INTRODUCTION
Alcohol is the world’s third-largest risk factor for disease burden. Consumption of alcohol results in 2.5 million deaths each year. Alcoholic hepatitis is an acute inflammation of the liver, accompanied by the destruction of individual liver cells and scarring. Symptoms may include fever, jaundice, an increased white blood cell count, an enlarged, tender liver, and spider-like veins in the skin. It may develop due to large amounts of alcohol for a long period and the outcome may range from abnormal liver functions with no symptoms to hepatic encephalopathy.

The World Health Organization (WHO) estimates that 140 million people worldwide suffer from alcohol dependency, causing damage to lives and economies. In India, 15 people die every day – or one every 96 minutes – from the effects of drinking alcohol, reveals an India Spend analysis of 2013 National Crime Records Bureau (NCRB) data. The per capita consumption of alcohol in India increased 38 percent. According to WHO data published in 2014 the total pure alcohol consumption among persons (age 15+) in liters per capita per year is 4.4 out of which 2.2 liters per capita per year is recorded consumption of alcohol where 2.2 liters per capita per year is unrecorded consumption. Mortality from alcoholic cirrhosis is declining in western nations but it is increasing in India.

EPIDEMIOLOGY
Incidence is unknown in India but prevalence varies among different states.

<table>
<thead>
<tr>
<th>Test</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST</td>
<td>Increased two to sevenfold, &lt;400 IU/L, &gt; ALT</td>
</tr>
<tr>
<td>ALT</td>
<td>Increased two to sevenfold, &lt;400 IU/L</td>
</tr>
<tr>
<td>AST/ALT</td>
<td>Usually &gt; 1</td>
</tr>
<tr>
<td>GGTP</td>
<td>Not specific to alcohol, easily inducible, elevated in all forms of fatty liver</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>May be markedly increased in alcoholic hepatitis despite modest elevation in alkaline phosphatase</td>
</tr>
<tr>
<td>ALP</td>
<td>Mildly elevated</td>
</tr>
</tbody>
</table>

ALT, Alanine aminotransferase; AST, Aspartate aminotransferase; GGTP, γ Glutamyl transpeptidase; ALP, Alkaline phosphatase.
assessment, various question formats, i.e. CAGE test, are helpful.

**PROGNOSTIC SCORES**
In clinical practice various scores are used to predict outcome of alcoholic hepatitis. The single most reliable indicator of severity is the presence of hepatic encephalopathy.

**Discriminant Function (DF) Score**
The DF of Maddrey and coworkers is based on PT and bilirubin levels and it is calculated as follows: DF = (4.6 × PT prolongation) + total serum bilirubin in mg/dL.

**MELD Score**
Several retrospective studies have shown that the MELD score is useful in predicting 30- and 90-day mortality in patients with alcoholic hepatitis. Moreover, the MELD score seems to contain some practical and statistical advantages over Maddrey’s DF in predicting mortality among these patients. In a cohort of 73 patients with alcoholic hepatitis at the Mayo Clinic, the MELD score was the only independent predictor of mortality [5]. Likewise, in another much larger retrospective study of 202 patients with alcoholic hepatitis, the MELD score was found superior to not only Maddrey’s DF but also to the classical Child-Turcotte-Pugh (CTP) score.6

**Glasgow Alcoholic Hepatitis Score (GAHS)**
The GAHS is one of the most recently described predictors of outcome in patients with alcoholic hepatitis. This scoring system uses 5 different variables, including age, bilirubin level, blood urea nitrogen (BUN) level, PT, and WBC count. The overall accuracy of GAHS, which was validated in 195 patients with alcoholic hepatitis, was 81%, when predicting 28-day outcome.7 In contrast, the modified DF had an overall accuracy of only 50%.7

**Asymmetric Dimethylarginine (ADMA) Score**
The ADMA score is the most recently proposed predictor of adverse clinical outcome in patients with severe alcoholic hepatitis. In a small prospective study of 27 patients with alcoholic hepatitis, the ADMA score was a better predictor of outcome than the CTP score, the DF, or the MELD scores.8

Other factors that correlate with poor prognosis include older age, impaired renal function, encephalopathy, and a rise in the WBC count in the first 2 weeks of hospitalization. Significantly raised serum γ-glutamyltransferase (γ - GT) and Mean Corpuscular Volume (MCV) are most important and valuable for detection of alcohol excess. However, moderate rise of γ-GT may be found in nonalcoholic fatty liver drugs like phenetoin causing enzyme induction. Liver function tests – Elevated Serum transaminase level ALT and AST are not specific. These are mildly raised in fatty liver. Characteristically, the AST: ALT ratio is about 2:1, and the absolute value of the ALT does not exceed 300 U/L unless a superimposed hepatic insult exists, such as paracetamol toxicity. If raised 5 times of normalreference range, other diagnoses such as viral or autoimmune hepatitis should be considered.10

**HISTOLOGICAL FINDINGS**
Liver biopsy is not routinely necessary to diagnose liver injury. For fatty liver, biopsy is rarely required and may be useful in excluding steatohepatitis or fibrosis. Inflammation and necrosis occurs, most prominently in the centrilobular area of hepatic acinus. Ballooning of
hepatocytes is classical. They compress sinusoids and lead to portal hypertension which is reversible.\textsuperscript{9}

**CONCLUSION**

Upto 40\% patients with severe alcoholic hepatitis die within 6 months of onset of clinical syndromes. Alcoholic liver disease and alcoholic hepatitis is increasing in India. Early diagnosis and treatment can prevent development of cirrhosis and decompensation. Abstinence is the key factor in the management of alcoholic hepatitis.

**ACKNOWLEDGEMENT**

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**REFERENCES**