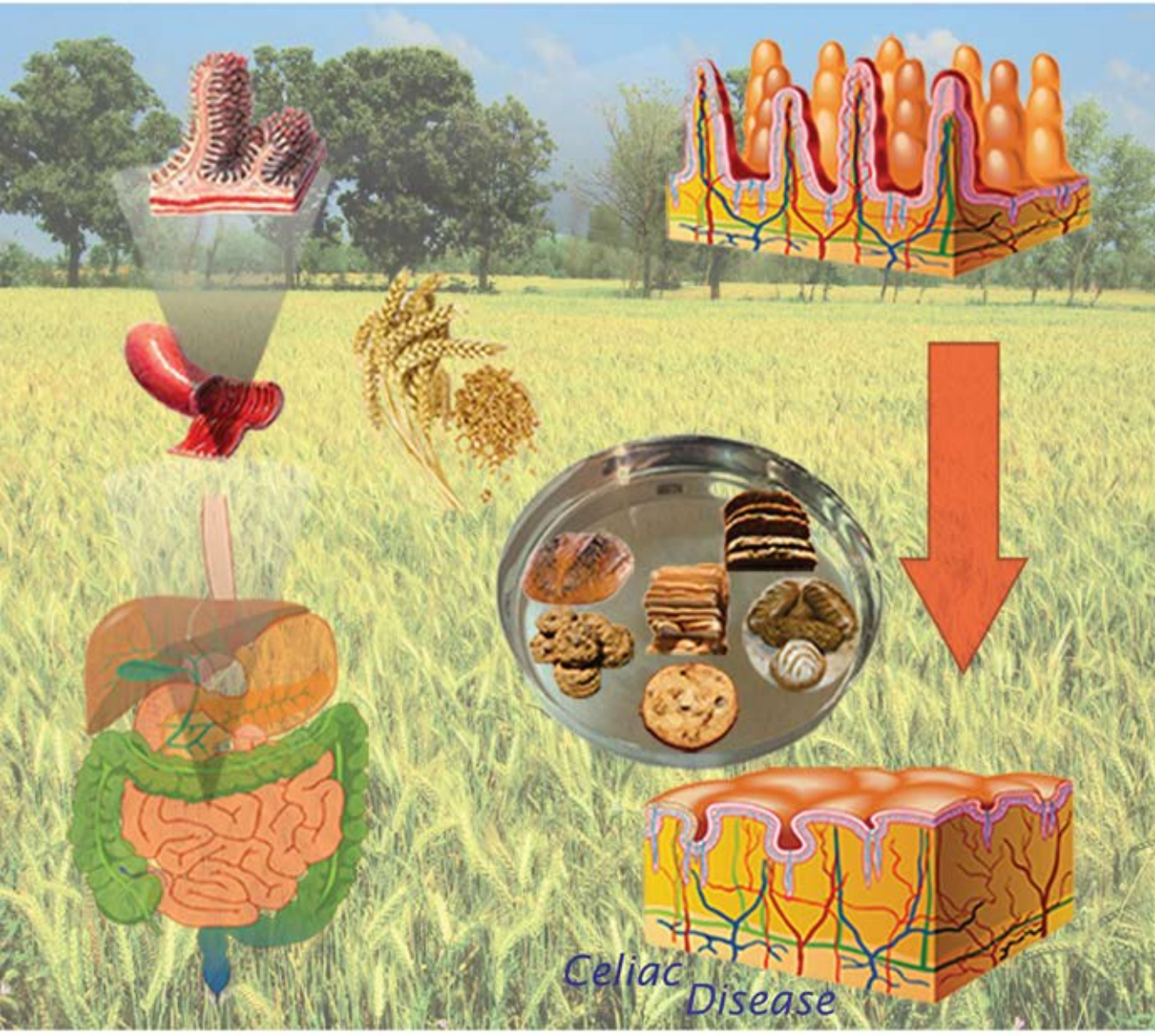
ICMR GUIDELINE ON DIAGNOSIS AND MANAGEMENT OF CELIAC DISEASE



Division of Noncommunicable Diseases
Indian Council of Medical Research
New Delhi

ICMR GUIDELINE ON DIAGNOSIS AND MANAGEMENT OF CELIAC DISEASE

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Indian Council of Medical Research
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ICMR Guideline on Diagnosis and Management of Celiac Disease in India

Coordinated by

Dr Prashant Mathur

Director, NCDIR, Bengaluru

(Formerly Scientist 'F' & Program Officer, Division of Noncommunicable Diseases, ICMR, New Delhi)

Members of Task Force Expert Group

Prof N.K. Arora, *Former Professor, Pediatric Gastroenterology, Hepatology & Nutrition, Department of Pediatrics, All India Institute of Medical Sciences & Executive Director, INCLEN Trust, New Delhi*

Prof Sita Naik, *Former Head of Department of Immunology, Sanjay Gandhi Postgraduate Institute, Lucknow*

Prof Meera Mathur, *Former Professor, Department of Pathology, All India Institute of Medical Sciences, New Delhi*

Prof M. Narendranathan, *Former Head, Department of Gastroenterology, Government Medical College, Thiruvananthapuram, Kerala*

Dr B. Sesikeran, *Former Director, National Institute of Nutrition, Hyderabad*

Members of Guideline Drafting Committee

Prof B.S. Ramakrishna, *Coordinator; Director, Institute of Gastroenterology, SRM Institutes for Medical Science*

Prof Govind K. Makharia, *Department of Gastroenterology, All India Institute of Medical Sciences, New Delhi*

Prof Siddhartha Datta Gupta, *Department of Pathology, All India Institute of Medical Sciences, New Delhi*

Prof Shinjini Bhatnagar, *Professor & Head, Pediatric Biology Centre, Translational Health Science and Technology Institute, Faridabad*

Prof A.S. Puri, *Head, Department of Gastroenterology, GB Pant Hospital, Delhi*

Prof Surender K. Yachha, *Head, Department of Pediatric Gastroenterology, Sanjay Gandhi Postgraduate Institute, Lucknow*

Prof Ajit Sood, *Head, Department of Gastroenterology, Dayanand Medical College & Hospitals, Ludhiana*

Prof Rakesh Kochhar, *Department of Gastroenterology, Postgraduate Institute of Medical Education & Research, Chandigarh*

Prof Meenu Singh, *Department of Pediatrics, Postgraduate Institute of Medical Education & Research, Chandigarh*

Dr Seema Puri, *Associate Professor, Department of Food and Nutrition, Institute of Home Economics, New Delhi*

Dr Kamal Chetri, *Senior Consultant Gastroenterologist, International Hospital, Guwahati.*

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एवं
महानिदेशक, आई सी एम आर
Dr. Soumya Swaminathan
MD, FASc, FNASc, FAMS
Secretary to the Government of India
Department of Health Research
Ministry of Health & Family Welfare
&
Director-General, ICMR

भारतीय आयुर्विज्ञान अनुसंधान परिषद
स्वास्थ्य अनुसंधान विभाग
स्वास्थ्य एवं परिवार कल्याण मंत्रालय
वी. रामलिंगस्वामी भवन, अंसारी नगर
नई दिल्ली-110 029 (भारत)
Indian Council of Medical Research
Department of Health Research
Ministry of Health & Family Welfare
V. Ramalingaswami Bhawan, Ansari Nagar
New Delhi-110 029 (INDIA)

PREFACE

I am happy to note that the ICMR Guideline on Diagnosis and Management of Celiac Disease has been prepared. This clinical condition affects many children and adults, who are unable to attain and maintain their full health potential. The present cornerstone of management remains early diagnosis and strict compliance with a life long gluten free diet. It fills a big void in having local and context specific guidelines for managing celiac disease.

This guideline has been prepared through wide stakeholder consultation, use of expert opinion and available scientific evidence. It shall help physicians, pathologists, nutritionists and other related disciplines to take care of these patients in an efficient and scientific manner. Several knowledge gaps identified during the process need to be addressed through appropriate research. It also lays the basis for working together across several sectors so as to spur initiation of several programmatic and policy relevant steps for addressing celiac disease.

I look forward to see the guideline helping in tackling celiac disease by various stakeholders in clinical and public health arena.

Soumya

(Soumya Swaminathan)

Tele. : (Off.) +91-11-26588204, 26589620; Fax (Off.) +91-11-26588662, E-mail: dg@icmr.org.in

THE INCLEN TRUST INTERNATIONAL
RESEARCH AND TRAINING FOR IMPROVING
EQUITY, EFFICIENCY AND QUALITY IN HEALTH CARE

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19th December, 2016

Foreword by Chairman

Celiac disease is caused by allergy to gliadin present in wheat and related grains with gastrointestinal as well as non-gastrointestinal manifestations and is estimated to have a prevalence of 1% to 1.5% in the general population in many Western countries. For a long time, celiac disease was generally believed to be rare in this part of the world and the clinicians considered celiac only sporadically in their differential diagnosis. The disease has been recognized in northern India, primarily in children, since the 1960s although true magnitude was not well known. A population-based study funded by the ICMR in 2012-2014 in Haryana, Tamil Nadu and Assam showed that seroprevalence of celiac disease as 1%, 0.2% and 0.8% respectively, indicating to the disease existence and patterns across the country.

Presently, the health professionals in India manage celiac patients based on the guidelines developed by European and North American associations; these have been considered insufficient in addressing unique features of the disease in India. Furthermore, the management practices were not standardized across the country. This felt need was taken up by ICMR and these guidelines were developed for the first time. The process of developing guidelines was kept objective, using high quality evidence, identifying the knowledge gaps, highlighting the limitations, keeping the language simple, consulting a wide range of stakeholders across different sectors and framing sections which can easily be identified for quick reference. The process was also used to identify key research questions to drive further research activities and also stimulate multi-disciplinary approach to address remaining challenges.

As Chairman of the ICMR Task Force on Celiac disease, it gives me immense satisfaction to see this document as a truly collaborative and participatory outcome to manage patients suffering from celiac disease in a systematic and scientific manner. This should improve management practices at all levels of health care. Due to rapid developments in this field the guideline would need to be revised periodically.

I wish it all the success in serving celiac disease.

NKArora 19.12.2016

Prof Narendra K. Arora
Chairman
ICMR Task Force on Celiac disease
Executive Director
The INCLEN Trust International

F-1/5, 2nd Floor, Okhla Industrial Area, Phase – I, New Delhi – 110 020 India
Phone no: (O) 91-11-47730000, Fax no: 91-11-47730001; Email: nkarora@inclentrust.org

डॉ आर.एस. धालीवाल
वैज्ञानिक 'जी' एवं प्रमुख
असंचारी रोग प्रभाग

Dr. R.S. Dhaliwal
Scientist 'G' & Head
Non-Communicable Disease

भारतीय आयुर्विज्ञान अनुसंधान परिषद
स्वास्थ्य अनुसंधान विभाग
स्वास्थ्य एवं परिवार कल्याण मंत्रालय
वी. रामलिंगस्वामी भवन, अंसारी नगर
नई दिल्ली – 110 029 (भारत)

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New Delhi - 110 029 (INDIA)

MESSAGE

The ICMR is the apex body for supporting medical research in the country and is one of the oldest such bodies in the world. The Council has developed several guidelines for various issues involved in biomedical research like stem cell research, assisted reproductive techniques, bioethics, etc. Division of Non-communicable Diseases has already brought out guidelines on management of different types of cancer (eight sites) and also type-II diabetes.

Celiac disease has been increasingly reported from several states of north India. Recognising its importance ICMR constituted a Task Force on Celiac disease which recommended that ICMR should develop guidelines for the diagnosis and management of celiac disease in India. This need was felt since in India, tropical enteropathy or environmental enteropathy is widely prevalent, and the incidence of parasitic and other infections of the small intestine is also significant. This meant that guidelines for management of celiac disease which are available internationally, have to be customised for the Indian conditions. Hence this activity of formulating Indian Guidelines was undertaken by the expert group. All statements in this document have been reached by consensus between all the experts and public comments received on the draft report placed on the ICMR website have also been incorporated.

I am sure that these guidelines will serve to rationalise the treatment of celiac disease in the country as well as serve as a reference for the postgraduate students interested in this area.

Head,
Division of Non-Communicable Diseases
Indian Council of Medical Research

Telefax (Off.) : 91-11-26588381, 26589811, E-mail: dhaliwalrs@icmr.org.in

NATIONAL CENTRE FOR DISEASE INFORMATICS AND RESEARCH
NATIONAL CANCER REGISTRY PROGRAMME
(INDIAN COUNCIL OF MEDICAL RESEARCH)
Department of Health Research, Ministry of Health and Family Welfare, Government of India
Nirmal Bhawan-ICMR Complex (II Floor), Poojanahalli Road, Off NH-7, Adjacent to Trumpet Flyover of BIAL
Kannamangala Post, Bengaluru - 562 110, INDIA

Dr Prashant Mathur *DCH, DNB, Ph.D., MNAMS*
Director
E-mail: director@ncdirindia.org

ACKNOWLEDGEMENT

The longstanding need to have an Indian guideline for diagnosis and management of Celiac Disease has been fulfilled, with the publication of this guideline. The process involved several expert meetings, public consultation and editing to bring out the present guideline.

I would like to express my gratitude to Dr. VM Katoch, Former Secretary, Department of Health Research and Director General, ICMR to agree to fund this activity at a point when funding position in ICMR was not very good. Following the completion of the process, Dr. Soumya Swaminathan, Secretary DHR and DG, ICMR kindly agreed to write a message highlighting the relevance of this guideline improving patient care and addressing public health needs. I was fortunate to have been encouraged by the Heads, Division of NCD, ICMR during this period (Dr. Bela Shah and Dr. DK Shukla) to complete the task with critical inputs. The guidance and support of Prof. NK Arora, Chairman, ICMR Celiac Disease Task Force along with Prof. Sita Naik, Prof. M Narendranathan, Prof. Meera Mathur and Dr. B. Sesikaran is gratefully acknowledged. The important role played by Prof. BS Ramakrishna in putting things together, carefully sifting through the available evidences, wading through controversial statements was immensely valuable and is much appreciated. The finalization process could not have been completed without critical insights provided by various experts and stakeholders during the consultation process. I am also thankful to Mr. PK Chawla, Mrs. Sunita Pahuja and Mr. Digambar Singh Rautela for the efficient administrative support provided to complete the task.

It will be fulfilment of a cherished dream to help patients with Celiac Disease get the best scientific management based on contextual circumstances.

(Prashant Mathur)

ICMR GUIDELINE ON DIAGNOSIS AND MANAGEMENT OF CELIAC DISEASE

Preamble

The Indian Council of Medical Research created a Task Force on Celiac disease in 2008, recognizing the need to focus on a disease that was beginning to be reported in sizeable sections of the population in several states of northern India. As recommended by the Task Force, data were collected on the prevalence of Celiac disease in three regions of India through a population-based study carried out in three regions of the country. The Task Force also recommended that ICMR develops guidelines for the diagnosis and management of celiac disease in India. While several international groups have generated guidelines for the diagnosis and management of celiac disease, these were largely rooted in the experience of western countries. There has been considerable skepticism over the applicability of these guidelines in a country like India where tropical enteropathy or environmental enteropathy is so widely prevalent, and where the incidence of parasitic and other infections of the small intestine is significant. Thus, the ICMR Task Force on celiac disease (CeD) recommended development of guidelines for diagnosis and management of CeD in the Indian context based on available evidence nationally and internationally.

Methodology

The sources of information included the following:

- International and national literature on the subject of celiac disease (CeD), identified through search of MEDLINE (1970 to present), EMBASE (1980 to present), AMED (1985 to present), HMIC (1983 to present) and IndMED (1985 to present).
- Data from a recently concluded multi-centre cross-sectional study carried out in Delhi, Guwahati and Vellore.
- A drafting committee comprising of prominent experts in the field was constituted. The panel of experts included the following:
    - B.S. Ramakrishna (adult gastroenterologist & coordinator of drafting committee)
    - Govind K Makharia (adult gastroenterologist)
    - Ajit Sood (adult gastroenterologist)
    - Rakesh Kochhar (adult gastroenterologist)
    - Amarender Puri (adult gastroenterologist)
    - Kamal Chetri (adult gastroenterologist)

- Shinjini Bhatnagar (paediatric gastroenterologist)
- Surender Yachha (paediatric gastroenterologist)
- Siddhartha Datta Gupta (histopathologist)
- Seema Puri (nutrition and dietetics)
- Meenu Singh (meta-analyses)
- Prashant Mathur, (Pediatric Gastroenterologist & ICMR Program Officer)

The Task force was coordinated at the Division of Noncommunicable Diseases, Indian Council of Medical Research, New Delhi.

Literature from various sources was searched in the guideline drafting coordinator's institution by a research fellow appointed for the purpose. Data was abstracted into data abstraction forms. The above experts were inducted into a Google emailing group. The literature was shared with the drafting committee. Based on the literature and the nation-wide ICMR study, the drafting committee identified priority areas and drafted a set of statements to address the major issues with regard to diagnosis and management of celiac disease in India. The draft statements were then circulated via Google groups to the entire committee and their feedback used to modify the statements. The drafting committee met in Delhi on 26.04.2015 to revise the guideline statements, and to provide write up for each statement, citing the appropriate source of evidence. The revised draft was circulated and was discussed in another face-to-face meeting organized at Indian Council of Medical Research, New Delhi on 14.07.2015. At this meeting, each of the statements was read out, the relevant literature on the topic presented and the members present agreed by consensus on the statement. Where consensus was not achieved, the statement was modified until a consensus was achieved. If this was not possible, the statement was dropped. The strength of the evidence available was graded. The draft version of the guideline was placed on the ICMR website between September to December 2015 for public comments. The comments received were examined by the core writing group and the relevant suggestions were incorporated at another meeting of the core writing group in Chennai on 12.01.2016. There are 9 sections and 49 statements in the guideline. The Task Force expert group reviewed and approved the final guidelines.

SECTION 1: DEFINITION

1. Celiac disease (CeD) is a chronic immune-mediated enteropathy, which is caused in genetically susceptible individuals by ingestion of gluten proteins present in wheat, barley and rye.

CeD was originally described as a disease causing chronic diarrhoea and malabsorption. Flattening of the villi, inflammatory cell infiltration in the mucosa and loss of surface area were the major reasons for the clinical manifestations. The understanding that this is an immune process in which the intestinal epithelium is damaged is now well accepted. Following from the original descriptions by Willem Karel Dicke relating wheat consumption to CeD,[1,2] a large number of studies have now established the central role played by proteins from wheat, barley and rye. The disease occurs only in individuals with a certain genetic predisposition but at the same time it does not necessarily occur in all such individuals. The nature of the immunological response to gluten is still being unravelled and T lymphocytes appear to play the primary role.[3,4] It is believed that CD4(+) T cells recognize gluten peptides bound to predisposing HLA-DQ molecules (DQ2 and DQ8), particularly when the gluten peptides are deamidated by the enzyme transglutaminase 2 (TG2). However, CeD is also characterized by the production of antibodies to gluten as well as to TG2 and a role for B cells in celiac disease pathogenesis is receiving increased recognition. Thus the precise definition of CeD incorporates all these characteristics, including the immune nature of the disease.

2. The disease occurs in individuals with specific genetic backgrounds including the presence of HLA DQ2 and/or DQ8.

CeD occurs only in genetically predisposed individuals who express certain specific human leukocyte antigens (HLA) related to the DQ region.[5] The DQ antigens are cell surface receptors that are present in many cells. In the antigen-presenting cells, specific DQ antigens are responsible for presenting the gluten peptides leading to immunogenicity and CeD. The DQ protein, located on the surface of antigen-presenting cells such as dendritic cells and macrophages, is composed of an alpha and a beta subunit, which together form a heterodimer. It presents antigen to CD4+ T cells through its T cell receptor(s). The HLA antigens are expressed based on the genetic composition of the HLA gene complex, which in humans is a large region on Chromosome 6, consisting of approximately 3 million nucleotides. The genes that determine the expression of the DQ antigens are to be found in 2 genetic loci, called DQA1 and DQB1, which lie next to each other, each gene having many alleles.[6] Typing of the HLA antigens is nowadays usually done by gene analysis. In a meta-analysis of 6 Western studies, the sensitivity of HLA-DQ2 and HLA-DQ8 typing for detection of CeD was 98% [95% confidence interval (CI) 97-99] and specificity 45% (95% CI 41-48).[6] If HLA-DQ2 or HLA-DQ8 was not detectable by typing, the negative likelihood ratio for CeD was 0.05 (0.03-0.09).[6] Due to its great sensitivity and low negative likelihood ratio, HLA-DQ2/DQ8 typing is more useful in ruling out CeD rather than for making a diagnosis of CeD.

3. The small intestine is the target organ most often affected in patients with CeD.

CeD classically presents with symptoms of diarrhea and nutritional deficiencies secondary to nutrient malabsorption. This indicates that the small intestine is the target organ most commonly affected in patients with CeD. Small intestinal changes, primarily an increase in intraepithelial lymphocytes associated with varying degrees of crypt elongation and villous blunting, form the primary histological hallmark of CeD. Studies have shown that the intestinal epithelium is the target of autoantibody deposition in CeD.[7-10]

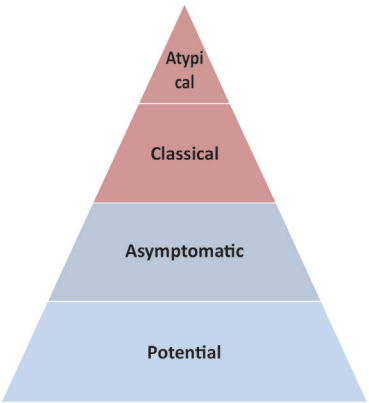
4. Organs other than the small intestine, including skin, reproductive system and bone may also be affected.

An association of CeD with dermatitis herpetiformis is well documented in the western literature, although there is a lack of reports from India. CeD is believed to directly target the skin.[11,12] Gluten ataxia is another immune manifestation of CeD indicating that it can also target the nervous system.[11] Joint and rheumatological disease may also occur independent of intestinal involvement.[11] Involvement of the oral mucosa (aphthous ulcers) and dental enamel hypoplasia are other features indicating that CeD often directly targets tissues outside the small intestine.[13,14] Infertility and metabolic bone disease are often features of CeD, but it is not clear whether this is due to involvement secondary to the nutritional consequences of small bowel involvement or whether this signifies direct involvement of reproductive system and bone.

5. The spectrum of disease varies from potential to clinical CeD.

CeD comprises a spectrum from asymptomatic to symptomatic individuals. Initial descriptions of CeD focused on the classical gastrointestinal presentation with diarrhea, malabsorption and nutrient deficiencies secondary to small bowel mucosal disease. With the advent of small bowel mucosal biopsy in the mid-20th century, the diagnosis focused on mucosal abnormalities characterized by intraepithelial lymphocyte infiltration, crypt elongation and villous shortening. This is now considered to be "classical" CeD. As understanding of the disease increased, it was recognized that CeD could present primarily with non-gastrointestinal manifestations such as iron deficiency anaemia, short stature, metabolic bone disease, or infertility. Investigation of the affected individuals also showed mucosal abnormalities characteristic of CeD and these presentations (where the gastrointestinal symptoms were minimal) were labelled "atypical" CeD. Among the above patients, where the presentation is mono-symptomatic (eg. stunting alone or anaemia alone) without even minor gastrointestinal symptoms, the disease may be called "non-classical" CeD. The diagnosis of disease then expanded and began to be recognized in apparently healthy individuals without any symptoms, in which situation they were labelled as having "asymptomatic" CeD. These individuals had mucosal abnormalities characteristic of CeD but did not have any symptoms to indicate the presence of CeD. The development of serological tests for CeD, in particular the IgA antibody to tissue transglutaminase (IgA anti-tTG), has unearthed yet another

category of individuals who are considered to have the potential to have CeD under the appropriate environmental circumstances, but do not have either mucosal abnormalities or symptomatology to suggest CeD. This last group of individuals is labelled as "potential" CeD.[15] Although the term "latent" CeD has been used interchangeably with "potential" CeD, the ICMR expert panel prefers the use of the term "potential" CeD to denote this group. The spectrum of CeD is such that only a small proportion is visible clinically, and the fact that most disease remains subclinical has led to the use of the term "celiac iceberg" to denote the various facets of the disease **(Figure 1).** Follow up of children with positive anti-tTG serology has shown conflicting results. In one study, 30.8% of 106 children with potential CeD developed villous atrophy over 3 years, while the others remained healthy.[16] Deposits of anti-TG2 IgA in the intestinal mucosa correlated with the development of villous atrophy and may serve to identify children at risk of developing clinical disease. However, in another study of children with potential CeD were followed for up to 9 years, 43% remained positive at the end of follow up, 20% became negative, and the remainder showed fluctuant results.[17] They did not develop mucosal damage after 9 years of follow up. In an adult hospital-based cohort, potential CeD accounted for 18.3% of all CeD, and some of these patients maintained a normal mucosa for many years.[18] In a case-finding study conducted in a school in Italy, 1.2% of the school children had CeD; the disease was asymptomatic in 64%, typical in 28%, atypical in 7%, and potential in 1%.[19] Comparable data do not exist for children in India. In the ICMR Task Force commissioned community based study recently completed at three sites in India, the pooled prevalence of CeD and potential CeD, respectively were 8.53/1000 and 3.70/1000 in northern, 4.66/1000 and 3.92/1000 in northeastern and 0.11/1000 and 1.22/1000 in the southern study sites. [20] This is clearly an area for further inquiry and evidence generation.



Symptoms	Mucosal lesion	Serology
Non-gastrointestinal	Present	Positive
Gastrointestinal	Present	Positive
None	Present	Positive
None	Absent	Positive

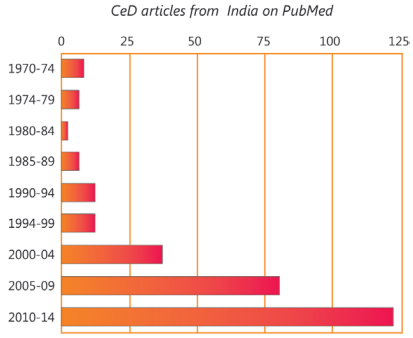
Figure 1. The "celiac iceberg". The upper two categories of the iceberg are symptomatic disease, and remain above water. The lower two categories are submerged and not usually recognized unless specifically looked for. The proportions of these categories likely vary from population to population and from time to time.

SECTION 2: EPIDEMIOLOGY

6. There has been an increase in reporting of CeD in India, which may be due to increased recognition (due to newer diagnostic techniques) or increased incidence or both.

Survey of the published literature indicates that there is a steady increase in the number of published papers dealing with CeD in India **(Figure 2) (Annex 1)**. This could be due to an increase in interest in the disease amongst physicians, an increase in recognition of the disease, or due to true increase in the incidence of the disease. In the absence of previous baseline data on the incidence and prevalence of the disease, the latter possibility is difficult to prove. However, the opinion of the expert group was that in addition to increased recognition, there was likely to have been an increase in true prevalence of the disease in recent years. Similar phenomena have been reported from other countries, particularly in the Asia-Pacific region.[21-23]

Figure 2. Number of publications on celiac disease from India, as listed in PubMed. Five year time intervals are shown



7. The prevalence of CeD in the northern states of India is greater than that in other parts of India.

Review of published studies from India indicates that publications on CeD are almost all from the northern states, particularly Punjab, Haryana, Uttar Pradesh, Rajasthan, Uttarakhand, Bihar and Delhi.[24] In the recently completed ICMR Task Force initiated study, the prevalence of CeD and potential CeD respectively were 8.53/1000 and 3.70/1000 in Haryana, 4.66/1000 and 3.92/1000 in Assam and 0.11/1000 and 1.22/1000 in Tamil Nadu respectively.[20]

8. CeD can affect individuals of any age starting from post-weaning to the elderly.

CeD does not occur before weaning since it requires exposure to the dietary gluten to get

manifested. This is a feature that can be used to clinically distinguish other causes of infantile diarrhea and malabsorption, that start prior to weaning. CeD can occur soon after weaning. It is present in all age groups thereafter and may even be recognized for the first time in the elderly.[25,26] Among adults, CeD could be noted in all age groups from 18 to 65 years of age in the recently completed ICMR Task Force initiated community study.

Risk factors

9. While wheat is the major cereal grain in the diet in the northern part of India, it is also consumed in the rest of India in variable quantities.

The expert group agreed that wheat is commonly accepted to be the major dietary cereal in northern India, in the states of Punjab, Haryana, Uttar Pradesh, Rajasthan, Bihar, and Uttarakhand, while states in eastern India and in southern India consumed rice as the major cereal in the diet. These findings were confirmed in the ICMR Task Force study, which compared 3 regions of India, and found that wheat intake in Haryana was nearly ten-fold higher than in either Assam or Tamil Nadu.[20]

10. CeD is a familial disease and a significant number of first-degree relatives are affected by CeD.

Studies from around the world provide convincing evidence that CeD is familial. The figures vary from 4% of 450 first degree relatives of CeD patients in Brazil to 9.5% of 45 first degree relatives in Turkey.[27-29] A recent meta-analysis concluded that the pooled prevalence of CeD among first degree relatives was 7.5%.[30] Additionally some first degree related children develop CeD if followed up for sufficiently long periods of time. The reported figures from India are concordant with these statistics, as 4.4% to 19% of first degree relatives of the Indian CeD patients were reported to have CeD.[31-34]

11. The risk of CeD varies in the first-degree relatives according to the their relationship with the index patient, the highest reported in siblings.

Studies of family risk for CeD suggest that carrying HLA-DQ2 (odds ratio 16.1; 95% CI 2.1-123) and being a sibling (odds ratio 2.5; 95% CI 1.1-5.8) are the highest family risk factors for CeD.[35] In a study from Scandinavia, the risk for CeD in multiple affected families was 26.3% for siblings and 12.9% for parents,[36] while a study from the US found that 21.3% of siblings were affected compared to 14.7% of offspring.[37] In Indian families, the prevalence of celiac disease among siblings (15.6%) was much higher as compared to that in parents (3.5%) or offspring (3%).[31]

12. The genetically predisposing HLA types (HLA-DQ2 and –DQ8) are common in the Indian population.

HLA-DQ serotypes 2 and 8 are widely recognized as being associated with CeD and provide the genetic background against which the disease develops.[5,6] These two serotypes have been reported in 13%-30% of different populations in India. Serotype DQ2 is encoded by the allelotype HLA-DQB1*02, while the most common allele encoding the DQ8 protein is HLA-DQB1*0302. The allele prevalence of HLA-DQB1*02 in northern India ranged from 16%-31% while that of HLA-DQB1*0302 ranged from 0%-5% and in southern India from 8%-14% and 6%-10% respectively.[38-40] Recent genome-wide association studies from other populations have identified a number of non-MHC genes that may influence the development of CeD.[41] A recent study evaluated the role of these non-MHC European CeD risk variants in 497 CeD cases and 736 controls of northern Indian origin.[42] Despite the inclusion of a number of non-MHC variants, this study found that the strongest association ($P=8.2\times10^{-49}$) was with a single nucleotide polymorphism in the intronic region of the HLA-DQB1 gene, suggesting that the non-MHC risk variants did not contribute much to CeD risk in India.

SECTION 3: CLINICAL PRESENTATIONS

13. The clinical spectrum of CeD varies from asymptomatic disease to severe manifestations.

As pointed out earlier in this document, patients with CeD may present with florid gastrointestinal symptoms (classical disease), overt non-gastrointestinal symptoms (atypical CeD) or be asymptomatic. In all these three states, it is expected that the individual will show celiac autoimmunity, ie. the presence of antibodies to transglutaminase 2 (TG2) detectable serologically as a positive IgA anti-tTG antibody test. All these forms of the disease should be accompanied by the presence of abnormal small intestinal mucosal histology characteristic of CeD. In the condition that has been variously called as potential CeD, latent CeD or celiac autoimmunity, the individual is positive for celiac autoantibody, but has a normal intestinal mucosa and has no symptoms consistent with the disease.[15]

14. Classical gastrointestinal manifestations are seen both in children and adults with CeD.

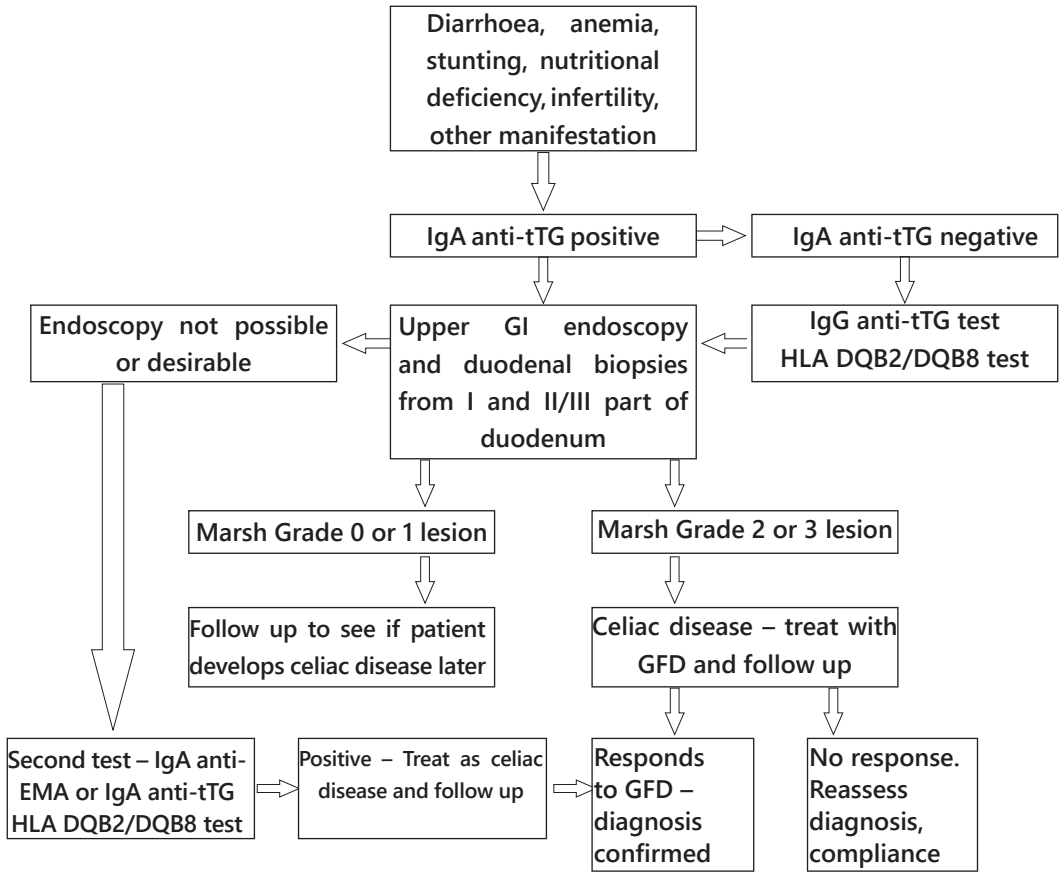
Several case series have reported that classical gastrointestinal manifestations, (including mainly chronic diarrhea defined by increased frequency of loose or bulky stools), weight loss and failure to grow, abdominal distension, vomiting, pedal edema, are the presenting symptom in 44-99% of patients with CeD.[43-52] These patients may have associated co-morbid conditions like anemia, recurrent oral ulcers, vitamin and other micronutrient deficiencies, recurrent abdominal pain and chronic fatigue and muscle weakness.

15. Many patients present with non-classical manifestations such as anaemia unresponsive to oral iron therapy, osteoporosis, increase in transaminases, infertility, short stature or failure to thrive.

Atypical CeD is now being recognized increasingly in India.[53-55] A significant proportion of patients with chronic anaemia unexplained by menstrual or other losses have CeD. Approximately 10% of patients attending a tertiary care hospital for investigation of short stature had CeD. Metabolic bone disease is another recognized co-morbid condition associated with CeD.[56-59] It may present with a spectrum of musculoskeletal signs and symptoms, such as bone pains, proximal muscle weakness, osteopenia, osteoporosis, and fractures. These conditions may be missed unless they are actively screened for. Approximately 2% to 3% of individuals with low bone mineral density may have asymptomatic CeD. There should be a high index of suspicion for CeD in patients with intractable epilepsy or where the epilepsy coexists with non-specific gastrointestinal symptoms.[59] Other neurological disorders like cerebellar ataxia, peripheral neuropathy, dementia, myoclonus and myelopathy have also been reported in CeD.[60] Late menarche, unexplained infertility, or unexplained abortions have also been reported to be associated with CeD. Recurrent oral ulcers and dental enamel defects are a presenting feature in some cases presenting without classical abdominal symptoms.

SECTION 4: DIAGNOSIS OF CED

ALGORITHM FOR DIAGNOSIS OF CELIAC DISEASE *



Anti-tTG = antibody to tissue transglutaminase

Anti-EMA = antibody to endomysial antibody

GFD = gluten free diet

** In situations where endoscopy is not feasible or desirable, kindly refer to para sections 19, 29 and 30.*

Serological tests

16. Serological tests are the first screening tests for CeD. IgA anti-tTG is currently the test of choice.

The definitive diagnosis of CeD is based on a combination of clinical history, serologic testing and examination of duodenal biopsies. However, a variety of serological tests were developed to assist in the diagnosis, and these have become the first line screening tests for CeD due to wide availability, ease of use and high reproducibility. Antigliadin antibodies, initially developed, are generally present in the blood of CeD patients, but may also be present in apparently healthy individuals, and in a variety of autoimmune or gastrointestinal diseases, and in non-celiac gluten sensitivity.[61] Therefore, they do not discriminate between CeD and controls and is not recommended for the diagnosis of CeD. Anti-endomysial IgA antibody (EMA) was first described in CeD diagnosis three decades ago.[62] The antibody is usually detected by immunofluorescence using monkey esophagus or human umbilical cord sections. However, the EMA test is expensive and it is very operator dependent due to the interpretation of the immunofluorescence pattern.[63]

In 1997, Dieterich et al.[64] found that tissue transglutaminase (tTG) was the autoantigen in CeD and part of the endomysial antigen. Based on this, a variety of ELISA tests to detect tTG-specific antibodies were subsequently developed, the target antigens used including guinea pig or human tTG (the latter may be recombinant or purified). Currently the preferred serologic test for the detection of CeD in subjects above 2 years of age is the IgA antibody to tissue transglutaminase (IgA anti-tTG), which shows a specificity and sensitivity of around 95%.[65] The test is available as enzyme linked immunosorbent assay or radioimmunoassay and has gained wider acceptance since the introduction of human recombinant substrates. The reported sensitivities and specificities of IgA-tTG ELISAs employing human tTG vary from 94% to 98%, and 95% to 99%, respectively.[66,67] A positive IgA-tTG ELISA using human tTG as antigen should lead to endoscopy and small bowel biopsies to confirm CeD. It is important to note that different tTG ELISA kits used by different diagnostic laboratories can have varying results and/or interpretation of the results while analyzing the same sample. The anti-endomysial antibody (EMA) test has a higher specificity (99%) and is sometimes used as a confirmatory test in cases of uncertain diagnosis where small intestinal biopsy is to be avoided.

Deamidated gliadin peptides contain CeD-relevant B cell epitopes and are very useful in the diagnosis of CeD.[68] Currently, there are a large number of commercial anti-deamidated gliadin peptide(DGP) ELISA tests available, including ones that detect IgA/IgG-DGPs individually or in combination with tTG which are quite sensitive and specific in both children and adults.[69] However a meta-analysis concluded that IgA-tTG ELISA has greater diagnostic accuracy than IgA-DGPs (sensitivities of 93% versus 87%

and specificities of 96% versus 94%).[67] Deamidated gliadin peptide (DGP) IgA and IgG, are used in combination with IgA anti-tTG in children who are less than 2 years old.[70] The sensitivity and specificity of serology in various settings depends on the pre-test probability of finding CeD. Thus, the specificity of serology is low when used as a screening test in healthy individuals in whom the pre-test probability is low, while in patients with a high probability of CeD, the specificity of serology is high.

Two studies[71,72] showed that patients with signs and symptoms suggestive of CeD and IgA-tTG levels >10x the normal range had a high likelihood for the presence of Marsh 3b or 3c villous atrophy. Based partly on this, ESPGHAN has suggested that, particularly in symptomatic children, CeD diagnosis can be performed without the need of intestinal biopsy if tTG serology is increased >10x normal range.[70] Under these circumstances the diagnosis of CeD should be confirmed by EMA staining and HLA typing in a second blood sample.[70]

IgA-tTG antibody may become negative in some children with subclinical CeD who were initially positive with titre <10x normal range.[73] Therefore in children without severe symptoms, serological follow-up is recommended before performing endoscopy with small bowel biopsies to confirm CeD. In children less than 2 years old IgG-DGP ELISAs may perform better than EMA tests and tTG ELISA with sensitivity and specificity approaching 100%.[74] However, some of these children may lose reactivity to DGP in course of time.[75] In general, because performance of all CeD-specific serology tests depends on the prevalence of the condition, the age of the subjects evaluated, and the amount of gluten ingested, these factors should be considered when interpreting CeD-specific serology results.

17. In a patient with strong clinical suspicion of CeD, negative serology should warrant exclusion of selective IgA deficiency.

Patients with clinical features of CeD may have a negative anti-tTG despite a strong clinical suspicion. Under these circumstances, the physician/paediatrician should consider using an alternative test for CeD such as an IgG anti-tTG/IgG anti-DGP test in conjunction with measurement of serum IgA levels. Partial IgA deficiency has been reported in 6.7% of 3818 healthy blood donors in northern India.[76] IgA deficiency is present in a similar number of patients with CeD.[77] In these patients, it is likely that the IgA serological tests may be negative.

18. In patients with IgA deficiency, an IgG based test such as IgG anti-tTG Ab or IgG anti-deamidated peptide Ab may be used.

Though IgA-tTG performs better than IgG-tTG in children and adults, the IgG-tTG test remains relevant in IgA-deficient cases.[70] IgG-deamidated gliadin peptide (DGP) ELISAs may also be used in IgA-deficient individuals. The sensitivity of IgG anti-tTG ranges from 75% to 95% and the specificity

from 94% to 100%, while sensitivity and specificity varies from 80% and 98% for IgG anti-DGP.[78]

Mucosal histological changes

19. Changes in small intestinal biopsy are essential for the diagnosis of CeD.

Originally the diagnosis of CeD hinged on the finding of characteristic abnormalities on small intestinal biopsy. As serological tests were developed, small intestinal biopsy began to be performed only after serological screening. Nevertheless, changes in small intestinal biopsy are considered essential for the diagnosis of CeD that manifests clinically. The use of anti-tTG titres in CeD diagnosis is not validated in India. False positive serology is noted in conditions other than CeD. Thus, in the Indian context, patients should not be put on lifelong GFD solely on the basis of serology.

20. The severity of enteropathy is demonstrated by villous abnormalities which vary from increase in intra-epithelial lymphocytes [IEL] to marked severe villous abnormality.

The enteropathy of CeD predominantly affects the proximal small intestine. It is often patchy. It is characterized by an increase in intraepithelial lymphocytes, elongation of the crypts, and shortening and atrophy of the villi. In severe cases, there may be subtotal or even complete villous atrophy. Other changes include thickening of the basement membrane under the surface epithelium, reduced numbers of goblet cells, inflammatory infiltration in the lamina propria, and changes in the intestinal epithelial cells including a cuboidal morphology, loss of basal nuclear orientation, and cytoplasmic vacuoles.[79]

21. The modified Marsh Oberhuber classification of villous abnormalities, which grades severity of villous atrophy based on two parameters i.e. increase in IELs and villous height to crypt depth ratio, is recommended for evaluating well-oriented CeD biopsies.

Michael Marsh was the first to quantify changes in the small intestinal mucosal histology in CeD.[80,81] The classification that he proposed was subsequently modified by Oberhuber[82] and has come to be known as the modified Marsh classification or the Marsh-Oberhuber classification **(Table 3)**. Although other classification systems have been developed, the Marsh-Oberhuber system is the most commonly used worldwide, and the expert group recommends its use in reporting small intestinal biopsies from patients with CeD. Grades 2 and 3a-3c in this classification are consistent with clinical CeD in patients with positive serology. The expert group also recommends the use of a cut-off of 40 IEL/100 enterocytes as elaborated in the next statement.

Table 3. Marsh-Oberhuber classification of small intestinal mucosal histology in CeD

Marsh Type	IEL / 100 enterocytes	Crypt elongation	Villous atrophy
0	<30	-	Absent
1	>30	-	Absent
2	>30	+	Absent
3a	>30	+	Mild
3b	>30	+	Severe
3c	>30	+	Complete

22. Increase in IEL (>30/100 epithelial cells) is not specific for CeD, and is present in many other conditions such as giardiasis, tropical sprue, bacterial overgrowth, etc.

Traditionally, counts greater than 40 IELs per 100 epithelial cells were considered to be abnormal.[83] Subsequently this was reduced to 30/100 epithelial cells. However, IELs may be increased in a number of conditions, particularly in giardiasis, tropical sprue and bacterial overgrowth. Thus, the expert group recommends that, in India, we retain the number of 40 IELs/100 epithelial cells when considering a diagnosis of CeD.

23. Four to six mucosal biopsies should be obtained from the mucosal folds in the second part of the duodenum and should be oriented well for interpretation.

The traditional recommendation for CeD diagnosis in the era of GI endoscopy is to obtain biopsies from the second or third part of duodenum and orient them well for interpretation. However a problem arises because CeD lesions may be patchy and there may be sampling error in obtaining biopsies. It has been shown that, especially in children, duodenal bulb biopsies may be just as good as biopsies from the second part of duodenum in diagnosing CeD, and indeed it is now recommended practice to sample both areas particularly when biopsying children for CeD.[84-86] With the new high definition endoscopes, it is possible to see areas of patchy villous atrophy particularly when incorporating narrow band imaging and this may be used to target biopsies in individuals with difficult problems.[87,88] Orientation of endoscopic biopsies is difficult. Studies show that less than 39% biopsies could be oriented properly.[89]

24. The biopsy report should include comments about IELs, villous height and crypt depth ratio and should be graded as per modified Marsh Oberhuber classification.

Reporting of biopsies in CeD should be done in a structured manner with comments about IEL number, villous to crypt ratio, and lamina propria inflammation. The expert group recommends the use of the Marsh Oberhuber classification for grading biopsy changes in CeD. Formal enumeration of IELs includes selecting suitably oriented villi in the biopsy specimens and then counting the total IELs present per 100–1000 epithelial cells along the luminal margin, excluding the crypt. The total number of IELs is expressed relative to 100 epithelial cells.

Genetic studies

25. While the majority of patients with CeD are HLA-DQ2/-DQ8 homozygous, heterozygous or compound heterozygous, HLA typing is not required routinely for the diagnosis of CeD.

Although a number of non-HLA genes have been associated with a risk for CeD, the HLA genes provide the strongest genetic risk for CeD. The majority of CeD patients express the HLA-DQ2.5 heterodimer encoded by HLA-DQB1*02 and HLA-DQA1*05 alleles. This is expressed either in cis on the DR3-DQ2.5 haplotype (DQB1*02:01, DQA1*05:01, and DRB1*03:01) or in trans (heterozygous for haplotypes DR5-DQ7 and DR7-DQ2.2), where the HLA-DQ2.5 heterodimer is encoded by DQB1*02:02 and DQA1*05:05. However, the diagnosis of CeD does not depend on the detection of the HLA genotype. This is because the above-described HLA makeup is present in a significant proportion of the normal population. It is needed for the development of CeD, but does not always result in the manifestation of CeD. Although a varying proportion of the Indian population, from 13% in certain communities to 30% in other communities, has the genetic background to express HLA-DQ2 and/or HLA-DQ8,[38-40] less than 1% of the population will actually have serological and clinical features of CeD. Thus the above HLA makeup is permissive for CeD development, but does not solely explain its development. Because of this, HLA study does not help in making a positive diagnosis of CeD. On the other hand, the vast majority of tested individuals with CeD in India have either DQ2 or the genetic makeup to express DQ2. Thus, 93-100% of children with CeD had DQ2 or DQB1*0201.[90,91] Therefore, absence of HLA-DQ2/-DQ8 types has good negative predictive value for the diagnosis of CeD. In other words, if there is doubt about likelihood of CeD in a child, HLA testing may exclude the diagnosis if DQ2/8 are absent. None of the genetic markers has any value at present in the diagnosis of CeD.

SECTION 5: WHO SHOULD BE SCREENED?

26. Screening for CeD should be considered in several groups of patients. These include adults or children with chronic diarrhea, chronic iron deficiency anaemia, unexplained short stature, failure to thrive in childhood, unexplained infertility, and unexplained osteopenia or osteoporosis.

CeD screening should be considered in siblings of patients with CeD. As noted earlier, there is a familial increased risk of CeD, which is higher in siblings than in parents or offspring. Hence siblings of index patients with CeD may also be screened for CeD and followed up if asymptomatic. In addition, there are a number of clinical conditions where CeD may explain the disease. These include chronic diarrhoea, iron deficiency or folate deficiency anaemia, particularly if refractory to oral therapy, in children with unexplained short stature or failure to thrive, unexplained infertility and unexplained osteopenia and fractures. Patients with unexplained infertility, recurrent miscarriage or intrauterine growth retardation have been found to have a significantly higher risk of CeD (Odds ratio 5.0, 95% CI 2.1-11.3 for infertility; OR 5.8, 95% CI 2.3-14.0 for recurrent miscarriage; and OR 8.73, 95% CI 3.2-23.5 for IUGR) than the general population.[92]

27. There is an association between Type 1 diabetes mellitus and CeD.

Western guidelines recommend targeted CD screening in patients with type 1 diabetes who have classic symptoms, such as abdominal pain, bloating, diarrhea, unexplained weight loss or labile metabolic control as they are at higher risk for microvascular comorbidities.[93] The global reported prevalence of CeD in T1DM is 2-10% while the data from studies in India shows a prevalence ranging from 7% to 14.9%.[94-97] The prevalence of T1DM in CeD in India is about 3.6% and 4.2%. Most diabetic children have silent or subclinical CeD. Children with subclinical CeD may develop growth failure, frequent hypoglycemia and osteopenia. Risk factors for development of CeD in T1DM include genetic predisposition, young age at diabetes onset, female gender and early introduction of gluten in the infant's diet. Targeted screening for CeD may therefore be useful in patients with type 1 diabetes mellitus.

28. Patients with CeD may have associated autoimmune diseases such as autoimmune thyroiditis and autoimmune liver disease.

Table 4 shows a number of diseases that are described to be associated with CeD.[98-120] It is advisable to maintain a high index of suspicion in these patients and to do targeted screening for CeD when indicated. A significant number of patients with CeD have increase in serum transaminases and they respond to gluten free diet. Pooled prevalences of positive CeD serology and biopsy-proven CeD in cryptogenic hypertransaminasaemia has been reported as 6% (95% CI 3% to 10%) & 4% (95% CI 1% to 7%) respectively. Pooled prevalence of abnormal serum transaminases in newly diagnosed CeD was 27% (95% CI 13% to 44%). Exclusion of gluten led to normalisation of serum transaminase levels in

63%-90% in less than 1 year. Persistent elevation of serum aminotransferase activity is a manifestation of liver damage related to CeD. Most frequent is a mild asymptomatic liver injury, which is reversible on a gluten-free diet. Autoimmune hepatitis is reported to be associated with CeD and reflects severe and progressive inflammatory liver damage, induced by an autoimmune process that may not respond to gluten withdrawal. Data from the paediatric patients shows a wide range of prevalence of CeD in autoimmune hepatitis of 11.5-46%.

Table 4: CeD associations with other diseases

Disease / Syndrome	Reference
Type 1 diabetes mellitus	Costa-Gomes et al, 2015 Nagesh VS et al 2015 Elfstrom P et al, APT 2014 Nijhawan S et al 2013
Grave's disease	Kyriacou et al 2015 Joshi AS et al 2014
Hypothyroidism	Kyriacou et al 2015 Nijhawan S et al 2013
Autoimmune polyglandular syndrome type II	Maturu A et al 2014
Unexplained infertility and recurrent miscarriage	Tersigni C et al 2014
Down syndrome	Costa-Gomes et al, 2015
Autoimmune hepatitis	Muratori P et al 2015 van Gerven NM et al 2014
Cryptogenic cirrhosis	Maiwall R et al 2014 Singh P et al 2013 Nijhawan S et al 2013
Primary biliary cirrhosis	Muratori P et al 2015
Non cirrhotic portal hypertension	Maiwall R et al 2014 Nijhawan S et al 2013
Pediatric rheumatologic disease	Sherman Y et al 2015
Dermatitis herpatiformis	Antiga E et al 2015
Lichen sclerosus	Jacobs L et al 2014
Psoriasis	Bhatia BK et al 2014
Enamel hypoplasia	Ferraz EG et al 2012
Gastric hyperplastic polyps	Galvez-Rios S et al 2014
Drug resistant focal epilepsy with focal seizures	Casciato S et al 2014
Myoclonus ataxia	Sarrigiannis PG et al 2014
Autistic spectrum disorders	Ludvigsson JF et al 2013
Pure red cell aplasia	Chatterjee S et al 2014

SECTION 6: DIAGNOSTIC CRITERIA

29. The diagnosis of CeD should be based on the basis of a combination of clinical manifestations, a positive serology and presence of villous abnormalities of at least Marsh grade 2 on duodenal biopsy.

As mentioned above, the diagnosis of CeD is based on the combination of clinical manifestations, positive IgA anti-tTG antibody, and a deep duodenal biopsy demonstrating the presence of villous abnormalities of at least Marsh grade 2. False positive serologic tests are well known to occur in a number of conditions including chronic liver disease, rheumatoid arthritis, inflammatory bowel disease and other autoimmune diseases. These diseases are not accompanied by the typical histologic changes of CeD on duodenal biopsy. Follow up over years may show that the celiac serology positivity changes over time in these patients and is not consistent. In order to negate a false diagnosis of CeD it is therefore considered advisable to include all three parameters in making a diagnosis of CeD. Patients who are serology positive but have normal biopsies can be followed up over time to determine whether they are likely to develop CeD.

30. Where duodenal biopsy is not considered feasible for any reason, a diagnosis of CeD can also be considered in presence of clinical manifestations, a positive serology of two different kinds such as anti-tTG Ab and a positive anti-endomysial Ab, and the presence of either HLA-DQ2 or DQ8 in the individual.

The European Society for Pediatric Gastroenterology, Hepatology and Nutrition indicates that the diagnosis of CeD may be made on the basis of two positive serological tests even in the absence of duodenal biopsy.[70] In particular it is suggested that the tests may include a recombinant antigen and deamidated gliadin peptides, or may include IgA anti-tTG and IgA anti-EMA. They appear to be also potentially applicable to less developed countries, but require the use of HLA testing in the absence of biopsy.[121,122] The criteria for diagnosis of CeD in the absence of biopsy are yet to be defined for India. There may be occasional situation, where intestinal biopsy is not possible due to various factors but clinical picture is strongly suggestive of CeD. The expert group recommends that, in the background of suggestive clinical manifestations and characteristic HLA profile, positive serology using two antibody tests can be used to confirm a clinical diagnosis of CeD. IgA anti-tTG titres x 10 times the ULN has been shown to be associated with histological changes of Marsh grade 2 or 3 in Indian patients with CeD.[123] In symptomatic patients with positive serology but where the degree of villous abnormality is not quite

significant, ie. Marsh grade 0 or Marsh grade I, the diagnosis of CeD may be considered on a case to case basis, basically relying more on clinical findings in the individual. Under such circumstances, the label of CeD should be avoided unless the patient shows response of both clinical symptoms and serological titre to a gluten free diet. An empirical trial of gluten free diet should generally be avoided in individuals with normal or indeterminate serology.

31. The diagnosis of CeD should not be made based only on a positive celiac serological test irrespective of its titre.

The expert group was of the opinion that a diagnosis of CeD should not be based solely on a positive celiac serology test, even if the test was positive in very high titre, eg 10 x ULN. In a study from India, a 10-fold rise in titre was associated with Marsh grade 2 or greater abnormality with a specificity of 95%.[123] Thus 5% of suspected patients but otherwise not CeD would be subjected to lifelong GFD if diagnosed with CeD on the basis of titre alone.

32. All asymptomatic patients who are tested positive for celiac serological test on screening and have villous abnormality of Marsh grade 0 or 1 can be labelled as having potential CeD.

In keeping with current international opinion, the expert group suggests that asymptomatic individuals who test positive for celiac serology using any of the recommended tests (IgA anti-tTG, anti-EMA, anti-DGP) can be labelled as having potential CeD. The group prefers this term to "latent" CeD. The term potential CeD implies that the participant may or may not progress to clinical CeD during the course of his/her lifetime.[124] Such individuals should be followed up. There is as yet little data to guide recommendations as to clinical indicators, frequency of serologic testing, indicators for intestinal biopsy and role of HLA typing for follow up in such cases.

SECTION 7: MANAGEMENT

Avoidance of gluten in diet remains the cornerstone for management of CeD. Optimal compliance must be followed by patients and checked by the treating physicians on every visit/follow up/ complication. A suggested list of best practices is provided in Table 5 below.

Table 5. Best practices for caregivers of Celiac Disease patients

Do's	Dont's
Use home prepared foods in which you can monitor presence of gluten	**Avoid eating outside homes or in places where wheat contamination of foods may be common**
Buy foods that are labelled as gluten-free	**Do not buy foods that do not have label and may be contaminated with wheat**
Use a separate grinder or chakki for preparing flour for a CeD patient	**Do not buy flour from local mills where wheat may also be grounded and can contaminate**
Reinforce the message of GFD to the patient periodically	
Educate the child's teachers about the importance of GFD for the child with CeD in order to avoid compulsion to eat wheat products in school	
Ensure that medication administered to the patient do not contain wheat flour as filler	

33. Lifelong and complete avoidance of gluten or gluten containing dietary items is the most effective and the main stay of treatment of CeD. While planning gluten-free diet, all patients should be counselled for a balanced diet as per their nutritional requirement.

The principal treatment for CeD is lifelong and complete avoidance of gluten in the diet.[125-129] Gluten is found in grains that contain prolamines from wheat or any Triticum species, such as spelt, durum wheat, rye, barley, or their crossbred varieties.[128-131] Because of its viscoelastic properties, gluten is used extensively in the food and other industry, and may be found in many items used daily such as lipsticks, postage stamps, beer, ice creams, sweets, confectionary, tablets, and excipients.[126-129]

It is important to understand that many people with CeD are nutritionally deficient because of malabsorption of nutrients. The diet of an individual with CeD should not only be free from gluten, but it should be healthy and well-balanced in terms of all the macro- and micronutrients.[126-134] The amount of the nutrients needed by an individual vary with his/her age, gender and level of activities which he/

she does in his/her day to day life. A balanced diet is one which contains different types of foods in such quantities and proportions that provides adequate calories, proteins, fats, minerals, vitamins and other nutrients. A balanced diet should comprise of 45-65% carbohydrate, 20-35% fats and 10-30% proteins.[135-136] The diet should be planned using right combination of five food groups such as cereal products; pulses and legumes; milk, egg and flesh foods; fruits and vegetable; and fats and sugar as suggested by the Nutrition Expert Group of Indian Council of Medical Research.[137]

34. Patient and family including caregivers should be counselled in detail about the nature and lifelong treatment of the disease.

CeD is a life-long disease "once celiac, always a celiac".[126-134] Patients and their family should be counselled about the nature of the disease and the requirement of life-long dietary adherence to GFD. The management of CeD is truly different and unique from the treatment of other medical or surgical diseases. The center stage of treatment of CeD is dietary counselling and regular reinforcement for adherence by a nutrition specialist/dietician.[126-133] It is not always easy for patients and family to believe that the patient cannot ever eat wheat products and therefore likely to go to alternative medicine. It is therefore, extremely important that patients and their families are counselled well about the disease and the treatment.[128,129,131]

35. A nutritionist or dietician should be involved in counselling of a patient and their family.

All patients with CeD should be referred to a dietician well versed in counselling patients and their families about the disease and GFD.[126-134] The dietary counsellor should have sufficient knowledge about the food and food products. It is not only about prescribing GFD but also is to provide an individual patient specific well balanced diet. While a physician can diagnose the disease; he/she may not have sufficient knowledge or time to explain to the patient about gluten-free diet; and therefore a nutrition specialist/dietician must be involved in the management of such patients. Insufficient education about the GFD can result in poor adherence, frustration and increased health-care costs due to patients seeking medical care for ongoing symptoms and/or complications. If left to a GFD on their own, patients encounter misinformation and may unnecessarily restrict intake. Other practical topics such as how to avoid contamination at home and school/work place, travel and restaurant tips should be discussed with patients and their families. Sufficient time should be spent in counselling and reinforcing messages periodically.[129-131,138]

36. A list of restricted food and allowed food should be provided.

All patients and their families should be provided with a list of food items which are safe and which are not safe for them. Printed formats are of great help. In case of non-availability of printed booklets, the physician and/ or dietician should provide all the instructions in the written format.[126,129,131] **(Table 6)** Flour may be ground at home using an electric or manual chakki in order to avoid contamination with wheat which may happen in commercial facilities.

Table 6: Some examples of diet recommendations for CeD @

Food products	Food allowed	Food not allowed
Flours @@	▪ Rice, Pulav, Biryani ▪ Dosa ▪ Poha ▪ Idli ▪ Sago ▪ Besan pakora ▪ Jowar flour ▪ Bajra roti ▪ Arrowroot flour ▪ Maize flour ▪ Gram flour ▪ Water chestnut flour ▪ Kuttu ka atta ▪ Soya bean flour ▪ Rice flour	▪ Wheat flour (atta) ▪ Refined wheat flour (maida) like in samosa, broken wheat (dalia) semolina (sooji), vermicelli (siwain) noodles, chowmein, pastas, spaghetti, macaroni ▪ Barley flour (jaun) (roti, paran-tha, poori, naan) ▪ Oats@ (jai) beer, malt, maltova
Bakery Products	▪ Home prepared biscuits or cake replacing wheat flour, with rice flour, arrowroot flour corn flour / millet flour	▪ Bread ▪ Burger ▪ Pastry ▪ Biscuits ▪ Cookies ▪ Nan khatai ▪ Patties ▪ Pizza ▪ Cakes ▪ Fanes
Sweet and confectionary	▪ Sugar candy (poppins, chlo romint, mango bite, orange candy) ▪ Home prepared sweets and ice-creams, jams, jellies ▪ Halwa (gajar halwa, aloo halwa) ▪ Besan ladoo ▪ Kheer (carrot kheer, rice kheer, sabo dana kheer, makhana kheer) ▪ Gajak ▪ Phirni ▪ Chikki ▪ Petha mithai	▪ Chocolates ▪ Toffees (with milk and chocolate) ▪ Chewing gums ▪ Most sweetmeats (mithai) ▪ Ice-cream and ▪ Ice-cream mixtures ▪ Custards ▪ Milk cakes ▪ Barfi ▪ Jalebi ▪ Atte ka ladoo

@@ Oats are better avoided as they are often contaminated with wheat, and about 5% of CeD patients may also be intolerant to pure oats;

@ The above list is indicative and there may be regional and state specific differences in recipes; please confirm the presence and absence of gluten from the nutritional counsellor

Food products	Food allowed	Food not allowed
Beverages and soups	▪ Milk ▪ Butter milk ▪ Coffee ▪ Tea ▪ Squashes ▪ Fruit juices. ▪ Home prepared clear soups (only stock no refined flour)	▪ Commercial Nutritional drinks e.g. bournvita, complan, boost, horlicks, maltova etc. ▪ Chocolate drinks ▪ Barley water ▪ Canned soup ▪ Soup mixes ▪ Thick soups (broth thickened by barley or macroni) ▪ Most stews (thickened by refined flour)
Other processed products	▪ Cottage cheese (preferred home prepared) ▪ Home prepared sauces e.g. tomato sauce, rice or cider vinegar ▪ Home prepared pickles	▪ Cheese (mozarella) ▪ Corn flakes (due to malt added) ▪ Sauces ▪ Puree ▪ Instant curry mixes ▪ White vinegar ▪ Mayonnaise, commercial salad dressing, pickles (in white vinegar) ▪ Mathri
Namkeens and snacks	▪ Dal namkeen ▪ Besan ke sev (home prepared) ▪ Potato chips ▪ Idli ▪ Finger chips ▪ Banana chips ▪ Popcorn ▪ Roasted channa ▪ Sprouted dal (lobia,moong and channa) ▪ Tikki (aloo, channa dal)	▪ Bread pakora ▪ Bread roll ▪ Samosa
Food Thickening	▪ Recipes thickened by corn starch ▪ Arrowroot flour ▪ Rice flour ▪ Potato starch ▪ Tapioca starch ▪ Water chestnut (singhary ka atta) ▪ starch ▪ Ground onion ▪ Coconut ▪ Poppy seeds ▪ Food coated by gram flour batter	▪ Recipes thickened by wheat flour ▪ Recipes with stuffing of bread crumb ▪ Food coated by wheat flour or bread crumb

@@ Oats are better avoided as they are often contaminated with wheat, and about 5% of CeD patients may also be intolerant to pure oats;

@ The above list is indicative and there may be regional and state specific differences in recipes; please confirm the presence and absence of gluten from the nutritional counsellor

37. Exposure to even small amounts of gluten is sufficient to maintain the disease.

Gluten intake varies from population to population and depends upon dietary practices in the particular region. The wheat intake is higher in Northern part of India in comparison to that in the Southern and North-Eastern part of India. A typical North Indian diet, where flat bread is the usual meal, contains about 25-30gm gluten per day; whereas average gluten intake in the Western countries varies from 10-20gm/day.[126,129,131,138,139] In a double blind, placebo controlled prospective study; Catassi, et al demonstrated that an intake of as little as 50 mg of gluten per day for 3 months was sufficient to cause a significant decrease in the intestinal mucosal villous height/crypt depth ratio.[140] This will equate to the amount of gluten present in one bite of chappati. A daily intake of gluten of lower than 10 mg is unlikely to produce significant histological abnormalities.[141,142] While complete adherence of GFD is essential, it is challenging for patients to maintain good adherence to GFD because of extensive use of gluten in the processed food industry. [126,129,131,143]

38. Patients with CeD are likely to have nutritional deficiencies and should receive appropriate nutritional supplementation. This may include vitamin D, calcium, iron, zinc, vitamin B12 and other macro and micronutrients.

During initial phase of the treatment, patients with CeD should be given supplements of vitamins, minerals, and extra protein supplement to overcome deficiencies and replenish nutrient stores.[144-145] Up to 80% of patients with CeD in India have anemia.[145,146,51]Anemia should be treated with iron, folate, or vitamin B12 depending on the type of anemia. Bone disease is frequent in patients with CeD, resulting from malabsorption of calcium and/or vitamin D, with subsequent osteopenia, osteoporosis, or osteomalacia.[147] All patients with CeD should receive elemental calcium and vitamin D3 supplementation.[126,129,148] Secondary lactose intolerance may occur in some patients with CeD, a low lactose diet may be useful in controlling symptoms, at least during initial few months of treatment.[129]

39. All patients should be encouraged to participate in celiac patient support groups.

All patients with CeD should be encouraged to join celiac disease patient support groups. The support group may play varied roles such as social and emotional support, awareness of local and national sources of gluten-free foods; practical advice for GF food preparation, dissemination of up-to-date information, travel information and/or experience. There are data to support that the members of celiac support groups have better dietary adherence.[151] At present, there are very few celiac support groups in India and there is need of creation of many such groups.

Monitoring of patients

40. Most patients respond to gluten-free diet within weeks to months.

Response to GFD is remarkable within weeks in most patients. Improvement in overall well-being is one of the early symptoms to recovery because of improvement in energy supply, correction of deficiencies of micro-nutrients and improvement in hemoglobin. [126,131,129,150-151] Serologic titres fall soon after initiating a GFD, with substantially lower antibody levels at 1 year with ongoing decline to negative/normal by 2 years.[152,153] Although antibody levels will decline in partially adherent patients, the rate of decline is less than with strict compliance. However, negative serology does not guarantee strict adherence to GFD; in addition, many patients have ongoing villous abnormalities despite negative serology. Histologic improvement is slow in adults and delayed compared with symptomatic or serologic improvement. Persistent injury is more likely in those who are non-adherent to GFD. Mucosal recovery is faster and more complete in children, with 95% recovery in 2 years and 100% recovery long- term in children following a GFD.[154-156] Follow-up visits can be scheduled after 1-3 months according to clinical presentation, and serology should be repeated after 6 months. Subsequently patients may be followed at one yearly interval. At follow up visits, patients should be assessed for clinical symptoms, dietary adherence and serology. A structured assessment should be done at clinic visits for the adherence to GFD. Patients can be assessed, if necessary, for histologic improvement after 2 years on a GFD.

41. CeD patients should be followed at regular intervals and should be monitored for compliance to gluten-free diet, their clinical parameters such as weight, height (for growing age), resolution of symptoms, and improvement of haemoglobin.

A key to the success of the GFD is adherence to GFD. Dietician-led evaluation by direct history taking, food records, and cross-check questioning is very useful for assessing the adherence to GFD.[126,129,131,133,157,158] Four targets of therapy have been proposed. The traditional target, relief of symptoms is readily assessable and important, especially because symptom avoidance is a major motivation for adherence to the GFD and is directly related to quality of life. The second target is correction of nutritional deficiencies.[133] This is of paramount importance in children because physical growth, rapid catch–up in height, and normalization of body mass index is associated with institution of GFD in a child with newly diagnosed CeD. The third potential target is to normalize immunological abnormalities such as normalization of serological titre. The final target is to achieve mucosal healing, which is an excellent surrogate for correction of immunological activation and is associated with improved outcomes in terms of morbidity and mortality.[133]

42. Dietary counselling is an ongoing process and should be periodically reinforced.

Patients and their families require counselling and education about the disease and an ability to identify (cross-contamination and hidden sources) gluten in food to enable them to make appropriate choice of food items. They should also be counselled about how to eat out of home and how to maintain an adequate nutritional intake. Repeated counselling is associated with better rate of adherence to GFD.[126,129,131,133,159-161] Patients with excellent and good adherence to GFD show significantly higher improvement not only in the celiac related symptoms but also in the level of hemoglobin and weight after 6-months of follow-up in comparison to those with poor adherence to GFD.[161]

43. The most common cause of either partial response or non-response is poor compliance to the dietary restrictions. Other causes are bacterial overgrowth, lactose intolerance, microscopic colitis, parasitic infection and refractory celiac disease.

Most common cause of partial or non-response to GFD in India is poor adherence to GFD.[126,129,133] There are many barriers to adherence to GFD in India such as inappropriate counselling, non-availability of gluten-free food, and lack of labelling for GF food. Non-response in symptoms of CeD even after 6 months of GFD despite compliance should raise the possibility of small intestinal bacterial overgrowth, autoimmune enteropathy, tropical sprue, drug-associated enteropathy (such as olmesartan), microscopic colitis, and eosinophilic gastroenteritis.[162-164] A persistent or recurrent elevation of serologic titers suggests ingestion of gluten either voluntary or adventitious.[126,129,131] There may be gluten exposure in non-food items such as medications, supplements, cosmetics, and glues.

44. Serological tests at 6 months and one year can be used to monitor adherence.

Serologic titers fall soon after initiating a GFD, with substantially lower antibody levels at 1 year with ongoing decline to negative/normal by 2 years. Normal serology does not guarantee strict GFD adherence; in addition, many patients have ongoing mucosal atrophy despite normal serology.[150,151]

Section 8: Complications

45. Complications of CeD include refractory CeD, celiac crisis, ulcerative jejunitis and malignancies including enteropathy T cell lymphoma [EATL], and other malignancies of small intestine and other part of GI tract.

Complications are uncommon in patients with CeD. The complications of CeD include refractory CeD, celiac crisis, ulcerative jejunitis and malignancies including enteropathic T-cell lymphoma and

non-Hodgkin's lymphoma.[126,128,129,133] Severely malnourished patients can develop refeeding syndrome after institution of GFD and nutritional supplements.[165]

46. Refractory CeD (RCD) may complicate CeD, but is not commonly diagnosed in India currently.

A few patients with CeD show a lack of response to GFD despite maintaining a good adherence to diet. Patients having persistence or recurrence, after initial response, of clinical symptoms and histological abnormalities despite strict adherence to the diet for more than 12 months are diagnosed as having RCD.[166-168] While exact prevalence of RCD is currently unknown, approximately 5% of patients with CeD are expected to have RCD. RCD is classified into two types such as RCD I and RCD II based on phenotypically normal and aberrant intraepithelial T lymphocytes in the small intestinal mucosa, respectively. Intraepithelial T lymphocytes are considered aberrant when they express cytoplasmic CD3, but lack surface expression of the T-cell markers CD3, CD4, CD84 and the T-cell receptor. To discriminate between RCD I and RCD II, a cut-off value of 20% aberrant intraepithelial T lymphocytes, determined by flowcytometry in small intestinal biopsies, is used. In the absence of flowcytometry, immunohistochemistry for CD3 and CD8 can be used as a first-line screening test for RCD.[166-168] RCD I has better prognosis and the 5-year survival rate in them is between 80% to 96%. RCD II is a more serious disease and the 5-year survival in them varies from 44% to 58%. The patients with RCD II are at higher risk of developing lymphoma as a consequence of clonal expansion and further transformation of aberrant intraepithelial T lymphocytes into enteropathy associated T-cell lymphoma (EATL). [166-168] RCD I can be treated effectively with prednisone with or without azathioprine. The options for type II RCD include cladribine therapy and autologous stem cell transplantation. Interleukin-15 blocking antibody is a promising new therapeutic alternative for RCD.[166-168]

CeD is associated with a 1.3-fold greater risk of malignancies, in particular lymphomas, than that in the general population. The principal malignancy associated with CeD is EATL. Unexplained weight loss, abdominal pain, fever and night sweating should alarm physicians of the presence of an overt EATL. Any part of GI tract can be affected in EATL, the most frequent site being proximal jejunum. EATL may even involve organs outside the gastrointestinal tract, for example in the lungs, ribs and spleen.[168-169]

Section 9: Special situations

47. Gluten re-challenge can be considered in patients with equivocal results in whom gluten free diet has not resulted in clinical improvement.

There are certain situations in which GFD may not result in the expected clinical improvement in a patient diagnosed with CeD. This may indicate poor adherence to GFD, occurrence of RCD, small intestinal bacterial overgrowth or microscopic/collagenous enteritis or colitis. Further investigation is

required in these patients. There are also situations in which gluten rechallenge may be considered. This may typically include patients in whom a diagnosis of CeD was made but the patient was simultaneously treated with a slew of measures including better nutrition, GFD, specific vitamin supplements, and antibiotics. These patients may have improved, but may then not wish to continue a GFD. At this stage, CeD diagnosis may be confirmed if gluten rechallenge results in recurrence of mucosal and/or clinical abnormalities. Gluten rechallenge should be done in graded manner under proper medical supervision and should be avoided during the development of dentition and during puberty.

48. Gluten rechallenge is not mandatory in children below 2 years with positive serology and histology and response to GFD.

Children below 2 years of age with classical CeD presentation may have negative serology. Due to the serious nature of their illness, they are sometimes started on GFD on the basis of abnormal mucosal histology and the children may improve. When they recover, the issue of lifelong continuance of GFD comes up as these children may have had a transient enteropathy due to some other illness. Under such circumstances, it is considered appropriate to rechallenge these children with gluten to confirm CeD diagnosis. Rechallenge with gluten was considered the gold standard for CeD diagnosis at a time when newer serological tests and HLA studies were not available. With the available of other corroborative evidence for CeD, gluten rechallenge may be unnecessary if there is positive serology, appropriate histological changes, and a clinical response to GFD.

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PUBLICATIONS ON CELIAC DISEASE IN INDIA (FIGURE 2) Annex 1

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