on aripiprazole but had an increased frequency of hallucination and anxiety. The aripiprazole was discontinued, and he received Electroconvulsive therapy (ECT). He initially had a long symptom-free period after the ECT treatment; however, his symptoms relapsed but with less frequency.

Considering the description of his symptoms, where symptoms of depression and anxiety are preceded by sudden onset olfactory or auditory hallucination, he was reevaluated. Blood work including complete blood count, basic metabolic panel, liver function test, lipid panel, thyroid function test, folate, vitamin B12 level, prolactin, urinalysis, and urine toxicology. All labs were within normal limits, except LDL-174 mg/dl and prolactin 67 ng/ml. He was referred for magnetic resonance imaging (MRI) to rule out prolactin-producing lesions due to the elevated prolactin level. On reviewing his MRI, a small atrophic lesion was found around the hippocampus. He was referred for electroencephalography studies (EEG) to determine any abnormal changes in his brain electrical function. His EEG report was inconclusive for any seizure activities. We decided to start him on Depakote for its anticonvulsant and mood-stabilizing effect, Lamotrigine to augment the effect of Depakote, recommended transcranial magnetic stimulation for his anxiety and depression, and referred him to a neurologist for the continuation of care due to the findings in the MRI.

DISCUSSION

It is often expected that professional judgments of medical practitioners are accurate, however, it can be challenging to always make accurate diagnoses especially in psychiatry due to overlap in symptoms and reliant on patient's symptom description to make accurate diagnosis. Our index patient was initially diagnosed and treated for schizophrenia, but subsequent history, supported by findings on his magnetic resonance imaging (MRI) suggested that he in fact has temporal lobe epilepsy (TLE), which is a common type of epilepsy disorder [5]. TLE and schizophrenia may present similarly, hence the need for a high suspicion in diagnosis [5]. A study done by Slater et al observed some degree of heterogeneity in their epileptic patients with psychosis resembling schizophrenia symptoms [6]. A small number of them presented with a predominance of hebephrenic and catatonic symptoms and with deterioration more typical of schizophrenia [6]. Slater thought that schizophrenia-like psychosis of epilepsy (SLPE) was schizophrenic in form but not in "etiology" and that it occurs in individuals lacking any special predisposition (i.e., no increased family history of schizophrenia and lack of premorbid schizoid traits) [6]. In Slater's view, the symptoms of his psychotic epileptic patients would have to be diagnosed as "schizophrenia" although he thought the "combination of symptoms shown by individuals differs slightly from the most usual schizophrenic pattern" [6]. Slater described a clinical picture with an average onset of 14 years from the beginning of epilepsy and with a prevalence of hallucinations and persecutory and mystical delusions with three-quarters of the patients having a temporal lobe focus [6]. Also, he observed that there was a decreased prevalence of schizophrenia in the families of patients with epilepsy enforcing that SLPE is a distinct diagnostic entity from schizophrenia [6], this supports the lack of family history of schizophrenia in our patient. His family history was only significant for depression in his mother and younger brother. His study presented patient had olfactory hallucination, auditory hallucination, anxiety, aggression, twisting feeling in his stomach, and other features related to

schizophrenia [7], which are symptoms similar to our index patient who presented with an olfactory and/or auditory hallucination that lasted only a few seconds to minutes, followed by bouts of depression and anxiety assessed as he is currently seeing the neurologist. Due to these similarities in symptoms of schizophrenia and TLE, patients with TLE are sometimes misdiagnosed to be schizophrenic.

Due to an elevated prolactin level of 67ng/ml, an MRI was requested to rule out prolactin-secreting lesions such as prolactinomas, which eventually led to the identification of atrophic lesions in the hippocampus in our patient. Studies have found that there is an association between elevated prolactin level and TLE. Most patients exhibit elevated levels of prolactin after complex focal seizures like TLE. This is usually most evident within 30 minutes after the seizure episode. In the study by Lin et al, postictal rise in serum prolactin level 15 to 20 minutes after the onset of the seizures in patients with TLE was reported [8]. Also, in the study by Jürgen et al, the findings support that serum levels of prolactin may increase as a consequence of epileptic seizures. He also reported that the rise in prolactin level is due to propagation of epileptic activity, usually from the temporal lobe to the hypothalamic-pituitary axis and it's seen in approximately 60% of complex partial seizures [9].

In diagnosing patients with TLE, an MRI of the brain is encouraged to look for changes in the temporal lobe such as reduction in hippocampal volume as seen in our patient presented in this case report. MRI may also be vital for the identification of other organic or structural anomalies which may precipitate temporal lobe seizures such as vascular malformations or tumors [1].

An electroencephalogram (EEG) should also be done to aid in the diagnosis. EEG will usually show a spike or sharp waves originating from the temporal lobe, however, seizures arising in more mesial (middle) temporal lobe areas, may only show rhythmic slowing during seizures. In some cases, it may be difficult to pick up these changes in an EEG due to patients declining to participate in the long duration of testing which may not yield a positive outcome since the result is dependent on the patient expressing an episode [10]. As seen in the index patient, the EEG was unremarkable even though MRI findings detected pathologic lesions in the temporal lobe.

CONCLUSIONS

Given the overlap in symptoms of TLE with other conditions, such as schizophrenia, as seen in our patient, it is important that psychiatrists, neurologists, and other physicians maintain a high index of suspicion for TLE in patients presenting with symptoms such as hallucinations, depression, and anxiety, as these might be a presentation of TLE. These measures will likely decrease the incidence of misdiagnosing TLE as a psychotic disorder which may have overlapping features. These would also decrease the health impact incurred from the side effects of medications used to treat the wrong conditions.

REFERENCES

[1] Health Jade Team. Temporal Lobe Epilepsy. HEALTHJADE. May 2019. https://healthjade.net/temporal-lobe-epilepsy/ Accessed Aug 31, 2021.

[2] Butler T, Blackmon K, McDonald CR, et al. Cortical thickness abnormalities associated with depressive symptoms in temporal lobe epilepsy. Epilepsy Behav. 2012 Jan; 23(1): 64-7.

[3] Kandratavicius L, Hallak JE, Leite JP. What are the similarities and differences between schizophrenia and schizophrenia-like psychosis of epilepsy? A neuropathological approach to the understanding of schizophrenia spectrum and epilepsy. Epilepsy Behav. 2014 Sep; 38:143-7. doi: 10.1016/j.yebeh.2014.01.005. Epub 2014 Feb 5. PMID: 24508393.

[4] Kiernan JA. Anatomy of the temporal lobe. Epilepsy Res Treat. 2012; 2012:176157. doi: 10.1155/2012/176157. Epub 2012 Mar 29. PMID: 22934160; PMCID: PMC3420617.

[5] McIntosh WC, M Das J. Temporal Seizure. [Updated 2021 Jul 10]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK549852/

[6] Slaterlater E, EardBeard AW, litheroGlithero E. The schizophrenia like psychosis of epilepsy. Br J Psychiatry. 1963 Jan; 109:95-150. doi: 10.1192/bjp.109.458.95. PMID: 13989174.

[7] Sadia Sultan, Ebtihaj Omar Fallata, "A Case of Complex Partial Seizures Presenting as Acute and Transient Psychotic Disorder", Case Reports in Psychiatry, vol. 2019, Article ID 1901254, 4 pages, 2019. https://doi.org/10.1155/2019/1901254

[8] Lin YY, Su MS, Yiu CH, Shih YH, Yen DJ, Kwan SY, Wu ZA, Chan SH. Relationship between mesial temporal seizure focus and elevated serum prolactin in temporal lobe epilepsy. Neurology. 1997 Aug;49(2):528-32. doi: 10.1212/wnl.49.2.528. PMID: 9270590.

[9] Jürgen Bauer. Epilepsy and prolactin in adults: a clinical review. Epilepsy Research, 24;1, 1996, Pg 1-7. https://doi.org/10.1016/0920-1211(96)00009-5.

[10] Javidan M. Electroencephalography in mesial temporal lobe epilepsy: a review. Epilepsy Res Treat. 2012; 2012:637430. doi:10.1155/2012/637430