

Ocular system involvement in the course of human trichinellosis. Pathological and diagnostic aspects

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Abstract

Pathomechanism of lesions in the course of trichinellosis was discussed, representing a sum of immunopathological, pathomorphological and biochemical phenomena. Particular attention was devoted to clinical pathology of the visual organ, which prevails at the acute stage of trichinellosis. In evaluation of clinical signs/symptoms manifested in the visual organ traits of its anatomic, morphological structure, function of the eyeball muscles and eyeball vascular system were taken into account. Ocular lesions in the course of trichinellosis reflect in principle angiomyositis due to immunopathology resulting from migration of *Trichinella* larvae to many organs and to structures of visual system.

Keywords

Trichinellosis, ocular system, pathology, differential diagnosis

Introduction

Within recent years decreasing numbers of trichinellosis epidemic foci have been detected. Nevertheless, the current potential for detection and confirmation of *Trichinella* spp. invasion manifestation in various environments, using modern investigative techniques, point to prevalence of the problem with a distinct character and scope.

Circulation of *Trichinella* spp. in synanthropic and sylvatic environments represents separate reservoirs, which in certain conditions may penetrate each other. Each reservoir may provide the source of human infection (Pozio 2001, Cabaj 2006, Gołąb and Sadkowska-Todys 2006). In the recent more than ten years consumption of wild boar meat increased, the meat not always subjected to a complete parasitological examination using mainly digestion techniques (Nowosad *et al.* 2004, Sadkowska-Todys and Gołąb 2013). On the other hand parasitological and molecular studies on wild animal – carnivores (foxes, wolves) and omnivores (wild boars, rats) in Poland demonstrated that they may be infected not only with *T. spiralis* but also with *T. britovi* (Nowosad and Pozio 1998, Cabaj *et al.* 2000, 2002, 2004). The detection of mixed invasion both *T. britovi* and *T. spiralis* in horse (from Poland) in 2008 should also be taken into account as a source infection for people (Liciardi *et al.*

2009). The first report of *T. pseudospiralis* in one red fox (*Vulpes vulpes*) in district of Nowy Targ in Poland (Moskwa *et al.* 2013) – is very important and uncommon finding from parasitological and epidemiological point of view; in group of 24 examined red foxes, the next three animals were infected with *T. britovi*. In 2010 the findings *T. nativa* in three red foxes in German (Güstrow district and Heidenheim district) and in Poland (Kętrzyn district) is a great epidemiological importance (Chmurzyńska *et al.* 2011).

Therefore, from the list of numerous *Trichinella* species and genotypes detected in different areas of the world, including new encapsulated species *T. patagoniensis* n.sp. (Krivokapich *et al.* 2012) – in Poland the presence of four species (*T. spiralis*, *T. britovi*, *T. nativa* and *T. pseudospiralis*) in domestic and wild animals is confirmed up to now.

In the last ten years two reported outbreaks of trichinellosis in France occurred due to consumption of wild boar meat infected with *T. britovi* (Gari-Toussant *et al.* 2005, De Bruyne *et al.* 2006). In Spain (Granada) in 2000 pork meat infected with *T. britovi* was identified as a source of outbreak of human trichinellosis (Gomez-Garcia *et al.* 2003). In Turkey (Izmir) in 2004 large epidemic was caused by the consumption of illegally mixed raw meat of beef with pork meat infected with *T. britovi* (Akkoc *et al.* 2008); group of 418 patients manifested typical clinical syndrome of acute trichinellosis. Only in

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Slovak Republic outbreak of trichinellosis in 2001 occurred by consumption of pork meat infected with *T. spiralis* (Reiterová *et al.* 2007).

A separate contemporary problem involves the fact that the reservoir and source of *Trichinella* invasion for humans may involve herbivorous animals. Evidence for this was provided by descriptions of epidemic foci of trichinellosis induced in France in 1975 and in 1985 by consumption of hoarse meat (Bourée *et al.* 1979, Dupouy-Camet *et al.* 1988, 1994a), in Italy in 1984 and 1986, as well other outbreaks have been described in Europe (Boireau *et al.* 2000). The morbidity was characterized by a benign clinical course, which was linked to a genetic type of *Trichinella britovi*. According to reports of the Coordination Group for Prevention and Treatment of Trichinellosis, in 1979–1980 and 1982–1984 in China numerous trichinellosis cases were noted following consumption of semi-raw mutton meat. Experimental studies demonstrated that rabbits (Chodera and Pawłowski 1974), goats (Reina *et al.* 1989) and merino sheep (Tomasovičová *et al.* 1991) are susceptible to *T. spiralis* infection. The list of herbivorous animals, the meat of which may provide the source of *T. spiralis* invasion for humans should include reindeers from Siberian tundra (Bessonov, 1981) and camels from Africa (Bommer *et al.* 1980).

The presented data indicate that herbivorous animals susceptible to *Trichinella* spp. invasion may provide an unexpected source of invasion in humans. The clinical course of trichinellosis may be benign or atypical, creating several diagnostic difficulties.

Therefore, the need arises for determination of *Trichinella* spp. species responsible for human morbidity using the accessible nowadays techniques (Zarlenga *et al.* 2001, Pozio and Zarlenga 2005, Pastusiak 2006, Gottstein *et al.* 2009, Pozio *et al.* 2009).

In cases of large epidemic foci the invasive dose of *Trichinella* originating from infected carcasses used to be dispersed among large numbers of individuals and, therefore, large epidemics used to manifest a generally more benign clinical pattern. When index-case manifest a benign, transient blepharodema and pseudo-influenza symptoms, it may cause a delayed action of sanitary-epidemiological and medical services.

The recent data indicate that within last years the number of familial and individual cases of trichinellosis increases in which, most frequently, it proves difficult to identify source of invasion and which used to provide diagnostic problems (Seroka 2000, Gołąb and Sadkowska-Todys 2006). In such individuals the course of the disease used to be severe, manifesting a high invasion intensity, which may be explained by a high invasive dose of *Trichinella*, reduced to a narrow group of persons. The diagnostic difficulties reflect also omission of the accurate epidemiological analysis in the patients, so significant and helpful in directing diagnostic efforts.

Ocular signs/symptoms provide one of the first signs/symptoms of the developing disease process, advising the

physician to suspect invasion of *Trichinella* spp. They are of significance in therapeutic management and in prognosis of the disease sequels.

Pathomechanism of lesions in trichinellosis

The pathomechanism of lesions in trichinellosis is conditioned by development of two *Trichinella* spp. generations, i.e. of its mature forms in small intestine and of larval forms in muscular tissue. The intestinal invasion determines development and subsequent course of the disease and its sequels. Elimination or destruction of the mature forms of *Trichinella* spp. small intestine using anthelmintics of benzimidazole group may alter the local and the systemic reaction of the host, may reduce invasion of larvae to muscles and alter the pattern of the disease (Kocięcka 2006).

Already in the 5th day of invasion newborn larvae (100–120 µm in length, around 6 µm in diameter) penetrate from the small intestine wall to lymphatic capillaries and, then, to blood circulation. Passing to the large circulation they may penetrate all organs and tissues, including organ of vision but they settle only in the selected biotope, in striated skeletal muscles. The migrating larva increases its dimensions at least tenfold and with a fully developed stichosome it invades cells of striated muscles in which it encapsulates or not, depends of *Trichinella* species (i.e. *T. pseudospiralis*). The nurse-cell-larva complex arises, forming a separate unit within the host tissues. Around the invaded muscular cells a network of capillaries develops (Baruch and Despommier 1991), of immense significance for metabolic exchange between the parasite and the host, which explains the prolonged penetration of *Trichinella* antigen to blood (Gomez-Morales *et al.* 2004, Bruschi *et al.* 2005). This is of a particular significance for maintenance of prolonged cell-mediated and humoral immune response at the late stage of trichinellosis.

Basophilic transformation of skeletal striated muscle cells (Gabryel *et al.* 1969, 1995) represents a typical trait, preconditioning their penetration and promote nesting, development and survival of *Trichinella* larvae in the altered cell. The excretory-secretory antigen (glycoprotein 49 kDa, 53 kDa – strong immunogenic) released from stichosome stichocytes (Despommier 1974, Despommier and Müller 1976) turns on the chain of immunopathological phenomena, among which the immediate type hypersensitivity reaction plays a leading role in acute pathology of trichinellosis with involvement of organ of sight.

The factors playing a significant role in pathomechanism of respective lesions include mast cells, neutrophilic granulocytes, lymphocytes T and B, their subpopulations and eosinophils. The cells provide source of several mediators and cytokines, inducing cell-mediated and humoral responses. The subsequent stage of the pathological process involves production of *Trichinella* antigen-specific antibodies (IgE, IgM, IgA, IgG), formation of immune complexes, their deposition on cell membrane of mast cells and their degranulation. The

released cellular mediators (slow reacting substance of anaphylaxis – SRS-A, prostaglandins PGD₂, PGF₂, PGI₂; platelet aggregating factor PAF) and preformed mediators (histamine, eosinophil chemotactic factors – ECHF-A); kinin protease together with the released bradykinin are responsible for development of disturbances in microcirculation, they lead to augmented permeability of capillary walls and, due to their dilatatory effect on smooth muscles of blood vessels, they aggravate the pathology: fluid, electrolytes and cell elements pass to the surrounding tissues. Due to its rich vascular supply, organ of vision is particularly susceptible to action of the factors at the early stage of the invasion: oedema of eyelids, periorbital oedema and occasionally facial oedema develop dynamically and are typical for early stage of trichinellosis.

Vasculitis in the course of trichinellosis, histologically expressed by fine intravascular thrombi, cellular infiltrates around capillaries and extravasations of various extent develops in the organ of vision and in many other organs (pulmonary tissue, brain, myocardium, wall of small intestine, muscles and nail beds).

Presence of immune complexes, accumulation of serotonin in high amounts, augmented numbers of neutrophils, their destruction and production of interleukin 1 (IL-1) provide source of endogenous pyrogen, which acts on center of thermoregulation promoting pyrexia at the acute stage of the disease. Involvement of cytokines network in pathomechanism of lesions in the course of trichinellosis has been suggested for several years. In experimental studies involvement of still other cytokines has been proven, mainly of IL-3, IL-4, IL-9, IL-10, which act as growth factors for mast cells, playing a significant role in immune processes in trichinellosis. In experimental animals IL-2, IL-3, IL-4 and INF gamma were found to accelerate clearance of mature forms from alimentary tract while IL-5 induced slower elimination of intestinal forms of *Trichinella* (Grencis *et al.* 1994, Mink *et al.* 1994). GM-CSF factor was found to induce proliferation, differentiation and activation of granulocytes and macrophages, the involvement of which in trichinellosis has been recognized longtime ago (Karmańska *et al.* 1997).

In experimental animals studies on the role and significance of free radicals and antioxidants in defence processes in the course of the invasion showed that macrophages represented the principal source of nitric oxide (NO) synthesis by inducible nitrogen oxide synthase (iNOS). This proved the significant role of macrophages in host's protective mechanisms in the course trichinellosis (Boczoń *et al.* 2004, Hadaś *et al.* 2002, Wandurska-Nowak and Wiśniewska 2002, Fabré *et al.* 2007). This points to a significant role of macrophages in host's protective mechanisms in the course of trichinellosis.

Eosinophilia represents an inherent trait of pathology even in trichinellosis of a benign or asymptomatic course. Eosinophils are induced by mediators (ECHF-A), released by mast cells, and IL-3 and IL-5 cytokines stimulated by TH1 and TH2 as well as by complement components C₅, C₆, C₇. Interaction between the factors and adhesin VCAM-1 and

eosinotactin plays an important role in accumulation of eosinophils at the place of parasite residence in tissues. The known for a long time bidirectional action of eosinophils involves, on one hand, release of enzymes, histaminase and arylsulphatase, which degrade, respectively, histamine and SRS-A, and on the other, eosinophils exert a cytotoxic effect on larvae mediated by their production of toxic superoxides (H₂O₂, O₂, OH). The phase of biochemical killing of the parasite involves action of eosinophiles-released major basic protein (MBP) and cationic peroxidases, ECP (eosinophil cationic protein) and EP (eosinophil peroxidase) (Wassom and Gleich 1979, McLaren 1980, Kazura and Aikawa 1980, Ruitenber 1981, Ruitenber and Buys 1986, Prin and Dubucquoi 1998, Bruschi and Murrell, 2002. Bruschi *et al.* (2008) in the review stressed that eosinophils could, have a protective role in trichinellosis because they are cytotoxic against the newborn larvae in an antibody-dependent cellular system. However, a prolonged exposure to activated eosinophils might cause tissue damage of many organs (muscles, myocardium, central nervous system and vascular walls) in severe form of human trichinellosis. In our opinion the damage of vascular system walls in the organ of vision due to intensive eosinophils activation and degranulation in the acute stage of trichinellosis – is possible.

Eosinophilia appears in blood already in the first days of invasion, before manifestation of clinical signs/symptoms and increases in intensity in days 6 to 12 of the invasion, i.e. during intestinal stage of trichinellosis. An abrupt increase in eosinophiles number is noted between days 18 and 24 following invasion, which is followed by a slow decrease in their number for a long time, occasionally reaching 3 to 4 months. Studies of clinicians demonstrated (Kassur *et al.* 1978, Kocięcka and Budzyńska 1985) than no correlation can be detected between the increase in eosinophilia on one hand and invasive dose of *Trichinella* and intensity of invasion in muscle tissue or severity of clinical course on the other.

Nevertheless, some patients with a severe clinical form of trichinellosis manifest in morphology of peripheral blood an eosinopenia accompanied by lymphopenia (6% to 8%), pointing to immunosuppression and usually forecasting an unfavourable course of the disease (Ozieretskorskaya 1978, 1994, Dupouy-Camet *et al.* 1994b).

Pathomechanism of muscular pain in the course of trichinellosis can be explained by invasion of *Trichinella* larvae to muscle cells, development of pathomorphological lesions in muscular tissue (basophilic transformation, cell infiltrates) and in microcirculation. Also, participation of neurokinin cannot be bypassed, particularly of substance P, playing role in pain transmission. The substance represents one of mediators in the first central neuron of pain perception while at the periphery it plays the role of a co-mediator for acetylcholine and serotonin. The muscle pathology is expressed also by metabolic and enzymatic disturbances. This pertains mainly to increased levels of muscle enzymes, i.e. creatine phosphokinase (CPK), lactate dehydrogenase (LDH), its

isoenzymes (LDH₄, LDH₅) and aspartate aminotransferase (AspAT). The disturbances arise due to an increased permeability of muscle cell membranes and leakage of the enzymes to serum (Kassur *et al.* 1978, Boczoń *et al.* 1981).

Thus, the complex of above mentioned pathological factors is responsible for development of acute morbid signs/symptoms characterizing trichinellosis and manifested by pyrexia, oedema of eyelids, periocular and facial oedema, haemorrhagic lesions, muscular pains in upper and lower extremities, trunk, nape and jaw (frequently accompanied by lockjaw), by various biochemical and histopathochemical lesions in muscular tissue and in inner organs.

Clinical pathology in the organ of vision

At the acute stage of trichinellosis pathology of visual organ affects its numerous structures. At the stage lesions of *vasculitis* and *perivasculitis* type prevail. They are expressed by subjective symptoms such as photophobia, blurring, a disturbed vision and pain of eyeballs. Appearance of symmetric oedema of eyelids and periocular oedema, involvement of eyeball muscles, vascular lesions in conjunctiva, uvea, retina and optic nerve are typical. Accumulation of fluid in palpebral subcutaneous tissue, the loose connective tissue, explains development oedema of eyelids and periocular oedema at the acute stage of the disease (Fig.1). Frequency of their manifestation in various epidemic foci of trichinellosis varied from 17% to 100%. Lower percent of ocular symptoms (26%–35%) noted in foci caused by consumption of infected horsemeat (Boireau *et al.* 2000). In some cases, oedema in orbital tissues may result in exophthalmos, sometimes accompanied by an increased intraocular pressure (Gould 1970, Kostrzewski and Skalska 1975).

Circulatory disturbances within great and lesser arterial circles of the iris (*circulus arteriosus iridis major et minor*) and anterior ciliary arteries (*a.a. ciliares anteriores*), which supply ciliary body, iris, sclera, conjunctiva result in haemorrhagic lesions of variable intensity; they may involve individual, fine foci or massive intra-conjunctival haemorrhages seen along the palpebral fissure (Fig.2). The wide spread haemorrhagic lesions used to be noted in patients with severe or moderately severe course of the disease, due to a massive invasion of *Trichinella spiralis* (Kostrzewski and Skalska 1975, Przybył-Ereńska and Kocięcka 1975, Klein 1976, Kassur *et al.* 1978, Ozieretskoykaya 1978, Kocięcka 1981a, Kocięcka 1996). Frequency of their manifestation in various epidemic foci of trichinellosis varied from 9.5% to 29.8% of affected patients.

In some cases hemorrhages to fingernail beds and in oral mucosa are noted in parallel (Kocięcka 1981a, 1996). They persist for a long time after acute signs of the disease subside, leaving brown elongated streaks under nails, which provide a valuable sign, helpful in retrospective analysis of trichinellosis, particularly in cases of individual morbidity (Fig.3). Capillaroscopic studies on capillary loops of nail folds conducted in trichinellosis patients (Kocięcka and Adamski, 1985) demonstrated variability of morbid patterns manifested in most patients (46.2%) by tortuous course or elongation (40.7%) of capillary loops. The authors stressed that the capillaroscopic lesions vanished in the course of glucocorticoid treatment at the acute stage of trichinellosis.

Wandering larvae, which enter ciliary arterioles and the central retinal artery, the anatomically terminal branch of ophthalmic artery, may induce injury to retina in the course of trichinellosis. This results in irreversible damage to sight, representing a severe sequel of past disease (Gould 1970). Harrenschwald (according to Gould 1970) histologically



Fig. 1. Symmetric periorbital and eyelids oedema in patient with re course of trichinellosis/according to Kocięcka 1996, p. 72



Fig. 2. Intensive conjunctival haemorrhage in patient with moderate course of trichinellosis/according to Kocięcka 1996, p. 74



Fig. 3. Finger-nail haemorrhages in patient with moderate course of trichinellosis / according to Kocięcka 1996, p. 81

demonstrated presence of *Trichinella* larvae in retinal blood vessels and wide spread lesions with traits of endothelial degeneration in retinal capillaries. In turn, Talkovskiy (according to Przybył-Ereńska and Kocięcka 1975) within *ora serrata* in retina demonstrated a tumour surrounded by inflammatory infiltrate containing *Trichinella* larvae.

Involvement of eyeball muscles in the course of trichinellosis used to manifest itself by pain accompanying movements of eyeballs (25% to 29% of patients). It may be accompanied by paralysis of extrabulbar muscles, resulting in diplopia or nystagmus; a disturbed accommodation may reflect involvement of ciliary muscles in course of the disease, noted in infrequent cases (Klemańska 1954, Kostrzewski and Skalska 1975, Przybył-Ereńska and Kocięcka 1975, Kocięcki and Kocięcka 2004). In accessible broader clinical studies on visual organ pathology in course of trichinellosis (Kassur *et al.* 1978, Ozeretskovskaya 1978, Kostrzewski and Skalska 1975, Przybył-Ereńska and Kocięcka 1975, Klein 1976, Marinçu *et al.* 2007) no descriptions of penetration of *Trichinella* larvae to ocular muscle cells or their encapsulation could be identified. This is consistent with the earlier opinion expressed by Weatherley (1983), who informed that in experimental investigations on rats infected with *T. spiralis* (Himisch *et al.* 1961) an intense invasion was accompanied by invasion of numerous larvae in retina and uvea but no encapsulated *Trichinella* larvae were identified in cells of ocular muscles. Gould (1970) in histological studies on visual organ in rats and rabbits experimentally infected with various doses of *Trichinella spiralis* larvae detected exclusively lesions of vasculitis type. In experimental studies on *Macaca mulatta* monkeys (Fig. 4) manifesting signs/symptoms resembling those in humans (symmetric blepharodema and periocular oedema, conjunc-

tivitis, swollen mouth, muscular pain) no descriptions were identified of encapsulated *Trichinella* larvae in muscles of eyebulb (Kocięcka *et al.* 1974, Kocięcka *et al.* 1981b). In a detailed parasitological evaluation of muscular invasion intensity in a man who died due to trichinellosis of a severe course (Gerwel *et al.* 1964) trichinoscopic examination of ocular muscles detected numerous *Trichinella* spp. larvae but no penetration of muscle cells or encapsulation of larvae were detected. This might indicate a transient presence of the *Trichinella* larvae in the tissues during their migration in blood vessels.

The presented above data prompt considerations on specificity of morphologic and anatomic structure of muscles, which move the eyeball.

It is significant that motor system of the eyeball includes six muscles: four straight muscles (straight muscle of eyeball superior, inferior, lateral, medial) and two oblique muscles of eyeball (inferior, superior), manifesting histological structure of striated muscles and, thus, muscles susceptible to *Trichinella* spp. invasion. However, it should be noted that the muscles are short (5.5 mm to 7.7 mm) and manifest connective tissue sheaths stemming from sheath of the eyeball

(*vagina bulbi s. Tenoni*). To certain extent, this may make penetration of *Trichinella* larvae to muscle cells difficult but it does not exclude their invasion. The problem requires further detailed studies. Orbicular muscle / *m. orbicularis oculi*/ of eye located in the upper lid (responsible for tightening the eyelids) belongs also to striated skeletal muscles. Larvae of *Trichinella* may penetrate tarsus, consisting of dense connective tissue with numerous elastic fibres, levator muscle of eyelid /*m. levator palpebrae*/ the smooth tarsal muscles superior and inferior, reaching them through blood vessels but they fail to remain there or to encapsulate. Similarly, *Trichinella* larvae do not settle in ciliary body, the main mass of which is formed by smooth muscle (Kocięcki and Kocięcka 2004). Also iris contains two smooth muscles, the sphincter and the dilatator of the pupil, the contraction of which develops under effect of light. Also the orbital muscle /*m. orbitalis*/ passing through the orbital fissure and stabilizing position of the eyeball consists of a layer of smooth muscles.

It should be stressed that smooth muscle cells contain low amounts of myoglobin, mitochondria and cytochromes while in contrast to striated muscles the source of energy is provided by anaerobic glycolysis. Thus, it cannot be excluded that the mentioned above anatomic, morphologic and biochemic characters of motile eyeball apparatus may impair penetration of *Trichinella* larvae to cells of the muscles, unfavorably affecting their settling and encapsulation.

It can be accepted that pathological signs/symptoms in the visual organ at the acute stage of trichinellosis might represent exponents of *vasculitis* and *angiomyositis* due to general immunopathological processes, developing in the course of migration and invasion of *Trichinella* larvae to multiple organs and tissues including structures of visual organ.

Differential diagnosis

Differential diagnosis is indispensable in every case of suspicion of *Trichinella* invasion. This is particularly true in patients with atypical or oligosymptomatic course of trichinellosis.

In patients who report sudden appearance of symmetric oedema of eyelids, periorbital oedema and signs of conjunctivitis, independently of the accompanying signs, one should exclude toxic-allergic reactions, serum sickness, acute glomerulonephritis and circulatory diseases.

In patients with haemorrhagic intraconjunctival lesions leptospiroses should be excluded and invasion of *Leptospira icterohaemorrhagiae* in particular. In persons returning from travel to endemic regions of Asia or Far East coexisting haemorrhagic fevers should be considered, induced by *Hantaviruses* and haemorrhagic fever with renal syndrome in particular, less frequently dengue haemorrhagic fever induced by *Flaviviridae*. In persons returning from tropical countries with generalised muscular pains, cyclic or irregular fever, drenching sweats and intraconjunctival extravasations malaria should be excluded or confirmed and appropriate treatment



Fig. 4. Macaca mulatta monkey with periorbital oedema and mouth swollen observed on 18th day after experimental invasion of *Trichinella spiralis* according to Kocięcka *et al.* 1974

should be implemented. A coexisting infection with *Cytomegalovirus* may promote haemorrhages development of diffuse lesions of *retinitis necroticans* type (Kocięcki *et al.* 2007) should be considered.

Differentiation of a fully symptomatic trichinellosis pattern with other diseases should not bypass Lyme borreliosis, the course of which may be accompanied by *uveitis* both in frontal uvea fragment (iris, ciliary body) and its posterior portion (*chorioretinitis*). Pain, photophobia and a decreased acuity of vision are symptoms common to several diseases, also those from the group of zoonoses, not only parasitic ones (Zagórski *et al.* 2012) and they may suggest preliminary stage of invasion with *Trichinella* spp.

Quantitative estimation of cytokines (IL-6, IL-8 and vascular endothelial growth factor – VEGF) in anterior chamber aqueous humor in patients with protracted visual acuity disturbances after severe course of trichinellosis could be very important to exclude coexistence of vascular and neovascular retinal changes (Shchuko *et al.* 2013)

Disturbances in alimentary tract are also worth attentions, manifested by abdominal pains and a protracted diarrhea. Such disturbances which accompany other signs/symptoms of trichinellosis may result not only from lesions in mucosa of small intestine upon protracted intestinal invasion of *Trichinella* larvae but also from accompanying infection of alimentary tract with other pathogens. It is indispensable to bacteriologically examine feces to exclude infection with *Salmonella* spp., *Shigella* spp., *Yersinia* spp. and *Clostridium difficile* and a parasitological examination is required to exclude infection with *Giardia*, *Cryptosporidium* and in patients returning from tropical countries to exclude infection with *E. histolytica*.

Diagnosis of trichinellosis is based on epidemiological anamnesis (source of infection, number of larvae present in consumed meat – encapsulated or not encapsulated, place and number of persons in the epidemic focus), clinical evaluation and laboratory tests (blood morphology demonstrating elevating eosinophilia, accompanied by hyperleukocytosis; immunodiagnostic and a set of tests supplementing or excluding the diagnosis).

The immunodiagnostic and molecular studies confirm infection with *Trichinella* spp. and provide an indication for implementing a therapeutic management. Polymerase chain reaction (PCR) permitting detection of DNA of *Trichinella* spp. in body fluids and in tissue of the host is valuable technique when used in immunosuppressed patients, in diagnostic disturbances of the doubtful cases, as well as in the early stages of infection when the other technique may be unreliable.

Conclusions

In the light of contemporary data trichinellosis is still important public health problem. New *Trichinella* species, new cycles and distribution of parasite to many environments – cause epidemiology and clinical evaluation more complicated.

The pathomechanism of lesions and multiorgan pathology in human trichinellosis is a complex one and depending upon the intensity of invasion, the species of *Trichinella* involved and many factors of immunopathological response of the host.

Ocular system involvement in the acute stage of the diseases is characteristic in syndrom of clinical signs and symptoms of human trichinellosis and might represent of *vasculitis* and *angiomysitis* due to general immunopathological process developing in the course of migration of *Trichinella* larvae to many organs and to structures of visual system. Differential diagnosis is indispensable in every case of suspicion of *Trichinella* invasion especially in case of sporadic incidence or in patients with atypical and oligosymptomatic course of trichinellosis.

The immunodiagnostic and molecular studies confirm infection with *Trichinella* spp. and provide indication for proper therapy

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