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STUDY OF CLINICAL PRESENTATION OF HEPATITIS C IN CASES OF ACUTE VIRAL HEPATITIS AND CIRRHOSIS OF LIVER

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ABSTRACT

BACKGROUND: Hepatitis C virus is a major causative agent of liver disease, resulting in acute and chronic infections that can lead to fibrosis, cirrhosis and hepatocellular carcinoma. Acute hepatitis is usually asymptomatic. The main objective was to study the clinical presentation of Hepatitis C in cases of acute viral hepatitis and cirrhosis of liver.

METHOD: The study was carried out for a period of one year from April 2002 to February 2003.The study was carried out in the department of Medicine, Pt. J.N.M. Medical College and associated Dr. B.R.Ambedkar Memorial Hospital, Raipur, Chhattisgarh. 52 cases of acute viral hepatitis and 42 cases of cirrhosis of liver were identified consecutively

RESULT: Out of 52 cases, icterus was noted in 98%, hepatomegaly in 80%, splenomegaly in 26% and cervical lymphadenopathy in 3% clinically. Abdominal distension was the commonest complaint reported by 94% of the cases followed by anorexia and flatulent dyspepsia in 86% cases. Ascites was the commonest clinical sign, detected in 96% of cases. Next to follow it was the splenomegaly noted in 60% of cases. In decreasing order of frequency were the pallor, icterus, hepatomegaly, veins, gynaecomastia and testicular atrophy, pedal edema and spider naevi accounting for 55%.38%, 31%, 29%, 22%, 18% and 12% of cases.

CONCLUSION: Improved prevention and expanded access to viral hepatitis treatments could greatly reduce the burden of these infections. Strategies to enhance HCV assessment are urgently needed

KEYWORDS : Hepatitis C, Acute Viral Hepatitis, Cirrhosis Of Liver.

INTRODUCTION:

Hepatitis could be defined as continuing disease without improvement for at least six months. Traditionally, chronic hepatitis has been classified as chronic persistent hepatitis (CPH) and chronic aggressive hepatitis or chronic active hepatitis (CAH). The committee of the international association for the study of the liver(IASL) pointed out that the traditional classification has often been misinterpreted in attempting to differentiate between separate disorders rather than in stating degrees of severity and has emphasized that the various forms of chronic hepatitis represent a clinical pathological syndrome and not a single disease.[1]

Chronic hepatitis following acute post transfusion hepatitis is no more common than that following sporadic or community acquired disease when anti -HCV – positive cases are compared. When anti-HCV tests are non reactive, chronic hepatitis is less common than when these tests are positive.[2]

90% of chronic hepatitis C patients had IgM anti HCV core IgM anti HCV – core levels were inversely correlated with the histological activity suggesting inconclusively that IgM anti HCV- core may play role in the pathogenesis of chronic hepatitis C.They found that 87% of patients had both markers simultaneously in their sera, which indicate that detection of IgM anti- HCV- core related with HCV replication. In this study, most of the non responder to IFN- alfa lost IgM anti – HCV core and HCV RNA with significantly greater frequency and both markers became undetectable in some transient responders during ALT normalization. IgM anti HCV should, therefore be considered as a sensitive marker of active HCV replication and may be useful in monitoring the course of anti viral therapy in chronic hepatitis C.[3]

The anti- HCV antibody assay systems detect mainly IgM type antibodies. In the early stage of acute hepatitis C, IgM is positive and IgG is negative or detected at low levels while in chronic hepatitis C, IgG is present in high levels irrespective of the presence or absence of IgM.[4]

Main clinical indicators for PCR in HCV are diagnosis of acute HCV infection, anti HCV- patients with chronic hepatitis, evaluation of HCV viremia in asymptomatic blood donors with normal ALT levels, Anti HCV + chronic hepatitis with anti- LKM 1 auto antibodies: decision for interferon therapy, Evaluation of HCV infections after liver transplantation, Mother to child HCV transmission and follow up antiviral therapy.[5]

METHOD:

The study was carried out for a period of one year from April 2002 to February 2003.The study was carried out in the department of Medicine, Pt. J.N.M. Medical College and associated Dr. B.R.Ambedkar Memorial Hospital, Raipur, Chhattisgarh. 52 cases of acute viral hepatitis and 42 cases of cirrhosis of liver were identified consecutively.52 cases of acute viral hepatitis included 42 males and 10 females ranging in age from 14 to 65 years and 42 cases of cirrhosis of liver included 25 males and 17 females, ranging in age from 15 to 70 years. Selection of cases was based mainly on clinical identification. Symptoms such as anorexia, nausea, vomiting, abdominal pain, fever, yellow discoloration of urine and sclera of less than one month duration were looked for acute viral hepatitis. Relevant past history was recorded. The patients enrolled for the study were subjected to routine investigations which included hemogram, urine examination and blood biochemistry. A complete liver function test was obtained. Tests of biosynthetic functions of the liver were carried out in cirrhotic that included estimation of cholesterol, albumin and globulin. Liver biopsy have been the most appropriate method to determine whether the patients had hepatitis/ cirrhosis or not but given the practical consideration ultra sonographic study was made use of to clinch the diagnosis. Serum samples were obtained from all the patients by venepuncture taking into account the strict aseptic precaution. The test device is meant for in vitro diagnostic use and is a qualitative, lateral flow immunoassay for the detection of antibody to HCV in serum or plasma. The recombinant HCV polyprotein used in the kit is encoded by the genes for both structural and non structural proteins including core, NS3, NS4 and NS5 regions, so it is a third generation immunoassay.

RESULT:

Table 1: Symptoms In 52 Cases Of Acute Viral Hepatitis

Symptoms	No of Cases	Percentage
Loss of appetite	47	92%
Yellow coloured urine	46	89%
Yellow coloured eyes	45	87%
Nausea and vomiting	39	75%
Fever	20	39%
Pain in abdomen	12	23%
Pruritis	4	9%

52 cases included in the study presented with the above mentioned symptoms in varied combinations. Loss of appetite was the commonest complaint in 92% cases. Yellow coloured urine and eyes

were the next common symptoms accounting for 89% and 87% respectively. Nausea and vomiting was seen in 75% of the cases. Fever mostly low grade, was seen in 39% cases while pain in abdomen was seen in 23% cases.9% were the cases with pruritis.

Table2: Symptoms In 42 Cases Of Cirrhosis Of Liver.

Symptoms	No of Cases	Percentage
Abdomen distension	39	94%
Anorexia and flatulent dyspepsia	36	86%
Yellow coloured urine/eyes	23	57%
Malaena	19	47%
Haematemesis	11	28%

Abdominal distension was the commonest complaint reported by 94% of the cases followed by anorexia and flatulent dyspepsia in 86% cases. Yellow discoloration of sclera and urine was noted in 57%. 47% of the cases complained of malaena while haematemesis was present in 28% cases.

Table 3: Clinical Signs In 52 Cases Of Acute Viral Hepatitis.

Signs	No of Cases	Percentage
Icterus	51	98%
Hepatomegaly	42	80%
Splenomegaly	13	26%
Lymphadenopathy Cervical	02	03%

Out of 52 cases, icterus was noted in 98%, hepatomegaly in 80%, splenomegaly in 26% and cervical lymphadenopathy in 3% clinically.

Table 4: Clinical Signs In 42 Cases Of Cirrhosis Of Liver.

Signs	No of Cases	Percentage
Ascites	40	96%
Splenomgaly	25	60%
Pallor	23	55%
Icterus	16	38%
Hepatomegaly	13	31%
Veins	12	29%
Gynaecomastia and testicular atrophy	09	22%
Paedal Oedema	07	18%
Spider naevi	05	12%

This table shows the ascites was the commonest clinical sign, detected in 96% of cases. Next to follow it was the splenomegaly noted in 60% of cases. In decreasing order of frequency were the pallor, icterus, hepatomegaly, veins, gynaecomastia and testicular atrophy, pedal edema and spider naevi accounting for 55%.38%, 31%, 29%, 22%, 18% and 12% of cases.

DISCUSSION:

Choo and colleagues used antibodies from an infected patient to detect virus – specific proteins in vitro at the Chiron corporations in California. They extracted nucleic acid from the plasma of chimpanzees experimentally infected with PT –NANBH in whom the titre of infectivity was high. The isolated nucleic acid was denatured and then subjected to reverse transcription for the production of complimentary DNA.[6]

Feinstone SM et al reported neither Hepatitis A nor Hepatitis B accounted for the post- transfusion hepatitis in a group of cardiac surgery patients monitored prospectively at the National Institute of Health.[7]

Cooreman MP et al reported that genotypes are numbered chronologically, according to the order of description, whereas subtypes are defined alphabetically. This practice is based on the assumption that no recombination of viruses occurs. Eg a virus with a nucleotide sequence corresponding to one genotype in its structural region and to another genotype in its non structural domain. As such recombinant viruses have not been identified, determining the genotype by selecting one part of the genome is accepted practice. This method of grouping allows integration of all identified genotypes and subtypes into one system that can be adapted a new types are discovered on the basis of nucleotide sequence divergence, without the need for reassessment.[8]

Zeuzem S et al argued against transmission by haemodialysis facilities. They studied 164 haemodialysis patients of which 19 were anti – HCV and 16 were HCV – RNA positive. Sequence analysis of virus isolates in 13/16 HCV – RNA positive patients revealed subtype distribution similar to respective subtype percentages in non –hemdialysis patients with chronic hepatitis C in Germany.[9]

Chiba et al reported that most HCV- related HCC arose in the cirrhotic liver. Cox proportional hazard regression analysis identified the progressive stages of liver disease as a primary risk factor for development of HCC. They concluded that continuous liver cell necrosis and multiple hepatocyte regrowth causes mutational genetic error to lead to the development of cancer. In their study anti- HBV positivity was found to be the second risk factor for generation of HCC in HCV related CLD. HBV infection through integration of HBV into host chromosomal DNA may activate cellular ontogenesis either directly or by disruption of tumors suppressor gene function. Alternatively chronic inflammation may increase the probability of the occurrence of a critical mutational event and subsequent tumor formation.[10]

CONCLUSION:

HCV is a major public health problem worldwide and it is more prevalent in our area. Improved prevention and expanded access to viral hepatitis treatments could greatly reduce the burden of these infections. Strategies to enhance HCV assessment are urgently needed. Elisa is the most popular but it is expensive and time consuming. It is not practical method for routine screening in our setting. The HCV test kit is used in this study will come handy because it is rapid, simple and needs no expensive equipment. The HCV test kit is very useful in situations like emergency blood transfusion.

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