Celiac Disease in Chennai

Celiac disease (CD), a permanent gluten intolerance is basically a T lymphocytic mediated small intestinal enteropathy induced by gluten in individuals with a genetic predisposition(1). This has been well documented from North India where wheat is the staple diet(2,3). However, there are no documented reports from Tamilnadu. We are reporting 3 cases seen in Chennai as shown in *Table I* with characteristics features of CD who responded well to gluten free diet (GFD). An interesting information obtained on history was that though the families of these 3 children were living in Chennai for several generations, their ancestral descent was from other states like Rajasthan and Andhra Pradesh. Travel and migration is now part of our life and one should consider CD especially in the migrant population from North India. Early recognition of this problem and avoiding gluten in these children will definitely help in decreasing the morbidity associated with this disease.

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TABLE I–*Clinical Profile of Cases.*

	Case 1	Case 2	Case 3
Age & sex	6 yrs, Male	7 yrs, Male	11yrs, Male
Age of onset of symptoms	4.5 yrs	5 yrs	5 yrs
Clinical presentation	Recurrent diarrhea, growth failure	Chronic diarrhea, pallor	Growth failure, Recurrent respiratory illness, recurrent. diarrhea.
Physical findings			
Height in cms	105 (exp113)	107 (exp119)	110 (exp143)
Weight in kg	15 (exp 20)	16.5 (exp 22)	15 (exp 30)
Pallor	–	+	+
Upper GI endoscopy	Normal	Scalloping of duodenal mucosa	Scalloping of duodenal mucosa
HPE- duodenal biopsy			
Villous atrophy	Subtotal	Severe	Severe
Crypt hyperplasia	+	+	+
↑lamina propria cellularity	+	+	+
Parasites	–	–	–
Serology EMA	+	+	Not done
AGA	↑	Not significant	+
tTGA	↑	↑	Not done
Response to Gluten Free Diet			
Symptomatic improvement	Marked	Marked	Marked
Improvement in Ht and Wt	3 cm & 2 kg in 5 months	2 cm & 2 kg in 5 months	3 cm & 2 kg in 4 months

EMA—Endomysial ntibody, AGA—Anti Gliadin antibody, tTGA—tissue transglutaminase antibody.

REFERENCES

1. Shamir R. Advances in celiac disease. Gastroenterol Clin North Am 2003; 32: 931-947.
2. Yachha S K, Misra S, Malik A K, Nagi B, Mehta S K. Spectrum of malabsorption syndrome in north indian children. Indian J Gastroenterol 1993; 12: 120-125.
3. Patwari A K, Anand V K, Kapur G, Narayanan S. Clinical and nutritional profile of children with celiac disease. Indian Pediatr 2003; 40: 337-342.

Wilson's Disease Presenting as Depressive Disorder

A 11-year-old girl was admitted with abnormal movements involving the left side of the body. Her symptoms started 9 months back when her father died. She became moody, irritable and lost interest in play and activities. She developed excessive fears, crying spells and disturbed sleep. She had loss of appetite and started losing weight. Her handwriting and academic performance deteriorated. The symptoms gradually increased and she was taken to a psychiatrist. A diagnosis of reactive depression was made and she was put on fluoxetine and thioridazine. About 4 months later she developed the abnormal movements and was admitted to our hospital.

She was born of non-consanguineous parents. She had normal developmental milestones and average academic performance before the illness. There was no past history of jaundice and no family history of any significant illness. On examination the child was conscious with occasional jerky movements of limbs. Her cranial nerves and optic fundi were normal. There was hypertonia involving all four limbs, slurred speech and ataxic gait. Deep tendon and plantar reflexes were normal with no sensory or cerebellar signs. Mental status examination revealed depressive features like depressed mood, prolonged reaction time, crying spells and ideas of hopelessness and worthlessness. There were no psychotic symptoms and her primary mental functions were intact. A diagnosis of major depressive disorder as per the DSM IV diagnostic criteria(1) and thioridazine-induced extra pyramidal symptoms was made.

The extrapyramidal symptoms persisted even after stopping thioridazine. Hence the possibility of organic extrapyramidal lesions was considered. Ophthalmology examination revealed bilateral KF rings, which pointed to the diagnosis of Wilson's disease. Investigations were also suggestive of Wilson's disease(2,3). Her serum ceruloplasmin level was very low and serum copper normal. Her complete blood count, ESR and peripheral smear were normal. Urine copper estimation could not be done due to technical reasons. Ultrasound scan showed increased echotexture of liver with no enlargement. The liver function tests were normal. CT scan head showed mild cerebral atrophy and hypo dense lesions involving the brain stem and putamen.

She was advised copper free diet and started on zinc 200 mg daily initially,