Delay in diagnosis and lessons learnt from a case of abdominal wall abscess caused by fishbone perforation

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ABSTRACT

Complications following foreign body (FB) ingestion are an uncommon clinical problem. A 59-year-old man presented with a 4-week history of left iliac fossa pain and 1 episode of dark red blood mixed with stools. Inflammatory markers were elevated, and computed tomography (CT) of the abdomen and pelvis showed an ill defined abdominal wall inflammatory collection in close contact with the small bowel loops. He was treated with antibiotics, and follow-up CT, colonoscopy and small bowel enema were mostly unremarkable.

The patient presented again ten months later with left iliac fossa cellulitis and fever. Multiplanar CT (the patient's fourth scan) demonstrated a 10cm abdominal wall collection with a linear hyperdense structure in the collection. The radiologists suspected a FB and on close scrutiny of the previous scans, they noted it to have been present on all of them. A targeted incision led to the removal of a 3cm fishbone from the collection. This case highlights the need to consider the possibility of a FB being the underlying cause in any unexplained intra-abdominal or abdominal wall inflammatory process so that the diagnosis is made in a timely manner.

KEYWORDS

Fishbone – Anterior abdominal wall – Intestinal perforation – Computed tomography – Foreign body – Multiplanar

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Ingested foreign bodies (FBs) usually pass through the gastrointestinal tract without complications. FBs such as fishbones, chicken bones and toothpicks have been reported to cause gastrointestinal perforation following ingestion.¹ These perforations can present either as an acute abdomen or later as an intra-abdominal collection. We present a case of anterior abdominal wall abscess caused by fishbone perforation. This has not been reported previously in the UK.

Case History

A 59-year-old man presented with stabbing left iliac fossa pain over a period of 4 weeks. He also had a three-week history of constipation and one episode of dark red blood mixed with stools. He did not complain of any history of loss of weight or loss of appetite. He was pyrexial at presentation and abdominal examination was unremarkable except for slight tenderness in the left iliac fossa on deep palpation. He did not have any other past medical history. He was a non-smoker and took alcohol rarely. There was no family history of bowel or other malignancy.

The inflammatory markers were elevated at presentation and the rest of the blood tests were unremarkable.

Computed tomography (CT) of the abdomen and pelvis showed an ill defined abdominal wall inflammatory collection in intimate contact with small bowel loops. The initial differential diagnoses included complicated diverticulitis, small bowel localised perforation due to inflammatory bowel disease and malignant perforation of the large/small bowel.

After consideration of the clinical picture, a conservative approach was adopted and the patient treated with a course of antibiotics, with resolution of the symptoms. He was discharged home well and follow-up CT six weeks later showed improvement in the appearances but there was a small fluid collection in the anterior abdominal wall with a linear density of uncertain significance adjacent to it. He remained asymptomatic and further imaging three months later showed only minimal abnormality. As the cause of the abscess remained obscure, colonoscopy and small bowel follow-through were performed but these were normal apart from mild diverticular disease.

The patient presented again six months later (ten months after the initial presentation) with a recurrence of left iliac fossa pain and fever. Clinical examination revealed a tender mass in the left mid/lower abdomen,

Figure 1 Axial computed tomography at initial presentation showing inflammatory change close to the small bowel loops but the fishbone is barely visible as tiny dot of high attenuation within it (arrow)

with inflammation of the overlying skin. CT showed a 10cm inflammatory change in the abdominal wall containing a large central fluid collection and a linear hyperdense structure in the collection. The radiologists suspected a FB and on close scrutiny of the previous scans, they noted it to have been present on all of them. It had migrated progressively towards the abdominal wall (Figs 1–3). Ultrasonography at this time confirmed the presence of a fishbone-like structure in the superficial collection (Fig 4) and its position was marked on the skin.

A targeted incision led to the removal of a 3cm fishbone from the abdominal wall collection (Figs 5 and 6). Histological examination of the tissue from the abscess cavity confirmed inflammatory granulation tissue only. The patient went on to make a full recovery. The scar from the abscess drainage healed completely and he did not have any recurrent symptoms.

Discussion

This case illustrates a rare delayed presentation of fishbone perforation of the small bowel. It highlights the need to consider the possibility of a FB being the underlying cause in any case of unexplained intra-abdominal or abdominal wall inflammatory process so that such FBs are identified and treated in a timely manner.

Accidental ingestion of fishbone is not uncommon but despite the sharp ends, the majority pass through the gastrointestinal tract uneventfully and perforation is rare.¹ They may on occasion, however, present in various ways from upper gastrointestinal impaction, dysphagia, bowel

Figure 2 Retrospective computed tomography coronal reconstruction showing the fishbone clearly in the inflammatory tissue (arrow)

obstruction or silent perforation to frank peritonitis. The clinical history may not be helpful, and establishing the cause is difficult because the episode of swallowing the bone frequently goes unnoticed and may have occurred several days or weeks previously.²

Our patient denied any history of FB ingestion. It was the recurrent episodes that led to careful scrutiny of the previous imaging, which eventually identified the culprit FB. This had been mistaken for a possible blood vessel on the initial imaging.

Figure 3 Computed tomography coronal reconstruction prior to surgery: The fishbone is seen clearly in the pus collection in the left iliac fossa (arrow).

Figure 4 Ultrasonography confirming the presence of a fishbone-like structure in the superficial collection (arrow). Ultrasonography was used to mark the location of the foreign body so as to guide with abdominal wall incision.

It is likely that the FB perforated the small bowel when the patient presented initially with abdominal pain and dark red rectal bleeding. This might have happened in a gradual manner owing to the size of the fishbone (3cm) and, consequently, the patient did not present with frank peritonitis.

Figure 5 Fishbone removed from the anterior abdominal wall collection (held in the tips of the curved clip)

Figure 6 3cm long fishbone removed from the anterior abdominal wall collection

The gradual movement of the fishbone would have allowed the bowel to seal off slowly over time, without causing any obvious findings on CT or small bowel follow-through. This inflammatory area would have adhered subsequently to the abdominal wall, resulting in collection, with gradual migration of the FB towards the path of least resistance, aided by intra-abdominal pressure driving it towards the skin.

There have been previous reported cases of such 'pseudotumoural' or 'inflammatory' masses in the anterior abdominal wall secondary to fishbone migration.⁵ Most of these cases were described in fish eating countries (eg China) and no such cases have been reported in the UK.¹ Bhatia *et al* reported a case of terminal ileal perforation in an 85-year-old patient due to fish fin ingestion.⁴ The patient was initially managed conservatively and, owing to worsening sepsis, needed laparotomy and terminal ileal enterotomy with removal of the offending FB. The initial CT of the abdomen and pelvis on this patient was reported incorrectly as colonic diverticulitis. Similar to our case, the small bowel FB in the report by Bhatia *et al* was demonstrated on the retrospective review of the initial scan.⁴

Radiography is often unrewarding in the diagnosis of fishbone perforation because of the minimal calcium content and the high kilovoltage of the x-ray beam. Even when fishbones are sufficiently radiopaque to be visualised on radiography, large soft tissue masses and fluid can obscure the minimal calcium content of the bone. An absence of free air in the peritoneal cavity can also obscure the aetiology.⁵

CT is frequently helpful in the detection of non-metallic FBs.⁵ The CT findings in FB perforation include the identification of the FB, localised pneumoperitoneum, thickened bowel wall, local soft tissue infiltration of fat, abscess or fluid collection and bowel obstruction. The main drawbacks of CT in the detection of non-metallic FBs relate to lack of observer awareness or clinical suspicion, the thickness of CT slices and the orientation of the FB with respect to the CT slices. Identification is far more readily achieved with modern multidetector scanners, which have substantially improved resolution over older machines, and with instant multiplanar reconstructions such FBs can be readily identified once suspected.⁵ Our patient's FB was identified only on the fourth CT scan (using a 64-slice scanner),

nearly a year after the initial presentation, despite it being visible in retrospect on all of the imaging.

Conclusions

Ingested fishbone induced intestinal perforation or secondary collections are seldom diagnosed preoperatively or at first presentation owing to incomplete patient history taking and difficulties in imaging evidence identification. In any case of unexplained intra-abdominal or abdominal wall inflammatory process, FBs should be considered by the clinicians and radiologists so as to diagnose the problem in a timely manner.

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