DRUG-INDUCED ACUTE PANCREATITIS IN A PATIENT WITH ACUTE MYELOID LEUKEMIA COMPLICATED BY DISTAL RENAL TUBULAR ACIDOSIS: A CASE REPORT

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ABSTRACT

Drug-induced pancreatitis (DIP) is a rare but significant complication of chemotherapy. We report an 18-year-old male with high-risk Acute Myeloid Leukemia (AML) who developed acute pancreatitis (AP) after beginning chemotherapy, complicated by refractory hypokalemia and Distal Renal Tubular Acidosis (Type 1 RTA). This case emphasizes the combined risk of chemotherapy and metabolic instability in the development of DIP.

Keywords: Acute Myeloid Leukemia, Drug-Induced Pancreatitis, Distal Renal Tubular Acidosis, Cytarabine, Hypokalemia.

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INTRODUCTION

Acute pancreatitis (AP) involves inflammation, edema, and potential pancreatic necrosis caused by autodigestion. 1,2 Although biliary disease and alcohol are common triggers, DIP is a rare but recognized cause, accounting for 0.1–2% of cases. 3, 4 Chemotherapeutic agents have been frequently linked, although the mechanisms underlying these associations are often unclear. This report examines a case of AP caused by combination chemotherapy in an AML patient with concurrent RTA. 5, 6

CASE PRESENTATION

An 18-year-old male, HCV PCR–positive, presented with severe epigastric pain and hypotension (80/40 mmHg). Initial labs showed a hemoglobin of 3.2 g/dL and circulating blasts. Bone marrow examination confirmed AML with adverse-risk cytogenetics, including monosomy 7 (classified as adverse risk according to ELN criteria).

The patient exhibited severe electrolyte imbalances, including sodium 156 mmol/L and chloride 115 mmol/L. Notably, he had profound, refractory hypokalemia (initial 2.49 mmol/L), which persisted despite aggressive IV supplementation. Urinary evaluation showed failure to acidify urine (pH 6.0) despite hypokalemia, suggesting Distal Renal Tubular Acidosis (Type 1 RTA).

After starting induction chemotherapy with Cytarabine and Daunorubicin, the patient developed clinical and radiological signs of acute pancreatitis. Serum amylase (472 U/L) and lipase (477 U/L) were elevated, and CT imaging showed peripancreatic inflammatory changes

Table No 1: Summary of laboratory findings, highlighting elevated pancreatic enzymes and persistent hypokalemia

Parameters	Result	Normal Range
WBC	5.2 × 10^9/L	4–11 × 10^9/L
Hemoglobin	3.2 g/dL	13–17 g/dL (male)
Platelets	670,000/μL	150,000–450,000/μL
Sodium	156 mmol/L	135–145 mmol/L
Potassium (initial)	2.49 mmol/L	3.5–5.0 mmol/L
Chloride	115 mmol/L	98–106 mmol/L
Magnesium	2.52 mg/dL	1.7–2.2 mg/dL
Creatinine/Urea	Normal	Creatinine 0.6–1.2 mg/dL, Urea 7–20 mg/dL
Amylase	472 U/L	30–110 U/L
Lipase	477 U/L	23–300 U/L
ABG	Normal	pH 7.45–7.35 (no acidosis/alkalosis)
Urinary potassium	Elevated	Low in hypokalemia; <20 mmol/L expected
Chest X-ray	Normal	
CT Abdomen	Pancreatic changes	
Potassium Level	□ 2.7 □ 2.49 □ 2.9 □ 1.9 3.2 mmol/L	5.0–3.5 mmol/L

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(figure 1) consistent with acute pancreatitis. Gallstones, alcohol use, hypertriglyceridemia, and hypercalcemia were ruled out. Chemotherapy was temporarily paused, and the patient was treated conservatively with bowel rest, intravenous fluids, pain relief, and electrolyte correction, leading to gradual clinical and biochemical recovery.

DISCUSSION

DIP is a diagnosis of exclusion. In this case, the temporal relationship between Cytarabine and Daunorubicin suggests a likely contributory role. 7-10 Furthermore, severe hypokalemia from Type 1 RTA may have increased the pancreas's vulnerability to injury by affecting cellular enzyme secretion.

11-14 The RTA itself may result from leukemic organ infiltration or early drug-related toxicity. Unlike typical cases, this patient's early presentation indicates a complex interaction between leukemia-associated organ dysfunction and chemotherapy-induced injury. Management focused on stopping the suspected drugs and aggressively correcting electrolytes, leading to clinical improvement. 12, 13

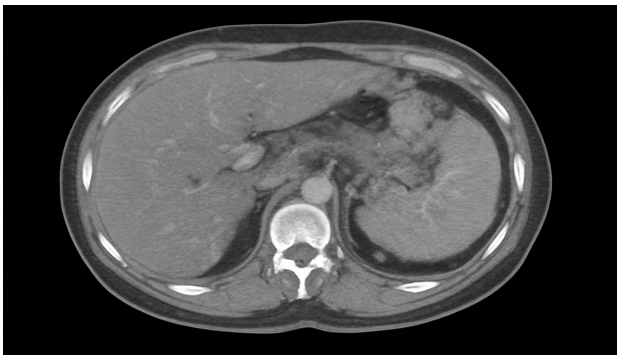

Fig- 1: contrast- enhanced CT abdomen showing peripancreatic inflammatory changes consistent with acute pancreatitis

CONCLUSION

Clinicians should consider monitoring pancreatic enzymes in patients with AML undergoing combination chemotherapy, especially when refractory electrolyte imbalances like RTA are present. Early detection and removal of the offending agent are crucial for recovery.

REFERENCES

1. Nitsche CJ, Jamieson N, Lerch MM, Mayerle JV. Drug-induced pancreatitis. Best Pract Res Clin Gastroenterol. 2010 Apr;24(2):143-55. doi: 10.1016/j.bpg.2010.02.002.
2. Johnson CD, Besselink MG, Carter R. Acute pancreatitis. BMJ. 2014 Aug 12;349:g4859. doi: 10.1136/bmj.g4859. PMID: 25116169.
3. McArthur KE. Review article: drug-induced pancreatitis. Aliment Pharmacol Ther. 1996 Feb;10(1):23-38. doi: 10.1111/j.1365-2036.1996.tb00174.x. PMID: 8871441.
4. Vinklerová I, Procházka M, Procházka V, Urbánek K. Incidence, severity, and etiology of drug-induced acute pancreatitis. Dig Dis Sci. 2010 Oct;55(10):2977-81. doi: 10.1007/s10620-010-1277-3. Epub 2010 May 25.
5. Yang QJ, Zheng J, Dang FT, Wan YM, Yang J. Acute pancreatitis induced by combination chemotherapy used for the treatment of acute myeloid leukemia: A case report. Medicine (Baltimore).
6. 2020 Aug 28;99(35):e21848. doi: 10.1097/MD.0000000000021848.
7. Balani AR, Grendell JH. Drug-induced pancreatitis : incidence, management and prevention. Drug Saf. 2008;31(10):823-37. doi: 10.2165/00002018-200831100-00002. PMID: 18759507.
8. 5. Singh T, Kumar R, Sharma A. Acute pancreatitis as an initial manifestation of pediatric acute myeloid leukemia. Thieme Med Case Rep. 2025;4(1):e15–e19.
9. Yang, Qiu-Jin MD; Zheng, Jie MD; Dang, Fu-Tao MD; Wan, Yue-Meng MD, PhD; Yang, Jing MD-. Acute pancreatitis induced by combination chemotherapy used for the treatment of acute myeloid leukemia: A case report. Medicine 99(35):p e21848, August 28, 2020. | DOI: 10.1097/MD.0000000000021848
10. Yang WX, An K, Liu GF, Zhou HY, Gao JC. Acute pancreatitis as initial presentation of acute myeloid leukemia-M2 subtype: A case report. World J Clin Cases 2023; 11(6): 1385-1392 [PMID: 36926134 DOI: 10.12998/wjcc.v11.i6.1385]
11. Bansal, Manju & Kapoor, Dhiraj & Dogra, V & Gargi, Gaveshna & Harita, & Kumari, Kavita & Singh, Gopal. (2025). Acute pancreatitis as presenting complication of Acute myeloid leukemia (A rare case and Literature Review). 2. 372-374.
12. Sumitani R, Hori T, Murai J, Kawata S, Oura M, Sogabe K, Takahashi M, Harada T, Fujii S, Miki H, Kagawa K, Abe M, Nakamura S. Acute Myeloid Leukemia Developing with Acute Pancreatitis Mimicking Autoimmune Pancreatitis. Intern Med. 2021 Jun 1;60(11):1753-1757. doi: 10.2169/internalmedicine.4916-20. Epub 2021 Jan 15
13. Jones MR, Hall OM, Kaye AM, Kaye AD. Drug-induced acute pancreatitis: a review. Ochsner J. 2015 Spring;15(1):45-51. PMID: 25829880; PMCID: PMC4365846.
14. Yokoe M, Takada T, Mayumi T, et al. Japanese guidelines for the management of acute pancreatitis: Japanese Guidelines 2015. J Hepatobiliary Pancreat Sci 22: 405-432, 2015.