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CASE REPORT

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Acute myocardial infarction complicating acute ulcerative colitis: A clinical conundrum

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Key Clinical Message

Inflammatory bowel disease (IBD) is an immune-mediated multisystem inflammatory disease that primarily affects the gastrointestinal tract, but it also has various extraintestinal manifestations like cardiovascular, dermatological, musculoskeletal, or hepatobiliary tract involvement. Herein, we describe a case of a 46-year-old female, who presented with acute coronary syndrome on the background of acute relapse of ulcerative colitis (UC), requiring a pragmatic clinical approach due to a labile balance between hemorrhagic and ischemic risk.

KEYWORDS

hypercoagulable, myocardial infarction, prothrombotic state, ulcerative colitis

1 | 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 (ASCVD) and thromboembolic events, due to a hypercoagulable state. The occurrence of acute coronary syndrome (ACS) can be due to atherosclerosis or non-atherosclerotic in causation. 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.[1] There is always a delicate balance between hemorrhagic and ischemic risk in the management of patients with coexisting active IBD and acute coronary syndrome.

2 | CASE

A 46-year-old female, who had a relapse of IBD-UC a month back, presented with intermittent chest pain and sweating for more than 24 h duration. Her electrocardiogram (EKG) showed ST elevations in inferior leads (II, III, aVF). 2D echocardiogram did show regional wall motion abnormality in mid basal inferior and posterior wall, with an ejection fraction of 35% and mild mitral regurgitation. Due to intermittent chest pain, electrocardiogram and echocardiogram findings suggestive of inferior wall myocardial infarction, she underwent coronary angiography which demonstrated the presence of thrombus distally in all three main coronary arteries including ramus intermedius (Figure 1). The presence of thrombi in all three main coronary arteries, had led us to consider for non-atheromatous cause of her presentation, likely systemic in

origin. On probing her for the detail history of IBD-UC, it was discovered that she had recent relapse of disease 2 months back, wherein she had hemorrhagic manifestation of gastrointestinal tract for which hospitalization was required for a week. Thereafter, she was discharged home on oral steroids and mesalamine. Laboratory investigations revealed mild anemia (Hb-10.7 g/dL), leukocytosis (11,000 per microliter) with neutrophilic predominance, and thrombocytosis (9,00,000 per microliter). Rheumatoid factor (RA factor) and high-sensitive C-reactive protein (hs-CRP) were elevated, the presence of which along with thrombocytosis suggest an active inflammatory state. The thrombophilia profile was negative (protein C, protein S antithrombin III, antiphospholipid antibodies). It became a precarious situation, where we were dealing on hand with coronary ischemia and on other hand there was a risk of gastrointestinal hemorrhage secondary to UC, which could be compounded with the dual antiplatelet therapy, if patient would have undergone percutaneous coronary intervention especially with stent deployment. Hence, it was decided to keep her on optimal medical therapy for acute coronary syndrome as benefits of PCI of infarct-related artery after delay in presentation is doubtful, and in the event after PCI, we would be committed to give her dual antiplatelets which will predispose her for bleeding risk. Also, in scenario where there is hemorrhagic complication post PCI, stopping antiplatelets could have adverse cardiovascular outcome, like stent thrombosis. Meanwhile, she had an episode of atrial fibrillation with fast ventricular rate which was managed medically with amiodarone and injectable anticoagulation (enoxaparin) and sinus rhythm was restored. She was also subjected to colonoscopy, the finding of which showed presence of sigmoid ulcer but with no active bleeding. She remained angina free during further stay in hospital. At the time of discharge, she was given apixaban, clopidogrel, beta blocker, angiotensin converting enzyme inhibitor and mineralocorticoid receptor antagonist along with oral steroids and mesalamine. A repeat coronary angiography 6 weeks later showed surprising results with a complete resolution of thrombus from all three major coronary arteries (Figure 2). A repeat 2D echocardiogram demonstrated a significant improvement in ejection fraction from 35% to 50%.

3 | DISCUSSION

Ulcerative colitis is a chronic relapsing and remitting inflammatory disease of colonic mucosa that is often associated with extraintestinal manifestations involving cardiovascular system, renal, skin, eyes, and musculoskeletal system. The frequently encountered cardiovascular manifestations are myocarditis, pericarditis, atherosclerotic cardiovascular disease (ASCVD), and thromboembolic events. Various mechanism implicated in pathogenesis of ASCVD in patients with IBD include local and systemic inflammation, gut microbiome irregularities, endothelial dysfunction, thrombosis, lipid dysfunction, and the negative effects of IBD therapies, particularly corticosteroids.[2]

There is scant literature available on the possible association between ASCVD and IBD. However, few epidemiological studies and case reports have suggested the causative link between them. The first such evidence came from Finnish epidemiological study which found a significantly increased prevalence of coronary artery disease in patients with IBD.[3] Similarly, a subgroup analysis of a retrospective study observed an increased association of myocardial infarction in women over 40 years age suffering from IBD.[4] A meta-analysis of multiple cohort studies had also found an increased risk of ischemic heart disease in patients with IBD.[5]

Our patient had a flare of IBD-UC a month back, following which she suffered an event of ACS. This finding is supported in the literature by the various authors. A systematic review by Jaaouani et al, had reported that the risk of ACS increases significantly with acute active flares, in addition to prolonged periods of active disease.[6] On the other hand, the risk of ACS is lower in patients who are in

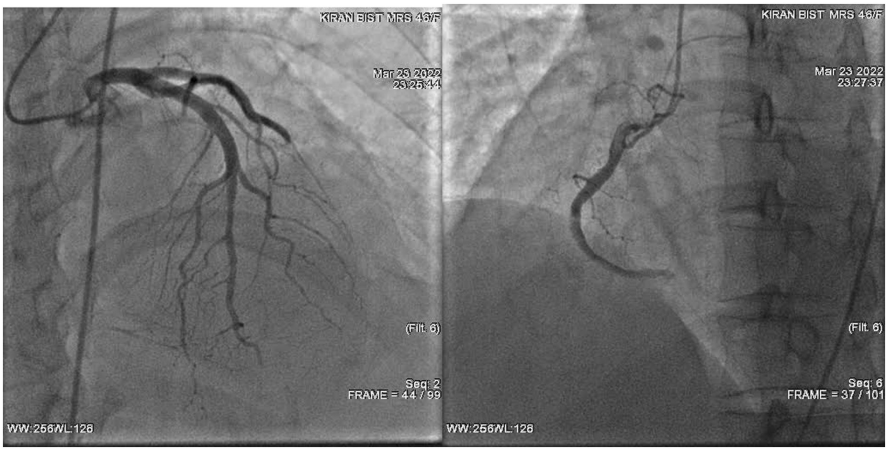

FIGURE 1 Coronary angiography shows thrombus in all three coronary arteries including ramus intermedius.

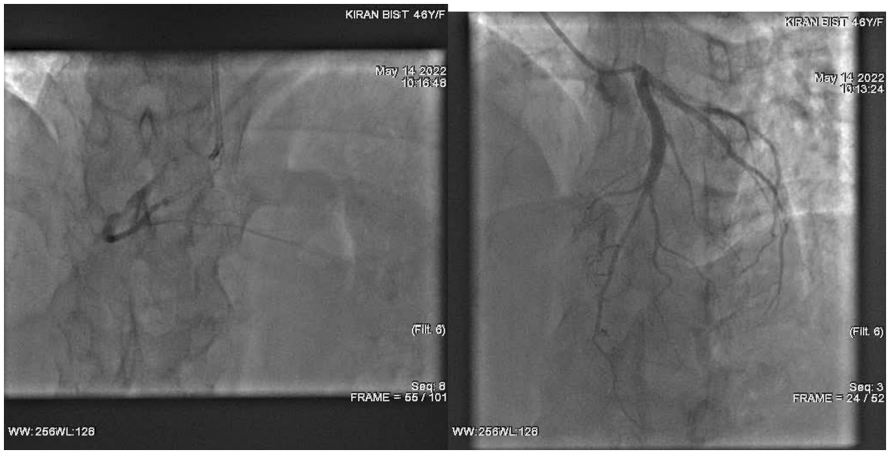

FIGURE 2 Repeat coronary angiography 6 weeks later showing complete resolution of thrombus from LAD, LCx, RCA, and ramus intermedius.

remission phase of the disease.[6] 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.[7] The similar observation was also noticed by Danish nationwide cohort study, where IBD flares were associated with an increased risk of MI, stroke, and even cardiovascular death.[8]

In a population-based cohort study, it is also observed that the traditional CVD risk factors are less prevalent in IBD patients than in general ACS subset population.[9] Our patient was also devoid of any conventional risk factors of CVD and elevated inflammatory markers were noticed which again indicate active systemic disease. Presence of thrombi in multiple coronary beds confirmed by coronary angiogram in our patient, which likely to be non-atherosclerotic in origin, indicates the existence of active systemic procoagulant state.

Drugs like corticosteroids may also have prothrombotic effects. It remains controversial whether corticosteroids may add risk to coronary artery disease in IBD patients. Among systemic corticosteroid users, the risk for ACS increased to fivefold compared with the controls and 2.5-fold for heart failure.[9] Our patient had a relapse of IBD-UC which was treated with injectable corticosteroids a month back and was later continued on oral steroids, following which she suffered an ACS, which again suggests that steroids may have some prothrombotic effects which further accelerates the systemic procoagulant state of IBD.

There exact pathogenetic mechanisms proposed for the development of ACS in IBD population remain unclear, but some of the proposed factors responsible are, like the disruption of the normal coagulation cascade, hyperhomocysteinemia, abnormalities in platelet-endothelial cell interactions, and increased fibrinolysis.[1] In our patient, we considered to follow conservative approach, after weighting the risk and benefits of it versus PCI, which were in the favor of medical management. Stent thrombosis is not uncommon after PCI, especially in the event when there is need to stop antiplatelets due to bleeding, and furthermore, the risk of stent thrombosis is increased in IBD patients owing to its hypercoagulable state. She was offered a non vitamin K antagonist oral anticoagulant (NOAC), which was apixaban along with clopidogrel for the purpose of dual pathways inhibition (DPI), in a such intricate clinical scenario, where multiple etiologies are implicated in the genesis of ACS. Both apixaban and clopidogrel have favorable gastrointestinal effects when compared with acetylsalicyclic acid and dabigatran or rivaroxaban.[10]

There is always a delicate balance between hemorrhagic and ischemic risk in the management of patients with coexisting active IBD and acute coronary syndrome. Adoption of conservative approach with a close follow-up of the patient is one of the safe options after weighing the risk and benefits of all available therapies for every patient in such an enigmatic clinical scenario.

4 | CONCLUSION

A multidisciplinary team-based approach should be followed for the management of IBD patients with the aim to induce disease remission along with aggressive reduction of cardiovascular risk factors. The risk and benefit ratio must be individualized due to fragile balance between ischemic and hemorrhagic complication in patients with coexisting IBD and ACS.

AUTHOR CONTRIBUTIONS

Krishna Vani Nemalidinne: Writing – original draft; writing – review and editing. **Abinay Siva Kumar Reddy Vanteru Vanteru:** Writing – original draft. **Amit Goel:** Writing – original draft; writing – review and editing. **Abhigan Babu Shrestha:** Writing – original draft. **Vikash Jaiswal:** Supervision; writing – original draft.

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None.

CONFLICT OF INTEREST STATEMENT

The authors have no conflict of interest to declare.

DATA AVAILABILITY STATEMENT

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

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.

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