Hepatitis: An Overview on the diseases its types and treatment.

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Doi: https://doi.org/10.5281/zenodo.22686276
Received: 01 August 2026
Accepted: 07 September 2026

Abstract

Hepatitis is a liver disease caused by viruses, alcohol, toxins, and drugs. This review article elaborates on the types of hepatitis, epidemiology, pathogenesis, diagnosis, and treatment of the disease.

Keywords: Hepatitis, liver cells, virus, toxins, hepatocytes.

Introduction

Hepatitis is an inflammatory liver disease mostly caused by viruses such as Hepatitis A and C, alcohol use, toxins, and autoimmune disorders. Today, many healthcare organizations are raising awareness of the disease and its treatment, and new treatment methods are being developed. Hepatitis disease can be classified into three types, namely 1. Toxic hepatitis: Hepatitis caused by certain drugs, toxins or excessive alcohol consumption. 2. Autoimmune hepatitis: This occurs when liver cells start attacking their own cells as an autoimmune response. 3. Viral Hepatitis: Viral hepatitis is caused by viruses and is the most common form of hepatitis. It can be classified into the following types: hepatitis A, B,C, D, and E. This review article focuses on the types of hepatitis, its causes, and the prevention diagnosis , Precision Medicine & AI in hepatits and public awareness of the disease.

Hepatitis diseases and its types

Hepatitis can be mainly classified into three types

- ✓ Viral hepatitis: Hepatitis diseases caused by viral infections include Hepatitis A, B,C,D,E.
- ✓ Alcoholic hepatitis: Hepatitis caused by excessive alcohol consumption .
- ✓ Toxic hepatitis: Hepatitis caused by the influence of toxic chemicals, certain drugs, and dietary supplements.
- ✓ Autoimmune hepatitis is a chronic condition in which the body's immune system mistakenly attacks its own liver cells.

I. Viral Hepatitis [1]

Viral hepatitis has been around for 5000 years and was first reported in China. There are five types:

1. Hepatitis A : This is was first discovered by Feinstone in 1973, an RNA virus which is spherical in shape 27 nanometer particle. Approximately 1.4 million cases are reported annually worldwide .

Pathogenesis: Hepatitis A is a contagious disease and spreads from person to person through contact or via the faecal-oral route, mostly due to the consumption of contaminated food (as seen in the outbreak in Shanghai 1988, where people were affected due to the consumption raw clams). Sometimes, the disease spreads through blood transfusion, in which case Polymerase Chain Reaction is conducted on the Blood Donor. HAV enters the liver through the portal vein after traversing the small intestinal mucosa.

[2] Structure of Hepatitis Virus A : HAV is a tiny virus with 27-32nm. It is a non-enveloped, hepatotropic picornavirus. There are two non-coding regions at the 5' and 3' ends of the HAV genome. Structural proteins are encoded by the P1 region, whereas non-structural proteins are encoded by the P2 and P3 regions. There is only one serotype of human HAV, as its antigenic properties are well preserved.

[3] Most HAV outbreaks in developed countries occur among specific high-risk groups: travellers, men who have sex with men, people who use substances, and people facing homelessness. Despite the availability of safe and effective vaccines for decades, high-risk groups in high-income countries remain unprotected due to vaccine hesitancy. Hepatitis A infections can be prevented by vaccination, and several countries have successfully introduced universal mass-vaccination programs for children. However, 2the development of specific HAV antiviral treatment could be of great use in containing outbreaks, which may be particularly important in developed countries where a universal vaccination program is not recommended.

2. Hepatitis B Virus [1]: Chronic hepatitis B is estimated to affect 257 million people worldwide, mostly in the Western Pacific and African regions.Pathogenesis: HBV is a double-stranded DNA Virus that belongs to the *Hepadnaviridae family*. This was identified in 1963 and named the Australia antigen because the protein reacted with hemophilic antigen. The virus has a special tropism for hepatocytes, to which it adheres after the initial infection. DNA integrates into the nucleus through covalently closed circular DNA (cccDNA) bonds; hence, there is a possibility of HBV reactivation in inactive chronic illness due to reverse transcriptase. New viral molecules are released through cccDNA, which are released by exocytosis, leading to immunemediated liver injury.

Viral Structure[4]: HBV exists in three types 1. Dane Particles 2. Non Pathogenic virus 3. Naked Nucleocapsids. HBV is a double-stranded DNA Virus with an intermediate RNA of 42 nm in size. It is partially double-stranded and circular, containing 30210-3320 nucleotides encoding the vital enzyme for replication, the polymerase gene. Viral DNA is encapsulated with HpcAg protein, which is also found in hepatocytes and aids the cellular immune response.

3. Hepatitis C Virus [1]: HCV is infecting more than 70 million people around the world and its prevalence is increasing from 1990 to 2010. There are seven main genotypes and 67 subtypes of HCV. HCV genotype 1 represents half of the genotypes, making it more prevalent

Pathogenesis[5] HCV-induced liver damage occurs due to host factors and innate and adaptive immune responses to HCV infection. Here, the HCV affects and triggers an adaptive immune response. Cytotoxic T lymphocytes target infected cells; strong T cells clear the infection, whereas weak and exhausted T cells persist in the infection and lead to chronic disease. In chronic infection, the ongoing immune attack on infected hepatocytes tends to persist, causing inflammation, hepatocyte death and regeneration. HCV alters lipid metabolism, promotes steatosis, and induces stress.

Viral Structure [6]: HCV is a small enveloped RNA Virus in the *Flaviviridae family*. The genome encodes a single polyprotein that is classified into structural and non-structural proteins. It is an icosahedral, nucleocapsid, and lipid-enveloped virus obtained from the host cells.

4. Hepatitis D Virus[7]: HDV affects people already suffering from HBV as it requires the HbsAg surface protein to enter liver cells. The chronic presence of HDV causes more liver damage than HBV and may also lead to death from liver cancer. The presence of cirrhosis leads to more damage to liver cells due to HDV, and sometimes a patient progresses to HDV infection without cirrhosis.

[1] It is estimated that 62-72 million individuals are infected with HDV globally. This virus is predominantly found in the Amazon Basin, Western and Middle Africa, Middle East, and parts of Asia. Highly affected individuals are those who carry the HbsAg protein.

Pathogenesis[7]: HDV affects only those who suffer from HBV carrying HBsAg. It enters the liver cells through the NTCP receptors and uses the host RNA Polymerase in the nucleus to replicate by producing large amounts of HDV RNA and delta antigen in infected hepatocytes.

Viral Structure: HDV is a small circular enveloped virus, approximately 36–43 nm in diameter. It is circular with single-stranded negative RNA that is highly folded into a rod-like structure. Viral RNA is tightly bound to HDAg delta RNA to form an inner antigen core.

5. Hepatitis E Virus: [1]Around 20 million people are infected with Hepatitis E virus globally, as reported by the WHO. Leading to 3.3million acute symptoms of hepatitis.[8] It is a small, non-enveloped viral RNA, and transmission occurs via the fecal-oral route. It is predominant in Africa, Asia, and Central America.

Pathogenesis: Hepatitis E Virus is transmitted through contaminated food or water. It enters the bloodstream through the intestinal mucosa and causes liver damage via immune-mediated mechanisms rather than cytopathic mechanisms. Infected hepatocytes present viral antigens that cause inflammation, hepatocellular injury and cholestasis.

Viral Structure: Small, circular, non-enveloped, positive-sense single-stranded RNA virus belonging to the *Hepeviridae family*. The genome organization is the 5’ prime head 3’tail, making it a cellular RNA virus. The capsid is icosahedral and 27–34 nm in diameter. There are three main open reading frames (ORFs): ORF1 encodes a large non-structural polyprotein with enzymatic domains required for replication., ORF 2 encodes the capsid protein that forms the outermost shell, and ORF3 encodes a small phosphoprotein involved in virion egress and modulation of host cell pathways.

II. Toxic Hepatitis

As the name suggests, liver inflammation is caused by exposure to toxins, drugs, and other toxic substances. [9]. Although the liver has a strong regenerative potential, exposure to drugs and toxins can cause irreversible harm; many drugs, especially neuropsychiatric drugs, have hepatotoxic effects. According to the author, administering antipsychotic drugs across all generations has an impact on somatic cells and causes physical disturbance; hence, it is extremely important to recognize the risks of prescribing them to psychotic patients on a long-term basis. As seen in the case report in August 2016, a patient suffering from schizophrenia complained of pain in the right rib cage; during that time, the patient was on antipsychotic drugs such as Quetiapine and Fluphenazine. Laboratory tests revealed increased liver enzymes like AST551U/L,ALT112IU/L, and GGT IU/L, while bilirubin levels remained constant. As a result, a hepatoprotective diet was prescribed, 200 mg of quetiapine was gradually discontinued, 2.5 mg of fluphenazine was discontinued, and 2 mg of clonazepam was introduced. Along with this, sulpiride 200 mg, clozapine 25 mg, and diazepam 5 mg were added, but the patient's condition deteriorated; therefore, the dose was changed to 50 mg clozapine and 20 mg diazepam, and the patient improved. Hence, it is essential to analyze the appropriate medicines for each patient. Based on the case report it is essential to have regular lab monitoring in order to prevent toxic liver damage due to administration of drugs risk factors should be notes which includes eating habits, harmful alcohol use, obesity, preferring monotherapy over combination therapy reduces the chance of toxic hepatitis.[10] Phytomedicine consumption can lead to toxic hepatitis. Dietary supplements are popular among people because they are believed to improve their well-being. Phytomedicine describes aloe as a herb with anti-inflammatory, antiproliferative, and anti-aging effects. In recent years, several cases of aloe-induced hepatotoxicity have been reported; however, the pharmacokinetics of Aloe vera have not been reported frequently. Three middle-aged women (57Y, 62Y, and 55Y) were admitted to the hospital after being diagnosed with acute hepatitis B. Various laboratory tests have concluded that oral consumption of Aloe vera, even for a few months, leads to hepatitis development. Upon discontinuation of the treatment with Aloe vera, all three patients recovered from acute hepatitis. The first patient 57 a old woman, consumed aloe vera supplements for 6 months and experienced dyspepsia for 2 months. She had no medical problems and did not consume alcohol. Laboratory testing revealed positive anti-HBs IgG results. Liver biopsy revealed moderate portal infiltration by eosinophils, neutrophils, and monocytes in the liver. Inflammatory cell infiltration and acidophilic bodies were observed in the hepatic lobule. The second patient 62 y woman, presented to the hospital with a week history of fatigue. She did not consume alcohol and was not on any medication. She had been consuming aloe vera powder for 3 months. Laboratory tests revealed Liver biopsy revealed severe portal and lobular infiltrates consisting of neutrophils and monocytes. Several acidophilic bodies and ballooning cell changes were observed in the hepatic lobule. Bile stasis and bile-stained Kupffer cells were observed, and when hepatitis recurred after re-challenge with the aloe extract, it was confirmed that the diagnosis was aloe-induced toxic hepatitis. When aloe vera consumption was stopped, the patient recovered from the hepatitis. The third patient was a 55Y old woman with epigastric dysfunction for three months. She had been consuming Aloe vera extract for five months. Physical examination was normal, but the epigastric region showed signs of tiredness. Laboratory reports revealed negative autoimmune markers and increased liver echogenicity. She recovered from hepatitis after discontinuing the consumption of Aloe vera extract. Hence, we can conclude that although Aloe vera is natural and considered safe as a dietary supplement, it should be consumed cautiously.

Exposure to organic solvents used in many industries, such as painting, metal cleaning, dry cleaning, and

healthcare, has a huge impact on liver health, the main organ responsible for chemical metabolism [11]. Many painters, chemical workers, and healthcare workers are highly affected as they are frequently exposed to hepatotoxic chemicals such as dimethylformamide, dimethylacetamide, trichloroethylene, tetrachloroethylene, carbon tetrachloride, xylene, toluene, and chloroform through inhalation, skin contact, and oral ingestion. The liver is the main organ for xenobiotic metabolism, and toxic metabolites produced during biotransformation are central to liver function. Occupational toxic hepatitis can be classified as hepatocellular, cholestatic, or mixed, among which hepatocellular toxicity is particularly dangerous. At the mechanistic level, this review focuses on four interconnected pathways: inflammation mediated by non-parenchymal liver cells, dysfunction of cytochrome P450 systems involved in xenobiotic metabolism, mitochondrial dysfunction, and oxidative stress leading to liver cell death via necrosis or apoptosis.

Oxidative stress leads to the development of toxic hepatitis when Reactive Oxygen Species cause liver cell damage and lead to disease progression [12]. Oxidative stress arises from an imbalance between reactive oxygen species (ROS) and antioxidant defenses in the liver. Hepatotoxic agents, such as drugs and chemicals, induce the production of Reactive Oxygen Species (ROS) and liver antioxidant defenses. ROS are primarily generated by metabolic activation via liver enzymes, such as the cytochrome P450 system. This surplus ROS initiates lipid peroxidation, damaging hepatocyte membranes and disrupting the cellular integrity. Zinc plays a major role as an antioxidant, stabilizing cellular membranes and inhibiting the production of Reactive Oxygen Species, making it a suitable option for therapy against toxic hepatitis due to oxidative stress.

III. Autoimmune Hepatitis

[13] Autoimmune hepatitis(AIH) is an immune-mediated liver disease in which the cells within our body attack the liver cells affecting all ages, genders and ethnicity.AIH is more common in females , certain types of haplotypes impacting AIH are A1-B8-DR3, DR4 mostly 25-34% are asymptomatic at diagnosis but are untreated , symtomsatic patients are often present with fatigue, jaundice and show signs of chronic liver disease.

Symptoms include fatigue, jaundice, and chronic liver disease. It can sometimes overlap with other liver diseases, such as primary biliary cholangitis (PBC), primary sclerosing cholangitis (PSC), drug-induced liver injury (DILI), and viral hepatitis, and can occur after liver transplantation. The common symptoms include fatigue, jaundice, and signs of chronic liver disease. AIH can occur acutely or chronically, with an increased risk of cirrhosis and hepatocellular carcinoma. AIH can overlap with Primary Biliary Cholangitis (PBC), Primary Sclerosing Cholangitis(PSC), drug-induced liver injury, and viral hepatitis. [14] Autoimmune diseases are chronic diseases with many triggers, including genetic predisposition, environmental triggers, and immune system dysregulation. Certain human leukocyte antigens (HLA), such as HLA-DR3 and HLA-DR4, influence antigen presentation and immune response, increasing the risk of liver cell damage. Environmental factors, such as drugs, toxins, and other chemicals, trigger an autoimmune response against liver antigens, causing hepatitis, especially in genetically predisposed individuals. The presence of autoantibodies,antinuclear antibodies (ANA), smooth muscle antibodies (SMA), and anti-liver kidney microsomal type 1 (anti-LKM1) antibodies causes liver inflammation. [15] AIH presents a wide range of clinical spectrum from asymptomatic enzyme elevation to acute liver failure. Diagnosis involves a combination of clinical, serological, and histological findings. Liver biopsy remains essential, especially for biochemical markers, which can be normal in cirrhosis or acute presentations of the disease. Common treatment options include a combination of steroids, such as prednisolone for induction and azathioprine

or mycophenolate mofetil (MMF) for maintenance. MMF shows better tolerance than azathioprine but requires pregnancy considerations because of its teratogenicity. The management of acute AIH involves administering high doses of steroids and rapid assessment for liver transplantation in patients using scores such as SURFASA and MELD. However, there is a strong need to find an alternative to steroid therapy because of its side effects. Future areas of treatment investigation include precision genetic therapy, Janus kinase (JAK) inhibitors, and immunotherapy.

IV. Alcoholic Hepatitis

[16] Alcohol-associated liver disease (ALD) is a significant global health burden, accounting for 5.9% of deaths worldwide. Major liver disorders include fatty liver alterations, fibrosis, and cirrhosis, which are driven by abnormal alcoholic metabolism, inflammation, oxidative stress, and disrupted liver metabolism. Some major risk factors include obesity, metabolic syndrome, and hepatitis. Smoking and alcohol consumption also lead to liver disorders, particularly in women. Even small amounts of alcohol consumption can lead to liver disease. Men who consume an average of 60-80 g of alcohol daily for over 10 years are prone to ALD, whereas women exhibit liver damage even with as low a consumption of 20–40 g. The early stages of ALD do not show any symptoms; therefore, diagnosis before liver damage is challenging. ALD has four stages: alcohol-induced fatty liver, alcoholic hepatitis, alcoholic steatosis, and cirrhosis. Corticosteroids are the preferred medications; however, they are not effective for ALD with 6 months mortality. [17] Steatosis can occur in 90% of patients with significant alcohol consumption (> 60 g/day for decades) and > 120–180 g/day, who are categorized as heavy drinkers. The diagnostic approach includes recognising alcohol use disorders, confirming diagnosis with validated criteria or liver biopsy and using the Model for End-Stage Liver Disease.

Corticosteroids are commonly used for the initial treatment of patients with severe AAH, and patients who do not improve with steroid treatment are recommended for liver transplantation at the earliest.

V. Diagnosis of Hepatitis

[18] Diagnosis and accurate treatment can eliminate chronic hepatitis infections by 2030. Chronic HBV and HCV infections are mostly prevalent in low- and middle-income countries. Many infections are acquired perinatally, in childhood, and in adulthood due to drug use and injection. Current diagnostic methods involve the use of serological assays for Hepatitis B virus (HBV) for HBsAg to detect whether it is Chronic or Acute Hepatitis. The primary screening test is performed to evaluate and identify whether further tests and assessments are required. Common methods include laboratory immune assays, such as ELISA/EIA or CLIA, and rapid diagnostic tests that use finger sticks or venous blood to identify the disease and determine whether further assessment is required. Similarly, HCV serological testing is performed to detect viremia and distinguish current from past infections in addition to primary screening tests. Virological tests are conducted on patients with active infections, including HBV DNA Testing(Viral Loading), which detects the presence and quantity of circular viruses. Apart from this, HBeAg and anti-HBe detect the level of replication similar to HCV, where the HCV RNS NAT test is performed to detect current active HCV infection after a patient shows a positive anti-HCV infection.

VI. Treatment of Hepatitis

[19] While most acute infections resolve on their own for viral hepatitis, chronic conditions, especially Hepatitis

B and C, if left untreated, will lead to cirrhosis and liver cancer. For Hepatitis A and E, hydration, rest, and supportive care are sufficient; the illness is usually self-limited and managed through vaccination. In Hepatitis E, self-care is sufficient in severe conditions of chronic disease, especially in immunocompromised patients, and ribavirin is recommended. The treatment for Hepatitis B virus aims to prevent viral replication, cirrhosis, and liver cancer. Some of the commonly used drugs are pegylated interferon-α finite course, which induces sustained response among limited people with certain side effects. Another drug is a nucleoside widely used for long-term oral therapy, which strongly suppresses HBV DNA and improves outcomes but rarely leads to a complete cure. For Hepatitis C, direct-acting antivirals for 12 weeks lead to a cure, as seen in >95% of patients.. In 1991, the FDA approved the use of interferon alpha for HCV, but the success rate was as low as 16%. As a result, ribavirin was added in the 1990s along with the interferons, slightly increasing the efficacy, but this also showed side effects. Pegylated IFN combined with ribavirin showed 41% efficacy but still had adverse side effects. Another approach is combination therapy, where antiviral combinations such as sofosbuvir/ledipasvir, ombitasvir paritaprevir/ritonavir, dasabuvir, and grazoprevir/elbasvir achieved sustained viral response (SVR) rates globally above 95% in all genotypes and around 85% in patients with advanced cirrhosis. No treatment is required for acute hepatitis D. The only approved drug for hepatitis is IFN-α which gives modest responses among 23-57% of patients.[20] For chronic hepatitis B include Interferon and Nucleotide analogs, which suppress viral replication and reduce the risk of cirrhosis and hepatocellular carcinoma. Novel aaantiviral and immunomodulatory agents are under clinical trails which can be better alternative.[21]Autoimmune hepatitis is treated with drugs such as prednisolone (30 mg/day) and azathioprine (1 mg/day). Patients intolerant to azathioprine are prescribed prednisolone. Budesonide was used in patients who were intolerant to Prednisolone and Azathioprine. Mycophenolate mofetil is an alternative immunosuppressant used for the treatment of autoimmune hepatitis.[21] Alcohol-associated hepatitis(AH) is the most severe form of hepatitis, with a 90 day mortality. The major 4 ways to treat AH include completely avoiding alcohol, habituating the consumption of nutritional food, careful prevention of infections, and management and control of alcohol avoidance. Common treatment methods include the administration of liver regeneration stimulants, such as obeticholic acid, introduction of anti-inflammatory and antioxidant strategies, such as N-acetylcysteine, immune and microbiome-targeted therapies, and liver transplantation, which is emphasized as a life-saving option and is no longer viewed as absolutely contraindicated by recent alcohol use. [22] For drug-induced hepatitis, novel therapies are repurposed therapeutic agents, as well as supportive and adjunctive interventions, such as N-acetylcysteine (NAC), a globally approved antidote effective in paracetamol overdose to prevent acute liver failure. Ursodeoxycholic acid is used empirically for treating cholestatic DILI drug-induced vanishing bile duct syndrome. Apart from this, a commonly used antidote is bicyclol, a synthetic hepatoprotective agent that scavenges free radicals, protects the mitochondria, and inhibits inflammatory cytokines. Current emerging trends include nanotechnology-based Drug Formulations proposed to optimize drug delivery and reduce hepatotoxicity.

VII. Transplantation for Hepatitis

[23] Liver transplantation is important for end-stage liver disease, and HBV/HCV are major causes of cirrhosis, hepatocellular carcinoma, and transplantation indications. Before the 1990s, HBV recurrence post-transplant was common, which led to rapid liver damage and fibrosing cholestatic hepatitis. High HBV DNA levels at the time of transplant are the key predictor of post-transplant recurrence; HBV recurrence is also linked to recurrent hepatocellular carcinoma, suggesting HBV can replicate in tumor cells from the introduction of hepatitis B

immunoglobulin (HBIG) and potent oral nucleos(t)ide analogs outcomes have changed: recurrence is much lower.[24] Liver transplantation has evolved over the past decade, noting major shifts in both candidates and donors as well as in allocation policy it uses MELD(Model For End Stasge Liver Disease) a decline in viral hepatitis as an indication for transplant, and a rise in metabolic liver diseases and cancer-related indications. Transplant candidates and donors are now typically older and have more comorbidities, such as metabolic syndrome, along with greater disease severity and sarcopenia/frailty. [25] For organ transplantation, the European Association for the Study of Liver (EASL) has set certain guidelines, a comprehensive update to the 2016 guidelines, focusing on guidelines for liver transplantation. Some of the major criteria and guidelines are as follows

- ✓ Candidate selection and Inclusion: Upper age limit has been relaxed , and the guidelines focus on the criteria of selection of factors such as cardiopulmonary status, frailty, sarcopenia, nutritional status , and certain traditional factors. The guidelines are suitable for patients with acute to chronic liver disease (ACLF) grade 3 and severe alcohol-related hepatitis.

- ✓ Donor Pool Expansion: There has been an increased donation of organs after a cardiac death (DCD) donors and HCV-infected grafts which can be transplanted into HCV negative recipients

- ✓ Equity and Access: New prediction models have been proposed to improve equal access to liver transplantation.

- ✓ Post-transplant Management: The guidelines focus on the recurrence of disease post-transplant, autoimmune liver disease, and metabolic-related fatty liver diseases.

VIII. Precision medicine in Hepatitis

[26]Viral hepatitis is a pressing issue, and lack of diagnosis can lead to cirrhosis and liver failure. Sometimes, the common methods used may not be ideal for all patients; hence, precision methods are under research, and their application is being studied even for hepatitis virus. Precision medicine can fill the gap in various aspects, such as the pathogenesis uncertainty, where the precise mechanism through which viral hepatitis develops and progresses remains unknown, the specific immune response of the host to viral hepatitis, and how the response between each patient remains unknown. It is essential to provide precise medicine and therapy for better results, as understanding the host-pathogen interaction plays a major role in the development of precision medicine. By studying the viral pathogen interaction with host factors, one can identify biomarkers that distinguish between patients and predict clinical outcomes. The identified biomarkers enable more accurate and early diagnosis, which is more convenient than conventional serological and viral testing practices. Another approach that is under study is the integration of multiple approaches, such as molecular approaches, including advanced molecular profiling techniques that characterize the viral genetic variants, host genetic variants, and expression patterns. In addition, the use of bioinformatic tools helps in extracting meaningful clinical insights from complex processes and multidimensional biological data. [27] Other precision medicine approaches include gene therapy, where RNA interference (RNAi) using small interfering RNA (siRNA) offers a promising strategy for treating chronic HBV by targeting HBV mRNA. Therapeutic vaccines such as ABX-203 (HeberNasvac), which contains HBsAg and HBcAg, have shown promise in clinical trials, achieving better viral load reduction and HBeAg seroconversion compared to Peg-IFNα. Another approach is next-generation sequencing NSG, which provides significantly enhanced sensitivity compared to Sanger Sequencing and plays a major role in detecting drug resistance mutations,

mixed genotypes, and viral quasi-species. Precision Pharmacogenomics explains how individual genetic variations influence drug response, focusing on pharmacokinetics and pharmacodynamics. Genome-wide association studies (GWAS), which identify single nucleotide polymorphisms (SNPs), influence liver disease development and progression, contributing to differences in immune responses.

IX. Artificial Intelligence in Hepatitis

[28] To overcome the challenges faced in traditional methods of diagnosis and treatment, it is essential for Artificial Intelligence (AI) to intervene at all stages. Deep learning and Machine Learning, which are applications of AI, help in improving the detection of liver cirrhosis, fibrosis, and HCC using ultrasound, CT, and MRI. Convolutional neural networks and decision tree models achieve high accuracy in classifying hepatitis status and staging the disease, often reducing the need for liver biopsies. AI models help in the development of personalized treatment by providing individual responses to antiviral therapy, risk of treatment failure, and hepatic encephalopathy. AI systems help in analysing real-time clinical and demographic data to forecast outbreaks, identify high-risk groups, guide vaccinations and screening programs, and support HCV and HBV control strategies.

X. Public Health awareness of Hepatitis

Public health awareness plays a major role in eradicating diseases. [29] Here a survey was conducted among the Indian population regarding awareness and vaccination rates for Hepatitis B where 40 million people suffer from chronic virus on 3540 individuals who are non healthcare workers (18y-60y) online the results concluded that the knowledge mean score was 6.89 out of 22 points , only 25% showed good knowledge , 44% showed poor information and 4% showed incorrect information , urban individuals had better knowledge and male respondents performed better. Only 22.7% had received all three doses of the HBV vaccine. Hence, it is essential to increase awareness about hepatitis. [30] Chronic Hepatitis is a public health issue worldwide where 2billion people are infected and 350 million people are chronic carriers HBV causes 600000-1200000 deaths annually. It is prevalent in Asia and Africa, where the disease is transmitted perinatally or in early childhood. The progression can be controlled by raising awareness about vaccines, diagnosis, addressing knowledge gaps, epidemiology, prevention, treatment, and implementation..

XI. Conclusion

Hepatitis is a disease that is prevalent even after advancements and awareness of the disease; hence, it is essential to focus on the development of precision medicine, develop better treatment options, and increase awareness among people.

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