Hematoxylin - eosin- Staining in a Dog Polyarteritys Nodosa

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Polyartetrtys nodosa (PAN) was diagnosed histologically in a dog 14 months old, that presented in the necropsy a hemorrhagic diathesis. The specific lesions were present in the muscular arteries of middle and small caliber and in the arterioles from heart. The modifications are segmentail, chronic, evolving from fibrinoid necrosis to the polyphasic, transmural, neutrophilic, leucocytodastic, macrophagic, lympho-plasmodtic and finally fibrotic vasculitis. The elastolisis predisposes to micro-aneurisms and micro-hemorrhages. Unlikethe perivascularitis, the inflammatory processis extended to the surrounding tissues.

Keywords: polyarteritis nodosa, dog, fibrinoid necrosis

Polyarteritis nodosa (PAN), or. panarteritis, periarteritis nodosa, Kussmaul disease is a condition of non-infectious primary medium caliber muscular arteries and small, peripheral or visceral lesions characterized by segmental and disseminated along multistage vessels, localized mainly in the bifurcation points. It is a complex disease process that coexists with acute injuries and chronic alternate with free wall. It is included in the group *collagen diseases* with pathogenesis (auto) immune [2, 8, 11, 13 - 17].

PAN succeed in developing multifocal fibrinoid necrosis of the media, transmural infiltration of inflammatory mononudear granulocyte followed by fibrosis and restorative adventitia invasion entire wall is associated with remote extension immune inflammation in the surrounding tissues [2, 3, 7, 8, 11, 13].

In etilogia PAN were indicted on several factors: HBV antigen, human, hypersensitivity to drugs (sulfonamides, penicillin, thiouracil etc.), infectious agents (PCM virus, the virus IEA, streptococci, staphylococci) [1] genetic factors. Experimental studies converge on the hypothesis of immunological pathogenesis that complex imine assets precipitated the endothelium or in the arterial wall and complexes formed *in situ (with circulating antibodies anti-cellular components)* initiates the activation of the complement system and triggers reactions and inflammatory yet however, the antigen responsible remain unknown [1, 4, 9, 19, 21].

The consequences are dependent thrombosis PAN acute or chronic ischemia followed by infarction in the kidney, spleen, lymph nodes, lungs, and nervous system, sometimes infarction, aneurysm and hemorrhage, secondary atrophy and fibrosis [6, 12].

Experimental part

Materials and metode

A cadaver dog aged 14 month post-mortem examination, showed the heart were observed thickening of blood vessels. Samples were taken from the heart which were fixed in 10% formalin, embedded in paraffin, sectioned at 6 microns and stained with HE method.

The samples preparation was carried out as follows: 24 h alcohol fixation at room temperature (prevent the tissue alteration due to the enzymes activity; preserve the tissue texture; improves the optical differentiation), alcohol dehydration (five steps: 70, 80, 90, 100% and 100% alcohol, each step for two hours), clearing with benzene, paraffin wax at 56°C, embedding tissues into paraffin blocks, trimming of paraffin blocks (6 μm), sections mounting on the glass slides (using Meyer albumin), hematoxylin - eosin -[22]. Staining was performed as follows: deparaffination of tissue sections in benzene, rehydration using decreasing concentrations of alcohol, rinsing in distilled water, hematoxylin staining, alcohol, eosin staining water removal using increasing concentrations of alcohol, cover slide mounting [22]. Hematoxylin will stain the nuclei in blue and the mucins in light blue. Eosin will stain the cytoplasm in pink, collagenin pale pink, red blood cells in bright red, and colloid in red. The microscopical examination is useful as differentiating diagnosis method only if chemical preparation of samples is applied [4, 5, 10, 22].

Results and discussions

A cadaver dog aged 14 months post-mortem examination showed the surface of the heart were observed thickening of blood vessels (fig. 1.)

PAN starts with edema with swollen endothelial intima and the subendothelial layer dilaceration. Plasma fibrin precipitation and average fibrinoid formation then the entire wall thickness, evolving into fibrinoid necrosis (fig. 2.)

Chemotactic properties of fibrin and fibrinoid induce massive infiltration with neutrophilic and eosinophilic

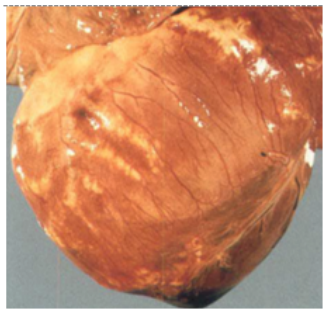

Fig. 1.Periarteritis nodosa. Yellow-white nodular thickening of subepicardial branches of coronary arteries Dog

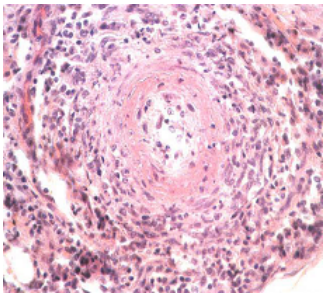

Fig.2.Periarteritis nodosa. Fibrinoid necrosis of the media of an artery. Infiltration of neutrophils and mononuclear cells predominantly in the adventitia. Dog. HE

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granulocytes wall and perivascular tissue. After phagocytic fibrin, granulocytes degenerates, their nuclear fragments, creating the appearance of *leukocytoclastic vasculitis* acute. Proteases released soaking produce arterial wall, followed by saciform expansion (true aneurysm) [15, 17].

Chronic form develops in connective tissue rich in collagen fibrosis scars wall making a deposit and fibers form concentric layers around the arteries and away in neighboring tissues, segmental artery thickening moniliformi forming nodules (hence the name *arteritis nodosa*) is the only Long Island Sound until vasculitis true distinguishing them from other inflammatory processes, there is no pressure fibrinoid necrosis or cellular invasion of the wall material, the inflammatory process is restricted only to the adventitia (perivascular) or only the intimate (endovasculitis) [7, 14, 21].

Conclussions

Polyarteritis (PAN) was diagnosed histologically a dog aged 14 months who presented necropsy bleeding diathesis and thickening heart sinuo surface.

Specific lesions were present in the muscular arteries of medium caliber.

Changes are segmental disseminated along the vessels, fibrinoid necrosis of the media of an artery. Infiltration of neutrophils and mononuclear cells predominantly in the adventitia.

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