Scientific Research Publishing

Open Journal of Gastroenterology, 2024, 14, 313-321
https://www.scirp.org/journal/ojgas
ISSN Online: 2163-9469
ISSN Print: 2163-9450

Hepatic Elastometry in the Management of Hepatitis B at National Hospital of Niamey

Ousseini Fanta[1,2*], Hamidine Illa[3,4], Moussa Saley Sahada[1,5], Abdourahamane Idrissa Fatouma[2], Abdou Nafissatou[2], Ali Cheik Rakia[6], Abdou Boubé[7], Abdou Djibo Ben Moctar[8], Ky Lawagoulé Joseph Emile[8], Daou Mamane[1,9]

[1]Faculty of Health Sciences, Abdou Moumouni University, Niamey, Niger
[2]Department of Hepato-Gastroenterology, National Hospital of Niamey, Niamey, Niger
[3]Laboratoire de Recherche Clinique et Système de Santé Niger, Université André Salifou of Zinder, Zinder, Niger
[4]Department of Hepato-Gastroenterology, Zinder National Hospital, Zinder, Niger
[5]Department of Infectiology, National Hospital of Niamey, Niamey, Niger
[6]Regional Hospital Centre of Niamey, Niamey, Niger
[7]Department of Hepato-Gastroenterology, General Reference Hospital of Niamey, Niamey, Niger
[8]Department of Hepato-Gastroenterology, The University Hospital, Ouagadougou, Burkina Faso
[9]Department of Internal Medicine, General Reference Hospital of Niamey, Niamey, Niger
Email: ousseinifanta0@gmail.com

How to cite this paper: Fanta, O., Illa, H., Sahada, M.S., Fatouma, A.I., Nafissatou, A., Rakia, A.C., Boubé, A., Ben Moctar, A.D., Emile, K.L.J. and Mamane, D. (2024) Hepatic Elastometry in the Management of Hepatitis B at National Hospital of Niamey. *Open Journal of Gastroenterology*, **14**, 313-321.
https://doi.org/10.4236/ojgas.2024.149033

Received: August 10, 2024
Accepted: September 21, 2024
Published: September 24, 2024

Abstract

Introduction: Viral hepatitis B is a public health problem in Niger which is classified as a high endemicity area with a prevalence ranging from 8 to 17% depending on the studies [1] and that of HBV-related cirrhosis is about 40.26% in 2024. The decision to treat is based on a combination of three parameters: viral load, ALT values and the degree of hepatic fibrosis [2]. The latter is assessed by hepatic elastography (Fibroscan), which is a decisive factor in treatment. In Niger, until 2024, the decision to treat or not to treat a patient with HBV was based on the determination of viral load B and transaminases, and no work evaluating the contribution of this third element, liver elasticity, has been done, hence the interest of our study. **Objective**: To study the contribution of Fibroscan in measuring hepatic elasticity in the management of patients with HBV. **Methodology**: This was a prospective descriptive study conducted from January 05 to November 30, *i.e.* a period of 11 months, on clinically asymptomatic HBsAg-positive patients who had undergone FibroScan liver elasticity measurement. The examination was carried out by a hepatogastroenterologist who had received training in the Fibroscan. The median of ten measures of liver elasticity at the same point with an IQR of less than 30% was considered the valid measure and no or minimal fibrosis was defined as a value <7 Kpa, moderate fibrosis as a value between 7 and 10 kpa, severe fibrosis as a value greater than 10 Kpa, and the existence of cirrhosis as

a value greater than 14 Kpa were analyzed using SPSS version 20 software. **Results**: Out of 398 patients monitored for HBV, 60 cases met the inclusion criteria, *i.e.*, a frequency of 15.07%. The mean age of the patients was 35.63 years, with extremes of 18 and 70 years. They were predominantly male, with a sex ratio of 3.2. Married patients accounted for 61.67% (n = 37). Jaundice was absent in 91.67% (n = 55). The circumstances of discovery of HBV were the routine health check-up, followed by blood donation with 50% and 46.67%, respectively. The viral load was >2000 UI/ml in 32.7% (n = 17). HBeAg was negative in 93.33% of cases (n = 56). ALT levels were normal in 47 patients (78.33%). Mean liver elasticity was 6.7 KPa. Fibrosis was classified as F0 - F1 in 75% (n = 45), F1 - F2 in 18.33% (n = 11) and F3 - F4 in 6.67% (n = 4) of patients. There was no significant relationship between viremia value, liver activity, degree of fibrosis and quantitative HBsAg. **Conclusion**: Measurement of hepatic elasticity has made it possible to diagnose cases of compensated cirrhosis and significant fibrosis in patients considered to be inactive carriers (viral load <2000 IU/ml and normal transaminases) in asymptomatic HBV+ patients. This made it possible to put these patients on Tenofovir in order to avoid decompensation for the first group and for the second the progression to cirrhosis. It is an excellent tool to aid in the decision to start treatment.

Keywords

Hepatic Elasticity, Fibroscan, Fibrosis, Hepatitis B, Niger

1. Introduction

Hepatitis B is an infectious disease caused by the hepatitis B virus (HBV). It represents a major public health problem worldwide, and is the leading cause of chronic liver disease [1]. The prevalence of viral B infection is high in sub-Saharan Africa, where modes of transmission are predominantly perinatal and horizontal intra-familial in early childhood [4]. The natural history of hepatitis B virus infection is characterized by a diversity of clinical profiles, with acute hepatitis, chronic hepatitis and progression to complications such as cirrhosis and/or hepatocellular carcinoma (HCC) [3] [5]. WHO estimates that there will be 296 million chronic HBV carriers worldwide in 2019, with 900,000 deaths per year, and chronic hepatitis B is responsible for around 50% of hepatocellular carcinomas (HCC) worldwide [3]. Chronic B viral infection comprises different phases, including the inactive carriage phase. This phase is defined by B viremia (HBV DNA) ≤ 2000 IU/ml and strictly normal alanine aminotransferase (ALAT) levels for at least one year of follow-up in an HBeAg-negative patient. However, in the absence of liver fibrosis evaluation, it may be difficult to distinguish true inactive HBV carriers with HBeAg-negative chronic HBV from those with viral B reactivation. This difficulty exposes the patient to a high risk of progression to complications in the absence of anti-B viral treatment. According to several authors, the combination of B viremia below 2000 IU/ml and HBsAg below 1000 IU/ml is associated with a low

risk of liver fibrosis progression in HBeAg-negative patients in Caucasian and Asian populations [6]. The majority of chronic HBV carriers in black Africa are HBeAg-negative, often with normal adult transaminases [7] [8]. Major advances have been made in recent years in the early diagnosis of cirrhosis, and in the management of HBV-infected patients through the use of non-invasive tests.

The fibroscan, which measures hepatic elasticity, is a simple, rapid, non-invasive method developed in 2002 that can be used to assess hepatic fibrosis by measuring the degree of liver elasticity. There is a risk of cirrhosis or HCC in patients with detectable B viremia but <2000 with normal transaminases and significant fibrosis, hence the need to systematically measure the degree of hepatic elasticity in any patient with chronic HBV, hence the interest of our study.

2. Methodology

The Hepato-Gastroenterology Department and the Day Hospital Department of the HNN served as study settings. It was a prospective descriptive and analytical study from January 05 to November 30, 2023, a period of eleven (11) months. Our study included all patients who met the inclusion criteria, in particular, all clinically asymptomatic HBs Ag-positive patients over 18 years of age who underwent liver elasticity testing. The parameters studied were: sociodemographic data, history, circumstances of discovery, liver elasticity, biological data and treatment. A pre-established individual survey form was used to collect the data.

We used the fibroscan mini 430 to measure liver elasticity. The examination was carried out by a hepato-gastroenterologist who had received Fibroscan training. The median of ten liver elasticity measurements at the same point with an IQR of less than 30% was considered the valid measurement, and the absence of fibrosis or minimal fibrosis was defined by a value <7 Kpa, moderate fibrosis by a value between 7 and 10 Kpa, severe fibrosis by a value greater than 10 Kpa and the existence of cirrhosis by a value greater than 14 Kpa. Data was entered using Microsoft Word 2010, and SPSS 25.0 was used for data analysis. We used the Chi2 test to compare data, with a significant threshold if p-value <0.005.

For the conduct of the study, we received: an authorization from the Faculty of Health Sciences/UAM, an authorization from the HNN administration. The study was conducted with strict respect for patient anonymity.

Limitations and/or difficulties encountered during the study were mainly: the lack of financial means of the patients, the repetitive lack of reagents, which are always in shortage, preventing the realization of complementary examinations such as: viral load, transaminases, AFP, quantitative HBs Ag.

3. Results

In the course of our study, we registered 1,855 outpatients, including 398 symptomatic HBV carriers with a frequency of 21.45%. 60 cases met our inclusion criteria (3.23%). The mean age of patients was 35.63 years, with extremes of 18 and 70 years, and the age range [18 - 30 years] accounted for 45% (n = 27) (**Table 1**).

Table 1. Patient distribution by age group.

Age Groups	Actual (n)	Percentage (%)
18 - 30	27	45
30 - 45	18	30
45 - 60	12	20
>60	3	5
Total	60	100

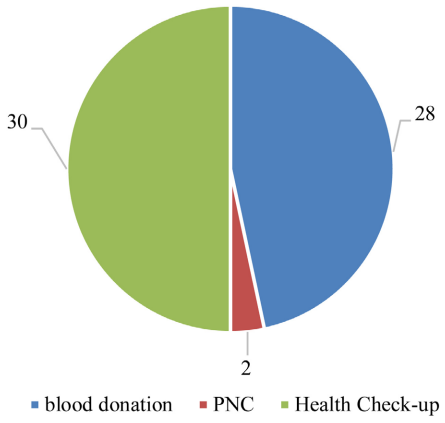
Males predominated, with a sex-ratio of 3.2. Our sample was composed of civil servants and farmers in 40% (n = 24) and pupils/students and housewives in 31.67% (n = 19). Our patients were married in 61.67% and had a higher level of education in 70% (Table 2).

Table 2. Distribution of patients by level of study.

Study Level	Actual (n)	Percentage (%)
Illiterate	1	1.67
Koranic school	1	1.67
Primary	11	18.33
Secondary	5	8.33
Superior	42	70

The antecedents found in our patients were jaundice, blood transfusion and a family history of liver disease in 8.33%, 6.67% and 6.67% of our patients respectively.

The circumstances in which HVB was discovered were the routine health check-up, followed by blood donation with 50% and 46.67% respectively (Figure 1).



*PNC: prenatal consultation.

Figure 1. Distribution of patients according to circumstances of discovery.

Of the 16 patients with quantitative AgHbs, 50% had a value greater than 1000 IU/ml (Table 3).

Table 3. Distribution of patients according to quantitative AgHbs results.

Quantitative AgHbs (UI/ml)	Actual (n)	Percentage (%)
500 - 1000	8	50
>1000	8	50
Total	16	100

HBe Ag negative in 93.33%. Co-infections were represented by one hepatitis C and one hepatitis delta. Cytolysis was present in 9.33% of our patients (n = 4). The mean ALT level was 39.87 IU/l.

The mean viral load was 10204383.7 IU/ml. A viral load greater than 2000 was found in 32.7% (n = 17) (Table 4).

Table 4. Distribution of patients according to viral load results.

HBV DNA (UI/ml)	Actual (n)	Percentage (%)
≤2000	35	67.3
>2000	17	32.7

Mean hepatic elasticity was 6.7 KPA. Fibrosis was significant in 25% (n = 15) of cases. It was moderate (F1 - F2) in 18.33% (n = 11) and advanced (F4) in 6.67% (n = 4) (Figure 2).

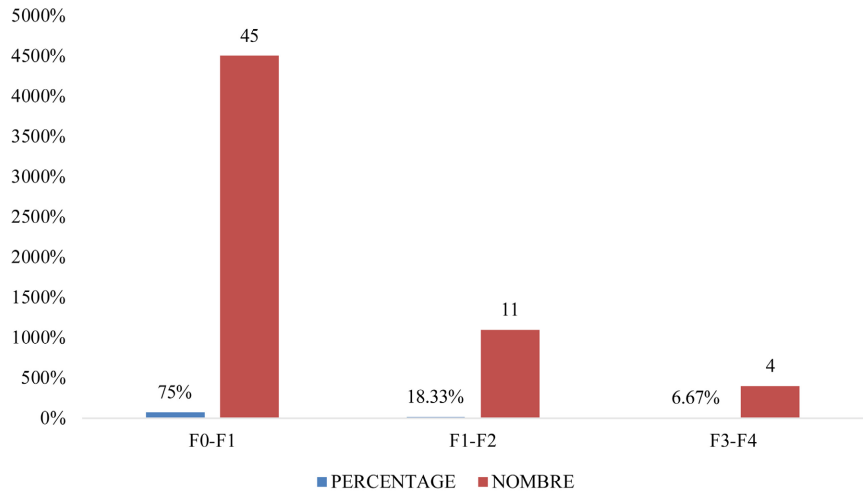


Figure 2. Distribution of patients according to fibroscan results.

Our patients were at the HBeAg-negative chronic hepatitis stage in 20.68% (n = 12) and at the HBeAg-positive chronic hepatitis stage in 1.72% (n = 1). Tenofovir-based antiviral therapy was prescribed in 13 patients (21.67%). The evolution of our patients under treatment was marked by biological improvement in 2

patients (15.38%), and Hbs seroconversion in 15.38% (n = 2). No cases of cirrhosis and/or hepatocellular carcinoma were recorded. Patients with quantitative Hbs antigenemia above 1000 IU/ml had a viral load above 2000 IU/ml in 50% (n = 4). There was no statistically significant relationship between B viral load and quantitative HBsAg (p = 0.3609). Among patients with an AgHbsQ greater than 1000, 66.67% had normal transaminases. Normal transaminases were found in 83.33% of patients with a B viral load greater than 2000 IU/ml. Advanced fibrosis was found in 11.76% of patients with a B viral load greater than 2000 IU/ml. There was no statistically significant relationship between B viral load and liver elasticity (p = 0.1494). No advanced fibrosis was found in patients with a quantitative HBsAg greater than 1000 IU/ml. There was no statistically significant relationship between quantitative HBsAg and liver elasticity (p = 0.1822).

4. Discussion and Comments

The in-hospital frequency of HBV in our study was 21.45%. This frequency is higher than that of Schweitzer A *et al.* in England in 2015 and Rachelle D in Mali in 2011, who found 11% and 13.97% respectively [5] [9]. This could be explained by the diversity of the study settings and the inequality of the samples.

This prevalence is considerable, as it is estimated that 15 to 40% of hepatitis B virus carriers will have a complication linked to this infection during their lifetime [10].

Viral hepatitis affects both sexes. A male predominance is observed in 76.70% of cases, giving a sex ratio of 3.2. This same predominance was found by de Diallo *et al.* in Senegal in 2018 and Traoré F *et al.* in Mali in 2020, who found 72.8% and 67% respectively [11] [12]. This predominance of males could be explained by men's lifestyle, which exposes them to risk factors more often than women (alcohol, tobacco, risky sexual behavior), and by their high participation in blood donations. The population was made up of young adults. The mean age was 35.63 years, with extremes of 18 and 70 years. Our results are comparable to those of Diallo *et al.* in Senegal in 2018 and those of Djieukam Toko D *et al.* in Madagascar in 2014, who found 33y and 35.11y, respectively [11] [13]. This could be explained by the youth of our populations. This high frequency of infection among young people merits particular attention in the fight against hepatitis B, as they constitute the productive and sexually active social stratum likely to transmit this virus.

Married people represented 61.67% of our patients. Our results are comparable to those of Lawson H in Niger in 2014, who found 72.6% [14]. This predominance of infection in married people can be explained by unprotected sexual intercourse, which is one of the risk factors, but also by their high level of participation in blood donations.

In our series, 4 patients (6.67%) had a family history of liver disease. Our results are comparable to those of Diallo *et al.* in Senegal in 2018, who found 3.7% [11]. A family history of liver disease is one of the risk factors for HBV infection. Follow-up and therapeutic indication must take this into account. HBV screening of

siblings and offspring of people with viral hepatitis B is important and should be systematic. The routine health check-up was the most common screening circumstance, followed by blood donation in 50% and 46.67% respectively. Our results differ from those of Lawson H in Niger in 2014, who found the health check-up and the prenatal check-up as the screening circumstance in 29.9% and 21%, respectively [14]. These 3 circumstances of discovery (health check-up, blood donation and antenatal check-up) are practically the same as in previous studies. This justifies the low-noise evolution of the disease. Indeed, most chronic carriers have few or no symptoms. As a result, the disease usually evolves silently, and is often discovered by chance [15] [16].

In 75% of our patients, hepatic elasticity was preserved. However, 25% had significant fibrosis, including 18.33% with moderate fibrosis and 6.67% with advanced fibrosis (F4). Our results are lower than those of Camara Toumin et al in Côte d'Ivoire in 2021, who found 38.9% [17]. This reflects the harmful consequences of late diagnosis of the condition, secondary to the often silent nature of viral hepatitis B. These patients were asymptomatic at the time of diagnosis, hence the importance of screening for early management. The consequences of this loss of elasticity are progression to cirrhosis with portal hypertension, hepatocellular insufficiency and hepatocellular carcinoma (HCC). Several studies have reported the association of hepatitis B virus with cirrhosis [7] [18]. Although cirrhosis is a precancerous state which favours the development of HCC, studies have also reported the role of this virus in determining the development of HCC [2] [7]. Our study population was predominantly composed of HBeAg-negative chronic carriers, with a frequency of 93.33%, compared with 3.33% of wild-type (HBeAg-positive) chronic carriers. Our results are similar to those of Lawson H in 2014, who found 90%. However, our results are higher than those of Camara Toumin et al in Côte d'Ivoire in 2021, who found 78%. This shows that the pre-c mutant virus is more frequent than the wild-type virus, but also by a prolonged duration of the immune tolerance breakdown phase, associated with a delay in HBe seroconversion, also justifying the severity of the liver lesions found. In our study 32.7% of our patients had a viral load greater than 2000 IU/ml. Our results are comparable to those of Abba Z in Niger in 2019, who reported 74.2% of viral loads above 2000 IU/ml in their studies [19]. Viral load is one of the parameters used to initiate antiviral treatment in all non-decompensated chronic HBV carriers and to assess the replicative capacity of HBV. According to the literature, not all patients with a viral load of less than 2000 IU/ml, compensated fibrosis and normal ALT are eligible for treatment.

Most of our patients (78.33%) had normal transaminases. However, 9.33% had elevated transaminases. Our results are lower than those of Lawson H in Niger in 2014, who reported elevated transaminases in 36%. This could be explained by the fact that HBV is not very symptomatic, even biologically. The normality of this marker does not rule out an active chronicity of hepatitis B. There is no correlation between the degree of fibrosis and the amount of quantitative AgHbs, between

liver elasticity and B viral load. This weak correlation has already been reported by the EASL 2017 recommendations [2] and also a study conducted by Rodriguez M in Spain. Indeed, it has been shown that a patient can develop cirrhosis and subsequently eliminate HBsAg, and conversely a patient can have an excessive amount of viral load without activity or fibrosis [19] [20]. Our patients were put on antiviral treatment based on Tenofovir diproxil fumarate (TDF) dosed at 300mg in 21.66% (n = 13). This same molecule has been used in several studies, such as those by Diallo et al in 2018 in Senegal, where out of 58 patients chronically infected with HBV, 55 patients were on TDF, i.e. 94.8%, and Abba Z in Niger in 2019, where TDF was used in all patients [11] [19].

The choice of Tenofovir diproxil fumarate could be explained by its potent antiviral activity, excellent tolerability for an indeterminate treatment duration, geographical and financial accessibility and the fact that it does not generate in vivo resistance, and is also recommended by the WHO [2]. Progression on TDF was marked by biological improvement in two patients. Two cases of HBsAg loss were noted during treatment. No cases of HCC were recorded during our follow-up.

5. Conclusion

Hepatitis B virus infection is a public health problem in our context. Our study population consisted of young adults, predominantly male. We recorded many patients with chronic hepatitis who benefited from antiviral treatment. There was no significant relationship between the degree of viremia, liver activity, and degree of fibrosis and quantitative HBs Ag. Fibroscan showed the absence of significant fibrosis in the majority of our patients, but also a non-negligible number of cirrhotic who benefited from tenofovir-based antiviral treatment.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Nigérien, G. (2024) De prise en charge des hépatites virales.

[2] European Association for the Study of the Liver (2017) EASL 2017 Clinical Practice Guideline on the Management of Hepatitis B Virus Infection. *Journal of Hepatology*, **67**, 370-398.

[3] World Gastroenterology Organization (2024) World Gastroenterology Organization (WGO) n.d. https://www.worldgastroenterology.org

[4] Inês, P., Horta-Vale, A., Cardoso, H. and Macedo, G. (2014) Hepatitis B Inactive Carriers: An Overlooked Population? *GE Portuguese Journal of Gastroenterology*, **21**, 241-249.

[5] Schweitzer, A., Horn, J., Mikolajczyk, R.T., Krause, G. and Ott, J.J. (2015) Estimations of Worldwide Prevalence of Chronic Hepatitis B Virus Infection: A Systematic Review of Data Published between 1965 and 2013. *The Lancet*, **386**, 1546-1555. https://doi.org/10.1016/s0140-6736(15)61412-x

[6] Leroy, P. (2024) Hépato-Gastro-Entérologie Chirurgie Digestive (3e éd.)—Les

Référentiels des Collèges—ELSEVIER/MASSON.
https://www.remede.org/documents/hepato-gastro-enterologie-colleges.html

[7] Chattin, J.L. (1994) Epidemiology of Hepatitis B Virus Infection in Tunisia. *Maghreb Medicine*, **278**, 15-18.

[8] Rodríguez, M., Buti, M., Esteban, R., Lens, S., Prieto, M., Suárez, E., *et al.* (2020) Consensus Document of the Spanish Association for Study of the Liver on the Treatment of Hepatitis B Virus Infection. *Gastroenterología y Hepatología*, **43**, 559-587. https://doi.org/10.1016/j.gastre.2020.03.008

[9] épouse Dakouo, R.D. (2011) Epidemiological and Semiological Profile of Hepatitis B Virus in an Urban Setting of Bamako. Faculty of Medicine, Pharmacy and Odonto-stomatology.

[10] Zoulim, F., Kay, A., Merle, P. and Trepo, C. (2006) Virology of Hepatitis B. *EMC* (*Elsevier SAS Paris*) *Hepatology*, **1**. 1-19.

[11] Diallo, S., Bassène, M.L., Gueye, M.N., Thioubou, M.A., Dia, D., Mbengue, M., *et al.* (2018) Hépatite virale B: Aspects cliniques, paracliniques et évolutifs dans le service d'Hépato Gastroentérologie de l'Hôpital Aristide Le Dantec: À propos de 728 cas. *Pan African Medical Journal*, **31**, 211-218. https://doi.org/10.11604/pamj.2018.31.82.14725

[12] Traoré, F. (2020) Knowledge, Attitudes and Practices of Interns on Hepatitis B Prevention at Point G University Hospital. Université des Sciences, des Techniques et des Technologies de Bamako.

[13] Djieukam, T.D. (2014) Prevalence of Hepatitis B and C Viral Serological Markers among Civil Servants in Madagascar. University of Antananarivo.

[14] Lawson, H. (2014) Chronic Viral Hepatitis B in Niger: Epidemiological, Clinical, Paraclinical and Therapeutic Aspects of 117 Cases Collected at Niamey National Hospital. Doctor of Medicine Thesis.

[15] Maiga, M.Y., Dembele, M., Diallo, F., Traore, H.A., Traore, A.K. and Guindo, A. (2002) Valeur diagnostique de l'endoscople digestive haute au cours de la cirrhose. *Acta Endoscopica*, **32**, 211-218. https://doi.org/10.1007/bf03016657

[16] Perret, J.l., Kombila, M.J.B., Demba, L.F., Boguikouma, J., Matton, T., *et al.* (2002) HBsAg and Hepatitis C Virus Antibodies in Complicated Liver Disease in Gabon: A Case-Control Study. *Gastroentérologie Clinique et Biologique*, **26**, 131-135.

[17] Camara, T., Kaba, I., Camara, M., Iv, B., Konan, M. and Toutou, T. (2021) Clinical and Paraclinical Aspects of Chronic Viral Hepatitis B at Treichville University Hospital. *The Journal of Medicine and Biomedical Science*, **22**, 16-19.

[18] Dembele, M., Maïga, I., Minta, D., Konate, A., Diarra, M., Sangare, D., *et al.* (2004) Study of HBs Antigen and Hepatitis C Virus Antibodies during Chronic Hepatopathy in Hospital Wards in Bamako, Mali. *Bulletin de la Societe de Pathologie Exotique*, **97**, 161-164.

[19] Abba, Z. (2019) The Importance of Viral Load in the Management of People Living with Hepatitis B Virus. Retro-Prospective Study about 296 Cases.

[20] Marcellin, P., Heathcote, E.J., Buti, M., Gane, E., de Man, R.A., Krastev, Z., *et al.* (2008) Tenofovir Disoproxil Fumarate versus Adefovir Dipivoxil for Chronic Hepatitis B. *New England Journal of Medicine*, **359**, 2442-2455. https://doi.org/10.1056/nejmoa0802878