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Original Research Article

Comparative Analysis of Individuals with Hepatitis B Suffering from Covid-19 for Symptomatology & Demographics: A Cross Sectional Study

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Abstract

Background: The global pandemic of the corona-virus infection in 2019 (COVID-19) has attracted attention from all across the world. Acute respiratory distress syndrome may develop in serious cases of the illness, which mostly affects the respiratory system and causes flu-like clinical features as fever, dry cough, and dyspnea.

Aims and Objectives: Comparative analysis of Covid-19 cases for symptomatology & demographics.

Material and Methods: The research was conducted in the Department of Biochemistry L.N. Medical College, Bhopal. 200 subjects who are Covid positive will be included in the research. Category-1 - 60 corona positive subjects who are Hepatitis B virus positive. Category-2 - 140 corona positive subjects.

Results: Out of 200 cases, 106 were males and 94 were females. When were compared for comorbidities then hypertension was most common overall but was more common in covid cases. All the individuals were suffering from one or the other clinical feature like fever, fatigue, myalgia, cough, dyspnoea, diarrhoea, headache & so on.

Conclusion: COVID-19's clinical features extend beyond the respiratory system. There have also been reports of multiple organ involvement, including the gastrointestinal, cardiovascular, and neurological systems. This multisystem illness may be caused by SARS-CoV-2 binding to the angiotensin-converting enzyme-2 (ACE2) receptors, which are widely distributed throughout.

Keywords: HBV, Covid, SARS CoV 2, Acute Respiratory Distress Syndrome.

Introduction

The global pandemic of the corona-virus infection in 2019 (COVID-19) has attracted attention from all across the world. Acute respiratory distress syndrome may develop in serious cases of the illness, which mostly affects the respiratory system and causes flu-like clinical features as fever, dry cough, and dyspnea [1]. The novel, extremely contagious

serious acute respiratory syndrome corona-virus (SARS-CoV-2) is the culprit responsible for COVID-19. Numerous studies have shown that individual with COVID-19 frequently experience hepatic damage [2]. There is a global spread of hepatitis-B. Hepatitis-B virus (HBV) is still the predominant cause of cirrhosis, hepatic infection, and hepato-cellular carcinoma at the moment.

Due to the fact that both viruses alter hepatic function, it is unclear how co-infection with HBV and SARS-CoV-2 would affect infection development and death [3]. If subjects with HBV infection have a greater risk of COVID-19. Over 40 million individual in India are HBV carriers, and hepatitis-B surface antigen (HBsAg) prevalence ranges from 3-4.2 percent [4]. Over 1,15,000 Indians pass away each year as a outcome of hepatitis-B-related illnesses.

In India, hepatitis-B, a condition that can be prevented through vaccination, affects a large number of individual, most of whom are unaware that they are infected [5]. Chronic hepatitis-B increases the chance of developing major hepatic conditions including cirrhosis (hepatic scarring) and carcinoma by progressively destroying the hepatic over a long period of time [6]. Second only to cigarettes as a cause of carcinoma in individual, hepatitis-B is the most common cause of hepatic carcinoma in the globe.

Even though everyone can have hepatitis-B, Asian and Pacific Islanders are more likely to develop it [7]. Individual with hepatitis-B have encountered more difficulties getting access to medical care and management during the COVID-19 pandemic in India because appointments have been delayed and routine hepatitis or primary care managements have been given lower priority [8]. Subjects not only had to get used to telemedicine but also had trouble acquiring antiviral refills and delays in mail-order deliveries. Others have experienced difficulty navigating particular medical information on COVID-19 for those with hepatic infection and have expressed concerns about the safety and effectiveness of COVID-19 vaccines for those with hepatitis-B and hepatic infection [9]. Community-based organisations (CBOs) that offer direct public health services like HBV education, screening, and immunisation found it particularly difficult.

Aims and Objectives

Comparative analysis of Covid-19 cases for symptomatology & demographics.

Material and Methods

The research was conducted in the Department of Biochemistry L.N. Medical College, Bhopal. 200 subjects who are Covid positive will be included in the research. Category-1 - 60 corona positive subjects who are Hepatitis B virus positive. Category-2 - 140 corona positive subjects.

Inclusion criteria: Corona positive willing participants, all age categories, Hepatitis-B positive individuals & Individuals with no physical sign & metabolic syndrome.

Exclusion Criteria: Unwilling participants, Individuals with physical sign & hepatic cirrhosis or any metabolic syndrome, Alcoholic consumption, cigarette smokers or drugs abusers & Hepatitis-B negative subjects.

Statistical Analysis

Data collected will be entered into Microsoft Excel Worksheet & statistically analyzed by using SPSS (Statistical package for social sciences) Version 20.

Results

Out of total 200 covid positive cases, 60 individuals were also HBV positive & rest were only having Covid positive status. Out of 200 cases, 106 were males and 94 were females. But when we specifically look for HBV positive cases then it shows male

preponderance with 41 male cases & 19 female cases.

Table 1: Gender wise distribution of overall cases

Gender	HBV+Covid	Covid	Total	p - value
Male	41	53	94	<0.051
Female	19	87	106	
Total	60	140	200	

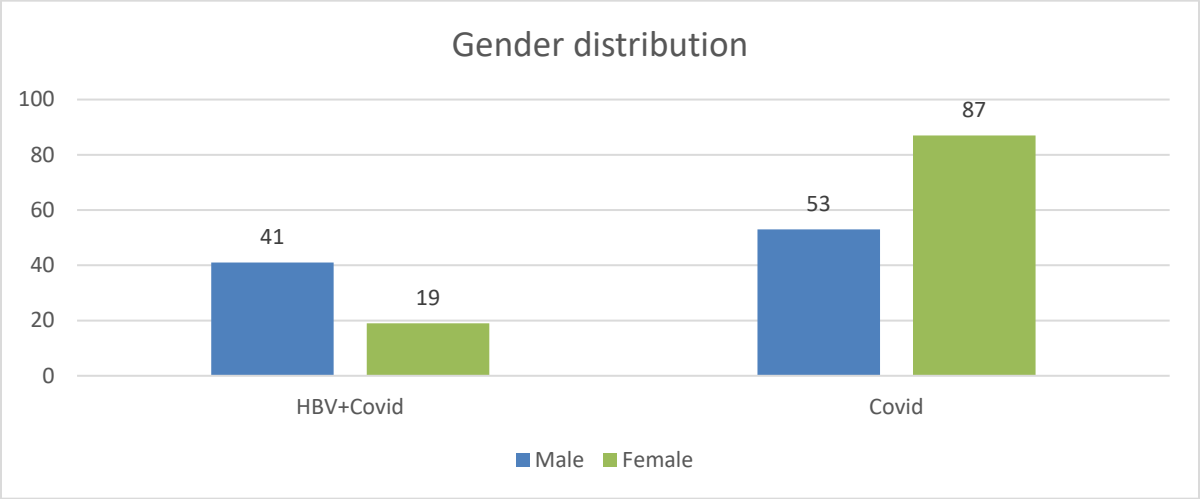

Figure 1

When we look for the average age of the cases, it is 54.6 years for covid cases suffering from HBV infection & it is 52.7 years for individuals who are only covid positive. With standard deviation for both in the range of 7.8 to 8.4.

Table 2: Age wise distribution of overall cases

Age	HBV+Covid	Covid	p - value
Mean	54.6	52.7	0.12
SD	8.4	7.8	

Table 3: Comorbidities wise distribution of overall cases

Comorbidities	HBV+Covid	%	Covid	%	p - value
Hypertension	4	6.7	25	18	0.03
CVD	0	0	12	9	0.019
Diabetes	4	6.7	12	9	0.64
Malignancy	12	20	4	3	<0.01
COPD	0	0	11	8	0.02
Liver Cirhosis	8	13.3	2	1	<0.01

When were compared for comorbidities then hypertension was most common overall but was more common in covid cases. Next was malignancy, which was most common in individuals having HBV infection. Also liver cirrhosis was more common in long standing HBV positive cases. Whereas diabetes & COPD was more common in individuals who were only covid positive.

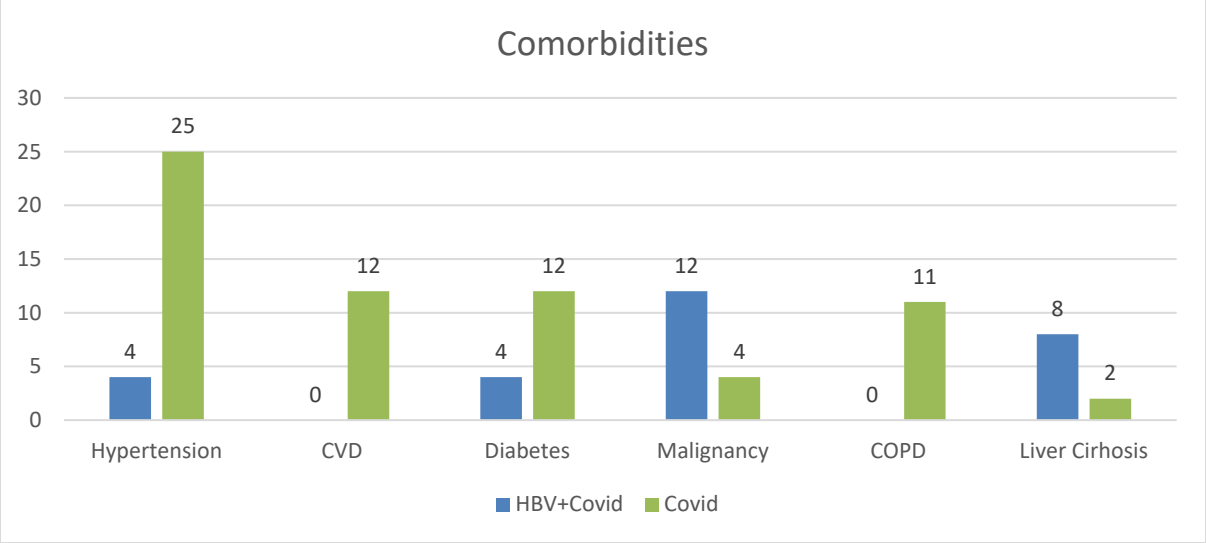

Figure 2

Table 4: Symptoms wise distribution of overall cases

Symptoms	HBV+Covid	%	Covid	%	p - value
Fever	32	53.3	112	80	<0.01
Fatigue	32	53.3	80	57	0.61
Myalgia	12	20	48	34	0.04
Cough	17	28.3	75	54	<0.01
Dyspnea	25	41.7	29	21	<0.01
Diarrhoea	9	15	24	17	0.71
Headache	9	15	25	18	0.62

All the individuals were suffering from one or the other clinical feature like fever, fatigue, myalgia, cough, dyspnoea, diarrhoea, headache & so on but when we looked for specific symptom then we found out that there is no relationship or effect of HBV positive status on the clinical symptoms on the individuals.

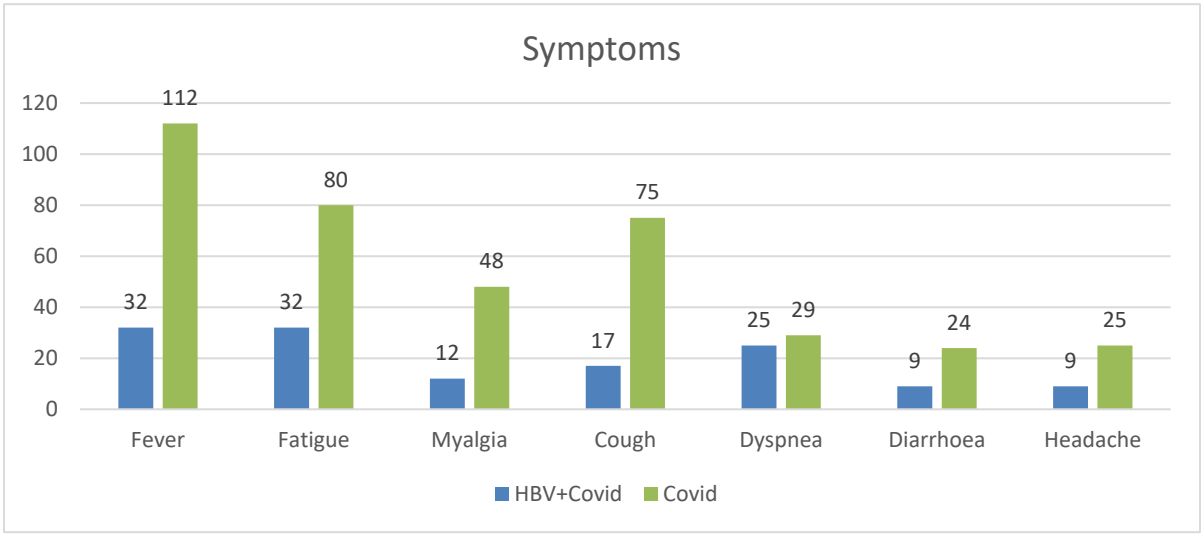

Figure 3

Table 5: Duration of hospital stay

Duration of hospital stay	HBV+Covid	Covid	P - value
Mean	13.8	13.9	0.75
SD	1.01	2.35	

Also when we compared for the duration of hospital stay in both the groups and found not much significant difference. This is in contrast to other studies where duration of hospital stay was more in HBV positive Covid cases.

Discussion

Numerous studies have assessed the frequency of raised hepatic chemistries in COVID-19 individuals. The following key findings were identified after a thorough analysis of 107 research from different nations:

a. COVID-19 has a pooled prevalence of underlying CLD of 3.6 percent;
b. Subjects with severe infections had a pooled prevalence of CLD of 3.9 percent.
c. Subjects with CLD have a 0.81-fold raised risk of developing severe COVID-19 compared to non-CLD subjects for the following reasons:
d. 23.1 percent of COVID-19 subjects have raised hepatic chemistries at initial presentation;
e. 24.4 percent of subjects have raised hepatic chemistries and severe hepatic injury during the illness; and
f. Subjects with raised hepatic chemistries have a higher risk of developing severe COVID-19.
g. Hypoalbuminemia is a sign of a serious infection.
h. There were 25.4 percent cases of DILI.
i. Remdesivir, lopinavir/ritonavir, and arbidol-related DILI is frequent but not life-threatening.

Additionally, they contrasted our findings with those of earlier meta-analyses. Study to document hepatic involvement in all COVID-19 subjects, including adults, pregnant subjects, and paediatric subjects [10]. We have thoroughly examined the hepatic's role in COVID-19, including the influence of high hepatic chemistries at the time of the illness's onset and on the patient's prognosis.

Additionally, we've mentioned any increases in AST, ALT, bilirubin, albumin, ALP, GGT, and PT prolongation for each variable of the hepatic function tests. From the data at hand, we also documented the prevalence of DILI and the medicines that were involved. Raised hepatic chemistries at the time of initial presentation or while unwell are a significant indicator of the severity of the disease [11].

A negative acute phase reactant called serum albumin also denotes serious illness. When compared to infections without COVID-19, hepatic damage occurs more frequently. According to hepatic biopsy results in COVID-19, there is mild lobular and portal activity as well as moderate micro-vesicular steatosis. Through the angiotensin-converting enzyme 2 (ACE2) receptor, SARS-CoV-2 penetrates cells [12]. Alveolar cells, bile duct epithelial cells, and hepatocytes all contain 128 ACE2 receptors.

Intriguingly, we found in our review that COVID-19 had hyper-bilirubinaemia, raised aminotransferases, and a considerable elevation in ALP and GGT, all of which pointed to direct or indirect hepatic impairment. While the virus was not found in three COVID-19 subjects' hepatic biopsies despite PCR and hepatic immunohistochemistry attempts, suggesting other possible pathways for hepatic harm. The cytokine storm, which causes an increase in inflammatory cytokines and damages the hepatic, is the second mechanism for hepatic damage [13].

The acute respiratory distress syndrome (ARDS) and systemic inflammatory response syndrome caused by SARS-CoV-2 can result in hepatic ischaemia and hypoxia-reperfusion

damage by causing hypoxia and shock. Direct endothelial cell infection by SARS-CoV-2 can cause extensive endotheliitis. Three SARS-CoV-2-infected subjects' post-mortem histological analysis revealed hepatocyte necrosis as well as lymphocytic endotheliitis in the lung, heart, kidney, and hepatic. Additional electron imaging of one of the transplanted kidney subjects' endothelial cells revealed viral inclusion formations. The authors postulated that extensive endothelial dysfunction associated with apoptosis may come from the recruitment of immune cells, whether by immune-mediated or direct viral infection of the endothelium [14]. Similarly, a post-mortem hepatic biopsy of 48 severely ill COVID-19 subjects revealed substantial vascular thrombosis, steatosis, lobular inflammation, and portal fibrosis.

Finally, taking medicines themselves may harm your hepatic. Due to the lack of any effective managements, the majority of subjects typically take multiple medications (polypharmacy). In addition to paracetamol, medications with hepatotoxic potential such as remdesivir, lopinavir, ritonavir, oseltamivir, umifenovir, and hydroxychloroquine may worsen hepatic damage in COVID-19 subjects [15]. Careful screening of novel managements, particularly in subjects with pre-existing high hepatic chemistries, is required in the quest for newer medications for the management of SARS-CoV-2, several of which have now been demonstrated to be hepatotoxic [16-18]. CLD is not common among COVID-19 subjects. Despite the limited and skewed data, subjects who contracted COVID-19 had a decreased risk of having a severe case. It is interesting how the ACE2 receptor, SARS-CoV-2, and angiotensin II (Ang II) interact. The SARS-CoV-2 is more easily able to enter type 2 pneumocytes thanks to the ACE2 receptor. After the viral combination is endocytosed, ACE2 is downregulated, which causes angiotensin II to build up unchecked and increase systemic problems. Subjects with COVID-19 appeared to have higher plasma Ang II levels in a recent small research, and these levels were linked to both the overall viral load and the severity of lung damage. It has been demonstrated that recombinant ACE2 infusions can lessen disease severity by reducing Ang II levels and raising ACE2 levels in respiratory viral infections [19,20].

Conclusion

COVID-19's clinical features extend beyond the respiratory system. There have also been reports of multiple organ involvement, including the gastrointestinal, cardiovascular, and neurological systems. This multisystem illness may be caused by SARS-CoV-2 binding to the angiotensin-converting enzyme-2 (ACE2) receptors, which are widely distributed throughout. Individuals with hepatic problems should be taken into consideration as a potential priority population for receiving the vaccination in the future as an outcome of preliminary findings showing raised COVID-19 mortality in these subjects. Researchers should be prepared to examine the effectiveness and safety of the vaccinations in both clinical trials and real-world registry data from individuals with hepatic infection.

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