Background and Aim

Most patients with hepatitis A virus (HAV) infection have a favorable clinical course and spontaneous recovery, and only a few suffer from serious complications of acute hepatitis A such as acute fulminating hepatitis, acute renal failure, or cholestatic hepatitis. In some previous reports, the clinical course and outcome of hepatitis A in patients with underlying chronic hepatitis B virus (HBV) infection was associated with higher peak laboratory abnormalities and more severe outcomes, including acute liver failure and mortality. However, there is controversy over the influence of HBV infection on the severity of acute hepatitis A.

There are very few comparative studies of clinical outcome in acute hepatitis A in the presence of HBV surface antigen (HBsAg). In this study, the researchers have retrospectively compared the clinical outcome of acute hepatitis A between HBsAg-positive and HBsAg-negative patients, and analyzed the incidence of acute liver failure as a measure of the severity of acute hepatitis A.

Materials and Methods

Between January 2000 and February 2010, 452 consecutive patients who were admitted to Samsung Medical Center, Seoul, Korea for acute hepatitis A were retrospectively reviewed. Three patients who were positive for hepatitis C virus (HCV) RNA with anti-HCV were excluded from this study. A total of 449 patients were included and classified into 2 groups: HBsAg-positive group (n = 30) and HBsAg-negative group (n = 419).

Acute hepatitis A was diagnosed when patients were hospitalized with typical symptoms of acute viral hepatitis and presence of serum IgM anti-HAV. The presence of IgM anti-HAV and HBsAg and the absence of IgM anti-HBV established the diagnosis of acute hepatitis A superimposed on chronic HBsAg carrier. The following blood tests were performed at the initial time of admission and regularly during admission—hemoglobin, platelet count, prothrombin time (PT), serum aspartate aminotransferase (AST), serum alanine aminotransferase (ALT), serum total bilirubin, alkaline phosphatase (ALP), serum albumin, γ-glutamyl transpeptidase (GGT), and serum creatinine. Patients who were HBsAg-positive were compared with patients without HBsAg with respect to complete blood count at the time of admission and the most severe laboratory abnormality of the biochemical profile, including liver function tests, at admission. The study also compared the incidences of prolonged jaundice, gastrointestinal bleeding, acute renal failure, acute liver failure, liver transplantation, death, and the mean durations of hospitalization between the 2 groups to determine the differences in clinical outcome.

Results

Clinical Characteristics

Among the 30 patients in the HBsAg-positive group, males 22 (73.3%) outnumbered the females 8 (26.7%). Mean age at the time of admission was 36.1 years and 31.8 years in the HBsAg-positive group and HBsAg-negative group, respectively, and the HBsAg-positive group was significantly older than the HBsAg-negative group (P = .004). In both HBsAg-positive and HBsAg-negative groups, the majority of patients were in the age group of 30 to 39 years. Two patients in the HBsAg-negative group had liver cirrhosis at the time of admission for acute hepatitis A. Of the 30 patients with acute hepatitis A and chronic hepatitis B infection, HBV DNA was detectable in 20 patients at the time of admission.

Laboratory findings

The study compared the values for hemoglobin level and platelet count, and the most severe value of PT, serum albumin, ALT, and AST, and serum total bilirubin during admission between the 2 groups of patients with acute hepatitis A. Mean platelet count at the time of admission was significantly lower in patients of the HBsAg-positive group compared with the HBsAg-negative group (137 ± 69 vs 177 ± 84 × 10^3/mm^3, P = .006). Mean peak values of PT (2.8 ± 2.3 vs 1.7 ± 1.9 INR, P = .016), serum total bilirubin (19.9 ± 17.6 vs 11.1 ± 9.3 mg/dL, P < .001), and serum creatinine (2.1 ± 2.1 vs 1.5 ± 2.2 mg/dL, P < .001) during admission were also significantly higher in patients of the HBsAg-positive group.

Clinical Outcomes

To evaluate the influence of chronic HBV infection on the severity of acute hepatitis A, the incidences of prolonged jaundice, gastrointestinal bleeding, ARF, ALF, liver transplantation, death, and the mean durations of hospitalization were compared in terms of clinical outcomes between the 2 groups (Table 1). Of the 30 patients who were carriers of HBsAg, 7 developed ALF. All 7 patients had detectable HBV DNA at the time of admission. HBV DNA level of 7 patients at the time of admission were varied from 65 to 45,919 IU/mL.

The incidence of ALF was significantly higher among patients with acute hepatitis A and chronic HBV infection than among patients with acute hepatitis A alone (29.3% vs 3.3%; odds ratio [OR], 8.80; P < .001). Among the total 449 patients with acute hepatitis A, 21 patients had ALF, 11 patients underwent liver transplantation, and 4 died secondary to ALF. ARF was noted in 46 of 449 patients with acute hepatitis A. Among the 46 patients with ARF, 23 were in the HBsAg-positive group and 23 were in the HBsAg-negative group.

The incidence of ARF in patients with acute hepatitis A and chronic hepatitis B infection was also significantly higher than in patients with acute hepatitis A alone (OR, 3.64; P = .007). In comparison of hospitalization time according to the presence of HBsAg, the average duration of hospitalization for the HBsAg-positive and -negative groups was 21.6 ± 4.1 days and 10.0 ± 4.0 days, respectively. Thus, the hospitalization time reflecting the morbidity caused by acute hepatitis A was significantly longer in the HBsAg-positive group than in the HBsAg-negative group (P < .001).

To evaluate the potential risk characteristics of acute hepatitis A-associated ALF, the researchers performed multivariate analysis. Logistic regression analysis showed that HBsAg positivity (OR, 7.43; 95% confidence interval [CI], 2.56–21.57) and age (OR, 1.07; 95% CI, 1.02–1.13) were independent predictors for the occurrence of ALF.

Discussion

ALF is a rare complication of HAV infection. Fulminant hepatitis in HAV infection occurs in approximately 0.1% to 0.5% of cases. HAV-related liver disease is known to be caused by immunologic mechanisms rather than direct toxicity of the virus. Interferon-γ produced by HAV-specific T cells may play an important role in the pathogenesis of infection. During infection with HAV and HBV, it is possible that INF-γ produced in response to HAV stimuli has an antiviral effect on HAV infection. It has also been reported that HAV superinfection in patients with chronic hepatitis B suppresses hepatitis B viral replication. However, as reduction in HBV viral load is associated with an increase in the immune response of the host, in some cases an excessive host response may induce severe damage of hepatocytes.

Table 1. Clinical Outcome of Patients with Acute Viral Hepatitis

<table>
<thead>
<tr>
<th>Clinical Outcome</th>
<th>HBsAg (+)</th>
<th>HBsAg (-)</th>
<th>OR (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal bleeding</td>
<td>2 (6.7)</td>
<td>3 (0.7)</td>
<td>9.91 (1.56–61.72)</td>
<td>.009</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>8 (26.7)</td>
<td>10 (2.3)</td>
<td>3.78 (1.10–12.97)</td>
<td>.037</td>
</tr>
<tr>
<td>Acute liver failure</td>
<td>7 (23.3)</td>
<td>14 (3.3)</td>
<td>8.80 (2.24–33.93)</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Liver transplantation</td>
<td>5 (16.7)</td>
<td>6 (1.4)</td>
<td>13.77 (3.04–68.22)</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Death</td>
<td>2 (6.7)</td>
<td>2 (0.5)</td>
<td>4.87 (1.02–20.71)</td>
<td>.046</td>
</tr>
<tr>
<td>Hospitalization (mean days ± SD)</td>
<td>21.6 ± 4</td>
<td>10.0 ± 4</td>
<td>.007</td>
<td></td>
</tr>
</tbody>
</table>

Kim KM, et al.

Meta-analysis of 50 Phase III Clinical Trials for Evaluation of Efficacy and Safety of Liv.52 in Infective Hepatitis

Kolhapure SA, et al.


Introduction

Hepatitis A (HA) infection has a worldwide distribution and occurs in both epidemic and sporadic patterns. Hepatitis A virus (HAV) belongs to enterovirus group of Picornaviridae family. Hepatitis A is a self-limiting disease and no specific treatment is available. Early renormalizations of hepatic functions with symptomatic and clinical recovery are the object. This was the clinical management of HA. This study was planned for meta-analysis of the efficacy and safety of Liv.52 tablet and syrup, in the management of hepatitis A, as reported in 50 published study reports.

Materials and Methods

All published study reports evaluating efficacy and safety of Liv.52 were included for the meta-analysis, regardless of the study design. Each study was abstracted with emphasis on the number and ages of enrolled patients. Changes in biochemical parameters of serum bilirubin (SB), serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), serum alkaline phosphatase (SAP), serum albumin (SA), serum globulin (SG), and prothrombin time (PT) were noted at baseline and at the end of the study.

The predefined primary endpoints of the study were to determine the level of statistical significance for the following parameters—symptomatic improvement, renormalization of biochemical parameters, and total duration of clinical recovery. The predefined secondary endpoints were incidence of adverse events during the study period and compliance to the drug treatment. Statistical analysis was done according to intention-to-treat principles.

Results

A total of 50 clinical studies that evaluated the efficacy and safety of Liv.52 in HA, conducted over a span of 30 years (from 1967 to 1997) were considered for this meta-analysis. The mean duration of these studies was 6.62 months and total study duration of all trials was 331 months.

A total of 4490 patients were enrolled in these studies and 3007 patients received Liv.52 for a mean period of 6 months; 785 patients (from control group) received placebo; and other patients received corticosteroids, multivitamins, or other treatment. A total of 235 children were part of a study population classified as per their age: 97 children <5 years, 117 children between 6 and 10 years, and 19 children between 11 and 15 years.

Cumulative data analysis showed a significant reduction in the mean serum bilirubin level, at the end of all studies in Liv.52 group. Similarly, elevated SGOT, SGPT, alkaline phosphatase, and prothrombin time levels were reduced significantly, when compared to the pretreatment values, in all the studies. Decreased serum albumin and serum globulin levels were renormalized compared to pretreatment values in patients who received Liv.52.

There was highly significant reduction in the mean period required for total (symptomatic clinical, and biochemical) recovery, as compared to placebo. No significant adverse events were observed or reported in any trial and the overall drug compliance was excellent.

Discussion and Conclusion

Meta-analysis, according to Huque, is defined as a statistical analysis that combines or integrates the results of several independent clinical trials considered by the analyst to be “combiable.” A single study often cannot detect or exclude with certainty clinically relevant differences in the effects of two treatments. Meta-analysis helps in the identification of the most promising or urgent research question, and may permit a more accurate calculation of the sample sizes needed in future studies.

In this study, meta-analysis of 50 clinical studies conducted over 30 years in 4490 patients, was performed to evaluate the efficacy and short- and long-term safety of Liv.52 in HA. Highly significant reduction in the mean recovery period and no adverse events was noted. Therefore, it may be concluded that Liv.52 tablets and syrup are effective and safe in the management of hepatitis A.

Hepatoprotective Role of Liv.52 Against Hepatitis Induced by Antitubercular Drugs

Khare A.


Introduction

Antitubercular treatment (ATT) is known to cause various harmful effects such as increased streptomycin toxicity in the aged and increased chances of retrobulbar neuritis in the pediatric group, even when given in properly calculated doses based on the age and body weight of the patient. Other common side effects range from temporary nausea and vomiting to severe jaundice.

Material and Methods

The present study was conducted over a period of 2 years at the Department of Medicine, Institute of Medical Sciences, Banaras Hindu University, correlating chest and tuberculosis with the nephrology unit. A total of 70 cases of pulmonary tuberculosis were studied to help reveal any hidden incidences of renal involvement and focus on modifications required in the line of treatment, with particular reference to adverse effects. The patients were in the age group of 12 to 60 years (average age 35.82 years) and the male to female ratio was 5:2.

Liv.52 was given to every alternate patient to judge its effectiveness in preventing hepatic damage. Patients weighing more than 30 kg received Liv.52 as a dosage of 2 tablets TID and those weighing more than 50 kg received Liv.52 at a dosage of 3 tablets TID.

All the cases were thoroughly investigated after obtaining a detailed clinical history. A thorough medical examination was conducted and the results of the following tests were recorded on a pre-determined proforma.

- Hematology tests (hemoglobin, total and differential counts, and erythrocyte sedimentation rate)
- Biochemistry tests (blood sugar, blood urea, liver function test, serum creatinine, and electrolytes)
- Radiological tests (x-ray chest, x-ray [kidney urinary bladder], and ultrasound of the abdomen were done in all the cases to assess the pulmonary region, kidney size, and damages, respectively)
- Histopathology test (kidney biopsy was done in all the cases where kidney size was within normal limits)

Observations and Discussion

In the first group (Liv.52), only one patient out of 35 cases (2.8%) developed clinically apparent jaundice. In the other group, where Liv.52 was not administered, 5 out of 35 cases (14.2%) developed jaundice. All the 6 patients who developed jaundice had raised levels of serum bilirubin, alkaline phosphatase, serum glutamic oxaloacetic transaminase, and serum glutamic pyruvic transaminase. Interestingly, the patient who was receiving Liv.52 along with ATT developed jaundice only on the 6th day, which could have been due to hypersensitivity caused by rifampicin. However, in the other group, two patients developed jaundice in the 8th week, one in the 10th week, and the remaining two in 14 weeks’ time. This long duration rules out any allergic (hypersensitivity type) reaction and confirms that the jaundice was due to liver damage.

Conclusion

The results clearly indicate that Liv.52 protects patients against hepatotoxicity due to antitubercular drugs.
Although most cases of immunoglobulin A (IgA) nephropathy are idiopathic, several diseases are associated with IgA nephropathy. Of these, chronic liver disease resulting from hepatitis B or C virus infection has been reported as a secondary cause of IgA nephropathy. Recently, hepatitis A virus (HAV)-associated kidney disease has received attention because acute kidney injury can occur as a complication of HAV infection, generally caused by acute tubular necrosis or interstitial nephritis. However, unlike IgA nephropathy related to hepatitis B or C, HAV-associated IgA nephropathy is extremely rare and long-term outcomes have not been reported yet. In this study, the researchers describe a case of spontaneous remission of IgA nephropathy associated with serologically documented HAV infection. The patient presented with microhematuria and moderate proteinuria, but acute kidney injury did not occur during active hepatic infection. Kidney biopsy specimens clearly showed mesangial IgA deposits with intact tubules and interstitium. Serum liver enzyme levels returned to reference values 1 month after the onset of acute hepatitis, but urinary protein excretion remained increased. Approximately 1 year later, urinary abnormalities were resolved and a second biopsy showed no mesangial IgA deposits. These findings suggest that IgA nephropathy can transiently accompany HAV infection, but may not progress to chronic glomerulonephritis after recovery from HAV.

**Expert comments**

Interview with Dr Payal Jain, MBBS, MD (Abd)

**Hepatitis A Infection**

How prevalent is hepatitis A infection?

Hepatitis A (HA) occurs sporadically and in epidemics globally, with a predisposition for cyclic recurrences. Worldwide, approximately 1.4 million people get infected with hepatitis A virus (HAV) annually.

The prevalence is high in developing countries with poor sanitary conditions and hygienic practices. Lifetime risk of HAV infection is >90% in these countries, is >90%. Most infections occur in early childhood. Epidemics are less common as older children and adults are generally immune. In developed countries with good sanitary conditions and hygienic practices, infection rates are low. HAV infection could occur among adolescents and adults in high-risk groups such as injecting-drug users, people travelling to high-risk regions, homosexual men, and in isolated populations.

What are the clinical manifestations of HA? How is HA transmitted?

The clinical manifestations of HA range from mild to severe, and include fever, nausea, malaise, loss of appetite, abdominal discomfort, diarrhea, dark-colored urine, and jaundice. Adults experience signs and symptoms of illness more often as compared to infants and young children, and the severity of infection and mortality increases in older age groups. Infected children of <6 years of age generally do not experience noticeable symptoms, and only about 10% develop jaundice. Among older children and adults, infection usually causes more severe symptoms, with jaundice occurring in >70% of the cases. These symptoms may appear 2 to 7 weeks after exposure (average 28–30 days).

HA is mostly spread by fecal-oral transmission. Waterborne outbreaks, though infrequent, are usually associated with sewage-contaminated or inadequately treated water. Blood borne transmission of HAV does occur but it is less common. Casual contact among people does not spread the virus.

What are the complications associated with hepatitis A infection? What measures should be taken to control the spread of hepatitis A?

Majority of patients with hepatitis A infection get recovered completely and the case fatality rate is low. Some patients could develop serious complications. About 15% of patients experience prolonged jaundice and/or relapses for several months. Certain patients can develop cholestatic hepatitis, in which bile channels get blocked. A few can develop fulminant (acute) liver failure and may require a liver transplant or if not treated can cause death.

HA can be effectively controlled by improved sanitation, regulation of food handling, and hepatitis A immunization. Supply of safe-drinking water and proper disposal of sewage within communities, in addition to personal hygiene practices, such as regular hand-washing, can help reduce the spread of HAV infection.

What is the role of Liv.52 in treating HA?

Liv.52 is a hepatoprotective medication helpful in the treatment of various liver disorders including viral hepatitis. Studies have shown that Liv.52 reduces clinical symptoms associated with viral hepatitis, normalizes hepatic enzyme levels, and plays a protective role in maintaining liver integrity and restoration of liver function. I regularly prescribe Liv.52 to my patients with infective hepatitis/hepatitis A.

**Hepatitis A Virus-related Late-onset Hepatic Failure: A Case Report**

Hayashi M, et al.


Late-onset hepatic failure, the least of the fulminating hepatic failures, has not occurred in patients with hepatitis A virus-related acute liver failure. This study reports a rare case of hepatitis A virus-related late-onset hepatic failure treated successfully by an emergent liver transplant. A 58-year-old Japanese woman who presented with fever and general malaise was diagnosed as having jaundice and liver dysfunction by a positive serum test for antihepatitis A virus IgM, which ultimately led to a diagnosis of acute hepatitis A virus associated hepatitis. Despite intensive treatment, her general condition was poor, and she developed a hepatic coma 79 days from the onset of the disease. Under a diagnosis of hepatitis A virus-related late onset hepatic failure, she was given a living-donor liver transplant 82 days from the start of the disease. The resected native liver revealed submassive necrosis with marked cholestasis, compatible with late-onset hepatic failure.

Today, 5 years after the transplant, she is alive and well with no signs of recurrent hepatitis A virus-hepatitis. This case should alert the physician to the clinical management of a patient with hepatitis A virus-related acute liver failure.

**Hepatitis A Virus Load in Relation to Severity of the Infection**

Hayashi M, et al.

Hepatitis A (HA) occurs sporadically and in epidemics globally, with a predisposition for cyclic recurrences. Worldwide, approximately 1.4 million people get infected with hepatitis A virus (HAV) annually.

The prevalence is high in developing countries with poor sanitary conditions and hygienic practices. Lifetime risk of HAV infection is >90% in these countries, is >90%. Most infections occur in early childhood. Epidemics are less common as older children and adults are generally immune. In developed countries with good sanitary conditions and hygienic practices, infection rates are low. HAV infection could occur among adolescents and adults in high-risk groups such as injecting-drug users, people travelling to high-risk regions, homosexual men, and in isolated populations.

A correlation between hepatitis A virus (HAV) genomes and the clinical severity of hepatitis A has not been established. The viral load in sera of hepatitis A patients was examined to determine the possible association between hepatitis A severity and HAV replication. One hundred sixty-four serum samples from 91 Japanese patients with sporadic hepatitis A, comprising 11 patients with fulminant hepatitis, 10 with severe acute hepatitis, and 70 with self-limited acute hepatitis, were tested for HAV RNA. The sera included 83 serial samples from 20 patients. Viral load was measured by real-time reverse transcription polymerase chain reaction (RT PCR). The detection rates of HAV RNA from fulminant, severe acute, and acute hepatitis were 10/11 (91%), 10/10 (100%), and 55/70 (79%), respectively. Mean values of HAV RNA at admission were 3.48 ± 1.30 log copies/mL in fulminant, 4.19 ± 1.03 in severe acute, and 2.65 ± 1.64 in acute hepatitis. Patients with severe infection such as fulminant hepatitis and severe acute hepatitis had higher initial viral load than patients with less severe infection (<P< .001). Viremia persisted for 14.2 ± 5.8 days in patients with severe infection and 21.4 ± 10.6 days in those with acute hepatitis after clinical onset (P> .19). HAV RNA was detectable quantitatively in the majority of the sera of hepatitis A cases during the early convalescent phase by real-time PCR. Higher initial viral replication was found in severely infected patients. An excessive host immune response might follow, reducing the viral load rapidly as a result of the destruction of large numbers of HAV-infected hepatocytes, and in turn severe disease might be induced.
In infective hepatitis...

**Liv.52**

(DROPS, SYRUP, TABLET, DS SYRUP, DS TABLET)

Unparalleled in liver care

Restores the metabolic efficiency of the liver
- Protects the hepatic parenchyma
- Promotes hepatocellular regeneration
- Prevents the loss of functional integrity of cell membranes
- Possesses antiperoxidative activity
- Normalizes the liver function tests
- Shortens the disease recovery period

**Dosage**

**Adults:**
- Syrup: 10 ml t.i.d.
- Tablet: 2 tablets t.i.d.

**Drops:**
- Infants: 5 to 10 drops t.i.d.
- Children: 10 to 20 drops t.i.d.

**Liver 52**

Unparalleled in liver care

**Conclusion**

In conclusion, this study indicates that acute hepatitis A superimposed on chronic HBV infection is associated with serious morbidity and mortality. Especially in terms of ALF, HBsAg-positive patients with acute hepatitis A are at an approximately 9-fold increased risk.

The main shortcomings of the present study are its retrospective nature and the fact that the study was conducted in a tertiary referral hospital, therefore there may be selection bias for patients who were referred for intensive care or liver transplantation. However, this study is significant in the sense that it is one of the few analyses on the difference in clinical features between HBsAg-positive and HBsAg-negative patients in this HBV endemic area. Furthermore, the results of this study also indicate that acute hepatitis A is no longer a mild disease, at least in HBsAg carriers.

**Laugh lines**

A man and woman were having marriage problems, and decided to end their union after a very short time together. After a most brief attempt to reconcile, the couple went to the court to finalize their break-up. The judge asked the husband, “What has brought you to this point? Were you not able to keep this marriage together?”

The husband said, “In the six weeks that we’ve been together, we haven’t been able to agree on one thing.”

The wife said, “Seven weeks.”

Fred, Jim, and Scott were at a convention together and were sharing a large suite on the top of a 75-story skyscraper.

After a long day of meetings, they were shocked to hear that the elevators in their hotel were broken and they would have to climb 75 flights of stairs to get to their room.

Bill said to Jim and Scott, “Let’s break the monotony of this unpleasant task by concentrating on something interesting. I’ll tell jokes for 25 flights, Jim can sing songs for the next 25 flights, and Scott can tell sad stories for the rest of the way.”

At the 26th floor, Bill stopped telling jokes and Jim began to sing. At the 51st floor, Jim stopped singing, and Scott began to tell sad stories.

“I will tell my saddest story first,” he said. “I left the room key in the car!”

**What Factors Determine the Severity of Hepatitis A-related Acute Liver Failure?**

The reason(s) that hepatitis A virus (HAV) infection may progress infrequently to acute liver failure are poorly understood. In this study, the researchers examined host and viral factors in 29 consecutive adult patients with HAV-associated acute liver failure enrolled at 10 sites participating in the US ALF Study Group. Eighteen of the 24 acute liver failure sera were polymerase chain reaction positive, whereas 6 patients had no detectable virus.

HAV genotype, and nucleotide substitutions were not associated with survival included detectable serum HAV RNA, while age, gender, liver failure within 1 month of each other. Predictors of spontaneous failure cases (chi-square test, <.01).

The difference between failure and success is doing a thing nearly right and doing a thing exactly right. — Edward Simmons

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**Study Reports Acute Pancreatitis in Acute Viral Hepatitis**

Several studies have reported association of acute viral hepatitis with acute pancreatitis. This study was conducted to find out the frequency of pancreatic involvement in acute viral hepatitis in the Nepalese population. Consecutive patients of acute viral hepatitis presenting with severe abdominal pain between January 2005 and April 2010 were considered for this study. Patients with history of significant alcohol consumption and gall stones were excluded. Acute viral hepatitis was diagnosed by clinical examination, liver function test, ultrasound examination, and confirmed by viral serology. Pancreatitis was diagnosed by clinical presentation, biochemistry, ultrasound examination, and computerized tomography (CT) scan.

The results of the study showed that severe abdominal pain was present in 38 of 382 serologically confirmed acute viral hepatitis patients. Twenty-five patients were diagnosed to have acute pancreatitis. Pancreatitis was mild in 14 and severe in 11 patients. The etiology of pancreatitis was hepatitis E virus in 18 and hepatitis A virus in 7 patients. Two patients died of complications and one was lost to follow-up. The remaining patients recovered from both pancreatitis and hepatitis on conservative treatment.

From the study findings, it was concluded that acute pancreatitis occurred in 6.5% of patients with acute viral hepatitis. Cholelithiasis and gastric ulcers are the other causes of severe abdominal pain. It was also concluded that a majority of patients can recover with conservative management.