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Portosystemic Encephalopathy in a Dog

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Abstract

Portosystemic shunt is an abnormal vascular connection between the portal and systemic venous system. Due to this abnormal network of vessels, portal venous blood and its toxic by-products by-pass the liver and directly mix into the systemic circulation. It is a well-known congenital cause of encephalopathy which is characterized by high ammonium levels. Our case; a two-year-old male dog was brought to our clinic with tonic-clonic epileptic seizures and allotriophagia presenting for the last four months.Diagnosis of portosystemic shunt was made, and a surgical operation was planned. After the operation, ammonium levels decreased sharply. However, ammonium levels have started to rise again the month following the procedure and leaded to more severe symptoms. Consequently, the patient was euthanized after three months.

Keywords: Portosystemic shunt; Encephalopathy; Hepatic encephalopathy

Introduction

Portosystemic shunt is an abnormal venous connection between the portal system and systemic circulation; this alternative route causes by-passing of blood and its toxic metabolites through the liver and directly drain into the systemic venous circulation. Many different types of congenital port vascular anomalies have been reported in dogs [1]. The incidence of congenital portosystemic shunt has been reported as 0.18% of in dogs[2]. Extrahepatic portosystemic shunts are usually seen in small-breed dogs. Affected dogs generally show symptoms before the age of two, but some patients do not develop clinical symptoms until they are older[3]. The most common symptoms are anxiety, lethargy and apathy while intrahepatic portosystemic shunts and cirrhosis cause hepatic encephalopathy more commonly[4,5]. Ultrasonography, magnetic resonance imaging and/or computed tomography were used for definitive diagnosis [6].

Case History

Two-year-old, male, mix-breed dog presented with an epileptic seizures and suspicion of foreign body ingestion. Anamnesis revealed that he had abnormal activities such as allotriophagia and increased aggression for the last four months. Body temperature, respiratory and pulse rate were in the normal range but there was abdominal discomfort during palpation. While the CBC's results were within normal ranges, serum ALT, AST and ALP levels were doubled. The serum ammonium level (SAL) was high (376 mmol/L).Urinalysis revealed proteinuria (2+) and bilirubinuria (2+), significant amount of ammonium biurate crystals were present in the urinary sediment. CT revealed multiple foreign bodies in the stomach stones in the bladder and both kidneys (Figure1). Generalized epileptiform waves were observed in electroencephalography (Figure 2).According to our findings, diagnosis of portosystemic shunt was made, and an operation was planned in order to correct it surgically.

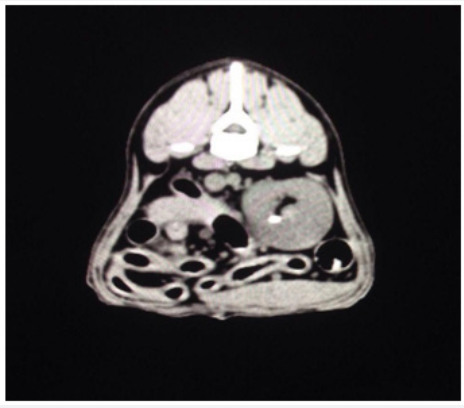

Figure 1: Stone detected in kidney by CT.

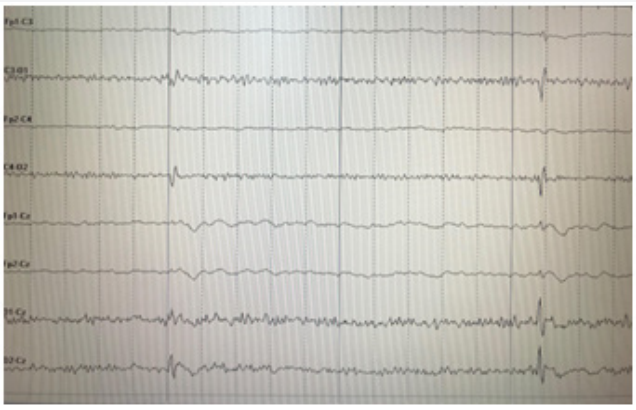

Figure 2: Generalized epileptiform discharged waves in electroencephalography.

Supporting therapy was begun immediately after the operation. Lactulose (5mls per 2.5 lbs. qid) was used for binding the ammonia present in the intestines. Phenobarbital (2mg/kg Bid) was given to inhibit the tonic-clonic epileptic seizures. After the operation SAL was decreased rapidly to 122 mmol/L. But the patient died three months after the operation. During the post mortem examination; stones of 4 mm diameter were detected in the kidneys and in the urinary bladder. There was also micro hepatica, portal fibrosis, thyroid gland degeneration and chronic interstitial nephritis.

Discussion

Porto-systemic encephalopathy is a reversible, complex neuropsychiatric syndrome characterized by disturbances in consciousness and behavior, personality changes and fluctuating neurological signs and distinctive electroencephalographic changes, which occur secondary to chronic liver disease[7,8]. Operative ligation of portosystemic shunts is effective in controlling chronic portosystemic encephalopathy, but also holds a high mortality rate[9,10]. In our patient presenting with high SAL and tonic-clonic seizures, we suspected presence of a portosystemic shunt. Surgery revealed an extrahepatic portosystemic shunt and it was corrected. However, the results were not satisfying; ammonium levels have started to rise again a month after the procedure and even leaded to more severe symptoms. We believe that there was also intrahepatic microvascular dysplasia. Quality of life was deteriorated day-by-day and patient was euthanized in accordance with the request by care-givers three months after the surgery.

Conclusion

Surgical correction of the portosystemic shunt may provide a cure for extra-hepatic portosystemic shunt induced encephalopathy, but success of treatment is low in the presence of portal vein hypoplasia, microvascular intrahepatic shunt or cirrhosis.

References

1. Van Straten G, Leegwater PAJ, De Vries M, Van Den Brom WE, Rothuizen J (2005) Inherited congenital extrahepatic portosystemic shunts in Cairn terriers. J Vet Intern Med 19(3): 321-324.
2. Tobias KM, Rohrbach BW (2003) Association of breed with the diagnosis of congenital portosystemic shunts in dogs: 2,400 cases. JAVMA 223(11): 1636-1639.
3. Worley DR, Holt DE (2008) Clinical outcome of congenital extrahepatic portosystemic shunt attenuation in dogs aged five years and older. JAVMA 232(5): 722-727.
4. Poordad FF (2007) The burden of hepatic encephalopathy. Aliment Pharmacol Ther 25: 3-9.
5. Van Straten G, Van Steenbeek FG, Grinwis GC, Favier RP, Kummeling A, et al. (2014) Aberrant expression and distribution of enzymes of the urea cycle and other ammonia metabolizing pathways in dogs with congenital portosystemic shunts. 9(6): e100077.
6. Tivers MS, Handel I, Gow AG, Lipscomb VJ, Jalan R, et al. (2014) Hyperammonemia and systemic inflammatory response syndrome predicts presence of hepatic encephalopathy in dogs with congenital portosystemic shunts. Plosone 9(1): e82303.
7. Maddison JE (1988) Canine congenital portosystemic encephalopathy. Aust Vet 65(8): 245-249.
8. Martin RA (1993) Congenital portosystemic shunts in the dog and cat. Vet Clin Nort Am Small Ani Pract, 23(3): 609-623.
9. Greenhalgh SN, Dunning MD, McKinley TJ, Goodfellow MR, Kelman KR, et al. (2010) Comparison of survival after surgical or medical treatment in dogs with a congenital portosystemic shunt. JAVMA, 236(11): 1215-1220.
10. Kraun MB, Nelson LL, Hauptman JG, Nelson NC (2014) Analysis of the relationship of extrahepatic portosystemic shunt morphology with clinical variables in dogs. JAVMA 245(5): 540-549.

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