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Case report

Gastric perforation by fish bone with hepatic abscess formation presenting as prolonged fever

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ABSTRACT

A 70-year-old woman presented to the emergency department with a 3-week history of prolonged fever, asthenia and anorexia, denying other symptoms. Physical examination was unremarkable and the patient admitted for further investigation.

Initial laboratory testing showed leucocytosis, elevated C-reactive protein and cholestasis, without hyperbilirubinemia or cytolysis. Abdominal ultrasonography found no abnormalities. Viral serologies, autoimmune tests and blood cultures were collected for further investigation of causes of prolonged fever with hepatic involvement. After two days, *Citrobacter koseri* was isolated in blood cultures and intravenous (IV) piperacillin–tazobactam initiated. Computed tomography (CT) scan of the abdomen showed a left lobe hepatic abscess with gas and a linear hyperdense image, possibly a foreign body, piercing through the gastric antrum into the abscess. Surgical exploration was done for source control. The abscess was drained and the foreign body, a 3.5 cm long fishbone, was removed. The patient's condition rapidly improved. Gastrointestinal perforation due to the ingestion of sharp and elongated foreign bodies usually occur in ileal loops, where the intestinal wall is thinner, causing extravasation of fluids and air into the peritoneum and typically presents with an acute abdomen. The uncommon location of perforation masked these symptoms leading to the unusual presentation with prolonged fever.

Introduction

Fever of Unknow Origin (FUO) has diverse differential diagnoses, each with its unique challenges. Infections, occult neoplasia, lymphoproliferative and autoimune diseases are the most common causes of FUO. Classically, Petersdorf and Beeson defined it as a prolonged fever of ≥38.3 °C for > 3 weeks that remains undiagnosed after one week of in-hospital work-up, but the concept has evolved since due to the advent of modern imaging and laboratorial tests [1]. Epidemiology of FUO is changing; while in developing countries infectious diseases remain the main cause of FUO, non-infectious diseases now amount for the majority of cases in developed countries [1,2]. Although not the most common cause of infectious FUO, intra-abdominal abscesses are recognized as a classical cause of FUO. History of previous abdominal surgery is common in patients with intra-abdominal abscesses, but other less common causes, like ingestion of foreign bodies, may be the actual culprits in some cases [1,2].

Ingestion of foreign bodies is common, but most pass the gastrointestinal tract without complications. Less than 1% of ingested foreign bodies perforate the bowel, mostly in the terminal ileum and rectosigmoid junction. Bone fragments, toothpicks and fish bones are the most common culprits. Patients generally don't recall ingesting the foreign body, which complicates the diagnosis, especially when typical findings such as an acute abdomen, abdominal pain, nausea, vomiting, pneumoperitoneum, intestinal obstruction or gastrointestinal bleeding are not present [3,4].

Case presentation

We report the case of a 70-year-old woman who presented to the emergency department (ED) with a 3-week history of fever and progressively worsening anorexia and fatigue. During the first two weeks of illness, patient had intermittent fever. The week prior to the presentation to the ED, patient had daily fever with

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temperature ranging from 38.0 °C to 38.5 °C. Review of the symptoms were negative except mentioned. Past medical history included hypothyroidism, obesity, gastroesophageal reflux disease, hypertension and depression. Chronic medications were levothyroxine, omeprazole, ramipril, hydrochlorothiazide, and escitalopram.

The patient was haemodynamically stable. She had one temperature spike in the ED. The physical examination was unsuspicious. Cutaneous inspection found no abnormalities, no lymphadenopathies were detected, cardiopulmonary auscultation was normal and abdominal palpation revealed non-tender abdomen without organomegaly. An initial laboratorial workup was performed, showing leucocytosis (19.3 × 10^9/L), elevated C-reactive protein (149.4 mg/dL) and sedimentation rate (89 mm/1st hour) and cholestasis (alkaline phosphatase 512U/L, gamma-GT 818U/L) without hyperbilirubinemia or cytolysis (Table 1). Serum albumin and total proteins were low, but there was no coagulopathy or other signs hinting at acute hepatic failure. Abdominal ultrasonography was normal. Acute kidney injury (AKI) was also detected at admission. It was interpreted as prerenal AKI, given the quick normalization of blood urea and creatinine with fluid administration and normal ultrasound, prompting no further evaluation.

In the light of these findings, the patient has questioned further. She denied contact with animals and was living in an urban area; skin inspection revealed no insect bite marks. Regarding potential toxics, the patient denied recent use of any other drugs besides her chronic medication, consumption of teas or other herbalist products, alcohol or other drugs or consumption of uncontrolled food or water sources. With no apparent cause for the fever and evidence of hepatic involvement, the patient was admitted in the internal medicine ward. Laboratorial testing was expanded with blood cultures, serology, PCR tests for hepatotropic virus, bacteria and parasites, and liver autoimmune markers (Table 2). All tests were negative, with the exception of blood cultures. *Citrobacter koseri* was isolated and IV piperacillin–tazobactam initiated after 48h of hospital admission.

Although both the physical exam and abdominal ultrasonography were benign, in the absence of another explanation for the fever and in the presence of cholestasis and bacteremia, an abdominal CT scan was obtained. This exam found a left lobe hepatic abscess with gas inside (50 × 41 mm) and a linear hyperdense image, likely a foreign body, piercing through the gastric antrum into the abscess (with arrow, Fig. 1). The abscess was drained through laparoscopic surgery and the foreign body, a 3.5 cm long fishbone, removed (Fig. 2). The patient's condition rapidly improved post-surgical drainage and piperacillin-tazobactam was discontinued 6 days post abscess drainage (total of 8 days of antibiotic therapy). The patient had no recollection of ingesting the fishbone, but it resembled a codfish fish bone, a fish the patient ate at Christmas's and New Year's Eve, as a traditional meal in our country, one week before the first spike of fever.

Discussion

Ingestion of foreign bodies is common but mostly benign as less than 1% of ingested foreign bodies perforate the bowel. Fish bones are the most common cause of perforation [3,4]. Perforation may

Table 1
Initial laboratorial findings.

Parameter	Value	Ref. Value	Parameter	Value	Ref. Value
Haemoglobin	13.0 g/dL	12.0–16.0	Total Bilirubin	0.81 mg/dL	<1.2
Leucocytes	**19.3 × 10^9 /L**	4.0–11-0	Direct Bilirubin	0.15 mg/dL	<0.4
Platelets	159 × 10^9/L	150–400	AST	**77 U/L**	10–31
CRP	**149.4 mg/L**	<5.0	ALT	**54 U/L**	10–31
SR	**81 mm/1st hour**	0–20	GGT	**818 U/L**	7–32
Creatinine	**1.67 mg/dL**	0.51–0.90	ALP	**512 U/L**	30–120
Urea	**83 mg/dL**	10–50	Albumin	**26.1 g/L**	38.0–51.0
Sodium	135 mEq/L	135–147	Total Proteins	**54.5 g/dL**	64.0–83.0
Potassium	4.7 mEq/L	3.5–5.1	aPTT	31.6 s	24.2–36.4
Chlorum	104 mEq/L	100–106	PT	12.7 s	9.9–13.9
Calcium (total)	2.1 mmol/L	2.0–2.6			

ALP, alkaline phosphatase; **ALT**, alanine aminotransferase; **aPPT**, activated partial thromboplastin time; **AST**, aspartate aminotransferase; **CRP**, C-reactive protein; **GGT**, gamma-glutamyl transferase; **PT**, prothrombin time.

Table 2
Further laboratorial aetiologic investigation.

Parameter	Value	Ref. Value	Parameter	Value	Ref. Value
HBs Ag	0.2	NR < 0.9	Transferrin saturation	21 %	20–50
HBc Ag	0.1	NR < 0.9	C4	20 ng/dL	12–36
HBs Ab	5.7 UI/L	–	C3c	189 ng/dL	83.0–177
HCV Ag	0.1	NR < 0.9	ANAs	Negative	ANAs
HIV (Ag + Ab, HIV 1 + 2)	0.1	NR < 0.9	Anti-dsDNA	< 10.0 UI/mL	< 100.0
CMV Ab IgG	234 AU/mL	R > 6.0	PR3-ANCA	< 2 U/mL	< 20
CMV Ab IgM	Negative	–	MPO-ANCA	< 2 U/mL	< 20
EBV Ab IgM (VCA)	negative	–	Anti-LKM	Negative	–
EBV Ab IgG (Early)	negative	–	AMAs	Negative	–
EBV Ab (EBNA)	3.8 RU/mL	R > 1.1	SMAs	Negative	–
EBV Ab (VCA)	2.5 RU/mL	R > 1.1	Anti-SLA	Negative	–
Ferritin	221 ng/mL	20–250	Anti-LP	Negative	–

Ab, antibodies; **AMAs**, anti-mitochondrial antibodies; **ANAs**, anti-nuclear antibodies; **Ag**, antigen; Anti-dsDNA, anti-double stranded DNA antibodies; **Anti-LKM**, anti-liver kidney microsomal antibodies; **Anti-LP**, anti liver–pancreas antigen antibodies; **Anti-SLA**, anti-soluble liver antigen antibodies; **C3c**, cleaved complement component 3; **C4**, complement component 4; **CMV,** Cytomegalovirus; **EBNA**, Epstein-Barr nuclear antigen; **EBV**, Epstein-Barr Virus; **HIV**, Human Immunodeficiency Virus; **HBs**, hepatitis B surface antibody; **HBc**, hepatitis B core antibody; **IgG**, immunoglobulin G; IgM, immunoglobulin M; **MPO-ANCA**, myeloperoxidase anti-neutrophil cytoplasmic antibodies; **NR,** non-reactive; **PR3-ANCA**, proteinase 3 anti-neutrophil cytoplasmic antibodies; **SMAs**, anti-smooth muscle antibodies; **VCA**, virus capsid antigen; **R,** reactive.

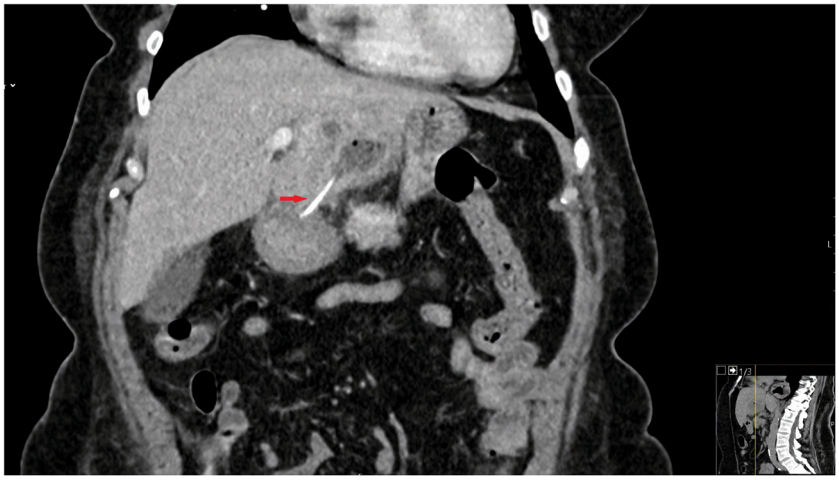

Fig. 1. Abdominal CT-scan showing a linear hyperdense image (highlighted with a red arrow), the foreign body later found out to be a fish bone, piercing through the gastric antrum into the left hepatic lobe abscess (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

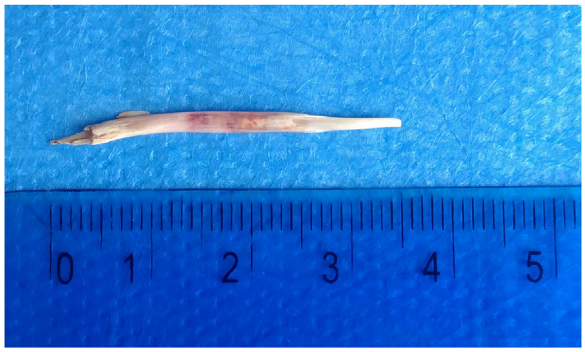

Fig. 2. Fish bone with 3.5 cm excised from the left hepatic lobe abscess during laparoscopic abscess drainage.

occur in any segment from the oesophagus to the rectum; sites with abrupt angulations and caliber transitions, such as the pyloric region, ileum, ileocecal junction and rectosigmoid colon are the most sites for perforation [3,4]. The stomach is a rare location of perforation and tends to occur more frequently in the lesser curvature [7]. Dental prosthetics, alcohol, fast eaters, mental retardation, elderly and infants are known risk factors for fish bone ingestion, mainly due to poor mastication and decreased awareness of sharp elements in the food bolus [4].

Several factors may difficult the diagnosis. Patients generally do not recall ingesting the foreign body and symptom onset may occur several days or even weeks after ingestion. Also, according to the site of perforation, symptoms may vary greatly: patients may present with unspecific abdominal pain, acute abdomen signs, vomiting, fever, melena or bowel obstruction; foreign body perforation may even mimic other entities, such as appendicitis or diverticulitis [4,5,8]. Acute abdomen is not always present; when the perforation is caused by gradual erosion of the wall, fibrin, inflammatory tissue and omental tamponade stop the leakage of air and fluids to the peritoneum, preventing acute abdomen [4,5]. In these atypical cases without acute abdomen or other typical signs, the diagnosis maybe until complications (bacteraemia, local abscess formation) occur [5,6]. Migration of the foreign body to other organs may also occur, and gastric or duodenal perforation may lead to hepatic abscess, usually in the left hepatic lobe [5,9].

Conventional abdominal radiograph rarely detects fish bones since they are radiolucent and ultrasonography is also not suitable for fish bone detection. CT-scan is the most sensitive method as fish bones appear as a thin, linear, high-density structure together with local inflammatory signs. Caution must be made regarding the interpretation of contrast-enhanced CT scans; if slice thickness is greater than 3 mm, small fishbones may be overlooked between slices given their small width or mistaken for small vessels due to low resolution and volume averaging [5,9]. When perforation is confirmed, laparoscopic surgery is the preferential approach, allowing the removal of the foreign body, abscess drainage and repair of the perforation site. In selected cases, if the perforation is contained by the gastric wall, endoscopic retrieval of the foreign body and clipping of the perforation site may be possible [4,5].

In this particular case, perforation occurred at the gastric antrum, an unusual site for bowel perforation. The absence of an acute abdomen hints for a gradual erosion of the gastric wall covered by fibrin, inflammatory tissue and omentum. Inflammation provoked adherence of the small curvature to the left hepatic lobe and migration of the fish bone, resulting in a hepatic abscess. Ultrasound failed to identify any abnormalities. As previously stated, ultrasound is not suitable to identify fishbones; also, abscess was not visualized due to patient's body habitus. The unremarkable physical exam at admission, unsuspicious ultrasound and history of prolonged fever made us approach etiologic investigation like a case of FUO. We broadened the investigation to search for infections, occult neoplasia, lymphoproliferative and autoimmune diseases as possible aetiologies. Among classic infectious causes for FUO, military tuberculosis, brucellosis and Q fever are the most common in case series reports, followed by intra-abdominal abscesses, like in this case [1,2].

Conclusions

Unknown ingestion of foreign bodies leading to perforation can pose a clinical challenge. Perforation site may occur in any segment, resulting in a broad spectrum of clinical presentations, from the acute abdomen to a more insidious clinical presentation when the perforation is contained. Contained perforations tend to complicate with local abscess formation or migration to adjacent organs, where an abscess may also be formed. These cases may present as prolonged fever leading physicians to approach the case as FUO. CT imaging is the best method for both the diagnosis of bowel perforation by a foreign body. When perforation is confirmed, laparoscopic removal of the foreign body, abscess drainage and repair of the perforation site is the preferred approach.

Conflict of interest

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Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editorin-Chief of this journal on request.

Ethical approval

The authors declare that compliance with ethical standards was a concern during all steps of the elaboration of this article, according to the practices endorsed by the hospital ethical committee.

Author contribution

All listed authors contributed directly in either some part of the patient in hospital care or in the manuscript draft and/or revision. JES and SP first received the patient in the ward and conducted the diagnostic workup, under orientation of MPatacho. Initial draft and bibliographical research for the manuscript was made by JES and MPacheco. Final approval and revision of the submitted version was done by MPatacho and JA.

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