**SUPPLEMENTARY FILE 1: ADDITIONAL METHODS**

**Quantification of GIP in stool samples**

The concentration of f-GIP was measured by sandwich enzyme-linked immunosorbent assay (ELISA) using the iVYDAL In Vitro Diagnostics iVYLISA GIP-Stool kit (Biomedal S.L., Seville, Spain).1 Briefly, stool samples were incubated for 60 minutes at 50° C with gentle agitation in 5 mL of Extraction Solution per gram of stool to release the GIP from the stool matrix. After extraction, samples were diluted 1:10 and incubated for 60 minutes in the provided microtiter plate coated with G12, together with the standards and the assay’s positive and negative controls. Wells were then washed and samples incubated with horseradish peroxidase-conjugated G12 antibody for another 60 minutes. Subsequently, plates were washed again and incubated with the horseradish peroxidase substrate. Colour development was stopped with sulphuric acid and absorbance was measured at 450 nm using a UVM340 microplate reader (Asys Hitech GmbH, Eugendorf, Austria). The results were expressed as μg GIP per gram of faeces. Each sample was run in duplicate and at least two different aliquots of each sample were tested on different days. The validity of this method for detecting gluten-free diet transgressions was determined by its analytical sensitivity (limit of quantification 0.08 μg GIP per gram of feces) and diagnostic sensitivity and specificity (98.5% and 100%, respectively).

**Coeliac serology**

Serum IgA anti-tTG2 (or IgG anti-tTG2 in IgA deficient patients) was analysed using a quantitative automated ELISA (Elia CelikeyTM, Phadia AB, Freiburg, Germany) with recombinant human tTG2 as antigen (positive values >2 U/mL).2 Anti-tTG2 at low titres was considered to be positive only if confirmed by positive EmA. EmA was performed by indirect immunofluorescence assay in serum samples at 1:5 dilution (commercial sections of monkey distal oesophagus; BioMedical Diagnostics, Marne-la-Vallée, France) in all patients with positive tTG2. Total serum IgA was measured using rate nephelometry (BN II, Siemens Healthcare Diagnostics SL, Marburg, Germany).

**Coeliac genetics**

Genomic DNA from whole blood was purified using the commercial Qiamp DNA Blood Mini kit (Qiagen, Düsseldorf, Germany). A commercial reverse hybridization kit for the detection of CD heterodimers HLA-DQ2.5 (A1\*0501/\*0505, B1\*0201/\*0202) and HLA-DQ8 (A1\*0301, B1\*0302) was used (GenID, GMBH, Strasburg, Germany).

**Histological studies**

Two endoscopic biopsies from the bulb and four from the second portion of the duodenum were obtained using a one-bite-per-pass technique with 2.8 mm biopsy forceps (Radial JawTM 4, Boston Scientific, USA) and were placed in separate vials for standard histological studies. Diagnostic and follow-up tissue slides were blindly evaluated by two expert pathologists and a consensus was reached in case of disagreement. Histopathological changes were classified according to the modified Marsh-Oberhuber classification.3,4 Lymphocytic enteropathy (Marsh type 1 lesion) was defined as 25 or more intraepithelial lymphocytes per 100 epithelial nuclei in samples stained with anti-CD3, and a normal villous architecture. The degree of concordance between the two pathologists was analyzed. Mucosal recovery was defined as the absence of villous atrophy at the follow-up biopsy (i.e., Marsh 0-2).

Special attention was paid to patients who did not show any signs of histological recovery after two years on dietary treatment.

**References**

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2. Mariné M, Fernández-Bañares F, Alsina M, et al. Impact of mass screening for gluten-sensitive enteropathy in working population. World J Gastroenterol 2009;15:1331-8.
3. Marsh MN. Gluten, major histocompatibility complex, and the small intestine. A molecular and immunobiologic approach to the spectrum of gluten sensitivity ('celiac sprue'). Gastroenterology 1992;102:330-54.
4. Oberhuber G, Granditsch G, Vogelsang H. The histopathology of celiac disease: time for a standardized report scheme for pathologists. Eur J Gastroenterol Hepatol 1999;11:1185-94.