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REVIEW ARTICLE

Clinical Approach and Treatment Strategies for Abdominal Compartment Syndrome in Cats and Dogs: A Review

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ABSTRACT

Abdominal compartment syndrome (ACS), which develops due to intra-abdominal hypertension, is a serious condition characterized by organ dysfunction and potential failure. In cats and dogs, this syndrome can occur due to various underlying causes such as trauma, gastrointestinal obstruction, or pancreatitis. The aim of this review article was to provide fundamental information on the pathogenesis, early diagnosis, and effective management of ACS in cats and dogs. Diagnosis of Abdominal compartment syndrome may involve a combination of clinical assessment, laboratory tests, and imaging techniques such as ultrasound. Treatment strategies for ACS generally focus on reducing intra-abdominal pressure and providing supportive care, while surgical intervention is preferred in severe cases. The prognosis depends on factors such as the type of underlying cause, the severity of organ dysfunction, and the importance of timely intervention. Ultimately, veterinary clinicians need to focus on gaining a better understanding of the pathophysiology of this syndrom in cats and dogs, and evaluating effective diagnostic and treatment approaches. This can lead to better outcomes for affected patients.

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INTRODUCTION

In veterinary medicine, abdominal compartment syndrome (ACS) in cats and dogs is a critical condition characterized by the impairment of abdominal organs function due to an increase in intra-abdominal pressure (IAP), leading to severe morbidity and mortality. Just as in human medicine, early diagnosis and management of ACS in veterinary practice can significantly improve chances of patient's survival (Conzemius *et al.*, 1995).

The basic pathophysiology involved in the abdominal compartment syndrome is the condition known as intra-abdominal hypertension (IAH) (Hoareau, 2014; Antoniou *et al.*, 2018). Various conditions such as acute abdominal trauma, peritonitis, excessive fluid overload, abdominal tumors and surgical complications can lead to this problem. Additionally, systemic diseases such as sepsis, pancreatitis, and intestinal perforation may also increase the risk of IAH and ACS (Bains *et al.*, 2019; Gardner and Schroeder, 2022). Intra-abdominal hypertension has systemic consequences such as hemodynamic instability, visceral organ hypoperfusion, and decreased pulmonary compliance (Fetner and Prittie, 2012). In cats, IAH can occur due to any of the factors summarized by the '8F' classification (Fluid, Fat, Food, Feces, Flatus, Fetus, Formidable organomegaly, and Feeble abdominal musculature) or a combination of these factors (Abrams-Ogg, 2006).

The process leading to the development of ACS can originate from an intra-abdominal, extra-abdominal or systemic problem (Abrams-Ogg, 2006). If the triggering cause involves a problem within the abdominal cavity, it is called Primary ACS; if it is extra-peritoneal, it is referred to as Secondary ACS. Primary ACS results from damage to structures present within the abdominal and pelvic cavities (such as gastric perforation, hemoperitoneum, uroabdomen, pancreatitis, hepatitis, gastric dilation-volvulus, peritonitis, rupture of the urinary bladder or urethra). Secondary ACS occurs as a result of conditions that do not originate from within the abdomen or pelvis, including fluid resuscitation, severe trauma, severe burns, pneumonia, and sepsis (Rader and Johnson, 2010; Nielsen and Whelan, 2012).

The pathophysiology of ACS is based on the mechanical effects of increased intra-abdominal pressure on the organs and the impairment of their blood supply. Increased pressure leads to compression of abdominal organs, reduced venous return, decreased cardiac output,

and impaired systemic organ perfusion. If not diagnosed and treated promptly, ACS can lead to organ dysfunction and even death (Sanches da Silva *et al.*, 2021; Gardner and Schroeder, 2022). In patients with ACS, signs such as severe abdominal pain, body swelling, respiratory distress, increased heart rate, and decreased urine output are commonly observed (Sanches da Silva *et al.*, 2021).

In cats and dogs, ACS is one of the critical conditions frequently encountered in veterinary emergency services that requires rapid intervention. Therefore, knowledge and awareness about this problem in the field of veterinary medicine are gradually increasing, helping veterinarians manage their patients more effectively (Mazzaferro, 2003; Fecteau *et al.*, 2018). However, this syndrome has not yet been thoroughly researched and fully understood in the veterinary literature (Joubert *et al.*, 2007; Smith and Sande, 2012).

Veterinarians should consider ACS after trauma or some severe intra-abdominal disease. Early diagnosis and treatment are important because delayed intervention can lead to organ damage and death. Treatment generally involves measures to reduce intra-abdominal pressure and requires close clinical monitoring (Fecteau *et al.*, 2018; Bains *et al.*, 2019; Csiszkó *et al.*, 2019).

This article aimed to compile the current knowledge and latest developments regarding ACS in dogs and cats. In this review, the pathogenetic mechanisms, diagnostic approaches, treatment strategies, prognosis and future directions of ACS have been discussed. Information regarding the impact of this problem on veterinary practice is expected to enable veterinarians to better understand ACS and develop effective diagnostic and treatment strategies against this problem in cats and dogs.

Etiology and Risk Factors: Abdominal compartment syndrome can occur in cats and dogs for a variety of reasons. These etiological factors and risks may contribute to the development of ACS and should be considered in the clinical management of patients (Table 1). Early diagnosis and appropriate treatment approaches, i.e. risk management of ACS, are critical to prevent its complications (Papavramidis *et al.*, 2011).

Pathophysiology: In veterinary medicine, knowledge and understanding of the pathophysiology of ACS can help clinicians in diagnosing this problem and implement appropriate interventions. This problem has been associated with various pathogenic mechanisms summarized in Table 2. The pathophysiology of ACS is related to the adverse effects of increased IAP on the abdominal organs and systemic circulation. Increased IAP leads to the formation of IAH. Prolonged continuation of IAH may result in ACS (Devey, 2013).

Developmental Mechanisms of Abdominal Compartment Syndrome

Mechanical compression of abdominal organs: This compression exerts pressure on abdominal organs, specifically on the intestines, liver, kidneys and diaphragm. Decreased blood flow to the intestines can lead to ischemic damage and disruption of the intestinal mucosal barrier function. Reduced blood flow to the liver can impair hepatic function and hinder the elimination of toxins from the body. Compression of the kidneys may lead to decreased renal perfusion and acute injury to the organ. The upward displacement of the diaphragm can disrupt respiratory mechanics, leading to ventilation-perfusion mismatch and hypoxemia (Bailey and Shapiro, 2000).

Decreased venous return: Increased IAP raises pressure on the inferior vena cava, thereby reducing venous return. A decrease in venous return reduces cardiac preload and leads to decreased cardiac output. This condition can lead to impaired systemic perfusion and organ failure (Abrams-Ogg, 2006).

Systemic inflammatory response: Abdominal compartment syndrome can trigger systemic inflammatory response syndrome (SIRS), which can lead to widespread inflammation in the body, increasing the risk of sepsis, multiple organ failure and death. Increased IAP can also impair microvascular circulation and lead to ischemic damage at the cellular level. Impaired microcirculation prevents oxygen and nutrients from reaching cells, leading to cell death and organ damage (Zarnescu *et al.*, 2023).

Impaired microcirculation: Impaired microcirculation also plays a crucial role in the pathophysiology of ACS. Increased IAP can lead to impaired microvascular circulation and ischemic damage at the cellular level. Impaired microcirculation prevents oxygen and nutrients from reaching cells, leading to cell death and organ damage (Csiszkó *et al.*, 2019).

Effects of Abdominal Compartment Syndrome: Abdominal compartment syndrome can significantly influence the clinical condition of an animal, which can be manifested by various symptoms (Table 3). This condition can be a primary cause or trigger of acute abdomen in cats and dogs. The significant effects of ACS are summarized as follows:

Respiratory system effects: In animals with ACS, increased intra-abdominal pressure pushes the diaphragm upward into the thoracic cavity, raising intrathoracic pressure. Due to the bidirectional compression, the movement of the diaphragm is restricted. The increase in intrathoracic pressure leads to extrinsic compression of the pulmonary parenchyma and can cause pulmonary dysfunction. Additionally, decreased chest wall compliance and reduced spontaneous tidal volume in these patients may result in arterial hypoxia and hypercapnia. Patients with IAH are also predisposed to pulmonary infections (Cheatham, 2009; Nielsen and Whelan, 2012).

Cardiovascular effects: High intra-abdominal pressure can reduce venous return and decrease cardiac output. This condition can contribute to the development of hypotension and shock, adversely affecting the animal's hemodynamic stability (Conzemius *et al.*, 1995; Nielsen and Whelan, 2012).

The IAH leads to cardiac compression due to the upward movement of the diaphragm into the thoracic cavity. This results in decreased ventricular compliance and reduced myocardial contractility (Conzemius *et al.*,

Table 1: Etiology and risk factors in Abdominal Compartment Syndrome.

Risk factors	Explanation	References
Surgical procedures	- Increased intra-abdominal pressure during prolonged or complicated surgical operations - Development of bleeding or infection in the abdominal cavity following laparotomy - Postoperative edema - Repair of diaphragmatic hernias - Repair of large defects in the abdominal wall (evisceration, abdominal hernia)	Formaggini *et al.* (2008) Cheatham (2009) Papavramidis *et al.* (2011) Przywara *et al.* (2014) Frikis and Zlateva (2018)
Trauma	- Serious abdominal trauma resulting from traffic accidents or falls - Penetrating injuries or damage to intra-abdominal organs - Pneumoperitomeum, hemoperitoneum, hemoretroperitoneum	Dörfelt *et al.* (2012) Fransson *et al.* (2015) Marwood *et al.* (2022)
Inflammation	- Peritonitis - Gastroenteritis - Cholangiohepatitis	Nielsen and Whelan (2012)
Infection	- Bacterial - Viral - Protozoal	Zygner *et al.* (2021)
Massive fluid resuscitation	- Edema development in abdominal organs and tissues in patients receiving intensive fluid therapy - Fluid accumulation in the abdomen after blood or plasma transfusion - Peritoneal dialysis - Uroabdomen	Papavramidis *et al.* (2011) Hall and Drobatz (2021_ Parlak *et al.* (2021) Marvel (2022)
Intraabdominal pathological conditions	- Intraabdominal and retroperitoneal tumors or cysts - Enlargement of intra-abdominal organs (organomegaly) - GDV - Ileus	Abrams-Ogg (2006) Leary and Sinnott-Stutzman (2018) Uemura and Tanaka (2018)
Obesity	- Excess body fat can increase intra-abdominal pressure - Obese animals are at higher risk of developing abdominal compartment syndrome	Bailey and Shapiro (2000) Turner *et al.* (2020)
Burns	- Burns covering a large surface area and edema developing due to these burns - Scar tissue after burns creates pressure on the abdominal wall	Joubert *et al.* (2007) Vaughn *et al.* (2012)
Metabolic and systemic diseases	- Conditions such as severe pancreatitis, liver failure or sepsis - Systemic inflammatory response syndrome (SIRS) - Development of multiple organ dysfunction syndrome (MODS)	Cioffi *et al.* (2012) Hoareau (2014) Zarnescu *et al.* (2023)
Pregnancy	- Increased IAP, especially in the late stages of pregnancy	Abrams-Ogg (2006) Chalifoux *et al.* (2022)

Table 2: Pathological mechanisms of Abdominal Compartment Syndrome.

Pathological Mechanism	Causes	Effects	References
Mechanical compression of abdominal organs	Increased IAP (GDV), mechanical compression of organs in the abdomen	Intestinal ischemia, bacterial translocation and sepsis, liver dysfunction, kidney failure, respiratory mechanics deterioration	Bailey and Shapiro (2000)
Decreased venous return	Increased IAP, increased pressure on the inferior vena cava	Decreased venous return, decreased cardiac preload, decreased cardiac output, impaired systemic perfusion	Abrams-Ogg (2006) Antoniou *et al.* (2018)
Systemic inflammatory response	ACS triggers systemic inflammatory response syndrome (SIRS)	Widespread inflammation, sepsis, multiple organ failure, increased risk of death	Hoareau (2014) Zarnescu *et al.* (2023)
Disruption of microcirculation	Increased IAP, impaired microvascular circulation	Ischemic damage at the cellular level, failure of oxygen and nutrients to reach cells, cell death and organ damage	Csiszkó *et al.* (2019) Zarnescu *et al.* (2023)

1995). Moreover, IAH functionally impairs blood flow in the inferior vena cava. This results in reduced venous blood flow in the lower extremities, increased peripheral edema, and a higher risk of deep vein thrombosis (Conzemius *et al.*, 1995).

Gastrointestinal effects: The intestines are the organs most sensitive to increase in intra-abdominal pressure. Intra-abdominal hypertension compresses the mesenteric vessels, leading to edema in the intestines, which further increases intra-abdominal pressure. This vicious cycle leads to further worsening circulatory insufficiency, intestinal ischemia, decreased mucosal pH, and lactic acidosis. As a result, insufficient blood flow in the intestines can lead to disruption of the mucosal barrier and subsequent migration of bacteria, sepsis, and multiple organ failure. Symptoms such as vomiting, diarrhea, and gastrointestinal motility are the main signs of ACS. These effects can negatively affect the nutritional status and digestive system of the affected animal.

Hepato-biliary effects: Increased intra-abdominal pressure in animals with ACS may adversely affect liver function. This may lead to increased hepatic enzyme activities, signs of liver dysfunction or hepatic insufficiency. Hepatic artery, hepatic vein and portal vein blood flow decreases due to extrinsic compression. As a result, liver failure occurs in advanced cases of reperfusion injury (Monnet, 2003; Antoniou *et al.*, 2018).

Hematological effects: Hematological disorders such as coagulopathy and thrombocytopenia may occur as a consequence of ACS and increase the patient's risk of bleeding. It may also contribute to the development of intravascular coagulation as a serious complication, which may further complicate the clinical course of ACS (Papavramidis *et al.*, 2011; Padar *et al.*, 2019).

Renal system effects: In ACS, renal functions are at high risk because the increased intra-abdominal pressure and the resulting decrease in cardiac output directly affects

Table 3: Effects of Abdominal Compartment Syndrome on various body ststems and physiological functions.

System	Effects	References
Respiratory system	- Restriction of movement of the diaphragm	Cheatham (2009)
	- Pulmonary dysfunction	Łagosz *et al.* (2022)
	- Decreased chest wall compliance	
	- Increased risk of pulmonary infection	
Cardiovascular system	- Decreased venous return and cardiac output	Gardner and Schroeder (2022)
	- Hypotension and shock	
	- Cardiac compression	
	- Peripheral edema and increased risk of deep vein thrombosis	
Gastrointestinal system	- Intraabdominal hypertension and intestinal edema	Nielsen and Whelan (2012)
	- Hypoperfusion and intestinal ischemia	
	- Mucosal barrier disruption and sepsis	
	- Vomiting, diarrhea, motility disorders	
	- Acute pancreatitis	
Hepatobiliary function	- Liver dysfunction	Antoniou *et al.* (2018)
	- Increased hepatic enzyme levels	
	- Decreased hepatic artery, hepatic vein, and portal vein blood flow	
Hematological activity	- Coagulopathy and thrombocytopenia	Csiszkó *et al.* (2019)
	- Increased risk of bleeding	
Renal system	- Renal dysfunction	Papavramidis *et al.* (2011)
	- Increased plasma renin activity, aldosterone and antidiuretic hormone	Zarnescu *et al.* (2023)
	- Decreased urine output	
Neurological function	- Increased intracranial pressure	Nielsen and Whelan (2012)
	- Cerebral ischemia and brain damage	
	- Restlessness, lethargy and confusion	
Ophthalmological function	- Increase in IOB	Jang *et al.* (2021)
Abdominal wall	- Decreased blood flow at the abdominal wall	Cheatham (2009)
	- Edema	Papavramidis *et al.* (2011)

renal blood flow. However, reduced renal blood flow is not the only cause of kidney dysfunction. Intra-abdominal pressure is also transmitted to the renal outflow tract. As pressure increases within the tubular system, it is inevitably transmited back to the glomeruli, reducing the filtration gradient. This results in a progressive decrease in urine output. Additionally, plasma renin activity, aldosterone, and antidiuretic hormone concentrations increase to levels higher than twice their baseline values (Papavramidis *et al.*, 2011; Zarnescu *et al.*, 2023).

Neurological effects: Intra-abdominal hypertension also increases intrathoracic pressure, inhibits venous return, and consequently leads to an increase in intracranial pressure. Short-term increases in intra-abdominal pressure, such as those caused by coughing, defecation or vomiting, result in a sudden rise in intracranial pressure. As a result, there can be a critical decrease in cerebral perfusion, leading to progressive cerebral ischemia and brain damage. Restlessness, lethargy, confusion, or other neurological signs may develop, particularly as a result of hypoperfusion and metabolic disturbances. The ACS can also have severe adverse effect on neurological functions (Nielsen and Whelan, 2012).

Ophthalmological effects: Sudden IAH, which occurs due to excessive volumes of gas insufflation into the abdominal cavity to expand the abdomen during laparoscopic surgery, can also cause an increase in intraocular pressure (IOP). The increase in IOP, like the increase in IOP caused by other factors, can predispose to glaucoma, optic nerve damage, and other eye problems (Jang *et al.*, 2021).

Abdominal wall effects: In ACS, the pressure on the abdominal wall may be increased due to the effect of high IAP, and this may be manifested by symptoms such as abdominal distension, stiffness or stretching of the abdominal wall (Cheatham, 2009). Intra-abdominal conditions such as visceral edema, abdominal masses and free intraperitoneal fluid stretch the abdomen and reduce abdominal wall compliance. Additionally, obesity, laparotomies that lead to excessive tension, large defect eviscerations, hernia repairs, edema in the abdominal wall, and compression from scars after extensive burns can also result in increased IAP. The IAH can also reduce abdominal wall blood flow directly through compression, affecting all the muscles that make up the abdominal wall. Secondary to shock and fluid resuscitation, abdominal wall edema may develop, leading to reduced abdominal wall compliance, which in turn exacerbates IAH (Papavramidis *et al.*, 2011; Łagosz *et al.*, 2022).

Diagnostic Approach to Abdominal Compartment Syndrome: Physical examination and evaluation of clinical findings play an important role in the diagnosis of ACS in cats and dogs (Łagosz *et al.*, 2022). Imaging techniques such as radiography, ultrasonography, Computed Tomography (CT), and Magnetic Resonance Imaging (MRI) confirm clinical findings and detect structural changes caused by increased intra-abdominal pressure (Shanaman *et al.*, 2013). However, intra-abdominal pressure measurement is essential for accurate diagnosis and treatment monitoring (Csiszkó *et al.*, 2019). Veterinarians should use a combination of these techniques to diagnose ACS and determine appropriate treatment strategies.

Clinical and physical examination: Clinical and physical examination findings in the diagnosis of ACS in cats and dogs can guide veterinarians in the early diagnosis and treatment of the problem. By adopting a multidisciplinary approach, clinical findings should be evaluated together with laboratory analyses and imaging results. In this way, the correct diagnosis of ACS can be made and appropriate treatment strategies can be adopted.

Clinical signs of abdominal compartment syndrome usually include the animal's overall condition, effects on respiratory and circulatory functions, and symptoms arising from increased intra-abdominal pressure. Clinical findings mostly include abdominal distension, abdominal pain, respiratory distress, tachycardia, hypotension, and oliguria. During physical examination, findings such as firmness, tenderness, and abdominal distension can be assessed through abdominal palpation (Nielsen and Whelan, 2012; Gardner and Schroeder, 2022). These clinical findings of ACS result from multisystemic effects that develop due to the increased intra-abdominal pressure.

The clinical signs of ACS can vary widely. Common symptoms include abdominal pain and distension with bloating, pain and discomfort in the abdominal area, and increased tenderness on palpation. Respiratory distress and hyperventilation may occur due to the upward displacement of the diaphragm, potentially leading to insufficient oxygenation as a result of lung compression. Hypotension can also occur due to increased intra-abdominal pressure, which may reduce venous return and cardiac output. Additionally, oliguria or anuria, characterized by decreased or absence of urine output, may develop as a consequence of decreased kidney perfusion. Tachycardia, an elevated heart rate, may be observed as a compensatory response to reduced perfusion. In some cases, changes in consciousness, such as confusion or lethargy, can arise due to diminished cerebral perfusion (Nielsen and Whelan, 2012; Smith and Sande, 2012).

Physical examination provides critical information in the assessment of abdominal compartment syndrome (ACS). One important finding is a noticeable increase in abdominal girth measurement, which may indicate a rise in intra-abdominal pressure; regular monitoring of these values is essential. Palpation of the abdominal area helps assess tenderness, hardness, and the presence of a mass, confirming abdominal distension. Abdominocentesis, a procedure involving percutaneous aspiration, allows evaluation of intra-abdominal fluid accumulation. Additionally, the upward displacement of the diaphragm may cause a decrease in respiratory movements and asymmetric breathing. Finally, the assessment of skin and mucous membranes can reveal pallor or cyanosis, which may indicate peripheral perfusion disturbances.

The findings of physical examination may be evaluated keeping in view findings of other diagnostic methods to confirm the diagnosis of ACS. These methods include imaging techniques (radiography, ultrasonography, CT) and intra-abdominal pressure measurements. Monitoring urine output is important for the evaluation of renal function (Smith and Sande, 2012).

Laboratory analysis: In the diagnosis of ACS in cats and dogs, laboratory analyses play a critical role in determining the severity of the disease and condition of the affected organs. Laboratory tests such as complete blood count, serum biochemistry, coagulation profile, lactate levels, arterial blood gas analysis, urine analysis and cardiac enzymes may be evaluated together with clinical findings and addressed with a multidisciplinary approach. These analyses are indispensable for the accurate diagnosis of ACS and the determination of efficacy of treatment strategies (Zacher *et al.*, 2010; Devey, 2013; Csiszkó *et al.*, 2019; Jang *et al.*, 2019).

Complete blood count (CBC): The complete blood count provides important clues for the diagnosis of ACS. Increased hematocrit (HCT) and hemoglobin (Hb) levels may indicate dehydration or hypovolemia. Changes in leukocyte count help identify conditions such as infection or sepsis. Leukocytosis may indicate the body response to infection, while leukopenia may suggest immunosuppression.

Serum biochemistry: Electrolyte imbalances are prominent laboratory findings of ACS. Electrolyte imbalances such as hyperkalemia (high potassium), hyponatremia (low sodium), and hypochloremia (low chloride) provide information about fluid management and renal function. Increased levels of creatinine and blood urea nitrogen (BUN) may indicate impaired renal function. Additionally, increases in liver enzymes such as ALT, AST, and ALP may indicate liver stress or damage.

Coagulation profile: Changes in coagulation parameters may be observed in animals with ACS. Changes in coagulation profile parameters such as prothrombin time (PT), activated partial thromboplastin time (aPTT), and fibrinogen levels may indicate the presence of disseminated intravascular coagulopathy (DIC) or other types of coagulopathy. These changes may help determine the severity of the disease and the patient's prognosis.

Lactate levels: Increased blood lactate levels indicate tissue hypoperfusion and hypoxia. High lactate levels may also indicate inadequate organ perfusion and tissue oxygenation problems. This is an important parameter in determining the severity and urgency of the problem.

Arterial blood gas analysis: Arterial blood gas analysis is used to assess the presence of acidosis or alkalosis. Metabolic or respiratory disorders, oxygenation status, and carbon dioxide levels can be determined by this analysis. Metabolic acidosis is common in ACS and may be due to lactate acidosis.

Urine analysis: Urine analysis is used to evaluate conditions such as kidney function and urinary tract infection. Parameters such as density, pH, presence of proteins, glucose and ketone in the urine are examined. In this way, the degree of severity of impaired kidney function can be detected.

Cardiac enzymes: Troponin levels are an important biochemical marker used to assess cardiac stress or damage. Given that cardiac function may also be affected in ACS, monitoring of troponin levels is important.

Imaging techniques: In addition to physical examination and clinical findings, imaging techniques also play an important role in the diagnosis of ACS. Imaging techniques play a critical role in the diagnosis of abdominal compartment syndrome and in determining the causes of the disease. Ultrasonography can be used to

visualize intra-abdominal organs and detect free fluids. Computed tomography (CT) can further evaluate anatomical changes caused by intra-abdominal pressure and detect complications (Zarnescu *et al.*, 2023). A combination of these diagnostic approaches may help accurately evaluate abdominal compartment syndrome and determine appropriate treatment strategies.

Chest X-ray, abdominal ultrasound and computed tomography scan are not specific for the diagnosis of IAH/ACS but may provide important clues about changes such as elevation of the diaphragm, basal atelectasis, inferior vena cava compression, retroperitoneal infiltration causing peritoneal disorders, massive abdominal distension, direct renal compression or displacement or bilateral inguinal herniation (Bains *et al.*, 2019).

Radiography: Direct and indirect radiography is one of the first-line imaging methods used to evaluate the general condition of the organs in the abdominal cavity. In ACS, findings such as expansion of the intestines and upward displacement of the diaphragm due to increased intra-abdominal pressure can be seen on radiography. However, radiography has low specificity in the diagnosis of ACS and is usually used with other imaging methods (Bischoff, 2003; Mavromatis *et al.*, 2018; Corrick, 2023).

Ultrasonography: Ultrasonography is a non-invasive method frequently used in the diagnosis of ACS. Findings such as intra-abdominal fluid accumulation, organ dislocation, and decreased intestinal peristalsis can be detected by ultrasonography. Ultrasonography is also useful in evaluating vascular compression and organ perfusion disorders caused by increased intra-abdominal pressure (Shanaman *et al.*, 2013; Corrick, 2023). Ultrasonography (point-of-care ultrasound or POCUS) provides more reliable information in determining the stomach contents (liquid vs. solid) compared to abdominal X-ray (Bains *et al.*, 2019).

Computed tomography (CT): Computed tomography (CT) provides more detailed, three-dimensional imaging of the abdominal cavity. It can be used to evaluate organ dislocation, bowel dilatation, and peritoneal fluid accumulation due to increased intra-abdominal pressure. This technique can also better visualize vascular compression and organ perfusion disorders caused by ACS. However, CT has disadvantages such as high cost and radiation exposure (Shanaman *et al.*, 2013; Zarnescu *et al.*, 2023).

Intraabdominal pressure measurement: Intraabdominal pressure (IAB) measurement is important in the diagnosis and follow-up of abdominal compartment syndrome. The IAB measurements allow the determination of the patient's intra-abdominal pressure and monitoring of the response to treatment. Intra-abdominal pressure can be measured using invasive and non-invasive techniques. Invasive methods include direct measurements such as intragastric or intrarectal catheterization, while non-invasive methods include indirect measurements such as intrabladder and intraperitoneal (Chopra *et al.*, 2015). These measurements should be selected considering the patient's clinical condition and available equipment. Intra-abdominal pressure measurements can provide valuable information in making decisions about the treatment process and determining prognosis (Fetner and Prittie, 2012).

The wide variety of species and breeds of veterinary patients hinders the establishment of reference data. Despite this, there are studies reporting IAP data in farm animals such as cattle (Fecteau *et al.*, 2018), horses (Munsterman and Hanson, 2009; Munsterman and Hanson, 2011), and pigs (Schachtrupp *et al.*, 2006; Wauters *et al.*, 2012; Antoniou *et al.*, 2018). In addition, there are experimental studies conducted in rat (Vincent *et al.*, 2023) and rabbit (Mahjoub *et al.*, 2012; Yoshino *et al.*, 2012) models; IAH and thus ACS are induced in experimental models by administering different volumes of liquid or gas to the intraperitoneal cavity. Information including IAP values in mice, rats, rabbits, sheep, pigs and dogs has also been reported in various review articles (Csiszkó *et al.*, 2019; O'Neill *et al.*, 2022).

In humans, IAP values have been standardized by taking the information established by the World Society of the Abdominal Compartment Syndrome (WSACS) in 2004 as reference, and accordingly, these values are taken into account in the clinical management of patients with ACS, including intensive care units (Cheatham, 2009; Luckianow *et al.*, 2012; Hoareau, 2014; Bains *et al.*, 2019; Padar *et al.*, 2019; Łagosz *et al.*, 2022). It is noteworthy that the WSACS criteria are also used as a reference in veterinary field research (Rader and Johnson, 2010; Fetner and Prittie, 2012; Mahjoub *et al.*, 2012; Nielsen and Whelan, 2012; Smith and Sande, 2012; Chalifoux *et al.*, 2022; Gardner and Schroeder, 2022).

According to the WSACS criteria, conditions in which the IAP is above 12 mmHg in humans are considered as ACS. Considering the diagnostic and therapeutic criteria, IAP values are graded into four categories. Accordingly, grades III and IV are considered fatal if left untreated (Bains *et al.*, 2019). While medical intervention is appropriate in any of these degrees, grade IV requires surgical decompression (Cheatham, 2009).

According to WSACS criteria, four grades of ACS are as follows (1 mmHg = 1.36 cmH_2O): Grade I: IAP 12-15 mmHg (16-20 cmH_2O); Grade II: IAP 16-20 mmHg (21-27 cmH_2O); Grade III: IAP 28-34 mmHg (16-20 cmH_2O) and Grade IV: IAP >25 mmHg (>34 cmH_2O).

Unfortunately, IAP pressure values have not been sufficiently standardized in the veterinary field. The variety of species and breeds of animals naturally results in data that show a wide range of distribution. It has been determined that even standing animals and those in lateral, sternal or supine positions can give different results (Rader and Johnson, 2010). The following rating has been suggested for cats and dogs (Smith and Sande, 2012) (conversion from cmH_2O to mmHg can be obtained by dividing by 1.36). Normal: 5-10 cmH_2O; Mild IAH: 10-20 cmH_2O; Moderate-Severe IAH: 20-35 cmH_2O and Severe IAH: >35 cmH_2O.

Many methods have been described for the measurement of IAP. The bladder, stomach, uterus, rectum, and intraperitoneal cavity are potential sites for IAP measurement. However, the transvesical technique is considered the gold standard in human medicine due to its limited invasiveness, reproducibility, and ease of access (Bains *et al.*, 2019; Fetner and Prittie, 2012). Intra-

abdominal pressure can be measured using "direct" and "indirect" techniques (Łagosz *et al.*, 2022).

Invasive (direct) techniques: Direct measurement of IAP is used as a reference for indirect methods. However, due to the risks to the patient, possible technical problems and cost, indirect methods, especially the transvesicular method, have become the gold standard for the diagnosis of IAH (Chopra *et al.*, 2015).

In direct (intraperitoneal/intra-abdominal) method, a catheter and an intraperitoneal pressure probe are inserted into the abdominal cavity (Yoshino *et al.*, 2012; Lopes *et al.*, 2016; Vincent *et al.*, 2023). The IAP is commonly measured via an intraperitoneal catheter during laparoscopic surgery (Bailey and Shapiro, 2000). Direct diagnosis of IAH is achieved by inserting a needle connected to a water manometer or a direct pressure monitoring transducer into the abdominal cavity under blind or ultrasound guidance. This invasive technique poses a risk of visceral perforation and peritonitis, especially in cases of massive gastrointestinal intraluminal distension. In addition, this technique may also yield erroneous values, since the procedure must be repeated at certain intervals during the patient's monitoring in the intensive care unit (Chopra *et al.*, 2015; Munguia and Thomovsky, 2024).\

Non-invasive (indirect) techniques: The use of indirect methods of IAP measurement is quite common, as they are less invasive, more cost-effective and easier to use. Some of these IAP measurement methods are described below.

Intra-bladder (intravesical) measurement: Since bladder pressure measurements reflect the general intra-abdominal pressure, IAP is commonly measured in cats and dogs via a catheter placed in the urinary bladder (Bailey and Shapiro, 2000). Measurements can be made with the animal in the lateral or sternal recumbency, but since IAP is affected by body position, repeated measurements are made with the animal in the same position (Lopes *et al.*, 2016; Kim *et al.*, 2022). It has also been shown that the measured value is affected by the volume of physiological serum administered to the bladder, body condition, pregnancy, external pressure applied to the abdomen, and the presence of abdominal wall or detrusor muscle contractions. As a standard, the reading is taken at the end of expiration. The frequency of IAP monitoring can be adjusted according to the patient's risk status and needs to be evaluated every four hours in critically ill patients (Luckianow *et al.*, 2012; Padar *et al.*, 2019).

First, a Foley urinary catheter is aseptically placed with the catheter tip just inside the trigone of the bladder. The Foley catheter is connected to a sterile urine collection system with two three-way stopcocks included in the collection system. A water manometer is attached to the first valve. A syringe is attached to the second valve to fill the manometer and infuse the bladder. The patient is placed in the lateral or sternal position, the bladder is emptied with the catheter and 1.0 mL/kg (maximum 25 mL/patient) of sterile saline is injected into the bladder. The manometer is zeroed at the patient's symphysis pubis midline and filled with sterile saline. The valve to the fluid source is closed to ensure that the manometer equalizes the pressure in the bladder. The measurement is made at the end of expiration. The difference between the equilibration point and the zero reference point reflects the IAP measurement value (Rader and Johnson, 2010; Smith and Sande, 2012; Way and Monnet, 2014).

Intragastric measurement: Another indirect method of measuring IAP is through an established nasogastric tube. However, intragastric use is not preferred because it may cause various complications and may be technically errors. (De Waele *et al.*, 2007; Gardner and Schroeder, 2022).

Intrarectal measurement: In this method, IAP measurement is performed with an apparatus placed in the rectum. It is performed using an open rectal catheter with continuous slow irrigation or a closed system using fluid-filled balloon catheters. It can cause rectal and anal sphincter lacerations and bleeding in the lower gastrointestinal tract, so its use is limited (Schachtrupp *et al.*, 2006; Fetner and Prittie, 2012; Smith and Sande, 2012).

Intrauterine measurement: Similar to rectal measurement, IAP is measured by placing an apparatus into the uterus via the vaginal route. However, bleeding and infection risks limit its use (Fetner and Prittie, 2012).

Airway pressure measurement: In this technique, a catheter that measures airway pressure is placed at the junction of the tracheal tube. Respiratory flow is measured with a pneumotachograph. All pressure and flow signals are recorded with a multi-channel recording system (Jacques *et al.*, 2011).

Measurement via inferior vena cava: In this method, measurement of IAP is performed by inserting a catheter into the inferior vena cava via the femoral vein. Although this monitoring technique provides continuous real-time data, there are more risks associated with this method, such as thrombosis, venous thromboembolism, venous or arterial laceration, femoral nerve injury, hematoma formation, pseudoaneurysm formation, and central line-related bloodstream infection (Luckianow *et al.*, 2012).

Treatment Strategies: Effective management of abdominal compartment syndrome requires a multidisciplinary approach that includes prompt intervention (Table 4). Effective management of ACS relies on early diagnosis in at-risk patients, severity of ACS, and optimal timing of medical therapy or abdominal decompression.

Prevention: The most effective way to prevent this disorder is through preventive interventions to minimize the chance of developing IAH in patients at risk. First of all, nonsurgical disorders that may cause deviations in the patient's physiology during laparotomy (hypothermia, acidosis, coagulopathy) should be considered and managed. In addition, optimal resuscitation and intensive care management of the patient and maintaining lactate, base deficit and gastric mucosal pH in balance are important. In addition, the use of absorbable mesh in high-

Table 4: Treatment Strategies for Management of ACS.

Strategy	Definition	Purpose of Use	Target	Explanation	References
Abdominal surgical decompression	Surgical intervention to reduce abdominal pressure	Emergency situations, in cases of refractory ACS	To control intraabdominal pressure	Used to reduce intra-abdominal pressure with surgical methods and to manage compartment syndrome.	Luckianow *et al.* (2012) Nielsen and Whelan (2012) Łagosz *et al.* (2022)
Fluid-electrolyte management and diuresis	Fluid therapy to increase urine output and reduce intra-abdominal pressure - Crystalloid and colloid solutions, diuretics may be used	In ACS cases with hemorrhagic shock and multiple organ failure	Improving fluid balance in the body	Important for maintaining fluid, electrolyte and acid-base balance	Luckianow *et al.* (2012) Bains *et al.* (2019)
Positive pressure ventilation (PPV)	Ventilation strategy to reduce intra-abdominal pressure	In ACS cases with acute respiratory failure	Optimizing respiratory functions	Aims to treat respiratory failure by reducing intra-abdominal pressure with ventilation settings	Csiszkó *et al.* (2019); Padar *et al.* (2019)
Pharmacological treatments	Drugs used to reduce intra-abdominal pressure (e.g. diuretics, vasodilators)	In cases of refractory ACS or as a supportive treatment after surgery	Providing hemodynamic stabilization with pharmacological adjustments	Treated to reduce intra-abdominal pressure and provide hemodynamic stabilization using pharmacological agents	Luckianow *et al.* (2012)
Nutritional support	Providing nutritional support by enteral or parenteral route	In cases of ACS requiring long-term treatment or to improve the nutritional status of seriously ill patients	Providing nutritional support and supporting the patient's recovery process	Enteral or parenteral nutritional support aims to optimize the patient's nutritional status and support the recovery process	Padar *et al.* (2019)

risk patients during laparotomy closure can reduce abdominal tension and thus prevent ACS due to IAH (Bailey and Shapiro, 2000).

Emergency intervention (Abdominal decompression): In cases where abdominal compartment syndrome is suspected, abdominal decompression may be required urgently to reduce intra-abdominal pressure. For this purpose, procedures such as peritoneal lavage or surgical fasciotomy can be applied to drain gas and fluid accumulated in the abdomen. This emergency intervention can help preserve organ functions and reduce the risk of complications by reducing intra-abdominal pressure (Gardner and Schroeder, 2022).

Supportive treatments: In patients with abdominal compartment syndrome, provision of supportive treatments is very important. Fluid therapy can be used to improve circulation in the body and increase organ perfusion. Appropriate use of analgesics and anti-inflammatory agents for pain management is also important. These can help the patient to relax and reduce stress (Hoareau, 2014; Łagosz *et al.*, 2022).

Surgical interventions: In cases with abdominal compartment syndrome, surgical interventions may often be required. Abdominal fasciotomy through incising the abdominal wall is a surgical procedure commonly used to reduce intra-abdominal pressure. In addition, surgical interventions may be required to determine the underlying cause of the problem and to consider an appropriate treatment plan. This can provide treatment for the underlying pathology of abdominal compartment syndrome and prevent complications (Luckianow *et al.*, 2012; Nielsen and Whelan, 2012).

The choice of treatment strategies may vary depending on the patient's clinical condition, intra-abdominal pressure level, and underlying causes. Therefore, it is important to adopt an individualized treatment plan for each patient.

Prognosis and Complications: The prognosis of abdominal compartment syndrome depends on factors such as early diagnosis, effective treatment, and management of complications.

Importance and impact of early diagnosis: Early diagnosis of abdominal compartment syndrome has an important influence on the prognosis of the disease. Early diagnosis can reduce the risk of complications due to the quick response of patient to treatment. In addition, early diagnosis can lead to timely start of the treatment which can protect organ functions. Therefore, it is critical to diagnose the problem and start treatment as early as possible in cases of suspected abdominal compartment syndrome.

Response to treatment and recovery process: The response to treatment and recovery process of patients with abdominal compartment syndrome depends on many factors. In patients which respond quickly to treatment, a decrease in intra-abdominal pressure, improved organ functions, and improvement in clinical symptoms can be observed. However, in some cases, response to treatment may not be satisfactory and complications may develop. The patient's general health status, underlying diseases, and the importance of starting treatment early are important factors affecting the recovery process.

Post recovery complications: Patients with abdominal compartment syndrome may develop certain complications after recovery. In particular, prolonged intra-abdominal pressure increase may worsen the risk of organ damage and systemic infection. Post-recovery complications include abdominal wound infection, intra-

abdominal abscess, and bowel obstruction. These complications should be managed through approaches like appropriate antibiotic therapy, surgical drainage, and supportive treatments.

Future Directions: The diversity of species and breeds among cats and dogs complicates the establishment of standardized data for ACS. Looking ahead, the focus in veterinary medicine will likely center on improving diagnostic methods and treatment strategies. Improved imaging techniques could significantly improve the diagnostic process, enabling earlier detection of ACS. Additionally, the development of more effective treatment strategies and approaches that enhance patient prognosis and quality of life seems to be crucial. Advanced technologies and surgical techniques hold the potential to make substantial progress in ACS treatment and improve patient condition.

Despite these prospects, remarkable deficiencies remain in ACS research. The pathophysiology and etiology of ACS are not yet fully understood. Basic scientific research is needed to elucidate the disease mechanisms and to develop effective treatment approaches. Furthermore, increasing emphasis on clinical trials and prospective follow-up studies seem essential for better understanding of the long-term prognosis of patients and making effective advancements in the ACS management.

Conclusions: Abdominal compartment syndrome in cats and dogs significantly influences clinical practice, especially in emergency settings where rapid intervention is crucial. This syndrome is a critical condition frequently encountered by veterinarians and requires prompt diagnosis and effective treatment. A multidisciplinary approach is essential to address the intensive care, general service, recovery, and reconstructive needs of these patients. To optimize outcomes for critically affected patients, it is often more effective to consider both medical and surgical intervention options concurrently. Following initial decompressive treatment, clinicians should also consider the option of transferring the patient to a more specialized center if needed.

Furthermore, ACS should be a key consideration not only in the postoperative management of surgical patients but also in determining anesthesia protocols and planning operations during the preoperative period. Establishing guidelines for the management of ACS and incorporating standard practices into routine care are vital. Advanced imaging techniques and laboratory tests should be employed in the monitoring and treatment processes, and current knowledge should be continuously updated through clinical research. By doing so, the management of ACS can be improved, leading to better patient outcomes and higher recovery rates.

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