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Efficacy of various colloid supplementations in management of ascites in dogs

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Abstract
The present study on role of colloids in diagnosis and management of ascites in dogs was carried out. On screening, 174 dogs were found to be suffered from abdominal distension out of 4372 dogs. Out of which 30 cases of ascites was confirmed. Total 18 cases of ascites were selected and divided into 3 groups as T_1 to T_3 having 6 dogs each. Group T_1 received the Polygeline polypeptides of degraded gelatin, Group T_2 received amino acids and group T_3 received Dextran 40 @ 5-10 ml/kg b.wt. of 3 doses on alternate day. On the basis of Serum ascitic albumin gradient ascites was low gradient (60%) and high gradient (40%). Total protein and albumin levels were significantly lowers before treatment however, they become under physiological range after treatment. On the basis of recovery pattern of ascites, group T_2 showed maximum recovery followed by T_3 group and least in T_1 group.

Keywords: Colloids, ascites, protein, dogs

1. Introduction
Ascites is referred as accumulation of serous fluid in peritoneal cavity, has been attributed to chronic hepatic failure, congestive heart failure, nephritic syndrome, malnutrition and protein losing enteropathy in canines. It results in abdominal swelling, dyspnea, lethargy, anorexia, vomition, weakness and discomfort (Regmi and Shah, 2017) [25]. However, recent evidences contradict these theories and suggest that renal mechanism leading to retention of sodium and water are primary events in development of ascites in hepatic diseases. Reduced albumin levels also contribute to onset of ascites (Richter, 1996) [26].
Diagnostic evaluation of an animal presented with ascites may include a complete blood count (CBC), biochemical evaluation, abdominal paracentesis (biochemical, cytological analysis of ascetic fluid, radiographs, organ function tests and ultrasonography. Moore (2005) [16] studied colloid therapy in emergency medicine and stated that combination of crystalloid and colloid @ 5-20 mg/kg of 6% hetastarch and dextran along with 15-30 ml/kg of crystalloid fluid was effective in hypoproteinemic dogs.
However, limited study was conducted to know the role of colloid in diagnosis and management of ascites in dogs. Hence, this study was aimed to determine the efficacy of various colloid supplementations in management of ascites in dogs.

2. Materials and Methods
The proposed work was conducted in the Department of Veterinary Medicine, College of Veterinary Science & Animal Husbandry, Nanaji Deshmukh Veterinary Science University (NDVSU), Jabalpur, Madhya Pradesh from September 2019 to March 2020.
A total of 174 dogs brought to veterinary clinical complex (VCC) with the complaint of abdominal distension were screened for ascites and were subjected to detail study. Out of 174 dogs, 18 dogs were selected for therapeutic study along with six apparently healthy dogs as healthy control group. All the suspected dogs were clinically examined for the presence of abdominal distension. The following clinical parameters were recorded on day 0 (pre-treatment) and on days 7 and 14 (post-treatment).

Approximately 3 ml of blood sample was collected aseptically from saphenous or cephalic vein from each dog, out of which 1 ml was poured into vial containing EDTA, subjected to routine haematology and 2 ml into clot activator vial for serum. Serum was harvested after centrifugation, frozen and stored at -20 °C until further biochemical analysis.

2.1 Haematological parameter

The haematological parameters were estimated on day 0 (pre-treatment) and on days 7 and 14 (post-treatment) using semi-automated haematology analyser (Abacus vet 5). Differential leucocytes count (DLC) was done as per standard procedure (Jain, 1986) [8].

- Haemoglobin concentration (Hb, g/dL)
- Total leucocytes counts (TLC, 10^3/µl)
- Differential leucocytes count (DLC, %)

2.2 Biochemical parameter

The blood biochemical profile was determined on day 0 (pre-treatment) and on days 7 and 14 (post-treatment). The following parameters were estimated by using commercially available kits with the help of auto-biochemical analyser (Erba CHEM -5 Plus) using readymade kits (Erba Mannheim, Transasia biochemical, India, Pvt. Ltd).

- Serum ALT (alanine amino transferase) (U/L)
- Serum AST (aspartate aminotransferase) (U/L)
- Serum creatinine (mg/dL)
- Serum total protein (g/dL)
- Serum albumin (g/dL)
- Serum globulin (g/dL)

3. Therapeutic Study

For the therapeutic study a total of 18 ascitic dogs were selected and divided into 3 groups as T_1 to T_3 having 6 dogs in each group. Six coeval apparently healthy dogs served as a healthy control group T_0. The therapeutic regimens T_1 to T_3 are specified as follows in table 01.

Table 1: Therapeutic protocol in various treatment groups

Treatment groups	No. of dogs	Treatment		
		Drug	Dose and route	Duration
T_0	6	Apparently healthy		
T_1	6	Polygeline polypeptides of degraded gelatin	@ 5-10 ml/kg B.wt. I/V	Alternate day, 3 doses
T_2	6	Amino acids	@ 5-10 ml/kg B.wt. I/V	Alternate day, 3 doses
T_3	6	Dextran 40	@ 5-10 ml/kg B.wt. I/V	Alternate day, 3 doses

- Fluid therapy (crystalloids), diuretics, symptomatic and supportive therapy was given as per clinical condition and organ(s)/ system(s) involved.

4. Therapeutic Response Evaluation

The response to colloid therapy with different specified colloid was evaluated on the dependable basis of the covert recovery of ascites by reduction of abdominal distension.

5. Statistical Analysis

The recorded experimental data were analysed by ANOVA and mean was compared by DMRT as per the standard procedure outlined by Snedecor and Cochran (1994) [30].

6. Results and Discussion

The mean value of temperature remains in normal physiological range in all the treatment groups. Pulse rate and respiration rate were within normal range in T_1 group and higher than normal physiological values in T_2 and T_3 group. Similar results were also observed by Kumar and Srikala (2014) [13]; Kashyap *et al.* (2015) [11]; Mukherjee *et al.* (2017) [18]; Regmi and Shah (2017) [25] however, high temperature was observed by Dabas *et al.* (2011) [2] and Dhillon *et al.* (2020) [3]. The reason behind increase in pulse and respiration in ascitic dogs might be due to the effect of catecholamines and other compensatory mechanism of heart to maintain oxygen supply to tissues. Higher respiration and pulse rates could be due to abdominal distension to meet out the oxygen and nutrients demand of the body tissues.

Hematological study revealed slight decrease in Haemoglobin concentration which is similar to the report of Kumar *et al.* (2003) [14]; Pradhan *et al.* (2008) [24]; Sarvanan *et al.* (2013) [27]; Mukherjee *et al.* (2017) [18]; Regmi and Shah (2017) [25]; Phom *et al.* (2019) [22]; Singh *et al.* (2019) [29] and Dhillon *et al.* (2020) [3]. Apart from kidney, liver is the main organ which is involved in the secretion of erythropoietin and other factors required for erythropoiesis and thereby liver dysfunction leads to anemia. Decrease in haemoglobin concentration is due to increased degradation of erythrocytes due to prolonged transit time through spleen because of decreased portal blood flow and/or increased fragility of erythrocytes due to high levels of bile acids. All these further impair bone marrow responses, decreased erythrocyte survival time and decreased nutrient uptake as a results of inappetance or anorexia and reduced availability of micronutrients from liver.

Higher physiological value of TLC occurs in pathological conditions like infection, neoplasia, liver cirrhosis and ascites in dogs. Elevated leucocytes are characteristic of inflammatory process. The findings of total leucocyte count in present study are in accordance with the observations of earlier workers Parker (2002) [21]; Kumar *et al.* (2003) [14]; Nottidge *et al.* (2003) [19]; Benjamin (2005) [1]; Pradhan *et al.* (2008) [24]; Phom *et al.* (2019) [22]; and Singh *et al.* (2019) [29]. Increase in neutrophils and monocytes in all treatment groups which are similar to the reports of Pradhan *et al.* (2008) [24] and Phom *et al.* (2019) [22]. Significant increase in mean values of lymphocytes in T_2 group was in accordance to the finding of Singh *et al.* (2019) [29] and contrary to Phom *et al.* (2019) [22] which found decrease in lymphocytes. Increased level of TLC and neutrophils are observed in liver cirrhosis and ascites in dogs (Parker, 2002; Pradhan *et al.*, 2008) [21, 24]. Monocytes increased in all treatment groups whereas eosinophils showed no significant change between intervals in all treatment groups which is similar to the findings of Mukherjee *et al.* (2017) [18].

Table 2: Result of various parameters in different treatment groups with time

Parameters	Groups	Day		
		0	7	14
Temperature (°F)	**T_0**	101.48±0.07	101.50^{ab}±0.11	101.31±0.24
	T_1	101.08^{B}±0.50	102.28^{aA}±0.22	101.71^{AB}±0.28
	T_2	101.90±0.49	101.46^{ab}±0.23	101.25±0.09
	T_3	101.26±0.80	101.31^{b}±0.43	101.21±0.28
Respiration rate (breaths/minute)	T_0	27.66^{b}±1.33	28.00^{b}±1.73	27.5 0±2.32
	T_1	35.66^{ab}±1.89	35.83^{ab}±1.89	32.50±2.68
	T_2	45.33^{aA}±3.73	33.83^{aAB}±1.22	32.66^{B}±2.34
	T_3	42.00^{a}±5.89	37.50^{a}±3.27	33.83±2.32
Pulse (beats/minute)	T_0	89.00^{b}±2.08	88.00^{b}±1.52	86.83^{b}±1.77
	T_1	101.33^{ab}±4.93	101.66^{ab}±4.38	93.50^{ab}±2.96
	T_2	106.50^{abA}±2.91	99.00^{abAB}±6.16	91.83^{abB}±1.85
	T_3	111.66^{a}±9.85	106.83^{a}±5.83	98.33^{b}±4.43
Haemoglobin concentration (g/dL)	T_0	14.83^{a}±0.94	15.50^{a}±0.42	15.66±0.66
	T_1	10.86^{b}±0.77	11.00^{b}±0.97	12.16±1.13
	T_2	10.48^{b}±1.00	11.18^{b}±0.94	12.28±1.11
	T_3	12.21^{ab}±1.71	12.28^{b}±1.35	12.08±1.48
Total leucocyte count (10^3/µl))	T_0	11.13^{c}±1.61	11.25^{c}±1.34	11.21^{b}±0.90
	T_1	16.23^{b}±2.20	16.24^{ab}±1.85	17.48^{a}±0.94
	T_2	13.16^{bc}±1.01	13.10^{bc}±1.56	12.04^{b}±1.46
	T_3	16.37^{b}±1.38	18.24^{a}±1.51	17.31^{a}±1.17
Neutrophil (%)	T_0	71.00±1.94	70.16±1.90	67.00^{b}±2.29
	T_1	76.66±3.11	73.16±1.93	75.00^{a}±1.89
	T_2	79.33±2.96	75.33±1.20	73.33^{a}±0.98
	T_3	77.16±2.62	75.33±1.42	75.50^{a}±1.38
Lymphocyte (%)	T_0	20.16^{b}±1.95	20.33±2.47	19.66±2.43
	T_1	18.66^{ab}±2.65	20.33±2.04	18.00±1.96
	T_2	12.66^{abB}±2.38	18.66^{A}±1.68	20.33^{A}±0.84
	T_3	18.33±1.74	18.50±0.80	15.66±1.78
Monocyte (%)	T_0	6.83^{a}±0.94	6.16±1.04	5.83±0.94
	T_1	3.33^{b}±0.84	4.33±0.88	5.00±0.51
	T_2	3.50^{b}±0.56	3.33±0.98	4.50±0.76
	T_3	2.66^{bB}±0.55	5.00^{A}±0.77	6.33^{A}±0.61
Eosinophil (%)	T_0	4.50^{a}±1.17	5.33^{a}±1.33	4.50^{a}±0.76
	T_1	1.16^{b}±0.79	2.00^{b}±0.68	2.00^{b}±0.73
	T_2	1.83^{ab}±1.07	2.66^{b}±0.76	2.00^{b}±0.89
	T_3	2.00^{ab}±0.63	1.16^{b}±0.54	2.50^{b}±0.42
Alanine aminotransferase activity (U/L)	T_0	56.33^{c}±09.54	54.50^{b}±06.40	56.00^{b}±07.48
	T_1	133.46^{a}±18.08	121.80^{a}±20.40	100.41^{a}±18.19
	T_2	86.02^{bc}±22.04	74.85^{b}±14.55	60.25^{b}±08.72
	T_3	163.91^{a}±20.95	133.20^{a}±13.89	121.73^{a}±12.69
Aspartate aminotransferase activity (U/L)	T_0	38.66^{b}±06.48	34.66^{c}±06.48	28.83^{b}±04.88
	T_1	70.68^{a}±09.67	68.63^{ab}±12.54	61.83^{a}±11.46
	T_2	71.33^{a}±06.32	62.06^{bc}±05.66	60.93^{a}±07.58
	T_3	98.53^{a}±12.84	94.90^{a}±11.45	82.43^{a}±13.00
Creatinine concentration (mg/dL)	T_0	7.28^{a}±0.59	6.70^{a}±0.50	7.90^{a}±0.25
	T_1	5.26^{b}±0.66	5.36^{b}±0.40	5.66^{b}±0.57
	T_2	4.96^{b}±0.34	5.33^{b}±0.38	5.95^{b}±0.38
	T_3	3.93^{bB}±0.35	4.63^{bAB}±0.32	5.20^{bA}±0.40
Serum total protein concentration (g/dL)	T_0	7.28^{a}±0.59	6.70^{a}±0.50	7.90^{a}±0.25
	T_1	5.26^{b}±0.66	5.36^{b}±0.40	5.66^{b}±0.57
	T_2	4.96^{b}±0.34	5.33^{b}±0.38	5.95^{b}±0.38
	T_3	3.93^{bB}±0.35	4.63^{bAB}±0.32	5.20^{bA}±0.40
Albumin concentration (g/dL)	T_0	2.73^{a}±0.35	2.71^{a}±0.33	2.33^{ab}±0.22
	T_1	1.56^{bB}±0.12	1.92^{bcAB}±0.11	2.33^{abA}±0.19
	T_2	1.87^{bB}±0.12	2.50^{abAB}± 0.15	2.90^{aA}±0.27
	T_3	1.62^{bB}±0.27	1.73^{cAB}±0.17	2.11^{bA}±0.22
Globulin concentration (g/dL)	T_0	4.58^{aAB}±0.58	3.63^{B}±0.64	5.86^{aA}±0.26
	T_1	3.70^{ab}±0.60	3.44±0.50	3.33^{b}±0.51
	T_2	3.09^{ab}±0.57	2.83±0.50	3.05^{b}±0.49
	T_3	2.30^{ab}±0.27	2.90±0.32	3.08^{b}±0.36

Mean values with different superscripts (uppercase) differ significantly (p<0.05)

Alanine aminotransferase study revealed increase in ALT concentration which is similar to the reports of Kumar *et al.* (2003) [14]; Saravanan *et al.* (2013) [28]; Kumar and Srikala (2014) [13]; Regmi and Shah (2017) [25]; Phom *et al.* (2019); Singh *et al.* (2019) [29] and Dhillon *et al.* (2020) [3]. The decreasing values of ALT from day 0 to day 14 suggest that the liver parenchyma is regenerating and started functioning physiologically. ALT is an enzyme normally found in the cytosol of hepatocytes. Consequently, serum levels will only be elevated in conditions where there is increased permeability of plasma membranes. In more chronic liver disease, plasma membrane permeability is often normal. Increased levels of ALT showed necrotic damage of liver resulting in leakage of enzymes from hepatic cells in the blood. Therefore, marked elevations are more commonly seen in diffuse than in localized liver disease. ALT levels also will vary with the stage of disease when the sample is collected. ALT has a circulatory half life of two to four days; therefore, a twofold elevation in ALT due to acute liver necrosis may be expected to have returned to normal range within two days.

Increased AST indicates hepatic insufficiency with extensive damage resulting into the leakage of enzymes from hepatic cell into blood stream. Similar results were also observed by Kumar *et al.* (2003) [14]; Pradhan *et al.* (2008) [24]; Kumar *et al.* (2016) [12]; Regmi and Shah (2017) [25]; Singh *et al.* (2019) [29] and Dhillon *et al.* (2020) [3]. It is important to remember that the magnitude of the increase in enzyme levels is proportionate to the damage. Lower AST values were indicating towards regeneration of hepatic parenchyma.

Increased creatinine values could be attributed to impaired kidney function associated with liver damage due to the decreased capacity of liver to detoxify the harmful products. Similar results were also observed by Pradhan *et al.* (2008) [24]; Kumar *et al.* (2016) [12]; Phom *et al.* (2019) [22] and Singh *et al.* (2019) [29]. Renal dysfunction is a frequent complication in patients with end stage liver diseases. The characteristic of hepato renal syndrome is severe renal vasoconstriction with major peripheral arterial vasodilation. Tubular function is damage with histological changes.

Liver is the primary site for the synthesis of major plasma proteins as well as site of synthesis and degradation of many other proteins therefore these are influenced by liver diseases in many ways (Webster, 2005) [33]. Similar finding of total protein was also reported by Kumar *et al.* (2003) [14]; Regmi and Shah (2017) [25]; Phom *et al.* (2019) [22]; Singh *et al.* (2019) [29] and Dhillon *et al.* (2020) [3]. Decreased serum total protein, albumin level were good indicator for diagnosis of ascites due to hepato biliary diseases (Saravanan *et al.* 2014) [27].

Hepatic failure of more than 75% can result in hypoalbuminemia. These findings are in accordance with the observations of earlier workers like Kumar *et al.* (2003) [14]; Regmi and Shah (2017) [22]; Phom *et al.* (2019) [22] and Singh *et al.* (2019) [29]. Hypoalbuminemia occurs due to low synthesis or higher loss of albumin, redistribution of albumin outside the intravascular space and dilution of albumin inside intravascular space. Many factors influence albumin synthesis, but most common are liver failure, inflammation and malnutrition. Ascites causes high level of albumin distribution and lowering the blood albumin concentration leading to decrease in plasma osmotic pressure and aggravates the formation of ascitic fluid (Tennant and Center, 2008) [32]. The findings of mean value of globulin are in accordance with the observations of earlier workers Singh *et al.* (2019) [29].

6.1 Recovery in clinical conditions

Recovery was determined on the basis of improvement in clinical conditions (*viz.* restoration of abdominal distension, respiratory distress appetite, alertness etc.). The recovery was graded as no, mild, moderate and complete recovery. In group T_2, three out of six cases were having complete recovery; two have moderate recovery and one mild recovery. While in group T_3, two out of six cases had complete recovery, one each had moderate and mild recovery and two had no recovery. In group T_1 none of the case had complete recovery while two cases each had moderate, mild recovery and no recovery.

Table 3: Recovery pattern of ascites in different treatment groups

Groups	Number of dogs				
	No recovery	Mild recovery	Moderate recovery	Complete recovery	Ranking
T_1	2	2	2	-	III
T_2	-	1	2	3	I
T_3	2	1	1	2	II

On the basis of recovery pattern of ascites (*viz.* clinical, haematobiochemical and ascitic fluid); dogs received amino acids showed maximum recovery followed by dogs on dextran 40 and least in dogs received polygeline polypeptides of degraded gelatin.

To our knowledge, this study was the first comparing clinical trial outcome after infusion of colloids in the dogs with ascites. Amino acids has been shown to be more effective than dextran 40 and polygeline polypeptides. In partial agreement to the present findings Gines *et al.* (1996) [5]; Moreau *et al.* (2006) [17]; Itou *et al.* (2009) [7] documented the variable patterns in recovery of ascites using the colloids alone or in combination. However, in present study the administration of amino acids supplementation not only reduces hypoalbuminemia but also ascites and improves quality of life in patients. Changes in amino acid metabolism are believed to participate in the pathogenesis of many complications such as hypoalbuminemia with edema and ascites. Amino acids increases synthesis and secretion rates of albumin by hepatocytes.

Amino acids promote albumin synthesis by improving amino acid imbalance. An increased supply of substrate for protein synthesis causes increases in albumin synthesis and osmotic pressure, which may in turn result in a decrease in extra cellular fluid and ascites. Albumin is regarded as treatment of choice of plasma expansion because it is known to be effective as safe, and has many biological properties that are thought to be a relevance to its action under physiological circumstances and diseases (Evans, 2002) [4]. Albumin functions as a major contributor to plasma

colloidal oncotic pressure (COP), as a carrier molecule and a scavenger of toxic substances generated during inflammatory states. Plasma oncotic pressure is maintained by the presence of protein molecule within the vascular space. One of albumin's primary functions is maintenance of intravascular oncotic pressure. In serum proteins albumin contributes approximately 80% of plasma COP (Griffel and Kauffman, 1992) [6]. COP is the principal force regulating transvascular fluid flow and is one of the forces opposing fluid exit from the vascular space. Together, the Gibbes-Donnan effect and albumin net negative charge govern albumin's water attraction (Kaminski and Haase, 1992) [9].

(a) (b)

Fig 1: a) Severe abdominal distension in Golden Retriever dog before treatment b) Recovery from abdominal distension after treatment in same dog

7. Conclusion

On the basis of recovery pattern of ascites and change in ascitic / peritoneal fluid, ascitic dogs of group (T_2) showed maximum recovery followed by dogs of T_3 group and least in dogs of T_1 group i.e. Dogs of group received amino acids (T_2 group) evoked a superior, prompt and solid response followed by Dextran 40 (T_3 group) and least in Polygeline polypeptides of degraded gelatin (T_1 group).

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