

# Investigations into feather abnormalities in racing pigeons

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## Summary

The present study reports cases of feather abnormalities in young racing pigeons from four different lofts. The pigeons from the first three sources were characterized by among other things loss of feathers, stunted feathers, vane discoloration, feather dystrophy. In pigeons from the fourth loft, vanes were unevenly developed, while the shafts in wing feathers were narrow and had an hourglass appearance. Investigation revealed a history of pox, vaccination and deworming in some cases. Clinical, microbiological, parasitological examinations, hematology, PCR assays, necropsy and histopathology were performed. The presence of *Eimeria* spp., *Trichomonas* spp., and single *Columbicola columbae* were demonstrated. Pigeon circovirus (PiCV) DNA was confirmed in tissues and affected feathers of pigeons from the first three sources. Histopathology revealed the lymphoid depletion and the presence of the pathognomonic inclusion bodies in the bursa of Fabricius. In the fourth loft, a paramyxovirus infection (APMV-1) was confirmed by RT-PCR. The microscopic lesions in the feathers were similar, despite their different macroscopic appearance compared to previous cases. It seems that feather abnormalities may be the effect of a combination of several factors (infectious and non-infectious).

**Keywords:** feather abnormalities, loss of feathers, racing pigeon

Feather diseases have a serious negative impact on the racing pigeon sport (competition flights) and cause losses. In general, feather abnormalities are commonly observed in pigeons; however the cause of problem is often difficult to identify, and requires the use of several diagnostic tools. Some of pigeon diseases are only accompanied by non-specific changes in plumage, including ruffled, dull and disheveled feathers, the presence of stress marks on feathers, dirty, discolored or mechanically damaged feathers. Feather lesions may be caused by variety of infectious or non-infectious factors. Infectious causes of skin and feather abnormalities may include: e.g. external parasites (mites, lice), bacterial (chronic salmonellosis), fungal or *Trichomonas* spp. infections (6). In the literature there is relatively little information indicating viral infections of the the order *Columbiformes* as one of the causes of abnormal plumage in birds (3, 8, 13). Feather disorders accompanying the most common pigeon viruses (pigeon circovirus – PiCV, pigeon paramyxovirus serotype 1 – PPMV-1) were not typically observed in pigeons. In the skin form of pigeon pox, birds develop scabby

proliferations (pocks), especially on areas of the head, legs and body that contain no feathers; nevertheless generalized lesions on the feathered skin may also be noted (6, 8, 14, 16). Non-infectious causes of feather abnormalities may include genetics, nutritional causes, mechanical factors, medicaments (e.g. fenbendazole toxicosis, corticosteroids) (5, 6).

The purpose of this study was to describe feather abnormalities in racing pigeons from four different lofts in Poland.

## Case descriptions

**Case 1.** Poor flying results and abnormal feather growth were noted in a flock of pigeons (10%, 8/80). Two young pigeons (in their first months of life) were submitted in June for investigation. Most birds in the loft came from the owner's breeding, but these two were purchased from other breeder. The pigeons were vaccinated against paramyxovirus, salmonellosis and mycoplasmosis. They were fed with standard pigeon diet (Ovigor, Agrol). In autumn, a pigeon pox outbreak was noted in the loft, but it remained unknown whether the affected pigeons had suffered from

pox. In spring, pigeons showed missing feathers initially on the crop and, subsequently, on the nape of the neck. The vitamin supplementation, antibiotic therapies were ineffective and intensified breaking feathers.

**Case 2.** (Provided by Leszek Szczepańczyk, DVM, Pszczyna.) Feather abnormalities were reported in only two racing pigeons from the second brood which had been kept in an aviary. One pigeon (approx. 5 weeks old) was submitted in August for investigations. The birds had not been previously dewormed or vaccinated, and they did not participate in races. Total breeding consisted of 100 birds, including 9 couples of varied origin. The pigeons with feather abnormalities were the offspring of two couples: a German male and a Polish female, and a German male and a Belgian female. The parents had undergone treatment for trichomoniasis before mating and they were dewormed with albendazole (Valbazen 10%, Pfizer).

**Case 3.** Feather abnormalities were observed in three racing pigeons, two of them (approx. 5 months old) were submitted in May for investigations. Changes in plumage had not been previously noted in the loft. Several new pigeons had been purchased and introduced for the breeding one year earlier. All birds in the loft were regularly vaccinated against pigeon pox, paramyxovirus, herpesvirus, salmonellosis and mycoplasmosis.

**Case 4.** Feather abnormalities were noted in nearly 100% of birds from the first brood at around 3 months of age, before weaning. The flock had been dewormed with oxfendazole (Systamex, Vetoquinol Biowet), but not vaccinated against common pigeon diseases. Two birds were submitted in April for investigations. New feather generation was normal or with shorter feathers.

## Material and methods

**Clinical examination.** A total of 7 pigeons were investigated. Clinical examination included the estimation of the body condition, general health status, behavior, skin, plumage status, joints and cloacal region. Crop swabs were analyzed for the presence of *Trichomonas columbae* according to the following scale: 0 – absence of parasites, + individual parasites, 1-3 per field of view, ++ average number of parasites, +++ numerous parasites, ++++ extremely numerous parasites. Parasitological examination of faecal samples involved a 4-point scale (according to the following scale: no invasion; mild invasion – up to 10 invasive forms; moderate – up to 20 invasive forms; massive invasion – uncountable number of eggs or oocysts). Specimens were viewed under a light microscope at 10 × total magnification with a 10 × ocular lens in 10 fields. Blood samples were collected for hematology in accordance with the method described in the literature (1).

**Molecular analysis.** PCR technique was used to detect the presence of genetic material of pigeon circovirus (PiCV). DNA was extracted from the feathers (quill of 3-5 feathers) with 5% Chelex (Bio-Rad Laboratories, Canada) and from the bursa, liver and spleen with a Sherlock kit (A&A Biotechnology, Poland) in accordance to the manufacturer's instructions. The reverse transcription PCR (RT-PCR) assay was used for detection of avian paramyxovirus serotype 1 (APMV-1) (2). Viral RNA was extracted from the brain and

kidney samples with QIAamp Viral RNA Mini Kit (Qiagen, Germany). Reverse transcription was performed with the use of the RevertAid First Strand Synthesis Kit (Thermo Fisher Scientific Inc., USA). Additionally, DNA samples from tissues were used for detection of herpesvirus (PiHV) and pigeon adenovirus (PiAdV) by PCR. Amplification primers and reaction conditions were obtained from the literature (2, 15, 16, 25).

**Necropsy and histopathological examination.** Tissue samples (liver, spleen, kidneys, lungs, heart, bursa of Fabricius, brain, pancreas and duodenum) and changed skin samples from 7 birds were collected for histopathological analysis. Paraffin sections were stained with hematoxylin and eosin (H-E). In some samples, additional staining methods were used, i.e. Ziehl-Neelsen (Z-N) for acid fast bacilli and periodic acid-Schiff (PAS) staining of fungi.

**Microbiological examination.** Cloacal swabs and tissue samples (liver, spleen) were cultured on standard bacterial growth media, selective and differential media (MacConkey's agar, SS agar, SF agar) and on Sabouraud agar.

## Results and discussion

**Feather abnormalities.** Case 1. Thickening of the feather pulp, feather deformation and loss of feathers on the nape of the neck were observed. The affected feathers were ruffled and had a greasy appearance (Fig. 1). Case 2. Significant symmetrical loss of feathers (mainly remiges) in both wings, loss of tail feathers, feather growth retardation, stunted feathers and quill congestion were observed (Fig. 2). Case 3. Vane discoloration in wing feathers was reported. The most advanced lesions with significant loss of plumage on the head and neck and abnormal feathers were noted in one pigeon (Fig. 3). Case 4. Loss of wing feathers, feather dystrophy and retarded vane development were observed. The rachis was soft, filled with blood, and narrow. Some feathers had a normal appearance along one-half to two-thirds of their normal length, but they were pinched off in distal parts (Fig. 4).



**Fig. 1. Case 1. Feather abnormalities on the base of the neck (arrow) in a racing pigeon**

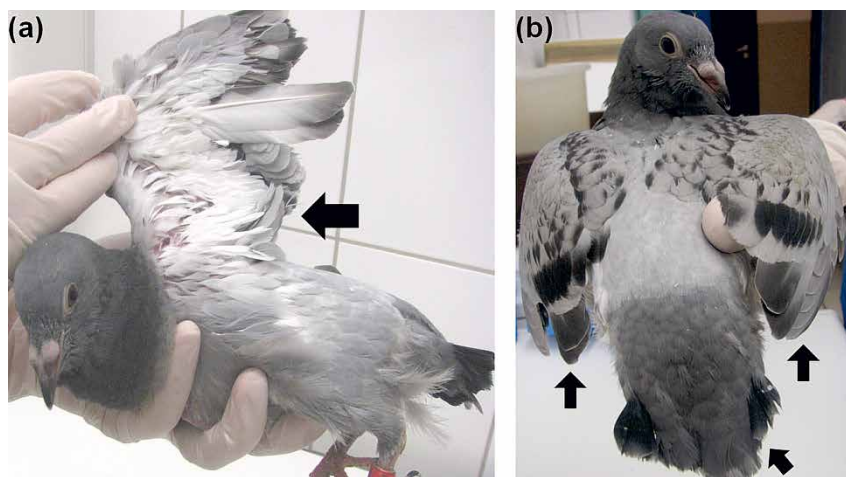


Fig. 2. Case 2. Significant symmetric loss of remiges in both wings, uneven feather growth (2a, arrow), and loss of wing and tail feathers (2b arrows) in a racing pigeon



Fig. 3. Case 3. Feather loss and feather dystrophy (arrows), vane discoloration in wing feathers (asterisk) in a racing pigeon

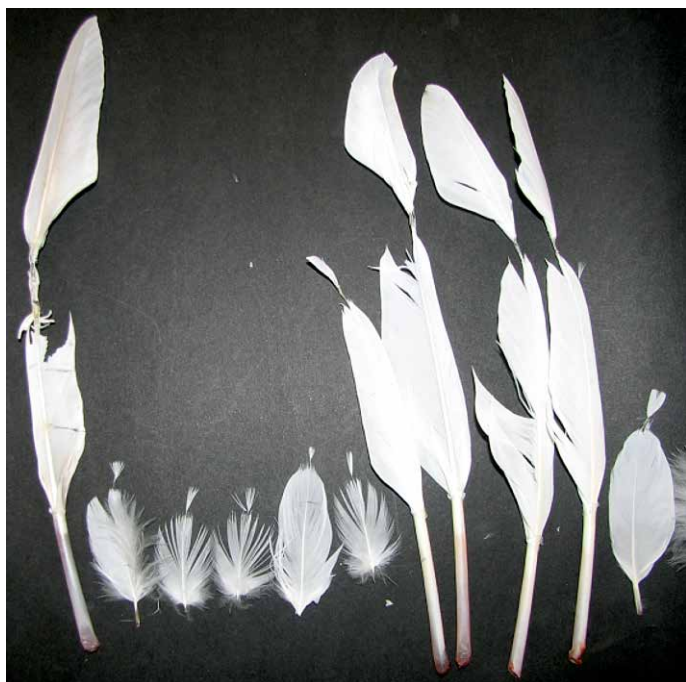


Fig. 4. Case 4. Feather abnormalities with a characteristic hourglass appearance in racing pigeons. Feathers showed pinched off distal parts and the regrowth of basal parts

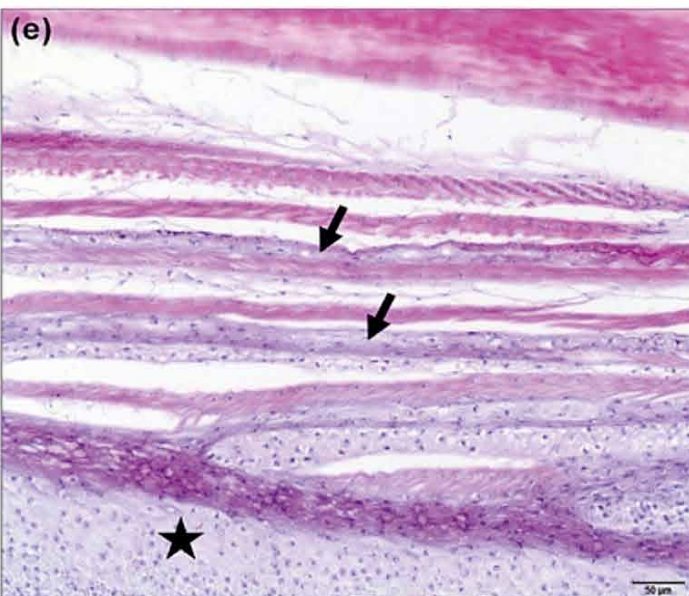
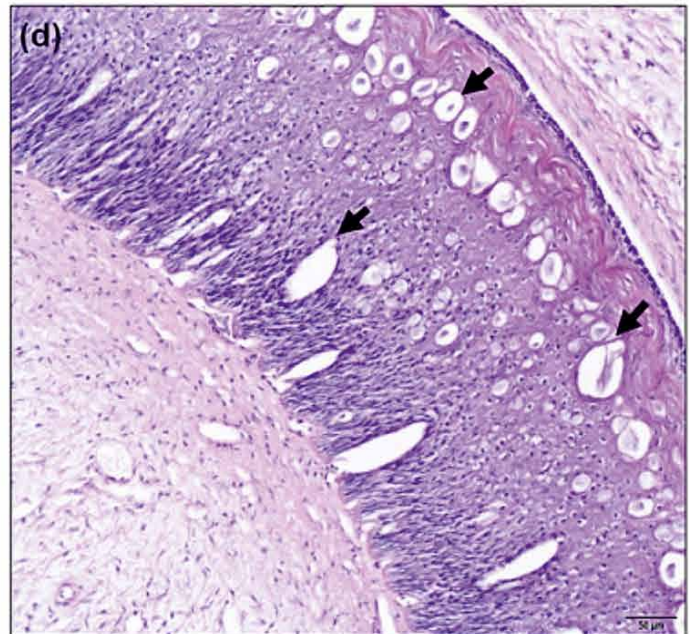
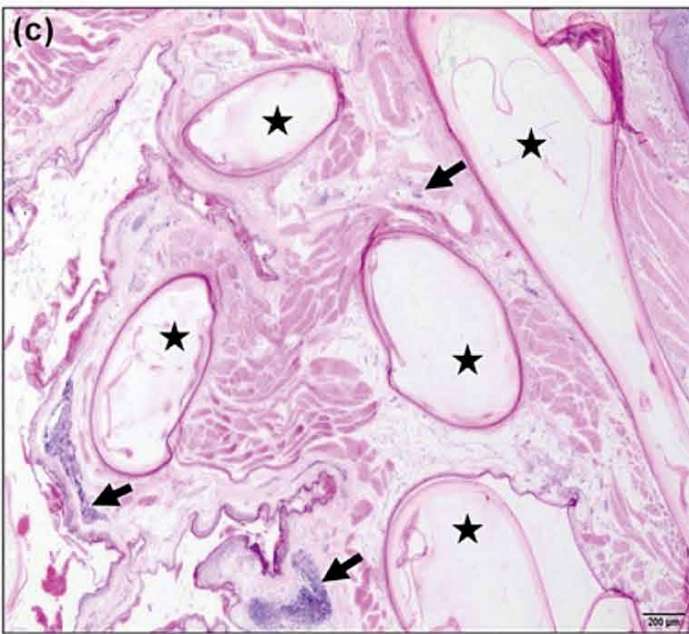
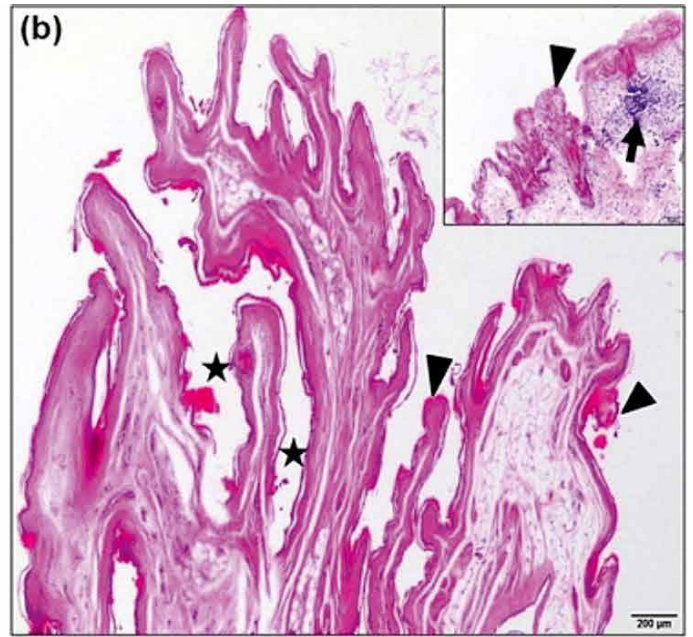
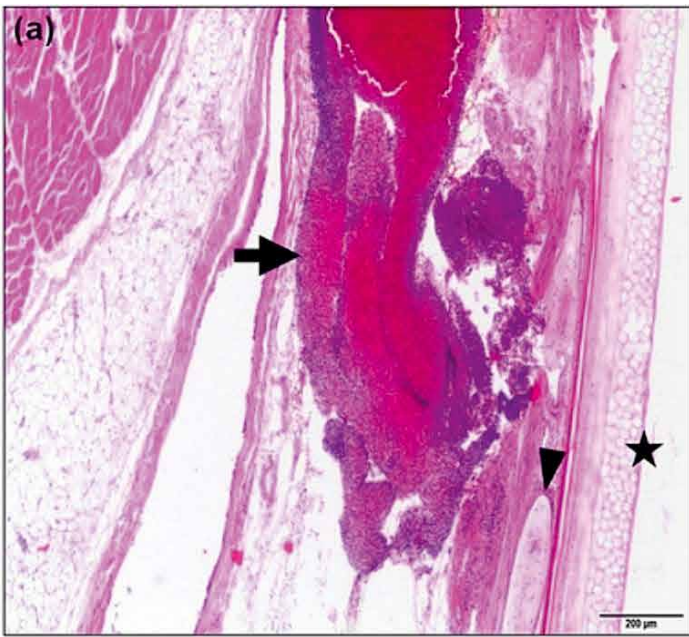
**Clinical examination.** The examined pigeons did not show symptoms of infectious diseases, except for one bird (case 2) in which watery diarrhea, depression and frequent sitting on the hocks were observed. Sternal deformity and disproportion were noted in one bird (case 3). Nutritional status and body condition were evaluated as good in all pigeons (mean body mass was 368.7 g). *Trichomonas* spp. was not detected in case 3, whereas single (case 1), numerous (case 2) and extremely numerous (case 4) trichomonads were detected in the remaining pigeons. Parasitological analyses did not reveal intestinal parasites or oocysts of *Eimeria* spp. (case 4). Single oocysts were reported in pigeons from case 2 and case 3,

numerous oocysts (++) in case 1. Single *Columbicola columbae* lice were reported in case 3. All of the evaluated birds were free of quill mites. The average values of hematological parameters were determined as follows: RBC  $3.0 \times 10^{12}/l$ , Ht 41.8%, Hb 13.2 g/dl, MCV131.9 fl, MCH 42.1 pg, MCHC 32.5 pg, WBC  $13.3 \times 10^9/l$ , lymphocytes 68.5%, heterophils 26.1%, eosinophils 0.1, basophils 1.3, monocytes 4.0%.

**Molecular analysis.** In pigeons of cases 1, 2 and 3 the presence of PiCV genetic material was found in feather, bursa of Fabricius and spleen. The positive result for APMV-1 (brain and kidney samples) was obtained only in pigeons from case 4. Case 4 was PCR negative for PiCV, including in the feather sample. All of the tested birds were free of the PiHV and PiAdV.

**Necropsy and histopathology.** Necropsy of case 1 pigeons revealed oval, soft, white and gray foci with pulpous consistency associated with subcutaneous tissue on the neck. A histopathological analysis of the skin revealed a granulomatous inflammation with multinucleated giant cells and central necrosis in the feather pulp, and hyperkeratosis of the feather sheath. The Z-N and PAS stains were negative. Marked depletion of lymphoid tissue associated with the presence of intracytoplasmic inclusion bodies in the bursa of Fabricius were observed.

In the pigeon of case 2 we observed significant splenomegaly. The liver, kidneys and pancreas were mildly enlarged in size. A microscopic analysis revealed massive hemorrhages and *panniculitis* in the region of the quills and sensory receptors (Fig. 5a), as well as loss of quills. The epidermis shows papillomatous hyperplasia with mild hyperkeratosis and small clusters of mononuclear cells (Fig. 5b). Moderate lymphoid depletion in the spleen and bursa of Fabricius, as well as splenic congestion, hyalinization of blood vessel walls in the spleen were noted. Parenchymatous degeneration of hepatocytes, multifocal infiltration by mononuclear cells around portal triads, blood vessels and congestion were observed in the liver.



In pigeons of case 3 we noted congestion of the kidneys, bursa of Fabricius and jejunal mucosa. A histopathological analysis of skin sections revealed a reduced number or absence of feather pulps. Quills were surrounded by infiltrating mononuclear cells (Fig. 5c). Marked depletion of lymphoid tissue with cyst formation and botryoid inclusion bodies in macrophages were noted in the bursa of Fabricius. Lesions in the spleen consisted of moderate lymphoid depletion. Reduced thickness of the cortical layer, lymphoid depletion and hemorrhages were observed in the thymus. Inflammatory infiltration constituted mainly by mononuclear cells (lymphocytes and plasma cells) was reported in the heart, kidneys and liver.

In case 4 we observed congested liver, pancreas and kidneys. Vacuolization (Fig. 5d) and necrosis of barb ridge cells in the feather pulp, as well as focal infiltration of mononuclear cells in connective tissue were noted in the analyzed skin sections. Disorganized columns of barb ridge cells with the presence of the cell debris were also reported (Fig. 5e, f). Moreover, interstitial nephritis, multifocal infiltration of mononuclear cells in the liver, focal cardiomyocyte atrophy and necrosis were observed. Congestion, edema, focal spongiosis and neuronal degeneration, moderate proliferation of glial cells and neuronophagia were noted in the central nervous system.

**Microbiology.** Microbiological analyses of cloacal swabs revealed moderate counts of non-hemolytic *E. coli*,  $\alpha$ -hemolytic *Streptococcus* spp. and *Corynebacterium* spp. (case 1). Moderate counts of non-hemolytic *E. coli* and numerous  $\beta$ -hemolytic *Streptococcus* spp. were isolated from tissue samples (case 2, 3). Single coagulase-positive *Streptococcus* spp. were isolated from the tissues of one bird (case 3). In case 4, mesophilic bacteria were not isolated from the supplied samples. All samples were negative for *Salmonella* spp. and fungal pathogens.

In this study, four independent cases of feather abnormalities included in seven young pigeons were investigated. Most of the pigeons were protected against infectious diseases by vaccination. They showed no clinical signs of diseases, with the exception of case 2 which was not vaccinated and dewormed. Almost all birds were affected in varying degrees by coccidia, *Trichomonas* spp. Only a single invasion of *Columbicola columbae* was recognized in one case. In some pigeons, *E. coli* and *Streptococcus* spp. were isolated from tissue samples. We confirmed PiCV infections in three cases and APMV-1 infection in one

case. The presence of PiHV or PiAdV were ruled out. In this study, pigeons were at age 5 weeks to approx. 1 year. According to the literature, PiCV poses the greatest threat for young pigeons, less than one year of age (16, 25). Tavernier et al. (23) reported the highest seasonal prevalence of PiCV infections between March and May. In our study infection was noted in May-August. According to the literature the feather abnormalities associated with circovirus have been reported in a variety of birds (11, 17, 18, 24, 26), but they appeared to be not a common symptom in pigeons in the course of pigeon circovirus (8). The clinical picture and pathological findings in pigeons (case 1-3) were similar to the changes described in other birds with circovirus (12-14, 21, 26), but the clear and unambiguous impact of PiCV on feathers cannot be confirmed in this study. Blood parameters of examined pigeons were within the reference range for this bird species (1). It is generally believed that hematological analyses do not have high diagnostic significance in pigeon circovirus (10). Pathological lesions are often associated with secondary infections because of an immunosuppression induced by PiCV (10, 23-25), which corresponded with our results. Moreover, we observed lymphoid depletion in lymphatic organs (mainly bursa of Fabricius), follicular atrophy of the bursa of Fabricius and the presence of intracytoplasmic botryoid inclusions, which were considered pathognomonic for pigeon circovirus infection (24). Botryoid inclusions in the bursa were not detected in all PiCV positive cases (only in case 1 and 3), which supported the previous suggestions that not every PiCV may be capable of producing these inclusions (4). The granulomatous inflammation of the skin was observed in one pigeon (case 1). We found that it could be induced by previous vaccination; however, it was absent in the second pigeons (with similar lesions on feathers) from the same case. We supposed that a history of pox infections in lofts, presence of lice together with the anti-parasitic drugs used might have an impact on the development of feathers in early stages. Based on the results of clinical examination and normal hematological parameters we excluded intoxication as a cause of feather lesions (5).

The results of PCR confirmed paramyxovirus (PPMV-1) infection and ruled out PiCV in pigeons of case 4. The feather abnormalities in this study were comparable with results presented by Lemahieu et al. (7). Similar feather lesions were observed to be also associated with the pinching off syndrome. The cause

**Fig. 5.** (a) Case 2. Massive hemorrhages (arrow) and mononuclear cell infiltration in the subcutis close to the calamus wall (asterisk) and lamellar corpuscle (arrowhead). (b) Case 2. Longitudinal section of the skin: loss of feathers (asterisks), papillary hyperplasia of the epidermis with mild hyperkeratosis (arrowheads) and inflammatory cell infiltration (arrows). (c) Case 3. Transverse section of quills (asterisks) surrounded by inflammatory cells (arrows), loss of feather pulp in dermis. (d) Case 4. Feather pulp with vacuolated cell barb ridge (arrows) and a mild inflammatory response. Longitudinal section of the feather filament, columns of barbules (arrows) in normal (e) and pathologically changed (f) quills. In comparison with a normal feather (e), the affected feather (f) was characterized by a loose meshwork of the pulp (asterisk), vacuolated barbule cells (arrowheads) and disorganized barbules (arrows). H-E, scale bar 200  $\times$  (a-d), 50  $\times$  (e-f)

of the syndrome has been attributed to quill mites, and to viral or genetic etiology (9, 26). The pinching off of old wing feathers from the base was followed by a regrowth of half-developed basal feather stumps to nearly full length. In opposition to the other authors, we found that tail feathers were unchanged, but more than one flight feather and smaller feather on both wings may be affected (7). On the other hand similar lesions may be observed after administration of an overdose of fenbendazole, e.g. during molting or in the pin-feather state (5, 6). We concluded that macroscopically the feathers of pigeons in cases 1-3 differed from the feathers of birds from case 4; however, their microscopic views were similar. The feathers in the pigeons of case 4 were pinched off to produce a characteristic hourglass-like appearance, which was not observed in other cases (1-3). Moreover, the structure of barb ridges was pathologically affected in pigeons of case 4, but not among other cases.

The instance of feather diseases in pigeons depends on many different factors and may have a complex etiology. The mechanism of the action of benzimidazoles in generating feather abnormalities in pigeons is still unknown. The role of PiCV and PPMV-1 in this process has not been sufficiently explained. According to the recent reports, the evolution of PPMV-1, genetic diversity of PiCV, and possibility of recombination can lead to the creation of emerging viral variants with new properties (19, 20, 22). More research on their pathogenesis is needed to determine if feather abnormalities have resulted from direct or indirect effects of infections, intoxications, liver dysfunctions or other factors. The present study is one of the few in which an analysis of feather lesions in young racing pigeons was discussed. To the best of our knowledge, there is a lack of sufficient data on similar analysis in the literature.

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