Pyogenic liver abscess caused by accidental ingestion of a wooden toothpick: role of preoperative imaging

To the Editor,

We have read with great interest the case report published by Katsinelos P et al in the last issue of *Journal of Gastrointestinal and Liver Diseases* (1). The article describes a rare complication of foreign body ingestion, with posterior penetration of the wooden skewer and development of a gastric abscess. Moreover, a minimal invasive option of endoscopic treatment was successful. We would like to add our recent experience with a similar case, where the ingested object penetrated the anterior gastric wall into the left liver lobe, with development of a pyogenic liver abscess. The importance of preoperative imaging tests that allowed a correct diagnosis, before the laparoscopic surgical intervention, is also highlighted.

A 58 year old female was admitted to the Gastroenterology Department for epigastric pain, vomiting, fever, symptoms that started after a copious meal and increased progressively over the following 3 days. The patient mentioned non-steroidal anti-inflammatory drug use during the past months. Clinical examination showed an anxious, febrile patient (39^{o}C), with tenderness in the epigastric area. Laboratory examinations showed high leukocyte count (15000 / mm^{3}) and a high erythrocyte sedimentation rate (95 mm). Transabdominal ultrasound (US) showed initially an inhomogeneous hypoechoic area in the middle of the left liver lobe, of approximately 1.5 cm in diameter, with multiple hyperechoic echoes inside, suggestive of air. Medical treatment with broad spectrum antibiotics as well as proton pump inhibitors was started. After 24 hours, a repeated US showed enlargement of the lesion, which was poorly delineated, containing a hypoechoic area of 3 cm in diameter, with a clear linear hyperechoic echo inside (Fig.1). Upper gastrointestinal endoscopy showed a compression on the anterior antral region with central erosion. Computed tomography (CT) confirmed the presence of a liver abscess (Fig.2), also showing a linear foreign body. An exploratory laparoscopy was further performed and allowed subsequent removal of the penetrating object: a wooden toothpick ingested accidentally during a party, embedded inside a sandwich (Fig.3).

Liver abscess is a rare complication of foreign body ingestion (2). However, different sharp objects, especially chicken/fish bones, as well as wooden/plastic tooth picks, can easily penetrate the gastric wall into the liver, with a consequent formation of a liver abscess. Although extremely rare, several cases are already described in the literature (1). Early diagnosis remains a challenge because the clinical signs are usually delayed and relatively non-specific, with no history of the ingestion of a foreign body.

The presence of linear hyperechoic structures has been already described during transabdominal US or endoscopic US as a hallmark of penetrating foreign objects (3, 4). Likewise, the visualization of a hyperdense linear image during CT scan evaluations might offer a clue for the diagnosis (5). An extensive and correct preoperative evaluation with state-of-the-art imaging tests like US and CT are critical not only in establishing the diagnosis of pyogenic liver abscess, but also for determining the cause of the abscess (6). The outcome is currently considered favorable in view of the possibility of miniinvasive treatment, which includes laparoscopy or percutaneous drainage (7). Fatal outcome after this complication has been reported in recent papers (8). Consequently, a high degree of suspicion should be maintained in order to diagnose the condition early.

In conclusion, we presented the problems of positive and differential diagnosis of hepatic abscesses induced by foreign body ingestion and penetration through the gastric wall, with special emphasis on the role of imaging methods that allow an early diagnosis and a mini-mum invasive treatment approach.

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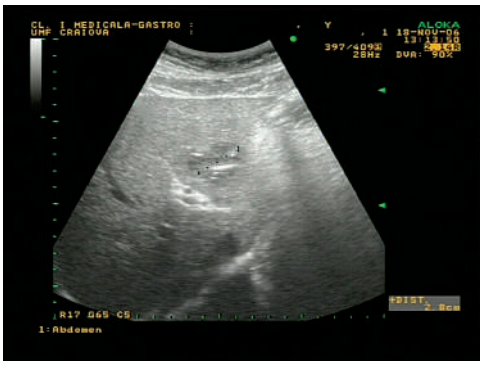

Fig.1 Transabdominal ultrasound: transverse section in the epigastrium showing a 3 cm hypoechoic area, with a linear hyperechoic echo inside, suggestive of liver abscess induced by a foreing body.

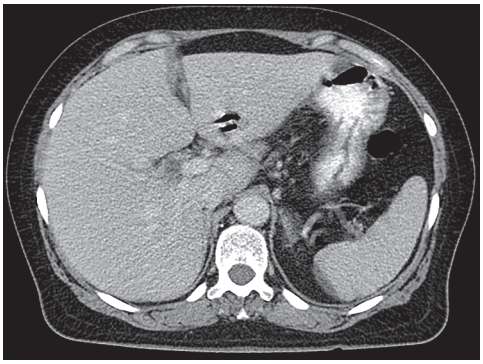

Fig.2 Contrast-enhanced computed tomography: air in the left liver lobe, suggestive for the diagnosis of liver abscess.

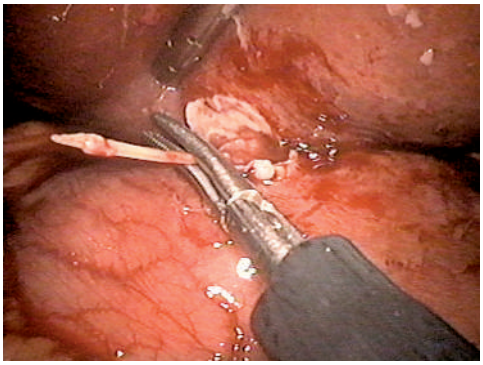

Fig.3 Laparoscopic extraction of the foreign body: a wooden toothpick ingested accidentally.

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Primary sclerosing cholangitis lesions in a patient with Crohn's disease and HCV infection

To the Editor,

It is well known that patients with chronic inflammatory bowel disease (IBD) have a higher incidence of primary sclerosing cholangitis (PSC) that varies from 2.5 to 7.5%. This association is more obvious for PSC ailing patients; a significant percentage, that varies from 50 to 75%, of these patients will develop IBD (1).

The etiology of PSC remains unknown. Bacteria, toxins, viral infections, immunological and genetic factors have all been proposed as etiological agents. Patients with PSC may have elevated levels of circulating immune complexes and autoantibodies (2). On the other hand, an etiopathological link between hepatitis C virus (HCV) and autoimmune liver disease has been suggested, as it is well known that autoantibodies, as well as autoimmune extrahepatic manifestations are often observed during the course of viral C hepatitis (3).

A smoker, 26-year-old female was admitted to our department with abdominal colicky pain, 4-5 diarrheic stools per day, weight loss and fever of 3 weeks duration. She mentioned appendectomy 20 years ago. Abdominal examination revealed hepatomegaly and right lower quadrant and periumbilical tenderness.

Initial laboratory studies showed: Ht 42%, Hb 13.7g/dl, MCV 89fl, PLT 300K/ul, WBC 14.6K/ul, ESR 35mm/h, C reactive protein 2.03mg/dl (normal value <0.8mg/dl), alkaline phosphatase 381u/l (90-270), AST 79u/l (9-48), ALT 148u/l (5-49), g-GT 179u/l (0-53), BUN, serum creatinine, total bilirubin normal, serum amylase 143u/l (0-96), urine amylase 658u/l (0-350), serum protein 7.5 g/dl, albumin 3g/dl, INR 1.03, aPTT 36.1". Autoantibodies (ANA, anti-dsDNA, AMA, ASMA, c/p ANCA) were negative. HCV: positive, HBsAg: negative, HIV 1/2 : negative, HCV-RNA: 1,324 000 IU/ml (Versant HCV RNA 3.0 Assay). Urinalysis revealed plenty of oxalic calcium crystals. Stool analysis revealed numerous leukocytes and no ova or parasites.

Barium contrast small-intestinal radiologic examination revealed diffuse mural edema of terminal ileum. Ultrasonography of the liver, gallbladder, intra- and extrahepatic bile ducts revealed no abnormalities. Abdominal CT scan identified mural thickening of terminal ileum without adenopathy. Colonoscopy demonstrated confluent eruption of the mucosa without ulcers, in a segmentary pattern, from the anal verge to the terminal ileum. Biopsy from terminal ileum during colonoscopy was compatible with Crohn's disease (CD). The mucosa showed architectural changes such as irregularity and focal shortening of the villi with inflammatory infiltrate consisting of histiocytes, lymphocytes, eosinophilic and neutrophilic leukocytes. The inflammation process involved also submucosa. Furthermore, granulomas were found supporting the diagnosis of CD.

Treatment was started with metronidazole and ofloxacin. The patient became afebrile with a significant decrease of the number of stools. She was in good general condition but mentioned mild colicky abdominal pain.

Laboratory findings of increased AST, ALT, γ-GT, alkaline phosphatase, serum and urine amylase persisted, serum bilirubin increased. ERCP revealed two stenoses of the bile duct - in both the initial and terminal segment - with mild dilatation of pancreatic and hepatic duct. No gallstones were observed in the gallbladder.

A liver biopsy showed histological features of non specific portal tract changes (mild inflammation with lymphocytes and few neutrophiles), interlobular bile duct epithelial changes with inflammatory infiltration of their walls surrounded by a ring of oedematous or hyaline fibrosis (periductular fibrosis), and no granulomas were detected (Fig.1).

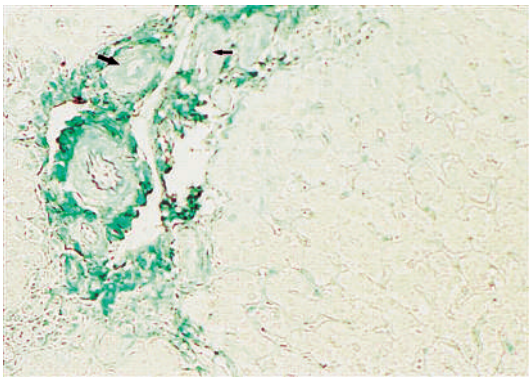

Fig.1 Periductal fibrosis of inter-lobular bile-duct. Masson stain x 250.

Mesalazine and ursodeoxycholic acid (UDCA) were added to treatment. The patient was discharged 8 days later without symptoms. After two months she was free of symptoms and laboratory findings were normal.

Hepatobiliary disorders are well known complications in patients with IBD. Histological findings at liver biopsies were unrelated to either activity or extent of colitis, except for the onion lesions which were seen exclusively in biopsies of patients with involvement of the total colon (4). Cholangiography and liver biopsy are both needed to evaluate the extent of the disease. The close association between PSC and IBD remains unexplained and both groups of patients have a high prevalence of autoantibodies. Possible triggers of liver autoimmunity have been explored and several sequences shared in common between autoantigenes and hepatotropic viruses, namely hepatitis B, C and cytomegalovirus have been identified (5). It has been hypothesized that HCV antigens could enhance immune cell sensitization and cytotoxicity by being homologous with host antigens or by molecular mimicry (6). Findings suggesting cross-reactivity between homologous sequences, especially between HCV and cytochromes, support the possibility that molecular mimicry plays a role in the induction of autoantibodies and autoreactive cytotoxic T cells.

To summarize, in the reported case, although a mere coexistence of PSC and HCV infection is probable, we hypothesize that HCV may be related, as a trigger of autoimmunity, to PSC type lesions of intrahepatic and possibly extrahepatic bile ducts.

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Acute pancreatitis attributed to the use of pegylated interferon in a patient with chronic hepatitis C

To the Editor,

Acute pancreatitis as a complication during interferon (IFN) (standard or pegylated) therapy of chronic hepatitis C (CHC) is rare (1-4). If the causative co-morbid HIV infection is excluded, there have been only 11 cases a 9 treated with standard IFNα, 2 with pegylated IFNα-2b) of acute pancreatitis, reported as a complication of the therapy of CHC patients, in the English literature (1-4). Here, we describe an additional case.

A 45-year-old man was admitted with newly diagnosed asymptomatic CHC with positive HCV RNA (10,540 000 copies/ml by PCR) and elevated aminotransferases (AST 92 UL, ALT 126 U/L) for the treatment. The genotype was 1b. He had no concomitant medical disorders, drug usage or alcohol abuse. The physical examination was normal. His complete blood count and abdominal ultrasonography were normal. Therapy was started with pegylated IFNα-2b subcutaneously 120 μg/week and ribavirin 1200 mg per oral daily. At the 5th week of the therapy he developed severe epigastric pain radiating to his back and nausea. Laboratory testing was notable for high levels of serum amylase 634 U/L (N: 28-100) and lipase 856 U/L (N: 7-60). White blood cell count, AST, ALT, alkaline phosphatase, total bilirubin, calcium, cholesterol and triglyceride were within normal limits. The abdominal ultrasound revealed pancreatic edema. The gallbladder and common bile duct appeared normal and no gallstones were noted. MR cholangiopancreatography was also normal. After the common risk factors for acute pancreatitis were excluded, the therapy was stopped and the patient was treated with supportive care. His epigastric pain resolved and the amylase level decreased to normal-limits 10-days after cessation of the treatment. The patient refused to continue the IFN therapy and did not develop any recurrence during 1-year of follow-up.

Our patient had no evidence of potential causes of acute pancreatitis such as gallstones, chronic or recent alcohol consumption, or other causes identified. The Naranjo's algorithm total score of our patient was 7, which showed a probable drug adverse reaction (5). Rapid resolution of symptoms after stopping the therapy also suggests a probable drug-induced pancreatitis (6). One year of recurrence free period in the follow up suggests that pegylated IFN usage was the most likely cause of acute pancreatitis.

Of the 11 cases of acute pancreatitis developed during IFN therapy in the literature, only two were related with pegylated IFN therapy (2,4). Table I shows the features of the other cases that developed acute pancreatitis during CHC therapy. Acute pancreatitis developed within 5 months (1-21 weeks) of therapy in these patients. The mechanism of IFN-induced acute pancreatitis is still unknown. We underline the importance of monitoring patients on pegylated IFN therapy, since a few signs and symptoms could be important for an early diagnosis before serious adverse events.

In conclusion, this is the third patient who developed acute pancreatitis, during the therapy with pegylated IFN for CHC. Although it is rare, the physicians should be aware of the possibility of acute pancreatitis when a hepatitis C patient on pegIFN therapy presents nausea and epigastric pain.

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Table I Summary of standard or pegylated interferon induced acute pancreatitis patients during the therapy for chronic hepatitis C

Reference and year	No case/ Gender	Age (yrs)	Medical history	Initial ALT	Initial HCV RNA copies/ml	Interferon and dose (SC)	Ribavirin and dose (oral)	Duration of therapy(weeks)	Amylase/ lipase (U/L)	Radiological findings
Eland et al (1) 2000	1/ M	40	Normal	NA	NA	3 MU IFN-α tiw	1200 mg daily	15 weeks, symptoms for 12 weeks	136 / 3972	US: Normal
Eland et al (1) 2000	2/ M	38	Meningitis, thrombosis of subclavian vein	NA	NA	5 MU IFN-α tiw	Not given	7 weeks, three times symptoms in 7 weeks	137 / 740	US: Sludge in the gall-bladder CT: Normal ERCP: Normal
Cecchi et al (2) 2004	3/ M	52	Normal	NA	NA	Pegylated IFNα-2b 80 μg/week	1000 mg qd	1 week	1714 / 1014	CT: Normal
Chaudhari et al (3) 2004	4/ M	49	Normal	NA	>5x 10^6	3 MU IFN-α tiw	1200 mg qd	12 weeks	764 / 2765	US, CT: Edematous pancreas
Chaudhari et al (3) 2004	5/ M	51	Gout, post-traumatic stress disorder	NA	850,000	3 MU IFN-α tiw	1200 mg qd	20 weeks	330 / 285	CT: Edematous pancreas
Chaudhari et al (3) 2004	6/ F	54	Reflux, and cholecystec-tomy	NA	597,267	3 MU IFN-α tiw	1000 mg qd	7 weeks	300 / 500	US, CT: Edematous pancreas
Chaudhari et al (3) 2004	7/ F	49	Mild depression	NA	2,57 x10^6	3 MU IFN-α tiw	1000 mg qd	20 weeks	426 / 1867	US: Edematous pancreas MRCP: Normal
Chaudhari et al (3) 2004	8/ M	60	Panic attacks, chronic lower back pain	NA	1,5x10^6	3 MU IFN-α, qd	1000 mg qd	10 weeks	1813 / 2278	CT: Edematous pancreas
Chaudhari et al (3) 2004	9/ M	52	Hypertension	NA	1,37x10^6	3 MU IFN-α, qd	1200 mg qd	4 weeks	182 / 460	CT: Normal
Chaudhari et al (3) 2000	10/ F	45	Depression	NA	4,1x10^6	1.5 MU IFN-α, 6x/ week	1000 mg qd	21 weeks	247 /171	US, CT: Edema in the tail of the pancreas
Ozdogan et al (4) 2007	11/ F	65	Normal	92	7,32x10^6	Pegylated IFNα-2b 120 μg/week	1000 mg qd	7 weeks	302 / 672	US: Pancreatic edema, HM MRCP: Normal
Present case	12/ M	45	Normal	126	10,54 x10^6	Pegylated IFNα-2b 120 μg/week	1200 mg qd	5 weeks	634 / 856	US: Pancreatic edema, MRCP: Normal

M: Male, F: Female, IFN: Interferon, SC: Subcutaneously, qd: daily, tiw: thrice weekly, NA: Not available, US: Ultrasonography, ERCP: Endoscopic retrograde cholangiopancreatography, MRCP: Magnetic resonance cholangiopancreatography, HM: Hepatomegaly