contrast enhancement, fat densification, adjacent fluid collections, or free fluid[3].

The definitive treatment for MD is surgery, which is always indicated in symptomatic patients. The approach can be by laparoscopy or laparotomy, which provide equally satisfactory results[12].

The data presented here underscore the importance of diagnostic suspicion for the identification of Meckel's diverticulitis. In patients with nonspecific abdominal symptoms, radiologists must be familiar with the imaging aspects of MD in order to interpret the imaging studies correctly.

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Subacute cortical infarct: the value of contrast-enhanced FLAIR images in inconclusive DWI

Dear Editor,

A 44-year-old patient presented with axial sensorimotor deficit, dating back approximately 10 days. The history was significant for diabetes, alcoholism, and cognitive impairment, making it difficult to assess the recent history and symptoms. The patient was submitted to brain magnetic resonance imaging (MRI) with T2-weighted imaging (T2WI), fluid-attenuated inversion recovery (FLAIR) sequences, susceptibility weighted imaging, and diffusion-weighted imaging (DWI), as well as T1-weighted imaging (T1WI), before and after intravenous gadolinium administration, in the axial, sagittal, and coronal planes. In the right postcentral gyrus, MRI revealed a cortical lesion, which showed a hyperintense signal on FLAIR image (Figure 1A). The lesion could be due to a new or older infarct. However, there was no restricted diffusion suggestive of a recent infarct (Figures 1B and 1C). Contrast-enhanced FLAIR imaging revealed marked cortical enhancement in the right postcentral gyrus, consistent with a subacute cortical infarct (Figure 1D).

Diabetes mellitus is a well-recognized risk factor for ischemic stroke, which is a leading cause of death and disability. MRI is quite sensitive in detecting ischemic changes. T2WI is more sensitive than is T1WI, and T1WI after gadolinium administration can provide valuable information for the accurate diagnosis[1,2]. Intravascular enhancement, although not specific, is considered a sign of ischemia on conventional MRI. Contrast enhancement

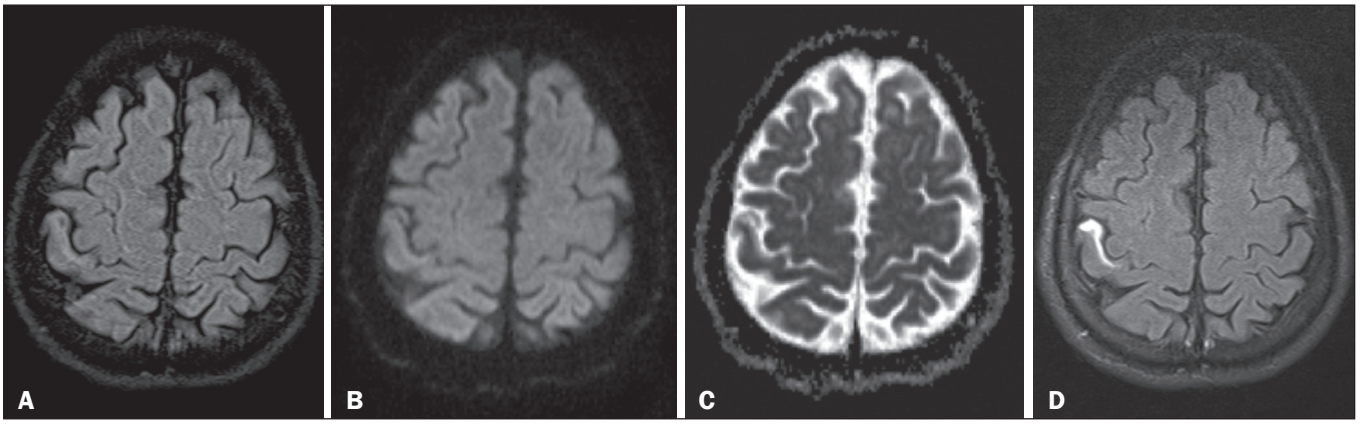

Figure 1. A: Axial FLAIR image. Note the subtly hyperintense signal in the anterior cortex of the postcentral gyrus. **B,C:** Corresponding axial DWI (**B**) and ADC map (**C**). There is a barely discernible hyperintense signal on DWI, although there is no evidence of low signal intensity on the ADC map (i.e., there is no restricted diffusion in the region). **D:** Gadolinium-enhanced FLAIR image showing marked contrast uptake in the affected area of the postcentral gyrus.

in the central nervous system is the result of a combination of disruption of the blood-brain barrier, high vascularity, and contrast leakage into the lymphatic system[3–6]. After one week, infarcts show parenchymal enhancement, due to breakdown of the blood-brain barrier[7].

New imaging techniques, such as DWI and perfusion-weighted imaging, have increased the accuracy of the diagnosis of acute cerebral ischemia, although there are some cases in which it cannot be distinguished from other entities[8,9]. In addition, because of pseudonormalization, subacute infarcts may not show restricted diffusion on DWI.

FLAIR is highly sensitive for the detection of ischemic lesions. Although it is considered to be heavily T2-weighted, rendering cerebrospinal fluid as dark, it also shows mild contrast enhancement on T1WI, which is responsible for the increased conspicuity of gadolinium enhancement. Pathologic conditions that present contrast enhancement on T1WI usually show marked enhancement on contrast-enhanced FLAIR[10]. This is exactly what occurred in the case presented here, in which DWI pseudonormalization did not help reveal the subacute cortical infarct. When a subacute cortical infarct is suspected, delayed contrast-enhanced FLAIR imaging is the best choice for demonstrating the lesion and for differentiating it from an older lesion with gliosis.

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Transmural migration of a gossypiboma: a rare cause of intestinal obstruction

Dear Editor,

An 83-year-old man presented to the emergency department with an approximately one-month history of diffuse abdominal pain, nausea, bilious vomiting, and abdominal distension, the symptoms worsening in the last three days. He reported having lost 6 kg since the onset of symptoms. Six months prior, he had undergone cholecystectomy at another facility. On physical examination, the abdomen was slightly rounded, with increased bowel sounds, and was painful to superficial palpation in the mesogastrium. No organomegaly or palpable masses were observed. Upper gastrointestinal endoscopy showed gastric antral vascular ectasia, with a large amount of undigested food. The pylorus was lateralized, retracted, and stenotic, which precluded the passage of the endoscope into the duodenal bulb. An abdominal X-ray (Figure 1A) showed marked dilation of the gastric antrum, with an air-fluid level and serpiginous radiopaque areas in the duodenal region, characteristic of a foreign body (gossypiboma). An abdominal CT scan with intravenous contrast administration (Figures 1B and 1C) confirmed the X-ray findings and better characterized the intraluminal mass in the first portion of the duodenum, showing metallic wires within the mass and confirming upper gastrointestinal obstruction, as well as enhancement of the duodenal and gastric walls, probably due to an inflammatory reaction. There were no signs of pneumoperitoneum or cavitary fluid collections/abscesses. The patient underwent laparotomy, with laparoscopic suture closure of the duodenum and jejunostomy for feeding access. The presence of a foreign body (gossypiboma) was confirmed intraoperatively (Figure 1D). The gossypiboma, which was located in the first portion of the duodenum, resulted in gastric outlet obstruction and gastric dilatation.

Acute abdominal conditions have been the subject of various recent studies in the radiology literature of Brazil[1–4]. Gossypibomas have been identified in 0.02–0.1% of patients undergoing abdominal surgery[5]. Transmural migration of a gossypiboma is extremely rare. When it does occur, it is typically in the bowel, bladder, or chest. Spontaneous expulsion of a gossypiboma has been reported in only a few cases, the mean time from surgery to diagnosis being 2.2 years[5,6].

Two types of reactions to foreign bodies have been described in the literature: fibroblastic and exudative. An aseptic fibrous response results in adhesion, encapsulation, and granuloma, usually remaining asymptomatic or causing chronic progressive symptoms over months to years. An exudative reaction causes the formation of a cyst or abscess that can establish fistulas to adjacent organs, the symptoms being more severe in such cases[5,7]. The increase in intra-abdominal pressure caused by a gossypiboma can result in partial or total necrosis of the intestinal wall[6,7]. The risk factors associated with the increase in the incidence of gossypiboma include emergency surgical procedures, prolonged surgical procedures, unplanned changes in the course of a procedure, the involvement of more than one surgical team, and a higher patient body mass index[7].

The imaging findings preceding transmural migration of a gossypiboma are variable, depending on the nature of the sponge, its radiopaque marker, the length of time the foreign body has been present, and the type of reaction to it. A CT scan can reveal a poorly defined, heterogeneous mass, containing metallic wires