Complications and outcome of cats with congenital extrahepatic portosystemic shunts treated with thin film: Thirty-four cases (2008-2017)

Paula Valiente1 | Mary Trehy2 | Rob White3 | Pieter Nelissen4 | Jackie Demetriou1 | Giacomo Stanzani5 | Benito de la Puerta2

1Dick White Referrals, Cambridgeshire, United Kingdom
2North Downs Specialist Referrals, Bletchingley, United Kingdom
3School of Veterinary Medicine and Science, University of Nottingham, Leicestershire, United Kingdom
4Tierklinik Haar, Haar, Germany
5Division of Medicine, Bloomsbury Institute for Intensive Care Medicine, University College London, London, United Kingdom

Correspondence
Paula Valiente, Dick White Referrals, Station Farm, London Road, Six Mile Bottom, Cambridgeshire, CB8 0UH, United Kingdom.
Email: pvd@dwr.co.uk

Abstract

Background: Congenital extrahepatic portosystemic shunts (CEHPSS) are rare in cats. Outcome after attenuation of CEHPSS with thin film has been described in a small number of cases.

Objectives: To describe the clinical presentation, postoperative complications, and outcome of cats treated with thin film to attenuate CEHPSS.

Animals: Thirty-four cats with CEHPSS were identified from the database of 3 institutions over 9 years.

Methods: Retrospective study. Medical records were reviewed to identify cats with a diagnosis of a CEHPSS that underwent surgical attenuation. Congenital extrahepatic portosystemic shunts were suspected from clinical signs, clinicopathologic findings, and diagnostic imaging, and confirmed at exploratory laparotomy. Cats treated with thin film band attenuation were included. Postoperative complications and follow-up were recorded.

Results: Complications were recorded in 11 of 34 cats. Deaths related to CEHPSS occurred in 6 of 34; 4 cats did not survive to discharge. Persistent seizures were the cause of death in 4 cats. Seizures were recorded in 8 of 34 cats after surgery; all these cats received preoperative antiepileptic drugs. Serum bile acid concentrations normalized in 25 of 28 of the cats for which data was available. Three cats had persistently increased serum bile acid concentrations and underwent a second exploratory laparotomy. One had a patent shunt, the other 2 had multiple acquired portosystemic shunts. Median follow-up was 8 months (0.5-84 months).

Conclusions and Clinical Importance: Congenital extrahepatic portosystemic shunts attenuation using thin film in cats carries a good short- and mid-term prognosis if they survive the postoperative period. Seizures were the most common cause of death.

KEYWORDS
bile acids, feline, seizures, shunt closure, thin film

Abbreviations: CEHPSS, congenital extrahepatic portosystemic shunts; CPSS, congenital portosystemic shunt.
INTRODUCTION

A congenital portosystemic shunt (CPSS) is an abnormal vascular communication between the portal and systemic venous circulatory system diverting portal blood away from the liver.1-9 A CPSS can be an extrahepatic or intrahepatic vessel.10 Congenital extrahepatic portosystemic shunts (CEHPSS) are the most common shunts in cats.7,11,12

Medical management of CPSS is aimed at treating clinical signs of hepatic encephalopathy without shunt correction.10 Surgical treatment allows for shunt closure11 and is the treatment of choice for most cats with CPSS.14

Although there are no prospective studies evaluating the medical management of cats with portosystemic vascular anomalies,1 long-term medical treatment of cats with CEHPSS is associated with a poor outcome.15 In a study with 25 cats, there was no clinical response to medical treatment in 3 (12%) cats, partial clinical response in 14 (56%) cats, and complete resolution of clinical signs occurred in 8 (32%) cats.11 In dogs, surgical treatment is associated with better outcomes compared to medical treatment.15 A similar study comparing the 2 has not been performed in cats.

Animals undergoing complete surgical shunt occlusion present a better prognosis compared to those with partial attenuation.14,17,18 However, dogs undergoing acute complete shunt ligation have a higher risk of developing portal hypertension.19,20 Complete, acute attenuation of a CPSS can be well tolerated in some cats.7,11,14

Two methods of slow occlusion using extravascular techniques have been reported in cats: ameroid constrictor and cellophane band.3,6,12,14,21,22 The aim was to produce complete shunt attenuation gradually without causing portal hypertension.14 Cellophane or similar materials produce a foreign body reaction responsible for progressive vascular occlusion.5,9,14,23

Postoperative complications in cats undergoing surgical attenuation are common, occurring in up to 75% of animals.1,6,7,21 The most common complications are signs of neurological disease, including generalized seizures in 8% to 22% of cats and central blindness in up to 44% of the cats.1,7,21

Cats with CEHPSS undergoing ameroid constrictor occlusion present a survival rate of 33%-75%, with an excellent long-term outcome in 22.2%21 (clinically normal, normal shunt fraction on scintigraphy) to 75% cats6 (clinically normal, receiving no medical treatment). In cats undergoing ligation, survival rate ranges from 66% to 75% with excellent outcome (clinically normal, receiving no medical treatment) reported in 56% of the cases.7 In the present study, 21 of 49 cats had complete shunt attenuation at the first surgery and 28 had partial attenuation.

Regarding cellophane banding attenuation, survival rates are reported as 66%-100%3,24 with 1 study reporting excellent outcome (clinically normal, receiving no medical treatment) in 57.1% of the cases.3

The objective of this study was to describe the clinical presentation, postoperative complications, and outcome of cats treated surgically with thin film to attenuate a CEHPSS. Our hypothesis was that thin film attenuation is an appropriate management strategy for cats with CEHPSS and preoperative variables are not associated with postoperative outcomes.
3.1 Presurgical findings

Clinical findings included abnormal behavior (28/34—82%), ptalism (25/34—73%), stunted growth (16/34—47%), ataxia (14/34—41%), copper-colored irides (13/34—38%), depression (12/34—35%), inappetence (10/34—29%), lethargy (10/34—29%), seizures (6/34—17%), cystoliths (5/34—14%), twitching (4/34—11%), vomiting (3/34—8%), polyuria and polydipsia (3/34—8%), hematuria (2/34—5%), and muscle hypotonicity (1/34—2%). Median duration of clinical signs before surgery was 4 months (0.4—48). No significant association between occurrence of postoperative seizures or survival to discharge and clinical signs of gastrointestinal disease (Seizures P > .99, Survival P = .56), clinical signs of urinary disease (Seizures P > .99, Survival P > .99), copper-colored irides (Seizures P = .44, Survival P = .63), or stunted growth (Seizures P = 0.70, Survival P > .99) was found. All cats presented at least 1 type of clinical signs of neurological disease, preventing meaningful analysis of this category. Due to their clinical relevance, seizures were analyzed independently. The presence of seizures before surgery was not significantly associated with an occurrence of postoperative seizures or survival to discharge (Seizures P = .13, Survival P > .99). Only 3 of the 6 cats with seizures before surgery developed seizures after surgery.

All cats received medical treatment before and after surgery. Median duration of medical treatment was 28 days (7—42 days) before surgery and 28 days (14—2046 days) after surgery. All cats were treated with an antibiotic. Ampicillin (Ampicillin, AAH Pharmaceuticals Ltd) was administered in 4 of 34 (11%) cats (dose unavailable from the medical records). Twenty-eight of 34 (82%) cats received potentiated amoxicillin (Synulox, Pfizer) at a median dose of 13 mg/kg (5—18 mg/kg) twice a day (dose information available for 18 cases). Two of 34 cats (5%) received metronidazole (dose unavailable). Thirty-three of 34 (97%) cats were treated with lactulose (Lactulose, Sandoz) at a median dose of 1 mL (0.2—3 mL) 3 times a day. Thirty-three of 34 (97%) cats received a low protein diet. Fifteen of 34 (44%) cats received an antiepileptic, which consisted of phenobarbitone (Phenobarbital elixir, Thornton and Ross) in 3 of 34 (8%) cats, at a median dose of 1.22 mg/kg (0.67—2 mg/kg), or levetiracetam (Keppra, GlaxoSmithKline) in 12 of 34 (35%) of the cases, at a median dose of 19 mg/kg (10—20 mg/kg) 3 times a day.

Of the cats pretreated with an antiepileptic (n = 15), 8 developed seizures after surgery, 4 of which subsequently died. Ten of 17 (58%) cats in the first institution were pretreated with antiepileptics, 5 of
and insertion of the shunts were not described in the surgical report.

Spleno-caval (5/34), left gastro-caval (4/34—11%), porto-caval (3/34—8%), and 1 each of pancreaticoduodenal-caval, left colo-caval, and porto-azygos. There were 7 cases (7/34—20%) in which the origin and insertion of the shunts were not described in the surgical report.

Cystotomy was not performed in any case. Gonadectomy was performed in 2 cases.

### 3.4 Postsurgical findings

Median duration of hospitalization was 3 days (range, 2-6) for the cats that survived to discharge (30/34—88%). In these cats, the median duration of follow-up after discharge was 8 months (0.5-84 months). Updated information could not be obtained at the time of manuscript preparation in 1 cat. Complications occurred in 11 of 34 (32%) cats and consisted of seizures in 8 of 34 (23%) cats, apnea and coma on recovery in 1 of 34 (2%) cats, ascites in 1 of 34 (2%) cats, reduced vision/blindness in 2 of 34 (5%) cats, and pancreatitis in 1 of 34 (2%) cats. Some cats had more than 1 complication. Of the cats that developed seizures postoperatively, 5 of 8 (62%) cats did not have seizures before surgery. All the cats that developed seizures after surgery had received antiepileptic drugs before surgery, consisting of levetiracetam in 6 cases and phenobarbital in 2 cases. In the cats that developed seizures during hospitalization (4 cases), these occurred within 5 days of surgery.

Six cats (6/34—17%) were euthanized after surgery due to CHPSS-related causes. Four of these deaths occurred during hospitalization (short-term). Cats did not survive to discharge due to refractory seizures (3 cases) or apnea and coma on recovery (1 case). The cat that died because of apnea and coma on recovery had a normal bile acid stimulation test before surgery; however, plasma ammonia concentration in this cat was markedly increased. The 2 cats that died after discharge developed multiple acquired portosystemic shunts, which were diagnosed during a second surgery. Both had a persistently abnormal bile acid stimulation test. One of them was euthanized due to worsening seizures 6 months after a second surgery, which was performed due to persistently abnormal bile acid stimulation test 3 months after the first surgery. The other cat was euthanized 6 years after surgery due to weight loss and anorexia. This cat had persistently abnormal bile acid simulation test, but it is unknown if the clinical signs of this cat were CHPSS-related, given the significant time gap between the original surgery and the euthanasia.

In the mid-term, 3 of 28 (10%) of the surviving cats presented CHPSS-related clinical signs: 2 cats developed postoperative seizures not present before surgery, 3 months after the first surgery and 6 months after a second surgery, respectively (the latter cat was euthanized). Postoperative persistence of preoperative seizures was present in a third case. In 2 cases, the seizures were successfully controlled with phenobarbital or levetiracetam. Two cats were also euthanized in the follow-up period due to non-CHPSS-related causes.

Ultrasound after surgery was performed in 4 of 30 (13%) cats. In 1 case, serial ultrasound were performed and revealed the presence of a very small residual flow 2 months after surgery; however, the liver received the majority of the blood flow and was increased in size. One cat had residual flow 4 months after surgery. This cat had a normal bile acid stimulation test and was clinically well up to the last follow-up 60 months after surgery and no subsequent ultrasounds...
were performed. The 2 remaining cats had no evidence of residual flow 2 months after surgery.

Postoperative bile acid stimulation test was performed in 28 of 34 (82%) cats. The cats that died during hospitalization and 2 of 30 (6%) cats that survived hospitalization did not have repeated bile acid stimulation test. Both of these cats were doing clinically well at their last follow-up at 36 and 40 months, respectively.

Both pre- (P < .001) and poststimulation (P < .001) serum bile acid concentration decreased significantly after surgery (Figures 1 and 2). Of the cats that survived to discharge and had a postoperative bile acid stimulation test performed, 25 of 28 (89%) cats had normal results; In these cats, bile acid stimulation test normalized within a median of 90 days (14–196 days). Of the 3 cats with an abnormal postoperative bile acid stimulation test, 1 had a patent shunt and 2 had multiple acquired portosystemic shunts. In the cat with a patent shunt, a second surgery was performed and bile acid stimulation test normalized subsequently. The 2 cats with multiple acquired portosystemic shunts were both euthanized and have been described in greater detail above.

3.5 | Follow-up

The median follow-up period was 8 months (0.5–84 months).

4 | DISCUSSION

In the present study, cats that underwent attenuation of a single CEHPSS by thin film and survived the short-term postoperative period had good mid-term outcome. However, cats that developed postoperative seizures had a poor outcome. In the short- or mid-term, seizures were the most common reason for euthanasia. In the mid-term, only 3 cats had persistent clinical signs. Bile acid stimulation test normalized after shunt attenuation in all but those 3 cats. In cases in which bile acid stimulation test remained abnormal, patent shunt or acquired portosystemic shunts were observed.

The population of cats in this study was comparable with previous similar studies3,6,7 and consisted of young cats. There was an overrepresentation of male cats1,6,21 and domestic shorthair cats, which is in agreement with previous literature.3,6,7 The most common type of CPSS was extrahepatic, similarly to previous reports.3,7,11,12 In our study, the most common anatomy of the portosystemic shunt was left gastro-phrenic. A shunt originating from the left gastric vein was the most common type of extrahepatic portosystemic shunt in a similar study.3

The clinical signs observed before surgical attenuation were similar to those described previously.3,6,7 All cats presented with signs of neurological disease that most commonly consisted of abnormal behavior (28/34—82%). Signs of gastrointestinal disease occur more commonly in dogs than cats1,26 and, in the present study, only 3 of 34 (8%) cats presented with vomiting. However, ptalism was frequent in this population (25/34—73%). Ptyalism is a common finding in cats with portosystemic shunts1,3,21 but is rarely described in dogs with this condition.6 Copper-colored irides (inappropriate for the breed) have been previously documented in cats1,3 and, in our study, 13 of 34 (38%) cats presented with this abnormality.

The total complication rate associated with thin film attenuation of CEHPSS in our study was 32% (11/34 cats), which is lower than previously reported. Cabassu et al2 reported 9 cats treated with thin film, and of the 7 surviving cats, 3 (43%) had persistent clinical signs. In another study using ameroid ring constrictors, 77% of the cats showed postoperative complications.5 In a study of cats treated with acute complete shunt ligation, 16 of 36 (44%) of the surviving cats had long-term complications.7 In a recent study27 in which outcomes of dogs treated with ameroid ring constrictor or cellophane banding were compared,
postoperative complication rates did not differ between the 2 methods and clinical outcomes were good to excellent.

In our population, postoperative seizures were reported in 23% cases (8/34 cats) which is lower in comparison to a similar study of cats treated with CB, in which 33% of cats experienced seizures. The proportion of cats developing seizures in our population was similar to the previously reported 22% using ligature but higher than in a study using ameroid ring constrictors (14%). It appears that seizures occur in similar frequency after treatment with gradual occlusion devices when compared to suture ligation.

As previously documented, development of uncontrollable seizures is the most complication after portosystemic shunt surgery in cats. In this study, the presence of seizures before surgery was not significantly associated with an adverse outcome; only 3 of the 6 cats with seizures before surgery continued to have seizures after surgery. However, death was due to seizure activity in 4 of 6 cats, which is in agreement with previous findings. The cause of post-ligation neurologic dysfunction remains unclear; potential etiologies include decreased endogenous inhibitory central nervous system benzodiazepine agonist concentrations and imbalances in excitatory and inhibitory neurotransmitters. Seizures might be secondary to hepatic encephalopathy or other metabolic abnormalities.

However, in 3 cats of this study, seizures persisted despite normal bile acid stimulation test (2 cases) and normal bile acid stimulation test and plasma ammonia concentration (1 case). All 3 cats had experienced seizures before surgery. It would have been useful to perform postmortem examination from the cats with seizures to determine whether irreversible changes were present despite normal bile acid stimulation tests.

The small sample size and the use of different medications and doses prevented us from evaluating any association between preoperative treatments, in particular, the use of antiepileptic drugs and postoperative outcomes. However, it is worth noting that all the cats that presented postoperative seizures were on treatment with antiepileptic drugs, including those that did not have seizures before surgery. Conversely, cats that were not treated with antiepileptic drugs before surgery did not develop seizures postoperatively. In addition, all the cats that developed new onset seizures after surgery were treated with levetiracetam.

The limitations of our study preclude speculation on the clinical importance of these findings; however, it raises questions on the benefit of preoperative antiepileptic drugs that will need to be addressed in future prospective studies. Interestingly, seizure aggravation after levetiracetam treatment was recently reported in 2 children with refractory epilepsies.

In a study of dogs with a CPSS surgically attenuated with ameroid ring constrictors, dogs pretreated with levetiracetam were at significantly lower risk of postoperative seizures and death and no dogs treated with levetiracetam experienced postoperative seizures. However, in a large recently published study, including 253 dogs treated with cellophane banding or either partial or complete suture ligation, prophylactic levetiracetam did not reduce postattenuation neurological signs or seizures.

No studies assessing the potential prevention of seizures in cats were published at the time of writing. Whether the use of prophylactic antiepileptic treatment has a positive, neutral, or negative effect in the development of seizures after surgery in cats remains unknown.

In this study, seizures that occurred during hospitalization did so on days 1, 2, 3, and 5, which emphasize the need for in-hospital monitoring after surgery.

Blindness or reduced vision occurred only in 2 cats (5%), whereas previously this has been reported as a common complication affecting up to 44% of cats undergoing surgery. It is not known why cats appear more likely to develop this complication than dogs but usually it resolves within 2 months after surgery. The lower number of cats with this abnormality could be due to a true lower prevalence of blindness in this population or as a consequence of the retrospective nature of the study and possible lack of information within their records.

Case fatality rate secondary to CEHPSS in our cat population was 17% (6/34) and mid-term survival rate was 82%, which is similar to previous reports in cats undergoing cellophane banding attenuation with 2 separate studies showing a survival rate of 66% at 3 years and a 100% survival rate at 2 weeks.

Regarding the correlation between postoperative serum bile acid concentration and clinical outcome, it seems that postoperative serum bile acid concentration correlated well with shunt correction given that all the cats with persistently abnormal bile acid stimulation test had either a patent shunt or multiple acquired shunts. However, postoperative repeated imaging (computed tomography angiography or ultrasound) was performed only in a limited number of cats and therefore persistent shunting could have been missed in the remaining cases.

Blindness or reduced vision occurred only in 2 cats (5%), whereas previously this has been reported as a common complication affecting up to 44% of cats undergoing surgery. It is not known why cats appear more likely to develop this complication than dogs but usually it resolves within 2 months after surgery. The lower number of cats with this abnormality could be due to a true lower prevalence of blindness in this population or as a consequence of the retrospective nature of the study and possible lack of information within their records.

Blindness or reduced vision occurred only in 2 cats (5%), whereas previously this has been reported as a common complication affecting up to 44% of cats undergoing surgery. It is not known why cats appear more likely to develop this complication than dogs but usually it resolves within 2 months after surgery. The lower number of cats with this abnormality could be due to a true lower prevalence of blindness in this population or as a consequence of the retrospective nature of the study and possible lack of information within their records.

Blindness or reduced vision occurred only in 2 cats (5%), whereas previously this has been reported as a common complication affecting up to 44% of cats undergoing surgery. It is not known why cats appear more likely to develop this complication than dogs but usually it resolves within 2 months after surgery. The lower number of cats with this abnormality could be due to a true lower prevalence of blindness in this population or as a consequence of the retrospective nature of the study and possible lack of information within their records.

Blindness or reduced vision occurred only in 2 cats (5%), whereas previously this has been reported as a common complication affecting up to 44% of cats undergoing surgery. It is not known why cats appear more likely to develop this complication than dogs but usually it resolves within 2 months after surgery. The lower number of cats with this abnormality could be due to a true lower prevalence of blindness in this population or as a consequence of the retrospective nature of the study and possible lack of information within their records.

Blindness or reduced vision occurred only in 2 cats (5%), whereas previously this has been reported as a common complication affecting up to 44% of cats undergoing surgery. It is not known why cats appear more likely to develop this complication than dogs but usually it resolves within 2 months after surgery. The lower number of cats with this abnormality could be due to a true lower prevalence of blindness in this population or as a consequence of the retrospective nature of the study and possible lack of information within their records.

Blindness or reduced vision occurred only in 2 cats (5%), whereas previously this has been reported as a common complication affecting up to 44% of cats undergoing surgery. It is not known why cats appear more likely to develop this complication than dogs but usually it resolves within 2 months after surgery. The lower number of cats with this abnormality could be due to a true lower prevalence of blindness in this population or as a consequence of the retrospective nature of the study and possible lack of information within their records.

Blindness or reduced vision occurred only in 2 cats (5%), whereas previously this has been reported as a common complication affecting up to 44% of cats undergoing surgery. It is not known why cats appear more likely to develop this complication than dogs but usually it resolves within 2 months after surgery. The lower number of cats with this abnormality could be due to a true lower prevalence of blindness in this population or as a consequence of the retrospective nature of the study and possible lack of information within their records.

Blindness or reduced vision occurred only in 2 cats (5%), whereas previously this has been reported as a common complication affecting up to 44% of cats undergoing surgery. It is not known why cats appear more likely to develop this complication than dogs but usually it resolves within 2 months after surgery. The lower number of cats with this abnormality could be due to a true lower prevalence of blindness in this population or as a consequence of the retrospective nature of the study and possible lack of information within their records.

Blindness or reduced vision occurred only in 2 cats (5%), whereas previously this has been reported as a common complication affecting up to 44% of cats undergoing surgery. It is not known why cats appear more likely to develop this complication than dogs but usually it resolves within 2 months after surgery. The lower number of cats with this abnormality could be due to a true lower prevalence of blindness in this population or as a consequence of the retrospective nature of the study and possible lack of information within their records.
histopathology in several cases, the low number of cases and events which limits statistical analysis, the lack of detailed information about the characteristics of the thin film in 2 institutions, the different anti-epileptic protocols used for each cat, the different diagnostic methods for diagnosis of the portosystemic shunts, and the lack of standardized times for reassessment of the cats and follow-up. Another limitation was the lack of postoperative gold-standard imaging techniques (i.e., computed tomography angiography) to confirm shunt closure.

5 | CONCLUSION

Congenital extrahepatic portosystemic shunt occlusion by thin film in cats carries a good short- and mid-term prognosis in cats that survive the postoperative period and do not develop postoperative seizures, the most common cause for death in this study. Bile acid stimulation test might be a good indicator of shunt attenuation and correlates well with clinical outcome.

ACKNOWLEDGMENTS

The authors thank our colleagues for assistance in providing the cases for the study and the private practitioners who referred cases for treatment. An abstract of this study (including 9 cases) was presented at the 27th ECVIM-CA Congress, Intercontinental, Saint Julian’s, Malta, 14-16 September, 2017.

CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Authors declare no IACUC or other approval was needed.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

ORCID

Paula Valiente https://orcid.org/0000-0002-9920-8113

REFERENCES

1. Berent A, Tobias K. Portosystemic vascular anomalies. Vet Clin Small Anim Pract. 2009;39:513-541.
2. Mankin TK. Current concepts in congenital portosystemic shunts. Vet Clin Small Anim Pract. 2015;45:477-487.
3. Cabasso J, Seim H, MacPhail C, Monnet E. ‘Outcomes of cats undergoing surgical attenuation of congenital extrahepatic portosystemic shunts through cellophane banding: 9 cases (2000-2007). J Am Vet Med Assoc. 2011;238(1):89-93.
4. Cocker S, Richter K. Diagnostic evaluation of the liver. In: Ettinger S, Feldman E, Cote E, eds. Textbook of Veterinary Internal Medicine. St. Louis, MO: Elsevier; 2017:1611-1621.
5. Frankel D, Seim H, MacPhail C, Monnet E. Evaluation of cellophane banding with and without intraoperative attenuation for treatment of congenital extrahepatic portosystemic shunts in dogs. J Am Vet Med Assoc. 2006;228(9):1355-1360.
6. Kyles A, Hardie E, Muhl M, Gregory C. 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(9):1341-1347.
7. Lipscomb V, Jones H, Brockman D. Complications and long-term outcomes of the ligation of congenital portosystemic shunts in 49 cats. Vet Rec. 2007;160:465-470.
8. Berent A, Tobias K. Hepatic vascular anomalies. In: Tobias K, Johnston S, eds. Veterinary Small Animal Surgery. 2nd ed. St Louis, Missouri: Elsevier; 2011.
9. Youmans K, Hunt G. Experimental evaluation of four methods of progressive venous attenuation in dogs. Vet Surg. 1999;28:38-47.
10. Tivers M, Lipscomb V. Congenital portosystemic shunts in cats: investigation, diagnosis and stabilisation. J Feline Med Surg. 2011;13(3):173-184.
11. Lipscomb V, Lee K, Lamb C, Brockman D. Association of mesenteric portovenographic findings with outcome in cats receiving surgical treatment for single congenital portosystemic shunts. J Am Vet Med Assoc. 2009;15(234):221-228.
12. Vogt JC, Krahwinkel DJ, Bright RM, et al. Gradual occlusion of extrahepatic portosystemic shunts in dogs and cats using and ameroid constrictor. Vet Surg. 1996;25:495-502.
13. Scavelli TD. Complications associated with the diagnostic, medical, and surgical management of portosystemic shunts. Probl Vet Med. 1989;1:145-158.
14. Tivers M, Lipscomb V. Congenital portosystemic shunts in cats: surgical management and prognosis. J Feline Med Surg. 2011;13:185-194.
15. Blaxter A, Holt P, Pearson G, Gibbs C, Gruffydd-Jones T. Congenital portosystemic shunts in the cat: a report of nine cases. J Small Anim Pract. 1988;29:631-645.
16. Greenhalgh SN, Dunning MD, McKinley TJ, et al. Comparison of survival after surgical or medical treatment in dogs with a congenital portosystemic shunt. J Am Vet Med Assoc. 2010;236:1215-1220.
17. Hottinger HA, Walshaw R, Hauptman JG. Long term results of complete and partial ligation of congenital portosystemic shunts in dogs. Vet Surg. 1995;24:331-336.
18. Hunt GB, Hughes J. Outcomes after extrahepatic portosystemic shunt ligation in 49 dogs. Aust Vet J. 1999;77:303-307.
19. Swalec KM, Seguin B, Johnston G. Surgical approaches to single extrahepatic portosystemic shunts. Compend Contin Educ Pract Vet. 1998;20:593-601.
20. Kummeling A, Van Sluijs FJ, Rothuizen J. Prognostic implications of the degree of shunt narrowing and the portal vein diameter in dogs with congenital portosystemic shunts. Vet Surg. 2004;22:17-24.
21. 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.
22. Bright RM, Vogt JC, Krahwinkel DJ, et al. Gradual occlusion of portosystemic shunts in dogs and cats using and ameroid ring constrictor. Vet Surg. 1997;26:253.
23. Macalinden A, Buckley C, Kirby B. Biomechanical evaluation of different numbers, sizes and placement configurations of ligacips required to secure cellophane bands. Vet Surg. 2010;39:59-64.
24. Hunt GB, Kummeling A, Tisdall PLC, et al. Outcomes of cellophane banding for congenital portosystemic shunts in 106 dogs and 5 cats. Vet Surg. 2004;33:25-31.
25. Webster C, Cooper J. Diagnostic approach to hepatobiliary disease. In: Bonagura J, Tweedt D, eds. Kirk’s Current Veterinary Therapy XV. St Louis, MO: Elsevier; 2014:569-575.
26. Winkler JT, Bohling MW, Tillson DM, et al. Portosystemic shunts: diagnosis, prognosis and treatment of 64 cases (1993-2001). J Am Anim Hosp Assoc. 2003;39:169-185.
27. Traverson M, Lussier B, Huneault L, Gatineau M, Gatineau M. Comparative outcomes between ameroid ring constrictor and cellophane banding for treatment of single congenital extrahepatic portosystemic shunts in 49 dogs (1998-2012). Vet Surg. 2017;00:1-9.
28. Fryer KJ, Levine JM, Peycke LE, Thompson JA, Cohen ND. Incidence of postoperative seizures with and without levetiracetam pretreatment in dogs undergoing portosystemic shunt attenuation. J Vet Intern Med. 2011;25:1379-1384.
29. Caraballo R, Cersosimo R, de los Santos C. Levetiracetam-induced seizure aggravation associated with continuous spikes and waves during slow sleep in children with refractory epilepsies. Epileptic Disord. 2010;12(2):146-150.
30. Strickland R, Tivers M, Adamantos S, Harcourt-Brown T, Fowkes R, Lipscomb V. Incidence and risk factors for neurological signs after attenuation of single congenital portosystemic shunts in 253 dogs. Vet Surg. 2018;1-11.
31. 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(1):57-64.

How to cite this article: Valiente P, Trehy M, White R, et al. Complications and outcome of cats with congenital extrahepatic portosystemic shunts treated with thin film: Thirty-four cases (2008-2017). J Vet Intern Med. 2020;34:117–124. https://doi.org/10.1111/jvim.15649