Case study

RISK FACTORS AND DIAGNOSITIC CHALLENGES OF ABDOMINAL TUBERCULOSIS: A CASE REPORT

Abstract

This a case of a 30 year old female who presented with a 4 month history of progressive abdominal swelling and mild abdominal discomfort. She had a past history of poorly treated pulmonary tuberculosis however during current symptoms the usually screening tests for tuberculosis such as the gene-xpert test, chest radiograph and acid fast bacilli smear and culture were negative creating a diagnostic challenge and considerations for alternative diagnosis to abdominal tuberculosis. Other supportive evidence and risk factors were sought for abdominal tuberculosis and treatment was initiated when other likely diagnosis where excluded. Patient made remarkable improvement after starting anti-Koch's therapy. .

Key Words : *Abdominal, Challenges, Diagnostic, Risk factors, Tuberculosis*

Introduction

Abdominal tuberculosis is one of the extra pulmonary manifestations of tuberculosis. It is caused by the mycobacterium tuberculosis complex that include *mycobacterium africanum, m. bovis, m caneitti, m.microti and m.tuberculosis.*[1] The prevalence of abdominal tuberculosis is said to be 1-3% worldwide.[2] There are various hospital estimates of prevalence across the world with the homeless, residents of long-term care facilities, prisoners and the immunocompromised being most vulnerable.[2-6] Nigeria is noted to one of the countries with a high burden of abdominal tuberculosis.[6] Abdominal tuberculosis is noted to make up to 11% of the extrapulmonary tuberculosis(EXPTB) cases.[7, 8]

Case Presentation:

A 30 year married female who was referred to the Pulmonology unit with complains of abdominal swelling of 4 months duration with associated history of moderate abdominal discomfort but had no swelling in any other part of the body. There was associated history of easy satiety and low grade fever. There were no gastrointestinal, pulmonary or other systemic symptoms during the current presentation. She had a past history of cough productive of non foul smelling whitish sputum, low grade fever, anorexia and drenching night sweat, 9 months prior to current complain for which she was diagnosed as pulmonary tuberculosis following a chest radiograph that showed patchy opacities in lung fields although gene- xpert was negative. She commenced anti-Koch's treatment for two months but stopped due to generalized persisted

joint pains following intake of the anti-Koch's medication. Her last menstrual period was a week prior to presentation. There was no history of consumption of unpasteurized milk. There was no history of exposure to anyone with chronic cough. She doesn't consume tobacco products in any form or take alcoholic beverages. She lived in a 3 bedroom flat with good ventilation in all rooms with her husband and 3 children.

Initial clinical diagnosis that was made on outpatient basis by the Gastroenterology team was of decompensated chronic liver disease(CLD) to rule out(r/o) abdominal TB, r/o ovarian cancer. Investigations including gene-xpert, acid fast bacilli smear and culture and abdominal scan which was requested for was not immediately done . She later presented a week later to the Accident and Emergency(A & E) where urea breathe test for Helicobacter pylori and fecal occult blood were both positive. A diagnosis of dyspepsia on background abdominal ascites to rule out ovarian malignancy was made and she was placed on amoxicilin/clarithomycin/rabeprazole combination therapy . She declined admission into the ward and was lost to follow up for the next two months. She later represented to A& E where she was seen and referred to the Gastroenterology clinic. Following a provisional diagnosis of decompensated CLD to r/o differential diagnoses of abdominal tuberculosis and ovarian cancer; she was then placed on oral spironolactone 25mg daily, carvedilol 3.125mg b.d livolin forte 1 T b. d tablets for a week; was asked to do the initial requested investigations and counseled on the need to be admitted into the hospital. She still declined in- patient hospital admission due to concern for her young children that she has to leave at home without any other care giver. Few days later on worsening of symptoms she went to the Obstetric and Gynaecology (O&G) clinic where gynaecological malignancy was ruled out clinically and abdominal tuberculosis was strongly considered. She subsequently accepted to be admitted into the internal medicine inpatient wards.

Physical Examination revealed that patient was underweight with Body Mass Index (BMI) of 16.3kg/m^2 and febrile with temperature of 38.0°C with no peripheral lymphadenopathy, no and no pedal edema and no stigmata of chronic liver disease. She had tachycardia. Abdomen moved with respiration and was uniformly distended, umbilicus was everted, and there were no visible umbilical veins. Abdominal girth was 96cm at the level of the umbilicus; it was doughy with periumbilical tenderness and there was no palpable organomegaly. Ascites was demonstrable by shifting dullness and bowel sounds reduced. Other systemic examination was unremarkable.

Table 1 : Investigations and results

No.	Investigation	Result(Normal value)	Remarks
1	Ascitic fluid macroscopy, microscopy culture and sensitivity(m/c/s)	Serosanguinous, No growth of mycobacterium tuberculosis	Negative but likely an exudate

2	Ascitic fluid Gene-Xpert test	Mycobacterium tuberculosis(MTB) not detected	Negative
3	Ascitic fluid cytology	There was absence of atypical inflammatory cells	Not suggestive of malignancy
4	Ascitic fluid biochemistry	Protein 57g/l (62-80g/l) , Glucose was 2.3mmol/l (4.2/6.4mmol/l)	Decreased Decreased
5	Ascitic fluid adenosine deaminase	87 IU//L (0- 30IU/L)	Elevated
6	Sputum Acid fast bacilli smear and culture	No growth of mycobacterium tuberculosis	Negative
7	Sputum Gene- Xpert test	(MTB) not detected	Negative
8	Full blood count	PCV was 36% (40-54%) WBC count was $5.2x10^9$/L(4.8-10.8X 10^9/L) Neutrophil- 53% (40-75) Lymphocytes-28% (20-45%) Mono/baso/eosinophils- 19%(1-6) Platelet was 334 $x10^9$/L (140-400X 10^9/L)	Decreased, Normal, Likely eosinophilia
9	Erythrocyte Sedimentation Rate(ESR)	37mm/hr(5-7mm/hr)- *Pre Anti-Koch's therapy* 67mm/hr	Elevated
10	Serology	Retroviral screen Hepatitis B surface antigen Hepatitis C antibody	Negative to HIV I/II Sero-negative Sero-negative
11	Chest X- ray	No patchy opacities, areas of consolidation or pleural effusion	Normal

		seen	
12	Abdominal Scan	Debritic ascites, No matted bowel loops or loculated collection	Most likely wet type peritonitis
13	Mantoux	17mm(< 5mm)	Most likely tuberculosis

While on admission the following investigations were done and the results are shown as above in Table 1. Other investigations were urinalysis which was normal while urine m/c/s showed no growth of any organism. No ova of any parasitic infection was seen on stool m/c/s. Liver function tests, fasting blood sugar and glycated haemoglobin were within normal limits. Therapeutic anti-Koch's therapy was strongly considered. The Directly Observed Treatment, Short course (DOTS) team was informed of the case. The results of the Mantoux and ascitic fluid adenosine deaminase showed elevated values. This made the decision to commence and continue therapeutic anti-Koch's stronger. The initiation phase was instituted with daily oral tablets rifampicin, isoniazid, pyrazinamide and ethambutol for the first two months and to continue with rifampicin and isoniazid for the next seven months with pyridoxine. Other adjunct therapy such as multivitamins, anti-helminthic and analgesic was also prescribed as requested. Patient subsequently improved as monitoring chart showed decrease in abdominal girth, weight, pulse rate, temperature and ESR after a week after commencement of Anti-Koch's therapy. There was initial reduction in weight due to loss of ascitic fluid and regain of actual muscle mass, resolved tachycardia to normal. The ESR became high initially after treatment but it gradually decreased to the normal limit. She had lost weight as a result of the resolution of the ascites however she did not gain much after other parameters such as fever has resolved. And ESR normalized. She confessed to have always been a very thin person and does not like to eat much since childhood during her follow up visits. Her BMI was 17.1 kg/m^2. Patient was counseled on the importance of weight gain and the risk of a low BMI in relation to the risk of tuberculosis. She was referred to the dietician to implement weight gaining diet as well to the primary DOTS care centre after the initial follow up visits at the Pulmonary clinic.

A -Pre Anti Koch's) B -1 week after C- 2 weeks after discharge

Figures A-C:Progressive response characterized by resolution of ascites

Discussion

This is a case of a Nigerian female patient who presented with abdominal tuberculosis. Abdominal TB was suspected as a differential from the onset as Nigeria is known to be one of the countries with a high prevalence of TB.[6] It is known globally that tuberculosis is twice as much as in men than women which the reasons have not been fully understood but it has been attributed to the socioeconomic and cultural factors related to exposure as well as biological mechanisms.[10] Extra-pulmonary tuberculosis particularly abdominal tuberculosis has been noted to be more common in females. The reasons are not clearly understood and this may as well be related to biological and socio-cultural differences.[11, 12] The past history of poorly treated TB is the strongest risk factor for development of abdominal TB in this patient. It would have been thought that she would have developed resistance after being on the drug for only a 2 month period however this was not so from the result of the gene -xpert test which also detects rifampicin resistance. This actually creates the dilemma of if this was a case of a reinfection, reactivation or partial resistance. It is known that those with pulmonary TB can also have extra-pulmonary manifestation. At the time of having the pulmonary symptoms she had no extrapulmonary manifestations. While when she had abdominal symptoms she had no respiratory symptoms. The time lag between these two systemic manifestations was 9 months. Accurate diagnosis was needed in this case because treatment would require prolonged antibiotics and the possibility of occurrence of side effects that may impair compliance and lead to antibiotic resistance. The patient may also loose confidence in the health care system if there was no certain proof that the cause of her previous illness was the same as the cause of this- atypical pneumonia caused by *mycobacterium tuberculosis*

Other risk factors for tuberculosis in this patient is being underweight. She had lost weight as a result of the resolution of the ascites however she did not gain much after other parameters such as fever has resolved and ESR normalized. She admitted to have always been a very thin person and does not like to eat much since childhood during her follow up visits. Low BMI has been associated with increased risk of having TB and a high TB mortality.[13, 14]

In making diagnosis of tuberculosis, the gene - xpert was negative using sputum and ascitic fluid samples.Gene - xpert test is a Cartridge Based Nucleic Acid Amplification Test(CBNAT) which is sensitive for diagnosing tuberculosis however it has been noted that the sensitivity is

influenced by the method of sample collection particular that of sputum collection.[15] The use of acid fast bacilli culture using body fluids have been noted to have poor yield hence the use of body fluid adenosine deaminase which has been shown to be more sensitive and specific.[16] It is also important to note that ADA can also be elevated in liver disease.[17] The method of sample collection for proper histologic and pathologic diagnosis of wet peritonitis type of abdominal TB the category which this patient falls into, has also been noted to be improved by carrying out an ultrasound-guided aspiration followed by laparoscopy if needed. [18]

In the management of TB contact tracing and adherence to therapy are two key steps in prevention of spread. This patient had poor adherence to therapy when she had pulmonary tuberculosis and this may have resulted in a dissemination of the bacilli and placed her at the risk of development of TB drug resistance.[19, 20] This patient was fortunate to have responded to the primary TB treatment regimen and needs continuous counselling, reassessment and monitoring..

Conclusion

Abdominal tuberculosis have different presentations and can mimic other diseases of the gastrointestinal system. A high index of suspicion is required to make accurate diagnosis of abdominal tuberculosis.

References

1) Sousa MD, Batista J, Pacheco P, Nunes V. 'Abdominal tuberculosis: an old disease surprising young doctors.' *BMJ Case Rep.* 2016 Nov 2; 2016
2) Sheer TA, Coyle WJ. 'Gastrointestinal tuberculosis'. *Curr Gastroenterol Rep.* 2003 Aug;5(4):273-8
3) Skopin MS, Batyrov FA, Kornilova ZKh. [The prevalence of abdominal tuberculosis and the specific features of its detection]. *Probl Tuberk Bolezn Legk.* 2007;(1):22-6. Russian. PMID: 17338349.
4) Cho, JK., et al. 'Clinical features and outcomes of abdominal tuberculosis in southeastern Korea: 12 years of experience.' *BMC Infect Dis, 2018,* 18, 699 .
5) Urabinahatti KA, et al. 'Abdominal tuberculosis: an epidemiological profile and management of 40 cases in a tertiary set up.' *Int Surg J* 2016;3:1502-8.
6) Affusim CC, Kesieme E, Abah VO. The Pattern of Presentation and Prevalence of Tuberculosis in HIV-Seropositive Patients Seen at Benin City, Nigeria. ISRN *Pulmonology* 2012;Article ID 326572:6pagesdoi:10.5402/2012/326572
7) Anonymous, 'Management Of Non-Respiratory Tuberculosis', *The Lancet,* 1968; 1:1423-4

8) Edafe, AE, Alasia, DD, Akpa, MR 'Abdominal Tuberculosis Mimicking Intra-abdominal Malignancy:A case report.' *The Nigerian Health Journal*,2015, Oct-Dec 15(4)
9) Chakinala, RC, Khatri, AM, 'Gastrointestinal Tuberculosis', Available at https://www.ncbi.nlm.nih.gov/books/NBK556115/, Accessed (10-09-2022)
10) Ferreira, C Pinto, T Neves, I. Duarte, R 'Gender differences in active tuberculosis' *European Respiratory Journal* 2014 44: P2688
11) Ben Jmaa M, Ben Ayed H, Koubaa M, Hammami F, Damak J, Ben Jemaa M. 'Is there gender inequality in the epidemiological profile of tuberculosis?' *Tunis Med.* 2020 Mar;98(3):232-240
12) Jemaa, MB et al '*762.* 'Sex Differences in the Epidemiology of Tuberculosis in Tunisia,' *Open Forum Infectious Diseases,* Nov, 2018, 5(suppl_1) :S274,
13) Yen YF, et al 'Association of Body Mass Index With Tuberculosis Mortality: A Population-Based Follow-Up Study.' *Medicine (Baltimore)*. 2016 Jan;95(1)
14) Kitonsa, PJ et al. 'Evaluation of underweight status may improve identification of the highest-risk patients during outpatient evaluation for pulmonary tuberculosis.' *PLoS ONE* , 2020, 15(12): e0243542.
15) Meyer A. et al. 'Sputum quality and diagnostic performance of GeneXpert MTB/RIF among smear-negative adults with presumed tuberculosis in Uganda.' *PLoS One*. 2017 Jul 7;12(7):e0180572
16) Baraua, and Hossana, M. 'Adenosine Deaminase in Diagnosis of Tuberculosis: A Review.' *Anwer Khan Modern Medical College Journal*, *5*(2), 43–48.
17) Atta, S., Kassem, A., Elhadidi, A., Esawy, H.E, The diagnostic value of adenosine deaminase activity in pulmonary tuberculosis: Comparison between sputum and serum, *Egyptian Journal of Chest Diseases and Tuberculosis*, 2015: 64(1) : 103-107
18) Abu-Zidan, F.M., Sheek-Hussein, M. 'Diagnosis of abdominal tuberculosis: lessons learned over 30 years: pectoral assay.' *World J Emerg Surg*, 2019 14, 33 .
19) CDC, *Treatment of Tuberculosis Disease,* Available at https://www.cdc.gov/tb/education/corecurr/pdf/chapter6.pdf, Accessed (25-09-2022)
20) Mohammed, TJM, Wafaa, H., Wafaa, B., 'Adenosine Deaminase (ADA) in Peritoneal Tuberculosis,' Available at https://www.worldgastroenterology.org/publications/e-wgn/e-wgn-expert-point-of-view-articles-collection/adenosine-deaminase-ada-in-peritoneal-tuberculosis, Accessed (25-09-2022)

: