Acute Pancreatitis as an Unusual Complication of Corrosive Ingestion

To the Editor,

We read with great interest the case report "Unusual presentation and complication of caustic ingestion. Case report" by Lionte *et al*. in the March 2007 issue of Journal of Gastrointestinal and Liver Diseases (1). We report a young patient who presented with acute corrosive ingestion and developed pancreatitis 10 days after ingestion.

A 25 year old young female presented as an emergency case following accidental ingestion of 100ml of hydrochloric acid (HCl). She vomited immediately after HCl ingestion. At admission, she was hemodynamically stable with saliva drooling. On examination, she had a red, swollen tongue and her oropharynx was edematous and inflamed. Nothing could be given by mouth, only intravenous fluids and intravenous proton pump inhibitor (PPI). Her liver function tests, hemogram, renal function tests, chest X-ray, urine routine examination, ECG, abdominal X-ray and ultrasonography were normal. The esogastroduodenoscopy performed two days after ingestion revealed erythema and edema of oropharynx, Grade II A injury in esophagus, grade II B injury in stomach and duodenum (2). On day 10 she had severe epigastric pain radiating to her back associated with vomiting. On examination she had epigastric tenderness. Her serum amylase and lipase levels were more than 3 times elevated. Her serum calcium, serum lipids were normal, abdominal ultrasound did not show sludge or stones in gallbladder, common bile duct was normal and abdominal X-ray was normal with no evidence of perforation. Contrast-enhanced CT of the abdomen showed a bulky pancreas. Continuous tube aspiration, intravenous fluids, prophylactic antibiotics, analgetics and PPI but nothing oral were given. Symptoms improved over the next 5 days. On day 16, a liquid diet was initiated, following which she had recurrent vomiting. She underwent repeated endoscopy which showed similar lesions in the esophagus, stomach, and also in the first part of the duodenum. There was stricture at D1 and D2 junction which could not be passed by the endoscope. The patient underwent a feeding jejunostomy.

After 3 weeks, a barium swallow and meal showed esophageal stricture with reduced capacity of the stomach. She underwent dilatation of the esophageal stricture with a 7mm Savary-Gillard dilator. Further three sessions of dilatation with a 9 mm dilator followed. The intention was to dilate the esophageal stricture and then to perform balloon dilatation of the duodenal stricture.

Acid injury is usually limited to the stomach: 6-20% of patients have associated esophageal and small intestinal injuries (2). The subsequent sparing of the duodenum is thought to be due to a pyloric spasm induced by the irritant acid in the antrum and the alkaline duodenal pH (3).

Our patient had circumferential ulceration at D1 and D2, which led to duodenal stricture. She developed pancreatitis, with high amylase and lipase levels in the setting of severe pain in the abdomen, due to papillary edema consecutive to corrosive ingestion.

Necrotizing pancreatitis has been reported in patients with transmural necrosis and perforation of the stomach (3). Our report supports the description of two out of nine patients in the surgical series who had pancreatic head necrosis and distal necrotizing pancreatitis with involvement of duodenal mucosa (4). However, even in initially stable patients, significant injuries of the duodenum and pancreas should be suspected and actively searched for.

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CT Aspects of Gastrointestinal Stromal Tumors: adding EUS and EUS Elastography to the Diagnostic Tools

To the Editor,

We have read with much interest the viewpoint by Lupescu *et al* about the role of CT examination in the detection and characterization of gastrointestinal stromal tumors (GISTs) (J Gastrointestin Liver Dis 2007; 16: 221-225). We take this opportunity to highlight the role of endoscopic ultrasound (EUS) and EUS Elastography for the accurate diagnosis of GISTs located on the wall of the gastrointestinal tract. GISTs are the most common mesenchymal neoplasms of the gastrointestinal tract, and can arise anywhere in the gastrointestinal tract but their most frequent location are the stomach (50%), small bowel (25%), colon and rectum (10%) (1). Therefore, a large proportion of GISTs are accessible for endoscopic examination and EUS characterization.

We already reported a case of ulcerated gastric stromal tumor hospitalized in our center for upper gastrointestinal bleeding, where EUS and CT examination concurred for accurate diagnosis (2). Recently, we identified retrospectively in our center 20 patients diagnosed by endoscopy, EUS, CT and confirmed by immunohistochemistry performed on surgical resection specimens (3). GISTs were found to be responsible for 0.94% of all digestive tract malignancies; they originated from the stomach (55%, n=11), small intestine (30%, n=6), colon (15%, n=3). 93% were KIT (CD117) positive. Curative surgical resection was possible in 60% (n=12) of cases; 2 patients received imatinib mesylate. EUS was performed in 11 patients with GIST using a radial scanning echoendoscope 7.5 MHz. On EUS, typically GISTs appear as large hypoechoic and homogeneous masses, usually well circumscribed, that can arise from either the 2nd hypoechoic layer (muscularis mucosae) or the 4th hypoechoic layer (muscularis propria) (Fig.1). The sensitivity and accuracy of diagnosing GISTs has been reported as 95% and 87%, respectively (4). Tumor size greater than 4 cm in GISTs showing irregular, poorly defined borders and heterogeneous echo texture with hyperechoic foci and/or anechoic spaces which indicate necrotic areas inside large tumours are good indicators of malignancy. The EUS sensitivity in detecting malignant GISTs ranges between 80% and 100% (5,6).

EUS elastrography (Hitachi 6500 Ultrasound System with SonoElastography module, Hitachi Medical System, Zug, Schwitzerland coupled with EG 3870 UTK linear

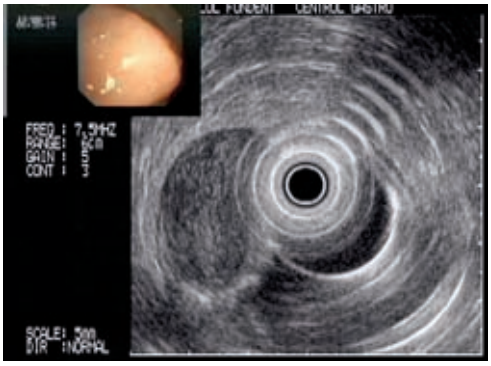

Fig.1 EUS appearance of a 2.5 cm hypoecoic homoge-neous mass arising from the 4th layer (muscularis propria) of the gastric wall.

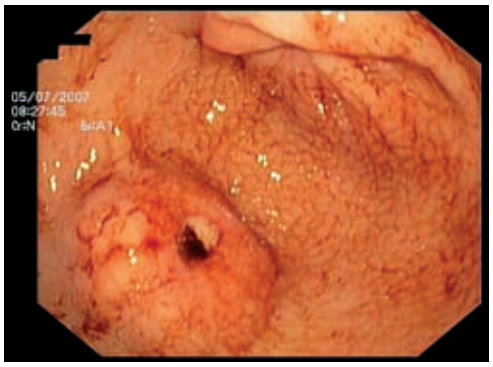

Fig.2 Endoscopic appearance of a bleeding gastric stromal tumour.

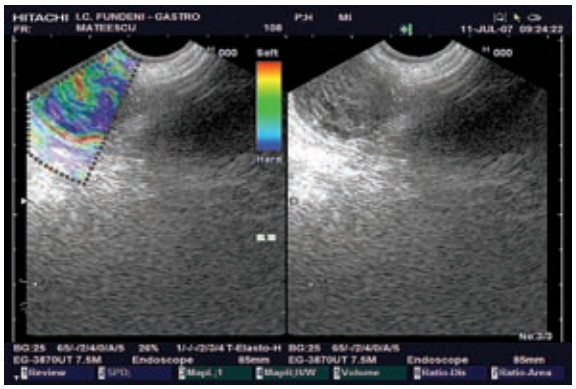

Fig.3 EUS elastography appearance of a 2.5 cm benign gastric stromal tumour showing a moderate and inhomogeneous increase in tumour stiffness (blue-green); the periphery is stiffer than the surrounding tissues with a softer central area (pattern 3, suggesting a benign lesion).

echoendoscope, Pentax, Hamburg, Germany) is increasingly used to improve the accuracy in the characterization of a benign or malignant nature of a lesion by analysing the tissue elasticity distribution inside the tumor. Either a qualitative assessment of the tumoral pattern according to Ueno classification for breast lesions (pattern 1 to 5), or a quantitative histogram analysis of the fundamental colours (red-green-blue) in images recorded during real time EUS Elastography (Adobe PhotoShop 7.0; Image J) may be used. Recently, we added to endoscopy (Fig.2) EUS with Elastography for the assessment of the GISTs (Fig.3). In

most cases, the benign or malignant nature of GISTs, leiomyomas, and schwanomas cannot be accurately differentiated by EUS or SonoElastography, thus biopsy or/and EUS-guided fine-needle aspiration (EUS-FNA) with histopathological examination and immunochemistry remain mandatory.

Due to its sensitivity and accuracy in diagnosing the benign or malignant nature of GISTs, EUS with EUS Elastography should be included routinely in the pre-operative assessment of these tumours.

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Hepatic Heterototopic Tissue on the Gallbladder Wall: an Incidental Finding with Pathological Potential

To the Editor,

The presence of histological normal tissue at an abnormal location has been termed choristoma by Albert (1904). Other terms such as heterotopic or ectopic tissue are synonyms with choristoma and used more frequently. Hepatic heterotopic tissue is an uncommon condition incidentally identified during the surgical exploration of the abdominal cavity for other causes (1). Hepatic choristomas have been found above and below the diaphragmatic muscle. Hepatic choristoma associated with the gallbladder (GB) is the most frequent abdominal location (2, 3). Despite this relative frequency, only 21 cases have been described in the literature. We present four cases of hepatic choristoma located on the wall of the GB in patients who underwent cholecystectomy for different indications. One of these cases has already been reported by us elsewhere (4).

The first case, a 47-year old man underwent laparoscopic cholecystectomy for symptomatic cholelithiasis. The preoperative abdominal ultrasound (US) reported the presence of gallstones and did not identify any mass associated with the GB. At laparoscopy a small solid mass (12 x 8 x 6 mm) adherent to the serosal surface of the vesicular body was observed. The histopathological examination showed chronic changes in the GB wall and diffuse lymphocytic infiltrates in the mucosa and muscularis mucosae. The hepatic nodule was adherent to the serosa and not to the GB wall. The hepatic histological architecture was preserved and diffuse lobulillar hyperemia was identified (4).

The second case, a 27-year old man was submitted to total gastrectomy for sub-cardial gastric cancer and as part of the procedure a cholecystectomy was performed. At histology, a hepatic choristoma (5 x 7 x 4 mm) was found adherent to the serosal surface of the GB. The histological architecture was normal (Fig.1). Preoperative abdominal US and CT scan did not report the presence of this small ectopic liver.

The third case, a 33-year old woman underwent laparoscopic cholecystectomy for symptomatic cholelithiasis diagnosed by abdominal US, which revealed only gallstones. During laparoscopy, a hepatic choristoma (17 x 6 x 5 mm) was identified over the serosa near the GB fundus; we noticed that the irrigation of the choristoma was provided by a branch of the cystic artery (Fig. 2). The histological parenchymal architecture was preserved.

The fourth case, a 35-year old man underwent exploratory laparotomy for a pancreatic pseudocyst after an episode of severe acute pancreatitis. As a part of the procedure the GB was removed and a hepatic choristoma (18 x 6 x 4 mm) was found (Fig.3), with histologically normal hepatic architecture. Preoperative abdominal US and CT scan failed to identify any mass adherent to the GB wall.

Anatomic anomalies of the liver have been classified as accessory hepatic lobe communicating with the liver by a stalk of normal hepatic tissue and heterotopic liver or hepatic choristoma without any vascular or parenchymal connection to the native liver (1, 5). Hepatic choristoma is the least common of these two abnormalities and as a consequence the clinical importance of hepatic heterotopia has only recently been established and its surgical resection has been recommended due to the high potential risk of hepatocarcinoma that was observed by some authors (5-7). The preoperative diagnosis of hepatic heterotopia has seldom been reported. Of 21 published cases only three have been operated on with the preoperative diagnosis of a mass attached to the GB wall: one patient had an abdominal US and the other two a computerized abdominal scan (1, 3), however the mass observed by these radiological studies was not identified as heterotopic liver. None of the studies performed in our patients identified any mass related to the GB. The hepatic choristoma was an incidental operative

finding in three cases and an incidental pathological finding in one case.

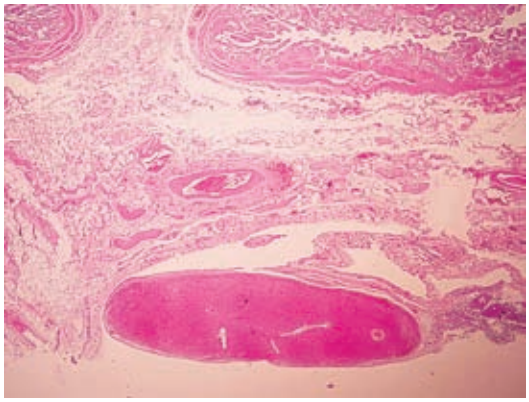

Fig.1 Hepatic heterotopic tissue.

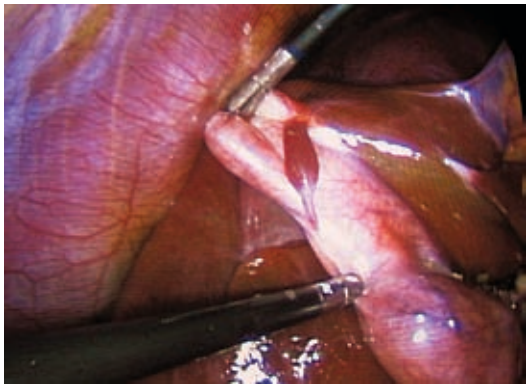

Fig.2 Laparoscopic view of a hepatic choristoma.

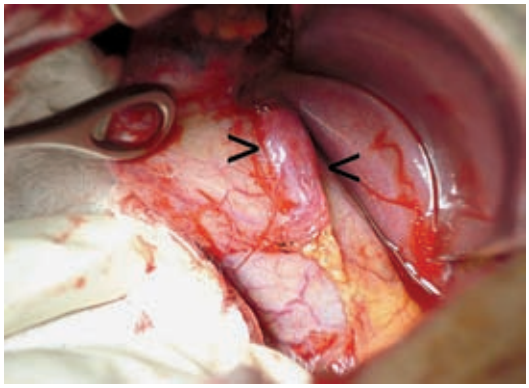

Fig.3 Hepatic choristoma (arrow-heads) on the gallbladder.

The cause of heterotopic hepatic tissue is aberrant migration during the embryologic development of the liver. This theory explains the most frequent locations of this choristoma near the liver: in the falciform ligament, GB, diaphragm, thorax intra and extrapleural, adrenal glands, pancreas, pylorus, omentum, spleen, esophagus, umbilicus and umbilical cord, retroperitoneum and pericardium. Of these locations, the most common is the GB (1,5). When the hepatic heterotopia is localized in the GB, it is usually overlying the serosal surface or is serosal encapsulated as in our cases, or less frequently intramuscular and submucosal (1,8).

Histopathological findings in hepatic heterotopia include normal parenchyma, fat infiltration, cirrhosis and hepatocellular carcinoma (1,5-7). Normal native liver and heterotopic hepatic tissue share the same histological characteristics or histopathological changes such as the coexistence of cirrhosis. A higher susceptibility and potential risk of malignant degeneration of hepatic heterotopic tissue secondary to metabolic alterations associated to abnormal bile and venous drainage has been reported (1,5,6). Currently, 25 cases of hepatocarcinoma in heterotopic liver associated to a native normal liver have been reported; in most of these cases cirrhosis coexisted in native and heterotopic liver (5). Based on these reports and other studies (7), the higher risk of hepatic heterotopic tissue to malignant degeneration was accepted, and supports the concept of curative surgery with the complete resection of the ectopic tissue and the affected organ. In some published cases in which the indication for surgery was acute cholecystitis, the histopathological findings in heterotopic liver were cholangitis and congestion secondary to the inflammatory process in the GB (9). Our patients were all electively operated. Even though the hepatic choristomas were all unsuspected findings, we considered it important to highlight the pathological implications of this ectopic tissue and its associated malignant potential.

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Reversible Acute Reactive Arthritis secondary to Iatrogenic Blind Loop Syndrome

To the Editor,

We report a case which highlights that small bowel bacterial overgrowth (SBBO) occurring in the setting of a surgically fashioned blind intestinal loop, particularly where this loop is partially obstructed, may result in a systemic inflammatory syndrome and that surgical decompression of the obstructed intestinal segments may result in rapid resolution of this syndrome.

A 57 year old man presented to our centre with a four month history of diarrhoea and arthralgia which began three weeks after undergoing a partial gastrectomy and vagotomy (Roux-en-y gastrojejenostomy with jejuno-jejunostomy) for a chronic non-healing prepyloric gastric ulcer. He had 6 to 8 loose bowel motions per day with associated intermittent postprandial upper abdominal discomfort. Arthralgia was described as pain and stiffness affecting hands, wrists, elbows, hips and feet. Abdominal examination was unremarkable. However, examination of the locomotor system revealed synovitis of proximal interphalangeal, metacarpophalangeal, ankle and metatarsophalangeal joints.

Routine blood profile revealed a normochromic normocytic anaemia (haemoglobin of 10.7 g / dL). The inflammatory markers were significantly elevated (CRP 25.1 mg/dL, ESR 84 mm/hr). The immunoglobulin profile revealed an acute phase response and albumin concentration was reduced at 28 g/dl. Of note rheumatoid factor, anti-nuclear factor, anti-nuclear cytoplasmic antibody and HLA-B27 typing were negative. Sacro-illiac joint radiographs were normal

Gastroscopy revealed significant food residue in stomach, but was otherwise unremarkable. Small bowel biopsies were normal. Colonoscopy to the terminal ileum was also performed, again no abnormality was found. Terminal ileal biopsies showed normal ileal mucosa. Antibiotic therapy including metronidazole, amoxycillin and oxytetracycline was ineffectual.

A small bowel follow through showed a slight increase in calibre of proximal small bowel loops, however no definite strictures or obstruction were identified. CT abdomen demonstrated thickening and dilation of afferent jejunal limb, the efferent jejunal limb being of normal calibre. On the basis of the above imaging a laparotomy was performed; dilatation was noted of both afferent and efferent jejunal limbs to beyond jejuno-jejunal anastomosis. The transverse mesocolon was seen to be exerting compressive effect on both jejunal limbs. Both anastomoses were repositioned and secured inferior to transverse mesocolon to avoid risk of further com-pression.

A clinical review was performed six weeks after the surgery. Diarrhoea, abdominal pain and arthralgia had completely resolved. Examination demonstrated no evidence of synovitis. Blood profile revealed resolution of anaemia (haemoglobin 13.3 g / dL), normal inflammatory markers (CRP 1.0 mg/dL, ESR 23 mm/hr) and an increased albumin concentration of 35 g/dL.

It is well known that alterations in the anatomy of the small intestine after surgical procedures, particularly the formation of blind intestinal loops predispose to the development of SBBO. Aside from causing disturbance of enteric function, SBBO may also result in systemic inflammatory complications. Fagan et al describe a series of three patients who had jejuno-ileal bypass surgery for morbid obesity and subsequently developed asymmetrical polyarthritis, tenosynovitis, sterile skin pustules, mucous membrane ulceration and retinal vasculitis. Antibiotic therapy did not ameliorate symptoms however dapsone produced a sustained remission. Bypass reversal was subsequently undertaken, but the systemic inflammatory condition recurred. The authors propose that SBBO may only be required to initiate, not perpetuate inflammatory complications (1). Goldman et al report two cases in which necrotizing vasculitis along with tenosynovitis / arthralgia / arthritis syndrome developed after intestinal bypass surgery. Interestingly each of these patients had jejunoileostomies and the excluded segment was placed in an ileocolic anastamosis. The authors speculate that connection of the excluded small bowel segment to the colon resulted in more significant SBBO and consequently a more florid clinical presentation (2). This report does not mention the treatment undertaken. Ross et al describe a case where arthritis was the primary manifestation of a blind intestinal loop syndrome. In this case surgical elimination of the blind loop was curative (3).

In conclusion, this report demonstrates that in the setting of a systemic inflammatory syndrome due to SBBO secondary to a blind intestinal loop, particularly where sub-acute obstruction of the loop is present, surgical correction should be considered and may result in resolution of the associated inflammatory syndrome.

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Hepatic Duct Compression Syndrome Diagnosed by Endoscopic Ultrasound

To the Editor,

Imaging abnormalities involving the hepatic duct are increasingly recognized on magnetic resonance cholangiopancreatography (MRCP). The differentiation of the benign from the malignant and the clinically relevant from the artifactual can be difficult. We describe a case where a patient with mild liver test abnormalities had an MRCP done which showed an eccentric filling defect in the common hepatic duct. A broad differential was offered. Endoscopic ultrasound (EUS) was crucial in making the diagnosis of common duct compression by the right hepatic artery. Familiarity with this increasingly recognized entity may avoid unnecessary invasive and costly procedures.

The right hepatic artery has a variable point of origin but in approximately 75 % of cases arises from the common hepatic artery and crosses the common hepatic duct posteriorly. This arrangement should predispose to an impingement syndrome but, fortunately, this is exceedingly rare.

We describe the first such case where EUS was crucial at arriving at the diagnosis avoiding further invasive procedures and expense. A 57 year old healthy Caucasian male who was found to have a significantly increased gamma-glutamyl-transferase (GGT) on an insurance physical was referred to us for consultation. Physical examination was entirely normal. Comprehensive laboratory evaluation and liver serology was unremarkable except for a minimally elevated alkaline phosphatase. An MRCP was obtained which showed an eccentric filling defect in the common hepatic duct (Fig.1). A differential diagnosis of crossing vessel, stone, polyp, fold or neoplasm was offered. An ERCP was suggested but the patient was referred for EUS instead. An examination with the Olympus GF-UM160 radial echoendoscope was carried out and no abnormalities were detected. A mass lesion was not present. To corroborate the findings, the Olympus GF-UC140P curved-linear array echoendoscope was employed. The Doppler study revealed that the common hepatic duct was crossed and compressed by the hepatic artery (Fig.2) and there was a slight proximal caliber step-up. The MRI was reevaluted in view of these findings and on the T2-weighted images (bile duct bright, vessels dark) the compression of the common hepatic duct by a vascular structure could be retrospectively identified (Fig.3). A CT scan obtained 2 years earlier showed similar features but was not diagnostic of compression (Fig.4).

Imaging abnormalities involving the hepatic duct may be related to stones, Mirizzi syndrome, polyps, strictures, cholangiocarcinoma or metastatic lymph nodes, and, as this case demonstrates, compression by vascular structures.

Clinically relevant common hepatic duct compression caused by the right hepatic artery is an extremely rare condition. When jaundice is present, the compression is often asociated with a hepatic artery aneurysm or aberrant anatomy (1). However, common duct compression by the

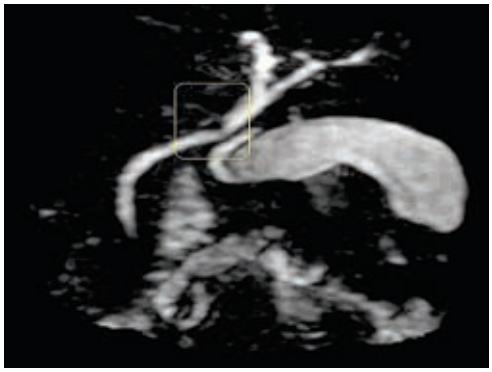

Fig.1 The MRCP showed an eccentric filling defect in the common hepatic duct.

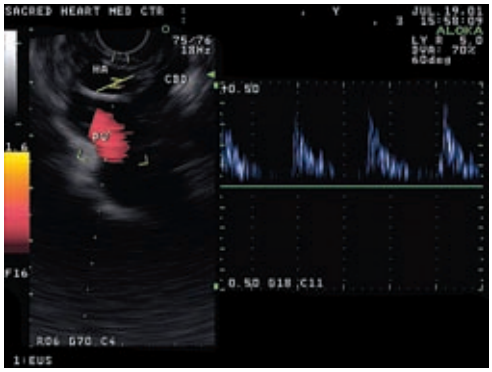

Fig.2 An EUS Doppler study revealed crossing of the common hepatic by the right hepatic artery.

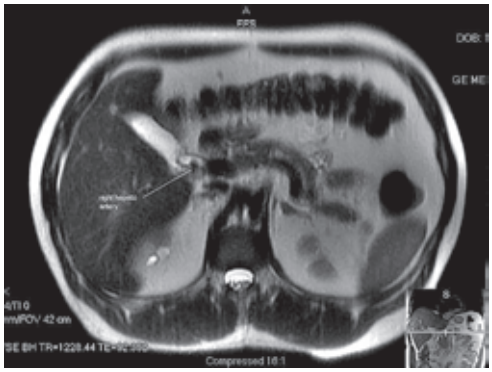

Fig.3 Reanalysis of the MRI T2-weighted images shows a vascular structure (dark) impinging on the bile duct (bright).

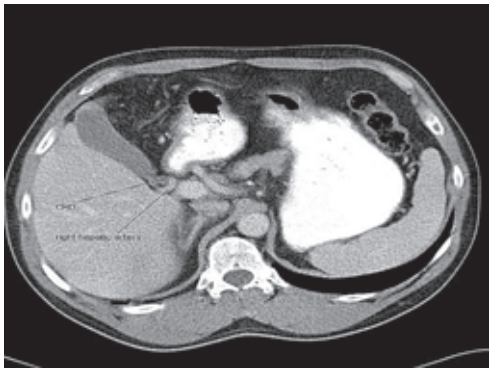

Fig.4 An earlier CT scan shows the anatomic relationships with a different modality.

right hepatic artery is increasingly recognized on MRCP as an incidental finding (2,3). More importantly, the MR abnormalities may be completely artefactual. Pulsatile compression can cause signal intensity loss in the extrahepatic bile duct, which may lead to apparent stenosis or obstruction of the extrahepatic bile duct on MRCP images (4). With the increasing utilization of MRCP the clinician will increasingly be confronted with findings of doubtful clinical significance but potentially important implications. A bile duct polyp is a premalignant lesion which requires further follow-up, mild compression of the common duct by a hepatic artery with a minimal elevation of liver tests does not. The first approach in the evaluation of a patient with an abnormal MRCP showing a hepatic duct defect should be the reanalysis of the images regarding the possibility of an arterial compression syndrome. Of all imaging modalities available to solve clinically doubtful cases, EUS combines the ability to scrutinize a specific area with high resolution images and allows for fine needle aspiration to be performed if a suspicious lesion is found. If the modality is not available, a CT cholangiogram especially with three-dimensional reconstruction may be helpful (3).

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