University of Mosul
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Website:

Lecture title: Septicemia, viremia, Toxemia

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- **Summary:** **Sepsis** is a suspected **or** proven bacterial infection in conjunction with the presence of systemic inflammatory response syndrome (SIRS), which is defined as systemic inflammation in response to injury, being caused by infectious agents (e.g., bacteria, viruses, protozoa, fungi) **or** by non-infectious causes (e.g., trauma, toxins, hyperthermia, burns).
- **Severe sepsis** is sepsis accompanied by organ dysfunction.
- **Septic shock** is defined as severe sepsis with hypotension (mean arterial blood pressure <65 mm Hg) despite aggressive intravenous fluid therapy.
- **Septicemia** is the acute invasion of the systemic circulation by pathogenic bacteria accompanied by septic shock with possible bacterial localization in various body systems **or** organs.
- **bacteremia,** bacteria are present in the blood stream for only transitory periods and do not produce clinical signs.
- **Viremia** is the invasion of the systemic circulation by pathogenic viruses with localization in various body tissues and in which the lesions produced are characteristic of the specific virus.

Etiology: septicemia or viremia All Species

- Many different infectious agents can result **in septicemia or viremia**. Some of the notable examples of septicemias and viremias are outlined next. **Anthrax, pasteurellosis, and salmonellosis are found in all species of food animal.**

PATHOGENESIS

- **Two mechanisms operate in septicemia:**
- The **exotoxins** or **endotoxins** produced by the infectious agents initiate a profound toxemia and high fever because of their initiation of the release of host mediators and because of the rapidity with which the agents multiply and spread to all body tissues (see also Toxemia, Endotoxemia, and Septic Shock).
- **The clinical manifestations** are the result of the effect of the pathogens on monocytes and lymphocytes, which initiate **SIRS**.
- **TNF-** α is associated with clinical septicemia in newborn foals and calves, and plasma.

- ♥ TNF-α concentration is associated with the severity of clinical signs.
- ♥ Localization of certain pathogens occurs in many organs and may produce severe lesions in animals that survive the toxemia.
- ♥ Direct endothelial damage and hemorrhages may also be caused.
- ♥ The same general principles apply to a viremia, except that **toxins are not produced by viruses.**
- ♥ It is more likely that the clinical manifestations are the result of direct injury of the cells invaded by the virus.
- ♥ **Transplacental infection** can occur, resulting in fetal **mummification**, **abortion**, or **infection of the fetus that may be carried to term**.

Disseminated IntravascularCoagulation

- ♥ Progression of SIRS can result in disseminated intravascular coagulation (DIC) caused by intravascular fibrin formation, particularly in severe septicemic diseases.
- ♥ Disseminated intravascular coagulation is initiated by vascular injury with partial disruption of the intima, caused by the circulation of foreign materials such as bacterial cell walls, antigen–antibody complexes, and endotoxin, with subsequent platelet adherence and the formation of platelet thrombi. Severe, uncontrolled

CLINICAL FINDINGS

✍ **The major clinical findings in septicemia** are fever, cardiovascular dysfunction and shock, and **submucosal and subepidermal hemorrhages that are usually petechial and occasionally ecchymotic**, **The hemorrhages are best seen under the conjunctiva and in the mucosae of the mouth and vulva.**

✍ Tachycardia, Tachypnea, and shock-induced organ dysfunction with cardiovascular hypotension, myocardial asthenia, and respiratory distress may occur in severe cases if the pathogen initiates the release of the host mediators, causing SIRS. These features are described under Toxemia, Endotoxemia, and Septic Shock.

TREATMENT

✍ The principles of treatment are similar to those described for the treatment of toxemia, endotoxemia, fever, and septic shock, and treatment should focus on **broad-spectrum antimicrobial agents and general supportive measures.**

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Toxemia, Endotoxemia

Toxemia is a clinical systemic state caused by widespread activation of host defense mechanisms to the presence of toxins produced by bacteria or injury to tissue cells.

- **Endotoxemia** is The most common form of toxemia in large animals, caused by the presence of lipopolysaccharide cell-wall components of **gram-negative bacteria in** the blood and characterized clinically by abnormalities of many body systems.
- **The abnormalities of endotoxemia include the following:**
- Marked alterations in cardiopulmonary function.
- Abnormalities in the leukon (neutropenia and lymphopenia)and thrombocytopenia that may lead to coagulopathies .
- Increased vascular permeability.
- Decreased organ blood flow and metabolism, leading to heart and renal failure.
- Decreased gastrointestinal motility.
- Decreased perfusion of peripheral tissues, leading to shock.
- The need for intensive and complex therapy.
- A high case fatality rate .

♥ ETIOLOGY:

- **Antigenic Toxins** These are produced by bacteria and to a lesser extent by helminths, divided into exotoxins and endotoxins.

♥ **Exotoxins** These are protein substances produced by bacteria that diffuse into the surrounding medium, *Clostridium* spp.

♥ Enterotoxins These are exotoxins that exert their effect principally on the mucosa of the intestine, causing disturbances of fluid and electrolyte balance, enterotoxigenic *E.coli.*

♥ **Endotoxins** :The endotoxins of several species of gram negative bacteria are a major cause of morbidity and mortality in farm animals, gains access to the blood when there is a severe localized infection, such as a coliform mastitis in dairy cattle.

♥ **Metabolic Toxins** accumulate as a result of incomplete elimination of toxic materials normally produced by body metabolism, or by abnormal metabolism.

♥ CLINICAL FINDINGS:

1- **Depression**, **anorexia,** and **muscular weakness** are common in acute endotoxemia.

- **Toxic or septic shock:**

- ♥ **The remarkable clinical findings are :**
- ♥ Severe peripheral vasodilatation with aconsequent fall in blood pressure
- ♥ Pallor of mucosa
- ♥ Hypothermia
- ♥ Tachycardia
- ♥ Pulse of small amplitude
- ♥ Muscle weakness

2- **Severe endotoxemia include the following**:

- ♥ Depression
- ♥ Hyperthermia followed by hypothermia
- ♥ Tachycardia followed by decreased cardiac output
- ♥ Decreased systemic blood pressure
- ♥ Cool skin and extremities
- ♥ Diarrhea
- ♥ Congested mucosae with an increased capillary refill time
- ♥ Muscular weakness, leading to recumbency

4- **Renal failure is common** and is characterized by **anuria.**

5- **If** disseminatedintravascular coagulation**(DIC) develops**, it is characterized by ***petechial and ecchymotic hemorrhages on mucous membranes and sclerae with a tendency to bleed from venipuncture sites.***

6- **Chronic Toxemia:** Lethargy, separation from the group, inappetence, failure to grow or produce, and emaciation are characteristic signs of chronic toxemia.

7- **Localized Infection**

Clinical Pathology:

a) Leukocytosis and neutrophilia occur with mild endotoxemia

b) leukopenia, neutropenia, and lymphopenia increase in severity and duration with increasing severity of endotoxemia.

c) A low plasma glucose concentration, high serum urea concentration, and a low serum albumin and total protein concentration

♥ Treatment

1) Removal of the foci of infection

2) Administration of antimicrobial agents with a gram-negative spectrum.

3) Aggressive fluid and electrolyte therapy to combat the relative hypovolemia, systemic hypotension, hypoglycemia, and electrolyte and acid-base disturbances

4) NSAIDs or glucocorticoids for the inhibition of products of the cyclooxygenase pathway.

Stress

- **Stress** is a systemic state that develops as a result of the long-term application of stressors, it includes pain.
- **Stressors** are environmental factors that stimulate homeostatic, physiologic, and behavioral responses in excess of normal .
- **The importance of stress:**
- Lead to the development of psychosomatic disease.
- Increase susceptibility to infection.
- Represent an unacceptable level of consideration for the welfare of animals.
- Reduce the efficiency of production.

- **Thirst:** is an increased desire for water manifested by excessive water intake**(polydipsia).**
- **Appetite** is a conditioned reflex depending on past associations and experience of palatable foods, and is not dependent on hunger contractions of the stomach.
- **Hyperorexia, or increased appetite**, caused by increased hunger contractions, is manifested by **polyphagia** or increased food intake.
- Partial absence of appetite **(inappetence)** and complete absence of appetite **(anorexia)** are manifested by varying degrees of decreased food intake (**anophagia**).

- Undernutrition can be defined as a prolonged inadequate supply of nutrients to sustain good health and, in the case of immature or underweight animals, growth potential.

- **Malnutrition** is a deficit, imbalance, or excess of nutrients with consequential adverse effects on health and growth potential.
- **Pica** is the ingestion of materials other than normal food and varies from licking to actual eating or drinking. It is associated in most cases with dietary deficiency, either of bulk or, in some cases, more specifically fiber, or of individual nutrients, particularly salt, cobalt, or phosphorus. It is considered as normal behavior in rabbits and foals

The type of pica:

- **Coprophgia** (eating of feces) incase B12 deficiency.
- **Ostesphagia** (eating of bone) incase Ca^{+} & Ph^{+}-def. it is normal in dog.
- **Infantophagia**(dam can eat aborted fetus and fetal membranes).
- **Alltrophagia or Geophagia** (clay eating from the ground).
- **Trichophagia** **(**hair ;wool, and other fibers eating).

Starvation

Complete deprivation of food causes rapid depletion of glycogen stores and a changeover in metabolism to fat and protein. In the early stages there is hunger, increase in muscle power and endurance, and a loss of body weight