pISSN 1598-298X
*J Vet Clin* 31(4) : 316-318 (2014)

Journal of Veterinary Clinics
www.ksvc.or.kr

# A Case of Portosystemic Shunt in a Domestic Shorthair Cat

**Eunkwang Cho, Ye Chan Jeong and Ul Soo Choi[1]**

*Bio-Safety Research Institute · College of Veterinary Medicine, Chonbuk National University, Jeonju-si, Jeollabuk-do 561-756, Korea*

(Accepted: August 11, 2014)

**Abstract :** A 3-year-old castrated male domestic shorthair cat was presented with a chief complaint of sudden onset of intermittent seizures occurring five times a day. Physical examination revealed the copper colored iris and loss of menace response at both eyes. Abnormalities of blood works and serum chemistry revealed mild erythrocytosis, severe microcytosis, and threefold increase in ALT activity. Additional liver function tests results were increased bile acid and $NH_3$ concentration. Radiographic study revealed multifocal nodules of the liver and an extrahepatic shunt was noted by ultraonography, which was confirmed by computed tomography as multiple extrahepatic shunts. The cat was scheduled for surgery applying an ameloid ring to occlude the shunt gradually. Diazepam and lactulose were instituted to the patient. However, clinical signs worsened despite medical management with shortened interval of seizures and the patient died due to cardiac arrest.

***Key words*** **:** portosystemic shunt, extrahepatic, Domestic shorthair cat.

## Introduction

In portosystemic shunt (PSS), portal blood bypasses the liver via systemic collateral vessels (13). Such vessel abnormality can be congenital or acquired condition that results from portal hypertension caused by severe liver cirrhosis or fibrosis (17).

Certain breeds seem to be predisposed to congenital portosystemic shunts in dogs (14), and in cats, Persian and Himalayan have been reported to be at increased risk for congenital PSS in small case series (8,16). In a report of 49 cats, median age was eight months old (6). Unlike dogs, main chief complaints of feline patients are neurologic signs associated with hepatic encephalopathy that includes ptyalism, intermittent seizure, ataxia, disorientation and blindness (1,17).

In laboratory tests, findings in some cats usually include low serum urea concentration, mildly increased liver enzyme activity, erythrocyte microcytosis, and increased serum bile acid and ammonia concentration (1,2,17). In radiography, liver size can be small (1,10). A definitive diagnosis is based on results of mesenteric portography or ultrasonography (8,10,17).

This paper describes a case of feline portosystemic shunt and review the significance of laboratory tests in screening cats with PSS.

## Case

A 3-year-old castrated male domestic shorthair cat was presented with a chief complaint of sudden onset of intermittent seizures. The seizures repeated at one or two hour intervals and lasted one minute. Physical examination revealed copper color iris and loss of menace response at both eyes. Blood works and serum chemistry revealed mild erythrocytosis ($11.34 \times 10^6$/uL, reference range 5.00-11.00 $\times 10^6$/uL), severe microcytosis (31.5fl, reference range 39.0-52.0fl), and increased ALT activity (314 U/L, reference range 12-130 U/L) (Table 1). Increased central pallor in RBCs was noted in blood film examination. Because the patient did not show clinical findings consistent with iron deficiency and chronic disease, further liver function tests were performed to rule in the possibility of portosystemic shunt.

Fasting ammonia concentration was 98 umol/L (reference range 0-95 umol/L) and preprandial and postprandial serum bile acid concentration was 16 umol/L (reference range 0-25 umol/L) and 52 umol/L (reference range 0-25 umol/L) respectively. Abdominal radiographs revealed normal liver size. Multifocal nodules in the liver and shunt vessel that connected to the caudal vena cava were identified by ultrasonography. Computed tomography angiography had been used in the identification of origin of the shunt. The shunting vessel originated from portal vein to caudal vena cava (Fig 1). The patient was scheduled to undergo surgery for application of an ameloid constrictor to the shunt vessel. Prior to surgery, diazepam (0.3 ml, IV) and lactulose were administered to manage seizure and hepatic encephalopathy. Despite medical management, interval and frequency of seizure was more shortened than before and the patient died due to cardiac arrest.

## Discussion

Congenital portosystemic shunt (CPSS) in cats are uncommon diagnosis with a reported incidence of 2.5 per 10,000 cats according to one report (9). No known incidence was reported in South Korea, although the number of companion cats has been increasing. The first CPSS in cats was reported only in 1980 in veterinary literature, and large case series were published since 1992. To the best of the authors' knowledge, this is the first documented feline PSS in Korea. The

[1]Corresponding author.
E-mail : uschoi@jbnu.ac.kr

**Table 1.** Complete blood cell count and biochemistry on the day of first admission

| Parameters | Value | Reference range | Parameters | Value | Reference range |
|---|---|---|---|---|---|
| WBC ($10^3$/μL) | 7.46 | 5.5-19.5 | Albumin (g/dl) | 3.1 | 2.2-3.9 |
| RBC ($10^6$/μL) | 8.80 | 5.00-11.00 | Total protein (g/dl) | 7.1 | 5.2-8.2 |
| Hematocrit (%) | 30.1 | 24.0-45.0 | BUN (mg/dl) | 24 | 16-33 |
| Hemoglobin (g/dl) | 11.4 | 8. 0-15.0 | Creatinine (mg/dl) | 1.4 | 0.6-1.6 |
| RDW (%) | 17 | 12.0-18.0 | ALT (U/L) | 314 | 12-130 |
| MCV (fl) | 31.5 | 39.0-52.0 | ALKP (U/L) | 65 | 14-192 |
| MCHC (g/dl) | 36.4 | 30.0-37.0 | Cholesterol (mg/dl) | 104 | 62-191 |
| Platelet ($10^3$/μL) | 325 | 150-800 | Ca (mg/dl) | 9.7 | 7.8-11.3 |
| | | | P (mg/dl) | 3.4 | 3.1-7.5 |
| NH3 (μmol/L) | 98 | 0-95 | Na (mmol/L) | 162 | 150-165 |
| bile acid, fasting (μmol/L) | 16 | 0-25 | Cl (mmol/L) | 125 | 112-129 |
| postprandial (μmol/L) | 53 | 0-25 | K (mmol/L) | 3.9 | 3.5-5.8 |

RDW = RBC distribution width; MCV = mean corpuscular volume; MCHC = mean corpuscular hemoglobin concentration; BUN = blood urea nitrogen; ALT = alanine aminotransferase; ALKP = alkaline phosphatase; Ca = calcium; P = phosphate; Na = sodium; K = potassium; Cl = chloride

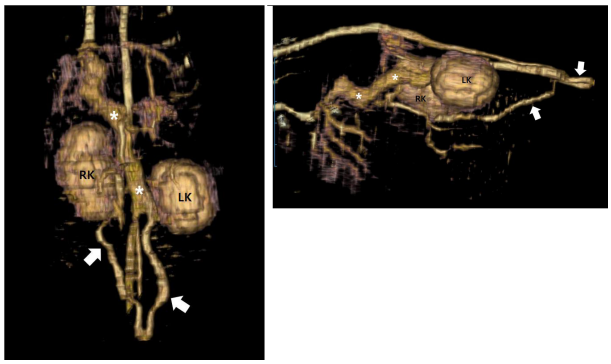


**Fig 1.** Volume rendering 3D reconstructed computed tomographic image of a cat with an extrahepatic portocaval shunt. The U-shaped shunt vessel (arrows) courses caudad at the level of urinary bladder and loops back craniad, connecting the portal vein and caudal vena cava (astericks) enlarged and tortuous. Left kidney (LK), right kidney (RK).

low incidence rate may be partly attributed to the fact that in many patients with PSS clinical signs may be intermittent and vague, waxing and waning, or appear normal (15).

Known clinical signs in the vast majority of affected cats include seizures, ataxia, central blindness, behavioral changes, aggression, lethargy, head pressing, tremors, and hypersalivation, among which hypersalivation has been the most frequently encountered clinical manifestation (1,2,15,17). Gastrointestinal disease common in dogs are less common in cats. The chief complaint of the current case was sudden onset of intermittent seizures.

Routine hematology and biochemistry are useful in the screening for cats with suspected PSS (15). Commonly reported clinicopathological abnormalities in affected cats include low urea, hypoalbuminemia, normal or increased ALT and ALP activities, increased fasting ammonia and bile acid concentration. In urinalysis like in dogs ammonium crystals can be seen although less common (1,5,7). In CBC microcytosis are more commonly reported, and approximately 27-54% of cats have microcytosis usually not accompanied by anemia (3,5,7). The sensitivity and specificity of ammonia for the detection of a CPSS has been reported as 83% and 86%, respectively and the sensitivity of preprandial and post prandial bile acids as 58-100% and 100% (13,15). The presence of microcytosis without signs or physical findings indicative of iron deficiency anemia or anemia of chronic inflammatory disease (2,4), low BUN, hypoabluminemia, and/or increased ALT and ALP activities can be used as a basis to determine to perform liver function tests, and/or imaging analysis. In the current case the presence of severe microcytosis in a young cat with no relevant clinical history suggestive of chronic disease or iron deficiency prompted further liver function tests and imaging analysis. It was difficult to demonstrate shunt vessels by ultraonography in the current case, although highly sensitive and specific for the diagnosis of PSS in cats (15). Microcytosis in conjunction with clinical signs can be a useful screening index for the diagnosis of CPSS in cats because it is cost effective and simple, and can be included in the general profile even in the normal appearing cats (12). However, for the confirmative diagnosis of CPSS liver function tests and imaging analysis should be pursued. Any feline breeds having congenital microcytosis have not been known unlike in dogs (15).

The shunt vessel in the current case was extrahepatic which is the most common form of CPSS in cats although the vessels were one large unusually long U shaped and another short vessels (15). Common communication include the left gastric vein to the caudal vena cava, portoazygous shunts, portocaval shunts, and shunts connecting the colonic vein or the gastrosplenic vein and the caudal vena cava of which portocaval shunt was revealed in the current case.

In cats with CPSS, stabilization is prerequisite prior to a definitive surgical treatment. Medical treatments include lactulose, antibiotics, dietary medication to reduce ammonia and other toxin levels, and anti convulsive drugs. Responses to these premedication may vary in cats with only 32% complete resolution of the clinical signs in one report (11). Diazepam, phenobarbitone, propofol or levetiracetam should be given to the cats with severe neurological signs or status epilepticus (15). Postattenuation neurological complications are

more prevalent in cats than in dogs (11). In this patient in an emergency state the seizures did not respond to diazepam, and lactulose, and the seizures became more severe. Poor response to this medication can be attributed to multiple complex form of shunts and severly decreased liver function.

Conclusively, CPSS should be included in the differential diagnosis in cats showing neurological signs. Microcytosis, low BUN, hypoalbuminemia, abnormal liver function tests and increased ALT/ALP activities are useful indices to rule in/out the presumptive diagnosis of CPSS.

## References


1. Bergerb B, Whiting PG, Breznock EM, Bruhl-Day R, Moore PF. Congenital feline portosystemic shunts. J Am Vet Med Assoc 1986; 188: 517-521.
2. Blaxter AC, Holt PE, Pearson GR, Gibbs C, Gruffydd-Jones TJ. Congenital portosystemic shunts in the cat: A report of nine cases. J Small Anim Pract 1988; 29: 631-645
3. Center SA, Magne ML. Historical, physical examination, and clinicopathological features of portosystemic vascular anomalies in the dog and cat. Semin Vet Med Surg (Small Anim) 1990; 5:83-93.
4. Dorothy PL, Edward AM, Sheila WA, David CT, Keith WP, Thomas LH. Microcytosis and iron status in dogs With surgically induced portosystemic shunts. J Vet Int Med 1994; 8: 212-216.
5. Havig M, Tobias KM. Outcome of ameroid constrictor occlusion of single congenital extrahepatic portosystemic shunts in cats: 12 cases (1993-2000). J Am Vet Med Assoc 2002; 220: 337-341.
6. Hunt GB. Effect of breed on anatomy of portosystemic shunts resulting from congenital diseases in dogs and cats: a review of 242 cases. Aus Vet J 2004; 82: 746-749.
7. Kyles AE, Hardie EM, Mehl M, Gregory CR. Evaluation of ameroid ring constrictors for the management of single extrahepatic portosystemic shunts in cats: 23 cases (1996-2001). J Am Vet Med Assoc 2002; 220: 1341-1347.
8. Levy JK, Bunch SE. Congenital portosystemic vascular shunts in cats. J Vet Int Med 1992; 6: 126.
9. Levy JK, Bunch SE, Komtebedde J. Feline portosystemic vascular shunts. In: Bonagura JD, ed. Kirk's current veterinary therapy XII small animal practice. Philadelphia: WB Saunders, 1995: 743-749.
10. Lipscomb VJ, Jones HJ, Brockman DJ. Complications and long-term outcomes of the ligation of congenital portosystemic shunts in 49 cats. Vet Rec 2007; 60: 465-470.
11. Lipscomb VJ, Lee KC, Lamb CR, Brockman DJ. Association of mesenteric portovenographic findings with outcome in cats receiving surgical treatment for single congenital portosystemic shunts. J Am Vet Med Assoc 2009; 234: 221-228.
12. Paepe D, Haers H, Vermote K, Saunders J, Risselada M, Daminet S. Portosystemic shunts in dog and cats: Laboratory diagnosis of congenital portosystemic shunts. Vlaams Diergeneeksundig Tijdschrift 2007; 76: 241-248.
13. Ruland K, Fischer A, Hartmann K. Sensitivity and specificity of fasting ammonia and serum bile acids in the diagnosis of portosystemic shunts in dogs and cats. Vet Clin Pathol 2010; 39: 57-64.
14. Simpson KW, Meyer DJ, Boswood A, White RN, Maskell IE. Iron status and erythrocyte volume in dogs with congenital portosystemic vascular anomalies. J Vet Int Med 1997; 11: 14-19.
15. Tivers M, Lipscomb V. Congenital portosystemic shunts in cats. Investigation, diagnosis and stabilization. J Feline Med Surg 2011; 13: 173-184.
16. Tobias KM, Rohrbach BW. Association of breed with the diagnosis of congenital portosystemic shunts in dogs; 2,400 cases (1980-2002). J Am Vet Med Assoc 2003; 223: 1636-1639.
17. Watson PJ, Bunch SE. Hepatobililary diseases in the Cat. In: Richard W. Nelson, C. Guillermo Couto., eds. Small Aniaml Internal Medicine. 4th ed.MOSBY ELSEVIER; 535-537.


# 한국 집고양이의 문맥전신션트 한 증례

**조은광 · 정예찬 · 최을수**[1]

전북대학교 수의과대학, 생체안전성 연구소

**요 약** : 3년령 한국 집고양이가 갑작스럽게 발병한 간헐적 경련 증상을 주 증상으로 내원하였다. 신체검사상 구리 빛 홍채가 관찰되었고, 양안에서 위협반사 소실이 관찰되었다. 혈액학 검사와 혈청 화학 검사에서 경증의 적혈구 증가증과 심한 소적혈구증, 증가된 ALT 활성도가 확인되었으며, 추가로 실시한 간기능 평가에서는 담즙산염 농도와 암모니아 농도가 증가하였다. 방사선 검사에서 간 내 다수의 결절이 관찰되었고, 간외성 션트 혈관을 발견하였으며, 추가로 실시한 컴퓨터 단층 촬영을 통해 복합 간외션트를 확인하였다. 고양이는 션트 혈관에 대한 외과적 처치술을 받기 위해 증상 완화를 위한 디아제팜과 락툴로즈를 투여하였으나 경련이 심화되었고, 심장 정지가 와서 폐사하였다.

**주요어** : 문맥전신션트, 간외성, 한국 집고양이