Prevalence of antibodies to hepatitis C virus in coastal Orissa

The epidemiology of hepatitis C virus (HCV) shows a wide geographic variation. There is limited data from India on HCV prevalence.14 We estimated the prevalence of HCV infection in coastal Orissa.

Blood samples were collected from 27,604 apparently healthy blood donors (25,582 men; 21,617 were replacement donors and 5,987 voluntary donors) during the period April 2002 to March 2003. The voluntary donors included 3030 who had donated at our blood bank and 2957 who donated blood at the blood donation camps conducted by the blood bank. All samples were tested for anti-HCV antibodies employing a third-generation enzyme-linked immunosorbent assay (LG HCD 3.0 Plus; LG, Korea). This assay employs six recombinant antigens: core, NS3, NS4, NS5, E1 and E2.

Only four of the samples tested were anti-HCV positive (prevalence 0.014%). All the four were from male replacement donors. Thus the prevalence was 0.02% in them and 0% in all other groups.

These figures are low as compared to those from other parts of the country. This is despite the fact that we employed ELISA-3, which is comparable to RIBA.7 The reported anti-HCV prevalence rates in other parts of India vary from 0.90% to 1.8%.14 The low prevalence in our study could be due to the fact that intravenous drug abuse is very uncommon in Orissa, and the practice of invasive methods of medicine too is relatively infrequent.

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Low vitamin E level as a possible cause of complications in celiac disease

Sood et al6 reported a woman with celiac disease with nonalcoholic steatohepatitis (NASH). The authors stated that pathogenic mechanisms for liver changes in celiac disease are not clear, and mentioned increased intestinal permeability to toxins and antigens as a possible cause.

The mechanism(s) for NASH are likely multifactorial; oxidative stress is likely to play a key role.2 Vitamin E may reduce oxidative stress and liver injury.23

Odenti et al4 reported that levels of markers of oxidative stress in celiac disease were elevated and concentrations of α-tocopherol in plasma were low. The development of neurological changes in celiac disease may be related to vitamin E deficiency,5 but no systematic studies have been done on vitamin E status in celiacs.

Table: Concentrations of α-tocopherol in newly diagnosed patients with celiac disease

<table>
<thead>
<tr>
<th>Sex/Age(y)</th>
<th>Plasma tocopherol (μg/L)</th>
<th>Plasma tocopherol/serum cholesterol ratio</th>
<th>Erythrocyte tocopherol (μg/mL)</th>
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</thead>
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<tr>
<td>M/2</td>
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<td>2.7</td>
<td>1.5</td>
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<td>M/4</td>
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<tr>
<td>F/17</td>
<td>17.3</td>
<td>4.0</td>
<td>2.2</td>
</tr>
</tbody>
</table>

References

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We assessed plasma and erythrocytes α-tocopherol levels in six newly diagnosed patients with celiac disease (Table). In all patients, concentrations of vitamin E in erythrocytes were below the normal. Vitamin E is safe and relatively inexpensive. In our opinion, it would be reasonable to investigate α-tocopherol status and liver function and to evaluate vitamin E therapy in celiac patients with NASH.

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Adult celiac disease in northern India

Sood et al.1 deserve credit for compiling the largest series of cases with adult celiac disease from northern India.

The most difficult aspect of celiac disease is to establish the diagnosis and ensure dietary compliance.2 Thirty-two patients (32.3%) in this series had typical non-diarrheal presentation. A repeat duodenal biopsy to demonstrate improvement in villous structure on gluten-free diet is advisable in such cases because clinical improvement is subtle and slow.2

Less than ten percent of patients in the series had a follow up of >3 years. In our experience, dietary compliance in patients with celiac disease is a major problem. Reasons for non-compliance are financial, social, ignorance and temptation. Compliance is usually good in the first 2 years. As they began to feel better, some degree of indiscretion (dietary cheating) is seen with most patients, especially since occasional dietary cheating does not produce immediate dire consequences. In northern India this problem is even more as wheat is consumed in some form in all meals.

Thus, there is reason to doubt the high level of compliance (90%) reported by Sood et al. The fact that the hemoglobin level increased from 8.6 g/dL to only 9.8 g/dL even after 2 years of follow up supports our stand. Normalization of hemoglobin (to more than 12 g/dL) occurs in almost all patients within 6 months of gluten-free diet. In a recent study from Italy, hemoglobin increased to >13 g/dL at 24 months, suggesting excellent dietary compliance.3

Adequacy of compliance is best checked by serial determination of anti-gliadin antibodies. In the sub-group of celiac disease patients with raised transaminases, normalization of AST/ALT may also be used as a cheaper alternative. Neither of these was used in the current study, and the authors relied only on the dietary history to assess compliance.

Ensuring compliance in patients with celiac disease is of utmost importance as the risk of development of two complications, viz., metabolic bone disease and T-cell lymphoma, is decreased only in patients who adhere to gluten-free diet.

Finally, although mention is made by the authors that vitamin B12 levels were estimated, this important data has not been provided. Also, the data regarding anti-transglutaminase antibody has some discrepancy; the authors state that it was done in only 2 cases but also that positive results were obtained in all 44 patients in whom it was tested.

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References

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Reply from the authors

We appreciate the interest Dr Puri and colleagues showed in our article; the points raised by them are very relevant.

We agree that 90% compliance as reported in our study is based only on the patients' version. Anti-gliadin IgA antibody testing was not done on follow-up, though this would have given more objective evidence.

Vitamin B12 levels at entry and on follow-up were not available in all patients; so they were not analyzed.

Finally, anti-endomysial antibody testing was done in 2 patients and anti-transglutaminase antibody in 44. We apologize for the typographical error.