Int. j. adv. multidisc. res. stud. 2024; 4(4):773-778

Received: 19-06-2024
Accepted: 29-07-2024

International Journal of Advanced Multidisciplinary Research and Studies

ISSN: 2583-049X

Clinical Trials Based on Aminoglucosides Against Viral Hepatitis B and C in Oriental Kasai (Democratic Republic of Congo)

[1] Kazadi Hyanda Espoir, [2] Bukasa Tshilonda Jean Christophe, [3] Tshieyi Mubambu Willy, [4] Musasa Tshibangu Jean Claude, [5] Matanda Tshibangu Jean Marie, [6] Kangudia Mukadi Gabriel, [7] Beya Beya Laurent, [8] Ngoyi Kadil Roger, [9] Mbenga Kasela Leon, [10] Ciamala Mukendi Paul

[1] Higher Institute of Medical Techniques of Kalenda, Laboratory Techniques Section, Medical Biology Option, Kalenda Baya, Democratic Republic of the Congo

[2] Higher Institute of Medical Techniques of Mbuji-Mayi, Nursing Science Section, Nursing Education and Administration Option, Mbuji-Mayi, Democratic Republic of the Congo

[3, 4] Higher Institute of Medical Techniques of Kalenda, Nursing Sciences Section, Teaching and Administration Option in Nursing, Kalenda Baya, Democratic Republic of the Congo

[5, 8] Higher Institute of Medical Techniques of Kamiji, Nursing Sciences Section, Nursing Education and Administration Option, Kamiji, Democratic Republic of the Congo

[6, 7] Higher Institute of Medical Techniques of Mbujimayi, Laboratory Techniques Section, Medical Biology Option, Mbujimayi, Democratic Republic of the Congo

[10] Higher Institute of Medical Techniques of Mbujimayi, Nursing Sciences Section, Teaching and Administration Option in Nursing, Democratic Republic of the Congo

[9] Higher Institute of Medical Techniques of Tshilengi Nursing Sciences Section, Teaching and Administration option in Nursing, Tshilengi, Democratic Republic of the Congo

DOI: https://doi.org/10.62225/2583049X.2024.4.4.3106
Corresponding Author: **Kazadi Hyanda Espoir**

Abstract

Hepatitis A, B and C are inflammations of the liver caused by a virus. In most cases, no treatment is necessary. Depending on the type of virus, hepatitis usually clears up on its own within two to six months after infection.

The objective of the present study was to contribute to improving the health of communities suffering from viral hepatitis B and C in the population through a clinical approach based on Aminoglucosides.

Aminoglucosides are an alternative to consider in the treatment of viral hepatitis B and C because bivariate statistical analyzes have proven that there is a very significant difference between variables studied before and after treatment.

Keywords: Therapeutic Trials, Hepatitis, Aminoglucosides

1. Introduction

Viral hepatitis are pathologies that affect the liver; they are characterized in clinically expressed forms by the onset of jaundice, ascites, gastritis, significant anorexia associated with asthenia, edema of the lower limbs.; in their biological forms, they are characterized by the increase in GOT (Glutamo-oxalo-acetate transaminase) and GPT (Glutamo pyruvate transaminase) transaminases, the increase in direct bilirubin and rarely the increase in urea and blood sugar. creatinine. In biological forms, serology by immunochromatography using reactive strips proves positive in both cases. (WHO, 2022) [13].

They are caused by hepatitis B viruses (hepatotropic virus, capable of causing acute infections, acute liver failure (ALF) and chronic infections in humans. It belongs to the *Hepadnaviridae family.*, family of enveloped viruses whose genetic information is carried by a molecule of partially double-stranded relaxed circular deoxyribonucleic acid (dsDNA), approximately 3,200 base pairs long) and C (hepatitis virus non A and not B" of the Arenaviridae family. This RNA genome has an organization close to that of flaviviruses with 9500 nucleotides (9.5 kbases), non-coding 5‟ and 3‟ ends, and starting from the 5‟ genes for capsid (C), envelope (E1 and E2) and non-structural proteins (NS2 to NS5), the NS3 protein being a viral protease and the NS5

protein being the RNA-dependent RNA polymerase).
Being enveloped viruses, their penetration into the host cell (hepatocyte) is by endocytosis, replication continues depending on the nature of the viral genome, one is an RNA virus (HCV) and the other is a DNA virus (HBSAg). This replication results in inducing the cytopathic effect which can cause either hepatocellular carcinoma or hepatic cirrhosis (Pierre and Marie Curie, 2017) [14].
Many studies indicate that HBV and HCV are important causes of cirrhosis and hepatocellular carcinoma. The prevalence of HBV and HCV infection in patients with cirrhosis or hepatocellular carcinoma varies between countries but generally reflects the variable prevalence of these infections in the population. In Egypt, where HCV prevalence is high, most patients with chronic liver disease initially have HCV infection that causes their disease. In contrast, HBV infection is frequently found in patients with cirrhosis or hepatocellular carcinoma in other African countries. The WHO estimates that more than 75% of cirrhosis and hepatocellular carcinoma in the population are attributable to chronic infection with HBV or HCV. Beyond this, we note that hepatitis has a high prevalence in developing and low-income countries, in this case the Democratic Republic of Congo. These data represent a cry of distress and require activity allowing early detection in order to spare the population from the related consequences by initiating treatment. (WHO, 2022) [13].
As part of this study, we would like to present the results of the effectiveness of the clinical trial based on aminoglucosides as an alternative against viral hepatitis B and C in a curative context. This is a scientific approach started in 2015 prospectively on a cohort made up of 112 patients selected in a non-probabilistic manner for convenience, from whom the laboratory investigations were carried out before adherence to treatment and one month after the last dose of treatment for the purpose of evaluation. This included serology by immunochromatography using strips which turned out to be positive and which allowed the selection of patients.
These patients were subsequently subjected to aminoglucosides in strict compliance with the protocol for a maximum period of five days with repeat doses within two months. It will thus be accompanied by an evaluation by execution of the different assessments to identify the variation using the chi square using SPSS software. There were considerable variations due to the treatment and exactly attesting to the effectiveness of the latter, without forgetting the therapeutic failures recorded which amounted to 5.4% or 6 failures out of 112 trials. These failures could possibly be due to the fact that these patients were consulted while their condition was in a very advanced clinical phase; some already had hepatocellular carcinoma and some others had liver cirrhosis, which made their outcome fatal despite the treatment initiated.
In the group of patients tested positive and submitted to the clinical trial under study, we noted a positive relationship in terms of recovery in the latter, revealed by a statistically significant variation in the variables studied, notably serology (HBV and HCV) and biochemistry (SGPT) as well as some clinical signs. This allowed us to put forward a hypothesis according to which the clinical trial based on Aminoglucosides responds better in the treatment of viral hepatitis B and C.

2. Method and Material

To carry out these clinical trials with aminoglucosides, we opted for a prospective cohort survey method. In this approach, we followed a cohort consisting of 112 cases tested positive for viral hepatitis including 90 cases for hepatitis B and 22 cases for hepatitis C. This sample was selected in a non-probabilistic manner for convenience and consisted of to take all cases whose biological diagnosis was made during premarital examinations and blood donation as well as all cases with pain in the right hypochondrium, jaundice and chronic gastritis.
This method was supported by the experimental analysis technique based on serological diagnosis by reagent strip by doubling dilution for HBV and simple serology for HCV, as well as the determination of transaminases GPT, GOT and finally urea. and creatinine.
For the analysis of these data, we used chi square as a statistical test.
Those materials were opted in this case.

- ✓ Spectrophometer Memmert 1250.
- ✓ Centrifuge model As023.
- ✓ Pipetting bulb or Micropupette with disposable tips 50-500 micromotor
- ✓ Incubator XP 60s.

Techniques
Serology
a) HBsAg serology
❖ **Principle**
The Determine® HBsAg test from ALERE uses a sandwich-type reaction for the detection of HBsAg surface antigen.
The sample is introduced into the deposition zone and migrates by capillary action along the strip. If the sample contains HBs antigens, these bind to the conjugate (mouse anti-HBs antibodies fixed to selenium colloids) at the migration zone to form antigenantibody-selenium immune complexes (Ag-Ac-Se).
These complexes migrate towards the reading zone and bind to the mouse monoclonal anti-HBs antibodies immobilized at the patient window of the strip, inducing the appearance of a red colored band.
The control band is obtained by the reaction between the anti-selenium antibodies immobilized at the control window which capture the free deselenium particles (not attached to the mouse anti-HBs Ab).
❖ **Operating mode**

- **Remove the plastic protection from each test.**
- **For serum or plasma samples:**
  - ✓ Distribute 50 µl of sample (using a precision pipette) to the sample deposit area,
  - ✓ Wait at least 15 minutes (maximum: 24 hours) and read the result.
- **For whole blood samples (venous puncture):**
  - ✓ distribute 50 µl of sample (using a precision pipette) onto the sample deposit area.
  - ✓ Wait one minute, then distribute a drop of migration buffer onto the sample deposit area,
  - ✓ Wait at least 15 minutes (maximum: 24 hours) and read the result.- For whole blood samples (fingertip):
  - ✓ Dispense 50 µl of sample (with a capillary tube containing EDTA) onto the sample deposition area.

- ✓ Wait for the blood to be absorbed by the deposition area, then distribute a drop of migration buffer onto the sample deposition area.
- ✓ wait at least 15 minutes and read the result. Stable coloring for 24 hours.

❖ **Results and interpretation**

▪ **Positive (two bars)**

The red bars appear in the control window (labeled "Control") and the patient window (labeled "Patient") on the strip. Any red color visible in the patient window should be interpreted as a positive result.

▪ **Negative (one bar)**

A red bar appears in the control window (labeled "Control"), the red bar of the patient window (labeled "Patient") does not appear on the strip.

▪ **Not Valid (no bar)**

If the red bar does not appear in the test strip control window and even if a red bar appears in the test strip patient window, the result is invalid and the test must be repeated.

Dilution

b) HCV serology

Biochemistry

1) Dosage of transaminases or determination of enzymatic activities of transaminases

A. Aspartateamino-transferase (**ASAT**), formerly known as Serum Glutamo-Oxaloacetate Transferase (**SGOT**).

❖ **Principle**

Kinetic determination of GOT activity after the reaction of the apoenzyme with pyridoxal-5' phosphate according to the reaction:

L-Aspartate + α-Oxoglutarate ⇌ L-glutamate + Oxaloacetate

Oxaloacetate + NADH + H+ $\xrightarrow{MDH}$ L-malate + NAD+

❖ **Determination of serum GOT level**

Reagents	
R1	800 Ul
R2	200 Ul
Sample	100 Ul

Mix, introduce into the device and measure the OD per minute for 3 minutes at the wavelength of 340 nm and then interpret the result in international units per liter (IU/L).

❖ **Usual values in serum**

▪ Men: 14 - 50 U/l.

▪ Women: 11 - 32 U/l

B. Alanine aminotransferase (**ALT**)

❖ **Principle**

Kinetic determination of GOT activity after the reaction of the apoenzyme with pyridoxal-5' phosphate according to the reaction:

L-alanine + α-oxoglutarate $\xrightarrow{GPT}$ Pyruvate + glutamate

Pyruvate + NADH+ H+ $\xrightarrow{LDH}$ Lactate+ NAD+

❖ **Determination of serum GPT level**

Reagents	
R1	800 uL
R2	200UL
Sample	100 uL

Mix, introduce into the device and measure the OD per minute for 3 minutes at the wavelength of 340 nm and then interpret the result in international units per liter (IU/L).

❖ **Usual values in serum**

▪ Men: 11 - 60 U/l;

▪ Women: 9 - 36 U/l.

2) Urea dosage

a. Berthelot method: Enzymatic and colorimetric test

▪ **Principle**

Urea is hydrolyzed to ammonia and CO_2. Ammonia reacts with salicylate and hypochlorite to form green indophenol. The color of this is proportional to the concentration of urea.

Urease

$$Urea + H_2O \longrightarrow CO_2 + NH_3$$

▪ **Composition of reagents:**

Reagent 1	Phosphate pH 6.7……..…50 mmol /l EDTA……..2 mmol/l Sodium salycilate…….….60 mmol/l Sodium nitroprusiate…….3, 2 mmol/l
Reagent 2	Sodium Hypochlorite ……..140 mmol /l Sodium hydroxide……..…150 mmol/l
Reagent 3	Urease……. 30000 U/l
Reagent 4	Urea……...50 mg/dl

▪ **Preparation and stability**

Dissolve the contents of vial R3 in buffer R1. This working solution is stable for 4 weeks at 2-8°C or one week at room temperature. The R2 reagent is ready to use.

▪ **Sample**

Serum, heparinized plasma

Urine diluted 1:50

▪ **Procedure**

	White	Standard	Standard
R1+R3	1.00	1.00	1.00
Standard	-	10µl	-
Sample	-	-	10µl

Mix and incubate at 37°C for 5 minutes or 10 minutes at 20-25°C. Then add reagent 2.

3) Creatinine dosage Jaffé kinetic method

a. Principle

Creatinine forms an orange complex in basic picrate solutions as described by Jaffé. The difference in absorbance, at predetermined times during the conversion, is proportional to the concentration of creatinine in the sample.

b. Composition of reagents:

Reagent 1 picric acid	Picrate solution ……..…17.5 mmol/l
Reagent 2 Alkaline reagent	Sodium hydroxide……...0.29 mol/l
Standard	Aqueous creatinine……..……2 mg/dl

c. Preparation and stability

The working solution is obtained by mixing proportionally 1:1 the reagents R1 picric reagent and R2 alkaline reagent. This solution is stable for 10 days between 15-25°C.

d. Samples

Serum, heparin plasma Creatinine in serum or plasma remains stable for at least 24 hours at 2-8°C.

e. Procedure

	Sample	Standard	White
Sample	100µl	-	-
Standard	-	100µl	-
Working solution	1000ml	1000ml	1000ml

Mix and start the timer. Read absorbance 1 (abs 1) after 30 seconds and absorbance 2 (abs 2)2 after 90 seconds of sample addition at a wavelength of 492 nm.

f. Calculation

Creatinine (mg/dl) = (Abs. Sample / Abs. Stand.) x 2 (Standard Conc.)

g. Reference values

Men: 0.7-1.4 mg/dl
Women: 0.6-1.1 mg/dl

3. Clinical trial results

3.1 Results of descriptive analyzes

Table 1: General profile of cases

Category	Number N = 112	Frequency
Hepatitis B	90	80.4
Hepatitis C	22	19.6
Coinfection B and C	3	2.6
HIV/B coinfection	0	00
HIV/C co-infection	2	2.6

Analysis of this table shows that the majority of cases which were the subject of the clinical trial with aminoglucosides were affected by viral hepatitis B with 80.4% of cases, followed by 19.6% affected by Hepatitis C; HIV/HCV coinfection represented only 2.6%.

Table 2: Presentation of cases by sex

Sex	Effective	Frequency
Male	106	94.6
Feminine	6	5.4
Total	112	100

This table indicates that the majority of cases were male subjects with 94.6%.

Table 3: Presentation of cases according to age

Age category (years)	Effective	Frequency
Children < 18	0	00
Adults (19 and 68)	108	96.4
Old people > 68	04	3.6
Total	112	100

In light of this table, it appears that the majority of cases were adults with 96.4% of cases.

Table 4: Presentation of average age by sex

Sex	Mean age ± standard deviation	Total/Frequency
Male	25±5	106 (94.6)
Feminine	28±3	6 (5.4)
Total	26.5	112

Looking at this table, it appears that the patients seen for hepatitis were on average 26 years old.

Table 5: Presentation of cases according to stage of disease

Stadiums	Effective	%
Acute hepatitis	2	1.8
Chronic hepatitis	108	96.4
Cirrhotic hepatitis (Cirrhosis)	1	0.9
Hepatocellular carcinoma	1	0.9
Total	112	100

Analysis of this table shows that the majority of cases, i.e. 96.4%, presented chronic hepatitis, followed by the minority who presented acute hepatitis with i.e. 1.8%.

Table 6: Presentation of cases according to diagnostic circumstances

Diagnostic circumstances	Effective	%
Right hypochondrium pain	3	2.7
Blood donation	27	24.1
Premarital exams	80	71.4
Systematic screening	2	1.8
Total	112	100

Through these results, we note that most cases, i.e. 71.4%, were diagnosed during premarital examinations, followed by 22.3% at the time of blood donation compared to 2.7% who showed pain at the level of the right hypochondrium.

Table 7: Presentation of cases according to clinical signs at admission

Clinical signs	Number=112	%
Edema	19	16.9
Ascites	07	1.8
Jaundice	05	4.5
Anorexia	106	94.6
Asthenia	106	94.6
Weight loss	91	81.3

In light of this table, we note that the majority of cases presented anorexia and physical asthenia with 94.6%, followed by 46.4% who presented jaundice against 1.8% who presented. ascites.

Table 8: Presentation of clinical trial results before and after treatment

Variables		Before treatment			After treatment			χ^2	*P*	*s*
			N=112	%		N=112	%			
Serology	HBsAg Dilution	Normal (<1/80)	2	1.9	Normal (<1/80)	85	75.9	**153**	**0.001**	****
		Pathological (≥ 1/80)	88	78.6	Pathological (≥ 1/80)	5	4.5			
	HCV	Negative	0	0	Negative	20	17.8	**36**	**0.012**	***
		Positive	22	19.4	Positive	2	1.8			
Biochemistry	Urea (mg%)	Normal (< 35)	98	87.5	Normal (< 35)	106	94.6	3.5	0.254	NS
		Pathological (≥ 35)	14	12.5	Pathological (≥ 35)	06	5.4			
	Creatinine (mg%)	Normal (<1.4)	102	91.1	Normal (<1.4)	106	94.6	1.08	0.352	NS

		Pathological (≥ 1.4)	10	8.9	Pathological (≥ 1.4)	06	5.4			
	SGPT (UI/L)	Normal (< 28)	08	7.1	Normal (< 28)	106	94.6	**179**	**0.001**	****
		Pathological (≥ 28)	104	92.9	Pathological (≥ 28)	06	5.4			
	SGOT (UI/L)	Normal (< 30)	98	87.5	Normal (< 30)	106	94.6	3.4	0.298	NS
		Pathological (≥ 30)	14	12.5	Pathological (≥ 30)	06	5.4			
Clinics	Edema	Yes	19	16.9	Yes	02	1.8	**156**	**0.001**	****
		No	93	83.1	No	110	88.4			
	Ascites	Yes	07	1.8	Yes	02	1.8	2.9	0.125	NS
		No	105	98.2	No	110	98.2			
	Jaundice	Yes	05	46.4	Yes	02	41.1	1.32	0.221	NS
		No	107	53.4	No	110	58.9			
	Anorexia	Yes	106	94.6	Yes	02	1.8	**192**	**0.001**	****
		No	06	5.4	No	110	98.2			
	Physical asthenia	Yes	106	94.6	Yes	03	2.3	**188**	**0.001**	****
		No	06	5.4	No	109	97.3			
	Weight loss	Yes	91	81.3	Yes	2	1.8	**145**	**0.001**	****
		No	21	18.3	No	110	98.2			

Analysis of this table shows that of all the variables studied, only serology (HBsAg, HCV), SGPT transaminases, physical asthenia, anorexia, edema and weight loss experienced a variation, the difference of which is statistically significant; with HBsAg and HCV (chi square: 153, p=0.001 and 36, p=0.001) and subsequently: 179, p=0.001 for SGPT, 192, p=0.001 for anorexia, 188, p =0.001 for physical asthenia and 145, p=0.001 for weight loss. These results sufficiently explain the impact of clinical trials based on aminoglucosides on the treatment of viral hepatitis B and C.

Table 9: Presentation of the results according to the effects observed during treatment

Effects observed	Number = 112	Frequency
Physical asthenia	5	4.5
Anorexia	9	8
Vomiting	2	1.8
Nausea	3	2.8
Polyuria	108	96.4
Polyphagia	98	87.5
Polydipsia	102	91.1
Diarrhea	2	1.8

Looking at this table, it appears that the majority of cases, i.e. 96.4%, presented polyuria, followed by 91.1% who presented polydipsia against 1.8% who presented diarrhea and vomiting.

Table 10: Presentation of the cure rate

Assessment	Effective	Frequency
Healing	106	94.6
Therapeutic failure	06	5.4
Total	112	100

In light of this table, it appears that clinical trials based on aminoglucosides presented a cure rate of 94.6% and a therapeutic failure of 5.4%.

4. Discussion

The analysis of Table 1 shows that the majority of cases which were the subject of the clinical trial with aminoglucosides were suffering from viral hepatitis B with 80.4% of cases, followed by 19.6% suffering from Hepatitis C; HIV/HCV coinfection represented only 2.6%. Our results corroborate those of Mamadou K *et al*, 2021, who found that 30% of the world population was affected by hepatitis B compared to 3% affected by hepatitis C. These results offer no explanation for this disproportionately, however, it is hepatitis B for which a vaccine exists.

Table 2 indicates that the majority of cases were male subjects with 94.6% and in light of Table 3, it appears that the majority were adults with 96.4% of cases, aged on average 26 years (Table 4).

The analysis of Table 5 shows that the majority of cases, i.e. 96.4%, presented chronic hepatitis, followed by the minority who presented acute hepatitis with i.e. 1.8%. Our results are similar to those of Mamadou K, cited above, who found that the majority of cases presented chronic hepatitis. According to Marc Gentillini in his 2002 edition, most cases of hepatitis present in chronic form and 90% of cases recover spontaneously. On the other hand, according to WHO 2021, people chronically infected with HBV have a higher risk of liver cirrhosis and hepatocellular carcinoma. The WHO estimates that approximately 600,000 people die each year from chronic liver disease linked to HBV. Without integration of hepatitis B vaccine into the EPI, it was estimated that approximately 100 000 people in each birth cohort in the Region would die from HBV-related liver disease or carcinoma hepatocellular.

Furthermore, there is no specific estimate of the HCV disease burden. However, if left untreated, 14 to 45% of patients infected with HCV develop chronic liver disease or cirrhosis 20 years after the onset of infection.

Through the results in Table 6, we note that most of the cases, i.e. 71.4%, were diagnosed during premarital examinations, followed by 22.3% at the time of blood donation compared to 2.7% who showed pain. at the level of the right hypochondrium. Screening for hepatitis B has been made compulsory in blood donors since December 1971 and for hepatitis C since 1990, so in 2012, among 383,000 new donors in France, 272 were found positive for HBsAg with i.e. 0.07% and 129 for HCV i.e. 0.03% **(HAS, 2014)** [7].

According to Nonon K and All 2018, the seroprevalence was respectively 3.9% for hepatitis B and 0.7% for hepatitis C among blood donors in the city of Kolwezi (Democratic Republic of Congo), of these results, a high seroprevalence was noted in the age group of 20 to 45 years, i.e. 4.2%, followed by those over 45 years of age, i.e. 2.3% and this difference was statistically significant (p = 0.047).

Moreover, it is since 1992 that screening for hepatitis B and C has been compulsory in premarital examinations, in order to avoid the vertical transmission of these diseases. In Algiers, in 2014 A. Nebab found 0.5% seropositivity to both viruses during premarital examinations. The prevalence of hepatitis B being 0.2% and that of hepatitis C 0.3%.

In light of Table 7, we note that the majority of cases presented anorexia and physical asthenia with 94.6%, followed by 46.4% who presented jaundice against 1.8% who presented. ascites. These results agree with the theory according to which viral hepatitis are pathologies which affect the liver, they are characterized in clinically expressed

forms by the onset of jaundice, ascites, gastritis, significant anorexia. associated with asthenia and edema of the lower limbs.

The analysis of Table 8 shows that of all the variables studied, only serology (HBsAg, HCV), SGPT transaminases, physical asthenia, anorexia, edema and weight loss experienced a variation, the difference of which is statistically significant; with HBsAg and HCV (chi square: 153, p=0.001 and 36, p=0.001) and subsequently: 179, p=0.001 for SGPT, 192, p=0.001 for anorexia, 188, p =0.001 for physical asthenia and 145, p=0.001 for weight loss. These results sufficiently explain the impact of aminoglucosides in the treatment of viral hepatitis B and C. Compared to transaminases, the results demonstrated that before treatment, their level was very high, which explained the degree of severity of trauma. inflammatory and infectious due to the virus on the liver. From their cytolytic effects, these viruses have the possibility of lysing the infected cell which is the hepatocyte, consequently its contents will be released into the blood circulation in this case the GPT transaminases which will see their serum value increased. This increase is therefore a reflection of the serious state of the already diseased liver.

In the absence of treatment and in states of immunodeficiency, the liver has two outcomes: Hepatocellular carcinoma and cirrhosis. The liver being endowed with a property of cellular self-regeneration, these viruses being oncogenic will induce a hepatocellular carcinoma by deactivation of the cytokines which are involved in the control of replacement (mitosis) of infected liver cells destroyed by apoptosis; consequently, there will be a disordered proliferation of cells. On the other hand, in certain states, chronic inflammation of the liver following the presence of the virus destroys hepatocytes creating liver fibrosis, newly formed scar tissue replaces the liver cells damaged during the inflammation. This fibrous tissue surrounds abnormal nodules formed by the uncontrolled regeneration of destroyed liver cells; cirrhosis sets in and cannot regress. In both cases, there will be an increase in the level of serum transaminases, particularly SGPT.

As part of this clinical trial using aminoglucosides, we noted a very significant variation in parameters considered to be strong signals during hepatitis disease (GPT transaminase level, HCV, HBV serology and certain clinical signs see Table 8) after administration of the protocol. This explains why the molecule acts directly on the virus by binding to the 30 S subunit of ribosomes. This interaction interferes with the translation of messenger RNAs by inducing errors in the decoding of codons; the virus is blocked by lack of production of early phase proteins. Thus, the infected cell receives a fratricidal signal and dies by apoptosis according to the infectious self theory. At the same time we are witnessing a reduction in viremia and a reestablishment of biological parameters that were disrupted during the infection.

Looking at Table 9, it appears that the majority of cases, i.e. 96.4%, presented polyuria, followed by 91.1% who presented polydipsia against 1.8% who presented diarrhea and vomiting. This is explained by the fact that the molecules based on this protocol (Aminoglucosides) are eliminated intact through the kidneys, which induces intense thirst and equally intense diuresis.

Table 10 indicates that clinical trials based on aminoglucosides presented a cure rate of 94.6% and a therapeutic failure of 5.4%. Compared to these results, it appears that late adherence to treatment as well as late diagnosis were major factors which induced therapeutic failure.

5. Conclusion

This was a scientific approach begun in 2015 prospectively on a cohort made up of 112 patients selected in a non-probabilistic manner for convenience, from whom the laboratory investigations were carried out before adherence to treatment and one month after the last dose of treatment for the purpose of evaluation. This included serology by immunochromatography using strips which turned out to be positive and which allowed the selection of patients.

Aminoglucosides are an alternative to consider in the treatment of viral hepatitis B and C because bivariate statistical analyzes have proven that there is a very significant difference between variables studied before and after treatment.

6. References

1. Nebab A. Prevalence and risk factors for transmission of viral hepatitis B and C among couples before marriage, Wilaya, Algiers, 2014.
2. Amuli Jiwe, *et al*. Scientific research methodology in care and health Volume I, GALIMAGE 103/2013, Kinshasa, DRC, 2013.
3. Anne DECOSTER, Jean-Claude Lemahieu, Eric Dehecq, Philippe Gontier, Marc Duhamel. Principles of virus classification. Link: http://anne.decoster.free.fr/d1viro/vgclass.html.
4. Daniel L Hartl, Elisabeth W Jones. Genetics: The main principles. Dunod, 3rd edition, 2003, 314.
5. François DABIS, *et al*. Epidemiology of intervention, Arnette, Paris, 1992.
6. Herbein G. Definition, structure, and classification of viruses, 2004, 13.
7. HAS. Pregnancy project, information, prevention messages, France, 2014.
8. https://www.vertex42.com/ExcelTemplates/simple-gantt-chart.html
9. Jean-Claude CALLEN. Cellular biology: From molecules to organisms. Dunod, 2nd edition, 2005, 25.
10. Jean-Marie HURAUX. Virology. Link: http://www.chups.jussieu.fr/polys/viro/poly/POLY.Chp.1.2.html.
11. Mamadou K, *et al*.
12. Nonon K, All. Seroprevalence of hepatitis B and C among blood donors in Kolwezi, DRC, RIC Journal. 2018; 2(1).
13. WHO. Prevalence of viral hepatitis B and C, Geneva, 2022, 56.
14. PIERRE ET MARIE CURIE. Virology and systematics of viruses, University of Louvain, 2017, 154.
15. PNTS. National Blood Transfusion Program, Kasai Oriental, 2022.
16. Prescott, Harley, Klein. Microbiology. De Boeck, 2nd edition, 2003, 369.
17. Prescott, Harley, Klein. Microbiology. De Boeck, 2nd edition, 2003, 377.
18. Prescott, Harley, Klein. Microbiology. De Boeck, 2nd edition, 2003, 378.
19. Prescott, Harley, Klein. Microbiology. De Boeck, 2nd edition, 2003, 21.