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Case Study

Assessment of Chemotherapy-Induced Toxicities of FOLFOX Regimen in Pancreatic Cancer Patients

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ABSTRACT

Pancreatic cancer is an aggressive gastrointestinal cancer with a poor prognosis and high fatality rate because of its rapid progression and delayed detection. The FOLFOX regimen, which includes 5-Fluorouracil, Leucovorin, and Oxaliplatin, is a common treatment for advanced and metastatic pancreatic cancer. Chemotherapy is typically well tolerated during the initial cycles; nevertheless, toxicities such as Myelosuppression, Hepatotoxicity, Nephrotoxicity, Electrolyte imbalance, and Peripheral Neuropathy may appear in subsequent cycles. Early toxicity diagnosis, dose modification, and appropriate supportive care are necessary to improve treatment outcomes, patient compliance, and quality of life.

INTRODUCTION

Pancreatic cancer (PC) has become more common in recent years. It is linked to 5% of cancer-related fatalities and makes up around 2% of all cancers. When the disease progresses to advanced pancreatic metastases, when tumor cells are very invasive, the majority of individuals do not exhibit any noticeable symptoms. It is now one of the most dangerous malignant tumors, and early diagnosis is challenging. Even with potentially drastic treatment, the majority of patients eventually relapse, and their 5-year survival rate is only 2% to 9%.3 Among PC patients, pancreatic ductal adenocarcinoma (PDAC) is the most prevalent. Diabetes, pancreatitis, and family history are some risk factors for Pancreatic Cancer.[1]

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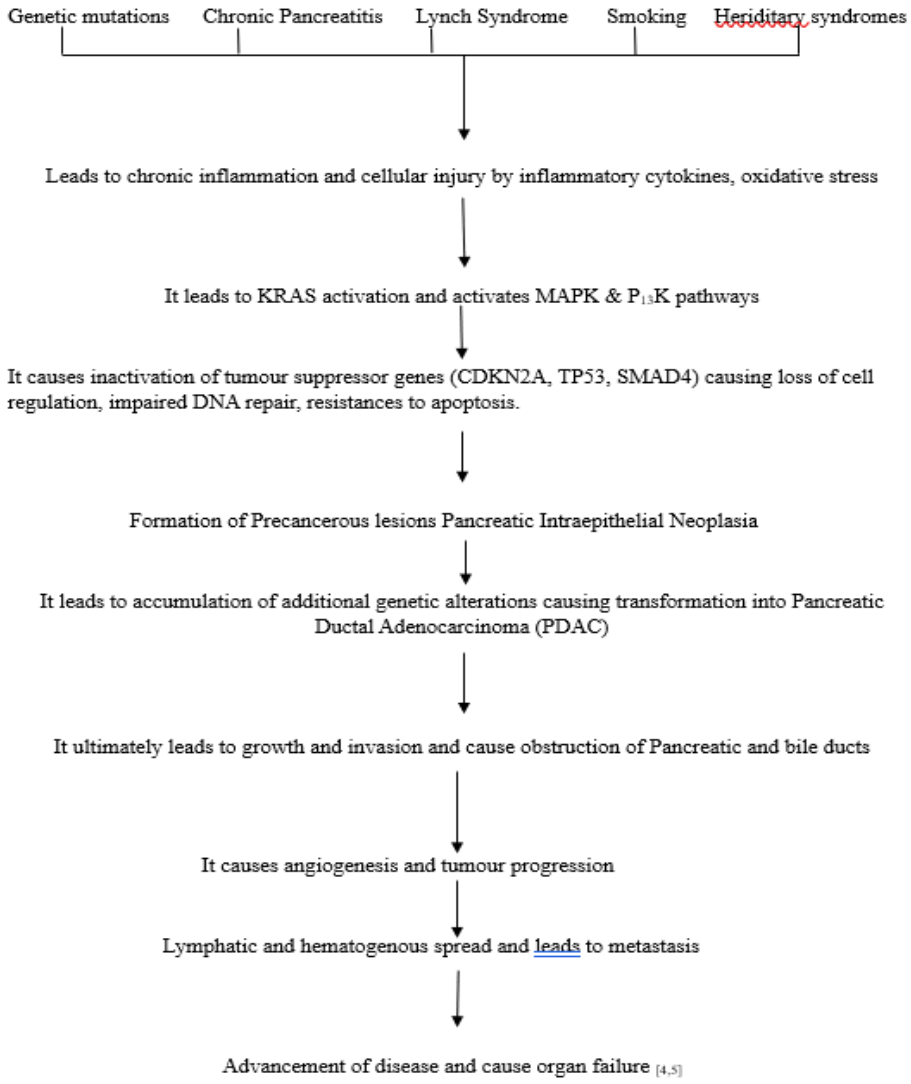
ETIOLOGICAL FACTORS:

The most common factors that account for Pancreatic Cancer includes smoking, genetic mutations, chronic pancreatitis, Lynch syndrome, Heriditary syndromes, family history of Pancreatic cancer, lifestyle factors. [2]

EPIDEMIOLOGY:

The Global Cancer Observatory (GLOBOCAN) 2020 estimates that 495,773 new cases of pancreatic cancer were detected globally in 2020, making it the 12th most common malignant tumor. The age-standardized incidence rate (ASR) was $4.9/10^5$, whereas the worldwide crude incidence rate was $6.4/10^5$. With a crude mortality of $6.0/10^5$, pancreatic cancer ranked seventh among all malignant tumors in 2020, accounting for an anticipated 466,003 deaths. [3]

PATHOGENESIS:



SIGNS & SYMPTOMS: [6]

The symptoms experienced by Pancreatic cancer patients include

- Loss of apetite
- Abdominal pain
- Jaundice
- Unexplained weight loss
- Diarrhoea or constipation
- Pale stools
- Unusual bloating

- Unusual belching
- Dark urine
- Fatigue

RISK FACTORS:

Risk factors for Pancreatic Cancer are classified as non-modifiable (age, sex, area, blood group, family history and genetic susceptibility, diabetes) and modifiable (intestinal microflora, smoking, alcohol, chronic pancreatitis [CP], obesity, dietary factors, infection). [7]

DIAGNOSIS:

CT Scan: Multi detector computed tomography is the tool for producing multi planar images for pancreatic adenocarcinoma.

MRI: MRI enables early diagnosis of tumours mainly morphological changes of pancreatic parenchyma as well as that of Pancreatic duct.

EUS with fine needle aspiration: In addition to being a safe and well-tolerated treatment, EUS offers the advantage of enabling fine needle aspiration for the purpose of obtaining a cytopathological diagnosis. It works very well for lesions less than 2 cm.

PET: PET with 18FDG is combined with CT to produce fusion images and enables staging of cancer.

ULTRASOUND AND ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY: It plays a prominent role in diagnosis of cancer and mainly used as a therapeutic modility. [7-8]

TREATMENT:

PHARMACOLOGICAL THERAPY

General chemotherapy regimens used in Pancreatic cancer include:

- Gemcitabine – 1000 mg/m², IV infusion over 30 minutes, Days 1, 8, and 15
- Nab Paclitaxel – 125 mg/m², IV infusion over 30 – 40 minutes, Days 1,8 and 15
- Oxaliplatin – 85mg/m², IV infusion over 2 hours, Day - 1
- 5-Fluorouracil – 400 mg/m² IV bolus, followed by 2400 mg/m² IV infusion over 46 hours
- Leucovorin – 400 mg/m² IV infusion, Day 1
- Irinotecan – 180 mg/m², IV Infusion over 90 minutes, Day 1

Dosing schedule:

- Gemcitabine + Nab – Paclitaxel regimen: Repeated every 28 days (4 week cycle)
- FOLFIRINOX regimen – Repeated every 14 days (2 week cycle)
- FOLFOX regimen - Repeated every 14 days (2 weeks cycle) [9]

NON - PHARMACOLOGICAL THERAPY:

Non-pharmacological management of Pancreatic cancer includes:

1. Surgery

- Primary treatment for resectable pancreatic cancer
- Procedures include pancreaticoduodenectomy (Whipple procedure), distal pancreatectomy, and total pancreatectomy with lymph node dissection. [10]

2. Radiation Therapy

- Mainly used in locally advanced pancreatic cancer and selected postoperative cases
- Includes external beam radiotherapy (EBRT) and stereotactic body radiation therapy (SBRT), often combined with chemotherapy [11]

3. Concurrent Chemoradiotherapy

- Used in borderline resectable and locally advanced pancreatic cancer

- Helps improve local tumour control and may increase surgical resectability [12]

4. Palliative Care

- Focuses on symptom relief and quality of life
- Includes pain management, nutritional support, pancreatic enzyme replacement, biliary stenting, and psychosocial care [13]

DRUG INFORMATION:

FOLFOX

COMPOSITION - 5-Fluorouracil + Leucovorin + Oxaliplatin

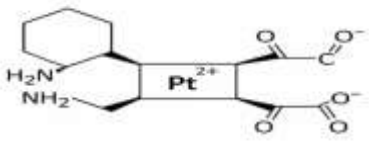
BRAND NAME - FOLFOX

CATEGORY - 5-Fluorouracil – Fluoropyrimidine agent

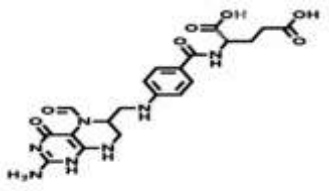
Oxaliplatin - Platinum containing antineoplastic agent

STRUCTURE:

5- Fluorouracil



Oxaliplatin



Leucovorin

MECHANISM OF ACTION:

5-Fluorouracil: 5-Fluorouracil is phosphorylated to form Fdump, FUTP. FdUMP inhibits thymidylate synthase enzyme responsible for DNA replication.

Leucovorin: It enhances the binding of FdUMP to Thymidyalte synthase enzyme and improves the inhibition of DNA repication.

Oxaliplatin: The compound causes inter and intrastrand formation in DNA and inhibits DNA repication by causing unwinging of DNA leads to tumour cell apoptosis [14]

PHARMACOKINETICS:

- **OXALIPLATIN**

Absorption: Oxaliplatin is rapidly and extensively absorbed after infusion. The bioavailability is 100%, the time taken to reach a peak concentration Tmax is 2 hours.

Distribution: Oxaliplatin is rapidly distributed. In blood platinum binds irreversibly to plasma proteins (predominantly serum albumin) and erythrocytes.

Metabolism: It rapidly and widely undergoes non-enzymatic biotransformation in the circulation by displacing its oxalate ligand, producing a number of reactive platinum-containing species. These species circulate with a range of inert conjugates that eventually bind to proteins and DNA, some of which are harmful.

Elimination: Oxaliplatin is mainly eliminated through renal route. About 30–50% of the platinum dose is recovered in urine within 2–5 days; renal clearance of ultrafilterable platinum correlates with glomerular filtration rate (GFR) [15]

- **LEUCOVORIN**

Absorption: Leucovorin is rapidly absorbed and bioavailability is 100%. Tmax of drug is 30 minutes.

Distribution: Leucovorin moderately binds to plasma proteins and distributes widely into body tissues including Liver and Gastrointestinal tract.

Metabolism: Leucovorin undergoes rapid enzymatic conversions in mucosa and Liver to 5-

methyl tetrahydrofolate. It enhances binding of FdUMP to thymidylate synthase.

Elimination: 80-90% is eliminated through urine and a very small fraction is eliminated through feces and bile. [16]

- **5- Fluorouracil**

Absorption: 5- Fluorouracil is rapidly absorbed with bioavailability of 100%

Distribution: It is rapidly distributed into tissues including Liver, GI tract. The plasma protein binding is only 10%

Metabolism: It is metabolized in Liver by enzyme Dihydropyridine dehydrogenase. Approximately 80-85% of administered drug is metabolized into its inactive metabolites.

Elimination: 60-90% of administered dose is excreted through urine and the remaining through bile. [17]

PHARMACODYNAMICS:

OXALIPLATIN: Oxaliplatin enters cancer cells and is activated non-enzymatically into reactive platinum species that covalently bind to DNA bases (mostly at the N7 sites of guanine and adenine). Intrastrand and interstrand DNA cross-links and bulky adducts are created as a result, distorting the DNA helix, preventing transcription and DNA replication, inducing DNA damage responses, and ultimately causing cell cycle arrest and apoptosis in tumour cells.

LEUCOVORIN: Leucovorin does not have intrinsic cytotoxicity; instead, it is transformed into reduced folates, which raise intracellular levels of folate cofactors, stabilize the FdUMP–thymidylate synthase–folate ternary complex, and increase and extend 5-FU cytotoxicity and thymidylate synthase inhibition.

5-FLUOROURACIL: When 5-FU enters cancer cells, it is transformed intracellularly into active metabolites like 5-fluoro-2'-deoxyuridine-5'-monophosphate (FdUMP), which inhibits thymidylate synthase, depleting thymidine and preventing DNA synthesis; other metabolites can be incorrectly incorporated into RNA/DNA, impairing their function and causing cell death in rapidly dividing cells. [18]

DOSE:

ADULTS [19]

5-Fluorouracil	400 mg/m^2 2400 mg/m^2	On day-1 Over 46 hours	IV Bolus IV Infusion
Leucovorin	200 mg/m^2	Over 2 hours	IV Infusion
Oxaliplatin	85 mg/m^2	Over 2 hours	IV Infusion

- Delivered every 14 days over 12 cycles

CLINICAL INDICATIONS:

Colon Cancer **-** Stage 2 (high risk), stage – 3 and stage - 4

Rectal Cancer **–** Adjuvant, metastatic cancer.

Gastric Cancer – Locally advanced, metasatic cancer

Pancreatic cancer – Second line agent

Cholangiocarcinoma – Second line agent [20]

ADVERSE EFFECTS:

Gastrointestinal Toxicity - Diarrhoea, nausea, vomiting, stomatitis, abdominal pain

Haematological Toxicity – Neutropenia, Thrombocytopenia, Anaemia

Peripheral Neuropathy - Paraesthesia, Dysesthesia, cold – induced neuropathy, sensory impairment

Dermatological toxicity – Hand Foot Syndrome, skin rash, nail changes

Hypersensitivity Reactions – Rash, fever, bronchospasm, anaphylaxis in severe cases

Hepatic Toxicity – Elevated liver enzymes, Sinusoidal liver injury, hyperbilirubinaemia. [21]

CONTRAINDICATIONS:

FOLFOX is contraindicated in patients with hypersensitivity to platinum or fluropyrimidine compounds, renal impairment, severe bone marrow suppression, dihyropyrimidine dehydrogenase deficiency, pregnancy and breast-feeding women. [22]

DRUG INTERACTIONS:

1. 5- Fluorouracil + Phenytoin

5- Fluorouracil will reduce the metabolism and clearance of Phenytoin leads to increased plasma concentration. This requires close monitoring of serum Phenytoin levels and dose reduction if necessary.

2. 5- Fluorouracil + Warfarin

5- Fluorouracil will reduce the metabolism of Warfarin causing increased anticoagulant effect. The combination need to avoided or monitor the increased INR levels.

3. 5- Fluorouracil + Allopurinol

The efficicacy of 5-Fluorouracil is reduced due to decreased activation of active metabolites. This combination requires close monitoring or the combination need to be avoided.

4. Oxaliplatin + Nephrotoxic drugs (Aminoglycosides, NSAID's, Amphotericin)

Oxaliplatin when combined with nephrotoxic drugs will cause increased nephrotoxicity. Try to avoid this combination or need to monitor closely.

5. Leucovorin + Pyrimethamine

Leucovorin may reduce the therapeutic efficacy of folate antagonist drugs due to reversal of folate inhibition. This combination need close monitoring or avoid the combination. [23,24]

CASE DISCUSSION:

CASE STUDY – 1

A 52 years old male patient was admitted in the Oncology department with chief complaints of progressive weight loss, upper abdominal pain radiating to back, loss of appetite and jaundice since 2 months. The patient was K/C/O Type 2 DM and HTN.

On further diagnosis, the patient was diagnosed with carcinoma head of pancreas with obstructive jaundice (cT2N1M0). Patient underwent Whipple procedure (Pancreaticoduodenectomy) on 18/06/2025. Later patient was advised for adjuvant chemotherapy cycles. On examination patient condition was BP- 120/85 mm hg, PR- 84 bpm, RR- 16/min, Temp- 98.1 F.

CECT ABDOMEN: Ill-defined hypo enhancing lesion measuring 3.2 × 2.8 cm in head of pancreas causing distal CBD obstruction with mild pancreatic duct dilatation.

MRI ABDOMEN: Soft tissue lesion involving pancreatic head with periportal lymphadenopathy.

X Ray CHEST: Mild bilateral basal fibrotic changes. No active lung lesions.

INTRA OP Findings: Growth involving head of pancreas with peri pancreatic lymph node enlargement. No liver deposits or peritoneal metastasis.

Histopathology: Features are suggestive of moderately differentiated ductal adenocarcinoma pancreas with lymph node metastasis.

PET CT: FDG avid lesion in pancreatic head with FDG avid peri pancreatic nodes likely neoplastic etiology.

On the basis of laboratory investigations the patient was diagnosed with Pancreatic carcinoma. The patient was advised for chemotherapy along with the pre medications including Inj. PALNOX 0.25mg + Inj. DEXA 8mg in 100ml NS over 15 minutes and Inj. Pantoprazole 40mg IV.

On 10/07/2025 – CYCLE 1 chemotherapy Inj. OXALIPLATIN 130mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV and Inj. 5

FLUOROURACIL 2400mg infusion over 46 hours.

On 31/07/2025 – CYCLE 2 chemotherapy Inj. OXALIPLATIN 130mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

The laboratory investigations were found to be normal in Cycle 1 and Cycle 2 of chemotherapy.

On 21/08/2025 – CYCLE 3 chemotherapy Inj. OXALIPLATIN 130mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

Few laboratory parameters were found to be abnormal, like Hb – 10.1 gm/dL, WBC – 3800 cells/cu mm, Platelets – 1.25 lakh/cu mm, Total bilirubin – 1.8 mg/dL.

CASE STUDY – 2

A 62 years old female patient was admitted in the Oncology department with chief complaints of severe epigastric pain, vomiting, anorexia and significant weight loss since 3 months. The patient had history of Hypertension.

The patient was diagnosed with locally advanced carcinoma body of pancreas (cT4N1M0). Patient was not suitable for surgery due to vascular invasion and was planned for chemotherapy. On examination patient condition was BP- 125/82 mm hg, PR- 86 bpm, RR- 18/min, Temp- 98.4F.

CECT ABDOMEN: Heterogeneous hypo dense lesion involving body of pancreas encasing splenic artery and proximal celiac axis.

MRI PANCREAS: Irregular infiltrative lesion in pancreatic body with peri pancreatic fat stranding.

X Ray CHEST: Mild cardiomegaly with no focal lung opacity.

PET CT: FDG avid lesion involving body of pancreas with regional nodal uptake.

Histopathology: Features suggestive of poorly differentiated adenocarcinoma pancreas.

Based upon the laboratory investigations the patient was diagnosed with locally advanced pancreatic carcinoma. The patient was advised for chemotherapy along with pre medications including Inj. Ondansetron 8mg IV, Inj. DEXA 8mg IV and hydration with NS.

On 05/08/2025 – CYCLE 1 chemotherapy Inj. OXALIPLATIN 130mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV, and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

On 26/08/2025 – CYCLE 2 chemotherapy Inj. OXALIPLATIN 130mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV, and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

The laboratory investigations were found to be stable in Cycle 1 and Cycle 2 chemotherapy.

On 16/09/2025 – CYCLE 3 chemotherapy Inj. OXALIPLATIN 130mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV, and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

Few laboratory parameters like Hb – 9.4 gm/dL, Neutrophils – 42%, Serum creatinine – 1.3 mg/dL, Serum potassium – 3.3 mEq/L were found to be abnormal.

CASE STUDY – 3

A 68 years old male patient was admitted in the Oncology department with chief complaints of jaundice, generalized weakness, pruritus and clay coloured stools since 1 month. The past medical history of the patient includes K/C/O CAD, Type 2 DM and Chronic Kidney Disease.

Upon further investigation the patient was diagnosed with metastatic carcinoma pancreas with liver metastasis (cT3N1M1). ERCP with biliary stenting was done on 12/05/2025 for obstructive jaundice. Patient was planned for palliative chemotherapy. On examination patient

condition was BP- 116/80 mm hg, PR- 86 bpm, RR- 22/min, Temp- 98F.

CECT ABDOMEN: Irregular mass lesion in head and uncinate process of pancreas with multiple hypodense liver lesions.

MRCP: Distal CBD narrowing with moderate intrahepatic biliary dilatation.

X Ray CHEST: Mild bilateral hilar prominence.

PET CT: FDG avid pancreatic head lesion with multiple FDG avid hepatic metastases.

Histopathology: Features are suggestive of adenocarcinoma pancreas.

Based upon the laboratory investigations the patient was diagnosed with metastatic pancreatic carcinoma. The patient was advised for palliative chemotherapy along with pre medications including Inj. PALNOX 0.25mg IV and Inj. DEXA 8mg IV.

On 20/05/2025 – CYCLE 1 chemotherapy Inj. OXALIPLATIN 85mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

On 10/06/2025 – CYCLE 2 chemotherapy Inj. OXALIPLATIN 85mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

Laboratory investigations showed mild improvement in bilirubin after biliary stenting.

On 01/07/2025 – CYCLE 3 chemotherapy Inj. OXALIPLATIN 85mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

Few laboratory parameters were found to be abnormal, like Hb – 8.9 gm/dL, Total bilirubin – 2.4 mg/dL, SGOT – 78 IU/L, Albumin – 2.9 g/dL.

CASE STUDY – 4

A 70 years old female patient was admitted in the Oncology department with chief complaints of abdominal discomfort, early satiety, back pain and loss of appetite since 2 months. The past medical history of the patient includes K/C/O Hypothyroidism.

Upon further investigation the patient was diagnosed with borderline ressectable carcinoma pancreas (cT3N0M0). Patient received neoadjuvant chemotherapy before surgery. On examination patient condition was BP- 124/78 mm hg, PR- 84 bpm, RR- 18/min, Temp- 97.8F.

CECT ABDOMEN: Hypo enhancing lesion measuring 4.1 × 3.5 cm in pancreatic neck abutting superior mesenteric vein.

PET CT: FDG avid lesion in pancreatic neck with no distant metastasis.

Endoscopic Ultrasound Guided Biopsy: Adenocarcinoma pancreas.

INTRA OP Findings: Tumour involving pancreatic neck with focal venous adherence. No liver deposits.

Histopathology: Moderately differentiated ductal adenocarcinoma.

Based upon the laboratory investigations the patient was diagnosed with borderline resectable pancreatic carcinoma. The patient was advised for neoadjuvant chemotherapy along with antiemetic and hydration support.

On 14/07/2025 – CYCLE 1 chemotherapy Inj. OXALIPLATIN 130mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

On 04/08/2025 – CYCLE 2 chemotherapy Inj. OXALIPLATIN 130mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

The laboratory investigations were found to be within acceptable range in first two cycles.

On 25/08/2025 – CYCLE 3 chemotherapy Inj. OXALIPLATIN 130mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV and Inj. 5

FLUOROURACIL 2400mg infusion over 46 hours.

Few laboratory parameters were found to be abnormal, like Hb – 10.3 gm/dL, Platelets – 1.1 lakh/cu mm, Serum magnesium – 1.5 mg/dL.

CASE STUDY – 5

A 64 years old male patient was admitted in the Oncology department with chief complaints of persistent abdominal pain, progressive jaundice, fatigue and weight loss since 4 months. The past medical history of the patient includes K/C/O HTN, Chronic smoker and Alcohol consumption.

Upon further investigation the patient was diagnosed with carcinoma tail of pancreas with peritoneal metastasis (cT4N1M1). Patient was treated conservatively with palliative intent chemotherapy. On examination patient condition was BP- 116/74 mm hg, PR- 94 bpm, RR- 20/min, Temp- 98.6F.

CECT ABDOMEN: Large irregular hypo dense lesion involving tail of pancreas with omental deposits and mild ascites.

PET CT: FDG avid lesion in pancreatic tail with metabolically active peritoneal deposits.

Ascitic Fluid Cytology: Positive for malignant cells.

Histopathology: Poorly differentiated adenocarcinoma pancreas.

X Ray CHEST: Mild bilateral pleural effusion.

Based upon the laboratory investigations the patient was diagnosed with metastatic pancreatic carcinoma. The patient was advised for palliative chemotherapy along with pre medications including Inj. Ondansetron 8mg IV, Inj. DEXA 8mg IV and IV fluids.

On 18/08/2025 – CYCLE 1 chemotherapy Inj. OXALIPLATIN 130mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

On 08/09/2025 – CYCLE 2 chemotherapy Inj. OXALIPLATIN 130mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

The laboratory investigations were found to be acceptable in first two cycles.

On 29/09/2025 – CYCLE 3 chemotherapy Inj. OXALIPLATIN 130mg IV in 5% dextrose over 2 hours, Inj. LEUCOVORIN 400mg IV and Inj. 5 FLUOROURACIL 2400mg infusion over 46 hours.

Few laboratory parameters were found to be abnormal, like Hb – 9.1 gm/dL, RBC – 3.2 million/cu mm, Serum sodium – 131 mEq/L, CA-19-9 – elevated.

CONCLUSION

One of the most popular chemotherapy regimens for managing pancreatic cancer is the FOLFOX regimen, particularly for patients with locally progressed, metastatic, recurring, or marginally resectable disease. For the majority of patients, the regimen demonstrated acceptable tolerance during the first cycles of chemotherapy. But after Cycle 2 or Cycle 3, toxicities were frequently noted, necessitating ongoing observation and supportive care.

CASE STUDY – 1; 52 years old, male with carcinoma head of pancreas tolerated initial cycles of FOLFOX, after cycle 3 patient developed anemia, thrombocytopenia and mild hyperbilirubinemia after Cycle 3.

CASE STUDY – 2; 62 years old female with locally advanced carcinoma body of pancreas treated with FOLFOX developed anemia, electrolyte imbalance and mild renal impairment after Cycle 3. The patient required hydration therapy and close biochemical monitoring.

CASE STUDY – 3; 68 years old male diagnosed with metastatic carcinoma pancreas with liver metastasis tolerated initial cycles of FOLFOX but

later developed anemia, elevated bilirubin, hypoalbuminemia and hepatotoxicity.

CASE STUDY – 4; 70 years old female with borderline resectable pancreatic carcinoma treated with neoadjuvant FOLFOX developed thrombocytopenia, anaemia and hypomagnesemia after Cycle 3. Electrolyte correction and supportive medications were administered.

CASE STUDY – 5; 64 years old male diagnosed with metastatic carcinoma tail of pancreas treated with FOLFOX developed myelosuppression, hyponatremia and elevated tumour marker levels after subsequent cycles.

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