A CASE OF MIXED GRANULOMATOUS INFLAMMATION IN A TURKEY HEN

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Abstract
Granulomatous inflammation may be caused in birds either by various infectious diseases (with the formation of Langhans type giant cells) or by the presence in different tissues and organs of foreign bodies (with the formation of Muller type giant cells), the most common being uric acid crystals in gout. We examined the cadaver of a two year old turkey hen that had multiple yellowish-white nodular formations located in the liver. Histopathological examination showed that these were formed mainly through an infectious type granulomatous inflammation caused by a chronic colibacillosis with the presence of bacterial colonies and necrotic debris in the center and Langhans giant cells with foamy cytoplasm. The liver also had early stage foreign body granulomas caused by the precipitation of uric acid crystals in the parenchyma. We were then able to establish that the bird suffered from both coligranulomatosis and also visceral gout, as a consequence of improper microclimate and feeding parameters. It is uncommon to find both types of granulomatous inflammations in the same patient or even in the same organ. The tophy gout had not yet started to form Muller type giant cells, but still, the architecture of the granulomas was clear enough to be compared to the adiacent infectious type one.
Key words: *chronic colibacillosis, gout, granulomatous inflammation*

Introduction

Granulomatous diseases in poultry may be caused by multiple and various factors. First of all, granulomatous inflammation may evolve as a reaction of the immune system towards an infectious agent or a foreign body. Amongst the first there are mostly bacteria (*Salmonella pullorum, Mycobacterium tuberculosis, Escherichia colli, Pasteurella multocida, Staphylococcus aureus, Streptococcus spp., Eubacterium tortuosum)* and parasites *(Tetratrichomonas gallinarum, Histomonas meleagridis,*) that may cause this type of lesion, but also some species of fungi *(Aspergillus fumigatus*) (Landman, 2017; Supartika, 2006; Paul, 2005).

Coligranulomatosis (also known as Hjarre's disease) appears when the immune system cannot eliminate the bacteria and so it chooses to encapsulate it in a focal lesion that is separated from the normal, healthy tissue through several layers of different cell populations. Granulomas may develop in the intestinal tract and liver. A histological characteristic of the coligranuloma is the particular aspect of the giant cells found in direct contact with the bacterial colony and the caseous necrotic debris. They are elongated cells, with a foamy cytoplasm and the nuclei located opposite from the center of the lesion (Zachary, 2017; Paul, 2005; Coțofan, 1992;).

The foreign body granulomatous type reaction is mostly caused by visceral gout, a dysmetabolic syndrome caused by the accumulation of urate crystals in various tissues, triggering a proliferative mesenchymal reaction. The main histological characteristic of this type of lesion is the aspect of the giant cells (hundreds of nuclei located opposite from the foreign body, irregular shape that follows the contour of the foreign body) and their display as a rosette around the urate deposit, in order to minimise the irritative action on the healthy tissue (Paul, 2001).

Material and methods

We received the cadaver of a turkey hen, two years of age, that came from a small farm. The necropsy exam showed multiple yellowish-white nodular formations in the liver. Tissue fragments were harvested and fixated in a 10% formaldehyde solution. After this they were included in paraffin, sectioned at 5 μm and stained following the Masson's trichrome method.

Histopathological slides were observed using a Leica DM 750 optical microscope.

Results and discussion

Histopathological examination showed that the nodular formations were formed mainly through an infectious type granulomatous inflammation caused by a chronic colibacillosis with the presence of bacterial colonies and necrotic debris in the centre of the formations and Langhans giant cells with foamy cytoplasm located on the margins of this area (Fig. 1, 2).

However, the liver also had early stage foreign body granulomas caused by the precipitation of uric acid crystals in the parenchyma (Fig. 3, 4). We were then able to establish that the bird suffered from both coligranulomatosis and visceral gout, the later one being a consequence of improper microclimate and feeding parameters (hyperproteic feed, lacking drinking water, vitamin A deficiency, kidney dysplasia) (Coțofan, 1992).

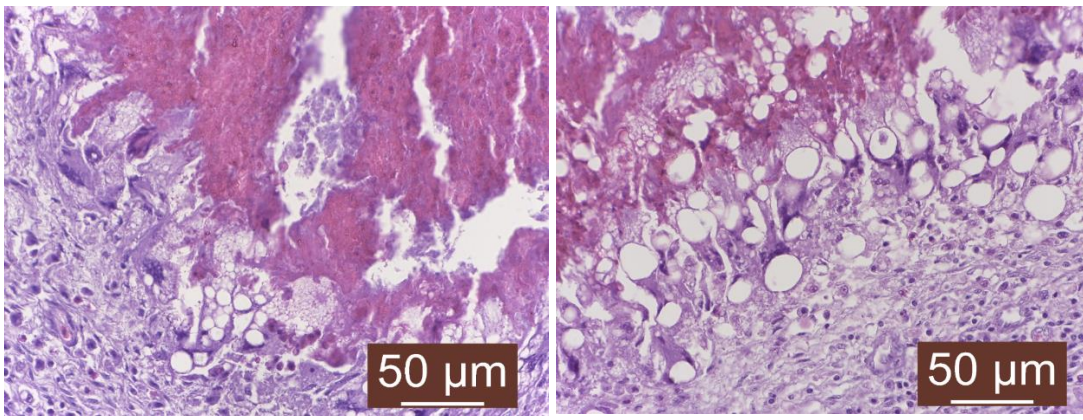

Fig. 1, 2 Coligranuloma in the liver of a turkey hen. Caseous necrotic debris and giant cells with foamy cytoplasm. Masson trichrome stain

It is uncommon to find both types of granulomatous inflammations in the same patient or even in the same organ. The tophy gout had not yet started to induce the formation of mature Muller type giant cells (dark rim of nuclei located opposite from the foreign body), but still, the architecture of the granulomas was clear enough to be compared to the adjacent infectious type one (Landman, 2017; Supartika, 2006).

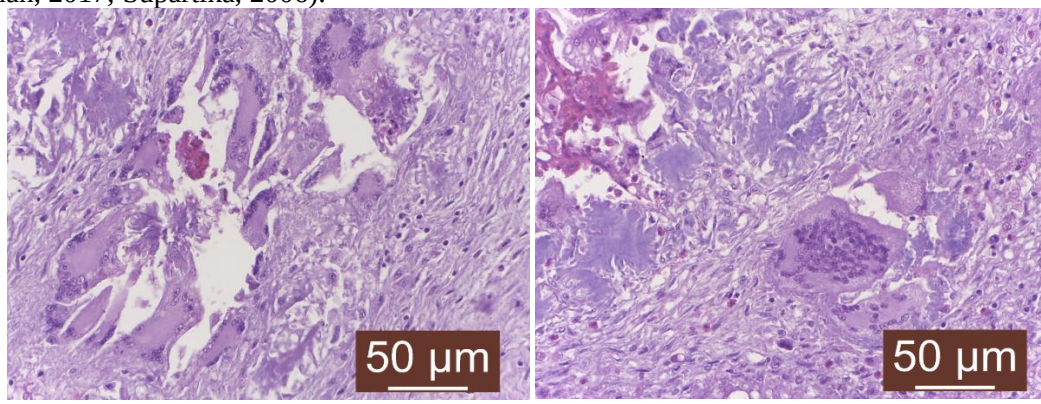

Fig. 3, 4 Tophy gout with young Muller type giant cells forming arround uric acid deposits, adiacent to a coligranuloma. Turkey hen. Masson trichrome stain

The number of the nuclei was much higher and also the aspect of the cytoplasm varied between the two giant cell types. The shape of the cells corresponded with the literature descriptions for these types of lesions, thus facilitating the indentification (Pașca, 2016).

Conclusions

Even though the literature tends to describe lesions in a separate, scholastic manner, it is not uncommon to find many lesions of different types evolving at the same time in the same organ or tissue. In this case, we were able to observe two types of granulomatous inflammation, caused completely different etiological factors, developing within the same macroscopical lesion in the same liver.

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