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A case of coeliac disease treated with *Lycopodium clavatum*

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Abstract
A case of coeliac 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 coeliac disease as well other than coeliac disease like physical complaints and mental complaints which may or may not be related to coeliac 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 coeliac disease i.e. TTG-IgA and which is overall a different and essential task and the result fundamentally depends upon the unadulterated prescription. Now with the help of Repertorization, symptoms have been converted to rubrics. Then most similar medicine has been selected. *Lycopodium* is selected, which covers maximum symptoms and marks. The well selected medicine *Lycopodium* 200 had great role in the eradication of disease. The aim of this article is to show the efficacy of Homoeopathic medicine in coeliac disease although each and every cases of coeliac is different from every case of celiac disease.

Keywords: Coeliac disease, TTG IgA, *lycopodium*, homoeopathy

Introduction
Coeliac disease also known as, coeliac sprue or gluten sensitive enteropathy, is characterized by Malabsorption resulting from inappropriate T cell mediated immune injury to mucosa of small intestine after ingestion of wheat gluten or related cereals like rye and barley. It has been also reported from Asian Countries like India and Western Pakistan and some Arabian countries. The first case in India reported in 1966 from New Delhi [1].

Coeliac disease is an important cause of malnutrition, classically children with coeliac disease present between ages 4 months - 24 months. But in our country there is significant delay in onset of symptoms and age of presentation. This delay is probably because of prolonged breast feeding practices in India, delayed weaning, late introduction of gluten.

Impaired growth, persistent diarrhoea, abdominal distention, vomiting, pallor (anaemia), abdominal pain, and vitamin deficiency are the typical symptoms of coeliac disease. It frequently also exhibits a number of uncommon or non-classical symptoms, including constipation, anaemia, short stature, association with other autoimmune conditions, and familial incidence [2, 3].

At risk groups
1. Asymptomatic iron deficiency anemia
2. IDDM (Type 1 DM)
3. Hypothyroidism
4. First degree relatives of coeliac disease
5. Williams's syndrome
6. Down's syndrome
7. Selective IgA deficiency
8. Polyglandular endocrinopathy
9. Addison's disease
10. Rheumatoid arthritis
11. Sjogren syndrome/ SLE/ hepatitis/ Auto immune myocarditis/primary sclecrosing cholangitis [4].

Early identification of coeliac disease patients in highly susceptible population may result in the treatment of sub clinical coeliac disease and improved control of associated disorders.

Among people with iron deficiency anemia that can't be explained by any other reason, there's high prevalence of coeliac disease [5]. For example, according to the American Gastroenterological Association (AGA), among people with iron deficiency and no gastrointestinal (GI) symptoms of coeliac disease, 2% to 5% will have positive coeliac disease blood tests and 3% to 9% will have positive biopsies. In patients with iron deficiency anemia who do have GI symptoms, the prevalence of coeliac disease is even higher 10% to 15%. Therefore the AGA recommends that any children with unexplained iron deficiency anemia be tested for coeliac disease.

When coeliac disease is not treated with a gluten-free diet, the small intestine's lining is harmed, resulting in iron and other nutrient Malabsorption.

A significant factor of malnourishment in children is coeliac disease or gluten sensitive enteropathy. It has been on the rise in our nation over the past two to three decades, especially in the northern and western states where wheat is a staple food.

Malnutrition is a serious health issue in developing nations like India. Poverty, a lack of food supply, a lack of health and nutritional knowledge, a number of sociocultural factors, diseases, population expansion, and last but not least, secondary malnutrition brought on by intestinal Malabsorption, are just a few of the reasons of malnutrition. Several pediatric diseases might include intestinal malabsorption, which is a major factor in the development of secondary malnutrition. It is not a diagnosis, rather a clinical condition.

"Malabsorption syndrome" is a condition that causes insufficient assimilation of ingested nutrients as a result of either maldisgestion or malabsorption. Children with generalized defect in assimilation of nutrients present with similar signs and symptoms like, abdominal distension, pallor, foul smelling and bulky stool, muscle wasting, poor weight gain or weight loss and growth retardation [6].

Clinical Manifestations

- **Gastrointestinal ("Classical"):** Diarrhea, vomiting, distended abdomen, failure to thrive, recurrent abdominal pain, flatulence, lactose intolerance.
- **Non gastrointestinal ("Atypical"):** Dermatitis herpetiformis, to decreased bone mineral density, oral mucosal lesions, dental enamel defects, occipital lobe epilepsy with cerebral calcification, cerebellar ataxia, chronic neuropathies, myoclonic ataxia, progressive leucoencephalopathy and dementia, infertility, mild liver abnormalities.
- **Asymptomatic:** In addition, coeliac disease may be associated with many other groups specially the relative of coeliac disease and various autoimmune disorders. These conditions are labeled as at risk groups for coeliac disease.

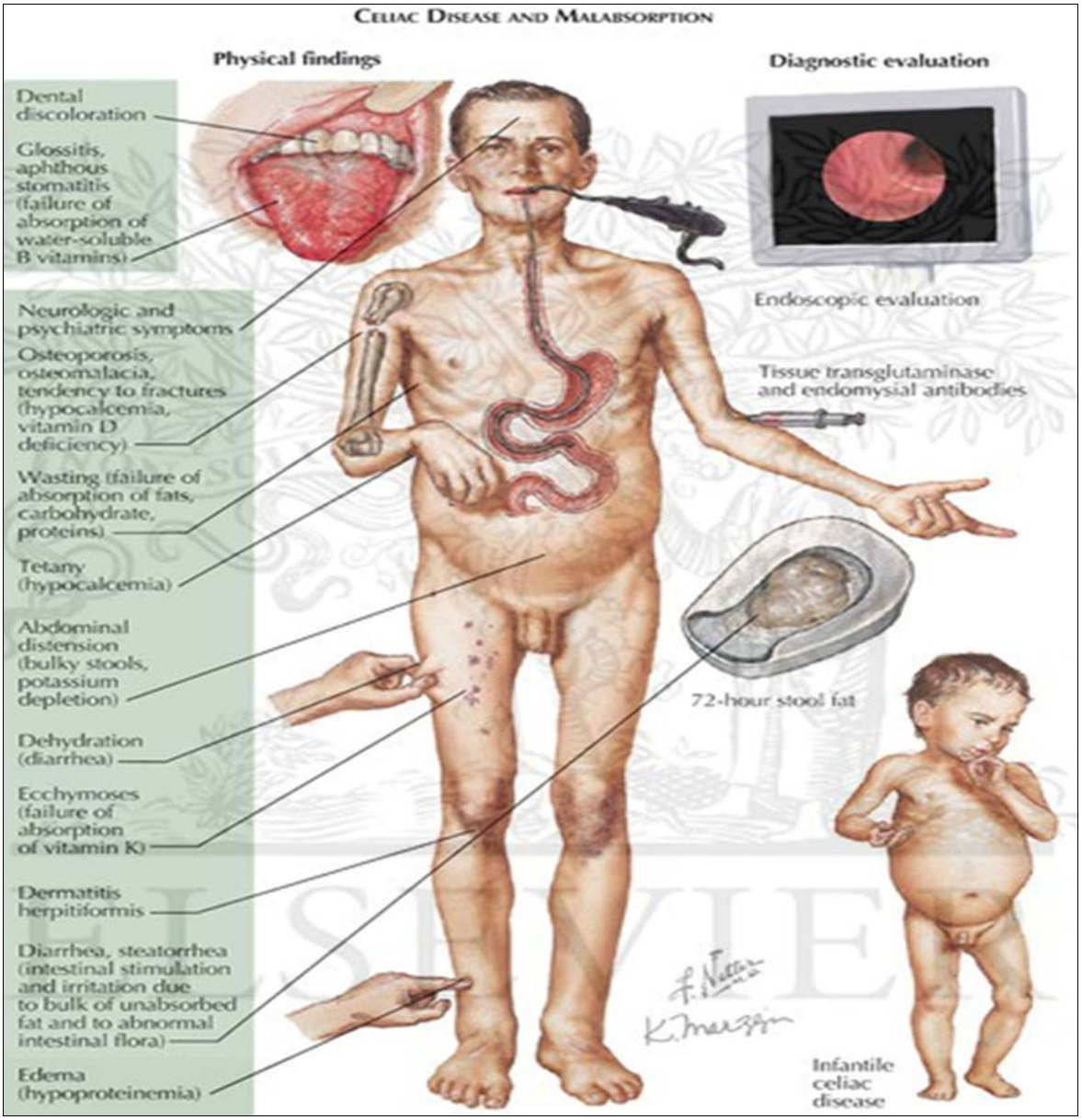
Gluten consumption causes coeliac disease in people who are genetically predisposed to the condition. Serum antibodies against endomysium, reticulin, gliadin, and tissue transglutaminase are what define it. Despite the fact that coeliac disease is frequently clinically asymptomatic or silent, the incidence of the condition is increased between 10 and 30 fold in several autoimmune illnesses when compared to the general population.

The detection of such coeliac disease instances is important since it may help in the management of Type 2 diabetes or endocrine functioning in general, as well as the avoidance of long-term CD consequences including cancer.

It is believed that CD may predispose an individual to other autoimmune disorders such as Type I diabetes. Hypothyroidism and other endocrine diseases and that gluten may be a possible trigger. The onset of Type I diabetes at an early age in patients with CD, compared to non CD, and the prevention or delay in onset of diabetes by gluten free diet in genetically predisposed individuals substantiates this antigen trigger hypothesis.

Treatment of subclinical coeliac disease and better control of related illnesses may arise from early detection of coeliac disease patients in a particularly sensitive group. When Ehlirchnd Morgenroth first described the condition known as "horror autotoxicus," or the fear of self-poisoning, the idea of autoimmunity was born. Several disorders were later identified as having an aberrant immune system response to self-antigens as their genesis [7].

Epidemiological studies have shown that genetic factors are involved in host susceptibility to autoimmune disease. For example, the castroen concordance rate of a particular autoimmune disease is much higher in monozygotic twins in comparison to fraternal twins. Moreover; this 2% to 5% will incidence is much higher in organs specific autoimmune disorders in comparison to non-organ specific disorders. Thus in hypothyroidism and type I diabetes, the concordance rate of the clinical conditions is as high as 50% in monozygotic twins, whereas in non-organ specific autoimmune disorders such as SLE and RA only 10% of identical twins are affected [9].



Diagnosis of coeliac disease

The European society of pediatric gastroenterology and nutrition ording to their document (ESPGAN) organized a panel discussion at its second annual movement on GED and also challenge. Conference in Inter-Laken in 1969, which lead to the publication in the following years of ESPGAN criteria for diagnosis of coeliac disease.

These are

1. Structurally abnormal jejuna mucosa when taking a diet containing gluten
2. Clear improvement of villious structure while on GFD
3. Deterioration of mucosa during challenge with gluten

Modified ESPGAN Criteria

In 1990, working group from the ESPGAN published modified criteria for diagnosis of childhood coeliac disease. These are:

1. Demonstration of characteristic histological changes in the small intestinal biopsy while on gluten.
2. Clear cut clinical remission within a few weeks of gluten free diet.
3. The findings of circulating antibodies to gliadin, reticulin, endomysial and tissue Tran's glutaminase at time of diagnosis and their subsequent disappearance on gluten free diet add weight to diagnosis.

According to these criteria, there is no need to document histological improvement on GFD and also of gluten challenge [9].

Case of coeliac disease

OPD No: 747XX
Name: XYZ
Age/sex: 27 yrs / Male
Father's name: XYZ
Marital status: Married
Religion: Hindu
Diet: Veg
Occupation: Family business
Address: Jagatpura, Jaipur
Date: 21/1/23

Chief complaints

Pain in abdomen on & off since 4-5 yrs
Nausea < empty stomach

Bloating & fullness of abdomen
Flatulence < night
Alternate Diarrhoea (watery offensive stool) and Constipation (hard stool has to strain, unsatisfactory offensive)
Sour Eructation < morning.
Weakness.

Past history
Dengue 6 yrs ago
Diagnosed case of coeliac disease patient was taking allopathic medicine from SMS Hospital with temporary relief.
Took ayurvedic treatment also.

Family history
Father DM & HTN since 4 yrs
Mother healthy & alive

Mental General
Anxiety and fear about health.
Consolation >
In dreams patient call everyone he is shouting for help but no one is listening, then suddenly wakes up from bed feels suffocated.

Physical generals
Appetite: Decreased does not feel like eating
Thirst: Increased dryness of mouth and throat
Desire: Sweet
Aversion: Sour
Stool: Alternate diarrhoea and constipation offensive unsatisfactory stool
Urine D5-6, N 0-1 No odour, No burning
Perspiration: Scanty, no staining, no odour
Sleep: Disturbed, unable to sleep unrefreshing
Addiction: Tea
Thermal: Toward hot

Physical Examination
Tongue: Clean moist Height- 165 cm
Pallor: Absent weight – 58kg
Icterus: Absent BMI - 21.3 Kg/m2
Oedema: Absent Bp – 120/90 mmHg
Cyanosis: Absent R/R – 17/min
Pulse: 83/ min

Systemic Examination
CNS: Conscious well oriented
CVS: S1, S2 heard, and no abnormal heart sound heard
Respiratory system- bilateral airway clear
Gastro- intestinal system-slightly tenderness in lower abdomen.

Analysis of symptoms

Mental General	Physical general	Particular
Anxiety about health Consolation > dreams feeling of suffocation	Appetite - decreased does not feel like eating Thirst – increased dryness of mouth and throat Desire – sweet Aversion – sour Stool – alternate diarrhoea and constipation offensive unsatisfactory stool Urine D5-6, N 0-1 No odour, No burning Perspiration – scanty, no staining, no odour Sleep – Disturbed, unable to sleep unrefreshing Addiction: Tea Thermal – Toward hot	Pain in abdomen on & off Nausea < empty stomach Bloating & fullness of abdomen Flatulence < night Alternate Diarrhoea (watery offensive stool) and Constipation (hard stool has to strain, unsatisfactory offensive) Sour Eructation < morning Weakness

Evaluation of symptoms
- Anxiety about health
- Consolation >
- Dreams feeling of suffocation
- Appetite- decreased does not feel like eating
- Thirst – increased dryness of mouth and throat
- Desire – sweet
- Aversion – sour
- Stool – alternate diarrhoea and constipation offensive unsatisfactory stool
- Perspiration – scanty, no staining, no odour
- Sleep – Disturbed, unable to sleep unrefreshing
- Thermal – toward hot
- Pain in abdomen on & off
- Nausea < empty stomach
- Bloating & fullness of abdomen
- Flatulence < night
- Alternate Diarrhoea (watery offensive stool) and Constipation (hard stool has to strain, unsatisfactory offensive)
- Sour Eructation < morning
- Weakness

Totality of symptoms
- Anxiety about health
- Dreams feeling of suffocation
- Appetite- decreased does not feel like eating
- Thirst – increased dryness of mouth and throat
- Desire – sweet
- Aversion – sour
- Nausea < empty stomach
- Sour Eructation < morning
- Pain in abdomen on & off
- Bloating & fullness of abdomen
- Flatulence < night
- Alternate Diarrhoea (watery offensive stool) and Constipation (hard stool has to strain, unsatisfactory offensive).

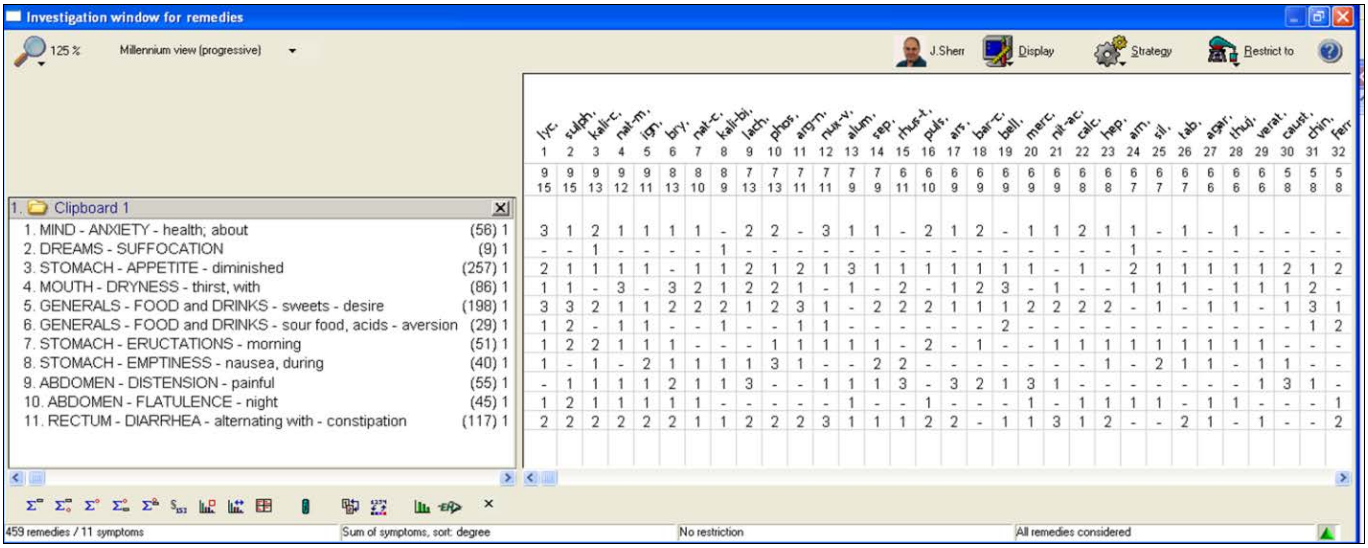
Miasm
- Anxiety about health - PSORA
- Dreams feeling of suffocation - SYCOTIC
- Appetite- decreased does not feel like eating - PSORA
- Thirst – increased dryness of mouth and throat - PSORA

- Desire – sweet -SYCOTIC
- Aversion – sour - SYCOTIC
- Pain in abdomen on & off -PSORA
- Nausea < empty stomach PSORA
- Bloating & fullness of abdomen - SYCOTIC
- Flatulence < night - SYCOTIC
- Alternate Diarrhoea (watery offensive stool) and Constipation (hard stool has to strain, unsatisfactory offensive) - SYPHILITIC
- Sour Eructation < morning - SYCOTIC

Rubrics

- Mind- Anxiety- health; about
- Dreams –Suffocation
- Stomach-Appetite- diminished
- Mouth- Dryness-thirst, with
- Generals-Food and Drink- sweets- desire
- General- Food and DRINK –sour food, acids- aversion
- Stomach- Eructation- morning
- Stomach- Emptiness- nausea, during
- Abdomen- Distension- painful
- Abdomen-Flatulence- night
- Rectum–Diarrhoea- alternating with- constipation

Repertory sheet



Repertorial Analysis

- *Lycopodium* – 15/9
- Sulphur – 15/9
- Kali-c - 13/9
- Natrum-m - 12/9
- Ign - 11/9

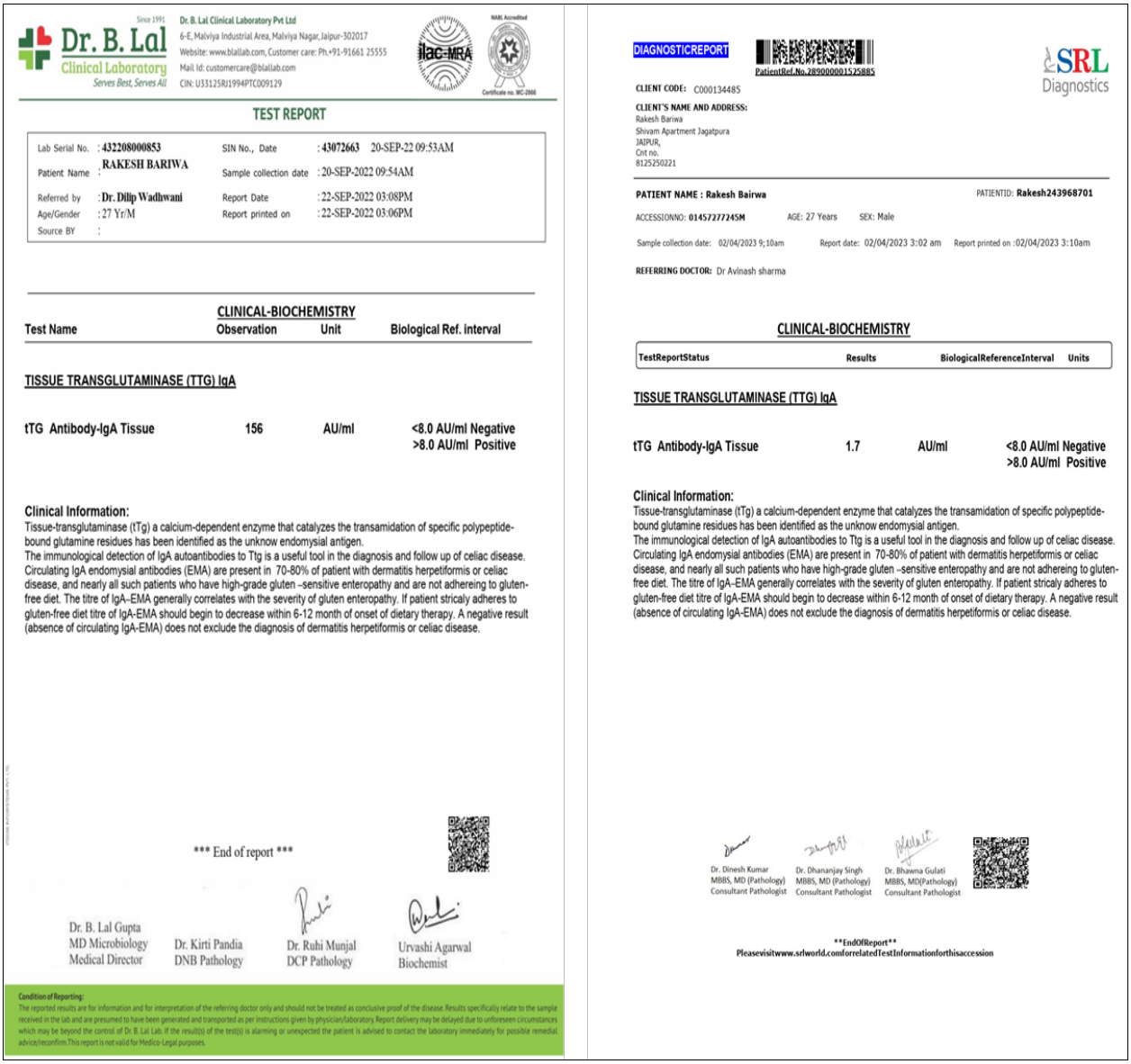
Depending upon the totality of symptoms *Lycopodium* was prescribed.

Prescription

Rx
Lycopodium 200 /1 Dose / Stat
Rubrum 30 TDS X 15 days

Selection of Remedy with Justification

Date	Symptoms	Prescription
3/2/23	Pain in abdomen has reduced Bloating & distension slightly reduced Sour Eructation + Flatulence + Nausea vomiting slightly better	Rx Rubrum 30 TDS X 15 days
17/2/23	No abdominal pain No nausea & vomiting Bloating and distension decreased Sour eructation +	Rx Rubrum 30 TDS X 15 days
4/3/23	Relief in abdominal pain	
	Bloating & fullness + Sour Eructation + Passing loose watery stool since 3-4 days, offensive Advice – Gluten free diet only	Rx *Lycopodium* 200 / 1 Dose / Stat Rubrum 30 TDS X 15 days
16/3/23	No Diarrhoea Normal regular bowel movement once a day Bloating fullness reduced Sour eructation decreased	Rx Rubrum 3o TDS X 15 days
1/4/23	No sour Eructation Passing soft stool once a day No abdominal pain or bloating	Rx Rubrum 30 TDS X 15 days



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CIN: U33125RJ1994PTC009129

TEST REPORT

Lab Serial No. : 432208000853 SIN No., Date : 43072663 20-SEP-22 09:53AM
Patient Name : RAKESH BARIWA Sample collection date : 20-SEP-2022 09:54AM
Referred by : Dr. Dilip Wadhwani Report Date : 22-SEP-2022 03:08PM
Age/Gender : 27 Yr/M Report printed on : 22-SEP-2022 03:06PM
Source BY :

CLINICAL-BIOCHEMISTRY

Test Name	Observation	Unit	Biological Ref. Interval
TISSUE TRANSGLUTAMINASE (TTG) IgA			
tTG Antibody-IgA Tissue	156	AU/ml	<8.0 AU/ml Negative >8.0 AU/ml Positive

Clinical Information:
Tissue-transglutaminase (tTg) a calcium-dependent enzyme that catalyzes the transamidation of specific polypeptide-bound glutamine residues has been identified as the unknow endomysial antigen.
The immunological detection of IgA autoantibodies to Ttg is a useful tool in the diagnosis and follow up of celiac disease. Circulating IgA endomysial antibodies (EMA) are present in 70-80% of patient with dermatitis herpetiformis or celiac disease, and nearly all such patients who have high-grade gluten –sensitive enteropathy and are not adhereing to gluten-free diet. The titre of IgA-EMA generally correlates with the severity of gluten enteropathy. If patient stricaly adheres to gluten-free diet titre of IgA-EMA should begin to decrease within 6-12 month of onset of dietary therapy. A negative result (absence of circulating IgA-EMA) does not exclude the diagnosis of dermatitis herpetiformis or celiac disease.

*** End of report ***

Dr. B. Lal Gupta, MD Microbiology, Medical Director
Dr. Kirti Pandia, DNB Pathology
Dr. Ruhi Munjal, DCP Pathology
Urvashi Agarwal, Biochemist

Condition of Reporting:
The reported results are for information and for interpretation of the referring doctor only and should not be treated as conclusive proof of the disease. Results specifically relate to the sample received in the lab and are presumed to have been generated and transported as per instructions given by physician/laboratory. Report delivery may be delayed due to unforeseen circumstances which may be beyond the control of Dr. B. Lal Lab. If the result(s) of the test(s) is alarming or unexpected the patient is advised to contact the laboratory immediately for possible remedial advice/reconfirm. This report is not valid for Medico-Legal purposes.

DIAGNOSTICREPORT
PatientRef.No.789000001525885
SRL Diagnostics

CLIENT CODE: C000134485
CLIENT'S NAME AND ADDRESS:
Rakesh Bariwa
Shivam Apartment Jagatpura
JAIPUR,
Cnt no.
8125250221

PATIENT NAME : Rakesh Bairwa PATIENTID: Rakesh243968701
ACCESSIONNO: 01457277245M AGE: 27 Years SEX: Male
Sample collection date: 02/04/2023 9:10am Report date: 02/04/2023 3:02 am Report printed on :02/04/2023 3:10am
REFERRING DOCTOR: Dr Avinash sharma

CLINICAL-BIOCHEMISTRY

TestReportStatus	Results	BiologicalReferenceInterval	Units
TISSUE TRANSGLUTAMINASE (TTG) IgA			
tTG Antibody-IgA Tissue	1.7	AU/ml	<8.0 AU/ml Negative >8.0 AU/ml Positive

Clinical Information:
Tissue-transglutaminase (tTg) a calcium-dependent enzyme that catalyzes the transamidation of specific polypeptide-bound glutamine residues has been identified as the unknow endomysial antigen.
The immunological detection of IgA autoantibodies to Ttg is a useful tool in the diagnosis and follow up of celiac disease. Circulating IgA endomysial antibodies (EMA) are present in 70-80% of patient with dermatitis herpetiformis or celiac disease, and nearly all such patients who have high-grade gluten –sensitive enteropathy and are not adhereing to gluten-free diet. The titre of IgA-EMA generally correlates with the severity of gluten enteropathy. If patient stricaly adheres to gluten-free diet titre of IgA-EMA should begin to decrease within 6-12 month of onset of dietary therapy. A negative result (absence of circulating IgA-EMA) does not exclude the diagnosis of dermatitis herpetiformis or celiac disease.

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EndOfReport
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Conflict of Interest
Not available

Financial Support
Not available

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