

Trichinella Infection with Multisystem Involvement in An Elderly Patient: A Case Report and Review of Literature

Nicolas Sandakly^{1,2}, Ayman Bahsoun¹, Georgio El Koubayati^{*}, Estelle Aoun¹, Randa Choueiri^{1,2}

¹Faculty of Medical Sciences, Lebanese University, Hadath Campus, Lebanon

²Department of Internal Medicine, Lebanese Hospital Geitaoui University Medical Center, Lebanon

***Corresponding author: Georgio EL KOUBAYATI, Faculty of Medical Sciences, Lebanese University, Hadath Campus, Lebanon**

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1. Introduction

Trichinosis, also known as trichinellosis, is a parasitic food-borne disease primarily transmitted through the consumption of raw or undercooked meat, most commonly pork containing *Trichinella* larvae. After ingestion, the larvae encyst in muscle tissue, leading to a wide spectrum of clinical manifestations [1]. Early symptoms, typically occurring within the first few days, include mild gastrointestinal disturbances such as nausea, vomiting, diarrhea, and abdominal pain. More severe systemic manifestations, such as myalgia, periorbital edema, myositis, myocarditis, encephalitis, or cerebral vasculitis, generally appear 10 to 14 days post-infection [2]. Although trichinosis can mimic other common illnesses, it is rare to observe severe cases with multi-organ involvement. Herein, we report the case of a 72-year-old male who presented with chest pain, generalized weakness, fever, and periorbital edema, and subsequently was found to have cardiac and neurological complications secondary to *Trichinella* infection.

2. Case Presentation

A 72-year-old male patient presented to the emergency department with severe, acute, non-radiating substernal chest pain at rest, described as a pressure-like sensation. His past medical history is significant for Parkinson's disease. Upon admission, the patient was febrile and tachycardic with normal blood pressure. Review of system revealed myalgia and generalized weakness. His face was notable for diffuse edema. The Electrocardiogram (ECG) performed upon admission revealed sinus tachycardia, ST-segment elevation in lead avR, and ST depression in the lateral leads. Initial laboratory investigations revealed elevated troponin T level at 2000 ng/L and leukocytosis at 11,000 cells/mm³ (reference range 4800-10800 cells/mm³) with eosinophilia (27.4%). Serum creatine kinase was elevated at 350 U/L (reference range 125-240 U/L). The patient was admitted to the critical unit for evaluation of suspected myocarditis and eosinophilia.

Further history revealed that two weeks prior to admission, the patient had consumed meat with his family. Three days later, he developed new-onset abdominal pain and diarrhea. Five days later, he noted periorbital edema, for which he was prescribed topical steroid eyedrops by his ophthalmologist. The edema got worse and extended to his face. His daughter and his wife who had dined with him reported similar manifestations.

Physical examination is significant for limb weakness that is more pronounced in the lower extremities with motor power graded at 1/5 in the lower limbs and 3/5 on the upper limbs. Tendon reflexes were diminished. A chest Computer Tomography (CT) scan revealed mild to moderate pericardial effusion. Brain Magnetic Resonance Imaging (MRI) showed white matter ischemic lesions features suggestive of inflammatory vasculitis. Cerebrospinal Fluid (CSF) analysis via lumbar puncture, did not reveal any albumin-

cytologic dissociation ruling out Guillain-Barre syndrome. Secondary causes of eosinophilia were investigated. No evidence of atopy was found. Auto-immune workup, including anti-neutrophil cytoplasmic antibodies (ANCA), was negative (Table 1).

Table 1: Autoimmune work-up

Investigation	Results	Normal values
ANA	Jan-80	Not Available
SS-A native (SSA)	10	0-91 U/mL
SS-B (SSB)	15	0-73 U/mL
Anti ds DNA IgG (IU/mL)	5.1	0-29.9
Anti Sm (U/mL)	2	0-7
ANCA-C (AU/mL)	3	≤19
ANCA-P (kU/L)	0.2	<1.4
C3 (g/L)	1.6	0.9-1.8
C4 (g/L)	0.3	0.1-0.4
Angiotensin-converting enzyme (U/L)	50	16-85

In light of these findings, the patient was started on albendazole 400mg twice daily with pulse methylprednisolone 500mg twice daily. Electromyography (EMG) of the lower limbs revealed fibrillation potentials and positive sharp waves in several muscles. Motor unit potentials were of short duration and low amplitude suggestive of myopathy with no conduction abnormalities. An MRI of the thigh was done to guide muscle biopsy to confirm the diagnosis. On the fourth day of hospitalization, serum anti-Trichinella spiralis IgG level measured using indirect ELISA test, came back positive. Moreover, the muscle biopsy obtained on the same day from the right quadriceps showed degenerative changes, bundles of skeletal muscles with eosinophilic and mononuclear cell infiltrates, as well as coiled larvae inside the muscle fibers confirming the diagnosis of trichinella infection (Figure1). The patient was transferred out of the critical unit following significant clinical improvement in the following days in consciousness and motor strength along with laboratory improvement. He was discharged to continue albendazole and tapering steroids with colchicine one milligram per day, along with rehabilitation. His daughter and wife were also treated with albendazole after testing positive for Trichinella infection. Follow-up revealed complete resolution of his symptoms.

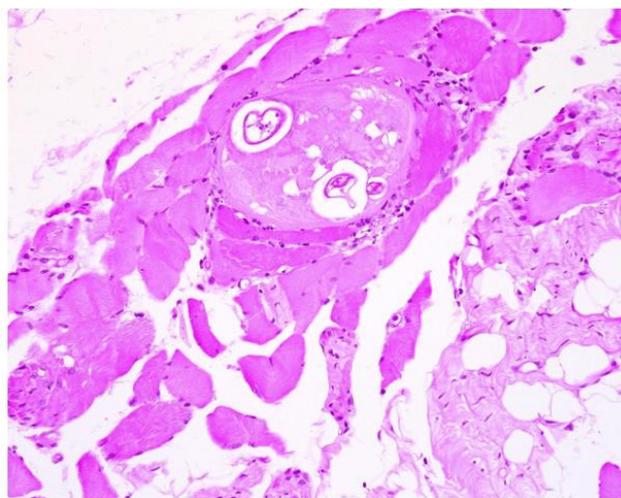


Figure 1: Muscle biopsy revealing Trichinella larvae

4. Discussion

In Lebanon, a trichinosis outbreak occurred in 1995, affecting approximately 200 individuals after the consumption of pork meat from an unregulated butcher [3]. Khalil et al. [4] raised concerns about a potential resurgence of trichinosis cases in Lebanon following the high inflation rate and the resort of some Lebanese to wild boar hunting, as an alternative source of animal protein. Trichinella has a well-defined life cycle. After ingestion, the acid medium of the stomach digests the cysts releasing larvae that invade the intestinal mucosa. After copulation, the male larvae die and the viviparous female adults produce newborns; marking the

enteral phase which lasts 24-48 hours. It is usually asymptomatic, however diarrhea, abdominal pain, nausea, vomiting may occur [5]. The newborn larvae then enter the lymphatic and hematogenous circulation preferentially invading the skeletal muscle fibers. This marks the parenteral phase which typically lasts 7-14 days. It is characterized by myalgia, periorbital edema and fever. Nevertheless, it has been suggested that the severity of clinical presentation in acute trichinosis might correlate with the host factors and the infectious dose [6].

As we describe above, in the case of this 72-year-old male, there were two distinct stages related to his clinical manifestations aligning with the known phases of *Trichinella* infection. In fact, he presented with non-specific symptoms related to the enteral phase, followed by complications characteristic of the parenteral phase. He developed myocarditis mimicking acute myocardial infarction, myopathy and cerebral vasculitis. A retrospective survey conducted in a Romanian hospital reported trichinellosis-related complications in 48 out of 558 patients. Among these, 44 had single complication, and only one patient presented with myocarditis, respiratory failure and central nervous system involvement [7]. Additionally, in an overview of 17 epidemiological studies including 5268 cases, cardiovascular events were reported in 26% patients, with myocarditis being the most commonly reported cardiac complications [8]. *Trichinella*-related myocarditis is thought to be caused by the larval migration into the myocardium, inducing an inflammatory response with eosinophilic degranulation. Lachkar et al. [9] reported the case of 59-year-old male patient who presented with the classical symptoms of trichinellosis complicated by an asymptomatic myocarditis detected by an elevated serum troponin despite normal ECG, electrocardiography and cardiac MRI. This finding suggests the potential utility of systematically screening for cardiac involvement using serum troponin levels in suspected cases of trichinellosis. Similarly, neurological complications in trichinellosis may arise through both direct parasitic invasion and immune-mediated mechanisms. Central nervous system involvement is reported in as few as 0.2%, however, almost half of the cases represent serious infection [10]. A systematic review of 168 neurotrichinellosis cases reported between 1906 and 2019, revealed that the most common clinical symptoms were headache, confusion and delirium, whereas sensitive impairment was uncommon. Of the 124 cases with documented outcomes, 20 results in death and almost half reported headache and disorientation and had normal cerebrospinal fluid (CSF) findings [11].

In our case, the initial presentation of chest pain with elevated troponin and ST-segment changes raised suspicion for acute coronary syndrome, however, peripheral eosinophilia, and fever coupled with the patient's clinical manifestations and a history of consuming undercooked pork, shared with two family members who developed similar symptoms raised strong clinical suspicion for trichinellosis, which was subsequently confirmed via muscle biopsy that remains the gold standard in confirming larval presence. Our patient received albendazole and a tapering course of corticosteroid resulting in significant clinical improvement over the following days. However, a residual motor deficit persisted at discharge requiring rehabilitation. At follow-up several months later, the patient had fully regained motor function, and his eosinophil count had returned to normal.

Conclusion

This case highlights the potential for *Trichinella* infection to present with severe and multi-system involvement, including myocarditis and cerebral vasculitis. It underscores the importance of maintaining a high index of suspicion for trichinellosis in patients presenting with non-specific gastrointestinal or systemic symptoms, eosinophilia, fever, orbital oedema and a relevant dietary history—especially in regions where wild or underregulated meat consumption may occur. Early recognition and timely initiation of anti-helminthic and supportive therapy are critical to preventing serious complications and improving outcomes.

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