Case Report

An Unusual Case of Obstructive Jaundice with an Ambiguous Stone Complicated by Disseminated Intravascular Coagulation and Acute Kidney Injury A Multifaceted Approach to Management

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Abstract

A 40-year-old man presented with obstructive jaundice complicated by DIC and AKI, highlighting the challenges in diagnosis and management. Obstructive jaundice can present with a spectrum of complications, including DIC and AKI, which significantly impact patient management and outcomes. Prompt recognition and intervention are crucial in such cases. ERCP with stent placement is an effective therapeutic modality for relieving biliary obstruction. Close monitoring and multidisciplinary management involving hepatologists, nephrologists, and intensivists are essential for optimizing patient care.

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Introduction

Obstructive jaundice is a clinical syndrome characterized by the accumulation of bilirubin due to impaired bile flow. It can result from various etiologies, including choledocholithiasis, malignancy and strictures. Cholestasis refers to disruptions in bile flow resulting from diseases affecting hepatocytes, intrahepatic bile ducts, or the extrahepatic biliary system.[1] The TG2013/2018 diagnostic criteria for acute cholangitis provide a structured approach to diagnosis. Systemic inflammation is assessed through fever or chills above 38°C and biological markers such as leukocyte count and CRP levels. Cholestasis is identified by jaundice or elevated total bilirubin levels, while abnormal liver function tests indicate hepatic involvement. Imaging showing bile duct dilatation or providing evidence of the underlying cause aids in diagnosis. The microorganisms commonly associated with acute cholangitis.[2] Choledocholithiasis occurs in 1–15% of cases and is found in 5–29% of patients with cholelithiasis. It can result in obstructive jaundice, biliary pancreatitis and potentially life-threatening acute obstructive suppurative cholangitis.[3] Disseminated intravascular coagulation (DIC) is a complex disorder involving systemic activation of coagulation, leading to widespread microvascular thrombosis and consumptive coagulopathy ultimately causing multi-organ dysfunction syndrome.[4] Difficult common bile duct stone is defined based on the characteristics of the stone, accessibility to papilla related to anatomical variations, and other clinical conditions or

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comorbidities of the patients.[5] Obstructive jaundice with DIC and AKI is a rare, complex condition. Post-cholecystectomy, stones can still form in the CBD due to residual or de novo factors. This case highlights the role of ERCP and the need for timely, multidisciplinary management to improve outcomes.

The institutional board of Fatima memorial hospital approved this study and written Informed consent was obtained from the participant.

Case Report

A 40-year-old male married and resident of okarah, businessman by profession presented to emergency room (ER) on 26 February 2024 with a 4-days history of vomiting and yellowish discoloration of the skin and sclera. Additionally, he experienced irritability for the past 1 day. The patient was in his usual state of health (USOH) 4 days back when he started vomiting after eating food from a restaurant. The vomiting was acute in onset, multiple episodes, large in volume containing food particles yellowish in color associated with food intake and right hypochondrial pain. It was relieved by antiemetic and non-bilious, having no blood or coffegrounnd content. He also complained of yellowish discoloration of the eyes and entire body, associated with itching and dark yellowish color of urine. The presence of small reddish/purplish spots (multiple petechial rashes) across his body were also reported. He also experienced generalized body pain and muscle weakness without any joint pain. The patient had a past medical history significant for type 1 diabetes spanning 20 years, managed with apidra (rapid acting insulin --16 units TID) & lantus (long-acting insulin---16 units OD) as well as a surgical history including appendectomy 15 year ago and cholecystectomy, Choledocholithotomy and pancreatico-lithotomy 12 year back. Additionally, he had a prior history of a liver abscess occurring 2 years back (treated with IV antibiotics). He smoked 4 to 5 cigarettes per day (10 packs per year) and admited to use illicit drugs intravenously and consuming ice over the past 6 months. However, he denied any known drug or food allergies. Family history reveals type 2 diabetes and hypertension in both parents. From a socioeconomic standpoint, the patient belonged to the upper class. On general physical examination, middle-aged male, average height and build, thin & lean, lying in bed anxiously, was conscious and oriented in time, place and person. He exhibited marked jaundice of skin. Vital signs revealed a pulse rate of 135 beats per minute, regular and of good volume, with no evidence of radio-radial or radio-femoral delay, blood pressure of 100/70 mmHg, temperature of 98°F, respiratory rate of 26 breaths per minute, oxygen saturation of 94% at 2 liters of oxygen and BSR was undetectable on glucometer. During gastrointestinal examination, inspection of the oral cavity was unremarkable, and the abdomen moved with respiration, exhibiting scar marks from previous cholecystectomy and appendectomy with intact hernial orifices. On Palpation epigastrium and right hypochondrium were mildly tender. Liver was palpable 3-4 finger breaths below right costal margin and spleen was palpable 3-4 finger breaths below left costal margin. Percussion revealed that lower border of liver percussible 3-4 finger breaths below right costal margin and upper border was percussible in right 5th intercostals space with liver span of 18 cm.spleen was percussible 3-4 finger breaths below left costal margin.No fluid thrill or shifting dullness and Bowel sounds were audible @3-5 per minute.Respiratory examination demonstrated respiratory rate 26 breaths/min and bilaterally equal chest wall movement.Auscultation showed normal vesicular breathing with decreased air entry at the bases bilaterally and no added sound.remaining respiratory examination was unremarkable. Cardiovascular examination was also unremarkable except pulse rate was 135/mi. Neurological examination indicated the patient was well-oriented with a Glasgow Coma Scale (GCS) score of 15/15. Motor examination revealed normal bulk with no wasting or atrophy, normal muscle tone, and full power in the upper and lower limbs, with all reflexes present and bilateral plantar reflexes being down going. Signs of meningeal irritation (SOMI) were also negative and there was no neck rigidity. The differential diagnosis considered for the patient's presentation include HIV/AIDS, acute viral hepatitis, diabetic ketoacidosis (DKA), AUTOIMMUNE DISORDER, disseminated intravascular coagulation (DIC) secondary to sepsis due to cholangitis, obstructive jaundice and symptomatic choledocholithiasis. Patient admitted to the High

Dependency Unit (HDU) and all baseline labs conducted along with a workup for Diabetic Ketoacidosis (DKA) and HIV serology, as well as Hepatitis B and C serology sent. Patient was in sepsis and mild cholangitis, didn't have any symptoms of shock. Patient was treated on the line of sepsis and DKA. Nasogastric (NG) tube and Foley catheters had been inserted. Patient was ordered nothing per oral (NPO). Treatment initiated with insulin Regular infusion at a rate of 6 units per hour along with normal saline infusion at 250ml per hour. Antibiotic therapy commenced with Piperacillin-Tazobactam 4.5 grams intravenous twice daily, Metronidazole 500mg intravenous thrice daily, Ondansetron 8mg intravenous thrice daily and Omeprazole 40mg intravenous twice daily. 2 FFPs had also been transfused.NSAID was avoided. Additionally, to minimize bruising from subcutaneous insulin administration, intravenous insulin was administered.

Table 1: *Critical Admission Labs: Marked Electrolyte Imbalance, Hyperglycemia and Liver Dysfunction*

TLC	RBC	HB	PLT	HCT
$10.87x10^3$ per microliter	$4.02x10^6$ per microliter	10.8 g/dl	$6x10^3$ per microliter	31%
Na	**K**	**Cl**	**Bicarb**	**CRP**
116 mmol/L	4.46 mmol/L	80 mmol/L	10.1 mmol/L	137.31 mg/L
Amylase	**Lipase**	**LDH**	**Creatinine**	**Uric acid**
16 U/L	56.3U/L	319	2.87mg/dl	9.2mg/dl
urea	**Ammonia**	**PT**	**APTT**	**INR**
219mg/dl	54.2mg/dl	16.9sec (control:11)	31.3sec (control:26)	1.57
Glucose Random	**ALT**	**AST**	**GGT**	**Albumin**
631 mg/dl	48U/L	31U/L	127U/L	2.5 g/L
Total bilirubin	**Indirect bilirubin**	**Direct bilirubin**	**ALP**	**D-DIMER**
24.31mg/dl	1.27 mg/dl	23.04 mg/dl	417U/L	2.05
PH	**PCO2**	**PO2**		
7.22	22	93		

Table 2: *Viral Screening: No Evidence of Viral Infections*

Anti HAV-IgM	Anti-HIV 1+2	Anti HEV IgM	HbsAg	Anti HCV	PCR HIV
Non-reactive	Non-reactive	Non-reactive	Non-reactive	Non-reactive	NOT DETECTED

The urine analysis indicated dark yellow color and hazy appearance with a specific gravity of 1.02. Glucose levels were significantly high, while ketones were absent. Despite numerous white blood cells, leukocyte esterase was negative. Red blood cell count ranged from 4 to 6, and moderate bacteria were observed. Bilirubin was moderately elevated and protein was detected at a lower level. Other parameters such as urobilinogen, pH, nitrite, yeast, and mucus were within normal ranges or absent. Chest X-ray and HRCT revealed heterogeneous areas of consolidation, particularly in the bilateral basal regions, greater extent to the left side, indicating an inflammatory process. The abdominal ultrasound (USG) revealed enlarged liver, measuring 17.8 cm, with a normal echotexture and no focal lesions identified. The gall bladder was noted to be surgically absent. Mild dilatation of the intrahepatic biliary channels was observed, attributed to ambiguous calculi in the common bile duct (CBD) alongside thick intramural sludge. Additionally, the pancreatic parenchyma was obscured. Bilateral increased echogenicity of the renal parenchyma was noted, indicating potential renal pathology. Furthermore, the urinary bladder contained thick, debris-laden contents. The portal vein Doppler study revealed no evidence of thrombus or obstruction in both the portal and hepatic veins, indicating normal blood flow through these vessels. However, intrahepatic biliary channels were noted to be dilated, and the common bile duct (CBD) measured 24 mm in diameter. Additionally, the CBD contained thick sludge, which was obscured by the presence of calculi within the duct. These findings suggest potential obstruction or impairment of bile flow within the biliary system. He had cholestatic jaundice, uncontrolled blood sugar levels, hyponatremia, hypokalemia, thrombocytopenia, acute kidney injury, pigment induced nephropathy and LRTI (lower respiratory tract infection). So according to the initial workup HIV/AIDS, acute viral hepatitis, diabetic ketoacidosis (DKA) had been excluded. The patient developed bicytopenia (anemia and thrombocytopenia) and disseminated intravascular coagulation due to sepsis (which may be due to cholangitis or some autoimmune cause). 1 mega unit of platelets had been transfused but platelet count

didn't improved.so the multidisciplinary approach was considered for this patient. The hematology team initiated treatment with hematinic and eltrombopag, gastroenterology planning an endoscopic retrograde cholangiopancreatography (ERCP) procedure contingent on platelet counts > 50k, nephrology addressing progressive AKI and serum potassium replacement, and pulmonology consulted for suspected lower respiratory tract infection.MTb gold assay was negative.RBC ANTIBODY screening was negative,corrected reticulocyte count 0.2%,serum iron 70 mg/dl and TIBC 155 mg/dl.one packed cell volume and one megaunit was transfused.Fresh frozen plasma added in BID daily.Hb and platelet count was not improved as patient developed antibodies and no blood was compatible.DAT positive and 3 cross match of PCV were incompatible.Antibiotic therapy was switched to oral moxifloxacin and ecasil, with intravenous fluids reduced to 50ml/h from 100ml/h.methylprednisolone was initiated at 125 mg twice daily.there Hb levels improved.COOMBS direct was weak positive and coomb's indirect was negative.ANA testing was conducted, revealing negative results for ANA/ENA. Further autoantibody-related workup was pursued for various syndromes including SLE, Sjogren's syndrome, SHARP syndrome, systemic sclerosis and CREST syndrome. RBC morphology exhibited anisocytosis, microcytosis, macrocytosis, along with pencil cells and fragmented RBCs, while WBCs were normal in nuclear morphology but showed hypersegmented neutrophilia. Marked thrombocytopenia was noted. Urine culture and sensitivity showed no growth while blood culture and sensitivity showed growth of E. COLI which was sensitive to amikacin, imipenem, meropenem, piperacillin and tazobactam.

Table 3: *Blood Count Improving Trends*

TLC (x103 per microliter)	17.09→15.45
Rbcs (x106 per microlitre)	2.78 →3.17
Hb %	8.7→9.2
PLT (x103 per microlitre)	7→46

All the above workups established the diagnosis of acute cholangitis leading to DIC and sepsis.

ERCP was performed after the platelet count reached 64k, with successful stent replacement to alleviate the biliary obstruction. Normal bile flow was restored. This stent replacement was performed via ERCP due to the presence of an 18mm stone in the common bile duct (CBD), which could not be removed due to structural constraints within the duct. The patient's condition showed significant improvement, allowing for discharge with recommended follow-up.

Table 4: *Discharge Labs Stabilized Blood Counts & Electrolytes*

TLC	Hb	PLT	Na	K	Bicarb	Cl
8.16(x103 per microlitre)	9.50%	150(x103 per microlitre)	130mmol /L	4.6 mmol/L	20.7 mmol/L	99 mmol/L
ALT	**GGT**	**AST**	**ALP**	**BILIRUBIN**	**Albumin**	**Creatinine**
19 (U/L)	83(U/L)	15(U/L)	172(U/L)	6.93 mg/dl	2.7 g/L	1.09 mg/dl

Upon discharge, the prescribed medications include Tablet folic acid, Syrup Ulcisanic, Capsule Urso, Tablet Deltacortil (2 tablets twice daily), Tablet Ossobon D, Tablet Neurobion, Injection Apidra (16 units x TID), Injection Lantus (16 units x OD).

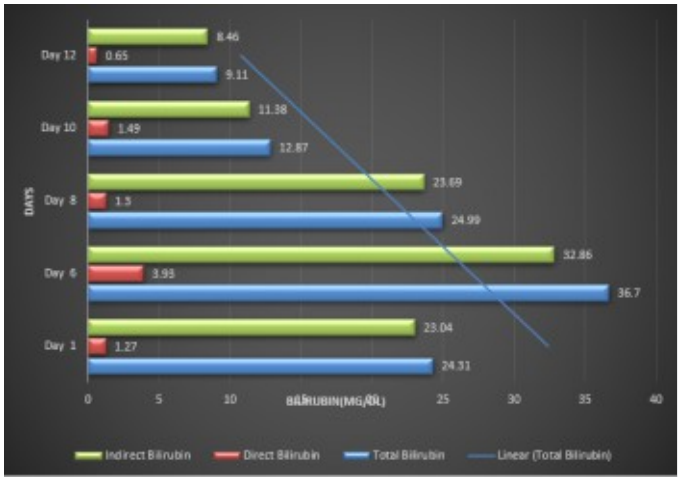

Figure-1: *Trend of bilirubin during hospital stay*

Figure 2: *ERCP----CBD with calculi*

Discussion

Gram-negative bacilli are predominant, with Escherichia coli being the most frequently isolated organism, accounting for 35% to 62% of positive blood cultures and 31% to 44% of biliary cultures.[2] The criteria for difficult bile duct stones, categorized into three main factors: stone characteristics, stone location, anatomical situation, and patient factors. Stone characteristics include large size (>15 mm), multiple stones (>3 stones, size >10 mm), hardness, and irregular shape, which pose challenges for lithotripsy and capture with a basket. Stone location factors involve stones in intrahepatic ducts, above strictures, impacted in the bile or cystic ducts, or associated with Mirizzi syndrome, complicating access. Anatomical situations such as altered anatomy (e.g., Bill Roth II/Roux-en-Y gastric bypass), periampullary diverticulum, or difficult biliary access further contribute to the complexity. Patient factors, including old age, poor general condition, unstable vital signs, bleeding tendency, and paradoxical responses, increase the risk of adverse events during intervention.[5] In obstructive jaundice, absence of gut luminal bile and accumulation of hepatic and circulating bile acids lead to gut bacterial overgrowth, mucosal atrophy, and systemic inflammation, with HMGB1 playing a significant role in promoting these pathological processes and contributing to multiple organ dysfunction.[6] A proposed risk assessment strategy for choledocholithiasis in symptomatic cholelithiasis patients relies on clinical predictors grouped by strength. Very strong predictors include CBD stone visualization, ascending cholangitis symptoms, and bilirubin levels >4 mg/dL. Strong predictors are a dilated CBD (>6 mm) and bilirubin levels of 1.8-4 mg/dL. Moderate predictors encompass abnormal liver tests, age >55, and signs of gallstone pancreatitis. High risk is indicated by any very strong predictor or both strong predictors, low risk by the absence of predictors, and intermediate risk otherwise. This strategy aids clinicians in timely intervention and treatment decisions.[7] Temporary biliary plastic stents post-ERCP prevent stone impaction and cholangitis, with potential for stone size reduction. Long-term stenting is for high-risk patients. Fully covered metal stents may aid incomplete clearance but pose migration risks. EPLBD is recommended initially, followed by cholangioscopy-guided lithotripsy or mechanical lithotripsy if needed, with ESWL as an alternative. Temporary stenting is a last resort, while indefinite stenting is for short life expectancy cases.[8] Current recommendations favor endoscopic papillary large balloon dilation (EPLBD) alone or combined with limited sphincterotomy for these cases. If extraction fails, options include mechanical lithotripsy, cholangioscopy-assisted lithotripsy, or extracorporeal shockwave lithotripsy. Surgery may be considered if lithotripsy is unavailable or unsuccessful.[9] Patients with altered anatomy require specific endoscopic approaches. For Billroth II or Roux-en-Y reconstruction, tailored procedures are necessary. Techniques like enteroscopy-assisted ERCP and EUS-guided approaches have been explored. Innovative methods, such as EUS-directed transgastric ERCP, show promise for Roux-en-Y patients. Post-liver transplantation, effective endoscopic management is crucial but complicated by factors like strictures and biliary casts, requiring careful planning.[10] Acute kidney injury (AKI) is a common complication associated with severe illness and can occur secondary to hemodynamic instability or direct renal injury.[11]The limitations of the study were that the patient's low platelet count poses a challenge for the ERCP procedure, as a minimum of 50,000 platelets is typically necessary for the ERCP. Furthermore, the patient's positive DAT reaction adds complexity to potential blood transfusions.

Conclusion

This case underscores the importance of considering a broad differential diagnosis and implementing comprehensive approach to the management of obstructive jaundice complicated by DIC and AKI. Timely intervention, supportive care, and definitive treatments such as ERCP with stent placement can lead to favorable outcomes in such complex cases.

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Author Contribution

TA: Conception and design, drafting the article or revising it critically for important intellectual content.

MKR: Conception and design, drafting the article or revising it critically for important intellectual content. final approval of the version to be published.

HJS: Drafting the article, final approval of the version to be published.

SA: Analysis & Interpretation, drafting the article.

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