Case- Report of Tuberculosis in a Tanzanian Male Sickle Cell Disease Patient

Evelyne Modestus Banda, Siya Sijabaje

Bombo Regional Referral Hospital, Tanzania

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ABSTRACT

Sickle cell disease is a group of inherited disorders that affect hemoglobin. Normally red blood cells are disc-shaped and flexible so they can move easily through the blood vessels. In sickle cell red blood cells are misshaped, typically crescent or ‟sickl‟ -shaped due to a gene mutation that affects the hemoglobin molecule.

Tuberculosis is an infectious disease caused by bacteria that most often affects the lungs. It spreads through infected droplets when people with TB cough, sneeze or spit.

Patients with sickle cell disease frequently suffer from recurrent crises, in particular affecting the lungs, skeletal system and the abdominal organs. Tuberculosis has rarely been reported in patients with sickle cell disease. Since there is an overlap of the possible presenting symptoms of SCD complications and TB, a low index for suspicion of TB in these patients is recommended hence it is difficult to diagnose TB in sickle cell patients especially when they present with dry cough.

Here we present a case of TB infection in a 25 years old male sickle cell anemia patient who presented mainly with chronic cough that was initially dry in nature and later became productive and was associated with fever which was more marked during the evenings and later developed difficulty in breathing, shortness of breath and hypoxia in room air. He had no known history of TB contact and no history of smoking. He was first diagnosed and treated as severe pneumonia but did not respond to treatment and later was treated with Rifampicin, Isoniazid, Pyrazinamide and Ethambutol in the intensive phase and continue with Rifampicin and isoniazid in the continuation phase resulted to marked improvement.

Keywords: Tuberculosis (TB), Sickle cell disease (SCD)

INTRODUCTION

Tuberculosis (TB) remains a global health burden. One-third of the world’s population is estimated to be latently infected with Mycobacterium tuberculosis. In Sub-Saharan Africa, the prevalence of TB and sickle cell disease (SCD) are both particularly high. Sickle cell disease (SCD) is a chronic hemolytic anemia. In SCD, occlusion of blood vessels by sickled red cells leads to ischemia of the tissue supplied, and to the protean complications of the disease. Repeated splenic crisis leads to asplenia early in the first months of life, and therefore infection is a major complication of SCD, [2] and a common cause of death. Bone is the common site of frequent local infarctions that may favor the occurrence of osteomyelitis. Chest involvement is frequent, consisting mainly in acute chest syndrome, infectious pneumonia, pulmonary hypertension, and lung fibrosis. [3] Since there is an overlap of the possible presenting symptoms of chest complications found in SCD and TB it is difficult to make a diagnosis of TB in SCD and it is often misdiagnosed.

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CASE PRESENTATION

A 25 years old male with a known history of sickle cell disease since childhood, managed with hydroxyurea and folic acid regularly presented with history of cough that was dry in nature for period of three weeks and later the cough became productive but was non blood stained. This was associated with chest pain and tightness to the extent that he had difficulty breathing and shortness of breath. He also reported of low-grade fevers that were more marked during the evenings. There was also history of drenching sweats especially during the nights. The patient also had history of on and off joint pain. Past medical history is remarkable for sickle cell anemia due to history of multiple admissions for blood transfusions and treatment of various sickle cell anemia crises. He works as a petty trader and had no known recent history of TB contact or working in mining. The patient neither smoke cigarette nor drank alcohol.

On examination he was moderately pale, mild jaundiced, and no signs of dehydration. No lymphadenopathy or cyanosis noted. Was tachypneic and with mild tachycardia, and he was febrile with temperature of 37.6 ^{O} C. On respiratory system he was saturating 88% in room air and had bronchial breath sounds. Body weight of 63kg.

He was investigated for pneumonia were by chest x- ray was done which reveled infiltration in the upper zone. Full blood count was done which was suggestive of infection and severe anemia. Other basic investigations done were normal which were ASAT, ALAT, Creatinine and urea.

Table 1

Investigations Results
Full blood count
WBC: 12: Elevated suggestive of infection
(3.5-10x10^{3}/UL)
Hb 4.5 g/dL
(12-16.5g/dL)
PLT: 200
(150-450x10^{3})
Blood grouping:
A positive and compatibility test was done so as to transfuse the patient due to very severe anemia.
Chest X- Ray:
Fig 1: Infiltrations on right upper zones of the lung which was suggestive of lobar pneumonia
Sputum for gene expert:
MTB was detected, No resistance to rifampicin
Biochemistry
ASAT/ALAT 29/30
(0-32 U/L)
(0-33 U/L)
Creatinine/Urea) 57/2.4
(50-100 micromoles/L)
(2.1-8.5 mmol/L)
HIV test
Rapid test: Negative
Blood culture and sensitivity
Revealed no growth after seven days of incubation

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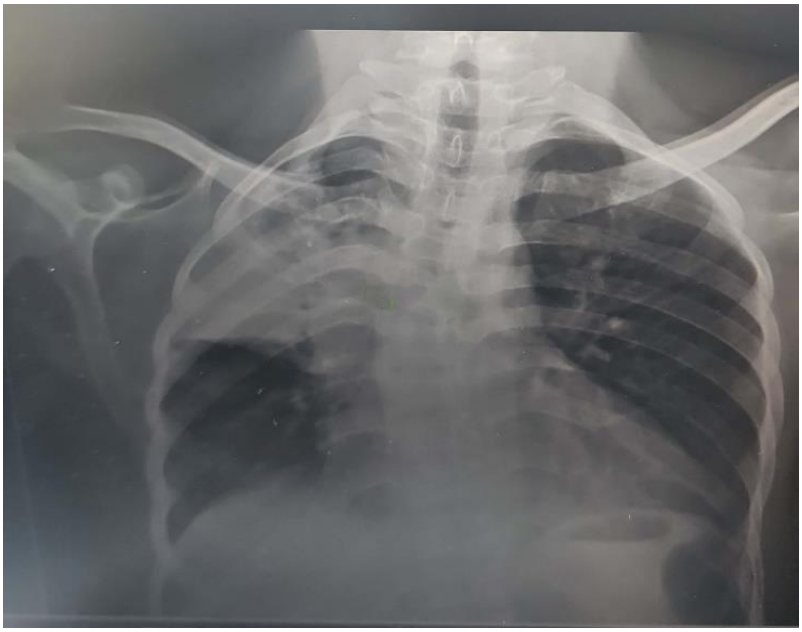

Figure 1: Chest X-ray

Management given:

Two units of blood transfusion was administered. He was also given analgesics for pain and he continued with folic acid and hydroxyurea daily. He was treated as pneumonia with amoxicillin combined with clavulanic acid together with clarithromycin but his condition worsened, he was still oxygen dependent, he still had pyrexia. After two weeks, he managed to produce sputum were by MTB was detected and anti TB was initiated which was RHZE (Rifampicin, Isoniazid, Pyrazinamide and ethambutol) in fixed combination, 4 tabs in intensive phase , he then had subsequently made an excellent recovery with marked clinical and radiological improvement and was then discharged home .After 4 weeks he came for follow up in hematology clinic , and he was better with no any B symptoms and was of good nutrition status. He completed his intensive phase for 2 months and continuation phase for 4 months. He is currently doing fine.

DISCUSSION

Sickle cell disease (SCD) is a common genetic disorder that characterized with the presence of Hemoglobin S (HbS) which changes the normal shape of red blood cells (RBCs) into crescent-shaped cells and alters their normal function of oxygen transportation and life span[4]. SCD is prevalent in Sub-Saharan, Africa, India, Saudia Arabia and Mediterranean countries. It has a diverse range of complications on health of SCD individuals. The main pathology arises from the crescent-shaped RBCs that blocks the capillaries when there is increased in viscosity [5]. SCD patients are at risk of diverse complications ranging from chronic persistent anemia to recurrent painful crisis, severe anemia and splenomegaly, as well as the recurrent encapsulated bacterial infections due to asplenism [7].

Tuberculosis (TB) is a communicable disease that is among the leading causes of death worldwide according to the World Health Organization (WHO) 2020 report, affecting around a quarter of the world population. Geographically, most people who developed TB in 2019 were in the WHO regions of South-East Asia (44%), Africa (25%) and the Western Pacific (18%). The causative organism is Mycobacterium tuberculosis, transmitted through inhalation of infected particles by coughing, sneezing or spiting. The disease mainly affects pulmonary but may affect other organs [8]. The common finding in pulmonary TB is cavitation. TB cavity site is normally in the apices of the lungs or apical part of lower lobes, the presence of this cavity forms a lifelong risk of reactivation and spreading when the cavities connect with airways releasing viable infectious bacilli, increasing the risk of spread to other humans. Another possible complication happens when infected monocytes distribute the myco-bacteria through dissemination in blood causing military TB[6].

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In spite of high prevalence of both SCD and TB with a similar geographical distribution, little is known about TB in SCD population. One paper describes the diagnosis of TB from clinical presentation of the patient which was only dry cough plus abnormality from the chest CT however our case diagnosis was confirmed from symptoms not responding to antibiotics and later sputum for gene x-pert analysis[9].

CONCLUSION

SCD is associated with impaired immune function. Loss of both humoral and cell mediated immunity are some of the mechanisms that account for immunosuppression in SCD patients. Due to immunosuppression and functional asplenia there have increased risk of infections, TB being one of them.

When compared non- SCD and SCD, TB diagnosis is similar although infectiousness seems to be lower in SCD because of in SCD usually smear is not positive and SCD are less likely to form cavitation due to low immunity.

Consent

Written informed consent was obtained from the patient and his caretaker for the publication of Case report.

Conflict of interests

The author declares that there is no conflict of interests

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