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An Outbreak of Human Trichinellosis in the Village of Subotinac near the Town of Aleksinac

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SUMMARY

An outbreak of trichinellosis affecting 13 individuals, of whom 8 with severe form presented as myocarditis, occurred in the village of Subotinac near the town of Aleksinac in Serbia. The source of this epidemic was smoked pork from a domestic pig that had not undergone meat inspection procedures. The most striking clinical features among all the 13 patients were muscular pain (84.6%), malaise (69.2%), upper eyelid edema (61.5%), and difficulty in chewing (53.9%). These symptoms and signs were associated with significant elevations of creatine phosphokinase and lactate dehydrogenase levels. As high as 61.5% of the patients with trichinellosis were diagnosed with myocarditis (determined by means of electrocardiography, echocardiography, troponin I and creatine kinase-MB measurements). The patients had *Trichinella*-specific IgG antibodies in an enzyme-linked immunosorbent assay and positive indirect immunofluorescence test results. Patients seemed to respond well to treatment with mebendazole. Corticosteroid therapy was administered to patients with myocarditis. Since high percentage of our patients presented with myocarditis, an infectious diseases specialist should always keep in mind doing cardiac biomarkers analyses, echocardiography and electrocardiography.

Key words: **outbreak, trichinellosis, myocarditis**

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INTRODUCTION

Trichinellosis is an infection caused by ovoviviparous nematodes of the genus *Trichinella*, most commonly by *T. spiralis* (1). Infection is initiated by ingestion of viable larvae in raw or undercooked meat. Larvae are liberated by digestion. The liberated larvae develop into adult forms in the duodenum and jejunum, where they mate and produce offspring. The adult worms are expelled into the stool. Newborn larvae penetrate the intestinal wall and enter the lymphatic system after which they are transferred by the bloodstream to areas of implantation. The newborn larvae have been involved in cardiac and neurologic manifestations of trichinellosis. The life cycle of this parasite is completed with a striated muscle cell invasion by the larvae (2). The larval presence triggers a strong allergic and inflammatory reactions which results in the disease. Patients with neurologic and cardiac dysfunctions have substantial hypereosinophilia associated with formation of arteriolar microthrombi inducing areas of cerebral and myocardial infarction as a result of increased number of larvae. Immunologic reactions are also the cause of one of the major clinical findings - palpebral edema. Other clinical features (eg, fever, muscle pain) are caused by the direct trauma of the larva encysting in muscle cells, coupled with the immunologic response, after which the intramuscular cysts eventually calcify (3). Outbreaks occur in the settings where more than one person consume the same *Trichinella*-infected meat. It is estimated that approximately 10,000 cases of trichinellosis occur every year worldwide. Several different species of *Trichinella* can cause human disease. The most common species is *Trichinella spiralis*, which has a worldwide distribution and is most commonly found in pigs (4).

Here we present the clinical end epidemiological features of the human trichinellosis outbreak caused by *T. spiralis*, that occurred in the village of Subotinac, near the town of Aleksinac.

PATIENTS AND METHODS

An outbreak of human trichinellosis took place in the village of Subotinac, near the town of Aleksinac. The presumed infestation occurred during the consumption of smoked pork, during an after-funeral reception in January 15th 2012 and a week later, during memorial service in January 22nd 2012. The same group of 24 people attended both events. Thirteen individuals were exposed to the infested pork and they recalled of the exact date of the smoked pork consumption. All of them were relatives. The pork was not tested for *Trichinella*. Since the history of illness and clinical symptoms were consistent with trichinellosis, serodiagnosis for *T.spiralis* was performed at the Department of Parasitology, Public Health Institute Niš (Serbia). Indirect immunofluorescence test (IIFT) and specific IgG *Trichinella* antibodies by an ELISA were performed. Serum samples with anti-*Trichinella* antibodies titre ≥ 1:20 using IIFT were considered seropositive.

Eight patients with cardiologic symptomatology and raised levels of muscle and cardiospecific enzymes were hospitalized ("inpatient"), while the rest underwent clinical observation, analyses and therapy as outpatients. All of them had positive *Trichinella* test results. Five outpatients also turned out to have positive results of *Trichinella* analysis. Eleven patients had titres of 1:40, one patient had titre of 1:20 and one patient had titre of 1:80. The test was repeated after two weeks. Then, eleven patients had titers of 1:40, one patient had titre of 1:80 and one patient had titre of 1 > 160. Regarding the outpatients, we preserved in our database only the results of the total blood counts analyses and serology analyses results. Test results of muscle enzymes, cardiospecific enzymes and acute phase reactant were given back to the outpatients after the ambulatory infectious diseases specialist confirmed that they were in the reference range. Electrocardiography and echocardiography of the heart were performed in each patient.

Inpatient treatment at the Clinic of Infectious Diseases of the Clinical Center Nis included mebendazole, 300 mg/8h for 14 days plus prednisolone 0.5-1 mg/kg/day with progressive dose reduction over 2 weeks. One eight-year-old boy (inpatient) was treated with oral solution of mebendazole, 100 mg/8h for 14 days, without corticosteroids. Inpatients were discharged after the normalization of cardiological symptoms and improvement of cardiospecific enzymes activities. Outpatients were treated identically with the exception of corticosteroid use. Due to the lack of test kits, we did not do an analysis of human immunoglobin E levels.

The outbreak was analyzed through descriptive methodology. The methodology included a negative control group of 11 people who attended the same events, but did not eat the infested meat. All of them also had negative serology results for *T.spiralis*. There were no individuals with trichinellosis (or reported trichinellosis in that period) who did not attend these events, so we could not form a positive control group.

RESULTS

Demographic and clinical features of all the patients with trichinellosis are given in Table 1. It comprises hospitalized and non-hospitalized patients

data. Clinical features of patients with myocarditis are shown in Table 2. The results of biochemical analyses of hospitalized patients are shown in Table 3.

Table 1. **Demographic and clinical features of patients with trichinellosis**

Variables	Inpatient n = 8	Outpatient n = 5	All the patients n =13
	(% in relation to inpatients only / % in relation to all the patients)	(% in relation to inpatients only / % in relation to all the patients)	
Average age (years)	41.3 ± 18.1	66.2 ± 7.9	50.8 ± 19.3
Range	9-59	63-80	9-80
Sex, n (%)			
Male	5 (62.5/38.5)	3 (60/23.1)	8 (61.5)
Female	3 (37.5/23.1)	2 (40/15.4)	5 (38.5)
Average inpatient time (days) Range	19 ± 5.2 11-27		
Clinical manifestations, n (%)			
Fever	5 (62.5/38.5)	2 (40/15.4)	7 (53.9)
Myalgia	7 (87.5/53.9)	4 (80/30.8)	11 (84.6)
Upper eyelid edema	6 (75/46.2)	2 (40/15.4)	8 (61.5)
Edema of extremities	3 (37.5/23.1)	1 (20/7.7)	4 (30.8)
Diarrhea	1 (12.5/7.7)	3 (60/23.1)	4 (30.8)
Photophobia	2 (25/15.4)	2 (40/15.4)	4 (30.8)
Conjunctivitis	4 (50/30.8)	2 (40/15.4)	6 (46.2)
Orbital pain	2 (25/15.4)	1 (20/7.7)	3 (23.1)
Abdominal pain	3 (37.5/23.1)	3 (60/23.1)	6 (46.2)
Vomiting	1 (12.5/7.7)	4 (80/30.8)	5 (38.5)
Difficulty in chewing	5 (62.5/38.5)	2 (40/15.4)	7 (53.9)
Palpitation	4 (50/30.8)	0 (0/0)	4 (30.8)
Malaise	6 (75/46.2)	3 (60/23.1)	9 (69.2)
Average incubation, days Range	22.4 ± 9.9 15-41	27.4 ± 7.5 18-36	24.3 ± 9.1 15-41

Table 2. **Symptoms and signs of the inpatients diagnosed with myocarditis**

Symptoms and signs, n (%)	Inpatient n = 8	All the patients n = 13
Elevated troponin	3 (37.5)	3 (23)
Pericarditis	2 (25)	2 (15.3)
ECG changes	3 (37.5)	3 (23)
Subjective symptoms palpitations)	4 (50)	4 (30.7)

Table 3. **Biochemical profile of the inpatients infected with *T.spiralis***

Parameter	Range	Average	Elevated parameters levels, n(%)	
			Inpatients n=8	All the patients n=13
Erythrocyte sedimentation rate	7-50	21.8 ± 17.5	5 (62.5)	5 (38.5)
C-reactive protein	1.3-35.9	10.7 ± 15.1	3 (37.5)	3 (23.1)
White blood cells	7.3-15.1	9.4 ± 2.5	3 (37.5)	3 (23.1)
Eosinophills, absolute count	0.2-4.3	1.5 ± 1.4	6 (75)	6 (46.1)
Eosinophilles, percentage	2.2-30.6	15.2 ± 1.4	6 (75)	6 (46.1)
Creatine kinase	111.8-2,695.2	626.6 ± 857.1	6 (75)	6 (46.1)
Creatine kinase MB	14.3-68.2	33.8 ± 17.1	6 (75)	6 (46.1)
Lactate dehydrogenase	274-1,230	771.2 ±291.4	7 (87.5)	7 (53.8)
Hydroxybutyrate dehydrogenase	88.6-373.6	256.6 ± 90.8	6 (75)	6 (46.1)
Myoglobine	43.87-1,852.2	629.72 ± 679.6	6 (75)	6 (46.1)
Aspartate aminotransferase	22-256	74.2 ± 75.3	6 (75)	6 (46.1)
Alanine aminotransferase	24.1-126.5	71.6 ± 37.2	6 (75)	6 (46.1)
Troponin	0-0.2	0.05 ± 0.06	3 (37.5)	3 (23.1)

DISCUSSION

Trichinellosis cases are reported to the Center for Diseases Control and Prevention (CDC) far less frequently now than in the past in the United States of America. In the decade 1940-1950, when the U.S. Public Health Service began recording the number of trichinellosis cases, an average number of 400 cases per year in the USA was reported. During 2008-2010, an average of 20 cases per year were reported to CDC each year. The overall number of an average number of cases reported has decreased by virtue of advanced and ameliorative pig farming practices, commercial and home handling with the pork meat and raised public awareness. The number of cases associated with raw or undercooked meats has not substantially changed over time. Over the past 40 years, only a small number cases of trichinellosis have been reported in the USA. The risk of trichinellosis from commercially raised and properly prepared pork meat is very low nowadays (4).

Over the past decade, trichinellosis has reemerged in Eastern Europe. An outbreak in Serbia, from December 2001 to January 2002, including 309 people, gave sufficient information regarding the causes for this reemergence. It has been proven by public health research that the immediate causes of the epidemics in Serbia were: the consumption of smoked sausages produced by private slaughterhouses and meat producers, failure of implementing the procedures of in-house control of meat and assurance of quality, insufficient number of experienced veterinary control personnel and their replacement with inexperienced stuff, replacement of large meat establishments with more than 1,000 small slaughterhouses which could not afford proper inhouse inspection, and an increase in smallholder swine farming with decreased level of government control. A 300% increase in pig infection in Serbia and a concomitant large increase in human trichinellosis outbreaks were a result of not providing high meat-producing standards. Before 1990, swine trichinellosis in Serbia was confined to four small districts. Nowadays, about one third of the Republic is considered endemic for trichinellosis (5).

The length of incubation period varies upon the severity of the disease. In severe forms, the incubation

period is not long (approximately one week). In moderate infestations, the incubation period is prolonged (approximately two weeks). In the benign forms, the incubation period usually lasts for 3 to 4 weeks. During the incubation period, a diarrheal syndrome can precede the typical signs and symptoms of the disease (6). As shown in Table 1, the incubation period in our patients is in keeping with the established incubation period, regardless of the severity of the disease. Having in mind that the number of patients is insufficient for adequate statistical analysis, we could not observe a relation between severity of disease and period of incubation. In spite of the facts mentioned above, the patients with severe form of trichinellosis did not have short incubation periods. However, one of our inpatients had an incubation period in duration of 41 days, and unexpectedly he was diagnosed with myocarditis (three days before hospitalization he felt heart palpitations and then elevated troponine values were found).

The presence of other clinical features shown in Table 1 is in accordance with the data available in scientific literature, with a small degree of deviation that leaves room for discussion. Heavy infections can cause severe cardiovascular or neurological complications, periorbital oedema, myositis, and rarely death (7, 8). In this case, having cardiovascular complications and myositis, our inpatients were diagnosed as having a serious form of trichinellosis; however, only 87.5% of them had myalgia, 75% had periorbital oedema, and the expected percentage is high above 90% up to 100% (8).

Myocarditis due to *Trichinella spiralis* is not caused by the direct larval invasion and encystation of the heart muscle. It is probably induced by an eosinophil-enriched inflammatory response which results in eosinophilic myocarditis. This process is similar to the pathogenesis of tropical endomyocardial fibrosis. Organ specific autoantibodies are produced in some patients with trichinellosis, but their role in the process of the disease is still not understood. The heart muscle could be affected by *T. spiralis* in 21-75% of infested patients (9, 10).

Cardiovascular complications can appear even in moderate and not only severe cases of trichinellosis. They occur usually in the later course of infestation between the third and the fourth week. Myocarditis is observed in 5-20% of all infested patients. The symptoms include chest pain, tachycardia and pathologic electrocardiogram (T waves flattening, ST segment lowering, lowered QRS complex, atrioventricular or interventricular conductance disturbances) (6). All of the inpatients of this study developed cardiac manifestations diagnosed as toxic myocarditis, as shown in Table 2. In relation to the total number of patients with trichinellosis (13 patients), patients with myocarditis make up 61.5%, which is markedly high. However, there are similar data of other epidemics analyses (9, 10). Our inpatients presented with subsequent disorders: atrial fibrillation, reversal of the arm leads and right axis deviation QRS(T) contour abnormality consistent with right lateral myocardial ischemia (associated with fine granulary appearance of interventricular septum seen in echocardiography) and low electrocardiographic QRS voltage in the limb leads (associated with pericarditis appearance in echocardiography).

We did not register any case of skin rash in our patients, although trichinosis patients may develop splinter hemorrhages of the fingernails, and nonspecific skin rashes (urticarial, maculo/paular, etc.) (11).

Erythrocyte sedimentation rates (ESR) are usually not increased in trichinellosis. However, increased ESR and C-reactive protein (CRP) could be a reflection of developed myocarditis, as was the case in our inpatients (12, 13).

Eosinophillia is the cause of leukocytosis. Leukocytosis can occur in 65% of patients, with the total white blood cells up to 24,000/μL. The neutrophil and lymphocyte counts are usually normal. Increased eosinophilia is an obligatory characteristic in trichinellosis. It appears early, 10 days after infection, before the appearance of clinical signs, and it achieves its maximum between the second and the fifth week of the disease. Eosinophilia regresses slowly and may persist from several weeks to three months. Increased eosinophil count is higher in patients with neurological complications. However, no linear relationship between the clinical course of trichinellosis and the hypereosinophillia level has been found. The total number of eosinophils could reach up to 8700/μL (40%-80% of total WBC) (14). As it could be noticed in Table 3, the highest eosinophil percentage in inpatients was 30.6%. Analyzing the patient data individually, patient by patient, we could not notice a relationship between eosinophil count and severity of diseases, particularly subjective symptomatology, muscle and cardiospecific enzyme activities. Also, outpatients had similar results of eosinophil count. Their mean leukocyte count was 6.7 ± 1.8, mean eosinophil value was 8.1 ± 7.5% , i.e, 0.5 ± 0.4 as an absolute count.

Activities of all muscle enzymes are increased in the serum during trichinellosis: creatine phosphokinase (CPK), lactic acid dehydrogenase (LDH), aldolase and occasionally aspartate aminotransferase (AST) and alanine aminotransferase (ALT). In 75%-90% of all trichinellosis patients, levels of CPK are increased (several-fold,

even up to 17,000 U/L) between the second and the fifth week of the disease. An increase in CPK blood activity does not correlate with the clinical severity of the disease, although it seems to correlate with the intensity of muscle pain. The CK isoenzyme myocardial band (CKMB) may indicate an affection of the hearth muscle. On the other hand, as many as 35% of patients without myocardial involvement may have elevated CK-MB activeties. Activities of LDH isoenzymatic forms (i.e LDH fraction 4 [LD4] and LDH fraction 5 [LD5]) are increased in 50% of patients. The total LDH is raised in 76% to 86% of patients. α-hydroxybutyrate dehydrogenase (LDH-1-iso-enzyme or HBDH) is increased in 62% (6, 15-19). Analyzing the percent of raised activities of muscle specific enzymes in our inpatients compared to the total number of patients with trichinellosis, we could note surprisingly low values. Compared to the number of the inpatients only, i.e patients with severe form, the percents are in accordance with the aforementioned scientific literature (Table 3).

Results of serology analyses are not positive until 2-3 weeks after infestation. The values reach their maximum levels around the third month and may persist for years. It is important to note that serology ratios do not correlate with the disease severity or the clinical course. A strong positive result of serology, i.e a high ratio, usually indicates an early infection (20).

Therapy of trichinellosis includes anthelmintics to eradicate adult worms, then corticosteroids for anti-inflammatory action, and nonsteroidal anti-inflammatory drugs or salicylates for pain management. Although anthelmintics have effect only in intestinal phase of the disease, most patients still continue to harbor adult worms in the intestines during the acute phase of infestation. Therefore, all patients with confirmed or suspected trichinellosis should be given antiparasitic medication to prevent continuation of newborn larvae production. Therapy with mebendazole is also effective in reducing the risk of increased deposition of the larvae, which is especially high in patients who receive corticosteroid therapy. We treated our patient in accordance with the contemporary guidelines (21, 22).

CONCLUSION

Unfortunately, *Trichinella* infection is not uncommon in Serbia due to insufficient public awareness. In order to save money, local pig farmers do not implement veterinary meat control. Therefore, they expose their families and people from their surroundings to possible *Trichinella* infection and its severe complications. In this case, there is a need for a better cooperation between veterinarians, epidemiologists and infectious disease specialists. Since high percent of our patients presented with myocarditis, infectious disease specialists should always keep in mind performing cardiac biomarkers analyses, echocardiography and electrocardiography.

References

1. Akkoc N, Kuruuzum Z, Akar S, et al. A large scale outbreak of trichinellosis due to Trichinella britovi in Turkey. Zoonoses Public Health 2009; 56(2):65-70. https://doi.org/10.1111/j.1863-2378.2008.01158.x

2. Blaga R, Durand B, Antoniu S, et al. Dramatic increase in the incidence of human trichinellosis in Romania over the past 25 years: impact of political changes and regional food habits. Am J Trop Med Hyg 2007; 76(5):983-6.

3. Bruschi F, Tassi C, Pozio E. Parasite-specific antibody response in Trichinella sp. 3 human infection: a one year follow-up. Am J Trop Med Hyg 1990; 43(2):186-93. https://doi.org/10.4269/ajtmh.1990.43.186

4. http://www.cdc.gov/parasites/trichinellosis/epi.html. Parasites - Trichinellosis (also known as Trichinosis). Epidemiology & Risk Factors.

5. Djordjevic M, Bacic M, Petricevic M, et al. Social, political, and economic factors responsible for the reemergence of trichinellosis in Serbia: A Case Study. J Parasitol 2003; 89(2):226-31. https://doi.org/10.1645/0022-3395(2003)089[0226:SPAEFR]2.0.CO;2

6. European Commission. Health & Consumer Protection Directorate-General, European Commission Health in Europe, ec.europa.eu>docs

7. Taylor WR, Tran GV, Nguyen TQ, et al. Acute febrile myalgia in Vietnam due to Trichinellosis following the consumption of raw pork. Clin Infect Dis 2009; 49(7):e79-83. https://doi.org/10.1086/605533

8. Rahul KS, Raghavendra N, Mohanty S, et al. Clinical & biochemical profile of trichinellosis outbreak in north India. Indian J Med Res 2014;140(3):414-9.

9. Kirschberg GJ. Trichinosis presenting as acute myocardial infarction. Can Med Assoc J 1972; 106 (8):898-9.

10. Compton SJ, Celum CL, Lee C, et al. Trichinosis with ventilatory failure and persistent myocarditis. Clin Infect Dis 1993;16(4):500-4. https://doi.org/10.1093/clind/16.4.500

11. Walsh DS, Jonqsakul K, Watt G. Hand rash in trichinosis. Clin Exp Dermatol 2001;26(3):272-3. https://doi.org/10.1046/j.1365-2230.2001.00813.x

12. Boroń P, Jezyna C, Klimowicz J. C-reactive protein (CRP) as an index of non-specific syndrome of the acute phase in correlation with leukocytosis and erythrocyte sedimentation rate in the study of trichinasis. Wiad Parazytol 1975;21(4-5):703-10.

13. Pozio E, Murrell KD. Systematics and epidemiology of trichinella. Adv Parasitol 2006; 63:367-439. https://doi.org/10.1016/S0065-308X(06)63005-4

14. Kociecka W. Trichinellosis: human disease, diagnosis and treatment. Vet Parasitol 2000; 93(3-4):365-83. https://doi.org/10.1016/S0304-4017(00)00352-6

15. van der Laarse A, Davids HA, Hollaar L, et al. Recognition and quantification of myocardial injury by means of plasma enzyme and isoenzyme activeties after cardiac surgery. Br Heart J 1979; 41(6): 660–7. https://doi.org/10.1136/hrt.41.6.660

16. Froscher W, Gullotta F, Saathoff M, Tackmann W. Chronic trichinosis. Clinical, bioptic, serological and electromyographic observations. Eur Neurol 1988; 28(4):221-6.

17. Tassi C, Materazzi L, Pozio E, Bruschi F. Creatine kinase isoenzymes in human trichinellosis. Clin Chim Acta 1995; 239(2):197-202. https://doi.org/10.1016/0009-8981(95)06113-R

18. Stumpf J, Kaduk B, Undeutsch K, et al. Trichinosis: epidemiology, clinical features and diagnosis (author's transl). Dtsch Med Wochenschr 1978; 103 (40): 1556-8, 1561-2. https://doi.org/10.1055/s-0028-1129300

19. Neghina R, Neghina AM, Marincu I. Reviews on trichinellosis (III): cardiovascular involvement. Foodborne Pathog Dis 2011;8(8):853-60. https://doi.org/10.1089/fpd.2010.0815

20. Rosenblatt JE. Laboratory diagnosis of infections due to blood and tissue parasites. Clin Infect Dis 2009; 49 (7):1103-8. https://doi.org/10.1086/605574

21. Gottstein B, Pozio E, Nöckler K. Epidemiology, diagnosis, treatment, and control of trichinellosis. Clin Microbiol Rev 2009;22(1):127-45. https://doi.org/10.1128/CMR.00026-08

22. Gilbert DN, Moellering RC, Eliopoulos GM, et al. The Sanford Guide to antimicrobial therapy 2011. Forty-First Edition. Table 13A (9), p.139.

Epidemija humane trihineloze u selu Subotinac u okolini Aleksinca

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SAŽETAK

U selu Subotinac, blizu grada Aleksinca, došlo je do epidemije trihineloze, od koje je obolelo trinaestoro osoba, od kojih je osmoro razvilo ozbiljnu formu bolesti u vidu miokarditisa. Izvor epidemije bila je dimljena svinjetina poreklom od domaće svinje koja nije podlegla procedurama provere mesa. Dominantni klinički znaci i simptomi kod ovih trinaestoro bolesnikabili su: bol u mišićima (84,6%), malaksalost (69,2%), otok gornjih kapaka (61,5%) i otežano žvakanje (53,9%). Pomenuti znaci i simptomi bili su udruženi sa značajno povišenom aktivnošću kreatin fosfokinaze i laktat dehidrogenaze. Kod čak 61,5% bolesnika sa trihinelozom utvrđen je miokarditis (prema nalazima elektrokardiografije, ehokardiografije, te nivoima troponina I i kreatin kinaze-MB). Kod pacijenata su bila prisutna Trichinella-specifična IgG antitela imunoenzimskog testa i pozitivni rezultati testa indirektne imunofluorescencije. Odgovor bolesnika na terapiju mebendazolom bio je zadovoljavajući. Kortikosteroidna terapija ordinirana je bolesnicima sa miokarditisom. S obzirom da je kod visokog procenta naših bolesnika bio prisutan miokarditis, infektolozi bi uvek trebalo da imaju u vidu upotrebu analiza srčanih biomarkera, ehokardiografije i elektrokardiografije.

Ključne reči: **epidemija, trihineloza, miokarditis**