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Original Research Paper

General Medicine

# A RARE CASE OF ANTI-TUBERCULAR DRUG INDUCED CARDIOMYOPATHY

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**KEYWORDS :**

**BACKGROUND:**

Anti-tubercular drugs are associated with some common and some rare adverse effects. We describe a case of a rare side effect- cardiomyopathy. In literature, none of the antituberculosis drugs are commonly mentioned among the drugs known to cause myocardial injury and cardiomyopathy. There have been only a handful of cases of anti-tuberculosis drug induced cardiomyopathy reported before. It is very important to recognize and treat this condition in a timely fashion which can otherwise be fatal as is shown in this case.

**Case Presentation:**

18 year old male patient with a known history of abdominal tuberculosis diagnosed on CT abdomen, 3 months ago presented with decreased appetite, weakness, sleep disturbances and pedal edema for 15 days. The patient at that time had completed 3 months of the standard four drug anti-tuberculosis treatment. On physical exam, there was decreased bilateral air entry, crepitation, elevated JVP, abdominal distention, pedal edema and icterus. Lab values revealed increased liver enzymes which hinted towards isoniazid induced hepatotoxicity. Isoniazid and rifampicin was discontinued and patient was started on ethambutol, amikacin and linezolid with daily monitoring of liver enzymes. There was no improvement. Subsequently, an echocardiogram was done which showed EF of 15%. The patient was then started on Lasix and spironolactone and all anti-tuberculosis drugs were discontinued. Ultimately, inotropes were started. In the end, unfortunately the patient passed away.

## INTRODUCTION

In literature, the most common documented side effect of the standard 4 drug anti tuberculous therapy is hepatotoxicity. Although about 85% of TB cases are successfully treated, treatment-related adverse events including hepatotoxicity, skin reactions, gastrointestinal and neurological disorders account for significant morbidity leading to reduced effectiveness of therapy.Hepatotoxicity is the commonest of all adverse effect leading to drug discontinuation in 11% of patients treated with combination of isoniazid,rifampicin and pyrazinamide.[3[ Here we describe a rare presentation of a rare antituberculous therapy adverse effect.

Here the patient with abdominal tuberculosis was presumed to have developed hepatotoxicity from the standard 4 drug therapy. When the liver enzymes remained elevated and the symptoms worsened despite stopping the antituberculous therapy, further investigations lead to the diagnosis of congestive cardiac failure and hepatic congestion. Amongst the lesser- known side effects of anti-tuberculous therapy, is its effect on cardiac function, which if not discovered can prove fatal as is shown from this case report

**Pathophysiology**

According to WHO, worldwide tb is the 13th leading cause of death, and the second leading cause of infectious disease death after Covid 19. In the light of this it is important to recognize some of the rare manifestations of tb and its treatment.The cardiovascular structures usually involved in tuberculosis are the pericardium, themyocardium, and the aorta.

(https://www.ahajournals.org/doi/10.1161/JAHA.120.019435)
https://njmonline.nl/getpdf.php?t=i&id=30#page=22

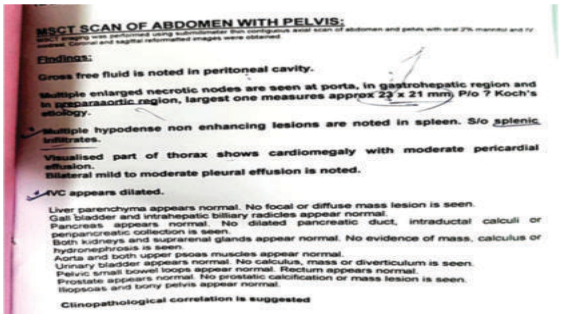
Etiology of dilated cardiomyopathy: [1]: The most common etiology of dilated cardiomyopathy (DCM) is idiopathic and without an identifiable cause. DCM can have a familial or genetic predisposition although these cases are usually classified under idiopathic if no clear genetic link is identified. DCM has been associated with mutations in genes for Desmin (cytoskeletal),Lamin C (nuclear membrane), or Myosin (contractile proteins). The secondary causes includeinfectious myocarditis (e.g., viral, Chagas disease, Lyme disease), ischemic disease,hypertension, medication-induced (e.g., Anthracyclines), alcohol abuse, humanimmunodeficiency virus (HIV), peripartum cardiomyopathy, or infiltrative disease. Ischemic cardiomyopathy caused by coronary artery disease (CAD) is the most common cause of congestive heart failure. However, ischemic cardiomyopathy is classified as its own disease entity and is only described as a cause of DCM in occult disease in patients without known CAD

Evaluation for secondary causes of dilated cardiomyopathy (DCM) always should be pursued prior to making the diagnosis of idiopathic DCM. Workup is focused on identifying any possible reversible causes. Recommended laboratory testing includes thyroid function tests, HIVserology, electrolytes, and iron studies (to rule out hemochromatosis). Urine toxicology screen and alcohol level can be checked when substance abuse is suspected. In certain familial cases,genetic testing should be considered. Serum B-type natriuretic peptide (BNP) levels may be obtained in cases where the diagnosis is unclear. Low levels of BNP are useful in ruling out CHF. In addition, levels of BNP are useful for prognosis.One should also rule out hypothyroidism and anemia.

## CONCLUSION

The common side effects of the first line anti tubercular therapy are hepatotoxicity, peripheral neuropathy, thrombocytopenia, hyperuricemia, optic neuropathy, arthralgia among others. The fact that this patient developed cardiomyopathy after the initiation of treatment for tb points towards a drug adverse effect rather than tuberculosis induced cardiomyopathy. Due to financial constraints we

could not obtain a biopsy or a cardiac MRI. However the circumstantial evidence indicates that it is a drug related phenomenon. We also ruled out other possible causes of cardiomyopathy. More such cases should be reported to decide which of the first line drugs is responsible, develop a management plan and to determine whether routine screening with 2D echo is needed along with CMPs and other labs in patients on anti-tubercular therapy.



MSCT SCAN OF ABDOMEN WITH PELVIS:

Findings:

Gross free fluid is noted in peritoneal cavity.

Multiple enlarged necrotic nodes are seen at porta, in gastrohepatic region and in preparaaortic region, largest one measures approx 22 x 21 mm. P/o ? Koch's etiology.

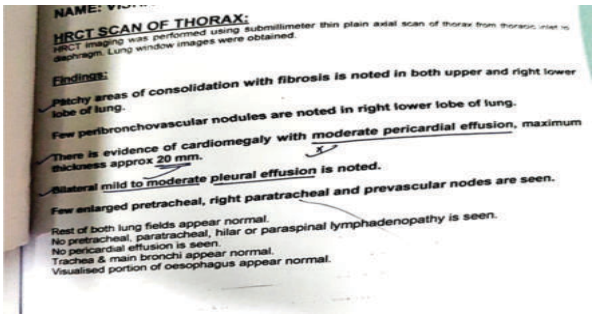
Multiple hypodense non enhancing lesions are noted in spleen. S/o splenic infiltrates.

Visualised part of thorax shows cardiomegaly with moderate pericardial effusion.
Bilateral mild to moderate pleural effusion is noted.

IVC appears dilated.

Liver parenchyma appears normal. No focal or diffuse mass lesion is seen.
Gall bladder and intrahepatic biliary radicles appear normal.
Pancreas appears normal. No dilated pancreatic duct, intraductal calculi or peripancreatic collection is seen.
Both kidneys and suprarenal glands appear normal. No evidence of mass, calculus or hydronephrosis is seen.
Aorta and both upper psoas muscles appear normal.
Urinary bladder appears normal. No calculus, mass or diverticulum is seen.
Pelvic small bowel loops appear normal. Rectum appears normal.
Prostate appears normal. No prostatic calcification or mass lesion is seen.
Iliopsoas and bony pelvis appear normal.

Clinopathological correlation is suggested



NAME:

HRCT SCAN OF THORAX:

HRCT imaging was performed using submillimeter thin plain axial scan of thorax from thoracic inlet to diaphragm. Lung window images were obtained.

Findings:

Patchy areas of consolidation with fibrosis is noted in both upper and right lower lobe of lung.

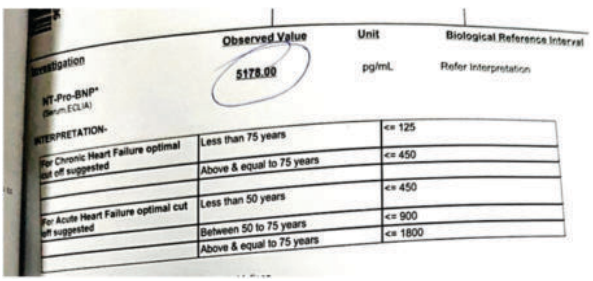
Few peribronchovascular nodules are noted in right lower lobe of lung.

There is evidence of cardiomegaly with moderate pericardial effusion, maximum thickness approx 20 mm.

Bilateral mild to moderate pleural effusion is noted.

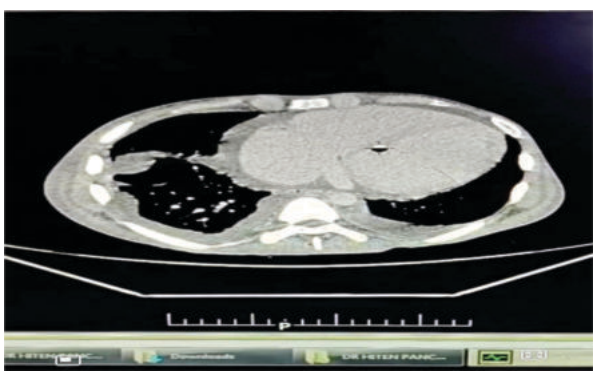
Few enlarged pretracheal, right paratracheal and prevascular nodes are seen.

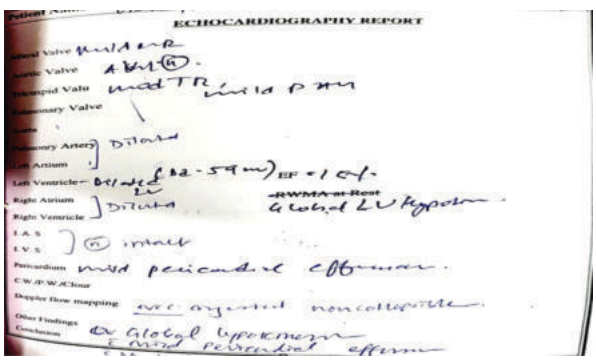
Rest of both lung fields appear normal.
No pretracheal, paratracheal, hilar or paraspinal lymphadenopathy is seen.
No pericardial effusion is seen.
Trachea & main bronchi appear normal.
Visualised portion of oesophagus appear normal.



| Investigation | Observed Value | Unit | Biological Reference Interval |
|---|---|---|---|
| NT-Pro-BNP* (Serum ECLIA) | 5178.00 | pg/mL | Refer Interpretation |

| INTERPRETATION- | | |
|---|---|---|
| For Chronic Heart Failure optimal cut off suggested | Less than 75 years | <= 125 |
| | Above & equal to 75 years | <= 450 |
| For Acute Heart Failure optimal cut off suggested | Less than 50 years | <= 450 |
| | Between 50 to 75 years | <= 900 |
| | Above & equal to 75 years | <= 1800 |





ECHOCARDIOGRAPHY REPORT

Aortic Valve AVR ®.
Tricuspid Valu mod TR
Pulmonary Valve
Pulmonary Artery Dilated
Left Atrium
Left Ventricle- Dilated (52-59 mm) EF = 1 0%.
Right Atrium Dilated
Right Ventricle RWMA at Rest Global LV Hypokin
I.A.S
I.V.S
Pericardium mild pericardial effusion.
C.W.P.W.Colour
Doppler flow mapping
Other Findings
Conclusion Dx Global hypokinesia mild pericardial effusion


**Citations:**

[1] https://www.ncbi.nlm.nih.gov/books/NBK441911/
[2] https://www. proquest.com/ openview/ e0b48e5d5d15e976bc0749e03cc1244c/1?pq-origsite=g scholar &cbl=43703
[3] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3940184/

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