International Journal of Science and Research (IJSR)
ISSN: 2319-7064
ResearchGate Impact Factor (2018): 0.28 | SJIF (2019): 7.583

Treatment of Celiac Disease with Single Medicine: A Case Report

Dr. Rajeev Saxena M.D. [1], Dr. Devi Lal Gangwal[2]

[1](Hom.- Repertory) Professor & HoD, PG-PhD Guide, Department of Repertory, Swasthya Kalyan Homoeopathic Medical College & Research Centre, Sitapura, Jaipur, India
Email address: drrajeevsaxena1800[at]gmail.com

[2]Department of Repertory, Swasthya Kalyan Homoeopathic Medical College & Research Centre, Sitapura, Jaipur, India
Email address: gangwalhomoeopathy[at]gmail.com

Abstract: ***A case of celiac disease is well taken with all the information regarding patient like personal information, family history, past history of any complaints and worked out according to the principles of Law of Simillia, took many symptoms regarding celiac disease as well other than celiac disease like physical complaints and mental complaints which may or may not be related to celiac disease so selected all the symptoms present in body. Then comes the follow up which is full of fluctuation of symptoms as well as reports regarding celiac disease i.e TTG-IgA and which is overall a different and essential task and the result fundamentally depends upon the unadulterated prescription. Further to processed the need for Repertory felt, so according to the case selected Kent Repertory because of much and more prominent physical symptoms. Now with the help of Repertorization, symptoms have been converted to rubrics. Then most similar medicine has been selected. Pulsatilla Nigricans is selected, which covers maximum symptoms and marks. The well selected medicine Pulsatilla Nigricans 200 had great role in the eradication of disease. The aim of this article is to show the efficacy of Homoeopathic medicine in celiac disease although each and every cases of celiac is different from every case of celiac disease.***

Keywords: Celiac Disease, Repertory, Pulsatilla Nigricans, Homoeopathy

1. Introduction

Celiac disease (CD) is a life-long autoimmune disorder characterized by an immunologically mediated enteropathy induced by the dietary gluten, which improves with the exclusion of the toxic protein from diet. The disorder affects genetically susceptible individuals carrying the human leukocyte antigen (HLA) class II DQ2 and/or DQ8.[1]

Celiac disease (CD) is associated with genetic characteristics and gluten exposure. In recent studies in which genetic markers were used, small intestinal histologic changes, serum immune reactivity and clinical features show that there are subjects with some but not all of itscharacteristics, posing a difficult question of how to de-fine the disease[2].

CD is associated with HLA molecules DQ2 (90%–95%) and DQ8 (5%–10%), and in the continued presence of gluten the disease is self-perpetuating. CD is one of the most common lifelong disorders worldwide and is characterized by a variety of clinical presentations. These include the typical malabsorption syndrome (classic symptoms) and a spectrum of symptoms potentially affecting any organ or body system (nonclassic symptoms). Because CD often is atypical or even clinically silent, many cases go undiagnosed and are exposed to the risk of long-term complications. There is growing interest in the social aspects of CD because the burden of illness related to this condition is doubtless higher than previously thought[3].

Abdominal symptoms are common in primary care, with an annual incidence of 35 to 40 per 1000 individuals. Chronic abdominal symptoms can adversely affect daily functioning and quality of life. For the primary care physician, the diagnostic challenge is to discriminate between patients with functional gastrointestinal problems only and those with organic disease, such as celiac disease[4]

1.1 What is Celiac Disease

Celiac disease, also spelled, Coeliac disease, Gluten allergy, Gluten intolerance, wheat allergy, Wheat intolerance, is an autoimmune disorder of the gut (small intestine) that occurs in hereditarily predisposed individuals of all ages from middle infancy onward.

Celiac disease is a unique autoimmune disorder in which the environmental precipitant, gluten, is known. Originally considered a rare malabsorption syndrome of childhood, celiac disease is now recognized as a common condition that may be diagnosed at any age and that affects many organ systems. This review discusses the pathogenesis, diagnosis, and management of the disease.[5]

In INDIA as per the data of J K Loan hospital Jaipur – According to the medical reports, there are about 2,000 children in Jaipur and about 3,50,000 in Rajasthan state have been suffered of wheat allergy. Worldwide celiac disease influences between 1 in 100 and 1 in 200 people; rates do, however, vary between different areas of the world from as few as 1 in 300 to as many as 1 in 40[6].

Wheat allergy is an allergy which usually indicates as a food allergy, but can also be a touch allergy consequential from occupational contact to wheat. Similar to all allergies, wheat allergy occupies immunoglobulin E and mast cell response. Typically, the allergy is limited to the seed storage proteins of wheat, some reactions are restricted to wheat proteins, while others can react across many ranges of seeds and other plant tissues. Wheat allergy may be a misnomer while there

are numerous allergenic constituents in wheat, such as protease inhibitors, glutens and proclaims and altered responses are often attributed to various proteins. 27 prospective wheat allergens have been effectively identified. Gluten sensitivity is not generally classified as a wheat allergy[7].

2. Pathogenesis

Celiac disease results from the interaction between gluten and immune, genetic, and environmental factors .

2.1 The Role of Gluten

Celiac disease is induced by the ingestion of gluten, which is derived from wheat, barley, and rye. The gluten protein is enriched in glutamine and proline and is poorly digested in the human upper gastrointestinal tract. The term "gluten" refers to the entire protein component of wheat; gliadin is the alcohol-soluble fraction of gluten that contains the bulk of the toxic components. Undigested molecules of gliadin, such as a peptide from an α-gliadin fraction made up of 33 amino acids, are resistant to degradation by gastric, pancreatic, and intestinal brush-border membrane proteases in the human intestine and thus remain in the intestinal lumen after gluten ingestion.4 These peptides pass through the epithelial barrier of the intestine, possibly during intestinal infections or when there is an increase in intestinal permeability, and interact with antigen-presenting cells in the lamina propria.

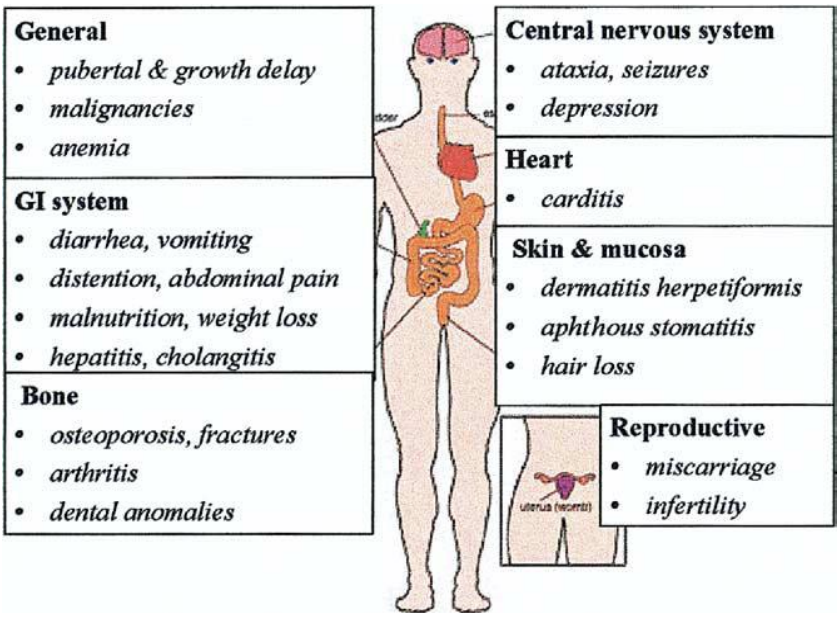
2.2 Mucosal Immune Responses

In patients with celiac disease, immune responses to gliadin fractions promote an inflammatory reaction, primarily in the upper small intestine, characterized by infiltration of the lamina propria and the epithelium with chronic inflammatory cells and villous atrophy (Fig. 1). This response is mediated by both the innate and the adaptive immune systems. The adaptive response is mediated by gliadin-reactive CD4+ T cells in the lamina propria that recognize gliadin peptides, which are bound to HLA class II molecules DQ2 or DQ8 on antigen- presenting cells; the T cells subsequently produce proinflammatory cytokines,5 particularly interferon-γ.6 Tissue transglutaminase is an enzyme in the intestine that deamidates gliadin peptides, increasing their immunogenicity.7 The ensuing inflammatory cascade releases metalloproteinases and other tissue-damaging mediators that induce crypt hyperplasia and villous injury. 8 Gliadin peptides also activate an innate immune response in the intestinal epithelium that is characterized by increased expression of interleukin- 15 by enterocytes, resulting in the activation of intraepithelial lymphocytes expressing the activating receptor NK-G2D, a natural-killer-cell marker.9 These activated cells become cytotoxic and kill enterocytes with surface expression of major-histocompatibility-complex class I chainrelated A (MIC-A), a cell-surface antigen induced by stress, such as an infection.10,11 The mechanism of the interaction between the processes in the epithelium and lamina propria has not been elucidated.[8]

3. Diagnostic Criteria

Growth problems, Failure to gain weight
Chronic diarrhea, which can be bloody or may be Chronic constipation
Vomiting, Fatigue
Abdominal bloating and pain,Irritability
Anemia, associated with iron deficiency, is most often due to increased blood loss, or impaired iron absorption. Iron-deficiency anemia is often recorded in newly diagnosed celiac disease[9].

System wise symptoms [10]



4. Prognosis

Complications of celiac disease include refractory disease, collagenous sprue, and intestinal lymphomas. Intestinal lymphomas affect 6 to 8% of patients with celiac disease, usually manifesting after 20 to 40 yr of disease. The incidence of other GI cancers (eg, carcinoma of the esophagus or oropharynx, small-bowel adenocarcinoma) also increases. Adherence to a gluten-free diet can significantly reduce the risk of cancer. If people who have

been doing well on a gluten-free diet for a long time once again develop symptoms of celiac disease, physicians usually do upper endoscopy with small bowel biopsy to check for signs of intestinal lymphoma[11].

The association between type 1 diabetes and celiac disease is well documented in young people, although reported rates vary. Prevalence rates from both cross-sectional and longitudinal studies range from 1.6% to 16.4% worldwide, with the majority of studies only including children and adolescents. In contrast, CD prevalence is 0.3% to 1.0% in the general population of all ages. A greater risk is conferred by female gender, younger age, and, in type 1 diabetes, younger age at diabetes diagnosis[12].

5. Diagnosing Celiac Disease

Until the 1950s, the diagnosis of CD was based on clinical observations focused on malabsorptive features. The peroral intestinal biopsies, introduced in 1956, marked a significant change in CD diagnosis. Since then, histological assessment of intestinal mucosa, with evidence of characteristic gluten-dependent mucosal damage, is considered the gold standard for CD diagnosis[13].

Capsule endoscopy (CE) is a useful tool for evaluating small-bowel disease, but appropriate indications and rates of detection, completion, and retention vary.[14]

Nowadays TTG-IgA test is most popular among because it is very cheap and very trustworthy. In this we can see how much allergy.

Case Study
A male Child (O.P.D. Regd. No: **94311**, X Kasana, 6 years old patient from Greatar Noida, Uttar Pradesh. He was diagnosed as a celiac patient at the age of 4 years.

The main symptoms present in X Kasana were —
- Diarrhea 2-3 times, watery stools
- Cramping type of pain before loose motions
- Excessive gas formation
- Abdominal pain due to gas formation which is relieved by passing flatus

5.1 Personal History

OPD No: 94311 Case No: 30 **Date: 24/02/219**	**Name: X Kasana** **Age/Sex: 6years/Male/child**
Father's Name: **Mr Vikram Kasana**	**Marital Status: Unmarried** **Religion: Hindu**
Address: Jaypee green spa court, greater noida (UP) 201308	
Occupation/Education: **Student** **Socio-economical Status: High**	**Diagnosis: Celiac Disease** **Miasmatic Diagnosis:** **Psora**
Result: Improvement	

Patient as a Person:

Appetite:	½- 1 chapati/day, twice a day.
Thirst:	2-2½ litres/day, satisfactory
Stool:	Diarrhea with cramping type of pain
Urine:	N/S
Desire:	Row foods
Aversion:	Fatty and rice foods
Addiction:	N/S
Thermal Reaction:	Hot
Bathing Habit:	Cold water in all seasons
Perspiration:	N/S
Sexual History:	N/S
H/o Vaccination:	Taken at that time
Menstruation:	N/A
Sleep/Position during Sleep:	Sound sleep, 7-8 hours

Mind:
- Anguish after defecation.
- Carried.
- Homesickness
- Envy.
- Impatience during sitting.

Family History	
Paternal	**Maternal**
Father – H/A (35yrs) **Brother** – H/A (8yrs)	**Mother –** H/A (33yrs) **Gr. Father** – Thyroid/ Alive (69yrs)

Physical Examination:

Weight: 19 kgms	**Tongue:** Moist, clean
Temperature: 98.6 ^{0}F	**Pallor:** Absent
Pulse: 88 b/m	**Icterus:** Absent
BP: N/S	**Cyanosis:** Absent
R/R: 20/m	**Edema:** Absent
Lymphadenopathy: Not palpable	**Clubbing:** Absent
Skin: Healthy	

Systemic Examination

Cardiovascular System:
Not abnormality found

Respiratory System:
Not abnormality found

Nervous System:
Not abnormality found

Gastrointestinal System:
Not abnormality found

GYNE/OBS HISTORY:
N/A

Laboratory Investigations
TTG-IgA
10-02-2019
TTG-IgA 41 U/ml

Analysis of Case (Kent's Method):

Mental Generals:
- Anguish after defecation.
- Carried.
- Homesickness.
- Envy.
- Impatience while sitting.

Physical Generals:
- Aversion to fatty and rice food.
- Desire for raw food.
- Hot patient.

Particulars:
- Diarrhea 2-3 times, watery stools
- Cramping type of pain before loose motions
- Excessive gas formation
- Abdominal pain due to gas formation which is relieved by passing flatus

Concomitants:
- Vomiting with loose motions

Common symptoms:
- General debility

Modalities:
- > Stool
- >Flatus

Evaluation of Symptoms:
1) Anguish after defecation.
2) Carried.
3) Homesickness.
4) Envy.
5) Impatience while sitting.
6) Aversion to fatty and rice food.
7) Desire for raw food.
8) Cramping pain relieved after passing flatus.
9) Cramping pain relieved after stool.

Repertorial Totality

S. No.	Symptom	Chapter/Rubric	Page No.
1.	Anguish before defecation.	MIND-ANGUISH-stool,before	3
2.	Carried.	MIND-CARRIED-desire to be	10
3.	Homesickness.	MIND-ENVY	39
4.	Envy.	MIND-HOMESICKNESS	51
5.	Impatience while sitting.	MIND-IMPATIENCE-sitting,while	54
6.	Aversion to fatty and rice food.	STOMACH-AVERSION-fat and rice food	480
7.	Desire for raw food.	STOMACH-DESIRE-row food	486
8.	Cramping pain relieved after passing flatus.	STOMACH-PAIN-flatus,passing,amel	513
9.	Cramping pain relieved after stool.	ABDOMEN-PAIN-cramping-stool,before	518

Repertorial Analysis:

S. No.	Remedies	Relative Value (No. of marks covered by remedy/No of rubrics covered by the remedy)
1.	Pulsatilla Nigricans	7/4
2.	Sepia	6/4
3.	Sulphur	6/3
4.	Staphysagria	5/3
5.	Arsenicum album	5/3

Prescription:
R_x
Pulsatilla Nigricans 200 1 dose EMES
Rubrum 30 T.D.S. x 7 Days 6 hourly

- **Justification of Selection of Remedy:** Pulsatilla Nigricans covers maximum rubrics and gets maximum marks with more suitable to the case, so phosphorus selected.
- **Justification of Selection of Potency:** 200 potency to start the treatment should be beneficial as per the susceptibility of the case.
- **Justification of Repetition of the Doses:** Dosage was given according to the severity of the case and complaints.
- **Diet and Regimen:** Take gluten free diets like rice, maze, soya bin, all fruits and vegetables.
- **Auxiliary Measures:** Avoid taking gluten contents like wheat, rye and barley. Take only gluten free diets.

International Journal of Science and Research (IJSR)
ISSN: 2319-7064
ResearchGate Impact Factor (2018): 0.28 | SJIF (2019): 7.583

सर्वे भवन्तु सुखिनः

Swasthya Kalyan Homoeopathic Medical College & Research Centre, Jaipur

REPERTORY SHEET

Name : Mst Adwik Kasana   Age & Sex: 6 yrs / Male / child   Date : 24/2/2019

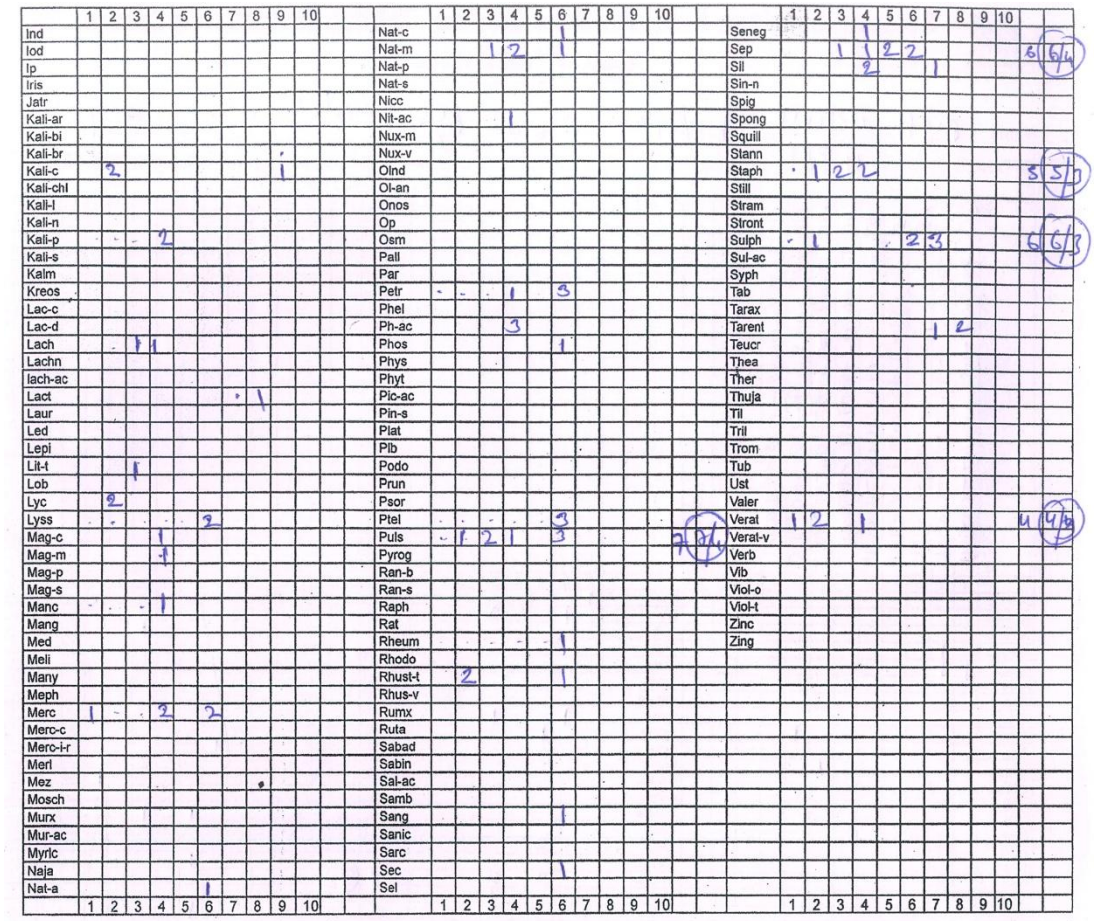
OPD No. : 94311   IPD No.: —   Ward/Bed No. : —

No.	Rubrics	Page
1	MIND- ANGUISH- stool, at	03
2	MIND- COVERED- desires to be	10
3	MIND- ENVY	39
4	MIND- HOMESICKNESS	51
5	MIND- IMPATIENCE- sitting, while	54
6	STOMACH- AVERSION- fat and rich	480
7	STOMACH- DESIRES- cold food	486
8	STOMACH- PAIN- eating, after	513
9	STOMACH- PAIN- cramping, after	518

	1	2	3	4	5	6	7	8	9	10	Total	Total Forward
Abies-c												
Abies-n												
Abrot												
Acet-ac		1										
Acon	1	1										
Act-s												
Aesc												
Aeth												
Agar								1				
Agn												
All							1					
All-c												
All-s												
Aloe												
Alumn												
Alum												
Ambr												
Am-c												
Am-m												
Anac												
Anan												
Ang						1						
Ant-c												
Ant-t												
Apis												
Apoc												
Aran												
Arg-m												
Arg-n												
Arn												
Ars-alb		2	1			2					5	5/3
Ars-iod												
Art-v												
Arum-t												
Asaf												
Asar												
Aspar												
Asper												
Aur				1								
Aur-iod												
Aur-m												
Bapt												
Bar-c												
Bar-m												
Bell				1		1						
Benz-ac		1										
Berb												
Bism												
Bor												
Bov												

	1	2	3	4	5	6	7	8	9	10	Total	Total Forward
Brom		1										
Bry			1			2						
Bufo												
Cact												
Cadm												
Calad												
Calc-ar												
Calc-c			1			1						
Calc-p				1								
Calc-f												
Calc-s												
Camph												
Cann-i												
Cann-s												
Canth												
Caps				3								
Carb-ac												
Carb-an				3		2					5	5/2
Carb-s						1						
Carb-v		1				2						
Card-m												
Carl				1								
Cast												
Caul												
Caust				2								
Cedr												
Cham		3										
Chel								1				
Chin						3						
Chin-a						1						
Chin-s												
Chlol												
Chlo												
Cic												
Cimx												
Cimic												
Cina		2										
Cinnb												
Cist												
Clem				2								
Cob			1									
Coca												
Cocc												
Coc-c												
Coff												
Colch						2						
Coloc									2			
Com												
Con												
Cop												

	1	2	3	4	5	6	7	8	9	10	Total	Total Forward
Cor-r												
Com												
Croc						1						
Cort-c												
Cort-h												
Crot-t												
Cupr												
Cupr-ars												
Cupr-s												
Cur			1									
Cycl						1						
Dig								1				
Dios												
Dros				1		1						
Dulc												
Echi												
Elaps				1								
Ery-a												
Eug												
Eup-per				1								
Eup-pur												
Euphr												
Eupi												
Fago												
Ferr												
Ferr-ar												
Ferr-i												
Ferr-p												
Fl-ac												
Gamb												
Gels												
Gins												
Glon												
Gran												
Graph												
Grat						1						
Guag												
Haem												
Ham												
Hell			1	1		1						
Helon												
Hep-s						2		2			4	4/2
Hipp				1								
Hura												
Hydr												
Hydr-ac												
Hyos				1								
Hyper												
Ign		1		2								
Indg												



Remark

1. pulsatilla nijsecam - 7/4
2. sepia - 6/4
3. sulphur - 6/3
4. staphysagria - 5/3
5. arsenicum album - 5/3

NOTE - pulsatilla covers maximum's and set max. marks, so pulsatilla is prescribed

Prescription -

℞ pulsatilla nijsecam 200 4 doses BMDS
Reborn 30 TDS 6 hourly x 15 days

Follow Up Sheet **OPD No:** 94311
Patient's Name: X Kasana **Age/Sex:** 6yrs.
Yrs/male/child

Date:	Symptom	Prescription
24/2/2019	Diarrhea 2-3 times, watery stools Cramping type of pain before loose motions. Excessive gas formation Abdominal pain due to gas formation which is relieved by passing flatus The TTG-IgA Was 41 U/ml.	Pulsatilla Nigricans 200 1 dose EMES Rubram 30 TDS 6 hourly X 15 days
12/3/2019	Relief in abdomen pain, loose motions and gas formation.	Rubram 30 TDS 6 hourly X 15 days
30/3/219	Pain in abdomen becomes reduced. He started gluten food.	Rubram 30 TDS 6 hourly X 15 days
15/4/2019	Further pain becomes reduced with gluten diets.	Rubram 30 TDS 6 hourly X 15 days
30/4/2019	Abdomen pain again appeared with relieved in loose motions.	Pulsatilla Nigricans 200 1 dose EMES Rubram 30 TDS 6 hourly X 15 days
17/5/2019	Relieved in abdomen and loose motions.	Rubram 30 TDS 6 hourly X 15 days
6/6/2019	Improvement in all symptoms but legs pain becomes appeared.	Pulsatilla Nigricans 200 1 dose EMES Rubram 30 TDS 6 hourly X 15 days
25/6/2019	Legs pain still persists with vomiting in morning.	Pulsatilla Nigricans 200 1 dose EMES Rubram 30 TDS 6 hourly X 15 days
13/7/2019	Improvement in all complains with normal appearance of abdomen.	Rubram 30 TDS 6 hourly X 15 days
7/8/2019	Improvement in all complains with normal appearance of abdomen.	Rubram 30 TDS 6 hourly X 15 days
22/8/2019	Improvement in all complains with normal appearance of abdomen. The TTG-IgA became normal with gluten diets. TTG-IgA Level 0.29 U/ml on 22/8/2019	Rubram 30 TDS 6 hourly X 15 days

Before Treatment

After Treatment

Comments: Patient was improving symptomatic as well as investigation gradually during treatment. So, case may be considered as "improved one"

References

[1] Bai J, Zeballos E, Fried M, et al. WGO-OMGE practice guideline: celiac disease. World Gastroenterology Organisation (WGO-OMGE); 2007.
[2] Araya M, Mondragon A, Perez-Bravo F, Roessler JL, Alarcon T, Rios G, Bergenfreid C. Celiac disease in a Chilean population carrying Amerindian traits. J Pediatr Gastroenterol Nutr 2000;31:
[3] 381–386Green PH, Cellier C 2007 Celiac disease. N Engl J Med 357:1731–1743
[4] van der Windt DA, Jellema P, Mulder CJ, et al. Diagnostic testing for celiac disease among patients with abdominal symptoms: a systematic review. JAMA. 2010 May 5;303(17):1738–46.
[5] Fasano A 2005 Clinical presentation of celiac disease in the pediatric population. Gastroenterology 128:S68 –S73
[6] Holmes GK. Screening for coeliac disease in type 1 diabetes.Arch Dis Child 2002;87:495– 498.
[7] http://www.drgangwal.com/specializations/celiac/
[8] Green PH, Fleischauer AT, Bhagat G, Goyal R, Jabri B, Neugut AI. Risk of malignancy in patients with celiac disease. Am J Med 2003;115:191–195.
[9] Halfdanarson TR, Litzow MR, Murray JA. Hematologic manifestations of celiac disease. *Blood* 2007; **109**: 412-421
[10] Gomez JC, Selvaggio GS, Viola M, Pizarro B, la Motta G, de Barrio S, Castelletto R, Echeverria R, Sugai E, Vazquez H, Maurino E, Bai JC. Prevalence of celiac disease in Argentina: screening of an adult population in the La Plata area. Am J Gastroenterol 2001;96:2700–2704
[11] Ilus T, Kaukinen K, Virta LJ, et al: Incidence of malignancies in diagnosed celiac patients: A population-based estimate. *Am J Gastroenterol* 109(9):1471–1477, 2014. doi: 10.1038/ajg.2014.194.
[12] Kaukinen K, Partanen J, Mäki M, Collin P: HLA-DQ typing in the diagnosis of celiac disease. *Am J Gastroenterol* 97(3):695–699, 2002. doi: 10.1111/j.1572-0241.2002.05471.x.
[13] Bai J, Zeballos E, Fried M, et al. Coeliac Disease. WGO-OMGE Practice Guideline World Gastroenterol News. 2005;10(2 suppl):S1–S8.
[14] Liao Z, Gao R, Xu C, et al. Indications and detection, completion, and retention rates of small-bowel capsule endoscopy: a systematic review. Gastrointest Endosc. 2010 Feb;71(2):280–6
[15] Kent.J.T : Repertory of Homoeopathic Materia medica ;6th Edition;New Central Book Agency(P)Ltd.,8/1 Chintamoni Das Lane,Calcutta;2004.