Congenital Portosystemic Shunts in Three Pigs and One Calf

T. S. G. A. M. van den Ingh, J. S. van der Linde-Sipman, A. Berrocal and J. H. Vos

Vet Pathol 1990 27: 56
DOI: 10.1177/030098589002700108

Vet. Pathol. 27:56–58 (1990)

BRIEF COMMUNICATIONS

Congenital Portosystemic Shunts in Three Pigs and One Calf

T. S. G. A. M. VAN DEN INGH, J. S. VAN DER LINDE-SIPMAN, A. BERROCAL, AND J. H. VOS

Key words: Calf; congenital portosystemic shunt; pig.

A congenital portosystemic shunt results from persistence of an anomalous embryological connection of the portal vein with a large vein of the systemic circulation.[3] In contrast to acquired portosystemic collaterals, which are multiple and often tortuous and result from portal hypertension, the congenital shunts are single and not associated with significant portal hypertension. Since 1974, many reports have documented the relatively high incidence of congenital portosystemic shunts in dogs and cats;[3,4,5,10,11] both intrahepatic and extrahepatic forms were recognized. Hepatoencephalopathy was one of the most obvious clinical sequelae of these shunts. In large domestic animals, only two cases have been reported thus far; these were an intrahepatic congenital portosystemic shunt (patent ductus venosus) in a calf[7] and an extrahepatic congenital portosystemic shunt (between splenic vein and caudal vena cava) in a foal.[8] In this report, we describe four cases of congenital portosystemic shunts: three in pigs and one in a calf.

Three unrelated pigs of the Dutch Landrace breed, two males and a female, 6, 12, and 16 weeks old, respectively, were submitted for post-mortem examination because of stunted growth. The animals were too small with respect to their age and weighed only 3.8, 10, and 12 kg, respectively. At necropsy the animals had a single, large extrahepatic portosystemic shunt covered by pancreatic tissue. All three animals had an identical vascular configuration. The portal vein communicated with the caudal vena cava over a distance of 0.5, 1, and 2 cm, respectively, at a site just caudal to the inlet of the splenic vein. The portal vein cranial to the shunt was markedly hypoplastic (Fig. 1). The livers were about ⅓ to ½ the normal size (98 g, 170 g, and 180 g, respectively) and had some superficial fibrosis. The kidneys were about two to three times the normal size (110 g, 140 g, and 280 g for both kidneys, respectively) but had a normal macroscopic appearance. Other, unrelated findings in the pigs included chronic focal peritonitis with abscessation around the urachus and acute fibrinous polyserositis and fibrinous pneumonia in the right apical lobe. Histologically, the livers in all three animals were hypercellular due to proliferation of Kupffer cells and sinusoidal leukocytosis. Hypoplastic portal vein tributaries and an increased number of arteriolar cross sections were present in the portal areas (Fig. 2). No significant lesions were seen in the kidneys. The central nervous system had extensive bilateral symmetric multifocal microcavitation, especially of the brain stem, the cerebellar nuclei, and at the border of the white and grey matter in the cerebral cortex. There was an increased number of protoplasmic astrocytes, usually with an irregular or indented outline and a clear nuclear membrane (Alzheimer type II reaction). The number of these cells varied, and they were most often seen in the cerebral cortex and the adjoining white matter.

A 12-week-old female calf of the Meuse-Rhine-Yssel breed was submitted for post-mortem examination with a history of dullness since birth, stunted growth, and recurrent ataxia. The animal was small with respect to age and breed (70 kg). At necropsy the animal had a single, large extrahepatic portosystemic shunt. The portal vein directly drained into the caudal vena cava, 2 cm caudal to the liver. No venous connection was found between the portal vascular system and the liver, the hepatic artery being the only vessel entering the liver hilus (Fig. 3). The liver was small (660 g); the kidneys were markedly enlarged (840 g), but otherwise grossly unremarkable. An exudative bronchopneumonia was present in the right and left apical lobes. Histologically, the portal triads had arteriolar proliferation and absence or hypoplasia of the portal vein tributaries. The hepatocytes were often atrophic or showed fatty infiltration. The kidneys were normal. The brain revealed severe bilateral symmetric multifocal microcavitation and some evidence of an Alzheimer type II reaction.

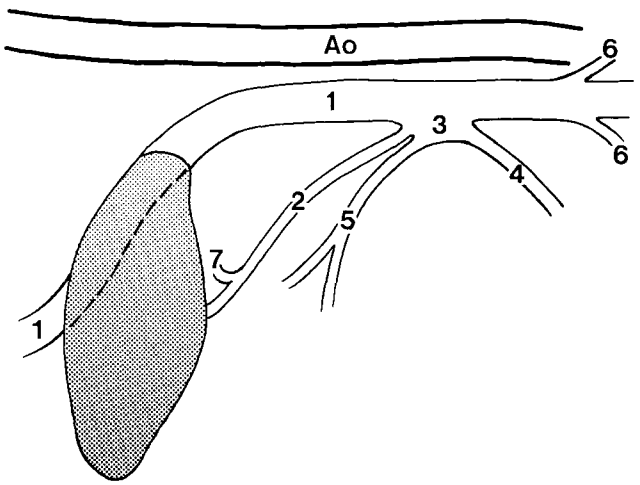

Fig. 1. Schematic drawing of the vascular configuration of the congenital portosystemic shunts in the three pigs. Aorta (Ao), caudal vena cava (1), portal vein (2), shunt (3), cranial mesenteric vein (4), splenic vein (5), renal vein (6), gastroduodenal vein (7).

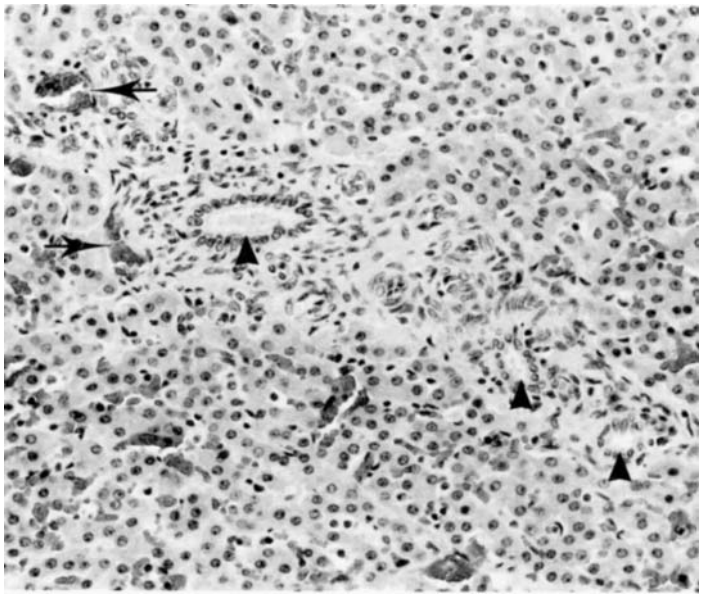

Fig. 2. Liver, pig. Portal area with hypoplastic portal vein (arrows), normal bile ducts (arrowheads) and many arteriolar cross sections. HE.

In the pigs and the calf the portosystemic shunts were the result of a persistent anomalous embryological anastomosis between the portal vein and the caudal vena cava as in the calf and foal described in the literature.[7,8] Two other reports of portal vein anomalies in horses[1,2] more likely describe acquired portosystemic collaterals due to portal hypertension as both cases showed multiple and tortuous vessels. The hypoplasia or even absence of the intra- and extrahepatic portal vein cranial to the shunt is most probably secondary to hemodynamic changes, as the portal blood avoids the higher flow resistance of the liver. Atrophy of the liver and degenerative changes of hepatocytes are the consequence of the shunting of portal blood past the liver. The many coiled branches of the hepatic artery observed on histologic examination of the liver are the morphologic expression of a compensatory increase in arterial blood flow.[6] Enlargement of the kidneys is a regular finding in dogs with congenital portosystemic shunts and is attributed to increased renal metabolic activity compensating for decreased liver function.[10] The microcavitation and the astrocytic changes in the brain, as seen in all four animals, are consistent with a diagnosis of hepatoencephalopathy. The astrocytic changes result from hypertrophy and subsequent degeneration of the cells. The hypertrophic changes reflect heightened metabolic activity perhaps for ammonia detoxification. This appears plausible since glutamine synthetase, the principal enzyme involved in ammonia detoxification, is exclusively found in astrocytes. Eventually, degenerative changes develop, possibly related to energy failure as two molecules of ammonia combine with α-ketoglutarate, a key intermediate in the citric acid cycle, to form glutamine. Accordingly, ammonia intoxication may deplete α-ketoglutarate, thereby reducing ATP production. Impaired astroglial function (water, electrolyte, pH, and neurotransmitter regulation) leads to an encephalopathic state.[9] The microcavitation, representing intramyelinic edema, may result from such impaired astroglial function.[9]

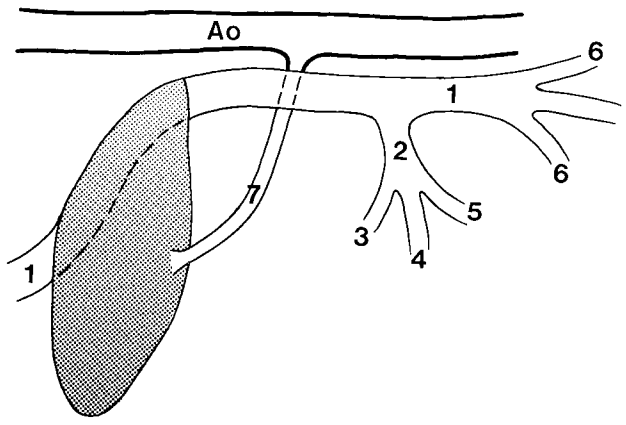

Fig. 3. Schematic drawing of the vascular configuration of the congenital portosystemic shunt in the calf. Aorta (Ao), caudal vena cava (1), portal vein (2), gastroduodenal vein (3), splenic vein (4), cranial mesenteric vein (5), renal vein (6), hepatic artery (7).

References

1 Beech J, Dubielzig R, Bester R: Portal vein anomaly and hepatic encephalopathy in a horse. J Am Vet Med Assoc **170:**164–166, 1977

2. Buonanno AM, Carlson GP, Kantrowitz B: Clinical and diagnostic features of portosystemic shunt in a foal. J Am Vet Med Assoc **192:**387–389, 1988

3 Ewing GO, Suter PF, Bailey CS: Hepatic insufficiency associated with congenital anomalies of the portal vein in dogs. J Am Anim Hosp Assoc **10:**463–476, 1974

4 Gandolfi RC: Hepatoencephalopathy associated with patent ductus venosus in a cat. J Am Vet Med Assoc **185:**301–302, 1984

5 Johnson CA, Armstrong PJ, Hauptman JG: Congenital

portosystemic shunts in dogs: 46 cases (1979–1986). J Am Vet Med Assoc **191**:1478–1483, 1987
6 Kaman J: Morphological reaction of the hepatic arteries of the dog at partial ligation of the portal vein. Zentralbl Veterinarmed **15**:718–728, 1968
7 Keane D, Blackwell T: Hepatic encephalopathy associated with patent ductus venosus in a calf. J Am Vet Med Assoc **182**:1393–1394, 1983
8 Lindsay WA, Ryder JK, Beck KA, McGuirk SM: Hepatic encephalopathy caused by a portacaval shunt in a foal. Vet Med (Praha) **83**:798–805, 1988
9 Norenberg MD, Gregorios JC: Central nervous system manifestations of systemic disease. *In:* Textbook of Neuropathology, ed. Davis RL and Robertson DM, pp. 403–467. Williams and Wilkins, Baltimore, MD, 1985
10 Rothuizen J, Van den Ingh TSGAM, Voorhout G, Van der Luer RJT, Wouda W: Congenital porto-systemic shunts in sixteen dogs and three cats. J Small Anim Pract **23**:67–81, 1982
11 Vulgamott JC, Turmwald GH, King GK, Ferring DS, Hansen JF, Boothe HW: Congenital portocaval anomalies in the cat: two case reports. J Am Anim Hosp Assoc **16**:915–919, 1980

Request reprints from Dr. T. S. G. A. M. van den Ingh, Department of Veterinary Pathology, Faculty of Veterinary Medicine, University of Utrecht, Yalelaan 1, 3508 TD Utrecht (The Netherlands).

Vet. Pathol. **27**:58–60 (1990)

Spleno-mesenteric-renal Venous Shunt in Two Dogs

R. W. VALENTINE AND J. L. CARPENTER

Key words: Ascites; dog; hepatic encephalopathy; spleno-mesentric-renal venous shunt.

This communication describes a congenital venous anomaly in two dogs. Our report is similar to a 30-year-old report that describes the condition, also in a dog, as an acquired shunt.[4,5] Both dogs in our study had ante-mortem diagnoses of ascites and an unclassified vascular anomaly involving the liver that had caused hepatic encephalopathy. One dog also had bilateral polycystic kidneys.

The first case was a 6-month-old male Golden Retriever with a history of lethargy, decreased appetite, and abdominal distension. The enlarged abdomen was caused by a transudate that was temporarily reduced by diuretic treatment. A tentative diagnosis of renal failure was based on urine specific gravity of 1.011 and sonographic findings that suggested bilateral, multiple, hyperechoic renal cortical foci. The dog was discharged but was returned because of several 1-minute seizures consisting of uncontrolled chewing, ataxia, hyperextension of extremities, and marked post-ictal depression. The bromsulfopthalein (BSP) retention test was 18.5% (normal, <5%) after 30 minutes (Table 1). Treatment with lactulose and enemas for hyperammonemia was begun before we received the report of a blood ammonia concentration of 3,674 μg/liter (normal, 0–700). Due to the guarded prognosis for complete recovery, the owner requested that the dog be euthanatized.

Necropsy findings included a cloudy serosanguineous pleural effusion (100 ml), clear, yellow to pink pericardial effusion (10 ml), serosanguineous ascitic fluid (10 ml) with a specific gravity of 1.009, and a small hiatal diaphragmatic hernia. Both kidneys were polycystic and a bilateral, chronic, severe, interstitial nephritis was confirmed histolopathologically. A venous plexus caudal to the left kidney was formed by anastomoses of splenic-omental veins, the left iliac, the left testicular (gonadal), and several mesocolic veins. A single tributary, analogous to the left gonadal vein, connected the plexus to the left renal vein, which was enlarged (Figs. 1, 2a, b). The shunt apparently provided venous drainage of high ammonia concentration, generated by the colic bacteria, to the caudal vena cava thus bypassing the portal circulation. The relationship of the embryonic vascular anomaly to that of the polycystic kidneys remains unknown.

Table 1. Clinical pathologic findings in two dogs with a spleno-mesenteric-renal venous shunt.

Test	Case I	Case II	Normal Concentrations
Blood urea nitrogen (mg/dl)	19.3	7.0	7–25
Creatinine (mg/dl)	2.6	0.7	0.6–1.6
Ammonia (μg/liter)	3,674.0	ND*	0–700
Bromosulfophthalein	18.5	ND	<5% at 30 minutes
Bile acids			
Pre-prandial (μM/liter)	ND	178.7	<5
Post-prandial (μM/liter)	ND	>400	<10

* ND = not done.