EOSINOPHILIA AND PARASITIC DISEASES

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ABSTRACT

Modification of eosinophil number may be due to more than one cause. One of these is a possible parasitic infection. Not every parasitic infection can cause change in peripheral blood eosinophil number. Usually, there is a growing eosinophilia in tissue helminth infections. In parasitic infections produced by protozoa or intraluminal worms eosinophilia is not significantly modified or altered. In the following are summarized the main nonparasitic diseases that can cause alteration of eosinophilia and the evolution of eosinophilia in the most common parasitic infections is analyzed. To determine the significance of eosinophilia within the broader clinical and laboratory diagnosis any investigation should start with fixing presence or lack of possible parasitic infections.

Keywords: eosinophilia, parasitic infections, nonparasitic diseases

REZUMAT

Modificarea numărului de eozinofile poate fi datorată mai multor cauze. Una dintre acestea este o eventuală infecție parazitară. Nu orice infecție parazitară determină modificarea numărului eozinofilelor în sângele periferic. De regulă, are loc o creștere importantă a eozinofiliei în parazitozele tisulare produse de helminți. Infecțiile determinate de protozoare parazite sau de helminți cavitari nu modifică sau modifică nesemnificativ eozinofilia.

În cele ce urmează, sunt prezentate pe scurt principalele afecțiuni nonparazitare ce pot determina modificarea eozinofiliei și, în continuare, este analizată evoluția eozinofiliei în cele mai răspândite infecții parazitare. Pentru a stabili semnificația unei eozinofilii în cadrul mai larg al unui diagnostic clinic și de laborator, se consideră că orice investigație trebuie să înceapă cu stabilirea prezenței sau absenței unei eventuale infecții parazitare.

Cuvinte-cheie: eozinofilie, infecții parazitare, afecțiuni nonparazitare

INTRODUCTION

The notion of "eosinophilia" means an increase in the relative or absolute number of circulating eosinophils in the blood, or existing in the bone marrow or other tissues [1, 2].

In adults, normal values are usually within the range of 1-5%, or about 300-400 elements/ mm^3 of peripheral blood.

Although physiological variations have been described depending on age, the stages of the menstrual cycle, or even the different moments of the day, it is believed that eosinophilia can be most commonly associated with parasitic or allergic diseases.

There are also non-parasitic eosinophils, such as:

Hypereosinophilic syndrome (> 1500/ mm^3), lasting more than 6 months, with or without splenomegaly that can affect:

- cardiac (Loeffler endomyocardial fibrosis with adiastolia, myocarditis with eosinophilia,
- neurological infiltrates (diffuse encephalopathy, peripheral neuropathy, diseases of the optic nerve), **respiratory** (chronic cough),
- polymorphic mucous membranes (urticaria, erythematous lesions),

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- abdominal (diarrhea, frequent Charcot-Leyden crystals in the stool),
- kidneys (kidney failure).

Systemic diseases with eosinophilia, such as**:**

- Carrington chronic eosinophilic pneumonia (more common in women), with dyspnea, general aneurysm, radiographic evidence of dense peripheral infiltration with progressive extension,
- Shulman eosinophilia**,** myalgia, arthralgia, edema at the distal extremities,
- Churg-Strauss syndrome – granulomatosis and angiitis (diffuse vasculitis, with asthma, heart failure, neuritis and paralysis).

Eosinophilic leukemia

Allergic diseases:

- urticaria, bronchial asthma, allergic rhinitis, iatrogenic hypereosinophilia in relation to drug allergies (β-lactam, iodine-produced, radiotherapy).
- dermatological disorders: eczema, herpetiform dermatitis and lichens, as well as: Hodgkin's disease, Duhring-Brocq disease, chronic myeloid leukemia, Addison's disease, liver cancer, some digestive cancers, dermatomyositis, rheumatoid arthritis, hemorrhagic rectocolitis, Crohn's disease, leprosy, tuberculosis.

Given this complex of possible etiologies, it is considered that a diagnosis algorithm of eosinophilia begins with the exclusion of a possible parasitic infection.

Features and functions of mature eosinophil

The mature eosinophil is a mobile cell capable of phagocytosis. It comes from an autonomous medullary line and has a bilobed nucleus and intracytoplasmic granules.

After leaving the bone marrow, it enters into the circulatory system and then through the diapedesis into the tissues.

It is considered that in the peripheral blood there are normally less than 400 eosinophils/mm^3. In the case of a number greater than 400-500 eosinophils/mm^3, it is considered to be hypereosinophilia.

If the number of eosinophils is between:

- 500/mm^3 and 1500/mm^3 = moderate hypereosinophilia
- 1500/mm^3 and 3000/mm^3 = forte hypereosinophilia
- > 3000/mm^3 = massive hypereosinophilia

There are two different populations of eosinophils:

- a population of eosinophils called "normodense", granular, present in normal subjects or during eosinophils in helminthiasis.
- An eosinophil population called "hypodense", cytotoxic potential, responsible for tissue lesions and encountered in hypereosinophilic syndrome, collagenase, hemopathies, asthma.

Three essential functions of eosinophils are currently accepted: cell with cytotoxic function, the participant cell in the inflammatory process and the immunoregulatory cell.

These functions are ensured by the content of specific granules, that appear in the promyelocyte stage. The granules contain a crystalline central core and a clear peripheral matrix. At the crystalline core level, the so-called "Major Basic Protein" (MBP) involved in cytotoxic effects of eosinophil, and activating function on neutrophils, mast cells and basophils was identified. In the matrix other major proteins have been identified, such as: a neurotoxin (EDN/EPX), a peroxidase (EPO) and a cationic protein (ECP) also involved in cytotoxic processes (including parasites), but also in activities ribonuclease catalysts. Dosage of such major proteins from different biological fluids (serum, alveolar lavage) brings important information in establishing certain diagnoses.

The disintegration of eosinophils makes the crystalline cores associated with the formation of the Charcot-Leyden crystals. The occurrence of Charcot-Leyden crystals in biological products has therefore the significance of an appreciable local eosinophilia.

The role of eosinophils in the inflammatory process is determined by the release of:

- lipid mediators: produced leukotrienes (LT) of arachidonic acid metabolism, pros-

taglandin, thromboxane B2, platelet activation factors (PAFs)
- some chemokines: RANTES (products of both T lymphocytes and chemotactic platelets for monocytes and eosinophils), MIP-1α (macrophage inflammatory peptides), interleukins (IL-8)
- some cytokines: (IL-1, IL-6, TNF-α)

The immunoregulatory function of the eosinophil is expressed by the production of complex functions cytokines:
- immunoregulatory factors (IL-2, IL-4, IL-10, Th-2, IFN γ),
- growth and activation factors: granulo-monocyte colony growth factor (GM-CSF), IL-3, IL-5, IL-16, RANTES, MIP-1α, a specific eosinophil chemoattractant (Eotaxin)
- Factors involved in tissue and fibrous remodeling processes: TNFα, Transforming Growth Factor (TGFα, TGFβ), IL-1, IL-6, IL-8).

There are normally less than 400 eosinophils/mm^3 in the peripheral blood. The vast majority of eosinophils are found at the tissue level where they reach by the diapedesis. They concentrate on gastrointestinal tissues, respiratory and genito-urinary apparatus, that is tissues that can be the input gates of potential antigens. A ratio of 1/300 between blood and eosinophilic tissues is estimated.

Hypereosinophilia is relatively common in current pathology. Its origin is, however, often difficult to establish. It is appreciated that besides a helminthic parasitic infection or a possible **myiasis** (protozoa does not produce significant growth), hypereosinophilia can occur in the following situations: allergic diseases, dermatoses, gastrointestinal diseases, tumor processes, immune deficiencies, some systemic diseases, hereditary eosinophils, cyclic eosinophils, syndromes due to special environmental conditions.

In the context of this complex of possible etiologies, it is considered and, in fact, it is already in use that a diagnostic algorithm of hypereosinophilia should begin with the exclusion of an even tiny parasitic infection [3].

Characteristics of eosinophilia in parasitosis [4, 5, 6]

Eosinophilia is a dynamic phenomenon that varies depending on:

- *The time elapsed in the evolution of the infection*

The evolution of eosinophilia according to the time factor is expressed by the so-called "curve of Lavier". The Lavier curve records the eosinophilia values at different stages of the infection. Its appearance depends on the parasitic load, the host reactivity, the stages of the parasites involved and their localization. The curve has the appearance of an asymmetric "bell" with a roughly upward initial portion (corresponding to the initial phase of the infection), a maximum plate (state phase) and a slow down line (corresponding to the healing phase).

- *Influence of the species and of the biological cycle peculiarities.*

Depending on these factors, eosinophilia may develop differently

When parasitosis evolves linearly, in one way (ascariasis, ancylostomiasis), eosinophilia follows the Lavier curve. When the infection develops in acute **attack**, with allergic phenomena (filariasis), each **attack** corresponds to an increase in eosinophilia. When **autoinfection** occurs (strongyloidiasis), eosinophilia is increased in frequent and persistent repetitions, sometimes for decades. Parasites, whose biological cycle involves a tissue phase, cause important eosinophils during the course of this phase. At the end of the tissue phase and the onset of the cavity, the eosinophilia returns to moderate values (ascariasis, filariasis, schistosomiasis, fasciolosis, etc.) **Some** parasites from animals cause important and long-lasting eosinophils (MVLS, MCLS). In the same way, a dead tissue parasite causes a more severe reaction (including eosinophilia) than a viable one (cysticercosis, dracunculiasis).

Intact or non-viable hydatid cyst does not produce an important eosinophilia, but a **fissured** hydatid cyst causes a significant increase in eosinophilia throughout the drainage of the content.

• *Localization of the parasite*

The localization of the parasite also greatly influences how eosinophilia evolves. Thus, cavitary parasites without significant tissue phases cause moderate eosinophils and with rapid decreases (oxyurosis, trichuriasis, taeniasis). However, if the infection is strong or the progression of the parasite is rapid, significant temporal increases in eosinophils may occur. Cause: massive release, in a short time, of metabolites that, being absorbable, are found to be powerful allergens.

• *Parasitic load:*

In a certain infection, eosinophilia reaches the maximum values for a certain number of parasites. Affected as the characteristic value, the increase in eosinophilia is no longer proportional to the number of parasites.

In relation: parasitic load - the value of eosinophilia, the metabolism factor may also be involved; a parasitic species with intense metabolism can cause significant eosinophilia.

• *Influence of the host's biological characteristics*

There are internal and external factors that can influence eosinophilia. Among the internal factors, the following can be mentioned: adrenal cortical hyperactivity stimulates eosinophil production, the estrogen secretion, the black race produces fewer eosinophils than the white race.

The external factors that can influence eosinophilia are: bacterial infections and corticoid treatments transiently decrease eosinophil levels. First infection can produce an important eosinophilia. After healing, a possible reinfection causes a moderate increase in eosinophilia. After effective treatment, eosinophilia decreases at the beginning faster, then slower and returns to normal in a few weeks.

The following are the most widespread parasitic infections and their relationship with eosinophilia.

Ascariasis

It is the infection produced by a geohelminth; *Ascaris lumbricoides*. The infection is widespread in all geographical areas, affecting a high percentage of the population, especially in less-favored areas. The infective stage is the embryoning egg on the ground. The infection is caused by digestive tract by faulty food hygiene. The final location is the small intestine, but there is an initial peripheral phase through the liver and lung. The **diagnosis** can be supported by other elements (apart from eosinophilia): Loeffler syndrome (in the peripheral phase), mild digestive manifestations, skin allergic phenomena, respiratory, eventual digestive complications (intestinal occlusions, wrong migration of the adult parasite) [4, 6].

Eosinophilia: average value is 25%; respect the "curve of Lavier".

Maximum eosinophilia time: approximately 20 days from infection under the conditions of the liver and lung circulation of the larvae. After the parasite is located in the intestine, the eosinophilia decreases to moderate values and then normalizes [5].

Recommended investigations: Initial radiological examination (migratory infiltrative images), sputum examination (the parasite larvae and/or Charcot-Leyden crystals can be highlighted). Later, repeated coproparasitological tests for egg evidence (the test is positive after at least 60 days from infection). Often there is no correlation between peak eosinophilia and positive coproparasitological examinations. For this reason, parasitological examinations may be negative although the parasite exists, but in the hepatic and pulmonary migration phase. If suspicion persists, it is recommended to repeat the examinations after a few weeks.

Hookworm infection

The infection is produced by two geohelminths with tropical and subtropical spread or possibly in temperate climate in mine biotope: *Ancylostoma duodenale* and *Necator americanus*. The infection is transcutaneous by active penetration of **strongylid** larvae (infective stage). These can be found on soil contaminated with faeces from infected persons. The final localization of the parasite is the small intestine after initial hepatic and pulmonary migration phase.

The most common symptoms are dermatitis, Loeffler syndrome under the conditions of

perienteric migration larvae through the liver and lung, intestinal irritation, digestive disorders with melenic stools, severe anemia [3, 6].

Eosinophilia: the mean eosinophilia is 35-40%. This value is recorded in acute infections. At this stage, the Lavier curve is respected. Maximum value, 60-70% is recorded approximately three months after the infection. It is worth noting that eosinophilia remains at high levels both in the perienteric and intestinal migration phase where the parasite, although it is located cavitary, produces tissue lesions. In chronic infections, eosinophilia no longer respects the Lavier curve and usually decreases [4].

Recommended investigations: initial radiological examination (migratory infiltrative images), possibly sputum examination (parasite larvae can be highlighted). The certainty diagnosis consists of viewing the parasite eggs by repeated coproparasitological examinations. Tests are positive after 30-40 days of infection.

Bothriocephalosis

The infection is due to a cestode; *Diphyllobothrium latum*. Man is infected by the consumption of non-heat-activated fish meat, which contains the infective stage; "plerocercoid larvae". The infection is particularly common in riparian human communities that regularly consume fish.

The parasite located in the small intestine can produce a mild nonspecific symptomatology, in more serious cases with avitaminosis B and/or anemia [4, 6].

Eosinophilia: being a cavitary parasite, there is often no increase in the number of eosinophils. If it does occur, it is irregular and around the 40th day of infection reaches maximum values [5].

The diagnosis is made by coproparasitological examinations, indicating the eggs about three weeks after the infection.

Cysticercosis

It is *Cysticercus celullosae* larvae infection of *Taenia solium*. It is a cosmopolitan infection in countries where pigs are grown for consumption. The infection is by digestive tract and is due to defective hygiene, the infective stage being the eggs of *Taenia solium*. Self-infection is also possible if the person is parasitized by the adult stage located in the small intestine. The larval stage, the cysticercus is usually located subcutaneously, muscularly, and nervously. The most severe symptoms occur in nerve locations. [4, 6].

Peripheral eosinophilia may reach 20-30%, but does not always occur. It is mainly determined by the inactivated cysticercus, the viable being weakly reactive. In nerve locations, eosinophilia appears elevated in the CSF [5].

Diagnosis is usually imaging in corroboration with serology.

Fasciolosis

Cosmopolitan infection produced by a trematode helminth; *Fasciola hepatica*. The infection is particularly prevalent in herbivorous animals, but accidentally can occur in humans as it feeds on spontaneous vegetation, usually riparian, contaminated with the larvae of the parasite; the infective stage. The infection develops in two stages, one caused by larval migration through the peritoneum and the other by the adult worms located in intrahepatic bile ducts.

The most common symptoms are: Loeffler's syndrome, abdominal pain sometimes simulating an acute abdomen, acute angiocholitis, pain in the liver lobe, icteric phenomena. [4, 6]

Peripheral eosinophilia can be 40-60% and is reached in the larvae migration phase through the peritoneum, in the first 5-6 weeks after infection. The maximum value can reach 80%. Evolution of peripheral eosinophilia respects the Lavier curve. A particular feature is that eosinophilia, although decreasing, is maintained at high levels and after the parasite becomes cavitary in intrahepatic bile ducts. [5]

The diagnosis of certainty is achieved by repeated coproparasitological examinations with the emphasis of the characteristic operculum eggs. Tests are positive over two months after infection. The appearance of the eggs in the faeces does not coincide with the installa-

tion of the peripheral eosinophilia, the latter preceding the positivity of the examinations. So, although eosinophilia and some epidemiological data might suggest fasciolosis, coproparasitological examinations may be negative for a period of time.

Lymphatic filariosis

These are tropical haemolymphatic infections produced by nematodes with vectorial transmission: *Wuchereria bancrofti*, *W. bancrofti* var. *Pacifica* and *Brugia malayi*. The infective stage is "microfilaria" transmitted by the stings of several **genera** and species of tropical mosquitoes.

The most common symptoms are varied and target lymphatic and blood vessels. The lymphatic vessels and superficial lymph nodes are affected first and then the deep ones. The gradual narrowing of lumen of large lymph vessels leads to their closure, chronic disease and the appearance of "elephantiasis". Depending on the genera and species of filarial, elephantiasis affects the legs, genitalia, upper limbs, breasts. Frequently, lymphatic vessels fistulate with lymph drainage in cavity organs. A special form of pathology is the so-called "Tropical Lung Eosinophilia Syndrome" [4, 6].

Peripheral eosinophilia varies between 30-50%, but can also reach 80% in the case of Tropical Lung Eosinophilia Syndrome, all amid a pronounced leukocytosis and increased IgE. In reactive processes at the level of lymphatic vessels and adjacent tissues, eosinophils prevail [5].

Diagnosis of certainty is made by highlighting microfilaries on Giemsa stained peripheral blood stain thin and thick smear. The frequency of larvae is taken into account. Serology can also be done.

Cutaneous dermal filariosis

There are tropical infections with vectorial transmission. Etiologic agents are *Onchocerca volvulus* (transmitted by *Simulium* dipter stings), *Loa loa* (transmitted by the *Crysops* dipter) *Dracunculus medinensis* transmitted via drinking water contaminated with microfilaria infected small crustaceans.

Clinical manifestations include: dermatitis (prurigo, depigmentation, lichenal tear, lymphatic sorts), edema (Calabar), allergic phenomena, subcutaneous nodules, ocular lesions (keratite, iridocyclite, retinitis) [4, 6]

Peripheral eosinophilia can reach values of 30-60%, especially for people coming for a short time from areas without filariosis in endemic areas. It remains at high levels all the time in infections with *Onchocerca* and *Loa.*

In dracunculosis, the values are lower, approximately 10% and kept at this value only the first year of infection [5].

The diagnosis of certainty is made by highlighting microfilariae on Giemsa stained peripheral blood stain thin and thick smear, the ELISA serological tests, the skin biopsy, possibly the Mazzotti test.

Hydatidosis

Hydatidosis is the larval stage infection of *Echinococcus granulosus*, a small parasite in the dog's intestine. The infection is cosmopolitan, but it is more common in breeding colleges with animals accompanied by dogs. Accidentally, man infects with parasite eggs eliminated by faeces of the infected dog. The infection is by the digestive tract, and the larval stage; hydatid cyst is usually located in the liver, lung, brain, and less commonly in the other organs.

Clinical manifestations depend on the affected organ, the hydatid cyst exerting a compression that causes alteration of the functions. In case of cyst cracking, allergic manifestations appear to anaphylactic shock and in the longer term secondary sowing can occur [4, 6].

Peripheral eosinophilia does not occur or if it occurs has low values in the situation where the hydatid cyst is intact. In the case of cracked or broken cysts, the eosinophilia values may be very high [5].

Diagnosis is usually imaging in corroboration with serology.

Enterobiosis

It is an infection commonly seen in children especially in the community and is produced by a small nematode called *Enterobius vermicularis*. Localization of the parasite is the large intestine, the area of the cecum and the

vermicular appendix. The female deposits eggs mainly in the anal mucous membranes causing intense pruritus. Originally, non-embryonic eggs quickly form the embryo in the presence of oxygen becoming infectious. Autoinfections are common.

Clinical manifestations are mild and largely due to pruritus. They consist of insomnia, agitation, nervousness, lack of concentration. Rare complications like vulvovaginitis [4, 6].

Peripheral eosinophilia is mild, the parasite being cavitary. In the case of strong infections and when some parasites penetrate into the mucosal thickness, eosinophilia can reach 15% [5].

Diagnosis is done through a coproparasitological examination and especially by "anal print" or NIH, with emphasis on characteristic eggs.

Schistosomiasis

Tropical parasitosis is produced by several species of the genus *Schistosoma*: *S. mansoni, S. japonicum, S. intercalatum* (produce intestinal schistosomiasis) and *Schistosoma haematobium* (uro-vesicular schistosomiasis). The biological cycle involves embryogenesis in water in the presence of intermediate hosts. The infection is caused by active skin patching by the infected stage "furco cercariae". People who wash or work in contaminated water are affected.

Clinical manifestations are severe and include: dysentery, hemorrhage, anemia, fibrosis, necrosis and hepatic failure, hepatosplenomegaly, cachexia, esophageal varices, hematuria, kidney failure, granulomas and polyps in the bladder and intestinal mucosa, severe cough and respiratory cough [4, 6].

Peripheral eosinophilia is on average 30%, higher in pulmonary affections; These values are reached around the 60th day from infection in urinary forms, but earlier in digestive tract [5].

Diagnosis of certainty is made by pointing out the eggs in the faeces, usually after 35 days of infection or 60 to 70 days in the urine. Rectoscopy, cystoscopy, biopsy of rectal or bladder mucosa, serology.

Strongyloidiasis

Widespread cosmopolitan infection. Like all geohelminth and strongyloidiasis infections, it is common where there is no running water, sewerage, in conjunction with a poor health education that leads to massive contamination of soil with faeces.

The parasite, *Strongyloides stercoralis*, is located in the small intestine.

Clinical manifestations are most often digestive and depend on the intensity of the infection. The parasite is not hematophagous, so there is no anemia. At the skin level, pruritic, urticaria eruptions susceptible to over-infections and irregular tracts produced by transcutaneous infective strongyloides larvae may occur. Transient pulmonary symptoms of the Loeffler syndrome can occur due to perienteric migration. Frequent autoinfection occurs [4, 6].

Peripheral eosinophilia. Since all stages of the parasite are tissue and due to frequent autoinfection, we expect a high peripheral eosinophilia. On average, it can often exceed 25%, but there are many cases where it can reach 90%. But it is fluctuating due to repeated autoinfections. Maximum values are recorded approximately 40 days after infection, namely when rhabditoid larvae appear in the faeces [5].

Diagnosis of certainty is obtained by recognizing rhabditoid larvae in the faeces by direct exams or coprocultures on charcoal.

Teniasis

There are infections with adult stage of *Taenia solium* and *T. saginata*. Infections spread in all geographical areas to pork or beef consumers in the absence of rigorous veterinary control. The infection is by larval stage; *Cysticercus cellulosae/bovis* of not adequately heat treated pork/beef. Localization is the small intestine, and digestive symptoms are mild, unspecific or absent [4, 6].

Peripheral eosinophilia is low, on average 7-10%, not respecting the Lavier curve. Maximum eosinophilia can reach 15-20% and is

recorded around when proglots or eggs (diagnostic elements) appear in the faeces, i.e. 8-10 weeks after the infection [5].

Trichinosis

In the temperate zone the infection is usually produced by the adult and larval stage of the *Trichinella spiralis* nematode. The infective stage is the parasite larvae in the pork. In other geographical areas, the disease is produced by other parasite species located in other hosts, but these cases are less prevalent.

Symptomatology is varied as forms of manifestation and intensity and is dependent on the extent of infection. There may be: fever, allergic skin and respiratory phenomena, pruritus, facial edema, glottis, headache, myalgia, heart attacks [4, 6].

Peripheral eosinophilia appears quite late after the onset of symptoms, has an average of 15-20%, but in severe infections it can reach 90%. Follows the Lavier curve. Maximum eosinophilia is recorded in weeks 2 to 4 after infection under the conditions of larvae localization, so much later to serological positivity and to the larva circulation phase which is clinically most dangerous [5].

Diagnosis can be supported by serology and epidemiological investigation, the disease being an outbreak.

Trichuriasis

The infection is caused by the *Trichuris trichiura* nematode. Like ascaridiosis, trichocephalosis is a cosmopolitan infection, widespread in the absence of current water, sewerage, soil faecal contamination, and poor food hygiene. It is very widespread in tropical areas.

The digestive symptomatology is unspecific. In mild infections it may be missing. Sometimes, a mild anemia can occur in massive infections [4, 6].

Peripheral eosinophilia is moderate, usually in strong infections and reaches peak values approximately ten days after infection [5].

Diagnosis is confirmed by repeated coproparasitological examinations with the emphasis on characteristic eggs. Exams are positive, however, after about one month of infection, and later than the time of recording of increased eosinophilia. So although eosinophilia could signal a parasitic infection, the coproparasitological exam might be negative for a while.

Toxocariasis (M. V. L. S.)

Toxocariasis or Migrans Visceral Larva Syndrome is an accidental digestive infection with embryonated eggs on the soil belonging to *Toxocara canis* nematodes, of dogs and cats. Larvae begin their perietheric cycle, but go into the so-called "parasitic deadlock." The body's reaction kills the larvae and in time they are removed.

Clinical manifestations are the ones associated with Loeffler Syndrome. The liver may be enlarged and allergic manifestations may occur. Cases of ocular toxocariasis, usually unilateral, are reported less frequently [4, 6].

Peripheral eosinophilia has high values, but usually fluctuating without respecting Lavier's curve.

The maximum values may exceed 60-70% and persist after the treatment is established, as the destroyed larvae are resorbed, the tissue antigens persisting for a long time. It is worth mentioning that an ocular toxocariasis as the only localization of the infection does not significantly change the number of eosinophils. Eosinophilia in toxocariasis is also accompanied by IgM, IgG and IgE hypergammaglobulinemia [5].

Diagnosis of an active toxocariasis is given by a positive serology for IgM and IgG associated with high eosinophilia. Typically, toxocariasis releases the investigations in its active phase, the presence of the parasites being detected by long-standing IgG antibodies.

Migrans Cutanata Larva Syndrome

It is a cutaneous and temporary parasitic infection caused by worms of some species of *Ancylostoma* parasite in animals. Accidentally, the infective strongyloides larvae transcutaneously enter humans.

Clinical manifestations. At the place of penetration, larvae cause the formation of a pruritic, indurated and red papule. From this point appears a red, serpentine line a few centimeters. This line corresponds to a tunnel in the thickness of the skin by the moving larvae. The area is pruritus, especially at night. The infection can affect any part of the body that comes into contact with the contaminated soil and can last for a few weeks, sometimes even a year [4, 6].

Peripheral and local eosinophilia are significantly increased [5].

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REFERENCES

1. Eosinophil - Definition of eosinophil in English by Oxford Dictionaries - English. Retrieved 2018.
2. What is an Eosinophil? Definition & Function CCED. www.cincinnatichildrens.org Retrieved 2018.
3. Dan Steriu. Eozinofilia in infecțiile parazitare, RoEqualm, Simpozionul cu tema "Asigurarea calității rezultatelor examinărilor în laboratorul medical" 27-28 Octombrie 2018 (In Romanian).
4. Lab.Diag. Infec. Dis. Princip. and Pract. Balows, Hausler, Lennette (1988).
5. Dan Steriu. Infectii parazitare, (2003), Edit. Brilliant.
6. Topley & Wilson's Microbiology and Microbial infections (1998) V 5 ninth ed. Arnold.