Treatment of Celiac Disease Permanently with Single Medicine

Devi Lal Gangwal

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. Sulphur is selected, which covers maximum symptoms and marks. The well selected medicine Sulphur 30 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, Sulphur, Homoeopathy

1. Introduction

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[1].

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[2].

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, gluten 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[3].

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. These activated cells become cytotoxic and kill enterocytes with surface expression of major-histocompatibility-complex class I chain-related A (MIC-A), a cell-surface antigen induced by stress, such as an infection. The mechanism of the interaction between the processes in the epithelium and lamina propria has not been elucidated.10

3. Diagnostic Criteria

Growth problems, Failure to gain weight Chronic diarrhoea, 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 disease5.

System wise symptoms:6

<table>
<thead>
<tr>
<th>General</th>
<th>Central nervous system</th>
<th>GI system</th>
<th>Heart</th>
<th>Skin &amp; mucosa</th>
<th>Bone</th>
<th>Reproductive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pubertal &amp; growth delay</td>
<td>ataxia, seizures</td>
<td>Diarrhea, vomiting</td>
<td>Carditis</td>
<td>Dermatitis herpetiformis</td>
<td>Osteoporosis</td>
<td>Miscarriage</td>
</tr>
<tr>
<td>Malignancies</td>
<td>Depression</td>
<td>Diarrhea, abdominal pain</td>
<td></td>
<td>Urticaria</td>
<td>Fractures</td>
<td>Infertility</td>
</tr>
<tr>
<td>Anaemia</td>
<td></td>
<td>Malnutrition, weight loss</td>
<td></td>
<td>Allergic stomatitis</td>
<td>Arthritis</td>
<td></td>
</tr>
</tbody>
</table>

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 (e.g., 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.7

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.8

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.9

Capsule endoscopy (CE) is a useful tool for evaluating small-bowel disease, but appropriate indications and rates of detection, completion, and retention vary.10

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 (Y, 6 years old patient from jaipur, Rajasthan. He was diagnosed as a celiac patient at the age of 3 years. The main symptoms present in Y were cramping type of abdominal pain since 15 days. Abdominal pain had became worsen by the farinaceous food and in morning. Bloating abdomen. Pain in lower extremities, headache after vomiting.

Mother history: she had taken lots of medicines for the treatment of allergic rhinitis for the whole 9 months which had created many difficulties in the pregnancy.

Personal History:
Marital status – unmarried; Occupation – Student Homoeopathic Generalities: A. Physical General.
Thermal reaction: hot patient.
Appetite: decreased; Desire: meat, charcoal; Aversion: row onions; Thirst: ½ to 1 lit./day; Salivation/ dryness of mouth: average; Taste: normal.
Bowel: stool; constipation, black hard stool. Urine: dark yellow urine with burning. Perspiration: average.
Sleep: normal.
B. Mental General:
Very slow to answer.
Confused while talking.
Aversion company even sight. Cosmopolitan, can’t stay at one place.

Clinical Examination

General examinations
Buit – lean thin; Nutrition – Emaciated; Anaemia – present; Jaundice – absent; Clubbing – not found; Oedema – not found; Neck vein – not engorged and not pulsatile; Neck gland – not palpable; Pulse ~102b /min; regular; Respiration ~26 /min; Obesity – absent; Weight- 8kg; Height- 78cms; B.M.I= 13.1; Depigmentation / hyperpigmentation – absent; Examination of palm, sole, vertex – normal; Tongue – moist.
Final Diagnosis: Celiac Disease.

Laboratory Investigations: On 10-08-2015 TTG-IgA was >160 U/ml

Confirmed Diagnosis: Celiac Disease as per expert’s opinion.

Miasmatic Diagnosis: Mixed- Miasmatic with predominance of Psora.

Analysis of symptoms:

Causation: This was may be due to lots of medication taken by her mother during pregnancy for treatment of allergic rhinitis.

Characteristic Mental Generals Symptom:
Very slow to answers.
Confused while talking.
Aversion company even sight.
Cosmopitian, can’t stay at one place.

Characteristics Physical Generals Symptom:
Aversion to row onions
Desire for meat
Desire for charcoal
Loss of appetite
Decreased thirst
Hot patient

Characteristics Particulars:
Bloated Abdomen
Cramping type of abdominal pain
Pain in lower extremities.
Constipation, black dark stool

Repertorization: Kent Repertory

Totality of the case:
1) Very slow to answers,
2) Aversion company even sight,
3) Confused while talking,
4) Aversion to row onions
5) Desire for charcoal
6) Desire for meat
7) Loss of appetite
8) Cramping pain in morning.
9) Bloated abdomen.

Analysis of Repertorial Result:
SULPHUR - 7/4
NUX MOS 6/4
THUJA 6/3
CUPRUM 5/4
CAUSTICUM 5/2

Repertorial Selection with Reasons: Sulphur is the repertorial selection because it covers maximum number of rubrics with highest score, it is found that Sulphur seems to cover the totality of symptoms as well as miasmatic background of the patient, so Sulphur is finally selected for the case

Final selection of Medicine (after consultation of Materia Medica and with reasons):

it is found that Sulphur seems to cover the totality of symptoms as well as miasmatic background of the patient, so Sulphur is finally selected for the case.

Prescription

On 15-08-2015: at that time she was on gluten free diet with above all symptoms and TTG-IgA was more than 161U/ml.

Rx Sulphur 30/tds 6 hourly; Doses; for 28 days

Follow Up

20-09-2015; improvement in appetite, bloated abdomen, cramps, legs pain, headache.

Rx PL 30/tds 6 hourly, Sulphur 30 single dose for 28 days.

29-10-2015: Relived in all complaints except bloated and prescribed. Now Started gluten diets for one time in alternate days Rx Sulphur 30 single dose for a month ; Doses; for 56 days;

11-01-2016: There was mild abdominal pain with relieved in all complaints and TTG-IgA gets down to 48 U/ml and prescribed PL 30/tds 6 hourly; 52 days.

23-03-2016: Patient feels better as a whole with gluten diets without previous complaints and prescribed Sulphur 30 single dose/month, early morning empty stomach, PL 30/tds 6 hourly 84 days.

16-07-2016: Patient feels better as a whole with complete gluten diets without previous complaints and prescribed PL 30/tds 6 hourly 28 days.

After 16th July he was on PL 30 for 6 months with gluten diets.

25-02-2017: Patient feels better as a whole, with complete gluten diets without previous complaints and prescribed PL 30/tds 6 hourly for hourly 56 days.

15-05-2017: Patient had slight abdominal pain, he taking gluten diets and prescribed Sulphur 30 single, PL 30/tds 6 hourly for 28 days.

20-06-2017: Patient feels better as a whole with complete gluten diets without previous complaints and prescribed PL 30/tds 6 hourly 84 days.

12-10-2017: Patient feels better as a whole with complete gluten diets without previous complaints and prescribed PL 30/tds 6 hourly 84 days.

22-01-2018: Patient feels better as a whole with complete gluten diets without previous complaints and prescribed PL 30/tds 6 hourly 84 days.

16-05-2018: Patient feels better as a whole with complete gluten diets without previous complaints also TTG-IgA gets down to 3.8 U/ml and prescribed PL 30/tds 6 hourly 84 days.

16-07-2016: Patient feels better as a whole with complete gluten diets without previous complaints and prescribed PL 30/tds 6 hourly 28 days.

Report Dated 13-11-2018; TTG-IgA was 0.29 AU/mL.

Conclusion :- The case of celiac was well taken and repertorized with the help of kent repertory and selected Lycopodium 30. Lycopodium 30 worked very well without complications.
Before Treatment

During Treatment

K.K DIAGNOSTIC CENTER
KHAWAS JI KA RASTA NEAR HAWA MAHAL JAIPUR
0141 - 2600834 , 3237997

Name  Mst. Ayan Qureshi
Lab. No  30
Ref.Dr. Dr. Gangwal Dr. D. L

Age  3 Yrs.  Sex  M
Date  02/11/2015  Rec. No.  1519-0159

Test Name  TTG (Tissue Trans Glutaminase)

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<th>Value</th>
<th>Unit</th>
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<tr>
<td>48.0</td>
<td>U/ml</td>
<td>Negative</td>
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Guide Value:

- <15 U/ml  Negative
- >15 U/ml  Positive

Type of Sample: SERUM

# EVERY TEST NEEDS TO BE CORELATED AND INTERPRETED CLINICALLY. IN CASE OF ANY QUERY TEST MAY BE REPEATED.

PATHOLOGIST: PROF. U.B. SHARMA M.D. PATHOLOGY, EX. PROF. S.M.S. MEDICAL COLLEGE, JAIPUR, & DR. KRITIKA SHARMA M.D PATHOLOGY

** End of Report **

TECHNOLOGIST

PATHOLOGIST

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After Treatment

K.K DIAGNOSTIC CENTER
KHAWAS JI KA RASTA NEAR HAWA MAHAL JAIPUR
0141 - 2600834, 3237997

<table>
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<tr>
<th>Name</th>
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<tr>
<td>Lab. No.</td>
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<tr>
<td>Ref. Dr.</td>
<td>Dr. Gangwal, D.L.</td>
</tr>
<tr>
<td>Age</td>
<td>3 Yrs.</td>
</tr>
<tr>
<td>Sex</td>
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<tr>
<td>Date</td>
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<td>Rec. No.</td>
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IMMUNOLOGY

**TTG (Tissue Transglutaminase)**

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<th>Test Name</th>
<th>Value</th>
<th>Unit</th>
<th>Reference Value</th>
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<tr>
<td>Serum for TTG</td>
<td>3.8</td>
<td>U/ml</td>
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<tr>
<td>Interpretation</td>
<td>Negative</td>
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<tr>
<td>Guide Value:</td>
<td>&lt;15</td>
<td>U/ml</td>
<td>Negative</td>
</tr>
<tr>
<td></td>
<td>&gt;15</td>
<td>U/ml</td>
<td>Positive</td>
</tr>
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</table>

Type of Sample: SERUM

**End of Report**

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

References


Author Profile

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