Case Report

Acute Biliary Pancreatitis: A Case Report

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Acute biliary pancreatitis (ABP) a condition caused by gallstones; can be a life-threatening condition if not treated early (mortality ~2-7%). Complications and symptoms of ABP can be ranging from mild (nausea, vomiting, and fever) to severe (necrosis, infections, hemorrhage, abscesses, renal failure, and adult respiratory distress syndrome). This paper presents a specific case of a middle-aged male diagnosed with ABP to illustrate the disease in a clinical setting, mainly looking at its presentation, diagnosis and focusing more on treatment and management. Clinical examinations and radiological investigations are crucial to recognize the diagnosis and foresee the prognosis of this condition. The medications that are administered to patients suffering from this condition include analgesics (like morphine), intravenous fluids, and antibiotics (e.g. ciprofloxacin). The limitations and lack of knowledge described above are immense concerns. It is highly encouraged that future research opportunities will compensate for the present gap in knowledge, contributing to current literature, as well as having practical implications for treatment and management of ABP.

Keywords: Acute biliary pancreatitis, Case report, Endoscopic retrograde cholangiopancreatography, Laparoscopic cholectectomy, Endoscopic sphincterotomy

INTRODUCTION

Cholecystitis is an inflammation of the gallbladder that occurs most commonly because of an obstruction of the cystic duct from cholelithiasis (stones in the gallbladder).[1] These gallstones may migrate out into the common bile duct and cause obstruction of the pancreatic duct. The blockage of ductal flow increases pressure leading to interstitial fluid accumulation, which is enzyme rich and causes local destruction. Hence, an inflammatory process will result, and acute pancreatitis (AP) is formed.[2] Gallstones account for 40% of all cases of pancreatitis and 90% of all cases of non-alcoholic pancreatitis. Pancreatitis arises in 3-8% of all patients with symptomatic gallstones, and 30% of patients with stones <3 mm in diameter.[3]

Although majority of the patients who suffer from this condition recover without significant sequelae, 15-30% may develop severe episodes requiring multidisciplinary care to ensure the best outcome. Complications of acute biliary pancreatitis (ABP), both local (necrosis, abscesses, hemorrhage) and systemic (pleural effusion, renal insufficiency, multi-organ failure) often require intensive care unit (ICU) management.[4]

The risk factors for developing gallstones include: Increasing age, obesity, and rapid weight loss (as is the case with this patient), being female, pregnancy, the presence of metabolic diseases such as ileal disease (Crohn's disease), diabetes mellitus, alcoholic cirrhosis, and primary biliary disease.[5]

If not treated, the overall mortality ranges from 2% to 7% despite aggressive intervention. The outcome of AP is determined by two factors: Organ failure and second is pancreatic necrosis. About half of the deaths in patients with AP occur within the first 1-2 weeks and are mainly due to multiple organ dysfunction syndromes. When not treated, the risk of recurrence in gallstone pancreatitis ranges from 32% to 61%.[4]

CASE REPORT

Mr. H.H a 32-year-old Pakistani male (date of birth is January 1, 1981) presented to Al-Shifa Al-Jazeera Medical Center on February 9, 2013 with history of abdominal pain. Abdominal ultrasonography was conducted and it showed that the gall bladder (GB) was distended with 3 calculi of 3 mm, 4 mm, and 7 mm. The wall of the GB was normal and the common bile duct was not dilated. Other abdominal organs investigated were unremarkable, including the liver, pancreas, kidneys, spleen, urinary bladder, prostate, right iliac fossa, aorta, and para-aortic areas. There were some abnormalities in the blood test results these are displayed in Table 1 (which can be found in Appendix 1).

Based on the existing findings, the patient was referred to Salmaniya Medical Complex (SMC), were he was admitted to Accident and Emergency on February 13, 2013. At that time, the patient had a 2/7 history of epigastric and right upper quadrant abdominal pain. The onset was sudden, continuous, radiating to the back, and increasing in intensity. It was worse with eating food and relieved slightly when drinking milk. The pain was also associated with: Nausea,

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vomiting once, dysphagia, weight loss of 3 to 4 kg (from 80 to 78 kg without dieting) within the period of 1 month, diarrhea, clay colored stool, dark urine, and jaundice. His vitals were as follows:

- Temperature: 36.2°C
- Respiratory rate: 12 breaths/min
- Blood pressure: 123/77 mmHg

During the physical examination, the patient was conscious and orientated. There were clear signs of jaundice in the skin and sclera. His abdomen was soft, with tenderness in the epigastric region; he did not display guarding, and Murphy's sign, Cullen's sign, and Grey Turner's sign were all found to be negative. The presence of breath sounds (BS) was noted. The patient's score on the Morse patient fall scale was 15 (no risk for falls).

The patient did not have any medical health problems besides this presenting complaint. He has never been admitted to the hospital before has not been operated upon. He is not known to have any allergies and does not take any prescribed, over the counter, herbal or recreational drugs.

Mr. H.H is employed at a travel agency. He is an ex-smoker, quitting about 6-8 months previously. He does not drink. He is single and does not have any children. Both his parents are alive and well although his father has diabetes mellitus, as well as his sister.

Investigations

On the day of admission, the patient's blood sample was sent for the various tests the abnormal results are seen in Table 2 (The details are tabulated in Appendix 1).

The raised alkaline phosphatase (ALP), alanine transaminase (ALT), and gamma-glutamyltransferase (GGT) are all indicators of liver involvement. ALP is raised in liver damage, and high levels of ALP and GGT indicate obstruction of the bile ducts (elevated GTT excludes other causes of raised ALP such as bone disease).[6] The elevated level of bilirubin is the cause of jaundice and scleral icterus in this patient. Elevated direct bilirubin usually indicates post-hepatic biliary disease,[7] while raised indirect bilirubin indicates hepatic damage.

Increased serum and urine amylase is a sign of AP. The values of amylase must be double the normal value in order to be a significant indicator.[8]

Increased lactate dehydrogenase is also found in pancreatitis.[9] In addition, pancreatitis also causes dehydration, which leads to increased serum globulin as well as an imbalance in electrolytes (Cl^-, HCO^{3-}, Na^+) White blood cell elevation is found in both cholecystitis[10] and pancreatitis.[11]

Stool samples were also examined but were unremarkable, and blood cultures were negative.

The day after admission (February 14, 2013) to Salmaniya Hospital, the patient was sent for an ultrasound, which is seen in Figure 1 (Please see details in Appendix 2). The radiologists confirmed some of the earlier findings contradicted others. They found that there was in fact thickening of the GB wall to 7 mm (normal <4 mm).[12] In addition, there was visible mild to moderate biliary sludge and mild pericholecystic edema. The report also stated that the CBD was dilated to a diameter of 1.8 cm (normal <6 mm)[12] with the suspicion of a 7 mm stone at its most distal end. They also found that the liver appeared normal but contained mildly dilated intrahepatic biliary ducts.

Diagnosis

- ABP
- Acute calculus choleycysitis
- Obstructive jaundice.

Treatment

The American Journal of Gastroenterology[13] states that the most important component of management of pancreatitis is supportive care. This includes recording vitals and arterial blood gases (ABGs) at regular intervals, which remained normal in our patient. Fluid resuscitation was administered and patient was kept non-post-operative (NPO) started on normal saline 500 ml on admission the switched to intravenous (IV) ringer's lactate solution 500 ml every 3 h on his 2nd day. He was also given the antibiotic IV flagyl 100 ml (metronidazole) and an injection of perfalgan 100 ml (paracetamol) for analgesia. In addition, IV omeprazole 20 ml (proton pump inhibitor) was administered to suppress acidity. On the third day of his hospital stay, the patient was also given IV rocephen 20 ml (ceftriaxone) as antibiotic prophylaxis for the endoscopic retrograde cholangiopancreatography (ERCP).

The consultant surgeon arranged for an ERCP for the patient. ERCP was indicated due to the results of the ultrasound and liver function tests, which prove the presence of gallbladder stones. ERCP is considered both a diagnostic and therapeutic procedure. It supported the diagnosis and confirmed the presence of sludge at the opening of the ampulla, as well as dilation of the CBD. The therapeutic procedures that were carried out during ERCP were biliary sphincterotomy (excision of the sphincter). In addition, a balloon was used in removal of the aforementioned sludge and stone fragments in the CBD. An image from this patient's ERCP can be seen in Figure 2 (Please see details in Appendix 2).

DISSCUSSION

The aim of this report is the presentation of specific case of an adult male diagnosed with ABP to illustrate the disease in a clinical setting, mainly looking at its presentation, diagnosis, treatment, and management.

ABP is the inflammation of the pancreas primarily due to biliary sludge and gallstones. There are three main types of gallstones: Cholesterol, black pigment, and brown pigment stones. Cholesterol and black pigment stones are located in the gallbladder, and they are mainly attributed to physical or chemical aggravation to the gallbladder. Brown pigment stones, however are located in the bile ducts and are due to infectious agents.[5]

The pathogenesis of AP has not been not fully understood. The specific mechanism by which the passage of gallstones induces pancreatitis is unknown. Two concepts have been identified as the etiology of AP in general: (a) Reflux of infected bile into the pancreas activating a cascade of proteolytic enzymes, and (b) obstruction of pancreatic duct causing acinar disruption from raised pressure. In fact an, yet unproven, hypothesis advanced by Dr. Frank G. Moody in 1993 states that gallstones initiate pancreatitis through obstruction of the pancreatic duct and that progression to necrosis and severe pancreatitis requires the reflux of bile.[4]

The pathophysiology of AP can be divided into three stages according to the severity of the condition (from mild to severe):[5]

- Stage 1: Pancreatic injury, e.g. edema, inflammation, fat necrosis.
- Stage 2: Local effects, e.g. retroperitoneal edema, extensive fat necrosis, ileus with "third spacing" of fluid and electrolytes.
- Stage 3: Systemic complications, e.g. hypotension, shock, metabolic disturbances, and organ failure.

Symptoms of ABP may include: Abdominal and epigastric pain (classically the pain is described as radiating to the back; it may be sudden and intense, or it may begin as a mild pain that is aggravated by eating and slowly grows worse), nausea and vomiting, diarrhea, gastro-intestinal hemorrhage, fat embolism (the exact mechanism of this phenomenon, whether traumatic in origin or related to the AP, is not clear), tetany (not a frequent finding, but may confuse the clinical picture), shock (not usually one of the early findings of the disease; however, in fatal cases, the patients pre-terminally develop evidence of profound peripheral vascular collapse), temperature (the elevation of the temperature during the early stages of the disease between 99°F (32.2°C) and 101°F (38.3°C) is an expression of the active inflammation or necrotic process in the pancreas), pulse (It usually is decreased in proportion to the temperature elevation. The patients who develop clinical shock have a tachycardia in proportion to the severity of the shock).[14] In this case, the patient was suffering from abdominal epigastric pain that radiated to the back, which was worsened by eating. The associated symptoms were nausea and vomiting.

During the clinical examination, a patient who has ABP may have epigastric tenderness, absent BS, tachycardia, dehydration, and moderate pyrexia. The patient may look unwell. Signs of hemorrhagic pancreatitis may be present such as Cullen sign (bruising around the umbilical region, which associated with hemoperitoneum) and Grey-Turner's sign (bruising in the flanks associated with retroperitoneal hemorrhage).[15] In this case, the patient had epigastric tenderness, positive BS, moderate pyrexia as well as he was looking unwell and exhausted.

There are many severity classification for AP like Scoring Systems: Ranson, Glasgow, acute physiology and chronic health evaluation score (APACHE II), Hong Kong score, New Haven, and C-reactive protein (CRP), hemoconcentration.[5] One of the most common classifications is Ranson's criteria[16] classification system shown in Table 3 (The details are tabulated in Appendix 1). Another important scoring system is the Glasgow Criteria scoring system[16] demonstrated in Table 4. (The details are tabulated in Appendix 1). An alternative scoring system is the APACHE II scoring system:[16] Evidence suggests APACHE II scoring at 24 h is at least as accurate as the Ranson and Glasgow scoring systems.

To assess severity through serum measurements, it seems that greater mortality is when creatinine >117 μmol/L[17] and blood glucose >13.8 mmol/L, which, in our patient, were 69 μmol/L and 6.2 mmol/L, respectively. The risk of necrotizing pancreatitis is greater if hematocrit >44 (42 in patient) and CRP is 150 mg/L (this test was not performed).

A study[18] evaluating the efficacy of laboratory results in predicting pancreatitis included bilirubin, ALP, ALT, and AST in the evaluation. They found that ALT was the most clinically useful and to a lesser extent AST. ALT must be greater than or equal to 150 IU/L (approximately a 3-fold elevation), for the positive predictive value to be 95%. On the other hand, total bilirubin and ALP may be useful in guiding diagnosis they were not predictive of gallstone pancreatitis. This study is relevant to our patient's laboratory results. As described his ALT levels were more than 6 times higher than normal.

As for imaging techniques, admission, a computed tomography (CT) scan[13] is indicated to differentiate between

pancreatitis and peptic ulcer disease (PUD). This should have been performed for the patient because he presented with symptoms similar to PUD: Epigastric pain that was slightly relieved when drinking milk. ^{99}mTc-IDA (^{99}mTc-dimethyl-acetanilide- iminodiacetic acid) hepatobiliary imaging was evaluated for its efficacy in distinguishing acute cholecystitis from AP, but it was not conducted on this patient.[19] A few day after admission, CT can be used to differentiate between interstitial and necrotizing pancreatitis. Table 5 shows comparison of imaging techniques for AP (The details are tabulated in Appendix 1).[4]

Differential diagnosis[16]

- With raised amylase: Diabetic ketoacidosis, perforated duodenal ulcer, or mesenteric ischemia/infarction.
- With similar pain: Small bowel perforation/obstruction, or ruptured/dissecting aortic aneurysm.

Vitals, ABGs, fluid balance, urinary output, and hematocrit are monitored and corrected when necessary to prevent hypoxemia, hypotension, and hypovolemia. Conservative medical therapy includes fluid resuscitation, which should be given to the patient immediately and aggressively to prevent progression to organ failure, pain control medication e.g. morphine, nutritional support such as nasojejunal tubes to reduce septic complications and IV nutrition (total parenteral nutrition), and antibiotics;[5] there is no consensus on the effectiveness of antibiotic prophylaxis. They are indicated in septic- looking patients,[20] but if cultures are negative then it should be given for a maximum of 14 days. It is, however, indicated in necrotizing pancreatitis. It was given to our patient in the absence of necrosis and infection, seemingly a routine choice of therapy for AP at SMC.

If pancreatitis is severe[20] then the patient must be transferred to the ICU, this may not be applicable for the patient in question, but must be considered in the elderly, organ failure, and hypoxemia. In regards to nutrition, patients should be kept NPO for 3-7 days. Once the condition has subsided (cessation of nausea, vomiting, abdominal tenderness, narcotics) then limited oral nutrition can commence.

A study[21] divided patients with biliary pancreatitis into two treatment groups to assess the effectiveness of each, early ERCP with sphincterotomy and conservative therapy (ERCP in 3 weeks if obstructive jaundice or sepsis arose). The results indicated that there is no difference between early ERCP and conservative therapy in the absence of obstructive jaundice. Therefore, since H.H had obstructive jaundice ERCP was effective in his condition. The indications for ERCP are presented in Table 6 (The details are tabulated in Appendix 1).

The risk of developing ERCP complications in this patient is 5-30%. These complications are detailed in Table 7 (The table can be found in Appendix 1).[16]

Definitive treatment of cholecystitis is needed to prevent recurrence, usually cholecystectomy.[13] Since our patient was discharged without a laparoscopic cholecystectomy, examination of the current literature is required to have an understanding of when the optimal time frame is to have that procedure for this patient. One such study[22] recommends that laparoscopic cholecystectomy should not be delayed for more than 4 weeks because of lower readmission rates (6%) compared to patients delayed for more than 4 weeks (25%). The readmissions were due to pancreatitis, cholecystitis, biliary colic, and pseudocyst.

CONCLUSION

In conclusion, this is a case of an adult male who was diagnosed by ABP, a condition by which is mainly caused by presence of stones in the gallbladder. The exact pathogenesis of this condition is still unknown. The symptoms and complications of this condition range from mild to severe. This patient presented with the classical symptoms of ABP such as epigastric tenderness, positive BS, moderate pyrexia as well as he was looking unwell and exhausted. After undergoing through thorough clinical examinations and radiological investigations, the patient was diagnosed by ABP. Early ERCP, and endoscopic sphincterotomy are the management options for this case. Conservative medical therapy, which includes fluid resuscitation, pain control medication e.g. morphine, nutritional support such as nasojejunal tubes, and antibiotics are administered to the patient to alleviate the severity of his disease and to prevent more complications.

There are not enough studies regarding the epidemiology of pancreatitis in Bahrain, including the risk factors and the most common causes for this condition. Therefore, this area is a good opportunity for research. In addition, the use of international severity scoring systems for AP is not prevalent in Bahraini hospitals.

This limitation should be corrected, as it is imperative in guiding treatment and management of pancreatitis.

It is strongly recommended that in the future more research should be conducted in understanding the pathophysiology of AP, as the current literature is lacking in this regard.

A double-blind randomized control trial comparing the effectiveness of using antibiotics in AP is required, including a comprehensive look into the indications of antibiotic

therapy and prophylaxis in acute, interstitial, necrotizing, and chronic pancreatitis.

The limitations and lack of knowledge described above are immense concerns. It is highly encouraged that future research opportunities will compensate for the present gap in knowledge, contributing to current literature, as well as having practical implications for treatment and management of acute biliary pancreatitis.

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APPENDIX 1

Table 1: Patient's abnormal blood chemistry test results in Al-Shifa Al-Jazeera Medical Center

Test	Values	Reference range	Units	High/low
White cell count	8.1	4.5-11	×IOVuL	Normal
Red blood count	5.3	4.2-5.7	×I0^{ft}/uL	Normal
Hemoglobin	133	130-162	g/dL	Normal
Hematocrit	42.9	0.32-0.5	%	Normal
MCV	80.9	76-100	fL	Normal
MCH	25.1	25-32	Pg	Normal
MCHC	31.0	31-35	g/dL	Normal
Platelets	327	140-350	×I0^{J}/uL	Normal
Lymphocytes	21.3	20.5-51.1	% of WBC	Normal
	1.7	3.5-9	×IOVuL	Low
Neutrophils	71.3	45-74	% of WBC	Normal
	5.8	1.8-7	×I0^{J}/uL	Normal
Monocytes	1	0-10	% of WBC	Normal
Eosinophils	3	1-6	% of WBC	Normal
AST	93	14-20	U/L	High
ALT	165	10-40	U/L	High

ALT: Alanine aminotransferase, AST: Aspartate transaminase, MCHC: Mean corpuscular hemoglobin concentration, MCH: Mean cell hemoglobin, MCV: Mean corpuscular volume, WBC: White blood cell

Table 2: Patient's abnormal blood chemistry test results in SMC

Test	Values	Reference range	Units	High/low
White cell count	11.9	3.6-9.6	×10^{9}/L	High
Mean cell volume	81.6	82-97	fl	Low
Red cell size distribution width	14	11.6-13.7	%	High
Polymorphs	91	42.2-75.2	% of WBC	High
	10.83	2-7	×I0^{y}/L	High
Lymphocytes	5	20.5-51.1	% of WBC	Low
	0.55	1-3	×10^{9}/L	Low
Basophils	0.01	0.02-0.1	×10^{9}/L	Low
LDH	247	100-190	pmol/L	High
Globulin	36	15-30	g/L	High
Total bilirubin	126	<18	pmol/L	High
Direct bilirubin	100	<7	pmol/L	High
Indirect bilirubin	26	<12	pmol/L	High
ALP	306	50-136	p/L	High
ALT	333	30-65	JJI/L	High
GGT	445	5-85	p/L	High
Sodium	130	137-148	mmol/L	Low
Potassium	3.7	3.9-5	mmol/L	Low
Chloride	83	100-107	mmol/L	Low
Bicarbonate	23	24-30	mmol/L	Low
Scrum amylase	1,128	23-85	p/L	High
Urine amylase	16,398	<400	pmol/L	High
APTT	24	28.1-42.9	s	Low
APTT ratio	0.8	0.9-1.4	s	Low

SMC: Salmaniya medical complex, LDH: Lactate dehydrogenase, WBC: White blood cell, ALP: Alkaline phosphatase, ALT: Alanine aminotransferase, GGT: Gamma-glutamyltransferase

Table 3: Ranson's criteria

At admission	At 48 h after admission
Age>55	Hct drop>10%
WBC>16×10^{9}/L	Urea increase>1.8 mmol/L
Glucose 10 mmol/L	Ca<2 mmol/L
LDH>350 IU/L	Arterial PO_2<60 mmHg
AST>250 IU/L	Base deficit>4
	Fluid needs>6 L

WBC: White blood cell, LDH: Lactate dehydrogenase, Hct: Hematocrit, AST: Aspartate transaminase

Table 4: Glasgow criteria

Glasgow criteria
PO_2<60 mmHg
Age>55 year
Neutrophils+all WBC 15×10^{9}/L
Calcium<2 mmol/L
Raised urea>16 mmol/L
Enzymes AST>200 U/L, LDH >600 U/L
Albumin<32 g/L
Sugar, glucose>10 mmol/L

LDH: Lactate dehydrogenase, WBC: White blood cell, AST: Aspartate transaminase

Table 5: Comparison of imaging techniques for acute pancreatitis

Imaging technique	Effectiveness
Contrast-enhanced computed tomography	78% sensitivity and 86% specificity for severe acute pancreatitis
Endoscopic ultrasonography	100% sensitivity and 91% specificity for gallstones
Magnetic resonance cholangiopancreatography	81-100% sensitivity for detecting common bile duct stones 98% negative predictive value and 94% positive predictive value for bile duct stones. As accurate as contrast-enhanced computed tomography in predicting severity of pancreatitis and identifying pancreatic necrosis
Magnetic resonance imaging	83% sensitivity and 91% specificity for severe acute pancreatitis
Transabdominal ultrasonography	87-98% sensitivity for the detection of gallstones

Table 6: Indications for ERCP

Before cholecystectomy	In hospital exacerbation
Concomitant cholangitis	After cholecystectomy
Obstructive jaundice	Unsuccessful laparoscopic/open common bile duct exploration
Severe disease	Smoldering disease±sphincter dysfunction/ductal disruption

ERCP: Endoscopic retrograde cholangiopancreatography

Table 7: Major ERCP complications

	Mild	Moderate	Severe
Pancreatitis	Hospital stay 2-3 days	4-10 days	10 days pseudocyst/ intervention
Bleeding	Hgb drop<3 g no transfusion	Transfusion<4 U no angio/ surgery	Transfusion>5 U angio/surgery
Perforation	Possible or very mild	Definite, treated medically	Medical Rx>10 days, intervention

ERCP: Endoscopic retrograde cholangiopancreatography

APPENDIX 2

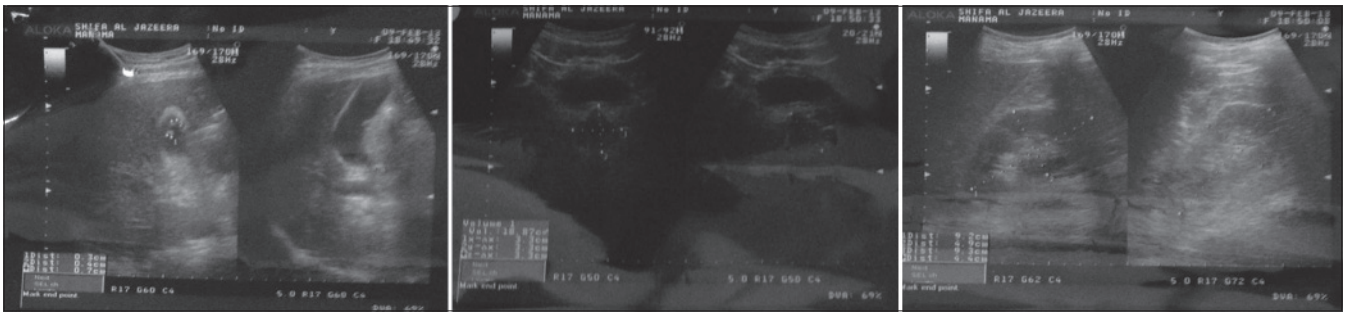

Figure 1: Ultrasound Images

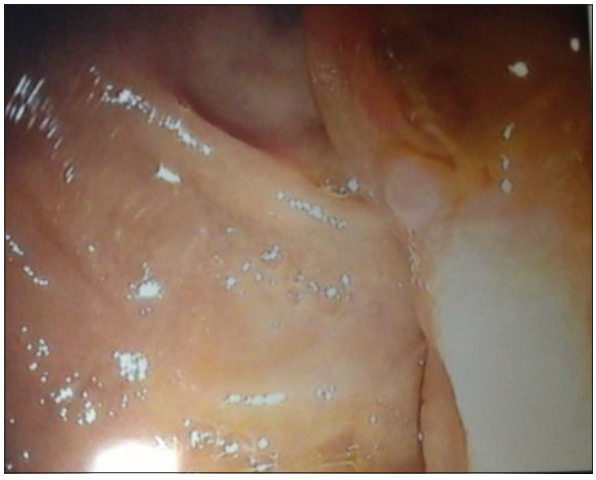

Figure 2: Endoscopic retrograde cholangio pancreatography