

犬隻黃麴毒素中毒病例報告

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摘要

97 年底台北縣某私人動物收容所內犬隻發生不明原因死亡，臨床症狀呈現厭食、消瘦與活力差等症狀，部份犬隻未顯現症狀即暴斃，12 月共送檢 26 頭死亡犬隻，就微生物學、有機磷、氰化物及病理學檢驗，並未檢出具病原性微生物，有機磷及氰化物檢驗呈現陰性，而病理學檢驗顯示毒性肝病之特異病變，剖檢可見皮下組織黃疸、出血、胃腸道管腔內充滿暗紅色血液、肝臟腫大呈黃白色至墨綠色，肝表面呈光滑至粗糙堅實以及膽汁鬱積致膽囊膨脹等變化；組織病理學檢查，可見不同程度的肝細胞壞死、變性與再生、纖維組織增生與膽管增生，亦可見明顯的再生性肝細胞結節的形成。飼料進行檢驗後發現存有黃麴毒素，檢驗值高達 151 至 238 ppb，綜合各項檢驗以及犬隻的臨床、剖檢與組織病變，診斷為毒性肝病，本報告結果與 Newman 等人於 2007 年發表之報告相近，分析該場犬隻死亡原因，係因攝入過量黃麴毒素致中毒死亡，機制為中毒性肝病引發肝衰竭、凝血不全、胃腸大量出血而導致出血性休克。全面改換保肝處方飼料後，犬群死亡頭數於 1 個月後明顯逐漸降低，表示犬隻肝臟具有強大修補能力，不同於人類肝硬化之預後，此發現值得後續追蹤及研究。

A case report of aflatoxicosis in dogs

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Abstract

An outbreak of unknown disease in dogs at the private animal shelter in Taipei County from late 2008 was investigated. Affected animals showed anorexia, weight loss and dullness but some dogs died suddenly without the appearance of clinical signs. 26 dogs were submitted to autopsy in December from the shelter and the examinations of pathogenic micro-organisms, organic phosphorus and cyanide were all negative. Surprisingly, the pathological examination revealed toxic hepatitis by the noted typical lesions. At necropsy, jaundice and hemorrhage in the subcutaneous tissue, gastrointestinal lumen filled with dark red blood were noted. Livers were moderately to severely enlarged, icteric and had pale yellow to blackish green and rough surfaces. Gall bladders were distended and had inspissated bile. Histopathologic findings included necrosis, degeneration and nodular regeneration of hepatocytes, perinodular fibrous tissue proliferation and biliary hyperplasia, which supported a diagnosis of toxic hepatitis. Analysis of feed confirmed exposure to aflatoxin. The analysis of stocked feed for aflatoxin revealed high levels of aflatoxin B1 (153 ppb, 238 ppb, 151 ppb, 192 ppb respectively). By examinations as mentioned above, the etiology and the sudden death of toxic hepatitis was due to excessive intake of aflatoxin. These results were similar to the scientific report published by Newman *et. al.* in 2007 . After aflatoxin exposure, the affected dogs presented with liver failure, hemorrhagic diatheses and clotting abnormalities led to hemorrhage in gastrointestinal lumen and then hemorrhagic shock. The mortality was declining gradually and markedly within 1 month since the commercial feeds containing drug prescription for hepatic protection were used. It means dogs have powerful and effective ability of liver repairs and the prognosis of liver damage was different from human cirrhosis. These results are worth keeping studying in the future.