

# Atypical Presentation of Acute Pancreatitis: Case Report and Review of Literature

Thamir Hashim<sup>1</sup>, Murad Mehmood<sup>1\*</sup>, Mazin Sharafeldien Elsayed Mohamed<sup>1</sup>, Hany A. Zaki<sup>1,2</sup>, Salah Idris<sup>1</sup>, Mohamed Elgassim<sup>1</sup>

<sup>1</sup>Emergency Medicine department, Hamad Medical Corporation, Qatar

<sup>2</sup>Clinical Assistant Professor of Emergency Medicine, College of Medicine, Qatar University (CMED - QU), Qatar

\*Correspondence: Murad Mehmood, Hamad General Hospital, Doha 3050 (Al Rayyan Rd.), Qatar, Tel: +974 4439 2549,

✉ mmehmood2@hamad.qa

Radiology Case. 2024 September; 18(9):39-46 :: DOI: 10.3941/jrcr.5436

## Acknowledgements

We would like to thank the nursing and medical staff at Hamad Medical Corporation for their assistance in the care of the patient.

## Disclosures

None of the authors have any financial or competing interests to disclose.

## Consent

Yes, the author obtained written informed consent from the patient for submission of this manuscript for publication.

## Ethical Statement/Human and Animal Rights

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and institutional guidelines for the reporting of clinical cases. Informed consent was obtained from the patient for publication, including the use of relevant clinical data and imaging. Given the retrospective nature of this report, formal approval from an Institutional Review Board was not required. No animal studies were conducted in relation to this research.

## Disclosures/Conflict Of Interest

The Authors Declare No Conflicts Of Interest In Relation To This Study. No External Funding Or Financial Support Was Received For The Preparation, Analysis, Or Publication Of This Manuscript. All Authors Contributed Substantially To The Conception, Data Collection, Analysis, And Writing Of This Case Report And Have Approved The Final Version Of The Manuscript For Submission.

## ABSTRACT

**Introduction:** Acute pancreatitis is a common inflammatory disorder of the pancreas, typically presenting with sudden upper abdominal pain, nausea, and vomiting. Despite its well-documented classic presentation, atypical manifestations can lead to initial misdiagnosis and complicate the clinical course.

**Case Presentation:** A 42-year-old female presented to the emergency department with left flank pain and watery diarrhea, initially treated as gastroenteritis or renal colic. Despite treatment with NSAIDs (ketorolac) and intravenous fluids, her pain persisted and worsened. Laboratory tests revealed an elevated white blood cell count ( $23 \times 10^3/\mu\text{L}$ ), normal pH and electrolytes, and a CRP of 7 mg/dL. Serum lipase was significantly elevated ( $>3000 \text{ U/L}$ ), and ultrasound imaging showed a bulky head and body of the pancreas with minimal peripancreatic fluid, confirming the diagnosis of acute pancreatitis. MRCP performed on day 4 revealed acute interstitial pancreatitis with focal changes in the body of the pancreas and minimal pancreatic fluid. The patient was managed conservatively and discharged on day 9.

**Discussion:** This case illustrates an atypical presentation of acute pancreatitis with flank pain and diarrhea, emphasizing the need for high clinical suspicion and thorough diagnostic evaluation. Acute pancreatitis can present with a wide range of symptoms, complicating timely diagnosis and management. While elevated serum lipase and characteristic imaging findings are diagnostic, atypical presentations may delay appropriate treatment. The patient's condition improved with conservative management, underscoring the importance of recognizing and managing atypical presentations of this condition.

**Conclusion:** This case highlights the diverse clinical manifestations of acute pancreatitis and the importance of considering this diagnosis even in the absence of typical symptoms. Increased awareness and further research are needed to understand the full spectrum of atypical presentations, ensuring prompt and accurate diagnosis and management.

## BACKGROUND

Acute pancreatitis is a well-recognized inflammatory disorder of the pancreas, most commonly presenting with sudden onset of severe epigastric pain, nausea, and vomiting. However, atypical manifestations pose a significant diagnostic challenge and can lead to delays in appropriate treatment. This case illustrates an unusual presentation of acute pancreatitis, initially misdiagnosed as gastroenteritis or renal colic due to predominant left flank pain and diarrhea, without the classic symptom of epigastric pain. The absence of traditional warning signs and the overlap with other gastrointestinal and renal conditions underscores the complexity of diagnosing atypical acute pancreatitis in emergency settings.

This case is particularly significant as it highlights the necessity of considering pancreatitis in the differential diagnosis of unexplained and persistent abdominal or flank pain, even when initial clinical suspicion is low. The importance of early laboratory evaluation, particularly serum lipase measurement, is evident in preventing delays in diagnosis. This case further demonstrates that imaging alone may not always be sufficient for early recognition, as conventional abdominal ultrasound was suggestive but not definitive. The utilization of MRCP on day four provided additional diagnostic clarity, reinforcing the role of advanced imaging in cases with atypical presentations.

The contribution of this case to the literature lies in its reinforcement of the growing recognition that acute pancreatitis does not always adhere to classic symptomatology. Although previous reports have documented atypical presentations, this case emphasizes the critical need for heightened clinical suspicion in patients with persistent or unexplained gastrointestinal symptoms. It also serves as a reminder that diagnostic pathways based on classical symptom profiles may not be universally applicable, and reliance on a rigid diagnostic framework can contribute to misdiagnosis. A broader perspective on symptom variability, coupled with timely laboratory investigations, can mitigate the risk of diagnostic delays and improve patient outcomes.

## INTRODUCTION

Acute pancreatitis, an inflammatory disorder of the pancreas, is one of the leading causes of abdominal pain contributing to significant mortality and morbidity worldwide [1]. It typically presents as a sudden intense pain in the upper abdomen (80-85%), radiating often to the back, accompanied by nausea and vomiting (40%) [1]. Fever, breathlessness, irritability, impaired consciousness and abdominal distension are often, but not always, associated with the typical presentation. Because of its similarity in clinical presentation to numerous other acute illnesses, diagnosis is made if 2 of the following 3 criteria are positive: (1) Typical abdominal pain, (2) Serum amylase/lipase  $\geq 3$  upper limit of normal, and (3) Characteristic imaging findings [2]. Despite the critical nature of the illness and heavy disease burden, there is no specific drug therapy that can change the course of acute pancreatitis, and management depends on timely recognition of the disease and its complications to prevent both short-term and long-term morbidity [1].

Literature indicates that while the classic presentation is well-documented, cases with symptoms such as isolated back pain, left-sided flank pain [9], and even scrotal pain [10] and inguinal swelling [12], can occur. These atypical manifestations often lead to initial misdiagnosis, ranging from renal colic to myocardial infarction, thus complicating the clinical course.

We present a case of a 42 year old female, who came to the emergency department with complaints of left flank pain and watery diarrhea. She was initially treated as a case of gastroenteritis vs renal colic, unresolving pain prompted the physician to order lipase which, along with the ultrasound findings, confirmed the diagnosis of acute pancreatitis. While there are case reports of atypical presentation of acute pancreatitis in literature, we believe more research is needed to increase awareness in considering the possibility of acute pancreatitis in patients with unresolving and unexplained symptomatology

## CASE PRESENTATION

A 42 year old lady presented to the emergency department with left flank pain and watery diarrhea for 1 day. Pain was sudden in onset, colicky, and was associated with nausea. She did not have fever, epigastric pain, vomiting, shortness of breath, dysuria. The patient denied any history of alcohol consumption, previous gall bladder disease, or illicit drug use. Past medical history was insignificant, and she was not on any medication. Physical examination was positive for left flank tenderness with voluntary guarding. She was vitally stable, afebrile.

Patient was given ketorolac 30mg and was started on intravenous fluids on the presumptive diagnosis of ureteric colic vs acute gastroenteritis. POCUS was negative for hydronephrosis. Initial measures didn't result in any improvement and her pain progressively worsened. Laboratory results included an elevated white blood cell count ( $23 \times 10^3/\mu\text{L}$ ), normal pH and electrolytes, and CRP of 7mg/dL. Serum lipase was ordered on account of non-resolving and non-specific flank pain and its value turned out to be  $>3000$  U/L. Abdominal ultrasound showed bulky head and body of pancreas with minimal peripancreatic fluid. She was admitted in the medicine ward with the diagnosis of acute pancreatitis.

MRCP done on day 4 of admission showed acute interstitial pancreatitis along with focal changes in the body of pancreas, with minimal pancreatic fluid and free fluid in the abdomen and CBD was dilated (1.1cm) till lower lobe. BISAP score remained 0 throughout the hospital stay. She was managed conservatively and was discharged on day 9.

## DISCUSSION

Acute pancreatitis is the inflammation of the pancreas caused by autodigestion of pancreatic parenchyma by pancreatic enzymes resulting in hemorrhage and necrosis. Pancreatic enzymes are normally released in inactive forms and are converted to active form in the duodenum. But certain conditions disrupt this protective mechanism causing premature activation of the autodigestive enzymes, triggering a cascade of inflammatory events that may lead to sepsis or acute respiratory

distress syndrome (ARDS) [3]. In spite of advancement in healthcare access, diagnostic and interventional techniques, acute pancreatitis remains a significant contributor to mortality and morbidity worldwide [4].

Alcohol and gallstones are the most common causative factors with trauma, hyperlipidemia, drugs, scorpion stings, post ERCP, sphincter of Oddi dysfunction and hypercalcemia also implicated. Common drugs causing pancreatitis include statins, amiodarone, thiazides, azathioprine, valproic acid, and didanosine [5].

Common clinical features include epigastric pain which is usually sharp and constant, moderate to severe in intensity, and occasionally radiating to the back. Low grade fever, abdominal distension and dyspnea are also commonly reported [5]. History of gallstones and alcohol intake, family history of hyperlipidemia, are important clues in the diagnostic process as the management differs in each case [4].

Uncommon symptomatology of acute pancreatitis reported in the literature include flank pain [9], scrotal ecchymosis (Bryant sign) [10], and rarely intrathoracic collections [11], and inguinoscrotal swelling [12]. Sabrina et al reported a case of acute pancreatitis manifesting solely as a septic fluid collection in the inguinal canal leading to the patient being misdiagnosed as inguinal hernia [13]. Symptomatic bradycardia (a form of viscerovisceral reaction) as the presenting complaint of acute pancreatitis has also been described, highlighting the necessity to keep a high index of suspicion of acute pancreatitis in patients with unresolving or vague abdominal pain [14].

Prompt diagnosis and severity assessment are integral to prevent morbidity and mortality. Diagnosis is done by clinical features, elevated serum lipase (>3 times upper limit of normal), with imaging done in diagnostic uncertainty and to look for complications [5]. Lipase is preferred over amylase owing to its increased sensitivity and earlier detection. Concomitant detection of amylase and lipase levels offers no significant improvement in mortality [4]. Serum triglycerides greater than 1000mg/dL suggest hyperlipidemia as the cause.

Cases of acute pancreatitis with normal amylase and lipase levels have been reported in the literature [5]. Serum trypsinogen activation peptide and trypsinogen-2 are more specific and early markers of acute pancreatitis but are not readily available and are costly to perform [7].

Misdiagnosis and delayed diagnosis of acute pancreatitis occurs because of delayed ED care, normal serum biochemistry, and, more rarely, absence of abdominal pain or symptoms related to the gastrointestinal tract [8]. Covino et al reported 2.6% cases of acute atypical pancreatitis in a population of Finland, defined as any symptomatology in the absence of typical abdominal pain. Factors leading to atypical acute pancreatitis include advanced age, systemic disease, and any distracting illnesses present. There is no significant difference in mortality associated with conventional and atypical acute pancreatitis [8].

Management of acute pancreatitis includes fluid resuscitation, analgesia, anti-emetics and addressing the underlying cause. Cholecystectomy is indicated for pancreatitis caused by gallstones. Biliary pancreatitis without cholangitis and choledocholithiasis, doesn't necessarily need ERCP [5]. Insulin infusion and plasmapheresis are treatment options for hypertriglyceridemia induced pancreatitis.

This case helps us understand the diverse clinical presentation of acute pancreatitis and underscores the need for heightened awareness and thorough diagnostic evaluation, particularly in case of non-specific abdominal pain. Further exploration is needed to understand the full spectrum of atypical presentations of acute pancreatitis.

### QUESTIONS

1. What are the typical symptoms of acute pancreatitis, and how did the patient in this case report present atypically?
2. What diagnostic tools can be used to confirm acute pancreatitis, and how did they assist in this case?
3. Why is early recognition of atypical symptoms in acute pancreatitis important for patient outcomes?
4. How does conservative management play a role in the treatment of acute pancreatitis, as highlighted by this case?
5. What are some of the rare presentations of acute pancreatitis, and how can they complicate the diagnostic process?

### Question 1: Which of the following statements about imaging for acute pancreatitis is TRUE?

1. Non-contrast CT is preferred for detecting pancreatic necrosis.
2. Ultrasound is the best imaging modality for evaluating pancreatic pseudocysts.
3. CT with IV contrast is used to assess the severity of acute pancreatitis.
4. Plain radiographs are highly sensitive for identifying pancreatic calcifications.
5. MRCP is generally not used for diagnosing biliary obstructions.

### Explanation:

1. [Non-contrast CT lacks the sensitivity to detect necrosis. Contrast-enhanced CT is required for this purpose.]
2. [Ultrasound can detect pseudocysts, but it is limited in resolution compared to CT or MRI, which are better for detailed evaluation.]
3. **CT with IV contrast is used to assess the severity of acute pancreatitis. (applies)** [Contrast-enhanced CT (CECT) is the gold standard for assessing the severity of acute pancreatitis, particularly in cases where necrosis or fluid collections need to be identified.]
4. [Plain radiographs can show calcifications, but they are not the most sensitive modality compared to CT, which offers much better resolution.]
5. [MRCP is, in fact, highly useful for diagnosing biliary obstructions and is often used to assess for gallstones and strictures.]

**Question 2: Which of the following statements about the role of MRI in acute pancreatitis is FALSE?**

1. MRI is typically more expensive and time-consuming than CT in the acute setting.
2. MRI is preferred over CT in evaluating pancreatic necrosis.
3. MRI is superior in visualizing soft tissue structures and detecting fluid collections.
4. MRCP (Magnetic Resonance Cholangiopancreatography) is highly sensitive for biliary obstruction detection.
5. MRI is generally not used as the first-line imaging modality in acute pancreatitis.

**Explanation:**

1. [This is true; MRI is more expensive and takes longer to perform compared to CT, especially in emergency settings.]
2. **MRI is preferred over CT in evaluating pancreatic necrosis. (applies) [Contrast-enhanced CT (CECT), not MRI, is the gold standard for detecting pancreatic necrosis, as it provides superior detail regarding perfusion deficits in the pancreatic tissue.]**
3. [This is true. MRI is better for soft tissue contrast and can detect fluid collections, but it is not the first-line modality for pancreatitis.]
4. [True. MRCP is highly sensitive and is frequently used to evaluate biliary obstructions, such as choledocholithiasis.]
5. [This is correct; CT is typically preferred for initial assessment of acute pancreatitis, while MRI is reserved for specific cases.]

**Question 3: Which of the following statements about the use of ultrasound in acute pancreatitis is FALSE?**

1. Ultrasound is commonly used to detect gallstones in patients with biliary pancreatitis.
2. Ultrasound can be limited by bowel gas, affecting the visualization of the pancreas.
3. Ultrasound is useful in detecting peripancreatic fluid collections.
4. Ultrasound is the imaging modality of choice for identifying pancreatic necrosis.
5. Ultrasound can assist in identifying complications such as biliary duct obstructions.

**Explanation:**

1. [True. Ultrasound is the first-line modality for detecting gallstones, especially in suspected biliary pancreatitis.]
2. [This is true. Ultrasound has limitations due to bowel gas and can struggle to fully visualize the pancreas.]
3. [True. Ultrasound is often used to detect fluid collections, but its sensitivity is lower compared to CT.]
4. **Ultrasound is the imaging modality of choice for identifying pancreatic necrosis. (applies) [False. Contrast-enhanced CT is the gold standard for detecting necrosis. Ultrasound lacks the resolution to evaluate necrotic tissue.]**
5. [True. Ultrasound is commonly used to detect complications related to the biliary system, such as obstructions.]

**Question 4: Which imaging modality is the most sensitive for detecting pancreatic calcifications in chronic pancreatitis?**

1. Ultrasound
2. Non-contrast CT
3. Contrast-enhanced CT
4. MRI
5. Plain abdominal radiograph

**Explanation:**

1. [Ultrasound is not as sensitive for detecting calcifications, especially when compared to CT or plain radiographs.]
2. [Non-contrast CT can detect calcifications, but it is not as effective as plain radiographs in detecting chronic calcifications in the pancreas.]
3. [CECT is excellent for evaluating necrosis and fluid collections but is not the most sensitive for detecting chronic calcifications.]
4. [MRI is not typically used for detecting calcifications, as it is better suited for soft tissue and fluid imaging.]
5. **Plain abdominal radiograph (applies) [True. Plain radiographs are often the best modality for detecting chronic pancreatic calcifications.]**

**Question 5: Which of the following is the main advantage of MRCP over CT in evaluating patients with acute pancreatitis?**

1. MRCP is faster than CT in emergency settings.
2. MRCP is more cost-effective than CT in detecting complications.
3. MRCP provides better visualization of pancreatic necrosis.
4. MRCP is non-invasive and superior for evaluating biliary duct obstructions.
5. MRCP is preferred for detecting pancreatic pseudocysts.

**Explanation:**

- [MRCP takes longer and is less available in acute settings than CT, which is preferred for emergencies.]
  - [MRCP is generally more expensive than CT and is not used for cost reasons in detecting complications.]
  - [CECT, not MRCP, is the gold standard for detecting necrosis.]
  - **MRCP is non-invasive and superior for evaluating biliary duct obstructions. (applies) [True. MRCP is non-invasive and highly sensitive for detecting obstructions in the biliary system.]**
- [CT is better for detecting pseudocysts, while MRCP is used for evaluating ductal systems.]

**TEACHING POINT**

This case emphasizes the importance of considering acute pancreatitis in patients with atypical presentations, such as left flank pain and diarrhea, which may initially mimic conditions like gastroenteritis or renal colic. Early recognition of atypical



symptoms and the use of diagnostic tools such as serum lipase and imaging can prevent misdiagnosis and ensure timely management, potentially avoiding complications associated with delayed treatment.

## REFERENCES

1. Lankisch PG, Apte M, Banks PA. Acute pancreatitis. *The Lancet*. 2015; 386(9988): 85–96.
2. Szatmary P, Grammatikopoulos T, Cai W, et al. Acute Pancreatitis: Diagnosis and Treatment. *Drugs*. 2022; 82(12): 1251–1276. PMID: 36074322.
3. Steer ML. Pathogenesis of acute pancreatitis. *Digestion*. 1997; 58: 46–49.
4. Greenberg JA, Hsu J, Bawazeer M, et al. Clinical practice guideline: management of acute pancreatitis. *Can J Surg*. 2016; 59(2):128–140. PMID: 27007094.
5. Banks PA. Epidemiology, natural history, and predictors of disease outcome in acute and chronic pancreatitis. *Gastrointest Endosc*. 2002; 56(6): S226–S230. PMID: 12447272.
6. Nadhem O, Salh O. Acute Pancreatitis: An Atypical Presentation. *Case Rep Gastroenterol*. 2017;11(2): 359–363. PMID: 28626384.
7. Shah AM, Eddi R, Kothari ST, Maksoud C, DiGiacomo WS, Baddour W. Acute pancreatitis with normal serum lipase: a case series. *JOP*. 2010; 11: 369–372. PMID: 20601812.
8. Covino M, Quero G, Ojetti V, et al. Atypical presentation of acute pancreatitis: a single center case-match analysis of clinical outcomes. *Eur Rev Med Pharmacol Sci*. 2020; 24(2): 813–820. PMID: 32016986.
9. Chen JH, Chern CH, Chen JD, How CK, Wang LM, Lee CH. Left flank pain as the sole manifestation of acute pancreatitis: a report of a case with an initial misdiagnosis. *Emerg Med J*. 2005; 22(6): 452–453. PMID: 15911961.
10. Ferreres Serafini J, Elvira López J, Memba Ikuga R, Jorba Martín R. Bryant sign, an atypical presentation of an acute severe pancreatitis. *Cir Esp (Engl Ed)*. 2022;100(9):586. PMID: 35700890.
11. C Alonso Belmonte, B Parra López. Large intrathoracic collection secondary to acute chronic pancreatitis displacing large hiatal hernia, an uncommon presentation. *Revista andaluza de patología digestiva online*. 2024; 47(2): 84–86.
12. Nazar MA, D'Souza FR, Ray A, Memon MA. Unusual presentation of acute pancreatitis: an irreducible inguinoscrotal swelling mimicking a strangulated hernia. *Abdominal radiology. Abdom Imaging*. 2007; 32(1): 116–118. PMID: 16680509.
13. Brar S, Watters C. A real ball ache: a case report of acute pancreatitis with an unusual sequelae of events. *J Surg Case Rep*. 2020; 2020(7): rjaa199. PMID: 32665840.
14. Mathew TL, Gonzales G, Hoang L. S1449 Symptomatic Bradycardia: An Unusual Presentation of Acute Pancreatitis. *The American journal of gastroenterology*. 2020;115(1): S694–4.

FIGURES

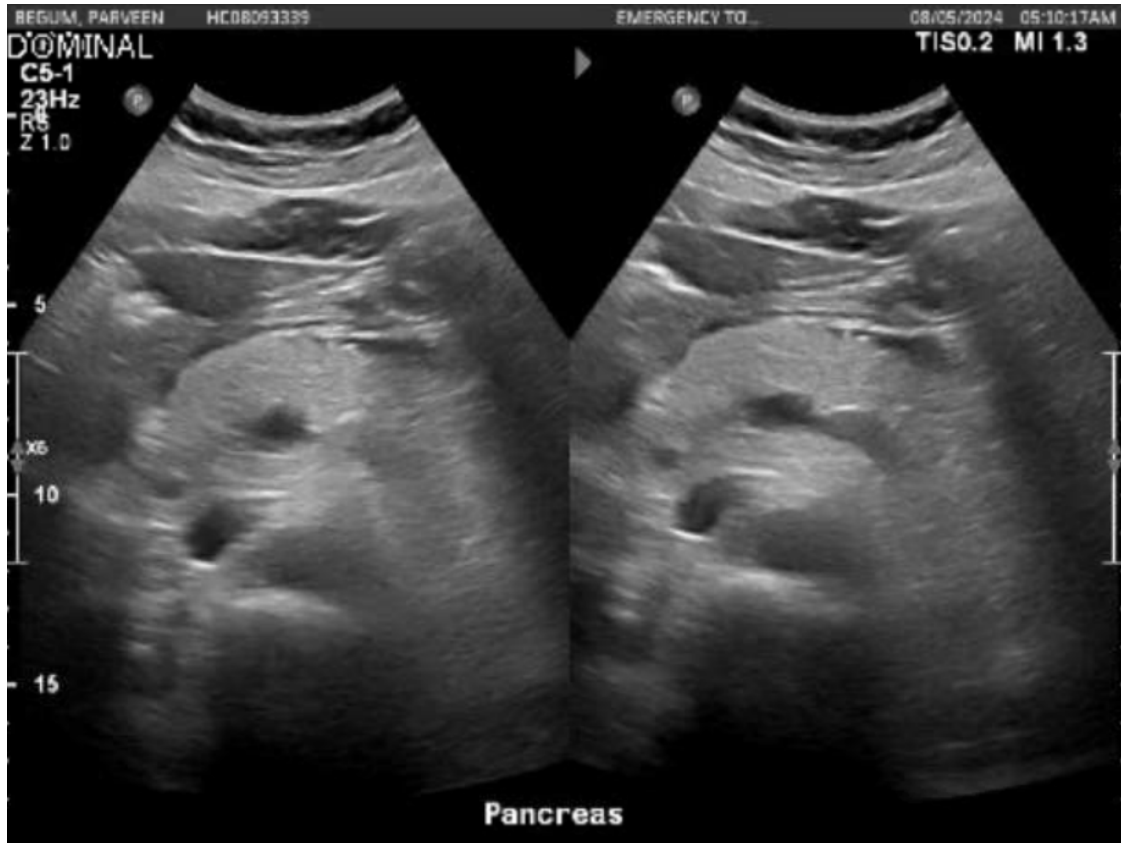
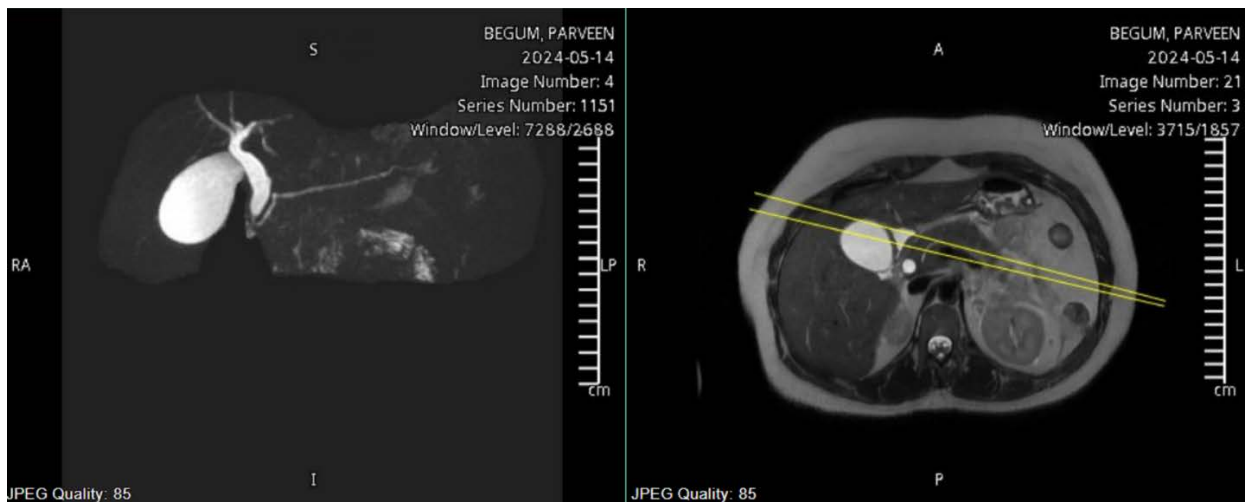


Figure 1: Abdominal ultrasound of 42 year old female. Acute Pancreatitis- Enlarged pancreas surrounded a rim of peri-pancreatic fluid.



Figure 2: Doppler Ultrasound imaging shows no signal in the small rim of peri-pancreatic fluid



**Figure 3:** MRCP. 42 year old female diagnosed with acute pancreatitis. There is abnormal signal intensity in the body of pancreas suggestive of focal pancreatitis changes along with diffuse interstitial pancreatitis changes. There is minimal peripancreatic fluid noted

**Table 1:** Laboratory values at admission and after 24 hours

Laboratory Parameter	Day 1	Day 2
WBC ( $\times 10^3/\mu\text{L}$ )	23	19
Hb (g/dL)	11.8	11.1
Platelets ( $\times 10^3/\mu\text{L}$ )	330	281
Urea (mmol/L)	4	2.2
Creatinine ( $\mu\text{mol/L}$ )	93	61
Glucose (mmol/L)	7.8	
CRP (mg/dL)	7.1	300
Lipase (U/L)	3000	1285
Calcium (mmol/L)	2.34	2.24
Triglycerides (mmol/L)	0.9	
ALT (U/L)	17	33
AST (U/L)	12	12

**KEYWORDS**

Acute Pancreatitis, Atypical Presentation, Flank Pain, Diarrhea, Serum Lipase, Conservative Management, Imaging, Gastroenteritis, Renal Colic

**ABBREVIATIONS**

WBC = White Blood Cell

CRP = C-Reactive Protein

MRCP = Magnetic Resonance Cholangiopancreatography

CBD = Common Bile Duct

ERCP = Endoscopic Retrograde Cholangiopancreatography

ARDS = Acute Respiratory Distress Syndrome

ALT = Alanine Aminotransferase

AST = Aspartate Aminotransferase

BISAP = Bedside Index for Severity in Acute Pancreatitis

POCUS = Point-of-Care Ultrasound

**Online access**

This publication is online available at:

[www.radiologycases.com/index.php/radiologycases/article/view/5436](http://www.radiologycases.com/index.php/radiologycases/article/view/5436)

**Peer discussion**

Discuss this manuscript in our protected discussion forum at:

[www.radiolopolis.com/forums/JRCR](http://www.radiolopolis.com/forums/JRCR)

**Interactivity**

This publication is available as an interactive article with scroll, window/level, magnify and more features.

Available online at [www.RadiologyCases.com](http://www.RadiologyCases.com)

Published by EduRad



[www.EduRad.org](http://www.EduRad.org)