Case Report

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Recognizing and Diagnosing Abdominal Epilepsy in Adult Male: A Case Study

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ABSTRACT

Abdominal Epilepsy (AE) is a very rare disorder; it is considered a category of temporal lobe epilepsies and was diagnosed by exclusion. AE is usually in the pediatric. However, there is a recorded case of its occurrence in adults. AE can present with unexplained, cruel, and recurrent gastrointestinal symptoms such as paroxysmal severe pain, nausea, vomiting bloating, and diarrhea that improve with antiepileptic therapy. It is commonly linked with electroencephalography (EEG) changes in the temporal lobes with symptoms that represent the involvement of the central nervous system (CNS) such as altered consciousness, confusion, and lethargy. That's Due to the mysterious nature of these symptoms; there is a greater chance of misdiagnosing of this case. We present the case of a 35-year-old man with AE who was misdiagnosed with psychogenic abdominal pain after doing multiple investigations with various medical branches in multiple hospital departments.

KEYWORDS: Abdominal pain; Abdominal Epilepsy; Temporal lobe epilepsies; Electroencephalography; Psychogenic

ABBREVIATIONS: AE: Abdominal Epilepsy; EEG: Electroencephalography; CNS: Central Nervous

INTRODUCTION

Abdominal epilepsy (AE) is a rare disorder, when seen in adults and presents with paroxysmal symptoms of abdominal pathology that result from seizure activity [1]. The range of this disorder is characterized by (a) unexplained, paroxysmal gastrointestinal symptoms; (b) symptoms indicate central nervous system (CNS) disturbance; (c) abnormal finding in the electroencephalography (EEG) with results specific for a seizure disorder; and (d) improvement of symptoms with anticonvulsant medications [2]. The history of abdominal epilepsy has reported only 36 cases in the English literature in the past 34 years [3]. Due to the rarity of reported cases, there is a precipitately needed for accurate diagnosis by increasing knowledge among physicians to avoiding misinterpretation of these symptoms as "functional" or "psychogenic" [4,5].

CASE PRESENTATION

The patient is a 40-year-old right-handed male. He presented to the gastrointestinal Clinic with recurrent episodes of severe acute epigastric abdominal pain for 2 months. These episodes were associated with nausea and sometimes vomiting followed by lethargy or confusion which lasted for a few minutes after each episode. Each episode of abdominal pain lasted for 15 to 20 minutes and occurred five to eight times per day. They were sudden onset and resolved spontaneously. His prenatal and natal developmental and past medical histories were unremarkable.

General and local examinations of the abdomen and nervous system examined did not show any significant findings. He was prescribed proton pump inhibitors and he was reviewed after one week. There's no significant improvement in his symptoms. His complete blood picture, stool analysis, 24 hr. Holter ECG, ultrasound abdomen as seen in Figure 1. Multisided post contrast CT Abdomen as seen in Figure 2. And upper GIT endoscopy with barium swallow studies was done as seen in Figure 3.

Multisided CT scan of urinary tract as seen in Figure 4. All the results were unremarkable. Following this he was referred to the immunity unit and he done multiple labs. Investigations as

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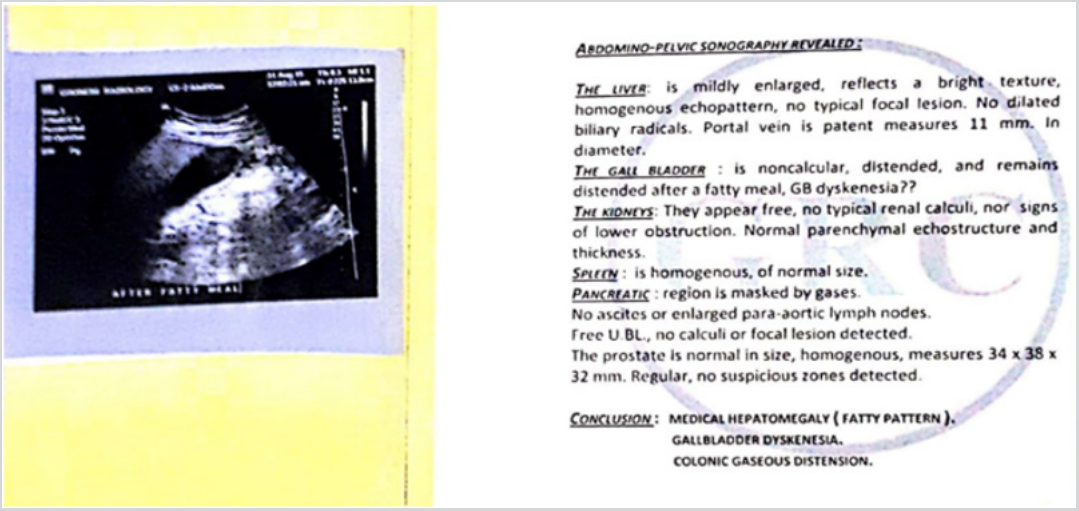
(ESR-CRP-Anti_DNA-Anti_CCP IgG-RNP_ab). All the results were unremarkable. Then he referred to the psychiatrist in view of psychogenic abdominal pain. He was taken antidepressants and antipsychotic medication without any improvement and he was referred to the neurology department for a second opinion. MRI brain was found to be normal as seen in Figure 5. At this time, epileptic etiology was suspected. a 30-minute awake EEG was made which revealed an abnormality in the form of repetitive sharp waves in the right temporal leads which were highly suggestive of an epileptogenic focus arising from the right temporal lobe as seen in Figure 6.

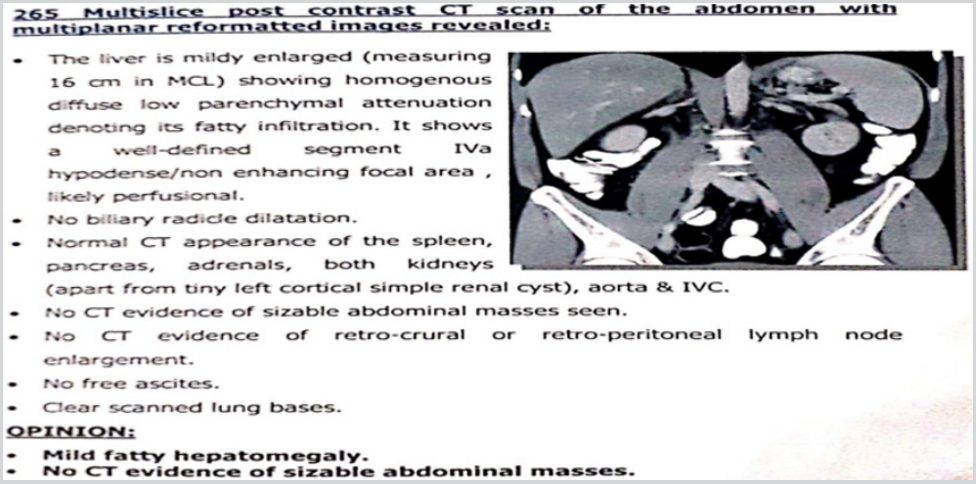


ABDOMINO-PELVIC SONOGRAPHY REVEALED:

THE LIVER: is mildly enlarged, reflects a bright texture, homogenous echopattern, no typical focal lesion. No dilated biliary radicals. Portal vein is patent measures 11 mm. In diameter.
THE GALL BLADDER : is noncalcular, distended, and remains distended after a fatty meal, GB dyskenesia??
THE KIDNEYS: They appear free, no typical renal calculi, nor signs of lower obstruction. Normal parenchymal echostructure and thickness.
SPLEEN : is homogenous, of normal size.
PANCREATIC : region is masked by gases.
No ascites or enlarged para-aortic lymph nodes.
Free U.BL., no calculi or focal lesion detected.
The prostate is normal in size, homogenous, measures 34 x 38 x 32 mm. Regular, no suspicious zones detected.

CONCLUSION: MEDICAL HEPATOMEGALY (FATTY PATTERN).
GALLBLADDER DYSKENESIA.
COLONIC GASEOUS DISTENSION.

Figure 1: Abdominal-pelvic ultrasound.



265 Multislice post contrast CT scan of the abdomen with multiplanar reformatted images revealed:

- The liver is mildy enlarged (measuring 16 cm in MCL) showing homogenous diffuse low parenchymal attenuation denoting its fatty infiltration. It shows a well-defined segment IVa hypodense/non enhancing focal area , likely perfusional.
- No biliary radicle dilatation.
- Normal CT appearance of the spleen, pancreas, adrenals, both kidneys (apart from tiny left cortical simple renal cyst), aorta & IVC.
- No CT evidence of sizable abdominal masses seen.
- No CT evidence of retro-crural or retro-peritoneal lymph node enlargement.
- No free ascites.
- Clear scanned lung bases.

OPINION:

- Mild fatty hepatomegaly.
- No CT evidence of sizable abdominal masses.

Figure 2: Multisided post contrast CT abdomen.

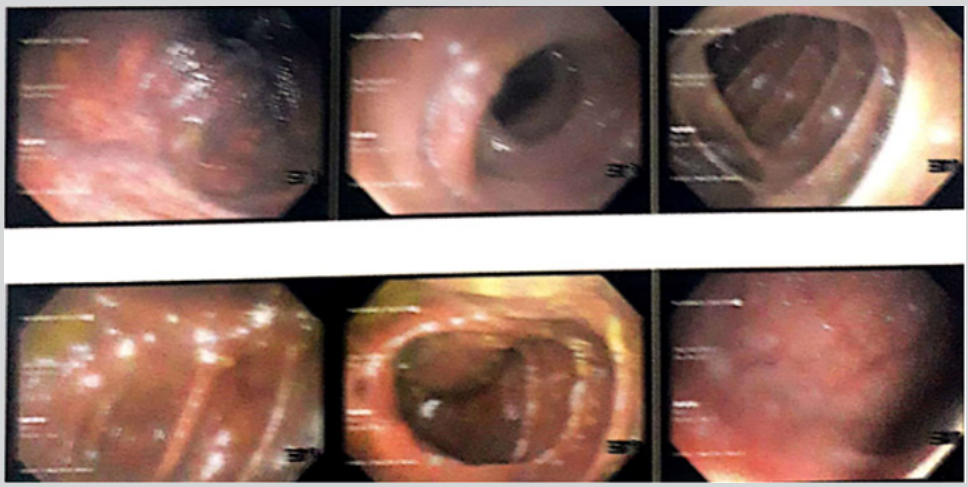
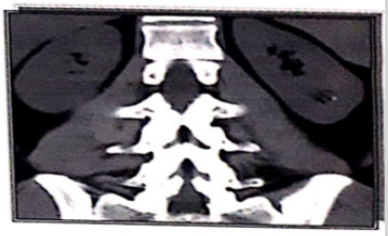


Figure 3: Upper GIT endoscopy with barium swallow.

REPORT

Multislice (64 channels) CT scan of the urinary tract with axial 3 mm sections and coronal reformatted images revealed:

- Tiny renal gravel is seen within the lower calyceal group of the left kidney.
- No urinary radio dense stones detected within the right kidney or along the abdominal or pelvic courses of the ureters or within the urinary bladder.
- Both kidneys are of average size showing smooth outline with preserved parenchymal thickness.
- No renal back pressure changes.
- Tiny prostatic concretions.
- The liver is of average size showing diffuse homogenous low attenuation of its parenchyma.



OPINION:

- **Tiny left lower calyceal renal gravel.**
- **Fatty liver.**

Figure 4: Multisided CT scan of urinary.

MRI of the brain using different pulse sequences in different planes:-

Techniques:

Plates 1, 2 & 3: Axial T1, T2 & FLAIR images.
Plate 4: Coronal T2 images.
Plate 5: Sagittal T2 images.

Findings:

- Diffuse mucosal thickening of all paranasal sinuses with bony rarefaction of the ethmoidal air cells, yet no solid evidence of intra-cranial extension.
- Mild dilatation of the ventricular system with no midline shift.
- Widened extra-axial CSF spaces.
- A small left external capsule about 3 mm area of high T2 and dark FLAIR signal intensity.
- No mass lesions.
- Normal cervico-medullary junction.

Conclusion:

- Pansinusitis, yet no solid evidence of intra-cranial extension, fungal etiology should be considered.
- Brain atrophic changes with left external capsule old lacunar infarction.

Truly yours;
Dr. Ahmad Rashad, MD

Figure 5: Brain MRI.

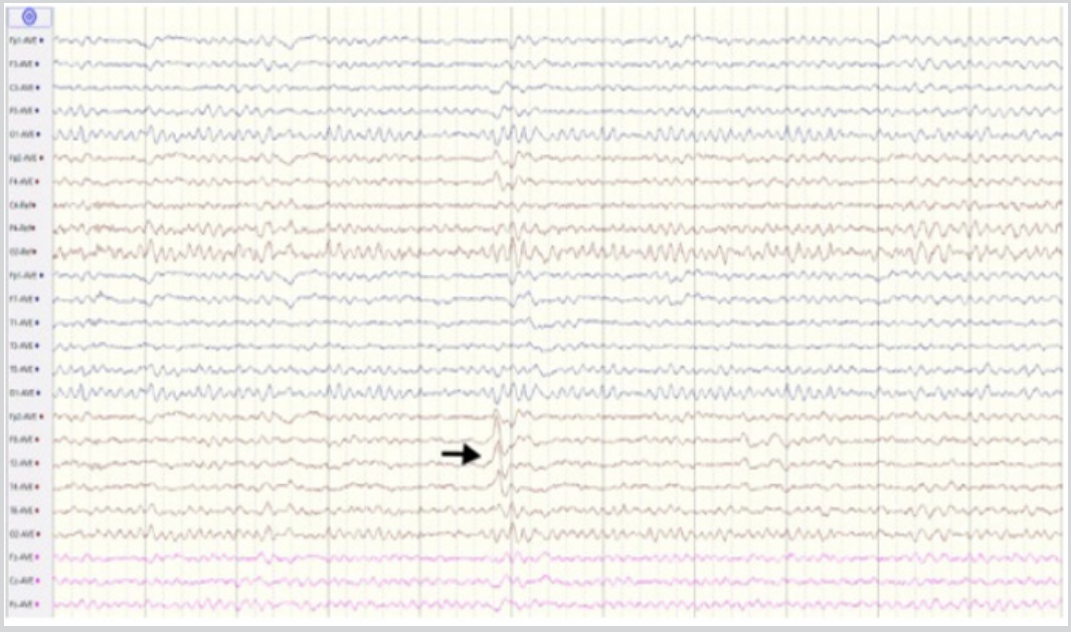


Figure 6: EEG findings of a 40-year-old male patient with abdominal epilepsy.

Then, the diagnosis of AE was made and he was started on oxcarbazepine 450mg BID. With regular follow-up visits every 2 weeks and was compliant with his medication. A follow-up over a 6-month period showed a progressive decrease in seizure frequency and has been asymptomatic.

DISCUSSION

AE is a rare disorder and has a vague nature of its symptoms; it is usually misdiagnosed or missed from diagnosis. It commonly occurs in the pediatric, but there is also documentation of its occurrence in adults [6]. It is characterized by unexplained, paroxysmal GI complaints, symptoms of a CNS disturbance, an abnormal EEG finding specific for a seizure disorder and improvement with anticonvulsant drugs [3]. AE has a variety of presenting symptoms. GI symptoms include paroxysmal pain, nausea, vomiting bloating, and diarrhea, whereas the CNS symptoms can comprise of dizziness, lethargy, headache, confusion, syncope, alternative levels of consciousness [3,7]. As AE is also a type of autonomic epilepsy, it can be associated with some autonomic symptoms such as pallor or cold sweating, dry mouth constipation [8]. Abdominal auras can also be found with manual and oral automatisms, that is, an auto motor seizure [9].

Although many explanations for abdominal epilepsy were outlined, the cause still remains unclear. We hypothesize that the Sylvain fissure and insular cortex lying right beneath it could be the origin of the seizure as they harmonize with the locations of the abdomen on the Sensory homunculus. Also, the M2 portion of the middle cerebral artery courses through the Sylvain fissure [10]. Any pathology of the vessel, particularly at this segment, could be assumed to play a role in epilepsies of the temporoparietal lobes. Phan et al. [11] recorded a case of ictal abdominal pain with parietal lobe hemorrhage and proposed the role of the somatosensory area I in pain perception. There have been previously reported cases of ictal abdominal pain with right parietooccipital encephalomalacia, biparietal atrophy and bilateral perisylvian polymicrogyria [12,13].

Recurrent abdominal pain is also seen in cases of visceral hyperalgesia, peptic ulcer disease and abdominal migraine [14,15]. The most common differential diagnosis for AE is abdominal migraine as they have many overlapping symptoms. Duration of the symptoms may be used to differentiate the two disorders; the duration is longer in abdominal migraine than in AE. The EEG result is usually abnormal in AE and may be used to confirm the diagnosis of AE [6].

Patients with AE may have the following possible features in the EEGs:

a) Patients may have normal EEG in the inter-ictal periods and diagnosis must not be purely based on an EEG [15].

b) Extra-temporal origins of epileptic foci; secondary generalization.

c) There are strong suggestions that EEG doing after the first 24 hours after the epileptic episode can detect abnormalities to a greater extent [16].

CONCLUSION

This case exhibits how conclusive diagnosis of AE can be challenging with longer time-consuming as the symptomatology is unclear and as the suspicion and incidence is low especially in adults. As AE might often be omission or misdiagnosed, it should be taken into account in patients presenting with episodic, recurrent and paroxysmal GI complaints along with symptoms suggestive of CNS disturbance that's do not improve with standard treatment guideline and after thoroughly excluding the more common causes in order to accurately diagnosis. Considering EEG, preferably a video-EEG for such patients so that their symptoms are not falsely labelled as psychogenic If the EEG findings are abnormal, the treatment involves the antiepileptic drugs with regular follow-up. 450 mg oxcarbazepine BID effective in the management of AE for our case.

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