# Microlithiasis in Recurrent Acute Pancreatitis : Diagnosis and Management

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## INTRODUCTION

### Recurrent Acute Pancreatitis

Recurrent acute pancreatitis is often caused by excessive alcohol intake and bile stones. Patients suffering from more than one episode of acute pancreatitis are diagnosed with recurrent acute pancreatitis. The etiology of recurrent acute pancreatitis is known in 70 to 90% of patients after evaluation incorporating history taking, physical examination, routine laboratory assessment, and transabdominal ultrasonography or CT scan. However, the etiology of 10 to 30% of patients with recurrent acute pancreatitis remains undetermined. Further more sensitive evaluation is often performed, such as ERCP, ultrasound endoscopy, or MRCP, in order to determine the cause for recurrent acute pancreatitis. These tests are usually able to diagnose microlithiasis, sphincter oddi dysfunction, or pancreatic division.[3,4,5,6,7]

Medina- Perez M, Garcia Ferris G, Caballow Gomes J, Hospital de la Merced, and Ossuna Sevilla reported a rare case on a 33-year old woman with recurrent acute pancreatitis related with an anatomical abnormality in the form of a duplicate duodenal ampulla with multiple stones.[2]

Even though several researches were able to detect microlithiasis in less than 10% of all patients with recurrent acute pancreatitis, most of them found microlithiasis in approximately two thirds of the patients.[3]

### Laboratory Assessment and Imaging

Increased serum amylase is often found during laboratory assessment. An increased serum amylase of 3x above normal (>330 u/l) could diagnose acute pancreatitis, but a normal serum amylase does not necessarily rule out the diagnosis of pancreatitis, since serum amylase levels are said to return to normal within 5 days (24-48 hours according to another literature). Assessment of urinary amylase levels may be useful in such cases, since amylase could survive for a long period of time in urine.

Assessments for amylase and lipase isoenzyme activity are more specific and sensitive, since lipase remains in serum for 8 to 10 days. Another assessment that can be performed is peritoneal fluid aspiration, routine blood examination, and blood biochemistry (leukocytosis, hyperglycemia, hypocalcemia, and increased CRP is usually found). Imaging modalities that can be used include ultrasound examination, CT scan, or MRI.[9,10,11]

### Treatment

Most patients (approximately 85-95%) with acute pancreatitis recover spontaneously, usually after 3-7 days of treatment. Treatment for mild acute pancreatitis is generally conservative, in the form of fluid replacement, administration of analgesics, parentheral nutrition, and resting the pancreatic gland. Insertion of nasogastric tubes has little proven benefit, but may be performed to prevent aspiration and decompression in cases of paralytic illeus in severe conditions.

Severe acute pancreatitis may require ICU admission, with the indications of respiratory failure, renal failure, shock, and sepsis.

Administration of anticholinegic agents, H2 antagonists, PPI, glucagons, as well as anti-inflammatory agents do not change the course of disease, while the administration of antibiotics is still argumentative, but this study found that it could alleviate complaints and accelerate healing in patients with severe acute pancreatitis.[8,9,10]

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## Microlithiasis

Microlithiasis refers to bile stones less than 3 mm in diameter, often associated with bile sludge, bile sand, bile sediments, microcrystal disease, false bile stones, or reversible bile stones. The risk factors for microlithiasis include prolonged fasting, total parentheral nutrition, accelerated weight loss, pregnancy, chronic disease, ICU patients, spinal injury, surgery, bone marrow or solid organ transplant, and treatment with Ceftrixone, Cyclosporine, or Ocreotide.[3,8]

Microlithiasis is usually diagnosed using transabdominal ultrasound or ultrasound endoscopy as well as microscopic assessment of bile fluids.

Treatment measures include surgical intervention, endoscopic sphincterotomy, and the administration of chemical solvents.[12,13,14,15]

For patients with symptomatic bile stones, gall bladder removal is considered greatly necessary, since it produces a 100% cure rate without recurrence, and prevents cancer of the gall bladder.[14]

## CASE ILLUSTRATION

Mr. N, 45 years, came with a chief complaint of epigastric pain from 1 year prior to hospitalization, with jabbing pains that spread to the left, accompanied with nausea and greenish vomiting and no fever. The patient had difficulty breathing but had no cough or palpitations. The patient also complained of pain in his waste and tea-colored urine with no sand, or stone in the urine, and no pain when urinating. Defecation was normal. The patient had sought medical advice to a doctor with no improvement.

The patients had been admitted to Cipto Mangunkusumo General Hospital twice with the same complaint. He was first admitted for 1 week (30th of January to 5th of February 2002) with a diagnosis of obstructive jaundice with laboratory results revealing elevated bilirubin levels, particularly direct bilirubin, slightly elevated SGOT and SGPT, bilirubinuria, as well as normal amylase and lipase levels. The patient was treated with Systenol as needed, and 3 x 1 capsule of curliv, 2 x 1 vial of ranitidine, and 3 x 1 vial of primperan. The patient was referred to the digestive surgery ward for cholesystectomy based on an ultrasound result revealing multiple minicholesystolithiasis and left kidney cyst, but the procedure was cancelled due to a lack of funds.

The patient was hospitalized for 11 days on his second admission (June 3rd to 14th, 2002) with a diagnosis of acute pancreatitis, the same complaints, and laboratory findings revealing leukocytosis, slightly elevated SGOT and SGPT, as well as greatly increased amylase and lipase levels. The patient was treated with 3 x 1 g of cefotaxim, 2 x 1 vial of ranitidine, and total parentheral nutrition in the form of 1 bottle of triofusin E 1000 and aminoleban for every 12 hours.

The patient had no history of alcoholism.

Physical examination revealed vital signs as follows: blood pressure 110/70 mmHg, pulse rate 84x/minute, respiratory rate 20x/minute, and body temperature 37 ° C. The patient's conjunctiva were not pale. His sclera demonstrated no jaundice. His heart and lungs were within normal limits. His abdomen not distended, there was no tenderness, and his liver and spleen were not palpable. His acral extremities were warm.

Laboratory assessment revealed leukocytosis, increased bilirubin levels, particularly direct bilirubin, slightly elevated SGOT and SGPT levels, elevated alkaline phosphatase levels, and increased amylase and lipase levels. Based on the following items:

1. Jabbing epigastric pain shooting to the left,
2. Two previous similar episodes,
3. Elevated amylase and lipase levels as well as a more elevated direct bilirubin level compared to indirect bilirubin,
4. The first abdominal ultrasound examination revealing multiple mini-cholesystolithiasis and left kidney cyst and the second one presenting no sign of acute pancreatitis and left kidney cyst.

The problem was first formulated as follows:

1. Recurrent acute pancreatitis due to bile duct stone
2. Obstructive jaundice due to bile duct stone
3. Bile stones
4. Cyst in the left kidney.

The patient was scheduled to undergo a repeat ultrasound examination and ERCP. The patient was to be treated with 3 x 1 g of Cefotaxim, 2 x 1 vial of primperan, 2 x 1 vial of ranitidine, and a nasogastric tube was to be inserted. If the production obtained from the nasogastric tube was less than 100 cc, the patient was to receive a liver diet I. If not, the patient was to receive total parentheral nutrition with Triofusin E 1000: Aminovel 600 = 1:1 for every 12 hours from the first infusion line, and 500 cc of 0.09% NaCl every 8 hours from the second line.

On the sixth day of treatment the patient had hyponatremia and hypocalcemia, suspected to be due to vomiting. NaCl infusion was continued, with the addition of 1 x 1 of KCR. On the seventh day of treatment the patient's condition improved. He was no longer

vomiting, and had reduced signs of jaundice in his sclera. On the 15th of August 2002, a repeat abdominal ultrasound was performed, revealing multiple microlithiasis (consultation diagnosed the patient with recurrent acute pancreatitis due to multiple microlithiasis) that had loosened and obstructed the choleductal and pancreatic ducts. On the next day, repeat evaluation of amylase and lipase levels revealed lowered levels. The patient was then consulted to the department of digestive surgery for cholesystectomy scheduled on September 2002 since the patient had no money. The patient was advised to visit the gastroenterology, hepatology, and digestive surgery out-patient clinic and was educated on the things that could initiate recurrent acute pancreatitis. The patient received 2 x 250 mg of CDCA and 2 x 250 mg of UDCA.

The patient was readmitted to the department of digestive surgery on October 3rd 2002, and underwent open cholesystectomy on the ninth of October 2002. On the 14th of that month, the patient was announced healthy and was released.

**Table 1. First Admission**

| DPL | | | Renal function | | | Urinalysis | |
|---|---|---|---|---|---|---|---|
| *Date* | *29/1* | *30/1* | *Date* | *29/1* | *30/1* | *Date* | *29/1* |
| *HB* | *14.9* | *14.9* | *Ur* | *35* | *19* | *BJ* | *10 25* |
| *Ht* | *42.1* | | *Cr* | *1.2* | *0.7* | *Ph* | *4* |
| *RBC* | *5.6 million* | | | | | *Protein* | *-* |
| *WBC* | *6,800* | *7,300* | *Electrolyte* | | | *Glucose* | *-* |
| *Platelet count* | *165,000* | *185,000* | *Date* | *29/1* | | *Acetone* | *-* |
| *Diff* | | | *Na* | *144* | | *Hb* | *+* |
| | | | *C* | *3.8* | | *Bilirubin* | *3+* |
| *Clto blood sugar* | | *117* | *Cl* | *104* | | *Urobilin* | *0.2* |
| | | | | | | *Bacteria* | *-* |
| *Liver function* | | | | | | *Epithel* | *+* |
| *Date* | | | | *5/2* | | *WBC* | *3-4* |
| *Albumin* | *4.1* | | *Amylase* | *65* | | *RBC* | *2-3* |
| *Globulin* | *2.0* | | *Lipase* | *133* | | *Cylinder* | *-* |
| *Indirect bilirubin* | *4.9* | | | | | *Crystal* | *-* |
| *Direct bilirubin* | *8.5* | | | | | *Yeast* | *-* |
| *Total bilirubin* | *13.4* | | | | | | |
| *SGOT* | *273* | | | | | | |
| *SGPT* | *595* | | | | | | |
| *Alkaline phosphatase* | | | | | | | |
| *CHE* | *5.23* | | | | | | |
| *GGT* | | | | | | | |

30-January-2002
HbS-Ag (-), Anti-HAV (-), anti-HCV (-), AFP 6.1
ECG: sinus rhythm, heart rate 80x/minute, normo-axis, ST – T change (-), LVH (-), RVH (-). Chest X-Ray: within normal limits 7-February-2002 Ultrasonogram:
1. Multiple mini-cholecystolitiasis with signs of chronic cholecystitis.
2. Simple renal cyst in the middle of the left kidney pool.

## DISCUSSION

Based on the patient's history of illness, during the patient's first admission, he was diagnosed with obstructive jaundice, since the patient's direct bilirubin level was higher than his indirect bilirubin level, and his SGOT and SGPT levels were not too hing (laboratory results demonstrated a total bilirubin level of 8.5, indirect bilirubin of 4.9, and total bilirubin of 13.4 as well as SGOT and SGPT levels of 273 and 595 respectively). Unfortunately, alkaline phosphatase was not evaluated in this patient. Literature states that an increase in alkaline phosphatase of over 4 times normal indicates obstructive jaundice.[11]

In this patient, amylase and lipase were also evaluated, with values 65 and 133 respectively. Nevertheless, such findings do not eliminate the diagnosis of acute pancreatitis, since the examination was performed 6 days after treatment, while amylase and lipase levels are expected to return to normal within 3 to 5 days after the onset of symptoms of acute pancreatitis. Urinary amylase assessment is thus recommended.

Ultrasound examination demonstrated minicholesystolithiasis, where small bile stones could

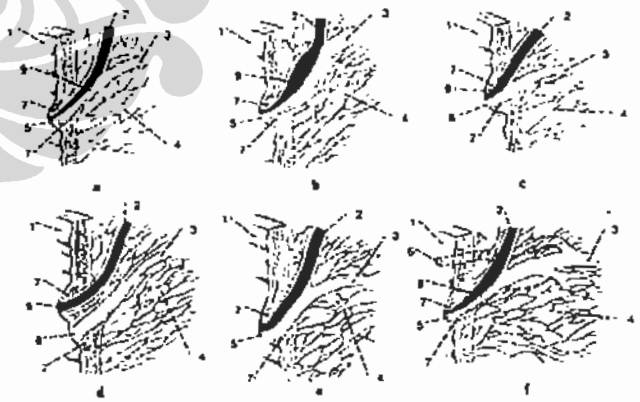

Figure 1. Minicholesystolithiasis

1. Duodenum.
2. Bile duct
3. Pancreatic duct
4. Pancreas.
5. Greater duodenal pappila
6. Minor duodenal pappila
7. Sphincter Oddi.
8. Accessory pancreatic duct
9. Hepatopancreatic ampulla

have descended into and obstructed the collecting duct, or descended into the duodenum once it could pass through.

Based on the findings above, if obstructive jaundice was found without acute pancreatitis, there are two possibilities. The first would be a bile stone obstruction proximal to the collecting duct. The second possibility is an obstruction distal to the collecting duct, consistent with certain anatomical structure where the collecting and pancreatic ducts do not unite, so that obstructions distal to the collecting ducts does not compress or obstruct the pancreatic duct, thus hindering acute pancreatitis in this patient.[1]

During the second admission, the patient was diagnosed with acute pancreatitis, with an amylase level of 1835 and a lipase level of 2136, as well as SGOT and SGPT levels of 298 and 281. It was concluded that the patient suffered from a concurrent acute pancreatitis and obstructive jaundice due to stone obstruction at the distal collecting duct, thus also obstructing the pancreatic duct, seen from a simultaneous increase of amylase, lipase, SGOT, and SGPT levels. The presumed anatomical structure in this patient is thus a uniting distal collecting duct and pancreatic duct. Unfortunately, bilirubin and alkaline phosphatase were not assessed to support obstructive jaundice.[1]

In the case of recurrent acute pancreatitis and obstructive jaundice, there is a great possibility of an obstructing gall stone, consistent with literature, which states that gall stone is the chief cause of acute pancreatitis. In the United Kingdome, for example, in 42% of males and 62% of females, acute pancreatitis is caused by bile stone, while in Bristol, 50% of the first episode of acute pancreatitis and 51% of recurrent acute pancreatitis is caused by bile stones.15 Another literature mentions two thirds of recurrent acute pancreatitis of unknown origin to be caused by microlithiasis. The frequency of microlithiasis in patients with recurrent acute pancreatitis could be found in Table 3.[3,8]

**Table 2. Second Admission**

| *Complete Peripheral Blood Analysis* | | | | *Renal function* | | | *Urinalysis* | |
|---|---|---|---|---|---|---|---|---|
| *Date* | *3/6* | *12/6* | *21/6* | *Date* | *3/6* | | *Date* | *3/6* |
| *HB* | *14.4* | *13.5* | *11.9* | *Ur* | *27* | | *BJ* | *1010* |
| *Ht* | | | | *Cr* | *1.1* | | *Ph* | *6* |
| *RBC* | | | | | | | *Protein* | *Trace* |
| *WBC* | *22,000* | *8,300* | *22,000* | *Electrolyte* | | | *Glucose* | *-* |
| *Platelet count* | *229,000* | *134,000* | *209,000* | *Date* | *3/6* | | *Acetone* | *-* |
| *Diff* | | | | *Na* | *141* | | *Hb* | *-* |
| | | | | *C* | *4.3* | | *Bil* | *-* |
| *Cito blood sugar* | | | | *Cl* | *109* | | *Urobilin* | *0.1* |
| | | | | | | | *Bacteria* | *-* |
| *Liver function* | | | | | *3/6* | *6/6* | *Epithelial cell* | *+* |
| *Date* | | | | *Amylse* | *1835* | *128* | *WBC* | *0-1* |
| *Albumin* | | | | *Lipase* | *2.136* | *206* | *RBC* | *0-1* |
| *Globulin* | | | | | | | *Cylinder* | *-* |
| *Bil I* | | | | | | | *Crystal* | *-* |
| *Bil D* | | | | | | | *Yeast* | *-* |
| *Bil T* | | | | | | | | |
| *SGOT* | *298* | | | | | | | |
| *SGPT* | *281* | | | | | | | |
| *Alkaline phosphatase* | | | | | | | | |
| *CHE* | *4.07* | | | | | | | |
| *GGT* | | | | | | | | |

*ECG: sinus rhythm, heart rate 80x/minute, normo-axis, ST – T change (-), LVH (-), RVH (-).*
*Chest X-Ray: within normal limits*

*Consultation from the subdivision of digestive surgery: no signs of obstructive illeus*

*13/6 – 2002 Ultrasonogram:*
*1. No signs of acute pancreatitis.*
*2. Findings consistent with solitary cyst in the left middle kidney pool*

**Table 3. The Frequency of Microlithiasis**

| | Frequency | Percentage |
|---|---|---|
| *Studies with a low frequency of findings* | | |
| *Venu 1989* | *8/116* | *7* |
| *Nash 1996* | *5/88* | *6* |
| | *13/204* | *6* |
| *Studies with a high frequency of findings* | | |
| *Ros 1991* | *37/51* | *73* |
| *Lee 1992* | *21/29* | *72* |
| *Sherman 1993* | *7/13* | *54* |
| *Caw 1996* | *15/25* | *60* |
| | *80/118* | *68* |

In this patient, microlithiasis was found in his first and third ultrasound examination, but not the second. Ultrasonography is the standard examination tool to detect bile stone, with a sensitivity rate up to 95% for large bile stones. For small stones (microlithiasis), however, the sensitivity is only 50 to 60%, according to the examiner, type of stone (cholesterol stones are more difficult to identify, and usually bile sludge and microlithiasis are intermittent in nature).[4,5] Thus, for more definite assessment, bile microscopy should be performed, with a sensitivity of 60-90%.[3,4,5,8,12,13]

During the third admission, the patient was diagnosed with recurrent acute pancreatitis based on an anatomy as mentioned above, caused by microlithiasis as identified from the ultrasound examination.

Based on the information above, it could be concluded that the patient suffered from recurrent acute pancreatitis and obstructive jaundice due to transient microlithiasis obstruction. The bile stone is believed to descend into the duodenum, consistent with references which mentions that 80 to 95% of acute pancreatitis achieves spontaneous recovery with conservative treatement.[9,10,11]

In this patient, a cholesystectomy should have been performed, but due to financial reasons, it was not performed, thus allowing recurrent episodes to occur.

References state that cholesystectomy should be performed in cases of obstructive jaundice or acute pancreatitis due to symptomatic stone, noting that the stone usually originates from the gall bladder. Other references state that the recovery rate after cholesystectomy is 100%, with a 0% recurrent rate and a very small rate of complication aside from that in elderly patients.

**Table 4. Third Admission**

| Complete Peripheral Blood Analysis | | Renal function | | | Urinalysis | |
|---|---|---|---|---|---|---|
| *Date* | *26/7* | *Date* | *26/7* | | *Date* | *26/7* |
| *HB* | *12.5* | *Ur* | *30* | | *BJ* | *1015* |
| *Ht* | | *Cr* | *0.9* | | *Ph* | *8* |
| *RBC* | | | | | *Protein* | *1+* |
| *WBC* | *12.400* | *Electrolyte* | | | *Glucose* | *-* |
| *Platelet count* | *152.000* | *Date* | *26/7* | *31/7* | *Acetone* | *Trace* |
| *Diff* | | *Na* | *138* | *123* | *Hb* | *-* |
| | | *C* | *3.7* | *3.2* | *Bilirubin* | *Trace* |
| *Clo blood sugar* | *91* | *Cl* | *107* | | *Urobilin* | *1* |
| | | | | | *Bacteria* | *0-2* |
| *Liver function* | | | | | *Epithelial cell* | *+* |
| *Date* | *30/7* | | *26/7* | *16/8* | *WBC* | *0-2* |
| *Albumin* | *3.2* | *Amylase* | *359* | *111* | *RBC* | *4-5* |
| *Globulin* | *4.1* | *Lipase* | *417* | *197* | *Cylinder* | *-* |
| *BIL I* | *2.0* | | *30/7* | | *Crystal* | *-* |
| *BIL D* | *2.7* | *Triglyceride* | *149* | | *Yeast* | *-* |
| *BIL T* | *4.7* | *Cholesterol* | *90* | | | |
| *SGOT* | *58* | *HDL* | *37* | | | |
| *SGPT* | *82* | *LDL* | *23* | | | |
| *Alkaline phosphatase* | *155* | | *30/7* | | | |
| *CHE* | | *Ca* | *6.7* | | | |
| *GGT* | | | | | | |

*ECG: sinus rhythm, heart rate 80x/minute, normo-axis, ST – T change (-), LVH (-) RVH (-). Chest X-Ray: within normal limits*
*15/8-2002 Ultrasonogram:*
*Multiple gallbladder stones.*

Roselyn et al collected data from 42,000 patients undergoing open cholesystectomy, representing 8% of all cases of cholesystectomy performed in the United States within the last 12 months, demonstrating a 0.17% mortality rate, but only 0.03% among patients over 65 years of age. Elderly patients had a high mortality rate of 0.5%. Bile tract damage occurred in approximately 0.2% of all patients.[3,14,15]

Another reference that compared open cholesystectomy, laparoscopic cholesystectomy, and medical (drug) treatment for bile stone could be found in Table 5.[8]

**Table 5. The Comparation Between Open Cholesystectomy, Laparoscopic Cholesystectomy, and Medical (Drug) Treatment**

| | Open Cholecystectomy | Laparoscopic Cholecystectomy | Chemical Solvent |
|---|---|---|---|
| *Cure rate* | *100 %* | *100 %* | *40-95 %* |
| *Recurrence rate* | *0 %* | *0 %* | *50 %* |
| *Mortality rate* | *0-1 %* | *0-1 %* | *0 %* |
| *Bile duct trauma* | *0.2 %* | *0.3 %* | *0 %* |

Endoscopic sphincterotomy is an alternative treatment for the elderly or those who are unable to undergo cholesystectomy. This procedure could improve the motility and injection fraction of the gallbladder.[3,5,9,10,11]

The patient was scheduled to undergo open cholesystectomy, particularly because inadequate finances causes the patient to postpone the procedure until the free open cholesystectomy offer from the digestive surgery division in September 2002.

The problem that came up due to the delay is whether this influences the patient's prognosis. The retrospective study by Runkel NS, Buhr HJ, and Herfart C, from the Department of Surgery of the University of Heidelbergh, Germany on the correlation between the time of surgery from the incidence of pancreatitis among 81 patients that underwent cholesystectomy, (rapid, within 3 days; slow, within 3-14 days, and elective, in over 14 days) demonstrated that a longer time lapse up to the operation produced a less satisfactory prognosis, but postponing the operation during an acute episode reduces the complication of the operation.

The patient received CDCA and UDCA, consistent with literature that states that this combination is able to prevent further stone formation.

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