Coeliac Disease
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Dr. Balwant Singh, MBBS, Ex. Capt. AMC

Introduction

Coeliac Disease was detected more than a hundred years ago, but its full implications could not be brought to light for a long time. Its hidden, devastating effects which are extremely common has enabled physicians to save many precious lives by taking adequate precautions. Its presentations are so uncertain that the doctors tend to do the test to rule out celiac disease when no other cause for sickness is found.

It can be responsible for damage to any system of the body. It can cause infertility, diabetes type 1, skin irritation in the form of dermatitis herpetiformis. It can cause unexplained increase in weight due to hypothyroidism. Hyperparathyroidism causing easy fractures due to decreased bone density. The diseased person can remain tired all the time and no medicine can help. This is caused by chronic fatigue syndrome. It can promote auto immune disorders affecting any system of the body. It may cause strictures in the intestine leading to surgical emergency. It may cause liver failure and hyposplenism making the body prone to easy uncontrolled infections. Brain damage and stunted growth in children. It may be the root cause of irritable bowel syndrome where all the tests are negative and we as doctors fail to help the patients for the lifelong malady. Easy bleeding or uncontrolled bleeding due to the deficiency of Vit K. Iron deficiency anemia and megaloblastic anemia due to deficiency of folic acid and vitamin B 12. It can lead to impaired vision due to deficiency of Vit A.

With such advancement in all spheres of medicine, we have failed to find a cure for such a killer disease. The only cure is not to take gluten for entire life or take gluten free diet for the rest of life. It becomes really difficult with small children where the entire family is taking normal food and the child is not allowed to share the food with the rest of the family. It is a heart wrenching for the parents to maintain that schedule for the rest of their lives. More over many times the precautions are not observed and child lands up in serious trouble again.

This precaution is possible only if you could judge the cause of the sickness. And till that time the life is in doldrums.

In the face of this reality see the grace of Shri Guru Granth Sahib, you may not know the cause of your sickness even then you are fully cured. And the cure is permanent and in the shortest possible time of two days. We have seen this miracle in hundreds of people. We are providing

you with contact numbers of 50 cured people for verification. It is not surprising that we get some time 15 -20 patients in one week who find permanent relief.

We organize the healing sessions at Mata Raj Kaur Rog Niwaran Kendre, 385-L, Model Town, Sarab Rog Ka Aukhad Nam Mission Trust, Ludhiana as per the following schedule.

Timings	Details	Days
5.30 am to 7.00 am	Healing Sessions	Saturdays, Sundays and Mondays
10.30 am to 11.00 am	Registration of the patients	Tuesday, Wednesday, Thursday and Friday
11.00 To 12.15 pm	Healing Session	------do-------
4.00 pm to 5.00 pm	Healing Session	------do-------

We are not the preachers but certainly we solely depend upon the power of Gurbani and we sing prayers and the patients join us in the prayers. The patients can do prayers at their respective homes and can enjoy the permanent lasting relief.

We don't charge anything from anybody and our services are free and open to any one of any sect, religion or group. We get patients of cancers and other chronic diseases and they too get relief over a period of time and not in four days.

For any help, please contact any of the following volunteers at the kendre.

Dr. Bawant Singh 94176 96268,

S. Jaswant Singh Chhabra 9988930174

S. Tarlochan Singh 96462 99560

S.Harmel Singh Bhardwaj 94176 96283

We plan to make more accommodation for holding of healing sessions in Model Town Ludhiana in the existing kendre and we do need funds for this work. You are requested to donate for the noble cause. We have been granted permanent exemption from Govt of India Income Tax Dep't, under section 80 –G. This trust is registered as a charitable institution with Govt of India.

For online donations, the following information will help.

Once the money is deposited in the bank we will send you the receipt for Income Tax Exemption claims on your postal address.

NAME of BENEFICIARY:

SARAB ROG KA AUKHAD NAM MISSION TRUST (REGD), LUDHIANA

STATE BANK OF INDIA, DUGRI ROAD, MODEL TOWN, LUDHIANA

ACCOUNT NO. 10007494464

IFSC CODE: SBIN0009089

MICR CODE: 141002007

List of permanently cured patients with contact numbers and before publishing the list we have physically verified their relief from their parents or from them.

1. Varleen Kaur age 8 yrs. Phone: 9417336229

2. Bableen Kaur age 8 yrs. Phone: 98156 25813

3.Harsimranjeet Singh 9 yrs. Phone: 94172 69271

4.Parvinder Singh age9 yrs. Phone: 9814222174

5.Prabhjot age 5 yrs Phone: 95011 60994

6. Palwinder Kaur age 20 yrs. Phone: 98141 50572

7.Rakshit Pahwa age 4yrs. Phone:093686 83701

8.Mannan age 6 yrs. Phone: 98721 65446

9.Manjeet Singh age 6yrs. Phone: 98154 97496

10. Ravinder Singh age 7 yrs. Phone: 98887 31499

11. Mehak Bhatia age 4 yrs. Phone: 98159 45247

12.Ragini age 7 yrs. Phone: 98728 32537

13. Kamini age 22yrs. Phone: 97793 16314

14. Karishpreet Singh age 7 yrs. Phone: 093558 25241

15. Jaskaran Singh age 8 yrs. Phone:81465 25340

16. Manpreet Kaur age 9 yrs. Phone: same as above

17. Harleen Kaur age 6 yrs. Phone:95011 00212

18. Bhoomi Bhandari age 6yrs. Phone:98147 43940

19. Ridhma age 5 yrs. Phone:84277 99996

20. Shaveta age 26 yrs. Phone: 98151 14702

21. Navdeep Singh age 3 yrs. Phone:93164 34265

22. Lavanaya age 3 yrs. Phone:98555 14651

23. Palvi Indra age 23 yrs. Phone: 98728 55104

24. Sajal age 3yrs. Phone:98723 01537

25.Paramjeet Kaur, age 34 yrs, Phone: 94658 65585

26.Santokh pal Singh, age 24 yrs, Phone:99886 38805

27. Devanshi Gupta, age 5 yrs, Phone: 98150 13132

28. Balwinder Singh, age 23 yrs, Phone: 84379 09998

29. Jasanpreet Singh, age 2 yrs, Phone:98156 96362

30.Akshit Kohli, age 11 yrs, Phone:98884 62699

31. Resham Singh, age 35 yrs. Phone: 98551 22127

32. Shashi Kapoor, age 43 yrs, Phone:98761 44600

33. Manpreet Kaur, age 22 yrs, Phone: 99159 02021

34. Kashvi, age 5 yrs, Phone: 098379 33000

35. Pushpa Rani, age 42 yrs, Phone: 96460 57566

36. Rohit Sharma, age 19½ yrs, Phone: ---- do---

37. Shayana, age 22 yrs, Phone:98787 77764

38. Manpreet Singh, age 6 yrs, Phone:98154 97496

39. Pawandeep, age 8 yrs, Phone:81461 90725

40. Sharanjit Singh, age 35 yrs, Phone: 87288 17413

41. Navya, age 8 yrs, Phone:96460 35775

42. Bhoomi Gupta, age 5 yrs, Phone:98550 91213

43. Simardeep Singh, age 3 yrs, Phone:98153 77170

44. Meenkashi Khanna, age 50 yrs, Phone:92169 08200

45. Simran Kaur, age 9 yrs, Phone: 98881 555250

46. Bachi Joana, age 4 yrs, Phone:95010 97771

47. Bhavraj Singh Sidhu, age 8 yrs, Phone: 98880 26236

48. Samardeep Singh, age 7 yrs. Phone: 98141 88551

49. Gursharan Singh, age 19 yrs. Phone: 92166 61819

50. Neena Behal, age 53 yrs. Phone: 0161 2770569 { The first patient who got well}

Celiac Disease, celiac disease, celiac spree, non-tropical spree, endemic spree, gluten enteropathy or gluten-sensitive enteropathy, and gluten intolerance

It is wrongly called wheat allergy, it is the reaction to wheat proteins. It is an autoimmune disorder of the small intestine that occurs in genetically predisposed people of all ages from middle infancy onward. Symptoms include chronic diarrhea, failure to thrive (in children), and fatigue, but these may be absent, and symptoms in other organ systems have been described.

Increasingly, diagnoses are being made in asymptomatic persons as a result of increased screening; the condition is thought to affect between 1 in 1,750 and 1 in 105 people in the United States Celiac disease is caused by a reaction to gliadin, a prolamin (gluten protein) found in wheat, and similar proteins found in the crops of the tribe Triticeae (which includes other common grains such as barley and rye)

Upon exposure to gliadin, and specifically to three peptides found in prolamins, the enzyme tissue transglutaminase modifies the protein, and the immune system cross-reacts with the small-bowel tissue, causing an inflammatory reaction. That leads to a truncating of the villi lining the small intestine (called villous atrophy). This interferes with the absorption of

nutrients, because the intestinal villi are responsible for absorption. The only known effective treatment is a lifelong gluten-free diet.

Signs and symptoms

Severe coeliac disease leads to the characteristic symptoms of pale, loose and greasy stool (steatorrhoea), and weight loss or failure to gain weight (in young children). People with milder coeliac disease may have symptoms that are much more subtle and occur in other organs rather than the bowel itself. It is also possible to have coeliac disease without any symptoms whatsoever. Many adults with subtle disease only have fatigue or anaemia.

Gastrointestinal

The diarrhoea that is characteristic of coeliac disease is (chronic) pale, voluminous and malodorous. Abdominal pain and cramping, bloatedness with abdominal distension (thought to be due to fermentative production of bowel gas) and mouth ulcers may be present. As the bowel becomes more damaged, a degree of lactose intolerance may develop. Frequently, the symptoms are ascribed to irritable bowel syndrome (IBS), only later to be recognised as coeliac disease; a small proportion of patients with symptoms of IBS have underlying coeliac disease, and screening for coeliac disease is recommended for those with IBS symptoms.

Coeliac disease leads to an increased risk of both adenocarcinoma (small intestine cancer) and lymphoma of the small bowel (enteropathy-associated T-cell lymphoma or EATL). This risk returns to baseline with diet. Longstanding and untreated disease may lead to other complications, such as ulcerative jejunitis (ulcer formation of the small bowel) and stricturing (narrowing as a result of scarring with obstruction of the bowel).

Malabsorption-related

The changes in the bowel make it less able to absorb nutrients, minerals and the fat-soluble vitamins A, D, E, and K.

The inability to absorb carbohydrates and fats may cause weight loss (or failure to thrive/stunted growth in children) and fatigue or lack of energy.

Anaemia may develop in several ways: iron malabsorption may cause iron deficiency anaemia, and folic acid and vitamin B12 malabsorption may give rise to megaloblastic anaemia.

Calcium and vitamin D malabsorption (and compensatory secondary hyperparathyroidism) may cause osteopenia (decreased mineral content of the bone) or osteoporosis (bone weakening and risk of fragility fractures).

A small proportion have abnormal coagulation due to vitamin K deficiency and are slightly at risk for abnormal bleeding.

Coeliac disease is also associated with bacterial overgrowth of the small intestine, which can worsen malabsorption or cause malabsorption despite adherence to treatment.

Miscellaneous

Coeliac disease has been linked with a number of conditions. In many cases, it is unclear whether the gluten-induced bowel disease is a causative factor or whether these conditions share a common predisposition.

IgA deficiency is present in 2.3% of patients with coeliac disease, and in turn, this condition features a tenfold increased risk of coeliac disease. Other features of this condition are an increased risk of infections and autoimmune disease.

Dermatitis herpetiformis; this itchy cutaneous condition has been linked to a transglutaminase enzyme in the skin, features small-bowel changes identical to those in coeliac disease, and may respond to gluten withdrawal even if there are no gastrointestinal symptoms.

Growth failure and/or pubertal delay in later childhood can occur even without obvious bowel symptoms or severe malnutrition. Evaluation of growth failure often includes coeliac screening.

Recurrent miscarriage and unexplained infertility.

Hyposplenism (a small and underactive spleen) this occurs in about a third of cases and may predispose to infection given the role of the spleen in protecting against bacteria.

Abnormal liver function tests (randomly detected on blood tests).

Coeliac disease is associated with a number of other medical conditions, many of which are autoimmune disorders: diabetes mellitus type 1, autoimmune thyroiditis, primary biliary cirrhosis, and microscopic colitis.[17]

Other grains

Wheat subspecies (such as spelt, semolina and durum) and related species such as barley, rye, triticale and Kamut also induce symptoms of coeliac disease. A small minority of coeliac patients also react to oats. It is most probable that oats produce symptoms due to cross contamination with other grains in the fields or in the distribution channels. Therefore, oats are generally not recommended. However, many companies assure the 'purity' of oats, and are therefore still able to be consumed through these sources.

Other cereals such as maize (corn), millet, sorghum, teff, rice, and wild rice are safe for patients to consume, as well as non cereals such as amaranth, quinoa or buckwheat. Non-cereal carbohydrate-rich foods such as potatoes and bananas do not contain gluten and do not trigger symptoms.

Pathophysiology

Coeliac disease appears to be polyfactorial, both in that more than one genetic factor can cause the disease and that more than one factor is necessary for the disease to manifest in a patient.

Almost all people with coeliac disease have either the variant HLA-DQ2 allele or (less commonly) the HLA-DQ8 allele. However, about 20–30% of people without coeliac disease have also inherited either of these alleles. This suggests additional factors are needed for coeliac disease to develop – that is, the predisposing HLA risk allele is necessary but not sufficient to develop coeliac disease. Furthermore, around 5% of those people who do develop coeliac disease do not have typical HLA-DQ2 or HLA-DQ8 alleles .

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Some individuals inherit DQ2.5 from one parent and an additional portion of the haplotype (either DQB1*02 or DQA1*05) from the other parent, increasing risk. 2% lack DQ2 or DQ8.

Tissue transglutaminase

Anti-transglutaminase antibodies to the enzyme tissue transglutaminase (tTG) are found in an overwhelming majority of cases.

Stored biopsies from suspected coeliac patients have revealed that autoantibody deposits in the subclinical coeliacs are detected prior to clinical disease. These deposits are also found in patients who present with other autoimmune diseases, anaemia or malabsorption phenomena at a much-increased rate over the normal population.

Endomysial components of antibodies (EMA) to tTG are believed to be directed toward cell-surface transglutaminase, and these antibodies are still used in confirming a coeliac disease diagnosis. However, a 2006 study showed that EMA-negative coeliac patients tend to be older males with more severe abdominal symptoms and a lower frequency of "atypical" symptoms including autoimmune disease.

In a large percentage of coeliac patients, the anti-tTG antibodies also recognise a rotavirus protein called VP7. These antibodies stimulate monocyte proliferation, and rotavirus infection might explain some early steps in the cascade of immune cell proliferation.

Villous atrophy and malabsorption

The inflammatory process, mediated by T cells, leads to disruption of the structure and function of the small bowel's mucosal lining and causes malabsorption as it impairs the body's ability to absorb nutrients, minerals and fat-soluble vitamins A, D, E and K from food. Lactose intolerance may be present due to the decreased bowel surface and reduced production of lactase but typically resolves once the condition is treated.

Risk modifiers

A 2005 prospective and observational study found that timing of the exposure to gluten in childhood was an important risk modifier. People exposed to wheat, barley, or rye before the gut barrier has fully developed (within the first three months after birth) had five times the risk of developing coeliac disease relative to those exposed at four to six months after birth. Those exposed even later than six months after birth were found to have only a slightly increased risk relative to those exposed at four to six months after birth.

Breastfeeding may also reduce risk. A meta-analysis indicates that prolonging breastfeeding until the introduction of gluten-containing grains into the diet was associated with a 52% reduced risk of developing coeliac disease in infancy; whether this persists into adulthood is not clear.

Diagnosis

There are several tests that can be used to assist in diagnosis. The level of symptoms may determine the order of the tests, but all tests lose their usefulness if the patient is already taking a gluten-free diet. Intestinal damage begins to heal within weeks of gluten being removed from the diet, and antibody levels decline over months. For those who have already started on a gluten-free diet, it may be necessary to perform a re-challenge with some gluten-containing food in one meal a day over 2–6 weeks before repeating the investigations.

Combining findings into a prediction rule to guide use of endoscopic biopsy reported a sensitivity of 100% (it would identify all the cases) in a population of subjects with a high index of suspicion for coeliac disease, with a concomitant specificity of 61% (a false positive rate of 39%). The prediction rule recommends that patients with high-risk symptoms or positive serology should undergo endoscopic biopsy of the second part of the duodenum. The study defined high-risk symptoms as weight loss, anaemia (haemoglobin less than 120 g/l in females or less than 130 g/l in males), or diarrhoea (more than three loose stools per day).

Blood tests

Serological blood tests are the first-line investigation required to make a diagnosis of coeliac disease. Antiendomysial antibodies of the immunoglobulin A (IgA) type can detect coeliac disease with a sensitivity and specificity of 90% and 99%, respectively. Serology for anti-tTG antibodies was initially reported to have a higher sensitivity (99%) and specificity (>90%) for identifying coeliac disease. However, it is now thought to have similar characteristics to anti-endomysial antibody.

Because of the major implications of a diagnosis of coeliac disease, professional guidelines recommend that a positive blood test is still followed by an endoscopy/gastroscopy and biopsy. A negative serology test may still be followed by a recommendation for endoscopy and

duodenal biopsy if clinical suspicion remains high due to the 1 in 100 "false-negative" result. As such, tissue biopsy is still considered the gold standard in the diagnosis of coeliac disease.

Guidelines recommend that a total serum IgA level is checked in parallel, as coeliac patients with IgA deficiency may be unable to produce the antibodies on which these tests depend ("false negative"). In those patients, IgG antibodies against transglutaminase (IgG-tTG) may be diagnostic. .

An upper endoscopy with biopsy of the duodenum (beyond the duodenal bulb) or jejunum is performed. It is important for the physician to obtain multiple samples (four to eight) from the duodenum. Not all areas may be equally affected; if biopsies are taken from healthy bowel tissue, the result would be a false negative.

Other diagnostic tests

At the time of diagnosis, further investigations may be performed to identify complications, such as iron deficiency (by full blood count and iron studies), folic acid and vitamin B12 deficiency and hypocalcaemia (low calcium levels, often due to decreased vitamin D levels). Thyroid function tests may be requested during blood tests to identify hypothyroidism, which is more common in people with coeliac disease.

Osteopenia and osteoporosis, mildly and severely reduced bone mineral density, are often present in people with coeliac disease, and investigations to measure bone density may be performed at diagnosis, such as dual energy X-ray absorptiometry (DXA) scanning, to identify risk of fracture and need for bone protection medication.

Treatment: Gluten-free diet

At present, the only effective treatment is a lifelong gluten-free diet. No medication exists that will prevent damage or prevent the body from attacking the gut when gluten is present. Strict adherence to the diet allows the intestines to heal, leading to resolution of all symptoms in most cases and, depending on how soon the diet is begun, can also eliminate the heightened risk of osteoporosis and intestinal cancer and in some cases sterility. Dietitian input is generally requested to ensure the patient is aware which foods contain gluten, which foods are safe, and how to have a balanced diet despite the limitations. In many countries, gluten-free products are available on prescription and may be reimbursed by health insurance plans.

The diet can be cumbersome; failure to comply with the diet may cause relapse. The term gluten-free is generally used to indicate a supposed harmless level of gluten rather than a complete absence. The exact level at which gluten is harmless is uncertain and controversial.

Epidemiology:

People of African, Japanese and Chinese descent are rarely diagnosed , this reflects a much lower prevalence of the genetic risk factors. Population studies also indicate that a large proportion of coeliacs remain undiagnosed; this is due, in part, to many clinicians being unfamiliar with the condition.

Coeliac disease is more prevalent in women than in men.

A large multicenter study in the U.S. found a prevalence of 0.75% in not-at-risk groups, rising to 1.8% in symptomatic patients, 2.6% in second-degree relatives of a patient with coeliac disease and 4.5% in first-degree relatives. This profile is similar to the prevalence in Europe. Other populations at increased risk for coeliac disease, with prevalence rates ranging from 5% to 10%, include individuals with Down and Turner syndromes, type 1 diabetes, and autoimmune thyroid disease, including both hyperthyroidism (overactive thyroid) and hypothyroidism (underactive thyroid).