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CASE REPORT

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Coronary thrombosis due to heparin-induced thrombocytopenia after percutaneous coronary intervention: Easy to miss, uneasy to prevent

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Abstract

In case of unexplained post-PCI coronary thrombosis, a HIT should be always considered. A pragmatic approach with bivalirudin may be reasonable, even in absence of confirmed laboratory diagnosis.

KEYWORDS

Coronary artery disease, Heparin-induced thrombocytopenia, PCI complication

1 | INTRODUCTION

Heparin-induced thrombocytopenia (HIT) is a rare immunological reaction to heparin leading to both arterial and venous thrombosis. HIT is infrequent and its clinical features inconstant, so after a percutaneous coronary intervention (PCI) a correct diagnosis may be missed with a negative prognostic impact. We describe an illustrative case.

Heparin-induced thrombocytopenia (HIT) is a well-known prothrombotic condition associated with the use of heparin or its derivatives. Treatment consists in the replacement with an alternative anticoagulant, but diagnosis may be challenging because of variable onsets and absence of the typical fall of platelet count. We describe an atypical presentation of HIT with a subsequent missed diagnosis discussing about possible preventive proceedings.

2 | CASE REPORT

A 68-year-old diabetic, hypertensive male patient was admitted to our cath-lab for anterior ST elevation acute myocardial infarction (STEMI) at 10 PM Four months before he had been hospitalized for pneumonia and respiratory distress. At admission, vital parameters were stable and blood samples were unremarkable except for a mild thrombocytosis (431 000/ mm^3) and elevated high-sensitive troponin. Coronary angiography (CA) revealed the occlusion of left descending artery (LDA) (Figure 1, Panel A). We performed a primary percutaneous coronary intervention (PCI) positioning a drug-eluting stent (DES) both at proximal LDA and at first diagonal branch with a good angiographic result (Figure, Panel B). During PCI, we administered unfractionated heparin (UFH) 7500 UI), acetyl-salicylic acid 300 mg intravenously and ticagrelor 180 mg orally. During PCI, the activated clotting time was 255 s. Soon after PCI, we did not observe any complication. Postprocedural biology revealed a significant increase of troponin and a mild hyperglycemia. On day 2, in the morning laboratory tests confirmed the alterations of the day before; platelet count was 339 000/ mm^3. At 2 PM of the day 2, 14 hours after PCI, we performed a new urgent CA because of chest pain with inferior ST elevation. A second CA showed an occlusion of proximal right coronary artery (RCA) (Figure, Panel C) and patent LDA. We performed a

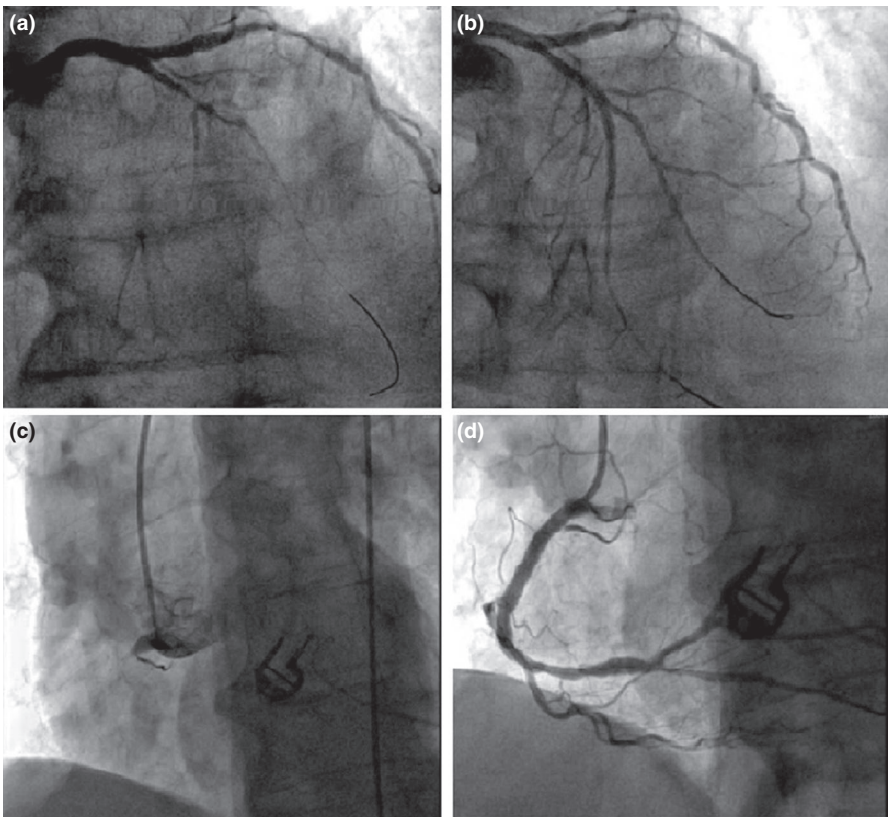

FIGURE 1 A, Occlusion of LDA at CA; B, Angiographic result after stenting of LDA and first diagonal; C, Occlusion of RCA; D, angiographic result after stenting of RCA. LDA: left descending artery; CA: coronary angiography; RCA: right coronary artery

primary PCI with DES of RCA obtaining ST-T normalization and remission of chest pain. During PCI, we administered UFH 7500 UI intravenously. An extraction for postprocedural blood count was performed, but 2 hour after the second PCI, the patient experienced a hemodynamic instability. A bedside echocardiogram showed a free wall rupture of left ventricle. Despite cardiopulmonary resuscitation maneuvers, inotropes, and pericardiocentesis, after 30 minutes, we ascertained the exitus. Biology, obtained 2 hours later, revealed a severe decrease in platelet count (88,000/mm^3). Because of the rapid exitus, we were not able to perform laboratory tests specific for HIT. Autopsy showed fresh thrombosis of both RCA and LDA. Following the 4 T's rule, a HIT was highly probable (1). In fact, the patient experienced a fall of platelet count superior than 50%, with a thrombotic event related to the administration of UFH without an alternative cause of thrombocytopenia.

3 | DISCUSSION

HIT is an immune-mediated decrease of platelet count associated with the use of heparin and its derivatives. The formation of heparin/platelet factor 4 (PF4) complexes and their immunogenicity is the pathological basis of the HIT that typically consists in a fall of the platelet count by more than 30%-50%. The prevalence varies from 0.1% to 5% in different observational studies.[1] In up to 50% of patients with a diagnosis of HIT, a thrombotic complication is described: this association is defined HIT with thrombosis (HITT).[1] The diagnosis of HIT begins from clinical aspects but should be confirmed by laboratory tests. The 4 T's Clinical Scoring System is the most used tool for excluding a HIT because of its strong negative predictive value and "ease of use".The positive predictive value varies from 10% for an intermediate score (4 points) to 80% for a high score (8 points). In case of intermediate or high probability of HIT, immunological or functional assays should be rapidly performed.[1]

Thrombocytopenia appears 5-10 days after heparin exposure in 60% of cases. In 30% of cases, a rapid onset, immediately after exposure, is also described. Rarely, a delayed onset may occur up to 3 weeks after exposure.[2] As UFH is the most used anticoagulant in the cath-lab, patients undergoing a PCI have a not negligible risk of HIT. In literature cases of coronary thrombosis after PCI with typical HIT but also atypical cases without thrombocytopenia are described.[3-5] The impact of HIT on coronary thrombosis after a PCI has been also described. In a retrospective study evaluating patients undergoing a PCI with an ischemic recurrence within 24 hours, one patient out of four had a diagnosis of HIT. Interestingly, the typical platelet count decrease was not always present.[6]

The management of HIT consists in the prompt interruption of heparin administration and the use of alternative anticoagulants as direct thrombin inhibitors (DTI). Bivalirudin, a synthetic analogue of hirudin, is strongly recommended for STEMI patients with HIT.[1,7]

In our report, the onset of thrombocytopenia is rapid, about 16 hours from the first UFH exposure and we missed the diagnosis of HIT because of the absence of typical fall of platelet count in the period between the first and the second STEMI. So, an early myocardial re-infarction postprimary PCI had been erroneously attributed to 'coronary' causes rather than a prothrombotic condition and a bolus of UFH had been administered during the second PCI. After a careful history, the high risk of HIT probably was related to the administration of enoxaparin during the recent hospitalization for pneumonia.

A recent use of UFH or derivatives should be routinely investigated before a PCI. Moreover, in case of early coronary thrombosis, even within 24 hours, especially if unexplained (eg, coronary thrombosis in nonculprit coronary arteries or not related to stent malapposition/under-expansion), a HITT should be always suspected. Nevertheless, at the same time the laboratory tests for HIT should be started. As this possibility is rare, a more extensive utilization of bivalirudin is reasonable. About an eventual over-utilization of bivalirudin, we should consider that it is a therapeutic option in the setting of acute coronary syndromes, independently from the diagnosis of HIT, even if with a lower level of recommendation.[8]

Atypical rapid HITT may be arduous to recognize. An approach based on thrombosis and clinical setting rather than on laboratory tests could prevent serious events.

4 | CONCLUSION

HIT is an infrequent complication after PCI. As frequently atypical, a correct and prompt diagnosis may be missed. A more extensive use of bivalirudin in unexpected and early coronary thrombosis after PCI could be a reasonable strategy to prevent HITT.

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Published with written consent of the patient.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

All authors were involved in the clinical management of this patient and contributed to the preparation of this manuscript.

INFORMED CONSENT

Informed consent was obtained from all individual participants included in the study.

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