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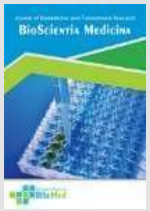

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Accidental Finding of Suspected Cysticercosis Without Symptoms in Balinese Male: A Case Report

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A B S T R A C T

Background: Cysticercosis is one of the illnesses that the WHO considers to be neglected tropical diseases (NTD). Prevalence of cysticercosis in Asia ranged from 0.8% to 41.8%. Mostly, the central nervous system is affected. **Case presentation:** A 56-year-old Balinese male came to emergency department with main complaint weakness on right-side of his body since 12 hours before being admitted to the hospital. He has history of eating lawar (Balinese traditional food) mixed with fresh pig blood; the last time he ate it was 6 months. Chest examination no nodule/swelling, abscess, or hypertrophy of muscle was found. On laboratory examination WBC $7.47x10^3$/ul, Hb 16.0 g/dL, percentage of eosinophil 5.4%, and eosinophil count $0.40x10^3$/ul. Head CT without contrast suggesting sub-acute cerebral infarction in the left capsule internal. The patient then performed chest radiograph and accidentally multiple rice grain calcification was found. The patient treated with albendazole 15mg/kg/bb/day for 14 days. Clinical presentations of cysticercosis can vary from those with no symptoms to those symptomatic, Patients with muscular involvement are mostly asymptomatic. Increasing eosinophil in complete blood count may indicate helminth infection, to clinch the early diagnosis radiological modalities can be used. However histological findings will give a definitive diagnosis. The use of praziquantel and albendazole is the suggested antihelminth in cysticercosis. **Conclusion:** Pulmonary muscle involvement of cysticercosis is a rare finding. Increasing awareness of such lesions may lead to early diagnosis and prevent irreversible damage.

1. Introduction

Cysticercosis, a parasitic disease caused by tapeworm larvae *Tape soles*, is one of the diseases categorized by the World Health Organization (WHO) as a neglected tropical disease (NTD). This disease has a significant impact on global public health, especially in areas with poor sanitation and unhygienic farming practices. Infection occurs through ingestion of *T. solium* eggs which contaminates food, water, or undercooked pork. Once swallowed, the eggs hatch in the upper digestive tract, releasing larvae which then penetrate the intestinal wall and migrate to various body tissues via the bloodstream. The central nervous system (CNS) and eyes are the main sites of cysticercosis infection, which can cause neurocysticercosis (NCC) and ocular cysticercosis (OCC). NCC is the most serious manifestation of cysticercosis, which can cause seizures, headaches, neurological disorders, and even death. OCC can cause vision problems, including blindness. Apart from the CNS and eyes, *T. solium* larvae can also infect striated muscle and subcutaneous tissue, although this is less common. The prevalence of cysticercosis varies throughout the world, with the highest rates of infection found in endemic areas such as Latin America, Sub-Saharan Africa, and Asia. In Asia, the

prevalence of cysticercosis ranges from 0.8% to 41.8%, with Thailand reporting the highest number of cases. Most individuals with cysticercosis of muscle do not show any symptoms, so the disease often goes undiagnosed and unreported.[1-4]

After eggs of *T. solium* are swallowed, oncosphere larvae are released in the small intestine. The oncosphere then penetrates the intestinal wall and enters the bloodstream, spreading to various body tissues. In the target tissue, the oncosphere develops into cysticercus larvae, which are the infectious stage of the parasite. Cysticerci can survive for years in host tissue, causing a chronic inflammatory reaction and granuloma formation. The location and number of cysticerci in the body determine the clinical manifestations of cysticercosis. NCC occurs when cysticerci infect the brain or spinal cord, causing a variety of neurological symptoms. OCC occurs when cysticercus infects the eye, causing visual impairment. Muscle cysticercosis occurs when the cysticercus infects striated muscle, usually without symptoms. However, in some cases, muscle cysticercosis can cause muscle pain, weakness, and swelling.[5,6]

Diagnosis of cysticercosis involves a combination of clinical examination, imaging (such as a CT scan or MRI), and serologic tests. Clinical examination can identify signs and symptoms associated with NCC, OCC, or muscle cysticercosis. Imaging can visualize cysticerci in infected tissue, while serology tests can detect antibodies against *T. solium* in the patient's blood. Treatment of cysticercosis depends on the location and number of cysticerci, as well as the patient's clinical symptoms. Antiparasitic drugs, such as albendazole or praziquantel, are used to kill cysticerci. Corticosteroids may be used to reduce inflammation and prevent neurological complications. Surgery may be necessary to remove a cysticercus that is causing severe or life-threatening symptoms.[7,8]

Pulmonary cysticercosis is a rare manifestation of cysticercosis, in which the cysticercus infects lung tissue. These cases often have no symptoms or only cause mild symptoms, such as coughing, shortness of breath, or chest pain. Diagnosis of pulmonary cysticercosis can be challenging because the symptoms are nonspecific and can resemble other lung diseases. Imaging, such as a CT scan or MRI, can help visualize cysticerci in the lungs, while serology tests can confirm the diagnosis.[9] In this case report, we present a case of suspected cysticercosis with pulmonary muscle involvement.

2. Case Presentation

A 56-year-old Balinese male came to the emergency department with the main complaint of weakness on the right side of his body for 12 hours before being admitted to the hospital. He also complained of difficulty speaking and one-sidedly dropping his face. He has a history of uncontrolled hypertension, other disease was denied. He has a history of eating lawar (Balinese traditional food) mixed with fresh pig blood; the last time he ate it was 6 months before admitted to the hospital. On physical examination, he was fully alert with GCS E4V5M6. Vital signs; blood pressure:171/87 mmHg, heart rate 112 times/min, respiration rate 22 times/min. Paresis on cranial nerve VII Dextra-Supranuclear, XII, flaccid hemiparesis dextra Gr 1/1; babinsky reflex +/-. Chest examination was normal, and no nodule/swelling, abscess, or hypertrophy of muscle was found. On laboratory examination WBC 7.47x10^3/ul, Hb 16.0 g/dL, Hematocrite 48.3 %, platelet 280x10^3/ul, percentage of eosinophil 5.4%, and eosinophil count 0.40x10^3/ul. Liver and kidney function within normal limits. Head CT without contrast was performed, the finding was sub-acute cerebral infarction in the left capsule interna and no finding that refers to involvement of cysticercosis in the central nervous system (Figure 1). On the chest radiograph, was found multiple rice grain calcifications on the soft tissue of the right and left hemithorax dd. Cysticercosis, and cardiomegaly with aortosclerosis (Figure 2). On chest CT without contrast there are multiple calcifications on right and left lung and pleura, suspecting pulmonary manifestation of cysticercosis; multiple rice grain in right and left intramuscular thorax anterior and posterior, right and left shoulder until

cervical suspecting cysticercosis and aortosclerosis (Figure 3). In this patient, we collaborated on treatment with a neurologist and cardiologist. Albendazole with dose 15mg/kg/day was given to the patient for 14 days.

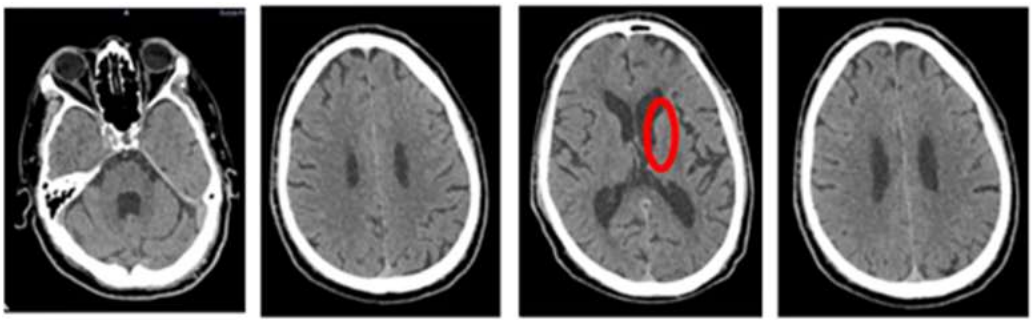

Figure 1. Sub-acute cerebral infarction in the left capsule internal (red circle).

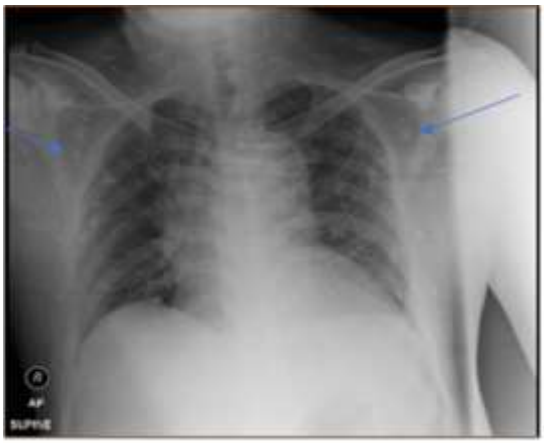

Figure 2. Chest radiograph. Multiple rice grain calcification (blue arrow).

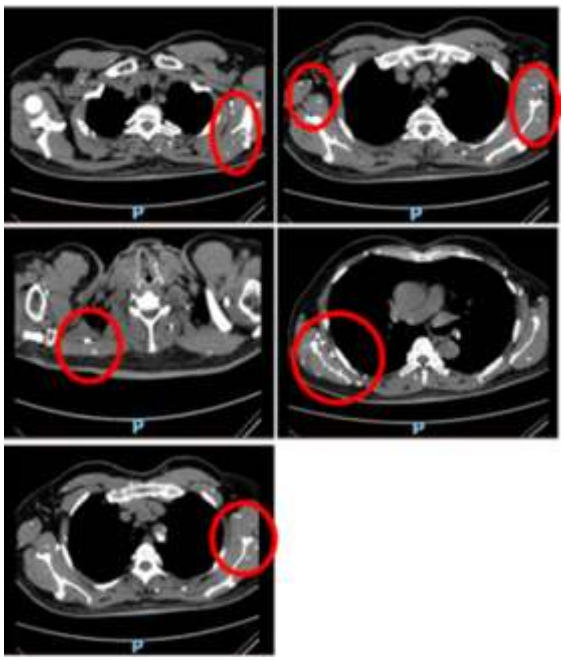

Figure 3. Chest CT. Multiple rice grain calcification (red circle).

3. Discussion

Pulmonary cysticercosis, a rare manifestation of *Taenia solium* parasitic infection, presents unique diagnostic and therapeutic challenges. The case we report highlights the complexity of this disease and underscores the importance of a multidisciplinary approach in clinical management. *T. solium* infection occurs through the consumption of raw or undercooked pork containing cysticercus larvae. Once ingested, the larvae migrate from the intestines to various body tissues, including the brain, muscles, eyes, and lungs. Infection begins with the consumption of raw or undercooked pork contaminated with cysticercus larvae from *T. solium*. In the small intestine, stomach acid and digestive enzymes break down the outer wall of the cysticercus, releasing viable larvae. Cysticercus larvae penetrate the intestinal wall and enter the blood vessels. Through blood circulation, larvae can spread to various organs, including the lungs. Cysticercus larvae have an affinity for lung tissue. After reaching the lungs, the larvae exit the blood vessels and penetrate the lung tissue. In the lung tissue, cysticercus larvae develop into fluid-filled cysts. This cyst can grow slowly and trigger an inflammatory reaction around it.[10-12]

The host's immune system recognizes the cysticercus as a foreign body through specific antigens on the surface of the parasite. Cysticercus antigen triggers the activation of various immune cells, including helper T cells, cytotoxic T cells, and B cells. Activated B cells differentiate into plasma cells that produce specific antibodies against cysticercus antigen. These antibodies bind to the parasite, marking it for destruction. Cytotoxic T cells play a role in destroying cells infected with cysticercus. Cytotoxic T cells release cytotoxic substances that damage the parasite cell membrane. The immune response to cysticerci often results in granuloma formation. Granulomas are collections of immune cells that attempt to confine and destroy parasites. The immune response that should protect the body from cysticercus infection can backfire if it is not controlled.[13-15]

Activated immune cells release various inflammatory mediators, such as cytokines and chemokines. These substances increase blood vessel permeability, attract more immune cells to the site of infection, and cause local inflammation. Immune cells also release proteolytic enzymes, such as matrix metalloproteinases (MMPs). This enzyme can damage lung connective tissue, including elastin and collagen fibers which are important for lung elasticity. During the inflammatory process, immune cells produce oxygen and nitrogen free radicals. These free radicals are reactive and can damage lung cells, proteins, and DNA. Damage to lung tissue can irritate the respiratory tract and trigger a cough reflex. Inflammation and damage to lung tissue can disrupt the exchange of oxygen and carbon dioxide gases, causing shortness of breath. Inflammation of the pleura (the membrane lining the lungs) can cause chest pain, especially when breathing deeply.[16,17]

Prolonged chronic inflammation resulting from cysticercus infection can trigger an abnormal healing process, known as fibrosis. Fibroblasts are the cells responsible for producing collagen, the main protein in connective tissue. In fibrosis, fibroblasts are overactivated and produce excessive collagen. Excess collagen is deposited in the lung tissue, forming dense, stiff scar tissue. The scar tissue that forms undergoes a remodeling process, where the collagen fibers are arranged randomly and irregularly. This disrupts the normal architecture of the lungs and reduces lung elasticity. Fibrosis reduces the lung's ability to expand and contract normally, thereby reducing lung capacity. Thick, stiff scar tissue inhibits the exchange of oxygen and carbon dioxide gases, causing hypoxemia (low blood oxygen levels). Fibrosis can cause increased blood pressure in the pulmonary arteries (pulmonary hypertension), which can lead to right heart failure. Tissue damage and fibrosis formation are serious consequences of an uncontrolled immune response to pulmonary cysticercus infection. This condition can significantly impair lung function and cause life-threatening long-term complications.

Therefore, it is important to control the immune response appropriately to prevent or minimize damage to lung tissue.[18,19]

Adult tapeworm *T. solium* lives in the human small intestine. These worms can grow up to several meters in length and consist of many segments called proglottids. Each proglottid contains thousands of eggs. The mature proglottids will be separated from the tapeworm's body and come out with human feces. *T. solium e*ggs are highly resistant to the environment and can survive in the soil for months. Pigs become infected after ingesting *T. solium* eggs found in environments contaminated with human feces. In the pig's intestines, the eggs hatch into oncosphere larvae. Oncosphere larvae penetrate the pig's intestinal wall and enter the blood vessels. Through blood circulation, the larvae spread to various tissues of the pig's body, especially skeletal muscles. In the muscle tissue of pigs, oncosphere larvae develop into cysticerci. Cysticercus is a cyst-shaped larva filled with fluid and the scolex (head) of tapeworms. Humans become infected with cysticercosis after consuming raw or undercooked pork containing cysticercosis. In the human intestine, the cysticercus develops into an adult tapeworm, and the cycle begins again. Cysticerci have an oval shape and vary in size, ranging from a few millimeters to several centimeters. The anterior part of the cysticercus is equipped with a scolex, which is the head of a tapeworm that has hooks and suckers. This scolex is important for attaching to the host's intestinal wall. The cysticercus is surrounded by a cyst wall consisting of several layers. The cyst wall functions as a protector against the host's immune system.[18-20]

Pulmonary cysticercosis often causes no symptoms or only causes mild symptoms such as coughing, shortness of breath, and chest pain. These symptoms are nonspecific and can resemble other lung diseases, making early diagnosis difficult. Imaging, especially CT scans and MRI, is essential in the diagnosis of pulmonary cysticercosis. Cysticercus cysts in the lungs can appear as round or oval lesions with well-defined walls. The calcifications and scolex visible within the cyst are characteristic features that help differentiate pulmonary cysticercosis from other conditions. Serological tests, such as ELISA and Western blot, are used to detect antibodies against *T. solium* in the patient's blood. This test has high sensitivity and specificity, so it can confirm the diagnosis of cysticercosis. It is important to increase awareness about pulmonary cysticercosis among medical personnel and the general public. Early diagnosis and appropriate treatment can prevent serious complications and break the chain of transmission of this disease.[19,20]

Treatment of pulmonary cysticercosis requires a comprehensive approach involving pharmacologic therapy, close monitoring, and surgical intervention if necessary. The main goals of treatment are to kill parasites, reduce inflammation, prevent complications, and improve the patient's quality of life. Anthelmintic drugs such as albendazole and praziquantel are the mainstay in the treatment of cysticercosis. This drug works by disrupting the parasite's metabolism, causing paralysis and death of the cysticercus. Anthelmintic therapy should be closely monitored because parasite death can trigger a severe inflammatory reaction. Increased neurologic symptoms, such as headache, seizures, or changes in mental status, should be evaluated promptly. Common side effects of anthelmintics include indigestion, headaches, dizziness, and allergic reactions. Patients should be informed of these potential side effects and instructed to immediately report any unusual symptoms. Corticosteroids, such as prednisone or dexamethasone, are often given with anthelmintics to reduce inflammation caused by parasite death. Corticosteroids help prevent neurological complications and reduce symptoms such as headaches and seizures. The dose and duration of corticosteroid therapy varies depending on the severity of the disease and the patient's response to treatment. Close monitoring is necessary to avoid long-term side effects from corticosteroid use.[21,22]

Surgery may be needed in cases where the cyst is large causing compression of vital organs, the cyst is

infected or has ruptured, and complications such as pneumothorax (air in the pleural cavity) or hemoptysis (coughing up blood). Common surgical procedures include thoracotomy (opening of the chest wall) to remove cysts, drainage of the pleural cavity, or repair of damaged lung tissue. After completing therapy, patients should undergo regular monitoring to ensure the effectiveness of treatment and detect possible relapses. Repeat imaging, such as a CT scan or MRI, may be performed to evaluate cyst resolution and monitor disease progression. Clinical management of pulmonary cysticercosis requires an individualized approach based on the patient's clinical condition, the size and location of the cyst, and the presence of complications. Collaboration between infectious disease specialists, pulmonologists, surgeons, and radiologists is essential to ensure optimal treatment results.[22,23]

4. Conclusion

Pulmonary muscle involvement of cysticercosis is a rare finding. The symptoms of muscular involvement are generally asymptomatic. While hematologic findings are non-specific and non-conclusive, radiological evaluation is often needed to clinch the diagnosis early. Increasing awareness of such lesions may lead to early diagnosis and prevent irreversible damage.

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