INTRODUCTION

A ‘Dog’ is considered as lovable; Loyal and friendly companion of human being. The indispensable services rendered by the dogs to the society are praise worthy. Alike human being, these pet animals also suffer time to time from variety of diseases frequently affecting the vital organs or system. The diseases affecting the vital organ like heart, lung, brain, kidney etc. are most important because these sometimes appear in life threatening condition. As kidney is one of the most important vital organs of the body, its dysfunction leads to critical condition, often proves to be fatal.

The kidney is complex, multi-functional organ of the body and it regulates essential homeostatic functions such as the balance of body water, acid-base status and electrolyte. Kidneys play a vital role in the excretion of waste products from the body and also in the reabsorption of valuable substances back into the blood circulation. Additionally, kidneys also perform many other functions like, regulation of arterial blood pressure, secretion of hormones, metabolism of vitamin-D etc. which prove that kidneys are important organ to maintain the good health and also to sustain life.

Renal failure is one of the common diseases encountered in dogs and it is a major cause of death in older animals. The underlying cause
of the disease may have occurred years earlier and remains unknown in most cases. In a study conducted by Deschepper, 1977, on 10920 cases he found that 1000 dogs had renal failure (13.8%). In a survey done on canine population the mean age of renal failure was 7 years (n=170) and in another 6.5 years (n=119) (Rubin, 1997).

Renal failure is the clinical syndrome that occurs when the kidneys are no longer able to maintain their regulatory, excretory and endocrine function results in retention of nitrogenous solutes and derangement of fluid, electrolyte and acid-base balance. Renal failure occurs when 75% or more of the nephron population is non functional (DiBartola, 2005).

The clinical signs associated with acute and chronic renal failure may be similar therefore differentiation of ARF from CRF is important for both prognostic and therapeutic reason. It is more likely that an animal with acute renal failure (ARF) will recover than an animal with chronic renal failure (CRF) (Cowgill and Francy, 2005; DiBartola, 2005 and Vaden, 1997), because ARF is potentially reversible. Accurate and early identification of ARF is crucial to allow the institution of aggressive treatment, thereby affording the best chance of animal survival (Vaden, 2000).
Acute renal failure (ARF) is a clinical syndrome characterized by rapid decline in renal function over a period of hours to days (Kraje, 2002). The causes of ARF may be divided into pre-renal, renal and post-renal categories. Pre-renal causes include any condition resulting in decreased renal perfusion such as shock or dehydration, post-renal causes include obstruction to urine flow or tear in the urinary tract. Renal causes are various and most commonly encountered are nephrotoxins and ischemia. Nephrotoxin can be therapeutic or non-therapeutic agents. Under therapeutic nephrotoxic drugs include indiscriminate use of aminoglycosides, non-steroidal anti-inflammatory drugs (NSAIDS), angiotensin-converting enzyme (ACE) inhibitors, amphotericin-B, cisplatin and non-therapeutic nephrotoxic agents like heavy metal (Mercury, uranium, lead, Bismuth salts, chromium, arsenic, gold, copper, silver, nickels, antimony etc.) and organic compounds (carbontetrachloride, ethylene glycol, chloroform, pesticides, herbicides etc.). Specific causes of ischemia include shock, dehydration, low cardiac output, thrombosis of renal vessels, hypotension. Other causes of acute renal failure include Leptospirosis, ehrlichosis, pyelonephritis etc. (Kraje, 2002; Cowgill, and Francy, 2005).

There are many similarities in the pathophysiologic findings of ARF as a result of nephrotoxins and ischemia. The kidneys are susceptible to ischemia and toxicants because of their unique anatomic and physiologic features (Labato, 2001). The kidneys receives about 20% of cardiac output
and the renal cortex receive 90% of that blood flow (Lane et al., 1994; Grauer, 1996) results in increased delivery of blood born toxicants to the kidneys compared with other organs (Labato, 2001). Within renal cortex, proximal tubules and thick ascending loop of Henle’s epithelial cells are most frequently affected by ischemia and toxicants because of their transport functions, high metabolic rates and vulnerable to hypoxia (Gleadhill, 1994; Bhatt and Patra, 2000). Hypoxia causes cellular damage by decreasing blood flow to the kidneys, which leads to depletion of tubular cells ATP reserve and dysfunction of the Na+, K+ - AT pase pump. This leads to cellular swelling and cells death (Kraje, 2002).

ARF, classically proceeds through three clinical phases, initiation, maintenance and recovery (Grauer, 1998). The initiation phase is the period during which animal is subjected to the renal insult (DiBartola, 2005). During this phase, therapy to decrease the renal insult can prevent established ARF (Kraje, 2002). The maintenance phase ensues after a critical amount of irreversible epithelial damage (DiBartola, 2005) characterised by tubular Lesions and established nephron dysfunction (Grauer, 1996). This phase may last 7 to 21 days (Labato, 2001) and most animal Will die (Kraje, 2002). The recovery phase is the periods where renal tissues undergoes regeneration and repair with restoration of renal function (DiBartola, 2005). During this phase resolution of azotemia, nephron repair and functional compensation occur (Lane et al., 1994).
Acute renal failure (ARF) may or may not be reversible depending on the degree of damage (Gleadhill, 1994). As long as basement membranes are intact and viable epithelial cells are present. Tubular injury can be repaired (Kraje, 2002).

The best time to intervene and stop the progression of ARF is the initiation phase where the initial injury can be minimized (Labato, 2001). Fluid therapy remains the mainstry of treatment for ARF. The goal of fluid therapy are to correct fluid and electrolyte imbalances, improve renal haemodynamics, increase tubular flow and initiate diuresis (Lane et al., 1994). When fluid therapy alone is in-effective to promote diuresis, routine use of diuretics and vasodilators has been advocated (Cowgill and Francy, 2005).

Mannitol and furosemide are being used by veterinary nephrologist over the past 10 years to initiate diuresis in oliguric/anuric patients. Furosemide frequently induces diuresis in patients who don’t respond to mannitol (Arthur, 1982).

Furosemide probably increases renal blood flow through activation of renal prostaglandin system, however, a diuresis often occurs without improvement of glomerular filtration rate (GFR). Although diuresis in general is thought to be beneficial. Low dose dopamine with minimal systemic
effects has been effective in some experimental model of ARF in dogs (Grauer, 1989).

Dopamine has a direct dilator effect on afferent arteriole in dogs. Recently, a combination of dopamine and furosemide is being considered to manage the ARF cases in a better way. It seems that dopamine and furosemide together have a synergistic effects. When conservative therapy fails to initiate diuresis, the prognosis is grave. Then Haemodialysis and peritoneal dialysis are the only option to manage the cases of ARF (Labato, 2001).

Haemodialysis and peritoneal dialysis have been used extensively in human medicine as a method of extra-renal means of excretion of metabolic waste products in the recent year but to a limited degree in the dogs. Peritoneal dialysis is simple procedure, equally effective and comparatively less expensive than haemodialysis which is expensive and requires trained technician and sophisticated instruments (Lane et al., 1994).

The main objective of peritoneal dialysis is to transfer of uremic solutes from blood into dialysate as a partial substitute for failed renal excretory function. Blood urea nitrogen, serum creatinine, phosphorous and many other uremic solutes can be removed by dialysis (Mahajan, 2000).
Peritoneal dialysis should not be regarded as cure for renal failure. It should be considered as temporary life saving measures which keeps the body chemistry in comparative balance until such time that the damaged kidney regain adequate function (Jackson, 1964).

The information available regarding management of renal failure are very limited and scanty in veterinary practice as per literature available in the present scenario. Hence the present investigation was undertaken with the following objectives.

1. To screen the clinical cases of renal failure.
2. To treat and manage the cases of renal failure with suitable therapy.
3. To compare the effects of different treatment in renal failure cases.
4. To adopt a suitable technique of peritoneal dialysis for the management of renal failure cases.
5. To study the haemato-biochemical changes pre and post treatment.
6. To assess the frequency of peritoneal dialysis.