CODEN [USA]: IAJPBB ISSN : 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES
SJIF Impact Factor: 7.187
https://doi.org/10.5281/zenodo.18523593

Available online at: http://www.iajps.com *Research Article*

# A PROSPECTIVE OBSERVATIONAL STUDY ON PREVALENCE AND MANAGEMENT OF HEPATITIS AT A TERTIARY CARE TEACHING HOSPITAL

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**Abstract :**

***Background:*** *This article focuses on viral hepatitis, which accounts for more than 50% of cases of acute hepatitis, mostly in the emergency department setting. Hepatitis is a general term for inflammation of the liver and can be caused by a variety of infectious (such as bacterial, fungal, viral, and parasitic organisms) and non-infectious (such as alcohol, drugs, autoimmune diseases, and metabolic diseases).Three viruses: hepatitis A (HAV), hepatitis B (HBV), and hepatitis C (HCV). All three of these viruses can cause an acute illness that manifests as jaundice, malaise, exhaustion, nausea, and stomach pain. Furthermore, chronic infection can result from acute HBV and HCV infection. Chronically infected patients may develop cirrhosis and hepatocellular carcinoma (HCC). Additionally, carriers of chronic hepatitis are still contagious and can spread the illness for many years.*

***Aims and Objectives :*** *To study the prevalence and management of hepatitis in the general medicine department and to analyse the prescribing patterns , and immunoglobulin administration for various types of hepatitis.*

***Methodology:*** *It is a prospective and observational study carried out over a period of six months at Osmania General Hospital Afzal Gunj , Hyderabad. Patients of both sex who are diagnosed with hepatitis age above 18 years were selected for the study.A total of 100 cases were collected using a predesigned data collection form and statistical approach to evaluate the prevalence and management of hepatitis.*

***Results:*** *There is a predominance of hepatitis in male ( 86%) in the study population. More cases are recorded for patients below ( 41-50 ) years of age group. Whereas, the diagnosis was made on the basis of laboratory findings and serological results which reveal viral hepatitis (A-E). Moreover, the clinical manifestation were found to be jaundice , yellowish discolouration of eyes and skin , abdominal pain. The commonly prescribed drugs for hepatitis were ceftriaxone , pantoprazole, metronidazole , furosemide , ampicillin , L-ornithine L-aspartate and meropenem.*

***Conclusion :*** *In the present study 100 cases of hepatitis were recorded and various prescription patterns were observed, however there is a predominance of alcoholic hepatitis in male. Majority of the cases are recorded in patients below 50 years of age, and other risk factors that trigger the disease condition include smoking , alcohol abuse for hepatitis. Likewise, laboratory investigations were carried out in most cases, and there was a lack or standard treatment pattern was recorded.*

***Keywords :*** *Hepatitis , Jaundice , Immunoglobulins , Cirrhosis , Hepatocellular Carcinoma.*

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***Please cite this article in press Hafsa Jabeen et al., A Prospective Observational Study On Prevalence And Management Of Hepatitis At A Tertiary Care Teaching Hospital, Indo Am. J. P. Sci, 2026; 13(01).***

## INTRODUCTION:

The word "hepatitis" describes inflammation of the liver. The liver is an essential organ that combats infections, breaks down nutrients, and filters blood. The liver is ill. Hepatitis can be caused by excessive alcohol use, pollution, some medicines, and certain medical conditions. If it is irritated or wounded, its performance may be affected. However, a virus usually causes hepatitis.[1]

Hepatitis is the most prevalent cause of acute and chronic liver diseases worldwide. The World Health Organisation (WHO) estimates that 240 million of the 2 billion people who are exposed to viruses are chronic carriers.[2]

Viral hepatitis has been a challenging issue since ancient times, as evidenced by reports of outbreaks in China 5000 years ago and Hippocrates' accounts of comparable jaundice on the island of Thassos in the fifth century BC [3,4]

**Hepatitis A, B, C, D, and E viruses are the main five agents that cause viral hepatitis**.

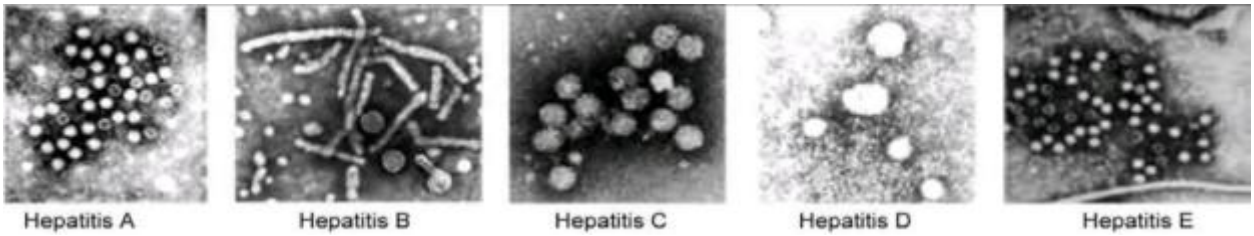


**Figure 1 : Electron micro-graph of viruses.**[5]

**Patients with viral hepatitis often experience 4 phases :**

Phase 1 (viral replication phase): During this phase, patients are typically asymptomatic and hepatitis-related indicators are detected in laboratory tests.

Patients in phase 2 (also known as the prodromal phase ) typically exhibit anorexia, nausea, vomiting, malaise, pruritis, urticaria, arthralgias, and exhaustion. These individuals frequently receive incorrect diagnoses of viral illness or gastroenteritis.

Patients in this phase have dark urine and light-colored feaces. Phase 3 (icteric phase). Some patients experience right upper quadrant pain and jaundice along with enlarged livers.

Phase 4 (convalescent phase): Patients usually start to feel better, and tests in the lab reveal that the liver enzymes are returning to normal ranges. [6]

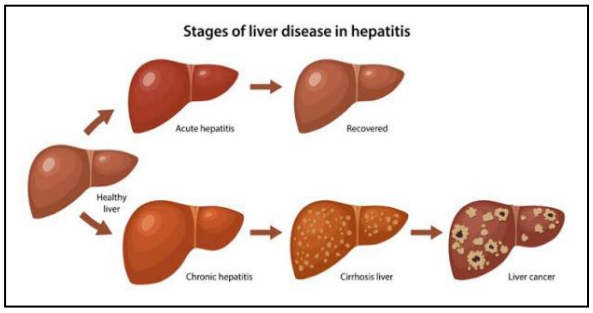


**Figure 2 : Stages of liver disease in hepatitis.**

**COMMON SYMPOMS IN HEPATITIS:**

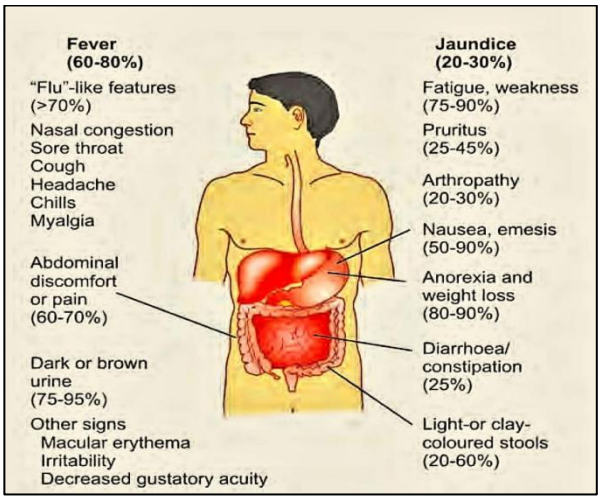


**Figure 3 : Common symptoms of hepatitis**.[7]

1.**CHRONIC HEPATITIS :**
It's defined as biochemical or serologic proof of inflammatory hepatitis that hasn't improved over the course of more than six months. Histologically, CH can be divided into three categories. The incidence of chronic hepatitis varies with age and virus type. Ninety percent of children who have hepatitis B at birth develop chronic hepatitis, but only one to five percent of adults develop chronic liver disease. Chronic hepatitis develops in 85–90% of cases of hepatitis C.[7]

**OLDER CLASSIFICATION :**
HBV, HCV, and HBV-HDV infections are frequently the cause of chronic persistent hepatitis. The portal tracts have been infiltrated by inflammatory cells, including lymphocytes, macrophages, and plasma cells. The hepatic

parenchyma is not affected; the infiltration is limited to the portal tracts.

**2. CHRONIC ACTIVE HEPATITIS:** This disorder is often caused by non-viral reasons such as auto-immune hepatitis, metabolic problems, antibiotics, and HBV, HCV, and HBV-HDV infections.
Pathologically, it is characterised by patchy fibrosis and necrosis that extend from the portal tracts into the hepatic parenchyma and cause cirrhosis. An overflowing portal inflammatory infiltration is leaking from portal tracts.
**3. CHRONIC LOBULAR HEPATITIS:** Chronic lobular hepatitis is characterised by lobular inflammation and spotty necrosis.
**NEW CLASSIFICATION:**

1. The reason.
2. Histologic grade or activity.
3. The degree or stage of development.

**Stages of hepatitis :**
This is divided based on the degree of fibrosis.

**Stage 0**- No fibrosis

**Stage 1**- Mild fibrosis

**Stage 2**- Moderate fibrosis

**Stage 3**- Severe fibrosis

**Stage 4**- Cirrhosis.

**Correlation between Earlier and Contemporary Classification of Chronic Hepatitis**

| *Old classification* | *New classification* | |
|---|---|---|
| | *Grade* | *Stage* |
| Chronic persistent | Minimal or mild | 0 or 1 |
| Chronic lobular | Mild or moderate | 1 |
| Chronic active | Mild, moderate or severe | 1, 2 or 3 |

**Figure 4 : classification of chronic hepatitis.**[7]

**GENERAL DIAGNOSTIC CRITERIA:**

1. Examining the abdomen for an enlarged liver.
2. Examining for jaundice in the skin and eyes Liver biopsies: Liver biopsies for the pathologic classification of chronic persistent hepatitis, chronic active hepatitis, or cirrhosis.
3. The amount of proteins and enzymes the liver produces in the blood can be used to gauge the extent of liver damage. Prothrombin time, albumin, bilirubin, gamma glutamine transferase, aspartate transaminase (AST), alanine transaminase (ALT), and lactate dehydrogenase are examples of enzymes and proteins. 8A number of serologic "markers" or combinations of markers are used to identify the different stages of HBV infection in patients who have an acute or chronic infection, are immune to HBV due to a previous infection or vaccination, or are susceptible to infection. Hepatitis B surface antigen, abbreviated HBsAg.
   Serological markers for HBV infection consist of HBsAg, anti-HBs, HBeAg, anti-HBe, and anti-HBc IgM and IgG.[9]

**TYPES OF HEPATITIS**

**HEPATITIS A :** Infection with HAV, an RNA virus, which is categorised as a picornavirus, causes hepatitis A. In 1979, it was isolated for the first time [10]. The ailment is typically mild and self-limiting. seldom progresses to FHF (fulminant hepatic failure).[11]
**Mode of transmission**: Faecal-oral route, person-to-person, ingestion of contaminated food and water, poor sanitary conditions, blood transfusions, and sexual contact.
**Epidemiology :** In 1973, Feinstone discovered a spherical 27 nanometre particle in the stool of hepatitis A patients using immunological electron microscopy [12]. Over half of the 1.4 million infections and 7134 deaths caused by the Hepatitis A virus (HAV), an RNA virus that is a member of the picornaviridae family, occurred in Asia in 2016.In 2006, the annual incidence rate in the United States was reported to be 2 cases per 100,000 individuals. Recent disease outbreaks indicate that, in comparison to 2013–2015, infections rose by 294% between 2016 and 2018.[13]
**Current outbreak :** In 1988, around 300,000 people in Shanghai experienced one of the biggest outbreaks in modern times after consuming raw clams.[14] In recent years, many outbreaks connected to specific food products have impacted more than 300 people from different parts of the United States (2013-2019). Fresh blackberries, frozen strawberries, pomegranate seeds, and raw scallops have all been connected to outbreaks.[15]

**Clinical presentation:** HAV takes 2-4 weeks to incubate. Common signs of a HAV infection include fever, malaise, and jaundice.Additional symptoms include weakness, exhaustion, nausea, vomiting, stomach discomfort, myalgia, arthritis, diarrhoea, and anorexia.[15]
**Laboratory tests:** Serum aminotransferases are usually higher than 1000 U/dL, total bilirubin is often 10 mg/dL, and alkaline phosphatase is frequently 400 U/L. Serum alanine aminotransferase (ALT) is usually higher than aspartate aminotransferase (AST). [16]
Severe hepatic disorders, hospitalisations, and increased mortality are often more common in older people.These results can be explained by the liver's decreased ability to regenerate and the elderly population's comparatively weaker immune system.Males are said to have a high mortality rate in addition to old age.[17]

**Diagnosis:** Serum contains anti-HAV (anti-HAV) specific antibodies. The diagnosis is confirmed by the presence of immunoglobulin (Ig) M anti-HAV. The antibodies can be found when the symptoms first appear. Serum IgM levels typically remain high for up to four months after the onset of symptoms, peaking during the acute illness[18]. Immunity is usually assessed using HAV total antibody to determine if exposure to HAV was unintentional or a result of immunisation [19].IgM antibodies may indicate a false positive test, a silent disease, or a previous HAV infection with persistent antibodies. when they exist but don't show any symptoms.

- Imaging studies or a liver biopsy are not required to make a diagnosis. When compared to non-HAV viral hepatitis, a liver biopsy may show substantial portal inflammation with generally less necrosis, Kupffer cell growth, acidophil bodies, or ballooning. [20]
- Treatment/control: Since there is no specific treatment for HAV, the primary focus is on symptom control. Improving sanitary conditions is still the main objective to lower transmission in the community. HAV infections have traditionally been prevented with immunoglobulins. With the exception of infants under 12 months old, the use of immunoglobulins has largely been ceased once an effective vaccine became available. Since 2007, the hepatitis A vaccine has been approved as a post-exposure prophylactic for immune-competent individuals without chronic liver disease who are between the ages of 12 months and 40. [21]

**GOALS OF THERAPY:**

- Complete clinical resolution
- Reducing complications
- Maintaining normal liver functions
- Prevention and reducing transmission.

**RECOMMENDED DOSING OF HEPATITIS A VACCINES**

TABLE 25-3 Recommended Dosing of Hepatitis A Vaccines

| Vaccine | Vaccinee's Age (years) | Dose | Number of Doses | Schedule (months) |
|---|---|---|---|---|
| Havrix | 1–18 | 720 ELISA units | 2 | 0, 6–12 |
| | ≥19 | 1440 ELISA units | 2 | 0, 6–12 |
| Vaqta | 1–18 | 25 units | 2 | 0, 6–18 |
| | ≥19 | 50 units | 2 | 0, 6–18 |
| Twinrix | >18 | 720 ELISA units | 3 | 0, 1, 6 |
| | >18 (accelerated schedule) | 720 ELISA units | 4 | 0, 7 days 21–30 days + 12 months |

**Figure 5:Recommended dosing of hepatitis A vaccines.** [22]

The combination hepatitis A and B vaccine also contains 20 mcg of hepatitis B surface antigen and requires a three-dose schedule

Liver transplantation's function: Acute liver failure occurs in less than 1% of cases with acute HAV infections. The majority of these patients recover with symptomatic treatment, with only 31% needing an immediate liver transplant to cure their fulminant sickness.[23]

**Prevention :**

Hepatitis A vaccination: if the patient is older than 16, administer 2 ml intramuscularly (IM) into the deltoid twice, separated by 2-4 weeks. The vaccine is a human diploid cell-grown inactivated protein vaccine (Harvix). The duration of immunity is one year. If a booster is given after six months, immunity lasts for ten years.[24]

**HEPATITIS B**

There are eight different genotypes of the DNA virus that causes hepatitis B. Hepatitis B is caused by an HBV infection. It was first isolated in 1965. Chronic hepatitis B (CHB) viral infection is a well-known global health issue.
The Orthohepadnavirus species, which causes the partially double-stranded DNA virus known as hepatitis B virus (HBV), is a member of the Hepadnaviridae family of viruses. This virus causes the illness known as hepatitis B.

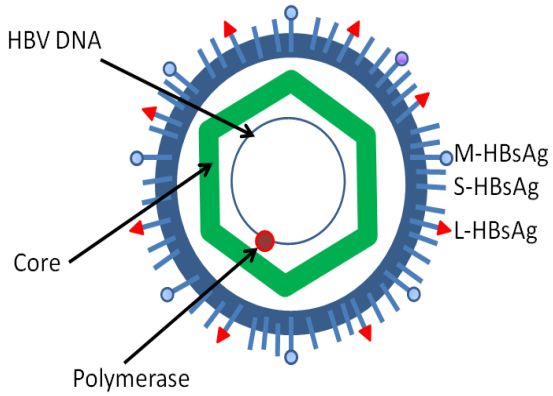


**Figure 6 : The structure of hepatitis B virus.** [25]

**Mode of transmission :**

- mother to neonate (vertical transmission)
- intravenous drug use
- male-to-male sex
- heterosexual sexual contact.[26]

**EPIDEMIOLOGY:** It was predicted that 257 million people globally would be infected with the chronic hepatitis B virus (HBV) in 2015, with North America having the lowest prevalence and the Western Pacific and African regions having the highest percentage of cases (68%). In 2017, the majority of liver cancer deaths and an estimated 29% of cirrhosis deaths globally were attributed to HBV.

**Clinical presentation:** Flu-Like syndrome,polyarticular arthritis, acute urticaria, membrabeous glomerularonephritis, IgA nephropathy.

**Risk Factors :** • The birth of an infected person (those born in the US who were not immunised as newborns and whose parents were born in a hepatitis B-endemic country). • Sharing tainted needles, syringes, or other equipment used in the manufacture of drugs.• Having sex with a partner who has hepatitis B.

**Laboratory Investigation :** During the acute phase: ALT and AST levels rise between 1,000 and 2,000 IU/L, total bilirubin levels rise, and the HBV viral load increases quickly in the range of 10,000–10,000 ng/mL. The diagnosis of acute HBV is confirmed by the development of positive HBsAg and IgM core antibody (HBcAb).[27]

**Diagnosis :** After the patient has recovered from the infection, both positive IgG HBcAb and HBsAb will be present. However, if the patient develops a chronic HBV infection and no HBsAb is detected, HBsAg will continue even if IgG HBcAb is present. Furthermore, the amount of aminotransferases, HBV DNA, and HBeAg status help determine the stage of the illness and direct therapy choices. The final category consists of people who have been vaccinated but have never come into contact with HBV.[28]

**Treatement :** IFN-α 2a and 2b—SC weekly or 2b either peginterferon 2a 180 mcg 100 mcg weekly for 48 weeks, or 5 million units daily or 10 million units three times a week for four to six months. When liver disease hasn't fully recovered, they shouldn't be utilised. (liver disease that is decompensated). The half-life and bioavailability of IFN are increased when it is pegylated with polyethylene glycol (PEG). Adverse effects include Fatigue, myalgia, arthralgia, fever, bone marrow suppression (neutropenia, thrombocytopenia, anaemia), depression, alopecia, and thyroiditis. For 48 weeks, take Lamivudine orally once a day.Adefovir and entecavir are used to treat patients with co-infections of HIV and HBV who develop early lamivudine resistance.For 48 weeks, take 10 mg of adefovir orally every day.
For 48 weeks, take 0.5 mg of entecavir orally and 1 mg if you are resistant to lamivudine.Clevudine, entricitabine, and telbivudine are undergoing trials.Lamivudine, adefovir, and entecavir should be taken in conjunction with hepatitis B immunoglobulin therapy to prevent HBV recurrence after liver transplantation.

**Role of Liver transplantation :** Different strategies have been developed depending on the donor and recipient's serologies. For example, if necessary, a combination of HBV immunoglobulin during the perioperative and postoperative phases. Long-term use of high-genetic barrier nucleoside/nucleotide analogues has been shown to have the lowest rates of HBV recurrence and post-transplant decompensation. [29]

**HEPATITIS C :**

Hepatitis C is a small, enveloped, positive single-stranded RNA virus that was first discovered in 1989 as a member of the Flaviridae family and is the only member of genus Hepacivirus. There are seven main genotypes and 67 subgroups. Genotype 1 is the most prevalent genotype worldwide, accounting for around 50% of all HCV infections. The second most common HCV genotype, which is more common in south Asia, Australia, and some European countries, accounts for about one-third of HCV infections. Genotype 4 is more common in the Middle East, North Africa, and central and eastern sub-Saharan Africa, whereas genotype 2 is more common in Asia and West Africa. [30]

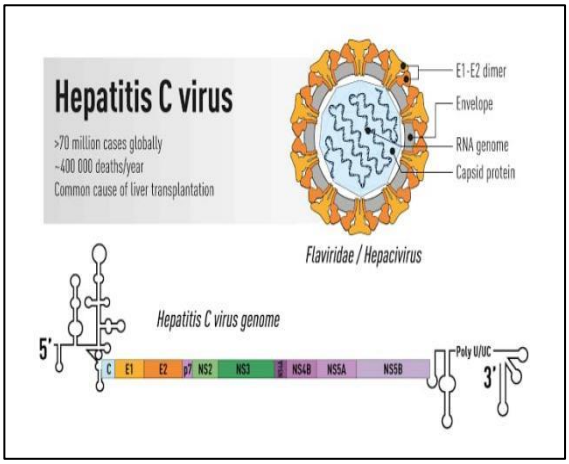


**Figure 7: structure of hepatitis C virus.** [31]

**Mode of Transmission :** Non-percutaneously through organ transplantation, blood transfusions, sexual activity, perinatal transmission, haemodialysis, religious scarification, body piercings, tattoos, and immunoglobulin injection. Percutaneously through needlestick injuries (including medical or intravenous drug use) containing contaminated blood. [32]

**Pathogenesis :** Four host-derived factors—Scavenger receptor class B type I, Occludin, Claudin-I, and CD81—help HCV enter the bloodstream [33] .Additionally, CD81 supports the entry of the viral particle into the hepatocyte by binding with it, either through the viral envelope protein (E)2 or other molecules.
HCV is regarded as a non-cytopathic virus, but once it reaches the hepatocyte, it multiplies and induces cell necrosis via inflammation brought on by

metabolism (hepatic steatosis, oxidative stress, and insulin resistance) and immune-mediated cytolysis (innate and adaptive immunity).
HCV's E2 regions and Non-structural Protein 5A (NS5A) are key players in the pathogenesis. Inhibiting the apoptotic process and triggering the release of anti-inflammatory interleukin, NS5A inactivates RNA dependent protein kinase (PKR) dis hepatocytes, facilitating viral multiplication. E2 protein blocks. [34]

**Clinical presentation:** weakness, anorexia, myalgia, arthralgia, nausea, diarrhoea, abdominal discomfort, sleep problems, depression, anxiety, and weight loss.

**Complications :** Within 20 years, 16% of people develop cirrhosis. Every year, 700,000 individuals worldwide pass away from cirrhosis, which is believed to develop in a liver infected with HCV after 20 to 30 years. Risk factors for the development of hepatic fibrosis in patients with chronic HCV infection include age > 40, alcohol consumption, HBV co-infection, immune-suppressed stage, marijuana usage, diabetic mellitus, schistosomiasis, severe hepatic necroinflammation, smoking, male sex, and white race. Viral load has not been linked to the chance of developing cirrhosis.
**Diagnosis:** Third generation enzyme immunoassay (EIAS) may detect antibodies against a variety of HCV antigens, such as HCV core, NS3, NS4, and NS5, as early as 8 weeks after exposure, with sensitivity and specificity of 99% after 2–6 months. Individuals with at least 10–15 IU/mL can be examined for HCV viremia using quantitative HCV RNA assays (Direct immunoassay). Although HCV core antigen immunoassay offers a substitute for HCV RNA testing, it is not sensitive enough to monitor treatment results.[35]

**Treatment:** Peg interferon alpha 2a 180 mcg SC once a week for HCV genotypes 1 and 4. Every week, provide 15 mcg/kg SC of interferon alpha 2b. For 48 weeks, take 1000–1200 mg of ribavirin daily orally. Peg interferon alpha 2a 180 mcg subcutaneously monthly for HCV genotypes 2 and 3. Adverse effects include haemolysis, Teratogenicity, and cough.

**Role of liver transplant:** Liver transplantation is usually the last resort for candidates whose HCV-decompensated cirrhosis or HCC does not respond to surgical resection. HCV infection continues to rank among the leading causes of liver transplants globally. Since 2012, the percentage of liver transplants for chronic HCV infection in the US has gradually decreased as a result of the introduction of DAAs. Using a liver transplant to treat acute liver failure brought on by an HCV infection has not been a routine procedure.[36]

**HEPATITIS D :**

Often called "delta hepatitis," the hepatitis D virus (HDV) is a liver infection that causes hepatitis D. Hepatitis D only develops in people infected with the hepatitis B and D viruses.The hepatitis D virus (HDV) cannot reproduce without the hepatitis B virus (HBV).
Hepatitis D virus (HDV) is present in about 5% of people with chronic hepatitis B virus (HBV) infections worldwide. HDV infection occurs when hepatitis B and D are contracted simultaneously (co-infection) or after hepatitis B has been contracted (super-infection).[37]
**Mode of Transmission :** contact with infectious blood, intravenous drug abusers, men who have sex with men.

**Epidemiology:** An extensive meta-analysis of 182 papers from 61 countries has found that between 62 and 72 million persons globally are infected with HDV. About 10.58% of HBsAg carriers also had HDV infection, which is far more than previously anticipated.[38]

**Clinical Presentation** : The clinical spectrum of HDV infection includes both inactive, asymptomatic carriers and acute liver failure. Concurrent HBV and HDV infection might cause a moderate, self-limiting illness or, less frequently, a severe acute hepatitis with spontaneous remission of both infections. On the other hand,, HDV **superinfection in chronic HBV carriers usually results in a prolonged clinical course.[39]**
**Treatment:** According to recent research, it is recommended to treat persistent HDV infection in people who have detectable viral RNA and signs of biochemical or histological active liver disease, especially if there is considerable fibrosis. Asymptomatic patients with normal liver enzyme levels, on the other hand, do not need treatment. [40]
**New Therapies :**

**BULEVIRTIDE: HDV ENTRY INHIBITORS**
Among the new therapies were bulevirtide and other HDV entry inhibitors. As previously mentioned, this medication was studied for 24 weeks as a monotherapy or in combination with tenofovir or PEG-interferon in patients who had HBV coinfection. The majority of bulevirtide users demonstrated a > 1 log10 decrease in HDV RNA after 24 weeks of treatment, with combination therapy outperforming monotherapy. Lastly, as mentioned in the hepatitis B section, REP 2139 was introduced as an inhibitor of virion release with the potential to decrease viral load in patients with HBV-HDV coinfection. Nevertheless, significant ALT increases were discovered in both studies. Individuals who have severe liver failure ought to think about getting a liver transplant. Plans for transfer to a transplant institution and

communication are crucial to saving these individuals. Despite appropriate medical care, the estimated rate of HDV infection recurrence following liver transplantation was 13.4%, with comparable outcomes for those with and without recurrence.[41]

**HEPATITIS E :**

Hepatitis E is an infection caused by the hepatitis E virus (HEV) that causes inflammation of the liver. A member of the Hepeviridae family, hepatitis E is an RNA virus.

**Mode of Transmission:** Sexual contact, haemodialysis, household, occupational, or prenatal exposure, the faecal-oral route, person-to-person contact, consumption of contaminated food or water, or drinking water contaminated with faecal matter are all potential routes of transmission. [42]

**Epidemiology:** According to the WHO, there are over 20 million new hepatitis E virus (HEV) infections per year in the world, which result in 3.3 million cases of acute hepatitis with symptoms and, according to another study, more than 44000 fatalities in 2015. The overall seroprevalence of the HEV in the United States between 1988 and 1994 was predicted to be 21%, despite the fact that the utility of modern antibody assays in seroprevalence studies is still up for debate. Other more recent investigations indicated a decreasing but increasing overall seroprevalence in Americans born between 2013 and 2014, peaking to 8.1% in 2015 and 2016, with age, female sex, and Asian ethnicity being important predictors of HEV seropositivity. [43]

**Current available treatment :** Prevention is still the best way to combat HEV-related illness. Maintaining the WHO's suggested hygienic and quality criteria for public water supplies can help achieve this. In 2011, China approved a recombinant vaccination to prevent HEV infection.A different study discovered that the HEV vaccine's protective efficacy was 86.8% 4.5 years after the initial immunisation. [44]

**Role of liver transplant**: Acute HEV infection seldom results in acute liver failure that necessitates a liver transplant, while chronic HEV may develop in the face of continuing immunosuppression. The prevalence and consequences of HEV infection varied significantly, according to an increasing number of epidemiologic research. For example, HEV is still the leading cause of acute viral hepatitis and acute liver failure (up to 44%) in a number of Asian countries, including Bangladesh and India. In contrast, it is an uncommon cause of acute liver failure in the US and Western Europe. [45]

**ALCOHOLIC HEPATITIS :**

Alcoholic hepatitis is a severe condition associated with alcohol-related liver damage. The abrupt development of jaundice, malaise, severe hepatomegaly, and subtle inflammatory features throughout the body are its defining features [46].Alcoholic hepatitis is a liver inflammation brought on by alcohol consumption.Individuals who drink excessively for a long time are more likely to acquire e than those who only drink moderately.

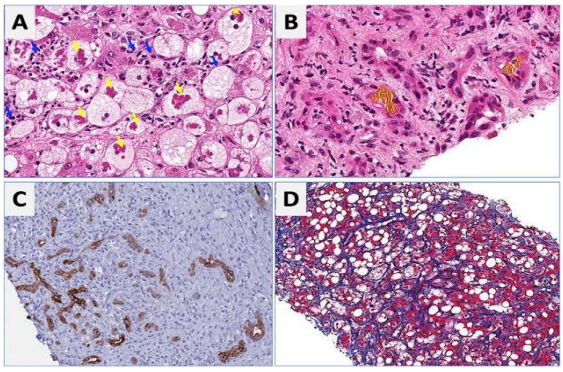


**Figure 8 : Characteristic histological finding in alcoholic hepatitis disease. [47]**

**Etiology :**The appearance of jaundice within 60 days of consuming a lot of alcohol (more than 50 grams per day for at least six months).Elevated aspartate aminotransferase (AST) of 50 U/L to 400 U/L and serum bilirubin greater than 3 mg/dL A ratio of AST:ALT (alanine aminotransferase) greater than 1.5. [47]

**Pathophysiology :** Alcohol enters an oxidative metabolic pathway in the hepatocytes, which reduces the ratio of nicotinamide adenine dinucleotide (NAD) to NADH. This promotes lipogenesis by stopping the oxidation of fatty acids and triglycerides.
Another known mechanism of alcohol-induced liver injury is the translocation of endotoxins from the intestines into the hepatocytes as lipopolysaccharides (LPS). When LPS binds to CD 14 and toll-like receptor 4 in hepatic Kupffer cells, reactive oxygen species (ROS) are released in a hail of particles. ROS cause the synthesis of cytokines such as interleukin-8, monocyte chemotactic protein 1 (MCP-1), platelet-derived growth factor (PDGF), and tumour necrosis factor-alpha (TNF alpha). Along with systemic clinical indications of alcohol toxicity, this leads to the increase of neutrophils and macrophages. [47]

**Clinical presentation:** Loss of appetite, Nausea and vomiting, Abdominal tenderness, fever often low grade, Fatigue and weakness, Jaundice, and ascites.

**Complications:** Variceal bleeding, Liver encephalopathy, Coagulopathy, Thrombocytopenia, Ascites, Bacterial peritonitis that develops suddenly, and Iron overload.

**Treatment :** The phosphodiesterase inhibitor pentoxifylline is thought to prevent the synthesis of tumour necrosis factor (TNF). Pentoxifylline monotherapy has been shown to be as effective as prednisolone and a placebo in treating patients with AH (Table 1). Prednisolone and pentoxifylline combined therapy has been compared to prednisolone or pentoxifylline monotherapy for effectiveness. [48]

**AUTOIMMUNE HEPATITIS :**

An immune system malfunction causes autoimmune hepatitis, a chronic liver disease. Antibodies released by your immune system inflame the liver tissues, resulting en hepatitis. These antibodies are frequently used to treat liver tissue infections. But autoimmune illnesses happen when your immune system unintentionally attacks and kills your own healthy cells. [49]

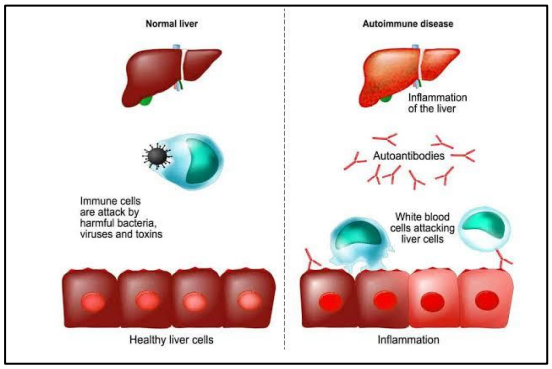


**Figure 9: Autoimmune liver disease.** [50]

**Treatment :**

Prednisone alone, 40–60 mg/day; Prednisone with azathioprine, 1-2 mg/kg/day, until remission is reached (more than 1-2 years). Retreatment is necessary in situations of relapse (20–30%).Liver transplantation for ESLD.Salvage therapy in refractory patients - Cyclosporine, tacrolimus, and mycophenolate mofetil • Lifelong low dosage therapy in some cases.[51]

**METHODOLOGY :**

**STUDY DESIGN**

A hospital based Prospective Observational study.

**LOCATION OF STUDY**

The study was carried out in Department of General Medicine, Osmania General Hospital, a Tertiary Care Teaching Hospital.

**STUDY PERIOD**

The study was carried out for the period of 6 months.

**STUDY POPULATION**

A sample size of 100 patients with complaints related to different types of hepatitis and liver diseases.

**SELECTION CRITERIA**

**(I) Inclusion criteria**

- Patients aged 18 years and above.
- All the patients admitted in the department of general medicine with hepatitis disease during the study period.
- Patients of either sex ( male and female ).

**(II) Exclusion criteria**

- Patients with COVID-19 infection
- Pregnant and lactating women.
- Patients with underlying diseases like HIV, AIDS and cancer.

**RESULTS AND OBSERVATIONS:**

Our study was carried out at Osmania General Hospital, a tertiary care teaching hospital, which provides an insight into the goals of observational research on different types of hepatitis, especially about their prevalence and prescribing practices for the management of hepatitis.

The data collected on patient profile form for Hepatitis during the study period , and was analysed according to various objectives. A total of 100 cases of hepatitis were collected from General Medicine Department ,Osmania General Hospital in 6 months duration which include both male and female population in a age of 18 - 70 years.

**1. DISTRIBUTION OF PATIENTS BASED ON AGE AND GENDER**

Out of the total number of 100 cases, 86% were male, and 14% of the patients were female. More cases are recorded in patients below 50 years of age. The maximum number of cases were seen in the age group range 31-40 years.

**Table 2 : DISTRIBUTION OF PATIENTS POPULATION BASED ON GENDER**

In the study there are male patients with distribution of (86%) and females ( 14%).

| Gender | No. Of patients | Percentage |
|---|---|---|
| **Male** | 86 | 86% |
| **Female** | 14 | 14% |

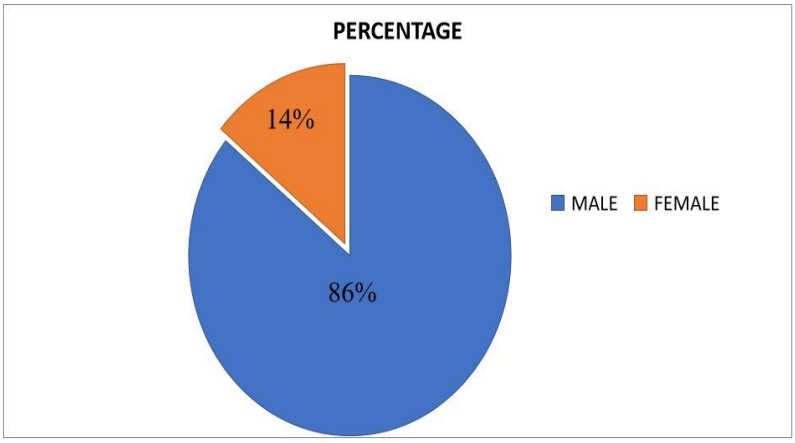


**Figure 10 : Distribution of patients based on gender.**

**TABLE 3 : DISTRIBUTION OF PATIENTS ACCORDING TO AGE GROUPS ALONG WITH GENDER DISTRIBUTION.**

In the study age groups between 18-20 years show (9%) , 21-30 years (19%), 31-40 years (27%), 41-50years (25%), 51-60 years (12%), 61-70 years (8%).

| Age Group | Male (n=86) | Female (n=14) | percentage |
|---|---|---|---|
| **18-20** | 6 | 3 | 9% |
| **21-30** | 18 | 1 | 19% |
| **31-40** | 22 | 5 | 27% |
| **41-50** | 21 | 4 | 25% |
| **51-60** | 11 | 1 | 12% |
| **61-70** | 7 | 1 | 8% |
| **Total percentage** | **85%** | **15%** | **100%** |

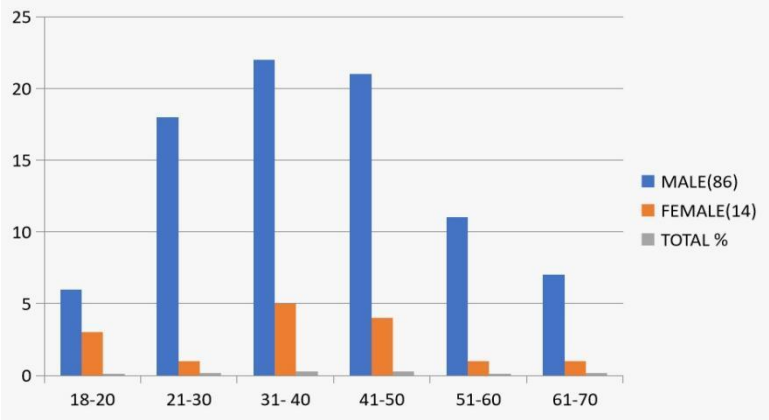


**Figure 11 : Graphical presentation of patients based on age groups.**

**Table 4 : DISTRIBUTION BASED ON PATIENTS WITH ADDICTIONS.**

In the study conducted, there are highest number of patients - alcoholic (38%), Both (S+A) (29%), No-Addiction (27%), Smokers (6%).

| Addiction | Total | Percentage |
|---|---|---|
| Alcoholic | 38 | 38% |
| Smoker | 6 | 6% |
| Both (Smokers+Alcoholics) | 29 | 29% |
| No Addiction | 27 | 27% |

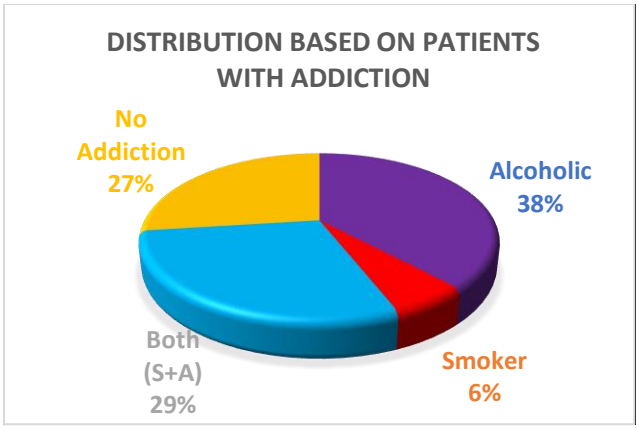


**Figure 12 : Patients with Addiction.**

**5.TREATMENT AND MANAGEMENT PATTERN :**

The most commonly prescribed drugs in types of hepatitis(Table /Figure ) are : ceftriaxone, cefotaxim, ampicillin, metronidazole,pantaprozole,furosemide,paracetamol,L-Ornithine L-Aapartate, urosodeoxycholic acid, levocitrizine,ondensetron.

**CLASS OF DRUGS**

- **ANTIBIOTICS :** ceftriaxone ,cefotaxim (cephalosporin), Ampicillin ( penicillin ), meropenem, rifaximin.
- **ANTIMICROBIALS :** Metronidazole
- **Proton Pump Inhibitors :** Pantaprozole.
- **DIURETICS :** Furosemide.
- **ANALGESICS :** Paracetamol.
- **HEPATOPROTECTIVE AGENTS :** L-ornithine L-aspartate( cholagogue)
- **GALLSTONE DISSOLUTION AGENTS :** ursodeoxycholic acid.
- **ANTI HISTAMINE :** Levocitrizine.
- **5-HT3 RECEPTOR ANTAGONIST :** Ondensetron.

**Table 5.1: DRUGS PRESCRIBED FOR DIFFERENT TYPES OF HEPATITIS**

In our study it is found that highest number of cases were of Alcoholic hepatitis ( 42%),Viral hepatitis (22%),Hepatitis B(17%),Hepatitis A(10%),Hepatitis C(7%), Drug induced hepatitis (2%).

| Infections | Drugs prescribed | No. of cases | percenatge |
|---|---|---|---|
| **Alcoholic hepatitis** | Ceftriaxone<br>Pantaprozole<br>Metronidazole<br>Furosemide<br>Ampicillin<br>Iron folic acid | 42 | 42% |
| **Viral hepatitis** | Metronidazole<br>Ondensetron<br>Levocitrizine<br>Ursodeoxycholic acid | 22 | 22% |
| **Hepatitis B** | Rifaximin,Ceftriaxone<br>Furosemide,Ondensetron<br>Ursodeoxycholic acid | 17 | 17% |
| **Hepatitis A (Acute infectious hepatitis)** | L-ornithine L-aspartate<br>Cefotaxim,Ondensetron<br>Ursodeoxycholic acid,<br>Pantaprozole. | 10 | 10% |
| **Hepatitis C** | Piperacillin/tazobactam<br>Meropenem, Rifaximin<br>Ceftriaxone,<br>Ursodeoxycholic acid | 7 | 7% |

| | | | |
|---|---|---|---|
| **Drug Induced hepatitis** | Ceftriaxone,ampicillin Pantaprozole, Furosemide,metronidazole L-ornithine L-aspartate | 2 | 2% |
| **Total** | | 100 | 100% |

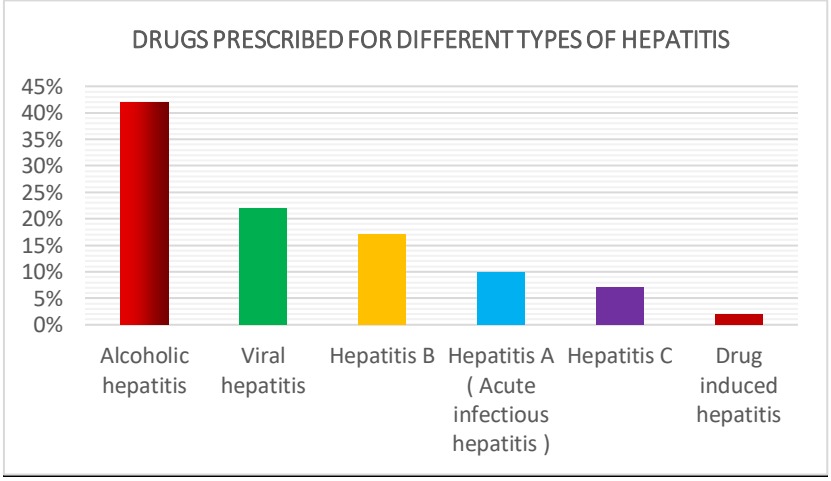


**Figure 13 : Drugs Prescribed for Different types of Hepatitis.**

**6. CO-MORBID CONDITIONS AND SYMPTOMS.**

In a study there are patients with co-morbid conditions with Hypertension (42%),HTN+DM (23%),Svere anemia (15%),AKI/CKD (10%),Diabetes (8%),Tuberculosis (2%).

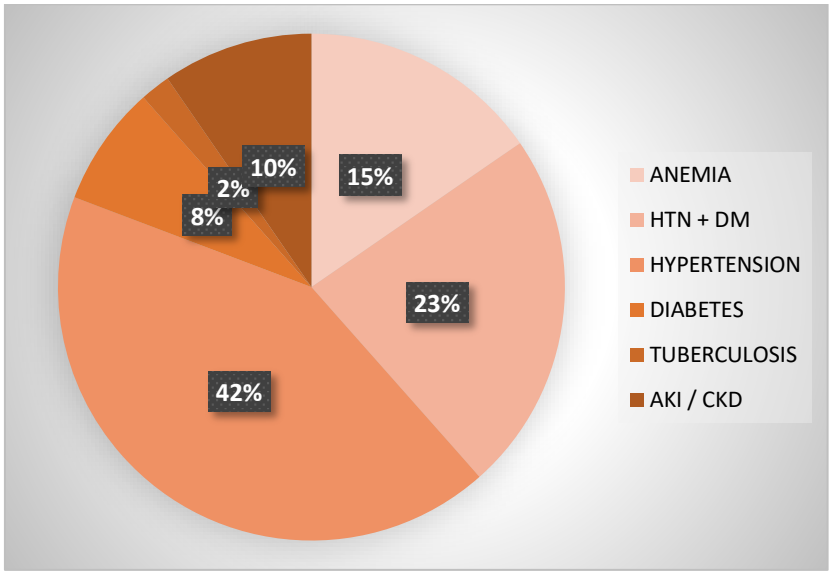


**Figure 14 : Distribution of patients based on co-morbid conditions.**

**Table 7 : DISTRIBUTION OF PATIENTS BASED ON SYMPTOMS**

In the study patient' distribution based on symptoms were with Jaundice (18%),slenomegaly (8%),Ascites(8%),Hepatomegaly ( 5%), Dyselectronemia (3%).As shown in the given table and figure below.

| Conditions | No. of cases | Percenatge |
|---|---|---|
| Jaundice | 18 | 18% |
| Spleenomegaly | 8 | 8% |
| Ascites | 8 | 8% |
| Hepatomegaly | 5 | 5% |
| Dyselectronemia | 3 | 3% |

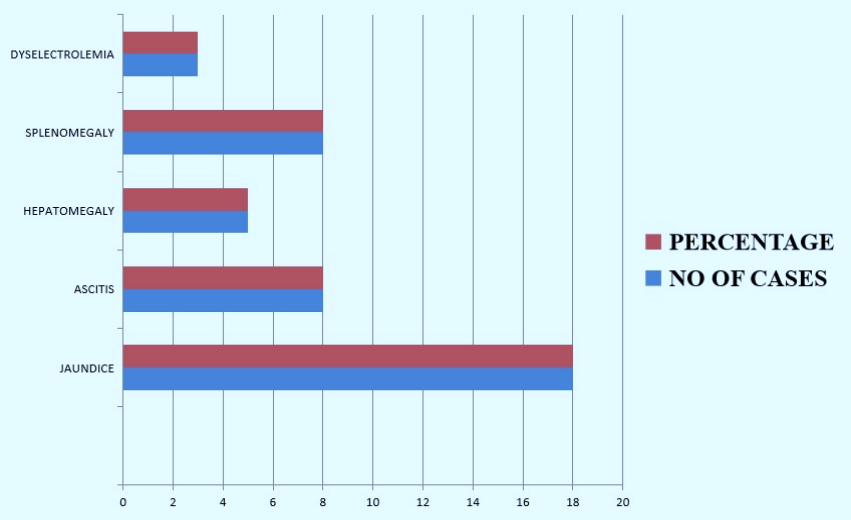


**Figure 15 : Graphical presenation of patients based on symptoms.**

**8 . PREVALENCE STUDY**

Prevalence is the proportion of a particular patient population found to be affected by a medical condition or disease at a specific time.

**Table 8.1 : Distribution of male population based on prevalence**

The total number of male patients were 86% and the most prevalent type of hepatitis among males is Alcoholic Hepatitis (46.51%) and the least prevalent is Hepatitis A (2.32%)

| Types of Hepatitis | No. of cases | percentage |
|---|---|---|
| Alcoholic hepatitis | 40 | 46.51% |
| Viral hepatitis | 19 | 22.09% |
| Hepatitis B | 15 | 17.44% |
| Hepatitis C | 6 | 6.97% |
| Acute infectious hepatitis | 4 | 4.65% |
| Drug induced hepatitis | 3 | 3.48% |

| | | |
|---|---|---|
| Hepatitis A | 2 | 2.32% |

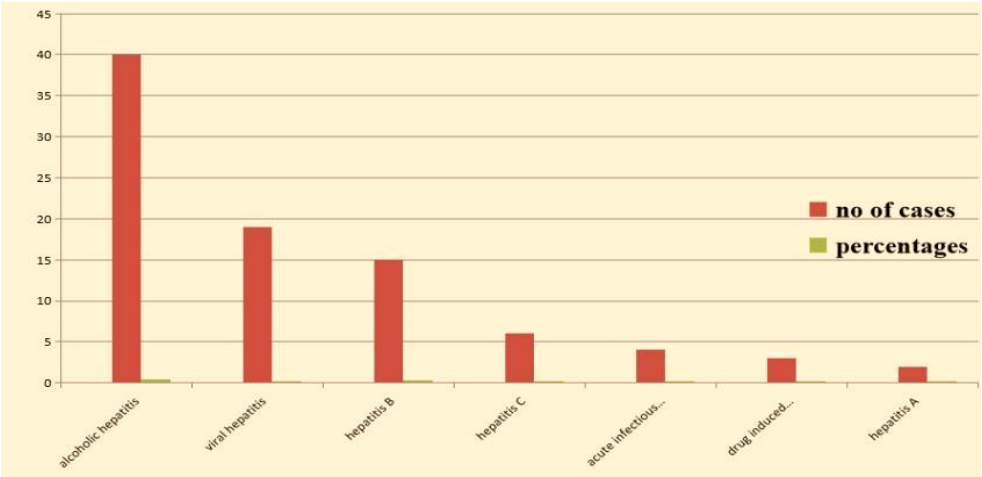


**Figure 16 : Graphical presenation of prevalence in male patient.**

**Table 8.2 : Distribution of female patients based on prevalence**

| Types of Hepatitis | No. of cases | percentage |
|---|---|---|
| Viral hepatitis | 4 | 28.5% |
| Hepatitis A | 4 | 28.5% |
| Acute infectious hepatitis | 3 | 21.4% |
| Hepatitis B | 1 | 7.14% |
| Hepatitis C | - | - |
| Alcoholic Hepatitis | - | - |
| Drug iinduced hepatitis | - | - |

**FEMALE PATIENTS = 14**

The total number of female patients were 14% and the most prevalent type of hepatitis among females is Viral Hepatitis (28.5%) and the least prevalent is Hepatitis A (7.14%).

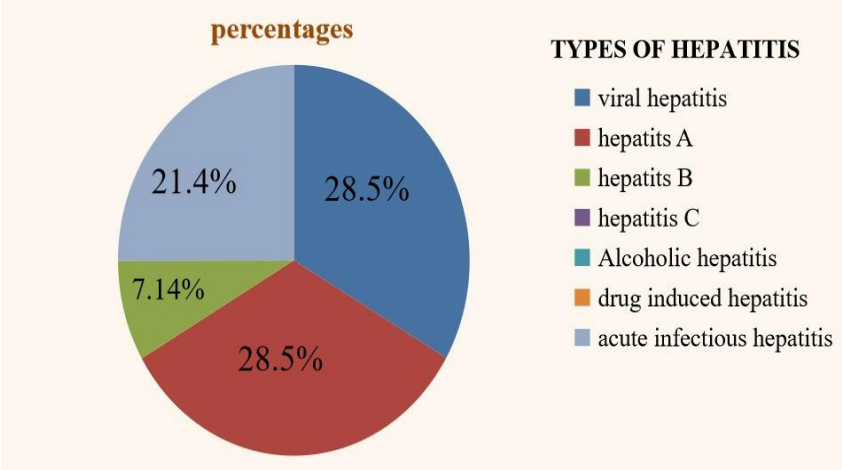


**Figure 17 : Graphical presentation of prevalence in female patients.**

## DISCUSSION:

In the present study we have performed a prospective observation study on prevalence and management of Hepatitis in tertiary care teaching hospital. In this present study evidence of different types of hepatitis were observed in 100 patients. Out of 100 patients 86 no. of cases were males which is 86% and 14 cases were observed in females that accounts for 14%. Hence, there is predominance) of male population in this study. This is accordance with a study conducted by simona leoni, alberto casabianca (2022) at division of internal medicine, hepatobiliary and immuno allergic diseases. IRCCS Azienda Ospedaliero universitaria di Bologna. Bolonga 40138, Italy. In a few other studies conducted by Gautam Ray et. Al (2017) in Br singh hospital gastro enterology unit, department of medicine, Kolkata, India and in contrast to simiar Debanjali Mitra, MA et. Al (2010) in Washington, DC, USA.

In this study we have also categorized the patient population according to their age groups and found that there is a increased prevalence in (31-40) years with a 27% as shown in the table 2. This study was as per the studies conducted by Debanjali Mitra, MA et. Al (2010) in Washington, DC, USA and Malewe Kolou et.al (2017).

In this study we have analyzed the distribution of patient population according to addictions (smoking, alcohol consumption) and found that there is a 38% of patient population with addiction of alcohol, 6% of patients with smoking as addiction and 29% of patients were found to be both (smokers and alcoholic) and 27% of patients with no addiction . This study was as per the studies conducted by Giovanni Addolorato et. Al (2016) at the Department of internal medicine, catholic university of Rome, Italy.

In this study, we have also categorised the patient population according to their age groups and found that there is an increased prevalence in the 31-40 years age group, with a 27% as shown in the table 2. This study was as per the studies conducted by Debanjali Mitra, MA et. Al (2010) in Washington, DC, USA and Malewe Kolou et al. (2017).

## CONCLUSION:

In summary, 100 cases of hepatitis were reported over a six-month period. According to the study, men are more likely than women to be exposed to the illness. The data indicate that the majority of patients with illness states are between the ages of 31 and 40. The study found that, out of all the kinds of hepatitis, alcoholic hepatitis had the largest number of cases (42).In the treatment of such situations, doctors recommend symptomatic treatments and supportive measures based on certain laboratory (LFT, CBP, serology) and radiographic examinations (liver ultrasound and liver biopsy). Addictions, including alcoholism and smoking, as well as a weakened immune system, are risk factors for the disease. However, the true aetiology of the illness is sometimes unknown. Abdominal discomfort (in the epigastric area), fever, anaemia, jaundice, diarrhoea, haematemesis, malena, constipation, decreased urine output, and anorexia are among the clinical symptoms that were frequently noted in cases and displayed by the majority of patients. According to the investigative reports completed throughout the patients' treatment, the liver function test was used to measure the levels of ALP, ALT, total bilirubin, direct and indirect bilirubin, and total protein in order to evaluate the liver's activities. In the majority of patients, a USG of the abdomen was recommended to assess the alterations in the morphology and physiology of the liver. Additionally, serological tests were performed to determine whether the patient had an A.B.C.D.E. viral infection. In the majority of instances, smoking and alcoholism are thought to be the primary causes of the illness. Ceftriaxone, Ursodeoxycholic acid, L-ornithine L-aspartate (hepamerz), cefotaxime,

lactulose solution, Pantoprazole, ondansetron, fluconazole, Doxycycline, pancreatin, aldactone, metronidazole, Rifaximin, and glutathione were the medications provided for the symptomatic therapy. Additionally, supportive measures were provided.

**ACKNOWLEDGEMENT**
Iwould like to extend my sincere gratitude to the professors of Mesco College of Pharmacy and the assistance of our guide. I would also like to acknowledge my peers for the discussions that inspired me throughout my academic journey. Special thanks to the ethics committee of Osmania General Hospital for the internship at their prestigious hospital department.

**LIST OF ABBREVIATIONS**
**HAV-Hepatitis A Virus**
**HBV-Hepatitis B Virus**
**HCV-Hepatitis C Virus**
**HDV-Hepatitis D Virus**
**HEV-Hepatitis E Virus**
**HbsAg- Hepatitis B surface antigen**
**HIV-Human immunodeficiency virus**
**AIDS-Acquired immunodeficiency syndrome**
**DILD-Drug induced liver disease**
**HCC-Hepatocellular carcinoma**
**ccDNA- Covalently closed circular DNA**
**CHB-Chronic hepatitis B**
**DAAs- Direct acting antivirals**
**ESLD-End stage liver disease.**

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