DISEASES OF THE VASCULAR SYSTEM

- A. Arterial Diseases
- B. Venous Diseases

Metabolic arteriopathies

- These are arterial diseases related to disorders of arteries that are not related to inflammation.

Metabolic arteriopathies

- Metabolic arteriopathies are divided into two main groups;

- a. Dystrophic arteriopathies,

- b. Degenerative arteriopathies

Dystrophic arteriopathies

- Dystrophic arterial diseases are relating to or caused by faulty nutrition of arterial wall.

Dystrophic arteriopathies

❑**Changes related to aging in arteries**:

Some changes occur in the walls of the artery as a result of events that are related to the progression of age

- **Fibrosis** in the aorta and A. pulmonalis
- **Thickening** of the artery wall
- **Fat accumulation** in the artery wall
- **Folding** of tunica intima in artery
- **Pregnancy sclerosis** in uterine vessels

Dystrophic arteriopathies

❑**Intima Sclerosis in Arteries :**

- Morphologically, it is divided into sub-sections as focal and diffuse - central and peripheral.
- It can be divided into subgroups about pathogenesis as reparative - degenerative - inflammatory and compensative -.
- Intima sclerosis usually occurs in the heart, kidney and thyroid arteries.
- Among animal species, it is most common in dogs with chronic kidney disease.

Dystrophic arteriopathies

❑Intima Sclerosis in Arteries :

The reasons for this can be listed as follows;

- **Degenerative and inflammatory arteriopathies**
- **Traumas in the arter wall**

- **Damage** to the intima layer - rupture - injury - lesions caused by parasites and thrombosis are **tried to be repaired by** proliferation of a **wide range of fibrils**. Hence, the tissues of these lesions occur in the **scar**. Thus, **regional intima thickening occurs.**

- In the case of **fibrinoid degeneration** in the arteries, the **degenerated parts are resorbed**, and then they recover in the remaining damaged areas.

- Thus, **intima sclerosis is formed.**

- Intima sclerosis is **permanent.**
- The lumens of such arteries **narrow.**
- Therefore, **nutritional disorders** are **seen in the organs** fed by arteries in which sclerosis occurs.
- Sometimes **calcifications** may occur in such lesions.

Dystrophic arteriopathies

❑ **Medial Hyperplasia in the Artery Wall:**

- An artery thickening is associated with an increase in muscle fibers in the media layer.
- It is mostly seen *in humans.*
- In animals, *the cats, rabbits and guinea pigs* are found only in the pulmonary arteries.

Dystrophic arteriopathies

- **Medial hypertrophy of the pulmonary arteries of cats:**

•Displays no age, sex, or breed predilection

•Appears to be normal anatomic variation in cats

•Similar pulmonary arterial lesions have been described in cats infected with the **parasites** *Aelurostrongylus abstrusus* and *Dirofilaria immitis.*

- Pulmonary hypertension does not result from the vascular change, right ventricular pressure does not increase, nor does the right ventricle hypertrophy.

✓ **The most severely affected vessels** may be grossly visible **on the cut surface of the lung** and through the pleura when the lungs are collapsed, and <u>are even palpable in some cases.</u>

✓ The histologic spectrum of arterial changes ranges from mild *sporadic or generalized hypertrophy* of the **tunica media.**

Dystrophic arteriopathies

- **Arterial aneurysm:**

- It is a limited and transverse **extension** of the artery wall.
- It is not a direct disease but a lesion that occurs as a result of many vascular diseases.
- It occurs more often **in horses** and often occurs in relation to **parasites**.
- Most of these events in horses occur in **Arteria ileocaecalis** and the less in branches of Arteria mesenterica cranialis.
- **Strongylus vulgaris** larvae form **verminous aneurysm** in horses.

Dystrophic arteriopathies

❑**Calcification in the artery wall (intima and media):**

- **Calcification (mineralization)** occurs quite frequently in the arteries of animals, either as a dystrophic or metastatic process.

- **Dystrophic mineralization,** or *mineralization of dying or dead tissue*, occurs in areas of inflammation, degeneration, and thrombosis, and not necessarily in association with pre-existing arteriosclerosis.
- **Metastatic mineralization** occurs as the *result of hypercalcemia and/or hyperphosphatemia.*

- **Causes of dystrophic calcification,**
  - Dystrophic, degenerative and inflammatory arteriopathies,
  - Verminous arteriopathies,
  - Tromboendoarteritis
  - Endoarteritis due to leptospirosis in the lung arteries,
  - Infarction and media sclerosis,
  - Lesions associated with diseases like Tuberculosis e.g.
- **Reasons for metastatic calcification,**
  - Hipovitaminosis D,
  - Common chronic interstitial nephritis of dogs,
  - Renal osteopathies,
  - Parathyroid hyperfunction and tumors.

❑**Arterial thrombosis and embolism:**

The 3 major predispositions to thrombosis are

1. ***Injury to endothelium***, for instance, via infectious, toxic, or immunologic mechanisms
2. ***Altered blood flow***, as occurs with stasis or turbulence
3. ***Hypercoagulability of the blood***, which may result from increased concentrations of activated procoagulants, increased numbers or stickiness of platelets, or decreased concentrations of inhibitors such as antithrombin in the nephrotic syndrome.

- ***Thrombi*** are of importance because they **occlude** the vessel.
- Arterial occlusion is of significance in organs with an **end-arterial blood supply**, such as the kidney, because of the absence of collateral circulation and the development of infarction.
- Simultaneous occlusion of a large number of pulmonary arterioles or arteries by thrombi can lead to **right heart failure (cor pulmonale)** and **death.**

❑Disseminated intravascular coagulation (DIC):

- *Disseminated intravascular coagulation (DIC) is a common and important intermediary mechanism of disease, but is not a disease in itself.*

DIC

- It may be defined as a pathologic activation of the **coagulation system** that leads to ***generalized intravascular clotting*** involving, in particular, arterioles and capillaries.
- The process may be *acute, subacute, or chronic,* and may be *localized or generalized.*
- The terms *"consumption coagulopathy," "defibrination syndrome," and "consumptive thrombohemorrhagic disorder"* are also used because of the massive consumption of coagulation factors that occurs and that may be sufficiently severe for hemorrhagic diathesis to result.

DIC

- *A wide array of agents and conditions will initiate coagulation* either by

❖Causing *widespread* ***endothelial damage*** and thus exposing thrombogenic subendothelial collagen, or

❖Directly ***activating the coagulation cascade*** via the intrinsic or extrinsic pathway.

DIC

- Exposure of monocytes, macrophages, and endothelial cells to disease agents or mediators will cause ***expression of tissue factor (tissue thromboplastin)*** on the cell surfaces and activation of the extrinsic pathway of coagulation, and is likely a predominant pathway of DIC.

Degenerative arteriopathies

- The degenerative events forming in tissues of the artery wall can be listed as follows:

❖Hyaline degeneration of arterial wall (Hyalinose),

❖Amyloid degeneration of the artery Wall (Amyloidose),

❖Fat degeneration of the artery wall (Lipoidose).

ARTERIOSCLEROSIS

Arteriosclerosis literally means "hardening of the arteries," and is more fully defined as: *chronic arterial change consisting of hardening*

- *loss of elasticity*
- *luminal narrowing (*resulting usually from proliferative and degenerative, rather than inflammatory)
- *changes of the media and intima*

ARTERIOSCLEROSIS

- Arteriosclerosis includes three main types of lesions.

a) **Atherosclerosis,**
b) **Monckeberg's medial sclerosis,**
c) **Arteriolosclerosis**

a) Atherosclerosis

- It is characterized by the formation of

✓ yellowish atheromatous plaques

✓ on the *inside of intima and medial layers*

✓ in large and medium size arteries and

✓ excessive fibrosis of these layers.

a) Atherosclerosis

- The term **atherosclerosis** is applied to lesions of arteriosclerosis in which ***degenerative fatty changes*** also occur.
- ***Atherosclerosis*** *is the most common and important type of arteriosclerosis in humans*, and the terms can thus be used interchangeably with little loss of meaning when discussing this species.
- *In domestic animals,* ***arteriosclerosis is common****, but of little clinical importance, and atherosclerosis is rare.*

a) Atherosclerosis

- The structure of atheromas consists of cholesterol, lipoid substances, destroyed cell and tissue residues and lipophages.
- Atheromas are softy masses.
- These masses surround the fibrous tissue that develops from the intima and form focal protrusions above the intima.
- Therefore, these masses are called ‘atheromatous plaque’ ”or fibrolipoid plaque’.

a) Atherosclerosis

- Atherosclerosis is the most common form of arteriosclerosis in humans.
- Atheromatous plaques occur not only in the aorta and its branches, but also in the coronary and cerebral arteries.
- Parrots and home pigeons are the most common species of atherosclerose.
- In mammalian species, only pigs have genuine atherosclerose.
- Meanwhile, atherosclerose events have been reported in cattle, horses and dogs.
- It has been reported that atherosclerosis of endocardial, aorta and renal arteries in Guernsey breed with **paratuberculosis** is similar to atherosclerosis in humans.

b) Monckeberg's medial sclerosis

Monckeberg's arteriosclerosis (medial calcific sclerosis),

- This form of sclerosis occurs in muscular medium-diameter arteries.
- It is characterized by excessive thickening/ hardening and calcification of the **medial layer of arteries.**
- The disease is also called 'Monckeberg degeneration'. It is mostly seen in older people.
- In animals, this form of disease is quite common. This type of calcification is often seen in the thoracic arteries of butchery cattle.

c) Arteriolosclerosis

- In peripheral small-diameter arteries (arterioles) is recognized by the thickening of the **intimal layer.**

- The disease is divided into two subgroups as **‘hyaline arteriolosclerosis’** and **‘hyperplastic arteriolosclerosis’**.

ARTERIOSCLEROSIS

REASONS:

- Factors considered as the cause of all forms of arteriosclerosis;
- Senility
- Hypertension (in humans)
- Genetic factors affecting the structure of the vessel wall
- Increased endothelial permeability and injury of endothelial layers of the arteries in infectious - toxic diseases.

ARTERIOSCLEROSIS

REASONS:

a) Atherosclerosis;

- General disorders of lipid metabolism,
- Excessive amounts of fatty foods through the digestive system, hence increasing plasma cholesterol levels,
- All factors leading to hyperlipidemia
- Hyperthyroidism
- Animals, especially poultry, due to not moving freely
- Excessive lubrication,
- Paratuberculosis events in Guernsey breed cattle.

ARTERIOSCLEROSIS

REASONS:

b) Monckeberg's arteriosclerosis ,

- Factors that are particularly vasotonic in this type of arteriosclerosis are considered to be responsible.
  - For example, excessive stimulation of smooth muscle tissue in the media layer of arteries through vasomotor.
  - Monckeberg's arteriosclerosis was created experimentally in dogs and rabbits by epinephrine (adrenaline) and nicotine injection.
- Hypervitaminosis D

ARTERIOSCLEROSIS

REASONS:

c) Arteriolosclerosis

- Hypertension,
- Nicotine (1-2 pack of cigarettes per person for a long time),
- Damage of the endothelial layer by endotoxins,
- Damage of the endothelial layer by various drugs,
- Diabetes mellitus (in humans).

ARTERIOSCLEROSIS

MACROSCOPIC FINDINGS:

a) Atherosclerosis,

- The vein wall is thickened and hardened especially in the big arteries. Typically **sclerotic atheromatous plaques** are seen on the inner face (intima layer) of these arteries.
- The new ones are in *yellowish spots and blisters.*
- In advanced cases, these plaques are enlarged and thickened.
- **In paratuberculosis of the cattle**, it has been reported that *petechial hemorrhages*, *necroses* and *ulcers* are also found in the aorta thoracica with arteriosclerosis.

ARTERIOSCLEROSIS

MACROSCOPIC FINDINGS:

b) Monckeberg's arteriosclerosis ,

- Since the calcification occurs in the form of rings in the vessel wall or less as plaques, the affected vessel becomes a stiff pipe.

MACROSCOPIC FINDINGS:

c) Arteriolosclerosis,

- In hyaline arteriolosclerosis, altered arterioles are seen as gray-white transparent foci (eg, nephrosclerosis).
- In hyperplastic arteriolosclerosis, the affected arterioles are in appearance resembling the onion (the walls of such vessels are thickened concentrically).

ARTERIOSCLEROSIS

MICROSCOPICAL FINDINGS:

a) Atherosclerosis,

- At the beginning, the intimal layer becomes edematous, follows it fibrous connective tissue proliferation, follows it the accumulation of cholesterine crystals or accumulation of neutral fats inside cholesterol clefts.
- Most of the lipoid substances were phagocytosed by foam cells - fat macrophages. This event is identified as 'atherosclerosis plaque'.
- When the event progresses, becomes old and widespread, it is observed that the tissues forming the artery wall are destroyed.
- It is observed that a giant cell granuloma tissue is formed in the environment. *In some cases (necrotic events), calcifications are also present. Metaplasic cartilage tissue may also be encountered in old events.*

ARTERIOSCLEROSIS

MICROSCOPICAL FINDINGS:

b) Monckeberg's arteriosclerosis,

In affected arterial walls, in the medial layer of the areas where fibrous connective tissue growth, it is observed that it becomes thick due to the circle and ring-shaped calcification zones.

ARTERIOSCLEROSIS

MICROSCOPICAL FINDINGS:

c) Arteriolosclerosis,

- **In hyaline arteriolosclerosis**, a homogenous hyaline accumulation is initially observed in the subendothelial tissue.

- **In the hyperplastic arteriolosclerosis**, it is observed that the smooth muscle cells initially progress towards the intimal layer. In advanced stages, these cells overgrowth and collagen accumulation is seen.

- **In old lesions**, such arteriolar walls (with respect to smooth muscle and fibrous connective tissue proliferation) appear to be a concentric layer resembling onion section.

VASCULITIS

- *Vasculitis, or inflammation of a vessel, is characterized by the presence of inflammatory cells within and around the blood vessel wall with concomitant vessel wall damage as indicated by fibrin deposition, collagen degeneration, and necrosis of endothelial and smooth muscle cells.*
- The term ’angitis‘ is also used instead.
- Arteritis
- Phlebitis

VASCULITIS

- In vasculitis;
- Vascular endothelium may be damaged and thrombosis may occur.
- Lymphohistiocytes and neutrophil leukocyte infiltrations may occur around the vein.
- Fibrinoid degeneration, necrosis, fibrosis, proliferation are observed in the vessel wall and calcification may occur.

VASCULITIS

Causes of vasculitis in domestic animals

- VIRAL;
  - Equine viral arteritis
  - Equine infectious anemia
  - African horse sickness
  - Hog cholera
  - Corysa Gangrenosa Bovum (Malignant Catarrhal Fever)
  - Bluetongue
  - Equine viral rhinopneumonitis (in the form of encephalomalacie)
  - Feline Infectious Peritonitis (FIP)
  - Aleutian disease (mink)

VASCULITIS

- CHLAMYDIAL;
  - Sporadic bovine encephalomyelitis (Chlamydia psittaci)
- RICKETTSIAL;
  - Rickettsia rickettsii
  - Ehrlichia canis, Ehrlichia equi
- BACTERIAL;
  - Salmonella spp.
  - Erysipelothrix rhusiopathiae
  - Hemophilus somnus, H. suis, H. parasuis, H. pleuropneumoniae
  - Corynebacterium pseudotuberculosis

VASCULITIS

- MYCOTIC;
  - mucormycosis
  - Aspergillus fumigatus
  - Histoplasma farciminosum
  - Sporothrix schenkii
- PROTOZOAL;
  - Encephalitozoon caniculi
  - Besnoitia besnoiti
- HELMINTHS;
  - Strongylus vulgaris, Dirofilaria immitis, Spirocerca lupi, Onchocerca spp.,
  - Elaeophora spp., Aelurostrongylus abstrusus, Angiostrongylus vasorum,
  - Schistosoma spp. (Phlebitis), Brugia spp. (Lymphangitis).

VASCULITIS

Non-infectious;

- Immune mediated,
  - Systemic Lupus erythematosus
  - Rheumatoid arthritis
  - Polyarthritis nodosa
  - Anaphylactoid purpura
  - Staphylococcal hypersensitivity
  - Serum disease.
  - Some drugs

- Immune mediated;
  - Uremia

ARTERITIS

Is the inflammation of the tissues forming the artery wall.

- Cause by infectious - toxic - parasitic - immunological effects.

According to location:

- Endarteritis (when located in the endothelial layer),
- Mesarteritis (when located in intimal and medial layer),
- Periarteritis (when located in adventitia),
- Panarteritis (if inflammation spread to all layers).

ARTERITIS

- This classificaiton of arteritis is not possible in practice. Because inflammation often involve several layers.
- Therefore, the classification of arterial inflammation can be done due the nature of the exudate and the pathogenesis of the case.

ARTERITIS

- According to this;

1. Arteritis serosa
2. Arteritis purulenta
3. Arteritis trombotica
4. Arteritis necroticans
5. Arteritis productiva

ARTERITIS

- ✓ Arteritis serosa: Serous exudate is present in the artery wall and the beginning of other inflammations.
- ✓ Arteritis purulenta: Purulent exudate penetrates the artery wall. Causes of different types of pus are responsible. It is a common type of arteritis in animals. It mostly occurs in the umbilical arteries in relation to the umbilical infections. Macroscopically, the vein is dirty-red-red and bulging.
  - Microscopically, neutrophil leukocyte-rich inflammatory edema, degeneration of the vascular wall, necrosis, desquamation of endothelial cells and lumen thrombosis can be seen.

ARTERITIS

- ✓ Arteritis thrombotica: is a purulent-trombotic inflammation that is about parasitic parasitic larvae occurs in all layers of artery wall.
- This type of arterial inflammation is seen with the invasion of Strongylus vulgaris larvae in horses.
- Parasitic larvae form embolus in arteries with blood. It causes injury to the endothelial layer. Therefore, local traumatic lesions develop. As a result of the accumulation of leukocytes and fibrin in intima and media, thromboses occur at the sites of injury and such inflammation occurs. If the thrombosis is organized over time, the vessel wall becomes thicker.
- Scar and calcification are formed.

ARTERITIS

- ✓ Arteritis necroticans: It is characterized by necrosis in the vein wall. Intima is rough-brown-greenish in color. Fibrin masses are noticeable. Thrombosis usually occurs in the lumen.
  - Microscopically, necrosis areas and cell debris are seen in pink homogenous appearance in the affected vessel. There are neutrophil leukocytes, lymphocytes and macrophage infiltrations around them. In chronic events, fibrous connective tissue proliferation is observed. In addition, aneurysms may be seen in place of these lesions.
- ✓ Arteritis productiva: Arterial walls are thick-hard-yellowish-brown. Excessive connective tissue proliferation, lympho-histiocytic cell infiltrations are seen. In the intima, connective tissue increase and thrombosis are formed. This type of features are seen in large vessels.

ARTERITIS

Important diseases with arteritis

- Equine Viral Arteritis (Pink Eye),
- African horse sickness
- Gangrenous Coryza (Malignant Catarrhal Fever),
- Hog Cholera (Pestis Suis, Schweinepest),
- Rocky Mountain Spotten Fever
- Uremic Arteritis,
- Panarteritıs Nodosa.

Equine viral arteritis

- **Equine viral arteritis.** This disease is caused by species ***Equine arteritis virus*** (EAV), an RNA virus of the family *Arteriviridae*, genus *Arterivirus*, which is pathogenic only for horses and is cytopathic in equine kidney culture.

Equine viral arteritis

- Transmission of virus occurs primarily by *respiratory and venereal routes* during the acute phase of infection.

Equine viral arteritis

Macroscopical findings

- Transudate with **fibrin** in pericardium, thorax and abdominal cavity,
- Subcutaneous edema,
- Hemorrhage in various organs (pleura, endo - epicard, larynx, pharynx, muscles, etc.),
- Lymph nodes are bulging due to edema and hyperemia,
- Edema and catarrhal inflammation in the lung,
- Edematous enteric wall and catarrhal hemorrhagic enteritis,
- Edema around the aorta.

Equine viral arteritis

Microscopical findings

- **The presence of fibrinoid degeneration** and **necrosis** in the small arteries (0.5 mm diameter) is the characteristic finding.
- Since there are *no degenerative changes in endothelial cells*, thrombosis is not formed.
- However, in some cases, thrombosis may occur in the colon and caecum, and Hemorrhage and necrosis may be formed.

African horse sickness

- The agent is orbivirus.
- It is transmitted by insects (culicoides).
- Dogs eating the flesh of infected horses can be infected even if they have disease.

African horse sickness

- **Clinically;** four forms were defined as lung, heart, mixed and horse fever.
- **Macroscopically;**

✓ **In the lung form**, edema in the lungs, hydrothorax is seen.

✓ **In the heart form**, hydropericardium, ecchymotic hemorrhages in epicard and endocard, these hemorrhages are concentrated along the coronary vessels, especially under the bicuspidal and tricuspital valves and at the connection sites of the chordae tendineae in the papillary muscles.

✓ **In mixed form**, lesions observed in lung and heart forms are observed.

- **Microscopically;** hemorrhages, edema, focal necrosis, inflammatory cell infiltrations and increase in connective tissue.

Corysa Gangrenosa Bovum (Malignant Catarrhal Fever)

- Microscopic lesions that are pathognomonic are necrosis in lymphocytes in lymphoid tissue and **fibrinoid - necrotic vasculitis** especially in the wall and adventitia of small arteries and mononuclear cell infiltration.

Hog Cholera (Classical swine fever)

- *Classical swine fever (CSF) is a highly contagious viral disease of swine; it may ocur as acute, subacute, chronic, or inapparent syndromes.*
- CSF is caused by a *Pestivirus*, a member of the family *Flaviviridae*.
- *Transmission* of the disease is usually by direct contact of infected pigs.
- The virus is found in the urine and faeces of the infected animals and the eye and nasal discharge and infects the environment.

Hog Cholera (Classical swine fever)

- ***Clinically,*** *classical swine fever is characteristically an* ***acute disease*** *of high morbidity and mortality*, most animals surviving only to 14 days after showing the first signs of illness.

Hog Cholera (Classical swine fever)

Macroscopically:

- The cadaver is dehydrated, the eyes are submerged.
- In the non-pigmented parts of the skin bleeding may occur.
- Petechia are observed in the periphery of the lymph nodes and in the kidneys.
- Infarction of spleen in acute cases is pathognomic.
- In the epicardium, petechae are common. The pericardium contains a small amount of exudate in the color of straw.

Hog Cholera (Classical swine fever)

Macroscopically:

- With transplacental infection nervous system findings such as:
  - ✓microencephalus,
  - ✓hydrocephalus,
  - ✓cerebellar hypoplasia,
  - ✓Hypomyelinogenesis
  - ✓ pulmonary hypoplasia

Macroscopically:

- Changes in veins are in the form of primary degenerative changes in the endothelium and sometimes in the form of proliferative changes.
- In the vein, **fibrinoid vasculitis and degenerative changes and perivascular lymphocyte - histiocyte infiltrations** occur.
- Infarcts with diagnostic significance occur in the spleen.

Rocky Mountain spotted fever

- **Rocky Mountain spotted fever.** Rocky Mountain spotted fever (RMSF), a febrile exanthema caused by ***Rickettsia rickettsii*,**
- is an important rickettsiosis of dogs and humans
- The *ticks* most commonly responsible for the transmission.
- The factors proliferate in endothelial cells of small blood vessels.
- Is seen in dogs especially under 2 years of age, it is reported that people are affected.

Rocky Mountain spotted fever

Clinically:

- Stagnation, fever, lymphadenomegaly, dyspnea, conjunctivitis, paralumbar hyperesthesia, edema in the face and extremities, petechiae or hemorrhagic diathesis, vomiting and diarrhea are seen.

Rocky Mountain spotted fever

Macroscopically:

- Nasal and ear edema, ulcerative glossitis, scrotal dermatitis and petechial hemorrhages in mucosa, abdominal skin, pleural and gastric wall are observed.
- Hemorrhagic colitis and hemorrhagic lymphadenopathy may also occur.

Microscopically:

- **Necrotic vasculitis in capillaries and arterioles** in small veins; **perivascular lymphocytes and macrophage infiltrations** are observed.
- Acute splenitis and interstitial pneumonia; multifocal necrosis occurs in the myocardium, adrenal glands and liver.

Uremic Arteritis

- In uremia, changes occur especially in *muscular arteries and arterioles.*
- The changes are localized mostly in vessels of the *gastric mucosa, tongue, colon, gallbladder, urinary bladder, kidney*, and rarely in the small *intestine, myocardium and other organs*.
- The disease is observed in dogs.

Uremic Arteritis

- Macroscopically, the small arteries in the organs are gray-yellowish, showing thickening and bulging.
- Microscopically; **fibrinoid necrotic vasculitis** in media and adventitia, **neutrophil leukocyte** infiltrations in the intima.
- As a result of the lesions, uremic gastritis and **calcification** are seen in the stomach in relation to ischaemia.

Panarteritis nodosa (polyarteritis, or periarteritis nodosa)

- The term “polyarteritis nodosa” has been applied to a *heterogeneous group of arteritides*, which occur sporadically in all species of domestic animals.
- It develops as a result of immune reactions (antigen-antibodies).
- *Small and medium-sized **arteries** undergo severe necrotizing inflammation, often in a sharply segmental (nodose) pattern*, and with a predilection for branching points.
- As all layers of the arterial Wall are involved, the lesion is also referred to as “*panarteritis*”.
- Arterioles, capillaries, and venules are not involved, and glomerulonephritis is not present.

Panarteritis nodosa

- It's a chronic event.
- After streptococcal infections in humans,,
- Aleutian disease in pox,
- Erysipelas in pork,
- Rarely seen in cattle, cats and dogs.

Panarteritis nodosa

It develops in three phases;

- **Fibrinoid deg. and necrosis phase:** Degeneration of muscle cells and multiple leukocytes.
- **Reperative phase:** First, leukocytes, lymphocytes, histiocyte infiltrations are seen, then develops proliferative events in adventitia (periarteritis nodosa).
- **Recovery phase:** Connective tissue proliferaiton and and peri-arterial cell infiltration are observed in the media.

Panarteritis nodosa

Depending on the vascular changes,

- aneurysm in vessels,
- ulcer can develop.
- and vessels.
- It is more common in organs such as heart (coronary vessels), kidney, gastrointestinal system.

Venous Disease

Dilation of a vein

Enlargemant inner diameter of the veins and is divided into two main groups;

Phlebectasia: It is a widespread extension of the vein, *along all vessel*.

Varix-Varicose: A dilation on swelling of a vein. Dilatation *locally* and like a *pouch*.

- The most common expansion is observed in varix.
  - For example;
- *Varicocel* in the vena plexus of the funiculus spermaticus
- *Haemorrhoids* of haemorrhoidal plexus
- *Telangiectasia* in liver or in the small veins in the skin are called.

The reasons;

- Stagnation of blood flow,
- Hereditary or acquired weakness of the vena wall tissues,
- Vena porta thrombosis,
- Cirrhosis of the liver

- **In the case of phlebectasia,** by increasing the functional needs, the veins **are expanding totally.** In this case, some areas of the vena wall are thinned, and some areas become thicker as a result of tissue hyperplasia.

  Phlebectasia cases are seen *in vena mammaria* in cows, and *vena thoracica externa*, *vena caphena* and *vena spermatica* in horses. Enlarged veins may reach the thumb thickness.

  In the case of varix, because of **the local disorder** in the veins wall and the blood flow, veins form varix by *expanding into bags or pouches.*

- Thrombosis or sclerosis may be sequelae.
- The acquired portosystemic anastomoses noted above usually result from dilation of pre-existing microscopic venous anastomoses to produce collateral venous drainage.

- In humans, varix is very suitable for **ruptur**ing.

  Complete or incomplete rupturs of varixs can result in fatal **bleeding**.

  Varix in the legs of people, *chronic subcutaneous edema*, the skin under the *fibrous connective tissue* reproduction leads to the formation of **elephant leg (elephantiasis).**

- Varix and phlebectasie lesions are permanent for life.
- Varixs cause stasis and circulatory disorders in the region where they occur.
- Since the animal body is covered with hairs, varix and phlebectasies are hardly noticeable.

Phlebitis

- **A) Periphelebitis,**
- **B) Endophlebitis**

A) Periphlebitis

The beginning of the inflammation in the adventitia layer.

- **Periphlebitis serosa**,
- **Periphlebitis purulenta**,
- **Periphlebitis gangrenosa**,
- **Periphlebitis productiva.**

- B) Endophlebitis
- In the intima and media layers of the inflammation.

  - **Endophlebitis purulenta**,
  - **Thromboendophlebitis**,
  - **Endophlebitis productiva**.

Pathology of lymph vessels

Lymphangiectasie

- The expansion of the Lymph vessels lumen.
- The enlargement is mostly seen in the serous and subserous lymph vessels such as mesenterium, peritoneum, pleura and epicart.
- The main reasons are the common inflammation in the tissues and tumor formation.

Lymphangitis

- Lymphangitis serosa(simplex)
- Lymphangitis purulenta:
- Lymphangitis productiva: