Adequacy of Non-Invasive Investigations for Coeliac Disease

Corresponding Author:
Mr. Abhijeet V Patil,
Senior Clinical Fellow, Department of General Surgery, The Great Western Hospital, SN3 6BB - United Kingdom

Submitting Author:
Mr. James M Williamson,
Speciality Training Registrar, Department of Surgery, Gloucestershire Royal Hospital, Great Western road, GL1 3NN - United Kingdom

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Author(s): Patil A V, Williamson J M, Burgess P

Abstract

Introduction
Coeliac disease can be an elusive diagnosis, partly due non-specific symptoms and the lack of unequivocal non-invasive investigations. Histology is the gold standard of diagnosis. The aim of this investigation was to assess if coeliac disease could be diagnosed using non-invasive investigations.

Method
The non-invasive investigations performed on all new, histologically proven, Coeliac's over a 3-year period were assessed.

Results
74 patients were identified; positive non-invasive investigations were: 45 (of 62) for ferritin (73%), 43 (of 62) for tTG (69%) and 5 (of 22) for radiology (23%). When both serum ferritin and tTG levels were recorded the sensitivity increased to 46 out of 54 (85%), when all three investigations were utilised the sensitivity was 13 out of 16 (81%)

Conclusions
At best the sensitivity of non-invasive investigation is 85% and thus histological diagnosis remains the gold standard for diagnosis. We would advise an increased use of endoscopic assessment.

Introduction
Coeliac disease is an inflammatory small bowel disorder characterised by severe villous atrophy, malabsorption, and malignancy. The gluten proteins of wheat, barley, and rye trigger are thought to trigger this disease. Recent studies place its prevalence between 1:300 and 1:100 (for Western populations). Patients with coeliac disease express the antigen-presenting molecules human leukocyte antigen-DQ2 (HLA-DQ2) and/or HLA-DQ8, which bind gluten peptides and thus activate the destructive intestinal T-cells. Untreated patients have circulating IgA auto antibodies to the enzyme tissue transglutaminase (tTG), a component of endomysium. Testing for serum IgA tTG has a high positive predictive value, although its sensitivity can be as low as 90%-6-8. The availability of IgA tTG and other serological markers - Endomysial antibody (IgA EMA) and Antigliadin antibody (IgA and IgG) - has dramatically facilitated the diagnosis of coeliac disease. Despite these immunological markers, histological identification of gluten sensitive enteropathy is the accepted basis for diagnosing coeliac disease. The classical characteristic inflammatory small bowel lesion is villous atrophy, with the presence of lymphocytes. In some patients, however, a less florid lesion is present, and this may cause diagnostic uncertainty. Unequivocal evidence that the lesion is sensitive to the withdrawal of gluten may require three biopsies; the first before treatment, the second after withdrawal of gluten and the third after challenge with gluten. This series of biopsies is rarely performed, and a single initial biopsy is usually sufficient.

Patients typically present with abdominal distension, alterations in bowel habit and malabsorption, but many have few gastro-intestinal symptoms. Coeliac disease may be an elusive diagnosis and extra-intestinal manifestations, such as osteoporosis, infertility and neurological disturbances, or iron and folate deficiency anaemia may be the only presentations. The most significant symptoms of malabsorption are diarrhoea, weight loss, meteorism, abdominal pain, vomiting and asthenia. A high level of suspicion is required in identifying the large number of undiagnosed patients that exist. A gluten-free diet is therapeutic for most patients and should prevent disease complications – dermatitis herpetiformis, small bowel lymphoma and adenocarcinoma, ulcerative jejuno-ileitis and microscopic colitis.

Infancy (Diarrhoea, abdominal distension, failure to thrive, anorexia, vomiting, psychomotor impairment (muscle wasting)
Childhood (Diarrhoea or constipation, anaemia, loss of appetite (short stature, osteoporosis)
Adulthood (Diarrhoea or constipation, anaemia, aphthous ulcers, glossitis, stomatitis, dyspepsia, abdominal pain, bloating, weight loss, fatigue, infertility, neuropsychiatric symptoms (anxiety, depression), osteoporosis, weakness (myopathy, neuropathy).

Box 1: Typical symptoms and signs of Coeliac disease depending on age.
Methods

Newly diagnosed, histologically proven, coeliac patients were retrospectively analysed over a 3-year period. Patient demographics and pre-endoscopic investigations were assessed. Non-invasive investigations were grouped into: anaemia, serum ferritin, serum IgA tTG and radiological small bowel series.

Results

Over the 3-year period, 74 newly diagnosed patients were identified. The population covered by our hospital is 300,000, thus our incidence is 1:1350. There was a slight female predominance (43F, 35M) with a male to female ratio of 1:1.24. The patients ranged from 17 to 86 years, with a mean age of 52 years old (figure 1). The majority of newly diagnosed patients were between 25 and 50 years of age (n= 36, 48%), followed by the 50 – 70 year group (n=26, 35%). 2 patients (3%) were below 25 years of age and 10 (14%) were aged over 70. The index of clinical suspicion, prior to diagnosis, was highest in the 25-50 years age group (n=25, 70%), followed by the 50-70 years group (n=13, 50%). One patient in the over 70 group (10%) and neither of the under 25 group were thought to have coeliac disease.

42 patients (57%) were anaemic; however, 31 (42%) patients had normal haemoglobin and 1 (1%) patient did not have a pre-diagnostic level taken (figure 2). Ferritin was low in 45 patients (61%), although it was normal in 17 (23%) patients and not requested in 12 (16%) patients (figure 3). Serum antibodies to tissue transglutaminase (tTG) were elevated in 43 patients (58%), but normal in 20 (27%) patients and no results were available for 11 patients (15%) (figure 4).

Small bowel radiological studies were carried out in only 22 of the patients (30%). Of these patients, 5 (23%) had findings suggestive of coeliac disease (figures 5 and 6).

Histological features in all patients showed duodenal mucosal villous atrophy, with or without features of chronic inflammation. These characteristic changes are diagnostic of coeliac disease. Sample histology is displayed in figures 7 and 8.

References

Illustrations

Illustration 1

Figure 1: Age Distribution

Illustration 2

Figure 2: Haemoglobin
Illustration 3

Figure 3: Serum Ferritin

Illustration 4

Figure 4: Antibodies to tissue transglutaminase
Illustration 5

Figure 5: Small Bowel Radiology

Illustration 6

Figure 6: Diagnostic Yield of Small Bowel Radiology
Illustration 7

Figure 7: Duodenal mucosa demonstrating loss of the normal villous architecture (flattening), with crypt hyperplasia and intra-epithelial lymphocytosis (x400 magnification)

Illustration 8

Figure 8: Duodenal mucosa showing intra-epithelial lymphocytosis (x200 magnification)
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