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Biochemical, Hematological, and Hepato-renal Study of Sendai Virus Infection in Wistar Rats

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Abstract: **Sendai virus (SeV) infection in Wistar rats was studied to evaluate its impact on hematological, biochemical, and hepato-renal parameters. The study aimed to understand the systemic effects of SeV infection beyond respiratory symptoms. Wistar rats were infected with SeV, and blood samples were collected at various time points. Hematological parameters, including red blood cell count, hemoglobin, hematocrit, platelet count, and white blood cell count, were analyzed. Biochemical parameters, such as liver enzymes (AST, ALT, ALP), total protein, total bilirubin, urea, and creatinine, were assessed to evaluate liver and kidney function. Results indicated significant alterations in hematological parameters, including reduced red blood cell count and hemoglobin, suggesting anemia. Liver function tests showed elevated levels of liver enzymes, decreased total protein, and increased bilirubin, indicating liver damage. Kidney function tests revealed increased levels of urea and creatinine, suggesting renal impairment. These findings highlight the systemic impact of SeV infection, extending beyond the respiratory tract to affect other vital organs. Understanding these effects is crucial for developing effective therapeutic interventions.**

Keywords: **Sendai virus, Wistar rats, viral infection, Hematological parameters and hepato-renal parameters.**

1. INTRODUCTION

The immune system is the body's basic defense against diseases (Marshall et al., 2018), such as viruses. Following viral infection, the immune system responds through innate and adaptive reactions, coordinating to eradicate the virus and prevent future infection (Li et al., 2020; Heim & Thimme, 2014).

Respiratory viral infections, such as those caused by the Sendai virus, are especially worrying due to their ease of transmission and the possibility of extensive epidemics (Mackenzie & Smith, 2020). These infections can cause symptoms ranging from moderate to severe, including pneumonia and Acute Respiratory Distress Syndrome (ARDS), which are potentially fatal. Long-term implications include chronic respiratory diseases, cardiovascular disease, and an increased risk of secondary infections (Mackenzie & Smith, 2020).

The human respiratory syncytial virus (RSV) is one of the most prevalent viruses that infect children globally, and it is increasingly recognized as a significant pathogen in adults, particularly the elderly and upper respiratory infections are the most common clinical situation associated with RSV infection (Jainet al., 2023).

Sendai virus (SeV) is a murine parainfluenza virus that has structural and antigenic similarities to human RSV. It is extensively used as an animal model to research RSV infection since it can elicit similar clinical alterations in the respiratory tract of rodents (Russell & Hurwitz, 2016; Russell & Hurwitz, 2021).

Animal research is regarded as an important step in the advancement of biomedical science. Animal models have a crucial role in studying disease physiopathology and developing new treatments, despite ethical concerns (Domínguez-Oliva et al., 2023). Wistar rats are often used as experimental animals in biomedical research. Their vulnerability to viral infections, combined with their physiological similarities to humans, make them a suitable model for studying the pathogenesis of respiratory viruses such as SeV (Bryda, 2013).

Viral infections can affect numerous organ systems, not just the respiratory tract (Kopańska et al., 2022). Common repercussions of severe viral infections include liver and renal failure. These organs perform critical functions in detoxification, metabolism, and electrolyte regulation. Understanding the consequences of viral infections on these organs is vital for establishing successful therapeutic strategies (Zhao et al., 2022).

In this study, we sought to evaluate the biochemical, hematological, and hepato-renal manifestation of SeV infection in Wistar rats. This was accomplished by assessing changes in several blood parameters.

Research Questions and Hypotheses

1. What are the hematological changes induced by SeV infection in Wistar rats?

2. How does SeV infection affect liver function in Wistar rats?

3. What are the effects of SeV infection on kidney function in Wistar rats?

Hypotheses:

1. SeV infection will lead to significant alterations in hematological parameters.

2. SeV infection will induce liver damage, as evidenced by elevated levels of liver enzymes

3. SeV infection will cause kidney damage, as indicated by increased levels of kidney function markers such as blood urea nitrogen (BUN) and creatinine.

2. METHODOLOGY

Animal Models and sample size

In this study, wistar rats were selected as the animal model. The sample size was determined based on statistical power calculations (Power method), ensuring that it included a sufficient number of rats to provide statistically meaningful results. Wister rats were chosen because they are commonly used in scientific research, and their physiology and response to treatments are well-documented, making them a suitable model for this study.

Ethical Clearance

All procedures in this study followed the guiding principles for animal research of the University of Port Harcourt Research Ethics Committee. The animals were housed in standard wooden cages maintained at a comfortable ambient temperature.

Procurement of Animals

Forty (40) adult male Wistar rats weighing 180–200g were procured from the Animal House of the Department of Pharmacology, Faculty of Basic Clinical Sciences, University of Port Harcourt. The rats served as the animal model for the study. The rats were housed in clean, disinfected wooden cages with sawdust as bedding. The animal house provided a controlled environment with a 12-hour light/dark cycle, 50–60% humidity, and a temperature of roughly 30°C. These conditions were maintained throughout the acclimatization and experimental periods. The rats were allowed to acclimatize for two weeks in the animal house. During this period, they had free access to clean water and standard animal feed. This acclimatization period ensured that the rats adapted to their new environment and stabilized before the commencement of the experiment.

Inducing the rats with Sendai virus

Sendai virus was procured from Nigeria institute of Research, Yaba, Lagos. Wistar rats were anesthetized using ketamine-xylazine to minimize stress and pain during injection of the virus. The virus was administered through the tail vein using a small gauge needle, allowing it to enter the bloodstream directly. Post-infection, the animals were closely monitored for signs of infection, such as changes in behavior, respiratory distress, weight loss, and reduced activity. infected animals were observed daily to assess the progression and effects of the viral infection

Blood Sample Collection and Organ Harvest

Blood samples were collected from each group of experimental animals before inducing with sendai virus, after inducing with sendai virus and after treatment. In order collect blood samples, the animals were first sedated using chloroform. This sedation procedure was carried out in a controlled environment within a desiccator to ensure safety and compliance with ethical guidelines. Following sedation, surgical incisions were carefully made in the chest region of the animals. These

incisions were instrumental in gaining access to the internal organs. By carefully opening both the chest and abdominal cavities, the heart became fully exposed. To facilitate subsequent analysis and preservation, transcardiac perfusion was conducted using a 10% formal The harvested organs, which include the livers, lung, kidney and heart, were preserved in a 10% formal saline solution. This preservation process is crucial in maintaining the structural integrity of the organs in preparation for subsequent histological examination.

Biochemical Assay

Haematological parameters

Determination of total white blood cell count by haemocytometry

The total white blood cell count was determined by haemocytometry following the method described by Ochei and Kolhatkar (2008).

Determination of platelet count

The platelet count was obtained using the haemocytometer.

Packed cell volume (PCV) estimation

The packed cell volume was determined using the microhaematocrit centrifuge (Jouan A13 model). (Cheesbrough, 2004).

Haemoglobin (Hb) concentration

The haemoglobin (Hb) concentration was measured spectrophotometrically by cyanomethaemoglobin method (Wameed & Bunea, n.d).

Determination of erythrocyte count by haemocytometry

The method of Math et al. (2016) was used.

Kidney and Liver Function Parameters

Determination of Aspartate Transaminase (AST) Activity in Plasma

Plasma sample of 0.1 ml was added to the sample tube, and 0.1ml of distilled water was added to the blank tube. The reagent buffer solution (0.5ml) was added to both tubes. This was well mixed and incubated for 30mins at 37˚C and reaction ensued. Later 0.5ml of solution 2 (2,4-dinitrophenylhydrazine) was added and they were mixed and allowed to stand for twenty minutes (20 mins) at 25˚C. Afterwards, 5.0ml of sodium hydroxide (NaOH) was pipetted into the test tubes: the contents were mixed and their absorbance read against the blank after 5 mins at 546nm in a spectrophotometer.

Determination of Alanine Transaminase (ALT) Activity In Plasma

Buffer solution of 0.5ml was added to reagent blank and sample test tubes and 0.1ml of sample and distilled water (0.1ml) were added into the sample and reagent blank tubes respectively. The mixture was incubated for exactly 30minutes at 37˚c (for the initiation of the reaction). Then 0.5ml of solution 2 (2,4-dinitrophenylhydrazine) was added to both tubes. Afterwards, the mixture was allowed to stand for exactly 20mins at a temperature of 25˚c (for colour development). Sodium hydroxide (0.4M) was added to both tubes, properly mixed and allowed to stand for 5minutes to stop the reaction. Absorbance of sample against the reagent blank was read at 546nm.

Determination of Alkaline Phosphatase (ALP) Activity in Plasma

Procedure: Three test tubes were set up, labeled reagent blank (T_1), test sample (T_2) and standard (T_3). To these were added 0.5ml of p-nitrophenyl phosphate and incubated at 37˚c for three minutes (3minutes). Distilled water (0.05ml), test sample (0.05ml) and standard (0.05ml) were added into the appropriate tubes and incubated for another 10 minutes. The ALP colour developer was added, the mixture was stirred properly and absorbance was read at 405nm.

Alkaline phosphatase (µ/L) = Change in absorbance at 405nm/min×2760

Alkaline phosphatase (µ/L) = ΔA405nm/min × 2760.

Total Protein

Procedure: The total protein was determined manually using a spectrophotometer at 546nm wavelength. The cuvette light path is 1cm and was measured against reagent blank. 0.02ml of distilled water was measured into the test tube labeled "reagent blank" and 1.0ml of R1 was added to it. To the tube labeled as standard, 0.02ml of standard (CAL) was measured into it and 1.0ml of R1 was added. To the test tube labeled as sample, 0.02ml of the serum to be tested was measured into it and 1.0ml of R1 added. The solutions in the tubes were shaken mix and incubated for 30 minutes at +20 to +25°C. the absorbance of the sample (A_{sample}) and of the standard ($A_{standard}$) was measured against the reagent blank.

Total Bilirubin

Procedure: The total bilirubin was determined manually using a spectrophotometer at 578nm wavelength. The cuvette light path was 1cm. The reagent compositions, R1, R2, R3 and the sample were mixed and allowed to stand at 25˚C for 10mins. The absorbance of the sample was read against the sample blank (A_{TB}).

Total Bilirubin (µmol/l) = $185 \times A_{TB}$ (578nm).

Urea

Procedure: Urea was determined manually using a spectrophotometer at 546nm wavelength. The cuvette light path is 1cm and was measured against Reagent blank at 37°C.

Three test tubes were labeled Blank, Standard and Sample respectively.10ul of distilled water was measured into the tube labeled blank, 10ul of standard into tube labeled standard and 10ul of samples serum into the tube labeled serum. 100ul of R1 was added respectively to each of the test tube and were shaken to mix, and incubated at 37°C for 10 minutes.

Subsequently, 2.50ml of R2 and 2.5ml of R3 were added to each tube, shaken to mix and incubated at 37°C for 15 minutes. The colour of the reaction is stable for at least eight hours. The absorbance of the sample (A_{sample}) and standard ($A_{standard}$) were read against the reagent blank.

Urea concentration = $A_{sample}/A_{standard} \times$ standard concentration (mmol/l)

Creatinine

Creatinine was determined manually using a spectrophotometer at 492nm wavelength. The cuvette light path is 1cm and was measured against air at 25/30/37°C. An aliquot 0.1ml of standard solution and 0.1ml of sample are measured into two separate cuvette and 1.oml of working reagent was added into each cuvette. They cuvettes were shaken to mix and after 30 seconds, the absorbance A_1 of the standard and sample was read. Exactly two minutes later, the absorbance A_2 of the standard and sample was also read.

$A_2 - A_1 = \Delta A_{sample}$ or $\Delta A_{standard}$

$\Delta A_{sample}/\Delta A_{standard} \times$ standard conc. (umol/l) = umol/l

Histological Examination

The tissue sections were stained using the method described by Alan & Ian in 1996. The 5 µm thick sections that were embedded and mounted previously were deparaffinized in xylene and rehydrated in descending concentrations of alcohol in a sequential manner. These were immersed in absolute alcohol, 90% alcohol, 80% alcohol, 70% alcohol and finally in distilled water. Samples were kept for 2–3 min in each solution. Thereafter, the sections were stained in Harris's Haematoxylin for 15 min and washed in running tap water until the brown colour disappeared. The slides were then immersed in a lithium carbonate solution for 10 s and washed in tap water. Washed slides were stained with Eosin (30 s) with agitation followed by washing with tap water. Sections were dehydrated in 70%, 90% and two changes of absolute alcohol with agitation (one minute each). Finally, the sections were cleared in xylene, and mounted and examined under a light microscope.

3. DATA ANALYSIS

The data obtained was analyzed using mean, standard deviation, simple percentage, and frequency counts, while the Statistical significance was tested using analysis of variance (ANOVA) and Turkey post Hoc test. The Analysis was performed using IBM SPSS software version 21

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4. RESULTS

Table 1.0: Haematological Parameters of Wistar Rats before Inducement with SeV

Parameters	Group 1	Group 2	Group 3	Group 4	Reference range
Red Blood Cell count (10^{12}/L)	6.43 ± 0.01[a]	6.40 ± 0.01[a]	6.56 ± 0.01[b]	6.40 ± 0.01 [a]	4.0 - 5.9
Haemoglobin (g/L)	16.91 ± 0.04[a]	16.81 ± 0.02[a]	16.79 ± 0.01[b]	16.13 ± 0.01[c]	14 - 17
Packed Cell Volume %	60.34 ± 0.01[a]	60.21 ± 0.01[a]	60.96 ± 0.04[b]	59.79 ± 0.23[c]	38.3 - 48.6
Red Cell Distribution Width %	11.67 ± 0.33[a]	10.33 ± 0.33[b]	13.00 ± 0.58[c]	12.33 ± 0.33[a]	11.5 - 14.5
Platelet (10^3/L)	221.67 ± 1.20[a]	229.33 ± 0.88[b]	227.33± 0.33[c]	232.00 ± 0.58[d]	150 - 400
White Blood Cell (10^9/L)	4.70 ± 0.06[a]	4.93 ± 0.03[b]	5.00 ± 0.10[c]	4.93 ± 0.03[b]	2.0 - 8.0
Neutrophil %	72.00 ± 1.15[a]	72.00 ± 1.00[a]	72.33 ± 0.33[a]	73.50 ± 0.50[a]	40 - 60
Monocyte %	0.33 ± 0.33[a]	0.67 ± 0.33[b]	0.33 ± 0.33[a]	0.33 ± 0.33[a]	2 - 8
Lymphocyte %	22.00 ± 0.58[a]	21.33 ± 1.45[a]	20.67 ± 0.88[a]	21.67 ± 0.88[a]	20 - 40

Values are presented in mean ± SEM. N=3. Mean values with same alphabet on same row have no statistically significant difference at $p \leq 0.05$

N.B: Group 1-4 are different mouse groups with the same treatment at the moment.

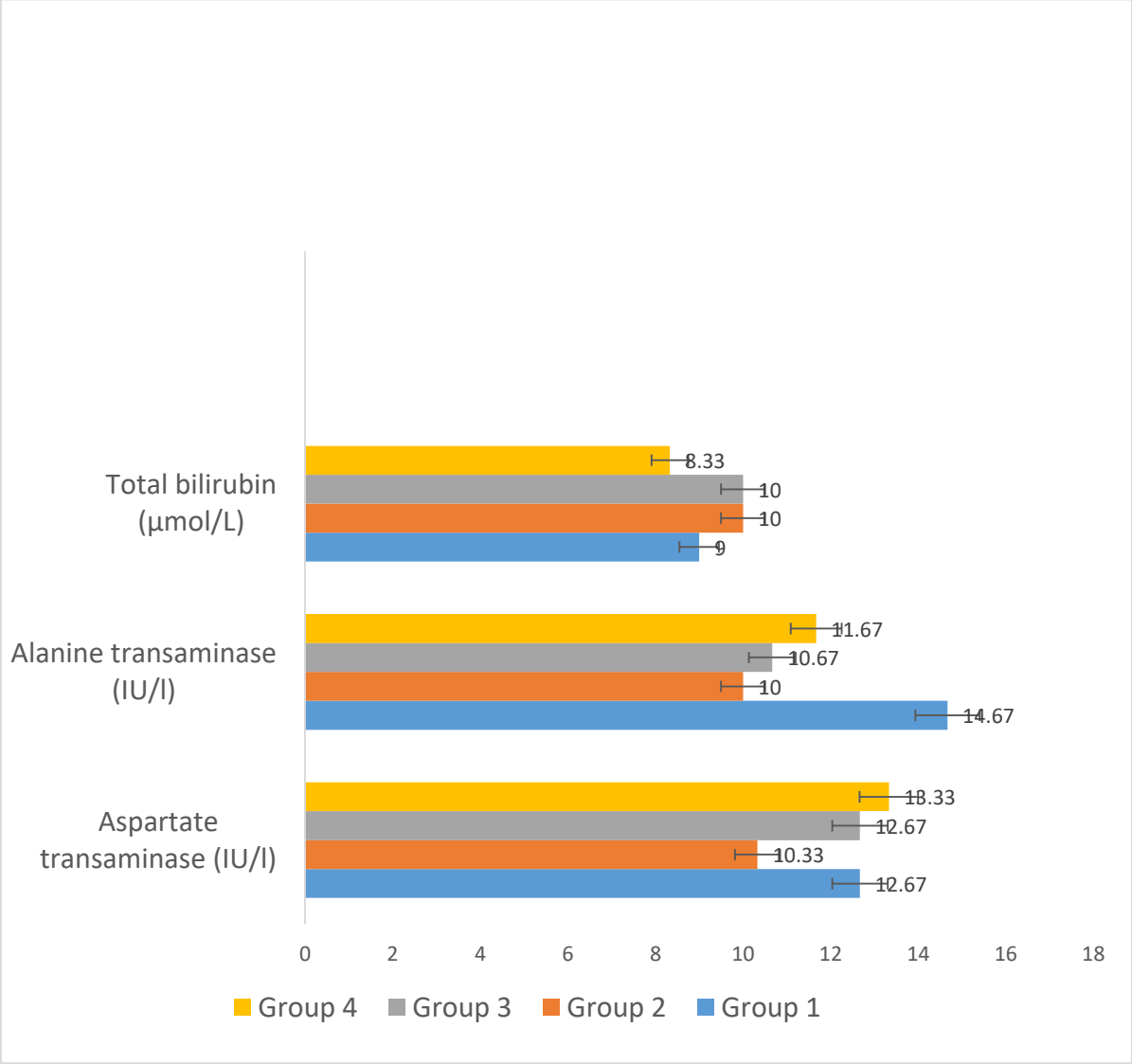

Figure 1.0a: Liver function parameters (AST, ALT and Total bilirubin) of Wistar Rats before Inducement

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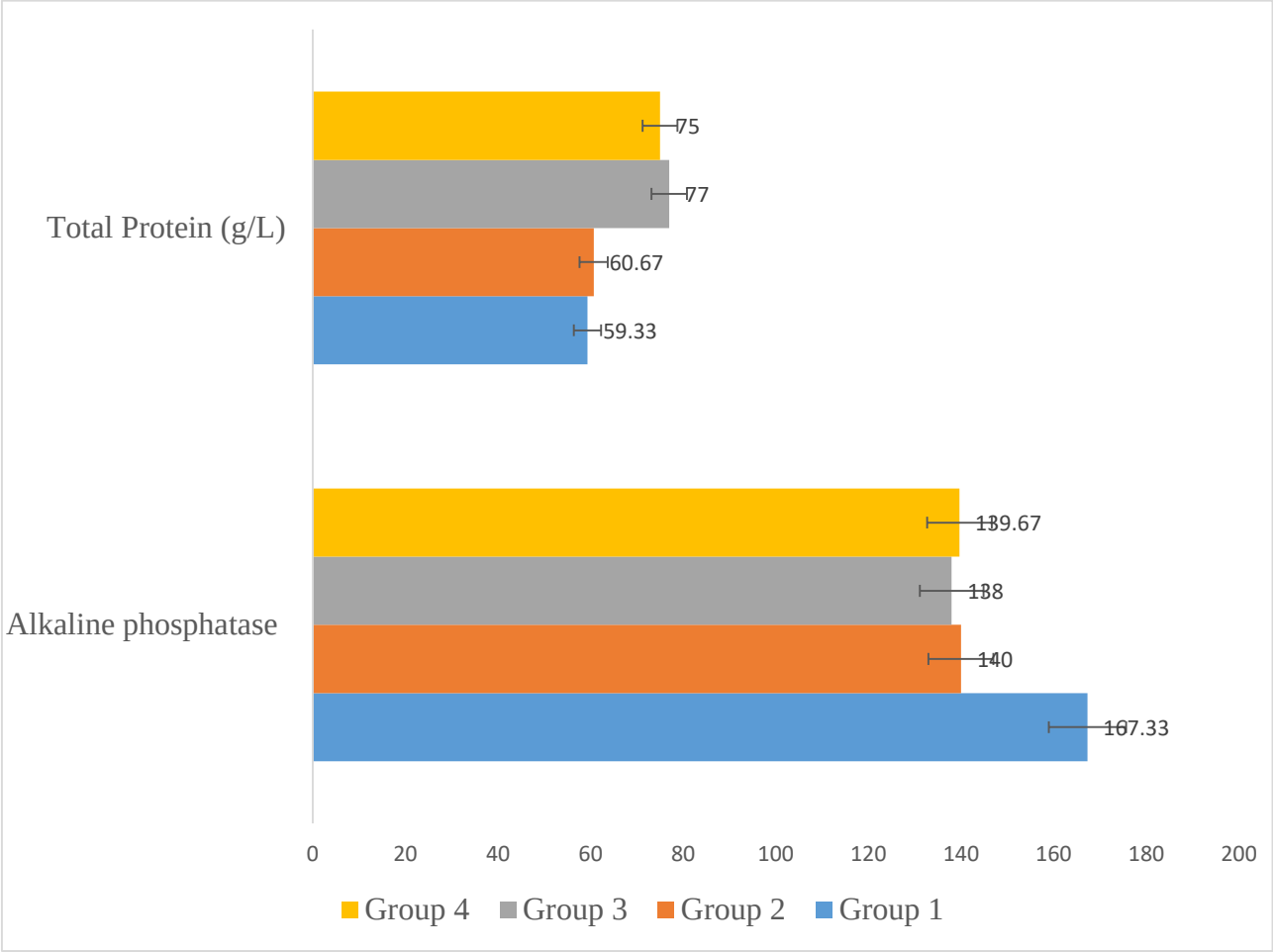

Figure 2.0: Liver function parameters (ALP and Total protein) of Wistar Rats before Inducement

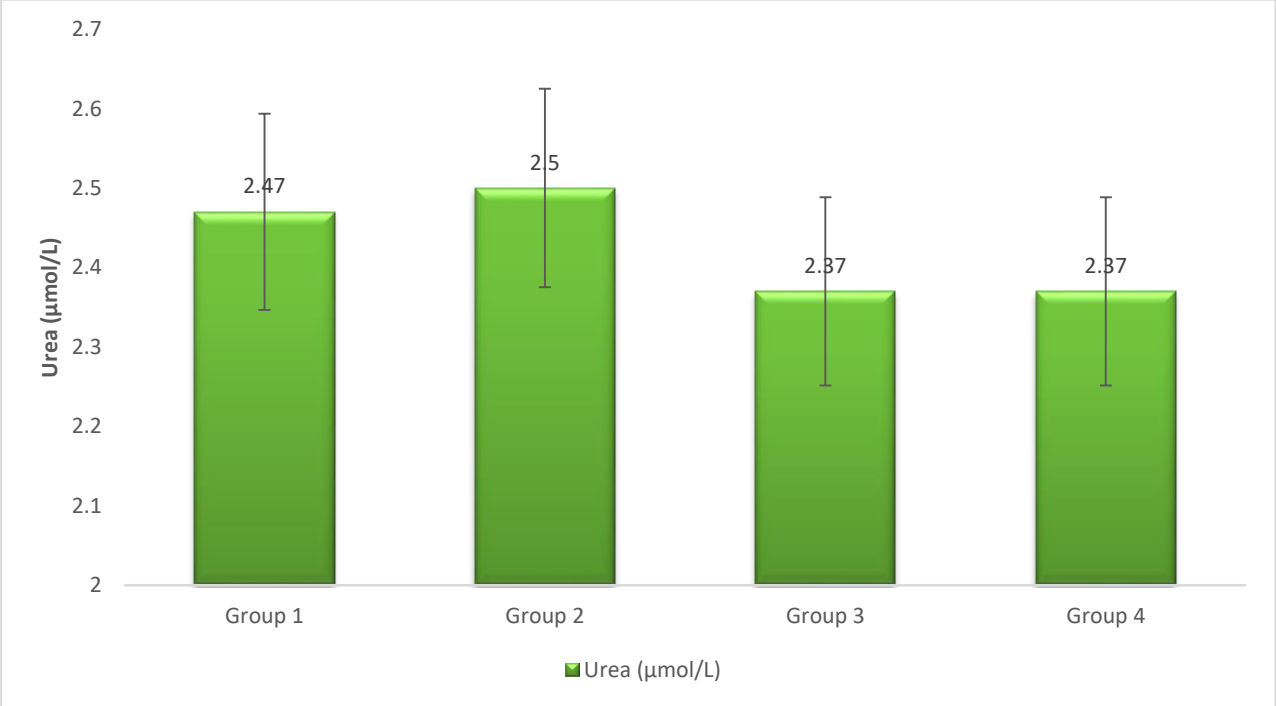

Figure 3.0: Kidney function parameter (urea) of Wistar Rats before Inducement

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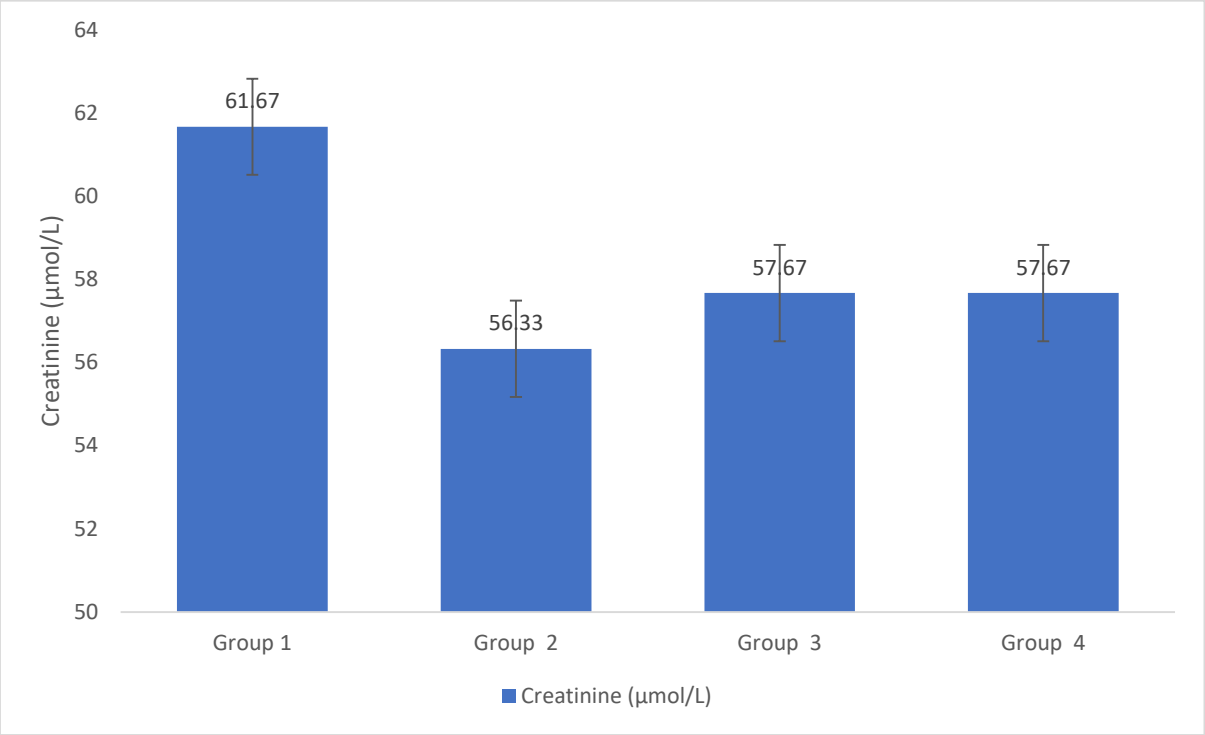

Figure 4.0. Kidney function parameter (Creatinine) of Wistar Rats before Inducement

Table 2.0: Haematological Parameters of Sendai Virus induced Wistar Rats

Parameters	Group 1	Group 2	Group 3	Group 4	Reference range
Red Blood Cell count (10^{12}/L)	6.60 ± 2.09^{a}	6.26 ± 0.02^{a}	6.27 ± 0.05^{c}	4.17 ± 0.01^{d}	4.0 - 5.9
Haemoglobin (g/L)	16.62 ± 0.03^{a}	15.45 ± 0.01^{b}	15.38 ± 0.00^{b}	15.44 ± 0.02^{b}	14 - 17
Packed Cell Volume %	59.96 ± 0.04^{a}	55.46 ± 0.36^{b}	57.37 ± 0.17^{c}	55.51 ± 0.53^{b}	38.3 - 48.6
Red Cell Distribution Width %	11.33 ± 0.67^{a}	16.67 ± 0.88^{b}	16.00 ± 1.15^{b}	17.00 ± 0.58^{b}	11.5 - 14.5
Platelet (10^{3}/L)	225.67 ± 0.33^{a}	227.00 ± 0.58^{a}	225.67 ± 1.20^{a}	227.33 ± 0.88^{a}	150 - 400
White Blood Cell (10^{9}/L)	4.77 ± 0.03^{a}	5.13 ± 0.09^{b}	5.43 ± 0.19^{c}	5.37 ± 0.12^{c}	2.0 - 8.0
Neutrophil %	74.67 ± 1.20^{a}	66.67 ± 0.88^{b}	66.33 ± 2.03^{b}	66.00 ± 1.15^{b}	40 - 60
Monocyte %	0.33 ± 0.33^{a}	0.67 ± 0.33^{a}	0.67 ± 0.33^{a}	0.67 ± 0.33^{a}	2 - 8
Lymphocyte %	19.67 ± 0.88^{a}	27.00 ± 0.58^{b}	26.33 ± 1.45^{b}	27.67 ± 1.45^{b}	20 - 40

Values are presented in mean ± SEM. N=3. Mean values with same alphabet on same row have no statistically significant difference at $p \leq 0.05$

Table 3.0: Liver and Kidney function parameters of Sendai Virus induced Wistar Rats

Parameters	Group 1	Group 2	Group 3	Group 4	Reference range
Aspartate transaminase (IU/l)	9.67 ± 0.33^{a}	18.67 ± 1.20^{b}	25.67 ± 0.88^{c}	17.33 ± 0.88^{d}	3 – 15
Alanine transaminase (IU/l)	12.00 ± 0.58^{a}	21.67 ± 0.67^{b}	28.00 ± 0.58^{c}	20.33 ± 0.88^{b}	10 - 40
Alkaline phosphatase	128.33 ± 2.40^{a}	174.67 ± 1.86^{b}	179.67 ± 4.06^{c}	179.67 ± 3.28^{c}	44-147
Total Protein (g/L)	65.67 ± 0.88^{a}	56.00 ± 0.58^{b}	49.33 ± 1.45^{c}	50.00 ± 1.15^{c}	60 - 83
Total bilirubin (µmol/L)	10.67 ± 0.33^{a}	15.33 ± 0.88^{b}	20.00 ± 0.58^{c}	18.33 ± 0.33^{c}	1.71 - 20.5
Urea (µmol/L)	2.27 ± 0.03^{a}	2.80 ± 0.06^{b}	3.27 ± 0.09^{c}	3.00 ± 0.06^{d}	1.8 - 7.1
Creatinine (µmol/L)	57.00 ± 0.58^{a}	66.00 ± 0.58^{b}	67.67 ± 0.33^{c}	63.00 ± 0.58^{d}	61.9 - 114.9

Values are presented in mean ± SEM. N=3. Mean values with same alphabet on same row have no statistically significant difference at $p \leq 0.05$

Group 1= control without inducement

Group 3-4= SeV induced wiatar rats. Grouped for the purpose of future treatment regimen

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5. DISCUSSION

Haematological Parameters

The haematological parameters of Wistar rats before Sendai virus induction are presented in Table 1.0. and the results are discussed as follows.

Red Blood Cell (RBC) Count: All groups exhibited RBC counts above the normal reference range (4.0-5.9 × 10^{12}/L). Group 3 showed the highest count (6.56 ± 0.01 × 10^{12}/L), while Groups 1 (control), 2, and 4 had slightly lower counts (6.43 ± 0.01, 6.40 ± 0.01, and 6.40 ± 0.01 × 10^{12}/L, respectively).

Haemoglobin (Hb) Concentration: Groups 1, 2, and 3 had Hb concentrations close to the upper limit of the normal reference range (14-17 g/L). Group 1 (control) had the highest concentration (16.91 ± 0.04 g/L), while Group 4 had a significantly lower concentration (16.13 ± 0.01 g/L).

Packed Cell Volume (PCV): All experimental groups showed significantly higher PCV values than the reference range (38.3-48.6%). Group 3 had the highest PCV (60.96 ± 0.04%), while Groups 1, 2, and 4 had slightly lower values, with Group 4 having the lowest (59.79 ± 0.23%).

Red Cell Distribution Width (RDW): Groups 1 to 4 had RDW values within the normal reference range (11.5-14.5%). Group 3 had the highest RDW (13.00 ± 0.58%), suggesting potential variability in red blood cell size. Group 2 had the lowest RDW (10.33 ± 0.33%), which is below the reference range.

Platelet Count: All groups had platelet counts within the normal reference range (150-400 × 10^3/L). Group 4 showed the highest count (232.00 ± 0.58 × 10^3/L), while Group 1 (control) showed the lowest count (221.67 ± 1.20 × 10^3/L).

White Blood Cell (WBC) Count: All groups had WBC counts within the normal reference range (2.0-8.0 × 10^9/L). Group 3 had the highest count (5.00 ± 0.10 × 10^9/L), while Groups 2 and 4 had slightly lower counts (4.93 ± 0.03 × 10^9/L). The control group (Group 1) had the lowest WBC count (4.70 ± 0.06 × 10^9/L).

Differential White Blood Cell Count:

Neutrophils: All groups had neutrophil percentages above the reference range (40-60%), with Group 4 having the highest percentage (73.50 ± 0.50%).

Monocytes: All groups had monocyte percentages below the reference range (2-8%), with Group 2 showing the highest percentage (0.67 ± 0.33).

Lymphocytes: All groups had lymphocyte percentages within the normal range, with Group 1 (control) showing the highest percentage (22.00 ± 0.58%).

Liver and Kidney Function Parameters

Figure 1 and 2 show the liver and kidney function parameters of Wistar rats before induction.

Aspartate Transaminase (AST): All groups had AST levels within the normal reference range (3-15 IU/L).

Alanine Transaminase (ALT): All groups had ALT levels within the normal reference range (10-40 IU/L).

Alkaline Phosphatase (ALP): Group 1 had an ALP level above the normal reference range (44-147 IU/L), while Groups 2, 3, and 4 had levels within the normal range.

Total Protein (TP): Groups 3 and 4 had TP levels above the normal reference range (60-83 g/L), while Groups 1 and 2 had levels within the normal range.

Total Bilirubin: All groups had total bilirubin levels within the normal reference range.

Urea: All groups had urea levels within the normal reference range (1.8-7.1 µmol/L).

Creatinine: All groups had creatinine levels within the normal reference range (61.9-114.9 µmol/L), indicating normal kidney function.

Following Sendai virus induction, significant alterations in haematological parameters were observed:

RBC Count: Group 4 displayed a marked decrease in RBC count, indicative of potential anemia.

Hemoglobin and PCV: Groups 2, 3, and 4 exhibited slight reductions in hemoglobin and PCV, suggesting diminished oxygen-carrying capacity.

RDW: Groups 2, 3, and 4 showed a significant increase in RDW, indicating variability in red blood cell size due to the viral infection.

WBC Count: Groups 3 and 4 exhibited significant increases in WBC count, reflecting an activated immune response to the viral challenge. Activation of immune response is wdely reported as the body's response to viral infection (Spudich, 2016; Silva et al., 2023)

Differential White Blood Cell Count:

A slight decrease in neutrophil percentage was observed in Groups 2, 3, and 4.

A significant increase in lymphocyte percentage was observed in Groups 2, 3, and 4, suggesting a robust immune response involving T and B lymphocytes. The findings of Rosendahl Huber, et al., (2014). Corroborates this finding.

Liver and Kidney Function Parameters After Induction

Post-infection, significant alterations in liver and kidney function parameters were observed:

Liver Enzymes: Groups 2, 3, and 4 exhibited elevated levels of AST, ALT, and ALP, indicative of liver damage.

Total Protein: Groups 3 and 4 showed decreased total protein levels, suggesting impaired liver function. (Chukwunonyelum, et al., 2016).

Total Bilirubin: Groups 3 and 4 displayed increased bilirubin levels, indicative of liver damage and impaired bile excretion (Guerra Ruiz, et al., 2021)

Kidney Function Markers: Groups 3 and 4 exhibited elevated levels of urea and creatinine, suggesting impaired kidney function (Brookes, & Power, 2022).

Conclusion: The outcomes of this investigation emphasize the far-reaching consequences of Sendai virus infection in Wistar rats. The observed alterations in hematological and biochemical parameters provide evidence of a systemic response to the viral challenge. The anemia, characterized by reduced red blood cell count and hemoglobin levels, suggests a compromised oxygen-carrying capacity. The changes in red blood cell morphology, as indicated by increased RDW, point to potential cellular stress or damage. The activated immune response, evidenced by elevated white blood cell count and lymphocyte percentage, reflects the body's defense mechanism against the viral infection. Furthermore, the elevated liver enzymes, decreased total protein, and increased bilirubin levels suggest liver damage, while the increased kidney function markers point to potential renal impairment. Collectively, these findings highlight the multifaceted nature of Sendai virus infection, extending its impact beyond the respiratory system to other vital organs.

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