Original Article

(a) Open Access



International Journal of Experimental Research and Review (IJERR) © Copyright by International Academic Publishing House (IAPH) ISSN: 2455-4855 (Online) www.iaph.in

Peer Reviewed



Analysing the association of *Helicobacter pylori* induced gastritis with neutrophillymphocyte ratio (NLR) and volume, conductance, scatter characteristics of LH780 Coulter Check for updates

Suresh Kumar Sinduja¹ and Rajesh Kanna Nandagopal Radha²*

¹Chettinad Hospital and Research Institute, Chettinad Academy of Research and Education, Rajiv Gandhi Salai (OMR), Kelambakkam, Chennai-603103, Tamilnadu, India; ²Department of Pathology, Chettinad Hospital and Research Institute, Chettinad Academy of Research and Education, Rajiv Gandhi Salai (OMR), Kelambakkam, Chennai- 603103, Tamilnadu, India E-mail/Orcid Id:

SKS, Sindhusk2407@gmail.com, https://orcid.org/0000-0002-0462-7762; RKNR, a rajeshthuva@gmail.com, b https://orcid.org/0000-0001-5354-670X

Article History:

Received: 27th Jan., 2023 Accepted: 08th Mar., 2023 Published: 30th Apr., 2023 **Keywords:** Conductance, H. pylori,

gastritis, LH780 Coulter neutrophilhemogram, lymphocyte ratio, scatter characteristics

Abstract: Helicobacter (H.) pylori is a bacterium that infects the stomach leading to chronic gastritis. In cases of extreme infection, it can lead to condition of painful sores called peptic ulcers. To determine the association of *H. pylori* infection in gastric mucosa with blood parameters like Neutrophil-Lymphocyte ratio (NLR) and volume, conductance and scatter (VCS) characteristics were studied using LH780 Coulter haemogram. Blood samples of 100 normal subjects and 100-gastritis cases were collected for testing complete blood count. Biopsies of the gastric tract were also conducted for the 100 gastritis patients. The total leucocyte count was significantly higher in the gastritis patients than in the control subjects. On the other hand, the control subjects' haemoglobin percentage, RBC count, Packed Cell Volume percentage, Basophil percentage, and Lymphocyte percentage were significantly higher. Absolute neutrophil count, Neutrophil conductance (Standard Deviation, SD), Neutrophil Scatter (SD), Lymphocyte volume (SD), Lymphocyte Conductance, Lymphocyte Scatter (SD) and Neutrophil-Lymphocyte ratio were significantly high among gastritis patients. Mean neutrophil scatter was significantly higher among control subjects. Haemoglobin percentage, RBC count, PCV percentage, MCV, eosinophil percentage, basophil percentage and lymphocyte percentage were significantly higher in control subjects compared to no activity gastritis patients. Absolute neutrophil volume, neutrophil conductance (SD), lymphocyte volume (SD), lymphocyte conductance (SD), lymphocyte scatter (SD) and neutrophil-lymphocyte ratio were significantly higher among no-activity gastritis patients. RBC count, haemoglobin percentage and packed cell volume percentage were significantly higher among the control subjects compared to the moderate activity gastritis patients. Lymphocyte volume (SD) and scatter (SD) were significantly higher among moderate-activity gastritis patients. Scatter parameters such as neutrophil conductance (SD), lymphocyte conductance SD and lymphocyte scatter (SD) were significantly higher among high-activity gastritis patients. Mean neutrophil scatter was significantly higher among control subjects compared to high activity gastritis patients. Based on our observation we propose that the N/L ratio shows signs of escalation due to the increased severity of the inflammation and intensified neutrophil infiltration of the gastric mucosa.

Introduction

Cancer Bacteria Helicobacter (H.) pylori cause multiple disorders of the gastric tract, such as chronic gastritis, lymphoid tissue, peptic ulcer, and gastric cancer associated with mucosa (Makola et al., 2007). H. pylori (Hp) related infection is more predominant among

^{*}Corresponding Author: rajeshthuva@gmail.com



middle-aged adults but may be present among populations of all ages (Makola et al., 2007). Hp is known for high morbidity but low mortality (Malaty et al., 2007). It is proven that Hp mainly stays outside the protective lining of the stomach mucosa and leads to inflammation (Winter et al., 2020). In infected individuals, acid secretions get higher than normal, and that causes chronic gastritis in the antrum of the stomach and gastric metaplasia of duodenum, favouring Hp to colonize in normal duodenum (Hamlet et al., 1999; Priyadharshini et al., 2022).

Role of Hp infection leading to gastritis and peptic ulcers is well established. There is a 90%-95% prevalence of HP-induced gastritis and duodenal ulcers (Hunt, 1996). Even after successfully eradicating Hp infection, peptic ulcer development is more likely in people with a previous history of infection (Kuipers et al., 1995). However, the investigation reveals that the peptic ulcer disease's recurrence decreases with the successful eradication of Hp infection compared to the non-cured patient (Hopkins et al., 1996). The progression of the infection is seen to be graded from no infection followed by mild to severe in biopsies, and their respective clinical picture shows asymptomatic to chronic gastritis and ulcers (Ruggiero, 2010). Umit et al. (2015) showed that acute infection tends to cause impairment in nongastrointestinal tissues. Gastritis also reduces the effectiveness of the body's ability to absorb iron (Kim et al., 2013).

Histopathological examination is the gold standard for detecting Hp infection. Other methods are rapid urease tests, PCR techniques or microbial culture (Thompson et al., 1982). There are other blood biomarkers available to assess the severity of inflammation in Hp induced gastritis, like C-reactive protein (CRP), tumour necrosis factor-alpha (TNF- α) and angiotensin-converting enzyme (Jafarzadeh et al., 2009; Mehmet et al., 2005). In addition, other biomarkers, including gastrin, interleukin-8, HLA class II molecules, reactive oxygen species and histological grades, correlate to the severity of inflammation (Naito et al., 2005).

Studies have shown that escalation in values of neutrophils and lymphocytes with several chemotactic proteins in the stomach induces inflammation in the gastric mucosa (Makola et al., 2007). The high leukocyte count and increased neutrophil-lymphocyte ratio (NLR) may be linked with the severity of stomach inflammation (Horne et al., 2005). The neutrophil-lymphocyte ratio is a reliable, inexpensive, highly sensitive, and specific marker for evaluating systemic inflammation and stomach malignancies (Farah et al., 2014). The neutrophil-lymphocyte ratio has been used as a prognostic value in assessing the severity of gastritis (Forget et al., 2017).

Choccalingam (2018) study demonstrated that the characteristics of volume, conductance, and scatter parameters (VCS) using the Coulter LH780 and the automated haematology analysers are much more effective in detecting changes in cell characteristics and provide better results in generating a comprehensive haematological profile.

In the present study, we aimed to determine the association of Hp infection causing regional chronic inflammation in gastric mucosa and assess the relation and differences in NLR and VCS characteristics using hemogram pictures derived from Coulter LH780. The other parameters that were evaluated as part of the study are the inflammatory process's status and ruling out microcytic hypochromic anaemia if present.

This study evaluates the prognostic values of the NLR ratio by helping us to identify the type of gastritis by ruling out the autoimmune cause in which lymphocytes are generally raised. By noting the VCS characteristics in hemogram of gastritis cases, it helps to check the success of the treatment.

Material and Methods

For the present study, we selected 200 individuals who visited the tertiary care of the multi-speciality Hospital for treatment. Out of them, 100 individuals were antral gastritis patients who have undergone endoscopic biopsies. Gastritis patients with criteria such as - (1) gastroscopy findings without antral gastritis; (2) systemic and chronic diseases; (3) gastrointestinal haemorrhage; (4) uncontrolled diabetes mellitus; (5) portal hypertension; and (6) patients on drugs such as nonsteroidal anti-inflammatory drug, proton pump inhibitors, H2 and cytotoxic medicines, were excluded from the study. The remaining 100 individuals were normal subjects without any gastritis symptoms and were used as control.

Out of 100 gastritis patients, 56 were male and 44 female. On the other hand, out of 100 normal subjects, 58 were male, and 42 were female. Based on age, subjects were segregated into three categories–Category I (15–30 years), Category II (31–50 years) and Category III (51–70 years). The number of gastritis patients in Categories I, II and II were 29, 47 and 24, respectively. Likewise, a number of normal subjects included in Categories I, II and II were 29, 47 and 24 individuals, respectively.

The histopathological features of the antral gastric biopsies were characterised following Sydney

Classification (1996) protocol and were subsequently analysed. We scored the changes due to Hp-induced gastritis-as 0 (no), 1 (mild), 2 (moderate), 3 (severe)based on the intensity of activity (neutrophil intensity) in the lamina propria of the gastric mucosa. We performed hemogram tests for all gastritis patients. We used Coulter LH 780 haematology analyser to record the total WBC count, neutrophil count, lymphocyte count, differential plot, and VCS positional characteristics for each group of patients. The Coulter LH 780 haematology analyser uses an advanced technology (Volume, Conductivity and Scatter (VCS) Principle) to improve cell enumeration and characteristics. We also recorded the Complete Blood Count (CBC) details of the patients and control subjects, and the mean and standard deviation of the parameters were correlated statistically with details of the biopsy report of gastritis patients. We used the neutrophillymphocyte ratio as a marker to evaluate whether the type of systemic inflammation is due to a bacterial cause like Hp or an autoimmune cause where lymphocytes play a major role.

Prior to conducting the statistical analyses, we conducted Shapiro-Wilk's test to assess the normality of the continuous variables. All parameters, other than RBC count, showed non-normal distribution. We conducted non-parametric tests to assess the significance of the non-normal variables and parametric tests for the normal variables. For non-normal variables, the difference in mean between the control subjects and the gastritis patients was assessed using the Mann-Whitney-Wilcoxon (W) test. The difference between control subjects and the gastritis patients was assessed for normal variables using Welch two sample 't'-test. Results with p<0.05 were considered significant. All analyses were conducted using R version 4.2.3 [Copyright (C): The R Foundation for Statistical Computing, 2023].

Results

Comparison of total blood count, leucocyte count and Volume, Conductivity and Scatter (VCS) parameters between control subjects and gastritis patients

Among the CBC parameters, Total leucocyte counts were significantly higher in the gastritis patients than in the control subjects (Table 1). However, the control subjects' haemoglobin percentage, RBC count, packed cell volume percentage, Basophil percentage, and Lymphocyte percentage were significantly higher (Table 1).

The VCS parameters such as absolute neutrophil count, neutrophil conductance SD, neutrophil Scatter SD, lymphocyte volume SD, lymphocyte conductance SD, lymphocyte scatter SD and neutrophil-lymphocyte ratio were significantly high among gastritis patients (Table 1). Neutrophil scatter means, however, was significantly higher among control subjects (Table 1).

Comparison of total blood count, leucocyte count and Volume, Conductivity and Scatter (VCS) parameters between control subjects and gastritis patients with no activity

During our study, we recorded significantly higher values of haemoglobin percentage, RBC count, packed cell volume percentage, mean corpuscular volume, eosinophil percentage, basophil percentage and lymphocyte percentage in control subjects compared to gastritis patients with no infiltration of neutrophils in the lamina propria of gastric mucosa (no-activity gastritis patients) (Table 2).

We observed that while the absolute neutrophil volume, neutrophil conductance SD, lymphocyte volume SD, lymphocyte conductance SD, lymphocyte scatter SD and neutrophil-lymphocyte ratio were significantly higher among the gastritis patients with no infiltration of neutrophils in the lamina propria of gastric mucosa (no-activity gastritis patients), absolute lymphocyte count and neutrophil scatter mean was significantly higher among the control subjects (Table 2).

Comparison of total blood count, leucocyte count and Volume, Conductivity and Scatter (VCS) parameters between control subjects and gastritis patients with mild activity

Among CBC parameters, haemoglobin percentage, RBC count and packed cell volume were significantly higher in control subjects compared to gastritis patients with mild infiltration of neutrophils in the lamina propria of the gastric mucosa (mild activity gastritis patients) (Table 3). Among scatter parameters, neutrophil scatter means were significantly high in control subjects. On the other hand, scatter parameters such as neutrophil conductance SD, neutrophil scatter SD, lymphocyte volume SD and lymphocyte scatter SD were significantly higher among gastritis patients with mild infiltration of neutrophils in the lamina propria of the gastric mucosa (mild activity gastritis patients) (Table 3).

Comparison of total blood count, leucocyte count and Volume, Conductivity and Scatter (VCS) parameters between control subjects and gastritis patients with moderate activity

We observed that RBC count, haemoglobin percentage and packed cell volume percentage were significantly higher among the control subjects compared to the gastritis patients with moderate infiltration of neutrophils in the lamina propria of the gastric mucosa

Table 1. Comparison of total blood count, leucocyte count and Volume, Conductivity and Scatter (VCS) parameters between control subjects and gastritis patients.

Parameters	Control Subjects	Gastritis Patients	Test For statistical significance			ance
	Mean±S.E.	Mean±S.E.	W	t- test	df	p- value
Haemoglobin (Hb %)	13.72±0.19	12.46±0.23	6557.5			< 0.001
Total Count						
Total Leukocyte count (Thousands/cu.mm)	7533.00±221.21	8390.00±259.38	4019.5			0.02
RBC count (millions/cu.mm)	4.84 ± 0.05	4.51±0.06		4.2	186.78	< 0.001
Differential Leucocyte Count						
Packed Cell Volume (PCV) (%)	41.85±0.54	37.96±0.62	6769.5			< 0.001
Mean Corpuscular Volume (MCV) (fl)	86.31±0.74	84.30±0.90	5796.5			0.05
Mean Corpuscular Haemoglobin (MCH) (pg)	28.36±0.28	27.64±0.35	5605.0			0.14
Mean Corpuscular Haemoglobin Concentration (MCHC) (%)	32.74±0.08	32.72±0.13	4586.5			0.31
Red Cell Distribution Width (RDW)	14.30±0.17	15.09±0.36	4494.5			0.22
Platelet Count (Lakhs/ c.mm)	2.61±0.08	2.86±0.09	4394.5			0.14
Neutrophil (%)	56.73±0.77	59.93±1.14	4281.5			0.08
Eosinophil (%)	4.25±0.36	4.07±0.32	5171.0			0.68
Basophil (%)	0.60±0.03	0.56±0.05	5996.0			0.01
Lymphocyte (%)	30.95±0.68	27.92±0.98	5980.0			0.02
Monocyte (%)	7.46±0.16	7.90±0.32	4848.5			0.71
Volume, Conductivity and Scatter	Parameter					
Absolute neutrophil count	4.39±0.18	5.22±0.24	3874.5			0.01
Absolute lymphocyte count	2.26±0.06	2.18±0.08	5500.0			0.22
Neutrophil Conductance, SD	5.01±0.07	5.62±0.09	2713.0			< 0.001
Neutrophil Scatter, Mean	148.78±0.48	145.28±0.67	6562.0			< 0.001
Neutrophil Scatter, SD	9.92±0.12	10.58±0.20	3929.0			< 0.001
Lymphocyte volume, SD	13.91±0.12	14.72±0.18	3528.5			< 0.001
Lymphocyte Conductance, SD	10.20±0.18	11.06±0.21	3744.0			< 0.01
Lymphocyte Scatter, SD	16.23±0.19	17.63±0.22	2815.0			< 0.001
N/L Ratio	2.03±0.09	2.99±0.29	4003.0			0.01

Table 2. Comparison of total blood count, leucocyte count and Volume, Conductivity and Scatter (VCS) parameters between control subjects and gastritis patients with no activity

	Control Subjects	Gastritis Patients	Test for statistical significance			
Parameters	Mean±S.E.	Mean±S.E.	W	t- test	df	p- value
Haemoglobin (Hb %)	13.72±0.19	12.11±0.43	2677.00			0.002
Total Count	I	1		1	1	
Total Leukocyte count (Thousands/cu.mm)	7533.00±221.21	8815.00±505.93	1612.50			0.074
RBC count (millions/cu.mm)	4.84±0.05	4.44±0.09		3.80	60.99	0.000
Differential Leucocyte Count						
Packed Cell Volume (PCV) (%)	41.85±0.54	36.90±1.17	2759.50			0.000
Mean Corpuscular Volume (MCV) (fl)	86.31±0.74	82.80±1.55	2461.50			0.033
Mean Corpuscular Haemoglobin (MCH) (pg)	28.36±0.28	27.10±0.62	2370.50			0.088
Mean Corpuscular Haemoglobin Concentration (MCHC) (%)	32.74±0.08	32.62±0.22	1837.50			0.454
Red Cell Distribution Width (RDW)	14.30±0.17	15.37±0.66	1798.00			0.352
Platelet Count (Lakhs/ c.mm)	2.61±0.08	2.92±0.16	1673.00			0.132
Neutrophil (%)	56.73±0.77	61.00±1.94	1585.50			0.056
Eosinophil (%)	4.25±0.36	3.25±0.45	2484.00			0.026
Basophil (%)	0.60±0.03	0.55±0.10	2570.50			0.008
Lymphocyte (%)	30.95±0.68	26.46±1.47	2525.50			0.015
Monocyte (%)	7.46±0.16	8.74±0.64	1622.50			0.082
Volume, Conductivity and Scatter Parame	eter					
Absolute neutrophil count	4.39±0.18	5.53±0.44	1472.50			0.015
Absolute lymphocyte count	2.26±0.06	2.13±0.14	2249.50			0.250
Neutrophil Conductance, SD	5.01±0.07	5.69±0.14	1085.00			0.000
Neutrophil Scatter, Mean	148.78±0.48	144.86±1.18	2647.50			0.003
Neutrophil Scatter, SD	9.92±0.12	10.85±0.43	1613.50			0.075
Lymphocyte volume, SD	13.91±0.12	14.85±0.30	1301.00			0.001
Lymphocyte Conductance, SD	10.20±0.18	11.35±0.28	1226.50			0.000
Lymphocyte Scatter, SD	16.23±0.19	17.80±0.30	973.00			0.000
N/L Ratio	2.03±0.09	3.22±0.41	1536.00			0.033

 Table 3. Comparison of total blood count, leucocyte count and Volume, Conductivity and Scatter (VCS)

 parameters between control subjects and gastritis patients with mild activity

Dowomotoro	Control Subjects	Gastritis Patients	Test for statistical significan			ficance
Parameters	Mean±S.E.	Mean±S.E.	W	t- test	df	p- value
Haemoglobin (Hb %)	13.72±0.19	12.66±0.32	2464.00			0.007
Total Count			1	•		
Total Leukocyte count (Thousands/cu.mm)	7533.00±221.21	7863.16±273.60	1603.50			0.158
RBC count (millions/cu.mm)	4.84±0.05	4.58±0.10		2.38	56.914	0.021
Differential Leucocyte Count						
Packed Cell Volume (PCV) (%)	41.85±0.54	38.67±0.86	2515.50			0.003
Mean Corpuscular Volume (MCV) (fl)	86.31±0.74	84.72±1.45	2193.50			0.163
Mean Corpuscular Haemoglobin (MCH) (pg)	28.36±0.28	27.72±0.57	2166.50			0.205
Mean Corpuscular Haemoglobin Concentration (MCHC) (%)	32.74±0.08	32.65±0.17	1853.00			0.824
Red Cell Distribution Width (RDW)	14.30±0.17	15.24±0.55	1514.50			0.066
Platelet Count (Lakhs/ c.mm)	2.61±0.08	2.90±0.14	1645.50			0.226
Neutrophil (%)	56.73±0.77	60.03±1.62	1637.50			0.212
Eosinophil (%)	4.25±0.36	4.51±0.49	1721.50			0.396
Basophil (%)	0.60±0.03	$0.57 {\pm} 0.07$	2211.00			0.136
Lymphocyte (%)	30.95±0.68	27.86±1.24	2287.50			0.065
Monocyte (%)	7.46±0.16	7.03±0.36	2245.50			0.100
Volume, Conductivity and Scatter Param	eter	• •				
Absolute neutrophil count	4.39±0.18	4.88±0.26	1498.00			0.056
Absolute lymphocyte count	2.26±0.06	2.15±0.12	2148.00			0.237
Neutrophil Conductance, SD	5.01±0.07	5.57±0.12	942.50			0.000
Neutrophil Scatter, Mean	148.78±0.48	145.22±0.96	2559.00			0.002
Neutrophil Scatter, SD	9.92±0.12	10.41±0.19	1322.50			0.006
Lymphocyte volume, SD	13.91±0.12	14.60±0.26	1403.50			0.018
Lymphocyte Conductance, SD	10.20±0.18	10.70±0.33	1585.00			0.134
Lymphocyte Scatter, SD	16.23±0.19	17.58±0.43	1213.00			0.001
N/L Ratio	2.03±0.09	2.75±0.39	1529.50			0.078

 Table 4. Comparison of total blood count, leucocyte count and Volume, Conductivity and Scatter (VCS)

 parameters between control subjects and gastritis patients with moderate activity

Parameters	Control Subjects	Gastritis Patients	Test For statistical significanc			ificance
Parameters	Mean±S.E.	Mean±S.E.	W	t- test	df	p- value
Haemoglobin (Hb %)	13.72±0.19	12.13±0.51	955.50			0.006
Total Count						
Total Leukocyte count (Thousands/cu.mm)	7533.00±221.21	8223.08±613.64	496.50			0.168
RBC count (millions/cu.mm)	4.84±0.05	4.25±0.19		3.10	13.71	0.008
Differential Leucocyte Count	•					
Packed Cell Volume (PCV) (%)	41.85±0.54	36.58±1.33	1019.00			0.001
Mean Corpuscular Volume (MCV) (fl)	86.31±0.74	86.91±2.32	618.50			0.780
Mean Corpuscular Haemoglobin (MCH) (pg)	28.36±0.28	28.75±0.87	547.50			0.359
Mean Corpuscular Haemoglobin Concentration (MCHC) (%)	32.74±0.08	33.09±0.52	546.00			0.351
Red Cell Distribution Width (RDW)	14.30±0.17	14.28±0.85	809.50			0.152
Platelet Count (Lakhs/ c.mm)	2.61±0.08	2.68±0.14	617.00			0.770
Neutrophil (%)	56.73±0.77	55.57±2.77	678.00			0.805
Eosinophil (%)	4.25±0.36	4.57±0.67	540.00			0.324
Basophil (%)	0.60±0.03	0.49 ± 0.04	781.50			0.235
Lymphocyte (%)	30.95±0.68	34.22±3.67	590.50			0.596
Monocyte (%)	7.46±0.16	8.09±0.57	526.00			0.266
Volume, Conductivity and Scatter Para	meter					
Absolute neutrophil count	4.39±0.18	4.63±0.43	571.00			0.480
Absolute lymphocyte count	2.26±0.06	2.40±0.24	609.50			0.718
Neutrophil Conductance, SD	5.01±0.07	5.38±0.24	486.50			0.142
Neutrophil Scatter, Mean	148.78±0.48	147.56±1.76	726.00			0.497
Neutrophil Scatter, SD	9.92±0.12	10.17±0.57	699.50			0.659
Lymphocyte volume, SD	13.91±0.12	15.07±0.64	406.00			0.028
Lymphocyte Conductance, SD	10.20±0.18	10.79±0.85	704.50			0.627
Lymphocyte Scatter, SD	16.23±0.19	17.25±0.49	399.50			0.024
N/L Ratio	2.03±0.09	2.10±0.25	603.50			0.679

Table 5. Comparison of total blood count, leucocyte count and Volume, Conductivity and Scatter (VCS) parameters between control subjects and gastritis patients with severe activity.

Parameters	Control Subjects	Gastritis Patients	Test For statistical significance			ificance
	Mean±S.E.	Mean±S.E.	W	t-test	df	p-value
Haemoglobin (Hb %)	13.72±0.19	13.70±0.60	461.00			0.908
Total Count						
Total Leukocyte count (Thousands/cu.mm)	7533.00±221.21	8966.67±1064.71	307.00			0.116
RBC count (millions/cu.mm)	4.84 ± 0.05	4.89±0.23		-0.22	8.72	0.832
Differential Leucocyte Count						
Packed Cell Volume (PCV) (%)	41.85±0.54	41.60±1.65	475.50			0.783
Mean Corpuscular Volume (MCV) (fl)	86.31±0.74	85.47±1.74	523.00			0.425
Mean Corpuscular Haemoglobin (MCH) (pg)	28.36±0.28	28.11±0.54	520.50			0.441
Mean Corpuscular Haemoglobin Concentration (MCHC) (%)	32.74±0.08	32.92±0.46	350.00			0.273
Red Cell Distribution Width (RDW)	14.30±0.17	14.39±0.35	372.50			0.396
Platelet Count (Lakhs/ c.mm)	2.61±0.08	2.65±0.22	459.00			0.925
Neutrophil (%)	56.73±0.77	61.04±4.94	380.50			0.447
Eosinophil (%)	4.25±0.36	5.21±1.70	425.50			0.792
Basophil (%)	0.60±0.03	0.66±0.17	433.00			0.855
Lymphocyte (%)	30.95±0.68	25.53±4.20	576.50			0.165
Monocyte (%)	7.46±0.16	7.56±0.80	454.50			0.965
Volume, Conductivity and Scatter Pa	rameter					
Absolute neutrophil count	4.39±0.18	6.12±1.40	333.00			0.199
Absolute lymphocyte count	2.26±0.06	2.18±0.33	493.00			0.639
Neutrophil Conductance, SD	5.01±0.07	5.90±0.34	199.00			0.006
Neutrophil Scatter, Mean	148.78±0.48	144.13±2.42	629.50			0.049
Neutrophil Scatter, SD	9.92±0.12	10.69±0.47	293.50			0.086
Lymphocyte volume, SD	13.91±0.12	14.17±0.48	418.00			0.729
Lymphocyte Conductance, SD	10.20±0.18	11.76±0.80	228.00			0.015
Lymphocyte Scatter, SD	16.23±0.19	17.57±0.58	229.50			0.015
N/L Ratio	2.03±0.09	4.28±2.03	334.00			0.204

(moderate activity gastritis patients) (Table 4). Whereas lymphocyte volume SD and lymphocyte scatter SD were significantly higher among gastritis patients with moderate infiltration of neutrophils in the lamina propria of gastric mucosa (moderate activity gastritis patients) (Table 4).

Comparison of total blood count, leucocyte count and Volume, Conductivity and Scatter (VCS) parameters between control subjects and gastritis patients with severe activity

None of the CBC parameters showed significant variations between the control subjects and gastritis patients with high infiltration of neutrophils in the lamina propria of the gastric mucosa (high-activity gastritis patients) (Table 5). On the contrary, scatter parameters such as neutrophil conductance SD, lymphocyte conductance SD and lymphocyte scatter SD were significantly higher among gastritis patients with high infiltration of neutrophils in the lamina propria of the gastric mucosa (high activity gastritis patients) (Table 5). However, the mean neutrophil scatter was significantly higher among control subjects compared to gastritis patients with high infiltration of neutrophils in the lamina propria of the gastric mucosa (high-activity gastritis patients) (Table 5).

Discussion

During our study, we recorded persistently low RBC percentages among gastritis patients compared to the control subjects. This low RBC count may have resulted from the gradual blood loss due to erosive and ulcerative gastritis triggered by *Hp* infection. Such prolonged loss of haemoglobin leads to microcytic hypochromic anaemia (iron deficiency). This has been highlighted by the MCV values and associated red cell distribution width (RDW) in gastritis patients. A similar observation has also been reported by Li et al. (2017).

We noticed a marked increase in the total leukocyte count among the gastritis patients, possibly related to the increase in the neutrophil population. Neutrophils are recruited to neutralise the inflammation caused Hp in the gastric mucosa. Considering that the Hp affects the external layer of the gastric mucosa, the neutrophils often fail to neutralise the bacteria properly and subsequent bactericidal actions of the mediators tend to harm the normal tissues. Guclu et al. (2017) showed that an increase in the intensity of Hp infection tends to reduce the neutrophil-lymphocyte ratio, which indicates increased lymphocyte presence among infected individuals. However, we observed that N/L ratio is increased among general gastritis patients and patients

with no to high intensity of Hp infections. Our observations are in concurrence with Farah et al. (2017). They also suggested that there is an appreciable link between Hp infection and inflammation-based NLR, but the study also indicated that the escalated N/L ratio gets normalized with treatment. Our study shows that there is a perceptible increase in the absolute neutrophil count that may be attributed to increasing Hp intensity. We observed that the intensity of Hp presence in the mucosa is also related to the severity of tissue inflammation in the infected individuals. We also observed that the percentage of lymphocytes in peripheral blood decreased in the initial stages, and this may be attributed to the subsequent increase of neutrophils. Based on our observation, we propose that the N/L ratio shows signs of escalation due to the increased severity of the inflammation and intensified neutrophil infiltration of the gastric mucosa.

Conclusion

In this study, we found that there was a correlation between the Hp in the mucosa and the severity of tissue inflammation. Moderate increase in the intensity of Hp infection does not lead to a significant change in N/L ratio. We observed an increased neutrophil-lymphocyte ratio (NLR) that helps to identify the type of gastritis by ruling out the autoimmune cause where lymphocytes show higher values. Though this study has not taken up autoimmune gastritis cases, the N/L ratio will definitely give clues to categorize the type of gastritis. The volume and conductance of neutrophils were enhanced, and their graph presents the left shift in cases of gastritis patients, which denotes diagnostic importance. The neutrophil scatter means that SD strengthens gastritis patients, symbolising the systemic inflammatory influence on these cases. So, analysing complete blood count with N/L ratio and a thorough analysis of the hemogram of cases in gastritis gives a clear advantage in knowing the probable cause, the grade of inflammation (severity scale) and treatment progression.

Funding

The grants from ICMR supported this work. The funding body aided in collecting, analysing and interpreting data and in writing the manuscript.

Conflict of interest

None

Acknowledgement

It is my pleasure to thank Chettinad Hospital and Research Institute for their support. Our thanks also go to the study participants, who consented to the present study assessing the data they provided.

Int. J. Exp. Res. Rev., Vol. 30: 46-56 (2023)

References

Choccalingam, C. (2018).Volume, conductance, and scatter parameters of neoplastic and nonneoplastic lymphocytes using Coulter LH780. *J. Lab. Physicians.*, 10(1), 85-88.
https://doi.org/10.4102/U.D.U.D.U.D. 65, 17

https://doi.org/10.4103/JLP.JLP_65_17

- Farah, R., & Khamisy-Farah, R. (2014). Association of neutrophil to lymphocyte ratio with presence and severity of gastritis due to *Helicobacter pylori* infection. J. Clin. Lab. Anal., 28(3), 219-23. https://doi.org/10.1002/jcla.21669
- Forget, P., Khalifa, C., Defour, J.P., Latinne, D., Van-Pel, M.C., & De Kock, M. (2017). What is the normal value of the neutrophil-to-lymphocyte ratio? *BMC Res. Notes*, 10(1), 12.

https://doi.org/10.1186/s13104-016-2335-5

- Guclu, M., & Faruq-Agan, A. (2017). Association of Severity of *Helicobacter pylori* Infection with Peripheral Blood Neutrophil to Lymphocyte Ratio and Mean Platelet Volume. *Euroasian J. Hepatogastroenterol.*, 7(1), 11-16. https://doi.org/10.5005/jp-journals-10018-1204
- Hamlet, A., Thoreson, A.C., Nilsson, O., Svennerholm, A.M., & Olbe, L. (1999). Duodenal *Helicobacter pylori* infection differs in cagA genotype between asymptomatic subjects and patients with duodenal ulcers. *Gastroenterology*, *116*(2), 259-268. https://doi.org/10.1016/S0016-5085(99)70121-6
- Hopkins, R.J., Girardi, L.S., & Turney, E.A. (1996). Relationship between *Helicobacter pylori* eradication and reduced duodenal and gastric ulcer recurrence: a review. *Gastroenterology*, *110*(4),1244-1252.

https://doi.org/10.1053/gast.1996.v110.pm8613015

- Horne, B.D., Anderson, J.L., John, J.M., Weaver, A., Bair, T.L., Jensen, K.R., Renlund, D.G., Muhlestein, J.B. (2005). Intermountain Heart Collaborative Study Group. Which white blood cell subtypes predict increased cardiovascular risk? *J. Am. Coll. Cardiol.*, 45(10), 1638-1643. https://doi.org/10.1016/j.jacc.2005.02.054
- Hunt, R.H. (1996). The role of *Helicobacter pylori* in pathogenesis: the spectrum of clinical outcomes. *Scand. J. Gastroenterol. Suppl.*, 220, 3-9. https://doi.org/10.3109/00365529609094743
- Jafarzadeh, A., Hassanshahi, G.H., & Nemati, M. (2009). Serum levels of high-sensitivity C-reactive protein (hs-CRP) in *Helicobacter pylori*-infected peptic ulcer patients and its association with bacterial CagA virulence factor. *Dig. Dis. Sci.*, 54(12), 2612-6. https://doi.org/10.1007/s10620-008-0686-z

Kim, H.K., Jang, E.C., Yeom, J.O., Kim, S.Y., Cho, H., Kim, S.S., Chae, H.S., & Cho, Y.S. (2013). Serum prohepcidin levels are lower in patients with atrophic gastritis. *Gastroenterol. Res. Pract.*, 2013, 201810. https://doi.org/10.1155/2013/201810

- Kuipers, E.J., Thijs, J.C., & Festen, H.P. (1995). The prevalence of *Helicobacter pylori* in peptic ulcer disease. *Aliment Pharmacol. Ther.*, *Suppl.*, 2, 59-69.
- Li, N., Zhou, H., & Tang, Q. (2017). Red Blood Cell Distribution Width: A Novel Predictive Indicator for Cardiovascular and Cerebrovascular Diseases. *Dis. Markers.*, 2017, 7089493. https://doi.org/10.1155/2017/7089493
- Makola, D., Peura, D.A., & Crowe, S.E. (2007). *Helicobacter pylori* infection and related gastrointestinal diseases. J. Clin. Gastroenterol., 41(6), 548-558.

https://doi.org/10.1097/MCG.0b013e318030e3c3

- Malaty, H.M. (2007). Epidemiology of Helicobacter pylori infection. Best Pract. Res. Clin. Gastroenterol., 21(2), 205-214. https://doi.org/10.1016/j.bpg.2006.10.005
- Mehmet, N., Refik, M., Harputluoglu, M., Ersoy, Y., Aydin, N.E., & Yildirim, B. (2004). Serum and gastric fluid levels of cytokines and nitrates in gastric diseases infected with *Helicobacter pylori*. *New Microbiol.*, 27(2), 139-48.
- Naito, Y., Ito, M., Watanabe, T., & Suzuki, H. (2005). Biomarkers in patients with gastric inflammation: a systematic review. *Digestion.*, 72(2-3), 164-180. https://doi.org/10.1159/000088396
- Priyadharshini, V., Dhande, S.K., Hanumanram, G., Surya, S.G.S., & Shankar, G.A. (2022). Analysis of the relation between elevated neutrophil lymphocyte ratio, and erythrocyte sedimentation rate in *Helicobacter pylori* positive chronic gastritis patients. *Int. J. Adv. Med.*, 9, 1023-1026. https://doi.org/10.18203/2349-3933.ijam20222400
- Ruggiero, P. (2010). *Helicobacter pylori* and inflammation. *Curr. Pharm. Des.*, *16*(38), 4225-4236. https://doi.org/10.2174/138161210794519075
- Satoh, Y., Ogawara, H., Kawamura, O., Kusano, M., & Murakami, H. (2012). Clinical Significance of Peripheral Blood T Lymphocyte Subsets in *Helicobacter pylori*-Infected Patients. *Gastroenterol. Res. Pract.*, 2012, 819842. https://doi.org/10.1155/2012/819842
- Thompson, C.B., Eaton, K.A., Princiotta, S.M., Rushin, C.A., Valeri, C.R. (1982). Size dependent platelet subpopulations: relationship of platelet volume to

ultrastructure, enzymatic activity, and function. Br. J. Haematol., 50(3), 509-519.

https://doi.org/10.1111/j.1365-2141.1982.tb01947.x

- Umit, H., & Umit, E.G. (2015). Helicobacter pylori and mean platelet volume: A relation way before immune thrombocytopenia? Eur. Rev. Med. Pharmacol. Sci., 19, 2818-2823.
- Winter, C., Hartl, S., Kolb, D., Leitinger, G., & Roblegg, E. (2020). Investigations to Evaluate Gastric Mucoadhesion of an Organic Product to Ameliorate Gastritis. Pharmaceutics, 12(4), 331. https://doi.org/10.3390/pharmaceutics12040331

How to cite this Article:

Suresh Kumar Sinduja and Rajesh Kanna Nandagopal Radha (2023). Analysing the association of Helicobacter pylori induced gastritis with neutrophil-lymphocyte ratio (NLR) and volume, conductance, scatter characteristics of LH780 Coulter. International Journal of Experimental Research and Review, 30, 46-56.

DOI: https://doi.org/10.52756/ijerr.2023.v30.005



This work is licensed under a Creative Commons Attribu-tion-NonCommercial-NoDerivatives 4.0 International License.