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Study on Prescription Pattern in Medical Management of Pancreatitis

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ABSTRACT
The pancreas is a crucial organ in the digestive and endocrine systems. Pancreatitis is an inflammation of the pancreas. Acute pancreatitis is characterized by severe pain in the upper abdomen and elevation of pancreatic enzymes in the blood. In the majority of patients, acute pancreatitis is a mild self-limiting disease that resolves spontaneously without complications but can be severe with organ failure, morbidity, and mortality. Chronic pancreatitis is characterized by permanent damage of pancreatic structure and function because of progressive inflammation and long-standing pancreatitis. To study the prescribing pattern in the management of pancreatitis. To study and analyse the pattern and usage of medications in the management of pancreatitis. Patients who meet the study criteria will be included in the study.A total of 108 subjects were evaluated, 50 subjects were diagnosed with acute pancreatitis, and 58 subjects were diagnosed with chronic pancreatitis.The majority of the subjects in both acute pancreatitis and chronic pancreatitis belong to the age group 31-45 years. A high female predominance was found in 57.1% of the total acute pancreatitis subjects, whereas in chronic predominance 55.3% was found. In acute pancreatitis was the most common comorbidity followed by diabetes mellitus (22%), Hypertension (22%), In chronic pancreatitis, Diabetes mellitus (32.8) was the most common co-morbidity followed by pancreatitis (27.5%), Hypertension (22.4%). These are the comorbidities observed in the study population. Serum amylase and serum lipase are the comorbidities tests obtained as biochemical markers for both acute and chronic pancreatitis. Among them, 45.4% had elevated serum amylase and 37% had elevated levels of serum lipase. Drugs used to treat pain in both acute pancreatitis and chronic pancreatitis are tramadol, Fentanyl, and Paracetamol. Tramadol was the most commonly used drug in both acute pancreatitis (88%) and chronic pancreatitis (75.9%). This study revealed that almost all the participants had a history of alcohol use, whereas biliary was the major cause of Acute pancreatitis.
Keywords: Acute Pancreatitis, Chronic Pancreatitis, gall stone, Exocrine Pancreatic Insufficiency (EPI)

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INTRODUCTION

Exocrine pancreatic insufficiency (EPI) results from the destruction of the pancreatic parenchyma with a sufficiently large loss of acinar cells and/or obstruction of pancreatic ducts that it is not possible to maintain the minimum production levels of digestive enzymes and ductal bicarbonate secretion required to adequately digest food [1]. Chronic pancreatitis (CP) is the most frequent cause of EPI in adults [2].

Acute pancreatitis is characterized by severe pain in the upper abdomen and elevation of pancreatic enzymes in the blood. In majority of the patients, acute pancreatitis is a mild self-limiting disease that resolves spontaneously without complications but can be severe with organ failure, morbidity and mortality [3].Chronic pancreatitis (CP) is a syndrome characterized by chronic progressive pancreatic inflammation, fibrosis, and scarring, resulting in damage to and loss of exocrine (acinar), endocrine (islet cells).Exocrine pancreatic insufficiency is a condition characterized by the inability of the pancreas to produce or deliver sufficient quantities of digestive enzymes and bicarbonate into the small intestine to ensure proper digestion and nutrient absorption. This deficiency results from significant destruction of the pancreatic parenchyma, predominantly involving the acinar cells, or from ductal obstruction that

impairs enzyme delivery. Symptoms of EPI include steatorrhea, weight loss, bloating, and malnutrition. As the exocrine function of the pancreas is impaired, the digestion of fats, proteins, and carbohydrates is compromised, leading to clinical and biochemical manifestations of malabsorption [4].

The etiological factors of acute and chronic pancreatitis are listed as follows:

Gallstones – The obstruction of the sphincter of Oddi by gallstones leads to pancreatic inflammation, making it the most common cause of acute pancreatitis [5].Alcohol – Excessive alcohol consumption, particularly binge drinking or chronic intake, triggers acute pancreatitis by inducing pancreatic inflammation and damage [6].Hypertriglyceridemia – Elevated triglyceride levels, especially >1000 mg/dL, cause acute pancreatitis by releasing free fatty acids, which damage pancreatic acinar and endothelial cells [7].

Hypercalcemia – High calcium levels lead to pancreatitis by depositing calcium salts in the pancreatic ducts and activating trypsinogen within pancreatic tissue.Medications – Certain drugs, including corticosteroids, diuretics, and antibiotics, can trigger acute pancreatitis through unclear mechanisms involving proinflammatory mediators [8-10].ERCP Procedures – Endoscopic Retrograde Cholangiopancreatography (ERCP) increases the risk of acute pancreatitis, especially in young women or patients with repeated bile duct cannulation attempts [11].Infections – Bacterial, viral, fungal, and parasitic infections can induce pancreatitis by obstructing the pancreatic duct or directly damaging pancreatic tissues. Idiopathic – In some cases, the cause of acute pancreatitis remains unknown despite extensive evaluation [8].

In 10–30% of cases, no specific cause is identified despite thorough clinical, laboratory, and imaging evaluations. Idiopathic pancreatitis may be associated with microlithiasis (tiny gallstones), genetic predispositions (e.g., PRSS1, SPINK1, CFTR mutations), or undetected autoimmune mechanisms. Recurrent idiopathic pancreatitis warrants further investigation, including endoscopic ultrasound and genetic testing.

A rare but increasingly recognized cause of chronic pancreatitis, autoimmune pancreatitis (AIP) is part of the spectrum of IgG4-related disease. It is characterized by lymphoplasmacytic infiltration, fibrosis, and responsiveness to corticosteroids. AIP may mimic pancreatic cancer in imaging and clinical presentation [12, 13]. Blunt or penetrating abdominal trauma can damage the pancreas directly or compromise its blood supply, leading to acute inflammation.

Complications

Acute pancreatitis leads to a wide range of local and systemic pathophysiologic alterations and to a large variability in the clinical manifestation and prognosis. Local complications of acute pancreatitis typically arise approximately 3 to 4 weeks after the initial attack and encompass acute fluid collection, pancreatic necrosis, infection, abscess formation, and pseudocyst development. Infections of necrotic tissue occur in 15% to 30% of cases and predominantly stem from secondary infections. Mortality often results from complications such as infected necrosis, pancreatic abscesses, and sepsis. Pancreatic ascites occurs when pancreatic secretions disseminate throughout the peritoneal cavity [14-18].

Lab Investigations

C-Reactive Protein (CRP): CRP is a liver-produced protein that increases in response to inflammation and helps distinguish between mild and severe pancreatitis. Its levels peak 48 hours after symptom onset, making it a useful diagnostic marker [19].

Gamma-Glutamyl Transferase (GGTP): GGTP is a liver enzyme used to monitor alcohol consumption, with levels rising even with small alcohol intake. Chronic drinkers show significantly elevated GGTP levels, indicating potential liver damage [20].

Serum Amylase: Serum amylase helps diagnose pancreatic inflammation, with levels rising within 4–8 hours of an attack and normalizing in 8–14 days. Amylase levels exceeding three times the normal limit indicate pancreatitis and possible pancreatic complications [21].

Serum Lipase: Lipase, an enzyme produced by the pancreas, aids in fat digestion and has a diagnostic sensitivity of at least 85%. Elevated lipase levels indicate pancreatic inflammation, remaining high until the condition resolves [22-24].

Prescription Pattern

Prescription pattern analysis is done to determine the prescribed frequency of commonly used drugs. The goal of rational drug prescribing is to use the least number of medications to achieve the best results in the shortest amount of time at a reasonable cost. Measurement of drugs used in health facilities is not only for explaining drug use patterns and prescribing behaviour but also helps in the identification of factors responsible for the practice of polypharmacy and the problems associated with it.

MATERIAL AND METHODS

Study Protocol

A prospective, observational study is conducted for six months after the approval of the Institutional Ethics Committee (IEC). Patients who meet the study criteria are included in the study. The required data is collected from the patient case sheets. After getting informed consent, the required data will be collected from the patients and their informants (primary caregivers). The data was analysed to evaluate the prescription pattern in the management of pancreatitis.

Study Design

An institutional-based prospective observational study was conducted for six months.

Study Site

The study has been conducted in the Department of Gastroenterology, Yashoda Hospital, Secunderabad.

Study Period

The study has been conducted for a period of six months from September 2023 to February 2024.

Study Population

108 consecutive patients presenting with features of pancreatitis on an outpatient and inpatient basis in the Gastroenterology department.

Study Criteria

Inclusion Criteria

Males and females of age group 18-70 years of age. Confirmed cases of pancreatitis (Acute pancreatitis and Chronic pancreatitis) are going to be included in this study. Both Alcoholic and non-alcoholic patients will be included.

Exclusion Criteria

Pediatric age groups are excluded. Pregnant and lactating women are excluded. Patients were diagnosed with cardiac abnormalities. Patients are diagnosed with chronic kidney disease, congestive heart failure, pancreatic carcinoma, and bowel and large intestine cancers.

Statistical analysis.

Software used SPSS version 24.P value less than 0.05 is considered significant since the CI is 95%. Test performed Chi-square [25,26].

RESULTS AND DISCUSSION

Out of 108 subjects included, 50 subjects were diagnosed with acute pancreatitis, and 58 subjects were diagnosed with chronic pancreatitis of the total 108 subjects included, in acute pancreatitis 14% belong to the age group 18-30 years, 40% belong to the age group 31-45 years, 36% belong to the age group of46-60 years, 10% belong to the age group of above 60 years. Whereas in chronic pancreatitis 32.8% belongs to the age group 18-30 years, 43.1% belongs to the age group 31-45 years, 20.7% belongs to the age group 46-60 years and 3.4% belongs to the age group above 60 years. The majority of the subjects in both acute pancreatitis and chronic pancreatitis belong to the age group 31-45 years. It was found that the age group of 31-45 years is most affected by acute and chronic pancreatitis Shown in Table 1 and Figure 1.

Table 1: Age Distribution

AGE		Acute pancreatitis	Chronic pancreatitis	Total
18-30 Years	Count	7	19	26
	Percentage	14.0%	32.8%	24.1%
31-45 Years	Count	20	25	45
	Percentage	40.0%	43.1%	41.7%
46-60 Years	Count	18	12	30
	Percentage	36.0%	20.7%	27.8%
Above 60	Count	5	2	7
	Percentage	10.0%	3.4%	6.5%
Total	Count	50	58	108
	Percentage	100.0%	100.0%	100.0%

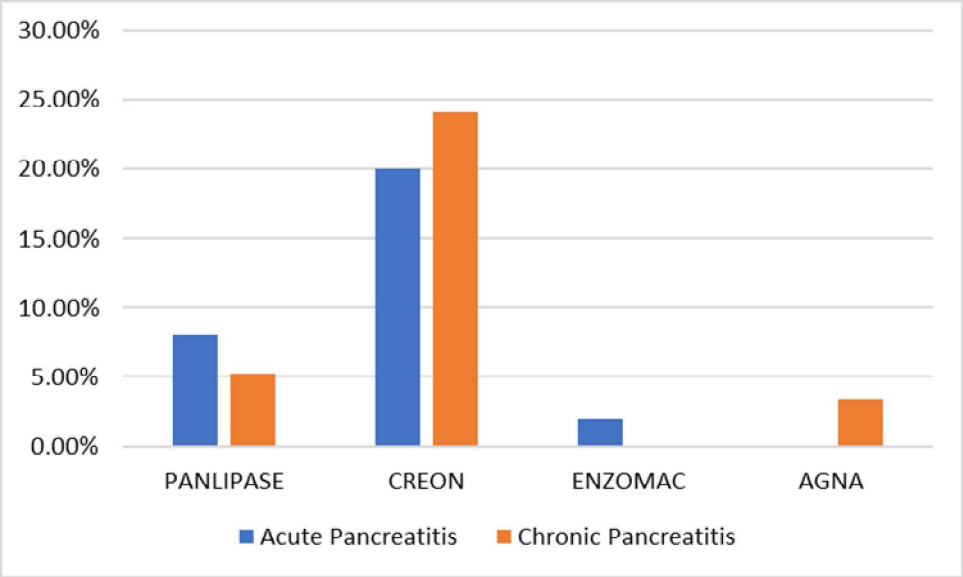

Figure 1: Distribution based on Age

A high female predominance was found in 57.1% of the total acute pancreatitis subjects, whereas in chronic predominance 55.3% was found. It was observed that acute pancreatitis is the most common in females compared to males. Whereas, in chronic pancreatitis, males are most affected **Table 2 Figure 2.** In subjects with acute pancreatitis, biliary was found to be a major cause 30%, whereas in chronic pancreatitis alcohol was found to be the major cause 50% **Table3.**

Table 2: Based on Gender

Gender		Acute pancreatitis	Chronic pancreatitis	Total
Female	Count	8	6	14
	Percentage	57.1%	42.9%	100.0%
Male	Count	42	52	94
	Percentage	44.7%	55.35%	100.0%
Total	Count	50	58	108
	Percentage	46.35	53.7%	100.0%

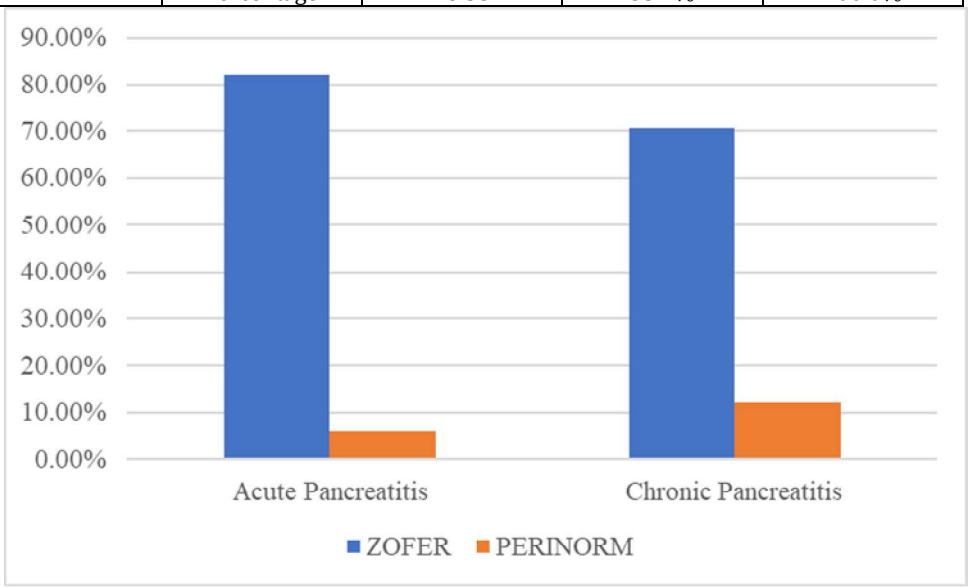

Figure 2: Based on Gender

Table 3: Based on Personal History

		Acute Pancreatitis	Chronic Pancreatitis
Alcoholic	Count	14	29
	Percentage	28.0%	50.0%
Smoker	Count	2	4
	Percentage	4.0%	6.9%
Biliary	Count	15	3
	Percentage	30.0%	5.2%

In acute pancreatitis, Pancreatitis (44%) was the most common comorbidity followed by diabetes mellitus (22%), Hypertension (22%), Cholelithiasis (20%), Hypothyroidism (4%) and Idiopathic (2%). In chronic pancreatitis, Diabetes mellitus (32.8) was the most common co-morbidity followed by pancreatitis (27.5%), Hypertension (22.4%), cholelithiasis (5.2%), Hypothyroidism (3.4%) and idiopathic (1.7%). These are the comorbidities observed in the study population **Table 4.**

Table 4: Based on Comorbidities

Comorbidities		Acute pancreatitis	Chronic pancreatitis
Idiopathic	Count	1	1
	Percentage	2.0%	1.7%
Hypertension	Count	11	13
	Percentage	22.0%	22.4%
Diabetes mellitus	Count	11	19
	Percentage	22.0%	32.8%
Cholelithiasis	Count	10	3
	Percentage	20.0%	5.2%
Pancreatitis	Count	22	16
	Percentage	44.0%	27.5%
Hypothyroidism	Count	2	2
	Percentage	4.0%	3.4%

Serum amylase and serum lipase are the comorbidities tests obtained as biochemical markers for both acute and chronic pancreatitis. Among them, 45.4% had elevated serum amylase and 37% had elevated levels of serum lipase **Table 5.** In acute pancreatitis both dextrose normal saline (75 ml/hr) and ringer lactate (75 ml/hr) were the most commonly given IV fluids, whereas in chronic pancreatitis IV fluids are not routinely recommended as per guidance but it was found in dehydration and nutritional insufficiency fluids were recommended **Table 6.**

Table 5: Based on Lab investigations

Value	Serum Amylase		Serum lipase	
	Frequency	Percentage	Frequency	Percentage
Below Normal	0	0	2	1.9
Normal	12	11.1	15	13.9
Above Normal	49	45.4	40	37.0

Table 6: Based on IV Fluids

IV fluids		Acute pancreatitis	Chronic pancreatitis
Normal Saline	Count	7	11
	Percentage	14.0%	18.9%
Dextrose Normal Saline	Count	19	22
	Percentage	38.0%	37.9%
Ringer Lactate	Count	17	20
	Percentage	34.0%	34.4%

Drugs used to treat pain in both acute pancreatitis and chronic pancreatitis are tramadol, Fentanyl, and Paracetamol. Tramadol was the most commonly used drug in both acute pancreatitis (88%) and chronic pancreatitis (75.9%) **Table 7 Figure 3.** In analgesics, Paracetamol was the most commonly used drug in acute pancreatitis (44%) and chronic pancreatitis (32.7%). Tab. Creon was the most commonly prescribed drug in both acute pancreatitis (20%) and chronic pancreatitis (24.1%) **Table 8 Figure 4.**

Table 7: Drugs used to treat pain in both acute pancreatitis and chronic pancreatitis

Drugs		Acute pancreatitis	Chronic pancreatitis
Paracetamol	Count	22	19
	Percentage	44.0%	32.7%
Tramadol	Count	44	44
	Percentage	88.0%	75.9%
Fentanyl	Count	8	9
	Percentage	16.0%	15.5%

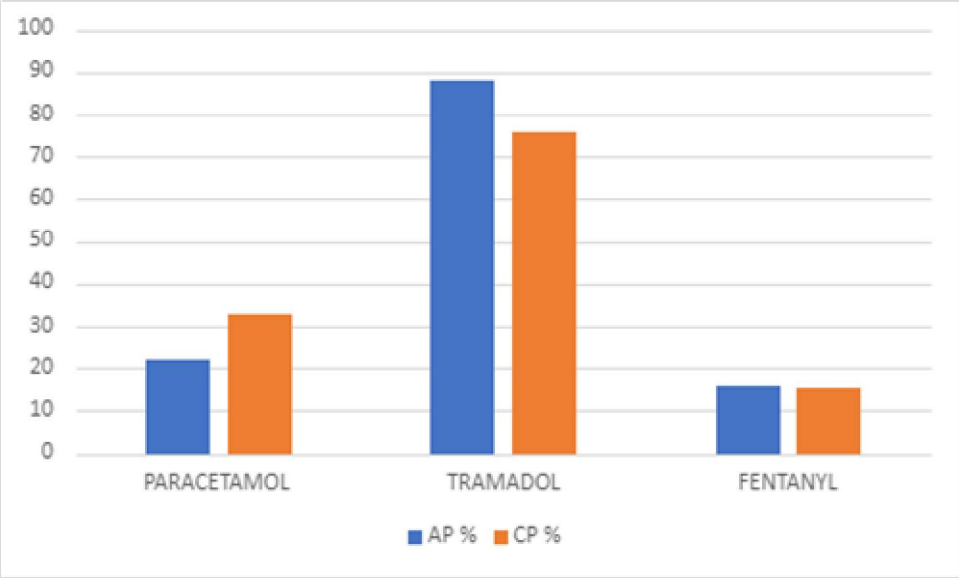

Figure 3: Based on the frequency of analgesics and opioids given

Table 8: Based on pancreatic enzymes supplements

Enzyme		Acute Pancreatitis	Chronic Pancreatitis
Panlipase	Count	4	3
	Percentage	8.0%	5.2%
Creon	Count	10	14
	Percentage	20.0%	24.1%
Enzomac	Count	1	0
	Percentage	2.0%	0.0%
Agna	Count	0	2
	Percentage	0.0%	3.4%

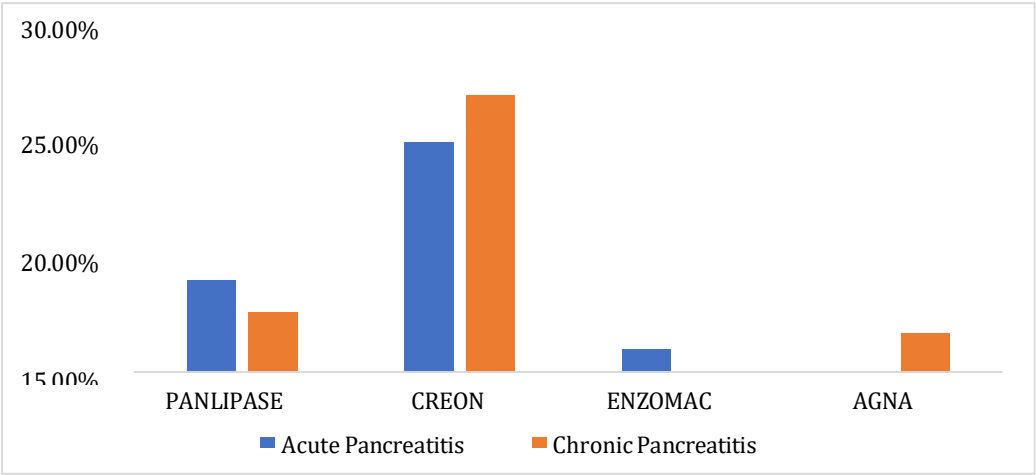

Figure 4: Based on enzymes

Inj. Magnex Forte was the most commonly used antibiotic in both acute pancreatitis (68%) and chronic pancreatitis (37.9%) **Table 9 Figure 5.** Inj.Optineuron was the most commonly used vitamin supplement in both acute pancreatitis (56%) and chronic pancreatitis (43.1%) **Table 10.** It was observed that pantoprazole was the only proton pump inhibitor used in pancreatitis **Table 11 Figure 6**. Inj. Zofer was the most commonly used antiemetic used in pancreatitis. **Table 12 Figure 7.**

Table 9: Based on antibiotics

Antibiotic		Acute Pancreatitis	Chronic Pancreatitis
Magnex Forte	Count	34	22
	Percentage	68.0%	37.9%
Metrogyl	Count	11	5
	Percentage	22.0%	8.6%
Cilanem	Count	5	2
	Percentage	10.0%	3.4%
Piptaz	Count	0	2
	Percentage	0.0%	3.4%
Monocef	Count	2	4
	Percentage	4.0%	6.9%

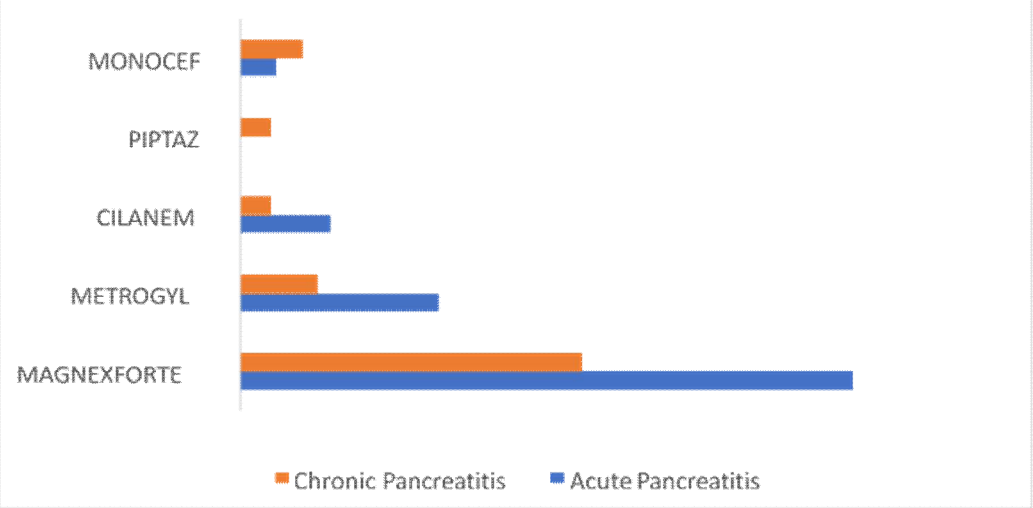

Figure 9: Based on Antibiotics

Table 10: Based on Vitamin supplements

Vitamin Supplement		Acute Pancreatitis	Chronic Pancreatitis
OPTINEURON	Count	28	25
	Percentage	56.0%	43.1%
THIAMINE	Count	11	10
	Percentage	22.0%	17.2%
ANTOXIPAN	Count	5	14
	Percentage	10.0%	24.1%

Vitamin supplement: Table 10: Based on Vitamin supplements: optineiuron most commonly prescribed in acute pancreatitis and antoxipan least prescribed vitamin supplement in acute pancreatitis. thiamine more prescribed in acute pancreatisn and antoxipan more prescribed in chronic pancreatitis.

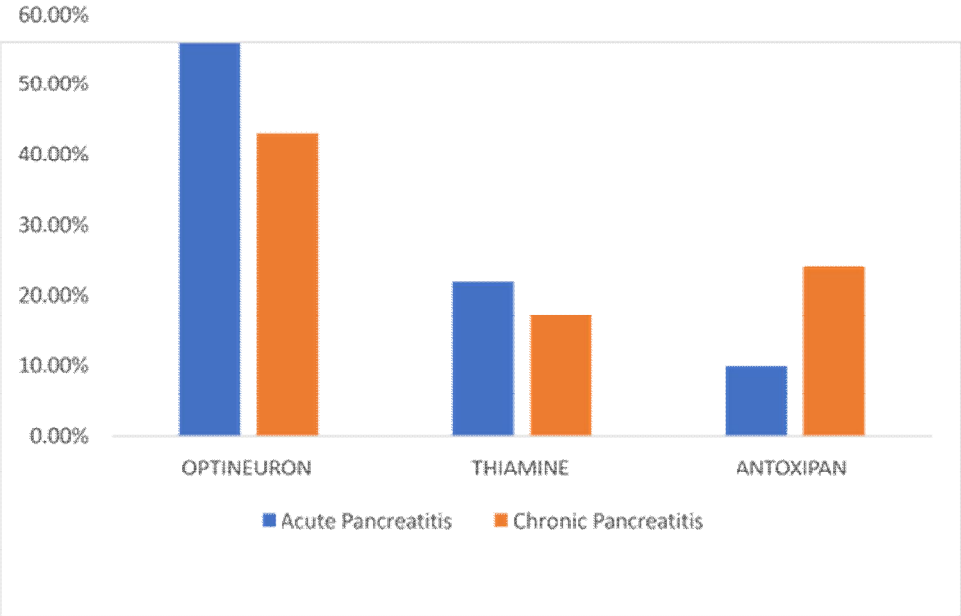

Figure 10: Based on Vitamin supplements

Table 11: Based on Proton pump inhibitors

		Acute Pancreatitis	Chronic Pancreatitis
PANTOPRAZOLE	Count	50	52
	Percentage	100.0%	89.7%

Pantoprazole more commonly prescribed in chronic pancreatitis compared to acute pancreatitis

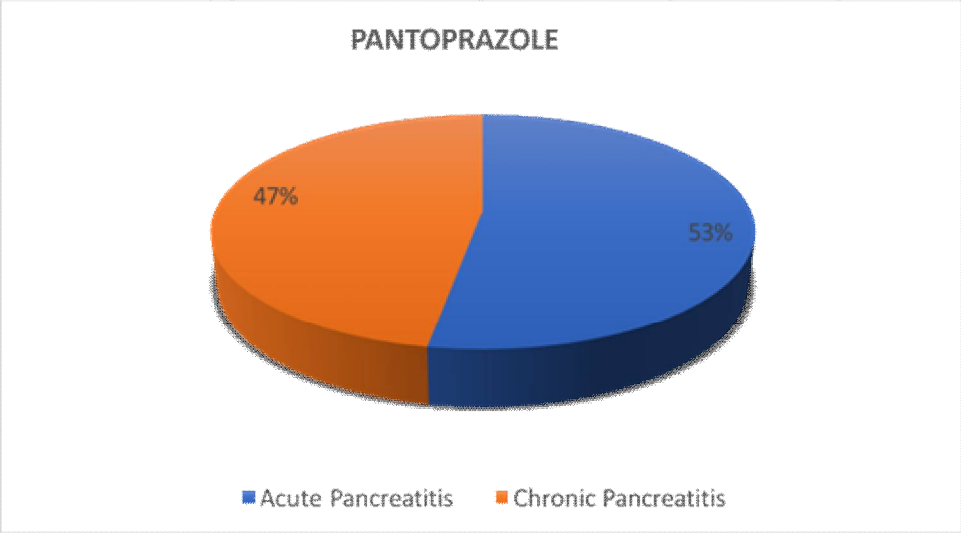

Figure 11: Based on Proton pump inhibitors

Table 12: Based on antiemetics

		Acute Pancreatitis	Chronic Pancreatitis
ZOFER	Count	41	41
	Percentage	82.0%	70.7%
PERINORM	Count	3	7
	Percentage	6.0%	12.1%

Inj. zofer is commonly prescribed in both acute pancreatitis and chronic pancreatitis, inj. perinorm more prescribed in chronic pancreatitis less in acute pancreatitis.

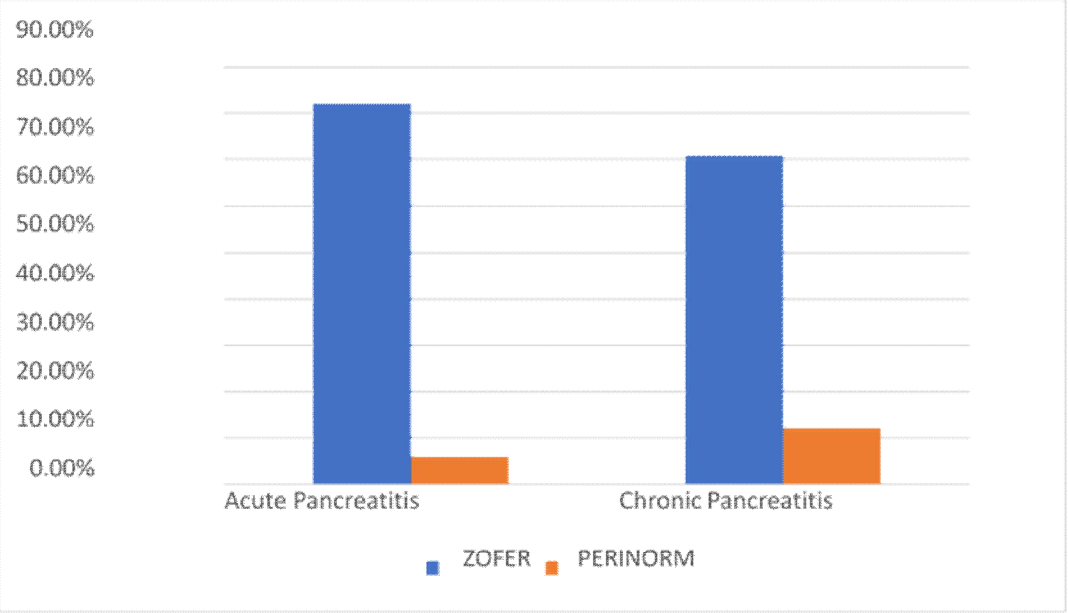

Figure 12: Based on antiemetics

LIMITATIONS

Due to a small sample size, demographic aspects may not be accurately represented. Due to alcoholism being less common in Indian women, we were unable to examine the clinical profile and treatment of alcoholic women who had chronic pancreatitis. We did not follow the patients; thus, we were unable to evaluate the efficacy of pain reduction from various therapies over a longer period.

CONCLUSION

This study was conducted in 108 subjects who had been diagnosed with Acute pancreatitis and Chronic pancreatitis. This study revealed that almost all the participants had a history of alcohol use, whereas biliary was the major cause of Acute pancreatitis. To detect pancreatic enzyme abnormalities, serum amylase and serum lipase were evaluated. IV fluids like Dextrose Normal Saline and Ringers Lactate were most used in Acute pancreatitis and Chronic pancreatitis. Opioid Analgesics like Tramadol were the 1st line agent used to treat pain and the Fentanyl patch was used to treat severe pain in Chronic pancreatitis. In Antibiotics Inj. Magnex Forte was the most predominantly used drug in both Acute pancreatitis and Chronic Pancreatitis. Pancreatic enzymes, supplementation, and vitamins help to prevent nutritional deficiencies and promote healing. Hypertension, Diabetes mellitus, Cholelithiasis, and a history of pancreatitis are the pre-existing comorbidities in pancreatitis.

Acknowledgement

We thank the management, staff and faculty of Anurag University, Hyderabad for helping us in this research study and also the physicians of Yashoda Hospital, Secunderabad for allowing us do the data collection from the cases.

Competing Interest

None

Ethical Approval

The study was approved by the Institutional Review Board of Anurag university bearing the research proposal number: IRB-AU/2023-2024/06.

REFERENCES

1. Capurso G, Traini M, Piciucchi M, Signoretti M, Arcidiacono PG. (2019). Exocrine pancreatic insufficiency: prevalence, diagnosis, and management. Clin Exp Gastroenterol.21;12:129-139. doi: 10.2147/CEG.S168266. PMID: 30962702; PMCID: PMC6432881.
2. Forsmark CE. (2018). Diagnosis and management of exocrine pancreatic insufficiency. Curr Treat Options Gastroenterol. 16(3):306–315. doi: 10.1007/s11938-018-0186-y.
3. Zahit Bolaman Adnan Menderes University Medical Faculty, Division of Hematology, Tip Fakultesi, Ic Hastaliklari AD, Aydin, Turkey 2006.

4. Majumder S, Chari ST: Chronic pancreatitis. 2016;387(10031):1957–66. 10.1016/S0140-6736(16)00097-0 [DOI] [PubMed]
5. Majbar AA, Cusick E, Johnson P, Lynn RM, Hunt LP, Shield JP. Incidence and clinical associations of childhood acute pancreatitis. Pediatrics. 2016 Sep;138(3):e20161198. [DOI] [PubMed]
6. Acute Pancreatitis: Diagnosis and Treatment Peter Szatmary 1,2,3, Tassos Grammatikopoulos 4, Wenhao Cai 1,2,5, Wei Huang 5, Rajarshi Mukherjee 1,3,7, Chris Halloran 2,3, Georg Beyer 6, Robert Sutton 1,2,3
7. Zhang R, Deng L, Jin T, Zhu P, Shi N, Jiang K, et al. Hypertriglyceridaemia-associated acute pancreatitis: diagnosis and impact on severity. HPB (Oxford) 2019;21(9):1240–1249. doi: 10.1016/j.hpb.2019.01.015.
8. Pravallika Chadalavada 1, C Roberto Simons-Linares 2, Prabhleen Chahal 1 2020 Oct;20(7):1281-1286. doi: 10.1016/j.pan.2020.07.401. Epub 2020 Aug 14.
9. Underwood TW, Frye CB. (1993). Drug-induced pancreatitis. Clin Pharm.12(6):440-8. PMID: 8403815.
10. Aydinli M, Bayraktar Y. (2007). Budd-Chiari syndrome: etiology, pathogenesis and diagnosis. World J Gastroenterol. 13(19):2693-6. doi: 10.3748/wjg.v13.i19.2693. PMID: 17569137; PMCID: PMC4147117.
11. Ali DN, Ahmed A, Al-Qaisi AHJ. (2024). Inhibitory Effect of Newly Prepared Pyrazole Derivatives against Alpha-Amylase in Pancreatic Cancer Cell. Adv J Chem Sect A.7(3):248–59.
12. Prasath H, Azhakesan A, Manikandan G. (2025). Analytical Methods for Quantification of Gemcitabine in Pharmaceutical and Biological Samples: An Overview of Developments in the Last Decade. Asian J Green Chem.; 9:457–75.
13. Steinberg W, Tenner S. (1994). Acute pancreatitis. N Engl J Med; 330:1198 –1210
14. Banks PA. (1997). Practice guidelines in acute pancreatitis. Am J Gastroenterol ; 92:377 –386
15. Beger HG, Rau B, Mayer J, Pralle U. (1997). Natural course of acute pancreatitis. World J Surg ; 21:130-135
16. Dervenis C, Johnson CD, Bassi C, et al. (1999). Diagnosis, objective assessment of severity, and management of acute pancreatitis: Santorini consensus conference. Int J Pancreatol ; 25:195 –210
17. Kloppel G. (1994). Pathology of severe acute pancreatitis. In: Bradley EL III, ed. Acute pancreatitis: diagnosis and therapy. New York, NY: Raven, 35 –46
18. Rizo-Téllez SA, Sekheri M, Filep JG. (2023).C-reactive protein: a target for therapy to reduce inflammation. Front Immunol. 14:1237729. doi: 10.3389/fimmu.2023.1237729. PMID: 37564640; PMCID: PMC10410079.
19. Abdel Aziz AM, Lehman GA. (2007). Pancreatitis after endoscopic retrograde cholangio-pancreatography. World J Gastroenterol. 2007 May 21;13(19):2655-68. doi: 10.3748/wjg.v13.i19.2655. PMID: 17569133.
20. Lippi G, Valentino M, Cervellin G. (2012). Laboratory diagnosis of acute pancreatitis: in search of the Holy Grail. Crit Rev Clin Lab Sci.49(1):18-31. doi: 10.3109/10408363.2012.658354. PMID: 22339380.
21. Mark Feldman, Lawrence S. Friedman, and Lawrence J. Brandt, (2020). Sleisenger and Fordtran's Gastrointestinal and Liver Disease E-Book, 11th Edition.
22. Ahmed SA, Salau S, Khan A, Saeed M, Ul-Haq Z. (2022). Inhibitive Property of Catechin and Chlorogenic Acid against Human Pancreatic Lipase: Molecular Docking and Molecular Dynamics Simulation Investigations. Adv J Chem Sect A. 5(3):226–40.
23. Putra DA, Wibowo MD, Susilo DH, Fandinata SS. (2025). The impact of concurrent diabetes mellitus and hypertension on hospital length of stay in patients with phlegmon: A cross-sectional study at rsud dr. soetomo surabaya. J Med Pharm Chem Res.7(2):150–60.
24. Younis CY, Qurtas DS, Muhammed AH. (2023). Evaluation of Thyroid Biomarkers in Patients with Alopecia Areata. J Med Chem Sci. 6(10):2480–93.