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Review Article

Fluid Overload in Small Animal Patients

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Fluid therapy is used daily by veterinary practitioners and is an essential part of treatment of many veterinary patients. However, as with all interventions, there is the potential for negative side effects resulting from fluid therapy. Fluid overload is a key side effect that has been increasingly recognized in human medicine as leading to significant negative sequelae. Evidence related to fluid overload in veterinary medicine is sparse but it is likely that the same types of negative sequelae are seen in our veterinary patients. The goal of this review is to present a definition for fluid overload in small animal veterinary patients and ways to both recognize and treat fluid overload. Additionally, ways to avoid the development of fluid overload are described.

Fluid overload is a commonly mentioned negative side effect of intravenous (IV) fluid administration. There are multiple publications in human medicine citing the negatives of excessive fluid administration.[1-5] Fluid overload is extremely prevalent in human hospitals and is a source of much discussion in the human critical care field based on the scientific program at the 2016 Society of Critical Care Medicine Annual Congress. A recent publication showed that 56% of septic patients in the intensive care unit (ICU) had clinically relevant indications of fluid overload on day 1 with 100% of patients exhibiting fluid overload by day 3 of ICU hospitalization.[6] In humans, fluid overload is related to increased mortality in many situations including adult patients with acute kidney injury[3] and children with critical illness.[4] Fluid overload has also been shown to increase the duration of mechanical ventilation[3] and, in 1 recent study,[5] fluid overload increased hospitalization costs by a factor of approximately 1.5 and approximately doubled ICU costs as compared to a matched cohort of patients who did not develop fluid overload.

In veterinary medicine, any discussion of fluid therapy includes a mention of the ills of fluid overload, but further characterization of the negative effects is largely anecdotal. The goal of this article is to present a possible definition for fluid overload, ways to recognize fluid overload in clinical veterinary patients, a brief discussion of the reasons for fluid overload, and strategies for treatment and avoidance of this condition.

Definition and Recognition of Fluid Overload

There is no clearly published definition for fluid overload in human or veterinary medicine. At its most basic, fluid overload occurs in the nondehydrated patient when the fluid entering the patient exceeds the amount of fluid being lost from the body.[7] This can be seen when the nondehydrated patient's body weight increases by more than 10% from baseline (with the increase in weight being attributed to fluid).[7]

In a clinical setting, fluid overload tends to manifest as either edema or cavity effusions (e.g., pleural effusion and peritoneal effusion), or other types of fluid deposition in the body. Thus, both physical examination findings as well as diagnostic imaging can be used to define patients with fluid overload (Box 1). It is important to realize that heart rate and blood pressure have not been shown to change in dogs with experimentally induced fluid overload, negating the use of these findings.[8]

Predispositions for Fluid Overload

In small animal practice, several patient populations are predisposed to fluid overload including those with heart disease, kidney failure with decreased-to-absent urine production (i.e., oliguric or anuric renal failure), hypoalbuminemic patients, or those suffering from vasculitis such as septic patients or those with the systemic inflammatory response syndrome. Heart disease may be a known entity in the dog or cat before fluid therapy or it might be suspected based on the presence of a heart murmur or arrhythmia in dogs[9] or cats.[10] However, many dogs and cats have heart disease and do not display either of these clinical signs.[9,10] Animals with oliguric or anuric renal failure commonly suffer from fluid overload because they are unable to create urine and therefore they cannot remove fluid from the body. Hypoalbuminemia leads to edema formation since fluid more readily leaves the capillary and deposits into tissues because of decreased colloid oncotic pressure. Finally, patients with vasculitis run the risk of fluid overload secondary to increased leakage of fluid out of their blood vessels and deposition into tissues.

Causes of Fluid Overload

The negative effects of fluid overload as well as predispositions for overload such as hypoalbuminemia, oliguric or anuric renal

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Box 1–Clinical Findings in Fluid Overload

Physical examination

- Peripheral edema formation,[18] commonly located in
  - o Intermandibular region
  - o Limbs and paws, unrelated to the site of IV catheter placement or other distal limb wraps
  - o Dependent regions (i.e., the side on which the patient has been lying)
- Increased respiratory rate or effort or both (even mild changes can be significant)
- Increased body weight (> 10% from baseline nondehydrated body weight)
- Fluid drainage (typically serous or serosanguineous) from incisions (if applicable)[11]
- Fluid drainage (typically serous or serosanguineous) from endotracheal tube (if applicable)[11]

Thoracic radiographic findings[19]

- Pulmonary edema
  - o Dogs: perihilar region
  - o Cats: no specific localization
- Pleural effusion
- Chest wall edema

Ultrasonographic findings

- Pleural effusion
- Peritoneal effusion
- Fluid deposition in soft tissues of the body wall

failure and vasculitis are related to the classic Starling's law of the capillary. As a review, Starling's law describes the forces controlling fluid flux across the capillary wall (a semipermeable membrane).[11] In short, the following two forces control fluid movement across a capillary membrane: hydrostatic pressure and oncotic pressure. Hydrostatic pressure is created largely by the volume of fluid within the blood vessel and works to push fluid out of the vessel and into the surrounding interstitium. Therefore, when the overall body fluid volume is increased, as in patients with oliguric or anuric renal failure who are receiving IV fluids, hydrostatic pressure in the capillary is increased, pushing fluid out of the capillary.

Conversely, oncotic pressure is predominantly created by the proteins in the bloodstream (primarily albumin) and the sodium particles associated with the albumin. Fluid follows the albumin and sodium, thus remaining in the capillary. Therefore, oncotic pressure works to "hold" fluid within the capillary. Animals with hypoalbuminemia are less able to hold fluid within the vascular space. A third component dictating fluid movement across the capillary involves the capillary vessel membrane. When there is inflammation of the cell membrane (vasculitis), intravascular fluid moves more readily across the cell membrane into the interstitium regardless of the oncotic and hydrostatic pressures (Fig).

Newer research has also described the presence of the endothelial glycocalyx, a cell-free region made up of polysaccharides that lines the interior aspect of the blood vessels.[12,13] The glycocalyx is involved in many processes in the body, including, but not limited to, inflammation, coagulation, the ability of the endothelial cell to interact with and respond to its environment, and the movement of fluid and other substances across the capillary cell membrane.[12,13] The presence of the glycocalyx adds another layer of complexity to the concept of cell membrane

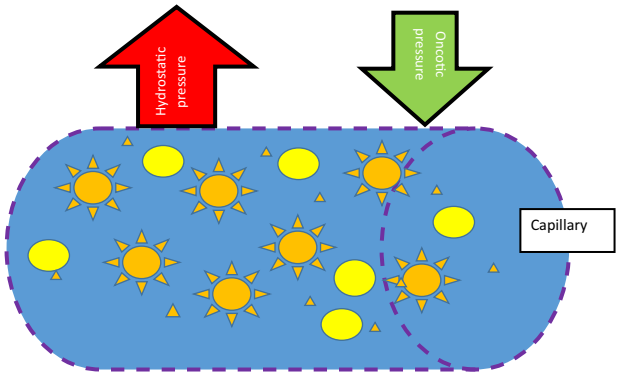

Fig. A review of the classic Starling's law of the capillary. Yellow and orange circles indicate albumin molecules. Triangles indicate sodium particles. The dotted purple line indicates the cell membrane of the capillary. Oncotic pressure derived from the albumin ± associated sodium particles works to hold fluid within the vascular space. Hydrostatic pressure largely determined by the fluid volume within the vascular space works to push fluid out of the vessel. The relative permeability of the cell membrane dictates how effectively fluid is held in the capillary vs. how easily the fluid moves out of the capillary. The net fluid movement (into or out of the vessel) is dependent upon the relative amount of oncotic pressure vs. hydrostatic pressure and is further regulated by the permeability of the capillary membrane. (Color version of figure is available online.)

permeability and fluid flux presented by Starling. Degradation of the glycocalyx alone and the increased membrane permeability resulting from this damage has been shown to increase the movement of fluid across the cell membrane by up to 60%.[12] In addition, there is some evidence that fluid therapy itself might lead to degradation of the glycocalyx, increasing vascular permeability and contributing to potential fluid overload.[13]

Sequelae of Fluid Overload

In a typical fluid overload situation, the overzealous administration of IV fluids is thought to increase hydrostatic pressure. As the hydrostatic pressure increases, the net fluid movement is out of the capillary and into the interstitium. In addition, degradation of the glycocalyx by aggressive fluid therapy may lead to increased fluid leakage out of the vessel.[13] Capillaries exist in all tissues so the end result of fluid overload is tissue interstitial edema anywhere in the body. Additionally, fluid can be pushed directly out of the blood vessels into the pleural and peritoneal cavity, creating effusions.

Typically on clinical examination, we can expect tissue edema in the limbs but it is important to remember that internal organ edema is also occurring. Organs with a capsule such as the liver and kidney would show effects of this edema sooner than other organs because the capsule would limit their ability to swell as fluid is deposited.[1] As organ edema worsens, pressure can increase within the organs. With enough pressure, decreased blood flow (and oxygen delivery) to cells of the organ occurs, leading to measurable organ dysfunction.[1] Clinically relevant effects can include decreased renal filtration (azotemia), decreased hepatic blood flow (liver enzyme elevations ± cholestasis), and decreased intestinal mobility (ileus, regurgitation or vomiting, and inappetence).

In some fluid overload situations, the increased size of the edematous intra-abdominal organs, in concert with free abdominal fluid, can also lead to increased intra-abdominal pressure that can further impede blood flow to organs.[13,14] At its most basic level, intra-abdominal hypertension results when the abdominal wall is less able to expand (i.e., after surgery or in obese patients) or when "extra things" are in the abdomen taking up space such as enlarged organs, fluid, or masses.[14]