# MORPHOPATHOLOGICALL QUANTIFICATION OF UNINFLAMMATORY PNEUMOPATHIES IN DOGS

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***Abstract:*** *Uninflammatory pneumopathies were microscopically and macrospically diagnosed in 22 cases (73.33%) out of a total of 30 necropsied dogs. A chronic evolution of the essential alveolar emphysema was morphopathologically diagnosed in 3 cases (13,63%). Macroscopically, the lungs were enlarged, with a tense pleura, pale, crepitant with a negative docimasy while microscopically, we could enhance the accentuated distension of the alveoli which presented flat pneumocytes and were accompanied by the rupture of the alveolar walls. Thrombosis of the pulmonary vein was noticed in one case (4,54%), with parietal thrombi present, microscopically, in the lumen of the vessel. A pulmonary red infarction was also diagnosed in one case (4,54%). Macroscopically, at inspection and section, we notice compact dense, dark-red areas, of triangular shape, with the base headed towards the pleura and the top pointed towards the pulmonary parenchyma, towards the obstructed vessel. Microscopically, a hemorrhagic infiltrate could be noticed in the area of necrosis and of coagulation of the pulmonary parenchyma.*
*In 7 cases (31,81%) we have set the diagnosis of active pulmonary congestion. Microscopically we could notice the ectasis of the alveolar capillaries and the congestion of the blood vessels, as well as the presence of well-individualized and intensely stained erythrocytes in the lumen. The passive congestion was identified in 3 cases (13,63%) and it was morphologically expressed through the presence in the capillary lumen and in the lumen of the blood vessels, of glued erythrocytes, accompanied by hemolytic phenomena, septal, mezenchymal reactions, reduction of the respiratory space and beginning of lung sclerosis. The pulmonary edema was morphpathologically identified in six cases (27,27%). Two cases (9,09%) had stasis pulmonary edema microscopically expressed through dilation of the pulmonary alveoli caused by transudate and ectasis of the alveolar capillaries; four cases (18,18%) were diagnosed with inflammatory pulmonary edema-showing leukocytes in the edema liquid and in the lumen of the alveoli and of the bronchi as well as capillary ectasis and desquamated endothelial cells. In one case, (4,54%) with pulmonary dirofilariosis, a calcification of the aorta media could be noticed and microscopically, the disorganization of the media through dilacerations and fragmentation of the elastic fibers, alongside precipitated calcium granules, could be distinguished.*

***Keywords****: circulatory pneumopathies, morphopathologicall, dog*

The modifications of the pulmonary circulation in dogs are exteriorized through passive and active congestions, pulmonary hemorrhages, thrombosis of the pulmonary vein, infarctions and pulmonary edema. They are the consequence of the singular or combined action of biotic and abiotic factors which directly or indirectly affect the respiratory apparatus (1, 5, 7, 10). The congestion or the pulmonary hyperemia presents itself differently from a morphopathological point of view, depending on the affected circulatory segment, arterial or venous. Thus, we mention the active or artherial congestion and the venous or passive congestion as well as the hypostatic pulmonary congestion. The pulmonary hemorrhages, from a morphoclinical point of view, may be intratisular, intraluminal and exteriorized (4, 9, 10). The pulmonary infarction micro-and macroscopically presents itself as a red infarction (hemorrhagic necrosis), frequently located in the diaphragmatic lobes (9).

The pulmonary edema may be a stasis edema in right heart failure, inflammatory edema in infectious diseases (tetanus, monocytic ehrlichiosis of dogs, etc.), toxic edema (intoxications with antifreeze, herbicides, etc.) (2, 5, 6, 8).

## MATERIALS AND METHODS

The research of the present study was conducted during the period October 2013-May 2015, through anatomopathological examination of lungs coming from 30 dog corpses of different ages, sexes and breeds, belonging to private owners, kennels or animal protection associations. The corpses were necropsied at the Veterinary Medicine Faculty in Timisoara, at Forensics.

The bodies were places in dorsal position and then necropsied according to the technique used for this species.

After a detailed examination of the lung, trachea and tracheobronchial lymph node, samples were taken for a histopathological exam. Subsequently, the pieces were prepared for the paraffin method. The obtained blocks were sectioned using a microtome at 6 micrometers and the sections were stained using the trichromatic method- hematoxylin eosin methylene blue (HEA) in order to enhance the modified structures. After staining, the sections were dehydrated and mounted in an anhydrous environment using Canada balm.

The histopathological preparations thus obtained were examined and photographed using an Olympus CX41 microscope.

## RESULTS AND DISCUSSIONS

*Exterior exam.* The bodies were in mediocre towards good shape, the skin lacked elasticity, and they presented enophtalmos, anemia and/or cyanosis of the apparent mucosae and skin. The fat tissue and the skeleton muscles were more or less developed, depending on the age and maintenance status.

*Interior exam.* The presence of a serosanguinolent liquid in variable quantities, in the thoracic cavity was noticed in 6 cases (80-120ml).

*Lung exam.* The research regarding the pulmonary lesions was systemized in two groups: volumetric pneumopathies and circulatory pneumopathies (Table 1, Fig.1.).

Three cases which were dogs aged 9, 11 and 14 years, presented enlarged lungs, with a tense pleura, pale color, crepitant and with negative docimasy. Microscopically, and accentuated distension of the alveoli could be noticed, with flat pneumocytes, accompanied by the rupture of the alveoli walls-essential alveolar pulmonary emphysema with a chronic evolution (Fig. 2.).

One case, the corpse of a three year old dog, was identified with thrombosis of the pulmonary vein which is a circulatory modification characterized by the formation of intravascular clots, during lifetime, due to blood constituents. Parietal thrombi were observed in the lumen of the vein. Macroscopically, on inspection, the affected venous segment is more dilated, presenting a dark red color and high consistency. On longitudinal section, the thrombus was adherent to the wall, dry, harsh, friable, and on transversal section the vascular lumen occupied by the thrombus could be seen.

Microscopically, the thrombus had an elongated shape, represented by a fibrin clot, red blood cells, leukocytes and altered platelets. It was adherent to the venal wall (Fig. 3.). The

parietal thrombi produce ischemia of different degrees which, if not compensated by the collateral circulation, causes the appearance of serious processes, infarctions or gangrenes.

The red pulmonary infarction was diagnosed in one of the cases, the body of a five year old dog. Macroscopically on inspection and on section, compact dark red areas could be noticed. They were dense, of triangular shape with the base towards the pleura and the top in the pulmonary parenchyma, towards the obstructed vessel. Microscopically, hemorrhagic infiltrates could be observed, with a triangular aspect in the area of pulmonary necrosis (Fig. 4.). In seven cases, macroscopically, the lungs were enlarged in volume and weight, with a tense pleura and on inspection and section, the pulmonary parenchyma was intense red, with a moist aspect, pasty consistency and negative docimasy-active pulmonary congestion (Fig. 5.). Microscopically, we noticed ectasis of the alveolar capillaries and congestion of the interstitial blood vessels with the presence of well individualized and intensely colored erythrocytes in their lumen-active pulmonary congestion (Fig. 6.).

Three cases, the body of a 14 month old, a 15 month old and of 1.7 year old dogs, were diagnosed with passive congestion. Microscopically, glued erythrocytes were noticed, in the lumen of the capillaries and blood vessels, alongside hemolytic phenomena, septal mezenchymal reactions, reduction of the respiratory space and pulmonary sclerosis.

In two cases, macroscopically, the lungs were enlarged in volume, of pink-yellowish color, expressing a foamy liquid on section, similar to whisked egg white. The same foamy liquid was seen in the airways (trachea and bronchi). Microscopically, we notice the dilation of the alveoli due to transudate and more or less accentuated hyperemia of the alveolar capillaries-pulmonary stasis edema (Fig. 7.).

In four cases, macroscopically the lungs were enlarged in volume, the pleura was tense, transparent, the parenchyma had a dark red color and on section, we noticed the same color and the presence of a foamy, dark red liquid (serous exudate) in the parenchyma, bronchi and trachea. The docimasy was between waters- inflammatory pulmonary edema (serous bronchopneumonia) (Fig. 8.). Microscopically, in the interstitium of the pulmonary lobules, we noticed an oxyphile mass that represents the edema liquid. In the lumen of the alveoli and bronchi we noticed the presence of leukocytes in the edema liquid which appears as a homogenous oxyphile mass. These morphological modifications define, in a histopathological plan, the inflammatory pulmonary edema (Fig. 9.).

The calcification of the aorta was signaled in one case- a 13 year old dog with cardio-pulmonary dirofilaria. Macroscopically, the wall of the aorta is rigid and the consistency is rough and highly friable. On section, in the media of the artery, we noticed the presence of white, hard grains and a reduction of the lumen. Microscopically, necrosis is enhanced and the desquamation of the endothelial cells, disorganization of the media due to dilaceration and fragmentation of the elastic fibers accompanied by precipitation of oxyphile calcium grains in the HEA staining method (Fig. 10.).

Table 1

Quantification of uninflammatory pneumopathies

| Nr. Crt | Necropsied dogs | **Uninflammatory pneumopathies** | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| 1. | 30 | Volumetric | | Circulatory | | | | Dystrophic | % |
| | | Atelectasis | Emphysema | Congestion | Edema | Trombosis | Infarct | Calcification | 73,33% |
| | | - | 3 (13,63%) | 10 (45,45%) | 6 (27,27%) | 1 (4,54%) | 1 (4,54%) | 1 (4,54%) | |

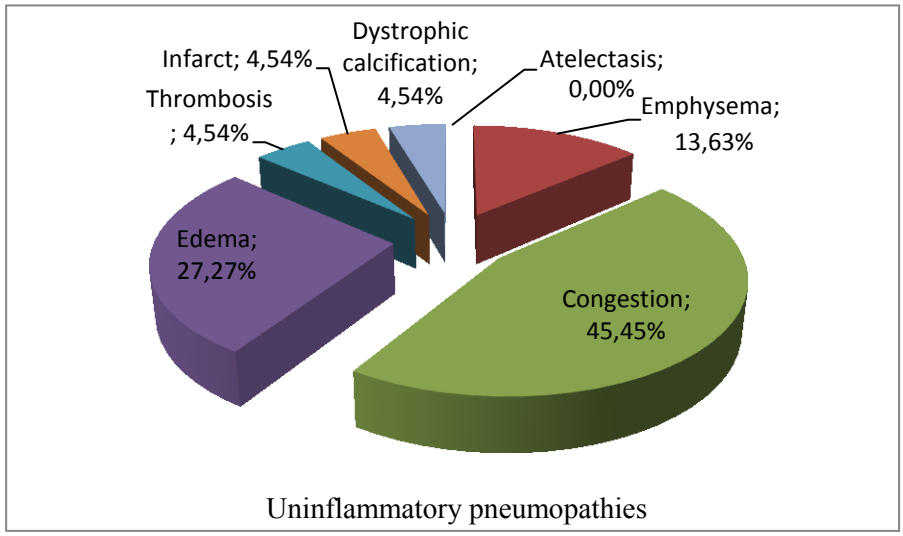


Fig. 1. Graphical representation of uninflammatory pneumopathies in dogs.

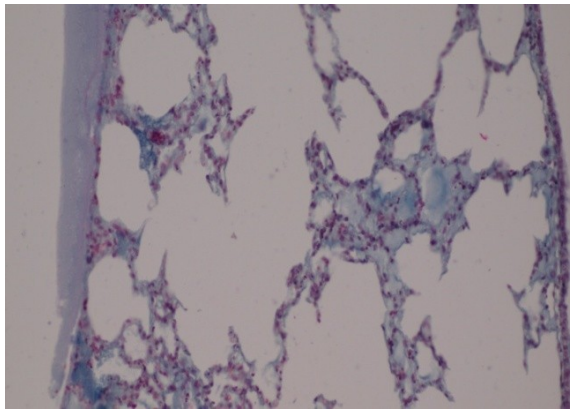

Fig. 2. Accentuated distension of the alveoli, with flat pneumocytes, accompanied by the rupture of the alveoli walls - essential alveolar pulmonary emphysema. Col. HEA x20

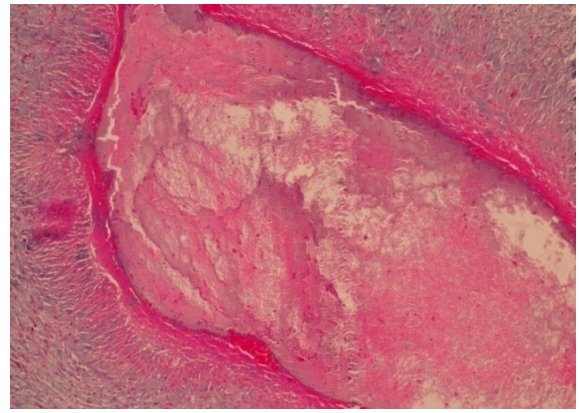

Fig. 3. Pulmonary vein – venous parietal thrombus. Col. HEA x10

Fig.4. Red pulmonary infarction - hemorrhagic infiltrates with a triangular aspect in the area of pulmonary coagulation necrosis. Col. HEA x4

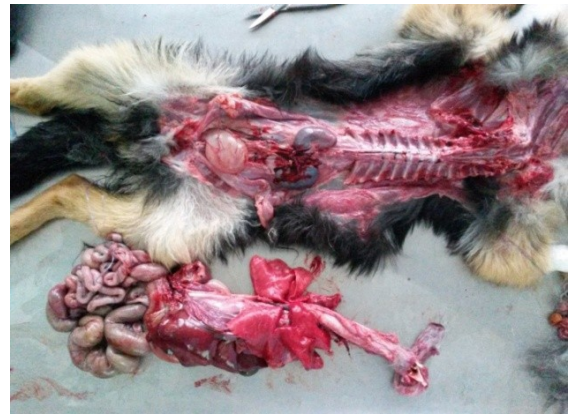

Fig. 5. Dog corpse: active pulmonary congestion.

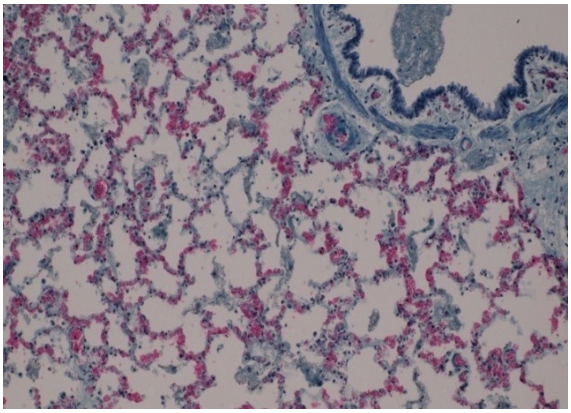

Fig. 6. Active pulmonary congestion : ectasis of the alveolar capillaries with the presence of well individualized and intensely colored erythrocytes . Col. HEA x 20.

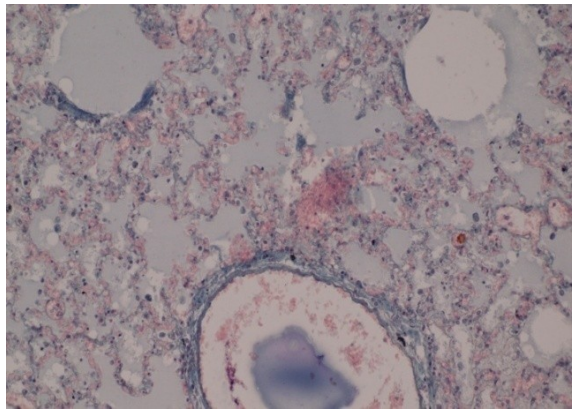

Fig. 7. Pulmonary stasis edema: the presence of edema fluid, basophil, in the lumen of the alveoli, bronchi and trachea. Col. HEA x 20.

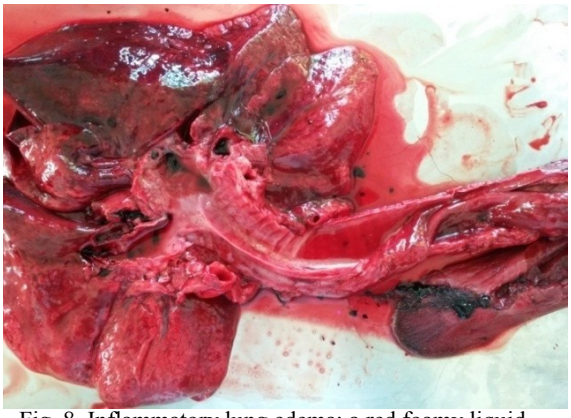

Fig. 8. Inflammatory lung edema: a red foamy liquid in the tracheal lumen, bronchi and lung parenchyma.

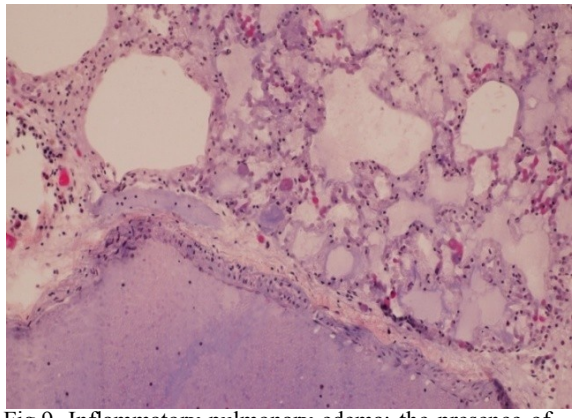

Fig.9. Inflammatory pulmonary edema: the presence of oxyphile mass that represents the edema liquid in the lumen of the alveoli and bronchi. Col. HEA x 20.

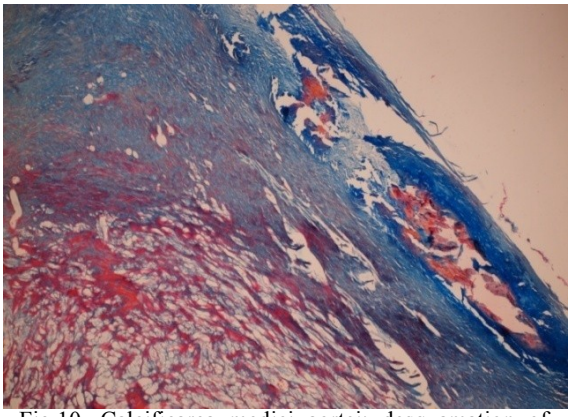

Fig.10. Calcificarea mediei aortei: desquamation of the endothelial cells, disorganization of the media, dilaceration and fragmentation of the elastic fibers, presence of oxyphile calcium grains. Col. HEAx4

## CONCLUSIONS

Out of a total of 30 necropsied dog corpses, 22 cases were morphopathologically diagnosed with uninflammatory pneumopathies (73,33%).

The following were diagnosed morphopathologically: volumetric pneumopathies, essential alveolar emphysema with a chronic evolution in three cases (13,63%); circulatory pneumopathies- thrombosis of the pulmonary vein, one case (4,54%), red pulmonary infarction, one case (4,54%), active pulmonary congestion (31,81%), passive pulmonary congestion (13,63%); stasis pulmonary edema, two cases (9,09%), inflammatory pulmonary edema-four cases (18,18%). Dystrophic calcification of the aorta media was signaled in one case which presented cardio-pulmonary dirofilaria.

Out of the circulatory pneumopathies, the most numerous were the active and passive congestions followed by the stasis edema and inflammations.

The dystrophic pneumopathies translated through calcifications of the aorta media were frequently signaled in dogs aged over 9 years.

The uninflammatory pneumopathies expressed through vascular thrombosis, infarctions, congestions, edema and pathological calcifications, which were macro and microscopically diagnosed in the examined cases, correspond to the pneumopathies signaled in the consulted specialty literature (4, 8, 9, 10).

In this paper, we made a prevalence of the uninflammatory pneumopathies of the cases taken into study, respecting the morphopathological presentation criteria of the fundamental pathological processes unanimously acknowledged in our country and abroad.

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