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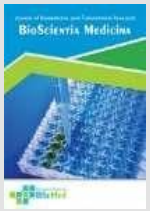

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Successful Long-Term Monitoring of Children with Scrofuloderma, Malnutrition, and HIV

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A B S T R A C T

Background: Scrofuloderma is a skin TB that occurs percontinuitatum from tissues underneath, such as lymph nodes, muscles, and bones. It often affects children and young adults. The incidence of TB disease is estimated to increase 20-fold in children with HIV infection. Poor nutrition can affect the mortality rate of children with HIV infection. This case report presents long-term monitoring of boys aged 1 year 5 months with scrofuloderma, malnutrition, and HIV infection. The case report aims to assist children and caregivers in undergoing treatment to prevent drug withdrawal and nutritional procedures and monitor the growth and development of children. **Case presentation:** A 1-year and 5-month-old boy with a complaint of ulcers on his right neck since 12 months ago. He also complained of diarrhea 12 months ago, accompanied by recurrent stomatitis and progressive weight loss since 3 months ago. He looked pale 1 month ago. Blood laboratory examination with hemoglobin 5.1 gr/dl, leukocytes 5280 / mm3, platelets 323,000 / mm3, hematocrit 20%, reactive anti-HIV test, CD4 examination with a value of 178 cells / □L, negative tuberculin test, a chest x-ray showed infiltrate in both lung fields, and bajah examination of the nodules in the Colli dextra region, with the impression of granulomatous inflammation that can be caused by mycobacterial infection. **Conclusion:** Tuberculosis is the most common opportunistic infection found in children with HIV infection and increases the mortality rate. There is a relationship between nutritional status and the incidence of mortality in HIV/AIDS children, so it is necessary to monitor children for adherence to treatment, improve nutritional status and reduce morbidity and mortality rates.

1. Introduction

Cutaneous tuberculosis accounts for approximately 1%–2% of tuberculosis cases.[1] Scrofuloderma is one of the cutaneous manifestations of tuberculosis and usually occurs when underlying tuberculosis, such as lymphadenitis, directly involves the skin.[2,3] Because scrofuloderma can appear similar to a bacterial abscess or malignancy both clinically and radiographically, a high index of suspicion is necessary for diagnosis. Although culture is the gold standard for diagnosis, smear for acid-fast bacilli and molecular techniques, such as polymerase chain reaction, canaid in diagnosis.[3] Scrofuloderma usually occurs in areas with underlying lymph nodes, such as the neck. The underlying focus of tuberculosis in lymph nodes (lymphadenitis) sometimes extends into the overlying skin and is diagnosed as scrofuloderma.[4]

Mycobacterium tuberculosis is a rather evolved complicated organism that impacts mainly the respiratory tract, despite the fact that disseminated lymphohematogenous infection can arise in an immunocompromised person. [5] Inside the host, the two pathogens, Mycobacterium tuberculosis and HIV kind 1 (HIV), seem to have a superb effect on every different, accelerating the deterioration of immune

capabilities.[6] Several research have proven lymph nodes because the maximum common of EPTB (extrapulmonary tuberculosis) infection among HIV-infected individuals.[7,8] Consistent with the latest observation, EPTB is the maximum not unusual form of tuberculosis in HIV sufferers with low CD4+ T-cellular counts, and the commonplace imparting symptoms of EPTB encompass fever, weight loss, and cough.[9]

HIV contamination is predicted to increase the threat of TB 20-fold in comparison with HIV-seronegative individuals in high HIV prevalence countries. Of the expected 8.7 million, those who evolved TB globally in 2012, 1.1 million (13%) had been expected to be HIV-coinfected.[10] Long-term observation of children with scrofuloderma, malnutrition, and HIV infection aims to assist children and caregivers in undergoing treatment to prevent drug withdrawal, monitor patient growth and development, and prepare children to face stigma as HIV sufferers.

2. Case Presentation

A 1-year and 5-month-old boy with a chief complaint is ulcers on his right neck since 12 months ago. He also complained of diarrhea 12 months ago, accompanied by recurrent stomatitis and progressive weight loss since 3 months ago. He looked pale 1 month ago. History of bleeding gums, nose, skin or gastrointestinal tract did not exist. A history of blood transfusion did not exist. Shortness of breath existed since 4 days ago, not shrinking, not accompanied by bluish, not affected by weather or activity. Ulcers on the neck, head, and back since 2 days ago. History of fever that disappears after coughing for more than 3 weeks. A history of discharge from the ear did not exist. The history of contact with long-standing cough sufferers was denied.

Physical examination appeared seriously ill, compos mentis, blood pressure 90/60 mmHg, pulse rate 120/minute, breath rate 48 x /minute, body temperature 36.6^{o}C. The child looked pale, neither ikterik nor cyanosis. Body height and weight were 70 cm and 5.8 kg, respectively, weight by age was below -3 Standard Deviation (SD), height by age below was -3 Standard Deviation, weight according to height (BB / TB) was below -3 Standard Deviation, upper arm circumference was 8 cm, the impression of nutrition status was marasmic type poor nutrition, with severe short stature. Palpable enlarged left colli lymph nodes left 2 x 1.5x 0.5 cm and submandibular 1 x 1 x 1 cm. The head is round and symmetric, with a head circumference (42 cm) and microcephaly impression according to Nellhaus standards. Hair is brownish and easily falls out. No palpebra edema, pale conjunctiva, or non-ikteric sclera was found. Normal chest shape, symmetrical chest movements, bronchivascular breath sounds, and ronki sounds in both lung basals. The heart with a normal limit of regular rhythm, and there was an inocene noise. The stomach had no distension, supel, palpable hepar 1/4-1/4 and intestinal noise was increased. On the back, there were palpable lumps as much as 2 pieces, size 2 x 1 x 0.5 cm, visible pus, and hyperemis. Buttocks examination found folded baggy pants. Warm acral limbs, good capillary refilling, positive pretibial edem, and no crazy pavement dermatosis.

Blood laboratory examination with hemoglobin 5.1 gr/dl, leukocytes 5280/mm3, platelets 323,000/mm3, hematocrit 20%, type count 0/0/4/40/46/10, ESR 86 mm, MCV 79 fL, MCH 20 pg, MCHC 26%, AST 22 u/l, ALT 9 u/l, sodium 136 mmol/L, potassium 3,2 mmol/L, calcium 9,4 gr/dl, albumin 2,1 gr/dl, ureum 15 mg/dl dan kreatinin 0,3 mg/dl, reactive anti HIV test and positive combs test result. Regular stool examination showed green color, liquid consistency, no blood, leukocytes 350-400 / LPB, erythrocytes in fecal preparations 2 / LPB, and no amoebas and parasites are found. Examination of fecal parasitology showed trophozoite Entamoeba histolytic and fecal cultures found in Escherichia colli. The results of thorax X-rays were found infiltrated in the perihiler and basal lungs. CD4 examination with a value of 178 cells / □L, negative tuberculin test, and BAJAH examination of the nodules in the Colli dextra region showed visible, microscopic clusters of

lymphocyte cells, neutrophils, macrophages, and epithelioid cells on the background of cellular debris, with the impression of granulomatous inflammation that can be caused by mycobacterial infections. The Denver examination explained the results of gross motor, fine motor, personal-social, and language were failed, with a global developmental delay.

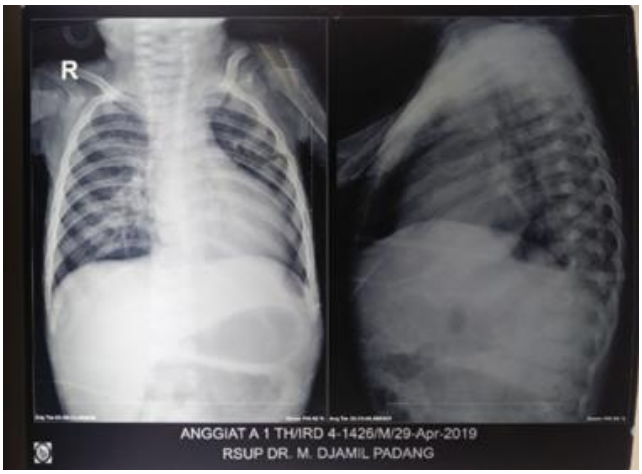

Figure 1. Chest x-ray imaging of the patient.

The child was diagnosed with sklofuloderma, opportunistic infection/HIV clinical stage IV, chronic diarrhea ec secondary immunodeficiency, oral candidiasis, pulmonary TB, marasmic malnutrition condition III, normocytic anemia, e.c. Chronic disease and global developmental delays. The child were treated with anti-tuberculosis drug, INH 1 x 75 mg, rifampicin 1 x 100 mg, pyranamid 1 x 250 mg, ethambutol 1 x 150 mg and vitamin B6 1 x 5 mg, F75 free lactose 6 x 100 cc / NGT, folic acid 1 x 5 mg PO, followed by 1 x 1 mg per oral, vitamin A 200,000 PO (single dose), zinc 1 x 20 mg and metronidazole 3 x 100 mg. Cotrimoxazole syrup 2 x 6 cc PO as adjuvant therapy in immunodeficiency patients. PRC transfusion with Hb target is 8 gr/dl to treat anemia in patients.

During the first month of observation, the child's weight was difficult to rise, and there was no vomiting and diarrhea. Children received F75 intakes per NGT at home. In the third month of observation, the child received advanced phase anti-tuberculosis drugs therapy (isoniazid and rifampicin), antiretrovirals (duviral and nevirapine), vitamin B6, and cotrimoxazole. The child's weight had begun to increase. The child received intakes of F100 per NGT. Nutritional status had increased, from malnutrition to undernutrition. Then, the child was given intake per oral.

In the fourth month of observation, the child's weight increases. The child got rice porridge and good tolerance. No longer installed NGT. The child got intake per oral. Nutritional status had increased, from undernourished to good nutrition. Anti-tuberculosis drug administration was given for 9 months.

In the ninth month of observation, the last CD4 examination was performed 932 cells/μL and CD8 740 cells/μL. Patients regularly take the drug, and there were no complaints in undergoing therapy. No therapeutic side effects were found in the patient. Lesions on the skin were no longer present and weight rised.

Denver's examination in the second semester showed improvements compared to before. Gross motorism and fine motor show passed, while personal social and language was still delayed. Monitoring in the third semester, Denver's examination showed

improvements and personal social being passed, but the language still failed. Monitoring in the fifth semester showed the child's height showed improvement, impression stunting only. Denver's examination showed passed on all developmental. The PedsQL examination showed a score of 81.25. It meant no physical, emotional and social problems were found.

3. Discussion

There have been reported cases, boys aged 1 year 5 months observed for 3 years with a working diagnosis of sclefuloderma, opportunistic infection / HIV clinical stage IV, chronic diarrhea ec secondary immunodeficiency, oral candidiasis, pulmonary TB, marasmic malnutrition condition III, normocytic anemia normochrome e.c. Chronic disease. The diagnosis was established based on anamnesis, physical examination, and supporting examination.

Scrofuloderma is a manifestation of endogenous tuberculosis infection caused by M.tuberculosis, less commonly M Bovis. The disease originates from the contiguous spread of a tuberculous focus to the overlying skin owing to local tissue destruction. This results in the formation of a painless subcutaneous nodule that breaks down and expresses purulent discharge.[2,3]

The definitive diagnosis of sklofuloderma is made by a fine needle aspiration biopsy / BAJAH / Fine Needle Aspiration Biopsy or open biopsy (open biopsy). At the examination was sought the presence of M.tuberculosis by means of culture and histopathological examination of tissues. The result of anatomical pathology can be granulomas with necrotic in the middle. There are datia langshans cells, epitheloid cells, lymphocytes, and BTA.[11]

Accelerated manufacturing of TNF has been recommended to cause expanded susceptibility to energetic TB. This has been proven in the context of individuals that certainly produce expanded ranges of TNF in response to M. tuberculosis contamination.[12] A dysregulation of Treg cells might be accountable for the sustained immune activation in HIV-infected individuals.[13] Both HIV and M. tuberculosis infect macrophages and cause the production of host inflammatory mediators that subsequently regulate the immune response and disease pathogenesis.[14] The current management of sufferers with HIV associated TB includes the provision of effective anti-TB, use of concurrent ART, prevention of HIV-associated comorbidities, management of drug cytotoxicity, and prevention/treatment of IRIS.[15]

M. tuberculosis and HIV share anatomical reservoirs together with the lung, and it has been counseled that TB patients have a microenvironment that allows HIV contamination.[16] M. tuberculosis and HIV share anatomical reservoirs together with the lung and it has been counseled that TB patients have a microenvironment that allows HIV contamination.[17]

WHO recommends the initiation of antiretroviral for any HIV-infected person who develops TB, no matter the CD4+ T-cells counts.[10] Except for the lack of ability to eradicate the virus from the infected host, a robust and sustained immune response in opposition to HIV is established on infection.[18] Endorsed first-line anti-HIV regimens are based on nonnucleoside opposite transcriptase inhibitors (NNRTI), with efavirenz as the drug of desire and nevirapine as an alternative.[15]

The management of sklofuloderma is with anti-tuberculosis drugs and local/topical procedures with a good compress or hygiene. The procedure of scrofuloderma in children must be thorough, in addition to medikamentosa must also be arranged with nutrition and the environment. Treatment in this disease is the same as treatment in pulmonary TB, by using anti-TB drugs aimed at providing faster healing, preventing resistance and recurrence.[11]

The factor that affects a person's likelihood of affecting TB infections is low endurance. One of the causes is malnutrition. The presence of TB infection and malnutrition problems in children can cause impaired growth. A child's growth can be monitored through weight per height and compared to the standard value of weight per height based on age.[19]

4. Conclusion

The treatment of pulmonary TB disease in children with HIV needs to get special attention in an effort to successfully treat and cure children. Family support is needed as an effort to make the child obedient to treatment. Families have a very important role in adherence to the treatment of children with TB-HIV co-infection.[20]

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