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Cerebellar Tremor Caused by Meningoencephalitis Tuberculosis in A 17-Year-Old Patient: A Case Report

Johansen[1]*
[1]Faculty of Medicine, University of Tarumanegara, Jakarta, Indonesia

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***CORRESPONDENCE:**
Johansentarumanagara@gmail.com

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Abstract: Meningitis Tuberculosis (TB) is a rare manifestation of extrapulmonary TB and can progress to Meningoencephalitis. Cerebellar tremor is a tremor in the head caused by cerebellar diseases such as strokes, tumors, multiple sclerosis, and certain drugs. This study describes a case of a 17-year-old boy who was infected with TB meningoencephalitis with cerebellar tremors. A 17-year-old boy came to the emergency room at K.R.M.T. Wongsonegoro Hospital with the main complaint of shaking head. The patient was diagnosed with meningoencephalitis TB and cerebellar tremors. Meningitis TB occurs due to the hematogenous spread of primary pulmonary TB or from rupture of the subependymal tubercles into the subarachnoid space and can progress to Meningoencephalitis. Clinical manifestations that can appear are a combination of meningitis and encephalitis and movement disorders caused by encephalitis. The management obtained in this patient was TB drugs, corticosteroids, and anti-seizures. Cerebellar tremor is one of the clinical manifestations caused by Tuberculosis encephalitis that must be watched out for to get adequate treatment.

Keywords: cerebellar tremor, meningoencephalitis, tuberculosis, child

INTRODUCTION

Tuberculosis (TB) is one of the oldest diseases known and the leading cause of death worldwide caused by the bacteria Mycobacterium tuberculosis.[1,2] The disease most often attacks the lungs, although other organs are involved in up to a third of cases. The bacteria that cause TB are most often transmitted from people with infectious pulmonary TB through droplets that spread through coughing, sneezing, or talking.[3] This disease is often seen in children in areas with high TB transmission. Progression of TB disease can occur, especially in children and people with immune disorders. Almost any organ system can be infected, and the organs most commonly involved are the lymph nodes, pleura, genitourinary tract, bones, joints, meninges, peritoneum, and pericardium.[2,4]

Tuberculous meningitis is a rare manifestation of extrapulmonary TB but remains the most common form of Central Nervous System (CNS) manifestation of TB. This disease can involve the meninges, brain parenchyma, and spinal cord (He et al., 2023). Tuberculous meningitis arises from hematogenous spread of infection from the lungs or lymph nodes to the brain parenchyma, forming small tubercles (Rich foci) that rupture into the subarachnoid space or ventricles in the early weeks after infection with airborne mycobacteria.[6,7] Clinical manifestations of TB meningitis begin from the first 2-4 weeks of the prodromal period with non-specific symptoms such as fatigue, malaise, myalgia, and fever. After prodromal symptoms, meningitis symptoms will appear, such as fever, headache, stiff neck, photophobia, and vomiting.[6]

Tuberculous meningitis is reported in 1 to 2% of individuals with active TB, of which 70 to 80% of CNS-TB cases appear as TB meningitis.[8] The highest prevalence of CNS-TB is reported in developing countries, and the main moderators are Human Development Index (HDI) and HIV infection.[9] Tuberculous meningitis has a morbidity rate of 25-50% in children and more than 10% in adults and can cause many complications due to the extreme inflammatory response it elicits in HIV-uninfected patients, even when on corticosteroid therapy.[7]

Tuberculous meningitis can progress to meningoencephalitis. Meningoencephalitis is a combined disease of meningitis and encephalitis that involves inflammation of the meninges, subarachnoid space, and brain parenchyma. [1] The most important predictors of TB meningoencephalitis in children are early diagnosis and prompt initiation of treatment. An incomplete understanding of the pathogenesis, nonspecific symptoms, suboptimal performance of diagnostic tests, and the paucibacillary nature of the disease often result in a lengthy process to obtain a definitive diagnosis and adequate therapy.[9]

Cerebellar tremor, also known as titubation, is a tremor in the head, and if the manifestation is severe, it can involve the body due to cerebellar disease.[10] The most prominent symptom is an involuntary tremor that looks like a "yes" or "no" movement and can occur at any time. Titubation can occur in patients who experience processes that cause damage to the brain, such as stroke, tumors, multiple sclerosis, chronic alcohol abuse, and certain drugs.[11,12] This study describes a case of a 17-year-old child infected with TB meningoencephalitis with cerebellar tremor from the history of the treatment given to the patient.[13] The severity of this type of tuberculosis is also highlighted by the unusual way in which this case is presented, with cerebellar tremor serving as a neurological symptom. After going through a seizure, the patient, a 17-year-old teenager with tuberculous meningoencephalitis, began to improve gradually. However, in order to control the long-term neurological outcome and guarantee a full recovery, it was determined that a referral for more specialized care was required due to the condition's severity and complexity.

CASES

A 17-year-old boy came to the emergency room at K.R.M.T. Regional Hospital Wongsonegoro, with the main complaint being that the patient had been frequently shaking his head for 3 days before entering the hospital. Based on the results of the anamnesis, the patient made unconscious shaking movements of his head. Complaints are also felt continuously throughout the day, especially at night, and when the patient opens his eyes, the complaints are relieved when the patient sleeps. The patient's complaint was also accompanied by fever, which had been felt for 7 days before entering the hospital. The patient's complaint was also accompanied by fever, which had been felt since 7 days before entering the hospital, more dominant at night, and the body temperature reached 39.4°C. When the patient has a fever, he also feels chills, shortness of breath, difficulty drinking, sore throat, and pain throughout the body, starting from the patient's back. Approximately 1 hour after the fever appeared, the patient also felt his vision become blurry. He denied cough complaints. The fever began to improve on the 4th day, and on the morning of the fifth day, the patient experienced a seizure; when the patient woke up from the seizure, the patient experienced agitation.

The patient and the patient's family had never experienced something similar. One week previously, the patient was hospitalized because the patient was suffering from dengue fever and was suspected of having bronchopneumonia. After the patient had felt the complaint, the patient was taken back to Pelita Anugerah Hospital and then referred to RSUD K.R.M.T. Wongsonegoro. The patient had a history of contact with the patient's grandfather, who was known to suffer from pulmonary tuberculosis.

On physical examination, apathy was found, E3M4Vaphasia, temperature 37oC, pulse 65x/m, respiration 24x/m, and SpO2 99%. Examination of the pulmonary thorax system revealed vesicular breath sounds (+/+), rhonchi (+/-), and wheezing (-/-). Examination of the neurological system revealed a stiff neck (+), lower extremity motor strength 2222/2222, and atrophy was found in all parts of the extremities. Other physical examinations were found to be within normal limits.

Supporting examinations were carried out, such as anteroposterior (AP) and lateral chest x-rays and an MRI of the head with contrast and blood examinations. Laboratory results on April 20, 2023, showed a decrease in hematocrit 36.20%, erythrocytes 4.26/uL, creatinine 0.5 mg/dL, and an increase in SGPT 199U/L, SGOT 93 U/L, and calcium 1.28 mmol/L. The results of AP (Figure 1) and Lateral (Figure 2) chest x-rays on April 19 2023 showed suspicion of bronchopneumonia. The results of an MRI of the head with contrast on May 3 2023 showed suspicion of meningoencephalitis (Figure 3). TCM sputum examination on April 25 was negative. When in the emergency room, the patient was given therapy with Diazepam 5mg, Dexamethasone 3x5mg, and Ceftriaxon 2x1.5mg. Then the patient underwent further management in the PICU and received therapy with citicolin 2x500mg, pirazepam 3x1gram, diphenhydramine 2x5mg, clonazepam 2x0.5mg, mecobalamin 1x500mcg, folic acid 1x1 mg, vitamin B complex 2x1mg and valproic acid 3x250mg and anti-tuberculosis drug 4FDC 1x3 tab to treat TB.

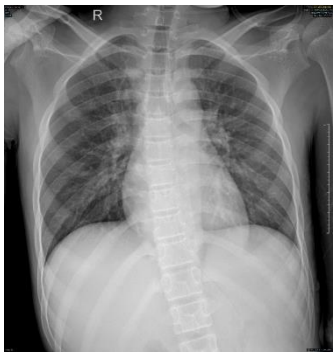

Figure 1. X-ray Thorax AP

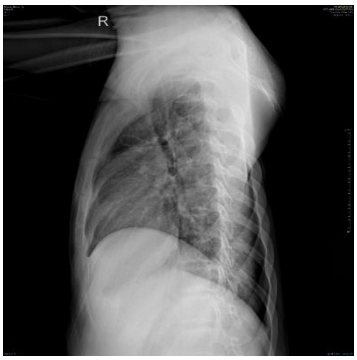

Figure 2. X-ray Thorax Lateral

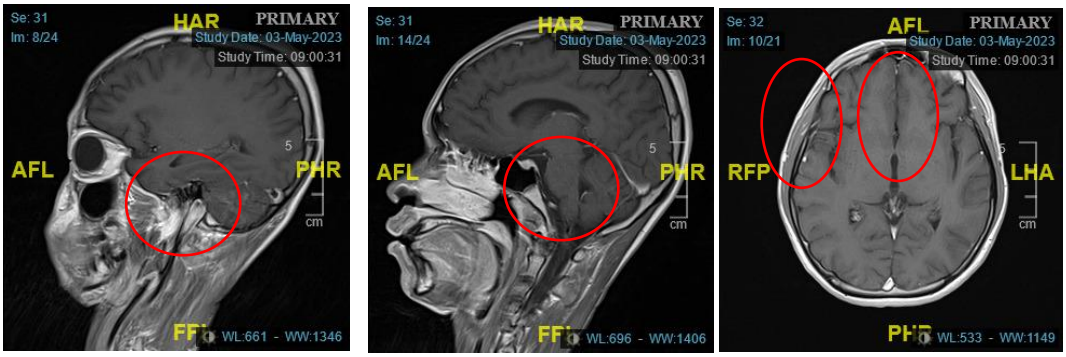

Figure 3. The red circle shows Leptomeningeal enhancement on an MRI of the head with contrast

Table 1. Laboratory Results (20/04/2023)

	Result	Unit	Normal Range
APTT Patient	23.1	second	26-34
Hemoglobin	14.1	g/dL	11-15
Hematocrit	42.2	%	40-52
Platelets Count	269	/µL	150-400
PT Control	12.2	second	
PT Patient	10.2	second	11-15
Erythrocytes	5.17	/µL	4.7-6.1
Leukocytes	8.1	/µL	3.8-10.6
INR	0.89		
Blood Glucose	79	mg/dL	70-110
Calcium	1.28	mmol/L	1-1.15
Kalium	4.00	mmol/L	3.5-5.0
Natrium	139.0	mmol/L	135-147
Creatinin	0.7	mg/dL	0.6-1.1
Ureum	41.3	mg/dL	17-43
APPT Control	27.2	Detik	
SGPT	199	U/L	0-50
SGOT	93	U/L	0-50
Procalsitonin	<0.5	ng/L	<0.5
Salmonella typhi IgM	2.00		<2: negative
Blood Culture	Tlp		

Table 2. Laboratory Results (23/04/2023)

	Result	Unit	Normal Range
Procalcitonin	<0.5	ng/L	<0.5
Blood Glucose	101	mg/dL	70-110
Calcium	1.31	mmol/L	1-1.15
Kalium	4.30	mmol/L	3.5-5.0
Natrium	136.0	mmol/L	135-147
Hemoglobin	13.5	g/dL	11-15
Hematocrit	40.7	%	40-52
Platelets Count	378	/µL	150-400
Erythrocytes	4.97	/µL	4.7-6.1
Leukocytes	14.8	/µL	3.8-10.6

Table 3. Laboratory Results (24/04/2023)

	Result	Unit	Normal Range
Epithel	4-7	/lpk	
Erythrocytes	>50	/LPB	
pH	8.0	4.8-7.8	
Protein	POS (2+)		Negative
Reduksi	Negative		Negative
Amorf	Negative		Negative
Silinder	1-2	/lpk	
Color	reddish yellow		
Crystal	Negative	/lpk	
Yeast	Negative		Negative
Leukocytes	2-4	/LPB	
Etc	Negative		
Trichomonas	Negative		
Turbidity	a bit murky		

Table 4. Laboratory Results (25/04/2023)

	Result	Unit	Normal Range
SGPT	101	U/L	0-50
SGOT	46	U/L	0-50
Amoeba	Negative		Negative
Bacteria	POS (1+)		
Smell	Typical		
Consistency	Mushy		Mushy
Worm egg	Negative		Negative
Protein Faeces	Negative		Negative
Yeast	Negative		
Carbohydrate	Negative		Negative
Etc			
Fat	POS (1+)		Negative
Leukocytes	0-2	/LPB	
Mucus	Negative		Negative
Color	Brown		
Blood	Negative		Negative
Erythrocytes	0-1	/LPB	

Table 5. Laboratory Results (30/04/2023)

	Result	Unit	Normal Range
Salmonella typhi IgM	2.00		<2 : negative
Blood Glucose	126	mg/dL	70-110
Creatinin	0.5	mg/dL	0.6-1.1
Ureum	29.8	mg/dL	17-43
Hemoglobin	11.5	g/dL	11-15
Hematocrit	36.2	%	40-52
Platelets Count	227	/µL	150-400
Erythrocytes	4.26	/µL	4.7-6.1
Leukocytes	51	/µL	3.8-10.6
SGPT	51	U/L	0-50
SGOT	19	U/L	0-50
Triglycerides	100	mg/dl	<150
Glucose (POCT) 02/05/23	120	mg/dl	70-110

DISCUSSION

Meningitis is inflammation of the meninges, which includes three membranes: the dura mater, arachnoid mater, and pia mater. This membrane lines the spinal canal, which encloses the spinal cord, and the skull, which encloses the brain. Inflammation that occurs directly in the brain is called encephalitis. If inflammation occurs in both parts, it is called meningoencephalitis.[14,15] Meningitis can occur in all age groups, from children to adults. The World Health Organization notes that meningitis cases occur worldwide with an incidence rate of 0.2 - 14 cases per 100,000 people. The highest incidence rate is in Sub-Saharan Africa, namely around 1,000 cases per 100,000 people.[16,17]

Meningitis can be caused by bacteria, viruses, fungi, and parasites. The most common bacteria that cause meningitis are *S. pneumoniae, N. meningitidis, group B streptococcus, H. influenzae dan L. monocytogenes.*[18] The most common viruses that cause meningitis are *Coxsackie Virus, Epstein Barr Virus, Herpes Simplex Virus,* and *Varicella-Zoster Virus*. Most causes of encephalitis are viruses similar to those that cause encephalitis, such as Herpes Simplex virus type 1, Varicella-zoster virus, and Epstein Barr virus. Apart from that, encephalitis can also be caused by Mycobacterium tuberculosis.[14,15,19] Risk factors for meningitis are sinusitis and otitis media caused by pneumococci, mastoiditis, alcoholism, diabetes, splenectomy, crescentic anemia, cancer, head trauma, and in individuals who do not receive vaccinations..[20] Other causes and risk factors for meningitis are immunocompromised patients and tuberculosis sufferers, especially pulmonary tuberculosis. Meningitis caused by tuberculosis is called tuberculous meningitis. It results from the reactivation of latent Mycobacterium tuberculosis infection, which causes chronic meningitis, usually developing over several weeks to several months.[10] The meningoencephalitis experienced by this patient caused the patient to show symptoms of shaking his head. It was caused by the cerebellar tremor he was experiencing, which was caused by encephalitis. As is known, cerebellar tremor has several types, one of which is palatal tremor. Slow movements in the pharynx muscles, lower face, and body characterize this tremor. The causes of palatal tremors are stroke, trauma, Behçet's disease, and encephalitis.[10]

Tuberculous meningitis results from the hematogenous spread of primary pulmonary TB or the rupture of subependymal tubercles into the subarachnoid space. Once TB bacilli gain access to the brain, the local immune system in the brain is restricted, allowing the survival and replication of TB bacteria as well as the development of TB lesions.(Davis, Rohlwink, Proust, Figaji, & Wilkinson, 2019; Manyelo, Solomons, Walzl, & Chegou, 2021) Based on postmortem studies, Rich and McCordock found that TB meningitis was preceded by the rupture of one of these lesions, a Rich focus located under the cortical pia or adjacent to the meninges or ventricles, which would release Mycobacterium tuberculosis bacilli into the subarachnoid space and cause granulomatous infection of the meninges and induce inflammation.[21,22] Tuberculous meningitis takes less than 12 months from primary infection in 75% of children. The poor outcome of this disease is caused by the body's inflammatory response, which will produce a thick exudate in the brain. The basal exudate blocks the basal subarachnoid cisterns by forming adhesions, blocking the flow of Cerebrospinal Fluid (CSF) and resulting in hydrocephalus and increased intracranial pressure.[21,22] Further progression of the exudate can result in obliterative vasculitis of small vessels proliferating and leading to the development of focal and diffuse ischemic brain changes. Blockage of larger arteries can cause infarction and perineuritis, resulting in

cranial nerve paralysis. Disease progression in severe cases is due to direct involvement of the brain parenchyma.(Davis et al., 2019; Manyelo et al., 2021) The course of the disease in this case follows the theory that has been explained, where the disease started from a TB infection contracted by the patient's grandfather and caused pulmonary TB disease in the patient. The infection develops into extrapulmonary TB, attacking the lining of the brain and brain tissue, causing TB meningoencephalitis.

Manifestations that can occur in meningoencephalitis are systemic symptoms such as headache, anorexia, low-grade fever, behavioral changes including apathy, and malaise, which usually appear for several weeks before signs of meningeal stimulation such as back and neck pain or stiff neck develop.[24] Some patients may have Other symptoms, such as lymphadenopathy cranial nerve defects (III, IV, VI). Cognitive impairment can occur gradually and can progress until the patient becomes comatose. Seizures can also occur in patients, especially children and those experiencing hyponatremia. Pediatric patients also often experience hydrocephalus, which will cause increased intracranial pressure.[2,24] A family history of pulmonary or extrapulmonary TB can be found in some patients.[4,24] Movement disorders can also occur due to brain lesions, manifesting as dystonia, chorea, ballism, myoclonus, and tremor (postural, kinetic, rarely at rest). This disorder may occur due to infarction, hematoma, cerebral vasculitis, tuberculoma, arachnoiditis, hydrocephalus, or brain edema.[25]

The clinical manifestations that occurred in this patient led to manifestations of TB meningoencephalitis where the patient had a history of TB contact with the patient's grandfather, involuntary movements of shaking the head, fever, pain throughout the body starting from the back, blurred vision, seizures, decreased consciousness, stiff neck, crackles in the right lung, atrophy in all extremities and inferior motor strength 2222/2222.

Supporting examinations that can be carried out consist of radiology and laboratory examinations. Radiological examinations that can be performed are CT scans and MRI, which can show hydrocephalus in 80% of children and 23% of adults. MRI examination can also show basal meningeal enhancement and thick meningeal plaque in the basal cisterns.[24] MRI examination in this case showed a picture of meningoencephalitis. Another radiological examination that can be carried out is a chest x-ray intended to see the presence of pulmonary TB.[24] Chest x-ray examination in this case showed suspicion of bronchopneumonia. Laboratory examinations that can be carried out are CSF analysis, Ziehl-Neelsen staining of CSF, and Polymerase Chain Reaction (PCR) tests, but in this case, these examinations were not carried out, but a rapid molecular test for TB was carried out, and the results were negative.[2,24] Based on the examinations that had been carried out, this patient was diagnosed with TB Meningoencephalitis with Cerebellar Tremor.

Treatment includes therapy for cerebellar tremors and tuberculous meningoencephalitis. Cerebellar tremor therapy with the choice of clonazepam 2x0.5mg. Pharmacological treatment is usually unsuccessful, but some cases show improvement after administration of carbamazepine, propranolol, or topiramate.[26] Treatment of meningoencephalitis depends on the etiology. This case was also given Ceftriaxone in the emergency room, but after a diagnosis of TB Meningoencephalitis was made, Ceftriaxone was stopped and replaced with 4FDC. Empirical treatment for TB meningoencephalitis is the same as treatment for pulmonary TB, namely rifampicin, isoniazid, pyrazinamide, and ethambutol. Empiric treatment is justified when the clinical picture and CSF findings are suggestive of TB, even before microbiological confirmation, as timely treatment will improve the prognosis of TB. Other drugs that can be given are corticosteroids. Corticosteroids are anti-inflammatory drugs and function as immunosuppression.[27] The patient was also given anti-tuberculosis medication in four Fixed-dose drug combinations (FDC) of 3 tablets. One 4FDC contains 150 mg rifampicin, 75 mg isoniazid, 400 mg pyrazinamide, 275 mg ethambutol, and 3x5 mg dexamethasone. Prognosis depends on the etiological cause, and those younger than 30 have a better prognosis than those older.[15,28]

CONCLUSION

A 17-year-old patient with cerebellar tremor ec TB meningoencephalitis who had a history of close contact with a TB patient was presented. This disease started from a TB infection that was contracted by the patient's grandfather and caused pulmonary TB in the patient. The infection develops into extrapulmonary TB, which attacks the lining of the brain and brain tissue, causing TB meningoencephalitis. The appearance of cerebellar tremors as a clinical symptom makes this case unusual and highlights the significance of prompt identification and thorough treatment.

CONFLICT OF INTEREST

The author declared that there are no conflicts of interest regarding the publication of this paper.

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