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RESEARCH ARTICLE

PSEUDOSEIZURES: A CASE REPORT.

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Abstract

Background: Pseudoseizures a disorder in which paroxysmal series of changed behavior are noticed, it misdiagnosed due to its similarity to true epilepsy.

Case presentation: A male patient of 22 years old with no medical illness admitted to emergency department with a history of repeated attack of involuntary movements affecting all four limbs, he has no medical or surgical history, his vital signs were recorded upon arrival, he experienced full examination and analysis, computed tomography, Electroencephalogram and Magnetic resonance image were performed on the patient.

Introduction:-

Pseudoseizures or Psychogenic Non-Epileptic Seizures (PNES) are disorders in which there are paroxysmal series of changed behavior that looks like epileptic seizures however it lacks organic causes such as dysfunction of central nervous system and the expected electro-encephalographical epileptic changes **[1]**. It is considered as somatoform disorder **[2].** Pseudoseizures diagnosis can be suspected by physical examination, personal and psychiatric history **[3],** however the exact and accurate diagnosis of Pseudoseizures is still a challenge **[4].** Its incidence was evaluated to be 6.5- 10.6 % in many studies **[5-8]** and 20 % of patients visiting the epilepsy centers **[9].** One from 5 patients with apparently epilepsy referred to specialist centers was found to suffer no organic reason for seizures but mistaken for epilepsy this is due to diagnostic error **[10],** so we must focus on history and symptoms of the patient to avoid the mistakes.

Case Presentation:-

The patient was a 22 years old male works as a military officer in the National Guard, he was not known to have any medical illnesses, he was admi0ed on 5th February 2017 AD as a case of query seizures versus pseudo seizures. The patient presented to our emergency department then transferred to neurology department. The patient has history of repeated attack of involuntary movements affecting all four limbs with neck hyperextension for three days each event lasting around 30 seconds. There were no preceding symptoms by an aura. He was aware of

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his surroundings during the event. There was no eye rolling, no tongue biting or loss bladder control (urinary incontinence). After each event he regained full consciousness and function. Trunk extension with opisthotonus movement with irregular arrhythmic asynchronous; jerky movement of all limbs with open eyes.

He initially presented to a different hospital where he stayed for two days then was discharged against medical advice. His systemic review was unremarkable. He was alcohol Consumer, his last drink was 3 days ago, also he was smoker (20 pack/year). He suffers from increased social pressure.

There was no past medical or surgical history and no history of drug or food allergy, he was not exposed to blood transfusion. The patient had an event which was witnessed by the neurology specialist. It was noted that the patient usually develops such event during physician rounds or visiting hours. Oxygen satura on was 96% during the event on room air, no dilatation or tachycardia were found and Plantars were downing. There was spontaneous recovery of consciousness without headache, vomiting or fatigue. There was no cranial nerve involvement, he has normal reflexes with intact motor and sensory functions, also cerebellar exam was normal and normal gait was found. According to Cardiovascular examination it was found that CVS: S1+S2+0, for chest examination it was found that B/L vesicular breath sound with no added breath sounds. Examining abdomen showed soft, Lax; no organomegaly and no palpable masses were felt.

His vital signs were as follow; T.: 36.2 C BP: 102/52mmHg P: 77bpm RR: 19breaths/min. spO2: 96% RA, RBS: 69 mgl/dl. Electrocardiography(ECG) showed Sinus Rhythm, his blood tests were found as follow; for complete blood count (CBC) his white blood cell count was 11×10^{9}/L, platelets was 211×10^{9}/L, ANC 7.04, HGB 15.5m MCV 81 and MCH 28, his coagulation profile showed that prothrombin time (PT), INR , aPTT were 13.9 sec, 1.04, 32.5 sec respec vely. By measuring kidney func on tests, Blood urea nitrogen(BUN)= 3.01mmol/L, Crea nine (Cr)=79 µmol/L, Na= 143 mmol/L, K= 4.1mmol/L, Cl= 106mmol/L, Ca= 2.3mmol/L, Mg= 0.8mmol/L, PO_4= 1.1mmol/L. Liver func on tests were; AST= 35 U/L, ALT= 29U/L, ALP= 110
U/L, Albumin= 38g/L, Amylase= 71 U/L, LDH =289 U/L. By estimation of Creatine kinas (CK) level=320U/L, CK-MB= 23 U/L, Prolac n = 314 nmol/ml, Thiamine level= 70 mmol/L, Vit B 12 level= 180 pg/ml, Venous blood gas = 155.

CT scan in brain was performed on patients' brain on the day of admission (5/2/2017) and it was normal. Electroencephalogram (EEG) was performed 9th February 2017 to find the problems related to electrical activity of the patients' brain, it was performed a1er giving 10mg IV midazolam as a sedative agent, results showed that it was essentially normal EEG recording during wakefulness, drowsiness and activating procedure demonstrating normal background with no abnormal focal slowing or epilliptiform discharges, also there was no clinical or electro graphic seizures. Excessive fast activities were noted which may be related to benzodiazepines that was given prior to this study.

Magnetic Resonance image (MRI) was performed 8th February 2017, the study was suboptimal to artifact produced by braces making evaluation of the images slightly difficult. There was no mass lesion or midline shift noted, hydrocephalus also was not found. Brain stem and both cerebellar hemispheres are unremarkable and both temporal lobes show normal signal intensity with no atrophic changes. We did not find cortical dysplasia or evidence of mesial temporal sclerosis also no diffusion restriction observed in diffusion weighted images. CP angle was unremarkable. Figures (1-3)

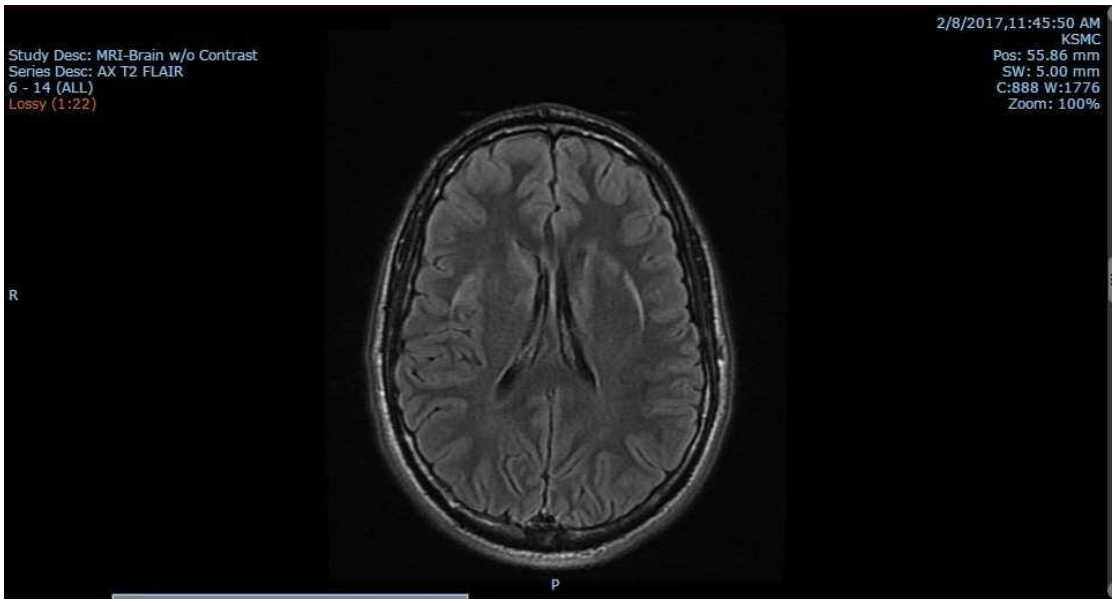

Fig.1.normal

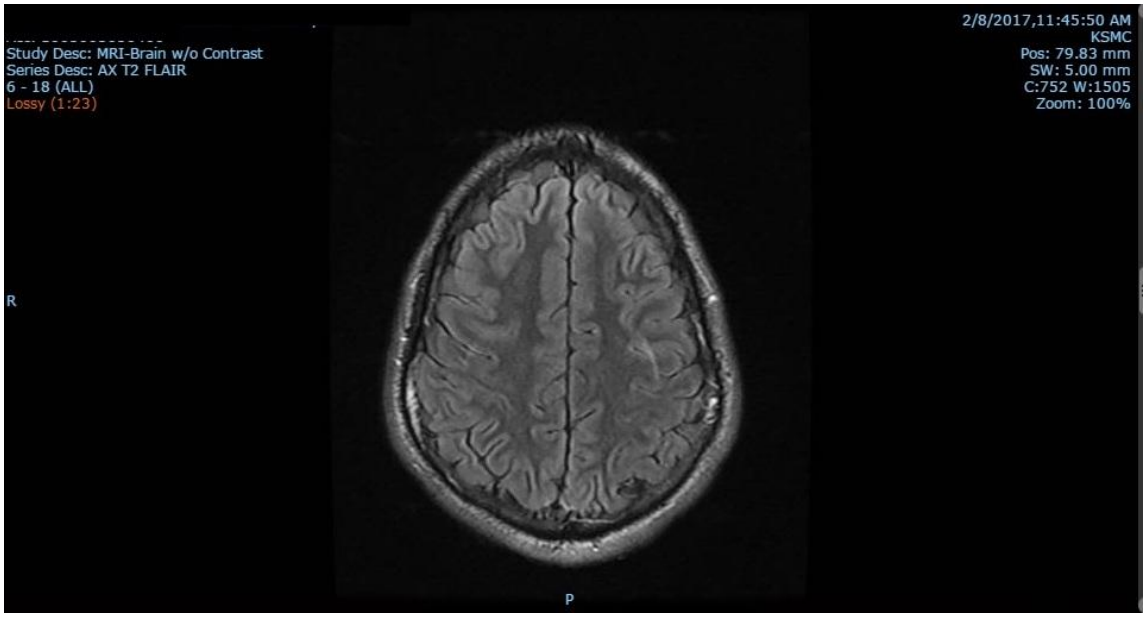

Fig 2 .normal

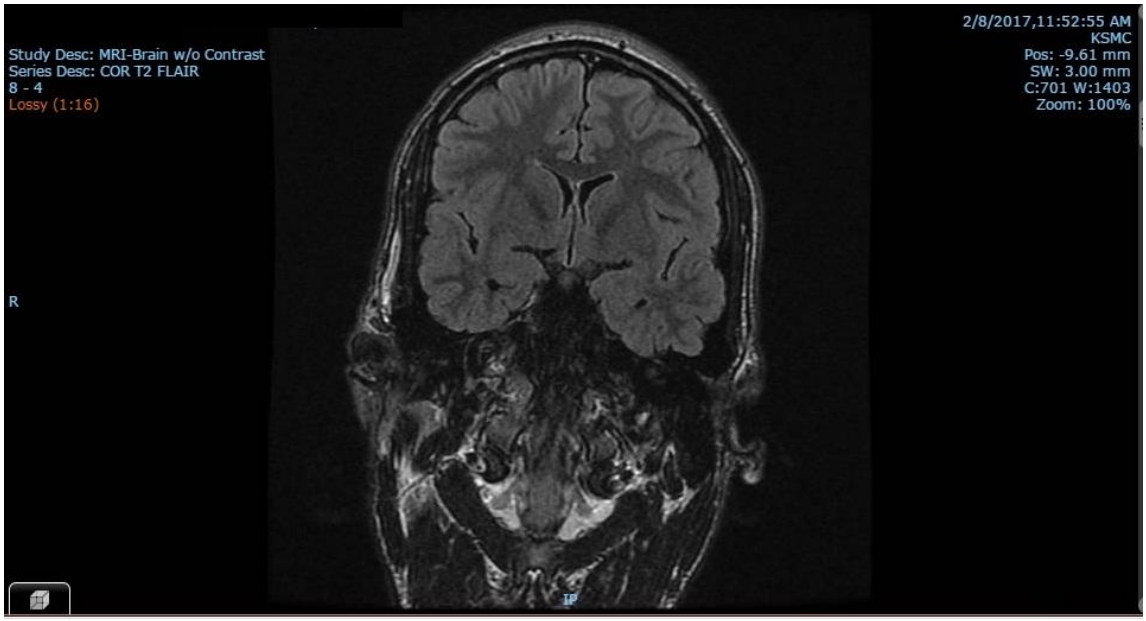

Fig.3.normal

A dose of 1 g of Phenytoin was given to the patient and he was put on a regular dose of 100 mg IV TID and Thiamine dose of 500 mg for three days. Folic acid dose of 1mg PO OD was given to the pa ent, D50 of 50 ml was given a**I**er thiamine injec on as a STAT dose. Keppra of 1 g PO BID was added a**I**er 2 days to op mize treatment if he was a true seizure patient. Also He was put on a placebo of 15 ml D5 water which he would stop his event a**I**er receiving the injection.

Discussion:-

The description of paroxysmal alterations in behavior that looks like epileptic seizures is called pseudoseizures but lack any organic cause **[9].** Pseudoseizures or Psychogenic Non-Epileptic Seizures (PNES) accounts for 17% to 30% of pa ents admi0ed to epilepsy units **[1].** Patients of pseudoseizures are often alcoholism and suffer pressure of performance **[9],** our patient was alcohol consumer and suffers from increased social pressure. The patient has normal laboratory tests and physical examination. Diagnosis of Pseudoseizures must be precisely recognized because mistake in diagnosis can be harmful **[9].** Actually it is very difficult to diagnose Pseudoseizures due to presence of overlapping in syndromes of real epilepsy with pseudoepilepsy **[11]**. There are many charachteristics of Pseudoseizures including no tongue bite, absence of urinary incontinence, normal pupillary reflex and normal pupil size where, it is dilated in organic seizures, occurrence of event in all times, especially when the patient is awake among all the relatives, or persons related to the issue**,** and plantar reflex is always flexor **[1,11].** In the present case almost all the previous conditions where exist with our patient, the patient did not suffer tongue biting and had no loss in bladder control, also there was no pupillary dilatation, the patient usually develops the events during physician rounds or visiting hours it was recorded that plantars were downing. All these indicate that this person was suffering pseudoseizures. He was suffering from repeated attack of involuntary movements affecting all his four limbs, but during the vent he was aware of his surroundings and he regained full consciousness and function after the event. Pupillary dilatation is not common, however it was observed a mild papillary dilatation **[11]**, in the present case there was no papillary dilatation. Bladder incontinence is rare in Pseudoseizures **[11],** our patient did not loss bladder control. The time for seizure episodes ranged from 4 seconds to 19minuts **[11],** in the present study the patients' neck hyperextension around 30 seconds every time.

Aura is unusual in pseudoseizures **[9]** this is what found in our patient who had no Aura. Serum prolactin concentration was found to be higher than 500 IU/ml in percent exceeding 90% of pa ents a**I**er a tonic-clonic seizure while it increases in 60% of pa ents a**I**er a complex partial seizure, prolactin does not increase after Simple partial seizures **[10],** in the present patient the Prolactin concentra on was 314 nmol/ml**.** Electroencephalogram (EEG) may be normal in 30% of epilepsy patients **[10],** however it is important to be

performed during the seizures to establish a correct diagnosis **[1]**. In the present case, normal EEG was recorded which demonstrating a normal background with no abnormal focal slowing or epilliptiform discharges. In our patient, his MRI showed no diffusion restriction in diffusion weighted images, no hydrocephalus or mass lesion or midline shift were noted**.** In conclusion we found that the present patient suffered Pseudoseizures, so misdiagnosis of Pseudoseizures is present due to similarity in some symptoms with true epilepsy**.**

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