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Treatment of celiac disease with single medicine

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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. Arsenicum Album is selected, which covers maximum symptoms and marks. The well selected medicine Arsenicum Album 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, arsenicum album, homoeopathy

Introduction

Celiac Disease (CD) is a genetic, autoimmune disorder in which gluten a protein from the grains wheat, rye and barley, damage the small intestine. Gluten is a main character that causes many derangements in the small intestine as well as other system in the body.

As the gluten enters through the wheat, rye and barley, it causes damage to inner linings of the small intestine, which make it hard for the body to absorb nutrients, especially fat, calcium, iron and folate. CD-specific antibodies comprise tissue transglutaminase (tTG) antibodies, endomysial antibodies (EMA) and antibodies against deamidated forms of gliadin peptides (DGP)^[1].

Celiac disease is a multi-system disorder, which is highly unpredictable in it's clinical terms, may occur at any age, and may be present with variety of manifestations. Owning to this, the diagnosis is often delayed. CD etiology is a multifactorial, originating from the interplay of genetic and environmental factors. CD is triggered by the ingestion of wheat gluten related prolamines from rye and barley. Celiac disease develops as a consequence of the encounter between an environmental trigger and a genetically predisposed host, with the possible participation of other environmental cofactors ^[2].

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 defined as an immune mediated systemic disorder elicited by gluten and related prolamines in genetically susceptible individuals ^[1]. Celiac disease is triggered by cereal gluten from wheat, barley and rye and it is strongly associated with the HLA molecules DQ2 and DQ8. It is common nutrient related chronic disorder in the world, affecting roughly 0.5-1% of the populations of Europe, North and South America, Oceania, the Middle East, South Asia and North Africa (Catassi 2005) ^[3]. The current therapy for the condition is a life-long gluten-free diet

Definitions [4,5].

Classic CD: presents with signs and symptoms of malabsorption. Diarrhoea, steatorrhoea,

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weight loss, iron deficiency anemia or growth failures are found.

Non-classic CD: presents without signs or symptoms of malabsorption (e.g., abdominal pain, constipation, transaminitis etc.).

Symptomatic CD: is characterized by clinically evident gastrointestinal or extra-intestinal symptoms.

Asymptomatic (silent) **CD:** is found in persons with positive diagnostic tests for Celiac disease but with no intestinal or systemic symptoms and no overt clinical response to gluten-free diet.

Atypical CD: Atypical form. Absence or few gastrointestinal symptoms, presence of extra intestinal signs and symptoms such as anemia due to iron deficiency, osteoporosis or osteopenia, infertility, low stature;

Potential CD: people with normal small intestinal mucosa who are at increased risk of developing CD as indicated by positive CD serology.

Refractory CD is characterized by persistence or recurrence of malabsorptive symptoms and signs together with villous atrophy on small intestinal biopsy despite maintenance of an adequate gluten-free diet for over 12 months.

Prevalence

Worldwide, the disease affects approximately 1% of the general population, though this prevalence varies between countries⁴. The prevalence of CD is increasing from a global prevalence of 0.03% in the 1970s to current reports of 0.5 to 1.26% in most parts of the world. The largest screening to date of a healthy population in the US reports a frequency of 1:105 in populations without risk factors ^[6]. The prevalence of CD is estimated to be 1: 100 in the UK ^[7].

An increase in number of patients with CD has been observed from many centers in India ^[8]. Furthermore, two community based prevalence studies have been reported, both from the Northern part of India. In the first report from Ludhiana (Punjab) Sood *et al.* reported prevalence of CD to be 1 in 310 ^[9]. Makharia *et al.* reported the prevalence of CD in the Northern part of India to be 1.04% (1 in 96) in another community-based study ^[10]. There are regional variations in the prevalence of CD due to genetic and dietary factors, that is, the wheat-rice shift from the north to the south in India ^[11]. Differences in CD prevalence between north and south India could be ascribed to differences in

dietary patterns (rice being the staple cereal in south India) or due to differences in genetic make-up. The HLA-DQ phenotype of the general population in north and south India is not adequately known. Studies suggest that the prevalence of HLADQ2 in a north Indian populations around 32% [11, 12]

Etiopathogenesis

CD is a multifactorial condition, originating from the interplay of genetic and environmental factors. Celiac disease develops as a consequence of the encounter between an environmental trigger and a genetically predisposed host, with the possible participation of other environmental cofactors [2].

Dietary exposure to gluten has a central role in triggering the mucosal damage (Shan *et al.* 2002) [13]. The term "gluten" refers to toxic or immunogenic peptides in the storage proteins i.e. prolamine of wheat (gliadin), barley (hordein), and rye (secalin). One region of α -gliadin stimulates membrane cells, enterocytes, of the intestine to allow larger molecules around the sealant between cells. Disruption of tight junctions allow peptides larger than three amino acids to enter circulation [14]. This innate response to gliadin results in immune-system signalling that attracts inflammatory cells and increases the release of inflammatory chemicals.

Genetic Predisposition: The necessary environmental trigger is gluten, while the genetic predisposition has been identified in the major histocompatibility complex region on chromosome 6p21, with over 90% of CD patients expressing HLA DO2 (DOA1*05/DOB1*02) or in the transposition in HLA-DR5/DR-7 heterozygous patients. The patients remaining celiac express (DQA1*0301/DQB1*0302). While these haplotypes confer the highest genetic risk for CD, the fact that only about 4% of DQ2/8 positive individuals exposed to gluten develop CD, and about 20-30% of people without celiac disease have inherited an HLA-DQ2 allele. 15 and about 5% of those people who do develop celiac disease do not have the DO2 gene has led to the recognition that other factors are also necessary [16].

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 [17].

System wise symptoms [18]

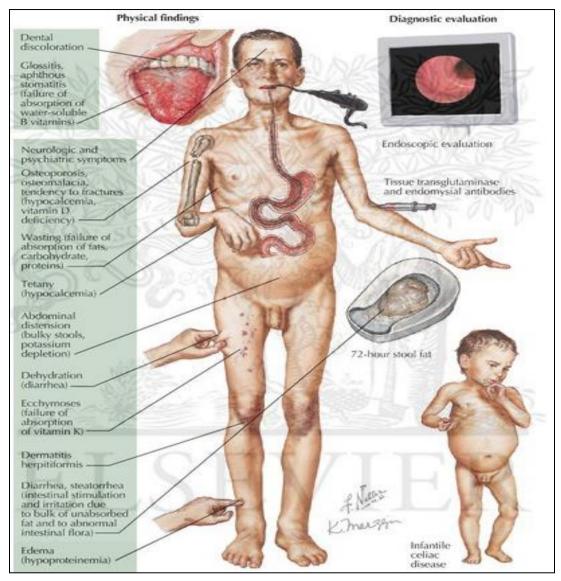


Fig 1: Celiac disease and malabsorption

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 [19].

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 [20].

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 glutendependent mucosal damage, is considered the gold standard for CD diagnosis [21].

Capsule endoscopy (CE) is a useful tool for evaluating small-bowel disease, but appropriate indications and rates of detection, completion, and retention vary [22].

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 boy (O.P.D. Regd. No: 95210, Y Kumar, 21 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 Kumar were-Abdominal pain in morning, bloated abdomen, constipation, knotty hard stool, regurgitation.

Personal History

Marital status: Unmarried; **Occupation:** Student

Homoeopathic Generalities

A. Physical General

General tendencies: Tendency to catch cold. **Thermal reaction:** Ambithermal patient.

Appetite: Average **Desire:** Ice cream Aversion: Warm food Thirst: 2 lit./day

Salivation/ dryness of mouth: Average

Taste: normal

Bowel: Hard stool 1 time/daily.

Urine: clear

Perspiration: average Sleep: Sound sleep

B. Mental General

Ardent **Fastidious**

Fright when complaints.

Impulsive

Desire to be strongly attract to dear one

Clinical Examination

General examintions Built: 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: 82 /min; regular;

Blood pressure: 108/78 mm Hg;

Respiration:16 /min;

Obesity: Absent Weight-38kgs, Height-101cms Pigmentation/hyperpigmentation: cheeks Examination of palm, sole, vertex: Normal

Tongue: White coated.

Final Diagnosis: Celiac Disease.

Laboratory Investigations: On 16-11-2017 TTG-IgA was

125 U/ml

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

Miasmatic Diagnosis: Mixed-Miasmatic with predominance of Psora.

Analysis of symptoms

Characteristic Mental Generals Symptom

Ardent Fastidious

Fright when complaints.

Impulsive

Desire to be strongly attract to dear one

Characteristics Physical Generals Symptom

Increased appetite, eat after eating also

Aversion to warm food

Characteristics Particulars

Bloated Abdomen relief by eructation

Abdomen pain in morning

Knotty hard stool

Characteristics concomitant

Headache relieved by eructations

Characteristics common symptoms

General debility

Characteristics modalities

Relieved by eructation

Repertorization: Kent Repertory [23]

Totality of the case

- 1. Ardent
- Fastidious
- 3. Fright when complaints.
- Impulsive
- Desire to be strongly attract to dear one 5.
- Increased appetite, eat after eating also 6.
- 7. Aversion to warm food
- 8. Bloated Abdomen relief by eructation
- 9. Knotty hard stool

Analysis of Repertorial Result: Arsenicum album – 11/6;

Phosphorus 10/4; Nux Vomica 9/5; Lycopodium clavatum -9/3: Silicea 8/4

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

Final selection of Medicine (after consultation of Materia Medica and with reasons): it is found that Arsenicum album seems to cover the totality of symptoms as well as miasmatic background of the patient, so Arsenicum album is finally selected for the case.

Prescription

On 27-02-2018 at that time she was on gluten free diet with above all symptoms

Rx Arsenicum album 200 single dose EMES; for 28 days

19-03-2018; improvement in Abdominal pain in morning, bloated abdomen, constipation, knotty hard regurgitation.

Rx PL 30/tds 6 hourly, Arsenicum album 200 1 dose for 28

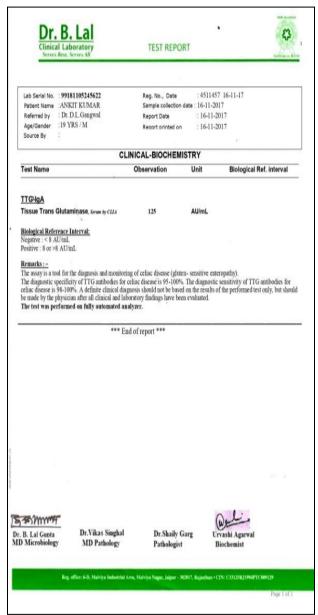
5-04-2018: Relived in all complaints better than before and prescribed, Now Started gluten diets for one time in alternate days Rx Arsenicum album 200 dose; for 42 days; 26-04-2018 There was mild abdominal pain with relieved in all complaints and prescribed Arsenicum album 200 1 dose; 42 days.

16-05-2018: Patient feels better as a whole with gluten diets without previous complaints and prescribed Arsenicum album 200 1 dose early morning empty stomach, PL 30/tds

6 hourly 28 days. 16-05-2018: Patient feels better as a whole with complete gluten diets without previous complaints and prescribed PL 30/tds 6 hourly 28 days.

03-06-2018: Patient feels better as a whole, with complete gluten diets without previous complaints and prescribed PL 30/tds 6 hourly for hourly 28 days.

28-06-2018: Patient feels much better, she taking gluten diets without previous complaints and prescribed PL 30/tds



Before Treatment

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

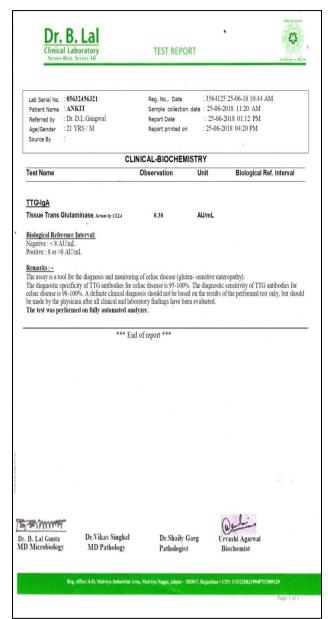
References

- 1. Husby S, Koletzko S, Korponay-Szabó IR, Mearin ML, Phillips A, Shamir R *et al.* ESPGHAN Working Group on Coeliac Disease Diagnosis; ESPGHAN Gastroenterology Committee; European society for pediatric gastroenterology, hepatology, and nutrition guidelines for the diagnosis of Celiac disease. J Pediatr Gastroenterol Nutr. 2012; 54:136-60. 1
- 2. Branski D, Troncone R. Gluten sensitive enteropathy

6 hourly for 28 days.

Report Dated 25-06-2018: TTG-IgA was. 3 AU/mL

Conclusion: The case of celiac was well taken and repertorized with the help of kent repertory and selected Arsenicum album 200. Arsenicum album 200 worked very well without complications.



After Treatment

(Celiac Disease). In: Kliegman RM, Stanton BF, St Geme JW, Schor NF, Behrman RE, editors. Nelson textbook of pediatrics. 20th ed. Philadelphia: Elsevier Saunders; 2016 p. 1835-38 2

- 3. Gujral N, Freeman HJ, Thomson ABR. Celiac disease: prevalence, diagnosis, pathogenesis and treatment. World J Gastroenterol. 2012; 18:6036-59. 20
- 4. Leonard MM, Vasagar B. US perspective on glutenrelated diseases. Clin Exp Gastroenterol. 2014; 7:25-37. 21
- 5. Murch S, Jenkins H, Auth M, Bremner R, Butt A, France S *et al.* Joint BSPGHAN and Celiac UK guidelines for the diagnosis and management of Celiac

- disease in children. Arch Dis Child. 2013; 98: 806-11. 4
- 6. Rajpoot P, Makharia GK. Problems and challenges to adaptation of gluten free diet by Indian patients with Celiac disease. Nutr. 2013; 5:4869-79. 22
- 7. Gupta R, Reddy DN, Makharia GK, Sood A, Ramakrishna BS, Yachha SK *et al.* Indian task force for Celiac disease: current status. World J Gastroenterol. 2009; 15:6028-33 23
- 8. Guandalini S. Celaic Disease. In: Guandalini S (ed). Textbook of Pediatric Gastroenterology and Nutrition. Florida, United States: CRC Press. 2004. p. 435-50. 8
- 9. Dinler G, Atalay E, Kalayci AG. Celiac disease in 87 children with typical and atypical symptoms in Black Sea region of Turkey. World J Pediatr. 2009; 5(4):282-6-7
- Ramakrishna BS. Celiac disease: can we avert the impending epidemic in India? Indian J Med Res. 2011; 133:5-8. 24
- Catassi C. The world map of celiac disease. ActaGastroenterol Latinoam. 2005; 35:37-55. 11
- 12. Shan L, Molberg O, Parrot I, Hausch F, Filiz F, Gray GM *et al.* Structural basis for gluten intolerance in celiac sprue. Science. 2002; 297:2275-2279. 25
- 13. Van Heel D, West J. Recent advances in coeliac disease". *Gut.* 2006; 55(7):1037–46. doi:10.1136/gut.2005.07511926
- 14. Van de Kamer, Dicke WK, Weijers HA. Celiac disease IV.An investigation into the injurious constituents of wheat in connection with their action on patients with celiac disease. (26) Paulley LW. Observations on the etiology of idiopathic steatorrhea. Br Med Jt 1954; 2(13):8-20. 17
- 15. Lionetti E, Catassi C. New clues in Celiac disease epidemiology, pathogenesis, clinical manifestations, and treatment.Int Rev Immunol. 211; 30:219-31. 3
- 16. Pallav, Kumar *et al.* "Clinical utility of celiac disease-associated HLA testing." Digestive diseases and sciences. 2014; 59(9):2199-2206. 27
- 17. Halfdanarson TR, Litzow MR, Murray JA. Hematologic manifestations of celiac disease. *Blood* 2007; 109:412-421
- 18. Gomez JC, Selvaggio GS, Viola M, Pizarro B, la Motta G, de Barrio S *et al.* Prevalence of celiac disease in Argentina: screening of an adult population in the La Plata area. Am J Gastroenterol. 2001; 96:2700-2704.
- 19. Ilus T, Kaukinen K, Virta LJ *et al.* Incidence of malignancies in diagnosed celiac patients: A population-based estimate. Am J Gastroenterol. 2014; 109(9):1471-1477. doi: 10.1038/ajg.2014.194.
- Kaukinen K, Partanen J, Mäki M, Collin P. HLA-DQ typing in the diagnosis of celiac disease. Am J Gastroenterol. 2002; 97(3):695-699. doi: 10.1111/j.1572-0241.2002.05471.x.
- 21. Bai J, Zeballos E, Fried M *et al.* Coeliac Disease. WGO-OMGE Practice Guideline World Gastroenterol News. 2005; 10(2 suppl):S1-S8.
- 22. 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; 71(2):280-6
- 23. Kent JT. Repertory of Homoeopathic Materia medica; 6th Edition; New Central Book Agency (P) Ltd.,8/1 Chintamoni Das Lane, Calcutta, 2004.