It is probably an undisputed fact that diabetes does reach the body parts that most other conditions do not, and the gastrointestinal (GI) tract is no exception. The association between diabetes and the GI tract is manifested at several aetiological levels: cause and effect (e.g. pancreatic damage, diabetic angiopathy and autonomic neuropathy), association (e.g. coeliac and thyrotoxicosis), side effects of therapeutic interventions (e.g. metformin, GLP-1 analogues, and bariatric surgery) or a mixture of those facets.

The paper by Cummings et al. in this issue of *Practical Diabetes* serves as a timely reminder of the close link between diabetes and abdominal complaints, with the particular spotlight on pancreatic exocrine insufficiency (PEI). The authors share their experience of introducing routine questioning about abdominal symptoms into the diabetes consultation, and their targeted screening of PEI using faecal elastase 1 (FE1) concentration.

To advocate the routine introduction of the above noble approach into the regular diabetes consultation requires further validation on logistical, evidence-based and cost-effectiveness grounds. Evidence is gathering on the high prevalence of PEI in diabetes, with reports putting this at 30–50% in type 1 and 20–35% in type 2 of some degree. Evidence regarding the impact of PEI on glucose metabolism and the efficacy of pancreatic enzyme replacement therapy (PERT) on abdominal symptoms and diabetes control is, however, less certain and is based on a small number of trial participants.

Such scarcity is apparent in the strength of evidence in the Australasian guidelines on treatment of PEI in diabetes, assigning low evidence for their recommendations for investigating it (level of 2b representing low quality randomised trials) and that for trialling PERT (level 5 based on expert opinion). Moreover, PERT can be associated with GI adverse effects such as diarrhoea, particularly at high doses, and requires the co-administration of proton pump inhibitors to enhance efficacy, resulting in further polypharmacy. Using FE1 as a diagnostic tool for PEI is emerging as the preferred non-invasive indirect method, and moving away from the unpopular three-day faecal fat ‘gold standard’ test. It is, however, only reliable in moderate to severe PEI and can be falsely low in patients with diarrhoea. FE1 testing also remains unpleasant for patients (one-third of patients offered FE1 testing did not provide a stool sample in Cummings et al.’s cohort). The test (circa £30) is comparatively cheap; however, when used more widely it would result in significant cost pressures.

While accepting the expanding remit of the diabetic consultation, which progressively shifted away from glucocentricity, the scope for an ad libitum venture into the GI tract requires further illumination. Maybe our gastroenterology colleagues could show us the way.

**Dr Ma’en Al-Mrayat, FRCP, Consultant in Diabetes & Endocrinology, St Mary’s, Isle of Wight, UK**

**References**


