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# CASE REPORT

# ATYPICAL CELIAC DISEASE AND TYPE 1 DIABETES MELLITUS: CASE REPORT

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**ABSTRACT**

Celiac disease (CD) is an immune-mediated multisystem disorder seen in genetically susceptible individuals triggered by gluten in wheat, rye and barley.[1] Celiac disease occurs more frequently in type 1diabetes than in the general population, with estimates varying from 2.4%10 to 16.4%. Between 3% and 6% of patients with type 1 *diabetes mellitus* have (atypical) CD.[2] We present a case of 27 year female who presented to us with diabetic ketoacidosis and was later diagnosed with atypical celiac disease.

## INTRODUCTION

Celiac disease (CD) is an immune-mediated multisystem disorder seen in genetically susceptible individuals triggered by gluten in wheat, rye and barley. The coexistence of type 1 diabetes mellitus (T1DM) and CD is due to intricate interaction between the environmental factors and genetic susceptibility. There is evidence of common genetic basis for disease expression as both the diseases are associated with the major histocompatibility complex class II antigen DQ2 encoded by the alleles, DQA1∗501 and DQB1∗201 and seven shared nonhuman leucocyte antigen (HLA) loci.[1] Celiac disease occurs more frequently in type 1 diabetes than in the general population, with estimates varying from 2.4%10 to 16.4%[2]

## CASE REPORT

We report a case of 27 year married female who presented to us in emergency with chief complaint of vomiting for last 5 days, and altered sensorium for 1 day. She was a known case of diabetes mellitus and was non complaint to treatment. On examination her bp was 122/76 mm of hg, pulse rate was 72, and RBS was high. She was married for 3 year and was having secondary ammenorhea for last 1 year. Her UPT was done which was negative. On clinical examination she had anemia. Chest and CVS examination was within normal limit. On abdominal examination there was no organomegaly. Her ABG was sent which was suggestive of metabolic acidosis and she was started on management for DKA with iv fluid and insulin infusion. On day 3 her sensorium improved and her sugar were under control. Her workup for anemia, diabetes and secondary amennorhea was sent.

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**Table 1.**

| | | | |
|---|---|---|---|
| Hemoglobin | 8g/dl | P/S | Microcytic hypochromic |
| Tlc | 12,600thou / ul | SGOT | 35 U/L |
| Platelets | 303 thou/ul | SGPT | 25 U/L |
| MCV | 85.6 | ALK PO4 | 79 U/L |
| MCHC | 33 | HBA1C | 16 |
| MCH | 28.2 | C peptide | <0.05 |
| CREAT | 0.7 | TSH | 2.07 |
| UREA | 14 | HIV, HBSAG, anti HCV | Negative |

**Table 2.**

| | |
|---|---|
| LH : 0.6 (1.5-9.3) | FERRITIN : 12( 4.63-204) |
| FSH : 4.2 (1.5-168.8) | |
| PROLACTIN :6.83( 5.18- 26.53) | |
| IG A TTG : 108.37 (> 18.0 U/ML) | |
| USG PELVIS : NORMAL | |
| IRON : 24 ( 60-180 ug/dl) | |
| TIBC: 435( 250-450 ug/dl) | |
| SATURATION : 4 ( 20-50%) | |

Her upper gi endoscopy was planned in view of iron deficiency for D2 biopsy and she was discharged keeping possibility: type 1 diabetes with diabetic ketoacidosis with anemia (iron deficiency anemia) and secondary amennorhea. Her D2 biopsy report show villous crypt ratio : 2:1. Moderate crypt hyperplasia. lamina propria shows infiltration by lympho mononuclear infiltrates admixed with eosinophill. Intraepithelial lymphocytes < 30 /100 epithelial cells. Since she does not had the classical symptoms of celiac disease such as diarrhea, weight loss, gluten sensitivity but had iron deficiency anemia with hypothalamic amennorhea and since her IGA TTG was positive along with d2 biopsy features

suggestive of celiac. She was diagnosed with atypical celiac diseae.

## DISCUSSION

Celiac disease (CD) is an immune-mediated multisystem disorder seen in genetically susceptible individuals triggered by gluten in wheat, rye and barley. The coexistence of type 1 diabetes mellitus (T1DM) and CD is due to intricate interaction between the environmental factors and genetic susceptibility. There is evidence of common genetic basis for disease expression as both the diseases are associated with the major histocompatibility complex class II antigen DQ2 encoded by the alleles, DQA1∗501 and DQB1∗201 and seven shared nonhuman leucocyte antigen (HLA) loci.[1] Approximately half of all CD patients have no overt gastrointestinal symptoms and many are asymptomatic, which may explain the underestimated epidemiologic data in the literature. Between 3% and 6% of patients with type 1 *diabetes mellitus* have (atypical) CD.[3,4] Celiac disease has been considered an "iceberg disease". Some have the classical symptoms related to nutrient malabsorbtion, diarrhea. Some have atypical celiac disease with manifestations that are not related to intestinal malabsorbtion (eg: anaemia, osteopenia, amenorhea and neurological symptoms. large number of patients have silent celiac disease. Celiac is diagnosed with the help of small intestinal biopsy in a patient who has symptoms of nutrient malabsorbtion as well as positive IgA tTg antibody test. The sequence of appearance of T1DM and CD cannot be predicted. A large proportion of CD cases are diagnosed within two years of T1DM and majority within 10 yr of screening in paediatric setting; however, the diagnosis can be made beyond this period. The diagnosis of T1DM usually precedes CD [6] though the order can also be reversed[7.] Since celiac disease has wide presentations and association we should screen patients for celiac disease, it being considered as an "iceberg "disease. Celaic is associated with T1DM, Down's syndrome, Turner's syndrome, dermatitis herpetiformis.

**Conclusion**

The coexistence of type 1 diabetes mellitus (T1DM) and CD is due to intricate interaction between the environmental factors and genetic susceptibility. Since celiac disease has wide presentations and association we should screen patients for celiac disease, it being considered as an "iceberg "disease.

**Conflict of Interest:** None.

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