

VETERINARY DERMATOLOGY



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Discoid lupus erythematosus in a dog

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Abstract

Introduction. Autoimmune dermatoses in dogs account for only 2–3% of all skin and coat pathologies encountered in veterinary dermatology practice. Discoid lupus erythematosus (DLE) in dogs is a chronic autoimmune disease. This condition was first described in dogs in 1965, but it was not until the 1970s that cutaneous variants were officially documented in dogs that developed chronic dermatitis following prolonged sun exposure. Lesions may be confined solely to the muzzle, the so-called facial form of discoid lupus erythematosus, or may be localized to other areas of the body — the generalized form of DLE. Olivry et al. developed a histopathological classification for DLE. It was also established that the histopathology of lupus shows similarities to the microscopic lesions present in cutaneous lupus erythematosus in humans. For veterinary specialists, this group of diseases raises many questions regarding diagnosis, selection of therapeutic methods, and monitoring of the patient's condition.

Case Description. A 5-year-old mixed-breed dog was presented to the veterinary clinic with lesions on the nasal planum, without systemic clinical abnormalities. The diagnosis of discoid lupus erythematosus was based on history, clinical examination, cytological examination, complete blood count and serum biochemistry, and histopathological examination of biopsy samples from the nasal planum. For cytological examination, impression smears were taken from ulcerated skin areas. The smears were fixed, stained with Diahem-Diff-Quick stain (Russia), and examined under a microscope at $\times 100$ magnification using an Olympus CX23 microscope (Japan). To establish the definitive diagnosis, a punch biopsy was performed under general anesthesia using a 3-mm punch; three tissue samples were obtained from the nasal planum and nasal wings. Histopathological examination was performed by veterinary pathologist I.V. Bogomolova.

Discussion. The presented clinical case illustrates the typical facial form of discoid lupus erythematosus (DLE) in a dog. The localization of lesions exclusively to the nasal planum, the absence of systemic symptoms, and normal blood parameters are consistent with the classic course of FDLE, highlighting the importance of differentiating this form from generalized and systemic lupus erythematosus. The diagnosis was verified based on histopathological examination, the gold standard for DLE diagnosis. The identified changes allowed the exclusion of other autoimmune dermatoses, particularly pemphigus, despite the presence of acantholytic cells on cytology. This finding confirms that cytological examination cannot serve as the sole diagnostic criterion.

Conclusion. The treatment outcomes and photographic materials presented in this article may be useful for practicing veterinarians in the diagnosis of discoid lupus erythematosus. The applied combination therapy proved to be effective.

Keywords: canine cutaneous lupus, discoid lupus erythematosus, histopathology of lupus in dogs, DLE

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Дискоидная красная волчанка у собаки

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Аннотация

Введение. Аутоиммунные дерматозы у собак составляют лишь 2–3 % от общего числа патологий кожи и шерстного покрова в практике ветеринарного дерматолога. Дискоидная красная волчанка (DLE) — хроническое заболевание аутоиммунной этиологии. Впервые данная патология была описана у собак в 1965 году, а в 1970-х годах были выделены и зарегистрированы кожные формы заболевания, связанные с развитием хронического дерматита после длительной инсоляции. Поражения при DLE могут ограничиваться областью морды; такую форму обозначают как лицевою дискоидную красную волчанку (FDLE). В других случаях очаги поражения могут

локализоваться на различных участках тела, что соответствует генерализованной форме DLE. Для заболевания характерны определенные гистопатологические признаки, при этом микроскопическая картина волчанки у собак имеет сходство с изменениями, наблюдаемыми при кожной форме красной волчанки у человека. В ветеринарной практике данная группа заболеваний нередко вызывает трудности, связанные с диагностикой, выбором терапевтической тактики и последующим контролем состояния пациента.

Описание клинического случая. Собака, метис, в возрасте 5 лет, поступила в ветеринарную клинику с поражениями в области мочки носа; системные клинические отклонения отсутствовали. Диагностика дискоидной красной волчанки основывалась на данных анамнеза, клинического осмотра, цитологического исследования, общего клинического и биохимического анализов крови, а также гистологического изучения биоптатов.

Для цитологического исследования отбирали мазки-отпечатки с эрозивных участков. Образцы фиксировали, окрашивали набором Диахим-Дифф-Квик (Россия) и подвергали микроскопии при увеличении $\times 100$ с использованием микроскопа Olympus CX 23 (Япония). Для верификации окончательного диагноза под общей анестезией была выполнена панч-биопсия: диаметр панча составлял 3 мм, было получено 3 образца тканей с мочки и крыльев носа. Гистологическое исследование было проведено ветеринарным патоморфологом.

Обсуждение. Описанный случай демонстрирует классическую лицевою форму дискоидной красной волчанки. Изолированная локализация очагов в области мочки носа и отсутствие системной симптоматики соответствуют течению FDLE. Это подчеркивает важность дифференциации данной формы от системной и генерализованной волчанки.

Диагноз был верифицирован на основании гистопатологического исследования, которое считается «золотым стандартом» диагностики DLE. Выявленные изменения позволили исключить иные аутоиммунные дерматозы, в частности пузырчатку, несмотря на обнаружение акантолитических клеток при цитологическом исследовании. Данный факт подтверждает, что цитология не может являться единственным критерием постановки окончательного диагноза.

Заключение. Представленные результаты лечения и фотоматериалы могут быть полезны практикующим специалистам при диагностике дискоидной красной волчанки. Примененная комплексная терапия продемонстрировала высокую эффективность.

Ключевые слова: кожная волчанка у собак, дискоидная красная волчанка, гистопатология волчанки у собак, DLE

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Introduction. Discoid lupus erythematosus in dogs (Discoid lupus erythematosus, DLE) is an autoimmune disease with an unknown etiology. The breed predisposition of German Shepherds and their mestizos indicates the possible involvement of a genetic component in the development of DLE [1]. The disease is divided into facial form (FDLE) and generalized form (GDLE) [2].

The most common clinical manifestation in dogs is a single chronic skin lesion; a typical localization in FDLE is the nasal mirror [1]. This form is characterized by erythema, depigmentation, ulceration, crusting, as well as partial or complete loss of the normal relief of the nasal mirror with atrophy and a violation of the architecture of the “cobblestone pavement” type. With FDLE, locally destructive changes in the nose are often recorded, accompanied by edema of soft tissues and lytic lesions of bone structures [2]. In GDLE, the pathological process has a generalized or multi-focal character; foci are usually localized on the neck, the back of the nose, the lateral surfaces of the trunk and the ventral region of the chest [3].

Histopathological examination remains the “gold standard” for differentiating subtypes of the disease: perivascular, superficial, periadnexal, and deep microscopic skin changes are detected [4]. To make a definitive diagnosis, it is necessary to correlate the clinical picture with typical morphological features [5].

The pathogenesis of DLE remains multifactorial and includes a genetic predisposition, the influence of environmental factors, as well as the interaction of innate and adaptive immune responses [6]. Ultraviolet radiation induces a cascade of pro-inflammatory cytokines, which leads to keratinocyte apoptosis and promotes the formation of DLE lesions [6]. Taking into account the complex pathogenesis of the disease is important in developing effective treatment strategies, which ideally should combine correction of external provoking factors and pharmacotherapy [6].

Currently, as in humans, there is no approved specific treatment for FDLE in dogs [7]. Since pathology is associated with hyperactivity of innate and adaptive immunity, various immunosuppressive and immunomodulatory approaches are used in therapy, both local and systemic [8]. Oral prednisone in immunosuppressive doses is usually used as first-line therapy, however, the clinical response may vary, and prolonged use of the drug is accompanied by side effects [1]. Cyclosporine (4.8 mg/kg orally twice daily) and azathioprine (1.5 mg/kg orally once daily) are recommended as alternatives, taking into account their effectiveness and safety profile [2]. For local immunosuppression, steroid creams

are used, more often with 0.1% betamethasone, but their use is difficult due to the licking of the drug by animals [3]. A number of studies have used immunomodulators — tetracycline in combination with niacinamide; The results were mixed: early studies indicated a good or excellent response in 70% of animals, while retrospective data showed a similar response in 67% of cases with a high recurrence rate [9]. The mechanism of their synergistic action remains unclear [10].

In chronic cases, oral vitamin E intake was recommended — no more than 400 IU per day before remission — as a means of promoting skin regeneration [11].

From the point of view of clinical practice, treatment in humans is proposed to be carried out in three stages: prevention, including minimizing sun exposure; first-line therapy using topical steroids or calcineurin inhibitors; second-line therapy involving the appointment of systemic steroids or other immunosuppressants. It is advisable to apply a similar approach in dogs [11]. The primary task is to inform owners about the chronic nature of the disease, the need to avoid trigger factors and strictly follow long-term therapy regimens [12].

DLE can lead to pronounced cosmetic defects and discomfort, which worsens the quality of animals' life [13]. With the progression of lesions, it is advisable to prescribe analgesia: its absence can significantly reduce the patient's well-being and the owner's motivation to continue treatment, therefore, an analgesia plan should be discussed and formulated in advance.

New targeted approaches to DLE therapy are emerging. Pro-inflammatory proteins play a key role in chronic lesions; transcriptomic studies of skin lesions in dogs and humans have revealed similar activation of innate immunity, the interferon signaling network, and the JAK–STAT pathway [14]. Experimental data demonstrate a rapid and pronounced response to oclacitinib in FDLE: clinical improvement was noted during the first two weeks at a dose of 1.8 mg/kg twice daily, and complete remission was achieved after four weeks of therapy [14]. Oclacitinib is a non-selective JAK inhibitor and affects key pathogenesis links. Although this protocol is still being studied, the early results represent an encouraging prospect for accelerating and improving the effectiveness of treatment [14].

Clinical case description. At the age of 5, a half-breed dog was admitted to a veterinary clinic with lesions in the area of the nose, without systemic clinical abnormalities. The diagnosis of discoid lupus erythematosus was based on medical history, clinical examination, cytological examination, general clinical and biochemical blood tests, as well as histological examination of nasal biopsies. For cytological examination, fingerprint smears were performed from ulcerated skin areas; the preparations were fixed and stained with a Diff-Quik kit (Russia). Microscopy was performed at magnification $\times 100$ using an Olympus CX23 microscope (Japan). To verify the diagnosis, a punch biopsy was performed under general anesthesia: the punch diameter was 3 mm, and 3 histological samples were obtained from the lobe and wings of the nose. The histological examination was performed by veterinary pathologist I.V. Bogomolova.

A five-year-old mongrel dog weighing 19.8 kg was admitted to the veterinary clinic “Center” (Rostov-on-Don). The main complaints of the owners/staff were peeling of the epidermis, depigmentation, ulceration and crusting in the area of the nose and nostrils. Prior to admission, local treatment with Bactroban ointment (mupirocin) was carried out for 14 days, but no clinical improvement was noted. The dog is kept in a shelter, and therefore it was impossible to collect a detailed medical history.

The examination revealed diffuse erythematous, depigmented and ulcerated areas of the nose and nostrils, which caused the animal severe local discomfort, constant friction and subsequent scratching (Fig. 1). Skin lesions were limited to the area of the nose; no other abnormalities were found during a general dermatological examination.

Taking into account the clinical picture, the ineffectiveness of previously prescribed local antibiotic therapy and the results of cytological analysis, the differential series was focused on an autoimmune epidermal lesion — discoid lupus erythematosus. Histological confirmation consisted in the identification of changes in the epidermis and dermis characteristic of DLE, which made it possible to establish a definitive diagnosis and determine subsequent treatment tactics aimed at suppressing the local inflammatory response and relieving symptoms.



Fig. 1. The dog at the initial examination before the start of treatment

Differential diagnosis was performed based on medical history and clinical examination results. The following autoimmune dermatoses were included in the differential series: mucocutaneous pemphigus; epidermolysis bullosa and discoid lupus erythematosus (DLE).

The volunteers were offered a biopsy to confirm the diagnosis and choose therapeutic tactics. Before the intervention, a general clinical blood test (Table 1) and a biochemical blood serum test were performed; no clinically significant abnormalities were detected (Table 2).

Table 1

General clinical blood test, increased red blood cells

Indicator	Result	Units of measurement	Normal range	Deviation
White blood cells (WBC)	7,1	K/uL	6,0 – 16,0	Within normal limits
Number of nuclear red blood cells/100 white blood cells	0		0 – 5	Within normal limits
Adjusted white blood cells	7,10	$\times 10^9 / l$	6,00 – 16,00	Within normal limits
Red blood cells (RBC)	8,57	M/uL	5,60 – 8,00	▲ 7%
Hemoglobin (HGB)	215	g/L	130 – 180	▲ 19%
Hematocrit (HCT)	61,6	%	38,0 – 55,0	▲ 12%
Mean Corpuscular Volume (MCV)	71,88	μm^3 (fl)	60,00 – 75,00	Within normal limits
Mean Corpuscular Hemoglobin (MCH)	25,09	Pg	21,00 – 27,00	Within normal limits
Mean corpuscular hemoglobin concentration (MCHC)	349	g/L	330 – 380	Within normal limits
Red Cell Distribution Width (RDW)	12,5	%	11,90 – 16,00	Within normal limits
Thrombocyte (PLT)	175	K/uL	160 – 550	Within normal limits
Erythrocyte Sedimentation Rate (ESR)	0	mm/hr	0 – 6	Within normal limits
Neutrophilic myelocytes	0	%	0	Within normal limits
Neutrophilic metamyelocytes	0	%	0	Within normal limits
Rod-shaped neutrophils	0	%	0 – 3	Within normal limits
Segmented neutrophils	64	%	60 – 70	Within normal limits
Eosinophils	9	%	0 – 5	▲ 80 %
Monocytes	6	%	0 – 7	Within normal limits
Basophils	0	%	0 – 1	Within normal limits
Lymphocytes	21	%	12 – 30	Within normal limits
Others	0	%	0,00	Within normal limits
Absolute values of leukocytes				
Rod-shaped neutrophils	0	$\times 10^9 / L$	0,00 – 0,33	Within normal limits
Segmented neutrophils	4,54	$\times 10^9 / L$	3,60 – 11,20	Within normal limits

Eosinophils	0,639	$\times 10^9/L$	0,00 – 0,80	Within normal limits
Monocytes	0,426	$\times 10^9/L$	0,00 – 1,12	Within normal limits
Basophils	0	$\times 10^9/L$	0,00 – 0,16	Within normal limits
Lymphocytes	1,491	$\times 10^9/L$	0,00 – 0,16	Within normal limits

Table 2

Biochemical blood analysis

Indicator	Result	Units of measurement	Normal range	Deviation
BILT	2,5	$\mu\text{mol/L}$	2,0 – 13,5	Within normal limits
AST	35,2	U/L	8,0 – 42,0	Within normal limits
ALT	51,8	U/L	10,0 – 58,0	Within normal limits
UREA	5,94	mmol/L	3,5 – 9,2	Within normal limits
CREA	108,6	mmol/L	26,0 – 130,0	Within normal limits
TP	65,8	g/L	55,0 – 75,0	Within normal limits
ALB	34,6	g/L	25,0 – 39,0	Within normal limits
ALKP	63	U/L	10 – 70	Within normal limits
AMY	887,0	U/L	300,0 – 1500,0	Within normal limits
GLUE	4,66	mmol/L	4,30 – 7,30	Within normal limits
LDH	71	U/L	23 – 220	Within normal limits
GLOB	31,2	g/L	30,00 – 36,00	Within normal limits
ALB/GLOB	1,109		0,600 – 1,300	Within normal limits
De Ritis ratio	0,680		–	–

A cytological examination of deep skin scrapings from the nasal mirror was performed and a large number of acantholytic keratinocytes and neutrophilic inflammation were diagnosed (Fig. 2).

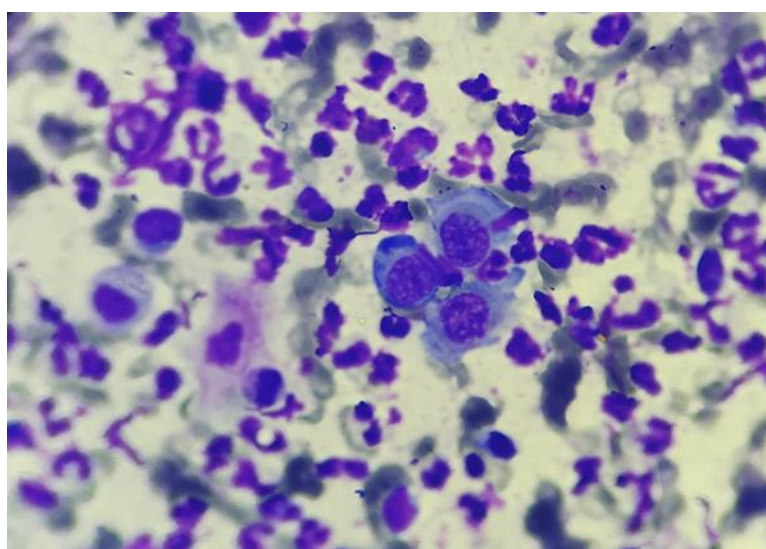


Fig. 2. Cytological examination, acantholytic keratinocytes, Diff-Quik staining

The biopsy was performed under general anesthesia by a punch biopsy using an instrument with a diameter of 3 mm. 3 histological samples were obtained from the lobe and wings of the nose; the material was transported in 10% formalin for subsequent histopathological examination (Fig. 3).



Fig. 3. Taking a punch biopsy

Serial histological sections (Fig. 4, 5) showed the following changes: the epidermis is moderately hyperplastic, with a predominance of acanthosis, irregular in places; there is also a focal minor hypergranulosis. The stratum corneum is indistinctly or moderately thickened, mainly due to orthokeratosis of both basket-shaped and compact types; rare foci of parakeratosis are revealed. Several intracorneal pustules of various sizes were found in the dermis, including large, partially damaged ones with transformation into scabs; their cavities are filled mainly with numerous neutrophils. There were no convincing signs of acantholytic keratinocytes. In some places, the stratum corneum is slightly “peeled off”, which may be partly due to processing artifacts; no obvious erosions or deep ulceration were found within the examined sections.

Spongiosis, that is, intercellular edema of the epidermis, is moderately pronounced and increases as it approaches the areas of pustulation and scab formation; in the same areas, moderate neutrophilic exocytosis is detected in places. There is no vesicle. Keratinocytes with signs of vacuolar degeneration corresponding to intracellular edema are present in significant numbers with a pronounced emphasis on the basal layer. Apoptosis-induced keratinocytes are detected occasionally, in the amount of 0-2 in one field of view $\times 400$, HPF, mainly in the basal layer. The interface zone in most fields of vision looks “blurred”; there are no stratification sites along the epidermis–dermis boundary within the sections.

The adnexal structures of the skin are relatively sparsely represented of all types; their ratio is generally close to normal, but the distribution is somewhat uneven. Hair follicles are found in all phases of growth; the anagen–telogen ratio is approximately 1:1. The epithelium in the funnel area is indistinctly or moderately hyperplastic, the stratum corneum is slightly thickened; there is no pustulation in the follicular region. Basal keratinocytes of hair follicles exhibit vacuolar degeneration and individual apoptotic cells, similar to the changes detected in the epidermis. In most cases, the hair shafts are preserved and have a relatively normal structure; the lumen of the follicles is relatively clean. Dilapidated follicles with the release of their contents into the surrounding soft tissues were not found within the sections.

The sebaceous glands are slightly atrophic in places; their excretory ducts have no significant features. The excretory ducts of the apocrine sweat glands are sometimes slightly dilated. Pigment granules were detected in moderate amounts and localized in the cytoplasm of epithelial cells of the epidermis and follicles; their distribution is somewhat uneven. Melanocytes are rare. Extracellular clusters of pigment granules and single melanophages are sometimes detected in the upper layers of the dermis, which corresponds to the phenomenon of “leakage” of pigment.

The soft tissues of the dermis are edematous and contain pronounced lichenoid-interface infiltration, represented mainly by small lymphocytes, to a lesser extent by neutrophils. Sometimes there are histiocytes and macrophages, including activated cells with vacuolized cytoplasm and signs of phagocytosis; less often there are plasmacytes and single eosinophils. The fibrotic reaction is mild. The vessels are represented in moderate numbers, have different calibers, and are slightly dilated in places; endotheliocytes are sometimes slightly swollen, while there is no pronounced infiltration of the vascular wall. Dilapidated vessels are not convincingly identified.

Subcutaneous fat is relatively sparsely represented, with no significant changes. There are rare single inclusions of skeletal muscle fibers along the edges of some sections. The mucocutaneous junction and the mucous membrane of the nasal cavity are absent in the preparation. No obvious necrosis was detected, fistulous passages were not convincingly

determined, and no signs of dystrophic mineralization were noted. Microorganisms are not visualized: bacteria, significant fungal forms, protozoa, mites and helminths are not found; significant foreign artifacts are also not found.

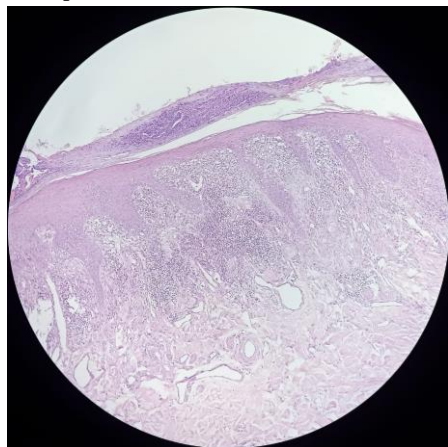


Fig 4. Lichenoid–interface inflammation and superficial pyoderma, ×10

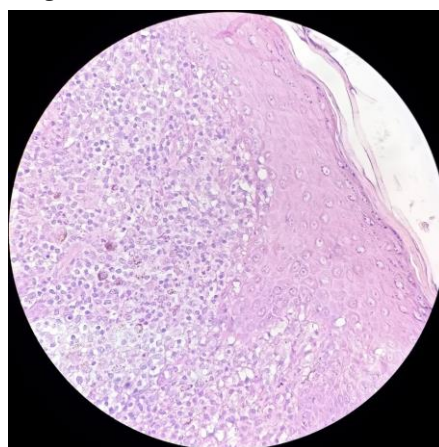


Fig 5. Apoptosis of keratinocytes of the basal layer, ×40

The final diagnosis was made based on clinical examination data and histological examination results — discoid lupus erythematosus (DLE).

Induction therapy mainly included systemic glucocorticosteroids, as well as combined treatment. The scheme and sequence of therapeutic measures are presented below:

- systemic glucocorticosteroids: “Prednisone” at a dose of 1 mg / kg once a day for 1 month; then a gradual dose reduction followed by a phased withdrawal of the drug;
- local therapy: apply “Mometasone” ointment in a thin layer to the nasal area 2 times a day, preventing the drug from being licked off for 5 minutes; the course treatment takes 7 days. Then reduce the frequency of application to 1 time per day every other day; the course takes 7 days. After completing the “Mometasone” treatment course, switch to “Hydrocortisone” ointment every other day; the course takes 14 days;
- antimicrobial-immunomodulatory combination: “Tetracycline” + “Nicotinamide” (nicotinic acid amide, vitamin PP or B3) 500 mg of each drug 3 times a day; after achieving a clinical effect, a reduction in the frequency of administration to 1-2 times a day;
- vitamin support: vitamin E 400 IU 2 times a day; course treatment — 4 months.

All of these drugs were prescribed orally, with the exception of local glucocorticosteroid treatment. It is recommended to keep the dog in an aviary with a canopy to limit prolonged exposure to ultraviolet radiation, which can exacerbate the course of the disease. The dynamics of therapy is shown in Fig. 6-9.

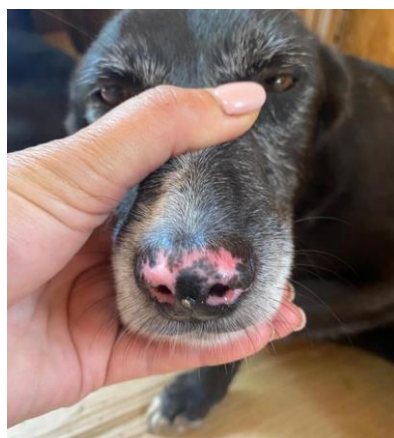


Fig. 6. The dog after 15 days of therapy



Fig. 7. The dog after 1 month of therapy

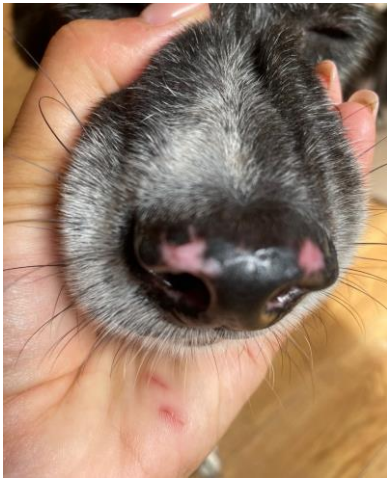


Fig. 8. The dog after 1,5 month of therapy



Fig. 9. The dog after 4.5 month of therapy

Conclusion. The purpose of this clinical report is to raise the awareness of veterinary specialists about discoid lupus erythematosus (DLE) in dogs, so that this disease is timely taken into account when conducting differential diagnosis and choosing clinical tactics. The prognosis for lupus is variable and directly depends on the severity of the course and the form of the pathology: both favorable and unfavorable outcomes are possible. The most cautious prognosis is typical for systemic lupus, especially in the presence of hemolytic anemia, thrombocytopenia or glomerulonephritis. According to the number of authors, dogs that respond quickly to glucocorticosteroid therapy have a higher survival rate and prolonged remissions.

It is crucial for dog owners to understand the need for regular monitoring of the clinical condition of an animal, as well as monitoring the indicators of general and biochemical blood tests, followed by correction of therapy if necessary. Patients with DLE should be protected from the adverse effects of ultraviolet radiation; however, as demonstrated by the described clinical case, complete exclusion of insolation is not always possible. It is recommended to use sunscreens, taking into account the risk of their licking off when applied to the nose [15].

Literature data on the epidemiology of DLE in dogs remain limited [15]. In this regard, further research is needed to better understand the pathogenesis of the disease, identify prognostic factors, and develop optimal treatment methods [16].

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