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Case Study

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CHOLANGIOCARCINOMA COMPLICATED WITH SEPSIS AND MULTIPLE ORGAN DYSFUNCTION SYSTEM (MODS) INDUCED CARDIOPULMONARY ARREST: A CASE STUDY

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ABSTRACT

Objectives: To know the desirable outcomes.; to weigh up the situation; and to explain the importance of early diagnosis. **Case Presentation:** We report a case of 70-year old patient diagnosed with Cholangiocarcinoma who developed complications and ended up with Multiple Organ Dysfunction System (MODS). **Conclusion:** Cholangiocarcinoma accounts for 10% of global mortality. Jaundice is the only presenting symptom in most patients. Therefore, patients presenting with jaundice and possessing risk factors for developing CCA should be rigorously screened for the presence of CCA. Serious complications of sepsis include end organ hypo perfusion, severe organ failure and death can be avoided by effective Early Goal Directed Therapies (EGDT).

KEYWORDS: Cholangiocarcinoma, Case report, Multiple Organ Dysfunction Syndrome, Early Goal Directed Therapy (EGDT).

INTRODUCTION

Cholangiocarcinoma (CCA) is a highly lethal adenocarcinoma of the hepatobiliary system, which can be classified as intrahepatic, perihilar and distal. Each anatomic subtype has distinct genetic aberrations, clinical presentations and therapeutic approaches. The CCA prognosis is considered to be dismal. 90% of pCCA patients present with jaundiceand 10% with acute cholangitis. 56% of pCCA patients complain anorexia, weight loss and fatigue (systemic signs of malignancy) as their initial presentation. Hepatobiliary malignancies account for 13% globally. The incidence of CCA are reported highest in Southeast Asia and the lowest in Australia.[1]

World Health Organisation defined sepsis as "life threatening organ dysfunction caused by dysregulated immune response to infection." In 2017, globally there were an estimated 49 million cases of sepsis. It is the leading cause of mortality and morbidity in higher rates despite taking proper medical attention.[2]

Multiple Organ Dysfunction Syndrome (MODS) relates to the critical illness characterized by reversible physiological abnormalities with the dysfunction of two or more organs that occur simultaneously. It requires extensive clinical management and huge healthcare resources. It may lead to prolong stays in the intensive care unit and/or may result in mortality (27%-100%). Therefore, it is important to identify the possible preventable predisposing factors of MODS in high-risk patients.[3]

Acute kidney injury (AKI) is defined as the sudden loss of kidney function. It is determined based on increased serum creatinine levels, oliguria and is usually limited to a duration of 7days.[4] The symptoms include changes in urinary habits (decreased urine output or urine discoloration), severe abdominal or flank pain, sudden weight gain.[5]

Acute pancreatitis is the inflammation of pancreas with variable involvement of regional tissues or remote organ system. It is characterized by elevation of pancreatic enzymes in the blood and severe pain in the upper abdomen.[5]

CASE PRESENTATION

A 70-year old male patient presented with complaints of jaundice, drowsiness since 1 week; loose motions, 3-4 episodes since 1 day; low urine output since 1 day. He was a known case of Diabetes Mellitus(DM), Hypertension(HTN), Coronary Artery Disease(CAD)-Post Percutaneous Transluminal Coronary Angioplasty

(PTCA)+Stent to Left Anterior Descending Artery (LAD), and on regular medications.

On Examination, the patient was conscious, coherent, body temperature was normal, blood pressure was 100/60 mmHg, pulse rate was 86 bpm, respiratory rate was 20/minute, S_1 and S_2 sounds heard; abdomen was palpated soft, SpO2 was 94% on 2 liters of oxygen.

Laboratory Reports

Day-1: Ultrasound of Abdomen showed hepatomegaly with coarse echotexture, mild bilobar Intra Hepatic Biliary Duct (IHBD), cystitis, minimal bilateral pleural effusion and mild ascites, aneurysmal dilatation of short segment of the abdominal aorta.

Magnetic Resonance Cholangiopancreatography (MRCP) showed Biliary Stricture involving the Common Bile Duct (CBD), Wall thickness: 3*3*4.6 cm, suspective of cholangiocarcinoma.

Figure-1: MRCP imaging of liver, gallbladder, bile ducts, pancreas and pancreatic duct.

Endoscopic Retrograde Cholangio-Pancreatography (ERCP) report was suspective of Hilar cholangiocarcinoma, Biliary sphincterotomy, right ductal stenting was done and stricture cytology was sent.

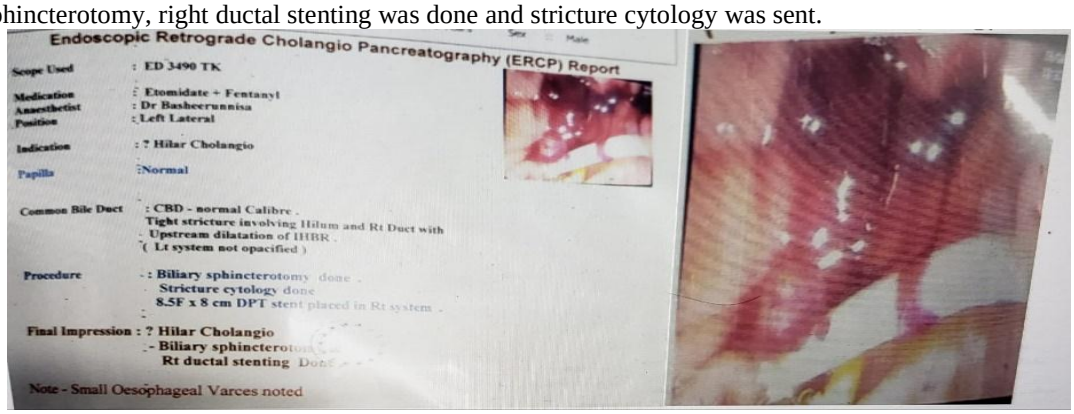

Figure-2: ERCP to evaluate cholangiocarcinoma.

CYTOLOGY INVESTIGATION: After ERCP was done, biliary brush cytology was sent to the lab to rule out the suspicion of CCA. Result showed the features of malignancy and carcinoma.

Day-3: Ultrasound of Abdomen reports shows Hilar cholangiocarcinoma, Status Post Percutaneous Transhepatic Biliary Drainage (PTBD) in bilateral ductal systems, Mild IHBD in the right lobe, moderate ascites, and minimal right pleural effusion.

Table 1: Day wise laboratory investigations' reports of patient along with the normal ranges of tests.

	Day-1	Day-2	Day-3	Normal Range
Total Bilirubin	**23.2**	**26.3**	-	0.2-1.3 mg/dL
Direct Bilirubin	**18.8**	**24.5**	-	Upto 0.25 mg/dL
Indirect Bilirubin	**4.4**	**1.8**	-	0.2-0.8 mg/dL
SGPT	**92**	**95**	-	13-69 U/L
SGOT	**104**	**437**	-	15-46 U/L
ALP	**134**	**400**	-	36-126 U/L
Prothrombin time	-	-	**16**	10-14 sec
Hb	**9.5**	**10.7**	**10.1**	12.5-17.5 Gms%
RBC	**3.0**	**3.0**	**3.33**	4.5-5.5 Milli/Cumm

WBC	**12,800**	**11,600**	**14,400**	4000-11000/Cumm
Platelets	1.75	1.90	2.1	1.5-4.5 Lakhs/Cumm
Creatinine	**3.6**	**4.3**	**4.5**	0.66-1.25 mg/dL
Urea	**54**	**65**	**67**	13-43 mg/dl
Sodium	**119**	**132**	140	137-145 mmol/L
Potassium	4.8	4.5	4.0	3.5-5.1 mmol/L
Chloride	**95**	102	102	98-107 mmol/L
Lipase	**-**	**120**	**140**	13-60 U/L
Amylase	**-**	**109**	**125**	1-100 U/L
ESR	**55**	**80**	**87**	0-20mm/hr
Procalcitonin	**-**	**0.9**	**1.4**	>0.5ng/ml

*The values marked in **bold** indicate abnormal

Diagnosis and Treatment

Based on the laboratory investigations' reports, the diagnosis was **Hilar Cholangiocarcinoma.**

In order to bring down elevated bilirubin, Ultrasound guided Percutaneous Trans hepatic Biliary Drainage (PTBD) was placed. The patient developed hypotension, for which he was shifted to Intensive Care Unit and administered inotropic supports (noradrenaline and vasopressin)

During his hospital stay, his creatinine levels raised above normal range suggesting **Acute Kidney Injury.** He had an increased procalcitonin levels, elevated ESR, increased white blood corpuscles (WBC) count indicating **Sepsis.** He also had increased levels of amylase and lipase indicating **Acute Pancreatitis.**

Despite the management with meropenem, piperacillin-tazobactam, sodium bicarbonate, ursodeoxycholic acid, rifaximin, sorbitol, tricholine citrate syrup, cholestyramine, amikacin, furosemide, atorvastatin, enoxaparin, vasopressin, etc. there was still an increase in serum bilirubin, serum creatinine, and WBC, thereby indicating a very poor prognosis. On day-3, patient developed metabolic acidosis with oliguria for which hemodialysis was advised (but was denied by the patient's kin).

On that night, patient developed cardiopulmonary arrest. Cardiopulmonary Resuscitation was initiated according to Advanced Cardiovascular Life Support (ACLS) protocol, but he could not be revived and was declared dead.

The cause of death was stated as cardiopulmonary arrest due to MODS (sepsis-induced), CCA, comorbidities like DM, HTN, CAD.

CONCLUSION

Cholangiocarcinoma accounts for 10% of global mortality. There is no disease specific symptom for CCA. Jaundice is the only presenting symptom in most patients. Therefore, patients presenting with jaundice and possessing risk factors (older age, family history, bowel diseases, social history) for developing CCA should be rigorously screened for the presence of CCA. An early diagnosis at benign stages and initiation of appropriate anti-neoplastic treatment helps the patient recover with minimal therapy; prevents the advancing of cancer and related complications.

A patient suffering with sepsis, is at a higher risk of ending up in septic shock which may lead to systemic hypotension, organ system insults and ischemia thus creating life-threatening complications. A timely detection of sepsis presence, cause and initiating appropriate therapy can significantly reduce mortality. The central elements for treating sepsis and septic shock are early recognition and intervention, antibiotics, fluid bolus, frequent clinical and laboratory assessment for severity, advanced hemodynamic support which can be achieved by adapting and implenting early goal directed therapies (EGDT), that includes preload, afterload, balancing of oxygen delivery and oxygen demand. EGDT can be tailored based on the monitoring of end organ perfusion, as the serious complications of sepsis include end organ hypoperfusion, severe organ failure and death can be avoided by effective EGDT.

DISCUSSION

The only curative option for Cholangiocarcinoma is surgical resection. However, most of the patients are diagnosed at late stages among which 10-45% turn out to be complicated during explorative laparotomy. Orthotopic liver transplantation is not recommended as monotherapy for cholangiocarcinoma because of high recurrence rates and long-term survival of less than 20%. Orthotopic liver transplantation (OLT) followed by neoadjuvant chemoradiation is an effective treatment option for perihilar cholangiocarcinoma. The rates of recurrence following this transplant protocol are 20%, and recurrence free 5-year survival 68%.[6] Nonsurgical therapies include chemotherapy which is considered to be resistant but a phase III randomized controlled trial treated with gemcitabine/cisplatin-combination therapy versus gemcitabine monotherapy showed 6 months survival benefit in cholangiocarcinoma patients.[1]

Sepsis is a medical emergency condition that narrates the body's immunological response to an infectious process that can lead to end-organ dysfunction and death. The transition of sepsis to Multiple Organ Dysfunction Syndrome can be prevented with appropriate and rapid resuscitation of shock.[6]

Prerenal sources of Acute Kidney Injury can be managed with volume replacement and hemodynamic support. If the cause is immune-related (interstitial nephritis or glomerulonephritis), immunosuppressive therapy should be started. Renal replacement therapies may be necessary to main fluid and electrolyte balance.[5]

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We sincerely thank the patient's family for their corporation in understanding & providing us all the required details.

Statement of Ethics
Since the patient had died, the written informed consent for publication of the details of the medical case and the images was taken from his kin. All attempts have been made to maintain confidentiality of the patient.

Conflict of Interest Statement
The authors have no conflicts of interest to declare.

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