An unusual cause for anaemia

To the Editor,

A 69-year-old lady presented with iron deficiency anaemia and a past history of hysterectomy and gastritis. She reported lethargy and tiredness as well as hot flushes associated with night sweats. She also admitted to 6kg of weight loss and complained of rather non-specific abdominal pains. She had enjoyed frequent holiday breaks abroad and she had recently returned from a walking excursion in Italy.

She was clinically euthyroid and her abdominal examination revealed mild tenderness over the left iliac fossa and a palpable but smooth liver edge. Her haemoglobin was 9.7g/dl with microcytosis and she had concomitant leukopenia, neutropenia and lymphopenia. Her platelet count was low at 126,000/dl. Initial C-reactive protein was raised and B12 and folate levels were normal. The liver function tests showed normal clotting but reversal of the albumin/globulin fraction (26/52 g/l). Her immunoglobulin estimation revealed a polyclonal raise of IgG and no paraprotein. A battery of autoantibodies was negative. She had normal ECG and chest X-ray while the ultrasound scan showed normal liver, biliary tree, spleen and pancreas. Upper and lower gastrointestinal endoscopies were equally unremarkable.

Few days later, clinically evident splenomegaly was present. The new finding prompted an urgent haematology review for exclusion of lymphoma. Both a repeat ultrasonography and a computed tomography scan showed splenomegaly but failed to detect any significant lymphadenopathy. Eventually, a bone marrow trephine biopsy was performed and confirmed reduced erythropoesis and a significant number of macrophages containing phagocytized micro-organisms. This finding was consistent with Donovan bodies. She received liposomal amphotericin with excellent response.

Anaemia in visceral leishmaniasis may be due splenic sequestration, haemolysis or bone marrow dyserythropoesis [1]. Splenomegaly and fever are sine qua non in symptomatic cases of kala azar (visceral leisdmaniasis) [2]. However, splenomegaly may be absent in the acute or early stages of the disease confusing the diagnostic process. Travel in endemic areas (i.e. India, Kenya, Nepal, Sudan) should be sought but is by no means prerequisite for diagnosis of the infection [2]. Atypical presentations are a rare occurrence, especially in non-endemic areas, but should be borne in mind when investigating anaemia with hypergammaglobulinaemia and splenomegaly.

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Successful treatment of *Clostridium difficile* colitis with intravenous immunoglobulin

To the Editor,

A 57 year-old-female was referred to the gastroenterology department with marked abdominal distension and profuse diarrhoea. She had been an in-patient for 7 months on the orthopaedic ward with an infected hip prosthesis. During her admission she had multiple courses of antibiotics for the infected prosthesis, bronchopneumonia and pressure ulcers. Two months prior to this she had been successfully treated for an episode of *Clostridium difficile* toxin (CdT)-associated diarrhoea with a course of metronidazole. Following this, she had cefuroxime, flucloxacillin and erythromycin for infected pressure sores. Her C-reactive

protein (CRP) was 219 mg/L (normal<16) and the white cell count 28.9x10^9/L (normal 3.8-11.0) with significant neutrophilia (26.5x10^9/L). Albumin was 22 g/L (normal 36-52) and faeces tested positive for CdT (ELISA). The CT scan of abdomen showed evidence of colitis. The antibiotics for her pressure sore were discontinued and she was commenced on metronidazole 400mgs t.d.s. Enteral feeding was initiated but her albumin remained low. Seven days later there was minimal symptomatic improvement despite normalisation of the white cell count and significant improvement of her CRP. A flexible sigmoidoscopy was performed and showed large pseudopolyp-like pseudomembranes (Fig.1). Histology confirmed acute infectious colitis. High dose vancomycin (250mg qds) was administrated together with probiotics and colestyramine. The patient showed modest improvement following a 10 day course of therapy. Unfortunately, the patient developed pyrexia and blood cultures grew Klebsiella pneumoniae. Intravenous Tazocin® was commenced and soon after she developed further diarrhoea and tested positive for CdT (ELISA). A combination of metronidazole and vancomycin was used but despite a 10 day course of therapy her diarrhoea and CRP failed to settle. At this point i.v. immunoglobulin (IVIG) was commenced. We used VIGAM®, in the form of two sets of three infusions one week apart, which was well tolerated by the patient. Twelve days after initiation of the above regime her bowel frequency improved and her CRP returned to normal. She remained well and continued with standard dose vancomycin (125mg qds) for 7 more days.

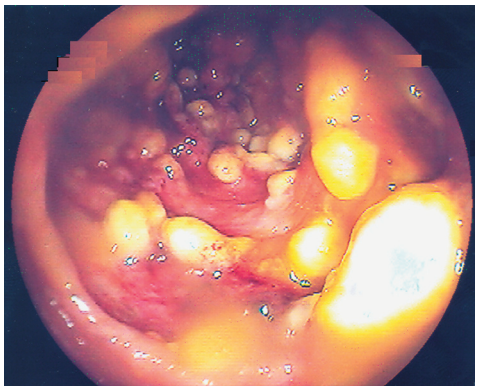

Fig 1. Sigmoidoscopy showing pseudopolyp-like pseudomembranes.

Clostridium difficile toxin-related diarrhoea used to be classically associated with clindamycin administration but now it is commonly seen after exposure to a wide range of other antibiotics. Clostridium difficile is a Gram-positive, anaerobic, spore forming rod and spreads by the faecal-oral route. The prevalence of asymptomatic colonisation in the community is 5% and in elderly in-patients is 20% [1]. Risk factors for developing CdT diarrhoea are: increasing age, comorbidities, long in-hospital stay, multiple antibiotic regimes, gastric acid suppressants and interestingly the presence of a nasogastric tube [2]. Recurrence of CdT diarrhoea will occur in up to one third of cases. It can be difficult to determine if this is due to eradication failure or simply recolonisation. Leung et al. were the first to report the successful use of IVIG in six children with relapsing CdT-induced enterocolitis [3]. Since then, there have been a limited number of case series reporting the beneficial effect of various regimes of IVIG in resistant CdT [1, 4- 8].

In a retrospective study of 14 cases, patients with resistant CdT-induced diarrhoea and a median duration of symptoms of 14 days (range: 3-90 days) were treated with IVIG. The patients received a median of 3 (range: 1-5) courses of vancomycin or metronidazole before IVIG. All tolerated the regime well. Nine responded with bowels normalizing in a median of ten (range: 2-26) days; one patient received two doses [4]. A recently published retrospective analysis however found no difference in colectomy and mortality rates between two matched groups of CdT infected patients treated with IVIG and conventional management [9].

In conclusion, there is growing interest in the use of IVIG therapy for persistent CdT diarrhoea which has failed to respond to traditional therapy. We advocate the use of IVIG as a final step in the treatment algorithm of intractable CdT diarrhoea. In frail, elderly or debilitated patients, where colectomy is not an option, IVIG therapy may prove lifesaving. Further prospective studies are needed.

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Hepatocellular carcinoma manifesting as cavitary lung metastases and leukemoid reaction

To the Editor,

Hepatocellular carcinoma (HCC) is rarely associated with paraneoplastic syndromes such as hypoglycemia, polycythemia, hypercalcemia, porphyria, carcinoid syndrome and watery diarrhea. Leukemoid reaction with cavitary lung metastases is rarely described with HCC [1]. We report the second case published in the medical literature.

A 43-year old male presented with jaundice for four months, abdominal distension and lower limb swelling for two months. Jaundice was fluctuating with no cholestasis. He had a history of alcohol intake of 100-140 g/day for 20 years. There was no history of fever, cough, hemoptysis or chest pain. On examination, he had pallor, icterus, pedal edema, decreased breath sounds in right axillary region, palpable liver 3 cm below xyphosternum and free fluid in the abdomen. Laboratory investigations revealed a hemoglobin of 10.3 g/dl, total leukocyte count of 110,000/mm^3 with a differential count of neutrophils 93%, lymphocytes 3% and monocytes 4% without any band forms, bilirubin 4.7mg/dl, serum aspartate aminotransferase 213 U/L, alanine aminotransferase 85U/L, alkaline phosphatase 719U/L, total protein 5 g/dl with albumin 2.5 g/dl, prothrombin time prolonged by 8.2 seconds and alpha fetoprotein > 300ng/ml. Blood sugar, urea, creatinine, electrolytes, calcium, platelets were within normal limits. Viral and autoimmune markers were negative. Leukocyte alkaline phosphatase level was elevated. Ascitic fluid analysis showed a high serum-ascites albumin gradient with no malignant cytology. Abdominal ultrasound showed coarse echotexture of liver and ascites. Chest X-ray revealed a cavitary lesion in the left lobe and a nodular lesion in the right lobe of lung. Contrast enhanced CT of chest and abdomen showed hypervascular nodular lesions in both lobes of liver, portal vein 16 mm with collaterals in perigastric, perisplenic region, ascites, splenomegaly, two large soft tissue attenuating lesions with cavitation in the right lower lobe and lingula of lung (Fig. 1). An ultrasound guided fine needle aspiration from liver lesions (Fig. 2) was performed and confirmed HCC. Esophagogastroduodenoscopy showed grade II esophageal varices and portal hypertensive gastropathy. Blood cultures, urine cultures and other workup of infective etiology were negative. Interleukin-6 (IL-6) level was elevated at 27 pg/ml (normal 0-12 pg/ml). He was managed with diuretics, intravenous albumin and beta blockers. The patient died after 10 days.

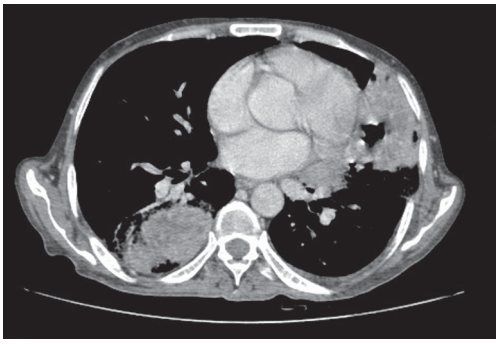

Fig 1. Contrast enhanced chest CT: two large soft tissue attenuating lesions with cavitation in right lower lobe and lingula of the lung.

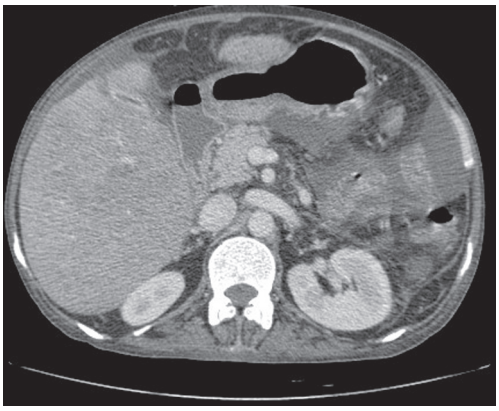

Fig 2. Dual phased contrast enhanced abdominal CT: enlarged liver with altered attenuation and a hypervascular nodular lesions in both lobes, portal vein 16 mm with collaterals in perigastric, perisplenic and mesenteric region, ascites, splenomegaly.

Leukemoid reaction refers to a reactive leukocytosis with cell counts of 30,000-50,000/mm^3. It is described in association with lung, gastrointestinal, genitourinary, ovarian and head and neck cancers [2]. HCC rarely presents with leukemoid reaction [1]. In this case, leukocytosis represented a true leukemoid reaction as suggested by the elevated LAP score and no blast cells.

Granulocytosis has an identifiable non-paraneoplastic etiology such as infection, tumor necrosis or glucocorticoid administration in about 50% of patients; none of these causes were identified in our patient. The other mechanism causing cancer associated leukemoid reactions are due to proteins in urine and serum that stimulate the proliferation of bone marrow cells like granulocyte colony-stimulating factor (G-CSF), granulocyte-macrophage colony stimulating factor (GM-CSF) or IL-6 in tumors of nasopharynx, kidneys and pancreas [3]. In this case, the IL-6 elevation and tumor progression associated with an increase in inflammatory cytokines may have resulted in leucocytosis. Paraneoplastic granulocytosis resolves with the treatment of underlying cancer.

HCC involves the lung in 25 percent of patients reported in autopsy series. Cavitary lung metastases with central

necrosis is more commonly a feature of primary lung tumors [4].

In conclusion, this is the second case of cavitary lung metastases and leukemoid reaction in a hepatocellular carcinoma patient.

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Removal of a metallic dental prosthesis from the duodenum using simultaneously a standard and an ultra-thin endoscope

To the Editor,

Since the first report on the removal of a foreign body with a flexible endoscope by McKechnie et al [1] in 1972, there have been major evolutions in therapeutic endoscopy.

In adults, foreign object ingestion is more common in those with mental impairment [2]. The majority will pass uneventfully through the entire gastrointestinal tract but, sometimes, endoscopic or surgical removal becomes necessary [2]. Impaction and perforation are higher with sharp, pointed metallic objects and so, special considerations and cautions are required with such kind of foreign bodies [2-8].

We report the case of a 25-year-old woman, with Machado-Joseph disease that swallowed, incidentally, her metallic dental prosthesis. The initial x-ray showed that the denture was apparently in the stomach and an urgent endoscopy under sedation took place.

With a standard endoscope (GIF-Q145; Olympus optical Portugal, Lisbon) the prosthesis was identified in the second portion of duodenum (Fig. 1). An alligator-jaws forceps was then used to secure the foreign body and push it in the direction of the stomach. However, the prosthesis' grasps became imbedded in the bulbar and pyloric mucosa and it was not possible to pass the pylorus (Fig. 2) even using a polypectomy snare. Maintaining the prosthesis held in place by the polipectomy snare, a second, ultra-thin endoscope (EG-1840, Pentax Europe GmbH, Hamburg Germany) was then introduced, simultaneously with the standard endoscope. When the ultra-thin endoscope reached the duodenal bulb, it was possible to determine where the metallic prosthesis was incarcerated and, by using the thin endoscope as a hook, it was possible to free the metallic grasps from the mucosa and bring the denture to the stomach (Fig. 3). It was then safely removed using a long overtube. Once removed we saw that the dental prosthesis was approximately 4 cm long and had three sharp grasps in one end and two in the other (Fig. 4). A second, immediate, endoscopic examination revealed only minor lacerations of the bulbar mucosa without signs

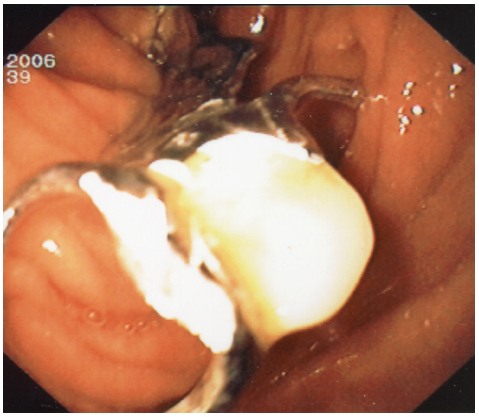

Fig 1. Dental prosthesis in the duodenum.

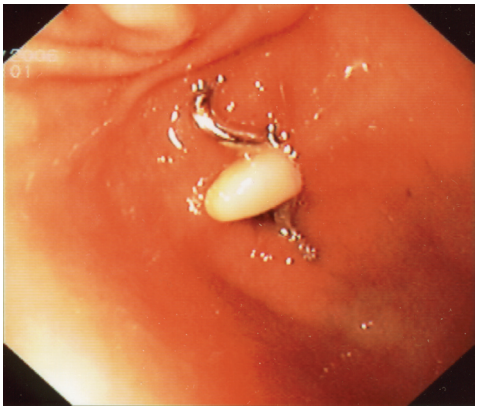

Fig 2. Prosthesis imprisoned in the duodenal bulb and the pylorus.

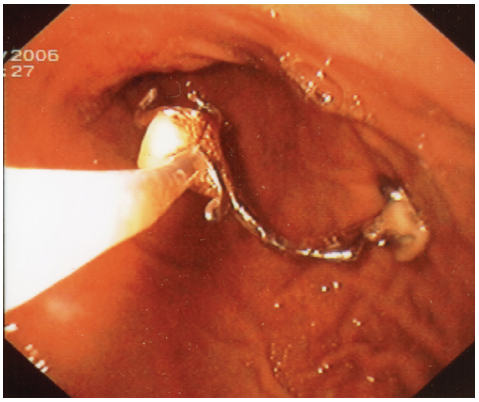

Fig 3. Prosthesis held with a polypectomy snare in the stomach.

Fig 4. Dental prosthesis after removal; it had three metallic grasps in the left end and two in the right one.

of perforation or bleeding.

Foreign body ingestion is frequent and the majority will pass through the gastrointestinal tract uneventfully. Non-operative intervention is necessary in 10-20% and surgery in 1% [2].

Some groups of patients are particularly susceptible to swallowing foreign bodies: children, elderly, mentally disabled/retarded, alcoholics, prisoners, psychiatric [2, 6, 7]. Patients with neurologic disorders such as Machado-Joseph disease are prone to incidental foreign body ingestion, including dental prosthesis.

Endoscopic removal of foreign objects is attractive since it avoids surgery for most of patients, it has reduced costs, is relatively accessible, allows simultaneous diagnosis of other diseases and has a low rate of morbidity [7]. Although safe for the majority of foreign objects, endoscopic removal has increased morbidity, mortality and technical difficulties with sharp-pointed objects [2-8]. Such objects may remain in the stomach or proximal duodenum and, if they pass into the intestine, they may cause a perforation in 15 to 35% of cases [3]. Therefore, endoscopic removal must be attempted in all possible cases.

The majority of prostheses have metal clasps that easily perforate the gastrointestinal wall during endoscopic extraction but, it is also difficult to ensure their natural expulsion without intervention [6]. Its endoscopic removal is challenging and, in fact, they represent the major group of endoscopic failures in removal of foreign bodies with a requirement for surgery, including laparoscopy [7, 8].

The method hereby described might represent an enormous advantage because it allows visualization of how and where the metallic denture is imprisoned, determining if endoscopic removal is still feasible or if the risk of perforation is too high and surgery must be considered. At the same time, the ultra-thin endoscope can serve as an accessory instrument to release the metallic grasps and ease the removal.

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Postprandial alcohol ingestion and gastric motility

To the Editor,

It is well known that alcoholic beverages may induce dyspeptic symptoms [1] and that this effect can be explained by their action on gastric secretion and motility [2]. Overviewing the difficulty to compare data about the effect of alcohol on gastric emptying, one can assume that alcohol inhibits the gastric emptying when administered on a fasting stomach or during the meal [3]. The emptying of the stomach depends on the calorie value of the meal, in physiological and pathological conditions [4], thus the effect of alcohol on stomach motility might be influenced by the contents of the stomach.

We read with interest the original study by Franke et al published in a recent issue of the *Journal of Gastrointestinal and Liver Diseases* [5] regarding the effect on the gastric emptying in healthy volunteers of the so-called „digestif" liqueurs administered in usual gastronomic dose after a standard solid meal. Gastric emptying was estimated with an established ultrasonographic method 9 times in each subject using the same test meal followed by four different liqueurs, in comparison with water, 40% pure ethanol, 70% glucose, coffee and a postprandial treadmill walk. The authors found no change in the gastric emptying with the four tested liqueurs as compared with water, with 40% pure ethanol, 70% glucose or coffee. Only slow postprandial physical

activity increased the speed of gastric emptying. These data lead to the conclusion that these digestifs did not improve the emptying of a full stomach.

The study is a nice contribution to the food culture and to the physiology of the stomach. We would like however to make some comments.

First, we think that the use of digestifs has been spread due to their palatable properties, not due to beliefs in any preventive or healing effect on dyspepsia after meals. Indeed, the term digestif is linked to the empirically supposed capacity to improve digestion, but digestion is mainly performed in the bowel, less in the stomach, and a faster gastric transit could even lead to impaired gastric digestion. Postprandial discomfort is rather linked to perception [6] and few data exist on gastric perception and alcohol. A putative mechanism by which chronic alcohol consumption is associated wih satiety and fullness may be represented by the alteration of ghrelin release [7, 8].

It would be interesting to know whether the authors found any correlation between the symptom score on the VAS and the parameters of gastric emptying, i.e. if certain subjects who have higher perception of satiety and fullness present delayed gastric emptying.

Second, the protocol has some limitations: due to ethical issues, the study was performed in healthy, moderately drinking volunteers. But postprandial fullness and satiety occur in dyspeptic patients rather than in healthy individuals, and dyspeptic patients frequently avoid alcohol consumption. A further study on dyspeptic patients would bring more light into the effect of the digestifs on postprandial gastric motility and symptoms.

The caloric value of the food (576 kcal) was lower than that ingested during a lunch or dinner given in the observation of a certain event, when digestifs are usually served; surprisingly, the dose of alcohol administered failed to significantly increase blood alcohol level (a good news for car drivers): in this condition it is also expectable to have no physiologic effect on gastric motility. Repeating the study with higher doses of calories and of alcohol in the test meal would be necessary to better understand the effect of postprandial alcohol ingestion.

This means that digestifs in the dose of one unit after a solid meal of about 600 kcal do not change gastric emptying and do not significantly increase alcoholemia. Their use remains thus a cultural matter linked to the palatable properties of these beverages. If they really improve digestion, as their name misleadingly suggest, this depends on their effects on digestive enzymes rather than on motility.

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Reply,

Consumption of hard liqueurs (digestifs) after comprehensive meals is a wide spread custom in order to relieve postprandial complaints such as fullness, bloating or epigastric discomfort. Scientific information about the potential effect of postprandial ingested alcoholic beverages on gastric motility was lacking before we performed our study. It was known that alcoholic beverages prolong gastric emptying [1]. Emptying of alcoholic beverages produced by fermentation (beer and red wine), but not those produced by distillation (whiskey) requires a 50% more time than their corresponding ethanol solutions (4, 10 and 40% v/v) [1]. Alcoholic beverages also affect gastric emptying of solid meals. Beer, red wine and whiskey prolong gastric emptying of a subsequlently consumed meal by 15 to 30 % [2, 3].

A possible positive effect of postprandial alcohol consumption may be induced by different mechanisms (e.g. increased gastrointestinal secretion, altered gastric perception). The effect on gastric emptying which was examined in our study may be another mechanism since fullness has been shown to be correlated with the antral gastric content [4]: increased gastric emptying might therefore be associated with a faster relieve of postprandial fullness.

However, in our study the postprandial consumption of various digestifs had no effect on the gastric emptying rate of a solid meal. Our study may have some limitations resulting from the limited sample size, the relatively low caloric content of the meal and the inclusion of young, healthy, male volunteers. Therefore, our results may not be valid for other groups of persons (e.g. dyspeptic patients). However, it was the first study dealing with the effect of postprandial alcohol consumption on gastric emptying and dyspeptic symptoms.

Further studies with a larger sample size, different amounts of postprandial alcohol and additional measurement of gastric perception might be helpful to understand if there is any scientific basis for the assumption of many people that postprandial alcohol consumption may relieve postprandial complaints.

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