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CLINICO-ETIOLOGICAL PROFILE OF ACUTE VIRAL HEPATITIS PATIENTS

General Medicine

Dr. Manoj Kumar M* Senior Resident, Department of Medicine, Government Medical College, Amritsar. *Corresponding Author

Dr. Aditi Sharma Senior Resident, Department of Transfusion Medicine, Government Medical College, Amritsar.

Dr Ajay Chhabra Professor, Department of Medicine, Government Medical College, Amritsar.

Dr Raman Sharma (Ex)Professor, Department of Medicine, Government Medical College, Amritsar.

Dr Kanwardeep Singh Professor, Department of Microbiology, Government Medical College, Amritsar

ABSTRACT

Background: Viral hepatitis refers to a primary infection of the liver by any of the heterogenous group of hepatitis virus types A, B, C, D and E. **Aims And Objectives:** To determine the clinical profile and the etiological profile of Hepatitis A, B, C, D and E as a cause of acute viral hepatitis. **Material And Methods:** The present study was conducted on 100 patients who attended outdoor department or were admitted in Guru Nanak Dev Hospital attached to Government Medical College Amritsar. All new patients aged more than 18yrs with clinical symptoms suggestive of acute hepatitis with raised levels of transaminases and positivity of serological markers for HAV, HBV, HCV, HDV and HEV were included in the study. **Results:** Most of the patients were from age group 21-40 years. In the present study most of the patients presented with symptoms such as fever(94%) followed by nausea and vomiting(85%) and signs such as icterus(51%), hepatomegaly(48%), pallor(24%) and hepatosplenomegaly(2%). In the present study most common risk factor from history was outside food intake(54%) followed by unsafe drinking water(41%), poor sanitization(32%), Drug abuse(12%), Surgery(4%), blood transfusion(1%) and Hemodialysis(1%). In the present study the most common form of hepatitis was hepatitis E(55%) followed by hepatitis A(27%), hepatitis B(8%), hepatitis C(7%) and mixed(3%). **Conclusion:** Viral hepatitis is an important heath care problem in India as it occurs epidemically and sporadically. It is very essential for health care professionals to be aware of all aspects of it so that it is detected and treated early.

KEYWORDS

acute viral hepatitis, clinic profile, etiological profile inflammation.

INTRODUCTION

Hepatitis is an inflammation of liver which results in damage to hepatocytes with subsequent cell death (necrosis), the condition can be self-limiting or can progress to fibrosis.[1] In general, hepatitis is classified as acute or chronic based on the duration of the inflammation and insult to the hepatic parenchyma.[2-4]

Acute hepatitis is a term used to describe a wide variety of conditions characterized by acute inflammation of the hepatic parenchyma or injury to hepatocytes resulting in elevated liver function indices.[2-4] Patients with acute viral hepatitis commonly present with symptoms such as fever, malaise, fatigue, loss of appetite, vomiting, diarrhea, and abdominal pain. Patients may also report yellowish discoloration of their sclera (icterus) and/or skin, dark-colored urine, and light-colored stools. Depending on the underlying etiology, physical exam findings can range from the presence of icterus to signs of acute encephalopathy, seizures, bleeding diathesis and hypotension.[5,6]

Almost all cases of acute viral hepatitis are caused by one of five viral agents: hepatitis A Virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), the HBV-associated delta agent or hepatitis D virus (HDV), and hepatitis E virus (HEV). All these human hepatitis viruses are RNA viruses, except for hepatitis B, which is a DNA virus but replicates like a retrovirus.

Acute viral hepatitis though a global problem, is more serious in nature in tropical and developing countries due to poor hygiene and sanitation and so we conducted a study on clinico-etiological profile of acute viral hepatitis patients.

MATERIALS AND METHODS

This study was conducted on 100 patients attending Outdoor department or admitted in Guru Nanak Dev Hospital attached to Government Medical College Amritsar after seeking permission from Institutional Ethics Committee and written informed consent obtained from the patients.

Inclusion Criteria:

All new patients aged more than 18yrs with clinical symptoms suggestive of acute hepatitis with raised levels of transaminases and positivity of serological markers for HAV, HBV, HCV, HEV included in the study. HDV tested in patients positive to HBV.

Exclusion Criteria:

- Patient not giving informed consent.
- Patients with non-viral cause of hepatitis including autoimmune, fatty liver, drug and toxic induced hepatits.

Serum samples collected from a total of 100 patients clinically diagnosed to have hepatitis and were screened for antibodies to HAV, HEV, HBV and HCV after obtaining informed consent. Associated LFT parameters also subjected to analysis. HDV was tested in all patients who were hepatitis-B positive.

Investigations performed were: Complete blood count, serum creatine liver function test, prothrombin time/INR Anti-Nuclear Antibody, serology marker for hepatotropic viruses (IgM for hepatitis A and E), (IgM-anti-HBc) for hepatitis B, IgM for hepatitis D in hepatitis B positive patients, Anti- HCV Antibody, USG-whole abdomen.

STATISTICALANALYSIS:

All relevant data were analysed using the statistical package for social science version 21.0 (SPSS, Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as count and percentage. Comparisons between groups were performed using Student's t test for continuous variables and the chi-square test for categorical variables.

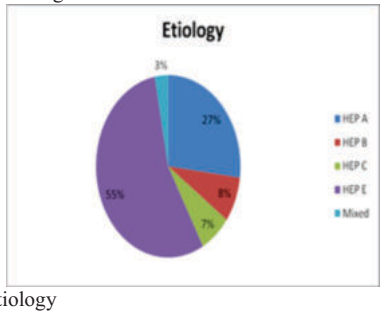

Figure 1: Etiology

OBSERVATIONS AND RESULTS

In the present study most of the patients are from age group 21-40 (60%) years followed by 41-60 years(23%), >60 years(10%) and 18-20 years(7%). In this study male predominance was seen with 53% males and 47% females. In our study 64% patients were from urban areas and 36% were from rural areas.

Table 1: Symptoms And Signs

Symptoms and signs	N	Percentage
Fever	94	94%
Jaundice	49	49%
Joint pain	14	14%
Abdominal pain	53	53%
Nausea and vomiting	85	85%
Diarrhea	7	7%
Anorexia	67	67%
Rash	1	1%
Pruritis	6	6%
Urine (dark)	51	51%
Clay Stool	4	4%
Black stool	1	1%
Bleeding manifestations	2	2%
Altered sensorium	6	6%
Pallor	24	24%
Icterus	51	51%
Lymphadenopathy	0	0%
Hepatomegaly	48	48%
Hepatosplenomegaly	2	2%

Table 2: Risk Factors

Risk factors		HEP A (27)	HEP B (8)	HEP C (7)	HEP E (55)	Mixed (3)	P value
Hemodialysis	Present	0	1	0	0	0	0.020
		.0%	12.5%	.0%	.0%	.0%	
Surgery	Yes	0	0	0	3	1	0.067
		.0%	.0%	.0%	5.5%	33.3%	
Blood transfusion	Yes	0	0	0	1	0	0.93
		.0%	.0%	.0%	1.8%	.0%	
Outside food intake	Often	21	3	1	27	2	0.016
		77.8%	37.5%	14.3%	49.1%	66.7%	
Drug abuse	Yes	0	5	4	0	3	0.00
		.0%	62.5%	57.1%	.0%	100.0%	
Poor Sanitization	Not proper	9	4	3	15	1	0.009
		28.1%	12.5%	9.4%	46.9%	3.1%	
Unsafe drinking water	No	13	5	3	19	1	0.536
		31.7%	12.2%	7.3%	46.3%	2.4%	

Table 3: Lab Parameters Among Study Participants

		HEP A	HEP B	HEP C	HEP E	Mixed	P Value
HEMOGLOBIN	<12	18	6	1	46	1	0.001
		66.7 %	75.0%	14.3%	83.6%	33.3%	
	≥12	9	2	6	9	2	
		33.3 %	25.0%	85.7%	16.4%	66.7%	
PLATELET COUNT	<1.5 lakh	6	0	2	12	0	0.514
		22.2 %	.0%	28.6%	21.8%	.0%	
	≥1.5 lakh	21	8	5	43	3	
		77.8 %	100.0 %	71.4%	78.2%	100.0%	
TLC	<4000	3	1	0	0	2	0.000
		11.1 %	12.5%	.0%	.0%	66.7%	
	≥4000	24	7	7	55	1	
		88.9 %	87.5%	100.0 %	100.0 %	33.3%	
SERUM BILIRUBIN TOTAL	>2.5	14	6	0	29	1	0.049
		51.9 %	75.0%	.0%	52.7%	33.3%	
	≤2.5	13	2	7	26	2	
		48.1 %	25.0%	100.0 %	47.3%	66.7%	
SERUM BILIRUBIN DIRECT	≥2	13	5	0	25	1	0.137
		48.1 %	62.5%	.0%	45.5%	33.3%	
	<2	14	3	7	30	2	
		51.9 %	37.5%	100.0 %	54.5%	66.7%	
SGOT	<400	8	4	7	22	1	0.019
		29.6 %	50.0%	100.0 %	40.0%	33.3%	
	≥400	19	4	0	33	2	
		70.4 %	50.0%	.0%	60.0%	66.7%	
SGPT	<400	7	4	7	20	1	0.009
		25.9 %	50.0%	100.0 %	36.4%	33.3%	
	≥400	20	4	0	35	2	
		74.1 %	50.0%	.0%	63.6%	66.7%	
INR	<1.1	5	3	6	24	1	0.059
		18.5 %	37.5%	85.7%	43.6%	33.3%	
	1.1-1.5	20	4	1	27	1	
		74.1 %	50.0%	14.3%	49.1%	33.3%	
	>1.5	2	1	0	4	1	
		7.4%	12.5%	.0%	7.3%	33.3%	
DSP	<3.5	9	2	1	19	3	0.119
		33.3 %	25.0%	14.3%	34.5%	100%	
	≥3.5	18	6	6	36	0	
		66.7 %	75.0%	85.7%	65.5%	0%	
SERUM CREATININE	<1.1	13	4	7	30	2	0.166
		48.1 %	50.0%	100.0 %	54.5%	66.7%	
	≥1.1	14	4	0	25	1	
		51.9 %	50.0%	.0%	45.5%	33.3%	

DISCUSSION

In the present study the most common form of hepatitis was hepatitis E(55%) followed by hepatitis A(27%), hepatitis B(8%), hepatitis C(7%) and mixed(3%). All the hepatitis B patients were screened for hepatitis D and found to be negative. The findings of our study are in concordance with the study done by Desai HD et al[10] who reported that among 70 acute viral hepatitis patients, majority were hepatitis E(70%), followed by hepatitis B(15.8%), hepatitis A(12.8%), and hepatitis C(1.4%). Similarly other study done by Dabadghao et al[7] found among 40 hepatitis cases, majority were hepatitis E(45%).

In the present study Hep A was seen predominantly in the age group 21-40 years (70.4%) followed by 41-60 years(18.5%) & 18-20 years(11.1%). Hepatitis B was seen only in the age group 21-40 years. Hepatitis C was predominant in the age group 21-40 years (57.1%) followed by 41-60 years(42.9%). Hepatitis E was predominant in the age group 21-40 years(50.9%) followed by 41-60 years(25.5%), >60 years(18.2%) & 18-20 years (5.5%). Mixed hepatitis was seen equally (33.3%) in the age groups 18-20 years, 21-40 years, 41-60 years but was not at all seen in >60 years age group. Difference in distribution among different age groups was not significant as p=0.06.

In the study done by Birajdar et al[8] out of 29 cases of hepatitis E majority of cases were 12 (41.3%) from 21-40 years of age. In the study done by Modi et al[11], the youngest patient was a child of 12 years age and the oldest patient was a 67-year-old male. Study done by Chakrabarti et al[9] also reported that the maximum number of hepatitis E patients in the study was clustered between the ages of 21-30 (33.3%).

In this study male predominance was seen with 53% males and 47% females. Hepatitis A was present more in males (51.9%) compared to females (48.1%). Hepatitis B was present more in males (75%) compared to females (25%). Hepatitis C was present more in males (85.7%) compared to females (14.3%). Hepatitis E was present more in females (56.4%) compared to males (43.6%). Mixed hepatitis was present only in males but not in females. Difference in distribution among different genders was significant as p=0.05.

The findings of our study are in concordance with the study done by Desai et al[10] who stated that males were affected slightly more than females. In the study done by Dabadghao et al[7] males were more affected compared to females (70%). Similar observation was also observed by Modi et al[11].

In case of hepatitis E most common symptom was fever (90.9%) followed by nausea and vomiting (87.2%), anorexia (76.4%), abdominal pain (52.7%) and jaundice (50.9%). 3 (5.5%) patients had hepatic encephalopathy indicative of acute liver failure with one patient had bleeding manifestation. In case of mixed infections fever was predominant symptom with a hepatitis B and C patient going in for hepatic encephalopathy. A study done by Shah et al[12], most common symptoms observed were jaundice (86.10%) followed by anorexia (76.50%), dark colored urine (73%), fever with chills (66.1%), and abdominal pain in (36.3%). Another study done by Zhang et al[13] also observed that the common clinical symptoms were jaundice, fatigue, and anorexia. In our study, the most common presenting symptoms was yellowish discoloration of urine (84.2%) followed by yellowish discoloration of sclera (81.2%), anorexia (65.7%), nausea and vomiting (40%), abdominal pain (38%), and fever (37%).

In this study most of the patients had signs such as icterus (51%), hepatomegaly (48%), pallor (24%) and hepatosplenomegaly (2%). In the present study majority of the hepatitis E patients (30.9%) had pallor followed by hepatitis B (25%), hepatitis A (14.8%) and hepatitis C (14.3%). Icterus was present in majority of hepatitis B patients (75%) followed by mixed hepatitis (66.7%), hepatitis E (54.5%) & hepatitis A patients (48.1%). Lymphadenopathy was not seen in any of the patients. Hepatomegaly was seen predominantly in hepatitis B (75%) followed by mixed hepatitis (66.7%), hepatitis A (55.6%), hepatitis E (43.6%) & hepatitis C (14.3%). Hepatosplenomegaly was observed predominantly in hepatitis B (12.5%) followed by hepatitis E (1.8%). Statistical significant difference was seen only in icterus in different types of hepatitis as p=0.04. This was consistence with other study done by Nagaich et al[14] who reported jaundice and hepatomegaly as most common sign.

In the present study most common risk factor from history was outside food intake (54%) followed by unsafe drinking water (41%), poor sanitization (32%), drug abuse (12%), Surgery (4%), blood transfusion (1%) and hemodialysis (1%). In hepatitis A the most common risk factor was frequent outside food intake (77.8%) followed by unsafe drinking water (31.7%) and poor sanitization (28.1%). Intravenous drug abuse was found in 65% of hepatitis B and 57.1% of hepatitis C with 100% cases of mixed infections. In hepatitis E frequent outside food intake (49.1%) and combined poor sanitization (46.9%) and no safe drinking water (46.37%) were found to be in almost equal proportions. Kumar et al[15] reported that high risk groups of hepatitis infection are injecting drug users (IDUs), truckers, and attendees of sexually transmitted infections (STI), persons suffering from thalassemia, hemophilia and other disease conditions requiring blood products transfusion.

In the present study 72% of the patients had haemoglobin <12 gm/dl and 28% had haemoglobin ≥12gm/dl. In our study, hemoglobin <12gm/dl was found in majority of the patients of hepatitis E (83.6%) followed by hepatitis B (75%), hepatitis A (66.7%), Mixed hepatitis (33.3%) & hepatitis C (14.3%). Hemoglobin>12 gm/dl was present in large percentage of hepatitis C (85.7%) followed by Mixed hepatitis (66.7%), hepatitis A (33.3%), hepatitis B (25%) & hepatitis E (16.4%). Difference in hemoglobin levels in different types of hepatitis was statistically significant p=0.001. In the study done by Birajdar et al[8], Haemoglobin level (<10 gm %) were seen in 10 (30.3%); 12 (27.9%); 1 (100%) and 9 (31.03%) in hepatitis A; B; C and E respectively. Desai et al[10] reported that in their study, anemia was seen in 27 (38.6%) patients. This is attributed to a temporary bone marrow suppression and autoimmune hemolytic anemia, which may accompany viral hepatitis.[16] Dilutional anemia is another possible explanation for this observation, as plasma volume is frequently increased in active hepatic disease.[17]

In the present study 6% of the patients had TLC <4000/µL and 94% had TLC ≥4000/µL. Total leucocyte count (TLC) <4000/µL was seen in Mixed hepatitis (66.7%) followed by hepatitis B (12.5%) & hepatitis A (11.1%).TLC>4000/µL was observed in all of hepatitis C & E (100%) followed by hepatitis A (88.9%), hepatitis B (87.5%) & mixed hepatitis (33.3%). In the study done by Birajdar et al[8] leukopenia (TLC count <4000/µL) was seen in 01 (3.03%); 02 (4.6%) and 05 (17.2%) cases of hepatitis A; B and E respectively. Study done by Ali SJ et al[18] also observed increase TLC in HBV patients. This is attributed to a virus interfering with leucopoiesis supports the more frequent finding of leucopenia rather than leukocytosis.[19]

20% of the patients had platelets <1.5 lakh/µL and 80% of the patients had platelets ≥1.5lakh/µL. Platelet count <1.5 lakh/µL was observed in hepatitis C (28.6%), hepatitis A (22.2%) & hepatitis E (21.6%). Platelet count >1.5lakh was observed in hepatitis B (100%), Mixed hepatitis (100%) followed by hepatitis E (78.2%), hepatitis A (77.8%) & hepatitis C (71.4%). Difference in platelet count in different types of hepatitis was not statistically significant p=0.51.In the study done by Birajdar et al[8] thrombocytopenia (platelet count <1.5 lakh) were observed in 02 (6.06%); 04 (9.3%); 1 (3.44%) in hepatitis A, B, and E respectively.

In the present study 50% of the patients had serum billirubin total >2.5mg/dl and 50% had serum bilirubin total ≤2.5mg/dl. 44% of the patients had serum billirubin direct ≥2mg/dl and 56% of the patients had serum billirubin direct <2mg/dl. Serum Bilirubin (Total) was >2.5mg/dl in high percentage of patients of hepatitis B (75%) followed by hepatitis E (52.7%), hepatitis A (51.9%) & mixed hepatitis (33.3%). It was ≤2.5mg/dl in all the patients of hepatitis C followed by mixed hepatitis (66.7%), hepatitis A (48.1%), hepatitis A (48.1%) & hepatitis E (47.3%). Difference in serum bilirubin total in different types of hepatitis was statistically insignificant p=0.49. Serum Bilirubin direct was ≥2mg/dl in majority of the patients of hepatitis B (62.5%) followed by hepatitis A (48.1%), hepatitis E (45.5%) & Mixed hepatitis (33.3%). Serum Bilirubin direct was <2mg/dl in all of the patients of hepatitis C followed by Mixed hepatitis (66.7%), hepatitis E (54.5%), hepatitis A (51.9%) & hepatitis B (37.5%). Difference in serum direct bilirubin in different types of hepatitis was statistically insignificant p=0.13. In the study done by Birajdar et al[8] Total serum bilirubin and direct serum bilirubin was raised in all cases of hepatitis A and E. In hepatitis B, total and direct serum bilirubin was raised in 36 (83.7%) and 34 (79.1%) respectively. Study done by Ashraf-uz-zaman et al[20] and Anand B et al[21] showed similar pattern of variations of serum bilirubin of viral hepatitis.

42% of the patients had SGOT <400U/L and 58% of the patients had SGOT ≥400U/L. 39% of the patients had SGPT <400U/L and 61% of the patients had SGPT ≥400U/L. SGOT levels <400U/L were found in all the hepatitis C patients followed by hepatitis B (50%), hepatitis E (40%), Mixed hepatitis (33.3%) & hepatitis A (29.6%). SGOT levels ≥400U/L were found in majority of hepatitis A patients (70.4%) followed by mixed hepatitis (66.7%), hepatitis E (60%) & hepatitis B (50%). Difference in levels of SGOT in different hepatitis was found to be statistically significant p=0.01. SGPT levels <400U/L were found in all the hepatitis C patients followed by hepatitis B (50%), hepatitis E (36.4%), Mixed hepatitis (33.3%) & hepatitis A (25.9%). SGPT levels ≥400 were found in majority of hepatitis A patients (74.1%) followed by mixed hepatitis (66.7%), hepatitis E (63.6%) & hepatitis B (50%). Difference in levels of SGPT in different hepatitis was found to be statistically significant p=0.009. In the study done by Birajdar et al[8] Raised SGPT and SGOT were observed in all cases of hepatitis A and E. Among 43 cases of hepatitis B cases SGPT and SGOT was raised in 32 (74.4%) and 31 (72.1%) respectively.

In the present study 39% of the patients had international normalized ratio <1.1, 53% had international normalized ratio 1.1-1.5 and 8% had international normalized ratio >1.5. In our study, INR < 1.1 was predominantly seen in hepatitis C patients (85.7%) followed by hepatitis E (43.6%), hepatitis B (37.5%), mixed hepatitis (33.3%) & hepatitis A (18.5%). INR 1.1-1.5 was seen predominantly in hepatitis A (74.1%) followed by hepatitis B (50%), hepatitis E (49.1%), Mixed hepatitis (33.3%) & hepatitis C (14.3%). INR >1.5 was predominantly seen in Mixed hepatitis (33.3%) followed by hepatitis B (12.5%), hepatitis A (7.4%) & hepatitis E (7.3%). Differences in INR in different types of hepatitis was not statistically significant p=0.059. Girish et al[1] in their study reported that PT INR varied from less than 1.5 to more than 3.5. PT INR of less than 1.5 seen in most of the cases that is in 44 cases (91.6%), INR of more than 3.5 seen in 2 cases (4.16%), INR between 1.5 and 2.5 was seen only in 1 case (2.08%).

In the present study 34% of the patients had DSP <3.5 and 66% of the patients had DSP ≥3.5. DSP <3.5 was seen in all the mixed hepatitis patients followed by hepatitis E (34.5%), hepatitis A (33.3%), hepatitis

B (25%) & hepatitis C (14.3%). DSP ≥3.5 was predominantly seen in hepatitis C patients (85.7%) followed by hepatitis B (75%), hepatitis A (66.7%) & hepatitis E (65.5%). Differences in DSP in different types of hepatitis was not statistically significant p=0.11. Similar results observed in study done by Birajdar et al. in 17.9% cases and by Dabadghao et al[7].

In the present study 56% of the patients had serum creatinine <1.1 and 44% of the patients had serum creatinine ≥1.1. Serum creatinine <1.1 was seen in all the patients of hepatitis C followed by mixed hepatitis (66.7%), hepatitis E (54.5%), hepatitis B (50%) & hepatitis A (48.1%). Serum creatinine ≥1.1 was seen predominantly in the patients of hepatitis A (51.9%) followed by hepatitis B (50%), hepatitis E (45.5%), & mixed hepatitis (33.3%). Differences in serum creatinine levels in different types of hepatitis was not found to be statistically significant p=0.16. Brehm et al[22] reported that patients with acute hepatitis E had significantly higher median serum creatinine levels (0.9 mg/dL vs. 0.8 mg/dL) and significantly lower median estimated glomerular filtration rate compared to patients with acute hepatitis A.

Given the higher prevalence of hepatitis E followed by hepatitis A quality standards for public water supplies should be maintained and proper sewage system should be established. At individual level hygiene practices should be maintained. The government of India should make the hepatitis A vaccine as priority in national immunization schedule. IV drug abuse being a major risk factor for hepatitis B and C in our current study, serology testing for these viruses should be made available in centers like OOAT clinics and other deaddiction centers apart from medicine and psychiatry departments. This aids in early screening, diagnosis and treatment.

CONCLUSION:

Viral hepatitis is an important heath care problem in India as it occurs epidemically and sporadically. The variability in nature of the disease regarding its onset, presenting symptoms, clinical course and development of complications are important aspects. So, it is very essential for health care professionals to be aware of all aspects of it so that it is detected and treated early.

REFERENCES:

1. Girish N, Sunil B, Devaranavadagi RA. A clinical study of viral hepatitis in children: a prospective hospital-based study. Int J ContempPediatr 2018; 5:563-8.
2. Cacciola I, Scoglio R, Alibrandi A, Squadrito G, Raimondo G., SIMG-Messina Hypertransaminasemia Study Group. Evaluation of liver enzyme levels and identification of asymptomatic liver disease patients in primary care. Intern Emerg Med. 2017 Mar;12(2):181-186.
3. Agrawal S, Dhiman RK, Limdi JK. Evaluation of abnormal liver function tests. Postgrad Med J. 2016 Apr;92(1086):223-34.
4. Woreta TA, Alqahtani SA. Evaluation of abnormal liver tests. Med Clin North Am. 2014 Jan;98(1):1-16.
5. Ryder SD, Beckingham IJ. ABC of diseases of liver, pancreas, and biliary system: Acute hepatitis. BMJ. 2001 Jan 20;322(7279):151-3.
6. Montrief T, Koyfman A, Long B. Acute liver failure: A review for emergency physicians. Am J Emerg Med. 2019 Feb;37(2):329-337.
7. Dabadghao V, Barure R, Sharma S, Mangudkar S. A study of the clinical and biochemical profile of acute viral hepatitis. Int J Biomed Adv Res 2015;6:68993.
8. Birajdar SV, Chavan SS, Mundhe SA, Bhosale MG. Clinical and biochemical profile of patients with viral hepatitis at tertiary care centre. Int J Adv Med 2017; 4:412-6.
9. Chakrabarti K, Ballala K, Rao K, Patil N. A study to assess the clinical and biochemical profile of patients diagnosed with hepatitis E in a large teaching hospital of Southern India. Asian J Pharm Clin Res. 2016;9(2):84-9.
10. Desai HD, Ansari AA, Makwana D, Jadeja DM, Gusani J. Clinical-biochemical profile and etiology of acute viral hepatitis in hospitalized young adults at tertiary care center. J Family Med Prim Care 2020; 9:247-52.
11. Modi TN, Patel SA, Mirani KM, Vaghasiya DR, Makadia GS, Usdadiya J. A study of clinical profile and outcome in acute viral hepatitis E. Indian J Clin Pract. 2013;23(10):635-7.
12. Shah NA, Kadla SA, Shafi PM, Dar IH, Ali I, Rasheed S, et al. Clinico-serological profile of acute sporadic viral hepatitis in Kashimiri adults: Hospital based prospective study. JMSCR 2014;2:3119-26.
13. Zhang S, Wang J, Yuan Q. Clinical characteristics and risk factors of sporadic Hepatitis E in central China. Virol J. 2011;8:152.
14. Nagaich N, Raghav M, Sharma R. Clinical profile of outbreak of viral hepatitis and its outcome at tertiary care Centre in Jaipur. Int J Current Advanced Res. 2016;5(1):577-80
15. Kumar M and Sarin SK. Viral hepatitis eradication in India by 2080-gaps, challenges & targets. The Indian Journal of Medical Research. 2014 Jul; 140(1):1.
16. Gumba SC, Chopra S. Hepatitis C: A multifactorial disease, review of extrahepatic manifestations. Ann Intern Med 1995;123:615-20.
17. Conrad ME, Schwartz FD, Young AA. Infectious Hepatitis-A generalized disease. A study of renal, gastrointestinal and haematologic abnormalities. Am J Med 1964;37:789-801.
18. Ali SJ. A correlative study between haematological and biochemical parameters in Hepatitis B. Ibn AL- Haitham J For Pure Appl Sci [S.l.] 2019;32:21-9.
19. Jones GP, Evans EG. Idiopathic thrombocytopenic purpura in infective hepatitis. BMJ 1951;2:451-2.
20. Ashraf-Uz-Zaman M, Begum B, Asad H. Biochemical parameters in common viral hepatitis. J Med. 2010;11:42-5.
21. Anand BS, Velez M. Assessment of correlation between serum titers of hepatitis C virus and severity of liver disease. World J Gastroenterol. 2004;10(16):2409-11.
22. Brehm TT, Mazaheri O, Horvatits T, Lutgehetmann M, Schulze zur Wiesch J, Lohse AW et al. Lower Levels of Transaminases but Higher Levels of Serum Creatinine in Patients with Acute Hepatitis E in Comparison to Patients with Hepatitis A. Pathogens. 2021;10:60.