A CASE OF ASPIRIN- INDUCED GASTRIC ULCER PRESENTING AS UPPER GASTROINTESTINAL BLEEDING IN AN ELDERLY PATIENT WITH CORONARY ARTERY DISEASE

ABITHA M[1], AGAYASUNDARI A[1] ,ASHMITHA N A[1]

DHIVYA B[1], ABINAYA S[1] , Dr VAISHNAVI DEVI C [1]

[1]Department of Pharmacy Practice, Swamy Vivekanandha College of Pharmacy.

Corresponding Author: Dr C VAISHNAVI DEVI [1]

ABSTRACT:

Upper gastrointestinal bleeding (UGIB) is a common medical emergency associated with significant morbidity and mortality, particularly in elderly patients with multiple comorbidities [1,2]. We report a case of a 78-year-old male with a history of diabetes mellitus, hypertension, and coronary artery disease who presented with melaena, vomiting, and fatigue. Laboratory investigations revealed severe anemia, elevated blood urea, and uncontrolled glycemic status. Stool occult blood was positive. Upper gastrointestinal endoscopy demonstrated a benign esophageal polyp, lax gastroesophageal junction, and drug-induced gastric ulcer. Chronic use of aspirin as part of antiplatelet therapy is a well-established risk factor for gastrointestinal mucosal injury and bleeding [3]. The patient was managed with proton pump inhibitors, antifibrinolytics, antibiotics, and supportive care, along with discontinuation of the offending drug. The patient showed clinical improvement. This case highlights the importance of early diagnosis, appropriate management, and careful monitoring of patients on long-term antiplatelet therapy [2,4].

INTRODUCTION:

Upper gastrointestinal bleeding (UGIB) represents a frequent medical emergency, with an annual incidence of 80-150 per 100,000 population and notable morbidity and mortality, especially among elderly patients with comorbidities. Chronic antiplatelet therapy, such as aspirin used for coronary artery disease, heightens the risk of drug-induced gastric ulcers and mucosal injury through mechanisms like cyclooxygenase inhibition and reduced prostaglandin synthesis.

UGIB often manifests as melena, hematemesis, or fatigue, with mortality rates ranging from 5-14%, rising steeply in those over 75 due to comorbidities and polypharmacy. "Upper

gastrointestinal bleeding (UGIB) is a common problem with an annual incidence of approximately 80 to 150 per 100,000 population," highlighting its public health burden.

CASE DESCRIPTION:

A 78 years old male patient was admitted to the CARDIOLOGY department with complaints of MELAENA,VOMITTING,LOOSE STOOLS, FATIGUE AND CHEST PAIN,TIREDNESS SINCE x 3 days. On physical examination the patient was conscious, oriented, and febrile. His past medical history was found to be a known case of DM/SHTN/ CAD and his past surgery S/P PTCA TO LCX (2017) .his family history was found to be Nil. past medication history was found to ROSVA GOLD 10 . On the examination of vital sign ,The blood pressure was slightly increased for the 1,5,6 days (150/80,140/70,140/70mmHg) and decreased for the next 4 days (110/60,120/70mmHg), The pulse rate was found to be normal for all days,The SPO2 was found to be normal for all days, temperature was found to be normal for all days was given in the table 1.ON laboratory investigation the RBC was decreased for the first day (2.80million/cumm),haemoglobin level was slighty decreased for the first day (8.90gm/dl) and gradually decreased for all the days (,4.8gm/dl,6.80gm/dl, 7.47gm/dl,7.1gm/dl,7.0gm/dl),Paced cell volume was decreased for the first day (25.3%),mean corpuscular hemoglobin concentration was increased for the first day (35.3gm/dl),basophils was decreased for the first day (0.35%) was given in table 2.on biochemistry the blood urea was increased for the days of examination(107mg/dl ,99 mg/dl,85mg/dl),creatinine was increased for the first day(1.9mg/dl) was normal for all days(1.2mg/dl,1.2mg/dl),potassium(5.0mmol/l ,4.3mmol/l ,4.1mmol/l)CPK-MB (09u/l),blood glucose fasting was increased for the day of examination (214mg/dl),blood glucose post prandial was increased for the day of examination (354mg/dl),HbA1C was increased for the day of examination(7.9%).was given in the table 3. On clinical pathology was shown that OCCULT BLOOD STOOL was positive table 4 , ENDOSCOPY REPORT: Patient was admitted with above mentioned complaints and underwent UGI scopy on day 4. Which shows ? BENIGN OESOPHAGEAL POLYP.LAX O-G JUNCTION. DRUG INDUCED GASTRIC ULCER.

The patient was treated withTAB.ROSVASTATIN10mgPO,INJ.PANTOPRAZOLE 40mg IV,INJ.ONDANSTRON 4mg IV,TAB.AZELINDIPINE 16mg PO,TAB.SITAGLIPTIN PHOSPHATE, METFORMIN 50/1/500 PO,TAB. METHYLCOBALAMIN, CALCIUM

CARBONATE, CALCITROL,FOLIC ACID , PYRIDOXINE CS PO,TAB.ALPRAZOLAM 0.5mg PO,TAB. LIPOSOMAL FERRIC DXROPHOSPHATE,VIT C,FOLIC ACID,ZINC PO,INJ. TRENAXAMIC ACID 500mg IV, INJ. PIPERAILLIN,TAZOBACTAM 4.5g IV,SYP.GELUSIL P/O ,TAB. TRENAXAMIC ACID 500mg P/O. WAS GIVEN IN THE TABLE 5. In this case the patient was taking rosva gold in past medication history for CAD long term of ASPIRIN CAUSES THE UGI EROSION so physician stopped the drug.the patient treated with the ROSVAS medication on his admission.The patient was treated with INJ.SOMEPRESE 40mg,INJ ONDANSETRON 8 mg ,SYP.MUCAIN GEL 10ml,INJ. ISOPAR 1gm ,INJ. TRENAXAMIC ACID 500mg,INJ LASIX 10mg,TAB WELMOL 1gm,IV RL+MVI,INJ.RL+NS,INJ. PIPERAILLIN ,TAZOBACTAM TEST DOSE 0.5ml was given in the table 6.the patient was discharged with improved patient compliance and discharged with the medication TAB. ROSVASTATIN10mg,TAB.METHYPREDNISONE 16mg,TAB.SITAGLIPIN+METFOMIN+GLIMEPIRIDE50/1/500mg,CAP.RABEPRAZOLE+ ITOPRIDE,TAB.ALPRAZOLAM 0.5mg,TAB.FERROUS ASCORBATE+FOLIC ACIDE,TAB.TRANEXAMIC ACIDE 500mg,TAB. AMOXICILLIN +CLAVULANIC ACIDE 625mg,SYP. GELUSIL was given in the table 7.

TABLE 1 - VITAL SIGN:

PARAMETER	DAY 1	DAY 2	DAY 3	DAY 4	DAY 5	DAY 6	DAY 7	DAY 8	NORMAL VALUE
BLOOD PRESSURE	150/80 mmHg	110/60 mmHg	120/70 mmHg	120/60 mmHg	140/70 mmHg	140/70 mmHg	110/60 mmHg	110/60 mmHg	120/80mmHg
PULSE RATE	90 beats per minute	98 beats per minute	86 beats per minute	80 beats per minute	94 beats per minute	76 beats per minute	78 beats per minute	80 beats per minute	60 to 100 beats per minute
SPO2	98%	99%	99%	97%	97%	98%	98%	98%	95-100 %

TEMPERATURE	97F	98F	98.2F	98.2F	100.6F	98F	97.2F	97.9F	97.7F-99.5F

TABLE 2 - LABORATORY INVESTIGATION: HAEMATOLOGY

S.NO	PARAMETER	OBSERVED VALUE DAY 1	OBSERVED VALUE DAY2	OBSERVED VALUE DAY3	OBSERVED VALUE DAY4	OBSERVED VALUE DAY5	OBSERVED VALUE DAY6	NORMAL VALUE
1	RED BLOOD CELLS	2.80 million/cu mm.	-	-	-	-	-	4.7-6.2million/ cu mm.
2	HAEMOGLOBIN	8.90gm/dl	4.8gm/dl	6.80gm/dl	7.47gm/dl	7.1gm/dl	7.0gm/dl	13-17gm/dl.
3	PACKED CELL VOLUME	25.3%	-	-	-	-	-	42-52%
4	MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION	35.3gm/dl	-	-	-	-	-	31-35gm/dl
5	BASOPHILS	0.35%	-	-	-	-	-	1.0-2%

TABLE 3 -BIOCHEMISTRY (RENAL FUNCTION TEST):

S.NO	PARAMETER	OBSEVERED VALUE 1	OBSEVERED VALUE 2	OBSEVERED VALUE 3	NORMAL VALUE
1	BLOOD UREA	107mg/dl	99 mg/dl	85mg/dl	19-43mg/dl
	CREATININE	1.9mg/dl	1.2mg/dl	1.2mg/dl	0.66-1.25mg/dl

3	POTASSIUM	5.0mmol/l	4.3mmol/l	4.1mmol/l	3.5-5.1mmol/l
4	CPK-MB	09U/L	-	-	UPTO 24
5	BLOOD GLUCOSE FASTING	214mg/dl	-	-	70-140mg/dl
6	BLOOD GLUCOSE POST PRANDAIL	354mg/dl	-	-	70-140mg/dl
7	HbA1C	7.9%	-	-	4-6.5%

TABLE 4- CLINICAL PATHOLOGY

S.NO	PARAMETER	OBSERVED VALUE	NORMAL VALUE
1	OCCULT BLOOD STOOL	POSITIVE	NEGATIVE

ENDOSCOPY REPORT:

Patient was admitted with above mentioned complaints and underwent UGI scopy on day 4. Which shows ? BENIGN OESOPHAGEAL POLYP.LAX O-G JUNCTION. DRUG INDUCED GASTRIC ULCER.

TABLE -5 THERAPUETIC CHART

S.NO	DRUG NAME	DOSE	ROUTE	FREQUENCY

1	TAB. ROSVASTATIN	10mg	PO	0-0-1
2	INJ.PANTOPRAZOLE	40mg	IV	BD
3	INJ.ONDANSTRON	4mg	IV	BD
4	TAB.AZELINDIPINE	16mg	PO	PO
5	TAB.SITAGLIPTIN PHOSPHATE, METFORMIN	50/1/500	PO	1-0-½
6	TAB. METHYLCOBALAMIN, CALCIUM CARBONATE, CALCITROL,FOLIC ACID , PYRIDOXINE	CS	PO	1-0-0
7	TAB.ALPRAZOLAM	0.5mg	PO	0-0-1
8	TAB. LIPOSOMAL FERRIC DXROPHOSPHATE,VIT C,FOLIC ACID,ZINC		PO	1-0-0
9	INJ. TRENAXAMIC ACID	500mg	IV	1-0-1
10	INJ. PIPERAILLIN,TAZOBACTAM	4.5g	IV	1-1-1
11	SYP.GELUSIL		P/O	10ml-10ml-10ml
12	TAB. TRENAXAMIC ACID	500mg	P/O	1-0-1

TABLE- 6 STAT CHART

DATE	DRUG NAME	INDICATION	DOSE	ROUTE

DAY1	INJ.SOMEPRESE	STAT	40mg	
DAY1	INJ ONDANSETRON	STAT	8 mg	IV
DAY1	SYP.MUCAIN GEL	STAT	10ml	IV
DAY1,4,5	INJ. ISOPAR	STAT	1gm	IV
DAY2	INJ. TRENAXAMIC ACID	STAT	500mg	IV
DAY2	INJ LASIX	STAT	10mg	IV
DAY 2	TAB WELMOL	-	1gm	P/O
DAY1	IV RL+MVI	STAT	-	IV
DAY4	INJ.RL+NS	STAT	-	IV
DAY5	INJ. PIPERAILLIN ,TAZOBACTAM	TEST DOSE	0.5ml	IV

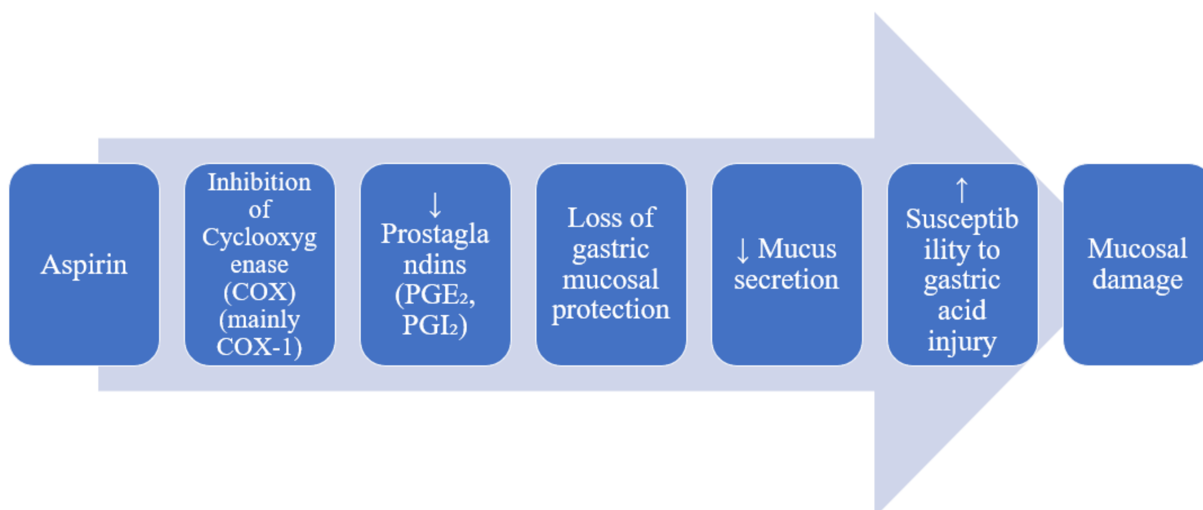
TABLE- 7 DISCHARGE CHART:

S.NO	**DRUG NAME**	**DOSAGE**	**ROUTE**	**FREQUENCY**	**DURATION**
1	TAB. ROSVASTATIN	10mg	PO	0-0-1	7 DAYS

2	TAB.METHYPREDNISONE	16mg	PO	0-0-1	7 DAYS
3	TAB.SITAGLIPIN+ METFOMIN+GLIMEPIRIDE	50/1/500mg	PO	½-0-½	7 DAYS
4	CAP.RABEPRAZOLE+ITOPRIDE		PO	0-0-1	7 DAYS
5	TAB.ALPRAZOLAM	0.5mg	PO	0-0-1	7 DAYS
6	TAB.FERROUS ASCORBATE+FOLIC ACIDE		PO	1-0-0	7 DAYS
7	TAB.TRANEXAMIC ACIDE	500mg	PO	1-0-1	3 DAYS
8	TAB. AMOXICILLIN +CLAVULANIC ACIDE	625mg	PO	1-0-1	3DAYS
9	SYP. GELUSIL		PO	10ml-10ml-10ml	7 DAYS

DISCUSSION:

A 78-year-old male patient was admitted with complaints of Malena, vomiting, and loose stools. The past medical history was CAD/Good LV dysfunction/DM/HTN. The patient has a GASTROINTESTINAL ULCER due to long-term use of ASPIRIN.



Direct injury and indirect injury are the two main components of the mechanism of aspirin-induced gastrointestinal damage. The irreversible inactivation of cyclooxygenase, which prevents arachidonic acid from being converted to prostaglandin H2 in platelets and consequently affects the generation of prostaglandins PGE2 and PGI2, is the indirect mechanism by which aspirin damages the gastrointestinal mucosa. Reduced PGE2 secretion lowers gastrointestinal blood flow as well as gastric mucus and HCO3 release. Reduced PGI2 output also reduces blood supply to the gastrointestinal tract. The gastrointestinal mucosa is extremely vulnerable to ischemia and hypoxia due to the reduced blood flow it receives. It may harm the gastrointestinal mucosa's epithelial cells, impair the function of the mucosal barrier, increase permeability, and increase the endotoxin.

CONCLUSION:

This case illustrates that long-term aspirin therapy can significantly increase the risk of upper gastrointestinal bleeding due to drug-induced gastric ulceration [3]. Elderly patients with comorbidities such as diabetes, hypertension, and coronary artery disease are particularly vulnerable [1].

Early recognition of symptoms and timely endoscopic evaluation are critical in reducing morbidity and mortality [4]. Standard management includes hemodynamic stabilization, proton pump inhibitor therapy, and endoscopic intervention when required [4,5].

Preventive strategies, including risk assessment and the use of gastroprotective agents, are essential in patients receiving long-term antiplatelet therapy [2]. Clinicians should carefully balance the cardiovascular benefits of aspirin against its gastrointestinal risks.

REFERENCES:

1. Wasserman RD, Abel W, Monkemuller K, Yeaton P, Kesar V, Kesar V. Non-variceal Upper Gastrointestinal Bleeding and Its Endoscopic Management. Turkish Journal of Gastroenterology [Internet]. 2024 May 28 [cited 2024 Nov 30]; Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC11363156/
2. Alali A, Barkun AN. An update on the management of non-variceal upper gastrointestinal bleeding. Gastroenterology Report. 2022 Dec 30;11.
3. Li DK, Laine L, Shung DL. Trends in Upper Gastrointestinal Bleeding in Patients on Primary Prevention Aspirin: A Nationwide Emergency Department Sample Analysis, 2016-2020. The American Journal of Medicine [Internet]. 2023 Sep 9;136(12):1179-1186.e1. Available from: https://www.sciencedirect.com/science/article/abs/pii/S0002934323005429
4. Bergin A, Christmas M, Tan WL, Ding Y, Lin L, Khaing MM, et al. Optimising admission to scope time for acute GI bleeding (UGIB): retrospective comparative analysis of the timing of endoscopy for UGIB in a Queensland quaternary centre following international recommendations for best practice. Clinical Medicine. 2023 Nov;23(6):96–7.
5. Maity R, Dhali A, Biswas J. Importance of risk assessment, endoscopic hemostasis, and recent advancements in the management of acute non-variceal upper gastrointestinal bleeding. World Journal of Clinical Cases [Internet]. 2024 Aug 26;12(24):5462–7. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC11269988/