The association between primary hypoparathyroidism and celiac disease (CD) is uncommon in paediatrics, even more if they are not part of an autoimmune polyglandular syndrome (APS, almost exclusively type II). We describe a case of autoimmune hypoparathyroidism coexisting with celiac malabsorption.

CASE REPORT

Valentina was a 7 year old female child when she was admitted in hospital because she had generalized seizures at home. She had normal weight and height, no dysmorphism; she had laryngospasm and Trousseau’s sign. No familiar history for tetania; no personal history of recurrent infections or mucocutaneous candidiasis. First blood exams: hypocalcemia (4.4 mg/dl) and low ionised calcium; hyperphosphatemia (9.1 mg/dl); Magnesium 1.6 mg/dl (n.v. 1.7-2.4). ECG: prolonged QTc. PTH not dosable, hypocalciuria, normal phosphaturia, normal alcaline phosphatase, 25OHD3 22 ng/ml (n.v. 30-100). She had hypocalcemia secondary to hypoPTH.

We administered her intravenous calcium gluconate and oral 25OHD3: the clinical signs regressed within some hours, but, when we tried to give the child oral calcium, calcemia went down again. In the next days we had a possible explanation to this problem: antibodies for celiac disease were positive! The duodenal biopsie was made: she had a celiac disease (HLA DQ 2 and DQ 8 were positive) and a duodenitis caused by helicobacter pylorii. CATCH22 and AIRE mutations were absent, cariotype was 46,XX, the anti-Calcium Sensing Receptor (CaSR) Antibody was positive

RESULTS

At 12 months from the diagnosis, the CD antibodies were negative, but PTH was still undosable. We could exclude the role of CD antibodies in the hypoparathyrodism origin. Her actual therapy is oral calcium and D vitamin.

CONCLUSIONS

Autoimmune hypoparathyroidism is an unusual association with celiac disease outside of an APS, We think that our child needs an adequate follow up to discover precociously other immune mediate disorders.