

# Spontaneous Bacterial Peritonitis in a Postpartum Female Secondary to Necrotizing Pancreatitis

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## Introduction

Spontaneous bacterial peritonitis (SBP) is an infection of the fluid that accumulates in the abdomen, typically caused by translocation of bacterial pathogens [1]. Failure in defensive factors to contain pathogens to the bowel and can lead to seeding other extra-intestinal sites. Most common pathogens isolated from SBP cases include, *Escherichia coli*, *Klebsiella* species, *Proteus* species, *Enterococcus faecalis*, and *Pseudomonas* [1,2]. Recurrence is a common sequela for patients with SBP, a large majority tend to be at an increased risk within 1 year, especially those with chronic liver cirrhosis and low protein concentration in ascitic fluid [1,2]. Clinical symptoms of SBP range from abdominal pain, pyrexia, nausea/vomiting, acute encephalopathy, decreased urine output [2]. Diagnosis involves ascitic fluid analysis whereby decreased pH and increased PMNL cell count provide adequate suspicion when clinically correlated [2,3]. Culture data and treatment with antibiotics are mainstays of management.

## Imaging

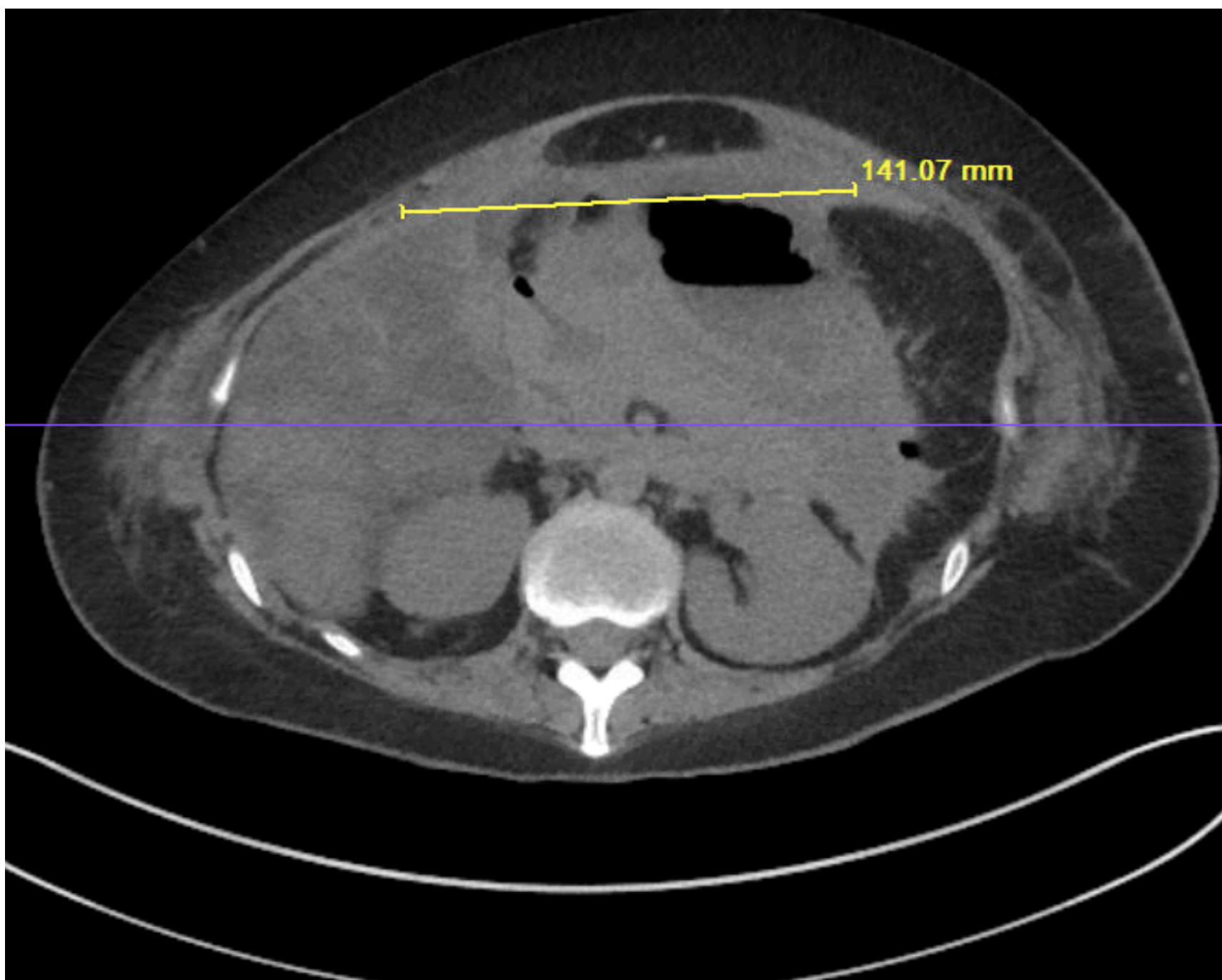


Fig. 1: Large fluid collection restituting along the anterior margin of the pancreas

## Case Presentation

A 27-year-old female presented to the emergency department with diffuse abdominal pain, nausea, vomiting, and diarrhea. Past medical history is significant for recent emergent low transverse cesarean section due to pre-eclampsia complicated by hypertriglyceridemia-induced pancreatitis. She was discharged from a nearby hospital 2 days prior to arrival following a 6 week stay for pancreatitis, complicated by ARDS requiring intubation, and further complicated by a *C. difficile* infection. On arrival to the ED, she was febrile, hypotensive, tachycardic, and tachypneic.

On admission, the patient was septic and had significant bilious emesis with severe, diffuse abdominal tenderness. Laboratory values revealed a white blood cell count 5.7, sodium 122, total protein 5.6, albumin 3.3. Creatinine, lipase, AST, ALT were within normal limits. Initial CT abdomen/pelvis revealed large volume ascites, fluid surrounding the pancreas, hepatic steatosis, small volume fluid and gas within the endometrial cavity. Initial paracentesis revealed brown, creamy fluid with 18K nucleated cells, 229K RBCs, fluid LDH 6824, fluid amylase 26, total protein 3.3, glucose 125, SAAG <1.1. MRCP conducted was unremarkable. Ascitic fluid culture was positive for pansensitive *E. coli*, and the patient was started on IV cefepime. *C. diff* stool toxin was positive, treated with vancomycin.

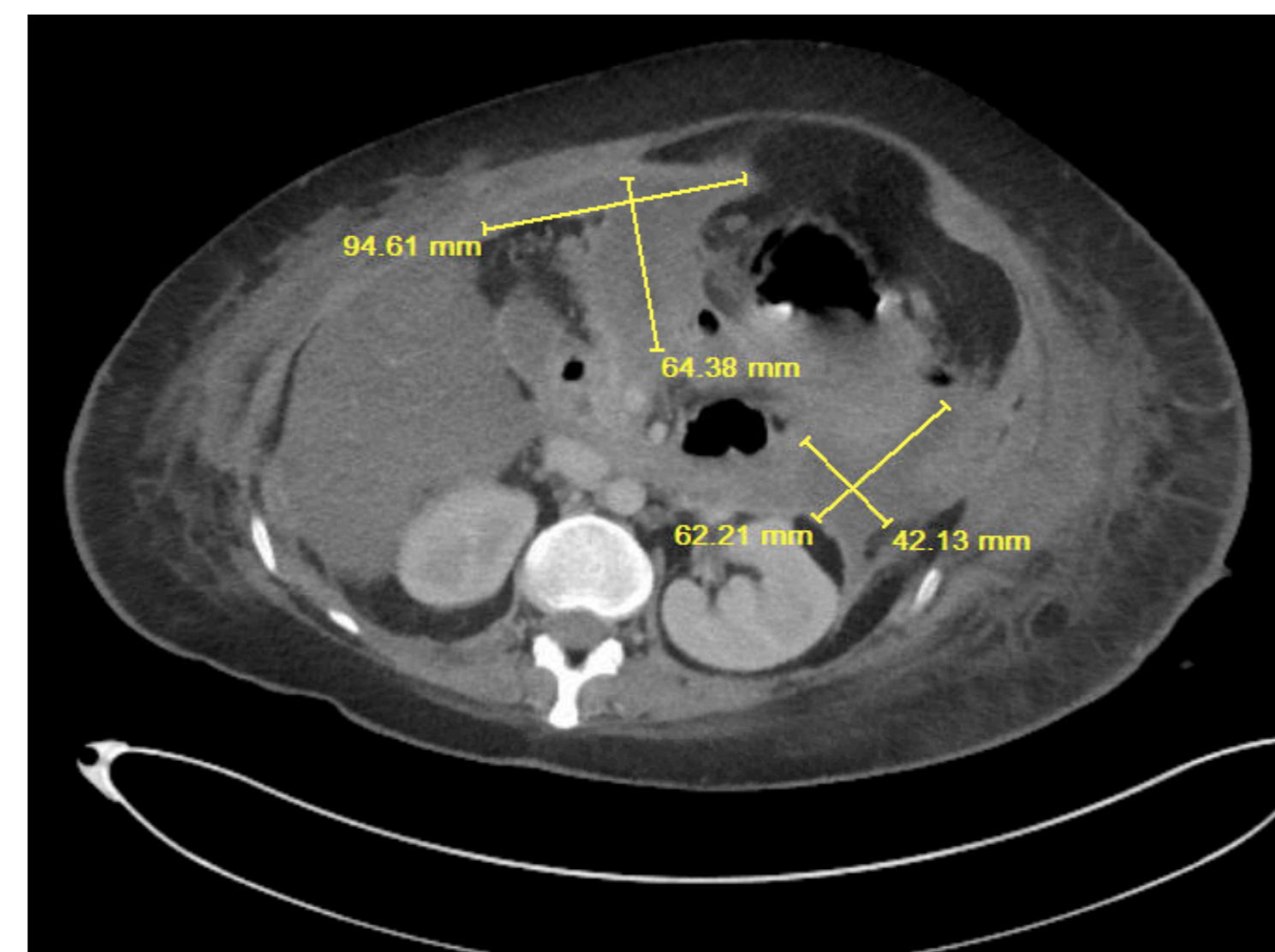


Fig. 2: Multiple likely abscesses in retroperitoneum and anterior body wall

## Cont.

Surgery, GYN, ID, and GI were consulted due to the complexity of the case with an unknown sepsis source. Concerns of a possible bowel perforation was ruled out through repeat contrast imaging. Continued ascitic fluid analysis revealed increasing nucleated cell counts peaking at >240K. Multiple abdominal drains were placed in order to mitigate peritoneal fluid collection, which had now settled in the retroperitoneal space. Stool pancreatic elastase was obtained to assess pancreatic insufficiency and found to be 168 indicating moderate insufficiency.

Throughout the patient's hospital course, concern for source control remained as the patient would continue to be cyclically febrile. Repeat ascitic fluid analysis did not reveal improving cell counts and cultures were persistently *E. coli* positive while on appropriate antibiotic treatment. An endometrial biopsy was obtained revealing fibrinopurulent material within the endometrial cavity. However, this result was thought to be secondary to primary source of infection. The patient was taken to the OR for site exploration. Bilateral retroperitoneal excisional debridement and washout was conducted revealing significant amounts of retroperitoneal saponified fat secondary to necrotizing pancreatitis. Furthermore, extensive abscesses with communications were evaluated and drained (Fig 1/2/3).



Fig. 3: Redemonstration of fluid surrounding pancreas

## Discussion and Conclusion

- Spontaneous bacterial peritonitis (SBP) is an infection of fluid that accumulates within the abdomen, classically in liver cirrhotic patients [1]
- Our patient was unique in that her SBP was characterized in the setting of a recent pregnancy complicated by pre-eclampsia resulting in emergent cesarean section, and further complicated by hypertriglyceridemia-induced pancreatitis
- Treatment was tailored to the ever-evolving clinical presentation including appropriate antibiotic management, consultation and collaboration with relevant providers, etc.
- Upon surgical intervention and exploration of the abdominal cavity, the diagnosis of necrotizing pancreatitis was made
- Seeding of pancreatic enzymes to the retroperitoneal cavity led to lipid saponification and communicating abscess formation, likely the source of SBP and our patient's clinical presentation (Fig.2)
- Other differentials for a primary diagnosis were explored including endometritis, recurrent *C. difficile* infection, protein-losing enteropathy [4,5]; each ruled out by appropriate diagnostic testing/imaging
- Due to the nature of the diagnosis, containment of the retroperitoneal abscesses proved very difficult resulting in multiple surgeries and a prolonged hospital course
- In conclusion, this case provided a unique presentation of SBP secondary to necrotizing pancreatitis in a postpartum patient. The diagnosis proved challenging due to the unremarkable diagnostic testing gathered prior to surgical intervention.

## References

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