Objective—To determine postoperative (≤ 6 days), short-term (≤ 90 days), and long-term (≥ 6 months) outcomes of cats undergoing ameroid constrictor occlusion of single congenital extrahepatic portosystemic shunts (PSS) and identify factors associated with outcome.

Design—Retrospective study.

Animals—12 cats.

Procedure—Cats with single congenital PSS that underwent surgical placement of ameroid constrictors were identified. Follow-up information was obtained through telephone interviews and facsimile correspondence with referring veterinarians and owners.

Results—All cats survived the surgery and were discharged from the hospital. One cat had seizures during the postoperative period. Five cats were clinically normal during follow-up evaluations within 90 days after the surgery. Long-term follow-up information was available for 9 cats. Three were clinically normal, 4 had been euthanatized because of progressive neurologic disease, and 2 had neurologic abnormalities that could not be controlled with medication. Four of 7 cats with continued or recurrent neurologic abnormalities 1 or more months after surgery had normal scintigraphic or hepatic function test results 2 to 6 months after surgery.

Conclusions and Clinical Relevance—Results suggest that the long-term outcome of ameroid constrictor occlusion of PSS in cats is poor. Owners of older cats and cats with preexisting neurologic signs should be made aware of the potential for a poor outcome when considering surgical correction of this disease. (J Am Vet Med Assoc 2002;220:337–341)

Portosystemic shunts (PSS) are abnormal vascular communications that divert venous blood from the portal to the systemic circulation, bypassing normal hepatic filtration and metabolism. Portosystemic shunts in humans, dogs, cats, horses, cows, and other species have been described; such shunts may be congenital or acquired, intrahepatic or extrahepatic, and single or multiple. Congenital PSS are uncommon in cats, being identified in 2.5 of every 10,000 cats examined at veterinary teaching hospitals in North America. Cats with congenital PSS most often have single extrahepatic shunts, although intrahepatic PSS have also been reported. No breed predilection has been documented in cats; however, some reports suggest that male cats are more likely to be affected.

Recommended treatments for PSS include medical and surgical management. The primary goal of medical management is to reduce the factors that contribute to hepatic encephalopathy, such as dietary protein and colonic bacteria. However, long-term medical treatment, without surgical occlusion of the PSS, has been associated with a generally unsatisfactory outcome in cats. Surgical treatment involves complete or partial occlusion of the shunt with nonabsorbable suture or, more recently, gradual complete occlusion with an ameroid constrictor. Before use of ameroid constrictors became popular, partial occlusion was often necessary in animals in which complete occlusion resulted in portal hypertension; however, clinical signs recurred in 75 to 100% of cats undergoing partial occlusion. The prognosis is reportedly good for cats with PSS that undergo complete occlusion of the shunt; however, the number of cats for which long-term follow-up information has been obtained is limited. In addition, the incidence of postoperative complications in and results of long-term follow-up for cats with PSS in which an ameroid constrictor is used for complete occlusion of the shunt are not well documented.

Therefore, the purposes of the study reported here were to determine postoperative (≤ 6 days), short-term (≤ 90 days), and long-term (≥ 6 months) outcomes of cats undergoing ameroid constrictor occlusion of a single congenital extrahepatic PSS and to determine whether age at the time of shunt occlusion, duration or severity of clinical signs, or results of diagnostic testing were associated with postoperative and long-term outcomes.

Criteria for Selection of Cases

Medical records of all cats admitted to the University of Tennessee Veterinary Teaching Hospital between January 1993 and November 2000 in which a diagnosis of portosystemic vascular shunting had been made were reviewed. Cats were included in the study only if a single extrahepatic PSS had been identified during exploratory laparotomy and an ameroid constrictor had been used to occlude the shunt.

Procedures

The following information was obtained from the medical records: signalment; history; physical examination findings; duration of clinical signs; results of clinicopathologic testing; radiographic and ultrasonographic findings; results of nuclear imaging; type of shunt and operation performed; and postoperative,
short-term, and long-term outcomes. Follow-up information was obtained by telephone interview and facsimile correspondence with referring veterinarians and owners. Postoperative outcome was defined as outcome during the hospitalization period immediately after surgery (days 1 through 6). Short-term outcome was defined as outcome 14 to 90 days after surgery. Long-term outcome was defined as outcome ≥ 6 months after surgery. Outcome was categorized as good if clinical signs did not recur or if the cat did not have any clinical signs with medical management. Outcome was categorized as poor if the cat had persistent or recurrent clinical signs that could not be resolved with medical management or resulted in euthanasia of the cat. The Fisher exact probability test was used to determine whether outcome was associated with age, sex, duration of clinical signs prior to surgery, neurologic status before surgery, results of diagnostic tests, or ameboid constrictor size; a value of P ≤ 0.05 was considered significant.

**Results**

Portosystemic vascular shunting was diagnosed in 14 cats during the study period. One of these cats had an intrahepatic PSS that was not corrected, and another had multiple acquired shunts; both cats were excluded from the study. The remaining 12 cats were included in the study.

Nine of the 12 cats were domestic shorthairs, 1 was of mixed breeding, and 2 were Persians. Age at the time of surgery ranged from 4 months to 10 years (median, 1.25 years). Nine cats were ≤ 2 years old, and 3 were ≥ 5 years old at the time of surgery. Seven cats were male (5 castrated and 2 sexually intact), and 5 were female (2 spayed and 3 sexually intact).

Duration of clinical signs ranged from 1 week to 1.5 years. Waxing and waning of clinical signs were reported in 7 cats. Ten cats were examined because of neurologic abnormalities, including seizures (8 cats), blindness (5), head shaking, disorientation, stupor, dementia, ataxia, conscious proprioception deficits, pelvic limb weakness, dull mentation, and anisocoria. Other findings included ptalism (8 cats); behavior changes (5); lethargy and weakness (5); cardiac murmur (4); small stature (3); a history of anesthetic intolerance (3); copper-colored irises (2); tremors (2); and vomiting, diarrhea, anorexia, tail twitching, vocalizing, and a history of urolithiasis (1 each). In 4 cats, clinical signs were worse after eating. In 2 cats, clinical signs were limited to lethargy (both cats) and inappetence, vomiting, diarrhea, or ptalism (1 cat each). In the cat with a history of urolithiasis, the type of calculus was unknown.

Results of a CBC and serum biochemical analyses were available for 11 cats; results of a urinalysis were available for 9. Hematologic abnormalities included anemia (1 cat; PCV, 16.6 to 24%), microcytosis (3; MCV, 33.9 to 38 fl; reference range, 39 to 55 fl), leukocytosis (2), lymphocytosis (1), and lymphopenia (3). The most consistent serum biochemical abnormalities were high alkaline aminotransferase activity (8 cats; range, 76 to 278 U/L; reference range, 20 to 75 U/L) and low blood urea nitrogen concentration (7; range, 10 to 13 mg/dl; reference range, 15 to 30 mg/dl). Other abnormalities included hypoglycemia (3 cats), hypoproteinemina (2), high alkaline phosphatase activity (2), high aspartate aminotransferase activity (1), high total bilirubin concentration (1), and low creatinine concentration (1). None of the cats had hypoalbuminemia or hypocholesterolemia. Urine was categorized as concentrated (urine specific gravity > 1.039) in 1 cat, dilute (urine specific gravity between 1.015 and 1.039) in 7 cats, and isosthenuric (urine specific gravity between 1.008 and 1.014) in 1 cat. Crystalluria was found in 3 cats; crystals were identified as ammonium biurate crystals in 2 cats and as amorphous crystals in 1. Urolithiasis was not noticed in any cat during physical examination or during palpation of the bladder at the time of surgery. In addition, uroliths were not seen in the 8 cats that underwent abdominal radiography or the 6 cats that underwent abdominal ultrasonography.

Results of hepatic function tests were available for all cats. Tests performed included measurement of baseline bile acids concentration (6 cats), measurement of postprandial bile acids concentration (5), measurement of baseline plasma ammonia concentration (9), and an ammonia tolerance test (1). Baseline plasma ammonia concentration was high in 9 of 10 cats in which it was measured (range, 1.14 to 8.34 µmol/ml; mean, 4.03 µmol/ml; reference range, < 1.0 µmol/ml). In the cat with a normal baseline plasma ammonia concentration, plasma ammonia concentration was greatly increased (5.01 µmol/ml) 30 minutes after oral administration of ammonium chloride. Baseline bile acids concentration was high in all 6 cats tested (range, 8.6 to 156.4 µmol/L; mean, 84.9 µmol/L; reference range, 0 to 5 µmol/L). Postprandial bile acids concentration was also high in all 5 cats tested (range, 141 to 314 µmol/L; mean, 230 µmol/L; reference range, 0 to 10 µmol/L).

Nuclear scintigraphy was performed before surgery in all cats. Sodium pertechnetate Tc 99m was administered per rectum, and the shunt fraction was calculated, as described.22 The shunt fraction was high in all cats (mean shunt fraction, 81.9%; range, 67 to 93%; reference range, < 15%).

Ten cats were treated medically prior to surgery. Preoperative treatment included 1 or more of the following: oral administration of lactulose, antibiotic therapy (neomycin, metronidazole, amoxicillin, amoxicillin-clavulanic acid, or ampicillin), and feeding a protein-restricted diet44 for 1 to 6 days before surgery. One cat had been fed a protein-restricted diet for 2 years because of a previous diagnosis of urolithiasis. Six cats were treated with phenobarbital or diazepam PO or with diazepam or pentobarbital IV for 2 to 14 days before surgery to control frequent seizures. One cat did not receive any medical treatment prior to surgery.

The most common PSS identified at surgery was a left gastric vein-to-caudal vena cava shunt (7 cats). Portocaval, gastroeplicenic-azygos, gastroduodenal-caval, mesenteric-caval, and portoazygos shunts were identified in the remaining cats. Size of the ameboid constrictor used to occlude the shunt ranged from 2.0 to 5.0 mm. During surgery, portal pressures were measured with a water manometer in 5 cats before place-
ment of the ameroid constrictor. Baseline portal pressure prior to application of the ameroid constrictor ranged from 10 to 14 cm H₂O (mean, 10.1 cm H₂O). After placement of the ameroid constrictor, portal pressures in 2 cats were 14 and 6 cm H₂O; however, it was not reported in the medical records of these 2 cats whether portal pressure catheters were flushed or positioned between pressure measurements.

No complications were identified during anesthetic recovery of the cats. Cats were hospitalized for 1 to 6 days (median, 2 days) after surgery. After surgery, fluids were administered IV for at least 16 hours, and cats were fed a protein-restricted diet. One cat had seizures 12 and 48 hours after recovery; this cat had been treated with phenobarbital for 1 week before surgery because of preoperative seizures. After surgery, seizures were treated by IV bolus administration of diazepam. None of the other cats had seizures during the hospitalization period after surgery; however, hypersalivation and neurologic signs, including ataxia, weakness, and trembling, persisted in 3 of the 10 cats that had neurologic signs prior to surgery.

At the time of discharge, owners were instructed to continue to feed a protein-restricted diet and to administer 1 or more of the following medications until hepatic function or clinical status returned to normal: lactulose, neomycin, cephalaxin, amoxicillin, or metronidazole. Four of the 6 cats that received anticonvulsant medications during the 2 weeks before surgery continued to receive anticonvulsant medications after surgery (phenobarbital, 3 cats; diazepam, 1). The remaining 8 cats did not receive anticonvulsant medications during the postoperative period.

All cats were returned to the veterinary teaching hospital or to the referring veterinarian for reevaluation within 90 days after surgery. Five of the 12 cats were clinically normal. Scintigraphy was performed in 3 of these cats, and results were normal in 2. The remaining cat had an abnormal shunt fraction (72% 2 3 of these cats, and results were normal in 2. The remaining 3 cats for which long-term follow-up information was available were clinically normal 10 months to 5 years after surgery. Two of these cats had no scintigraphic evidence of shunting. The third had a high shunt fraction on follow-up scintigrams but was free of clinical signs 10 months after surgery; as long as it was maintained on a protein-restricted diet and lactulose.

Long-term outcome was considered good in 3 cats and poor in 6. Outcome was not associated with age, sex, duration of clinical signs prior to surgery, results of clinicopathologic tests or scintigraphy, or ameroid constrictor size. Two cats without neurologic signs before surgery had good outcomes, whereas only 1 of 7 cats with neurologic signs before surgery had a good outcome. However, neurologic status before surgery was not significantly (P = 0.08) associated with long-term outcome.

Discussion

The age range of cats included in the present study was much broader than the age range of cats with congenital PSS described in previous reports.2-5,13 This suggests that congenital PSS should be investigated as a possible differential diagnosis in older cats in which history, clinical signs, and clinicopathologic test results are suggestive of the disease. Some studies20,25-25 report that older dogs with PSS may have a higher risk of developing postoperative complications than younger dogs and that age may be associated with outcome, whereas other studies26,27 do not identify age as a prognostic factor. In the present study, all 3 cats that were ≥ 5 years old at the time of surgery had poor long-term outcomes; however, number of cats was too small for meaningful statistical analysis of whether age was associated with outcome.

As with previous studies,1-4,5,7,11,12,18 all cats in the present study were most often examined because of neurologic abnormalities. In dogs with congenital PSS, a lack of signs of hepatic encephalopathy is a positive prognostic indicator that the shunt can be completely occluded, and complete shunt occlusion may, in turn, be associated with a better long-term prognosis after surgical correction.20,28 In the present study, both cats

Lactulose was continued to feed a protein-restricted diet and to administer anticonvulsant medications during the postoperative period.
without neurologic signs before surgery had good long-term outcomes, whereas only 1 of 7 cats with neurologic signs before surgery had a good long-term outcome. However, the small number of cats precluded meaningful statistical analysis of the association of neurologic status and long-term outcome.

Results of CBC, serum biochemical analyses, urinalyses, and hepatic function tests were generally similar to those previously reported,1,4,5,11,12,13,19 with the exception that hypoalbuminemia and hypocholesterolemia were not detected in any cats in the present study. Results of preoperative clinicopathologic testing and scintigraphy were not associated with long-term outcome in this study.

Seizures reportedly develop during the immediate postoperative period in 10 to 18% of dogs20,21,23,27,29 and 5% of cats2 undergoing surgery for correction of a PSS. Results of the present study were similar, in that 1 of 12 cats had seizures during the postoperative period. Such seizures may be secondary to hepatic encephalopathy, hypoglycemia, or other metabolic abnormalities. However, seizures and neurologic deficits may also develop in dogs without metabolic abnormalities.27 In these dogs, clinical signs, including ataxia, generalized motor seizures, and status epilepticus, usually develop within 3 to 7 days after surgery.20,21,23,27,29 Older dogs and dogs with portoazygos shunts are reportedly predisposed to these nonmetabolic seizures and neurologic deficits.20,23,24,27,29,30 However, seizures and status epilepticus do not necessarily develop in dogs with hepatic encephalopathy after administration of benzodiazepine receptor antagonists.19,24,30 In addition, concentrations of endogenous benzodiazepines are high in portal blood from dogs with PSS.30 However, if an acute reduction in the endogenous benzodiazepine concentration is the primary cause of nonmetabolic seizures after PSS occlusion, then animals in which an ameroid constrictor is used to gradually occlude the shunt would theoretically have a lower risk of postligation seizures. However, shifting of the constrictor ring or use of a constrictor that is too small may acutely compress the shunt.37,31 The 1 cat that had seizures in the postoperative period in the present study was being treated with lactulose, a protein-restricted diet, and 5% dextrose solution, IV, when the seizures occurred; however, the cat responded well to IV infusion of diazepam. In dogs, diazepam may provide temporary cessation of seizures, but most animals require heavy sedation for 12 to 24 hours with propofol or pentobarbital to halt seizures and prevent their recurrence.21,29 Some authors recommend prophylactic use of anticonvulsant medications before and after surgery to prevent seizures.6,23,32 In the cat in the present study, seizures occurred despite the use of anticonvulsant medications prior to surgery.

Chronic neurologic abnormalities in animals that have undergone surgical occlusion of a PSS may be attributable to continued shunting through the original shunt or another congenital shunt not identified at the time of surgery, through multiple shunts acquired secondary to portal hypertension, or through dysplastic vessels in the hepatic parenchyma (hepatic microvascular dysplasia).17,31,35,36,37 The diagnosis is made on the basis of results of portography, scintigraphy, and hepatic biopsy. In animals with congenital or acquired PSS, shunting would be evident on portograms or scintigrams, whereas in animals with hepatic microvascular dysplasia, results of portography and scintigraphy would be normal but hepatic biopsy specimens would be abnormal. Four cats in the present study were assumed to have continued hepatic encephalopathy on the basis of results of scintigraphy or liver function testing; liver biopsy specimens were not available for any of these cats. Three of these cats did not respond or responded only poorly when treated with a protein-restricted diet and lactulose; therefore, neurologic disorders other than hepatic encephalopathy may also have been present.

Four cats in the present study had persistent or recurrent seizures despite normal shunt fractions or hepatic function test results. Persistence or recurrence of neurologic signs without evidence of postoperative shunting or hepatic dysfunction may be secondary to metabolic encephalopathy (eg, hypoglycemia, altered serum electrolyte concentrations, and acid-base abnormalities), systemic hypertension, or primary alterations in CNS structure or function.21 Blood pressure was not measured in any cat in this study; however, serum electrolyte and glucose concentrations, when measured, were unremarkable. A necropsy was not performed on any cat; therefore, the brains could not be examined for structural changes. Reported histologic abnormalities in patients with hepatic encephalopathy include polymicrocavitation of the white matter and development of Alzheimer type-II cells.35,36 It is unknown whether these changes resolve in animals after ligation of PSS. Functional changes may occur with hepatic encephalopathy because of altered cerebral metabolism. Such changes are usually considered reversible,24,30,31,37; however, some authors propose that chronic abnormal metabolism associated with hepatic encephalopathy alters biochemical features of the brain to a degree that it can no longer function once the shunt has been occluded.20,25

Structural or functional neurologic damage may also occur during status epilepticus or cluster seizures,30,33 as the increased rate of oxidative metabolism during seizures may lead to cerebral acidosis, cerebral edema, and ultimately neuronal death. In some cats in the present study, chronic seizures may have contributed to the progressive deterioration of their neurologic condition.

In the present study, the long-term outcome after ameroid constrictor occlusion of extrahepatic PSS was considered good in only 3 of 9 cats because of recurrent or persistent neurologic abnormalities in the remaining 6 cats. In contrast, good to excellent outcomes after acute partial or complete shunt occlusion have been reported for 59% of cats in previous studies.1,4,6,12,14-16,17 However, many of these studies included limited numbers of cats, and follow-up times were gen-
eraly short. It is possible that outcomes would have been poorer had follow-up times been longer. Approximately 90% of dogs described in previous studies\(^{1,2}\) and approximately 85% of Yorkshire Terriers treated at the University of Tennessee Veterinary Teaching Hospital are clinically improved or normal after amiodor constrictor occlusion of PSS.\(^{1,2}\) It is unknown why so many cats in the present study had progressive neurologic abnormalities after amiodor constrictor placement. Medical treatment is generally considered only palliative in cats with extrahepatic PSS, and results of medical management alone for treatment of cats with extrahepatic PSS have been reported infrequently. In 2 studies,\(^{3,4}\) however, only 1 of 5 cats receiving medical treatment alone survived for > 6 months. Comparison of these results with results of the present study would suggest that the long-term prognosis may be slightly better in those cats treated surgically versus those treated medically.

In conclusion, results of the present study suggest that gradual occlusion of extrahepatic PSS with an amiodor constrictor does not prevent recurrence or progression of neurologic signs in cats. Owners of older cats and cats with preexisting neurologic signs should be made aware of the potential for a poor outcome when considering surgical correction of this disease.

References