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Pupillary hippus as a component of postattenuation neurologic signs in a dog

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Background

Post-attenuation neurological signs (PANS) are a well-documented complication following the partial or complete surgical attenuation of a congenital extrahepatic portosystemic shunt (EHPSS) in dogs. PANS has been defined as manifestation of any postoperative neurologic signs between surgery and discharge, most commonly within the first seven days of surgery, and may include seizures or more subtle signs such as behavioural changes, depression, ataxia or tremors.¹ PANS is considered distinct from hepatic encephalopathy (HE), as it can occur despite full ligation of the EHPSS, is refractory to medical management for HE and occurs despite often normal blood ammonia concentrations.²

Pupillary hippus (also known as pupillary athetosis and pupillary nystagmus) is a well-recognised phenomenon in human medicine and has been defined as exaggerated constant motion of the pupil that is spasmodic, cyclic and bilaterally in phase, and occurs in the absence of changes in external influence such as luminance, mood and fixation.³ Although the underlying mechanism is poorly understood, it has been reported as a feature of non-convulsive status epilepticus⁴, vestibular migraine⁵ and as a predictor of early mortality in hospitalized patients.⁶

Case Description

A four-year-old male neutered Pug dog presented with a two-month history of polyuria-polydipsia, sporadic vomiting and hyporexia. Bile acid stimulation testing showed elevations of both pre-prandial (124.1 umol/L; ref. 0.0-14.9) and post-prandial (>180.0 umol/L; ref. 0.0-29.9) bile acid concentrations. Fasting blood ammonia was normal (39.0 umol/L; ref. 0.0-57.0). Computed tomography angiography identified the presence of a left gastro-azygos EHPSS. After two weeks of medical management, the dog was re-admitted for the purposes of surgical shunt management. A midline celiotomy was performed and a 5mm ameroid constrictor was placed around the shunt on the abdominal side at the level of the esophageal hiatus.

Initial recovery from surgery was uneventful. However, 48-hours later the dog became acutely obtunded and suffered generalised tonic-clonic seizures. Interictal neurological examination documented marked obtundation with intermittent periods of dysphoria and hyperaesthesia, absent menace responses bilaterally with cortical blindness and rhythmic oscillatory dilation and constriction of both pupils (**Fig. 1, Supplementary Video**). The combination of clinical signs was consistent with a diagnosis of PANS.



Treatment was initiated with diazepam (0.5mg/kg IV, twice) and levetiracetam (60mg/kg IV). As two further generalised tonic-clonic seizures occurred, phenobarbitone (3mg/kg IV q12hrs) was started alongside levetiracetam (60mg/kg IV q8hrs), as well as a dexmedetomidine continuous rate infusion (4mcg/kg/hr) to control inter-ictal dysphoria and vocalization. Supportive treatment otherwise consisted of lactulose (5ml PO q12hrs), omeprazole (1mg/kg IV q24hrs) and metoclopramide continuous rate infusion (0.5mg/kg/24hrs). No further seizures were witnessed in hospital following the addition of phenobarbitone, and the pupillary abnormalities resolved within 24 hours. Neurologically, the dog returned to normal over the following 8 weeks .

Discussion

The most widely reported cause of pupillary hippus in humans is those with non-convulsive status epilepticus.^{4, 7-8} A cluster of generalised tonic-clonic seizures were a feature of the PANS of the current case, which resolved following initiation of phenobarbitone treatment. It is possible that the hippus in the present case could have represented non-convulsive seizure activity, however electroencephalography (EEG) was not performed to interrogate this. However, the other PANS signs resolved spontaneously over varying timeframes, independent of anti-seizure medication. It is therefore also possible that the pupillary hippus was an independent neurological sign that was self-limiting in nature.



Video here!

References

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