Abdominal epilepsy in a Nigerian child



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Abdominal epilepsy is an exceptionally rare cause of abdominal pain that is more likely to occur in children than in adults. We report on a child with episodic paroxysmal abdominal pain, accompanied by flatulence, neck pain, tiredness and bilateral weakness of the lower limbs. The findings on physical examination were normal except for Mongolian spots. Haematological investigations, radiographs and an ultrasound scan were normal. The electro-encephalogram showed temporal lobe dysrhythmia during a typical attack. The patient responded well to carbamazepine and remained asymptomatic during the 6 months prior to our writing this article, while taking her treatment regularly.

Abdominal epilepsy (AE) is an extremely rare syndrome of epilepsy that is more likely to occur in children than adults. Gastro-intestinal complaints, most commonly abdominal pain, result from seizure activity.¹ The syndrome is characterised by: (*i*) otherwise unexplained, paroxysmal gastro-intestinal complaints; (*ii*) symptoms of a central nervous system (CNS) disturbance; (*iii*) an abnormal electro-encephalogram (EEG) with findings specific for a seizure disorder; and (*iv*) improvement with anticonvulsant medication.^{1,3} To the best of our knowledge, a clinical syndrome in keeping with abdominal epilepsy has hitherto not been reported in Nigeria.

Case report

We report on a 4-year-old girl who was diagnosed with AE at Federal Medical Centre Azare, Nigeria. Her problem started at the age of 2.5 years, and she was repeatedly admitted with recurrent paroxysmal abdominal pain for 18 months before the diagnosis was made. The abdominal pain seemed to be generalised, but further details could not be ascertained because of the patient's age. The attacks occurred suddenly, resolved spontaneously and lasted for 20 minutes to 1 hour. There was no associated headache, convulsions or loss of consciousness, but each episode was usually followed by flatulence, tiredness, nuchal pain and brief parapheresis with hypotonia of the lower limbs. No sensory deficit was detected.

The findings on examination were essentially normal except for two hyperpigmented spots (Mongolian spots) on the back and thighs. Several diagnoses were made at different times, including irritable bowel syndrome and suspected lead poisoning. The patient underwent exhaustive investigations including stool examinations for ova and parasites, a full blood count, haemoglobin genotyping and a blood film.

Other investigations included an abdominal ultrasound scan and upper and lower gastro-intestinal tract barium studies. No abnormalities were found. Finally an EEG done during an episode of the abdominal pain revealed left temporal dysrhythmia.

The patient did well on an anticonvulsant (carbamazepine) and has been free of symptoms for the 6 months prior to our writing this article. She has been taking her treatment regularly and has resumed school. Repeat EEGs done during the first and third months of treatment were essentially normal.

Discussion

AE is an uncommon cause for abdominal pain in children and adults. Although its symptoms may be similar to those of functional gastro-intestinal abnormalities, detailed history taking and a high index of suspicion enable it to be distinguished from the latter conditions. The most common gastro-intestinal symptoms are abdominal pain, nausea and vomiting, while the most common neurological symptoms are lethargy and confusion. After exclusion of more common causes for the presenting complaints, work-up should proceed with an EEG. Treatment typically begins with anticonvulsant medication, and resolution of symptoms with therapy helps to support the diagnosis. Our case fits the above scenario except that the patient had the additional central nervous symptom of post-ictal bilateral weakness of the lower limbs.

AE is well documented among children in the Western world but is infrequently recognised among African children. The paucity of reports in our setting could result from failure to recognise this condition or lack of diagnostic aids such as EEG, computed tomography and magnetic resonance imaging to support the diagnosis when it is suspected. Our patient only had an EEG; the other two investigations were not done owing to lack of funds.

The spectrum of gastro-intestinal disturbances reported in AE includes recurrent abdominal pain, nausea, vomiting, bloating and diarrhoea, or combinations of these. Similarly, diverse CNS symptoms reported have included confusion, fatigue, headache, dizziness and syncope. 1,2,5 Our patient presented with paroxysmal abdominal pain and flatulence, and the CNS manifestations were tiredness, neck pain and bilateral lower limb weakness.

An EEG is a simple, non-invasive and cheap investigation that may be helpful in differentiating AE from other diagnoses, since patients with AE usually have specific EEG abnormalities, particularly of a temporal lobe seizure disorder. The EEG abnormality in our patient was a burst of temporal lobe dysrhythmia. This is in agreement with previous reports suggesting that the most common EEG findings in patients with AE are bursts of sharp waves and/or spikes from one or both temporal regions. Other EEG findings that have been reported include generalised spike and wave discharges and frontal lobe discharge. Other EEG findings that have been reported include generalised spike and wave discharges and

One of the accepted criteria for the diagnosis of AE is sustained response to anticonvulsants.¹⁻⁴ Anticonvulsants that have been shown to be effective include carbamazepine, phenytoin and

sodium valproate.¹ Carbamazepine was tolerated well by our patient. She had control of symptoms within 1 month of starting treatment and was symptom free at 6 months' follow-up.

We suggest that AE should be suspected in African children with paroxysmal episodes of abdominal pain and flatulence associated with CNS symptoms during or after the attack. A high index of suspicion is key to its diagnosis, and an EEG should be performed, preferably during an attack, after excluding other possible causes. Treatment with an anticonvulsant such as carbamazepine is usually effective.

References

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