

# A Study of Liver Checkup in Patients with Hepatitis C in the Region of Batna

A. Zidani, M. Yahia K. Belhadi, and S. Benbia

**Abstract**—Hepatitis C is an infectious disease transmitted by blood and due to hepatitis C virus (HCV), which attacks the liver. The infection is characterized by liver inflammation (hepatitis) that is often asymptomatic but can progress to chronic hepatitis and later cirrhosis and liver cancer. Our problem tends to highlight on the one hand the prevalence of infectious disease in the population of the region of Batna and on other hand the biological characteristics of this disease by a screening and a specific diagnosis based on serological tests, liver checkup (measurement of haematological and biochemical parameters).

*The results showed:*

The serology of hepatitis C establishes the diagnosis of infection with hepatitis C. In this study and with the serological test, 24 cases of the disease of hepatitis C were found in 1000 suspected cases (7 cases with normal transaminases and 17 cases with elevated transaminases). The prevalence of this disease in this study population was 2.4%.

The presence of hepatitis C disrupts liver function including the onset of cytolysis, cholestasis, jaundice, thrombocytopenia, and coagulation disorders.

**Keywords**—Disease hepatitis C, serology, liver checkup

## I. INTRODUCTION

**H**EPATITIS means inflammation of the liver, this condition can have many causes. One of these causes, hepatitis C, known as infection HCV is a virus that attacks the liver and can cause inflammation and scarring. The diagnosis of hepatitis C begins with serological and blood tests to detect antibodies to HCV. Monitoring of chronic hepatitis involves performing successive liver checkup which allow to assess enjoy the impact of the virus on liver function [1]. The aim of this study with 1000 subjects suspected of a population living in the region of Batna is to know the biological diagnosis of the disease, highlighting the prevalence of hepatitis C in the population of the region of Batna and clarifying the biology of this disease.

## II. MATERIALS AND METHODS

### A. Patients and Methods

Our study was conducted on 1000 subjects suspects living in the region of Batna, in both sexes and various ages, including screening was done after a routine health check showing hypertransaminasemia during a review of viral hepatitis B or HIV infection, in a review of blood donor or to

Authors are from the Department of Biology, University of Batna, Republic of Algeria Biotechnology's laboratory of the bioactive molecules and the cellular physiopathology (✉corresponding author to provide phone: 00 213 778796334; fax: 00 213 33 86 23 71 E-mail: abla.biologie@yahoo.com

non-specific clinical signs such as fatigue and abdominal pain. This study was conducted in the microbiology laboratory of the CHU of Batna, Central Laboratory of Hematology University Hospital of Batna, biochemistry laboratory of the sanatorium of Batna and the laboratory of anatomy and pathological cytology of the Batna University Hospital. The study lasted 12 months, from July 2009 to June 2010.

HCV serology is to investigate the presence of HCV antibodies, evidence of a specific reaction of the body against the virus and therefore a HCV infection. A positive test must be confirmed by a second blood test.

We used the HCV test Monalisa® Ag - Ab ULTRA for serological diagnosis is an immunoassay - enzyme for the detection of HCV infection based on antibody and core antigen associated with infection with hepatitis C in serum or plasma.

The main strengths are that we realized the determination of biochemical parameters (transaminases [GOT and GPT], alkaline phosphatase [ALP], gamma-GT and bilirubin) and the determination of haematological parameters (prothrombin time, count of blood platelets and factor V).

### B. Statistical analysis

The statistical method used is the "t" test of student to compare the sample means. The results are expressed as mean  $\pm$  sem, calculations were performed using the software Graph Pad Prism 5.00.

## III. RESULTS AND DISCUSSION

From 1000 serological diagnosis of suspected cases, the disease was only found in 24 patients, this means that the frequency is estimated at 2.4% from which they received a liver checkup.

After the determination of transaminases, 7 of 24 positively tested patients have normal transaminases, against the rest of the patients (17 cases) which has a high transaminases.

TABLE I  
VARIATION IN THE RATE OF TRANSAMINASES (GOT, GPT) IN PATIENTS WITH HEPATITIS C IN NORMAL TRANSAMINASES.

State / Biological test	Normal Control	Patients with normal transaminases	P
GOT rate (IU / L)	27 $\pm$ 2,6	29 $\pm$ 3,5	0.5934
GPT rate (IU / L)	28 $\pm$ 1,7	30 $\pm$ 2,4	0.5412

Normal value: 5-40 IU / L

The results show that there is no statistically significant difference in the rate of GOT ( $p = 0.5934, > 0.05$ ) in these patients (29  $\pm$  3.5) by contribution to controls (27  $\pm$  2.6). The result is the same rate for the GPT, there is no statistically

significant difference ( $p = 0.5412, > 0.05$ ) in these patients ( $30 \pm 2.4$ ) by contribution to controls ( $28 \pm 1, 7$ ).

A number of patients with chronic HCV infection have normal transaminases continuously despite the presence of detectable viremia (viral RNA detectable by PCR in serum). These patients are often identified during screening. The

TABLE VI

VARIATION OF PROTHROMBIN TIME AND FACTOR V IN PATIENTS WITH HEPATITIS C

State / Biological test	Normal Control	Patients	P
Prothrombin time (%)	$95 \pm 1,8$	$68 \pm 2,6$	$<0.0001$
FV rate (%)	$89 \pm 4,6$	$55 \pm 2,7$	$<0.0001$

Normal values: PT : 70 – 100 %, FV : 70 – 120 %

definition of this group of patients must be strict: positivity for anti-HCV positivity of HCV RNA by PCR and strictly normal transaminases. This requires at least 3 doses of transaminases over a period of at least 6 months [2].

Factors associated with normal transaminases, the female is the one found most consistently. Other factors are inconsistently found as young or a low viral load. In contrast to earlier studies, the genotype does not appear to be an associated factor. Finally, few data are available physiopathologic. These include the more frequent association

TABLE II

VARIATION IN THE RATE OF TRANSAMINASES (GOT, GPT) IN PATIENTS WITH HEPATITIS C AT ELEVATED TRANSAMINASES.

State / Biological test	Normal Control	Patients at elevated transaminases	P
GOT rate (IU / L)	$27 \pm 2,6$	$82 \pm 9,5$	$0.0002$
GPT rate (IU / L)	$28 \pm 1,7$	$87 \pm 9,2$	$<0.0001$

Normal value: 5-40 IU / L

with certain HLA groups including HLA-DRB1 \* 11. The mechanisms responsible for chronic hepatitis C with normal aminotransferase activity are mainly related to an impaired immune response of the host against the viral infection [3].

Our results reveal the existence of a statistically significant difference in the rate of GOT ( $p = 0.0002, <0.05$ ) in our patients ( $82 \pm 9.5$ ) by contribution to controls ( $27 \pm 2.6$ ) and a statistically very highly significant rate GPT ( $p <0.0001$ ) in these patients ( $87 \pm 9.2$ ) by contribution to controls ( $28 \pm$

TABLE III

VARIATION IN THE RATE OF ALKALINE PHOSPHATASE AND GGT IN PATIENTS WITH HEPATITIS C

State / Biological test	Normal Control	Patients	P
ALP rate (IU / L)	$75 \pm 4,1$	$140 \pm 12$	$0.0040$
GGT rate (IU / L)	$19 \pm 1,5$	$87 \pm 9,2$	$0.0190$

Normal values: ALP : 35 – 104 IU/L, GGT : 5 – 36 IU/L

1.7). Elevated transaminase levels are a sign of cytolysis indicating a breach of the hepatocyte membrane. This achievement is a destruction of the membrane that defines the hepatocyte necrosis. Therefore, substances normally contained in the hepatocyte will be released into the sinusoids and their concentration in peripheral blood will increase [4].

There has been a statistically significant difference in alkaline phosphatase ( $p = 0.0040, <0.05$ ) between controls ( $75$

$\pm 4.1$ ) and patients ( $140 \pm 12$ ). Examination of these results allows to evidence a significant increase ( $p = 0.0190, <0.05$ ) of GGT in these patients ( $87 \pm 9.6$ ) by contribution to controls ( $19 \pm 1.5$ ).

The increase in alkaline phosphatase in hepatobiliary disorders is a reflection of cholestasis. Cholestasis is the disruption of bile flow by impaired bile formation or impede the flow through the biliary tree. Most liver diseases cause of cholestasis by disrupting intracellular transport mechanisms or by altering the small interlobular bile ducts. Under the influence of cholestasis, hepatocyte produces an excess number of substances whose serum concentration increases: alkaline phosphatase and GGT. If elevations of alkaline phosphatase, the assay of  $\gamma$ -GT is useful to confirm that this increase is of hepatic origin [5].

According to the analysis of these results, there was a significant difference in the rate of total bilirubin ( $p = 0.0127, p <0.05$ ) between patients ( $13 \pm 1.6$ ) and controls ( $6.3 \pm 0.64$ ).

TABLE IV  
VARIATION IN THE RATE OF BILIRUBIN IN PATIENTS WITH HEPATITIS C

State / Biological test	Normal Control	Patients	P
Total bilirubin (mg / L)	$6,3 \pm 0,64$	$13 \pm 1,6$	$0.0127$
Direct bilirubin (mg / L)	$1,4 \pm 0,18$	$4,7 \pm 1,1$	$0.0484$

Normal values: Total bilirubin  $\leq 10$  (mg / L), Direct bilirubin  $\leq 2,5$  (mg / L)

The same for the direct bilirubin, there is a significant difference in the rate of direct bilirubin ( $p = 0.0484, p <0.05$ ) between patients ( $4.7 \pm 1.1$ ) and controls ( $1.4 \pm 0.18$ ).

The bilirubin is a natural substance in the body. It comes from the breakdown of hemoglobin (bilirubin free). Then she is captured by the liver (bilirubin) and degraded. An turgor injured hepatocytes causes obstruction for bile ducts, bilirubin conjugated posthépatique accumulates in the bloodstream, causing jaundice [6].

TABLE V  
VARIATION IN THE RATE OF PLATELET IN PATIENTS WITH HEPATITIS C

State / Biological test	Normal Control	Patients	P
Platelet rate (103/mm <sup>3</sup> )	$220 \pm 15$	$140 \pm 14$	$0.0020$

Normal value: 150 – 500 x 103/mm<sup>3</sup>

Our results show that there is a significant decrease in platelet rate ( $p = 0.0020, <0.05$ ) in these patients ( $140 \pm 14$ ) by contribution to controls ( $220 \pm 15$ ).

A reduced rate of platelets is a condition called thrombocytopenia. This condition is dangerous because it poses a risk of bleeding internally and externally.

The frequent presence of thrombocytopenia in patients chronically infected with HCV, may belong to several mechanisms: peripheral arthritis (hypersplenism, autoimmune thrombocytopenia by anti-platelet or anti-phospholipid antibodies, HCV RNA in platelets) Central or damage (HCV RNA in megakaryocytes, lymphoproliferative malignancy, decreased hepatic production of thrombopoietin) [7]The results show the existence of a very highly significant decrease in prothrombin time ( $p <0.0001$ ) in our patients ( $68 \pm 2.6$ ) by contribution to controls ( $95 \pm 1.8$ ). For factor V, there

was a very highly significant decrease ( $p < 0.0001$ ) in these patients ( $55 \pm 2.7$ ) by contribution to controls ( $89 \pm 4.6$ ).

The decrease in the PT results in the absence of hepatic synthesis of factors, or lack of absorption of vitamin K in case of cholestasis.

The factor V is a reflection more specific hepatobiliary function as prothrombin time. Its rate does not decrease when cholestasis because it causes a vitamin K deficiency and factor V does not require vitamin K for its synthesis [8].

#### IV. CONCLUSION

Hepatitis C is a relatively common disease. It is estimated that 3% of world population has a chronic infection with hepatitis C virus (HCV) and HCV accounts for about 70% of cases of chronic hepatitis, a major cause of cirrhosis and hepatocellular carcinoma. The silent evolution of the disease and the high incidence of chronicity explain the existence of a large reservoir of infected individuals. Thus, although HCV is not highly contagious, it is widely transmitted mainly by parenteral route. Acute hepatitis C is usually asymptomatic, which explains why the diagnosis is rarely in the acute stage of the disease. Chronic hepatitis is usually asymptomatic and its diagnosis is incidental in most cases, sometimes at a late stage of the disease. It is based on laboratory tests.

The diagnosis of the disease of hepatitis C that was performed on 1000 suspected subjects of a population living in the region of Batna allowed us to demonstrate that:

The prevalence of hepatitis C in the region of Batna was 2.4% contribution to the study population. Were detected twenty-four cases of hepatitis C. The prevalence of this disease in this study population the prevalence is close to theoretical.

This pathology leads to the onset of cytolysis, cholestasis, jaundice, thrombocytopenia, and clotting disorders including the determination of transaminases explores the cytolysis, assay of alkaline phosphatase and gamma-GT explores cholestasis, the bilirubin explores jaundice, platelet count explores thrombocytopenia, the prothrombin time with factor V explore coagulation disorders

In fact the frequency and severity of the disease, hepatitis C has been and is the subject of numerous researches, which allowed, despite the absence of effective systems for viral culture, to understand molecular biology and the viral replication cycle.

#### REFERENCES

- [1] D. Paul, R. Dominique, (2003). "Virus de l'hépatite C". Elsevier, Ed. Paris, p. 190, 2003.
- [2] A. Piton, T. Poynard, F. Imbert-Bismut, and al, "Factors associated with serum alanine transaminase activity in healthy subjects: consequences for the definition of normal values, for selection of blood donors, and for patients with chronic hepatitis C", *Hepatology*, vol. 27, pp. 1217-9, 1998.
- [3] B. Kronenberger, B. Rüster, J. H. Lee, and al, "Hepatocellular proliferation in patients with chronic hepatitis C and persistently normal or abnormal aminotransferase levels", *Hepatology*, vol. 33, pp. 640-7, 2002.
- [4] J. P. Capron, "Augmentation modérée et prolongée de l'activité sérique des transaminases", *Presse médicale*, vol. 18, pp. 913-6, 1989.
- [5] E. U. Sotil, D. M. Jensen, "Serum enzymes associated with cholestasis", *Clin Liver Dis*, vol. 8, pp. 41-54, 2004.
- [6] B. Jean-Pierre, B. Johannes, M. Neil, R. Mario, R. Juan, "Hépatologie clinique", 2nd ed. Médecine-Sciences Flammarion, Ed. Paris, p.2148, 2002.
- [7] T. Lisman, T. N. Bongers, J. Adelmeijer, H. L. Janssen, MP. de Maat, PG. de Groot, "Elevated levels of von Willebrand Factor in cirrhosis support platelet adhesion despite reduced functional capacity", *Hepatology*, vol. 44, pp. 53-61, 2006.
- [8] C. Eugène, L. Costentin, S. Beaulieu, "Les hépatites virales", 2nd ed. Masson, Ed. Paris, p. 194, 2004.