Supplement 1: Definition of Chronic Hepatitis[1]

Chronic hepatitis is characterized by hepatocellular apoptosis or necrosis, a variable mononuclear or mixed inflammatory infiltrate, regeneration, and fibrosis. The proportion and distribution of these components vary widely and it is necessary to include in the diagnosis the activity and stage of the disease as well as the possible cause. The activity of the disease is determined by the amount of inflammation and extent of hepatocellular apoptosis and necrosis. The stage of the disease, and the prognosis, may be determined by the extent and pattern of fibrosis and the possible presence of architectural distortion. Fibrosis may be porto-portal, porto-central and centro-central bridging, or it may dissect the lobule. It may occur associated with interface hepatitis, following collapse and condensation of the reticulin network, or by direct activation of hepatic stellate cells with perisinusoidal deposition of collagen. Regeneration and regenerative nodules of hepatic parenchyma are often seen, as well as proliferation of ductular structures at the periphery of the parenchyma and within fibrous septa. Histochemical stains for connective tissue may be helpful in detecting the amount and pattern of fibrosis, particularly in early and mild disease. (Van den Ingh TS, WSAVA, 2006; Cullen et al VCNA, 2009)

Cirrhosis is the end stage of chronic hepatitis and is defined as a diffuse process characterized by fibrosis of the liver and the conversion of normal liver architecture into structurally abnormal nodules. In cirrhosis, two morphological categories can be distinguished, i.e. micronodular cirrhosis with nodules less than 3 mm that are all the same size, and macronodular cirrhosis with nodules greater than 3 mm that are of different sizes.

Lobular dissecting hepatitis is microscopcially characterized by bands of fibroblasts and thin strands of extracellular matrix between individual and small groups of hepatocytes which cause dissection of the original lobular pattern. Inflammation and hepatocellular apoptosis/necrosis are slight to moderate. Marked ductular proliferation is seen as well.

Non-specific reactive hepatitis* represents a nonspecific response of the liver to a wide variety of extrahepatic disease processes, especially febrile illnesses and inflammation somewhere in the splanchnic bed, or it represents the residual lesion of a previous inflammatory intrahepatic disease. The lesion is characterized by an inflammatory infiltrate in portal areas and parenchyma without evidence of necrosis/apoptosis or fibrosis.

[1]**van den Ingh TSGAM**, Van Winkle TJ, Cullen JM, et al. Morphological classification of parenchymal disorders of the canine and feline liver: 2 Hepatocellular death, hepatitis, and cirrhosis. In: WSAVA Standards for Clinical and Histological Diagnosis of Canine and Feline Liver Diseases. Society of Comparative Hepatology; Available at: http://www.vetvisuals.com/lms/moodle/mod/book/view.php?id=1001&chapterid=52859

*The panel prefers the term non-specific reactive hepatopathy as inflammation is only a single manifestation of the type of damage inflicted by disruption of the splanchnic circulation. Other changes include vacuolar degeneration and lipidosis.

Supplemental Table 1: Breeds with reported increased prevalence of chronic hepatitis or breed specific type of chronic hepatitis, including age and breed predisposition and role of copper

Breed	Strength of evidence for breed related disease (Groups A, B and C described below)*	Age	Sex pre-disposition	Countries and Era	Role of copper
Labrador retriever	**STRONG (many reports in groups A and C)** Group A; [1-4] Group B: Strombeck et al 1988[5]; Bosclair et al 2001; Schultheiss et al 2002; Raffan et al 2009 ; Favier et al 2013; Fieten et al 2014 ; Gomez-Selgas 2014[6-11] Group C: Hoffman et al 2006; Shih et al 2007; Smedley et al 2009; Johnston et al 2013; Langlois et al 2013; Fieten et al 2014[12-17]	Mean: 5.6 to 7 yrs Median: 8.3 to 9.3 yrs Ranges 1.8 to 15 yrs	**Strong female predominance in 6 studies from 2:1 to 4:1** Andersson & Sevelius 1991[1] ; Bexfield et al 2012; Hirose et al, 2014; Hoffman et al 2006; Smedley et al, 2009 Fieten et al 2014:[3,4,12,14,17] **No sex bias in 3 studies** Shih et al 2009; Johnston et al 2013; Langlois et al 2013[13,15,16]	Europe; USA; Japan. 1991-2014 First reported in 1988 but no increase in prevalence	**Copper involved in about 1/3 of cases reported in US and Netherlands** Smedley et al 2009[14] Hoffman et al 2006[12] Poldevarart et al 2009[2] **Copper not reported in UK** House et al[a] 2013 **Concurrent copper-associated proximal convoluted tubule dysfunction** Langlois et al 2013[16]

American Cocker spaniel	**STRONG (many reports in group A and one in group C)** Group A: 1-4 Group B: Sevelius et al 1994 and 1995; Bosclair et al 2001; Poldervaart et al; Strombeck et al 1988; Fuentealba et al; Schultheiss et al 2002 [18,19] [2,5,7,20,21] Group C: Kanemoto et al JVIM 2013 [22]	Mean 4.6-5 yrs Median 5.5 yrs Range 1.9-11.25 yrs Bexfield et al[3] Noted ACS younger than ECS	**Male predominance in 2 studies from 2.7:1 to 5:1:** Andersson & Sevelius 1991[1]; Bexfield et al 2012[3] **No sex bias in 1 study** Kanemoto et al[22]	Europe; USA; Japan. (1988) 1991-2014 (first report in breed 1988 but unclear if really over-represented)	**Not copper associated**
English Cocker spaniel	**STRONG (many reports in group A)** Group A; Ref [1-3] Group B: Sevelius et al 1994 and 1995; Favier et al 2013; Gomez-selgas 2014; Strombeck et al 1988; Fuentealba et al; Schultheiss et al 2002 [18,9,11,5,21,7]	Mean 5 years Median 8.75 yrs Range 1.25- 14 years	**No strong gender bias.** **Female predominance in 1 study 1.8:1** Bexfield et al 2012[3] **Male bias 1 study 1.9:1** Andersson & Sevelius 1991[1]	Europe 1991-2012 Whether reported in US/Canada or not depends on whether ECS or ACS in Stombeck; Fuentalba and Schultheiss [5,7,21]	**Not copper associated**

Doberman pinscher	**STRONG (many reports in groups A and C)** Group A: 1,3, 6 and 7 Group B: Sevelius et al 1994 and 1995; Bosclair et al 2001; Schultheiss et al 2002 [18,6,7] Group C: Johnson et al 1982[23]; Crawford et al 1985 [24] ; Van den Ingh et al 1988 [25]; Mandigers 2004 and 2005 [26,27]; Spee et al 2005; [28]Thornburg 1998 [29] ; Speeti et al 2003 [30] Dyggve et al 2010 [31]	Mean 5.1 yrs Median 4 - 5.33 yrs Range 2-10 yrs Bexfield et al [3] – younger than Lab, Cockers and Cairns	**Strong female predominance ALL studies** **2:1 up to 25:1.**	Europe including Scandinavia and USA 1982-2012	**Copper associated in most references, >60% of cases** Johnson et al; Crawford et al; Van den Ingh et al; Mandigers et al 2004 and 2005; Spee et al 2005; [23-28] **Some papers particularly from Scandinavia NO copper and suggestive of immune-mediated.** Speeti et al[30]; Dyggve et al 2010[31]
West Highland white terrier	**STRONG (Two reports in each of groups A and C)** Group A: 1,2 Group B: Sevelius et al 1994 and 1995 [18,19]Favier et al 2013[9] Fuentealba et al; Schultheiss et al 2002 [7,21] Group C: Thornburg et al 1996 [32]	Mean 4.8 yrs (Andersson & Sevelius 1991[1]) Range 2 yrs to 14 yrs (Thornburg 1996[32])	**One study female predominance 1.8:1** Thornburg 1996[32] **One study no sex bias** Andersson & Sevelius 1991[1]	Europe and USA 1991-2009	**About 20% copper associated** Poldevaart et al,2009[2] Thornburg et al, 1996[32]

Scottish terrier	**Moderate (one report in Group A)** Group A: Andersson and Sevelius, 1994[1] Group B: Fuentealba et al (1 case) Schultheiss et al 2002 [21,7]	No reports	No reports	Sweden only 1991 but single reports USA and Canada	Not reported
Dalmatian	**STRONG (one report Group A and several Group C)** Group A: Bexfield et al 2012[3] Group B: Fuentealba et al; Schultheiss et al 2002 [21,7] Group C: Napier 1996; Noaker et al 1999 ; Webb et al 2002; Cooper et al 1997 [35,33,34]	**Generally younger:** Mean 6 yrs (Webb) Median 4 yrs 7 m (Bexfield) Range 2 yrs to 12 yrs (Bexfield [3]– sig younger than Lab, Cocker and Cairn)	**Female predominance** 1.5:1 - 9:1	USA first report one dog 1996- UK first report 2012+	**All dogs copper associated** Webb et al, 2002 [33] Cooper et al, 1997 [34] Napier et al, 1996 [35] Noaker et al., 1999 [35,36]
English Springer spaniel	**STRONG** **(2 references group A and one group C)** Group A: Bexfield et al 2012 and	**Tend to be younger** Median 3.6 yr or 5 yrs (Bexfield 2012 and	**Marked female predominance 2.4:1 - 3:1**	UK only 2011 - 2014 One dog Holland	Not copper associated

	Watson et al 2010 [3,42] Group B: Raffan et al 2009; Favier et al 2013; Gomez-Selgas 2014[8,9,11] Group C: Bexfield et al 2011[42]	2011[3,42]) Range: 7 m to to 11 years Bexfield et al 2012 – sig younger than Lab; Cockers and Cairns			
Bedlington terrier	**STRONG** Group C: see Twedt et al 1979[37] Hultgren et al 1986; [38] Herrtage at al 1987 a and b – case report of 2 cases + 62 sub clinical cases presented to Cambridge;[39,40] Hardy Stevens and Stowe 1975[43]	**Mean age with CHRONIC signs 5 years** **(range 5-12 yrs – Twedt et a, 1979l)** **Dogs with acute hepatitis younger.** (Twedt et al, 1979[37] < 6 yrs old) Dogs with clinical signs 2-11 years	**No sex predisposition** Twedt et al 1979; Hultgren et al 1986 [37,38]	First reported 1975 USA. Also publications from UK; Holland; Finland; Australia	**Copper associated**

		(Schultheiss et al[7])			
Miniature Schnauzer	**MODERATE** Group A : Hirose et al, 2014[4]	NA	NA	Japan 2014	Not reported
Pomeranian	**MODERATE** Group A: Hirose et al, 2014[4]	NA	NA	Japan 2014	Not reported
Miniature Dachshund	**MODERATE** Group A: Hirose et al, 2104[4]	NA	NA	Japan 2014	Not reported
Cairn terrier	**MODERATE** Group A: Bexfield et al, 2012[3]	Median: 10 yrs Range: 7 to 13 yrs	**No sex pre-disposition**	UK 2012	Not reported
Great Dane	**MODERATE** Group A: Bexfield et al, 2012[3] Group B Raffan et al 2009[8] Bosclair et al 2001[6]	Median: 6 ys 2 m Range: 1 yr 2 m to 7 ys 11 m	**No sex pre-disposition**	UK only 2009- 2012 1 dog Canada 2001	Not reported
Samoyed	**MODERATE** Group A: Bexfield et al, 2012[3]	Median: 10 yrs Range: 3 yrs 1 m to 11 yrs	No sex pre-disposition	UK 2012 1 dog USA 2002	Not reported

	Group B: Schultheiss et al 2002[7]				
German pointer	**MODERATE** Group A : Poldervaart et al, 2009[2] Group B: Favier et al 2013[9]	NA	NA	Holland 2009	Not copper associated
Yorkshire terrier	**MODERATE** One group a reference from UK (Watson et al 2011[45]) and 1 group B reference Schultheiss et al 2002[7]	NA	NA	UK 2010 1 dog USA 2002	Not reported
Jack Russell terrier	**MODERATE** Group A: Watson et al 2011[45]			UK only 2010.	Not reported
Standard poodle	**MODERATE** Group C : Jensen et al 1999 [41] Bradley et al 2015[b]	Median 7.9 yr (2.9-12.7)	None	USA 1991 and 2015	Not reported

***Group A**: There are 6 or 7 published studies where increased prevalence of chronic hepatitis was demonstrated statistically based on comparison with some form of control population.

1. **Andersson M and Sevelius E, 1991[1]:** 250 cases of histopathologically confirmed chronic hepatitis one diagnostic laboratory in Sweden + 49 dogs with histopathologically confirmed CH at the Animal Hospital of Helsingborg 1984 – 1989. Control population: Swedish Kennel Club registrations 60-70% of all dogs in Sweden)
2. **Poldevaart JH, et al. 2009:[2:]** Retrospective study of histologically confirmed hepatitis at University of Utrecht 2002-2006. 21 acute hepatitis; 67 chronic hepatitis. Control population: total clinic population

3. **Bexfield N, et al. 2012:**[3] 551 cases histopathologically confirmed chronic hepatitis from 6 histopathology labs in the UK 2001-2008. Control population: microchip data from one company from 2001 and 2008; 175,442 and 311,085 dogs respectively
4. **Hirose N, et al. 2014:**[4] 463 canine liver biopsies at Veterinary Medical Center of University of Tokyo 2006-2012. Odds ratios compared with all cases with chronic hepatitis. Some dogs were classified as cholangiohepatitis
5. **Watson PJ, et al. 2010:**[44] which was a post mortem study in Scotland where all liver pathology was included but breed predisposition for CH was calculated using relative risks
6. **Fuentealba C, et al. 1997:**[21] pathology study from Canada 34 dogs with chronic hepatitis – only one breed over-represented - Doberman pinschers accounted for 17.6% of the cases of chronic hepatitis, but only 2.6% (122/4687) of the total canine submissions.
7. **Strombeck DR, et al. 1988:**[5] also give some comparison with the general hospital population, but only one breed (Dobermans) had large enough numbers for the authors to consider this significant

Group B: Published studies describing chronic hepatitis in dogs in which breeds identified as having increased prevalence in the controlled papers described above were also represented.

Group C: Published studies describing a distinctive breed specific form of chronic hepatitis but which did not demonstrate an increased prevalence by reference to any control population.

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Supplemental Table 2: Comparison of different hepatic biopsy techniques

Technique/Characteristic:	**Percutaneous ultrasound guided needle biopsy**	**Laparoscopic biopsy using biopsy forceps**	**Surgical biopsy during laparotomy**
Invasiveness	- Least invasive - Minimal pain	- Less invasive than surgery - Requires pain medication	- Most invasive - Requires pain medication
Sedation/anesthesia	- Requires heavy sedation/short anesthesia	- General anesthesia	- General anesthesia
Adequacy of samples	- Poor to adequate - Depends on needle gauge - Only 2-4 portal tracts with 18 G o 16 G: less than 42% had > 6 portal tracts (V)	- Typically adequate. - Using 5 mm forceps, mean of 16-18 and 8-13 portal triads per biopsy o Only 3.4 (2.7-4.2) in one	- Typically adequate - >30 per biopsy

	- Fragmented especially with fibrotic livers - Small livers may be difficult to biopsy - Typically biopsies from L lateral and L medial lobe only - Recommend 14G in most dogs, 16G in small dogs (<12 kg)	study	
Number and location of samples	- Limited, especially in small livers	- Unlimited	- Unlimited

Recognition and control of hemorrhage	- No direct visualization, rely on post-procedural imaging (US) and monitoring of clinical status and PCV, TS - Highest risk for bleeding	- Direct visualization of bleeding, with control of hemorrhage through application of electrocautery or topical coagulation agents (e.g., Gelfoam®, Pfizer, NY, NY) if necessary	- Direct visualization of bleeding with electrocautery or application of topical coagulation agents (e.g., Gelfoam®) if necessary
Cost	- Least expensive	- Intermediate expense	- Most expensive in some locations
Availability	- Many practices have access to ultrasound	- Requires specialized equipment and training	- No specialized equipment or training necessary beyond sound surgical skills
Post procedure	- +/- pain medications	- Pain medications	- Pain medication

monitoring/care	- PCV, TS, HR, RR, CRT, BP q 2 hrs for 6-8 hr - Monitoring at home or in hospital for up to 24 hrs	- Same day discharge or overnight stay depending on case - HR, RR, CRT, PCV, TS, BP (usually q 2 hrs for 6-8 hr)	- Monitor in hospital for 24 hr - HR, RR, CRT, PCV, TS, BP (usually q 2 hrs for 6-8 hr)
Time to Complete	- 10 to 15 minutes	- 35 min (13-60 min) depending on skill	- Varies with surgeon's skill
Complication Rate	- 1.9%, 2.4%, 4.3%	- 1.9% and 3.4%	- Undocumented
Ability for further assessment and/or intervention	- Ultrasound used to image the biliary tree and pancreas - No ability for therapeutic interventions - Can safely obtain bile by cholecystocentesis - Contraindicated if	- Direct visualization of the biliary tree and pancreas - No ability for therapeutic interventions - Can safely obtain bile by cholecystocentesis	- Direct visualization of the biliary tree and pancreas - Ability for therapeutic interventions - Can safely obtain bile or gallbladder

	suspected extrahepatic bile duct obstruction		wall biopsy

BP= blood pressure, CRT= capillary refill time, HR= heart rate, PCV= packed cell volume, RR= respiratory rate, TS= total solids.

Supplemental Table 3: Summary of studies looking at survival in dogs with chronic hepatitis

Histopathologic Diagnosis	**Breed**	**Study Population**	**Survival time Days**	**Reference**
Chronic hepatitis	Mixed	n=97*	541 (21-1323)	Strombeck 1977-1985 (163)
Chronic hepatitis	Mixed	n=20	630 (1-2610)	Sevelius 1987-1994 Excludes dogs with cirrhosis (169)
Chronic hepatitis	Labrador	n=24	374 (1-2645)	Shih 2000-2005

				(167)
Chronic hepatitis	American Cocker Spaniel	n=13	913 (63-1981)	Kanemoto 2003-2009 (164)
Chronic hepatitis	English Springer Spaniel	n=68	189 (1-1211)	Bexfield 2006-2010 (160)
Chronic hepatitis	Mixed	n=39	197 (1-2677)	Gomez-Selgas 1999-2010 (175)
Chronic hepatitis	Mixed	n=20	729 (342-1113)	Raffan 1996-2005 Excludes dogs

				with ascites (176)
Chronic hepatitis	Mixed	n=43	549 (0-1470)	Poldervaart 2002-2006 Idiopathic CH (90)
Chronic hepatitis	Mixed	n=23	507 (204-810)	Poldervaart et al 2002-2006 Copper associated CH (90)
Chronic hepatitis	Mixed	n=17	990	Favier * excluding dogs

				with cirrhosis (approximately 600 days with cirrhotics (165)
Chronic hepatitis	Dobermans	n=16	44 (2-284)	Speeti*** Survival after onset of clinical signs (156)
Cirrhosis	Mixed	n=20	< 7 (0-60)	Sevelius 1997-1984 (169)

Cirrhosis	Mixed	n=19	39	Favier (165)
Ascites	Mixed	n=7	32	James (184)
Ascites	Mixed	n=14	12	Raffan (176)
Lobular Dissecting Hepatitis	Mixed	n=7	21 (12-810)	Poldevaart 2002-2006 (90)
Lobular Dissecting Hepatitis	Mixed	n=6	75 (0-150)	Bennett 1983 (178)

Supplemental Table 4: Prognostic Factors in chronic hepatitis in dogs

Category	Predictive Factors	REF	Finding
Clinical Pathology	Anemia	Kilpatrick (187)	Shorter overall survival
	Thrombocytopenia	Shih (167)	Survival less than 2 months
	Hyperbilirubinemia	Gomez-Selgas (175) Poldevaart (90) Kilpatrick (187)	Shorter overall survival Shorter overall survival* Shorter overall survival
	Hypoalbuminemia	Strombeck (163) Poldervaart (90)	Shorter overall survival Shorter overall survival*
	Increased PT	Strombeck (163) Shih (166) Poldevaart (90)	Predicted death within 1 wk Survival less than 2 months Shorter overall survival
	Increased aPTT	Strombeck (163) Shih (167) Poldevaart (90)	Predicted death within 1 wk Shorter overall survival Shortened overall survival*
	Low glucose	Strombeck (163)	Predicted death within 1 wk

	Hypoglobulinemia	Sevelius (169) Shih (167)	Shorter overall survival (alpha globulins) Shorter overall survival
Histopathology	Severity of fibrosis	Strombeck (163) Poldevaart (90)	Predicted death within 1 wk Shorter overall survival
	Degree of necrosis	Strombeck (163)	Predicted death within 1 wk
	Bridging fibrosis	Strombeck (163)	Shorter overall survival
	Marked fibrosis	Hoffmann (abstract)*	Shorter overall survival
Imaging	Ascites	Raffan (176) Poldevart (90)	Shorter overall survival Shorter overall survival*
	Small liver on US	Poldevaart (90)	Shorter overall survival
	Enlarged portal lymph nodes	Poldevaart (90)	Shorter overall survival
Clinical Scores	SIRS	Kilpatrick (187)	Shorter overall survival
	Clinical Score	Shih (167)	Shorter overall survival

*__Hoffman AR__, Gold RM, Sucholdolski JS,

Steiner JM, Lidbury JA. Hepatic Fibrosis is Associated with Shortened Survival Times in Dogs with Chronic Hepatitis.ACVIM 2017, National Harbor, MD

Supplemental Table 5: Treatment of complications of chronic hepatitis in dogs

Complication	Therapeutic Approach	Dose/Modulation	Side Effects
Portal hypertension	Careful attention to volume status to avoid hypervolemia No drugs shown to decrease portal pressure in the dog	NA	-NA
Ascites	Dietary Na restriction	<0.05 g/100 kcal	-None
	Spironolactone	1-2 mg/kg/day if no response in 7-10 days titrate dose up q 3-5 days to maximum of 4 mg/kg day	-Hyperkalemia -Dehydration
	Furosemide	0.5-1.0 mg/kg/day	-Hypovolemia -Metabolic alkalosis -Hypokalemia
	Paracentesis	Remove 50% of ascitic volume*	-Hypovolemia and worsening of hypoalbuminemia especially with removal of large volumes

Hepatic Encephalopathy	Dietary protein modulation	< 5g/100 kcal diet titrated to maximum tolerated amount High biologic value, highly digestible, soy, dairy and vegetable sources. Avoid red meat	-Hypoalbuminemia that worsens ascites -Muscle catabolism that worsens HE
	Lactulose	0.25-0.5 ml/kg PO q 8-12 hrs titrate to 2-3 soft stools a day	-Diarrhea -Flatulence -Abdominal cramps
	Metronidazole	7.5 mg/kg PO BID	-Neurotoxicity (rare) -Cytopenia (rare)
	Neomycin	20 mg/kg PO BID	-Not recommended due to oto- and renal toxicity
	Amoxicillin/ampicillin	10 mg/kg BID	-Diarrhea -Vomiting
Coagulation	Severe prolongation of PT, aPTT (>1.5 X ULN) with	Fresh frozen plasma (FFP) and/or use	-Prothrombotic -Proinflammatory

	sign of bleeding	whole blood if anemia or overt signs of hemorrhage 10-15 ml/kg IV	-Volume overload -Immunological reactions
	Thrombocytopathia	DDAVP 1-2 ug/kg SubQ	
	Vitamin K deficiency	0.5 mg/kg SQ BID for 3 days then weekly	-Anaphylactic if given IV
	Hypofibrinogenemia	FFP (above) Cryoprecipiate 1 unit/10 kg IV	
	Hyperfibrinolysis	Aminocaproic acid 50-100 mg/kg IV or 15 mg/kg PO q 8	-GI upset
	Evidence of acute thrombosis	Clopidogrel 2 mg/kg PO q 24 Enoxaparin 0.8 mg/kg SQ q 8 hrs Dalteparin 150 units SQ q 8 hrs Rivaroxaban Dose not well established	-Bleeding

Infection	Ideally base antibiotic therapy on culture and sensitivity Empiric choices: Fluoroquinolone in combination with penicillin/beta lactamase inhibitor	Enrofloxacin 10 mg/kg PO/IV q 24 plus Clavulanic acid/amoxicillin 13.75 mg/kg PO q 12 hrs or Ampicillin/sulbactam 30 mg/kg IV q 8 hr	-Vomiting -Diarrhea
Gastroduodenal ulceration	Omeprazole Famotidine Sucralfate Avoid NSAID use	1 mg/kg PO BID 1 mg/kg PO BID ¼- ½ of a1gram tablet (250 – 500 mg) for toy breed dogs; up to 1 gram per large dog PO TID	-Nausea, vomiting -Acute kidney injury (humans) -Rare -Constipation -Interference with drug absorption

*Removal of larger volume of ascitic fluid can, although rarely, precipitate decreased renal perfusion or due to depletion of albumin an oncotic emergency. For larger volume paracentesis, IV administration of crystalloids or colloids may be indicated.

DDAVP= desmopressin, FFP= fresh frozen plasma, NSAID = nonsteroidal anti-inflammatory agent