To the Editor: It is a challenge for a physician to manage acute ST-elevation myocardial infarction (STEMI) in a patient with upper gastrointestinal bleeding. We present a case of gastrointestinal bleeding due to antiplatelet therapy after percutaneous coronary intervention (PCI) and secondary in-stent thrombosis due to discontinuation of antiplatelet drugs. The experience of treatment of this case may shed some light on antiplatelet therapy in this clinical dilemma for patients with both high bleeding and high ischemia risk.

A 73-year-old man was admitted to the Department of Cardiology, First Affiliated Hospital, Dalian Medical University, with 3 h of severe chest pain on March 31, 2017. Initial electrocardiogram revealed a marked ST-segment elevation in leads II, III, AVF, and V7–V9, and serum cardiac troponin I was 67.9 µg/L. The diagnosis was acute STEMI (inferior and posterior). Coronary risk factors included hypertension and diabetes. The patient had received the first PCI with a sirolimus-eluting stent (2.5 mm × 33.0 mm) in the posterior descending branch of right coronary artery (PDA) due to acute non-STEMI on December 18, 2015, and the second PCI (sirolimus-eluting stent, 2.5 mm × 25.0 mm) in the left anterior descending artery due to unstable angina pectoris on March 16, 2016. The patient routinely received dual antiplatelet therapy (aspirin and clopidogrel) but did not receive proton pump inhibitor (PPI) and gastric mucosal protective agent. On March 11, 2017, upper gastrointestinal bleeding occurred with hemoglobin of 66 g/L, and gastric mucosal protective agent. On March 21, 2017, severe chest pain on March 31, 2017. Initial electrocardiogram revealed a marked ST-segment elevation in leads II, III, AVF, and V7–V9, and serum cardiac troponin I was 67.9 µg/L. The diagnosis was acute STEMI (inferior and posterior). Coronary risk factors included hypertension and diabetes. The patient had received the first PCI with a sirolimus-eluting stent (2.5 mm × 33.0 mm) in the posterior descending branch of right coronary artery (PDA) due to acute non-STEMI on December 18, 2015, and the second PCI (sirolimus-eluting stent, 2.5 mm × 25.0 mm) in the left anterior descending artery due to unstable angina pectoris on March 16, 2016. The patient routinely received dual antiplatelet therapy (aspirin and clopidogrel) but did not receive proton pump inhibitor (PPI) and gastric mucosal protective agent. On March 11, 2017, upper gastrointestinal bleeding occurred with hemoglobin of 66 g/L, shock and gastric ulcer were diagnosed; antiplatelet therapy was discontinued and 6 units of red blood cells was transfused; then, the hemoglobin increased to 81 g/L on March 21, 2017. During the current admission, a loading dose of aspirin (300 mg) and ticagrelor (180 mg) was given and primary PCI was immediately performed. Total occlusion with in-stent thrombus was found in PDA, and no residual stenosis remained after thrombus aspiration confirmed [Figure 1a and 1b]. During PCI procedure, bivalirudin was used. Antiplatelet monotherapy with ticagrelor 90 mg twice daily was given after PCI. The PPI and gastric mucosal protective agent were also given. At the 1-, 3-, and 6-month follow-up examination, the patient’s medical condition was stable. The maximum aggregation ratios (MAR%) in response to arachidonic acid (AA) and adenosine diphosphate (ADP) were 39.2% and 40.6% on May 10, 2017; 21.0% and 31.6% on July 13, 2017; and 46.1% and 38.7% on July 13, 2017, respectively.

Antiplatelet therapy with aspirin and P2Y12 inhibitors is the cornerstone of medical treatment in patients after PCI, which prevents ischemic events with increased risk of bleeding. The risk of upper gastrointestinal bleeding remains a significant problem and the reported incidence was 1.1% within 1 year after PCI, even up to 10.4% within 4 years in STEMI patients after primary PCI. In this case, antiplatelet therapy was discontinued after upper gastrointestinal bleeding and hemorrhagic shock since hemorrhagic events might be associated with a comparable or even higher mortality risk to thrombotic events. Acute STEMI occurred 20 days after discontinuation of antiplatelet therapy in this patient, which further confirmed that the discontinuation of antiplatelet therapy was associated with an increased risk of ischemic events after PCI. No residual stenosis after thrombus aspiration confirmed that discontinuation of antiplatelet therapy was the most important factor for drug-eluting stent-associated late thrombosis.

![Figure 1](image1.png)

**Figure 1:** (a) Total occlusion with in-stent thrombus was found in the posterior descending branch of right coronary artery before thrombus aspiration. (b) No residual stenosis remained after thrombus aspiration.

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This case challenged clinical decision-making, especially the antiplatelet strategy after primary PCI. There were several reasons for ticagrelor monotherapy as an alternative for long-term platelet inhibition in this patient receiving primary PCI and thrombus aspiration. First, a higher prevalence of gastrointestinal complications was demonstrated in STEMI patients after primary PCI, especially in patients with upper gastrointestinal bleeding and hemorrhagic shock. However, more intense platelet-directed therapy is necessary for late stent thrombosis. This is a particularly intractable problem. Second, aspirin was discontinued due to the mechanism of action of blockade of the COX-1 enzyme, and some studies have proved no episodes of stent thrombosis in patients who discontinued aspirin yet maintained P2Y12 inhibitor therapy. Ticagrelor is superior to clopidogrel and is the preferred P2Y12 inhibitor for acute coronary syndrome patients. The risks about inhibition of antiplatelet action of combined use of clopidogrel and PPIs should be considered. In addition, it was reported that the long-term incidence of major adverse upper gastrointestinal events was still relatively high (16%) in STEMI patients undergoing PCI with in-hospital major adverse upper gastrointestinal events although clopidogrel and PPI were together used after discontinuation of aspirin. Finally, TWILIGHT study has proved that ticagrelor monotherapy could significantly reduce bleeding compared with ticagrelor plus aspirin in high-risk PCI patients, with comparable efficacy to conventional double antiplatelet therapy, after 3-month course of ticagrelor plus aspirin. One study further proved that P2Y12 receptors were critical to the generation of irreversible aggregation through the TXA2-dependent pathway. As a result, strong P2Y12 receptor blockade alone caused inhibition of platelet aggregation that was minimally enhanced by aspirin. The platelet function monitoring showed that both AA-MAR% and ADP-MAR% were <50% at three follow-up visits after discharge, which suggested that ticagrelor monotherapy could also reduce the production of TXA2, and strong P2Y12 receptor will also challenge the current paradigm mandating the universal need for aspirin after PCI.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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