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BASIC APPROACHES TO INTENSIVE CARE OF GASTROENTERITIS CAUSED BY PARVOVIRUS INFECTION IN DOGS

In the article developed basic approaches and regimens of intensive care for parvovirus infection complicated by various etiological factors.

Inflammation of gastrointestinal mucosa is one of the most common diseases in dogs. The main forms include gastroenteritis, enterocolitis, colitis, and gastroenteritis or gastritis caused by parvovirus and complicated by various pathogenic or opportunistic bacteria which virulent and toxigenic properties define their pathogenic profile. The symptoms include acute diarrhoea, vomiting, abdominal pain and, in severe cases, strong intoxication and dehydration [29, 30, 45]. In turn, infectious diseases localized primarily in the gastrointestinal tract (GIT) and accompanied by the abovementioned symptoms are commonly referred as *acute intestinal infections* (AIIs). The aetiology of AIIs however can hardly be traced either clinically or laboratory in more than half of cases [3]. Toxaemia may develop in severe forms of infection, especially, in cases of parvovirus gastroenteritis and immunodeficiency [24, 33]. A characteristic feature of AIIs is a quick and often rapid increase in the severity of clinical symptoms (such as toxicosis, diarrhoea or exicosis) which may cause a severe condition or even death just in a few hours unless properly treated. Therefore, the quality of early diagnosis of these diseases is of particular importance. The diagnostics should not be restricted to establishing a nosological form of the disease, but requires a veterinarian to establish and assess the reserves of a dog's body and the prognosis that is very important when intensive care is planned. Intensive care, when delayed for just a few hours, is crucial and largely determines the development of life-threating conditions associated with hypovolemic and infectious-toxic shock, acute renal failure, and acute disorders of cerebral circulation [23, 31, 46]. Diagnostics as well as the choice of pathogenic treatment may hardly be decided at the intensive care stage. Therefore, a veterinarian should firstly focus his/her efforts on:

– improving severe vital dysfunctions
– differentiating the infectious, therapeutic or surgical genesis of the disease

The aim of our research was to develop basic approaches and regimens of intensive care for parvovirus infection complicated by various etiological factors.

Intensive care refers to a system of therapeutic measures aimed at addressing or preventing disorders in vital functions to maintain the protective response and prevent transition into pathological conditions. Intensive care is intended to rapidly impact the main link in the chain of pathological disorders and, at the same time, to support functions of other less affected systems and to take measures to prevent secondary complications of such systems and organs. Intensive care can address functional disorders only. When vital organs are irreversible damaged, if, for example, anatomic integrity is damaged, intensive care will not give the desired effect [12, 20].

In dogs, parvo- and coronaviruses appear to play a major role in aetiology of acute viral intestinal infections associated with diarrhoea. When a parvovirus infection develops, the intestinal mucosa is deeply damaged. Therefore, the etiologic role of opportunistic pathogenic microflora in the development of AIIs appears to increase, though many elements of such intestinal flora may be harmless for a healthy dog under normal conditions [36, 38]. Diseases caused by pathogenic bacteria are often caused by activation of endogenous flora as a result of the failure of macroorganism's protective system which explains the severe course of the disease and significant problems in the treatment. Among the most common pathogens are *Citrobacter*, *Klebsiella*, *Proteus*, *Staphylococcus aureus*, *Clostridium spp.*, *Yersinia, Campylobacter jejuni* (Table) [37, 41, 44, 47].

The regimen of veterinary care in acute intestinal infections should be clearly planned and should include all veterinarians' measures to establish the

Table – **Pathogens and clinical forms of AIIs**

Pathogen	Disease	Major clinical forms
Salmonella spp., *S. typhimurium*, *S.enteritidis*, *Shigella spp.*	Salmonellosis Shigellosis	Gastroenteritis, colitis; generalized infection is rare. Lesion of the distal colon; gastroenteritis, general intoxication and bacteraemia may also develop
Escherichia coli – toxigenic and enteropathogenic strains;	Escherichiosis	Intoxication, GIT lesions; mucosal lesions in large intestine are more frequent
Yersinia	Yersiniosis	Gastroenteritis
Campylobacter spp.	Campylobacteriosis	Gastrointestinal form, gastroenteritis, enteritis; generalized infection may also develop
Different aerobic and some anaerobic bacteria	Acute bacterial diarrhoea	Enteritis, enterocolitis, gastroenteritis
Parvoviruses, coronaviruses	Viral diarrhoea	Gastroenteritis, enteritis. General intoxication may also develop

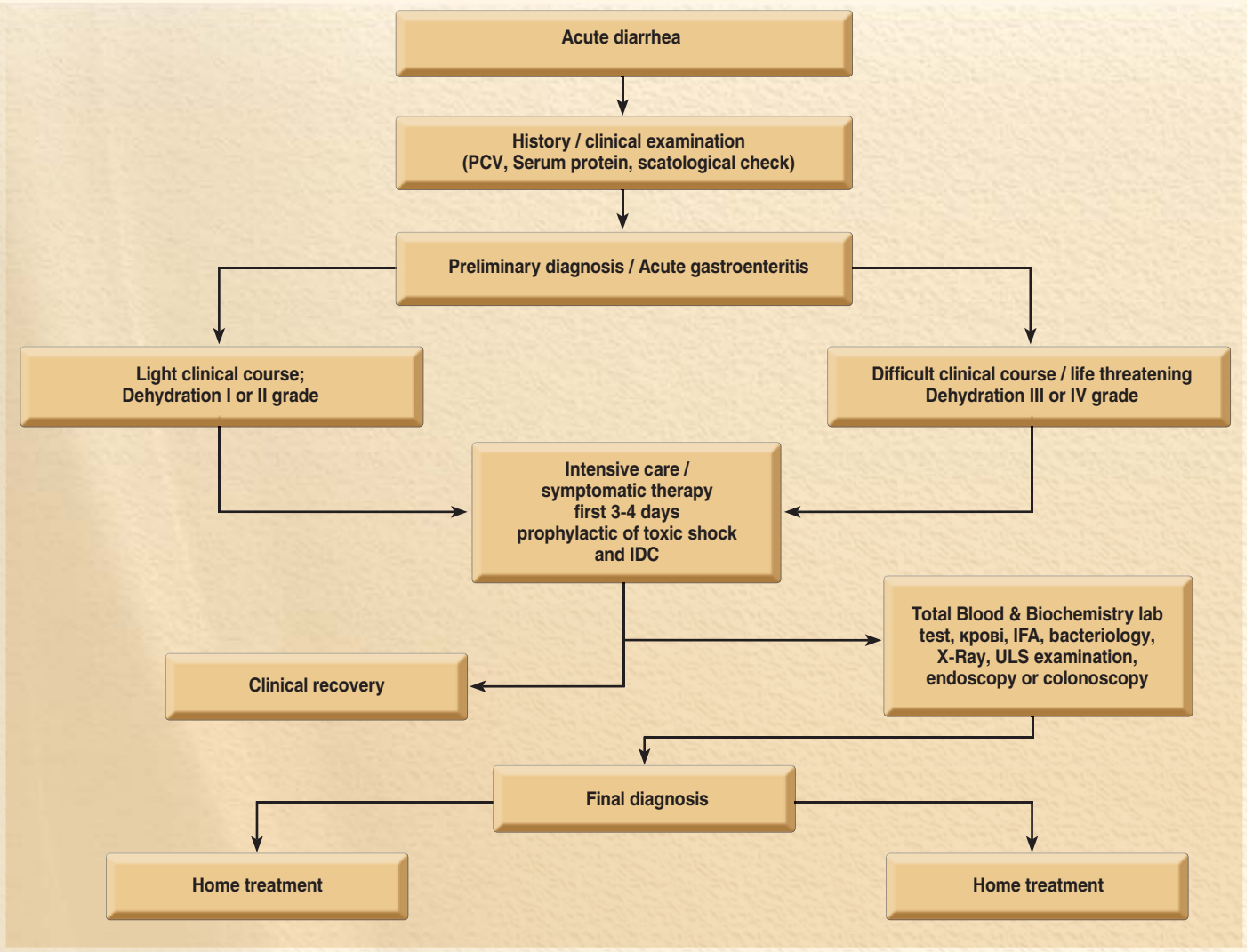

Fig. 1. Veterinary care for acute intestinal infection in dogs

preliminary diagnosis before therapy is initiated (Fig. 1).

Dehydration and the related arterial hypotension caused by intoxication causing drop in blood pressure and dysfunction of the central nervous system appears to be the major threat for dogs suffered from acute gastroenteritis. The scope of intensive care for AIIs is based on the monitoring of vital functions: consciousness and respiratory function, blood pressure and hydration. Therefore, this stage of therapy should be focused on: restoring of the heart rhythm, optimizing of the circulating blood volume and expelling of hypoxia and normalizing acid-alkaline balance [15].

Infusion therapy is one of the main approaches to intensive care [21, 28]. The indications for infusion care include severe dehydration (grade III); infectious-toxic shock; exicosis of any degree combined with severe intoxication; oliguria or anuria remained after the first stage of rehydration therapy; uncontrolled vomiting; inefficient oral rehydration therapy for 12 hours.

Considering literature data and practical experience, water and electrolyte imbalances have certain patterns which we have used to develop the intensive care regimen based, in particular, on parenteral solutions.

Some authors suggest administration of polyion crystalloid solutions such as Trisolum, Quartasolum, Chlosolum, and Acesolum for intravenous rehydration (IVR) therapy, consequently after analysis of the literature data, we applied polyion solution Ringer's Lactate which showed superior efficacy in our research [9, 40].

The volume of liquid for IVR therapy depends on the clinical condition of an animal when priority tasks are to restore circulating blood volume and total water and electrolyte losses of animal's body. There are three phases of rehydration therapy: urgent phase; compensatory phase; maintenance phase.

The volume of liquid for infusion is based on the assessment of body's needs in liquid to achieve the following goals: restore fluid deficit (to normalize the animal's condition); maintain the volume of liquid due to the ongoing losses; maintain the volume of liquid due to pathological losses.

Assessment of the volume of liquid depends on the results of the clinical ex-

	Na^+	Cl^-	K^+	HCO_3^-	Volume	Solution
Diarrhoea	↓	↓	↓	↓	↓	NORMOSOL^R-R + KCl or Ringer's lactate + KCl
Obstructed pylorus	↓	↓	↓	↑	↓	0,9 % NaCl + KCl
Dehydration	↑	↑	H	H / ↓	↓	NORMOSOL^R-R + KCl, Ringer's lactate + KCl, 0,9 % NaCl + KCl, 5 % dextrose

Fig. 2. Intensive care in the application of parenteral solutions

amination (percent of dehydration) or changes in animal's body weight.

It should be kept in mind that complete restoration of fluid deficit within 24 hours is an extremely difficult task because this can lead to loss of urine and further dehydration. Therefore, only 75 % to 80 % of the fluid deficit should be restored for the first 24 hours. Moreover, if the animal does not refuse to eat and drink, the volume may be increased to compensate for current and, if any, pathological losses. Rehydration therapy requires control of diuresis.

The volume of solution to be administered to compensate for fluid losses is calculated using the formula:

Fluid deficit (mL) = % dehydration × animal's weight (kg) × 1,000 × 0.80

Due to the lack of data on water requirements in dogs, recommendations of many authors vary greatly. It is commonly known that water and energy needs are numerically the same (1 kcal of energy = 1 mL of water). Recent researches indicate that the energy consumption is less than that reported in previously published formulas and recommendations. The assessment of water needs includes 50 mL/kg/day, 132 kcal × kg ^{0,75}, 156 × kg ^{0,667} (30 × kg) + 70, 70 × kg ^{0,75}. Indirect calorimetry is used today to assess dog's needs in energy (and water). These studies confirm the need to revise the previously recommended formulas for assessing dog's needs in energy (water). Therefore, the volume of liquid to maintain the current losses in dogs for the next 24 hours is determined by the formula:

Maintenance volume of liquid for 24 hours (mL) = 30 × animal's weight (kg) + 70

The question on sick animal's needs in energy and, therefore, in water due to the ongoing abnormal losses of fluid remains disputable. Traditionally, diseases, traumas, surgeries are believed to be associated with increased needs in energy (water) in humane medicine. These approaches are based on data obtained in humans and rodent experiments. As for dogs, there is growing evidence and publications confirming the lack of increased needs in energy and water in case of diseases, injuries or surgical interventions. Moreover, from an evolutionary point of view, a sick or injured dog is expected to adapt to preserve its energy. Under such extreme conditions, the reserves are already minimal and there is no sense to increase metabolic needs in order to survive. In other words, it would be more rational to preserve the existing energy and, therefore, water and to reduce metabolic losses. Studies of intensive care in dogs demonstrate that the function of the thyroid gland decreases. Therefore, the metabolic demand is reduced, and the most optimal method to determine the current pathological losses of liquid is to double the volume assessed by estimating such losses (vomiting, diarrhoea, and urination) [48].

Therefore, the total volume of liquid for infusion is determined by the formula:

Total volume of liquid (mL) = fluid deficit + maintenance volume / 24 h + current pathological losses / 24 h

The rate of intravenous infusion depends on the volume and speed of dehydration. Intensive fluid loss requires rapid recovery.

Calculate the rate of intravenous infusion of water-electrolyte mixture using the formula:

$$\frac{\text{Infusion rate, mL/h}}{\text{60 min}} \times \text{drops/mL (infusion system)} = \text{drops per minute}$$

In severe condition, start the infusion of water-electrolyte mixtures to a sick dog at a flow rate of 80 to 100 mL/kg/h or 30 to 35 mL/kg/h when the severity is moderate. Once the blood pressure is stabilized, reduce the infusion rate to 3 to 10 mL/kg/h. To prevent progressive dehydration, pulmonary oedema or hemodynamic insufficiency, the volume of liquid to be infused after stabilization of dog's condition may be 30 to 60 ml per kg of body weight. Once rehydration therapy is completed, administer KCl-solution based on serum potassium level. However, when electrolyte levels cannot be determined, KCl 10 to 15 mmol/L per litre of Ringer's lactate solution is considered safe during the maintenance phase of water and electrolyte infusion therapy. When KCl-

containing solution is administered, the infusion rate should not exceed 0,5 mmol/L/kg/h [39].

Antibiotics in acute gastroenteritis are associated with high risk of septicaemia. This may include parvovirus enteritis and haemorrhagic gastroenteritis. Signs of bacterial mucosal lesions include haemorrhagic diarrhoea, high WBC stool test, severe leukopenia, leucocytosis with a left shift or clinical signs of sepsis (such as fever, depression or shock).

The empirical therapy with ceftriaxone 20 mg per kg of body weight was determined and subsequently justified by microbiological studies in parvovirus infection complicated by enterotoxigenic strains of *E. coli* and other bacterial pathogens. The treatment lasted for 4 days and reduced the duration of diarrhoea syndrome up to 2 days. The purpose of the short-term parenteral antibiotic therapy at the early intensive care was to prevent life-threatening primary or secondary bacterial infections which diagnostics takes time while the clinical condition requires urgent symptomatic therapy.

Administration of enterosorbents, in particular, Enterosgel appears to be an important element of intensive care. Hydrogel of methyl silicic acid, the active ingredient of this medicinal product, selectively adsorbs toxic substances and products of incomplete metabolism in the gastrointestinal tract. The drug does not penetrate into cells of the mucous membrane but clears the blood from various endo- and exotoxins, products of metabolism and immune complexes (via membrane of capillaries of villi). When the latter are removed from the circulation, the ability of monocytes/macrophages and other phagocytes to clear the blood from antibodies and, as a consequence, from immune complexes is then restored. Enterosgel is reported to actively affect the colonization of normal intestinal flora by binding with pathogenic microorganisms and toxins [10, 27, 34].

As a part of our research, we have evaluated the efficiency of Enterosgel in intensive care of acute gastroenteritis accompanied by diarrhoea syndrome. Our studies confirmed that Enterosgel normalised main clinical symptoms in dogs significantly earlier than in the control group by the end of the first day. For example, subjective improvement has been observed in 80% of dogs by the end of the first day of treatment. At Day 3, the number of defecations decreased to 2 times a day (from 7/8 to 3/4 times a day) in 85 % of dogs in the main group and fences tended to become more solid. By the end of Day 4, decreased or no flatulence was observed in all animals, faeces normalized, and stool test results improved: WBC and RBC counts decreased or normalized and no mucus was found. Colon microflora profile improved in most animals (94,1 %) of the main group.

Under normal conditions, the antioxidant system counteracts the effects of free radicals. Under pathological conditions of the digestive tract, the intake of natural antioxidants with food is decreased due to absorption disorders. Oxidative damage to cellular components plays an important role in the development of acute gastroenteritis in dogs, especially of viral aetiology [19, 26]. Such lesions are the result of oxidative stress which develops when free radicals and active oxygen are formed in tissues quicker than the normal antioxidant mechanism can respond. This results in oxidization of cellular macromolecules such as lipids, nucleic acids, and proteins. Antioxidants provide the basic protective function of the organism against oxidative stress. The combined effect of antioxidants reduces the risk of cell damage due to such stress. We used the ethanolic extract of *Rhodiola rosea* 10 drops twice daily orally as an adaptogen in our studies to improve resistance to stress factors. Biologically active substances of *Rhodiola rosea* which show therapeutic effect include salidroside, rozin, rosavin, rosarin and tyrosol contained mainly in the rootstock of the plant. These substances increase the overall resistance of cells and the whole body against the harmful environmental effects, protect the cardiovascular system against stress and arrhythmias and demonstrate some antioxidant effects. There are data in support of hepatoprotective properties of Rhodiola products and their ability to stop the growth of malignant tumours and metastases in the liver and reduce the level of C-reactive protein and blood creatinine kinase after physical exercise indicating anti-inflammatory and protective effects of Rhodiola on muscle tissue during exercise. Adaptogenic, cardiopulmoprotective properties of the drug are mainly associated with properties of Rhodiola able to influence levels and activity of monoamines and opioid peptides such as beta-endorphins. Adding *Rhodiola rosea* to the intensive care regimen under development normalized the activity of aspartate aminotransferase (AST) and alkaline phosphatase, the level of medium molecular peptides, urea, bilirubin, and decreased the activity of alanine aminotransferase, inhibited the activity of alpha amylase and partially inhibited the increase of blood serum glucose level in dogs with symptoms of acute gastroenteritis. Therefore, the inclusion of the extract of adaptogens with a high content of polyphenols and 10% solution of ascorbic acid at a dose of 40 mg/kg to the treatment schedule provided not only adaptogenic properties, but also reduced the risk of complications caused by oxidative stress [22, 42, 43].

Thiotriazoline is another representative antioxidant compound with strong antioxidant effect used in intensive care of acute gastroenteritis in dogs. Intramuscular administration of 2,5 % thiotriazoline solution 2 mL a day improved the functional status of the liver: total protein level and serum albumin normalized, total bilirubin decreased, the activity of AST and ALT decreased, and glucose levels reduced showing positive hepatoprotective effect of thiotriazoline on hepatocytes in infected animals. This drug increased compensatory activation of anaerobic glycolysis, reduced inhibition of oxidation processes in the Krebs cycle, contributed to the preservation of ATP in tissue, demonstrated

strong antioxidant and immunomodulatory activity, and increased blood rheology [32, 35].

In case of ischaemic tissue damage, thiotriazoline is likely to normalize utilization of the reserves of glucose and glycogen in a cell and activity of glucose-6-phosphate dehydrogenase, increased NAD/NADH ratio and activity of cytochrome-C-oxidase, increased pyruvate, malate, isocitrate and succinate levels. At the same time, it reduced lactate overproduction, manifestations of uncompensated acidosis and its antioxidant action. Intensification of oxidative carbohydrate metabolism with thiotriazoline is known to increase ATP levels against increased deposits of ADP and, more crucially, to reduce the level of AMF [5, 16]. Mitochondria play a key role in the energy supply of cells in which thiotriazoline activates the process NAD+N-reoxidation of ethanol. In case of ischemia development, thiotriazoline firstly promotes utilization of recovered pyridine nucleotides (NADH) in the malate-aspartate shunt in mitochondria. Secondly, it activates the oxidation of NADH in lactate dehydrogenase reactions in cytosol. Positively affecting the utilization of recovered forms of pyridine nucleotides, thiotriazoline significantly inhibits pathways of reactive oxygen intermediate (ROIs) and activates oxidative phosphorylation by increasing ATP production. In ischemia, the protective effect of thiotriazoline is achieved by activating the malate-aspartate «shuttle» mechanism that supplies protons to the electron transport chain. In this case, compensatory increase in the power of malate shunt is accompanied by inhibition of the formation of acetyl-CoA from carbohydrates (in pyruvate dehydrogenase reaction) which, in case of ischemia, affects the synthesis of free fatty acids. Thiotriazoline-induced activation of the malate-aspartate mechanism not only promotes ATP production, but also inhibits abnormal synthesis of lipids [1, 2, 4, 6, 8, 11, 13, 14, 17, 18].

The therapeutic regimen including thiotriazoline is likely to normalize the activity of the antioxidant protection system and leads to rapid regression of clinical and morphological symptoms of gastroenteritis.

Acute gastroenteritis of any aetiology is accompanied by a reduction or acceleration of the motor activity of the digestive tract. Two disorders play the leading role in the mechanism of motor disorders of the digestive system: change in sensitivity and the structure of receptor system or bioelectric properties of muscle cells and imbalance of neurotransmitters, neuropeptides, and gastrointestinal hormones. Peristalsis of the digestive tract (DT) is controlled by neurotransmitters and hormones: motor function is stimulated under the influence of acetylcholine, serotonin, histamine, cholecystokinin, angiotensin, motilin, and gastrin and is slowed down by the inhibitory action of dopamine, noradrenaline, glucagon, vasoactive intestinal polypeptide, and somatostatin. To inhibit motility, drugs that stimulate the sympathetic nervous system, opiate and purinergic receptors or inhibit the parasympathetic nervous system and regulate the intracellular content of Ca^{2+} in muscle cells are used. Metoclopramide (Cerucal) is widely used in clinical practice. The latter has a dual mechanism of action. Metoclopramide blocks central and peripheral D2 receptors, stimulates the release of acetylcholine from postganglionic nervous fibre endings and is an agonist of 5 HT4 and an antagonist of 5 HT3 receptors. These mechanisms contribute to prokinetic effects of metoclopramide, mainly at the level of the proximal areas of DT. The drug reduces motor activity of the oesophagus, increases the tone of the lower oesophageal sphincter, and improves the evacuation of food from the stomach by toning up its body and increasing phase activity of the antrum. At the same time, the upper regions of the small intestine are relaxed. As a result, the development of duodenogastric reflux and vomiting is prevented, and the transit through the small intestine is then accelerated. Since advisability and possibility of long-term administration of metoclopramide as a prokinetic agent have not been established and studied yet, the latter is prescribed as a short-term therapy in secondary motor dysfunction of the upper regions of the digestive tract, dyspeptic disorder accompanied by comorbidities. In intensive care of acute gastroenteritis in dogs, metoclopramide should be used in a dose of 0,2 mg/kg every 6 to 8 hours in case of frequent vomiting unless bowel obstruction is suspected. Reduced frequency of vomiting along with intensive therapy to restore water and electrolyte losses and acid-alkaline balance contributed to more rapid reduction of acidosis in the first days of therapy [7, 25].

Thus, the analysis of literature data and our research indicate that the developed therapeutic regimen and, in particular, every component of the intensive care is highly efficient for acute gastroenteritis in dogs. The use of antioxidants significantly enhances pharmacotherapy.

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Основні підходи до інтенсивної терапії гастроентериту за парвовірусної інфекції у собак. *А.В. Дідух*

У статті розроблено основні підходи та схеми інтенсивної терапії парвовірусної інфекції, ускладненої різними етіологічними факторами.

Основные подходы к интенсивной терапии гастроэнтерита при парвовирусной инфекции у собак. *А.В. Дидух*

В статье разработаны основные подходы и схемы интенсивной терапии парвовирусной инфекции, осложненной различными этиологическими факторами. ○

З версією статті українською мовою можна ознайомитися на web-сторінці журналу ***– http://vmu.org.ua***

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КАМПІЛОБАКТЕРІЇ – ЗБУДНИКИ ІНФЕКЦІЇ, ЯКА МАЄ ПЕРЕБІГ З ОЗНАКАМИ ТОКСИКОІНФЕКЦІЇ

У статті висвітлено дані про збудників біологічних ризиків – бактерій роду* Campylobacter*, які спричиняють інфекції, що мають перебіг з ознаками токсикоінфекції. Описано їх морфологічні, культуральні властивості й особливості інфікування людей і тварин. Висвітлено поширення різних видів кампілобактерій в Україні та світі.

Забезпечення мікробіологічної безпеки харчових продуктів щодо збудників емерджентних зоонозних харчових токсикоінфекцій є актуальною проблемою в більшості країн світу. Останнім часом у світі найчастіше реєструють харчові отруєння, причиною яких є бактерії роду *Campylobacter*. Результати останніх наукових досліджень демонструють збільшення кількості випадків, коли тварини стають носіями збудників кампілобактеріозу, що призводить до зростання ризику виникнення харчових токсикоінфекцій у разі вживання контамінованих продуктів тваринного походження. Численними мікробіологічними й епідеміологічними дослідженнями доведено, що основними джерелами ризику кампілобактеріозу є молоко, вода та м'ясо сільськогосподарських тварин і птиці.

Мета роботи – вивчити й проаналізувати відомості про кампілобактерій – збудників інфекції, які мають перебіг з ознаками токсикоінфекцій.

Кампілобактеріоз – це зоонозне інфекційне захворювання, спричинюване бактеріями роду *Campylobacter*, який налічує близько 13 видів (G. Morris, Ch. Patton, 1985) (*див.* таблицю).

Раніше вважалося, що ці мікроорганізми уражують лише свійських тварин (велика рогата худоба, вівці, свині), доки в 1972 р. не виділили кампілобактерії з фекалій людей, хворих на діарею.

Морфологічно всі види й підвиди кампілобактерій ідентичні, різняться лише за патогенними властивостями для різних видів тварин, а також за серологічними й деякими культурально-біохімічними (не чітко) показниками. Кампілобактерії – це рухливі, грамнегативні, поліморфні, мікроаерофільні мікроорганізми зігнутої або спіралеподібної форми, спор і капсул не утворюють, залежно від виду містять один чи кілька джгутиків [9].

Для їх розвитку потрібні знижений уміст кисню та підвищений – вуглекислого газу. Оптимальне газове середовище для росту бактерій – суміш 5 % кисню, 10 % вуглецю, 85 % азоту. Кампілобактерії вимогливі до складу живильних середовищ. Обов'язковою умовою є наявність у середовищі 7–10 % еритро-