previously, with variable results.[1-6] We present our data on such an association in a largely in-bred population of about 400,000 persons residing in the Maltese Islands in the Mediterranean Sea.

Patients previously diagnosed to have CD (based on serological tests and duodenal biopsy) and attending a medical out-patient clinic answered a questionnaire designed to determine whether they had previously been diagnosed to have asthma or allergic rhinitis. They were also asked about symptoms suggestive of asthma; patients with such symptoms but no prior diagnosis of asthma underwent physiological lung tests to look for undiagnosed asthma. All patients provided informed consent. The frequency of asthma and allergic rhinitis in CD patients was compared with data from the International Study of Asthma and other Allergic Conditions in Childhood in the Maltese Islands (ISAAC1997), using *chi*-squared analysis.

All 86 patients (age range 16-69 [median 43] years; 65 female) answered the questionnaire about CD and asthma. They constituted 21% of the 409 patients with CD in the Maltese islands included in a register kept for controlling free prescription of gluten-free foods.

Of 86 respondents, 24 (27.8%; 21 female) had asthma, including 22 with known asthma and 2 with previously undiagnosed asthma; the frequency of asthma in CD patients was higher than that reported in the general Maltese population (11.1%; $p<0.00005$).[7] In addition, four non-asthmatic patients (one smoker, two ex-smokers, one non-smoker) reported wheezing in the absence of respiratory tract infection in the past; they however had normal pulmonary function tests. Another woman with CD gave history of wheezing and cough after exercise. She was a non-smoker, had family history of asthma, and her lung function tests showed 10% reversibility in FEV1 after the administration of bronchodilator. Another patient had nocturnal cough; he was an ex-smoker, had family history of asthma, and had normal lung function tests.

In 16 patients, asthma preceded CD by 3 months to 39 years (median 20 years). Among these patients, gluten-free diet had led to improvement in asthma in 6 patients, possible improvement in 2 patients, and no change in 8 patients. In the remaining 8 patients, asthma followed CD by 2 to 14 years (median 8). Thirty-one of 86 patients with CD and 11 of 24 patients with CD and asthma gave family history of asthma among first-degree relatives.

Eighty-two patients (62 female) answered the questionnaire about allergic rhinitis. Of these, 36 (44%) suffered from allergic rhinitis; this frequency was higher than that reported in the general Maltese population (32.3 %; $p<0.05$).[7]

Our findings suggest that asthma and allergic rhinitis are more common in CD patients than in the general population in Malta. In patients with atopic diseases, index of suspicion for CD should be high.

Pierre Ellul, Mario Vassallo, Stephen Montefort

St. Luke's Hospital, Malta

References

1. Zauli D, Grassi A, Granito A, Foderaro S, De Franceschi L, Ballardini G, *et al.* Prevalence of silent coeliac disease in atopics. *Dig Liver Dis* 2000;32:780-1.
2. Collin P, Reunala T, Pukkala E,Laippala P, Keyrilainen O, Pasternack A. Coeliac disease: associated disorders and survival. *Gut* 1994;35:1215-8.
3. Bottaro G, Cataldo F, Rotolo N, Spina M, Corazza GR. The clinical pattern of subclinical/silent celiac disease: an analysis on 1026 consecutive cases. *Am J Gastroenterol* 1999;94:691-6.
4. Greco L, De Seta L, D'Adamo G, Baldassarre C, Mayer M, Siani P, *et al*. Atopy and coeliac disease: bias or true relation? *Acta Paediatr Scand* 1990;79:670-4.
5. Hodgson HJF, Davies RJ, Gent AE, Hodson ME. Atopic disorders and adult coeliac disease. *Lancet* 1976;i:115-7.
6. Tarlo SM, Broder I, Prokipchuk EJ, Peress L, Mintz S. Association between celiac disease and lung disease. *Chest* 1981;80:715-8.
7. Montefort S,Lenicker HM, Caruana S, Agius Muscat H. Asthma and other allergic conditions in childhood in the Maltese Islands. *ISAAC* 1997.

Correspondence to: Dr Ellul, 29 Heapfold, Norden, Rochdale, Lancashire, OL 12 7NR, England. E-mail: pierre_ellul@yahoo.co.uk

Pancreatic pseudocysts in brothers: familial, environmental or incidental?

A 42-year-old non-alcoholic man presented with upper abdominal pain and gradually increasing mass of 2 years and 6 months duration. There was no past history of acute pancreatitis, trauma, gallstone disease, jaundice or tuberculosis. Examination revealed a 6 cm x 7 cm ill-defined intra-abdominal mass in the epigastrium. The rest of the examination was unremarkable. Laboratory investigations and plain radiographs of the chest and abdomen were normal. Ultrasonography revealed a thick-walled cystic mass, 6 cm x 6 cm, in the lesser sac with internal echoes and echogenic debris. The head and body of the pancreas was irregular and appeared shrunken. A diagnosis of chronic pancreatitis with pancreatic pseudocyst was made. He underwent laparoscopic cystogastrostomy and made satisfactory recovery.

One year later, when he came for a routine check up (still well), he brought his younger brother (aged 36 years) who lived with him and worked with him in the fields. The brother had complaints of upper abdominal pain of 6 months' duration. He was also not a consumer of alcohol and there was no significant past medical or surgical history. General examination was unremarkable and there was no palpable mass in the abdomen. Ultrasonopraphy revealed a 4 cm x 3 cm cystic mass in the lesser sac with internal echoes suggestive of pancreatic pseudocyst. The pancreas appeared normal. This patient also underwent laparoscopic cystogastrostomy. A year after this both brothers are well and symptom free.

Both our patients had no past history of pancreatitis, were not consumers of alcohol or known to have gallstone disease. Additionally they had adequate nutritional intake and there was no history of trauma. The similar presentations and identical pathologies suggest a common etiology. They were farmers who lived and worked together, and although an environmental (exposure) factor cannot be ruled out, we could not identify any. Occurring in brothers, there is a possibility of this having a familial or hereditary etiology.[1] Pancreatic pseudocysts without antecedent symptomatic pancreatitis, to our knowledge, have not been described to occur in siblings.

A Dildeep Pillai, Ashish Choudhrie, Naveen Eipe*

Departments of Surgery and *Anesthesia, Padhar hospital, Padhar, Betul, Madhya Pradesh 460 005

Reference

1. Toskes PP, Greenberger NJ. *Disorders of the Pancreas. In:* Braunwald E, Fauci AS, Kasper DL, Hauser SL, Longo Dl, Jameson JL, Eds. Harrison's Principles of Internal Medicine, 15th ed. New York: McGraw Hill. 2003: p. 1788-804.

Correspondence to: Dr Eipe. Fax: (7141) 26 3346. E-mail: neipe@yahoo.com

Small bowel volvulus around feeding jejunostomy tube

Placement of tube or needle catheter jejunostomy as an adjunct to major upper gastrointestinal surgery and following upper gastrointestinal corrosive acid injury is now widely accepted.[1] Minor functional disturbances associated with feeding jejunostomy are well understood and are acknowledged in most studies.[2] However, major complications requiring emergency re-laparotomy related to jejunostomy feeding tube are rare.

A 19-year-old girl had a surgically placed feeding jejunostomy tube for corrosive stricture of esophagus and stomach antrum following acid ingestion. She underwent antrectomy with Billroth I gastro-duodenal anastamosis three months following corrosive injury, and was on regular antegrade endoscopic esophageal dilatation using Savary-Gilliard dilators.

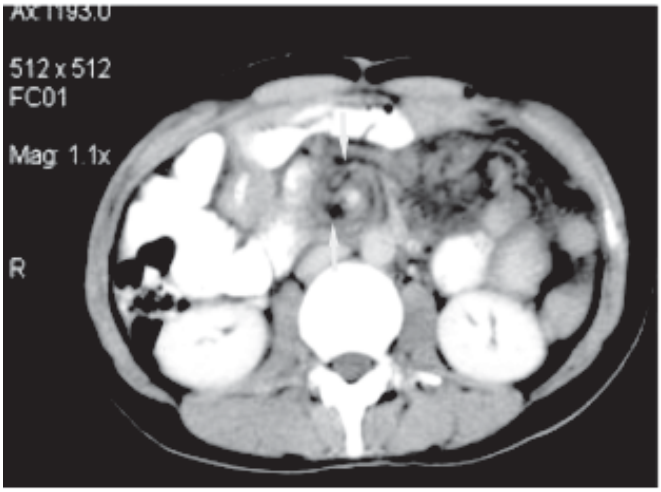

Fig. : Contrast - enhanced CT scan showing "whirlpool sign"

After 9 months, she was admitted with acute severe abdominal pain. On examination, abdomen was not distended. There were no clinical signs of peritonitis. X-ray abdomen was normal. Contrast-enhancement CT scan (Fig) showed jejunostomy feeding tube *in situ*, with a "whirlpool sign",[3] i.e., convergence of mesenteric vessels toward the twisted site in the small bowel mesentery, with mesenteric edema and no free fluid in the abdomen, suggesting small bowel volvulus with impending vascular compromise with no intraperitoneal leak.

At emergency re-laparotomy the jejunal loops were twisted around the jejunostomy tube fixation site on the abdominal wall, resulting in small bowel volvulus with mesenteric edema and bowel ischemia with no peritoneal contamination. Untwisting of the small bowel was done, which resulted in a pink viable small bowel with good mesenteric pulsation. The previous jejunostomy site was dismantled and closed with 3/0 vicryl-interrupted sutures. The patient had an uneventful recovery.

Despite the advantages of feeding jejunostomy, serious complications do occur and can be life-threatening. In a large study, intestinal occlusion and volvulus occurred in 0.14% of all needle catheter jejunostomy applications.[4] Zapas *et al*[5] reported that routine insertion of feeding jejunostomy as an adjunct to major upper abdominal procedures may not be justified, as its benefit-risk ratio was low.

Small bowel volvulus at the anchored site of jejunostomy tube can be prevented by broad-based fixation (6-10 cm) of the jejunal loop to the parietal peritoneum of the anterior abdominal wall using three or four 3/0 silk sutures.

A Sivasankar, M Johnson, S Jeswanth, S Rajendran, R Surendran

Department of Surgical Gastroenterology, Stanley Medical College Hospital, Chennai