TRICHINOSIS

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Introduction

Trichinosis is an intestinal and tissue infection of humans and other mammals caused by the nematode *Trichinella spiralis*. The disease is characterized by diarrhoea during the development of the adult parasites in the intestine and by myositis, fever, prostration, periorbital oedema, eosinophilic leucocytosis and occasionally, evidence of myocarditis, pneumonitis or encephalitis during the stage of larval migration in tissues. Trichinosis is a common disease throughout the world contracted by eating meat containing viable cysts of *Trichinella spiralis*. The disease is transmitted by the ingestion of inadequately cooked meat. Smoked meat are also dangerous because they are not cooked. We report a case of Trichinosis detected in a patient who presented at the civil OPD with features of severe generalized muscle aches and urticaria and diagnosis confirmed by a muscle biopsy.

Case Report

A 30-year-old female presented at the civil OPD with features of diarrhoea of two days duration associated with abdominal pain and nausea. This was followed by high grade fever associated with chills and prostration. She had severe generalized muscle ache, a red eye suggestive of conjunctivitis and also maculopapular rashes more on the trunks. She had muscle tenderness, more on the deltoid muscle area. A swelling was detected around both the eyes. A history of uncooked pork ingestion was given by the patient.

Relevant investigations revealed Hb 10gm%, TLC 12000/cmm., with eosinophilic leucocytosis of more than 50%. Blood for Malarial parasite was negative. Urine RE detected albumin 1+, Stool RE was negative for ova/cysts. Creatine phosphokinase, and aspartate transaminase were elevated. Based on above findings and a high degree of clinical suspicion, a deltoid muscle biopsy was performed; part of the biopsy material was compressed between glass slides and examined under low power of microscope for presence of larvae. The rest of the portion was submitted for routine processing. H & E sections revealed larvae of *T spiralis* enclosed within a membrane produced by host muscle cells (nurse cell). There was severe myositis and the inflammatory reaction was both lymphocytic and eosinophilic. There was also focal baso-

philic degeneration of muscle fibres (Fig-1).

Discussion

Trichinosis in humans is contracted by ingestion of meat containing the encysted larvae of *Trichinella spiralis*. The meat has almost always been pork, but for the past several years 10 percent of cases reported in the USA have been attributed to feral meat, usually bear or walrus [1]. There are no intermediate hosts, and both the adults and larval stages develop in the same animal. Soon after ingestion, the larvae are liberated from their cysts by gastric digestion and migrate into the intestinal mucosa, where copulation takes place. The male dies, and within a week, the viviparous female is discharging larvae which enters vascular channels and are distributed throughout the body. The larvae enters skeletal muscle, grow and begin encysting within three weeks; calcification of cysts begins in six to eighteen months. The life span of the encysted organ has been estimated to be upto ten years. The muscle of the diaphragm, tongue, eye, del-

Fig. 1 : Lower arrow depicts the coiled larvae of trichinella spiralis within a skeletal muscle cell. Upper double arrow shows a collagenous capsule. Surrounding the coiled larvae is lymphoplasmacytic inflammatory infiltrate.

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toid, pectoral, gastrocnemius and intercostal muscles are often affected. Large outbreaks are usually caused by consumption of ready to eat pork sausage prepared in noninspected facilities at home. The chief reservoir for man is the infected pig. More stringent regulations, a decrease in garbage feeding of pigs, sterilization of garbage and deep freezing of pork have markedly reduced the incidence of the disease in many countries [2]. The larvae becomes enclosed within a membrane produced by the host muscle cells which persists for the life of the larvae. This remarkable biological coexistence of skeletal muscle cell and larvae has led to the muscle cell being called a "nurse cell" [3]. Death of the larva and its nurse cell incites an inflammatory reaction characterized principally by lymphocytes and eosinophils. In time the dead larva calcifies. Therefore, infection in both animals and man can be recognized by microscopic identification of the viable or calcified larvae within striated skeletal muscle fibres. During active Trichinosis there is focal basophilic degeneration of muscle fibres. During invasive phase cell destruction can be widespread and lethal. Thus, in the heart, acute inflammatory changes are found during the early stages in the form of a patchy but scattered interstitial myocarditis, but the larvae do not become encysted within the myosites. Invasion of the CNS is reflected by a diffuse lymphocytic and monocytic infiltration in leptomeninges and development of focal gliosis. The most typical features of the disease are fever, muscle tenderness, eosinophilia and swelling of the eyelids [4]. The eosinophilia can account for 70% of the TLC. After the third week of the disease, serologic tests are positive. A skin test is also available. Muscle biopsy best taken near the tendinous insertion of the deltoid is definitive even earlier. Following methods in diagnosis are adapted: stool test for adult worms-rarely recovered [5], blood exam for leucocytosis with an ascending eosinophilia, serological tests like complement fixation, precipitin and bentonite flocculation test. Skin test by intradermal injection of 0.1 ml of 1 in 10000 dilution of Bachman antigen is available. These serological tests all become positive during the third week of the disease and may remain positive for a few years. A newly introduced, highly sensitive ELISA test is able to detect specific antibody in the first week of infection [6,7]. Other serological tests include countercurrent electrophoresis and the indirect fluorescent antibody test. Muscle biopsy when carried out during third week of the disease remains the most useful test for demonstrating the larva. Differential diagnosis includes Hodgkins disease, eosinophilic leukaemia and polyarteritis nodosa

and dermatomyositis [7]. Treatment with Thiabendazole 25 mg/kg bid for 7 days, has resulted in relief of muscle pain and tenderness and lysis of fever. Patients with angioedema, urticaria, CNS and myocardial manifestations should be treated with prednisolone 20 to 60 mg/day. Mebendazole 400 mg tid for 2 weeks has also been used [7,8]. Prevention includes adequate cooking of pork which involves heating all portions of meat at 110°F. Freezing procedures require a temperature -15°C for 20 days. Proper smoking and pickling also destroys larvae.

Conclusion

Trichinella spiralis infection is not commonly encountered in our country and has not been frequently reported. Usually the disease is endemic in countries like the USA and in Greenland where feral meat is widely consumed besides pork. Even in our modern generation where meat hygiene is strictly adhered to we still may come across a case like this.

The commonest lesion expected in the muscle biopsy in this case will always be cysticercosis and we were surprised to identify the coiled encysted larvae of *Trichinella spiralis* instead. The most typical features of the disease as mentioned earlier are fever, muscle aches and periorbital oedema. Our case presented with diarrhoea as the predominant symptom followed by fever and muscle aches.

The case highlights a pathological lesion in a skeletal muscle fibre not often encountered in our country. We should be aware of this clinical entity to take necessary action in case of a break-out of Trichinosis. Treatment with Thiabendazole 25 mg/kg x 7 days is effective and relieves symptoms in a short time.

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