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ORIGINAL ARTICLE

To determine Hepatitis A virus infection among hospitalized patients

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ABSTRACT
Background: Hepatitis A virus (HAV) infection is one of the most common causes of acute hepatitis worldwide. The present study was conducted to determine Hepatitis A virus infection among hospitalized patients. **Materials & Methods:** The present study was conducted on 126 patients of viral Hepatitis A infection of both genders. A thorough clinical examination was done in all patients. Clinical features were recorded. **Results:** Out of 126 patients, males were 86 and females were 40. Common clinical features in patient was nausea/ vomiting in 104, jaundice in 87 and abdominal pain in 110 patients. The mean AST level in patients was 3765.4 IU/L, AY was 2672.8 IU/L, ALP was 251.9 IU/L, GGT was 280.1 IU/L, total Bilirubin was 19.2 mg/dl and serum creatinine was 2.3 mg/dl. **Conclusion:** Authors found that common clinical features in patient were nausea/vomiting, jaundice and abdominal pain.
Key words: Abdominal, Jaundice, Hepatitis A virus

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Introduction

Hepatitis A virus (HAV) infection is one of the most common causes of acute hepatitis worldwide. Approximately 1.5 million clinical cases occur worldwide annually, but the rate of infection is probably 10 times higher. Hepatitis A virus is a positive-sense, single-stranded RNA virus classified within the genus Hepatovirus of the family Picornaviridae. The chief mode of transmission for HAV is through the faecaloral route, including person-to-person spread, and contaminated water or food products, but it has also been associated with outbreaks in injecting drug users and men who have sex with men.[1]

Hepatitis A is an ancient disease that has likely afflicted mankind since humans first began to live in groups large enough to sustain transmission of the causative agent, hepatitis A virus (HAV). In reviewing what was known as 'catarrhal jaundice' in 1912, Cockayne[2] noted descriptions of epidemic jaundice extending back to antiquity. The infectious nature of the disease was proven several decades later in deliberate human transmission studies. Such experiments led to a clear distinction between hepatitis A ('infectious hepatitis') and hepatitis B ('homologous serum jaundice') and recognition of the lack of cross immunity between these two forms of transmissible hepatitis by as early as 1945.[3]

The mechanisms underlying synthesis of HAV RNA have not been intensively investigated, but are thought to be similar to those of other, well studied picornaviruses. RNA replication proceeds slowly, and like all positive-strand RNA viruses, within the cytoplasm and in close association with membranes.[4]

The present study was conducted to determine Hepatitis A viral infection among hospitalized patients.

Materials & Methods

The present study was conducted in the department of Internal Medicine. It comprised of 126 patients of viral Hepatitis A infection of both genders. All were informed regarding the study and written consent was obtained. Ethical approval was obtained from institute prior to the study.

General information such as name, age, gender etc. was recorded. A thorough clinical examination was done in all patients. Clinical features were recorded. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

Results

Table I Distribution of patients

Total- 126		
Gender	**Males**	**Females**
Number	86	40

Table I, graph I shows that out of 126 patients, males were 86 and females were 40.

Graph I Distribution of patients

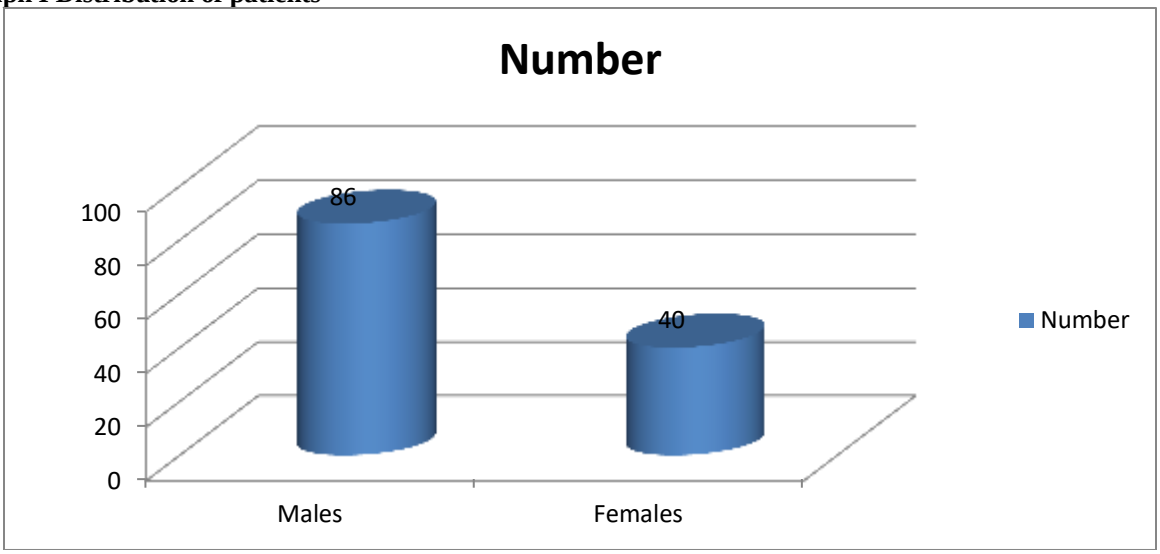


Table II Clinical features in patients

Clinical features	Number	P value
Nausea/Vomiting	104	0.84
Jaundice	87	
Abdominal pain	110	

Table II, graph II shows that common clinical features in patient was nausea/ vomiting in 104, jaundice in 87 and abdominal pain in 110 patients.

Graph II Clinical features in patients

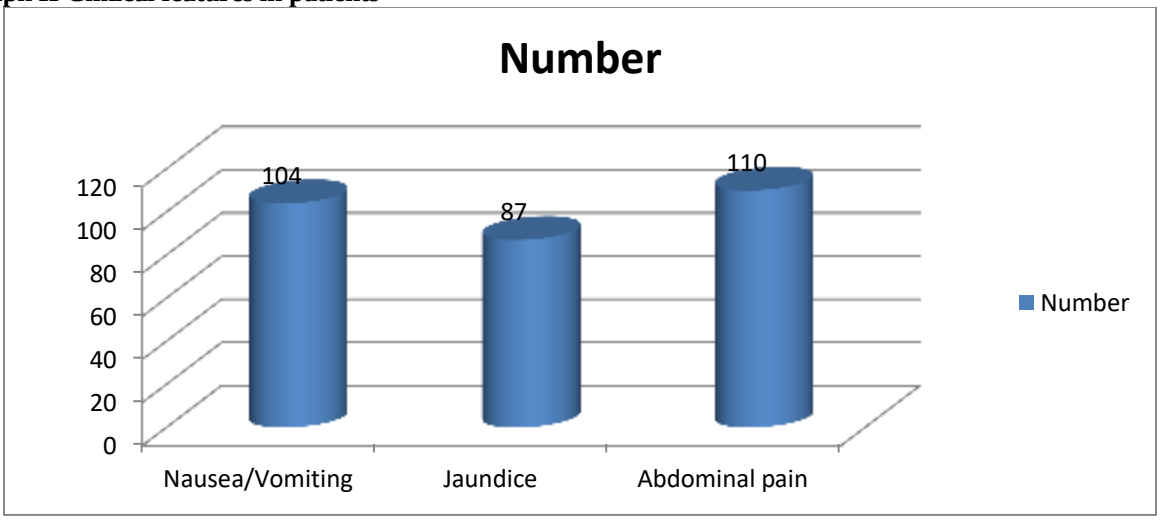
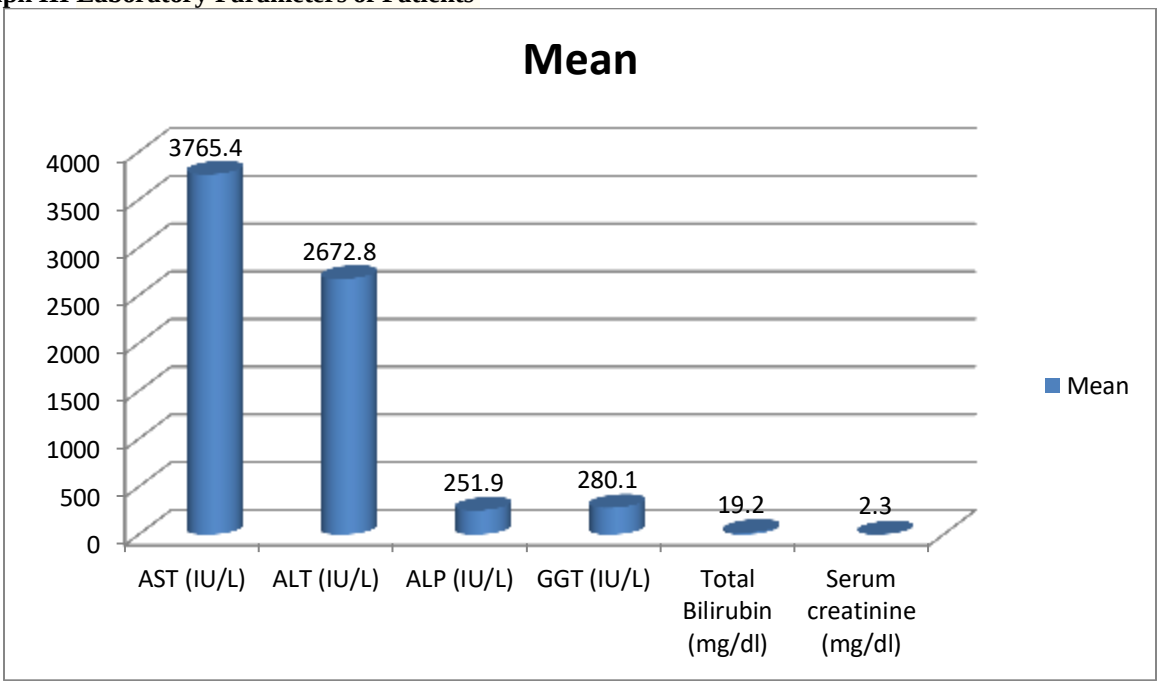


Table III Laboratory Parameters of Patients

Laboratory Parameters	Mean
AST (IU/L)	3765.4
ALT (IU/L)	2672.8
ALP (IU/L)	251.9
GGT (IU/L)	280.1
Total Bilirubin (mg/dl)	19.2
Serum creatinine (mg/dl)	2.3

Table III, graph III shows that mean AST level in patients was 3765.4 IU/L, AY was 2672.8 IU/L, ALP was 251.9 IU/L, GGT was 280.1 IU/L, total Bilirubin was 19.2 mg/dl and serum creatinine was 2.3 mg/dl.

Graph III Laboratory Parameters of Patients



Discussion

Hepatitis A is caused by infection with hepatitis A virus (HAV), a non-enveloped RNA virus that is classified as a picornavirus. HAV was first identified by immune electron microscopy in 1973 and initially replicated in mammalian cell culture in 1979.[5] Humans are the only natural host, although several nonhuman primate species have been infected in laboratory settings. Depending on conditions, HAV can be stable in the environment for months. The virus is relatively stable at low pH levels and freezing to moderate temperatures, but can be inactivated by high temperature (185°F [85°C] or higher for one minute) or through disinfection of surfaces with a 1:100 dilution of sodium hypochlorite in water.[6]

Hepatitis A is typically acquired through fecal-oral transmission, either from direct person-to-person contact or consumption of contaminated food or water. HAV replicates in the liver, is excreted in bile, and is shed in the stool of infected people in high concentrations 2–3 weeks before and 1 week after onset of illness.[7] Peak infectivity occurs during the 2 weeks prior to onset of clinical signs and symptoms (jaundice or elevated liver enzymes). Most persons cease to be infectious 1 week after jaundice appears. Although virus is present in serum of an infected person, its concentration is several orders of magnitude less than in feces. Infected children and infants may excrete virus longer than adults.[8]

Janaszek et al[9] retrospectively analyzed 449 patients hospitalized for acute hepatitis and compared clinical outcomes based on the presence of HBsAg. Of the 449 patients, 30 patients were in the HBsAg-positive group and 419 in the HBsAg-negative group. The HBsAg-positive group was older than the HBsAg-negative group however, other baseline characteristics were similar between the 2 groups. Mean peak values of prothrombin time, serum total bilirubin, and serum creatinine at admission were significantly higher in the HBsAg-positive group. When comparing clinical

outcomes between the 2 groups, gastrointestinal bleeding, acute renal failure, and acute liver failure were more frequently observed in the HBsAg-positive group. In particular, the incidence of acute liver failure was approximately 9-fold higher in the HBsAg-positive group than in the HBsAg-negative group. Multivariate analysis showed that HBsAg and age were independent risk factors for the occurrence of acute liver failure.

Polz-Dacewicz et al[10] showed that higher prevalence of antibody to HAV was found in subjects older than 50 years (75.8%). In all patients, the clinical course of hepatitis A infection presented symptomatically with abrupt onset and jaundice. The main factor that influences the clinical course of primary HAV infection is the age of an infected patient. Non-immune travellers are at risk of contracting the disease during travels to countries of high or intermediate endemicity. Currently, with improvements in sanitary conditions, the risk of infection for non-immune travellers who visit high or medium-endemic areas has been reduced. In the past the risk of HAV infection in unvaccinated travellers was considered to be 3 per 1,000 individuals per month of travel.

Conclusion

Authors found that common clinical features in patient were nausea/vomiting, jaundice and abdominal pain.

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