Acute myocardial infarction complicating acute ulcerative colitis: A clinical conundrum

Krishna Nemalidinne[1], Abinay Siva Kumar Reddy Vanteru Vanteru[1], Amit Goel[1], Abhigan Shrestha[2], and Vikash Jaiswal[3]

[1]Blackpool Health Authority
[2]M Abdur Rahim Medical Medical College Hospital
[3]AMA School of Medicine

May 19, 2023

Introduction

IBD is a chronic systemic inflammatory disease that predominantly involves the gastrointestinal tract. At the same time, it exerts numerous cardiovascular manifestations, like atherosclerotic cardiovascular disease and thromboembolic events, due to a hypercoagulable state. Although the disease mechanism is unclear, factors responsible, like the disruption of the normal coagulation cascade, hyperhomocysteinemia, abnormalities in platelet-endothelial cell interactions, and increased fibrinolysis, are implicated.

Case

A 46-year-old female, who is a known case of IBD-UC, presented with intermittent chest pain for more than 24 hours. Her history was suggestive of a recent relapse of IBD-UC, for which she was on mesalamine and steroids. Electrocardiogram (EKG) showed ST elevations in inferior leads (II, III, aVF). She was loaded with dual antiplatelet and high-dose statins. 2D Echocardiogram revealed regional wall motion abnormality with an ejection fraction of 35%. Coronary angiography revealed thrombus in all three main arteries with >90% occlusion in LAD (left anterior descending artery), LCX (left circumflex artery), and near-complete occlusion of RCA (right coronary artery), and ramus. (Figure 1). Considering her delayed presentation, prothrombotic state, and keeping in mind the immediate complications of stent placement, only thrombosuction was done, achieving grade 3 TIMI flow. Intravenous antiplatelet (abciximab) infusion was administered for 48 hours. Laboratory investigations revealed anemia (Hb-11gmdl), leucocytosis (11,000 per microliter) with neutrophilic predominance, and thrombocytosis (9,00,000 per microliter). Her troponin was 6.5(reference range 0-0.02). RA factor and hs-CRP were elevated, suggesting an active inflammatory state. The Thrombophilia profile was negative (protein C, protein S antithrombin III, antiphospholipid antibodies). She was negative for ANA, vasculitis profile, AMA, Anti smooth muscle, and anti-LKM antibodies. Complement levels were normal. Sigmoid ulcers with no active bleeding were seen on colonoscopy. Bone marrow biopsy reported hypercellular marrow with no evidence of any myeloproliferative neoplasm. During her hospital stay, she had an episode of atrial fibrillation, for which she was put on amiodarone and NOACs (apixaban) as there was no evidence of bleeding from sigmoid ulcers in colonoscopy. She was discharged on single antiplatelet and NOACs and was advised for further follow-up in OPD (outpatient department). She did not report any chest pain for the next two weeks. However, a repeat coronary angiography six weeks later showed surprising results with a near-complete resolution of thrombus from RCA and complete resolution from LCX and LAD(Figure 2). A repeat 2D Echocardiogram revealed a significant increase in ejection fraction from 35% to 45-50%.

DISCUSSION

Ulcerative colitis is often associated with extra-intestinal manifestations affecting the liver, skin, kidneys, eyes, and joints. Several processes chronically stimulated in IBD patients have been involved in the pathophysiology of ASCVD. These include local and systemic inflammation, gut microbiome irregularities, endothelial dysfunction, thrombosis, lipid dysfunction, and the negative effects of IBD therapies, particularly corticosteroids.

Previous literature has suggested possible causative links between IBD and coronary artery disease. [1,2]

A Finish epidemiological study first demonstrated an association between IBD and IHD. Similarly, a meta-analysis of multiple cohort studies also reported an increased incidence of ACS in IBD.(3)

A recent retrospective cross-sectional study showed that the incidence of thromboembolic events in IBD patients was increasing over the past decade, and more arterial thrombotic events were observed compared with venous thrombotic events.[4], suggesting an increased risk of CAD.

Our patient had a flare of IBD-UC a month back, followed by ACS. This can be explained by a systematic review which stated that the risk of ACS increases significantly with acute active flares and hospitalization, in addition to prolonged periods of active disease. On the other hand, IBD patients in remission present with a lower risk for ACS. (5)A cross-sectional observational study on the Egyptian population also showed that the risk of ASCVD is higher in IBD patients, particularly during active disease, with increased carotid intimal thickness and wall stiffness. (6) The same was depicted in a recent Danish nationwide cohort study showing that IBD flares are associated with an increased risk of MI, stroke, and even cardiovascular death. (7)

It was found that traditional CVD risk factors were less prevalent in IBD patients than in the general ACS population in a population-based cohort study(8). Our patient was in a procoagulant state with elevated inflammatory markers. Despite the absence of traditional ASCVD risk factors, our patient also presented in ACS, presumably from a non-atherosclerotic thrombus as confirmed by Coronary angiography. Case reports of Hyon He and Yong Zhang also support the same. (9,10)

Corticosteroids may also have some prothrombotic effects. It remains controversial whether corticosteroids may add risk to coronary artery disease in IBD patients. Among systemic corticosteroid users, the risk for AMI increased to 5-fold compared with the controls and 2.5-fold for Heart failure (8). Our patient had a relapse treated with corticosteroids a month back, followed by an ACS presentation indicating that steroids may have some prothrombotic effects. But, further studies are needed to confirm this association.

The risk-benefit ratio must be individualized for every patient before stent placement. Stent thrombosis is not uncommon after angioplasty, and its risk is increased in IBD patients owing to its hypercoagulable state. Our patient with elevated inflammatory markers had a greater risk of stent thrombosis; hence, a conservative approach was made.

Our patient with delayed presentation of MI, triple vessel blockade, and active inflammatory state (suggested by elevated inflammatory markers – thrombocytopenia, elevated hs-CRP, and RA factor) was in a procoagulant state. And if angioplasty was planned, subsequent long-term dual antiplatelet drugs pose an increased risk of bleeding with a high risk of stent thrombosis. Our decision to manage patients with antiplatelet infusions and avoid stent placement benefited the patient.

So, while managing such cases, a conservative approach can be made with a close follow-up of the patient.

CONCLUSION

A multidisciplinary team-based approach should be considered for managing IBD, i.e., for the remission of IBD disease-based activity and aggressive reduction of cardiovascular risk factors. Although the immediate treatment for MI is angiography +/- angioplasty, a conservative approach can be tried in IBD patients to avoid future complications like bleeding, stent thrombosis, etc.

Consent of patient: Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

Conflict of interest and funding: Nil

Ethical approval

N/A.

Data Availability Statement:

Data sharing does not apply to this article as no datasets were generated or analyzed during the current study

Registration of research studies

Not applicable.

Consent

Written*informed consent* was obtained from the parent's patient for publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

REFERENCES

1. Nuutinen, H.; Reunanen, M.; and Farrkkila, M. Association of ulcerative colitis and ischemic heart disease. Gastroenterology, vol. 108, p. A886, 1995.
2. Ha, C.; Magowan, S.; Accortt, N.A.; et al. Risk of arterial thrombotic events in in*fl* ammatory bowel disease. Am J Gastroenterol 2009;104:1445– 51.
3. Feng, W.; Chen, G.; Cai, D.; et al. Inflammatory bowel disease and risk of ischemic heart disease: An updated meta-analysis of cohort studies. J Am Heart Assoc. 2017;6:e005892. DOI: 10.1161/JAHA.117.005892.
4. Kuy, S.; Dua, A.; Chappidi, R.; et al. The increasing incidence of thromboembolic events among hospitalized patients with in*fl* ammatory bowel disease. Vascular 2015;23:260– 4.
5. Jaaouani, A.; Ismaiel, A.; Popa, S.-L.; Dumitrascu, D.L. Acute Coronary Syndromes and Inflammatory Bowel Disease: The Gut–Heart Connection. J. Clin. Med. 2021, 10, 4710. https://doi.org/10.3390/jcm10204710.
6. Ali, W.A.; Abdel Aal, A.M.; Abokresha, M.W.; et al. Predictors of Subclinical Atherosclerotic Cardiovascular Disease in Inflammatory Bowel Disease Patients. The Egyptian Journal of Hospital Medicine (April 2022) Vol. 87, Page 1666-1673.
7. Kristensen, S.L.; Ahlehoff, O.; Lindhardsen, J.; et al. Disease activity in in*fl* ammatory bowel disease is associated with increased risk of myocardial infarction, stroke and cardiovascular death — a Danish nationwide cohort study. PLoS ONE 2013;8:e56944.
8. Aniwan, S.; Pardi, D.S.; Tremaine, W.J.; Loftus, E.V. Increased Risk of Acute Myocardial Infarction and Heart Failure in Patients with Inflammatory Bowel Diseases. Clin. Gastroenterol. Hepatol. 2018, 16, 1607–1615.e1.
9. Garza, H.H.; Truong, T.; Patel, R.; et al. Arterial Embolism causing Myocardial Infarction in a patient with Ulcerative colitis. JACC March 17, 2015, Volume 65, Issue 10S.
10. Zhang, Y.; Hao, X.; Zheng, X.; et al. Acute myocardial infarction in a young woman with ulcerative colitis. Medicine (2017) 96:47/.

Figure legends: Figure 1: Coronary angiography shows thrombus in all three main arteries with >90% occlusion in LAD (left anterior descending artery), LCX (left circumflex artery), and near-complete occlusion

of RCA (right coronary artery), and ramus. Figure 2: Repeat coronary angiography six weeks later showed complete resolution from RCA, LCX and LAD

Hosted file

`fig1.docx` available at https://authorea.com/users/620126/articles/644320-acute-myocardial-infarction-complicating-acute-ulcerative-colitis-a-clinical-conundrum

Hosted file

`fig2.docx` available at https://authorea.com/users/620126/articles/644320-acute-myocardial-infarction-complicating-acute-ulcerative-colitis-a-clinical-conundrum