



# INTERNATIONAL JOURNAL OF RESEARCH IN PHARMACEUTICAL SCIENCES

Published by JK Welfare &amp; Pharmascope Foundation

Journal Home Page: <https://ijrps.com>

## Interpretations of hematological findings among Iraqi children with celiac disease in Kerbala city

Lames H. Almanseekanaa<sup>\*1</sup>, Raed H. Ogaili<sup>2</sup>, Sameem S. M. Baker<sup>3</sup><sup>1</sup>Department of Basic Medical Science, College of Dentistry, Kerbala University, Iraq<sup>2</sup>Department of Oral & Maxillofacial Surgery, College of Dentistry, Kerbala University, Iraq<sup>3</sup>Departments of Dentistry, Ibn Hayyan University College, Iraq

### Article History:

Received on: 23.09.2018

Revised on: 16.12.2018

Accepted on: 19.12.2018

### Keywords:

Celiac disease,  
Iron deficiency anaemia,  
Gluten intolerance

### ABSTRACT

Celiac Disease is a common systemic disorder including immunologic, genetic, and environmental factors. To describe the different haematological finding of Celiac Disease in Karbala city. This is a cross-sectional study conducted in the Children Teaching Hospital in Karbala city comprising data sheets of complete blood count (CBC) for 119 children, 70 males and 59 females (with the average age of 7). Tissue transglutaminase antibody (IgA and IgG) are performed for all patients after recording their history and clinical examination. CBC results are divided into three categories as 1) red blood cells (RBC) showing iron deficiency or anaemia, 2) white blood cells (WBC) showing the status of disease if it's in the acute or chronic state serological obtained from tissue transglutaminase antibody (IgA and IgG) and 3) clinical examination providing a new scientific explanation for the samples bordered to detect IgA and IgG. Celiac Disease is associated with a diversity of haematological findings especially Iron deficiency anaemia characterised by no response to iron supplement, leukopenia, lymphopenia, neutropenia, and eosinophilia, as well as thrombocytopenia and thrombocytosis. Due to the interaction between the clinical signs of food intolerance against gluten and celiac disease, few other studies have been suggested.



### \* Corresponding Author

Name: Lames H. Almanseekanaa  
Email: [lames.h@uokerbala.edu.iq](mailto:lames.h@uokerbala.edu.iq)

ISSN: 0975-7538

DOI: <https://doi.org/10.26452/ijrps.v10i1.1894>

Production and Hosted by

IJRPS | <https://ijrps.com>

© 2019 | All rights reserved.

### INTRODUCTION

Celiac Disease is a common systemic disorder with immunologic, genetic, and environmental factors including 1% incidence rate of the population. Celiac Disease patients have a long duration of symptoms before diagnosis; the disease is considered to be underdiagnosed. The diagnostic period between the beginning of symptoms and diagnosis of Celiac Disease is still unacceptably long. One of the

most common problems in Celiac Disease diagnosis is the long duration between the beginning of symptoms and diagnosis of community awareness expected to be increased to decrease this interval so that the symptoms might be diagnosed in first phases by patients and physicians. Disability of iron absorption leading to anaemia is diagnosed in Celiac Disease (Bao *et al.*, 1999). Therefore, the iron malabsorption symptom has been continued for different times after starting of the treatment with food free gluten (Bingley, Norcross, Lock, Ness, & Jones, 2004).

Iron deficiency anaemia recorded from school students and adults have strong prevalence to show the symptoms of Celiac Disease (Bottaro, Cataldo, Rotolo, Spina, & Corazza, 1999; Carroccio *et al.*, 2002). Celiac Disease in children has an arousing curiosity relation with their iron malabsorption and deficiency maintaining less estimation between the haematologists as well as subspecialty physicians (Cook, 2005). Hematopoietic deficiency

seen in Celiac Disease patients has appeared as an unexpected experience in undiagnosed adults (Carmel, 2004) so that the mentioned deficiency in Celiac Disease has shown multifactorial causes.

The Hematopoietic deficiency in Celiac Disease is described as hyperproliferative haemoglobin in the intestinal mucosa, leading to malabsorption of iron and other essential nutrients such as vitamins, folate and cobalamin appeared to ameliorate after starting a gluten-free diet (Dahele & Ghosh, 2001). One of the most important factors that explain the mechanism of anaemia in Celiac Disease is abnormal iron absorption; respectively, Iron is absorbed in the first part of small intestine depending on several factors such as the intraluminal intestinal pH and intact mucosal surface. In CD patient, the intestinal mucosa is Villous atrophy negatively reflected in iron absorption (Poggi, Conenna, Fiorillo, & Scippa, 1992).

### Aims of the study

The purpose of this study is to interpret the relationship among the haematological findings, serological results and clinical symptoms of the children with Celiac Disease in Karbala.

### METHODS

The current study has been conducted at AL-Hussain Medical City and Children Teaching Hospital as a tertiary teaching hospital for children in Karbala, Iraq. The study samples are 111 children (2-12 years old) admitted to the hospital in three months (between July - September of 2017).

Based on the clinical examination, children have been suffered from symptoms suspected to be celiac. To perform the haematological and serological investigation, the blood samples are collected in labelled 5ml tubes, stored and delivered at 4°C cool containers. Also, the sera are stored in 0.2 ml aliquots at -20°C till testing.

Red blood cells, White blood cells, Hemoglobin, Hematocrit, MCV, MCH, MCHC, RDW, and Platelets are measured through the tests. Subsequently, human tissue trans-glutaminase IgG and IgA antibodies are measured according to - Euroimmun Medizinische Labordiagnostik AG (ELIZA) by Luebeck, Germany. Test process has started by adding 100uL of calibrated, positive or negative or diluted patient samples to each well plate, then incubated for 30 minutes at room temperature (+18°C bis 25 C) emptying the wells and rinsing 3 times by 300 ul washing buffer/wash, then leaving the wash buffer in each well for 30 to 60 seconds per rinsing cycle. 100µL of the conjugated enzyme (peroxidase-labelled anti-human IgA) is added to each plate and incubated for 30 minutes at 37°C (+18°C bis 25 C) while empty and rinsed. Further,

100 µL chromogen/substrate solution has been added to each plate and incubated for 15 minutes at 37°C (+18°C bis 25°C) followed by the addition of 100 ul of stop solution to each plate. The optical density (OD value) of each well plate is measured in 30 minutes of adding stop solution by using a microplate reader set at 620-650 nm resulted in pg/mL" (Meyer, Scheper, Lehmann & Stocker, 2003).

### RESULTS

**Table 1: Hematological results in female-group**

Test Name	No. of children with abnormal results
RBC (Erythrocytes)	31
HCT (Haematocrit)	58
HGB (Hemoglobin)	55
MCV	43
MCH	59
MCHC	41
RDW-SD	31
RDW-CV	30
WBC (Leukocyte)	23
NEU %	23
NEU #	15
LYM %	35
LYM #	30
MON#	14
MON%	20
EOS#	23
EOS%	11
BASO#	0
BASO%	0
PLT (Platelet Count)	10
MPV	11
PDW	16
PCT	13
P-LCR	21

119 children, 70 males and 59 females (with an average age of 7 years old) have been participated to AL-Hussain Medical Hospital for Children in Karbala for three months (July - September of 2017). They are suffered from various occasional symptoms such as gaseousness, bloating, nausea and vomiting as transitory, esophagus spasm, difficulty in swallowing (dysphagia), noncardiac chest pain, abdominal pain, unexplained weight loss, and constipation or diarrhoea. All haematological results are measured by automated haematology device through the Sysmex kit. During the test, most children have positive tissue transglutaminase antibody IgA, IgG or both as a result of the serological examination. The haematological finding represented by CBC results (complete blood count) (Table, 1 & Table, 2) are lower than normal value. Both groups have been suffered from anaemia as a result of the low concentration of haemoglobin. Accordingly, the haemoglobin concentration of 64

samples from 70 male children and 55 samples from 59 female children is lower than normal value synchronized with HCT percentage (Hematocrit) and RBC numbers (Erythrocytes) for both genders. Regarding all samples, there is a decline in haemoglobin concentration (hypochromic) in 59 females and 70 males. Meanwhile, small cells size (microcytic) is also seen in 63 samples from 70 male children align with 43 samples of 59 children from another group.

**Table 2: Hematological results in male-group**

Test Name	Abnormal results
RBC (Erythrocytes)	34
HCT (Haematocrit)	67
HGB (Hemoglobin)	64
MCV	63
MCH	70
MCHC	43
RDW-SD	45
RDW-CV	42
WBC (Leukocyte)	23
NEU %	34
NEU #	15
LYM %	36
LYM #	48
MON#	30
MON%	43
EOS#	13
EOS%	18
BASO#	0
BASO%	2
PLT (Platelet Count)	12
MPV	11
PDW	2
PCT	14
P-LCR	21

Regarding leukocytes account, there is a high increment in lymphocytes (lymphocytosis), monocyte (monocytosis), and eosinophilia (eosinophilia).

## DISCUSSION

Celiac Disease is an autoimmune inflammatory disease in the small intestine (Nelsen, 2002). Also, anaemia is the most common laboratory manifestation of celiac disease (Iovino *et al.*, 1998; Clemens, 1996). According to the haematological results, there is a decline in erythrocyte numbers, haemoglobin concentration and hematocrit level. These results have been strongly related to Iron deficiency anaemia characterised by no response to iron supplement as chronic cases. Therefore, this type of anemia might be considered as the first choice in diverse diagnosis for Celiac Disease identified through serological laboratory tests (tissue transglutaminase antibodies) and histopathol-

ogy examination (Bao *et al.*, 1999). Leukopenia, especially neutropenia, lymphopenia, eosinophilia and thrombocytosis plus clinical signs of Celiac Disease are aligned with acute and chronic cases. To sum up, the majority of children who are sensitive to gluten are diagnosed during chronic cases. Therefore, CBC interpretation in reducing white blood cells (leukopenia), especially lymphopenia, and neutropenia have completely matched to these results.

## REFERENCES

- Bao, F., Yu, L., Babu, S., Wang, T., Hoffenberg, E. J., Rewers, M., & Eisenbarth, G. S. 1999. One-third of HLA DQ2 homozygous patients with type 1 diabetes express celiac disease-associated transglutaminase autoantibodies. *Journal of Autoimmunity*, 13(1), 143-148.
- Bingley, P. J., Norcross, A. J., Lock, R. J., Ness, A. R., & Jones, R. W. 2004. Undiagnosed coeliac disease at age seven: population based prospective birth cohort study. *BMJ*, 328(7435), 322-323.
- Bottaro, G., Cataldo, F., Rotolo, N., Spina, M., & Corazza, G. R. 1999. The clinical pattern of subclinical/silent celiac disease: an analysis of 1026 consecutive cases. *The American Journal of Gastroenterology*, 94(3), 691-696.
- Carmel, R. 2004. Megaloblastic anaemias: disorders of impaired DNA synthesis. *Wintrobe's Clinical Hematology*, 1, 1367-1395.
- Carroccio, A., Giannitrapani, L., Di Prima, L., Iannitto, E., Montalto, G., & Notarbartolo, A. 2002. Extreme thrombocytosis as a sign of coeliac disease in the elderly: case report. *European Journal of Gastroenterology & Hepatology*, 14(8), 897-900.
- Clemens, P. 1996. Coeliac disease in adults with atypical symptoms. *The Lancet*, 347(9007), 1050.
- Cook, J. D. 2005. Diagnosis and management of iron-deficiency anaemia. *Best Practice & Research Clinical Haematology*, 18(2), 319-332.
- Croese, J., Harris, O., & Bain, B. 1979. Coeliac disease. Haematological features, and delay in diagnosis. *The Medical Journal of Australia*, 2(7), 335-338.
- Dahele, A., & Ghosh, S. 2001. Vitamin B 12 deficiency in untreated celiac disease. *The American Journal of Gastroenterology*, 96(3), 745.
- Iovino, P., Ciacci, C., Sabbatini, F., Acioli, D. M., D'Argenio, G., & Mazzacca, G. 1998. Esophageal impairment in adult celiac disease with steatorrhea. *The American Journal of Gastroenterology*, 93(8), 1243-1249.

- Meyer, W., Scheper, T., Lehmann, H., & Stocker, W. 2003. EUROIMMUN Medizinische Labordiagnostika AG. Selbstklebende Blotmembranen. Registered German utility model DE, 202(15), 268-5.
- Nelsen, J. D. A. 2002. Gluten-sensitive enteropathy (celiac disease): more common than you think. *American Family Physician*, 66(12), 2259-2266.
- Poggi, V., Conenna, R., Fiorillo, A., & Scippa, L. 1992. Iron absorption and iron deficiency in infants and children with gastrointestinal diseases. *Journal of Pediatric Gastroenterology and Nutrition*, 14(1), 21-26.