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Double Mimicry: Chronic Diverticulitis and Post-pancreatitis Fat Necrosis Mimicking Metastatic Colonic Cancer

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Abstract

Inflammatory intra-abdominal conditions can occasionally mimic advanced malignancy. Post-pancreatitis fat necrosis masquerading as peritoneal carcinomatosis is a rare entity. We report a case of “double mimicry” in which severe diverticulitis and post-pancreatitis fat saponification simultaneously simulated a metastatic colonic neoplasm.

A 59-year-old man with a recent episode of severe acute necrotizing pancreatitis presented with large bowel obstruction. Computed tomography (CT) and sigmoidoscopy showed an abrupt sigmoid stricture with irregular, friable mucosa, suggestive of an obstructing carcinoma. At emergency laparotomy, a hard sigmoid mass and widespread solid omental and peritoneal nodules resembling carcinomatosis were found. A diverting loop transverse colostomy was performed, and the peritoneal nodules were biopsied.

Histopathology of the nodules showed fat necrosis, saponification, and calcification, with no malignancy, and endoscopic biopsies showed only inflammation. Tumor markers were normal. Considering these findings and a prior colonoscopy documenting left-sided diverticulosis, a benign etiology was favored, and the patient underwent elective laparoscopic sigmoidectomy. Final pathology confirmed severe chronic diverticulitis with reactive fibroblastic proliferation and no neoplasia.

This case illustrates how two concurrent benign processes can create a convincing picture of metastatic colonic cancer. Integrating the patient’s full history and obtaining histopathological confirmation are essential to avoid erroneous terminal staging and to allow definitive, curative treatment.

Categories: General Surgery, Pathology
Keywords: diverticulitis, fat necrosis, intestinal obstruction, pancreatitis, peritoneal neoplasms

Introduction

Benign intra-abdominal inflammatory conditions can occasionally present with clinical, radiological, and macroscopic features that mimic advanced malignancy [1]. Peritoneal carcinomatosis most commonly arises from gastrointestinal and ovarian primaries, and its detection usually implies advanced, often incurable disease; therefore, a false-positive diagnosis can lead to inappropriate staging and abandonment of curative treatment [2]. Differentiating non-neoplastic processes from true neoplastic dissemination is a significant diagnostic challenge that requires careful multidisciplinary evaluation [1,3].

Severe acute pancreatitis, particularly when necrotizing, triggers an enzymatic cascade in which lipases released into the peritoneal cavity hydrolyze extrapancreatic triglycerides, causing fat necrosis, saponification and subsequent calcification within the peritoneal cavity, mesentery and omentum [4-6]. At surgical exploration or on cross-sectional imaging, these solid necrotic nodules, which form locally rather than through transcoelomic spread, can masquerade as metastatic peritoneal deposits [3,7]. This presentation is a recognized but rare entity, described in the surgical literature as a “surgical trompe l’oeil” [8].

Independently, chronic diverticular disease of the sigmoid colon can produce localized strictures, mucosal friability and abrupt luminal narrowing that closely resemble a primary obstructing colonic neoplasm, and the two entities are often difficult to distinguish on imaging [9,10]. Their simultaneous occurrence in a single patient, however, is exceptionally rare.

Case Presentation

A 59-year-old man presented to the emergency department with large bowel obstruction, reporting severe abdominal pain and obstipation. Four months earlier, he had been admitted to the Intensive Care Unit with

severe acute necrotizing pancreatitis of biliary etiology (Bedside Index for Severity in Acute Pancreatitis (BISAP) score of 3 [11]), complicated by type 2 respiratory failure, mild thrombocytopenia, and acute kidney injury (Kidney Disease: Improving Global Outcomes (KDIGO) stage 1 [12]). A CT scan during that admission showed a heterogeneous, swollen pancreas with peripancreatic fat stranding and a fluid collection containing gas bubbles (Figure *1*). His medical history also included a prior non-ST-elevation myocardial infarction treated with percutaneous coronary intervention, dyslipidemia, hypertension, and obstructive sleep apnea. A previous colonoscopy had documented numerous diverticula in the left colon.

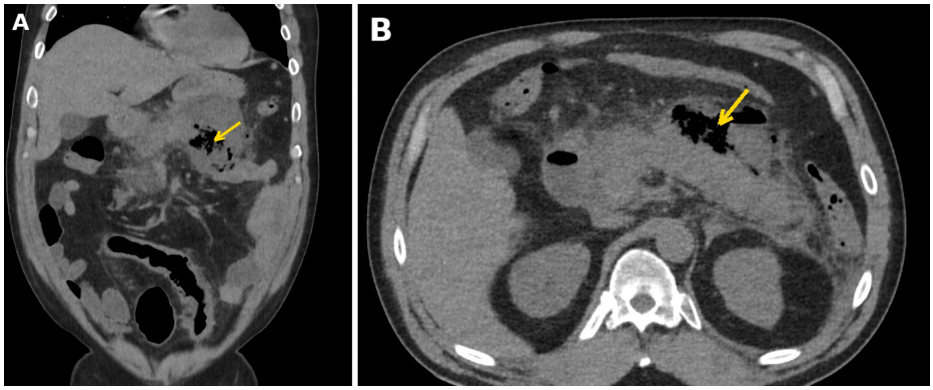


FIGURE 1: Computed tomography during the admission for acute necrotizing pancreatitis.

(A) Coronal and (B) axial images showing a heterogeneous, swollen pancreas with extensive peripancreatic fat stranding and a peripancreatic collection containing gas bubbles (arrows).

On admission, the patient was in marked distress, with severe abdominal pain and no position of relief. Blood pressure was 150/93 mmHg, heart rate 109 beats per minute, and peripheral oxygen saturation 97%, and he was afebrile. The abdomen was distended, tympanic, and tender to palpation, particularly in the epigastrium, with guarding and absent bowel sounds. Digital rectal examination revealed normal sphincter tone and soft brown stool in the rectal ampulla. Laboratory tests showed leukocytosis and an elevated C-reactive protein, with mildly elevated urea and amylase, and normal creatinine, liver enzymes, lipase, and lactate. Tumor markers (carcinoembryonic antigen (CEA) and carbohydrate antigen 19-9 (CA 19-9)) were within normal limits (Table *1*).

Parameter	Patient value	Reference range	Unit
White blood cell count	16.3	4.0-10.0	×10³/μL
Hemoglobin	12.1	12.0-17.7	g/dL
Platelets	260	150-500	×10³/μL
C-reactive protein	22.6	<5	mg/L
Urea	8.6	2.8-7.2	mmol/L
Creatinine	0.89	0.67-1.18	mg/dL
Alanine aminotransferase	7	3-45	U/L
Aspartate aminotransferase	16	15-50	U/L
Amylase	92	22-80	U/L
Lipase	50	22-67	U/L
Sodium	137	136-146	mmol/L
Potassium	4.1	3.6-5.1	mmol/L
Lactate	0.7	0.5-1.6	mmol/L
Carcinoembryonic antigen	0.7	<3	ng/mL
Carbohydrate antigen 19-9	2.7	<35	U/mL

TABLE 1: Laboratory findings on admission.

On presentation, an abdominal radiograph and CT scan showed significant colonic distension, with focal thickening of the proximal sigmoid colon causing an abrupt change in caliber, and a small omental nodule suspicious for a peritoneal metastasis (Figure *2*). Urgent sigmoidoscopy revealed a luminal stricture approximately 30 cm from the anal verge, with irregular, nodular, highly friable mucosa and an irregular vascular pattern, highly suspicious for malignancy. The endoscope could not be advanced beyond this point, and biopsies were obtained.

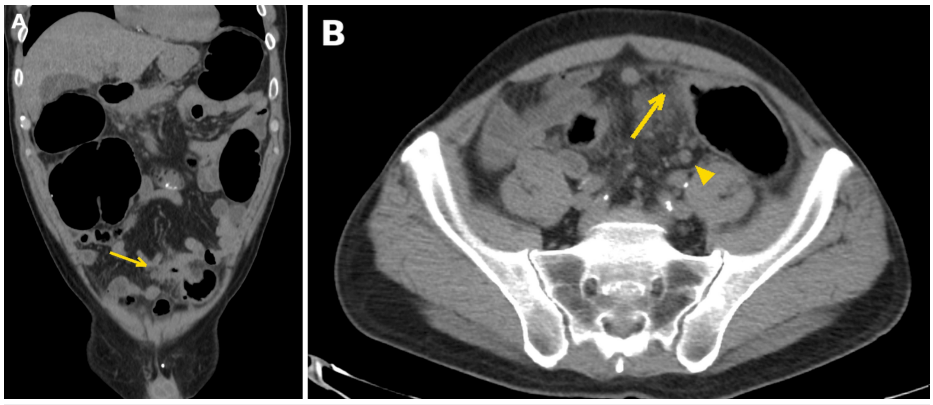


FIGURE 2: Computed tomography at presentation with large bowel obstruction.

(A) Coronal image showing the sigmoid mass (arrow) causing obstruction of the upstream colon. (B) Axial image showing the sigmoid mass (arrow) and a small omental nodule (arrowhead), initially interpreted as a peritoneal metastasis; biopsies of the omental nodules later revealed fat necrosis.

Given the obstructive presentation and the strong suspicion of an occlusive sigmoid tumor, the patient underwent emergency laparotomy. Exploration revealed a hard, neoplasm-like mass in the sigmoid colon. Widespread, solid, whitish nodules were scattered across the left paracolic gutter, the ileocecal region, the greater omentum, and the transverse mesocolon, macroscopically indistinguishable from multifocal

peritoneal carcinomatosis. Intraoperative frozen section analysis was not available in this out-of-hours emergency setting. Given the presumed peritoneal carcinomatosis, a diverting loop transverse colostomy was performed to relieve the obstruction, with the intention of referring the patient for systemic chemotherapy and evaluation for cytoreductive surgery and hyperthermic intraperitoneal chemotherapy (HIPEC), rather than performing an oncological resection in the emergency setting. Biopsies of the omental and ileocecal nodules were obtained.

The endoscopic biopsies showed fragments of granulation tissue with exuberant polymorphic inflammatory infiltrate and no evidence of neoplasia. Histopathological analysis of the peritoneal nodules showed adipose tissue with connective tissue septa, foci of fat necrosis, histiocytic infiltration, and calcifications, consistent with fat saponification secondary to the previous necrotizing pancreatitis, with no malignant cells (Figure *3*).

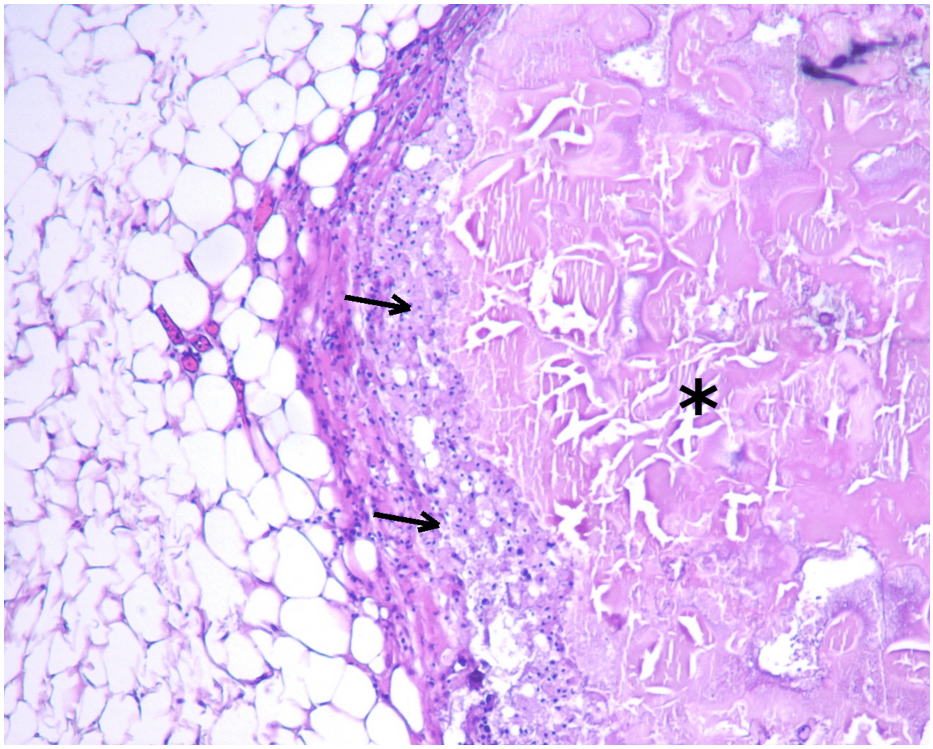


FIGURE 3: Histopathology of an omental nodule (hematoxylin and eosin, ×40).

Fat necrosis with saponification (asterisk), surrounded by a rim of foamy histiocytes and fibrosis (arrows), adjacent to viable adipose tissue.

Further workup was undertaken. Repeat lower endoscopy, performed after the index operation, again showed a severe, ulcerated, friable circumferential stricture (Figure *4*), but multiple repeat biopsies showed only ulceration and inflammation. Staging CT and abdominal MRI showed segmental thickening of the sigmoid colon with adjacent fat stranding and reactive lymph nodes, without distant metastases.

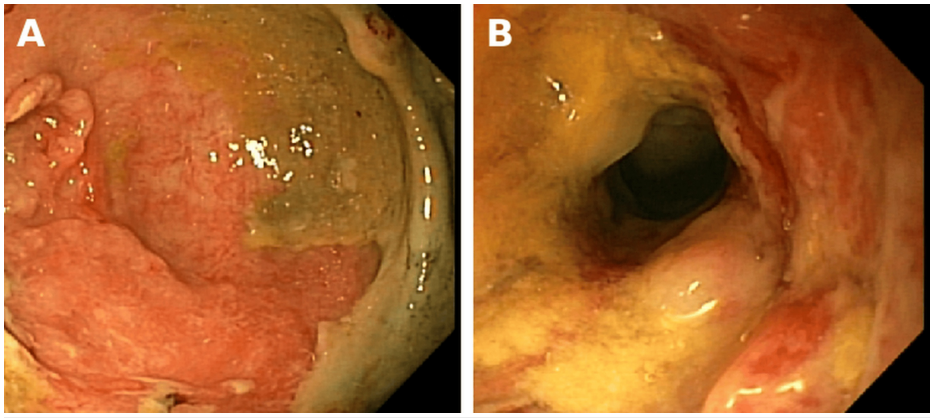


FIGURE 4: Colonoscopy performed after the index operation.

(A) Distal aspect of the sigmoid stricture showing irregular, ulcerated and friable mucosa with fibrinous exudate.
(B) Central aspect of the stricture showing circumferential luminal narrowing with ulcerated, inflamed mucosa.

Following multidisciplinary discussion favoring a benign etiology, but given the persistent symptomatic stricture and the impossibility of completely excluding malignancy, the patient underwent elective laparoscopic sigmoidectomy two months after the index operation. Final pathology confirmed severe chronic diverticulitis with prominent reactive fibroblastic proliferation (Figure 5). No neoplastic cells were identified, and all lymph nodes were reactive. The patient recovered uneventfully and later underwent elective laparoscopic cholecystectomy and colostomy closure. He remains well at three months of follow-up.

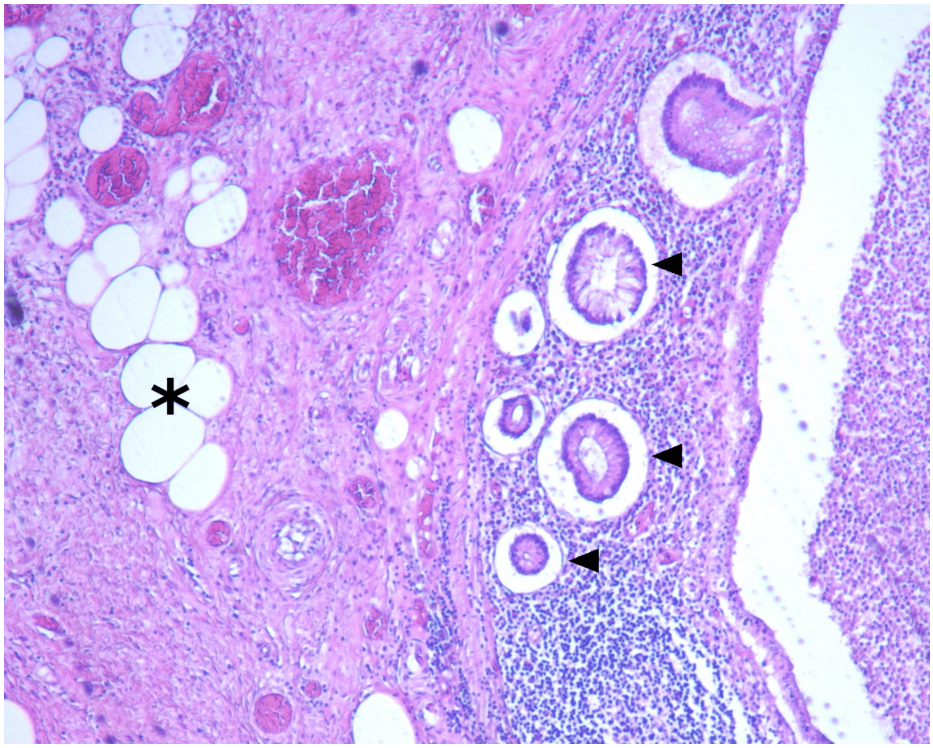


FIGURE 5: Histopathology of the sigmoidectomy specimen (hematoxylin and eosin, ×40).

Diverticulum with benign colonic mucosa (arrowheads) extending into the pericolic adipose tissue (asterisk), with dense chronic inflammation and fibrosis and no evidence of neoplasia.

Discussion

Differentiating benign intra-abdominal inflammatory conditions from advanced malignancy remains a challenge in both surgical and radiological practice [1,2]. In severe acute pancreatitis, enzymatic

degradation of extrapancreatic adipose tissue results in fat necrosis, saponification, and eventual calcification [4,5]. These nodules do not result from transcoelomic spread; rather, they form locally wherever the omental, mesenteric and peritoneal fat is exposed to the enzymes and inflammatory mediators released from the inflamed pancreas, particularly lipase. When widespread, they can closely resemble peritoneal carcinomatosis at surgical exploration [7], and may lead the surgical team to assume a terminal oncological diagnosis [8]. In our patient, the recent episode of severe necrotizing pancreatitis provided the pathophysiological basis for the diffuse peritoneal nodules found at laparotomy.

The differential diagnosis of multiple peritoneal nodules and solid omental masses is broad. Malignant causes include peritoneal carcinomatosis from gastrointestinal, ovarian, or pancreatic primaries, primary peritoneal tumors such as mesothelioma, and peritoneal lymphomatosis [2]. Several benign entities can produce a similar picture [1,2]. Peritoneal tuberculosis may present with ascites, peritoneal thickening, and omental or mesenteric nodules; smooth, regular peritoneal thickening with marked enhancement, low-attenuation or calcified lymph nodes, and ileocecal involvement favor tuberculosis, and histology shows granulomatous inflammation [2,13]. Sclerosing mesenteritis typically appears as a “misty” mass at the mesenteric root, often with a fat halo around vessels and nodes and a peripheral pseudocapsule [1]. Endometriosis shows hemorrhagic foci that are hyperintense on T1-weighted magnetic resonance imaging and is confirmed by endometrial glands and stroma on histology [2]. Splenosis enhances like normal splenic tissue and occurs after splenic trauma or splenectomy, whereas foreign-body granulomas arise near previous surgical sites and contain multinucleated giant cells [2].

Some features can help distinguish fat saponification from true metastatic deposits. On imaging, post-pancreatitis fat necrosis tends to appear after a documented episode of pancreatitis, shows a fatty or heterogeneous center, sometimes with a peripheral rim or calcifications, displays absent, weak, or delayed enhancement, and may regress on follow-up, whereas metastatic deposits are typically solid, enhancing soft-tissue nodules and are often associated with ascites and a detectable primary tumor [4,6]. On histology, fat necrosis shows anucleate “ghost” adipocytes, basophilic calcium soap deposits, foamy histiocytes, and fibrosis, without atypical cells, whereas metastases contain malignant epithelial cells, frequently within a desmoplastic stroma [6,14]. These features were present in our patient’s biopsies (Figure *3*). However, none of these imaging findings are entirely specific, and tissue diagnosis remains the reference standard [3,15].

Post-pancreatitis fat necrosis mimicking peritoneal carcinomatosis has been reported mainly as isolated case reports. Smith et al. described post-pancreatitis fat necrosis with an imaging appearance suggestive of carcinomatosis, and Tan et al. reported diffuse peritoneal nodules found at surgery after acute gallstone pancreatitis, coining the term “surgical trompe l’oeil” [7,8]. Moldovanu et al. described a 50-year-old man with alcohol-induced pancreatitis and a large omental mass initially suspected to be a liposarcoma, which proved to be fat necrosis after omentectomy [14]. Mejia et al. reported a 14-year-old girl with biliary pancreatitis in whom widespread peritoneal micronodules found during laparoscopic cholecystectomy were confirmed as fat necrosis on biopsy [6], and further cases with similar radiological presentations have been described recently [3,5]. In most of these reports, the diagnosis was only established after surgical exploration or tissue sampling. To our knowledge, the concurrence of this entity with an obstructing diverticular pseudotumor of the sigmoid colon has not been previously described.

Complicating the picture, our patient simultaneously had an obstructing sigmoid mass that appeared malignant on endoscopic and intraoperative evaluation. The differential diagnosis of a sigmoid stricture includes adenocarcinoma, chronic diverticular disease, ischemic stricture, inflammatory bowel disease, endometriosis in women, and extrinsic compression [9,10]. Chronic diverticular disease is a well-known mimic of sigmoid carcinoma, producing fibrotic strictures, mucosal friability, and reactive lymphadenopathy; imaging features overlap considerably, and a proportion of presumed diverticular masses prove to be cancer, and vice versa [9,10]. A colonic stricture related to the previous necrotizing pancreatitis was also considered; such strictures result from ischemia or spread of the peripancreatic inflammatory process and usually involve the splenic flexure, while more distal involvement is exceptionally rare [16]. The sigmoid location, the previously documented left-sided diverticulosis, and the final histology supported a diverticular origin in our patient. When the diverticular pseudotumor coexisted with diffuse fat saponification, the combined presentation strongly suggested metastatic colonic cancer.

This diagnostic trap directly influenced the initial surgical strategy. In the presence of apparent peritoneal carcinomatosis, a diverting stoma was chosen to relieve the obstruction and allow prompt referral for systemic therapy and evaluation for cytoreduction. Upfront resection of the primary tumor has not shown a survival benefit in patients with synchronous unresectable metastases [17], and in potential candidates for cytoreductive surgery, resection can be performed at the time of cytoreduction. This coincidence is unlikely to be recognized pre-operatively, as no clinical, laboratory, or imaging feature reliably distinguishes it from malignancy, and in our patient it was established only through tissue diagnosis. In retrospect, intraoperative frozen section of the peritoneal nodules could have clarified the diagnosis and allowed a definitive resection at the index operation; when available, it should be considered whenever suspected carcinomatosis is found in a patient with a history of severe pancreatitis.

Ultimately, integrating the patient’s history, the prior colonoscopy findings and definitive tissue biopsies, together with multidisciplinary discussion, prevented a permanent misdiagnosis and allowed curative

treatment. Surgeons should maintain a high index of suspicion for fat necrosis mimicking carcinomatosis in any patient with recent severe pancreatitis, and definitive management should be guided by histopathological confirmation.

Conclusions

This rare case of "double mimicry" illustrates the diagnostic challenges that arise when separate benign inflammatory processes simultaneously masquerade as advanced metastatic cancer. The concurrence of chronic diverticulitis and post-pancreatitis fat saponification created a near-perfect illusion of obstructing colonic carcinoma with peritoneal carcinomatosis. In the emergency setting, when histological results are not yet available, surgical management should aim to resolve the acute complication while preserving future curative options. Definitive oncological staging should only be established after histopathological confirmation and multidisciplinary discussion, particularly in patients with a recent history of severe pancreatitis.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Patrícia Bárbara, Sónia Martins, Maria Cunha, Paulo Clara, Ana Rodrigues

Acquisition, analysis, or interpretation of data: Patrícia Bárbara, Sónia Martins, Maria Cunha, Paulo Clara, Ana Rodrigues

Drafting of the manuscript: Patrícia Bárbara

Critical review of the manuscript for important intellectual content: Patrícia Bárbara, Sónia Martins, Maria Cunha, Paulo Clara, Ana Rodrigues

Supervision: Sónia Martins, Maria Cunha, Paulo Clara, Ana Rodrigues

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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