

ORIGINAL RESEARCH

Celiac disease in children with iron-deficiency anemia

Dr. Prabhat Kumar

Assistant Professor, Department of Paediatrics, Hind Institute of Medical Sciences, Barabanki, UP, India

Corresponding Author

Dr. Prabhat Kumar

Assistant Professor, Department of Paediatrics, Hind Institute of Medical Sciences, Barabanki, UP, India

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ABSTRACT

Background: Unknown iron deficiency anemia without symptoms related to the gastrointestinal tract is a well-known sign of celiac disease (CD). The present study was conducted to assess celiac disease in children with iron-deficiency anemia. **Materials & Methods:** 66 children of 1 to 14 years of age with iron deficiency anemia of both genders were divided into 2 groups of 33 each. Group I comprised children with anemia and group II had healthy children without anemia. **Results:** Group I had 20 males and 13 females and group II had 18 males and 15 females. The mean height for age (z score) was -1.42 and -1.14, weight for age (z score) was -1.04 and -0.85, weight for height (z score) was -0.46 and -0.17 and midarm circumference was 15.1 cm and 16.2 cm in group I and II respectively. The difference was significant ($P < 0.05$). **Conclusion:** Children who are anemic are much more likely to have celiac disease.

Key words: celiac disease, Children, iron deficiency anemia

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INTRODUCTION

Unknown iron deficiency anemia without symptoms related to the gastrointestinal tract is a well-known sign of celiac disease (CD). This autoimmune enteropathy is typified by the development of malabsorption due to villus injury brought on by the humoral and cellular immune system's activation against gluten, which is widely present in wheat, barley, and rye.¹ However, there is a dearth of information, particularly from countries like India where nutritional anemias are prevalent, about the proportionate contribution of CD to unexplained anemia in children. Significant dietary changes are necessary for children with CD in addition to nutritional anemia treatment.²

Iron, folic acid, and vitamin B12 malabsorption causes anemia frequently in people with CD. The most frequent hematological consequence of CD is anemia, which has a prevalence of 12% to 69% upon diagnosis.³ Furthermore, it can be the first clinical observation of silent or subclinical CD. The most prevalent type of anemia is called iron deficiency anemia (IDA), which is typically linked to increased iron loss or decreased iron absorption.⁴ Furthermore, the most frequent extraintestinal sign of CD is IDA unresponsive to oral iron supplementation, which may

be the only indication of the illness in cases when there is no evident malabsorption. The main cause of the iron deficit in CD is the enteropathy, which is characterized by small intestine mucosal destruction that impairs iron absorption. However, occult blood loss in the gastrointestinal tract may also be present.⁵ The present study was conducted to assess celiac disease in children with iron-deficiency anemia.

MATERIALS & METHODS

The present study comprised of 66 children of 1 to 14 years of age with iron deficiency anemia of both genders. Parental consent was obtained before starting the study.

Data such as name, age, gender etc. was recorded. Patients were divided into 2 groups of 33 each. Group I comprised children with anemia and group II had healthy children without anemia. Both groups' serum IgA-tissue trans-glutaminase levels were measured. Upper gastrointestinal endoscopy and duodenal biopsy were performed on all children whose celiac serology tested positive; a biopsy result of Marsh grade 3 was deemed indicative of celiac disease. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS**Table I Distribution of patients**

Groups	Group I (Anemia) (33)	Group II (Without anemia) (33)
M:F	20:13	18:15

Table I shows that group I had 20 males and 13 females and group II had 18 males and 15 females.

Table II Baseline parameters

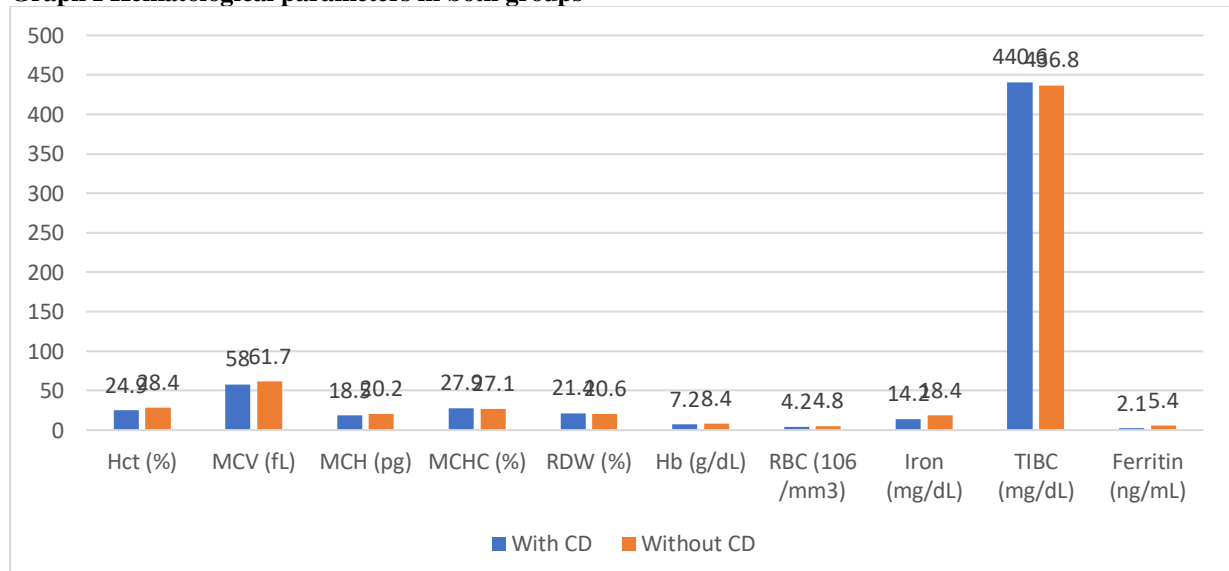
Parameters	Group I	Group II	P value
Height for age (z score)	-1.42	-1.14	0.03
Weight for age (z score)	-1.04	-0.85	0.05
weight for height (z score)	-0.46	-0.17	0.03
Mid-arm circumference (cm)	15.1	16.2	0.04

Table II shows that mean height for age (z score) was -1.42 and -1.14, weight for age (z score) was -1.04 and -0.85, weight for height (z score) was -0.46 and -0.17 and midarm circumference was 15.1 cm and 16.2 cm in group I and II respectively. The difference was significant ($P < 0.05$).

Table III Hematological parameters in both groups

Parameters	With CD (21)	Without CD (45)	P value
Hct (%)	24.9	28.4	0.05
MCV (fL)	58.0	61.7	0.98
MCH (pg)	18.5	20.2	0.04
MCHC (%)	27.9	27.1	0.19
RDW (%)	21.4	20.6	0.12
Hb (g/dL)	7.2	8.4	0.05
RBC (106 /mm ³)	4.2	4.8	0.94
Iron (mg/dL)	14.2	18.4	0.05
TIBC (mg/dL)	440.6	436.8	0.65
Ferritin (ng/mL)	2.1	5.4	0.01

Table III, graph I shows that mean Hct (%) was 24.9 and 28.4, MCV (fL) was 58.0 and 61.7, MCH (pg) was 18.5 and 20.2, MCHC (%) was 27.9 and 27.1, RDW (%) was 21.4 and 20.6, hemoglobin (g/dL) was 7.2 and 8.4, RBC (106 /mm³) was 4.2 and 4.8, iron (mg/dL) was 14.2 and 18.4, TIBC (mg/dL) was 440.6 and 436.8 and ferritin (ng/mL) was 2.1 and 5.4 in group I and II respectively. The difference was significant ($P < 0.05$).

Graph I Hematological parameters in both groups**DISCUSSION**

A chronic enteropathy affecting the proximal small intestine, celiac disease (CD) is typified by a persistent intolerance to gluten resulting from dietary consumption of gluten-containing foods in people

who are genetically predisposed to the condition.⁶

There are two clinical subgroups that have been identified for CD presentation. Weight loss, abdominal distention, persistent diarrhea, and failure to flourish are typical CD presenting symptoms.⁷ The

majority of people with CD have the silent or atypical (subclinical) type of the illness. Symptoms of CD might appear slowly, such as osteoporosis, neurological symptoms, iron-deficiency anemia (IDA), or cryptogenic hypertransferrinemia. Anemia is the most prevalent hematologic condition that CD is known to cause. Micronutrient malabsorption, including that of iron, folic acid, and vitamin B12, is typically the cause of anemia in CD patients.⁸The most prevalent form of anemia in humans, iron deficiency anemia (IDA) is typically brought on by either increased iron loss or decreased iron absorption. One typical extraintestinal symptom of CD is IDA resistance to oral iron supplementation, which has been characterized as the only extraintestinal symptom of the disease without overt malabsorption.^{9,10} The present study was conducted to assess celiac disease in children with iron-deficiency anemia.

We found that group I had 20 males and 13 females and group II had 18 males and 15 females. Kalayci et al¹⁰ determined the prevalence of coeliac disease in children with iron deficiency anaemia without significant gastrointestinal symptoms. There were 135 children with iron deficiency anaemia in the patient group (group 1), and 223 healthy children without iron deficiency anaemia in the control group (group 2). Antiendomysial antibody (EMA) IgA test was given to both groups. Antiendomysial antibody-positive patients underwent small intestine biopsy. The mean age was 7.2±4.6 (2-16) y in the patient group (group 1) and 8.2±3.8 (2-16) y in the control group (group 2), and no significant difference between the two groups was detected. In terms of gender, there was a significant difference between groups 1 and 2 (M/F: 74/61 and 98/125, respectively). EMA was positive in six cases in group 1 (4.4%), and villous atrophy and/or inflammation in the lamina propria with increased intraepithelial lymphocytes was seen on small intestine biopsy in these patients. In the control group, EMA was negative in all children. In detailed histories of patients with coeliac disease diagnosis, recurrent iron deficiency anaemia/pica was found in four patients (66.7%) and occasionally foul-smelling or watery stool attacks were seen in four patients (66.7%). Three of these six patients (50%) had short stature.

We observed that mean height for age (z score) was -1.42 and -1.14, weight for age (z score) was -1.04 and -0.85, weight for height (z score) was -0.46 and -0.17 and midarm circumference was 15.1 cm and 16.2 cm in group I and II respectively. Haapalahti et al¹¹ described the nutritional status in patients with screen-detected celiac disease (CD). Nutritional status was assessed by serum tests and anthropometric measures in 26 subjects (16 to 25 years of age) with biopsy-proven CD and 29 healthy control subjects (16 to 21 years of age) with negative tissue transglutaminase antibodies (16 to 22 years of age); all the subjects were selected from the cohort of 3654

schoolchildren. Compared with control subjects, CD patients had lower median values of whole blood folic acid (91 versus 109 nmol/L; P = 0.01), serum ferritin (14 versus 27 microg/L; P = 0.028) and pre-albumin (0.21 versus 0.28 g/L; P ≤ 0.001) and higher transferrin receptor (1.3 versus 1.1; P = 0.008) and serum transferrin receptor-ferritin index (1.2 versus 0.7; P = 0.006). Folic acid concentration was subnormal in 31% of the CD subjects (versus 14% of the controls) and iron status (transferrin receptor-ferritin index) was subnormal in 30% (versus 14%). Body mass index was not different in females of the CD and control groups (22 versus 22 kg/m²) or in the males of the respective groups (25 versus 24 kg/m²). Females with CD were shorter than the controls (mean 162 versus 167 cm; P = 0.018), but no difference was found in males. No association was found between the nutritional status and the markers of mucosal injury (villous-crypt measures), but titer of transglutaminase was associated with whole blood folic acid (r = -0.5; P = 0.016) and with transferrin receptor-ferritin index (r = 0.4, P = 0.05).

CONCLUSION

Authors found that children who are anemic are much more likely to have celiac disease.

REFERENCES

1. Corazza GR, Valentini RA, Andreani ML, et al. Subclinical coeliac disease is a frequent cause of iron-deficiency anaemia. *Scand J Gastroenterol.* 1995;30:153-156.
2. McIntyre AS, Long RG. Prospective survey of investigations in outpatients referred with iron deficiency anaemia. *Gut.* 1993;34:1102-1107.
3. Mandal AK, Mehdi I, Munshi SK, et al. Value of routine duodenal biopsy in diagnosing coeliac disease in patients with iron deficiency anaemia. *Postgrad Med J.* 2004;80:475-477.
4. Emami MH, Karimi S, Kouhestani S. Is routine duodenal biopsy necessary for the detection of celiac disease in patients presenting with iron deficiency anemia? *Int J Prev Med.* 2012;3:273-277.
5. Hill ID, Dirks MH, Liptak GS, Colletti RB, Fasano A, Guandalini S, et al. Guideline for the Diagnosis and Treatment of Celiac Disease in Children: Recommendations of the North American Society for Pediatric Gastroenterology, Hepatology. *J Pediatr Gastroenterol Nutr.* 2005;40:1-19.
6. Bainton DF, Finch CA. The diagnosis of iron deficiency anemia. *Am J Med.* 1964;37:62-70.
7. Marsh MN, Crowe PT. Morphology of the mucosal lesion in gluten sensitivity. *Baillieres Clin Gastroenterol.* 1995;9:273-93.
8. Kapur G, Patwari AK, Narayan S, et al. Iron supplementation in children with celiac disease. *Indian J Pediatr.* 2003;70:955-958.
9. Mandal AK, Mehdi I, Munshi SK, Lo TC. Value of routine duodenal biopsy in diagnosing celiac disease in patients with iron deficiency anemia. *Postgrad Med J.* 2004;80:475-7.
10. Kalayci AG, Kanber Y, Birinci A, Yildiz L, Albayrak D. The prevalence of coeliac disease as detected by

- screening in children with iron deficiency anaemia. Acta Paediatr. 2005;94:678-81.
11. Haapalahti M, Kulmala P, Karttunen TJ, et al. Nutritional status in adolescents and young adults with screen-detected celiac disease. J Pediatr Gastroenterol Nutr. 2005;40:566-70.