International Journal of General Medicine and Pharmacy (IJGMP)
ISSN (P): 2319-3999; ISSN (E): 2319-4006
Vol. 6, Issue 6, Oct - Nav 2017; 13 - 22

International Academy of Science, Engineering and Technology
Connecting Researchers; Nurturing Innovations

LIMBIC PSYCHOTIC TRIGGER REACTION, A MOTIVATIONAL HOMICIDE: A CASE REPORT

DARAKHSHAN AHMAD[1], ABDUL GHAFOOR[2], FAREED MINHAS[3] & MAHMOOD ALI KHAN[4]

[1]Holy Family Hospital, Rawalpindi, Pakistan

[2, 3, 4] Institute of Psychiatry, WHO Collaborating Centre, Benazir Bhutto Hospital, Rawalpindi, Pakistan

ABSTRACT

Limbic Psychotic Trigger Reaction is a type of Non Convulsive behavioral seizures. As described by Pontius, memories triggered by an unrelated stimulus kindle seizure like activity in the limbic system, which combined with the disharmony of front limbic interactions, results in sudden, ”bizarre, out of character, emotionless, non voluntary and unplanned antisocial acts”

To the best of our knowledge, There have been a total of 24 reported cases of Limbic Psychotic Trigger Reaction worldwide, and this is the 25th. Four of these involved fire setting, one case of bank robbery and the rest of homicide (with one case involving attempt to rape).

All these cases were evoked rather than provoked and involved the classic 3 components of seizure i-e aura (comprising perceptual distortions, hallucinations, etc.), ictal phase (usually associated with autonomic dysfunction) and postictal confusion.

The objective of this case report is not only to highlight the symptomatology of this disease, but also emphasizes on progression with time and long term sequel in the form of depression and brain damage.

KEYWORDS: Behavioral Seizures, Kindling, Fronto Limbic Disharmony, Kindling, Limbic Psychotic Trigger Reaction, Temporal Lobe Epilepsy, Partial Seizures

INTRODUCTION

Limbic Psychotic Trigger Reaction (LPTR) is a type of non convulsive behavioral seizures. As described by Pontius, 2004[1], memories triggered by an unrelated stimulus kindle seizure like activity in the limbic system, which result in sudden, “bizarre, out of character, non voluntary antisocial acts”. [1]

To the best of our knowledge, there have been a total of 24 reported cases of limbic psychotic trigger reaction worldwide, and this is the 25th. Four of these involved fire setting, [1-4]one case of bank robbery[5]and the rest of homicide (with one case involving attempts to rape) [6]

PATHOPHYSIOLOGIC MECHANISMS

The Limbic Psychotic Trigger reaction is a poorly understood condition. Various pathophysiologic mechanisms have been proposed; these are as follows.

Kindling

Goddard (1967) was the first to describe the mechanism of seizure kindling due to repeated stimuli.

[7]Mark and Ervin demonstrated that the limbic system, particularly amygdala is the most susceptible to kindling mechanism. [8]The kindling theory of LPTR was further substantiated by studies by Weiser 1983, Gloor*et al* 1982, Goddard 1986 and Trimble *et al* 1997.[9-12]

In primates, kindling produces behavioral seizures. [13]Likewise in humans, it is postulated that long term stresses/ previous traumatic experiences stored in memory kindle these behavioral seizures, with the trigger being a mild, remotely related or completely unrelated stimulus. Halgren*etal*[14] proved during his research on Temporal Lobe Epilepsy (TLE) that synaptic circuits of memory formation and Temporal Lobe Epilepsy were largely similar. [15-18]

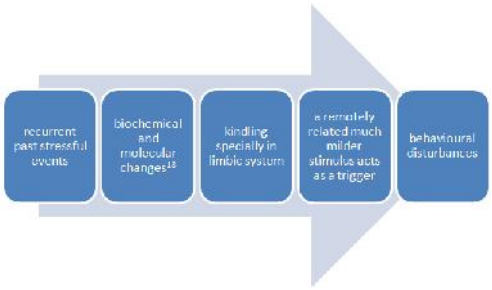


Figure 1

Frontal Lobe Dysfunction/ Limbic System Dysfunction/ Erratic Interplay between Frontal Lobe and Limbic System

Limbic system consists of hypothalamus, amygdala, and hippocampus. These are involved in motivation, emotion, learning, and memory. [19]

The Frontal lobes on the other hand are involved in impulse control, and modulation of actions and emotions generated by the limbic system as per social and moral appropriateness and consequences.

The frontal lobe functions in harmony with limbic system [20,21]and enables us to modulate our actions and responses when deemed inappropriate. The hypothalamus, a part of the limbic system, can be implicated in the transient imbalance of the autonomic nervous system that is seen with LPTR.[6, 22, 23]

This is the reason most of these patients score poorly on tests assessing frontal lobe functions, e.g. Trail making test and clinical narratives test. [24]

Role of Neurotransmitters

Henriksen et al studied the EEG patterns of rat brains, after administration of subtherapeutic doses of beta endorphins. They demonstrated that beta endorphins cause increased activity in the limbic brain areas of rats. [25]

Vagal Stimulation[26]

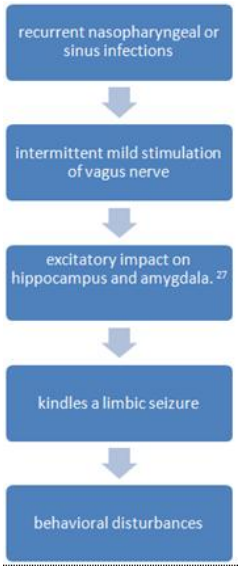


Figure 2

5 of the 24 patients with proposed limbic psychotic trigger reaction had a history of recurrent nasopharyngeal infections. 4 of these patients also had positive findings on MRI.

Traumatic Brain Injury

Most of the patients studied till date had some form of traumatic brain injury at some point in their lives. [24] Given the fact that frontal and temporal lobes are most susceptible to damage, [28]

The possible contribution of TBI cannot be ignored.

CASE HISTORY

My patient, 35 yr old female, Ms. XYZ, a diagnosed case of Limbic Psychotic Trigger Reaction, with no known co-morbids, was in usual state of health when she developed symptoms of physical and verbal aggression, over talkativeness, decreased sleep and decreased awareness of surroundings, visual disturbances and diplopia. The symptoms were slowly progressive with waxing and waning character, but never reached the baseline. All these symptoms started after an episode of sore throat and apthous ulcers. These symptoms persisted for three days before she was brought to the hospital. The morning before she was brought to the emergency of the Psychiatry Department, she threw her 8 month old child on the sofa and verbally as well as physically abused her husband and sister in law.

Her illness started 17 years back, when her first child was only 7 days old. As per the patient's account, she was at a party when she developed a strange anxiety, started feeling feverish, drowsy and developed profuse sweating and palpitations. The patient had visual hallucinations that someone wants to take her child away. Panicked, Ms XYZ fainted and hit her head on the ground. She soiled her clothes with urine. Behavioral disturbances started to manifest once she regained consciousness. Ms. XYZ used to run away from home, tear her clothes and tried to strangulate her child. The family members intervened, symptoms subsided and no medical help was sought at that time.

The second exacerbation occurred when Ms. XYZ was 25 yrs of age. By that time, she had 6 children, the youngest one 8 months old. The patient's own words," I started having vivid dreams, spaced a few days apart. I saw

that I have gone to a lake with my 6 children, where I lose the youngest 4 of the lot." So disturbed was she with this dream that the patient attempted suicide twice, once by ingesting insecticides and the other by trying to hang herself with a rope but was unsuccessful as family members intervened.

She again dreamt that her hands are blood tinged. That night she mixed sleeping pills in the food. Once everyone was asleep, Ms XYZ drowned her youngest four children in an underground water tank in her house. She then picked up a chisel and wounded her husband and the other two children. Upon being asked, she told her husband what she had done with a blunted, emotionless face. She was reported to the police. On realizing what she had done, she took full responsibility, had deep feelings of guilt and remorse and had no idea why she had committed the act. It was after this incident that the court requested the patient's checkup by a medical board to rule out psychiatric disease. Bender Gestalt Test, administered by doctors in civil hospital Karachi, revealed organist as the cause (as indicated by distortions and rotations in drawing). EEG performed at that time revealed seizure discharges over the right frontotemporal region on a background of alpha activity. These discharges persisted during eye opening, during and after hyperventilation and photic stimulation. MRI brain in 2007 revealed mucosal thickening in bilateral maxillary and sphenoid sinuses. The repeat MRI did in 2016 revealed maxillary sinusitis and no epileptogenic focus could be identified. All other lab tests were normal. A third episode occurred 2 years back, at the age of 33, when the patient left home, lost awareness of surroundings and was found from EDHI homes the next day. During the current (fourth) episode, the patient is severely agitated. She is preoccupied with guilt for killing her children. Her mood is labile. During the course of the day, her mood alternates from low with frequent weeping spells and suicidal ideation to sudden outbursts of excessive talking, over familiarity, aggression and self laughing. On mental state examination, the patient has no formal thought disorder or perceptual disturbance, no hallucinations or delusions. Her memory is intact and she has partial insight into her illness. While giving history, the patient often gets lost in details.

DISCUSSIONS

The symptoms of our patient fit most of the inclusion and exclusion criteria, for Limbic Psychotic Trigger Reaction described by Pontius.[1] Ms. XYZ had visual hallucinations, autonomic symptoms in the form of profuse sweating, urinary incontinence, flat affect and intact consciousness while committing infanticide. She has complete memory of the event step by step, no motivation, planning, no history of dementia or drug use and no family history of seizure disorder. Like all other patients of LPTR, Ms XYZ assumes full responsibility for infanticide and out of guilt and remorse, has developed severe depression with suicidal ideation and agitation. All her episodes have been preceded by subjective fever and some kind of upper respiratory complaint. No specific trigger stimulus could be isolated in our patient.

Due to slow response, an EEG was repeated in April, 22-2016. EEG revealed generalized low voltage sharp slow theta and delta waves on a background of theta waves –suggestive of diffuse cortical dysfunction; which suggests marked deterioration from her previous EEG.

The exacerbations are completely unpredictable, with the slightest of stimulus evoking the Psychotic Trigger Reaction. In our patient, there is a remote history of head injury. One notable finding in our patient is that there seems to be a relation between exacerbations and nasopharyngeal infections. We also suspect that she exacerbations are somewhat related to her pregnancies (with the episodes occurring 8 months, 8 months, 1.5 yrs and one year after delivery respectively). However, unlike most other reported cases, we could not find any repetitive stress in our patient, or any relation of the perception of hallucinations with a past traumatic event.

DIAGNOSTIC TESTS

- EEG: 2 EEGs done 9 years apart, both abnormal
- MRI: revealed maxillary sinusitis
- Luria's 3 step test: normal
- Alternating Hand movements: normal, but very slow
- Abstract thinking: impaired
- Perseveration test: abnormal
- Glabellar tap test: normal
- Written alternate sequence test: abnormal

Diagnosis: The patient was diagnosed as having Limbic Psychotic Trigger reaction. This fits into the following psychiatric diagnoses:

ICD-10: F07.8: Other Organic Personality and Behavioral disorders due to brain disease, damage and dysfunction.

DSM 4: Psychotic disorder due to a general medical condition; hallucinations and delusions occurring in paroxysmal episodes

DSM 3: Atypical Psychotic Disorder

TREATMENT

Ms XYZ was being treated with lamotrigine 100 mg for the past 10 years and was in remission and fully functional till 2 years back. After the current episode, Sodium Valproate was added to her treatment regimen. We also started her on escitalopram for depression and low dose haloperidol for aggression.

Patient Perspective Regarding her Illness

'I am not mad, just a bit ill. I have a headache, sore throat and visual disturbances'

Differential Diagnosis

Due to lack of any definitive tests, the following conditions must be ruled out before making a diagnosis of limbic psychotic trigger reaction.

- Temporal lobe epilepsy (discussed in the table below)
- Paranoid schizophrenia
- Gilles de la Tourette disease (vocal and motor tics associated with behavioral disturbance)
- Drug intoxication or withdrawal
- Personality disorders (narcissistic, borderline and antisocial personalities)

- Reflex epilepsy

Among the conditions described above that need to be ruled out, temporal lobe epilepsy bears most resemblance to LPTR, the differences however are outlined below.

Table 1

Limbic Psychotic Trigger Reaction	Temporal lobe epilepsy
Severe behavioral disturbances e.g. homicide, fire setting, rape	Milder aggression
Fully conscious during the episode	Clouding of consciousness
Complete recall of the events	Amnesia for the act
Associated with autonomic symptoms	Commonly associated with sexual problems
Usually visual or auditory hallucinations although one case of LPTR with olfactory hallucinations has been reported (the 23rd case which involved motiveless firesetting).[1]	Olfactory hallucinations usually seen
Vagal stimulation is believed to be an etiological mechanism	Vagal stimulation is sometimes used for treatment

CONCLUSIONS

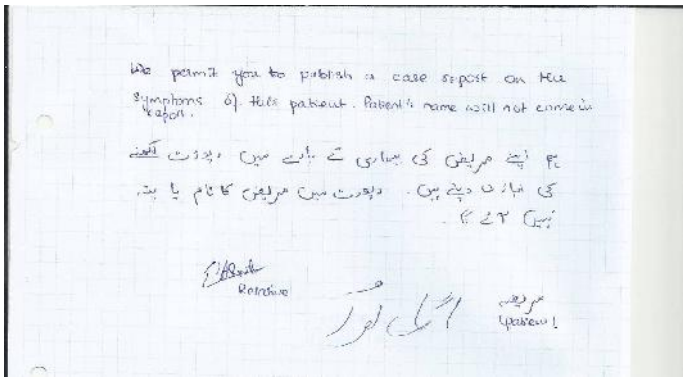
There is no standard single diagnostic test, and no known best treatment modality, although till date, it has been treated according to treatment guidelines for partial seizures.

Considering the unpredictable nature of behavioral disturbances, the danger that such patients present to themselves and those around them cannot be underscored. Due to lack of standardized treatment, poorly understood pathophysiological mechanisms, and unpredictability of results of diagnostic tests (EEG, MRI, PET scans), the rates of under reported cases is believed to be substantial.

As pointed out by Proust, there are probably many more "sleeper cases" with milder symptoms. These sleeper cases need to be actively sought for and monitored for disease progression. [29]

Increased awareness about the illness may help in timely diagnosis and prevent progression of disease. It may as well, bring to light as yet unrecognized manifestations of the disease.

CONSENT FOR REPORT


We permit you to publish a case report on the symptoms of this patient. Patient's name will not appear.

Relative

Witness

Figures

Bender Gestault Test

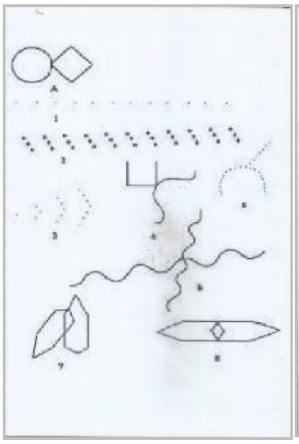
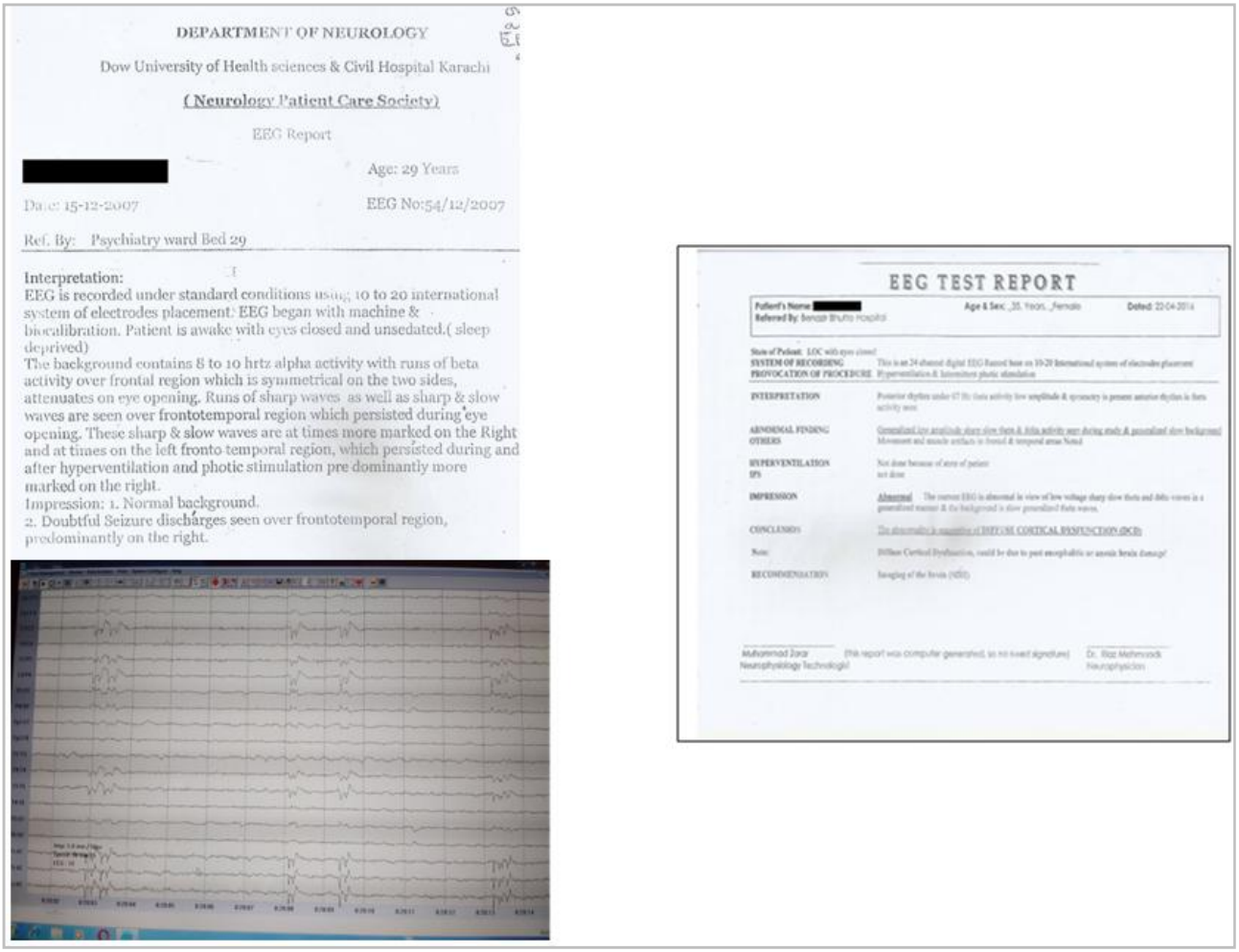


Figure 3 **Figure 4**

Electroencephalograms



DEPARTMENT OF NEUROLOGY

Dow University of Health sciences & Civil Hospital Karachi

(Neurology Patient Care Society)

EEG Report

[redacted] Age: 29 Years

Date: 15-12-2007 EEG No:54/12/2007

Ref. By: Psychiatry ward Bed 29

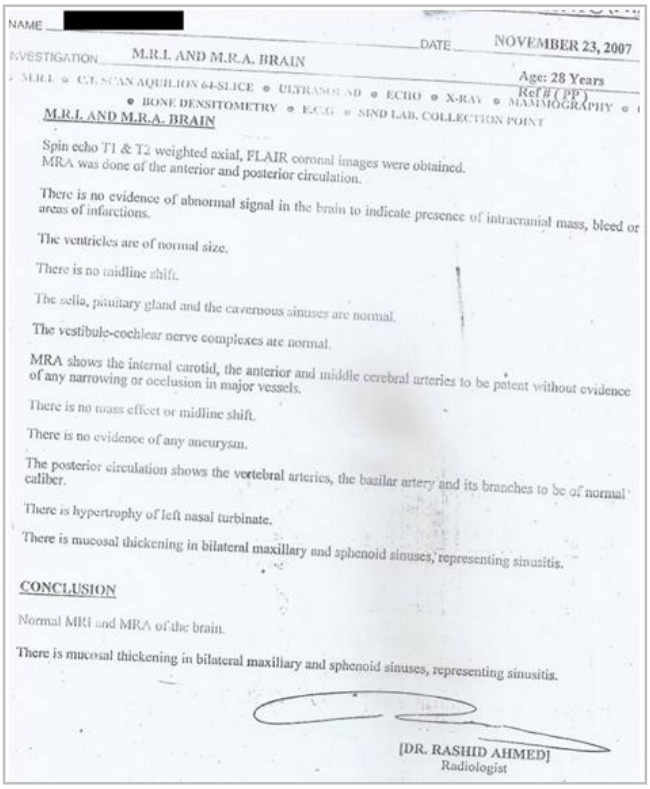
Interpretation:
EEG is recorded under standard conditions using 10 to 20 international system of electrodes placement. EEG began with machine & biocalibration. Patient is awake with eyes closed and unsedated.(sleep deprived)
The background contains 8 to 10 hrtz alpha activity with runs of beta activity over frontal region which is symmetrical on the two sides, attenuates on eye opening. Runs of sharp waves as well as sharp & slow waves are seen over frontotemporal region which persisted during eye opening. These sharp & slow waves are at times more marked on the Right and at times on the left fronto temporal region, which persisted during and after hyperventilation and photic stimulation pre dominantly more marked on the right.
Impression: 1. Normal background.
2. Doubtful Seizure discharges seen over frontotemporal region, predominantly on the right.

EEG TEST REPORT

Figure 5

MRI Reports

2007 and 2016



NAME [redacted] DATE NOVEMBER 23, 2007

INVESTIGATION M.R.I. AND M.R.A. BRAIN Age: 28 Years

Ref # (PF)

M.R.I. ● CT SCAN AQUILION 64-SLICE ● ULTRASOUND ● ECHO ● X-RAY ● MAMMOGRAPHY ● BONE DENSITOMETRY ● E.C.G. ● SIND LAB. COLLECTION POINT

M.R.I. AND M.R.A. BRAIN

Spin echo T1 & T2 weighted axial, FLAIR coronal images were obtained.
MRA was done of the anterior and posterior circulation.

There is no evidence of abnormal signal in the brain to indicate presence of intracranial mass, bleed or areas of infarctions.

The ventricles are of normal size.

There is no midline shift.

The sella, pituitary gland and the cavernous sinuses are normal.

The vestibule-cochlear nerve complexes are normal.

MRA shows the internal carotid, the anterior and middle cerebral arteries to be patent without evidence of any narrowing or occlusion in major vessels.

There is no mass effect or midline shift.

There is no evidence of any aneurysm.

The posterior circulation shows the vertebral arteries, the basilar artery and its branches to be of normal caliber.

There is hypertrophy of left nasal turbinate.

There is mucosal thickening in bilateral maxillary and sphenoid sinuses, representing sinusitis.

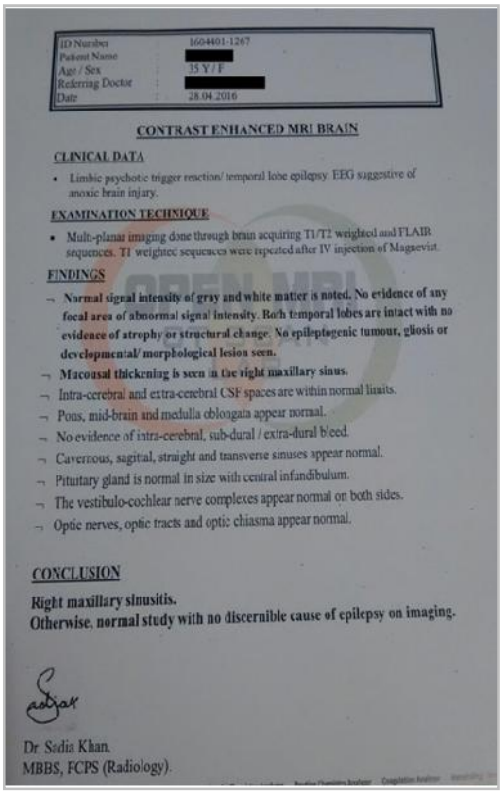
CONCLUSION

Normal MRI and MRA of the brain.

There is mucosal thickening in bilateral maxillary and sphenoid sinuses, representing sinusitis.

[DR. RASHID AHMED]
Radiologist

Figure 6



ID Number 160401-1267
Patient Name [redacted]
Age / Sex 35 Y / F
Referring Doctor [redacted]
Date 28.04.2016

CONTRAST ENHANCED MRI BRAIN

CLINICAL DATA

- Limbic psychotic trigger reaction/ temporal lobe epilepsy. EEG suggestive of anoxic brain injury.

EXAMINATION TECHNIQUE

- Multi-planar imaging done through brain acquiring T1/T2 weighted and FLAIR sequences. T1 weighted sequences were repeated after IV injection of Magnevist.

FINDINGS

- **Normal signal intensity of gray and white matter is noted. No evidence of any focal area of abnormal signal intensity. Both temporal lobes are intact with no evidence of atrophy or structural change. No epileptogenic tumour, gliosis or developmental/ morphological lesion seen.**
- **Mucosal thickening is seen in the right maxillary sinus.**
- Intra-cerebral and extra-cerebral CSF spaces are within normal limits.
- Pons, mid-brain and medulla oblongata appear normal.
- No evidence of intra-cerebral, sub-dural / extra-dural bleed.
- Cavernous, sagittal, straight and transverse sinuses appear normal.
- Pituitary gland is normal in size with central infundibulum.
- The vestibulo-cochlear nerve complexes appear normal on both sides.
- Optic nerves, optic tracts and optic chiasma appear normal.

CONCLUSION

Right maxillary sinusitis.
Otherwise, normal study with no discernible cause of epilepsy on imaging.

Dr. Sadia Khan
MBBS, FCPS (Radiology)

Figure 7

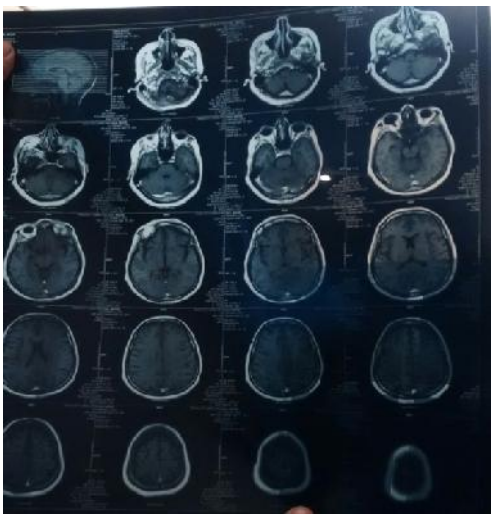


Figure 8

Written Alternate Sequence Test

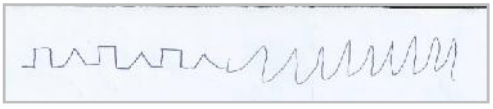


Figure 9

REFERENCES

1. Pontius AA, Wieser HG. Can memories kindle nonconvulsive behavioral seizures in humans? Case report exemplifying the "limbic psychotic trigger reaction". EpilepsyBehav. 2004 Oct; 5(5):775-83.
2. Pontius AA: Motiveless firesetting, implicating partial limbic seizure kindling by revived memories of fires in limbic psychotic trigger reaction. Percept Mot Skills 88:970 – 82, 199
3. Pontius AA: Limbically kindled arson in juveniles: memories of fire revived by fire-related stimuli. Presented at the 24th Annual Meeting of the American Academy of Psychiatry and the Law, San Antonio, TX, 1993
4. Sherman C: Limbically kindled seizures might provide the spark for some irrational acts of violence. ClinPsychiatr News 22:1–2, 1994
5. Pontius AA. Two Bankrobbers With “Antisocial” and “Schizoid/Avoidant” Personality Disorders, Comorbid With Partial Seizures: Temporal Lobe Epilepsy and Limbic Psychotic Trigger Reaction, Respectively. Journal of Developmental and Physical Disabilities. June 2001, Volume 13, Issue 2, pp 191-197
6. Pontius AA (1984) specific stimulus-evoked violent action in psychotic trigger reaction: a seizure-like imbalance between frontal lobe and limbic systems (Monograph 1-V59). Perceptual and Motor Skills: Volume 59, Issue, pp. 299-333.doi: 10.2466/pms.1984.59.1.299
7. Goddard, G. V. (1967). Development of epileptic seizures through brain stimulation at low intensity. Nature, 214,1020–1021
8. Mark, V. H., & Ervin, F. R. (1970). Violence and the brain. New York, NY: Harper and Row.
9. Wieser HG. Electroclinical features of the psychomotor seizure: a stereo-electroencephalographic study of ictal symptoms and chronotopographical seizure patterns including clinical effects of intracerebral stimulation. Stuttgart/London: G. Fischer–Butterworths; 1983
10. Gloor, P., Olivier, A., Quesney, L. F., Andermann, F., & Horowitz, S. (1982). The role of the limbic system in experiential phenomena of temporal lobe epilepsy. Annals of Neurology, 12, 129–142.
11. Goddard, G. V., & McIntyre, D. C. (1986). Some properties of lasting epileptogenic trace kindled by repeated electrical stimulation of the amygdala in mammals. In B. K. Doane, & K. E. Livingston (Eds.), The limbic system: Functional organization and clinical disorders (pp. 95–105). New York, NY: Raven.
12. Trimble MR, Mendez MF, Cummings JL J Neuropsychiatry Clin Neurosci. 1997 Summer; 9 (3): 42938. Neuropsychiatric symptoms from the temporolimbic lobes.
13. Wada JA. The clinical relevance of kindling: species, brain sites and seizure susceptibility. In: Livingston KE, Hornykiewicz O, editors. Limbic mechanism, the continuing evolution of the limbic system concept. New York: Plenum; 1978. p. 369–88.
14. Halgren E, Stapleton J, Domalski P, et al. Memory dysfunction in epilepsy patients as derangement of normal physiology. Advances in Neurology. New York: Raven Press; 1991;55: p. 385–410

15. Pontius AA. Homicides with partial limbic seizures: is a chemical kindling the culprit? Int J Offender Ther Comp Criminol 2001; 45 : 515–26
16. Pontius, A. A. (1981). Stimuli triggering violence in psychoses. Journal of Forensic Sciences, 26, 123–128
17. Pontius AA. Retroductive reasoning in a proposed new subtype of partial seizures evoked by limbic ‘‘kindling’’. Psychol Rep 1995; 76:55–6.
18. Cromie WJ. Brain changes in learning measured. HarvUnivGaz 2002;47:1–4
19. https://en.wikipedia.org/wiki/Limbic_system.
20. Nauta WJ. The problem of the frontal lobe: a reinterpretation. J Psychiatr Res. 1971 Aug; 8 (3): 167-87.
21. Pontius AA, Yudowitz BS. Frontal lobe system dysfunction in some criminal actions as shown in Narratives Test. J NervMent Dis 1980;168:111
22. Bear DM. Commentary to AA Pontius: psychotic trigger reaction: neuro-psychiatric and neuro-biological (limbic?) aspects of homicide, reflecting on normal action. Integrative Psychiatry 1987; 5:125–7.
23. Pontius AA, Psychological Reports, 1993, 73, 875-892. O Psychological Reports, 1993 Neuropsychiatric Update Of The Crime "Profile" And "Signature" In Single Or Serial Homicides: Rule Out Limbic Psychotic Trigger Reaction
24. Henriksen SJ, Bloom FE, Mccoy F, Ling N, Guillemin R. Proc. Nati. Acad. Sci. USA Vol. 75, No. 10, pp. 5221-5225, October 1978 Neurobiology f-Endorphin induces nonconvulsive limbic seizures (electroencephalogram/opiates)
25. Pontius AA, Marjorie JL Aggression in temporal lobe epilepsy and limbic psychotic trigger reaction implicating vagus kindling of hippocampus/amygdala (in sinus abnormalites on MRIs)
26. Paul Mc Lean. The tribune in brain evolution – Paleocerebral functions
27. Andrew Siegal, Nils. R. Varney. Failure To Recognize Neurobehavioral Disorders: The Gateway To "The Primrose Path"
28. Proust MA. La recherche du temps perdu. Paris: Editions Galimard; 1954 (original 1913). perpetrator