Pseudomyxoma Peritonei Mimicking Liver Cirrhosis: A Case Report and Literature Review

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ABSTRACT

Pseudomyxoma peritonei (PMP) is a rare condition caused by rupture of a primary mucinous tumor, followed by dissemination of tumor cells within the peritoneal cavity and excessive mucin production. The signs and symptoms are generally nonspecific, making early diagnosis difficult. We present a man, age 57, who showed abdominal distention accompanied by nausea and vomiting for approximately four months, which was referred to as liver cirrhosis. Carcinoembryonic antigen level was elevated and postoperative histology revealed low-grade peritoneal mucinous neoplasia. The signs and symptoms of PMP are generally nonspecific, making it difficult to diagnose earlier and leading to delayed appropriate treatment. Computed tomography (CT) scan is the preferred imaging modality for determining the extent of the disease preoperatively. Elevated tumor marker level, especially CEA, CA19-9, and CA-125, is usually suggestive of the diagnosis of PMP. Immunohistochemistry was used to determine the origin of primary malignancy. PMP management needs to be individualized; therefore, discussion among multidisciplinary teams is needed. Currently, the most common treatment option is cytoreductive surgery (CRS) in conjunction with hyperthermic intraperitoneal chemotherapy (HIPEC) and has been associated with improved survival in several studies. However, if there are limitations in therapeutic modalities or patient populations, alternative therapies can be used. This case report aims to highlight the diagnostic challenges of pseudomyxoma peritonei presenting as ascites that mimics liver cirrhosis.

Keywords: *Pseudomyxoma Peritonei, Mucinous Ascites, Jelly-Belly Ascites, Gelatinous Ascites, Carcinoembryonic Antigen*

ABSTRAK

Pseudomyxoma peritonei (PMP) adalah suatu kondisi langka yang disebabkan oleh rupturnya tumor mukus primer yang diikuti oleh implantasi sel tumor di dalam kavum peritoneum dan sekresi mukosa yang berlebihan. Kami melaporkan seorang pria berusia 57 tahun yang mengalami distensi abdomen disertai mual dan muntah selama kurang lebih empat bulan yang dirujuk dengan sirosis hepatis. Kadar carcinoembyronic antigen ditemukan meningkat dan gambaran histopatologi menunjukkan neoplasia mukinosa peritoneal derajat rendah. Tanda dan gejala PMP umumnya tidak spesifik, sehingga sulit untuk didiagnosis lebih awal dan menyebabkan keterlambatan intervensi. Computed tomography (CT) scan merupakan modalitas pencitraan yang lebih dipilih untuk menentukan derajat penyakit sebelum operasi. Peningkatan kadar penanda tumor, terutama CEA, CA19-9, dan CA-125 biasanya sugestif terhadap diagnosis PMP. Imunohistokimia dapat dilakukan untuk menentukan

primer dari keganasan. Manajemen PMP dilakukan secara individual, oleh karena itu diperlukan pendekatan multidisipliner. Bedah sitoreduktif (CRS) yang dikombinasikan dengan kemoterapi intraperitoneal hipertermik (HIPEC) saat ini merupakan pilihan utama pengobatan dan dikaitkan dengan tingkat kelangsungan hidup yang lebih lama dalam beberapa penelitian. Alternatif terapi dapat dipertimbangkan apabila fasilitas atau kondisi pasien tidak memungkinkan dilakukan CRS-HIPEC. Laporan kasus ini bertujuan untuk menyoroti tantangan diagnostik pseudomyxoma peritonei yang bermanifestasi sebagai asites yang menyerupai sirosis hati.

Kata kunci: *Pseudomyxoma Peritonei, Mucinous Ascites, Jelly-Belly, Gelatinous Ascites, Carcinoembryonic Antigen*

INTRODUCTION

Pseudomyxoma peritonei (PMP) is an intra-abdominal malignancy due to the growth of mucin-producing tumors in the peritoneal cavity and leading to mucinous ascites (*jelly-belly*).[1] The incidence of PMP is around 0.2 per 100,000 persons annually.[2] With a median age of 53, women are more likely than men to have PMP (9:11).[3] The majority of PMP cases start in the appendix, while they can also occasionally start from mucinous tumours in the colon, ovaries, or pancreas.[1]

PMP begins with the rupture of a mucinous tumor from the organ of origin and massive tumor cells colonize the peritoneal cavity and produce mucus.[4] Diagnosis of PMP is challenging for clinicians as it frequently manifests as nonspecific symptoms like distension, blockage, or abdominal pain.[5] A case report in Beirut in 2024 reported misdiagnosis of a patient with PMP as cirrhosis hepatitis.[6] An abdominal CT scan with notching of the liver or lien due to external compression by gelatinous mass, and mucinous carcinoma peritonei on histopathology can establish the diagnosis of PMP.

We reported a 57-year-old male patient with pseudomyxoma peritonei and secondary bacterial peritonitis with an initial diagnosis from the referring hospital of suspected hepatic cirrhosis. This case report aims to highlight the diagnostic challenges posed by pseudomyxoma peritonei presenting as ascites that mimics liver cirrhosis.

CASE ILLUSTRATION

A 57-year-old man came to Ngoerah Hospital's emergency ward with complaints of an enlarged abdomen, nausea, and vomiting that had progressively worsened for 4 months before the visit to Ngoerah Hospital, accompanied by a feeling of fullness, shortness of breath, and a full stomach when filled with food. The patient also experienced nausea and vomiting with increasing frequency, along with abdominal enlargement. The patient denied fever, weight loss, vomiting blood, or black stools. The patient had no symptoms of obstruction related to defecation or micturition. Alcohol consumption, history of hepatitis, malignancy, autoimmune diseases, and other chronic diseases were also denied.

The patient was compos mentis, blood pressure 114/86 mmHg, respiratory rate 22 times per minute, VAS 1/10, and other vital signs measured within normal limits. On physical examination, the patient showed anemic conjunctiva, abdominal distension with a positive undulation test, but the enlarged hepatic organs or lien were difficult to evaluate; the patient complained of mild tenderness in all abdominal quadrants. There were no signs of chronic liver disease or cirrhosis such as spider navy, gynecomastia, palmar erythema, or caput medusae.

Laboratory examination revealed mild hypochromic microcytic anemia (Hb 10.90 g/dL; MCV 68.40 fL; MCH 23.40 pg), hyponatremia (130 mmol/L (136 - 145)), also elevated levels in alkaline phosphatase (ALP) (159 U/L (40 - 150)), gamma GT (71 U/L (<59)), and globulin (5.5 g/dL (2.2 - 3.7)). Anti-HCV and HBsAg tests were non-reactive. Ascitic fluid puncture was performed in the hospital room. Only 5 cc of ascitic fluid was obtained, with a yellow color and thick gelatinous consistency suggestive of mucinous ascites. The patient complained of pain during aspiration. Analysis of ascitic fluid obtained ascites-albumin serum gradient (SAAG) 3 (>1.1), with a polymorphonuclear cell count of 520.5 cells. Cytologic examination of ascitic fluid revealed no malignant cells. There was an elevated CEA level of 88.34 ng/mL (0 - 5), while alpha fetoprotein (AFP) levels were found to be normal.

The patient was given initial management of fluid restriction, furosemide 40 mg every 24 hours intraoral, spironolactone 100 mg every 24 hours intraoral, metoclopramide ten mg every 24 hours intra oral if necessary, and ceftriaxone two grams every 24 hours intravenous. Antibiotic treatment was given for 7 days, and diuretic medication was discontinued because there was no improvement. The patient then

underwent laparoscopic biopsy and ascites drainage, with postoperative evaluation finding serous ascites with a *jelly-belly* like consistency, and a humped hepatic surface **(Figure 1A).** Postoperatively, the patient was attached to two ascites drainage tubes with ascites fluid production of 2,000 cc in one hour, serous colour **(Figure 1B).** Histopathological examination was performed, and the peritoneal tissue and abdominal wall showed extensive extracellular mucinous material and few glandular epithelial components with mild atypia and mucin vacuoles suggestive of low grade peritoneal mucinous neoplasia **(Figure 2)**, consistent with PMP.

Post laparoscopic contrast CT scan of the abdomen, showing normal hepatic size, irregular surface with obtuse angle, homogeneous normal parenchymal density, no EHBD/IHBD and vascular dilation, no nodules/masses/cysts as seen in **(Figure 3).** There was also *scalloping* of the visceral surfaces of the right and left hepatic lobes, and a fluid-density appearance remained in the abdominal and pelvic cavities, with two drain catheters in place in the abdominal region. The patient underwent a colonoscopy to trace the origin of the tumour, with the findings of the rectum to the terminal ileum within normal limits.

In this case report, we did not find the origin of the tumor. The patient was planned to have a follow-up examination at Hemato-Oncology Division, but the patient had returned to his home region and had been receiving systemic chemotherapy.

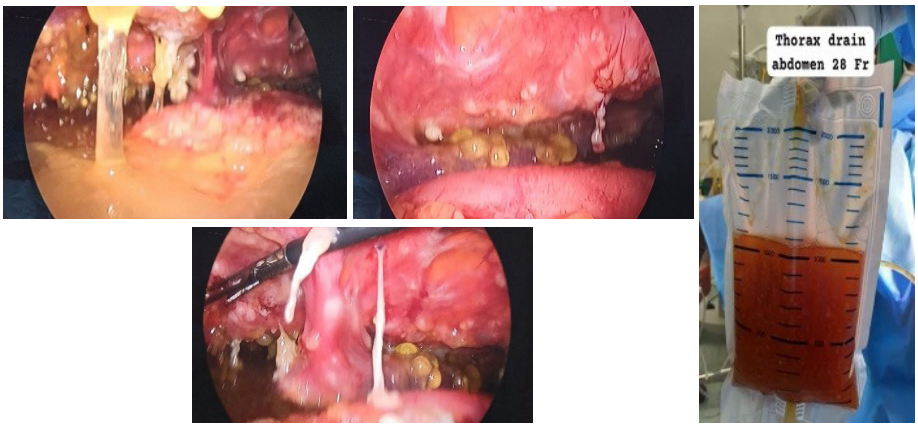

Figure 1 (A) Evaluation during surgery found turbid yellow and thick ascitic fluid, (B) The patient was fitted with thorax drain 28 Fr cavum abdomen, with 1-hour postoperative ascites production of 2,000 cc serous color

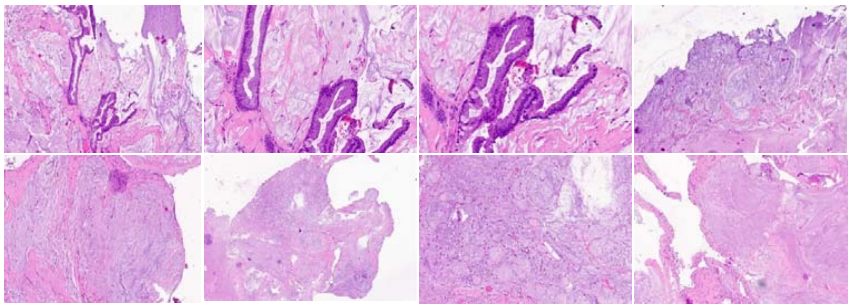

Figure 2. Histomorphological features of peritoneal and abdominal wall tissue biopsies showing extensive extracellular mucinous material and little glandular epithelial component with mild atypia and mucin vacuoles suggest low-grade peritoneal mucinous neoplasia (corresponding to pseudomyxoma peritonei)

Figure 3. Post operative CT Scan Abdomen with contrast. Red arrows showing a liver *scalloping*

DISCUSSION

This case highlights an unusual presentation of pseudomyxoma peritonei mimicking liver cirrhosis. The patient initially presented with progressive abdominal distension and ascites, which led to suspicion of chronic liver disease. However, further evaluation revealed distinctive findings, including thick gelatinous "*jelly-belly*" ascites and difficulty during ascitic fluid aspiration due to its viscous consistency. Because PMP often presents with nonspecific clinical manifestations, it may lead to diagnostic pitfalls. The condition may initially be mistaken for other causes of ascites, particularly liver cirrhosis, peritoneal carcinomatosis, or peritoneal tuberculosis. In some cases, PMP may also mimic malignant ascites associated with gastrointestinal or ovarian malignancies, leading clinicians to suspect more common conditions such as chronic liver disease.[7,8]

Mucin buildup in the peritoneal cavity as a result of the proliferation of mucin-producing cells on the peritoneal surface is a hallmark of PMP.[1] PMP most commonly originates from primary appendiceal lesions, but can originate from other gastrointestinal organs (small bowel, colon, pancreas, stomach, bile duct, gallbladder).[9] A 10-year retrospective study found 1,482 (0.9%) appendiceal epithelial lesions out of 167,744 appendectomies, with all of them having a PMP.[2] A mucinous tumour ruptures from its origin, allowing tumour cells to migrate into the peritoneal cavity and giving rise to pseudomyxoma peritonei. The flow of peritoneum fluid in the abdominal cavity will distribute mucin and tumor cells to the pelvic cavity, hepatic cavity, and omentum, known as the redistribution phenomenon. Ascites will result from the increased mucus production by tumour cells in the peritoneal cavity.[4,10,8]

Pseudomyxoma peritonei can manifest as a collection of symptoms, one of the most common symptoms is abdominal or low back pain, flatulence and indigestion due to the accumulation of mucin in the abdominal cavity causing intestinal obstruction. Physical examination may reveal abdominal distension.[9] It generally occurs between the ages of 50 and 60 years and is more common in women (male:female ratio 9:11).[3] In this case, the patient complained of progressive abdominal enlargement accompanied by minimal tenderness, with a *jelly-like* ascitic fluid consistency. The patient in this report had elevated CEA levels and PMN count >500 cells, suggestive of secondary bacterial infection. Carcinoembryonic antigen (CEA), carbohydrate antigen 125 (CA-125), and carbohydrate antigen 199 (CA19-9), are three tumor markers associated with PMP. Between 56% and 75% of patients had elevated levels of CEA, while 58% to 67% had elevated CA19-9 levels. The baseline values of these tumor markers are reported to be associated with tumor extent, aggressiveness, recurrence risk, and the amount of tumor remaining post resection.[11,12,13]

A study reported that PMP patients with transverse colon cancer who were positive for all three tumor markers had a poor prognosis, with a survival rate of 29 months. A poor prognosis was also reported in PMP patients with rectal cancer with elevated levels of all three tumor markers, with a survival rate of less than 36 months.[14,15] Unlike ascites associated with liver cirrhosis, spontaneous bacterial peritonitis is relatively uncommon in mucinous ascites because it is not primarily related to portal hypertension or bacterial translocation from the gut. However, infection may still occur, particularly following invasive procedures, repeated paracentesis, tumor necrosis, or bowel perforation associated with advanced disease. In such circumstances, secondary bacterial peritonitis may develop, contributing to clinical deterioration.[16]

Analysis of immunohistochemical pulses, such as cytokeratin (CK) 7, CK 20, and CDX-2 can be used to determine the origin/primary malignancy.[17] In 2008, the Peritoneal Surface Oncology Group International (PSOGI) issued a consensus on the preoperative evaluation of PMP cases including serum tumor marker examination, CT scan and three-dimensional reconstruction, as well as laparoscopic exploration and cytology.[18] A cystic or cystic-solid mass with calcifications in the right lower abdomen may be seen on an abdominal CT scan **(Figure 4A),** mucinous ascites in the abdominal cavity **(Figure 4B),** and compression or invasion of other intra-abdominal organs **(Figure 4C-F).**[18] *Scalloping* features indicate external compression of the organs (liver and lien) due to the gelatin mass. Intraperitoneal production of accumulated mucin causes indentations to form on the organs.[19] In this case, a *scalloping* image of the liver was found. Other imaging, such as thoracic and pelvic CT scans and colonoscopy, is also recommended by the American Society of Colon and Rectal Surgeons (ASCRS) to detect primary sources of malignancy.[20]

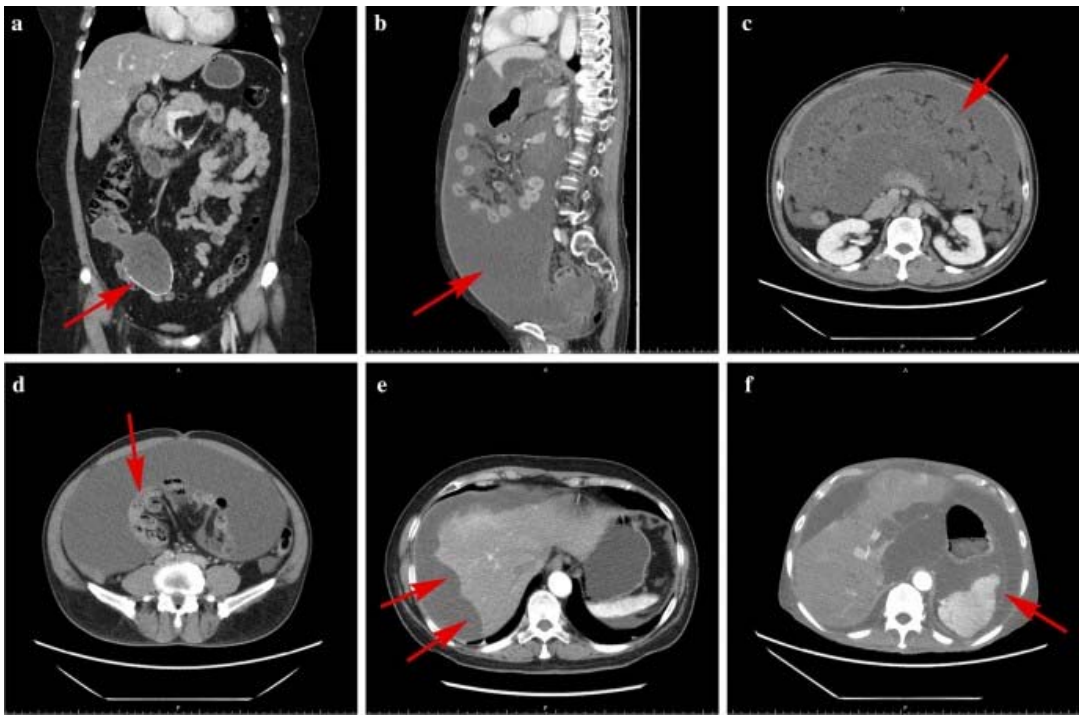

Figure 4. CT scan abdominal features of PMP: (A) Enlargement of the appendiceal cavity and calcification of the appendiceal wall; (B) Enlargement of the abdominal circumference due to intraperitoneal mucus volume increase (*jelly-belly*); (C) Thickening of the greater omentum (*omental cake*); (D) Small intestine depressed by mucus causing central displacement; (E) *Scallop* image on the surface of the liver; (F) Deformation of the spleen contour.[18]

Peritoneal Surface Oncology Group International issued special terminology in the classification of PMP to facilitate the selection of appropriate therapy, namely low-grade mucinous carcinoma peritonei, high-grade mucinous carcinoma peritonei, and high-grade mucinous carcinoma peritonei with signet ring cells **(Table 1).** Fewer epithelial cells or gland-like structures are often found in patients with low-grade mucinous carcinoma peritonei **(Figure 5A&B).** Some cell clusters will appear as islands in mucin. Atypical cytology includes low-grade lesions, and in some cases, the cells look non-neoplastic. High-grade lesions are usually more cellular and have the characteristics of large and vesicular nuclei, irregular nucleus contours, and numerous mitoses or cribiform growths **(Figure 5C).** Organ-destructive invasion is also among the hallmarks of high-grade lesions. Signet ring cell-containing tumours are categorized individually due to their poor prognosis **(Figure 5D).** A lesion is considered a signet ring cell carcinoma if >50% of the cells show signet ring morphology, but if only a few cells in the mucin show signet ring morphology and appear degenerative, it can be ignored.[1] In this case, the biopsy results of the patient's peritoneal wall showed a histomorphology picture of extensive extracellular mucin and few glandular epithelial components with mild atypia and mucin vacuoles, so it was consistent with the picture of low grade peritoneal mucinous neoplasia.[1]

Table 1. Diagnostic Criteria for PMP[1]

Lesion	Criteria
Acellular mucin	• Mucin in peritoneal cavities without neoplasm epithelial cells
Low-grade mucinous carcinoma peritonei / DPAM	• Little epithelial component • Gland-like structures or collections of small cells • Minimal atypical cytology • Sporadic mitosis • Invasion by pressing on organs
High-grade mucinous carcinoma peritonei / PMCA	• Generally more cellular • Cribriform growth • High-grade atypical cytology • Many mitoses • Organ invasion is infiltrative and destructive
High-grade mucinous carcinoma peritonei with *signet ring cells* / PMCA-S	• Any lesion with a signet ring cell component (round cells with mucin in the cytoplasm that pushes nuclei to the cell membrane)

DPAM, disseminated peritoneal adenomucinosis; PMCA, peritoneal mucinous carcinomatosis, least squares; PMCA-S, peritoneal mucinous carcinomatosis with signet rings cells

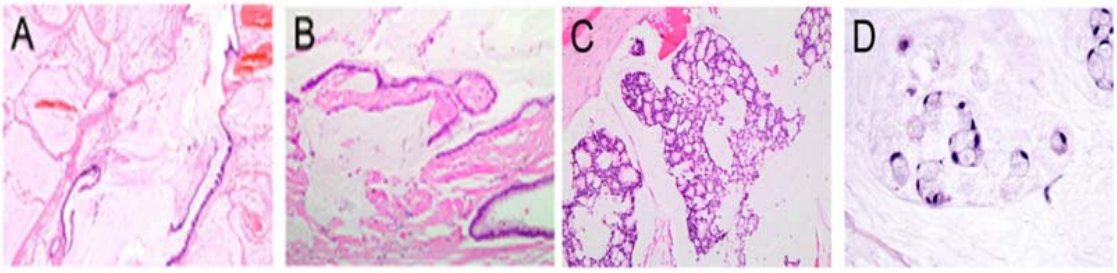

Figure 5. (A & B) Low grade mucinous carcinoma peritonei; (C) High grade mucinous carcinoma peritonei; (D) High grade mucinous carcinoma peritonei with signet ring cells.[5]

Cytoreductive surgery (CRS) combined with hyperthermic intraperitoneal chemotherapy (HIPEC) is the treatment of choice in PMP.[21] Hyperthermic intraperitoneal chemotherapy can be administered during the surgical procedure or postoperatively (early postoperative intraperitoneal chemotherapy (EPIC)), with the aim of preventing recurrence and progression of residual microscopic to minimal macroscopic tumors, where the cytotoxic effect of HIPEC can reach a tumor depth of 2.5 mm.[22]

The five-year survival rate of low-grade stage patients undergoing CRS-HIPEC procedure is 60-100% while that of high-grade stage patients is 0-60%.19 In conditions where the tumor cannot be resected, or the hospital/surgeon cannot perform HIPEC, or the patient's condition is not feasible, alternatives such as debulking surgery, systemic chemotherapy (adjuvant, neoadjuvant) and palliative care can be performed.[20,23] For both low- and high-grade PMP, neoadjuvant or adjuvant systemic chemotherapy may be recommended. An alkylating drug (such as oxaliplatin) and fluoropyrimidine are advised if neoadjuvant or adjuvant systemic chemotherapy is needed. Chemotherapy with a bevacizumab regimen may contribute to prolongation of progression-free survival (PFS).[24] A limitation of this report is that the exact primary origin of the pseudomyxoma peritonei remains undetermined. Despite several diagnostic evaluations, no definite primary source was detected. Further investigation was limited because the patient continued treatment at another institution.

CONCLUSION

We reported a male with nonspecific symptoms of an enlarged abdomen with thick *jelly* ascites, diagnosed as PMP. The unusual disorder known as PMP results from the rupture of a primary mucinous tumor, followed by implantation of tumor cells in the peritoneum and excessive mucous secretion. Signs and symptoms are generally nonspecific, making early diagnosis difficult. Abdominal CT scan, histopathologic examination, serum tumor marker, and immunohistochemistry are recommended to support the diagnosis of PMP and to detect the primary source of malignancy. Elevated serum tumour markers before surgery typically indicated an aggressive disease and a high chance of recurrence. CRS combined with HIPEC is the primary choice of PMP management and is associated with better survival rates. Alternatives such as debulking surgery, systemic chemotherapy, and palliative care may be an option in patients with inoperable tumors, unfit patients, or hospital/surgeon limitations.

CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest regarding the publication of this article.

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AUTHOR CONTRIBUTIONS

D.P.P. contributed to patient management, data collection, literature review, and manuscript drafting. G.S. contributed to patient management, conceptualization, supervision, and critical revision of the manuscript. All authors read and approved the final manuscript.

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DATA AVAILABILITY

The datasets are available in the manuscript.

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