We read the article titled “Tuberculosis in cirrhosis: A diagnostic and management conundrum” by Mishra S et al. published in your esteemed journal online with great enthusiasm. We congratulate the authors for focusing on a rather common clinical problem.1 As authors have rightly pointed out that extrapulmonary tuberculosis (EPTB) is highly prevalent in cirrhotic patients due to the reasons mentioned.

We would like to share our own experience regarding diagnosis, treatment, and outcome on peritoneal TB in cirrhosis. We had a total of 30 patients with peritoneal TB with cirrhosis; alcohol was the most common etiology (17, 56.66%), followed by hepatitis C virus infection (6, 20%). Hepatitis B virus infection, Budd Chiari syndrome, and nonalcoholic steatohepatitis (NASH), were the underlying etiology in two patients each while one was cryptogenic. Of the total 30, 8 had mixed ascites, while 22 had low SAAG (serum-ascites albumin gradient) ascites. We recently published the article on the role of adenosine deaminase (ADA) for the diagnosis of peritoneal tuberculosis in cirrhotic patients.2 At a cut-off value of 39.9 IU/L, we had 93% sensitivity and 94% specificity. Mishra S, et al.1 reported sensitivity and specificity of 80%, 90%, respectively, at a cutoff of 26 IU/L. But the authors have analyzed pleural and peritoneal TB together, and the method used for estimation of ADA was Giusti method while we used Slaat’s method. Additionally, our study was a prospective one, and we have used histopathology as the diagnostic gold standard. Biopsy sample obtained by laparoscopy (from parietal peritoneum and/or omentum) in 23 cirrhotic peritoneal TB patients while in rest seven cirrhotic patients underwent ultrasound-guided omental biopsy. In our population, 4 patients (8.5%) had an adverse event related to the biopsy, 3 (10%) in the cirrhosis group and 1 (2%) in the noncirrhotic group. Two patients with cirrhosis developed decompensation and worsening of underlying liver disease, with one patient succumbing to progressive organ failures. These findings underline the importance of ADA as a screening and supportive diagnostic tool in high TB prevalent countries like India. It is possible that varying severity of underlying liver disease (reflected in MELD scores or Child-Pugh score) may affect actual ADA values. Given that very few studies have been published on the role of ADA in cirrhotic TBP patients and the small sample size in the individual studies, it is difficult to draw a final conclusion. Given the difficulty in making tissue diagnosis (in traditional Paustian3 and Logan4 criteria) in cirrhotic TBP patients, ADA still has a role in the diagnosis of TB in cirrhosis.

Regarding ATT to be used and outcome in cirrhosis, available data is limited.5 The current recommendations of two hepatotoxic drugs for Child A, one hepatotoxic drug for Child B, and no hepatotoxic drug in Child C is more of an expert recommendation due to little evidence.7 In current study authors have given details of treatment used from which it is clear that all three hepatotoxic drugs are still used by clinicians simultaneously in cirrhotic, which authors have inferred eventually led to increased drug-induced liver injury (DILI) (30%).

In our series, we did not use pyrazinamide in any cirrhotic patient. Child C patients were not given any hepatotoxic drug at start; child B patients were given one hepatotoxic drug, preferably rifampicin (R), while child A patients were given both isoniazid (H) and rifampicin. Ethambutol (E) is common in all, while streptomycin (S) and levofloxacin (L) were used on a case-to-case basis. In total, 13 patients received SHRE regimen, 8 patients received SERL regimen, and SER and SEL were received by three patients each. Four patients died in the cirrhotic group on follow up within three months versus none in the noncirrhotic TBP group (13% vs. 0%, p-0.009). All patients were followed for six months or till completion of ATT, whichever was later. Three patients among cirrhotic TBP were lost to follow up after three months. DILI is defined as the rise in bilirubin by 2.5 mg/Dl and/or increase in transaminases by >2 times from baseline value.5

Overall adverse event rate was higher in cirrhotic TBP than in noncirrhotic TBP (7, 23% vs.3, 6%, p-0.02). In a study by Mishra S, et al adverse event rate was higher by 30% but was attributed to the simultaneous initiation of all three hepatotoxic drugs. We strictly adhered to recommended guidelines, but despite this, 16% developed DILI, which is a significant number.

Tuberculosis has defied human medical and technological evolution. Multicenter studies with adequate sample size are the need of the hour for a uniform diagnostic approach, treatment, and predictors of DILI in cirrhotic patients with EPTB.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

Amol Sonyabapu Dahale: Conceptualization, Writing – original draft, Data Collection. Amarendra Singh Puri: Conceptualization, Writing – original draft. Anushka
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**Verma:** Data collection, Formal analysis, Validation. **Sanjeev Sachdeva:** Visualization, Investigation. **Ashok Dalal:** Supervision, Revision. **Debabrata Banerjee:** Writing – review & editing.

**CONFLICTS OF INTEREST**

The authors have none to declare.

**REFERENCES**