BJCR
Brazilian Journal of Case Reports

PCAA
Publicações Científicas de Acesso Aberto

Case Report

Sclerosing Mesenteritis: Case Report of a Malignancy Mimicker

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Abstract: Sclerosing mesenteritis (SM) is a rare, non-malignant, disorder marked by mesenteric chronic inflammation and fibrosis. We present a case report of a 52-year-old woman who presented with pleuritic chest pain and left sided rib pain, leading to diagnosis of bilateral pulmonary emboli, and was incidentally found to have a mesenteric mass. After 13 months of extensive workup with multiple imaging modalities for malignancy and biopsy, this patient was ultimately diagnosed with SM. This case report emphasizes the importance of considering SM in patients outside of the typical demographic and explores strategies for distinguishing it from other conditions and malignancies.

Keywords: Fat Necrosis; Fibrosis; Mesentery; Mesenteric Ischemia; Peritoneal Panniculitis.

Citation: Ahmad A, Bhakta R, Yalamanchili B. Sclerosing Mesenteritis: Case Report of a Malignancy Mimicker. Brazilian Journal of Case Reports. 2026 Jan-Dec; 06(1):bjcr229.

https://doi.org/10.52600/2163-583X.bjcr.2026.6.1.bjcr229

Received: 15 September 2026
Accepted: 27 September 2026
Published: 1 October 2026

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1. Introduction

Sclerosing mesenteritis (SM) is a rare, idiopathic, condition characterized by chronic inflammation and fibrosis of small bowel mesentery adipose tissue [1]. It is a spectrum of mesenteric inflammatory processes, including mesenteric lipodystrophy and mesenteric panniculitis [2,3]. It is reported more often in men, with mean onset between age 60 – 65 years [4,5]. It can present with non-specific symptoms: abdominal pain, fever, or signs of obstruction or ischemia [4,5]. Imaging findings often overlap with malignancy, but certain radiographic features can be used to distinguish SM from other diagnoses.

A 52-year-old woman with incidental mesenteric mass underwent an extensive 13 month evaluation before SM diagnosis. This case of an asymptomatic, middle-aged woman, outside the typical demographic, underscores its clinical variation. It also highlights the need to identify SM earlier to avoid excessive healthcare utilization, as well as unnecessary psychological and financial burden on the patient.

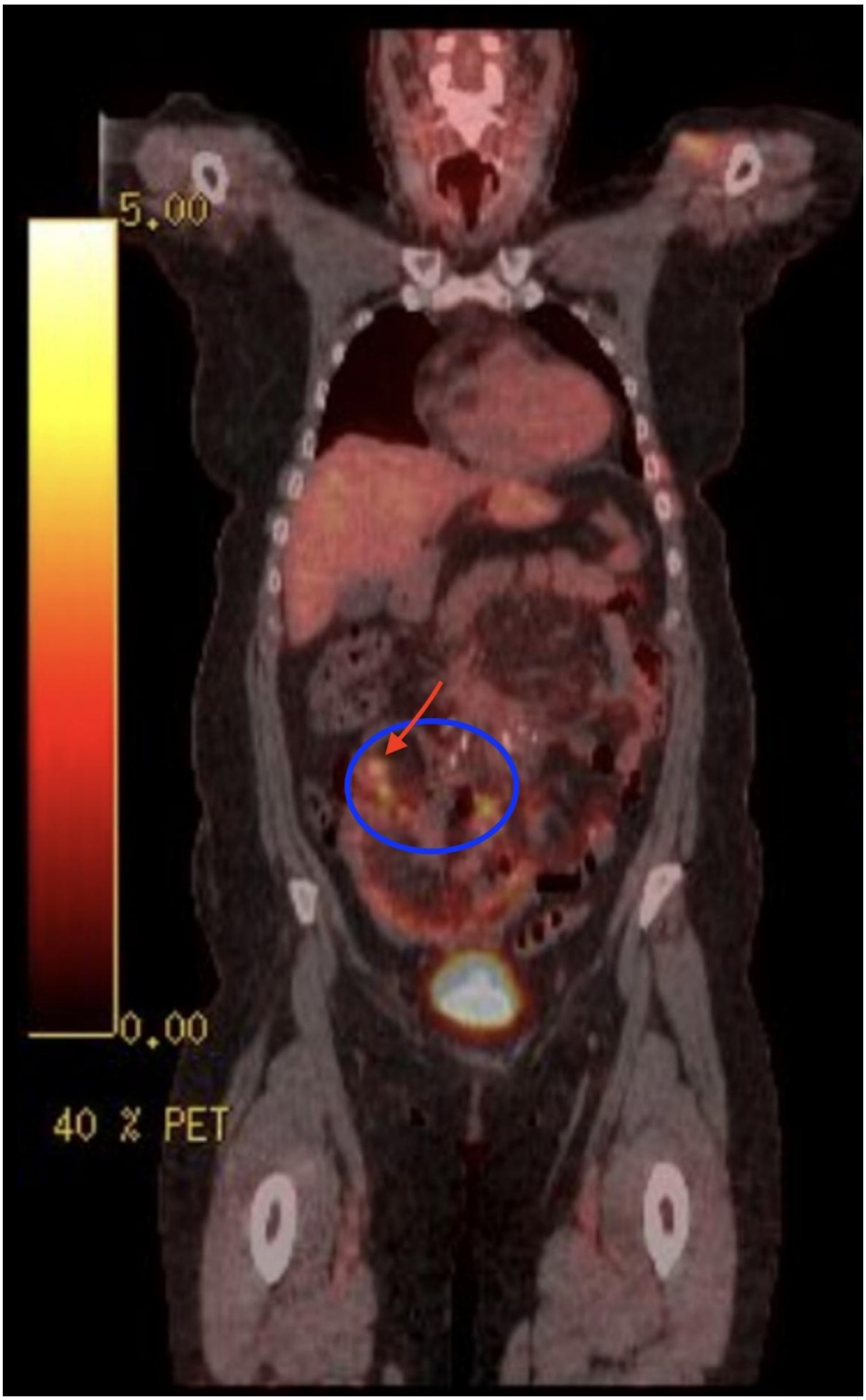
2. Case Report

A 52-year-old woman presented to the emergency department for pleuritic chest pain and left sided rib pain. Computed tomography angiography (CTA) revealed bilateral pulmonary emboli (PEs) and incidental mesenteric mass. Past medical history was significant for trauma six months prior, requiring cervical spine fusion. Initial labs were unremarkable, including hemoglobin, inflammatory markers (ESR, CRP), albumin, tumor markers (CEA, CA 19-9, CA 27-29), and extensive autoimmune work-up. CA-125 was mildly elevated - 62.8 U/mL (normal <37 U/mL).

Computed tomography (CT) enterography 1 month later demonstrated central mesenteric plaque-like calcification with a few lower mesentery spiculated noncalcified soft tissue nodules. Esophagogastroduodenoscopy and colonoscopy were deferred due to PEs and anticoagulation. Fluorodeoxyglucose positron emission tomography (FDG PET) (Figure 1) noted a mildly metabolically active, partially calcified mesenteric mass (standardized uptake value (SUV) max 1.3), with surrounding mildly metabolically active mesen-

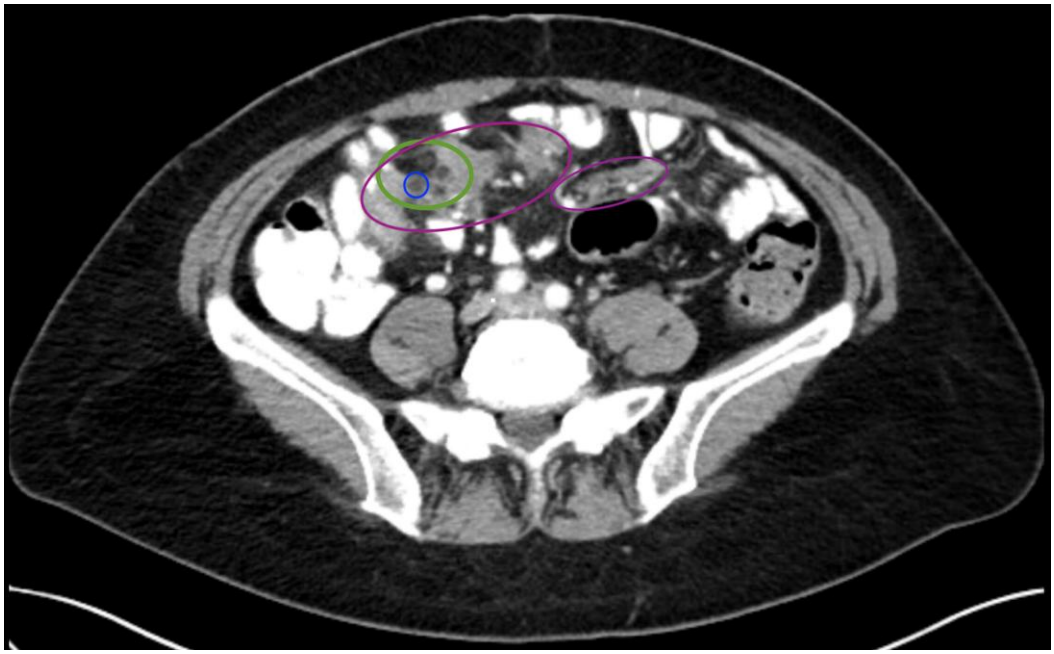
teric lymph nodes (SUV max 4.0). A large metabolically active left lower lobe lung opacity, 5.9 cm (SUV max 4.0), was noted but smaller from prior CTA, and attributed to resolving inflammation.

Figure 1. Initial Fluorodeoxyglucose positron emission tomography computed tomography (FDG PET CT) coronal view demonstrating mildly metabolically active mesenteric lymph nodes SUV max 4.0 (blue oval), one of which is highlighted with the red arrow.



CT of chest, abdomen, and pelvis 5 months later demonstrated increase in mesenteric mass with internal calcification of 8.1 cm, and sub-centimeter bilateral iliac chain lymph nodes. CT-guided core biopsy of mesenteric lymph node revealed fibroadipose tissue and mild chronic inflammation without evidence of malignancy. Repeat CT abdomen and pelvis with contrast at 5 months post-biopsy showed a stable calcified mesenteric lesion (6.3 × 1.6 cm), an enlarging left mesenteric soft-tissue lesion now measuring 2.6 × 2.4 cm (previously 1.9 × 1.6 cm) with similarly stable mesenteric lymphadenopathy, and a stable subpleural lung nodule (1.2 × 1.0 cm) (Figure 2).

Figure 2. Computed tomography abdomen and pelvis with contrast axial view 11 months after initial presentation showing two mesenteric soft tissue masses (magenta ovals). Also demonstrated are four fat ring signs (green oval) with one of the four highlighted with blue circle, though this was not documented on radiology report, as detailed in the discussion section.



At month 13, Ga-DOTATATE PET CT and 24-hour urine 5-HIAA were ordered to evaluate for a neuroendocrine tumor. Ga-DOTATATE PET CT demonstrated marginally increased size of multiple partially calcified and non-calcified mesenteric lesions (Figure 3). Patient did not complete 24-hour urinary 5-HIAA. She denied unintentional weight loss, nausea, vomiting, abdominal pain, and diarrhea. She reported flushing (which she attributed to menopause) and 10 pound intentional weight loss over 1 year.

This case was presented at tumor board. Based on imaging findings, including infiltrative calcified mesenteric masses without avidity on Ga-DOTATATE PET CT, mesenteric swelling, and fat ring signs, as well as pathology demonstrating fibroadipose tissue and chronic inflammation, diagnosis of SM was established. Conservative management with gastroenterology follow-up for esophagogastroduodenoscopy/colonoscopy and clinical monitoring was recommended. It took 13 months from time of presentation to diagnosis (Figure 4).

3. Discussion and Conclusion

SM is a rare, non-neoplastic inflammatory process of small bowel mesentery adipose tissue, often mistaken for neoplasm [1]. It is a spectrum progressing from fat necrosis (mesenteric lipodystrophy) to chronic inflammation (mesenteric panniculitis), and ultimately fibrosis (SM) [2,3]. Prevalence is 1.3% to 7.8%, with men affected at twice the rate, and presentation onset age 50-70 (mean onset 60-65) [1]. Etiology is unclear but associated

with autoimmune conditions, infections, prior abdominal surgery, and trauma [1]. In this case, the patient suffered a major trauma 6 months prior to presentation. In patients with SM, 75% present with abdominal pain, 30% - 40% with change in bowel movements, 25% with weight loss, 20% with vomiting, 15% with anorexia, and 5% to 20% with fever [4,5]. Laboratory findings may include elevated CRP and ESR, anemia, or hypoalbuminemia [4,5]. Normal labs and absence of symptoms made this case atypical.

Figure 3. Ga-DOTATATE PET CT coronal view depicts multiple partially calcified (green arrow indicates one) and non-calcified (yellow arrow denotes one) mesenteric lesions without increased fluorodeoxyglucose (FDG) avidity. On further review, this image also demonstrates "misty mesentery" (purple arrow showing area of increased density of the mesenteric fat), not documented on radiology report, as outlined in the discussion section.

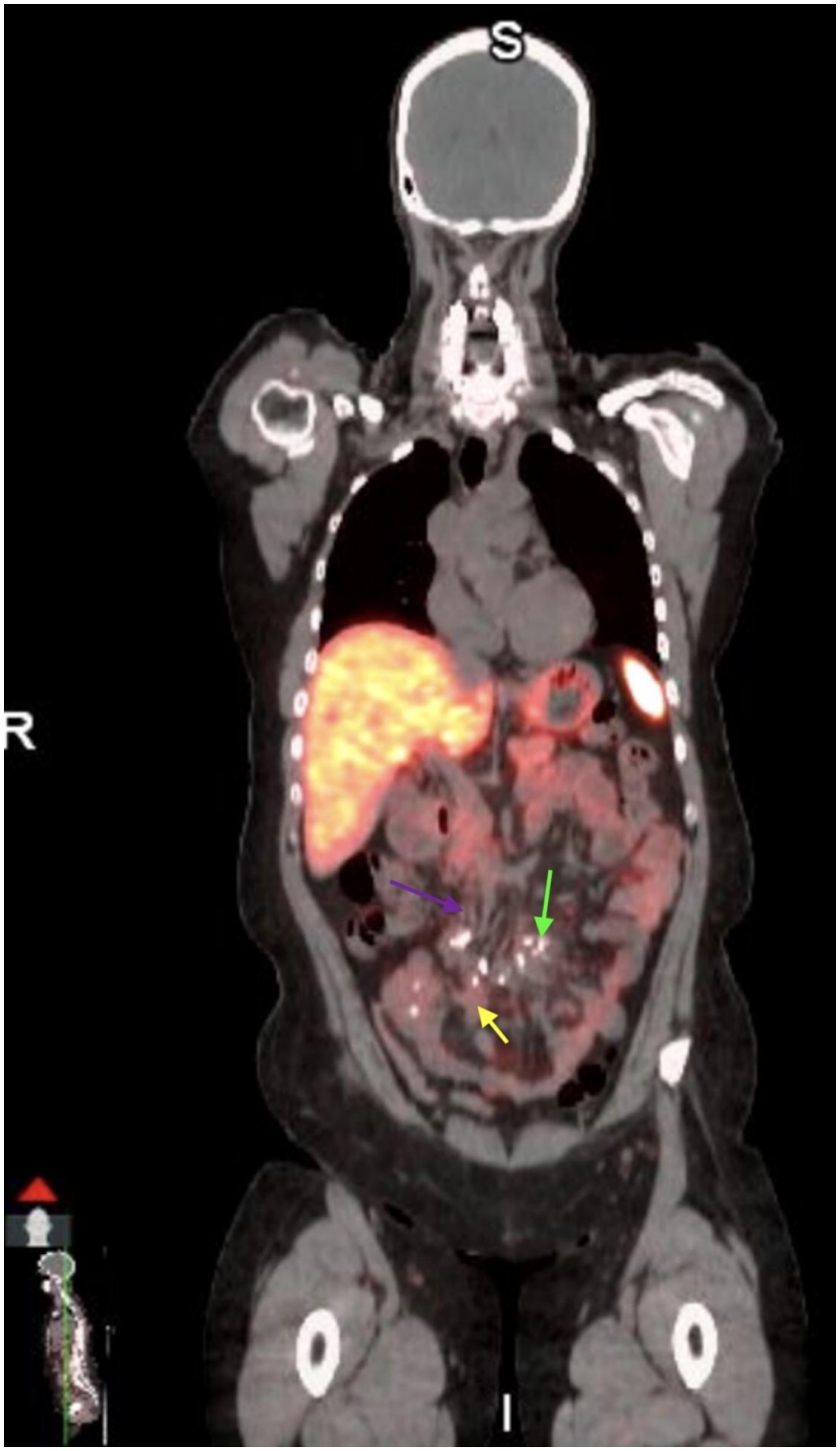
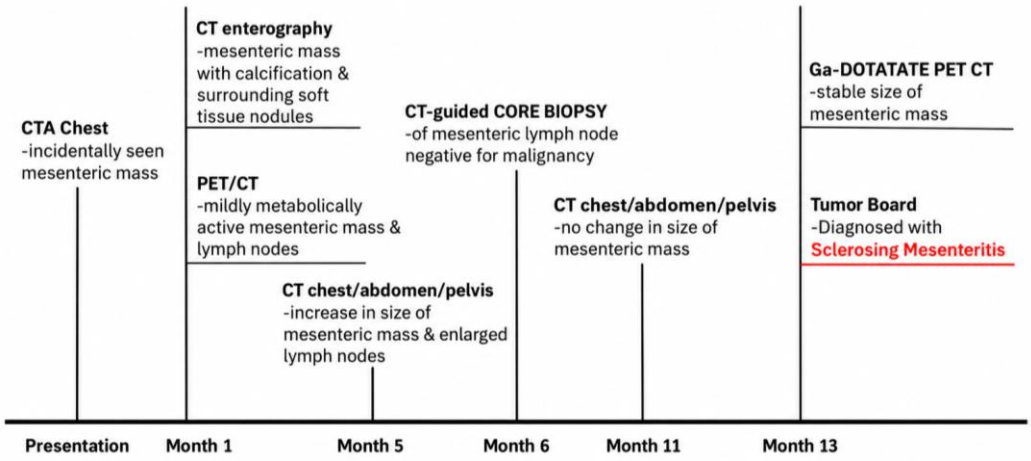


Figure 4. Timeline from initial presentation of patient to final diagnosis 13 months later.



A distinct finding of SM on CT imaging is the presence of a circumscribed mesenteric mass encased in a fibrotic pseudocapsule, with a border of fat, called the "fat rin sign", though a similar finding can be found in malignancy [6-8]. Upon review of patient's CT, the fat ring sign is visible - though not noted in the radiology report (Figure 2). Another radiological finding of SM is "misty mesentery," an increase in density of mesenteric fat, which can also be seen in malignancy [1,9]. Misty mesentery is appreciated on review of Ga-DOTATATE PET CT (Figure 3), which was also not documented by radiology. If recognized earlier, imaging findings may have enabled a timelier diagnosis, underscoring the need for increased awareness of SM, including among radiologists.

Differential diagnosis of mesenteric mass includes neoplasm. Features favoring malignancy include lymph nodes >1cm, extra-mesenteric lymphadenopathy, and "B symptoms" (fevers, night sweats, unintentional weight loss) [1]. PET-based imaging can yield a false positive result in the setting of inflammatory processes, potentially complicating evaluation for malignancy [10,11]. In this case, the initial FDG PET CT demonstrating avidity in the mesenteric masses increased the concern for malignancy. The final Ga-DOTATATE PET CT was negative for metabolically active sites and aided in leading to the final diagnosis with tumor board review.

Histologically, SM is characterized by fat necrosis, chronic inflammation, and fibrosis [3]. Natural progression is slow growth in 80% of patients [5]. Management is symptom dependent so asymptomatic patients can be monitored. Complications include bowel obstruction, ascites, and mesenteric ischemia due to enlarging mass [4,12]. Non-obstructive symptoms (abdominal pain, vomiting, diarrhea) can be treated with corticosteroids and/or tamoxifen, with stepwise escalation to immune modulators [1,4]. Prognosis is favorable, with rare cases of spontaneous regression, years after diagnosis [13].

Identifying SM remains a diagnostic challenge due to non-specific symptoms, imaging overlap with malignancy, and rarity. While the "fat ring sign" on imaging is a characteristic and highly suggestive feature of SM, it is not pathognomonic and may occasionally be seen in other disease processes [6-8]. Thus, biopsy remains the gold standard for diagnosis [1,2]. High index of suspicion for SM is needed to reduce diagnostic delays, unnecessary imaging, and healthcare costs.

Funding: None.

Research Ethics Committee Approval: Written informed consent was obtained from the patient's legal representative, and the study was conducted in accordance with the ethical principles established in the Declaration of Helsinki.

Acknowledgments: We are grateful to the patient for generously allowing us to share her experience so that others may learn from this case.

Conflicts of Interest: All other authors declare no conflicts of interest.

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