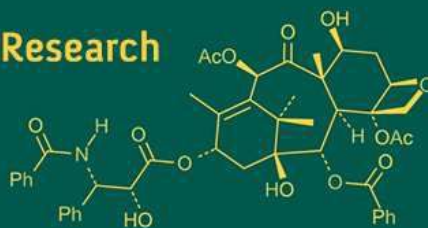
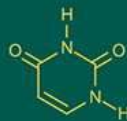


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Case review: Clinical management of cholelithiasis and ascites in dog

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Abstract

One of the most prevalent conditions in dogs is ascites, which has a variety of underlying causes. Here, at PetVet Veterinary Clinic, a male dog, two years old, with a distended abdomen from ascites and other concomitant symptoms was presented. With the use of laboratory testing, diagnostic techniques, and suitable therapy, such as double diuretic regimens, choleretics, liver enzyme, intravenous infusions of critical amino acids, etc. the following case was treated. The animal recovered after receiving medication for 15 days.

Keywords: Ascites, cholelithiasis, dog's gall bladder, hyperlipidaemia, hypoproteinaemia

Introduction

A transudate or modified transudate accumulating in the peritoneal cavity is known as ascites. Although it is rarely enough on its own to produce fluid accumulation, hypoalbuminemia plays a significant role in the development of ascites; portal hypertension is crucial. Renal sodium retention appears to be a corollary of the development of ascites in liver disease patients. Systemic hypotension and increased renal sodium retention are common. This is caused in part by decreased sodium delivery to the tubules and a decreased glomerular filtration rate, as well as increased release of renin-angiotensin-aldosterone (RAAS), which increases sodium retention in the distal tubules. As a result, there is an increase in the volume of fluid in circulation, which speeds up the development of ascites. This starts a vicious cycle that results in renal sodium retention and ascites, limits venous return, and increases pressure on the caudal vena cava. When furosemide is administered alone, hemoconcentration and subsequent increases in RAAS activation might further lower systemic blood pressure, which can be ineffectual or, in certain situations, even increase the volume of effusion.

Materials and Methods

Case History and Clinical findings

"Lucky" a 2-year dog, sex-male, breed- labrador, fully vaccinated weighing 25 kg with complaint of bulged abdomen, inappetence, frequent vomiting was presented to PetVet veterinary clinic, Kalyan-421301. Prima facie dog appeared active, alert but slight discomfort was exhibited while walking mostly due to bulged abdomen hindering the locomotion.



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Inappetence since, 4-5 days, bulged abdomen since last 3 days, water intake-normal, urine output-normal. The dog been dewormed regularly in every 2 months. The food offered to dog was home based, high carbs-rice, high fat and sweet in nature. It came to notice that food offered since puppyhood lacks fibre and protein in diet.

On Physical examination - Abdominal palpation - No pain but bulging was there and fluid thrill was felt indicating

ascites. The body temperature was 101.5 F. Lymph nodes were normal in size. On auscultation no abnormal lung sounds heard.

After careful examination the blood was drawn from cephalic vein of dog for complete blood count (cbc), liver function test (LFT), Kidney function test (KFT) and Serum cholesterol estimation.

Sr. No.	Parameters	Test value
1.	Haemoglobin	10.9 mg/dl
2.	Total erythrocyte count (TEC)	$5.2 \times 10^6/\mu\text{l}$
3.	Total leucocyte count (TLC)	$8.2 \times 10^3/\mu\text{l}$
4.	PCV	32.1%
5.	Platelets	$149 \times 10^3/\mu\text{l}$
6.	Blood urea nitrogen (BUN)	22 mg/dl
7.	Creatinine	1.1 mg/dl
8.	ALT	95 U/L
9.	AST	67 U/Ls
10.	Total Billirubin	0.40 mg/dl
11.	Albumin	1.9 g/dl
12.	Total Protein	4.8 g/dl
13.	Serum Cholesterol	330mg/dl

Post blood collection, dog subjected to ultrasonographic examination to ensure presence of ascitic fluid inside abdominal cavity. Abdominal USG revealed anechoic area, free fluid surrounding the liver and externally shown severe

abdominal distension. USG also carried out to ascertain gall bladder which resulted in observation of presence of large stone of 2.3 cm in length.



Pic 1: Indicating presence of gall bladder stone



Pic 2: Indicating presence of 2.33 cm gall stone

To provide emergency care and mitigate the dangerous discomfort being observed in dog, abdominocentesis was performed using 22G scalp and 150 ml of ascitic fluid removed. This abdominocentesis was performed for next 4 days with equal amount of fluid being removed. Also, owner was advised to perform continue monitoring of abdominal girth to ascertain reduction in abdominal fluid externally. Since the day abdominocentesis process was initiated, dog showed marked improvement in overall appearance.

To arrive at line of treatment to be administered to the dog, few points were considered based on the investigation carried out. The absence of low fibre and protein diet with high carbohydrates and Fat predisposes canine to cholelithiasis and hyperlipidemia in serum sample also reiterates this fact. One of the reason of presence of ascitic

fluid inside the abdominal cavity is hypoproteinemia and Hypoalbuminemia which causes leakage of fluids which percolates and accumulates in the ventral portion of body thereby manifesting ascites.

Treatment

1. Tab. Ursodeoxycholic acid @Ursocol @5 m/Kg P.O 12 hrs interval for 15 days.

As per Allan *et al.*, 2021 ^[1], By dislodging hydrophobic bile acids, ursodeoxycholic acid (UDGA), a synthetic hydrophilic bile acid, reduces oxidative and hepatocellular damage brought on by bile acid retention in the liver.

Additionally, UDGA has anti-inflammatory and cholerectic effects on the gall bladder. (Meyer *et al.*, 1997) ^[5] (Guarino *et al.*, 2005) ^[3] (Yanaura and Ishikawa, 1978) ^[10].

2. Tab. Lisybin (Large) P.O B.i.d for 15 days

Contains S-Adenosyl-L-Methionine (SAME). One of the necessary amino acids, methionine, is the precursor of SAME, which SAME synthetase activates. Liver disease causes a significant reduction in this enzyme's activity. Methionine accumulates as a result of decreased utilization, and SAME is also reduced. As a result, SAME becomes an essential nutrient and needs to be supplied exogenously as a supernutrient to make up for its deficiency. (Lieber, 2002) [4].

3. Tab. Furosemide @3 mg/kg P.O q 8 hrs interval for 10 days.

4. Tab. Hydrochlorothiazide @2 mg/Kg P.O for 12 hrs interval for 10 days.

When furosemide is administered alone, hemoconcentration and subsequent increases in RAAS activation might further lower systemic blood pressure, which can be ineffectual or, in certain situations, even increase the volume of effusion. So here in this case Tab Hydrochlorothiazide was administered per-oral with Tab. Furosemide. It is crucial to monitor serum Potassium (K) levels as hydrochlorothiazide consistently expels K in urine. So, to avoid hypokalaemia and related symptoms continuous monitoring of K is important.

1. Inj Hermin @100 ml/day I.V for 5 days.

Injection of essential amino acids to correct low levels of albumin and total protein.

2. Inj. D25% 270 ml/day I.V for 5 days.

Result and Discussion

15th day post treatment: Blood sample collected to estimate the changes in blood parameters after 15 days of treatment.

Sr. No.	Parameters	Test value
1.	Haemoglobin	11.2 mg/dl
2.	Total erythrocyte count (TEC)	$5.5 \times 10^6/\mu\text{l}$
3.	Total leucocyte count (TLC)	$7.7 \times 10^3/\mu\text{l}$
4.	PCV	33.0%
5.	Platelets	$158 \times 10^3/\mu\text{l}$
6.	Blood urea nitrogen (BUN)	20 mg/dl
7.	Creatinine	0.9 mg/dl
8.	ALT	75 U/L
9.	AST	63 U/Ls
10.	Total Billirubin	0.30 mg/dl
11.	Albumin	2.9 g/dl
12.	Total Protein	5.4 g/dl
13.	Serum Cholesterol	295 mg/dl

Significantly reduced serum cholesterol, ALT, AST and evident increase in total protein and serum albumin was concluded.

Abdominal USG

Revealed total absence of effusion and free fluid around liver with no external ascites manifestation. Gall bladder stones size reduced to 0.4 cm. Active and alert dog whose appetite increased considerably within 7 days of treatment.



Pic 3: Indicating presence of 0.94 cm of gallstone (reduction) post 15th day of treatments

Cholelithiasis and Diet aiding its formation

Specifically, dietary patterns that are associated with higher caloric intake, high fructose intake, poor fiber content, high fat, fast food consumption, and low vitamin C intake raise the risk of gallstone formation. (Di Ciaula *et al.*, 2019) [2]. A diet high in carbohydrates and lipids and lacking in protein and fiber led to cholelithiasis. (Smith and Gee, 1979) [8]. It seems that the synthesis of lithogenic bile, which is saturated or supersaturated with cholesterol, is indispensable for the development of gallstones. (Small and Rapo, 1970) [7].

The production of lithogenic bile is aided by an increase in the relative quantity of cholesterol.

Low fiber consumption has been proposed as a significant indicator of the development of cholelithiasis.

The value of a fiber-rich diet to prevent gallstone development According to a 1980 theory by Story J.A., fiber may bind bile salts in the gut, preventing the formation of micelles and lowering cholesterol absorption. This may facilitate the excretion of cholesterol in feces. Being overweight also plays a role in the development of cholelithiasis. A diet rich in dietary fiber and one rich in protein can effectively and nearly equally reduce body weight, according to statistical studies of the results. However, a diet rich in fiber also significantly reduces the risk of biliary slug accumulation. (Sulaberidze, 2014) [9].

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