Disseminated Tuberculosis in a Vaccinated 9-Year-Old: A Case Report

Chinyelu Uchenna Ufoaroh[1,4]; Victor Ifeanyichukwu Modekwe[2,4]; Somto Celestine Ngonadi [3]; Ebube BelieveGod Akosa[4]; Ejike Chiedozie Okoli[4], Tito Omokhowa Asekhame[1]

[1]Department of Internal Medicine Nnamdi Azikiwe University Awka, Nigeria. [2]Department of Surgery Nnamdi Azikiwe University Awka, Nigeria. [3]Department of Paediatrics Nnamdi Azikiwe University Teaching Hospital, Nnewi, Nigeria. [4]Daystar Specialist Hospital Anambra State, Nigeria

ABSTRACT

Tuberculosis (TB) is uncommon in children vaccinated at birth as Bacille Calmette-Guerin (BCG) vaccine offers protection for a 10- to 15-year period. However, disseminated TB has been increasingly recognized in children due to the increased prevalence of immune suppression secondary to AIDS, malnutrition and immunosuppressive therapies for various medical disorders as well as increasing awareness leading to more diagnoses. We report a 9-year-old female who presented with recurrent fever, rashes, bilateral neck and abdominal swelling of 3 years duration in order to raise awareness of the occurrence of disseminated TB in our locality, the importance of a high index of suspicion and tissue diagnosis in immunosuppressed children with a negative microbiology result.

Keywords: Disseminated tuberculosis; Immunosuppression; Vaccinated child.

INTRODUCTION

Tuberculosis is an important public health problem in developing countries. In India, despite substantial efforts targeting TB and its associated risk factors, the number of cases remains high, with 2.7 million new cases per year, and paediatric TB accounting for about 10% of cases. [1]. The high burden of TB in children is well recognized, but not well quantified in many endemic areas due to problems with making a definitive diagnosis.[2]. Generally, there is still no satisfactory diagnostic reference or standard for childhood TB diagnosis.[3]. Definitive diagnosis is usually arrived at via bacteriological approach as against clinical, immunological and radiological approaches.[3].

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***Correspondence**:
Email: believegod1995@gmail.com

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Microbiological diagnosis of TB in children is challenging due to the inability to produce sputum effectively and the paucibacillary nature of the infection.[1]

Here we reviewed a case of a vaccinated 9-year-old child who came with disseminated tuberculosis.

CASE REPORT.

A 9-year-old female presented with a 3-year history of recurrent fever, rashes, and neck and abdominal swelling. Fever was initially high grade, the first episode was associated with vomiting and passage of loose stool, cough and sore throat which resolved after 1 week following treatment at a peripheral hospital. However, the fever persisted but became low grade. There was no history of convulsion or neck stiffness. There was no history of contact with a chronically coughing individual or drenching night sweats. A generalized body rash was noted about the same time as the onset of the fever, mostly itchy and pustular. Some became ulcerated as the disease progressed. Bilateral neck swelling and abdominal swelling appeared subsequently, and became progressive with associated bilateral leg oedema and facial swelling. However, there was no history change in volume nor frothiness of the urine. The patient was fully vaccinated for age. For the above complaints, she was taken to various hospitals and then to herbal homes where scarification marks were made on the abdomen without any resolution of symptoms.

Examination revealed a chronically ill-looking school-aged child, malnourished and lethargic, febrile (37.9°C), pale, bilateral pitting pedal oedema, and a generalised pustular rash. There were bilateral multiple cervical lymphadenopathies involving all groups, some measuring up to 3cm×3cm; some were matted, tender, not attached to overlying skin. The abdomen was distended with visible scarification and scratch marks. Her liver was enlarged up to 12cm below the right costal margin, along mid-clavicular line, and firm. There was massive splenomegaly and ascites.A provisional diagnosis of lymphoproliferative disorder with generalised scabies was made. Abdominopelvic ultrasound showed abdominal lymphadenopathy and hepatosplenomegaly and she had an ESR of 43mm in the first hour. The retroviral screening was negative and her genotype was AA. Serum protein revealed severe hypo-albuminaemia - 0.7g/dl, haemogram was 6.7g/dl, total white blood cell count was 6500 cells/mm^3 with relative lymphocytosis (60%). The liver function test showed elevated alkaline phosphatase. Gene expert of early morning salivary fluid was trace for *Mycobacterium tuberculosis*. Lymphoid tissue histology report showed complete effacement of nodal architecture by granulomatous nodules. These granulomatous foci were composed of epithelioid cells and a few admixed multinucleated giant cells, mostly the Langhans and foreign body types. There were foci of extensive necrosis with evidence of nuclear dusting. The overall features suggested chronic granulomatous lymphadenitis. A definitive diagnosis of disseminated TB in a malnourished child was made.

Empirical treatment with oral prednisolone, oral ivermectin, topical permethrin, antibiotics, antipyretics and blood transfusion were commenced pending the result of sputum gene expert and lymph node biopsy. Fever persisted even as the empirical treatment was ongoing. Following the results of the lymph node biopsy, anti-Koch's therapy and pyridoxine were commenced. During the initial two-month intensive phase, rifampicin, isoniazid, pyrazinamide and ethambutol were administered daily, followed by a continuation phase using daily doses rifampicin and isoniazid over a period of four months. After about 3 days of commencement of anti-Koch's her clinical condition improved steadily from less frequent fever spikes until her temperature stabilised within the normal range. Gradually the neck and abdominal swelling began to subside. She was discharged after about 2 weeks of commencement of anti-Koch's. At follow-up after two months, the hepatosplenomegaly, ascites and cervical lymphadenopathy had all completely resolved.

DISCUSSION

Reaching a definitive etiological diagnosis is vital in planning treatment while managing any clinical case.[1]. Often, clinicians evaluate patients based on detailed clinical history and systemic examination alongside minimally invasive investigations.[1]. Invasive procedures are usually resorted to when a diagnosis is difficult to make. In this index case, the fever had lasted for quite a long time and gene expert was trace for TB. This suggests that diagnosing TB sometimes can be challenging due to low sensitivity of sputum smear examination and culture.[1]. A similar challenge was noted in a study done in India where a 5-year-old child was diagnosed with disseminated TB following bone marrow biopsy as the sputum smear examination and culture were negative.[1]. In this study, the recurrent fever lasted for about 1-month before a definitive diagnosis was made as we resorted to a more invasive procedure when the traditional way of clinching the diagnosis of TB failed to solve the dilemma. Additionally, malignancy as a possible cause of the recurrent fever was ruled out just like in this index case using lymph node biopsy which also confirmed the diagnosis of TB. In this index case, there was a protein-calorie deficit which could possibly lead to immunosuppression. Even with BCG vaccination, there are still possibilities of TB infection if the immune system is compromised. Pancytopenia can occur in disseminated TB due to hypersplenism, maturation arrest, or the granulomatous infiltration of the bone marrow. Tubercular granuloma may cause pancytopenia by the replacement of marrow cells or its suppression through the release of interferon and lymphotoxin.[4]. However, this was not the case in our study as the only reduced cellular component of the blood was the red cells. BCG has about 60-80% protective efficacy against severe forms of tuberculosis in children particularly meningitis,[5,6], and its efficacy against pulmonary disease varies geographically depending on the method of administration, vaccine strain and nutritional status at the time of vaccination.[7-9]. Marais et al showed that though children less than 2 years of age were at risk of developing miliary TB, most children suffering from tuberculosis in endemic areas were older which correlates with the index case.[10]. Krujishaar and Abubakar from their study in the UK discovered that more cases of miliary TB occurred in older children and this indicated reactivation of a latent disease.[11]. This might be the case of the patient we reviewed where there may be reactivation of latent TB because of immunosuppression precipitated by malnutrition.

CONCLUSION

Disseminated tuberculosis in a BCG-vaccinated child can occur from the reactivation of latent TB or from a new infection following severe immunosuppression. A high index of suspicion and a tissue diagnosis are essential for making an accurate diagnosis especially in the face of a confounding differential such as a malignancy, and in situations where traditional methods of making a diagnosis of TB, including sputum smear examination, have proved abortive.

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