International Surgery Journal
*Choudhury NA et al. Int Surg J. 2026 Jan;13(1):158-162*
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: https://dx.doi.org/10.18203/2349-2902.isj20254347

**Case Report**

# Chyle leak after laparoscopic cholecystectomy in a patient with necrotizing pancreatitis: reporting a rare case

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**Received:** 11August 2025
**Revised:** 16 December 2025
**Accepted:** 16 December 2025

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## ABSTRACT

Chyle leak after laparoscopic cholecystectomy is an extremely rare post operative complication, usually related to inadvertent lymphatic injury in patients with distorted anatomy. We report a 70-year-old male with type 2 diabetes mellitus, hypothyroidism, and a recent history of necrotizing pancreatitis, who underwent elective laparoscopic cholecystectomy. Intraoperatively, frozen Calot's anatomy and dense adhesions were encountered. A postoperative subhepatic drain revealed milky fluid, which was confirmed biochemically as chyle. The patient was successfully managed conservatively with dietary modifications, medium-chain triglyceride supplementation, and octreotide. Although rare, chyle leak should be considered in patients with postoperative milky drain output after laparoscopic cholecystectomy. Conservative management is effective in most cases, but requires early recognition, multidisciplinary input, and close follow-up.

**Keywords:** Chyle leak, Laparoscopic cholecystectomy, Necrotizing pancreatitis, Conservative management

## INTRODUCTION

Laparoscopic cholecystectomy is one of the most frequently performed surgical procedures worldwide, with a well-established safety profile. However, rare complications such as chyle leak can occur. This entity is typically associated with intraoperative injury to lymphatic channels, particularly in patients with altered anatomy or dense adhesions from prior inflammation.[1-3] Chyle leak usually manifests as milky postoperative drain output, confirmed by high triglyceride content in the fluid. While uncommon, reported cases demonstrate that conservative measures, including dietary modification and somatostatin analogues, can be highly effective.[2-5]

A rare case of chyle leak following laparoscopic cholecystectomy in a patient with necrotizing pancreatitis, successfully managed without the need for re-operation.

## CASE REPORT

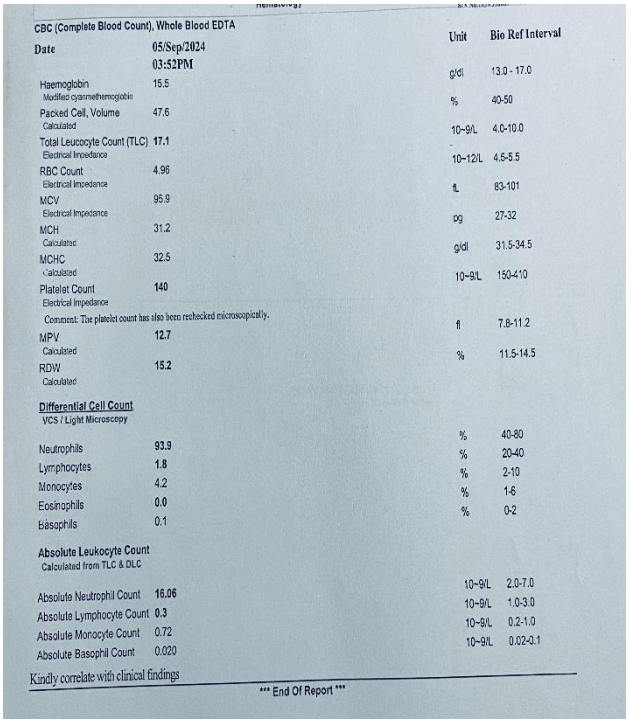
A 70-year-old male with a background of type 2 diabetes mellitus and hypothyroidism presented to the emergency department with diffuse abdominal pain radiating to the back, recurrent vomiting, progressive abdominal distension, and inability to pass stools or flatus for 1 day. There was no history of trauma, alcohol intake, or previous abdominal surgery.

### *Examination*

Patient was conscious, oriented to time, place and person. No icterus was noted. Vitals: BP - 160/70 mmHg, pulse rate- 104 bpm, $SpO_2$ 94% on room air. Per abdominal examination - Abdomen distended with right upper quadrant tenderness, tympanic on percussion, without guarding or rigidity. Bowel sounds were sluggish.

### *Investigations*

Laboratory findings included leukocytosis (TLC 17.1 × $10^3$/µl), elevated serum amylase (2074 U/l), lipase (1520 U/l), CRP (431.8 mg/dl), and deranged liver function tests. Electrolyte abnormalities included hyponatremia (Na 128.8 mmol/l) and hypocalcemia (7.7 mg/dl). Random blood sugar level- 218 mg/dl.



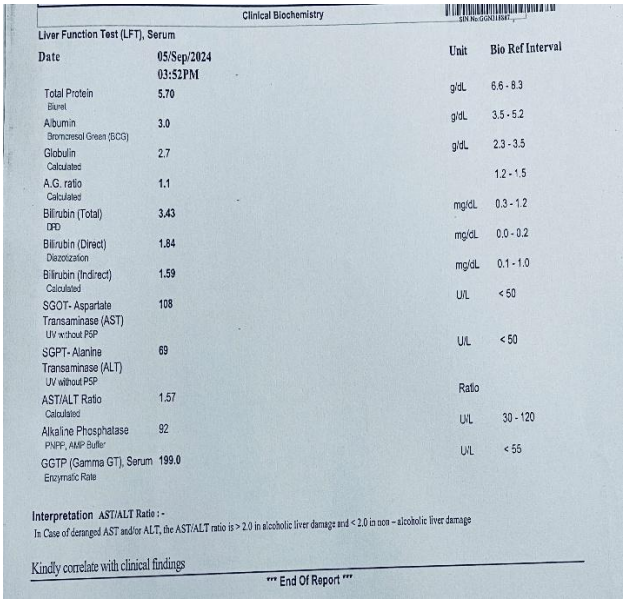
CBC (Complete Blood Count), Whole Blood EDTA

| Date | 05/Sep/2024 03:52PM | Unit | Bio Ref Interval |
|---|---|---|---|
| Haemoglobin (Modified cyanmethemoglobin) | 15.5 | g/dl | 13.0 - 17.0 |
| Packed Cell, Volume (Calculated) | 47.6 | % | 40-50 |
| Total Leucocyte Count (TLC) (Electrical Impedance) | 17.1 | 10~9/L | 4.0-10.0 |
| RBC Count (Electrical Impedance) | 4.96 | 10~12/L | 4.5-5.5 |
| MCV (Electrical Impedance) | 95.9 | fL | 83-101 |
| MCH (Calculated) | 31.2 | pg | 27-32 |
| MCHC (Calculated) | 32.5 | g/dl | 31.5-34.5 |
| Platelet Count (Electrical Impedance) | 140 | 10~9/L | 150-410 |
| Comment: The platelet count has also been rechecked microscopically. | | | |
| MPV (Calculated) | 12.7 | fl | 7.8-11.2 |
| RDW (Calculated) | 15.2 | % | 11.5-14.5 |
| **Differential Cell Count** (VCS / Light Microscopy) | | | |
| Neutrophils | 93.9 | % | 40-80 |
| Lymphocytes | 1.8 | % | 20-40 |
| Monocytes | 4.2 | % | 2-10 |
| Eosinophils | 0.0 | % | 1-6 |
| Basophils | 0.1 | % | 0-2 |
| **Absolute Leukocyte Count** (Calculated from TLC & DLC) | | | |
| Absolute Neutrophil Count | 16.06 | 10~9/L | 2.0-7.0 |
| Absolute Lymphocyte Count | 0.3 | 10~9/L | 1.0-3.0 |
| Absolute Monocyte Count | 0.72 | 10~9/L | 0.2-1.0 |
| Absolute Basophil Count | 0.020 | 10~9/L | 0.02-0.1 |

Kindly correlate with clinical findings

*** End Of Report ***

**Figure 1: Complete blood count report.**



Clinical Biochemistry

Liver Function Test (LFT), Serum

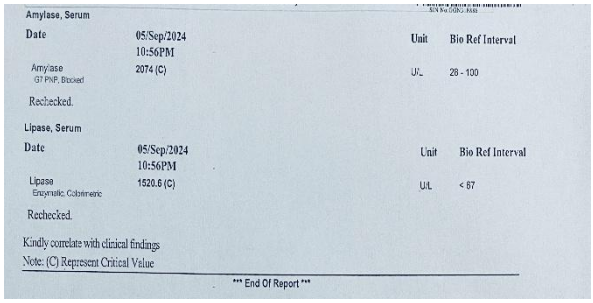
| Date | 05/Sep/2024 03:52PM | Unit | Bio Ref Interval |
|---|---|---|---|
| Total Protein (Biuret) | 5.70 | g/dL | 6.6 - 8.3 |
| Albumin (Bromocresol Green (BCG)) | 3.0 | g/dL | 3.5 - 5.2 |
| Globulin (Calculated) | 2.7 | g/dL | 2.3 - 3.5 |
| A:G ratio (Calculated) | 1.1 | | 1.2 - 1.5 |
| Bilirubin (Total) (DPD) | 3.43 | mg/dL | 0.3 - 1.2 |
| Bilirubin (Direct) (Diazotization) | 1.84 | mg/dL | 0.0 - 0.2 |
| Bilirubin (Indirect) (Calculated) | 1.59 | mg/dL | 0.1 - 1.0 |
| SGOT- Aspartate Transaminase (AST) (UV without P5P) | 108 | U/L | < 50 |
| SGPT- Alanine Transaminase (ALT) (UV without P5P) | 69 | U/L | < 50 |
| AST/ALT Ratio (Calculated) | 1.57 | Ratio | |
| Alkaline Phosphatase (PNPP, AMP Buffer) | 92 | U/L | 30 - 120 |
| GGTP (Gamma GT), Serum (Enzymatic Rate) | 199.0 | U/L | < 55 |

Interpretation AST/ALT Ratio :-
In Case of deranged AST and/or ALT, the AST/ALT ratio is > 2.0 in alcoholic liver damage and < 2.0 in non – alcoholic liver damage

Kindly correlate with clinical findings

*** End Of Report ***

**Figure 2: Liver function test report.**



Amylase, Serum

| Date | 05/Sep/2024 10:56PM | Unit | Bio Ref Interval |
|---|---|---|---|
| Amylase (GT PNP, Blocked) | 2074 (C) | U/L | 28 - 100 |

Rechecked.

Lipase, Serum

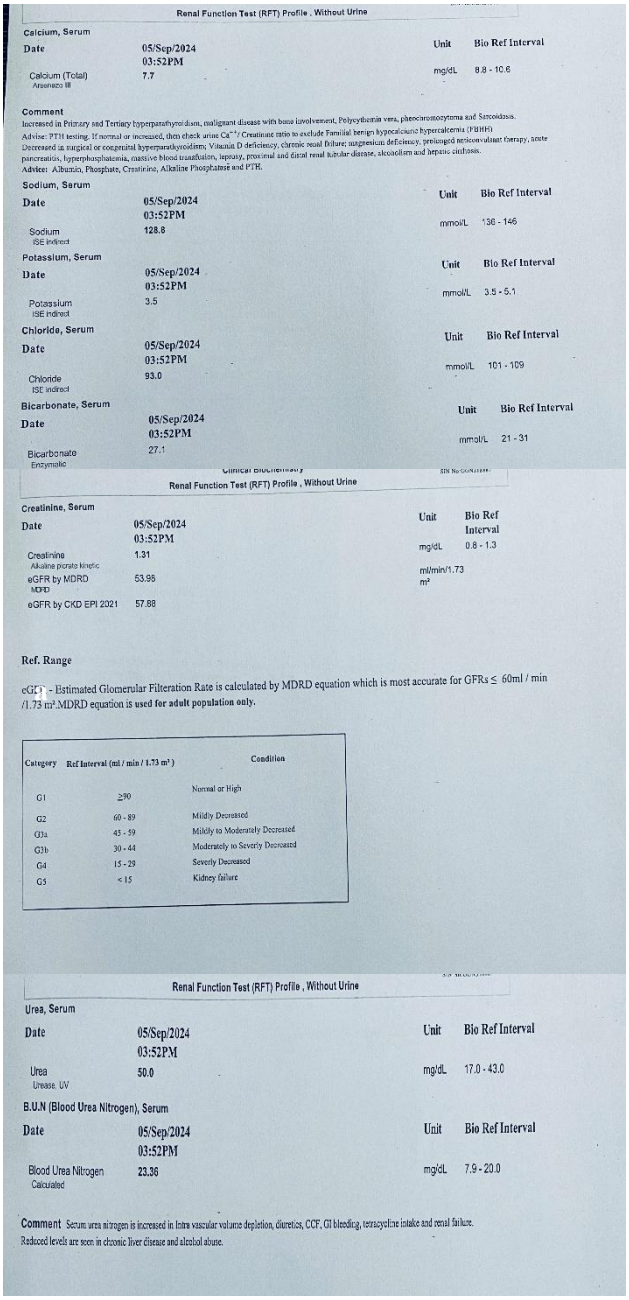
| Date | 05/Sep/2024 10:56PM | Unit | Bio Ref Interval |
|---|---|---|---|
| Lipase (Enzymatic, Colorimetric) | 1520.6 (C) | U/L | < 67 |

Rechecked.

Kindly correlate with clinical findings
Note: (C) Represent Critical Value

*** End Of Report ***

**Figure 3: Serum amylase and lipase report.**



Renal Function Test (RFT) Profile , Without Urine

Calcium, Serum

| Date | 05/Sep/2024 03:52PM | Unit | Bio Ref Interval |
|---|---|---|---|
| Calcium (Total) (Arsenazo III) | 7.7 | mg/dL | 8.8 - 10.6 |

Comment
Increased in Primary and Tertiary hyperparathyroidism, malignant disease with bone involvement, Polycythemia vera, pheochromocytoma and Sarcoidosis.
Advise: PTH testing. If normal or increased, then check urine $Ca^{++}$/Creatinine ratio to exclude Familial benign hypocalciuric hypercalcemia (FBHH)
Decreased in surgical or congenital hyperparathyroidism, Vitamin D deficiency, chronic renal failure; magnesium deficiency, prolonged anticonvulsant therapy, acute pancreatitis, hyperphosphatemia, massive blood transfusion, leprosy, proximal and distal renal tubular disease, alcoholism and hepatic cirrhosis.
Advice: Albumin, Phosphate, Creatinine, Alkaline Phosphatase and PTH.

Sodium, Serum

| Date | 05/Sep/2024 03:52PM | Unit | Bio Ref Interval |
|---|---|---|---|
| Sodium (ISE Indirect) | 128.8 | mmol/L | 136 - 146 |

Potassium, Serum

| Date | 05/Sep/2024 03:52PM | Unit | Bio Ref Interval |
|---|---|---|---|
| Potassium (ISE Indirect) | 3.5 | mmol/L | 3.5 - 5.1 |

Chloride, Serum

| Date | 05/Sep/2024 03:52PM | Unit | Bio Ref Interval |
|---|---|---|---|
| Chloride (ISE Indirect) | 93.0 | mmol/L | 101 - 109 |

Bicarbonate, Serum

| Date | 05/Sep/2024 03:52PM | Unit | Bio Ref Interval |
|---|---|---|---|
| Bicarbonate (Enzymatic) | 27.1 | mmol/L | 21 - 31 |

Renal Function Test (RFT) Profile , Without Urine

Creatinine, Serum

| Date | 05/Sep/2024 03:52PM | Unit | Bio Ref Interval |
|---|---|---|---|
| Creatinine (Alkaline picrate kinetic) | 1.31 | mg/dL | 0.8 - 1.3 |
| eGFR by MDRD (MDRD) | 53.98 | ml/min/1.73 m² | |
| eGFR by CKD EPI 2021 | 57.88 | | |

Ref. Range

eGFR - Estimated Glomerular Filteration Rate is calculated by MDRD equation which is most accurate for GFRs ≤ 60ml / min /1.73 m².MDRD equation is used for adult population only.

| Category | Ref Interval (ml / min / 1.73 m²) | Condition |
|---|---|---|
| G1 | ≥90 | Normal or High |
| G2 | 60 - 89 | Mildly Decreased |
| G3a | 45 - 59 | Mildly to Moderately Decreased |
| G3b | 30 - 44 | Moderately to Severly Decreased |
| G4 | 15 - 29 | Severly Decreased |
| G5 | < 15 | Kidney failure |

Renal Function Test (RFT) Profile , Without Urine

Urea, Serum

| Date | 05/Sep/2024 03:52PM | Unit | Bio Ref Interval |
|---|---|---|---|
| Urea (Urease, UV) | 50.0 | mg/dL | 17.0 - 43.0 |

B.U.N (Blood Urea Nitrogen), Serum

| Date | 05/Sep/2024 03:52PM | Unit | Bio Ref Interval |
|---|---|---|---|
| Blood Urea Nitrogen (Calculated) | 23.36 | mg/dL | 7.9 - 20.0 |

Comment Serum urea nitrogen is increased in intra vascular volume depletion, diuretics, CCF, GI bleeding, tetracycline intake and renal failure.
Reduced levels are seen in chronic liver disease and alcohol abuse.

**Figure 4: Renal function test reports.**

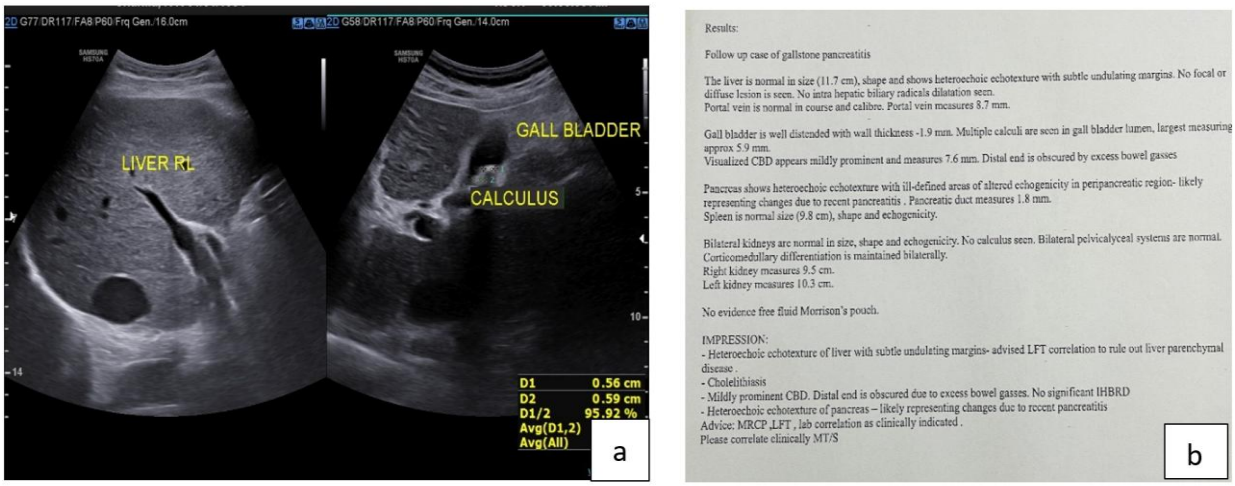



**Figure 5: USG whole abdomen. (a) Image, (b) report revealing multiple gallstones and a mildly prominent CBD.**

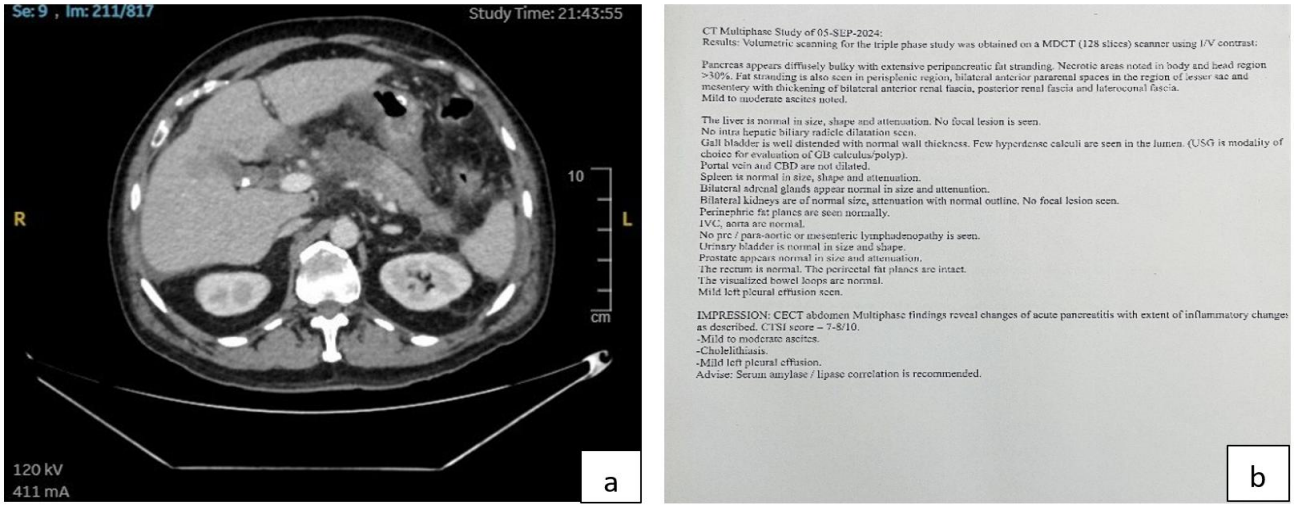



**Figure 6: (a) Image, (b) report of contrast enhanced multiphase CT- abdomen.**

Contrast-enhanced CT (CECT) of the abdomen showed - Acute necrotizing pancreatitis (CTSI score 7–8/10), mild ascites, cholelithiasis, and a left pleural effusion.

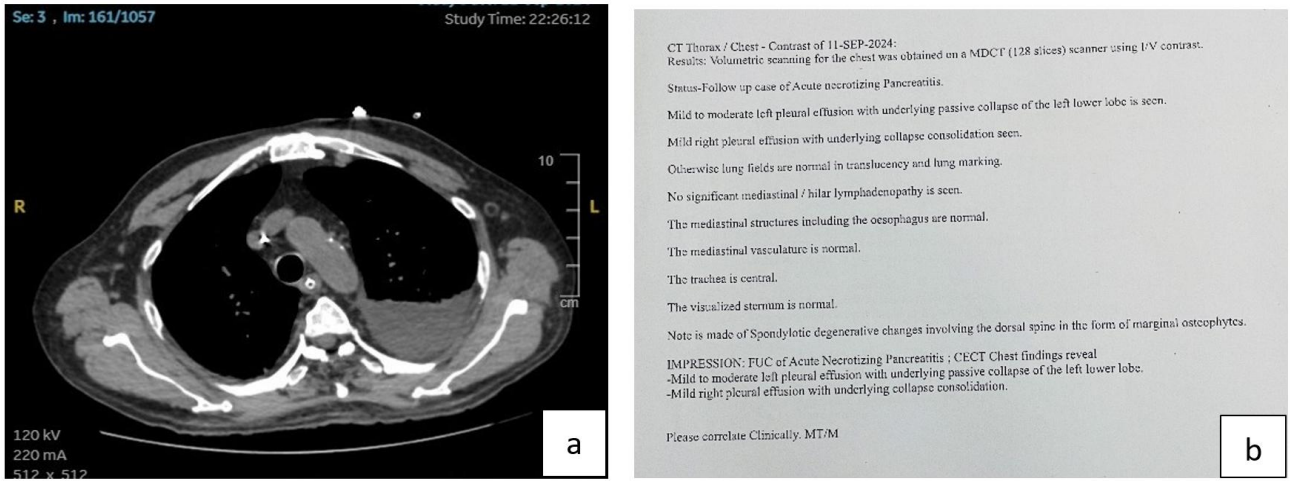



**Figure 7: (a) Image, (b) report of CT- Thorax- showing- mild to moderate left sided pleural effusion with underlying passive collapse of the left lower lobe. Mild right sided pleural effusion with underlying collapse consolidation was also noted.**

***Diagnosis***

Acute necrotizing pancreatitis secondary to gallstones.

***Initial management***

The patient was admitted to ICU and managed conservatively with aggressive IV fluids, kept nil per oral and nasogastric decompression, broad-spectrum antibiotics, analgesia, and electrolyte correction. His condition gradually improved and he was discharged with advice for interval cholecystectomy. Operative course and postoperative events: after 8 weeks, he underwent elective laparoscopic cholecystectomy. Dense omental and bowel adhesions to the gallbladder and obliteration of Calot's triangle were noted intraoperatively. A subhepatic drain was placed due to difficult dissection.

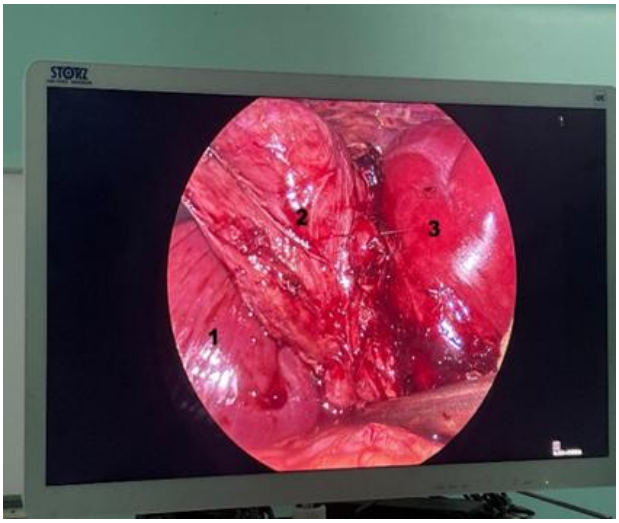



**Figure 8: Intra-op image: 1. right lobe of liver 2. gall bladder with frozen calot's region 3. left lobe of liver.**

The patient was discharged on postoperative day 2 with drain output of 200 ml (Serosanguinous fluids). By day 4, he returned with 400 ml of milky drain output, which was confirmed as chyle on biochemical testing.


**Figure 9: Drain- output (milky white in colour).**

| Test Name | Result |
|---|---|
| Chyle Examination-(L)*, Staining, Microscopy | |
| Type of Specimen: | Pancreatic fluid |
| Result. | Positive |
| Kindly correlate with clinical findings | |
| | *** End Of Report *** |

**Figure 10: Drain fluid analysis**

***Management of chyle leak***

Conservative management included high-protein, low-fat diet enriched with medium-chain triglycerides (MCTs) Supportive care and serial drain monitoring Subcutaneous octreotide initiated in week 2.

*Outcome:* Drain output progressively declined (90 ml/day at week 1, 40 ml/day at week 2 and 8 ml/day at week 3). The drain was removed, and the patient recovered fully without recurrence. Histopathology of gall bladder confirmed chronic cholecystitis with cholesterolosis.

## DISCUSSION

Chyle leak after laparoscopic cholecystectomy is extremely rare. The lymphatic system may be injured inadvertently during dissection, especially when the anatomy is distorted by inflammation or fibrosis, as was the case here.[3-5]

The diagnosis of a chyle leak is primarily clinical, with milky postoperative drain fluid being a hallmark. This is confirmed by elevated triglyceride levels in the fluid (>110 mg/dl).[7]

Initial management is conservative, including dietary modifications (low-fat diet with MCTs), which reduce lymph flow, as MCTs are absorbed directly into the portal venous system.[2-8] Pharmacologic therapy such as octreotide, a somatostatin analogue, can further reduce intestinal lymphatic output and hasten recovery.[6-8] Surgical or interventional options are considered only when conservative measures fail or the output remains high over an extended period. In this case, conservative management led to complete resolution without surgical intervention, consistent with outcomes reported by Jensen and Weiss[1], Gogalniceanu et al.[3], and Ong et al.[5]

## CONCLUSION

Chyle leak is an uncommon yet significant postoperative complication of laparoscopic cholecystectomy, particularly in patients with distorted biliary anatomy. Early diagnosis, close monitoring, dietary modifications, and the judicious use of octreotide can lead to successful conservative management and avoid invasive procedures.

A multidisciplinary approach is essential for optimal outcomes in such complex patients.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: Not required*

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**Cite this article as:** Choudhury NA, Jyoti V, Yadav R. Chyle leak after Laparoscopic Cholecystectomy in a patient with Necrotizing Pancreatitis: Reporting a rare case. Int Surg J 2026;13:158-62.