

AKT INDUCED HEPATITIS: A CASE REPORT

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ABSTRACT

Background: Tuberculosis (TB) is a highly contagious bacterial infection that can affect almost any part of the body, but the lungs are the most common site of infection. INH, RMP, PZA, and PZA are hepatotoxic among the first-line combination therapy medications (INH, RMP, PZA, EMB), as they are mostly processed by the liver.

CASE: This is a case report of 23 year female who has history of potts spine (2 months ago), diagnosed with hepatitis due to Antituberculosis drug. She was admitted to hospital for nausea, Loss of appetite, Fever, Lower leg pain, Backache, Dry cough. Lab investigation shows elevated liver enzymes. In this case patient was receiving antituberculosis drug since 1.5months and later developed hepatitis which is severe adverse drug reaction. **Conclusion:** Hepatotoxicity is

serious side effect of antituberculosis medication we had a positive outcome after standard therapy and care of the patient.

INTRODUCTION

Tuberculosis (TB) is a potentially fatal infectious illness affecting mostly the lungs. Tuberculosis bacteria are communicated from person to person via minute droplets discharged into the air by coughs and sneezes. In 1985, tuberculosis infections began to rise in affluent countries, mainly due to the advent of HIV, the virus that causes AIDS. HIV affects a person's immune system, making it unable to combat tuberculosis germs. In the United States, tuberculosis began to decline again in 1993 as a result of improved control strategies. However, it is still a source of concern.^[1]

Spinal tuberculosis, also known as Pott's disease or tuberculous spondylitis, is a rare infectious condition that causes the vertebrae to collapse, resulting in a kyphosis deformity

(hunchback). Pott's disease, also known as bone tuberculosis, begins in the vertebra and slowly spreads to surrounding areas. It is named after Dr. Percivall Pott, who first described the condition in the 1700s.^[2]

DOTS is the main strategy for tuberculosis management, according to the Revised National Tuberculosis Control Program (RNTCP). Rifampin [R], Isoniazid [H], Pyrazinamide [Z], and Ethambutol [E] are the first-line agents for tuberculosis treatment. They will be given for two months, followed by four months of Isoniazid and Rifampin. Anti-tubercular medications have been linked to an increased risk of liver and kidney damage. Recent anti-TB treatment guidelines have also proposed approaches to manage common side effects such as GI discomfort, rash, drug fever, hepatotoxicity, and optic neuritis. Acute renal damage, on the other hand, has no treatment guidelines. Hepatotoxicity caused by ATT affects 2-28 percent of people. An elevated liver enzyme of 5 times the upper limit of normal can be used to confirm ATT-induced hepatitis in the absence of jaundice or other symptoms; in the presence of jaundice or other symptoms, an elevated liver enzyme of 5 times the upper limit of normal can be used to confirm ATT-induced hepatitis.^[3]

CASE REPORT

A 23-year female patient came with complaints of vomiting since 3-4 days, Nausea since 5-6 days, Loss of appetite since 5-6 days, Fever since 2-3 months which is high grade associated with chills, B/L Lower leg pain since 10 months, Backache since 1 year, Dry cough since 1 month, Burning sensation in stomach and giddiness since 15 days. Patient was addicted to tobacco. Patient was diagnosed with Pott's spine since 2 months. Patient started on gov AKT around 1.5 months back.

On admission, physician was confirmed that patient was suffering from Pott's spine which is also known as spinal TB, additionally patient vitals were normal according to lab values (SGOT= 191, SGPT=201), normal range up to 40 IU/L and USG report of abdomen/pelvis patient was also suffering from borderline hepatitis which is not dangerous but this condition is required to be controlled otherwise it leads to liver cirrhosis and hepatocellular carcinoma which is very dangerous for that physician has changed medication from AKT4 (Isoniazide, Rifampicin, Ethambutol, Pyrazinamide) to streptomycin (0.5 gm iv), Levofloxacin (500 mg, 1-0-0), Ethambutol (800 mg, 1-0-0) as a result on 5th day patient SGOT (59 IU/L) and SGPT (112 IU/L) were returned to normal and rest of treatment was same without any modification.

Finally patient was discharged from dhiraj hospital after stay of 10days. Patient was received following type of treatment.

	Brand name	Dose	Frequency
1	Inj streptomycin	0.5gm	1-0-0
2	Inj levofloxacin	500mg	1-0-0
3	Inj ethambutol	800mg	1-0-0
4.	Tab. Pandeep DSR	40/10mg	1-0-0
5.	Tab tonofolic DS	15/300/1.5mg	1-0-1
6.	Inj normal saline	1 ampule	
7.	Inj emset	4mg	1-0-1
8.	Tab.tramdeep P	37.5/325mg	1-0-1
9.	Tab. Pegs SR-M	1500ug/75mg	1-0-1
10	Inj dexona	4mg	1-0-1

DISCUSSION

Anti-TB medications are one of the most common causes of idiosyncratic hepatotoxicity over the world. The incidence of anti-TB drug-induced hepatotoxicity varies greatly depending on the cohort's characteristics, treatment regimens utilised, hepatotoxicity thresholds used, and monitoring and reporting techniques. Hepatotoxicity caused by anti-TB medications has been observed in 5%–28% of persons who have been treated with anti-TB drugs. However, determining how many of these cases fit into a more current international consensus case definition of drug-induced liver injury is problematic (DILI). The majority of the reports used an elevated alanine (ALT) or aspartate transaminase (AST) of 3 times upper limit of normal range (ULN) with symptoms (abdominal pain, nausea, vomiting, unexplained fatigue, or jaundice) attributable to liver injury, or 5 times ULN of ALT with symptoms (abdominal pain, nausea, vomiting, unexplained fatigue, or jaundice) attributable to liver injury.

When compared to acute viral hepatitis, anti-TB drug-induced fulminant liver failure appears to have a worse result, with a case fatality rate ranging between 0.042 and 0.07 per 1000 people at any given point during treatment. When accessible, liver biopsy results demonstrate lobular hepatitis, sub-massive to major necrosis, and hydropic hepatocyte degeneration in severe cases. On histology, localised hepatocellular necrosis and apoptosis in zone 3 as well as cholestasis have been observed in cases of rifampicin hepatotoxicity.

CONCLUSION

A case of anti-tuberculosis drugs-induced hepatitis in an 23-year-old female patient was reported. Hepatotoxicity is a serious side effect of anti-tubercular drugs. By providing

standard treatment and care we have achieved outcome. It is important for pharmacist and physician to follow up and counsel patient about sign and symptoms of hepatitis.

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